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Alcohol-Impaired Driving: The Influence of Adverse Rearing Environments, Alcohol, Cannabis Use, and the Moderating Role of Anxiety

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Abstract

Background—The rate of alcohol-impaired driving (AID) increases during the college years and students who have reported adverse rearing environments appear to be at increased risk for the development of alcohol and drug use behaviors. Alcohol and cannabis are the most commonly used drugs by college students, and these substances are particularly predictive of substance-impaired driving.

Objectives—The present study aimed to investigate whether adverse rearing environment experiences and level of alcohol and cannabis use are related to the frequency of alcohol-impaired driving and whether anxiety might buffer or accelerate this effect.

Methods—Data regarding adversity, drug use, anxiety, and AID were obtained from 1,265 students annually, from first to final year of college, over four waves (Mean Age at wave 1 = 18.5 years).

Results—Structural equation modeling supported associations among childhood adversity, alcohol, cannabis, and anxiety symptoms. A significant mediation effect was found such that adversity was predictive of AID via alcohol use and cannabis use. Among men, anxiety symptoms accelerated the path from increased cannabis use and decelerated the path from increased alcohol use to AID frequency.

Conclusions/Importance—Childhood adversity is a developmental risk precursor to drug use and AID, whereas anxiety might serve a risk or protective factor to AID, contingent on the drug used.

Keywords

Alcohol-impaired driving; substance use; anxiety; childhood adversity; risk taking; gender

Introduction

Alcohol-impaired driving (AID) has been a major public health issue and a cause of preventable injury and mortality in the United States (U.S.) during the past several decades. In 2013, 10,076 individuals lost their lives due to AID crashes, accounting for nearly one-third (31%) of all traffic-related deaths in the U.S. (National Highway Traffic Safety Administration, 2014). The rate of alcohol-involved fatal crashes is highest among young adults (21–24), with men significantly more likely than women (24% vs. 14%, respectively) to die in fatal crashes as a result of AID (National Highway Traffic Safety Administration, 2013). Although available information from national surveys on AID suggests a decreasing trend in the prevalence of AID among young adults (Berning et al., 2015), college populations remain at a particular risk (Hingson et al., 2009).

Early adversity and substance use

Adverse rearing environments, such as child abuse and inter-parental violence, are associated with the development of alcohol and drug use behaviors in young adulthood (Rogosch, Oshri, & Cicchetti, 2010; Shin, Edwards, & Heeren, 2009). Pertinently, reports on drug use indicated that, among college students, alcohol and cannabis are the most commonly used substances (Johnston, O'Malley, Bachman, & Schulenberg, 2011), and that alcohol and drug use patterns in college are a major risk factor for AID (Fell et al., 2010; Norstrom & Rossow, 2013). From a developmental perspective, it has been hypothesized that young adults who had experienced rearing adversity engage in drug use to self-medicate against painful childhood memories. Tension-reduction theories of substance use (Dennhardt & Murphy, 2011; Ham et al., 2013) have purported that coping with negative affect (e.g., anxiety symptoms related to past experiences) is one of the mechanisms by which early onset and progression of substance use may be explained. Beyond the differential pharmacological effects of alcohol and cannabis, less is known about the distinct behavioral phenotypic etiology of these two drugs relative to risk behaviors.

In order to understand the processes that underlie the progression of psychopathology and drug use in relation to childhood adversity experiences, a developmental perspective is necessary (Masten, 2011; Tarter, 2002). As such, risk behaviors in young adulthood are conceptualized as product of a developmental trajectory that commences via adverse family experiences during childhood. Indeed, childhood adversity has been linked to general risk behaviors (Bruce et al., 2013; Wilson & Widom, 2009) including substance use and misuse among college students (Oshri et al., 2015). Although alcohol and cannabis use have been shown to form different indirect paths in predicting the link between early adversity and risk behaviors (Oshri et al., 2012), less is known whether these paths exert differential effects on AID. The psychosocial substance pathways to AID require empirical delineation in order to inform preventive interventions targeting AID among collegiate young adults.

