Cannabis use as a risk factor for causing motor vehicle crashes: a prospective study

Jeffrey R. Brubacher[1] 🔘, Herbert Chan[2], Shannon Erdelyi[2], Scott Macdonald[3], Mark Asbridge[4], Robert E. Mann[5], Jeffrey Eppler[6], Adam Lund[7], Andrew MacPherson[8], Walter Martz[9], William E. Schreiber[2], Rollin Brant[2] & Roy A. Purssell[2]

Vancouver General Hospital and University of British Columbia, Vancouver, BC, Canada,[1] University of British Columbia, Vancouver, BC, Canada,[2] University of Victoria, Victoria, BC, Canada,[3] Dalhousie University, Halifax, Nova Scotia, Canada,[4] Centre for Addiction and Mental Health, Toronto and University of Toronto, Toronto, Ontario, Canada,[5] Kelowna General Hospital and University of British Columbia, Kelowna, BC, Canada,[6] Royal Columbian Hospital and University of British Columbia, New Westminster, BC, Canada,[7] Victoria General Hospital and University of British Columbia, Victoria, BC, Canada[8] and Institute of Forensic Medicine, Justus Liebig University, Giessen, Germany[9]

ABSTRACT

Aim We conducted a responsibility analysis to determine whether drivers injured in motor vehicle collisions who test positive for Δ-9-tetrahydrocannabinol (THC) or other drugs are more likely to have contributed to the crash than those who test negative. **Design** Prospective case–control study. **Setting** Trauma centres in British Columbia, Canada. **Participants** Injured drivers who required blood tests for clinical purposes following a motor vehicle collision. **Measurements** Excess whole blood remaining after clinical use was obtained and broad-spectrum toxicology testing performed. The analysis quantified alcohol and THC and gave semiquantitative levels of other impairing drugs and medications. Police crash reports were analysed to determine which drivers contributed to the crash (responsible) and which were 'innocently involved' (non-responsible). We used unconditional logistic regression to determine the likelihood (odds ratio: OR) of crash responsibility in drivers with $0 < THC < 2$ ng/ml, 2 ng/ml $\leq THC < 5$ ng/ml and $THC \geq 5$ ng/ml (all versus $THC = 0$ ng/ml). Risk estimates were adjusted for age, sex and presence of other impairing substances. **Findings** We obtained toxicology results on 3005 injured drivers and police reports on 2318. Alcohol was detected in 14.4% of drivers, THC in 8.3%, other drugs in 8.9% and sedating medications in 19.8%. There was no increased risk of crash responsibility in drivers with $THC < 2$ ng/ml or $2 \leq THC < 5$ ng/ml. In drivers with $THC \geq 5$ ng/ml, the adjusted OR was 1.74 [95% confidence interval (CI) = 0.59–6.36; $P = 0.35$]. There was significantly increased risk of crash responsibility in drivers with blood alcohol concentration (BAC) $\geq 0.08\%$ (OR = 6.00; 95% CI = 3.87–9.75; $P < 0.01$), other recreational drugs detected (OR = 1.82; 95% CI = 1.21–2.80; $P < 0.01$) or sedating medications detected (OR = 1.45; 95% CI = 1.11–1.91; $P < 0.01$). **Conclusions** In this sample of non-fatally injured motor vehicle drivers in British Columbia, Canada, there was no evidence of increased crash risk in drivers with Δ-9-tetrahydrocannabinol < 5 ng/ml and a statistically non-significant increased risk of crash responsibility (odds ratio = 1.74) in drivers with Δ-9-tetrahydrocannabinol ≥ 5 ng/ml.

Keywords Alcohol, cannabis, drugs, motor vehicle crash, *per se* limits, tetrahydrocannabinol.

INTRODUCTION

The legal status of cannabis is changing rapidly. Cannabis has been legal for medical use in Canada since 2001, and 25 US States have legalized or decriminalized medical cannabis [1]. At present, four US states and several countries have gone further and legalized cannabis for recreational use. The Canadian government recently legalized the production, possession, distribution and sale of cannabis for recreational use.

Cannabis contains more than 60 cannabinoids, but most impairing effects are caused by Δ-9-tetrahydrocannabinol (THC) [2], the main psychoactive compound. After smoking a 'joint', whole blood THC levels typically peak at

> 100 ng/ml within 15 minutes and then drop rapidly, so that THC is usually < 2 ng/ml within 4 hours after a single acute exposure [3]. Psychotrophic effects typically peak at 20–30 minutes and resolve by 4 hours. Ingesting cannabis delays the onset and extends the duration of effect. The main THC metabolite, 11-nor-9-carboxy-delta-9-tetrahydrocannabinol (THC-COOH), is not psychoactive and persists in blood and urine long after impairment has resolved. Thus, THC-COOH provides evidence of previous cannabis exposure but does not necessarily indicate impairment or recent use. Urine tests for cannabis measure THC-COOH, and cannot confirm recent use [4–12]. THC is also found in oral fluid of cannabis users due to local absorption of THC in the oral cavity during smoking [13,14]. Oral fluid is easier to obtain than blood and is useful for screening [15–17], but THC concentration in oral fluid correlates poorly with blood level or impairment [17–19], and blood is considered to be the best medium for measuring THC in the impairing range [20].

