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Comorbidity and Temporal Relations of Alcohol and Cannabis Use Disorders from Youth through Adulthood

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Abstract

Background—Alcohol and cannabis are among the most widely used and abused drugs in industrialized societies. Investigations of patterns in comorbidity and temporal sequencing between alcohol use disorders (AUDs) and cannabis use disorders (CUDs) from childhood to adulthood are important for understanding the etiologies of these disorders.

Methods—The sample comprised 816 individuals (59% male, 89% white). Dichotomous measures indicated whether or not a participant was in an AUD or CUD episode during three developmental periods —youth (childhood through adolescence), early adulthood, and adulthood. Structural equation modeling was used to determine relations between AUDs and CUDs across the three developmental periods, and to test for gender differences.

Results—Concurrent associations between AUD and CUD were significant. Both AUD and CUD in previous developmental periods significantly predicted the same substance disorders in subsequent periods. Cross-lagged paths from youth AUD to young adult CUD and youth CUD to young adult AUD were both significant. However, only the cross-lagged path from youth CUD to adult AUD was significant. The cross-lagged paths from young adult AUD to adult CUD and young adult CUD to adult AUD were both nonsignificant. Males and females were mostly similar with only three differences found between genders.

Conclusions—Comorbidity of AUDs and CUDs was evident from youth through adulthood but the strength of the relationship lessened in adulthood. Temporal sequencing influences of AUDs

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Contributors

SD and RF had the initial idea and conducted background literature searches. PL and JS were involved in the original study protocol and data collection. JG conducted statistical analyses. SD and JG wrote the first draft of the manuscript. RF, JS, DK, and PL contributed to interpreting the findings and writing further drafts of the manuscript. All authors reviewed and have approved the final manuscript.

Conflict of Interest

No conflict declared.

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and CUDs on each other were similar in youth and adulthood but not young adulthood. Same substance stability was greatest in adulthood.

Keywords

Alcohol use disorders; Cannabis use disorders; Comorbidity; Gender differences; Youth; Young adulthood

1. INTRODUCTION

Alcohol and cannabis use disorders (AUDs, CUDs) are among the most common psychiatric disorders in industrialized countries, and are associated with substantial personal, societal, health, and economic costs (Boden et al., 2006; Compton et al., 2007; Haberstick et al., 2014; Hall and Degenhardt, 2009; Rehm et al., 2009). Studies have found evidence of homotypic comorbidity (i.e., the cooccurrence of disorders within a diagnostic grouping; Angold et al., 1999) between alcohol and cannabis use and abuse (Degenhardt et al., 2001a, 2001b). Evidence of comorbidity is based on similar initial onset ages, strength of associations of use and abuse patterns, and temporal relations between the two substances (Flory et al., 2004; Hix-Small et al., 2004; Jackson et al., 2008; Kirisci et al., 2013).

Documenting comorbidity patterns of AUDs and CUDs in terms of strengths of comorbid associations and temporal relations is important for improving our understanding of the etiology of these disorders and for informing prevention efforts (Flory et al., 2004; Hayatbakhsh et al., 2009). There are a number of possible explanations for comorbidity and associations between AUD and CUD. Their comorbidity, for example, could be due to common risk factors such as socioeconomic disadvantage, ineffective parenting, or affiliations with deviant peer groups (Kagan, 2013; Patterson et al., 1989). Alternatively, there may be a liability to addiction (e.g., genetic vulnerabilities) that underlies the likelihood of substance misuse in general (Vanyukov et al., 2003, 2012), or a causal sequence of stages associated with the use and abuse of these substances, as posited by the “gateway” hypothesis (Fergusson et al., 2006; Kandel et al., 1992).