Alcohol and cannabis use: Associations with AID

Alcohol is classified as a depressant drug that inhibits the function of the central nervous system (CNS). In particular, alcohol comprises biphasic effects over the course of the blood alcohol curve, including feelings of euphoria or excitation during the ascending limb and

feelings of sedation during the descending limb. In contrast, cannabinoids (chemicals residing within the cannabis plant) are known to induce perceptual changes, anxiety reduction, analgesia, and mild sedation. Based on the self-medication theory, the psychopharmacological soothing associated with alcohol and cannabis may be particularly reinforcing for youth with histories of adverse rearing environment (Hayatbakhsh et al., 2009; Oshri et al., 2011). The alcohol myopia theoretical model (Steele & Josephs, 1990) postulates that excessive alcohol use can impair inhibition and lead to riskier decision-making. Consequently, individuals who drink heavily allocate their attentional resources to the most salient, easy-to-process, proximal stimuli (e.g., pleasing friends and fitting in), undermining long-term risks (e.g., risk of AID). In addition to the pharmacological effects of drugs upon cognition and behavioral disinhibition (Giancola et al., 2010), individual psychological state (e.g., level of anxiety) in which drug use often occurs may exacerbate or prevent AID (Testa & Livingston, 2009). For example, given that drinking in college often occurs in a social context, general anxiety may induce fear to engage in AID or alternatively accelerate worries regarding peers' approval and influence to commit AID.

Alcohol and cannabis use and AID: The moderating role of anxiety

Despite the common co-occurrence of anxiety disorders and problem drinking, the association between anxiety and drinking among college students is not well understood. Studies have documented that social anxiety, in particular, is negatively linked to weekly alcohol use (e.g., Ham & Hope, 2005; Meade Eggleston et al., 2004), whereas other studies found anxiety a potent risk factor for alcohol use (Birrell et al., 2015). Thus, the literature suggests that the association between social anxiety and drinking is not necessarily linear, thereby purporting that it may play either a jeopardizing or protective role contingent on the drinking context. Specifically, it is conceivable that high social anxiety may potentiate risk for problem drinking as self-medication, whereas subclinical social anxiety may lead youth to inhibit imbibing due to concerns about possible negative evaluation by others (Bruch et al., 1992; Bruch et al., 1997).

Interestingly, recent studies have linked cannabis use and anxiety, suggesting that undergraduates with higher social anxiety may be particularly vulnerable to cannabis use and attendant symptoms of cannabis use disorders (Buckner et al., 2006). In fact, studies have reported that adolescents with anxiety disorders are significantly more likely to develop cannabis dependence as young adults, even after controlling for a wide array of relevant comorbid psychopathology (Buckner & Schmidt, 2008). Hence, acute anxiety may serve as a risk factor in cannabis use and associated sequela. Although the link between drug use and AID is well documented (e.g., LaBrie et al., 2011; Vassallo et al., 2008), little is known about the impact that subclinical levels of general anxiety symptoms may have on this path.

The specific aims and hypotheses of the present study were as follows: (a) to test the links between severity of early adversity and frequency of drug use; we hypothesize that childhood adversity would predict higher levels of alcohol and cannabis use. (b) to test alcohol and cannabis use as mediators of the association between childhood adversity and AID; we hypothesize that early adversity would be linked to AID via increased level of alcohol and cannabis use, independently. (c) to evaluate the role of anxiety in the link

between alcohol, cannabis use and AID; research findings on the impact of anxiety symptoms on substance use have been equivocal, hampering a directional hypothesis. (d) given women's growing representation in AID arrest statistics (Schwartz, 2008), gender differences were explored as a moderator in all examined models.

