Risk Assessment for Asbestos-Related Cancer From the 9/11 Attack on the World Trade Center

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Objective: *We sought to estimate the lifetime risk of asbestos-related cancer for residents of Lower Manhattan attributable to asbestos released into the air by the 9/11 attack on New York City's World Trade Center (WTC).* ***Methods:*** *Exposure was estimated from available data and reasoned projections based on these data. Cancer risk was assessed using an asbestos risk model that differentiates asbestos fiber-types and the US Environmental Protection Agency's model that does not differentiate fiber-types and combines mesothelioma and lung cancer risks.* ***Results:*** *The upper limit for the expected number of asbestos-related cancers is less than one case over the lifetime of the population for the risk model that is specific for fiber-types and 12 asbestos-related cancers with the US Environmental Protection Agency's model.* ***Conclusions:*** *The cancer risk associated with asbestos exposures for residents of Lower Manhattan resulting from the collapse of the WTC is negligible.* (J Occup Environ Med. *2005;47:817–825*)

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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 but unforgettable. Twenty-four hours later, the airborne concentration of dust was markedly lower, but it remained uncertain as to the extent to which asbestos exposures would be above background during the 10 months required to remove the 1.5 million tons of debris resulting from the collapse of the buildings. This article will estimate the risk of developing lung cancer and mesothelioma from the asbestos exposure, including its 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? What is the asbestos related cancer incidence likely to be as a consequence of these asbestos exposures?

The airborne asbestos monitoring undertaken by the US Environmental Protection Agency (EPA) after the attack was not based on health benchmarks or on acquiring data for a risk assessment.[1] 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

The exterior of WTC was built of steel, with no masonry used. There-

fore, the concrete floors (40,000-ft^2 per floor), fireproofing (5000 tons), insulation, and interior dry walls were the main sources of the resulting dust.[2,3] Two photographs taken seven miles away during the first 8 minutes reveals how quickly the air pressure generated by the collapsing tower raised a dust cloud. The cloud reached such a height that no skyscraper in the vicinity of the WTC (several over 800-ft in height) was visible At the street level, the dust moved like a wall of volcanic ash (Fig. 1A–C). Five hours later, the dust had cleared sufficiently for the New York City skyline to be partially visible, now missing its two tallest and largest buildings (Fig. 2).

There was obviously mixing occurring within the cloud that indicates the dust that settled during the 6 days after 9/11, the period for our study, would be representative of the particulate matter in the dust cloud. That the cloud we sampled during the first 6 days is indeed representative is one of our important assumptions. The day after the collapse of the WTC, the airborne dust concentration was markedly lower but remained elevated above background. The removal of the 1.5 million tons of debris required 20,000 to 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 (Fig. 3). If exposures had remained elevated for an extended period of time, contrary to our airborne asbestos analysis, an increase in the risk of asbestos-related cancer would be expected.

Materials and Methods

Six representative settled dust samples were collected at least 6 days after 9/11 (locations shown in Fig. 4) 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 site in Lower Manhattan and prepared by direct-transfer for analysis by ATEM.[4] Historical air samples collected in NYC and in the chrysotile asbestos mining town of Asbest City, Russian Federation, were used respectively as low and high background controls.[5,6]

Fig. 1. (A) One and a half minutes after the collapse of the South Tower the lighter color construction dust became more visible and quickly reached higher than the tallest building in Lower Manhattan. (B) Within 8 minutes the entire skyline disappeared in a cloud of dust. (C) The dust cloud moving in very sharp zones around the Woolworth Building on Broadway.

Fig. 2. Five hours after the collapse of the first tower the skyline was partially visible.

Fig. 3. Trucks on Chambers Street waiting to pick up debris from World Trade Center during the first week in October, 2001. Note that the streets surrounding the WTC were kept continuously wet to suppress the dust.

