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Intergenerational Congruence in Adolescent Onset of Alcohol, Tobacco, and Marijuana Use

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Abstract

We examined alcohol, tobacco, and marijuana (ATM) use onset across early to late adolescence in a sample of fathers and their offspring. We tested a theory of developmental congruence in polysubstance use, or the extent to which fathers' ATM use onset in early adolescence increased risk for earlier ATM onset by their offspring. Average rates of adolescent ATM use onset were also compared across generations, which may reflect intergenerational discontinuity and secular trends. Children (n = 223, 44% boys) and their fathers (n = 113; originally recruited as boys at neighborhood risk for delinquency) contributed repeated prospective self-reports of their ATM use across adolescence (as late as age 18 years). Mothers' ATM use (retrospective) through age 18 years was available for 205 children. Data were analyzed using discrete-time survival mixture analysis. Compared with their fathers, boys and girls showed later onset for tobacco use, and girls showed later onset alcohol use. Developmental congruence was partially supported: children showed earlier ATM use onset if their fathers were assigned to the early adolescent polysubstance use onset class, compared to the late-adolescent predominantly alcohol and tobacco onset class; mothers' ATM use in adolescence attenuated this effect. Consistent with national secular trends, rates of adolescent onset tobacco and alcohol use declined across generations, whereas marijuana use onset did not. However, there was intergenerational transmission of risk for early polysubstance use onset. Prevention that delays early substance use may have early lifespan effects as well as transgenerational implications.

Keywords

adolescence; intergenerational; marijuana; polysubstance use; discrete-time survival mixture analysis

Several prospective intergenerational studies support that parents' substance use during their adolescence predicts their children's substance use decades later (e.g., Henry, Fulco, Agbeke, & Ratcliffe, 2018; Hill, Sternberg, Suk, Meier, & Chassin, 2018; Kerr, Capaldi, Pears, & Owen, 2012). A clear implication is that prevention could begin long before children are even conceived by altering the early developmental pathways of future parents (Cheng, Johnson, & Goodman, 2016; Patton et al., 2018). Intergenerational associations in substance use are relatively modest in magnitude, however. One explanation for small effects

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is that intergenerational transmission pathways may be indirect and subject to considerable moderation, or stronger for some forms of substance use than for others (e.g., Hill et al., 2018; Nadel & Thornberry, 2017). For example, two studies found that parental cannabis use disorder was more strongly linked with substance use in the next generation than were other measures of parents' cannabis use (Henry & Augustyn, 2017; Hill et al., 2018). In order to inform prevention, researchers should clarify which forms or patterns of substance use are most strongly transmitted from one generation to the next. Thus, in the present study we examine whether two higher risk features of substance use in adolescence that have been less considered—the use of multiple substances and earlier onset—show *congruence* across generations. We do so in a sample of fathers assessed prospectively from late childhood to middle adulthood, their offspring, as well as mothers who retrospectively reported on their own ATM use during adolescence.

Onset and rapid growth of alcohol, tobacco, and marijuana (ATM) use are common during adolescence (Duncan, Duncan, & Strycker, 2006), and earlier onset portends heavier substance use, use of illicit drugs, and substance use disorders in early adulthood (e.g., Moss, Chen, & Yi, 2014). Examining use of a particular substance can be informative regarding risk. For example, earlier onset alcohol use predicts a more problematic drinking trajectory through age 31 years (Warner, White, & Johnson, 2007). Yet studies of a single substance do not take into account the fact that polysubstance use is common among adolescents (Leatherdale, Hammond, & Ahmed, 2009; Tomczyk, Isensee, & Hanewinkel, 2016). For example, using AddHealth data on ATM use, Moss and colleagues found that many 16-year olds had used alcohol (52%), marijuana (43%), or cigarettes (defined as regular use; 29%)—however, only a small proportion of youth had used *only* that substance (approximately 14%, 6%, and 4%, respectively), whereas 40% had used two or all three substances. Furthermore, onset of one substance increases onset risk for another (Kosterman, Hawkins, Guo, Catalano, & Abbott, 2000), and early polysubstance use generally is a stronger predictor of later (young adult) substance use disorder and other problem outcomes than is early use of a specific substance (Moss et al., 2014). Given the serious long-term consequences of substance abuse (Bossong & Niesink, 2010; Broman, 2009; Moore, Florsheim, & Butner, 2007) and the recognition that early adolescence may be a particularly sensitive time for prevention (Tucker, Ellickson, Orlando, Martino, & Klein 2005), a better understanding of risk for *early onset polysubstance use* is needed.

Parents' substance use is a likely risk factor for early polysubstance use by offspring, given its well-established associations with adolescents' substance use, such as smoking initiation and alcohol experimentation (Gilman et al., 2009; Kerr et al., 2012). However, studies of whether parents' use of a substance predicts adolescents' use of the same substance may not actually illuminate any process that is substance specific. Genetic and environmental influences on substance use and dependence overlap considerably across drug classes and with the broader class of problem behaviors (Dishion & Patterson, 2006; Hicks, Krueger, Iacono, McGue, & Patrick, 2004; Hoffman, & Maynard, 2008; Kendler et al., 2012). Therefore, it may be meaningful to examine parents' substance use in relation to their children's use of *multiple* substances, which too few studies have done. For example, Tomczyk and colleagues (2016) found that only 2 of 23 studies in a systematic review of

latent class analyses of adolescents' polysubstance use examined parental substance use as predictors.

Just as adolescents' polysubstance use has been insufficiently considered, so too has the risk factor of parents' polysubstance use, which is also more common than is use of a single substance (Cohn et al., 2018). A limited number of studies have demonstrated that parental use of multiple substances during their children's lives make unique contributions to adolescent substance use onset. Mothers' use of alcohol and tobacco additively increased and fathers' use of alcohol and marijuana multiplicatively increased risk for earlier alcohol use onset in their offspring (Capaldi, Tiberio, Kerr, & Pears, 2016). Similarly, Bailey, Hill, Oesterle, and Hawkins (2006) found that a latent score of parental substance use (binge drinking, tobacco, and marijuana use) when children were aged 13–14 years explained most of the variance in a similar latent score of offspring substance use at ages 15–18 years. Focusing on more serious problems, however, Henry (2017) found that when fathers' lifetime histories of alcohol and cannabis use disorders were considered simultaneously, only the latter was associated with their children's early alcohol or cannabis use.

Prospective intergenerational studies that follow parents through their own adolescence, as well as that of their offspring, can contribute knowledge in this area in ways that traditional longitudinal studies of parents' and children's substance use cannot. First, most parents of adolescents are beyond the peak years of onset of use and dependence (Vega et al., 2002; Wagner & Anthony 2002) and of heavier use of marijuana and alcohol (Schulenberg et al., 2018). Thus, if parents are assessed in adulthood, aspects of their substance use histories are not observed. In contrast, intergenerational studies can reliably determine whether parents' adolescent histories of early onset and polysubstance use present risks to children's adjustment. Relatedly, intergenerational studies trace the implications of teenagers' early onset polysubstance use not just for themselves but for their future offspring. This perspective may inspire a broader view on prevention opportunities and, in particular, those that are intergenerational in scope (Cheng et al., 2016), rather than focused solely on parent substance use and other behaviors during a child's life. Third, only prospective intergenerational designs allow examination of continuities or similarities in the substance use trajectories of parents and their children across the same developmental periods.

