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Sent: Monday, November 21, 2005 1:12 PM
To: zzMSHA-Standards - Comments to Fed Reg Group
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Subject: Comments on Asbestos Exposure Limit-RIN:1219-AB24

Please find attached our comments on the above referenced matter. I have attached a pdf of one of our references which may not be part of the rulemaking record. The other reference by Ross and Nolan (2003) in available as a pdf at www.rpnolan.com under publications. All the other paper we refer to our in the rulemaking record. If you have any questions or need additional information please feel free to contact me. Cordially.

Comments on Part II, Department of Labor, Mine Safety and Health Administration 30 CFR Parts 56, 57, and 71 RIN 1219-AB24 Asbestos Exposure Limits; Proposed Rule

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November 21, 2005

MSHA's proposal to simply adopt OSHA's air monitoring methodology and permissible exposure limit (PEL) *will not* be effective for the mining environment unless changes are made to the asbestos exposure methodology to ensure that only asbestos fibers are counted (Proposed Rule: Asbestos Exposure Limit). Also, the assumption implicit in the asbestos PEL that the risk of asbestos-related disease is the same for all six asbestos fiber-types was never well supported and now is clearly known to be incorrect. In 1971 the asbestos PEL was 12 f/mL for both MSHA and OSHA. Since then, OSHA has lowered it 4-times to 0.1f/mL. During the same period MSHA has lowered it once to 2f/mL, without ever presenting a justification for the assumption that all the asbestos fiber-types are the same. In this proposal, yet again MSHA has failed to justify or explain this assumption.

MSHA must clearly define what they mean by the word asbestos. Do they wish to go back to the ancient Greek, Pliny, who first described asbestos and gave it the name that means "does not burn"? If not, they must recognize that the hazards of the different asbestos fiber-types are so different that each must be separately defined. Or do they mean a silicate mineral which has grown in a natural fibrous form (asbestiform) used commercially as asbestos (Ross et al. 1984, Ross and Nolan 2003). Then they must decide between two basic alternatives: Are all asbestos forms equally dangerous until they are proved different or are all asbestos forms different in their hazards until they are proven the same? It is far preferable for these questions to be faced directly rather than implied by a particular interpretation of the data.

The best evidence now indicates a strong fiber-type risk gradient for asbestos-related mesothelioma, the risk decreasing from riebeckite (crocidolite) asbestos, to grunerite (amosite) asbestos to chrysotile asbestos in the ratio 500:100:1 (Hodgson and Darnton 2000). The lung cancer risk gradient is not as clear and of a lesser magnitude. But the best available evidence for both asbestos-related cancers is that a single asbestos PEL is not adequate to address the potency differences. This is not a new concept, the British asbestos PEL for riebeckite (crocidolite) asbestos was 0.2f/mL and chrysotile & grunerite (amosite)

asbestos was 2f/mL in 1968. It would be another18 years before the OSHA asbestos PEL would be reduced to this level. MSHA does not propose to differentiate among the asbestos fiber-types but has engaged in a rulemaking which leaves open the possibility that the asbestos PEL will include mineral fibers that are clearly not asbestos.

The phase-contrast optical method (PCOM) used by OSHA to quantify occupational exposure to asbestos was developed in the 1960s and uses only morphology to determine which fibers should be counted. PCOM cannot be used to determine if the fibers being counted are asbestos. For the occupational workplace as monitored by OSHA, the presence of asbestos in the manufacturing process or in end-products use is already established, asbestos bags and asbestos-containing products are labeled, and therefore it is not necessary for the air monitoring method used to be analytical for asbestos. One simply assumes that all of the fibers meeting the dimensional criteria of the fiber counting rules are asbestos. In contrast, in the mining environment many other airborne fibers may be present that are then incorrectly counted as asbestos because of their morphology. In their 1992 final rule OSHA clearly stated these non-asbestos fibers were less active than asbestos and should not be counted in the asbestos PEL (Federal Register 1992).

When the permissible exposures limit (PEL) for asbestos was 12 f/mL, adopting the PCOM methodology in the asbestos industries was a significant advance from simply counting all particles, and assuming that the airborne fibers were all asbestos gave reasonable reliable results. As the asbestos PEL has been lowered more than 100-fold and different working environments have been monitored where non-asbestos fibers are present the limitations of the PCOM have become better understood. In occupational workplaces where tremolitic talc, other types of fibrous minerals or indeed any other fibrous materials (with lengths ≥5μm with 3 to 1 aspect ratios) are present, the airborne fiber concentrations are generally low but, as the asbestos PEL is lowered PCOM will not be able to be effectively used to control these workplaces for airborne asbestos without modification of the method. In the past, when the asbestos PEL was high, the relatively low concentrations of other fibers were not a significant source of error. With the proposed lowering of the

PEL, the contribution of non-asbestos fibers to the asbestos fiber counts will become a significant proportion of the PEL. *It is entirely possible that in specific mining environments when the asbestos PEL is exceeded the predominant fiber counted will not be asbestos.*

This limitation *has not* been particularly important in the OSHA monitored work places as the commercial use of asbestos has largely, if not completely, been abandoned. Therefore few occupational exposures continue to be monitored for asbestos. The rationale for OSHA not reducing the asbestos PEL to less than 0.1f/mL was in part based on this being the lowest value at which the PCOM method could effectively be used. The evidence indicates that, while this may be true for the OSHA workplace, it is not a reliable method for monitoring for asbestos in the mining environment where crushed rock is a primary source of non-asbestos fibers.

