The Relationships of Parental Alcohol Versus Tobacco and Marijuana Use With Early Adolescent Onset of Alcohol Use

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ABSTRACT. Objective: This study examined whether the use of tobacco and marijuana by fathers or mothers predicted onset of alcohol use in their offspring over and above effects of parental alcohol use. **Method:** The present study included 146 children of 93 parents (90 fathers and 85 mothers). The fathers were originally recruited as boys to the Oregon Youth Study, a study of community, familial, and individual risk factors for delinquency. **Results:** Only mothers' but not fathers' alcohol use was associated with children's age at onset. Children's age at onset was predicted by mothers' tobacco use and by the interaction of fathers' marijuana use and alcohol use. These effects were observed when controlling for parental education, child's gender, and also child's antisocial behavior—a general developmental risk factor for substance use onset in adolescence. **Conclusions:** Mothers' substance use played a major role in childhood onset of alcohol use, yet the role of maternal substance use as a risk factor for their children has previously received less attention than the role of paternal substance use. Also, findings imply that it may be important to identify children of polysubstance-using parents for targeted prevention programs. (*J. Stud. Alcohol Drugs, 77,* 95–103, 2016)

STUDIES OF EFFECTS OF PARENTAL substance use on offspring alcohol use have focused on effects from parental alcohol use rather than also examining effects of use of other substances (Chassin et al., 2004; White et al., 2000). However, many adults who drink alcohol also use other substances, particularly tobacco and marijuana, and there are significant associations between alcohol use disorders and other substance use disorders (Stinson et al., 2006). Thus, there may be an additional risk for onset of alcohol use in offspring (which typically begins before marijuana and other illicit drugs; Kandel, 2002) from parental use of tobacco and marijuana. Understanding such risk is a crucial issue because early onset predicts later problematic use in adulthood (Pitkänen al., 2008; Warner et al., 2007). The present study examines whether use of tobacco and marijuana by fathers or mothers predicts onset of alcohol use in their offspring over and above effects of parental alcohol use. In addition, interaction effects of parental use of tobacco and marijuana, along with alcohol, on childhood onset of alcohol use were examined.

Age at onset of alcohol use is a risk factor for a later alcohol use disorder. Grant and Dawson (1997) found for the National Longitudinal Alcohol Epidemiologic Survey that rates of lifetime dependence were at more than 40% for those who started drinking at age 14 years or younger, and about 10% for those who started drinking at age 20 years or older. DeWit et al. (2000) found a rapid progression to alcohol-related harm among those having their first drink at ages 11–14 years; after 10 years, 16% of individuals who had a first drink at ages 11–12 years met a diagnosis of alcohol dependence, and 9% of those who initiated drinking at ages 13–14 years met such criteria, whereas only 1% of those who began drinking at age 19 years or higher met such criteria. However, a limitation of these studies and many studies of age at onset of drinking is that age at onset is reported retrospectively. For adolescents, it has been shown that retrospective reporting of age at onset of alcohol use is subject to forward telescoping whereby reported age at onset moves up to older ages as the adolescent ages, and retrospective reporting by adults may be subject to similar biases. In the present study, age at onset was assessed four times from ages 11–12 to 17–18 years for the children. Note that for the Oregon Youth Study (OYS) men themselves, who were assessed yearly across adolescence, we examined the association of age at first full alcoholic drink and later diagnosis of an alcohol use disorder (assessed by lifetime reports at ages 25–26 and 35–36 years) and found that men reaching diagnostic criteria were on average 1.5 years younger at age

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of first full drink (14.5 vs. 16.0), $t(186) = 3.22$, $p = < .01$, than men who did not reach such criteria (unpublished finding). Thus, an earlier age at onset of alcohol use is associated with problematic use later.

