Age of cannabis use onset and adult drug abuse symptoms: a prospective study of common risk factors and indirect effects

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Charlie Rioux, B.Sc. - PhD Candidate, Department of Psychology, Université de Montréal; CHU Sainte-Justine Hospital Research Centre.

Natalie Castellanos-Ryan, Ph.D. - Assistant professor, School of Psychoeducation, Université de Montréal; Researcher, CHU Sainte-Justine Hospital Research Centre.

Sophie Parent, Ph.D. - Professor, School of Psychoeducation, Université de Montréal; Vice-Dean of undergraduate studies and digital strategies, Faculty of Arts and Sciences, Université de Montréal.

Frank Vitaro, Ph.D. - Professor, School of Psychoeducation, Université de Montréal; Researcher, CHU Sainte-Justine Hospital Research Centre.

Richard Ernest Tremblay, Ph.D. - Emeritus professor, Department of Psychology, Université de Montréal; Associate professor, Department of Pediatrics, Université de Montréal; Professor, School of Public Health and Population Sciences, University College Dublin; Researcher, CHU Sainte-Justine Hospital Research Centre.

Jean Richard Séguin, Ph.D. - Professor, Department of Psychiatry, Université de Montréal; Researcher, CHU Sainte-Justine Hospital Research Centre.

Corresponding author: Dr Natalie Castellanos-Ryan, École de Psychoéducation, Université de Montréal, Pavillon Marie-Victorin, 90 av. Vincent-d'Indy, Outremont (QC), H2V 2S9, Canada; Telephone number: 514 343 6111 ext. 28511; E-mail: natalie.castellanos.ryan@umontreal.ca.

Abstract

Objective: The present study examined (1) whether the associations between cannabis use (CU) age of onset and drug abuse by 28 years remain when controlling for risk factors in childhood, adolescence and early adulthood; and (2) developmental pathways from early risk factors to drug abuse problems.

Method: Participants from a longitudinal sample of low SES boys (N=1030) were followed from 6 to 28 years. Self-reported CU onset between 13 and 17 years of age and drug abuse symptoms by 28 years were examined.

Results: The odds of developing any drug abuse symptoms by 28 years were reduced by 31% for each year of delayed CU onset (OR = 0.69). Cannabis, alcohol and other drug frequency at 17 years mediated this association. Still, even when taking that frequency of use into account, adolescents who started using cannabis before 15 years were at higher risk of developing drug abuse symptoms by age 28. Significant indirect effects were found from early adolescent delinquency and affiliation with deviant friends to drug abuse symptoms at 28 years through CU age of onset and substance use frequency at 17 years.

Conclusions: Results suggest more clearly than before that prevention programs should aim at delaying CU onset in order 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.

Keywords: cannabis, marijuana, early onset, substance abuse disorder

Abbreviations: CU = cannabis use

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. While two 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 years found that CU onset before 16 years was positively associated with cannabis dependence at 20 years [8] and CU frequency and dependence at 24 years [9]. However, using dichotomous variables with a cut-off at 16 years 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 associated with higher odds of later alcohol and drug dependence [13-15]. While 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 it is only a minority of cannabis users that 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 is 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] and alcohol, cigarette and illicit drug use [5,6]. Although these previous studies collectively included a variety of covariates, each individual study controlled only for a limited number of these. 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 years by examining: 1) whether the associations between CU age of onset and drug abuse symptoms by 28 years remain when taking into account (a) early common risk factors (6-13 years), (b) cannabis and other substance use frequency (17 years), and (c) explanatory variables in early adulthood (20-23 years); and 2) the developmental pathways (indirect effects) from early common risk factors to drug abuse symptoms by 28 years through CU age of onset, substance use frequency during adolescence, and explanatory variables in early adulthood.

Method

Participants

Participants come from the Montreal Longitudinal and Experimental Study of low SES boys (MLES). In 1984, 1037 French-speaking boys (mean age = 6.1; SD = 0.31) were recruited from schools in low socio-economic 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, and at ages 20 and 28. 952 boys participated at least once during the adolescent period and 536 participated at 28 years. The total sample for this study consists of 1030 boys with data on any outcome variable between 17 and 28 years of age, i.e., CU age of onset, high school graduation, substance use at 17, delinquency at 20 and drug abuse symptoms at 28. 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 years old, 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 months. The scale includes 7 items referring to the absence (scored 0) and presence (scored 1) of problems over the past 12 months. 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 years, adolescents were asked whether they had consumed cannabis in the last twelve months. 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 years) to 6 (no CU onset by 17 years).

