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The impact of marijuana decriminalization on California drivers

Robin A. Pollini*, Eduardo Romano, Mark B. Johnson, John H. Lacey

Pacific Institute for Research and Evaluation, 11720 Beltsville Drive, Suite 900, Beltsville, MD 20705, United States

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ABSTRACT

Background: The liberalization of marijuana laws has led to concerns that such changes will increase "drugged driving" and crash-related mortality. California decriminalized marijuana effective January 1, 2011; we examine the impact of this change on marijuana-involved driving.

Methods: We used laboratory testing from roadside surveys and the Fatality Analysis Reporting System (FARS) to assess impacts on weekend nighttime drivers and fatally injured drivers, respectively. We calculated marijuana prevalence (measured by laboratory-confirmed delta-9-tetrahydrocannabinol [THC] in roadside surveys and cannabinoids in FARS) and compared corresponding 95% confidence intervals (CI) to identify statistically significant changes post-decriminalization. We also conducted multiple logistic regression analyses to determine whether the odds of marijuana-involved driving increased significantly after controlling for potential confounders.

Results: There was no statistically significant change in the prevalence of THC-positive driving among weekend nighttime drivers ($n = 894$) in 2012 (9.2%; 95% CI: 6.3, 12.2) compared to 2010 (11.3%; 95% CI: 8.5, 14.0) or in the adjusted odds of testing positive for THC (adjusted odds ratio [AOR] = 0.96; 95% CI: 0.57, 1.60). In contrast, we found a statistically significant increase in the prevalence of cannabinoids among fatally injured drivers in 2012 (17.8%; 95% CI: 14.6, 20.9) compared to the pre-decriminalization period 2008–2010 (11.8%; 95% CI: 10.3, 13.3). The adjusted odds of testing positive for cannabinoids were also significantly higher in 2012 (AOR = 1.67; 95% CI: 1.28, 2.18).

Conclusions: Our study generated discrepant findings regarding the impact of decriminalization on marijuana-involved driving in California. Factors that may have contributed to these findings, particularly methodological factors, are discussed.

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1. Introduction

The United States is in the midst of an historic change related to the legal status of marijuana. By December, 2014, 23 states and the District of Columbia (DC) had legalized the use of marijuana for medicinal purposes (ProCon.org, 2014) and 17 states and DC had decriminalized marijuana, thereby reducing possession of small amounts for personal consumption from a criminal offense to a civil offense punishable by fine (NORML, 2014). In addition, in November 2012, voters passed ballot measures in both Colorado and Washington to legalize marijuana for recreational use, regulating it in a manner similar to alcohol that includes authorizing and taxing retail sales for customers ages 21 and older.

This liberalization of marijuana laws has been accompanied by concerns that such changes will lead to increases in "drugged driving" and crash-related fatalities. These concerns stem from experimental studies showing that marijuana can impair neurocognitive and psychomotor functions important for safe driving including reaction time, road tracking, and multiple task processing (Downey et al., 2013; Hartman and Huestis, 2013; Lenné et al., 2010; Ménétrey et al., 2005; Ramaekers et al., 2008, 2006a,b, 2000; Ronen et al., 2008). These concerns are also fueled by recent epidemiological studies suggesting increases in marijuana-involved driving and increased risk of fatal injury among marijuana-involved drivers (Li et al., 2013, 2012), although studies with similar methodologies have found no association between marijuana and fatal crashes (Romano et al., 2014).

Despite growing concerns regarding marijuana policy changes and their impacts on driving, few studies have directly examined whether laws that expand access to marijuana are directly associated with increases in its use among drivers and, subsequently, fatal crashes. A 2014 study found that cannabinoid prevalence among fatally injured drivers increased significantly in only three

* Corresponding author. Tel.: +1 301 755 2755; fax: +1 301 755 2799.
E-mail addresses: rpollini@pire.org (R.A. Pollini), romano@pire.org (E. Romano), mjohnson@pire.org (M.B. Johnson), lacey@pire.org (J.H. Lacey).

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of 12 states that passed medical marijuana laws (Masten and Guenzburger, 2014). There have been no similar studies of the effects of marijuana decriminalization on drivers in the U.S.

