

**Are parents' beliefs about substance use risks associated with their adolescent child's  
substance use?**

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### **Abstract**

Although specific parenting behaviors have been identified as protective factors for adolescent substance use (SU), the relationship between parents' beliefs about the risks associated with SU and their adolescent child's SU remains unclear. Furthermore, shared genes between parents and their biological offspring may confound this relationship. This analysis draws data from a prospective, longitudinal adoption study of both non-biologically related parent-child dyads and biologically related dyads to investigate the relationship between parents' beliefs about the risks associated with SU and their adolescent child's self-reported SU (*M* age = 18.9 years). The sample included 284 parent-adolescent dyads, of which 227 were not biologically related (54.2% of adolescents were male and 57.7% were White). A confirmatory factor analysis of the Parent Substance-Related Beliefs Scale revealed that parents' risk perceptions differed by substance type. Structural equation modeling was used to regress the adolescent's use of a specific substance on their parent's perception of risks associated with that substance. Parents' greater perceived risk associated with alcohol use was associated with a decrease in the probability of their adolescent ever drinking by age 18 but not their alcohol use frequency; however, parents' perceptions of cannabis-related risks were not associated with their adolescent's initiation of cannabis use or frequency of use. In addition, prenatal substance exposure was associated with adolescent SU. The parents' biological relation did not moderate any identified associations, suggesting that they were likely environmental in nature. Parents' perceptions of alcohol-related risk may be a viable target for preventive interventions.

**Keywords:** parenting, adolescence, substance use, drug use, risk perceptions, adoption design

## **Are parents' beliefs about substance use risks associated with their adolescent child's substance use?**

Substance use (SU) during adolescence can disrupt brain development (Hamidullah et al., 2020), is associated with truancy and lack of school engagement (Trucco, 2020), and increases risk of addiction later in life (Viner & Taylor, 2007). Despite the potential for negative consequences of adolescent SU, 58% of adolescents in the US have used at least one substance before the end of 12<sup>th</sup> grade (Miech et al., 2025). Adolescence is a developmental period marked by rapid physical and brain development, high social pressure, and increased risk-taking, which could make SU particularly appealing (Pechmann et al., 2020). Parents play a critical role in whether their adolescent uses substances (Trucco, 2020). As parents' beliefs about the risks associated with SU likely influence their own SU (Thornton et al., 2013), their disapproval of their child's use (Brooks-Russell et al., 2015), and their child's beliefs about SU (Shin & Miller-Day, 2017; Willoughby et al., 2021), it is plausible that parents' beliefs are related to their adolescent's SU. We sought to test this possibility in the current study.

In addition to parental influences on adolescent SU via environmental exposure to specific parental behaviors (e.g., modeling SU, communicating disapproval of adolescent SU), parents also pass down genes to their biological offspring that may increase or decrease their offspring's risk of SU (Huibregtse et al., 2016). Much of the research to date on parental influences on adolescent SU has sampled biologically related parent-child dyads. Because specific genes have been found to increase adolescent risk of SU (Dick et al., 2016), the shared genes between the parent and child in samples of biological families may confound the relationship between parenting factors and adolescent SU. In some cases, genetic factors could also moderate the effect of parenting on adolescent SU, such that the effectiveness of direct

parenting practices can depend on the child's genotype (Pieters et al., 2012). By drawing data from a longitudinal parent-offspring adoption study that included adolescents reared with non-biological parents, we can more clearly isolate environmental influences. Specifically, we will examine whether parents' beliefs about the risks associated with SU will predict their adolescent's subsequent SU, after adjusting for genetic relatedness.

### **Adolescence as a Key Developmental Period for Substance Misuse Prevention**

The adolescent brain is prone to reward seeking, impulsivity, and risk-taking while top-down executive control systems are still developing, resulting in less reliable behavioral regulation (Pechmann et al., 2020). As the adolescent brain is still developing, it is highly susceptible to the negative effects of SU (Hamidullah et al., 2020). Earlier onset of SU is associated with an increased risk of lifetime SU disorder (Viner & Taylor, 2007), highlighting the importance of preventing adolescent SU onset. Additionally, adolescents experience more negative emotions than any other age group due to rapid bodily changes and high social pressure and can often lack the coping skills necessary to address these stressors (Pechmann et al., 2020). Difficulty coping may make SU appealing, particularly during this period where peers increasingly influence behaviors and the social rewards for risk-taking are salient (Ellis et al., 2012).

### **How Parental Beliefs May Influence Adolescent Substance Use**

Parents play a critical role during adolescence because most adolescents live with their parents and rely on them to meet their basic needs. Not surprisingly, specific parenting practices such as parental modeling of SU, parental monitoring, and permissiveness towards SU have been linked to adolescent SU (Trucco, 2020). Further, parents who believe that SU is harmful are less likely to use substances themselves (Thornton et al., 2013), thereby reducing their child's

exposure to SU and access to substances in the home, both of which are known risk factors for adolescent SU (Nawi et al., 2021).

Parental beliefs have been linked to observable parenting behaviors (Gattis et al., 2022). Although not measured here, one possible mechanism through which parental beliefs about the risks associated with SU may influence adolescent SU is via parenting behaviors. For example, when parents perceive SU as harmful, they may convey that to their children via strict rule-setting, which has a protective effect against adolescent SU (Bacikova-Sleskova et al., 2019). The protective effect of rule-setting regarding one substance may also confer protection against the use of other substances (i.e., strict rule-setting regarding alcohol use has been found to protect against adolescents' use of tobacco and cannabis; Koning et al., 2020). Parents who believe that SU is harmful may exert a protective influence on their children by communicating their beliefs to their children. Perceived parental disapproval of SU has been found to be protective against SU in adolescence and emerging adulthood (Brooks-Russell et al., 2015).

Children also tend to adopt beliefs similar to their rearing parents' beliefs. Children of parents who communicated their disapproval of media depictions of adolescent SU tended to report personal anti-SU norms, which was linked to less frequent SU (Shin & Miller-Day, 2017). In one study on political attitudes, adult children tended to express political beliefs that were similar to their rearing parents, even when they were not biologically related (Willoughby et al., 2021). Adult children who were reared by biologically related parents had political beliefs more similar to their rearing parents than adult children reared by non-biologically related parents, indicating that there was an additive effect of genes and the environment. Although political beliefs are substantively different from beliefs about the risks associated with SU, this study established that parental beliefs can be passed onto children and lends support for our hypothesis

that these beliefs will have a stronger effect on biologically related children. When parents believe that SU is harmful, it is plausible that their children will adopt these beliefs themselves, which is associated with lower risk of adolescent SU (SAMHSA, 2013). In summary, parental beliefs that SU is harmful are expected to reduce the risk of adolescent SU through the child's adoption of the parent's belief that SU is harmful. As parental beliefs may be a modifiable risk factor for adolescent SU, if this hypothesis is confirmed, it could be an important target for prevention.

