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## **Cannabis use and violence: Is there a link?**

**Thor Norström**  
**Ingeborg Rossow**

Swedish Institute for Social Research (SOFI), Stockholm University, Stockholm, Sweden  
Norwegian Institute for Alcohol and Drug Research (SIRUS), Oslo, Norway

## **Cannabis use and violence – is there a link?**

Thor Norström<sup>1,2</sup> & Ingeborg Rossow<sup>2</sup>

<sup>1</sup>Swedish Institute for Social Research (SOFI)  
Stockholm University  
S-109 91 Stockholm, Sweden  
Fax: +468154670  
E-mail: [totto@sofi.su.se](mailto:totto@sofi.su.se)

<sup>2</sup>Norwegian Institute for Alcohol and Drug Research (SIRUS)  
P.O. Box 565 Sentrum  
0105 Oslo  
Norway.  
E-mail: [ir@sirus.no](mailto:ir@sirus.no)  
Fax: + 47 22340401

Correspondence: Thor Norström, Swedish Institute for Social Research, Stockholm University, S-106 91 Stockholm, Sweden,  
tel: +46-8-162314, fax: +46-8-154670, e-mail: [totto@sofi.su.se](mailto:totto@sofi.su.se)

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## **Abstract**

**Background:** Although several studies suggests that cannabis users are at increased risk of interpersonal violence, it is not clear to what extent the association is causal..The aim of this paper is to assess the association between cannabis use and violence using a method that diminishes the risk of confounding. **Methods:** We analysed data on cannabis use and violent behaviour from the second (1994) and third (1999) waves of the Young in Norway Longitudinal Study (cumulative response rate: 68.1%,  $n = 2681$ ). We applied fixed-effects modelling to estimate the association between these behaviours, implying that changes in the frequency of violence were regressed on changes in the frequency of cannabis use. Hence, the effects of time-invariant confounders were eliminated. In addition, we included two time-varying covariates. **Results:** The elasticity estimate implied that at 10% increase in cannabis use frequency was associated with a 0.4% increase in frequency of violence ( $P=.024$ ). **Conclusion:** Analyses of panel data on Norwegian youths revealed a statistically significant association between cannabis use and violence.

**Key words:** cannabis, violence, panel data, Norway

## **INTRODUCTION**

Does cannabis use play a causal part in violence? In the mid 1930's cannabis was considered the 'killer weed' in the United States (1), and the assumption that cannabis use caused violence contributed to the Marihuana Tax Act in 1937 (2), which radically limited legal access to cannabis. A quarter of a century later, the production, trade, and possession of cannabis became criminal offences in countries under the Single Convention on Narcotic Drugs (1961) (3). These days cannabis is the most widely used illicit drug in most parts of the world (4), and most people who use cannabis do so in adolescence or young adulthood (5). Thus, the possible health consequences of cannabis use have been intensely studied (3, 6). Yet, the answer to the opening question is still ambiguous. While the public image of the drug's effects on aggressive behaviour has changed significantly and has been replaced by that of a drop-out drug and a calming substance (1), the scientific evidence of a possible causal effect of cannabis use on violence is still limited and the findings and interpretations are mixed (7, 8). In this study we will address the question of a possible causal role of cannabis in violence empirically in a longitudinal study of young people in Norway.

### **Cannabis – violence: possible mechanisms and previous studies**

While the majority of studies suggests that cannabis users are at increased risk of interpersonal violence (7), it is not clear to what extent the association is causal. However, the literature suggests several mechanisms that may underlie a possible causal link between cannabis use and violence. First, although the acute effects of cannabis generally comprise mild euphoria and relaxation, adverse acute psychopharmacological effects manifested as panic attacks, confusion, hallucinations, suspiciousness and paranoia may also occur, all of which may affect emotions and cognition in ways that enhance aggressive responses to provocations (7, 9). Second, withdrawal symptoms resulting from an abstinent period in

frequent users of cannabis often include irritability, anger, and aggression (7, 8), and this could be another - possible causal - effect (8). Third, it is suggested that violent behaviour may occur in connection with market transactions (e.g. fights over money) or to uphold rules related to the drug market (7).

As to empirical studies on the link between cannabis and various forms of violence and aggression, animal studies report positive as well as null findings (7, 10). Also, the few laboratory studies of humans seem to be inconclusive (7, 10). In cross-sectional studies it is generally found that cannabis users are at increased risk of violent behaviour (7). However, the observed cross-sectional associations between cannabis use and violence are compromised by the likely presence of confounders (6). These may be stable (i.e. time-invariant) factors, such as genetic or temperament traits, antisocial personality disorder, and parental modelling (11), while other potential confounders, such as drinking and socialising with non-normative peers, may vary over time in adolescence. Heavy episodic drinking is associated with violence (12) as well as with cannabis use (13); actually, most cannabis use among young people seems to occur when drinking (14). Regarding the possible influence of non-normative peers, research suggests that young people's cannabis use and violent behaviour are associated with that of their peers (11, 12). Thus, there are both stable individual factors (many of which are unobservable in practice, such as genetic factors), and time-dynamic individual and environmental factors that are likely to confound an association between cannabis use and violence (6, 15).

