Contextual risks linking parents' adolescent marijuana use to offspring onset

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Abstract

Objective: We studied the extent to which parent marijuana use in adolescence is associated with marijuana use onset in offspring through contextual family and peer risks. **Method**: Fathers assessed (*n* = 93) since childhood, their 146 offspring (*n* = 83 girls), and offspring's mothers (*n* = 85) participated in a longitudinal study. Using discrete-time survival analysis, fathers' (prospectively measured) and mothers' (retrospective) adolescent marijuana use was used to predict offspring marijuana use onset through age 19 years. Parental monitoring, child exposure to marijuana use, peer deviance, peer marijuana use, and perceptions of parent disapproval of child use were measured before or concurrent with onset. **Results**: Parents' adolescent marijuana use was significantly associated with less monitoring, offspring alcohol use, the peer behaviors, exposure to adult marijuana use, and perceptions of less parent disapproval. Male gender and the two peer behaviors were positively associated with children's marijuana use onset, controlling for their alcohol use. Parents' adolescent marijuana use had a significant indirect effect on child onset through children's deviant peer affiliations and a composite contextual risk score. **Conclusions**: Parents' histories of marijuana use may contribute indirectly to children's marijuana use onset through their influence on the social environments children encounter; specifically, those characterized by more liberal use norms, exposure to marijuana use and deviant and marijuana-using peers, and less adult supervision. Given that alcohol use onset was controlled, findings suggest that the contextual factors identified here confer unique risk for child marijuana use onset.

Keywords: Marijuana, Onset, Adolescence, Deviant peers, Intergenerational studies, Fathers

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1. Introduction

Marijuana use is relatively common among adults in the U.S. (30.2% among 19-28 year olds; Johnston et al., 2013a) and, as with use of other substances, becomes increasingly prevalent across adolescence. In 2012, lifetime prevalence of marijuana use among 8th, 10th, and 12th graders in the Monitoring the Future (MTF) Study was 15.2%, 33.8%, and 45.2%, respectively (Johnston et al., 2013b). Recent legalization of recreational marijuana use in several U.S. states may reflect increasingly liberal use norms and may lead to increased availability and modeling of marijuana in the homes and communities of adolescents. At the same time, however, there is increasing evidence that marijuana use may have serious effects on the developing brains of adolescents, including increased risk for disorders such as schizophrenia (Arseneault et al., 2004; Bossong and Niesink, 2010; Moore et al., 2007). Earlier onset also is associated with heavier and more persistent use, marijuana use disorder, and negative socioeconomic consequences during early adulthood (Broman, 2009; DeWit et al., 2000). Consequently, there is good reason to delay onset among youth. The identification of modifiable risk and protective factors will inform prevention efforts to do so.

Many risk and protective factors relevant to marijuana use may be of similar relevance to other commonly used substances (Hansen et al., 1987). Social influences such as those related to parenting, peer group, and neighborhood on use of any specific substance in adolescence often overlap with those for use of other substances and for the general category of problem behaviors (Dishion and Patterson, 2006; Hicks et al., 2004). Additionally, polysubstance use is common in adolescence (Leatherdale et al., 2009), and onset of one kind of substance use hastens onset of others (Kosterman et al., 2000). Thus, models of risk for marijuana use onset should

accommodate the likelihood that some risks tend to be generalized rather than substance specific. Identifying pathways of association that are of special importance in relation to marijuana use would aid the refinement of prevention programs.

Parent substance use is an important risk factor for child use, and some research concerns marijuana specifically (Duncan et al., 1995; Washburn and Capaldi, 2014a, 2014b). Most studies measure parent substance use in adulthood (e.g., Bailey et al., 2009). However, across early adulthood, marijuana use becomes less probable and quantity of use decreases even among chronic users (Washburn and Capaldi, 2014a). Thus, parental use in middle adulthood may represent atypical and problematic behavior, and variability in parents' prior use, which may have long-term influences, is ignored. We focus here on marijuana use during parents' adolescence, and examine the extent to which it is associated with family and peer contexts that lead to their children's marijuana use onset.

