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Disentangling longitudinal relations between youth cannabis use, peer cannabis use, and conduct problems: developmental cascading links to cannabis use disorder

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ABSTRACT

Aims To determine whether cannabis use during adolescence can increase risk not only for cannabis use disorder (CUD) but also for conduct problems, potentially mediated by exposure to peers who use cannabis. **Design, Setting, Participants** Longitudinal study analyzing four waves of longitudinal data from 364 racially and socio-economically diverse, urban, US community youth (at baseline: M_{age} = 13.51 (0.95); 49.1% female). **Measurements** Self-reports of cannabis use, conduct problems, proportion of peers using cannabis and CUD criteria at the final wave were analyzed using a method sensitive to changes over development, the random-intercept cross-lagged panel model. **Findings** Change in cannabis use did not predict changes in conduct problems or peer cannabis use over time, controlling for gender, race–ethnicity and socio-economic status. Instead, increases in conduct problems predicted increases in cannabis use and ultimately CUD, with some of the effect mediated by increases in the prevalence of peer cannabis use [β = 0.12, 95% confidence interval (CI) = 0.07, 0.20]. Additionally, affiliation with peers who used cannabis predicted subsequent CUD via increased personal cannabis use (β = 0.08, 95% CI = 0.04, 0.14). Significant within-person betas for the cross-lagged effects ranged between 0.20 and 0.27. **Conclusions** Cannabis use in adolescence does not appear to lead to greater conduct problems or association with cannabis-using peers apart from pre-existing conduct problems. Instead, adolescents who (1) increasingly affiliate with cannabis-using peers or (2) have increasing levels of conduct problems are more likely to use cannabis, and this cascading chain of events appears to predict cannabis use disorder in emerging adulthood.

Keywords Adolescence, cannabis use, cannabis use disorder, conduct problems, longitudinal, peer influence.

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INTRODUCTION

More youth use cannabis than smoke cigarettes in the United States [1], and in other parts of the world cannabis use has become almost as regular as tobacco use among adolescents and young adults [2]. In the United States, many states and jurisdictions have relaxed laws against possession of small amounts of this drug, and more states are considering taking a similar course. One of the first meta-analyses on the effects of these trends did not show that legalization of medical cannabis until 2014 in the United States increased the prevalence of adolescent cannabis use [3]. However, the currently limited research is mixed on whether trends toward legalization of recreational cannabis lead to greater access to cannabis for youth, which could subsequently lead to greater use of this drug [4–7]. This is an important question, as increased adolescent cannabis use could potentially have adverse consequences, considering that some adolescents are neurobiologically vulnerable to experimentation and subsequent addiction to such substances [8,9]. Hence, it is critical and timely to examine the precursors and potential adverse consequences associated with patterns of cannabis use among youth and whether these associations lead to cannabis use disorder (CUD).

One concern is that the use of cannabis might lead not only to CUD but to other unhealthy outcomes, such as conduct problems (CP) (e.g. school truancy, stealing), which typically peak during adolescence [10]. It is well documented that substance use and conduct disorder

(i.e. the clinical variant of CP) are interconnected [11,12]. In fact, substance use including cannabis use during adolescence is more strongly associated with conduct disorder than to any other psychiatric disorder [13]. Even in community samples, a longitudinal link between cannabis use and CP has consistently been replicated [14,15]. However, the sequencing of this link is still debated, i.e. it remains unclear whether cannabis use predicts subsequent CP and/or whether CP predicts subsequent cannabis use.

When addressing the sequencing between youth cannabis use and CP, it is essential to consider the peer context. Similar to other substance use and problem behaviors, cannabis use during adolescence is primarily a social behavior [16], and peers can play a critical role in predicting such behaviors [17–21]. Specifically, if adolescents affiliate with peers who use cannabis, this could increase their risk for both cannabis use and CP via social learning processes [16,19,22]. However, most studies on the link between youth cannabis use and CP do not account for peer influences. In this study, we specifically test three ways in which peer cannabis use can explain the developmental associations between youth cannabis use and CP (Fig. 1).