While past research indicates cooccurrence between alcohol and cannabis use in adolescence and young adulthood (Flory et al., 2004; Jackson et al., 2008; Mason et al., 2013), there have been comparatively few prospective studies using diagnostic data of either AUDs or CUDs (e.g., Hayatbakhsh et al., 2009; Stinson et al., 2005). Hayatbakhsh et al. (2009) investigated early risk factors of CUDs in young adulthood but did not include relations with AUDs, and Stinson et al. (2005) documented the 12-month prevalence of AUDs, other substance use disorders (SUDs), and comorbidity of different SUDs in the U. S. population. Research is extremely limited on the comorbid relations between AUDs and CUDs during different developmental periods, specifically from youth through early adulthood. The temporal sequencing of relations between AUDs and CUDs across developmental periods from youth through adulthood also has been largely unexplored. Coffey et al. (2003) found no statistically significant evidence of comorbidity between cannabis dependence during early adulthood and frequent or heavy alcohol use during adolescence. The authors concluded that their findings might illustrate a social process whereby individuals select into either a predominantly alcohol-using or cannabis-using lifestyle, thus attenuating or negating

comorbidity in early adulthood. In general, the longitudinal comorbid relations between AUDs and CUDs and the temporal sequencing of these relations across developmental periods from youth through adulthood are not well defined and are largely unknown.

In addition, limited research has investigated possible gender differences in comorbidity and temporal relations between AUDs and CUDs. Differences in levels of alcohol and cannabis use, abuse, and dependence have been documented as a function of gender, with males generally exhibiting higher rates for each substance than females (Coffey et al., 2003; Hayatbakhsh et al., 2009; Lev-Ran et al., 2013; Mason et al., 2013; Perkonig et al., 2008). Schulte et al. (2009) suggested that prevalence rates of alcohol use are similar for boys and girls in adolescence, but in young adulthood boys become increasingly more at risk for problematic drinking and AUDs. In general, there is a dearth of research that has documented gender similarities or differences in the strength of comorbidity between AUD and CUD or evaluated the temporal relations between AUDs and CUDs through youth/adolescence, early adulthood, and adulthood.

The current longitudinal study included prospective data as well as diagnostic data to define AUD and CUD episodes in youth (childhood and adolescence; ages 6–17.9), through early adulthood (ages 18–23.9) to adulthood (ages 24–30). Low prevalence rates prohibited the examination of childhood separately from adolescence. The primary aim was to examine comorbidity patterns and temporal relationships between AUDs and CUDs across these three developmental periods to determine whether AUD and CUD comorbidity patterns and temporal relationships differed across males and females. Knowledge of the temporal sequencing of AUDs and CUDs and possible gender differences within each stage of development would help clarify the nature of their relationships and whether both disorders share common or distinct etiological processes.

2. METHODS

2.1 Participants

Oregon Adolescent Depression Project (OADP) participants were followed at four time points (T_1 through T_4) between 1987 and 2001. An institutional review board granted approval prior to the collection of study data. Participants completed informed consent procedures.

The OADP sample was randomly drawn from nine high schools in two urban and three rural communities in western Oregon. Three cohorts were recruited for a total T_1 sample of 1,709 (M age = 16.6, SD = 1.2) with an overall participation rate of 61%. Demographic characteristics of the T_1 sample were very similar to corresponding regional census data, and follow-up phone contacts with nonparticipants revealed no demographic differences with participants in head of household gender, family size, number of parents in household, parents' employment status, middle class socioeconomic status, or race (Lewinsohn et al., 1993). These findings suggest that the T_1 sample was representative of the regional population from which it was drawn.

Approximately one year later (T_2), 1,507 participants (88%) were reassessed (M age = 17.7, SD = 1.2). Between 1993 and 1999, as participants reached their 24th birthday, all individuals with a history of psychopathology (n = 644) and a randomly selected set of participants with no history of mental disorder (n = 457) were invited to participate in a third (T_3) evaluation. Sampling of the no-disorder comparison group was proportional to age and gender within age. To increase racial and ethnic diversity within the sample, all participants with non-White ethnicity were retained for the T_3 sample. Of the 1,101 T_2 participants selected for the T_3 assessment, 941 (85%) completed the evaluation. At age 30, all T_3 participants were asked to complete another interview assessment. Of the 941 who participated in T_3 , 816 (87%) completed the T_4 assessment. Thus, from T_1 to T_2 , T_2 to T_3 , and T_3 to T_4 , retention rates for eligible participants were 88%, 85%, and 87%, respectively. Detailed published analyses revealed minimal sample biases related to attrition (Farmer et al., 2013; Lewinsohn et al., 1993). Recent analyses also considered attrition based on participants who (a) dropped out between T_1 and T_2 , (b) were recruited at T_3 but did not participate, or (c) dropped out between T_3 and T_4 (Farmer et al., 2013). Specifically, the T_4 panel was compared with the attrition group with respect to psychiatric history (i.e., any lifetime DSM-defined disorder diagnosis) and the cumulative number of lifetime psychiatric disorders at T_1 . The T_4 panel was not statistically different from the attrition group with respect to positive psychiatric histories (p = .96) or the cumulative number of lifetime disorders (p = .23) at T_1 .

