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THE BENDIX CORPORATION

CORPORATE ENGINEERING STAFF

SOUTHFIELD, MICHIGAN

HEALTH-HAZARDS OF ASBESTOS -
A REVIEW OF THE MEDICAL LITERATURE

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EXECUTIVE SUMMARY

HEALTH-HAZARDS OF ASBESTOS

A. CURRENT MEDICAL KNOWLEDGE

1. Nature of Asbestos-Related Diseases

Present medical knowledge associates asbestos with three primary diseases: Asbestosis; Bronchogenic (lung) Cancer; and Mesothelioma, a rare form of cancer.

All three diseases affect the lungs and in the case of mesothelioma, the abdominal cavity may also be affected. Asbestosis can cause death but it is not always fatal, bronchogenic cancer is fatal and mesothelioma is the most deadly of them all.

2. Is there a safe exposure level?

There is general agreement that the risk of developing one of the asbestos-related diseases is positively correlated with the intensity and duration of exposure to asbestos dust.

However, there is no conclusive proof of a safe threshold level of exposure. If and when such a threshold level is determined present indications are that it will vary from type of asbestos fiber to type of asbestos fiber; from occupation to occupation; and from disease to disease.

3. What is the relationship between asbestosis and lung cancer?

There are reported cases of lung cancer victims showing symptoms of asbestosis, but if an individual does not smoke, asbestosis does not lead to lung cancer. The medical evidence is that asbestosis do occur irrespective of one's smoking habits while an excess risk of lung cancer has thus far been only associated with cigarette smokers.

4. What are the relative risks following exposure to only one variety of asbestos?

All commercial forms of asbestos cause asbestosis, bronchogenic cancer and mesothelioma. However, the risk is greatest with crocidolite, less with amosite and perhaps, still less with chrysotile.(10b)

5. What is the mechanism of disease causation in asbestos-related diseases?

Existing evidence appear to indicate that extracellular processes and the physical features of fibers constitute a major part of the causation of the lung diseases associated with exposure to asbestos dust. The fiber diameter appear to be the controlling factor in the causation of disease.

The association of "asbestos-caused" cancer with cigarette smoking, coupled with the physical nature of the process of disease causation, as present studies indicate; perhaps, permits the speculation that asbestos fibers or other fibers of similar physical dimensions, once in the lungs act only as a mechanical confactor in enhancing retention, inhibiting clearance and modifying the distribution of inhaled insoluble cigarette smoke particles which are proven to be carcinogenic. It may be inferred, therefore, that other insoluble particulate matter of similar physical dimensions as asbestos may be equally cocarcinogenic as asbestos.

6. What other causes of mesothelioma are there?

There have been reported cases of mesothelioma that cannot be linked to asbestos but there are no known other cuases of the disease other than exposure to asbestos dust.

7. Can asbestos cause other diseases?

Yes. There is ample evidence of an association between pleural plaques and all types of exposure to asbestos, and to all types of asbestos fiber. Asbestos is not the only cause of plaques but it is the most common. (33)

8. Is it possible to detect asbestosis early enough to permit a cure

With regular x-ray examinations it is possible to detect the onset of asbestosis to at least permit its control. However, the latent period of asbestos-related diseases is estimated to be about 20 years. (18)

B. COMMENTARY

There is no doubt that the inhalation of substantial amounts of asbestos can lead to increased rates of various types of lung disease, including two forms of cancer. The medical literature is full of solid evidence linking asbestos to disease. Eliminating the emission of asbestos dust into the working environment appears to be an obvious way of dealing with the problem. This, however, may not be the most feasible approach in light of economic considerations. It then becomes necessary to examine what other alternatives exist.

There is strong evidence to indicate that all the diseases associated with asbestos is exacerbated by cigarette smoking. In fact, the evidence is that there is no excess risk of bronchogenic cancer in asbestos workers who do not smoke. In this case it appears that asbestos is merely a catalyst to the causation of bronchogenic cancer and the primary carcinogen is cigarette smoke

An alternative solution to the problem that perhaps would be economically attractive to all concerned may thus be a requirement that workers in the asbestos industry be non-smokers. This option becomes even more attractive when recognition is taken of the fact that existing evidence, even though inconclusive at this time, indicates that other fibers with certain physical dimensions (below 3 μm diameter) irrespective of chemical composition may have the same effect as asbestos.

