Injury Epidemiology: Fourth Edition

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Chapter 8. THE USE AND ABUSE OF CAUSAL ANALYSIS

On an exam in a course on the use of epidemiology for injury control, I asked the students, "What is wrong with this statement: 'Alcohol is consistently associated with unintentional injury deaths, therefore alcohol abuse must be reduced to lower rates of death and disability due to injury?'". Only one in twenty came close to writing a correct answer. Yet in presentations during the course, numerous instances to the contrary were noted. Motor vehicle injuries, for example, have been reduced by improved vehicle crashworthiness, lighting dark sections of road, and channeling traffic, without reducing alcohol or otherwise changing drivers.

In explaining the answer to the students, I rewrote the statement: "Gender is consistently related to injury; therefore gender must be changed to reduce injury". The nervous laughter, particularly from the gender often at higher risk (males), suggests that some might have got the point.

The statement about alcohol on the exam was not original. Prominent leaders in public health published the statement (Brown, et al., 1990). There is an assumption inherent in much public health literature that specification of complex interaction of factors -- sometimes called causal webs -- is necessary for injury and disease reduction (Krieger, 1994). To the contrary, while the notion of the causal web may be useful to call attention to the complexities of multiple causes of diseases and injuries, it may disable our minds in thinking about prevention (Renwick, 1973).

The "public health model" is usually presented as follows: 1. identify the problem (surveillance), 2. identify risk factors, 3. develop and test interventions, 4. implement interventions and measure effectiveness (e.g., Powell, et al., 1996). In fact, many interventions and therapies have been successful without the second step. One does not need to know the "risk factors" for a headache to use an analgesic. Focusing on multiple "risk factors" in causal webs may lead astray rather than toward injury control.

Consider the New York Health Department's study of children's fatal falls that resulted in barriers over windows in high rise buildings and a huge reduction in child deaths (Chapter 7). What would have happened if the researchers had

attempted an analysis of numerous "risk factors" that could contribute to children falling from windows, such as no adult present, intoxicated adults, numerous factors that distracted adults, and children's' inquisitiveness, hyperactivity or numerous other characteristics? Would there have been attempts to change those factors rather than install window barriers? If so, it is unlikely that the falls would have declined nearly to the extent produced by the window barriers.

Multiple causation of disease and injury, for which the causal web is the most common metaphor, is repeatedly demonstrated. It does not logically follow, however, that multi-factorial theory or analysis leads to rational prevention policy. This was recognized in the original conceptualization of the "causal web" (McMahon, et al., 1960), but it is commonly forgotten. Those of us who study injuries in the U.S. are not fond of metaphors regarding guns, but there were instances in which "magic bullets" were found despite multiple risk factors for infectious diseases (Evans, 1993).

If we have to deal with causal webs, perhaps we need a metaphor for something more effective than bullets against webs and spiders -- like brooms. To Krieger's (1994) provocative question regarding causal webs: "Has anyone seen the spider?" should be added, "Has anyone seen the housekeepers with the preventive brooms?"

Proponents of the search for complex causes before consideration of preventive action argue that, without understanding of causes, prevention may fail because of confounding factors or may have unintended consequences. Confounding refers to the attribution of risk to a factor that has little or no effect but is mistakenly thought to have an effect because it is correlated to a factor that is important.

In rare instances, confounding has misled preventive efforts and adverse "side effects" have also occurred, but the nature of certain types of causes is such that the potential for unintended consequences is minimized. Complete understanding is an elusive goal that is yet to be accomplished for most phenomena. How much, then, do we need to know for injury control?

To eliminate an injury (or anything else that is undesirable), one need only find a controllable necessary condition for the outcome, and control that condition. Even the least resilient of human anatomies can tolerate some mechanical energy load (commonly measured in pounds per square inch or kilograms per centimeter). In the road environment, any combination of energy management by vehicle components or the surrounding environment that keeps loads below that tolerance will eliminate injury. While energy management to that extent has not been accomplished, and may not be feasible in the extreme for economic or other reasons, substantial reduction in motor vehicle death rates occurred due to increased vehicle crashworthiness, increased visibility of side running lights and high mounted brake lights and automatic corrective action by electronic stability control. There are claims of unintended consequences in increased risky driving

by those protected, but these claims are not supported by the better research (Chapters 10 & 11).

Causal analysis of injury can inform preventive approaches when it specifies factors that are substantially changeable and that account for a proportion of a given type of injury, and rules out factors that are spuriously correlated to injury. Attention to the nature of types of causes gives guidance to the extent that a given injury might be reduced if a given factor were changed.

In examining data on potential causes of injuries, it is useful to bear in mind the types of statistical distributions of injuries that are found when conditions are necessary, sufficient or contributing factors. Examples are shown in Tables 8-1 through 8-3 where the designation of "none", "some" and "more" cases refers to a proportion of all cases in the table.