The 816 probands (59% female, 89% White, 53% married, M age = 30.4, SD = 0.7) who participated in the T_4 panel constitute the reference sample for the present study.

2.2 Diagnostic Measures

During T_1 , T_2 , and T_3 , participants were interviewed with a version of the Schedule for Affective Disorders and Schizophrenia for School-Age Children (K-SADS) that combined features of the Epidemiologic and Present Episode versions (Chambers et al., 1985; Orvaschel et al., 1982). Follow-up assessments of disorders at T_2 and T_3 also involved the joint administration of the Longitudinal Interval Follow-Up Evaluation (LIFE; Keller et al., 1987) that, in conjunction with the K-SADS, provided detailed information related to the presence and course of disorders since participation in the previous diagnostic interview. Thus, at each assessment, both current and “since last interview” diagnostic assessments were performed (in the case of T_1 , the retrospective timeframe was any time before T_1). The T_4 assessment included administration of the LIFE and the Structured Clinical Interview for Axis I DSM-IV Disorders–Non-Patient Edition (SCID-NP; First et al., 1994). Different diagnostic interviews were used at different time points so that assessments would be age-appropriate (K-SADS for adolescence and emerging adulthood, and SCID-NP for adulthood). While the K-SADS and SCID-NP differed marginally on phrasing of items, both were comparable in that they allowed for evaluation of all symptoms necessary to code AUD and CUD diagnoses. Symptom reports were evaluated in accordance with DSM-III-R diagnostic criteria at T_1 and T_2 , and DSM-IV diagnostic criteria at T_3 and T_4 . In the case of both AUDs and CUDs, both abuse and dependence were assessed.

Diagnoses of AUDs at all assessment periods were performed with good to excellent interrater reliability (κ s: $T_1 = .76$, $T_2 = .89$, $T_3 = .69$, $T_4 = .79$), as were diagnoses of CUDs (κ s: $T_1 = .72$, $T_2 = .93$, $T_3 = .83$, $T_4 = .82$). For purposes of this report, diagnosis and onset data were used to create six dichotomous measures that indicated whether (coded 1) or not (coded 0) a participant was in an AUD or CUD episode during each of three developmental periods—youth (ages 6–17 years), early or young adulthood (ages 18–23 years), and adulthood (ages 24–30 years).

2.3 Statistical Methods

Analyses were conducted using structural equation modeling (SEM). Initially, latent growth modeling (LGM) analyses were conducted to determine if there were significant trajectories of AUDs and CUDs over time. No significant developmental trajectories were found; therefore, autoregressive panel modeling in SEM was used to examine relationships between AUDs and CUDs over time. First, we tested a preliminary unconditional SEM model (without regression effects) to determine whether there were any mean differences in AUDs and CUDs at each of the developmental periods across males and females. The only significant difference was for young adult AUD (ages 18–23.9 years), where males had significantly higher means than females ($\chi^2_{\text{diff}[1]} = 4.15$, $p < .05$). Next, multiple sample (by gender) SEM was used to test an autoregressive model that included lagged and cross-lagged paths across the three developmental periods and between AUDs and CUDs (see Figure 1). Models were estimated using Mplus 7.1 software (Muthén and Muthén, 1998–2012) with a weighted least square estimator and sampling design weights to account for the binary observed data and stratified sampling at T_3 .