The NIOSH recommended PCOM method as used by OSHA has historically been very useful for monitoring airborne asbestos but it has limitations. It does not give reliable results when monitoring environments where tremolitic talc or elongate cleavage fragments of non-asbestos amphibole are present. NIOSH has for decades expressed the opinion that if a fiber meets the dimensional criteria of the fiber counting rules used to monitor asbestos in the workplace then it must be asbestos or should be controlled to the same permissible exposure limit as asbestos. Their logic here is overly protective of the methodology without giving proper consideration to the small overlap in the size distribution between asbestos and non-asbestos fiber nor the lack of epidemiological or experimental animal studies to demonstrate a health hazard remotely similar to asbestos (Fig. 1, Langer at al. 1991, Nolan et al. 1991).

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OSHA had a rulemaking to determine if the evidence available justified regulating elongate cleavage fragments of the non-asbestos amphiboles – tremolite, anthophyllite and actinolite as asbestos and concluded that the non-asbestos amphibole minerals should not to be regulated as asbestos. OSHA expressed the following conclusions in its final rule (Federal Registry 1992):

> NIOSH recommended to OSHA the following "… for regulatory purposes that cleavage fragments of the appropriate aspect ratio and length from the nonasbestiform minerals should be considered as hazardous as fibers from the asbestiform minerals" OSHA evaluated the NIOSH's recommendation and in response stated "… OSHA does not believe the current record provides an evidentiary basis to determine "the appropriate aspect ratio and length" for determining pathogenicity." OSHA was not accepting the claim that the morphological similarity between cleavage fragments and asbestos justified regulation. More than 10 years later NIOSH made similar recommendations to MSHA expressing their concern about the hazards of non-asbestos fiber.
>
> OSHA concluded "… the discussion indicates that populations of fiber and populations of cleavage fragments can be distinguished from one another when viewed as a whole. For example one can look at the distribution of aspect ratios or even widths for a population of particles as being asbestiform or nonasbestiform. However when one looks at individual particles (e.g. particles from air sampling filters) sometimes these mineralogical distinctions are not clear." The methodology that MSHA is proposing to adopt will not address these individual particles that OSHA correctly recognized as being indistinguishable from asbestos on the air filter.
>
> OSHA concluded "… for most mineral deposits, asbestos and nonasbestiform habits are distinguishable."

> "OSHA has determined that nonasbestiform ATA and asbestos anthophyllite, actinolite and tremolite should be defined separately for regulatory purposes to conform to common mineralogic usage." *OSHA concluded that mineral fibers should be regulated based on the mineralogical criteria used to define them.* In the recent public hearing NIOSH stated that the "definition of asbestos needed to be improved". We would argue that is *not NIOSH's task* to develop mineralogical definitions but rather it should use the mineralogical definition of asbestos which is already fully developed (Ross et al. 1984). What is and is not asbestos is very well understood. What is measured by the PCOM method recommended to MSHA by NIOSH is not specific for asbestos and does need development (Langer et al. 1991).

OSHA *expressed serious concern* about the potential health hazard from occupational exposure to non-asbestiform minerals but they concluded that, "…currently available evidence is not sufficiently adequate for OSHA to conclude that these mineral types pose a health risk similar to asbestos." It is more than 12 years since OSHA came to that conclusion and NIOSH has neither improved its air sampling methodology to differentiate elongate cleavage fragments nor articulated a position on the type of health hazard or cancer risk these non-asbestos fibers might present.

OHSA went further and concluded that although the non-asbestos amphiboles were not shown to be non-carcinogenic the evidence available was adequate to demonstrate that their carcinogenic potency was clearly less than that of asbestos. OSHA recognized that a small percentage of populations of cleavage fragments and asbestos would be indistinguishable but accepted that each is a unique mineral with different potential for causing a health hazard. *One critical point is that it is difficult to find environments where a high concentration of cleavage fragments or alteration products are present in the air.* However, as the PEL is lowered to 0.1 f/mL, many mining environments will exceed this PEL because non-asbestos amphiboles are very common in the rock and soil of the earth's

crust, and will always produce a few regulatory fibers when crushed (Fig. 1). As the asbestos PEL is to be lowered by 20-fold in the mining environment this lack of specificity of the PCOM method needs to be addressed, a task MSHA has not done.

By way of background MSHA made comments at both public hearings about the health hazards at associated with vermiculite exposure at Libby, Montana. The first epidemiological study reporting increased mortality from asbestos-related disease among miners was published by McDonald et al. (1986a, b) and sponsored by WR Grace and Company. These results were largely confirmed by the MSHA sponsored study by NIOSH which followed a year later (Amandus et al. 1987a, b, c). McDonald et al. 2002, 2004 recently updated the mesothelioma mortality in the Libby cohort.