Results

Settled Dust

Powder diffraction patterns of the settled dust indicated that three major crystalline phases were present: gypsum, calcite, and quartz, which are consistent with the known composition of the WTC construction materials.[2,3] In addition, each diffraction pattern was examined for the most intense peaks of the asbestos minerals. None were found, indicating that if asbestos was present, it was only present to less than 1% by mass. No asbestos minerals were visible by PLM in any of the settled dust samples, further reducing the limit of asbestos concentrations to less than 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. We

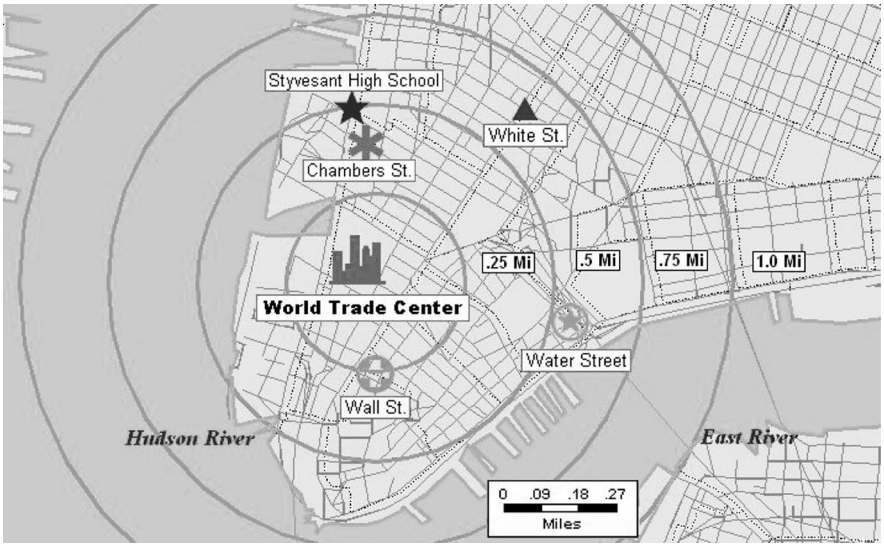

Fig. 4. The highlighted area of lower Manhattan has 57,511 residents according to the 2000 U.S. Census and was used in the risk assessment as the general population. Three settled dust samples were collected in the area of Stuyvesant High School on the Westside Highway (★, one from a motor vehicle on Chambers Street (*, one from an auto van on White Street (▲) and one southeast of WTC (⊕). The six ambient air samples were collected at Water Street (✪) near the Brooklyn Bridge.

estimate the concentration of chrysotile asbestos in the representative 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 major crystalline phases (quartz, gypsum, and calcite) identified by XRD and PLM analysis also were found by ATEM. Each contained trace amounts of chrysotile asbestos, consistent with reports describing the uses of asbestos in the WTC.[7]

Airborne Concentration of Asbestos After 9/11

Airborne particulates were collected on six membrane filters during a 3-week period in October to determine the type and concentration of asbestos present. All the samples taken after 9/11 were collected at a single site during the day and at night because the WTC debris removal program performed different tasks at night and the movement of airborne particles are affected by thermal change due to sunlight (Fig. 4). Samples were collected outside of buildings to determine whether 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.

All of the particulates in 11,244 to 14,293 mL of air were examined in the six samples at 20,000× magnification by ATEM. This procedure 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 than usual for such tests 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 and 25-fold more sensitive than the Asbestos Hazard Emergency Response Act (AHERA) protocol favored by US EPA.[1,5] Not a single asbestos fiber was found in the 73,475 mL of the outside air examined (Table 1). For the exposure calculations that follow, we use the upper 95% confidence limits shown in Table 1, which are upper bounds for the true airborne asbestos concentration based on our measurement. The concentration of asbestos in the outdoor air in Lower Manhattan 26 days post-9/11 was approximately 500-fold lower than the ambient air in a chrysotile mining community and at the low end of the worldwide background level reported by the World Health Organization (Fig. 5).[6,8]

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 × years). Outdoor air samples in NYC pre-9/11 were consistently less than 0.0008 f/mL for all asbestos fiber types having lengths ≥5 μm (Figs. 5 and 6).[5] Because the settled dust gave no indication of any amphibole asbestos being present, we only considered chrysotile asbestos in our discussion of the upper limit (similar results regarding the asbestos fiber type present have been reported by others).[9]

To our knowledge, no air sampling data have been reported for the initial dust cloud on 9/11, and it is doubtful whether such a particulate dense aerosol could have been meaningfully analyzed for the type and concentration of respirable asbestos. 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 the exposures measured historically in uncontrolled

TABLE 1
Results of ATEM Analysis of Six Outdoor Air Samples Collected on Water Street in October 2001

