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Cannabis and crash responsibility while driving below the alcohol per se legal limit



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ABSTRACT

There is a growing interest in how extensively the use of marijuana by drivers relates to crash involvement. While cognitive, lab-based studies are consistent in showing that the use of cannabis impairs driving tasks, epidemiological, field-based studies have been inconclusive regarding whether cannabis use causes an increased risk of accidents. There is ample evidence that the presence of cannabis among drivers with a BAC ≥ 0.08 g/dL highly increases the likelihood of a motor vehicle crash. Less clear, however, is the contribution of cannabis to crash risk when drivers have consumed very little or no alcohol. This effort addresses this gap in knowledge. We took advantage of a unique database that merged fatal crashes in the California Statewide Integrated Traffic Records System (SWITRS) and the Fatality Analysis Reporting System (FARS), which allows for a precise identification of crash responsibility. To account for recent increase in lab testing, we restricted our sample to cover only the years 1993-2009. A total of 4294 drivers were included in the analyses. Descriptive analyses and logistic regressions were run to model the contribution of alcohol and drugs to the likelihood of being responsible in a fatal crash. We found evidence that compared with drivers negative for alcohol and cannabis, the presence of cannabis elevates crash responsibility in fatal crashes among drivers at zero BACs (OR = 1.89) and with 0 < BAC < 0.05 g/dL (OR = 3.42), suggesting that emphasis on curbing impaired driving should not be solely focused on heavy-drinking drivers. Data limitations however caution about the generalizability of study findings. Special efforts to understand the effect of cannabis on fatal crashes, in particular in the absence of alcohol, are needed.

1. Introduction

There is a growing interest in how extensively the use of marijuana by drivers relates to crash involvement (e.g., Dobbs, 2005; Sewell et al., 2009; Rogeberg and Elvik, 2016). Cognitive, lab-based studies are consistent in showing that the use of cannabis impairs driving tasks (Sewell et al., 2009). Epidemiological, field-based studies on the other hand have been inconclusive regarding whether cannabis use causes an increased risk of accidents. Recently, the European Driving Under the Influence of Drugs, Alcohol and Medicines (DRUID) study reported an unadjusted serious or fatal crash risk associated with marijuana use similar to that faced by drivers with a blood alcohol concentration (BAC) between 0.01 g/dL and 0.05 g/dL. These estimates however require some cautions, as difficulties in pooling data from different sources amounted to unavoidable contradictions (Hels et al., 2011). Using information from the 2007 National Roadside Survey to serve as "controls" for the fatal crashes reported in the Fatality Analysis Reporting System (FARS), Li and colleagues reported a crude odds ratio (OR) for marijuana relative to that by non-marijuana users of 1.83 (Li et al., 2013). More recently, Chihuri et al. (2017) also used the matched FARS-NRS databases to report a synergistic contribution of alcohol and cannabis to crash risk. Also, using similar databases, Romano et al. (2014) found that although cannabis was a significant contributor to crash risk when studied alone (OR = 1.55), once adjusted by the presence of alcohol, the crash risk associated with cannabis became nonsignificant. A recent review of previous research on the role of cannabis in motor vehicle crashes including an updated meta-analysis of 21 observational studies, further revealed the studies' heterogeneity of results (Rogeberg and Elvik, 2016). The authors also pointed that such heterogeneity is related to the quality of the data and approach applied in each study, with higher risk estimates usually associated with case-control studies, low study quality, limited control of confounders, medium quality use data, and failure to control for alcohol intoxication (Rogeberg and Elvik, 2016).

The previous discussion suggests that the noted inconsistencies regarding the role of cannabis on crash risk may be at least in part related

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to the quantity of alcohol consumed. There is ample evidence that the presence of cannabis among drivers with a BAC \geq 0.08 g/dL highly increases the likelihood of a serious or fatal motor vehicle crash (e.g., Hels et al., 2011; Li et al., 2013; Romano et al., 2014). Less clear, however, is the contribution of cannabis to crash risk when drivers have consumed very little or no alcohol. Understanding this contribution is relevant to the design of policies targeting marijuana and driving under the influence of drugs (DUID).

