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Woo, Youngki; Willits, Dale W.; Stohr, Mary K.; Hemmens, Craig; and Hoff, Staci, "Wreck on the Highway: The Intersectionality of Driver Culpability, THC, Other Intoxicants and Fatalities in Washington State" (2019). *Criminal Justice Faculty Publications and Presentations*. 5.
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Wreck on the Highway: The Intersectionality of Driver Culpability, THC, Other Intoxicants and Fatalities in Washington State

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Word count: 5,650 words text + 6 tables x 250 words (each) = 7,150 words

Submission Date: July 16, 2018

ABSTRACT

Objective: Given the legalization of recreational cannabis in 2012 in Washington State and recent mixed results regarding the effects of cannabis on driver safety, we examine the link between Delta-9-Tetrahydrocannabinols (THC) and driver's behavior, including speeding and driver errors which may have contributed to a particular fatal crash.

Methods: The current study utilized data from the Washington State Fatality Analysis Reporting System Analytical File (WA FARS) in years 2008-2016. A series of logistic regression were employed to compare THC positive and negative drivers, as well as drivers who tested positive for other intoxicants.

Results: The results of the study were mixed as Delta-9 THC positively predicted speeding, but not other driver errors. Interestingly, Carboxy THC, a non-psychoactive chemical that can be detected for a longer period of time, was a significant predictor of both speeding and driver errors.

Conclusions: This research further demonstrates that cannabis is a risk factor for fatal crashes, though it is not nearly a risk factor of the same magnitude as alcohol. Additional research is needed to better understand why Carboxy THC is a stronger and more robust predictor of poor driving behavior than Delta-9 THC.

Keywords: Delta-9 THC, Carboxy THC, Speeding, Driver errors, FARS

INTRODUCTION

Considerable research has examined the role of cannabis in traffic safety and crashes, with some research suggesting that cannabis is an important risk factor (*1-4*) and other studies finding little to no relationship between cannabis consumption and traffic crashes (*5,6*). The mixed results are noteworthy, given that early evidence highlighted the link between cannabis consumption and reductions in driving-related skills, including coordination, attention, and reaction time, with greater skill reduction occurring at higher doses. Many of these same early studies noted that drivers under the influence of cannabis were aware of the effects and drive more carefully to compensate (for a review, see *7*). To date, research tends to suggest that cannabis is a “low to medium” level risk factor for motor vehicle crashes (*4*, p.1348), but that cannabis when used in conjunction with alcohol was a much stronger risk factor (*8*).

Much of the prior literature showing this “low to medium” effect on the likelihood of crashing is based on simulated driving programs or road experiments. Yet given the potential increased use of cannabis following legalization in states like Washington, more work remains to be done examining the link between cannabis consumption and specific driving errors and behaviors in actual fatal crashes in a state where blood testing for cannaboids is increasingly common. We attempt to address this gap in the literature. Specifically, we examine the link between positive tests for Delta-9-Tetrahydrocannabinols (THC) and Carboxy-THC and dangerous driving behaviors, including speeding and driver errors using Fatality Analysis Reporting System (FARS) and toxicology results from Washington State. Though FARS data only include fatal crashes (and thus cannot be used to examine the causal connection between cannabis and crashes generally), the identification of links between cannabis consumption and risky driving behaviors would both support prior literature which identifies cannabis use as a crash risk factor and could also highlight the manner in which cannabis use affects driving safety. Based on prior research, we hypothesize that cannabis use, as evident by a positive blood test for THC or Carboxy-THC, increases the likelihood that a driver involved in a fatal crash was speeding or committed other driver errors. Moreover, we hypothesize that the effects of cannabis on these outcomes are greater when used in conjunction with alcohol and/or other drugs.

Background

While more recent epidemiological research typically finds that cannabis consumption is a significant driving impairment factor (*9*), the degree of this effect and its link to THC levels in the bloodstream of drivers is not yet fully established in the research or the law (*10-12*). Much of the current discussion revolves around legal thresholds, with Logan et al. (2016, p. 5) noting that “a quantitative threshold for *per se* laws for THC following cannabis use cannot be scientifically supported.” The unresolved nature of research on when cannabis use results in driver impairment has real world implications. For example, in a recent state appellate court decision in Arizona the judge ruled that a driver with a medical cannabis card who had 26.9 ng/mL of THC in his blood could not be deemed drugged because the medical science about the level of THC needed to impair a driver was not settled among medical practitioners (*13*). The public, as well, is still uncertain about the cannabis-traffic link. For example, in a 2014 roadside survey conducted for the National Highway Traffic Safety Administration by the Pacific Institute for Research and Evaluation in Washington, it was revealed that a majority (61.9%) of drivers who admit they use cannabis believe that cannabis does not adversely affect their driving (*14-15*).

Yet the need for additional research on cannabis and traffic safety is perhaps more pressing than ever, given the increased trend toward cannabis decriminalization and legalization across the United States. Indeed, research documented that the prevalence of cannabis in drivers and especially in fatal crashes was increasing before states began to experiment with recreational cannabis laws (*16-17*). It is difficult to parse out whether this increase in cannabis-involved driving was a reflection of a shift in behavior or a shift in testing, yet research documents some increase in cannabis-positive tests for drivers following the passage of medical cannabis laws (*18-20*). Most of the notable increases in cannabis-positive test results in states with medical cannabis laws occurred in fatal crashes where driver toxicology outcomes are readily available. This is despite other research indicating that medical cannabis laws and the presence of medical marijuana dispensaries were negatively related to traffic fatalities overall (*21*). This is perhaps because research suggests that medical marijuana laws had little to no impact on cannabis use among young adults (*22*).

A considerable amount of cannabis is being sold in states which have legalized recreational cannabis. In Washington State over $1 billion dollars in sales have been made (*23*), with income from taxes and licensing rising to $319 million in 2017 alone (increasing from $189 million in 2016) (*22*, *24*, p.15). Early evidence suggests that at least initially, legalization increased use among minors in Washington State, but not in Colorado (*25, 26*). Still, given the prevalence of young drivers in fatal crashes (*27*) and the potential for increased usage among this population, it is reasonable to infer that recreational marijuana laws could increase the prevalence of drivers under the influence of cannabis. The potential for increased traffic crashes and fatalities was, in fact, a central part of the argument against cannabis legalization in states like Washington (*28*). Importantly, research shows that since legalization the number of cannabis related driving cases, both Delta-9-THC and Carboxy-THC in Washington State, have increased markedly (*29*). To date, however, no research has examined the link between cannabis consumption and risky driving behaviors in a state with legalized recreational cannabis.

