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Risk assessment for asbestos-related cancer from the 9/11 attack on the World Trade Center


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Summary

Background The 9/11 terrorist attack on New York City’s World Trade Center (WTC) released an enormous amount of asbestos-containing dust into the outdoor air. The airborne concentrations of asbestos in Lower Manhattan were above background on and post-9/11 and inhalation of asbestos containing dust can increase the risk of developing some types of cancer.

Methods Analysis of settled dust and air sampling were used to estimate the extent to which the concentration of airborne asbestos was increased, how long it remained so and the type(s) of asbestos found.

Findings Chrysotile was the only asbestos mineral in the settled dust, at a concentration of less than 0.01% by volume; outdoor air sampling and modeling estimated the cumulative asbestos exposure to be equivalent to inhaling 0.064 fibers of asbestos per mL for a year.

Interpretation We conservatively assume the risk of developing an asbestos-related cancer increases linearly with exposure. For the residential population of Lower Manhattan the asbestos exposure associated with 9/11 might cause 1-2 asbestos-related cancer deaths over the lifetime of the population.
Introduction
In the aftermath of the September 11th atrocity, which destroyed New York City’s World Trade Center (WTC), questions have been raised concerning the risk of asbestos-related cancer from inhaling the dust. The initial dust cloud caused an enormously high concentration of airborne particulates which was brief and unforgettable. Twenty-four hours later the airborne concentration of dust was markedly lower but it remained uncertain the extent to which asbestos exposures would be above background during the 10 months required to remove the 1.5 million tons of debris. This report will address this uncertainty by asking the following questions: What were the asbestos fiber type(s) and concentration(s) in the air? When did the outside airborne asbestos levels post-9/11 return to the historical background levels for asbestos in NYC and elsewhere? What are the asbestos-related cancer risks likely to be as a consequence of these asbestos exposures?
Asbestos health hazards due to the events of 9/11 have been the focus of much media attention. The airborne asbestos monitoring undertaken by the US Environmental Protection Agency (EPA) was not based on health benchmarks. Little, if any, attention has been given to undertaking the type of air sampling necessary to perform a modern asbestos-related cancer risk assessment for 9/11.

Sources of the Cloud Dust
No masonry was used in the steel construction of the WTC. Therefore, the concrete floors (40,000-ft² per floor), fireproofing (5,000 tons), insulation and interior dry walls were the main sources of the resulting dust. Two photographs taken seven miles away over the first eight minutes reveals how quickly the air pressure generated by the collapsing tower raised a dust cloud, reaching such a height that no skyscraper in the vicinity of the WTC (several over 800-ft
in height) was visible (figure 1-2). Five hours later the dust had cleared sufficiently for the skyline to be partially visible, missing the two tallest and largest buildings in NYC (figure 3). At the street level the dust moved like a wall of volcanic ash (figure 4). There was obviously mixing which indicates the settled dust collected, for our study, would be representative of the particulate matter in the dust cloud.

The following day, the airborne dust concentration was markedly lower but remained elevated above background. Removing the 1.5 million tons of debris would require 20,000-30,000 truckloads and 10 months to complete. The movement of heavy equipment and other vehicles could promote re-entrainment of the asbestos containing settled dust; even allowing for efforts to suppress it by keeping the streets wet and the use of trucks capable of vacuuming (figure 5). If exposures remained elevated for an extended period of time, an increase in the risk of asbestos-related cancer would be expected.

**Methods**

Five settled dust samples were collected six days post-9/11 and one additional sample southeast of the WTC a week later (figure 6, table 1) and each was analyzed for the presence of asbestos minerals using powder X-ray diffraction (XRD), polarized light microscopy (PLM) and analytical transmission electron microscopy (ATEM). During the month of October, high volume outdoor air samples were collected at a single site in Lower Manhattan and prepared by direct-transfer for analysis by ATEM (figure 6). Historical air samples collected at three different sites in NYC and in the chrysotile asbestos mining town of Asbest City, Russian Federation were used respectively as low and high background controls.
Role of funding source

The State of New York provided an equipment grant but had no role in the study or the decision to submit this report for publication. Two of the authors (RP Nolan and CW Axten) are directors of the public foundation sponsoring the study.

