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# Developmental epidemiology of drug use and abuse in adolescence and young adulthood: Evidence of generalized risk

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## Abstract

Past studies highlight a narrowing gender gap and the existence of a shared etiology across substances of abuse; however, few have tested developmental models using longitudinal data. We present data on developmental trends of alcohol, tobacco, and marijuana use, abuse and dependence assessed during adolescence and young adulthood in a community-based Colorado twin sample of 1733 respondents through self-report questionnaires and structured psychiatric interviews. Additionally, we report on the rates of multiple substance use and disorders at each developmental stage, and the likelihood of a substance use disorder (SUD; i.e., abuse or dependence) diagnosis in young adulthood based on adolescent drug involvement. Most notably, we evaluate whether the pattern of multiple substance use and disorders and likelihood ratios across substances support a model of generalized risk. Lastly, we evaluate whether the ranked magnitudes of substance-specific risk match the addiction liability ranking. Substance use and SUDs are developmental phenomena, which increase from adolescence to young adulthood with fewand inconsistent gender differences. Adolescents and young adults are not specialized users, but rather tend to use or abuse multiple substances increasingly with age. Risk analyses indicated that progression toward a SUD for any substance was increased with prior involvement with any of the three substances during adolescence. Despite the high prevalence of alcohol use, tobacco posed the greatest substance-specific risk for developing subsequent problems. Our data also confirm either a generalized risk or correlated risk factors for early onset substance use and subsequent development of SUDs.

### Keywords

Adolescence; Young adulthood; Risk; Alcohol; Tobacco; Marijuana



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Authors John Hewitt, Thomas Crowley, Mike Stallings, Susan Young, Robin Corley, and Christian Hopfer designed the study, wrote the protocols, and managed the data collection. Author Thomas Crowley is also the principal investigator of the Center for Antisocial Drug Dependence. Author Robin Corley managed the incorporation of data for analysis. Authors Rohan Palmer, Christian Hopfer, and Susan Young managed the literature searches and summaries of previous works. Author Rohan Palmer undertook the statistical analyses and wrote the first draft of the manuscript under John Hewitt's mentorship. All authors contributed to and have approved the final manuscript.

**Conflict of interest**

All of the listed authors declare that they have no conflicts of interests.

## 1. Introduction

Epidemiologic studies have demonstrated that the prevalence of drug use and abuse increases with age during adolescence and peaks in young adulthood (Johnston et al., 2008; Substance Abuse and Mental Health Services Administration (SAMHSA), 2006, 2008). Substance experimentation is common in adolescence and substantially elevates the risk for persistent substance use, substance use disorders (SUDs; i.e., abuse/dependence) and other comorbid disorders in later life stages (Bauman and Phongsavan, 1999; Brook et al., 1999; Gould et al., 1977; Kapusta et al., 2007; Riggs et al., 2007;Winters and Lee, 2008). Additionally, involvement with multiple substances and the risk for substance use problems appear to be driven by either a common risk factor or correlated risk factors (Grant and Dawson, 1998; Gil et al., 2004; Grant et al., 2006; Kendler et al., 2007, 2008; Rhee et al., 2003, 2006; Young et al., 2006). Despite this wealth of information, our understanding of substance use and related problems in youth remains limited by the fact that most data are cross-sectional. This permits investigation of cohort differences or temporal change, but does not allow for developmental models to be explicitly tested.

This study extends our previous work on adolescent substance use patterns among community adolescents in Colorado (Young et al., 2002) who have now been followed longitudinally into young adulthood, by addressing two primary questions that remain largely unanswered. First, are there gender differences in the developmental trends in substance use, abuse, and dependence? Second, is there epidemiological evidence to support a model of generalized risk across alcohol, tobacco, and marijuana use and disorders?

### 1.1. Gender differences in developmental trends of substance use and disorders

Although gender differences in alcohol use in adolescence are typically modest (Hicks et al., 2007; Johnston et al., 2008; Young et al., 2002), adult studies consistently show higher levels of alcohol consumption and a greater prevalence of alcohol use disorders amongmen(Prescott, 2001, 2003; SAMHSA, 2008; Sher et al., 2005). However, recent reports show that the gender gap in drinking behavior has narrowed in recent decades (Holdcraft and Iacono, 2002; Keyes et al., 2008; Rice et al., 2003), whichmay be attributed, in part, to changes in cultural attitudes toward drinking (Holmila and Raitasalo, 2005). Based on large epidemiologic surveys such as the Monitoring the Future Survey (MTF; Johnston et al., 2008), we would expect only modest gender differences in cigarette and marijuana smoking in adolescence. Their data on young adults show that while rates of marijuana use are higher among males in this age range, gender differences in rates of cigarette smoking remain small. Although these cross-sectional data do not address developmental issues, they are consistent with one of the few longitudinal studies of substance use in adolescence and young adulthood, which also shows that substance related problems in males escalate in young adulthood at a greater rate than in females (Hicks et al., 2007). Based on these data, we anticipated only modest gender differences in our Wave 1 adolescent data, and that these differences would expand in our Wave 2 young adult data, particularly for alcohol and marijuana.

### 1.2. Generalized versus substance-specific risk of substance use and disorders

The substance abuse literature documents an ongoing interest in understanding the risk factors underlying substance use and related disorders. While substance-specific factors have been supported, the high rates of comorbidity among nicotine, alcohol, and illicit substances have led many researchers to concentrate on risk factors that may be common across multiple substances. For example, Rhee and colleagues (2003) utilized simulated family data to discriminate between 13 different models of comorbidity defined by Neale and Kendler (1995), and then applied this methodology in a clinically ascertained adolescent sample (Rhee et al., 2006). Results suggested that two models were equally likely to explain the patterns of

comorbidity observed. The first model, referred to as the correlated liability model, hypothesizes that each substance has its own set of risk factors (i.e., liability) and that these factors are correlated, accounting for poly-substance use and comorbid SUDs. The second plausible model, the alternate forms model, hypothesizes that comorbidity is driven by a single risk factor which manifests itself as an array of deviant behaviors, including substance use and SUDs. Both models are consistent with observed prevalence rates of comorbidity that exceed those predicted from the rates of use, abuse, and dependence on individual substances when assuming independent liabilities. In the current study we examine the patterns of single and multiple drug involvement in both adolescence and young adulthood while making comparisons to expected rates based on a model of independent liabilities. By comparing the expected and observed rates of multiple substance use and SUDs we are able to test if these liabilities are independent. We hypothesized that the patterns of multiple substance use and abuse in adolescence and young adulthood would support a model of generalized risk. Additionally, given that young adulthood is a period during which drug initiation and progression to problematic use remains frequent (Wagner and Anthony, 2002), we also hypothesized that the prevalence of lifetime multiple substance use and abuse would increase from adolescence to young adulthood.