METHODS

Data

Data for studying traffic fatalities are commonly drawn from the National Highway Traffic Safety Administration's Fatality Analysis Reporting System (NHTSA FARS), yet Pollini et al. (2015) note that drug information is relatively sparse in the national FARS data (see also *30*). Here, we use data from the Washington State Fatality Analysis Reporting System Analytical File (WA FARS) and supplement it with toxicology outcomes from the Washington State Laboratory on drivers. The FARS data provide information on all fatal crashes in the State of Washington and are organized into person and incident-level records. Unlike the NHTSA FARS, the WA FARS includes specified THC results (Delta-9-THC versus Carboxy-THC) and drug blood levels for all drivers in fatal crashes who were blood tested for intoxicants. Conversely, NHTSA FARS can only be used to identify the presence of unspecified cannabinoids and does not include drug levels (*30*).

For the purposes of our analyses, the fatal crash-involved driver (both surviving and deceased) is the unit of analysis and we merge data about these incidents with data about the toxicology result of the driver. We utilize WA FARS data from January 2008 to December 2016, as blood test derived THC levels are not available before this time period. There were a total of 10,155 individuals of all types involved in fatal crashes in Washington from 2008 to 2016. Among these individuals, 5,931 drivers were involved in fatal crashes, of which 2,432 were only blood tested for intoxicants with 2,425 complete cases.

Driver Error Outcome Variables

The WA FARS data includes a variety of measures that might reflect driver culpabilities, like evidence of speeding, lane deviations, distraction, and other driving errors. Since many of these individual variables occurred relatively infrequently, we combined these measures into two dichotomous variables representing driver culpability: 1) speeding and 2) driver errors (identified by police). The first dependent variable, speeding was dichotomous in nature, but the other outcome, driver errors, contains several sub-categories. For example, in the driver error variable, there were 34 sub-types of driving errors or fault, such as driving in an erratic reckless, negligent manner, or abrupt speed change, driving on the wrong side, improper lane changing, intentional illegal driving on the road shoulder, failure to yield or obey the signal, and overcorrecting. These measures were dummy coded into a dichotomous variable representing driver error.

Covariates of Fatal Crash Risks

In addition to measures of driver error, the WA FARS data also includes information on the presence of alcohol and other drugs in the blood by the driver. Given that prior research has highlighted the confounded nature of cannabis and alcohol (*8, 17*), we include measures of both, as well as of other drugs in order to examine whether Delta-9-THC and Carboxy-THC have an independent and/or contingent relationship with speeding and driver errors. We included two dummy variables that indicate whether the driver tested positive for Delta-9-THC at less than 5 nanograms per mL of blood or 5 or more nanograms per mL of blood (the per se limit in Washington state). In addition, we include a dummy variable measure of Carboxy-THC (hereafter referred to simply as Carboxy) in our analysis. Carboxy results are included in the drug tests for fatal crashes in Washington and are indicative that a person had consumed cannabis, but perhaps not recently, as Carboxy is an non-psychoactive metabolite formed after cannabis consumption which can stay in the bloodstream for a longer period of time (*31*), with some research suggesting Carboxy can be detected up to 30 days after consumption (*29*). Given that all Delta-9-THC positive drivers, regardless of whether they were above or below the per se limit, also include positive results for Carboxy, we constructed a modified dummy variable for Carboxy that was scored a 1 if *only* Carboxy (and *not* Delta-9 THC) were positive in the test results. Thus, a positive result (either below or above the per se limit) for Delta-9-THC indicates recent consumption and potential impairment, while a positive result for Carboxy indicates less recent consumption.

In order to measure alcohol impairment, we include two dummy variables that indicate whether the driver's blood alcohol content (BAC) was greater than or equal to 0.08 (the per se limit in Washington state) or less than 0.08. In addition, we constructed a single dummy variable indicating whether the driver tested positive for other drugs, including narcotics, stimulants, hallucinogens, phencyclidine, inhalants, and other drugs. As a robustness check, we estimated all of the models below using actual Delta-9-THC and BAC levels instead of just the dummy variable indicators. Compared to these results, however, results across all the models using dummy variables were generally better with improved goodness-of-fit logistic models. Thus we only present results with dummy variables of alcohol, Delta-9-THC, and Carboxy in this paper.

We also include a variety of driver characteristics in our modeling strategy. Specifically, we include driver's age (in years), gender (where 1 equals male and female is the reference category), whether the driver had an active license (1= licensed, 0 = unlicensed), and prior traffic convictions (including previous DWIs, driver's license suspensions, and speeding citations over past three years). These variables are included as control variables, as these factors might also be linked to driver speeding and errors. Table 1 presents descriptive statistics for all of the outcome and driver characteristics variables.

TABLE 1 Outcome Measures and Driver Characteristics (*N* = 5,915)[a]

Variable	Mean/%	SD	Range
Outcome Measures			
Speeding	0.25	0.43	0-1
Driving error (identified by police)	0.27	0.45	0-1
Driver Characteristics			
Age (year)	42.45	18.43	16-99
26-35	19.0%		
36-45	15.3%		
46-55	16.2%		
56-65	13.4%		
Over 65	12.7%		
Gender (1 = male)	0.75	0.43	0-1
Unlicensed driver	0.13	0.33	0-1
Driver History (during the previous three years)			
Driving while intoxicated (DWI)	0.03	0.17	0-1
Driver license suspensions	0.19	0.39	0-1
Speeding convictions	0.27	0.44	0-1
Other traffic convictions	0.32	0.47	0-1
Drug and Alcohol Involvement			
Total number of blood tested drivers for intoxication	42.0%		
Alcohol positive			
BAC < .080	0.04	0.18	0-1
BAC ≥ .080	0.19	0.39	0-1
Delta-9-THC positive			
THC < 5.00	0.03	0.17	0-1
THC ≥ 5.00	0.04	0.20	0-1
Carboxy THC positive (without Delta-9-THC)	0.03	0.18	0-1
Other drugs[b] positive (except for cannabinoid)	0.12	0.33	0-1

Note: *a* Drivers in fatal crashes in the state of Washington FARS data, 2008-2016. *b* other drugs include narcotics, depressants, stimulants, hallucinogens, phencyclindine, inhalants, and other unknown forms of drugs.

