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Testing Whether and When Parent Alcoholism Uniquely Affects Various Forms of Adolescent Substance Use

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Abstract

The current study examined the distal, proximal, and time-varying effects of parents' alcohol-related consequences on adolescents' substance use. Previous studies show that having a parent with a lifetime diagnosis of alcoholism is a clear risk factor for adolescents' own substance use. Less clear is whether the timing of a parent's alcohol-related consequences differentially predicts the adolescent's own substance involvement. Using a multilevel modeling approach, we tested whether adolescents showed elevated rates of alcohol, heavy alcohol, marijuana and other illegal drug use (a) at the same time that parents showed alcohol-related consequences (time-varying effects), (b) if parents showed greater alcohol-related consequences during the child's adolescence (proximal effects), and (c) if parents had a lifetime diagnosis of alcoholism that predated the child's adolescence (distal effects). We tested these effects in a high-risk sample of 451 adolescents assessed over three waves beginning at ages 11–15 from 1988 to 1991 (53 % male, 71 % non-Hispanic Caucasian, 54 % children of alcoholic parents and 46 % matched controls). Strong and consistent distal effects of parent alcoholism on adolescent's substance use were found, though no additional risk was associated with proximal effects. Limited time-varying effects were also found. The importance of differentiating the timing effects of parent alcoholism in identifying underlying mechanisms of risk for adolescent substance use is discussed.

Keywords

Parent alcoholism; Substance use; Intergenerational transmission; Time-varying effects

A consistent tension in developmental theory lies in evaluating the relative impact of early experiences in childhood and those that occur later in ontogeny as formative to the development of psychopathology (Cairns and Cairns 2006). Sometimes framed as the effects of 'timing' (i.e., Cicchetti 2006), a fundamental question is whether the same risk factor has

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a different impact on children's adjustment depending on when in development it occurs. For example, studies of pubertal development suggest that an earlier timing of pubertal onset may increase risk for substance use in girls (at least within certain contexts, Dick et al. 2001; Stattin and Kerr 2011), though a later onset appears unrelated (Ge et al. 2006). Studies of children of divorce also indicate that timing may be important, with parent separation and divorce occurring before adolescence (earlier in time, Lansford et al. 2006 or in mid-childhood versus adolescence, Palosaari and Aro 1994) generally being associated with more negative later outcomes.

Similarly, the question of the developmental timing of risk onset is also emerging in the study of substance use and disorder (Zucker et al. 2008). Observing associations between risk indicators such as externalizing and internalizing symptoms in early childhood and substance use in adolescence, Zucker (2006) theorized that a key to understanding the development of these problems is in studying children earlier in the life course when such risk forms. In contrast, much of the study of substance use and disorder focuses on risk processes assessed within adolescence and adulthood as proximal influences on the development of these behaviors. Importantly, the potential mechanisms underlying risk for substance use and disorder that are associated with more distal influences on adolescent substance use and those that are associated with more proximal influences differ. For example, contributions associated with early stress exposure (i.e., maltreatment, parental separation) and certain forms of genetic liability may exert distal influences over development on child's adjustment (Hussong et al. under review; Gunnar and Quevedo 2007) whereas peer selection and socialization influences may be more proximal influences on children's adjustment (Dishion et al. 1994). Thus distinguishing between distal and proximal influences on children's adjustment has implications for understanding etiology and, as a result, mechanisms to target in empirically-informed prevention and treatment efforts.

In the current paper, we focus on this question of timing for a robust risk factor for adolescent substance use, namely parent alcohol-related consequences and disorder (Chassin and Ritter 2001). Moving beyond the question of whether parent alcohol-related consequences increase risk for adolescent substance involvement, we test whether the effects of these consequences differ when they are distal to adolescence versus proximal. Moreover, we test whether parent alcohol-related consequences that occur during their offspring's adolescence identify who is at risk for substance use (between-person proximal effects) as well as when a given adolescent might use substances (within-person 'time-varying' effects). This work extends a line of inquiry regarding different ways in which parent alcoholism may impact adolescent functioning more generally. In this series of papers (Hussong et al. 2008; Hussong et al. 2010), we distinguish among three different effects of parent alcoholism on adolescents' functioning. We refer to these effects as the distal, proximal, and time-varying effects of parents' alcohol-related consequences.

Parent Alcoholism and Alcohol-Related Consequences

Although findings regarding the effects of parents' alcohol use on adolescent's substance use are mixed, results consistently show that adolescents whose parents abuse alcohol are at greater risk for substance use and later disorder compared to their peers (Chassin et al. 1999; Sher 1991). In fact, children of alcoholic parents initiate drinking earlier, escalate in their use faster, and more quickly transition from initiation to alcohol use disorder than do children of nonalcoholic parents (Chassin et al. 1996; Hussong et al. 2008). Estimates from a community-based study indicate that by early adulthood just over half (53 %) of children of alcoholic parents evidence an alcohol use disorder and 21 % meet criteria for a drug use disorder, in comparison to 25 % and 9 %, respectively, of matched controls (Chassin et al.

1999). Moreover, after controlling for effects due to co-occurring parent psychopathology, environmental stress and family disruption, parent alcoholism is a unique risk factor for substance use in adolescence as well as substance use disorder in young adulthood (Chassin et al. 1991; Chassin et al. 1999). These results simultaneously demonstrate that parent alcoholism is a significant risk factor for alcohol and drug use disorders in offspring and that there remains significant heterogeneity in the outcomes of children of alcoholic parents, with nearly half not evidencing an alcohol or substance use disorder by young adulthood.

