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Cannabis and crash risk among older drivers

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ABSTRACT

The purpose of this research was to reanalyze data collected from the National Highway Traffic Safety Administration's Drug and Alcohol Crash Risk Study to investigate whether driving under the influence of cannabis (THC-positive) was associated with elevated crash risk for younger and older drivers. The data came from a case-control relative risk study collected from Virginia Beach, VA, over a 20-month period. Data collectors gathered driver information from the scene of vehicle crashes and, in some cases, from hospitals. Non-crash controls were sampled from the same locations, days, and times as crashes. Key data items included driver demographics and oral fluid and blood samples, which were assayed for licit and illicit drugs. We found no overall association between cannabis use and risk of crash involvement. However, when age and age^2 were allowed to interact with THC, significant interaction effects emerged. THC was associated with increased risk of crash involvement for older drivers. Difference between THC-positive and sober drivers emerged as significant at age 64. The research underscores the value of examining drugged driving in the context of driver age. Age-related declines in neurocognitive and psychomotor functioning were not measured but might be important in explaining the results.

1. Introduction

Despite recent increased attention by the scientific community, uncertainty remains as to whether and to what extent cannabis use contributes to risk of vehicle crash involvement. Double-blind placebo control studies have routinely found that cannabis impairs cognitive and psychomotor skills, as well as simulated driving (Hartman et al., 2015; Hartman and Huestis, 2013). But recent epidemiological research, which examines actual crashes at the population level, has proved quite inconsistent.

In the past decade, four major relative crash risk studies have been conducted. Brubacher et al. (2019) examined the responsibility of crash-involved drivers in Canada as a function of tetrahydrocannabinol (THC) and found no significant differences between that THC-positive and sober drivers in terms of crash responsibility. On the other hand, Drummer et al. (2020) found a statistically significant but modest increase in crash culpability among THC-positive drivers in Australia. Bernhoft et al. (2012) measured the drug presence among seriously injured drivers in Europe and found a statistically significant but small increase in the risk of crash involvement among THC-positive drivers. But a major case-control crash risk study conducted by the National Highway Traffic Safety Administration (NHTSA) in the United States found an adjusted odds ratio of 1.0 for predicting crash involvement from drivers being THC-positive (Compton and Berning, 2015; Lacey et al., 2016). In one recent meta-analysis, one in three studies reviewed found no association between cannabis and increased crash risk (Gjerde et al., 2015).

There is no clear explanation for the divergent results of these major data collection initiatives. It is worth noting that results from epidemiologic research on *alcohol*-involved crashes has been remarkedly consistent. For example, the risk curves from Borkenstein's seminal study in Grand Rapids, Michigan (Borkenstein et al., 1964) closely mirrored curves produced 30 years later in Germany (Krüger and Vollrath, 2004; Krüger et al., 1995). More recently, the curves from the Dunlap and Associates crash risk study in Florida (Blomberg et al., 2005) closely matched those from Lacey et al. (2016) in Virginia Beach, despite marked differences in refusal rates. The lack of consistency in the epidemiologic literature regarding cannabis and crash risk does not appear to simply be a product of methodologies used.

Diverging results may indicate that the underlying population-level effect is quite modest, but it also might suggest the presence of unmeasured or unmodeled variables. This research explores the latter. We

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hypothesize that *driver age* shapes the relationship between cannabis use and crash risk and in this article examine that relationship across the lifespan.

The relationship between driver age and crashes, independent of alcohol or drug use, is well-known and described by a U-shaped curve. Younger drivers have substantially higher risk of being involved in a crash than middle-aged drivers (Cassarino and Murphy, 2018; Freeman et al., 2012; Klauer et al., 2014; Preusser, 2003; Williams, 2003), as do older drivers (Cicchino and McCartt, 2014; Fraade-Blanar et al., 2018; Loughran and Seabury, 2007; Regev et al., 2018). Cannabis may exacerbate this risk.