There are a number of reasons parental use of substances other than alcohol may predict earlier onset of a child's alcohol use. First, the mechanisms by which parents transmit substance use risk to children may be general, rather than substance specific. Familial alcoholism is associated with higher impulsivity and lower agreeableness in children (Chassin et al., 2004). Similarly, conduct problems in childhood are a risk factor for the early onset of alcohol, tobacco, and marijuana use (Dishion et al., 1999), and conduct problems or antisocial behavior are associated across generations (Capaldi et al., 2012). Regarding genetic influences, risk for substance use and dependence overlap considerably across drug classes (Kendler et al., 2012). Second, although parental modeling is usually examined in a substance-specific fashion (e.g., alcohol-to-alcohol; White et al., 2000), we posit that use of any psychoactive substance by the parent involves parental modeling of substance use and also may be viewed by the child as indicating the parents' tolerance for use of such substances by the child. Use of multiple substances by the parent (e.g., tobacco and alcohol) may make it even more likely that the parent is either tolerant of the child's use of substances or that the child ignores their verbal disapproval of early use. Third, parents' marijuana intoxication may interfere with their ability to effectively monitor children (Robertson et al., 2008) and prevent access and use of alcohol in and outside the home.

No study of which we are aware has examined the effects of parental use of substances other than alcohol on offspring alcohol use while controlling for parental alcohol use. Cox and Pritchard (2007) found that high school students who reported that their fathers smoked were more likely to use alcohol and engage in heavy episodic drinking than were other adolescents. However, they did not account for any effects of parental alcohol use. Li et al. (2002) examined associations of parental smoking, alcohol use, and marijuana use with adolescent alcohol use for early adolescents. They found that parental smoking, alcohol use, and marijuana use predicted adolescent alcohol use 18 months later. However, they did not examine these three risk factors in multivariate prediction models. Thus, little is known about the extent to which parental use of tobacco, alcohol, and marijuana may uniquely or interactively contribute to alcohol use onset in their children. Nonetheless, these studies do provide some indication that parental use of substances, other than alcohol, is associated with offspring alcohol use.

A few studies have examined whether associations from parents' substance use to children's use can be characterized as general risk rather than substance-specific risk. Hansen et al. (1987) defined latent constructs of both parental substance use (using indicators of smoking, alcohol use, and marijuana use) and a similar construct for the youth and found both general and substance-specific (for alcohol) intergenerational associations. However, the study was cross sectional and limited by a reliance on the youth to report their own and their parents' substance use. Similarly, Bailey et al. (2006) examined the association of a latent parental substance use variable (with indicators of heavy episodic drinking, tobacco use, and marijuana use) assessed at offspring ages 13–14 years, and a similar latent score of offspring substance use at ages 15–18 years, and the general latent scores were significantly associated. Additional substance-specific effects were found for only tobacco use. These studies indicate that intergenerational associations are not limited to substance-specific effects but did not address the issues of (a) whether parental use of marijuana and tobacco predict earlier onset of alcohol use during adolescence, when accounting for effects of parental alcohol use; (b) whether offspring alcohol use onset is more strongly predicted by parental marijuana, tobacco, or alcohol use; or (c) interactive effects of parental marijuana or tobacco use with parental alcohol use.

A further limitation of prior work is that few studies have involved assessment of both maternal and paternal substance use. In a sample of children of alcoholics and matched controls, Hussong et al. (2012) examined the association of maternal and paternal alcohol diagnoses with both frequency of alcohol use and frequency of heavy alcohol use. They found that the fathers' alcohol diagnosis was associated with both childhood variables, whereas the mothers' diagnosis was only related to heavy use. Thus, problematic levels of alcohol use by both mothers and fathers have been found to be predictive of alcohol use in adolescence by their children. In prior work on intergenerational associations in alcohol use with the current sample, Kerr and colleagues (2012) examined the association of maternal and paternal alcohol use with a construct score of children's alcohol use in early adolescence, controlling for the association expected via father-to-child transmission of externalizing behavior. Both paternal and maternal alcohol use were significantly and marginally associated with child's use, respectively, controlling for the significant effect of common pathway risk via intergenerational transmission of antisocial behavior. Thus, substance use by either or both parents may influence alcohol use by children. The present study builds on prior work in two main ways: first, by addressing risk for age at onset of alcohol use and second, by examining effects of both mothers' and fathers' use of marijuana and tobacco on such risk, in addition to effects of mothers' and fathers' alcohol use. We hypothesized that both maternal and paternal alcohol use would be associated with an earlier age at onset of alcohol use in offspring. In addition, we predicted that both parental tobacco use and marijuana use would make additional contributions to an early age at onset, whether via main effects or interactive effects with alcohol use. Lower parental education

and children's antisocial behavior were hypothesized to increase risk for earlier alcohol onset in children and thus were included as controls. We tested hypotheses using discrete-time survival analyses of data on a sample of parents and children assessed prospectively at ages 11–18 years.

