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Internal
Memorandum

Bendix Research
Laboratories

Date August 21, 1973 Letter No.

To Distribution

From W. M. Spurgeon

Subject PRESENTATION AND CORRESPONDENCE ON ASBESTOS

Southfield, Michigan



Attached are copies of documents received on August 17 from the Friction Materials Standards Institute.

On first reading it appears that the experts are not in agreement. The fact that ingested or inhaled asbestos is harmful to health is not questioned. What Drs. Selikoff and Lewinsohn are both saying, in effect, is that the safe exposure levels, if any, are not known. The various parties differ in the rigor, and hence the cost, of the protective measures to be adopted.

To some, Dr. Selikoff appears to be an overzealous crusader. In fairness to him it should be realized that an epidemiologist has to be a crusader in order to be effective in his work. Further, it is not a tenable position to advocate that rigorous control methods should be postponed until safe exposure levels are established. It is not possible to establish them, since well-designed experiments on human subjects are both impermissible and impractical in this matter.

In such a situation it is better to err on the safe side, pushing ahead with our programs for control of asbestos in our factories, for labeling our aftermarket friction materials, and our search for new fibrous reinforcing materials.

W. M. Spurgeon
ew

WMS/ew

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AUG 17 1973

August 14, 1973

W. M. SPURGEON

TO: MEMBERS OF ASBESTOS STUDY COMMITTEE

SUBJECT: DR. SELIKOFF PRESENTATION TO AMERICAN INDUSTRIAL HYGIENE ASSOCIATION

Your Chairman, Mr. I. H. Weaver, has sent the enclosed papers for distribution to the Committee.

1. Presentation Dr. I. J. Selikoff to the American Industrial Hygiene Association on May 21, 1973 (Includes Question & Answer Session).
2. Letter from H. C. Lewinsohn and S. Holmes of Turner Brothers Asbestos of July 12, 1973 to Dr. Selikoff.
3. Letter from H. Swetonic, Asbestos Information Association N.A. to J. H. Stender of OSMA dated July 24, 1973. (Forwarding Dr. Selikoff's allegations and the comments of Lewinsohn and Holmes).
4. Letter from G. F. Scannell, OSMA, of July 31, 1973, to H. Swetonic acknowledging receipt and advising on distribution of enclosures.
5. Mr. Swetonic letter of August 3, 1973 covering distribution of correspondence to members of AIA.

Mr. Swetonic's letter of August 3, 1973 to the AIA Membership summarizes the presentation and review of a talk Dr. Selikoff gave to the American Industrial Hygiene Conference in Boston, Massachusetts, on May 21, 1973. In essence, Dr. Selikoff is attacking the 2 fiber/cc asbestos exposure level in the work place as being too loose. This is the 2 fiber limit that goes in effect in 1976 (not the 5 fiber limit now in effect).

For your information.

E. W. Drislane
Executive Director

EWD:llz
Enc.

Presentation on Asbestos
by
Dr. Irving J. Selikoff
at
American Industrial Hygiene Conference
Boston, Massachusetts
May 21, 1973

This year, 1973, marks 50 years since the examination of the first case of death due to asbestosis. Dr. Cooke in Leeds described this fatal disease in 1924. He might well have been asked the question which points a dilemma in which we still are. If he had been asked what exposure of this asbestos textile worker resulted in her fatal pulmonary fibrosis, he would have had to say, "I don't know." Because 20 years ago when she began work in this textile factory, no dust counts were done. Now, the dilemma that he faced is still very much with us at this time because the disease that we are now seeing is the result of exposures at a time when we were not alerted and environmental measurements were not made. We find this a great difficulty in our own work and perhaps it may be well demonstrated by the first slide which I will read to you in view of the circumstances.

This is a slide of the x-ray results in 1,000 employee workers in the New York area examined by us and it demonstrates that out of 725 people with less than 20 years from onset of exposure, most had normal x-rays. When they were abnormal, they tended to be minimally so. On the other hand, more than 20 years from

onset of exposure, of the over 300 men so examined, few had normal x-rays; and when they were abnormal, not infrequently, the fibrosis was extensive. So the 20 year rule which was found in 1924 is present with us today. The description of lung cancer, 1935, and subsequently of pleural mesothelioma in 1954 and afterwards, in 1955 of peritoneal mesothelioma and subsequently of other tumors, has done nothing to resolve this question because they too have had this difficulty which we still face. We have found it so in our studies.

