Paternal and Maternal Prescription Opioid Use and Misuse: General and Specific Risks for Early Adolescents' Substance Use

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Abstract

Background: Parent substance use is a risk factor early adolescents' substance use. Theoretical models of deviance and general substance use risk may not apply to risk-transmission pathways involving parents' prescription opioid misuse (POM) and child outcomes. Thus, we examined predictions of children's alcohol, tobacco, and marijuana (ATM) use in early adolescence, from parental POM, delinquency, depressive symptoms, and ATM use.

Method: Children (*n* = 216; 121 female) participated from early childhood to ages 11-12 or 13-14 years with their 111 fathers and 136 mothers. At all available waves, self-reports were collected on each parents' POM, ATM, prescription opioid use (POU), depressive symptoms, and delinquent behavior, and children's ATM use.

Results: Poisson regressions were run separately by parent, controlled for child age and gender and paternal age at child's birth, and accounted for clustering of children in families. Child ATM use was predicted by paternal POM, but the effect was better explained by paternal ATM use, which was a stronger effect in families with higher father–child residential contact. In contrast and unexpectedly, mothers' POU but not POM predicted child ATM use, and the effect was not explained by the significant predictions from maternal ATM use and delinquency.

Conclusion: Fathers' POM and mothers' POU predicted child ATM use by early adolescence. Findings generally were consistent with parent–child risk-transmission processes described for other substances. Resident fathers' substance use and multiple maternal risk factors are worthy foci for prevention of the intergenerational transmission of substance use.

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1. Introduction

According to the Centers for Disease Control (2018), drug overdoses killed over 60,000 Americans in 2016, 66% of which involved a prescription or illicit opioid. National survey data indicate young adult parents are more likely to report prescription opioid use (POU) than their nonparent peers, and parents reporting POU are more likely than other parents to report prescription opioid misuse (POM) and alcohol or other substance dependence (Austin & Shanahan, 2017). As part of the effort to understand and prevent opioid abuse, it is critical to identify how the current generation of children may be responding to POM by their parents. Yet there is little information about whether parental POM is associated with risk for child substance use.

The opioid epidemic raises questions about the value of models that place behavioral deviance or emotional distress at the center of understanding why substance abuse develops. Whereas antisocial traits, deviant contexts, and negative affect are relevant to the development of substance use and abuse (Chassin et al., 2004b), otherwise healthy and socially integrated individuals may develop an opioid-use disorder, with prescribed use recognized as an entry point to abuse (Volkow & McLellan, 2016).

Two studies support that prevailing models of substance use development are at least somewhat relevant to POM. First, in a recent cross-sectional national study (NSDUH) of parent–child dyads, correlates of adolescent prescription opioid abuse were highly consistent with deviance models, and included adolescents' delinquency, depression, other substance use, and perceived peer substance use—as well as parental smoking, prescription opioid abuse, and low monitoring (Griesler et al., 2019). Parents' depression history was controlled, but their deviant behavior and—of potential relevance to parental modeling and other social transmission mechanisms—the extent to which parents and children were in contact was not. Second, findings from the longitudinal Oregon Youth Study (OYS) of at-risk community men supported a pathway predicting POM in adulthood from antisocial behavior in adolescence (Capaldi et al., 2019). Specifically, among the risk factors during adolescence for adult POM, delinquency was the primary predictor, whereas parental substance use and adolescent use of other substances were not uniquely associated. Predictors of adult POM from risk factors in adulthood were more complex; in multiple regression models, significant predictors included adult alcohol, tobacco, and marijuana (ATM) use and antisocial behavior, but not depressive symptoms. The study supported a general pathway of risk for POM involving deviance in adolescence and adulthood and also highlighted the importance of risk from other substance use in adulthood. The question addressed in the present study concerns how parental POM may be related to early substance use by their children, after accounting for parental deviance (assessed by delinquency), depressive symptoms, use of other substances, and parent–child contact.

