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Directions of the relationship between substance use and depressive symptoms from adolescence to young adulthood

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Abstract

Purpose—Both substance use and depression are common in adolescence and often comorbid. Past research has produced conflicting results on whether there is a temporal relationship and if so, in which direction it operates and how it may vary by sex. The purpose of this paper is to explore the longitudinal, potentially bidirectional, relationships between high-frequency substance use and depressive symptoms from adolescence into young adulthood for males and females.

Methods—Using data from the National Longitudinal Study of Adolescent to Adult Health we investigated longitudinal associations between high frequency substance use (alcohol, cigarettes, and marijuana) and depressive symptoms. The linear mixed effects models were stratified by sex and used a lagged measure of the dependent variable to test temporal relationships. A random intercept was used for respondent ID.

Results—Increases in depressive symptoms were significantly associated with a later increase of about a half day in marijuana use frequency for males and nearly a two day increase in smoking frequency for females. Conversely, increases in smoking frequency were significantly associated with approximately a 0.6-point increase for females and 0.4-point increase for males in depressive symptoms at a later wave.

Conclusions—Results indicate a bidirectional relationship between smoking and depressive symptoms for females. For males, there was evidence supporting self-medication with marijuana and for smoking being associated with later increases in depressive symptoms. Results inform how substance use and depression screening, prevention and treatment efforts should be paired and targeted for males and females.

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Keywords

Substance use; depression; adolescence; young adulthood; gender

1. Introduction

Substance use and depression are common in adolescence, frequently co-morbid, and have serious short- and long-term health implications (Chassin, Hussong, & Beltran, 2009; Fletcher, 2010; Kann, Kinchen, Shanklin, & et al., 2014). Despite substantial research, the directionality between the two, and whether directionality varies by sex, remains unclear. The self-medication hypothesis asserts that risk taking is used to ameliorate depressive symptoms possibly through lowering impulse control or motivation (Chassin et al., 2009; Khantzian, 1997). Several studies support this pathway. For example, one study followed over 4,000 adolescents from grades 9 to 12 and found those reporting higher depressive symptoms in grade 9 reported faster increases in cigarette and marijuana use (Hooshmand, Willoughby, & Good, 2012). Burns et al. followed a small group of rural adolescents for two years and found baseline depression scores were associated with later tobacco use (Burns et al., 2004). Below we illustrate the mixed findings from research in this area, focusing on papers using advanced longitudinal methods.

Sex complicates the self-medication hypothesis as adolescent females generally report more depression and less risk taking; the aforementioned studies did not examine sex differences (Chassin et al., 2009; Kann et al., 2014). Studies examining sex differences show conflicting results. For example, symptoms of depression were positively associated with alcohol, tobacco, and drug use in a clinical sample of 400 youths but relationships were not moderated by sex (Schwinn, Schinke, & Trent, 2012). In contrast, in a sample of ninth and 10^{th} grade students, overall negative mood predicted rapid smoking escalation in boys, but only those with affect-related motives for smoking; for girls, negative mood variability predicted smoking escalations indicating moderation by sex (Weinstein & Mermelstein, 2013). Another study that followed students through high school found an increase in depressive symptoms predicted trying smoking for boys but not for girls (Killen et al., 1997). Similarly, with a sample of over 600 African American adolescents who were interviewed annually for six years starting in high school, depressive symptoms predicted marijuana use in males but not in females (Repetto, Zimmerman, & Caldwell, 2008). However, a small study of 200 undergraduate women found support for self-medication with alcohol (Mushquash et al., 2013).

Some sex differences in self-medication could be due to differences in the substances. A study of cannabis-dependent respondents found cannabis increased depressive symptoms among those with a history of depression though it improved control of aggression, which is more common among males (Arendt et al., 2007; B. Green & Ritter, 2000; Zahn-Waxler, Shirtcliff, & Marceau, 2008). This may explain findings supporting male' self-medication with marijuana (Henry et al., 1993; Repetto et al., 2008; Schuster, Mermelstein, & Wakschlag, 2013). The Nicotine Dependence in Teens study revealed cigarette smoking does not seem to reduce depressive symptoms but can slow rate of change over time

(Chaiton, Cohen, O'Loughlin, & Rehm, 2010). As depressive symptoms can be less gender-normative for males, they may have stronger motivations than females to self-medicate and slow symptom progression (Evans, Frank, Oliffe, & Gregory, 2011; Tamres, Janicki, & Helgeson, 2002). Finally, in a sample of 400 eighth grade students, Tomlinson and Brown (2012) found depressive symptoms did predict heavier, more frequent, and solitary drinking but the relationship may be moderated by social anxiety and alcohol expectancies, which may vary by sex (Tomlinson & Brown, 2012).