In addition to these driver characteristics, the WA FARS data includes a number of factors related to the context of fatal crashes. Given that weather, road, and vehicle conditions might also affect driver behavior, we include measures of these variables in our models and crash specific factors as additional control variables. Descriptive statistics for these environmental factors are presented in table 2.

TABLE 2 Environmental Contexts of Fatal Crashes (*N* = 5,915)[a]

Variable	Mean/%	SD	Range
Natural Conditions			
Weather condition			
Clear	71.8%		
Cloudy	12.9%		
Rain	10.9%		
Fog/smoke	2.2%		
Snow	2.2%		
Time of crash (1 = night: 5 A.M. to 5 P.M.)	0.40	0.49	0-1
Road Conditions			
Road alignment (1 = Straight)	0.70	0.46	0-1
Road grade (1 = Level)	0.66	0.47	0-1
Intersection involved	0.27	0.44	0-1
Surface type			
Concrete	11.0%		
Asphalt	85.8%		
Others (brick, slag, stone, etc.)	3.2%		
Surface condition			
Dry	73.8%		
Wet or Water	20.3%		
Snow or Frost	4.3%		
Others (sand, dirt, mud, oil, etc.)	1.6%		
Posted speed limit	46.37	12.67	5-70
Number of traffic lanes in crash	2.46	0.93	1-5
Drivers' Vehicle Conditions			
Vehicle type			
Motorcycle	11.7%		
Medium/heavy truck	5.8%		
Passenger vehicle (sedan, SUV, van, light truck)	80.0%		
Others (bus, motorhome, etc.)	2.6%		
Other External Conditions			
Number of occupants in vehicle	1.51	0.91	1-5
Number of vehicles in crash	1.81	0.81	1-4
Number of non-motorists in crash	0.13	0.34	0-1
Lap and shoulder belt used	0.62	0.49	0-1
Heavy truck involved	0.11	0.32	0-1
Head-on involved	0.20	0.40	0-1
Traffic control device present	0.14	0.35	0-1

Note: *a* Drivers in fatal crashes in the state of Washington FARS data, 2008-2016

Analytic Strategy

Since each of the outcome variables (speeding and driver error) are measured dichotomously, we present a set of two logistic regression models examining main effects, each with robust standard errors. As the WA FARS data includes a number of factors that might be predictive of the outcome variables (see Tables 1 and 2), we utilized a stepwise backward selection process for independent variables, based on prior evidence and literature. We began by estimating two regressions with all independent variables presented in Tables 1 and 2, then removed independent variables that were not significant at the p value less than .10, except for a couple of non-significant variables which have been found and documented as substantial factors related to fatal crashes, such as gender, time of crash, alcohol BAC test positive, and Delta-9-THC/Carboxy/other drugs positive. In order to ensure that our model selection process did not unduly affect our results, we compared the results of our backwards selection models to the full models, which were substantively similar, though more cumbersome to present (available upon request).

Next, in order to explore the possibility of an interactive relationship between Delta-9-THC, Carboxy, alcohol and other drugs, we also estimated a series of interaction models for each outcome variable. In summary, we examined the following interactions: Delta-9-THC by BAC, Carboxy by BAC, Delta-9-THC by Other Drugs, Carboxy by Other Drugs, BAC by Other Drugs, and three-way interactions examining Delta-9-THC by BAC by Other Drugs and Carboxy by BAC by Other Drugs. Following best practices with interaction modeling, we first estimated each potential interaction and two-way interaction in separate models and included all nested two-way interactions in the models containing the three-way interactions (*32*).

Lastly, we also conducted sub-group analyses on 1) drivers who were given a blood test for drugs (n=2,425, accounting for listwise deletion); 2) drivers who were given a blood test for drugs and tested positive for alcohol (n=860); and 3) drivers who were given a blood test for drugs and tested positive for alcohol greater than or equal to 0.08 (n=714). Though the WA FARs data provide more information on chemicals present in the blood than other data, not all drivers in Washington are tested. In the full sample models, all drivers who were not tested would be counted as having not consumed any substances, yet it is possible that some of these untested drivers had in fact used drugs or alcohol. These sub-group models provide a more direct comparison between clean and potentially impaired drivers, though they do so at the expense of omitting a great number of drivers who were likely unimpaired.

RESULTS

Speeding Models

Results for the main effects and the three interaction models where speeding is regressed on driver characteristics, contextual factors, and drug and alcohol involvement are presented in Table 3. The results using all drivers involved in fatal crashes indicate that younger drivers, males, motorcycle drivers, and drivers who test positive for alcohol, Carboxy or Delta-9-THC or other drugs, were more likely to be speeding when involved in a fatal crash than drivers without these risk factors. In addition, driving in poor weather conditions (e.g., fog/smoke and snow) and on curvy wet roads were also risk factors for speeding during fatal crashes. The odds of speeding for drivers who tested positive for Delta-9-THC (over the 5 nanograms per mL per se limit) were 48% more likely to be speeding than those who did not test positive for Delta-9-THC at all, controlling for other factors. There was not a similar effect for those drivers who tested positive for Delta-9-THC at a level less than the per se limit. The presence of Carboxy, however, was also statistically significantly associated with speeding: The odds of speeding for drivers who tested positive for Carboxy were 54% greater than for those who did not, controlling for other factors.

TABLE 3 Logit Models of Drug and Alcohol on Speeding (n = 5,310 drivers from 2008-2016 WA FARS data)

