



Self-control and its relation to joint developmental trajectories of cannabis use and depressive mood symptoms

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ABSTRACT

Background: Cannabis use and depressive mood symptoms in adolescence have been found to co-occur. In exploring the nature of this relationship and in the search for mechanisms that explain this link, scholars have postulated the idea for a 'common liability model'. According to this model, the link between cannabis use and depressive symptoms can be explained by an underlying risk factor. One important candidate for this underlying risk factor may be self-control, as a reflection of immature self-regulatory systems in adolescence. In the present study, we will test the extent to which joint development of cannabis use and depressive symptoms can be explained as an expression of self-control.

Methods: A total of 428 adolescents participated in a five-wave longitudinal design. Main study outcomes were self-reports of self-control (age 12) and cannabis use and depressive symptoms (ages 12–16).

Results: We established six trajectories of joint development of cannabis use and depressive symptoms. Conditional probabilities indicated that cannabis use and depressive symptoms were symmetrically related. Levels of self-control were lowest for adolescents following the joint developmental pathway of cannabis use and high depressive symptoms.

Conclusions: Low levels of self-control are predictive of joint development of cannabis use and depressive symptoms. Future studies should concentrate on the role of self-control in co-occurrence of other health risk behaviors and on psychological and physiological mechanisms underlying self-control and its relation to co-occurrence.

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1. Introduction

Developmental roots of a variety of lifetime problems are generally set in adolescence (Dahl, 2004), a period that is marked by the emergence of several changes on multiple domains. One of these domains concerns the development of adolescent risk behavior, among which experimentation with substance use. From a more internalizing perspective, another domain concerns the development of emotions and affect. In some cases, changes across different domains follow a parallel development and appear together. In the present study, we focus on the joint development of cannabis use and depressive mood symptoms. In exploring the nature of the relationship between cannabis use and depressive symptoms, and in the search for mechanisms that explain this link, scholars have postulated the idea for a 'common liability model'. In accordance with this model, we will test the extent to which joint development of cannabis use and depressive symptoms can be explained

as an expression of a common underlying risk factor (Marmorstein et al., 2010). Since immature self-regulatory mechanisms, reflected in deficits in self-control have been found related to externalizing behaviors (Shedler and Block, 1990) and internalizing problems or emotional distress (Davey et al., 2008), self-control may be an important candidate for the common underlying risk factor in the relationship between cannabis use and depressive symptoms.

1.1. Co-occurrence of cannabis use and depressive symptoms

Cannabis users have been identified as an at-risk group for impaired emotional functioning (Dorard et al., 2008) and psychosocial and mental health difficulties (Arseneault et al., 2002; Fergusson et al., 2002; Moore et al., 2007). Some studies found evidence suggesting that cannabis use may increase depressive symptoms (Bovasso, 2001; Holden and Pakula, 1998; Degenhardt et al., 2003), by certain physiological mechanisms or related effects on interpersonal functioning (Bovasso, 2001; Dorard et al., 2008; Holden and Pakula, 1998; Degenhardt et al., 2003). With respect to physiological mechanisms that may underlie the relationship between cannabis and depressive symptoms, psychotropic prop-

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erties of cannabis may increase levels of interferongamma that indirectly augment the synthesis of serotonin (Holden and Pakula, 1998). Regarding effects on interpersonal functioning, cannabis use has been found to interfere with adolescents' social relationships (Brook et al., 1989).

A second group of studies found support for the 'secondary substance use disorder model' that posits that mental health problems increase the risk for substance use. Regarding depressive symptoms or dysthymia, cannabis has been found to have a self-medicating function, producing mood altering symptoms (D'Souza et al., 2004; Renault et al., 1974).

Finally, there is the possibility that cannabis use may not be causally related to depressive symptoms at all, as it was shown by Fergusson and Horwood (1997) and by Boys and Marsden (2003). Here, accordingly with a 'common liability model', the comorbidity rates of depressive symptoms and cannabis use can be explained as an expression of a common underlying risk factor. One important candidate for this underlying risk factor in the common liability model may be self-control.

1.2. Self-control

A major source of problems in adolescence is related to difficulties in the control of behavior and regulation of emotions (Dahl, 2004). These difficulties are partially due to the fact that specific regions of the brain implicated with self-regulation are still in progress (Giedd, 2004). Specifically, it is during this specific period that functional and structural development of subcortical and prefrontal regions in the brain take place, regions in the brain associated with self-regulatory control (Galvan et al., 2006). Self-control can be referred to as a person's capacity to (a) inhibit socially unacceptable and undesirable impulses and (b) alter and regulate one's behavior, thoughts, and emotions (Baumeister et al., 1994; Muraven and Baumeister, 2000; Tangney et al., 2004). Individual differences in self-regulatory control have been found to increase engagement in risk-taking (Magar et al., 2008). In particular, individuals who show lower levels of self-control have been found to be more delinquent (De Kemp et al., 2009) and to be more at-risk to use alcohol, tobacco, and cannabis (Wills and Stoolmiller, 2002). Here, self-control may entail a coping component: individuals with low self-control may be incapable to inhibit impulsivity, which is a marker for substance use (Shedler and Block, 1990; Verdejo-García et al., 2008).

Besides its relation to substance use, recent studies have found support for the assumption that an immature self-regulation mechanism reflected in low self-control is also associated with emotional problems such as low self esteem (Finkenauer et al., 2005) and depression (Kaslow et al., 1988). Specifically, a recent study by Davey and colleagues provided an overview of models that aim to explain how the vulnerability for depressive symptoms arises in adolescence (Davey et al., 2008). One of these models postulates that an increase in depression/depressive symptoms during adolescence is due to an ineffective social information processing network. Particularly, yet-to-mature self-regulatory mechanisms may be incapable to process important social stimuli adequately, which in turn may precipitate depressive symptoms (Nelson et al., 2005). According to a second model, depression or altered depressive symptoms in adolescence are a result of decreased approach and increased risk avoidance (Ernst et al., 2006). Finally, Forbes and Dahl argue that depression or depressive symptoms in adolescence may be due to development of the neurological systems that underlie reward in adolescence (Forbes and Dahl, 2005). All these models refer to the immature self-regulatory system and the mismatch between different systems as a consequence of this immaturity. In other words, the underlying process here refers to the incapability of individuals low on self-control to influence their emotions

and thoughts, and therefore an increased risk to develop depressive symptoms.

