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

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ABSTRACT

Aim We conducted a responsibility analysis to determine whether drivers injured in motor vehicle collisions who test positive for Δ9-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 ≤ THC < 5 ng/ml and THC ≥ 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 ≥ 5 ng/ml. In drivers with THC ≥ 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) ≥ 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 Δ9-tetrahydrocannabinol < 2 ng/ml and a statistically non-significant increased risk of crash responsibility (odds ratio = 1.74) in drivers with Δ9-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 Δ9-tetrahydrocannabinol (THC) [2], the main psychoactive compound. After smoking a ‘joint’, whole blood THC levels typically peak at...
with blood THC probably the case. In addition, many case rates of drug use than those who participated, as is deemed responsible for the crash versus in those deemed crash-involved drivers and compare cannabis use in drivers acute use or impairment. In fact, the most recent review on either presence of THC-COOH or any THC above the

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> 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 psychotropic 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 ≤ THC < 5 ng/ml and THC ≥ 5 ng/ml versus THC = 0 ng/ml), (5) BAC level (0 < BAC < 0.08% and BAC ≥ 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 ≤ 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 ≤ 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. 3).

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%
Table 1 Characteristics of 2318 drivers with crash reports.

| Count (% of total) | All drivers | Responsible | Non-responsible | Indeterminate |
|--------------------|-------------|-------------|----------------|---------------|
| n = 2318 (100%)    | n = 1178 (100%) | n = 647 (100%) | n = 493 (100%) |

Age (years)
- Mean (SD): 44 (18) 43 (18) 46 (16) 46 (18)
- Range: 16, 93 16, 93 17, 89 17, 93

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: n = 730 (31.5%) n = 564 (47.9%) n = 86 (13.3%) n = 80 (16.2%)
- Night-time: 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.

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We also found that crash risk in moderately impaired drivers (BAC ≥ 2 ng/ml) 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 driving 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 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 driving drivers (BAC > 0) who also used cannabis had a
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$^1$ | Adjusted model$^2$ | Model with THC in ng/ml$^3$ | Model with interaction$^4$ |
|-----------------------------|-----------------------|-------------------|-----------------------------|-----------------------------|
| **Intercept**               |                       |                   |                             |                             |
| n = 637 (62.0%)             | 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)** |                       |                   |                             |                             |
| 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)** |                       |                   |                             |                             |
| 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)**  |                       |                   |                             |                             |
| 0 < THC < 2 ng/ml           |                       |                   |                             |                             |
| n = 77 (72.7%)              | 1.53 (0.89, 2.60)     |                             |                             |                             |
| THC ≥ 2 ng/ml               |                       |                   |                             |                             |
| n = 88 (75.0%)              | 1.72 (1.07, 2.87)$^*$ |                             |                             |                             |
| **Alcohol 1 (ref: BAC = 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)$^***$ |                             |                             |                             |
| **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)$^**$     |

