Asbestos as a metaphor for teaching risk perception

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ABSTRACT
Asbestos -- the word means different things to different people, ranging from a mineral form to a nightmare for a parent whose child may be exposed to it in school. The public perception of and public policy for asbestos are derived from popular media stories; the academic community often does not take enough of an active role in public policy issues. For instance, does the financial cost of asbestos abatement outweigh the proportionate reduction in asbestos-related illnesses? This question can only be addressed by a rational, factual, unbiased overview of the data, which requires integration of science, medicine, philosophy, and law. More involvement from the academic community is needed to answer these sorts of questions. A critical evaluation of the asbestos issue provides an outline for evaluating other related environmental issues which, hopefully, will make our students better able to make rational public policy decisions.

Introduction

I became interested in asbestos in 1984 when I was a graduate student studying amphiboles in a crystal chemistry course. One chapter in the book we were reading entitled, "The geological occurrences and health hazards of amphibole and serpentine asbestos" (by Malcolm Ross, in Reviews in Mineralogy, 1981), indicated the health hazards from exposure to asbestos were over exaggerated, especially for low-level (non-occupational) exposures. My interest never waned, and now, as a faculty member, I use the asbestos case history as an example of how to evaluate risk in health-related environmental issues. I teach a semester-long seminar for honors-students on critical risk assessment, spend a week in my large introductory geology class discussing these issues, and have given several seminars on this subject the past three years in various universities in the United States and Europe.

The purpose of this paper is not to provoke controversy but to provide an overview of the asbestos problem and to show a pedagogical approach to teaching similar cross-discipline environmental issues. It includes such diverse fields as engineering, law, medicine, mineralogy, philosophy, and the media; it is important to understand the relationships among these seemingly unrelated disciplines. For asbestos, or any other health-related issue, which involves human suffering, discussions and decisions must be based on factual matter. We must not allow irrationality, ignorance, or greed to determine our nation's public policy. Asbestos abatement is a multibillion dollar industry in the United States - one in which many people have a vested interest. The facts (case studies, medical reports, etc.) not anecdotes, must be used to analyze the issue and make public policy decisions.

What is asbestos?

The question yields different answers depending upon the audience. To a mineralogist, asbestos is a mineral form. To an engineer, it is an industrial material with several useful properties. To a medical doctor, it is an agent that might cause certain diseases. The third answer invokes several more issues: to a lawyer, a possible lawsuit; to a news reporter, a story; to an asbestos abatement worker, a job; and to a public school administrator or a parent, a nightmare.

Mineral: Asbestos is a mineral from, generally thought of as fibrous. If the aspect ratio (the length:width) is greater than 10:1, the term fiber is applied (Skinner and others, 1988). By this definition, at least 400 of the nearly 4,000 known minerals could be classified as fibrous. Precise definitions of asbestos are difficult. For example, Ross (1981) notes the commonly accepted Occupational Safety and Health Administration (OSHA) definition of asbestos as any product containing "any of the naturally occurring amphibole minerals (amosite, crocidolite, anthophyllite, tremolite, and actinolite) and the serpentine mineral chrysotile with dimensions greater than 5 μm long and less than 5 μm in diameter and an aspect ratio of 3:1."

There are two major groups of asbestos minerals: chrysotile (one of the forms of serpentine) and certain amphiboles (the five listed in Table 1). The aspect ratio is a matter of debate. Mossman and Gee (1989) state a 3:1 value, but the mineralogy community thinks the aspect ratio should be higher. Table 1 lists the common asbestos minerals. The three most abundant asbestos minerals have special names when they occur in a "fibrous" habit. Chrysotile is by the far the abundant asbestos mineral, followed a distant second and third by crocidolite and amosite.

These minerals form in metamorphic terrain undergoing uniaxial tensional strain. The fibers grow parallel to the principal strain axis during deformation (Figures 1 & 2). Amosite and crocidolite occur in banded ironstones, while the majority of chrysotile, about 85%, occur in alpine-type metamorphic rocks with only minor amounts coming from stratiform ultramafic intrusions and serpentinized limestones (Ross, 1981).

Because the physical properties of minerals are directly related to their crystal structure, there should be structural reasons why amphiboles and chrysotile occur as fibers. All silicate minerals are based upon the polymerization of silicate tetrahedra. Amphiboles consist of two cross-linked chains of silicate tetrahedral parallel to the c-crystallographic axis (Figure 3). These chains paralleling the c axis cause the amphiboles to be elongated parallel to c, and, in the case of asbestos amphiboles, the c axis parallels the fiber length. Their basic building block is $(Si_8O_{22})^{12-}$. Other available cations enter into four possible octahedral sites between the double chains to complete the structure.

