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Is the relationship between early-onset cannabis use and educational attainment causal or due to common liability?

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Abstract

Background—Several studies have shown that early cannabis use is correlated with poor educational performance including high school drop-out. The predominant explanation for this relationship is that cannabis use causes disengagement from education. Another explanation is that the association between early cannabis use and educational attainment is not causal, but the result of overlapping risk factors that increase the likelihood of both early cannabis use and disengagement from education. These confounding factors could be of genetic and/or environmental origin.

Methods—Here we use data from a large community-based sample of adult twins (N=3337) who completed a comprehensive semi-structured telephone interview. We first apply the classical twin-design to determine whether genetic and/or environmental influences underlie the relationship between early-onset cannabis use (prior to age 18) and early school leaving. Next, with a co-twin control design we investigate whether the relationship between the two variables is more likely due to direct causality or overlapping risk factors.

Results—We find a significant phenotypic correlation between early-onset cannabis use and early school leaving ($r=0.26$), which could be explained by familial influences (of genetic and/or shared environmental origin). The pattern of odds ratios found in the co-twin control design is not consistent with direct causation, but rather suggests that the association is due to shared environmental factors influencing both variables.

Conclusion—Our findings suggest that the relationship between early-onset cannabis use and school leaving is due to shared environmental risk factors influencing both the risk of early-onset cannabis use and early school leaving.

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Contributors

KJHV was responsible for the study concept and design of the study. NGM and MTL contributed to the data acquisition. KJHV performed the data analysis and interpretation of findings. KJHV drafted the manuscript. ACH, AA, NGM, and MTL provided critical revision of the manuscript for important intellectual content and approved the final version for publication.

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Keywords

cannabis; education; environment; genetics; school drop-out; twin research

1. INTRODUCTION

Cannabis is the most widely consumed illicit drug worldwide, and prevalence of use is especially high among adolescents and young adults (United Nations Office on Drugs and Crime, 2010). A large body of research has demonstrated the adverse effects of cannabis use; it is associated with accidents, violence, and use of other drugs (Hall, 2009; Huas et al., 2008) and regular use can cause physical or psychological problems (Fergusson and Horwood, 1997, 2000; Hall, 2009; Hall and Babor, 2000; Hall and Degenhardt, 2009; Hall and Solowij, 1998). Public health costs, law enforcement, and loss of productivity and work potential because of cannabis use are an economic drain on society (Hall and Babor, 2000).

Cannabis use is typically initiated during adolescence (Chen and Kandel, 1995) and given the widespread use and its negative effects on cognitive tasks and psychological functioning, there has been increasing concern about its adverse effects on a host of psychosocial outcomes, including educational attainment. Several studies have shown that early cannabis use is correlated with poor educational performance, including higher rates of absenteeism, worse school performance, higher drop-out rates, and failure to attend tertiary education (Bray et al., 2000; Fergusson et al., 2003, 1996; Horwood et al., 2010; Lynskey et al., 2003). In turn, educational attainment has a severe impact on future life opportunities; individuals that drop out of school are at risk for a range of negative outcomes, including reduced occupational opportunities and income, poorer health, and involvement in crime (Beauvais et al., 1996).

The predominant explanation for the relationship between cannabis use and educational attainment is that cannabis use causes educational difficulties (Brook et al., 1999; Fergusson and Horwood, 1997; Yamada and Kendix, 1996). Possible mechanisms underlying this hypothesised causal association are that (heavy) cannabis use may lead to cognitive or motivational deficits, which encourages decreased participation in education (see Lynskey et al., 2003).

An alternative explanation is that poor educational performance causes cannabis use. Rates of cannabis use are higher among adolescents not attending school (Swaim et al., 1997), leading to the hypothesis that early school leaving leads to cannabis use. However, this elevation in rates of cannabis use in early school leavers disappears when correcting for cannabis use prior to school drop-out (Fergusson et al., 2003), suggesting this hypothesis is incorrect.