Unlike driving under the influence (DUI) of alcohol, which is governed by a well-established legal framework that uses BAC as legal evidence of impaired driving, complexities involving cannabinoid pharmacokinetics, including how cannabinoids are disposed into biological fluids and tissues (Huestis, 2007), have made it difficult to design a legal framework for driving under the influence of marijuana comparable to that for alcohol. Nevertheless, concerns about marijuana and DUID have motivated the promotion of state laws to reduce drugrelated crashes (DuPont et al., 2012; Withers, 2011). Five states have passed per se limits for tetrahydrocannabinol (THC), cannabis's main psychoactive constituent, and 12 states have passed zero-tolerance laws for DUID, establishing that a positive test for marijuana constitutes legal evidence of impaired driving (GHSA, 2016). It is unclear the extent to which these laws account for the complexity of the drug-crash relationship (Reisfield et al., 2012). It is also unclear if in the case of cannabis the deleterious impact of the drug takes place regardless of the level of alcohol consumed. Assessing the contribution of cannabis to crash risk in the absence of alcohol and/or at low BACs would be relevant to policymakers.

Acquiring information on cannabis's contribution to motor vehicle crashes at zero or low BACs not only would be relevant to the design of DUID laws, but also to DUI laws. For instance, if those who use alcohol and other drugs (such as marijuana) in combination were more likely to be involved in crashes for which they are culpable, such outcome would suggest the need for different penalties for alcohol + marijuana DUI drivers.

Currently, all U.S. jurisdictions have a 0.08 per se law in place, making it illegal for a driver to drive with a BAC \geq 0.08 g/dL. There is, however, an ongoing policy debate over whether the legal BAC limit should be lowered to 0.05 g/dL (e.g., Chamberlain and Solomon, 2002; Fell and Voas, 2006). Relevant to this debate is the apparent reduction in crash risk (relative to zero BAC) at BACs between 0.01 g/dL and 0.03 g/dL. This reduction in risk was first reported by Borkenstein et al. (1974) (the "Grand Rapids Dip") and replicated in several other studies (e.g., Blomberg et al., 2005) and appeared to support the conclusion that drivers with small amounts of alcohol were safer drivers than sober drivers.

The validity of such an assertion was questioned by Allsop (1966), Hurst (1973), and Hurst et al. (1994), who argued that such a dip was an artifact-an example of the Simpson Paradox in which correlations within groups are reversed when the groups are combined. Also arguing against the concept of low BAC drivers being safer operators was Marowitz (1996), who examined the recidivism rate (a measure of crash risk) of 53,217 drivers convicted of impaired driving in California between January and June of 1993 and found that the risk of recidivism increased as the arrest BAC declined from 0.09 g/dL to 0.00 g/dL. Thus, drivers convicted of DUI at low BACs were more likely to be rearrested for the same offense than drivers with BACs as high as 0.09 g/dL. To some extent, this rise in recidivism at low BACs relates to the enforcement procedures implemented to apprehend impaired drivers in the United States, which begin with the detection of vehicle maneuvers associated with impaired driving (Stuster, 1997), followed by sobriety tests (Burns, 2003; Burns and Moskowitz, 1977; Stuster, 1997). Despite being stopped under the presumption of drinking and driving, an increasing number of DUI arrested drivers are found to have BACs below 0.08 when a breath test is conducted (Basich, 2015). Also of special interest, therefore, is what caused arrested drivers with low BACs to behave as impaired.

It could be argued that some of the low BAC cases may be due to the delay between arrest and transporting the suspect to the police station for breath testing during which the body eliminates alcohol at approximately 0.10 g/dL to 0.015 g/dL percent per hour (Jones, 2010). Alternatively, however, it is also possible that a non-negligible proportion of low BAC drivers include high-risk drivers for whom alcohol consumption is not the only risk factor. Thus, sources of crash risk other than alcohol would be responsible for the Grand Rapids Dip as well as for the relatively elevated number of arrestees at low BACs. One such unaccounted sources of risk could be drowsiness or fatigue (Corfitsen, 2003). Another possibility is Marowitz's (1996) contention that the zero BAC arrest cases are drug-impaired drivers. This possibility is currently receiving the greatest attention. With the recent surge in states enacting medical marijuana laws and/or legalizing recreational use of cannabis-as well as with the evidence from the 2007 National Roadside Survey (Lacey et al., 2007) that 14.4% of drivers on U.S. roads test positive for a drug-interest has increased in the number of drug-impaired drivers being arrested with current enforcement procedures (DuPont et al., 2012; Voas et al., 2013). The hypothesis that low or zero BAC cases among DUI offenders involve cannabis is at the center of this effort. Although there is some evidence in support of this hypothesis, the evidence is weak. Dubois et al. used the Fatality Analysis Reporting System (FARS) to report that even at BAC = .00 g/dL, the presence of cannabis contributes to crash culpability (Dubois et al., 2015). Unfortunately, by using a proxy for crash responsibility developed from the same database (rather than applying an independent measure of culpability); by excluding drivers younger than 21 years old, an age group with increasing rates of cannabis use (NIDA, 2016) and at elevated crash risk (Peck et al., 2008), and by lumping crashes that occurred all over the United States (failing to account for the severe statebased and annual-based limitations in drug reporting present in the FARS) (e.g., Berning and Smither, 2014; Pollini et al., 2015), the report casts some doubts on their findings.