The crystal structure of the serpentine minerals can be schematically represented by sheets of silicate tetrahedral linked to sheets of Mg octahedral. Mg is six-coordinated to four OH^- groups and 2 oxygens (Figure 4). The two oxygens of the octahedron are the apical oxygens of the polymerized sheet of silicate tetrahedra. The distance between the apical oxygens of the tetrahedra is less than the distance between the oxygens for the octahedron. When these two sheets combine, they must curve around each other for structural stability. This curving causes chrysotile to form scrolls elongated parallel to its a-crystallographic axis, which also parallels the fiber elongation.

Industrial mineral: Asbestos has many commercial uses based upon its resistance to heat, friction, and acidic conditions. Its working definition in the industrial arena is: slender, easily separable, flexible fibers with high tensile strength, chemical stability, and incombustibility. The combination of its resistance and the fact it occurs as a fiber creates several applications. It is an excellent fireproofing material and is used in clothing, gloves, face masks, stage currents, roofing products, and spray-on insulation in buildings. It is used as a binding agent in floor tile and cement pipes and as a friction agent in brake linings for cars and trucks.

Approximately 95% of the asbestos mined and used in the United States is chrysotile, while crocidolite and amosite comprise about 5%, (Ross, 1981). Commercial terms have been applied to these three asbestos minerals; chrysotile is known as "white" asbestos, crocidolite as "blue" asbestos, and amosite as "brown" asbestos (see Figure 1).

Health threat: Three pulmonary diseases, asbestosis, mesothelioma, and lung cancer, are associated with asbestos exposure. All three diseases have been associated with inhalation of asbestos fibers. The etiology (cause of the disease) of the asbestos-related diseases is poorly understood (Skinner and others 1988; Mossman and Gee, 1989; Mossman and others, 1990).

Asbestosis is a pneumoconiosis, a lung disease caused by foreign particles deposited in the lung through inhalation. It is the result of long-term inhalation of large amounts of asbestos. The lung tissue encapsulates the asbestos and hardens, thus decreasing its efficiency in O_2-CO_2 exchange. The heart, in turn, must work harder, and death results from heart failure. Asbestosis is not a cancer. There are several other similar diseases that result from inhalation of particulate matter. Black lung is caused by inhalation of coal dust, brown lung is caused by inhalation of cotton fibers, berylliosis is caused by inhalation of beryllium, and silicosis is caused by inhalation of silica dust. Approximately 200 deaths per year in the United States can be attributed to asbestosis (Table 2). All deaths from asbestosis are directly linked to long-term occupational exposure in the preregulated workplace.

Mesothelioma is a rare disease of the lining of the lung and stomach. There are two types occurring in the lung lining: localized benign, which is not related to asbestos exposure, and diffuse malignant, which is. Approximately 80% of the diffuse mesothelioma cases can be linked to asbestos exposure (Mossman and Gee, 1989); the cause of the remaining cases is unknown. Diffuse mesothelioma usually results in death within 1-2 years of its diagnosis (Ross, 1984). Its latency period is 35-40 years, and most deaths occur in patients over 60 years old (Mossman and Gee, 1989). Mesothelioma causes 400 deaths per year (Table 2), and 320 of these (i.e., 80%) are attributable to asbestos exposure. This is the most feared asbestos-associated disease, because death comes as a cancer so long after the exposure. It is assumed that children exposed to asbestos in school may contract the disease when they are only 40-50 years old.

Lung cancer by far causes the greatest number of annual deaths in the United States, approximately 130,000 (Table 2). Cigarette smoking is the main cause of lung cancer, resulting in approximately 110,000 deaths per year (Hoffman, 1992). The remaining deaths occur in non-smokers and might be caused by other environmental agents (e.g., radon, second-hand cigarette smoke, or asbestos). A synergetic relationship probably exists between smoking and asbestos in lung cancers (Mossman and Gee, 1989). The relationships between these diseases and asbestos exposure are discussed later.

Government regulations

Because asbestos was shown to cause diseases in the work place, the federal government imposed regulations setting limits for asbestos exposure. In 1972, OSHA set a limit of 5 fibers/cm^3 in the workplace. OSHA acceptable levels declined during the next two decades (Table 3).

If large levels of asbestos are harmful, then possibility low levels are, too. For a carcinogenic material there is no acceptable minimal exposure level. This train of thought led to a congressional act to limit exposure of children to asbestos in public schools. The Asbestos Hazard Emergency Response Act of 1986 (AHERA) requires public schools to locate and monitor the condition of asbestos-containing material (ACM). If the ACM begins to deteriorate, allowing fibers to become airborne, it has to be removed or encapsulated. The removal of this material from buildings such as public schools is very difficult and very expensive. The legislation does not provide any funds for asbestos abatement. The legislation also requires action to be taken based upon the condition of the ACM and not by monitoring the airborne asbestos levels, as is done with OSHA regulations in the workplace. Table 3 lists average asbestos levels found in outdoor and indoor air. The data show that ACM levels in buildings are much lower than OSHA regulations for the workplace; thus, the AHERA sets asbestos levels far below what OSHA considers acceptable.