Another alternative explanation is that the association between cannabis use and poor educational attainment is not causal, but the result of overlapping risk factors increasing the likelihood of both early cannabis use and poor educational achievement. These confounding factors could be of genetic and/or environmental origin. Both cannabis use and educational achievement are found to be substantially heritable, with genetic factors estimated to account for ~45% of variance in cannabis use initiation and ~60% of variance in educational achievement (Baker et al., 1996; Szanton et al., 2009; Verweij et al., 2010). Overlapping environmental risk factors could include social disadvantages, family dysfunction, parental substance use, and peer influences (Fergusson et al., 2003; Lynskey and Hall, 2000).

While this common causes explanation has been proposed before (Fergusson et al., 2003; Lynskey and Hall, 2000), only two studies have directly investigated this with a genetically informative sample. Surprisingly, Bergen et al. (2008) did not find a significant association between illicit drug use and educational attainment, but for drug abuse/dependence their

findings suggested that the relationship with educational attainment is likely due to overlapping genetic factors that influence both traits. However, due to a lack of power they were not able to rule out a role for overlapping shared environmental influences. A limitation of their study is that they had no information on when the participants started using drugs, so they did not know whether drug use preceded or followed educational dropout. In the second paper, Grant et al. (2012) tested whether alcohol, nicotine, and illicit drug use and dependence were associated with educational attainment (measured as more or less than 16 years of education). By using twin data they were able to investigate the role of substance use in educational attainment controlling for shared familial factors. They found that the association of cannabis initiation, early use, and dependence with educational attainment could be explained by overlapping familial factors, but were unable to distinguish between genetic and shared environmental influences. While this study had a large sample size and the findings make an important contribution to unravelling the relationship between substance use and educational attainment, their study also had some limitations. The sample was drawn from the Vietnam Era Twin Registry, so consisted only of males who had almost all completed high school (a requirement for military service at the time) and who had access to military educational benefits. Therefore, the findings may not be generalisable to the general population and to females. Also, as completion of high school was a requirement, the researchers were not able to examine the more immediate relationship between substance use and high school drop-out.

These two studies were the first genetically informative studies to empirically show that the relationship between cannabis use and educational attainment is not likely to be causal, but due to overlapping familial factors. However, neither of them provided information about the relative importance of genetic and environmental influences to the relationship between cannabis use and educational attainment.

In the present study, we use a sample of 3337 male and female twins to more closely examine the relationship between cannabis use and educational attainment. We investigate the strength of the relationship between cannabis use before the age of 18 and early school leaving as well as the extent to which genetic, shared environmental, and residual factors contribute to this relationship. Furthermore, using the co-twin control design, we examine whether the association between early-onset cannabis use and school leaving is more likely explained by direct causation or by genetic or environmental factors influencing both variables.

2. METHODS

2.1 Participants

Twins participating in this study were drawn from the Australian Twin Registry (ATR). Between 2006 and 2009 these twins participated in a comprehensive computer-assisted telephone interview administered by trained interviewers. This interview was aimed primarily at assessing links between correlates of cannabis use phenotypes. Written informed consent was obtained from all participants.

A total of 3337 twins (1173 males and 2164 females) completed the questions regarding cannabis use and educational attainment. Of these, 2302 (69%) reported lifetime (ever) cannabis use. Participants were aged between 27 and 40 years ($\text{mean} \pm \text{S.D.} = 31.9 \pm 2.5$) and the sample included 571 identical (monozygotic; MZ), 653 non-identical (dizygotic; DZ) twin pairs, and 889 single twins (where only one twin of the pair participated). The zygosity of the twin pairs was determined based on responses to standard items about physical similarity, a procedure that has been found to have at least 95% concurrence with DNA typing (Ooki et al., 1990). For further details about the recruitment procedure and other study characteristics, see Lynskey et al. (2012).

2.2 Measures

2.2.1 Early-onset cannabis use—Twins were asked whether they had ever used cannabis, and (if they did) how old they were the first time they used cannabis. We grouped twins into two categories: those that used cannabis before the age of 18 (i.e. during high school), and those that never used cannabis or used for the first time after age 18. Table 1 shows the prevalences for lifetime cannabis use and use before the age of 18.