First, youth cannabis use could lead to CP either directly [17,18,23,24] or via exposure to peer cannabis use (e.g. [19] (model A; Fig. 1). Such links have been observed even when accounting for effects of peers and other common confounding factors, such as socio-economic status (SES) and gender [17–19,24]. Consistent with a 'psychopharmacological model', a direct link from cannabis use to CP could occur because cannabis use induces cognitive impairment that can affect judgment and decision-making more broadly [2,25,26]. We are not aware of studies that have found whether or not these cascading chain of events could predict later CUD, however.

As far as we know, only one study [19] has attempted to investigate the developmental cascades contained in model A. In a birth cohort of 935 adolescents who were born in New Zealand in 1977, a series of regression models with two waves of longitudinal data found support for the links in model A. However, that study [19] did not consider whether engaging in CP also potentially influences cannabis use and/or CUD (see model B; Fig. 1). Of note, that study [19] was based on self-reports of property or violent offences, whereas other studies reviewed here investigated other types of conduct problems such as non-violent offences. For the sake of consistency, we refer to all these externalizing behaviors as conduct problems.

In the alternative model B, CP could lead to cannabis use directly or via exposure to peers who use cannabis. Several studies have found a direct link from CP to cannabis use [27–29], even when controlling for common confounding factors [30–32]. These findings suggest the often-overlooked hypothesis that youth with CP might use cannabis as a coping mechanism to deal with disapproval of their CP [25] and perhaps to self-medicate [31,33]. Indeed, recent research revealed that 'feeling stressed out' is the primary reason adolescents use cannabis [16]. Alternatively, an 'under-control-disinhibition' hypothesis suggests that CP in childhood and preadolescence predicts substance use, because CP are manifestations of

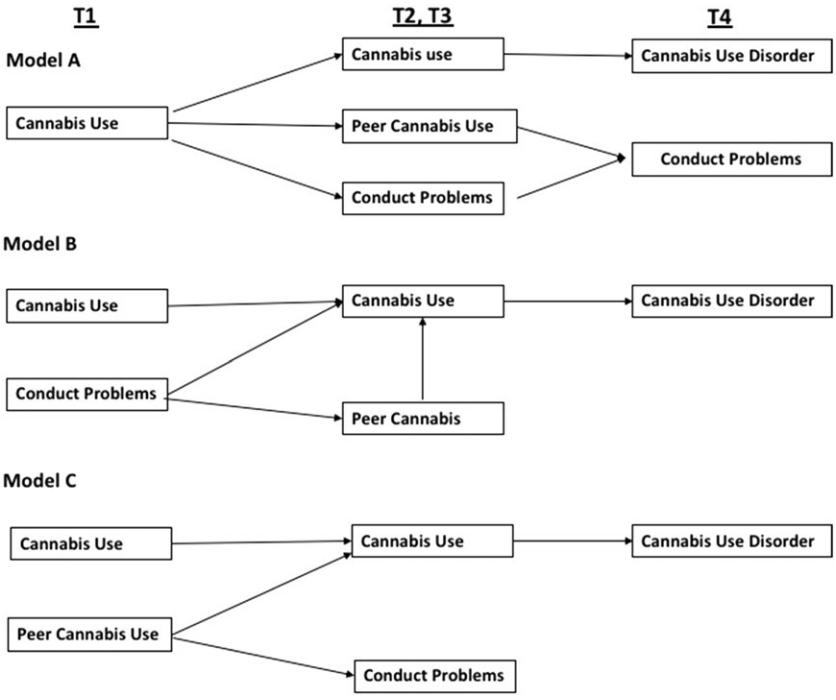

Figure 1 Hypothesized interrelations between cannabis use, conduct problems and peer cannabis use and cannabis use disorder (CUD)

underlying behavioral under-control that lead to problem behaviors such as drug use, and ultimately drug use disorders, as adolescents age [34–36]. Additionally, an indirect pathway from CP to cannabis use could also emerge if youth with CP affiliate with other problem-behavior peers who use cannabis, which could also induce cannabis use that potentially leads to CUD. We could not identify any empirical studies that have explicitly investigated such cascading developmental chains of events. Nevertheless, scholars have speculated that affiliation with delinquent and cannabis using peers could, in turn, cause heightened cannabis use in youth because these peers are likely to reinforce the use of such substances [16,37]. Prior clinical research also suggests that substance use develops later as a symptom of conduct disorder [38].

