Asbestos Risk Assessment

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Abstract **The aim of this paper is to investigate the hazard, exposure, and toxicity associated with asbestos fibres as a model to assess the risks it poses on human health and public safety. For this purpose, information acquired from research that has explored aspects of asbestos were collectively integrated and analyzed in an unbiased, scientific approach. It was discovered that people who are constantly exposed to asbestos dust from work, are family members of the occupationally exposed, or are residents living near factories that utilize asbestos are most susceptible to asbestos exposure. Toxicity assessments revealed that prolonged exposure to asbestos ultimately causes lung tissue scarring, leading to various lung diseases such as asbestosis, mesothelioma, and lung cancer. The degree of asbestos potency is highly dependent on chemical composition, size, shape, durability, and clearance. However, a lack of consistent information provided in literature that indicate these factors, along with information based on sampling size, duration and amount of exposure, air sampling techniques, and appropriate controls, continue to plague stakeholders from reaching appropriate conclusions based on the risks identified. Finally, despite the success of asbestos monitoring methods and risk communication via the mass media in reducing the use and production of asbestos, asbestos is still a major public health concern. Consequently, more involvement from the academic community is still needed to precisely quantify the probability and characterize the risks of exposure in order to fill in the knowledge gaps that remain uncertain for both the hazard and exposure aspect of the risk equation.**

Introduction

Risk perception is a subjective judgment that assessors make to characterize the severity of a risk before deciding its priority (Bebbington *et al*., 2001). When a certain hazard is imminent in the environment and is expected to spread beyond a particular safety boundary, it can quickly modify a stakeholder's perception of risk, especially if it threatens to decrease the quality of life and human health of those exposed to the substance. There are many contributing factors that lead people to associate certain substances as being harmful. For many, mass media, as well as the influence of culture on society, collectively play a role in providing information about certain issues to the public, which help stakeholders decipher whether it is relevant to their lives. Unfortunately, the media has a tendency to distort facts or to report them with a slant to make headlines; consequently, most people fail to integrate, analyze, and reach a conclusion based on the assumed problem (Gunter, 1994). The word *Asbestos* means different things to different people, ranging from an excellent all-in-one sound absorbing wall insulator from an engineer's perspective, to a nightmare for a bedridden lung cancer patient surrounded by asbestos-insulated walls. In order to accurately address and evaluate the health risks associated with the exposure to asbestos fibres and its related compounds, a thorough investigation involving unbiased, factual information is required. The purpose of this document is to provide an unbiased, detailed overview of the research that has explored aspects of asbestos in Canada and internationally and to evaluate the risks it poses to human health and public safety addressed from a diverse field of disciplines.

What is Asbestos?

Depending on the audience, the answer to this question can be given in numerous ways. To a scientist, asbestos is a generic term for a number of geologically similar, but not necessarily chemically related minerals (Milano, 2009). To a physician, it is a carcinogen capable of damaging the lungs. To a civil engineer, it is an inexpensive, thermal insulator. A lawyer may answer with a possible lawsuit in mind, while a stakeholder may perceive it as a health concern.

The term *asbestos* comes from the Greek word *asbesta*, meaning *indestructible*, *imperishable*, or *noncombustible* (Cugell & Kamp, 2004). It is a naturally occurring silicate mineral with long, thin fibrous crystals. Many minerals possess asbestiform characteristics, but only six specific minerals from two distinct groups are used industrially: Amphibole and serpentine. The amphibole fibres are straight and are arranged in long linearly organized chains (Finley *et al*., 2008); it encompasses amosite, crocidolite, anthophyllite, actinolite, and tremolite. The serpentine group, which represents 95 percent of manufactured asbestos, is composed of chrysotile – a softer, more fibrous form of asbestos arranged in large parallel sheets (Finley *et al*., 2008). A list of properties for each type of asbestos mineral is presented in Table 1. These minerals form in metamorphic terrain where amphibole and serpentine rocks are subjected to uniaxial tensile strain (Gunter, 1994). This causes the fibre to grow

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parallel to its principal strain axis during geological formation (Figure 1).

Due to its wide range of desirable physical properties, asbestos has been used in many industrial applications. It is highly resistant to mechanical traction, possesses low electrical conductivity, flame-retardant and high-temperature resistant, able to absorb and retain micro-organisms in the interweaving of the fibres (Kivman *et al*., 1978), tensile, and able to withstand wear and abrasion. Finally, asbestos fibres have no detectable flavour or odour; this property makes detecting its presence expensive.

Problems Associated with Asbestos

After 20 years from the beginning of the industrial use of asbestos in Europe, as described in Normandy, France, in 1906, the first cases of pulmonary fibrosis – deep tissue scarring of lungs – were documented in the textile industry that used asbestos (Greenberg, 1994). The first case of *asbestosis* was observed in Britain in 1900, where all ten individuals working at the facility died of lung disease around the age of 30 (Greenberg, 1994). To date, three major diseases are associated with asbestos exposure: Asbestosis, mesothelioma, and lung cancer.

Asbestosis: This term defines a particular pneumoconiosis, a progressive, irreversible parenchyma lung disease caused by the inhalation of fine silica dust, as a result of long-term exposure to asbestos. Its diagnosis is based on clinical history, occupational history, and chest x-rays, in accordance to the guidelines outlined in the International Classification of Radiographs of Pneumoconiosis. X-rays in category zero are considered normal, while categories one (a), two, and three, represent abnormal results (ILO, 1980) (Figure 2).

Mesothelioma: Epidemiological studies suggest that 75 to 80 percent of cases related to malignant mesothelioma of the pleura are associated with exposure to asbestos. About 80 percent of cases occur among workers exposed to asbestos in the workplace (Gee & Morgan, 1989). In this disease, malignant tumour cells develop from mesothelial cells of peritoneum, pleura or pericardium – protective lining that covers most internal organs. Mesothelioma usually results in death within one to two years of its diagnosis and has a latency period of 35 to 40 years (Ross, 1984).