Method

Participants

The fathers were originally recruited as boys to the OYS. Of all boys in the fourth-grade classes (ages 9–10 years) of schools in neighborhoods with higher-than-average rates of delinquency in a medium-sized metropolitan area in the Pacific Northwest, 74% (n = 206) agreed to participate; 90% were White, and 77% were from families of low socioeconomic status (Hollingshead, 1975). Of living participants, 89% or more participated at each of the yearly assessments from ages 9–10 to 40–42 years.

The ongoing Three Generational Study (3GS) recruited offspring of the OYS men and these children's mothers (who might still be in a romantic relationship with the father). Originally, all children and cohabitating stepchildren were allowed to participate. Because of budget limitations, recruitment was later limited to only the first two biological children per pairing of an OYS man with a woman (i.e., OYS men who fathered children with more than one woman could have more than two children followed in 3GS). The N available for each wave is determined by the ages of the maturing children. The present study included 146 children of 93 parents (90 fathers and 85 mothers). In early adolescence (ages 11–13 years), 40% of the children lived with both biological parents, 17% with one biological parent, 31% with the biological mother and a stepfather, 6% part time with each biological parent, and 6% with other guardians. Of these children in the present study (as of March 2014), 28.8% (n = 42) were old enough to participate at ages 17–18 years, 29.4% (n = 43) had reached ages 15–16 years, 28.8% (n = 42) had reached ages 13–14 years, and 13.0% (n = 19) had reached ages 11–12 years. Fifty-seven percent of the children in Generation 3 were girls. Parents identified the children as European American (n = 106), African American (n = 10), Asian American (n = 1), Native American (n = 12), Hispanic or Latino (n = 9), or biracial (n = 8).

Procedures

Annual assessments of the OYS boys/men included interviews and questionnaires, and mothers were assessed at the same time as their children. Parents participated until their sons were ages 17–18 years. Assessments of children in the 3GS similarly included parent and child questionnaires and interviews. Fathers' substance use data collected as part of the OYS were used in the present analyses if data were unavailable from the fathers' 3GS assessments and the assessments occurred within a year window of one another.

Measures

Time-varying scores of child's alcohol use onset were created for each of the discrete-time periods across children's adolescence, whereas all predictor and control variables were aggregated over time. For parents' substance use and children's antisocial behavior, scores were averaged across children's adolescence, whereas for parents' level of education, the maximum was calculated. All predictor variables were then standardized across children (i.e., mean of 0, variance of 1). Thus, inter-individual differences in the predictor and control variables (e.g., standardized differences in parents' average tobacco use during their children's adolescence) were used to explain differences in the rate with which children had experienced onset of alcohol use across adolescence.

Children's alcohol use onset. A variety of operational definitions have been used to define alcohol onset (Donovan, 2004). These range from sips or tastes (Donovan & Molina, 2008) to definitions including regular drinking (Donovan, 2004), depending on the study purpose. Whereas onset of sips is an important issue as predictive of later use (Donovan & Molina, 2011), a large proportion of relatively young children would be defined as having experienced onset by this criteria (35% of 8-year-olds and 48% of 10-year-olds; Donovan & Molina, 2008), and it may be a number of years before such children progress to a full drink. Thus, for the present study, use of a full drink was deemed a more appropriate definition of onset, indicating use beyond very minimal levels. This has been frequently used as an onset criterion in other studies (e.g., King & Chassin, 2007).