A multitude of mechanisms appear to account for this risk (Chassin and Ritter 2001), with each offering partial explanations for why COAs show greater substance use than their peers. Indeed, parent alcoholism has been described as a distal indicator for a host of negative risk factors (e.g., family conflict and disruption, maltreatment history) that may mediate the effects of parent alcoholism on adolescent outcomes. The mechanisms for which parent alcoholism and alcohol-related consequences may serve as a marker may in part depend on the timing of these parental behaviors within children's lives. The emphasis on how the timing of parent alcoholism impacts adolescent's substance use is a unique contribution of the current study.

To evaluate this question, we distinguish among three types of effects of parent alcoholism and alcohol-related consequences. The first is a within-subjects or time-varying effect that indexes whether children show increased (or decreased) functioning, over their usual baseline, at those times when their parents also show increased (or decreased) alcohol-related consequences. The second effect 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 dysfunction during that same period compared to children whose parents do not have alcohol-related consequences during this period. Thus, time-varying effects focus on the specific timing of effects (whether children's functioning gets 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 under study helps us to identify those children showing the greatest amount of dysfunction during this time). Finally, the third effect of parent alcoholism is a baseline and (relatively) distal effect. 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.

These three effects are conceptually distinct. For example, for a given child followed over ages 11 to 13, the time-varying effect is the within-person elevations and reductions in functioning associated with parents' alcohol-related symptoms at each specific age; the proximal effect is the between-person differences in functioning associated with parents' average level of alcohol-related symptoms between ages 11 and 13; and the distal effect is the between-person differences in functioning associated with parents' lifetime alcohol use disorder status assessed at baseline (in this example, by age 11 of the child). Moreover, the distal, proximal and time-varying effects of parent alcoholism have potentially unique information to provide regarding developmental 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. The proximal effects of parent alcoholism indicate that factors identifiable close in time to the child's assessment identify who is at risk for dysfunction whereas time-varying effects indicate that risk factors are also present to identify when dysfunction is most likely to occur.

Between-person comparisons for studying the effects of parent alcoholism on adolescents' substance use dominate the literature and contribute to our understanding of variability in risk for adolescent substance use. For example, findings drawn from the same sample as that analyzed in the current study show that children had higher risk for alcohol use if their fathers had continuous rather than remitted alcohol-related consequences (Chassin and Barrera 1993; Chassin et al. 1991). Moreover, 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 parents' alcohol dependence over time. Rarely studied, however, are the within-person or time-varying effects of parent alcoholism on adolescents' substance use. These time-varying effects answer the question of when (i.e., at what point in time) risk occurs for substance use, with the hypothesized effect being that youth will show greater risk than usual when their parents are reporting greater alcohol-related consequences.

Hops et al. (1996) studied the time-varying effects of parents' substance use on adolescents' alcohol, cigarette and marijuana use, taking into account differences in parent and adolescent gender in a community sample of 763 families. They reported that mother's and father's marijuana use uniquely predicted adolescent's own marijuana use. Effects for parents' use of alcohol and cigarettes on adolescents' own substance use were also found, depending on the age and gender of the adolescent. Thus, these findings indicate that when parents' increase their use of these substances, adolescents show increased risk for their own substance use. However, the analytic model used in these analyses did not take into account individual differences in rates of change in substance use over time (i.e., random effects in change or initial levels of substance use), meaning that increases in the child's substance use were relative to the group of children studied in general rather than to that child's own substance use over time. These models also did not control for the effects of between-effect predictors (i.e., distal and proximal effects) in evaluating the time-varying effects of parents' substance use on adolescents' substance use. Given that these effects are naturally highly correlated, it is important to determine whether time-varying effects add uniquely to our understanding of when adolescents are at risk for substance use above and beyond simply knowing who is at risk as a function of distal and proximal effects. Finally, the community sample used by Hops et al. did not have a high-risk sampling design which yields higher base rates of parental alcohol use disorder, a more salient risk indicator for adolescent alcohol use than rates of parental alcohol use as measured by Hops et al.

Variants of the Timing Effect

We anticipate that the effects of timing of parents' alcoholism and alcohol-related consequences on adolescents' substance use may vary as a function of three factors: which parent is showing consequences (mother or father), the gender of the adolescent, and the type of substance outcome examined. We predict that the impact of maternal alcoholism may be greater than that of paternal alcoholism. Given the impact of assortative mating (Maes et al., 1998; particularly in COAs, Boye-Beaman, Leonard & Senchak, 1991) and the lower base rates of alcoholism in women (Grant, Dawson, Stinson, Chou, Dufour & Pickering, 2004), a family with an alcoholic mother often also has an alcoholic father, so the impact of maternal alcoholism is often not practically distinguishable from that of having two alcoholic parents. As such, families with alcoholic mothers may convey significantly greater risk because the primary caretaker for the child is more likely to be affected, the familial stress load and dysfunction within the home is likely heightened (Chassin et al. 1991; Hussong & Chassin, 2004), and, if indeed both parents are affected or a second caretaker is not present, the child lacks the potential protective influence of a non-affected

parent (Werner, 1986; though this influence is not always supported, Curran & Chassin, 1996).