It is well established that parental substance use and abuse increase risk for adolescents' substance use (Bailey et al., 2006; Capaldi et al., 2016; Chassin et al., 2004b). We consider three risk-transmission pathways. First, *general problem-risk pathways* implicate the broad classes of parental deviance (e.g., antisocial behavior) and psychopathology (e.g., depression) associated with children's genetic and contextual risk for problem behaviors of which early substance use is but one (Chassin et al., 2004b; Hicks et al., 2004; Nadel & Thornberry, 2017). Second, a *general substance-use-risk* pathway distinguishes risks for substance use from those explained by more general problems (i.e., deviance and depression). Consistent with this pathway, intergenerational studies have found evidence that some associations between parents' and children's problem behaviors are domain specific (e.g., Nadel & Thornberry, 2017).

General substance-use-risk pathways may not implicate parental use of any particular substance as a risk factor for children's substance use, however, as most individuals who use one substance also use others (e.g., Moss et al., 2014). Yet some prior studies have indicate parental use of a specific substance may confer unique risk to their children; for example, predicting child use of the same substance, beyond more general parental and contextual risk indicators (Bailey et al., 2006; Chassin et al., 2004a; Kerr et al., 2012). Thus, in the present study, we also assess a third pathway involving potential contributions of parental POM to risk for child ATM use that are unique from those explained by parental deviance and depressive symptoms (*general problem-risk pathway*) and parental ATM use (*general substance-use-risk pathway*).

Models of general and specific risk inform prevention efforts, as they signal whether targeting very broad (e.g., parent antisocial behavior) or more specific factors (e.g., exposure to drunk adults; Kerr et al., 2012) will be most impactful. To date, such models have not been applied to whether parental POM increases risk for child substance use.

1.1. Present Study and Hypotheses

The OYS–Three Generational Study (OYS–3GS) data are highly relevant to the present questions. For one, the opioid crisis has differentially affected the demographic groups (male, primarily White, lower-socioeconomic status [SES]) to which one or both participating parents belong (Cicero et al., 2014). Furthermore, the opioid crisis coincided temporally (late 1990s to 2010s) with OYS–3GS parents' early adulthood when vulnerability to substance abuse is highest.

Using data from this study of children assessed regularly from early childhood to middle adolescence, we tested the hypothesis that parental POM during their children's lives would be associated with children's use of ATM by age 14 years. We examined child use of any or all of the three substances as: we had no basis for differential hypotheses by substance type; use of only one substance is unusual among adolescents; early polysubstance use is a higher-risk behavior (Kerr et al., 2019; Moss et al., 2014); and focusing on a single substance could reduce statistical power. Measurement capitalized on the longitudinal assessment design but models did not temporally separate predictors and outcomes due to the relatively low prevalence of parent POM and of early adolescent ATM use.

In separate models for fathers and mothers, these hypotheses and questions were evaluated:

1.   We tested whether parent POM would be positively associated with child ATM use beyond other control variables, including parent POU.

2.   Consistent with a *general substance-use-risk pathway*, we examined whether parents' ATM use would be related to child ATM use and whether parent ATM use would wholly explain associations between parents' POM and child ATM use.

3.   In line with a *general problem-risk pathway*, we predicted that parents' delinquency and depressive symptoms would be positively associated with child ATM use, and we tested whether these parent risks would account for associations between parents' POM and child ATM use.

4.   Next, we examined whether a unique association persisted between parent POM and child ATM use, after including general risk pathways in the model.

5.   Finally, many fathers do not live with their children, precluding or limiting socially mediated transmission processes such as substance use modeling and parental monitoring. Therefore, we expected father–child contact to moderate associations that fathers' POM and other substance use had with children's ATM use (i.e., greater risk from substance-using fathers when father–child contact was high vs. low), as has been found in intergenerational studies of antisocial behavior (Thornberry et al., 2009).