Many North Americans drive after using cannabis [21–23] and there is concern that this practice will increase following legalization, resulting in more crashes due to cannabis impairment. Controlled experiments show that cannabis impairs the psychomotor skills required for safe driving, with participants displaying slower reaction time, impairment in automated tasks such as tracking ability (e.g. staying within a lane) or monitoring a speedometer, impaired divided attention performance, impaired working memory and more errors in simulated driving tests [19,24–29]. However, there is also evidence that cannabis users are aware of their impairment and compensate by driving more slowly, leaving more headway and taking fewer risks [25–27]. Epidemiological evidence is required to understand the 'real-world' crash risk associated with acute cannabis use.

Several recent meta-analyses concluded that cannabis increases crash risk, with estimated odds ratios (ORs) ranging from 1.36 to 2.66 [30,31]. Most studies employed either case–control designs which compare cannabis use in crash-involved drivers with non-crash-involved drivers [32–43] or responsibility analyses which include only crash-involved drivers and compare cannabis use in drivers deemed responsible for the crash versus in those deemed non-responsible [44–51]. Unfortunately, most studies had significant limitations. Cannabis exposure was often based on either presence of THC-COOH or any THC above the limit of detection, neither of which necessarily indicates acute use or impairment. In fact, the most recent review found only five studies that calculated crash risk for drivers with blood THC > 2 ng/ml [31]. All case–control studies had high refusal rates (> 15%), potentially resulting in selection bias if drivers who refused participation had different rates of drug use than those who participated, as is probably the case. In addition, many case–control studies

employed different methods to detect cannabis exposure in cases versus in controls (e.g. blood THC in cases and saliva THC in controls). Another common problem was use of non-comparable controls (e.g. patients visiting hospital for medical problems) to estimate THC use in the general driving population. A responsibility analysis design has several advantages. Because all drivers are involved in a crash, this method minimizes the problem with differential ascertainment of THC in cases versus controls. Furthermore, responsibility analyses typically eliminate bias due to refusals by taking advantage of mandatory THC testing performed as part of routine police [50] or coroner [49] investigation. Responsibility analyses are limited due to the inherent difficulty in retrospectively determining responsibility, combined with the fact that all included drivers 'failed to avoid crashing'. As a result, some 'non-responsible' drivers may differ from the general driving population. Previous responsibility analyses had mean delays of > 3 hours from crash until blood collection for THC measurement [47,49–51], which is important because THC levels decline rapidly after smoking marijuana, so levels measured > 3 hours after a crash will be significantly lower than at the time of the crash [52]. Many responsibility analyses used THC from coroner reports, but interpretation of those levels is complicated by postmortem redistribution of THC [53–55].

A large 2015 case–control study from Virginia warrants comment [43,56]. Researchers accompanied police to 2682 crashes and measured oral fluid THC in crash-involved drivers and in 6190 roadside control drivers, matched for time and place of crash. No associations between THC and crash risk were observed (adjusted OR = 1.00). This study, like all roadside surveys of drug use in drivers, is limited by high refusal rates in both crash-involved drivers (20.4%) and controls (17.7%). Other limitations include use of limit of detection for THC in oral fluid (and therefore inclusion of unimpaired drivers in the THC positive group) and a focus on minor crashes (no injuries in 76.4%), where the prevalence of driver impairment may differ.

As evidence-based legal limits (*per se* limits) are effective in preventing drunk driving, many jurisdictions have set *per se* limits for THC. Unfortunately, given limited evidence, setting evidence-based *per se* levels for THC is challenging. Some experts suggest that many drivers with blood THC > 3 ng/ml [57] or > 3–5 ng/ml [29] have significant impairment and should be prohibited from driving. A recent simulator study suggested that drivers with blood THC > 8.2 ng/ml were as impaired as drivers with blood alcohol content (BAC) > 0.05% [19]. Based on these reports, many jurisdictions, including many US states and Canada, have set THC *per se* limits of 2 or 5 ng/ml. These levels, especially the 2 ng/ml level, have been criticized because they may not indicate impairment, especially in frequent users

who develop tolerance to some THC impairing effects [24,58,59]. In addition, because cannabinoids accumulate in fat, some daily users may have blood THC > 2 ng/ml after a week or more of abstinence [10,60]. Advocates of lower *per se* levels note that THC concentration drops rapidly after smoking, so a driver could be impaired with high THC levels at the time of driving but be below 5 ng/ml several hours later if there is a delay in obtaining blood samples [52], a fact that supports lower *per se* limits for THC.

Better estimates of the crash risk associated with acute cannabis use are required to guide policy, public education, enforcement and resource allocation strategies aimed to prevent impaired driving. Here we report a prospective observational study which quantifies the relationship between acute cannabis use and crash risk while avoiding many limitations of previous research. We specifically study crash risk associated with THC levels of 2–5 ng/ml and > 5 ng/ml.