Table 8-1. Distribution of Observations When A Is a Necessary Condition for B

	B	Not B
A	Some	Some
Not A	None	Some

Table 8-2. Distribution of Observations When A Is a Sufficient Condition for B

	B	Not B
A	Some	None
Not A	Some	Some

Table 8-3. Distribution of Observations When A Increases the Probability of B

	B	Not B
A	More	Some
Not A	Some	More

If A is a necessary condition for B, then B will not occur in the absence of A, and the joint distribution of A and B will look like that in Table 8-1 when the measurement of both is accurate. For example, keeping the height of playground equipment below the level that can produce enough energy in a fall to injure the heaviest users will eliminate such injury (Chapter 2).

If A is sufficient for B, then B will always occur in the presence of A, but the absence of A does not imply the absence of B unless A is also necessary for B (Table 8-2). A competently used guillotine will behead anyone placed under it,

but all beheadings are not by guillotine. Occupants of cars that run under trailers of tractor-trailer rigs are sometimes beheaded.

As seen in Table 8-3, if the lower left and upper right cells were both zero, factor A would be both necessary and sufficient for B, but such effects are very rare. In Table 8-3 as shown, A increases the probability of B, and more cases of B are found in the presence of A, but A is neither necessary nor sufficient for B. Some people who drink and drive are not in crashes and many drivers in crashes have not consumed alcohol so alcohol is a contributing factor, neither necessary nor sufficient to cause a crash.

Since prevention of a necessary condition for harm completely reduces the harm, the more closely the distribution of injuries of a given type and severity (B) in relation to the presence of some factor (A) resembles the distribution in Table 8-1, the greater the number of such injuries that will be prevented if A is eliminated.

CRITERIA FOR CAUSATION. The old Henle-Koch criteria for attributing causes to infectious diseases required that a microorganism or other factor be found both necessary and sufficient to produce a given infectious disease, but clearly those criteria were too stringent. Many microorganisms were found necessary but not sufficient for the infection to occur (Kelsey, et al., 1986). Nevertheless, by controlling the microorganism, its access to the host, or host resistance, the disease could be controlled.

Similarly, as noted in Chapter 2, an energy exchange with the human organism is a necessary and specific condition for injury, but the degree of the energy exchange necessary in individual cases may vary by the nature of the tissues affected, which makes them more or less tolerant of energy insults. Nevertheless, if the energy exchange can be kept below the tolerance of the most vulnerable tissue, injury will not occur. Where it is not possible to reduce energy exchanges to that degree, it is nevertheless possible to greatly reduce severity by reducing energy exchanges.

Several criteria must be met to make a strong inference of causation. First, the cause must precede the effect in time. For example, a study of alcohol measured in the breath of emergency room patients found that some claimed to have consumed alcohol after the injury (Wechsler, et al., 1969). In those cases, if true, alcohol could have not contributed to the incidence, although it might affect recovery.

Second, the hypothesized cause must be correlated to the effect, that is, they must have joint distributions similar to one of those in Tables 8-1 through 8-3, or in the case of non-categorical variables such as blood alcohol concentration, there should be a dose-response correlation. The cliché among some scientists that "correlation doesn't mean causation" is not precise. Correlation is a necessary but not sufficient condition for inference of causation.

The absence of correlation does not totally rule out causation if measurement is unreliable, biased, invalid or other factors intervene in such a way as to mask the correlation. Occasionally claims of lack of correlation are disproved when better research designs controlling for other factors are used (e.g., Zador, et al., 1984). In other cases, the effect may be under or overestimated for lack of control of relevant factors. For example, the National Highway Traffic Safety Administration estimated that placement of fuel tanks in front of rear axles of passenger cars reduced fatal rear impact fires by 29 percent (Tessmer, 1994). The analysis did not account for the fact that cars were being reduced in size during the period that gas tanks were being relocated. When vehicle size and tank location were considered simultaneously in a multivariate analysis, forward located tanks were found to reduce rear fire fatalities by more than half (Robertson, 1993).

Third, the correlation must be demonstrated as large enough, given the numbers of observations in the sample, which it is unlikely to have occurred from random fluctuations in drawing samples of that size. If the sample size is too small, however, a causal connection may be falsely rejected. Statistical power is a function of sample size. Studies often lack a large enough sample to detect magnitudes of correlation that would have practical use if detected. One study of prominent medical journals, such as the Journal of the American Medical Association, found that 70 percent of articles with statistically insignificant results did not discuss the issue of statistical power, that is, the number of cases studied was often not large enough to detect important differences beyond chance variation (Hebert, et al. 2002). Textbooks in statistics contain criteria for sampling and tests for random fluctuation in samples (e.g., Armitage, 1971, Selvin, 1991). Computer programs such as EPIINFO, used for data entry and statistical calculations, include features for estimating statistical power before a study is undertaken (e.g., Dean, et al., 1994).

