

**IN THE UNITED STATES DISTRICT COURT  
FOR THE DISTRICT OF DELAWARE**

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In re	: Chapter 11
	: :
FEDERAL-MOGUL GLOBAL, INC., et al.	: Jointly Administered
T&N LIMITED, et al.,	: :
Debtors.	: :
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THE OFFICIAL COMMITTEE OF	: :
ASBESTOS CLAIMANTS and	: :
ERIC D. GREEN, as the	: :
LEGAL REPRESENTATIVE FOR	: :
FUTURE ASBESTOS CLAIMANTS,	: :
Plaintiffs,	: :
	: :
v.	: Case No. 05-00059 (JHR)
	: :
ASBESTOS PROPERTY	: :
DAMAGE COMMITTEE,	: :
Defendant.	: :
	: :
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**COMMENTS ON THE REPORT OF DR. LAURA S. WELCH, M.D.  
BY HANS WEILL, M.D.**

## **The Asbestos-related Diseases: Current and Future Status**

**Hans Weill, M.D**

**May 12, 2005**

### ***Introduction***

I have been asked by counsel to provide a statement of my opinions regarding medical explanations for the past, present, and likely future incidence and prevalence of asbestos-related disease. My curriculum vitae is attached.

### ***Background***

Knowledge that workplace exposure to asbestos-containing dust has caused adverse effects (disease) on the respiratory system has evolved since the early decades of the last century. Asbestosis (diffuse lung fibrosis) was recognized in the 1920s, lung cancer risks were confirmed epidemiologically in 1955 and mesothelioma was linked to exposure in 1960. These conditions can all cause structural and functional intrathoracic abnormalities and can shorten life-span. They are all dose-dependent, i.e. increased level and total amount of fiber exposure results in increased risk and/or severity of the diseases. Conversely, as workplace exposures have been substantially reduced in the last several decades, asbestos-related health effects have become less prevalent.

Reduction in airborne asbestos fiber levels in the various divisions of industry has been documented by OSHA and MSHA air sampling data(1). There is abundant quantitative evidence of dose-response relationships and consequent risk assessment pertaining to the asbestos health effects in the scientific literature(2,3). There are also published data on relevant disease incidences and mortality(1). It is this information, primarily from government and academic sources, and my personal clinical and research experience, which

informs the judgments expressed below regarding the likely current and future burden of asbestos-related diseases.

### ***Asbestosis***

Diffuse lung fibrosis is called asbestosis when it results from workplace exposure to asbestos dust. Asbestosis is a non-malignant condition. It is a type of pneumoconiosis which is usually diagnosed by chest x-ray film and review of patient occupational and environmental history, but when lung tissue is available (which rarely occurs), pathologic examination should be considered the "gold standard" in the establishment of this diagnosis. Pathologic criteria have been developed to assist in this assessment(4). Radiographic evaluation is often based on an international classification, revised in 2000, published in 2003(5), which categorizes the type and extent of small opacities which may be consistent with diffuse scarring of the lungs. Standard films showing varying levels and types of opacities are used for comparison in order to classify the films of individuals being evaluated for occupational lung disease. The radiographic pattern of asbestosis, is consistent with a generalized interstitial pattern, is non-specific and a diagnosis (of asbestosis) must be considered along with an appropriate occupational exposure history, the absence of another, more likely, condition causing the x-ray pattern, and other probable asbestos-associated findings, such as pleural plaques.

The experience of most chest physicians (pulmonologists) during the last couple of decades has been that of a markedly diminishing number of new cases of asbestosis; many have not seen a new case in years. This experience is in contrast to the increasing number of claims for asbestosis being filed. The reason this is so in the litigation arena is the direct result of the striking over-reading of chest films by predictable readers who have a credential called "B-reader" certification(6). A recent study quantified the differing pattern of readings between plaintiff's experts and other chest radiology consultants(7).

While this study has limitations, it provides useful information and strongly suggests that there has been systematic over-reading of chest x-ray films by asbestos screening facilities, not simply random variability between B-readers in film classifications. There is no registry of asbestosis cases in the U.S., and even if there were, it would be grossly unreliable because of the inordinate influence of litigation on the validity of these diagnoses (diagnostic bias). Any temporal trends which were generated from these flawed diagnoses would be meaningless.

