Papers

Cannabis intoxication and fatal road crashes in France: population based case-control study

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Abstract

Objectives To evaluate the relative risk of being responsible for a fatal crash while driving under the influence of cannabis, the prevalence of such drivers within the driving population, and the corresponding share of fatal crashes.

Design Population based case-control study.

Participants 10 748 drivers, with known drug and alcohol concentrations, who were involved in fatal crashes in France from October 2001 to September 2003.

Main outcome measures The cases were the 6766 drivers considered at fault in their crash; the controls were 3006 drivers selected from the 3982 other drivers. Positive detection of cannabis was defined as a blood concentration of Δ^9tetrahydrocannabinol of over 1 ng/ml. The prevalence of positive drivers in the driving population was estimated by standardising controls on drivers not at fault who were involved in crashes resulting in slight injuries.

Results 681 drivers were positive for cannabis (cases 8.8%, controls 2.8%), including 285 with an illegal blood alcohol concentration (≥ 0.5 g/l). Positive cannabis detection was associated with increased risk of responsibility (odds ratio 3.32, 95% confidence interval 2.63 to 4.18). A significant dose effect was identified; the odds ratio increased from 2.18 (1.22 to 3.89) if $0 < \Delta^9$tetrahydrocannabinol < 1 ng/ml to 4.72 (3.04 to 7.33) if Δ^9tetrahydrocannabinol ≥ 5 ng/ml. The effect of cannabis remains significant after adjustment for different cofactors, including alcohol, with which no statistical interaction was observed. The prevalence of cannabis (2.9%) estimated for the driving population is similar to that for alcohol (2.7%). At least 2.5% (1.5% to 3.5%) of fatal crashes were estimated as being attributable to cannabis, compared with 28.6% for alcohol (26.8% to 30.5%).

Conclusions Driving under the influence of cannabis increases the risk of involvement in a crash. However, in France its share in fatal crashes is significantly lower than that associated with positive blood alcohol concentration.

Introduction

Experimental studies have shown that consumption of cannabis diminishes the faculties needed for vehicle driving.[1][2] These effects are sometimes perceptible on driving simulators[3–5] or in real situations.[6][7] Epidemiological studies have often focused on responsibility for a crash; results have varied with respect to the increase in responsibility attributable to cannabis consumption.[8][9] The underlying difficulty is in the absence of a synchronous relation between a change in behaviour and the presence of cannabinoids in the blood or urine.[10] Recent studies have highlighted the importance of focusing analyses on the detection of Δ^9 tetrahydrocannabinol in the blood.[11][12] However, the low number of drivers positive for Δ^9 tetrahydrocannabinol and the common association of cannabis and alcohol hamper the detection of effects entirely attributable to cannabis.[13]

In 1999, before considering changes in drug legislation, the French government wished to obtain reliable epidemiological data, especially on the role of cannabis in the occurrence of crashes. Systematic research was organised in France, from October 2001 to September 2003, into drug consumption in drivers involved in fatal road crashes.

Methods

Study population and drug detection process

We included all fatal crashes resulting in immediate death (including pedestrian fatalities) in the study. All the drivers involved were taken as soon as possible to the hospital, under the control of the police, for compulsory urine testing to detect four major drug families (cannabis, amphetamines, opiates, and cocaine). If the test was positive or impossible a blood sample was taken. This information was associated with the blood alcohol concentration in the police reports.

These reports provided 10 748 drivers who had had full tests for drugs and alcohol. We considered urinary screening for drugs as positive above a concentration of 1000 ng/ml of urine for amphetamines, 300 ng/ml for cocaine and opiates, and 50 ng/ml of acid tetrahydrocannabinol for cannabis. We considered blood tests for drugs (using gas chromatography-mass spectrometry) positive above a concentration of 50 ng/ml for amphetamines and cocaine, 20 ng/ml for opiates, and 1 ng/ml of Δ^9 tetrahydrocannabinol for cannabis. We considered drivers negative if their urine tests were negative or their blood concentrations below these thresholds. However, during the analyses of dose and effect, we no longer considered non-null below threshold concentrations as "negative."