Genetic studies generally support a heritable component to substance dependence, but environmental influences are stronger in adolescence and for earlier stages of use (e.g., onset) (Dick, 2011; Kendler et al., 2008; Lynskey et al., 2010). Thus, parents transmit risk for marijuana use, in part, through the social contexts in which offspring are raised. Social contextual models of marijuana use in adolescence are derived from those proposed for alcohol use (e.g., Conger and Rueter, 1996; Kerr et al., 2012) and emphasize parents' influence on both home and peer environments that model use, communicate deviant norms, and offer (or fail to limit) access to marijuana. Consistent with these notions, the age trends in marijuana use prevalence identified in MTF were paralleled by clear trends toward older youth more often having friends who use marijuana, personally approving of trying it, less often believing that occasional use is harmful, and being able to easily get it (Johnston et al., 2013b).

In the present study, fathers who have been studied since childhood participated with their offspring and their offspring's mothers in an ongoing prospective study of risk for alcohol and drug abuse. Factors from children's family and peer contexts potentially linking parents' adolescent marijuana use with risk for child onset were examined, including those known to confer generalized risk for adolescent problem behaviors (parental monitoring and deviant peers; e.g., Dishion and Patterson, 2006). Then—as in our prior work on alcohol-specific risk (Kerr et al., 2012) and drawing on prior studies of marijuana (e.g., Ellickson et al., 2004) — outcome-specific risk factors were examined; specifically, having friends who use marijuana, exposure to marijuana use, and perceived parent disapproval of child marijuana use. Models also controlled for whether children had shown onset of alcohol use. This approach highlighted predictive paths to marijuana use onset that were not better explained by generalized risk processes shared with use of this more commonly encountered substance.

The study offers several other advances over prior work. Given the design of the study, adolescent marijuana use histories were known for all fathers, who tend to be less represented in developmental research. Additionally, most risk factors were measured using multiple informants, and substance use by fathers and their children were measured prospectively. Finally, the discrete-time survival analysis approach is especially relevant given the sensitivity needed to model onset and examine how risk may accumulate with development.

1.1. Hypotheses

Study hypotheses were as follows: (a) parents' marijuana use during their own adolescence will be associated with an earlier onset of marijuana use among their children; (b) the intergenerational transmission of such risk will be largely indirect through general contextual risks in the family and peer contexts, including peer deviance and less parental monitoring; (c)

parent marijuana use will be associated with several outcome-specific risks for child marijuana use: namely, having friends who use marijuana, exposure to marijuana use, and low perceived parent disapproval of child use; (d) these general and specific factors will hasten the onset of marijuana use, beyond what would be predicted from child alcohol use. We also control for child gender, given the earlier substance use onset observed in boys in this and other samples (Capaldi et al., under review; Kosterman et al., 2000).

2. Methods

2.1. Participants

The present study was based on 93 fathers (recruited as children to the Oregon Youth Study [OYS]; Capaldi and Patterson [1989] and assessed regularly to the present day); their biological children (n = 146; 83 girls), 85 of the children's mothers, and 90 of the fathers also participated in the Three Generational Study (3GS). Children had to have participated in at least one of the four waves between ages 11 and 19 years as of March, 2014 to be included in the present analyses. A minority (38.4%, n = 56/146) were living with both biological parents at the earliest wave considered here. Children were 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).

2.2. Procedures

Fathers' reports of adolescent marijuana use were collected annually from ages 11-12 to 17-18 years. 3GS assessments started in early childhood and four occurred across adolescence. Mothers, fathers, and children were interviewed separately. The N available for each 3GS wave is determined by the ages of the maturing children; total N = 136, 126, 84, and 42 at the age 11-13, 13-15, 15-17, and 17-19 year assessments, respectively.