Date	Asbestos ≥5 μm	Asbestos <5 μm	Volume of Air Scanned (mL)	Sensitivity Fiber/mL	Total Airborne Chrysotile Asbestos Concentration 95% UCL* Fiber/mL
10/08/01	0	0	11,244	0.00009	0.00027
10/09/01	0	0	11,319	0.00009	0.00026
10/10/01	0	0	11,371	0.000088	0.00026
10/21/01	0	0	13,530	0.000074	0.00022
10/25/01	0	0	11,718	0.000085	0.00026
10/30/01	0	0	14,293	0.00007	0.00021
Pooled	0	0	73,475	0.00001	0.00004

Although no asbestos was found in any sample, we calculated using the Poisson distribution the upper 95th percentile, which is 3 fibers in each case. The upper 95th percentile of the pooled measurement or 0.00004 f/mL was used in the risk assessment to establish the airborne asbestos level had returned to background 27 days after 9/11.

*Upper confidence limit.

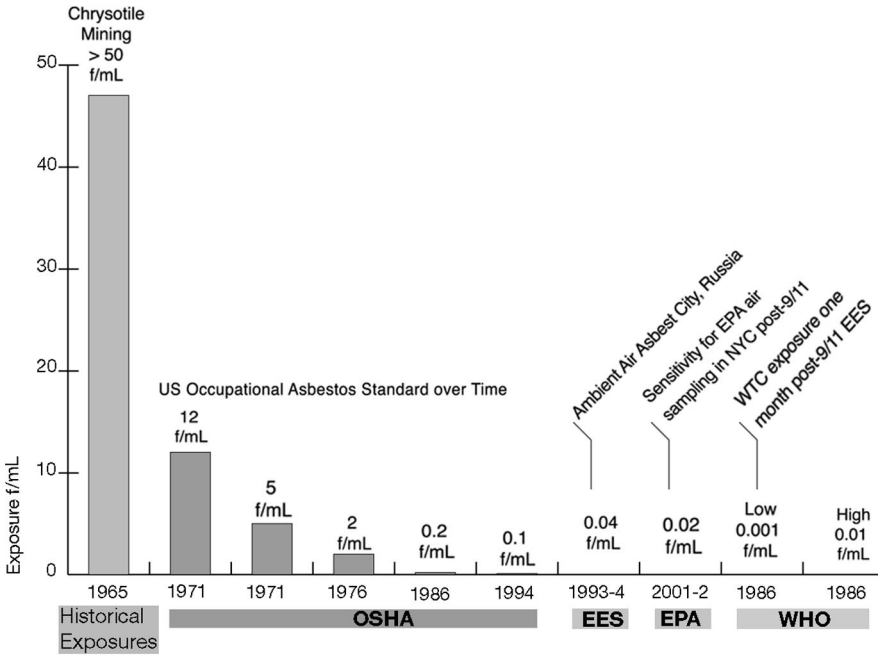

Fig. 5. Comparison of Asbestos Exposures from the collapse of WTC complex with historical, permissible and background asbestos exposures. Note the United States Environmental Protection Agency (EPA) does not determine the actual airborne concentration of asbestos but only reports the number of sturctures per unit area of the collection filter (EPA f/mL above is estimated). EPA does not define structure as any of the six regulated types of asbestos therefore asbestos fiber type is not known. Earth and Environmental Sciences (EES) found the historical airborne asbestos concentration in NYC to be indistinguishable from those 26 days post-9/11.

chrysotile asbestos mines and mills where the ore contains a minimum of 2% to 4% asbestos −100 times more than in the dust from the Twin Towers. This is therefore likely to be a pessimistic assumption and we assume it as an upper limit (Fig. 5).[10]

Photographs taken approximately 5 hours after the collapse of the first tower indicate that the suspended dust settled rapidly (Fig. 2). We assumed the airborne concentration of chrysotile asbestos to have decreased during that initial 5 hours by 50-fold to no more than 1 f/mL ≥5 μm in length. Then, we assumed that the concentration further decreased linearly to background by the time we collected our first air sample 26.4 days later on October 8th (Fig. 1, Fig. 6). Because the decrease was more likely to have been exponential, this linear assumption is conservative.