Our prediction, in part, follows from research on alcohol-impaired driving. Alcohol substantially increases the risk of crash involvement for drivers under 20 (Voas et al., 2012; Zador et al., 2000a,2000b), possibly by contributing to impulsive and risky driving decisions (Fillmore et al., 2008). And while epidemiological studies on drunk driving have not focused on the upper ranges of the age distribution, experimental research suggests that older adults are particularly sensitive to alcohol affecting their driving skills (Price et al., 2018; Sklar et al., 2014, 2012).

As with alcohol, cannabis may have particularly notable effects on younger and older drivers as well. Cannabis consumption is known to impair impulse control (Ramaekers et al., 2016; Wrege et al., 2014) as well as neurocognitive and psychomotor functioning (Bossong et al., 2014; Crane et al., 2013; Crean et al., 2011; Curran et al., 2002; Gonzalez, 2007; Ramaekers et al., 2006; Wrege et al., 2014). For younger drivers, cannabis use may contribute to recklessness through impulse control, or skill impairment may interact with general inexperience to increase risk. For older adults experiencing age-related decline (Murman, 2015; Salthouse, 2009), cannabis use may produce compounding effects on neurocognitive and psychomotor functioning. Age-related cognitive decline has been linked with *linear* increases in crash risk (Fraade-Blanar et al., 2018), and it follows that cannabis use by individuals with preexisting cognitive risk factors would likely result in elevated crash risk.

Publicly available data from the National Survey on Drug Use and Health (https://www.datafiles.samhsa.gov/) show that over 6.6 million young adults (30.5 % of those aged 16–20) used cannabis in 2018, with over 2 million using habitually (3 times per week or more). But cannabis use is not limited to younger adults. Over 2 million adults 65 and older use cannabis, with 475,000 habitual users. Moreover, there are 6.8 million cannabis users, and 1.9 million habitual users, in their 50 s and early 60 s. Over the next decade, as these younger cohorts age, the country may see a dramatic increase in drugged driving older adults. If and to what extent cannabis use exacerbates the effects of age-related decline on crash risk, the country could witness a serious public health threat on the roadways.

2. Methodology

Data to test this hypothesis came from the Drug and Alcohol Crash Risk project, which was conducted by NHTSA in Virginia Beach, VA, over a 20-month period. Additional support for the data collection came from a grant from the National Institute on Alcohol Abuse and Alcoholism (R01 AA018352, PI: Robert Voas, PhD). The project's final report is available on the NHTSA website (https://www.nhtsa.gov/behavioral-research/drug-and-alcohol-crash-risk-study), in which the data collection procedures are described in detail. The raw data are publicly available on the NHTSA website (https://www.nhtsa.gov/drug-and-alcohol-crash-risk-study/drug-and-alcohol-crash-risk-study-databases). Data processing and analysis code for the research described herein are available from the authors upon request.

2.1. Data collection procedures

Data collection involved four primary components: crash data, control data, hospital data, and biological assays. All research design and data collection procedures were reviewed and approved by an Institutional Review Board.

2.1.1. Crash data

Data collection occurred 24 h a day, seven days a week, and at least one data collection team was on duty at all times. Teams (including a phlebotomist) traveled with a law enforcement officer who was hired to assist with the research and waited to be alerted to calls about vehicle crashes over police radio. When the research officer was alerted to a vehicle crash, the team would travel to the crash site to collect data. Although other on-duty officers responded to the crash in an official capacity, the research officer was present to add authority to the data collection efforts and help ensure the safety of the site. He or she served as a liaison between drivers and the study team, but otherwise had no interaction with research participants.

Assuming drivers were medically fit to be interviewed and consented, the data collector recorded observations, asked survey questions, and requested breath, oral fluid, and blood samples. Drivers were interviewed separately in multivehicle collisions. Drivers responded to interview questions on demographics, driving practices, drinking and drug use history, problem drinking and drug use, and other risk factors (sleepiness, cell phone use, seat belt use). However, only driver demographics (age, sex, and race/ethnicity) were included in this research. A $10 incentive was offered to participants who provided an oral fluid sample; an incentive of $50 was offered for a blood sample.