Lung Cancer: The risk of lung cancer depends on a number of factors, such as the dose, fibre type, type of work and duration of exposure, smoking history, and the presence of pulmonary fibrosis (Gee & Morgan, 1995). There is no clinical, radiological or pathological test that can distinguish lung cancer caused by smoking or other carcinogenic potential, since the histological distribution of asbestos-related cancers is similar to lung cancers that affect smokers without asbestos exposure (Gefter *et al*., 1992). In Canada, asbestos accounted for 219 documented cases of lung cancer deaths from 2000 to 2005 (Statistics Canada, 2009).

Exposure Assessment

To assess the level of asbestos toxicity on human health, exposure assessments using models and simulations must be integrated as a starting point in understanding the short-term and long-term effects to which it contributes. For nearly a century, the presence of asbestos fibres lingering in the air has been acknowledged as a health hazard by the scientific community; consequently, an extensive amount of human exposure data exists in literature, both in occupational and natural environmental exposures (Erdal & Esmen, 1990). Like most toxicants, asbestos is only harmful when it enters the body; therefore, modeling its route of exposure must be evaluated prior to making assumptions that certain diseases are linked to its presence.

Although some mesotheliomas can occur without a history of exposure (Arblaster *et al*. 1999), nearly all cases reported in Canada are thought to be related to asbestos exposure. Unlike PAHs and DDT, asbestos is part of the natural environment; therefore, the likelihood of unintentional exposure should not come as a surprise, especially if one has lived or worked in a building that contained asbestos or came in contact with the clothing of industrial workers.

To identify the presence of asbestos fibres within a given location, ambient air samples are collected and observed for the following mineralogical properties: Morphology, crystallography, colour, appearance, optical properties, and hardness of a specimen (Erdal & Esmen, 1990). Compared to data obtained in occupational cohort-based studies, nonoccupational data is to some extent haphazard in terms of consistency. For instance, some studies indicate that a considerable amount of asbestos fibres exist in schools and public buildings, while data obtained from other sources are mainly scattered (Table 2). In a study conducted by Case *et al*. (2002), pleural mesothelioma among female residents of Québec's asbestos mining regions were compared to controls for residential, domestic

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and occupational asbestos exposures. They learned that between 1970 and 1989, ten women suffering from mesothelioma resided in the mining region at the time they were diagnosed (ten cases or 22.7 cases per million), while 108 others lived elsewhere in Québec at the time of diagnosis (108 cases or 2.1 per million). Thus, women in the mining regions had 10.8 times more mesothelioma than women elsewhere in Québec. This study suggests that the risks associated with asbestos-exposure are higher in places where asbestos is more likely to be present, such as in an occupational or paraoccupational setting. This relationship is highlighted in Table 2, where amosite levels analyzed downwind of a factory were far greater than most public areas.

In a similar study conducted by Arblaster *et al*. (1999), it was investigated whether the amount of asbestos retained in the lungs of mesothelioma sufferers differed in three different groups: Occupational, paraoccupational, and residential exposure. Although many of the paraoccuptational mesothelioma cases showed similar lung fibre concentrations to occupational, it was apparent that the nonoccupational mesothelioma cases ranged several orders of magnitude lower than occupational in fibre count. This study suggests that people who work in asbestos-based product manufacturing industries are more susceptible to asbestos exposure than nonoccupational groups. In addition, the author speculates that merely living near an industry that uses asbestos, such as the examined population, can increase the risk of developing mesothelioma.

The ingestion of asbestos fibres as a route of exposure has been investigated in past literature and also deserves mention. Generally, asbestos is introduced into water by the dissolution of asbestos-containing minerals, as well as from industrial effluents, atmospheric pollution, and erosion of asbestos-insulated pipes in water distribution systems (WHO, 1996). In a national survey of the water supplies of 77 communities in Canada, chrysotile was the predominant type of asbestos detected, with fibres ranging in length from 0.5–0.8 μm. It was estimated that concentrations were >1 million fibres per litre (MFL) in the water supplies of 25 percent of the population, >10 MFL for 5 percent of the population, and >100 MFL for 0.6 percent of the population. Concentrations were also higher in untreated water than in treated drinking water (WHO, 1996).

Toxicity Assessment

Despite reductions in use and production worldwide, the risk of asbestos-related illnesses is still a major health concern, especially since vast amounts of asbestos-containing products were manufactured, processed, and used over the past century (Kamp, 2009) and its long latency period that averages between 35 to 40 years from initial exposure to the first appearing symptoms (Ross, 1984). The toxic effects of asbestos inhalation depend on the cumulative dose, the time of initial exposure, and both the physical and chemical properties common to the different asbestos fibres (Kamp, 2009).

To gain an appreciation of the mechanism of damage induced by asbestos fibres, the four quintessential pillars of toxicity assessment must be explored, namely: Absorption, distribution, metabolism, and excretion. Since the main route of exposure is inhalation, the lungs, which comprise the tracheobronchial and the pulmonary or alveolar region, are the focal point where asbestos fibres initiate toxicity (Bignon *et al*. 1978). Histologically, fibrosis begins around the respiratory bronchioles in the lower lobes, where asbestos tends to accumulate and get absorbed upon inhalation (Gefter *et al*., 1992). In an acute study conducted by Brody *et* al. (1989), it was reported that lung damage to endothelial and smooth muscle cells of the arterioles and venules near the bifurcations surrounding this region, occurred within 19 to 72 hours subsequent to exposure in rats treated with four million fibres per m^3 of chyrsotile. Fibres deposited in the bifurcations of alveolar ducts induce alveolar macrophages to attack at the site of deposition (Kamp, 2009). When the macrophage engulfs the foreign fibre, it produces cytokines such as fibronectin – a glycoprotein that recruits and initiates the proliferation of fibroblasts. In addition, the macrophages release excessive amounts of growth factors which act synergistically to produce an event cascade that leads to proliferation of more fibroblasts and collagen fibre deposition (Maxim & McConnell, 2001). As a result, fibrosis and scar accumulate which irreversibly alter pulmonary structure and function. Accumulating evidence also suggests that once macrophages engulf asbestos fibres, it forms and releases reactive oxygen species, such as hydrogen peroxide and superoxide anion O^{2-} (Kamp & Weitzman, 1999), as well as plasminogen activators which, in turn, activate urokinase-type plasminogen activator receptors on the surface of human mesothelial cells (Hamilton *et al*., 1999). Once activated, damage to lung tissue occurs which further contributes to the future remodelling of the lung tissue attacked (Hamilton *et al*., 1999). Overtime, asbestos fibres deposited in the lungs are encapsulated by proteinaceous iron-containing mucopolysaccaride, forming redox-active *asbestos bodies* (Kamp, 2009). A histological characteristic of asbestosis is the presence of pulmonary fibrosis in association with numer-