2. 3. Measures

Child marijuana use onset was modeled across early to late adolescence. Parents' adolescent marijuana use (i.e., the antecedent) chronologically preceded all other variables. For the mediating and control variables, scores were averaged across all waves prior to and including the wave of marijuana use onset; scores for children who did not onset were averaged across all waves in which they were at risk for doing so (i.e., through their final wave of participation). All predictors were aggregated by using a mean score (after standardizing within reporters and assessments); the only exceptions were child gender and alcohol use (binary). The temporal sequence of the antecedent, mediators, controls, and outcome variables affords a longitudinal examination of the indirect effects of parents' adolescent marijuana use on child onset mediated through prior and concurrent contextual risk factors.

Child marijuana use onset. At each assessment, children were asked if they had ever tried marijuana ("yes" or "no") and, if so, age at first use. The minimum age of first reported use (age 11 years) corresponded to the minimum age at the first assessment (ages 11-13 years). It was therefore unnecessary to incorporate left censoring (i.e., having onset prior to the initial assessment) into the survival models. New reports of having ever used marijuana at the three later assessments were used to define onset for each subsequent period, creating four binary variables for marijuana use onset at ages 11-13, 13-15, 15-17, and 17-19 years. Once a child onset, all subsequent scores were set to missing values as s/he was no longer at risk for onset at those ages. Right censoring of onset due to age (e.g., if a child was too young to have participated yet at the age 17-19 year assessment) also was represented with missing data codes.

Parent marijuana use during adolescence. During their initial 3GS assessment, mothers reported retrospectively how often they had used marijuana during adolescence (from ages 13 to

18 years). Response categories included “1 = never”, “2 = hardly ever”, “3 = sometimes”, and “4 = often”. For mothers, quantities of adolescent use were not queried. Fathers’ adolescent marijuana use was annually and prospectively measured from ages 11-12 to 17-18 years; specifically, as boys they were asked how often they had used marijuana in the last year (i.e., frequency) and how much they had used on a typical occasion (i.e., quantity). Frequency was capped at 999 times in the last year (i.e., having used more than twice daily for the last year). Response categories for quantity of marijuana use included various methods of use and were equated to grams as follows: “one joint” = 1 gram, “one toke or bong hit” = 1/10 gram, “one ounce” = 28 grams. Fathers’ adolescent marijuana use scores were calculated as the product of frequency and quantity of use at each annual assessment, then log-transformed to reduce positive skew, and averaged across all (OYS) adolescent waves. Mothers’ and fathers’ scores were significantly associated (r = .25, p = .003) and averaged, creating parental adolescent marijuana use scores. Most mothers (63.4%) and fathers (53.8%) reported at least some marijuana use during adolescence.

Parental monitoring. Parental monitoring included child, mother, and father reports at each 3GS assessment as follows: (a) Children’s reports (6 items scaled from “1 = Never or almost never” to “5 = Always or almost always”: e.g., How often do you check in with your parents or babysitter before going out?). Reliabilities by wave were α = .61, .68, .78, and .81, respectively. (b) Mothers’ and fathers’ monitoring was assessed by: (i) *direct monitoring* of children’s whereabouts and activities (7 items, scaled “1 = Never or almost never” to “5 = Always or almost always”: e.g., How often is your child at home or a friends without adult supervision?). Reliabilities were α = .41, .70, .72, and .71 for mothers and α = .61, .76, .69, and .87 for fathers. (ii) *indirect monitoring* via time spent and communication with children (5 items:

e.g., How many days per week [0 - 7] do you spend with you child, talk to your child about plans for the coming day, what happened during the day/with his or her friends?). Reliabilities by wave were α = .87, .87, .82, and .86 for mothers, and α = .92, .92, .94, and .88 for fathers. Note that the low reliabilities for parents' reports of direct monitoring at the initial assessment were due to the fact that essentially all parents reported high monitoring. Direct and indirect monitoring scores were positively associated within wave for mothers, r = {.16, .33, .46, .48}, p = {.07, <.001, <.001, .002}, and fathers, r = {.43, .36, .53, .36}, p = {<.001, <.001, <.001, .08}. Composite mother- and father-report scores were created by averaging these two forms of monitoring. Finally, the child-, mother- and father-reported scores were averaged, yielding one monitoring score at each assessment.

