

Much more on the role of alcohol in  
Chapter 10 *Alcohol* of *Traffic Safety*

## THE FRACTION OF TRAFFIC FATALITIES ATTRIBUTABLE TO ALCOHOL

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**Abstract**—The vast literature on alcohol's effect on traffic safety does not contain even a moderately satisfactory answer to one of the most basic questions, namely "What is the fraction of all traffic fatalities attributable to alcohol use?" A published estimate of 23.7% based on an erroneous calculation has been widely quoted. This paper combines 1987 Fatal Accident Reporting System (FARS) data from 26 states that recorded blood alcohol concentrations for over 84% of fatally injured drivers with published estimates on how alcohol affects crash risk. By categorizing all traffic fatalities as either nonoccupants of vehicles, or occupants killed in single-vehicle, two-vehicle or three-or-more-vehicle crashes, and developing calculation procedures appropriate for each category, the fraction of all fatalities due to alcohol was inferred. The main finding was that eliminating alcohol would reduce traffic fatalities by  $(47 \pm 4)\%$ . It was also concluded that alcohol use changes from 1982 to 1987 have reduced traffic fatalities by 12% (6,400 fatalities), which helps explain the absence of the fatality increase predicted because of a buoyant economy. Reducing the fraction of fatalities due to alcohol from the 1987 value of 47% to 42% (say) would reduce all traffic fatalities by 8%.

### INTRODUCTION

A vast technical literature addresses the role of alcohol in traffic safety; one recent review (National Highway Traffic Safety Administration 1985) cites 560 references, while Moskowitz and Robinson (1987) identify 557 citations on the narrower subject of the influence of alcohol on skills performance. It is therefore surprising that this literature does not contain even a moderately satisfactory answer to what must surely be one of the most basic and fundamental questions about alcohol's role in traffic safety. That question is "What fraction of all traffic fatalities would be prevented if alcohol did not exist?" The same basic question can be expressed in alternative forms, such as "What is the fraction of all traffic fatalities attributable to alcohol use?" It is the purpose of the present research to answer this question.

It is widely reported and well known that alcohol consumption is involved in about 50% of fatal crashes. Such information by itself does not address how many fatalities could be prevented by eliminating alcohol, as is readily apparent when we consider that coffee consumption is probably involved in nearly all crashes, yet eliminating coffee is unlikely to have much effect on fatalities. Because all drivers have some risk of crash involvement, eliminating alcohol would not change alcohol-consuming drivers into risk-free drivers. Therefore, for single-vehicle crashes, eliminating alcohol necessarily reduces fatalities by a lesser proportion than the proportion of all such crashes that involve alcohol; for multiple-vehicle crashes the situation is more complex.

The observation that about 50% of fatal crashes involve alcohol has contributed to a popular impression that about 50% of fatalities are attributable to alcohol use. Such an understanding is further enforced by the opening sentence of the summary of the 1968 landmark Department of Transportation report to Congress (U.S. Department of Transportation 1968), which reads "The use of alcohol by drivers and pedestrians leads to some 25,000 deaths" (that is, half of the total deaths in the mid-1960's). This 50% value was arrived at subjectively after examination of a large body of data. Although, as we shall see, remarkably insightful given the limitations of the data and analyses then available, this value was not based on any specified calculation or direct inference from the data presented.

The only study in the technical literature that provides a quantitative answer to the

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question we posed based on analyzing data is one published by Reed (1981) under the auspices of the National Academy of Sciences. The estimate obtained was 23.7%. This estimate has been highly influential and much quoted, as in the following recent (1986 to 1988) examples: "The National Academy of Science's estimate is that about one-fourth of deaths and considerably less of injuries and property damage can be attributed to alcohol" (Ross 1988, p. 78); ". . . if every drunk driver could be permanently removed from the roads—an ideal hypothesis at best—the wisest estimates suggest that the death toll would decline by only one-fourth" (Ross 1986, p. 169); "The National Academy of Sciences estimated that if no one drove after drinking, 11,000 fewer persons annually would die in crashes, approximately a 20%–25% annual reduction" (Hingson et al. 1988); ". . . drunk driving can be held causally responsible for only about a quarter of traffic fatalities . . ." (Ross 1988, p. 75); ". . . alcohol is implicated as a causal factor in a large proportion of serious crashes in the United States and Canada, although this proportion is not nearly as large as the 50% and higher frequency mentioned in the media, but more like a quarter of fatal crashes . . ." (Ross 1987; p. 475).

This paper addresses the question by bringing together two information sources. First, the distribution of driver fatalities by alcohol use from a national traffic fatality data file. Second, risk factors available in the literature that enable us to estimate how many of these fatally injured drivers would have been killed if they had been alcohol-free. Different calculations are required for single-vehicle and multiple-vehicle crashes; for the single-vehicle case, calculating the number of excess deaths attributable to alcohol is relatively simple, because the only occupants affected are the driver and the other occupants in that driver's vehicle.

Multiple-vehicle crashes are more complex, because the risks faced by alcohol-free drivers are influenced by alcohol-consuming drivers. The finding that drivers killed in multiple-vehicle crashes tend to have consumed less alcohol than those killed in single-vehicle crashes has encouraged a tendency to assume that alcohol effects in multiple-vehicle crashes are proportionately lower. The following (extremely hypothetical) construct is offered to show that making inferences from the distribution of alcohol in those fatally injured in multiple-vehicle crashes is not that simple.

Consider an imaginary population of drivers who never crash. Now, let some very small fraction of these drivers consume a substance which makes them likely to crash into the next object they encounter, be it a tree or another vehicle. Examining fatally injured drivers would reveal 100% substance use in single-vehicle crashes compared to barely more than 50% use in two-vehicle crashes, notwithstanding that eliminating the substance would eliminate the same fraction of driver deaths (namely, 100%) in single- and two-vehicle crashes.

In the present paper we develop a calculation procedure that uses the observed distribution of alcohol in drivers fatally injured in multiple-vehicle crashes to infer how many of these drivers would not have been killed if all drivers had been alcohol-free. By summing occupants killed in two-vehicle crashes, three-vehicle crashes, and crashes involving more than three vehicles, together with occupants killed in single-vehicle crashes and all nonoccupants killed, the total number of fatalities attributable to alcohol is estimated. All the estimates depend on assuming that when drunk drivers are sober, their crash risk is the same as that for average sober drivers. While there are indications in the literature that this may not be so (for example, Hurst 1973), the unavailability of any quantitative relationship makes it impossible to include such effects in the present analysis.

#### METHODS AND MATERIALS

Data on fatal crashes are obtained from the Fatal Accident Reporting System (FARS), a computerized data file maintained by the National Highway Traffic Safety Administration containing detailed information on all traffic crashes occurring in the United States since January 1, 1975 in which anyone was killed (National Highway Traffic Safety

Administration 1988). Because the contribution of alcohol to traffic fatalities has been changing (National Highway Traffic Safety Administration 1988, Table 2-2), we confine our attention to just one year's data, namely, that for 1987.