Fourth, the research design must be adequate to rule out co-variation between the hypothesized cause and other factors that could explain the same variation, or specify how such co-variation represents a causal sequence. This is the previously mentioned problem of "confounding". Research projects that demonstrate a nonrandom correlation between risk factor A and injury B are often criticized by saying: "You didn't control for X." The criticism may be legitimate if X could reasonably be expected to affect both A and B strongly enough to account for their correlation, but some such criticisms have no basis in terms of plausible causal mechanisms or potential magnitude of the effect.

Fifth, the mechanism of the causation should be plausible in terms of what is known about the phenomena in the relevant discipline. Occasionally the theories of physics, chemistry, biology or behavior are modified by some seemingly implausible research finding, but often such a finding is subsequently shown to be the result of faulty research methodology. See Appendix 8-1 for an analysis of

an implausible claim of the cause of a divergence in trends of motor vehicle fatality rates among countries.

CAUSAL MODELS. The more plausible hypotheses are those that are deduced from what is known. Some important discoveries occur as a result of hypotheses based on hunches, observations of a few cases in a clinic or morgue, and the like, but the odds of finding something useful are greater when the hypothesis to be tested provides a plausible link in a causal model.

In the case of injury, a hypothesis is more plausible if the proposed cause has a likely connection to the concentration of energy, an energy exchange with tissue, or the vulnerability of tissue. If the hypothesized cause could not directly affect one or more of these factors, it is likely to be a relatively weak and indirect contributor to the incidence or severity of injury. To aid thinking about the research that is needed to fill gaps in knowledge, a diagram of the hypothesized causal paths of a given set of injuries is often useful.

For example, it is well known that age is correlated to severe motor vehicle injuries, but age is merely a measure of how often one has circled the sun. While age indicates differential risk that can be used to target injury control efforts at overinvolved age groups such as middle-aged opioid users, it is not a cause of injury. Age is an inexact proxy measure of human limitations and behaviors, including exposure to more or less risky energy sources (knowingly or unknowingly), which result in concentration of energy or energy exchanges, and physical conditions that affect tissue vulnerability.

Correlating such factors as age and gender to injury rates has been called "black box" epidemiology (Susser and Susser, 1996). Without precise knowledge of the exposures to energy and behaviors in the presence of those exposures, by age and gender, the causal mechanisms involved are lumped in a box, the contents of which are unknown.

Figure 8-1 illustrates some possible paths of causation whereby correlates of age may contribute to the necessary and specific causes of motor-vehicle-occupant injury or severity. The direction of the arrows represents the known or hypothesized direction of effect of a given factor on another.

Energy generated by speed and mass interacts with vulnerable tissue in a crash, exacerbated by insufficient room for vehicle occupants to decelerate as indicated by vehicle size. The power of an engine, measured as horsepower in given increments, is necessary for given increments in speed. Vehicle size and horsepower accounted for about 55 percent of the variation in occupant fatalities per 100,000 passenger cars of particular makes and models in the 1980s (Robertson, 1991). Observations of age, gender and speed of drivers at sites where vehicles rolled over indicated younger drivers were driving faster than the average speed at the sites, but there was no correlation of speed and gender. Age, however, was not correlated with rollover and speed was not correlated to

vehicle stability. Therefore, speeding or young drivers did not confound the effect of vehicle stability on rollover (Robertson and Maloney, 1997).

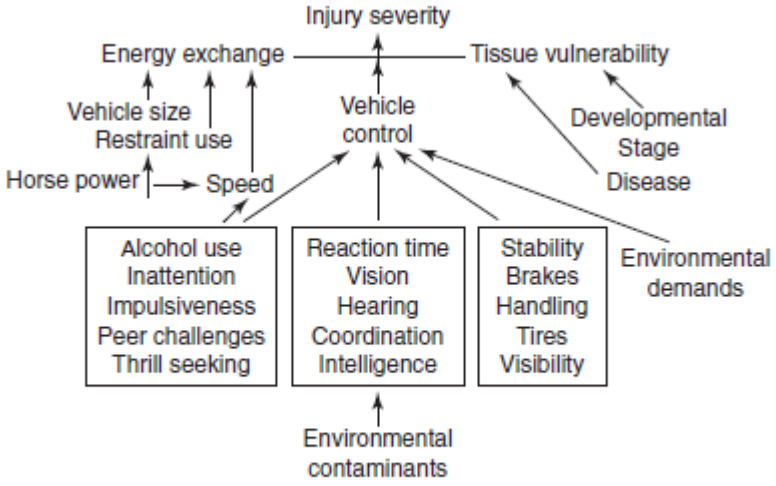

Figure 8-1. Causal Model of Motor Vehicle Injury

Loss of control of one or more vehicles usually precedes a crash. Loss of control is affected by vehicle stability, steering characteristics, braking capacity and tire characteristics in interaction with inputs from the driver -- speed, steering, braking. Vehicle stability accounts for about 62 percent of the variation in fatal rollover crashes per 100,000 utility vehicles, independent of other major known risk factors. These vehicles have stability less than the vast majority of cars (Robertson, 1989). Automatic Stability Control Systems reduced fatal crashes generally, including rollovers, but the effect of low stability still accounts for a significant proportion controlling for the effects of other factors (Appendix 9-1).