Alternatively, there is support for the reverse pathway that substance use leads to depression, though results are again mixed. Using data from Waves I and II of Add Health, one study found both sexual risk taking and substance use predicted an increased likelihood of future depression; the self-medication pathway was not supported in their analyses (Hallfors, Waller, Bauer, Ford, & Halpern, 2005). Similarly another study using Add Health data from Waves I and II, found no relationship between depression and later smoking but did find support for the reverse (Goodman & Capitman, 2000). Longitudinal analyses for over 1,000 New Zealand youth indicated the best-fitting causal model was from alcohol abuse or dependence to depression, though they tested both directions (Fergusson, Boden, & Horwood, 2009). A longitudinal study of African Americans from age 6 to 42 found increased substance use (alcohol, marijuana, cocaine) in adolescence predicted psychological distress in young adulthood but only for men (Green, Zebrak, Robertson, Fothergill, & Ensminger, 2012). These latter findings conflict with other theoretical and empirical findings indicating females are more vulnerable to increased depression resulting from substance use, perhaps due to their greater interpersonal sensitivity and vulnerability to interpersonal stress (Ge, Lorenz, Conger, & et. al, 1994; Hallfors et al., 2005; Rudolph, 2002).

Previous studies of the association between depression and substance use in adolescents are limited by cross-sectional design or, when longitudinal, by using non-representative samples, short time periods, or not examining both directions or sex differences (Brook, Brook, Zhang, Cohen, & Whiteman, 2002; Chinet et al., 2006). This paper prospectively examines directionality over a longer time period using the population-based Add Health sample and stratifying by sex. Based on prior research, we hypothesized greater support for the self-medication hypothesis for males and greater support for the reverse pathway for females.

2. Methods

2.1 Sample

Add Health is a school-based longitudinal study that includes a nationally representative U.S. sample of adolescents who were in grades 7-12 in the 1994-95 school year (Wave I). There have been four in-home interview waves since. The analysis sample is restricted to respondents subsequently interviewed at ages 18 to 26 (Waves III, 2001) and ages 24 to 32 (Wave IV, 2007-2009), with valid sampling weights (N=12,288) and who had complete data on all variables of interest (N=12,017, missing 2.2%). Data from Wave II were not used as Wave I seniors were not followed by design. Details of the Add Health study and design are described elsewhere (Harris, 2013). All Add Health processes were approved by the

Institutional Review Board at the University of North Carolina, Chapel Hill, these analyses were deemed exempt.

2.2 Measures

2.2.1 Depression—We used the nine items from the Center for Epidemiologic Studies Depression scale (CES-D) that appeared at each interview wave, as prior Add Health studies have done (alpha=0.8). The psychometrics of a seven-item CES-D have been previously validated (Levine, 2013). Questions ask about frequency of symptoms in the past week, though 12-month re-test reliability is high (Eaton, Muntaner, Smith, Tien, & Ybarra, 2004). Answers are scored from 0 to 3, indicating rarely to most of the time; the summed score ranges from 0 to 27. The CES-D captures depressive symptoms but is not a diagnostic tool (Eaton et al., 2004).

2.2.2 Substance use—Substances include alcohol (binge drinking), cigarettes, and marijuana. In Add Health, substance use is assessed with either continuous or ordinal variables, and the time frame varies. For cigarette smoking, respondents are asked, at all waves, how many days they smoked in the past thirty days. At Waves I and III, the question is very similar for marijuana use but captures instances of use in the past 30 days (e.g., 0 to >900). At Wave IV, the question changes to measure how many days respondents used marijuana in the past thirty using a 0 to 6 ordinal scale for none to nearly every day or every day. Finally, binge drinking was assessed for the past year using the same ordinal variable (Harris, 2013). To make the measures of marijuana use frequency comparable across the waves, days of marijuana use was derived from the measures at Waves I and III and then these frequencies were made ordinal to align with the measure of marijuana use at Wave IV. We modeled each substance individually controlling for use of the others.

