PLAINTIFF'S EXHIBIT

MBL-127

USAF OEHL REPORT
84-125C0111B0B

ASBESTOS - AN UPDATE OF EPIDEMIOLOGY AND PATHOLOGY SINCE 1976

USAF Occupational and Environmental Health Laboratory
Aerospace Medical Division (AFSC)
Brooks Air Force Base, Texas 78235

NOTICES

When U.S. Government drawings, specifications, or other data are used for any purpose other than a definitely related Government procurement operation, the Government thereby incurs no responsibility nor any obligation whatsoever, and the fact that the Government may have formulated, furnished, or in any way supplied the said drawings, specifications, or other data, is not to be regarded by implication or otherwise, as in any manner licensing the holder or any other person or corporation, or conveying any rights or permission to manufacture, use, or sell any patented invention that may in any way be related thereto.

The mention of trade names or commercial products in this publication is for illustration purposes and does not constitute endorsement or recommendation for use by the United States Air Force.

Do not return this copy. Retain or destroy.

Please do not request copies of this report from the USAF Occupational and Environmental Health Laboratory. Additional copies may be purchased from:

National Technical Information Service
5285 Port Royal Road
Springfield, Virginia 22161

Government agencies and their contractors registered with the DTIC should direct requests for copies of this report to:

Defense Technical Information Center (DTIC)
Cameron Station
Alexandria, Virginia 22314

This report has been reviewed by the Public Affairs Office and is releasable to the National Technical Information Service (NTIS). At NTIS, it will be available to the general public, including foreign nations.

This technical report has been reviewed and is approved for publication.

William E Mabson

WILLIAM E. MABSON, Colonel, USAF, BSC
Commander

UNCLASSIFIED

SECURITY CLASSIFICATION OF THIS PAGE *(When Data Entered)*

REPORT DOCUMENTATION PAGE		READ INSTRUCTIONS BEFORE COMPLETING FORM
1. REPORT NUMBER 84-125 C0111BOB	2. GOVT ACCESSION NO.	3. RECIPIENT'S CATALOG NUMBER
4. TITLE *(and Subtitle)* Asbestos - An Update of Epidemiology and Pathology Since 1976		5. TYPE OF REPORT & PERIOD COVERED FINAL - February 1984 6. PERFORMING ORG. REPORT NUMBER
7. AUTHOR(s) R.T.P. deTreville, et al.		8. CONTRACT OR GRANT NUMBER(s)
9. PERFORMING ORGANIZATION NAME AND ADDRESS USAF Occupational and Environmental Health Laboratory, Brooks AFB TX 78235		10. PROGRAM ELEMENT, PROJECT, TASK AREA & WORK UNIT NUMBERS
11. CONTROLLING OFFICE NAME AND ADDRESS USAF Occupational and Environmental Health Laboratory, Brooks AFB TX 78235		12. REPORT DATE February 1984 13. NUMBER OF PAGES 50
14. MONITORING AGENCY NAME & ADDRESS(*if different from Controlling Office*)		15. SECURITY CLASS. *(of this report)* UNCLASSIFIED 15a. DECLASSIFICATION/DOWNGRADING SCHEDULE

16. DISTRIBUTION STATEMENT *(of this Report)*
Approved for public release; distribution unlimited.

17. DISTRIBUTION STATEMENT *(of the abstract entered in Block 20, if different from Report)*

18. SUPPLEMENTARY NOTES

19. KEY WORDS *(Continue on reverse side if necessary and identify by block number)*

asbestos	mesotheliomia	ferruginous	chrysotile
crocidolite	asbestosis	fibrous dust	amosite

18-0000573

20. ABSTRACT *(Continue on reverse side if necessary and identify by block number)*
Since 1976, there has been a relative epidemic—both of asbestos-related occupational diseases and of public anxiety over possible environmental exposures resulting in cancer.[1,2,3] Increasingly, environmental and occupational health professionals and technicians are being called upon for advice by Air Force co-workers, union representatives, personnel specialists (especially those responsible for managing "Environmental Differential Pay" or EDP), supervisors (regarding compensation claims) and commanders (concerning public inquiries). As a result of consultation provided Air Force

001992

DD FORM 1 JAN 73 1473 EDITION OF 1 NOV 65 IS OBSOLETE

UNCLASSIFIED

SECURITY CLASSIFICATION OF THIS PAGE *(When Data Entered)*

wide in such matters, the USAF Occupational and Environmental Health Laboratory's (USAF OEHL) staff has spent many hours keeping abreast of progress. By communicating our experience (including Appendix I), it is hoped that those having responsibilities locally will be able to act effectively, by providing accurate and helpful information as soon as questions arise, referring problem cases promptly for assistance. In this way, larger numbers of well prepared and motivated health service personnel may contribute more meaningfully towards educating workers and the public concerning existing facts regarding asbestosis and cancer hazards, and their prevention by use of proper standards for control of harmful environmental and occupational exposures.

Report No. 84-125C0111BQB

USAF OCCUPATIONAL AND ENVIRONMENTAL

HEALTH LABORATORY

Brooks AFB, Texas 78235

Asbestos - An Update of Epidemiology and Pathology Since 1976

Prepared by:

ROBERT T.P. deTREVILLE, Col, USAF, MC
Consultant, Occupational Medicine

DANA MIRKIN, Maj, USAF, MC
Occupational Physician

WILLIAM D. CHRISTENSEN, Lt Col, USAF, BSC
Chief, Industrial Hygiene Branch

PETER A. LURKER, Capt, USAF, BSC
Consultant, Industrial Hygiene Engineer

CHARLES R. JONES, Lt Col, USAF, BSC
Chief, Consultant Services Division

Reviewed by:

R.B. GRAHAM, Colonel, USAF, BSC
Vice Commander

Approved by:

JOHN J. COUGHLIN, Colonel, USAF, BSC
Commander

Foreword

An earlier report[1] on Asbestos prepared in 1976 by the USAF Environmental Health Laboratory, Kelly AFB TX, reviewed the epidemiology and pathology of the several commercially important, naturally occurring, usually hydrated, mineral silicates which are incombustible in air and separable into filaments.

Chrysotile	Tremolite
Amosite	Anthopyllite
Crocidolite	Actinolite

The purpose of the current report is to update certain epidemiologic data and standards information contained in the earlier report. Hopefully, it will also provide the field with sufficient orientation concerning current concepts of asbestos bioeffects and occupational health practices in the Air Force to allow proper utilization of existing guidance and consultation where problems arise.

As this report is being written, OSHA has just announced an emergency temporary standard (as an intended change in its 1976 Permissible Exposure Limit), and EPA is considering environmental control standards. As a result, numerous questions concerning asbestos hazard at work, at play, in schools and at home are certain to be asked. Availability of the information contained herein may suffice to allow local medical problem-solving. In any event, it should be of value to environmental and occupational health personnel, wherever located, in recognizing problems and referring them to USAF Occupational Environmental Health Laboratory's professional and technical staff of consultants for assistance.

EXECUTIVE SUMMARY

When our first publication, Asbestos: A Brief Review of the Epidemiology and Pathology, appeared in 1976, there was an existing national consensus standard of 5 fibers > 5 μ/cc, which the Occupational Health and Safety Administration (OSHA) had adopted directly from the American Conference of Governmental Industrial Hygienists (ACGIH) Threshold Limit Values (TLVs) for 1968 along with all other TLVs. In 1976, the TLV had been lowered to 2 fibers > 5 μ/cc based on British data and OSHA followed suit 1 Jul 76.

The National Institute for Occupational Safety and Health (NIOSH) had been given the responsibility, under the Occupational Safety and Health Act of 1970, of developing criteria documents and providing recommendations to OSHA to help guide standards setting. ACGIH TLVs had been set to prevent occupational diseases, i.e., impairment leading to disability and death. Because of a long latency period it was not possible to be certain that the ACGIH TLV would protect against occupationally induced lung cancer and malignant mesotheliomas. The British Occupational Hygiene Society suggested a standard of safety of 100 fiber years (or 2 fibers > 5 μ/cc for a 50-year working lifetime) in view of the excess mortality being attributed to asbestos exposures considerably above this level. Reports on the British cohort in 1955, 1965, 1968 and 1977 (and a 1978 review by the same authors) have been reassuring. Preliminary claims based on data presented in NIOSH's recommendation for the equivalent of zero level threshold for asbestos have been effectively refuted by subsequent, more extensive studies summarized in the ACGIH's documentation of TLVs since 1981. It must be recognized that there are certain areas of controversy which remain and which require further research.

The primary purposes of this report are: (1) to share with Air Force occupational and environmental health specialists, physicians and other interested personnel, the experience of our consulting staff as outlined in Appendix I, which contains the text of direct testimony in an environmental differential pay issue; (2) to review advances in knowledge and understanding of asbestos, generically, but more specifically to review the bioeffects of the most widely used types which have different TLVs based on important differences in bioeffects in vivo and in vitro; and, (3) to stress the hazard of lung cancer from cigarette smoking as being two times more likely than from uncontrolled asbestos exposure by itself--but 50 times as likely from smoking with uncontrolled asbestos exposure. While not as high as had been originally estimated, the risk of smoking and asbestos exposure is great and must be communicated to all personnel concerned with employee health conservation--supervisors, unions, workers and the public. The public must understand the need to protect against high level asbestos exposure in nonoccupational settings even for short periods of time, e.g., as a homeowner or summer worker tearing out old asbestos containing insulation in the process of renovating old apartments, buildings, boilers, steam lines, plumbing or other equipment.

This report will be valuable to anyone interested in obtaining a total perspective of the complex issues involved in asbestos exposure risk assessment.

TABLE OF CONTENTS

List of Illustrations

I. INTRODUCTION

Since 1976, there has been a relative epidemic—both of asbestos-related occupational diseases and of public anxiety over possible environmental exposures resulting in cancer.[1,2,3] Increasingly, environmental and occupational health professionals and technicians are being called upon for advice by Air Force co-workers, union representatives, personnel specialists (especially those responsible for managing "Environmental Differential Pay" or EDP), supervisors (regarding compensation claims) and commanders (concerning public inquiries). As a result of consultation provided Air Force-wide in such matters, the USAF Occupational and Environmental Health Laboratory's (USAF OEHL) staff has spent many hours keeping abreast of progress. By communicating our experience (including Appendix I), it is hoped that those having responsibilities locally will be able to act effectively, by providing accurate and helpful information as soon as questions arise, referring problem cases promptly for assistance. In this way, larger numbers of well prepared and motivated health service personnel may contribute more meaningfully towards educating workers and the public concerning existing facts regarding asbestosis and cancer hazards, and their prevention by use of proper standards for control of harmful environmental and occupational exposures.

II. DEFINITION OF ASBESTOS

Geologically, seams of fibrous minerals develop within rocks of the more common structure. When crushed and processed, these separate longitudinally into finer and finer fibers. Governmental agencies and industrial organizations restrict the term asbestos to those fibrous silicates in the group which are commercially exploitable (Fig 1).[2] Most important of these are the serpentine mineral chrysotile, which has curved fibers, and the amphibole minerals crocidolite, amosite and anthopyllite, all of which have straight fibers.

Chrysotile (white asbestos) is mined chiefly in Canada and the Union of Soviet Socialist Republic (USSR). Crocidolite (blue asbestos) is found predominantly in the Cape region of South Africa, amosite (brown asbestos) in the South African Transvaal and anthopyllite in Finland.

Asbestiform varieties of various minerals which are encountered widely in nature (e.g., tremolite, a contaminant of taconite, a low grade iron ore mined in northern Minnesota) are excluded, as are Turkish tuff (erionite, a zeolite mineral), man-made fibers (e.g., fiberglass) and rock wool.

The remarkable strength, durability and insulative properties of asbestos, known since antiquity, had not been truly exploited until the industrial revolution. Total world production estimates have risen from 50 tons a century ago to five million tons.[3] Industry in the U.S. alone now uses about one million tons annually (95% being chrysotile, imported chiefly from Canada).

Background concentrations of asbestos fibers and dusts vary widely in nature. The dynamics of fibers within branching pulmonary airways are dependent chiefly upon <u>width</u>,[4,5] rather than length. The lungs of all living persons receive and retain some fibers regardless of where they live and work.

