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

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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

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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

nonmalignant disorders most closely associated with exposures to asbestos in other countries. Ideally, a mortality study using conventional techniques would have been proposed. However, given the very crude state of health statistics in the country generally, and the recognized fact that cause of death can rarely be ascertained for rural populations, such an approach was not feasible. On the other hand, the nation has had a functioning Pneumoconiosis Bureau for almost 40 years. Despite obvious limitations to this existing source, it was determined that information at the Bureau could be utilized to make certain qualitative and even semiquantitative judgements regarding the occurrence and rate of some disabling and lethal sequelae of asbestos exposure. The strategy, results, and interpretation of these data are presented here. Subsequent to this effort, a cross-sectional health survey of current asbestos miners and millers was conducted to elucidate dose-response relationships for nonmalignant respiratory disease; these data appear as a companion article [Cúllen et al., this issue].

MATERIALS AND METHODS

Pneumoconiosis Medical Bureau Practice

Zimbabwe has had a Pneumoconiosis Bureau since 1949. In 1984, a law was passed requiring universal surveillance of miners and others in "dusty trades," primarily those directly associated with mines; prior to that time, X-rays were performed primarily on more skilled or managerial personnel only. Chest X-rays representing "initial" (first ever) or "periodic" (routine follow-up) examinations are obtained by employers and mailed to the central facility in Harare. Films are accompanied by a brief questionnaire and medical report, which generally provides age, summary work history, and job title. After registration, films and records are reviewed by a Medical Panel consisting of a chest surgeon, a radiologist, a public health physician, and mine doctors. The purpose of the review is to certify: 1) fitness for work; and 2) eligibility for compensation on a four-grade scale. In addition to miners, workers from nonmining industries suspected of having pneumoconiosis may apply to the Bureau for compensation, although routine X-rays are required only at mining facilities; few other industries routinely obtain them.

Since this study would be limited to cases selected by the Medical Bureau, the selection process itself was investigated in a preliminary step. After observation of several working sessions, a sample of approximately 500 newly arrived (unread) films were blindly read using the ILO system by one of the authors (M.R.C.), a NIOSH-certified B reader, and results were compared to subsequent readings by the Bureau. In this comparison, it became evident that fewer than 10% of grade 1/0 of 1/1 films (about 5% of total) were designated abnormal by the panel, whereas most films grade 1/2 or higher (about 1% of films) were identified for compensation. Thus it was appreciated at the outset that the identified cases would, in general, reflect those of moderate or greater severity, at least radiographically, rather than the group of all miners and millers with abnormal findings.

Case Selection Evaluation and Analysis

The records of all cases of occupational lung disorder certified since 1980 were reviewed to establish place of employment. All those from places in which asbestos was known to be mined, processed, or used were selected and an effort made to locate the full X-ray folder and compensation file. Once located, the entire case was reviewed in order to establish best diagnosis, using all available data sources, including

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:

1. Three men, all with exposure exclusively in Zimbabwean mines and/or mills, had evidence of asbestos-associated pleural plaques, without parenchymal changes. All were alive and working, apparently without impairment.
2. Three men with otherwise normal X-rays presented with clinical and radiographic features of chest malignancies. One case was proven at open resection to be malignant mesothelioma. In the other two, no pathologic diagnosis was achieved—one radiographically resembled mesothelioma, the other a primary bronchogenic tumor.
3. Twenty-one men had radiographic and/or pathologic evidence of asbestosis. Of these, one had been exposed making asbestos cement only, and 2 others had been extensively exposed to asbestos outside Zimbabwe; the other 18 had been exposed exclusively in the Zimbabwe mines and/or mills.

The 18 asbestosis cases associated with Zimbabwe mine/mill exposures only were further analyzed. Eight of these men were established to be dead by 1988, three from likely malignancies (one confirmed mesothelioma), five from progressive respiratory failure and/or cor pulmonale. Among the ten who survived, the severity level of disease, judged radiographically, was generally high, all but three being ILO profusion grade 1/2 or greater at the time of diagnosis. In general, exposures among these cases were of long duration, ranging from 17 to 37 years, with a mean of 22.3; latency prior to disease recognition was comparable (range 17–42 years, mean 27.2).

DISCUSSION

Despite the obvious limitations of a case series of the kind presented here, limited inferences may be drawn from these data, which could provide a rationale for some changes in clinical and public health practice in Zimbabwe as well as a basis for more substantial scientific inquiries in the future. Coupled with data from the cross-sectional study of the mining industry [Cullen et al., this issue], a more secure justification has been developed for undertaking these costly activities.

Looking first at the shortcomings, the most obvious problem is the underrecognition bias introduced by looking at a workers' compensation-derived series. Although the active workers at the asbestos mines and mills have been under medical surveillance during the study period, there is no capacity in the system to follow-up miners who have quit or retired from the industry, except for those who remain in the mining area and voluntarily utilize mining health services; the demographics of the workforce suggest that the majority return to their families in their region of origin. Among these, it is unlikely that most asbestos-related diseases would be recognized or a case filed given general lack of awareness about the problem in most rural areas and the impediments to filing. The problem is compounded by the fact that lung cancer and mesothelioma have not been formally listed as compensable disorders, which could further reduce reporting for these conditions. That this bias is operating is suggested by the fact that over three-fourths of cases studied in this series were actively working when first diagnosed.