We do know from the National Center for Health Statistics that asbestosis *mortality* rates declined significantly in the 1990s in those under age 65 at the time of death(1), this group having been at work during the time that asbestos exposures were declining. It should be kept in mind, however, that diagnoses on death certificates of non-malignant respiratory disease, including asbestosis, (almost always without pathological examination) are not nearly as reliable as an objective diagnosis made during life by a qualified examiner using objective chest x-ray film readings. Both death certificate and hospital discharge diagnoses of asbestosis have in recent years been highly unreliable, due mainly to the effect of litigation and "mass-screening" resulting in many former workers having been told that they have "asbestosis". This litigation diagnosis can be transmitted by the patient or family to their physicians - but not independently verified outside the litigation-screening context-- and therefore invalid diagnoses are accepted and recorded.

As regards the current and future burden of asbestosis we know the following: 1) the prevalence and severity of the disease has repeatedly been shown to be dose-related; 2) exposure levels have been declining significantly during the last three decades because of government regulation and improved industrial hygiene; and 3) the anecdotal experience of consultant pulmonologists indicates marked reduction in the number of asbestosis cases being seen, as one would expect from 1) and 2). The default assumption must be that

asbestosis is a gradually vanishing disease. Any estimate of the incidence of asbestosis that does not take into account the systematic over-diagnosis of the disease in the litigation arena through a statistically valid sampling approach and application of objective diagnostic criteria and methodology will necessarily overestimate the incidence of valid asbestosis claims.

### ***Benign Pleural Effects (Plaques and Diffuse Pleural Thickening)***

Asbestos exposure can result in focal (plaques) or diffuse pleural thickening, due to fibrosis (scarring) of the pleural surface of the lungs. In a very high percentage of such cases, no functional abnormality results (as measured by lung function tests) and an increased risk of future asbestos-related malignancies has not been convincingly demonstrated(8). In fact, the *absence* of elevated lung cancer or mesothelioma risk, in the presence of pleural effects alone (in the absence of asbestosis) and taking exposure into account, has been found(9). These pleural effects, while dose-dependent, can probably result from lower levels of asbestos exposure than asbestosis, but no hard data exist in regard to temporal trends of their prevalence.

### ***Lung Cancer***

Lung cancer related to asbestos exposure does not differ clinically or pathologically from that most commonly seen as the result of cigarette smoking. Thus, knowing the temporal trends for lung cancer in the U.S. (which we do) does not inform on the central issue of the course of those "caused" by asbestos exposure. If, however, there was a necessary linkage between asbestosis and asbestos-attributable lung cancer, (i.e. no excess lung cancer risk in exposed populations in the absence of asbestosis), then the question about time trends of asbestos-related lung cancer becomes one of trends in asbestosis incidence. In other words, declining risk of asbestosis means declining risk of lung cancer as well.

In 1988 and 1991, we reported results of a prospective study in which there were both individual estimates of cumulative dust exposure and health data, including smoking and radiographic status at the outset of observation(9,10). The subjects were 839 men in two asbestos cement plants, who were examined in a cross-sectional morbidity study in 1969 and then followed into the mid-1980s for cause-specific mortality. Twenty or more years after hire, no excess of lung cancer was found among workers without radiographic evidence of lung fibrosis; nor was there a trend in risk by level of cumulative exposure to asbestos among such workers. By contrast, employees with small opacities ( $\geq 1/0$ ; ILO classification with films read by three independent experts for *research* and not in a litigation context) experienced a four-fold increase in risk of lung cancer, even though their exposures to asbestos were similar to the exposures of long term workers without opacities. We concluded that the excess risk of lung cancer in that population was restricted to workers with radiographic evidence of asbestosis, a finding consistent with the view that asbestos is a lung carcinogen because of its fibrogenicity. Other studies of asbestos-exposed workers are consistent with these findings and no convincing contrary evidence has been published. Other causes of lung fibrosis (or idiopathic - no known cause) have also been shown to be associated with increased risk of lung cancer(11), lending biologic plausibility to the hypothesis linking inflammation (the pre-fibrosis process), fibrogenicity and carcinogenesis.

