

## **Quartz - the most abundant mineral species in the earth's crust and a human carcinogen?**

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### **ABSTRACT**

Quartz -- the most abundant mineral species in the earth's crust -- has recently been deemed a human carcinogen, with the main threat to humans being from inhalation and, in turn, the development of lung cancer. This action was taken by the International Agency for Research on Cancer (IARC) and was based upon sufficient clinical and experimental evidence in humans and animals and *in vitro* experiments. As a result, the U.S. Occupational Safety and Health Administration (OSHA) now requires all manufacturers of materials that contain above 0.1% quartz to label their products as "probable human carcinogens." Concurrently, the U.S. Environmental Protection Agency (EPA) required communities to monitor PM10 (particulate matter less than 10 microns in diameter) in the air, because high levels of PM10 have been linked to increased risk of respiratory diseases.

A major component of PM10 is quartz. Long-term inhalation of large amounts of quartz-rich dust has led to various respiratory diseases in the mining industry and other dusty trades, but could low-level exposure to quartz lead to lung cancer in the general population? These recent rulings, and current and future regulations, have serious economic implications on the mining and farming industries in the United States and may even limit the rock and mineral collections students are "exposed" to in academic settings.

### **Introduction**

It has been known since Biblical times (Goldsmith, 1994) and documented since the 1500's (Paracelsus, 1567) that miners and those who work in dusty trades for long periods of times suffer from various respiratory diseases. In fact, based upon observations of miners, Paracelsus arrived at the underlying theory behind toxicology, that "the dose makes the poison." This simple statement becomes lost in today's over-regulated, litigious society. The most common example of over reaction in the mineralogical arena is asbestos exposure. Without question, there were elevated death rates in the pre-regulated asbestos trade industries, but the evolution in belief that high exposure in the trades can kill to the current thought that inhalation of one fiber of asbestos can kill led to a series of regulations and lawsuits which destroyed the asbestos industry in the United States and resulted in considerable panic in the general population (Gunter, 1994; Ross, 1981; Skinner and others, 1988).

Quartz has long been associated with lung disorders in workers in the dusty trades (Goldsmith, 1994; Ross and others, 1993), with the main disease being silicosis. Attempts to correlate quartz to lung cancer in the dusty trades are very difficult because smoking histories for most workers do not exist, but the fear exists. In general, silicosis, and asbestosis, result from long-term inhalation of large amounts of quartz and asbestos. These diseases are not cancers but, nevertheless, are debilitating lung disorders that lead to death in many cases. Regulations and actions that reduce dust levels have greatly minimized the frequency of these diseases, but the fear of even the smallest amount of these minerals has been firmly implanted in the mind of public by the media.

Two types of dust exist: inorganic (i.e., minerals that occur naturally) and human-made combustion products (e.g., hydrocarbons resulting in burning fossil fuels). The general population has been exposed to inorganic dust in the environment since human life began. Currently, the EPA requires communities to monitor PM<sub>10</sub> values and attempt to reduce the values if they exceed EPA guidelines. EPA is proposing new guidelines for PM<sub>2.5</sub>; this smaller size fraction can penetrate deeper into the lungs (EPA 1995, 1996).

Many geologists have a poor understanding of the working of the human lungs and how they are affected by inhalation of dust particles, but most geologists would link silicosis, not lung cancer, to quartz inhalation. The medical community has difficulty understanding mineralogical terminology (e.g., what is meant by polymorphs, crystalline vs amorphous, etc.). In some of the earlier animal studies, the medical community did not distinguish between the different types of asbestos (Gunter, 1994). The regulatory community also struggles with mineral nomenclature; for instance, in some regulations they grouped all the silica polymorphs into one group called crystalline silica (IARC 1987a, 1987b). This paper reviews and summarizes each of the separate areas (i.e., silica mineralogy, lung functions and diseases, epidemiology, and regulations) needed to understand the health effects of inhalation of quartz dust and should provide a good introduction to students in the geosciences of the interdisciplinary methods required to understand problems faced by our society and how we, as geoscientists, might help solve these problems.

### **Silica - forms and geological occurrences**

Silica is the term given to the oxide SiO<sub>2</sub>. Approximately 47 weight % of the earth's crust is composed of O and 28 weight % is composed of Si, making O and Si by far most abundant elements in the earth's crust (Heaney and others, 1994). Silica, in turn, is by far the most abundant oxide in the earth's crust. Pure silica can occur in either crystalline form (i.e., minerals) - with quartz as the most abundant species- or in amorphous form (e.g., opal). Silica is also the major component of the silicates. For instance, the chemical formula for the Na feldspar albite, NaAlSi<sub>3</sub>O<sub>8</sub>, can be rewritten in terms of its oxide components as 0.5(Na<sub>2</sub>O), 0.5(Al<sub>2</sub>O<sub>3</sub>), and 3(SiO<sub>2</sub>). While the feldspars are the most abundant mineral *group* in the earth's crust, approximately 60%, quartz is the most abundant mineral *species*, approximately 12% (Klein, 1993).

There are six naturally occurring polymorphs of SiO<sub>2</sub>: quartz, tridymite, cristobalite, coesite, stishovite, and moganite. All of the silica polymorphs are composed of silica tetrahedral, (SiO<sub>4</sub>)<sup>4-</sup>, in which each oxygen is shared by two tetrahedrons. This sharing provides for charge balance, and the different polymorphs are just different arrangements of the tetrahedrons. Different arrangements occur at different pressures and temperatures (Figure 1) and result in different physical properties (Table 1).

Quartz is the stable polymorph on the earth's surface. It forms in cooling magma chambers and in near-surface aqueous environments. In Figure 1, there are two fields for quartz - low quartz and high quartz (also referred to as α-quartz and β-quartz). The low and high distinctions can relate to low and high temperatures or to low and high symmetry. Figure 2A represents the low quartz structure and Figure 2B represents the high quartz structure. The phase transformation from low to high quartz requires only bond bending and is nearly instantaneous; thus, only low quartz exists at surface temperatures. This type of polymorphic transformation is termed as displacive, because structural units are displaced. Low quartz has a three-fold

screw axis in the center of the ring, while high quartz has a six-fold screw axis in the center of the ring.

