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Disaggregating the Distal, Proximal, and Time-Varying Effects of Parent Alcoholism on Children's Internalizing Symptoms

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Abstract We tested whether children show greater internalizing symptoms when their parents are actively abusing alcohol. In an integrative data analysis, we combined observations over ages 2 through 17 from two longitudinal studies of children of alcoholic parents and matched controls recruited from the community. Using a mixed modeling approach, we tested whether children showed elevated mother- and child-reported internalizing 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). No support for time-varying effects was found; proximal effects of mothers' alcohol-related consequences on child-reported internalizing symptoms were found and distal effects of mother and father alcoholism predicted greater internalizing symptoms among children of alcoholic parents. Implications for the time-embedded relations between parent alcoholism and children's internalizing symptoms are discussed.

Keywords Parent alcoholism · Internalizing symptoms · Integrative data analysis · Intergenerational transmission · Time-varying effects

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Introduction

Previous studies of children of alcoholic parents (COAs) document an elevated risk not only for the early onset of symptoms beginning in childhood (e.g., Edwards et al. 2001; Loukas et al. 2001; Puttler et al. 1998; Tubman 1993) but also for troubling developmental trajectories that indicate a continuance of risk for symptom disturbance and disorder. Such trajectory-based analyses are increasingly common in developmental studies of risk factors for child psychopathology. Most of these studies adopt a "launch" or "catapult" model in which risk factors assessed at baseline are evaluated as static predictors of change over time in child outcomes (Kinderman and Skinner 1992). Within studies of COAs, these trajectory analyses show that parent alcoholism is associated with higher rates and faster escalation in substance use over time (e.g., Chassin et al. 1996). On the other hand, parent alcoholism does not predict changes in children's internalizing symptoms or social competence over time (Hussong et al. 2007; Hussong et al. 2005).

One explanation for this lack of prediction of changes in children's symptoms is that the launch model is not sensitive to the more immediate impact and time-varying fluctuations of parent alcoholism on children's functioning.

Supporting this possibility, Loukas et al. (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 et al. (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. Further evidence for the importance of the timing of parent alcoholism comes from studies showing that children whose alcoholic parents are recovered show intermediate risk compared to children with currently alcoholic parents and children of non-alcoholic parents (for substance use outcomes, Chassin and Barrera 1993, and for social competence outcomes, Hussong et al. 2005). These results indicate that some effects of parent alcoholism may be more proximal and that fluctuations in parent impairment due to alcohol involvement may impact patterns of child functioning over time.

Although parent alcoholism has a broad impact on child functioning (Chassin et al. 1991), we focus here on internalizing symptoms specifically. Internalizing symptoms show a high rate of comorbidity with other indices of child functioning and, unlike externalizing symptoms and substance use, show little change over time in relation to parent alcoholism in launch models. For example, in previous analyses of data in the current study, we found that although subgroups of COAs showed stable elevated mother- and child-reported internalizing symptoms from ages 2 through 17, parent alcoholism did not predict fluctuations or changes in internalizing symptoms over time (Hussong et al. 2007).

In the current study, we follow-up these analyses to distinguish between three effects of parent alcohol-related consequences on children's internalizing symptoms. The first is a within-subjects or time-varying effect that indexes whether children show increased (or decreased) internalizing symptoms, over their usual baseline, at those times when their parents also show increased (or decreased) alcohol-related symptoms. The second is a between-subjects proximal effect that indexes whether children whose parents show greater alcohol-related consequences during a developmental period in turn show greater internalizing symptoms during that same period. Thus, time-varying effects focus on issues of timing, whether children's internalizing 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 internalizing symptoms during this time. The third effect is a baseline and (relatively) distal influence; this is also a between-subjects effect but the focus is on the impact of parent symptomatology that largely predates the developmental period and is not influenced by changes in parent symptomatology over the developmental period.