Hence, both cannabis use as well as depressive mood symptoms in adolescence can possibly be subscribed to immaturity of self-regulatory systems and mismatches of different systems which is typical for adolescence.

1.3. The present study

The present study responds to the need for more research on co-occurrence of behavior and potential explanatory factors. Specifically, it concentrates on the role of self-control in the joint development and co-occurrence of cannabis use and depressive mood symptoms in an at-risk age group. We will concentrate on those adolescents who show cannabis use and report elevated depressive symptoms. We will use longitudinal data and because we concentrate on an early age group, we will focus on robust profiles of cannabis use. A previous study that concentrated on trajectories of cannabis use in early adolescence found three trajectories (early onset – late onset – non-users). The late onset group was a split-off from the non-users trajectory at around age 15 (Flory et al., 2004). In the present study we will also expect two or three developmental pathways of cannabis use: a large group of respondents that has hardly developed any use of cannabis, and one or two expectantly smaller groups that report different levels of frequency of use. Regarding depressive symptoms, we expect three or four robust profiles of development of depressive symptoms (for three trajectories see Brendgen et al., 2010, for four trajectories see Rodriguez et al., 2005). Specifically, we expect one group that shows consistently low levels of depressive symptoms, one large group that shows medium levels of depressive symptoms, and one or two smaller groups that show consistently high or increasing levels of depressive symptoms. By combining these trajectories we will create trajectories of joint development of cannabis use and depressive symptoms. If self-control functions as a common liability factor in the joint development of cannabis use and depressive symptoms, those individuals who follow the joint trajectory illustrated by high levels of cannabis use and high levels of depressive symptoms would show lowest levels of self-control. In other words, whereas other studies assume that there are causal relationships between cannabis use and depressive symptoms, we postulate that the joint development of cannabis use and depressive symptoms can be explained by one underlying factor that is known to be important in adolescence (i.e., self-control).

We analyzed the data to (i) establish separate developmental trajectories of cannabis use and depressive symptoms; (ii) to establish joint developmental trajectories of cannabis use and depressive symptoms; and (iii) to determine if we could discriminate subgroups of joint developmental trajectories of cannabis use and depressive symptoms by using indicators of self-control accordingly with a common liability model. We thus examined self-control in relation to subgroup differences in the development of cannabis use and depressive symptoms in adolescence.

2. Methods

2.1. Sample

Participants were from the Family and Health Study, a prospective study among 428 families that were selected from registers of 22 municipalities in the Netherlands (e.g., Harakeh et al., 2005; Van der Vorst et al., 2005). The families were visited in their homes by interviewers. To maintain confidentiality, questionnaires were filled out in private by each family member. Participants were asked not to discuss the questionnaire with other family members. In the present study, we use data from five waves with 1-year interval of the youngest child in the family. At year 1, participants for this study were between 13 and 15 years ($M = 13.36$, $SD = .50$). The distribution of males and females was almost equal. More than 95% of the family members were of Dutch origin. With respect to education, adolescents were equally

divided over three educational levels; one-third of the respondents followed special or lower education, one-third followed intermediate, general education, and the remaining group followed the highest level of secondary school in the Netherlands (preparatory college and university education). Parental consent was obtained for all adolescents who participated and the study was approved by a local ethics committee.

2.2. Measures

2.2.1. Self-control. To assess self-control, a Dutch translation of the self-control scale developed by Tangney, Baumeister, and Boone was employed (2004) that showed adequate psychometric qualities (DuBois et al., 1995; Kuijter et al., 2008; Mathews et al., 2007). We used a short version of the original scale (De Kemp et al., 2009; Finkenauer et al., 2005). The self-control scale aims to assess people's ability to control their impulses, alter their emotions and thoughts, and to interrupt undesired behavioral tendencies and refrain from acting on them. On a five-point scale ranging from 1 *not at all* to 5 *very much*, participants were asked to indicate the extent to which items applied to them. Examples items are: "I am lazy", "I have a hard time breaking bad habits", "I wish I had more self-discipline", and "I have trouble concentrating", "I change my mind fairly often" ($M=2.26$, $SD=.54$). The alpha was sufficient (11 items, $\alpha=.75$) and in line with other studies (De Kemp et al., 2009; Finkenauer et al., 2005).

2.2.2. Cannabis use. Information was collected using self-reports at each assessment point following two items: (1) have you ever used cannabis (0 = Yes, 1 = No); (2) How many times have you used cannabis during the last 4 weeks (1 = Not, 2 = 1–2 times, 3 = 3–4 times, 4 = 5 times or more). We created a composite score, ranging from 0 to 4, which represented the frequency of cannabis use for each data collection wave (0 = Never used, 1 = Used, but not during the last 4 weeks, 2 = 1–2 times during the last 4 weeks, 3 = 3–4 times during the last 4 weeks, 4 = 5 times or more during the last 4 weeks (M (SD 's): .10 (.47), .24 (.81), .34 (.89), .43 (1.00), .51 (.98)) (Monshouwer et al., 2006).

2.2.3. Depressive mood symptoms. To assess the extent to which adolescents experience negative moods, the 6-item depressive mood list developed by Kandel and Davies (1982, 1986) was used in its Dutch translation (Dékovic, 1996; De Vries et al., 2003). The depressive mood list is extensively used in adolescent surveys (Engels et al., 2001; Compas et al., 1993; Otten et al., 2009) and showed sufficient psychometric properties in terms of internal consistency, reliability and stability over time (Van den Eijnden et al., 2008). Respondents were asked to report the frequency of experienced negative feelings over the last 12 months. Studies have shown that depressive symptoms are rather stable (Kendall et al., 1989). On a five-point scale items asked how often they felt unhappy, sad, or depressed and how often they felt nervous or tensed. Cronbach's alpha's for the depressive mood list used in the present study were consistently $>.80$ at each point of measurement (M (SD 's): 2.47 (.64), 2.54 (.71), 2.53 (.70), 2.31 (.72), 2.30 (.71)).