At higher temperatures, but still low pressures, cristobalite and tridymite are the stable forms of  $\text{SiO}_2$ . These minerals form in volcanic eruptions and also occur as alteration products of opal and silica-rich volcanic ashes. The structure of these two minerals is based upon a six-member ring of  $\text{SiO}_4$  tetrahedrons in which alternating tetrahedrons point up and down (Figure 2C). These rings polymerize into a sheet (Figure 2C), and then these sheets are stacked in different arrangements to produce the two polymorphs. Low and high structure types also exist for these minerals and, like low to high quartz, the low to high conversion is nearly instantaneous with temperature; thus, only the low polymorphs for quartz, cristobalite, and tridymite are given in Table 1. Cristobalite and tridymite exist on the surface because the conversion from the sheet-like structures which compose them to the low quartz structure requires breaking the Si-O bonds which hold the tetrahedrons together. This type of polymorphic transformation is termed reconstructive, and it requires considerable energy and time.

The rare high-pressure polymorphs, coesite and stishovite, were only discovered in 1950's in a meteor crater. Coesite also has recently been found associated with diamonds (Heaney and others, 1994). Stishovite is the only polymorph not composed of silica tetrahedrons. In stishovite, six oxygens surround the Si, resulting in a denser mineral (Table 1). This is good example of the crystal chemical relationship that higher pressures favor higher coordination numbers. The remaining polymorph, moganite, was first observed microscopically in the 1890's as intergrowths with quartz in chalcedony. Structural analysis of moganite obtained from volcanic rocks in Spain confirmed this new silica polymorph (Heaney and others, 1994). Its density is between that of quartz and cristobalite.

Amorphous silica occurs in the geological environment as opal and in silica-rich volcanic ashes. Opal is commonly given the chemical formula  $\text{SiO}_2 \cdot n\text{H}_2\text{O}$  to denote a variable amount of non-structural water. A gradation exists from amorphous to crystalline silica. Silica compounds are based on the silica tetrahedron as the basic building block. "Amorphous" silica lacks a long-range (~100 nm) ordering of silica tetrahedrons, and opals occur with different crystallinity. Opal-A (the A denotes amorphous) does not diffract X-rays. It is unstable and will alter to quartz through the steps: opal-A  $\rightarrow$  opal-CT  $\rightarrow$  opal-C  $\rightarrow$  moganite plus quartz  $\rightarrow$  quartz. The "C" and "T" refer to cristobalite and tridymite. During these alterations, the water content of the opal decreases. These alterations are common in volcanic ashes, and these ashes are quite often a complex mixture of different phases of amorphous and crystalline silica.

### **Diseases associated with inhalation of silica and lung function**

Most people think of silicosis when quartz and lung disease are associated; however, with the recent classification of quartz as a human carcinogen (IARC, 1987, 1996) lung cancer is now also associated with quartz inhalation. Silicosis is most certainly related to inhalation of large amounts of quartz dust, especially in the pre-regulated workplace. It is important to note that silicosis is *not* a cancer. It belongs to the family of diseases called pneumoconiosis (from the Greek, pneuma - breath and konis - dust). Asbestosis and black lung are other examples of pneumoconiosis. Silicosis is a rare disease; only 117 deaths were reported in the U.S. in 1992. The number of lung cancer deaths for the same period was 145,953 (Table 2).

A basic knowledge of how the lung works and the process of particle clearance from the lung are important in understanding these respiratory diseases. (See Skinner and others (1988) and Goldsmith (1994) for a more detailed discussion on these subjects.) The function of the lung is to replenish  $O_2$  while removing  $CO_2$  from the blood. Air is inhaled through the nose or mouth and passes through the trachea to the bronchus and into the lungs (Figure 3). The  $O_2$ -rich air continues into smaller and smaller bifurcations of the airways (bronchioles, terminal bronchiole, respiratory bronchiole, alveolar duct) until it finally reaches an alveolus, or air sac (Figure 3). An alveolus is hollow sphere with a diameter of approximately 0.3 mm. It is one cell layer thick, with the inside literally exposed to the atmosphere and the outside wrapped in small veins and arteries, the pulmonary arteriole and pulmonary venule (Figure 3). Blood from a venule arrives at an alveolus rich in  $CO_2$  and depleted in  $O_2$ . In the blood-rich sphere encapsulating the alveolus, the  $O_2$  and  $CO_2$  exchange through the single cell thickness of the air sac, and  $O_2$ -rich blood leaves the alveolus in the arteriole. There are approximately 300 million alveolar in the human lungs, and they have a surface area of approximately  $90\text{ m}^2$ .

The heart receives  $CO_2$ -rich blood from the body and pumps it into the lungs through a series of bifurcating veins with ever-decreasing size. The veins “transform” into arteries after  $O_2$ - $CO_2$  exchange has occurred, and the  $O_2$ -rich blood returns through smaller arterioles joining each other until one large artery enters the heart. The heart, in turn, pumps the  $O_2$ -rich blood back through the body for another circuit. Any disease that reduces the lungs’ efficiency for  $O_2$ - $CO_2$  exchange places a larger demand on the heart, because as exchange rates drop, larger volumes of blood must be pumped into the lungs, straining the heart. For this reason, heart disease kills more smokers than lung cancers do.

When foreign particles are inhaled, they can be removed from the respiratory tract by different means as a function of their size. If they are not removed, they are deposited in different portions of the respiratory tract as a function of their size, and this may result in different diseases. Particles with a diameter greater than  $15\text{ }\mu\text{m}$  are deposited in the upper nasal tract, those with  $10\text{-}15\text{ }\mu\text{m}$  diameter are deposited in the lower nasal tract, those with  $5\text{-}10\text{ }\mu\text{m}$  diameter are deposited in the trachea or the bronchus, and those below  $5\text{ }\mu\text{m}$  diameter can penetrate into the terminal bronchiole and alveolus and disrupt the lungs’ function (Goldsmith 1994).

A human inhales approximately  $10\text{ m}^3$  of air per day (Skinner and others, 1988). Air contains particles of various sizes and types. In 1986, the EPA set guidelines for PM10 because this size particle was linked to increases in respiratory diseases (EPA, 1986). “Clean” outside air has PM10 levels of approximately  $10\text{ }\mu\text{g}/\text{m}^3$ . Assuming a density of  $2\text{ g}/\text{cm}^3$  for the PM10, there would be approximately 250,000 five-micron diameter particles in one cubic meter of air. Thus, a human would inhale about 2,500,000 five-micron diameter particles per day.