In the self interest of Bendix the apparently physical nature of the mechanism of disease causation of asbestos fibers should be carefully considered in our search for alternate material for our brake lining composition.

P.S: SEE ATTACHMENT A FOR SUPPORTING DETAIL AND BIBLIOGRAPHY OF THE MEDICAL LITERATURE REVIEWED.

ATTACHMENT A

HEALTH-HAZARDS OF ASBESTOS
A REVIEW OF THE MEDICAL LITERATURE

ASBESTOS-RELATED DISEASES

Three primary diseases are known to be caused or induced by the exposure to and inhalation of asbestos fibers in industrial environments. They are; asbestosis; bronchogenic (lung) cancer; and mesothelioma, an extremely rare form of cancer which affects the lining of the pleural (lung) cavity or the peritoneal (abdominal) cavity.

Asbestosis

This is the most common of the three asbestos-related diseases. It is one of the lung diseases classified as "pneumoconioses." Among others are silicosis, from crystalline silica dust; byssinosis from cotton dust, talcosis from talc; and anthracosis from coal dust. Asbestosis is a non-malignant fribotic lung condition which shows a high frequency of occurrence in populations exposed to asbestos dust, if the dust concentration is high or the duration of exposure is long. (1,2)

When an asbestos fiber is inhaled into the body and it is not captured and eliminated by the normal cleansing mechanisms, two different actions can occur. It can be encapsulated with iron-rich protein - in which case it is then referred to as an "asbestos body" or "ferruginous body." Or, it can remain in a naked state-uncoated. For all intents and purposes, the asbestos fiber if coated is harmless. On the other hand, if the fiber remains in a naked state in the lung, then an almost uncontrolled growth of cells may begin, resulting in the formation of collagen, or scar tissue. When collagen forms in the lungs, it alters the normal tissue so that it no longer functions properly.

When this biological reaction occurs, the body's vital capacity is greatly reduced and the oxygen-carbon dioxide exchange function within the lungs is altered. The overall effect is poor ventilation and labored breathing, which are signs of asbestosis. Other physical symptoms of asbestosis are rales - unusual sounds produced in the chest cavity, and finger clubbing. (3a)

Asbestosis has been shown in numerous studies to cause death but it is not necessarily always fatal. (3b)

All the commercially significant varieties of asbestos; chrysotile, amosite, crocidolite and anthophyllite, have been shown to cause asbestosis. (4-9) However, there is considerable evidence to suggest that the risk is greatest with crocidolite, less with amosite and perhaps still less with chrysotile. (10a,b, 11, 12)

While exact dose-response relationships are not well established, there is general agreement that there exists a positive correlation between intensity of exposure and the frequency of occurrence of asbestosis.

Studies indicate that pulmonary fibrosis is augmented in asbestos workers by cigarette smoking. (13) It has also been shown that the risk of death from asbestosis may be increased by cigarette smoking. (14)

Bronchogenic (lung) Cancer:

A high frequency of bronchogenic cancer greater than that expected on the basis of the general male population has been shown to be manifested among persons who have had exposure to asbestos in industrial settings. (15-18) Studies (19-25) have shown that all types of asbestos can give rise to an excess of bronchogenic cancer under some circumstances. It has been forcefully demonstrated that the risk of bronchogenic cancer is greatest with crocidolite, less with amosite and perhaps, still less with chrysotile. Evidence from studies using dust exposure assessments has generally shown that the excess risk of bronchogenic cancer is related to dose and duration of exposure. Perhaps the most significant conditional factor associated with the excess risk of bronchogenic cancer related to industrial exposure to asbestos is the requirement of cigarette smoking. Existing evidence indicates that non-smokers do not show any excess risk of bronchogenic cancer regardless of their exposure to asbestos. (26, 27)

The biological mechanisms involved in the development of bronchogenic cancer and mesothelioma are not yet clearly established. However, evidence based on studies thus far conducted on this subject permits reasonable speculation on a probable course of events.