### ***Mesothelioma***

Trends in U.S. mesothelioma incidence have recently been reported(12). Among the asbestos-related diseases enumerated above, mesothelioma is the most sensitive and specific indicator of the adverse health effects that have resulted from airborne exposures to asbestos fibres. It is sensitive because it is likely that this tumor can result from lower levels and shorter duration of

exposure than the other conditions, and specific because mesothelioma, a rare tumor, is likely to be the consequence of asbestos exposure in a high proportion of cases, especially in men. Mesothelioma risk is dependent on the dose of asbestos exposure, with substantially enhanced risk related to amphibole exposure (crocidolite and amosite) in comparison to chrysotile exposure. The median latency period (time between first exposure and clinical manifestation of the tumor) is around 30 years. The reduction in asbestos exposure referred to above, particularly to amphibole asbestos, would be expected to result in diminishing mesothelioma risk beginning about three decades after reduced exposure. In the U.S., peak mesothelioma incidence occurred in the early to mid-1990s and has started to decline since then. This is probably primarily related to reduction in amphibole use since its peak importation into the U.S. in the 1960s. Projections of the number of future cases of mesothelioma should be based on the use of this trend information and estimates of annual mesothelioma incidence.

### ***Quantitative Risk Assessment (QRA)***

Apart from the above discussion regarding asbestosis as a necessary precursor to asbestos-attributable lung cancer, the clear and repeated demonstration of dose-dependency of malignancies caused by exposure to asbestos has led to approaches to quantify these risks for use by regulatory agencies, and others(13). The QRA process requires selection of suitable models and studies utilizing valid quantitative estimates of exposure and disease. Since the exposures of interest (e.g. promulgation of standards for current permissible exposure limits - PELs) are far lower than those found in air sampling data and estimates from past, much higher, exposures, the risk estimates depend on extrapolation from these past occupational exposures to the recent lower exposure levels for which there are no data. The wide acceptance of QRA in the federal government (e.g. EPA, OSHA, CPSC) supports the credibility of dose-dependency of asbestos-related risks. The lowering of asbestos PELs over

the years(14) is a main cause of the reduced exposures in recent decades. The expected consequence of this regulatory/exposure history for asbestos is the continuing marked decline of the conditions which have resulted from past exposures to asbestos. To believe that the asbestos diseases have increased (or even remained at past levels) in recent years and will continue to do so is strikingly counterintuitive.

The perceived asbestos disease burden *will* "increase", however, if systematic over-diagnosis of asbestos-attributable diseases continues. Regrettably, the recent American Thoracic Society (ATS) official statement on asbestos(15) will not lead to more credible diagnoses of non-malignant asbestos-related disease since the diagnostic criteria set out in the statement have been unjustifiably broadened. The statement lacks a sufficiently comprehensive literature review and needed balance in citing of the literature in the presentation of medical issues on which there is more than one side. There was blatant omission of evidence contrary to the opinion of committee members. Criteria on imaging for the diagnosis of asbestos-related non-malignant respiratory disease are vague and appear to include any present or future unsupported notions in regard to what may constitute evidence for an asbestos-related lung or pleural effect. Assertions regarding increased risk of malignancy due to presence of pleural plaques is based on 2 papers by a committee member without reference to substantial and *far stronger* contrary evidence. The ATS has set out guidelines for the development of official society statements indicating what is expected of the committees charged with this responsibility(16). This statement fails to meet these published ATS objectives and will not help in bringing more rationality to the diagnosis of asbestos-related diseases. It is of interest that several letters to the editor criticizing the statement have subsequently been published in the same journal and, because of widespread dissatisfaction with the statement, a special public forum regarding the

statement is being held this month at the annual meeting of the American Thoracic Society, in San Diego.

### ***Opinions of Dr. Laura Welch***

I have been asked to comment on the report of Dr. Laura Welch, submitted in April, 2005 in this matter. A problem that applies to the entirety of Dr. Welch's report is that the bases for her various opinions are not referenced by specific papers in the scientific literature. A list of papers on asbestos-related disease at the end of her report is not an adequate substitute for indicating *which* papers in the literature are specifically being relied on for the many opinions contained in the text of her report.

Dr. Welch refers to a 1982 paper by Nicholson, et al, giving projections for asbestos-related mortality in subsequent decades. More than two decades after they were published, these estimates have thus far been shown to be overstated, and cannot accurately reflect recent exposure conditions and therefore the current and future burden of asbestos-related disease associated with the markedly lower exposures as compared to the observed period of Nicholson.