2.2.2 Early school leaving—Participants were asked for their highest completed educational level. Response options ranged from (1) ‘primary incomplete’ to (10) ‘post-graduate degree’ (see Table 1 for all response options and corresponding frequencies). In Australia, most people attend school for 12 years. Everyone is obliged to finish year 10 after which they are allowed to stop, but in order to be accepted for some tertiary education (e.g. university) they need to pass the year 12 exams. This variable was recoded into a dichotomous variable, where participants that did not finish year 12 (i.e. who reported year 11 or less as their highest level of education) were marked as early school leavers.

2.3. Statistical analysis

2.3.1 The classical twin-design—We used the classical twin design (see Neale and Cardon, 1992; Posthuma et al., 2003) to partition the variance in early-onset cannabis use and early school leaving into additive genetic (A), shared (family) environmental (C), and residual (E) components. A refers to variance resulting from the sum of allelic effects across all segregating genes. C includes environmental influences shared by family members which may include shared home environment and parental style. E denotes environmental factors that are not shared by twin pairs, stochastic biological effects, and measurement error. The variance decomposition can be achieved because identical twins share all their genes, while non-identical twins share on average half their segregating genes. A, C, and E influences therefore predict different patterns of MZ and DZ twin pair correlations, and structural equation modelling is used to determine the combination of influences that best matches the observed data. In the same way the cross-twin cross-trait correlations are used to partition the covariance between early-onset cannabis use and early school leaving into A, C, and E influences. In this way we obtain a measure of overlap in the genetic and environmental variation underlying both variables.

Structural equation modelling of twin data is performed in the flexible matrix algebra program Mx (Neale et al., 2006), which employs maximum likelihood modelling procedures. We fitted models to the raw dichotomous data, where it is assumed that a normally distributed continuum of liability underlies the dichotomous observed categories. Age and sex effects were accounted for by including them as covariates. The goodness-of-fit of a model is summarised by a statistic distributed as a chi-square (χ^2). By testing the change in model fit ($\Delta\chi^2$) against the change in degrees of freedom (Δdf), we can test whether constraining parameters to zero or constraining them to be equal across groups, significantly worsens the model fit. For ease of interpretation, the bivariate model was transformed into a correlated factors model (see Loehlin, 1996).

2.3.2 The co-twin control design—To test whether the association between cannabis use and early school leaving is more likely explained by direct causality or by underlying factors overlapping between both variables, we applied the co-twin control design (see Kendler et al., 1993; Ligthart and Boomsma, 2012). With this design we compare the risk of early school leaving of individuals discordant for early-onset cannabis use within MZ pairs, within DZ pairs, and in unrelated individuals. If cannabis use causes school drop-out, it is expected that in the general population those who use cannabis prior to age 18 will be at increased risk of leaving school (i.e. odds ratio [OR]>1); the same pattern is expected within discordant MZ and DZ twin pairs: the twins using cannabis at an early age will be at greater risk of leaving school

than their non-early-using co-twins and the strength of the association will not be attenuated by the degree of relatedness.

If, on the other hand, the relationship is not causal, but overlapping underlying factors influence both the risk of early-onset cannabis use and early school leaving, we would expect different patterns of ORs depending on the source (A, C, or E) of the covariation. If overlapping genetic influences explain the entire association, the expected OR for the general population is similar to the one above ($OR > 1$). However, within MZ twin pairs discordant for early cannabis use, we expect no elevation in risk of early school leaving in the early-onset cannabis using twin relative to their non-early using co-twin ($OR = 1$), because both twins inherited the same risk genes that influence liability to early-onset cannabis use and school leaving. For discordant DZ twins we expect the odds ratio to be intermediate between the OR for the general population and the OR for MZ twins, because DZ twins share on average 50% of their segregating genes.