2.   Method

2.1.   Participants

Data from children ($n = 216$; 121 female) of 111 fathers and 136 mothers were analyzed presently. Fathers were enrolled in the OYS when they were ages 9–10 years in 1984. OYS boys were at-risk for delinquency due to attending grade 4 at schools in higher-delinquency neighborhoods in a medium-sized metropolitan area in the U.S. Pacific Northwest; 74% were recruited (Capaldi & Patterson, 1987). OYS boys were primarily White (90%) and from less-educated (17% and 8% of fathers and mothers were college graduates, respectively) and lower-SES families (75%; Hollingshead, 1975), many families (33%) received welfare or food stamps.

Men who became fathers ($n = 154$ of 206 OYS men to date) were recruited with their children ($n = 337$ to date) and their children's mothers to the OYS–3GS. Initially, all children and cohabitating stepchildren were included; budget constraints later constrained recruitment to the first two biological children per mother. Families were targeted for assessment at child ages 1.5, 3, 5, 7, 9–10, 11–12 and 13–14, although some children were older than the targeted age.

Thus, the present analyses are limited to the 216 children who completed self-reported assessments of substance use at either the age 11–12 ($n = 204$) or 13–14 ($n = 181$) year assessment. Substance use was first queried at ages 9–10 years for 106 children. Although

that wave was suspended in 2008 (budgetary reasons), we used the data in the present study. Of the children, 38 participated at only one wave, 81 at only two, and 97 at all three of the waves spanning ages 9 to 14 years. Fathers had one ($n = 36$), two ($n = 50$), or three or four ($n = 25$) children in the study; the 136 biological mothers had one ($n = 67$), two ($n = 58$), or three ($n = 11$) children in the study.

2.2. Procedures

Fathers' delinquency and substance use were assessed through OYS, continuing to age 42 years. Fathers and mothers also were assessed during 3GS visits with their children. OYS and 3GS assessments included questionnaires, interviews, and observations (not considered here). Adult participants provided informed consent. All procedures were approved by the IRB of Oregon Social Learning Center. Participants were compensated.

2.3. Measures

Parent measures were based on all OYS and 3GS assessments collected between the dates of the child's birth and the date the child was last assessed. Means across time were *z*-standardized for use in regressions.

2.3.1. Parent delinquency.—Scores were sums of 16 items of a self-report delinquency measure of minor and major index crimes (e.g., purposely destroying or damaging property; sold marijuana; attacked someone; Elliot, 1983) coded if the behavior did (1) or did not (0) occur.

2.3.2. Parent depressive symptoms.—Scores on the 20-item Center for Epidemiologic Studies, Depression (Radloff, 1977) collected at 3GS visits had acceptable internal consistency across waves (fathers *M*[*SD*] α=.92[.03], range .87–.95; mothers α=.92[.01], range .89–.93).

2.3.3. Parent ATM use.—Use of each substance during the child's life was assessed repeatedly for each parent; for each self-report scale, the mean across time was calculated for each parent. *Tobacco use* was an aggregate of cigarettes, pipes, and chewing tobacco, converted to mg doses of nicotine per week (Capaldi et al., 2016). For the *alcohol use* scale, beer, wine, and hard liquor was measured from (a) any use in the past year and (b) for users, the number of times used (capped at 365 times per year to reduce skew) and the amount consumed on a typical occasion (i.e., in units of <1 drink, 1, 2, 3, 4 to 5, 6 or more). Units were placed on a common alcohol-content scale (Capaldi et al., 2015). Volume of alcohol use was calculated from frequency of use multiplied by the usual amount consumed for beer, wine, and hard liquor, separately; the three values were summed to create the total yearly alcohol-volume score. *Marijuana use* was the reported number of times in the past year, and the quantity usually used per occasion—converted to grams as follows: 1 joint=1 gram; 1 toke or hit=1/10 gram; 1 ounce=28 grams. Grams of marijuana used per year were estimated as the product of these two variables.