Data from prospective longitudinal studies have consistently shown that the onset of substance use in adolescence confers a particularly potent risk for persistent use and the development of substance use disorders (Bonomo et al., 2004; Brook et al., 1999; Duncan et al., 1997; Gil et al., 2004; Lewinsohn et al., 1999; McGue and Iacono, 2008; Timberlake et al., 2007). These data can provide another mechanism for examining possible generalized risk factors by comparing the probability of developing a SUD on substance A, given adolescent use of substance A versus adolescent use of substance B. For example, does smoking in adolescence primarily predict tobacco/nicotine dependence in young adulthood and only minimally predict problems with other substances (evidence of specific risk)? Alternatively, is adolescent smoking as predictive of later marijuana abuse or dependence as it is predictive of later nicotine dependence (evidence of generalized risk)? Using longitudinal data collected in adolescence and young adulthood, we asked whether the likelihood of developing an alcohol, tobacco, or marijuana SUD during young adulthood depends on adolescent drug involvement with a particular substance or any substance.

With our third analytical approach we investigated whether onset of use in adolescence predicts the development of a SUD in young adulthood to the same degree for tobacco, alcohol, and marijuana. Goldstein and Kalant (1990) rank ordered the relative risk of addiction of several drug categories by the “addictiveness” of each drug based on animal studies examining self-administration of substances, engagement in drug seeking behavior, and latency to relapse after enforced abstinence. The addiction liability ranking suggested that tobacco was the most addictive of the three substances, followed by alcohol, and lastly marijuana (Goldman et al., 2005; Goldstein and Kalant, 1990). Based on these findings we predicted that the magnitude of risk for a SUD in young adulthood, given adolescent onset of use, would be consistent with the addiction liability ranking. If supported, this pattern would suggest that in addition to generalized risk factors, there are also important substance-specific mechanisms that increase the risk for developing SUDs.

## 2. Methods

### 2.1. Sample description

Subjects in the study were 1733 individual members of twin pairs drawn from the Colorado Community Twin Study (CTS) and Longitudinal Twin Study (LTS) who also participated in the Center for Antisocial Drug Dependence (CADD) study at the University of Colorado (Rhea et al., 2006). Participants in this study are part of a community-based twin sample of individuals

that had completed two waves of assessment. The CTS sample was identified for recruitment through the Colorado Department of Health (CDH), Division of Vital Statistics, supplemented later by Colorado school districts. All members of the LTS were recruited at birth through the CDH and most have participated in the study repeatedly from infancy to the present. A total of 1142 CTS and 591 LTS respondents participated at both waves. Details of the recruitment procedures are provided by Rhea et al. (2006).

The interval between the two waves of assessment was approximately 5 years (mean = 5.06; standard deviation (S.D.) = 0.65). DuringWave 1 the twin sample had an age range of 11.5–18.49 years (mean age = 14.73 years; S.D. = 2.14). At the Wave 2 follow-up, the sample had an age range of 16.50–25.49 years (mean age = 19.79 years; S.D. = 2.35). We define adolescence as the age range 12–18 and young adulthood as ages 17–25 (with the overlapping ages 17 and 18 representing transitional years into young adulthood). With the exception of age category 23+, in our analyses and presentation of results the age of each respondent was rounded to the nearest year and placed into separate age categories (12–22); the age 23+ category was composed of respondents between the ages of 22.5 and 25.49 (mean age = 23.47, S.D. = 0.63). Table 1 provides the racial composition and age distribution by gender at each wave. The racial distribution of the twin sample is comparable to that of the state of Colorado between the years of birth of the respondents (1981–1990) (Colorado Department of Public Health and Environment, State birth rates from 1979 to 2004). The sample was predominantly made up of complete twin pairs (818 pairs; 97 single twins) and an equivalent distribution of participants of each gender across all age categories. A comparison of subjects without data available at Wave 2 and those being examined in this study revealed similar rates of substance use and disorders in Wave 1.

### 2.2. Assessments

Data gathered on lifetime substance experimentation and repeated-use were collected using a MTF-based (Johnston et al., 1986) self-report questionnaire. Data on abuse and dependence were collected using the Composite International Diagnostic Interview-Substance Abuse Module (CIDI-SAM) structured interview (Cottler et al., 1989; Üstűn et al., 1997). The CIDI-SAM is a face-to-face interview administered by lay interviewers that has been demonstrated to be a reliable and useful diagnostic tool for gathering data on drug abuse and dependence in both clinical and general research settings (Cottler et al., 1989; Crowley et al., 2001; Üstűn et al., 1997). At Wave 1, the CIDI-SAM was administered using a paper and pencil interview protocol while at Wave 2 a computerized version was employed.

Experimentation was defined for all substances as having used a substance one or more times in a person's lifetime. Repeated alcohol use was defined as having had six or more drinks in a respondent's lifetime. Repeated tobacco (cigarette) use was defined as 'recently smoking regularly' or 'smoking regularly in the past', allowing each respondent to define "regular smoking" themselves. Repeated marijuana use was defined as having used marijuana (grass, pot, or hashish) six or more times in a respondent's lifetime. Abuse and dependence categories adhered to the 1+ and 3+ symptom endorsement guidelines (for abuse and dependence, respectively) of the Diagnostic and Statistical Manual of Mental Disorders 4$^{th}$ Ed. (DSM-IV; American Psychiatric Association (APA), 1994) but without imposing the clustering criterion or distinguishing between dependence with or without physiological symptoms. At Wave 1, clustering would have slightly reduced the prevalence of SUD diagnoses for alcohol (6-5.88%), marijuana (3.81-3.70%), and tobacco dependence (3.46-2.37%); based on this observation we chose not to impose clustering. Unlike the experimentation and repeated-use categories, DSM-IV abuse and dependence are mutually exclusive diagnoses, such that a diagnosis of dependence precludes a diagnosis of abuse. Furthermore, abuse and dependence (with and without physiological symptoms) are both outcomes of drug use; as such we created a

composite measure, lifetime SUD, that indicated the presence or absence of an abuse or dependence diagnosis.

