Alcohol-Related Risk of Driver Fatalities in Motor Vehicle Crashes: Comparing Data From 2007 and 2013–2014

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ABSTRACT. Objective: Using data from 2013–2014, this article aims to update alcohol-related fatal crash relative risk estimates, defined as the risk of dying in those crashes at different blood alcohol concentrations (BACs) relative to the risk of dying in a crash when sober (BAC = .00 g/dl), and to examine any change in risk that could have taken place between 2007 and 2013–2014. More specifically, we examine changes in risk among BAC = .00 g/dl drivers and among BAC > .00 g/dl drivers. **Method:** We matched and merged crash data from the Fatality Analysis Reporting System (FARS) and exposure data from the National Roadside Survey (NRS). To the matched database we applied logistic regression to estimate the changes in relative risk. **Results:** We found that

among sober (BAC = .00 g/dl) drivers, the risk of dying in a fatal crash decreased between 2007 and 2013–2014. For drinking drivers, however, no parallel reduction in the overall contribution of alcohol to the fatal crash risk occurred. Compared with 2007, in 2013–2014 the oldest group of drivers (age ≥ 35 years) were at an elevated crash risk when driving at low BACs (.00 g/dl < BAC < .02 g/dl). **Conclusions:** Although the decrease in crash risk for drivers with a BAC of .00 g/dl is encouraging, the consistency of the alcohol-related risk estimates over the last two decades suggests the need to substantially strengthen current efforts to abate drinking and driving. (*J. Stud. Alcohol Drugs*, 79, 547–552, 2018)

LABORATORY RESEARCH clearly shows that alcohol consumption impairs driving skills (e.g., Moskowitz et al., 1985; National Institute on Alcohol Abuse and Alcoholism, 1997). To corroborate these laboratory findings, several field-based studies have been conducted. The contribution of alcohol to fatal crashes was explored by Zador (1991) and Zador et al. (2000) by linking driving data from the 1996 National Roadside Survey (NRS)—a quasi-decennial effort sponsored by the National Highway Traffic Safety Administration (NHTSA) and others—with fatalities from the Fatality Analysis Reporting System (FARS)—a census of all fatal crashes in the United States. Like in the laboratory-based studies, the authors also reported an increase in crash risk associated with increases in blood alcohol concentration (BAC).

In 2012, this research team updated the estimates of Zador et al. (2000) by linking the 2007 NRS with FARS data (Voas et al., 2012). Following a study design that repeated as closely as possible the analytical procedures of the previous study, the authors reported estimates of fatal crashes similar to those reported a decade earlier by Zador et al. (2000). The sole exception occurred among young female drivers (ages 16–20), who in 2007 faced crash risks comparable to those

of their male counterparts. We also found that young drivers were more likely to crash than ever before, even if they were sober (BAC = .00 g/dl), a finding we hypothesized was related to the increasing popularity of texting and electronic devices.

The completion of the 2013–2014 NRS conducted by NHTSA and the Pacific Institute for Research and Evaluation (PIRE) (Compton & Berning, 2015; Ramirez, 2016) provides an opportunity to further update these risk estimates. By matching the 2007 and 2013–2014 NRSs with single-vehicle fatal crash data from the FARS and following analytical procedures used in previous studies as close as possible, this article aims to (a) update fatal risk estimates for sober (BAC = .00 g/dl) drivers killed in single-vehicle crashes. We hypothesize that an increased use of in-vehicle communication devices (e.g., texting) and in risk-taking driving behaviors by women has increased the crash risk for sober (BAC = .00 g/dl) drivers, particularly if they are young. We also aim to (b) update alcohol-related fatal crash risk estimates, that is, the relative risk (RR) of dying in a crash at different BACs relative to the risk of dying in a crash when sober (BAC = .00 g/dl) and (c) examine any change in fatal RR (by age, gender, and BAC) that could have taken place between 2007 and 2013–2014.

Method

Data

Exposure. Exposure data for this study come from the 2007 and 2013–2014 NRSs, studies aimed at estimating the

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incidence of alcohol and drug use in drivers on our nation's roadways. More than 9,000 drivers were interviewed in 2007 (Compton & Berning, 2009; Lacey et al., 2009b) and another 9,400 provided breath samples in 2013–2014 (Berning & Smither, 2014) to determine the prevalence of drivers at various BACs, as well as the prevalence of drivers having various over-the-counter, prescription, and illegal drugs in their systems.