This distal influence has been the focus of most research indexing COAs' risk for a variety of negative outcomes and reflects a host of influences that impact the child through a launch process. Most pertinent to the current study, stress-coping models of internalizing symptoms and depression suggest that an interaction of genetic vulnerability and environmental press (e.g., family conflict and adversity, major life stressors, abuse and neglect) may overtax coping resources and result in emotional dysregulation, eventually culminating in depressive symptoms (Graber 2004). This may be a particularly relevant model for understanding the effects of parent alcoholism on children's internalizing symptoms as family linkage studies suggest the possibility of co-transmission for depression and alcoholism, such that COAs may receive a dual genetic vulnerability for both disorders (Kendler et al. 1994; Merikangas et al. 1985; Zucker 2006). Moreover, COAs show a clear risk for greater life stress in the form of family conflict and disturbance, child abuse and neglect, and more stressful life events (Chassin et al. 1991; Sher et al. 1997). In turn, some evidence suggests that these stressful life events may mediate the relation between baseline parent alcoholism and adolescents' internalizing symptoms (Chassin et al. 1993).

Less clear in this model, however, is the role played by the timing of parent alcoholism. Distal effects of parent alcoholism index a mix of genetic vulnerability and environmental stress that in turn set children and adolescents on a negative course for internalizing symptoms. The risk resulting from the distal effects of parent alcoholism does not require that children be directly exposed to their parents' alcoholism, but merely to a more stressful environment or more intense genetic diathesis at some point prior to the study period. Consistent with this notion is evidence that children and adolescents experience physiological changes and develop patterns of biased information processing as a result of major stress events that may in turn place them at long-term risk for negative outcomes, including internalizing symptoms (Alloy et al. 2006).

However, equally plausible is that distal effects of parent alcoholism index a persistently stressful environment, such that intervening events have a more immediate impact on children and adolescent's internalizing symptoms. As such, children whose parents are actively abusing alcohol may experience greater stress during a given developmental period and thus be at greater risk than their peers for internalizing symptoms. As a result, proximal effects of

parent alcoholism (i.e., greater parent symptoms during the developmental period) may actually be a stronger index of children's risk for internalizing symptoms than distal parent alcoholism. Moreover, if environmental stress is directly related to parents' alcohol-related symptoms, then children may experience the greatest risk for internalizing symptoms when their parents' symptoms are elevated; this hypothesis posits a time-varying effect of parent alcoholism predicting the timing of elevations in, and not just level of, children's internalizing symptoms.

The Current Study

In the current study, we examined three effects of parent alcoholism on children's internalizing symptoms that we refer to as distal, proximal, and time-varying effects. These effects differentially focus on inter-individual (i.e., distal and proximal effects) and intra-individual (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 examined these effects using an integrative data analysis approach in which we simultaneously analyzed two nationally prominent prospective studies of COAs and matched controls recruited from the community (see Hussong et al. 2007). We also study the effects using mother-reports of symptomatology, permitting the inclusion of young children, as well as child-reports, drawing on the relatively stronger validity of adolescents' reports of internalizing symptoms. Building on results of our previous analyses of these samples showing significant distal effects of parent alcoholism on the level (but not changes) of children's internalizing symptoms (Hussong et al. 2007), we focus here instead 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 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.[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 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 3-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 siblings of the male target child in cohort one who were aged 3–11 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, 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, 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 that was blind to family diagnostic status. Although protocol length varied by wave of assessment, parent assessments typically involved 9–10 h

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

of data collection and child assessments were typically 7 h 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 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 (DIS) 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 et al. 1991, 1992). 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 SES 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 and West 2000), mother- and child-report samples are considered with respect to the underlying age distribution rather than assessment waves. Across MLS and AFDP, 781 mothers reported on their children's internalizing symptoms in at least one assessment period between ages 2 and 17, reporting on 1,026 children and adolescents and providing a total of 2,801 observations (see Table 1). This mother-report sample was 63% male, 13% minority (primarily Hispanic), and 66% COA, with 44% having parents with a high school education or less and 26% having at least a college degree. For the child-report sample, only AFDP participants were included (see rationale in results), resulting in a sample of 454 adolescents and 1,349 observations between ages 10 and 17. The child-report sample was 53% male, 29% Hispanic, and 54% COA, with 27% of families having parents with less than a high school education and 30% having at least a college degree.