Air samples were collected by EPA starting on September 15th and continued through October 8th. The EPA collected 8870 air samples in Lower Manhattan after 9/11 for analysis by analytical transmission electron microscopy.[11] Twenty-two air samples (0.24%) exceeded the AHERA standard of 70 structures per square millimeter (S/mm^2), having a length greater than or equal to 0.5 μm. The samples that exceeded the AHERA standard we mainly collected at the perimeter of Ground Zero and the landfill on Staten Island. The AHERA standard is not a health benchmark but rather reflects the upper limit of possible asbestos contamination of the collection filter. Based on the area of the filter examined for fibers and the volume of air sampled required in the AHERA protocol, the 70 S/mm^2 corresponds to approximately 0.02 S/mL ≥0.5 μm in length. Not all structures are fibers and the number of S/mL will always be equal to or greater than the number of f/mL. Therefore when S/mm^2 are converted to f/mL, the exposure values are upper limits.

The first air samples collected after 9/11 (by the EPA on September 15th) correspond to 0.038 f/mL and 0.048 f/mL. An additional 10 air samples (range 0.021–0.164 f/mL, mean 0.04 f/mL ≥0.5 μm in length

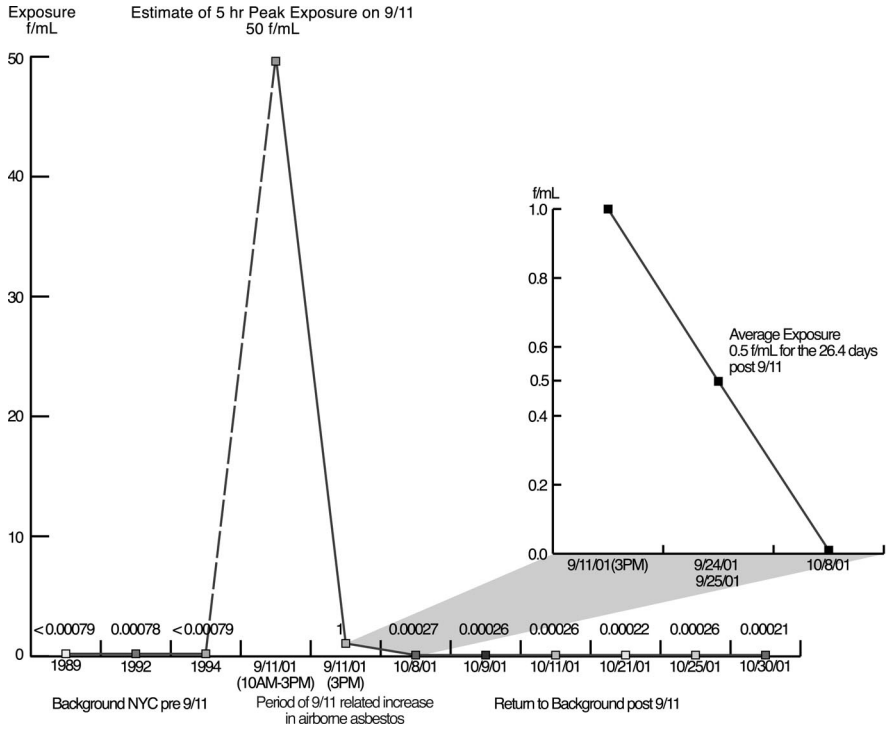

Fig. 6. Estimates of the chrysotile asbestos exposure to the general population from the dust released when the Twin Towers collapsed and while the airborne concentration of asbestos was elevated. The best estimate of the maximum cumulative chrysotile asbestos exposure to the general population of Lower Manhattan during the period post 9/11 prior to returning to background is 0.064 f/mL × years.

for all 12 air samples) were above 0.02 f/mL before collecting our first air sample on October 8th. Although these air samples are of limited use for risk assessment that requires that the number and type of airborne asbestos fiber ≥5 μm in length be determined, it is interesting to know that the numbers for total fibers present are below what we have assumed for the ≥5 μm in length fraction.

In our exposure estimate, we assume a mean exposure to 0.5 f/mL of chrysotile asbestos ≥5 μm in length for the 26.4-day period from the initial clearing of the heavy airborne dust after 5 hours until the background level is re-established on October 8th. The mean of the 12 highest concentrations reported by EPA during that time period, 0.04 f/mL ≥0.5 μm in length, is an order of magnitude lower than our assumed value and would be even lower if fiber length were considered. As with the initial 5-hour exposure period, we have assumed what is likely to be a worst-case estimate of exposure prior to establishing background on October 8th.

