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Darren Grant

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# THE DYNAMICS OF DRINKING AND DRIVING IN THE U.S.: THE ROLE OF SOCIAL FORCES AND THE ROLE OF LAW*

Darren Grant
Department of Economics and International Business
Sam Houston State University
Huntsville, TX 77341-2118
dgrant@shsu.edu

Abstract: Among U.S. drivers involved in fatal accidents, blood alcohol concentration conditional on drinking is static. Thus the dynamics of drunk driving can be described using the fraction of accident-involved drivers who have been drinking (HBD). Changes in this variable imply significant reductions in vehicle fatalities between 1975 and 2004, and correlate mildly with the underlying "general risk factor" that accounts for the remaining reduction in fatalities. But seven important drunk driving laws influence HBD only weakly, and together explain only one-sixth of its reduction over this period. The estimated effect of social forces over twice as large.

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Keywords: drunk driving; traffic safety legislation; panel data analysis

*** This paper has several pages of figures that are best viewed in color. ***

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This manuscript is linked to two companion papers, each part of a series on the economics, political economy, and statistical analysis of drunk driving legislation. Grant (2010b), which motivates Section V, documents how academic findings on the effects of three major laws have become much less favorable over time, and explains this evolution in terms of changes in study design and an "early adopter effect." Grant (2010c) explores how the federal government assesses the efficacy of drunk driving laws through an extended narrative of the process surrounding the passage of the National Minimum Drinking Age Act. Its findings on the political economy of deterrence-based drunk driving countermeasures complement the empirical findings in Section VI. A fourth paper, Grant (2010d), completes the series.

Drunk driving countermeasures try to reduce traffic accidents by reducing drunk driving. A thorough understanding of their effects would therefore require an understanding of two causal links: that between countermeasures and the distribution of blood alcohol concentration (BAC) among drivers, and that between BAC and crash probabilities. The latter is chiefly an epidemiological question, to which we have a reasonably satisfactory answer (Blomberg et al., 2005, 2009). The former is a question for policy analysts, and we have virtually no answer to it at all. It has been addressed by only a handful of studies, discussed below, which focus solely on the incidence of drinking and use inferior methods that are generally eschewed by economists.

This knowledge would have two practical uses. First, it would provide an alternate method of assessing the effect of drunk driving laws on traffic safety. Such a mechanism is vitally needed. Most studies relate laws directly to outcomes, such as fatalities, without using BAC or the incidence of drunk driving as mediating factors. Generally, however, these estimates are highly variable across studies, with the greatest variability (and some bias) occurring in those early studies that have the greatest influence on policymakers (Grant, 2010b). Because these mediating factors are far less variable than crashes are, and are subject to fewer influences (see below), analyzing them instead could lead to improved estimates that are less affected by these problems.

Second, this knowledge would help us better understand the relative importance of law and "social forces" (such as media campaigns, public awareness of the risks of drunk driving, and safety attitudes) in reducing drunk driving over the decades. While laws, demographics, social forces, road quality, and automobile safety technology all affect crash incidence, only the first three of those should substantively affect drunk driving. If we can approximate the contribution of laws and demographics, then, the remainder approximates the contribution of social forces.

Among drivers involved in fatal accidents in the United States, blood alcohol concentration

conditional on drinking has remained unchanged for the past generation. Thus the evolution of drunk driving over this period can be described using the fraction of accident-involved drivers who have been drinking. The dynamic properties of this variable suggest that it is, in fact, a superior instrument with which to assess the effects of effectiveness of drunk driving legislation, and panel regressions using this variable imply that the effects of several major drunk driving laws on fatalities are at the low end of the range estimated in the literature. A decomposition that builds on this finding, in turn, suggests that new laws have affected drunk driving far less than social forces have.

## I. The Basic Dynamics of Drunk Driving.

We begin by documenting the basic dynamics of drunk driving and traffic fatalities. Ideally, this would have been done at the very inception of the literature, to understand the properties of the dependent variable(s) one seeks to explain, but this was not possible because of data limitations. This understanding proves useful in several ways, identifying the drunk driving statistic on which to focus our investigation, delineating the temporal and spatial scales over which it evolves, and revealing sources of bias and inefficiency in standard empirical analyses.

To do this we rely, by necessity, on the only long, nationwide panel of traffic outcomes available: the Fatality Analysis Reporting System (FARS) of the National Highway Traffic Safety Administration (NHTSA). This records accident, vehicle, and driver characteristics for all fatal traffic accidents on public highways since 1975. BAC is directly reported in a fraction of all accidents and imputed for the others, most of which involve nondrinkers. Imputations are present only from 1982 onward, and our data end in 2004, leaving a total span of 30 years, 23 of which have reported or

imputed BAC for each involved driver. National statistics are reported for all fifty states plus the District of Columbia; following the precedent in many recent studies (e.g., Dee, 1999; Freeman, 2007), regression analyses are conducted on 48 states, omitting Alaska, Hawaii, and the District of Columbia. While the FARS data are not a sample, for convenience we use the term "sample period" and refer to the random variation inherent in any probabilistic process, such as traffic fatalities, as "sampling error." The underlying fatality risk at any point in time and space is only imperfectly revealed by the observed fatality rate, because, fortunately, fatal accidents are infrequent.

These data have two major limitations. First, they contain only those accidents that involve a fatality. Accidents involving drinkers are somewhat more likely to be fatal (Blincoe et al., 2002; Grant, 2010d). Still, our empirical analysis is unaffected as long as this relation remains constant over time–if BAC does not have sizeable complementarities with road quality and automobile technology in the "production of accident severity." There little reason to suspect such a complementarity. Furthermore, while only about 1% of all accidents are fatal, these generate half of all accident-related economic costs (Blincoe et al., 2002).

The other limitation is presence of imputed BACs. Using these imputations, as we do, limits the sample period and could affect estimates of state laws' effects, as imputations are not conditioned by state. This should not be a major problem, because few *drinkers'* BACs are imputed and because the strongest predictors of driver BAC are accident-specific factors such as driver age, passenger BAC, and police reported drinking involvement. Still, to provide a robustness check and extend the sample period, we also present some results using just unimputed data.[1] Neither limitation has

[1] Two caveats should also be reported. First, NHTSA's imputation system provides ten imputed values for every non-reported BAC. The first value is utilized in the results reported in this paper; the use of the others yields similar findings. Second, while BAC reporting has increased

prevented NHTSA from using these data to analyze the effects of laws on drinking incidence, most recently by Dang (2008), whose work we extend in the final section of this paper.

We begin by examining the evolution of BAC conditional on drinking (BAC > 0). This, astonishingly, is static. Over the past generation, people have changed *whether* they drink and drive, but not the amount they drink *when* they drive. Figure 1 documents the 25th, 50th, and 75th percentiles of BAC conditional on driving after drinking for all drivers involved in fatal accidents in the U.S. between 1975 and 2004. The BAC distribution is essentially normal, with a mean of about 0.16, a standard deviation of about 0.08, and an interquartile range of about 0.05, whether the imputed BACs are included or excluded. Crucially, in both cases, these numbers are virtually constant across time.

This stability extends to two levels of disaggregation. We calculated BAC (conditional on drinking) at each of these percentiles within state*year cells, and then regressed them on a full set of state and year dummy variables. In these regressions, the standard deviations of the state dummies were only about .008, indicating geographic stability. Furthermore, the standard deviations of BAC at each percentile, calculated across all pooled state*year cells, were between .01 and .015, indicating that temporal stability extends to the state level.

In consequence, the dynamics of drunk driving can be described using a single statistic: the

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gradually in many states over time, several initially low-reporting states dramatically increased reporting in a discrete jump at various years in the 1980s. While any effects of these secular trends in reporting will be adequately captured by the year dummies in the regressions below, the discrete changes are associated with discrete jumps in HBD, and thus can bias estimates of effects of drunk driving legislation. (This occurs, not surprisingly, most strongly with the minimum drinking age, which was changed frequently during this period; this problem biases estimates of its effect toward zero.) Thus, the regressions below omit from the sample those few sample years prior to the jump in reporting in those states. The affected states and the last year of omitted data are as follows: AL, 1982; AR, 1989; FL, 1985; ID, 1984; IN, 1985; IA, 1982; KS, 1987; MS, 1991; MD, 1985; NC, 1982; ND, 1984; TX, 1985.

fraction of accident-involved drivers who have been drinking, or HBD (had been drinking). While only a small fraction of drivers drink, the typical drinking driver is far more risky than a sober driver is. In an exhaustive study that follows and extends decades of epidemiological research, Blomberg et al. (2005, 2009) carefully assess how crash risk is influenced by BAC. Colloquially, this risk doubles with each standard drink beyond two; Grant (2010d) argues that this holds for fatalities as well as crashes, and that these risks, relative to those of sober drivers, have not changed substantially over time. Given the high BACs of accident-involved drinkers, simple calculations show that the average crash risk of drinking drivers is sixteen times that of sober drivers.

In consequence, while drinkers are well-represented among drivers involved in fatal accidents, they form a small percentage of all drivers. This, in turn, implies that a one percent decrease in the number of drinking drivers will lead to a one percent decrease in crashes attributable to drinking drivers, virtually no increase in crashes attributable to sober drivers, and thus a decrease in overall crashes of about HBD percent. This holds true whether the decrease is caused by fewer drivers drinking, or by fewer drinkers driving. By algebraically backing out the change in the number of drinking drivers from changes in HBD, one can calculate the implied the percentage reduction in fatalities. This equals the percentage *increase* in the fraction of drivers who have *not* been drinking.

While BAC conditional on drinking is stable, this is not true for drinking itself. HBD has declined nationwide, and it is instructive to document the systematic, evolutionary way in which it has done so. Figure 2 illustrates using age-HBD profiles at five-year intervals, both including and excluding the imputed data. Including the imputations sharply increases the age-HBD gradient for adults and the absolute magnitude of change but leaves the relative rates of change unaffected. These profiles indicate that HBD fell "from the outside in," initially declining most among the youngest and

oldest drivers, with middle aged drivers catching up a decade later. The largest declines in HBD first occurred among drivers over forty, with HBD falling ten percentage points or more between the late 1970s and the early 1990s. Next came drivers under twenty, where HBD declined about fifteen percentage points between the early 1980s and the mid 1990s. Finally, among middle-aged drivers aged 20-40, HBD fell most rapidly during the 1990s. Drivers in their early thirties, laggards during the previous decades, were the only group reducing HBD as the new century dawned. Using the relation given above, these changes in HBD generated fatality reductions of about 30% for young drivers, 20% for middle-aged drivers, and 10% for older drivers. These account for a large, but not dominant, share of the reduction in fatalities per vehicle mile traveled over this period, which fell by half (NHTSA, 2009).