### 2.3. Statistical analysis

Prevalence rates for lifetime substance experimentation, repeated-use and SUDs, as well as odds-ratio estimates were computed using SAS® (SAS Institute Inc., 1987). Chi-square analyses and Fisher's exact tests were used to compare experimentation, repeated-use and SUD rates by gender after accounting for the non-independence in the data by creating a weighting scheme based on the number of individuals in a family unit. Individuals from a family of complete twin pairs were given a weight of 0.50 while individuals from an incomplete twin pair were given a weight of 1.0 (Young et al., 2002). This provided a conservative estimate of gender and group differences in the sample. Weighted and unweighted results are provided in the tables. Log-linear analyses were utilized to assess the propensity to repeatedly use multiple substances or to be diagnosed with multiple SUDs. The odds in favor of developing a lifetime diagnosis of a SUD as opposed to a lifetime report of either abstinence or some form of non-problematic use (i.e., defined here as experimentation or repeated-use without an abuse or dependence diagnosis) 5 years later were determined using weighted logistic regression. Odds-ratios in favor of a SUD diagnosis for a given substance were adjusted for age (mean deviated), gender, and substance use and SUD patterns for other substances at Wave 1. Because we utilized lifetime measures of drug involvement, respondents could not report a lesser level of drug involvement with a given substance in the second wave. For instance, a person who was identified as a repeat-user (with no SUD diagnosis) can later be classified as still being a repeat-user (with no SUD diagnosis) or as having a SUD, but not as an abstainer. Respondents who violated this protocol were not included in the weighted logistic regression analyses predicting SUDs at Wave 2.We found that roughly 2.4–4% of the sample gave inconsistent responses, depending on the predicted substance.

## 3. Results

### 3.1. Substance experimentation, repeated-use, and SUD overall prevalence rates

Prevalence rates of experimentation, repeated-use, and SUDs at each wave for each gender are presented in Table 2. At each wave, alcohol was the substance most experimented with, repeatedly used, and abused, while tobacco was the substance with the highest prevalence of dependence. As expected, the prevalence of each category markedly increased between waves of assessment. At Wave 1, there were no significant gender differences overall with respect to each level of substance involvement; however, by Wave 2 a significantly larger proportion of males were involved with most substances.

### 3.2. Substance experimentation, repeated-use and SUD developmental trends and gender differences

Table 3 presents the prevalence rates of experimentation, repeated-use and SUDs for each substance at each age and wave of assessment for each gender. The prevalence of experimentation, repeated-use, and SUDs gradually increased between 12 and 22 years of age with few and inconsistent gender differences except in the case of alcohol and marijuana SUDs in young adulthood. The data also show a difference in the prevalence rates between 17- and 18-year olds tested at Wave 1 and Wave 2. The decreases from Wave 1 to Wave 2 may in part reflect the cohort effect observed in the MTF and other studies. An alternative explanation may be that our LTS sample (ascertained from birth records and followed annually from birth) which contributed many of the 17- and 18-year-old subjects at Wave 2, may have lower substance involvement than our CTS sample (ascertained as adolescents through the school system) that contributed the 17- and 18-year olds at Wave 1. Another possibility is that the computerized administration of the CIDI-SAM at Wave 2 may have led to slightly different prevalence

estimates than those elicited by the paper and pencil interview format at Wave 1. Finally, being assessed for a second time may have resulted in lower endorsement of behavior. Although we are not able to disentangle these contributions to the lower prevalence estimates for 17- and 18-year olds at Wave 2, it is apparent that at older ages the prevalence rates for Wave 2 are in line with what would be expected projecting forward from Wave 1.

### 3.3. Single and multiple substance repeated-use and SUDs

Table 4 outlines the prevalence of having repeatedly used one or multiple substances in a person's lifetime, the prevalence of being diagnosed with one or multiple SUDs (i.e., for different substances) in a person's lifetime, and the expected prevalence of multiple repeated-use and SUDs at adolescence and young adulthood. The expected prevalence for multiple substances is computed on the assumption of independence among substances (details of the computation of the expected values to follow). The prevalence of lifetime multiple substance repeated-use and multiple SUDs increased between waves of assessment. Repeated-use of alcohol alone and alcohol SUDs alone were the most prevalent categories at each wave of assessment while "alcohol and marijuana" repeated-use was the most prevalent multiple substance repeated-use category at each wave. Of the multiple SUD categories, "tobacco and marijuana" was the most prevalent category at Wave 1 while "alcohol, tobacco, and marijuana" was the most prevalent category at Wave 2.

A log-linear analysis was used to assess the significance of multiple substance repeated-use or non-use compared to expectations based on the prevalence for each substance independently. For example, based on the Wave 2 prevalence of repeated alcohol use (65.73%), repeated tobacco use (20.50%), and repeated marijuana use (33.45%), we would expect the prevalence of using all three substances to be 4.51% (i.e., 0.6573×0.2050×0.3345×100) and the expected prevalence of repeatedly using only alcohol to be 34.78% (i.e., 0.6573×(1−0.2050)×(1−0.3345) ×100) if the risk for use for each substance was independent of the use of another substance. At each wave, the observed prevalence of the lifetime repeated-use of all three substances exceeded expectations. At Wave 1, 4.86% of the sample reported repeatedly using all three substances at some point in their lifetime, compared to the expected prevalence of 0.17%. This observation provided evidence of a general tendency to repeatedly use all three substances. By Wave 2, the prevalence of repeated-use of all three substances had increased to 15.45%. Similarly, the observed prevalence of non-repeated-use of all three substances at each wave (Wave 1, 75.29%; Wave 2, 32.77%) exceeded what was expected (Wave 1, 64.48%; Wave 2, 18.13%). These excess observations were balanced by corresponding deficits in the numbers using only one or two substances repeatedly or non-repeatedly. The overall $\chi^2$ (with four degrees of freedom) testing both two way and three way interactions was highly significant (Wave 1: $\chi^2 = 754.62$, $p<0.001$; Wave 2: $\chi^2 = 470.42$, $p < 0.001$). Similarly, the observed prevalence of having a SUD on two or more substances also exceeded chance expectations at both developmental stages. At each wave, the overall $\chi^2$ was highly significant (Wave 1: $\chi^2 = 292.17$, $p<0.001$; Wave 2: $\chi^2 = 570.77$, $p < 0.001$). Consequently, the elevated prevalence of the multiple SUD categories was balanced by the decline in the prevalence of the single SUD categories at each Wave. Further examination revealed that the observed prevalence of repeated-use and abuse of all three substances was increasingly greater than the expected prevalence as age increased. Overall, these trends in single and multiple substance repeated-use and disorders imply that the risk for repeated-use and SUDs of alcohol, tobacco, and marijuana are not independent.