METHODS

This study was approved by the University of British Columbia research ethics board (REB).

Study design

We studied moderately injured drivers who were treated in hospital after a crash. Moderate injury was defined pragmatically as meaning that bloodwork (blood count or electrolyte measurement) was required for clinical assessment. We used a responsibility analysis design [61,62] and compared THC levels in drivers deemed responsible for the crash (cases) versus in drivers deemed non-responsible (controls). Because we used excess blood remaining after clinical use, and had procedures to protect personal information, the REB approved waiver of consent.

Sampling

We prospectively sampled drivers from seven participating British Columbia (BC) trauma centres (January 2010–July 2016). All injured automobile drivers for whom police crash reports were available and blood samples were obtained as part of clinical care were included. The decision to obtain blood was made by treating physicians based on their assessment of the driver's clinical condition, and not based on suspicion of drug use. Most samples contained whole blood [in ethylenediamine tetraacetic acid (EDTA)] obtained to measure complete blood counts (CBC); the remainder contained plasma that had been obtained to measure electrolytes. Note that excess blood used in this study had not been obtained for toxicology testing and clinicians did not receive the results of drug testing from this study. Research assistants regularly reviewed emergency department records to identify eligible drivers and obtained excess

blood before it was discarded. Blood was frozen for later toxicology analysis. Drivers with minor injuries who did not require bloodwork were excluded. Drivers were also excluded if blood samples were obtained more than 6 hours after the crash, no excess blood remained after clinical use or if police did not investigate the crash. Drivers of motorcycles or commercial vehicles were excluded, because the responsibility tool is not validated for these vehicles.

Health records

We reviewed medical records and recorded basic demographic and medical information as well as all medications given as part of the driver's clinical care prior to phlebotomy. All 'post-crash' medications given prior to phlebotomy were identified by review of paramedic and emergency department nursing notes and accounted for when reporting the medications detected in a driver's blood samples.

Toxicology analysis

Broad-spectrum toxicology testing on whole blood samples was conducted at the BC Provincial Toxicology Centre [63]. Toxicology testing detected alcohol and cannabinoids, other recreational drugs (cocaine, amphetamines including designer drugs and opiates), as well as psychotrophic pharmaceuticals (including antihistamines, benzodiazepines, other hypnotics and sedating antidepressants). The laboratory methods detected opium alkaloids (codeine and morphine), semisynthetic opioids (oxycodone, hydromorphone) and synthetic opioids (methadone, fentanyl). Detection limits were 0.2 ng/ml for THC and 1 ng/ml for other drugs.

Police crash reports

We obtained police reports via probabilistic linkage based on driver's name, age, sex and date of crash. Responsibility for the crash was determined by standardized scoring of police reports by computerized algorithm, using a validated scoring system as reported elsewhere [64]. The algorithm considers seven categories that could contribute to a crash (road conditions, weather, vehicle factors, action of other drivers, the difficulty of the manoeuvre being performed at time of the crash, action of the index driver, obedience of road laws and crash configuration). Each category is given a score between 1 and 5 based on factors that police believe contributed to the crash (contributory factors) and/or other standardized data recorded in BC police reports. High total scores (≥ 16) indicate that external factors contributed to the crash and the driver was considered non-responsible. Scores ≤ 13 indicate that the only explanation for the crash lay with the index driver, and the driver is considered responsible. For example, if the police report lists road conditions as a contributory factor, the

driver would receive a score of 5 for road conditions. Conversely, if the police report indicates that the crash occurred on a dry paved road, the score for road conditions would be 1. Drivers with indeterminate scores (14 or 15) were excluded from the analysis. The scoring system does not consider police impression of driver impairment or other 'human condition' factors.

Explanatory variables

We considered the following explanatory factors for crash responsibility: (1) driver age ($<$ 20, 20–30 and $>$ 50 years versus 31–50 years), (2) sex, (3) health authority of the visited hospital (Fraser, Interior, and Vancouver Island versus Vancouver Coastal), (4) THC level ($0 <$ THC < 2 ng/ml, $2 \leq$ THC < 5 ng/ml and THC ≥ 5 ng/ml versus THC $= 0$ ng/ml), (5) BAC level ($0 <$ BAC $< 0.08\%$ and BAC $\geq 0.08\%$ versus BAC $= 0\%$), (6) other recreational drugs detectable (e.g. cocaine, amphetamines) and (7) medications detectable (including benzodiazepines, antidepressants, antipsychotics, tricyclics, Z-drugs and anticonvulsants).

Analysis

For all drivers with a police crash report, we computed a crash responsibility score and categorized the driver as either responsible (1), non-responsible (0) or indeterminate (excluded from analysis) [64]. For each explanatory factor, we computed unadjusted odds ratios (ORs) for responsibility and corresponding 95% confidence intervals (CIs) via univariate logistic regression. To obtain adjusted ORs, we fitted a logistic regression model that included all explanatory factors as predictors.