Studies of gender differences in risk for alcohol-related outcomes associated with parental alcoholism also indicate that daughters may be more vulnerable than sons (Russell, Cooper, & Frone, 1990; Sher 1991). However, converging studies suggest that boys may be more sensitive to the effects of other family-related stress than are girls. Studies of divorce, family conflict, maternal depression and non-responsive caregiving show greater negative effects of these family stressors on externalizing symptoms in boys than in girls (Dadds, Atkinson, Turner, Blums & Lendich, 1999; Essex, Klein, Cho & Kraemer, 2003; Malone et al., 2004; Martin, Maccoby, & Jacklin, 1981; Shaw, Keenan, & Vondra, 1994; Shaw et al., 1998). The extent to which this sensitivity to family-related stress also results in greater substance use in male versus adolescent female COAs is unclear.

Finally, stage theories indicate that different forms of substance use may be sequenced to indicate differences in severity (Kandel and Yamaguchi 1999). Although the sequencing of substances varies over study and perhaps historical time, alcohol is typically a gateway experience for other forms of drug use, with marijuana often preceding the use of other forms of illicit drug use. To the extent that alcohol use is developmentally typical, we would expect to see parent alcoholism predict adolescent illicit drug use and marijuana use more strongly than adolescent alcohol use. By comparing the effects of the timing of parent alcohol-related consequences across these three substances in the current study, we test this hypothesis.

The Current Study

In the current study, we test the hypothesis that parent alcoholism exerts risk for adolescents' substance use via distal, proximal and time-varying effects. We examine this hypothesis over four indicators of adolescents' substance use, namely alcohol use, heavy alcohol use, marijuana use, and other illegal drug use. Moreover, we focus on a high-risk, community recruited sample of children of alcoholic parents and matched controls. This study extends a line of inquiry regarding how the timing of parent alcoholism impacts adolescent functioning broadly conceived to include internalizing symptoms, externalizing symptoms, and now substance use.

Method

Sample and Procedure

In the Adolescent/Adult Family Development Project (AFDP; Chassin et al. 1991), 454 adolescents and their parents completed three annual, computerized, in-home beginning in 1988. Of these, 246 included a biological and custodial alcoholic parent whereas 208 were matched controls. Children of alcoholic (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 (using the property value code from the reverse directory; Chassin et al. 1992). As we did with COA families, we directly interviewed parents (or their spouses when parents were not available) in control families to confirm that neither biological nor custodial parents met criteria for a lifetime alcoholism diagnosis. Recruitment biases have been found to be minimal (Chassin et al. 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. After dropping three cases missing relevant data, the remaining 451 adolescents we included for the analysis provided 1,317 observations on up to three different time points. The age range of these measurements is from 11 to 17 with a mean of 13.82 and standard deviation of 1.59. Among these adolescents, 53 % are boys, 71 % are non-Hispanic Caucasians and 8 % of their parents received a high school education or less (see 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 alcohol-related consequences and diagnosis was assessed by parent-report. Parents were directly interviewed at baseline using a computerized version of the Diagnostic Interview Schedule (Robins et al. 1981, 1982) to assess diagnostic status. In cases where a biological parent was not 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 et al. 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. This lifetime diagnosis formed the distal risk indicator for parent alcoholism.

Both proximal and time-varying indicators of parent alcoholism were based on parents' self-reports at each wave of whether they had experienced each of 11 alcohol-related DSM-III symptoms in the past year. The symptoms are consistent with 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. 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; Curran and Bauer 2011). The time-vary covariate (TVC) was

assessed for each parent at each age of the child; this captured the within-person effect (after person-mean centering). The age-specific means of the TVCs for mothers ranged from 0.14 to 0.31 (SDs ranging from 0.62 to 1.0) and for fathers ranged from 0.44 to 0.91 (SDs ranging from 0.90 to 1.61). The grand mean of the set of TVCs was computed for each parent; this captured the between-person effect. The mother-specific grand mean of the set of TVCs was equal to 0.27 (*sd*=0.81; range 0–7.33), with 74 % of mothers reporting no consequences at any wave and between 6 and 13 % of mothers reporting consequences within any given age period. The father-specific grand mean of the set of TVCs was equal to .77 (*sd*=1.14; range 0–6.75), with 44 % of fathers reporting no consequences at any wave and between 30 and 35 % of fathers reporting consequences within any given age period.

We created four variables indexing *adolescent-reported alcohol, heavy alcohol, marijuana and other drug use*. These variables were based on six items assessing the use of beer or wine, the use of hard liquor, drinking 5 or more in a row, drunkenness, marijuana use and other illegal drug use within the past year using an 8-point response scale ranging from never to daily. As expected for this age range, responses for all items were highly skewed and we collapsed across response options to take into account sparseness in the upper response options. To index alcohol use, we created a four-level ordinal response variable (i.e., never, annually, monthly, and at least weekly) indexing the maximum frequency reported on two alcohol use variables (i.e., use of beer/wine and hard liquor; see Table 2). The range of this variable was from 0 to 3, with 9.4 % endorsing at least some use at age 11 and 80 % doing so at age 17 and rates across waves of 34, 39 and 48 %. To index heavy alcohol use, we similarly categorized responses on the maximum frequency reported on two items (i.e., drinking 5 or more in a row and drunkenness) using the same four-level ordinal response scale. The range of this variable was from 0 to 3, with 3 % endorsing at least some heavy alcohol use at age 11 and 51 % doing so at age 17 and rates across waves of 15, 19 and 25 %. Given highly skewed responses, we dichotomized responses (i.e., no use versus use) to items assessing marijuana use (a single item) and other illegal drug use (7 items assessing amphetamines, barbiturates, tranquilizers, cocaine, opiates, inhalants and hallucinogens which we combined due to lower base rates of these individual substances) to form these last two indicators of adolescent substance use. Percentages for marijuana and other illegal drug use ranged from 0.93 % and 1.87 % at age 11 endorsing use to 20.34 % and 13.56 % doing so at age 17, respectively. Marijuana use rates were 7, 10 and 11 % across waves and other illegal drug use rates were 6, 10 and 7 % across waves