Covariates		Main Effects Model		Interaction Model 1		Interaction Model 2		Interaction Model 3	
		Logit (*Robust SE*)	**OR**	**Logit (*Robust SE*)**	**OR**	**Logit (*Robust SE*)**	**OR**	**Logit (*Robust SE*)**	**OR**
Driver Characteristics									
Age		-.03(.00)	0.97***	-.03(.00)	0.97***	-.03(.00)	0.97***	-.03(.00)	0.97***
Gender (1 = male)		.40(.10)	1.49***	.39(.10)	1.48***	.40(.10)	1.48***	.40(.10)	1.49***
Prior Speeding convictions (in the past three years)		.10(.08)	1.11	.10(.08)	1.11	.11(.08)	1.11	.11(.08)	1.11
Natural Conditions									
Weather condition	*Clear (reference)*	--	--	--	--	--	--	--	--
	Cloudy	.07(.12)	1.07	.07(.12)	1.07	.07(.12)	1.07	.07(.12)	1.07
	Rain	-.33(.17)	0.72*	-.33(.17)	0.72*	-.34(.17)	0.72*	-.34(.17)	0.71*
	Fog/Smoke	.83(.28)	2.29**	.82(.29)	2.28**	.84(.29)	2.31**	.83(.29)	2.28**
	Snow	1.09(.29)	2.98***	1.11(.29)	3.03***	1.11(.29)	3.04***	1.11(.29)	3.04***
Time of crash (1 = night)		.04(.08)	1.04	.03(.08)	1.03	.03(.08)	1.03	.03(.08)	1.03
Road Conditions									
Road alignment (1 = straight)		-.73(.08)	0.48***	-.73(.08)	0.48***	-.73(.08)	0.48***	-.73(.08)	0.48***
Road grade (1 = level)		-.20(.08)	0.82*	.20(.08)	0.82*	-.21(.08)	0.81*	-.20(.08)	0.82*
Surface condition (1 = dry)		-.67(.12)	0.51***	-.66(.12)	0.52***	-.67(.12)	0.51***	-.67(.12)	0.51***
Surface type (1 = Asphalt)		-.17(.12)	0.84	-.17(.12)	0.85	-.17(.12)	0.84	-.17(.12)	0.85
Intersection involved		-.44(.10)	0.65***	-.44(.10)	0.65***	-.43(.10)	0.65***	-.44(.10)	0.65***
Posted speed limit		-.02(.00)	0.98***	-.02(.00)	0.98***	-.02(.00)	0.98***	-.02(.00)	0.98***
Drivers' Vehicle Conditions									
Vehicle type	*Passenger vehicle (Reference)*	--	--	--	--	--	--	--	--
	Heavy truck	-.60(.23)	0.55**	-.59(.23)	0.56*	-.58(.23)	0.56*	-.58(.23)	0.56*
	Motorcycle	1.21(.11)	3.37***	1.21(.12)	3.35***	1.21(.12)	3.36***	1.21(.12)	3.34***
	Others	-.11(.34)	0.89	-.11(.34)	0.90	-.11(.34)	0.90	-.11(.34)	0.89
\Other External Conditions									
Number of occupants in vehicle		.11(.04)	1.11**	.11(.04)	1.12**	.11(.04)	1.12**	.11(.04)	1.12**
Number of vehicles in crash		-.49(.06)	0.62***	-.48(.06)	0.62***	-.49(.06)	0.62***	-.48(.06)	0.62***
Drug and Alcohol Involvement									
BAC < .080		.89(.17)	2.43***	.88(.17)	2.41***	.86(.17)	2.37***	.86(.17)	2.36***
BAC ≥ .080		1.26(.10)	3.51***	1.37(.11)	3.94***	1.36(.11)	3.88***	1.34(.11)	3.84***
THC < 5.00		.12(.20)	1.13	.11(.20)	1.12	.11(.20)	1.11	.11(.20)	1.11

THC ≥ 5.00	.39(.17)	1.48*	.37(.17)	1.45*	.58(.19)	1.78**	.59(.19)	1.80**
Carboxy only without THC	.43(.18)	1.54*	.42(.18)	1.52*	.41(.18)	1.51*	.65(.23)	1.91**
Other drugs[a]	.59(.10)	1.80***	.76(.12)	2.13***	.81(.12)	2.24***	.85(.12)	2.35***
Interactions[b]								
BAC ≥.080*Other drugs	--	--	-.57(.22)	0.57**	-.54(.22)	0.58*	-.53(.21)	0.59*
THC ≥ 5.00*Other drugs	--	--	--	--	-.80(.37)	0.45*	-.85(.37)	0.43*
Carboxy*Other drugs	--	--	--	--	--	--	-.72(.38)	0.49†
Model χ^2	1455.405***		1460.003***		1460.432***		1464.263***	
Nagelkerke R^2	.380		.381		.382		.383	

Note: Significant interaction terms in the models are presented. OR = odds ratios. BAC = blood alcohol concentration. THC = Delta-9-tetrahydrocannabinol. *a* other drugs include narcotics, depressants, stimulants, hallucinogens, phencyclindine, inhalants, and other unknown types of drugs. *b* Two interaction terms, BAC<.080*Carboxy and BAC<.080*Carboxy*Other drug were excluded due to multicollinearity and the zero cell issue. Sample sizes vary by models due to the use of a list-wise deletion method.
† p <.1, *p <.05, **p <.01, ***p <.001

Though this is a substantial increase, it falls far short of the magnitude of the effect of alcohol on speeding. Drivers who tested below the 0.08 limit were 143% more likely to have been speeding and drivers who tested positive equal to or over the 0.08 BAC limit were 251% more likely to have been speeding during a fatal crash than drivers who did not. On the other hand, drivers with elevated BACs and those who tested positive for other drugs were less likely to be speeding during a fatal crash than drivers who only had a positive BAC as noted by the negative coefficients for the interactions between BAC and other drugs. These data indicate that the addition of “other drugs” to alcohol tends to dampen the tendency to speed.

The interaction models indicate no statistically significant two- or three-way interaction between Delta-9-THC and alcohol. These results indicate that drivers who were under the influence of both cannabis and alcohol simultaneously were no more likely to speed in fatal accidents than other drivers. There is however a statistically significant interaction between other drugs and BAC equal to or over the 0.08 limit, Delta-9-THC equal to or over the per se limit, and Carboxy in interaction models 1, 2, and 3 (respectively). These interaction were negative. In terms of cannabis, this suggests that drivers who had used cannabis recently and blood tested positive for both Delta-9-THC and another other drug were less likely to speed, though it should be noted that the overall relationship between Delta-9 THC, Carboxy, alcohol, other drugs and the outcome variable speeding remain positive. These interactions only provide modest improvements to model fit as evidenced by the small increases in Nagelkerke R^2 values. Put simply, these results suggest that consumption of any of these substances increases the likelihood that a driver was speeding during a fatal crash, but that the consumption of multiple substances does not further increase this risk and, in fact, might diminish, but not eliminate the risk caused by one substance.

In order to explore the possible relationship between certain types of drivers and blood toxicology results on speeding, sub-group analysis was conducted using the same logit models on three sub-groups of drivers: 1) drivers who were given a blood test for drugs (n=2,201, which is lower than the 2432 due to listwise deletion); 2) drivers who were given a blood test for drugs and tested positive for alcohol (n=763); and 3) drivers who were given a blood test for drugs and tested positive for alcohol greater than or equal to 0.08 (n=634). The results are presented in table 4.