In a study, conducted by M. F. Stanton (28) of the Laboratory of Pathology, National Center Institute, Bethesda, U.S.A., various structural forms of asbestos, fibrous glass and aluminum oxide were tested for carcinogenicity on the pleura (lung) of rats. Results from all three materials indicate that carcinogenicity is related primarily to fibrous structure rather than to physicochemical properties. A comparison of the dimensional distribution of fibers in those samples of asbestos and glass producing high and low tumor incidence indicate that carcinogenicity may be related to fibers below 2.5 μm (micron) in diameter and between 10 to 80 μm in length.

Another study by V. Timbrell (29) of the MRC Pneumoconiosis Unit, Llandough Hospital, Penarth, UK. concluded that

> "extracellular processes and the physical features of fibers appear to constitute a major part of the etiology (causation) of the lung diseases associated with exposure to asbestos dust, including the cancers. The biological exposures which different types of asbestos fibers produce when inhaled seem to be governed largely by the aerodynamic properties of the fibers. The physical characteristics of the fibers which form the basis of these suggested explanations are fiber diameter, fiber length and fiber morphology, the central parameter being fiber diameter."

Further evidence supporting the thesis of physical processes as etiological (causation) mechanisms of asbestos-related cancers is provided by Dr. Edward A. Martell of the National Center for Atmospheric Research. In two articles [30,31] published in 1974 and 1975, Dr. Martell asserted and provided evidence to support the following:

> "Airborne ^{210}Pb is concentrated on small Aitken particles which accumulate on tobacco trichomes. Tobacco curing and the combustion of trichomes in burning cigarettes produce insoluble particles of high ^{210}Pb radioactivity which are inhaled and deposited in the bronchi of smokers. The subsequent ingrowth of ^{210}Po results in high local alpha irradiation which may account for bronchial cancer among smokers."

The significance of this finding is that coupled with the cited evidence of the physical nature of the causation of asbestos-related cancers, and other related facts, the suspected carcinogenicity of asbestos (or fibers of an appropriate physical dimensions) may be subject to reinterpretation.

It has been observed by Selikoff et al [26,27] that bronchial carcinoma deaths among asbestos workers who smoke cigarettes are about eight times that expected for cigarette smokers in general, whereas there were no excess bronchial carcinomas among asbestos workers who did not smoke. It thus follows that asbestos is not a primary carcinogen. It is indeed a cocarcinogen.

It may be concluded, therefore, that it is likely that an accumulation of the small asbestos fibers (or other similar fibers) in the lungs of the workers acts only as a mechanical cofactor in enhancing retention, inhibiting clearance, and modifying the distribution of inhaled insoluble alpha-emitting radioactive particles from cigarette smoke which are proven to be carcinogenic.

Mesothelioma

Mesothelioma is the third asbestos-related disease. It is an extremely rare form of cancer which affects the lining of the lung cavity and of the abdominal cavity. Mesothelioma is by far the most serious of the three diseases because at the present time, once it is diagnosed, it is inevitably fatal -- there is no known treatment. Death usually occurs within 18 months after diagnosis.

Although there are reported cases of mesothelioma that can not be linked to asbestos, asbestos remains the prime suspect as the carcinogenic agen

According to P. C. Elmes [32], the tumor usually starts in the pleura (lung), but a small proportion starts in the peritoneum (abdomen). In either case, pain is usually the first symptom, followed later by breathlessness and loss of weight. The time course of the disease is short, "averaging 13-14 months for the pleural cases and six months for the peritoneal."

Even though the evidence [26] is not conclusive, it appears that cigarette smoking aggravates the occurence of mesothelioma.

The causation of this disease in so far as it relates to the role of asbestos, is similar to bronchial cancers as previously described.

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3a. Johns-Manville Corporation, "Asbestos and Health" Environmental Facts, Asbestos, March 1974.

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8. Cooper, W.C., and Miedema, J., "Asbestosis in the Manufacture of Insulating Materials."

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12. Wagner, J.C. "Asbestosis in Experimental Animals," British Journal of Industrial Medicine, 20, 1, 1963.