Perceived parental disapproval of child marijuana use. Children who abstained from marijuana use were asked how upset their parents would be if they had used marijuana (i.e., hypothetical), and children who reported using marijuana were asked how upset their parents would be if (or were when) they found out they had used marijuana. Response categories included "1 = not at all upset", "2 = a little upset", "3 = somewhat upset", and "4 = very upset".

Child exposure to marijuana use. At the first two assessments (child ages 11-13 and 13-15 years), mothers and fathers were asked if their children had seen anyone use marijuana in the last year. Response scales of "1 = never", "2 = hardly never", "3 = sometimes", and "4 = often" were recoded to binary responses of "yes = 1" and "no = 0", and the maximum of the mother's and father's reports was taken to yield one binary variable at each assessment.

Child peer marijuana use. At each assessment, children were asked how many of their friends had used marijuana in the last year. Response scales included "1 = none", "2 = some", and "3 = most".

Child deviant peer association. Composite scores were created using child and parent reports. Items pertaining to substances were eliminated. Child reports (from the interview) involved seven items (e.g., my peers steal, burglarize, damage property). Response categories included "1 = none", "2 = some", and "3 = most". Reliabilities were α = .69, .74, .78, and .69, respectively, by wave. Mothers and fathers reports involved four items from the Peers Questionnaire (Dishion and Capaldi, 1985; e.g., my child's peers steal, are a bad/good influence) and one item from the Child Behavior Checklist (CBCL; Achenbach, 1991; i.e., my child socializes with children who get into trouble). Response categories ranged from "1= Never or almost never" to "5 = Always or almost always" for the Peers Questionnaire items, and 3 options recoded from 0, 1, and 2 to "1 = Not true", "3 = Sometimes true", and "5 = Often true" for the CBCL item, respectively. Reliabilities were α = .83, .81, .88, and .73 for mothers and α = .58, .75, .74, and .78 for fathers, respectively, by wave. The children's, mothers', and fathers' constructs were significantly associated at all assessments (r = {.28 to .57}, p = {<.001 to .003} for child-mother; r = {.31 to .49}, p = {<.001 to .002} for child-father; and r = {.51 to .58}, p <.001 for mother-father), except for the child-father (r = .12, p = .619) and mother-father (r = .34, p = .163) constructs at the last assessment (ages 17-19 years). Scores were averaged, yielding a single score of deviant peer association at each assessment.

Overall contextual risk factor. Excluding parental monitoring, all but one of the bivariate associations among the mediating predictor variables were significant (see Table 2). Exploratory factor analyses confirmed a unidimensional solution for an overall contextual risk factor that was comprised of: parental disapproval of child marijuana use (reverse scored), child exposure to marijuana use, child peer marijuana use, and child deviant peer association. The common factor explained 29.9% of the total variance. Contextual risk scores were created by averaging these

four variables.

Child alcohol use. At each assessment, children were asked if they had ever consumed at least one whole alcoholic drink (yes/no) and, if so, the age at which they had first done so. Child alcohol use scores equaled "1" if onset occurred prior to or concurrent with marijuana use onset or "0" if onset did not occur or occurred after marijuana use onset.

2.4. Data Analytic Plan

Discrete-time survival analysis (DTSA; Muthén and Masyn, 2005) was used to model children's marijuana use onset across early to late adolescence using Mplus version 7.3 (Muthén and Muthén, 1998-2012). Child age of onset categories were 0-11, 11-13, 13-15, 15-17, and 17-19 years. Models were estimated assuming proportional odds, which imply that the effects of the predictors and covariates are equal across children's adolescence. Independent variables were standardized, except for the binary variables of child gender (coded as "male = 1", "female = 0") and alcohol use. Dependence among siblings' scores was accounted for by adjusting the standard errors using a sandwich estimator.