As a result of a collaboration between the Pacific Institute for Research and Evaluation (PIRE) and the California Department of Motor Vehicles, we took advantage of a unique database merging fatal crashes in the California Statewide Integrated Traffic Records System (SWITRS, maintained by the California Highway Patrol) and the FARS. The merged SWITRS-FARS database allows for a precise identification of crash responsibility, while allowing for a control of the FARS's weaknesses in recording drug use information (Berning and Smither, 2014). By taking advantage of that database, our goal was to evaluate the hypothesis that cannabis use may help explain the relatively high incidence of low and zero BACs among arrested drivers, as well as the Grand Rapids Dip. The relevancy of this aim is apparent, as it should illuminate the role that cannabis plays in fatal crashes, in particular at zero or low BACs.

2. Methods

For this study, we drew extensively from Brar (2012) and took advantage of a unique database merging fatal crashes in the FARS and the SWITRS. The FARS contains data on crashes that resulted in the death of a vehicle occupant or non-motorist within 30 days of the crash. The FARS informs about the victims' actual BAC. In 1982, only 54% of the drivers in the database had been tested for alcohol. That figure climbed to 65% in 2004 (Hedlund et al., 2004). For those with no actual measure available, FARS provides BAC measures developed using a multiple imputation technique by Subramanian (2002). When the driver was not tested for alcohol, we used the imputed measure. Results from drug tests are codified and stored in FARS in three variables, each informing the outcome of the lab test. The following list shows the correspondence between these codes and drug classes in the FARS: 000 (Not Tested for Drugs); 001 (No Drugs Reported/Negative); 100-295 (Narcotics); 300-395 (Depressants); 400-495 (Stimulants); 500-595 (Hallucinogens); 600-695 (Cannabinoids); 700-795 (Phencyclidine/PCP);

800-895 (Anabolic Steroids); 900-995 (Inhalants); 996 (Other Drugs); 997 (Tested for Drugs, Results Unknown); 998 (Tested for Drugs, Drugs Found, Type Unknown/Positive); and 999 (Unknown if Tested/Not Reported). We followed this list. The FARS provides the following seven separate codes for cannabinoids (codes 600-695): "Delta 9," "Hashish Oil," "Hashish," "Marijuana," "Marinol," "Tetrahydrocannabinoid," "THC," and "Cannabinoid, Type Unknown" (NHTSA, 2011). Presence of any of these codes in one of the three drug variables was considered indicative of the driver being positive for cannabis. Presence of different drugs in the three variables denoted multidrug use. Drug information in FARS should be handled with extreme caution. There is an abysmal lack of consistency across states and over time regarding the types of drugs tested, the testing protocols, and even the criteria to decide who is tested (Berning and Smither, 2014). Regarding cannabis, not every state tests for the drug and not for the same set of analytes (Romano et al., 2017). As just described, the FARS database does not clearly distinguish between delta-9-THC and its metabolites, nor informs about concentration levels.

SWITRS contains data on all police-reported crashes that occurred in California on public roadways. While both of these sources provide data on fatal crashes, it was necessary to access both because neither alone provided all the information needed for the current study. Specifically, only FARS contains drivers' BAC and drug information, and only SWITRS contains information on which driver the investigating law enforcement officer deemed to have caused the crash.

Such a merging allowed an examination of crash culpability (present in the SWITRS) and BAC information (present in the FARS). Data merging was based on the following variables: crash date (year, month, day); crash time (hour, minute); driver age; driver sex; and vehicle model year. The matching was done at the party (driver) level. To be included, a crash had to meet all of the following criteria: (1) involve exactly two vehicles (drivers); (2) have at least one of the drivers identified as at fault; (3) have both drivers identified as validly licensed, with a suspended or revoked license, or unlicensed; and (4) involve only passenger cars or pickup trucks as the driven vehicles. As described by Brar (2012, pp. 7–8), "For purposes of the above selection, a driver

was considered at fault if they were listed as such in SWITRS, and not at fault otherwise. A crash was excluded from the analysis if neither of the two drivers was listed as being at fault. Since SWITRS does not list more than one driver as being at fault in any given crash, no crashes had to be eliminated due to both drivers being considered at fault." Because of limitations in the drug measures available in the FARS (see Berning and Smither, 2014), the examination of data from 1993 to 2009 minimizes the impact that recent interest on cannabis driving may have had on lab results. Aiming to detect all cannabis-positive drivers, labs may have recently improved the screening and confirmation protocols they were using for THC identification on fatally injured drivers (Pollini et al., 2015). Such changes over time should caution the mixing of recent and past years in the FARS database when conducting drug analyses. Because all drug measures were collected by the same state, by focusing only on California data and only from 1993 to 2009, we minimize those potential biases caused by recent variations in lab procedures.