A combination of models A and B is also possible, which would constitute a bidirectional link between cannabis use and CP leading eventually to CUD, with peer cannabis use operating as an intervening factor. A direct bidirectional relation would be consistent with the 'under-control-disinhibition' hypothesis' [34–36] as well as problem behavior theory (PBT; [38]), which postulates that problem behaviors such as cannabis use and CP are interrelated (suggesting bidirectional links) and predicted by a shared set of psychosocial risk factors in the personality, perceived environment and behavior systems [39]. We could not locate any empirical studies that have explicitly investigated the cascading chain of events in model B. One study investigated bidirectional links between cannabis use and violence and found a bidirectional relationship in a UK young adult male sample [40]. However, this study did not investigate CUD. There is no longitudinal adolescent study with at least four waves that employ sensitive analytical techniques to simultaneously test such bidirectional and cascading effects for cannabis use and CP.

In a third model (C), peer cannabis use could serve as a common confounding factor and fully account for any links between youth cannabis use and CP. Specifically, considering that peer cannabis use could lead to both youth CP and cannabis use, any links between cannabis use and CP would be spurious and no longer exist when peer cannabis use is controlled. This would be consistent with the hypothesis that adolescents experiment with CP and substance use because they are both manifestations of an underlying problem behavior syndrome (such as 'deviant peer affiliation' in the environment system) that predicts both of these behaviors [39,41]. However, specifically in relation to cannabis use and CP, studies show that the cannabis use–CP link does not (completely) vanish when accounting for common confounding factors, such as deviant peer influences [17–19,31].

The current study was designed to investigate which of the above-described models (or a combination thereof) provides the best explanation for the well-documented link between youth cannabis use and CP, the potential role of peer cannabis use in this link and how these three variables could lead to subsequent CUD. To test the various links, we used a longitudinal design with four waves of observations of a US urban sample starting at age 13.5 in 2006 and approximately age 19 at the last wave. A recent and advanced version of the standard cross-lagged panel model (CLPM) was utilized, which is often regarded as a stronger methodology for inferring causality [42], when experimental research is not possible. Whereas the standard CLPM confounds between- and within-person associations, we used the random intercept cross-lagged panel model (RI-CLPM), which isolates changes within-persons as predictors of subsequent within-person changes [42,43]. When it is possible to identify such within-person processes, it provides a more stringent test of potential causal relations between variables.

METHODS

Participants

Participants were enrolled into the Philadelphia Trajectory Study (PTS), a six-wave study that began in 2004 (see [44] and Supporting information). The study was approved by the Institutional Review Board of Children'Hospital of Philadelphia. At wave 1, participants (n = 387) were between ages 10–12 years and were tested annually from 2004 to 2010, and then in 2012 for a final 2-year follow-up [44]. Attrition was 3.9, 5.3, 5.7, 13.4 and 24.8% from waves 2–6, respectively. The current study used the last four waves (i.e. waves 3–6) of PTS data, henceforth referred to as T1 (baseline), T2, T3 and T4, as the levels of cannabis use at earlier waves were too low to permit tests of sequencing. At baseline, valid data were available for 364 participants [49.1% female; age range = 12–14 years; M_{age} = 13.51 (0.95)]. At T4, participants were between ages 18–21 [M_{age} = 18.78 (0.72)]. The sample was diverse regarding race–ethnicity; 56% non-Hispanic white, 26% non-Hispanic black, 9% Hispanic and 9% in other categories [44,45]. Most participants came from low–middle SES families, and the mean Hollingshead Two-Factor Index of Social Status was 47.0 ± 15.8, reversed-scored; higher scores indicate higher SES [45].

