Age of Cannabis Use Onset and Adult Drug Abuse Symptoms: A Prospective Study of Common Risk Factors and Indirect Effects

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Abstract

The present study examined 1) whether the associations between cannabis use (CU) age of onset and drug abuse by 28 y remain when controlling for risk factors in childhood, adolescence and early adulthood; and 2) the developmental pathways from early risk factors to drug abuse problems. Participants from a longitudinal sample of boys of low socioeconomic status ($N = 1,030$) were followed from 6 to 28 y. We examined the self-reported CU onset between the ages of 13 and 17 y and drug abuse symptoms by 28 y. The odds of developing any drug abuse symptoms by 28 y were reduced by 31% for each year of delayed CU onset ($OR = 0.69$). Cannabis, alcohol and other drug frequency at 17 y mediated this association. Still, even when taking that frequency of use into account, adolescents who started using cannabis before 15 y were at a higher risk of developing drug abuse symptoms by age 28 y. Significant indirect effects were found from early adolescent delinquency and affiliation with deviant friends to drug abuse symptoms at 28 y through CU age of onset and substance use frequency at 17 y. The results suggest more clearly than before that prevention programs should aim at delaying CU onset to prevent or reduce drug abuse in adulthood. Furthermore, prevention programs targeting delinquency and/or affiliation with deviant friends in childhood or early adolescence could indirectly reduce substance abuse in adulthood without addressing substance use specifically.

Abrégé

Objectif : La présente étude a examiné (1) si les associations entre l'âge du début d'utilisation du cannabis (UC) et l'abus de drogue à 28 ans demeurent après avoir contrôlé pour des facteurs de risque à l'enfance, l'adolescence et l'âge adulte; et (2) les trajectoires développementales des facteurs de risque précoces aux problèmes d'abus de drogue.

Méthode : Les participants d'un échantillon longitudinal de garçons de faible statut socio-économique (SSE) ($N = 1\,030$) ont été suivis de 6 à 28 ans. Le début d'UC auto-rapporté entre 13 et 17 ans et les symptômes d'abus de drogue à 28 ans ont été examinés.

Résultats : Les probabilités de développer des symptômes d'abus de drogue à 28 ans étaient réduites de 31% pour chaque année de délai du début d'UC ($RC = 0,69$). La fréquence d'utilisation du cannabis, de l'alcool et d'autres drogues à 17 ans agissaient comme médiateurs de cette association. Cependant, même en tenant compte de cette fréquence d'utilisation, les

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adolescents qui ont commencé à utiliser le cannabis avant 15 ans étaient à risque plus élevé de développer des symptômes d'abus de drogue à 28 ans. Des effets indirects significatifs ont été observés, allant de la délinquance, l'impulsivité et l'affiliation avec des pairs déviants au début de l'adolescence aux symptômes d'abus de drogue à 28 ans, au travers l'âge de début d'UC et la fréquence d'utilisation de substances à 17 ans.

Conclusions : Les résultats suggèrent plus clairement qu'auparavant que les programmes de prévention devraient viser à retarder l'âge de début d'UC afin de prévenir ou de réduire l'abus de drogue à l'âge adulte. En outre, les programmes de prévention qui ciblent la délinquance et/ou l'affiliation avec des pairs déviants à l'enfance ou au début de l'adolescence pourraient réduire indirectement l'abus de substances à l'âge adulte sans aborder spécifiquement l'utilisation de substances.

Keywords

cannabis, marijuana, early onset, substance abuse disorder

Cannabis is the most widely used substance worldwide after alcohol and tobacco, including among Canadian adolescents.¹⁻³ Indeed, 10% of Canadian adolescents consumed cannabis in Grade 8, a rate that increases to 29% by Grade 12.³ This early-onset cannabis use (CU) may be problematic, as it is associated with several adverse outcomes, including other drug use and substance use disorders.⁴ However, most studies to date on the association between adolescent CU and later substance use defined early-onset as any CU in adolescence without examining age of onset specifically. Whereas 2 studies suggest that an earlier CU age of onset is associated with higher risk of cannabis abuse and dependence as well as alcohol use problems, differences between earlier and later onset were not formally tested^{5,6} and one of these studies relied partly on retrospective data,⁵ which is less reliable than prospective data.⁷ Two more studies using the same sample and comparing CU onset before and after 16 y found that CU onset before 16 y was positively associated with cannabis dependence at 20 y⁸ and CU frequency and dependence at 24 y.⁹ However, using dichotomous variables with a cut-off at 16 y may mask significant information about age of onset across adolescence.