Results

Our analytic approach proceeded in three phases following a similar strategy as in our prior studies (Hussong et al. 2008; Hussong et al. 2010). First, we addressed the issue of missing data, both by design and attrition. Second, we estimated longitudinal trajectories of substance use. Finally, we tested our specific study hypotheses. In all cases, the longitudinal ordering of the data was based on adolescent age rather than on wave of assessment, capitalizing on our cohort sequential design.

Phase 1: Imputing Missing Data

We addressed the issue of missing data in our time-invariant and time-varying covariates through multiple imputation (Schafer 1997a). 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. Cases (n=451) with missing data for the time-invariant covariates were about 0.2 % and observations (1,317) with missing time-varying predictors were 26 % due to missing parent

reports. The primary source of missing time-covariate data was a parent refusal from a participating family. 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.

Phase 2: Constructing Trajectories

We then identified the shape of substance use trajectories for each of our four outcomes: alcohol use, heavy alcohol use, marijuana use, and other illegal drug use. (See Chassin et al. 1996 for previous trajectory analyses of these data.) First, we plotted the log odds for each outcome across age and then examined iterative analyses using data from the multiple imputations (above) to examine the optimal functional form of the resulting trajectories (i.e., linear, quadratic and piecewise liner) following the strategies described by Bollen and Curran (2006). Intercepts for the trajectories were centered at age 14 for all outcomes. We used Mplus to fit linear, piecewise and quadratic growth models to each outcome separately, using non-linear link functions to account for the binary and trichotomy outcomes. 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; for complete results contact author). Based on these criteria, the optimal functional form for indicators of both alcohol and heavy alcohol use were quadratic in form such that drinking increased more rapidly early in adolescence before stabilizing in later adolescence (see Fig. 1). Both marijuana and other illegal drug use also showed this quadratic pattern, but for marijuana use the stabilizing effect was less evident as captured by an underlying significant linear effect of age as well (see Fig. 2).

Phase 3: Hypothesis Testing

To test our hypotheses, we estimated a series of conditional multi-level or random coefficients 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 and proximal effects, we followed Raudenbush and Bryk (2002, p. 134–141; see also Curran and Bauer 2011). Specifically, we person-mean centered (i.e., subtracted each repeated assessment for an individual from the average of those repeated assessments for that individual) the variables indexing mothers' and fathers' alcohol-related symptoms prior to including them in the multi-level model as repeated measures and the report of these symptoms averaged over repeated assessments as the proximal effect.

In baseline models, we first included control variables (i.e., child gender, ethnicity and parent education) and interactions between control variables and the age-indicated time trends. This was a conservative strategy and we thus reduced non-significant interactions for subsequent analyses. Only one such significant interaction was found which reflected gender differences in the linear trend underlying heavy alcohol use, with boys showing steeper increases in heavy drinking after age 14 than girls.

As reported in Table 3, we then added the three types of effects (i.e., distal, proximal, and time-varying) for each parent's alcohol-related symptoms. As in our previous studies, having a parent with a baseline diagnosis of alcoholism was associated with strong and consistent increased risk for substance use in adolescents. In all but one instance, this increased risk of baseline or distal effects of parent alcoholism were present and independent for both mothers and fathers reported alcoholism.1 No proximal effects of parent alcoholism were found to identify which adolescents were more likely to engage in substance use. However, time-varying elevations in mother's alcohol-related drinking consequences served to decrease the risk of children's coinciding alcohol use (β=−0.38, t=−2.42; this effect was

clarified by moderating effects tested subsequently, see below) and father's alcohol-related drinking consequences served to increase risk at the level of marginal significance (β=0.18, *t*=1.86).

We next tested whether gender differences may obscure significant time-varying effects of parent alcoholism on adolescent's substance use. We tested this hypothesis by adding to these models the interaction between adolescent gender and the mother and father time-varying indicators. Out of these eight possible interactions across substance use, two were significant and showed moderating effects of gender on the effect of mother's alcohol-related symptoms on adolescents' alcohol use and on the effect of father's alcohol-related symptoms on adolescent's other illegal drug use. Plotting of these interactions revealed that the effect of father's alcohol-related symptoms on adolescent's illegal drug use was non-significant for girls (*b*=0.25, *t*=1.26, p = ns) but significant and negative for boys (*b*=−0.43, *t*=−2.07, *p*=0.04). A similar pattern was found for the effects of mother's alcohol-related consequences on adolescents' alcohol use, though neither simple slope for boys or girls reached levels of statistical significance. However, due to the inconsistent and limited findings from these repeated tests, we did not consider their interpretation further.