### 3.4. Risk estimates for the development of a substance use disorder at Wave 2

Table 5 describes the odds-ratio in favor of a lifetime diagnosis of a SUD for each substance as a function of a person's Wave 1 status (i.e., abstinence, use 1+ times without a disorder diagnosis, and SUD) with each of the three substances. Odds-ratios were determined for the

following comparisons: 'Use 1+ times without a SUD vs. abstinence', 'SUD vs. abstinence' and 'SUD vs. Use 1+ times without a SUD'. The table reports the odds-ratio for two separate models. The unadjusted model (OR; Eq. (1)) determined each odds-ratio controlling for the mean deviated age (i.e., age of each respondent minus the mean age of sample), and gender. The model also included an interaction of the Wave 1 comparison predictor with age and gender.

Equation 1—OR model (odds-ratios adjusted for age and gender)

$$\begin{aligned}&\text{Log (Odds of Alcohol SUD at Wave 2 is TRUE)}\\&=\beta_0+\beta_1(\text{Wave 1 Alcohol Status Comparison})+\beta_2(\text{Age})\\&+\beta_3(\text{Gender})+\beta_4(\text{Wave 1 Status Comparison}\times\text{Age})\\&+\beta_5(\text{Wave 1 Status Comparison}\times\text{Gender})+\varepsilon_i\end{aligned}\tag{1}$$

The adjusted model (AOR; Eq. (2)) was similar to the OR model but also controlled for the level of adolescent involvement with the other two substances not being predicted by the model.

Equation 2—AOR model (odds-ratios adjusted for age, gender, and drug involvement in Wave 1)

$$\begin{aligned}&\text{Log (Odds of Alcohol SUD at Wave 2 is TRUE)}\\&=\beta_0+\beta_1(\text{Wave 1 Alcohol Status Comparison})+\beta_2(\text{Age})\\&+\beta_3(\text{Gender})+\beta_4(\text{Wave 1 Status Comparison}\times\text{Age})\\&+\beta_5(\text{Wave 1 Status Comparison}\times\text{Gender})\\&+\beta_6(\text{Wave 1 Level of Tobacco Involvement})\\&+\beta_7(\text{Wave 1 Level of Marijuana Involvement})+\varepsilon_i\end{aligned}\tag{2}$$

All analyses were weighted by family structure in order to obtain conservative odds-ratios. Controlling for levels of involvement with substances not predicted by the model removed the effects of these substances on the likelihood of the predicted outcome; providing a direct relationship between the Wave 1 comparisons and the likelihood of the disorder at Wave 2 within and between substances.

### 3.5. Generalized and substance-specific risk for young adult substance problems

The distribution of significant odds-ratios of the OR models presented in Table 5 revealed that the risk of abuse/dependence in young adulthood was elevated once a person had used any substance at least once. For instance, the likelihood of developing a lifetime diagnosis of an alcohol SUD at Wave 2 was at least two times greater when comparing persons who experimented with alcohol, tobacco, or marijuana in adolescence to persons who abstained (OR model), and at least eight times greater if they were dependent on tobacco as opposed to being abstinent. More importantly, correcting for involvement with other substances during adolescence revealed significant relationships within and between substances across all three Wave 1 comparisons. For example, use or dependence on tobacco during adolescence elevated the likelihood of not only tobacco dependence in young adulthood but also alcohol and marijuana problems. It is important to note that, among statistically significant findings, the comparison between the odds-ratios of the OR and the AOR models revealed minor differences between the models, providing strong evidence of a common or shared liability between the substances. Significant odds-ratios from the OR model that are no longer significant in the AOR model indicate that these associations may have been driven by the other substances. Lastly, the rank ordering of the magnitude of the substance-specific risk based on adolescent drug use was greatest for tobacco (5.89), followed by marijuana (2.91) and alcohol (1.93).

## 4. Discussion

This study goes beyond its predecessors by employing prospective longitudinal data from a community sample in order to (1) assess the developmental patterns of substance use and SUDs, including a gender comparison, and (2) examine whether comorbidity across substances as well as the progression from use to SUD support a generalized versus substance-specific model of liability for alcohol, tobacco, and marijuana related problems. Furthermore, it adds to the limited number of studies that have explored the comorbidity between substances across developmental stages. Significant age trends confirm that alcohol, tobacco, and marijuana use and SUDs are developmental phenomena which increase gradually with age. Risk models suggest that while being exposed to a particular substance in adolescence increases the risk for problems with the same substance later in young adulthood, the risk is also elevated once there is involvement with other substances. Lastly, the magnitude of substance-specific risk differs between substances; tobacco confers the greatest degree of risk of a SUD diagnosis followed by marijuana and alcohol.

### 4.1. Comparison of prevalence rates and trends with nationally representative samples

Overall, our rates and ranking of alcohol, tobacco, and marijuana use and disorders were comparable to recent findings on national samples such as the MTF (Johnston et al., 2008) and the National Survey on Drug Use and Health (NSDUH; SAMHSA, 2008). Likewise, the level of alcohol use and SUD prevalence rates during young adulthood is consistent with past studies (Sher et al., 2005). Our data also confirm that experimentation, repeated-use, and SUD prevalence rates increase approximately linearly with age up to young adulthood. This was consistent with the earlier findings by Young et al. (2002) and other nationally representative samples (Bauman and Phongsavan, 1999; Johnston et al., 2008; SAMHSA, 2006, 2008). Despite the low prevalence of adolescent SUDs in our community-based sample, within 5 years these rates were at least doubled. Alcohol accounted for most of the abuse diagnoses in our population, while, not unlike findings from the National Comorbidity Survey, the highest rate of substance dependence was observed with tobacco at both waves of assessment (Anthony et al., 1994). Overall, despite the high prevalence of alcohol, tobacco, and marijuana repeated-use in our sample, the rates of disorders are consistent with the fact that not all users go on to develop problems of substance abuse or dependence (Anthony and Helzer, 1995; SAMHSA, 2008; Young et al., 2002).