2.3.4. Parent POM and POU.—From ages 20–21 years, OYS men were asked specifically asked about opioid use. At 3GS assessments, parents were shown a card of

common names of opioids and asked to list the types they had used. For each prescription opioid used, parents were asked whether they always got the drug from a doctor and, if so, whether they ever took more than prescribed. Obtaining the opioids from a source other than a doctor or taking more than prescribed was considered POM. For each parent at each measurement occasion, two binary variables were recorded for whether or not in that year they reported (1) POU or (2) POM. POU was only scored in a given year if POM was denied that year, and POU was initially considered as a control variable.

2.3.5.   Child ATM use.—At each of up-to-three interviews (ages 9–14), children were asked about ATM use. First, they were asked, "Have you ever tried smoking cigarettes or chewing tobacco?" Second, if children reported trying beer, wine coolers, wine, and hard liquor (queried separately), they were asked for each endorsed alcohol-type follow-up questions regarding frequency and volume-of-use. The interviewer then determined, "Has the target child *ever* had one whole alcoholic drink (not just sips)?" Third, children were asked, "Have you ever tried marijuana or pot?" Responses were coded 1 for each substance type to which they answered yes at any of the three waves and 0 if all reports were no. The final score was a count ranging from 0-3 of the number of the three substances ever used at any assessment.

2.3.6.   Father–child contact.—At each 3GS wave, parents each reported on the timing of when children lived with parents and parent figures since birth, as well as on joint-custody (50:50 joint-custody), with changes recorded at the level of month/year. Averaging each parents' reports, the percentages of mother–child ($M[SD]$ =.87[.19]) and father–child ($M[SD]$ = .72[.30]) contact were calculated from estimates of the total number of days each child lived with the parent from birth to the latest available wave through the age 13–14 wave. Estimates were available for 198 fathers and 204 mothers. Due to nonindependence and lower variability in mother–child contact, only father–child contact was analyzed.

2.3.7.   Control variables.—*Child gender* and *Child age at last assessment* were controlled because we expected that children who were too young to have completed the age 13–14 wave to be less likely to have used each substance, and children who were older than a targeted wave age were expected to be more likely. *Father age* at child's birth was controlled, as children born to younger parents were at increased risk for early substance use.

2.4. Data Analysis

Models used poisson regressions to predict child ATM use as a count variable from maternal and paternal behaviors (separately by parent). Dependence among siblings' scores was accounted for using a sandwich estimator using the *TYPE=COMPLEX* option in MPlus (Muthén & Muthén, 1998–2017). A series of models were tested separately for mothers and fathers. Model 0 included child gender and age, and father age. Model 1 tested associations that parental POM and POU had with child ATM use and the extent to which such associations persisted when controlling for parents' ATM use. Model 2 examined the extent to which associations in Model 1 persisted when controlling for parents' psychopathology. Model 3 tested the full model with all predictors. Model 4 (fathers only) examined

moderation of effects of father substance use on child ATM use by father–child contact; that is, whether the prediction of child ATM use from paternal POM and ATM use differed for fathers who had more or less contact with their child.

3. Results

3.1. Descriptive Statistics

Descriptive statistics on unstandardized predictor variables are presented in Table 1, based on children as the focal participant, and do not represent that children are clustered in families. Children's mothers and fathers reported POU at 27% and 18% of measurement occasions, and POM at 4% and 6% of measurement occasions, respectively. On at least one measurement occasion, 67.4% and 67.1% of children's mothers and fathers reported POU, and 14.0% and 27.3% reported POM. Father–child contact expressed as percentages of the child's life were <25% ($n = 20$, 10.1% of sample), 25-49% ($n = 33$, 16.7%), 50-74% ($n = 35$, 17.7%), or 75-100% ($n = 110$, 55.6%); father-contact was 100% for 58 children (29.3%). Children used only one ($n = 38$, 17.6%), only two ($n = 13$, 6.0%), all three ($n = 7$, 3.2%), or none ($n = 158$, 73.2%) of the three ATM substances.