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ous asbestos bodies (Gee & Morgan, 1995). A schematic depicting these cellular events is illustrated in Figure 3.

Unlike other carcinogens, asbestos cannot be distributed through the bloodstream. If penetration does occur in lung or gastrointestinal tissue, it is extremely limited (Cook, 1983). Consequently, asbestos cannot be metabolized – once inhaled, it is retained for life. Seshan (1983) reported that simulated gastric juices can alter the physical and chemical properties of chrysotile and crocidolite fibres, but it does not stimulate metabolism. The low pH inside alveolar macrophages has also been speculated to weaken inhaled chyrsotile fibres and facilitate destruction (Finley *et al*., 2008). Finally, nearly all *ingested* fibres are excreted in the feces within 48 hours.

The idea linking asbestos genotoxic and carcinogenic potential through ingestion is a controversial supposition based on the notion that ingested fibres that come in contact with epithelial cells lining the lumen will penetrate the gastrointestinal tract and be distributed throughout the body. However, in a chronic study conducted by Chouroulinkov & Truhaut (1989), rats that were exposed to high *oral* doses of chrysotile and a mixture of chrysotile and crocidolite over a two year period showed no toxic or carcinogenic effects. Moreover, rats that were administered 0.5 to 50 mg chrysotile for 14 months, in another study showed significant increases in thymidine incorporation in the gastrointestinal tract (RAIS, 1995), suggesting that ingestion may interfere with DNA metabolism. As of yet, no oral studies have been conducted on humans, largely because of the difficulty in controlling external factors that influence the exposed tissue. Overall, to be a significant health concern, asbestos must either be inhaled, or to a lesser extent, ingested.

Size and shape of fibres play an important role in the potency and pathogenesis of asbestos-related illnesses. In a chronic study conducted by Layard *et al.* (1981), the occurrence of neoplastic tissue in rats correlated well with the dimensional distribution of asbestos fibres. They discovered that fibres ≤1.5 μm in *diameter* and >8 μm in *length* yielded the highest incidence of soft tissue sarcoma of the pleural cavity. If a fibre is longer than the macrophage diameter, it has the potential to reside in the lung for longer periods of time (Maxim & McConnell, 2001). In contrast, fibre ≤8 μm are often inactivated by phagocytosis (Layard *et al.*, 1981) and removed from the lung either directly to the conducting airways or via lymphatics to regional lymph nodes (Maxim & McConnell, 2001). Pott (1978) provides a hypothetical view of how the carcinogenicity of asbestos varies in terms of length and diameter (Figure 4).

To date, all levels of asbestos exposure studied have demonstrated clinical effects. However, most literature published on dosimetry of the carcinogenic potency of asbestos is based on rats, as opposed to humans. Consequently, there are still many difficulties outlining precise doses and of what type of fibre will initiate pathogenic events in lung tissues. Although the risk of asbestos-related diseases is dose-response related (IPSC, 1998), a generalized dose-response curve is inexistent for asbestos in human studies due to, and not limited to, time constraints (long latency period), extent of control, nature of the material, and external influences such as cigarette smoking (Kava, 2007). Dosimetry models that are available from cohort studies indicate that, on a normalized basis, fibre deposition and clearance rates are lower in humans than rats; this is because humans are generally much more sensitive (by a factor of 10^2 to 10^3) to fibres than rodents (Maxim & McConnell, 2001). Consequently, the use of rats as a model species to assess asbestos toxicity is limited. An interspecies comparison of various asbestos exposure parameters in rodents and humans are shown in Table 3.

Risk Characterization

Before characterizing the risks attributed to asbestos exposure, it is helpful to understand the relationship that exists between risk and probability. Risk is the probability of an event multiplied by the consequence. Hence, if the consequence is very large, such as developing a disease, the risk will be high even if the probability is very low. On the contrary, if the consequences are minor, the risk will be low even if the probability is very high. Since the consequences associated with asbestos-related illnesses lead to death, the exposure to asbestos poses great risk even through the probability of inhaling toxic doses is low.

Three methods are predominantly used to evaluate risk of a particular hazard, namely: (1) In vitro testing, where the effects of the substance are determined at the cellular level, (2) in vivo testing, where laboratory animals are exposed to substances under controlled conditions and monitored for the spontaneous development of a disease, and (3) cohort studies of humans exposed to a material, where the death rates and exposure levels are known (Gunter, 1994). Based on the majority of the results obtained in all three approaches, one aspect that remains consistent is that the greater the amount of exposure and the longer the time of exposure, the greater the risk of as-

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bestos-related illnesses. Figure 5 depicts how the exposure to various asbestos fibre sizes can contribute to the number of deaths per 1000 workers. Unfortunately, the same problem exists with this model as in other models that suggest an association between exposure to asbestos and asbestosis, mesothelioma, and lung cancer, that is, there is a lack of consistent information indicating fibre type, size, and duration of exposure. This limits the ability to quantify the probability and to characterize the risks to humans, for both the hazard and exposure aspect of the risk equation (Kava, 2007).