Measures

Demographic variables included participant gender (0 = female; 1 = male), age, ethnicity (0 = Caucasian, 1 = Hispanic or African American)[2] and parent education (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).

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 DIS (version III; Robins et al. 1981, 1982), the Short Michigan Alcohol Screening Test (Selzer et al. 1975), and the Drinking and Drug History Questionnaire (Zucker et al., unpublished manuscript). 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 also directly interviewed at baseline using a computerized version of the DIS 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 (FH-RDC) (Andreasen et al. 1977). Thus, a lifetime diagnosis at

[2] Because the sample contained very few African American participants, we were not able to look at effects separately for African American and Hispanic participants.

Table 1 Demographic characteristics within and across studies and reporters

	Mother-report			Self-report
	MLS	AFDP	Total	AFDP
% male	71	53	63	53
% Caucasian	98	72	87	71
Parent education				
% with high school education or less	57	26	43	27
% college graduate	23	30	26	30
% COA	75	54	66	54
% with an alcoholic mother	32	13	24	13
% with an alcoholic father	72	47	62	48

Self-report N=454, mother-report N=1,026

the time of the baseline assessment was made based on DIS self-reports or FH-RDC 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 consequences in the past year. The consequences are related to indicators of DSM-IV criteria for alcohol abuse and dependence and include problems with friends and family due to drinking, drinking-related offenses, problems at work or school due to drinking, suffering blackouts, signs of tolerance, loss of control over drinking, feeling guilty about drinking, and drinking first thing in the morning. All items were dichotomized and summed within wave to form the repeated measures indicating the time-varying effects of parent alcoholism. Across observed ages, means ranged from 0.25 (0.94) to 0.84 (1.74) for mothers' consequences and 0.22 (0.78) to 1.02 (1.61) for fathers' consequences in child-report models and 0.14 (0.77) to 2.61 (2.00) for mothers' consequences and 0.87 (1.46) to 2.83 (2.12) for fathers' consequences in parent-report models. These time-varying indicators were then averaged across wave (within-person) to create the proximal indicator of parent alcoholism. By creating time-varying (or within-person) and proximal (or between-person) effect 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 et al., 2007b).

Child internalizing symptoms were assessed via mother- and child-report. In both studies, participants completed the Child Behavior Checklist (CBCL) (MLS) or an adapted form of the CBCL (AFDP; Achenbach and Edelbrock 1978). In the current study, we examined 13 items from the CBCL anxiety-depression subscale (although three items were not administered to adolescents in AFDP and thus coded as missing for these participants). The response scale ranged from 0–2 for parent-report and for child-report in MLS and from 0–4 for child-report in AFDP, with an assessment window of past 6 months for MLS and past 3 months for AFDP.[3] For the current study, we chose to dichotomize these items as absent or present because of sparse response frequencies in the other response category.

We then derived scale scores from these indicators using item response theory (IRT; Embretson and Reise 2000). IRT has several strengths in measurement evaluation and development including the abilities to consider differential item contributions to the scale scores, to capture greater true score variability in the pattern of symptom endorsement than proportion scores, to create a true interval-level metric for the latent construct, and to test and incorporate measurement invariance across discrete groups, such as those defined by study membership, age, and gender (see Curran et al. 2007a; Khoo et al. 2006).