The body rids itself of these particles by physical or chemical means. The main line of defense is the ciliated-lined upper airway. Cilia cells, hair-like in shape, move inhaled particles up the respiratory tract to be purged from the body in saliva. If the particles are deposited in the smaller bronchioles or alveolar, they are removed by white blood cells in the lungs, called macrophages. These macrophages engulf foreign particles and may dissolve them if they are soluble. Quartz is basically insoluble, while chrysotile asbestos appears to be soluble (Gunter, 1994). Also, the particles can be carried away in the pulmonary lymphatic system and deposited in lymph nodes (Goldsmith, 1994).

Three types of silicosis exist which relate to length of exposure and particle size. *Acute silicosis* is very rare and results from very high, short-term exposure to very fine quartz dust. Large amounts of quartz inhaled over a short time period cause fluid to accumulate in the alveolar of the lung, similar to pulmonary edema (Goldsmith, 1994; Lang 1998).

*Chronic silicosis*, the most common form of silicosis, results from long-term inhalation (i.e., greater than 10 years) of quartz particles (Goldsmith, 1994). Particles with a median size range of 0.5 to 0.7  $\mu\text{m}$  are phagocytosed by macrophages (i.e., encapsulated by white blood cells) (Ross and others, 1993). In the process, the cells release materials which form scar tissue in the lung. The scar tissue destroys portions of the lungs and reduces the  $\text{O}_2$ - $\text{CO}_2$  exchange process, causing the heart to beat faster to increase blood flow. Death results from heart failure.

*Accelerated silicosis* is intermediate between acute and chronic silicosis in exposure times (i.e., 5-10 years exposure). Both acute and accelerated silicosis seem to result from exposure to freshly fractured quartz, which appears to be more reactive (Goldsmith, 1994). As with chronic silicosis, scar tissue forms in the lung, and death results from heart failure.

The causes of lung cancer, like all cancer, are still poorly understood, but cell mutations and uncontrolled cell growth are the result. The cells no longer function as they were intended. In the lung, or any organ, these cancer cells form tumors and disrupt the lungs' function, reducing the lungs' ability to  $\text{O}_2$ - $\text{CO}_2$  exchange. Also, the cancer spreads throughout the organ and body. It is postulated that when a macrophage engulfs a quartz particle in the lung, the macrophage in turn releases chemicals, such as oxygen free-radicals, that set off a cascading effect, resulting in tumor formation (IARC, 1997).

### **Linking a material to a disease - epidemiology of silica-related diseases**

Substances are linked to diseases by three methods: (1) human case studies, (2) animal studies (i.e., *in vivo* studies), and (3) "test tube" studies (i.e., *in vitro* studies). The first indication that a substance may affect human health usually comes from case studies of people who were exposed to the substance, usually in large amounts for long periods of time. Animal studies are often used to determine safe dose levels for humans. *In vitro* studies are helpful in determining the actual cause of the disease at the cellular level. If the material is being tested as a carcinogen, there must be some biological effect, (such as DNA mutation). It is also necessary to establish a dose-response relationship for the material and the disease in question (i.e., that higher doses yield higher disease rates). A dose-response curve can be made either from case studies of humans or, more frequently, from animal studies. In human case studies, the dose is commonly unknown.

The field of epidemiology involves the study of the causes and relationships of diseases in humans. To determine if one group, for instance granite workers, has a higher incidence of disease, for instance silicosis, than the normal population, epidemiologists would calculate the standard mortality ratio (SMR) for the granite workers with the following equation:

$$SMR = \left[ \frac{O}{E} \right] * 100$$

where,  $O$  is the number of observed deaths in the study group and  $E$  is the number of expected deaths estimated from a reference population. This reference population might be the general population of the U.S., or as is commonly used in studies of miners, a male population with the same age distribution. If the SMR is above 100, the observed group has a higher than expected death rate. If the SMR is below 100, the observed population has a lower than expected death rate.

As is the case with any measurement, it is important to know its variability. The standard error of the SMR can be approximately calculated by the following (Kahn and Sempos, 1989):

$$SE(SMR) \cong \frac{SMR}{\sqrt{O}}$$

where,  $O$  is the number of deaths in the observed population. Thus, as the number of deaths increases, the  $SE(SMR)$  decreases. With large  $SE$ 's, no conclusion can be drawn from an SMR. For instance, if the SMR was 200 but the  $SE$  was 75, a 95% confidence interval would be approximately plus or minus two times the  $SE$  or an SMR of 50-350. When the 95% confidence interval includes 100 in its range, the SMR value would have to be judged non-significant.

For silica, the first sign of concern came from high incidences of silicosis in people employed in quartz-rich, dusty environments. The top part of Table 3 lists some example SMR's for silicosis and lung cancer taken from Ross and others (1993). The table shows SMR values as high as 900 (i.e., nine times the rate of the reference population) for silicosis in granite workers. Also notice that for the granite workers, the silicosis SMR decreases with time, a result of improved quarrying operations (e.g., wet drilling) resulting in lower dust levels. Figure 4 is a plot of deaths in the U.S. from silicosis and asbestosis from 1970 to present. The graph shows that silicosis deaths have decreased since the advent of better dust control. Interestingly, asbestosis deaths are increasing, probably as a result of better diagnosis methods and/or increased litigation.

While a clear dose-response relationship between exposure to quartz-rich dust and silicosis has been shown in granite miners (Table 3), could lung cancer be related to inhalation of quartz particles? To answer this question, we need to examine human case studies, animal studies, and *in vitro* tests. Figure 5 shows a plot of lung cancer deaths in the U.S. from 1970 to 1992. The number of deaths almost doubled in this time. This increase is, of course, directly related to cigarette smoking (Hammond and others, 1978). The risk of dying from lung cancer increases by a factor of 15 for those who smoke and smoking rates increased during the 1950's and 1960's (Goldsmith, 1994). Smoking also destroys the body's first line of defense for clearing inhaled particles, the hairlike cells in the upper respiratory system, and reduces the efficiency of the lung, placing a strain on the heart (Lehnert, 1993). SMR's calculated for any case study attempting to relate lung cancer to an environmental agent, such as quartz, must take smoking rates into account. Very few case studies have been adjusted for smoking rates between the observed and reference populations, thus putting into question any reported lung cancer SMR for quartz inhalation.