A baseline autoregressive model was estimated with all model parameters constrained to be equal across gender with the exception of the young adult AUD thresholds where significant mean differences between genders had already been determined. Lagged and cross-lagged parameters were added and a chi-square difference test of nested models was used to determine if the fully lagged and cross-lagged model provided a significantly better fit to the data than did the autoregressive only model. Finally, gender differences were examined by testing the invariance of each parameter, one at a time, and by computing a chi-square difference test between nested models. The parameter associated with the lowest significant p -value ($< .05$) was allowed to remain free and the process repeated until no significant gender differences were detected. Model fit was assessed with the chi-square test of model fit and based on recommended cutoff values by Hu and Bentler (1999), comparative fit index (CFI) $> .95$, root mean square error of approximation (RMSEA) $< .06$, and weighted root mean square residual (WRMR) $< .90$.

3. RESULTS

Rates of AUD and CUD by developmental period are shown in Table 1 and are comparable to ranges of AUD and CUD found in other community-based studies. For example, Moffitt et al. (2010) reported a 31.8% lifetime rate of alcohol dependence, and lifetime estimates of AUDs have ranged from 15.9% to 32.4% in other studies (e.g., Kringlen et al., 2001; Reinherz et al., 1993; Wittchen et al., 1998). Similarly, two prospective studies from New Zealand suggest that the risk for developing a cannabis dependence disorder is between

12.5% and 18% (Boden et al., 2006; Moffitt et al., 2010), and Wittchen et al. (2007) reported a 13.7% lifetime rate of CUDs.

As noted earlier, results of the unconditional model showed significant gender differences in the means for young adult AUD (Males .336, Females .232, $p < .05$). Fit indices for the autoregressive model and the fully lagged and cross-lagged models are shown in Table 2. The autoregressive model showed moderate but not adequate fit as evidenced by failures to meet recommended cutoff values for all fit indices. The fully lagged and cross lagged model fit the data significantly better than the autoregressive only model ($\chi^2_{diff}[8] = 58.27, p < .001$). In order to examine the proposed research questions this model was retained and the invariance of the parameter estimates tested between males and females.

Results of invariance tests showed two additional parameters significantly differed by gender: the lagged path from youth CUD to adult CUD ($\chi^2[1] = 8.40, p = .004$) and the autoregressive path of adult CUD regressed on young adult CUD ($\chi^2[1] = 4.13, p = .042$). Allowing these parameters to be estimated separately for males and females resulted in a final lagged and cross-lagged model with good fit to the data (see Table 2). Final parameters estimates are shown in Table 3. The correlations between young adult AUD and CUD and between adult AUD and CUD were both significant. The relationship between AUD and CUD was stronger for young adults than adults (.53 vs. .30), suggesting greater comorbidity between AUDs and CUDs in early adulthood and less comorbidity as individuals aged.

The autoregressive paths showed that both AUD and CUD in the previous developmental period significantly predicted AUD and CUD in the subsequent period indicating relative stability of AUDs and CUDs from youth through adulthood. For both AUD and CUD, stability was stronger from the young adult to adult periods compared to the youth to young adult periods. The standardized coefficients also suggest the young adult to adult CUD relationship was more stable than the young adult to adult AUD relationship. Furthermore, the young adult to adult CUD relationship showed significantly greater stability for males compared to females. The lagged path of youth AUD to adult AUD was nonsignificant, whereas the lagged path from youth CUD to adult CUD was significant and negative for females (standardized estimate = $-0.21, p < .001$) and nonsignificant for males (standardized estimate = $0.01, p = .943$). This relationship indicates that for women only, in addition to proximal influences, there may be a more distal influence whereby having a CUD in youth is related to not having a CUD in adulthood.

The cross-lagged paths from youth AUD to young adult CUD and from youth CUD to young adult AUD were both significant and the magnitudes similar, indicating that each specific substance use disorder during youth is a similar predictor of the other substance use disorder during young adulthood. However, the cross-lagged paths from youth CUD to adult AUD and youth AUD to adult CUD were different, whereby the path from youth CUD to adult AUD was significant while the path from youth AUD to adult CUD was nonsignificant. This suggests that having a CUD in youth may be more predictive of later AUDs than having an AUD in youth is of later CUDs. The cross-lagged paths from young adult AUD to adult CUD and young adult CUD to adult AUD were both nonsignificant. The overall cross-lagged path results suggest that the influences of AUDs and CUDs on each

other appear to lessen throughout the three developmental periods, from childhood through age 30.