2.3. Attrition and missing data

Of the original dataset 98% of the cannabis use data were available at T2; 92% at T3; and 71% of the data were available at T4 and T5. At T2, complete depressive symptoms data was available for 97% of the original data, 94% at T3, 79% at T4 and 71% at T5. To make use of all available data participants with at least one data point on cannabis use and depressive symptoms were allowed in the trajectory analyses (99.3%). Participants who had missings on either cannabis use data or depressive symptoms data on one of the five measurement points were likely to be lower educated than those respondents for who complete data over all time five points were available.

2.4. Analyses

After presenting descriptive data on trends in cannabis use and depressive symptoms across the study age range, the analyses proceeded in three steps. In step 1, in preparation of the joint trajectory analysis of cannabis use and depressive symptoms, models for the developmental trajectories were separately estimated for cannabis use and depressive symptoms. We used growth mixture models (GMMs) to estimate the trajectories in Mplus Version 4.1 (Muthen and Muthen, 1998–2003). GMMs are designed to identify clusters of individuals who follow unique developmental trajectories, each of which may reflect distinct etiologies and/or outcomes. These clusters are identified on the basis of growth parameters such as intercepts (starting values) and slopes (linear or quadratic growth). Missing data were handled through Full Information Maximum Likelihood (Enders and Bandalos, 2001). To account for the non-normal distributions of the cannabis use scores, we used censored data for the clustering of the scores at the scale minimums (i.e., a preponderance of zeros). A series of models was fitted beginning with a one-trajectory model and moving to a four-trajectory model. There are different ways to decide the choice for the correct number of trajectories. First, it can be decided by inspection of the BIC (the Bayesian Information Criterion), a measure that balances the fit of the model, with lower values indicating a more parsimonious model. Second, one can also look at the entropy that indicates the precision of group assignment, with values

Table 1
Mean levels of cannabis use and depressive symptoms.

Variable	Boys		Girls		Total	
	Mean	SD	Mean	SD	Mean	SD
Cannabis use						
Wave 1	.12	.52	.08	.41	.10	.47
Wave 2	.32	.94	.17	.66	.24	.81
Wave 3	.48	1.09	.22	.64	.34	.89
Wave 4	.63	1.26	.25	.65	.43	1.00
Wave 5	.68	1.13	.35	.79	.51	.98
Depressive symptoms						
Wave 1	2.35	.63	2.57	.64	2.47	.64
Wave 2	2.37	.70	2.69	.68	2.54	.71
Wave 3	2.38	.70	2.66	.68	2.53	.70
Wave 4	2.19	.71	2.42	.72	2.31	.72
Wave 5	2.16	.69	2.43	.70	2.30	.71

closer to 1 indexing greater precision (range 0–1). Finally, it is important to take the utility of classes into account. This can be based on the knowledge about a certain development, and the fact that classes need to have reasonable numbers of participants (Jordan et al., 2006) and the extent to which these numbers are theoretically meaningful (Muthen, 2003).

In step 2, the joint trajectories of depressive symptoms and cannabis use were estimated. We used the most robust trajectory models for depressive symptoms and cannabis use as the starting point for the joint models. Key outputs of a joint model are the joint probabilities and the conditional probabilities. Joint probabilities of belonging to trajectories of depressive symptoms and cannabis use (e.g., the probability of following chronic depressive symptoms and chronic cannabis use trajectories), and conditional probabilities (e.g., the probability of following a high depressive symptoms trajectory conditional on following a high cannabis use trajectory) are useful for describing the developmental overlap between two types of distinct but related phenomena (Barker et al., 2007).

In step 3, we classified the adolescents based on their probabilities of belonging to the different joint trajectories and examined mean differences between the joint trajectories in self-control. The extent to which self-control could differentiate the joint trajectories was tested with multinomial and logistic regression analyses with weighted data. When data are weighted, each participant is represented in each cell as a function of his or her probability of being assigned to that joint trajectory group. This approach preserved the continuous nature of the classification variable and corrected for potential uncertainty in trajectory assignment.

3. Results

Table 1 shows the prevalence of cannabis use and depressive symptoms at each wave for the total group and for boys and girls separately. Overall cannabis use increased over time. At age 13 only 5% had used cannabis at some point in time, at age 14 this was 10.5%, at age fifteen 17.9%, at age sixteen 22.5%, and at age seventeen 29.3% of the respondents had used cannabis at some point in time. Overall, depressive symptoms remained stable over time.

3.1. Trajectory models of cannabis use and depressive symptoms

The segregate trajectories for cannabis use and depressive symptoms are shown in Figs. 1 and 2, respectively. Both trajectories show a slight decline in the end which may be due to some selec-

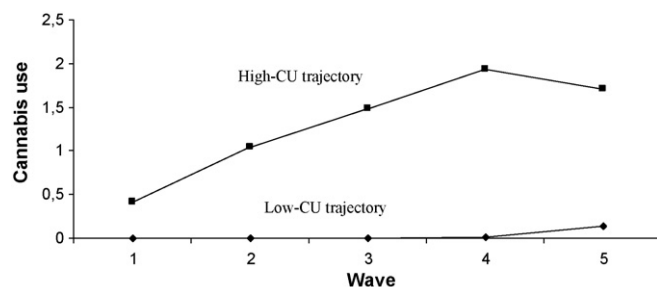


Fig. 1. Longitudinal profiles of cannabis use (CU) over five measurements (with 1-year intervals).