2.2.3 Controls—Substance use and depressive symptoms can vary along sociodemographic lines so covariates were included for respondent's self-identified race/ethnicity (Hispanic and non-Hispanic White, Black, Asian, Native American, and Other) and educational attainment of both the parents and the respondent (less than high school, high school graduate, some college, or college graduate or higher) as a proxy for socioeconomic status (Chassin et al., 2009). Respondent's age was also included as substance use can vary substantially by age and the age ranges are fairly wide within waves.

2.3 Analysis

Linear mixed effects models with lagged measures of the dependent variable were used to evaluate both the Self-Medication and reverse pathways. Mixed effects models control for time invariant unobserved characteristics, reducing vulnerability to endogeneity, and are fairly robust to violations of normality (Fitzmaurice, Laird, & Ware, 2011). Also, the inclusion of a prior measure of the dependent variable in the models will effectively test the relationship between an earlier measure of the predictor variable and a later change in the dependent variable. In the models, the CES-D score was scaled by 5 so the regression results display a substantively meaningful change. For the figures displaying predicted changes in substance use frequency or depressive symptoms for each sex, race was set to White, parental education and respondent education were held at the referent value (college

graduation or higher), and all other covariates were held at their means. We used the original, rather than the scaled, CES-D to ease interpretability in the figures. All analyses were stratified by sex and used longitudinal weights to adjust for unequal probability of selection into the sample and nonresponse over time. Additionally, we adjusted variance estimates for clustering at the primary sampling unit and stratification by region. We used Stata, version 13.0 (Stata Corp, College Station TX, 2013). We test our assumptions of linearity in the ordinal substance use measures by treating the variables as ordinal and continuous in two different models and comparing the BIC between the two models. We also test our assumption that the strengths of the association from Wave I to Wave III will be equivalent to the association between Wave III and Wave IV with an interaction term between a Wave IV indicator variable and the key predictors.

3. Results

Table 1 outlines the analysis sample's demographic characteristics. Across all waves, the mean CES-D score was approximately one point higher for females than males ($p<0.001$). Males consistently reported more substance use than females, except for cigarette smoking at Wave I.

Table 2 shows the results of the three linear mixed effects models with frequency of each substance rotating as the dependent variable to test the self-medication pathway, CES-D was the independent variable. The intercept of this model was allowed to vary randomly by respondent ID. The results indicate that for males, a 5-point increase in depressive symptoms was associated with a 0.08 ($p<0.01$) later increase in marijuana use frequency. For females, a 5-point increase in depressive symptoms was associated with a 0.31 ($p<0.05$) later increase in cigarette smoking frequency. Also, the standard deviation of the intercept indicates meaningful variation in the intercept by respondent ID.

Using the results from Table 2, the predicted mean marijuana use frequency (for males) and smoking frequency (for females) as depressive symptoms increase are displayed in Figures 1 and 2 below, respectively. A maximum increase on the CES-D is predicted to increase male mean monthly marijuana use frequency from approximately 0.5 to 1, or one day of marijuana use in the past month. For females, a maximum increase in depressive symptoms is predicted to increase mean monthly smoking frequency by nearly 2 days.

Table 3 shows the results of the linear mixed effects models testing the relationship between substance use frequency and later depressive symptoms. The intercept of this model was allowed to vary randomly by respondent ID. The results indicate a 5-day increase in smoking frequency was associated with a 0.02 ($p<0.01$) increase for males and a 0.05 ($p<0.001$) increase for females in later depressive symptoms. The standard deviation of the intercept indicates some variation in the intercept by respondent ID. Further, the linearity assumption for the ordinal substance use measures was supported as the BIC estimates from models with the measures coded as continuous and then categorical were very similar. The assumption that the strengths of associations between the waves would be equivalent was partially supported as the interactions between the Wave IV indicator variable and the substance use predictors were not statistically significant. But, the interactions between the

Wave IV indicator variable and the depressive symptoms predictors in the marijuana self-medication model for males and the cigarette self-medication model for females were both statistically significant.

