THREE MINERALS, THREE EPIDEMICS — ASBESTOS MINING AND DISEASE IN SOUTH AFRICA

Marianne A. Felix
National Centre for Occupational Health
Johannesburg, South Africa

Jean-Patrick Leger
Sociology of Work Program
University of the Witwatersrand
Johannesburg, South Africa

Rodney I. Ehrlich
Department of Community Health
University of Cape Town Medical School
Cape Town, South Africa

INTRODUCTION

At the 1969 International Conference on Pneumoconiosis held in Johannesburg, Dr. J.C. Gilson (1970) observed that "[in South Africa] you have got three of the principal types of asbestos being mined. You have ... a unique opportunity to look at the effects of the mining and milling of these three types." Was Gilson's advice heeded? To what extent has the burden of asbestos-related disease in South Africa, one of the premier asbestos mining countries, been measured? Finally, when and how was such information used to reduce or eliminate this hazard?

This chapter traces the history of the mining of each of the three major commercial forms of asbestos in South Africa. As the mining of these minerals developed, so too did three epidemics of asbestos-related diseases. Environmental, as well as occupational, effects were felt. Furthermore, disease was not restricted to South Africa. Asbestos in South Africa was mined predominantly by transnational companies for international markets. As the exportation of asbestos increased in the 1920s and 1930s, so too were workers and communities around the world affected.

We examine how the local epidemics were initially identified. From the 1920s to the early 1960s, South African scientists contributed meaningfully to the understanding of asbestos-related diseases. Thereafter, it seems that the anxiety of

1. Corresponding Author: Dr M.A. Felix, National Centre for Occupational Health, Box 4788, Johannesburg 2000, SOUTH AFRICA.
2. Key Words: asbestos-related disease, asbestosis, mesothelioma, South Africa, amosite, crocidolite.
3. Abbreviations: CSIR, Council for Scientific and Industrial Research; GME, Government Mining Engineer; MWU, Mineworkers' Union; PRU, Pneumoconiosis Research Unit; SAIMR, South African Institute for Medical Research.

mining interests over losing their local and export markets, combined with the acquiescence of state agencies, led to the stifling of any research that was critical of the industry, until the 1980s. In this unequal contest of interests, the welfare of those exposed to the hazard was unlikely to prevail (Myers, 1980).

The chapter concludes with a summary of the efforts made to control the epidemics of asbestos-related disease. We conclude that state controls on asbestos production reflected more of an interest in encouraging mining and the use of asbestos products rather than an effort to protect miners and their communities from harm.

In recent years, the doctrine of self-regulation, which delegates responsibility for control of health and safety to the workplace level, has been promoted by government officials and incorporated in legislation (Republic of South Africa, 1983, 1991). In most cases in South Africa this entails self-regulation by plant or mine management. The lesson that should be drawn from the history of the South African asbestos mining industry is that in the absence of countervailing power exercised by workers and active intervention by the state, self-regulation is ineffective as a means of securing a safe and healthy workplace or environment.

THE DEVELOPMENT OF THE ASBESTOS MINING INDUSTRY IN SOUTH AFRICA

Cape Crocidolite

The first discoveries of crocidolite were made in the northern Cape Province near Prieska between 1803 and 1806 by Lichtenstein, an explorer and geologist. Prieska is situated in the crocidolite belt, a region with numerous asbestos deposits extending some 400 km in length and 50 km in width. In 1893, the Cape Asbestos Company started mining north of Prieska and created a market in the United Kingdom for this variety of fiber (Hall, 1930). Mining development extended northward to the then Bechuanaland Protectorate (now Botswana) border, as the demand for crocidolite grew during and after the First World War.

By 1930, there were 24 crocidolite mines in the Cape, as well as small diggings controlled by tributors and farmers, producing 7000 tons per year. Mining and milling were highly labor-intensive, with most mining taking place by way of open-cast workings. Fiber was cobbed from waste rock by handheld hammers before hand sorting and sieving or mechanical sorting were used (Hall, 1930).

Occupational exposure was not confined to adult men. Women, children, and infants were involved directly and indirectly in production. As Hall (1930) noted, "Native labour is paid according to the grades per standard amount of cobbed fibre ... He arranges to live close to the scene of operations, and his family help to swell the budget by picking over the accumulated debris for the most promising seams and cobbing them by hand." Women carried their infants and were assisted by their children in cobbing and picking.