Many case studies have been done to examine the relationships between lung cancer incidence in various dusty industries. Ross and others (1993) provide several case studies in which both SMR's from silicosis and for lung cancer are listed

(Table 3). Several of the SMR's for lung cancer are slightly above 100, but smoking histories were not available so it is difficult to interpret them. Goldsmith (1994) and IARC (1997) provide a more extensive review of case studies than Ross and others (1993). Table 3 summarizes their SMR's for lung cancer. The case studies are divided into four areas: ore mining, granite production, foundries, and ceramics. The table lists the number of SMR's, or other statistical tests, for lung cancer and the number of these tests showing a significant increase in lung cancer for these occupations. Next, a percentage of SMR's greater than 100 for each industry in each review paper was calculated. Overall, Goldsmith (1994) found a significant increase in lung cancer rate for 77% of his case studies, while IARC (1997) found an increase in only 37% of the cases. However, in both review papers it was noted that smoking data were unavailable for most studies and that without it, it is impossible to determine if these increased rates of lung cancer relate to quartz exposure or smoking. There is also a synergetic relationship between smoking and other lung diseases because, as stated previously, smoking reduces the lungs' defense mechanisms.

All of the above studies involved high doses in the workplace. What is needed are studies on lower dose levels in the general population. Norton (1996) and Norton and Gunter (1999) conducted a state-wide study in Idaho on respiratory diseases and dust levels. Idaho is a dusty state, and the dust composition is 10-15% quartz statewide. The lung cancer SMR for Idaho, when compared to the U.S., was 84(1). Adjusting for lower smoking rates in Idaho and a younger population raised the SMR to 93(1), still below the national average. We also studied farmers vs. nonfarmers and found the SMR for farmers, 88(3), was well below that for non-farmers, 116(1). Thus, we found no evidence in our study supporting increased rates of lung cancer for those living in a dusty environment.

The IARC (1997) also provides an in-depth review of animal studies. In these studies, quartz is usually injected into the animal trachea. Different amounts are injected into the animals and the animals are "sacrificed" after a certain period of time and lung tumors counted. Quartz was shown to cause lung tumors in rats but not in hamsters and mice. Many people question the validity of animal testing and how well it represents what would happen in a human (Ames and Gold, 1990). *In vitro* testing showed some biological activity (i.e., mutations), but again there is controversy around these methods. In fact, even Bruce Ames, who developed the *in vitro* testing method, questions its usefulness (Ames and Gold, 1990).

## **Regulations**

Occupational exposure to quartz inhalation has been detrimental to workers' health in dusty occupations and reductions in dust levels have led to reduced deaths from silicosis in the U.S. (Figure 4). Reductions in occupational hazards usually result from some type of government regulation. In the U.S., OSHA and EPA are the two main agencies that regulate human exposure to "hazardous" materials. OSHA is involved with regulatory issues in the workplace; their mandate is to regulate occupational exposure. The EPA is involved with exposure limits in the general population; their mandate is to set guidelines for non-occupational exposure. Also, Congress can enact laws it deems appropriate, for instance to protect school children from exposure to asbestos (Gunter, 1994).

Currently, OSHA regulations exist on occupational exposure to mineral dusts in the workplace. The limit for inert respirable dust is 5 mg/m<sup>3</sup> of air, time-averaged over an eight-hour work day. Inert dust is defined as any dust which has shown no toxic effect to humans. Limits are also placed on the amount of respirable quartz,

cristobalite, and tridymite as function of the dust content of the air and the composition of the dust. For quartz, the current exposure limit is  $(10 \text{ mg/m}^3)$  divided by  $(\% \text{quartz} + 2)$ . If the composition of the dust is 100% quartz, the exposure limit would be  $0.1 \text{ mg/m}^3$  of dust, while if it is 10% quartz, the exposure limit would be  $1.0 \text{ mg/m}^3$  of dust. The exposure limits for cristobalite and tridymite are one-half those of quartz. Thus, OSHA considers the risk from cristobalite and tridymite to be the same and twice that of quartz. For amorphous silica, the exposure limit is  $80 \text{ mg/m}^3$  divided by the percent of  $\text{SiO}_2$  in the dust, so if the dust were 100% amorphous silica, the exposure limit would be  $0.8 \text{ mg/m}^3$ .

OSHA has regulations on carcinogenicity of certain materials; the 1983 OSHA Hazard Communication Act requires that any product containing above 0.1% of a probable human carcinogen be labeled as a "probable human carcinogen." OSHA does not make rulings on a material's carcinogenicity; they use IARC's. IARC's role is to review existing data on the carcinogenicity of materials and classify them into one of five groups: in terms of decreasing carcinogenicity, they are group 1, 2A, 2B, 3, and 4. (See Table 4 for a more thorough discussion of each group.) IARC's reviews are based on the degree of evidence for carcinogenicity in both humans and animals. They use three listings: the material showed sufficient (S), limited (L), or inconclusive(I) evidence as a carcinogen. Based upon these overall ratings, they place materials into one of their five groups (Table 4).

IARC (1987) evaluated crystalline and amorphous silica. Their results (Table 5) classified crystalline silica as a group 2A human carcinogen, meaning that it is probably carcinogenic to humans, and amorphous silica as group 3, unclassifiable as a human carcinogen. In the 1987 ruling, all forms of crystalline silica were treated as one. This 1987 upgrade of crystalline silica (i.e., quartz) required OSHA, based on the Hazard Communication Act, to require manufacturers to label their products as probable human carcinogens if they contained above 0.1% crystalline silica. Although this was not widely publicized, The Wall Street Journal published an article entitled "How sand on a beach came to be defined as a human carcinogen" (Stiff, 1993).

Based upon new experimental data and more evidence for quartz-induced lung cancer in humans, IARC (1997) reevaluated crystalline and amorphous silica. In this evaluation, shown in Table 5, they separated quartz, cristobalite, and tridymite. Sadly, quartz, the most abundant mineral species in the earth's crust, is now listed as a group 1 human carcinogen. Before a material can be classified to this group it must show sufficient evidence for cancer in humans. Based on epidemiological studies, IARC's panel of 19 experts, none of whom were mineralogists or geologists, found this link. Cristobalite was also evaluated to group 1 status, while tridymite remained in group 2A and amorphous silica in group 3.