Figure 3 shows the predicted mean CES-D score as smoking frequency increases for males and females. For a 30-day increase in smoking frequency, there was a predicted nearly 0.4 point increase in the mean CES-D score for males and a 0.6 point increase for females.

4. Discussion

We assessed the relationship directionality between depressive symptoms and high frequency substance use from adolescence into young adulthood using a nationally representative sample. Overall, we found support for each pathway. Depressive symptoms were associated with increases in later smoking frequency for females and marijuana use frequency for males, consistent with the self-medication hypothesis though contrary to our hypothesis of a stronger relationship for males (Khantzian, 1997). Smoking frequency was also associated with later increases in depressive symptoms for both males and females, and the relationship was stronger for females, consistent with our hypothesis.

Our most robust finding is that an increase in depressive symptoms among females is significantly associated with a nearly two-day later increase in monthly smoking frequency. Evidence of depression self-medication with cigarette smoking has been found elsewhere, perhaps because cigarettes have been linked with increases in positive affect and decreases in negative affect, both of which are implicated in depression (Audrain-Mcgovern, Rodriguez, & Leventhal, 2015; Burns et al., 2004; Hedeker, Mermelstein, Berbaum, & Campbell, 2009; Hooshmand et al., 2012). However, our finding of self-medication with cigarettes only among females contrasts with prior longitudinal analyses. Two analyses of high school students, one sample followed for 15 months and the other for four years, found increases in depressive symptoms predicted smoking initiation and escalation only for males (Killen et al., 1997; Weinstein & Mermelstein, 2013). Another analysis, using Add Health data from Waves I and III, found females with high depressive symptoms were less vulnerable to subsequent increases in cigarette use (Needham, 2007). One potential explanation for our finding is our use of a longer developmental timespan than previous studies—from adolescence into young adulthood— allowed for a more complete picture. Also, there is empirical support for why these associations may be stronger for females. A longitudinal survey of adolescents found both low positive affect and high negative affect increased the risk of smoking for females and only high negative affect increased risk for males; this suggests females have a stronger impulse to self-medicate (Audrain-Mcgovern et al., 2015).

We also found support for males self-medicating depressive symptoms, though with marijuana instead of cigarettes. The size of the association was smaller; an increase in depressive symptoms was associated with approximately a half-day increase in monthly use frequency. This result has been found previously but is somewhat perplexing as other studies have found marijuana use may aggravate rather than soothe depressive symptoms (Arendt et al., 2007; Green & Ritter, 2000). One possible explanation is that males are not self-

medicating depression but aggression, for which marijuana appears effective (Arendt et al., 2007). Aggression may be comorbid with depression in males as they are more likely to be antagonistic and anti-social when depressed compared to females (Nolen-Hoeksema, 1990; Zahn-Waxler et al., 2008). Alternatively, depressive symptoms can predict delinquency in males and marijuana may be appealing for this as an illicit substance (Schuster et al., 2013). Finally, although a half-day increase in marijuana use frequency may not seem clinically relevant, it should be noted that the association reflects a time span of approximately seven years; past studies with shorter time spans have found larger effect sizes (Henry et al., 1993; Repetto et al., 2008; Schuster et al., 2013).

Despite the contention that men are more likely to cope by drinking alcohol, we found no support for males self-medicating with binge drinking (Nolen-Hoeksema, 2012). This may be a function of our measure of alcohol use. Binge drinking in adolescence and early adulthood is relatively common and generally a social activity. Past studies have found associations between solitary drinking and depression (Hooshmand et al., 2012; Needham, 2007; Tomlinson & Brown, 2012). Alternatively, if males do self-medicate depression with delinquent behavior, alcohol may be less appealing as the most prevalent form of substance use (Hooshmand et al., 2012; Schuster et al., 2013).