Lawsuits begin to appear in the courts based upon presumed risk of low-level exposure to asbestos. This led to the "one-fiber" theory that states one-fiber of asbestos is sufficient to cause an asbestos-related disease. This theory goes hand-in-hand with the perception of many that we should have a zero threshold level for carcinogenic materials in the environment. The implication is that if something is bad in large amounts, it must have a negative impact even at very, very low-levels. As a point of interest, the average human inhales approximately 10 m^3 of air a day (Skinner and others, 1988). In outdoor air, a normal human would inhale 3,900 fibers of asbestos per day (Table 3).

Abatement questions

Is removal of asbestos from existing buildings necessary? The main points here are the health risk associated with low-level exposure and the financial cost of abatement. Before these two issues are addressed, some philosophical points need to be made.

Risk imposed upon us is viewed much differently than risk we choose to impose upon ourselves. The chosen risk of a child being killed in a car accident is far greater than the risk imposed from exposure to asbestos. However, we choose the former risk and the later is imposed upon us. Anecdotally, many of the people I have heard complain the loudest about asbestos exposure also smoke cigarettes and, in turn, expose their children to second-hand smoke, putting their children at much greater risk of lung disease than asbestos exposure does.

Anyone should expect a concerned parent not to want their child exposed to undue or avoidable risks. However, parents' fears are often unfounded and based solely upon ignorance. Unfortunately, some of the responsibility for the public's ignorance on these issues rests with our educational system. We rarely address these issues in an unbiased manner in higher education, often because of our fear of becoming involved in public policy issues and the difficulties of explaining scientific issues to non-scientifically literate individuals.

The general public's response to environmental risks are extremely variable. In general, the population does not seem very concerned about the physical environment, evidenced by urban development on floodplains, near the ocean, and on active faults. The passivity we display to the physical environment is contrasted by a paranoia to the chemical environment. For instance, if a local paper ran a

headline, "City water supply contains selenium," the public would most likely panic. Selenium is required in animal diets from 0.04 ppm to 0.1 ppm (Keller, 1992), but above 4 ppm it becomes a toxin. Many chemicals display this sort of beneficial/harmful dosage scenario. Thus, zero-level for an element like selenium could actually be harmful to our health.

Despite mounting evidence showing it is unnecessary, asbestos abatement continues, encouraged by those who profit from the business. The Environmental Protection Agency (EPA) estimates cost for abatement of 733,000 public and commercial buildings is $53 billion over the next 30 years (Mossman and others, 1990). Uncertainties in their estimate may cause the actual cost to range as high as $100 - $150 billion. Recent actual annual costs are $1.8 billion for 1987, $4.2 billion for 1989, and $2.7 billion for 1991 (Newsweek, April 13, 1992, p. 59). For comparison, the 1990 budget for the Department of Education was approximately $23 billion (Hoffman, 1992) and NSF's research budget was approximately $2 billion. The University of Idaho's annual budget is approximately $70 million, and Geology's operating expense (supplies, phones, travel, copying, etc.) portion is approximately $30,000. The university spends approximately $300,000 annually on abatement, 10 times more than operating expenses for Geology. This "rule of 10" holds for several geology departments nation-wide in which I have given my asbestos seminar. The asbestos abatement industry is large and has a vested interest in maintaining the status quo of federal regulations in the workplace and public schools.

Government regulations require us to continue abatement or face potential lawsuits. The judgments of the lawsuits are often more than the cost of abatement. It is often financially prudent for a company to settle out of court, even though they are not guilty, than to go to court.

So there are at least three major reasons, which have nothing to do with human health or safety, why we continue abatement practices: public perception, financial gains of the abatement industry, and the legal system. Figure 5 graphically demonstrates how our fear of death and the dollar amount of lawsuits are inversely related to the probability of death.

There is the problem, especially true in the early days of abatement, of increased airborne asbestos due to abatement. Another problem is the exposure of a whole new group of asbestos workers to higher levels of asbestos during the abatement process.

Determination of asbestos-associated health risks

There are three methods used to evaluate risk of a particular material to humans. From least to most expensive and reliable, they are: *in vitro* testing (determining the effects of a material on individual cells, e.g., the Ames test), *in vivo* testing (exposing laboratory animals to materials and checking for disease development), and case studies of humans exposed to a material where the death rates and exposure levels are known.