2.1. Data processing

Data for this study come from an effort initiated by Brar (2012), which was an extension and refinement of an earlier study by DeYoung et al. (1997) that focused on examining crash responsibility among licensed and non-licensed drivers of passenger vehicles and light pickups who died in a crash in California from 1987 to 2009. As indicated in the original report, from a sample of 27,483 crashes in the FARS, the authors found that 1229 did not match with the SWITRS. Of the remaining crashes, Brar discarded 16,722 matched records to exclude vehicle body types other than passenger vehicles or light pickups, missing and/or conflicting FARS and SWITRS information (e.g., non-matching vehicle type, or license status); 197 because of missing crash responsibility; and 565 because neither of the drivers were at fault, which left a sample of 8770 crashes involving two vehicles, both of which had to be either passenger vehicles or light pickups, where the crashes could be successfully matched between SWITRS and FARS, and where there was information indicating which of the two drivers was at fault for the crash.

Table	1
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BAC and drug class distribution, and percentage at fault, among	drivers in two-vehicle fatal crashes in California, 1993–2009
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BAC	N	% AF	95% LCI	95% UCI
BAC=.00 (ref)	3,052	59.1%	57.4%	60.9%
.00 <bac<.05< th=""><th>247</th><th>58.7%</th><th>52.3%</th><th>64.9%</th></bac<.05<>	247	58.7%	52.3%	64.9%
.05≤BAC<.08	121	73.6%	64.8%	81.2%
BAC≥.08	874	84.7%	82.1%	87.0%
Total	4,294	64.7%	63.3%	66.2%
Drug Class	N	% AF	95% LCI	95% UCI
Drug Negative (ref)	3,316	61.5%	59.8%	63.2%
Cannabinoids	192	74.5%	67.7%	80.5%
Depressants	46	80.4%	66.1%	90.6%
Narcotic/Analgesics	80	68.8%	57.4%	78.7%
Stimulants	330	77.9%	73.0%	82.2%
Other Drugs	185	70.3%	63.1%	76.8%
More than One	145	81.4%	74.1%	87.4%
Total	4,294	64.7%	63.3%	66.2%

BAC stands for blood alcohol concentration, and it is measured in g/dL. *At fault* indicates a driver was at fault in the crash. Drug classes as identified by the FARS manual (NHTSA, 2011). *More than One* denotes a driver testing positive for drugs in more than one class. *%AF, 95% LCI*, and *95% UCI* denote percent of drivers at fault, and its respective 95% confidence interval's lower and upper limit, respectively. Cells with %AF in bold indicate significant difference ($\alpha = 0.05$) with the %AF of the respective reference levels (indicated as 'ref'). For instance, in each year the %AF among BAC ≥ 0.08 g/dL was higher than among the %AF among BAC = 0.00 g/dL. Significant ($\alpha = 0.05$) comparisons between drug classes and 0.00 < BAC < 0.05 are indicated by highlighted grey cells. Thus, the %AF among those positive for cannabinoids, depressants, stimulants, or more than one drug was significantly higher than the %AF among drivers at 0.00 g/dL < BAC < 0.05 g/dL (as well than drivers at BAC = 0.00 g/dL).

For the present study, further reductions in the sample was deemed necessary. Drug information among fatally-injured Californian drivers started to be recorded in the FARS in 1993. Because they were noninformative for this study, we deleted the 1987-1992 years from the SWITRS, leaving 5664 crashes (11,328 drivers) in the 1993-2009 SWITRS-FARS database. Next, of the 11,328 drivers in the selected file, only 4294 had a known lab test result. These drivers constitute the final sample used for this study. Of particular notice is that because of the need to include only drivers with a known lab test result, only 19% of the two-car crashes in the original sample (n = 693) have drug information available for drivers. Also of notice is the decision not to add recent (after 2009) FARS and SWITRS years to the study. This decision was based on the recent surge in interest for testing the presence of cannabis among drivers, which may have induced states to change (enhance the detection capabilities) of testing protocols among crashed drivers (Pollini et al., 2015). Unfortunately, as Pollini et al. (2015) pointed out, these changes in lab testing protocols have not been documented. Because of such data uncertainty, we considered the use of recent years of FARS data for drug-related analyses questionable.