In this paper we address the knowledge gap regarding marijuana decriminalization and driving using California as a case study. Marijuana possession was considered a misdemeanor criminal offense in California until January 1, 2011, when Senate Bill 1449 (SB1449) decriminalized the possession of up to 28.5 grams (≤1 ounce), making it an infraction punishable by maximum $100 fine with no criminal record (State of California, 2010). Did this change result in an increase in marijuana-related "drugged driving" and related fatalities in California? We examine pre- and post-decriminalization marijuana-positive driving among (a) weekend nighttime drivers, and (b) fatally injured drivers, to address this question.

2. Methods

We used drug testing data from roadside surveys and the National Highway Traffic Safety Administration's Fatality Analysis Reporting System (FARS) to quantify the prevalence of marijuana-positive driving before and after marijuana decriminalization among weekend nighttime drivers and fatality injured drivers, respectively. Our goal was to identify any significant increases in marijuana-positive driving that could reasonably be attributed to decriminalization.

2.1. Roadside surveys

2.1.1. Site selection. The 2007 National Roadside Survey (NRS) used survey jurisdictions identified in the National Automotive Sampling System/General Estimates System (NASS/GES) to select a nationally representative sample of 60 survey sites (Lacey et al., 2009). The NASS/GES uses data from hundreds of thousands of vehicle crashes to identify survey locations representative of the continental U.S. as a whole. Five of the 2007 NRS sites were in California (Contra Costa County, Los Angeles County, Orange County, Ventura County, and the City of San Jose).

In 2010, the California Office of Traffic Safety (OTS) funded a replication of the California NRS data collection to generate state-level longitudinal data on alcohol and drug use among drivers (Johnson et al., 2012). Surveys were conducted in San Rafael (at the border with Contra Costa County), Torrance (Los Angeles County), Anaheim (Orange County) and Bakersfield (near Ventura County). We were unable to recruit cooperating police agencies within the City of San Jose, so Fresno was selected as a replacement. For geographic balance we also added the city of Eureka, located in Northern California (Humboldt County).

OTS funded roadside surveys again in 2012 to continue generating longitudinal data. Of the six sites surveyed in 2010, four were surveyed again in 2012: Anaheim, Eureka, Fresno, and San Rafael (we were unable to recruit police cooperation in Torrance and Bakersfield). These four sites constitute the roadside survey sample for our pre- and post-decriminalization analysis.

2.1.2. Data collection procedures. The procedures for the 2010 and 2012 surveys were similar to the 2007 NRS (Lacey et al., 2009) and were approved by the Pacific Institute for Research and Evaluation's Institutional Review Board. At each location, four specific 1-mile square areas were selected at random and a specific survey site was chosen in each area. The roadside surveys were stratified by day (Friday and Saturday) and time of night (10PM-midnight and 1–3AM), with each survey site assigned to one of the strata. Sites were located in lit parking areas alongside roadways with ample space for vehicles to enter and depart, and each was organized into three to four research bays. A police officer (or officers) was positioned on the roadway outside the interview area to manage traffic and vehicle recruitment and help ensure safety. Orange traffic signs that read "Voluntary Survey Ahead" were situated by the roadside several blocks upstream of the survey site and at the survey site entrance to alert drivers to the data collection activity.

For each survey, the police officer would attract the attention of a driver in an oncoming vehicle and wave that vehicle into the survey site. To minimize selection bias, the officer waved the third approaching vehicle into the site until all survey bays were filled. The next approaching vehicle was waved into survey bays as they became vacant. There were no consequences for drivers who ignored the police officer or failed to pull into the survey area as directed.

As a vehicle pulled into a research bay, the driver was immediately approached by a research assistant who said "you have done nothing wrong," and informed the driver that he or she was selected at random to take part in a survey. All potential participants were informed that the survey was voluntary and anonymous, and that they would earn $20 for participating.

2.1.3. Roadside survey interview. Data collection on consenting drivers involved five parts: (a) an interviewer-administered interview concerning general driving practices, demographics, drinking history (frequency and quantity); (b) breath test using a calibrated Intoxilyzer 400™ preliminary breath test (PBT); (c) a self-administered pencil-and-paper survey on drug use; (d) a pencil-and-paper survey on alcohol and drug problems based on the Alcohol Use Disorder and Associated Disabilities Interview Schedule (AUDASIS) (Grant et al., 1995); and (e) an anonymous oral fluid sample using the Quantisal™ data collection kit (Immunalysis Corporation, Pomona, CA).