EPA's job seems even more difficult. They set guidelines for exposure for the general public. Prior to 1986, total suspended solids (TSP's) were monitored in the air. As the name TSP implies, the size fractions were undefined. As discussed earlier, smaller size materials can penetrate deeper into the lung, resulting in greater damage. After reviewing the existing literature on respiratory diseases and dust levels, EPA (1986) set guidelines for PM10 for outside air at  $150 \text{ } \mu\text{g/m}^3$  for a 24-hour period and  $50 \text{ } \mu\text{g/m}^3$  for a yearly period. These guidelines also required cities to monitor their PM10 levels and, if they exceeded EPA guidelines, to develop plans to reduce them.

EPA (1996) evaluated new experimental data since 1987 to determine if their PM guidelines required modification. Since 1987, many studies have been written relating hospital emergency room visits with high PM<sub>10</sub> levels. One of the most famous of these studies, the "Six Cities Study" (Dockery and others, 1993), showed a direct link between air pollution and human health in metropolitan areas. Schwartz (1996) also linked emergency room visits with high PM levels. Many of these studies found that PM<sub>2.5</sub> appeared to be more of a health risk than PM<sub>10</sub>. PM<sub>2.5</sub> is composed primarily of combustion products (i.e., hydrocarbons from cars and factories). Currently, EPA (1996) is proposing to keep its standards for PM<sub>10</sub> but also add new standards for PM<sub>2.5</sub> to be in the range of 25-65  $\mu\text{g}/\text{m}^3$  for a 24-hour period and 12.5-20  $\mu\text{g}/\text{m}^3$  for a one-year period.

The regulatory agencies in the U.S. have not yet responded to the reclassification of quartz as a group 1 human carcinogen. The impact of the reclassification could be devastating to many important industries in the U.S., including mining and farming. Quartz occurs everywhere, and there is no feasible way to obtain low levels (i.e., less than 0.1%) in the natural geological environment.

### **Conclusions**

There is no doubt that long-term inhalation of high levels of quartz-rich dust has caused silicosis in the occupational setting and that regulations were required to reduce these dust levels to curb this rare disease. Based upon case studies, there may be a slight increase in the risk of lung cancer for people who work in jobs that expose them to high levels of quartz-rich dust; however, smoking clouds these case studies.

Confusion exists about such terms as silica, crystalline silica, and amorphous silica in the regulatory agencies. Many regulations have been made, and continue to be made, with little or no input from the mineralogical or geological communities. Geologists also tend to associate quartz and silicosis, and many have no knowledge of the recent classification of quartz as a human carcinogen. The geological community needs to learn more about the health effects of mineral dusts and become involved in this public policy issue before it is too late.

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### Tables

Table 1: The names of the six naturally occurring silica polymorphs, their crystal systems, and density.

mineral	symmetry	density (g/cm <sup>3</sup> )
quartz	hexagonal	2.65
moganite	monoclinic	2.61
crystalobalite	tetragonal	2.50
tridymite	orthorhombic	2.32
coesite	monoclinic	3.01
stishovite	tetragonal	4.35

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Table 2: Most recent death statistics in the United States for respiratory diseases. COPD's refer to chronic obstructive pulmonary diseases (e.g., bronchitis, emphysema, and asthma). All data from the 1992 (most recent) U.S. Vital Statistics (Sondik, 1996).

total: 2,175,613	black lung: 631
lung cancer: 145,943	asbestosis: 285
COPD's: 91,938	silicosis: 117

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Table 3: Summary of review papers by Ross and others (1993), Goldsmith (1994), and IARC (1997). The top part of the table gives lung cancer / silicosis SMR's for four occupational settings. The first SMR is for lung cancer and the second is for silicosis. The numbers in parentheses represent standard errors for the SMR's. The bottom of the table shows the number of case studies with a significant increase in lung cancer vs. the number of case studies given in these two review papers. Also, a percentage is calculated for the number of studies that show a significant increase in lung cancer rates for these occupations (e.g., IARC (1997) reviewed 137 case studies in four occupations and found a significant increase in lung cancer rates for 50 of the studies, or 37%).

Ross and others (1993): lung cancer SMR / silicosis SMR

Homestake gold miners:	100(15) / 279(38)	
Australian gold miners:	140(18) / 640(193)	
Diatomaceous workers:	143(19) / 329(51)	
Vermont granite workers:		
pre 1930	1930-39	1940-49
128 / 919	125 / 421	95 / 90
		1950-59
		51 / 0
		1960-69
		91 / 0

Case studies taken from	Goldsmith (1994)	IARC (1997)
Ore mining	23 vs 35 -> 66%	11 vs 46 -> 24%
Granite production	9 vs 10 -> 90%	11 vs 30 -> 37%
Foundries	16 vs 17 -> 94%	8 vs 15 -> 54%
Ceramics	none	20 vs 46 -> 43%
Overall	48 vs 62 -> 77%	50 vs 137 -> 37%

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Table 4: Classification system used by IARC. IARC places a material into one of these five groups based upon reviews of existing scientific literature (IARC, 1997).

- Group 1:** The agent (mixture) is carcinogenic to humans. The exposure circumstance entails exposures that are carcinogenic to humans.
- Group 2A:** The agent (mixture) is probably carcinogenic to humans. The exposure circumstance entails exposures that are probably carcinogenic to humans
- Group 2B:** The agent (mixture) is possibly carcinogenic to humans. The exposure circumstance entails exposures that are probably carcinogenic to humans
- Group 3:** The agent (mixture, or exposure circumstance) is unclassifiable as to carcinogenicity in humans.
- Group 4:** The agent (mixture, or exposure circumstance) is probably not carcinogenic to humans
-

Table 5: IARC's evaluation of the carcinogenic risk of silica (IARC 1987, 1997), where S represents sufficient evidence, L limited evidence, and I inadequate evidence.

agent	degree of carcinogenicity		overall evaluation
	human	animal	
1987 evaluation:			
silica, crystalline	L	S	2A
silica, amorphous	I	I	3
1997 evaluation:			
quartz	S	S	1
cristobalite	S	S	1
tridymite	I	L	2A
silica, amorphous	I	I	3

### Figures Captions – Figures at end

Figure 1. SiO<sub>2</sub> phase diagram showing 5 of the 6 silica polymorphs. Cristobalite and tridymite occur in high and low phases similar to quartz, but these fields are not shown because only the low phase occurs at surface conditions.