Changes in HBD are also not uniform geographically. To focus on the age group with the greatest drinking involvement and also reduce variation in driver age and accident type, we analyzed HBD in all single vehicle accidents involving drivers aged 21-40 for the 1982-2004 period. Calculating HBD by state by year (1173 state*year cells), we then regressed these values on state and year fixed effects. The state effects have a standard deviation of 6.9 percentage points, with heavy-drinking Wisconsin at the top and light-drinking Utah at the bottom, thirty percentage points below. Together, they and the year fixed effects explain about two-thirds of the variation in the dependent variable. Still, the residuals, which track within-state variation in HBD that has been purged of national trends, are quite variable, with a standard deviation of 5.7 percentage points.

The properties of this "purged HBD" variable are of great import, as most modern analyses of drunk driving laws, panel regression analyses, include state and year fixed effects as control variables. Therefore we also estimate this variable's serial correlation, of the current value with its

one year lag, and its spatial correlation. This is calculated across matched pairs of states that are geographically and demographically similar, like those used in many case-control studies of drunk driving laws. (Vermont is matched with New Hampshire, for example, and New Mexico with Arizona. The full set of pairs is identified in the note to the table.) The results are shown in the first row of Table 1: the serial correlation and the spatial correlation are both quite small.

Within state*year cells, however, sampling error can contribute substantially to the variation in HBD. That is, HBD can be a relatively inaccurate measure of the underlying rate of drinking and driving in that state in that year, because it is calculated from only those drivers involved in fatal accidents. Because HBD estimates the population proportion of "successes" in a binomial distribution, however, this sampling error can be approximated and extracted, thus allowing the variance of the underlying latent variable to be calculated, along with its serial and spatial correlation.

To do this, let $H$ be HBD observed within a state*year cell, $h$ be the unobservable latent variable or "true HBD" around which $H$ varies, $H^*$ be the HBD predicted from state and year fixed effects alone, $S$ be the number of observations within each state*year cell, and $C$ be the total number of state*year cells. The capitalized variables are observed and the lower case variables are not, a convention maintained throughout. Then, summing across state*year cells:

$$\begin{aligned} \Sigma(H-H^*)^2 = \Sigma(H-h)^2 + \Sigma(h-H^*)^2 &= \Sigma\frac{h\cdot(1-h)}{S} + \Sigma(h-H^*)^2 \\ &\approx \Sigma\frac{H^*\cdot(1-H^*)}{S} + \Sigma(h-H^*)^2 \end{aligned} \qquad \textbf{(1)}$$

where sampling error is approximated by replacing $h$ with its unbiased and reasonably close estimate $H^*$. We wish to know the properties of $h$ - $H^*$, that is, $h$ net of state and year fixed effects. Because this has an expectation of zero, the sample analog of its variance is then:

$$\Sigma \frac{(h - H^*)^2}{C} \approx \Sigma \frac{(H - H^*)^2}{C} - \Sigma \frac{H^* \cdot (1 - H^*)}{C \cdot S} \qquad (2)$$

The extensions to serial and spatial correlation are straightforward.

The results are presented in the second row of Table 1. Sampling error in fact generates most of the variance of HBD at the state*year level; the standard deviation of the underlying latent variable is only about two percentage points. Its spatial correlation is a modest 0.4, but its serial correlation is much higher, about 0.8. Just as it does at the national level, state-level drinking and driving evolves gently and persistently, but at different rates in different states. These inter-state differences might suggest the relevance of states' legal environments, but also could reflect the influence of social forces, which plausibly affect drinking and driving in the systematic, evolutionary manner that we have just documented.

## II. HBD and Fatalities.

The algebraic link between HBD and fatalities noted in the previous section helps us impute the change in fatalities that can be attributed to changes in HBD. But it is also useful to understand the statistical relationship between these two variables, as fatalities are affected by factors other than drunk driving. While are theoretical developments are general, our calculations focus, as before, on values within state*year cells after extracting time-invariant state factors and year fixed effects.

Above we distinguished between observed HBD and the underlying, unobservable latent variable around which these observations varied, and estimated the variance of each. We can do the same for fatalities, which are distributed Poisson around their expected value. Let $F$ be actual

fatalities within each state*year cell, $f$ be the underlying latent variable (expected fatalities), and $F^*$ be the number of fatalities predicted from state and year fixed effects and vehicle miles traveled. The relationship needed to extract sampling error is as follows:

$$\Sigma\left(\frac{F-F^*}{F^*}\right)^2 = \Sigma\left(\frac{F-f}{F^*}\right)^2 + \Sigma\left(\frac{f-F^*}{F^*}\right)^2 = \Sigma\frac{f}{F^{*2}} + \Sigma\left(\frac{f}{F^*}-1\right)^2 \approx \Sigma\frac{1}{F^*} + \Sigma\log^2\left(\frac{f}{F^*}\right) = \Sigma\frac{1}{F^*} + \Sigma(\log(f)-\log(F^*))^2 \quad (3)$$

The results are presented in the next two rows of Table 1. As with HBD, much of this variable's variation is due to sampling error, and once this is removed the spatial correlation of the underlying factor is also about 0.4 and the serial correlation about 0.8.[2]

Given that the covariance between HBD and log fatalities should be unaffected by sampling error, we can calculate both the raw correlation between these two variables and a correlation that is adjusted for sampling error–that is, the correlation of the latent variables. Even after extracting sampling error, this correlation is only 0.32, as seen in the last column of the table. Changes in drinking and driving account for only a modest fraction of the variation in log fatalities. In consequence, regressions using HBD could be quite different from those using fatalities.

The relationship between HBD and fatalities can be better understood by setting out a simple, first-order model for fatalities that is consistent with our previous discussion and with previous work on the subject. In addition to the variables defined previously, add the following:

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[2] These findings call into question a design feature of a number of early traffic safety studies (e.g., Williams et al., 1983; Arnold, 1985) that draw on matched state pairs to infer the effects of law changes. In this design, the paired states are deemed to be identical in their characteristics except for the passage of the law, which is valid only if the adjusted spatial correlation for fatalities is very high.

- $s$ = the miles driven by sober drivers
- $d$ = the miles driven by drinking drivers
- $r$ = the general riskiness of driving, due to weather, road quality, automobile technology, general safety laws, etc.
- $M = s + d$ = miles driven

Also let $K$ represent the average crash risk of drinking drivers relative to sober drivers. Evidence provided previously indicates $K \approx 16$. The latent variable $h = Kd / (s+Kd)$.

Observed fatalities, $F$, are distributed Poisson around their expected value, $f$, a function of the miles driven by sober drivers, $s$, the miles driven by drinking drivers, $d$, and the general risk factor, $r$, as follows: $f = (s + Kd)*r$. In this equation a ten percent reduction in the general risk factor leads to a ten percent reduction in fatalities among both sober and drinking drivers, while a ten percent reduction in the number of drinking drivers will lead to a ten percent reduction in fatalities involving those drivers and a 10*HBD percent reduction in overall fatalities, as in Section I. One can think of reasons why these statements might not be strictly true, but they are likely to be second-order.[3]

Algebra yields:

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[3] Behaviorally, the compensating variation hypothesis suggests that sober drivers would be less careful if they knew that there were fewer drunk drivers on the road. This hypothesis would suggest that the HBD regressions below, which find mild effects, are biased *away* from zero. If dominant, it would also imply a negative correlation between drinking incidence and the general risk factor, instead of the positive correlation that obtains, as shown shortly.

Alternatively, sober and drinking drivers may have different risk factors. Even then an HBD analysis is probably preferred to a fatality analysis. To see this, consider the expression:

$$\frac{f_{DRINKING}}{f_{SOBER}} \approx \log(K) + \log(M) - \log(d) + \log(r_{DRINKING}) - \log(r_{SOBER}) \approx \log(\frac{h}{1-h})$$

If the two risk factors are positively correlated regression error is reduced, as is bias if the common risk component is correlated with $d$, as empirical estimates presented shortly will indicate.

The dependent variable in most previous, related work in the literature is the empirical analog of the leftmost expression in the equation above, which, as shown, is a simple transformation of H.

$$f = M \cdot r \cdot [1 + \frac{h}{(\frac{K}{K-1})(1-h)}] \approx M \cdot r \cdot [1 + h/(1-h)] = M \cdot r/(1-h) \quad \textbf{(4)}$$

where the approximation is very close whenever $K >> 1$, as it is here. Then:

$$\log(f/M) \approx \log(r) - \log(1-h) \quad \textbf{(5)}$$

so that expected per mile fatalities are directly proportional to the general risk factor and, as in Section I, inversely proportional to the fraction of crash-involved drivers who have not been drinking.