Regarding the reverse pathway, we do find that an increase in smoking frequency is significantly associated with modest later increases in depressive symptoms among both males and females. Our findings are stronger for females, in line with prior analyses and our hypotheses (Ge et al., 1994; Hallfors et al., 2005; Rudolph, 2002). Two previous similar studies found smoking associated with a 200% later increase in the odds of depression (Goodman & Capitman, 2000; Hallfors et al., 2005). There are three important differences between these studies and our own that may contribute to differences in the strength of the association. First, the earlier studies examined a one-year timespan, compared to our seven year span. Second, they treated depression as a binary rather than using frequency of symptoms; the latter relationship is likely more subtle. Third, one of these earlier studies used clusters of risk behaviors and found the strong association between cigarette smoking and later depression when smoking was combined with sexual risk behavior, suggesting combinations of risk behaviors are more potent predictors of depressive symptoms (Hallfors et al., 2005).

Taken together, our findings suggest a bidirectional relationship between depressive symptoms and cigarette smoking for females. This could be interpreted as evidence that self-medication of depressive symptoms with nicotine does not ameliorate the symptoms (Chaiton et al., 2010). Further, as females increase their cigarette smoking frequency, they are engaging in a non-normative activity that could increase interpersonal stress, thereby increasing depressive symptoms (Ge et al., 1994; Nelson et al., 2008; Rudolph, 2002). Past studies that tested for bidirectional associations using Add Health data (Goodman & Capitman, 2000; Hallfors et al., 2005) and a sample of Canadian adolescents (Hooshmand et al., 2012) did not find them. However, they used different measures of smoking and different time frames and one explained the bidirectional association away with potential confounders.

Our findings should be considered in the context of this study's limitations. Limitations include assumptions of linearity in the measures of use frequency for marijuana and binge drinking, and that the strengths of the associations between high frequency substance use and depressive symptoms are equivalent from Wave I to Wave III and from Wave III to Wave IV. However, we tested both assumptions and they were partially supported, though there appears to be variation by wave in the self-medication pathways for both males and females, something future research could examine. Third, the use of lagged measures of the dependent variable could introduce bias that would decrease the magnitude of the associations, though Monte Carlo simulations reveal their use is acceptable when we expect prior conditions will influence the current process. Further, if the sample is larger than 50, the bias is small and the chance of making incorrect inferences is less than 1% (Keele & Kelly, 2006). A fourth consideration is that the magnitude of identified associations, especially for smoking predicting later increases in depressive symptoms, is small. However, as seven years separate each of the interview waves and we are using continuous measures of depressive symptoms, this is not surprising. Fifth, though the prevalence of adolescent substance use has changed since 1994 when the first wave of data were collected, we have no reason to suspect the relationship between substance use and depression has changed. Finally, our results may be influenced by the bidirectional relationship between depression and substance use. To investigate this, we ran all models with only the youngest respondents at Wave I as they had a very low prevalence of substance use, and found similar results (available upon request).

Present findings support the self-medication hypothesis, but also indicate it is not the only explanation. We find support for males and females self-medicating depressive symptoms with marijuana and cigarettes, respectively, though we also find cigarette smoking significantly associated with later increases in depressive symptoms. Future research is needed to understand potential mediators of this pathway. In the meantime, study results can inform adolescent health care. The United States Preventive Services Task Force recently supported depression screening for adolescents and the Affordable Care Act supports substance use screening for adolescents (U.S. Department of Health and Human Services, 2012; Williams, O'Connor, Eder, & Whitlock, 2009). This research reveals screening for both is important and, where resources are limited, females screening positive for either smoking or depression should be targeted for both. For males, those screening positive for depression should also be targeted for marijuana use prevention and those screening positive for smoking should be screened for depression.

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Highlights

- Depressive symptoms associated with later increases in smoking for females
- Depressive symptoms associated with later increases in marijuana for males
- Smoking linked with later increases in depressive symptoms, especially in females