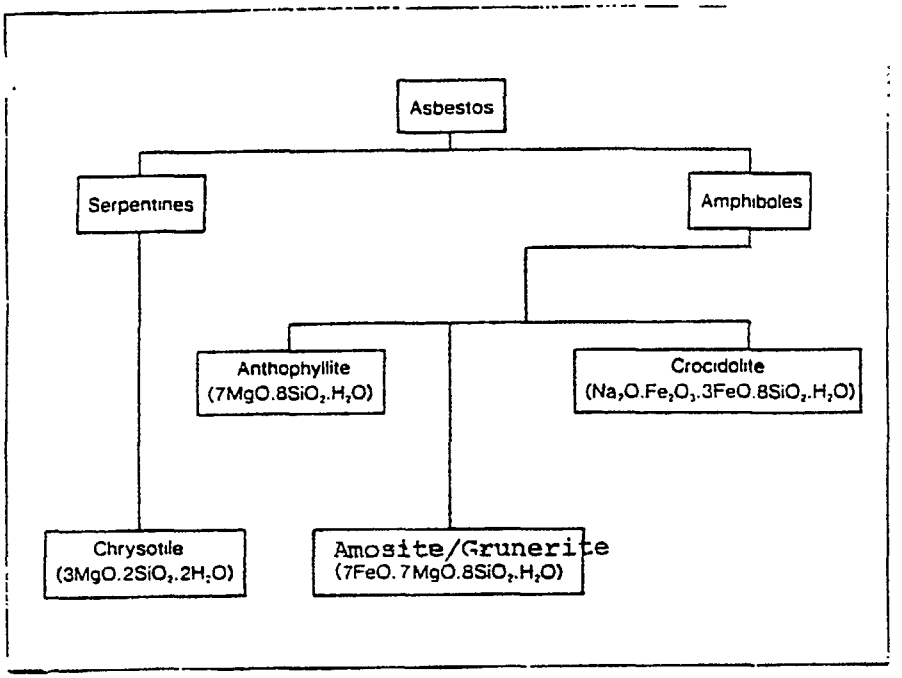

Figure 1. Types of Asbestos Used in Industry* (Modified from N.E. Jour. of Med., 306:1446, 1982).

*Mineralogic classification and chemical composition of common commercial types of asbestos.

III. ASBESTOS BODIES

Considered the hallmark of asbestos exposure[2] (not of asbestosis), asbestos bodies typically appear as rod-shaped structures with clubbed ends and are capable of demonstration at necropsy almost universally if carefully sought.[6] Capable of production by nonasbestos fibers[7] (including coal, glass, aluminum silicate, carborundum, elastin, vegetable dusts and man-made textiles), the proper terminology where the central core is unknown (or known to be other than asbestos) would be ferruginous body.[8] This reflects the iron containing mucoprotein coating which accounts for its golden brown color with hematoxylin and eosin stains, and the classic Prussian blue iron-staining reaction with ferrocyanide.[9] Experimentally, where asbestos exposure was known to be chrysotile, electron diffraction analysis of the central cores showed the distinctive pattern expected. In random autopsies of lungs of city dwellers studied in Pittsburgh at the Mellon Institute during 1968, however, none of the 28 ferruginous bodies showed the typical hollow, tubular structure characteristic of chrysotile. Measurements of the diffraction patterns obtained (to an accuracy of ± 1%) on each of the cores showed that none of the fibers examined were identifiable as being any of the forms of asbestos used commercially in U.S. industry. As chrysotile use predominates in this country, failure to find it as the central core of any of the 28 asbestos bodies studied was unexpected, to say the least. The crystalline diffraction pattern observed

ruled out fibrous glass, glass wool and other vitreous materials capable of producing ferruginous bodies. It was concluded that: (1) the fibers were crystalline (i.e., probably mineral); (2) they were long chain silicates; and (3) they were not chrysotile asbestos. Further studies with microprobe chemical analysis have identified amphiboles elsewhere. Such would be required, qualitatively and quantitatively, to determine the atomic composition and permit identification of unknown fibers in nonindustrially exposed persons' asbestos bodies.[10,12] In view of these findings, the significance of asbestos bodies, even as the hallmark of asbestos exposure, is now questionable and has been of very limited value in forensic evaluations of asbestosis and lung cancer (described later).

Before proceeding, however, an interesting question arises concerning relevance of the asbestos bodies' coated fibers or central cores to the much more numerous bare fibers found in the lungs of city dwellers.[12] Fibers outlined in Figures 2 and 3 present detailed observations of the wide variety of shapes, while Table 1 shows concentrations of fibers per milligram of dry lung weight in seven city dwellers. Also shown, for comparison, are similar findings in two long-time workers from a fibrous glass factory, and one from an asbestos products plant, who died of natural causes. It was estimated that some of the lungs contained at least 3-4 million fibers.[13] In addition, fractured crystalline plates, characteristic of talc, not shown here but seen occasionally while counting naked fibers, helped confirm the ubiquity of respirable fibers in man's environment. Two comparative estimates of concentrations of asbestos fibers in urban areas of the U.S. are shown in Table 2.

Some rather basic experimental observations have helped explain the relationship between asbestos bodies and bare fibers.[9] The finding that all dust fibers become coated with something, in early calcification, suggested that only a few fibers were in a suitable chemical environment allowing asbestos body formation. There is a great increase in acid mucopolysaccharides in calcifying tissue and this was shown to be present by histochemical staining, including colloidal iron (Hale's stain). This helped indicate the method of formation of asbestos bodies. First, coated with acid mucopolysaccharide, this material would become impregnated by any colloidal iron in the area, the most abundant source being ferritin. In cellular tissues, the supply of ferritin should be good in comparison to acellular, avascular collagen--hence the paucity of iron in calcifying tissues. Further observations have shown the stepwise saturation of the coating by ferritin granules extending centrally from the periphery of newly forming asbestos bodies. These interesting findings of research from Cambridge University, U.K., must be kept in mind in attempting to understand and explain the relative significance (or lack thereof) of asbestos bodies in presence of pathogenic changes (described later).

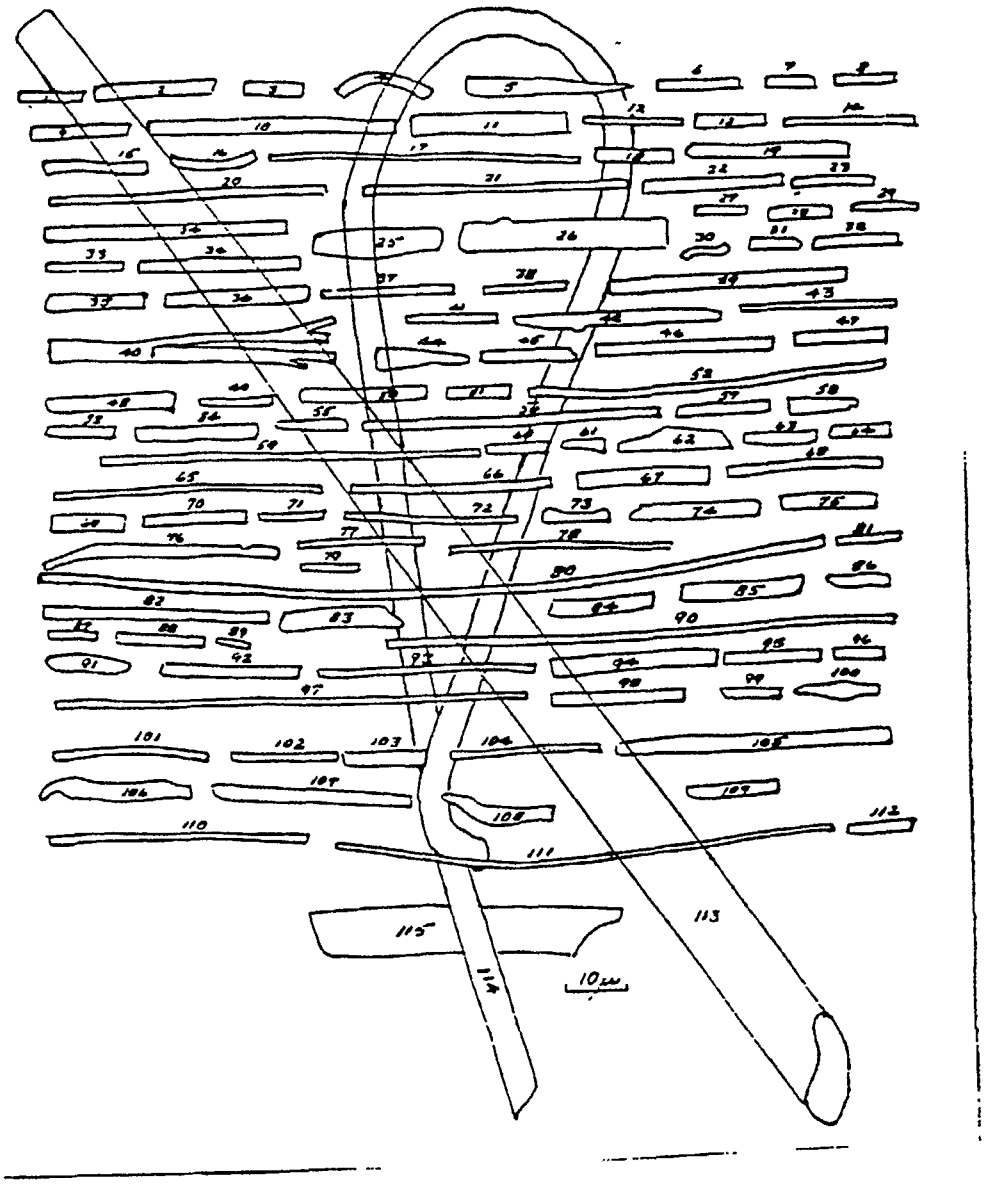

Figure 2. Bare Fibers* (Modified from Gross[13])

*Fiber outline tracings from images projected form 35-mm negative film by means of enlarger. Most fibers have diameters of 2 µ and lengths less than 25 µ. Rare fiber is gigantic (up to 1-mm in length) and too large to be accomodated in alveolar tissue. These were probably lodged within smaller airways and were not included in the quantitations.

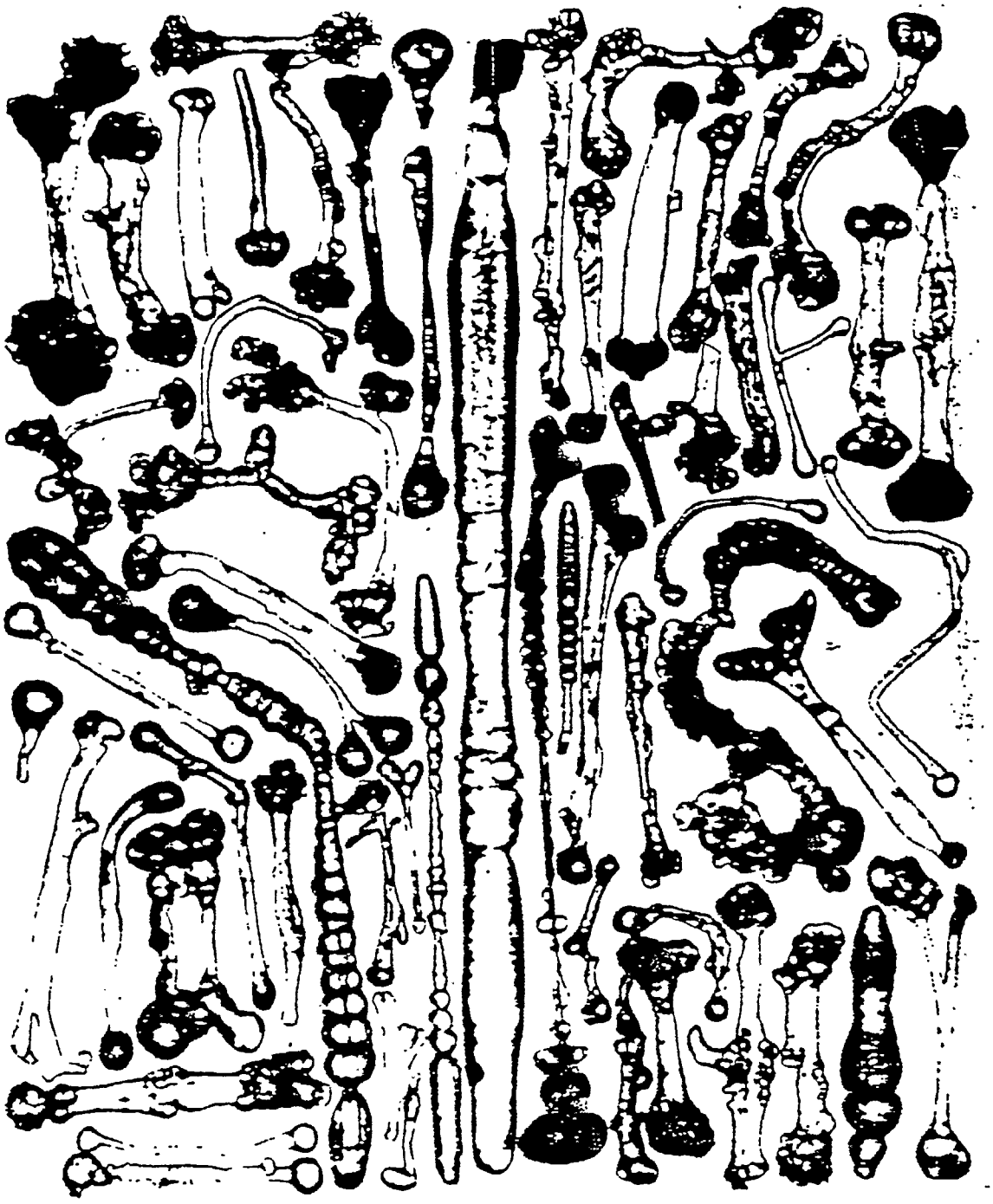

Figure 3. Ferruginous Bodies* (Modified from Gross[13])

*Collection of individually photographed ferruginous bodies from lungs of 74-year old Pittsburgh woman (A69-80) who died of Escherichia colisepticemia (X 1,000).