Studies on the role of CU on other outcomes and on alcohol use age of onset highlight the importance of examining age of onset throughout adolescence instead of before and after an arbitrarily chosen cut-off age. Indeed, studies found an earlier CU age of onset during adolescence was associated with age at onset of psychosis,^{10,11} decline in verbal IQ and tasks tapping trial and error learning and reward processing, and lower rates of high-school graduation.¹² Studies on alcohol use age of onset also report that an earlier alcohol use age of onset is associated with higher odds of later alcohol and drug dependence.¹³⁻¹⁵ Although these studies suggest that examining age of onset across adolescence may be particularly important to fully understand the association between adolescent substance use and future outcomes, no study so far has formally examined the association between CU age of onset and future substance use disorders. Such information is needed to improve our understanding of the pathogenesis of substance use disorders. Indeed, more complete information on the significance of an earlier onset of CU will better guide prevention and intervention programs.

It is also important to look at explanatory variables and mechanisms linking CU onset with later substance use problems, especially considering that few cannabis users become dependent and develop substance use problems.^{16,17} This can help determine whether the association between CU age of onset and later substance use problems can be explained by common early risk factors for both early onset CU and later drug abuse, or other risk factors for substance use disorders.⁴ One study has found that the association between CU onset and cannabis dependence was explained by antisocial behaviours and smoking.⁸ However, other studies found that the association between early CU onset and later substance use was not explained by school grades, antisocial behaviours, parent education,⁶ parental separation, parent smoking,⁹ or alcohol, cigarette, and illicit drug use.^{5,6} Although these previous studies collectively included numerous covariates, each study controlled only for a limited number. Furthermore, studies did not include other important adolescent covariates of substance use, such as parental supervision,¹⁸ impulsivity,¹⁹ and affiliation with deviant peers.²⁰ Besides simply entering these variables as covariates, understanding their role in developmental pathways from early risk factors to later substance use could help identify prevention and intervention targets.

The Present Study

This study aims to investigate the associations between CU age of onset and drug abuse symptoms by 28 y by examining: 1) whether the associations between CU age of onset and drug abuse symptoms by 28 y remain when taking into account (a) early common risk factors (6 to 13 y), (b) cannabis and other substance use frequency (17 y), and (c) explanatory variables in early adulthood (20 to 23 y); and 2) the developmental pathways (indirect effects) from early common risk factors to drug abuse symptoms by 28 y through CU age of onset, substance use frequency during adolescence, and explanatory variables in early adulthood.

Method

Participants

Participants were from the Montreal Longitudinal and Experimental Study of boys of low socioeconomic status

Table 1. Sample Characteristics and Substance Use Prevalence.

Sample Characteristics ^a	
Mother age at first child	23.3 (4.1)
Father age at first child	26.4 (5.1)
Mother occupational prestige ^b	38.3 (12.1)
Father occupational prestige ^b	39.4 (12.8)
Family adversity ^c	0.33 (0.25)
Intact family structure, % (n)	68.5 (683)
Substance use prevalence	
Any CU during adolescence, % (n)	58.3 (484)
Onset at 13 y	3.7 (31)
Onset at 14 y	8.9 (74)
Onset at 15 y	16.9 (140)
Onset at 16 y	16.4 (136)
Onset at 17 y	12.4 (103)
Cigarette use at 17 y, % (n)	47.6 (376)
Alcohol use at 17 y, % (n)	79.7 (628)
Other drug use at 17 y, % (n)	23.7 (186)
Any drug abuse symptoms by 28 years, % (n)	36.9 (198)
Drug abuse symptoms by age of onset of CU, % (n)	
Onset at 13 y	76.9 (10)
Onset at 14 y	65.0 (26)
Onset at 15 y	44.3 (35)
Onset at 16 y	41.4 (29)
Onset at 17 y	47.8 (32)
No CU during adolescence	23.9 (52)

CU, cannabis use

^aData are mean (SD) unless indicated otherwise^bOccupational prestige based on Canadian norms; scale range 17.8 to 101.7; population average 42.7, SD 13.3.^cFamily adversity range is 0 -1.