For example, in the New York area, we have been following 632 members of the Asbestos Workers Union; these are the insulation workers, who were on the union rolls on January 1, 1943. You will see in this slide that for the first 10 years from 1943 to 1947 and 1948 to 1952, the total number of deaths was almost exactly what was expected. It was not until after this 10 year period had passed that we began to see the extraordinary mortality patterns that are still with us. We found, for example, that after 20 years wherein there should have been 6 or 7 deaths of cancer in the lungs and pleural mesothelioma, there were 45. There should have been 9 or 10 deaths of cancer of the stomach, colon and rectum. There were three times as many, 29, and, of course, there were 12 deaths of asbestosis where none had been expected. Yet, as we go longer with this group - we have followed them now through December 31, 1971 (and these are unpublished data; I thought you might find

... interesting) - we have found that of the survivors of this 343 group, there should have been another 85 deaths, but 168 have occurred. There should have been 4 or 5 deaths of lung cancer. There were 42. There should have been no deaths of mesothelioma, which almost never occurs in the general population. There were 25. There was the small increase, 3 or 4 times increase, in gastro-intestinal cancer and the asbestosis deaths, so that the longer we go from onset of exposure, the more evident the risk appears.

We have, in order to further study this question, undertaken a second study and these are the first reports of this second study, not yet published.

On January 1, 1967, we registered every single union insulation worker in the United States. There were 17,800 members of this union on that date, and Dr. Hammond and I have followed them from January 1, 1967 through December 31, 1971. These are the results found in that study.

Whereas there should have been 805 deaths among them, given their ages, there actually were almost 1,100. And, once again, the lung cancer death rate was extraordinarily increased. Whereas there should have been 44 expected, 213 bronchogenic carcinomas actually occurred. There should have been no deaths of mesothelioma; there were 77. There should have been a small number of gastro-intestinal deaths; there were 3 times as many as expected. And,

of course, there should have been no deaths of asbestosis, which doesn't occur in the general population, and there were 78.

When we analyze these deaths by duration from onset of work, among these 17,800 men, we found that in the group less than 15 years from onset of exposure, there was no significant increase in either lung cancer or mesothelioma. We did not see one mesothelioma in less than 15 years from onset of work, although we were observing almost 10,000 men in this category. On the other hand, once the 15 years passed, we found that the ratio of expected to observed deaths was 5, 6 and 7 times; now the mesothelioma deaths began to be seen.

We have also looked to see whether this was so in an asbestos products factory making insulation and many other products. To study this, we made a list of every single production worker in this plant on January 1, 1959 who had on that date worked at least 20 years. We have followed this group, 689 people, through December 31, 1971. And, once again, we have found that whereas there should have been 8 or 9 deaths of respiratory cancer, there were 35; of these 27 were lung cancers, 8 were pleural mesotheliomas, 7 were peritoneal mesotheliomas, and the same increase, a modest increase, in gastro-intestinal cancer and asbestosis. Finally, to try to get some sense out of the duration from onset versus the actual exposure, because most of the men in the three

cohorts that I have just reported to you had worked continuously and in one group, we were able to find a very significant difference in the amounts of work. This was a factory which opened in June, 1941, to make insulation for the U. S. Navy. Nine hundred thirty-three men were employed for the first time, for '41 to '45. The factory closed in 1954. Some 11 men worked for only 1 day. They didn't like it. Some worked until the plant closed in '54. Following this group of 933 people (it is a darn good plant - they got their Army-Navy "E"), we have found that in these 933 men there should have been 300 deaths by the end of '71; there actually were 484. And, once again - it's almost monotonous - same 7 times increase in lung cancer, pleural mesothelioma, peritoneal mesothelioma, gastro-intestinal cancer and asbestosis. But, of these men, approximately one-third worked for less than 3 months. One-third worked from 3 to 11 months, and one-third worked for more than a year. We have found that although the group with more than a year of exposure had a very significantly greater ratio of expected to observed deaths, 11 times as many lung cancers as expected, still even less than 3 months of work was enough to give a 3 or 4 times increase in bronchogenic carcinoma provided they lived long enough - provided you looked at them 30 years later. Now all of these data indicate that what we are seeing now, the current mortality experience, is the result of the inadequately controlled exposures of the past. However, the

problem is with this long latent period, how you find out what the exposures of the past were to relate them to the epidemiological evidence of disease at the present time. Unfortunately, in the United States we are at a loss for this. There are no sets of dust counts and resulting disease observations, from the first case reported in the United States in 1930, to permit judgment here of acceptable levels. There are two approaches. Both have been used. First of all, if there were measured dust levels 20 years ago, and if we could identify the population exposed to those levels 20 years ago, we could make some correlations. One, of course, would be asbestosis.

