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RISK ASSESSMENT FOR LOW LEVELS OF ASBESTOS EXPOSURE

-- Reg had 1 hr to talk about the techniques by which we generate data and I have only one half that time to talk about interpret data. Unfortunately, I think that the proportion of time at this conference devoted to interpreting asbestos sampling results is much greater than the proportion of time and effort devoted to interpretation of actual sampling results. We collect mountains of data, yet little effort is spent in learning what it all means or communicating the results in an understandable fashion to those who are exposed, or to those who are responsible for exposures.

-- This is a deplorable situation. As I.H.s we may be called upon to interpret sampling data. Indeed some legal authorities tell us that we have a duty to warn those who are at risk.

-- Thus, the goal of my presentation is to convey to you some information that you may find helpful if need to assess and communicate the risk of asbestos exposure.

1. First I'll present some of the basic information on asbestos risk.

2. Then, look at an example of risk assessment and communication of hazard.

3. Some of the basic information plus a bibliography has been handed out.

Risk assessment-- The determination of the risk to a populations (humans or other organisms) from exposure to a hazardous agent. The scope of risk assessment can be extremely broad including:

(1)

--source of agent
--distribution in environment
--routes of exposure
--populations exposed
--degree of exposure
--hazards associated
--dose-response relationship
--excess risk to populations
--remedial actions

Assessments of this scope are often associated with contaminants in the outdoor environment. Fortunately, in I.H., concern is typically focused on small populations of people whose exposure may be characterized by direct measurement.

e.g. workers on an abatement project
or
children in a school with ACM.

~~Our~~ The objective is often to estimate the excess risk to population members resulting from exposure and to communicate that risk so that informed actions may be taken.

--There are at least 3 levels of risk assessment studies:
1. Fundamental
2. Summary and Evaluative
numerous papers in the literature on asbestos
3. Applications to specific circumstances

Some of the components of our risk assessment are obvious:
Source- usually known
Routes of Esposure- inhalation
Degree of exp.- air sampling results

(2) Effects:

-- We are usually concerned with mortality from asbestos related diseases since the data is available. Much less is known regarding morbidity and subclinical effects.

-- Mortality is expressed as some measure of excess risk associated with asbestos exposure. Excess over what?

--often comparison is made with the general population, but workers are healthier than the general population. They show ~80% of the death rate of the gen. population when matched for age and sex.

(3) --Measures:
Relative Risk, or
SMR -- for rare diseases, such as mesotheliomas, these measures reach absurdly high values since the denominator is very small.

An alternative is Excess Risk--this is the absolute probability of an outcome. Here I will usually use deaths per 100,000 persons exposed.

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(4)

Dose-Response

This is the most complex part of the risk assessment for asbestos. But, it is something of a misnomer since the true dose is seldom, if every known-- only exposure levels are measured.

Factors affecting dose-response.

Smoking

Nicholson (1983) of Mt. Sinai in his recent study for EPA cites work by Selikoff, Hammond, Berry and McDonald in support of the assertion that the effects of smoking and asbestos exposure on lung cancer are multiplicative. A study of over 12,000 asbestos workers showed that both smokers and non-smokers had 5 time the risk of dying from lung cancer. However, the risk is low for non-smokers; therefore, mulitplying it by five does not result in many cases, although any excess is undesireable. On the Other hand, smoking itself caused a significant increase (10 X controls). Thus, that high risk multiplied by 5 gives an immense increase.

(5)

-- In a dissenting opinion Hughes and Weill cite the same studies, but come to the conclusion that the percentage increase in lung CA risk may be "somewhat less" for smokers than for non-smokers.

-- Smoking behavior does no seem to affect the relationship between mesothelioma and exposure.

Age at which Exposure Occurs

Lung CA- (Relative Risk Model)

(6)

-Relative risk increases after time of first exposure until ~35-40 years. The shape of these curves is independent of the age at first exposure.

