PLAINTIFF'S EXHIBIT
UC-3590

Cadwalader, Wickersham & Taft

March 30, 1979

MEMORANDUM TO: Asbestos Information Association/NA
Legal Research Committee

FROM: Carey E. Gross
Cadwalader, Wickersham & Taft

RE: Asbestos-Associated Disease:
With Particular Reference to
U.S. Shipyards, a Conference at
the Mount Sinai Medical Center
November 27-29, 1978

On November 27, 28 and 29, 1978 I attended a conference given at the Post-Graduate School of Medicine of the Mount Sinai School of Medicine in New York. The conference was a post-graduate course and was part of the 1978-1979 program of the University's Occupational Safety and Health Education Center, supported by the National Institute for Occupational Safety and Health.

Approximately 300 people attended the conference, roughly broken down as follows: 40% doctors; 40% attorneys; and 20% workers and union members. The conference was well organized. I have summarized below some of the more interesting discussions at the conference. All of the statements below were made by participants at the conference and do not necessarily reflect the opinions or beliefs of the writer. A complete tape recording of the proceedings is available from this office.

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Much of the presentation was medically oriented, with a lot of attention to historical data and epidemiological studies. Dr. Irving J. Selikoff and Dr. Kaye H. Kilburn directed the conference. A majority of the speakers seemed to direct their presentations to the needs and situations of plaintiffs in asbestos-related disease litigation.

The first day of the conference involved very little "new" information. I sensed that many members of the audience were new to this area and a thorough historical presentation was being made for their benefit. Events and studies with which we are all familiar were presented and emphasized.

The conference began with an introduction by Dr. Selikoff, quoting the August 8, 1978 statement of HEW: "approximately 8-11 million workers have been exposed to asbestos...1.5 to 2.5 million are now being exposed...effects of exposure won't appear for 15 to 30 years."

Without being exhaustive, some of the names and dates that were presented in that first day of the conference were: (1) 1907 - Dr. H. Montague Murray reported first recorded death of asbestosis in Great Britian; (2) 1930 - first case report study on asbestosis in U.S.A., exposure was in South Africa; (3) 1938 - Dreessen, et al. proclaimed the TLV of 5mppcf; (4) 1955 - Wright pioneered analysis of pulmonary function patterns; and (5) 1957 - O'Donnell and Ward called attention to lung cancer (in 13 out of 27 cases in hospital near factory, lung cancers were in the lower lobe).

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Further, many general statements about asbestos-related diseases were made. Examples are: (1) changes in normalcy of x-rays are largely seen after long latency period - approximately 20 years; (2) one out of every five insulation workers in this country dies of lung cancer; (3) historically, cancer has been seen much more in the lower lobes of the lungs and linings instead of the non-peritoneum and higher in the lung where those carcinomas normally occur; and (4) asbestos-related cancers are often beyond the skill of a surgeon's knife (autopsy photos supporting this proposition were presented). Dr. Selikoff stated that recent data shows a statistically significant increase in the appearance of carcinomas in the larynx, pharynx, and kidney in asbestos workers. The clinical latency period for these cancers appears to be roughly 15 to 40 plus years.

Dr. Selikoff feels that the real problem with exposure to asbestos is cancer not asbestosis. A significant factor in this area is the end product use. Dr. Selikoff seems to feel that workers in companies manufacturing asbestos-containing end products have more health problems than those in the production and mining segment of the industry.

Dr. Selikoff asserted that, in 1959, doctors began to see mesothelioma cases in workers exposed to only amosite. This was suprising, since earlier medical opinion held that mesothelioma was caused by crysotile exposure. References were: Wagner-1974, British Journal of Medicine, and Molly Newhouse. He further asserted that brief exposure to asbestos, if excessive, may be as hazardous as long term exposure to lower concentrations. Emphasis was placed on reports that an

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increased incidence of cancer was found in workers who had been exposed to asbestos for only one day at the Patterson plant (such workers were purportedly exposed to very high dust levels and bad conditions).

Another aspect of asbestos-related diseases was discussed by Dr. Selikoff. In the profession it is now called "bystander" disease. This does not refer to those working directly with asbestos-containing materials. Rather, bystanders are individuals who contract an asbestos-related disease without primary occupational exposure to asbestos-containing materials. These are generally, but not necessarily, family members of the asbestos worker.