### 4.2. Gender differences in substance use and SUDs in adolescence and young adulthood

With the exception of alcohol disorders, gender differences were few, inconsistent, and did not become apparent until late adolescence and young adulthood, similar to trends observed in the MTF and NSDUH studies (Johnston et al., 2008; SAMHSA, 2008). Gender differences in alcohol disorders beyond age 18 were consistent with past studies (Sher et al., 2005) which showed that young adult males were more frequently diagnosed with alcohol SUDs. On the other hand, rates of tobacco dependence were similar for males and females during and after adolescence. Gender differences in marijuana SUDs were not marked during adolescence but SUDs were more prevalent in males in young adulthood. Thus, as we expected, the gender gap was minimal when our sample was in adolescence; however, the overall prevalence for males rose to a much greater degree from adolescence to young adulthood than it did in females. Previous studies reporting similar changes across these developmental periods have attributed these differentially increasing rates of SUDs to the fact that alcohol and illicit substance use problems are frequently comorbid with other psychopathology such as antisocial personality disorder, which is much more common among males (Hicks et al., 2007).

### 4.3. Evidence of both generalized and specific liabilities to addiction

Evidence from both methods used to assess the existence of a generalized risk to use and abuse multiple substances supported the alternate forms and correlated liability models identified by Rhee and colleagues (2003, 2006). In many instances, the observed prevalence rates of multiple substance use and disorders were greater than the expected rates (derived under the assumption of independent liabilities). The comparison between the observed and expected rates revealed that (1) the factors contributing to these behaviors are not independent, and (2) the risk for use of multiple substances and the development of multiple SUDs increases with age. Similarly, the prediction models supported a model of generalized risk since SUDs on any substance in young adulthood could be predicted by involvement with any of the three substances in adolescence. Adolescent smoking not only increased an individual's odds of developing tobacco/nicotine dependence, but also increased the odds of developing abuse or dependence on alcohol and marijuana in young adulthood. This finding was consistent with that of Bonomo and colleagues (2004) who also found an increased risk for alcohol dependence among tobacco users in adolescence using a community sample. Like previous studies (Brook et al., 1999; DeWit et al., 2000; Duncan et al., 1997; Gil et al., 2004; Grant and Dawson, 1997; Grant et al., 2006), adolescent use of alcohol and marijuana predicted both substance-specific and generalized substance problems in late adolescence/young adulthood. Likewise, the association between any tobacco use and marijuana use problems was similar to the findings of Lewinsohn et al. (1999). However, we did not replicate the association between marijuana involvement and tobacco dependence observed in Timberlake et al. (2007) possibly because of differences in the criteria for repeated-use between their study and our own. The evidence also suggests that the risk of alcohol, tobacco, and marijuana problems in young adulthood was greater if persons were diagnosed with a SUD on another substance in adolescence. Most importantly, the comparison of the adjusted versus the non-adjusted odds-ratio indicated that the associations are substantially influenced by involvement with other substances, providing evidence of a common vulnerability.

In addition to the evidence of a generalized risk across substances from Table 4 and Table 5, there was substantial evidence of substance-specific risk. Furthermore, the degree of specific risk was greatest for tobacco, followed by marijuana, and lastly alcohol. Although this was not entirely consistent with the literature on relative risk for addiction (Goldman et al., 2005;Goldstein and Kalant, 1990), tobacco was still ranked as the substance with the highest risk of SUD onset.

Overall, our findings converge on a central position regarding the nature of the liability for the development of SUDs in young adulthood in cases where substance use begins in adolescence. That is, early users do not appear to limit their use to a single substance, and this broad sampling of substances generally increases the risk for developing problems of abuse and dependence across a number of substances. Moreover, a SUD diagnosis on any given substance in young adulthood is not entirely predicted from involvement with the same substance during adolescence but also with other substances. These observations suggest that there is either a generalized risk factor or correlated risk factors for early onset substance use and subsequent development of substance use disorders. These patterns seen in our longitudinal analyses are consistent with findings from previous epidemiological and biometrical studies that have inferred a generalized liability to SUDs (Goldman and Bergen, 1998; Kendler et al., 2007, 2008; McGue and Iacono, 2008; Petraitis et al., 1995; Rhee et al., 2003, 2006; True et al., 1999; Vanyukov et al., 2003; Young et al., 2006). Other studies examining risk factors underlying substance involvement should not overlook the importance of data regarding the use of multiple drugs.

### 4.4. Proposed mechanisms of generalized risk: genetic vulnerability

Twin studies have provided the leverage needed to disentangle the relative importance of genetic and environmental influences on the generalized liability for substance use problems. Findings from these studies suggest that genetic factors explain a large degree of the overlapping risks among alcohol, tobacco, and marijuana use problems in youth (Hopfer et al., 2003; Kendler et al., 2007, 2008; Young et al., 2006). The search for specific genes that may explain this inherited liability for substance use problems is challenging and ongoing. Meta-analyses across published linkage and association studies point to polymorphisms in genes involved in the metabolism of drugs (e.g., polymorphisms in the alcohol dehydrogenase gene; Dick and Beirut, 2006; Li et al., 2008) and polymorphisms involved in neurological systems (e.g., brain-reward; Dick and Beirut, 2006; Gardner, 2002; Koob and Le Moal, 2001) that are widely known to be involved in the addiction process. Despite this progress, efforts to identify specific genetic polymorphisms that account for the generalized biological risk underlying substance use and SUDs continue.

### 4.5. Proposed mechanism of generalized risk: neurologic and environmental factors

Several areas of research propose alternate mechanisms underlying the liability for substance problems. One possible mechanism of risk specificity is that early involvement with alcohol in adolescence possibly increases the risk of later dependence by altering a person's normal developmental trajectory (DeWit et al., 2000). Intuitively, the same claim could be made for all drugs of abuse, given their impact on the still developing adolescent brain. A possible mechanism for the generalized risk is the interaction of the neural pathways associated with addictive drugs and the mesolimbic dopaminergic system which is the target pathway for the reinforcing effects of drugs (Dick and Beirut, 2006; Gardner, 2002; Koob and Le Moal, 2001). From an environmental perspective, it is also possible that drug use and abuse in adolescence increases the possibility of continued exposure to high-risk environments which may limit the attainment of adult roles, impair physical and mental health, and foster the development of antisocial and criminal behaviors which are alternative pathways to addiction (Jessor, 1998; White et al., 1998).