We have already identified the variation in log fatalities that is attributable to sampling error. With this new relationship we can now break down the remaining variation into components associated with the latent variable log($r$), the latent variable log(1-$h$), and their interaction, and use the method of moments to identify each from the data.[4] The results of this exercise are found in the

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[4] Define $n = 1 - h$, and $N$ and $N^*$ accordingly. Then, the key relationships are as follows:

$$\Sigma(\frac{N-N^*}{N^*})^2 = \Sigma(\frac{N-n}{N^*})^2 + \Sigma(\frac{n-N^*}{N^*})^2 = \Sigma\frac{n\cdot(1-n)}{S\cdot N^{*2}} + \Sigma(\frac{n}{N^*} - 1)^2$$
$$\approx \Sigma\frac{1-N^*}{S\cdot N^*} + \Sigma(\log(n) - \log(N^*))^2$$

$$cov(\log(F/M),\log(N)) = cov(\log(r) + \log(n),\log(n)) = cov(\log(r),\log(n)) + var(\log(n))$$

$$\Sigma(\frac{F-F^*}{F^*})^2 - \Sigma\frac{1}{F^*} = \Sigma(\log(f) - \log(F^*))^2 = \Sigma(\log\frac{r/n}{r^*/N^*})^2$$
$$= \Sigma([\log(r) - \log(r^*)] - [\log(n) - \log(N^*)])^2$$
$$= C\cdot[var(\log(r)) + var(\log(n)) + 2cov(\log(r),\log(n))]$$

The first relationship identifies the variance of log($n$), the second the covariance of log($r$) and log($n$), and the third the variance of log($r$).

final two rows of Table 1. The variation in log fatalities that is not attributable to state fixed effects, year fixed effects, miles driven, and sampling error has a standard deviation of 8.1 percentage points. Each of the components listed above contributes about 1/3 of this variation. The interaction component is generated by a weak positive correlation between drinking and the general risk factor ($\rho = 0.20$), which suggests either that drinking sentiment and general "driving safety sentiment" move together, or that new drunk driving laws are associated with other traffic safety initiatives, such as an increased police presence. A practical implication is that fatality regressions may yield "effect sizes" (the estimated effect of laws) that are both biased and more variable than those in regressions analyzing HBD, unless these risk factors can be directly observed and controlled for.

## III. Legislation and HBD: Estimation.

Using the relationships derived above, one can estimate the effect of laws (or other factors) on HBD and then impute the implied reduction in fatalities caused by the attendant reduction in drinking. Following the literature, many of our regressions take a natural, panel analysis form:

$$Y_{s,t} = \beta S_{s,t} + \gamma L_{s,t} + \sigma_s + \tau_t + \epsilon_{s,t} \qquad \textbf{(6)}$$

with $s$ indexing states and $t$ indexing time; ß the coefficients on statewide, time-varying controls $S$; $\gamma$ the coefficients on the law dummies, $L$;[5] $\sigma$ the state fixed effects; and $\tau$ the time fixed effects.

For the dependent variable, $Y$, there are three options, which are not all equally good. The

[5] The coding of all laws is taken from the *Digest of State Alcohol-Highway Safety Related Legislation,* supplemented occasionally with Dang (2008) or Grant (2010a).

first, suggested by equation **(5)**, is to use the log of the fraction of crash-involved drivers who have not been drinking in that state in that year, log(1-*H*). Then $\gamma$ is interpreted as the reduction in log fatalities implied by the reduction in drinking and driving attributed to law L. If the number of fatal crashes within any given state*year cell is sufficiently small, however, *H* can deviate substantially from its expected value, *h*, due to sampling error. Then it easy to show that $E(\log(1-H)) \neq \log(1-h)$, that this difference is larger when *h* is larger, and (numerically) that this biases regression coefficients in a favorable direction. This problem is severe in panel analyses of crashes for a small age range, such as young drivers, because there are few such fatalities in less-populated states.

A solution is to use the second option: employ *H* as the dependent variable, so sampling error causes no bias, and then calculate the implied change in log(1-*h*) from the estimate of $\gamma$. Because variation in *h* around its mean is not large, a Taylor series approximation is feasible. Replacing log(1-*h*) with its first-order approximation around the grand mean of the sample, $H_M$, yields:

$$\log(1-h) \approx \log(1-H_M) - \frac{h-H_M}{1-H_M} = \log(1-H_M) - \frac{H-H_M}{1-H_M} + \nu \qquad (7)$$

The expected increase in log(1-*h*) is closely approximated by the decrease in *H* divided by (1- $H_M$). That is, it suffices to use HBD directly as the regressand in the regressions, and then scale the coefficient estimate by -1/1-$H_M$ to infer the effect on log fatalities. Both the effect of the law on HBD and its implied effect on fatalities are reported. (A slightly more accurate approximation can be calculated using individual state means rather than $H_M$.) Estimation is conducted using least squares, weighted by the number of accident-involved drivers in each state*year cell.

The final option tailors the specification for use with individual microdata. The probability that an accident-involved driver has been drinking is estimated with a logit model that includes not

just the state-level variables listed above but also individual demographic and accident-specific information (see equation **(9)** below). The average marginal effect, calculated numerically, indicates how the law affects HBD, from which the implied effect on fatalities is calculated as above.

In the panel analyses, the vector of time-varying state covariates $X$ can be smaller than in fatality regressions, because many of their controls plausibly affect fatalities but not drinking. Economic factors, which are known to affect the drinking incidence, should be included, along with alcohol prices or consumption. Our estimations also focus on single vehicle accidents, to which the model in Section II best applies, using multiple-vehicle accidents as a robustness check. This is not greatly limiting: many studies in the traffic safety literature have focused on single vehicle accidents, in which responsibility for the accident is naturally attributed to the driver.

## IV. Estimation Results.

To compare how HBD regressions of laws' effects compare with fatality regressions, we initially focus on the three most studied drunk driving laws in the literature, with one hundred published academic studies between them (Grant, 2010b): a minimum legal drinking age (MLDA) of twenty-one, zero tolerance (ZT) laws that lower the per se illegal BAC for youth to .01 or .02, and laws that lower the per se illegal BAC for adults to .08. These are now universally enacted within the United States, mostly during our sample period and partly due to Congressional pressure (the threatened loss of highway funds).

In the panel regressions, each law variable is set to zero if the law was not in effect in that state in that year, to one if the law covered the entire intended population in that state in that year,

and to a fraction less than one if the law covered only part of the relevant population or was in effect only part of the year.[6] In the logit models, in contrast, we can specifically determine whether that driver was covered by the law in question and then assign a value of zero or one. All regressions include controls for the annual, state unemployment rate and dummies for .10 per se laws and administrative license revocation (ALR) laws that allow the state to suspend or revoke an individual's license immediately upon testing positive for drunk driving or refusing to be tested. These are not the only viable controls, just those that plausibly influence drunk driving and regularly appear in the large panel fatality studies in the literature. (Expanded specifications are used in Section VI.)

Estimates for Adults. We first conduct estimations for "adults" aged 21-60, for which all three estimation methods are acceptable, and for which we can ignore the laws that pertain to "youth" aged 18-20. These are presented in Table 2.

We begin with the basic specification in the fourth row of the table, which uses HBD directly as the dependent variable and then present the implied reduction in fatalities, the coefficient divided by 1-$H_0$, in brackets. For the per se laws, the .10 coefficient estimate is insignificant but the .08 estimate implies a sizeable 2.5 percentage point reduction in HBD and a concomitant five percent reduction in fatalities. ALR reduces HBD by one percentage point and fatalities by two percent.

Moving upward from this row of the table we encounter alternative specifications. In the next row up, the specification uses -log(1-$H$) as the dependent variable. The coefficient on this variable, multiplied by one hundred, gives the implied percentage reduction in fatalities yielded by reductions

[6] The value of the dummy equals the fraction of the relevant population covered by the law during that state in that year. For MLDA laws, then, this value was determined by multiplying the fraction of 18-20 year olds covered by the law by the fraction of the year the law was in effect.

in drinking and driving. These estimates tend to be slightly more negative than those in the HBD regressions, which is to be expected because of the bias discussed above. Finally, the top two rows use logit models. The first, in the top row of the table, includes just the controls used in the other regressions along with driver age, while the model in the second row also includes driver gender and sets of dummy variables for the hour, day, and month of the accident. After adding these controls, the estimates become less favorable (more positive), falling about one-third for both .08 and ALR.

Below the basic HBD regression in the fourth row of the table are found robustness checks using alternative sets of accidents and using only the unimputed data. The gross effect of .08 laws on fatalities (relative to no per se law) is a little smaller than before, two to three percent, but its significance and that of ALR is unaffected.

Overall these findings, while confirming the value of .08 per se laws and ALR, are less favorable than in the literature as a whole. The median estimate of .08 laws' effects on fatalities in Grant's (2010b) review is five percent, slightly higher than the average estimated effect in Table 2, and most of these studies find that a .10 (or higher) per se law also reduces fatalities substantially, while here it has no estimated effect. The estimated effects of ALR, a one or two percent reduction in fatalities, are also far lower than in most studies (see the review in McArthur and Kraus, 1999).

Estimates for Youth. Table 3 presents estimates of the effects of MLDA and ZT laws for both youth and "young adults" aged 21-25, a natural control group. These law variables are added to those used in the previous regressions; thus ALR, .08, and .10 law dummies are retained as controls.

We initially focus on the MLDA coefficients in the basic HBD regressions, which are in the first three rows of this table. These estimates are consistently positive and generally significant,

suggesting that making more people eligible to drink increases HBD. Thus, raising the MLDA from 18 to 21 reduces HBD by three or four percentage points and brings fatalities down by about seven percent. In contrast, the coefficients for the young adult control group, while also positive, are smaller and insignificant.

The next three rows of the table utilize only the unimputed data. As before, these results support the findings we have just reported.

Finally, in the bottom two rows of the table, we run individual-level logit models. In addition to facilitating the inclusion of demographic and accident controls, these regressions allow the values of the two law variables to be precisely determined using driver age and accident year and month, as described above. These MLDA coefficient estimates imply even smaller effects on fatalities.

On the whole, these results are at the low end of the range of estimates produced in the literature. Grant (2010b) surveys all studies of minimum drinking age (MLDA) laws published by 2009; effect estimates are widely dispersed and often large, with predicted reductions in fatalities generally exceeding ten percentage points. Many of these studies, however, include cross-sectional variation or analyze just a few states; effect sizes tend to be lower when a nationwide panel analysis is conducted. The one panel study of *reductions* in the MLDA, Cook and Tauchen (1984), estimates a 7% effect on fatalities; the largest-scale studies of MLDA *increases*, Polnicki, Gruenwald, and LaScala (2007) and Miron and Telebaum (2009), get estimates of 5-10%. Our findings suggest even these estimates are too high, maybe because of the correlation between HBD and the general risk factor that was uncovered in our decompositions.