(MLES). In 1984, 1,037 French-speaking boys (mean age [SD], 6.1 [0.31] y) were recruited from schools in low socioeconomic neighbourhoods of Montreal, Canada (for more information, see Tremblay et al.²¹). Sex, ethnicity (mostly Caucasian boys), and socioeconomic status were homogeneous because of the selection procedure. Sample characteristics are presented in Table 1.

Boys were assessed annually from ages 12 to 17 y, and at ages 20 and 28 y; 952 boys participated at least once during the adolescent period and 536 participated at 28 years. The total sample for this study consists of 1,030 boys with data on any outcome variable between 17 and 28 y; i.e., CU age of onset, high school graduation, substance use at 17 y, delinquency at 20 y and drug abuse symptoms at 28 y. The University of Montreal and CHU Ste-Justine Institutional Review Boards approved this project. Written informed consent from parents and teachers, as well as verbal assent from the boys, were obtained during adolescence. In adulthood, written consent was obtained from all participants.

Measures

Drug abuse symptoms. At 28 y, participants completed a domain-specific scale adapted from the Problem Severity scale of the Personal Experience Screening Questionnaire,²² which measures problems associated with the use of any

drug (excluding alcohol and tobacco) over the past 12 mo. The scale includes 7 items referring to the absence (scored 0) and presence (scored 1) of problems over the past 12 mo. These items were used to compute a dichotomous variable representing the presence or absence of drug abuse symptoms corresponding to DSM-IV criteria (0 = abuse symptoms absent; 1 = at least one abuse symptom present).

CU age of onset. Each year from 13 to 17 y, adolescents were asked whether they had consumed cannabis in the last 12 mo. These scores were used to compute an age of onset score, representing the first year the adolescents answered that they consumed cannabis. Computed scores ranged from 1 (CU onset by 13 y) to 6 (no CU onset by 17 y).

Early common risk factors. Paternal alcohol use problems were assessed when the boys were 12 y through an interview with the mother and a subset of fathers. Trained interviewers conducted the interview through a telephone survey using the Short Michigan Alcoholism Screening Test (SMAST).²³ The SMAST allowed mothers to rate fathers reliably, as supported by a study of a subset of 160 fathers showing a high concordance between mother-reported and father-reported alcohol use problems in this sample.²⁴

Family adversity at 6 y was assessed using 6 variables reflecting the quality of the family environment: 1) mothers' occupational status, 2) fathers' occupational status, 3) mothers' educational level (number of years in school), 4) father's educational level, 5) mothers' age at the birth of their first child, and 6) family structure (intact or non-intact).

Academic achievement at 12 y was obtained from elementary school teachers, who reported on students' achievement in French (first language) and mathematics. Scores ranged from 1 (academic failure) to 5 (excellent academic performance).

Verbal IQ at 13 y was assessed using the Sentence Completion Test.²⁵

Impulsivity at 13 y was assessed through self-reports with a short form of the Eysenck Impulsiveness-Venturesomeness-Empathy scale, which included the 5 items ($\alpha = 0.69$) with the highest factor loadings on the original scale.^{26,27}

Affiliation with deviant friends at 13 y was obtained by asking boys whether they were part of a group or a gang that carried out reprehensible acts.

Delinquency at 12 y was measured using 17 items from the Self-Reported Delinquency Questionnaire (SRDQ)²⁸ assessing involvement in delinquent behaviours over the last 12 mo by measuring physical violence (5 items, e.g., used a weapon during a fight), theft (7 items, e.g., stole \$100 or more), and vandalism (5 items, e.g., vandalised a car). Each question was rated on a 4-point scale (0 = never; 3 = very often). Cronbach's alpha for this scale was 0.79.