When the relationship between the traits is entirely due to overlapping shared environmental factors (C), the expected OR for the general population again is similar to the one above ($OR > 1$). For MZ and DZ twins we expect that both twins have a similarly increased risk of early school leaving ($OR = 1$); the early-onset cannabis using twin will not be at higher risk for early school leaving, as both twins share the same environmental factors that predispose to the two traits. Last, if residual (individual-specific) factors (E) explain the association between cannabis use and school drop-out, only the twin exposed to these E factors will be at increased risk of both variables, which results in a pattern that resembles the causal model. Figure 1 summarises the pattern of expected ORs for the different mechanisms. In reality, the association between the two variables may result from a combination of these possible underlying mechanisms.

We selected all same-sex discordant twin pairs from our sample for this analysis. To obtain an OR for the general population we used the singletons and one twin per pair that was not part of a discordant MZ/DZ pair. Because there are sex differences in the prevalence of cannabis use and early school leaving, we selected these individuals such that the proportion of males and females matched the proportion in the discordant twin pairs (63% females) and excluded opposite-sex twin pairs from the DZ analysis. This yielded a total of 115 and 126 pairs of discordant MZ and DZ twin pairs, and 1872 unrelated individuals.

3. RESULTS

3.1 Preliminary analyses

Before modelling the variance components, we tested the effects of age, sex, and zygosity on the prevalences (thresholds) of early-onset cannabis use and school leaving ($\alpha = 0.01$).

As mentioned above, there were significant sex effects on the prevalence of both variables, such that males reported more early-onset cannabis use and early school leaving than females. Levels of early-onset cannabis use and early school leaving did not significantly differ between MZ and DZ twins in either sex. There was a significant age effect on the prevalence of early-onset cannabis use ($\Delta\chi^2_1 = 30.02$, $P < 0.0001$), indicating that younger participants were more likely to report cannabis use before the age of 18. There was no such age effect for school leaving ($\Delta\chi^2_1 = 1.25$, $P = 0.26$). The effects of sex and age were accounted for in subsequent modelling.

Polychoric twin pair correlations for each zygosity group (corrected for age and sex effects) are displayed in Table 2. There were no sex differences between male and female MZ nor between male, female, and opposite-sex DZ twin pair correlations for both variables, suggesting no qualitative or quantitative sex differences in the sources of variance between

sexes. The pattern of MZ versus DZ twin pair correlations suggest A and C influences on both traits - this is formally tested below.

3.2 Genetic modelling

Table 3 shows the results of the bivariate genetic models fitted to the observed cannabis use and school leaving data, controlling for the effects of sex and age. Based on preliminary analyses described above, we equated the A, C, and E estimates between males and females (model 1). The parameter estimates in this model (transformed into a correlated factors model) are presented in Figure 2. Shown are the proportions of variance in adolescent cannabis use and early school leaving accounted for by genetic (A, or heritability; h^2), shared environmental (C), and residual (E) influences, as well as the genetic (r_g), shared environmental (r_c), and residual (r_e) correlations (correlations between the latent A, C, and E influences). The parameter estimates suggest that adolescent cannabis use is strongly influenced by genetic factors (A=57%) and only moderately by shared environmental (C=20%) and residual factors (E=23%), while early school leaving is most strongly influenced by shared environmental factors (46%) and to a lesser extent by genetic (29%) and residual factors (25%). Furthermore, we estimated a significant phenotypic correlation between early-onset cannabis use and school leaving of $r=0.26$, primarily explained by shared environmental factors underlying both traits, and to a lesser extent by overlapping genetic factors.

To statistically test the significance of each parameter, genetic and environmental parameters were dropped from the base model and the fit of the reduced models was compared to the fit of the baseline model (see Table 3). To summarise, early-onset cannabis use was significantly influenced by genetic influences, but the shared environmental influences were not significant (see models 7 and 8). Conversely, the shared environmental influences were significant for early school leaving, but the genetic influences were not (see models 9 and 10). The phenotypic correlation between early-onset cannabis use and early school leaving was highly significant ($p<.001$, see Model 2). However, the genetic (r_g), shared environmental (r_c), and residual (r_e) correlations were not significant on their own (see models 3 to 5), but we could not drop *both* r_g and r_c from the model at once ($p<.001$, see model 6). This indicates that the relationship between early cannabis use and school leaving is at least partly due to overlapping familial factors (genetic and shared-environmental).