3.2. Poisson Regression Models: Predicting Child ATM Use

3.2.1. Maternal models.—Results of Model 1 (Table 2) indicate that mothers' POU was positively associated with child ATM use; whereas contrary to predictions, mothers' POM was not. As expected, older child age and younger father age at child's birth were associated with children's ATM use. Subsequent models indicated that the effect of mothers' POU remained significant after adjustment for the significant positive association between mothers' and children's ATM use (Model 1), and for mothers' depressive symptoms (nonsignificant) and antisocial behavior (significant; Models 2 and 3).

3.2.2. Paternal models.—Results for Model 1 (Table 3) support the hypothesis that fathers' POM, but not fathers' POU, was significantly positively associated with child ATM use (again, accounting for control variables). In Model 1, the effect of fathers' POM was no longer significant when fathers' ATM use was controlled (although fathers' ATM was not significant). In Model Set 2, neither fathers' depressive symptoms or delinquency appeared to account for the association between fathers' POM and child ATM use. Lastly, in Model 3, neither fathers' POM or ATM use were associated with child ATM use when fathers' depressive symptoms and delinquency were controlled—neither of which were significantly associated with child ATM use.

3.2.3. Moderation of fathers' substance use effects by father–child contact.—In the final model, the interaction of father–child contact and father ATM use predicted child ATM use, whereas the interaction involving father POM did not (Table 3, Model 4). When father–child contact was high (e.g., 1 SD above the mean), fathers who had used more ATM conferred greater risk for child ATM use compared to fathers who had used less ATM (e.g., yielding a change in the predicted values of .54 and −.62, assuming father ATM use was 1 SD above and below the mean, respectively). When father contact was low (e.g., 1 SD below the mean), there were little to no differences in conferred risk for child ATM use given

fathers' ATM use was 1 *SD* above and 1 *SD* below average (e.g., yielding a change in the predicted values of .12 and −.04, respectively).

4. Discussion

Mothers' use and fathers' misuse of prescription opioids were linked to their children's ATM use by early adolescence. Parents' POU, POM, and ATM use were averaged across repeated assessments across their children's development, and children's ATM use was measured through early adolescence. The study spanned critical-developmental periods during which exposure, influence, experimentation, and early onset occur. Few prior studies have considered a community rather than clinical sample of parents (including robust participation by resident and non-resident fathers) using prescription opioids, or have examined parental use of a specific substance other than ATM, as a risk factor for children's substance use. A further contribution of this study was the application to POM of theoretical models developed to study various forms of substance use. This is important, as prescription opioid abuse can begin very differently—for example, through a painful physical injury, legitimate prescribed use, and an escalation to abuse (Cicero et al., 2014). Thus it was not clear that similar models of familial risk for ATM use would be relevant to POU and POM. However, many of the findings were consistent with parent–child risk-transmission processes that have been described for other substances.

Support for the *general problem-risk* pathway linking parent behavior to child substance use was inconsistent. Mothers' delinquency was associated with child ATM use, but did not account for the associations that either mothers' ATM use or POU had with child outcomes. Fathers' delinquency during their children's lives was univariately associated with child ATM use, but did not explain the links between fathers' POM and ATM use of their offspring. Next, and consistent with a *general substance-use-risk* pathway, fathers' POM was not uniquely associated with child ATM use once paternal ATM use was controlled. Notable was that a general substance-use-risk pathway was evident for fathers who had more frequent or longer-term contact with their children (i.e., paternal ATM use was a significant predictor), whereas fathers' and children's ATM use were not associated when fathers were more absent from their children's lives. Similar patterns have been observed for intergenerational transmission of antisocial behavior (Thornberry et al., 2009). Links between maternal and child substance use went beyond a general substance-use-risk pathway, as maternal ATM use was associated with that of their children, and maternal POU contributed further to children's increased risk. Finally, there was no evidence for unique risks conferred from parental POM as neither fathers' or mothers' POM were associated with children's ATM use after controlling for more general risk pathways.