Production of crocidolite escalated after the Second World War. Mining was increasingly by way of underground workings. In towns such as Koegas, Kuruman, Prieska, and Griquatown, central mechanized mill facilities were commissioned. The mills operated dry and their exhaust stacks spewed asbestos dust over hundreds of square kilometers (Sluis-Cremer, 1965).

In the northern Cape, an extremely arid region, settled asbestos dust becomes easily airborne, facilitating its spread. In addition to the dust produced by the mills, asbestos pollution was spread inadvertently by local municipalities which used asbestos tailings to surface roads. Asbestos waste was also used to cover playing fields and create golf course greens.

Production of crocidolite peaked as recently as 1977 when 200,000 tons were produced (Hart, 1988). In the 1960s and 1970s, the crocidolite mines employed between 12,000 and 14,000 workers. Since then, the demand for crocidolite has been dramatically reduced as a result of litigation in the United States, trade union and consumer campaigns in first world countries, and stricter legislative controls, particularly over crocidolite and amosite. By 1992, crocidolite production had dropped to 12,000 tons (Hart, 1992) — a figure first exceeded in the 1920s.

With these developments came severe employment cuts in the crocidolite mines. Currently, crocidolite mining occurs only at the Coretsi and Wandrag mines near Kuruman, with a total work force of fewer than 500 workers.[1]

More than 95% of the crocidolite mined in South Africa has been exported. Accompanying the fall in local production has been the diversion of trade away from first world countries over the past decade. The primary markets from the 1940s to the 1960s were Western Europe, the United States, and the United Kingdom (May, 1965). According to the managing director of the two companies that today control practically all local asbestos mining, there is currently a strong market for blue asbestos in North Africa and the Middle East (Hart, 1992).

Amosite and Transvaal Crocidolite

Details of the initial discovery of the amosite deposit in the Lydenburg district in the northeastern Transvaal are not known. Hall, an intrepid and eloquent geologist, recorded finding several old prospecting pits in 1907. He derived the name amosite from the acronym for "asbestos mines of South Africa." Amosite mining started in 1914 at Penge after difficulties encountered in marketing the new fiber were overcome (Hall, 1930). The striking features of these deposits were the length of their amphibole fibers and the great extent of the ore. With

[1] H.P. Hart (1992), personal communication.

the establishment of an international market, amosite production soon overtook crocidolite because of the ease of mining the Penge deposits (De Kun, 1965).

Amosite production peaked at 100,000 tons in 1970 (Hart, 1988) when 7000 workers were employed. In the 1980s, Japan was the main consumer of amosite for the manufacture of calcium silicate insulation boards used extensively in their building industry. In 1988, the Japanese Government declared to industry in that country that the use of amosite was no longer acceptable. By 1991, amosite in insulation boards had been phased out completely. The collapse of this Far East market led to the closure of the Penge Mine in June 1992.

The Pietersburg asbestos fields lie northwest of Penge in a 45 kilometer arc extending from Mafefe in the south to Bewaarkloof in the north. Molengraaff, a geologist, first found a specimen of crocidolite at Mafefe in 1905. The poorer grade Transvaal crocidolite deposits are contained in extensions of the Penge geological formation. They were exploited only after the economically more important amosite production at Penge was established. By 1917, both amosite and crocidolite were being mined at numerous places in the Pietersburg fields. These operations generally remained small. Numerous tributors supplied the mills in the area with cobbed fiber. The mills were crudely designed and caused extensive pollution in the valleys. By the mid-1970s, most of the small operations had closed, with some of the larger mines continuing until the early 1980s.

Chrysotile in the Eastern Transvaal

Chrysotile deposits were discovered in the Barberton district in 1905. In contrast to the arid crocidolite and semi-arid amosite fields, this area is part of the "mist belt," an escarpment characterized by heavy rainfall. Systematic prospecting for chrysotile followed in 1915 and a number of mines started shortly thereafter at Kaapsche Hoop and Kalkkloof (Hall, 1930).

The most significant commercial find of chrysotile in South Africa was made some years later in the Msauli River valley, a location also near Barberton, where mining started in 1937. Today, Msauli is the only operating chrysotile mine in the country, with the exception of two marginal operations at the old Kaapsche Hoop and Stella mines where asbestos is extracted from tailings dumps.

Unlike the crocidolite and amosite mines, Msauli, with its 1650 employees, is working close to its production capacity of 110,000 tons per annum (Hart, 1992). Ninety percent of the chrysotile obtained is exported, and is shipped mainly to Taiwan, Korea and Japan. The market for South African chrysotile firmed during the 1970s. It has remained stable at approximately 100,000 tons per annum since 1975.