The long latency for asbestos-related cancer – mesothelioma and lung cancer – indicates the asbestos exposures important in the etiology of these increased cancer risks occurred more than 20-years earlier. In 1963, the year WR Grace took over operation of the vermiculite mine, the earlier exposure and latency period for asbestos-related disease had largely been established and the outcome of the two epidemiology studies would have been very similar had the mine closed in 1963. The point we wish to emphasize is that the different asbestos PELs of OSHA and MSHA had little or no impact on the epidemiological results of the two studies. The early exposures at Libby were reported to be as high as 130 f/mL in the dustiest jobs and MSHA had an asbestos PEL of 30f/mL in 1968 when the British PEL was 2f/mL. The asbestos-related disease associated with the lower exposures which started in the 1960s is less clear and additional epidemiological studies of workers exposed starting in the mid -1960s until the mine closed in 1990 would be helpful. Had the British 2f/mL asbestos PEL been adopted by the US government in 1968 it would most likely have had a significant impact on this later cohort of Libby miners.

Although asbestiform winchite and asbestiform richterite have been reported at Libby using the methodology that EPA recommends for monitoring airborne asbestos in the non-

occupational environment –analytical transmission electron microscopy- we found the predominant asbestos fiber-type was tremolite asbestos (Nolan et al. 1991). While the presence of these sodium-rich calcic amphiboles is interesting their importance as an etiological agent in asbestos-related disease is weak when compared to the evidence for tremolite asbestos. From our review of the recent studies at Libby there was no effort made to distinguish tremolite asbestos or the two asbestiform amphiboles- winchite and richterite from the elongate cleavage fragments of amphiboles. As OSHA has recognized the significant difference between these two classes of fiber it would be useful if other government agencies would differentiate between them otherwise these reports cannot be used for health hazard evaluation.

We remain more optimistic than others that, in the fullness of time, the non-occupational asbestos-related disease in Libby will be found to be significantly less severe than it is currently being portrayed. There is a considerable record of the United States government over-estimating the hazards of asbestos-related disease. For example the Department of Health, Education and Welfare, then led by Joseph Califano, projected 2,000,000 asbestos-related deaths in the United States. The seminal error there was to project that the highest risk of asbestos-related cancer found would be experienced by anyone exposed to any asbestos. E. Cuyler Hammond of the American Cancer Society said he was "slightly puzzled" by the projections which used "tricky arithmetic". We recommend Edith Efron's *The Apocalyptic's* for the details. In Libby the evidence for asbestos-related disease in the general population is largely based on the mortality experience of the miners (who were also counted among the general population of the small town) and radiological surveys of the general population which included those with non-occupational exposure. If much of the "asbestos" dust in the Libby turns out to be cleavage fragments and non-asbestos fibers it would not surprise us if later these asbestos-related pulmonary abnormalities were found to have less pulmonary impairment and lower risk of asbestos-related cancer than are currently being predicted.

We make to specific recommendation to MSHA concerning the asbestos rulemaking:

1. It is not clearly stated in the proposal that it is possible and relatively simple to differentiate asbestos from non-asbestos fibers. The discussion in the proposal, particularly the analysis of the Libby minerals done at the US Geological Survey (Meeker et al. 2003), indicates the opposite. Asbestos has been known to be present in the vermiculite mined near Libby for about 75 years. The types of asbestos minerals present have been known in detail for at least 30 years. The report coming out of the USGS does not clearly state this and adds little, if any, useful information. The minerals regulated as asbestos in the OSHA asbestos standard are well known and methodology is highly developed to determine their presence or absence. The proposal as written does not reflect the need to use a polarized light microscopy and a geological survey of the mine for asbestos (Langer et al. 1991, Nolan et al. 1999, Ross and Nolan 2003) and we recommend MSHA use both.

2. The proposed rule is based on a 1986 asbestos risk assessment now thoroughly out-of-date. If the rule were to be enacted, the risk assessment would be 20 years old when it appeared; we recommend it be updated. A lot has changed in 20 years and the risk assessment needs to be re-evaluated and brought current with modern knowledge of asbestos-related disease. It is without question that asbestos fiber-type is an important factor in risk assessment. MSHA dismisses the importance of asbestos fiber-type without clearly stating a rationale for doing so. For example, Hodgson and Darnton (2000) reported that the mesothelioma risk is 500-fold higher for riebeckite (crocidolite) asbestos when compared to chrysotile asbestos. In comparison the most recent (1993) EPA Integrated Risk Information System

(http:www.epa.gov/iris/subst/0371.htm) under-estimates the risk for asbestos-related cancer by a factor of at least 10 for riebeckite (crocidolite) asbestos and overestimates the risks from chrysotile by a similar amount (Nolan et al. 2005). This is like saying you can drink the same amount of 151-proof rum as you can light beer and not notice the difference. Mesothelioma is a rapidly fatal malignancy which occurs five times more commonly in males than females due to occupational exposure among the males. It accounts for about 5% of the deaths in many cohorts exposed to amphibole asbestos. It is the only occupational malignancy that can be tracked in the general population based on incidence. This is possible because perhaps 75% of all the mesothelioma cases among males are asbestos-related. No other tumors are predominantly caused by an occupational exposure. The extremely high mesothelioma risk from riebeckite (crocidolite) asbestos has been known since 1960 and in 45 years the federal government has yet to enact policies to eliminate this trend. The principal reason for the continuing high male incidence of mesothelioma is the failure to address the issue of asbestos fiber-type. The incidence of mesothelioma is elevated in only a few mining locales most prominently in the vermiculite miners near Libby, Montana. Few, if any, of the other mining locales in the US have any increased incidence of this disease related to mining.