2.1.2. Control data

When a crash-involved driver participated, the team returned to the crash site one week later—on the same day of week and time of day—for non-crash "control" data collection. Using standard roadside survey recruitment methods (Kelley-Baker et al., 2016), vehicles were randomly selected from the traffic flow and directed into an off-road survey bay. Drivers were recruited, and those who consented were administered the same survey (including breath, oral fluid, and blood draws) as were crash-involved drivers. For each crash, control collection continued until two drivers were recruited and consented, or two hours elapsed.

2.1.3. Hospital data

The study team collaborated with the Virginia Medical Examiner's Office and the Sentara Hospital Organization for offsite data collection. If medical staff were treating the driver, a time was arranged for data collection, which mirrored the previous procedure. For seriously injured drivers, medical personnel drew and stored an additional 10 mL of blood for the research, but which was only provided after consent was obtained. For drivers who died in the crash, the medical examiner drew a vial of blood for the study.

2.1.4. Biological assays

Oral fluid and blood samples were assayed for quantitative drug concentrations. Biological samples were screened and confirmed for the drugs (Lacey et al., 2016, Table 7) using enzyme-linked immunosorbent assay (ELISA) micro-plate technology. The lab provided all confirmations via gas chromatography-mass spectrometry (GC/MS) or liquid chromatography-mass spectrometry (LC/MS) (Moore et al., 2007).

2.2. Missing data

Not all drivers who were stopped as part of the research and who were eligible to take part agreed to participate. Lacey et al. (2016) reports 82.2 % of crash-involved drivers and 93.8 % of control drivers provided an oral fluid and/or blood sample. Note, however, that the *alcohol* risk curves presented in Lacey et al. (2016, Figure 7) closely matched alcohol risk curves obtained by Blomberg et al. (2005), which use the identical case-control methodology, in which participation rates

were quite high. This suggests that differences in participation rates between crash and control cases did not contribute to biased results.

2.3. Analytic strategy

All analyses were conducted using *generalized linear mixed modeling* in SAS 9.4. This procedure accommodates random effects / nested dated and can be applied to data that follow a variety of distributions (e.g., Gaussian, binary, Poisson) by specifying the appropriate link function (e.g., a logit-link function for binary outcomes). For each analysis, each crash was grouped with its matched controls to form a cluster, which was treated as an R-side (residual) random effect in the model (SAS Institute Inc., 2010). The analysis also applied the empirical *sandwich estimator* to produce robust and unbiased error terms.

In all analyses, crash outcome (non-crash versus crash) was the binary outcome variable. Driver age was the main fixed-effect predictor and was treated as a continuous variable. In the first analysis we dichotomized THC status (sober versus THC-positive), based on assays of biological samples. Age was treated a continuous variable; we did not bin age into categories to reserve statistical power (Altman and Royston, 2006; MacCallum et al., 2002). Rather, in order to capture the known curvilinear relationship between age and crashes, age-squared (age^2) was included in the model. Driver THC, age, age^2, and the THC x Age and THC x Age^2 interactions were the primary focus of the research.

Driver sex (male or female), race (White, Black, or other), and ethnicity (non-Hispanic or Hispanic) were included as demographic covariates. As detailed below, we also controlled for alcohol, but excluded polydrug use in our analysis. Blood alcohol concentrations (BACs) was treated as a continuous variable for the same reasons we treated age as continuous (discussed above), and we modeled BAC^2 as well to capture known curved relationships between alcohol and crash risk (Blomberg et al., 2005; Zador et al., 2000a,2000b). Finally, because people who drive more miles crash more, we also included two rough driving exposures: a three-level annual miles variable (more than average, average [15,000 miles], or less than average), and a four-level miles-from-home item (0−5, 6−10, 11−20, 21+).