We also fitted a secondary logistic regression model with THC in ng/ml as a continuous variable and other factors unchanged. We explored the possibility of quadratic and cubic relationships between THC and the log odds of responsibility, but likelihood ratio tests indicated that these higher-order polynomials did not improve the model fit. We also considered a model with log-transformed THC, but this model had only a marginally higher Akaike's information criterion (AIC) than the model without transformation.

In a third model, we examined the interaction between alcohol and cannabis but simplified the categorization of each substance due to insufficient data. Of the drivers who tested positive for both alcohol and cannabis, none of these drivers had THC ≥ 5 ng/ml and $0 <$ BAC < 0.08, so no interaction could be estimated. Furthermore, all the alcohol-impaired drivers with $2 \leq$ THC < 5 ng/ml were classified as responsible, resulting in unreasonably large standard errors. In light of this, our interaction model categorized alcohol as either positive or negative; cannabis as either THC $= 0$ ng/ml, $0 <$ THC < 2 ng/ml, or THC ≥ 2 ng/ml; and all other explanatory factors as

described previously. We used Firth's penalized likelihood to address separability in the model with interaction.

We conducted two sets of sensitivity analyses. First, we excluded drivers whose blood was drawn more than (i) 1 hour, (ii) 2 hours or (iii) 4 hours after the crash. Secondly, we studied the effect of coding indeterminate cases as either (i) responsible or (ii) non-responsible to explore possible bias related to exclusion of these drivers. Alpha < 0.05 was considered statistically significant.
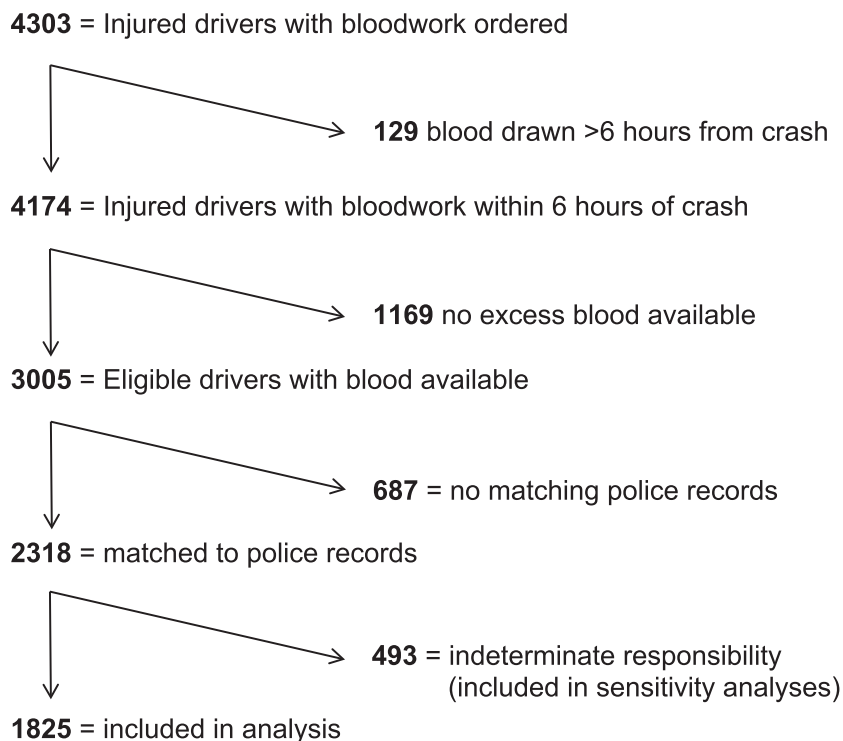
RESULTS

During the course of the study (January 2010 to July 2016), 3005 drivers meeting inclusion criteria presented to a participating hospital and had excess blood available for analysis. Police reports were available for 2318 drivers (Fig. 1). Most drivers (63.2%) were male. The mean age was 44 (range = 16–93); 596 (25.7%) were admitted to hospital (Table 1).

At least one potentially impairing substance was detected in 886 drivers (38.2%). Alcohol was detected in 334 drivers (14.4%), THC in 192 (8.3%), other recreational drugs in 207 (8.9%) and sedating medications in 460 (19.8%). Polysubstance use was common, and many drivers (11.4%) tested positive for more than one impairing substance (Table 2).

Overall, 1178 drivers (50.8%) were deemed responsible for the crash, 647 (27.9%) were not responsible and 493 (21.3%) had indeterminate responsibility. Drivers aged $<$ 20 years were more likely to be responsible than drivers aged 31–50 years (OR = 4.00; 95% CI = 2.14–8.17). There was no difference in responsibility between males and females (OR = 0.99; 95% CI = 0.80–1.22).