### 4.6. Study limitations

When employing survey data on adolescents the most common methodological problem is the underestimation of prevalence rates because the most severe cases are not usually recruited. Fortunately, the sample used in the current analyses was recruited at a young age and did not exclude respondents with behavioral problems or psychiatric disorders. Second, because additional data sources (such as parents and teachers) were unavailable we had to assume that respondent's reports were truthful. Interestingly, only 2.4–4% of the sample violated our model of progression (i.e., reported a lower level of lifetime involvement at Wave 2 than at Wave 1), suggesting a high level of accuracy in reporting. Third, other covariates of substance abuse (e.g., conduct disorder, attention deficit hyperactivity disorder, depression) (Button et al., 2007; Crowley et al., 1998; Ferdinand et al., 2001; Riggs et al., 1999; Tarter, 2002; Whitmore et al., 1997) were not included in the logistic regression models. While we recognize that omitting these predictors is a limitation of this study, the focus of this paper was determining whether the risks for substance problems in young adulthood were common or specific across these substances. Our approach is mirrored in an expanding literature of biometrical studies (e.g., Goldman and Bergen, 1998; Kendler et al., 2007, 2008; Rhee et al., 2006; True et al., 1999; Young et al., 2006) that provide evidence of a common genetic and/or environmental factor contributing to the comorbidity among these substances in both adolescence and adulthood. Fourth, dependence measures do not impose the clustering criterion of the DSM-IV or distinguish between dependence with or without physiological symptoms. Unlike previous adolescent epidemiological studies, we used a threshold of three symptoms of

dependence, to be consistent with the threshold for an adult diagnosis and to allow for comparison to adult studies. We chose not to impose clustering because the prevalence of disorders was only slightly changed across substances when clustering was required. Lastly, several of the confidence intervals in Table 5 are substantial. We believe this is a result of the low prevalence rates of SUDs in Wave 1 resulting in a small cell sizes in the odds-ratio analyses. Despite these instances, many of the odds-ratios are significantly greater than one, with small confidence intervals; we can be confident in the significance of these results, but less certain about the upper limit of the true odds-ratio.

### 4.7. Conclusions

As substance experimentation and use among adolescents continues, the evidence suggests that multiple drug involvement will increase, as will the impact on the development of substance disorders in young adulthood. At present, data from epidemiological surveys highlight the tendency of adolescents to ignore the negative risks associated with underage drug use (Johnston et al., 2008). In the MTF's most recent report on risk perception, only 46%, 60%, and 57% of $12^{th}$ graders were aware of the negative consequences of using alcohol, tobacco, and marijuana, respectively (Johnston et al., 2008). Given the generalized vulnerability to substance abuse/dependence a robust strategy for prevention and intervention enterprises would be a generalized substance risk approach targeted at young adolescents.

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**Table 1**

Sample characteristics.

| **Racial distribution** | | | **Age distribution by gender (*N*)** | | | | | |
|---|---|---|---|---|---|---|---|---|
| **Racial category** | ***N*** | **%** | **Wave 1** | | | **Wave 2** | | |
| | | | **Age** | **Females** | **Males** | **Age** | **Females** | **Males** |
| Caucasian | 1538 | 88.75 | 12 | 189 | 157 | 17 | 246 | 234 |
| Mixed ethnicity | 117 | 6.75 | 13 | 154 | 157 | 18 | 76 | 61 |
| Unknown | 32 | 1.85 | 14 | 107 | 119 | 19 | 103 | 128 |
| Asian | 21 | 1.21 | 15 | 115 | 99 | 20 | 102 | 91 |
| African American | 14 | 0.81 | 16 | 103 | 86 | 21 | 128 | 81 |
| Native American | 8 | 0.46 | 17 | 113 | 94 | 22 | 101 | 108 |
| Pacific Islander | 3 | 0.17 | 18 | 133 | 107 | 23+ | 158 | 116 |
| Totals | 1733 | 100 | | 914 | 819 | | 914 | 819 |

**Table 2**

Prevalence (%) of substance experimentation, repeated-use and SUD at each wave for each gender.

| | Wave 1 (ages 12–18) | | | | Wave 2 (ages 17–23+) | | | |
|---|---|---|---|---|---|---|---|---|
| | *N*[a] | Overall | Female | Male | *N*[a] | Overall | Female | Male |
| Experimentation (%) | | | | | | | | |
| Alcohol | 1720 | 53.14 | 50.99 | 55.53 | 1675 | 83.64 | 82.81 | 84.59 |
| Tobacco | 1721 | 26.73 | 27.26 | 26.13 | 1673 | 48.06 | 44.43 | 52.17* |
| Marijuana | 1722 | 17.48 | 18.47 | 16.38 | 1674 | 48.57 | 43.95 | 53.74** |
| Repeated-use (%) | | | | | | | | |
| Alcohol | 1720 | 22.27 | 23.4 | 21.01 | 1675 | 65.73 | **63.37** | **68.41** |
| Tobacco | 1721 | 7.61 | 8.61 | 6.50 | 1673 | 20.50 | **18.67** | **22.58** |
| Marijuana | 1722 | 10.22 | 11.06 | 9.29 | 1674 | 33.45 | 28.70 | 38.78** |
| Abuse (%) | | | | | | | | |
| Alcohol | 1733 | 4.62 | 4.27 | 5.01 | 1733 | 10.04 | 7.88 | 12.45* |
| Marijuana | 1733 | 1.96 | 2.19 | 1.71 | 1733 | 8.25 | 5.14 | 11.72** |
| Dependence (%) | | | | | | | | |
| Alcohol | 1733 | 1.38 | 1.64 | 1.10 | 1733 | 12.35 | 8.86 | 16.24** |
| Tobacco | 1733 | 3.46 | 3.72 | 3.17 | 1733 | 16.27 | 14.11 | 18.68 |
| Marijuana | 1733 | 1.85 | 1.97 | 1.71 | 1733 | 4.27 | 3.50 | 5.13 |
| SUD[b] (%) | | | | | | | | |
| Alcohol | 1733 | 6.00 | 5.81 | 6.11 | 1733 | 22.39 | 16.74 | 28.69** |
| Cannabis | 1733 | 3.81 | 4.16 | 3.42 | 1733 | 12.52 | 8.64 | 16.85** |

[a] Several subjects did not provide answers to experimentation and repeated-use in the MTF-based questionnaire.

[b] SUD category combines abuse and dependence diagnoses. Tobacco is not included as it is has only a dependence diagnosis. Bolded values indicate unweighted gender difference analyses with a *p*-value less than 0.05.

* Represents weighted gender difference analyses with a *p*-value less than 0.05.