Blood Alcohol Concentration (BAC, measured as the percent, by weight, of alcohol in the blood) values are available for some, but not nearly all, persons coded in FARS. Availability is higher for drivers than for other road users, higher for the fatally injured than for survivors, and varies widely from state to state. For example, the percentage of fatally injured drivers with known BAC varies from 95.6% in Delaware to 9.3% in Mississippi. In order to avoid, to the extent possible, the types of biases which can arise when missing data are correlated with phenomena being investigated (Feinstein 1988), we focus on drivers fatally injured in states which measure BAC for a large fraction of such drivers. Specifically, we use data from 26 states that measured BAC for over 84% of fatally injured drivers, the average for these states being 88%.

Table 1 shows information on all drivers (of any motorized vehicle, including motorcycles) and nonoccupants (essentially pedestrians) killed in traffic crashes in these 26 states in 1987. The six BAC ranges recur in the paper, and will be referred to as  $i = 1$  (for BAC = 0) through  $i = 6$  (for BAC  $\geq 0.2\%$ ). For expository convenience, the first range is designated BAC = 0, rather than the more strictly technically correct BAC < 0.001%. The additional space in Table 1 at BAC  $\geq 0.1\%$  delineates those in violation of drunk driving laws.

Starting from the number of fatally injured drivers at a given BAC level, we estimate the number who would still have been killed even if alcohol-free, using the same type of calculation earlier applied to another risk-increasing factor, namely occupant ejection (Evans and Frick 1989). The risk-increasing effect of alcohol is quantified in terms of a

Table 1. Distributions of Blood Alcohol Concentrations (BAC) of fatally injured persons in the 26 states\* in 1987 FARS data with BAC known for over 84% of fatally injured drivers. All values, except those in the bottom row, are percents

i	Blood alcohol concentration, BAC (%)	Drivers killed in crashes involving:-				Non-occupants of vehicles
		One vehicle	Two vehicles	Three vehicles	> Three vehicles	
1	0.00	35.23%	65.67%	75.17%	72.41%	58.48%
2	0.001 - 0.049	3.35	4.22	3.55	6.21	3.54
3	0.050 - 0.099	6.75	5.24	5.24	3.45	4.27
4	0.100 - 0.149	12.11	5.61	3.55	5.52	4.63
5	0.150 - 0.199	16.80	7.31	3.72	4.14	6.89
6	$\geq 0.200$	25.76	11.95	8.78	8.28	22.19
All with known BAC		100.00	100.00	100.00	100.00	100.00
BAC unknown		10.55	14.06	13.32	12.12	27.08
Number of fatalities		5677	5016	683	165	3405

\*The 26 states, identified by their postal codes, are: CA CO CT DE HI ID IL IN ME MD MA MN MT NE NV NJ NM NC OR SD VT VA WA WV WI WY

relative risk factor,  $R$ , defined as

$$R(i) = \frac{\text{Risk that driver with BAC in range } i \text{ causes a crash}}{\text{Risk that driver with BAC} = 0.0 \text{ causes a crash}}, \quad (1)$$

in which we use the term "cause" for convenience and consistency with Borkenstein et al. (1964) while avoiding it in general for the reasons stated by Haight (1980) and Evans (1991). Estimates of  $R$  are available using the data in Table 2, which are from the landmark study conducted by Borkenstein et al. (1964) in Grand Rapids, Michigan, in the early 1960s. BAC levels were measured for 5,985 crash-involved drivers (only 622 in single-vehicle crashes) and 7,590 control drivers (the same study also described in Borkenstein et al. 1974). We focus on the subset of crash-involved drivers estimated to have caused the crash (see Table 43, page 230 and Chart XV of Borkenstein et al. 1964), because, as discussed previously, examining total involvement in multiple-vehicle crashes necessarily underestimates the causative role of any crash-rate-increasing factor.

In terms of the data shown in Table 2,  $R$  is given by

$$R(i) = \frac{A(i)}{C(i)} \times \frac{C(1)}{A(1)}, \quad (2)$$

where  $A(i)$  and  $C(i)$  represent the number of crash-causing and control drivers, respectively. Assuming the driver populations arise from a Poisson process, the standard error in  $R$  is given by

$$\Delta R(i) = R(i) \sqrt{1/C(i) + 1/A(i) + 1/A(1) + 1/C(1)}. \quad (3)$$

The values of  $R$  calculated using eqn 2 form the basis of this study. Later, in order to explore the sensitivity of results to  $R$  we use the lower and upper limits. All errors in this paper are standard errors, so there is a 68% probability that the true value is within the one standard error, and a 95% probability it is within two standard errors. The lower than unity value of  $R(2)$  is probably an artifact of the data as discussed by Allsop (1966). Rather than set  $R(2) = 1$ , which is probably more correct conceptually, we perform the calculations using the actual values shown in Table 2.

Table 2. Data from Borkenstein et al. (1964) for estimated accident-causing-driver group and control driver group.  $R(i)$  is the risk that a driver in BAC range  $i$  causes a crash impaired to the risk for a driver with BAC = 0

i	BAC (%)	Number of drivers		R(i)	$\Delta R(i)$	Lower limit	Upper limit
		Crash-causing	Control			R - $\Delta R$	R + $\Delta R$
1	0	2604	6756	1	-	-	-
2	0.001 - 0.049	200	589	0.88	0.075	0.81	0.96
3	0.050 - 0.099	143	187	1.98	0.22	1.76	2.21
4	0.100 - 0.149	172	44	10.1	1.7	8.4	11.9
5	0.150 - 0.199	123	10	31.9	10.5	21.4	42.4
6	>= 0.200	63	4	40.9	21.1	19.8	62.0

The following quantities will recur as we develop the results for the different types of crashes:

$$F(i) = \text{number of fatally injured with BAC in category } i \quad (4)$$

$$f(i) = \text{fraction of fatally injured drivers with BAC in category } i \quad (5)$$

$$G(i) = \text{number of drivers in population at risk with BAC in category } i \quad (6)$$

$$g(i) = \text{fraction of drivers in population with BAC in category } i. \quad (7)$$

## RESULTS

### *Single-vehicle crashes*

Suppose we have a population of  $N$  drivers killed in single-vehicle crashes, with  $F(i)$  of these in BAC range  $i$ . If the BAC of these  $F(i)$  drivers had been zero rather than in range  $i$ , a smaller number,  $F(i)/R(i)$ , would have been killed. Thus, instead of the original  $N$  fatalities, there would be

$$\text{Number of fatalities, assuming all at zero BAC} = \sum F(i)/R(i), \quad (8)$$

where the summation in this and all other cases is over the 6 BAC categories.

If  $T$  represents the ratio of the actual number of fatalities to the number that would have occurred if all drivers had zero BAC, then

$$T = N/[\sum F(i)/R(i)] = 1/[\sum f(i)/R(i)]. \quad (9)$$

As the normalized distribution of driver fatalities by BAC,  $f(i) = F(i)/N$ , is given in Table 1, and the values of  $R(i)$  are given in Table 2, the quantity  $T$  is readily computed as  $T = 2.232$ .