The 2007 and 2013–2014 NRSs limited the area covered to the 48 contiguous states. Surveys were conducted on Friday and Saturday nights (10 P.M.–midnight and 1 A.M.–3 A.M.), when heavy drinking is most likely to occur and alcohol-involved crashes are most frequent (Lestina et al., 1999). Because of logistic and safety concerns, counties with populations of less than 20,000 were not surveyed, and in counties with larger populations, only roadways with 2,000–4,000 average daily traffic counts were surveyed. Finally, commercial vehicle operators were excluded. Thus, the NRS studies provide information on private four-wheel vehicle operators at randomly selected locations during periods when drinking and driving is most prevalent. Survey details appear in Ramirez et al. (2016).

The overall participation rates for the 2007 and 2013–2014 NRSs were fairly high (83.4% for the 2007 NRS and 79.3% for the 2013–2014 NRS, for the total sample of drivers able to participate in the survey). Results were summarized by Berning et al. (2015), who reported an overall decrease in the prevalence of drinking drivers over time, with those at BACs $\geq .08$ decreasing from 7.5% in 1973 to 1.5% in 2013–2014.

Fatal crash data. Data on motor vehicle crash fatalities came from the FARS. To match the 2007 and 2013–2014 NRSs, we selected drivers age 16 years and older at the time of the fatal crash, driving four-wheel passenger vehicles, involved in fatal crashes occurring on a weekend night, in a county with a population of at least 20,000, outside of special jurisdictions (i.e., reservations), and on a paved road not classified as an interstate, other urban freeway, or expressway. There were only two notable differences between the exposure and the crash-screening criteria, and both were disregarded to increase the sample size for drivers retained for the analyses. First, we accepted crashes that occurred between midnight and 1 A.M. Exposure measures at that time interval were not recorded, as the survey teams moved to another location. However, because BAC distribution between midnight and 1 A.M. is thought to be similar to neighboring hours, crashes occurring in that interval were nevertheless included in the analyses. Second, we did not restrict crashes to the weekend nights that the surveys were conducted but include weekend nights for the whole year. This decision increased sample sizes almost 12-fold and introduced no substantial difference in the distribution of drivers' BACs because BACs will vary little between the survey period and the rest of the year.

Analyses

Like Voas et al. (2012) and Zador et al. (2000), we used a population-based case-control study in this work. We applied logistic regression to estimate the RR of crashing at different BACs relative to that at BAC = .00 g/dl for single-vehicle as well as all-vehicle fatal crashes. We approximated the RR of a fatal crash by computing its odds ratio (OR) based on a logistic model (Agresti, 2002). The logistic regression involved a binary response that took a value of 0 or 1 depending on whether the driver belonged to the "case" population (FARS) or the control population (NRS), respectively. We included sex, age, and BAC—and all the possible interactions among them—as possible predictors for the RR of a fatal crash. To avoid extremely small coefficient estimates, the actual and imputed BAC values were rescaled by a factor of 1,000 (Zador et al., 2000). Because the number of underage drivers (ages 16–20) with BAC $\geq .08$ g/dl in the 2013–2014 NRS was very small, we grouped these drivers to deal with very large standard errors of estimates. As in previous efforts, drivers were classified into the following age groups: 16–20, 21–34, and 35 and older. We separately ran regressions for all drivers and for those at BAC = .00 g/dl only. New to this effort, we also included a "year effect" to account for changes in risk between the years 2007 and 2013–2014.

As in Voas et al. (2012) and Romano et al. (2014), we applied a resampling method to account for the variability attributable to the NRS sample design (Brick et al., 2000). We also applied the delete-one jackknife method, which is appropriate for multistage designs with a small sampling fraction of units selected in the first stage (Levy & Lemeshow, 2009). For the 2007 and 2013–2014 NRSs, the sampling fraction in the first sampling stage was approximately 6% of all primary sampling units. We tried 60 replicate weights to estimate standard errors corresponding to the 60 primary sampling units included in the first stage of the sampling design in each of the NRS samples (Lacey et al., 2009a; Ramirez et al., 2016). The replicate weights were calculated with the information of the different stages involved in the sampling design (Lacey et al., 2010). We used the PROC SURVEYLOGISTIC procedure of the SAS software, Version 9.4 of the SAS System for Windows (SAS Institute, Cary, NC). Inference for parameters in the logistic models based on multiple imputations was performed using SAS PROC MIANALYZE (Berglund, 2010). Estimates were thus obtained by using each imputed data set and the 60 replicate weights based on the delete-one jackknife method, and this procedure was repeated for each of the 10 imputed FARS data sets. The resulting estimates were subsequently combined to obtain the final estimates of interest with their respective standard errors. To determine whether a term in the logistic models was significant, a combined Wald-type test statistic based on the multiple imputations was calculated (O'Kelly & Ratitch, 2014). For each model, we also