Parental supervision at 12 years was measured using 2 items ($\alpha = 0.73$) asking 'Do your parents know where you are when you are not at home?' and 'Do your parents know

Table 2. Results from Logistic Regressions Predicting Drug Abuse Symptoms by 28 Years.

	Model 1			Model 2			Model 3			Model 4		
	B	OR	95%CI	B	OR	95%CI	B	OR	95%CI	B	OR	95%CI
CU age onset	−0.37 ^c	0.69	0.61–0.78	−0.37 ^c	0.69	0.60–0.79	−0.06	0.95	0.74–1.14	−0.04	0.97	0.80–1.17
Father AUP				0.25	1.28	0.69–2.36	0.19	1.21	0.63–2.30	0.19	1.21	0.62–2.33
Family adversity 6 y				0.10	1.10	0.47–2.58	0.05	1.05	0.42–2.60	−0.05	0.95	0.37–2.45
Academic achievement 12 y				−0.11	0.89	0.73–1.09	−0.05	0.95	0.77–1.17	−0.01	0.99	0.80–1.23
Verbal IQ 13 y				0.10	1.10	0.98–1.23	0.10	1.11	0.98–1.25	0.12	1.12	0.99–1.28
Impulsivity 13 y				−0.04	0.96	0.84–1.10	−0.04	0.96	0.84–1.10	−0.04	0.96	0.84–1.11
Deviant friends 13 y				−0.11	0.90	0.52–1.57	−0.12	0.89	0.50–1.57	−0.23	0.80	0.43–1.46
Delinquency 12 y				−0.01	0.99	0.93–1.04	0.00	1.00	0.95–1.06	−0.01	0.99	0.93–1.05
Parental supervision 12 y				−0.08	0.92	0.80–1.10	−0.07	0.93	0.80–1.08	−0.06	0.94	0.81–1.10
Cigarette frequency 17 y							0.06	1.07	0.98–1.16	0.06	1.06	0.97–1.16
Alcohol frequency 17 y							−0.11	0.90	0.79–1.01	−0.14 ^a	0.87	0.77–0.99
Cannabis frequency 17 y							0.28 ^c	1.32	1.14–1.53	0.30 ^c	1.35	1.16–1.59
Other drug frequency 17 y							0.13	1.14	0.94–1.39	0.11	1.12	0.91–1.37
High School graduation										−0.22	0.80	0.47–1.35
Delinquency 20 y										0.13	1.14	0.99–1.31

AUP, alcohol use problems; CU, cannabis use; y, years.

^a $P < 0.05$, ^b $P < 0.01$, ^c $P < 0.001$.

who you are with, when you are not at home?’ rated on a scale ranging from 1 (never) to 4 (always).

Substance use frequency at 17 y. Cigarette, alcohol, cannabis and other drug use frequency at 17 y was obtained by asking adolescents whether they had smoked cigarettes, drunk alcohol, consumed cannabis and consumed drugs other than cannabis (hallucinogens, cocaine, amphetamines, barbiturates, tranquillisers, heroin, inhalants, and other drugs) in the last 12 mo (1 = never, 7 = 40 or more times).

Explanatory variables in early adulthood. Data on high school graduation was obtained when the boys were 23 y old from the Education Department’s official records. Because the probability of completing secondary school dramatically decreases after 20 to 24 y,²⁹ graduation status at 23 y is likely to be definitive for most participants.

Finally, delinquency at 20 y was measured using the Self-Reported Delinquency Questionnaire (see early common risk factors section). Cronbach’s alpha for this scale at 20 y was 0.63.

Data Analyses

Analyses were carried out using Mplus 7.0.³⁰ The estimator used was maximum likelihood with robust standard error estimation (MLR), except for models testing indirect effects, which used weighted least squares means and variance adjusted estimation (WLSMV). First, logistic regressions were conducted to test the association between CU age of onset and drug abuse symptoms, and whether other variables reduced this association. Regressions were conducted in 4 steps: a first model with CU age of onset only, a second model with early common risk factors added to the model,

a third model including substance use frequency, and a final model including early adulthood explanatory variables. Path models were conducted to examine indirect effects. Indirect effects were tested in Mplus with a 1,000 samples bias-corrected bootstrap, which reduces bias and yields a more precise type I error.³¹