References

Amandus HE, Wheeler R, Jankovic J, Tucker J (1987a) The morbidity and mortality of Vermiculite Miners and Millers Exposed to tremolite-actinolite: Part I. Exposure Estimates. *Am J Ind Med* 11: 1-14.

Amandus HE, Wheeler R (1987b) The morbidity and mortality of vermiculite miners and millers exposed to asbestiform tremolite-actinolite. Part II. Mortality. *Am J Ind Med* 11: 15-26.

Amandus HE, Althouse R, Morgan WKC, Sargent EN, Jones R (1987c) The morbidity and mortality of vermiculite miners and millers exposed to asbestiform tremolite-actinolite. Part III. Radiographic findings. *Am J Ind Med* 11: 27-37.

Efron, E (1984) *The Apocalyptics.* Simon and Schuster, New York, NY pp. 1-590.

Federal Register (1992) Occupational Exposure to Asbestos, Tremolite, Anthophyllite and Actinolite; Final Rule 57: 24310, June 8, 1992.

Hodgson JT, Darnton A (2000) The quantitative risks of mesothelioma and lung cancer in relation to asbestos exposure, *Ann Occup Hyg* 44: 565-601.

Langer AM, Nolan RP, Addison J (1991) Distinguishing between Amphibole Asbestos Fibers and Elongate Cleavage fragments of their Non-Asbestos Analogues. In: *Mechanisms in Fibre Carcinogenesis*. Brown RC, Hoskins JA, Johnson NF (Eds), Plenum, New York, pp 253-267.

McDonald JC, McDonald AD, Armstrong B, Sebastien P (1986a) Cohort study of mortality of vermiculite miners exposed to tremolite, *Br J Ind Med* 43: 434-444.

McDonald JC, Sebastien P, Armstrong B (1986b) Radiological survey of past and present vermiculite miners exposed to tremolite, *Br J Ind Med* 43: 445-449.

McDonald JC, Harris J, Armstrong B (2002) Cohort mortality study of Vermiculite miners exposed to fibrous tremolite: an update. *Ann Occup Hyg* 46 (Suppl 1): 93-94.

McDonald JC, Harris J, Armstrong B (2004) Mortality in a cohort of vermiculite miners exposed to fibrous amphiboles in Libby, Montana. *Occup Environ Med* 61: 363-366.

Meeker, G.P., Bern, A.M., Brownfield, I.K., Lowers, H.A., Sutley, S.J., Hoefen, T.M., Vance, J.S. (2003) The composition and morphology of amphiboles from the Rainy Creek Complex, near Libby, Montana. *American Mineralogist*, 88: 1955-1969.

Nolan RP, Langer AM, Oechsle GW, Addison J, Colflesh DE (1991) Association of Tremolite Habit with Biological Potential: Preliminary Report. In: *Mechanism of Fibre Carcinogenesis*. Brown RC, Hoskins JA, Johnson NF (Eds.), Plenum, New York, pp 213-251.

Nolan RP, Langer AM, Wilson R (1999) A Risk Assessment for Exposure to Grunerite Asbestos (Amosite) in an Iron Ore Mine. PNAS USA 96(7): 3412-3419.

Nolan RP, Ross M, Nord GL, Axten CW, Osleeb JP, Domnin SG, Price B, Wilson R (2005) Risk Assessment for Asbestos-Related Cancer from the 9/11 Attack on the World Trade Center, J Occup Environ Med 47: 817-825.

Ross M, Kuntze RA, Clifton RA (1984) Definition of Asbestos. *Special Technical Publication 834*, Am Soc Testing Mat, Philadelphia, pp 139-147.

Ross M, Nolan RP (2003) History of Asbestos Discovery and Use and Asbestos-Related Disease in Context with the Occurrence of Asbestos within Ophiolite Complexes. Geological Society of America, Special Paper 373: 447-470.

Brief Biographies of the Commenters

ROBERT P. NOLAN received a PhD degree in chemistry from The City University of New York in 1986. He was been awarded a Fellowships from the Stony-Herbert Wald Foundation, National research Council, Fulbright and the International Union for Pure and Applied Chemistry and a; he is the Deputy Director of the Center for Applied Studies of the Environment and a member of the doctoral faculty in Chemistry and Earth and Environmental Sciences in The Graduate School and University Center of The City University of New York. He is the author of more than fifty scientific and is internationally recognized as an expert in the characterization and health hazard evaluation of asbestos and other minerals.