Our results are also smaller than those of the four other multi-state studies that examine the effect of the MLDA on HBD (or its transformation) using the FARS data: Robertson (1989), Voas,

Tippetts, and Fell (2003), Fell et al. (2008), and Dang (2008).[7] These studies find that the MLDA decreases fatalities by at least 11% (and similarly large effects for the other laws included in those studies). But each of them omits state fixed effects, allowing cross sectional variation to influence the coefficient estimate. In our models these state fixed effects are highly significant, and their omission affects coefficient estimates substantially, just as it often does in fatality regressions. Therefore, the large effects estimated in these studies are probably due to omitted variable bias.

Regarding zero tolerance laws, the findings in Table 2 are even weaker. Across the full set of regressions the coefficient estimates, while negative, are insignificant, and nearly matched by those in the control group. This suggests zero tolerance laws do not materially affect HBD or fatalities. This too contrasts with most estimates reported in the literature (Grant, 2010b), but these often analyze a small number of states or allow cross-sectional variation. Later, more comprehensive panel studies tend to be less favorable, and the most recent of these find no effect whatsoever (Dee, Grabowski, and Morrisey, 2005; Grant, 2010a). The latter study reveals the existence of unmeasured confounders that were not accounted for in earlier work, which lead to equivalent effect sizes in the target group and multiple control groups–the kind of correlation suggested by the decomposition in Section II. This study also finds no substantial effect of ZT laws on HBD among youth or control groups, using a shorter sample period that brackets the years over which these laws were adopted.

Overall, then, our estimates for MLDA and ZT laws are far smaller than in the weaker studies in those literatures and somewhat smaller than those of the stronger studies (large panel analyses). Estimating the effects of drunk driving laws on fatalities via their effects on HBD leads to relatively

[7] Hingson, Heeren, and Winter (1996) find more modest effects in a before-after study of the effects of .08 laws. These are the only published, multi-state studies that relate laws to drinking incidence in traffic fatalities.

conservative estimates of their effectiveness. It may be argued that two of these laws, .08 and ZT, which lower allowable BAC limits, should not affect HBD but instead affect BAC conditional on drinking and driving. For ZT laws, however, Grant (2010a) has examined this contention specifically and found no evidence to support it. For .08 laws, the number of fatal traffic crashes involving affected drivers (with BACs of .08 or .09) is less than three percent, suggesting that even with nearly full compliance the effect on fatalities would not exceed a couple of percentage points, which is less than the .08 law-.10 law difference implied by the Table 2 regressions. Section I offers an explanation for these laws' impotence: drinkers seem to adjust whether they drive, but drivers do not adjust how much they drink, which is what these laws are asking them to do.

## V. HBD Effects and "Early Adopters."

While a retrospective evaluation of the effects of these three laws is valuable, its practical import is somewhat limited because they are now long-established. The diffusion of these laws throughout the country is most strongly impacted by studies of the experience of those states that adopt the law first. Carefully reviewing the academic literatures on MLDA, ZT, and .08 laws, Grant (2010b) finds that all three follow a familiar pattern: large and variable estimates of the law's effect in those early studies that were available at the time of Congressional action, converging to smaller, less variable estimates decades later, after more data has been collected and improved analytical techniques employed.

Furthermore, following Miron and Telebaum (2009), Grant shows that conditional on estimator, specification, and data source, in all three literatures the law has larger estimated effects

in early-adopting states, which he calls an "early adopter effect." Thus, even with the best data and techniques available, assessments of laws' effects in early-adopting states are likely to overpredict their effects in later-adopting states. A possible explanation for this early-adopter effect is the existence of other law changes, enforcement initiatives, or changes in safety attitudes that accompany the new law in question. These are more likely to be found in those states that combat drunk driving aggressively, and less likely to be found in states incentivized to adopt these laws by the threatened loss of federal highway funds. These factors should also affect fatalities more than HBD, and thus should bias fatality regressions more strongly than HBD regressions. Thus, the techniques introduced here may be especially valuable for uncovering the effects of drunk driving legislation in early-adopting states, and thus for contemporaneous, rather than retrospective, policy analysis.

To find out whether HBD regressions also exhibit an early-adopter effect, we must estimate the effect of the law in question in each state using a constant estimation method. A simple and revealing way to do this, following Grant (2010b), is to estimate the following regression:

$$H_{s,t} = \beta S_{s,t} + \gamma_s L_{s,t} + \sigma_s + \tau_t + \epsilon_{s,t} \quad \textbf{(8)}$$

Unlike its predecessor, this regression estimates a separate coefficient for each law-adopting state in the data, and its standard error. To determine the cumulative estimated effect based on all states adopting the law up to date T, we calculate the weighted average of all of those states' $\gamma$ estimates, using the inverses of the squares of the standard errors as weights, and calculate the standard error of this average accordingly. We calculate this cumulative estimate at every date (month and year) in which a new state passed the law in question, and graph the results on a cumulative "stock plot" that has time on the horizontal axis and the cumulative effect size on the vertical.

For fatalities, Grant (2010b) performed such an analysis, quasi-replicating three recent, comprehensive studies of each laws' effects: Miron and Tetelbaum (2009) for MLDA laws, Grant (2010a) for ZT laws, and Freeman (2007) for .08 laws. Each uses the standard state*year panel regression design; each contains numerous supplementary tests that strengthen its conclusions; each takes care to replicate others' findings and explain any differences from those findings; and each finds a smaller effect of the law than is typical in the literature.

These stock plots are presented in the left panes of Figure 3-5 for MLDA laws, ZT laws, and .08 laws respectively.[8] The right pane of each figure presents the stock plot calculated here for HBD. Crucially, the axis has been scaled in the graph on the right by $(1-H_0)$, so that an equal vertical distance on the two graphs implies an equal change in fatalities. A cubic trend, which fits the data reasonably well, is included. The leftmost point on each graph includes just the coefficient estimate from the first state to adopt the law in question over the sample period; the rightmost contains the cumulative, or "final," estimate from all states adopting the law in question over the sample period. Confidence intervals extending two standard deviations from the mean are depicted in the vertical lines that surround the dot that represents the cumulative effect size.

In the left panes of Figures 3-5, the cumulative stock plots for fatality regressions, a large, consistent upward trend, toward zero, is clearly visible. Effect sizes in early-adopting states are more than double, and at least five percentage points, higher than the final estimate. This is the early-adopter effect.

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[8] A few states passed early "partial" zero tolerance laws that affected only some youth drivers or that set a BAC limit above .02. These are rarely included in recent studies of ZT laws but did receive some early analysis, and are included here because of the focus on early-adopters. In Figure 4, separate estimates for "partial laws" and "full laws" are included for those states that adopted both.

The right panes of Figures 3-5 are different. While early estimates of each law are naturally quite variable, they are not always strongly favorable. More importantly, in each case the cumulative estimate closely approaches the final estimate before half of the sample period has passed, by which time only about one-quarter of the states have passed the law in question. This convergence is much more rapid than is observed for the fatality estimates. Standard errors at each point in time are similar between the fatality and HBD estimates.[9] Therefore, HBD regressions better inform policymakers about the experience of drunk driving laws in early-adopting states–though great caution should be used drawing strong conclusions from the experiences of a small number of states.

## VI. The Role of Social Forces and the Role of Law.

Reductions in drunk driving have contributed significantly to the decline in per-mile crash fatality rates in the U.S. over the past generation. These reductions could come from three sources: legal incentives, demographics, and general social forces such as moral suasion and public information about the dangers of drunk driving. The first two factors are observable but the third is not. Instead, its effect must be inferred, as the residual after accounting for the effects of demographics and laws.

We can do this by conducting estimates on microdata that includes individual driver demographics. Let $\eta$ be a dummy variable indicating whether a particular driver is coded as having a positive BAC. Expand the vector L to include indicator variables for seven key drunk driving laws,

[9] The standard errors should be smaller in the HBD regressions, but this is obscured because these regressions omit years 1975-1981 from their sample. One can show analytically that sampling error is always larger in the fatality regressions, and if important general risk controls are omitted, these standard errors will be even larger still.

described below. Define D as a vector of accident (hour, day of the week, and month of the accident) and demographic variables (driver age, gender, and state), S as two "state factors," the state unemployment rate and per capita alcohol consumption, and Λ as the logistic function. A standard fixed effects specification relating these variables is as follows:

$$P(\eta_{i,s,t} = 1) = \Lambda(\gamma L_{i,s,t} + \phi D_{i,s,t} + \beta S_{i,s,t} + \tau_t) \quad \textbf{(9)}$$

where i indexes individuals and ϕ is a vector of coefficients. This is estimated on all drivers aged 15-60 involved in fatal single-vehicle accidents.

In addition to the five laws examined in Section IV, the laws vector, L, includes open container laws and dram shop laws, based on empirics supporting their effectiveness in comprehensive panel traffic safety studies (Ruhm, 1995; Eisenberg, 2003; Whetten-Goldstein et al, 2000; Benson and Rasmussen, 1999). The *practical* case for legislative drunk-driving countermeasures rests squarely on these seven laws, as no others have received appreciable support in the academic literature, strong financial incentives from Congress, or emphasis from NHTSA. The demographics vector, D, now subsumes the state fixed effects expressly written out before. The alcohol consumption measure included in S could be influenced by economics, social forces, or laws, and the statistical analysis will indicate the relative importance of each. This regression is estimated single-vehicle accidents involving drivers aged 15-60 over the period 1986-2004. Several states have poor BAC reporting prior to 1986, but from this year forward reporting is reasonably high and reasonably steady, ranging from 56%-59.5% in each year.

Now, define t=0 as a base year, and consider the following four equations:

$$H_t = \bar{\eta}_t = \overline{\hat{\eta}_t} = \overline{\Lambda(\phi D_{i,s,t} + \gamma L_{i,s,t} + \psi S_{s,t} + \tau_t)} \quad \textbf{(10)}$$

$$E(H_t \mid L = L_0) = \overline{\Lambda(\phi D_{i,s,t} + \gamma L_{i,s,0} + \psi S_{s,t} + \tau_t)} \quad \textbf{(11)}$$

$$E(H_t \mid L = L_0, S = S_0) = \overline{\Lambda(\phi D_{i,s,t} + \gamma L_{i,s,0} + \psi S_{s,0} + \tau_t)} \quad \textbf{(12)}$$

$$E(H_t \mid L = L_0, S = S_0, t = 0) = \overline{\Lambda(\phi D_{i,s,t} + \gamma L_{i,s,0} + \psi S_{s,0} + \tau_0)} \quad \textbf{(13)}$$

The difference between HBD in any given year, $H_t$, and HBD in the base year, $H_0$, can be broken down into four components: laws, the difference between the first two equations; state factors, the difference between the next two equations; social forces and other residual factors, the difference between the next two equations; and demographics, the difference between equation **(13)** and $H_0$. These components are calculated for each year of the sample, using 2004 as the base year, and graphed in Figure 6.