Results

Descriptive Statistics

Table 1 presents substance use prevalence for the sample. The prevalence of participants with drug abuse symptoms by 28 y decreased with increasing age of CU onset, dropping from 77% in boys who initiated CU at 13 y or before to 48% in boys who initiated CU at 17 y, and to 24% in boys who did not use CU during adolescence. A full correlation table and descriptive statistics for all study variables is provided as Supplementary Material (Table S1). The number of years cannabis was consumed and the rates of other substance use as a function of CU age of onset can also be found in Supplementary Tables S2 and S3.

Does the Association Between CU Age of Onset and Drug Abuse Symptoms by 28 Y Remain When Accounting for Other Risk Factors?

The results from the logistic regressions predicting the presence of any drug abuse symptoms are presented in Table 2. Without considering any covariates, the odds of developing any drug abuse symptoms by 28 y were reduced by 31% for each year of delayed CU onset (model 1; $R^2 = 0.09$). Early common risk factors (model 2; $\Delta R^2 = 0.02$) were not directly associated with drug abuse symptoms by 28 y, and their inclusion did not affect the association between CU age

Table 3. Odds of Drug Abuse Symptoms by 28 Years for Each Year of Delayed Onset of Cannabis Use^a.

Onset by	Drug Abuse Symptoms	
	OR	95% CI
13 y	3.44	1.05–11.22
14 y	1.97	1.08–3.61
15 y	1.10	0.68–1.80
16 y	0.87	0.51–1.49
17 y	1.14	0.64–2.03

^aComparison group for each analysis is later cannabis use. Analyses control for significant covariates from model 4: alcohol use frequency and cannabis use frequency at 17 y.

of onset and later drug abuse symptoms. Once substance use frequency at 17 y was added to the model (model 3; $\Delta R^2 = 0.09$), the contribution of CU age of onset was reduced to non-significance. Cannabis use frequency at 17 y was associated with 1.32-times greater odds of reporting any drug abuse symptoms by 28 y. When early adulthood explanatory variables were added to the model (model 4; $\Delta R^2 = 0.03$), only alcohol use frequency at 17 y reached significance (OR = 0.87).

Odds of Having Any Drug Abuse Symptoms by Age of CU Onset

To examine whether a specific age of CU onset was associated with greater odds of having any drug abuse symptoms, the odds of developing any drug abuse symptoms by age 28 were examined by age of CU onset, with each age compared to later CU (see Table 3). The odds of developing any drug abuse symptoms by age 28 y were non-significant if CU had its onset between 15 and 17 y but were significant and almost doubled each year if onset was before 15 y (OR = 1.97, onset by 14; OR = 3.44, onset by 13). Indeed, rates of having any drug abuse symptoms by 28 y were 68% in cannabis users who started early (at or before 14 y) compared to 44% in users who started later ($\chi^2 [1, N = 269] = 9.39, P = 0.002$).

To clarify how CU frequency contributed to the increased risk of developing any drug abuse symptoms in adolescent cannabis users, a cross-tabulation across different patterns of cannabis use frequency was conducted. Within adolescent cannabis users, rates of having any drug abuse symptoms by 28 y were significantly higher in cannabis users who consumed cannabis frequently at 17 y (20 or more times in the last year) than in cannabis users who consumed less frequently (72% v. 40%; $\chi^2 (1, N = 227) = 22.29, P < 0.001$).

Is CU Age of Onset Part of an Indirect Pathway from Early Risk Factors to Drug Abuse Symptoms?

When all variables were combined in a path model (see Figure 1), earlier age of CU onset was significantly predicted by higher verbal IQ and delinquency at 12 y, impulsivity and

affiliation with deviant peers at 13 y, and having a father with alcohol use problems. In turn, earlier CU age of onset was associated with a lower rate of high school graduation, a higher frequency of cigarette, alcohol, cannabis and other drug use at 17 y, and delinquency at 20 y. Finally, a higher frequency of cannabis and other drug use at 17 y as well as a lower frequency of alcohol use were associated with having any drug abuse symptoms by 28 y.