### **Prenatal Substance Exposure**

Adolescents with prenatal substance exposure may experience increased reward responses to SU, putting them at greater risk of developing a substance use disorder (Grecco & Atwood, 2020). Although other genetically-conscious studies have found mixed effects of prenatal substance exposure on adolescent SU (Bidwell et al., 2017; Knopik et al., 2009), the potential for prenatal exposures to influence adolescent SU warrants attention to this potential risk factor.

### **Genetic Influences on SU and the Adoption Study Design**

Adoption and twin study designs have illuminated genetic influences on adolescent SU, specifically finding that the role of genetics depends on the substance type, the child's sex, and the child's age (Huibregtse et al., 2016). It is critical to parse out the role of genetics because it is possible that biologically related parent-child dyads share genes that drive both the parent's beliefs about the risks associated with SU and their adolescent's actual SU. One unique advantage of parent-offspring adoption study designs is that they provide an opportunity to study genetic influences on behavior without the potentially confounding effects of a shared rearing environment that occurs when children live with parents to whom they are genetically related.

The current parent-offspring adoption study design is well-suited to evaluate the effect of parents' beliefs on adolescent SU because it includes non-biologically related parent-child dyads. In this analysis, including both non-biologically related dyads and biologically related dyads allows for moderation analyses to see if the relationship between parents' beliefs and adolescent SU depends on their genetic relatedness.

### **Current Study**

Given the pathways through which a parents' beliefs about the risks associated with SU can protect their adolescent child from SU, we predicted that when parents perceive greater risk associated with SU, their adolescent will be less likely to have initiated SU by age 18 or use less frequently during adolescence. Parental SU, the adolescent's prenatal substance exposure, sex, and the genetic relatedness of the parent-child dyad will be included as covariates. Furthermore, we hypothesize that genetic relatedness between rearing parents and adolescents (yes/no) will moderate the effect of parental beliefs on adolescent SU: the effect of parents' beliefs on adolescent SU will be stronger within biologically related parent-child dyads than within biologically unrelated parent-child dyads (i.e. adoptive parent and child). The results of this analysis will clarify the effect of parents' beliefs about the risks associated with SU on adolescent SU, which may indicate if strategies to promote parental education on the risks associated with SU could be an effective preventive intervention.

## **Method**

### **Original Study**

The data used in this analysis were drawn from the Early Growth and Development Study (EGDS), a prospective longitudinal adoption study (Leve et al., 2019). The study team identified potential participants via 45 adoption agencies across 15 US states to recruit eligible families.

Children adopted by non-biologically related caregivers within three months of their birth were recruited between 2003-2010 from across the United States ( $n = 561$ ), with data collected from infancy through late adolescence. To participate, children were required not to have had any known major medical conditions (e.g., extreme prematurity) and parent participants had to understand English at the eighth-grade level. An expansion of the study allowed for data collection from other children in the adoptive and birth family homes (e.g., other adopted children in the home, biological siblings of the adoptee who are reared in the birth home) in middle childhood and adolescence, resulting in a sample of 856 parent-child dyads who had provided data on parents' substance-related beliefs or child self-reported SU at 11y, 15y, or 18y when these measures were collected.

### **Inclusion Criteria**

In this analysis, we drew data from the 15-year (15y) and 18-year (18y) study visits that occurred between 2018 and 2023. To be included in this analysis, participants must have been eligible for both the 15y and 18y visits at the time of data collection and provided data in at least one of the visits. Sixteen participants were not eligible to complete the 15y visit: 13 children were too old (18 or older), one was in the custody of the state, one was unable to participate due to a family emergency, and one did not live with their parent. At the 18y visit, 501 participants were not eligible: 499 child participants were too young (under 18) and two participants were too old (over 21). We additionally excluded 55 parent-child dyads who provided no data at both the 15y and 18y study visits due to refusal and/or non-response from both the parent and child. The resulting sample included  $n = 284$  parent-child dyads.

### **Study Participants and Procedures**



Over half of adolescents in this sample identified as White (57.7%), 25.7% identified as more than one race, 15.8% were Black/African American, 0.4% were Asian, and 0.4% of the adolescents' race was unknown (see Table 1). About half of the adolescents (49.1%) were prenatally exposed to SU. There were slightly more male adolescents in this sample (54.2%) than female adolescents (45.8%). Most parent participants were female (85.5%). Of parents who reported their family income, over half (54.8%) reported that their combined annual family income was \$100,000 or more. Most adolescents were adopted ( $n = 201$ ; 70.8%). Two hundred and twenty-seven (79.9%) of the parent-child dyads were not biologically related because in addition to the 201 adoptees, one adolescent in a birth family home was a stepchild and 25 adolescents in adoptive family homes were other adopted children.

When adolescents were approximately 15 years old, their rearing parent reported on their own beliefs about the risks associated with SU. Adolescents self-reported their own SU at approximately ages 15 and 18. Demographic information about the parents and adolescents in this sample, including prenatal substance exposure, was collected during earlier waves of the study.

## **Measures**

### ***Parent Substance-Related Beliefs Scale – Age 15***

Parents reported on their beliefs about substance-related risks using the Parent Substance-Related Beliefs Scale when their child was 15 years old. This scale includes 16 statements about beliefs related to 4 substances: smoking, alcohol, cannabis, and other drugs. Example items include: “If you are young and healthy, smoking is not dangerous” and “A person has to drink for many years before it is likely to affect his/her health” (Chassin et al., 1998). The parent was asked to rate their agreement with each statement on a scale from 1 to 5, with higher scores

indicating higher levels of agreement with the statement. The 16-item scale is structured such that the same four questions are asked about each substance type (smoking, alcohol, cannabis, and other drugs; see Online Resource 1). All 16 items on the Parent's Substance-Related Beliefs Scale demonstrated acceptable internal reliability ( $\alpha = 0.81$ ), as did the smoking-related items ( $\alpha = 0.72$ ), alcohol-related items ( $\alpha = 0.78$ ), cannabis-related items ( $\alpha = 0.90$ ), and other drug-related items ( $\alpha = 0.74$ ) when used as substance-specific subscales. When added to the models as a predictor, items were reverse-coded such that higher scores indicated a greater perception of substance-related risk.