3.3 Co-twin control design

The phenotypic correlation between early-onset cannabis use and school leaving could be explained by a number of possible underlying mechanisms. Using the co-twin control design we tested whether the association is more likely explained by a direct causal relationship (cannabis use causes early school leaving), or by underlying factors that are overlapping between both variables.

Figure 3 presents the results of the co-twin control analysis, comparing the ORs for early school leaving between early-onset cannabis users versus those who did not use cannabis before the age of 18. In the unrelated sample, early-onset cannabis users had a 2.73 (95% Confidence intervals [CI]: 2.04–3.65) increased odds for early school leaving. This elevated risk was not present for discordant DZ and MZ twins - their ORs were close to 1 (DZ twins: OR [95% CIs] = 0.90 [0.37–2.20], MZ twins: 0.92 [0.41–2.05]), both significantly lower than the OR for unrelated individuals ($p<0.05$). This pattern of ORs is not consistent with cannabis use causing early school leaving. Rather, this pattern is most consistent with overlapping shared environmental factors that increase the likelihood of both early-onset cannabis use and early school leaving.

4. DISCUSSION

The aim of this study was to examine whether the known relationship between early-onset cannabis use and low educational attainment (as indexed by early school leaving) is more likely due to direct causality or to genetic or shared environmental factors underlying both variables. Using a bivariate twin model we found a significant phenotypic correlation between the two variables that was due to familial influences. The observed pattern of ORs in the co-twin control design suggests that this relationship is most likely due to shared environmental factors influencing both vulnerability to early cannabis use and early school leaving. This finding would imply that the leading explanation that adolescent cannabis use causes low educational attainment (e.g. Fergusson and Horwood, 1997) is not correct.

Our results are consistent with the findings from Bergen et al. (2008) and Grant et al. (2012) who showed that the relationship of illicit drug abuse/dependence and cannabis use, respectively, with educational attainment is not due to a causal mechanism but rather to overlapping familial liability. However, Bergen et al. (2008) found that this familial liability is most likely of genetic origin, while we found that a role for overlapping shared environmental influences is more likely. However, due to the wide CIs around the ORs we are unable to rule out the role of underlying genetic factors, and based on the estimates from our bivariate genetic model it is likely that both genetic and shared environmental factors underlie the relationship between both variables.

Our results have implications for the prevention and intervention of cannabis use and early school leaving. Early identification of at-risk adolescents could reduce both school drop-out and cannabis use, so it is important to identify the specific risk factors. Potential shared environmental risk factors may include family factors, such as parenting style (parental monitoring, support, communication), parental knowledge, family dysfunction, and parental substance use and deviant behaviour (Bohnert et al., 2012; Cohen and Rice, 1997; Ensminger and Slusarcick, 1992; Fergusson et al., 2003; Korhonen et al., 2008; Swadi, 1999). Other potential risk factors may relate to neighbourhood, school characteristics or peer influences (Battin-Pearson et al., 2000; Croninger and Lee, 2001; Huizink et al., 2010; Kuntsche and Jordan, 2006). In a 25-year longitudinal study, Fergusson et al. (2003) showed that accounting for socio-demographic factors, family functioning, parental problem behaviour, and various personal characteristics attenuated the association between cannabis use and early school leaving.

Our findings are probably not specific to cannabis use and early school leaving. Cannabis use is associated with use of other substances, conduct disorder, and risky behaviour, and these relationships (whether causal or due to overlapping liability) have been shown to be partly due to overlapping shared environmental and genetic influences (Button et al., 2006; Kendler et al., 2003; Korhonen et al., 2012; Miles et al., 2001). It is therefore plausible that an overlapping set of (environmental) risk factors (such as social disadvantage, family dysfunction, low parental support, parental substance use, and peer affiliations) is associated with a wide range of adverse social outcomes, including substance use and reduced educational attainment, but also delinquency, risky sexual behaviour and psychiatric disorder. This suggests that identification of overlapping risk factors will not only aid prevention and intervention efforts targeted at cannabis use and school drop-out, but will be applicable to a broad range of negative outcomes. For example, it is known that low parental monitoring is associated with cannabis and other substance use (Martins et al., 2008; Tragesser et al., 2007) as well as reduced educational attainment (McNeal, 1999), so guiding and advising parents about their parenting style and stimulating their level of monitoring and involvement could reduce the risk of a broad range of negative outcomes.