On the basis of the analysis of settled dust and air sampling, we estimated the 9/11-based incremental increase in the ambient asbestos exposure for a typical resident of Lower Manhattan. Our objective was to assess the incremental cancer risk associated with this exposure by applying established quantitative risk assessment models. We calculated two exposure indexes: cumulative lifetime exposure for use with separate risk models for lung cancer and mesothelioma, which was developed by Hodgson and Darnton,[12] and lifetime average daily exposure (LADE) for use with EPA's aggregate risk model for lung cancer plus mesothelioma.[13] The assumed exposure levels from 9/11 until our first air sample was collected on October 8th is shown graphically in Fig. 6. The cumulative exposure for this time period is calculated below:

Although the initial level of 50 f/mL fell to approximately 1 f/mL during the first 5 hours after the first tower collapsed on 9/11, our estimate is an upper bound on exposures by assuming 50 f/mL throughout the 5-hour interval:

$$50 \text{ f/mL } [5 \text{ hours}/(24 \text{ hours/d} \times 365 \text{ days/yr})] = 0.029 \text{ f/mL} - \text{years};$$

for the next 26.4 days, our estimate is as follows:

$$(1.0 \text{ f/mL} - 0.0004 \text{ f/mL})/2) \times (26.4 \text{ days}/365 \text{ days/yr}) = 0.036 \text{ f/mL} - \text{years}$$

The total cumulative exposure is the sum of the exposures for these two time-periods is as follows:

$$\text{Total Cumulative Chrysotile Asbestos Environmental Exposure} = 0.065 \text{ f/mL} \times \text{years}$$

Anyone not exposed to the initial 5-hour dust cloud on 9/11 had less than half the cumulative environmental chrysotile asbestos exposure given. Because no asbestos fibers were detected in any of the air samples, the upper 95% confidence limit for the combined samples, 0.00004 f/mL, was used as the background concentration of asbestos fibers.

The risk models we used were derived from occupational exposure data. Therefore, we must restate our continuous environmental exposure estimates as equivalent occupational exposures. Occupational exposures occur over the course of 250 days per year for 8 hours per day. Continuous environmental exposure occurs over 365 days per year 24 hours per day. Therefore, multiplying continuous exposure by the ratio (365 days/yr × 24 hours/d)/(250 working days /yr × 8 hours/d) = 4.38 produces equivalent occupational exposure. The equivalent occupational exposure associated with the events of 9/11 is 4.38-fold larger

than the environmental exposure, or 0.28 f/mL − years.

The Lifetime Average Daily Exposure (LADE), the exposure index used with EPA's aggregate cancer risk model for asbestos is a measure of exposure for 24 hours per day every day of the year. LADE, therefore, is the environmental exposure calculated above, 0.065f/mL − years, divided 70 years, the lifetime duration EPA uses for risk assessment. LADE for the 9/11 exposure is 0.0009 f/mL (= 0.065/70).

Risk Assessment for Asbestos-Related Cancer

The number of asbestos-related mesothelioma (O_M) depends on the type of asbestos one is exposed to, the cumulative exposure and the age at which exposure first occurs[12] and can be calculated by the following:

$$O_M = \frac{R_M \times E_{CA} \times T_{pop}}{100} \quad (1)$$

Where R_M is the risk of mesothelioma as a percentage of the total expected mortality. The R_M used, 0.001, is obtained from Table 1 of Hodgson and Darnton[12] (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 of R_M. This is derived from occupational exposure, assumed to be 8 hours/d for 250 days per year. E_{CA} is the cumulative chrysotile asbestos environmental exposure (assumed to be continuous) 0.065f/mL × years is multiplied by 4.38 to the equivalent occupational exposure 0.28 f/mL × years (Fig. 6). T_{pop} is the adjusted total exposed population for Lower Manhattan. The total population is 57,514 residents estimated from United States Census 2000 (see Fig. 4 for area included). Multiplying the T_{pop} by 0.47 adjusts the age at first exposure to the average age of Lower Manhattan residents of 38.[12] T_{pop} used in the calculation is 57,514 × 0.47 = 27,302.

Solving for O_M:

O_M = 0.08 mesothelioma cases due to 9/11 exposure to chrysotile asbestos and the lifetime risk of mesothelioma is $O_M/O_{pop} = 1.39 \times 10^{-6}$.