***Outcome: Adolescent SU – Age 18***

**Lifetime Use.** At the 18y visit, the Youth Risk Behavior Survey (YRBS; CDC, 2015) was used to assess adolescents' lifetime SU. Participants who indicated that they had ever used cigarettes, alcohol, cannabis, or other drugs were coded as '1' for that specific substance. If they indicated that they had not ever used that substance, their response was coded as '0' for that substance.

**Frequency of Use.** If participants indicated that they had used alcohol, cannabis, or cigarettes in their lifetime on the YRBS, they were then asked how often they used that substance, on average, over the past 12 months. Response options were coded from 0 to 5 indicating: "never", "less than once a month", "less than once a week", "1-2 days per week", "3-5 days per week", and "almost every day or every day". Participants who indicated that they had never used one of these substances in their lifetime were not prompted with the frequency question for that substance. To minimize missingness on the frequency items, all participants who indicated no lifetime use of each substance were recoded to "never" for the frequency

question for that substance. Participants completed the YRBS at the 15y and 18y visits, so their 15y use frequency was added to the frequency models as a covariate to adjust for earlier use.

### ***Moderators and Covariates***

**Rearing parent's biological relation to the adolescent.** A dichotomous variable was used to indicate whether the parent who was reporting on their own substance-related beliefs was biologically related to the adolescent. '0' indicated no bio-relation ( $n = 227$ ) and '1' indicated that the reporter was biologically related ( $n = 57$ ). When used in an interaction term, this variable was recoded as  $-1$  and  $1$ .

**Prenatal Exposure to Substances.** Birth mother medical record data were coded for prenatal SU. When medical record data were missing, the birth mother's self-report was used. This variable was coded such that adolescents who were prenatally exposed to any amount of any substance were marked '1' on this variable ( $n = 111$ ) and '0' if they were not ( $n = 115$ ). When used in an interaction term, this variable was effects coded ( $1$  and  $-1$ ).

**Parent substance use.** Parental SU was measured using the reporting parent's response to "During your lifetime, did you have a strong desire or urge to [use drug] that you could not keep from [using drug]?" and "Have you OFTEN smoked a lot more than you intended or for more days in a row than you intended? For example, smoking half a pack or more when trying to limit yourself to only 1 or 2 cigarettes?". If the reporting parent responded "yes" to the first question regarding alcohol, sedatives, tranquilizers, amphetamines, prescription painkillers, inhalants, cannabis, cocaine, hallucinogens, or heroin, or they responded "yes" to the second question, the parental SU variable was coded as '1' to indicate that they had lifetime serious use of at least one substance ( $n = 30$ ). If they responded "no" to the first question regarding all the substances listed above and "no" to the second question, their response was coded as '0' ( $n =$

191). Birth parents responded to these questions when the child they placed for adoption was between 3 and 6 months old and adoptive parents responded when the child was 18 months old.

**Child sex.** Adolescent participants who were assigned female at birth were coded as ‘0’ ( $n = 130$ ) and participants assigned male at birth were coded as ‘1’ ( $n = 154$ ).

### **Missing Data**

Missingness on each of the included variables ranged from 0% to 34.5%. Little’s MCAR test revealed that the data were not missing completely at random ( $\chi^2(1641) = 2020, p < .001$ ; Little, 1988), however, a “missing” variable was created and coded as ‘1’ for cases with missingness on any included variable ( $n = 200$ ) and ‘0’ for cases without missingness ( $n = 84$ ; see Table 1 and Online Resources 1 and 2 for count of respondents for all included variables). When regressed onto all the included variables, this variable was not significant, indicating that the data were likely missing at random, so the MICE package (van Buuren & Groothuis-Oudshoorn, 2011) was used for multiple imputation ( $m = 20$ ). Multiple imputation allowed for the full sample ( $n = 284$ ) to be used in all models. Model estimates from the 20 multiply imputed datasets were pooled using Rubin’s rules (Rubin, 1987).

### **Analytic Approach**

All analyses were conducted in R version 4.3.3 (R Core Team, 2024). A confirmatory factor analysis was performed to determine whether parents’ beliefs about all four substance types could load onto a single factor and meaningfully predict adolescents’ SU of each type or if parents’ perceived risks associated with the use of each SU type varied and it would be logical to use parents’ substance-specific beliefs to predict their adolescent’s use of that substance (i.e., alcohol-related beliefs predicting the adolescent’s alcohol use). A higher-order factor structure was also modeled to see if the parents’ belief items loaded onto substance-specific latent factors,

which can then indicate a higher-order factor representing the parents' overall beliefs. To compare models, the chi-square statistic, comparative fit index (CFI), Tucker-Lewis Index (TLI), root mean square error of approximation (RMSEA), and standardized root mean squared residual (SRMR) were used.

As 49 adolescent participants shared a home with another participant in the study and SU frequency was modeled over time, clustering both within-household and within-individual was assessed. If the design effects were above 2, multilevel modeling would be used. If the design effects were below 2 but the intraclass correlation coefficients (ICC) were above .05, a robust estimator would be used to adjust for this non-independence of observations (Lai & Kwok, 2015).

If the confirmatory factor analysis reveals that all the parents' substance-related beliefs loaded onto one global beliefs latent variable, either with or without the higher-order structure, that latent variable would be used to predict the adolescent's lifetime use or frequency of use of each substance type by age 18 in separate models. The following covariates would also be added to the model: parent's biological relation to the adolescent, parent's lifetime serious SU, prenatal substance exposure, and child sex. If the parents' beliefs fit better when loaded onto four substance-specific latent variables, each of those latent variables would be used in two models, with one model predicting adolescents' lifetime use of that specific substance (yes/no) and the second model predicting adolescents' use frequency of that substance while adjusting for the covariates. As the adolescent lifetime SU outcome variables were dichotomous, a probit regression model was fit using a weighted least squares estimator in the lifetime use models. The use frequency outcomes were treated as continuous, so a linear regression model was fit using a

maximum likelihood estimator in the frequency models. The null hypothesis was rejected at a  $p$ -value of less than 0.05.

## **Results**

Parents generally perceived the risks associated with SU. Nearly every parent in the sample strongly disagreed with each of the smoking and other drug-related items, indicating that they perceived a high level of risk associated with using both of those substance types (see Online Resource 1). Parents' average level of agreement with the four smoking items ranged from 1.1–1.3 and their average level of agreement with the four other drug-related items ranged from 1.1–1.2, with one being the highest level of risk perceived. There tended to be less perceived risk associated with alcohol and cannabis use, with the mean scores on each of those items ranging from 1.6–3.1 and 1.6–1.9, respectively.