Our findings should be interpreted with caution. While the co-twin control design is one of the best methods to distinguish between causal and non-causal relationships, there are some limitations that should be kept in mind. Although the pattern of ORs seems to clearly point toward an overlapping shared environmental liability, the broad CIs around the ORs mean that we cannot rule out the possibility that the relationship is due to shared genetic liability. And of course, it is also possible that a combination of the described mechanisms as well as other mechanisms (e.g. gene-environment interactions and correlations) underlie the relationship between early-onset cannabis use and educational attainment, especially as the bivariate twin model suggests a role for both genetic and shared environmental factors.

Despite the large sample size, there was insufficient power to disentangle genetic and shared environmental sources of variance and covariance between the traits in the bivariate model. The use of dichotomous traits, combined with the relatively low frequency of high school drop-out also decreased our power.

There are also several limitations of our data. First, they are based on retrospective self-report, which can be influenced by social desirability and is subject to recall bias. The latter could explain the significant age effect on prevalence of early-onset cannabis use, as older participants might less well remember the age of initiation.

Also, our measure of early school leaving is based on participants' highest educational attainment and will therefore not capture individuals as early leavers who left high school but continued their education at a later age (although it is unlikely that this is a substantial percentage of people). Vice versa, some participants who may have decided to leave high school at a legitimate age to start working, do an apprenticeship or engage in other education than the ones mentioned in the questionnaire will have been classified as early school-leavers in this research.

The operationalisation of early-onset cannabis use is also noteworthy; never users were combined with those who initiated at age 18 or later and no distinction was made between those that used cannabis a few times during high school and regular or heavier users, which precludes capturing likely effects such as heavier use being more strongly associated with reduced educational attainment. Also, we cannot rule out that there may be individuals who left school prior to the initiation of cannabis use.

Notwithstanding these considerations, our findings suggest that cannabis use does not directly cause early school leaving, but rather that both traits are influenced by the same family environmental factors.

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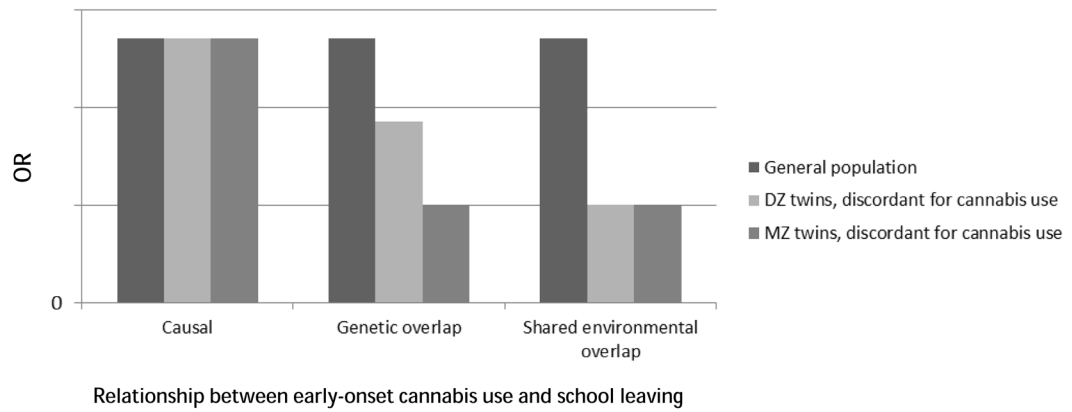


Figure 1. Expected pattern of odds ratios when the relationship between early onset cannabis use and school leaving is causal, or due to overlapping genetic or shared environmental influences. Results are shown for the general population, and discordant dizygotic (DZ) and monozygotic (MZ) twins.