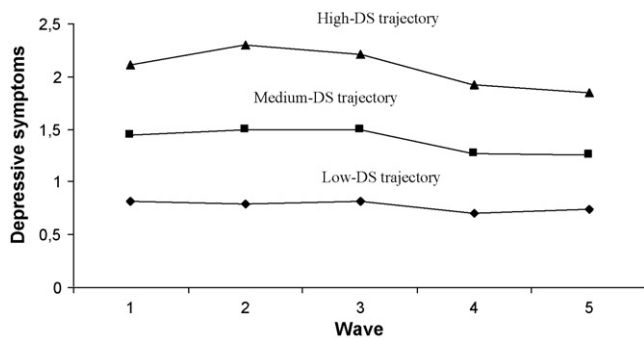


Fig. 2. Longitudinal profiles of depressive symptoms (DS) over five measurements (with 1-year intervals).

tiveness in the attrition/retention. In our study, boys were more likely to score higher on cannabis use than girls, and girls were more likely to score higher on depressive symptoms than boys; however, the shape of the trajectories for both groups was similar. Therefore, we analyzed the data on the total sample, controlling for the confounding effect of sex.

The fit measures indicated that respectively a three-group model for cannabis use (BIC=1863.35; entropy=.93) and a four-group model for depressive symptoms (BIC=3428.88; entropy=.75) fitted slightly better to the data than a two-group model (BIC=1944.88; $p=.00$; entropy=.90) and a three-group model (BIC=3464.06; entropy=.72). However, we have deliberately chosen for the two-trajectory solution of cannabis use and a three-trajectory solution for depressive symptoms. Specifically, we aimed at combining the most robust developmental trajectories of cannabis use and depressive symptoms. Combining a three-group model with a four-group model would lead to 12-joint trajectories, with some joint combinations comprised of only few or no respondents. One criterion for the decision for a correct number of trajectories is that each class needs to have a reasonable number of children to be theoretically meaningful (Muthen, 2003). Hence, we decided to choose the more robust solution (i.e., a two-trajectory solution of cannabis use and a three-trajectory solution for depressive symptoms). The argument that a model with a smaller number of groups would imply more robustness was supported by the fact that the second and the third trajectory for cannabis use in the three-group model were parallel and as a sum included the exact number of respondents as in the second trajectory of the two-group model (this was also found for depressive symptoms). The choice for two groups with respect to cannabis use would also be in line with Moffitt's theory of antisocial behavior (1993). Specifically, according to this theory there are two pathways of antisocial behavior: a life-course persistent pathway and an adolescent-limited pathway. Individuals that follow the life-course persistent pathway are likely to show antisocial behavior that begins in childhood and persists into adolescence and adulthood. Those who follow the adolescent-limited pathway start to show antisocial behavior in mid-adolescence (around age 15), however, here it is limited to adolescence and therefore considered to be more normative. Finally, there is a third group that does not engage in antisocial behavior. The two-trajectory solution for cannabis use may reflect a similar development with a relatively small group engaging in cannabis use and a larger group that consists out of abstainers and late starters (at around age fifteen: the adolescence-limited group).

Regarding cannabis use, individuals in the low trajectory were not engaged in cannabis use or rarely so (76%), with no significant intercept, slope, or quadratic trend (estimates based on censored data: intercept = -13.709 , $SE = 12.972$, $p = .291$; slope = 5.322 , $SE = 6.166$, $p = .388$; quadratic trend = $-.593$, $SE = .746$, $p < .427$). A proportion of 24% of the sample followed the increasing cannabis

use trajectory (intercept = -2.407 , $SE = .898$, $p < .0001$; slope = 2.671 , $SE = .491$, $p < .0001$; quadratic trend = $-.414$, $SE = .08$, $p < .0001$).

For depressive mood symptoms, the lowest trajectory included 25% of the sample and represented those who hardly reported having experienced depressive symptoms over the last 12 months (intercept = $.813$, $SE = .14$, $p < .0001$; slope = $-.009$, $SE = .05$, $p = .863$; quadratic trend = $-.004$, $SE = .02$, $p = .802$). The second trajectory included the largest part of the sample (50%) which represented those who reported overall medium levels of depressive symptoms (intercept = 1.460 , $SE = .16$, $p < .0001$; slope = $.062$, $SE = .04$, $p = .126$; quadratic trend = $-.031$, $SE = .004$, $p < .01$). The highest depressive symptoms trajectory identified 25% of the sample (intercept = 2.142 , $SE = .10$, $p < .0001$; slope = $.153$, $SE = .06$, $p < .009$; quadratic trend = $-.061$, $SE = .01$, $p < .0001$). These findings are in line with the idea that depressive symptoms, rather than symptoms that belong to a clinical depression, are rather common and their prevalence follows a relatively normal distribution, with most adolescents reporting depressive symptoms to some extent, and less individuals scoring relatively high or low.

3.2. Step 2: joint trajectories of cannabis use and depressive mood symptoms

The top part of Table 2 shows the joint probabilities of trajectory membership. In this part of the table, probabilities across all cells sum to 1. Most individuals scored low on cannabis use and followed the medium trajectory of depressive symptoms (38.3% of the sample). Most important for our study, the sixth and most at-risk trajectory describes the developmental overlap between two types of distinct but related phenomena and consisted of individuals who scored positive on cannabis use and high on depressive symptoms (8.2% of the sample).

3.2.1. Conditional probabilities of depressive symptoms given cannabis use. The middle part of Table 1 contains the likelihood of following one of the depressive symptoms trajectory groups conditional on membership in the cannabis use trajectory. Most importantly, the likelihood to follow the high depressive symptoms trajectory was higher for those following the high cannabis use trajectory than for those following the low cannabis use trajectory (.35 versus .24, respectively).

3.2.2. Conditional probabilities of cannabis use given depressive symptoms. The third part of the table contains the likelihood of following one of the two cannabis use trajectory groups conditional on membership in one of the depressive symptoms trajectories. Here, most importantly, the likelihood to follow the cannabis use trajectory was higher for those who followed the high depressive symptoms trajectory than for those who followed the medium or low depressive symptoms trajectories (.31 versus .18 and .21, respectively).