2.1.4. Drug analysis and screening. Oral fluid samples were sent to Immunalysis Corporation for processing. All samples were initially screened using enzyme-linked immunosorbent assay (ELISA) microplate technology. For positive screening results, confirmation was performed using gas chromatography–mass spectrometry (GC/MS) or liquid chromatography–mass spectrometry (LC/MS/MS) technology. All the analytical procedures used for drug testing were fully validated according to established protocols. Negative, low-level, and high-level controls were run for each batch, along with calibration standards.

The drugs tested by bioassay included delta-9-tetrahydrocannabinol (THC; the active drug only and not metabolites) and a variety of other legal and illegal drugs (Lacey et al., 2011). The method for assaying oral fluid for THC has a limitation of quantification of 1 ng/mL, linearity of 0.5–32 ng/mL, intraday precision of 7.1% at 3 ng/mL and 2.9% at 12 ng/mL, and interday precision of 4.9% at 3 ng/mL and 1.6% at 12 ng/mL (6 replications over 5 days). The assay methods and limitations for THC did not differ for the 2010 and 2012 studies, nor did the proportion of recruited participants providing oral fluid samples at the four sites (78.4% in 2010 and 78.9% in 2012).

2.1.5. Key measures. The key dependent variable for this analysis was the presence or absence of THC. The key independent variable for the analysis was year (2012 vs. 2010).

2.1.6. Data analysis. We calculated THC prevalence and related 95% confidence intervals (95% CIs) for each of the four survey communities and compared site-specific confidence intervals by year to identify statistically significant changes in THC-positivity between 2010 and 2012. Then, using THC positivity as the dependent variable and year as the primary independent variable of interest, we aggregated data by site and calculated univariate odds ratios for all potential confounding variables; those that achieved significance at $p < 0.10$ were included in a multiple logistic regression model to determine whether there was a statistically significant increase in the odds of testing THC-positive in 2012 compared to 2010 after controlling for potential confounders.

2.2. Fatality Analysis Reporting System (FARS)

FARS is a census of all crashes on U.S. public roads that result in a death within 30 days. It contains an estimate of the blood alcohol content (BAC) of every driver involved in a fatal crash, consisting of either an actual BAC measurement or an imputed value based on other factors in the crash (Subramanian, 2002). Drug information is more limited but 20 states (including California) provide drug testing results for at least 80% of their fatally injured drivers, which minimizes the likelihood of selection bias that might arise from reporting only occasional, court-mandated analyses (Hingson et al., 2010).

2.2.1. Case selection. We used FARS data for 2008–2012. Because we did not have FARS data specific to the communities where we conducted roadside surveys (FARS data is only available by county, and would have provided only $N = 266$ cases for the four counties in the 2008–2012 study period) we used statewide data for this portion of the analysis. We limited our cases to drivers who were tested for drugs and had a known lab results for cannabinoids ($N = 6776$; see key measures below). To ensure proper identification of crash responsibility, we excluded drivers who: (i) presented a condition signaling them as mentally challenged; (ii) were involved in a police chase; (iii) were driving a bus, snowmobile, motorized wheelchair, construction or farm equipment; or (iv) were parked or in the process of parking a vehicle ($N = 781$). We also excluded multiple vehicle crashes ($N = 3135$) to limit our sample further to fatally injured drivers (surviving drivers are rarely tested for drugs) in single vehicle crashes (in which the driver was probably responsible for the crash), which is a selection strategy typical of many studies using driver fatality data (Haddon and Bradess, 1959; Romano et al., 2011; Romano and Pollini, 2013; Williams and Shabanova, 2003). This approach yielded a total study sample of $N = 2860$.

2.2.2. Key measures. FARS uses three variables to account for up to three drugs detected per case. Each variable is assigned a drug code: 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). The "Cannabinoids" category included test results for delta 9, hashish oil, hashish, marijuana, marinol, tetrahydrocannabinoid, THC, and "Cannabinoid, Type Unknown" (National Highway Traffic Safety Administration, 2011). For this study we considered drivers to be positive for cannabinoids if they tested positive for any substance in the cannabinoid category.