ARTHUR M. LANGER received a PhD degree in geology from Columbia University in 1965. He was elected a Fellow of both the Geological Society of America and Mineralogical Society of America. Dr. Langer was a founding member of the Environmental Sciences Laboratory at Mount Sinai School of Medicine, New York, NY. He is the Director of the Center for Applied Studies of the Environment and a Professor of Earth and Environmental Sciences in The Graduate School and University Center of the City University of The New York. Dr. Langer has authored more than 125 scientific publications and is internationally recognized as an expert in the characterization and health hazard evaluation of asbestos and other minerals. He has co-authored documents for the International Agency for Research on Cancer and the International Programme on Chemical Safety of the World Health Organization.

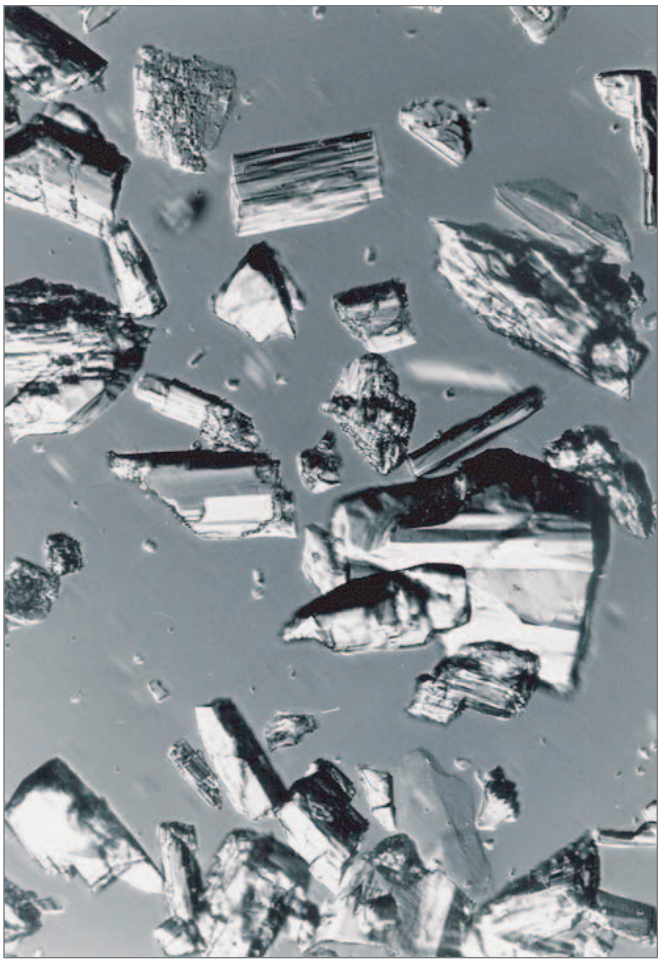
MALCOLM ROSS received a PhD in geology from Harvard University in 1962. He was elected a Fellow of both the Geological Society of America and Mineralogical Society of America. Dr. Ross has been honored to serve as President of the MSA in 1991. He conducted scientific research at the United States Geological Survey for more than 35 years, retiring in 1995. He is a Senior Scientist in the Center for Applied Studies of the Environment of The City University of New York. He has authored more than 100 scientific papers, many pertaining to amphibole minerals and asbestos. Dr. Ross has lectured nationally and internationally on the identification and heath hazard evaluation of asbestos.

JOHN ADDISON graduated with an honors degree in Geology from the University of Glasgow in 1969. He was Assistant Keeper of mineralogical and petrological collections at the Hunterian Museum of Glasgow University before being appointed as mineralogist at the Institute of Occupational Medicine in Edinburgh, one of the leading research institutions in occupational health and hygiene in the world. He was the head of the

mineralogy section there until 1993 when he resigned to establish himself as a scientific consultant in the field of asbestos and mineral analysis, toxicology and control. He has been a consultant to a wide range of mining companies including the vermiculite, talc and quarrying industries. He is recognized as an international expert on asbestos and other minerals and is the author or co-author of more than 45 scientific papers on asbestos and other minerals with particular reference to their health effects. For almost 20 years he has been a member of the UK Health and Safety Executive working groups charged with the tasks of developing analytical methods for asbestos monitoring and identification and with the oversight of the UK asbestos analytical proficiency testing schemes.

RICHARD WILSON is the Mallinckrodt Research Professor of Physics at Harvard University and immediate past Director of the Regional Center for Global Environmental Change at Harvard University. He is an Affiliate of the Center for Science and International Affairs and the Center for Middle Eastern Studies at Harvard University. He is a past Chairman of the Department of Physics at Harvard University, a past Chairman and current member of the Cyclotron Operating Committee. He is a founder of the Society for Risk Analysis. He is and has been a consultant to the United States government and the governments of numerous countries on matter of nuclear safety, toxicology, epidemiology, public health and safety and risk assessment, most recently *Risk-Benefit Analysis* (EAC Crouch)(Harvard University for Risk Analysis, 2nd Ed. 2001).

