# BAC and Fatal Crash Risk

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**Abstract**

Induced exposure, a technique whereby not-at-fault driver crash involvements are used as the denominator in a risk estimate calculation, was used to estimate fatal crash risk by driver BAC. One important advantage of induced exposure is that large existing data sets provide substantial sample size for risk estimation. Risk ratios were calculated for BACs ranging from .01% to .20%. The results indicated that whereas case/control methods suggest an exponential or curvilinear relationship between crash risk and BAC, both induced exposure and laboratory findings suggest a linear relationship.

**Introduction**

Estimation of driver crash risk by specific BAC level has been difficult using existing laboratory and case/control methods. Nevertheless, BAC limits of precisely .02%, .04%, .06% and .08% have been established for various offenses affecting various classes of drivers. The objective of the present study was to estimate fatal crash risk for precise BAC levels using an alternative procedure, induced exposure, then compare the risk estimates obtained to both laboratory and case/control findings.

Moskowitz (2001) summarized laboratory and case/control research as follows: 1) Laboratory studies have found impairment at BACs as low as .01%; 2) Young people are more impaired at low BAC levels than older persons; 3) Excluding the young and the old, and using each subject as his/her own control, laboratory studies indicate that there is no differential alcohol effect as a function of age; 4) Laboratory studies suggest a linear increase in impairment with increasing BAC whereas case/control studies suggest an exponential increase. Each of these findings can be compared to results found using induced exposure risk estimation.

**Induced Exposure**

Induced exposure (see Preusser et al., 1998), is based on the concept that any driver on the road may be the victim in a multiple vehicle crash of some other driver's mistake. These "not at fault" multiple vehicle crash involvements can be used as a surrogate measure of exposure to highway risk. Drivers involved in a single vehicle crash and drivers who made a critical error

leading to a multiple vehicle crash are considered to be "at fault."

In effect, at fault crash involvement becomes the numerator (i.e., measure of risky driving) and not at fault involvement in multiple vehicle crashes becomes the denominator (i.e., measure of exposure). Crash risk can then be calculated in relation to some reference group such as drivers ages 35-49 at .00% BAC (Table 1) or all drivers at .00% BAC (Table 2; after Clayton, 1977).

$$\text{Risk} = \frac{T_f B_{nf}}{T_{nf} B_f}$$

T = number of crash involvements for the target age driver at a specified BAC level (e.g., 16-20 year old drivers at .01% BAC)
B = number of crash involvements for drivers in the base driver group (e.g., ages 35-49 at .00% BAC)
f = at fault involvements
nf= not at fault involvements

One strength of the induced exposure technique is that it requires no assumptions for time of day, road type, vehicle type, type of area or other variables that might be related to high risk or low risk driving situations. Types or groups of drivers who drive more in high risk situations should have proportionately greater opportunity for "induced" exposure than groups of drivers who drive more in low risk situations.

Fatally injured drivers of passenger vehicles were identified in FARS for the years 1987-99. Each driver was categorized as being "at fault" or "not at fault" in the crash. At fault was defined as: being involved in a single vehicle crash; or being assigned one or more driver level factor codes 20 to 59 (i.e., behavioral errors). Passenger vehicles were defined as cars, vans, light trucks and utility vehicles. Drivers of motorcycles, motor homes, farm equipment, buses, medium trucks and heavy trucks were excluded as were drivers for which BAC or age was unknown. Also excluded were crashes involving a pedestrian or bicyclist. The full data set, after these exclusions, contained 192,282 fatally injured drivers of which 33,146 were "not-at-fault."

**Results**

Table 1 shows risk calculations by driver age and BAC. Calculations are normalized to a risk of 1.00 for drivers ages 35-49 at .00% BAC. As shown in the first column, per unit of exposure, young drivers ages 16-20 at .00% BAC are three times more likely to become fatally injured in a motor vehicle crash than drivers ages 35-49 at .00% BAC. Drivers ages 65 and older are two times more likely. Remaining columns show risk for each age group with increasing BAC.