Most adolescents reported SU by age 18. The most frequently used substance was alcohol, with 81.3% of participants reporting use of alcohol by age 18. Over half of adolescents (58.5%) self-reported using cannabis, 30% used cigarettes, and 20.5% used other drugs. As expected, frequency of cigarette, alcohol, and cannabis use all increased from the 15y visit to the 18y visit (see Online Resource 2).

### **Confirmatory Factor Analysis**

The confirmatory factor analysis revealed that a four-factor, substance-specific model and both global parents' beliefs models (higher-order structure and not) fit poorly due to the lack of variance on both the smoking-related beliefs latent variable and the other drug-related beliefs latent variable. This result was unsurprising as nearly every parent in this sample strongly disagreed with each of those items. This lack of variance precluded us from using those items in subsequent analyses. Instead, we compared a two-factor model with the parents' alcohol and

cannabis-related beliefs items loaded onto two separate latent factors and a higher-order model with both of those latent factors indicating an alcohol- and cannabis-related beliefs factor (see Online Resource 3). The higher-order model had estimation issues, so the two-factor model was adopted.

### **Clustering**

All outcomes assessed demonstrated non-negligible clustering ( $ICC > .05$ ) and design effects below 2 for clustering within household and within-individual. ICC values ranged from 0.01–0.47 and design effects fell between 1.00–1.15. Based on these high ICC values, robust estimators were used in all models to account for non-independence; that is, the weighted least squares mean and variance adjusted [WLSMV] estimator for predicting lifetime use and the maximum likelihood with robust standard errors [MLR] estimator in the frequency models.

### **Effect of Parents' Beliefs on Alcohol and Cannabis Use**

Parental beliefs about alcohol and the adolescent's prenatal exposure to substances were significantly associated with alcohol use initiation by age 18 (see Figure 1a). Parents' greater perceived risk associated with alcohol use was associated with a decrease in the probit probability of their adolescent ever drinking by age 18 ( $b = -0.57$ ,  $SE = 0.20$ ,  $t = -2.95$ ,  $df = 124.68$ ,  $p = .004$ ). To aid interpretation, we calculated predicted probabilities using the probit link and model estimates. Holding all other predictors at their reference levels, the predicted probability of drinking by age 18 was 87.8% for an adolescent whose parent scored 1 SD below the mean on the Parent's Substance-Related Beliefs Scale (i.e., perceived alcohol as less harmful) and 65.9% for an adolescent whose parent scored 1 SD above the mean (i.e., perceived alcohol as more harmful). Thus, stronger parental beliefs about the risks of alcohol use were associated with a lower predicted probability of adolescent alcohol use initiation. Additionally, adolescents

who were prenatally exposed to substances had a significantly higher probit probability of initiating alcohol use by age 18 ( $b = .76$ ,  $SE = .24$ ,  $t = 3.20$ ,  $df = 158.74$ ,  $p = .002$ ). Holding all other predictors at their reference levels, the predicted probability of drinking by age 18 was 93.9% for an adolescent who was prenatally exposed to substances and 78.5% for an adolescent who was not prenatally exposed. Parents' cannabis-related beliefs were not associated with their adolescent's initiation of cannabis use by age 18, nor were any of the other covariates in the model (see Figure 1b).

Parents' alcohol-related beliefs did not predict their adolescent's alcohol use frequency at 18y (see Figure 2a). Both the adolescent's sex and their alcohol use frequency at 15y did predict their use frequency at 18y, after adjusting for the other covariates. Male adolescents were estimated to drink less frequently than female adolescents ( $b = -.39$ ,  $SE = .13$ ,  $t = -2.93$ ,  $df = 219.02$ ,  $p = .004$ ) and greater drinking frequency at the 15y visit predicted greater drinking frequency at the 18y visit ( $b = .59$ ,  $SE = .14$ ,  $t = 4.09$ ,  $df = 167.26$ ,  $p < .001$ ). Parents' cannabis-related beliefs did not predict their adolescent's cannabis use frequency at 18y (see Figure 2b). Adolescents who used cannabis more frequently at the 15y visit ( $b = .77$ ,  $SE = .14$ ,  $t = 5.44$ ,  $df = 61.54$ ,  $p < .001$ ) and those who were prenatally exposed ( $b = .49$ ,  $SE = .25$ ,  $t = 1.98$ ,  $df = 174.04$ ,  $p = .049$ ) were estimated to use cannabis more frequently at the 18y visit.

### **Moderating Effect of Reporting Parent's Biological Relation to the Child**

#### ***Biological relation moderating the effect of prenatal exposure on alcohol use initiation and cannabis use frequency***

Although this analysis was not planned a priori, the finding that prenatal exposure significantly predicted adolescent alcohol use initiation and cannabis use frequency warranted further investigation. As adolescents living in their birth homes who were prenatally exposed to



SU may be more likely to be exposed to postnatal environmental risk factors for adolescent SU (Dodge et al., 2019), a prenatal exposure x biological relation interaction term was created and added to the model. The interaction term did not significantly predict adolescent alcohol initiation, but prenatal exposure remained a significant predictor. When this interaction term was added to the cannabis use frequency model, it was not significant, nor were any of the other predictors. Thus, prenatal exposure appears to be a risk factor for adolescent alcohol use initiation regardless of the adolescent's biological relation to their rearing parent, whereas its association with cannabis use frequency was not robust after accounting for the shared variance with the interaction term.

***Biological relation moderating the effect of beliefs on alcohol and cannabis use***

When a latent variable indicated by the interaction of biological relation and each of the parents' beliefs items was added to the models, biological relation did not moderate the effect of parents' alcohol-related beliefs on adolescent lifetime alcohol use, nor did it moderate the effect of parents' cannabis-related beliefs on adolescent cannabis initiation. This interaction was also not a significant predictor of alcohol use frequency or cannabis use frequency when added to those models.

**Discussion**

This secondary analysis of data from a prospective longitudinal parent-offspring adoption study revealed that parents' perceptions of risk associated with alcohol use when their adolescent was 15 years old significantly predicted adolescent alcohol initiation by age 18. Conversely, parents' perceptions of risks associated with cannabis use did not predict adolescent cannabis use. Parents' beliefs about the risks associated with alcohol and cannabis use when their adolescent was 15 also did not predict adolescent frequency of use for either substance at age 18.

The adolescent's 15y use frequency consistently predicted their later use. Finally, prenatal exposure significantly predicted adolescent alcohol use initiation and cannabis use frequency, and genetic relatedness did not moderate those relationships. There was no evidence that the effect of parents' beliefs on their adolescent's SU was moderated by genetic relatedness.