2.2. Analyses

We examined BAC and drug class distribution among crash responsible and non-responsible drivers in each of the years under study. Following the FARS coding criterion, the following seven drug classes were examined: Cannabinoids, Depressants, Narcotic/Analgesics, Stimulants, Other Drugs (i.e., drugs not included in any of the other classes), More than One (i.e., drivers who tested positive for more than one class), and Drug Negative. Next we examined the distribution of drivers testing positive for cannabis in each of the four BAC groups under study (BAC = 0.00 g/dL, 0.00 g/dL < BAC < 0.05 g/dL, $0.05 \text{ g/dL} \leq \text{BAC} < 0.08 \text{ g/dL}$, and BAC $\geq 0.08 \text{ g/dL}$), separated by their crash culpability. In this analysis, drivers were considered positive for cannabis if they were included either in the drug class Cannabinoids, or in the More than One class and Cannabinoids was one of them. Comparisons were made using 95% confidence intervals (CIs).

We also ran a logistic regression to model the contribution of alcohol and drugs to the likelihood of being responsible in a fatal crash, adjusted by driver gender and age (aged < 21, 21–34, 35–54, and 55+). To facilitate the interpretation of results, the otherwise continuous BAC and age variables were categorized into levels of specific interest. To avoid unnecessary data partition and because of our focus on cannabis, drug classes were further collapsed into the following three levels: positive for cannabis (i.e., either alone or in combination with other drugs); positive for any other drug; and drug negative. These three classes were then combined with the four BAC groups to yield a 12-level drug and alcohol variable. Being at the same time negative for alcohol and drugs was the reference level. We used SAS v9.4 (SAS Institute, Cary, NC) to conduct the analyses.

3. Results

Table 1 shows the BAC and drug class distribution of drivers in the file, indicating the proportion of them that were at fault. Overall, the proportion of drivers at fault (%AF) increases with BAC, with drivers at BAC ≥ 0.08 g/dL being significantly higher than drivers at 0.05 < BAC < 0.08 g/dL ($\alpha = 0.05$), with both these drivers more likely to be at faults than drivers at BAC = 0.00 g/dL. Compared to those at BAC = 0.00 g/dL, there is a decrease in %AF drivers at 0.00 g/dL < BAC < 0.05 g/dL. Such a dip however, is not statistically significant. Among drug classes, analysis for the 1993–2009 period shows that compared with drug-negative drivers, the %AF among drivers positive for cannabinoids, depressants, stimulants, or multi-drug users was significantly higher ($\alpha = 0.05$).

Table 1 also allows comparisons in the %AF between drug classes and BACs. Highlighted grey cells within each year indicate significant Ū

BAC	Positiv	<i>i</i> e for Cannab	is		Positive	for Other Dru	60		Drug Neg	ative			IIA			
	z	%AF	L95CI	U95CI	z	%AF	L95CI	U95CI	N	%AF	L95CI	U95CI	N	%AF	L95CI	U95CI
BAC = 0.00 (ref)	101	63.4%	53.2%	72.7%	468	73.1%	68.8%	77.1%	2483	56.3%	54.4%	58.3%	3052	59.1%	57.4%	60.9%
0.00 < BAC < 0.05	16	75.0%	47.6%	92.7%	85	67.1%	56.0%	76.9%	146	52.1%	43.6%	60.4%	247	58.7%	52.3%	64.9%
$0.05 \le BAC < 0.08$	12	91.7%	61.5%	99.8%	38	81.6%	65.7%	92.3%	71	66.2%	54.0%	77.0%	121	73.6%	64.8%	81.2%
BAC ≥ 0.08	63	88.9%	78.4%	95.4%	195	85.6%	79.9%	90.2%	616	83.9%	80.8%	86.7%	874	84.7%	82.1%	87.0%
All	192	74.5%	67.7%	80.5%	786	76.0%	72.8%	78.9%	3316	61.5%	59.8%	63.2%	4294	64.7%	63.3%	66.2%
			-		-			-			0000000		-	-		
5AC stands for blood alcol.	vol concent	tration, and it	is measured in	g/dL. At fault	indicates a c	anver was at r	ault in the cras	h. Drug classe	s as identified	DV The FAKS I	nanual (NH15)	A 2011) (ret)	indicates the I	reference level	WAF. 95%LUI	30%

Percentage of drivers at fault among drivers involved in two-vehicle fatal crashes in California, 1933–2009, who were positive for marijuana, other drugs, or drug negative.

denote percent of drivers at fault, and its respective 95% confidence interval's lower and upper limit, respectively. Cells in bold indicate statistically significant difference with drivers at BAC = 0.00 g/dL

1 1

Table 2

differences in %AF ($\alpha = 0.05$) between drivers positive for drug classes and drivers at different BACs. Analysis for the 1993–2009 period shows that the %AF ($\alpha = 0.05$) among drivers positive for cannabinoids, depressants, stimulants, or more than one drug was significantly higher than the %AF for drivers at BAC = 0.00 g/dL or 0.00 g/dL < BA-C < 0.05 g/dL. Conversely, the %AF among drivers at BAC \geq 0.08 g/ dL was higher than the %AF among drivers positive for any drug, although such a difference was statistically significant only for cannabinoids, narcotic/analgesics, and other drugs.