Inputs to the vehicle by the driver are known to be altered by impairment or other effects of alcohol and other drugs, and variations in human limitations such as reaction times (time from signal of need to change inputs to actual behavior), vision, hearing, intelligence and coordination of senses and motor function. The effect of each is not known precisely and is more or less contingent on the demands of the driving environment. Therefore, the extent of reductions in injuries that could be achieved by changing the factors that are changeable is in more or less doubt for each one. The other listed behavioral factors may affect speed, reaction time, steering or braking, and degree of vehicle use, but how often is largely unknown. Factors related to age may also affect tissue vulnerability including developmental stages, alcohol or other drugs, and certain diseases.

As depicted in Figure 8-1, the effects of given variables are straightforward in causal chains, but some of the links among factors may be more complicated. While the effect of alcohol is usually referred to as impairment, there are several aspects of the correlation of alcohol and injuries that suggest a more complex

causal pattern. Alcohol in drivers in motor vehicle crashes is more correlated to injury severity than to incidence (Haddon, et al., 1968). Alcohol is found more often in the victims of assault and homicide than in drivers killed in motor vehicle crashes (Wechsler, et al., 1969; Baker et al., 1971). Therefore, it is unlikely that the effect of alcohol is just to impair performance or make tissue more vulnerable. Based on these and other findings, a more elaborate model of alcohol use and its effects has been suggested (Robertson, 1983), as displayed in Figure 8-2.

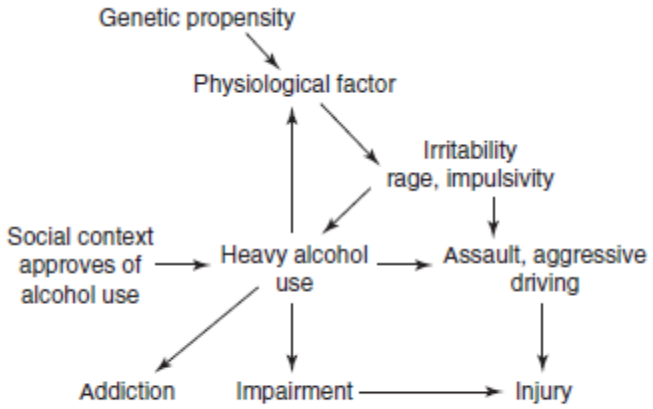

Figure 8-2. A General Theory of Alcohol Use and Effects

In biological and other systems, the system goes out of control when there is positive feedback, that is, factor A increases factor B which, in turn, increases factor A directly or through its effect on other factors. In the model, alcohol use is hypothesized as partly a consequence of inherited biological factors that affect emotions that may then contribute to increase in drinking in an uncontrolled feedback. If such feedback occurs, the individual effects in the loop do not have to be large to have an enormous effect in a few iterations. For example, if B=1.02A, C=1.03B and A=1.02C, the three factors will about double in 10 cycles and about quadruple in 20 cycles.

If the alcohol model is supported by research evidence, then elimination of alcohol might not eliminate all of the injuries with which it is associated. The emotions that contributed to drinking may also contribute to the behaviors leading to injuries and would be present in the absence of alcohol, albeit to a lesser degree (McClelland, 1984). Chapter 15 includes an analysis that indicates that removal of all alcohol from drivers would reduce fatal crashes about half the amount presumed from data on alcohol "involvement". Too often, the human mind converts "involvement" to "caused".

This dynamic model of the causes and consequences of alcohol use is more comprehensive regarding the possible reasons for the involvement of alcohol in assaults as well as other injuries, and for eventual alcohol addiction. It suggests that if the physiological factor possibly intervening between the genes and

increased use can be identified and controlled, a variety of problems would be alleviated (Kapoor, et al. (2013).

Feedback is apparently a factor in violence ranging from interpersonal disputes to war. From the Hatfield and McCoy family feuds to youth in inner cities in the U.S. today, violence led to more violence through retaliation for real or perceived wrongs. Throughout much of the world, violence of members of one ethnic group against another is often claimed to be justified by past wrongs (McConnell, et al, 2014). And President George W. Bush pushed the invasion of Iraq based at least in part on the allegation that Saddam Hussain tried to have Bush's father murdered. In a U.S. city, retaliatory homicide was found significantly more often in gang and drug killings but not so in those altercations among friends, family, strangers or those in which alcohol was involved (Kubrin and Weitzer, 2003).

These are relatively simple examples of causal models. Numerous variations have been suggested just for driver factors in motor vehicle injury (Michon, 1985). The value of a model is not necessarily its complexity or completeness, but whether or not it suggests testable hypotheses regarding variables that have a major influence, particularly variables that are controllable for injury prevention or severity reduction.