Figure 6 depicts a drop in HBD of twelve percentage points, corresponding to a one-quarter decrease in fatalities, over the period 1986-1997, after which is stasis (HBD for all accidents behaves similarly). Concomitantly, the contribution of each factor is largest in 1986 and falls more or less steadily thereafter. The effect of laws, a total reduction of two percentage points in HBD (and therefore 4% in fatalities), is equal to the contribution of state factors and somewhat smaller than the contributions of demographics (three percentage points) and social forces (four percentage points).

The interpretation of the state factors component can be clarified by additional regressions whose results we now report. First, it simply reflects changes in per-capita alcohol consumption (and is labeled as such in the figure), because the coefficient on unemployment disappears after this is controlled for. Second, the component's downward trend is not explained by the demographic

(Greenfield, Midanik, and Rogers, 2000) and law variables in our regressions, nor by prices, which closely tracked inflation throughout the period. Thus, the decline in per capita alcohol consumption is itself best attributed to social forces. By this measure social forces, which have both reduced alcohol consumption and drunk driving conditional on alcohol consumption, are more than twice as important in reducing drunk driving fatalities as are seven major drunk driving laws.

We acknowledge the inherent imprecision in inferring the effect of social forces indirectly, as a residual, when it is not feasible to control for every extant drunk driving law. To the extent that important laws are excluded, the estimated effect of social forces is overstated. But we also believe that, heretofore, the excess has run in the other direction. Because the effect of social forces can be imputed only as the "remainder" after accounting for the effects of laws (and demographics), the larger those effects are determined to be, the smaller must be the effects of social forces. Accordingly, if the effects of law have been overstated, the effects of social forces have been correspondingly understated, or "crowded out." This study and the evidence in a companion paper, Grant (2010b), suggest that this is frequently the case. It is not unusual for laws' estimated effects to exceed the entire change in fatalities that can be attributed to reductions in HBD in the appropriate age range over the appropriate time period. Yet this disconnect has drawn no attention whatsoever.

The most striking instance of this disconnect is found in Voas, Tippetts, and Fell (2003), a frequently-cited, pooled time-series cross-section regression analysis of the effect of several laws on drunk driving fatalities among drivers under 21. Among this group, they find crashes are reduced 19% by the raised MLDA, 24% by ZT laws, 18% by .10 per se laws, and 19% by administrative license revocation (Table 3). The combined effect is to lower crashes by 59%, twice the amount implied by the raw reduction in HBD among this group (see Section I and Figure 2). These effects

are described as "modest" and "generally consistent with the results of other studies" (p. 585).

But this phenomenon is far more pervasive that this one study. Consider, for example, zero tolerance laws, universally adopted in the U.S., mostly during the mid-1990s. As Grant (2010a, 2010b) shows, many studies found that these laws reduced fatal accidents by fifteen percent or more, though the implied fatality reduction from reduced drinking among the affected age group between the early 1990s and the early 2000s is only about ten percent (see Section I and Figure 2). Similarly, .08 laws, universally adopted by all states, mostly around the turn of the century, are often found to have double-digit (percentage) effects on fatalities, though the aggregate reduction in fatalities attributable to general reductions in HBD among the affected age group (twenty-one and older, as all drivers under twenty were subject to zero tolerance laws) between the mid-1990s and the mid-2000s was no more than five percent (again, see Figure 2), and though fewer than three percent of all fatalities involved drinkers in the BAC range directly affected by the law, as mentioned above.

If the effects of drunk driving legislation have historically been overstated, and the importance of social forces understated, our efforts to combat drunk driving are, to some degree, misdirected. This possibility could be more easily disregarded if HBD had not remained flat since 1997, despite increasing numbers of legal disincentives.

One corollary issue that defies quantification, and hence a simple resolution, is the relationship–or, better put, relationships–between laws and social forces. The two variables are associated, mutually causal, and complementary (see Grant, 2010c, which elaborates on the following points at length). They are associated because both social forces and laws can be fostered by changes in the political environment and in scientific knowledge, as in the early 1980s. They are mutually causal because changes in social attitudes can presage changes in the law, as many economists

recognize, and because new laws can change social attitudes, as many politicians recognize. Finally, as emphasized among some criminologists and sociologists, the social acceptance of drunk driving laws can be vital to their success, making the two variables complementary.

Generally, these relationships would imply that the estimated effect of laws in standard traffic safety analyses is favorably biased. The difficulty of disentangling them, empirically, should caution anyone who would declare the efficacy of any drunk driving law based on such an analysis, especially if the legislation is to be "imposed" on states against their will.

## VII. Conclusion.

From a mathematical perspective, the process governing traffic fatalities is preternaturally elegant, allowing repeated use of scale analysis and elementary probability theory to unlock the secrets harbored within the data. In this paper we have tried to harness the full power of these tools to help staunch the tragic and heartbreaking carnage that gives rise to these data. The results illuminate the evolutionary, local nature of drunk driving dynamics; generate an alternative method of estimating the effects of legislation, which has several practical advantages; and reveals the limits of this legislation in modifying deviant driving behavior.

There are several reasons why laws' effects, while present, are not large. One is suggested by the dynamic analysis in Section I of this paper: people do change whether they drink and drive, but not the amount they drink when they drive. Accordingly, .08 or ZT laws aimed at accomplishing the latter purpose are not particularly effective. A second, as previously mentioned (Grant 2010b, 2010c), is that social acceptance of drunk driving laws is vital to their success. Consequently, laws

passed in certain states solely because of strong Congressional incentives are not effective, generating the "early-adopter effect" discussed in Section V of this paper. A third reason, suggested by Grant (2010d), is that, given the low probability of being apprehended if driving drunk and the modest penalties imposed on those who are convicted, the expected legal costs of drunk driving are dwarfed by the expected costs to one's own life, health, and property. Most drivers who are sensitive to these costs, therefore, will remain sober; those who do not are unlikely to be swayed by tougher laws, which generate only a small increase in total expected costs.

To the extent that legal factors have been overemphasized in research on the effects of drunk driving legislation, the effect of social forces have been underemphasized. While difficult to quantify with precision, these forces appear to be at least as important as laws are. For over a decade, drinking involvement in fatal crashes has remained flat. The secret to reinstituting a downward trend may be a renewed emphasis on social suasion.

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Table 1. Standard Deviations and Various Correlations of HBD, Log Fatalities, and More.

| | Standard Deviation | Spatial Correlation | Serial Correlation | Cross Correlation |
|---|---|---|---|---|
| HBD–Raw (percentage points) | 5.7 | 0.07 | 0.15 | 0.08 |
| HBD–Adjusted | 2.3 | 0.41 | 0.81 | 0.32 |
| Log Fatalities–Raw (times one hundred) | 13.2 | 0.18 | 0.32 | 0.08 |
| Log Fatalities–Adjusted | 8.1 | 0.46 | 0.84 | 0.32 |
| Implied General Risk Factor (percent) | 5.2 | 0.43 | 0.82 | 0.20 |
| Implied Drinking Factor (percent) | 5.4 | 0.41 | 0.81 | 0.20 |

Note: All observed and latent variables are measured (or presumed measured) at the state*year level, in deviations from state and year fixed effects (and log vehicle miles, for fatalities). Spatial correlations are calculated across matched state pairs. Using postal codes, the pairs are as follows: ME/MA, VT/NH, CT/RI, NY/NJ, TX/OK, KS/NE, ND/SD, WA/OR, CA/NV, UT/CO, ID/MT, MN/WI, MI/OH, IL/IN, IA/MO, AR/LA, AL/MS, TN/KY, GA/FL, NC/SC, VA/WV, MD/PA, DC/DE, AK/HI. "Adjusted" means that sampling variance has been removed. Cross correlations are the correlation of HBD and log fatalities, and the general risk factor with the implied drinking factor. There are 1173 observations (51 states * 23 years).

Table 2. Regressions, Adults Aged 21-60 (WLS or logit coefficient estimates, with standard errors in parentheses, the predicted percentage effect on the number of drivers involved in fatal accidents in straight brackets, and the average marginal effect on HBD in curly brackets when necessary).

| Dependent Variable<br>Change in Sample or Controls | .10 per se law | .08 per se law<br>(effect relative to<br>.10 per se) | Administrative<br>License<br>Revocation |
|---|---|---|---|
| Dummy for Driver BAC > 0<br>(logit model with age dummies) | 0.002<br>(0.017)<br>{0.04}<br>[0.08] | -0.095*<br>(0.014)<br>{-1.80}<br>[-3.75] | -0.033*<br>(0.014)<br>{-0.63}<br>[-1.31] |
| Add Other Driver and<br>Accident Controls | 0.016<br>(0.020)<br>{0.30}<br>[0.63] | -0.083*<br>(0.026)<br>{-1.57}<br>[-3.27] | -0.031*<br>(0.016)<br>{-0.59}<br>[-1.23] |
| $-\log(1-HBD_{s,t})$ | -0.008<br>(0.013) | -0.046*<br>(0.011) | -0.026*<br>(0.010) |
| **$HBD_{s,t}$ , Single Vehicle Accidents<br>(mean = 52%)** | **-0.03<br>(0.60)<br>[-0.06]** | **-2.45*<br>(0.49)<br>[-5.10]** | **-0.94*<br>(0.47)<br>[-1.96]** |
| Single Vehicle Night<br>(mean HBD = 71%) | 1.34<br>(0.71)<br>[4.62] | -2.28*<br>(0.59)<br>[-7.86] | -0.55<br>(0.55)<br>[-1.90] |
| Vehicles ≤ 2<br>(mean HBD = 36%) | 0.54<br>(0.50)<br>[0.84] | -1.46*<br>(0.40)<br>[-2.28] | -0.98*<br>(0.38)<br>[-1.53] |
| *No Imputed Data*<br>Single Vehicle | -1.69*<br>(0.78) | -1.73*<br>(0.66) | -1.98<br>(0.64) |
| Single Vehicle Night | -1.08<br>(0.76) | -1.10<br>(0.65) | -1.17<br>(0.61) |
| Vehicles ≤ 2 | -1.10<br>(0.68) | -0.73<br>(0.58) | -2.43*<br>(0.55) |