Interpretation of results was aided by the presentation of odds ratios as well as model-based proportions (computed using the ESTIMATE command in SAS and then applying the SAS *ilink* function (Wicklin, 2019). Interactions between categorical (e.g., THC category) and continuous (e.g., age) variables were probed using planned comparisons in SAS. Curves were plotted using the PROC PLM function in SAS.

2.4. Models tested

We first tested a model where THC status was obtained from assays of oral fluid and dichotomized. We focused on THC × Age and THC × Age^2 interactions as the primary predictors, with crash-control cluster modeled as a random effect and the covariates described above. Next we conducted *exploratory* analyses involving interactions among age, THC, and BAC (including curvilinear functions). We had no explicit hypotheses regarding interactions with alcohol. Finally, we conducted sensitivity analyses to demonstrate that THC × Age interactions were not an artifact due to age and drug outliers. All models tested crashes as the outcome.

3. Results

3.1. Data reduction

The official final data set provided by NHTSA went through several rounds of reduction before it was analyzed for the research herein. The primary purpose for this reduction was to eliminate cases that included drug use other than cannabis in order to remove the potential for confounding. The official data set (N = 12,790) included 5375 crashes and 7415 controls. Of these, 2700 refused to provide an oral fluid sample; these cases were excluded given they could not provide useful information for our hypothesis. Of the remaining 10,090 cases, 111 refused to provide a breath sample. However, BACs were estimated from blood (n = 25) and oral fluid (n = 86) for those missing cases, so all 10,090 drivers (n = 3171 crashes and n = 6919 controls) had measures of alcohol consumption.

Next, we identified and indicated cases of cannabis use and polydrug use. Of the 10,090 oral fluid samples collected from drivers, 8589 tested negative for all potentially impairing drugs (Lacey et al., 2016, Table 7). Alcohol was considered separately. A total of 555 cases tested positive for THC only, with no polydrug use, while 114 cases showed polydrug use that included THC. (Note that we considered only active THC, but no metabolites, in this selection). We also identified drivers that tested positive for other drugs (e.g., sedatives, stimulants, antidepressants or opioids). Given the focus of this paper on cannabis and driving and the desire to avoid confounds with other drugs, we did not include drivers with other drugs in their systems in the analysis. Our final design, therefore, involved a comparison of crash risk for drug-free drivers versus THC-only drivers across the lifespan, controlling for alcohol consumption and other covariates.

3.2. Sample

This process of data reduction produced a dataset of 2859 crash-drivers and 6285 non-crash control drivers (N = 9114) for which there was both drug and alcohol data. While everyone in the sample provided an oral fluid specimen, only about half (53.2 %) of the drivers in the sample provided blood samples (n = 1197 crashes and n = 3649 controls).

There were no missing data regarding THC, BAC, race, ethnicity, or annual miles driven, but 40 cases had missing driver sex information and 27 cases had missing driver age. We also excluded one case where the driver age was listed as 13. After removing these cases, the final data set (n = 2839 crashes and 6238 controls) was 52.6 % male, 70.9 % White, and 21.7 % Black, and 6.4 % identified as having a Hispanic ethnicity. Drivers' ages ranged from 16 to 92 with a median of 36 and quartiles of 25 and 49.

A total of 551 drivers (6.1 %) tested positive for THC (in oral fluid) but no other drugs. 288 (6.1 %) drivers tested positive for cannabis (but no other drug) in blood. Table 1 shows the distribution of THC positive cases (based on oral fluid) by age category for crashes and controls.

3.3. Statistical tests

3.3.1. Age × THC model

A preliminary model included THC category, age, age^2, BAC, BAC^2, sex, race, ethnicity, annual miles driven and distance from home. This analysis confirmed the original study finding of no overall effect of THC (Compton and Berning, 2015; Lacey et al., 2016) and is not discussed further. The primary analysis for this research, however, included THC × Age and THC × Age^2 interactions as the key predictors. Results are shown below in Table 2 (Stroup, 2013, 2015). It should be noted that

Table 1
Counts of by age, THC status (based on oral fluid), and crash status.