4. DISCUSSION

This study examined patterns of comorbidity and temporal relations between AUDs and CUDs across three developmental periods between childhood and age 30: youth (ages 6 to 17.9), young adulthood (ages 18.0 to 23.9), and adulthood (ages 24.0 to 30). We also evaluated whether sequencing and comorbidity patterns differed as a function of gender. The findings of this study show evidence of comorbidity between AUDs and CUDs, particularly during the earlier stages of development. In addition, the cross-lagged paths from youth AUD to young adult CUD and from youth CUD to young adult AUD were significant, indicating temporal patterns of comorbidity. Thus, these findings lend further support for the successive comorbidity between these two substance use disorders, at least during these developmental periods.

An interesting difference between the two substances was that the cross-lagged path from youth CUD to adult AUD was significant while the path from youth AUD to adult CUD was not, highlighting some possible differences in patterns of influence between AUDs and CUDs across developmental periods. This result may indicate, for example, that having a CUD in youth is more predictive of later AUDs in adulthood, and may therefore be a more potent risk factor for later AUDs than early AUDs are for later CUDs. This result is in concordance with other research that indicates early and heavy cannabis use may be a particularly salient risk factor for the development and continuity of later substance use disorders (Degenhardt et al., 2010; Fergusson et al., 2002). It also provides some support, albeit limited, for the gateway hypothesis where a causal sequence of substance use and abuse has been proposed (Fergusson et al., 2006; Kandel et al., 1992). However, while some temporal sequencing was evident in the current study, the cross-lagged relations for AUD and CUD were generally similar. In addition, the consistent findings for comorbidity of AUDs and CUDs, as well as stability of AUDs and CUDs from youth through adulthood, indicate less of a causal sequence and more of a similarity in AUDs and CUDs patterns across developmental periods.

The current study's findings support a general comorbidity of AUDs and CUDs, but also point to developmental period influences and a lessening in comorbidity between AUDs and CUDs as young people enter adulthood. The correlation between adult AUDs and CUDs (.30), while significant, was considerably weaker than between young adult AUDs and CUDs (.53). In addition, the cross-lagged temporal paths from young adult AUD to adult CUD and young adult CUD to adult AUD were both nonsignificant, while they had been significant during earlier developmental periods. The overall cross-lagged path results suggest that the relations between and influences of AUDs and CUDs on each other lessen throughout the three developmental periods spanning childhood through age 30. Our findings suggest less comorbidity in AUDs and CUDs in the oldest developmental period of adulthood (ages 24–30) as well as less temporal influence of AUDs and CUDs on each other in adulthood. It may be that comorbidity is greater earlier on, during childhood, adolescence, and young adulthood, but that prior use of the same substance (stability) starts to take over as a stronger

predictor of continued substance-specific use or abuse during adulthood. Others have reported similar results. Patton et al. (2007), for example, found that levels of cannabis and alcohol use were associated in adolescence and adulthood, but the strength of the association declined as the cohort aged and there was a tendency for higher level users to use one substance predominantly. Coffey et al. (2003) similarly concluded based on their findings that some adults may select into either a predominantly alcohol-using or cannabis-using lifestyle. Our findings illustrate a similar process whereby individuals are more likely to select into either an alcohol-abusing or cannabis-abusing lifestyle as they entered adulthood.