Parents' POU was controlled to rule-out the possibility that unmeasured factors (e.g., chronic pain) that are associated with POU might account for apparent effects of POM on child substance use risk. Indeed, POU did not explain the negative association between fathers' POM and child ATM use. Surprisingly, however, mothers' POU predicted child ATM use, even after accounting for mothers' delinquency and other substance use (including POM), as well as nonsignificant effects of depressive symptoms. A recent large study found maternal POM was more strongly related to adolescents' POM than was paternal POM after

accounting for multiple parent (including depression and other alcohol, tobacco, and other drugs use) and adolescent characteristics; however, maternal POU and antisocial behavior were not controlled in that report (Griesler et al., 2019). Potential mechanisms of the present finding that maternal POU was independently associated with child ATM use may include effects of debilitating pain or opioids' psychoactive properties on parenting, or transmission of attitudes and beliefs about prescription drug use that generalize to other substances (Chassin et al., 2016). The unexpected effect of maternal POU requires replication but may provide further reason to promote more cautious opioid prescription guidelines (see Dowell et al., 2016; Shah et al., 2017).

4.1. Limitations

The present study was not originally designed to assess POU and POM or abuse. One limitation is that neither the reasons for the initial prescription (e.g., surgery) nor experience of physical pain were assessed. Parental POM to relieve chronic pain versus to experience the drug's psychoactive effects may have different consequences for children. Second, there was not sufficient variability to consider frequency of POU and POM or opioid use disorder, or to consider longitudinal or developmentally specific predictions (e.g., effects of POM during children's early childhood versus adolescence). It seems unlikely that child ATM use played any causal role in parental substance use. Still, we cannot rule out alternative temporal sequences (e.g., maternal depression at child age 7 influences both child alcohol use at age 10 and maternal POU at child age 11 years). Third, prevalence of child substance use was too low at this stage of the study to evaluate prediction of serious use patterns. Finally, the demographic characteristics of the sample that make it highly relevant to questions about POM (i.e., predominantly White and lower-SES) also potentially limit generalizability.

4.2. Conclusions and Prevention Implications

Findings indicate that fathers' POM and mothers' POU are associated with child ATM use by early adolescence. Intergenerational associations involving fathers' POM were better explained by their more general substance use, and these latter effects were stronger for fathers who had more residential contact with their children than for those with less contact. One implication is that alternative pain prescriptions or prevention of parent POU and ATM use may have secondary prevention effects on ATM use for children. Unexpectedly, findings also hinted that POU, and not just POM, may have detrimental effects on child ATM use. Overall, models yielded robust prediction of child ATM use from maternal risk factors, including maternal general substance use and delinquency. Thus, findings from this sample of predominantly lower-SES, White families suggest mothers should be a major focus for prevention of the intergenerational transmission of substance use.

Acknowledgements

Abbreviations:

(ATM)	alcohol, tobacco, and marijuana
(POM)	prescription opioid misuse
(POU)	prescription opioid use

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Highlights

- Fathers' prescription opioid misuse was associated with children's substance use by age 14 years

- Mothers' prescription opioid use but not misuse was correlated with child substance use

- Risk from mothers' opioid use was not explained by mothers' more general behavioral risks

- Risk transmission was stronger with higher father–child residential contact

Table 1.

Descriptive statistics for study variables

	Mean (*SD*) or %	Range
Mothers		
POU	.27 (.27)	.00 - 1.00
POM	.04 (.12)	.00 - 1.00
ATM		
Alcohol	1.20 (.64)	.00 - 2.80
Tobacco	.66 (.78)	.00 - 2.00
Marijuana	.38 (.88)	.00 - 4.34
Delinquency	.63 (.75)	.00 - 4.00
Depressive symptoms	11.20 (7.72)	.00 - 40.00
Age at child's birth	23.13 (4.29)	14.97 - 36.95
Fathers		
POU	.18 (.20)	.00 - .91
POM	.06 (.15)	.00 - .80
ATM		
Alcohol	1.86 (.79)	.00 - 3.20
Tobacco	1.05 (.84)	.00 - 2.00
Marijuana	.47 (.70)	.00 - 2.48
Delinquency	.72 (.81)	.00 - 4.77
Depressive symptoms	8.56 (5.99)	.22 - 30.60
Age at child's birth	24.38 (3.89)	16.74 - 32.47
Father–child contact	.72 (.30)	.00 - 1.00
Children		
ATM use count	.39 (.75)	0 - 3
Any ATM use (yes/no)	27%	0 - 1
Alcohol	14%	0 - 1
Tobacco	14%	0 - 1
Marijuana	11%	0 - 1
Female	56%	0 - 1
Age at last assessment	13.82 (1.00)	11.32 - 16.68