Method

Participants

Data were obtained from a longitudinal study of social experiences in college (Longitudinal Study of Violence Against Women; White et al., 2001). Four time-points (waves) were utilized in the current study: Freshman (T1; mean age 18.5 years), Sophomore (T2), Junior (T3), and Senior (T4). Data from each time-point were collected at the beginning of each academic year. In the present study, only cohorts that were asked about AID were included in the analyses ($N = 1265$; 59.3% female; 69.2% white).

Measures

Adverse rearing environments—At Time 1, respondents reported on three types of negative experiences as a child: physical abuse (PA), sexual abuse (SA), and witnessing violence between parents (PV). The three items were adapted from a study reported by Malamuth and colleagues (Malamuth et al., 1991) and were used to create a latent factor of adverse rearing environments (see measurement model). *PA* was assessed by one item (*how often did parents hit you?*). Possible responses ranged from 1 to 5 (*never to more than twenty times*). *SA* was assessed by four items ($\alpha = .78$). Participants were asked how often the following experiences occurred to them during childhood (e.g., *another person fondled you in a sexual way*). Possible responses to each item ranged from 1 to 5 (*never to more than five times*). Responses were summed to create a single sexual abuse index. *PV* was assessed by youth report on the violence that occurred between their parents with one item (i.e., *how often did your parents hit each other?*). Possible responses ranged from 1 to 5 as noted in PA above.

Alcohol and cannabis use—Participants reported on their alcohol and cannabis use separately, at each wave (*how often do you drink alcohol? how often do you use marijuana?*). Possible responses ranged from 1 to 5 (*never to more than two times a week*).

Alcohol-impaired driving—At T4, participants reported on how often they drove under the influence of alcohol over the past year (*have you driven a car or another vehicle after having been drinking alcohol*). Possible responses ranged from 1 to 4 (*never to did this a lot*).

Anxiety—General anxiety symptoms were assessed at each time point via multiple items ($\alpha = .78$). Participants were asked to rate past month anxiety symptoms, and possible responses ranged from 1 to 5 (*not at all like me to very much like me*). Responses to each item were averaged to create a single anxiety score.

Analytic plan

Structural equation modeling was used to test the proposed hypotheses. Data were modeled using Mplus Version 7.11 (Muthen & Muthen, 1998–2012). Indirect effects were assessed with the product-of-coefficients ($\alpha^*\beta$) approach (Fritz & Mackinnon, 2007). Conditional indirect effects (moderated mediation paths) were tested using a procedure described by Preacher and colleagues (2007). The recommended statistical fit criteria from Hu and Bentler (1999) were utilized to assess model fit, including the Comparative Fit Index (CFI; Bentler, 1990), the Root Mean Square Error of Approximation (RMSEA; Steiger, 1990), and the Root Mean Square Residual and Standardized index (SRMR; Bentler, 1995). The CFI ranges from 0 to 1 with higher values indicating better fit. The RMSEA and SRMR are bound by 0 and 1 with values ranging from .05 to .08 indicating acceptable fit.

Results

Table 1 displays bivariate correlations between and descriptive statistics of modeled variables.

Measurement model

The adverse rearing environment measurement model was assessed by confirmatory factor analysis (see Figure 1). Maximum likelihood estimator was used to estimate the model. The model was just-identified: $\chi^2(0) = 0$, $p = .00$; CFI = 1.00; RMSEA = .00; SRMR = .00. Loading values ranged from medium to large ($.56 < \lambda < .83$; Brown, 2015).

