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Johns-Manville

Internal Correspondence

To: Paul Kotin, M. D. Date: July 23, 1975

From: George W. Wright, M. D.

Copies: file & C

Subject: MEETING ORGANIZED BY DR. JOHN FINKLEA OF NIOSH TO CONSIDER A REPORT BY DR. SELIKOFF AND HIS ASSOCIATES ON THEIR PRELIMINARY FINDINGS WITH RESPECT TO THE ENVIRONMENT AND THE HEALTH OF WORKERS EMPLOYED IN THE REPLACEMENT OF BRAKE LININGS.

The meeting was held in the afternoon of July 21, and those attending are indicated on the attached exhibit. The agenda is also attached.

Dr. Selikoff opened the meeting by stating that their interest in the health of brake lining repair workers arose because of a report of several cases of mesothelioma occurring in auto repair workers. There was some confusion in my mind at first as to whether this was solely from the New York area or a larger population. It later developed that there were 4 and possibly 5 cases that had come to his attention from workers in this trade throughout the United States. He estimated that at least one million men might be thus employed and exposed to asbestos. He repeated several times that in his judgement it was not possible at this point in the development of information to say whether or not this represented an excess risk of mesothelioma but that he thought the observation merited further exploration. He also indicated that he had reviewed this problem with appropriate people at the Ford Motor Company. He then asked Dr. Langer to discuss the nature of the material found in the dust that accumulates in brake drums and which is by customary practice blown off the surface and into the surrounding air at the time that work on the brake drums is being carried out.

Langer opened his comments by saying that others had reported that 30 percent of the asbestos in brake lining is converted by high heat to Forsterite. He indicated that "modifiers" present prevented the formation of Forsterite and he has found Forsterite in brake drum dust only in one instance. The only person I know of who says that the asbestos is converted to Forsterite is Paul Gross. Others say that it is converted to an amorphous and essentially non-fibrous material. Dr. Langer indicated that the residue was not pristine chrysotile fiber but he did not speak about amorphous material. Langer, in addition, said that he

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found pure asbestos fibers. This too has been reported by others in quantities of anything from 1 to 3 percent of the residual dust. He then asked Dr. Rohl to discuss the fiber counts in the dust that was obtained.

It should be indicated that brake drum dust was obtained from cars in taxi repair shops, municipal bus repair shops and I believe some other similar workplaces. Dr. Rohl indicated that studies of the material blown from, or obtained from the brake drum contained fibers in large numbers and these were chrysotile. None of the fibers were more than 0.5 microns long and the bulk were under 0.3 microns. In diameter they varied from 250 to 500 angstroms. On the other hand, he reported that the fiber count varied from 0.4 to 29.0 fibers per ml at the worker breathing zone level where brakes were being cleaned by air jet. It was my understanding that these were fibers that were countable under the OSHA regulations and therefore would be visible with the light microscope and longer than 5 microns. It was not entirely clear to me whether these optically recognizable fibers actually came from the brake drum dust or not. If they did, I have a conflict in reconciling his electron microscope study with his light microscope studies. Rohl also reported that in brushing the brake drums rather than blowing them off the fiber count (OSHA criteria) varied from 1 to 4 per ml, during grinding the fiber count varied between 1 and 5. Most important was the finding that when men were bevelling the brake linings by grinding the counts varied between 20 and 75 fibers per ml. During punching, riveting or sweeping the fiber count varied between 2 and 4 fibers per ml. It is my understanding that other kinds of grinding of the brake lining goes on in the fitting process. Ed Fenner tells me that they do something called "arcing" and this involves direct grinding of the brake lining and is a very dusty job. Rohl also showed data to indicate that there was a very rapid fall off in the level of count as one moved away from the source so that fiber counts 10 or 20 feet more away from the source of origin were lower than 1 fiber per ml and in general were in the neighborhood of 0.2. On some questioning it developed that these shops were what would be considered to be very well run and therefore tend to have lower dust counts than would the average smaller shop. In the pictures that were shown and the comments that were made there was no evidence to indicate that any sort of dust exhaust or suppression systems were in use. I was surprised that no direct referrence to extensive data relative to brake drum dust by Lynch, by Jacko et al, by Anderson et al, and by Johns-Manville as well as a small study of the work place during brake maintenance by Hickish and Knight was not made by either Langer or Rohl.