To conceptualize intergenerational associations, Capaldi, Kerr, and Tiberio (2017) posited a theory of *developmental congruence*. The term is adapted from geometry, where congruence refers to figures of a similar shape and size. Developmental congruence refers to the expectation that ages at initiation of ATM use and patterns of growth across development will show similarity across generations, given intergenerational continuities in social risk contexts and similarities in individual vulnerabilities. It is based on the theory that child factors (e.g., temperament), some of which are heritable, increase risk for problem behaviors when they are expressed at sensitive developmental periods (Witt, 2010). Thus, the timing of these developmentally related manifestations (e.g., susceptibility to deviant peer influences) is expected to be similar across generations within families. Such similarity is made more likely by continuities within and across generations in social risk context (Capaldi, Pears, Kerr, Owen, & Kim, 2012; Gavin, Hill, Hawkins, & Maas, 2011; Lipman, Georgiades, & Boyle, 2011; Scaramella, Neppl, Ontai, & Conger, 2008; Schofield et al., 2011; Thornberry,

Freeman-Gallant, & Lovegrove, 2009) and family mechanisms such as parenting (Kerr & Capaldi, 2019). Furthermore, parents' expectations regarding substance use during adolescence may guide monitoring and socialization processes that delay or hasten child onset. As parents' expectations are likely to be influenced by their own histories of adolescent substance use, intergenerational transmission would be predicted.

Although developmental congruence is predicted, the ages of ATM use initiation may change over generations, possibly reflecting shifts in secular trends. For the present sample of fathers, 2–3 decades have passed between their adolescent years (late 1980s to early 1990s) and those of their children. During this time, contextual factors affecting ages of onset of substance use have changed, including decreased availability of tobacco to youth because of increased restrictions on sales (Forster et al., 1998), increasingly liberal attitudes toward marijuana use (Pacula, 2010), and legalization policies (Johnson & Guttmannova, 2019). Indeed, there is considerable evidence of changes in age of ATM use onset over the past 25 years. Findings from Monitoring the Future (Miech et al., 2019) indicate there have been declines in 30-day use rates of cigarettes among 8–12th graders, from approximately 14–28% in 1991 to 2–8% in 2018. For alcohol use, 11–30% of 8–12th graders in 1991 reported having had five or more drinks in a row at least once in the past 2 weeks, compared to 4–14% in 2018. In contrast, 6%, 17%, and 24% of 8th, 10th, and 12th graders, respectively, reported marijuana use in the last 12 months in 1991, compared to about 10%, 28%, and 36% in 2018. Furthermore, marijuana is increasingly the first substance used by adolescents in the sequence of adolescent substance use onset (Keyes, Rutherford, & Miech, 2019). Thus, given recent trends, children were expected to show later ages of onset for alcohol and tobacco use compared to their fathers, but earlier ages of onset for marijuana use.

Research Questions

The present study focused on father-child similarities in polysubstance use onset, given that fathers' onset histories were assessed prospectively beginning in late childhood. During adulthood, mothers contributed retrospective data on their ATM use in adolescence and this variable was used in some analyses.

We evaluated the first study question regarding intergenerational changes in the timing of first substance use by comparing average rates of father's and offspring's onset during adolescence. The offspring generation was posited to show later ages of onset for tobacco and alcohol and earlier ages of onset for marijuana than did their fathers. In addition, it was predicted that substance use onset would be earlier among boys relative to girls (Capaldi et al., 2016; Kerr, Tiberio, & Capaldi 2015; Trim, Schuckit, & Smith, 2010), and thus that there would be more pronounced differences in onset timing between fathers and daughters than between fathers and sons.

Second, the theory of developmental congruence in polysubstance use onset across generations was tested. Fathers' earlier ATM use onset during adolescence was expected to predict risk for earlier ATM use onset in their offspring. Of note, most studies consider only onset of a single subtance (e.g., first tobacco use), and onset typically can only be considered as an outcome not as a predictor. Thus, to relate three simultaneous onset processes between

two generations, a mixture component was added to study models, therefore enabling a unified test of intergenerational congruence in polysubstance use onset. Finally, it was expected that men with histories of early onset ATM use would father children with women who reported adolescent onset ATM use (i.e., assortative mating). Furthermore, whether mothers' ATM use histories would be uniquely associated with their children's ATM use onset risk, and perhaps attenuate father-child congruence, was examined.

Method

Participants

Participants were 223 children, 154 fathers, and 205 mothers. Fathers were originally recruited as boys ages 9–10 years from schools in neighborhoods with higher-than-average rates of delinquency, as part of the Oregon Youth Study (OYS). The OYS sample was boys only, but was otherwise representative of the area at the time; 90% were White, and most were from families of low socioeconomic status (Capaldi & Patterson, 1989; Hollingshead, 1975). High retention of an at-risk sample is a strong feature of the OYS. Of the 206 OYS men, 6 have died, and the participation rate for the remaining 200 men in the OYS was 88% at the last wave completed.

Men who became fathers were invited to participate in the OYS–Three Generational Study (3GS) with the first two biological children they had per female partner (initially all of their children were invited). Participation by invited (i.e., located and eligible) children was 98% with 90% retention over time. The 3GS sample has grown as children were born and matured, and the ages of the maturing children determines the *n* of children, fathers, and mothers available for analysis at each wave. As of September, 2018, 223 children (44% boys, $n = 99$) were old enough to have participated in at least one assessment of substance use, the focus of the present analyses. The sample size available for each adolescent age category (shown in Table 1) ranged from approximately 90% of the sample at ages 11–12 years to just less than one half of the sample at ages 17–18 years.

Procedures

The OYS and 3GS were approved by the Oregon Social Learning Center's Internal Review Board. Following parental consent and youth assent, assessments were conducted involving phone or in-person interviews and questionnaires.

Measures

Offspring's and fathers' ATM use onset before age 19 years.—Alcohol, tobacco, and marijuana use onset were determined from self-report questions. Adolescents in both generations were asked at each assessment whether they had ever used tobacco (cigarettes, pipes, chewing tobacco) or marijuana and, if so, the age at which they had first used each substance. They also indicated at each assessment whether they had used alcohol, and if so the age of their first use, and whether they had ever consumed a whole drink. Although sips and tastes of alcohol generally are predictive of future use, these behaviors show a high prevalence in childhood and typically are not followed closely in time with further use that would indicate a significant marker of onset (Grant, 1998). We instead focused on onset of

first whole drink, which is considered a stronger indicator of onset. Alcohol onset timing was set as the adolescent's age at the assessment they first reported they had consumed a whole drink

Offspring were asked biannually at targeted assessment ages of 9–10, 11–12, 13–14, 15–16, and 17–18 years about ATM use. Note that the age 9–10 year assessment was closed midway through the study for budgetary reasons. Given the onset definitions for tobacco and marijuana (age at first use) versus alcohol (age when first whole drink was reported), this wave closure affected sample sizes for alcohol only; ultimately, however, alcohol onset was so unusual at the early assessments that it appears unlikely that it was misspecified for any case.