$^1$P-value < 0.05; $^2$P-value < 0.01; $^3$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 1, alcohol 2, cannabis 3 × alcohol 2, other recreational drugs and sedating medications.
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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References
1. Marcoux R. M., Larut E. P., Vogenberg F. R. Medical marijuana and related legal aspects. Pharm Ther 2013; 38: 612–9.
2. Huestis M. A. Human cannabinoid pharmacokinetics. Chem Biodivers 2004; 1: 1770–804.
3. Hunault C. C., Bocker K. B., Stellato R. K., Kenemans J. L., de Vries I., Meulenberg J. Acute subjective effects after smoking joints containing up to 69 mg Delta-9-tetrahydrocannabinol in recreational users: a randomized, crossover clinical trial. Psychopharmacology 2014; 231: 4723–33.
4. Grotenhermen F. Cannabinoids. Current Drug Targets - CNS & Neurological Disorders 2005; 6: 507–30.
5. Grotenhermen F. Pharmacology of cannabinoids. Neuroendocrinol Lett 2004; 25: 14–23.
6. Grotenhermen F. Pharmacokinetics and pharmacodynamics of cannabinoids. Clin Pharm 2003; 42: 327–60.
7. Heishman S. J., Huestis M. A., Henningfield J. E., Cone E. J. Acute and residual effects of marijuana: profiles of plasma THC levels, physiological, subjective, and performance measures. Pharmacol Biochem Behav 1990; 37: 561–5.
8. Huestis M. A., Henningfield J. E., Cone E. J. Blood cannabinoids. II. Models for the prediction of time of marijuana exposure from plasma concentrations of delta 9-tetrahydrocannabinol (THC) and 11-nor-9-carboxy-delta 9-tetrahydrocannabinol (THCCOOH). J Anal Toxicol 1992; 16: 283–90.
9. Huestis M. A., Henningfield J. E., Cone E. J. Blood cannabinoids. I. Absorption of THC and formation of 11-OH-THC and THC-COOH during and after smoking marijuana. J Anal Toxicol 1992; 16: 276–82.
10. Bergamaschi M. M., Karschner E. L., Goodwin R. S., Scheidweiler K. B., Hirvonen J., Queziz R. H. et al. Impact of prolonged cannabinoid excretion in chronic daily cannabis smokers’ blood on per se drugged driving laws. Clin Chem 2013; 59: 519–26.
11. Karschner E. L., Schvilke E. W., Lowe R. H., Darwin W. D., Pope H. G., Herning R. et al. Do Delta-9-tetrahydrocannabinol concentrations indicate recent use in chronic cannabis users? Addiction 2009; 104: 2041–8.
12. Skopp G., Richter B., Potsch L. Serum cannabinoid levels 24 to 48 hours after cannabis smoking. Arch Kriminol 2003; 212: 83–95.
13. Huestis M. A., Cone E. J. Relationship of Delta 9-tetrahydrocannabinol concentrations in oral fluid and plasma after controlled administration of smoked cannabis. J Anal Toxicol 2004; 28: 394–9.
14. Kauert G. E., Ramakers J. G., Schneider E., Moeller M. R., Toennes S. W. Pharmacokinetic properties of delta-9-tetrahydrocannabinol in serum and oral fluid. J Anal Toxicol 2007; 31: 288–93.
15. Gjerde H., Langel K., Favretto D., Verstraete A. G. Estimation of equivalent cutoff thresholds in blood and oral fluid for drug prevalence studies. J Anal Toxicol 2014; 38: 92–8.
16. Gjerde H., Langel K., Favretto D., Verstraete A. G. Detection of illicit drugs in oral fluid from drivers as biomarker for drugs in blood. Forens Sci Int 2015; 256: 42–5.
17. Vindenes V., Lund H. M., Andresen W., Gjerde H., Ikdahl S. E., Christophersen A. S. et al. Detection of drugs of abuse in simultaneously collected oral fluid, urine and blood from Norwegian drug drivers. Forens Sci Int 2012; 219: 165–71.
18. Langel K., Gjerde H., Favretto D., Lilsunde P., Øiestad E. L., Ferrara S. D. et al. Comparison of drug concentrations between whole blood and oral fluid. Drug Test Anal 2014; 6: 461–71.
19. Hartman R. L., Brown T. L., Milavetz G., Pierce R. S., Gorelick D. A. et al. Cannabis effects on driving lateral control with and without alcohol. Drug Alcohol Depend 2015; 154: 25–37.
20. The Talloires Report. 2007. Guidelines for Drugged Driving Research. Walsh JM (chairman), Sponsored by The National Institute on Drug Abuse. Available at: https://www.drugabuse.gov/sites/default/files/pdf/talloiresreport.pdf (Archived by WebCite® at http://www.webcitation.org/77fW656U) (accessed 19 March 2019).
21. Berning A., Compton C., Wochinger K. Results of the 2013–2014 National Roadside Survey of Alcohol and Drug Use by Drivers. (Traffic Safety Facts, Research No. DOT HS 812 118) Washington, DC: US Department of Transportation, National Highway Traffic Safety Administration; 2015.
22. Beasley E. E., Beirness D. J. Alcohol and Drug Use Among Drivers Following the Introduction of Immediate Roadside Prohibitions in British Columbia: Findings from the 2012 Roadside Survey. Ottawa: Canadian Centre on Substance Abuse; 2012.
23. Beirness D., Beasley E., McCafferty K. Alcohol and drug use among drivers in Ontario: Findings from the 2014 roadside survey Toronto. Ontario: Ontario Ministry of Transportation; 2015.
24. Desrosiers N. A., Ramakers J. G., Chauzard E., Gorelick D. A., Huestis M. A. Smoked cannabis’ psychomotor and neurocognitive effects in occasional and frequent smokers. J Anal Toxicol 2015; 39: 251–61.
25. Sewell R. A., Poling J., Sofuoglu M., Sewell R. A., Poling J., Sofuoglu M. The effect of cannabis compared with alcohol on driving. Am J Addict 2009; 18: 185–93.
26. Ramakers J. G., Berghaus G., van Laar M., Drummer O. H. Dose related risk of motor vehicle crashes after cannabis use. Drug Alcohol Depend 2004; 73: 109–19.
27. Downey L. A., King R., Papaditiou K., Swann P., Ogden E., Boorman M. et al. The effects of cannabis and alcohol on simulated driving: influences of dose and experience. Accid Anal Prev 2013; 50: 879–86.
28. Berghaus G., Scheer N., Schmidt P., editors. *Effects of Cannabis on Psychomotor Skills and Driving Performance—a Metaanalysis of Experimental Studies*. Adelaide, Australia: International Council on Alcohol Drugs and Traffic Safety (ICADTS); 1995: 60.