Early common risk factors. Paternal alcohol use problems were assessed when the boys were 12 years old 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 years was assessed using six 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 years 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 years was assessed using the Sentence Completion Test [25].

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

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

Delinquency at 12 years was measured using 17 items from the Self-Reported Delinquency Questionnaire (SRDQ)[28] assessing involvement in delinquent behaviours over the last 12 months 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 two items ($\alpha = 0.73$) asking "Do your parents know where you are when you are not at home?" and "Do your parents

know 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 years. Cigarette, alcohol, cannabis and other drug frequency at 17 years were 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 twelve months (1 = never, 7 = 40+ times).

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

Finally, delinquency at 20 years was measured using the SRDQ (see early common risk factors section). Cronbach's alpha for this scale at 20 years 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 four 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 1000 samples bias-corrected bootstrap, which reduces bias and yields 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 years decreased with increasing age of CU onset, dropping from 77% in boys who initiated CU at 13 years or before to 48% in boys who initiated CU at 17 years, 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 Tables S2 and S3, provided as supplementary material.

Does the association between CU age of onset and drug abuse symptoms by 28 years remain when taking into account other risk factors?

Results from 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 years 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 years and their inclusion did not affect the association between CU age of onset and later drug abuse symptoms. Once substance use frequency at 17 years 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 years was associated with 1.32 times greater odds of reporting any drug abuse symptoms by 28 years. When early adulthood explanatory variables were added to the model (model 4; $\Delta R^2 = 0.03$), only alcohol use frequency at 17 years 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 were non-significant if CU had its onset between 15 and 17 years, but were significant and almost doubled each year if onset was before 15 years (OR = 1.97, onset by 14; OR = 3.44, onset by 13). Indeed, rates of having any drug abuse symptoms by 28 years were 68% in cannabis users who started early (at or before 14 years) compared to 44% in users who started later (χ^2 (1, N = 269) = 9.39, p = .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 years were significantly higher in cannabis users who consumed cannabis frequently at 17 years (20+ times in the last year) than in cannabis users who consumed less frequently (72% vs 40%; χ^2 (1, N = 227) = 22.29, p < .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 years, impulsivity and affiliation with deviant peers at 13 years 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 drugs use at 17 years, and delinquency at 20 years. Finally, a higher frequency of cannabis and other drug use at 17 years as well as a lower frequency of alcohol use were associated with having any drug abuse symptoms by 28 years.

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

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 had

shown that any CU during adolescence was associated with more substance use problems later in life [4] and that onset before 16 years was associated with cannabis dependence [9]. The present study extends those results not only by showing that any CU during adolescence is associated with higher odds of reporting drug abuse symptoms by 28 years, 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 years 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 years, and partly by other drugs. Still, even when taking frequency of use into account, adolescents who started using before 15 years were at higher risk of developing drug abuse symptoms. Furthermore, although earlier CU was associated with higher alcohol use frequency at 17 years, alcohol frequency was negatively associated with drug abuse symptoms by 28 years. Nonetheless, it must be noted that alcohol frequency at 17 years was associated with increased odds of alcohol abuse symptoms by 28 years (OR = 1.16; 95% CI = 1.04 - 1.30; see supplementary material 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 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, two different independent pathways to drug

abuse symptoms through early onset CU were found, one stemming from high levels of delinquency, and the other from affiliation with deviant peers in early adolescence.

Clinical implications

Some school-based prevention programs have already been shown as effective in reducing substance use among 12- to 19-year-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 years, 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 years 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. While 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 a variety of 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 explaining the associations that were found. 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 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 not show causal relationships. The current sample included only French-speaking Caucasian boys from low SES neighbourhoods in Montreal and 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 two decades [39] 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

While previous studies looked at how CU onset before 18 or 16 years was associated with later substance use problems [5-9], the present study examined CU onset between 13 and 17 years of age. 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 years was still associated with higher odds of developing drug abuse symptoms by 28 years, 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 from 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 aiming at delaying CU.