TABLE 1. Logistic regression coefficients (standard errors) in models for risk of driver fatalities in single-vehicle, and all crashes as a function of year of NRS survey, age, sex, and BAC

Variable	Sober drivers (BAC = .00)		All BACs	
	FSV Coeff. (SE)	FAV Coeff. (SE)	FSV Coeff. (SE)	FAV Coeff. (SE)
Main effects				
Intercept	-2.305 (0.229)	-1.649 (0.227)	-2.303 (0.206)	-1.677 (0.209)
Year: 2013–2014 (ref.: 2007)	-0.578 (0.251)	-0.683 (0.244)	-0.556 (0.230)	-0.653 (0.228)
Female (ref.: male)	-0.732 (0.125)	-0.433 (0.114)	-0.803 (0.048)	-0.519 (0.042)
Age 16–20 years	1.180 (0.132)	0.994 (0.127)	1.200 (0.087)	0.998 (0.042)
Age ≥35 years (ref.: age 21–34 years)	-0.202 (0.105)	0.004 (0.093)	-0.171 (0.074)	0.075 (0.062)
BAC	—	—	0.035 (0.003)	0.031 (0.002)
Interactions				
Age 16–20, 2013–2014	-0.149 (0.169)	-0.082 (0.159)	-0.225 (0.135)	-0.139 (0.12)
Age ≥35, 2013–2014 (ref.: age 21–34, 2007)	0.265 (0.150)	0.108 (0.124)	0.206 (0.114)	0.104 (0.087)
Female, 2013–2014 (ref.: male, 2007)	-0.030 (0.154)	-0.034 (0.136)	—	—
Age 16–20, female	-0.001 (0.181)	-0.146 (0.172)	—	—
Age ≥35, female (ref.: age 21–34, male, 2007)	0.210 (0.158)	0.126 (0.128)	—	—
Age 16–20, female, 2013–2014	-0.298 (0.225)	-0.132 (0.215)	—	—
Age ≥35, female, 2013–2014 (ref.: age 21–34, male, 2007)	-0.280 (0.227)	-0.103 (0.175)	—	—
Age 16–20, BAC	—	—	0.004 (0.005)	0.004 (0.005)
Age ≥35, BAC (ref.: age 21–34, BAC)	—	—	-0.001 (0.004)	-0.003 (0.003)
2013, BAC (ref.: 2007, BAC)	—	—	0.002 (0.004)	0.003 (0.004)
Age 16–20, 2013–2014, BAC	—	—	0.030 (0.014)	0.027 (0.012)
Age ≥35, 2013–2014, BAC (ref.: age 21–34, 2007, BAC)	—	—	-0.001 (0.005)	-0.001 (0.005)
Max. rescaled R^2	.077	.065	.737	.656

Notes: Sober drivers (BAC = .00) denotes regressions based only on BAC = .00g/dl drivers. All BACs denotes regressions based only drivers at any BAC. Data come from the 2013–2014 National Roadside Survey and matching FARS databases. Coefficients (coeff.) in **bold** indicate statistical significance ($p < .05$) using a pooled Wald-statistic calculated over multiple imputations. NRS = National Roadside Survey; BAC = blood alcohol concentration; coeff. = coefficient; FAV = fatalities in all vehicle crashes; FSV = fatalities in single-vehicle crashes; ref. = reference.

computed the maximum of rescaled R^2 , a modified version of the generalized coefficient of determination that is used to measure the goodness-of-fit in logistic models (Menard, 2000).

Results

As expected, among sober (BAC = .00 g/dl) drivers (the leftmost half of Table 1), male drivers ages 16–20 have the highest risk of dying in a crash—2.8 (fatalities in single-vehicle crashes [FSV]) and 2.5 (fatalities in all vehicle crashes [FAV]) times higher than that of male drivers ages 21–34. Relative to 2007, the risk a sober driver (BAC = .00 g/dl) would die in a crash in 2013–2014 decreased regardless of the crash type and driver's age.