Objectives and study design

We estimated the relative risk of responsibility for fatal crashes while driving under the influence of cannabis and evaluated the corresponding share of fatal crashes. This also implied estimating the prevalence of cannabis in the driving population (drivers not involved in a crash). Under certain conditions, these variables can be estimated from a case-control study.[14]

Cannabis intoxication may favour fatal crash occurrence in two ways: either by increasing the risk of causing a crash (resulting in death), or by increasing the risk of being killed (in a crash caused by another driver) because of greater vulnerability. Our analysis only dealt with the first hypothesis. We considered the

second hypothesis only as a potential source of selection bias of the control group. As such, we evaluated drivers' risk of being responsible for a fatal crash, resulting in their own death or that of another person.

We sought a dose-effect relation between responsibility and cannabis dose, took potential confounding factors into account (focusing specifically on alcohol), and evaluated the representativeness of the cases and controls.

Assessment of responsibility

We determined responsibility of the driver by adapting the method proposed by Robertson and Drummer,[15] which takes into account the different factors liable to reduce driver responsibility. We considered several categories of factors: driving offences, responsibility assignment by the police, factors characterising vehicle and road conditions, and crash types. We also asked experts to evaluate the responsibility for a representative subsample of 2683 drivers in a crash involving two or more vehicles. We carried out these two evaluations without considering alcohol and drug intoxication or related factors, such as sex and age. We therefore compared the two responsibility assessments for this common subsample.

Selection of cases and controls

The cases were the 6766 drivers at fault, including those responsible for their own death. We selected the controls from the 3982 drivers not at fault (a smaller sample because in single vehicle crashes, the driver was generally at fault). In reality, only a comparison with the driving population allowed us to estimate relative risks by using odds ratios (as the event of interest must be a rare event). We therefore used the exposure prevalence estimated from this control group to estimate corresponding attributable risks. As previously found with alcohol,[16] analysis showed a significant increase in the risk of death of drivers not at fault who tested positive to cannabis.[17] Because this phenomenon would lead to a selection bias compared with the driving population—because of the over-representation of crashes in which the only person killed is a driver detected as positive for cannabis (or alcohol)—we excluded the 976 drivers not at fault who were the only fatally injured parties in the crash and therefore selected 3006 controls who were neither at fault nor the only fatally injured party (figure 1).

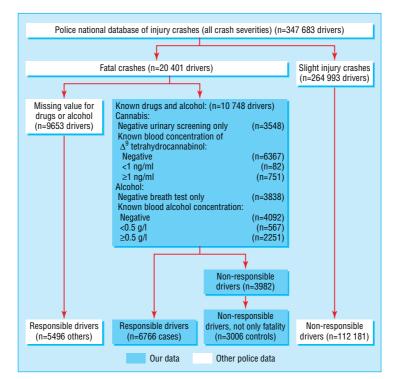### Validation of cases and controls

We used the police national database of injury crashes (all crash severities) to identify the 112 181 drivers not at fault who were involved in a crash resulting in slight injury (a group we assumed best to represent the driving population). We standardised the prevalence for our control group according to cannabis related factors that were found to be significant between the two groups to estimate the prevalence of cannabis in this group and made the same comparison for alcohol. We discussed the representativeness of our controls on the basis of a comparison, on the one hand between these observed and standardised prevalences and on the other hand between these same prevalences for alcohol and those estimated elsewhere for the driving population by using other methods.

We also used this method when comparing our cases to the 5496 drivers at fault who were responsible for a fatal crash, who were not subjected to full drug and alcohol screening.

Confounding factors

The main variable is the positive detection of cannabis. However, considering alcohol simultaneously has a twofold advantage: it takes into account a major confounding factor, and it validates new results on drugs with analogous results on alcohol, already well represented in the literature. A dose-effect is sought for both.

The other cofactors we considered are the other three families of drugs, the driver's age and sex, the type of vehicle driven, and the time of crash. We excluded certain other factors for two reasons: they were not relevant for all vehicle types (such as

Flow of included drivers through the study

wearing a seat belt), or they were the result of the crash (such as the number of vehicles involved), which would lead to over-adjustment. We used unconditional logistic regression to estimate odds ratios.

Estimation of attributable risk

Once we considered the representativeness of our cases and controls acceptable, and given that the relative risks were significant, we then estimated the fraction of fatal crash risks attributable to cannabis and alcohol. We used the bootstrap method to estimate confidence intervals. We calculated attributable risk from estimated odds ratios and prevalence rates for each of 2000 replications. The 95% confidence interval limits were the 2.5th and 97.5th percentiles of the bootstrap distribution, which avoids any normality assumption.[18 19] In the absence of certainty regarding the confounding role of certain cofactors, we propose a double estimation, adjusted for all the cofactors used in the analysis, or on cannabis and alcohol alone (and other drugs, when appropriate). We used SAS, version 9 (SAS Institute Corporation, Cary, NC, 2003) for our analysis.