Appendix 8-1. False Inference of Causation (Adapted from Robertson, 2006)

A retired General Motors employee, Leonard Evans, published a book in which trends in motor vehicle death rates in the U.S. were compared to other countries (Evans, 2004). The decline in U.S. rates slowed in the 1990s compared to those of several other industrialized countries. As a result, the U.S. no longer holds its historically leading position -- the lowest rate. Evans said the trends indicated a "Dramatic Failure of U.S. Safety Policy". His "analysis" was confined to eyeballing the trends and assertion regarding the cause. The changed slope in U.S. rates he attributed to personal injury lawyers who, he said, have an interest in focusing on vehicles to the neglect of programs to change driver behavior. He said that the emphasis on vehicle factors that resulted in the Motor Vehicle Safety Act of 1966 was the beginning of the problem and singled out the airbag controversy of the 1970s and 1980s as a subsequent major culprit.

Actually, the Motor Vehicle Act of 1966 was inspired by a book co-edited by a physician and two social scientists, who had no interest in injury lawsuits (Haddon, et al., 1964). That book emphasized that the energy in car crashes (and other injurious events) could be managed by product and environmental modifications to reduce injury severity. Based on that analysis, Senator Abraham Ribicoff began hearings on vehicle manufacturer responsibility to improve vehicle safety. When private detectives hired by General Motors were caught trying to entrap Ralph Nader in a scandal, and GM's Chairman apologized in a Senate hearing, the issue gained wider public attention (McCarry, 1972). Whether

the Motor Vehicle Safety Act would have been enacted without that incident is problematic but it was a positive step to reduce vehicle fatalities. The initial regulations substantially improved the crashworthiness of passenger cars. The manufacturers subsequently improved crashworthiness, possibly in response to publicized crash tests (Robertson, 1996).

Evans says that Joan Claybrook (lawyer) in the Carter Administration, egged on by Ralph Nader (lawyer) imposed the "air bag mandate" in the U.S. In fact, the only air bag mandate was introduced by non-lawyers Douglas Toms, head of the National Highway Traffic Safety Administration (NHTSA) and a former state motor vehicle administrator, and John Volpe, Secretary of Transportation, a former owner of a construction company, in the Nixon administration prior to the Carter Administration. Because their mandate was not a performance standard as required by U.S. law, the standard was later revised to require minimum forces on crash dummies in frontal impacts at 30 miles per hour, which is not an "air bag mandate". The Reagan administration tried to overturn the standard but was overruled by the courts as a result of a lawsuit by insurance companies and others, not personal injury lawyers. Automakers chose to use air bags to meet the standard but were not compelled to do so if they had chosen to design the vehicles differently.

Even more bizarre than Evans' false assertions were the favorable reviews of Evans' book in leading medical and other journals. The reviewer in the Journal of the American Medical Association called the chapter on U.S. policy failure a "show stopper" and devoted most of the review to an uncritical repetition of Evans' allegations regarding the history of vehicle regulation (Eisenberg, 2005).

So what are the available data that Evans failed to analyze? One prominent trend in U.S. vehicle sales in the 1990s was the increase in large "sport utility vehicles" (SUVs) built on pickup truck frames. (Readers outside the U.S. unfamiliar with SUVs may see pictures by typing SUV in any Internet search engine). Vehicles with center of gravity too high relative to their width have excessive rollover death rates (Robertson and Kelley, 1989). Stiff frames in certain SUVs, pickup trucks and vans are also a factor in increased risk (Gabler and Hollowell, 1996). Evans mentioned a possible effect of SUVs but said they could not make a difference of more than "a few thousand" deaths per year, "up or down". A few thousand deaths per year affect the rankings among countries substantially. He did not mention the fact that pickup trucks with high centers of gravity also increased in sales. It has long been known that vehicles with their weights distributed higher off the ground contribute disproportionately to the deaths of occupants of other vehicles, as well as pedestrians and bicyclists, the latter probably because of longer braking distances of heavier vehicles (Robertson and Baker, 1976).

Using data from the countries that Evan's claimed superior in safety policy to the U.S., trends in the sales of trucks were examined to see if those countries had a similar increase in truck use compared to the U.S. (Binder, 1990-2003). The

major separation in the rates of the U.S. and other countries occurred in the 1990s. Figure 8-3 illustrates the change in truck sales during that period. In the U.S., trucks and SUVs were 25 percent of sales in 1991 and steadily increased to more than 40 percent of sales in 2002. None of the other countries experienced a parallel increase. In Japan, the percent of trucks sales declined substantially but the trend was relatively flat in most countries. Trends among other European countries mentioned by Evans (Denmark, Finland, Luxembourg and the Netherlands) were similar to those of their nearby neighbors shown here. The Canadian percentage started lower but rose similar to that of the U.S. until 1998 and then declined. These trends led to substantial differences in vehicle mix among the countries.