Several significant indirect effects were also found (see Figure 1). Significant indirect paths from CU age of onset through CU frequency and other drug use frequency at 17 y positively predicted having any drug abuse symptoms by 28 y, and negatively predicted any drug abuse symptoms through alcohol use frequency at 17 y. Indirect effects from early risk factors to any drug abuse symptoms were also found (see Figure 1). Delinquency at 12 y indirectly predicted having any drug abuse symptoms by 28 y through CU age of onset and cannabis frequency at 17 y. Affiliation with deviant friends at 13 y indirectly predicted having any drug abuse symptoms by 28 y through CU age of onset and cannabis, other drug and alcohol frequency at 17 y.

Discussion

The results of the present study highlight that cannabis users in early adolescence are at higher odds of developing drug abuse symptoms in adulthood. Previous research has shown that any CU during adolescence was associated with more substance use problems later in life⁴ and that onset before 16 y was associated with cannabis dependence.⁹ The present study extends upon those results not only by showing that any CU during adolescence is associated with higher odds of reporting drug abuse symptoms by 28 y, but also that an earlier age of onset increased these odds further. A second contribution of this study was to qualify and quantify the risk conveyed by early age of onset for developing drug abuse symptoms by 28 y by including early risk factors in the models and examining potential explanatory mechanisms. Much of the effect found between CU age of onset and drug abuse symptoms was explained by the frequency of use of cannabis at 17 y, and partly by other drugs. Still, even when taking frequency of use into account, adolescents who started using before 15 y were at higher risk of developing drug abuse symptoms. Furthermore, although earlier CU was associated with higher alcohol use frequency at 17 y, alcohol frequency was negatively associated with drug abuse symptoms by 28 y. Nonetheless, it must be noted that alcohol frequency at 17 y was associated with increased odds of alcohol abuse symptoms by 28 y (OR = 1.16; 95% CI = 1.04 to 1.30; see Appendix Figure S2), suggesting a specific association between alcohol use in adolescence and alcohol problems in adulthood. These specific associations between adolescent alcohol frequency and later alcohol use disorders, but not cannabis or other drug disorders, have previously been shown in other research,^{32,33} but future studies are needed to clarify how frequent adolescent cannabis or

Figure 1. Significant direct and indirect paths from early risk factors to drug abuse symptoms by 28 y through cannabis use (CU) age of onset. AUP: alcohol use problems. Double lined arrows indicate indirect paths going through cannabis age of onset. Model fit: $\chi^2 = 9.68$, $df = 9$, $P = 0.38$; CFI = 1.00; TLI = 0.99; RMSEA = 0.009 (90%CI, 0.000 to 0.037). Correlations among variables within a developmental period (i.e., among early common risk factors, and among substance use frequency variables at 17 y) were included in the model. Standardized coefficients are provided. * $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$. Significant indirect effects from CU age of onset to drug abuse symptoms by 28 y included: CU age of onset → CU frequency at 17 y → drug abuse symptoms by 28 y ($ab = -0.196$, $SE = 0.058$, $P = 0.001$); CU age of onset → other drug frequency at 17 y → drug abuse symptoms by 28 y ($ab = -0.071$, $SE = 0.032$, $P = 0.026$); and CU age of onset → alcohol frequency at 17 y → drug abuse symptoms by 28 y ($ab = 0.077$, $SE = 0.034$, $P = 0.024$). Significant indirect effects from early factors through CU age of onset predicting drug abuse symptoms by 28 y included: delinquency at 12 y → CU age of onset → CU frequency at 17 y → drug abuse symptoms by 28 y ($abc = 0.035$, $SE = 0.014$, $P = 0.011$); deviant friends at 13 y → CU age of onset → CU frequency at 17 y → drug abuse symptoms by 28 y ($abc = 0.047$, $SE = 0.016$, $P = 0.004$); deviant friends at 13 y → CU age of onset → other drug frequency at 17 y → drug abuse symptoms by 28 y ($abc = 0.017$, $SE = 0.008$, $P = 0.038$); deviant friends at 13 y → CU age of onset → alcohol frequency at 17 y → drug abuse symptoms by 28 y ($abc = -0.018$, $SE = 0.009$, $P = 0.039$). Other significant direct and indirect paths (predicting high school graduation, other drug frequency, cigarette use frequency, alcohol use frequency and delinquency) are provided in the Supplementary Materials (Figure S1).

alcohol use, or both, increase the risk of developing specific substance use problems in early onset substance users (be it early onset-cannabis and/or alcohol users). Finally, we found 2 different independent pathways to drug abuse symptoms through early onset CU: one stemming from high levels of delinquency and the other from an affiliation with deviant peers in early adolescence.