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Table 1. Sample characteristics and substance use prevalence

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

[a] Occupational prestige based on Canadian norms; scale range 17.8 to 101.7; population average 42.7, SD 13.3.
[b] Family adversity range is 0-1.

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%C I	B	OR	95%CI	B	OR	95%CI
Cannabis – age onset	-0.37***	0.69	0.61 - 0.78	-0.37***	0.69	0.60 - 0.79	-0.06	0.95	0.74 - 1.14	-0.04	0.97	0.80 - 1.17
Father AUP [a]				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 6y				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 12y				-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 13y				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 13y				-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 13y				-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 12y				-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 12y				-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 17y							0.06	1.07	0.98 - 1.16	0.06	1.06	0.97 - 1.16
Alcohol frequency 17y							-0.11	0.90	0.79 - 1.01	-0.14*	0.87	0.77 - 0.99
Cannabis frequency 17y							0.28***	1.32	1.14 - 1.53	0.30***	1.35	1.16 - 1.59
Other drug frequency 17y							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 20y										0.13	1.14	0.99 - 1.31

[a] AUP: Alcohol Use Problems

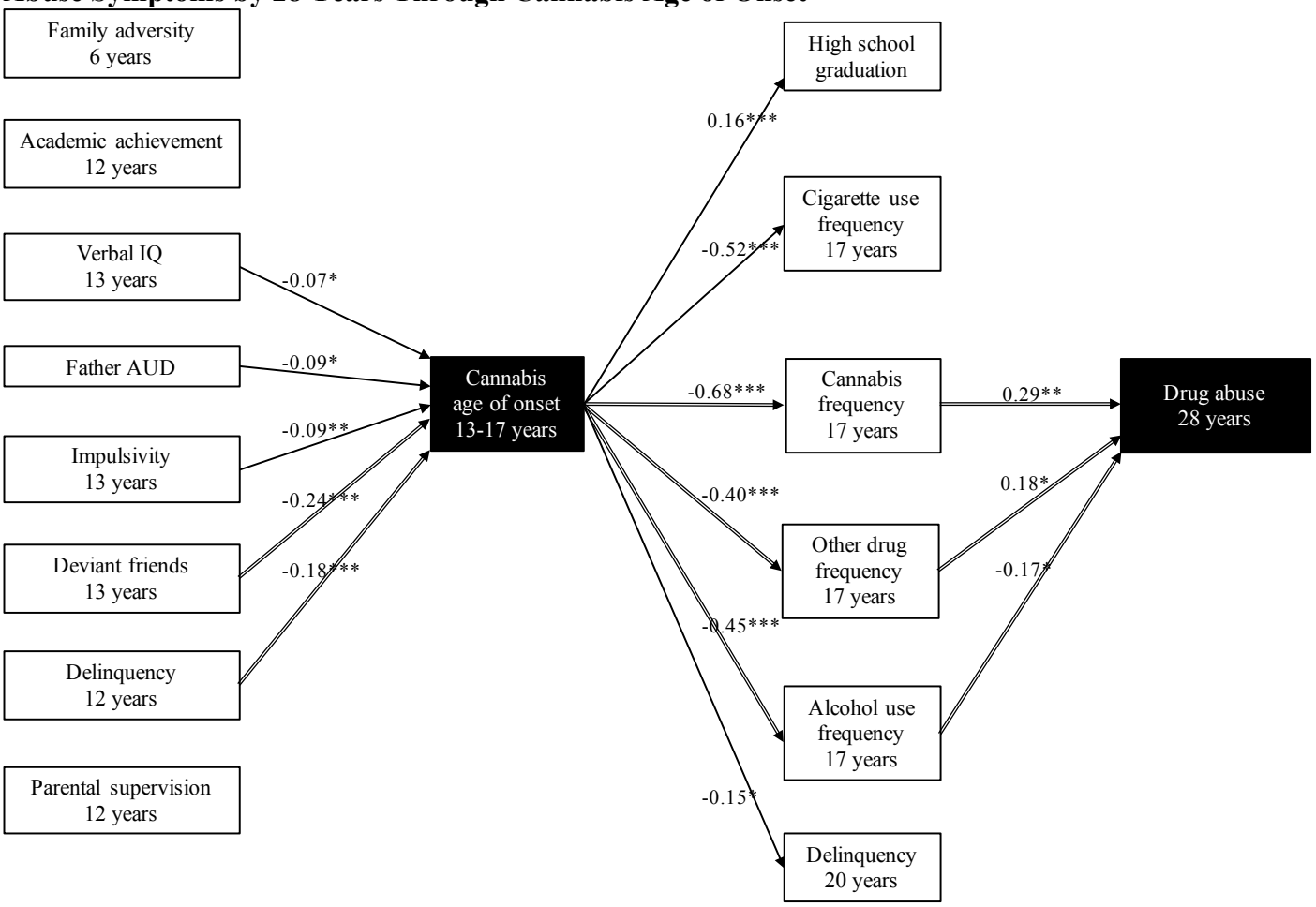
*p<.05, **p<.01, ***p<.001.