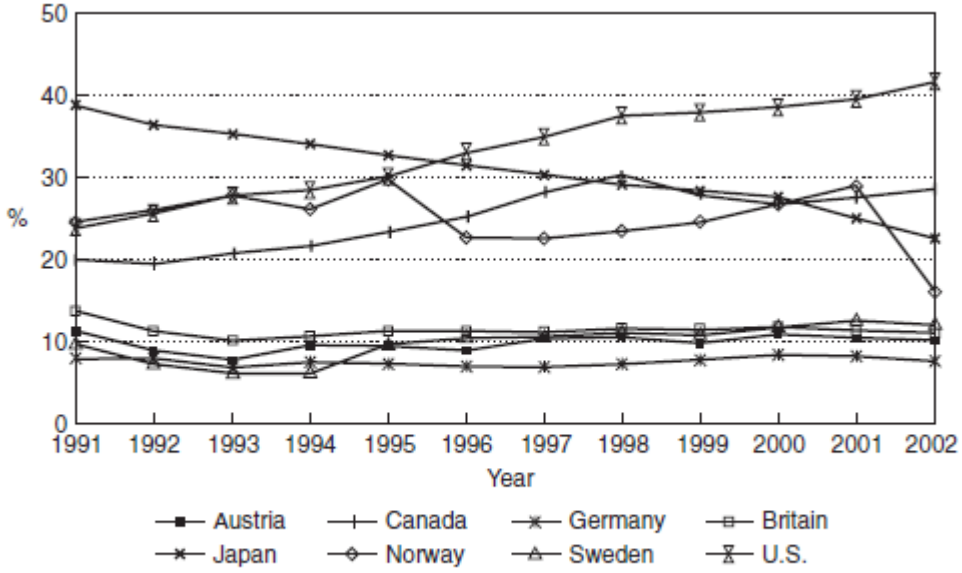

Figure 8-3. Truck Sales 1991–2002—Selected Countries

Of course, we cannot say with certainty that trends in truck sales explain the differences in fatality trends among countries without additional data but the fact that trucks have a higher death rate than other vehicles and truck sales increased in the U.S. disproportionately favors that explanation over the "lawsuit" explanation, which has no plausible causal nexus.

More convincing data is available based on Canadian vehicle sales. Evans gives special emphasis to the decline in Canadian death rates because, he claims, Canada has the same mix of vehicles and drivers as the U.S. The Canadian motor vehicle death rate per vehicles in use declined 63 percent from 1979 to 2002, similar to other industrialized countries, compared to a reduction of 46 percent in the U.S. rate.

Canada does not keep records of the makes and models of vehicles in fatal crashes but the data from death rates for each make and model in the U.S. can be applied to the same make-models in use in Canada to calculate an expected

death rate, adjusted for average miles traveled per vehicle. The following steps were undertaken to obtain an expected death rate for Canada:

1. Count the number of deaths of each make and model in use in the U.S. and divide by the number in use during 2001-2002.
2. Multiply the death rate for each make and model in the U.S. times the number of each make and model in use in Canada during the same period to obtain expected deaths for each make and model in Canada.
3. Sum the number of expected deaths across makes and models and divide the total by the total number of vehicles in use in Canada. Multiply the expected rate per 100,000 vehicles by the ratio of average annual miles per vehicle in Canada to average annual miles in the U.S.

The fatalities in the U.S. by make and model of vehicle in the years 2001-2002 were obtained from the National Highway Traffic Safety Administration's Fatal Analysis Reporting System. Only 1991 and later models were included. All deaths in a fatal crash in which a given vehicle was involved were counted. In collisions of two or more vehicles, only the occupants who died in each vehicle were counted for that vehicle to avoid double counting. Vehicles in use in the U.S. and Canada were estimated by tabulating the annual sales by make and model in each country during 1991-2002 and subtracting the number scrapped before 2002, using published data on vehicles remaining after a given number of years of use for cars and trucks separately in each country (Binder, 1990-2003).

Table 8-4. Percent Vehicles in Use in the United States and Canada and U.S. Fatality Rates: 2001–2002

	Percent Use		Total Deaths/100,000 Vehicles	Occupant Deaths/100,000 Vehicles
	U.S.	Canada		
Cars <3,000 lbs	30.4	38.8	18.2	16.5
Cars ≥3,000 lbs	25.9	17.5	16.9	14.0
Vans	9.6	16.5	13.4	9.7
SUVs	12.8	8.4	19.9	14.4
Pickup trucks	18.0	16.3	22.7	15.0
Motorcycles (single vehicle)	3.1	2.4	33.4	33.4

Differences in types of vehicles in use in 2001-2002 between the U.S. and Canada are shown in Table 8-4, based on examination of specific make-model sales from 1991-2002, discounted for scrapped vehicles. U.S. drivers used proportionately larger cars that had somewhat lower death rates than smaller cars while Canadian drivers disproportionately used vans – the vehicles with the lowest aggregate death rates. U.S. drivers used proportionately more SUVs and pickup trucks that have higher total death rates mainly because of their higher involvement in deaths to road users other than their occupants. The higher

weights of trucks and SUVs result in longer stopping distances, contributing to increased deaths of pedestrians, bicyclists and motorcyclists. Motorcycles are also used more in the U.S. Even with deaths to motorcycle riders counted in the total rate of other vehicles that collided with motorcycles, the single-vehicle death rate of motorcycles is higher than the total death rate of the other classes of vehicles.