Table 1

Quantitation of Fibrous Dust Particles Ferruginous Bodies in Human Lungs
(Modified from Gross[18])

					No. of Fibers									Ferruginous Bodies			
Case	Sex	Age	Total Dust as % Dry Wt/Lungs	Per G Dry Wt/ Lungs X 10^3	Per mg/Dust	Mean Vol Fibers, cm, μ	% Fiber/ Wt Dust/ Wt	Mean Diam. Fibers, μ	% Fibers w/Diam. ≤2μ	≤3μ	Mean Length Fibers, μ	% Fibers w/Lengths 5μ–25μ	>50μ	No. Per mg Dust	No. Per G/Dry Lung	No. Fibers/ Body	% Fibers as Bodies
A[1]	M	66	1.5	27.9	1,840	153	0.09	2.2	69	91	28	64	8	*	*	*	*
B[1]	M	59	1.3	23.9	1,850	133	0.06	2.3	58	87	25	68	5	12	160	149	0.7
C[1]	M	69	3.2	84.8	26,400	44	0.28	1.4	95	98	17	90	2	2,280	73,000	12	8
A68-299	M	75	2.7	15.4	564	84	0.01	2.0	74	97	25	67	8	11	300	51	2
A68-300	M	59	2.3	66.4	2,845	88	0.06	2.0	70	95	25	68	8	108	2,450	26	4
A68-301	F	69	1.9	15.1	783	143	0.02	1.9	61	93	20	75	4	*	*	*	*
A68-302	F	70	1.8	20.8	1,150	54	0.02	2.0	76	97	18	80	3	153	2,700	7.5	13
A69-80	F	74	1.3	21.4	1,560	88	0.04	2.2	71	97	22	70	5	292	3,927	5	18
A69-144	F	81	0.6	28.8	4,700	121	0.14	2.2	57	92	27	63	8	440	2,700	11	9
A69-198	F	46	0.7	63.3	9,070	210	0.47	2.8	33	70	28	58	9	43	300	211	0.5

[1]Workers from a fibrous glass factory
[2]Worker in an asbestos products plant
*Not done; all of the lung tissue was digested with formamide, which destroyed the ferruginous bodies.

Table 2

Concentrations of Asbestos in Urban Areas of the U.S.
(Modified from Enterline[14])

Asbestos concentration ng/m³	Mt Sinai Number of samples	Battelle Institute Number of samples
<1	61	27
1-2	58	33
2-5	45	42
5-10	12	22
10-20	8	1
20-50	1	2
50-100	2	0
Mean	3.58	3.62
Median	1.57	2.29

IV. PLEURAL PATHOLOGY

The parietal pleural plaque has been called the most common asbestos-related disorder.[3] While also caused by other filamentous dusts (such as mica, talc, diatomaceous earth, zeolite and other fibrous silicates), asbestos exposure is considered to be the most important.[2] The pleural plaques are neither harmful nor precancerous.[3] However, as they characterize passage of at least 20 years since first filamentous dust and/or asbestos exposure, they are considered indicators that sufficient time has elapsed since initial exposure to increase the risk both of asbestosis and cancer.[3]

Once started, the plaques tend to progress and to calcify, depending on length of exposure, time since first exposure and a degree of individual predisposition or susceptibility. However, this process is not related to average or cumulative doses of fibers, as in progression of asbestosis[3] (described later).

Microscopically, plaques are composed of collagenous connective tissue with few cells. Normal mesothelium covers them and normal parietal pleural connective tissue lies beneath them. Electron microscopy has disclosed uncoated asbestos fibers within the plaques, more numerous in calcified areas. Radiologically, only a small proportion (about 15%) of plaques detectable at necropsy are detectable by standard posteroanterior (PA) chest radiography. According to the Mayo Clinic,[3] special right and left oblique chest x-ray views should be added to the PA view routinely in examining individuals suspected of having asbestos-related disease. Noncalcified pleural plaques are visualized best by radiography at 110-140 kV, while calcification within plaques is more clearly demonstratable at 80 kV. Computerized axial tomography (CT) may be helpful in detecting (or excluding) plaques in the diaphragm, in paravertebral locations and in areas near the heart. However, as CT involves greater radiation exposure, time loss and cost, its use is limited to research and forensic applications.

It must be noted that the Occupational Safety and Health Administration (OSHA) standards specify annual chest x-rays for those exposed to atmospheric concentrations of asbestos fibers without specifying the type of x-rays or the extent of exposure. The DoD 1982 Manual on Occupational Health Surveillance used the Navy's former action level of 0.5 fibers/cc for inclusion in a mandatory periodic occupational medical evaluation, including PA chest x-ray. The 1933 revision (Table 3) has reduced th action level to 0.1 fibers/cc 8-hour time weighted average (TWA), making it consistent with OSHA regulations. As the OSHA action level represents the maximum sensitivity of the environmental sampling procedure now in use, the intended interpretation seems to be: if an ambient level of asbestos can be detected, it is high enough to require biological monitoring. The stated goal of OSHA is to prevent appearance and/or progression of asbestos-related pathology and the goal set is the equivalent of zero exposure. However, as will be seen in Table 4, there is still a significant difference between background and the permissible occupational exposure level (PEL). It will be important to document, eventually, the adequacy of existing OSHA standards to accomplish the goals of employee health protection, as environmental survey techniques being developed will doubtless progressively increase sensitivity of environmental survey methods.

Persons with localized, benign parietal pleural plaques reportedly live normal lives without significant symptoms or disorders of pulmonary function.[3] The only observable change may be progressive calcification of the plaques. Fortunately, persons with calcified pleural plaques appear not to develop the more serious asbestos-related pleural thickening known as hyalinosis complicata. In the latter, the visceral pleura and underlying lung are more diffusely involved, with evidence of painful, exudative, sterile pleural effusion, followed by progressive, extensive pleural thickening, beginning low and moving upward into the thorax. Resulting restrictive changes in chest motion and pulmonary function may also be accompanied by pericardial constriction. Death in such cases may result from asphyxia, cor pulmonale or pleuropneumonia, described later. Differentiation from malignant mesothelioma requires prolonged observation and is often difficult except by exclusion.

Exudative pleural effusion may be secondary to asbestosis, bronchogenic carcinoma, cor pulmonale, or malignant pleural mesothelioma. It may also occur as a primary clinical event in persons exposed to asbestos, in which case, it has been termed benign asbestos effusion. Criteria for diagnosis are: prior history of asbestos exposure, absence of any other predisposing cause (e.g., thromboembolic disease) and spontaneous remission. Of greatest concern is the protracted effusion that masks an evolving bronchogenic carcinoma or malignant mesothelioma. Treatment is directed at relief of symptoms and development of the basis for being able to reassure the patient that more serious causes of effusion are not demonstrable.

Table 3

Occupational Health Surveillance in Potential Asbestos Hazard
(Modified From DoD 6055.5-M[84,86])

CHEMICAL AGENT	MEDICAL SURVEILLANCE PROCEDURES		COMMENTS
	A. PREPLACEMENT or BASELINE	B. PERIODIC	
Asbestos (1910.1001 and OSHA CPL 2-2.21A)	1. Comprehensive medical and work histories to elicit symptomatology of respiratory disease, smoking history and any past exposure to asbestos. 2. Physical exam 3. Clinical laboratory studies a. 14" x 17" post/ant chest x-ray b. Pulmonary function (FVC, FEV_1)	Same as in A. Frequency as follows: 1. For those currently exposed at or above the action level: ANNUALLY. 2. On termination of employment: ± 30 DAYS OF TERMINATION DATE if not examined within the last year. 3. For those not currently exposed per B.1. but whose previous exposures did meet the B.1. criteria: a. And who are less less than 45 years old: EVERY 5 YEARS. b. And who are 45 years old or older: ANNUALLY. 4. For workers in B.1. or B.3., above, who show signs or symptoms of any asbestos-related disorder: ANNUALLY.	Requirements apply to: 1. Workers currently exposed to an airborne concentration of 0.1 fibers/cubic centimeter (f/cc), as an 8-hr/day time weighted average (TWA-8) on any occasion (action level). NOTE: The permissible exposure limits (PELs) for unprotected personnel are currently 2.0 f/cc (TWA-8) and 10 f/cc (ceiling). 2. Workers currently exposed to an airborne concentration of: a. 0.5 f/cc, for any length of time on a regular basis (routine ceiling) [NOTE: "Regular basis" is defined at 3.a. below]; or b. 10 f/cc (or greater) for any time (absolute ceiling). 3. Workers not currently exposed, but whose previous exposures can be reasonably estimated to have exceeded: a. Either the action level or the routine ceiling for 15 day in any one quarter or 45 days in any one calendar year; or b. The absolute ceiling value at any time.

Note: The following additional procedures and inclusion criteria are required in the Navy:

Table 4

Lifetime Lung Cancer Deaths Resulting From
Various Levels of Asbestos Exposure Per Million Exposed
(Modified from Enterline[14])

Level of exposure (ng/m^3)	Equivalent fibers/cc (> 5 μm long)	Lung cancer deaths
1	0.000037	0.85
1.5	0.00006	2.08
5	0.0002	4.6
50	0.002	46.06
500	0.02	460.6
5,000	0.2	4,606
50,000	2*	46,060

*1975 OSHA PEL (see Table 3)

V. PARENCHYMAL FIBROSIS OR ASBESTOSIS

The pulmonary fibrosis characteristic of asbestosis is located interstitially within the lung and in the visceral pleura. This definition excludes pleural plaques and fibrosis of the parietal pleura (described above).[3]

Microscopically, early changes tend to focus upon the terminal respiratory bronchiole, where reticulin fibers, macrophages and asbestos fibers accumulate, resulting in the basic lesion of asbestosis: peribronchiolar fibrosis. The process then tends to spread, forming a diffuse interstitial fibrosis, often associated with patches of solid scarring. The pathogenesis of asbestosis has been studied experimentally and found to be different from the fibrosis caused by free silica. Experimentally, it has been impossible to produce diffuse asbestosis in animals with fibers shorter than 5-10 microns (μ), even when the average exposure of 125 million particles per cubic foot (mppcf) has been maintained. In contrast, experiments using only 1/3 as many of the longer fibers produces well-marked fibrosis in the animals after about two years. This discrepancy has been supported by observations in humans, e.g., workers in the Thetford Mills in Canada who had had exposures to both extremely heavy dust concentrations of serpentine and to very fine, short, chrysotile fibers, have not been found to develop clinical asbestosis of the type observed in American asbestos textile plants. In 1938, the U.S. Public Health Service (USPHS) studied four such textile plants where massive exposures to chrysotile dust (up to 35 mppcf in opening and picking, the dustiest operation) had reportedly occurred.[11,21] Only three doubtful cases of asbestosis were found in workers exposed to dust concentrations under 5 mppcf, whereas numerous cases were found above that level. Based on such observations, the American Conference of Governmental Industrial Hygienists (ACGIH) in 1938 set a Threshold Limit Value (TLV) for asbestos exposure at 5 mppcf. Its implementation resulted in greatly improved working conditions and health protection.

Incorporating downdraft ventilation in the 1940s not only attained the TLV effectively, but also allowed workers to see other operators at adjacent machines clearly for the first time. Addition of filter systems in the 1950s, each with over 1,000 canvas-type sacks and the ability to conduct over 50,000 ft³/min of exhausted air, lowered dustiness further to levels approaching 2 mppcf by 1955. Continuing epidemiological studies in the asbestos textile industry in the U.S. and the U.K. have shown a prompt and sustained reduction of asbestos-related diseases of all types as a result.[31] Similar reductions occurred when occupational exposure to free silica of respirable size was brought below the TLV.*

British environmental asbestos concentrations have tended to be area rather than breathing zone estimates, making comparisons with U.S. data difficult. For example, in the British literature, historically, uncontrolled exposures to asbestos dust were estimated at up to 50 fibers per cc. In the U.S., a 1979 report estimated that similarly employed workers at plants operated by Johns-Manville (JM) were exposed to 10 mppcf. If 1 mppcf = 3 fibers/cc, the exposure of JM employees would have averaged 30 fibers/cc, causing an asbestos-associated cancer (discussed later) death rate of less than 1%. Table 5 shows remarkable unanimity of British and American study results, rectifying the erroneous impression conveyed in one governmental report of 1978. Figure 4 also shows this discrepancy quite clearly.

