Marijuana use and car crash injury

Stephanie Blows[1], Rebecca Q. Ivers[1], Jennie Connor[2], Shanthi Ameratunga[2], Mark Woodward[1] & Robyn Norton[1]

The George Institute For International Health, Sydney[1] and School of Population Health, University of Auckland, New Zealand[2]

Correspondence to:
Stephanie Blows
The George Institute For International Health
Level 10 King George V Building
Royal Prince Alfred Hospital
PO Box M201
Missenden Road
Sydney
NSW 2050
Australia
Tel: +61 29993 4585
Fax: +61 29993 4501
E-mail: sblows@thegeorgeinstitute.org

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ABSTRACT

Aims To investigate the relationship between marijuana use prior to driving, habitual marijuana use and car crash injury

Design and setting Population based case–control study in Auckland, New Zealand.

Participants Case vehicles were all cars involved in crashes in which at least one occupant was hospitalized or killed anywhere in the Auckland region, and control vehicles were a random sample of cars driving on Auckland roads. The drivers of 571 case and 588 control vehicles completed a structured interview.

Measurements Self reported marijuana use in the 3 hours prior to the crash/survey and habitual marijuana use over the previous 12 months were recorded, along with a range of other variables potentially related to crash risk. The main outcome measure was hospitalization or death of a vehicle occupant due to car crash injury.

Findings Acute marijuana use was significantly associated with car crash injury, after controlling for the confounders age, gender, ethnicity, education level, passenger carriage, driving exposure and time of day (OR 3.9, 95% CI 1.2–12.9). However, after adjustment for these confounders plus other risky driving at the time of the crash (blood alcohol concentration, seat-belt use, travelling speed and sleepiness score), the effect of acute marijuana intake was no longer significant (OR 0.8, 95% CI 0.2–3.3). There was a strong significant association between habitual use and car crash injury after adjustment for all the above confounders plus acute use prior to driving (OR 9.5, 95% CI 2.8–32.3).

Conclusions This population-based case–control study indicates that habitual use of marijuana is strongly associated with car crash injury. The nature of the relationship between marijuana use and risk-taking is unclear and needs further research. The prevalence of marijuana use in this driving population was low, and acute use was associated with habitual marijuana use, suggesting that intervention strategies may be more effective if they are targeted towards high use groups.

KEYWORDS Cannabis, case control study, motor vehicle injury.

INTRODUCTION

Marijuana is an increasingly widely used drug, particularly among young people [1–3], and there is concern about its effects on road safety. Many research studies and several reviews of the literature have examined the relationships between marijuana use, driving ability and crashes [4–11]. Driving ability and crash risk may be

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affected by both acute marijuana use prior to driving and habitual use of marijuana [9]. The effects of acute marijuana use have been studied in both laboratory/simulator studies and epidemiological studies of drivers. Laboratory studies of the effect of acute marijuana intake on various tasks show impairment in reaction time, attention, coordination and motor skills, which are likely to be important in driving [1]. However, there is less evidence that this translates into actual driving impairment in simulator studies [9]. There have been few studies of the prevalence of acute marijuana use among on-road drivers, but these estimate it to be around 1–6% in those driving populations that have been studied [4,9,12]. Studies of crash-involved and/or injured drivers have found evidence that marijuana use is higher is these populations, at 4–12% [4,9,12–14]. This suggests that there is a positive relationship between marijuana use and car crashes and injury. However, epidemiological studies of the effect of acute marijuana intake on crash risk have had variable results. Some studies have suggested marijuana use may increase the likelihood of culpability in a crash; in particular, a recent case–control study by Drummer *et al*. found that drivers who tested positive for marijuana use were more likely than drug-free drivers to be culpable for the crash [7,11]. However, other studies have failed to find an association between marijuana use and car crash injury, and recent literature reviews conclude that overall the evidence for the role of marijuana use in car crashes remains inconclusive [5,6,9,10,15]. These reviews note that it is difficult to draw conclusions because of methodological problems, including selection bias, due particularly to low response rates; measurement bias relating to difficulty in accurately measuring marijuana use; and failure to adjust for important confounders, including alcohol consumption.