	Controls		Crashes	
Age	THC-	THC+	THC-	THC+
16−19	309	32	347	56
20−29	1595	176	817	81
30−39	1190	85	468	32
40−49	1202	36	381	11
50−59	947	24	315	8
60−69	448	4	197	4
70−79	150	1	90	0
80+	39	0	31	1
	5880	358	2646	193

Table 2
Results from THC × Age model.

Effect	Category	Odds Ratio (95 % CI)	Statistical Test
Sex	Male	–	F (1, 1713) = 5.2, p = .023
	Female	1.115 (1.015–1.225)	
Race	White	–	F (2, 1724) = 6.8, p = .001
	Black	0.840 (0.748–0.944)	
	Other	1.173 (0.975–1.411)	
Ethnicity	Non-Hispanic	–	F (1, 472) = 0.6, p = .444
	Hispanic	1.082 (0.884–1.323)	
THC Category	THC negative	–	F (1, 477) = 4.0, p = .046
	THC positive	1.071 (0.807–1.423)	
Annual Miles	Above Avg	–	F (2, 2837) = 49.3, p < .001
	Average	1.409 (1.254–1.582)	
	Below Avg	1.824 (1.620–2.053)	
Miles Home	0–5 miles	–	F (3, 2686) = 27.2, p < .001
	6–10 miles	1.321 (1.176–1.483)	
	11–20 miles	1.477 (1.282–1.702)	
	21+ miles	1.863 (1.589–2.183)	
†BAC	1-unit (.1)	1.041 (0.941–1.153)	F (1, 6757) = 0.6, p = .430
BAC^2	1-unit (.01)	1.008 (0.999–1.102)	F (1, 6757) = 3.5, p = .062
Age	1-unit (Year)	0.900 (0.887–0.914)	F (1, 6757) = 43.0, p < .001
Age^2	1-unit² (Year)	1.001 (1.001–1.001)	F (1, 6757) = 32.0, p < .001
Age × THC	THC -	–	F (1, 6757) = 4.3, p = .038
Age × THC	THC +	0.908 (0.829–.995)	
Age^2 × THC	THC -	–	F (1, 6757) = 4.6, p = .028
Age^2 × THC	THC +	1.001 (1.000–1.003)	

† Prior to analysis, 3-decimel BACs (.001) were multiplied by 100 to aid with interpretation, so a change in BAC of .001 equals a change of .1 for computing estimated values.

the procedure used by SAS for binary data is focused both on testing as well as accuracy of estimation and relies on F-statistics (Stroup, 2013, 2015).

Most covariates examined were significantly related to crash involvement. Controlling for all other variables in the model, we found that women were slightly but statistically significantly more likely to crash than men, Black drivers were significantly less likely to crash than White drivers, and greater driving exposure—measured both by annual miles driven and miles from home—predicted greater likelihood of crashing.

Central to this research, however, were the significant THC × Age and THC × Age^2 interactions. These indicate the relationship between age and crash-risk was different for those who tested positive versus negative for THC. The risk curves produced by our model are shown in Fig. 1. Accordingly, consistent with the extant literature, both younger and older drivers demonstrated greater likelihood of crash involvement. But for drivers who were THC-positive, the crash rate was clearly elevated, particularly at the higher end of the age distribution. In interpreting Fig. 1, it is important to note that by nature of our data collection design, approximately 1/3 of cases (31.3 %) were crashes, and thus crash probability of .313 can be considered baseline.