*Interestingly, in 1929 an ACGIH-TLV of 0.1 mg/m³ of free quartz dust of respirable size had been proposed, based upon the amount present in 9 mppcf of granite dust. Subsequent to implementation of dust controls in Vermont granite quarries resultingin exposures at or below 5 mppcf TWA, no new cases of silicosis were detected over an ensuing 20-year period of observation. The suggestion of a similar 5 mppcf TLV for asbestosis prevention was more than coincidental, therefore.

Table 5

Annual Number of Cancer Deaths in the U.S. Due to Occupational Exposure to Asbestos—Various Estimates (Modified from Enterline[14])

Source	Exposed Population Alive 1980	Annual Number of Cancer Deaths	Percent of All U.S. Cancer Deaths
McDonald* (1981)		3,330	<1%
Higginson (1979)		4,000	1%
Enterline (1981)	7,600,000	4,084	1%
Nicholson et al. (1981)	9,200,000	8,500	2%
Hogan and Hoel (1981)	7-8,000,000	12,000	3%
U.S. Govt.(1978)	7-10,000,000	58-75,000	13-18%

*U.S. and Canada

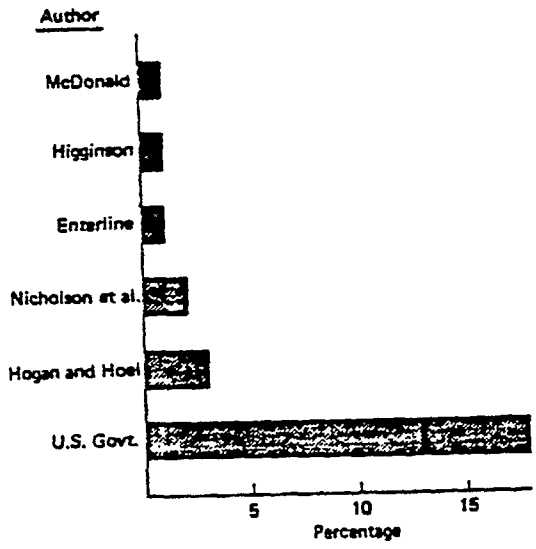

Figure 4. Estimates of the Percentage of all Cancer Due to Occupational Exposure to Asbestos in the U.S. in 1980. The McDonald estimate reflects U.S. and Canada. (Modified from Enterline[14])

The pathogenesis of asbestos is poorly understood. Asbestos-induced fibrosis is diffuse and differs markedly from the nodular fibrosis produced by free silica. Furthermore, the effect of a given weight of free silica is greatly magnified by reducing the particle size, thus increasing surface area. Reduction of the length of chrysotile fibers below 5-10 μ results in virtual elimination of asbestotic changes in animals and man according to observations on animals and man reported by Johnstone[15] in 1948. These were based chiefly on research performed at the Saranac Laboratory by Gardner, who had concluded that the reason for the difference between respirable free silica dust and long fibers asbestos could be explained by chemical action of the former and physical (or mechanical) irritation of the latter. Interestingly, in 1972, studies at the National Cancer Institute by Stanton, involving intrapleural injections of high doses of fibers longer than 4 μ and less than 0.25 μ wide, appeared to confirm a concept of solid state or foreign body carcinogenesis (discussed later) as the so-called Stanton hypothesis. More recently, evidence has been accumulating concerning important physicochemical differences between chrysotile and amphibole fibers' bioeffects, both in vitro and in vivo. It appears that there is need for further research to explain discrepancies not covered by the Stanton hypothesis.[30] Electron microscopy has demonstrated that only a small proportion of asbestos fibers in the lungs are asbestos bodies--the great majority being uncoated and submicroscopic (see Asbestos bodies above). Asbestos bodies are considered inert. Fibers shorter than 5 μ, as mentioned, appear to lack fibrogenic potential. When inhaled, longer fibers less than 3 μ in diameter are quite capable of reaching the depths of the lungs, where attempts of alveolar macrophages to engulf them cause altered secretory and immunologic activity; e.g., release of leukocyte chemotactic factor and apparent alteration of fibroblasts from normal connective tissue synthesis towards production of scarring. Sophisticated tests (cell-mediated immunity, antinuclear antibodies, rheumatoid factor and autoantibodies) may show changes indicating altered host reactivity rather than evidence of other diseases. Regrettably, however, at this time no biochemical and/or serological markers uniquely associated with exposure to asbestos have been identified, nor are there any reliable tests predictive of asbestosis or indicative of its earliest stages.[3] In evaluating past asbestos exposure in the presence of a case of lung cancer or mesthelioma (described below) today's occupational health specialist often must depend upon internal medical, radiological and nuclear medical consultations. Such consultations should come from physicians, pathologists and other scientists, fully qualified by training and experience to provide expert advice and assistance in diagnosing or ruling out the possibility of asbestos-related disease (see reference to Dr Weill below).

VI. LUNG CANCER

Whereas asbestotic changes in human lungs were described as early as 1907, the potential for development of lung cancer in asbestos textile workers was not reported until considerably later (1934 in U.K.; 1935 in the U.S.). However, the magnitude of this association in asbestos textile plants did not emerge until 1955 when results of epidemiological studies in a group of British asbestos textile workers were reported. A number of studies in Europe and the U.S. which tended to support the earlier findings were published in

the mid-1960s. However, an epidemiological investigation, begun in 1956 and published in 1958, reported the health status of a cohort of Canadian chrysotile miners who had at least five years of exposure and who were in the industry in 1950.[17] In this review which contained 92 references, the authors questioned the ability of asbestos, per se, to cause cancer. Further studies, including the same population, made by McDonald and published in 1971, extended the period of observation 16 years to 1966 and concluded that there was a five-fold difference in lung cancer rates between those maximally and minimally exposed.[18] A multiplicative risk was noted for the combined effects of asbestos dust and cigarette smoking. Concurrently, it was shown experimentally that finely milled chrysotile dust exposure incapable of producing diffuse asbestosis in rats, produced a high incidence of lung tumors.[19] This was despite very heavy exposure averaging 86 mg/m^3 for six hours-a-day, 5 days-a-week for up to 62 weeks. Wearing away of the steel mill-hammer, which occurred in the process of milling the asbestos, introduced increased concentrations of trace metals (nickel, chromium and cobalt). Metallic contamination was thought to be responsible, but subsequent National Cancer Institute (NCI) studies by Stanton have tended to reinforce the importance of fiber length, regardless of chemical composition. (However, also see Stanton hypothesis, questioned, below.) Epidemiologically, a 1973 observation by Hammond and Selikoff[20] (i.e., that exposure to asbestos dust did not appear to increase the risk of bronchogenic cancer in insulation workers the absence of regular cigarette smoking) was responsible for the suggestion by George Wright[5] that the role of asbestos might be restricted to that of a cocarcinogen. However, the validity of the Hammond and Selikoff data has been disputed in 1980 by Demopoulos[21] (Table 6). Meanwhile, at a clinical symposium on Asbestos and Lung Cancer, the relative risk of asbestos-related lung cancer was shown to be about equal, using the same insulation workers' data for smokers and non-smokers (Table 7); e.g., 4.9 vs 4.4, respectively.[22] According to Enterline, when lung cancer was a rare disease, asbestos apparently acted simply to multiply the risk by some factor, e.g., 4, but a four-fold increase in a rare disease will still be rare. So, we did not see much asbestos-related lung cancer in asbestos workers until lung cancer became fairly common.[22]

Table 6

Lung Cancer Mortality in Asbestos Tradesmen[1]
(Modified from Demoponlos[21])

	Expected by National Average	Expected by Estimating Variables	Observed[1]
Lung Cancers (non-mesothelioma)	106	406[2]	485
Non-Pulmonary	104		340 (169 mesotheliomas excluded)

[1]The total observed in approximately 18,000 asbestos workers over 10 years was 994, according to a report by Selikoff which considered only adjustments for age and sex; the data was not controlled for smoking; 169 mesotheliomas were directly caused by high level exposure to asbestos. The question addressed in this analysis is whether asbestos is a carcinogen for other anatomic areas, particularly the bronchi and bronchioles; consequently, the lung cancers were evaluated independently of the other anatomic locations.

[2]Calculations and estimations as follows: (1) Approximately 75% of involved tradesmen smoke cigarettes--this increases the expected number of lung cancer deaths to about 160, instead of 106 since 50% of adult males smoked in the national average 10-20 years ago. (2) Most of the involved tradesmen are in urban centers--there is a striking difference between urban/rural lung cancer rates among smokers. It amounts to approximately a 90% greater risk. If the urban/rural distribution of the national population was about 50/50 in the 1950s then applying just one half of the urban correction, i.e., 45% greater, increases the expected 160 cases of cigarette induced lung cancers by 45% and yields an expected number of 232 (160 x 45% + 160 = 232) among cigarette smoking tradesmen in urban areas. (3) Involved tradesmen smoke approximately 30% more cigarettes than the national average of one pack/day. This increases the lung cancer mortality ratio from 8.62 to 14.69, an increase of about 75%, which will cause 174 more cancers (232 expected so far x 75% = 174), yielding an expected 406 (232 + 174). Values for the urban lung cancer rates and the dose-response relationship were obtained from Persons At High Risk Of Cancer, J.F. Fraumeni (Editor), Academic Press, New York, 1975, p. 345 and p. 134, respectively. Guidance in estimating cigarette consumption in the construction industry, which includes shipyard and insulation workers, was obtained from: a) R.R. Williams, N.L. Stegens, and J.R. Goldsmith, Associations of cancer site and type with occupation and industry from the Third National Cancer Survey Interview, J.N.C.I. 59:1147-1185, 1977; b) N.E. Breslow and J.E. Enstrom, Geographic correlations between cancer mortality rates and alcohol-tobacco consumption in the United States. J.N.C.I. 53:631-639, 1974; and c) cited references. This estimation of what the expected lung cancer mortality might be is an exercise in demonstrating how different the potential interpretations could be, depending on whether the controls include the major variables or not. The thought that asbestos exposure causes a very striking synergism of cigarette induced lung cancer is based largely on comparisons with national averages. Comparison with well matched controls might demonstrate a far lower degree of synergism.

Table 7

Lung Cancer Death R. 's by Smoking History
(Rates per 100,000 Per Year)
(Modified from Enterline[22])

Cigarette Smoking	Asbestos Insulators	U.S. Males	Relative Risk
Yes	362.0	74.4	4.9
No	40.4	9.2	4.4

A symposium was held in 1980 to discuss attributability of lung cancer to asbestos exposure in cigarette smokers.[22-25]

Regarding chances in an individual who smoked that lung cancer was caused by occupational exposure, Enterline arrived at a 97.5% probability that either smoking or asbestos exposure was responsible, as follows:

> In calculating the probability (.794) that an asbestos worker with lung cancer who smoked had the disease as the result of asbestos exposure, the traditional public health approach uses the concept of a prior or predisposing condition which, if removed, would have prevented the disease. Clearly, smoking magnified the effects of asbestos and for the same worker there is the probability (.888) that his lung cancer was due to smoking. Moreover, it can be calculated as before but with the relative risk (from Table 7) as the lung cancer death rates among asbestos insulators who smoke and U.S. males who do not:
>
> $$RR = \frac{362.0}{9.2} = 39.4$$
>
> Using this ratio produces the desired probability (i.e., 0.975).
>
> Using another approach, and if relative risks were actually independent of smoking and asbestos exposure, this can be calculated in a more conventional way:
>
> $$P(S \text{ or } A) = P(S) + P(A) - P(S \text{ and } A)$$
>
> While this requires an estimate of relative risks under conditions of independence, substituting the

probabilities of 0.794 and 0.888 gives a good approximation:

$$P = 0.794 + 0.888 - (0.794)(0.888)$$

$$= 0.794 + 0.888 - .705$$

$$= 0.977$$

The second method is informative in that the degree of interaction between asbestos exposure and smoking becomes more clearly apparent—i.e., for an asbestos worker with lung cancer who smoked cigarettes, the probability that both asbestos and cigarettes played a role is 0.705, or 70.5%.

The objective of the above analysis of probabilities was to review epidemiologic evidence. It does not provide a basis for the clinician to be definite in a given case; it merely permits us to recognize apparent trends on which to base attempts at prevention (see Table 8).

Table 8

Multiplicative Effects of Cigarette Smoking and Uncontrolled Asbestos Exposure at Work (Modified from Enterline[22])

Nonsmoker:	Relative Risk	Smoker:	Relative Risk
Asbestos Worker	5	No Asbestos Work	10
No Asbestos Work	1	Asbestos Worker	50

As pointed out in the above mentioned 1980 Symposium on Asbestos and Lung Cancer,[23] there is an analogy to the buildup of evidence relating cigarettes to lung cancer which led the Royal College of Physicians to comment in 1971: "...objections to the conclusion that cigarette smoking is a cause of lung cancer...are...without substance." Their first such report issued a decade earlier, based upon a review of 216 reports, had stated: "...none of the facts contradict the conclusion that cigarette smoking is an important cause of lung cancer." In the U.S., suspicions had been raised concerning such an etiological association as early as 1941, based on clinical (thoracic surgical) epidemiological observations of apparent clustering. Though not mentioned in 1948 in an editorial in the JAMA entitled The Advertising of Cigarettes, it was again implicated in 1959 by the USPHS Surgeon General, Dr L.E. Burney, as being "...the principal etiologic factor." Subsequently, in 1964 and based upon a review of 1011 scientific papers, a report of an advisory committee to the USPHS Surgeon General went one step further, cautiously suggesting that cigarette smoking was causally related to lung cancer.