Dr. Welch emphasizes "other" cancers as a consequence of asbestos exposure (e.g. larynx, gastrointestinal) despite the fact that the asbestos health effects literature is increasingly skeptical that causal inferences as regards asbestos are well supported(17-27), particularly since most of the evidence of "excess risk" comes from one group of investigators (Selikoff and colleagues). Such findings have not been replicated by the many other investigators who have published mortality studies of occupational cohorts exposed to asbestos, including our research group(3).

I agree, in general, with the discussion on lung function but fail to see the relevance of it to the issues in this case since physiology offers little information regarding *causation* of diffuse lung diseases. Referring to the recent ATS statement on asbestos(15) the suggestion is repeated that the pulmonary diffusing capacity (DLCO) is "important" in the evaluation of asbestos disease. In addition to the fact that reliable DLCO measurements are almost never available in claimants, we and others have shown that the DLCO is no better in assessing asbestos-associated impairment than are simple spirometric measures (e.g. forced vital capacity - FVC)(28).

In Dr. Welch's report, there is the suggestion that substantial exposures are not always necessary for the development of asbestos-related disease and that individual (host) factors may play a role (no evidence is cited for this proposition). In our research(29,30), we looked for many years for evidence that, in addition to exposure dose, host factors influence the risk or severity of nonmalignant asbestos-related disease in large exposed populations and never found such evidence. Nor has anyone else.

Dr. Welch suggests that asbestosis may be present in an undefined percentage of individuals the absence of abnormalities on the chest x-ray film. I agree that lung fibrosis (from any cause, including asbestos exposure, or no known cause) may indeed exist while the chest film is read as normal. This is not, however, relevant to the *diagnosis* of asbestos-related non-malignant disease because one needs either imaging or pathologic evidence to make a diagnosis. In regard to high resolution CT (HRCT) scans, it has not been adequately demonstrated in epidemiologic studies that changes on the more sensitive HRCT are dust dose-dependent, as have the changes on the chest x-ray film (as classified by ILO categories). Importantly, I am not aware of credible studies which have shown dose-response relationships for the HRCT changes, taking into account other factors (e.g. smoking) which could influence the images. Failing this demonstration, causal inferences as regards HRCT scans are

problematic and cannot be viewed as being “widely accepted” as a technique for the diagnosis of asbestosis. Additionally, Dr. Welch’s report does not demonstrate the wide-spread diagnostic use of HRCT scans to detect asbestosis.

In making a case in regard to the utility of HRCT, as with other matters, Dr. Welch relies heavily on the recent ATS asbestos statement and mentions “expert consensus” without identifying which experts. I have discussed the limitations of the ATS statement earlier in this report, citing a lack of balance and comprehensiveness in their review of the literature. The committee chosen to prepare this society document regularly failed to cite literature contrary to the well-known opinions of committee members. As indicated previously, letters to the editor critical of the statement have recently been published.

Dr. Welch discusses asbestos exposure, asbestosis and asbestos-attributable lung cancer. Dr. Welch posits that such a lung cancer does not require asbestosis as an intermediary. I have discussed our work on this issue in this report and refer the reader to our published research reports and review of the literature(9-11). Dr. Welch offers the recent paper by Cullen, et al as support for her opinion. Quite to the contrary. The Cullen study suffers from very serious flaws: 1) as well illustrated by the title of this paper, the investigation was not designed for the purpose of examining the link between lung cancer causation and the absence or presence of asbestosis - it was done to determine whether certain vitamins (beta-carotene and retinol) prevented lung cancer in high risk individuals (mainly smokers). They did not. 2) The reading of chest x-ray films to determine the presence of changes indicating asbestosis was performed by different single readers in various clinics, not a standard approach in the use of chest film reading for epidemiological studies. Multiple readers (usually three), reading all the films is the accepted way of studying the effects of mineral dust exposure, the method used in all of our studies of asbestos-exposed populations. And, 3) there is no quantitative exposure

information available in the Cullen study, making it impossible to rule out confounding by exposure differences as an explanation for their findings.

Further, in regard to lung cancer risk, Dr. Welch mentions the "Helsinki Criteria", implying international "consensus" on this and other issues. Rather, that document constitutes consensus of those with like opinions. Using a putative asbestosis threshold for cumulative asbestos exposure (25 fibers/cc-years) in assessing asbestos attributability for lung cancer might be an acceptable approach if there were a valid method to quantitatively estimate past asbestos exposure in claimants. There is not.