The other approach has been to take dust measurements now and get some sense of how these measurements now relate to what existed in practice 20, 30 and 40 years ago. This has been done but there are significant differences with this technique. First of all, in the past very few dust counts were done. The current techniques, membrane filter methods, phase microscopy, etc. were not used at that time. For example, the ^{Dresden} ~~Dreesen~~ study. Also at that time all particles were counted rather than only fibers, and as we try to convert particles per cubic foot to fibers per cc, the results are unfortunately uncertain. We have, on the other hand, had some measurements of what exists today in the construction industry with the use of insulation material, and we have found in using fibers per cc, that in general somewhere around 5 to 10 fibers

per cc exists in these environments. We don't really know whether this was true 20 years ago. There are 4 studies that provide evidence. First, there is the original study of Dr. Fleischer and his colleagues in the U. S. Navy shipyards, published in '46 and done in '44 and '45. A different instrument was used and a time weighted average of 25 fibers per cc could be obtained, but other glass fibers and so forth were included. Murphy and Ferris and their colleagues have looked at shipyard data published in '68 and '71 and they came to somewhere around 10 fibers per cc. Balzer and Cooper, in San Francisco insulation workers, have come to around 6 fibers per cc. And in our laboratories, looking at construction work in shipyards throughout the United States, we have found exactly what Balzer and Cooper have found; somewhere around 6 fibers per cc. But this is reconstruction. And we don't really know that this existed 20 years ago. While this is the dilemma that we faced, we thought that 6 fibers per cc, somewhere around that, 10 fibers per cc, existed in the past but we weren't sure. We know what the disease is; I've just shown it to you. Therefore, we were all very much impressed and grateful when the British Occupational Hygiene Society gave us the answer.

In 1968, the British Occupational Hygiene Society published a report stating that 4 fibers per cc or 2 fibers per cc would likely be safe. The basis for their report was the report of their sub-committee on asbestos published in the Annals of Occupational Hygiene

1966. It read, "The results of recent work, the study of John F. Knox and Steven Holmes, have been made available to the committee by the asbestos industry and it is on these recent results that the new standard is primarily based." What they came to is that it would be safe to have 100 fiber years, such as 2 fibers per cc for 50 years or 4 fibers per cc for 25 years. The work of Dr. Knox and Dr. Holmes (they were both members of this committee) was valid. Dr. Knox was Chief Medical Officer of Turner Brothers Asbestos Company in Rochdale and Dr. Holmes was their Industrial Hygiene Scientist.

The bases for these data were then published by the British Occupational Hygiene Society. Dr. Knox had x-rayed all production workers at Turner Brothers Asbestos Company in Rochdale as of June 30, 1966. There were 931 workers in this plant, of whom a large number had been more than 10 years in their employ. They had done dust counts, very good dust counts, from 1951 on, and therefore we had a population, for the first time, where dust counts could be related to clinical evidence of disease. In the departments where the dust counts were 10 to 15 fibers per cc, of the large number of workers there employed, 7 showed some x-ray abnormality which could have been asbestosis. On the other hand, of 70 or 80 people working at 3 to 4 fibers per cc, only one was reported to have an abnormal x-ray. This being so, the subcommittee, which itself did not review the data but reviewed the numbers

submitted to it, then reviewed the data given it and decided that since only one worker exposed for 20 years or more at 4 fibers per cc showed evidence of asbestosis, that half that level, 2 fibers per cc, would be prudent and safe. And if I were on that committee, I would have come to exactly the same conclusion.