Chyrsotile and amphibole asbestos are the types most commonly used in factories and commercial applications, and growing evidence suggests that they differ in respect to toxicity and their potential for disease production (Finley *et al.,* 2008). According to the EPA, recent studies concluded that amphiboles are four times and 800 times as potent as chrysotile at inducing lung cancer and mesothelioma, respectively (Berman & Crump, 2003). Likewise, Darnton and Hodgson (2000) reported that the exposure specific risk of mesothelioma from the three most commonly used commercial asbestos – chrysotile, amosite, and crocidolite – is broadly estimated in the ratio 1:100:500, respectively.

Although there are dozens of published epidemiological studies quantifying the asbestos exposure-response relationship for lung cancer and mesothelioma (Finley *et* al., 2008), estimated no observed adverse effect levels (NOAEL) reported by some studies are highly inconsistent with ranges reported by other studies. For example, Fry *et al*. (1984) observed a significant increase in respiratory cancer when cohorts were exposed to <14 fibres per cubic centimetre per year (f/cm^3-yr; equivalent to f/mL-yr). This effect level conflicts with other studies which range from >25 to 1600-3200 f/cm^3-yr (Finley *et al*., 2008). Similarly, Brown *et al*. (1994) observed significant increases in lung cancer risk in cohorts who were exposed to a range of 2.7 to 6.8 f/cm^3-yr (NOAEL: 1.4–2.7 f/cm^3-yr); this is also inconsistent with lung cancer NOAELS reported in other studies. Surprisingly, one author, mentioned by Finley *et al*. (2008), reported that no increased risks occur at estimated cumulative asbestos exposures of 1600–3200 f/cm^3-yr. This is well beyond the threshold values stated earlier. Finley *et al*. (2008) states that this variability may be due to a number of factors, including, and not limited to: (i) air sampling techniques, (ii) use of appropriate controls, (iii) cohort sizes, and (iv) biases. Overall, the accepted derived NOAELs for lung cancer and mesothelioma fall in the range of 25–1000 f/cm^3-yr and 15–500 f/cm^3-yr, respectively (Finley *et al*., 2008). Fortunately, this is well below the nonoccupational asbestos exposure levels listed in Table 2. Figure 6 shows a comparison of upper bound cumulative chyrsotile exposures from various studies analyzed by Finley *et al*. (2008).

Currently, the permissible exposure limit for asbestos is 0.1 f/cm^3 of air averaged over an eight-hour work shift (29 CFR 1910.1001, OSHA) or 1.0 f/cm3 of air averaged over a sampling period of 30 minutes (29 CFR 1910.1001, OSHA). Although there is no safe level of exposure to any carcinogen such as asbestos, this standard would reduce worker asbestos-related illnesses to less than four cases per 1000 workers, half of the current illness rate. Moreover, the EPA (2009) specifies that water containing greater than seven MFL could increase one's risk of developing benign intestinal polyps. Thus, depending on the accuracy of the method and reliability of the information supplied by Edmen & Erdal (1990) on chrysotile levels observed in Québec's water quality (Table 2), drinking water in Québec could potentially be fatal.

As mentioned earlier, risk can also be evaluated using in vitro studies. In a study conducted by Layard *et al*. (1981), fibres of various sizes were implanted in the pleurae of rats for periods of more than one year. It was discovered that the probability for the development of pleural mesotheliomas was highest for fibres with a diameter of ≤0.25 μm and lengths ≥8 μm. Not surprisingly, these dimensions correlate with the average dimension of a single chrysotile fibril. Table 4 shows the correlation coefficients for the logit of tumour probability for the development of pleural mesotheliomas in rats. This discovery also correlates with Pott's (1978) hypothesis on the carcinogenic potency of a fibre with respect to size (Figure 4).

Although much scientific progress has been made in understanding the mechanisms involved in asbestos-induced diseases, the EPA (2003) notes that at least two critical data gaps remain:

- No one has yet been able to *track* a specific lesion induced by asbestos in a specific cell through to the development of a specific tumor. Studies have demonstrated that tumors of the type that result from asbestos exposure exhibit patterns of DNA alteration (or other kinds of cellular damage) that are sometime (but not always) consistent with the earlier cellular changes associated with asbestos exposure. There are also studies that show that exposure to asbestos can lead ultimately to development of tumors. However, these types of studies have yet to be linked.

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- The specific target cells that serve as precursors to tumors in various target tissues are not known with certainty.

Since researchers tend to report a broad range of tissue and cellular affects to denote the toxicity of asbestos-fibre exposure (EPA, 2003), there is no *consistent* endpoint to be used as a marker to track for asbestos-induced carcinogenesis. Consequently, this is a limitation on the hazard side of the risk assessment evaluation of asbestos (Kava, 2007).

Since asbestos abatement (removal) is a multibillion dollar industry and one in which many people have invested interest in (Gunter, 1994), innovative risk-based decision making must be based on factual matter and not one that is subject to ignorance, fear, and irrationality. Many developed countries, including Canada, have stopped using asbestos because of its detrimental effects to human health. However, is the removal of asbestos from existing building necessary? The two main issues involving this debate are the health risks associated with low-level exposure and the financial cost of removal (Gunter, 1994). To address the first issue, the NOAEL established by Finley *et al*. (2008) clearly indicates that what people are exposed to, on average, in buildings or schools (Table 2) is far lower than the threshold. Secondly, despite evidence showing it is unnecessary, the EPA estimates costs more than $53 billion over the next 30 years for the abatement of public and commercial buildings (Gunter 1994). It is now up to the stakeholders (tax-payers) to decide whether this is necessary or just a waste of time.

