IPCB HEARING
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Thank you very much for the opportunity to appear in support of your regulations controlling the use and production of asbestos materials and spray insulation.

For purposes of identification, my name is William J. Nicholson. I am Assistant Professor of Community Medicine at the Mount Sinai School of Medicine of the City University of New York. As a physicist, I have served as coordinator of several projects undertaken by the Environmental Sciences Laboratory of Mount Sinai to evaluate the effects of asbestos exposure. These include an industrial hygiene research program designed to identify and control asbestos hazards in the insulation industry, the development of analytical techniques for the quantitation of asbestos in ambient air, and a study of asbestos air pollution in New York City and throughout the United States. Additionally, I am engaged in several epidemiological studies of disease associated with occupation or environmental exposures to various substances. Two of these studies involve the effects of asbestos on defined populations.

The justification is strong for controlling the use of asbestos on basis of the health effects produced by its use in the past. The evidence that heavy occupational exposure to asbestos is associated with pulmonary disease dates from 1900 when massive fibrosis was found at post mortem in the lungs of a man who was the last survivor of a group of 10 men who worked since 1886 in the carding room of an asbestos textile mill. Cases of asbestos-induced scarring of the lungs, asbestosis, were seen with increasing frequency in subsequent years as the use of asbestos increased. However, a hint of additional disease risk from occupational asbestos exposure was seen in 1935. Two cases of lung cancer were found in individuals having asbestosis. This portent was grimly verified in

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later studies. In 1947, the Chief Inspector of Factories in Britain found lung cancer in 13 per cent of deaths in which asbestosis was present.

To bring us up to date, 70 years after the first knowledge of asbestos disease, and to complete the picture of occupational risk from exposure to asbestos, let me present some data obtained by Dr. Irving J. Selikoff, Director of the Environmental Sciences Laboratory, Mount Sinai School of Medicine. He has followed since January 1, 1943 the 632 members of the New York and New Jersey Locals of the asbestos workers union, the International Association of Heat and Frost Insulators and Asbestos Workers. These men apply the insulation to high temperature pipes and boilers in apartment, office building and individual plants. Some of the material they use contains a small amount of asbestos -- 7 to 15 per cent, usually. Often it contains none. In comparison to the factory exposures during early decades of this century, their exposures are relatively light.

Knowing their ages in 1943 and using standard tables, it was possible to calculate the expected mortality experience of these 632 workers. This was done by Dr. E. Cuyler Hammond, Chief Epidemiologist of the American Cancer Society, and the results, along with those actually seen, are given in Table I for the years 1943 through 1962.

From all causes, 203 deaths were expected of those who had worked 20 years. In fact, there were 255. More than 50 men who died should not have died. Looking at the table, we can see the causes of these "excess death": 45 died of cancer of the lung, trachea and pleura where only six or seven were expected; 12 died of asbestosis although none

should have died; three times as many died of cancer of the G. I. tract as were expected.

Results of continued observation of the group through June 30, 1971 are shown in Tables II and III. They represent a catastrophe. Over half of the deaths were from some form of cancer or asbestosis. Nearly 40% could be termed "occupational" deaths.

In these latter data, special reference should be made to the high death rate from mesothelioma. Twenty-seven cases of this, as yet, incurable disease were seen. A diffuse cancer of the lining of the chest or abdomen, it is so rare that it has yet to be separately classified in the International Classification of Causes of Death. Its occurrence in the general population may be so infrequent as to cause only one death in 10,000. Here, it was the cause of death in one of every 16 individuals. Moreover, in other studies, its appearance in increased amounts has always been associated with asbestos exposure.

Two other aspects of the research by Doctor Selikoff are important. The first is the evidence of the long lapsed period between onset of asbestos exposure and the incidence of disease. Lung cancer usually appears between 20 and 40 years after first exposure to asbestos, and mesothelioma most often after a 35-year lapsed period.