Results

Of the 9772 drivers studied, 681 were positive for cannabis (7.0%) and 2096 for alcohol (21.4%), including 285 for both (2.9%). The other three families of drugs were, proportionally, more often associated with cannabis than with alcohol (table 1). Men, more often involved in crashes than women, were also more often positive for both cannabis and alcohol, as were the youngest drivers, and users of mopeds and motorcycles. Positive detection was more commonly associated with nighttime crashes.

Agreement between the two responsibility evaluation methods was seen to be satisfactory (κ test 0.71). Table 2 shows the estimated odds ratios of responsibility, according to the Robertson and Drummer method, associated with drugs and alcohol. Cannabis was significantly related to responsibility of the driver. Using expert evaluation did not modify this non-adjusted odds ratio (2.87, 95% confidence interval 2.07 to 3.97).

Amphetamines, cocaine, and alcohol were also significantly related to responsibility, although opiates were not. We highlighted a significant dose effect for cannabis (and for alcohol), adjusted or not for alcohol (respectively for cannabis), shown in table 3 (test for trend for non-null concentrations or Δ^9 tetrahydrocannabinol $P < 0.01$; for non-null blood alcohol concentration $P < 0.001$). These dose effects were taken into account in the rest of the analysis.

Case drivers and control drivers also differ according to sex ($P < 0.05$), age ($P < 0.001$), vehicle type ($P < 0.001$), and time of crash ($P < 0.001$). We performed multivariate analysis to adjust the relation between responsibility and blood concentration of Δ^9 tetrahydrocannabinol for these cofactors, added to blood alcohol concentration, amphetamines, and cocaine. In addition to blood concentrations of Δ^9 tetrahydrocannabinol ($P < 0.001$) and alcohol ($P < 0.001$), shown in table 3, the final model included driver's age ($P < 0.001$), type of vehicle ($P < 0.001$), and time of crash ($P < 0.001$). It excluded amphetamines ($P = 0.20$), cocaine ($P = 0.07$), and driver's sex ($P = 0.08$). Table 4 shows adjusted odds ratios associated with these cofactors, included or not in the final model. No interaction reached significance, particularly that between blood concentrations of Δ^9 tetrahydrocannabinol and alcohol ($P = 0.99$): we estimated the adjusted joint effect corresponding to blood concentrations of both Δ^9 tetrahydrocannabinol and alcohol present at any dose to be 14.0 (8.00

to 24.7), very close to the value obtained from the product of the adjusted individual effects ($1.78 \times 8.51 = 15.1$).

Comparing our controls with drivers not at fault involved in a slight injury crash allowed us to identify their distinguishing characteristics: driver's sex and age; type of vehicle; and place, time, and type of crash. The prevalence of cannabis in our controls was 2.8%, compared with 2.9% when standardised for these variables; both these prevalences were 2.7% for alcohol.

Similarly, comparing our cases with the other drivers responsible for a fatal crash enabled us to estimate the prevalence of cannabis and alcohol for the other drivers: for cannabis, the standardised prevalence was 8.5% compared with 8.8% for the cases, and for alcohol, it was 29.3% compared with 29.8%.

The adjusted fraction of fatal crashes attributable to cannabis (present at any dose) was 2.5% (4.3% when adjusted only for alcohol). The adjusted fraction for alcohol was 29% and 31% when adjusted only for cannabis. When considering only blood

Table 1 Characteristics for all drivers, and for those tested positive for cannabis and for alcohol. Values are numbers (percentages) of drivers