29. Grotenhermen F., Leson G., Berghaus G., Drummer O. H., Kruger H. P., Longo M. et al. Developing limits for driving under cannabis. *Addiction* 2007; 102: 1910–7.

30. Asbridge M., Hayden J. A., Cartwright J. L. Acute cannabis consumption and motor vehicle collision risk: systematic review of observational studies and meta-analysis. *BMJ* 2012; 344: e536.

31. Rogeberg O., Elvik R. The effects of cannabis intoxication on motor vehicle collision revisited and revised. *Addiction* 2016; 111: 1348–59.

32. Movig K. L., Mathijssen M. P., Nagel P. H., van Egmond T., de Gier J. J., Leulkenis H. G. et al. Psychoactive substance use and the risk of motor vehicle accidents. *Acad Adv Prev 2004; 36: 631–6.

33. Dussault C., Brault M., Gaulier J. M., Marquet P., Martin-Dupont S. et al. Comparison of the prevalence of alcohol, cannabis and other drugs between 900 injured drivers and 900 control subjects: results of a French collaborative study. *Forens Sci Int* 2003; 133: 79–85.

34. Assum T. The prevalence and relative risk of drink and drug driving in Norway—a case–control study in the Oslo and Bergen areas. Oslo, Norway: Institute of Transport Economics; 2003.

35. Blows S., Ivers R. Q., Connor J., Ameratungu S., Woodward M., Norton R. Marijuana use and car crash injury. *Addiction* 2005; 100: 605–11.

36. Woratatarap P., Insgasithit A., Suriyawongpaisal P., Rattanasiri S., Chatchaipun P., Wattayakorn K. et al. Alcohol, illicit and non-illicit psychoactive drug use and road traffic injury in Thailand: a case-control study. *J Anal Toxicol* 2009; 41: 651–7.

37. Kuypers K. P. C., Legrand S.-A., Ramakers J. G., Verstraete A. G. A. Case-control study estimating accident risk for alcohol, medicines and illegal drugs. *PLoS ONE* 2012; 7: e43496.

38. Hels T., Jyljegård A., Simonsen K. W., Steenfort A., Bernhoff I. M. Risk of severe driver injury by driving with psychoactive substances. *Acad Adv Prev 2013; 59: 346–56.

39. Gjerde H., Christophersen A. S., Normann P. T., Morland J. Associations between substance use among car and van drivers in Norway and fatal injury in road traffic accidents: a case-control study. *Transport Res Part F Traffic Psychol Behav* 2013; 17: 134–44.

40. Li G., Brady J. E., Chen Q. Drug use and fatal motor vehicle crashes: a case–control study. *Acad Adv Prev 2013; 60: 205–10.

41. Romano E., Torres-Saaavedra P., Voss R. B., Lacey J. H. Drugs and alcohol: their relative crash risk. *J Stud Alcohol Drugs* 2014; 75: 56–64.

42. Compton R. P., Berning A. Drug and Alcohol Crash Risk. Report no.: DOT HS 812 117. Washington, DC: National Highway Traffic Safety Administration; 2015.

43. Terhune K. W. An evaluation of responsibility analysis for assessing alcohol and drug crash effects. *Accid Adv Prev 1983; 15: 237–46.

44. Williams A. F., Peat M. A., Crouch D. J., Wells J. K., Finkle B. S. Drugs in fatally injured young male drivers. *Public Health Reps* 1985; 100: 19–25.

45. Terhune K. The incidence and role of drugs in fatally injured drivers. Report. Report no: DOT HS 808 065. Washington: NHTSA, US Department of Transportation; 1992.

46. Lengo M. C., Hunter C. E., Lokan R. J., White J. M., White M. A. The prevalence of alcohol, cannabinoids, benzodiazepines and stimulants amongst injured drivers and their role in driver culpability; part II: the relationship between drug prevalence and drug concentration, and driver culpability. *Accid Adv Prev 2000; 32: 623–32.

47. Lowenstein S. R., Koziel-McLain J. Drugs and traffic crash responsibility: a study of injured motorists in Colorado. *J Trauma Inj Infect Crit Care* 2001; 50: 313–20.

48. Drummer O. H., Gerostamoulos J., Batziris H., Chu M., Caplehorn J., Robertson D. M. et al. The involvement of drugs in drivers of motor vehicles killed in Australian road traffic crashes. *Accid Adv Prev 2004; 36: 239–48.