Adverse rearing environment, substance use, and AID

Statistical model—Structural equation modeling was used to evaluate the longitudinal stability and cross-lagged nature of alcohol use, cannabis use, and anxiety. The rank order correlation (i.e., stability) of the examined variables over time was established (see Figure 2) and cross-lagged effects were controlled for (see Figure 3) per recommendations by Maxwell, Cole, and Mitchell (2011). Adverse rearing environments, AID, and anxiety symptoms by substance use interaction terms were added to the model, and age, minority status, and gender were used as covariates (see Figure 4 for final model). See Table 2 for model fit indexes and parameter estimates of the examined model. Non-significant pathways were trimmed out of the final model with no subsequent significant reduction in model fit. Higher levels of adverse rearing environments significantly predicted higher levels of T1 alcohol use ($\beta = .29$, $p < .01$), cannabis use ($\beta = .23$, $p < .01$), and anxiety symptoms ($\beta = .13$, $p < .01$). In addition, alcohol use, cannabis use and anxiety maintained statistically significant rank order stability over the three time points (i.e., rank order correlations of alcohol from Time 1 to Time 2 and Time 2 to Time 3). Alcohol ($\beta = .39$, $p < .01$) and cannabis use ($\beta = .13$, $p < .01$) at T3, significantly predicted AID at T4, while anxiety did not. Men reported significant higher frequency of AID than did women ($\beta = -.08$, $p < .05$).

Mediation—Indirect effects from adverse rearing environments to AID via alcohol and cannabis use over Waves 1, 2, and 3 were tested with the product-of-coefficients ($\alpha^*\beta$) approach (Shrout & Bolger, 2002). The direct link from adverse rearing environments to AID was controlled for. Analyses revealed significant indirect effects of adverse rearing

environment and AID via alcohol ($\beta = .008$, $SE = .002$, 95% $CI = [.004, .012]$) and cannabis use over time ($\beta = .005$, $SE = .002$, 95% $CI = [.002, .009]$).

Conditional indirect effects—A conditional indirect effect approach was used to examine whether anxiety moderated the path from alcohol and cannabis use at T3 to AID at T4 (Preacher, Rucker & Hayes, 2007). Alcohol, cannabis, and anxiety at T3 were mean-centered before the alcohol $\times$ anxiety and cannabis $\times$ anxiety interaction terms were created. The interaction term of cannabis $\times$ anxiety use significantly predicted AID ($\beta = .07$; $p < .05$). The interaction term of anxiety and alcohol use was not significantly predictive of AID ($\beta = -.05$; $p = n.s.$) and was trimmed out from the SEM model. To interpret these results, we plotted the estimated levels of cannabis use and AID at low (-1 SD) and high ($+1$ SD) levels of anxiety after centering covariates to account for their effects on AID (Aiken, West & Reno, 1991). Individuals who reported higher levels of anxiety and cannabis use reported higher levels of AID than individuals who reported lower levels of anxiety and similar levels of cannabis use (see Figure 5). In order to probe the interaction in the context of indirect effect of adverse rearing environments on AID, the Johnson and Newman (1936) technique was used (see Figure 6). The regions of significance of the mediation were plotted with respect to levels of anxiety. Individuals who reported higher levels of anxiety showed significant indirect associations between adverse rearing environments and AID via cannabis use, whereas individuals who reported lower levels of anxiety showed insignificant indirect associations between adverse rearing environments and AID via cannabis use.

Multiple group analyses—To further explore the effect of gender on AID, multiple group analyses by gender were performed. Model fit was excellent: $\chi^2(136) = 232.28$, $p = .00$; CFI = .98; RMSEA = .03; SRMR = .04. For men, anxiety inversely buffered the link from alcohol use ($\beta = -.24$; $p < .01$) and potentiated the link from cannabis use ($\beta = .21$; $p < .05$) to AID (see Figure 7). Men who reported higher levels of alcohol use and anxiety reported less frequent AID than men who reported relatively lower levels of anxiety or alcohol use. Conversely, men who reported higher levels of anxiety and cannabis use reported increased levels of AID than men who reported relatively lower levels anxiety or cannabis use. For women, anxiety did not significantly moderate the paths from alcohol use ($\beta = .02$; $p = n.s.$) and cannabis use ($\beta = .07$; $p = n.s.$) to AID. In order to test for moderation by gender, chi-square difference tests were computed. Analyses revealed a significant chi-square difference between men and women in the interaction of alcohol and anxiety in predicting AID ($\chi^2(1) = 7.87$); higher levels of anxiety buffered the link from alcohol use to AID among men. In contrast, the interaction between cannabis and anxiety was not statistically different in predicting AID across gender ($\chi^2(1) = 2.45$).