Details of our IRT analyses are reported in Hussong et al. (2007). In brief, we conducted both Differential Item Functioning (DIF) testing and item calibration using all available participants from AFDP and MLS for mother-reports (N=1,026) and child-reports (N=971). (Note that child-reports were calibrated by including MLS participants for greater generalizability, although these participants are not included in subsequent analyses because parents reports of consequences and child reports of symptoms only co-occurred in one wave.) We tested for DIF in age (age 2 to 11 [n=475] vs. age 12 to 17 [n=551] for mother-reports and age 10 to 13 [n=429] vs. age 14 to 17 [n=542] for child-reports), child gender, and study sequentially. Items with DIF were split into two "sub-items" (e.g., one for young participants and one for old participants, with the sub-item not pertaining to a particular group coded as missing) to derive separate item parameter estimates as a function of age or gender so that subsequent scoring would account for DIF (see Flora et al. 2007). In the calibration phase, we estimated the discrimination and severity parameters for each of the items and sub-items by fitting the 2PL model to the item responses from the calibration sample using MULTILOG (Thissen 1991). In the scoring phase, we used MULTILOG to calculate *maximum a posteriori* scores for each participant's set of repeated observations based on her or his item responses and our parameter estimates from the calibration phase (Thissen and Orlando 2001). These scores served as the observed dependent variables in the growth models described below. Across observed ages, means ranged from 0.22 (0.78) to 0.57 (0.75) for child-reports and −0.67 (0.44) to 0.32 (0.85) for parent-reports.

[3] Differences in the assessment window for this instrument are part of the study effect which was tested in all aspects of analyses. As detailed later, results of testing in the IRT analyses, however, suggested no study differences in measurement structure after accounting for gender and age effects.

Results

Our integrative data analysis occurred in three phases involving missing data imputation, constructing trajectories, and hypothesis testing. For analyses of mother-reports of children's internalizing symptoms, we included data from both the MLS and AFDP studies. However, because the MLS had only 1 year where parent alcohol-related symptoms were assessed simultaneously with child- (not parent-) reported internalizing symptoms, we only examined time-varying models pertaining to child-reports of internalizing symptoms in AFPD. Below we present an overview of the analytic strategy applied to the samples for each reporter of symptomatology and then present results for each separately.

Analytic Approach

Our statistical approach permits simultaneous analysis of data drawn from different longitudinal studies. Although we are drawing on existing methodologies, we combine these techniques in novel ways consistent with what McArdle and Horn (2002) refer to as mega-analysis. First, we addressed the issue of missing data in our time-invariant and time-varying covariates through multiple imputation (Rubin 1987). For these analyses, we used the internalizing IRT scores from 2,081 assessments of 1,026 participants in the mother-report sample and 1,349 assessments of 454 participants in the child-report sample. We used SAS PROC MI (SAS 1999) to impute missing data in the time-invariant covariates and the R package PAN (Schafer, unpublished manuscript) 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.[4]

[4] Because both imputation packages rely on Markov-Chain Monte-Carlo methods to draw samples from the posterior predictive distribution of the missing data given the observed data, we used graphical methods reviewed by Cowles and Carlin (1996) to assess whether the simulated "chains" had properly converged. We found that due to the relatively small proportion of missingness (i.e., the highest proportion of missing cases is 20.4% for the Father's Depression diagnosis, and the fraction of missing information for this variable 20.1%.), the chains converged very fast, generally moving out of the initial phase and converging to the target (posterior) distribution in as few as 200 iterations. The low fraction of missing information in most variables ensured that with just $M=10$ imputations, we achieved "relative efficiency" of at least 0.98 (see Rubin 1996).

We next examined the functional form of trajectories characterizing mother- and child-reported internalizing symptoms over time. Similar preliminary analyses are presented in Hussong et al. (2007) and thus are not a focus here. However, these analyses differ from those previously presented in that (a) here we used a random coefficients modeling approach rather than a structural equation modeling approach and thus require (b) the imputation of missing time-invariant covariates (i.e., these cases were omitted in Hussong et al.), and (c) here we included only AFDP in the analysis of child-reported symptoms. (MLS was included in Hussong et al. because the unavailability of repeated assessments of parent alcohol-related symptoms did not preclude testing of the baseline influence of parent alcoholism.) Thus we briefly present replications of these trajectory analyses.