Table 3. Odds of drug abuse symptoms by 28 years for each year of delayed onset of cannabis use [a]

	Drug abuse symptoms	
Onset by	OR	95% CI
13 years	3.44	1.05 - 11.22
14 years	1.97	1.08 - 3.61
15 years	1.10	0.68 - 1.80
16 years	0.87	0.51 - 1.49
17 years	1.14	0.64 - 2.03

[a] Comparison 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 years.

FIGURE 1. Significant Direct and Indirect Paths From Early Risk Factors to Drug Abuse Symptoms by 28 Years Through Cannabis Age of Onset



[a] AUP: alcohol use problems. Double lined arrows indicate indirect paths going through cannabis age of onset. Model fit: $\chi^2 = 9.68$, dl = 9, p = 0.38; CFI=1.00; TLI = 0.99; RMSEA=.009 (90%CI=.000-.037); Correlations among variables within a developmental period (i.e., among early common risk factors, and among substance use frequency variables at 17 years) were included in the model. Standardized coefficients provided. *p<.05, **p<.01, ***p<.001.

Significant indirect effects from cannabis use age of onset to drug abuse symptoms by 28 included: cannabis age of onset → cannabis frequency at 17 years → drug abuse symptoms by 28 (ab=-.196, SE=.058, p=.001); cannabis age of onset → other drug frequency at 17 years → drug abuse symptoms by 28 (ab=-.071, SE=.032, p=.026); and cannabis age of onset → alcohol frequency at 17 years → drug abuse symptoms by 28 (ab=.077, SE=.034, p=.024). Significant indirect effects from early factors through cannabis age of onset predicting drug abuse symptoms by 28 included: delinquency at 12 → cannabis age of onset → cannabis frequency at 17 → drug abuse symptoms by 28 (abc=.035, SE=.014, p=.011); deviant friends at 13 → cannabis age of onset → cannabis frequency at 17 → drug abuse symptoms by 28 (abc=.047, SE=.016, p=.004); deviant friends at 13 → cannabis age

of onset → other drug frequency at 17 → drug abuse symptoms by 28 (abc=.017, SE=.008, p=.038); deviant friends at 13 → cannabis age of onset → alcohol frequency at 17 → drug abuse symptoms by 28 (abc=-.018, SE=.009, p=.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 supplementary materials (Figure S1).

Supplementary material

To accompany: Rioux C, Castellanos-Ryan N, Parent S, Vitaro F, Tremblay RE, Séguin JR. Age of cannabis use onset and adult drug abuse symptoms: a prospective study of common risk factors and indirect effects.