Clinical Implications

Some school-based prevention programs have already been shown to be effective in reducing substance use

among 12- to 19-y-old adolescents, the most effective being interactive programs that incorporate a motivational social component, where adolescents notably develop skills to resist peer pressure, but still include other aspects such as knowledge and affective components.³⁴ The finding that a younger CU age of onset during adolescence, particularly between 13 and 15 y, increases the odds of developing drug abuse symptoms stresses the importance of targeting this period for preventing or reducing CU, and thus it may be important to implement these programs by the end of elementary school, at least for the prevention of CU.

However, targeting early adolescence risk factors upstream could also prevent drug abuse symptoms without explicitly addressing substance use. Indeed, because early delinquency and affiliation with deviant friends were indirectly associated with drug abuse symptoms by 28 y through early CU onset, targeting these risk factors before adolescence or in early adolescence, either through community, health, or education approaches, may reduce the odds of initiating CU early and developing later drug abuse symptoms. Whereas studies have already shown that interventions in childhood and early adolescence targeting delinquent behaviours can be effective in reducing substance use during adolescence,^{35,36} future studies could look at whether such interventions have longer-term effects on adult substance use problems.

Strengths and Limitations

The main strength of this study was its prospective design, which allowed the examination of the predictive relationships and indirect effects of CU age of onset from a developmental perspective. The use of prospective longitudinal data also allowed for a more reliable measure of CU age of onset, as retrospective reports have been found to be only moderately reliable, especially for participants with an earlier age of onset.⁷ Still, some limitations should be considered. While the current study included various risk factors for substance use and examined indirect effects from early risk factors to drug abuse symptoms in adulthood, there may still be other factors to explain the associations. Notably, since adolescence is an important period for brain development,³⁷ early risk factors and CU may have an impact on neurological development and cognitive function,¹² which could be tested as potential mediators of the association between the risk for CU onset and substance abuse in future studies. Regarding the study design, although its prospective nature was a strength, it remains correlational and thus does not show causal relationships. The current sample included only French-speaking Caucasian boys from low socioeconomic neighbourhoods in Montreal and the results should be replicated with girls and in more diverse geographical populations. Substance use data were obtained by self-report, which is susceptible to bias, notably social desirability. That said, participants were guaranteed confidentiality and self-reports have been shown as reliable in assessing substance use.³⁸ Furthermore, quantity, potency, and mode of administration of cannabis were not considered and future studies should look at whether these factors affect the association between CU age of onset and drug abuse symptoms. Notably, considering that the potency of cannabis products increased over the last 2 decades³⁹ and that adolescent CU was assessed from 1991 to 1995, it is possible that the higher content of Δ -9-tetrahydrocannabinol in the cannabis available today would be associated with higher rates of drug abuse symptoms.

Conclusions

Previous studies have examined how CU onset before 18 y or 16 y was associated with later substance use problems.⁵⁻⁹ The present study builds on this previous data, examining CU onset between 13 and 17 y of age. Our results showed that CU frequency explained much of the association between CU age of onset and later drug abuse symptoms but that CU onset at 13 or 14 y was still associated with higher odds of developing drug abuse symptoms by 28 y, even after controlling for cannabis and other substance use frequency. Explanatory mechanisms were examined and indirect pathways to drug abuse symptoms through early onset CU were found to be associated with delinquency and affiliation with deviant peers in early adolescence. This suggests that targeting these risk factors in late childhood or early adolescence may help prevention efforts aimed at delaying CU.

Declaration of Conflicting Interests

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Supplemental Material

Supplementary material for this article is available online.

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