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). Below, we report the combined results.

Results of Mother-Report Sample

In previous analyses of the current data, we found that change over time in mother-reported internalizing symptoms was best characterized by a piecewise growth curve in which the first linear piece describes change from age 2 to 7 and the second describes change from age 7 to 17 (see Raudenbush and Bryk 2002, pp. 178–179). The random intercept effect in this model represented internalizing at age 13. To control for cross-study effects, we added a dummy covariate to represent the study effect. Because only participants from the MLS were observed during age 2 to 7, the interaction of the study covariate with the first linear piece was not included. The unconditional model was a linear multilevel model with 3 levels of nesting, namely repeated measures within participants and (in MLS) participants within families. We fitted this model using SAS PROC MIXED with the restricted maximum likelihood estimator. To account for individual variability in the growth trajectories, we let the three parameters that characterized the growth curve (the intercept, the first piece, and the second piece) vary randomly among participants. We also estimated a random intercept at the family level, thus including a variance component to account for the between-participant correlation in the same family. Consistent with Hussong, Flora, et al, this model also fits well with data in the

current study.[5] Results indicated that internalizing scores increase during childhood and begin to decrease after age 7, though to a greater extent in MLS than in AFDP.

We next added baseline demographic variables (gender and parent education) as individual level covariates to the unconditional model as well as cross-level interactions between demographic variables and time (i.e., as indexed by the first and second piece of linear growth). We also tested whether interactions between the demographic variables and study were associated with variability in the linear slope, but found no such effects (all $p>0.05$). Overall, these results imply that no study effects are present in the impact of demographic factors on internalizing scores. For parsimony, we did not include non-significant study interactions in our final model.

To test the effects of within- versus between-person effects, we followed Raudenbush and Bryk (2002, p. 134–141). Specifically, we added person-mean centered time-varying covariates for mothers' and fathers' alcohol-related consequences as repeated measures (i.e., the within-person or time-varying effect) and the report of these consequences averaged over repeated assessments as time invariant covariates (i.e., the between-person or proximal effect). We also added interactions between each of these predictors with study to test for differences in findings based on MLS versus AFDP. No significant study effects were found, and thus these interactions were trimmed in the final model. (No substantive differences between the full and trimmed models were found.) As shown in Table 2 (model 1), we found no time-varying or proximal effects of either mother's or father's alcohol-related consequences on mother-reported child internalizing.

We next added the baseline effects of mothers' and father's lifetime diagnosis of alcoholism to this same model and as well as interactions between each of these effects and study. A significant interaction of father's lifetime diagnosis with study was found, and thus was retained in the final model (presented in Table 2, model 2). Both mothers' and fathers' lifetime diagnosis predicted greater mother-reported child internalizing symptoms, although father's alcoholism diagnosis was a significant predictor only in AFDP ($\beta=0.20$, $t=3.05$, $p<0.001$) and not in MLS ($\beta=-0.05$, $t=0.77$, $p>0.10$).

To examine whether the lack of time-varying effects of parent alcoholism was due to moderating gender differences, we next included cross-level interactions between child's gender and the time-varying effects of mothers' and fathers' alcohol-related symptoms in these models. As shown in Table 2 (model 3), no gender differences were found.

