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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.

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

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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 has focused on 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.

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.

2.3.1. 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.