Discussion

Alcohol-impaired driving during college years is a major public health concern. The prevalence rate of AID is high in young adulthood, a developmental phase when substance use behaviors are peaking. Experiencing childhood adversity is a robust developmental risk precursor that can eventuate in increased alcohol and cannabis use during college and attendant risk for AID. The present study tested mechanisms hypothesized to link childhood adversity experiences to substance use and AID among college youths, while exploring the

role of anxiety symptoms within the examined paths. Our results confirmed the hypotheses regarding the longitudinal relations between severity of childhood adversity and alcohol and cannabis use, as well as anxiety symptoms throughout the college years. Importantly, multivariate interaction analyses with anxiety and gender revealed the presence of idiosyncratic pathways to AID.

In the present study significant indirect effects were found to the link from adverse rearing environment to AID at T4 via alcohol and cannabis use at T3. In addition, these indirect effects were further examined for moderation by anxiety, following a moderated mediation tests (Preacher et al., 2007). Finding revealed that individuals with higher levels of anxiety and cannabis use showed significant elevations in AID than individuals with lower levels of anxiety and similar levels of cannabis use. Last, gender differences were tested and confirmed. Among men, higher levels of anxiety served as a moderating factor to the indirect path from early adversity to AID via alcohol use, and as an exacerbating factor of the indirect path via cannabis use. No differences in the conditional indirect effect with anxiety were found for women.

Our findings lend partial support to the self-medication hypothesis. Individuals who were exposed to adverse rearing environments may have used substances to cope with aversive experiences they encountered in childhood. This maladaptive tension-reduction strategy has been previously documented in clinical and community youth samples (Oshri et al., 2012). As expected, the mediation modeling in the present study indicates that alcohol use during college is predictive of increased AID. Commensurate with previous reports on the association between patterns of drinking and AID (Flowers et al., 2008), the present results confirm and extend these data in agreement with psychophysiological evidence and the alcohol myopia model (Giancola et al., 2010). Specifically, individuals who report acute alcohol consumption are more likely to impair their controlled effortful cognitive processing and, thereby, potentiate impulsive decisions such as AID.

Despite the comorbidity of AID and alcohol use, relatively few youth risk driving under the influence. The decision to drive a car while being under the influence of alcohol necessitates significant discounting of the potential risk associated with the behavior. In the present study, anxiety was not found to impact the association between alcohol use and AID. In line with the alcohol myopia conceptual model, our findings demonstrate that alcohol bears a particular strong effect on risky decision making such as AID. However, anxiety symptoms served only as a risk factor for the link between cannabis use and AID. These findings suggest that cannabis use is a particularly important risk factor when used by individuals who report higher levels of anxiety symptoms. It seems plausible that youths who self-medicate to combat the effect of anxiety by using cannabis are more risk-prone compared to those who use alcohol for a similar purpose. It is possible that this distinct effect between alcohol and cannabis on AID in relation to anxiety stems from the differential psychoactive impact cannabinoids have on the brain compared to alcohol. Functional changes in the cannabinoid receptors may result in changes in the levels of various neurotransmitters associated with decision making, in particular dopamine and norepinephrine.