Fig. 1 includes confidence intervals, but it is important to note that

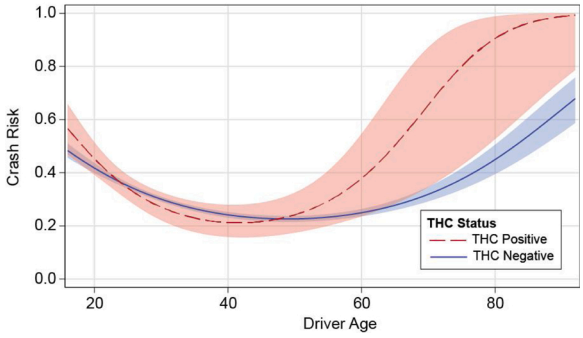

Fig. 1. Crash risk as a function of driver age and THC category.

eyeballing overlap between confidence intervals is not identical to significance tests (Cumming and Finch, 2005; Goldstein and Healy, 1995); significance tests are based on pooled errors around differences, not errors around independent estimates. Accordingly, we probed the curves for significant differences using planned contrasts between THC-positive and sober drivers at different specific age points (ages 16, 24, 40, 64, and 76) along the risk curves. These were strategically selected to cover the meaningful range of ages, and no contrasts at any other ages were performed. Importantly, these tests were not comparisons among age "groups." Rather, the estimates along the risk curves were *derived from the entire data set*. Tests near the ends of the distribution, where sample sizes were thinner, produced commensurately larger standard errors as a result (as shown in Fig. 1).

At age 16, the difference in expected crash rate between THC-positive and sober drivers was not statistically significant (p = .11); neither were differences at age 24 or 40 (p-values > .38). However, tests of THC-positive versus sober drivers at age 64 and 76 were statistically significant, (F (1, 6757) = 3.8, p = .049; F (1, 6757) = 4.8, p = .028. This provides indication of at which age point the increased risk associated with driving under the influence of cannabis become statistically significant.

Fig. 2 displays the *difference* in crash risks between THC-positive and THC-negative drivers as a function of age against a 0-reference line. Confidence intervals are based on the pooled standard errors of the individual series. Using the pooled standard errors, we begin to see differences emerge at earlier ages than shown in Fig. 1.

3.3.2. *Age × THC × BAC model*

We conducted exploratory analyses examining interactions between BAC and the other variables in the model. Prior research (e.g., Dubois et al., 2015) has demonstrated interactions between alcohol and cannabis on driving. Thus, beyond the covariates, our model included: age, age^2, THC, Age × THC, Age^2 × THC, BAC, BAC × THC, BAC × Age, BAC × Age^2, BAC × THC x Age, BAC × THC × Age^2, BAC^2, BAC^2 ×

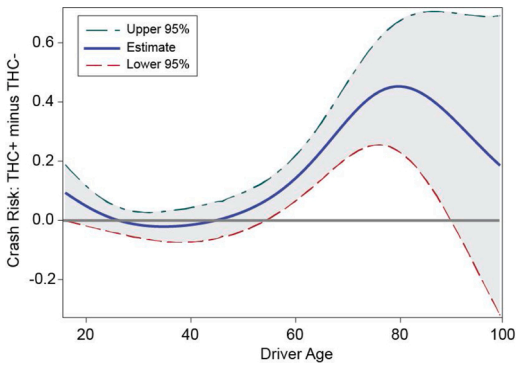

Fig. 2. Difference is risk between THC-positive and THC-negative drivers as a function of age.

THC, BAC^2 × Age, BAC^2 × Age^2, BAC^2 × THC × Age and BAC^2 × THC × Age^2. However, even after following model reduction procedures, none of these new higher order effects approached statistical significance.

3.3.3. Sensitivity analysis

In formal analysis, the penalty for small sample sizes is increased standard errors. As is shown in Fig. 1, the confidence intervals around the THC-positive curve, and particularly at the upper end of the age distribution, are quite wide. Nevertheless, areas of clear non-overlap are evident, and formal planned tests revealed statistically significant differences between the curves. It is understood, however, that in regression a small number of cases at the ends of a distribution can exert undue leverage on the shape of curves. Although our risk curves were informed by thousands of drivers each, it is possible, at least, that the few older THC-positive crash drivers in the sample created observed differences by exaggerating these curves.