In a final set of analyses, we added to the models reported in Table 3 the interaction between the time-varying and distal effects of parents' alcohol-related consequences and disorder. In these analyses, we tested whether increases in parents' alcohol-related consequences differentially impacted the timing of adolescent's substance use depending on whether the parent had at one time met lifetime criteria for an alcohol use disorder. Two marginally significant interactions emerged out of eight tested. We probed these interactions following Bauer and Curran (2005). Simple slopes showed that father's alcohol-related drinking consequences showed a stronger effect on the timing of adolescent drinking behavior when the father did not have a lifetime alcohol diagnosis (*b*=0.61, *t*=2.35, *p*<0.05) versus when he did (*b*=0.11; *t*=1.10, p = ns). Similarly, mother's alcohol-related drinking consequences only predicted concurrent elevations in adolescent's marijuana use when the mother had no lifetime alcohol diagnosis (*b*=1.02, *t*=1.96, *p*=0.05) versus when she did (*b*=−0.04, *t*=−0.16, p = ns). Again, due to the inconsistent and limited findings from these repeated tests, we do not consider their interpretation further.

Discussion

Our findings do support differences in the timing of parent alcoholism and alcohol-related consequences on adolescent's substance use. As with previous studies, our findings indicate that having a parent with a lifetime alcohol use disorder is a strong and consistent risk factor for adolescents' alcohol, marijuana and other illicit drug use (Chassin et al. 1991; 1999; Sher 1991). After controlling for this dominant distal effect of parent alcoholism, we found no additional risk associated with the proximal effects of parent alcoholism. However, we did find some support for the time-varying effects of parent alcohol-related symptoms. These time-varying effects indicated that adolescents' risk for substance use decreases when their mothers report greater alcohol-related consequences themselves. Although these time-varying effects of parent alcoholism were weaker and more inconsistent than the distal effects, they nonetheless were unique above and beyond other indicators of parent alcoholism. The overall lack of interaction effects suggested that the pattern of effects varied little over gender of the adolescent and parent. However, time-varying effects were apparent for alcohol use and not for other substance use indicators. These findings have implications

[1]In sensitivity analyses, we included indicators of mothers' and father's depression and antisocial personality disorders. Substantive interpretations of the results remained unchanged, with one exception. The distal effects of mother's alcoholism diagnosis became non-significant in predicting heavy alcohol use and other illicit drug use, though they remained unique predictors of marijuana use.

for furthering our understanding of sources of heterogeneity in functioning and developmental outcomes among children of alcoholic parents, for identifying mechanisms that impact the trajectories of adolescents' substance use, and for identifying who and when risk for substance use emerges so as to inform prevention efforts.

Timing and Mechanisms

The current study completes a series of investigations examining whether the timing of parent alcoholism and alcohol-related consequences impacts adolescent functioning. Across these studies we found a clear pattern in which the distal effects of parent alcoholism were stronger and more consistent than were proximal and time-varying effects on adolescents' internalizing symptoms, externalizing symptoms, and substance use. The dominance of this distal effect is consistent with broad-based, developmentally cumulating risk mechanisms that predict impairment in children's functioning. These include some models of genetic vulnerability, such as that shown to account for the co-occurrence of antisocial behaviors, conduct problems, alcohol use and drug use in behavioral genetics studies of adults (Kendler et al. 2003). Moreover, these dominant distal effects are also consistent with proposed early emerging pathways of risk that link risk processes underlying externalizing or internalizing symptoms in childhood to later substance use in adolescence and beyond (e.g., Hussong et al. 2011; Zucker et al. 2011). In each of these examples, risk for substance use and symptomatology is already present in youth before they enter adolescence, and the impact of these early risk mechanism may be little deterred by the pattern of parents' alcohol-related consequences, in the absence of intervention and significant protective factors, by what happens in adolescence itself.

In previous studies, we have found some support for proximal effects indicating an additional risk for externalizing and internalizing symptoms (above and beyond that attributed to distal effects) for children whose parents had greater alcohol-related consequences during the child's adolescence (Hussong et al. 2008; 2010). For internalizing symptoms, this proximal risk was more limited and conveyed only via mothers' alcohol-related consequences on adolescents' self-reported symptoms. For externalizing symptoms, the proximal risk was more consistently identified and was present across parents (i.e., fathers' alcohol-related consequences consistently predicted parent-reported symptoms in children and mother's alcohol-related consequences predicted mother- and adolescent-reported symptoms in children). These proximal effects are consistent with the interpretation that youth with active alcoholic parents face an additive risk for these symptoms and are at greater risk for such functioning deficits than are their peers whose parents' alcoholism is remitted (Chassin et al. 1991). However, we did not find such proximal effects in predicting adolescents' substance use in the current study. This discrepancy may in part be due to the larger effect of father's lifetime alcoholism diagnosis on adolescents' substance use versus other forms of symptomatology. Previous studies show that a parent's lifetime alcoholism diagnosis is a specific risk factor for substance use in adolescents above and beyond co-occurring parent psychopathology, environmental stress and family disruption (Chassin et al. 1991). Perhaps the specificity of mechanisms associated with the distal effects of parent alcoholism dilute the potential additive risk associated with proximal effects in the prediction of substance use versus children's symptomatology.