One kilometer across the border into Swaziland is the Bulembe mine (previously known as Havelock), a slightly older operation than Msauli, in which an extension of the same chrysotile deposit is mined. The asbestos mined at Bulembe is transported via an aerial cableway to Barberton in South Africa.

IDENTIFYING THE EPIDEMICS

Chrysotile — The Earliest Studies

The first reports from South Africa relating asbestos to disease were published in the late 1920s. Local scientists were aware of the work being performed in Britain at the time. In a 1928 *British Medical Journal (BMJ)* article, Simson, a senior pathologist at the South African Institute of Medical Research (SAIMR), reported one of the earliest accounts of deaths due to asbestosis (Simson, 1928). He studied lung specimens that were sent by a medical officer employed in a chrysotile mine in Southern Rhodesia (Zimbabwe). Simson noted golden yellow segmented structures with club-shaped ends embedded in fibrous tissue — asbestos bodies, described by McDonald in the *BMJ* the previous year. In the 1929 Annual Report of the SAIMR, a case of "malignant pleural endothelioma" was reported in a white male — probably the first recorded case of mesothelioma in South Africa (SAIMR, 1930).

The first comprehensive survey of asbestos-related disease in South Africa was a doctoral thesis on the health of workers who were employed in the mining and milling of chrysotile. Between 1926 and 1930 — at the same time that Merewether and Price (1930) were performing their ground-breaking studies of the British asbestos textile industry — Slade (1931) investigated "the incidence of respiratory disability in workers employed in asbestos mining, with special reference to the type of disability caused by the inhalation of asbestos dust."

Slade was a medical officer at a Transvaal chrysotile mine, probably the New Amianthus Mine, a subsidiary of Turner and Newall (Hall, 1930). He investigated the effects of dust underground and in the mill, relying on clinical observations in the absence of x-ray facilities.

While dust in the wet underground workings was not visibly in evidence, Slade (1931) observed that: "Several years of experience of the mill has shown that the concentration of dust in the atmosphere in that building is at all times excessive, and frequently sufficiently so [as] to render indistinguishable objects at a distance of a few yards." The incidence of respiratory disease was therefore consistently highest in mill workers.

Slade examined a sample of 100 mill workers whose average length of service was short. Only 14% had more than five years service; the longest span of employment was nine years. Yet every worker experienced a cough productive of mucoid sputum, 72% suffered dyspnea on exertion, and 47% reported loss of weight.

Slade divided his findings into cases with "early physical signs" (clinically normal except for fine short dry crackling sounds heard by stethoscope toward the end of inspiration and during the early part of expiration), and those with "late physical signs" (evidence of respiratory insufficiency, restricted thoracic expansion, abnormalities on percussion of the chest, or abnormal breath sounds and various added sounds heard by stethoscope).

A summary of Slades's results are provided in Table 1. The very high proportion of workers with advanced physical signs despite their short employment records is striking. It is noteworthy that the prevalence rates of cough, dyspnea, and presumptive asbestosis in the mill studied by Slade were more than double those observed in the British textile factories by Merewether and Price (1930).

TABLE 1. Proportions of Chrysotile Mill Workers Diagnosed with Abnormal Physical Signs by Slade (1931)

Years of Service	Number (n)	Dyspnea (%)	Early signs* (%)	Advanced signs* (%)	Any abnormal sign
0.25 - 1	22	45	36	9	45
1 - 3	35	63	63	14	77
3 - 5	29	90	55	24	79
5 - 9	14	100	36	64	100
Total	100	72	51	23	74

* See text.

Incredibly, Slade's study, the first of any local asbestos mine or manufacturing operation, was also the last study of chrysotile mines and mills in South Africa. To this day no further study of disease in a local chrysotile mine has been published, and the authors are unaware of any unpublished surveys.

The only other studies of chrysotile workers in the region have been performed in Swaziland and Zimbabwe. The Swaziland study is relevant because of the proximity of the mine to South Africa and to the Msauli operation. McDermott et al. (1982) measured the prevalence of asbestosis among two groups of workers at the Havelock (Bulembe) Mine. Employees with more than 10 years of exposure had a pneumoconiosis prevalence rate of 29%. The second group — younger men (mean age 33 years) employed in the mill — had a 30% prevalence rate.