Given the cost of AID to individuals and society (in terms of economics, injury, and death) among male college students, the study of risk and protective factors by gender is critical. Pertinently, when analyzed by gender, the impact of anxiety changed per substance type among men in our study. Anxiety served as a protective factor in the link between alcohol use and AID and in the path from cannabis use to AID. Thus, experiencing anxiety prior to driving under the influence can serve both as a protective function or a risk factor, contingent on gender and the drug used. These nonlinear associations may shed light on the inconsistencies among reports regarding the association between anxiety and drug use. The present findings indicate that men who experience anxiety and who self-medicate with cannabis are at greater risk for AID. Indeed, driving under the influence of substances is likely to involve an impaired decision making as has been suggested by research on the impact cannabis use has on cognitive functioning (Crane, Schuster, & Gonzalez, 2013; Solowij, Stephens, Roffman, & Babor, 2002). Moreover, given that women's prefrontal cortex gray matter matures earlier than in men, it is possible that early use of cannabis use may have a greater neurobehavioral effect on men relative to women. In contrast, increase use of alcohol in conjunction with higher anxiety among men was negatively associated with AID and, as such, anxiety was found to buffer the association between alcohol use and risk for AID. These findings may be attributable to the fact that alcohol use in college years is a normative social behavior that could occur, in specific contexts, without increasing the participation in risky behaviors such as AID (Sznitman, 2008).

Extant strategies for preventing AID have often focused on educational content to discourage youth from operating a vehicle while intoxicated. In addition, empirical research on AID has focused chiefly on prevalence, legal deterrence, and assessment of risk for alcohol abuse seeking to inform universal prevention strategies (Anderson et al., 2009; Centers for Disease Control and Prevention, 2011; Gustin & Simons, 2008). Our findings support the notion that despite the high prevalence of young adult substance use, some are at higher risk for AID than others. College students who had experienced varying degrees of childhood adversity appear to be at increased risk for AID and, as such, this information is important to incorporate into preventive intervention programs targeting college students. Of note, the methodological and conceptual approach adopted in the current study illuminates the developmental history of these youth, delineating specific etiological pathways to AID. The heterogeneity and complexity in the longitudinal associations between childhood diversity and alcohol and cannabis use, and anxiety levels support the need to further adopt a developmental approach to the elucidation of the AID etiology. The disparate paths we found between childhood adversity and AID through substance use may well be associated not only with the specific effects of substance type (alcohol vs. cannabis) but also due to individual differences in risk and protective factors for substance use such as attendant anxiety.

Limitations

This study is not without limitations. First, the study uses self-report measures that may have confounded the results due to shared method effects and recall bias. Forensic records or peer/relative reports would strengthen our findings. Despite the fact that young adults are known to underreport their usage of substances, our study had enough statistical power to

detect the hypothesized effects, thereby mitigating this drawback (Brown, Kranzler, & Del Boca, 1992; Lacey et al., 2011; Ramo et al., 2012). Second, while causal inferences are common in mediation analyses that involve longitudinal data, such inferences should be made cautiously in the absence of experimental methodology. Third, because anxiety symptoms measured in the present study were general and sub-clinical, inferences to specific anxiety disorders are limited.

Implications for prevention

Our findings indicate that exposure to an adverse rearing environments in childhood may potentiate a “domino effect” that culminates in AID via drug use and anxiety symptoms. These results inform competence-enhancement prevention programs that have already been tested among youth (Griffin et al., 2004). The present study sought to inform growing prevention research that has been integrating life-span development research into preventive intervention trials. Specifically, the present study identified empirically verifiable factors (early adversity) and processes (substance use and anxiety) that affect the likelihood of health risk behaviors (AID).

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Figure 1.

Confirmatory factor analysis of adverse rearing environments ($N = 1,265$). The model is a just-identified model. Model fit was good: $\chi^2(00) = 0.00$, $p = .00$; CFI = 1.00; RMSEA = .00. $^{**}p < .01$.

Figure 2.

The stability of alcohol use, cannabis use, and anxiety over time ($N = 1,265$). Covariance is shown in grey. Model fit was good: $\chi^2(18) = 170.81$, $p = .00$; CFI = .91; RMSEA = .08. * $p < .05$; ** $p < .01$.

Figure 3. Cross-lags between alcohol use, cannabis use, and anxiety over time ($N = 1,265$). Covariance is shown in grey. Non-significant pathways removed for clarity. Model fit was good: $\chi^2(18) = 76.38$, $p = .00$; CFI = .97; RMSEA = .03. * $p < .05$; ** $p < .01$.