When these data, this conclusion and this recommendation reached the United States, it did, as I say, impress all of us. So much so that on February 28, 1969, Howard Ayer sent a letter, containing this statement, to 28 asbestos companies and 50 state health departments: "From these standards (that is, the British Occupational Hygiene Society recommended standards), one would gather that concentration below 2 fibers per cc are probably safe." A fair statement. Indeed when the criteria document was submitted by NIOSH last year, 1972, it stated, "The development of a standard for asbestos dust in Great Britain and the evaluation made by the British Occupational Hygiene Society Subcommittee on Hygiene Standards for Asbestos, which considered data to reduce the risk of asbestosis, was given great weight in the development of this asbestos standard." That is the origin of the 2 fiber per cc standard. Two fibers per cc obviously is 2 million fibers per cubic meter. A working man inhales somewhere around 8 to 10 cubic meters of air in an eight-hour working day, which means that we have told him that he can safely inhale with an uncertain, unmeasured retention, somewhere around 20 million fibers longer than 5 microns

in an eight-hour day. As Mr. Dement has just told us, anywhere from 20 to 99% of fibers are smaller than 5 microns, and therefore the total number of fibers of all sizes is somewhere around 20 to 40 billion.

Now some new data has recently appeared which I thought might be of interest to you. In 1968, Dr. Knox retired as Chief Medical Officer of Turner Brothers and was replaced by a young South African, Dr. Hilton Lewinsohn. Dr. Lewinsohn has re-x-rayed all of the workers at Rochdale in December, 1970. The populations, however, were not entirely identical. Dr. Knox had examined only men in the scheduled areas - that is, the production areas, 913 - whereas Dr. Lewinsohn included all employees, including the office personnel, and therefore had a larger number. To some extent you could say that this diluted the population, but I think it was a wise thing to do. Also, some of the people working there in 1966 were no longer employed; obviously they had retired or died and some new workers had been added. The very large majority, of course, were in both surveys. Dr. Lewinsohn reported the results of his December 1970 re-x-ray of the Rochdale personnel in the Royal Society of Health Journals in 1972. Dr. Lewinsohn's data are on this slide.

It demonstrates that of those with, let's say, 20 to 29 years from onset of exposure, of 114 x-rayed, 51 had abnormal x-rays. All together, total abnormalities were 55% of all people who had

reached 20 to 29 years from onset, and of these, actual pulmonary fibrosis was seen in 21 out of 114. Of those who had reached 30 to 39 years from onset, 78% had abnormal x-rays, and 17 out of the 42 had actual pulmonary fibrosis. Of those 40 to 49 years, there was too small a number to show.

When we put these two sets of data together, we can find that as of 400 fiber years, Dr. Knox had found, in reading the films, that somewhere around 7 or 8% had abnormal x-rays, whereas in the same population a different film reader had found that somewhere between 40 to 50 % had abnormal x-rays. There is obviously a considerable discrepancy in the two sets of film readings. Now when these discrepancies were discussed with our British colleagues, last October in Lyon, France, Dr. Holmes made the following statement: "Thus, the information, although the best available at the time, was, to say the least, scanty for the purpose. And some of us who were associated with it have become increasingly concerned with the authority with which it has become invested in the international field (meaning the United States). The time has therefore probably arrived for an up-to-date reappraisal of the original data." I have communicated with Dr. Lewinsohn in the last three weeks and am informed that there are definite plans to undertake this review in the future. It has not yet been done.