Results of Child-Report Sample

Because the child-report analyses included data only from AFDP, we did not rely on our previous analyses of the child-report data (which included MLS) in guiding our analyses. Thus, we first fit a series of unconditional growth curve models to specify the optimal functional form of growth for the internalizing scores and to establish a firm basis for moving to conditional growth models examining the effects of covariates on random growth factors (see McArdle 1988). These forms of growth included a simple linear model, a quadratic model, and a set of piecewise linear models with varying ages for the "knot point" at which the two linear pieces were joined (see Bollen and Curran 2006, p. 103–106). Because these models were not formally nested, we compared their fit to the data by examining the match between the model-implied growth trajectories and the observed means at each age. Results indicated that child-reports of internalizing symptoms were best represented by a quadratic model in which linear and quadratic effects of age (centered at age 13) were included in the model as predictors of the repeated measures. The unconditional model was a linear multilevel model with 2 levels of nesting, namely repeated measures within participants. We fitted this model using SAS PROC MIXED with the REML estimator. To account for individual variability in the growth trajectories, we let the intercept and linear effects characterizing the growth curve vary randomly among participants and fixed the random component of the quadratic effect to zero. This model fit the data well and results indicated that child-reported internalizing generally decreased from ages 10 to 13 and then remained fairly stable through age 17.

As in mother-reports, we included baseline demographic variables (gender and parent education) as individual level

[5] Because indices of global model fit are not available within the mixed modeling framework, we judged fit by examining convergence among several indicators. We examined Akaike information criterion (AIC) and Bayesian information criterion (BIC) values (for these non-nested models reflecting linear, quadratic, and various piece-wise models) to determine functional form of the trajectories. (Actual AIC and BIC values available from first author by request.) Given that there are no formal thresholds for these model indices (rather model adequacy is based on relative comparisons across models) we used these indices more as guidelines than as absolute indices of fit. Our decision about model fit was based on a combination of these information-theory criteria tests, significance testing of the fixed and random effects (i.e., the linear, quadratic and piece-wise components), and visual inspection of graphs (e.g., plotting observed and model-implied means as a function of sample size).

Table 2 Effects of parent alcoholism on mother-reported child internalizing symptoms

Predictors	Model 1		Model 2		Model 3	
	Estimate	t-value	Estimate	t-value	Estimate	t-value
Control variables						
Linear age 2–11 years	**0.13**	**9.29**	**0.13**	**9.19**	**0.13**	**9.07**
Linear age 11–17 years (T2)	**−0.03**	**−2.36**	**−0.03**	**−2.23**	**−0.03**	**−2.27**
Study membership	**−0.44**	**−8.01**	**−0.32**	**−4.22**	**−0.32**	**−4.21**
T2 × study	**0.04**	**2.60**	**0.04**	**2.54**	**0.04**	**2.56**
GEN	*−0.08*	*−1.83*	−0.08	−1.68	−0.08	−1.65
EDU	−0.02	−1.26	−0.02	−1.29	−0.02	−1.29
T2 × GEN	**−0.03**	**−3.18**	**−0.03**	**−3.25**	**−0.03**	**−3.17**
Within-person effects						
MAC	0.02	1.42	0.02	1.35	0.03	1.03
DAC	0.00	0.45	0.00	0.44	−0.01	−0.37
MAC × GEN					−0.01	−0.27
DAC × GEN					0.02	0.77
Between-person effects						
MAC	0.03	1.64	0.02	1.05	0.02	1.07
DAC	0.01	0.94	0.01	0.48	0.01	0.49
Baseline effects						
MAC			**0.17**	**3.41**	**0.17**	**3.40**
DAC			**0.20**	**3.05**	**0.20**	**3.05**
DAC × study			**−0.26**	**−2.93**	**−0.26**	**−2.94**

Within-person predictors are the repeated annual assessments of parents' alcohol-related consequences within the past year, between-person predictors are the average of these repeated annual assessments within each person, and the baseline predictors are the wave one lifetime reports of parents' alcohol-related consequences. Bold estimates are significant at $p<0.05$, italicized are significant at $p<0.10$
GEN = child gender, EDU = parent education, MAC = mom alc con, DAC = dad alc con

covariates to the unconditional model as well as cross-level interactions between demographic variables and time (i.e., as indexed by the liner and quadratic slopes). However, because no interactions of demographic variables and slopes were significant, we did not retain them in subsequent analyses. We next tested time-varying versus proximal effects again adding the person-mean centered time-varying covariates and time-invariant (aggregated) covariates for mothers' and fathers' alcohol-related consequences to this model (see Table 3, model 1). We found no time-varying effect of parents' alcohol-related consequences, but the proximal effect of mothers' consequences was a significant predictor of child-reported internalizing symptoms ($\beta=0.11$, $t=3.40$, $p<0.001$). As such, adolescents reported greater internalizing symptoms if their mothers' reported greater alcohol-related consequences during the study period.