(7)

- Median time from first exposure to death was 32 years (Selikoff)

-Life time risk seems to be only related to cumulative exposure (nicholson, 1983)

-The latency period has been shown to be function of both exposure and age. The higher the exposure, the shorter the latency period. The older the person, the shorter the latency period.

Bottom Line: life time risk of lung CA mortality seems to be dependent only on the degree of exposure.

Mesothelioma- (Absolute Risk Model)

(3)

-Incidence of death is independent of the age of first exposure and increases according to a power of time after a "delay period" of about 10 years (Nicholson, 1983).

- Median time to death after first expsoure is about 36 years.

- The rates of mesothelioma increases with time more rapidly than those of lung CA and may equal those of lung CA 45 years or so after first exposure.

- Thus, age of exposure is of considerable consequence for estimating mesothelioma risk. ~~However, this is often not accounted for explicitly in risk assessment.~~ Nicholson's work for EPA has look at this in detail.

Sex

- Does not affect dose-response for mesothelioma, but males seem to have 2.5 to 3 time the excess risk of women for death by lung CA from asbestos exposure.

Types of Asbestos

- All forms of asbestos are capable of producing cancer. However, there is controversy regarding whether crocidolite or amphiboles in general are more carcinogenic than chyrsotile. Standards in other countries, such as Britain and Sweden take the type of asbestos into account, while U.S. Standards do not. The epidemiological data are quite contradictory, possible because good particle size distribution data were not available for the study exposures.

ACM composition or Process

-- Gradients in lung CA rates have been observed in the progressive processing of asbestos. Lowest rates of lung CA are associated with mining and friction product production, and the highest with asbestos textile production. It seems likely that these gradients could be explained by differences in particles size distributions.

-- PCM microscopy yields an index of exposure; it is a surrogate for the more complete characterization of exposure and thus may yield annomalies in dose-response relationships.

-- There is insufficent evidence to determine if there are gradients in mesothelioma rates by industrial process after accounting for the type of asbestos used.

Dose-Response

Lung Cancer-

Two hypotheses seem to be widely accepted by toxicologist and epidemiologists who have examined the dose-response for asbestos.

1. After an appropriate latency period, the relative risk of lung CA is independnent of the elapsed time between exposure and observation.

2. The relative risk of lung CA increases linearly with cumulative exposure.

Correllaries:

a. We can use linear extrapolation from the dose-response of industrial workers exposed at high levels in years past to determine the risk of exposures several orders of magnitude less.

b. Cumulative dose may be represented by the product of the average concentration and the duration of exposure

Although is is doubtful that the dose-response relationship for low exposures can ever be determined by direct observation, these assumptions are consistent with observations at higher levels of exposure and is supported by the mathematical theory of multistage carcinogenesis

Also, there is general agreement that it is unlikely that the risk increases more rapidly than linearly at low levels. Thus, it is believed that these assumptions lead to conservative estimates of risk.

Mesothelioma-

(9) In exposed workers, the incidence rate increases rapidly with age and duration of exposure, at least until the age at which other causes of death become important. Relatively little quantitative dose-response data is available. Thus, mathematical models have play a large role in estimating risk of mesothelioma mortality.

Nevertheless, Schneiderman (1981) observed that two industrial studies which investigated exposure gradients found response rates per unit dose which were of the same order of magnitude. Thus, he assumed a linear dose-response relationship. In light of the nature of mesothelioma risk, the use of his approach may not be valid for assessing the risk of childhood exposures.

Summary Dose-Response Data

-Lifetime excess risk of death for exposure to 1 fiber-year/cc.

-Excess risks are in relatively good agreement compared with risk assessments for other agents.

Summary of Asbestos Concentrations

PCM-TEM Conversion

-Adds an additional degree of uncertainty.

-Most risk assessments deal with concentrations in fibers/cc. If TEM is used, ng/cu.M are converted to fibers/cc. Validity is dubious.

Excess Cancer Deaths at Various Exposure Levels

Case Study