Notes. ATM = alcohol, tobacco, and marijuana use; POM = prescription opioid misuse; POU = prescription opioid use. Mean (*SD*) = mean (standard deviation) across assessment occasions.

Table 2.

Poisson regressions predicting children's ATM use from maternal behaviors

| | Model 0 | Model Set 1 | | Model Set 2 | | Model 3 |
| | Controls | POU/POM | POU/POM + ATM | POU/POM + depressive | POU/POM + delinquency | All predictors |
	B (SE)	B (SE)	B (SE)	B (SE)	B (SE)	B (SE)
Child is male	.50(.26)†	.44(.26)†	.32 (.25)	.44(.26)†	.26(.26)	.24(.26)
Child age	.36(.15)*	.35(.15)*	.38(.15)*	.34(.15)*	.39(.13)**	.38(.14)**
Father's age	−.36(.16)*	−.38(.16)*	−.33(.16)*	−.37(.16)*	−.26(.15)†	−.28(.15)†
POU		.32(.11)**	.31(.11)**	.30(.12)*	.32(.11)**	.31(.11)**
POM		.09(.13)	−.06(.13)	.08(.14)	−.02(.13)	−.09(.14)
ATM			.41(.10)***			.30(.11)**
Depressive				.07(.10)		.004(.11)
Delinquency					.34(.06)***	.20(.09)*

Notes.

†
p < .10.

*
p < .05.

**
p < .01.

***
p < .001. ATM = alcohol, tobacco, and marijuana use; POM = prescription opioid misuse; POU = prescription opioid use. *B (SE)* = Regression estimate (standard error).

Table 3.

Poisson regressions predicting children's ATM use from paternal behaviors and father–child contact

	Model 0	Model Set 1		Model Set 2		Model 3	Model 4
	Controls	POU/POM	POU/POM + ATM	POU/POM + depressive	POU/POM + delinquency	All predictors	Moderation by father contact
	B (SE)	*B (SE)*	*B (SE)*	*B (SE)*	*B (SE)*	*B (SE)*	*B (SE)*
Child is male	.50 (.26)†	.53 (.27)†	.51 (.28)†	.54 (.27)*	.53 (.29)†	.53 (.28)†	.56 (.31)†
Child age	.36(.15)*	.36(.15)*	.37(.15)*	.37(.15)*	.36(.15)*	.37(.15)*	.38 (.15)*
Father's age	−.36(.16)*	−.35(.16)*	−.27(.15)†	−.33(.16)*	−.34(.16)*	−.30(.16)†	−.24(.17)
POU	.	.04(.15)	−.01(.16)	.001(.14)	.03(.16)	−.01(.16)	−.08 (.18)
POM	.	.23(.09)**	.10(.14)	.24(.09)**	.22(.11)*	.13(.16)	.10 (.14)
ATM	.	.	.30 (.16)†	.	.	.29 (.16)†	.33 (.17)*
Depressive	.	.	.	.16 (.11)	.	.08 (.11)	.
Delinquency	.	.	.	.	.02 (.14)	−.06 (.14)	.
Father contact	.	.	.	.	.	.	−.04 (.13)
POM x Father contact	.	.	.	.	.	.	.09 (.10)
ATM x Father contact	.	.	.	.	.	.	.25 (.11)*

Notes.

† *p* < .10.

* *p* < .05.

** *p* < .01.

*** *p* < .001. ATM = alcohol, tobacco, and marijuana use; POM = prescription opioid misuse; POU = prescription opioid use; Father age = father's age at the birth of the target child. *B (SE)* = beta (standard error).