Materials

Cannabis use was assessed with two items to assess ever (yes = 1; no = 0) and past 30 days use. Namely, 'Have you ever used marijuana (also called weed, grass, pot, or blunts)?'. If participants answered 'yes', the second question was: 'In the past 30 days, how many times have you used marijuana?' [never (= 0); not in past 30 days (= 1); one to nine times (= 2); and more than 10 times (= 3)].

The two above-described questions were collapsed to create a new variable that was re-coded as never (= 0); not in past 30 days (= 1); 1 one to nine times (= 2); and more than 10 times (= 3). At T4, this frequency question measured days instead of times; namely, 'In the past 30 days, how many days have you used marijuana?'. Here we also used a combined score of ever use and last 30-day use. Answer categories ranged from never (= 0) to 6–9 days (= 3). There were answer categories signifying more than 9 days, but no participants chose those categories.

CUD was measured at T4, using items that tapped abuse and dependence as defined in the Diagnostic and Statistical Manual of Mental Disorders, 4th edition (DSM–IV) [46,47]. Participants who self-reported cannabis use in the past year were asked follow-up questions pertaining to abuse and dependence related to cannabis use that were derived from the National Survey on Drug Use and Health (NSDUH), which has adequate validity and reliability (see [46]). Although these items are based on the DSM-IV criteria, there were also items assessing craving and withdrawal/dependence, which are part of the DSM-5 criteria. We matched the DSM-5 criteria to these NSDUH questions to create criterion scores for CUD, which no longer distinguish between abuse and dependence and simply categorize people in terms of varying levels of CUD. The following categories were used: 0 = no symptoms, 1 = one symptom; and 2 = two to three symptoms to three to four or more symptoms [48]. DSM-5 classifies a mild disorder as meeting two to three criteria. We used these continuous criterion scores, signifying number of criteria met, which thus represents a continuum of severity of CUD. A total of 14.5% of the participants met criteria for a mild CUD.

CP in the past 6 months were measured with the conduct problems scale of the Youth Self Report (YSR [49]; see also [48]) at T1–T3. The 15 items on this scale overlap with the symptoms of conduct disorder as described in the DSM–IV [47,48]. The answer categories are: not true (= 0), somewhat or sometimes true (= 1) and very true or often true (= 2). At T4, the participants were aged 18 or older, thus the DSM-oriented 'antisocial personality problems' scale on the Adult Self-Report (ASR [48]) was used instead, which is the adult equivalent of the YSR. The 20 items on the antisocial personality problems scale tap the symptoms of antisocial personality disorder as described in the DSM. Reliabilities for these DSM-oriented scales from the YSR and ASR [50] are high (Cronbach alphas > 0.70). We used mean scores for both scales.

Peer cannabis use at T1–T4 was measured with the question: 'Of your friends and the people your age that you spend time with, how many smoke marijuana?'. Answer categories were: 1 = none; 2 = a few; 3 = about half; 4 = most; and 5 = all.

Statistical analyses

The RI-CLPM [42] was specified in Mplus 7.3 [51]. Similar to the standard CLPM, the RI-CLPM included stability paths for cannabis use, CP and peer cannabis use, and cross-lagged paths between these variables. Additionally, concurrent associations between these variables were also included in the models. We further controlled for gender, race–ethnicity and SES by regressing cannabis use, CP and peer cannabis use on these variables at each wave. However, unlike the CLPM, in the RI-CLPM the variance between people (i.e. stable time-invariant traits) was parceled out from the variance of the observed scores, in order to capture the within-person variance (i.e. fluctuations over time, which technically refers to deviations of individuals from their own intercepts) (for technical details see [43]). Furthermore, we extended this model by examining whether within-person increases in T1 cannabis use, peer cannabis use and CP predict T4 CUD (it was not possible to control for the random intercept for this variable). We followed-up with mediation analyses using bootstrapped standard errors to test the significance of the mediational paths from T1 cannabis use, T1 peer cannabis use and T1 CP to T4 CUD [52]. A robust maximum likelihood (MLR) estimator was used to account for non-normality [53]. We used the $P < 0.05$ level in all tests of model parameters. Finally, we verified that missing data patterns were unrelated to study variables, and thus we used full information maximum likelihood (FIML) to estimate missing data [51].