** Represents weighted gender difference analyses with a *p*-value less than 0.01.

**Table 3**

Prevalence rates of alcohol, tobacco, and marijuana experimentation, repeated-use, and SUDs by age and gender.

| Level of Drug Involvement | Age | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | **12** | **13** | **14** | **15** | **16** | **17** | **18** | **19** | **20** | **21** | **22** | **23+** |
| Alcohol experimentation | | | | | | | | | | | | |
| Female (Wave 1) | 25.40 | 23.68 | 42.86 | 42.98** | 73.53 | 89.38 | 82.44 | | | | | |
| Male (Wave 1) | 24.36 | 30.77 | 54.62 | 69.39 | 78.82 | 86.17 | 80.19 | | | | | |
| Female (Wave 2) | | | | | | 67.76 | 65.79 | 86.00 | 88.66 | 93.50 | 98.99 | 90.67 |
| Male (Wave 2) | | | | | | 65.67 | 80.33 | 91.94 | 92.94 | 94.52 | 97.09 | 94.34 |
| Alcohol repeated-use | | | | | | | | | | | | |
| Female (Wave 1) | 2.65 | 4.61 | 11.43 | **9.65** | 35.29 | 56.64 | 58.78 | | | | | |
| Male (Wave 1) | 2.56 | 3.21 | 13.45 | **22.45** | 34.12 | 43.62 | 50.94 | | | | | |
| Female (Wave 2) | | | | | | 33.47 | 43.42 | 67.00 | 65.98 | 78.86 | 96.97 | 83.33 |
| Male (Wave 2) | | | | | | 39.48 | 55.74 | 68.55 | 78.82 | 87.67 | 95.15 | 91.51 |
| Alcohol SUD[a] | | | | | | | | | | | | |
| Female (Wave 1)[b] | 0.00 | 0.00 | 0.93 | 0.87 | 13.59 | 18.58 | **12.78** | | | | | |
| Male (Wave 1)[b] | 0.00 | 0.00 | 1.68 | 4.04 | 9.30 | 11.70 | **23.36** | | | | | |
| Female (Wave 2) | | | | | | 6.10 | **6.58** | 17.48 | 11.76** | **26.56** | 27.72* | 25.95* |
| Male (Wave 2) | | | | | | 8.12 | **21.31** | 23.44 | 41.76 | **40.74** | 47.22 | 43.97 |
| Tobacco experimentation | | | | | | | | | | | | |
| Female (Wave 1) | 4.26 | 8.61 | **11.43** | 26.32 | 39.22 | 61.95 | 55.64 | | | | | |
| Male (Wave 1) | 3.87 | 5.73 | **24.37** | 35.71 | 34.12 | 57.45 | 47.66 | | | | | |
| Female (Wave 2) | | | | | | 23.17 | 27.63* | 49.00 | **44.33** | 57.85 | 59.60 | 64.00 |
| Male (Wave 2) | | | | | | 29.18 | 52.46 | 51.61 | **60.00** | 71.23 | 70.59 | 66.04 |
| Tobacco repeated-use | | | | | | | | | | | | |
| Female (Wave 1)[b] | 0.00 | 0.66 | 0.95 | 7.02 | 9.80 | 24.78 | 22.56 | | | | | |
| Male (Wave 1)[b] | 0.00 | 1.27 | 4.20 | 8.16 | 5.88 | 15.96 | 16.82 | | | | | |
| Female (Wave 2) | | | | | | 8.13 | 6.58* | 21.00 | 17.53 | 26.45 | 26.26 | 30.00 |
| Male (Wave 2) | | | | | | 9.44 | 27.87 | 22.58 | 25.88 | 28.77 | 32.35 | 32.08 |
| Tobacco dependence | | | | | | | | | | | | |

| Level of Drug Involvement | Age | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | **12** | **13** | **14** | **15** | **16** | **17** | **18** | **19** | **20** | **21** | **22** | **23+** |
| Female (Wave 1)[b] | 0.00 | 0.00 | 0.93 | 2.61 | 2.91 | 12.39 | 9.77 | | | | | |
| Male (Wave 1)[b] | 0.00 | 0.64 | 0.00 | 2.02 | 2.33 | 9.57 | 11.21 | | | | | |
| Female (Wave 2) | | | | | | 4.88 | **3.95**[*] | 17.48 | 14.71 | 21.09 | 20.79 | 20.89 |
| Male (Wave 2) | | | | | | 5.98 | **22.95** | 17.97 | 24.18 | 29.63 | 29.63 | 20.69 |
| Marijuana experimentation | | | | | | | | | | | | |
| Female (Wave 1)[b] | 0.00 | 2.68 | **2.86** [*] | 15.65 | 29.41 | 46.02 | 45.11 | | | | | |
| Male (Wave 1)[b] | 1.27 | 1.91 | **15.13** | 13.13 | 18.82 | 44.68 | 37.38 | | | | | |
| Female (Wave 2) | | | | | | **24.49** | **32.89** | 46.00 | 51.02 | 55.37 | 66.33 | 51.70 |
| Male (Wave 2) | | | | | | **34.62** | **55.74** | 54.40 | 62.79 | 63.51 | 71.84 | 62.26 |
| Marijuana repeated-use | | | | | | | | | | | | |
| Female (Wave 1)[b] | 0.00 | 0.67 | 2.86 | 6.09 | 14.71 | 28.32 | 31.58 | | | | | |
| Male (Wave 1)[b] | 0.00 | 0.64 | 5.88 | 5.05 | 9.41 | 30.85 | 24.30 | | | | | |
| Female (Wave 2) | | | | | | 13.47[*] | 21.05 | 28.00 | **28.57** | 43.80 | **43.88** | 36.05 |
| Male (Wave 2) | | | | | | 23.50 | 31.15 | 37.60 | **47.67** | 51.35 | **58.25** | 43.40 |
| Marijuana SUD[a] | | | | | | | | | | | | |
| Female (Wave 1)[b] | 0.00 | 0.65 | 1.87 | 2.61 | 3.88 | 11.50 | 11.28 | | | | | |
| Male (Wave 1)[b] | 0.00 | 0.64 | 0.00 | 1.01 | 4.65 | 8.51 | 13.08 | | | | | |
| Female (Wave 2) | | | | | | **3.66** | 6.58 | 8.74 | **5.88**[**] | 18.75 | **7.92** | **11.39** |
| Male (Wave 2) | | | | | | **8.55** | 16.39 | 16.41 | **24.18** | 24.69 | **19.44** | **20.69** |

[a] SUD category combines abuse and dependence diagnoses. Tobacco is not included as it is has only a dependence diagnosis.

[b] Fisher's exact test or Chi-square difference test could not be computed in cases where the prevalence rates were zero. Bolded values indicate unweighted gender difference analyses with a *p*-value less than 0.05.