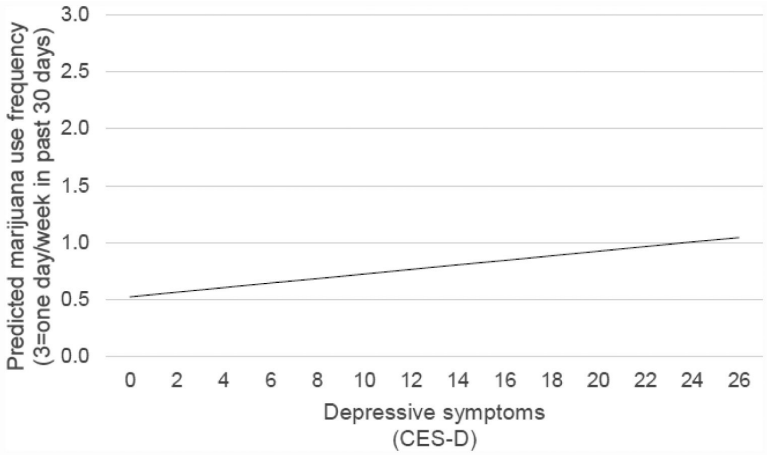

Figure 1. Predicted change in marijuana use frequency as depressive symptoms increase, Males[a]

[a] The figure shows results from the same model as outlined in Table 2, but the horizontal axis was changed to the unscaled measure of depressive symptoms to maximize interpretability.

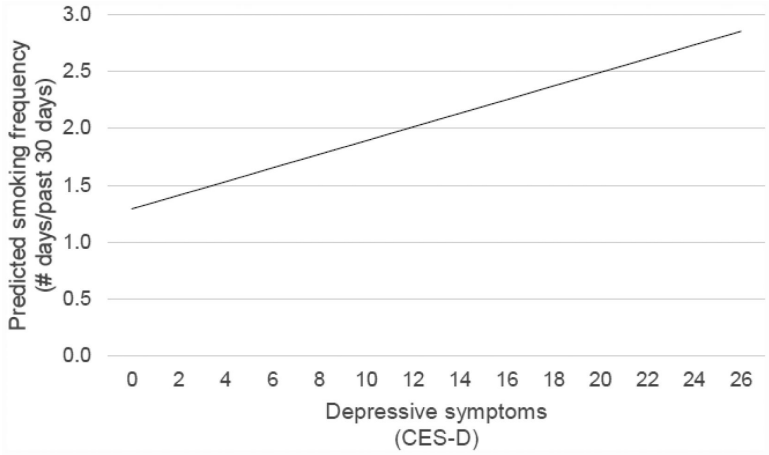

Figure 2. Predicted change in cigarette smoking frequency as depressive symptoms increase, Females[a]

[a] The figure shows results from the same model as outlined in Table 2, but the horizontal axis was changed to the unscaled measure of depressive symptoms to maximize interpretability

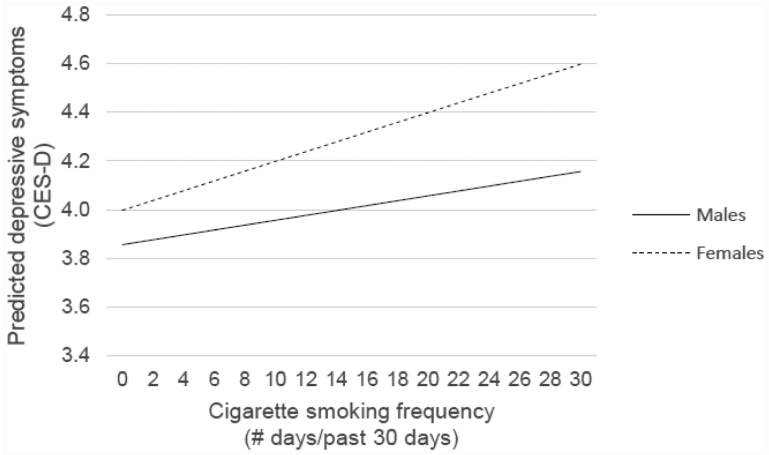

Figure 3. Predicted change in depressive symptoms as smoking frequency increases[a]

[a] The figure shows results from the same model as outlined in Table 3, but the vertical axis was changed to the unscaled measure of depressive symptoms to maximize interpretability.