The above simple calculation answers the question for single-vehicle crashes; it has little uncertainty, and does not require knowledge about the population of drivers on the road. In order to prepare the ground for the more intricate computations involving more than one vehicle, we develop the same single-vehicle result starting from the different perspective of vehicles on the road.

Consider a population of  $D$  drivers, with  $G(i)$  of these in BAC range  $i$ . If the average crash rate for zero BAC drivers is  $k$  per year, then the  $G(i)$  drivers are expected to be involved in  $kG(i)R(i)$  crashes per year. If the probability of a fatality is proportional to the probability of crash involvement, then the number of fatalities to drivers in BAC range  $i$  is  $KG(i)R(i)$ , where  $K$  is  $k$  times the probability that a crash proves fatal. The total number of fatalities,  $N$ , is

$$N = K\sum G(i)R(i). \quad (10)$$

If all drivers had zero BAC (that is,  $R(i) = 1$  for all  $i$ ), then the total number of fatalities becomes  $K\sum G(i) = KD$ . Dividing eqn 10 by this value gives

$$T = \sum R(i)G(i)/D = \sum g(i)R(i) \quad (11)$$

where  $g(i) = G(i)/D$  is the fraction of all drivers in BAC range  $i$ . The fraction of all driver fatalities in BAC range  $i$  can be written

$$f(i) = g(i)R(i)/\sum g(i)R(i) = g(i)R(i)/T, \quad (12)$$

yielding

$$g(i) = Tf(i)/R(i). \quad (13)$$

The values of  $g(i)$  and  $f(i)$  are displayed in Table 3. Note also that combining eqns 11 and 2, and treating the  $G$ s and  $C$ s as equivalent, and taking  $f(1) = A(1)/\Sigma A(i)$  gives the additional result that

$$T = g(1)/f(1). \quad (14)$$

The set of  $g$  values derived should not be interpreted to refer to the overall population of all drivers, but rather to this population weighted by the various factors that are associated with involvement in single-vehicle crashes by zero BAC drivers; that is, it refers to a population preferentially consisting of young males driving at night, and in which the large number of daytime commuting drivers plays a lesser role. Specific values of  $g$  do not play a central role in any of the calculations that follow because they always appear as products with  $R$  values, namely  $g(i)R(i)$ . A different choice of  $R(i)$  leads to an essentially proportionate change in  $g(i)$ , but, for  $R(i) \gg 1$ , only a modest change in  $g(i)R(i)$ .

The value  $T = 2.232$  (from eqns 5, 11, or 14) gives that eliminating alcohol use reduces driver deaths in single-vehicle crashes by 55.2%.

#### Two-vehicle crashes

Consider the population of drivers to be at risk in two-vehicle crashes to contain  $D$  drivers,  $G(i)$  of them in BAC range  $i$ ; we use the same symbols as before, though meanings and values may differ. Assume that a specific vehicle with a zero BAC driver has a probability  $k$  (per year, say) of striking another specific zero BAC vehicle; assume further that the other vehicle has likewise a probability  $k$  of striking this specific vehicle. The number of expected two vehicle crashes between the specific vehicle and any zero BAC vehicle is  $kR(1)G(1) + kG(1)R(1)$ , where  $R(1)$ , the risk factor for the sober driver, has the value one; in this and the other cases below we ignore minor effects arising because vehicles cannot crash into themselves. The risk that this zero BAC driver crashes into any particular driver with BAC in some other range, say  $j$ , is greater than the risk of crashing into a specific sober driver by the nonsobber driver's risk factor  $R(j)$ , so that the risk that a specific zero BAC vehicle crashes into any vehicle in BAC range  $j$  is  $kR(1)G(j)R(j) + kG(j)R(j)R(1)$ . As the zero BAC vehicle may crash into any vehicle in the population, and any vehicle in the population may crash into it, the total crash risk (or expected number of crashes) is given by

$$\text{Crash risk for specific zero BAC vehicle} = kG(1)G(j)R(j) + kG(j)R(j)G(1). \quad (15)$$

Table 3. Results for single-vehicle crashes

$i$	BAC (%)	$R(i)$	$f(i)$	$g(i)$
1	0	1	35.23%	78.64%
2	0.001 - 0.049	0.88	3.35	8.50
3	0.050 - 0.099	1.98	6.75	7.61
4	0.100 - 0.149	10.1	12.11	2.68
5	0.150 - 0.199	31.9	16.80	1.18
6	$\geq 0.200$	40.9	25.76	1.41

The number of crashes for all zero BAC drivers,  $n(1)$ , is given by

$$\begin{aligned} n(1) &= kG(1)R(1) \sum G(j)R(j) + k[\sum G(j)R(j)]G(1)R(1) \\ &= 2kG(1)R(1)\sum G(j)R(j). \end{aligned} \quad (16)$$

If, instead of concentrating on zero BAC drivers, we consider drivers in BAC range  $i$ , then extending eqn 16 gives the number of two-vehicle crashes,  $n(i)$ , in which drivers in this range are involved as

$$n(i) = 2kG(i)R(i)\sum G(j)R(j). \quad (17)$$

The total number of two-vehicle crashes is then given by

$$\sum n(i) = 2k[\sum G(j)R(j)]^2. \quad (18)$$

If we assume that fatality risk is proportional to crash involvement, then the fraction of all two-vehicle-crash driver fatalities that are to drivers in BAC range  $i$  is given by the ratio of eqns 17 and 18 to give

$$f(i) = G(i)R(i)/\sum G(j)R(j). \quad (19)$$

By dividing numerator and denominator by the number of drivers,  $D$ , the number of drivers (the  $G$ s) become replaced by the fraction of drivers in the population, represented by  $g$ s, to give

$$f(i) = g(i)R(i)/\sum g(j)R(j), \quad (20)$$

from which we derive

$$g(i) = [f(i)\sum g(j)R(j)]/R(i). \quad (21)$$

The ratio,  $T$ , of observed fatalities to the number expected if all had zero BAC is readily computed from eqn 18 as

$$T = [\sum g(j)R(j)]^2, \quad (22)$$

and can be numerically estimated using the values computed by eqn 21.

Not only is the above model of the two-vehicle-crash process plausible on intuitive grounds, but it has been successful in explaining other two-vehicle crash phenomena (Evans 1985). In the Appendix some quantities calculated using the model are compared to corresponding quantities obtained directly from the FARS data. Even though the calculations ignore increasing risk of death from the same impact with increasing BAC (Waller et al. 1986; Anderson and Viano 1987), the analysis in the appendix suggests that other confounding factors play an even larger role, so that the net effect is probably that the model underestimates the role of alcohol as a crash-risk-increasing factor in two-vehicle crashes. Stein (1989) also models two-vehicle crashes and finds large effects due to alcohol. Note that increased risk of death with increasing BAC would have zero influence on the results for drivers killed in single-vehicle crashes. Eliminating alcohol would eliminate the excess number of such driver deaths irrespective of whether their source was increased crash risk, or increased fatality risk in the same crash.

