PLAINTIFF'S
EXHIBIT
BRK-245

THE INDUSTRIAL COMMISSION OF OHIO
Division of Safety and Hygiene

Richard F. Celeste
Governor

Corporate Plaza Office Building
6500 Poe Avenue, Suite 330
Dayton, Ohio 45414

May 4, 1989

Judy Spencer
Legal Section
Industrial Commission of Ohio
246 North High Street
Columbus, Ohio 43215

RE: OD 31870-22
Claimant: Beulah Webb
Company: Inland Division
General Motors
P.O. Box 1224
Dayton, Ohio 45401

Dear Ms. Spencer:

On April 5, 1989, I visited Inland Division to conduct the requested occupational disease claim investigation. Representing Inland Division were Mr. Marvin Aukerman, Safety Coordinator, and Ms. Edna Scheuer, Attorney-at-Law, Smith and Schnacke.

EMPLOYMENT HISTORY

From the Inland employment record provided by the company (attached), Ms. Webb began working for Inland in October 1957 and retired on medical (T & P) disability in March 1986. She apparently stopped working in March 1984, making her time on the job 27 years. She has held various jobs with Inland over these years. Most have been within the manufacturing of weather strips completed in Building 27 of the "hill complex". Jobs directly related to weather stripping include end joining, inspect and patch, repair and salvage, and miscellaneous bench. She was also involved with the manufacture of ice trays.

End joining was a process that required splicing or corner attachment of weather strips using a latex foam. Inspect and patch is a self-descriptive process completed on weather strips. Repair and salvage required removal of defective portions of a strip which were salvaged; repairable portions were separated for that purpose. Miscellaneous bench was described as a variety of small tasks completed on weather strips.

The weather strip molding process was described as a steady state business that experienced minimal changes over

the years. No asbestos usage ever occurred in this process. The company reported that the only place where asbestos was ever used at Inland was in the Brake Lining Department. Ms. Webb's only association with this process was a 2-week stint in November 1960 in the same building as Brake Linings, but in an unrelated department.

MEDICAL EVALUATION

From David W. Wiltse, M.D., in a letter dated October 18, 1988, to Ms. Scheuer, he concludes the following:

"My overall assessment is that there no sign, whatsoever, of any asbestos-induced lung or pleural disease. In fact, Mrs. Webb's asbestos exposure was so minimal that one would not really believe that she is at high risk for any asbestos-induced lung disease. In spite of her inconsistent performance, the pulmonary function studies and her clinical picture do suggest that she is suffering from obstructive airways disease of a moderate degree. This is entirely related to her cigarette smoking. There seems to be elements of both asthmatic bronchitis , as well as emphysema, seen on both the chest X-ray and the pulmonary function studies."

From R. Michael Kelly, M.D., impressions upon examination of Mrs. Webb completed June 13, 1987, include asbestosis, early peripheral vascular disease, probable carpal tunnel, myofascitis to the back, and gastritis. "There is sufficient clinical and laboratory evidence to establish a diagnosis of asbestosis."

Mrs. Webb's smoking history is varied. She has reportedly smoked 1 pack per day for most of her adult life. Another account portrays one-half pack per day for fifteen years. Another account details a 10 pack year history.

TOXICOLOGY

ASBESTOS

The term "asbestos" is applied to a group of naturally occurring fibrous silicate minerals. These minerals fall into two major mineralogical subdivisions: chrysotile, which belongs to the serpentines; and the amphiboles, including crocidolite, asbestiform actinolite, asbestiform tremolite, amosite, and anthophyllite. Only amosite, chrysotile, and crocidolite are of economic importance. More than 90% of all asbestos usage in the United States is of the chrysotile variety. Asbestos cement products constitute the major use of asbestos followed closely by floor products or materials used in the construction industry. Materials containing asbestos have been used extensively in construction and shipbuilding for purposes of fireproofing and for

decoration. These have often been applied by spray application. (1, page 287)

Asbestos has over 3,000 commercial uses and is ubiquitous in the general environment. Because of the mineral's resistance to thermal and chemical degradation, exposures may take place starting from initial mining of the fibers through manufacture, use, and eventual burial of asbestos containing waste. (1, page 288)

ASBESTOSIS

Asbestosis is a name of the pneumoconiosis produced by the inhalation of asbestos fibers. It is characterized by diffuse interstitial fibrosis of the lung parenchyma, often accompanied by thickening of the visceral pleura and sometimes calcification of the pleura. Clinical findings include dyspnea on exertion, non-productive cough, rales at the lung bases, bronchi, and in advanced cases, finger clubbing. Lung function measurements usually demonstrate a restrictive impairment with reduced diffusing capacity. (1, page 287)

A total of 2.3 to 2.5 million workers are estimated to be currently (potentially) exposed to asbestos. However, because of the long latency (20 to 30 years) required before asbestos related diseases become clinically manifest, past asbestos workers must also be considered at risk. These estimates are especially difficult to develop and are subject to controversy (29). Nonetheless, large numbers of previous asbestos workers are now completing their latency period and are at risk of asbestos related disease. (1, page 289)