There were non-statistically significant increases in unadjusted risk of responsibility for drivers with $0 <$ THC < 2 ng/ml (OR = 1.53; 95% CI = 0.93–2.60), for those with $2 \leq$ THC < 5 ng/ml (OR = 1.59; 95% CI = 0.94–2.82) and for those with THC ≥ 5 ng/ml (OR = 2.29; 95% CI = 0.83–8.01). Unadjusted risks were increased in drivers with THC ≥ 2 ng/ml (OR = 1.72; 95% CI = 1.07–2.87; $P = 0.03$). After adjustment for age, sex and other impairing substances, none of these associations were statistically significant (Table 3, Fig. 2). Sensitivity analyses that included only drivers with blood samples obtained within 1, 2 or 4 hours after the crash yielded comparable results. Additional sensitivity analyses with indeterminates coded as either responsible or non-responsible did not find a statistically significant association between cannabis and responsibility. ORs were smaller when indeterminates were coded as responsible, and larger when they were coded as non-responsible.

With THC modelled as a continuous variable, there was a statistically significant but small increase in unadjusted risk for each 1 ng/ml increase in THC (OR = 1.13; 95%

4303 = Injured drivers with bloodwork ordered

→ **129** blood drawn >6 hours from crash

4174 = Injured drivers with bloodwork within 6 hours of crash

→ **1169** no excess blood available

3005 = Eligible drivers with blood available

→ **687** = no matching police records

2318 = matched to police records

→ **493** = indeterminate responsibility
(included in sensitivity analyses)

1825 = included in analysis

Figure 1 Study flow-chart

Table 1 Characteristics of 2318 drivers with crash reports.

Count (% of total)	All drivers n = 2318 (100%)	Responsible n = 1178 (100%)	Non-responsible n = 647 (100%)	Indeterminate n = 493 (100%)
Age (years)				
Mean (SD)	44 (18)	43 (18)	46 (16)	46 (18)
Range	16, 93	16, 93	17, 89	17, 93
< 20	n = 107 (4.6%)	n = 85 (7.2%)	n = 11 (1.7%)	n = 11 (2.2%)
20–30	n = 553 (23.9%)	n = 308 (26.1%)	n = 130 (20.1%)	n = 115 (23.3%)
31–50	n = 809 (34.9%)	n = 395 (33.5%)	n = 242 (37.4%)	n = 172 (34.9%)
> 50	n = 849 (36.6%)	n = 390 (33.1%)	n = 264 (40.8%)	n = 195 (39.6%)
Male	n = 1466 (63.2%)	n = 766 (65.0%)	n = 397 (61.4%)	n = 303 (61.5%)
Health authority				
Vancouver Coastal	n = 1402 (60.5%)	n = 660 (56.0%)	n = 413 (63.8%)	n = 329 (66.7%)
Fraser	n = 319 (13.8%)	n = 161 (13.7%)	n = 98 (15.1%)	n = 60 (12.2%)
Interior	n = 291 (12.6%)	n = 164 (13.9%)	n = 79 (12.2%)	n = 48 (9.7%)
Vancouver Island	n = 319 (13.8%)	n = 161 (13.7%)	n = 98 (15.1%)	n = 60 (12.2%)
Crash type				
Single-vehicle crash	n = 730 (31.5%)	n = 564 (47.9%)	n = 86 (13.3%)	n = 80 (16.2%)
Night-time crash	n = 859 (37.1%)	n = 473 (40.2%)	n = 233 (36.0%)	n = 153 (31.0%)
SVNC	n = 349 (15.1%)	n = 283 (24.0%)	n = 34 (5.3%)	n = 32 (6.5%)
Admitted	n = 596 (25.7%)	n = 353 (30.0%)	n = 134 (20.7%)	n = 109 (22.1%)
Time from crash to blood draw (min)				
Mean (SD)	101 (64)	100 (66)	104 (63)	98 (57)
Median (IQR)	84 (55)	81 (56)	88 (54)	85 (53)
Within 60 min	n = 557 (24.0%)	n = 311 (26.4%)	n = 128 (19.8%)	n = 118 (23.9%)
60–120 min	n = 1206 (52.0%)	n = 588 (49.9%)	n = 356 (55.0%)	n = 262 (53.1%)
120–240 min	n = 456 (19.7%)	n = 222 (18.8%)	n = 135 (20.9%)	n = 99 (20.1%)

SD = standard deviation; SVNC = single-vehicle night-time crash; IQR = interquartile range.

Table 2 Prevalence of substance use in 2318 drivers with crash reports.