2.2.3. Data analysis. We estimated cannabinoid prevalence and computed 95% CIs for the pre-decriminalization period (2008–2010) and for each year post-decriminalization (2011, 2012). Comparisons between prevalence estimates were conducted by identifying any overlap in 95% CIs. We repeated these calculations stratifying by sex and age categories (16–20 years, 21–34 years, 36–64 years, and ≥65 years). We then calculated univariate odds ratios and 95% CIs for year, sex, and age, and conducted multiple logistic regression modeling to determine whether there was a significant increase in the odds of testing positive for cannabinoids in 2011 and/or 2012 after controlling for sex and age as potential confounders.

3. Results

3.1. Roadside surveys

Table 1 shows THC prevalence by year at each of the four roadside survey sites. Prevalence varied widely by site, with the Northern California sites (Eureka and San Rafael) demonstrating substantially higher prevalence than those in Central (Fresno) and Southern (Anaheim) California. However, prevalence did not differ significantly between 2010 and 2012, either overall or by site.

Table 2 presents results from our aggregated analysis of THC-positivity and its correlates. Several variables were significantly associated ($p < 0.05$) with THC-positivity in the univariate analysis including survey site, survey session, BAC, past year drinking, self-perceived drinking type, meeting criteria for alcohol abuse, educational attainment, employment status, age, sex, and race/ethnicity; however, year of survey (2012 vs. 2010) was not significantly associated with THC-positivity (odds ratio [OR] = 0.80; 95% CI: 0.52, 1.25). After controlling for potential confounders ($p < 0.10$), survey year still was not independently associated with THC-positivity (adjusted odds ratio [AOR] = 0.96; 95% CI: 0.57, 1.60).

Table 1
THC prevalence among roadside survey respondents in four California communities, 2010 and 2012.

Site	2010		2012	
	N	% THC-positive (95% CI)	*N*	% THC-positive (95% CI)
Anaheim	140	4.3 (0.9, 7.7)	136	6.6 (2.4, 10.8)
Eureka	148	18.9 (12.5, 25.3)	88	19.3 (10.9, 27.7)
Fresno	96	4.2 (0.1, 8.2)	92	3.3 (0.0, 7.0)
San Rafael	131	15.3 (9.0, 21.5)	63	9.5 (2.1, 17.0)
Total	515	11.3 (8.5, 14.0)	379	9.2 (6.3, 12.2)

Table 2
Odds of testing positive for THC among roadside survey respondents in four California communities, 2010 and 2012.