Table S1. Correlations and descriptive statistics for all study variables

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
1. Cannabis age of onset	–															
2. Father AUP [a]	-.12**	–														
3. Familial adversity at 6	-.05	.13**	–													
4. Academic performance at 12	.04	-.04	-.29**	–												
5. Verbal IQ at 13	-.04	.01	-.22**	.41**	–											
6. Impulsivity at 13	-.19**	.05	-.11**	-.14**	-.10**	–										
7. Deviant friends at 13	-.33**	.04	.10**	-.08*	-.02	.23**	–									
8. Delinquency at 12	-.29**	.07	.10**	-.14**	-.15**	.25**	.35**	–								
9. Parental supervision at 12	.20**	-.06*	-.05	-.13**	.01	-.28**	-.20**	-.39**	–							
10. Cigarette frequency at 17	-.49**	.08	.09*	-.12**	-.04	.13**	.16**	.11**	-.06	–						
11. Alcohol frequency at 17	-.45**	.01	-.07*	.10**	.21**	.09*	.14**	.13**	-.07	.40**	–					
12. Other drug frequency at 17	-.43**	.07	.07	-.03	.01	.15**	.18**	.21**	-.09*	.28**	.36**	–				
13. Cannabis frequency at 17	-.66**	.06	.04	-.02	.12**	.11**	.18**	.17**	-.13**	.47**	.56**	.67**	–			
14. High school graduation	.17**	-.14**	-.41**	.50**	.41**	-.16**	-.12**	-.18**	.09*	-.25**	.03	-.10**	-.10**	–		
15. Delinquency at 20	-.22**	-.01	.08*	-.08*	.00	.12*	.29**	.27**	-.15**	.11	.18**	.17**	.16**	-.09*	–	
16. Drug abuse symptoms by 28	-.33**	.09*	.03	-.04	.10*	.01	.08*	.08*	-.12**	.26**	.15**	.38**	.43**	-.08*	.23**	–
Mean	4.50	.15	.33	2.88	9.04	1.64		27.26	6.58	3.35	3.77	1.64	2.79		21.66	
Standard deviation	1.55	.36	.25	1.16	2.14	1.58		4.33	1.44	2.78	2.06	1.42	2.31		2.63	
Skewness	-.58	1.97	.38	.04	-1.08	.61		2.11	-.81	.47	.08	2.39	0.88		2.13	
Kurtosis	-.91	1.90	-.73	-.69	1.20	-.81		4.52	.01	-1.71	-1.30	4.84	-0.89		4.63	
% Yes							18.5							53.3		36.9

[a] AUP: Alcohol Use Problems

*p<.05, **p<.01, ***p<.001.

Table S2. Number of years of cannabis use by cannabis age of onset.

Cannabis age of onset	Number of years of cannabis use from 13 to 17 years				
	1	2	3	4	5
13 yrs	6.5%	12.9%	9.7%	16.1%	54.8%
14 yrs	16.2%	12.2%	4.1%	54.1%	
15 yrs	12.9%	21.4%	65.7%		
16 yrs	27.2%	72.8%			
17 yrs	100%				

Table S3. Percentage using other substances at 17 years by cannabis age of onset.

Cannabis age of onset	% using alcohol	% using tobacco	% using other drugs
13 yrs	88	83	57
14 yrs	95	75	57
15 yrs	93	77	46
16 yrs	91	57	38
17 yrs	95	57	28
Any use (13-17 yrs)	93	67	42