The first two DTSA models identified the associations of child gender and then alcohol use with onset of marijuana use across early to late adolescence. All subsequent models included these controls. Next, Model I tested whether children's marijuana use onset could be predicted by their parents' adolescent marijuana use. Models II - VI examined whether parents' adolescent marijuana use indirectly increased risk for marijuana use onset in their offspring via contextual risk factors; the first five separately examined each mediating risk factor. Finally, Model VII examined mediation by the overall, aggregated contextual risk. Mediation was tested in Mplus version 7.3 by creating interaction terms between (a) the effects of the antecedent on the mediators and (b) the effects of the mediator in the outcome, and testing whether these

parameters were significantly different from zero.

3. Results

3.1. Descriptives and Correlations

Descriptive statistics for all study variables are given in Table 1. Child marijuana use onset increased across adolescence, from 2% or less by age 13 years, to approximately 50% at ages 17-19 years; 36.3% showed onset. Boys' rates of marijuana use onset exceeded girls at the first three assessments but were essentially equal by ages 17-19 years. Relative to girls, boys were monitored less by their parents (t[144] = 3.10, p = .002), 4.46 times more likely to have consumed at least one whole alcoholic drink in adolescence (p = .035), and had marginally higher deviant peer association (t[144] = 1.76, p = .081). Bivariate correlations among the independent variables are given in Table 2. Parent adolescent marijuana use was significantly associated with all proposed mediators in hypothesized ways. Parental monitoring was not associated in the expected manner with mediators other than deviant peer associations; other variables were interrelated—generally significantly—in the posited directions.

3.2. Discrete-Time Survival Analysis Model Results

In the two initial models, onset was significantly more likely among boys than girls (OR = 2.01, p = .018) and (marginally so) among children with prior or concurrent alcohol use (OR = 2.02, p = .073). Then, Model I revealed a positive but nonsignificant association of parents' adolescent marijuana use with child marijuana use onset (Table 3, Model I). Next, each mediating contextual factor was tested individually (Table 3, Models II-VI). Only greater peer marijuana use and deviant peer association were related to child marijuana use onset. Furthermore, the only support for an indirect effect of parents' adolescent marijuana use on their children's earlier onset was through greater deviant peer association; those parents who had used

more marijuana as adolescents had offspring who associated with peers higher in antisocial behavior, increasing their risk for earlier marijuana use onset. Finally, the composite contextual risk factor also operated as a mediating factor (Table 3, Model VII); greater parental use of marijuana as adolescents predicted greater child overall contextual risk, which in turn increased risk of child marijuana use onset.

4. Discussion

Findings of this prospective intergenerational study indicate that adolescents who more often used marijuana were more likely to raise children in family and peer contexts that encouraged or failed to inhibit children's early onset of marijuana use. In such families, parents less closely monitored their children's whereabouts and associates, children had more contact with deviant peer groups, more often had seen someone use marijuana, and believed their parents would disapprove less if they tried the drug. Such contexts were expected to perpetuate risk for marijuana use in the next generation and are known to be associated with the host of related problem behaviors for adolescents (e.g., Dishion and Patterson, 2006). To our knowledge, this is the first intergenerational study documenting how adolescents' marijuana use is associated with the contexts in which they raise their future offspring. Prior studies (e.g., Bailey et al., 2009; Washburn and Capaldi, 2014a) have tended to consider parent use of any substance or have measured use during the child's life. Such use may have proximal adverse effects on the caregiving environment through parental intoxication and impairment. Thus, parental marijuana use during adolescence versus adulthood represents rather different risk processes for offspring.

As expected, the family and peer contextual factors examined were generally interrelated and formed a risk composite through which parents' adolescent marijuana use was associated with children's marijuana use onset. This association was demonstrated even when children's

histories of having previously or concurrently used alcohol (which also was associated with the risk composite) was controlled. This suggests that parent marijuana use and the contextual risk composite confer both general and marijuana-specific risk for children's onset. Also notable, deviant peer association was the only contextual factor that, on its own, mediated the relations between parent marijuana use in adolescence and offspring onset risk.