Figure 2: SiO<sub>4</sub> tetrahedral arrangements and the basic building blocks for quartz, tridymite, and cristobalite. A. The basic building block for low quartz with a three-fold screw axis in the ring's center. The numbers next to each tetrahedron represent the Si atom's height above the page. The lower drawing combines the basic building block above to make the structure. B. The basic building block for high quartz with a six-fold screw axis in the ring's center. The numbers next to each tetrahedron represents the Si atom's height above the page. The lower drawing combines the basic building block above to make the structure. The transformation between low and high quartz structures is instantaneous and only requires bond bending between the SiO<sub>4</sub> groups (i.e., a change in the Si-O-Si angle). C. The basic building block for tridymite and cristobalite. These six-membered rings have three tetrahedrons pointing up and three pointing down. These rings, in turn, form sheets (bottom of drawing) which stack together in different ways to produce cristobalite and tridymite.

Figure 3: A schematic view of the human lung at three different scales (modified from Skinner and others, 1988). The left view shows the respiratory tract leading into the lungs with bifurcating airways; the position of the heart is also shown. The middle sketch shows an enlargement of the air sacs, alveolar, located at the end of the respiratory bronchiole. The venule leading from the heart and the arteriole leading back to the heart are also shown. The lower right drawing is an enlargement of the previous sketch showing more details of the individual alveolus and the site of O<sub>2</sub>-CO<sub>2</sub> exchange.

Figure 4: Relationship between the number of deaths from silicosis (solid diamonds) and asbestosis (open boxes) in the United States from 1970 to 1992 (Sondik, 1996). During this time period, silicosis deaths decreased, probably due to better mining procedures (i.e., lower dust levels), while the asbestosis deaths increased, probably due to better diagnosis and/or increased litigation.

Figure 5: A plot of the number of deaths from lung cancers in the United States from 1970 to 1992. Lung cancer deaths continue to increase due to increased smoking rates in the 1950's and early 1960's.