Although we cannot collect new data, we can examine the influence of older drivers on the risk curves by assigning different weights to cases according to age. Across a series of analyses (otherwise identical to that which created Fig. 1), we assigned drivers 50 and older weights of 0.01, 0.1, 0.5, 2, 10, and 20, respectively, to vary their influence on the regression curves. Drivers 49 and younger were always weighted 1. Fig. 3 shows risk curves produced from these analyses, ranging from circumstance where the influence of drivers age 50 and older was negligible (panel 3a: weight = 0.01) to where the influence of older driver cases is increased twentyfold (panel 3f: weight = 20.0). Confidence intervals are provided for completeness, but we are not interested in post-hoc hypotheses testing with artificially reweighted data, nor do we argue that the confidence intervals are easily interpretable.

These descriptive results show several things. First is that the *general shape* of the sample risk curves is consistent regardless of whether drivers over age 50 are given minimal or exaggerated weight. Although the curves are distorted in panel A due to ceiling effects, overall, the observed increasing elevation in crash risk for older THC-positive drivers does not appear uniquely influenced by the older drivers in the sample. Instead, the pattern is informed by the three-quarters of the sample under age 50. s, rather than inflate expected risk, the data from older drivers appears to flatten the risk curves for both THC-positive and sober drivers; when data from drivers age 50 and older are given greater influence in the analysis, risk levels for those older drivers decrease.

Third, not surprisingly, changes in weighting affected risk curves for THC-positive (which were based on fewer cases) more than they did sober driver. Finally, there appears to be diminishing returns by granting further weight to older drivers. While these analyses do not tell us what would happen if more data were collected, they give confidence that our findings of elevated crash risk for older THC-positive drivers are not artifacts of those older drivers exerting undue leverage on the risk curves.

4. Discussion

As previously documented, both younger drivers and older drivers experience elevated risk of crash involvement compared to middle-aged adults. We hypothesized that cannabis use would exacerbate this risk, although we speculated the reasons would be different for younger drivers than older drivers. We thought loss of impulse control might account for increased risk among younger drivers, while drug-induced cognitive impairment might compound with age-related declines to elevate crash risk for older drivers. Prior research has been inconsistent as to whether and what extent cannabis increases the risk of crash involvement in the general population (Brubacher et al., 2019; Drummer et al., 2020; Lacey et al., 2016), but it might predict crashes at the upper and lower ends of the age distribution.

Using data previously collected and analyzed as part of the NHTSA Drug and Alcohol Crash Risk study, we tested additional models that included interactions between THC and age and age^2, the latter being included to capture curvilinear age relationships. Although we confirmed the original findings of no overall effect for THC (Compton and Berning, 2015; Lacey et al., 2016), after including interactions involving driver age, we found that crash risk among older drivers were significantly divergent as a function of THC. This divergence began to appear in the sample around age 58, and we detected differences as statistically significant at age 64. We did not find strong evidence that younger drivers who tested positive for cannabis experienced increased crash risk.

One strength of the research was the assayed driver specimens for a large number of prescription and illicit drugs that could impair driving. We were able to identify and excluded polydrug users from the analyzed sample, as well as control for alcohol. This makes it easier to attribute observed associations to cannabis alone and not cannabis mixed with other drugs.

The regression analysis compared the upper ends of the THC-positive and the sober crash risk curves, and it is noted that our sample included relatively few cases of THC-positive older drivers involved in crashes. Although the risk curves were informed by thousands of cases each, the