The second aspect is the strong synergism found between asbestos exposure and cigarette smoking in the incidence of lung cancer. By personal interview in 1962, the smoking habits of 370 of the 632 men were established. By following the mortality experience of these 370 men, a special risk of cigarette smoking was established. From January 1, 1963 through February 1, 1971, in 283 men with a history of cigarette smoking, 40 lung cancers were observed. This is nearly ten times the number ex-

pected in an equivalent cigarette smoking population. In 87 non-smokers, only one lung cancer was seen. Thus, it is not asbestos alone, or cigarette smoking alone, but the combination of the two that produced these grim data. In fact, it is calculated that asbestos workers who smoke run more than 90 times the risk of dying of lung cancer than men who neither smoke cigarettes nor work with asbestos.

These data I have presented to you were from occupational exposures to asbestos. At most, 200,000 men and women in the United States have such exposures. Hopefully, 70 years after the first awareness of asbestos disease, working conditions will be improved to reduce their risk of disease. But were this the only risk of asbestos exposure, it would not be germane to the regulations under consideration here. Unfortunately, in recent years the evidence has accumulated that the problem of asbestos disease has disseminated from the occupational area into the general environment.

Evidence is accumulating that mesothelioma is rapidly increasing among men who worked in the vicinity of asbestos application. Recall that mesothelioma is virtually nonexistent in people who have had no exposure to asbestos. In Holland, 22 cases were found during a 6-year period in workers of the de Shelde shipyard. These cases occurred, however, not in asbestos workers, but in members of other trades, welders, carpenters, or painters, all of whom only worked in an area where asbestos insulation was applied or removed. Similar data have appeared from Great Britain. Here, 80 cases of mesothelioma were found in a survey of the material in the pathology departments of Scottish hospitals and medical schools from the years 1950 through 1967. An investigation of the occupational background of these cases revealed a strong association

with shipbuilding or marine work. Here again, however, the cases were not often found in insulators but in other trades working near the application or removal of asbestos. A further disconcerting feature of the Scottish data is the rapid rise in the rate of occurrence of mesothelioma; 51 of the 80 cases were seen in the years 1964-67, approximately 30 years after the extensive shipbuilding undertaken during World War II.

The spectrum of disease associated with asbestos now extends even to environmental exposures. In 1960, Wagner, Sleggs and Marchand described 47 cases of mesothelioma found during a four-year period in the northern Cape province of South Africa, an area with extensive crocidolite asbestos mines. None were seen in the neighboring Transvaal Province or in 10,000 autopsy cases of workers who had died with known exposures to silica. Of the 47 cases, approximately half were found to have had either industrial or mining exposures to asbestos. Virtually all of the remaining cases were in individuals who simply lived or worked in the vicinity of the asbestos mines or mills.

Confirmation of the possibility of environmental asbestos-associated disease soon appeared. Newhouse and Thompson reviewed all mesothelioma cases at the London Hospital. They corroborated the close association with asbestos, 31 of the 76 cases having had documented occupational exposure. Of the remainder, 11 had lived, decades before, within one-half mile of an asbestos factory. A similar distribution of cases was found by Lieben and Pistawka in Pennsylvania. One insidious form of indirect asbestos exposure is that of families of asbestos workers. In the previously mentioned studies, 9 of the London mesotheliomas were in family members, as were 3 of the 46 Pennsylvania cases.

Unfortunately, these data on the incidence of mesothelioma from environmental or indirect occupational exposure to asbestos are severely limited. The exposures in question took place beginning 20, 30, 40 or more years ago. No knowledge exists of the associated asbestos dust exposure levels. Thus, we have no dose-response information on low-level asbestos exposure. We only have knowledge of a potential risk of cancer, at exposures much below occupational ones. Obviously, additional research and continued surveillance are highly desirable.

Let us now turn to specific items in your regulations. Firstly, the general requirements of Section 2 are excellent, particularly with regard to responsibility and education. We find a great problem to be the ignorance of a potential hazard among marginal users of asbestos. Such ignorance can easily result in lax procedures.

Secondly, the banning of asbestos in spray insulation is entirely appropriate. In New York City we have found this to be a major contributor to ambient air asbestos levels. For the past 10 years, during which spray fireproofing came to be extensively used, it was not uncommon to see extensive snowfalls of asbestos-containing material over widespread areas of New York and other metropolitan centers. In some cases, the fireproofing was even done with no attempt to enclose the spray area by tarpaulins or other means.