Fathers were asked annually from ages 9–10 to 18–19 years about their ATM use in the past year, and this information was collapsed into biannual time periods to match the 3GS-assessment design. Specifically, fathers' onset was coded as prior to age 11 years if they reported use of a given substance use at their age 9–10-year or 10–11-year assessment and were confirmed younger than age 11 years at the time, or if they reported such timing on the lifetime-use questions at the first assessment they reported use. From the subsequent assessments of fathers, new reports of ATM use were used to define onset during each period (i.e., ages 11–12, 13–14, 15–16, and 17–18 years). As the timing of the offspring's and fathers' assessments varied by person, participants' exact ages were used to determine whether or not onset had occurred within each discrete-time category.

Thus, for tests of differences in secular trends in adolescent ATM use onset across generations, five binary variables were created for offspring and fathers for each substance at each of the following five age categories: prior to 11, 11–12, 13–14, 15–16, and 17–18 years. Once an individual had onset, all of his or her subsequent scores at older ages for that particular substance were set to missing values (Muthén & Masyn, 2005). Thus, only those adolescents who remained at risk for onset were included at each time period of adolescence. Likewise, onset variables for offspring who were not old enough to reach the later assessments were set to missing at these later waves.

Mothers' ATM use onset.—Mothers reported retrospectively on their ages of onset of *any* use of alcohol (whole drink was not queried), tobacco, and marijuana. Mothers' adolescent ATM use onset was defined as the number of substances (zero to three) the mother reported using before age 19 years.

Analytic Approach

In all models that included offspring data, standard errors were estimated using a sandwich estimator in Mplus version 8.3 (Muthén, & Muthén, 1998–2017) to account for the dependence among siblings' scores. As background for tests of our research questions nine, discrete-time survival analysis (DTSA; Muthén & Masyn, 2005) models were fit to separately describe fathers', boys', and girls' onset on each of the three substance. Paternal models included no predictors (i.e., unconditional) and the offspring models included the design effect of child's age at his or her last wave of participation to account for right

censoring. Models allowed for fixed effects only (i.e., no random frailty effects) and assumed proportional odds (i.e., effects were fixed to be equal across adolescence).

To evaluate the first research question regarding intergenerational changes in onset, DTSA models were fit simultaneously for fathers and boys, and then again for fathers and girls, separately for each substance (six models total). Differences in mean ATM use onset rates across generations were tested by comparing a null model that assumed equal onset rates to an alternative model that assumed unequal onset rates, and then assessing change in overall model fit via the Wald statistic. This approach is akin to the log-rank test. Retrospective data on mothers' ATM use histories were not examined in relation to this question, as the measurement approach was not appropriate for use in DTSA models.

In preparation for tests of the second set of research questions regarding intergenerational congruence in polysubstance use onset, ages of onset of *multiple substances* were modeled by simultaneously fitting alcohol, tobacco, and marijuana use onset as three discrete-time processes across the five discrete-time periods of adolescence. Specifically, discrete-time multiple event survival process survival mixture (Dean, Bauer, & Shannon, 2014) models were separately estimated for each generation. These models account for dependence among multiple, non-repeatable discrete-time events by modeling heterogeneity in the timing of such events using latent classes. The addition of latent classes allows for tests of theoretical models that posit distinct populations of individuals who differ according to various traits—such as adolescents who are (versus those who are not) at heightened risk for earlier ATM use onset. These subpopulations can then be predicted from posited risk factors—in the present case, their fathers' earlier ATM use onset. The number of classes necessary to most adequately and parsimoniously summarize the variance in adolescent ATM use onset rates was examined using information criteria and the Lo-Mendell-Rubin likelihood ratio test (Lo, Mendell, & Rubin, 2001), as well as considering the interpretability of the classes. For the best-fitting models, fathers' and children's predicted ATM use class memberships (based on their most likely assignments), and the classification uncertainty in assignments were saved for use in the final analyses. For the offspring generation, child gender and child's age at his or her last assessment of participation (to account for right censoring due to developmental age) were included as predictors of class membership.

The second research question regarding developmental congruence in ATM use onset was examined. This was tested by predicting children's ATM use onset classes from their fathers' ATM use onset classes while still accounting for uncertainty in class assignments using a manual three-step approach (Asparouhov & Muthén, 2013). Thus, fathers' and children's probabilities of being in each ATM use onset class were used in the analyses, rather than simply the observed categorical variables determined from their most likely class assignments. Models also controlled for child gender and the design effect of child's age at his/her last participation.

Finally, we examined how fathers' and mothers' (retrospective) histories of earlier onset substance use were associated, and whether any paternal intergenerational effects were attenuated after adding mothers' reports of ATM use onset as a predictor of child onset class.

Results

Descriptive Statistics and Design Effects

Substance use onset rates are listed in Table 1. Onset rate is different from prevalence[1], and corresponds to the proportion of adolescents who showed onset by the end of an age period who were still at risk of onset at the start of that period. In general, offspring risk for onset increased with age, as reflected in the increasing rates of alcohol, tobacco, and marijuana use onset across adolescence, equaling 2%, 5%, and 6%, respectively, at ages 11–12 years and increasing to 47%, 38%, and 28%, respectively, at ages 17–18 years. For fathers, rates of ATM use onset varied more across adolescence than those of their children. A substantial number of fathers reported onset on ATM use prior to age 11 years. From ages 11–12 years onward, fathers' rates of substance use onset generally increased or remained constant.

The mothers' retrospective reports of substance use indicated the following patterns of use: 59.6% (n = 133) had used all three substances before age 19, 4.5% (n = 10) used only tobacco, 11.2% (n = 25) used only alcohol, 13.5% (n = 30) used tobacco and alcohol only, and 3.1% (n = 7) used alcohol and marijuana but not tobacco; six cases (2.7%) were missing.

Offspring were 16.54 years of age, on average, at their last wave of participation, and right-censoring due to age could influence our models, results, and interpretation. We thus partitioned variance due to this type of missingness by including child's age at his or her last wave of participation as a predictor of ATM use onset, which was indeed a significant predictor of offspring tobacco, alcohol, and marijuana use onset ($b[se]$ = 17[.06], p = .003; $b[se]$ = 18[.08], p = .029; $b[se]$ = 14[.07], p = .040, respectively). Thus, the models controlled for the design effects that offspring who were old enough to have participated at the later waves were more likely to have reported using ATM earlier in adolescence.

Changes in Adolescent ATM Use Onset Across Generations

The estimated baseline survival curves from the nine separate DTSA models for fathers', sons', and daughters' adolescent ATM use onset are depicted in Figure 1, and the tests of intergenerational differences in adolescent ATM use onset are shown in Table 2. Results supported predictions that, on average, children showed onset of alcohol and tobacco use at significantly older ages than did their fathers (though the father-son comparison was not significant for alcohol; p = .055). Contrary to the study hypothesis, onset of marijuana use did not significantly differ across generations.