The effect of habitual marijuana use on car crash risk has been studied in several cohort studies [8,16–18], but not all have suggested a significant positive relationship. Many of these studies also have methodological problems, including long time delays between measurement of exposure and outcome [18] and use of self-reported and non-injury crashes as outcomes [16]. Furthermore, although these studies adjust for confounding variables such as age and gender, due to their prospective nature the results are usually not adjusted for important variables, such as speed and blood alcohol level, at the time of the crash. The joint associations between habitual and acute marijuana use and car crash injury have not been described.

This analysis examines the relationships between acute marijuana use, habitual marijuana use and car crash injury, using data from a population-based case–control study with 1159 participants. A wide range of crash-related variables were measured in this study, allowing us to adjust for many important confounding factors. The outcome factor was severe injury or death of a vehicle occupant as a result of a crash.

METHODS

Recruitment and data collection

The study was conducted in the Auckland region of New Zealand, which has a population of about 1.1 million [19] and includes both urban and rural areas. Details of the study methodology have been published previously [20]. Recruitment of participants occurred from March 1998 to July 1999. Case vehicles were defined as all cars involved in crashes in which at least one occupant (driver or passenger) of the car was hospitalized with injuries, or killed, during the study period, in the Auckland region. For control selection, random cluster sampling was used to obtain a representative sample of all driving in the study region. Random points were selected from a list of roads in the region and control cars, identified randomly proportional to the volume of traffic at the site, were stopped as close as possible to these points. One control participant per case was recruited at approximately the same rate and over the same time period as cases.

Drivers of all vehicles completed a face-to-face or telephone-administered questionnaire-based structured interview. For controls, this was related to the time that they were recruited during the roadside surveys. The next of kin or other suitable person (proxy respondent) was interviewed when a driver was fatally injured or otherwise unable to complete the interview. The interview contained 155 questions on potential risk factors for car crashes, including demographics; circumstances of the crash; and personal, vehicle and environmental factors. An environmental survey of the crash and control recruitment sites was also conducted to record road and traffic characteristics. The medical records of case participants were examined for information on injuries and other relevant variables. Blood alcohol levels were estimated using a breathalyser for controls and from hospital and police records for cases. Missing data for blood alcohol level were imputed according to self-reported alcohol consumption and whether or not consumption and impairment were suspected by ambulance and hospital staff. Full details of blood alcohol measurement and imputation have been published previously [21]. Marijuana use was measured using two questions. For acute use, participants were asked if they had used any marijuana in the 3 hours prior to the crash/roadside survey. For habitual use, participants were asked about frequency of marijuana use during the past 12 months. The interviewer reminded participants that all information

was strictly confidential and would not be disclosed to authorities.

Study population

Interviews were completed for 571 drivers of case vehicles, a response rate of 92.8%. Non-responders included 30 (4.9%) case vehicles for which the selected participant declined to participate in an interview and 14 (2.3%) that could not be contacted.

From the roadside surveys, 746 cars were identified as control vehicles. Of these, interviews were completed for 588 drivers (78.8%). Non-responders included 92 (12.3% of total) who declined to participate, 60 (8.0%) who could not be contacted and six (0.8%) who could not participate for other reasons.

Analysis

Frequencies, odds ratios and 95% confidence intervals were calculated from linear logistic regression models using SUDAAN software, which accounts for intracluster correlation of control data sampled from the same site. Proportions of controls were adjusted for the clustered sampling design. This was conducted by weighting control data by the inverse of the sampling fraction at the recruitment site and adjusting the variances of the estimates to account for intracluster correlation of data from the same site. We identified potential confounders from the epidemiological literature and adjusted for these in the analyses if they were associated significantly with car crash injury in our data after adjusting for driver's age and sex. To investigate the relationship between marijuana use and other acute risky driving variables, and to allow for the possibility that risky driving variables may be on the pathway between marijuana use and car crash injury, we obtained odds ratios for the association between acute and habitual marijuana use first adjusting only for other non-risk taking variables (age, sex, ethnicity, education level and driving exposure of the driver; age of vehicle; number of passengers; and time of day) then adding other variables measuring acute risky driving at the time of the crash/survey (sleepiness score, blood alcohol level, seat-belt use and speed) to each model.

