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Chrysotile Asbestos and Health in Zimbabwe: I. Analysis of Miners and Millers Compensated for Asbestos-Related Diseases Since Independence (1980)

Mark R. Cullen, MD, and Rabelan S. Baloyi, PhD

Data on the health effects caused by locally mined chrysotile asbestos in Zimbabwe have been very limited. The prevailing local view has been that risk is minimal. In this report we critically reassess the cases of 51 individuals with asbestos exposure who have been compensated by the Central Pneumoconiosis Bureau since independence in 1980. Results demonstrate that the major health risks of exposure reported elsewhere—morbid asbestosis, nonmalignant pleural disease, malignant mesothelioma, and lung cancer—all occur in Zimbabwe, at least among workers in the asbestos mines and mills. It is concluded that further investigation and control measures in the industry are warranted.

Key words: asbestosis, pleural disease, mesothelioma, lung cancer, chrysotile miners, chrysotile millers

INTRODUCTION

As one of the world's major suppliers of chrysotile asbestos and producer of several important asbestos products, Zimbabwe has a sizeable population exposed to the mineral, occupationally and environmentally. Currently, some 7,000 men are engaged in mining and milling of the major ore bodies in the Midlands, with another 3,000–4,000 engaged in the manufacture of asbestos construction materials (cement, asphalt, tiles) and automotive products (brakes, gaskets, clutches). An undetermined number of people are further exposed in the use, repair, and disposal of these materials. Some 40,000–45,000 people live within a few kilometers of the mills and mines; a large portion of the population lives and works in buildings made from asbestos materials.

Despite these exposures, some of which have been ongoing for decades (the mines opening about 1910), there is general optimism among local observers that

Yale New Haven Occupational Medicine Program, Yale University School of Medicine, New Haven, Connecticut (M.R.C.).
Department of Occupational Health, Safety and Workers' Compensation, Ministry of Labour, Manpower Planning and Social Welfare, Harare, Zimbabwe (R.S.B.).
Address reprint requests to Dr. Mark R. Cullen, Yale University School of Medicine, Occupational Medicine Program, 333 Cedar Street, New Haven, CT 06511.
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health effects have been limited and a suggestion that the product itself—a hugely important export commodity—may be safer than amphibole or even chrysotile asbestos mined and processed elsewhere in the world. Although some of this impression may result from perhaps premature acceptance of some published opinions from Europe and North America regarding the lesser toxicities of chrysotile generally [Cullen, 1987], at least some opinion has been founded in observation, or rather lack of observations of anticipated sequelae, e.g., asbestosis, lung cancer, or mesothelioma.

Four papers have been published on the subject. Gelfand and Morton [1969] reviewed the asbestos-associated cases certified by the Pneumoconiosis Bureau between 1963 and 1967. Only 37 cases met international criteria for asbestosis, once cases of apparent silicosis or tuberculosis were excluded. The authors, without critical analysis of their data source, extrapolate from the results quantitative evidence of a very low rate of asbestosis given the total numbers apparently at risk.

In 1977, Walker, a mine physician, described the absence of even a single case of malignant mesothelioma either in his personal experience or, to his knowledge, anywhere in the country. He also commented on the rarity of asbestosis and asbestos-related pleural disease in the mining populations he had cared for but did not provide quantitative information.

Two years later, Mostert and Meintjes [1979] reported six cases of railwaymen who had developed asbestosis (four cases) and mesothelioma (two cases). Their careful review of exposures, however, clearly documented that each man had had some exposures to nonlocally mined amphibole fibers; indeed, each had worked during the time when crocidolite asbestos was being imported and used by the railroads.

In a note published in 1983, Mossup commented again on the rarity of asbestos-related diseases in his experience, claiming further that few cases were being identified by the Pneumoconiosis Bureau despite expansion of its role. He presented, as well, results of a proportional cancer mortality analysis of asbestos miners done using the cancer registry of the Mpilo Central Hospital, which draws referrals from the mine area. These data show that only 12% of all cancers registered among asbestos-exposed workers were primary bronchogenic, a rate similar to other occupational groups; this would suggest the absence of an asbestos effect, which would be expected to elevate the proportion of lung cancers as well as the total incidence of cancer cases (which could not be assessed by this method).

In an as yet unpublished study, Armstrong and Elmes [1985] performed a cross-sectional survey of all active asbestos cement plant workers and a sample of asbestos mine and mill workers with more than 10 years of exposure. Among cement workers, for whom measured and extrapolated exposure levels were available, a clear dose-response of asbestos fiber on forced vital capacity and radiographic changes was demonstrated, consistent with previous studies of the cement industry in other countries. At the mines and mills, parenchymal radiographic abnormalities (ILO grade 1/0 or greater) were noted in 20% (mines) and 26% (mills), although the representativeness of the sample was not verified. As well, no dose-response could be shown on lung function using a surrogate index of exposure, years of exposure. No actual or reconstructed air levels were available at that time for the analysis.

Given these very limited data sources, it was the aim of the present investigators to gain a clearer picture of the risks in the various exposure settings for malignant and

the X-rays, demographic data, medical and occupational histories, and laboratory reports. The basis for best-evidence diagnoses and vital status when known was also recorded. Final analysis was limited to cases meeting the following criteria. 1) Best evidence diagnosis was of an asbestos-related disease (asbestosis, benign pleural disease, lung cancer, or malignant mesothelioma); 2) Exposure to asbestos occurred exclusively at a Zimbabwe asbestos mine or mill, since general knowledge of exposures to the total workforce is available and because universal X-rays have been performed during the period of interest.