Table 4 shows the values of  $g(i)$  calculated using eqn 21. Substituting these into eqn 19 gives  $T = 1.817$ . This gives that eliminating alcohol reduces driver fatalities in two-vehicle crashes by 45.0%; a 17.0% contribution from drivers with BAC = 0 and a 28.0% contribution from drivers with BAC > 0. If  $R(i) = 1$  for all drivers, then two-vehicle fatality risk to BAC = 0 drivers would decrease by  $17.0/65.67 = 25.9\%$ . The use of

Table 4. Results for multiple-vehicle crashes

i	BAC (%)	R(i)	Two-veh crashes		Three-veh crshs	
			f(i)	g(i)	f(i)	g(i)
1	0	1	65.67%	88.52%	75.17%	91.08%
2	0.001 - 0.049	0.88	4.22	6.46	3.55	4.89
3	0.050 - 0.099	1.98	5.24	3.57	5.24	3.21
4	0.100 - 0.149	10.1	5.61	0.75	3.55	0.43
5	0.150 - 0.199	31.9	7.31	0.31	3.72	0.14
6	>= 0.200	40.9	11.95	0.39	8.78	0.26

alcohol by other drivers increases the number of zero BAC drivers killed in two-vehicle crashes by 35%.

#### Three-vehicle crashes

The mathematical derivations are essentially parallel to those for the two-vehicle crash case, with a set of  $g(i)$ 's being calculated by applying eqn. 21 (which applies to  $n$ -vehicle crashes for all  $n$ ) to the  $f(i)$  data to give the results in Table 4. Note that the  $g$  values differ from those for the two-vehicle crashes, which differed from those for the single-vehicle crashes; this is because three-vehicle crashes tend to occur at times of higher traffic density (morning and afternoon) when alcohol use is less, in contrast to the low density nighttime single-vehicle crashes. The two-vehicle crash  $g$  values are intermediate between those for three- and single-vehicle crashes. The  $g$  values for two-vehicle crashes are most similar to those observed directly in the control sample by Borkenstein et al. (1964), which are readily computed from Table 2, and are also close to those in Stein (1989). These values are not for random drivers, but for drivers observed at the same times and places as those involved in crashes (predominantly two-vehicle crashes).

The ratio,  $T$ , of drivers killed in three-vehicle crashes to the number expected if all drivers had zero BAC is given by

$$T = [\sum g(i)R(i)]^3. \quad (23)$$

For the  $n$ -vehicle case, the exponent becomes  $n$ . Substituting the values in Table 4 gives  $T = 1.779$ , so eliminating alcohol reduces driver fatalities in three-vehicle crashes by 43.8%.

#### Estimates of overall fatality reductions associated with zero alcohol use

In order to estimate the overall reduction in all traffic fatalities, we apply the results derived above for drivers in 26 states to drivers in all states, but later make a small adjustment to account for slightly higher alcohol use in the 26 states. To estimate effects for the entire nation, we use the distribution of all fatalities in the complete 1987 FARS file shown in Table 5.

We assume that the percentage increase in fatalities to drivers in crashes of any type applies equally to all occupants in vehicles driven by these drivers. This assumption is unlikely to add any appreciable error and is not violated just because passenger fatality risk may differ from that of the driver as a function of sex, age, or seating position (Evans 1988a; Evans and Frick 1988a). Such differing fatality risk is equally present independent of the BAC level of the driver.

The three-vehicle result is assumed to apply to crashes involving more than three

Table 5. Distribution of all 46,386 fatally injured persons in the 1987 FARS data by type of person and number of vehicles involved in the fatal crash

Persons fatally injured	No. of vehs involved in crash			Total
	One	Two	> Two	
Drivers	12,986	11,954	1,891	26,831
-----	-----	-----	-----	-----
All vehicle occupants	18,666 (40.24%)	17,164 (37.00%)	2,795 (6.03%)	38,625 (83.27%)
Non-occupants	7,167 (15.45%)	487 (1.05%)	107 (0.23%)	7,761 (16.73%)
Total	25,833 (55.69%)	17,651 (38.05%)	2,902 (6.26%)	46,386 (100.00%)

vehicles, which is why all crashes involving more than two vehicles are combined into one category in Table 5. The similarity of the two- and three-vehicle results leads one to expect that effects are similar for crashes involving more vehicles. Less than 2% of all traffic fatalities result from crashes involving more than three vehicles, so the overall estimate is highly insensitive to whatever assumption is made regarding crashes involving four or more vehicles.

The question of nonoccupants (essentially pedestrians) is more uncertain. If one assumes that the distribution in time of pedestrian risk is similar to that for driver fatalities in single-vehicle crashes, and that vehicles strike pedestrians in proportion to the rate at which they strike other objects (Evans 1984), then eliminating alcohol would reduce pedestrian fatalities in the same proportion it reduces single-vehicle driver fatalities, namely, 55%. However, pedestrian deaths tend to occur at times more like those for driver deaths in two-vehicle, not single-vehicle, crashes. It might, therefore, seem appropriate to consider drivers with the distribution of alcohol use given by the  $g(i)$ 's in Table 4, and to assume that they strike randomly distributed pedestrians in proportion to their  $R(i)$  factors. Such a calculation estimates that eliminating driver alcohol use would reduce pedestrian fatalities by 26%. However, this calculation ignores alcohol use by pedestrians, an assumption that the data in the last column of Table 1 unmistakably refute.

Clearly, alcohol use by pedestrians contributes to pedestrian fatality risk, a contribution that has been estimated in a field study by Blomberg and Fell (1979) using the same case-control method as Borkenstein et al. (1964). The increased risk of pedestrian involvement in traffic crashes found by Blomberg and Fell (1979) is consistent with the increased risk of involvement for drivers reported by Borkenstein et al. (1964). Therefore, the risk factors in Table 2 are consistent with risk factors for pedestrians being responsibly involved in, rather than just involved in, crashes. If the risk factors for pedestrians and drivers were identical, and the  $f(i)$ 's for drivers involved in two-vehicle crashes were identical to those for pedestrians, this would imply that eliminating alcohol would reduce pedestrian fatalities in the same proportion as for two-vehicle crashes, namely a 45% reduction. Because fatally injured pedestrians show higher BAC values than those for drivers killed in two-vehicle crashes, this 45% estimate is probably low. However, in the absence of firmer information, we assume that eliminating alcohol reduces nonoccupant deaths by 45.0%, keeping in mind that this is more likely an underestimate than an overestimate.