The Symposium's chairman, Dr Margaret R. Becklake, in view of the above caution, asked: "Who would be prepared to answer the legal question.

In which year, Doctor, was it known that smoking causes lung cancer?"[23] Obviously, the same held true--even to a greater degree--with asbestos and lung cancer.[24] The situation was summarized as follows in 1982 in a special issue of the AMA Archives of Pathology and Laboratory Medicine on the subject of Asbestos Associated Diseases: "The relationship of the number of asbestos bodies and uncoated fibers in the lungs with the pathogenesis of (lung cancer) has not been resolved. The greater the cumulative exposure to asbestos, the higher the risk of lung cancer developing. All types of asbestos seem to be carcinogenic, however, the relative carcinogenicity of each type of asbestos is not defined and remains a matter of controversy."[2]

Speaking on clinical decision-making regarding the role of asbestos in causing lung cancer in individual cases, Dr Hans Weill stated: "The highest probability of such association will be in workers having histologic and radiographic evidence of asbestosis, characterized by diffuse interstitial pulmonary fibrosis. While this issue has not been fully resolved, there is some evidence that a carcinogenic dose of asbestos exposure will also be fibrogenic, producing evidence of lung and/or pleural scarring. Since it is unlikely that agreement can be reached soon concerning the minimum required time and concentration of exposure which in the presence of bronchogenic cancer makes it an asbestos-attributable tumor, the current procedure is to require radiographic and/or histologic evidence of asbestosis in association with the lung cancer."[25] The presence of asbestosis thus serves as an indicator of degree of total past hazardous asbestos exposure, for purposes of determining its adequacy in production of another more serious pathologic condition: lung cancer.

VII. MALIGNANT PLEURAL MESOTHELIOMAS

Described more than a century ago and noted to be associated with asbestos exposure 37 years ago--malignant pleural mesothelioma (also see Pleural Pathology above) was considered a rarity until 1960 when 33 cases were linked to the crocidolite type of amphibole asbestos exposure in the northwest Cape of South Africa.[3] By 1975, 4,539 mesotheliomas reported in asbestos workers worldwide (the vast majority were in workers who had been exposed to crocidolite alone, or combined with other forms of asbestos) constituted 6.1% of deaths from all causes (range 2.42% to 16.07%). By comparison, where only chrysotile was used, mesothelioma deaths made up only 0.3% (range 0.24 to 0.87). An even greater contrast is found in anthopyllite workers in Finland where a study of 216 deaths among 900 miners and millers did not reveal any mesothelioma cases from of this form of amphibole asbestos. Nationally, mesotheliomas were the cause of death in nonasbestos exposed persons, at a rate of 7 per million for males and 2 per million for females, according to cancer registry data from the early 1970s. A detailed study of 188 cases diagnosed in Los Angeles between 1972 and 1979, where the incidence in each sex was close to the estimated national rate, suggests that about 70-80% of male and 10-20% of female patients had been specifically exposed to asbestos. If applied nationally, it follows that some 500-600 mesotheliomas were caused by asbestos each year between 1970 and 1975.

According to Doll and Peto, if alarming predictions in the extremely controversial "Estimates of the Fraction of Cancer in the U.S. Related to

Occupational Factors" (OSHA, 15 Sep 78) should materialize with 350,000-575,000 mesotheliomas to be expected from asbestos exposures, at least 10,000 mesotheliomas cases per year should be occurring now instead of the 500-1,000 cases found. So there is more than a 10-fold shortfall at a time when we should be experiencing peak rates.[26]

Because the Borough of Manhattan in New York City had been found to have the highest urban asbestos air pollution levels (Table 9), the New York University (NYU) Coordinated Investigative Tumor Registry reviewed the NYU Medical Center experience with mesotheliomas over a 10-year period, from 1967-76, in search of a trend. In comparison with the National Cancer Institute's Third National Cancer Survey which found 154 pleural/peritoneal mesotheliomas among 163,000 cancers cataloged (a ratio of 1:1000), the rate at NYU was 2.6:3000/year for 10 years or 0.86:1000 cases/year. No upward trend in annual admissions for mesotheliomas was found, despite a relatively high risk urban population served, from the standpoint of asbestos exposure (Table 10).[21]

Table 9

Chrysotile Content of Ambient Air in New York City by Borough (Modified from Demopoulos[21])

Sampling Locations	Number of Samples	Asbestos air level in 10^{-9} g/m^3 Range	Average
Manhattan	7	8-65	30
Brooklyn	3	6-39	19
Bronx	4	2-25	12
Queens	4	3-18	9
Staten Island	4	5-14	8

Table 10

Mesotheliomas at the New York University Medical Center (1967-1976)[1]
(Modified from Demopoulos[21])

	Probable or Compatible[1]	Definite	Total
1967	0	3	3
1968	1	1	2
1969	0	3	3
1970	2	2	4
1971	0	1	1
1972	0	2	2
1973	0	1	1
1974	1	2	3
1975	2	2	4
1976	2	1	3
TOTALS	8	18	26

[1]NYU Medical Center is a general, acute care institution and admits 3,000 new cancer cases per year, including those from a prime affiliate in Queens, Booth Memorial Hospital. As a general facility, it "sees" a representative sampling of initially diagnosed mesotheliomas, in contrast to special mesothelioma-referral facilities that receive many cases after initial diagnosis elsewhere.

The well known difficulty in establishing the diagnosis is reflected in the need to categorize some as only probable.

Complicating the matter of asbestos related etiology, per se, is the reported widespread occurrence of mesothelioma (with an incidence rate of 2.30%) in the village of Karain, Turkey, the population of which has remained about 600 for almost 1000 years because of the incredible incidence of this chest disease.[27] The name of the town means pain in the midriff. Environmentally; the only difference between Karain and a similar village nearby is in the volcanic rock or tuff. At Karain the tuff contains a large number of amphibole-like, long fibers, 75% of which are less than 0.25 μ in diameter. By analysis, however, the fibers appear to be zeolite materials, with composition most like erionite.[28]

Most published cases of malignant mesotheliomas reportedly have come from seaports with shipyards.[1] Incidence rate was equal to matched controls for black males and black and white females. There was 4 times the nationally recorded rate for white males among whom (to judge by the control group) 28%

at the ages studies had been employed in shipbuilding.[16] Insulation workers were said by Selikoff and Seidman to be at over 10 times that risk, i.e., 46 times matched controls.[3] Their data has been reviewed critically by Doll and Peto, however, who commented that 68% of their mesotheliomas were miscertified.[16]. Other occupations with recognized mesothelioma hazard, including plumbing and heating trades (exclusive of insulation), asbestos production, manufacture and construction as well as shipyard work, are said to have relative risks ranging from 3-6X controls.[3] (Enterline has also called attention to the fact that Selikoff's estimate of cancer deaths caused by occupational exposure to asbestos is 10 times higher than the approximately 1.0% estimated by all other observers).[14] It is important to recognize that the alarming predictions, in the above mentioned U.S. government report of 1978, also included unsupported estimates over an order of magnitude in excess of all other existing scientific knowledge according to Enterline[14] (Table 5 and Fig 4). Despite the efforts of the U.S. Congress to restore scientific objectivity and accuracy,[14] it has been predicted that factual arguments will not be accepted by people who wish to emphasize the importance of occupational factors.[22] Nevertheless, these facts, which have been discussed widely in scientific meetings without dissent, should be known to all occupational health personnel having responsibility for advising Air Force workers, unions and supervisors concerning true relative risks, e.g., in job placement, hazard control and environmental differential pay.

The ability of asbestos to affect adversely individuals exposed nonoccupationally over a long period of time has also been cited, e.g., the example of a the shipyard pipe insulator who brought his work clothes home for cleaning. He died of bronchogenic adenocarcinoma, but his wife and daughter both died later of malignant mesotheliomas.[3] The fact that such clustering is not reported more frequently, despite the widespread public exposure to asbestos (as shown in Tables 2 and 4), points to a complex etiology. Unusual immunologic reactions have been postulated.[3] Peto, et al., however, have suggested that, rather than progressive generalized changes in immune function, there may be a relatively constant incidence of genetic or epigenetic accidents. This theory is based upon two observations: 1) that mesothelioma incidence is dependent upon time since first exposure, but is unrelated to age at first exposure and 2) that the incidence pattern over time is identical for pleural and peritoneal tumors.[29]

Mesothelioma incidence is unrelated to smoking. Among nonsmokers, it may exceed the increase in lung cancer risk, without respect to age at first exposure to asbestos. The ratio of asbestos-induced lung cancer (observed minus expected) to mesotheliomas was 3.9 among smokers first exposed to asbestos at age 25 or over (1.8 below age 25) and 0.2 among nonsmokers. "The ratio could change at much lower asbestos dust levels," Peto comments, "but it seems reasonable, in the absence of direct evidence of such, to assume that the effect is simply proportional to dust level for both diseases, and that their ratio will depend upon age at first exposure in the same way for nonoccupational exposure. The qualitative conclusion that asbestos acts chiefly at an early stage in mesothelioma induction but affects a later stage or stages for lung cancer seems reasonably secure...."[29]

Interestingly, Bignon reports that the risk of developing mesothelioma is substantially lower in humans whose exposure to chrysotile is reduced or stopped than in those whose exposure continues.[10] By contrast, even brief

exposure to crocidolite produces a substantial incidence of mesothelioma many years later. This difference is consistent with the facts (1) that chrysotile fibers are largely eliminated from the lungs whereas amphiboles remain almost indefinitely and (2) that chrysotile fibers which remain become leached in vivo, and thus cease to be biologically active in the body, while crocidolite fibers remain active (or even become more active, based on observed increased cytotoxicity in vitro with leaching). The striking modification in the biological response of acid-treated asbestos suggests that reactive sites at the surface of the fibers (i.e., as well as fiber length, per se) could play a role in determining the pathogenic effects of fibers. A difference in survival time between chrysotile and crocidolite in rats injected intrapleurally suggests that chrysotile is immediately reactive in inducing inflammation and subsequently cancer, whereas crocidolite needs in vivo modification to become inflammatory and carcinogenic. These in vitro and in vivo research findings (also discussed above under Asbestosis) cast doubt upon the fiber length alone, i.e., the Stanton hypothesis.[30] They provide the basis for more and better research studies to help understand the true pathogenicity of various types of fibers, including those grouped under the generic term: asbestos.

Practically speaking, according to Peto, et al., long fine fibers are particularly liable to cause mesotheliomas, whether or not they are asbestos. Chemical differences appear less important, e.g., airborne chrysotile fibers in a factory environment may be considerably finer than those in a mine, and the incidence of mesotheliomas at a given nominal fiber count appears quite low among miners. Peritoneal mesotheliomas are common among amosite workers and their being rare or absent among chrysotile miners may reflect physical fiber differences--just as in the case of the peritoneal-pleural mesothelioma ratio difference between Australian crocidolite miners (0:26) and crocidolite gas-mask workers in Canada (6:3). It may, therefore, be an error to attribute the substantial incidence of pleural mesothelioma among chrysotile factory workers to occasional crocidolite exposure, merely because mesothelioma is rare among chrysotile miners.[29]

Parenthetically, the overall excess of lung cancer is also relatively low among chrysotile miners. The only safe conclusion must be that dose response relationships cannot be expected to apply outside of the environment in which they are established, at least until the range of fiber sizes to be included in the fiber count has been chosen less arbitrarily. A single universal standard is liable to be too stringent for certain working conditions and dangerously high for others.[29]

Basis for the Existing Consensus Standard - According to the TLVs-ACGIH's Documentation of TLVs,[31] the only reliable exposure data upon which to base a threshold limit come from England. There, it has been observed that asbestos textile workers with less than 50 fiber years of exposure (e.g., 1 fiber per cc for 50 years $\cong$ 2 fibers per cc for 25 years) would cause development of asbestosis (manifested by persistent high-pitched basilar rales heard in the lungs) in no more than 1% of exposed workers. This departure from the previous 1938 dust standard of 5 mppcf (see Asbestosis above) was in recognition: 1) that there was marked variability of the asbestos fiber content in factory dust and 2) that the disease was related to the number of asbestos fibers inhaled and not to the amount of nonfibrous dust particles. Also, it was specified that the counted fibers were to be longer than 5 μ--partly because it is

not practical to attempt to count shorter fibers with an optical microscope (400 to 450 x magnification under phase contrast illumination with a 4 mm objective). It is recognized that for every asbestos fiber longer than 5 μ, there may be as many as 100 or more shorter and thinner that are not visible under the optical microscope. However, experimental evidence has shown the fibers shorter than 5 μ to be nonfibrogenic (see Asbestosis above).