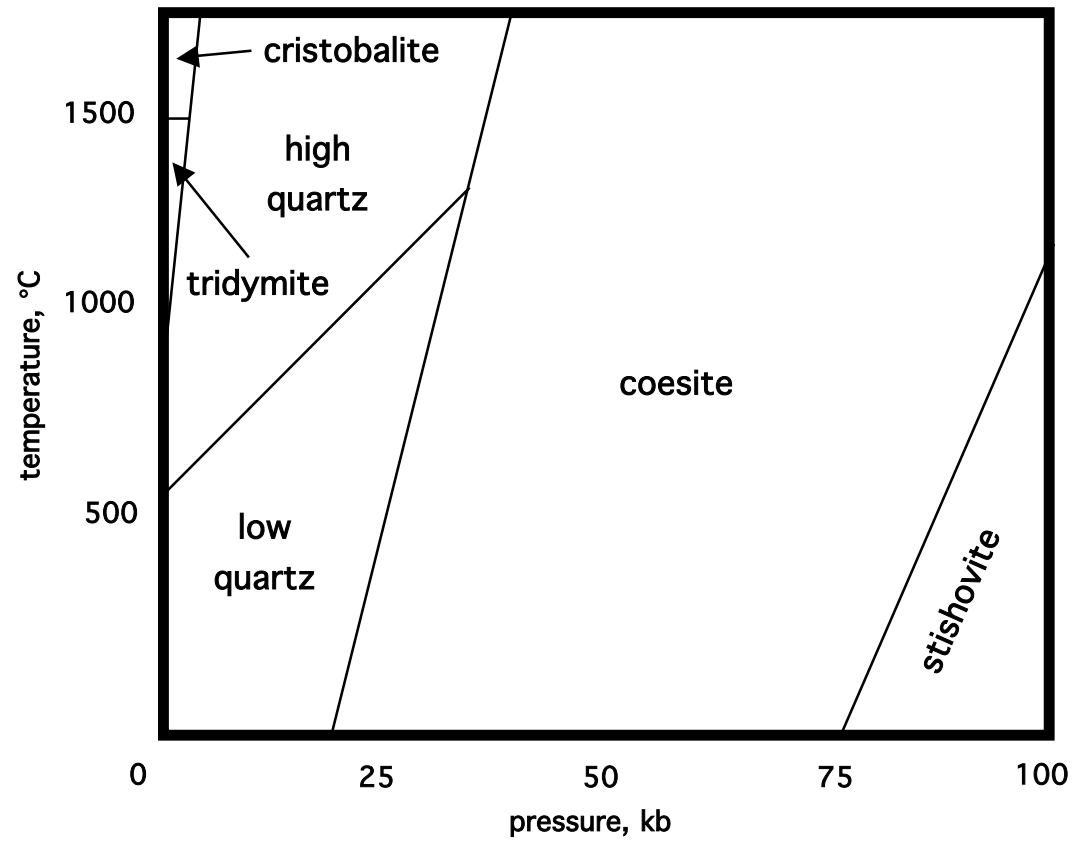
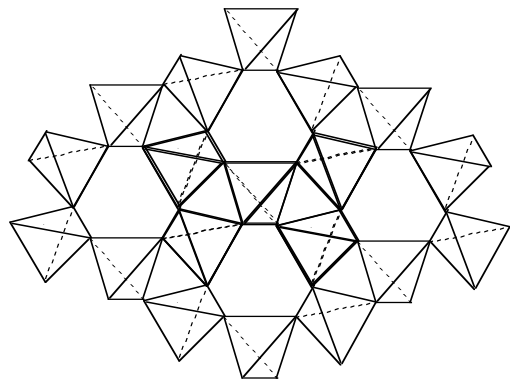
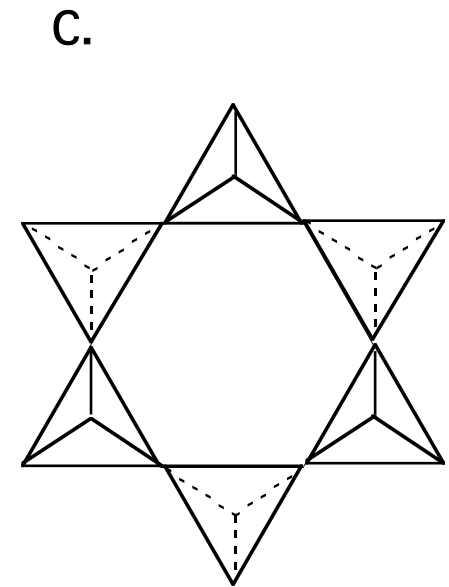
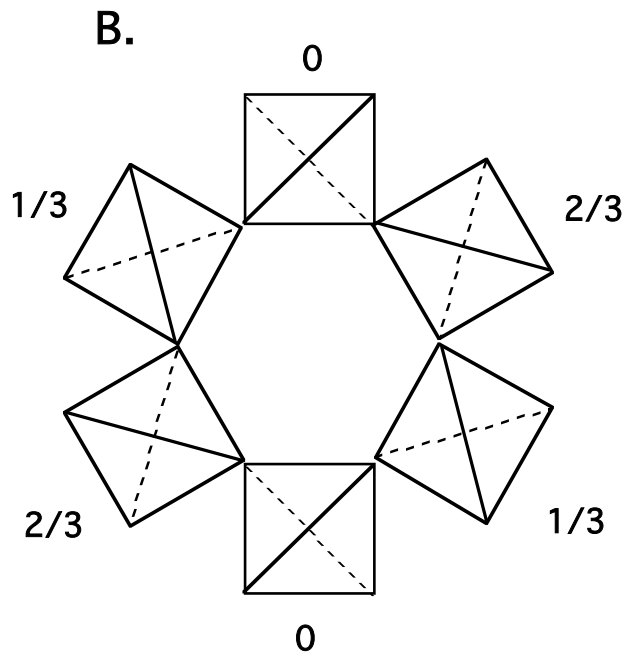
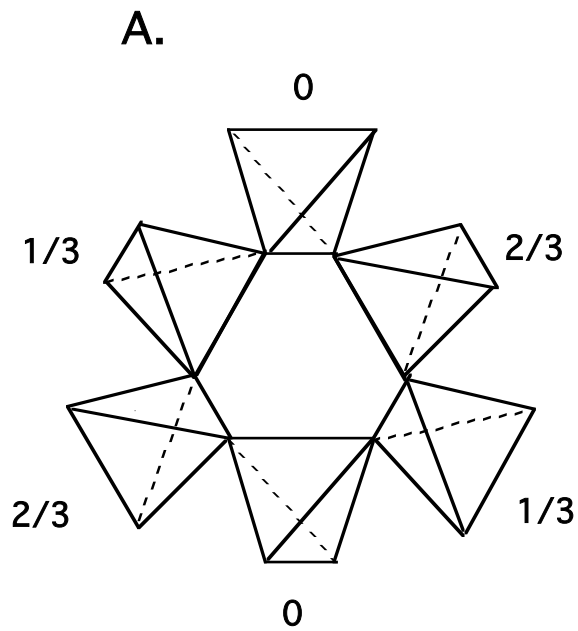
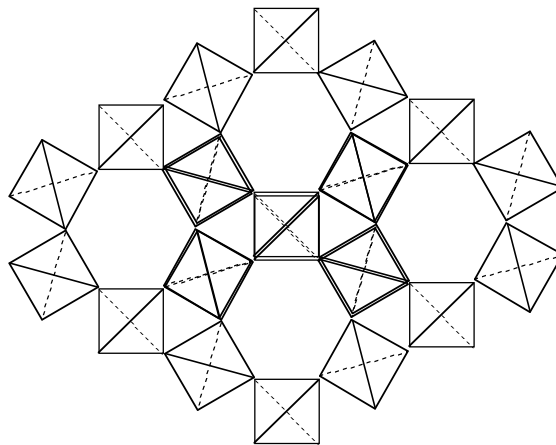