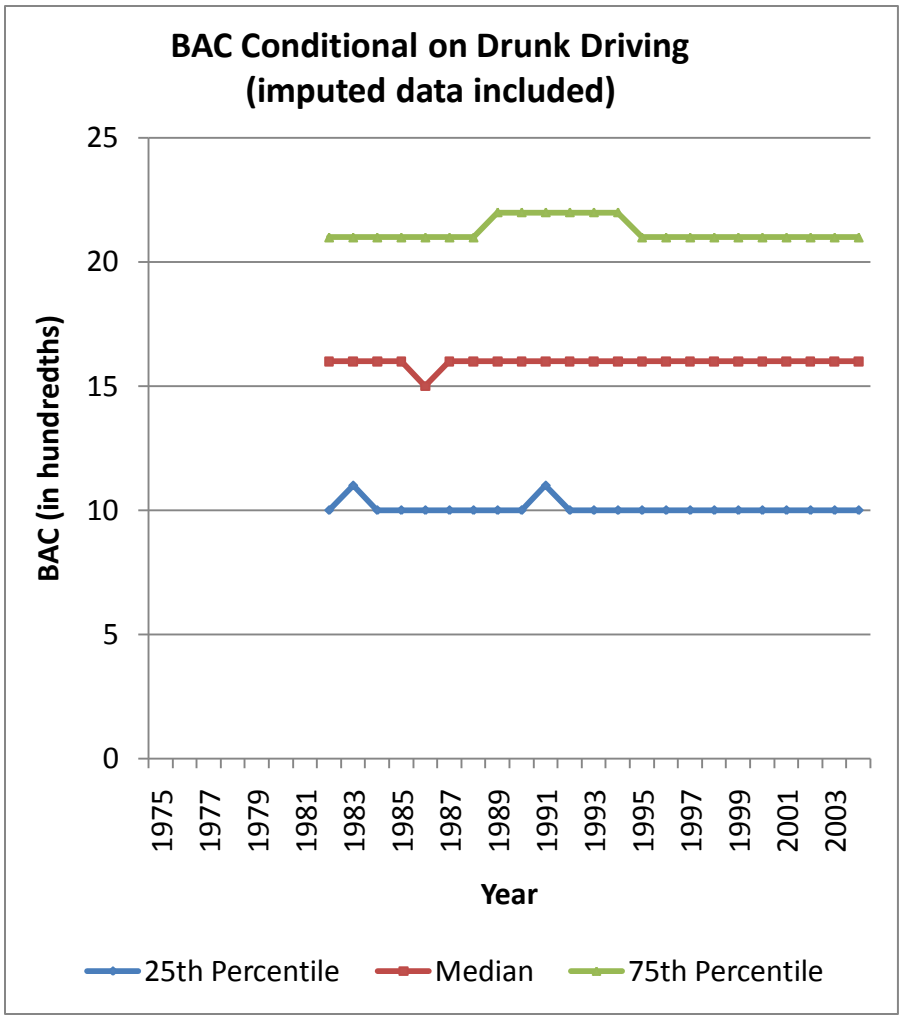
Note: All regressions use HBD as the dependent variable, analyze single vehicle accidents, and include imputed data unless otherwise indicated. Except in the logit models, N = 1058, 48 states (not AK, DC, HI) for 23 years, subtracting out years prior to discrete jumps in BAC reporting in twelve states. Controls include state and year fixed effects and unemployment, along with a dummy variable to distinguish one car accidents from two car accidents in the "vehicles ≤ 2" regressions. The full logit uses 327,560 individual observations and includes dummies for age, gender, hour of the day, day of the week, and month. * means the coefficient estimate differs significantly from zero at $\alpha = .05$.

Table 3. Regressions, Drunk Driving Laws Aimed at Youth (WLS or logit coefficient estimates, with standard errors in parentheses, the predicted percentage effect on the number of drivers involved in fatal accidents in straight brackets, and the average marginal effect on HBD in curly brackets when necessary).

| | MINIMUM DRINKING AGE | | ZERO TOLERANCE | |
|---|---|---|---|---|
| Dependent Variable<br>Change in Sample/Controls | Youth<br>(aged 18-20) | Young Adult<br>(aged 21-25) | Youth<br>(aged 18-20) | Young Adult<br>(aged 21-25) |
| **$HBD_{s,t}$ in Single Vehicle Accidents (youth mean = 47%, young adults = 60%)** | **3.80***<br>**(1.57)**<br>**[7.17]** | **0.40**<br>**(1.29)**<br>**[1.00]** | **-2.17**<br>**(1.27)**<br>**[-4.09]** | **-1.00**<br>**(1.06)**<br>**[-2.50]** |
| Single Vehicle Night<br>(mean HBD = 63%, 75%) | 2.59<br>(1.92)<br>[7.67] | -0.36<br>(1.47)<br>[-1.44] | -1.34<br>(1.62)<br>[-3.62] | -1.59<br>(1.23)<br>[-6.36] |
| Vehicles ≤ 2<br>(mean HBD = 35%, 46%) | 3.67<br>(2.29)<br>[5.17] | 1.52<br>(1.00)<br>[2.81] | -2.12*<br>(0.88)<br>[-3.09] | -1.03<br>(0.81)<br>[-1.91] |
| *No Imputed Data*<br>Single Vehicle | 4.55*<br>(2.05) | 0.33<br>(1.60) | -2.63<br>(1.63) | -0.67<br>(1.29) |
| Single Vehicle Night | 3.37<br>(2.22) | 0.01<br>(1.54) | -0.87<br>(2.22) | -1.54<br>(1.26) |
| Vehicles ≤ 2 | 5.94*<br>(1.56) | 2.06<br>(1.39) | -2.49*<br>(1.23) | -0.92<br>(1.10) |
| *Single Vehicle Accidents*<br>Dummy for Driver BAC > 0<br>(logit model with age dummies) | 0.088*<br>(0.041)<br>{1.63}<br>[3.07] | ---- | -0.082<br>(0.043)<br>{-1.54}<br>[-2.91] | ---- |
| Add Other Driver and Accident Controls | 0.068<br>(0.047)<br>{1.26}<br>[2.38] | ---- | -0.080<br>(0.048)<br>{-1.49}<br>[-2.81] | ---- |

Note: N = 1058, 48 states (not AK, DC, HI) for 23 years, subtracting out years prior to discrete jumps in BAC reporting in twelve states. State and year fixed effects, unemployment, .08 and .10 BAC laws, and ALR laws controlled for, along with a dummy variable to distinguish one car accidents from two car accidents in the "vehicles ≤ 2" regressions. The full logit uses 59,490 individual observations and includes dummies for age, gender, hour of the day, day of the week, and month. * means the coefficient estimate differs significantly from zero at $\alpha = .05$.

Figure 1. BAC Conditional on Driving after Drinking, Drivers Involved in Fatal Accidents, Nationwide: with Imputed Data (on left) and without.