	Total (n = 894)	Odds ratio (95% CI)	Adjusted odds ratio (95% CI)
Year			
2010	515 (57.6)	1.00	1.00
2012	379 (42.4)	0.80 (0.52, 1.25)	0.96 (0.57, 1.60)
Site			
Anaheim	276 (30.9)	1.00	1.00
Eureka**	236 (26.4)	4.10 (2.22, 7.57)	3.46 (1.68, 7.11)
Fresno	188 (21.0)	0.67 (0.27, 1.68)	0.58 (0.22, 1.56)
San Rafael**	194 (21.7)	2.69 (1.39, 5.23)	2.94 (1.39, 6.22)
Session			
Friday early	252 (28.2)	1.00	1.00
Friday late*	174 (19.5)	1.88 (0.98, 3.62)	1.41 (0.69, 2.87)
Saturday early	269 (30.1)	1.45 (0.78, 2.70)	1.45 (0.74, 2.84)
Saturday late**	199 (22.3)	1.95 (1.04, 3.68)	1.32 (0.65, 2.68)
BAC > 0**	57 (6.5)	2.17 (1.08, 4.36)	1.80 (0.77, 4.16)
Ever drink past 12 months**	630 (71.0)	2.12 (1.21, 3.71)	0.95 (0.10, 8.73)
Type of drinker			
Non-drinker[a]	260 (29.4)	1.00	1.00
V/F light**	494 (79.0)	1.83 (1.03, 3.28)	2.09 (0.24, 17.85)
Moderate**	114 (18.2)	3.65 (1.83, 7.25)	4.26 (0.49, 36.99)
V/F heavy	17 (2.7)	0.95 (0.12, 7.65)	–
Weekly alcohol consumption			
None	244 (39.0)	1.00	1.00
1–2 drinks	241 (38.6)	1.27 (0.76, 3.22)	0.72 (0.38, 1.35)
>2 drinks*	140 (22.4)	1.70 (0.99, 2.92)	0.64 (0.29, 1.40)
Drove after drinking too much in past 12 months	53 (8.5)	1.57 (0.71, 3.43)	
Meet criteria for alcohol abuse**	51 (8.1)	2.54 (1.25, 5.14)	2.78 (1.21, 6.40)
Meet criteria for alcohol dependence	31 (4.9)	1.89 (0.76, 4.70)	
Education			
≤HS grad	209 (23.7)	1.00	1.00
Some college**	376 (42.6)	1.89 (1.05, 3.41)	1.77 (0.91, 3.43)
≥College	297 (33.7)	1.12 (0.58, 2.13)	1.24 (0.58, 2.66)
Employed**	676 (77.1)	0.62 (0.39, 0.99)	0.66 (0.38, 1.16)
Median age in years (IQR)**	30 (22–43)	0.98 (0.96, 1.00)	0.98 (0.96, 1.00)
Sex**			
Female	334 (39.5)	1.00	1.00
Male	512 (60.5)	1.80 (1.11, 2.91)	1.98 (1.16, 3.67)
Race/ethnicity			
White non-Hispanic/Latino	426 (48.3)	1.00	1.00
White Hispanic/Latino**	128 (14.5)	0.35 (0.16, 0.79)	0.56 (0.23, 1.38)
Black	39 (4.4)	1.83 (0.83, 4.05)	2.01 (0.81, 5.00)
Asian**	86 (9.8)	0.30 (0.11, 0.84)	0.42 (0.14, 1.26)
>1 race**	181 (20.5)	0.43 (0.23, 0.83)	0.53 (0.25, 1.16)
Other	22 (2.5)	0.29 (0.04, 2.20)	0.30 (0.04, 2.38)

Columns may not sum to *N* due to missing values.
[a] Did not drink in past year.
* $p < 0.10$.
** $p < 0.05$.

Table 3
Cannabinoid prevalence among fatally injured drivers in California, Fatality Analysis Reporting System, 2008–2012.

	2008–2010 (*N* = 1718)		2011 (*N* = 574)		2012 (*N* = 568)	
	N	% (95% CI)	*N*	% (95% CI)	*N*	% (95% CI)
Overall						
Cannabinoid positive	203	11.8 (10.3, 13.3)	74	12.9 (10.2, 15.6)	101	**17.8 (14.6, 20.9)**
Cannabinoid negative	1515	89.4 (88.4, 90.4)	500	87.4 (85.5, 89.3)	467	**82.0 (79.8, 84.2)**
Males						
Cannabinoid positive	173	12.7 (10.9, 14.5)	66	14.4 (11.2, 17.6)	87	**18.1 (14.7, 21.5)**
Cannabinoid negative	1190	87.3 (85.5, 89.2)	392	85.6 (82.4, 88.8)	394	**81.9 (78.5, 85.3)**
Females						
Cannabinoid positive	30	8.5 (5.6, 11.3)	8	6.9 (2.3, 11.5)	14	16.1 (8.3, 23.8)
Cannabinoid negative	325	91.6 (88.7, 94.4)	108	93.1 (88.5, 97.7)	73	83.9 (76.2, 91.6)
Age 16–20						
Cannabinoid positive	41	21.1 (15.4, 26.9)	14	25.9 (14.2, 37.6)	14	35.0 (20.2, 49.8)
Cannabinoid negative	153	78.9 (73.1, 84.6)	40	74.1 (62.4, 85.8)	26	65.0 (50.2, 79.8)
Age 21–34						
Cannabinoid positive	97	16.0 (13.1, 19.0)	30	13.3 (8.9, 17.8)	44	20.4 (15.0, 25.7)
Cannabinoid negative	508	83.9 (81.0, 86.9)	195	86.7 (82.2, 91.1)	172	79.6 (74.3, 85.0)
Age 35–64						
Cannabinoid positive	60	8.3 (6.3, 10.4)	27	10.9 (7.0, 14.8)	41	**16.2 (11.7, 20.8)**
Cannabinoid negative	660	91.6 (89.6, 93.7)	220	89.1 (85.2, 93.0)	212	**83.8 (79.3, 88.3)**
Age 65+						
Cannabinoid positive	4	2.0 (0.0, 4.0)	3	6.3 (0.0, 13.1)	2	3.5 (0.0,8.1)
Cannabinoid negative	193	98.0 (96.0, 99.9)	45	93.8 (86.9, 99.9)	56	96.6 (91.9, 99.9)

Bold font indicates statistically significant difference compared with the 2008–2010 period ($p < 0.05$).