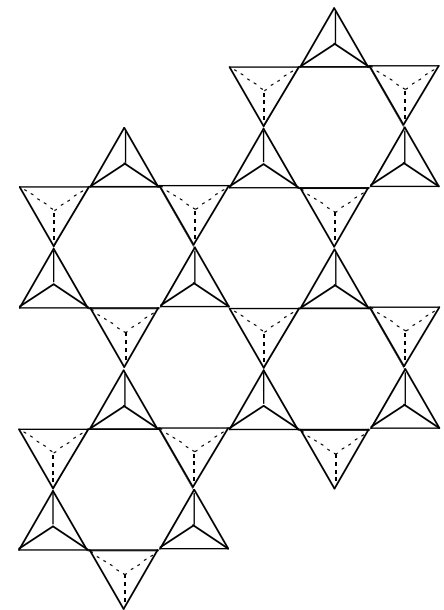
Figure 1



low quartz



high quartz



tridymite - cristobalite

Figure 2

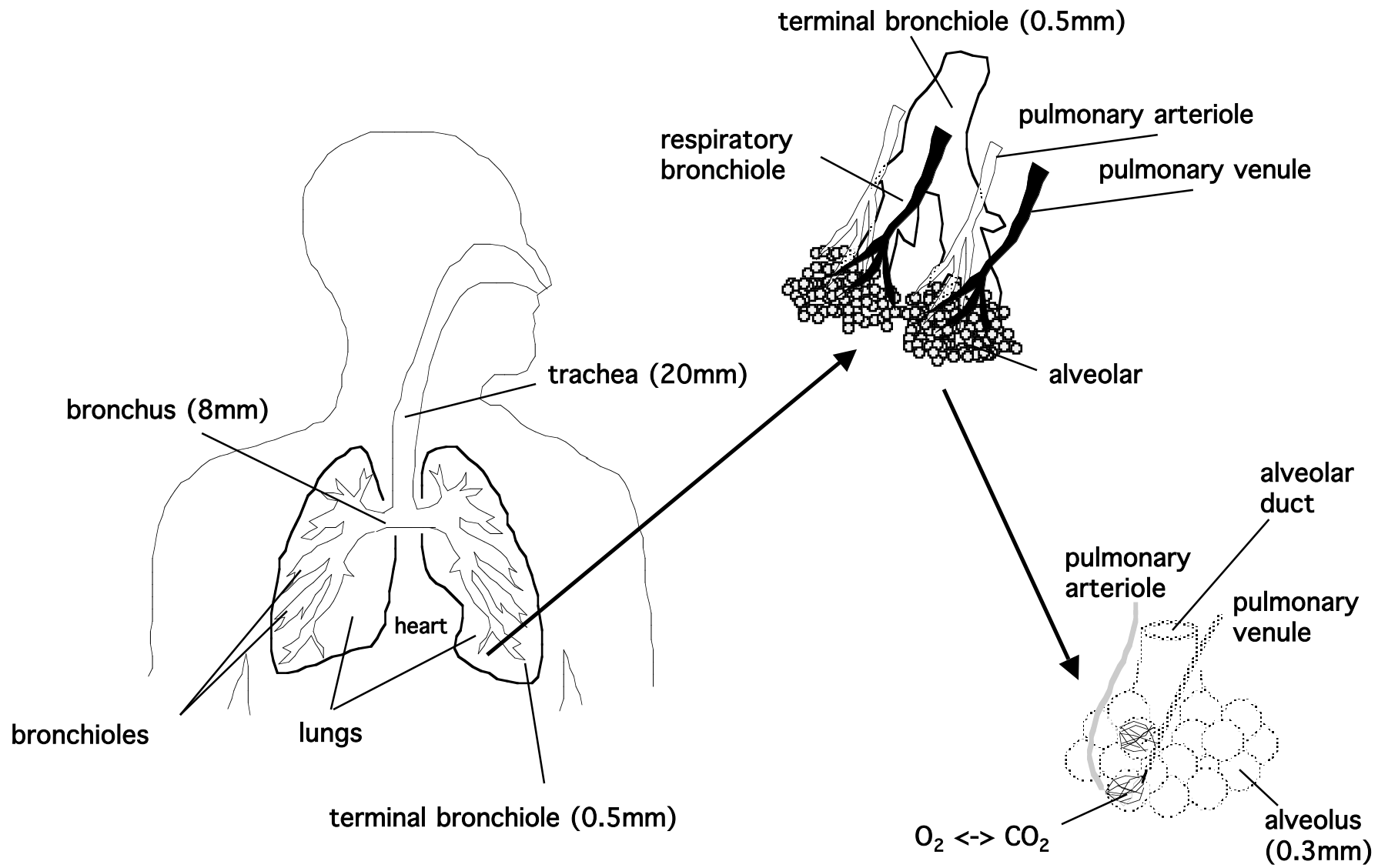


Figure 3

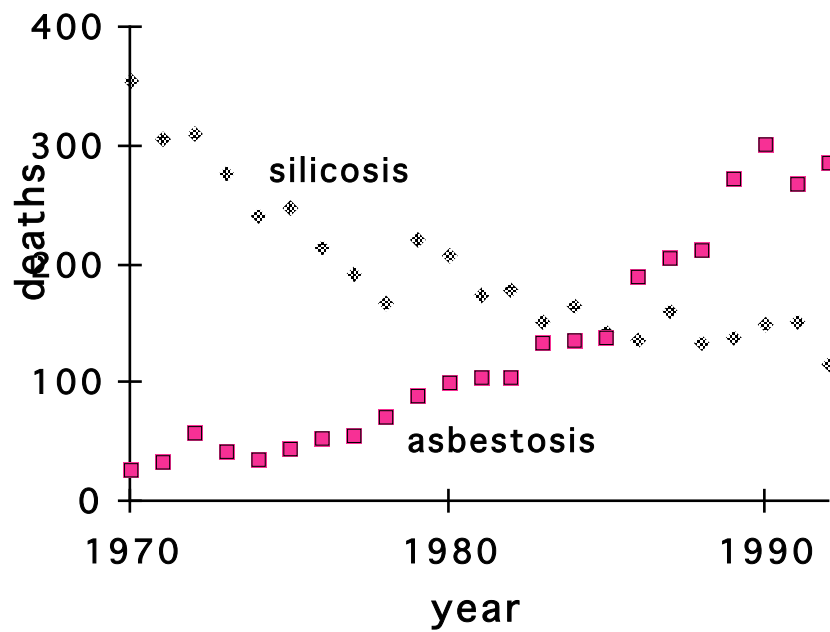


Figure 4

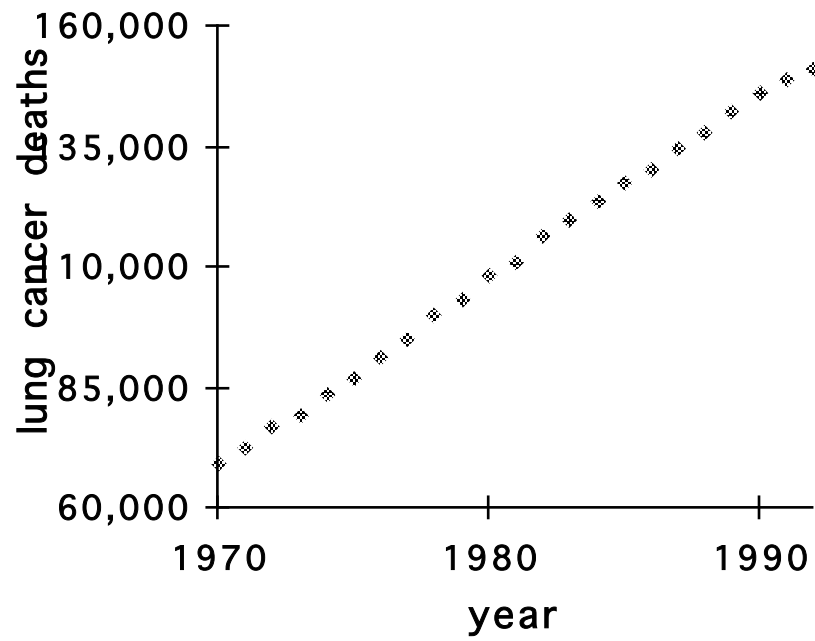


Figure 5