TABLE 4 Sub-Group Analysis: Logit Models of Drug and Alcohol on Speeding

Covariates		Blood Tested Drivers (n=2,201) Logit (*Robust SE*)	OR	BAC Positive Drivers (n=763) Logit (*Robust SE*)	OR	BAC ≥.08 Drivers (n=634) Logit (*Robust SE*)	OR
Driver Characteristics							
Age		-.04(.00)	0.96***	-.05(.01)	0.96***	-.05(.01)	0.96***
Gender (1 = male)		.48(.14)	1.61**	.66(.22)	1.93**	.82(.24)	2.26**
Prior Speeding convictions (in the past three years)		.00(.12)	1.00	-.07(.18)	0.94	-.11(.20)	0.90
Natural Conditions							
Weather condition	*Clear (reference)*	--	--	--	--	--	--
	Cloudy	.01(.18)	1.01	-.11(.26)	0.89	.12(.29)	1.12
	Rain	-.22(.25)	0.80	-.41(.39)	0.66	-.15(.44)	0.86
	Fog/Smoke	.41(.39)	1.50	.37(.64)	1.45	.64(.68)	1.89
	Snow	.64(.45)	1.90	-1.37(.83)	0.25†	-1.24(.79)	0.29
Time of crash (1 = night)		-.04(.12)	0.97	.09(.19)	1.09	-.08(.22)	0.92
Road Conditions							
Road alignment (1 = straight)		-.85(.12)	0.43***	-.77(.18)	0.46***	-.78(.20)	0.46***
Road grade (1 = level)		-.37(.12)	0.69**	-.29(.18)	0.75	-.25(.20)	0.78
Surface condition (1 = dry)		-.63(.18)	0.54**	-.32(.28)	0.73	-.26(.32)	0.78
Surface type (1 = Asphalt)		-.02(.17)	0.98	.05(.26)	1.05	.03(.28)	1.03
Intersection involved		-.24(.14)	0.79†	-.24(.22)	0.79	-.21(.24)	0.81
Posted speed limit		-.03(.01)	0.97***	-.05(.01)	0.95***	-.04(.01)	0.96***
Drivers' Vehicle Conditions							
Vehicle type	*Passenger vehicle (Reference)*	--	--	-70(.25)	0.50**	-.29(.27)	0.75
	Heavy truck	-.17(.32)	0.85				
	Motorcycle	1.01(.15)	2.74***				
	Others	.03(.55)	1.03				
Other External Conditions							
Number of occupants in vehicle		.10(.06)	1.11†	-.03(.10)	0.97	-.04(.11)	0.96

Number of vehicles in crash	-.26(.09)	0.78**	-.35(.12)	0.71**	-.35(.13)	0.71**
Drug and Alcohol Involvement						
BAC < .080	.88(.20)	2.42***	--	--	--	--
BAC ≥ .080	1.18(.13)	3.26***	--	--	--	--
THC < 5.00	-.05(.21)	0.95	-.25(.26)	0.78	-.22(.29)	0.80
THC ≥ 5.00	.36(.17)	1.43*	.35(.25)	1.42	.39(.28)	1.48
Carboxy only without THC	.34(.19)	1.40†	.48(.30)	1.62	.44(.32)	1.55
Other drugs[a]	.45(.12)	1.57***	.29(.19)	1.34	.32(.21)	1.38
Model χ^2	639.536***		189.494***		145.119***	
Nagelkerke R^2	.376		.299		.281	

Note: No significant interaction terms in the models are found. OR = odds ratios. BAC = blood alcohol concentration. THC = delta-9-tetrahydrocannabinol. *a* other drugs include narcotics, depressants, stimulants, hallucinogens, phencyclindine, inhalants, and other unknown types of drugs. Sample sizes vary by models due to the use of a list-wise deletion method.

† $p < .1$, *$p < .05$, **$p < .01$, ***$p < .001$

The results for the sub-group analysis are somewhat consistent with the results for the speeding model presented above. The results from a model of blood tested drivers indicate that young males, driving a motorcycle, on curvy wet roads, who test positive for alcohol, Carboxy alone, and Delta-9-THC over 5 nanograms per mL, and other drugs, were more likely to be speeding when involved in a fatal crash, though some of these results are only marginally significant.

For the subset of drivers who tested positive for alcohol, the results similarly indicate that age, sex, and road conditions predict speeding during fatal crashes. Importantly, the variables Delta-9-THC, Carboxy, and other drugs are unrelated to speeding for alcohol positive drivers (both lesser and greater than or equal to 0.08 drivers). No significant interaction terms in the three sub-groups are found.

Driver Error Models

Results for the main effects and three interaction models where driver errors are regressed on driver characteristics, contextual factors, and drug and alcohol involvement are presented in Table 5. The results for the main effects model for driver errors are similar to the results for the speeding model presented above. Particularly, a number of driver characteristics (including expected risk factors, like age, unlicensed driver, and other prior traffic conviction) and contextual factors significantly predict driver error during a fatal crash. Similar to the findings from the speeding models, drivers with elevated BAC levels and drivers who tested positive for other drugs were significantly more likely to yield driver error during a fatal crash. However, while the presence of Carboxy was significantly and positively associated with driver error, the presence of Delta-9-THC is not significantly related to driver error. Indeed, drivers with Delta-9-THC in their blood that tested greater than or equal to 5.00 nanograms per mL were somewhat less likely to engage in driving error during a fatal crash, though this result only achieves marginal statistical significance. The interaction models reveal significant interactions between BAC ≥ .08 and Carboxy and other drugs, with all interactions suggesting that the combination of alcohol and other drugs seems to decrease the likelihood of driver error. This might indicate something of a self-correcting measure in that drivers under the influence of multiple substances might attempt to drive more carefully to compensate for the drugs effect. Alternatively, given that much of these data are derived from police reports, it is also possible that when an officer notes evidence of impairment that other smaller factors may be deemed less important and not recorded on the report.