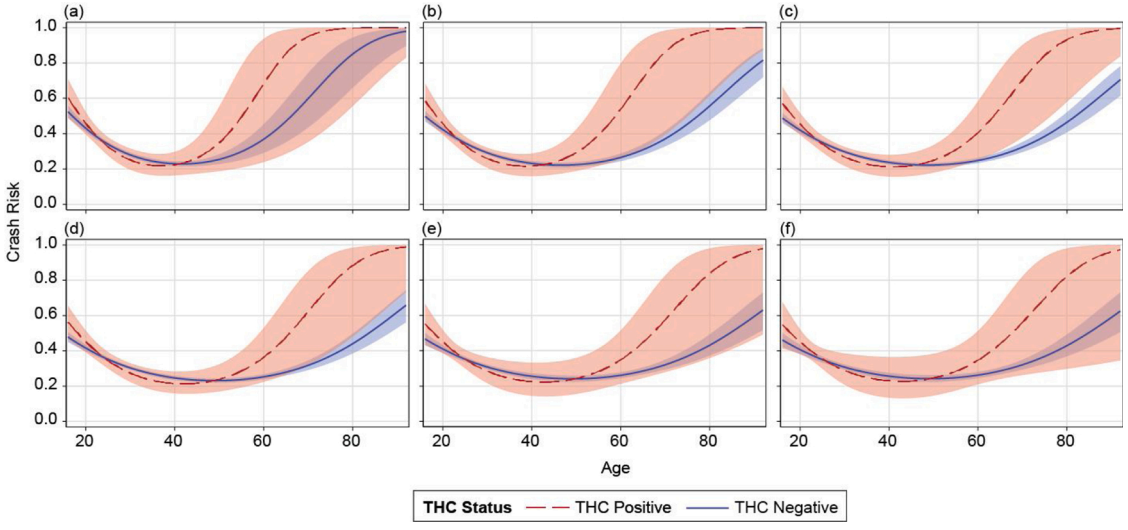

Fig. 3. Crash risk by THC-status and driver age where data from drivers age 50 and older are weighted.
Note: Weights for drivers age 50 and older were 0.01, 0.1, 0.5, 2.0, 10.0, 20.0 for panels A–F, respectively.

confidence intervals around the THC-positive curve (shown in Fig. 1) were quite wide, and only large differences were detected as statistically significant.

We conducted sensitivity analyses to explore how older drivers in our sample contributed to the shape of the risk curves. The key characteristic of our results—increasingly elevated risk curves for THC-positive (relative to sober) older drivers—was persistent; whether the contribution of data from older drivers was minimized or exaggerated, the same general pattern emerged. Furthermore, the actual contribution of data from older drivers was to flatten the curves for both THC-positive and sober drivers, not exacerbate them. When data from older drivers were weighted more heavily, risks were moderated. Although this analytic exercise does not tell us what would happen if we had more data, it does suggest strongly that our observed results are not an artifact of having relatively few THC-positive older drivers in our sample.

Although we hypothesized the results would be caused due to compounding cognitive decline (for older drivers) and increased impulsivity (for younger drivers), we have no data to directly assess this. We have no information, for example, on the cognitive or psychomotor functioning of those older adults involved in crashes, nor do we have information on other medical conditions. It is possible that drivers' underlying medical conditions or chronic pain contributed to the crash but also motivated their cannabis use, and that the association between cannabis and crashes was spurious. This is limitation of the result.

It is important to remember that vehicle crashes are a population-level problem. Fatal crashes and crashes with serious injuries are rare events, but they represent a public health problem burden due to the 3.2 trillion miles that Americans drive annually. It is not trivial that 5.2 % of daytime drivers aged 65 and older (based on analysis of National Roadside Survey data, Kelley-Baker et al., 2017) drive under the influence of cannabis, even if representation in our sample was small. The fact that cannabis use is growing among older adults (Bobitt et al., 2019; Lloyd and Striley, 2018) while more and more drivers are operating vehicles at older ages, we might observe population-level increases in motor vehicle crashes over the next years.

To date, the research on cannabis and driving safety has focused on the problem as one relevant to the general population. However, it is possible that the risks of driving under the influence of cannabis are less global. Our research suggests that the combined trajectories of an aging, driving population and increased cannabis use among older Americans has potential to contribute to public harm. Further research is warranted.

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CRediT authorship contribution statement

Mark B. Johnson: Conceptualization, Formal analysis, Writing - original draft. **Laszlo Mechtler:** Conceptualization, Writing - review & editing. **Bina Ali:** Writing - original draft, Writing - review & editing. **David Swedler:** Writing - review & editing, Formal analysis. **Tara Kelley-Baker:** Writing - review & editing.

Declaration of Competing Interest

The authors report no declarations of interest.

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