Using the information derived above, and summarized in Table 6, gives that the percent fatalities prevented by eliminating alcohol in the 26 states is

$$0.4024 \times 55.2\% + 0.3700 \times 45.0\% + 0.0603 \times 43.8\% + 0.1673 \times 45.0\% = 49.0\%. \quad (24)$$

Table 6. Summary of results (based on 46,386 traffic fatalities in 1987)

Source of fatalities	Percent of all fats	Pct due to alcohol		No. of fatalities preventable by eliminating alcohol
		26 states	All USA	
Occupants killed in:				
1-vehicle crashes	40.24%	55.2%	52.6%	9,818
2-vehicle crashes	37.00	45.0	42.9	7,363
> 3-vehicle crashes	6.03	43.8	41.8	1,168
Non-occupants	16.73	45.0	42.9	3,329
Total (or average)	100.00	(49.0%)	(46.7%)	21,678

This large role of alcohol in traffic fatalities is supported in two recent investigations. Donelson et al. (1989) conclude that 50% of fatal crashes in British Columbia, Canada, were due, at least in part, to alcohol use among drivers and pedestrians. Data in Stein (1989, Table 13) indicate that 66% of single-vehicle driver fatalities are attributable to alcohol, somewhat higher than the 55% found here for such crashes.

In order to examine the extent to which results from the 26 states apply to the entire nation, we use estimates of BAC levels for all fatally injured drivers (there is no BAC measurement for about 25% of them) obtained using discriminant factor analysis (Klein 1986; Fell and Klein 1986; Fell and Nash 1989). The results\* of such a calculation for 1987 are that 46.6% of fatally injured drivers had BAC > 0; the corresponding value for the 26 states from Table 1 is 48.9%, showing a greater role for alcohol in the 26 states. In order to estimate the fraction of U.S. traffic fatalities attributable to alcohol, we consider how the fraction of drivers with BAC > 0 relates to the fraction of fatalities attributable to alcohol. When one of these quantities is zero, so is the other; when one is 100%, so is the other. For the 26 states, 48.9% of fatally injured drivers had BAC > 0 and 49.0% of traffic fatalities were attributable to alcohol. Thus it appears that one of the quantities is approximately proportional to the other. To obtain the fraction of U.S. fatalities attributable to alcohol we therefore multiply the estimate for the 26 states by a scaling factor reflecting the ratio of fatally injured drivers with BAC > 0 in the U.S. to the value in the 26 states to obtain

$$49.0\% \times 46.6/48.9 = 49.0\% \times 0.953 = 46.7\%. \quad (25)$$

The contributions from the various crash types are similarly multiplied by 0.953 to give the estimates for the United States shown in Table 6.

*Why is the present answer so different from the earlier estimate?*

The main data supporting the 23.7% estimate of Reed (1981) are from 106 drivers fatally injured in Vermont from July 1, 1967 to April 30, 1968. The same paper also gives data for other injury levels obtained from various studies using methodology similar to that used by Borkenstein et al. (1964).

Reed (1981) applied a calculation essentially equivalent to that reflected in our eqn 14 to all data *except* the Vermont data (see Table 7). These data were treated differently because an estimate of the effect of eliminating alcohol on all, not just driver, fatalities was sought. Therefore, an attempt was made to correct the driver distribution so that

\*The values used are the same as the published values (National Highway Traffic Safety Administration 1988), but were kindly provided by James Fell to the additional decimal point required for the present analysis.

Table 7. Data for 106 drivers killed in crashes in Vermont in 1967–1968, as given in Table A-8, page 378 of Reed (1981). The distribution for crash-involved drivers is what we have called  $f(i)$ , and that for control drivers a direct observation of  $g(i)$

$i$	BAC (%)	Fatally-injured drivers	Control drivers	$R(i)$
1	0	44.92%	83.74%	1
2	0.001 - 0.049	5.08	9.32	1.02
3	0.050 - 0.099	8.49	4.80	3.27
4	0.100 - 0.149	13.21	1.16	21.07
5+	$\geq 0.150$	28.30	0.98	53.43

it would apply to all fatally injured persons by performing various arithmetical manipulations using data on the number of drivers responsible for fatal crashes (mainly pedestrian) in Boston.

The following considerations show that the arithmetical manipulations in Reed (1981), which do not appear to have any coherent basis, generate an estimate which is logically inconsistent with the data presented. Applying the identical calculation that was applied by Reed (1981) to all the other data (that is, eqn 14) gives that eliminating alcohol reduces driver fatalities by 46.4%. Reed (1981) states that 59% of all Vermont traffic fatalities were driver fatalities. Combining these values implies that eliminating alcohol generates a  $0.59 \times 46.4\% = 27\%$  reduction in overall traffic fatalities due to reducing driver deaths alone. That is, the contribution from reducing driver deaths alone exceeds the overall estimate of 23.7%. The erroneous result does not flow from some specific error, but from applying a series of steps that not only lack plausible justification, but generate a total result that is smaller than one of its parts. It should be stressed that the source of most of the large discrepancy between the former and present estimates is the invalid arithmetical manipulations performed in the earlier estimate; the basic assumptions and underlying conceptualization are similar for both studies.

#### *Sensitivity of result to assumptions*

As estimates without some indication of uncertainty are of limited value (Evans 1988b; 1991), we proceed to estimate confidence limits for the 46.7% value. As is often the case, including many instances in the physical sciences, this involves including judgmental estimates of some components in the error calculation.

The 46.7% value depends on the  $R(i)$  values in Table 2, which contribute to many steps in the calculation. Repeating the complete calculation using the lower limit set of  $R$ s in Table 2 gives an estimate of 44.9%. The reason why so large a change in the risk factors leads to so small a change in the result is that once a risk factor comfortably exceeds unity, further increases do not much influence the calculation. Using the upper limit set of  $R$  values in Table 2 has an even smaller effect, increasing the estimate from 46.7% to 47.9%. The set of  $R$  values used is compatible with other estimates (Reed 1981; Donelson et al. 1987) based on smaller studies; for example, using the set of  $R$  values in Table 7 gives an estimate of 49.6%. Some confounding effects may increase estimates of  $R$ , because alcohol use is correlated with other risk-enhancing characteristics (youth, male). However, because the control and crash-involved drivers are matched in day of week and time, such effects should not excessively distort the  $R$  estimates. Basically, no different realistic set of  $R$  values will influence the solution by more than a percent or so. We thus conclude that variations in the choice of  $R$  may generate a 2%

absolute error in the estimate. Relative to the 47% estimate, this is a 4.3% error; below, percent errors are, like the 4.3%, relative to the estimated, and not absolute, value.

The distributions of the  $f(i)$ 's are based on sufficiently large samples that statistical uncertainty is relatively unimportant (Table 1 shows 5,078 driver fatalities in single-vehicle crashes and 4,311 in two-vehicle crashes). Cases with fewer data (592 for three-vehicle crashes) contribute so little to the overall estimate that, again, statistical uncertainty is negligible. The distribution of all fatalities into the four categories is based on a census for the study year, so there is no statistical uncertainty.