RESULTS

Descriptive statistics

Table 1 shows that cannabis use and peer cannabis use increased from T1 to T4, whereas CP declined after T3.

Table 1 Descriptive statistics of study variables per year.

	Minimum	*Maximum*	*Mean (SD)*
T1 Cannabis use	0	3	0.10 (0.41)
T2 Cannabis use	0	3	0.17 (0.54)
T3 Cannabis use	0	3	0.44 (0.86)
T4 Cannabis use	0	4	10.03 (10.16)
T4 CUD	0	3	0.44 (89)
T1 Conduct problems	0	1.40	0.22 (0.23)
T2 Conduct problems	0	1.00	0.24 (0.21)
T3 Conduct problems	0	1.27	0.27 (0.24)
T4 Conduct problems	0	1	0.22 (0.19)
T1 Peer cannabis use	1	4	10.26 (0.59)
T2 Peer cannabis use	1	5	10.62 (0.94)
T3 Peer cannabis use	1	5	20.13 (10.13)
T4 Peer cannabis use	1	5	20.51 (10.15)

CUD = conduct use disorder; SD = standard deviation.

Table 2 Concurrent and longitudinal correlations between the study variables.

	1	*2*	*3*	*4*	*5*	*6*	*7*	*8*	*9*	*10*	*11*	*12*	*13*
1. T1 Cannabis use	–												
2. T2 Cannabis use	0.52**	–											
3. T3 Cannabis use	0.32**	0.52**	–										
4. T4 Cannabis use	0.15**	0.22**	0.44**	–									
5. T4 CUD	0.15**	0.26**	0.38**	0.70**	–								
6. T1 Conduct problems	0.29**	0.29**	0.33**	0.21**	0.21**	–							
7. T2 Conduct problems	0.12*	0.29**	0.40**	0.28**	0.23**	0.68**	–						
8. T3 Conduct problems	0.09	0.22**	0.46**	0.38**	0.31**	0.64**	0.69**	–					
9. T4 Conduct problems	0.12*	0.18**	0.29**	0.38**	0.43**	0.40**	0.43**	0.57**	–				
10. T1 Peer cannabis use	0.61**	0.49**	0.32**	0.16**	0.21**	0.44**	0.29**	0.27**	0.22**	–			
11. T2 Peer cannabis use	0.34**	0.53**	0.51**	0.26**	0.22**	0.41**	0.45**	0.36**	0.13*	0.57**	–		
12. T3 Peer cannabis use	0.24**	0.34**	0.61**	0.43**	0.35**	0.36**	0.38**	0.51**	0.26**	0.37**	0.64**	–	
13. T4 Peer cannabis use	0.25**	0.21**	0.32**	0.54**	0.42**	0.27**	0.30**	0.36**	0.39**	0.21**	0.22**	0.44**	

*$P < 0.05$; **$P < 0.01$.

Table 2 shows that these behaviors were positively concurrently and longitudinally correlated at all waves, with one exception (T1 cannabis use and T3 CP).

RI-CLPM

Direct links

The results (Fig. 2 and Table 3) showed significant cross-lagged paths from CP to cannabis use except from T1 to T2. Additionally, significant paths from peer cannabis use to cannabis use existed over the four waves. As for CUD, links from T4 cannabis use and CP to T4 CUD were also observed. No significant paths from cannabis use to CP or peer cannabis use were observed.

CP also served as a predictor of peer cannabis use from T1 to T2, whereas peer cannabis use predicted cannabis use at all waves. All the cross-lagged paths were significant above and beyond the significant within-person correlations and correlated residuals (Table 3).