3.2. Fatality Analysis Reporting System (FARS)

The percentage of fatally injured drivers in each of California's 58 counties who tested positive for cannabinoids ranged from 0 to 40.0% during 2008–2010, 0 to 50.0% in 2011, and 0 to 100.0% in 2012. Table 3 shows that the overall prevalence of cannabinoids among fatally injured drivers statewide for these same time periods was 11.8% (95% CI: 10.3, 13.3), 12.9% (10.2, 15.6), and 17.8% (14.6, 20.9). This constitutes a statistically significant increase in 2012 compared to the pre-decriminalization period of 2008–2010. The increase from 2008–2010 and 2011 was not statistically significant. Table 3 also provides a more detailed breakdown of the gender and age characteristics of fatally injured drivers. The increase in cannabinoid prevalence in 2012 was statistically significant for males and for those ages 35–64 years but was not significant for any subpopulation in 2011.

Table 4 presents the results of multiple logistic regression modeling. After controlling for sex and age, there was a statistically significant increase the odds of testing positive for cannabinoids among fatally injured drivers in 2012 vs. 2008–2010 (AOR = 1.67; 95% CI: 1.28, 2.18) but not for 2011 vs. 2008–2010 (AOR = 1.10; 95% CI: 0.82, 1.46).

Table 4
Odds of testing positive for cannabinoids among fatally injured drivers in California, Fatality Analysis Reporting System, 2008–2012.

	Adjusted odds ratio (95% CI)
Year	
2008–2010	1.00
2011	1.10 (0.82, 1.46)
2012	1.67 (1.28, 2.18)
Sex	
Female	1.00
Male	1.54 (1.12, 2.10)
Age (years)	
16–20	2.85 (2.05, 3.97)
21–34	1.67 (1.30, 2.13)
35–64	1.00
65–74	0.27 (0.14, 0.53)

4. Discussion

This study used two different data sources – roadside survey data from randomly selected nighttime weekend drivers and FARS data from fatally injured drivers involved in single-vehicle crashes – to assess the impact of California's marijuana decriminalization law on driving. We did not anticipate that our analyses of these two data sources would produce discrepant findings; nonetheless, we found a significant post-decriminalization increase in cannabis-positive driving among fatally injured drivers while finding no significant changes in THC-positive driving among nighttime weekend drivers.

These conflicting findings between roadside-surveyed drivers and drivers killed in crashes are puzzling. The primary mechanism by which decriminalization would contribute to increased crash fatalities is through increased prevalence of marijuana use in the driving population, which in turn would increase the number of drivers at risk of marijuana-involved crash fatalities. The fact that we did not find a significant increase in THC-positive nighttime weekend driving (as we did find among fatally injured drivers) requires further examination.

One explanation is that decriminalization led to an increase in the risk of fatal crashes among drivers who were already using marijuana – perhaps by increasing the typical dosage – but did not expand the prevalence of marijuana use among drivers in general. This might account for an increase in THC-positive fatalities but no change in our roadside survey sample. However, examination of THC concentration data from our roadside surveys indicated no statistically significant difference between 2010 and 2012 (27.5 vs. 26.0 ng/mL, $p = 0.282$). Thus, our data suggest no change in THC concentration before and after decriminalization.

Another explanation for the observed discrepancy between the roadside survey and FARS findings is that the roadside surveys missed a number of THC-positive drivers. Unlike the FARS (which is a census of fatal crashes), not all drivers that we attempted to recruit agreed to provide oral fluid samples. If THC-positive drivers are systematically more likely to refuse oral fluid testing, then our estimates of the prevalence of THC-positive drivers would be biased down. However, if the prevalence of THC among drivers actually increased in 2012, consistent with our FARS findings,

then the degree of selection bias would have to have increased substantially from 2010 to 2012 to yield statistically similar results. This is possible but unlikely given that all other aspects of the roadside survey were consistent over the 2 years.