[*] Represents weighted gender difference analyses with a *p*-value less than 0.05.

[**] Represents weighted gender difference analyses with a *p*-value less than 0.01.

**Table 4**

Prevalence (%) of single/multiple substance repeated-use and SUDs at each wave.

| Mono/multiple substance category | Wave 1 (%) | | Wave 2 (%) | |
|---|---|---|---|---|
| | Observed | Expected[a] | Observed | Expected[a] |
| Repeated-use | | | | |
| No repeated-use | **75.29** | **64.48** | **32.77** | **18.13** |
| Alcohol only | 12.06 | 18.47 | 29.52 | 34.78 |
| Tobacco only | 1.23 | 5.31 | 0.36 | 4.68 |
| Marijuana only | 0.70 | 7.34 | **0.96** | **0.09** |
| Alcohol and tobacco | 1.17 | 1.52 | 4.03 | 8.97 |
| Alcohol and marijuana | **4.33** | **2.10** | 16.54 | 17.48 |
| Tobacco and marijuana | 0.35 | 0.60 | 0.36 | 2.35 |
| Alcohol, tobacco and marijuana | **4.86** | **0.17** | **15.45** | **4.51** |
| SUD | | | | |
| No SUD | **91.17** | **87.29** | **68.67** | **56.85** |
| Alcohol only | 3.35 | 5.57 | 9.64 | 16.40 |
| Tobacco[b] only | 0.87 | 3.13 | 4.56 | 11.05 |
| Marijuana only | 0.87 | 3.46 | 2.83 | 8.14 |
| Alcohol and tobacco[b] | **0.81** | **0.20** | **4.62** | **3.19** |
| Alcohol and marijuana | **0.87** | **0.22** | 1.73 | 2.35 |
| Tobacco[b] and marijuana | **1.15** | **0.12** | **2.60** | **1.58** |
| Alcohol, tobacco[b], and marijuana | **0.92** | **0.01** | **5.37** | **0.46** |

Bold numbers represent instances where the observed prevalence exceeds the expected prevalence.

[a] Expected rates of repeated-use and disorders were computed on the assumption of independence among substances using the overall prevalence rates in Table 2. For example the prevalence of repeatedly using alcohol only is: (Prevalence of repeatedly using alcohol/100)×(Prevalence of NOT repeatedly using tobacco/100)×(Prevalence of NOT repeatedly using marijuana/100)×100.

[b] Tobacco dependence.

**Table 5**

Odds of being diagnosed with alcohol, tobacco, or marijuana abuse/dependence in young adulthood (Wave 2) based on Wave 1 drug use status.

| Wave 1 status | Wave 2 | | | | | |
|---|---|---|---|---|---|---|
| | Alcohol abuse/dependence (95% CI) | | Tobacco dependence (95% CI) | | Marijuana abuse/dependence (95% CI) | |
| | OR[a] | AOR[b] | OR[a] | AOR[b] | OR[a] | AOR[b] |
| Use 1+ times (w/no SUD[c]) vs. abstinence | | | | | | |
| Alcohol | 2.72** (1.77,4.19) | 1.93** (1.21,3.09) | 2.97** (1.78,4.96) | 1.28 (0.70,2.31) | 3.34** (1.77,6.28) | 1.77 (0.87,3.59) |
| Tobacco[d] | 3.19** (2.10,4.85) | 1.94** (1.20,3.15) | 7.73** (4.81,12.43) | 5.89** (3.46,10.05) | 4.83** (2.92,8.00) | 2.34** (1.30,4.22) |
| Marijuana | 3.44** (1.93,6.12) | 1.52 (0.79,2.93) | 4.12** (2.26,7.51) | 1.19 (0.59,2.42) | 6.03** (3.18,11.43) | 2.91** (1.41,6.00) |
| SUD[c] vs. abstinence | | | | | | |
| Alcohol | – | – | 11.43** (5.23,24.95) | 3.17* (1.28,7.84) | 12.56** (5.39,29.29) | 3.86* (1.47,10.09) |
| Tobacco[d] | 8.31** (3.17,21.84) | 3.05* (1.01,9.26) | – | – | 16.27** (6.03,43.89) | 4.51* (1.37,14.86) |
| Marijuana | 8.78** (3.15,24.53) | 2.93 (0.88,9.78) | 8.61** (3.15,23.56) | 1.12 (0.33,3.82) | – | – |
| SUD[c] vs. Use 1+ times (w/no SUD[c]) | | | | | | |
| Alcohol | – | – | 5.43** (2.18,13.51) | 1.55 (0.54,4.41) | 5.32** (1.97,14.31) | 1.80 (0.59,5.51) |
| Tobacco[d] | 2.38 (0.86,6.61) | 1.25 (0.40,3.94) | – | – | 2.46 (0.83,7.26) | 0.77 (0.22,2.72) |
| Marijuana | 1.77 (0.54,5.78) | 1.32 (0.36,4.76) | 2.61 (0.78,8.72) | 1.34 (0.34,5.24) | – | – |

OR and AOR models were based on the following log-linear equations: OR model: Log(Odds of Alcohol SUD at Wave 2 is TRUE) =$\beta_0$ + $\beta_1$(Wave 1 Alcohol Status Comparison) + $\beta_2$(Age) + $\beta_3$(gender) + $\beta_4$(Wave 1 Status Comparison)×Age + $\beta_5$(Wave 1 Status Comparison)×Gender + $\varepsilon_i$.

AOR model: Log(Odds of Alcohol SUD at Wave 2 is TRUE) = $\beta_0$ + $\beta_1$(Wave 1 Alcohol Status Comparison) + $\beta_2$(Age) + $\beta_3$(gender) + $\beta_4$(Wave 1 Status Comparison)×Age + $\beta_5$(Wave 1 Status Comparison)×Gender + $\beta_6$(Wave 1 Level of Tobacco Involvement) + $\beta_7$(Wave 1 Level of Marijuana Involvement) + $\varepsilon_i$.

[a] Estimates generated controlling for age (mean deviated), sex and interactions.

[b] Estimates generated controlling for age (mean deviated), sex, interactions, and other substance patterns at wave 1.

[c] SUD category combines abuse and dependence diagnoses. Tobacco is not included as it has only a dependence diagnosis.

[d] Tobacco dependence.

* Represents weighted analyses with a *p*-value less than 0.05.

** Represents weighted analyses with a *p*-value less than 0.01.