This effect remained after controlling for the distal influence of parents' alcoholism diagnoses (see Table 3, model 2). In addition, both mothers' and fathers' lifetime alcoholism diagnosis predicted greater internalizing symptoms in adolescents, controlling for the time-varying and proximal effects of parent alcoholism ($\beta=0.20$, $t=1.99$, $p<0.05$ and $\beta=0.20$, $t=3.20$, $p<0.001$, respectively). Moreover, no significant gender differences in the time-varying effects of parent alcoholism were found (see Table 3, model 3).

Discussion

Using an integrative data analysis framework, we examined the time-varying, proximal and distal effects of parent alcoholism on mother- and child-reports of children's internalizing symptoms. There were several strengths to our approach, including the use of large, longitudinal samples drawn from two studies (for mother-reports), the inclusion of multiple reporters of children's symptoms, direct ascertainment of parent alcoholism in a community recruited sample with a matched contrast group, and inclusion of all cases through the use of longitudinal analyses that incorporated imputed data to account for missingness. These strengths lend confidence to our findings which show few proximal and no time-varying effects of parent alcoholism beyond distal influences.

The dominance of distal influences over proximal and time-varying effects in capturing the impact of parent alcoholism on children's internalizing symptoms runs counter to theories of child psychopathology that conceptualize parent–child influences as dynamic and driven by

Table 3 Effects of parent alcoholism on child-reported internalizing symptoms

Predictors	Model 1		Model 2		Model 3	
	Estimate	t-value	Estimate	t-value	Estimate	t-value
Control variables						
Linear age effect	**−0.03**	**−2.08**	*−0.03*	*−1.92*	*−0.03*	*−1.84*
Quadratic age effect	*0.01*	*1.86*	*0.01*	*1.84*	*0.01*	*1.81*
GEN	**−0.16**	**−2.66**	**−0.15**	**−2.51**	**−0.15**	**−2.51**
EDU	−0.04	−1.33	−0.04	−1.29	−0.04	−1.29
Within-person effects						
MAC	0.01	0.32	0.01	0.32	0.04	1.14
DAC	0.00	0.00	0.00	0.01	0.02	0.79
MAC × GEN					−0.06	−1.18
DAC × GEN					−0.04	−1.03
Between-person effects						
MAC	**0.11**	**3.40**	**0.08**	**2.31**	**0.08**	**2.32**
DAC	0.01	0.60	−0.01	−0.31	−0.01	−0.31
Baseline effects						
MAC			**0.20**	**1.98**	**0.20**	**1.99**
DAC			**0.20**	**3.20**	**0.20**	**3.20**

Within-person predictors are the repeated annual assessments of parents' alcohol-related consequences within the past year, between-person predictors are the average of these repeated annual assessments within each person, and the baseline predictors are the wave one lifetime reports of parents' alcohol-related consequences. Bold estimates are significant at $p<0.05$, italicized are significant at $p<0.10$
GEN = child gender, EDU = parent education, MAC = mom alc con, DAC = dad alc con

real-time processes (e.g., Granic and Patterson 2006). However, alternate views note the 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.

Alternatively, and more specific to the focus of the current study, internalizing symptoms may too narrowly define child functioning to capture the ways in which COAs respond to those periods of life when their parents are actively abusing alcohol. This possible explanation is consistent with previous studies showing that the strongest specific effects of parent alcoholism on child functioning indices are, not surprisingly, for alcohol involvement itself (Chassin et al. 1991). Moreover, we might expect the time-varying and proximal effects of parent alcoholism on children's externalizing symptoms to be more evident even in childhood because externalizing symptoms consistently show stronger relations with alcohol involvement than do internalizing symptoms (e.g., Hussong et al. 1998). 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.