A challenge to the ACGIH TLV of 2 fibers/cc was raised when, in 1976, a study of 440 hard rock gold miners, who had been exposed to an asbestiform mineral, cummingtonite-grunerite, found 10 respiratory cancer deaths (including a carcinoma of the maxillary sinus and a mediastinal carcinoma), whereas only 2.74 has been expected. While the diagnosis asbestosis was not mentioned, the authors emphasized the findings of an average of 4.82 fibers/cc, 80% of which were fibrous amphiboles, and 60-70% of the latter were fibrous grunerite (i.e., amosite). Fibers longer than 5 μ averaged 0.36 fibers/cc and about 94% of the airborne fibers average 0.13 μ in diameter by 1.1 μ in length. Based on these observations, the authors questioned adequacy of OSHA's PEL for asbestos, i.e., 2 fibers longer than 5 μ/cc.

Another investigator did a more extensive study of the same gold mine. Using a larger cohort consisting of 1321 men who had completed 21 years service with the mining company. Results (see Table 11), which showed no excess of observed over expected respiratory cancers, failed to support the claims of the earlier study concerning inadequacy of the 2 fiber longer than 5 μ/cc OSHA standard. The excesses of pneumoconiosis and silicotuberculosis shown reflected the serious hazard from silicosis (due to hard rock dust, containing 13% free silica)—not cancer.

Table 11

Tabulation of Significant Findings in Two Epidemiologic Investigations of Workers in the Same Hard Rock Gold Mine (Modified from Documentation of TLVs[31])

		McDonald et al.		Gillam et al.	
Size of Cohort		1321		440	
Number of Deaths		Observed	Expected	Observed	Expected
Respiratory Cancer		17	16.5	10	2.74
Nonmalignant	Pneumoconiosis	39*	0	---	---
Respiratory	Respiratory TBC Silico-TBC	39	3.6	5	1.85
	Other Nonmalignant Respiratory Disease	?	?	---	---

*37 of the 39 deaths were ascribed to silicosis

According to the ACGIH TLV committee, workers in the asbestos textile factory, from which the British standard of 2 fibers/cc was derived, have been studied continuously for 30 years with reports published in 1955, 1965, 1968 and 1977. They represent the only cohort of asbestos workers in the world in which health effects have been correlated with definite exposure data defined as fibers per cc. It would be premature and ill-advised, they feel, to change the present consensus standard of 2 fibers longer than 5 μ/cc for chrysotile without indications from this study population of the advisability for such change.[31] The same TLV is assigned to other forms of asbestos not specifically named by ACGIH (i.e., other than crocidolite and amosite mentioned below). The most widely used amphiboles (crocidolite and amosite) appear to have greater potential hazard than chrysotile and have been awarded lower TLVs, as follows:

amosite 0.5 fiber > 5 μ/cc

crocidolite 0.2 fiber > 5 μ/cc

Despite the existence of the above consensus standards, a NIOSH/OSHA Asbestos work group in 1980 concluded: 1) that there are no data to support existence of a no-effect threshold (virtually all levels of asbestos exposure studies to date demonstrated an excess of asbestos related disease) and 2) on the basis of available information, there is no scientific basis for differentiating between asbestos types for regulatory purposes. Accordingly, the single standard recommended to be applied to all asbestos fiber types, was to be 0.1 fibers > 5 μ/cc TWA, representing the sensitivity level of the most widely

used authorized industrial hygiene method of environmental quantitation. As stated previously, the intent appeared to be to require biological monitoring whenever asbestos exposure of any degree could be detected by existing methods. Furthermore, a change in the 1972 PEL of 2 fibers to 0.1 was recommended as an Emergency Temporary Standard. OSHA responded on 4 Nov 83 by announcing an Emergency Temporary Standard of 0.5 fibers > 5 μ/cc, expressing its intention that it serve also as a proposal to revise the current asbestos standard. The Asbestos Information Association (AIA) objected that the emergency rule making procedure had denied AIA participation in the standards setting process, despite its attempts for the past two years to initiate rule making to develop an improved standard. In filing suit against the emergency standard, asbestos manufacturers claimed it to be a "precipitous action based on the unfounded conclusion that a health emergency existed under the 2 fiber standard."[33]

VIII. CONCLUSIONS AND RECOMMENDATIONS

Military Applications - Generally, the action level, at which biological monitoring of exposed workers becomes necessary, is one-half the TLV. However, in the case of asbestos according to AFOSH Standard 161-4, dated 20 June 1977, preplacement (baseline) and annual medical examinations are mandatory for Air Force workers exposed (8-hour TWA) to 0.1 fibers/cc of asbestos determined by the USAF OEHL, using phase contrast microscopy with 400-450 x magnification (4 mm objective). (As pointed out above, fibers shorter than 5 μ are not detected by this method.) A termination medical examination performed within the year prior to release is provided for any employee previously included in the periodic surveillance program. All such records are maintained at least 40 years after termination of employment.

As noted above (under Pathology), the 1982 DoD Occuptional Health Surveillance Manual incorporated an action level which had been used by the Navy, i.e., 0.5 fibers/cc for any length of time on a regular basis (i.e., 15 days in any one quarter or 45 days in any one calendar year).[34] In order to reconcile DoD guidance with that contained in OSHA, the 1983 modification has been changed to reflect the action level as being 0.1 fiber/cc for an 8-hour TWA on a regular basis (Table 3).[35] The absolute ceiling TLV of 10 fibers > 5 μ/cc (for any period of time with or without proper protection) remains unchanged as a basis for inclusion in the periodic surveillance program.

Air Force Status - In view of existing knowledge and standards, the health of Air Force workers with known potential asbestos exposure hazard has been well protected with environmental and, where indicated, biological documentation. Despite this, claims for asbestos-related diseases are expected to originate among former Air Force civilian employees who have worked in jobs in which asbestos exposures may have occurred, e.g., plumbers and maintenance workers.

A leading pulmonary specialist, Dr Hans Weill, speaking in 1980 on the question of attributability of respiratory malignancy in workers or former said:

> Pleural or peritoneal mesothelioma, diagnosed by one or more pathologists expert in the difficult recognition of these

> tumors, in an individual or in his family, should be considered work-related and appropriately compensated without further proof of a causal association.
>
> Bronchogenic carcinoma is another matter...and an attempt must be made to assess quantitatively the cumulative burden of asbestos fiber exposure in these cases so that a probability of attributability can be estimated....If the relative risk is similar for nonsmokers and smokers (although the excess cases are far greater among smokers) one can clearly disregard smoking in attempting to determine the role of asbestos exposure in a given case. We are still left with the burden of assessing asbestos dose. Since it is rarely possible to construct an estimate of past individual exposures...considerable weight must be placed on the presence and extent of another dose-related asbestos health effect, namely pulmonary fibrosis or asbestosis. As pointed out by Becklake, however, Compensation Boards must act, today and now. Some accept attributability in the absence of fibrosis; some do not.[23]

Another asbestos-related problem requiring clinical decision-making is: "What action is to be taken in the case of the currently employed asbestos-exposed worker with early but definite fibrogenic effects on the lungs and/or pleural surfaces?" As shown in Table 12, the greater the past cumulative dust exposure, the higher the probability of progressive pulmonary fibrosis, Dr Weill advises against further exposure by changing such workers to a job where measurable asbestos exposure does not exist. This should minimize excessive risk of lung cancer development.[25]

Table 12

P-Values for Relationships Between Progression of Radiographic Abnormalities and Possible Influencing Variables (Modified from Weill[25])

	Progression of		
	Irregular Small Opacities	Pleural Thickening	Pleural Calcification
Age	NS	NS	NS
Smoking	NS	NS	NS
Length of Exposure	NS	.02	.03
Average Exposure	.0001	NS	NS
Cumulative Exposure	.0002	NS	NS

NS = Not significant, p >.10

Pleural thickening and plaques, as shown in Table 12, have been shown to be related to length of exposure and time since first exposure but not to cumulative dust dose. Thus, their progress is not likely to be influenced

by modest additional average or cumulative exposure. Hence, Dr Weil recommends continued monitoring of respiratory health, but that job transfer is not necessary; however, he points out that the worker, who should be informed concerning the pleural effects of asbestos exposure, may subsequently elect to change to a job without further asbestos exposure.[35]

In contrast to other employers lacking fully developed preventive industrial hygiene programs, the Air Force should have no unknown, unprotected asbestos-hazardous jobs. Where this has been documented by careful bioenvironmental engineering surveys and compliance with OSHA standards of control, the decision not to authorize environmental differential pay has been upheld.[36]

References

1. Melvin, W.W., Asbestos: A Brief Review of the Epidemiology and Pathology, USAF Environmental Health Laboratory, Kelly AFB TX (1976).

2. Craighead, John E. et al., Asbestos - Associated Disorders (Special Issue) A.M.A., Archives of Pathology and Laboratory Medicine, 106:544-597 (1982).

3. Fontana, Robert S. et al., A National Correspondence Course on Lung Cancer and Asbestos-Related Pulmonary Disease, Co-sponsored by the Americ an College of Chest Physicians and the National Cancer Institute, Bethesda MD (1981).

4. Hatch, T.F. and Gross, P., (ALHA and USAEC Monograph), Pulmonary Deposition and Retention, Academic Press, New York, pp 31-32, 54 (1964).

5. Wright, George W., (Chapter 7), The Pulmonary Effects of Inhaled Inorganic Dust In: Patty Industrial Hygiene and Toxicology, Third Revised Edition, Vol I - General Principles, George D. Clayton and Florence E. Clayton, Editors, John Wiley and Sons, New York, p 194-Asbestos, (1978).

6. Hunter, Donald, The Diseases of Occupations, 6th Ed., Hodder and Stoughton, London, pp 997-998 (1978).

7. Gross, P.; R.T.P. DeTreville; L.J. Cralley and J.M.G. Davis, Pulmonary Ferruginous Bodies: 1. Their Development in Response to Filamentous Dusts; 2. A Method of Isolating and Concentrating Them, Arch. Path., 85:539 (1968).

8. Davis, John M.G.; Robert T.P. DeTreville and Paul Gross, Asbestos Bodies and Bioeffects: A Detective Story, 32nd Annual Meeting, Transactions of the Industrial Hygiene Foundation of America Inc., Mellon Institute, Pittsburgh, p 83 (1967).

9. Davis, John M.G., Pathological Studies of Ferruginous Bodies Basic Considerations in Med. Series Bull. No. 16-70: Fibrous Dust Seminar Proceedings, Industrial Hygiene Foundation of America Inc., Pittsburgh, pp 69-75 (1970).

10. Haller, Martin N., "Pathological Studies of Ferruginous Bodies Instrumentation," in Med. Series Bull. No. 16-70: Fibrous Dust Seminar Proceedings, Industrial Hygiene Foundation of America Inc., Pittsburgh, pp 69-75 (1970).

11. Goodman, J.L., "Asbestos Exposure in the U.S. Textile Industry, 1930-date," in Med. Series Bull. No. 16-70: Fibrous Dust Seminar Proceedings, Industrial Hygiene Foundation of America Inc., Pittsburgh, pp 69-75 (1970).

12. Gross, P; R.T.P. DeTreville and W.W. Haller, Pulmonary Ferruginous Bodies in City Dwellers, A Study of Their Central Fiber, Arch. of Environ. Health, 19:186-188 (1969).

13. Gross, P; Jiri Tuma; R.T.P. DeTreville, Fibrous Dust Particles and Ferruginous Bodies Methods for Quantitating Them and Some Results from the Lungs of City Dwellers, Arch. of Environ. Health, 21:38-46 (1970).

14. Enterline, Philip E., Cancer Produced by Non-Occupational Asbestos Exposure in the U.S., J. Air Pollution Control Assoc., 33:318-22 (1983).

15. Johnstone, R.T., Chapter 14, Asbestosis in Occupational Medicine and Industrial Hygiene, C.V. Mosby Co, St Louis MO (1948).

16. Doll, R., Mortality from Lung Cancer Among Asbestos Workers, Brit J. Ind. Med., 12:81 (1955).

17. Braun, D.C. and T.D. Truan, An Epidemiological Study of Lung Cancer in Asbestos Miners, AMA Archives of Ind. Health, 17:634 (1958).

18. McDonald, J.C., et al., Mortality in the Chrysotile Asbestos Mines and Mills of Quebec, Arch. Environ. Health, 22:677 (1971).

19. Gross, P., R.T. DeTreville, E.B. Tolker, Marianne Kaschak and Mary Ann Babyak, Experimental Asbestosis, Arch. Env. Health, 15:343 (1967).