U.S. drivers drove an average 12,655 miles in 2002 compared to 10,733 miles driven by Canadian drivers. Applying the U.S. rates for each make-model to the numbers of vehicles of the same make model in use in Canada, and correcting for mileage differences, results in an expected death rate in Canada of 15.9 per 100,000 vehicles compared to 18.9 per 100,000 in the U.S. That difference is exactly the difference in the total death rates between the countries in 2002.

Contrary to Evan's assertion, the U.S. and Canada did not have the same mix of vehicles. The difference in death rates between Canada and the U.S. is predicted by the difference in vehicle mix and miles driven between the two countries. The results suggest that if Canadian drivers had driven the same mix of vehicles the same miles per vehicle as U.S. drivers, they would have the same total death involvement rate as U.S. drivers.

One of the possible reasons that Canadians drive less is a higher gasoline tax. Gasoline taxes in the U.S. are the lowest among the industrialized countries (Perry, 2002). The attenuation in the decline in U.S. fatality rates in the 1990s was at least partly attributable to increased miles driven due to the decline in gasoline prices, adjusted for inflation. Controlling for various state laws and economic factors, "a 10 percent decrease in the real gasoline price is associated with a 1.6 percent increase in fatal crashes per capita" (Grabowski and Morrissey, 2004).

There is no evidence for Evans' claim that the differences between the U.S. and Canada resulted from too little emphasis on behavioral factors in the U.S. Indeed, the U.S. federal government spends large amounts on such programs. According to officials at Transport Canada, there is no Canadian federal government expenditure on behavior programs. In addition to the Motor Vehicle Safety Act, in 1966 the U.S. Congress enacted the Highway Safety Act that provides grants to the states for safety programs. Study of the early effects of such grants found an adverse effect on state motor vehicle fatality rates of high school driver education expenditures but a favorable effect of other programs (Robertson, 1984). Driver education is no longer federally funded. The grants more than doubled in the 1990s. From 1998 to 2002, the grants to the states were incremented from $236.1 million to $556.8 million – targeted substantially toward alcohol abuse, seat belt use and child restraint use. U.S. states and Canadian Provinces may have spent additional funds but there was certainly no paucity of attention to behavioral factors in the U.S.

References – Chapter 8

Armitage P (1971) Statistical Methods in Medical Research. New York: John Wiley and Sons.

Baker SP, Robertson LS, and Spitz WU (1971) Tatoos, alcohol, and violent death. J Forensic Sci 16:219-225.

Binder AK (ed.) (1990-2003) Automotive Yearbook; Southfield, MI: Ward's Communications.

Brown ST, Foege WH, Bender TR and Axnick N (1990) Injury prevention and control: prospects for the 1990s. In Breslow L, Fielding JE, and Lave LB (eds) Ann Rev Pub Health 11:251-266.

Dean AD, Dean JA, Coulombier D, Burton JH, et al. (1994) Epi Info Version 6: A Word Processing, Database, and Statistics Program for Epidemiology on Micro-computers. Atlanta, GA: Centers for Disease Control. The latest version is free at: http://wwwn.cdc.gov/epiinfo/

Eisenberg D. (2005) Review of "Traffic Safety" JAMA 294:746-747.

Evans AS (1993) Causation and Disease: A Chronological Journey. New York: Plenum Publishing Co.

Evans L. (2004) Traffic Safety. Bloomfield Hills, MI: Science Serving Society.

Gabler, HC and Hollowell WT. (1996) Aggressivity of light trucks and vans in traffic crashes. Society of Automotive Engineers Paper Number 980908.

Grabowski DC, Morrisey MA. (2004) Gasoline prices and motor vehicle fatalities. J Policy Anal and Management, 23:575–593.

Haddon W Jr, Kelley AB and Waller JA (anonymously) (1968)1968 Alcohol and Highway Safety Report. Washington, DC: U.S. Government Printing Office.

Haddon W Jr., Suchman EA, Klein D. (1964) Accident Research: Methods and Approaches. New York: Harper and Row. Free online at: http://www.mvhap.org/noteworthy_doc.php,

Hebert RS, Wright SM, Dittus RS and Elasy TA (2002) Prominent medical journals often provide insufficient information to assess the validity of studies with negative results. J Negat Results Biomed. 2002;1:1. Free online at: http://www.jnrbm.com/content/1/1/1

Joudis M, et al. (2014) A comparison and non-domestic homicides: Further evidence for distinct dynamics and heterogeneity of domestic homicide perpetrators. J Fam Vio 29:299-313.