	All drivers (n=9772)	Δ^9 tetrahydrocannabinol ≥1 ng/ml (n=681)	Alcohol ≥0.5 g/l (n=2096)
Blood concentration of drugs:			
Δ^9 tetrahydrocannabinol ≥1 ng/ml	681 (7.0)	681 (100.0)	285 (13.6)
Amphetamines ≥50 ng/ml	47 (0.5)	20 (2.9)	17 (0.8)
Cocaine ≥50 ng/ml	22 (0.2)	9 (1.3)	5 (0.2)
Opiates ≥20 ng/ml	83 (0.8)	23 (3.4)	14 (0.7)
Blood concentration of alcohol ≥0.5 g/l	2096 (21.4)	285 (41.9)	2096 (100.0)
Male sex	8298 (84.9)	643 (94.4)	1951 (93.1)
Female sex	1474 (15.1)	38 (5.6)	145 (6.9)
Age in years:			
≤24	2399 (24.5)	412 (60.5)	628 (30.0)
25 to 34	2379 (24.3)	199 (29.2)	616 (29.4)
35 to 69	4436 (45.4)	66 (9.7)	804 (38.4)
≥70	558 (5.7)	4 (0.6)	48 (2.3)
Vehicle type:			
Moped	276 (2.8)	35 (5.1)	106 (5.1)
Motorcycle	816 (8.4)	73 (10.7)	233 (11.1)
Car	6987 (71.5)	531 (78.0)	1678 (80.1)
Van	329 (3.4)	17 (2.5)	38 (1.8)
Truck	1069 (10.9)	20 (2.9)	23 (1.1)
Other*	295 (3.0)	5 (0.7)	18 (0.9)
Crash time:			
Monday to Friday, daytime	5330 (54.5)	271 (39.8)	581 (27.7)
Saturday, daytime	1059 (10.8)	61 (9.0)	194 (9.3)
Sunday, daytime	974 (10.0)	67 (9.8)	233 (11.1)
Sunday to Thursday, nighttime	1228 (12.6)	123 (18.1)	474 (22.6)
Friday night	240 (2.5)	29 (4.3)	94 (4.5)
Saturday night	941 (9.6)	130 (19.1)	520 (24.8)

*Including 60 bicycles.

Table 2 Drivers' responsibility associated with drugs and alcohol. Values are numbers (percentages) of drivers unless otherwise indicated

	Cases (n=6766)	Controls (n=3006)	Unadjusted odds ratio (95% CI)
Blood concentration of drugs:			
Δ^9 tetrahydrocannabinol ≥1 ng/ml	596 (8.8)	85 (2.8)	3.32 (2.63 to 4.18)
Amphetamines ≥50 ng/ml	42 (0.6)	5 (0.2)	3.75 (1.48 to 9.47)
Cocaine ≥50 ng/ml	20 (0.3)	2 (0.1)	4.44 (1.04 to 19.0)
Opiates ≥20 ng/ml	56 (0.8)	27 (0.9)	0.92 (0.58 to 1.46)
Blood concentration of alcohol ≥0.5 g/l	2016 (29.8)	80 (2.7)	15.5 (12.4 to 19.5)

Table 3 Odds ratios of driver responsibility associated with blood concentration of Δ^9 tetrahydrocannabinol and alcohol

	No of drivers	Odds ratio (95% confidence interval)		
		Unadjusted	Adjusted for alcohol or Δ^9 tetrahydrocannabinol	Multivariate model*
Concentration of Δ^9 tetrahydrocannabinol (ng/ml):				
Negative	9013	1.00	1.00	1.00
<1	78	2.18 (1.22 to 3.89)	1.89 (1.03 to 3.47)	1.57 (0.84 to 2.95)
1 to 2	298	2.54 (1.86 to 3.48)	2.04 (1.47 to 2.84)	1.54 (1.09 to 2.18)
3 to 4	143	3.78 (2.24 to 6.37)	2.78 (1.61 to 4.78)	2.13 (1.22 to 3.73)
≥5	240	4.72 (3.04 to 7.33)	3.06 (1.93 to 4.84)	2.12 (1.32 to 3.38)
Present at any dose	759	3.17 (2.56 to 3.94)	2.37 (1.89 to 2.97)	1.78 (1.40 to 2.25)
Blood concentration of alcohol (g/l):				
Negative	7181	1.00	1.00	1.00
<0.5	495	3.41 (2.67 to 4.35)	3.30 (2.59 to 4.22)	2.70 (2.10 to 3.48)
0.5 to 0.8	211	8.00 (4.80 to 13.4)	7.74 (4.64 to 12.9)	6.29 (3.74 to 10.6)
0.8 to 1.2	304	9.32 (5.91 to 14.7)	8.73 (5.53 to 13.8)	7.56 (4.75 to 12.0)
1.2 to 2.0	739	15.0 (10.4 to 21.6)	14.1 (9.79 to 20.2)	13.2 (9.11 to 19.1)
≥2.0	842	41.8 (24.1 to 72.4)	40.0 (23.1 to 69.4)	39.6 (22.7 to 68.9)
Present at any dose	2591	9.97 (8.44 to 11.8)	9.50 (8.04 to 11.2)	8.51 (7.15 to 10.1)

*Included variables: blood concentration of Δ^9 tetrahydrocannabinol, blood concentration of alcohol, age, vehicle type, time of crash.