Table 2 shows the percent at fault among fatally injured drivers positive for cannabis and other drugs at different BACs. Regardless of drug presence, the %AF tend to increase with BAC, with the largest at BAC ≥ 0.08 g/dL. The "dip" in %AF noticed among all drivers at 0.00 g/dL < BAC < 0.05 g/dL relative to the %AF of drivers at BA-C = 0.00 g/dL is also noticeable among drug-negative drivers, although such a dip was not statistically significant either. For those positive for cannabis and other drugs, such a "dip" does not occur.

Fig. 1 examines the percentage of drivers positive for cannabis by BAC group and crash responsibility. For instance, among drivers at BAC = 0.00 g/dL, 4.1% of the drivers not-at-fault and 6.7% of those at fault were positive for cannabis. As illustrated in Fig. 1, the presence of cannabis by BAC among at fault and not at fault drivers appears to follow opposite patterns. Among those not at fault, the prevalence of cannabis increases with drivers' BAC, being higher at BAC \geq 0.08 gdL. Among at fault drivers, the prevalence of cannabis is higher at intermediate BACs than at the extremes, albeit not statistically different. The elevated prevalence of cannabis among at fault drivers at intermediate BACs also provides some support to the hypothesis that the use of cannabis contributes to crash responsibility in crashes in which the responsibility of alcohol is under the legal threshold (BAC \leq 0.08 g/dL).

Regression analysis provided a further examination of drug involvement to crash responsibility. As shown in Table 3, regardless of drug presence, the likelihood of finding a driver responsible for the crash is about 5 times higher among drivers with a BAC ≥ 0.08 g/dL than among drivers negative for both alcohol and drugs. Drivers at $0.05 \text{ g/dL} \le \text{BAC} < 0.08 \text{ g/dL}$ who were drug positive also were about 5 times more likely to be responsible for the crash than drivers negative for alcohol and drugs. On the other hand, drivers at 0.05 g/ $dL \le BAC < 0.08 \text{ g/dL}$ who were drug negative were statistically as likely to be at fault as drivers negative for alcohol and drugs. However, the lower limit of the confidence interval for this group was very close to 1, suggesting that the lack of significance we found may have been related to chance. Drivers with 0.00 g/dL < BAC < 0.05 g/dL had a significantly higher culpability odds ratio (OR 3.42 [1.28-916 LCL-UCL]) than drug- and alcohol-negative drivers only when also positive for cannabis.



For drivers with BAC = 0.00 g/dL, having a positive result for drugs other than cannabis also bore a significant relationship to increased crash responsibility. Taken together, these findings provide evidence that marijuana and other drugs in general contribute to increased crash risk.

4. Discussion

The literature provides ample evidence that the mixing of cannabis and alcohol contributes to crash risk (e.g., Li et al., 2013; Peck et al., 1986; Romano et al., 2014). However, in the absence of alcohol or at low BACs, the contribution of cannabis to crash risk is unclear. While cognitive studies have linked the use of cannabis with impairing driving tasks, epidemiological studies on the other hand have yielded inconsistent results (Sewell et al., 2009). As a possible explanation for the lack of consistency, Sewell et al. suggested that cannabis may have a larger deleterious impact on highly automatic driving functions, but a less damaging effect on complex tasks that require conscious control-a pattern that the authors pointed out is the opposite from that which occurs with alcohol. Related to this argument is the suggestion made by Romano and Voas (2011) that the contribution of cannabis to crash risk varies with the type of crash. For instance, it would be reasonable to expect cannabis to contribute differently to the risk of crashes caused by inattention than to those caused by rage or speeding; subsequently differing between fatal and non-fatal crashes. The noted inconsistencies and variations in measuring the impact of cannabis on crash risk suggests the need to avoid generalizations, examining specific driving situations instead.