49. Laumon B., Guadegbeku B., Martin J. L., Biecheler M. B. Cannabis intoxication and fatal road crashes in France: population based case–control study. *BMJ* 2005; 331: 1371.

50. Poulsen H., Moar R., Pirie R. The culpability of drivers killed in New Zealand road crashes and their use of alcohol and other drugs. *Accid Adv Prev 2014; 67: 119–28.

51. Hartman R. L., Brown T. L., Milavetz G., Spurgin A., Gorelick D. A., Gaffney G. R. et al. Effect of blood collection time on measured DELTA9-tetrahydrocannabinol concentrations: implications for driving interpretation and drug policy. *Clin Chem* 2016; 62: 367–77.

52. Brunet B., Huetet T., Hebrard W., Papet Y., Maucou G., Mura P. Postmortem redistribution of THC in the pig. *Int J Leg Med* 2010; 124: 543–9.

53. Holland M. G., Schwope D. M., Stoppacher R., Gillen S. B., Huestis M. A. Postmortem redistribution of Delta9-tetrahydrocannabinol (THC), 11-hydroxy-THC (11-OH-THC), and 11-nor-9-carboxy-THC (THCCOOH). *Forens Sci Int* 2011; 212: 247–51.

54. Lemos N. P., Ingle E. A. Cannabinoids in postmortem toxicology. *J Anal Toxicol* 2013; 37: 394–403.

55. Lacey J. H., Kelley-Baker T., Berning A., Ramírez A., Yao J. et al. Drug and alcohol crash risk: a case–control study. Report. Report no: DOT HS 812 355. Washington, DC: National Highway Traffic Safety Administration; 2016.

56. Vindenes V., Jordbru D., Knapskog A.-B., Kvan E., Mathieson J. Impairment based legislative limits for driving under the influence of non-alcohol drugs in Norway. *Forens Sci Int* 2012; 219: 1–11.

57. Broyd S. J., van Heij H. H., Beale C., Yucel M., Solowij N. Acute effects of cannabis intoxication on neuropsychological functioning of occasional and heavy cannabis users during THC intoxication. *Psychopharmacology* 2012; 220: 341–50.

58. Karschner E. L., Swortwood M. J., Hlinoveny J., Goodwin R. S., Bosker W. M., Ramakers J. G. et al. Extended plasma cannabinoid excretion in chronic frequent cannabis smokers during sustained abstinence and correlation with psychomotor performance. *Drug Test Anal* 2016; 8: 682–9.
61. Robertson M. D., Drummer O. H. Responsibility analysis: a methodology to study the effects of drugs in driving. Accid Anal Prev 1994; 26: 243–7.

62. Lenguerrand E., Martin J. L., Moskal A., Gadegbeku B., Laumon B. Limits of the quasi-induced exposure method when compared with the standard case–control design. Application to the estimation of risks associated with driving under the influence of cannabis or alcohol. Accid Anal Prev 2008; 40: 861–8.

63. Brubacher J. R., Chan H., Martz W., Schreiber W., Asbridge M., Eppler J. et al. Prevalence of alcohol and drug use in injured British Columbia drivers. BMJ Open 2016; 6: e009278.

64. Brubacher J., Chan H., Asbridge M. Development and validation of a crash culpability scoring tool. Traffic Inj Prev 2012; 13: 219–29.

65. Brubacher J. R., Chan H., Brasher P., Erdelyi S., Desapriya E., Asbridge M. et al. Reduction in fatalities, ambulance calls, and hospital admissions for road trauma after implementation of new traffic laws. Am J Public Health 2014; 104: e89–e97.

66. Blomberg R. D., Peck R. C., Moskowitz H., Burns M., Fiorentino D. The Long Beach/Fort Lauderdale relative risk study. J Safety Res 2009; 40: 285–92.

67. Schulze H., Schumacher M., Urmeew R., Alvarez J., Bernholt I. M., de Gier H. et al. Driving under the influence of drugs, alcohol and medicines in Europe—findings from the DRUID project. Lisbon, Portugal: European Monitoring Centre for Drugs and Drug Addiction; 2012.

68. World Health Organization. Drug use and road safety: a policy brief. Geneva, Switzerland: World Health Organization; 2016.

69. af Wahlberg A. E. The relation of non-culpable traffic incidents to bus drivers’ celeration behavior. J Safety Res 2008; 39: 41–6.

70. af Wahlberg A. E., Dorn L. Culpable versus non-culpable traffic accidents; what is wrong with this picture? J Safety Res 2007; 38: 453–9.

71. Ramaekers J. G., Kauert G., Theunissen E. L., Toennes S. W., Moeller M. R. Neurocognitive performance during acute THC intoxication in heavy and occasional cannabis users. J Psychopharmacol 2009; 23: 266–77.