Parents reported a high level of perceived risk associated with smoking and using other drugs, which demonstrates the effectiveness of public health campaigns that have emphasized the health risks posed by using those substances. Unfortunately, the lack of variance on the parents' smoking- and other drug-related beliefs items precluded our ability to examine their relationship to adolescent use of these substances. Although parents generally endorsed that smoking and using other drugs was harmful, adolescents had smoked cigarettes and used other drugs at a rate of 30% and 20.5%, respectively. These findings, coupled with the finding that parents' cannabis-related beliefs were not a significant predictor of adolescent cannabis use, indicate that parental perceptions of high risk associated with use of cigarettes, other drugs, and cannabis are not enough for effective prevention. It is critical to identify effective preventive interventions for these substances based on their potential to cause long-term impairments when used heavily during adolescence (Hamidullah et al., 2020).

### **Implications for Prevention Science**

Our finding that adolescents with parents who perceived a high level of risk associated with alcohol use were less likely to have initiated alcohol use by age 18 suggests that parental education about the risks associated with alcohol use may be an effective preventive intervention. As biological relation did not moderate this relationship, it appears that parents' beliefs can predict their adolescent's initiation of alcohol use even when they do not share a genetic relation to their child. The finding that prenatal exposure increased the probability of

adolescent alcohol initiation after adjusting for the interaction between parents' biological relation and prenatal exposure implies that prenatal exposure may be a risk factor for adolescent alcohol use even when postnatal risk factors are absent. Genetically informed studies have found mixed effects of prenatal exposure on adolescent alcohol use (Bidwell et al., 2017; Knopik et al., 2009), although the authors are not aware of any study that previously examined this relationship using an adoption design. This analysis makes a unique contribution to the literature because we included adoptive parent-child dyads, allowing us to unpack the relationship between parents' beliefs and adolescent SU by statistically adjusting for shared genes and testing biological relation as a moderator.

### **Limitations**

A primary limitation of this analysis was that all data were self-reported (either by the parent or adolescent), so responses were likely influenced by social desirability bias. The longitudinal design allowed for a time-ordered investigation into SU frequency; however, it is possible that adolescent initiation of SU preceded the collection of their parent's beliefs in the lifetime SU models. Using a lifetime use measure of SU did not allow for analyses on the timing of SU initiation (i.e., adolescents who initiated use at 14y were treated the same as those who initiated use at 18y). Although this was not an issue in the SU frequency models which looked at past-year use, adolescents who reported not using in the past year were combined with those who had never used. This approach may have obscured important distinctions in pathways to adolescent SU and the prevention needs of these distinct groups. Most reporting parents were female, so it is possible that these findings do not generalize to fathers. Finally, these results were drawn from a sample of mostly adopted adolescents from primarily upper middle-class families, so they cannot be generalized to all adolescents.

## **Future Directions**

The current study did not address mechanisms that might account for the association between parental beliefs and adolescent SU outcomes. There are potentially influential mediators and moderators of this relationship that should be tested, such as parenting practices (e.g., monitoring, open communication, limit-setting) and peer SU. Adolescents have been found to affiliate with peers who hold similar attitudes towards SU as their parents (Mrug & McCay, 2013), indicating that for parents who perceive low risk in SU, associations with adolescent SU may be amplified by peers with similar perceptions of low risk or attenuated for peers perceiving high risk of SU. Additionally, as relational tension between parents and their children is associated with an increased risk of adolescent substance use (Latendresse et al., 2009) and may affect the adolescent's receptivity to their parent's substance-related beliefs, relationship quality also should be tested as a moderator. The parent study continues to collect data from the parent-child dyads included in this analysis, providing future opportunities to explore how parental beliefs could influence SU beyond adolescence and into emerging adulthood.

## **Compliance with Ethical Standards**

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### **Ethics Approval**

The original study was conducted in compliance with the Institutional Review Boards that oversaw the original study.

### **Conflicts of Interest/Competing interests**

The authors have no relevant financial or non-financial interests to disclose.

### **Consent to participate**

All adult participants provided informed consent and child participants provided assent prior to participating in the original study.

### **Clinical Trial Registration**

Clinical trial number: not applicable.

### **Author Contribution Statement**

A.T. conceived of the idea, conducted the analyses, wrote the original draft, and edited subsequent drafts. K.M., J.N., J.G., and L.D.L. secured funding, selected the measures, and oversaw data collection activities. K.M., J.N., J.G., M.N., D.S., and L.D.L. reviewed and edited manuscript drafts. L.D.L. supervised this research project.

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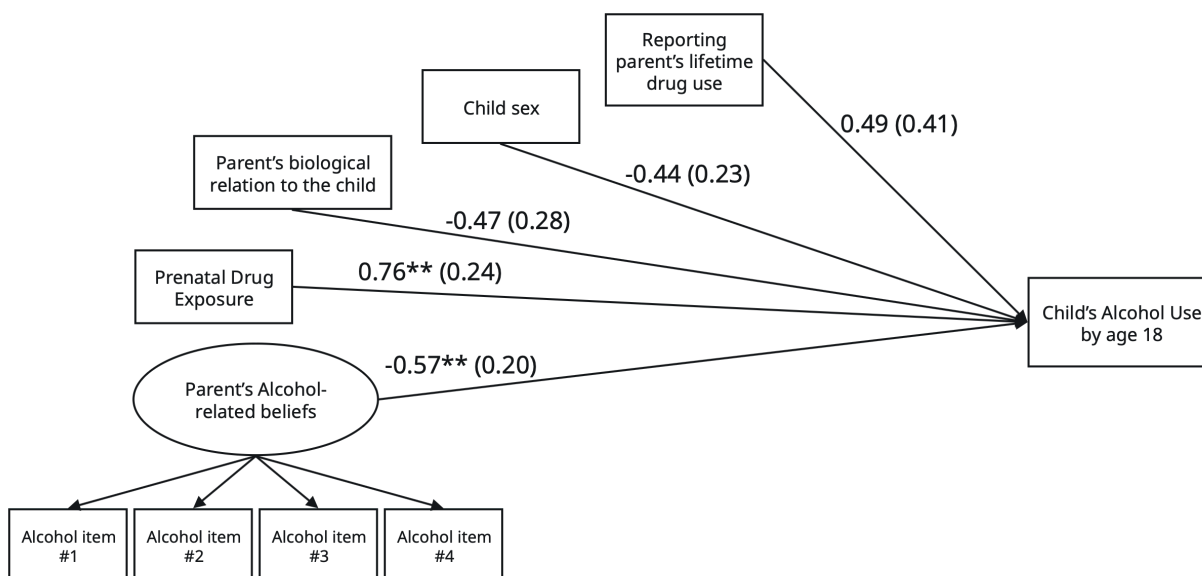
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## Tables and Figures