Kaplan MS, McFarland BH and Huguet N (2009) Characteristics of male and female firearm suicide decedents: Findings from the National Violent Death Reporting System. Inj Prev 15:322-327.

Kapoor M et al. (2013) A meta-analysis of two genome-wide association studies to identify novel loci for maximum number of alcoholic drinks. Human Genet 132:1141-1151. Free online at: http://europepmc.org/articles/pmc3776011.

Kelsey JL, Thompson WD and Evans AS (1986) Methods in Observational Epidemiology. New York: Oxford University Press.

Krieger N (1994) Epidemiology and the web of causation: has anyone seen the spider? Soc Sci Med 39:887-903. Free online at: http://havenscenter.wisc.edu/files/krieger1.pdf

Kubrin CE and Weitzer R. (2003) Retaliatory homicide: concentrated disadvantage and neighborhood culture. Soc Problems 50:157-180).

McCarry C. (1972) Citizen Nader. New York: Saturday Press Review.

McClelland DC (1984) Drinking as a response to power needs in men. In McClelland, D.C. Motives, Personality, and Society. New York: Praeger.

McConnell F. Megoran N and Williams P. (eds.) (2014) Geographies of Peace. London: I.B. Tauris. Sections free online at: http://books.google.com/books?hl=en&lr=&id=HjLkAwAAQBAJ&oi=fnd&pg=PA194&dq=retaliatory+violence&ots=YwgOjr0FuB&sig=1I0QGwipujgxrnQ2K5zZYOrl69U#v=onepage&q=retaliatory%20violence&f=false

McMahon B, Pugh TF and Ipsen J (1960) Epidemiologic methods. New York: Little Brown and Co.

Michon JA (1985) A critical view of driver behavior models: what do we know: what should we do? In Evans L and Schwing RC (eds) Human Behavior and Traffic Safety. New York: Plenum.

Parry IWH (2002) Is gasoline undertaxed in the United States? Resources 148:29-33. Free online at: http://www.rff.org/RFF/Documents/RFF-Resources-148-gasoline.pdf

Powell EC, Sheehan KM, and Christoffel KK (1996) Firearm violence among youth: public health strategies for prevention. Ann Emergency Med. 28:204-212.

Renwick JH (1973) Analysis of cause -- long cut to prevention. Nature 246:114-115.

Robertson LS (1983) Injuries: Causes, Control Strategies and Public Policy. Lexington, MA: DC Heath.

Robertson LS. (1984) Federal funds and state motor vehicle deaths. J Pub Heal Policy 5:376-386.

Robertson LS (1989) Risk of fatal rollover in utility vehicles relative to static stability. Am J Pub Heal 79:300-303. Free online at: http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1349551/pdf/amjph00229-0046.pdf

Robertson LS (1991) How to save fuel and reduce injuries in automobiles. J Trauma, 31:107-109.

Robertson LS (1993) Fatal fires from rear-end crashes: the effects of fuel tank placement before and after regulation. Am J Pub Health. 83:1168-1170. Free online at: http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1695178/pdf/amjph00532-0098.pdf

Robertson LS. (1996) Reducing death on the road: the effects of minimum safety standards, publicized crash tests, seat belts and alcohol. Am J Pub Heal 86:31-

34. Free online at:
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1380356/pdf/amjph00512-0033.pdf
Robertson LS. (2006) Motor Vehicle Deaths: Failed Policy Analysis and Neglected Policy. J Pub Health Policy 27:182-189.
Robertson LS, Baker SP. (1976) Motor vehicle sizes in 1440 fatal crashes. Acc Anal Prevent 8:167-175.
Robertson LS and Kelley AB.(1989) Static stability as a predictor of overturn in fatal vehicle crashes. J Trauma.29:313-319.
Robertson LS, Maloney A (1997) Motor vehicle rollover and static stability: an exposure study. Am. J. Pub. Health, 87:839-841, 1997. Free online at:
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1381060/pdf/amjph00504-0129.pdf
Robinson WS (1950) Ecological correlation and the behavior of individuals. Am Soc Rev 15:351-357. Free online at:
http://ije.oxfordjournals.org/content/38/2/337.full
Selvin S (1991) Statistical Analysis of Epidemiologic Data. New York:Oxford University Press.
Susser M and Susser E (1996) Choosing a future for epidemiology: I. eras and paradigms. Am J Pub Health 86:668-673. Free online at:
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1380474/pdf/amjph00516-0062.pdf
Tessmer J (1994) An analysis of fires in passenger cars, light trucks and vans. Washington, DC: National Highway Traffic Safety Administration.
Wechsler H, Kasey EH, Thum D and Demone HW Jr (1969) Alcohol level and home accidents. Pub Health Rep 84:1043-1050. Free online at:
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2031532/pdf/pubhealthreporig01072-0021.pdf
Zador PL, Jones IS and Ginsburg M (1984) Fatal front-to-front car collisions and the results of 35 mph frontal barrier impacts. 28th Ann Proc Am Assoc Auto Med.