TABLE 5 Logit Models of Drug and Alcohol on Driver Errors (n = 5,455; drivers from 2008-2016 WA FARS data)

Covariates	**Main Effects Model**		**Interaction Model 1**		**Interaction Model 2**		**Interaction Model 3**	
	Logit (*Robust SE*)	**OR**	**Logit (*Robust SE*)**	**OR**	**Logit (*Robust SE*)**	**OR**	**Logit (*Robust SE*)**	**OR**
Driver Characteristics								
Age	-.00(.00)	1.00†	-.00(.00)	1.00	-.00(.00)	1.00†	-.05(.11)	1.00†
Gender (1 = male)	.12(.07)	1.13†	.12(.07)	1.13†	.11(.07)	1.12	.12(.07)	1.12
Unlicensed driver	.54(.10)	1.71***	.54(.10)	1.71***	.53(.10)	1.71***	.53(.10)	1.71***
Other traffic conviction (in the past three years)	.16(.07)	1.17*	.16(.07)	1.17*	.16(.07)	1.17*	.16(.07)	1.17*
Natural Conditions								
Time of crash (1 = night)	-.34(.07)	0.71***	-.34(.07)	0.71***	-.35(.07)	0.70***	-.36(.07)	0.70***
Road Conditions								
Asphalt	.19(.09)	1.21*	.19(.09)	1.21*	.19(.09)	1.21*	.19(.09)	1.22*
Posted speed limit	.01(.00)	1.01***	.01(.00)	1.01***	.01(.00)	1.01***	.01(.00)	1.01***
Drivers' Vehicle Conditions								
Vehicle type: *Passenger vehicle (Reference)*	--	--	--	--	--	--	--	--
Vehicle type: *Heavy truck*	-1.41(.19)	0.24***	-1.41(.19)	0.24***	-1.40(.19)	0.25***	1.40(.19)	0.25***
Vehicle type: *Motorcycle*	-.71(.11)	0.49***	-.72(.11)	0.49***	-.72(.11)	0.49***	-.73(.11)	0.48***
Vehicle type: *Others*	-.84(.29)	0.43**	-.84(.29)	0.43**	-.83(.29)	0.44*	-.84(.29)	0.43**
Other External Conditions								
Number of vehicles in crash	-.20(.04)	0.82***	-.20(.04)	0.82***	-.20(.04)	0.82***	-.20(.04)	0.82***
Heavy truck involved	.56(.12)	1.76***	.57(.13)	1.76***	.56(.13)	1.76***	.57(.13)	1.76***
Head-on involved	.64(.08)	1.89***	.64(.08)	1.89***	.63(.08)	1.88***	.63(.08)	1.87***
Traffic control device present	1.10(.09)	3.02***	1.11(.09)	3.02***	1.11(.09)	3.02***	1.10(.09)	3.01***
Lap and shoulder belt used	-.33(.08)	0.72***	-.32(.08)	0.72***	-.31(.08)	0.74***	-.31(.08)	0.73***
Drug and Alcohol Involvement								

BAC < .080	.77(.16)	2.16***	.77(.16)	2.15***	.76(.16)	2.13***	.76(.16)	2.13***
BAC $\geq$ .080	.76(.09)	2.14***	.80(.09)	2.22***	.94(.10)	2.57***	.93(.10)	2.52***
THC < 5.00	.17(.18)	1.18	.16(.18)	1.17	.14(.18)	1.15	.14(.18)	1.15
THC $\geq$ 5.00	-.28(.16)	0.75†	-.29(.16)	0.75†	-.31(.16)	0.73*	-.31(.16)	0.73*
Carboxy only without THC	.66(.17)	1.93***	.92(.22)	2.51***	.88(.22)	2.42***	.88(.22)	2.42***
Other drugs[a]	.40(.08)	1.49***	.39(.08)	1.48***	.58(.09)	1.78***	.58(.09)	1.78***
Interactions								
BAC $\geq$.080*Carboxy	--	--	-.68(.34)	0.52†	-.61(.33)	.54†	-.31(.36)	.73
BAC $\geq$.080*Other drugs	--	--	--	--	-.76(.19)	.47***	-.66(.19)	.52**
BAC $\geq$.080*Other drugs*Carboxy	--	--	--	--	--	--	-1.27(.62)	.28*
Model χ^2	598.837***		602.716***		618.057***		622.614***	
Nagelkerke R^2	.143		.144		.148		.149	

Note: Significant interaction terms in the models are presented. OR = odds ratios. BAC = blood alcohol concentration. THC = delta-9-tetrahydrocannabinol. *a* other drugs include narcotics, depressants, stimulant, hallucinogens, phencyclindine, inhalants, and other unknown types of drugs. Sample sizes vary by models due to the use of a list-wise deletion method.

† p <.1, *p <.05, **p <.01, ***p <.001

Sub-group analyses were then conducted, including drivers who were given a blood test for drugs (n=2,251); 2) drivers who were given a blood test for drugs and tested positive for alcohol (n=783); and 3) drivers who were given a blood test for drugs and tested positive for alcohol greater than or equal to 0.08 (n=651). The results are presented in table 6. The results from a model of blood tested drivers indicate that unlicensed young males, who had other traffic conviction records, and those who test positive for alcohol or Carboxy were more likely to commit driver error when involved in a fatal crash, though again some of these results are only marginally significant. Interestingly, drivers who test positive for Delta-9 THC equal to or over 5.00 ng per mL are 38% less likely to make driver errors in fatal accidents than drivers who were blood tested and found to not have Delta-9-THC in their blood.