Figure 4.

The impact of adverse rearing environments on drunk driving through substance use and anxiety (N = 1,265). Age, gender, and minority status were used as covariates. Non-significant paths were removed for clarity. Covariance is shown in grey. PA = Physical abuse; SA = Sexual abuse; PV = Parental violence. Model fit was good: $\chi^2(93) = 337.77$, $p = .01$; CFI = .93; RMSEA = .05; SRMR = .05. * $p < .05$; ** $p < .01$.

Figure 5.
Effect of cannabis $\times$ anxiety on AID ($N = 1,265$).

Figure 6.

Conditional effect of anxiety on the indirect association between adversity rearing environments and driving under the influence via cannabis use. The Johnson–Neyman technique was used to examine the moderating influence of anxiety on the pathway from adverse rearing environments to driving under the influence. The vertical line represents the limit of significance.

Figure 7. Effects of cannabis $\times$ anxiety and alcohol $\times$ anxiety on AID for males ($N = 515$).

Table 1

Descriptive statistics and bivariate correlations among study variables.

Variables	1	2	3	5	6	7	8	9	10	11	12	13	14	15	16
1. PA	–	.20**	.39**	.18**	.11**	.06	.16**	.16**	.13**	.07	.10*	.07	.11*	–.00	–.02
2. SA	.19**	–	.53**	.11**	.05	.08	.16**	.05	.06	.07	.05	.01	.07	–.03	.06
3. PV	.39**	.17**	–	.16**	.04	.04	.13**	.05	.08	.03	–.01	–.05	–.00	–.02	.05
5. Alc1	.18**	.15**	.13**	–	.70**	.60**	.45**	.40**	.36**	.12**	.07	.01	.34**	–.03	–.16**
6. Alc2	.05	.03	.04	.23**	–	.66**	.39**	.44**	.35**	.04	.10*	.05	.39**	–.02	–.21**
7. Alc3	.01	.18**	–.02	.64**	.20**	–	.32**	.36**	.41**	.08	.10*	.10*	.39**	.03	–.20**
8. Cann1	.12**	.16**	.11*	.50**	.12*	.30**	–	.69**	.52**	.05	.08*	.01	.25**	–.03	–.06
9. Cann2	.09	.18*	.10	.65**	.26**	.55**	.53**	–	.60**	.03	.04	.04	.25**	–.06	–.12**
10. Cann3	.06	.21**	.08	.36**	.13*	.40**	.45**	.52**	–	–.01	.06	.03	.26**	.04	–.08
11. Anx1	.14**	.03	.10*	.09*	–.05	.11	.14**	.09	.08	–	.47**	.39**	.09	.02	–.09*
12. Anx2	.04	.01	.04	.17**	.02	.08	.17**	.13*	.04	.58**	–	.52**	.07	–.00	–.12**
13. Anx3	–.05	–.01	.01	.04	–.16*	.11	.07	–.01	.06	.36**	.43**	–	.10*	.02	–.11**
14. AID	–.03	.10	–.01	.54**	.22**	.52**	.28**	.46**	.32**	.10	.09	–.02	–	–.01	–.05
15. Age	–.04	–.06	–.04	–.06	–.35**	–.08	–.00	–.19*	–.01	–.05	–.07	.06	–.01	–	–.05
16. Min	–.01	–.09*	–.06	–.05	–.45**	–.12*	.03	–.26**	–.08	.01	–.05*	.09	–.17*	.54**	–
Mean	1.37	5.46	1.13	2.35	2.74	2.38	1.35	1.54	1.44	1.5	2.33	2.24	2.27	2.86	1.30
(SD)	.74	2.81	.57	1.16	1.35	1.14	0.79	1.01	.91	.819	.87	.83	.69	.66	.46
Skewness	2.66	4.51	8.35	.53	.35	.49	2.69	1.98	2.50s	.71	.88	1.01	1.66	–.42	.86