The fact that lung cancer risk is related to amount of asbestos exposure in an occupationally-exposed cohort in no way weakens the position that asbestos-attributable lung cancer risk requires the presence of asbestosis. And in those cohort studies (including our own early mortality study [3]), the excess risk of lung cancer may very well have been limited to those with asbestosis. To my knowledge, no *retrospective* mortality studies have been able to distinguish between those with asbestosis and those without. That is why we did our most recent *prospective* mortality study, since it *could* (9,10), and we found that the excess lung cancer risk was *limited* to those workers whose chest x-ray film showed evidence of asbestosis (as measured by an ILO reading of small opacities of  $\geq 1/0$ ). As indicated previously, I believe that the weight of the credible evidence on this point supports the position that lung cancer can be judged to be "caused" by asbestos exposure only in the presence of asbestosis.

Dr. Welch says that even in the *absence* of x-ray changes, asbestosis may still be present, and thus lung cancer may be caused by asbestos exposure even when there is no radiographic evidence of exposure.<sup>1</sup> To support this opinion, Dr. Welch relies on the Kipen study (31). We also cited Kipen in *support* of our

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<sup>1</sup> As previously demonstrated, even assuming this is true, it ignores that a diagnosis of asbestosis generally requires radiographic or pathologic evidence.

findings of a linkage between lung fibrosis and attributable lung cancer. One can't have it both ways. Kipen's study showed that in their insulators, there was *more* than enough asbestosis to fully account for the *excess* lung cancer risk (see discussion above).

In conclusion, I believe that the totality of the scientific and medical evidence leads to the firm conclusion that the occurrence of asbestos-related diseases is currently at quite a low level, in comparison to past decades, and these diseases will continue to become more and more infrequent in the coming years.

### ***Compensation***

Drs. Weill is being compensated for their work on this project at the hourly rate he customarily charges for expert consulting services and court testimony, plus out-of-pocket expenses. Dr. Weill's rate is \$400 per hour, or \$4,000 per day when traveling.

### ***Prior Expert Testimony***

A list of cases in which Dr. Weill has given expert deposition or trial testimony in the past four years is attached.

Respectfully submitted,

A handwritten signature in black ink, appearing to read "Hans Weill". The signature is written in a cursive, flowing style with some loops and flourishes.

Hans Weill, M.D.  
Professor of Medicine Emeritus  
Tulane University School of Medicine

## References:

1. Work-Related Lung Disease Surveillance Report 2002; Division of Respiratory Disease Studies, National Institute for Occupational Safety and Health, U.S. Department of Health and Human Services, Public Health Service; Centers for Disease Control and Prevention, December 2002.
2. Hughes JM, Weill H. Asbestos exposure - quantitative assessment of risk. *Am Rev Respir Dis* 1986; 133:5-13.
3. Hughes JM, Weill H, Hammad YY. Mortality of workers employed in two asbestos cement manufacturing plants. *Br J Ind Med* 1987; 44:161-174.
4. Craighead JE (Chairman), Abraham JL, Churg A, Green FHY, Kleinerman J, Pratt PC, Seemayer TA, Vallyathan V, Weill H. The pathology of asbestos-associated diseases of the lungs and pleural cavities: diagnostic criteria and proposed grading schema. Report of the Pneumoconiosis Committee of the College of American Pathologists and the National Institute for Occupational Safety and Health. *Asbestos-Associated Diseases (special issue), Archives of Pathology and Laboratory Medicine* 1982; 106:541-596.
5. International Labour Office. International classification of radiographs of pneumoconioses. Geneva, Switzerland, 2003.
6. Wagner GR, Attfield MD, Kennedy RD, Parker JE The NIOSH B reader certification program. An update report. *J Occup Med.* 1992 Sep;34(9):879-84.
7. Gitlin JN, Cook LL, Linton OW, Garrett-Mayer E. Comparison of "B" readers' interpretations of chest radiographs for asbestos related changes. *Acad Radiol.* 2004 Aug;11(8):843-56.
8. Weiss, W., Asbestos-related pleural plaques and lung cancer. *Chest,* 1993. 103(6): p. 1854-9.
9. Hughes JM, Weill H. Asbestosis as a precursor of asbestos-related lung cancer: results of a prospective mortality study. *Br J Ind Med* 1991; 48:229-233.
10. Hughes JM, Weill H. Pulmonary fibrosis as a determinant of asbestos-induced lung cancer in a population of asbestos cement workers.