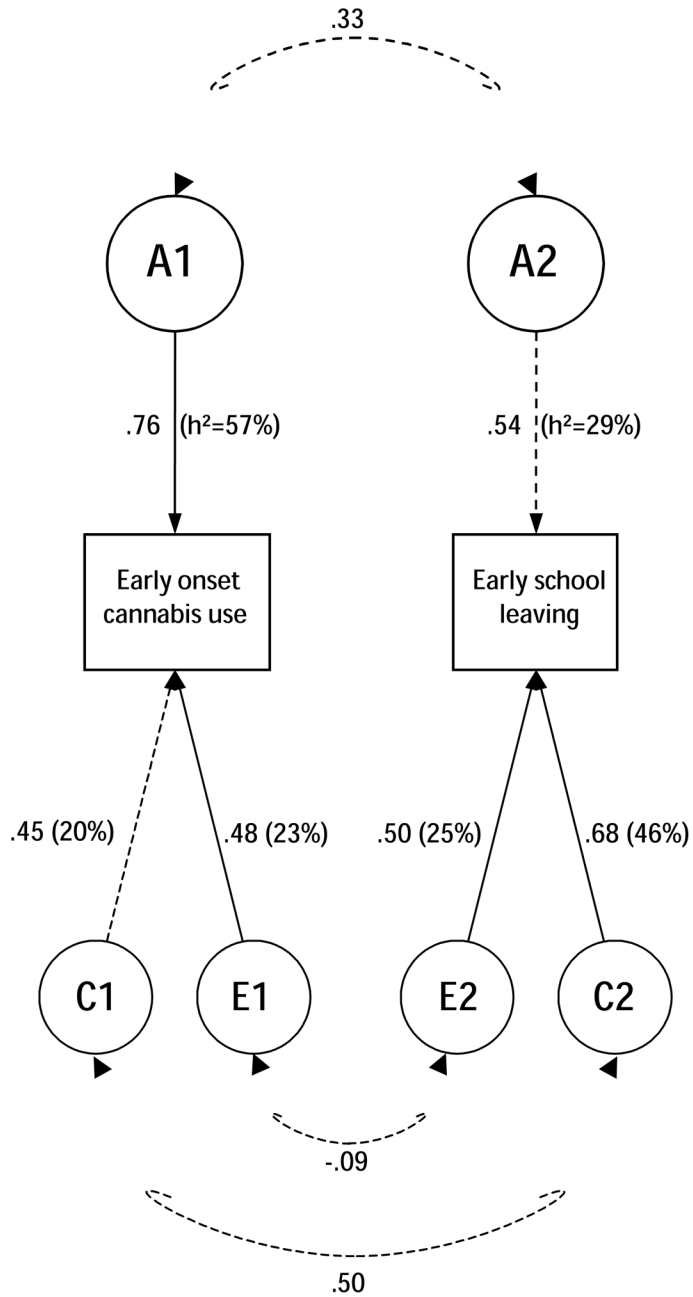


Figure 2. Correlated factor model. Graphical presentation of the parameter estimates and proportions of variance in early-onset cannabis use and early school leaving accounted for by additive genetic (A), shared environmental (C) and residual influences (E). h² (heritability) is the percentage of variance accounted for by genetic factors. The double-headed arrows represent the genetic, shared environmental and residual correlations, indicating the degree to which the same genetic or environmental factors are influencing the two traits. Broken lines represent non-significant paths (p>=0.05).