Risk Communication

Risk information is often communicated to the public through the workplace, the media, and personal experience. For the mass media, news about risks can easily gain one's attention, even when the risk news story is incomplete. Illnesses associated with asbestos exposure are often communicated in daytime television commercials. Mesothelioma lawyers advertise their service by discussing the risks associated with occupational asbestos-exposure, symptoms, and the possibility of being entitled to monetary damages. Interestingly, lawyers rarely offer their service to asbestos-related lung cancer patients, since lung cancer is often associated with smoking history and to prove a case would be time-consuming and confounded.

Generally, the public's perception of asbestos are derived from popular media stories. For instance, in the *Baltimore Sun*, a medical physician reported that 'whether [asbestos] is pulled out of a mountain, scraped off a steam pipe or shed from a brake shoe, asbestos causes cancer' (Schneider, 2006). Although the points made are true, to the reader, a statement like this would instantly cause alarm, even though it fails to discuss any of the factors that influence the potency of asbestos fibres. As a result, stakeholders will identify specific concerns about asbestos without ever having analyzed the accuracy of the reported data. In fact, many non-scientifically literate individuals on the internet still hold the view that 'one fibre of asbestos can kill,' which holds no scientific validation.

Furthermore, since most asbestos-related diseases are correlated to elevated concentrations on the job, asbestos-related health risks are often communicated to stakeholders prior to first starting a job, by taking part in mandatory training sessions to help reduce exposure potential. Stakeholders working in factories that have adopted the Occupational Safety and Health Administration (OSHA) standards, and whose duties require them to perform maintenance or custodial work must learn and follow general precautions like those listed in Table 5. In the 1970's, the mean concentration of fibres in the mining and milling industries in Québec, Canada, often exceeded 20 fibres/mL. Owing to the success of risk communication and the introduction of monitoring methods, average fibre concentrations are now less than 1.0 fibre/mL and continue to decline substantially (IPCS, 1998). Thus, asbestos risk is well communicated and, to some degree, successful in making stakeholders aware of inherent risks.

Conclusion

Although the word *asbestos* means different things to different people, as suggested at the beginning of this document, one definition that all parties can agree on is the established fact that asbestos exposure has the potential to cause severe adverse human health effects over prolonged periods of exposure. Asbestos exposure assessments clearly indicate that fibres are omnipresent, whether inside a public building or a residential area, and those who are at greater risk of developing asbestos-related illnesses are people who are occupationally exposed, are family members of the occupationally exposed, or are residents that live near factories that utilize asbestos. Toxicity assessments also reveal that prolonged exposure to asbestos fibres causes irreversible scarring in lung tissue, which ultimately leads to life threatening diseases. However, since there is a lack of consistent information provided in studies that indicate fibre type, size, duration of exposure, amount of exposure, air sampling techniques, and appropriate controls, a risk characterization challenge

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still remains open to debate. These factors collectively limit the stakeholder's ability to evaluate whether asbestos exposure poses a major health risk in their lives. Although there are significant gaps in the knowledge, the end result of asbestos-related illnesses is ultimately death; whether there is a definitive safe level or not, preventative actions should not be taken lightly by stakeholders. Safety precautions should continue to be promoted, practiced, and communicated to help reduce any possibility of asbestos-induced illnesses.

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Table 1. The six common asbestos minerals.

Asbestos Mineral	Property			
	Mineral	*Chemical Formula*	*Colour*	*Melting Point/Decomposition Temperature (°C)*
Amphibole				
Amosite	Cummingtonite, grunerite	$(Mg,Fe)_7Si_8O_{22}(OH)_2$	Light gray to pale brown	600-900
Crocidolite	Riebecktite	$Na_2Fe_3Fe_2^{+3}Si_8O_{22}(OH,F)_2$	Blue	800
Anthophyllite	Anthophyllite	$(Mg,Fe)_7Si_8O_{22}(OH,F)_2$	White to Grey, pale brown	950
Actinolite	Actinolite	$Ca_2(Mg,Fe)_5Si_8O_{22}(OH)_2$	Pale to dark green	1400
Tremolite	Tremolite	$Ca_2Mg_5Si_8O_{22}(OH,F)_2$	White to Grey	1040
Serpentine				
Chrysotile	Antigorite, lizardite	$Mg_6(OH)_8Si_4O_{10}$	White to pale green yellow, pink	800-850

* Adapted from Erdal & Esmen (1990) & ATSDR (1995).

Table 2. Selected asbestos exposure values.

Type	Location[a]	Level[‡]	Method*
Nonoccupational			
Chrysotile	Drinking Water (Québec)	1.1-1300 million fibres/L	TEM
Amosite	Downwind of Factory	500–2000 fibres/mL	TEM
Asbestos	Recreational Area	0.3–5.3 fibres/mL	TEM
Mixed	Offices (22)	0.022 fibres/mL	TEM
Chrysotile	School (71)	0.0083 fibres/mL	SEM
Mixed	Buildings (43)	<0.001–0.04 fibres/mL	TEM
Asbestos	Pacific Ocean[†]	0.002 fibres/mL	TEM
Type	**Process/Job**	**Level**	**Method**
Occupational			
Chrysotile/Amosite	Insulation	28–1015 fibres/mL	MFPCOM
Chrysotile	Drywall Installation	4–8 fibres/mL	SEM

* Method Descriptors: TEM, Transmission Electron Microscope; SEM: Scanning Electron Microscope; MFPCOM: membrane filter and phase contrast optical microscopy.
[†] Approximately 13 miles out on the Pacific Ocean on the California Coast.
[‡] Some original values were converted to fibres/mL for simplicity's sake.
[a] Number in brackets indicates the number of locations sampled.
[b] Modified from Erdal & Esmen (1990).

Table 3. A comparison of asbestos-induced disease parameters between rodents and humans.