Dr. Selikoff summarized lessons concerning asbestos-related diseases learned during the last 15 years:

1. Dose-disease response relationship;
2. Pre-eminence of cancer is replacing asbestosis;
3. Pleural mesotheliums presenting with scant exposures;
4. Importance of end-product use; and
5. Cigarette smoking and its effect on cancer in asbestos workers.

Researchers are now finding that, in animal models, glass fibers can cause the same mesotheliums as is seen in humans exposed to asbestos. However, it is not yet known if the results will appear in humans exposed to glass fibers.

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Dr. Nicholson asserted that, in the shipbuilding industry, those associated with thermal insulation have the greatest chance of contracting asbestos-related diseases. The speaker emphasized importance of the total working environment, rather than just the particular trade. The prevalence of fibers and dust throughout the shipyard is, according to Dr. Nicholson, evidenced in abnormalities among all shipyard workers. Guards without previous yard jobs and draftsmen without any experience in the yards have been diagnosed as having some of the asbestos-related diseases. It is estimated that approximately 4-1/2 million different individuals were employed in shipyards during World War II.

Dr. Selikoff discussed the fact that researchers have seen in utero transfer of asbestos exposure in animals but not in humans. He also asserted that children of workers who contract asbestos-related diseases show symptoms 20-30 years after birth. The assumption is that they inhaled fibers in their home, probably carried by their father's clothing or hair or body, not from in utero exposure. Dr. Selikoff noted that exposure cannot really be stopped once a single exposure has occurred because the inhaled fibers continue to expose the lungs and the lung lining.

The speakers pointed out that, for laymen reading some of the epidemiological data, it is important to understand the "healthy worker effect". In general, the best comparison for all studies is the U.S. population data. But for the first

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five years of most studies of any type of workers, there seems to be 30-40% fewer deaths than among the general population. This is because the general population has a higher proportion of sick people, while the working population is generally healthier. Therefore, for the first five to ten years of employment, the working population is healthier than the general population. As a result, if researchers find identical statistics from the general population and from workers early in their careers, something is wrong.

Dr. Selikoff discussed the fact that workers usually die of lung cancer with asbestosis, though there are cases where death is due to asbestosis (very short of breath) with the presence of lung cancer. Pulmonary scarring and cor pulmonale cause difficulty in pulmonary functions, although there are no good data yet about correlations between these two conditions and heart attacks.

There was an interesting and in-depth presentation of the mineralogy of asbestos. In parts it was quite specific chemically, but most of what was discussed should be common knowledge to members of the asbestos industry. Apparently deposits of crocidolite are contaminated with amosite. This raises an interesting point with respect to litigation in which the specific types of asbestos or asbestos fibers is important.

Dr. Langer discussed "Brownian movement" which plays an important part in the airborne quality of asbestos fibers.

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The very small fibers do not fall rapidly due to other atoms and molecules moving in the atmosphere. These other particles push the small asbestos fibers around - often pushing them upwards. In a vacuum, the smallest fibers would settle - but this is not so in the atmosphere. Therefore, some fibers may never settle regardless of gravity. This leads to an increased duration of possible exposure to the fibers.

Dr. Langer asserted that there are no known safe exposure levels to asbestos. However, he believes that, as dose exposures decrease, there should be a decrease in the incidence of the various diseases. He said that there is no known exposure standard in the United States to prevent cancers. As a result, the industries and sciences work on a regulatory system trying to minimize exposure and possible related diseases.

Dr. Nicholson discussed difficulties with early dust measurements. The basic problems noted were:

1. Few counts were done;
2. Those that were done did not use current techniques;
3. All particles (mppcf) were counted rather than only fibers; and
4. Conversion factors were uncertain.

Mr. Michael Wood, Director of Safety and Communications, International Brotherhood of Boiler Makers, read from a Chicago Tribune newspaper article: "The federal government has the heaviest of responsibilities in finding solutions to problems of millions of workers exposed." Mr. Wood then asserted that

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management must collectively bargain over the safety and health "insult" it has rendered its workers.

