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SHOULD COMPENSATION SCHEMES BE BASED ON THE PROBABILITY OF CAUSATION OR EXPECTED YEARS OF LIFE LOST?

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INTRODUCTION

The purpose of this essay is to give a succinct and accessible summary of why I believe that compensation in tort suits should be based not on probability of causation but rather on years (or quality-years) of life lost.[1] I only consider cases in which the plaintiff claims that a hazardous exposure some years in the past is the cause of a current medical illness, such as cancer or heart disease. As I understand it, current legal standards hold that a judgment should favor the plaintiff if and only if "it is more probable than not" that the exposure "causally contributed to" or "was a substantial contributing factor" to her disease.[2] This standard is often expressed as the requirement that the "probability of causation" exceed ½, where the "probability of causation" (PC) is the probability that the exposure causally contributed to the development of the individual's disease. It has been suggested that

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[1] For complete scientific, mathematical, and philosophical arguments for this position, see Sander Greenland & James M. Robins, *Epidemiology, Justice, and the Probability of Causation*, 40 JURIMETRICS 321 (1999); James M. Robins & Sander Greenland, *Estimability and Estimation of Expected Years of Life Lost Due to a Hazardous Exposure*, 10 STAT. MED. 79 (1991); James M. Robins & Sander Greenland, *The Probability of Causation under Stochastic Model for Individual Risk*, 45 BIOMETRICS 1125 (1989).

[2] L.A. Bailey et. al., *Reference Guide on Epidemiology*, *in* REFERENCE MANUAL ON SCIENTIFIC EVIDENCE 168-69 (FED. JUDICIAL CTR. ED. (1994).

this standard can be operationalized via the formula

PC= (RR-1)/RR

(formula (1)), where RR is the rate ratio (equivalently, relative risk) calculated from epidemiologic data comparing the disease rate in a cohort of individuals exposed to the hazard to that in an unexposed cohort. If one plugs a value of 2 for the rate ratio into formula (1) one obtains:

PC=(2-1)/2=1/2

On the basis of this calculation, it has been argued that whenever the rate of disease in the exposed cohort is less than twice the rate in the unexposed (so the rate ratio is less than two), PC must be less than ½ and thus the tort claim is without merit.

I. IS THE PROBABILITY OF CAUSATION ESTIMABLE FROM EPIDEMIOLOGIC DATA

But is formula (1) correct? For if not, the above argument fails. I will now show that formula (1) may be incorrect even if the rate ratio is based on data from an ideal epidemiologic study with an enormous sample. Specifically we will suppose we have data from a (hypothetical) very large well conducted randomized trial. A very large well-conducted randomized trial represents an ideal epidemiologic study because, in a large study, randomization guarantees that there is no confounding by unrecorded common causes of the exposure and disease: exposure is only determined by the flip of a coin and a coin flip does not cause disease. Indeed, in a large randomized study of 2n subjects, one can view randomization as producing n pairs of carbon copies with respect to the disease outcome of interest, with exactly one copy in each pair being exposed and the other being unexposed. That is, if one member of the pair would get the disease at age 50 when exposed, and at age 60 when unexposed, then the same is true for her carbon copy. This statement is more than mere metaphor. Specifically, it is a mathematical theorem that this statement becomes closer and closer to being precisely correct as the size of the trial increases, and furthermore any logical conclusion one draws concerning the results of a large randomized trial under the assumption that each exposed person has an exact unexposed carbon copy (with respect

to disease outcome) will be a valid conclusion. This theorem gives readers without advanced mathematical training a license to use the carbon copy assumption as a walking stick or crutch (as needs be) to aid them in analyzing a large trial.