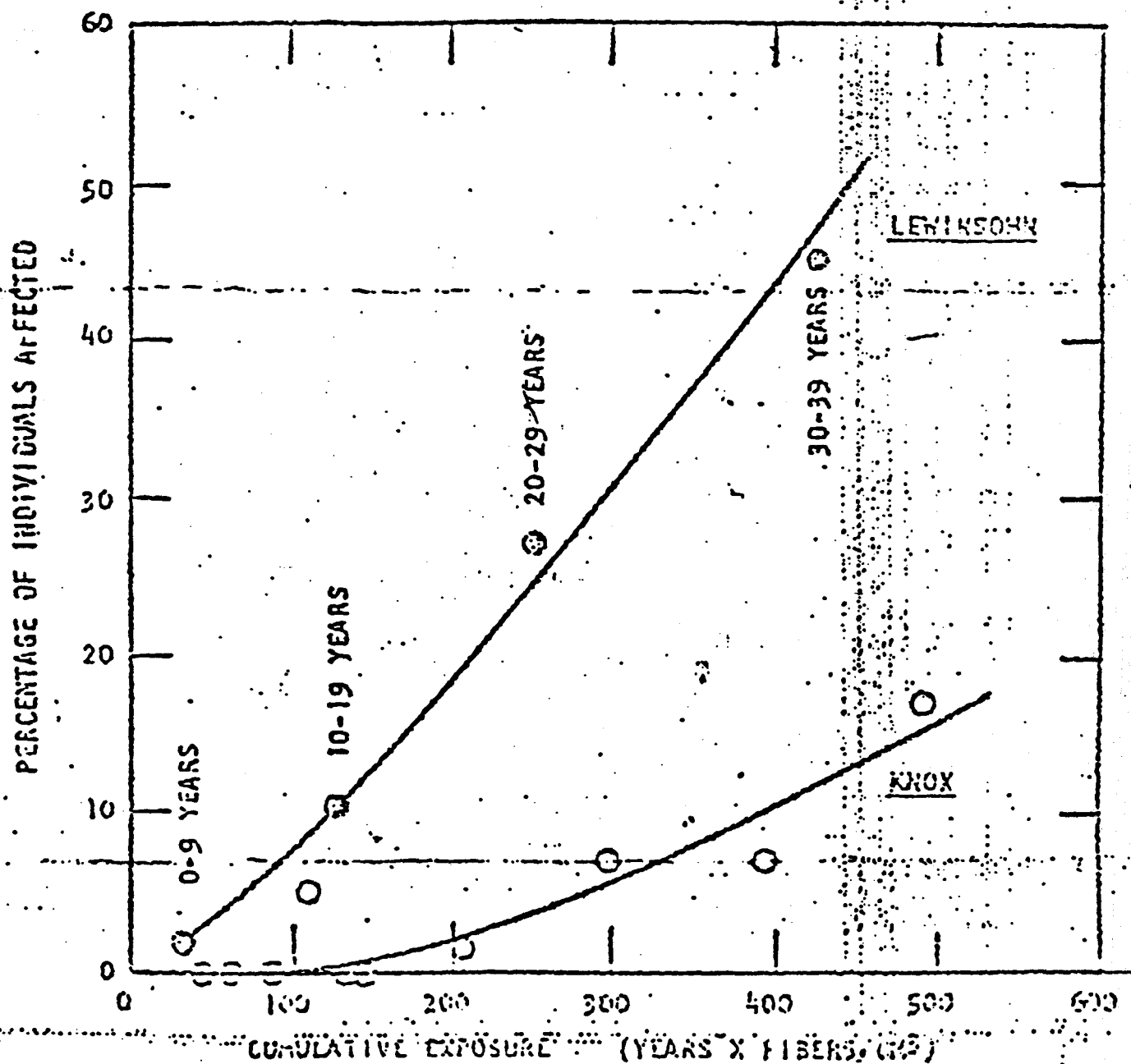
In conclusion, then, as a result of these new data from Turner Brothers Asbestos Company, we can say that if the 1972 report of

the Chief Medical Officer of Turner Brothers Asbestos Company correctly represents the disease incidence associated with prior levels of asbestos exposure in the company, then the asbestos standard of the Department of Labor places U. S. asbestos workers at serious risk of irreversible, often fatal disease, and in my opinion, it should immediately be reconsidered.

Unfortunately, then, I feel that we are in the same dilemma still - after 50 years - that Dr. Cooke found himself in 1923.

Thank you very much.

PREVALENCE OF X-RAY EVIDENCE OF ASBESTOSIS.
IN ASBESTOS TEXTILE WORKERS



QUESTION AND ANSWER SESSION
FOLLOWING DR. SELIKOFF'S PRESENTATION

- Q. In the asbestos products factory in which men employed for only three months were found to have a significant increase in lung cancer, were smoking histories obtained?

(Dr. Selikoff)

- A. The answer is "Yes", and those data are now being analyzed, but I would be amazed if we found any lung cancers or any more than a trivial number among those who did not smoke cigarettes. We have failed to see any significant increase in the incidence of lung cancer among asbestos workers who do not smoke cigarettes. We have been following 2,066 non-smoking insulation workers from January 1967 through 1971 and we have only seen two lung cancers among them. On the other hand, of 9,590 cigarette smoking insulation workers, we have found 145 lung cancers whereas only some 30 were expected. Bronchogenic carcinoma is almost limited to cigarette smoking asbestos workers. This is not true of peritoneal mesothelioma; it is not true of asbestosis; and it is probably, although our data is too scant yet to be sure, it is probably not true of pleural mesothelioma. But of lung cancer - there is no question - this will not occur by and large among asbestos workers who do not smoke cigarettes.

- Q. I would like to ask Dr. Selikoff if he thinks the new standard should include both a total dust count - if there is a new standard - both a total dust count and a per cent fiber count. At least in our experience, we seem to vary comparatively. A standard set on one rather than the other could be quite misleading.