Case studies: In a case study, cohorts (groups of individuals with similar traits) of exposed individuals are compared to similar cohorts of nonexposed individuals where the only difference is exposure to the material of interest. An estimate can be obtained from the control cohort of the background, or normal death rate, of a particular disease and a comparison made to the group exposed to the material of concern. The standard mortality rate (SMR = number of deaths in exposed cohort

divided by the number of deaths in the control cohort) is often calculated to show this relationship. The material under question poses a significant health threat if the SMR is significantly greater than 1.0.

Ross (1981, 1984) provides the basis for analyzing asbestos trade workers and their causes of death. In these case studies he differentiates between chrysotile and amphibole asbestos, the three associated diseases, and occupational vs. non-occupational exposure. One problem with the available data is that lung diseases are confounded because many of the asbestos workers were smokers and many of them worked in environments in which they were exposed to more than one type of asbestos. Sufficient data are available for only chrysotile and crocidolite to evaluate the three diseases. It appears that asbestosis was a significant occupational disease prior to government regulations. Currently, deaths from asbestosis are declining and it is not a threat outside the workplace (Mossman and Gee, 1989; Ross, 1981).

Chrysotile: Ross (1981) presents 13 case studies for the asbestos trades relating mortality from lung cancer and mesothelioma. In all of these studies but one, study #13, the workers were exposed to more than one kind of asbestos; in study #13 they were only exposed to chrysotile. The study #13 group consisted of 264 workers of which 66 were dead - 4 from lung cancer and none from mesothelioma. The lung cancer death rate was 6.1%. Is that number higher than expected for an average population?

Ross (1981) discusses the impact of smoking on lung cancer and the confounding problem that most asbestos workers smoked. Approximately 70% of the workers smoked in studies by Selikoff and Hammond (1975) and Saracci (1977). In a study of chrysotile asbestos miners and millers, it was found that 85% smoked (McDonald and others, 1974). The mortality rate for lung cancer in which 75% of the people smoke ranges from 6 to 7.5%, regardless of occupation (Ross, 1981).

The cancer mortality rate of five nations, including the United States and Canada, was 5.7% for lung cancer and 0.03% for mesothelioma (Ross, 1981). Ten case studies of asbestos miners, millers, and hard rock miners yielded an average lung cancer death rate of 5.7%, excluding the crocidolite miners. Some of the individual groups of asbestos miners and millers show an increase in lung cancer rates, but only after long periods of heavy exposure to chrysotile and anthophyllite; however, even these groups do not show an increase in mesothelioma.

McDonald et al. (1980) links lung cancer to exposure levels in the chrysotile mines of Quebec. For miners exposed to levels of asbestos between 10-21 fibers/cm^3 for 20 years, their total SMR was 0.94 and lung cancer SMR was 1.15, slightly significant. These fiber counts are two orders of magnitude higher than current OSHA standards and five orders of magnitude higher than those found in schools containing ACM (Table 3). In 1970, a British study showed no excess lung cancers, or any other asbestos related diseases, when workers were exposed to chrysotile levels of 0.5 - 1.0 fibers/cm^3 (Mossman and others, 1990).

Other reviews of the asbestos problem (Mossman and Gee, 1989; Mossman and others, 1990; Lippmann, 1992; Sivak, 1991) confirm the above findings that chrysotile may cause a slight increase in lung cancer but that it is difficult to evaluate how smoking affects the death rate. Also, even for those individuals who do not smoke, they are exposed to second-hand smoke from their smoking peers.

From the various case studies, it appears that chrysotile does not cause mesothelioma. One such case study involves a school in Ambler, Pennsylvania. Next to the school is a 150,000-ton pile of chrysotile-containing material. For the past 100 years, thousands of children have been exposed to this material; however, there has not been a single reported case of mesothelioma in any of the students who attended the school (Harvey and Rollinson, 1987).

Crocidolite: There is current agreement that crocidolite poses more of a health threat than chrysotile (Ross, 1981, 1984; Mossman and Gee 1989; Mossman and others, 1990; and Lippmann 1992). The lung cancer death rate is 11.6% in a case study for crocidolite miners, which is almost twice as high as expected, and the death rate from mesothelioma was 3.3%, compared to a normal value of 0.03% (Ross, 1981). Mesothelioma death rates are as high as 10.6% for individuals exposed only to amphiboles (Mossman and Gee, 1989). Mossman and others (1990) point out that mesothelioma may account for up to 18% of the mortality in crocidolite workers. Several other case studies (Ross, 1984) support these findings. Ross (1984) also reviews non-occupational data for exposure to asbestos. There is a higher incidence of mesothelioma in the crocidolite mining towns and no increase in asbestos-related diseases in the chrysotile mining areas.