In agreement with such point of view, our study focuses on the contribution of cannabis to motor vehicle crashes among drivers at zero or low BACs. Our study indicates that, albeit marginally, cannabis contributes to fatal crash responsibility in the absence of alcohol. Our findings also seem to support Marowitz's hypothesis that the Grand Rapids Dip may be at least in part related to drugged driving or particularly cannabis-impaired driving among BAC = 0.00 g/dL drivers. However, caution with this interpretation is also required. The Grand Rapids Dip was first detected during times in which the levels of marijuana consumption were much lower than those registered nowadays. According to Gallup, in 1973 about 12% of Americans admitted to having ever tried marijuana. In 2013, that figure climbed to 38% according to Gallup (Saad, 2013) or 48% according to the Pew Research Center (2013). It is therefore unlikely that cannabis was a significant contributor to the Grand Rapids Dip as it was first noticed by Borkenstein et al. (1974). However, it is reasonable to surmise that the Grand Rapids Dip is not attributable to a single phenomenon, but rather to a variety of factors that may change in prevalence and relevancy over time. In other words, any crash-contributing factor behaving with some

Fig. 1. Percentage of drivers killed in two-vehicle fatal crashes in California that were positive for cannabis—by BAC and crash responsibility (1993–2009).

BAC stands for blood alcohol concentration, and it is measured in g/ dL. *At fault* indicates a driver was at fault in the crash. N denotes sample size. Percentages of drivers positive for cannabis among drivers of different BAC and crash responsibility. For instance, among drivers at BAC = 0.00 g/dL, 4.1% of the drivers not-at-fault and 6.7%of those at fault were positive for cannabis.

Table 3

Odds ratios for being at fault in fatal crashes in California, 1993-2009, according to the driver's recorded drug and alcohol condition.

Odds Ratio (OR) Estimates				
	OR	95% Wald	95% Wald	
		Confidence Limits		
BAC = 0.00 & Marijuana Positive (N = 172) vs BAC & Drug NEGATIVE (N = 2483)	1.89	1.34	2.66	
BAC = 0.00 & Other Drugs Positive (N = 397) vs BAC & Drug NEGATIVE (N = 2483)	2.14	1.69	2.71	
0.00 < BAC < 0.05 & Drug NEGATIVE (N = 146) vs BAC & Drug NEGATIVE (N = 2483)	0.85	0.60	1.19	
0.00 < BAC < 0.05 & Marijuana Positive (N = 26) vs BAC & Drug NEGATIVE (N = 2483)	3.42	1.28	9.16	
0.00 < BAC < 0.05 & Other Drugs Positive (N = 75) vs BAC & Drug NEGATIVE (N = 2483)	1.46	0.90	2.36	
$0.05 \le BAC < 0.08 \& Drug NEGATIVE (N = 71) vs BAC \& Drug NEGATIVE (N = 2483)$	1.60	0.97	2.65	
$0.05 \le BAC < 0.08$ & Marijuana Positive (N = 16) vs BAC & Drug NEGATIVE (N = 2483)	5.55	1.25	24.60	
$0.05 \le BAC < 0.08$ & Other Drugs Positive (N = 34) vs BAC & Drug NEGATIVE (N = 2483)	4.26	1.75	10.36	
BAC \geq 0.08 Drug NEGATIVE (N = 616) vs BAC & Drug NEGATIVE (N = 2483)	4.48	3.54	5.67	
BAC \geq 0.08 Marijuana Positive (N = 81) vs BAC & Drug NEGATIVE (N = 2483)	5.49	2.88	10.47	
BAC \geq 0.08 Other Drugs Positive (N = 177) vs BAC & Drug NEGATIVE (N = 2483)	5.44	3.49	8.48	
Aged 20 or less (N = 608) vs Aged 35-54 (N = 1222)	2.11	1.69	2.63	
Aged 21-34 (N = 1417) vs Aged 35-54 (N = 1222)	1.22	1.03	1.44	
Aged 55 or more (N = 1047) vs Aged 35-54 (N = 1222)	1.56	1.30	1.86	
Female (N = 1319) vs Male (N = 2975)	1.03	0.90	1.18	

BAC stands for blood alcohol concentration, measured in g/dL. Other drugs positive denotes a driver positive for a drug other than cannabis or alcohol. NA denotes too small a sample for meaningful odds ratio estimates. Cells in bold denote a statistical significant difference in the likelihood of being at fault between the BAC/drug combination and being negative for both BAC and drugs, as well as being of ages other than 35–44 or being female.

independence of alcohol use could elevate the crash responsibility of drivers at BAC = 0.00 g/dL and, if left unaccounted, could generate a dip in crash risk such as that detected by Borkenstein et al. (1974). For example, drugs (Marowitz, 1996), drowsiness (Corfitsen, 2003), or other crash-contributing but not alcohol-related factors (e.g., distractions, texting) could also lead to the generation of an anomaly similar to the Grand Rapids Dip.