Results

Settled Dust

XRD patterns of the settled dust indicated the presence of three crystalline phases: gypsum, calcite and quartz which are consistent with the known composition of the construction materials.\textsuperscript{2,3} In addition, each diffraction pattern was examined for the most intense peaks of the asbestos minerals. None were found indicating if asbestos is present it is less than the usual XRD detection limit of 1\% by mass. No asbestos minerals were visible by PLM in any of the settled dust samples using a procedure where asbestos would be ready apparent at 0.1\% by volume. ATEM examination found no amphibole asbestos of any type but traces of chrysotile asbestos were present in all six settled dust samples. The amount of sample examined decreases as you go from XRD to PLM to ATEM, using all three methods increases confidence in the estimate of the percentage of asbestos present. We estimate the concentration of chrysotile asbestos in the settled dust to be less than 0.01\% by volume. The composition of the settled dust is a guide to understanding the airborne asbestos exposure which is the basis of the risk assessment. All of the settled dust samples were of similar composition and the three crystalline phases (quartz, gypsum and calcite) identified by XRD and PLM analysis were also found by ATEM (table 1). Each contained trace amounts of chrysotile asbestos consistent with reports describing the uses of asbestos in the WTC.\textsuperscript{7}
Airborne concentration of asbestos post-9/11

Airborne particulates were collected on six membrane filters over a three-week period in October to determine the type and concentration of asbestos present. All the post-9/11 samples were collected at a single site, during the day and at night because the WTC debris removal program performed different tasks at night and movement of airborne particles are affected by thermal changes due to sunlight (figure 8, table 2). Samples were collected outside to determine if measurable increases in airborne asbestos concentration could be associated with the residual dust from the massive dust cloud containing traces of chrysotile asbestos and the ongoing debris removal.

The sampling strategy called for examining, at 20,000X magnification by ATEM, all of the particulates in an 11,244 to 14,293mL subpopulation of the total volume in each air sample. ATEM is the most sensitive method for the detection of airborne asbestos; the direct-transfer preparation of the air filter causes minimal changes in size distribution and any asbestos fiber present will be visible under these conditions. By sampling higher volumes of air and examining a larger area of the filter, the sensitivity was ~10-fold greater than what is normally used to monitor airborne asbestos for the purpose of risk assessment in the non-occupational environment.\(^5\) Not a single asbestos fiber was found in the 73,475mL of the outside air examined (table 2). For any air sample with a measurement of 0, the 95 percent upper confidence limit calculated from a Poisson distribution is 3 fibers. Therefore the upper limit for the airborne concentration based on the pooling of samples is 3 fibers in 73,475mL which equals 0.0004 f/mL. This concentration of asbestos in the outdoor air in Lower Manhattan 26 days post-9/11 was at the low end of the worldwide background level reported by the World Health Organization (figure 7).\(^8\) For the subsequent calculation we therefore assume that the effects of 9/11 on airborne asbestos concentrations were over 26 days later.
Estimation of cumulative asbestos exposure associated with 9/11

A modern risk assessment for asbestos-related cancer uses knowledge of the type of asbestos and the cumulative exposure, which represents the intensity and duration of exposure usually given in fibers per milliliter multiplied by years (f/mL x years). It is then compared to historical occupational exposures associated with increases in asbestos-related cancer, based on an 8-hour work day and 250 days per year. Outdoor air samples in NYC pre-9/11 were consistently less than 0.0008 f/mL for all asbestos fiber types having lengths = 0.5 µm (figure 8). Since the settled dust gave no indication of any amphibole asbestos being present we only consider chrysotile asbestos in our discussion of the upper limit.