Laboratory studies: Lippmann (1992) presents a good review of the current status of animal testing with asbestos. The data obtained from these tests support the observations made in case studies. Unfortunately, it is these animal studies that are used to set guidelines and policy for asbestos exposure limits. A test is conducted at high concentrations (high dose rates) which, in turn, yields high death rates. A model (Figure 6) is used to extrapolate back to low-level (Nally, 1984; Lippmann, 1992). The accepted level of risk that a material poses a threat to human health varies from 1 in 10,000,000 to 1 in 100,000. If we accept the center of this range as our acceptable risk, then whatever dose corresponds to a one in a million death rate is considered the upper limit for human exposure. Using this mentality (or lack of it), if two people die per year in the United States from "something," that "something" should be considered bad and removed from the environment. However, cigarette smoking causes approximately 300,000 deaths per year (Almanac, 1992), and cigarettes are allowed to remain in our environment.

An estimated increased chance of lung cancer of two in a million is predicted by Doll and Peto (1985) for an exposure at 0.0002 fibers/cm^3 over a period of 20 years of 8-hour days. As Lippmann (1992) points out, based on the data in Table 3, mean asbestos concentration in buildings is seldom higher than in outside air, so much of this small risk may be based upon outdoor air entering the building.

Integration of all of the case studies and *in vitro* studies suggests that fiber size and composition may play a role in the etiology for the three pulmonary asbestos diseases. Only sufficiently small fibers can enter the lungs. According to the Stanton hypothesis (Mossman and others, 1990), the fiber size believed to cause most problems, are fibers greater than 8 μm in length with a diameter less than 0.25 μm. This size range correlates to fibers capable of entering different portions of the lung and having an adverse effect on the lungs' operation. For instance, the smaller fibers can pass through a lung into the lung lining and cause mesothelioma. Lippmann (1992) proposes fiber lengths approximately 2 μm for asbestosis, 5 μm for mesothelioma, and 10 μm for lung cancer and fiber diameters above 0.15 μm for asbestosis and lung cancer and fibers below 0.1 μm for mesothelioma.

The chemical composition of the fibers also have been related to etiology. The amphibole fibers appear to be the most dangerous - thus, the origin of the Amphibole hypothesis linking higher risks with exposure to amphibole asbestos types as compared to chrysotile. The chemical composition may be indirectly related to diseases because of the biodurability of the fiber. Hume and Rimstidt (1992) show chrysotile dissolves in the lung in about nine months and amphibole remains basically forever - in terms of a human life. Lippmann (1992) reviews solubility data for chrysotile and crocidolite and supports Hume's and Rimstidt's observations. Thus, *size* of the fiber and *duration* of stay may be the determining agents for asbestos-related diseases.

Summarizing all the available data, it appears that chrysotile, by far the most commonly used type of asbestos, has questionable health effects for inducing lung cancer and does not cause mesothelioma, even at occupational doses. The amphiboles, especially crocidolite, cause an increase in both lung cancer and mesothelioma at occupational exposures because they are retained longer in the lungs. Thus, in the public schools, where chrysotile is the main form of asbestos and fiber/cm^3 levels are approximately three orders of magnitude lower than accepted occupational levels, there appears to be no real health threat.

Further reading

This article attempts to integrate all of the issues surrounding asbestos. Several books and many research articles have been written on certain aspects in more depth. Books of special interest are *Asbestos in the Schools* (Harvey and Rollinson, 1987) and *Asbestos and Other Fibrous Materials* (Skinner and others, 1988). Good review articles include Guthire (1992), Lippmann (1992), Mossman and Gee (1989), Mossman and others (1990), Ross (1981, 1984), and Stone (1991).

Other problems for evaluation

Table 4 lists several other current environmental issues. The blank in the table is for the endless addition of new problems that we face. Each of these issues can be discussed and analyzed to see if it presents a significant health threat by using the following procedure:

1. Provide an overview of the problem.
2. Define the material. It will have more than one definition depending upon the audience.
3. Describe some of the analytical methods used to identify and quantify this material.
4. Describe the material's properties. Why do we (or did we) want to use this material?
5. Identify the associated diseases and their relationships to the material. Define as much of the etiology as is known.
6. Describe any government regulations.
7. Add the risk factor and compare associated deaths with available data.
8. Describe the financial impact of not using this material or removing this material from the environment.
9. Use case studies and/or laboratory test data to determine the health effects of exposure to the material.
10. Conclusion - Is this a problem our society should deal with or does it just make a good TV news show piece?