Thus armed with our crutch, let us analyze a trial. Table 1 presents the data on a representative subset of size 10 from a hypothetical large randomized trial in which at age 20, 500,000 subjects are randomized to the purported hazardous exposure and five hundred thousand subjects remain unexposed. To keep matters simple the disease outcome is death from any cause and all subjects are followed to extinction. In Table 1 the 5 exposed subjects die at ages 40, 50, 60, 70, and 80 while the five unexposed die at ages 50, 60, 70, 80, and 90. We will suppose the data on the total 1,000,000 subjects is just 100,000 copies of Table 1, in order to spare us keeping track of 1,000,000 different individuals. The rate ratio in Table 1 is by definition the death rate in the exposed divided by the death rate in the unexposed. To calculate the death rate in the exposed, we divided the number of deaths in the exposed (5) by the total number of years lived by exposed subjects (beginning at age 20 when the study began). Thus the rate in the exposed is 5/(20 +30 + 40 + 50 + 60)= 5/200 deaths per year and the rate in the unexposed is 5/(30 + 40 +50+60+70) = 5/250 deaths per year. Thus the overall rate ratio is 5/200 divided by 5/250 which is 1.25. Thus if we use formula (1) to calculate the probability of causation, we obtain PC = (1.25-1)/1.25 = 0.2 = 1/5 which is less than ½ and thus no compensation would be awarded to the plaintiff.

TABLE 1—AGE AT DEATH FOR 10 STUDY SUBJECTS

	Age in years								Death rate in deaths/year
	20	30	40	50	60	70	80	90	
5 exposed subjects			1	1	1	1	1		5/200
5 unexposed subjects				1	1	1	1	1	5/250

$$RR = \frac{5/200}{5/250} = 1.25 \qquad RR = \frac{(1.25-1)}{1.25} = 0.2$$

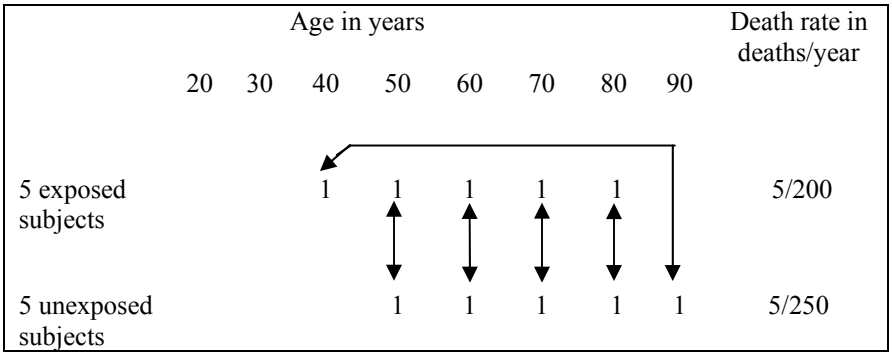
Let us now see if formula (1) gave the correct answer. To do so we will use the carbon copy assumption but before doing so we must think a bit more about just what the assumption entails. Consider a given exposed subject, Helen, who is observed to die at age 60. Helen would have died at some unknown age x if she had been randomized to non-exposure. Under the carbon copy assumption we are assured that, among the unexposed subjects in Table 1, there is a carbon copy for Helen who dies at exactly the age x that Helen would have died if unexposed. But, since we do not know x, we cannot determine which of the unexposed persons is Helen's carbon copy. We conclude that although we may assume each exposed person has an unexposed carbon copy, nonetheless the data collected in the trial provides absolutely no information as to which unexposed person is the carbon copy of a given exposed person. Thus there are many possibilities consistent with the trial data in Table 1.

Table 2a shows one such possibility. In the table, carbon copies are linked by arrows. According to this linkage, four of the five exposed individuals would have died at the same age had they been unexposed, while the fifth exposed person died prematurely due to her exposure. Thus the probability of causation is 1/5, which agrees with that obtained using formula (1). If a PC of 0.5 was the mandated legal cutoff, the judge would have been correct in his decision to dismiss the suit based on formula (1).