Heterogeneity in Adolescent Onset Classes

Next, we examined whether fathers and children exhibited significant heterogeneity in ages of adolescent ATM use onset. For children, a two-class solution denoting an earlier and later ATM use onset class was chosen to have fit the data best (Table 3, Panel 1; and Figure 2). Note that a three-class solution indicated by the Lo-Mendell-Rubin test was untenable because gender was confounded with the earliest ATM use onset class (all were boys), this

[1]Cumulative prevalence by age period can be visualized in Figure 1, which is based on subsequent analyses.

class size was small (n = 14), and the model fixed approximately 50 parameters. Thus, we pursued the two-class solution. For the *early adolescent polysubstance use* onset class (n = 95, 42.6% of children), essentially all children (97.2%) were estimated to have onset on tobacco, and most were estimated to have onset on alcohol (87.6%) and marijuana (89.7%) use by the end of adolescence (ages 17–18 years). In contrast, in the *late-adolescence polysubstance use* onset class (n = 128, 57.4% of children), many fewer youth had shown onset for tobacco, alcohol, and marijuana by ages 17–18 years (13.9%, 31.9%, and 20.2%, respectively). Regarding the control variables, children who had participated at the older assessments were significantly more likely to be assigned to the early adolescent compared to the late-adolescent onset class ($b[se]$ = .39[.10], p < .001). There was not support for a gender difference in the assignment to the earlier versus later onset class ($b[se]$ = .79[.46], p = .089). However, child gender was retained as a control variable when testing the associations between father's and children's onset classes, given that many other studies suggest that boys show earlier onset of ATM use than girls (e.g., Trim et al., 2010).

For fathers, a three-class solution was chosen to fit the data best (Table 3, Panel 2; and Figure 3). Note that although the Lo-Mendell-Rubin test indicated a four-class solution, class sizes were quite small (ranging from n = 23 to 36), and many of the parameters (i.e., thresholds) had to be fixed in the model due to all of the fathers having onset on many of the substances prior to the end of adolescence. In the *early adolescent polysubstance use* onset class (n = 43, 38.1% of fathers), essentially all adolescents had used alcohol (94.7%) and tobacco (97.4%) by ages 15–16 years, and 92.1% of adolescents had used marijuana by the end of adolescence (ages 17–18 years). In the *late-adolescent polysubstance use* onset class (n = 23, 20.4% of fathers), 45.0% and 70.5% of the adolescents had used alcohol and tobacco, respectively, by ages 15–16 years; whereas no adolescents had used marijuana by ages 15–16 years. By the end of adolescence (ages 17–18 years), all adolescents had used alcohol and tobacco, and 90.0% had used marijuana. Finally, for the *latest predominantly alcohol and tobacco use* onset class (n = 47, 41.6%) the majority of fathers were estimated to have onset on tobacco (61.2%) and alcohol (80.9%) whereas marijuana use onset was estimated to have occurred for only 22.4% of fathers by the end of adolescence.

Associations of Mothers' and Fathers' Adolescent Polysubstance Use

Next, the number of substances mothers used as adolescents (indicating use of alcohol, tobacco, and/or marijuana) was included as a predictor of the fathers' adolescent ATM use onset classes. The R3STEP procedure was used in Mplus version 8 (Muthén & Muthén, 1998–2017) to account for uncertainty in fathers' adolescent ATM use onset classes. Of note, using the R3STEP procedure ensured that the fathers' classes were estimated prior to including mothers' adolescent ATM use as a predictor (i.e., mothers' ATM use did not influence class solution). The model indicated that mothers' adolescent ATM use was a significant predictor of fathers' being in the early adolescent and late-adolescent polysubstance use classes compared to the latest predominantly alcohol and tobacco use onset class ($b[se]$ = 1.42[.43], p = .001 and $b[se]$ = .80[.33], p = .015, respectively).

Developmental Congruence in Adolescent Polysubstance Use Onset

To test the theory of developmental congruence across fathers and children in adolescent ATM use onset, children's onset class was predicted from their fathers' class, again accounting for classification uncertainty and controlling for offspring age and gender. Results (Table 4, column 1) indicated that children were significantly more likely to be in the early versus late-adolescent polysubstance use onset class if their fathers were in the early adolescent polysubstance use onset class versus the late-adolescent predominantly alcohol and tobacco use onset class. However, children were no more or less likely to be in the early versus late-adolescent polysubstance use onset class if their fathers were in the late-adolescent polysubstance use class versus the late-adolescent predominantly alcohol and tobacco use onset class. Boys were significantly more likely than girls to be assigned to the early versus late-adolescent onset class, and offspring age at his/her last assessment was positively associated with early versus late-adolescent polysubstance use onset classes. In the final model, mothers' adolescent ATM use predicted assignment of children to the early as opposed to late-adolescent polysubstance use class, whereas the effect of fathers' adolescent polysubstance use onset class on offspring child class was attenuated (Table 4, column 2).

Discussion

The theory of developmental congruence in adolescent polysubstance use onset was examined in the present study by testing similarities between fathers and their offspring in ATM use onset using fully prospective data across early to late adolescence in each generation. Developmental congruence was supported, as fathers' early polysubstance use onset conferred greater risk for earlier ATM use onset in their offspring compared to fathers' later onset on fewer substances. In addition, this pattern was evident when the gender difference in offspring onset—earlier for boys than girls—was controlled (Capaldi et al., 2016; Kerr et al., 2015). We recommend that further attention be devoted to understanding why early onset polysubstance use is a risk factor for offspring, and how familial or individual life-course processes can be modified to prevent intergenerational transmission. Overall, the present findings are consistent with the notion that early adolescence is a particularly important period for prevention of substance use involvement (Tucker et al., 2005), whereas later onset use, which may be more normative, is less transmissible to offspring. The findings also demonstrate how valuable well-timed interventions could be, as they promise to alter the developmental course of not only adolescents but of their future offspring. The study results also suggest that assessment of both parents' early histories of polysubstance use may inform which families and youth should be targeted for selective or tailored modular prevention approaches.

In addition to support for intergenerational congruence in polysubstance use onset, we found differences in rates of onset by generation and gender. There was evidence of later ages of onset for tobacco use among offspring compared with their fathers, which is consistent with national data on secular trends. Furthermore, daughters and, to a lesser extent, sons (nonsignificant trend) showed later onset of alcohol use than did their fathers. Some of the differences between fathers and children in this sample may be due to the original sampling

of fathers based on neighborhood risk and the attenuation of this risk in the next generation. Still, the present findings extend prior research by establishing increases in ages of onset not just among unrelated individuals across time (i.e., cross-sectional designs) but between fathers' and children's alcohol and tobacco use onset measured prospectively across adolescence. Changes over the past 20 years in restriction of availability of alcohol and tobacco for minors (Forster et al., 1998; Johnston, O'Malley, Bachman, & Schulenberg, 2012) and increased stigmatization of tobacco use and awareness of its negative health effects (Gutman, 2011; Johnston et al., 2012), may contribute to such delays in ages of onset (including continued abstinence). Notably, we anticipated children would show earlier onset on marijuana use relative to their fathers, given recent cannabis liberalization trends and the fact that the proportion of adolescents reporting marijuana use nationwide was relatively low during the fathers' adolescence (late 80s to early 1990s) compared to that of their children's generation (Miech et al., 2019). However, this hypothesis was not supported, perhaps also due to the aforementioned tempering of fathers' risk.