Lengthy articles could be written on each item in Table 4. A final comment on one of the entries seems especially appropriate for a geology journal. The International Agency for Research on Cancer (IARC) designated quartz dust a carcinogen based upon *in vitro* studies. OSHA rules are invoked by IARC's decision, and a level of 0.1% free silica (i.e., the silica polymorphs) was set as the upper safe exposure (Ross, 1991). Materials capable of producing dusts above 0.1% free silica must be labeled as a possible carcinogen. This means every gravel road, field, desert, beach, etc. in the United States must be labeled as a possible carcinogen!

Conclusions

An integration of facts from several different disciplines is required to understand the asbestos issue in the United States. If the data are then analyzed in a rational, unbiased, scientific approach, it appears the billions of dollars a year we spend on asbestos abatement in the public schools is not necessary. The current government regulations should be changed to distinguish between the different types of asbestos, and actual monitoring should occur in the schools. Asbestos removal should only occur when levels are significantly higher than that found in outside air. Much higher acceptable levels for chrysotile should exist than for the amphibole varieties.

The asbestos issue can serve as a model for critical thinking. A thorough comprehension of this issue requires a student to glean data from several sources, integrate it, analyze it, and reach his/her own conclusion of the significance of the assumed problem to our society.

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Tables

Table 1: The six common asbestos minerals. The first three minerals in the list have specific names when they occur in "fibrous" form. Their non-fibrous name is listed in parentheses.

chrysotile (serpentine):	$Mg_3 Si_2 O_5 (OH)_4$
crocidolite (riebeckite):	$Na_2 (Fe^{2+}, Mg)_3 Fe^{3+}_2 Si_8 O_{22} (OH)_2$
amosite (grunerite):	$(Fe^{2+})_2 (Fe^{2+}, Mg)_5 Si_8 O_{22} (OH)_2$
anthophyllite:	$Mg_7 Si_8 O_{22} (OH)_2$
actinolite:	$Ca_2 (Mg, Fe)_5 Si_8 O_{22} (OH)_2$
tremolite:	$Ca_2 Mg_5 Si_8 O_{22} (OH)_2$

Table 2: Annual death statistics in the United States (population approximately 250 million). Total deaths for 1988 = 2,167,999. All data from the 1988 (most recent) U.S. Vital Statistics (Feinleib, 1991).

Cancer	970,096	Asbestosis	213
Heart disease	969,400	Silicosis	135
Lung cancer	133,284	Lightning	82
Auto accidents	46,300	Bee stings	34
Commercial air	536	Spider bites	4
Mesothelioma	400	Snake bites	0

Table 3: Fibers/cm^3 of asbestos (>5 μm with 3:1 aspect ratio). The first series indicates numbers in the workplace and the changing OSHA regulations from 1972 to 1992. The second set indicates average values found in outdoor air, indoor air, schools, and public buildings with ACM in different states of repair (Mossman and others, 1990).

> 100	preregulated workplace
5	OSHA (1972)
2	OSHA (1976)
0.5	OSHA (1983)
0.2	OSHA (1986)
0.1	OSHA (1992)
0.00039	outdoor air
0.00024	schools
0.00099	indoor air, no ACM
0.00054	indoor air, nondamaged ACM
0.00073	indoor air, damaged ACM

Table 4: Other problems that might be discussed in a risk assessment course.

Pb in drinking water	Low-level radiation
Al in cans	Hg in dental fillings
Alar on apples	Radon in houses
EMF in air	Free quartz
Fluoride in water	_____

Figures

Figure 1: Crocidolite (left) and chrysotile (right) in hand specimen (a Swiss Franc for scale). Both samples show the fiber axes are parallel to the direction of greatest strain.