At each assessment, children were asked if they had ever consumed one whole alcoholic drink (yes or no) and, if so, at what age they had first done so. Alcohol use onset before the first assessment included in the present study (at ages 11–13 years) was determined by using children's reports of their age at first alcohol use from the first assessment; subsequently, new reports of having ever drunk a whole drink were used to define onset for each period. Thus, five binary variables were created denoting alcohol use onset response patterns for onset before age 11 years and onset during four periods of adolescence (ages 11–12, 13–14, 15–16, and 17–18 years). Responses were coded as "1" (onset) and "0" (no onset), respectively, at each time point. Once a child experienced onset of alcohol use, all of his or her scores at later assessments were set to missing values because, once children experienced onset (e.g., at ages 13–14 years), they are no longer at risk for alcohol use onset at all subsequent ages (e.g., ages 15–16 and 17–18 years). There was also missing data on alcohol use onset for those children who were right censored because of their age at their last assessment (e.g.,

a 16-year-old adolescent who had not experienced onset would have a response pattern of {0, 0, 0, missing, missing}, respectively, across the five discrete-time periods).

Parental substance use. Separately for tobacco, alcohol, and marijuana, mothers and fathers self-reported at each interview on their frequency and quantity of use on a typical occasion in the last year. Response scales for frequency of use were either continuous (i.e., open-ended items) or categorical. Categorical responses were recoded to reflect the midpoints of the categories, denoting frequency of use in the last year, capped at daily use (*once or twice* = 1.5, *once every 2–3 months* = 5, *once a month* = 12, *once every 2–3 weeks* = 20.8, *once a week* = 52, *2–3 times a week* = 130, *once a day and 2–3 times a day* = 365). Tobacco use included the aggregate use of cigarettes, pipes, and chewing tobacco (which were converted to mg doses of nicotine per week). Likewise, alcohol use included the aggregate use of beer, wine, and distilled spirits (denoted as the number of drinks on a typical occasion in the last year). Quantity of marijuana use was converted into grams as follows: one joint = 1 g, one toke or bong hit = 1/10 g, one ounce = 28 g. Separately for each type of substance, parental substance use scores were calculated as the product of frequency and quantity of use, log-transformed to reduce positive skew, and standardized over the children's assessments. Prevalence of tobacco, alcohol, and marijuana use during at least one point in their children's adolescence were respectively 43.3%, 82.8%, and 13.4% for mothers, and 62.0%, 89.1%, and 33.3% for fathers. Of the fathers and mothers who reported using alcohol during their children's adolescence, on average, 23.6% and 55.9% consumed less than 1 drink per week, 23.6% and 13.5% consumed 1 to 3 drinks per week, 13.8% and 13.5% consumed more than 3 but no more than 7 drinks per week, and 39.0% and 17.1% consumed more than 7 drinks per week, respectively. Significant associations among parental use variables were found only for mothers' use of alcohol and marijuana ($r = .24$, $p < .01$), mothers' and fathers' alcohol use ($r = .47$, $p < .001$), and mothers' tobacco and fathers' marijuana use ($r = .22$, $p < .05$).

Child's antisocial behavior. Children's antisocial behavior in the past year was assessed by child, mother, and father reports. The child Self-Report Delinquency Scale (Elliott et al., 1985) included 35 non–substance-use-related items that varied in terms of severity and forms of delinquency, including theft (e.g., robbery), property damage (e.g., damaged or destroyed family member's property), and violence (e.g., threatened/hit parent/student/coworker). Item response scales were coded as binary (*yes* = 1, *no* = 0). Reliabilities for the 35-item scales equaled $\alpha = .85$, .83, .81, and .61, respectively, by assessment. Counts of the different types of acts endorsed by the children were computed at each assessment and then standardized within assessments. The response scale for the Child Behavior Checklist (Achenbach & Edelbrock, 1983; 32 items; e.g., argues a lot, lying or cheating, physically attacks people, and sets fires), completed by mothers and fathers, included *not true* = 0, *sometimes true* = 1, and *often true* = 2. Across the four assessments, reliabilities ranged from $\alpha = .92$ to .94 for mothers' and $\alpha = .90$ to .96 for fathers' reports. Composite scores were created at each assessment by summing the items. Mothers' and fathers' scores were then averaged within assessments, and standardized *z* scores were created at each assessment.

The scores from child reports and combined parental reports were significantly associated within each of the first three assessments ($r = .24$, $p = < .01$; $r = .32$, $p < .001$; and $r = .31$, $p = .01$, respectively), but not at the last assessment ($r = .07$, $p = .66$). These two standardized scores were then averaged first within assessments and then across assessments, yielding one score for each child reflecting his or her average antisocial behavior across adolescence.