Figure S1

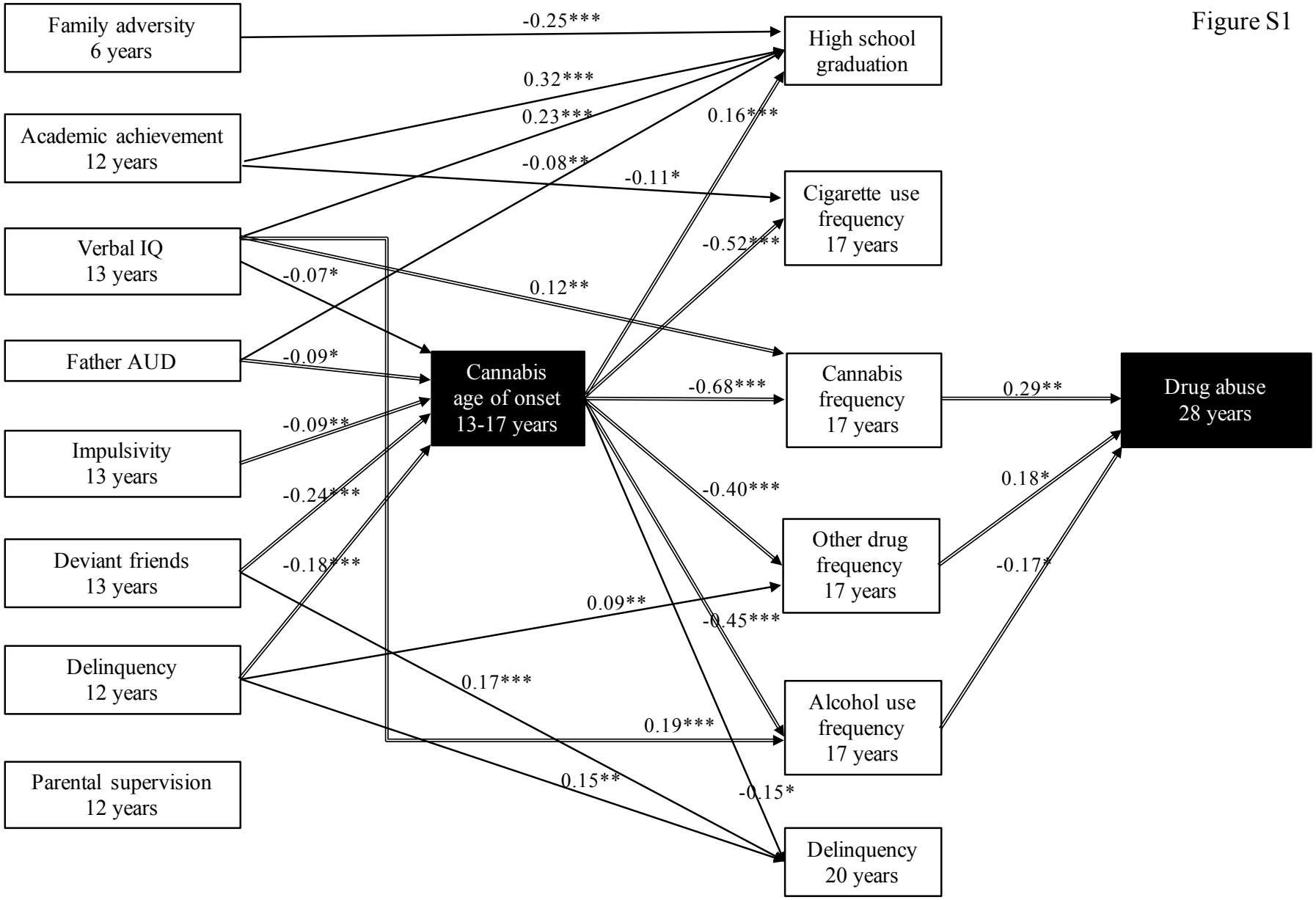

Family adversity 6 years
Academic achievement 12 years
Verbal IQ 13 years
Father AUD
Impulsivity 13 years
Deviant friends 13 years
Delinquency 12 years
Parental supervision 12 years
Cannabis age of onset 13-17 years
High school graduation
Cigarette use frequency 17 years
Cannabis frequency 17 years
Other drug frequency 17 years
Alcohol use frequency 17 years
Delinquency 20 years
Drug abuse 28 years
-0.25***
0.32***
0.23***
-0.08**
-0.11*
0.16***
-0.52***
0.12**
-0.07*
-0.09*
-0.09**
-0.24***
-0.18***
-0.68***
-0.40***
-0.45***
0.09**
0.17***
0.19***
0.15**
-0.15*
0.29**
0.18*
-0.17*

Figure S1. All significant paths in tested model. AUP: alcohol use problems. Double lined arrows indicate indirect paths. Standardized coefficients provided. †p=.05 *p<.05, **p<.01, ***p<.001.

Indirect effects predicting high school graduation through cannabis age of onset included: delinquency at 12 → cannabis age of onset → high school graduation (ab=-.027, SE=.010, p=.005); impulsivity at 13 → cannabis age of onset → high school graduation (ab=-.014, SE=.007, p=.038); deviant friends at 13 → cannabis age of onset → high school graduation (ab=-.037, SE=.012, p=.002).