In contrast to findings concerning proximal effects, the time-varying effects of parent alcoholism were found to predict both substance use in the current study and other forms of symptomatology in our previous work. The present findings showed that adolescents decreased their own heavy alcohol use during those years when their mothers reported more alcohol-related consequences. To account for the unique effect of time-varying risk associated with parents' alcohol-related consequences, risk mechanisms must be dynamic

with short-term impact. For example, adolescents may bear more responsibility for care of younger siblings or self-care during these years or other care takers may play a bigger role in childcare and provide more stability and compensatory support, each of which may result in temporary resources or demands on teens that create a transitory decrease in risk for substance use. Further work is needed to determine whether the effects of parent alcoholism are consistent with the time-varying effects of parent alcohol-related consequences specifically or with more distal effects of parent alcohol-related consequences that accumulate over time. Moreover, future studies should consider whether the effects of proximal and time-varying processes are more evident in general population, rather than high-risk, samples where alcohol use disorders are less prevalent.

Both mother's and father's alcohol-related behaviors predicted adolescent outcomes across our previous studies, with minor variations in pattern. This was of course most clear for the distal effects of parent alcoholism. Although previous studies show a more consistent pattern of proximal and distal effects of fathers', than mothers', alcohol-related consequences on adolescents' externalizing symptoms, no clear differences between mothers' and father's alcohol-related consequences emerged in analyses of internalizing symptoms or various forms of substance use perhaps due to the isolated findings for each. These findings reinforce the importance of both parents' functioning for adolescent adjustment. However, they do not consider more nuanced differences in parenting roles and the impact on family functioning that may be important for understanding how mother's and father's uniquely influence children's adjustment. Previous work shows that having two alcoholic parents is more impairing for youth than having one and that comorbid psychopathology in alcoholic parents can also increase risk for poor youth outcomes, including accelerated substance use (Hussong et al. 2008). Given the complexity of family constellations, particularly for families with an alcoholic parent, the question of contrasting mother's and father's influences on children's outcomes may need to be more broadly cast within this question of overall impairment across caregivers. Future work is needed to unravel this complex issue.

In Conclusion

In sum, the current study found strong support for distal and limited support for time-varying effects of parent alcoholism on adolescents' substance use. This was the third in a series of studies indicating that the impact of parent alcoholism and alcohol-related consequences on adolescent outcomes depends on the timing of parents' impairment. Strengths of the current study include use of a high-risk, community based sample of children of alcoholic parents and matched controls, direct ascertainment of parent alcoholism, repeated assessments over adolescence, and use of analytic models to parse between- and within-person effects. These strengths lend credence to our conclusions, though limitations should also be considered including the lack of temporal precedence in the time-varying predictors and large confidence intervals for some of the estimated parameters for naturally small sub-groups (e.g., young children using marijuana with an alcoholic mother; see Table 1), the limited ethnic variation in the sample, the skewed indicators of parents' alcohol-related symptoms which may create challenges in parsing effects, potential differences in the severity of parental impairment associated with distal and proximal indicators of parent alcohol-related consequences and alcoholism, and the reliance on self-reports for parents' own and adolescents' own substance use. These limitations require for cross-validation of the findings.

Nonetheless, these results are consistent with the call for early prevention programming in which children of alcoholic parents are targeted early in life to prevent later, cumulating risk for internalizing symptoms, externalizing symptoms and substance use (Masten et al. 2008). Moreover, parents' alcohol-related consequences may also signal periods of risk for

adolescents, but to a lesser extent. Future research concerning the mechanisms underlying these time-varying effects of parent alcoholism are needed to better understand the best ways to support these families and prevent risk for externalizing symptoms and substance use in these youth.