Parents' education level. At each assessment, mothers and fathers reported on their highest level of education completed. Categorical responses were recoded to reflect the midpoints of the categories *(<Grade 7* = 6, *Grade 7–9* = 8, *Grade 10–11* = 10.5, *high school graduate* = 12, *1–3 years of college* = 13.5, *4-year college graduate* = 16, *graduate degree* = 19.5). The maximum level of education completed by the mother and father across assessments was then calculated. On average, parents had completed 13.90 years of education.

Data analytic plan

Children's alcohol use onset across adolescence was modeled using discrete-time survival analyses (Muthén & Masyn, 2005) in Mplus version 7.3 (Muthén & Muthén, 1998–2012). Dependence among siblings' scores was accounted for by adjusting the standard errors using a sandwich estimator. Discrete-time categories were defined by the child ages of 0–10, 11–12, 13–14, 15–16, and 17–18 years. Models were estimated assuming proportional odds, which imply that the effects of the predictors and covariates are equal across children's adolescence. In addition, all models controlled for child's gender, and all of the parental substance use predictors were allowed to correlate. Except for child's gender, all independent variables were standardized, and a series of five models was fitted. The first three models examined the main effects of mothers' and fathers' tobacco, alcohol, and marijuana use, respectively. The fourth model simultaneously examined both the main and interactive effects of parents' tobacco, alcohol, and marijuana use. All two-way interactions between types of substances within a parent were tested (i.e., tobacco by alcohol, tobacco by marijuana, and alcohol by marijuana use, separately for mothers' and fathers' use), but only significant interactions (in addition to the main effects) were retained in the final model. Finally, the last model examined whether associations between child's alcohol use

Table 1. Descriptive statistics for child alcohol use onset

Variable	Boys	Girls	Children
Sample size, *n* (% of total)	63 (43%)	83 (57%)	146
Child alcohol use onset, *n* who experienced onset/ *n* at risk for onset (% who experienced onset)			
Ages 0–10 years	4/63 (6%)	1/83 (1%)	5/146 (3%)
Ages 11–12 years	1/55 (2%)	1/74 (1%)	2/129 (1%)
Ages 13–14 years	11/49 (22%)	8/70 (11%)	19/119 (16%)
Ages 15–16 years	14/28 (50%)	16/36 (44%)	30/64 (47%)
Ages 17–18 years	5/7 (71%)	5/8 (63%)	10/15 (67%)

onset and parental substance use were attenuated by child's antisocial behavior and parents' education level.

Results

Descriptives

Descriptive statistics are given in Table 1. Rates of child's alcohol use onset increased across adolescence, with the rate at onset increasing at ages 15–17 years, particularly. Across adolescence, boys' rates of alcohol use onset exceeded those of girls. In addition, boys had significantly higher levels of antisocial behavior than did girls, $t(144) = 2.31$, $p = .022$. Parental substance use and education levels did not significantly differ by child's gender.

Prediction to child's alcohol use onset

Model results for the discrete-time survival analyses are shown in Table 2. Boys showed earlier onset of alcohol use than did girls. Examination of the main effects of parental substance use by type of substance (Table 2, Models 1–3) indicated that mothers'—but not fathers'—use of tobacco, alcohol, and marijuana were associated with earlier alcohol use onset in their children. Only the main effects model for parental nicotine use yielded significant explained variance in childhood onset, equaling 15.5%.

Next, the joint influences of multiple types of parental substance use on child's alcohol use onset were considered, along with their interactive effects (Table 2, Model 4). This model explained 27.0% of the variance in onset and indicated that greater use of tobacco and alcohol (but not marijuana) by mothers was associated with increased risk for onset in their children. Interestingly, however, none of the two-way interactions between types of substances used by mothers were significant (not shown). A different pattern emerged for fathers' substance use. None of the main effects of fathers' use of tobacco, alcohol, and marijuana was significant, but the interactive effect of fathers' alcohol by marijuana use predicted earlier alcohol use onset in their offspring. Neither of the other two-way interactions of fathers' use (tobacco by alcohol and tobacco by marijuana) significantly predicted child's alcohol use onset (nonsignificant interactions not shown). Finally, in the fully adjusted model (Table 2, Model 5), children's antisocial behavior significantly increased their risk for alcohol use onset, whereas parents' education level did not, and an estimated 30.6% of variance in child's alcohol use onset was explained by this model. Moreover, both of the main effects of mothers' tobacco and alcohol use and the interactive effect of fathers' alcohol use by marijuana use remained significant in the presence of the control variables. In this model, the effect of maternal tobacco use on childhood onset of alcohol use was found to be significantly greater than was the effect of maternal marijuana use (logit