A final explanation for the observed differences relates to possible changes in the crash data collected by FARS. Unlike the roadside surveys, FARS was not specifically designed to facilitate comparisons of drug prevalence over time. Specifically, although FARS seeks to provide uniform recording and presentation of fatal crash data, it relies on information from individual officers, investigators, and laboratories that is not necessarily collected uniformly across jurisdictions and/or over time. For example, changes in vigilance regarding "drugged driving" enforcement over time could bias case selection; in this study, the proportion of fatally injured drivers testing positive for cannabinoids in FARS increased from a maximum of 40% in 2008–2010 to a maximum of 100% in 2012, suggesting that targeted testing surely is in effect and may have increased over the study period. The Office of National Drug Control Policy (ONDCP) named "drugged driving" as one of three national drug policy priorities in its 2011 National Drug Control Strategy (Office of National Drug Control Policy, 2011), which may have contributed to increases in drug testing among fatally injured drivers. If efforts were made by California law enforcement to increase cannabinoid detection during the study period then our findings of increased prevalence may have been caused by changes in case selection rather than by an actual increase in cannabinoid prevalence among fatally injured drivers.

Lack of standardization in laboratory testing for cannabinoids, and changes in testing protocols over time, could also influence the number of drivers who screen positive for cannabinoids in FARS. Some jurisdictions do their own drug testing while others contract out to private laboratories, and testing methods may vary. The types of drugs and metabolites tested for and the sensitivity of tests and their "cut-off" levels can all vary across jurisdictions and over time; when a jurisdiction adds metabolites to its testing panel, or increases the sensitivity of a screening test, this can result in increases in the detection of cannabinoid-positive cases and an increase in prevalence estimates independent of any actual increase in cannabinoid-positive driving. Methods for cannabinoid testing across jurisdictions are poorly documented; however, personal communications with experts and preliminary results of a pilot survey in California conducted by our research team suggest that the types of cannabinoids and metabolites tested for differ across jurisdictions and changes over time do occur, making time-based comparisons of drug results in FARS vulnerable to bias.

Similar concerns about case selection have been raised with regard to state-based differences in testing protocols, and some authors have attempted to correct for such bias by applying mixed models that treat state as a random variable in their analyses (Romano and Pollini, 2013; Romano et al., 2014). Although this approach allows for meaningful broad studies (e.g., national estimates of prevalence), valid comparisons of drug use between states are not possible as long as case selection methods and lab protocols in each state are unknown. By the same token, direct comparisons of drug prevalence between two time periods (even within a state) may yield meaningless results if changes in lab testing protocols occurred during the study period.

Our study is subject to other limitations. Because of the small number of driver fatalities in the communities that participated in our roadside survey, it was not possible to limit our FARS analysis to those communities. Second, FARS data reported laboratory results for a wider range of substances than the roadside survey; changes in use of these substances (exclusive of THC) during the study period could also have influenced our results. It should be noted that while the roadside survey reported all drugs present among tested drivers, the FARS dataset allows only three drugs to be entered into the dataset; in the event that marijuana was present in a fatally injured driver but was listed after at least three other drugs the case would not have been included neither in the FARS data set nor in our study. Finally, it is important to note that our study examines the presence of THC and/or other cannabinoids (including inactive metabolites) among drivers, not impairment, and our findings should not be interpreted as providing information on impaired driving.

In summary, this study found no increases in THC among night-time weekend drivers following marijuana decriminalization but an increase in cannabinoid prevalence among fatally injured drivers over the same period. A definitive interpretation of these discrepant findings requires further investigation; however, our study demonstrates the need for better, standardized data to inform policy making on "drugged driving."

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Contributors

R.A.P, E.R., M.B.J., and J.H.L designed the study. J.H.L. provided datasets for the roadside surveys. R.A.P. and E.R. analyzed the datasets in consultation with M.B.J. R.A.P. and E.R. wrote the first draft of the manuscript. All authors contributed to and have approved the final manuscript.

Conflicts of interest

No conflict declared.

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