- Proceedings of the VIIth International Pneumoconioses Conference, Pittsburgh, Pennsylvania, August, 1988. DHHS (NIOSH) Publication No. 90-108, Part I, September, 1990, pp. 370-374.
11. Jones RN, Hughes JM, Weill H. Asbestos exposure, asbestosis, and asbestos-attributable lung cancer. *Thorax* 1996; 51(Suppl 2): S9-S15.
  12. Weill H, Hughes JM, Churg AM. Changing Trends in US Mesothelioma Incidence. *Occup Environ Med* 2004;61:438-441.
  13. Hughes JM, Weill H. Asbestos exposure - quantitative assessment of risk. *Am Rev Respir Dis* 1986; 133:5-13.
  14. Weill H, Hughes JM. Asbestos as a public health risk: disease and policy. *Ann Rev Publ Health* 1986; 7:171-192.
  15. Diagnosis and Initial Management of Nonmalignant Diseases Related to Asbestos. *Am J Respir Crit Care Med* 2004;170(6):691-715.
  16. <http://www.thoracic.org/adobe/guidelines.pdf> Accessed on September 27, 2004.
  17. Richard Doll and Julian Peto Asbestos: Effects on health of exposure to asbestos Report to the UK Health & Safety Commission, Her Majesty's Stationery Office, 1984
  18. Gamble JF. Asbestos and colon cancer: a weight-of-the-evidence review. *Environ Health Perspect.* 1994 Dec;102(12):1038-50.
  19. Garabrant DH, Peters RK, Homa DM Asbestos and colon cancer: lack of association in a large case-control study. *Am J Epidemiol.* 1992 Apr 15;135(8):843-53.
  20. Reid A, Ambrosini G, de Klerk N, Fritschi L, Musk B. Aerodigestive and gastrointestinal tract cancers and exposure to crocidolite (blue asbestos): incidence and mortality among former crocidolite workers. *Int J Cancer.* 2004 Sep 20;111(5):757-61.
  21. Cocco P, Ward MH, Dosemeci M Risk of stomach cancer associated with 12 workplace hazards: analysis of death certificates from 24 states of the United States with the aid of job exposure matrices. *Occup Environ Med.* 1999 Nov;56(11):781-7.
  22. Liddell D. Cancer mortality in chrysotile mining and milling: exposure-response. *Ann Occup Hyg.* 1994 Aug;38(4):519-23, 412.

23. Browne K, Gee JB. Asbestos exposure and laryngeal cancer. *Ann Occup Hyg.* 2000 Jun;44(4):239-50.
24. Goodman M, Morgan RW, Ray R, Malloy CD, Zhao K. Cancer in asbestos-exposed occupational cohorts: a meta-analysis *Cancer Causes Control.* 1999 Oct;10(5):453-65.
25. Liddell FD, McDonald AD, McDonald JC. The 1891-1920 birth cohort of Quebec chrysotile miners and millers: development from 1904 and mortality to 1992. *Ann Occup Hyg.* 1997 Jan;41(1):13-36
26. Kraus T, Drexler H, Weber A, Raithel HJ. The association of occupational asbestos dust exposure and laryngeal carcinoma. *Isr J Med Sci.* 1995 Sep;31(9):540-8
27. McLaughlin JK, Lipworth L. Epidemiologic aspects of renal cell cancer. *Semin Oncol.* 2000 Apr;27(2):115-23
28. Weill, H., et al., Lung function consequences of dust exposure in asbestos cement manufacturing plants. *Arch Environ Health*, 1975. **30(2):** p. 88-97.
29. deShazo RD, Daul CB, Morgan JE, Diem JE, Hendrick DJ, Bozelka BE, Stankus RP, Jones R, Salvaggio JE, Weill H. Immunologic investigations in asbestos-exposed workers. *Chest* 1986; 89:162S-165S.
30. Weill H, Hughes JM. Asbestos health effects: resolving the scientific uncertainties. *Postgrad Med J* 1988; 64 (supplement 4):48-55.
31. Kipen HM, Lilis R, Suzuki Y, Valciukas JA, Selikoff IJ. Pulmonary fibrosis in asbestos insulation workers with lung cancer: a radiological and histopathological evaluation. *Br J Ind Med.* 1987 Feb;44(2):96-100