Table 2. Predicted probability of child alcohol use onset given the predictor variables and percentage of overall variance explained from the survival analyses

	Model				
Variable	Model 1: Parental tobacco use	Model 2: Parental alcohol use	Model 3: Parental marijuana use	Model 4: Parental tobacco, alcohol, and marijuana use	Model 5: Parental tobacco, alcohol, and marijuana use with controls
Child alcohol use onset predicted by:					
Child's gender (male = 1, female = 0)	2.10*	1.73*	1.92*	2.08*	1.74†
Mothers' tobacco use	1.88***	–	–	2.20***	2.13***
Fathers' tobacco use	0.77	–	–	0.72†	0.78
Mothers' alcohol use	–	1.54*	–	1.61*	1.58*
Fathers' alcohol use	–	1.02	–	1.14	1.27
Mothers' marijuana use	–	–	1.32*	1.11	1.12
Fathers' marijuana use	–	–	1.06	0.82	0.83
Fathers' alcohol × marijuana use	–	–	–	1.52*	1.49*
Parents' education	–	–	–	–	1.14
Child's antisocial behavior	–	–	–	–	1.63*
% variance explained (R^2)	15.5**	7.4	5.4†	27.0***	30.6***

Notes: All parental substance use predictor variables and control variables of child's antisocial behavior and parental education were standardized across all children (i.e., means of 0 and variances of 1).
$^{\dagger}p < .10$; *$p < .05$; **$p < .01$; ***$p < .001$.

