FILE NAME: Uniroyal (UNIR)

DATE: 1964 Nov 17

DOC#: UNIR020

DOCUMENT DESCRIPTION: Letter to US Rubber Co. from John G. Wells RE Conference on 'Biological Effects of Asbestosis'

JOHN G. WELLS, M. D.
41 JEFFERSON STREET
NEWNAN, GEORGIA
TELEPHONE AL 3-0611

12 November 1964

Mr. A.C. Link
United States Rubber Company
Hogansville, Georgia

Dear Mr. Link:

I should like to thank you and the United States Rubber Company for the opportunity and very great pleasure of attending the recent conference on "Biological Effects of Asbestosis" on October 19-21, 1964.

This conference was sponsored by the New York Academy of Sciences; and was truly an international conference, attended by active workers in the field of Asbestos from the British Isles, Finland, Germany, Italy, The Union of South Africa, Canada and several areas in the United States.

The majority of the discussants traveled from the British Isles and South Africa where interest and continuing investigations had been initiated by the signal report of Merewether and Price in 1930, which established the occupational origin of asbestosis and made this disease a "certifiable" and compensatable disease.

The recent conference on biological aspects of asbestosis can be divided into two parts; the first relating to the broad aspects of asbestos and asbestos disease; and the second, to the seemingly striking increase in incidence of bronchogenic carcinoma and mesotheliomata of pleura and peritoneum. These neoplasic disease have been found with increasing frequency in individuals whose initial exposure to Asbestos dust has exceeded twenty years.

Rather impressive data to demonstrate this development were presented by Selikoff, Smither, Buchanan and others. However, no evidence was given which was of correlative significance in regards to the type of asbestos dust; or to the nature, amount and conditions of initial and subsequent exposure.

Of perhaps even greater significance in this regard was the report of Dr. John Knox of Turner Brothers Limited, who compared two groups of people with twenty years or more exposure gained before and after 1933.

In the first group, there was a very significant increase in the number of deaths from all causes; all neoplasms, and cancer of the lung and pleura.

However, in the group of the twenty-years-exposure men studied after 1933, the number of observed deaths is only slightly greater than the expected mortality rate; and the number of deaths due to all neoplasms, and cancer of the lung and pleura, is in fact, less than that which was anticipated for the population at large.

No conclusions were drawn from these observations by Dr. Knox; but it was pointed out both by him and Mr. Hill, of the same company, that following 1933, extensive and continuing efforts at dust control have been employed in their factory.

A great amount of experimental work has been done both in this country and abroad purporting to demonstrate the ability of asbestos fibers to produce bronchogenic carcinoma and mesotheliomata of the pleura.

Of some interest is the work of Smith and Miller at Fairleigh Dickinson University, Madison, New Jersey, who, using chrysotile and amosite fibers, inoculated hamsters both intratracheally and intrapleurally with massive doses of the fibers. They did not report one case of neoplasia by the intratracheal route; but did report numerous cases of neoplasia following the intrapleural route of injection, which is of course, not experienced by human beings.

In summary, in regards to neoplasia, the reported findings related largely to a group of workers in various asbestos industries, including large bodies of insulation workers (non-textile workers) whose exposure occurred several decades ago; and prior to a time when increasing awareness of a potential occupational hazard generated a continuing interest and activity towards effective dust control.

The first half of the conference pertained largely to the descriptions of Asbestos disease in various parts of the world; the experimental production of asbestosis; and the efforts of various industries and fact findings bodies--most particularly in the British Isles and South Africa--towards investigating the gross anatomic, microscopic, radiologic, and pulmonary functional aspects of asbestos disease.

It was demonstrated that not only did microscopic particles of asbestos dust of various size enter into the lungs and produce asbestos bodies and subsequent pulmonary fibrotic changes; but it was also demonstrated most remarkably by Dr. Davies of the Asbestos Research Council at the University of Cambridge in England, using electron microscopy and selective iron staining dyes, that ultra-microscopic particles were also incorporated into the lung and picked up by the lung phagocytes. This particular work is being carried on actively and the results in terms of production of asbestos disease are not at present known. No conclusion was drawn, but one must entertain the speculation that asbestos fibers of not only microscopic but of also ultra-microscopic size may be implicated in the formation of pulmonary asbestosis.

A most interesting study was reported by Kiviluoto of Finland who described a rather peculiar type of pleural calcification not only in the workers in local Asbestos mines, but also in the population at large in an area extending peripherally from the mines.

Kiviluoto, measuring anthophyllite, found that at a distance of one-half kilometer from the mines there were twelve to thirty-four grams of anthophyllite dust per hundred square meters; and that at a distance of one and one-half kilometers there were six to twelve grams; at twenty-five kilometers there were 1.38 grams; and at fifty kilometers there was no measurable dust.

These findings, again, pointed out the theoretical and practical importance of dust control, not only for immediately exposed workers, but for the community at large.