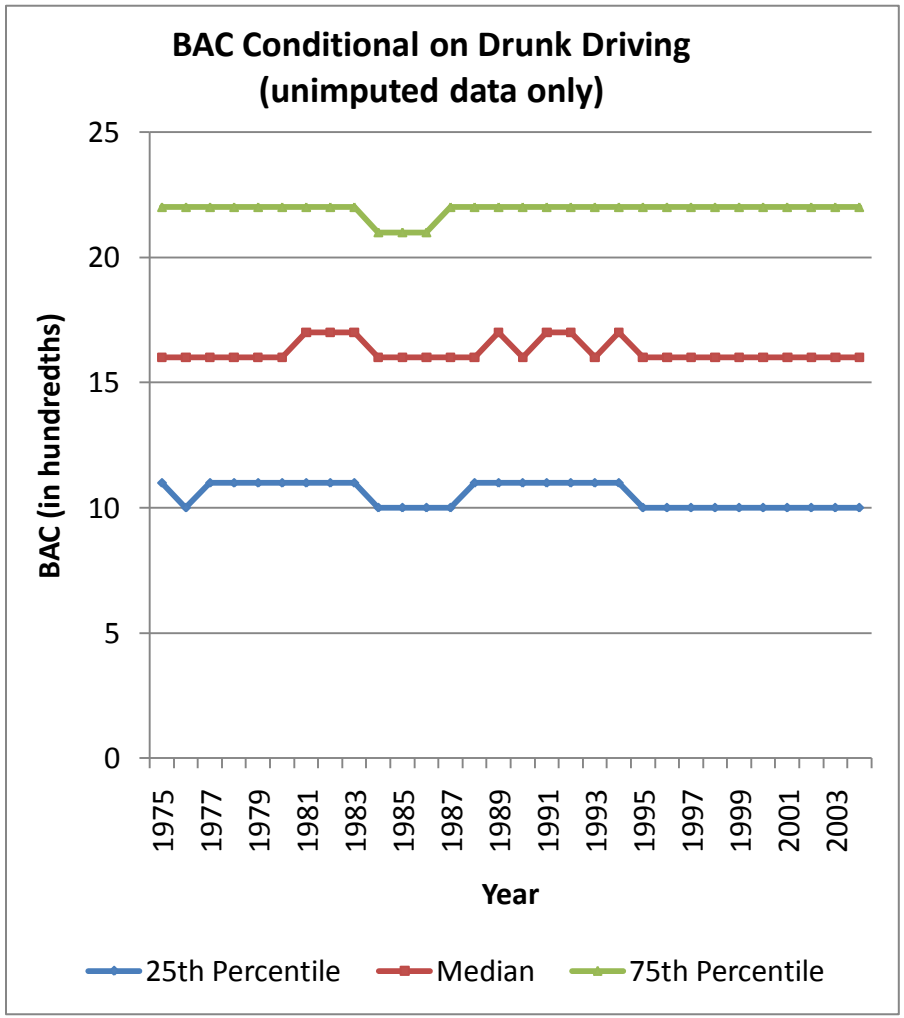
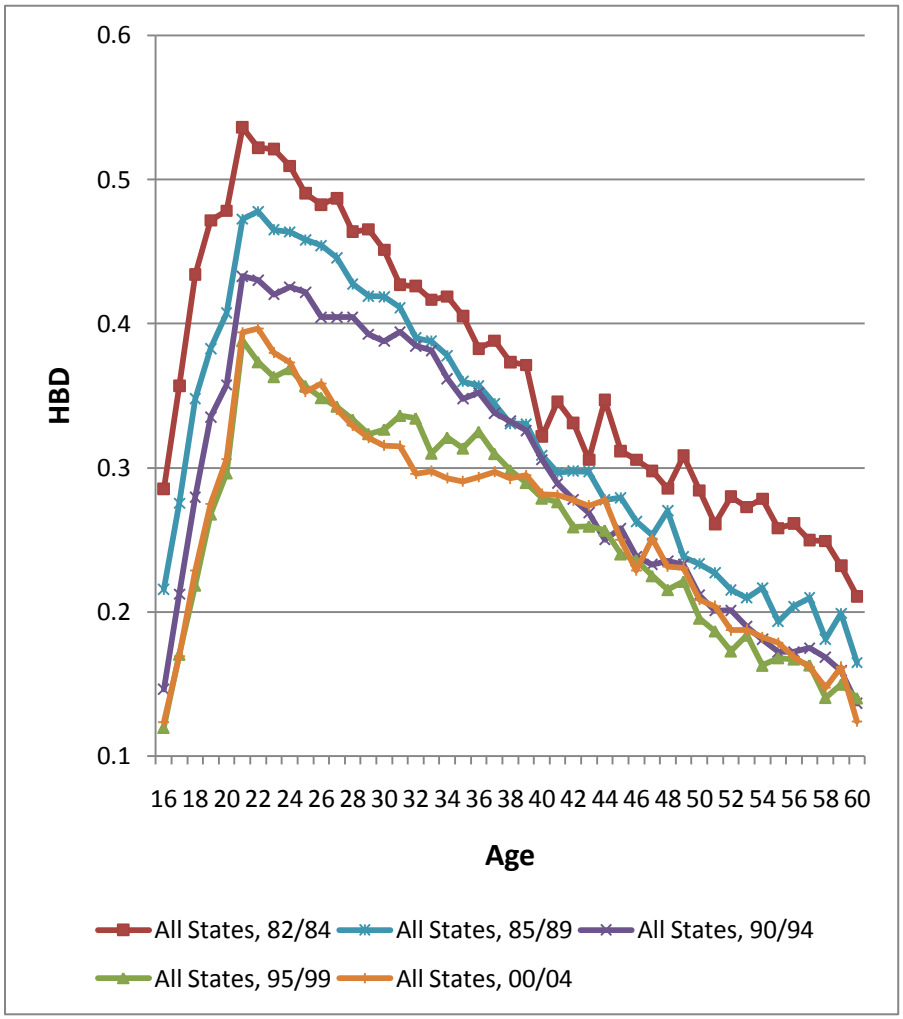



**Figure 2. Evolution of HBD in the U.S.: Profiles by Age, with Imputed Data (on left) and without.**



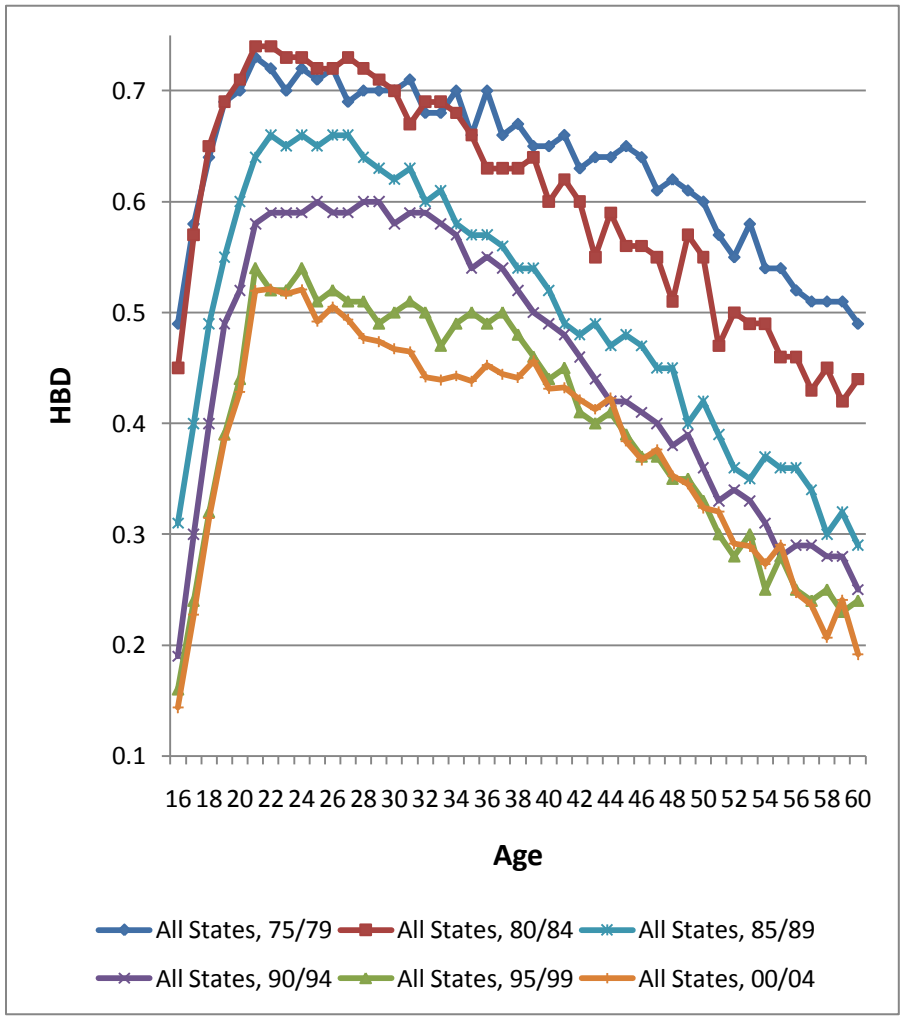
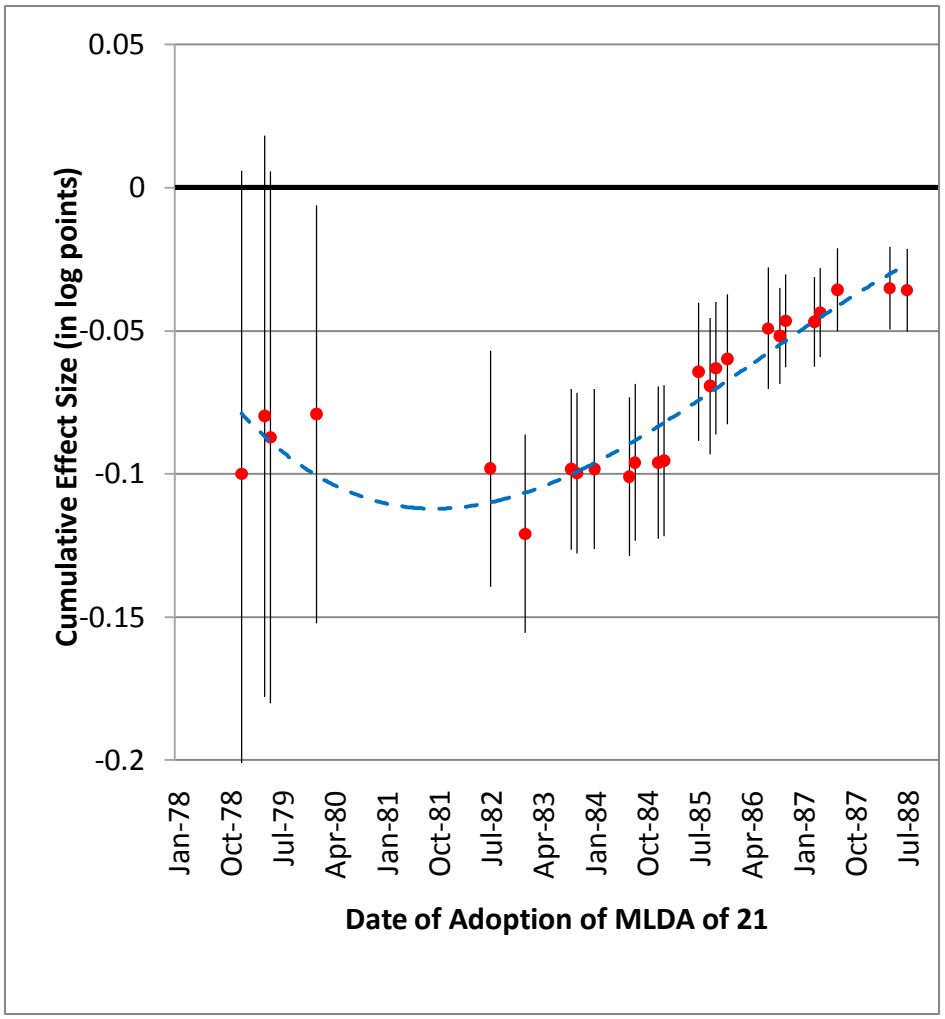



Figure 3. Timing of MLDA Adoption and Estimated Effect Size. (For description, see the text.)