Note. Correlations for males are presented below the diagonal ($n = 515$); correlations for females are presented above the diagonal ($n = 750$). 1, 2, and 3 refer to time points. PA = Physical abuse. SA = Sexual abuse. PV = Parental violence. Alc = Alcohol use. Cann = Cannabis use. Anx = Anxiety. AID = Alcohol-Impaired Driving. Min = Minority status. Minority status coded: 1 = white, 2 = minority. Gender coded as 0 = male, 1 = female. Age = number of years born after 1970.

* $p < .05$ ** $p < .01$

Table 2Parameter estimates of direct, indirect, & conditional effects of final model ($N = 1,265$).

	B(SE)	β	95% CI
<i>Direct effects</i>			
ARE $\rightarrow$ Alcohol Use T1	.69 (.11)	.29	[.21, .36] **
ARE $\rightarrow$ Cannabis Use T1	.38 (.07)	.23	[.16, .30] **
ARE $\rightarrow$ Anxiety T1	.24 (.07)	.13	[.06, .21] **
ARE $\rightarrow$ AID	.10 (.10)	.06	[-.05, .17]
Alcohol Use T1 $\rightarrow$ Alcohol Use T2	.52 (.03)	.44	[.40, .49] **
Alcohol Use T1 $\rightarrow$ Cannabis Use T2	.26 (.02)	.30	[.25, .35] **
Cannabis Use T1 $\rightarrow$ Cannabis Use T2	.62 (.04)	.48	[.43, .52] **
Cannabis Use T1 $\rightarrow$ Anxiety T2	.09 (.03)	.08	[.02, .14] **
Anxiety T1 $\rightarrow$ Anxiety T2	.49 (.03)	.51	[.46, .55] **
Alcohol Use T2 $\rightarrow$ Alcohol Use T3	.15 (.03)	.17	[.11, .27] **
Cannabis Use T2 $\rightarrow$ Cannabis Use T3	.34 (.03)	.39	[.31, .43] **
Cannabis Use T2 $\rightarrow$ Alcohol Use T3	.18 (.04)	.16	[.10, .23] **
Anxiety T2 $\rightarrow$ Anxiety T3	.34 (.03)	.41	[.35, .47] *
Alcohol Use T3 $\rightarrow$ AID	.28 (.03)	.39	[.32, .46] **
Cannabis Use T3 $\rightarrow$ AID	.11 (.04)	.13	[.04, .21] **
Anxiety T3 $\rightarrow$ AID	.03 (.04)	.03	[-.05, .10]
<i>Indirect Effect ($\alpha^* \beta$)</i>			
ARE $\rightarrow$ Alcohol T1, T2, T3 $\rightarrow$ AID	.014 (.004)	.008	[.004, .012] **
ARE $\rightarrow$ Cannabis T1, T2, T3 $\rightarrow$ AID	.009 (.003)	.005	[.001, .009] **
<i>Conditional Effect ($W^* \beta$)</i>			
Anxiety T3 $\times$ Cannabis Use T3 $\rightarrow$ AID	.10 (.05)	.07	[.002, .146] *
<i>Covariates</i>			
Gender $\rightarrow$ Alcohol Use T2	-1.08 (.07)	-.40	[-.44, -.35] **
Gender $\rightarrow$ Cannabis Use T2	-.46 (.05)	-.22	[-.26, -.18] **
Gender $\rightarrow$ AID	-.15 (.07)	-.09	[-.16, -.01] *

Note. Model fit was acceptable: $\chi^2(93) 337.77, p = .00$; CFI = .93; RMSEA = .05; SRMR = .05. T1, T2, and T3 refer to time points. AID = Alcohol-Impaired Driving. ARE = Adverse rearing environment. Gender coded as 0 = male, 1 = female.

* $p < .05$

** $p < .01$.