Table 2, first row, shows risk calculations normalized to 1.00 for drivers of all ages at .00% BAC. The comparable risk calculations from Grand Rapids (Borkenstein, 1964 as summarized by Hyman, 1968) are shown in the second row of Table 2.

Table 1
Crash Risk by Age and BAC
(FARS 1987-99, N = 192,282)

| | Blood Alcohol Concentration | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| AGE | 00 | 01 | 02-03 | 04-05 | 06-07 | 08-09 | 10-14 | 15-19 | 20+ |
| 16-20 | 3.31 | 4.37 | 4.12 | 5.44 | 8.17 | 10.10 | 15.77 | 25.30 | 28.19 |
| 21-24 | 1.79 | 2.18 | 2.59 | 4.42 | 6.11 | 8.13 | 10.73 | 16.43 | 26.00 |
| 25-34 | 1.25 | 1.38 | 1.89 | 2.32 | 2.94 | 4.37 | 7.27 | 11.61 | 16.08 |
| 35-49 | 1.00 | 1.09 | 1.49 | 1.78 | 2.62 | 3.56 | 5.64 | 10.44 | 16.99 |
| 50-64 | 1.02 | 0.93 | 1.17 | 1.24 | 2.03 | 2.23 | 4.71 | 8.48 | 13.24 |
| 65 + | 2.04 | 1.97 | 2.49 | 2.50 | 2.50 | 3.55 | 4.83 | 7.48 | 9.48 |

Table 2
Crash Risk by BAC
(FARS 1987-99 - Grand Rapids)

| | Blood Alcohol Concentration | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | 00 | 01 | 02-03 | 04-05 | 06-07 | 08-09 | 10-14 | 15-19 | 20+ |
| FARS | 1.00 | 1.07 | 1.36 | 1.72 | 2.44 | 3.28 | 5.21 | 8.27 | 11.37 |
| G. R. | 1.00 | 0.91 | 0.89 | 1.13 | 1.46 | 1.89 | 5.70 | 17.11 | 23.62 |

1) Impairment can be found at BACs as low as .01%.

Table 1 indicates somewhat increased risk at .01% BAC for all age groups through age 49; decreased risk for age groups above age 49. However, none of the increases or decreases shown in Table 1 for any of the age groups, .00% BAC versus .01% BAC, were statistically significant. At .02% BAC, a statistically significant increase in risk was found for age groups 21-24, 25-34 and 35-49. For 16-20 year-olds the first BAC level to show a statistically significant increase in risk was .03%. For 50-64 year-olds the first BAC level was .06% and for drivers ages 65 and older the first level was .08%.

Table 2 shows that for drivers of all ages fatal injury crash risk was 1.07 at .01% BAC versus 1.00 at .00% BAC. This difference was not statistically significant ($\chi 5 = 1.66$, ns with 1 df). However, at .02% BAC fatal crash risk increased to 1.24. This difference, 1.24 versus 1.00, was statistically significant ($\chi 5 = 14.49$, $p<.001$ with 1 df). Therefore, induced exposure methods indicate a

statistically significant increase in crash risk beginning at .02%.

2) Young people are more impaired at low BAC levels than older persons.

As shown in Table 1, young drivers are clearly at greater risk at lower BACs than older drivers. However, they are also at greater risk at .00% BAC, moderate BAC and high BAC.

3) Excluding the young and the old, and using each subject as his/her own control, there is no differential alcohol effect as a function of age.

This is one of the more interesting findings from the laboratory studies. While each age group may start at a different level, the ratio or multiplicative increase in impairment arising from increasing BAC is similar.

The results shown in Table 1 are consistent with this finding. For all age groups 20 through 64, risk increases by a factor of about five or six at .10% BAC; by a factor of about thirteen for BACs of .20% and above.