To establish error limits we make judgmental estimates of the errors associated with each step of the calculation. For single-vehicle crashes, few assumptions were required, and those made are likely to be closely obeyed. I judge the single-vehicle crash estimate to be reliable to within 5%. As the model for the multiple-vehicle crashes is more uncertain, let us assume that it has an associated uncertainty of 10%. All multiple vehicle effects are combined because errors in the two- and three-vehicle cases are likely in the same direction, so treating them as independent would underestimate the net error. Let us assume an error of 20% of the estimated reduction for nonoccupants, the most uncertain component in the calculation; that is, we consider a range, centered at 45.0%, that extends from 36% to 54%. This is plausible in view of the earlier discussion. Assuming that the single-vehicle, multiple-vehicle, and nonoccupant errors of 5%, 10%, and 20% operate independently, we use standard formulae in the propagation of errors (Young 1962, pp. 97-99) to estimate a percentage error in the overall estimate from these sources of 6.4%. This is then combined with the earlier discussed 4.3% error contribution associated with the  $R$  values; assuming that these act independently, the combined error is the square root of the sum of squares, or 7.7%. Thus, we estimate an error of 7.7% of the estimated effect of 46.7%, or, converting to absolute errors, an error of  $0.467 \times 7.7\% = 3.6\%$ . Our estimate of the percentage of fatalities that would be prevented by eliminating alcohol is therefore  $(47 \pm 4)\%$  for 1987; this is equivalent to a reduction of between 20,000 and 24,000 fatalities annually.

#### *Effect of eliminating drunk driving rather than alcohol*

The calculation was reported comparing observed fatalities to those estimated assuming all road-users had BAC levels that did not exceed various levels, rather than just zero BAC. Setting the maximum BAC equal to the highest of the categories still legal in most (but not all) U.S. states, namely 0.05% to 0.099%, for which  $R(3) = 2.98$ , leads to an overall reduction in fatalities of 41%, compared to the 47% estimate for all drivers at zero BAC. That is, all drivers with illegal levels of BAC acquiring a distribution of BAC similar to that presently observed for drivers with BAC between 0.050% and 0.099% would reduce traffic fatalities by 41%. Setting the maximum risk level at other values generates the following percentage declines in fatalities: 44% for a maximum risk of 1.5; 39% for a maximum risk of 3; 34% for a maximum risk of 6; 26% for a maximum risk of 10. The results of Borkenstein et al. (1964) suggest that a driver at 0.10% BAC has a risk of causing a crash about 6 times that of a sober driver, so our results indicate that if all legally drunk drivers changed to marginally legal levels of just under 0.10% BAC, traffic fatalities would decline by 34%.

## DISCUSSION AND CONCLUSIONS

The estimate that eliminating alcohol would reduce traffic fatalities by  $(47 \pm 4)\%$  is based on the assumption that if drivers with a given BAC level did not consume alcohol, they would drive at the same risk level as drivers with BAC = 0 observed at similar places on similar occasions. Although there are clear indications that heavy users of alcohol differ from the general public in ways other than their alcohol use (Donovan et al. 1983; Stutker et al. 1980), and that such differences could lead to different crash rates even when alcohol-free (McCord 1984), there does not appear to be sufficiently firm quantitative information to modify our estimate. Any such effect would tend to lower the fatality reductions associated with eliminating alcohol.

In 1987, the study year, 46.6% of fatally injured drivers had BAC > 0, compared to 53.1% in 1982 ([Klein 1986; Fell and Klein 1986; Fell and Nash 1989]). Replacing 46.7 by 53.1 in eqn 25 gives that eliminating alcohol in 1982 would have reduced traffic fatalities by 53.2%. Hence, in 1982, alcohol increased fatalities to 2.14 times the zero-alcohol number, compared to 1.88 in 1987, implying a reduction in fatalities of  $0.26/2.14 = 12.1\%$  from 1982 to 1987 attributable to reduced alcohol use; the decline in the crashes specifically attributable to alcohol (all minus those not attributable to alcohol), from 1.14 to 0.88, represents a 23% decline. The origin of such reductions may be evolving social norms encouraged in part by grass-roots activism (Howland 1988). The reduction, equivalent to 6,400 fewer fatalities in 1987, helps explain why national fatalities did not increase during a time of economic expansion, as predicted by the multivariate model of Partyka (1984). This reduction is much larger than the 2%–3% decline that Hedlund and others (1984) found associated with reduced alcohol involvement in an earlier period (1980 to 1982) before grass-roots activism became so prevalent.

Apart from such overall behavioral factors as alcohol use, traffic safety measures generally focus on specific subsets of the driving population, or on specific crash types, so that their effectiveness is limited necessarily to some subset of all crashes or road users. Even if such interventions are effective in specific applications, their influence on total fatalities is diluted by all the cases to which they do not apply. For example, if the 55% of car drivers and right front passengers presently not wearing belts (National Highway Traffic Safety Administration 1989) became wearers, overall fatalities would decline by about  $0.56 \times (0.36 + 0.13) \times 43\% = 9\%$  (Evans 1986; 1990); all cars having airbags for drivers and right front passengers compared to no one using any restraint system generates an 8% reduction in overall traffic fatalities (Evans 1990); if the 35% or so of motorcyclists presently not wearing helmets (Evans and Frick 1988b) were all to become wearers, national traffic fatalities would decline by about  $0.35 \times 0.10 \times 28\% = 1\%$ .

The magnitude of the fatality reduction associated with eliminating alcohol found here suggests that interventions aimed at reducing drunk driving have the potential to produce larger fatality reductions than hitherto thought. For example, any measure that reduces the fraction of fatalities attributable to alcohol from the 1987 value of 47% to 42% (that is, a reduction of 10% in the 1987 value) is calculated to generate an overall fatality reduction of 8.6%; a decline of 12% was previously calculated to have occurred from 1982 to 1987. Rather than applying to a subset of traffic fatalities, broad measures to reduce alcohol use, such as reducing or deglamorizing its portrayal in the mass media (Evans 1987), changes in availability (Hauge 1988), or changes in taxation policy (Saffer and Grossman 1987; Phelps 1988a; b; Walsh 1987), have the potential to not only reduce all types of traffic fatalities, but to generate additional reductions in fatalities from nontraffic causes such as homicide and alcohol-related disease.

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## REFERENCES

- Allsop, R. E. Alcohol and road accidents. Report no. 6. Crowthorne, Berkshire, U.K.; Transport and Road Research Laboratory; 1966.
- Anderson, T. E.; Viano D. C. Effect of acute alcohol intoxication on injury tolerance and outcome. In: Noordzig, P.; Roszbach, R., eds. Alcohol, drugs and traffic safety—T86. Amsterdam: Excerpta Medical Elsevier Science Publisher; 1987; 251–254.
- Blomberg, R. D.; Fell, J. C. A comparison of alcohol involvement in pedestrians and pedestrian casualties. Proceedings of the 23rd annual meeting of the American Association for the Advancement of Medicine, p. 1–17; 1979.
- Borkenstein, R. F.; Crowther, R. F.; Shumate, R. P.; Ziel, W. B.; Zylman, R. The role of the drinking driver in traffic accidents. Department of Police Administration, Indiana University; 1964.
- Borkenstein, R. F.; Crowther, R. F.; Shumate, R. P.; Ziel, W. B.; Zylman, R. The role of the drinking driver in traffic accidents. Blutalkohol 11 (supplement 1); 1974.