RESULTS

Among the approximately 300 men certified by the Bureau as having occupational lung disease since 1980, 54 were identified as having worked at some time at an asbestos mining or manufacturing facility. Complete Bureau records and X-ray files were available on 51 of these men, who thus formed the study population.

An attempt was made first to assign a clinical-radiologic diagnosis in each case, based on the records. In most cases, this was accomplished by radiographic interpretation coupled with review of reports of physical examination and sputum smears and culture for acid-fast bacteria. This strategy was necessary because, in the majority of cases, no detailed clinical evaluation, e.g., functional studies, bronchoscopy, biopsy, etc., had ever been conducted and patients were dispersed geographically. Even among the 17 confirmed to have died, only five had received postmortem examinations, but two others had had premortem lung biopsies. Notably, biopsy or autopsy results were the initial basis for compensation in six of these seven cases; only one man in the series had been certified for compensation on clinical grounds and subsequently had histologic (postmortem) confirmation of diagnosis.

Using this classification strategy, diagnoses were as follows: Ten cases had tuberculosis, smear- and culture-proven in six, suspected and treated clinically in the other four. In none of these cases could underlying pneumoconiosis be established. Eight cases met radiographic criteria for silicosis (i.e., symmetric small rounded opacities), simple in two and complicated in six. Among the latter, two were demonstrated to have superimposed pulmonary tuberculosis by culture or biopsy; the remainder had typical features of progressive massive fibrosis. Interestingly, in five cases, no exposure to dust other than asbestos mining or milling could be documented.

Six cases appeared not to have diseases primarily associated with dust or fiber exposures. One had chronic obstructive pulmonary disease demonstrated functionally and radiographically. A second had autopsy-documented adenocarcinoma of the prostate. Two had focal infiltrates most consistent with infectious pneumonitis. One had clinically recognized congestive cardiac failure. The last had a diffuse, symmetric profusion of thick irregular opacities (ILO U/U) associated with hilar and paratracheal node enlargement, most consistent with some granulomatous lung disease, likely sarcoidosis; insufficient data were available for better classification.

This left 27 men with findings consistent with one or more of the asbestos-associated diseases. Detailed clinical and occupational data on these workers appears in Table I. The findings can be summarized as follows:

10	64/1985	Asbestosis	Chest X-ray 2/2 s/t bilateral plaques	1951–1980	1	Mill attendant	Alive	
11	65/1985	Probable lung cancer	Chest X-ray	1932–1980	2	Mine laborer	Unknown	Negative AFB studies in 1985; no follow-up available
12	72/1986	Asbestosis	Chest X-ray 1/1 t/t bilateral plaques with calcification	1955–1971	2	Underground manager	Alive	Croccidolite mining 1951–1955; nonasbestos mining 1953–1951 and 1971–1983
13	46/1986	Asbestosis	Chest X-ray 1/0, s/q bilateral plaques	1966–1986	1,2	Sanitation worker	Alive	
14	46/1986	Asbestosis	Chest X-ray 2/2, t/u normal pleura	1957–1986	2	Mill operator	Dead (1987)	No postmortem examination
15	Unknown/ 1986	Asbestosis	Chest X-ray 1/1, s/t unilateral plaque	1952–1986	1	Welder, plater	Alive	
16	Unknown/ 1985	Asbestos-related pleural disease	Chest X-ray 0/0, bilateral plaques	1962–1966	3	Mine laborer	Alive	Gold and copper mining exposures after 1966
17	70/1983	Asbestosis	Postmortem	1946–1967	1	Mine laborer	Dead (1983)	Preterminal X-ray 2/2 t/u diffuse pleural thickening
18	Unknown/ 1984	Asbestosis	Chest X-ray 1/1, s/s normal pleura	1971–1984	Cement factory	Sweeper	Alive	
19	52/1984	Malignant mesothelioma	Biopsy	1969–1981	2	Fitter, turner	Unknown	Premalignancy X-ray (1987) normal
20	78/1984	Malignant mesothelioma	Postmortem	1951–1952	4	Manager	Dead (1954)	Premalignancy chest X-ray 1/0, s/t, normal pleura (1978)
21	Unknown/ 1984	Asbestosis	Chest X-ray 2/3 s/t, bilateral plaques	1963–1973	5	Loader	Alive	Healed apical tuberculosis
22	69/1983	Asbestosis	Chest X-ray 2/2 t/t, bilateral plaques	1960–1983	2	Lasher	Alive	
23	62/1950	Asbestosis	Chest X-ray 1/2 t/q, normal pleura	1957–1980	1	Mill sweeper	Alive	Nodules midzones; ?mixed dust pneumoconiosis
24	Unknown/ 1980	Asbestosis, probable terminal mesothelioma vs lung cancer	Chest X-ray 1/0 s/t, normal pleura/pleural mass 5 years later	1960–1985	1	Gang leader	Dead (1986)	No postmortem examination
25	43/1981	Asbestosis	Chest X-ray 1/1 s/t bilateral plaques	1960–1972	1	Mill operator	Alive	
26	43/1982	Asbestos-related pleural disease	Chest X-ray 0/0 calcified bilateral plaques	1956–1982	1,5	Blaster	Alive	
27	61/1982	Asbestosis	Chest X-ray 3/3 s/t normal pleura, open-lung biopsy	1940–1957	1	Driller	Alive	

*Sites: 1, 2, active asbestos mines/mills, 3–5, closed asbestos mines/mills.

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