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, 90% of the lung cancers are found in smokers, and 25% of the residents of Lower Manhattan smoke. The risk of lung cancer increases linearly with cumulative asbestos exposure following the relationship:

$$Obs_L = Exp_L + \frac{R_L \times E_{CA} \times Exp_L}{100} \quad (2)$$

We wish to calculate the increase in the observed number of lung cancers (Obs_L) caused by exposure to chrysotile asbestos. Exp_L is the expected background of lung cancer deaths, 1,278, among the 57,514 residents of Lower Manhattan. This background rate is determined by solving equations that reflect the relationship between the percentage of smokers who get lung cancer and the percentage of lung cancers that occur in smokers. Specifically, 0.9 × (no. lung cancers) = 0.08 × (no. smokers) = 0.08 × 0.25 × 57,514/0.9 = 1,278.

R_L is the risk of lung cancer expressed as a percentage of lung cancer deaths per f/mL × years of asbestos exposure. The R_L used is 0.062 obtained from Table 2 of Hodgson and Darnton[12] and is specific for chrysotile asbestos. E_{CA} is the cumulative chrysotile asbestos environmental exposure (assumed to be continuous) 0.065 f/mL × years is converted to the equivalent occupational exposure of 0.28 f/mL × years (Fig. 6). Using these values Obs_L = 0.22 and the relative risk of lung cancer associated with the events of 9/11 is $Obs_L/Exp_L = 1.7 \times 10^{-4}$.

The US EPA's aggregate asbestos cancer risk model does not differentiate asbestos fiber types. The risk for the sum of lung cancer and mesothelioma is calculated as 0.23 × LADE, where the increment to LADE (lifetime average daily exposure) for the events of 9/11 is 0.0009 f/mL. The risk of cancer equals 2.1×10^{-4}, which is equivalent to 12 excess cancers, for the population of Lower Manhattan.

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 (Figs. 1 and 2). Our analysis of representative settled dust samples by XRD, PLM, and ATEM indicates that of the six regulated asbestos fiber types, only chrysotile asbestos was present. The chrysotile asbestos concentration was less than 0.01% by volume. Although estimating the airborne concentration of asbestos on and shortly after 9/11 has limitations, it undoubtedly was above the background in the air for some period of time.[9,11] The potential for an increased incidence of asbestos-related cancer from 9/11-related exposure depends principally on two factors: asbestos fiber type(s) and the cumulative asbestos exposure. For mesothelioma age at first exposure is an additional important factor. For lung cancer the synergy between asbestos and cigarette smoking can be important, although only at higher cumulative asbestos exposures than those associated with 9/11 (Fig. 7).

This risk assessment makes two fundamental assumptions about the carcinogenicity of chrysotile asbestos. First, it is assumed, following Hodgson and Darnton,[12] that chrysotile is a less potent inducer of mesothelioma and lung cancer than amosite or crocidolite. Second, it is assumed that at low doses there is a linear dose–response. Our approach was 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. Epide-

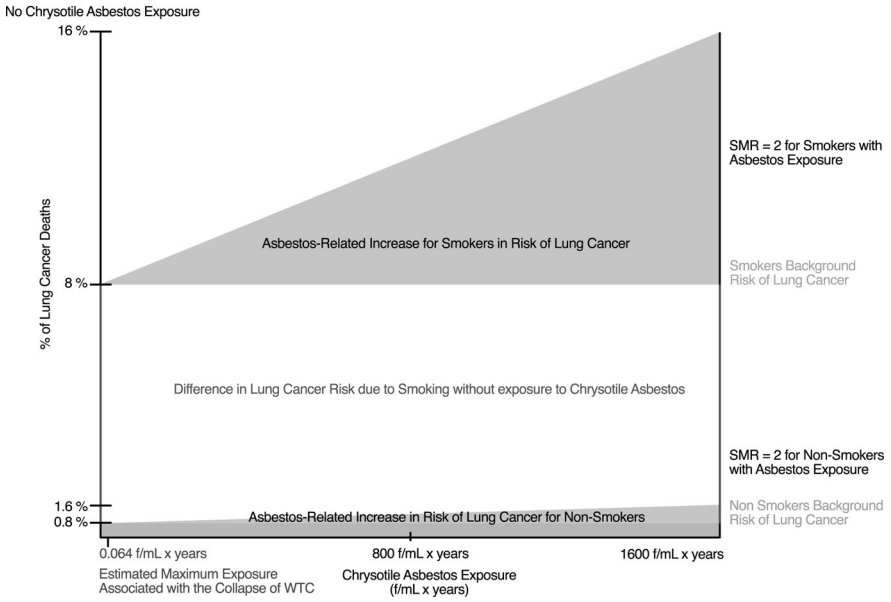

Fig. 7. Comparison of the risk of lung cancer for nonsmokers and smokers as a function of exposure to chrysotile asbestos.