Table 3 presents the proportion of boys and girls in the trajectory groups as well as the results of a logistic regression analyses predicting membership with gender. Boys were more likely than girls to follow joint trajectory 2 (low cannabis–medium depressive symptoms), group 4 (high cannabis–low depressive symptoms), and group 5 (high cannabis–medium depressive symptoms). Girls were more likely than boys to follow group 3 (low cannabis–high depressive symptoms). There were no significant differences in proportions of boys and girls following group 1 (low cannabis–low depressive symptoms) and group 6 (high cannabis–high depressive symptoms).

Table 2
Joint and conditional probabilities of depression and cannabis use trajectories.

Cannabis use	Depressive symptoms		
	Low	Medium	High
Probabilities of trajectory groups membership ^a			
Low	(1) 0.202	(2) 0.383	(3) 0.182
High	(4) 0.046	(5) 0.105	(6) 0.082
Probabilities of depressive symptoms conditioned on cannabis use ^b			
Low	(1) 0.264	(2) 0.500	(3) 0.237
High	(4) 0.196	(5) 0.452	(6) 0.352
Probabilities of cannabis use conditioned on depressive symptoms ^c			
Low	(1) 0.816	(2) 0.784	(3) 0.689
High	(4) 0.184	(5) 0.216	(6) 0.311

^a Cells sum to 1.^b Rows sum to 1.^c Columns sum to 1.**Table 3**
Between group proportions of boys and girls.

Joint trajectory groups	Boys		Girls		Odds ratio
	N	%	N	%	
1. Low CU–Low DS	72	35.3	79	41.1	1.26
2. Low CU–Medium DS	50	24.5	37	16.5	.61*
3. Low CU–High DS	26	12.7	52	23.2	2.08***
4. High CU–Low DS	29	14.2	16	7.1	.46*
5. High CU–Medium DS	15	7.4	4	1.8	.25**
6. High CU–High DS	12	5.9	23	10.3	1.85

Note: CU = cannabis use and DS = depressive symptoms.

* $p < .05$.** $p < .01$.*** $p < .001$.

3.3. Step 3: self-control, cannabis use, depressive symptoms

First, we calculated self-control scores for the separate trajectories of cannabis use and depressive symptoms. Regarding cannabis, the mean scores for individuals in the low and the high trajectory were, respectively, 2.28 ($SD = .53$) and 2.19 ($SD = .57$). With respect to depressive symptoms, the mean scores for individuals in the low, medium, and high trajectory were respectively 2.27 ($SD = .49$), 2.56 ($SD = .50$), and 1.96 ($SD = .51$). The mean scores of self-control for the joint trajectories were 2.26 ($SD = .47$) (low cannabis–low depressive symptoms), 2.30 ($SD = .55$) (high cannabis–low depressive symptoms), 2.56 (SD 's = .51 and .49) (both low and high cannabis and medium depressive symptoms), 2.02 ($SD = .52$) (low cannabis–high depressive symptoms) and 1.85 ($SD = .47$) (high cannabis–high depressive symptoms). Note that regarding the joint development of cannabis use and depressive symptoms, the self-control level for those individuals following the joint developmental pathway of cannabis use and high depressive symptoms was lower than the lowest levels of self-control in the separate trajectories ($M = 1.85$, $SD = .47$).

Consequently, multinomial regressions analyses were used to predict joint trajectory group membership from self-control measured at measurement 1. Sex and age were entered as covariates.

In this study we are interested in individuals who follow the high cannabis-high depressive symptoms developmental trajectory, so we concentrated on the most important comparisons with this specific trajectory. Table 4 shows that lower scores on self-control increase the odds of following the high cannabis–high depressive symptoms trajectory as compared to one of the other trajectories with factor 4.30.¹ Results show that lower scores on

self-control increase the odds of following the low cannabis–high depressive symptoms trajectory with factor 2.83 compared to other trajectories. Finally, although the mean scores appeared different (2.02 vs. 1.85), the difference on self-control between participants following the low cannabis–high depressive symptoms group and those following the high cannabis–high depressive symptoms trajectory was marginally significant (binomial probability for trend, $OR = 2.04$, $p = .09$), tentatively suggesting that self-control was stronger related to depressive symptoms than to cannabis use. Interactions with the effect of gender on the relationship between self-control and the joint trajectories of cannabis and depression failed to reach significance.

4. Discussion

The aim of this study was to look at the extent to which the joint development of cannabis use and depressive symptoms could be explained as an expression of a common underlying risk factor (i.e., self-control) (Marmorstein et al., 2010). Of the two trajectories for cannabis use, a majority included adolescents with no use or minimal levels of use and a smaller group of adolescents reporting high/increasing levels of cannabis use. This is in line with Moffitt's theory of antisocial behavior (1993) suggesting that there are two pathways of antisocial behavior: a life-course persistent pathway and an adolescent-limited pathway. The adolescent-limited pathway is normally a split-off of the non-antisocial group (in our case the abstainers group) that takes place during mid-adolescence. Regarding depressive symptoms, the largest proportion of adolescents experienced medium levels of depressive symptoms, one fourth of all respondents experienced high levels, and one fourth of

¹ To illustrate the robustness of the effects of self-control: the effects of self-control remained significant after correcting for other confounding factors than age

and sex (i.e., extraversion, emotional instability, smoking, delinquency and alcohol use).

Table 4
Multinomial regression model comparing joint trajectories.

Variable	Trajectory comparison								
	High CU–High DS vs. Others			Low CU–High DS vs. Others			Low CU–High DS vs. High CU–High DS		
	OR	95% CI	p-value	OR	95% CI	p-value	OR	95% CI	p-value
Age	1.47	0.72–3.00	.29	0.90	0.55–1.52	.70	1.43	0.64–3.17	.85
Girls	1.42	0.65–3.12	.38	1.80 [*]	1.05–3.10	.03	0.85	0.36–2.01	.71
Low self-control	4.30 ^{***}	2.06–9.02	.00	2.83 ^{***}	1.67–4.80	.00	2.04	0.89–4.70	.09 ^a

Note: CU = cannabis use, DS = depressive symptoms, OR = odds ratio, and CI = confidence interval.