To our knowledge no air sampling data have been reported for the initial dust cloud on 9/11 and it is doubtful if such a particulate dense aerosol could have been meaningfully analyzed for what was respirable and at what concentration. Considering the trace amount of chrysotile asbestos in the settled dust, we estimate the maximum concentration of airborne asbestos at 50 f/mL with a length = 5 µm. It is problematic to use the analysis of settled dust to determine the extent to which these asbestos fibers, when airborne, were respirable and at what concentration. The high exposure assumed is similar to uncontrolled historical exposures in chrysotile asbestos mines and mills where the ore contains a minimum of 2-4% asbestos and likely to be the worst case. After approximately 5 hours, we assume the airborne concentration of chrysotile asbestos to have decreased by 50-fold to no more than 1 f/mL = 5 µm in length. Then we assume it decreased linearly to background when the first air sample was collected 26 days later on October 8th (figure 1 and 8). This assumption is conservative since the decrease is more likely to have been exponential.

Based on the analysis of settled dust and air sampling, the exposure estimate for the residential population need only consider exposure to chrysotile asbestos. The assumed cumulative
exposure from 9/11 until the first air sample was collected on October 8th is shown graphically in figure 8 and calculated below:

The two-time periods of cumulative exposure can be added

\[0.028 \text{ f/mL} \times \text{years (initial 5 hrs after first tower collapses on 9/11)}\]
\[+ 0.036 \text{ f/mL} \times \text{years (next 26.4 days)} = 0.064 \text{ f/mL} \times \text{years}\]

Anyone not exposed to the initial 5-hour dust cloud on 9/11, had about half this cumulative chrysotile asbestos exposure.

**Risk assessment for asbestos-related cancer**

The number of asbestos-related mesotheliomas \((O_M)\) depends on the type of asbestos one is exposed to, the cumulative exposure and the age at which exposure first occurs\(^{10}\) and can be calculated by:

\[
O_M = \frac{R_M \times E_{CA} \times T_{pop}}{100}
\]

Where

- \(R_M\) - Risk of mesothelioma as a percentage of the total expected mortality. The \(R_M\) used, 0.001, is obtained from Reference 10 (their table 1) (adjusted to 30 years of age at first exposure) and over estimates the chrysotile asbestos risk as some exposure to amphibole asbestos occurred in the cohorts used to determine the value. This is derived from occupational exposure, assumed to be 8-hour work day and 250 days per year.
• $E_{CA}$ - The cumulative chrysotile asbestos environmental exposure (assumed to be continuous) 0.064 f/mL x years is converted to be an equivalent occupational exposure of 0.28 f/mL x years (figure 8).

• $T_{pop}$ - Total exposed population for Lower Manhattan is all 57,511 residents estimated from United States Census 2000 (see figure 6 for area included).

Solving for $O_M$:

$$O_M = 0.16 \text{ mesothelioma cases due to 9/11 exposure to chrysotile asbestos.}$$

For a given cumulative asbestos exposure, the risk of developing lung cancer will increase as a percentage of the existing lung cancer risk in the population. We will assume that on average 8% of cigarette smokers develop lung cancer while among those that choose not to smoke only 0.8% will develop lung cancer$^{11}$ and that the risk of lung cancer increases linearly with cumulative asbestos exposure following the relationship:

$$Obs_L = Exp_L + \frac{R_L \times E_{CA} \times Exp_L}{100}$$

We wish to calculate the increase in the observed number of lung cancers ($Obs_L$) due to exposure to chrysotile asbestos.

• $Exp_L$ - Expected percentage of lung cancer deaths assumed to be 8% among smokers.
• **R\textsubscript{L}** - Risk of lung cancer expressed as a percentage of lung cancer deaths per f/mL x years of asbestos exposure. The **R\textsubscript{L}** used is 0.062 obtained from reference 10 (their table 2) and is specific for chrysotile asbestos.