Table 1

Characteristics of the analysis sample

Characteristic	Males (n=5474) n (weighted %)	Females (n=6521) n (weighted %)
Race/Ethnicity[a]		
Hispanic	886 (12.2)	967 (11.4)
Black	1014 (14.9)	1482 (16.0)
Asian	412 (3.8)	397 (3.4)
Native American	111 (2.3)	113 (1.8)
Other	50 (1.0)	54 (1.0)
White	2970 (65.8)	3507 (66.5)
Parental Education		
Less than high school	630 (11.9)	843 (11.5)
High school graduate	1318 (26.1)	1680 (28.2)
Some college	1603 (30.0)	1845 (28.7)
College graduate or higher	1892 (32.1)	2152 (31.6)
Respondent Education (Wave IV)		
Less than high school	458 (9.3)	377 (6.9)
High school graduate	1007 (20.8)	837 (13.4)
Some college	2361 (41.6)	2872 (44.2)
College graduate or higher	1616 (28.3)	2434 (35.6)
Age (mean (SE[b]))		
Wave I	15.5 (0.12)	15.3 (0.12)
Wave III	21.9 (0.12)	21.7 (0.12)
Wave IV	28.4 (0.12)	28.2 (0.12)
CES-D (mean (SE))[c]		
Wave I	5.1 (0.09)	6.3 (0.12)***
Wave III	4.0 (0.07)	4.9 (0.09)***
Wave IV	4.8 (0.09)	5.6 (0.09)***
Binge Drinking (mean (SE))		
Wave I	0.8 (0.05)	0.5 (0.03)***
Wave III	1.7 (0.06)	0.9 (0.04)***
Wave IV	1.5 (0.04)	0.9 (0.04)***
Cigarettes (mean (SE))		
Wave I	4.3 (0.31)	4.7 (0.37)
Wave III	9.4 (0.32)	8.3 (0.42)**
Wave IV	9.6 (0.34)	7.7 (0.40)***
Marijuana Use (mean (SE))		
Wave I	0.5 (0.04)	0.4 (0.03)**
Wave III	1.1 (0.05)	0.6 (0.04)***

Characteristic	Males (n=5474) n (weighted %)	Females (n=6521) n (weighted %)
Wave IV	0.9 (0.04)	0.4 (0.03)***

*p<0.05;

[a]All other race groups are non-Hispanic

[b]SE: Standard error

[c]The sex differences in the means of the key measures were tested and stars (*) were used to indicate if the mean for females was significantly different from the mean for males

**p<0.01;

***p<0.001

Table 2

Linear mixed effects models of the relationship between depressive symptoms at an earlier wave and substance use frequency at a later wave, Males and Females

	MALES (n=5474)			FEMALE (n=6521)		
Coefficients (SE)	**M1: Binge drinking**	**M2: Cigarettes**	**M3: Marijuana**	**M1: Binge drinking**	**M2: Cigarettes**	**M3: Marijuana**
CES-D	−0.01	0.24	0.08**	0.00	0.31*	0.03
	(0.03)	(0.22)	(0.03)	(0.02)	(0.15)	(0.02)
Binge drinking		1.24***	0.25***		1.69***	0.27***
		(0.14)	(0.02)		(0.18)	(0.02)
Cigarettes	0.03***		0.04***	0.02***		0.03***
	(0.00)		(0.00)	(0.00)		(0.00)
Marijuana	0.23***	1.78***		0.27***	2.11***	
	(0.02)	(0.13)		(0.02)	(0.14)	
Age	0.10***	0.61***	0.02***	0.03***	0.59***	0.00
	(0.01)	(0.05)	(0.01)	(0.00)	(0.03)	(0.00)
Race/ethnicity						
Hispanic	−0.11	−2.68***	0.22*	−0.04	−3.13***	0.10*
	(0.07)	(0.52)	(0.10)	(0.05)	(0.44)	(0.04)
Black	−0.49***	−3.61***	0.32***	−0.28***	−4.77***	0.14***
	(0.06)	(0.36)	(0.05)	(0.03)	(0.26)	(0.03)
Asian	−0.49***	−0.99	0.00	−0.21***	−2.15***	0.06
	(0.06)	(0.51)	(0.07)	(0.05)	(0.45)	(0.05)
Native American	−0.15	0.67	0.25	−0.03	−0.22	0.16
	(0.14)	(1.03)	(0.14)	(0.09)	(1.11)	(0.10)
Other	0.12	−2.53*	0.22	−0.45***	0.06	0.26
	(0.21)	(1.26)	(0.20)	(0.12)	(1.38)	(0.18)
White	(referent)	(referent)	(referent)	(referent)	(referent)	(referent)
Parental education						
< High school	−0.06	−0.80	−0.18	−0.04	0.58	−0.19***
	(0.09)	(0.70)	(0.11)	(0.05)	(0.49)	(0.05)
High school	0.08	0.66	−0.26***	−0.03	1.74***	−0.17***
	(0.07)	(0.55)	(0.07)	(0.04)	(0.40)	(0.04)
< College	0.01	0.06	−0.12	0.05	0.55	−0.07
	(0.05)	(0.45)	(0.06)	(0.04)	(0.34)	(0.04)
College or higher	(referent)	(referent)	(referent)	(referent)	(referent)	(referent)
Respondent education						
< High school	−0.32***	6.99***	0.26**	−0.17*	7.44***	0.15*
	(0.09)	(0.74)	(0.09)	(0.07)	(0.63)	(0.07)