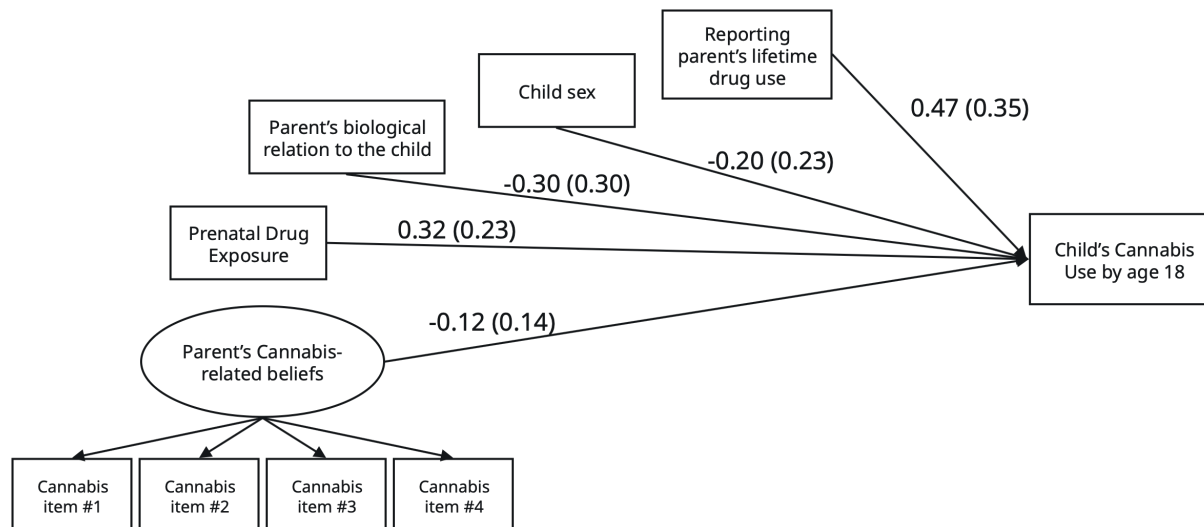
**Table 1.**

Child and Parent Participant Characteristics

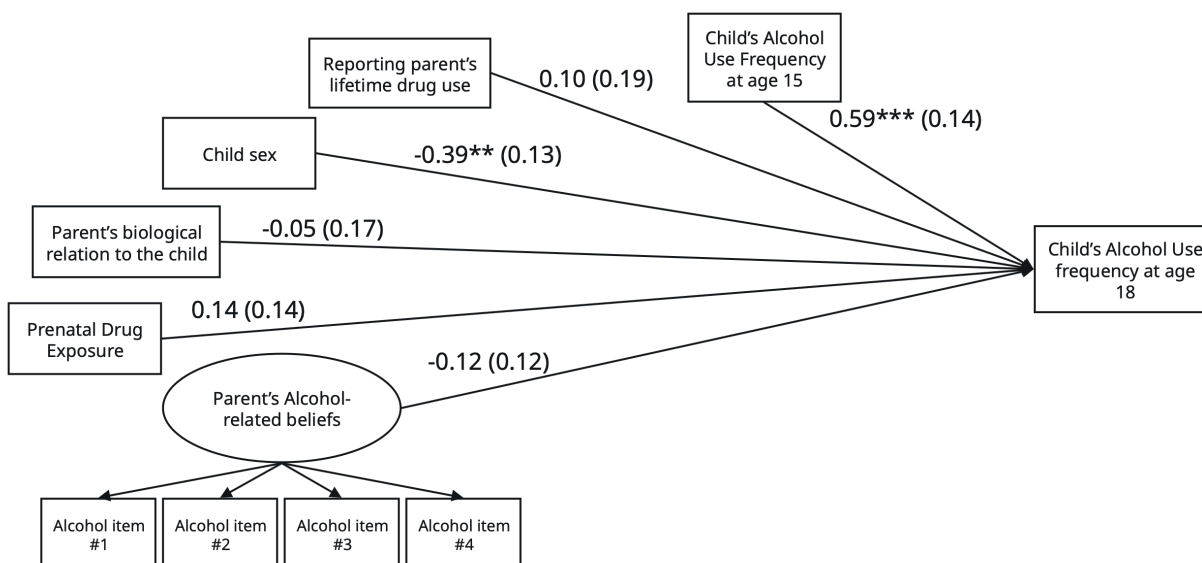
	<b>Mean</b>	<b>SD</b>	<b>N</b>	<b>Valid %</b>
<b>Child Age at 15y visit</b>	16.0	0.6		
<b>Child Age at 18y visit</b>	18.9	0.7		
<b>Child Type</b>				
Adoptee			201	70.8
Adoptee's sibling in the Adoptive Home			43	15.1
Adoptee's sibling in the Birth Home			40	14.1
<b>Parent's Biological Relation to Child</b>				
Biologically Related			57	20.1
Non-biologically Related			227	79.9
<b>Child Race</b>				
White			164	57.7
More than 1 race			73	25.7
Black/African American			45	15.8
Asian			1	0.4
Unknown			1	0.4
<b>Child Ethnicity</b>				
Hispanic or Latino			37	13.0
Not Hispanic or Latino			247	87.0
<b>Child Sex Assigned at Birth</b>				
Female			130	45.8
Male			154	54.2
<b>Prenatal Exposure to Substances</b>				
Yes			111	49.1
No			115	50.9
<b>Parent Sex</b>				
Female			201	85.5
Male			34	14.5
<b>Parent's Lifetime History of Serious Substance Use</b>				
Had lifetime serious use of at least one drug			30	13.6
Did not have lifetime serious use of at least one drug			191	86.4