• **E\textsubscript{CA}** - The cumulative chrysotile asbestos environmental exposure (assumed to be continuous) 0.064 f/mL x years is converted to be an equivalent occupational exposure of 0.28 f/mL x years (figure 8).

\[
\text{Obs}\textsubscript{L} = 8\% + 0.0014\% = 8.0014\%
\]

If the entire population of Lower Manhattan, 57,511, smoked cigarettes, the lung cancer mortality would be expected to be 8\% (or 4,600 lung cancer cases) and increases to 8.0014\% (or 2 additional case as a result of the events of 9/11). If the population chooses not to smoke the incidence of lung cancer would be 10-fold lower and 9/11 would be associated with two tenths of a lung cancer case. Approximately 80-85\% of the lung cancer risk is attributed to smoking, making it the most significant risk factor for developing lung cancer.\textsuperscript{12} About 25\% of all persons over 18 years of age smoke and approximately 86\% of the residents in Lower Manhattan are over 18 years of age so the actual increased lung cancer incidence would be less than one case.\textsuperscript{13} At this very low cumulative asbestos exposure, the synergy with smoking is expressed solely as a difference between smokers and non-smokers in the assumed linear risk coefficient (figure 10).
Discussion

The attacks on NYC’s WTC and the collapse of both towers created a pressure wave, which dispersed an enormous amount of dust containing asbestos into the outside air of Lower Manhattan (figure 1-5). The only type of asbestos present in the settled dust was chrysotile at a concentration less than 0.01%. Crucial assumptions were made about estimating the airborne concentration of asbestos: that the dust cloud immediately after the collapse of the first tower was equivalent to highest concentration reported for uncontrolled chrysotile use and that after five hours it fell to below the level of the 1976 occupational exposure standard for asbestos in the US, before it fell to the measured background (figure 7).

This risk assessment also makes two fundamental assumptions about the carcinogenicity of chrysotile asbestos. Firstly, it is assumed, following Hodgson and Darnton\(^{10}\), that chrysotile is a less potent inducer of mesothelioma than amosite or crocidolite. Secondly, it is assumed that at low doses there is a linear dose-response. Our approach is to interpolate linearly the increased risk from high cumulative exposures for which there is a known risk for the asbestos-related cancer, to very low exposure. The dose-response may be sublinear (leading to a smaller risk at low doses), and this is an additional reason why our estimates are only an upper limit. For a cumulative asbestos exposure of 0.064 f/mL x years, the 57,511 residents of Lower Manhattan may experience at most 1 mesothelioma case (estimated expected number equals 0.16) more than would otherwise be expected (probability of more than one equals 0.01). The background for mesothelioma has been conservatively estimated to be 3.6 cases per 10,000.\(^{14}\) Therefore approximately 21 mesotheliomas would be expected in this population with perhaps one additional 9/11 related case.
If the exposure was to crocidolite asbestos, the mesothelioma risk would be almost 500-fold higher and 80 mesothelioma cases would be expected (figure 9). This justifies our claim that determining asbestos fiber type(s) is important. Environmental exposure to airborne crocidolite and tremolite asbestos have been shown to increase the risk of mesothelioma where mine tailings or local outcrops have been used in the construction of unpaved roads or building materials.\textsuperscript{11}

For chrysotile asbestos the evidence is different, in chrysotile producing areas where the outdoor airborne concentration of asbestos is 0.04f/mL environmental mesotheliomas rarely if ever occur.\textsuperscript{15}

If the entire population smoked cigarettes approximately two asbestos-related lung cancer cases would be expected. If no one smoked, the risk of lung cancer would be 10-fold lower (figure 10).

\textit{Contributors}

R P Nolan and M Ross had the original idea for the project, wrote the study protocol and analyzed the samples along with G L Nord. All authors discussed the protocol and contributed to the design. C W Axten modeled the exposure data. J P Osleeb contributed the population data. S G Domnin undertook the Russian air sampling. R Wilson and B Price did the risk assessment. R P Nolan wrote the final version which was discussed, edited and revised by the all the authors.

\textit{Conflict of Interest Statement}

None declared.
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