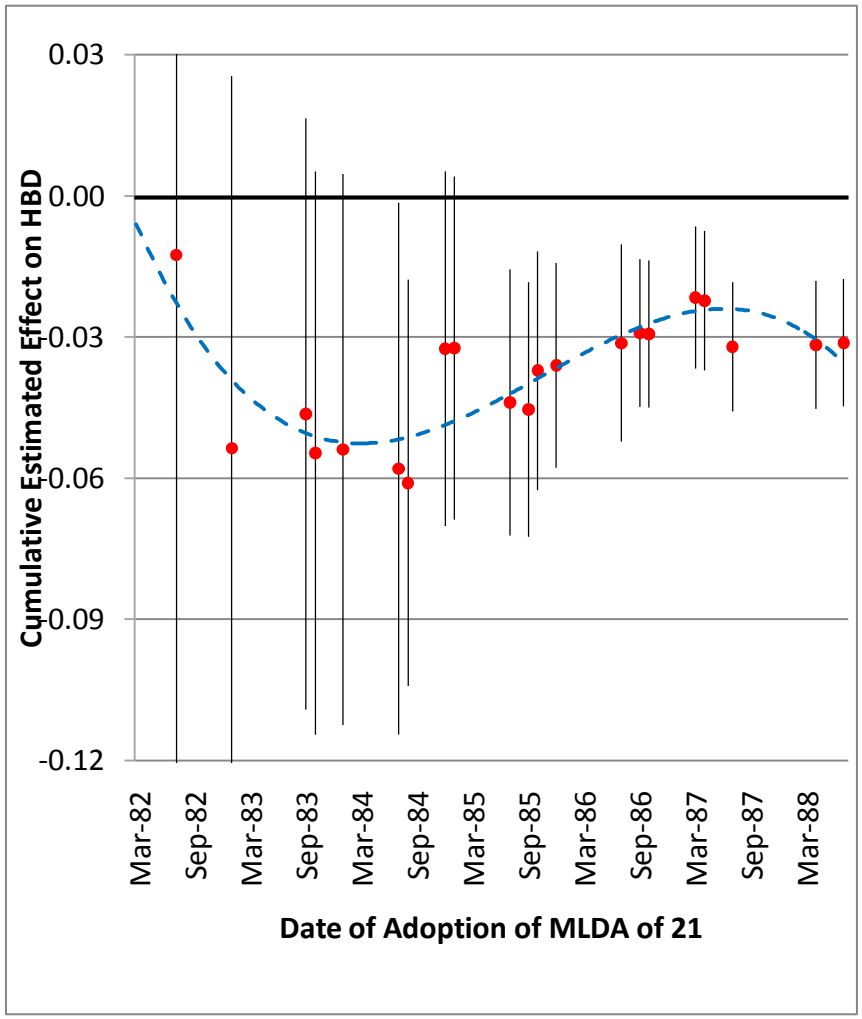



**Figure 4. Timing of ZT Adoption and Estimated Effect Size. (For description, see the text.)**

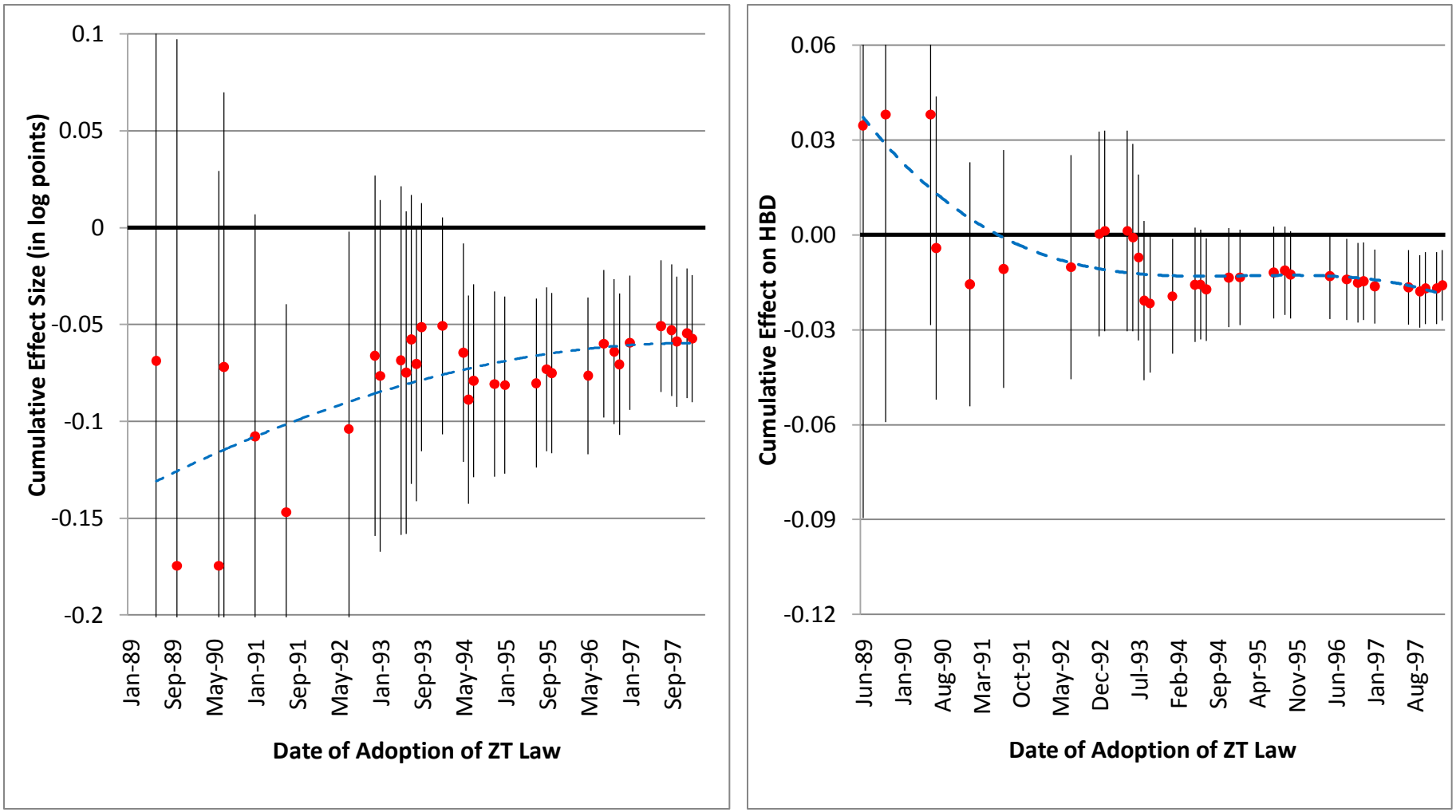


Figure 5. Timing of .08 Law Adoption and Estimated Effect Size. (For description, see the text.)

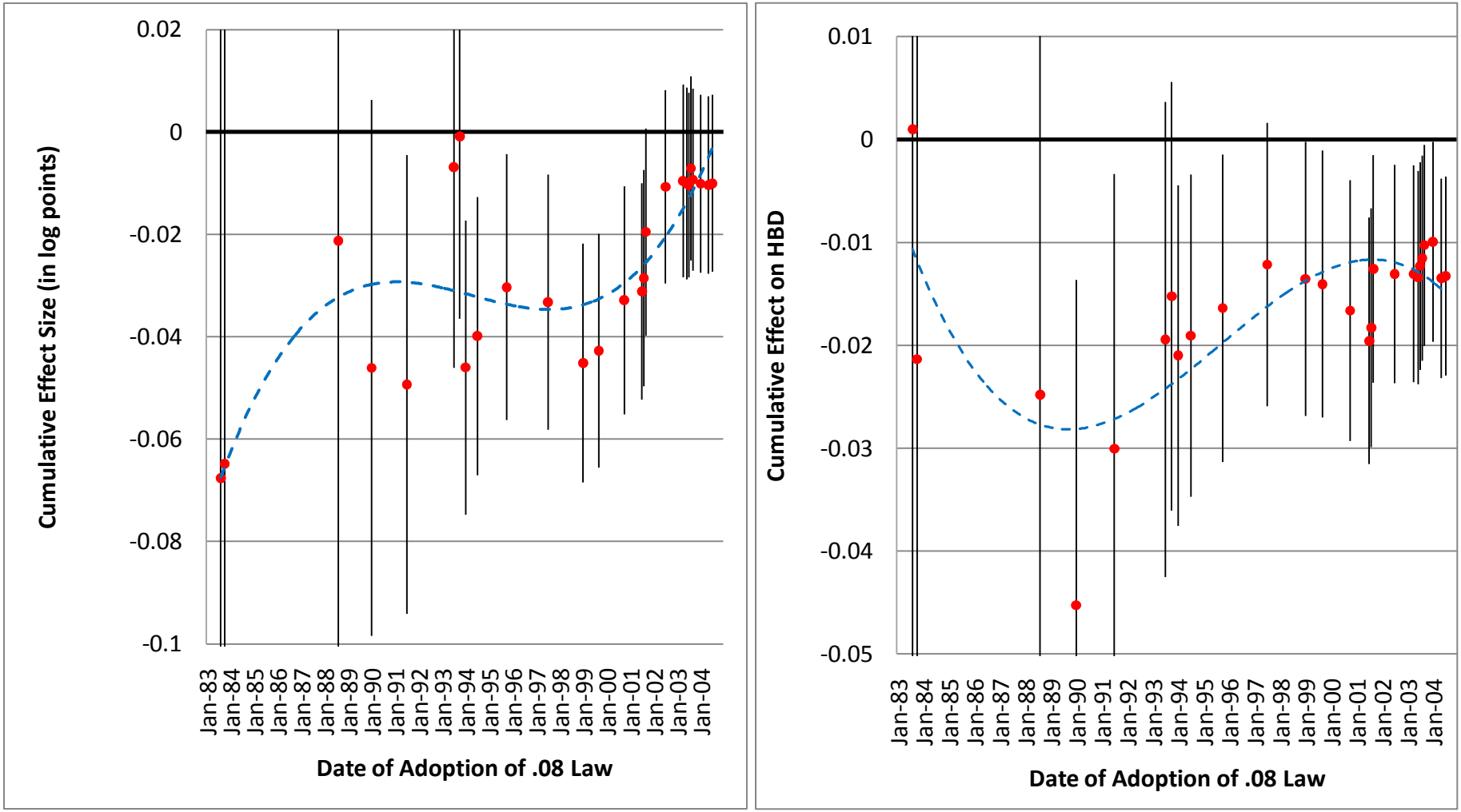


Figure 6. Decomposition of the Reduction in HBD in Single-Vehicle Accidents, 1986-2004.