**Figure 1a.****Alcohol Lifetime Use Model**

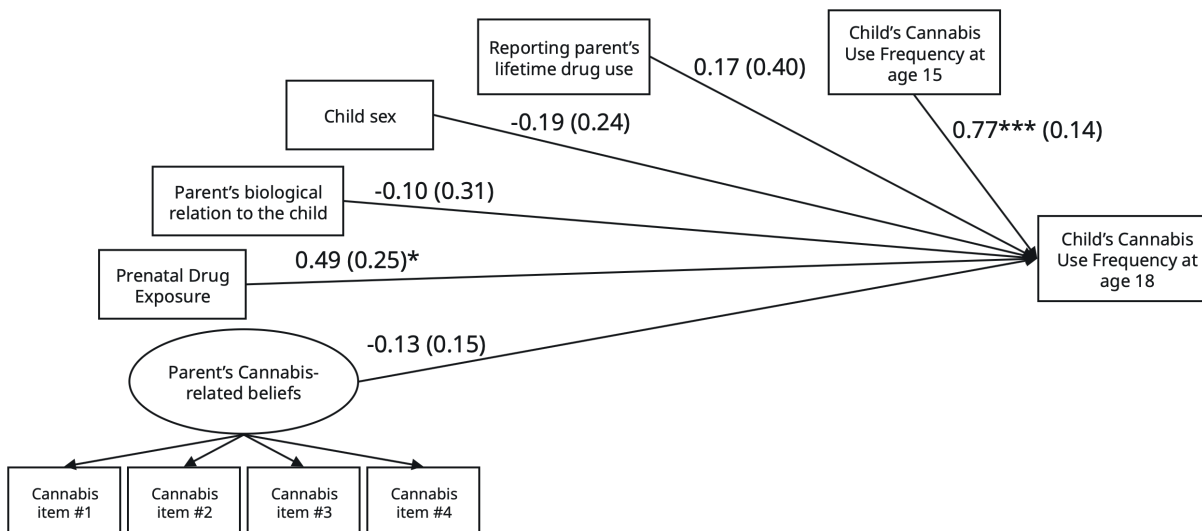
*Note.* Estimates reflect probit regression coefficients and standard errors from a WLSMV structural equation model for binary outcomes.

**Figure 1b.****Cannabis Lifetime Use Model**

*Note.* Estimates reflect probit regression coefficients and standard errors from a WLSMV structural equation model for binary outcomes.

**Figure 2a.****Alcohol Use Frequency Model**

*Note.* Estimates reflect unstandardized coefficients and standard errors from a linear regression model fit using the MLR estimator.

**Figure 2b.****Cannabis Use Frequency Model**

*Note.* Estimates reflect unstandardized coefficients and standard errors from a linear regression model fit using the MLR estimator.

### Online Resource 1

**Supplementary Table 1.**

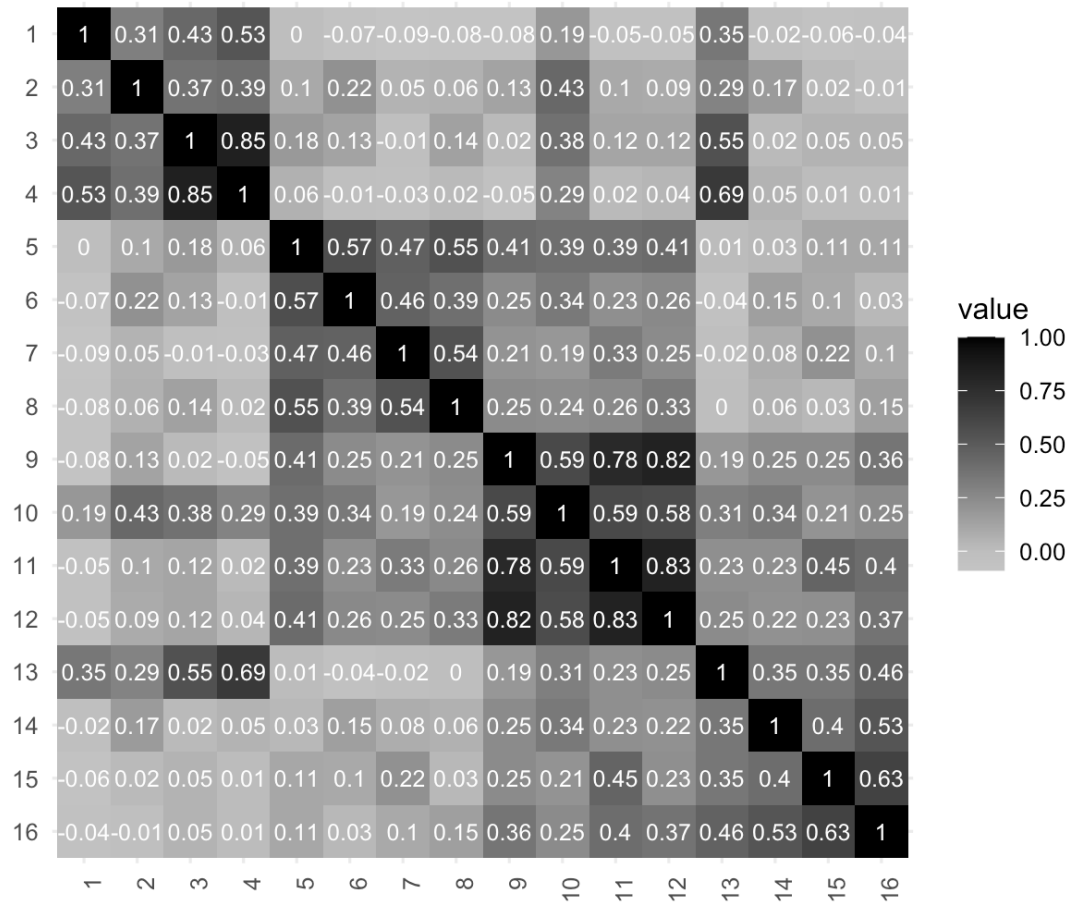
Parent's Substance-Related beliefs items

	<b>Mean</b>	<b>SD</b>	<b># of Respondents</b>
1. If you are young and healthy, smoking is not dangerous.	1.2	0.8	224
2. A person has to smoke for many years before it is likely to affect his/her health.	1.3	0.9	224
3. Smoking is OK as long as you don't use them too much.	1.1	0.5	224
4. A person who eats right and exercises regularly can smoke without harming his/her health.	1.1	0.4	224
5. If you are young and healthy, drinking is not dangerous.	1.6	0.9	224
6. A person has to drink for many years before it is likely to affect his/her health.	1.7	1.1	224
7. Drinking is OK as long as you don't use them too much.	3.1	1.4	224
8. A person who eats right and exercises regularly can drink without harming his/her health.	2.1	1.2	223
9. If you are young and healthy, marijuana is not dangerous.	1.6	1.0	222
10. A person has to use marijuana for many years before it is likely to affect his/her health.	1.6	1.0	221
11. Marijuana use is OK as long as you don't use them too much.	1.9	1.2	222
12. A person who eats right and exercises regularly can use marijuana without harming his/her health.	1.7	1.1	222
13. If you are young and healthy drug use is not dangerous.	1.1	0.5	223
14. A person has to use drugs for many years before it is likely to affect his/her health.	1.1	0.4	223
15. Using drugs is OK as long as you don't use them too much.	1.2	0.7	223
16. A person who eats right and exercises regularly can use drugs without harming his/her health.	1.1	0.4	222

*Note.* Participants rated their agreement from 1-5 on each of these items, which a higher score indicating a higher level of agreement with the item. When used as a predictor in structural equation models, these items were reverse coded so that higher scores indicated a greater perception of risk.