Count (% of total)	All drivers n = 2318 (100%)	Responsible n = 1178 (100%)	Non-responsible n = 647 (100%)	Indeterminate n = 493 (100%)
Cannabis				
THC = 0 ng/ml	n = 2126 (91.7%)	n = 1056 (89.6%)	n = 604 (93.4%)	n = 466 (94.5%)
0 < THC < 2 ng/ml	n = 91 (3.9%)	n = 56 (4.8%)	n = 21 (3.2%)	n = 14 (2.8%)
2 ≤ THC < 5 ng/ml	n = 79 (3.4%)	n = 50 (4.2%)	n = 18 (2.8%)	n = 11 (2.2%)
THC ≥ 5 ng/ml	n = 22 (0.9%)	n = 16 (1.4%)	n = 4 (0.6%)	n = 2 (0.4%)
Alcohol				
BAC = 0%	n = 1984 (85.6%)	n = 920 (78.1%)	n = 614 (94.9%)	n = 450 (91.3%)
0 < BAC < 0.08%	n = 57 (2.5%)	n = 39 (3.3%)	n = 11 (1.7%)	n = 7 (1.4%)
BAC ≥ 0.08%	n = 277 (11.9%)	n = 219 (18.6%)	n = 22 (3.4%)	n = 36 (7.3%)
Cannabis and alcohol				
0 < THC < 2 ng/ml × BAC > 0%	n = 24 (1.0%)	n = 21 (1.8%)	n = 1 (0.2%)	n = 2 (0.4%)
THC ≥ 2 ng/ml × BAC > 0%	n = 24 (1.0%)	n = 21 (1.8%)	n = 1 (0.2%)	n = 2 (0.4%)
Other recreational drugs detectable	n = 207 (8.9%)	n = 139 (11.8%)	n = 34 (5.3%)	n = 34 (6.9%)
Sedating medications detectable	n = 460 (19.8%)	n = 276 (23.4%)	n = 102 (15.8%)	n = 82 (16.6%)
Any substance	n = 886 (38.2%)	n = 574 (48.7%)	n = 164 (25.3%)	n = 148 (30.0%)

THC = Δ-9-tetrahydrocannabinol; BAC = blood alcohol concentration.

CI = 1.03–1.28; $P = 0.03$). However, after adjustment for other predictors, there was no statistically significant association between THC level and risk of responsibility (OR = 1.07; 95% CI = 0.98–1.20; $P = 0.19$)

Drinking drivers had higher odds of being responsible for the crash and the risk increased with higher BAC levels. The adjusted risk was OR = 6.00 (95% CI = 3.87–9.75) for drivers with BAC ≥ 0.08% (Table 3). In the model that included a cannabis and alcohol interaction, ORs for BAC > 0% and THC ≥ 2 ng/ml were 1.62 (95% CI = 0.34–15.7) times larger when both substances were detected compared to the individual effects of alcohol and cannabis alone, but this interaction was not statistically significant ($P = 0.58$). We also found an increased adjusted risk of crash responsibility in drivers who tested positive for sedating medications (OR = 1.45; 95% CI = 1.11–1.91) and in drivers who tested positive for recreational drugs other than marijuana (OR = 1.82; 95% CI = 1.21–2.80)

DISCUSSION

We found no evidence of increased crash risk in moderately injured drivers with THC < 5 ng/ml. For drivers with THC ≥ 5 ng/ml there may be an increased risk of crash responsibility. The best estimate for crash risk in this group was OR = 1.74, but this finding was not statistically significant ($P = 0.35$). Our null findings for THC < 5 ng/ml are consistent with the recent Virginia Beach study, that also investigated non-fatal crashes, and found no evidence of increased risk in drivers with THC > 0 (adjusted OR = 1.0) [43,56]. However, unlike our study, the Virginia Beach study reported presence of THC in oral fluid and did not report crash risk at higher THC levels. We also found that drinking drivers (BAC > 0) who also used cannabis had a

higher risk (OR = 7.3 for 0 < THC < 2 ng/ml; OR = 6.8 for THC ≥ 2 ng/ml) than drinking drivers who did not use cannabis (OR = 4.2), but there was no statistically significant alcohol-cannabis interaction.

Our findings, of a low prevalence of drivers with THC > 5 ng/ml (0.9%), combined with a modest (OR = 1.74) and statistically non-significant risk of crash responsibility, suggest that the impact of cannabis on road safety is relatively small at the present time. However, it is possible that the impact may increase following cannabis legalization if more people drive after using cannabis, especially if this includes occasional users with less tolerance to the impairing effects of cannabis. It is also important to caution that the risk associated with cannabis may be higher in young drivers who have a high crash risk at baseline, or in inexperienced cannabis users who may be less able to compensate for cannabis-induced impairment. Furthermore, our findings do not necessarily apply to fatal crashes where the association with cannabis may be stronger. A recent systematic review, which excluded low-quality studies, reported cannabis-associated risk separately for non-fatal crashes (OR = 1.74; 95% CI = 0.88–3.46) and for fatal crashes (OR = 2.1; 95% CI = 1.31–3.36) [30].

Our findings also suggest that the road safety risk associated with alcohol or with other impairing substances is higher than for cannabis, consistent with conclusions by Sewel *et al.* [25]. In our sample, 14.4% of drivers had been drinking and 11.9% had BAC > 0.08%. The relatively low prevalence of alcohol in this sample is probably explained by the effectiveness of BC traffic laws from 2010 that give police authority to impound the vehicles of drinking drivers at the roadside [65]. Consistent with previous research [66], we found a high risk of crash responsibility in

Table 3 Unadjusted and adjusted risk estimates—this analysis includes the 1825 drivers with determinate responsibility scores; drivers with indeterminate scores ($n = 493$) were excluded from the analysis.