Indirect effects predicting cigarette use frequency at 17 through cannabis age of onset included: delinquency at 12 → cannabis age of onset → cigarette use frequency at 17 (ab=.093, SE=.023, p<.001); father AUD → cannabis age of onset → cigarette use frequency at 17 (ab=.047, SE=.022, p=.036); impulsivity at 13 → cannabis age of onset → cigarette use frequency at 17 (ab=.047, SE=.019, p=.016); deviant friends at 13 → cannabis age of onset → cigarette use frequency at 17 (ab=.124, SE=.025, p<.001).

Indirect effects predicting cannabis frequency at 17 through cannabis age of onset included: delinquency at 12 → cannabis age of onset → cannabis frequency at 17 (ab=.120, SE=.028, p<.001); father AUD → cannabis age of onset → cannabis frequency at 17 (ab=.060, SE=.028, p=.032); impulsivity at 13 → cannabis age of onset → cannabis frequency at 17 (ab=.061, SE=.025, p=.014); deviant friends at 13 → cannabis age of onset → cannabis frequency at 17 (ab=.160, SE=.030, p<.001).

Indirect effects predicting alcohol use frequency at 17 through cannabis age of onset included: delinquency at 12 → cannabis age of onset → alcohol use frequency at 17 (ab=.081, SE=.020, p<.001); father AUD → cannabis age of onset → alcohol use frequency at 17 (ab=.040, SE=.019, p=.036); impulsivity at 13 → cannabis age of onset → alcohol use frequency at 17 (ab=.041, SE=.017, p=.016); deviant friends at 13 → cannabis age of onset → alcohol use frequency at 17 (ab=.107, SE=.021, p<.001).

Indirect effects predicting other drug frequency at 17 through cannabis age of onset included: father AUD → cannabis age of onset → other drug frequency at 17 (ab=.036, SE=.017, p=.038); impulsivity at 13 → cannabis age of onset → other drug frequency at 17 (ab=.036, SE=.015, p=.018); deviant friends at 13 → cannabis age of onset → other drug frequency at 17 (ab=.094, SE=.019, p<.001). Indirect effects not going through cannabis age of onset included: Verbal IQ at 13 → cannabis frequency at 17 → drug abuse by 28 (ab=.036, SE=.015, p=.018); Verbal IQ at 13 → alcohol frequency at 17 → drug abuse by 28 (ab=-.032, SE=.016, p=.046).