Fig. 1. Light photomicrograph of two tremolite specimens: (A) Non-Asbestos Tremolite from Shinness, United Kingdom and (B) Tremolite Asbestos from California, United States. The non-asbestos tremolite has not produced cancer in experimental animals by injection while the asbestos specimen has done so by inhalation and injection. Both specimens contain fibers with lengths equal to or greater than 5μm having aspect ratios of 3 to 1 or more. Fibers meeting the MSHA asbestos counting criteria in the non-asbestos specimen are rare. However, as the asbestos PEL is lowered to 0.1 f/mL many mining environments will exceed this PEL as the non-asbestos amphiboles are common in the earth's crust, but rarely form fiber so the non-asbestos fiber exposures will almost always be less than 1 f/mL.



(A)

(B)

Received 11/21/05
MSHA/OSRV

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*)

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-

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Supported by a Higher Education Advanced Technology grant from the State of New York and the International Environmental Research Foundation of New York City.

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DOI: 10.1097/01.jom.0000167273.17109.6d

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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

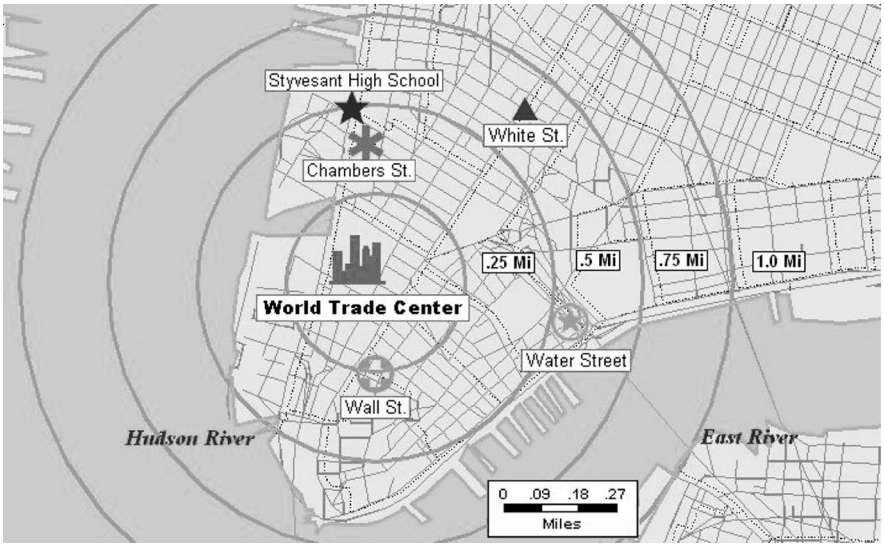

Fig. 4. The highlighted area of lower Manhattan has 57,511 residents according to the 2000 U.S. Census and was used in the risk assessment as the general population. Three settled dust samples were collected in the area of Stuyvesant High School on the Westside Highway (★, one from a motor vehicle on 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.

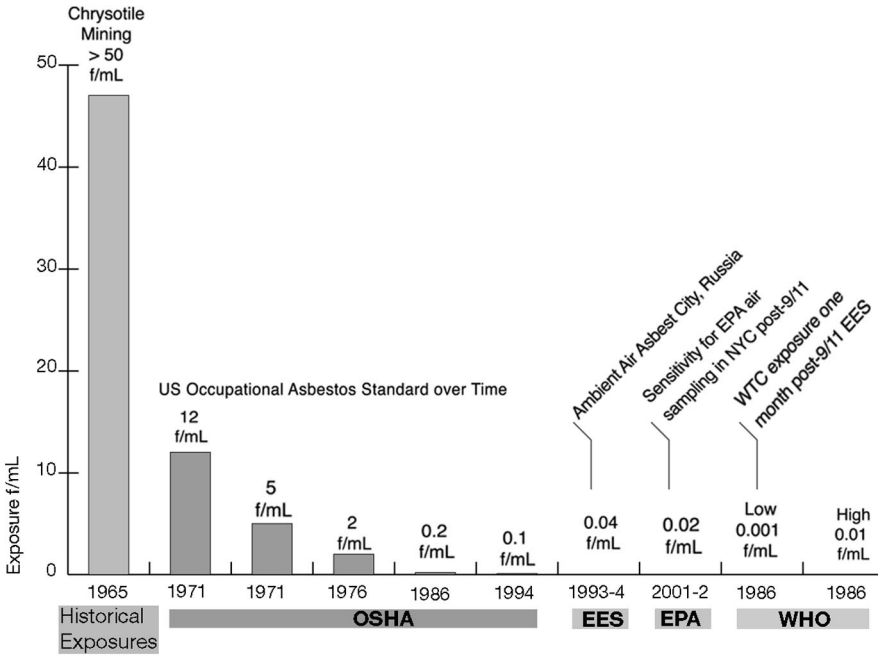

Fig. 5. Comparison of Asbestos Exposures from the collapse of WTC complex with historical, permissible and background asbestos exposures. Note the United States Environmental Protection Agency (EPA) does not determine the actual airborne concentration of asbestos but only reports the number of sturctures 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