[Technical note: The perceptive reader might disagree by

arguing as follows. If (i) the judge believed that the PC was 1/5 in the exposed population and thus that exactly one of the five exposed subjects was harmed by exposure and (ii) from the study data in Table 1, the judge had no way of knowing which of the 5 exposed subjects was the subject who died prematurely, he would be correct in assigning a probability of causation of 1/5 to each and thus in dismissing the suit. But in fact, given (i), the judge can logically deduce from the data in Table 1 that the exposed person who died at 40 must be the individual harmed and thus that (ii) is false. As a consequence the judge should have concluded that PC for the 4 other exposed subjects was zero, but that PC was one for the subject dying at 40 and therefore the latter subject should have been compensated. However, although (ii) is false in our highly simplified and stylized example in which all subjects die at one of 6 distinct ages and the earliest death age in the exposed is strictly less than in the unexposed, nonetheless (ii) will be true in a realistic study. This is because in a realistic study both the exposed and unexposed study subjects will have a positive (albeit small and different) probability of dying at any age. As a consequence, given (i), in a realistic study, the judge is correct to dismiss the suit.]

TABLE 2A—AGE AT DEATH FOR 10 STUDY SUBJECTS—ARROWS LINK CARBON COPIES



Probability of causation 1/5 =.2
Total years of life lost due to exposure = 1 x 50 = 50

However Table 2b considers a second possible carbon copy linkage that, like Table 2a, is also consistent with the data in Table

1. Under this possibility, each exposed individual died ten years prematurely due to her exposure. Thus the probability that exposure was a substantial contributing factor to an exposed subject's death is 5/5=1, since it contributed to every exposed subject's demise. Since the PC is 1, we conclude under this carbon copy linkage scenario, the quantity 1/5 calculated using formula (1) is not the PC and the judge's ruling to dismiss the case was in error.

TABLE 2B—AGE AT DEATH FOR 10 STUDY SUBJECTS—ARROWS LINK CARBON COPIES

	Age in years								Death rate in deaths/ year
	20	30	40	50	60	70	80	90	
5 exposed subjects			1	1	1	1	1		5/200
5 unexposed subjects				1	1	1	1	1	5/250

Probability of causation = 1.0
Total years of life lost due to exposure = 5 x 10 = 50

This example has taught us that epidemiologic data, even ideal epidemiologic data such as that in Table 1, although sufficient to determine the rate ratio is not sufficient to determine the probability of causation. The probability of causation is not in general given by formula (1). Rather its magnitude depends critically on which unexposed person is each particular exposed person's carbon copy and the data provide absolutely no information on the matter. Indeed, whenever the rate ratio exceeds 1.0, the data will always be consistent with a carbon copy linkage scenario analogous to Table 2b in which exposure contributes to each exposed subject's death and thus the PC is 1. Therefore an

epidemiologist who testifies (as many have) that PC is given by formula (1) is either giving false testimony or has failed to read and/or understand the published literature, since the relevant papers refuting formula (1) were published over 15 years ago in leading epidemiologic, biostatistical, and risk analysis journals.

II. EXPECTED YEARS OF LIFE LOST AND COMPENSATION

Since the probability of causation cannot be determined from even ideal epidemiologic data, how should compensation claims be adjudicated? Let us return to Tables 2a and 2b but now, let us calculate the total number of years of life lost (YLL) due to the exposure. Under the carbon copy linkage scenario of Table 2a, the YLL due to exposure is 50, the entire loss being suffered by a single individual. Under the scenario in Table 2b the YLL is also 50, 10 years being lost by each one of the five exposed subjects. These calculations are an example of a general phenomenon. In a large ideal epidemiologic study, one can accurately determine the total number of years of life lost due to exposure because the total YLL depend only on the data and not on the unknown carbon copy linkage scheme. On the other hand neither the probability of causation nor the correct apportionment of the total number of YLL among the exposed subjects can be empirically determined from the data, since both depend on the unknown and empirically unverifiable true carbon copy linkage scheme.

Nonetheless, I believe that awards in compensation cases should be proportional to expected years of life lost calculated under some reasonable agreed upon linkage scheme. Only in this way will the award system serve the public health and "law and economics" goal of providing rational properly-calibrated economic incentives for defendants to avoid exposing plaintiffs to hazardous substances. Specifically, under such an award scheme, and given good epidemiologic data, the total payment made by the defendant to the plaintiffs will be proportional to the total number of YLL (and thereby does not depend on the chosen carbon copy linkage scheme). The defendant then appropriately pays for each year of life that he has cost the plaintiffs, providing a clear incentive to minimize years of life lost. Under this scheme, the

remaining question is how to divide the total award to the plaintiff class among the individual plaintiffs. This must be negotiated within the class because the distribution of YLL among the class members cannot be empirically determined even from ideal epidemiologic data, as the distribution depends on the unknown true carbon copy linkage scheme.