As expected, findings in the present research also showed considerable stability in AUDs and CUDs across youth, early adulthood, and adulthood. Having an AUD or CUD in one developmental period significantly predicted having an AUD or CUD, respectively, in the subsequent developmental period. This finding was consistent across all three developmental periods. The stability coefficients for both AUDs and CUDs were stronger from the young adult to adult periods when compared with the youth to young adult periods, indicating greater stability of disorders within each substance at the older developmental periods. At the same time there was also a lessening in comorbidity between substances as participants aged. Prior studies have demonstrated stability in cannabis and alcohol use in early adulthood. Perkonig et al. (2008), for example, found stable frequent cannabis use in their sample throughout the third decade of life and low rates of remission until about 34 years of age. AUDs generally show a strong age gradient with typical onset during adolescence, and while some adolescents who experience AUDs “mature out,” others display a more persistent course beyond adolescence (Sher et al., 2005).

Results suggested more similarities than differences across genders. The only significant difference in prevalence was for AUDs in young adulthood where males had a higher rate of AUDs than females. This is in line with results of other studies where men are often shown to be at greater risk for problematic drinking and AUDs during young adulthood and adulthood compared to women (Nolen-Hoeksema and Hilt, 2006; Schulte et al., 2009). There was also a significant gender difference where men in the young adult to adult CUD relationship showed significantly greater stability compared to women. Limited research (Lynskey et al., 2006) has documented differences across genders for stability in cannabis dependence, with men demonstrating greater lifetime durations of dependence episodes than women. Reasons for gender differences in stability observed here and in other research are less clear, but may be related to genetic factors (Lynskey et al., 2006) or gender differences in maturation processes during the transition from young adulthood to adulthood (e.g., age at marriage, parenthood). Additionally, as has been suggested with regard to AUDs (Nolen-Hoeksema and Hilt, 2006), women, when compared to men, may carry certain protective factors against continued CUDs, including lower expectations of positive outcomes and greater perceived social sanctions for cannabis use.

Another significant gender difference was found for the lagged path from youth CUD to adult CUD, which was significant and negative for women and nonsignificant for men (the same path for AUDs was nonsignificant for all). This relationship may indicate that for women only, in addition to proximal influences, there may be a more distal influence whereby having a CUD during youth acts as a protective factor against having a CUD as an

adult. Again, lower perceived positive outcomes and greater social sanctions and negative consequences among women compared to men may help explain this finding (Nolen-Hoeksema and Hilt, 2006).

Although the present study has several strengths, there are also limitations that suggest some caution in the interpretation of the findings. Study strengths include OADP's study design, which is mostly prospective, as well as the use of diagnostic data and the focus on alcohol and cannabis use disorders rather than simply substance use. Prospective studies allow for more accurate investigations of temporal relations among the substance use disorders. Childhood and early adolescent diagnostic data in the present research, however, were based on retrospective assessments collected at T₁ (~ age 16). Retrospective assessments are known to introduce recall-related biases that generally favor the underreporting of psychopathology (Moffitt et al., 2010). Different interview methods and versions of DSM were used to assess and diagnose disorders across waves. It is possible that the use of different interviews and changes in diagnostic criteria for some disorder categories could have introduced method bias into the study that, in turn, differentially influenced decisions concerning disorder occurrences across assessment waves. Past year and lifetime diagnostic agreement between DSM-III-R and DSM-IV for alcohol and drug use disorders, however, is good to excellent (Grant, 1996), and is unlikely to be a source of significant bias. Limitations also include the predominantly White sample, which precluded an evaluation of models separately as a function of racial and ethnic subgroups, and the possibility that AUD and CUD rates for contemporary youth may differ to some extent from the current data.

In addition, this study focused predominantly on comorbidity and relationships between AUD and CUD, and consequently did not include potential confounding or explanatory variables. Future studies are encouraged to include variables that might help explain not only the comorbidity between AUD and CUD but also the temporal relations between AUD and CUD from youth through adulthood. For example, the inclusion of common risk factors such as socioeconomic disadvantage, ineffective parenting, or affiliations with deviant peer groups (Kagan, 2013; Patterson et al., 1989) might help explain the comorbidity and temporal relations between AUD and CUD, as may a genetic or biological vulnerability to substance abuse in general (Vanyukov et al., 2003, 2012).