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(19 through 26, 28, 29, 32, and 33 were presented at IARC Conference on Biological Effects of Asbestos, Lyon, France, 1972)

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EXPLANATORY NOTES

1. Because the three major types of asbestos differ chemically and physically, they also differ in biological effect in animal experiments. The significance of this difference in relation to human health is uncertain. Current knowledge indicates that crocidolite, the type least used in the United States, is most clearly associated with health hazards for people. This is the concensus of a panel of nine medical experts appointed by the British Ministry of Labour. -- Christy, R.K. and members of the panel, "Problems Arising from the Use of Asbestos," Her Majesty's Stationery Office, 1967. Wagner et al., reported a high number of mesothelioma cases among crocidolite miners in one area of South Africa, but no cases among amosite miners. -- Wagner, J.C., Sleggs, C.A. and Marchand, P., "Diffuse Pleural Mesothelioma and Asbestos Exposure in the North Western Cape Province," British Journal of Industrial Medicine, 1960, 22, 261. By contrast, chrysotile mining areas in other parts of the world have not exhibited a high incidence of mesothelioma. Wagner also published experimental evidence that, compared with other asbestos fibers, crocidolite produces the most severe asbestosis in laboratory animals. -- Wagner, J.C., "Asbestosis in Experimental Animals," British Journal of Industrial Medicine, 1963, 20, 1.

2. Only in occupational exposure do asbestos dust levels appear great enough to become possible health hazards. This point is related to dosage levels, as pointed out by Enterline and Kendrick: "Asbestos dust at levels to which general populations are exposed probably is of little importance in the etiology (causation) of disease." -- Enterline, P.E. and Kendrick, M.A., "Asbestos Dust Exposures at Various Levels and Mortality," Archives of Environmental Health, August 1967. In addition investigators who report so-called "asbestos bodies" in human lungs, note that these findings are not related

to cause of death, or indeed to any disease. The comment of Thompson and Graves is representative of those in other studies: "But to convert the scanty or very scanty bodies which have demonstrated to be present in so many urban dwellers to the frequency present in a minimal basal asbestosis would require an increase by hundredfolds and to get a more diffuse classical asbestosis with pulmonary disability the multiplying factor might well be in many millions." -- Thomson, J.C. and Graves, W.M., "Asbestos as an Urban Air Contaminant," Archives of Pathology, May 1966.

3. So-called "asbestos bodies" in the lung may be produced by other substances. Gross et al., have offered experimental proof: "So-called 'asbestos' bodies were produced in the lungs of hamsters injected intratracheally with respirable filamentous particles composed of aluminum silicate... Instead of the term 'asbestos' body, the designation of ferruginous body is suggested." -- Gross,P., Cralley, L.J. and deTreville, R.T.P., "'Asbestos' Bodies: Their Nonspecificity," American Industrial Hygiene Association Journal, November-December 1967. Similar results have been reported by Davis, working at Cambridge University, England. Davis said that hamsters were injected in the trachea and the pleura with dust from aluminum silicate, glass fiber, carborundum and man-made textile fiber. "In both injection sites all these foreign materials produced bodies which with the light microscope appeared very similar to asbestos bodies. The basic assumption that asbestos like bodies can only be produced from asbestos has proved incorrect." -- Davis, J.M.G., Gross, P. and deTreville, R.T.P., "Asbestos Bodies and Bioeffects -- A Detective Story," Annual Meeting, Industrial Hygiene Foundation, Pittsburgh, October 1967.

4. One of the substances capable of producing ferruginous bodies in human lungs is talc, which has numerous industrial uses, one of the least important being the manufacture of talcum powder. The United States consumes annually about 100,000 more tons of talc than asbestos. As for the ubiquitous talcum powder, an analysis by Cralley et al., revealed that the average drug store product contains about 20 percent of respirable fibers. -- Cralley, L.J., Key, M.M., Groth, D.H., Lainhart, W.S. and Ligo, R.M., "Fibrous and Mineral Content of Cosmetic Talcum Products," American Industrial Hygiene Association Journal, July-August 1968.

5. Gross et al., used electron microscopy in an effort to find chrysotile asbestos in 28 random samples of ferruginous bodies in city dwellers. "Chrysotile, which comprises more than 90% of the asbestos used in this country, has a characteristic electron diffraction pattern... On the basis of the electron diffraction pattern, chrysotile was decisively excluded as a constituent of the cores of all 28 ferruginous bodies isolated from lungs of urban dwellers not occupationally exposed to asbestos. This exclusion is considered highly significant because if the ferruginous bodies in the above city dwellers had been caused by the inhalation of asbestos dusts, then some of the cores should logically be composed of chrysotile." -- Gross, P., deTreville, R.T.P. and Haller, M.N., "Pulmonary Ferruginous Bodies in City Dwellers," Archives of Environmental Health, August 1969.