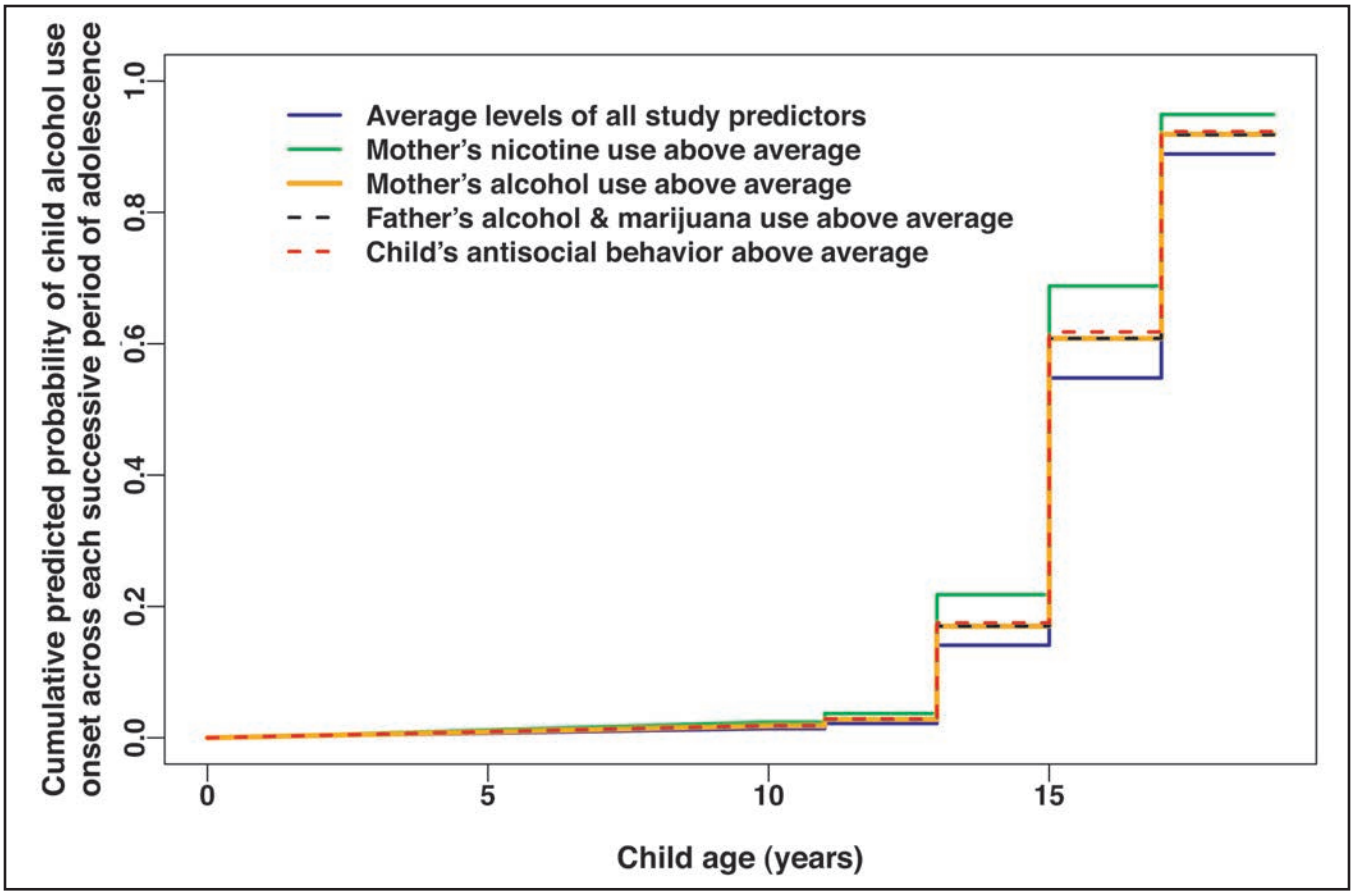

FIGURE 1. Children's cumulative predicted probability of alcohol use onset across each successive period of adolescence given parental substance use and child's antisocial behavior. *Note:* "Above average" denotes predictor values 1 *SD* above the mean.

difference of $b = 0.638$, $p = .005$), but other comparisons of the magnitude of main effects (relevant to maternal predictors only) were not significant (i.e., tobacco vs. alcohol use or alcohol vs. marijuana use). Children's predicted probabilities of adolescent alcohol use onset—which reflect cumulative estimates of onset through each successive period of adolescence, given parental substance use and the control variables—are depicted in Figure 1.

Discussion

The contribution of maternal and paternal use of alcohol, tobacco, and marijuana to their adolescent children's age at onset of alcohol use in adolescence was examined. The parents showed relatively high levels of use of the three types of substances. The hypothesis that both mothers' and fathers' use of alcohol would show main effects in predicting children's age at onset of alcohol use was partially supported; specifically, only mothers' alcohol use was associated with age at onset. The hypothesis that parental use of tobacco and marijuana would make additional contributions to their children's early onset of alcohol use, via either main or interaction effects, was supported for both mothers and fathers. Specifically, children's age at onset was predicted by mothers' tobacco use and by the interaction of fathers' marijuana use and alcohol use. These effects were observed when we controlled for parental education, child's gender, and also child's antisocial behavior, a general developmental risk factor for substance use onset in adolescence (Kerr et al., 2012) and an indicator of poor behavioral regulation (Calvete & Orue, 2012; Kim & Cicchetti, 2010).

Our findings indicate the importance of the role of mothers' substance use on children's onset of alcohol use. In fact, mothers' tobacco use was the most salient and robust predictor of adolescent onset. Fathers' substance use has received more attention as a risk factor for childhood use (Andreas & O'Farrell, 2007; Eiden et al., 2004; Jacob & Windle, 2000). This may be partly because men show higher levels of use and substance use disorder diagnoses than do women (Grant et al., 2004). On average, however, mothers spend more time with their children than do fathers (Craig, 2006); thus, their behavior may be both more visible and more influential. Exposure to maternal nicotine use could be particularly salient, considering that it typically occurs multiple times a day. In

addition, in the present study, a number of parents had separated—with about 40% of children living with both biological parents in early adolescence, likely contributing further to higher levels of contact with mothers. A further possibility is that, despite some leveling off since 2004, smoking is now less normative than in the past in the United States (Centers for Disease Control and Prevention, 2010); thus, smoking may be indicative of risk contexts and other risk behaviors of the mothers. Finally, substance-using mothers may either be more tolerant of their child's alcohol use than are other mothers, or engage in lower levels of tracking or monitoring of their child's behavior—as poor parental monitoring is a robust predictor of offspring alcohol use (Siebenbruner et al., 2006; Windle et al., 2009). Future studies should examine mechanisms accounting for these associations.

