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Whodunit? Liability for Mesothelioma Cases

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It is possible to apportion blame for mesothelioma incidence to previous exposure in relation to an exponent of the time since that exposure. This article demonstrates the application of a formula to calculate the probability that a specific exposure caused an individual mesothelioma case.

Although asbestos is widely accepted as the cause for the majority of mesotheliomas, there is often a question as to which of many exposures caused a particular mesothelioma. Risk of mesothelioma is generally accepted to be proportional to exposure, providing that the patient's exposure preceded diagnosis by over 10 years.

Workers in some of the highest risk categories of occupations (eg, insulators) usually have had several jobs and many exposures to asbestos. For the legal system, this means a continuing struggle to decide which job was likely to have caused the worker's disease, although under the principle of joint and several liability, the courts may abdicate this allocation of blame. As a result, an asbestos manufacturer (more usually, a former asbestos manufacturer) may be forced to defend and pay for claims in which that particular manufacturer had little probability of causing the disease, either because the patient had relatively little exposure to the particular product or had exposure relatively late in a series of asbestos exposures.

Although mesothelioma risk is proportional to "dose" of asbestos, the major predictor for mesothelioma appears to be years since first exposure. This fact was clearly demonstrated by Peto et al,[1] who developed a model for mesothelioma incidence. The model is stated $p = b \cdot t^k$, where p is the incidence in any one year, b is a constant, t is the time in years since first exposure, and k an exponential between 3 and 4. The authors suggest that 3 more likely represents a short-term exposure and 4 a continuing exposure.

It follows that the model can be used to work backward from disease to probable cause, in that the annual incidence is a conditional probability, which, in the case of a known mesothelioma, is equal to 1.0. The model assumes that after the first exposure, each subsequent exposure has the same b and k as developed by Peto et al for first exposures. Each year of exposure can be considered capable of causing risk many years later, in proportion to an exponential of time to the third or fourth power. Each year is considered to have a probability of creating disease, under the assumption that previous exposures did not necessarily create a "hit" to cause the disease. We can then adapt the model to read

$$p_0 + \sum_{i=1}^{n} p_i = 1$$

with p_0 representing background. Assuming that the disease in this instance is caused by asbestos, and not background, the model becomes

$$\sum_{i=1}^{n} p_i = 1$$

where $p_i = t_i^k / \sum t_i^k$.

As an example of an application, consider a former insulator with mesothelioma diagnosed in 1988, who was exposed to asbestos from 1942 through 1944 and again in 1950.

The table provides the data that allows the conclusion that for this person there was an 84.4% likelihood that his 1942 through 1944 job caused the disease, and a 15.6% likelihood for the 1950 exposure.

The exponential used (somewhere between 3 and 4) can vary at the choice of the investigator, with lighter exposure given a smaller exponential, but that generally makes little difference in the conclusions. The necessary numbers are easily set up with any common spreadsheet program to handle the calculations.

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TABLE
Probability of Each Year as Cause

Exposure	Time (t)*	t^n	Causal Probability $t^n/\Sigma t^n$
1942	46	980,186	.503
1943	45	611,285	.281
1944	44	565,047	.260
1950	38	338,254	.156
Total		2,174,751	

*t = time in years since first exposure.

The model proposed here has the advantages of simplicity and derivation from empirical data, and numbers that can be calculated easily on a simple spreadsheet program.

Reference

1. Peto J, Seidman H, Selikoff IJ. Mesothelioma mortality in asbestos workers: implications for models of carcinogenesis and risk assessment. *Br J Cancer*. 1982;45:124–35.