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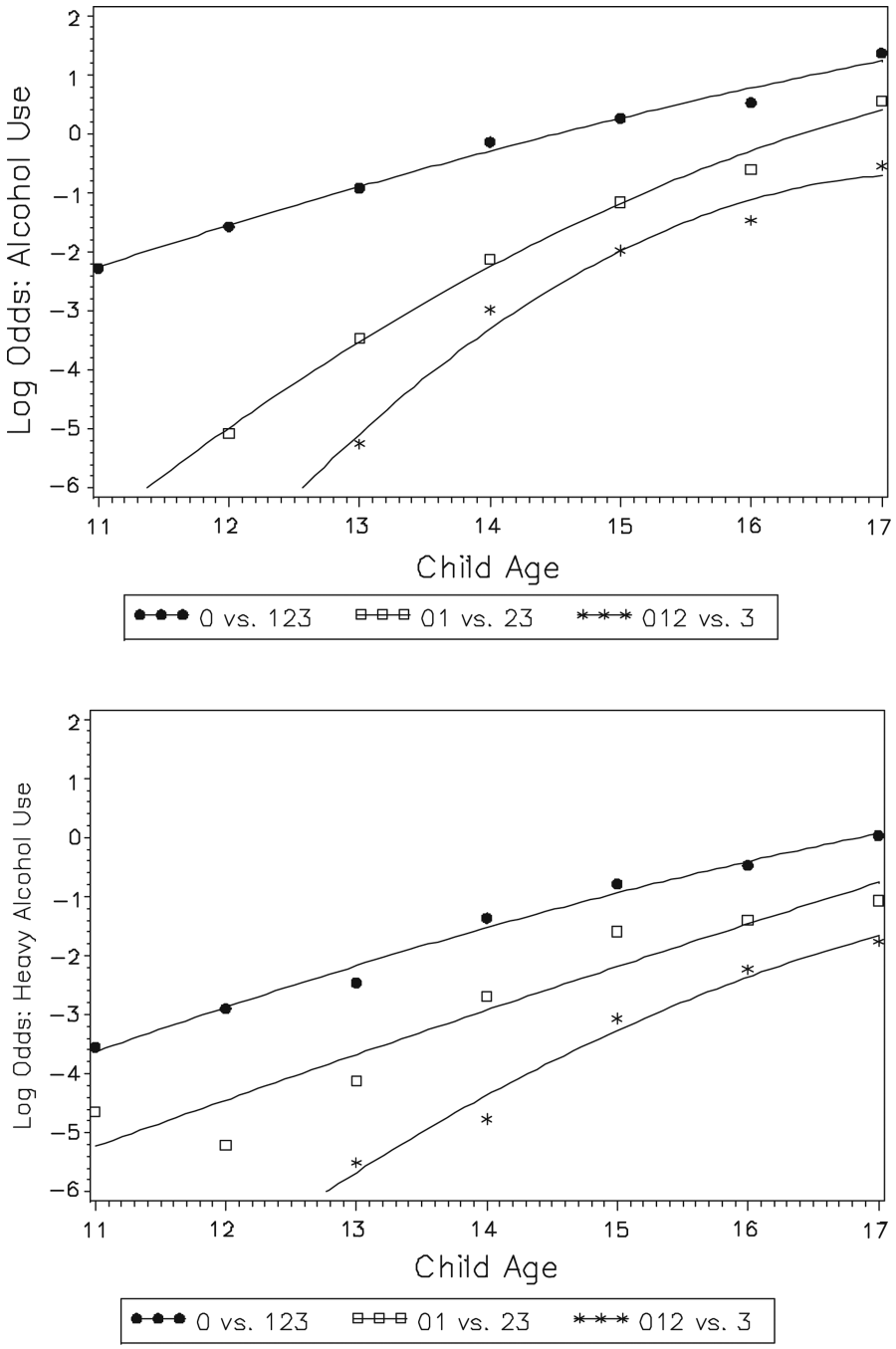