RESULTS

The mean age of case drivers was 36.6 years and of control drivers 40.8 years. The case group was 65% male and the control group was 59% male. There were no significant differences in age group, sex or driving conditions, between drivers who were interviewed and all eligible drivers, for both cases and controls. Table 1 shows the distributions of acute and habitual marijuana use and all confounding variables by case–control status. Proportions of controls in this table are adjusted for the clustered sampling design. The proportion of drivers reporting marijuana use in the 3 hours prior to the crash/survey was 5.6% among cases and 0.5% among controls. Habitual marijuana use of an average of at least once per week over the past 12 months was reported by 10.0% of cases and 0.9% of controls. Of those who had used marijuana acutely in the 3 hours prior to the crash/survey, 87% of cases and 88% of controls were also habitual users, compared to 6% and 0.5%, respectively, for those who had not used it acutely. Missing data were less than 10% for all variables included in these analyses after imputation for blood alcohol level [21].

Table 2 shows the associations between acute and habitual marijuana use and car crash injury. Acute use of marijuana in the 3 hours prior to the crash was significantly associated with car crash injury in the univariate model (odds ratio (OR) 11.4, 95% confidence interval (CI) 3.6–35.4). This association remained significant after adjustment for the non-risk taking confounders age, sex, education, ethnicity, driving exposure, age of vehicle, time of day and number of passengers (OR 3.9, 95% CI 1.2–12.9). However, after adjustment for these confounders plus other risky driving at the time of the crash (sleepiness, blood alcohol concentration, seat-belt use and travelling speed), the effect of acute marijuana intake was no longer significant (OR 0.8, 95% CI 0.2–3.3). The association between habitual marijuana use and car crash injury was significant in both the univariable and age and sex-adjusted models, and remained significant after adjustment for all risk-taking and non-risk taking confounders, plus acute marijuana use prior to the crash/survey (OR 9.5, 95% CI 2.8–32.3). Restricting the analyses to cases in which the driver was hospitalized or killed did not alter the significance of the odds ratios.

DISCUSSION

This population-based case–control study is notable for measurement of multiple confounding factors, both acute and chronic; random selection of controls from a regional driving population; and the use of validated records on injury-related hospitalizations or death as the outcome. The results suggest that habitual users of marijuana have about 10 times the risk of car crash injury or death compared to infrequent or non-users, after adjustment for other crash-related variables including an objective measure of blood alcohol level. In addition, this study measured self-reported acute marijuana use prior to driving in a random selection of the Auckland regional driving population (our control participants) and compared

Table 1 Frequency distributions of acute and habitual marijuana use and other confounding variables by case–control status, Auckland Car Crash Injury Study.