The Northern Cape: Linking Asbestos to Cancer

After the Slade study, no published work on asbestos-related diseases appears to have been carried out until the mid-1950s. In 1955, Dr. Chris Sleggs, superintendent of a tuberculosis referral hospital in the diamond mining town of Kimberley, was treating patients with gross pleural disease. He grew increasingly alarmed as these men and women failed to respond to anti-tuberculous therapy. Most had been referred from communities situated in the asbestos belt of the northern Cape.

One of these patients was investigated by Martiny (1956), who published the first case report of a locally identified primary mesothelioma of the pleura. The patient was a 36-year-old male born in the Kuruman district, whose case was submitted for compensation to the Silicosis Medical Bureau in 1955. The patient died in 1956 and diagnosis was made on post-mortem.

The large number of patients with this unusual tumor prompted Sleggs to collaborate with Wagner, a pathologist, and Marchand, a thoracic surgeon, to elucidate its cause (Sleggs 1960; Wagner et al., 1960). Their initial series of 33 mesothelioma cases (which grew to a total of 47 by June 1960), assembled over a mere four years, was at the time the largest recorded number (worldwide) of this hitherto rare tumor.

Wagner et al. (1960) identified circumstantial evidence of exposure to asbestos in all but one of their patients; this included fifteen patients whose exposure was non-occupational. The authors concluded that their "findings tend to add support to asbestos being the common factor in the development of these tumours" and that "mesothelioma occurs 20 to 40 years or more after exposure to dust." Internationally, this work was valuable in confirming the link between asbestos and mesothelioma. Closer to home, it demonstrated to government officials and mine management the direct link between asbestos mining and cancer.

At the 1959 Pneumoconiosis Conference held in Johannesburg, Sleggs (1960) and Wagner (1960) each presented preliminary versions of their papers which were later published in the *British Journal of Industrial Medicine.* A resolution was adopted to further investigate the relationship between asbestos exposure and mesothelioma. Consequently, the Pneumoconiosis Research Unit (PRU) of the government-sponsored Council for Scientific and Industrial Research (CSIR) embarked on a field survey in the northern Cape and at Penge in 1961. The first year of the study cost R12,000. Asbestos mining companies contributed R8000 and the South African Cancer Association contributed R4000.

Important interim data generated by the study encouraged Walters, who was then PRU director, to suggest that the study be extended to the Transvaal chrysotile fields. However, "as a result of certain deliberations at the end of the first year of the survey, it was decided that the financial contributions of the Industry and the Cancer Association would not be renewed" (CSIR, 1964). Instead, the proposal was deferred, "pending the outcome of the industry's representations to the

Government and the Government's decision in this regard" (CSIR, 1964).[2] The field work never continued.

Instead, the CSIR contributed R10,000 to finalize a report on the research that had been undertaken. This action followed an agreement by the PRU's Research Advisory Committee that "such a 'report' would not be published or made available outside the Unit [PRU], other than to sponsors and the various members of the working committees that had been concerned with the conduct of the 'survey'."[2]

The PRU study had sought to investigate the relationship between asbestos dust and mesothelioma and asbestosis. The most notable conclusions were that dust levels in the regions were so high that "people who live or who have lived in proximity to asbestos mines or mills are in danger of contracting asbestosis even though they have had no industrial exposure to asbestos dust inhalation." Second, "an alarmingly high number of cases with mesothelioma of the pleura have been discovered ... and that there is evidence to suggest that mesothelioma is associated with an exposure to asbestos dust inhalation which need not be industrial" (CSIR, 1964).

While most of the data in the PRU report were subsequently published (Sluis-Cremer, 1965), the attempts to restrict publication had certain negative consequences. Research momentum and opportunities were lost, and the field work was never extended to the chrysotile mines.

Asbestos mine management played a crucial role in influencing research until the 1980s, when independent research emerged. Management representatives were members of the steering committee for the asbestos research project, which was run under the auspices of the PRU (known then as the National Research Institute for Occupational Diseases, and now called the National Centre for Occupational Health). Apart from vetting research proposals and offering "editorial advice," mine management representatives were privy to research results and papers prior to publication. Publication of at least one study was delayed for eight years as a result of the intervention of mine management (Flynn, 1982). Studies that were performed relied heavily upon management cooperation, a process not conducive to criticism of the industry.

Nevertheless, the study that did follow was important because it was one of the first to study African[3] asbestos workers (Talent et al., 1980). In South Africa,

[2] Letter accompanying CSIR report written by Dr. Walters, director of the PRU.