During a question and answer period there was a discussion concerning the devalued American dollar and the effect it may have on asbestos-related diseases. A speaker asserted that many foreign ships are being brought to the United States for repair. Therefore, shipworkers may be working on these ships without knowing what materials they are being exposed to. This may increase the likelihood of their exposure to asbestos and asbestos-containing materials.

It was stated that medical expertise in clinical and pathological interpretation throughout this country is generally sufficient. Further, the American College of Radiology is now training its technicians to read and interpret x-rays so that there will be a higher degree of accuracy in the specific area of pulmonary problems and x-rays relating to pulmonary diseases.

Dr. Selikoff gave a brief presentation on the ILO (International Labor Office) classification system. This system was designed to classify films and x-rays for epidemiologists, statisticians and others in the field. Its function is purely one of classification (the preamble to its design states that this classification is not for workmen's compensation and the like). What follows is a very brief summary of what Dr. Selikoff said.

Pneumoconioses are dust diseases of the lung. ILO began labeling x-rays to provide a standard classification system for lung diseases. The classifications originally showed

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what was occurring in those exposed to coal dust and some silica. The classifications described nodules, but when the medical profession began seeing asbestosis films they could not use the same descriptions for two reasons. First, there were no nodules. Second, asbestos-related problems were apparent not only in the lungs, but also in the pleura, and the initial classification slides did not relate to non-lung problems.

Dr. Selikoff reiterated that it is important to remember that there is a much greater amount of pathology in the lungs than can be seen on x-rays. X-rays always underestimate what is there. Because of this, a whole new set of standards and classifications were developed regarding asbestos and asbestos-related diseases and films showing each of these. The result is a new (1978) classification system. This will include a proposed set of standard films so that anyone looking at the films will know exactly what is meant by the various classifications. In other words, doctors can compare the films of their patients to the standard films that come with the classification package. There is also a proposal that there be a set of end-category films. These films would show both ends of each category so that a particular classification will include a film from the middle (or average) of that category and both extremes of that category.

Dr. Rabin asserted that mesothelioma of the pleura is rarely encountered among people without any asbestos exposure. He stated that autopsies will reveal less than one out of 10,000

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persons have mesotheliomas of the pleura, yet 2 to 10% of persons with asbestos exposure develop pleural mesotheliomas. Therefore, according to Dr. Rabin, there is a definite correlation between exposure to asbestos (either occupationally or by contact with one so exposed) and the development of pleural mesotheliomas. Note, however, that according to speakers at the conference, studies in Turkey have revealed pleural mesotheliomas as a result of exposure to xeolite.

Diffuse pleural thickening, diffuse pleural plaques (mainly on the diaphragm) and pleural calcification are often seen in asbestos workers. In pleural thickening, the pleura often creates a case around the lung causing shortness of breath.

Dr. Tierstein stated that a conglomerate shadow on the lung x-ray does not necessarily mean cancerous. Also, the presence of an asbestos fiber on a biopsy does not necessarily mean asbestosis. However, primary cancerous of the lung is metastatic to the pleura. There may be a very sophisticated new instrument on the horizon which will help differentiate between cancerous and pulmonary fibrosis. It is called a "Neta scan," (spelling phonetic).

The following is a summary of points made by Dr. Tierstein with respect to primary bronchogenic carcinoma.

1. Pulmonary asbestosis may mask a tumor;
2. The cancer is more frequent in the lower lobes;
3. The cancer is usually "peripheral";

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4. Carcinoma is increasingly common without pulmonary asbestosis; and

5. It is difficult to differentiate between pleural carcinoma, pleural metastases and mesothelioma.

There appears to be a possibility of using nuclear medicine in determining primary bronchogenic carcinomas. A gallium scan may be able to detect some of the fibers in the lung tissues. Those patients with malignant mesothelioma would be detected by gallium scanning and differentiated from those with benign pleural involvement.

Dr. Rabin stated that lung cancer is eight times as great in those exposed to asbestos as in the general U.S. population. He also asserted that blood-gas studies are unlikely to help diagnose asbestosis. Apparently it is common to have negative x-rays although various pulmonary problems exist. It is Dr. Rabin's view that asbestosis and carcinomas have no dose-response relationship to one another -- each has their own dose-response curve. Diagnostic thoracotomies are not recommended by Dr. Rabin. He noted that there have been three deaths due to diagnostic thoracotomies at Mt. Sinai. There appears to be a low resistence to post-thoracotomy infection.