^{*} $p < .05$.

^{***} $p < .001$.

^a Binomial probability for trend.

all respondents reported low levels of depressive symptoms during the years in the study. The joint analysis of the two sets of trajectories illustrated that one group consisting out of 8% of the total sample ($N = 35$) followed the highest trajectory for cannabis use and depressive symptoms, indicating co-occurrence. Moreover, accordingly with the common liability model, results showed that low levels of self-control are predictive of co-occurrence of cannabis use and depressive symptoms. Specifically, baseline levels of self-control were lowest in those adolescents who followed the joint developmental pathway of cannabis use and high levels of depressive symptoms. The level of self-control in the group of adolescents who engaged in cannabis use and who followed the pathway with high levels of depressive symptoms was also lower than self-control reported by adolescents in the low cannabis use trajectory but who followed the pathway with high levels of depressive symptoms, although here the difference was only marginally significant.

The present study extends knowledge of previous studies in a few ways. First, while most studies that look at co-occurrence and comorbidity with cross-sectional designs, the present study uses a relatively new methodology: i.e., joint developmental trajectories. This statistical technique allows to look at the joint development and co-occurrence of different behaviors and psychological phenomena. Second, this study is the first to show that the link between cannabis use and depressive symptoms can, at least to some extent, be explained by a common liability model as suggested by Marmorstein et al. (2010). While there are different candidates for a common liability factor, this study shows that self-control is a potentially important factor that may explain co-occurrence of cannabis use and depressive symptoms. Both cannabis use as well as depressive symptoms in adolescence can possibly be subscribed to the relative immaturity of self-regulatory systems and mismatches of different systems in adolescence. The fact that the self-control levels for those individuals following the joint developmental pathway of cannabis use and high depressive symptoms was lower than the lowest levels of self-control in the separate trajectories indicates that low levels of self-control or self-regulations are reflected in different developmental domains. Although it is likely that these effects are the result of immature self-regulatory mechanisms, additional studies are needed to test exactly which brain regions or systems are responsible for these effects. For instance, the effects may be a result of an ineffective information processing network (Nelson et al., 2005), a disturbed approach and avoidance system (Ernst et al., 2006), an immature reward system (Forbes and Dahl, 2005), or a combination of different systems.

Furthermore, it would be interesting to test the extent to which self-control plays a role in for instance co-occurrence of development in other domains of health risk behavior (e.g., alcohol use or binge eating). Moreover, in this study we concentrated on depressive symptoms as a reflection of emotional distress and imbalanced affect regulation. However, additional studies are needed to test

whether the effects are different for, for instance, anxiety which often occurs to overlap with depression/depressive symptoms (Ollendick et al., 2003), but may be differently related to self-control. Finally, it would be interesting to test the role of self-control in clinical samples.

4.1. Limitations

Some limitations of this study should be mentioned. First, all included variables were assessed with self-reported frequency which is prone to error and could in some cases have lead to under-report. Although measurement of cannabis use by physiological measures is also difficult due to variation in biologically available cannabinoids concentrations, a combination of both self-report and more objective measures (e.g., immunoassay screening and gas chromatography/mass spectrometry confirmation) (Buchan et al., 2002) would have provided more valid measures of cannabis use (Moore et al., 2007). A second limitation refers to the sample and sample size. In the first part of the analyses we calculated separate trajectories for both cannabis use and depressive symptoms. A larger sample size would have allowed us to look at more different developmental profiles of both cannabis use and depressive symptoms. As a consequence, one might argue that results regarding the effect of self-control could have been more precise and specific if we had a larger sample. For instance, it would have been interesting to see the predictive value of self-control in a joint developmental trajectory that consists of adolescents with a cannabis use disorder according DSM-IV criteria and clinical levels of depressive symptoms. On the other hand, while this group consisted of relatively young adolescents, it made more sense to assume two rather than three developmental trajectories of cannabis use: one group that experimented and tried cannabis use over time and one group that did show little use or no use at all. Third, there is discussion about the statistical analysis that we used (i.e., trajectories) (Bauer, 2007). We do acknowledge these concerns; however we think that for the purpose of this paper the use of joint trajectories was appropriate. We aimed at identifying robust groups of adolescents that showed consistently different levels of cannabis and depressive symptoms. By identifying these trajectories we were able to identify those adolescents that scores high on cannabis use and depressive symptoms. Finally, although participating families were carefully selected on the basis of several characteristics (e.g., educational level), results cannot be generalized to, for instance, adolescents from single-parent families or other cultures.

4.2. Conclusions

This study provided support for the idea that self-control plays a role in the joint development and co-occurrence of cannabis use and depressive symptoms. Identifying early adolescents with low self-control should need extra attention since these adolescents are possibly most at-risk for health problems. Specifically,

these children may also be more at-risk for joint development and co-occurrence of other health problems than presented in this study. Therefore, future studies should concentrate on the role of self-control in co-occurrence of other health risk behaviors and on psychological and physiological mechanisms underlying self-control and its relation to co-occurrence. Particularly when turns out that self-control is a major risk factor for joint risk behaviors, prevention programs could target at children who score low on self-control. Potentially by means of prevention programs that, for instance, aim at strategies to increase one's self-efficacy to refuse peer pressure and by providing children with adequate coping skills.

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Contributors

Roy Otten, Edward D. Barker, Barbara Maughan, Louise Arseneault, Rutger Engels. Otten designed this present study conducted the analyses and wrote the first draft of the manuscript. Barker contributed to the design, assisted in the statistical analyses and writing of the results section. Arseneault and Maughan contributed to the theoretical underpinnings of the study. Engels designed the larger study from which the data were drawn. All authors substantially contributed to the revision of the intellectual content of the manuscript.

Conflict of interest

No conflict declared.