Parameter	Endpoint	Rodent	Human
Latency Period	Mesothelioma	13-26 months	30-40 years
	Lung Cancer	12+ months	20 years
Cytotoxicity	Viability of alveolar macrophages	~50 %	~12 %
Inflammation/Fibrosis	Production of tumour necrosis factor α	4505 pg/mL	724 pg/mL
Cell Transformation	Clastogenesis (breaking of chromosomes)	Sensitive	More Sensitive

* Adapted from Maxim & McConnell (2001).

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Table 4. Correlation coefficients of logit of tumour probability with common logarithm of number of particles per microgram in different dimensional ranges.

Fibre diameter (μm)	Fibre length (μm)		
	≤ 4	> 4–8	> 8
> 4	–0.22	–0.28	–0.30
> 1.5–4	–0.45	–0.24	0.13
> 0.25–1.5	0.01	0.45	0.68
≤ 0.25	0.20	0.63	0.80

* Adapted from Layard *et al*. (1981).

Table 5. General Precautions for reducing exposure potential when working with or around known asbestos-containing materials.

- Never cut through pipe insulation.
- Never drill holes or hammer nails in ceilings or surfaced walls.
- Do not remove ceiling tiles or light fixtures from suspended ceiling grids.
- Never install curtains, drapes, or blinds in a way that damages any potential asbestos-containing material
- Try to avoid scraping floor tiles, walls, or ductwork when moving furniture.
- Never remove ventilation system filters or shake the filters to remove dust.
- Do not dust, sweep up debris or vacuum carpets in areas that may contain asbestos-contaminated waste.
- If you find any material that you suspect may contain asbestos, notify supervisor.

***** Adapted from EHSO (1996).

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Figures

Figure 1. Crocidolite rock (left) and chrysotile rock (right). Both samples show the fibre axes indicated by the red arrow are parallel to the direction of greatest strain.

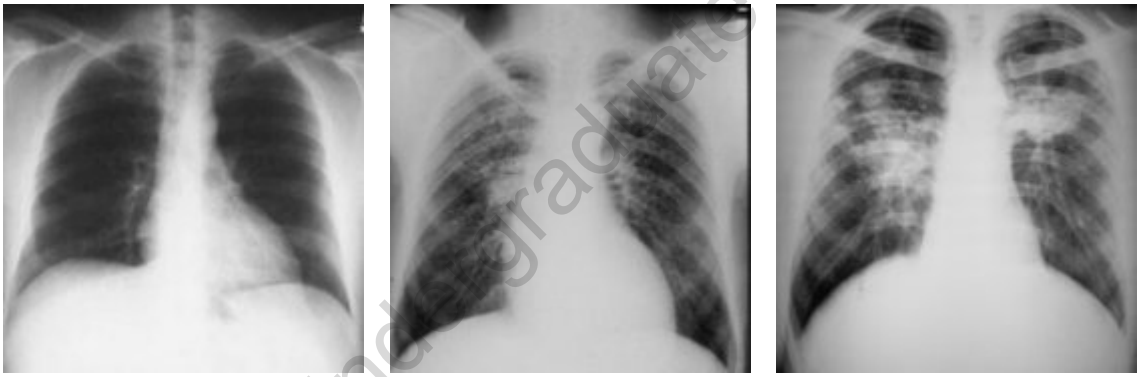

Figure 2. Examples of normal and abnormal radiographs according to the *International Classification of Radiographs of Pneumoconiosis. Left: Normal Radiograph; Middle:* Small parenchymal opacities in coal worker's pneumoconiosis; Right: Large parenchymal opacities (progressive massive fibrosis) in coal worker's pneumoconiosis.

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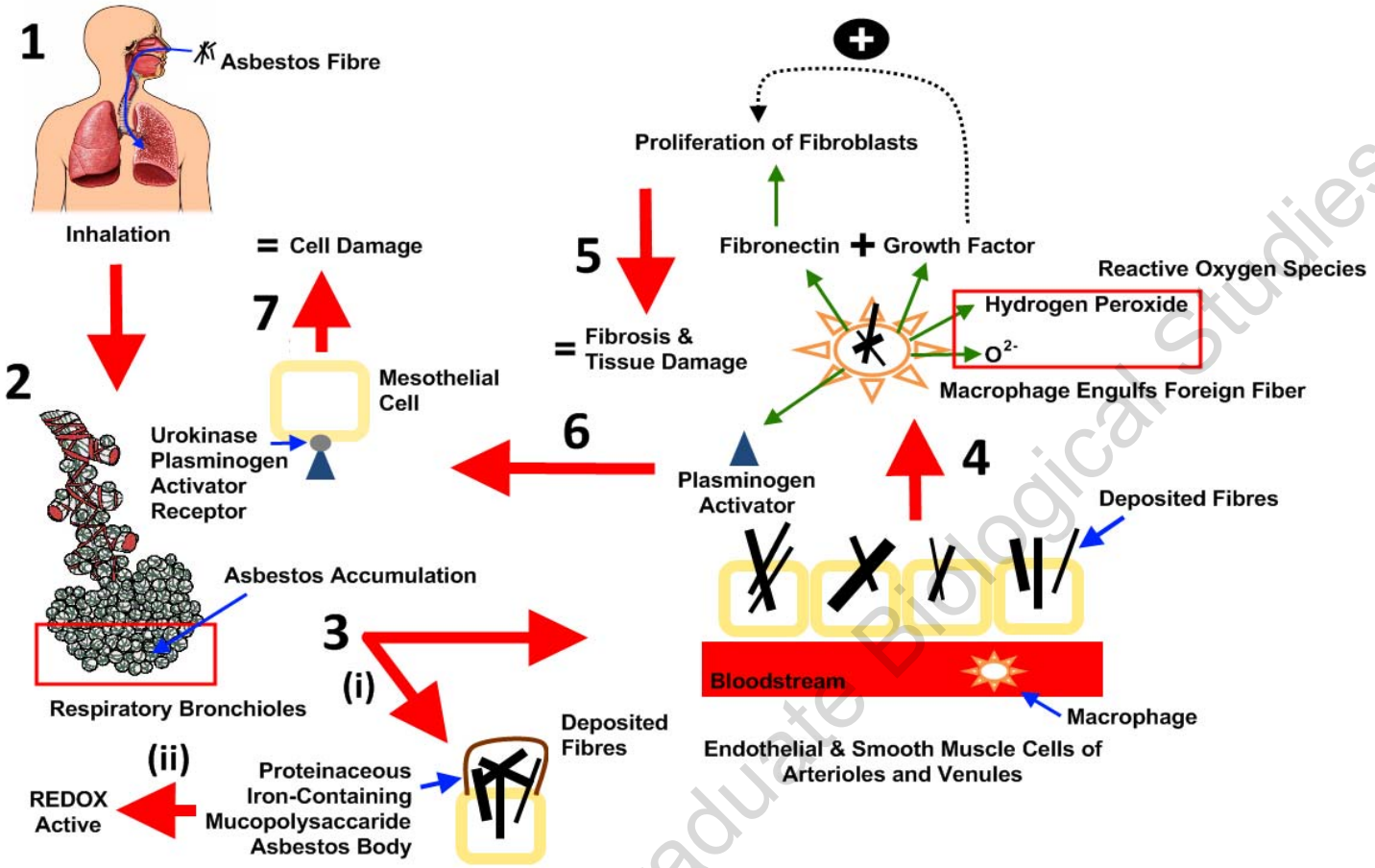