Indirect links

Significant mediational cascading links from T1 CP (via peer and personal cannabis use) to T4 CUD also emerged (total indirect: B = 0.57, 95% CI = 0.32,0.93; β = 0.12, 95% CI = 0.07, 0.20; see Table 4). Additionally, significant mediational cascading links from T1 peer cannabis use (via cannabis use) to T4 CUD existed (total indirect: B = 0.13, 95% CI = 0.07, 0.21; β = 0.08, 95% CI = 0.04, 0.14). The presence of both significant overall and specific indirect links provide strong evidence of mediation. Similar results were found for the standard CLPM (see Supporting information).

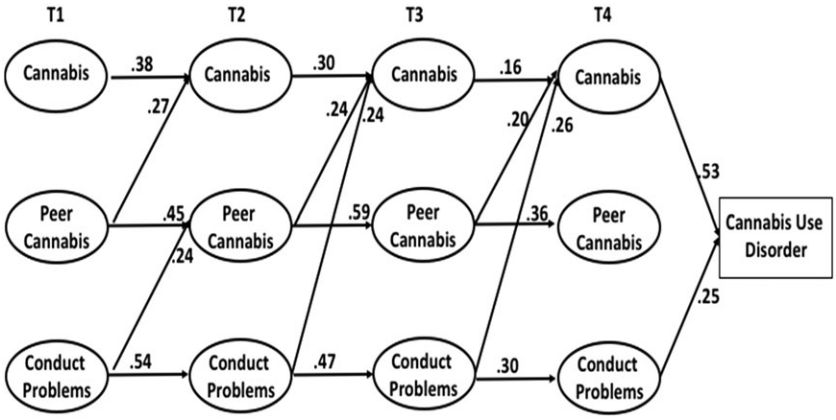

Figure 2 Path model of the standardized significant paths of the random intercept-cross-lagged panel model (RI-CLPM) (χ^2 = 43.59 [35], P = 0.151). Non-significant paths, concurrent associations and control variables (gender and socio-economic status and race–ethnicity) are not depicted. Model fit was good according to: confirmatory fit index (CFI) (0.995), root mean square error of approximation (RMSEA) (0.026) and standardized root mean square residual (SRMR) (0.032)

Table 3 Random intercept-cross-lagged panel model (RI-CLPM): links between cannabis use, peer cannabis use, conduct problems, and cannabis use disorder (CUD).