- A. Ray Murphy has asked me a very embarrassing question. He asked me to give an opinion where I can't give it even on fiber counts. He now asked me whether I would do it on total counts rather than just fibers. The answer is, "I don't know." But I'm not very impressed (if I can upstage Duncan Holiday a bit who is going to review this tomorrow morning) - we're not very impressed with asbestos standards which rely upon dust counts being done. May I give you some data? Very depressing data, if you think these are depressing. On June 7, 1972, the asbestos standard came into effect. And it said that every company using asbestos had to do at least one dust count, one dust count, in the next six months. That is, by December 6. We, on December 7, did a survey of insulation workers in the United States and we now have some 5,000 ccd replies. Each man was asked about

the dust counts that he personally had seen in the six months when every company was supposed to do a dust count. In the United States insulation industry, fewer than 3% of the insulation workers saw even one dust count done from June 7 to December 6, 1972. Obviously then, dependence upon dust counting as a means of control in this unusual industry has many deficiencies.

- Q. It is really one question in two parts, perhaps. The fibers at the Turner Asbestos Company (Knox-Holmes study), what were these and could you comment, would you comment on the possible differences that you think may be between crocidolite and chrysotile and amosite?
- A. The fibers in Turner Brothers, since it was a textile operation, were largely chrysotile. We can give you no information concerning crocidolite because none of the workers studied by us had any crocidolite exposure. They had chrysotile exposure and many of them had amosite exposure. Indeed, in the plants, in the factory making the Navy insulation, this was almost entirely amosite. So our data apply only to amosite and to chrysotile and, unfortunately, I can give you no information on the relative hazard of crocidolite. I would suggest, however, that if crocidolite is more dangerous than amosite or chrysotile, it is going to be almost as bad as bis-chloromethylether. (Laughter)
- Q. Dr. Selikoff, are you aware of a particular type of asbestos fiber mined in Ontario called Headman? fibers which have been found to be relatively inert, by Paul Gross and others?
- A. I presume that you mean the fibers mined in Quebec, not Ontario. (No, mined in Headman Mine in Ontario.) No, I do not know these particular fibers and I would be delighted to know of an asbestos fiber which is comparatively inert.
- Q. They claim - and Paul Gross and others have done experimental work - and they have found that this particular fiber which is chrysotile has been, in experimental work, found to be inert.
- A. Well, I would be delighted to see those, indeed I'd be delighted to see those in industrial use.

Thank you, Dr. Selikoff.

ECL/AN
12.7.73.

Professor Irving J. Selikoff,
Mount Sinai School of Medicine,
Fifth Avenue and 110th Street,
NEW YORK NY 10029,
U.S.A.

Dear Professor Selikoff,

We have recently received a transcript of a tape recording of your address to the American Industrial Hygiene Conference in Boston on 21st May, 1973 which was sent to us because of its references to our work. If the copy enclosed herewith is a true representation of that address and the graph shown by you, we must tell you that we find your assumptions and conclusions to be extremely misleading.

The case made at the end of your paper appears to rest on the fact that the data which formed the basis of our Hygiene Standard¹ was scanty. You then attempt to extract data from the Figures in ECL's publication² in order to show a discrepancy between the original data and these Figures. We are astonished at your having communicated the contents of a private correspondence to the meeting which you addressed without having obtained our agreement. We are also very displeased at the manner in which you have used the information we provided in order to launch what seems to us to be a political campaign in the USA aimed at discrediting our Hygiene Standard for asbestos. We cannot accept the implication that the ECL publication allows doubt to be cast on the validity of the BCES Standard and are of the opinion that such a conclusion is unwarranted.

Comparisons such as those you have made between the Knox data and the ECL data are invalid for the following reasons :-

1. The paper by ECL was not intended to justify or verify the BCES Standard, and no reference is made in this paper to environmental dust measurements. The results presented in ECL's paper were preliminary results and in the discussion section it was clearly stated that 'from the information currently available only tentative opinions can be expressed'. We are

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surprised therefore that you have been able to read far more into the incomplete picture presented than the author was able to do or even intended to do.