To verify that such fireproofing operations were a significant source of respirable asbestos fiber, air sampling was conducted in lower Manhattan about construction sites where extensive spraying of asbestos-containing fireproofing was taking place. This work was done by our Laboratory in conjunction with the Department of Air Resources of New York City. The results demonstrated that spray fireproofing can indeed con-

tribute significantly to asbestos air pollution.

Up to ¼ mile away, levels exceeding 0.1 micrograms per cubic meter were observed, which exceeds by 100 times background levels found in urban areas distant from construction sites. While 0.1 micrograms may appear to be a small amount, it could represent more than 10 million minute fibers of asbestos. Moreover, the large clumps of asbestos-containing material covering the sidewalk serve as a continuing source to be dispersed by passersby.

As I mentioned earlier, lacking a dose-response relationship, we cannot state that environmental exposure levels from this practice are certain to produce disease. However, it is hard to imagine factory emissions, even 40 years ago, being more excessive. Since the extensive spraying of asbestos-containing fireproofing material has only existed in the construction industry for the last 10 years, disease caused by the exposure resulting from this practice is 10 or 20 years away. However, continuing the uncontrolled spraying practices of the past decade in the face of the mounting evidence of risk to all metropolitan residents would be playing asbestos roulette with the lives of millions of people.

This method of fire protection for high rise buildings presents an additional, continuing risk to the occupants of those buildings. Often, the space between the false room ceiling and the under side of the floor above, which has been sprayed with asbestos, serves as a return plenum for the air supply system. After filtration, inadequate to remove most asbestos fibers that may have eroded into the air stream, this return air is recirculated throughout the building. The magnitude of this problem has yet to be defined. Until recently in New York City, we have not been allowed to analyze the air in buildings that has been in direct contact

with asbestos-containing insulation.

Finally, this spray procedure leaves a legacy of potential risk to future generations. The lifetime of modern office buildings is short and the procedures for the safe future demolition of buildings that could contain more than 100 tons of asbestos as fireproofing have yet to be devloped. Perhaps these procedures will be found and perhaps they will be followed but we have no such assurance at this time.

Unfortunately, safe procedures developed for the spray application of asbestos-containing materials have not been followed either voluntarily or under control regulations by contractors. Because of this failure and because alternative procedures or materials are available, action banning this use of asbestos is indeed appropriate.

Moreover, because of uncertain knowledge of the health effects of exposure to mineral fibers other than asbestos, it is entirely appropriate that strict control be exercised over other spraying of such substitute materials. While some forms of "mineral wool" and other mineral fibers have been in use since the 1930s there are still no definitive studies on exposed populations that would, at this time, provide assurance that respirable mineral fibers, other than asbestos, are biologically inert in humans. In the absence of such assurace, caution is warranted and I support, in its entirety, Section 3 of your regulations.

On the other items, in Section 4, you might wish to add a qualifying statement that when there is potential release of asbestos to the air, a special enclosure shall be used. Many asbestos products used in construction can be installed with no release of fiber. Asbestos-vinyl floor tile is one example. In fact, with proper procedures, even asbestos cement can be applied with minimal release of fiber.

I cannot emphasize too strongly the need for stringent controls during demolition. Those listed here may not be adequate and close surveillance should be maintained to verify their adequacy. For example, I am not sure that any asbestos should be transported via chutes to trucks. It is hard to imagine that the debris would be completely wetted.

In reference to Section 6 A, I would like to raise some of the problems assocated with air monitoring which may be severe. In ambient air samples, it is difficult, and often impossible, to identify asbestos fibers by optical microscopy alone. There are many birefringent fibers with a similar appearance. Even the sampling of stacks containing only asbestos has its problems. Such sampling does not evaluate any emissions from windows, room exhausts or uncontrolled sources. Moreover, the use of optical microscopy can misestimate actual concentrations, even assuming accurate counting of all fibers longer than 5 microns. Often as many as 100 times more fibers shorter than 5 microns may be present. If those counted were a constant fraction of the total present, this would be no problem. However, such is not the case. In different uses of chrysotile we have found the ratio of the number of fibers longer than 5 microns to the total number to vary from 1/250 to 1/20. Since bag houses have a size dependent efficiency, the results obtained by optical analysis may be misleading and not comparable to other samples analyzed by similar means.