Returning to the discussion of congruence, findings indicated that fathers who showed ATM use in early adolescence more often had children with women who recalled greater use of ATM by age 18 years. Additionally, mothers' histories of ATM use in adolescence contributed uniquely to the prediction of their children's ATM use in early adolescence and attenuated the effect of father onset class on child onset class. These findings are consistent with assortative mating by polysubstance use history and the conclusion that both mothers' and fathers' adolescent histories of polysubstance use may contribute to risk for polysubstance use onset in the next generation. Prevention research should determine whether reducing deviant peer associations and unplanned and early pregnancies can affect assortative mating on early substance use—that is, by influencing adolescents' romantic partner selection and preventing their short-term sexual partners from becoming lifelong co-parents.

The present study did not examine mechanisms by which early onset polysubstance use was linked across generations. It is possible that among fathers with histories of earlier rather than later adolescent onset there is stronger continuity of substance use into adulthood, which influences their children's onset risk. Mechanisms consistent with congruence may involve intergenerational continuities in social risk contexts and gene-environment correlations and interactions. For example, parents and children may share traits that confer risk for early polysubstance use because they are genetically related and because they experienced similar environments. Furthermore, traits associated with substance use in adolescence, when expressed in adulthood, may interfere with parenting behaviors (e.g., monitoring) that could otherwise offset the impact of contextual risks on the next generation. Thus, identifying how to intervene to interrupt these cascades and who is most likely to benefit are important next steps. Of note, a recent set of papers explored how the efficacy of prevention may depend on child genotype (Dick, 2018), and indeed there is evidence that the family based Family Check-Up intervention moderated effects of a polygenic risk score on alcohol dependence in early adulthood (Kuo et al., 2019).

Several environmental mechanisms that may contribute to congruence in early polysubstance use suggest areas of focus for prevention. It is well established that early

onset substance use patterns complicate adolescents' early life course, including escalation of substance use problems and other problem behavior and negative educational and relationship outcomes (e.g., Broman, 2009; Mason et al., 2010; Moore et al., 2007). Adolescents with problematic social and behavioral developmental histories are poorly positioned as they enter adulthood and—as suggested here—may select vulnerable partners. This perpetuation of social risk contexts from the family of origin to the family of procreation provides an explanation for how teenage boys' substance use patterns can adversely affect their future children's behavioral outcomes via indirect pathways (Nadel & Thornberry, 2016). It follows that at-risk adolescents and young adults may benefit from assistance not just in reducing substance use but also in resuming protective developmental and educational trajectories. It is also likely that parenting behaviors—such as modeling substance use, communicating lax norms and expectations regarding early use, and poor monitoring—contribute to congruence, as prior studies with this and other samples indicate that parents' substance use histories in adolescence predict these factors (e.g., Kerr et al., 2012; 2015). Prior work suggests that before adolescence children can identify alcoholic beverages, know cultural rules about use, and have expectations regarding cognitive and behavioral effects of use (Buchmann et al., 2009). Moreover, parents' norms regarding alcohol use and parent–child discussions of these norms are associated with children's alcohol use norms, intentions to use alcohol, and alcohol use (Brody, Flor, Hollett-Wright, & McCoy, 1998; Gaines, Brooks, Maisto, & Dietrich, 1988; Miller, Smith, & Goldman, 1990). Likewise, maternal and paternal drinking motives have been demonstrated to predict adolescent drinking motives, which in turn increase adolescent alcohol use and alcohol-related problems (Mares, Lichtwarck-Aschoff, & Engels, 2013). Helping parents modify the many ways they communicate about and expose their children to substance use is a common theme in preventive interventions. The present study suggests parents with histories of early polysubstance use may be in particular need of such interventions. Overall, the findings emphasize the likely importance of familial and contextual influences on transmission of risk for early adolescent ATM use onset. Future research should examine factors that facilitate the intergenerational transmission of marijuana use, in particular, given that 1) marijuana legalization has been associated with changes in parent beliefs and behaviors (Kosterman et al., 2016), and 2) in the present study, marijuana did not exhibit the decrease in average age of onset across generations that was observed for tobacco and alcohol.

There were some limitations of the present study. The tests of congruence were limited by the small number of ATM onset classes identified for offspring. For example, given that no intermediate onset offspring class was identified, then father-child congruence in later onset could not be evaluated. The limited number of offspring classes may be due to right censoring, given the sample's ongoing maturation or to inadequate sample size. Second, for mothers, onset of first whole alcoholic drink was not assessed, and only retrospectively estimated ATM use onset ages were available, which are known to be biased and positively associated with age at recall (Johnson & Schultz, 2005) and could be sensitive to mothers' contemporary behavior (especially current ATM use). The lack of prospective data on mothers during adolescence precluded examination of heterogeneity in maternal ATM use histories (e.g., early and late-onset ATM use) in relation to offspring risk or interactive effects of maternal and paternal ATM onset. Likewise, inconsistencies in the literature

regarding the relative importance of maternal versus paternal effects could not be addressed (e.g., Gilman et al. [2009] vs. Melchior, Chastang, Mackinnon, Galera, & Fombonne [2010] regarding smoking). Third, statistical power did not permit tests of gender differences in the associations of fathers' adolescent substance use onset on boys' versus girls' onset (i.e., father class by offspring gender interactions). This is important considering that Gilman and colleagues (2009) found that fathers' smoking was more strongly associated with earlier smoking initiation in sons than in daughters. Fourth, the sample was predominantly White non-Latino; thus, generalizability of findings to other ethnic groups is unknown. Finally, the analytic design was not well-suited to examining mediators of the associations between fathers' and children's adolescent ATM onset patterns. Determining whether parents' adult use of alcohol, tobacco, or marijuana at different points in their children's development may explain congruence is of interest.

Despite these limitations, this study suggests that risk for earlier ATM use onset was transmitted from fathers to children. Thus, children of such fathers should be a prime focus of early prevention efforts. The strong co-occurrence of substance use in adolescence and the close temporal sequencing of onsets implies that successfully delaying onset of one substance may stall onset of others. Delaying early polysubstance use is a clear prevention goal, and multiple studies make this case by highlighting that prevention of early ATM use could avert problems that have cascading negative consequences across the lifespan, including delinquency and school failure (Duncan, Duncan, & Strycker, 2000), health-risking sexual behavior (Dogan, Stockdale, Widaman, & Conger, 2010), suicide risk (Hendershot, Magnan, & Bryan, 2010), and dating violence (Guo, Collins, Hill, & Hawkins, 2000; Mason et al., 2010). Intergenerational researchers and theorists make what is an even stronger case for resource allocation and action—that modifiable risks to development emerge long before children are even conceived (Cheng et al., 2016; Patton et al., 2018) and that prevention may have intergenerational effects.