Presenting a third option, the proximal and time-varying effects of parent alcoholism on children's internalizing symptoms may only be evident given the absence of 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 et al. 1980). Moreover, some work has suggested that the functioning of the non-alcoholic parent may play a protective role, though findings are mixed (Curran and 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 and Chassin 1997). Each of these protective factors appears to have the greatest implications for dispelling the proximal or 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.

Fourth, distal risk processes may 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 become depressed as a result of parent drinking problems but once

they become depressed, they may withdraw from social activity. The lack of positive social activities (and maybe associated social skills deficits) results in these children becoming more depressed. As such, the original depression trajectory starts because of parental drinking but then it becomes attached to other predictors over time.

Finally, we may see that time-varying effects differ over varying short-term time intervals. Our model of time-varying effects tested simultaneous associations between parent symptomatology and child internalizing symptoms. However, such effects may only become evident after a certain period of exposure, such that, for example, cross-sectional associations are not evident but time-varying associations over a 1 year lag are. This may occur because parental disturbance that does not last for a sufficient interval will not overcome the child's normal orthogenic "righting tendencies". It is only when parental disturbance is severe or prolonged enough that children will begin to evidence resulting internalizing symptoms.

Despite the general lack of support for proximal effects of parent alcoholism on children's internalizing symptoms, we did find greater proximal, though not time-varying, effects of maternal alcohol-related symptoms on child-reported internalizing symptoms. Although this singular finding begs replication prior to generalization, it may be that, particularly during adolescence, having an actively alcoholic mother increases risk for internalizing symptoms in youth that go largely undetected by these parents. Because mothers typically serve as primary caregivers and alcoholic women are often married to alcoholic men (confounding the effects of maternal alcoholism with those of having two alcoholic parents), this proximal effect may be limited to maternal alcoholism. Nonetheless, no effects were found for fluctuations in child symptomatology to mirror those of their parents.

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 internalizing symptoms, mechanisms that operate early and provide a stable influence over the life course should be primary targets for exploration. The lack of proximal effects has developmental implications as well, indicating that much of the risk for internalizing symptoms shown by COAs is evident early in the lifecourse (Hussong et al. 2007) and persistent at least though young adulthood (Curran et al. 2007a, b). Moreover, the dominance of distal effects indicates that prevention efforts targeting COAs' internalizing symptoms per se should occur in early childhood and, likely, be embedded in a larger intervention addressing other negative outcomes that may more proximally impact the course of internalizing symptoms in COAs over time.

However, these implications should be tempered by limitations of the current study. Limited ethnic diversity in our samples 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 internalizing 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 internalizing symptoms rely on symptom checklists, which may not relate directly to risk for disorder, and targets the internalizing spectrum broadly. As such, we were unable to effectively distinguish between symptoms of depression and anxiety, limiting specificity (Graber 2004). In addition, data were only conducive for the child-report analyses in one of our two contributing studies. Although this results in greater power for our mother-report analyses, the dominance of distal over time-varying and proximal effects of parent alcoholism on children's internalizing symptoms was evident regardless of reporter. 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 results of the current study call into question the proximal influence of parent alcoholism on the specific outcome of children's internalizing symptoms. Although such influences may emerge for other negative outcomes and in the absence of important protective factors, the primary influence of parent alcoholism on children's internalizing symptoms appears to be a distal one. As such, COAs show elevated internalizing symptoms as young as 2–3 years old (Hussong et al. 2007). COAs then continue to show elevations relative to their peers that continue through adolescence and the alcohol-related symptoms of parents do little to mitigate or exacerbate this risk within this period of development. Further understanding the role of this problematic trajectory for internalizing symptoms within the larger array of negative outcomes for which COAs show elevated risk is needed to better understand the multiple ways in which parent alcoholism may impact children's functioning over development.

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