20. Hammond, E.C. and I.J. Selikoff, Relation of Cigarette Smoking to Risk of Death of Asbestos - Associated Disease Among Insulation Workers in the U.S., In Biological Effects of Asbestos: Proceedings of a Working Conference held at the International Agency for Research on Cancer, Lyon, France, Oct 2-6, pp 312-317 (1972).

21. Demopoulos, Harry B., NY University Medical Center, Co-Sponsored by the NYU Medical Center and the American Cancer Society, In Demopoulos, H.B. and M.A. Melilinam, Editors, Cancer and the Environment: Summary of Conference: Major Discussion Points and Societal Interrelationships, Pathtox Publishers Inc., 2405 Bond St, Park Forrest South IL, pp 473-481 (1980).

22. Enterline, P.E., Attributability in the Face of Uncertainty, Chest, 78:377-9.

23. Becklake, M.R., Asbestos and Lung Cancer: The Clinician's Questions Chest 78:372-3 (Suppl) (1980).

24. McDonald, J.C., Asbestos and Lung Cancer: Has the Case Been Proven? Chest, 78:380-1 (1980).

25. Weill, H., Asbestos and Lung Cancer: Basis for Clinical Decision Making, Chest 78:382-3 (1980).

26. Doll, R. and R. Peto, The Causes of Cancer: Quantitative Estimates of Available Risks of Cancer in The U.S. Today, The OSHA Paper (NCI/NIEHS. NIOSH) of 1978, pp 1240-1245, Appendix F: Examination of the Arguments and Conclusions in Estimates of the Fraction of Cancer in the U.S. Related to Occupational Factors, pp 1305-1308 (OSHA, Sept 15, 1978).

27. Wagner, J.C., The Complexities in the Evaluation of Epidemiologic Data of Fiber-Exposed Populations, In: R. Lemen and J.M. Dement - Editors, Dust and Disease, Proceedings of a Conference on Occupational Exposures to Fibrous and Particulate Dust and Their Extension into the Environment, Pathotox Publishers Inc., pp 37-39 (1979).

28. Pooley, F. D., Evaluation of Fiber Samples Taken from the Vicinity of Two Villages in Turkey, Dust and Disease, Proceedings of a Conference on Occupational Exposure to Fibrous and Particulate Dust and Their Extension into the Environment, Pathotox Publishers Inc., pp 41-44 (1979).

29. Peto, J., Seidman, H. and Selikoff, I.J., Mesothelioma Mortality in Asbestos Workers: Implications for Models of Carcinogenesis and Risk Assessment, Br. J. Cancer, 45:124-135 (1982).

30. Bignon, J. and M.C. Jaurand., Biological in Vitro and in Vivo Responses of Chrysotile Versus Amphiboles, Environmental Health Perspectives, 51:73-80 (1983).

31. Documentation of TLVs, 4th Ed., ACGIH, Cincinnati OH (1980).

32. Workplace Exposure to Asbestos: Review and Recommendations, DHHS (NIOSH) Publication No. 81-103 (Nov 80).

33. International Environmental Reporter: Current Report, p 533 (14 Dec 83).

34. DoD 6055.5-M, "Occupational Health Surveillance" (Jul 82).

35. NavMed Cominst 6260.3, "Occupational Health Medical Surveillance," MedCom-24 (29 Aug 83).

36. Lurker, Peter A., USAF OEHL/ECH, Personal Communication (Nov 83).

Appendix I

Testimony in Environmental Differential Pay Dispute (1981)

Capt Peter A. Lurker

Appendix I

Testimony in Environmental Differential Pay Dispute (1981)

Capt Peter A. Lurker

Capt Lurker is of lawful age, being duly cautioned and solemnly sworn as hereinafter certified, was examined and testified as follows:

Direct Examination (by Maj Schlabs, assigned by USAF Staff Judge Advocate)

Q. State and spell name for the record:
A. Peter A. Lurker

Q. What is your duty title and station?
A. Consultant, Industrial Hygiene Engineer, USAF Occupational and Environmental Health Laboratory, (USAF OEHL), Brooks AFB TX.

NOTE: All answers were directed to the Arbitrator, regardless of the source of questions.

Q. Explain your responsibilities as Consultant, Industrial Hygiene Engineer.
A. I perform surveys and contract monitoring Air Force-wide and make recommendations based on standard practices, independent of management. I am also co-author of several AFOSH standards, including one on workplace monitoring (now under review by HQ USAF prior to publication).

Q. What is your educational background in your special field of Industrial Hygiene Engineering?
A. I earned a B.S. in Chemical Engineering in 1971 at the New Jersey Institute of Technology and a M.S. also in Chemical Engineering in 1973 from the University of Pennsylvania; then a PhD in Environmental Health from the University of Cincinnati's Kettering Institute in 1980 (under Dr Eula Bingham who was the Institute's Associate Director).

Q. Are you a member of any professional organizations?
A. I am certified in Industrial Hygiene by the American Board of Industrial Hygiene and am a member of the American Industrial Hygiene Association as well as the American Conference of Governmental Industrial Hygienists.

Q. Please list your Air Force experience.
A. Computer officer for one year (Air Force line officer). I was Bioenvironmental Engineer at the Air Force Aerospace Medical Research Laboratory (HQ AFAMRL/THD) for two and one-half years during which I conducted environmental degradation studies on toxic chemicals unique to Air Force use. Then I served as base Bioenvironmental Engineer at Kunsan AB, Korea, for a year, following which I spent three years in the doctoral program at the Kettering Institute as an Air Force fellow, receiving the PhD degree in Environmental Health in 1980. Subsequently, I was assigned to my present

job in the USAF OEHL Consultant Services Division. In July 1980, I became certified in Industrial Hygiene by the American Board of Industrial Hygiene.

Q. What experience have you had in industrial hygiene?
A. I have conducted 15 field surveys for USAF OEHL involving measurements of industrial hazards such as carbon monoxide, ozone, chlordane, asbestos, carbon, composite carbon fibers, styrene and methyl ethyl ketone peroxide.

Q. Indicate to the Arbitrator your particular experience with asbestos.
A. I conducted the field study at Fairchild AFB WA on the central heating plant. Included were both the heating plant personnel and the steam pipe fitters who work in steam tunnels and steam pits. Also, during my three years at the University of Cincinnati, I assisted in performing both sampling and analysis for asbestos in support of ongoing laboratory studies.

Q. What health problems have been related to asbestos dust exposure hazards?
A. Asbestosis, lung cancer and mesothelioma.

Q. How does asbestos cause all of these problems?
A. Generally, through inhalation of fibers generated in the work place, although some 15% of all mesotheliomas are thought to result from nonoccupational exposure to asbestos or other fibers of respirable size.

Q. When did asbestos become widely publicized as a possible health hazard?
A. Just prior to World War II--about 1939--in the industrial setting in the United Kingdom, but not until after World War II in the United States. In 1968 the British Occupational Health Society published an epidemiological study of 240 asbestos workers relating worker's exposure to health status and providing the best data available on which to base the current estimate of occupational hazard control requirements published by the American Conference of Governmental Industrial Hygienists.

Q. Describe to the Arbitrator the development of the Occupational Safety and Health Act (OSHA) and the intent of the OSHA standard for asbestos.
A. The OSHA Act of 1970 created an administrative agency (also called OSHA) within the Department of Labor (DOL). Its purpose was to promulgate and enforce standards to protect all persons from hazards at work so that no worker would have to pay with his life or health to make a living.

Q. How does OSHA do this?
A. OSHA publishes a proposed standard in the Federal Register and announces that a public hearing will be held, usually within six months, at which any interested party may attend and present evidence to aid the standards setting process. Unions as well as corporation and trade associations may be represented. Scientists and technicians from private institutions engaged in teaching and research also frequently attend to present their data and opinions. Also, private citizens may participate without need for sponsorship. All opinions and data are then assembled and presented in the Federal Register as an intended final standard with a deadline for last minute corrections. After the deadline, the finalized document is adopted as a legally binding regulation under the Act and distributed to OSHA

compliance officers for enforcement. The OSHA standard for asbestos has two parts: first, an 8-hour time weighted average (TWA) of 2 fibers > 5 μ/cc and second, a ceiling limit of 10 fibers > 5 μ/cc for any period of 15 minutes or less (i.e., the time required to collect the sample is 15 minutes).

Q. What is an AFOSH standard?
A. An internal Air Force standard intented to be at least as stringent as the OSHA standard in meeting the intent of the law.

Q. What is the current AFOSH standard for asbestos?
A. The same as OSHA, both for TWA and ceiling limit.

Q. You mentioned the ACGIH--what is that?
A. The American Conference of Governmental Industrial Hygienists. In general, to be a member you must be an industrial hygienist employed by a local, state or federal governmental agency. One of the most important functions of the ACGIH is the Threshold Limit Committee which sets limiting concentrations for airborne levels of chemical substances in the work environment to assure worker protection 8 hours-a-day, 5 days-a-week for a working lifetime (which could be up to 50 years). Exposures below that threshold limit value (TLV) should not induce occupational illness or impairment in all, or the great majority of workers, i.e., 95%+. When OSHA (the administration) was enacted, one of its first acts was to adopt the ACGIH TLVs, in toto, as OSHA standards.

Q. How does this affect the current asbestos standards?
A. The initial OSHA standard was the existing ACGIH TLV of 5 fibers > 5 μ/cc (which was based on the British Occupational Hygiene Society's (BOHS) Standard published in 1968). In 1975 the BOHS recommended moving the standard to 2 fibers/cc. The ACGIH TLV and the OSHA permissible exposure limit (PEL) were modified to reflect this change.

EDITOR'S NOTE: Concurrrently in 1975 OSHA announced an intention to consider lowering the PEL to 0.5 fibers/cc. In Oct 83 it published 0.5 fibers > 5 μ/cc as an Emergency Temporary Standard, with intent to change the PEL by this means. The Asbestos Information Association (AIA) has filed suit in Federal Court against such action based on the 1975 OSHA position, indicating the AIA has been urging downward revision using the normal procedure, including public hearings. Emergency action, by passing public hearings is not justified, therefore, according to AIA. The court has agreed to set aside the Emergency Temporary Standard pending review and decision.

Q. Has the ACGIH made any recent announcement regarding the basis for its existing TLV?
A. Its Documentation of the TLVs is updated annually and continues to list the TLV for most forms of asbestos as 2 fibers > 5 μ/cc 8-hour TWA. There is no ceiling concentration specified by ACGIH, however, as appears in the OSHA and AFOSH standards, i.e., 10 fibers > 5 μ/cc.

Q. Will you tell the Arbitrator what the acronym NIOSH represents?
A. The National Institute for Occupational Safety and Health.

Q. And how does it function under the law?
A. It was also created by the OSHA Act of 1970 and is intended to develop and present to OSHA criteria for standards to be adopted by OSHA after completion of the process involving public hearings, described earlier.

Q. Are NIOSH recommendations tantamount to OSHA standards necessarily?
A. No. They must be considered an early stage in the evolution of OSHA standards. You will often see NIOSH contract out the development of criteria documents to independent facilities such as universities and private laboratories or individuals. The resulting product is submitted to a Project Officer at NIOSH. A cover letter is then added in order to forward the document to OSHA for consideration; there is no guarantee of peer review by a fully qualified and experienced industrial hygienist or other reputable persons with scientific and technical background and recognized competence in the field.

Q. Before proceeding further, will you identify this exhibit for the record?
A. It is the ACGIH's Documentation of the TLV for Asbestos, dated 1981.

Q. Thank you, I am giving one copy to the Union and hereby offer it into evidence as Management Exhibit No. 9.

ARBITRATOR: Any objections?

UNION REPRESENTATIVE: No

ARBITRATOR: So ordered. Continue Maj Schlabs.

Q. Returning to NIOSH, from what you have said, it appears that the NIOSH recommendation does not necessarily represent a consensus standard on safety and health?
A. That is correct.

Q. Regarding asbestos, what is the current (1981) recommendation by NIOSH for exposure limits?
A. 0.1 fibers > 5 μ/cc 8-hour TWA.

Q. What is the justification?
A. The NIOSH goal is to provide a standard as close to zero as possible to measure and 0.1 fibers represents the sensitivity of the method specified and in use.

Q. What is the meaning of a zero threshold?
A. If we assume that a single hit can result in cancer after many years, the only way to prevent cancer would be to prevent the single hit.

Q. When did NIOSH first recommend 0.1 fibers?
A. In the Criterion Document for Asbestos dated 1976.

Q. So it has been over five years?
A. Yes.

Q. What change has been made by OSHA?
A. There has been no revision in the asbestos standard since then. (NOTE: i.e., prior to 1981. See above note also.)

Q. You mentioned studying under Eula Bingham at the University of Cincinnati. Who is she?
A. In 1977 she was appointed by then-President Carter as an Assistant Secretary of Labor to head the Occupational Safety and Health Administration.

Q. Was the NIOSH recommendation (for lowering the standard for asbestos from 2 to 0.1 fibers > 5 μ/cc) before OSHA during her tenure as the Assistant Secretary of Labor for OSHA?
A. That was made just prior to her coming on board as OSHA administrator.