Parameters	*B*	*SE*	*P*	*95% CI of B*
Cross-lagged paths				
T1 Pcannabis ➔ T2 Conduct	0.02	0.02	0.277	−0.016, 0.055
T1 Cannabis ➔ T2 Conduct	−0.05	0.04	0.173	−0.130, 0.023
T2 Pcannabis ➔ T3 Conduct	0.04	0.02	0.051	0.000, 0.071
T2 Cannabis ➔ T3 Conduct	−0.01	0.03	0.863	−0.072, 0.060
T3 Pcannabis ➔ T4 Conduct	−0.00	0.01	0.832	−0.029, 0.023
T3 Cannabis ➔ T4 Conduct	0.04	0.02	0.054	−0.001, 0.081
T1 Pcannabis ➔ T2 Cannabis	0.26	0.09	0.002	0.092, 0.429
T1 Conduct ➔ T2 Cannabis	0.40	0.25	0.115	−0.097, 0.897
T2 Pcannabis ➔ T3 Cannabis	0.21	0.07	0.004	0.066, 0.348
T2 Conduct ➔ T3 Cannabis	10.16	0.32	< 0.001	0.536, 1.780
T3 Pcannabis ➔ T4 Cannabis	0.20	0.07	0.006	0.058, 0.346
T3 Conduct ➔ T4 Cannabis	10.45	0.57	0.011	0.327, 2.557
T1 Cannabis ➔ T2 Pcannabis	0.16	0.18	0.371	−0.195, 0.523
T1 Conduct ➔ T2 Pcannabis	10.22	0.34	< 0.001	0.559, 1.871
T2 Cannabis ➔ T3 Pcannabis	−0.05	0.16	0.778	−0.356, 0.266
T2 Conduct ➔ T3 Pcannabis	0.81	0.42	0.052	−0.007, 1.629
T3 Cannabis ➔ T4 Pcannabis	0.03	0.11	0.802	−0.185, 0.239
T3 Conduct ➔ T4 Pcannabis	0.76	0.44	0.080	−0.092, 1.619
Final links to T4 CUD				
T4 Conduct➔T4 CUD	10.43	0.42	0.001	0.603, 2.250
T4 Cannabis➔T4 CUD	0.42	0.06	< 0.001	0.306, 0.535
T4 PCannabis➔T4 CUD	0.04	0.05	0.411	−0.054, 0.131
T1 Correlations				
T1 Conduct - T1 Cannabis	0.02	0.01	0.003	0.002, 0.044
T1 Conduct - T1 Pcannabis	0.04	0.01	< 0.001	0.020, 0.063
T1 Cannabis - T1 Pcannabis	0.13	0.05	0.005	0.040, 0.227
Correlated residuals				
T2 Conduct - T2 Cannabis	0.02	0.01	0.019	0.002, 0.027
T2 Conduct - T2 Pcannabis	0.04	0.01	< 0.001	0.020, 0.057
T2 Cannabis - T2 Pcannabis	0.10	0.03	0.002	0.035, 0.160
T3 Conduct - T3 Cannabis	0.04	0.01	< 0.001	0.021, 0.061
T3 Conduct - T3 Pcannabis	0.06	0.01	< 0.001	0.030, 0.082
T3 Cannabis - T3 Pcannabis	0.24	0.05	< 0.001	0.156, 0.332
T4 Conduct - T4 Cannabis	0.04	0.01	< 0.001	0.022, 0.064
T4 Conduct - T4 Pcannabis	0.04	0.01	< 0.001	0.018, 0.057
T4 Cannabis - T4 Pcannabis	0.37	0.06	< 0.001	0.245, 0.486

Cannabis = cannabis use; Pcannabis = peer cannabis use; Conduct = conduct problems; CI = confidence interval.

Table 4 Significant indirect mediational links from T1 conduct problems and T1 peer cannabis use to T4 cannabis use disorder (CUD).

Parameters	*B*	*95% CI*
T1 CP- > T2 CP- > T3 CP- > T4 Cannabis use- > T4 CUD	0.166	0.036, 0.359
T1 CP–>T2 PCannabis use- > T3 PCannabis use- > T4 Cannabis use- > T4 CUD	0.071	0.018, 0.168
T1 PCannabis–>T2 PCannabis use- > T3 PCannabis use- > T4 Cannabis use- > T4 CUD	0.044	0.010, 0.095

Pcannabis use = peer cannabis use; CI = confidence interval.

DISCUSSION

The present findings showed that, consistent with model B and with previous studies [27–29], CP predicted cannabis use but not vice versa, particularly during mid–late adolescence. Thus, unlike other studies that did not use RI-CLPM or a similar methodology, we were able to demonstrate for the first time that increases in

CP precede increases in cannabis use within individuals. Specifically, youth whose CP change at one time-point are likely to engage in a corresponding change in cannabis use at follow-up, regardless of the level of those problem behaviors at the prior time-point. Finally, we also found that increases in T4 CP were related to T4 CUD. These direct links from CP to cannabis use and CUD were found even while accounting for effects of peer cannabis use, gender, race–ethnicity and SES. The current findings showing that CP robustly predicts youth cannabis use support some previous studies, although those studies did not examine within-person links or control for peer effects (e.g. 21,25).