The findings of this study highlight possible similarities and differences in comorbidity and temporal relations between AUD and CUD from youth through adulthood (age 30 years) across men and women. More longitudinal, prospective studies using diagnostic data are needed to determine whether these findings are replicated in other samples and subgroups. Future studies might also include family history as a predictor of AUD and CUD, as well as potential moderating factors (e.g., family influences) of temporal relations between AUD and CUD in order to better understand the etiology of dual cannabis and alcohol use disorders and relationships between the hazardous use of these two substances as a function of gender. It also would be of interest to determine the comorbidity and temporal relations between AUD, CUD, and other psychiatric disorders. The comorbidity findings in this study underscore the importance of directing prevention efforts at high risk youths prior to their first substance exposure to alcohol or cannabis (Kirisici et al., 2013; Perkonig et al., 2008)

in order to minimize early and frequent experiences with cannabis and alcohol and, consequently, the lessening of AUD and CUD stability in young adulthood and adulthood.

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Highlights

- We examined relations between AUDs and CUDs from youth through adulthood.
- Comorbidity of AUDs and CUDs was evident but lessened in adulthood.
- Temporal sequencing influences of AUD and CUD were similar in youth and adulthood.
- Same substance stability was greatest in adulthood.

Figure 1.

Autoregressive and fully lagged and cross-lagged model where *a* through *f* represent autoregressive parameters, *g* through *h* lagged parameters, and *i* through *n* represent cross-lagged parameters. Youth AUD and CUD are exogenous indicators, and with the delta parameterization specified only contribute regression parameters, thus no correlation is estimated. AUD = alcohol use disorder, CUD = cannabis use disorder. Dashed lines significantly differ by gender. ***p* < .01, ****p* < .001.

Table 1
Rates of alcohol use and cannabis use disorders at different developmental periods.

	Youth		Young Adult		Adult	
	N	%	N	%	N	%
Alcohol use disorder						
Female (n = 480)	42	6.9	111	21.8	71	13.1
Male (n = 336)	33	7.7	133	35.6	83	22.1
Total (n = 816)	75	7.2	244	27.9	154	17.0
Cannabis use disorder						
Female	39	6.9	58	11.5	29	5.7
Male	43	9.4	66	17.6	47	12.8
Total	82	8.0	124	14.2	76	8.8

Note: Ns are observed and percentages are weighted based on sampling stratification procedures implemented at T3.

Table 2

Model fit statistics.

Model	χ^2	df	p-value	CFI	RMSEA	WRMR
Autoregressive	76.17	21	<.001	.92	.08	2.15
Fully lagged and cross-lagged	29.67	13	.020	.98	.05	1.27
Final fully lagged and cross-lagged	7.95	11	.718	1.00	.00	0.63

Note: df = degrees of freedom; CFI = confirmatory fit index; RMSEA = root mean square error of approximation; WRMR = weighted root mean square residual.

Table 3

Parameter estimates for final multiple group model.

Path	Parameter	Female		Male	
		Estimate	p-value	Estimate	p-value
a	Youth AUD → Young adult AUD	0.27	<.001	0.33	<.001
b	Young adult AUD → Adult AUD	0.65	<.001	0.41	<.001
c	Youth CUD → Young adult CUD	0.32	<.001	0.34	<.001
d	Young adult CUD → Adult CUD	0.81	<.001	0.87	<.001
e	Young adult AUD ↔ Young adult CUD	0.53	<.001	0.53	<.001
f	Adult AUD ↔ Adult CUD	0.30	.003	0.30	.003
g	Youth AUD → Adult AUD	-0.07	.189	-0.05	.189
h	Youth CUD → Adult CUD	-0.21	.007	0.01	.943
i	Youth AUD → Young adult CUD	0.15	<.001	0.15	<.001
j	Youth AUD → Adult CUD	-0.07	.244	-0.06	.244
k	Young adult AUD → Adult CUD	-0.08	.539	-0.06	.539
l	Youth CUD → Young adult AUD	0.12	.004	0.16	.004
m	Youth CUD → Adult AUD	0.16	.003	0.13	.003
n	Young adult CUD → Adult AUD	0.01	.918	0.01	.918

Note: Path letters correspond to Figure 1. Standardized estimates are provided. Bolded entries significantly differ between genders at $p < .05$.