The true carbon copy linkage scheme is determined by the unknown biological mechanisms (at the sub-cellular and genetic level) by which exposure causes disease.

III. FURTHER PROBLEMS WITH COMPENSATION BASED ON THE PROBABILITY OF CAUSATION

Suppose, contrary to fact, we could determine the PC from epidemiologic data via formula (1). Even then a compensation scheme that pays in proportion to years of life lost would be much preferable to one that only compensates plaintiffs whose PC exceeds ½ (or equivalently that RR exceeds 2). To see why consider the workers at a nuclear facility. If "RR greater than 2" were the legal doctrine, companies would have no incentive to decrease the amount of daily radiation exposure sustained by the workforce. Instead the companies' incentive would be to rotate or replace workers just before their cumulative exposure was large enough for RR to exceed 2. Indeed such a rotation policy is common in the nuclear industry. If the cancer response to radiation is linear in accumulated dose and without a threshold, then rotation saves the company compensation costs at the expense of workers lives. Specifically the total number of deaths caused by radiation exposure would depend only on the total radiation dose received by the worker population (regardless of the dose per worker). Thus rotation has no effect on the number of radiation-induced deaths. It only serves to distribute those radiation-attributable deaths over a larger worker population, allowing the company to avoid paying any compensation. This is disastrous public health policy.

Even compensation schemes that would pay in proportion to the probability of causation (without a threshold of ½) have a serious problem with fairness. Under such a payment scheme, the estate of a 40-year old and of an 80-year old would be equally

compensated for the same probability of causation. But the forty year old has suffered a greater loss, because the 40-year old had some 45 years of expected life cut short by her exposure, while the 80-year old had less than 10. This examples points to the fact that even if one wanted to use probability of causation to determine culpability, damages should be assessed in terms of expected years (or quality years) of life lost.

IV. A BACKDOOR INTO COMPENSATION BASED ON EXPECTED YEARS OF LIFE LOST

Therefore, consider a compensation scheme that mandates an award proportional to the expected number of years of life lost on the hypothesis that exposure to the defendant's product caused the plaintiff's death (i.e., the damages) multiplied by the probability of culpability (i.e, by the probability the hypothesis is true). This probability is, of course, precisely the probability of causation. To implement this scheme one must assume a particular empirically unverifiable carbon copy linkage scheme. It can be proved that this compensation scheme is mathematically equivalent to simply paying in proportion to overall expected years of life lost under the assumed carbon copy linkage scheme. It therefore follows from our earlier discussion that the total damages paid by the defendant will be proportional to the total years of life lost due to the hazardous exposure and will not depend on the particular carbon copy linkage scheme. Indeed this conceptual separation of the award into a product of a damages part proportional to expected years of life lost (on the hypothesis that exposure to the defendant's product caused the plaintiff's death) times a culpability part proportional to the probability of causation may be a politically attractive approach to pushing for change in the current compensation policy in the direction of a more rational policy that pays in proportion to years of life lost.

V. THE PROBABILITY OF CAUSATION VERSUS YEARS OF LIFE LOST: SOCIAL, ETHICAL AND LEGAL CONSIDERATIONS

I have argued that award schemes that pay in proportion to years of life lost are economically rational but that award schemes based on probability of causation are not. More generally, expected years of life lost is a more informative summary of exposure effect than the probability of causation, since it takes into account when (instead of just whether) exposure caused the death. Despite these advantages of years of life lost, in certain settings there may be ethical, social, or legal reasons for preferring to base compensation on the probability of causation.