	Cases (n = 571)		*Controls[1] (n = 588)*	
	No.	*(%)*	*No.*	*(%)*
Acute marijuana use (in past 3 hours)				
No	520	(91.1)	582	(99.2)
Yes	32	(5.6)	5	(0.5)
Don't know/missing	19	(3.3)	1	(0.2)
Habitual marijuana use (in past 12 months)				
Less than once per week	494	(86.5)	579	(98.7)
Once per week or more	57	(10.0)	7	(0.9)
Don't know/missing	20	(3.5)	1	(0.4)
Age of driver (years)				
< 25	195	(34.2)	91	(13.7)
25–34	133	(23.3)	125	(22.3)
35–44	85	(14.9)	154	(24.5)
45–54	61	(10.7)	107	(19.6)
55–64	39	(6.8)	80	(14.2)
65 +	58	(10.2)	31	(5.6)
Sex				
Female	198	(34.7)	226	(41.3)
Male	373	(65.3)	362	(58.7)
Education level				
Post-secondary	178	(31.5)	276	(49.3)
Secondary school, > 3 years	137	(24.2)	154	(25.1)
Secondary school, ≤ 3 years	252	(44.4)	157	(25.6)
Ethnicity				
White/European	313	(54.8)	444	(74.7)
Maori	117	(20.5)	61	(9.2)
Pacific Islander	86	(15.1)	36	(6.1)
Other	55	(9.6)	47	(10.0)
Time of day				
Not between 2 and 5 a.m.	525	(91.9)	571	(99.6)
Between 2 and 5 a.m.	46	(8.1)	17	(0.4)
Stanford Sleepiness Score				
1–3 (sleepy)	447	(87.7)	578	(99.0)
4–7 (not sleepy)	63	(12.3)	8	(1.0)
Number of passengers				
0	285	(50.3)	355	(62.9)
1	140	(24.7)	144	(23.4)
2 or more	142	(25.0)	88	(13.7)
Seat-belt use				
Yes	469	(82.1)	568	(97.4)
No	81	(14.2)	4	(0.8)
Blood alcohol concentration (mg percentage)[2]				
< 3	397	(69.7)	565	(96.6)
3–50	41	(7.2)	16	(2.6)
> 50	132	(23.2)	6	(0.8)
Travelling speed at time of crash				
0–30 kph	87	(16.9)	78	(13.9)
31–50 kph	113	(21.9)	229	(41.5)
51–80 kph	196	(38.1)	220	(33.7)
> 80 kph	119	(23.1)	55	(11.0)

[1]Proportions of controls are adjusted for the clustered sampling design. [2]Missing data imputed [16].

Table 2 Univariable, age- and sex-adjusted and multivariable adjusted odds ratios (95% confidence intervals) for the associations between acute marijuana use, habitual marijuana use and car crash injury, Auckland Car Crash Injury Study.

	Univariable		*Age- and sex-adjusted*		*Multivariable*[1]		*Multivariable*[2]	
	OR	*95% CI*	*OR*	*95% CI*	*OR*	*95% CI*	*OR*	*95% CI*
Acute marijuana use (in past 3 hours)								
Yes	11.4	(3.6–35.4)	6.0	(1.8–20.3)	3.9	(1.2–12.9)	0.8	(0.2–3.3)
No	1.0	Reference	1.0		1.0		1.0	
Habitual marijuana use (in past 12 months)								
Less than once per week	12.7	(5.3–30.6)	8.6	(3.3–22.0)	9.1	(3.8–22.0)	9.5	(2.8–32.3)
Once per week or more	1.0	Reference	1.0		1.0		1.0	

[1]Adjusted for age, sex, education, ethnicity, driving exposure, age of vehicle, time of day and number of passengers. [2]Adjusted for the above confounders, plus blood alcohol concentration, seat-belt use and travelling speed. The model for habitual marijuana use is also adjusted for acute marijuana use.

this to crashed drivers from the same population, which has been achieved by few previous studies [9]. Marijuana use prior to the crash/survey was reported by 5.6% of case drivers and 0.5% of control drivers. Acute use of marijuana prior to the crash was associated with four times the risk of car crash injury with adjustment for non-risk taking variables, but after adjustment for speed, seat-belt use, blood alcohol level and sleepiness this association was no longer present. Acute use of marijuana was also highly correlated with habitual use: 87% of cases and 88% of controls who had used marijuana acutely prior to the crash/survey were also habitual users, compared to less than 10% who had not.

The relationship we observed between habitual marijuana use and car crash injury has been found previously in a number of cohort studies [17,18,22]. These have suggested associations between frequent habitual marijuana use and alcohol-related, single vehicle and at-fault crashes [17]; and crashes causing injury [18] and hospitalization [22]. Unlike most of these studies, we were able to control for marijuana use prior to the crash, minimizing the possibility that habitual users were at increased risk because they had been smoking marijuana prior to driving. However, the mechanism by which habitual marijuana use increases the risk of car crash injury has not been identified. For example, habitual users may be more likely to engage in other risk taking behaviours. Fergusson & Horwood [16], in a New Zealand-based cohort study, found that habitual marijuana users were more likely to report risky/illegal driving behaviors and attitudes, which were associated with increased risk of car crashes. We included the risky driving variables speed, non-use of seat-belts, sleepiness and high blood alcohol level in our models but found that this did not remove the significant effect of habitual marijuana use. However, heavy users may be more likely to engage in other risky driving behaviours associated with car crashes that we did not measure, and our analysis may thus be subject to uncontrolled confounding. The possibility that habitual heavy marijuana use induces mental impairment, that in turn affects driving ability, is less likely. Although there is some evidence that habitual marijuana use can cause long-term changes in brain functioning [23,24] the results are inconclusive and this has not been studied in relation to driving. There is some evidence that heavy habitual marijuana users build up continuously high levels of the drug and may display ongoing acute effects [25]. If so, the association we observed may be due to these acute effects rather than due solely to the effect of habitual marijuana use.