This constant relationship by age does not hold for younger or older drivers. For drivers ages 16 through 20, risk starts at 3.31 for .00% BAC. Risk shows multiplicative increases similar to other age groups through .10% BAC; then risk increases more slowly. For drivers ages 65 and older, the increase in risk as a function of BAC is much smaller as compared to other age groups over the entire BAC range.

4) Laboratory studies suggest a linear increase in impairment with increasing BAC whereas epidemiological studies suggest an exponential increase.

Currently, the primary available epidemiological case/control data set is Grand Rapids (Borkenstein et al., 1964). Table 2 compares Grand Rapids with the current study. The results indicate that the Grand Rapids risk estimates are well below the induced exposure estimates for all BAC levels up to .09%. Grand Rapids is approximately equal to induced exposure at .10% BAC then rises rapidly. For BACs of .20% and above, crash risk estimated from Grand Rapids is approximately double the crash risk estimated by induced exposure. The Grand Rapids results clearly suggest a curvilinear relationship between BAC and risk whereas the induced exposure results, consistent with laboratory findings, clearly suggest a linear relationship.

Why do case/control results differ from both induced exposure and the laboratory?

Case/control studies are based on comparisons between crash involved drivers and similarly exposed, yet non-crash involved, controls sampled same time of day, same day of week at the crash location. An inherent bias of this methodology is that any real differences between the crash and non-crash groups will be diminished to the extent that drinking itself is correlated with time of day, day of week and location irrespective of any increased risk due to alcohol. For instance, if the

incidence of drinking correlates 100% with time of day, day of week and location, then it is a logical impossibility to find a crash versus control difference based on the presence or absence of alcohol. Each control subject will be found to have been drinking every time the crash involved driver was drinking; each control subject will be found not to have been drinking every time the crash involved driver was found not to have been drinking. This is true whether the increased risk due to alcohol is 0 percent, 10 percent or 1,000 percent.

Consider, for instance, the following extreme example. There is a dead-end road leading only to a social club and a religious facility. Every driver going to or from the club has been drinking. Every driver going to or from the religious facility has not been drinking. By mutual agreement, the club and the religious facility are never open at the same time of day, day of week. Thus, every time there is a crash involving a driver from the club, that driver will have been drinking as will every control driver sampled to correspond with that driver. Similarly, every time there is a crash involving a driver from the religious facility, that driver will not have been drinking and every control driver sampled to correspond with that driver will not have been drinking. No increase in risk associated with alcohol will be found even though there may be five, twenty-five or fifty crashes involving a driver from the club for every one crash involving a driver from the religious facility. That is, overall risk for drivers who had been drinking will be calculated at 1.00; overall risk for drivers who had not been drinking will be calculated at 1.00.

While the overall risk for the presence or absence of alcohol may be 1.00 in the above example, it is not necessarily true that calculated risk will be 1.00 for every BAC level. The high BAC drivers from the club could be the ones that are crashing while drivers at lower BAC may be found more often in the control group. Still, overall risk must sum to 1.00. Algebraically, then, we would expect risk estimates that are less than 1.00 for low BACs; higher than 1.00 for high BACs; summing to an overall risk of 1.00. Any such downward bias for low BACs, and upward bias for high BACs, in case/control studies would have the effect of creating the appearance of an exponential risk by BAC relationship.

If the true relationship between BAC and risk is linear, then it follows that countermeasures need to be concerned with the full range of BAC levels. It is just as important to lower a BAC level of a drinking driver from .05% to .04% as it is to lower a level from .15% to .14%. Moreover, viewed on a population basis, it may be more important to lower the .05% BACs since there are far more drivers on the road with low as opposed to high BACs.

**Conclusion**

The present results, consistent with laboratory findings, indicate that BACs as low as .02% increase fatal crash risk. Relative increases in crash risk by BAC appear to be consistent across all age groups, young and old excluded. The shape of the BAC by risk function is likely linear, not exponential as suggested by earlier case/control studies. If linear, it becomes even more important to address low BACs in any comprehensive effort to reduce alcohol related crashes.

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