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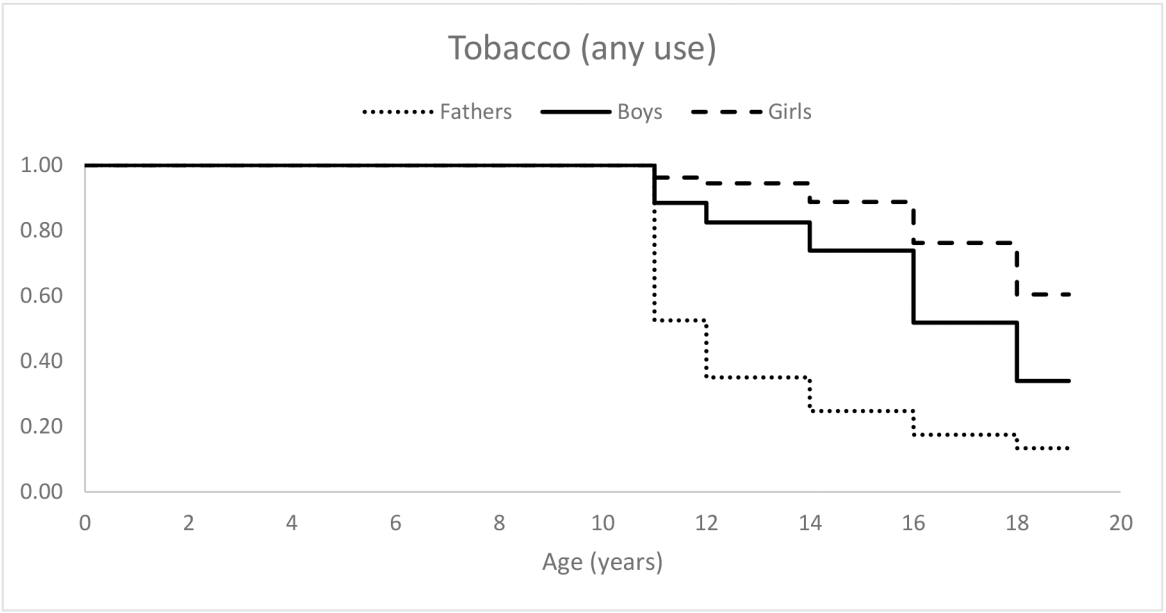


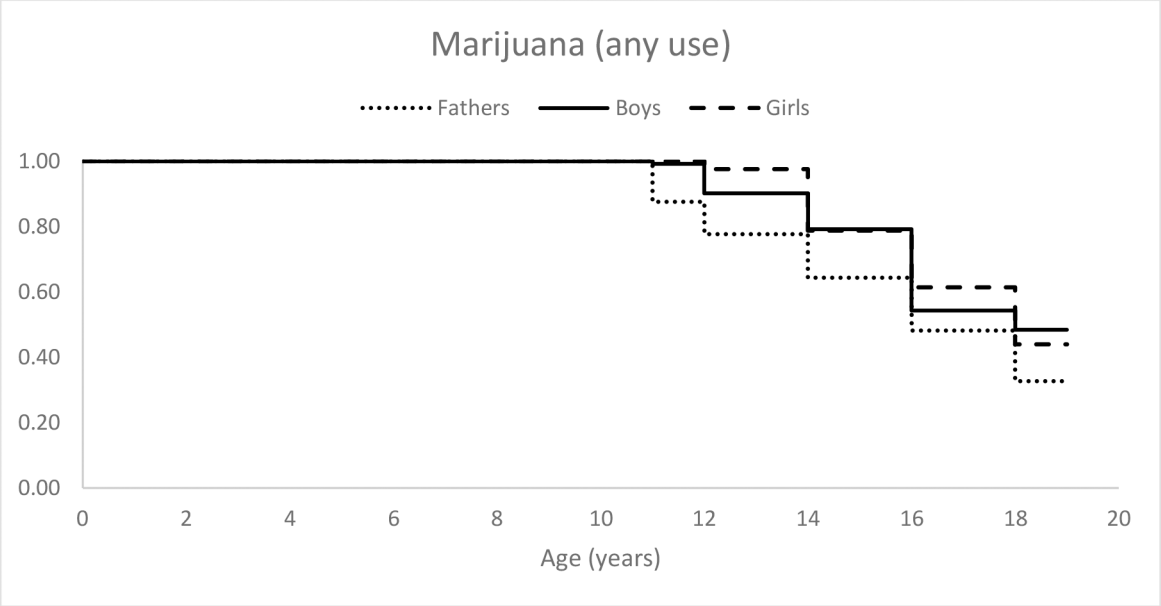


Figure 1.
Estimated baseline survival curves for alcohol, tobacco, and marijuana use onset by fathers, boys, and girls.