According to Dr. Churg, mesothelioma is much less common than the lung cancers but much more specific. Carcinoma of the pleura is lung parenchyma surrounded by mesothelioma. Histologically, mesothelioma may look very similar to carcinoma of the pleura. Any tumor that gets into the pleura will grow

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in the same manner. Mesotheliomas are 70-75% epithelial.

Dr. Suzuki noted that a histological section is not a good way to determine asbestosis.

A light microscope may be used, according to Dr. Langer, as long as one understands the caveats to its use. More sophisticated instruments include the light beam and the electron beam microscopes.

An interesting caveat to the British asbestos standards was discussed by Dr. Nicholson. The British standards were designed to prevent (or lower the incidence of) asbestosis. He asserted that no one knew what were "safe" standards to prevent cancer. The problem with the standard for carcinogens, as stated at the conference, is that even at the present time we do not know thresholds for carcinogens. Therefore, Dr. Nicholson recommended reducing exposure levels as much as possible.

In discussing current standards he noted that there is no historic or scientific basis for the "two fiber standard." Further, the five micron length was stated to be an arbitrary cut-off to allow the counting of fibers with the light microscope. Dr. Nicholson stated that we don't know that shorter fibers do not cause problems. Similarly, he asserted that there are 100 times as many fibers shorter than five microns as those more than five microns in length.

Dr. Selikoff believes that it is very difficult to have dust counts done. Further, he asserted that approximately

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15% of a man's life is lost if he becomes an asbestos worker. He did note that it is important to remember that we are talking strictly averages and that the results are different for every worker. The passage of time from onset of exposure is critical. It is important to remember that for research purposes a study period of less than 20-25 years is not as significant as the period after 30 years.

Dr. Selikoff continued by stating that there is a sharp dose-response relationship for lung cancer - the longer a worker has worked with asbestos, the more likely he is to get lung cancer - but again the latency period is crucial. The tissue burden continues to affect with no further exposure other than the initial exposure which can be one month, one year, even one week. The smaller the initial dose, the longer the induction period (dose-induction period). These were said to be the biological parameters that must be taken into consideration.

One segment of the conference was directed toward discussing "family contact diseases." Dr. Anderson explained that this relates to children and women exposed to workers who were occupationally exposed to asbestos. It is believed that the dust and fibers brought back into the home by the worker were carried on their bodies, in their hair and on their work clothes. There is a relative risk factor of ten for women with mesothelioma. This means that a woman with mesothelioma is ten times as likely to live (or have lived) in a home with an asbestos worker than a woman in the control group.

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According to Dr. Anderson, asbestos fibers persist in the atmosphere. In some homes that were tested, asbestos fibers were found on the walls, ceilings and framework over the door as much as 30 years after the last known working date of the asbestos worker. Wives have been found to have the greatest chance of contracting asbestos-related diseases. Curiously, daughters were found to develop asbestos-related diseases the least, less than their male siblings. No one is sure why this is so.

Following the above background in the medical and epidemiological aspects of asbestos-associated diseases, presentations were made relating to workmen's compensation, product liability and legal aspects of the asbestos disease problem. I will now turn to a summary of some of those presentations.

In discussing the scientific basis for regulatory determination of disability, Dr. Kilburn noted that "disability is an opinion." He explained that there are four classes of pulmonary respiratory impairment that correspond to the four classes of cardiovascular impairment. Mr. Howard Vincent then said that the length of time from time of injury to the first disability check received by the claimant is approximately 60 days in injury cases and approximately one year in occupational illnesses. He continued that one out of every ten injury cases is contested and there is an average award of $25,000. Six out of every ten illness cases are contested and there is an average award of less

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than $10,000.

According to Mr. Paul Gillenwater the earliest form of occupational diseases are set apart from accidental injuries for two reasons:(1) the disease is not accidental but is usually expected; and (2) the disease has developed over a long period of time versus a sudden onset (like an accident).

Compensable disability can also be divided into two categories -- a disability of a medical or physical nature and a de facto inability to earn wages. The two types usually occur simultaneously, but they can occur independently.