miology studies of some workers with low chrysotile exposures found no increased risk of lung cancer even though the workers smoked.[14] Indicating the dose–response may be sub linear and this is an additional reason why our estimates are only an upper limit. The average age of Lower Manhattan residents at the time of the exposure was 38 years. Applying the adjustment indicated;[12] we calculated the risk of mesothelioma associated with the incremental ambient asbestos exposure as the result of the events of 9/11 to be 1.39×10^{-6}.

For a cumulative asbestos exposure of 0.28 f/mL × years, the increment in mesothelioma for the 57,514 residents of Lower Manhattan would be less than 1 case (expected number of cases equals 0.08). The probability of more than one case occurring is less than 0.01. Mesothelioma is a very rare tumor with a lifetime background rate estimated to be 3.6×10^{-4}; therefore, in a population of 57,514, the expected number of background mesothelioma cases is 21.[15] The 9/11 related increase is less than 1% of the background and cannot be observed using epidemiological methods. If 9/11 caused even one asbestos-related cancer case, it would be indistinguishable among the background cases by any pathologist.

If the exposure was to crocidolite asbestos, the mesothelioma risk would be almost 500-fold higher.[12] 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,[16] whereas there is a paucity of epidemiological evidence demonstrating similar occurrences of nonoccupational mesotheliomas in chrysotile mining communities.[6] The latter have experienced for the last 100 years much greater cumulative exposures to chrysotile asbestos than in Lower Manhattan after 9/11 without convincing evidence of chrysotile related environmental mesotheliomas occurring.[6]

Lung cancer risk resulting from asbestos exposure is modeled as an increment relative to the background risk of lung cancer. If the entire population smoked cigarettes, approximately one asbestos-related lung cancer case would be expected. If no one smoked, the risk of lung cancer would be 10-fold lower. Given the smoking rates for the residents of Lower Manhattan of approximately 25%, 1278 background lung cancers cases would be expected (1150 in smokers).[17–19] The model projects a relative risk of lung cancer associated with incremental exposure to asbestos from the events of 9/11 equal to 1.7×10^{-4}.[12] The expected number of lung cancers is 0.22 and the probability of more than one incremental case occurring is approximately 0.02. If an additional 9/11 related case were to occur, it would be indistinguishable among the 1278 background lung cancer cases. 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 (Fig. 7).

On the basis of the results presented above, we conclude that the exposure to asbestos in ambient air after the collapse of the WTC towers has resulted in no more than a negligible increase in the risk of cancer for the residents of Lower Manhattan. The critical underpinnings of this conclusion are (1) assuming that the dust particles sampled were representative, both in space and time of the dust from the collapse; (2) identifying the asbestos fiber-type as chrysotile; (3) expending sufficient resources on air sampling and analysis to produce accurate estimates of airborne asbestos concentrations and establish the return to background following 9/11; and (4) assessing the risks of mesothelioma and lung cancer separately rather than as an aggregate of asbestos-related cancers. Differentiating mesothelioma from lung cancer and chrysotile asbestos from other asbestos fiber-types are both essential for meaningful risk calculations.