Whereas main effects of both maternal tobacco use and alcohol use were predictive of their child's age at onset of alcohol use in the multivariate model, the only predictive effect from father's use was for the interaction of alcohol use and marijuana use. It is possible that as alcohol use at moderate to high volumes is common among the men in this study (Capaldi et al., 2015), it was only when combined with marijuana use, which showed more variance among the men (Washburn & Capaldi, 2015), that a detrimental effect on children's age at onset was found. Again, this suggests that polysubstance use by parents may be particularly detrimental for the children. In addition, statistical power, and the fact that correlations among the predictor variables can reduce power, should also be considered. Note that fathers' alcohol use was positively associated with mothers' alcohol use, and both were included in the model. However, post hoc survival analyses that included only child's gender (OR = 1.85, p = .035, given child is male) and fathers' alcohol use indicated a nonsignificant association to earlier child's alcohol use onset (OR = 1.28, p = .22) and similar results for a survival analyses that included only fathers' marijuana use (OR = 1.12, p = .50).

Given the findings of the present study that both maternal and paternal use of substances in addition to alcohol influence children's age at onset of alcohol use, future research should examine these associations further, as well as whether parental use of other substances influences such issues as frequency and quantity of alcohol use and problematic use. It could be that polysubstance use is an indicator of more severe genetic or environmental risk transmitted from parents to children; thus, a factor of overall substance use also should be considered. Possible mediators of the parent–child association (e.g., exposure to substances, availability of alcohol, poor supervision) also should be examined.

There were a number of strengths of the design of the present study. First, repeated prospective assessment of both parents' use of multiple substances, as well as use of alcohol by the child across adolescence, is unusual. Second, discrete-time survival analysis is ideally suited to examine prediction to age at onset, and key control factors were included in the models. There were, however, some design limitations. First, age at onset of alcohol use could have been right censored for younger adolescents. That is, given that the youth were the third generation in a longitudinal study, they were not from a single cohort; therefore, they had not all reached the age for participation in the older adolescent assessments. In addition, right censoring occurred for youth who had not experienced onset by the end of the study period (ages 17–18 years). However, it is plausible that onset will occur at older ages when alcohol use becomes more normative and legal. Second, although gender was controlled for in the analyses, it is possible that a differential effect of parental substance use on a child's alcohol onset might have been found for boys and girls. This may be examined for the present sample in a future study when there is a larger sample of adolescents. Third, estimates of explained variance should be interpreted with caution, given that censoring can bias such measures. Finally, although models were fitted to include the simultaneous prediction of both maternal and paternal use of multiple substances (as well as interactive effects across substances), multiple regression models control for use of one substance relative to another. This does not mean, however, that children who were only exposed to alcohol use were found to be at lower risk for earlier alcohol use onset compared with children who were exposed to polysubstance use by their parent(s). Such a test would require a design with categories of exposed children (e.g., to only nicotine or alcohol use, or to alcohol and marijuana use exposure, from mother and/or father), which the current sample was not sufficiently powered to address.

Notwithstanding these limitations, the present findings illustrate that use of substances, in addition to alcohol, by both mothers and fathers may contribute to children's onset of alcohol use in adolescence. Early onset puts children at higher risk for future problematic levels of use of alcohol (Pitkänen et al., 2008; Warner et al., 2007)—as well as other negative consequences (Griffin et al., 2010)—and is thus a matter of public health significance. It may be important to identify children of polysubstance-using parents for targeted prevention programs and to identify children of substance-using mothers regardless of father's use levels. Further, prevention and treatment of parental use of tobacco, alcohol, and marijuana may delay their children's initiation of alcohol use in adolescence.

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