A. Example 1

Suppose two apparently healthy 40-year old men were killed instantly when the brakes of a car in which they were traveling failed due to a manufacturing error. Upon autopsy, one of the men was found to have undiagnosed metastatic lung cancer. The second had no underlying medical illness. If compensation is to be paid in proportion to the probability of causation, then full compensation would be paid to the estates of both men, since the probability is 1 that their deaths were due to brake failure. On the other hand, if compensation is to be paid in proportion to years of life lost, the man with the metastatic lung cancer would receive much less compensation than the man with no underlying illness. It is clear that one could raise legal, ethical, and social questions as to the propriety of differentially compensating the families of the two men in this setting.

Examples such as this show that choice of a measure can involve issues beyond science, public health, or mathematics. To further illustrate the sort of legal, social, and ethical questions brought out by this example, consider the following modification.

B. Example 2

Suppose in the previous example that the first man had in fact a recently diagnosed localized peripheral lung cancer for which he

was to be operated on in one week's time (instead of undiagnosed metastatic lung cancer). Medical experts had given him a 60 percent chance of a complete cure and a 40 percent chance of death from metastatic disease within 5 years. Should the damages assessed against the car manufacture be less for the first rather than the second man if the first man had a 40 percent chance of being dead of lung cancer in 5 years? What if, one year later, a cure for the dead person's cancer was discovered?

VI. NON IDEAL EPIDEMIOLOGIC DATA

Heretofore, I have assumed that data from a large ideal epidemiologic study are available. In practice, sample sizes may not be large and there may be unquantified biases due to uncontrolled confounding, misclassification, and measurement error. These sources of uncertainty add greatly to the difficulty and complexity of evaluating epidemiologic data for one can no longer assume that the empirical rate ratio, calculated from the epidemiologic data is equal to the rate ratio that would be found in a large well-conducted randomized trial. This latter rate ratio we will call the causal rate ratio. I conclude by briefly discussing the consequences of having less than ideal epidemiologic data.

When assessing causality, epidemiologists measure the strength of association by the empirical rate ratio: the ratio of then rate of disease in the exposed to that in the unexposed. If the rate ratio is 10 or greater it is hard-to-imagine unmeasured risk factors or unconsidered biases could be responsible. On the other hand, if the empirical rate ratio is 1.2, it is relatively easy to suppose that unmeasured confounders or uncontrolled biases could explain the association and therefore that the causal rate ratio is 1. This is true even if the study population were of sufficient size to produce narrow confidence intervals for the empirical rate ratio (e.g., (1.1, 1.3)) and an extreme p-value (e.g., $p < 0.001$).

Unfortunately, the public health benefits of controlling (regulating) exposure are best measured in terms of the number of lives saved (or the amount of serious morbidity prevented) per thousand individuals exposed multiplied by the expected number of years of additional life per life saved (which, in total, is the YLL

from failing to regulate the exposure). The number of lives saved is proportional to the causal rate difference rather than the causal rate ratio.

To help understand the public health implications of these facts consider the following example. The death rate from coronary heart disease (CHD) is over 200 times that of soft tissue sarcoma. Thus, a causal rate ratio of 1.2 for CHD corresponds to a much larger causal rate difference than a causal rate ratio of 15 for soft tissue sarcoma [since $200\times(1.2-1)=40$ is approximately three times greater than $(15-1)=14$]. Therefore, it is far more important to regulate an exposure associated with a causal rate ratio of 1.2 for CHD than an exposure associated with a causal rate ratio of 15 for soft tissue sarcoma. However, if the causal rate ratio for CHD is 1.2, it is likely that the empirical rate ratio will be less than 1.5 in any study with sufficient power. In such circumstances, it will not be possible to reach a scientific consensus that the true causal rate ratio is greater than 1 because of concerns about unquantified potential biases. In contrast consensus would exist that the causal rate ratio for soft tissue sarcoma is greater than 1. Therefore the exposure which actually takes far fewer lives would be preferentially regulated. Similarly, the infrequent exposure-caused deaths from soft tissue sarcoma would be compensated in the courts while the more frequent exposure-caused deaths from CHD would go uncompensated.

It follows that an improved tort system will never, in itself, succeed in adequately protecting the public health; one must also regulate exposure to substances that, based on current evidence, are suspected of being harmful, even in the absence of scientific consensus as to harm.