In addition to the cross-sectional studies, which are limited as a basis for inferring causal relationships (16), the relatively few longitudinal studies have produced somewhat mixed findings. White and Hansell (17); Brook et al. (18); Fergusson et al. (19); Marie et al. (20);

and Friedman et al. (21, 22) found a positive association between cannabis use and violent behavior after adjustment for confounding factors, Green et al. (23) did not. Although all these studies did include control variables, there is always the risk of remaining residual confounding. Moreover, these studies typically assess the possible long-term effects of cannabis use in adolescence on violence later in life. Such a long-term effect is compatible with a hypothesis of cannabis use as a gateway into a deviant career, but it does not reveal much about the possible acute effects of cannabis.

An alternative way of analysing longitudinal data in this context is fixed-effects modelling that addresses the question ‘Is a *change* in cannabis use associated with a *change* in violence?’ This approach is more feasible for assessing possible acute effects of cannabis; in addition, it eliminates confounding due to shared risk factors between cannabis use and violence that are stable across time (see below). The study by Fergusson et al. (15) is the only one that has applied such an approach; in addition to fixed-effects modelling they included a set of time-dynamic co-variates. However, the relevance of their study is somewhat limited in the present context, as they did not address violent behaviour specifically, but applied a composite measure of violent crimes and property crimes. Thus, notwithstanding the fairly large number of studies, the empirical evidence on a possible causal effect of cannabis use on violence is inconclusive.

## **AIMS**

The main aim of our study was to assess the association between cannabis use and violence by applying a method that is expected to minimise bias due to unobserved confounders.

## **MATERIAL AND METHODS**

### **Data**

We applied data from a longitudinal cohort study of Norwegian adolescents, the *Young in Norway Longitudinal Study*, which has been described in detail elsewhere (24). The data we required were collected in the second (1994) and third (1999) waves. The initial sample (1992) was obtained by selecting schools from a national register of all junior and senior high schools. The sampling procedures were designed to obtain a nationwide, representative cross-section of this student population in Norway. The response rate in the 1992 survey was 97.0%, while the corresponding figures for the 1994 and 1999 surveys were 91.8% and 83.8%, respectively. In both 1992 and 1994, questionnaires were distributed and completed in the classroom, while a postal survey was carried out in 1999. The 1999 survey was completed by 2924 respondents, with a cumulative response rate of 68.1%. After list-wise exclusion of respondents with missing data on the measures we analysed, we were left with 2681 respondents for analyses. In the following, we will refer to the 1994 survey and the 1999 survey as T1 and T2, respectively. The sample comprised slightly more females (56.1%) than males, and the mean age (SD) was 16.5 years (1.9) at T1, and 21.6 years at T2.

### **Strategy of analysis**

We first describe the relation between cannabis use and violence by means of bivariate and multivariate modelling of cross-sectional data. The multivariate model included a set of potential confounders. Besides age and sex, impulsivity was included, as it may be related to cannabis use (25), as well as to violence (26). Heavy episodic drinking and non-normative peers were included for the same reason. Next, we applied fixed-effects modelling (27), which eliminates the risk for confounding that is due to covariates that are stable across time. However, it does not remedy bias due to time-varying factors that affect violence as well as cannabis use, and some identified time-varying factors are therefore included in the fixed-effects model.

## Measures

*Violence* was measured identically at T1 and T2 by 2 questions: (1) ‘During the past 12 months, how many times have you been in a fight (without a weapon)?’, and (2) ‘During the past 12 months, how many times have you been in a fight (with a weapon)?’ There were 6 response options: ‘Never’ (coded 0), ‘Once’ (1), ‘2 to 5 times’ (3.5), ‘6 to 10 times’ (8), ‘11 to 50 times’ (30), and ‘More than 50 times’ (55) and a sum-score for violence frequency at T1 and T2 was constructed, with a possible range from 0 to 110.

*Cannabis use (CAN)* was measured identically at T1 and T2 by the question: ‘During the past 12 months, how many times have you used hash or marijuana?’ The response options were identical to those for violent behaviour.