Q. Did Miss Bingham--did Dr Bingham in any way cause the NIOSH recommendation to become the national consensus standard, the OSHA standard?
A. What she did during her tenure was to establish a NIOSH/OSHA working group and this group published a revised criteria document for asbestos in 1980.

Q. Notwithstanding any of those actions, she did not cause the OSHA standard to be lowered to the level recommended by NIOSH, did she?
A. She did not.

Q. How would you characterize the NIOSH approach to standards setting for asbestos?
A. Zero dose concept.

Q. Is there any other scientific basis for standards setting?
A. Yes. It's called the Threshold Limit Concept.

Q. Please explain this concept to the Arbitrator.
A. The Threshold Limit is a level of concentration in the worker's breathing zone below which the great majority of exposed workers will not develop occupational illness, disease or death. Those unduly susceptible can be screened from such employment or placement. If found to be adversely affected after beginning exposure, the susceptible worker should be protected from permanent harm by removal from further exposure at the earliest evidence suggestive of harm (e.g., x-ray evidence of asbestosis).

Q. O.K., Has OSHA leaned towards the zero dose or Threshold Limit Concept in the past in standards setting?
A. The ACGIH TLV premise since its origin prior to World War II has been based on controlling potentially hazardous exposures below the Threshold Limit. And, as I mentioned earlier, the initial standards published by OSHA accepted the ACGIH TLVs, in toto, as PELs for purposes of OSHA standards of enforcement.

Q. Thank you. I believe that answers the question. Now, are you familiar with the data presented by Maj Collins at Tab 5 of Joint Exhibit No. 2?
A. Yes. I would prefer to discuss the categories separately, i.e., the Carpenter Shop first and then the mechanical rooms.

Q. What about the carpenters?
A. Personnel doing power sawing appear to have been exposed above the OSHA ceiling of 10 fibers > 5 μ/cc, but Maj Collins stated that they wore NIOSH-approved respirators which provided a protection factor of 10 and would adequately protect a worker up to a ceiling of 100 fibers > 5 μ/cc. I noted that there is local exhaust which reduces the fiber concentration effectively as it is generated.

Q. I need to get something straight--you have indicated that OSHA has not accepted the NIOSH recommended standard for asbestos as being based on a different premise than the one to which ACGIH, and you yourself, depend. Yet, you appear to approve of reliance upon an NIOSH-approved respirator for protection of exposed personnel. A. NIOSH has also the responsibility for certifying personal protective equipment including respirators.

Q. A separate function from standards setting?
A. Correct.

Q. Have you any reason to disagree with NIOSH on their certification of protective gear?
A. No.

Q. What if these workers were not wearing the NIOSH-approved protection?
A. Then they would be exceeding the OSHA standard, the AFOSH standard, and thus, would not be adequately protected.

Q. Would it be your opinion then that those workers (in the Carpentry Shop) were, in fact, not exposed to a hazard from asbestos of an unusual nature?
A. That would be my opinion.

Q. For the entire period they were performing that operation?
A. Yes. Incidentally, I concur with Maj Collins' recommendation that they no longer use this cement asbestos board.

Q. For the record, it should be cemestabord?
A. Yes. And go to a suitable substitute that does not contain asbestos.

Q. Thank you. Now, from your review of the data, is there any indication of an unusual hazard to employees not involved in sawing who might be in the Carpentry Shop?
A. Maj Collins' data collected in adjacent work areas show asbestos levels to be well below 2 fibers > 5 μ/cc, and I do not believe there is hazard to the health of workers in adjacent areas.

Q. O.K. Is there any other category of work in which you believe there might be unusually hazardous asbestos exposure based on your review of all data?
A. On 22 Jul 80, personnel sampling of a worker conducting filter change out exceeded the 2 fibers > 5 μ/cc 8-hour time weighted average standard set by OSHA and AFOSH.

Q. That would be on the page that ran from Nov 78 through 4 Sep 80?
A. Yes.

Q. O.K. Now, have you an opinion on--having other data, would you base an opinion on that finding alone as to the degree of hazard to those workers?
A. The other data subsequent to these samples were much lower.

Q. Very well. And what about the practice of unsupervised vs supervised sample collection in such matters?
A. Maj Collins advised us that he just reported to this base about 22 Jul 80 and that neither he nor his Environmental Health Technicians supervised or monitored workers during this filter change out. It is standard industrial hygiene practice to observe and document the work practice in such cases. Subsequent sampling, which was conducted under Maj Collins' personal supervision, was much lower and did not, in fact, exceed the 2 fiber standard. Nevertheless, he recommended appropriate steps to improve hazard control, including the wearing of respirators which I said earlier give a 10X protection factor. So the workers were very well protected indeed.

Q. Why does supervised sampling carry greater weight over unsupervised in industrial hygiene surveys?
A. You can't be sure what occurred in the unwitnessed, unsupervised sample collection.

Q. Something accidental may have happened?
A. A contamination. An unusual work practice that would need to be sampled further is another possibility.

Q. Thank you. What would be the effect on any degree of hazard, high or low, of wearing a respirator?
A. Wearing a NIOSH-approved respirator provides an additional protection factor of at least 10--and that's a conservative estimate.

Q. Are there any other tasks which indicate an unusually severe hazard from asbestos?
A. None that I can observe from the data.

Q. I will refer you specifically to a sample taken on 17 Mar 81 designated No. 0064.
A. That is designated as an area sample from the Boiler Room. I noted in the data that there were four subsequent samples of that room, and each of these were well below 2 fibers > 5 µ/cc. Therefore, I concluded that this particular value of 5.996 fibers should be considered a statistical sampling outlyer.

Q. What is that?
A. If, in the presence of a pool of data, a single value presents as being high or low, it should be considered suspect and possibly rejected altogether in view of the other data. It could be a sampling or analytical error or something unexplained during the measurement that was not observed.

Q. Is there any health hazard from the area exposure data?
A. Except for that one outlyer, no.

Q. I believe you answered this but in the presence of a hazardous concentration, how would the asbestos get into the body to cause harm?
A. By inhalation.

Q. One point I neglected and had meant to ask; you mentioned earlier that the NIOSH recommendation is not characterized by peer review of the type which ACGIH uses in arriving at a consensus in setting its TLVs. Now I would like for you to compare the OSHA process. Please explain to the Arbitrator what goes on in making an OSHA standard in comparison with a NIOSH recommendation.
A. As I stated earlier, the proposed OSHA standard is published in the Federal Register with a date or dates selected for public hearings--usually within six months--and the NIOSH recommendations in the NIOSH criteria document would be subjected to review by all interested parties, including labor, industrial management and trade associations and universities--usually represented by industrial hygienists and technical experts or scientists who have good basis in experience and education for their expressed views. Once the public hearings have been completed, OSHA reviews and finalizes its standard based on total evidence presented at the hearing. By contrast, NIOSH usually goes out on contract to develop a literature review of the known occupational health hazards. That document is then returned to NIOSH where a cover letter is added in order to forward it to OSHA for their action as I have described.

Q. Now I will show you what has been marked as Union Exhibit No. 2 and ask if you recognize it.
A. Yes, this is a NIOSH/OSHA publication dated Nov 80, prepared by a NIOSH/OSHA working group on asbestos.

Q. What is its recommendation?
A. They recommend 0.1 fiber > 5 μ/cc for the TWA exposure for workers.

Q. Is this different from the one NIOSH first recommended several years ago?
A. No. If fact, the authors of the original 1976 document, Leman and Derment with the consultation of Wagner, are three of the four NIOSH members of the working groups. The other three members are from OSHA. The creation of such a NIOSH/OSHA working group is unique and appears puzzling to me. This is the first document to my knowledge that has attempted to include OSHA participation in the preparation of the NIOSH recommendations.

Q. Why?
A. Because from my understanding, the OSHA--the Act--developed NIOSH to be an independent agency to present technical information to OSHA for its action.

Q. Having examined the information contained in the working group's report, could you please tell the Arbitrator your impressions?
A. This particular document is in essence a restatement of the 1976 NIOSH document with no additional evidence to support their zero threshold concept. In 1976, the most experienced, best qualified speakers from the asbestos industry stated that there is no substantial evidence to support the NIOSH recommendation. I have reviewed the data, and I basically agree with the position specified in the ACGIH TLV Documentation--which was previously entered.

Q. That, for the record, is Management Exhibit No. 9?
A. Yes. While it was attacked in the NIOSH/OSHA working group document, may I refer to it here?

Q. Go ahead. He is referring to Management Exhibit No. 9.
A. The key difference between A..IH and the NIOSH recommendations related to the need to postulate a zero threshold, and I would like to direct attention to the last paragraph on page 28; whether or not there is a dose-effect relationship has been answered affirmatively by a number of epidemiological surveys (referring here to five such references). Whereas this relationship is clear-cut in the regard to asbestosis and lung cancer, it is less well marked with regard to mesothelioma but is nevertheless positive.

Q. What is the significance of that?
A. ACGIH states that there is a threshold limit value for exposure to asbestos.

Q. And that is?
A. For most fibers, 2 fibers > 5 μ/cc.

Q. Thank you.
A. Continuing, if I may, I find the major disagreement to be a paper authorized by Gillam, Tell, Leman and Dement. Please note that Leman and Dement are also co-authors of the NIOSH criteria document on asbestos of 1976 and of the NIOSH/OSHA working group recommendation of 1980.

Q. Why is that significant?
A. The Gillam study reported in 1976, the finding of an increase in the amount of lung cancer in hard rock gold miners exposed to an asbestiform mineral (Cummington-grunerite or amosite) at an exposure level below 2 fibers > 5 μ/cc. They found 10 respiratory cancer deaths in 440 miners (including a carcinoma of the maxillary sinus and a mediastinal carcinoma) where only 2.74 such deaths were expected. As pointed out in Management Exhibit No. 9, pages 29 and 30, however, and summarized in the table on the last page, an expanded study of 1,321 workers in the same company by McDonald et al., reported an expected respiratory cancer rate of 16.5 with only 17 found, which is not significantly different. So the ACGIH has cited McDonald's study results as the basis for disregarding the claims of Gillam, et al. The ACGIH TLV documentation also comments on the existence of a single epidemiological study with a cohort of workers who have had known, well documented asbestos exposure. That was the cohort published initially in 1955 by the British Occupational Health Society (BOHS). These workers represent the only cohort of asbestos workers in the world in which health effects have been correlated with definitive exposure data defined as fibers per cc. Despite acceptance of the ACGIH position by the concensus procedure, the NIOSH/OSHA working group's report continues to attack McDonald's report, pressing for zero threshold. The 0.1 fiber > 5 μ/cc standard proposed is at the level of sensitivity of the method commonly used for detecting asbestos in air, so their intent is obviously to have OSHA enforce a standard that states that if you can find any asbestos level present, the area measured is out of compliance. This viewpoint has not been supported by peer review, however, as manifested by the ACGIH position in Management Exhibit No. 9.

Q. What about cigarette smoking?
A. According to Hammond and Selikoff, cited in the same exhibit, on page 28, asbestos insulators who smoked had 5.4 times the expected rate of lung cancer. However, smoking insulators lung cancer rate was 14 times greater than nonsmoking workers' rate. It means that there is greater risk of lung cancer from smoking cagarettes than from uncontrolled asbestos exposure as an insulator—but that asbestos exposure further multiplies the risk of lung cancer in smokers. The need or prevent cigarette smoking, per se, is of the greatest concern in attempting to prevent lung cancer, whether or not there is exposure to asbestos.

Q. No further questions.

ARBITRATOR: Ms Rains—Cross examination?

MS RAINS:
Q. Of the two schools—zero or threshold—you subscribe to the latter?
A. Yes.

Q. What about dormancy of asbestos-related disease for many years?
A. I concur that the latent period for lung cancer is more than 15-20 years usually and perhaps twice as long for mesothelioma.

Q. Can you be sure the ACGIH won't lower the TLV?
A. If there should be good evidence that the existing standard does not protect adequately based on firm data, they will lower it. As indicated in Management Exhibit No. 9, they are following the BOHS cohort results closely. This cohort follows workers who entered scheduled areas since 1951 and on whom reports were published in 1955, 1965, 1968 and 1977 by highly qualified investigators. Is is apparent that all excess mortality which has occurred until now can be attributed to asbestos exposures far above 2 fibers > 5 μ/cc for a 50-year expected working lifetime.

Q. The study of asbestos is relatively young. Do you agree it may take 20 years before we get concrete evidence on this?
A. No.

Q. You don't agree?
A. The cohort in England mentioned previously has been exposed 30 years already, with reports in 1955, 1965, 1968 and 1977. The lack of evidence of harm at the 2 fiber exposure level becomes increasingly significant with time, of course, in arguing against need for a zero threshold.

MS RAINS: No further questions.

MAJ SCHLABS: I have no questions.

ARBITRATOR: Thank you very much.