At the request of the Unions Dr. Selikoff also did a preliminary examination of 104 workers currently employed in the places where the dust counts had been made. The men were not selected on the basis of duration or intensity of exposure, some working only once a week where they would be exposed to brake lining operations but the majority of them had worked for between 10 and 20 years. Of the 104, 29 had no direct exposure to asbestos or pulmonary disease but worked in the same area. The data was presented so rapidly that I could not follow all of it but in part at least it is as follows: Of the 64 men known to have direct shop exposure 16 had chest films that were read as category "s or t" small irregular densities with a profusion of 1/0 or more. No controls for the reading were spoken of and I assume that these were read on one occasion by one person. The inference was that these readings constituted "disease" attributable to the exposure. There was no breakdown of how many had profusion readings in excess of 1/0 and thus I have no knowledge about the extent of the changes. Later in the meeting I raised the question of the interpretation of this kind of information. In the absence of controls, there is no way to estimate the influence of bias in this kind of reading. It is obvious that the reader knew the films came from an exposed group and the tendency under these circumstances is to find something in the way of an abnormality. The best way to counteract this bias is to have a group of controls dispersed among the exposed group and have the entire set read "blind". In the absence of this there are two other ways of going at it, neither of which were utilized in these readings. The first of these is to have several readers look at the films independently. The accepted reading is then the one that has the majority of the readers designation. For example, if the majority of the readers class a film as a 1/1 and a minority classify it as a 0/1 the ultimate reading used is 1/1. Some use reading by "consensus" but this is a poor and unacceptable method because usually there is one dominant reader who is able to persuade the others as to what he thinks it is. The other way of going at it is to know what the exposure history is and be able to rank the observed population interms of intensity of exposure. The films are then read with no knowledge of the intensity of the exposure and the distribution of readings has slightly less bias than otherwise because the least exposure serves as a control for the more heavily exposed. This method is not as good as having multiple readers or actual control films interspersed amongst the films of the population under study. With the information I have I don't know how to interpret their x-ray findings other than to say that the inference that came from them was that all 16 of these

individuals had disease. The UICC classification is not a diagnostic classification in any sense of the word and serves only an epidemiological purpose. I know of no basis on which one can conclude that people with a reading of 1/0 have actual disease. Additional data was presented along the lines of pulmonary function measurements. The data showed a substantial percentage of those who were directly exposed to have what was called restrictive disease. I don't know what criteria were used for arriving at this designation and hence can't evaluate the finding. If it was based on actually measured total lung volumes, rather than just on vital capacity, then the data has useful meaning. The strong suggestion made by the Sinai group was that in these 104 individuals they had demonstrated the presence of asbestos related disease. Maybe this was not their intention but in contrast to Dr. Selikoff's statement that 4 cases of mesothelioma did not constitute sufficient evidence to incriminate the exposure, nothing was said to the effect that the x-ray and pulmonary function measurements may not have been very sound evidence of disease.

On the basis of these reports from Dr. Selikoff's laboratory several suggestions were forthcoming. First of all there was an inference that Langer's studies had a strong implication that a public health hazard might exist. At the very least the implication both from Langer and from Rohl was that the short fibers found in large numbers in the dust from the brake lining posed a potential threat to health of great magnitude. In my comments I pointed out that there was growing experimental evidence that fibers of lengths under 10 micron did not produce disease in terms of pulmonary fibrosis and very likely did not produce mesothelioma. On this basis I would think the presence of the very short fibers has little or no biological significance until proven otherwise. The longer fibers are quite another matter since they are capable of producing asbestos associated biological effects. In my comments I stated that the observation showing fiber concentrations of 20 to 70 fibers per milliliter in the bevelling operation did not surprise me. In other settings, the grinding of the surface of new brake lining is a known quite dusty process. Depending upon the accumulated duration of exposure in this kind of operation one had every reason to expect that in those who have been doing this for a substantial length of time there would be a risk of pulmonary fibrosis and the other kinds of asbestos associated disease. Even though this might occur only once or twice a week, these high intermittent exposures might have an adverse effect. Since no data in the way of cummulated dosage was presented there is no way in which the risk can be evaluated.

It was quite clear that the Union representatives expect a study of this category of workers to be made by

Dr. Selikoff's group. Mr. Cefalo of the Machinists Union made the blunt statement that if funds were not available he could promise that they could become available. Whether this meant from Union sources or from the influence of the Unions on the Government was not clearly stated .