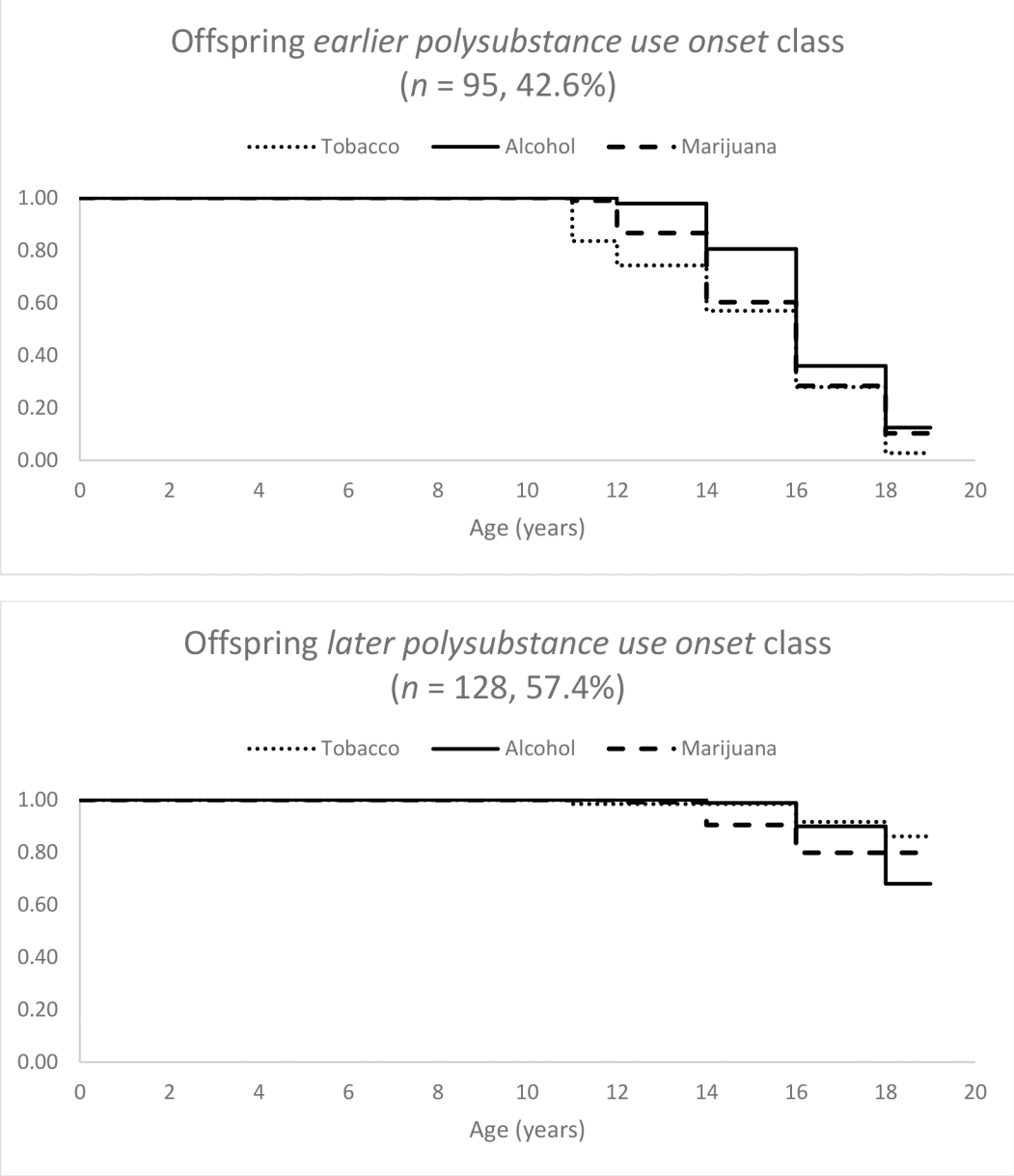
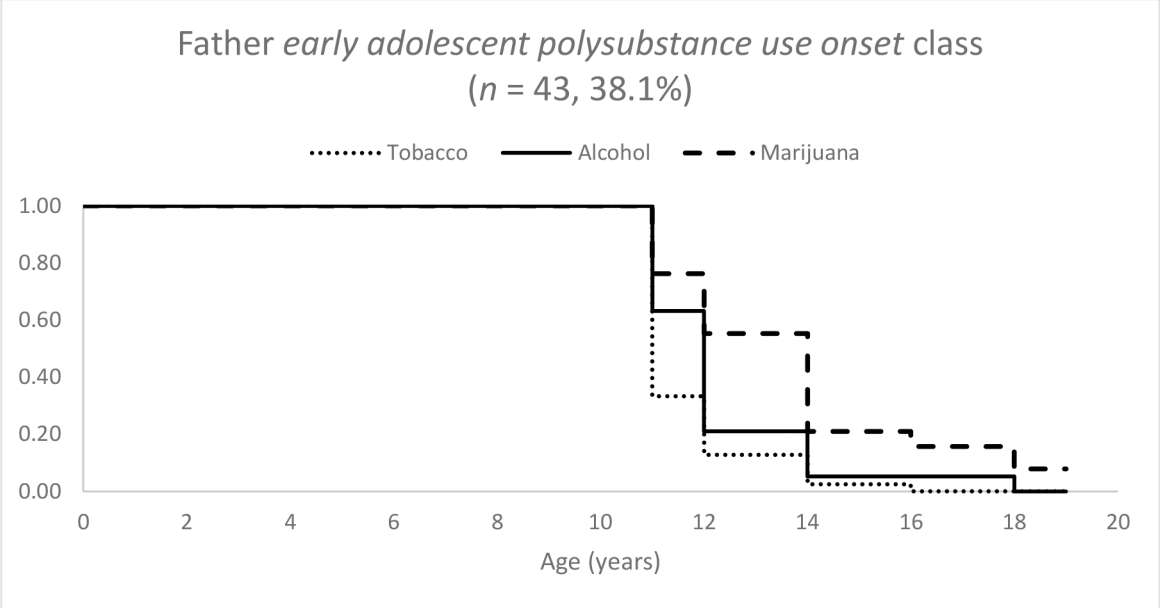
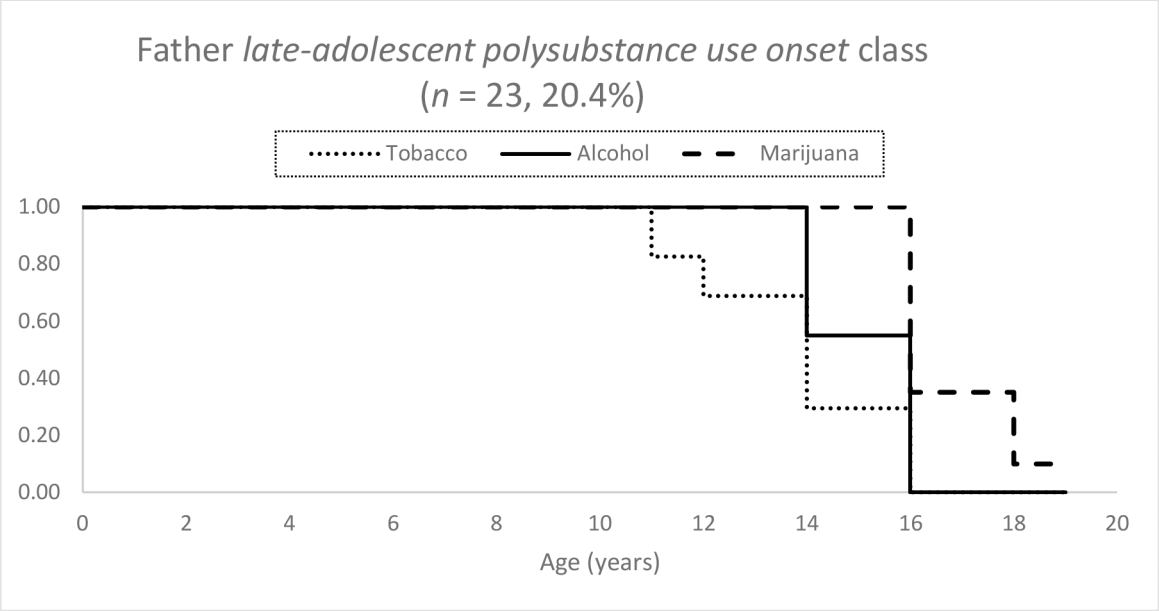


Figure 2.
Survival curves for offspring polysubstance use onset two-class solution.