Interestingly, our finding that the use of cannabis increases fatal crash responsibility even in the absence of alcohol to some extent contradicts previous studies by this research team and others, which failed to detect a contribution of cannabis to crash risk (e.g., Pollini et al., 2015; Romano and Voas, 2011; Romano et al., 2014). Our previous studies have focused on examining the contribution of cannabis to crash risk without taking crash responsibility into account. However, not every alcohol- or drug-positive driver involved in a crash is responsible for that crash; nor inversely does each alcohol- or drug-negative driver have no crash culpability. Accounting for crash responsibility may have eliminated unwanted noise from the model. It could be argued therefore that by including a measure of crash responsibility into our analyses we caused the contribution of cannabis to fatal crashes to surface.

This interpretation, however, is far from conclusive and open to several considerations. First, we must consider the limitations of the datasets used. Drug information in the FARS database has been shown to present several limitations, in particular sharp and not-well-documented inter-state variations in drug testing that also occurs over time (Berning and Smither, 2014). We attempted to minimize those limitations by working with only one state (California) and using a capped time frame. Nevertheless, it is possible our efforts were not completely successful, particularly if missing drug information in the FARS was not random (an important caveat to consider, since only about 38% of the sample [4294 out of 11,328 drivers] had valid information on drugs). Related to this limitation was our decision to analyze the data by applying a simple logistic regression model rather than with one conditional to the crash in which the original pair of drivers was involved. Only few of these crashes (n = 222) contained valid drug information on both drivers. As expected, conditional logistic regression ran on such a small sample (not shown for brevity), yielded non-significant results, largely because of the reduced sample size. Nevertheless, the limitations associated to the impossibility of using conditional logistic

regression in this study should be noted.

Another limitation of the FARS database is that it presents drug results only as either present or absent. By not informing on drug concentration, some of the results we found may be biased. Lack of clarity regarding the analytes included in the database added to the possibility of bias and made examining their joint effect (polydrug use) inadvisable.

Limitations on the assignment of crash responsibility should not be ignored either. Information on crash responsibility in the SWITRS database comes from police reports. It is possible that errors or prejudice could have affected the accuracy of these reports. Issues such as whether the crash type was a hit-and-run or involved alcohol has been shown to affect the officer's assignment of crash responsibility (Jiang et al., 2012). Evidence obtained from surviving drivers and passengers may also affect officers' assessment (Jiang et al., 2012). For instance, of the 4294 drivers with drug information in the file, 64.7% (2779) were at fault in the crash, with 35.3% (1515) not at fault. As such and coupled with the FARS database having drug information available for drivers in only 19% of the two-car crashes in the original SWITRS sample, the asymmetric distribution of drug test results between at fault and not at fault, may not be indicative of a drug contribution to crash; but of officers' decision to test for drugs only the at fault drivers.

Finally and as suggested by Romano and Voas (2011), the findings of this effort may be relevant only to fatal crashes, for the contribution of cannabis to crash may differ by type crash.

Nevertheless, despite the noted limitations, reliance in fatal crash data for drug driving analyses seems unavoidable for current drug driving studies, since such types of crashes are more likely than others (even those involving injuries) to involve testing for drugs and therefore, showing less severe missing data problems (in particular if non-randomly missing).

In summary, although they should be taken with extreme caution, our findings suggest that i) alcohol remains the main contributor to crash responsibility and efforts to abate this problem should not be reduced; and ii) even if inconclusive and relatively marginal, the contribution of cannabis and factors other than alcohol to fatal crash involvement among drivers at zero and low BAC should not be ignored. The relevance of the later is compounded by the sheer number of zero and low BAC drivers, who are the vast majority of drivers on the road (Kelley-Baker et al., 2013). Studies able to eliminate the limitations that

plagued this effort are needed. To this regard, recent calls to improve the homogeneity and accuracy of the drug information present in the FARS (e.g., Slater et al., 2016; Romano et al., 2017) are important steps towards addressing this issue.

Our study also suggests the need to conduct research on the possible benefits of including a measure of crash responsibility in modelling crash risk estimates, a possibility this study suggests (but not shows) may increase the accuracy of crash risk estimates.

In any case, one of the few inconclusive results coming from this effort is that emphasis on curbing impaired driving should not be solely circumscribed to heavy (BAC $\ge 0.08 \text{ g/dL}$) drinking drivers. To this regard, this study indicates that a better understanding of the role cannabis and other factors play in driving impairment among drivers at low or zero BAC is imperative, in order to implement efficient crash-reduction policies.

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