	Driver count (% responsible)	Unadjusted models[1] OR (95% CI)	Adjusted model[2]	Model with THC in ng/ml[3]	Model with interaction[4]
Intercept			1.14 (0.91, 1.43)	1.14 (0.91, 1.43)	1.15 (0.92, 1.43)
Age, years (reference group = drivers aged 31 to 50 years)					
< 20	$n = 96$ (88.5%)	4.73 (2.58, 9.56)***	4.00 (2.14, 8.17)***	3.98 (2.14, 8.14)***	3.79 (2.05, 7.63)***
20–30	$n = 438$ (70.3%)	1.45 (1.12, 1.89)**	1.17 (0.89, 1.54)	1.16 (0.88, 1.53)	1.18 (0.90, 1.55)
> 50	$n = 654$ (59.6%)	0.91 (0.72, 1.13)	0.99 (0.79, 1.25)	1.00 (0.79, 1.26)	0.99 (0.79, 1.25)
Sex: male versus female	$n = 1163$ (65.9%)	1.17 (0.96, 1.43)	0.99 (0.80, 1.22)	0.99 (0.80, 1.22)	0.99 (0.81, 1.23)
Health authority (ref: Vancouver Coastal)	$n = 1073$ (61.5%)				
Fraser	$n = 259$ (62.2%)	1.03 (0.78, 1.36)	0.93 (0.69, 1.25)	0.93 (0.69, 1.25)	0.94 (0.70, 1.26)
Interior	$n = 243$ (67.5%)	1.30 (0.97, 1.75)	1.21 (0.89, 1.65)	1.20 (0.89, 1.64)	1.18 (0.87, 1.61)
Vancouver Island	$n = 250$ (77.2%)	2.12 (1.55, 2.94)***	1.63 (1.17, 2.30)**	1.63 (1.17, 2.30)**	1.60 (1.15, 2.25)**
Cannabis 1 (ref: THC = 0 ng/ml)	$n = 1660$ (63.6%)				
0 < THC < 2 ng/ml	$n = 77$ (72.7%)	1.53 (0.93, 2.60)	1.09 (0.63, 1.92)		
2 ≤ THC < 5 ng/ml	$n = 68$ (73.5%)	1.59 (0.94, 2.82)	1.16 (0.66, 2.13)		
THC ≥ 5 ng/ml	$n = 20$ (80.0%)	2.29 (0.83, 8.01)	1.74 (0.59, 6.36)		
Cannabis 2: THC (ng/ml)		1.13 (1.03, 1.28)*		1.07 (0.98, 1.20)	
Cannabis 3 (ref: THC = 0 ng/ml)	$n = 1660$ (63.6%)				
0 < THC < 2 ng/ml	$n = 77$ (72.7%)	1.53 (0.93, 2.60)			0.99 (0.56, 1.79)
THC ≥ 2 ng/ml	$n = 88$ (75.0%)	1.72 (1.07, 2.87)*			1.15 (0.67, 2.02)
Alcohol 1 (ref: BAC = 0%)	$n = 1534$ (60.0%)				
0 < BAC < 0.08%	$n = 50$ (78.0%)	2.37 (1.24, 4.89)*	1.93 (1.00, 4.04)	1.93 (1.00, 4.04)	
BAC ≥ 0.08%	$n = 241$ (90.9%)	6.64 (4.33, 10.71)***	6.00 (3.87, 9.75)***	6.01 (3.88, 9.77)***	
Alcohol 2: BAC > 0% versus BAC = 0%	$n = 291$ (88.7%)	5.22 (3.63, 7.73)***			4.18 (2.84, 6.34)***
Cannabis 3 × alcohol 2					
0 < THC < 2 ng/ml × BAC > 0%	$n = 22$ (95.5%)				1.75 (0.37, 17.1)
THC ≥ 2 ng/ml × BAC > 0%	$n = 22$ (95.5%)				1.62 (0.34, 15.7)
Other recreational drugs	$n = 173$ (80.3%)	2.41 (1.66, 3.61)***	1.82 (1.21, 2.80)**	1.83 (1.22, 2.80)**	1.79 (1.20, 2.74)**
Sedating medications	$n = 378$ (73.0%)	1.63 (1.28, 2.11)***	1.45 (1.11, 1.91)**	1.46 (1.12, 1.91)**	1.45 (1.11, 1.90)**