Although both CP and peer cannabis use directly predicted youth cannabis use, we further found that CP could also predict cannabis use via affiliation with cannabis-using peers. For example, mediation analyses showed that increasing levels of T1 CP predicted increasing levels of T2 peer cannabis use, and peer cannabis use at T3 subsequently predicted increasing levels of youth cannabis use at T4, which ultimately predicted T4 CUD. These cascading links could be interpreted as CP leading adolescents to associate more with cannabis-using peers (or that their peers began to use cannabis) which, in turn, led to more cannabis use by adolescents and ultimately to CUD during emerging adulthood. Similar cascading links have been hypothesized in the past [37]. However, as far as we know, the current study is the first to empirically investigate these cascading links at a within-person-level in a design such as the RI-CLPM, which more sensitively determines the temporality of these longitudinal associations, and can test the mediational links. Similarly, we also found that increased affiliation with cannabis-using peers at T1 predicted CUD via increased adolescent cannabis use.

We found no evidence that cannabis use predicted either CP or peer cannabis use as suggested by model A or that peer cannabis use predicted conduct problems as suggested by model C. Although some prior research has found evidence for models A and C (e.g. [19,24]), the data in those studies were collected decades ago and may not accurately reflect more current adolescent cannabis use patterns and their relations with CP. Moreover, the current study controlled for prior levels of CP, cannabis use and cannabis using peers, in addition to controlling for other stable between-person differences. Thus, the stringency of our model might account for why current results are not consistent with those of past studies. Nevertheless, our finding that CP predict cannabis use in adolescence concurs with the results of numerous other studies [27–32,54]. Our findings also clearly show that peer cannabis use cannot alone account for relations between CP and cannabis use. Thus, taken together, the results are most consistent with model B.

It has been put forward that youth with CP use cannabis to cope [25] and self-medicate [33], and this could perhaps explain the direct link we found from CP to cannabis use and CUD. Alternatively, other substance use literature suggests that CP lead to substance use and ultimately substance use disorder (and not vice versa), because CP are underlying components of behavioral under-control [34–36]. This explanation is consistent with findings suggesting that such under-control is a marker for liabilities to drug addiction [34–36].

The current findings could be meaningful for current policy-related debates on cannabis, and accordingly they could also have prevention implications. Namely, our findings imply that while a potential increase in cannabis use [4,5] might not increase CP in youth, it appears that particularly youth who show heightened levels of CP and/or affiliate with cannabis-using peers might end up using more cannabis which, in turn, predicts CUD. However, importantly, youth with increasing CP are susceptible to more cannabis use and CUD regardless of whether or not their friends are increasingly using it. Additionally, these findings might also be relevant for clinical practice, as they suggest that in cases of comorbidity between cannabis use and CP [13] CP could receive treatment priority as it predicts cannabis use, not vice versa. However, when determining treatment priority, the level of cannabis use should also be taken into account, as high levels of cannabis use could possibly diminish treatment effectiveness. Finally, with regard to both policy-making and clinical practice, the current results demonstrate that although there appears to be a direct link from CP to cannabis use, adolescent'peer context should not be neglected, as this could undermine or hamper the effects of policies and clinical interventions.

Despite the numerous strengths inherent in the current study, possible limitations of self-reports should be kept in mind. Future studies could also examine these pathways in relation to other drugs and parental influences.

CONCLUSIONS

In sum, at a within-person level, increases in CP predicted increases in cannabis use and ultimately CUD, partially via increases in affiliation with cannabis-using peers. As cannabis use becomes more normative, access to the drug will inevitably increase. Our results suggest that this will increase risks for CUD, especially for youth with CP who are at higher risk for cannabis use and affiliation with cannabis-using peers. If youth with CP use unprescribed cannabis to cope with their condition, then healthier alternative coping strategies and support should be made available.

Declaration of interests

None.

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Supporting Information

Additional supporting information may be found online in the Supporting Information section at the end of the article.

Table S1 Significant mediational Links from T1 peer cannabis use and T1 conduct problems to T4 Cannabis use disorder.

Figure S1 A simplified path model of the standardized significant paths of the standard Cross-lagged panel Model (CLPM). Concurrent associations are not depicted, but those results are embedded in the text. Control variables (Gender & SES & Race-Ethnicity) are not depicted. Chi-square = 56.54 (41), P = .054; CFI (.99), RMSEA (.03) and SRMR (.02).