As you know I had to leave before the meeting ended and I don't know what the upshot of the matter was. I asked myself the question; what is to be gained by mounting a complete epidemiologic study based on observation of these workers and the environment of their workplace. If the question is solely whether or not asbestos has the propensity to produce a biological reaction along the lines we already know I would say that making this study would be a very poor use of Dr. Selikoff's and his co-workers time and of expenditure of tax payers money. We already have that kind of information and nothing in the presentation of the afternoon indicated that the exposure in this group was appreciably different from that which has been reported in other occupational groups. If adequate information can be obtained then two questions might be approached. There is the implication fostered primarily by Dr. Selikoff's group that short asbestos fibers (<5 μ long) produce all of the known biological effects in workers substantially exposed. They have taken this attitude on the basis primarily that short fibers are found in the lungs at autopsy and therefore must be the cause of the disease. On the basis of animal experimentation others do not accept the hypothesis that fibers shorter than 5 to 10 microns in length have biological capacity. There appears to be a reluctance on the part of the Selikoff group to accept the inferences of the experimental animal data. The difficulty of looking to human exposure for resolution of the question is obvious. Those groups exposed ocupationally, at least in studies up to the present, are exposed to a mixture of long and short fibers and there is thus no way of separating the affects of short versus those caused by long fiber. Not even autopsy data will help in this respect since long fibers are subject to shortening by fracture with a passage of time. If a group exposed only to the dust from the brake drums as described by Langer and Rohl could be separated out and looked at in terms of their mortality and morbidity experience then a study of the brake lining workers population would be emminently worth while. If this cannot be done and the exposures are mixed, the population will be of no use for examining the effects of short fibers versus long fibers. Another question might be looked at by studying this group. There still remains a question in the minds of some as to whether or not the ? fiber per cc level in the ambient air (OSHA standard) is adequate to protect the workers who are exposed throughout a lifetime. If Dr. Selikoff's group can reconstruct the exposure histories of these workers in a manner that will give a ranking of

the workers in terms of intensity and duration of exposure then a study of the relationship between this ranking and the biological effects would be useful. If this cannot be done and if the population cannot be divided into those exposed solely to short fibers and those exposed to either solely long fibers or a combination of short and long fibers then I see nothing to be learned from an extensive study of this population that is not already known. To be sure, the employees in this trade might want an assessment of the potential hazards and this kind of information might be used in persuading both the employers and employees to create a safe work place. I think this can be done by utilizing knowledge already existing with respect to biological effects and relating it to a thorough survey of the environment with respect to the level of asbestos fibers in the various kinds of jobs being done in this category of employment.

It seems to me that Dr. Selikoff's group constitutes a resource that should husband its time and facilities. I would hope they would not embark on a study except in those areas where something useful could be obtained. I think that a thorough investigation into the environment could be thought of as useful study on the basis of what is now known. In fact, under the regulations set forth by OSHA the employers should already have developed that information. The regulations require that when employees handle materials in processes through which they might be exposed to the inhalation of asbestos fibers certain environmental and physical examination requirements must be satisfied. I would think this would be the most useful avenue to pursue since the requirements under OSHA are quite clear cut. I don't know at this time what can be anticipated in the way of reconstructing exposure histories or in dividing the population into the two groups with the respect to length of fiber. On the basis of past experience I would not be very hopeful that this could be accomplished with a degree of accuracy permitting any conclusions from a biological survey to be drawn.

Several other points arose from this meeting that seemed to me to be important. First of all, insofar as I can learn, labeling of brake lining materials in a way that would inform the user and the worker of the inherent hazard related to the various ways in which the material might be handled has not been a common practice if done at all. I would hope this would be corrected at once. Mr. Sam Meyers of the United Auto Workers Local 259, showed posters his group have developed, warning of the possible effects of brake lining handling on health. He also made constructive remarks during the meeting. It seems clear that some mechanism must be found for alerting all workers in this trade to the potential hazards and the need to join

with the employers in finding ways to work safely. An educational program through the Unions as well as the employers should be mounted as quickly as possible. Since this trade may involve only a few people in a given place and presumably is not totally unionized I believe this may be a matter of considerable magnitude. The discussions of this particular afternoon demonstrate as well as anything I know the obligation on the part of manufacturers to know where their products containing potentially toxic material go and the way in which they are used. In those circumstances where a hazard can be created by the use of the product a means of informing the worker must be found. At the very least this should be labelling and in all probability it should be a joint educational effort on the part of the workers organizations and the employers and the producers. Unfortunately labelling is often times either ignored or loses its impact with the passage of time. For this reason more persistent and active methods should be found for accomplishing the purpose. In view of the nature of the problem and the fact that the current OSHA regulations should have brought it to the fore I was surprised that no representative of OSHA was present. Representatives of EPA were present even though their concern would be expected to be less at this point than should be true for OSHA.

I regret that I was unable to attend the meeting beyond 3:40. I will call Dr. Finklea and see if he can give me more information and if so I will transmit this to you.

George W. Wright, M. D.
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