Smoking and asbestos exposure are more than additive in their combined ability to increase the risk of lung cancer. Hammond et al. reported results of their 10-year follow-up of 8,220 asbestos insulation workers with known smoking status (38). The mortality experience of these workers was compared with that expected among smokers and nonsmokers of the American Cancer Society's prospective cancer prevention study. Asbestos workers who did not smoke showed approximately a fivefold risk of lung cancer compared to the nonsmoking control population. On the other hand, a more than sixtyfold risk of lung cancer was observed for smoking asbestos workers compared to nonsmoking controls. A similar multiplicative effect was observed by Selikoff et al. among a factory cohort producing amosite insulation (129). (1, page 298)

The relationship between pleural thickening and calcification and subsequent mortality is important insofar as surveillance of asbestos workers is concerned. Edge and Sheers completed independent mortality studies of workers

with and without plaques; neither of these studies established causality between pleural changes and subsequent development of mesothelioma or lung cancer because neither asbestos exposure or latency were controlled for in the analysis. However, plaques and pleural thickening do indicate an asbestos exposure and this fact alone places the workers at an increased risk for lung cancer and asbestosis. (1, page 298)

Recent papers on lung function among those with asbestosis have suggested that a mixed restrictive and obstructive pattern and obstructive defect are also commonly found among those with asbestosis. In 1972, Muldoon and Turner-Warwick reported 13 of 60 asbestos workers evaluated at the Brompton Hospital had a pure obstructive ventilatory defect; 3, a mixed pattern; 32, restriction; and 12 were normal (72). In 1975, Fournier-Massey and Becklae reported that among 1,000 Canadian asbestos miners and millers, 12.8% had a restrictive pattern (30). Murphy et al. in a study of shipyard workers, found no more obstruction among asbestos workers than matched controls (94). However, Rodriguez-Roisen et al. recently reported an obstructive pattern, defined by reductions in forced expiratory flow at 75% of the vital capacity, in 34 of 40 asbestos workers referred to the Pneumoconiosis Medical Panel and the Brompton Hospital, London (114). Although only 7 of 34 were considered nonsmokers, the authors suggest that airways obstruction, particularly affecting small airways, is a common functional abnormality attributable to asbestos exposure. This view is consistent with pathological observations which show peribronchiolar fibrosis to be an early lesion in asbestosis. The extent and severity of obstructive defects among asbestos workers, however, still needs full epidemiological evaluation with attention to other risk factors, especially smoking. (1, page 317)

CHRONIC OBSTRUCTIVE PULMONARY DISEASE

Emphysema and chronic bronchitis make up the disease category called chronic obstructive pulmonary disease (COPD). At one time they were viewed as distinct clinical conditions, but it is now clear that in most patients they coexist, although one or the other may predominate. These are chronic disorders and are characterized by progressive limitation of the air flow into and out of the lungs. Emphysema is identified by its characteristic alteration of lung architecture. A person with emphysema has destruction of the walls of the smallest air passages, called bronchioles, and the walls of the small air sacs, called alveoli, that function as the gas exchanging units of the lung. The alterations result in abnormally enlarged air spaces. Chronic bronchitis is characterized by excessive mucus secretion in the bronchial tree which leads to a persistent, productive cough. An individual is considered

to have chronic bronchitis if cough and sputum are present on most days for a minimum of three months for at least two successive years or for six months during one year.

In emphysema, the limitation of airflow is caused by abnormalities of lung elastin, as supporting tissue in the bronchioles and the walls of the alveoli. This abnormality in elastin results in collapse or narrowing of the small airways which in turn limits airflow out of the lung. Once this damage occurs, it is irreversible. Emphysema can follow chronic bronchitis or any other lung condition in which the airways become seriously clogged or obstructed. (A few people have an inherited enzyme deficiency that predisposes them to emphysema, regardless of other lung diseases or exposure to hazards.)

In chronic bronchitis, airway obstruction is due to inflammatory changes in the walls of the bronchi which causes them to thicken. This thickening, together with excessive mucus production obliterates some of the smaller air passages and narrows larger ones. These changes are to some degree reversible.

The three most significant factors contributing to the disease are smoking, air pollution, and occupational exposures. The effects of cigarette smoking are massive and obscure the effects of environmental agents such as dusts. (2, pages 331-332; 436-438)

CONCLUSION

This claim alleges that asbestosis-related disease has developed in the claimant as the result of her employment with Inland Division of General Motors Corporation. Her employment history shows that she worked in weather strip manufacturing operations for most of her career. Asbestos has not been used in the manufacture of weather strips. Medical diagnoses offer conflicting interpretations of her condition. The claim file contains an extensive history for medical treatment of chronic obstructive pulmonary disease and chronic bronchitis. From the information presented, it does not appear that asbestosis-related disease could develop when occupational exposures to asbestos have not occurred. Further review of the claimant's medical condition appears warranted in light of the conflicting diagnoses.

Respectfully Submitted,

Chris Ottoson

CHRIS OTTOSON
Certified Industrial Hygienist

REFERENCES

1. "Occupational Respiratory Diseases", James A. Merchant, M.D., Dr. P.H., Editor, Division of Respiratory Disease Studies, Appalachian Laboratory for Occupational Safety and Health, U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health, September, 1986.

2. "Encyclopedia of Occupational Health and Safety", International Labor Office, Geneva, Switzerland, 3rd Edition, Vol. 1, 1983.