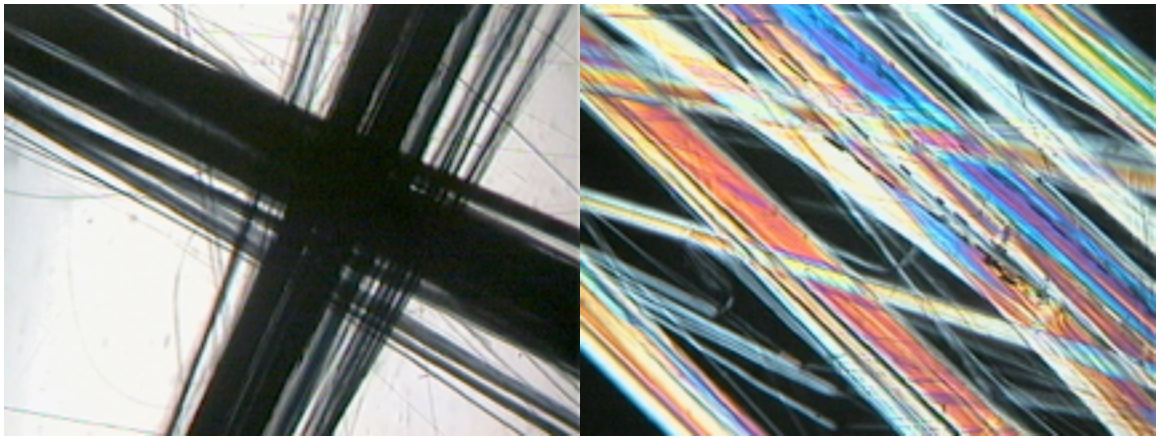

Figure 2: Photomicrograph of crocidolite (left in cross-polarized light) and chrysotile (right in plane-polarized light). (Field of view approximately 2.5 mm wide.)

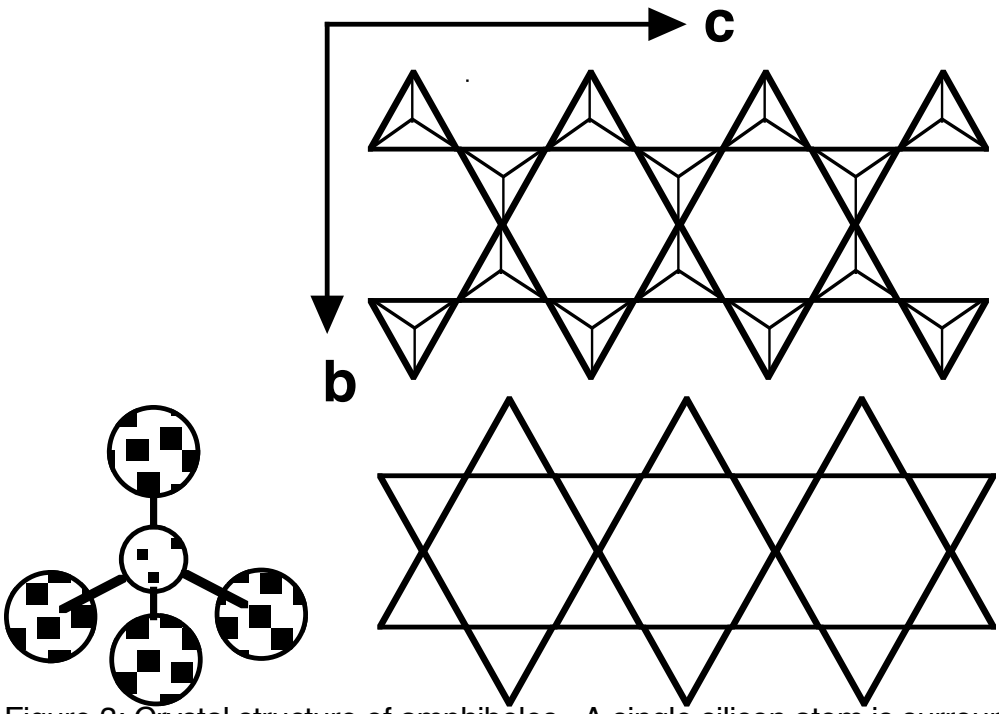

Figure 3: Crystal structure of amphiboles. A single silicon atom is surrounded by four oxygens atoms to form a silicate tetrahedra (left). These tetrahedra then link to form double chains parallel to the c-crystallographic axis (right), which parallels the long axis of the fibers.