TABLE 6 Sub-Group Analysis: Logit Models of Drug and Alcohol on Driver Errors

Covariates	Blood Tested Drivers (n=2,251)		Interaction Model of Drug Tested Drivers (n=2,251)		BAC Positive Drivers (n=783)		BAC >.08 Drivers (n=651)	
	Logit (*Robust SE*)	**OR**	**Logit (*Robust SE*)**	**OR**	**Logit (*Robust SE*)**	**OR**	**Logit (*Robust SE*)**	**OR**
Driver Characteristics								
Age	-.01(.00)	0.99**	-.01(.00)	0.99**	-.02(.01)	0.98***	-.02(.01)	0.98**
Gender (1 = male)	.13(.11)	1.14	.13(.11)	1.14	-.20(.20)	0.82	-.42(.23)	0.66†
Unlicensed driver	.37(.13)	1.45**	.36(.13)	1.44**	.56(.18)	1.75**	.60(.20)	1.82**
Other traffic conviction	.17(.10)	1.18†	.17(.10)	1.19†	-.03(17)	0.97	.01(.18)	1.01
Natural Conditions								
Time of crash (1 = night)	-.34(.10)	0.71**	-.35(.10)	0.70**	-.31(.18)	0.74	-.29(.21)	0.75
Road Conditions								
Asphalt	.15(.14)	1.16	.15(.14)	1.17	.33(.22)	1.39	.24(.24)	1.27
Posted speed limit	.01(.00)	1.01†	.01(.00)	1.01†	.01(.01)	1.01	.02(.01)	1.02*
Drivers' Vehicle Conditions								
Vehicle type: *Passenger vehicle (Reference)*	--	--	--	--	.44(.23)	1.55†	.50(.26)	1.65†
Vehicle type: *Heavy truck*	-1.60(.30)	0.20***	-1.58(.30)	0.21***				
Vehicle type: *Motorcycle*	-.68(.15)	0.51***	-.67(.15)	0.51***				
Vehicle type: *Others*[a]	-.99(.48)	0.37*	-.99(.48)	0.37*				
Other External Conditions								
Number of vehicles in crash	.17(.07)	1.19*	.16(.07)	1.18*	.23(.14)	1.26	.40(.17)	1.49*
Heavy truck involved	.65(.21)	1.92**	.66(.21)	1.93**	.87(.51)	2.38†	.66(.54)	1.94
Head-on involved	.94(.13)	2.56***	.94(.13)	2.55***	1.50(.34)	4.49***	1.49(.40)	4.45***
Traffic control device present	1.12(.15)	.08***	1.12(.15)	3.08***	1.45(.33)	4.28***	1.53(.37)	4.63***
Lap and shoulder belt used	.01(.11)	1.01	.02(.11)	1.02	.25(.18)	1.28	.24(.20)	1.27
Drug and Alcohol Involvement								
BAC < .080	.81(.20)	2.24***	.81(.20)	2.26***	--	--	--	--
BAC ≥ .080	.71(.12)	2.03***	.84(.14)	2.32***	--	--	--	--
THC < 5.00	-.07(.18)	0.93	-.08(.18)	0.92	-.24(.27)	0.79	-.29(.31)	0.75
THC ≥ 5.00	-.48(.16)	0.62**	-.50(.16)	0.61**	-.79(.23)	0.46**	-.94(.26)	0.39***
Carboxy only without THC	.43(.19)	1.53*	.41(.19)	1.50*	.42(.27)	1.52	.17(.29)	1.18
Other drugs	.13(.10)	1.14	.25(.12)	1.28*	-.06(.18)	0.94	-.22(.20)	0.80
Interactions								
BAC ≥.080*Other drugs	--	--	-.42(.21)	0.66†	--	--	--	--

Model χ^2	306.301***	309.405***	132.768***	119.787***
Nagelkerke R^2	.172	.174	.220	.243

Note: Significant interaction terms in the models are presented. OR = odds ratios. BAC = blood alcohol concentration. THC = delta-9-tetrahydrocannabinol. *a* other drugs include narcotics, depressants, stimulants, hallucinogens, phencyclindine, inhalants, and other unknown types of drugs. Sample sizes vary by models due to the use of a list-wise deletion method.
† p <.1, *p <.05, **p <.01, ***p <.001

The results also indicate that drivers who were given a blood test were more likely to have been involved in head-on collision and with other vehicles, like heavy trucks, in a fatal crash. Notably, it might be that those involved in head-on collisions were more likely to be drug tested because they died. There is a marginally significant interaction between other drugs and BAC equal to or over the 0.08 limit. However, this interaction was negative, meaning that drivers who had consumed alcohol and tested positive for some other drug were less likely to commit driver error. The interaction, however, only provides modest improvements to model fit as evidenced by the small increases in Nagelkerke R^2 values.

With regard to a model of BAC positive drivers, the results show that age, driving without a license, and using a passenger vehicle were positive risk factors driver error when involved in a fatal crash. The significance of prior traffic convictions and time of accident disappears in this sub-group analysis. In addition, the presence of other drugs and Carboxy is no longer associated with driver error. Delta-9-THC, Carboxy, and other drugs have some mixed effects on driver error on these sub-group drivers and no significant interaction terms in the BAC positive sub-groups are found.

DISCUSSION

This study examined the degree to which those drivers tested positive for cannabis, measured both as Delta-9-THC and Carboxy in the blood, were related to speeding and driver error that may result in fatal crashes in Washington State. We hypothesized that cannabis use increases the likelihood that a driver involved in a fatal crash was speeding, and committed an error. Moreover, we hypothesized that the effects of Delta-9-THC and Carboxy on these outcomes are greater when used in conjunction with alcohol and/or other drugs.

The results of this study were mixed. Delta-9-THC (over 5ng/mL) was positively associated with speeding, but Delta-9-THC (over 5ng/mL) was negatively related to driver error. The positive link between Delta-9-THC and speeding was particularly strong, as these results were statistically significant in the full model and for the subset of all drivers who were blood tested. It is important to note though that while this was a robust result, the relationship between Delta-9-THC and speeding was than the relationship between alcohol and speeding, Carboxy and speeding, and other drug consumption and speeding. This suggests that while marijuana consumption is a risk factor for speeding, it is, like the research on marijuana and crashes suggest (*4*), a low to medium-level risk factor. Much like prior research, however, alcohol intoxication was a strong and positive predictor of speeding and driver error (8, *17*, *33*, *34*). Again, while our results cannot speak to the etiology of crashes or fatal crashes, these results suggest that alcohol intoxication is more likely to result in driver errors in fatal accidents than cannabis consumption.

The negative link between Delta-9-THC and driver errors was even weaker, as this was only significant in the full sample during interaction models and in the subset models including only drivers who were blood tested. Put simply, drivers with Delta-9-THC seemed marginally less likely to make driver errors in fatal crashes, but this effect was not particularly robust. At a surface level, the null or very weak negative relationship between Delta-9-THC and driver error might seem to run contrary to prior research which finds that cannabis intoxication is a risk factor for crashes. We caution against this interpretation, as our data consist of only crashes resulting in fatal injuries and therefore due to this selection bias cannot be used to identify predictors of non-fatal crashes generally. Instead, our results simply state that drivers in Washington who tested positive for Delta-9-THC were no more likely to commit an error included in the WA FARS data than drivers involved in fatal crashes in general. In fact, when examining the subset of blood tested drivers, individuals who tested positive for more than 5ng of Delta-9-THC were less likely than drivers who tested negative for Delta-9-THC to make driver errors. This result may in fact be in line with prior research, as it is possible that cannabis impaired drivers recognize their impairments (*7*, *17*) and take active steps to drive slower and make less driving errors (*35*). There are other potential explanations for this result, however. It may be that police are less likely to note driver error's during the crash reports when they suspect recent marijuana use, as it is possible that the evidence of drug consumption is enough to press forward with charges.