There were many other papers on the radiologic changes of pulmonary asbestosis; and there were reports relating to the sequential pulmonary physiological changes seen in asbestosis; and there were encouraging data relating to the decreased incidence of Asbestos disease in areas where good dust control had been engineered.

It can be said that the bulk of the reported work related to isolated parameters of asbestos disease.

Moreover, it was my personal observation, that with some very significant exceptions, the major portion of material and evidence presented described experiences and findings long past and not immediately applicable to the present.

It was lamented by Dr. John C. Gilson of the Pneumoconisosis Medical Research Council of Great Britian, one of the chief protagonists of this meeting, that none of the studies made significant correlations of clinical, radiologic, physiologic and dust counting data.

With this thinking in mind, and at the end of the meeting I was given the opportunity to describe some things about the work and findings of our own program at the United States Rubber Mill in Hogansville, Georgia.

Having been originally requested to limit the remarks to three minutes, I was allowed to speak for some fifteen or more minutes, and it appeared that the comments were well received.

It has been the purpose of our own study to learn as much as possible regarding the nature of asbestosis in this particular Hogansville environment; and of greater correlary significance, to ascertain the earliest moment at which significant changes occurred, in order that workers might be removed from the asbestos atmosphere.

The work already accomplished and criteria established have, in our mill, gone a long way to protect the workers and the company. Vanished is the air of concern and even apprehension which was nascent in the mid nineteen fifties.

The company has been virtually free of litigation, and the employees have developed confidence that their health interests are being protected. In fact, numerous unrelated medical conditions have been uncovered sufficiently early in many instances for workers to seek preventive care and remain healthy.

A few basic conclusions and observations can be presented.

Exposure to asbestos dust can produce pulmonary asbestosis.

The advent of the disease process appears to be grossly relate to an accumulative dust count and time factor. Decreasing the dust exposure by suitable engineering should prolong the working time and effectiveness of the trained employee. Current evidence indicates this to be the case.

Accumulative dust exposure (measured here in million particles per cubic foot-years) appears to be not the only factor implicated in the production of pulmonary asbestosis. There are several instances of grossly equal total exposure in different workers, some of whom have asbestos disease, while others do not.

The explanation is not immediately forthcoming. However, there are some interesting leads pointing to the type of work (eg, spinner, weaver, etc), type of responsibility (eg, supervisor or worker); mouth breathing versus nose breathing, presence or absence of chronic bronchitis; and perhaps an hereditary increased susceptibility as in mucoviscidosis.

There remains the further presently enigmatic possibility that initial exposure to high dust concentrations, such as occurred in the early operations of this mill, may be more important in the production of pulmonary asbestosis than the prolonged exposure to low concentrations, against which the workers own physiological defense mechanism may provide sufficient protection. Studies in progress should serve to resolve this problem.

We have not, as yet, observed a single worker with cancer of the lung, pleura, stomach, colon or rectum.

All reports at the recent conference pointed towards the increased incidence of these malignancies twenty years or more following initial exposure.

Fragmentary evidence suggested greater risk from exposure to crocidolite dust. Since this mill has used chrysotile fibers almost exclusively since the initial installation of a fly frame and winder as a pilot operation in 1938, it must be hoped that our workers will escape this misfortune. A close watch will be kept for signs of trouble.

Accepting the stipulation that the pre-eminent cause of pulmonary asbestosis is exposure to asbestos dust fibers, it is a logical assumption that the prevention of asbestosis rests on effective dust control.

The criteria of effective dust control are not only the factual evidence of diminished dust counts; but also the abolition of the sequelae of exposure--pulmonary asbestosis.

The second"line of defense" therefore rests on early detection of the disease process.

We have studied this condition--as it occurs in Hogansville--using many points of view.

We are now able to make a clinical diagnosis of pulmonary asbestosis at a relatively early stage. There is some evidence at hand suggesting that the disease process is not progressive once the worker has been removed from the dust exposure. It is too early to draw conclusions in this regard.

However, the criteria for diagnosis still rest upon the demonstration of observable evidence brought about by the pathologic process which is occurring within the lungs.

By the time these early changes are demonstrable, significant fibrotic disease of an irreversible nature has occurred.

The importance of the earliest possible detection is thus readily apparent.

It would now appear that the most sensitive means available is the utilization of diffusion studies which measure the rate of diffusion of a gas from the pulmonary alveolus to the pulmonary capillary--the site at which the earliest significant pathologic changes occur in asbestosis.

We have been using a two level of oxygen method, which while of great informative value, is time consuming and potentially dangerous in older workers of whom we have a considerable number.

Carbon monoxide diffusion studies, using very low concentration of the gas, are feasible, safe and relatively rapid; and I would hope that we could further our work using this technique. Infra red analyzer engineered to measure carbon monoxide and carbon dioxide concentrations, are commercially available.