Fig. 1.
Sample cumulative log-odds for alcohol use and heavy alcohol use

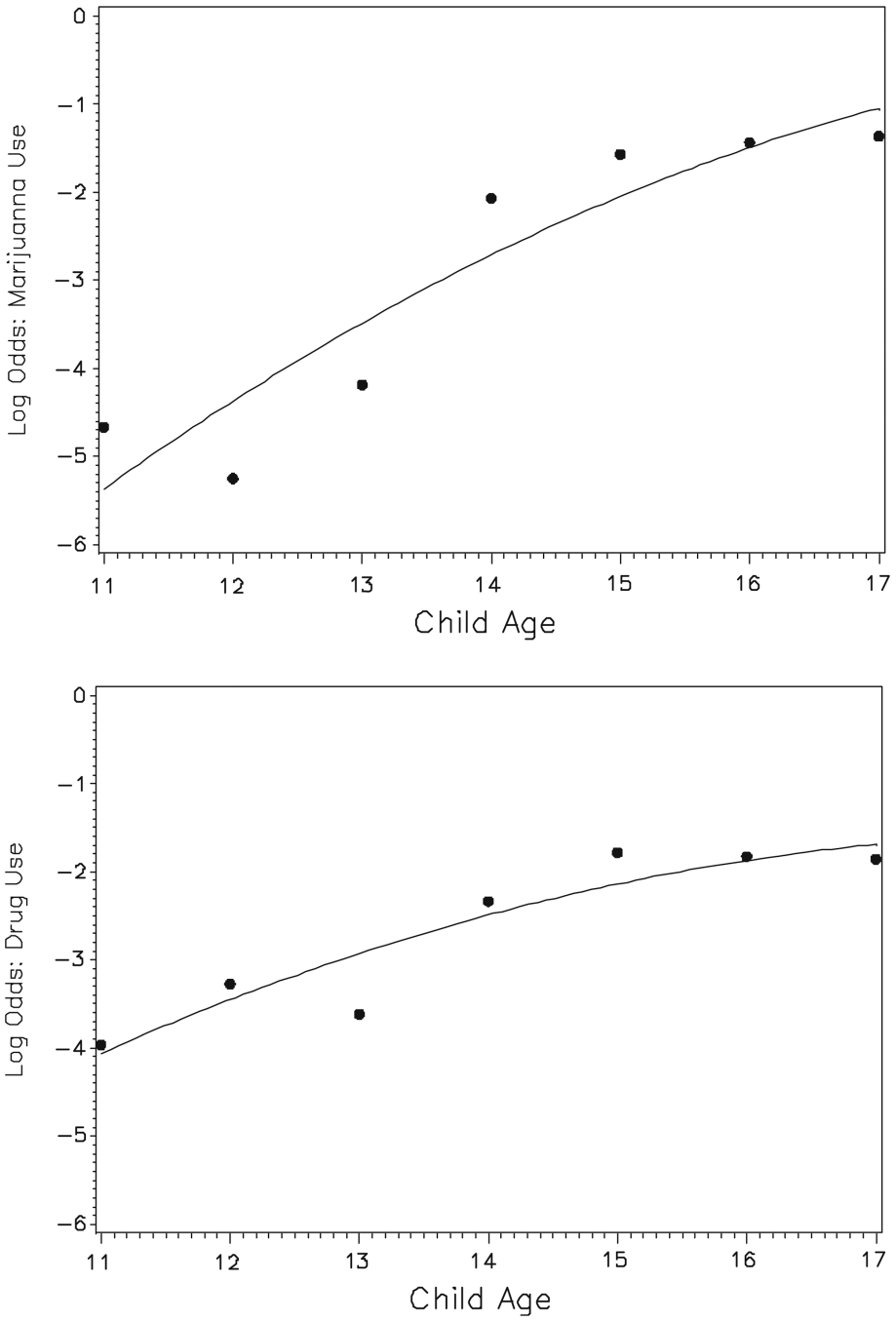

Fig. 2.
Sample log-odds for marijuana and other illegal drug use

Table 1

Sample descriptive statistics

Characteristic	Representation
% Male	53
% Hispanic	29
Parent Education	–
% with high school education or less	27.11
% college graduate	16.86
% COA	54
% with an alcoholic mother	13
% with an alcoholic father	47
Average Age (SD; range)	13.82 (1.59; range 11–17)

Table 2

Percent of adolescents using substances by age

	Age						
	11	**12**	**13**	**14**	**15**	**16**	**17**
Alcohol Use	9.35	17.28	28.57	46.6	56.63	62.91	79.66
Heavy Alcohol Use	2.8	5.24	7.89	20.41	31.33	38.41	50.85
Marijuana Use	0.93	0.52	1.5	11.22	17.27	19.21	20.34
Other Illicit Drug Use	1.87	3.66	2.63	8.84	14.46	13.91	13.56

Table 3

Time-varying, proximal and distal effects of parent alcoholism on adolescent's substance use

	Alcohol use			Heavy alcohol use			Marijuana use			Other illegal drug use		
PREDICTORS	**Log Odds**	**t-value**	**OR (95 % CI)**	**Log Odds**	**t-value**	**OR (95 % CI)**	**Log Odds**	**t-value**	**OR (95 % CI)**	**Log Odds**	**t-value**	**OR (95 % CI)**
Control variables												
Intercept	−1.49	−2.90	0.23 (0.08, 0.62)	−3.40	−5.32	0.03 (0.01,0.12)	−6.48	−3.79	0 (0.00, 0.04)	−4.63	−4.52	0.01 (0.00, 0.07)
Linear Age 11–17	0.88	9.10	2.42 (2, 2.93)	0.59	3.31	1.8 (1.27,2.55)	1.96	2.20	7.06 (1.24, 40.34)	0.45	1.28	1.57 (0.79, 3.11)
Quadratic Age 11–17	−0.03	−.9	0.97 (0.9, 1.04)	−0.16	−2.06	0.85 (0.73,0.99)	−0.80	−2.91	0.45 (0.26, 0.77)	−0.48	−2.01	0.62 (0.39. 0.99)
Parent Education	0.05	0.40	1.05 (0.82, 1.36)	−0.09	−0.63	0.91 (0.68,1.22)	−0.20	−0.71	0.82 (0.46, 1.43)	0.04	0.18	1.04 (0.68, 1.59)
Child Gender	−0.17	−0.63	0.84 (0.5, 1.43)	0.40	1.27	1.49 0.8,2.78)	−0.41	−0.63	0.66 (0.18, 2.38)	−0.78	−1.79	0.46 (0.20, 1.08)
Child Ethnicity	0.38	1.25	1.45 (0.81, 2.62)	0.22	0.63	1.25 (0.63,2.48)	0.42	0.60	1.53 (0.38, 6.07)	0.52	1.06	1.68 (0.64, 4.40)
Child Gender × Linear Age 11–17	–	–		0.65	3.16	1.91 (1.28,2.85)	–	–		–	–	
Time-varying effects												
Mom alc con (TVC)	−0.04	−0.24	0.96 (0.66 1.38)	−0.38	−2.42	0.69 (0.5,0.93)	0.23	0.93	1.25 (0.78, 2.01)	0.24	0.83	1.27 (0.72, 2.24)
Dad alc con (TVC)	0.18	1.86	1.19 (0.99, 1.44)	−0.07	−0.73	0.93 (0.77,1.12)	−0.18	−0.59	0.84 (0.46, 1.51)	−0.04	−0.24	0.97 (0.73, 1.29)
Between-person proximal effects												
MAC	0.20	1.16	1.22 (0.87, 1.71)	0.23	1.31	1.26 (0.89,1.78)	0.42	1.28	1.52 (0.80, 2.89)	0.06	0.26	1.06 (0.67, 1.68)
DAC	0.13	1.08	1.14 (0.9, 1.44)	−0.01	−0.08	0.99 (0.76,1.29)	0.30	0.95	1.35 (0.72, 2.53)	0.00	0.00	1 (0.65, 1.54)
Between-person distal effects												
Mom alc diagnosis	0.55	1.11	1.73 (0.65, 4.57)	1.14	2.17	6.49 (3.26,12.95)	2.63	2.02	13.93 (1.09, 178.39)	1.63	2.30	5.11 (1.28, 20.46)
Dad alc diagnosis	1.38	4.51	3.96 (2.18, 7.21)	1.87	5.32	3.13 (1.12,8.75)	1.98	2.48	7.22 (1.51, 34.50)	1.11	2.31	3.02 (1.18, 7.72)