Interestingly, the interaction of Delta-9-THC and alcohol and the interaction between Delta-9-THC and other drugs were not risk factors for speeding or driver error; in fact, these results have demonstrated only a modest, but negative, association with speeding. Sub-group analyses have also shown the null or very weak relationship between Delta-9-THC (above and below 5ng/mL) and speeding. Our interactions and subgroup analyses suggest that the combination of recent cannabis consumption with other drugs or alcohol do not greatly impact the likelihood that a driver was speeding or made an error during a fatal crash. This seems to run contrary to prior work highlighting the interactive nature of marijuana and alcohol (*8*), though it is important to remember that our work only describes behavior during crashes and does not explain why the crash occurred in the first place.

These data do indicate that the per se limit for Delta-9-THC stood as a potential demarcation line on driver culpability, albeit with mixed results. As mentioned in the preceding discussion, in terms of speeding, Delta-9-THC over the 5 nanograms per mL limit was strongly and statistically significantly related to speeding in both the main and interaction models (see Table 3) as it was for Blood Tested Drivers in the sub-group analysis (see Table 4). For drivers who committed errors, the relationship with a Delta-9-THC level that met or exceeded the per se limit was more modestly, and paradoxically negatively, related in both the main and interaction models and in the sub-group analyses (see Tables 5 and 6). In contrast, Delta-9 THC levels that tested below the per se limit did not show up as statistically significantly related to either speeding or driver error in any of the models (with the exception of a modest and negative relationship with BAC Positive Drivers and Speeding – see Table 4). Clearly, replication of this kind of analysis is warranted before any conclusions can be drawn, but these findings do indicate there is a difference in outcomes when the level of Delta-9-THC intoxication varies. As already mentioned in this paper, the appropriate per se limit in the states is not a settled matter and this matter requires more attention as determining the point at which Delta-9 THC levels are most likely to result in driving impairment might be useful for policymakers as they grapple with whether a limit makes a difference and whether it is defensible in court.

One of our most interesting results regards the relationship between Carboxy and driver behaviors during fatal crashes. These results indicate that drivers who had consumed cannabis recently, but not necessarily in the immediate time period before the crash, were significantly more likely to speed during a fatal accident and this effect was greater than for Delta-9-THC. Moreover, our results indicate that drivers who had consumed cannabis, but not recently, were also more likely to make driver errors during fatal crashes. This suggests that the link between cannabis use more generally and recent cannabis use and driving behaviors need not be the same.

We cannot definitely explain why Carboxy has stronger and more deleterious effects on driving behaviors than Delta-9-THC, but we offer some suggestions for investigation in future research. One possibility is that Carboxy, given the length of time it remains in the bloodstream, is a proxy measure for regular cannabis use. If so, it is possible that cannabis use impairs cognitive functioning and thereby leads to poor driving decisions. Indeed, research suggests that persistent cannabis use may have both acute and long-term effects on decision-making (*36, 37*). The negative correlation between Delta-9-THC concentration and driving performance has been shown to be inconsistent and to vary for chronic versus occasional users (*3*), indicating that over time, regular cannabis users may become worse drivers. Alternatively, a positive result for Carboxy might simply be a proxy for impulsivity or low self-control. A large body of research links impulsivity to both drug consumption (*38, 39*) and risky behaviors, including risky driving behaviors (*40, 41*). This explanation moves the causal emphasis away from cannabis and suggests, instead, that both cannabis use and driving problems are the result of the same underlying personality traits and characteristics. It is important to note that FARS data are not suited for testing these explanations, as Carboxy is a crude proxy for variables like regular cannabis use and an even weaker proxy for cognitive functioning. More work is absolutely needed in this area.

The current research suffers from a number of limitations. The WA FARS data only examine fatal crashes and therefore provide a sample of incidents in which driving, for whatever reason, has gone awry resulting in the death of a driver, passenger, or non-motorist. More research is needed on the effects of cannabis in a variety of driving contexts, including non-fatal crashes and traffic citations (not involving a crash). Moreover, the current analysis uses data only from Washington State. Though the WA FARS data provide more detailed drug information than the NHTSA FARS data, these results should be replicated in other states. Related to this, while the WA

FARS data is notable in their inclusion of drug-testing results, drug tests were not administered in all crashes. Though we suspect that crash investigators were likely to order testing if there was evidence of recent cannabis use, we cannot rule out measurement error in our key independent variables.

CONCLUSIONS

As more and more states experiment with the legalization and decriminalization of cannabis, it is important that research is conducted to examine the consequences for traffic safety. Our results are mixed. The consumption of cannabis appears to increase the likelihood that a driver was speeding during a fatal crashes, but only Carboxy, an inactive resultant chemical, is a risk factor for driver errors during fatal crashes. Thus, while we find some evidence that cannabis consumption is a risk factor for dangerous driving behaviors, we do not find uniform evidence of this. Moreover, the negative effect of cannabis as never as strong as the negative effect of alcohol in any of our models. Lastly, we find no evidence that cannabis interacts with alcohol or other substances to increase the likelihood of dangerous driving behaviors during fatal crashes. Even given these mixed results, it is clear that more work is needed in this area, especially on data which are not limited to only fatal crashes. In addition, we strongly suggest that additional work be conducted examining the link between Carboxy and driving behaviors, as the FARS data are not suited for addressing why the non-psychoactive metabolites produced by consuming cannabis are a bigger predictor of driver error than the primary psychoactive ingredient in cannabis. Given our analysis and the limitations of our data, we conclude by noting that there is evidence that cannabis use is associated with risky driving behaviors during fatal crashes, but that the effect is low to medium in size and that alcohol remains a much larger problem.

ACKNOWLEDGEMENTS

This research was supported in part by the Washington Traffic Safety Commission (Grant-1484: The Role of THC and Other Intoxicants on Traffic Accidents in Washington State). The contents of the manuscript are solely the responsibility of the authors and do not necessarily reflect the official views of the funding agency.

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