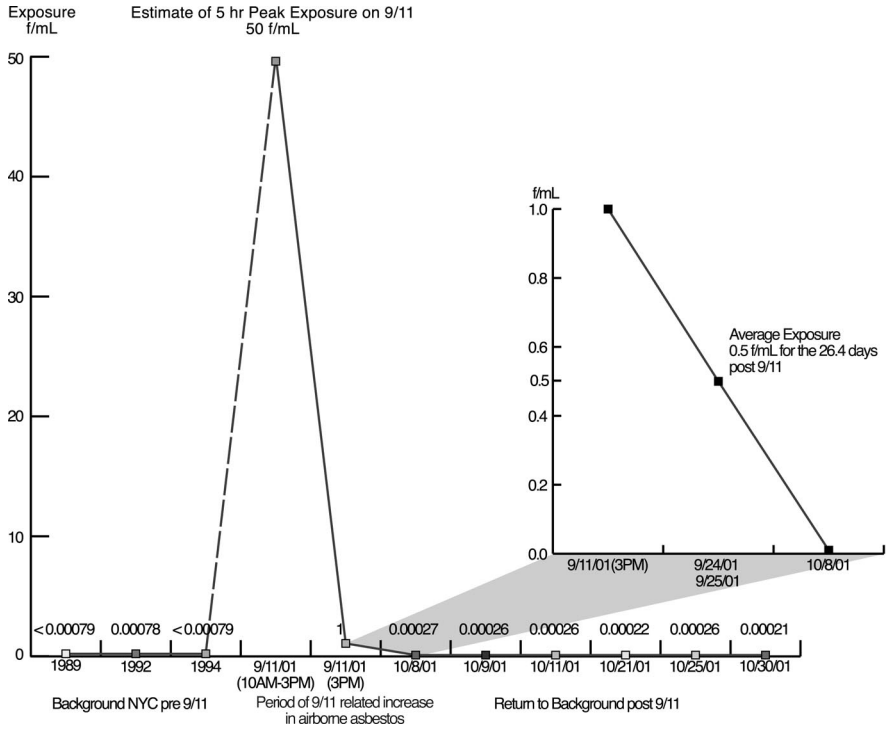

Fig. 6. Estimates of the chrysotile asbestos exposure to the general population from the dust released when the Twin Towers collapsed and while the airborne concentration of asbestos was elevated. The best estimate of the maximum cumulative chrysotile asbestos exposure to the general population of Lower 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-

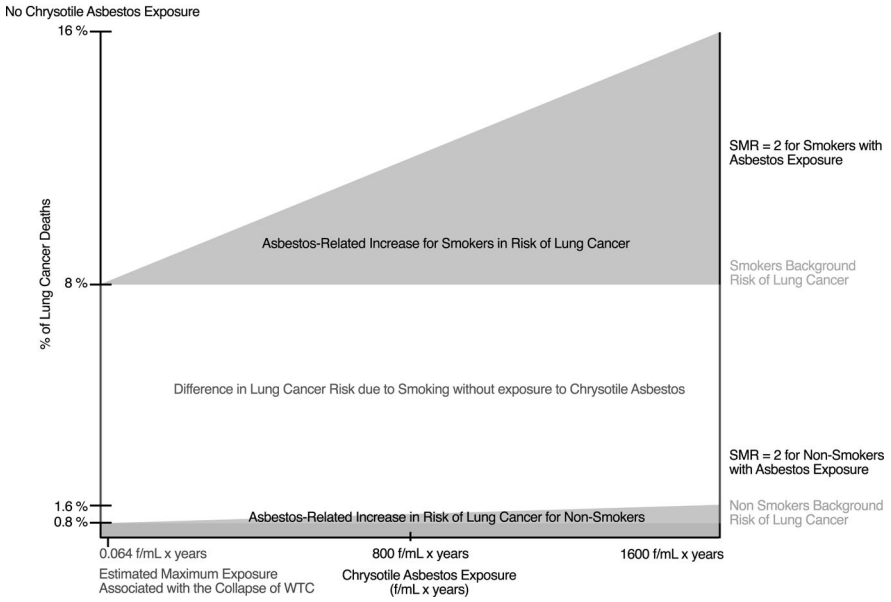

Fig. 7. Comparison of the risk of lung cancer for nonsmokers and smokers as a function of exposure to chrysotile asbestos.

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.

Acknowledgments

We thank Drs. Arthur M. Langer, J.B.L. Gee, and E.E. McConnell for very helpful suggestions that led to improvements of this report. The authors would also like to thank Jesse W. Liss for his valuable help in collecting the air samples and Barbara G. Mensch, Robert Essel NYC/Corbis, and John Ricasoli for the photographs and Angelina Nikolova for the graphics.