### Supplementary Figure 1.

Correlation Matrix of Parent's Substance-Related Beliefs Items



*Note.* See Supplementary Table 1 above for scale items.



## Online Resource 2

Supplementary Table 2.

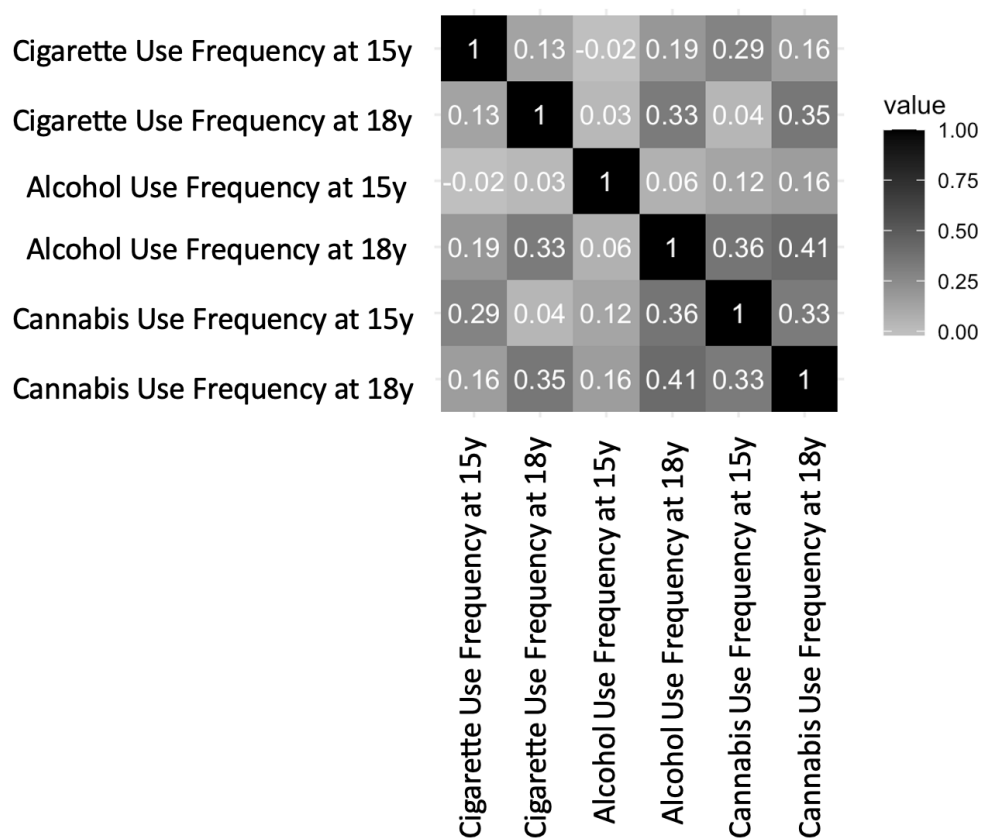
Youth drug use frequency

	Count (15y)	% (15y)	Count (18y)	% (18y)
<b>Cigarettes</b>				
Never	203	98.1	168	89.4
Less than once per month	4	1.9	11	5.9
Less than once per week	0	0	6	3.2
1-2 days per week	0	0	6	3.2
3-5 days per week	0	0	1	0.5
Almost every day or every day	0	0	1	0.5
<b>Alcohol</b>				
Never	202	97.1	131	69.7
Less than once per month	4	1.9	34	18.1
Less than once per week	1	0.5	17	9.0
1-2 days per week	0	0	4	2.1
3-5 days per week	0	0	0	0
Almost every day or every day	0	0	0	0
<b>Cannabis</b>				
Never	187	90.3	131	69.0
Less than once per month	9	4.3	11	5.8
Less than once per week	4	1.9	10	5.3
1-2 days per week	2	1.0	18	9.5
3-5 days per week	3	1.4	22	11.6
Almost every day or every day	0	0	0	0

*Note.* The number of respondents varies by item because there was differential missingness on each item. Other drug use frequency was not reported here because it was captured using different measures for each type of other drug.

## Supplementary Figure 2.

Youth Substance Use Frequency Correlation Matrix



**Supplementary Table 3.**

Youth lifetime substance use by age 18

	<b>Count of Respondents indicating use</b>	<b>Total Respondents</b>	<b>Valid Percent</b>
Cigarettes	61	203	30.0%
Alcohol	182	224	81.3%
Cannabis	121	207	58.5%
Other Drugs	39	190	20.5%

*Note.* The number of respondents varies by item because there was differential missingness on each item.

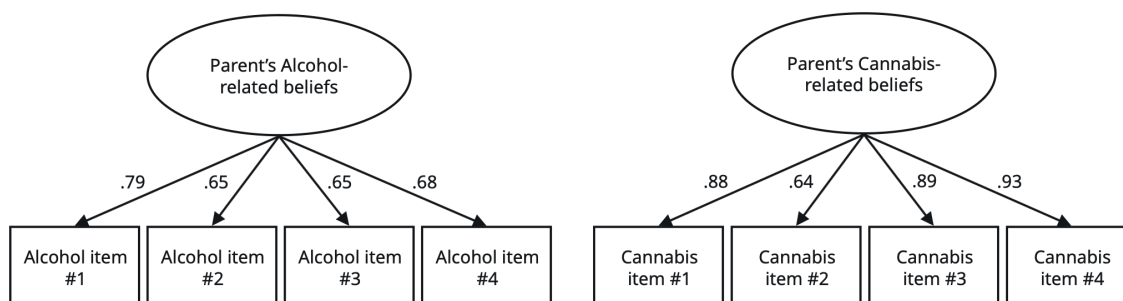
## Online Resource 3

Supplementary Table 4.

Two-factor model fit

Fit Indices	Two-factor Model (selected model)	Higher-order two-factor Model
Chi Square	47.15***	44.91***
CFI	0.95	0.95
TLI	0.92	0.92
RMSEA	0.07	0.07
SRMR	0.05	0.05
Model Issues	<ul style="list-style-type: none"> <li>None detected</li> </ul>	<ul style="list-style-type: none"> <li>The model was not identified when fit to 11/20 imputed datasets</li> </ul>

*Note.* \*\*\*  $p < .001$ ; Model fit indices were pooled across 20 imputed datasets using the D2 method and the MLR estimator, which provides robust standard errors and scaled test statistics that account for non-normality.

**Supplementary Figure 3.****Two-factor Substance-specific Latent Variables**

*Note.* Standardized factor loadings are displayed for each path.