References

- Arseneault, L., Cannon, M., Poulton, R., Murray, R., Caspi, A., Moffitt, T.E., 2002. Cannabis use in adolescence and risk for adult psychosis: longitudinal prospective study. *BMJ* 325, 1123–1212.
- Barker, E.D., Séguin, J.R., White, H.R., Bates, M.E., Lacourse, É., Carbonneau, R., Tremblay, R.E., 2007. Developmental trajectories of male physical violence and theft: relations to neurocognitive performance. *Arch. Gen. Psychiatry* 64, 592–599.
- Bauer, D., 2007. Observations on the use of growth mixture models in psychological research. *Multivar. Behav. Res.* 42 (4), 757–786.
- Baumeister, R.F., Heatherton, T.F., Tice, D.M., 1994. *Losing Control: How and Why People Fail at Self-Regulation*. Academic Press, San Diego, CA.
- Bovasso, G.B., 2001. Cannabis abuse as a risk factor for depressive symptoms. *Am. J. Psychiatry* 158 (12), 2033–2037.
- Boys, A., Marsden, J., 2003. Perceived functions predict intensity of use and problems in young polysubstance users. *Addiction* 98 (7), 951–963.
- Brendgen, M., Lamarche, V., Wanner, B., Vitaro, F., 2010. Links between friendship relations and early adolescents' trajectories of depressed mood. *Dev. Psychol.* 46 (2), 491–501.
- Brook, J.S., Gordon, A.S., Brook, A., Brook, D.W., 1989. The consequences of marijuana use on intrapersonal and interpersonal functioning in black and white adolescents. *Genet. Soc. Gen. Psychol. Monogr.* 115 (3), 349–369.
- Buchan, B.J., Dennis, M.L., Tims, F.M., Diamond, G.S., 2002. Cannabis use: consistency and validity of self-report, on-site urine testing and laboratory testing. *Addiction* 97, 98–108.
- Compas, B.E., Ey, S., Grant, K.E., 1993. Taxonomy, assessment, and diagnosis of depression during adolescence. *Psychol. Bull.* 114 (2), 323–344.
- Dahl, R.E., 2004. Adolescent development and the regulation of behavior and emotion: introduction to part VIII. *Ann. N. Y. Acad. Sci.* 1021, 294–295.
- Davey, C.G., Yucel, M., Allen, N.B., 2008. The emergence of depression in adolescence: development of the prefrontal cortex and the representation of reward. *Neurosci. Biobehav. Rev.* 32 (1), 1–19.
- De Vries, H., Engels, R., Kremers, S., Wetzels, J., Mudde, A., 2003. Parents' and friends' smoking status as predictors of smoking onset: findings from six European countries. *Health Educ. Res.* 18 (5), 627–636.
- Degehard, L., Hall, W., Lynskey, M., 2003. Testing hypotheses about the relationship between cannabis use and psychosis. *Drug Alcohol Depend.* 71 (1), 37–48.
- De Kemp, R.A.T., Vermulst, A.A., Finkenauer, C., Scholte, R.H.J., Overbeek, G., Rommes, E.W.M., Engels, R.C.M.E., 2009. Self-control and early adolescent antisocial behavior: a longitudinal analysis. *J. Early Adolesc.* 29, 497–517.
- Dékovic, M., 1996. *Vragenlijst Depressie bij Adolescenten*, VDA. Intern Rapport: Universiteit Utrecht.
- D'Souza, D.C., Perry, E., MacDougall, L., Ammerman, Y., Cooper, T., Wu, Y.T., Braley, G., Gueorguieva, R., Krystal, J.H., 2004. The psychotomimetic effects of intravenous delta-9-tetrahydrocannabinol in healthy individuals: implications for psychosis. *Neuropsychopharmacology* 29 (8), 1558–1572.
- Dorard, G., Berthoz, S., Phan, O., Corcos, M., Bungener, C., 2008. Affect dysregulation in cannabis abusers: a study in adolescents and young adults. *Eur. Child Adolesc. Psychiatry* 17 (5), 274–282.
- DuBois, D.L., Felner, R.D., Bartels, C.L., Silverman, M.M., 1995. Stability of self-reported depressive symptoms in a community sample of children and adolescents. *J. Clin. Child Psychol.* 24, 386–396.
- Enders, C.K., Bandalos, D.L., 2001. The relative performance of full information maximum likelihood estimation for missing data in structural equation models. *Struct. Equ. Model.* 8 (3), 430–457.
- Engels, R.C.M.E., Finkenauer, C., Meeus, W., Dekovic, M., 2001. Parental attachment and adolescents' emotional adjustment: the associations with social skills and relational competence. *J. Couns. Psychol.* 48 (4), 428–439.
- Ernst, M., Pine, D.S., Hardin, M., 2006. Triadic model of the neurobiology of motivated behavior in adolescence. *Psychol. Med.* 36, 299–312.
- Fergusson, D.M., Horwood, L.J., 1997. Early onset cannabis use and psychosocial adjustment in young adults. *Addiction* 92 (3), 279–296.
- Fergusson, D.M., Horwood, L.J., Swain-Campbell, N., 2002. Cannabis use and psychosocial adjustment in adolescence and young adulthood. *Addiction* 97 (9), 1123–1135.
- Finkenauer, C., Engels, R.C.M.E., Baumeister, R.F., 2005. Parenting and adolescent externalizing and internalizing problems: the role of self-control. *Int. J. Behav. Dev.* 29, 58–69.
- Flory, K., Lynam, D., Milich, R., Leukefeld, C., Clayton, R., 2004. Early adolescent through young adult alcohol and marijuana use trajectories: early predictors, young adult outcomes, and predictive utility. *Dev. Psychopathol.* 16 (1), 193–213.
- Forbes, E.E., Dahl, R.E., 2005. Neural systems of positive affect: relevance to understanding child and adolescent depression? *Dev. Psychopathol.* 17 (3), 827–850.
- Galvan, A., Hare, T.A., Parra, C.E., Penn, J., Voss, H., Glover, G., Casey, B.J., 2006. Earlier development of the accumbens relative to orbitofrontal cortex might underlie risk-taking behavior in adolescents. *J. Neurosci.* 26 (25), 6885–6892.
- Giedd, J.N., 2004. Structural magnetic resonance imaging of the adolescent brain. *Ann. N. Y. Acad. Sci.* 1021, 77–85.
- Harakeh, Z., Scholte, R.H., De Vries, H., Engels, R.C.M.E., 2005. Parental rules and communication: their association with adolescent smoking. *Addiction* 100 (6), 862–870.
- Holden, R.J., Pakula, I., 1998. Marijuana, stress and suicide: a neuroimmunological explanation. *Aust. N. Z. J. Psychiatry* 32 (3), 465–466.
- Jordan, N.C., Kaplan, D., Nabors Olah, L., Locuniak, M.N., 2006. Number sense growth in kindergarten: a longitudinal investigation of children at risk for mathematics difficulties. *Child Dev.* 77 (1), 153–175.
- Kandel, D.B., Davies, M., 1986. Adult sequelae of adolescent depressive symptoms. *Arch. Gen. Psychiatry* 43 (3), 255–262.
- Kandel, D.B., Davies, M., 1982. Epidemiology of depressive mood in adolescents: an empirical study. *Arch. Gen. Psychiatry* 39 (10), 1205–1212.
- Kendall, P.C., Cantwell, D.P., Kazdin, A.E., 1989. Depression in children and adolescents: assessment issues and recommendations. *Cogn. Ther. Res.* 13, 109–146.
- Kuijjer, R., de Ridder, D., Ouweland, C., Houx, B., van den Bos, R., 2008. Dieting as a case of behavioural decision making. Does self-control matter? *Appetite* 51 (3), 506–511.
- Magar, E.C.E., Phillips, L.H., Hosie, J.A., 2008. Self-regulation and risk-taking. *Pers. Individ. Differ.* 45, 153–159.
- Marmorstein, N.R., Iacono, W.G., Malone, S.M., 2010. Longitudinal associations between depression and substance dependence from adolescence through early adulthood. *Drug Alcohol Depend.* 107 (2–3), 154–160.
- Mathews, J., Youman, K., Stuewig, J., Tangney, J., 2007. Reliability and validity of the brief self-control scale in a sample of incarcerated offenders. Paper presented at the Annual Meeting of the American Society of Criminology, Atlanta Marriott Marquis, Atlanta, Georgia, 2007, November 13. Retrieved February 9, 2009, from http://www.allacademic.com/meta/p202012_index.html.
- Moffitt, T.E., 1993. Adolescence-limited and life-course-persistent antisocial behavior: a developmental taxonomy. *Psychol. Rev.* 100 (4), 674–701.
- Monshouwer, K., Van Dorselaer, S., Van Verduren, J., Ter Bogt, T., De Graaf, R., Vollebergh, W., 2006. Cannabis use and mental health in secondary school children. Findings from a Dutch survey. *Br. J. Psychiatry* 188, 148–153.
- Moore, T.H., Zammit, S., Lingford-Hughes, A., Barnes, T.R., Jones, P.B., Burke, M., Lewis, G., 2007. Cannabis use and risk of psychotic or affective mental health outcomes: a systematic review. *Lancet* 370 (9584), 319–328.
- Muraven, M., Baumeister, R.F., 2000. Self-regulation and depletion of limited resources: does self-control resemble a muscle? *Psychol. Bull.* 126, 247–259.
- Muthen, B., 2003. Statistical and substantive checking in growth mixture modeling: comment on Bauer and Curran. *Psychol. Methods* 8 (3), 369–377, 319–328.
- Muthen, L., Muthen, B., 1998–2003. *Mplus User's Guide*. Muthen Muthen, Los Angeles, CA.
- Nelson, E.E., Leibenluft, E., McClure, E.B., Pine, D.S., 2005. The social re-orientation of adolescence: a neuroscience perspective on the process and its relation to psychopathology. *Psychol. Med.* 35 (2), 163–174.