Overall, rates of marijuana use onset were relatively low in the sample (approximately 36%), in part because many participants had not yet reached the ages of peak onset. Though this fact may have reduced statistical power, it was notable that the direct association between parents' adolescent marijuana use and child onset was not significant. Also surprising was that low parental monitoring was not associated with children's marijuana use onset, although this parenting behavior was predicted by parents' adolescent marijuana use. Monitoring has been associated with the range of problem behavior outcomes such as delinquency; earlier and health-risking sexual behavior; and use of alcohol, tobacco, and other drugs (e.g., Capaldi et al., 2002). Additionally, prior studies have found that parents who in adolescence showed more positive adjustment and lower rates of these problem behaviors later showed higher levels of effective parenting, including monitoring, of their own children (Bailey et al., 2009; Kerr et al., 2009; Neppl et al., 2009), thus highlighting parental monitoring as potential mechanism disrupting or linking problem behaviors over generations. To our knowledge, this is the first time this pathway has been examined specifically for marijuana use, and we found no support for it as an explanatory mechanism in predicting onset. Still, power was limited, and we examined only first onset. Child onset of patterned use may have stronger connections with parental use and monitoring.

4.1. Strengths and Limitations

The present study had numerous strengths, including fully prospective data from fathers' adolescence, use of DTSA, a powerful and developmentally sensitive approach to examining prediction to age of onset, and control for child gender and alcohol onset. Thus, the study offers particularly strong evidence that the contextual factors tested make substance-specific contributions to risk for marijuana use onset. The study also had some important limitations. First, age of onset of marijuana use was right censored for many adolescents; most youth had not shown onset to date and, given the study design, many had not yet reached the age for the later adolescent assessments when onset becomes increasingly likely. However, the analytic approach ameliorates these concerns, as onset hazard estimates are based only on participants assessed at that age. A second limitation was that genetic or other biological mechanisms of transmission (e.g., prenatal exposure; Day et al., 2006) were not examined. Third, adolescent marijuana use measures were not equivalent for mothers and fathers. Finally, the sample was predominantly European American (73%) and lived in a region of the U.S. with more liberal marijuana use norms (e.g., early legalization of recreational use). Future research will determine whether such laws lead to changes in contextual factors— such as modeling, communication of norms, and drug access—that hasten child onset relative to prior cohorts.

4.2. Conclusions

Early onset of marijuana use may expose adolescents to more years of risk through a variety of processes, including detrimental effects on the developing brain (e.g. Arseneault et al., 2004). Prevention efforts directed at decreasing contact with deviant peers may delay or forestall the onset of marijuana use, and limiting contact with marijuana-using peers may be uniquely important. These efforts may have an impact on marijuana use onset independent of whether children use alcohol. Another key finding was that marijuana-using adolescents more often grew

up to form families in which substance use is encouraged or not discouraged. As noted in our prior intergenerational work, such findings hold out the promise that successful prevention may benefit not only its proximal targets, but also these individuals' future partners and offspring. From this perspective, effective prevention can be expected to have legacy effects.

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Table 1

Descriptive Statistics.