- Donelson, A. C.; Beirness, D. J.; Mayhew, D. R.; Simpson, H. M. Blood alcohol concentration (BAC) limits in impaired-driving laws: Their history, scientific basis and effectiveness. Traffic Injury Research Foundation of Canada, Ottawa, Canada, Sept. 1987.
- Donelson, A. C.; Beirness, D. J.; Hass, G. C.; Walsh, P. J. The role of alcohol in fatal traffic crashes: British Columbia, 1985-1986. Traffic Injury Research Foundation of Canada, Ottawa, Canada, 1989.
- Donovan, D. M.; Marlatt, G. A.; Salzbeg, P. M. Drinking behavior, personality factors and high-risk driving—A review and theoretical formulation. *J. Stud. Alcohol* 44:395-428; 1983.
- Evans, L. Driver fatalities versus car mass using a new exposure approach. *Accid. Anal. Prev.* 16:19-36; 1984.
- Evans, L. Involvement rate in two-car crashes versus driver age and car mass of each involved car. *Accid. Anal. Prev.* 17:155-170; 1985.
- Evans, L. The effectiveness of safety belts in preventing fatalities. *Accid. Anal. Prev.* 18:229-241; 1986.
- Evans, L. Young driver involvement in severe car crashes. *Alcohol, Drugs and Driving* 3(3-4):63-78; 1987.
- Evans, L. Risk of fatality from physical trauma versus sex and age. *J. Trauma* 28:368-378; 1988a.
- Evans, L. Comments (on two papers on mandatory safety belt use laws, and reflections on broader issues). In: Graham J. D., ed., *Preventing automobile injury—New findings from evaluation research*. Dover, Ma: Auburn House; 1988b:73-83.
- Evans, L. Restraint effectiveness, occupant ejection from cars, and fatality reductions. *Accid. Anal. Prev.* 22:167-175; 1990.
- Evans, L. *Traffic safety and the driver*. New York: Van Nostrand Reinhold; 1991.
- Evans, L.; Frick, M. C. Seating position in cars and fatality risk. *Am. J. Public Health* 78:1456-1458; 1988a.
- Evans, L.; Frick, M. C. Helmet effectiveness in preventing motorcycle driver and passenger fatalities. *Accid. Anal. Prev.* 20:447-458; 1988b.
- Evans, L.; Frick, M. C. Potential fatality reductions through eliminating occupant ejection from cars. *Accid. Anal. Prev.* 21:169-182; 1989.
- Feinstein, A. R. Scientific standards in epidemiologic studies of the menace of daily life. *Science* 242:1257-1263; 1988.
- Fell, J. C.; Klein, J. M. The nature of the reduction in alcohol in U.S. fatal crashes. SAE Paper No. 860038. Warrendale, PA: Society of Automotive Engineers; 1986.
- Fell, J. C.; Nash, C. E. The nature of the alcohol problem in U.S. fatal crashes. *Health Educ. Q.* 16:335-343; 1989.
- Haight, F. A. What causes accidents—a semantic analysis. SAE Paper 80039. Warrendale, PA: Society of Automotive Engineers; 1980. (Also included in *Accident Causation*. SAE Special Publication SP-461; pp. 51-54; 1980).
- Hauge, R. The effects of changes in availability of alcoholic beverages. In: Laurence, M. D.; Snortum, J. R.; Zimring, F. E., eds. *Social control of the drinking driver*. Chicago, IL: The University of Chicago Press; 1988; 169-187.
- Hedlund, J.; Arnold, R.; Cerrelli, E.; Partyka, S.; Hoxie, P.; Skinner, D. An assessment of the 1982 traffic fatality decrease. *Accid. Anal. Prev.* 16:247-261; 1984.
- Hingson, R.; Howland, J.; Morelock, S.; Heeren, T. Legal interventions to reduce drunken driving and related fatalities among youthful drivers. *Alcohol Drugs and Driving* 4:87-98; 1988.
- Howland, J. Social norms and driving countermeasures. In: Graham J. D., ed. *Preventing automobile injury—New findings from evaluation research*. Dover, MA: Auburn Publishing Company; 1988:163-180.
- Hurst, P. M. Epidemiological aspects of alcohol in driver crashes and citations. *J. Safety Res.* 5:130-148; 1973.
- Kahane, C. J. An evaluation of occupant protection in frontal interior impact for unrestrained front seat occupants of cars and light trucks. Report No. DOT HS 807 203. January 1988.
- Klein, T. M. A method for estimating posterior BAC distributions for persons involved in fatal traffic accidents. Document DOT HS 807 094. National Highway Traffic Safety Administration; July 1986.
- McCord, J. Drunken drivers in longitudinal perspective. *J. Stud. Alcohol.* 45:316-320; 1984.
- Moskowitz, H.; Robinson, C. Driving-related skills impairment at low blood alcohol levels. In: Noordzig, P.; Roszbach, R., ed. *Alcohol, Drugs and Traffic Safety—T86*. Amsterdam: Excerpta Medical Elsevier Science Publisher; 1987:79-86.
- National Highway Traffic Safety Administration. Fatal Accident Reporting System 1987. Document DOT HS 807 360. December 1988.
- National Highway Traffic Safety Administration. 19-city safety belt and child safety seat use observation survey. Washington, DC: NHTSA; 1989.
- National Highway Traffic Safety Administration: Alcohol and highway safety 1984: A review of the state of the knowledge. Document DOT-HS-806-569. February 1985.
- Partyka, S. C. Simple models of fatality trends using employment and population data. *Accid. Anal. Prev.* 16:211-222; 1984.
- Phelps, C. E. Alcohol taxes and highway safety. In: Graham J. D., ed. *Preventing automobile injury—New findings from evaluation research*. Dover, MA: Auburn Publishing Company; 1988a: 197-219.
- Phelps, C. E. Death and taxes—an opportunity for substitution. *J. Health Economics* 7:1-24; 1988b.
- Reed, D. S. Reducing the costs of drinking and driving. In: Moore, M. H.; Gerstein, D. R., eds. *Alcohol and public policy—Beyond the shadow of prohibition*. Washington, DC: National Academy Press; 1981:336-387.
- Ross, H. L. Costs, benefits and alternatives: Another look at deterring drinking drivers: Comment on Snortum, Hauge and Berger. *Justice Quarterly* 3:167-169; 1986.
- Ross, H. L. Brewers view of drunk driving: A critique. *Accid. Anal. Prev.* 19:475-477; 1987.
- Ross, H. L. Deterrence-based policies in Britain, Canada, and Australia. In: Laurence, M. D.; Snortum, J. R.; Zimring, F. E., ed. *Social control of the drinking driver*. Chicago, IL: The University of Chicago Press; 1988:64-78.
- Saffer, H.; Grossman, M. Beer taxes, the legal drinking age, and youth motor vehicle fatalities. *Journal of Legal Studies* 26:351-374; 1987.
- Stein, S. K. Risk factors of sober and drunk drivers by time of day. *Alcohol, Drugs and Driving* 5(3):215-227; 1989.