Figure 3. Mechanism outlining the major responses initiated by asbestos-fibre exposure in lung tissue. 1. Asbestos is inhaled into the tracheobronchial and the pulmonary or alveolar region. 2. Asbestos is absorbed and accumulates in respiratory bronchioles in the lower lobes. 3. The absorbed fibres are (i) encapsulated by proteinaceous iron-containing mucopolysaccaride, forming asbestos bodies and (ii) are REDOX active. 4. Asbestos fibres deposited in alveolar ducts induces alveolar macrophage to attack, producing fibronectin and growth factor which act synergistically to produce fibroblast. Macrophages also release reactive oxygen species and plasminogen activators. 5. This cause's fibrosis and tissue damage. 6. Plasminogen activators bind to urokinase-type plasminogen activator receptors at the surface of human mesothelial cells. 7. This induces direct cell damage to lung tissue.

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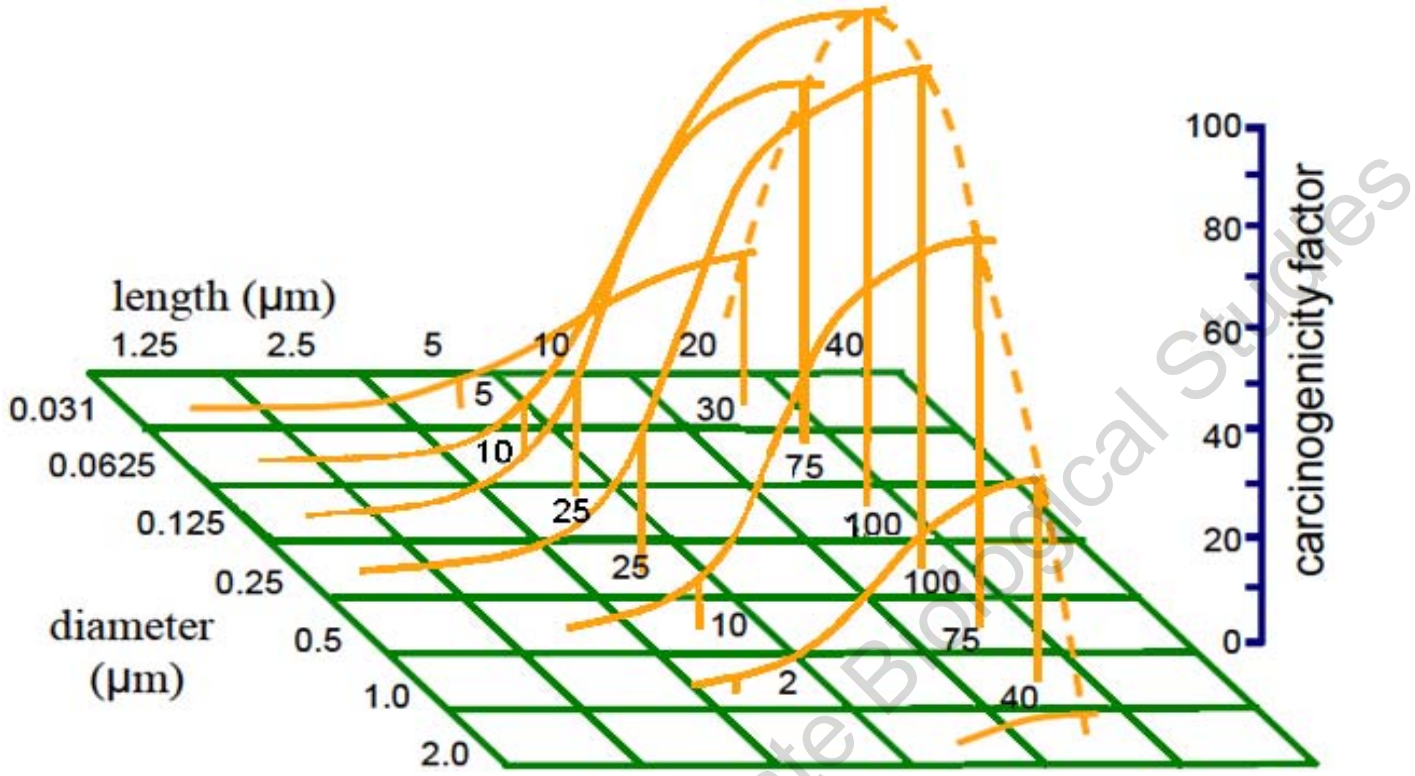

Figure 4. Hypothesis on the carcinogenic potency of a fibre as a function of its size with some with some data on the carcinogenicity factors. Modified from Pott (1978).