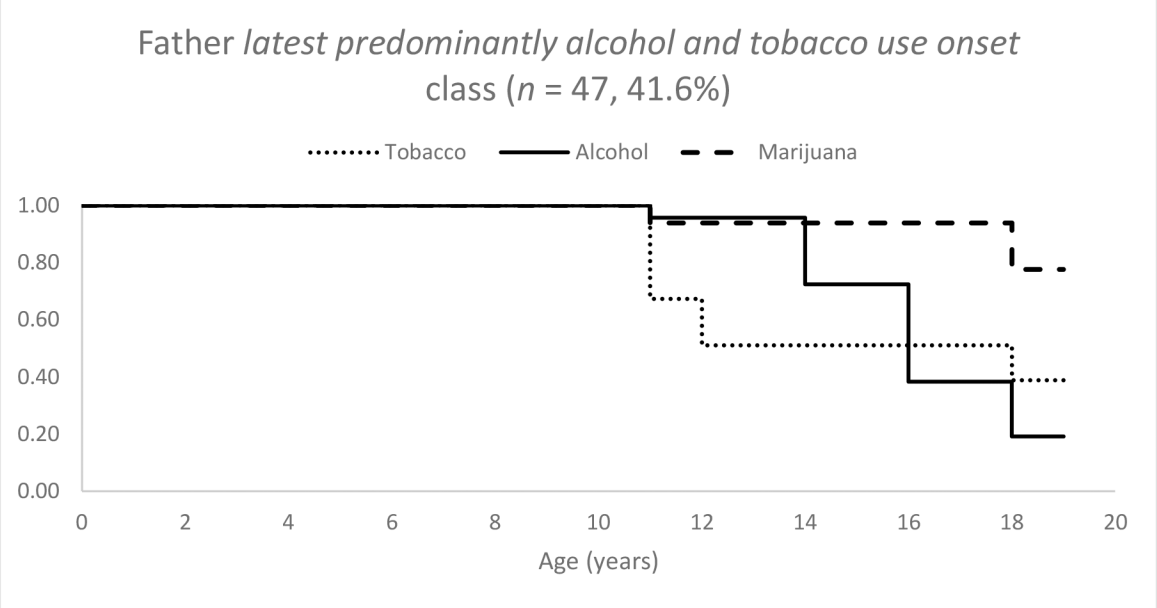


Figure 3.
Survival curves for fathers' adolescent polysubstance use onset three-class solution.

Table 1

Descriptive Statistics and Alcohol, Tobacco, and Marijuana Onset Rates at Each Age Category

	Fathers	Boys	Girls	Children
Total sample size (*N*)	113	99	124	223
Offspring sample size by age (*n*)				
Ages 9–10 years		54	59	113
Ages 11–12 years		85	108	193
Ages 13–14 years		75	102	177
Ages 15–16 years		64	80	144
Ages 17–18 years		49	58	107
Onset rates at each age category:				
n who onset / *n* at-risk to onset (% who onset)				
Alcohol use:				
Prior to age 11 years	16/107 (15%)	0/56 (0%)	0/68 (0%)	0/124[a] (0%)
Ages 11–12 years	19/96 (20%)	1/90 (1%)	2/116 (2%)	3/206 (2%)
Ages 13–14 years	27/80 (34%)	14/79 (18%)	7/100 (7%)	21/179 (12%)
Ages 15–16 years	31/52 (60%)	24/55 (44%)	21/73 (29%)	45/127 (35%)
Ages 17–18 years	16/27 (59%)	15/27 (56%)	15/37 (41%)	30/64 (47%)
Tobacco use:				
Prior to age 11 years	46/111 (41%)	12/99 (12%)	6/124 (5%)	18/223 (8%)
Ages 11–12 years	21/66 (32%)	6/83 (7%)	3/115 (3%)	9/198 (5%)
Ages 13–14 years	14/47 (30%)	8/65 (12%)	8/95 (8%)	16/160 (10%)
Age 15–16 years	9/35 (26%)	15/42 (36%)	14/65 (22%)	29/107 (27%)
Ages 17–18 years	7/28 (25%)	8/19 (42%)	12/34 (35%)	20/53 (38%)
Marijuana use:				
Prior to age 11 years	12/110 (11%)	1/99 (1%)	0/124 (0%)	1/223 (.004%)
Ages 11–12 years	9/100 (9%)	10/94 (11%)	3/121 (2%)	13/215 (6%)
Ages 13–14 years	14/92 (15%)	11/70 (16%)	21/100 (21%)	32/170 (19%)
Ages 15–16 years	17/78 (22%)	18/43 (42%)	15/59 (25%)	33/102 (32%)
Ages 17–18 years	19/63 (30%)	3/18 (17%)	11/33 (33%)	14/50 (28%)

[a]The age 9–10 assessment wave was closed midway through the study for budgetary reason, thus precluding a determination of whether or not onset of a whole drink had occurred for the children who were not assessed until later; affected cases were treated as missing.

Table 2

Tests of Differences Between Generations in ATM Use Onset Trends

	Fathers versus Boys	Fathers versus Girls
Substance	**Estimate$_{(df)}$, *p* value**	**Estimate$_{(df)}$, *p* value**
Tobacco onset	$44.92_{(5)}$, $p < .001$	$80.11_{(5)}$, $p < .001$
Alcohol onset	$14.80_{(4)}$, $p = .055$	$56.94_{(4)}$, $p = .001$
Marijuana onset	$8.07_{(4)}$, $p = .089$	$7.15_{(4)}$, $p = .128$

Note: df = degrees of freedom. Tabled estimates denote Wald statistics, assessing the change in overall model fit for a null model that assumed equal, to an alternative model that assumed unequal, onset rates across generations.

Table 3.

Class Enumeration Results for Offspring (Panel 1) and Fathers during Their Adolescence (Panel 2)

Panel 1: Offspring								
Number of classes	**Number of parameters**	**Log likelihood**	**AIC**	**BIC**	**Sample size adjusted BIC**	**Entropy**	**Class sizes**	**Lo-Mendell-Rubin *k* versus *k*+1 classes**
1 class	14	−688.00	1403.99	1451.69	1407.32	NA	223	NA
2 classes	30	−614.75	1289.50	1391.71	1296.64	.74	95/128	144.82, $p < .001$
3 classes	46	−593.32	1278.64	1435.37	1289.59	.86	75/134/14	42.45, $p = .029$
4 classes	62	−593.32	1310.64	1521.89	1325.40	.89	14/0/75/134	11.50, $p = .387$
Panel 2: Fathers during adolescence								
Number of classes	**Number of parameters**	**Log likelihood**	**AIC**	**BIC**	**Sample size adjusted BIC**	**Entropy**	**Class sizes**	**Lo-Mendell-Rubin k versus k+1 classes**
1 class	15	−565.15	1160.29	1201.21	1153.80	NA	113	NA
2 classes	31	−519.89	1101.79	1186.34	1088.36	.81	44/69	89.33, $p = .006$
3 classes	47	−497.35	1088.71	1216.90	1068.35	.82	43/23/47	44.60, $p = .009$
4 classes	63	−481.05	1088.10	1259.93	1060.81	.92	23/23/31/36	33.37, $p = .006$
5 classes	79	−473.79	1105.57	1321.03	1071.35	.90	24/11/19/34/25	14.46, $p = .187$

Note: AIC = Akaike information criterion. BIC = Bayesian information criterion.

Table 4

Tests of Father–Child Transmission of Polysubstance (ATM) Use Onset in Adolescence

Child in earlier versus later ATM onset class given	Primary adjusted model *b(se)*	Controlling for mothers' ATM use *b(se)*
Father in EA vs. LA polysubstance use onset class	1.79(.81), $p = .028$	.89(.88), $p = .314$
Father in LA polysubstance use class vs. latest predominantly alcohol and tobacco onset class	.67(.91), $p = .491$	.27(.88), $p = .760$
Child's maximum age	.66(.11), $p < .001$	.70(.13), $p < .001$
Child is male	1.54(.66), $p = .019$	1.66(.72), $p = .021$
Mothers' adolescent ATM use onset		.81(.35), $p = .021$

Note: ATM = alcohol, tobacco, and marijuana; EA = early adolescent; LA = late adolescent; Tabled numbers denote *b(se)* on logit scale.