- Stutker, P. B.; Brantley, P. J.; Allain, A. N. MMPI response patterns and alcohol consumption in DUI offenders. *J. Consult. Clin. Psychol.* 48:350-355; 1980.
- U.S. Department of Transportation. 1968 Alcohol and Highway Safety Report, Report from the Secretary of Transportation to the U.S. Congress. Washington, DC: USDOT; 1968.
- Viano, D. C.; Culvert, C. C.; Evans, L.; Frick, M. C.; Scott, R. Involvement of older drivers in multi-vehicle side impact crashes. Proceedings of the 33rd Annual Meeting of the Association for the Advancement of Automotive Medicine, Baltimore, MD, Oct 1-4, 1989. *Accid. Anal. Prev.* 22:177-188; 1990.
- Wagenaar, A. C. Alcohol, young drivers, and traffic accidents: Effects of minimum-age laws. Lexington MA: Lexington Books, D. C. Heath Company; 1983.
- Waller, P. F.; Stewart, J. R.; Hansen, A. R.; Stutts, J. C.; Popkin, C. L.; Rodgman E. A. The potentiating effects of alcohol on driver injury. *JAMA* 256:1461-1466; 1986.
- Walsh, B. M. Do excise taxes save lives? The Irish experience with alcohol taxation. *Accid. Anal. Prev.* 19:433-448; 1987.
- Young, H. D. Statistical treatment of experimental data. New York: McGraw-Hill; 1962.

## APPENDIX

## TWO-VEHICLE CRASH MULTIPLICATIVE MODEL COMPARED TO DATA

Two-vehicle crash effects were modelled by assuming that the number of crashes between vehicles driven by drivers in BAC category  $i$  and drivers in BAC category  $j$  was proportional to  $g(i)R(i)g(j)R(j)$ , and that fatalities were proportional to crash involvements. To further explore the plausibility of this model, two-vehicle crashes were examined directly, using FARS data for the 26 states, with the results shown in Table A1. Each cell shows the number of drivers with BAC in category  $i$  who were killed in crashes with vehicles driven by drivers in BAC category  $j$ , irrespective of whether the other driver was fatally injured. For example, 53 drivers with zero BAC died in crashes with drivers with BAC from 0.05% to 0.099%, whereas 27 drivers with BAC from 0.05% to 0.099% died in crashes with zero BAC drivers. This invites the nominal interpretation that drivers with lower BAC levels were less likely to survive. Indeed, out of the 14 cases that show a difference, 10 indicate that the lower BAC driver was more likely to be killed.

This should not be taken as evidence refuting the findings of Waller and others (1986) and Anderson and Viano (1987) that survivability decreases with increasing BAC, because many factors strongly associated with involvement in two-vehicle crashes are also associated with alcohol use. For example, alcohol use is associated with younger drivers (Wagenaar 1983), who have higher survivability in crashes (Evans 1988a). Vehicles driven by older drivers are more likely to be struck on the side [Viano et al. 1989]; when one vehicle crashes into the side of another, fatality risk is much higher in the side-impacted vehicle. Any estimate of factors influencing survivability in two-vehicle crashes, such as that in Kahane (1988), necessarily involves multivariate considerations requiring much more data than that presented in Table A1. It is because of such considerations that the simple model in the text was used.

Table A2 shows the above data normalized so that each cell gives the percentage of drivers in the indicated cell. Table A3 shows  $g(i)R(i)g(j)R(j)$  values calculated using the  $R$  values from Table 2 and the  $g$  values for two-vehicle crashes from Table 4. The simple model does a reasonable job of reproducing the data, especially for aggregate quantities. For example, the data indicate that 41% of fatally injured drivers with BAC = 0 are killed in crashes with BAC > 0 drivers and 31% of BAC > 0 drivers are killed in crashes with BAC = 0 drivers; the model makes no distinction between these two cases, estimating 34% of each. It is not possible to estimate directly from the data how many of the fatalities are attributable to alcohol without a model and assumptions essentially as complicated and uncertain as those used. The general comparison of the model

Table A1. The number of fatally injured drivers with BAC in category  $i$  (as defined in Tables 1-4) killed in two-vehicle crashes in which the other involved driver had BAC in category  $j$ . From FARS 1987 data for 26 states

		BAC category, $j$ , of other involved driver						All
		1	2	3	4	5	6	
BAC category, $i$ , of fatally injured driver	1	525	33	53	79	94	99	883
	2	33	7	5	6	11	10	72
	3	27	1	4	6	12	14	64
	4	34	2	8	12	4	6	66
	5	47	8	9	5	8	8	85
	6	89	6	14	12	15	12	148
	All	755	57	93	120	144	149	1318

with the data suggests that our calculation more likely underestimates than overestimates the fraction of two-vehicle fatalities attributable to alcohol, and that ignoring differential survivability as a function of BAC is more than compensated for by influences in the opposite direction from driver age distributions and crash types. The advantage of the model is that we can use it to infer the fraction of fatalities attributable to alcohol without additional assumptions.

Table A2. The data in Appendix Table 1 normalized so that each entry gives the percent of fatally injured drivers with BAC in category  $i$  killed in two-vehicle crashes in which the other involved driver had BAC in category  $j$

		BAC category, $j$ , of other involved driver						All
		1	2	3	4	5	6	
BAC category, $i$ , of fatally injured driver	1	39.8	2.5	4.0	6.0	7.1	7.5	67.0
	2	2.5	0.5	0.4	0.5	0.8	0.8	5.5
	3	2.0	0.1	0.3	0.5	0.9	1.1	4.9
	4	2.6	0.2	0.6	0.9	0.3	0.5	5.0
	5	3.6	0.6	0.7	0.4	0.6	0.6	6.4
	6	6.8	0.5	1.1	0.9	1.1	0.9	11.2
	All	57.3	4.3	7.1	9.1	10.9	11.3	100.0

Table A3. Calculated number of fatally injured drivers with BAC in category  $i$  killed in two-vehicle crashes in which the other involved driver had BAC in category  $j$ . Each cell is computed as  $g(i)R(i)g(j)R(j)$  times a normalizing constant which insures that all entries add up to 100 percent

		BAC category, $j$ , of other involved driver						All
		1	2	3	4	5	6	
BAC category, $i$ , of fatally injured driver	1	43.2	2.8	3.4	3.7	4.8	7.8	65.7
	2	2.8	0.2	0.2	0.2	0.3	0.5	4.2
	3	3.4	0.2	0.3	0.3	0.4	0.6	5.2
	4	3.7	0.2	0.3	0.3	0.4	0.7	5.6
	5	4.8	0.3	0.4	0.4	0.5	0.9	7.3
	6	7.8	0.5	0.6	0.7	0.9	1.4	11.8
	All	65.7	4.2	5.2	5.6	7.3	11.8	100.0