There are four classes of disability: temporary-total; temporary-partial; permanent-total; and permanent-partial. A more detailed discussion of these four classes of disability is contained in the American Medical Association "guide" to permanent disability. It is important to remember that in determining disability the focus is not on the loss of earning capacity but on the cause of the loss of earning capacity (i.e., is it occupational?). As of October 1, 1978 the maximum rate of compensation was $396 per week.

Mr. Matthew Shafner revealed that a determination of disability is not only dependent on a diagnosis but also on age, education, industrial history and available employment which the claimant can obtain after injury. Further, disability in a legal sense does not become permanent until it reaches maximum medical improvement. This determination is made by an administrative law judge. Mr. Shafner said that written medical reports may not be introduced into evidence in disability hearings and in asbestosis cases it is generally desirable - and often necessary -

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to have medical expert witnesses testify. If a claimant returns to work after injury this does not preclude a finding of permanent or total disability. Mr. Shafner emphasized that disability is a forward-looking concept, not just an examination of past events. Mr. Henderson added that the statute of limitations on disability claims begins to run when the claimant knows or reasonably should have known about his (1) exposure or (2) disease or (3) harmful exposure.

There was a discussion of some particularly difficult scientific and legal question which arise in workmen's compensation proceedings particularly when there is a long latency period. Dr. Selikoff reiterated that tissue change is generally undetectable in asbestos-related diseases until a 20-year latency period has run. Therefore, there is a progression of disability and burden on the tissues which follows the cessation of exposure. It is possible that the incremental insults may not is coming from only one source. This was related to the combined effects of smoking, environmental problems, pollutants in the water and air, and the like.

Mr. Shafner continued by mentioning that the last employer or last insurance carrier is generally responsible for compensation. In filing multiple claims there should be no overlapping of benefits, but there should also be no gaps. The government has a right to a third-party lien if the employee has been treated for compensable disease by a VA clinic. Workmen's compensation may affect the amount of social security received although social security does not affect workmen's compensation

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awards. It is hard to obtain total compensation through social security.

Mr. Gene Locks posed the following crucial questions for determining legal points in workmen's compensation claims: Does a disease exist at a sub-clinical stage? or only when it is detectable at radiographic stages? or when pulmonary function changes become evident?

With respect to the varying statutes of limitation, in Mr. Henderson's opinion, the time when the cause of action accrues could be: (1) when the plaintiff suffers harm; (2) when the plaintiff becomes aware of the disease process; (3) when the plaintiff recognizes the causual realtionship between exposure and the disease; or (4) the defendant's date of duty (last exposure date).

Mr. Ronald Motley asserted that a good radiologist should be able to distinguish between fibrosis caused by smoking and fibrosis caused by asbestos exposure. He also stated that asbestos fibers and cigarette smoking act independently and either can cause lung cancer. He pointed out that a non-smoking asbestos worker has a five times greater chance of contracting lung cancer then the general population.

Mr. Roy Steinfurth stated that compensation claims are usually too late or inadequate to halt the chain of "collateral" events that occur when a worker has contracted an occupational disease. Some of those events are depression, break up of the family, depletion of savings, loss of wages, and the like. He

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asserted that it is of utmost importance to have attorneys who are well versed in workmen's compensation and products liability law. He noted that the time from application to the award of benefits is often two or three years during which time the plaintiff often dies. The burden of proof is then on the widow or other representative, especially in state compensation cases.

There are two states which, as yet, have not awarded any compensation to asbestos workers. Those states are Mississippi and Louisiana.

Mr. Albert Millus pointed out there is no such thing as an inert dust, but there are great individual differences in the susceptibility to dust exposure and pulmonary insult. Dr. Selikoff added that in 15% of all mesotheliomas cases asbestos cannot be identified as the cause and that it does not make biological sense that only one agent could cause a cancer. The last statement was supported by evidence from Turkey of two different types of zeolite causing this type of cancer.

On the last day of the conference approximately two hours of the afternoon session were used to demonstrate a workmen's compensation hearing. There was a direct examination and cross-examination of the plaintiff's pathologist, a direct examination and cross-examination of the defendants' clinician, closing arguments and a rebuttal. Complete tapes of these presentations are available.

If any AIA\NA member wishes further information concerning this conference, or wishes to obtain copies of any of the tapes, please contact me.

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