References

1. Office of Inspector General. Evaluation Report on EPA's Response to the World Trade Center Collapse: Challenges, Successes and Areas of Improvement. Report No. 2003-P-00012, August 21, 2003. Available from: URL: http://www.epa.gov/oigearth/index.htm; Internet; accessed April 22, 2005.
2. Reitz WB, Nicholson WJ, Holaday DA, Selikoff IJ. Application of sprayed inorganic fiber containing asbestos: occupational health hazard. *Am Indust Hyg Assn J*. 1972;33:178–191.
3. Federal Emergency Management Agency (FEMA) 2002 World Trade Center Building Performance Study Data Collection, Preliminary Observations and Recommendations, FEMA 402/May 2002. Federal Insurance and Mitigation Administration, Washington, DC and FEMA Region II, New York Available from: URL: http://www.fema.gov/library/wtcstudy.shtm; Internet; accessed April 22, 2005.
4. ISO 10312 International Standard Ambient Air— Determination of Asbestos Fibers—Direct Transfer. Transmission Electron Microscopy Method, 1995.
5. Nolan RP, Langer AM. Concentration and type of asbestos fibers in air inside buildings In: Nolan RP, Langer AM, Ross M, Wicks FJ & Martin RF, eds. *The Health Effects of Chrysotile Asbestos: Contribution of Science to Risk-Management Decisions.* Ottawa, Canada: Canadian Mineralogist, Spec Publ 5; 2001:39–51.
6. Shcherbakov SV, Kashansky S, Domnin SG, et al. The health effects of mining and milling chrysotile: The Russian experience. In: Nolan RP, Langer AM, Ross M, Wicks FJ, Martin RF, eds. *The Health Effects of Chrysotile Asbestos: Contribution of Science to Risk-Management Decisions.* Ottawa, Canada: Canadian Mineralogist, Spec Publ 5; 2001:187–198.
7. Langer AM, Morse RG. The World Trade Center catastrophe: was the type of spray proofing a factor in the collapse of the towers? *Indoor Built Environ*. 2001; 10:350–360.
8. World Health Organization (1986) International Programme on Chemical Safety (IPCS), Environmental Health Criteria 203, Chrysotile Asbestos. Available from URL: http://www.who.int/docs/ehc_203.htm.
9. NYC Department of Health and US Agency for Toxic Substances and Disease Registry, Final Report of the Public Health Investigations to Assess Potential Exposure to Airborne and Settled Surface Dust in Residential Areas of Lower Manhattan. September, 2002:20.
10. Gibbs GW, DuToit RS. Environmental data in mining. In: *Biological Effects of Asbestos*. Geneva: IARC Publication No. 8; 1973:311–320.
11. US-Environmental Protection Agency. Exposure and Human Health Evaluation of Airborne Pollution from the World Trade Center Disaster. External Review Draft, October, 2002.
12. Hodgson JT, Darnton A. The quantitative risk of mesothelioma and lung cancer in relationship to asbestos exposure. *Ann Occup Hyg*. 2000;44:565–601.
13. Asbestos File in EPA's Intergrate Risk Information System (IRIS) Available for http://www.epa.gov/iris/subst/0371.htm; Internet; accessed April 22, 2005.
14. Gibbs GW. Health effect associated with mining and milling chrysotile asbestos in Quebec and the role of tremolite. In: Nolan RP, Langer AM, Ross M, Wicks FJ, Martin RF, eds. *The Health Effects of Chrysotile Asbestos: Contribution of Science to Risk-Management Decisions.* Ottawa, Canada: Canadian Mineralogist, Spec Publ 5; 2001:165–175.
15. Price B, Ware A. Mesothelioma trends in the United States: an update based on SEER data for 1973 through 2003. *Am J Epidemiol*. 2004;159:107–112.
16. Browne K, Wagner JC. Environmental exposure to amphibole-asbestos and mesothelioma. In: Nolan RP, Langer AM, Ross M, Wicks FJ, Martin RF, eds. *The Health Effects of Chrysotile Asbestos: Contribution of Science to Risk-Management Decisions.* Ottawa, Canada: Canadian Mineralogist, Spec Publ 5; 2001: 21–28.
17. Doll R, Peto R. The causes of cancer: quantitative estimates of avoidable risks of cancer in the United States today. *J Natl Cancer Inst*. 1981;66:1191–1380.
18. Zang EA, Wynder EL. Smoking trends in the United States between 1969 and 1995 based on patients hospitalized with nonsmoking related disease. *Prev Med*. 1998; 27:854–861.
19. Wilson R, Crouch EAC. Risk-benefit analysis. Cambridge, MA: Harvard University Press; 2001:230–231.
20. Camus M, Siemtiatycki J, Case BW, Desy M, Richardson L, Campbell S. Risk of mesothelioma among women living near chrysotile mines versus US EPA Asbestos Risk Model: Preliminary Findings. *Ann Occup Hyg*. 2002;46: 95–98.
21. Camus M, Siemiatycki J, Meek B. Nonoccupational exposure to chrysotile asbestos and the risk of lung cancer. *N Engl J Med*. 1998;338:1565–1571.
22. Final Draft: Technical Support Document for a Protocol to Assess Asbestos-Related Risk. EPA 9345.4-06, October 2003.
23. Airborne Asbestos Health Assessment Update 1986. EPA/600/8-84/003F.