Marijuana use prior to driving was reported by 0.5% of control drivers in this study. These figures may be subject to under-reporting, but are consistent with the estimates of 1–6% that have been reported previously for similar populations [4,9]. They suggest that the prevalence of marijuana use prior to driving in the general population is low. More cases than controls reported using marijuana acutely in the 3 hours prior to the crash; however, acute marijuana use was not associated with car crash injury after adjustment for the crash-related risky driving variables of speed, seat-belt use, blood alcohol level and sleepiness. This supports several previous studies and literature reviews that have not found an effect of acute marijuana intake on crash risk [4,7,16]. However, some studies have suggested that acute marijuana use does have an effect on crash risk [6,12] and in particular, a recent large, well-conducted study of fatally injured drivers by Drummer *et al*. [11] suggested that those who tested positive for marijuana were almost three times more likely to be culpable for the crash. Our odds ratio for this relationship is close to 1 and the confidence intervals are wide, so we cannot rule out an effect. Furthermore, these risky driving variables may be on the causal pathway between acute marijuana use and car crash injury; that is, because drivers are under the influence of marijuana, they may be less likely to wear seat-belts and more

likely to be sleepy and drive at higher speeds due to the influence of the drug. In this case, it may be inappropriate to adjust for these variables [26]. In particular, sleepiness is known to be an acute effect of marijuana use [27]. It would be appropriate to test for an association between sleepiness, risky driving and marijuana use in the control group, but because of small numbers of controls with positive risk taking outcomes in this study we were not able to do so. Other studies have also indicated that there may be a relationship between marijuana use, risky driving and car crash injury [16,28] and this area needs further research.

This study has several limitations and the results may be subject to bias. We relied on a retrospective, self-reported measure of marijuana consumption in the past 3 hours and did not differentiate between levels of consumption. We also did not measure marijuana levels in the body or any resulting impairment. This may have produced an inaccurate result for marijuana consumption or resulted in differential reporting of marijuana consumption for cases and controls. It is difficult to estimate to what extent and in what direction this may have biased our results. It will remain a challenge for future investigators to obtain an accurate measure of both marijuana consumption and impairment within the short time window available following a crash (or roadside survey) in which these variables can be measured. Although the amount of missing data in this study were small, there were more missing data for cases than controls. If cases with missing data were more likely to have been using marijuana than cases who answered the question, then we may have underestimated the effect of marijuana use on car crash injury. The response rate in cases was also higher than that for controls, and although there were no differences in age, gender or driving conditions between case and control non-responders we were not able to assess differences in marijuana use by case–control status in non-responders. The result may be subject to selection bias as a result of these missing participants.

CONCLUSIONS

This population-based case–control study suggests that habitual marijuana use is associated with a 10-fold increase in the risk of car crash injury. The relationship between both habitual and acute marijuana use and car crashes is complex and is likely to be related to other risk-taking behaviours, particularly risky driving. Because of the challenges involved in conducting high-quality research into these relationships, converging evidence from a variety of sources is required before potentially costly policy and practice decisions are made. In this study the prevalence of self-reported recent marijuana use in the Auckland driving population was less than 1%, and those who did use marijuana prior to driving were highly likely to be habitual users. This suggests that interventions targeting high-risk marijuana use groups may be more cost-effective than general population interventions such as random roadside testing.

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