2. The populations reported by Knox and HCL were not identical nor were 'the very large majority' of asbestos workers 'common to both surveys', as you suggested. 'Office personnel' were not included. The population studied by ECL consisted of workers in the recognised asbestos areas, together with maintenance, quality control and research staff.
3. Knox reported on 290 men (not 913 as stated by you) with at least 10 years exposure since 1st January, 1933, when the Asbestos Industry Regulations 1931 took effect, and who were still in the Company's employ on the 30th June, 1966. ECL on the other hand has analysed the X-rays of a much larger population (1,287) as of November, 1970 irrespective of date of first employment; some were in fact first employed before 1st January, 1933.
4. Knox sub-divided his population according to actual cumulative exposure whereas ECL's was sub-divided by decades elapsed since first exposure, irrespective of the actual number of years exposed. In view of these basic differences and others which we will point out in due course, we fail to see how you derived the relationship between cumulative exposure and percentage of individuals affected on the graph which you showed (copy attached). The heading on the graph is obviously misleading as ECL at no stage in his report equated the radiological changes with 'asbestosis'.
5. Because it was possible to estimate dust exposures for the Knox population, the BOHS Sub-Committee were able to arrive at figures for cumulative exposure expressed in fibre years/cc. No such exercise was ever attempted by ECL. This is a further invalidation of the graph since you have attempted to relate radiological features described by ECL to what we can only regard as speculative exposure data.
6. Knox only recorded X-ray changes as being present which are possibly asbestotic, after taking the whole clinical picture into consideration. 'Radiological changes considered significant included those of increased general opacity of the lower lobes, blurring of the cardiac outline, pleural thickening and adhesions'.¹

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HCL on the other hand attempted to analyse his radiological findings according to the UICC/ILO classification, an entirely different set of criteria, and took no account of the clinical picture as a whole.

7. The BOES sub-committee decided that basal rates were the 'key symptom' since all the men with X-ray changes, by the Knox criteria, had basal rates, but some with basal rates had no X-ray changes.¹ HCL points out in his paper that he has not yet attempted to correlate physical signs and X-ray changes.

8. We are obliged to point out that the use of the words 'pulmonary fibrosis' by HCL in his paper may have misled you, as they were used to describe small opacities which prior to the advent of the UICC/ILO classification may have been regarded as consistent with fibrotic changes. The use of this term does not allow for sub-division into profusion and type of opacity as is now possible with the UICC/ILO classification. For simplicity in presentation, HCL grouped all cases showing evidence of these changes together, irrespective of severity. Knox only included X-ray changes consistent with the clinical diagnosis of asbestosis. It is not possible to say, from the data that HCL has compiled, how many persons would fulfil the criteria for a clinical diagnosis of asbestosis, and to attempt to illustrate these two very different sets of data on the same graph is simply not permissible. The clinical significance of the radiological changes observed cannot as yet be defined.

You appear to have ignored completely the fact that the number of cases of asbestosis in 1968 among 355 employees (men and women) exposed for 10 years or more since 1933 in Rochdale and still employed, was 1.12% (i.e. 4 cases)³ as compared with Merewether's finding of 44.4% (i.e. 59 cases) among 133 workers exposed 10 years or more in the same industry in 1929.³ This lower incidence has occurred in spite of the fact that the Pneumoconiosis Medical Panels are better equipped to diagnose asbestosis than Merewether was. In addition, during the years immediately following 1933, there still was substantially greater exposure in certain jobs than in more recent times.

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In view of your comments and our previous correspondence we should like to remind you that we are in the process of reviewing and bringing up to date all the available data, and hope to present it for publication in the near future.

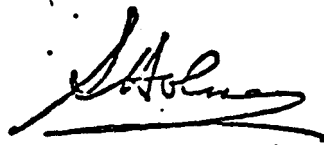
Dr. Holmes' remarks at Lyon were never intended to suggest that the BOES Standard was not stringent enough. The BOES Standard is 100 fibre years/cc and therefore application of a 2 f/cc limit for periods of less than 50 years has the effect of providing an ample safety margin. The BOES Sub-committee, of which we are both members, recently reviewed the Hygiene Standards for Chrysotile Asbestos⁴ in the light of the latest available data and recommended that no change be made at the present time.

We are forwarding a copy of this letter to the President of the American Industrial Hygiene Association, the Chairman of the ACGIH, the Director of NIOSH and also the Asbestos Information Association of North America in view of their obvious interest in this important subject.

Yours sincerely,



H.C. LEWINSOHN, M.B. B.Ch. D.I.H. :
Chief Medical Officer.



S. HOLMES,
Chief Health Physicist.

Encl:

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