*Impulsivity (IMP)* was measured at T2 only, through 6 items based on Eysenck’s Personality Questionnaire (28) with the stem ‘Decide whether the statements below correspond to you’: (1) I’m an impulsive person; (2) I rarely do anything carefully; (3) I act on the spur of the moment; (4) I rarely take chances; (5) When I’m having fun, I don’t think of the consequences; and (6) I make up my mind quickly. There were four response options, ranging from ‘Corresponds very poorly’ (coded 1) to ‘Corresponds very well’ (4). On the basis of the 6 items we constructed an additive index which was then divided by 6 (*IMP* with a theoretical range of 1–4, and Cronbach’s alpha = 0.72).

### *Time-varying control variables*

*Heavy episodic drinking (HED)* was measured identically at T1 and T2 by the question: ‘During the past 12 months, have you had so much to drink that you felt clearly intoxicated?’ The response options were identical to those for violent behaviour.

*Non-normative peers* was measured identically at T1 and T2 by an additive index (denoted *NNP*) based on whether one or both of the respondent's two closest friends (i) smokes regularly, (ii) has used cannabis; (iii) has been in contact with the police due to illegal activities (range 0–6).

All variables were log-transformed (as zero cannot be logged we first added 0.1 to variables containing zeros). This transformation alleviates skewness and yields an effect measure (elasticity) that is easy to interpret.

### **Statistical analyses**

The association between cannabis use and violence was first analysed in ordinary linear least square (OLS) regression models. The multivariate model included a set of available potential confounders: age and sex, impulsivity, heavy episodic drinking, and non-normative peers. The outcomes for T1 and T2 were pooled into average estimates. Next, we applied fixed-effects (FE) modelling (27). In practice this means that we calculated change scores for the dependent variable (violence) as well as the explanatory variables by subtracting the value at T1 from the value at T2. Then, these variables were used in ordinary OLS regression. The main advantage of fixed-effects modelling is that it reduces the risk of bias due to confounding. More precisely, if a common cause of cannabis use and violence is stable across time (e.g. some environmental or individual determinant), the differencing means that the impact of this causal factor is cancelled out, as shown below:

$$(1) V_{i1} = \beta_1 CAN_{i1} + \beta_2 C_i + e_{i1}$$

$$(2) V_{i2} = \beta_1 CAN_{i2} + \beta_2 C_i + e_{i2}$$

In model (1)  $V_{it}$  and  $CAN_{it}$  are violence and cannabis use, respectively, for individual  $i$  at T1.  $C_i$  denotes other causes of violence that are time invariant. If  $CAN$  and  $C_i$  are correlated, the estimate of  $\beta_1$  will be biased. Model (2) is equivalent, but refers to T2. It can be seen that if one subtracts (1) from (2), that is, applies fixed-effects modelling,  $C_i$ , and thereby that particular source of bias, is eliminated. Two time-varying factors that affect violence as well as cannabis use were identified above (HED and NNP), and the difference scores from T1 to T2 of these variables were included in the fixed-effects model.

## RESULTS

Table 1 and 2 show that whereas cannabis use increased, violence decreased between T1 and T2. This suggests that any possible effect of the upward shift in cannabis use on violence is masked by much stronger countervailing effects of factors related to increasing age. The frequency of both violent behaviour and cannabis use was stable from T1 to T2 for the majority of the respondents, but for both types of behaviour, there were also some who had increased the frequency and some who had decreased the frequency (Table 3).

Table 1 -3 about here

The simple bivariate analysis (Table 4) suggested a positive and statistically significant association between cannabis use and violence. The inclusion of the control variables, that is, age, sex, heavy episodic drinking, and non-normative peers, reduced the association markedly. This outcome was by and large replicated by the fixed-effects estimates; although the estimated associations between violence, on the one hand, and cannabis use and drinking, on the other, were additionally weakened, they were still statistically significant. With log-transformed variables the parameter estimates express the percentage change in violence

frequency given a 1% increase in the frequency of cannabis use. The outcome from the fixed-effects model thus implies that at 10% increase in cannabis use frequency is associated with a 0.4% increase in the frequency of violent behaviour.

Table 4 about here

## **DISCUSSION**

In this study we found that an increase in cannabis use was associated with an increased risk of violence. We used an analytic strategy that strengthens the case for a causal interpretation of the observed association. The applied modelling technique thus accounts for time-invariant unobserved confounders; in addition, we controlled for time-dynamic covariates. The study thus adds to the meagre literature on observational studies of the cannabis–violence association that attempts to control for confounding in a rigorous way. Our finding is in line with that of Fergusson and co-workers (15), who applied the same modelling technique. However, the latter study used a broader outcome measure, combining violence and property crime, and thus, the findings are not directly comparable.