Figure S2

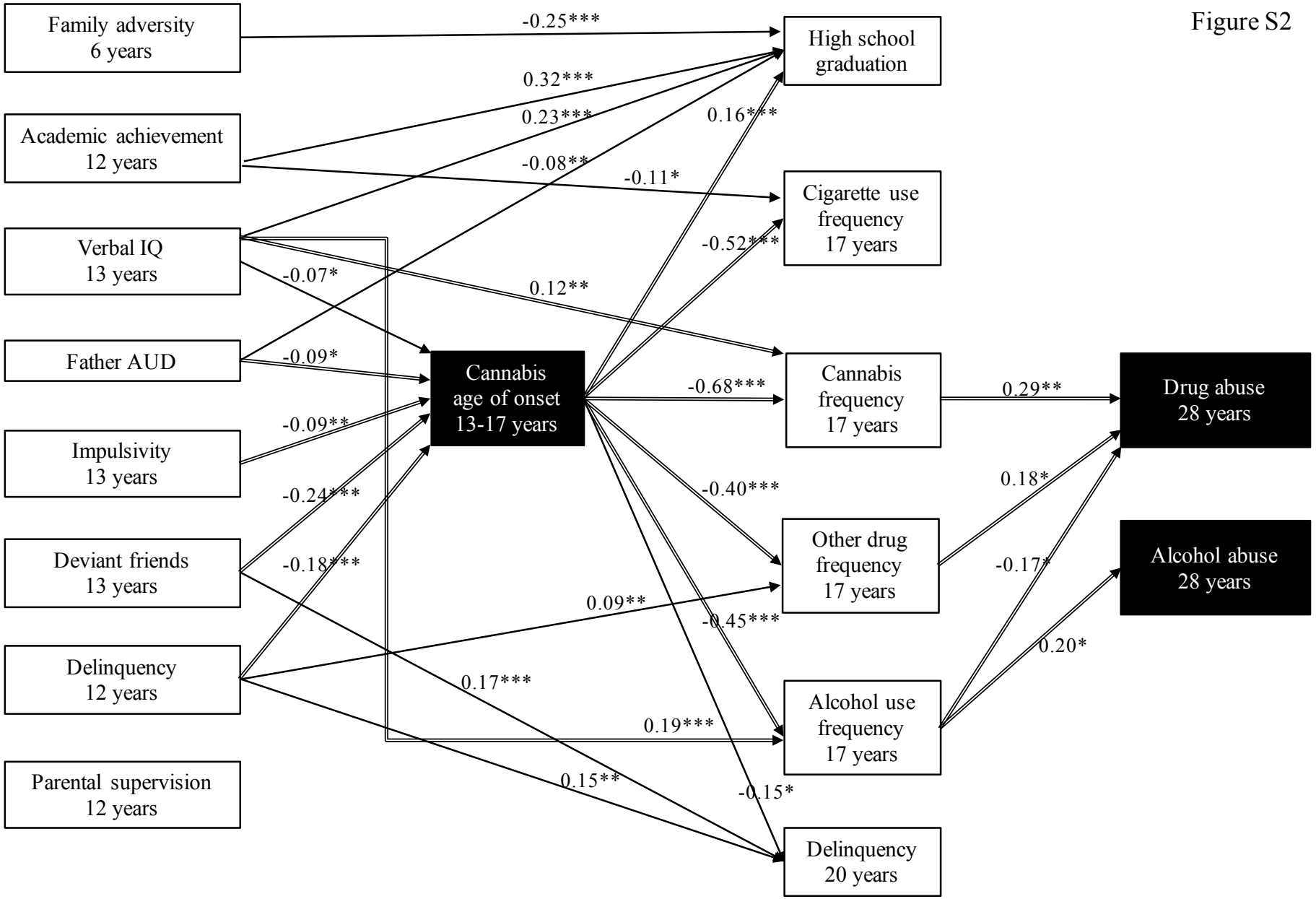

Family adversity 6 years
Academic achievement 12 years
Verbal IQ 13 years
Father AUD
Impulsivity 13 years
Deviant friends 13 years
Delinquency 12 years
Parental supervision 12 years
Cannabis age of onset 13-17 years
High school graduation
Cigarette use frequency 17 years
Cannabis frequency 17 years
Other drug frequency 17 years
Alcohol use frequency 17 years
Delinquency 20 years
Drug abuse 28 years
Alcohol abuse 28 years
-0.25***
0.32***
0.23***
-0.08**
-0.11*
0.16***
-0.52***
-0.07*
0.12**
-0.09*
-0.68***
0.29**
-0.09**
-0.24***
-0.40***
0.18*
-0.18***
-0.17*
0.09**
-0.45***
0.20*
0.17***
0.19***
0.15**
-0.15*

Figure S2. Path model including alcohol abuse symptoms by 28 years. The alcohol abuse scale was computed from the same questionnaire as the drug abuse symptoms scale, but with items asking about problems related to alcohol use specifically. The variable represents the presence or absence of alcohol abuse symptoms corresponding to DSM-IV criteria (0 = abuse symptoms absent; 1 = at least one abuse symptom present). Double lined arrows indicate indirect paths. Model fit: $\chi^2 = 9.68$, dl = 9, p = 0.38; CFI=1.00; TLI = 0.99; RMSEA=.009 (90%CI=.000-.037); *p<.05, **p<.01, ***p<.001.
Indirect effects predicting alcohol abuse symptoms by 28 years included: Cannabis age of onset → alcohol use frequency at 17 → alcohol abuse by 28 (ab=-.092, SE=.039, p=.018); Verbal IQ at 13 → alcohol use frequency at 17 → alcohol abuse by 28 (ab=.039, SE=.018, p=.031); Deviant friends at 13 → cannabis age of onset → alcohol use frequency at 17 → alcohol abuse by 28 (abc=.022, SE=.010, p=.031).