EPA's aggregate risk model does not differentiate fiber types and combines mesothelioma and lung cancer. The EPA aggregate model indicates a risk of cancer equal to 2.1×10^{-4}, which is equivalent to 12 excess cancers, for the incremental ambient

TABLE 2.
Expected Number of Mesotheliomas Resulting from Asbestos Exposure due to Events of 9/11 Based on Two Risk Assessments by EPA[22,23]

EPA's "All Fiber-Types Are the Same" Mesothelioma Potency Factor: ($K_M = 1 \times 10^{-8}$)[22]

	Lower Manhattan Asbestos Exposure Resulting from 9/11	Lifetime Mesothelioma Risk for an Asbestos Exposure of 0.01 f/mL for one year (per 100,000)	Lifetime Mesothelioma Risk for Lower Manhattan Asbestos Exposure Resulting from 9/11 (per 100,000)	Number of Residents	Expected Number of Mesotheliomas
Males					
Age					
0	0.065	11.2	72.6	265	0.2
10	0.065	7	45.4	3,342	1.5
20	0.065	4.1	26.6	4,473	1.2
30	0.065	2.1	13.6	5,846	0.8
50	0.065	0.3	1.9	15,317	0.3
Total				29,242	4.0
Females					
Age					
0		14.6	94.6	256	0.2
10		9.4	60.9	3,231	2.0
20		5.6	36.3	4,324	1.6
30		3.1	20.1	5,652	1.1
50		0.6	3.9	14,809	0.6
Total				28,272	5.5
		Total Number of Mesotheliomas Expected = 9.5			

Mesothelioma Potency Factor for Chrysotile Asbestos ($K_M = 4 \times 10^{-10}$)[23]

	Lower Manhattan Asbestos Exposure Resulting from 9/11	Lifetime Mesothelioma Risk for an Asbestos Exposure of 0.01 f/mL for one year (per 100,000)	Lifetime Mesothelioma Risk for Lower Manhattan Asbestos Exposure Resulting from 9/11 (per 100,000)	Number of Residents	Expected Number of Mesotheliomas
Males					
Age					
0	0.065	0.45	2.9	265	0.0
10	0.065	0.28	1.8	3,342	0.1
20	0.065	0.16	1.1	4,473	0.0
30	0.065	0.08	0.5	5,846	0.0
50	0.065	0.01	0.1	15,317	0.0
Total				29,242	0.2
Females					
Age					
0		0.58	3.8	256	0.0
10		0.38	2.4	3,231	0.1
20		0.22	1.5	4,324	0.1
30		0.12	0.8	5,652	0.0
50		0.02	0.2	14,809	0.0
Total				28,272	0.2
		Total Number of Mesotheliomas Expected = 0.4			

asbestos exposure during and after 9/11 in Lower Manhattan. Camus and coworkers evaluated the two component parts of EPA's aggregate risk model, the model for lung cancer and the model for mesothelioma.[20,21] They found that both the lung cancer model and the mesothelioma model substantially overstated risk when compared to actual cases in areas of environmental chrysotile exposure in Canada.

To further demonstrate the importance of differentiating among fiber-types, we applied the mesothelioma model used by EPA to exposures in Lower Manhattan but incorporated a potency factor specific for chrysotile rather than EPA's potency factor that treat all fiber-types alike. The chrysotile potency (K_m) factor for mesothelioma developed in research conducted for EPA[22] is 4×10^{-10}, EPA's all-inclusive fiber-type potency factor (K_m) for mesothelioma is 1×10^{-8}.[23] We projected the number of expected mesothelioma cases using EPA's mesothelioma risk model (their Table

6–3[23]), adjusted to reflect an average continuous exposure of 0.065 f/mL for 1 year, and the population age distribution of Lower Manhattan. With EPA's all-inclusive fiber-type potency factor for mesothelioma, 9.5 mesothelioma cases are expected corresponding to the asbestos exposures resulting from the events of 9/11. With the chrysotile potency factor, 0.4 mesothelioma cases are expected (Table 2).

The studies by Camus and coworkers and our analysis of EPA's mesothelioma model described above further support our estimates of less than one expected mesothelioma and less than one expected lung cancer. The difference between EPA estimates and our estimates is the consideration of asbestos fiber-type, which clearly is an important risk factor for mesothelioma. Recent estimates of the relative mesothelioma potencies are 500:100:1 for crocidolite, amosite and chrysotile respectively,[12] and 750:1 for amphibole fibers (amosite and crocidolite) versus chrysotile fibers.[22] By averaging the mesothelioma risk for the three different asbestos fiber-types the EPA substantially overstates the mesothelioma risk for chrysotile, which is the most common and least potent of the three fiber- types.

Conclusion

This report shows that the risk of developing cancer *from asbestos exposures* during, and subsequent to, the collapse of the World Trade Center towers is negligible; we make no estimate of the risk from inhaling fine particulate matter.

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