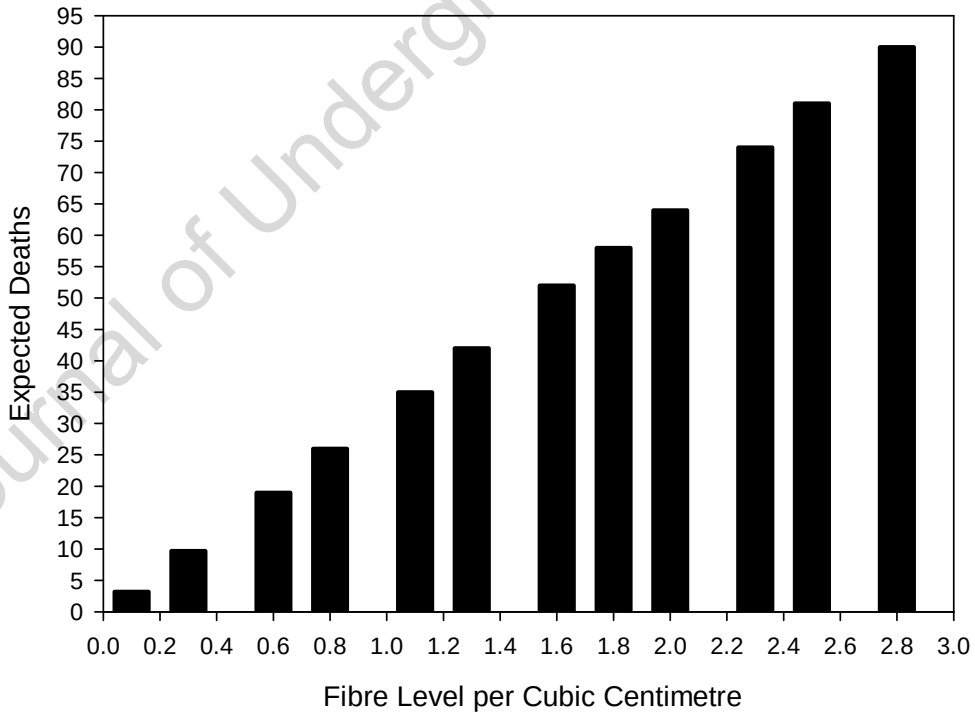

Figure 5. The number of expected deaths per 1000 workers based on airborne asbestos fibre exposure levels.

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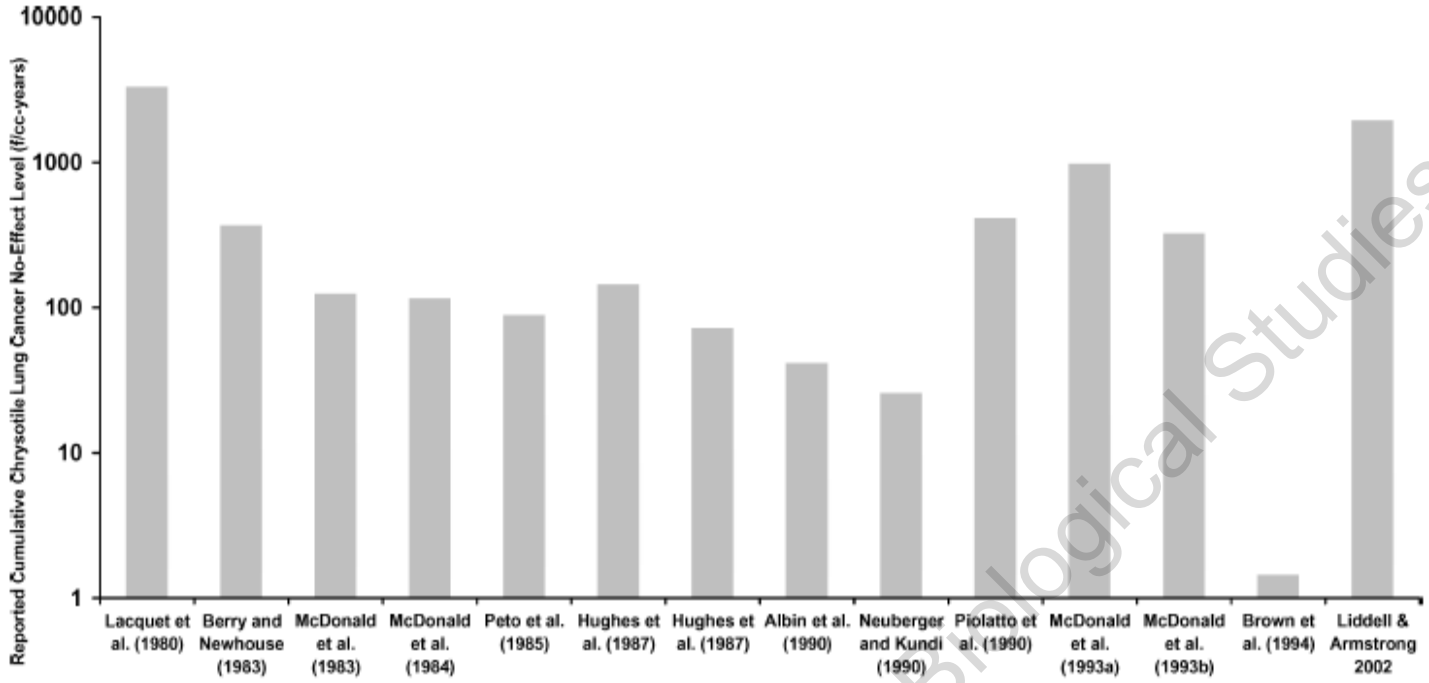

Figure 6. Comparison of upper bound cumulative chrysotile NOAELs reported in various occupational studies. Adapted from Finley *et al*. (2008).

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