It is also clear that the series severely underrepresents workers or ex-workers with grade 1 parenchymal changes on X-ray. In the series, only three nonfatal cases had changes below category 1/2, whereas eight had died, implying a highly skewed series in terms of case severity. That a large number of lower grade cases have

TABLE I. Cases Certified by Zimbabwe Pneumoconiosis Bureau Between 1980 and 1987 With Suspect Asbestos-Related Diseases

Case no.	Age and year of diagnosis	Diagnosis	Basis for diagnosis	Exposure period	Site[a]	Job title	Vital status (year of death)	Comments
1	35/1980	Asbestosis	Biopsy/postmortem	1965–1980	1	Mill laborer	Dead (1982)	Rapid progression 1980–82; last chest X-ray 2/3 t/t with bilateral plaques
2	54/1987	Probable mesothelioma	Chest X-ray	1949–1982	1	Mill sweeper	Dead (1987)	Normal X-rays 1983; pleural mass noted premortem; no postmortem examination
3	64/1987	Asbestosis	Chest X-ray; 1/2 t/t pleural calcifications	1969–1970	1	Electrician	Alive	Prior exposure in shipyards, power stations, railroads beginning 1943
4	55/1980	Asbestos-related pleural disease	Chest X-ray 0/1 bilateral plaques with calcifications	1947–1980	1	Mill operator	Alive	
5	44/1982	Asbestosis	Chest X-ray 2/2 t/t slight pleural thickening	1957–1962	1	Mine driller	Dead (1983)	Death certificate; cor pulmonale; no postmortem
6	53/1987	Asbestosis	Chest X-ray 1/2, s/t bilateral	1964–1980	2	Mine laborer	Alive	Prior nickel, gold mine exposure
7	63/1986	Asbestosis	Chest X-ray 1/2 s/s	1949–1986	1	Mine laborer	Alive	
8	Unknown/ 1980	Asbestosis; probable lung cancer	Lung biopsy; chest X-ray	1948–1970	1,2	Mine laborer	Dead (1980)	Mass on X-ray in 1979, negative bronchoscopy, 1979; open lung—asbestosis; cancer not identified
9	51/1983	Asbestosis	Postmortem	1962–1983	1	Mill operator	Dead (1983)	Premortem X-rays of poor quality; postmortem revealed advanced fibrosis with ferruginous bodies
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	Crocidolite 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, pluter	Alive	

10	64/1985	Asbestosis	Chest X-ray 2/2 s/t bilateral plaques	1951–1980	1	attendant	Alive	
11	65/1985	Probable lung cancer	Chest X-ray	1932–1980	2	3 laborer	Unknown	Negative AFB studies in 1985; no follow-up available
12	72/1986	Asbestosis	Chest X-ray 1/1 s/t bilateral plaques with calcification	1955–1971	2	Underground manager	Alive	Crocidolite mining 1951–1955; nonasbestos mining 1953–1954 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.

escaped detection by this method has been proven by the findings in our own cross-sectional study [Cullen et al., this issue], in which 8.7% of workers with more than 10 years of exposure had such changes (categories 1/0 and 1/1), and in the previous study by Elmes and Armstrong [1985], who found over 20% in this category range. Given that each of the approximately 3,000–4,000 active workers with this amount of experience has been X-rayed at least once, an additional 240–800 cases can be assumed to be missing from a theoretically complete series due to this bias alone.

Another justifiable concern about the case series is the accuracy of diagnoses given the limitations of the available data and the use of a single clinician (M.R.C.) as the diagnostic standard. This is especially true of the six apparent malignancies, of which only two (both malignant mesotheliomas) were pathologically confirmed. To a lesser extent, this is also a problem with the asbestosis cases, in which other, albeit less probable, causes could rarely be excluded from the records. Similarly, the radiographic categorization may certainly be askew when based on the judgement of a single reader [Musch et al., 1985]. This is unlikely to introduce too much overdiagnosis, however, since each film had already been judged to represent asbestosis by the Medical Panel.

These problems notwithstanding, the data appear to confirm that each of the major diseases associated with asbestos in other countries—asbestosis, mesothelioma, nonmalignant pleural disease, and carcinoma of the lung—all occur among Zimbabweans exposed exclusively to the domestic chrysotile industry. Obviously very little can be said quantitatively about the cancers from this series, since case ascertainment and confirmation are so far from complete; even if they were complete, comparisons would be difficult because of the lack of adequate mortality data among unexposed men in the population from which the miners and millers were drawn. Nonetheless, this is the first report to confirm the occurrence of these most dread consequences of asbestos exposure and should serve to emphasize the need to develop diagnostic vigilance among physicians who treat exposed workers and enhanced surveillance strategies at the mines and nationally.

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