Table 4 Adjusted odds ratios of driver responsibility associated with the cofactors of blood concentrations of Δ^9 tetrahydrocannabinol and alcohol, either included in the final model (age, vehicle type, crash time) or not (amphetamines, cocaine, sex). Values are numbers (percentages) of drivers unless otherwise indicated

	Cases (n=6766)	Controls (n=3006)	Odds ratio (95% confidence interval)
Blood concentration of drugs:			
Amphetamines* ≥50 ng/ml	42 (0.6)	5 (0.2)	1.96 (0.73 to 5.27)
Cocaine* ≥50 ng/ml	20 (0.3)	2 (0.1)	4.23 (0.91 to 19.6)
Sex*:			
Male	5780 (85.4)	2518 (83.8)	1.00
Female	986 (14.6)	488 (16.2)	1.12 (0.98 to 1.27)
Age in years			
≤24	1941 (28.7)	458 (15.2)	1.89 (1.65 to 2.16)
25 to 34	1628 (24.1)	751 (25.0)	1.03 (0.91 to 1.16)
35 to 69	2751 (40.7)	1685 (56.1)	1.00
≥70	446 (6.6)	112 (3.7)	2.56 (2.04 to 3.20)
Vehicle type:			
Moped	255 (3.8)	21 (0.7)	3.09 (1.94 to 4.93)
Motorcycle	761 (11.2)	55 (1.8)	5.94 (4.47 to 7.91)
Car	4995 (73.8)	1992 (66.3)	1.00
Van	183 (2.7)	146 (4.9)	0.67 (0.53 to 0.85)
Truck	418 (6.2)	651 (21.7)	0.41 (0.36 to 0.47)
Other	154 (1.5)	141 (4.5)	0.62 (0.49 to 0.80)
Crash time:			
Monday to Friday, daytime	3512 (51.9)	1818 (60.5)	1.00
Saturday, daytime	709 (10.5)	350 (11.6)	0.73 (0.62 to 0.85)
Sunday, daytime	695 (10.3)	279 (9.3)	0.78 (0.66 to 0.92)
Sunday to Thursday, nighttime	917 (13.6)	311 (10.3)	0.92 (0.78 to 1.08)
Friday night	182 (2.7)	58 (1.9)	0.74 (0.53 to 1.05)
Saturday night	751 (11.1)	190 (6.3)	0.75 (0.61 to 0.91)

*Estimates shown when individually added to the final model.

alcohol concentration over 0.5 g/l, it was 25% and 27% respectively (table 5).

Discussion

The risk of responsibility for fatal traffic crashes while driving under the influence of cannabis has a significant dose effect that shows a causal relation between cannabis and crashes.

Strengths of the study

We used a case-control study to overcome the difficulty of finding appropriate controls for the cases involved in road crashes. Cannabis and alcohol prevalences, when standardised according to the factors distributed differently between the controls and drivers not at fault involved in crashes resulting in slight injury, were found to be very close to the raw values. Additionally, the prevalence of alcohol in the controls is comparable to that estimated for the French driving population (about 2.5%)[20] when other methods are used. These similarities help validate the comparison of our controls with the driving population, and in particular the prevalence of cannabis observed as an acceptable estimation for the driving population. This approach, which excludes certain over-represented drivers not at fault from the control group, also helps to answer questions raised by some authors who obtain higher odds ratios when comparing cases to the driving population (not involved in crashes), than when comparing the same cases to drivers not at fault (involved in crashes).[21]

We did not exclude any drivers at fault and therefore took into account the overall increase in cannabis related risk in causing crashes fatal to either the individual or to others. This

explains why we found higher risks than in studies that only include fatally injured drivers.[21 22] The increased fatality risk of drivers not at fault under the influence of cannabis or alcohol is a phenomenon that deserves further investigation. It can be partly explained by greater exposure to the secondary risk of death in a crash, via higher vulnerability (such as the use of motorised two-wheelers), riskier behaviour[23 24] (such as not wearing seat belts), or socio-economic disparities (such as the age of their vehicle), and partly by a reduced ability to avoid a crash.[17]

Likewise, studying only drivers at fault for whom drug and alcohol detection had been carried out could have constituted a biased sample of all drivers responsible for fatal crashes. Standardised and raw estimates of the prevalences of cannabis and alcohol were very close, which shows that differences between our cases and other drivers responsible for fatal crashes have no significant effect on prevalence estimates.