	MALES (n=5474)			FEMALE (n=6521)		
Coefficients (SE)	**M1: Binge drinking**	**M2: Cigarettes**	**M3: Marijuana**	**M1: Binge drinking**	**M2: Cigarettes**	**M3: Marijuana**
High school	−0.38***	5.14***	0.13	−0.30***	4.37***	0.08
	(0.08)	(0.61)	(0.09)	(0.05)	(0.42)	(0.05)
< College	−0.33***	4.31***	0.12*	−0.26***	4.08***	0.02
	(0.07)	(0.42)	(0.06)	(0.04)	(0.34)	(0.04)
College or higher	(referent)	(referent)	(referent)	(referent)	(referent)	(referent)
Constant	−1.16***	−14.51***	−0.52***	−0.02	−13.78***	−0.01
	(0.16)	(1.13)	(0.15)	(0.09)	(0.91)	(0.09)
Variance estimates (SE)[a]						
Respondent ID	0.15	28.0	0.29	0.06	24.80	0.16
	(0.04)	(2.27)	(0.05)	(0.02)	(2.06)	(0.03)
Residual	1.95	79.83	1.95	1.14	68.55	1.05
	(0.07)	(2.78)	(0.08)	(0.05)	(2.54)	(0.05)

*p<0.05;

**p<0.01;

***p<0.001

[a]When the slope was allowed to randomly vary by age, there was not meaningful variation and so the final models only allow for random intercepts.

Table 3

Linear mixed effects models of the relationship between substance use frequency at an earlier wave and depressive symptoms at a later wave, Males and Females

Coefficients (SE)	MALES (N=5474)	FEMALES (N=6521)
Binge drinking	−0.01	0.01
	(0.01)	(0.01)
Cigarettes	0.003**	0.01***
	(0.00)	(0.00)
Marijuana	0.01	0.02
	(0.01)	(0.01)
Age	−0.02***	−0.02***
	(0.00)	(0.00)
Race/ethnicity		
Hispanic	0.13**	0.17***
	(0.04)	(0.04)
Black	0.15***	0.20***
	(0.03)	(0.03)
Asian	0.25***	0.30***
	(0.04)	(0.05)
Native American	0.09	0.07
	(0.07)	(0.08)
Other	0.00	−0.04
	(0.17)	(0.11)
White	(referent)	(referent)
Parental education		
< High school	0.20***	0.18***
	(0.05)	(0.04)
High school	0.07*	0.11***
	(0.03)	(0.03)
< College	0.02	0.09**
	(0.03)	(0.03)
College or higher	(referent)	(referent)
Respondent education		
< High school	0.16***	0.45***
	(0.05)	(0.05)
High school	0.08*	0.27***
	(0.03)	(0.04)
< College	−0.02	0.11***
	(0.03)	(0.02)
College or higher	(referent)	(referent)

Coefficients (SE)	MALES (N=5474)	FEMALES (N=6521)
Constant	1.26***	1.27***
	(0.07)	(0.08)
Variance estimates		
(SE)		
Respondent ID	0.16	0.20
	(0.01)	(0.01)
Residual	0.35	0.51
	(0.02)	(0.02)

[a] When the slope was allowed to randomly vary by age, there was not meaningful variation and so the final models only allow for random intercepts.

* p<0.05;

** p<0.01;

*** < 0.001