- Ollendick, T.H., Seligman, L.D., Goza, A.B., Byrd, D.A., Singh, K., 2003. Anxiety and depression in children and adolescents: a factor-analytic examination of the tripartite model. *J. Child Family Stud.* 12, 157–170.
- Otten, R., Ven, M.O.M., van de, Engels, R.C.M.E., Eijnden, R.J.J.M., van den, 2009. Depressive mood and smoking onset: a comparison of adolescents with and without asthma. *Psychol. Health* 24 (3), 287–300.
- Renault, P.F., Schuster, C.R., Freedman, D.X., Sikic, B., Nebel de Mello, D., Halaris, A., 1974. Repeat administration of marihuana smoke to humans. *Arch. Gen. Psychiatry* 31 (1), 95–102.
- Rodriguez, D., Moss, H.B., Audrain-McGovern, J., 2005. Developmental heterogeneity in adolescent depressive symptoms: associations with smoking behavior. *Psychosom. Med.* 67, 200–210.
- Shedler, J., Block, J., 1990. Adolescent drug use and psychological health: a longitudinal inquiry. *Am. Psychol.* 45, 612–630.
- Tangney, J.P., Baumeister, R.F., Boone, A.L., 2004. High self-control predicts good adjustment, less pathology, better grades, and interpersonal success. *J. Pers.* 72, 271–324.
- Van den Eijnden, R., Meerkerk, G., Vermulst, A., Spijkerman, R., Engels, R., 2008. Online communication, compulsive Internet use and psychosocial well-being among adolescents: a longitudinal study. *Dev. Psychol.* 44 (3), 655–665.
- Van der Vorst, H., Engels, R., Meeus, W., Dekovic, M., 2005. The impact of alcohol-specific socialization on adolescents' drinking behaviour. *Addiction* 100 (10), 1464–1467.
- Verdejo-García, A., Lawrence, A.J., Clark, L., 2008. Impulsivity as a vulnerability marker for substance-use disorders: review of findings from high-risk research, problem gamblers and genetic association studies. *Neurosci. Biobehav. Rev.* 32 (4), 777–810.
- Wills, T.A., Stoolmiller, M., 2002. The role of self-control in early escalation of substance use: a time-varying analysis. *J. Consult. Clin. Psychol.* 70 (4), 986–997.