The contribution of alcohol to fatal crash risk is shown in the rightmost half of Table 1. Also as expected, crash risk was higher for drivers who were ages 16–20, male, and had a positive BAC. The older group (age ≥ 35) showed the lowest risk of dying in a single-vehicle crash but higher than those

ages 21–34 when all crashes are considered. Female drivers were less likely to be involved in a fatal crash than their male counterparts. Only one of the interactions was found to be significant: In the examination of all crashes, we found the Age 16–20 × 2013–2014 × BAC interaction to be positive and statistically significant. This finding seems to suggest that the crash risk for drivers ages 16–20 with positive BACs increased between 2007 and 2013–2014. However, this finding must be accepted with caution because of the limited number of underage drivers (16–20 years old) in the selected 2013–2014 NRS sample. To address this concern, we fitted a variety of alternative models, including a model with drivers' age groups re-classified into 16–21, 22–34, and 35 and older; models exploring a variety of interaction terms; and models treating BAC as a categorical variable. Although not shown, the results were essentially similar to those in Table 1. The only difference was that the Age 16–21 × 2013–2014 × BAC interaction was no longer significant, a finding that emphasizes the need to accept the significance of this interaction with extreme caution.

TABLE 2. Relative risk estimates [and 95% confidence intervals] of driver fatalities as a function of drivers' BAC by crash type and drivers' age, relative to sober drivers of the same age group

Crash type	Model	Age group	BAC					
			.001 g/dl– .019 g/dl	.020 g/dl– .049 g/dl	.050 g/dl– .079 g/dl	.080 g/dl– .099 g/dl	.100 g/dl– .149 g/dl	≥.150 g/dl
FSV	Model I	16–20	2.87 [2.04, 4.03]	11.69 [5.3, 25.8]	96.23 [22.13, 418.4]	<i>a</i>	<i>a</i>	<i>a</i>
		21–34	1.74 [1.61, 1.89]	3.66 [3.05, 4.39]	11.13 [7.93, 15.63]			
		≥35	1.67 [1.59, 1.75]	3.32 [2.96, 3.71]	9.27 [7.53, 11.42]			
	Model II	16–21	2.09 [1.85, 2.35]	5.56 [4.2, 7.37]	24.23 [14.39, 40.8]	82.57 [40.13, 169.9]	459.45 [168.64, 1,251.72]	48,476.87 [8,307.37, 282,883.38]
		22–34	1.73 [1.59, 1.88]	3.58 [2.94, 4.36]	10.68 [7.42, 15.37]	26.56 [16.05, 43.97]	95.09 [47.22, 191.49]	3,030.41 [883.9, 10,389.59]
		≥35	1.67 [1.59, 1.75]	3.32 [2.97, 3.71]	9.27 [7.53, 11.42]	21.83 [16.36, 29.14]	72.43 [48.51, 108.14]	1,876.92 [927.01, 3,800.24]
FAV	Model I	16–20	2.66 [1.96, 3.61]	9.78 [4.79, 19.97]	69.03 [18.32, 260.03]	<i>a</i>	<i>a</i>	<i>a</i>
		21–34	1.67 [1.55, 1.8]	3.30 [2.78, 3.92]	9.17 [6.67, 12.62]			
		≥35	1.58 [1.51, 1.66]	2.93 [2.62, 3.28]	7.36 [5.98, 9.06]			
	Model II	16–21	1.98 [1.77, 2.2]	4.90 [3.8, 6.33]	19.17 [11.95, 30.75]	59.69 [31.03, 114.83]	292.78 [118, 726.44]	21,933.34 [4,430.96, 108,569.96]
		22–34	1.66 [1.53, 1.79]	3.24 [2.7, 3.9]	8.90 [6.31, 12.54]	20.62 [12.81, 33.18]	66.89 [34.54, 129.53]	1,631.66 [509.91, 5,221.19]
		≥35	1.58 [1.51, 1.66]	2.93 [2.62, 3.28]	7.36 [5.98, 9.06]	15.85 [11.89, 21.14]	46.43 [31.14, 69.24]	858.21 [424.79, 1,733.89]

Notes: Data come from the 2013–2014 National Roadside Survey and matching FARS databases. Models I and II differ only on how the age groups were constructed. BAC = blood alcohol concentration; FSV = fatalities in single-vehicle crashes; FAV = fatalities in all fatal crashes. *a*Relative risk estimates are not reported due to model instability caused by the relative low numbers of drivers ages 16–20 at BAC ≥ .08 g/dl who participated in the 2013–2014 NRS.