The only way to accurately asses environmental exposures is by electron microscopy. Here, however, an analysis may cost $200 - $300 and be very time-consuming. You may wish to consider procedural controls with the requirement of no visible emissions of asbestos from any building or site. If the facility inspected questions the judgement of an inspector they could pay for adequate air analysis by electron microscopy.

I enclose in Table IV data from our work analyzing samples of ambient air from nearby 50 U. S. cities. These data show that a background of some asbestos fiber is found in all cities, but occasional peaks appear. From this occasional occurrence and from the fiber size distribution on high samples, it appears that the contribution of some specific source is important in these high levels.

Finally, one comment on the proposed ban of asbestos in brake linings after 1975. The problem here is there is a dearth of information. We have some very preliminary information that asbestos air levels near sites of extensive braking may be 3 times higher than background. Since wind conditions are highly variable, much more analysis of this question is required. All our data really show is that brakes are not as obvious a problem as asbestos-containing fireprooofing spray in New York City. Perhaps you may wish to qualify 7 B by the words "Unless evidence of safety is demonstrated the use of asbestos...."

In summary, your regulations in general are excellent, your intent is proper, and I am pleased to support you.

Table I

Observed and Expected Number of Deaths
Among 632 Asbestos Workers Exposed to
Asbestos Dust 20 Years or Longer, 1943-1962

Cause of Death	Years 1943-1947	1948-1952	1953-1957	1958-1962	Total, 1943-1962
Total, all causes.....					
Observed (asbestos workers)	28	54	85	88	255
Expected (US white males)..	39.7	50.8	56.6	54.4	203.5
Total cancer, all sites.....					
Observed (asbestos workers)	13	17	26	39	95
Expected (US white males...	5.7	8.1	13.0	9.7	36.5
Cancer of lung and pleura....					
Observed (asbestos workers)	6	8	13	18	45
Expected (US white males)..	0.8	1.4	2.0	2.4	6.6
Cancer of stomach, colon and rectum.....					
Observed (asbestos workers)	4	4	7	14	29
Expected (US white males)..	2.0	2.5	2.6	2.3	9.4
Cancer of all other sites combined.....					
Observed (asbestos workers)	3	5	6	7	21
Expected (US white males)..	2.9	4.2	8.4	5.0	20.5
Asbestosis.....					
Observed (asbestos workers)	0	1	4	7	12

Death rates as reported annually by the U.S. National Office of Vital Statistics. A five year average is given here for each period.

Death rates include cancer of lung, bronchus, pleura, mediastinum and trachea, assigned to international list code No. 47b-f prior to 1949 and to No. 160-164 thereafter.

Table II

Expected and observed deaths among 370 asbestos insulation workers, Jan. 1, 1963 - June 30, 1971.

	Observed deaths	Expected deaths*
Total cancer (all sites)	92	15.1
Cancer of lung, pleura, trachea, bronchus	47	4.5
Lung cancer	43	+
Pleural mesothelioma	4	+
Peritoneal mesothelioma	19	+
Cancer of stomach, colon and rectum	12	3.0
Cancer all other sites	14	7.6
Asbestosis	20	‡
All other causes	51	63.5
Total deaths	163	78.6

*Expected deaths are based upon age-specific rates for U.S. white males in 1967. Smoking habits are disregarded.

+United States data not available but figure should be only slightly less than 4.5.

‡United States data not available but these are rare causes of death in general population.

Table III

Mortality experience of 632 members of IAHFIAW, Locals 12 and 32
January 1, 1943 to June 30, 1971

Cause of Death	Number	Per Cent
Lung cancer	85	20.0
Pleural mesothelioma	7	1.6
Peritoneal mesothelioma	20	4.8
Gastro-intestinal cancer	40	9.3
All other cancers	36	8.4
Total cancer = 188		
Asbestosis	32	7.7
All other causes	205	48.2
	425	100.0

Table IV

Asbestos Air Levels in United States Cities

Analysis of 187 Quarterly Composite Samples

Asbestos Air Levels (10^{-9} grams/m^3)	Number of Samples With Given Vaule
0.0-0.9	61
1.0-4.9	103
5.0-9.9	12
10.0-19.9	8
20-49	2
50-99	1