### *Study strengths and limitations*

We applied a strong study design to assess the link between cannabis use and violence, which, so far, has rarely been seen in observational studies on this topic. Moreover, the study sample was fairly large, which reduced the risk of discarding a moderate association. While the follow-up rate was fairly good, there was nevertheless an attrition bias; previous analyses suggest that adolescents with deviant behaviour were to a larger extent lost to follow up (29). This may have led to a reduced variance in the input and outcome variables, with the concomitant risk of downwards bias of the effect estimates. On the other hand, we cannot

preclude the possibility that there is time-dynamic confounding that was not accounted for in the analyses, which may have biased the effect estimates upward. Further, the measures of cannabis use and violence were fairly crude, which is likely to have inflated the standard error of the estimated association between the two. Another limitation is that we do not have information about whether the reported incidents of violence co-occur with the use of cannabis; we only know that those who reported an increase in cannabis use also reported an increase in violence involvement. Finally, we rely on data that were collected in the late 1990s in Norway. The generalizability of our findings should thus be probed by analyses of more recent data pertaining to other cultural contexts.

### *Implications*

Over the past decade cannabis legislation has changed in various parts of the world; e.g. in many European countries possession and use have been de-penalised. Moreover, in the United States 14 states have de-penalised recreational use, more than a third of the states have legalised cannabis for medical use, and initiatives to legalise recreational use have been on the ballot in a number of states in the past few years. Although the literature on the likely effects of such changes on cannabis use is very sparse, there are some indications that de-penalisation may lead to further increase in cannabis use (30). Thus, given that de-penalisation may lead to increased cannabis use, and that cannabis use may spur violence, the likely consequences of such legislative changes are clearly of relevance to public health and safety.

Against this background it is important that future research seeks to obtain a stronger empirical basis for the assessment of possible health and social consequences of cannabis use.

Further studies of possible causal effect of cannabis use on violence and assessment of likely underlying mechanisms are warranted.

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The authors are listed alphabetically, and contributed equally to this paper.

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Table 1. Descriptive statistics

|     | T1   |       | T2    |       |
|-----|------|-------|-------|-------|
|     | Mean | SD    | Mean  | SD    |
| VIO | 0.88 | 4.11  | 0.42  | 2.83  |
| CAN | 0.51 | 3.84  | 2.41  | 9.53  |
| HED | 7.29 | 12.72 | 16.74 | 16.75 |
| NNP | 1.46 | 1.34  | 1.52  | 1.37  |
| IMP |      |       | 2.50  | 0.48  |

Note: VIO = violent behavior, CAN = cannabis use; HED = heavy episodic drinking; NNP = non-normative peers; IMP = impulsivity.

Table 2. Frequency (number of times during the past 12 months) of violence and cannabis use at T1 and T2 ( $n=2681$ ). Per cent

| Frequency | Cannabis use |      | Violence |      |
|-----------|--------------|------|----------|------|
|           | T1           | T2   | T1       | T2   |
| 0         | 94.3         | 83.8 | 78.7     | 89.0 |
| 1-5       | 4.0          | 9.6  | 18.6     | 10.0 |
| 6-        | 1.7          | 6.6  | 2.7      | 1.0  |

Table 3. Change in violent behaviour and cannabis use from T1 to T2 ( $n=2681$ )

|              | Change from T1 to T2 (%) |        |          |
|--------------|--------------------------|--------|----------|
|              | Decrease                 | Stable | Increase |
| Violence     | 17.5                     | 76.1   | 6.4      |
| Cannabis use | 3.0                      | 81.9   | 15.1     |

Table 4. Estimated models (log-log) with violence as outcome. The bivariate and multivariate models are cross-sectional models where estimates for T1 and T2 have been pooled ( $n = 2681$ ).

| Predictor | Bivariate model |       |       | Multivariate model |       |       | Fixed-effects model |       |       |
|-----------|-----------------|-------|-------|--------------------|-------|-------|---------------------|-------|-------|
|           | Estimate        | SE    | p     | Estimate           | SE    | P     | Estimate            | SE    | P     |
| CAN       | 0.141           | 0.016 | <.001 | 0.056              | 0.016 | <.001 | 0.041               | 0.015 | 0.008 |
| HED       |                 |       |       | 0.050              | 0.009 | <.001 | 0.041               | 0.010 | <.001 |
| AGE       |                 |       |       | -0.097             | 0.009 | <.001 |                     |       |       |
| SEX       |                 |       |       | -0.554             | 0.031 | <.001 |                     |       |       |
| NNP       |                 |       |       | 0.090              | 0.012 | <.001 | 0.059               | 0.017 | 0.001 |
| IMP       |                 |       |       | 0.634              | 0.085 | <.001 |                     |       |       |

Note: CAN= cannabis use; HED = heavy episodic drinking; NNP= non-normative peers; IMP =impulsivity.