Table 2 shows the RR estimates and their respective 95% confidence intervals (CIs) for FSV and FAV as a function of age and BAC for 2013–2014. For each BAC class, the numbers correspond to the estimates of the RR for the midpoint of the BAC class and a given age group relative to sober drivers (BAC = .00 g/dl) in the same age group. As expected, Table 2 shows that in 2013–2014, the RR of being fatally injured in a crash increased considerably as the BAC increased, with this pattern being more remarkable among fatalities involving the youngest drivers and in FSV crashes. Unexpectedly, however, Table 2 shows a significant increase in RR for drivers ages 35 and older at low but positive BACs (.00 g/dl < BAC < .02 g/dl) compared with a BAC = .00 g/dl, both in FSV and FAV crashes. This finding contradicts previous reports based on the 2007 matching of the NRS and FARS databases, even by this research team (Voas et al., 2012; Zador et al., 2000).

Discussion

A point of interest coming from this study is that between 2007 and 2013–2014, there was a reduction in crash risk among sober (BAC = .00 g/dl) drivers of all ages, teenagers in particular. This finding is encouraging, as it reverses our previous estimates showing that in 2007 and compared with 1996, young sober drivers were at an increased risk of dying

in a crash. Although the reasons for such a decline could not be examined by this study, we speculate that it might relate to improvements in vehicle safety technologies over the last decade (Jermakian et al., 2017).

In contrast with this decline, the contribution of alcohol to the risk of fatal crash involvement did not change significantly over the 17-year period covered by the three NRS–FARS risk studies. The consistency of the RR estimates over the last two decades suggests that whatever caused a decrease in crash risk among sober (BAC = .00 g/dl) drivers failed to induce a parallel reduction in alcohol-related crash risk. Drinking and driving seems to remain a deadly behavior that may be resilient to advances in vehicle safety and road designs.

Researchers have posited several ideas to resume more substantial reductions to alcohol-related crashes like those experienced between 1982 and 1995, including strengthening the use of high-visibility enforcement methods (Voas & Fell, 2013) and lowering the legal BAC limit to .05 g/dl (Fell & Voas, 2014), as well as widening the use of vehicle alcohol interlocks as a sanction for impaired driving (e.g., Kaufman & Wiebe, 2016; McGinty et al., 2017).

Also intriguing is the significant increase in RR for drivers ages 35 and older at .00 g/dl < BAC < .02 g/dl compared with that at BAC = .00 g/dl, a finding that contradicts previous reports showing no such increase (Voas et al., 2012;

Zador et al., 2000). The reasons for such an increase in risk at low BAC levels are unclear and may be related to specific changes over time in the matching NRS and FARS databases, as well as the recent development of additional sources of risk for older drivers (e.g., opioids) that may interact negatively with alcohol even at low doses that were unaccounted for in this study (e.g., Han et al., 2017; West et al., 2015; Wu & Blazer, 2011). The wide range of ages included in the “35+” group adds to this concern, for adults of different ages within this group tend to vary in the drugs they use/abuse the most.

This study has a number of limitations. By failing to account for the impact of drugs other than alcohol on crash risk, our RR estimates may be biased. Although the sampling procedures of the NRS studies were standardized insofar as possible to maintain continuity with previous national roadside surveys (Berning et al., 2015; Compton & Berning, 2009; Lacey et al., 2009a), some inevitable variations in collection procedures may have contributed to error in the comparison of the RR levels between 2007 and 2013–2014. Also, the detection of factors significantly associated with fatal crashes is complicated, as the standard errors tend to be large because of the different sources of error and uncertainty. It is possible, therefore, that standard errors associated with our estimates are also large, which may have led to the declaration of differences between groups as nonsignificant, when perhaps such differences exist. It is also possible that the economic recession that affected our country around the year 2007 may have had an impact on travel exposure, subsequently affecting some of our results. Despite its limitations, this study points to the need to find mechanisms to resume progress in abating alcohol-related deaths.

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