	Boys	Girls	Children
Sample size (*n*, % of total)	63 (43%)	83 (57%)	146
Child marijuana use onset (*n* who onset / *n* at-risk to onset, % who onset)			
Prior to age 11 years	0/63 (0%)	0/83 (0%)	0/146 (0%)
Age 11-13 years	3/59 (5%)	0/77 (0%)	3/136 (2%)
Age 13-15 years	8/51 (16%)	7/72 (10%)	15/123 (12%)
Age 15-17 years	14/33 (42%)	9/38 (24%)	23/71 (32%)
Age 17-19 years	4/9 (44%)	8/16 (50%)	12/25 (48%)
Parental monitoring	-0.29 (1.13)	0.22 (0.83)	0 (1.00)
Parental disapproval of child marijuana use	-0.01 (0.97)	0.01 (1.02)	0 (1.00)
Child exposure to marijuana use	-0.07 (0.96)	0.05 (1.03)	0 (1.00)
Child peer marijuana use	0.05 (1.13)	-0.04 (0.89)	0 (1.00)
Child deviant peer association	0.17 (1.03)	-0.13 (0.97)	0 (1.00)
Overall contextual risk	0.06 (1.03)	-0.05 (0.98)	0 (1.00)
Child alcohol use onset prior to or concurrent with marijuana use onset (n, %)	33 (52.4%)	29 (34.9%)	62 (42.5%)
Parent adolescent marijuana use	-0.16 (0.98)	0.12 (1.01)	0 (1.00)

Note: Tabled values denote mean (standard deviation) unless noted otherwise.

Table 2

Bivariate correlation matrix of predictor and control variables.

	2.	3.	4.	5.	6.	7.	8.	9.
1. Parent adolescent marijuana use	-0.17*	-0.18*	0.18*	0.18*	0.23**	0.29***	0.16M	-0.14
2. Parental monitoring		0.13	0.03	-0.15M	-0.40***	-0.25**	-0.11	-0.25**
3. Parental disapproval of child marijuana use			-0.23**	-0.17*	-0.04	-0.54***	-0.24**	-0.01
4. Child exposure to marijuana use				0.29***	0.36***	0.71***	0.22**	-0.06
5. Child peer marijuana use					0.47***	0.72***	0.43***	0.05
6. Child deviant peer association						0.70***	0.15M	0.15M
7. Overall contextual risk							0.39***	0.05
8. Child alcohol use (yes = 1, no = 0)								0.18*
9. Child gender (male = 1, female = 0)								

Note: *** $p < .001$. ** $p < .01$. * $p < .05$. M $p < .10$.

Table 3

Model Results: Child Marijuana Use Onset given Parent Adolescent Marijuana Use (Antecedent) and Mediating Contextual Risk Factors Controlling for Child Gender and Alcohol Use Onset.

	Direct Effects Model	Models with Mediation by					
	I. Parent use	II. Monitoring	III. Parental Disapproval	IV. Child Exposure	V. Peer Use†	VI. Deviant Peer	VII. Contextual Risk
Child marijuana use onset predicted by: (*OR*)							
Antecedent:							
Parent adolescent marijuana use	1.34	1.31	1.29	1.33	1.34	1.13	1.19
Mediators:							
1. Parental monitoring	--	0.87	--	--	--	--	--
2. Parental disapproval	--	--	0.82	--	--	--	--
3. Child exposure to	--	--	--	1.30	--	--	--
4. Peer use	--	--	--	--	**3.10*** **	--	--

5. Deviant peer	--	--	--	--	--	**2.47*** **	--
6. Contextual risk	--	--	--	--	--	--	**2.42*** **
Controls:							
Child gender (male = 1)	**2.14***	1.97 [M]	**2.16***	**2.30***	**3.33****	1.78	**2.64****
Child alcohol onset	1.68	1.67	1.55	1.48	0.76	1.51	0.93
Indirect effect of parent use on child onset via mediator	--	1.02	1.04	1.05	1.22	**1.23***	**1.29****
Mediator predicted by: (standardized beta)							
Parent use	--	-0.17 [M]	-0.18	**0.18***	0.18 [M]	**0.23***	**0.29*** **

Note: OR = Odds ratio.

† An alternative model that also controlled for child deviant peer association was fit to the data; results indicated that child deviant peer association marginally predicted child marijuana use onset (*OR* = 1.74, *p* = .054) but did not attenuate the effect of child peer marijuana use on child marijuana use onset (*OR* = 2.54, *p* = .001).

[M] $p < .10$. *$p < .05$. **$p < .01$, ***$p < .001$.