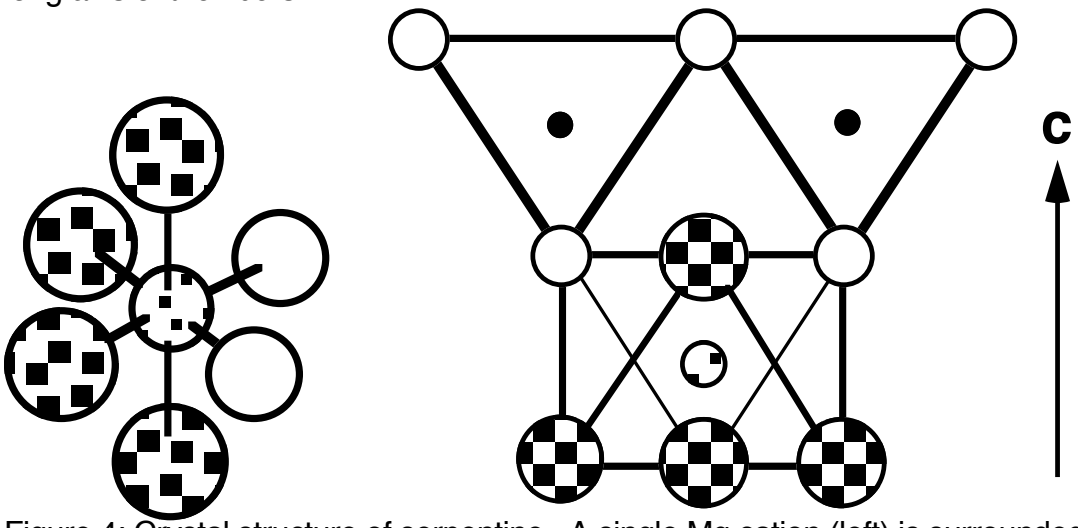

Figure 4: Crystal structure of serpentine. A single Mg cation (left) is surrounded by four OH groups (stippled) and two oxygens (non-stippled) to form an Mg octahedron. The oxygen-oxygen spacing in the Mg octahedron is similar, but slightly larger, than the oxygen-oxygen spacing for the apical oxygens for a sheet of polymerized silicate tetrahedra. These two units combine to form the basic building block for the serpentine mineral group (right). In chrysotile, the difference in oxygen-oxygen spacing causes the octahedral layer to cure around the tetrahedra layer, resulting in a scrolled tube structure with the scrolls parallel to the a-crystallographic axis, which parallels the long axis of the fiber.

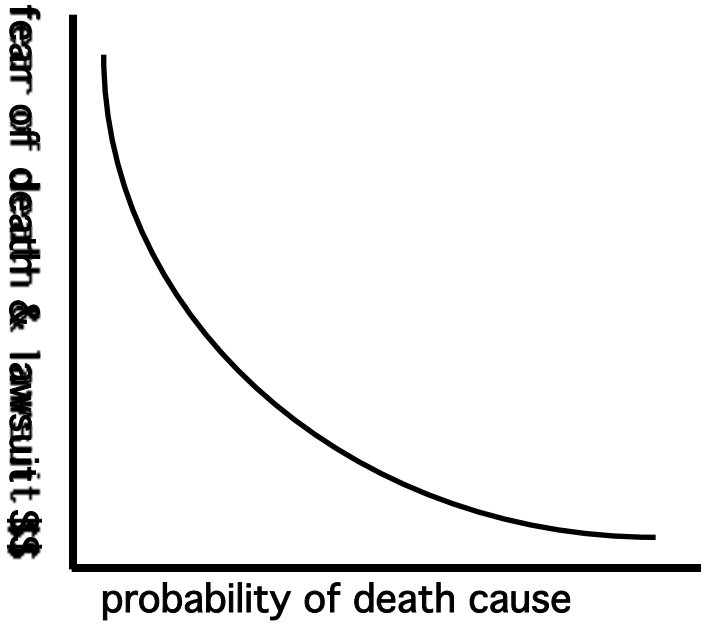

Figure 5: A philosophical view of the inverse relationship between lawsuit awards or fear of death vs. probability of death from a certain cause. Relate this graph to Table 2. Many people are afraid of snakes, yet in 1988 no one died from snake bites. Many people fear flying, yet 86 times more people died in auto accidents than in commercial air traffic in 1988.

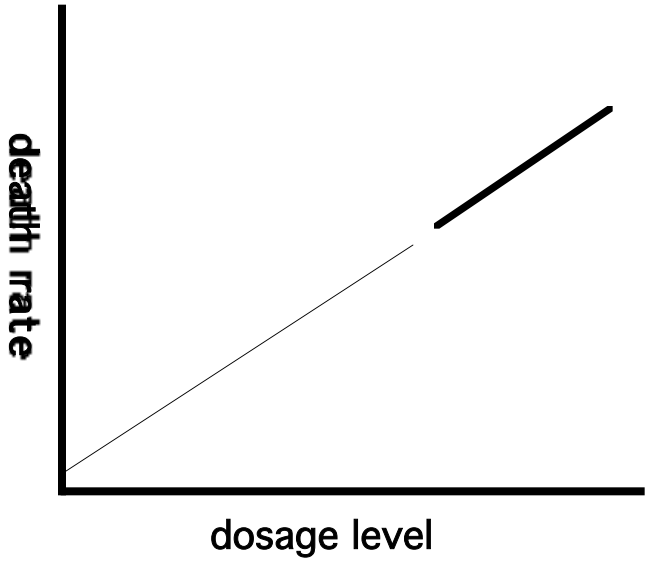

Figure 6: Plot of animal death rate vs. dosage level. The heavy line in the upper-left portion of the graph is based upon death rates of laboratory animals at given high dosage rates. The lighter line is an extrapolation to a chosen risk level (death level) at which a safe dosage can be estimated. If a risk level of one in a million is assigned as acceptable, the corresponding dosage level can be found on the graph.