*P-value < 0.05; **P-value < 0.01; ***P-value < 0.001. THC = Δ-9-tetrahydrocannabinol; BAC = blood alcohol concentration. [1]Separate logistic regression models for each explanatory factor. The intercept is not shown for these models. [2]Logistic regression with adjustment for age, sex, health authority, cannabis 1, alcohol 1, other recreational drugs and sedating medications. [3]Logistic regression with adjustment for age, sex, health authority, cannabis 2, alcohol 1, other recreational drugs, and sedating medications. [4]Logistic regression with adjustment for age, sex, health authority, cannabis 3, alcohol 2, cannabis 3 × alcohol 2, other recreational drugs and sedating medications.
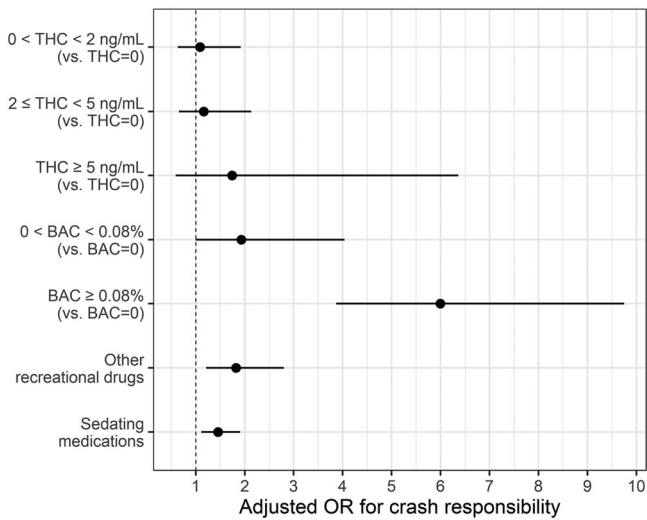
Figure 2 Adjusted odds ratios. This figure shows the risk of crash responsibility for drivers with various ranges of Δ-9-tetra-hydrocannabinol (THC) concentration or blood alcohol concentration (BAC). Risk estimates are adjusted for age, sex, health authority and presence of other impairing substances

drinking drivers (OR = 6.00 for BAC ≥ 0.08%). Sedating medications such as antihistamines or benzodiazepines, and recreational drugs such as cocaine, amphetamines or heroin, are known to impair the psychomotor skills required for safe driving [67,68]. In our study, more drivers tested positive for a sedating medication or for other recreational drugs than for THC, and we found statistically significant increases in responsibility risk in drivers who used recreational drugs other than cannabis (OR = 1.82) and in those who used sedating medications (OR = 1.45).

Interpreting risk estimates from responsibility studies hinges on how responsibility is defined. Modern responsibility studies assign responsibility by objectively scoring detailed crash information, and not according to legal liability [61,64]. Scoring is based on the paradigm of whether the driver should have been able to avoid the crash. In theory, non-responsible drivers are representative of other drivers on the road at the time of the crash and therefore have the same risk factor profile as roadside controls in a standard case–control study [69,70]. If this assumption is true, then responsibility studies should generate higher risk estimates than standard case–control studies [31]. Conversely, all drivers in a responsibility analysis failed to avoid crashing, making it likely that some control drivers (deemed non-responsible) contributed to the crash and should have been classified as cases, a misclassification that would produce lower risk estimates.

STRENGTHS AND LIMITATIONS

Our study has several advantages over previous studies of cannabis and crash risk. We studied moderately injured drivers instead of focusing exclusively on fatal cases. We measured THC in blood (instead of urine or saliva), and obtained samples more than an hour sooner after the crash than previous responsibility studies. Responsibility was

determined by automatic computerized scoring of police reports, eliminating bias that could occur if reviewers were unblinded to toxicology results. Most importantly, because we had REB approval for waiver of consent, we avoided the bias common in standard case–control studies that could arise if drivers who used drugs were more likely to refuse participation.

Our study also has limitations. Although better than previous studies, we had an average delay of 101 minutes between crash and blood draw. In addition, despite a large sample size, only 20 drivers with determinant responsibility scores had THC > 5 ng/ml. Based on a priori power calculations, we would require 51 drivers with THC > 5 ng/ml to have 80% power to detect an OR of 2.5 or higher. Thus, we were underpowered to detect small increases in crash risk in this group of drivers. Although waiver of consent is a strength, the trade-off is that we were unable to interview or assess participants and do not know when they last used cannabis or whether they were impaired. In particular, some drivers with low THC levels may be chronic users who last used many hours previously [10,11] and/or have tolerance to some effects of THC [24,71]. This problem is less likely to be an issue for drivers with higher THC levels (>5 ng/ml), as THC in this range usually represents recent use [24,60]. Finally, our results apply to non-fatally injured drivers whose injuries were severe enough that they required bloodwork and the association between cannabis use and crash responsibility may be different for fatal crashes or property damage only crashes.

CONCLUSIONS

In this multi-site observational study of non-fatally injured drivers we found no increase in crash risk, after adjustment for age, sex and use of other impairing substances, in

drivers with THC < 5 ng/ml. For drivers with THC ≥ 5 ng/ml there may be an increased risk of crash responsibility (OR = 1.74), but this result was statistically non-significant and further study is required. With THC modelled as a continuous variable, there was a statistically significant but small increase in unadjusted risk for each 1 ng/ml increase in THC (OR = 1.13). However, after adjustment for other predictors, there was no statistically significant association between THC level and risk of responsibility. There was significantly increased risk in drivers who had used alcohol, sedating medications or recreational drugs other than cannabis.

Declaration of interests

None.

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