6. An uninformed speculation, frequently stated as if it were a fact, is that the wearing of automobile brakes releases dangerous quantities of asbestos fiber into the air. A study by Lynch, of the U.S. Public Health Service, has demonstrated that this statement is erroneous. Lynch performed laboratory tests of automotive brake linings and found that normal wear

releases insignificant amounts of asbestos fiber into the air. He concluded that "the free fibers from brake lining wear appear to be an inconsequential health factor in urban air pollution." -- Lynch, J.R., "Brake Lining Decomposition Products," Journal of the Air Pollution Control Association, 7. A time factor in asbestosis cases is demonstrated by the study of McVittie showing that clinical asbestosis takes on the average about 17 years to develop. -- McVittie, J.C., "Asbestosis in Great Britain," Annals N.Y. Academy of Sciences, December 31, 1965.

8. Selikoff et al., reported a much higher rate of lung cancer among asbestos workers who smoked than among cigarette smokers generally. However, they reported not one case of lung cancer among nonsmoking asbestos workers. The authors concluded that their evidence "suggests that exposure to asbestos does not lead to an extremely high risk of lung cancer among nonsmokers." -- Selikoff, I.J., Hammond, E.C. and Churg, J., "Asbestos Exposure, Smoking and Neoplasia," Journal of the American Medical Association, April 8, 1968. A 1969 update of this study reported one case of lung cancer among nonsmokers -- Selikoff, I. J., Hammond, E. C. and Churg, J., "Mortality Experience of Asbestos Insulation Workers 1947-1968," presented at International Conference on Pneumoconiosis, Johannesburg, South Africa, April-May 1969.

9. That dust control measures effectively reduce the incidence of lung cancer among asbestos workers is evidenced in the continuing study by Knox, Doll and Hill. These investigators studied one plant in the British asbestos textile industry. In 1955 the workers in this plant exhibited a high incidence of lung cancer as was reported by Doll at that time. But in 1965, and again in 1968, subsequent to dust control measures, the incidence of lung cancer among these workers was approximately the same as for the general British population. -- Knox, J.F., Holmes, S., Doll, R.S. and Hill, I.D., "Mortality

from Lung Cancer and Other Causes Among Workers in an Asbestos Textile Factory," British Journal of Industrial Medicine, October 1968.

10. The varying prevalence of mesothelioma in the two crocidolite mining areas of South Africa has prompted comment by Wright: "That something other than, or in addition to, asbestos plays a role in mesothelioma formation seems inescapable." -- Wright, G.W., "Asbestos and Health in 1969," American Review of Respiratory Disease, October 1969.

11. Not a single case of mesothelioma has been reported among the anthophyllite asbestos miners of Finland. -- Kiviluoto, R. and Meurman, L., "Results of Asbestos Exposure in Finland," Proceeding, International Conference on Pneumoconiosis, Johannesburg, South Africa, 1969. Primary malignant mesothelioma is very rare in Canada among all segments of the population, including the chrysotile asbestos miners. -- McDonald, A.D., Harper, A., El Attar, O.A. and McDonald, J.C., "Epidemiology of Primary Malignant Mesothelial Tumours in Canada," Proceedings, International Conference on Pneumoconiosis, Johannesburg, South Africa, 1969.

12. About 50 cases of mesothelioma are reported annually in Great Britain. -- Gunter, R., Minister of Labour. Official Report to the House of Commons, April 17, 1967.

13. Typical of the doubt involved in any diagnosis of mesothelioma is the observation of Demy and Adler: "The mesothelial tumors, as usual, were the subject of doubt, debate, review and re-review, but as usual, no other primary lesion was found and so they were classified as mesotheliomas." -- Demy, N.C. and Adler, H., "Asbestosis and Malignancy," American Journal of Roentgenology, July 1967. Similarly, Wright comments: "There is lively controversy

among pathologists regarding the requirements for establishing the diagnosis of primary mesotheliomas... Further study will be needed to demonstrate whether mesothelioma is being over or under diagnosed." -- Wright, G.W., "Asbestos and Health in 1969," American Review of Respiratory Disease, October 1969.

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