Table 5 Adjusted fractions of attributable risks of fatal crashes associated with blood concentrations of Δ^9 tetrahydrocannabinol and alcohol. Values are percentages (95% confidence intervals)

Substance and concentration	Adjusted for Δ^9 tetrahydrocannabinol or alcohol	Multivariate model*
Δ^9 tetrahydrocannabinol >0.0 ng/ml	4.3 (3.4 to 5.3)	2.5 (1.5 to 3.5)
Alcohol:		
>0.0 g/l	31.2 (29.8 to 32.5)	28.6 (26.8 to 30.5)
≥0.5 g/l	26.8 (25.6 to 28.0)	25.2 (23.5 to 26.9)

*Includes blood concentrations of Δ^9 tetrahydrocannabinol and alcohol, driver's age, type of vehicle, and time of crash.

Consequently, our approach allows the estimation of the fraction of fatal crashes attributable to cannabis, providing that all drivers are taken into account. We had to consider drivers who had negative urine tests without any blood confirmation as negative. This choice is acceptable as the Syva RapidTest, a commonly used urinary test, has a sensitivity of 93% and a specificity of close to 100%.[25] What is more, the odds ratios linked to urine tests alone (1.83, 95% confidence interval: 1.47 to 2.27) and to blood concentrations from tests taken immediately after the crashes (2.85, 2.03 to 4.02)[17] are lower than those estimated here for all drivers. The former concerns drivers who were not killed and does not differentiate between cannabis consumers, not under its influence at the time of crash, and those who were driving really under its influence. The latter mainly concerns fatally injured drivers and therefore evaluates only an increase in risk for this subpopulation, masking the increase in risk linked to cannabis causing the death of its consumer. For most cases, the time elapsed between the crash and blood sampling was less than four hours, indicating that the results are reliable.[25]

We determined responsibility according to the Robertson and Drummer method, as used in many previous studies. This allowed us to estimate responsibility for all drivers involved in injury crashes (and thus validate the cases and controls). The high concordance with the expert responsibility assessment strongly reduced the risk of misclassification.

In other respects, for the subsample of crashes involving two or more drivers, data should also be considered as matched, and conditional logistic regression should be used. Taking these dependences into account would not significantly modify our results.[17]

Comparison with other studies

We used alcohol as a plausibility indicator for the results obtained for cannabis: our study concurs with previous studies on crash risk related to alcohol.[21 22 26 27] We were therefore able to confirm the confounding role of alcohol, although we were not able to highlight any interaction: consumption of both cannabis and alcohol would only multiply the risks related to consumption of either cannabis or alcohol alone, without specific potentiation of the effects of one by the other. This result consolidates several previous experimental and epidemiological studies.[28] The existence of a dose effect gives credence to a causal relation between cannabis and road crashes.

The other cofactors may to some extent lead to over-adjustments.[29 30] This is why odds ratios and attributable risk fractions for cannabis and alcohol are given in the form of a range of values estimated by taking these cofactors into account or by ignoring them: in both cases, consumption of cannabis increases the risk of responsibility for fatal road crashes, while remaining significantly lower than the risk associated with alcohol.

Limitations of the study

It was not possible to perform an adjusted analysis of the effects of amphetamines, cocaine, and opiates, mainly because of the small number of drivers positive for these substances. This highlight, however, that these drugs are not a major issue in France at the moment (2003). Psychoactive medical drugs were only sought in the case of positive blood testing. No further study of this confounding effect was possible.

Conclusions

This study answers many questions left unanswered by previous studies,[21 22 26] in particular by considering what some call an "at fault, not at fault" study[31] as a specific case-control study.

What is already known on this topic

Cannabis consumption, even in low doses, hampers certain faculties necessary for driving a vehicle

Epidemiological studies provide conclusions that are heterogeneous and not robust enough to prove that such consumption represents a crash risk factor of significant magnitude

What this study adds

The risk of responsibility for fatal road crashes while driving under the influence of cannabis has a significant dose effect that shows a causal relation between cannabis and crashes

Reliable estimates of the share of fatal crashes attributable to cannabis and alcohol enable comparison of the respective road safety issues

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However, in addition to the number of deaths linked to the responsibility of drivers, further work should include the share of all deaths attributable to the greater vulnerability of users under the influence of cannabis. This latter calculation was possible for drivers but is yet to be shown for other road users, namely passengers and pedestrians.

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