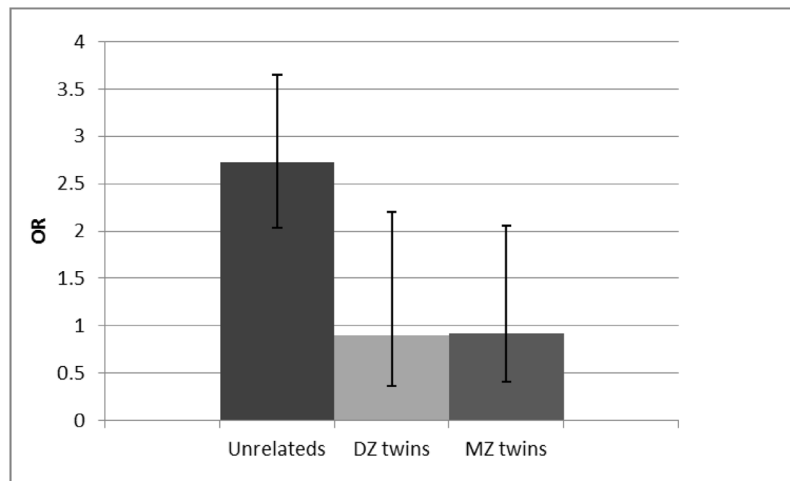


Figure 3. Observed odds ratios for and early school leaving as compared between individuals who used cannabis before the age of 18 and those who did not. Bars show results for unrelated individuals, and DZ and MZ twins discordant for adolescent cannabis use. Error bars denote the 95% confidence intervals.

Table 1

Prevalences of highest level of educational attainment, early school leaving, lifetime cannabis use, and cannabis use before the age of 18, for males and females separately.

	Males N=1173 (35%)	Females N=2164 (65%)
Primary school – incomplete	0 (0.0%)	1 (0.0%)
Primary school – complete	0 (0.0%)	1 (0.0%)
Year 8 complete	3 (0.3%)	6 (0.3%)
Year 9 complete	10 (0.9%)	11 (0.5%)
Year 10 complete	76 (6.5%)	102 (4.7%)
Year 11 complete	61 (5.2%)	82 (3.8%)
Year 12 complete	160 (13.6%)	301 (13.9%)
TAFE/Technical college	369 (31.5%)	573 (26.5%)
Undergraduate degree	296 (25.2%)	627 (29.0%)
Post-graduate degree	198 (16.9%)	460 (21.2%)
Early school leaving	150 (12.8%)	203 (9.4%)
Lifetime cannabis use	898 (76.6%)	1404 (64.9%)
Cannabis use initiated before the age of 18	525 (44.8%)	712 (32.9%)

Table 2

Tetrachoric twin pair correlations (and 95% confidence intervals) for cannabis use and early school leaving by zygosity.

Zygosity	N pairs	Cannabis use before the age of 18		Early school leaving	
		Tetrachoric twin pair r (95% CI)	MZ versus DZ Twin pair r (95% CI)	Tetrachoric twin pair r (95% CI)	MZ versus DZ twin pair r (95% CI)
MZ F	399	0.80 (0.71; 0.87)		0.76 (0.59; 0.88)	0.76 (0.62; 0.86)
MZ M	172	0.71 (0.55; 0.83)	0.77 (0.69; 0.83)	0.76 (0.47; 0.91)	
DZ F	302	0.58 (0.42; 0.71)		0.64 (0.41; 0.80)	
DZ M	120	0.31 (0.03; 0.55)	0.46 (0.35 – 0.56)	0.52 (0.09; 0.80)	0.60 (0.44; 0.72)
DZ OS	231	0.38 (0.18; 0.56)		0.58 (0.31; 0.77)	

F=female, M=male, OS=opposite-sex, MZ =monozygotic, DZ= dizygotic, CI= confidence interval, MZ versus DZ =pooled MZ and DZ twin pair correlations (i.e. equated across sexes).

Table 3

Goodness-of-fit statistics for bivariate models of early-onset cannabis use and early school leaving.

Model	versus	$\Delta\chi^2$	Δdf	P-value
1 General ACE model				
2 Drop phenotypic correlation	1	51.27	3	<0.001
3 Drop genetic correlation (r_g)	1	1.04	1	0.31
4 Drop shared environmental correlation (r_c)	1	1.79	1	0.18
5 Drop residual correlation (r_e)	1	0.28	1	0.60
6 Drop genetic and shared environmental correlations (r_g and r_c)	1	36.02	2	<0.001
7 Drop A influences on early-onset cannabis use	2	20.51	1	<0.001
8 Drop C influences on early-onset cannabis use	2	2.43	1	0.12
9 Drop A influences on school leaving	2	2.92	1	0.09
10 Drop C influences on school leaving	2	6.69	1	<0.01

A=additive genetic influences, C=shared environmental influences, E=residual influences