[3] Apartheid legislation allocated South Africans at birth to one of four groups: Asian, black (replaced by the term "African" in this text), colored (mixed descent), and white.

the occupational health status of African miners has been badly neglected, even though they have formed the bulk of the mining work force, and limited available evidence shows African workers to have been exposed to the worst workplace conditions. Reasons that are generally advanced for the lack of disease studies among African workers and communities include migrancy, the lack of administrative infrastructure, poor health services serving African communities, and the poor quality of service records and death certification (Flynn, 1982; Frumkin et al., 1989; Sluis-Cremer et al., 1992).

Talent and co-workers sought to investigate the health status of 1185 asbestos miners who had worked in crocidolite operations of the northern Cape and lived in the Bophuthatswana "homeland." Working from a research clinic set up at St. Michael's hospital near Kuruman, the team traced 755 miners. Irregular small opacities were detected on the chest x-rays of 122 (16.2%) of these workers. In another 134 miners (17.7%), pleural abnormalities alone were present. Six miners were diagnosed with mesothelioma and one was diagnosed with bronchogenic carcinoma.

Prevalence studies cannot reflect the rate of rare fatal diseases such as mesothelioma. Even the risk of chronic nonfatal diseases may be underestimated if the observation period after initial exposure is short or if a large proportion of the study group is not traced. Of the original sampling list in Talent's study, 123 (10%) were not traced or examined, and 215 (18%) had died prior to the survey. The cause of death was not known for practically all of these cases. Among those examined, the period following initial occupational exposure to asbestos was less than twenty years for 519 (69%) of the workers. Also, 518 (69%) of the sample had fewer than five years of occupational exposure.

A key outcome of the study was the large number of cases of mesothelioma identified incidentally at the research clinic during the survey (1974-79). Sixty one cases were diagnosed among patients who presented to the hospital, or who met the study requirements but were not on the original sample list. Twenty-five of these mesothelioma cases had asbestos mining experience; 12 were women who had cobbed asbestos, and 24 had only environmental exposure. Given the remoteness of the St. Michael's Hospital, these incidental findings are suggestive of the scale of the epidemic in the northern Cape/Bophuthatswana region.

A high incidence of mesothelioma in the region is borne out in preliminary results of a birth cohort study of whites born in Prieska between 1917 and 1936 (Reid, 1990). Initially, 399 (87%) of the cohort members born between 1932 and 1936 were traced. Of the 66 people who had died, 6 had died from mesothelioma, a cumulative mortality rate of 15/1000.

Further data have been collected in this study.[4] Of 2391 white births registered between 1917 and 1936, 1762 (74%) have been traced, and of this group 532

[4] G. Reid (1992), personal communication.

have died. A total of 209 members of this cohort (39%) died before the age of 25 years. Mesothelioma was diagnosed in 19 (3.6%) of the deaths, other respiratory neoplasms in 20 (3.8%) of the deaths, and other cancers were diagnosed in 46 (13%) of the deaths. The cumulative mortality rate for mesothelioma in the revised statistics is 11/1000, which is 1000 times higher than the cumulative rate calculated for all South Africans (Zwi et al., 1989).

Two important studies have sought to examine the scale of crocidolite-related diseases other than mesothelioma. Irwig et al. (1979) measured the prevalence of asbestosis and pleural changes among white and colored asbestos mine workers. They found that the prevalence of irregular chest x-ray opacities ranged from 2.3% in men with occupational exposure of less than one year, to 48% in miners with more than 15 years exposure.

Botha et al. (1986) carried out a geographic mortality study to investigate excess mortality in crocidolite mining districts. They found significant excess mortality from asbestosis and mesothelioma, malignant neoplasms of the bronchus and lung, and malignant neoplasms of the stomach. This is the only South African study which has demonstrated a link between possible asbestos exposure and a gastrointestinal neoplasm. A recent mortality study of white amphibole miners, however, failed to confirm this association (Sluis-Cremer et al., 1992).

These studies nevertheless confirmed the earlier findings of Oettle (1964) of the National Cancer Association, who found a threefold increase in lung cancer in the Cape asbestos mining districts.

Penge and Environs: The Hidden Epidemic

The first scientists to examine disease in the Transvaal amphibole mines encountered a sordid sight. In 1949, Schepers of the Silicosis Medical Bureau visited Penge Mine as part of the first official governmental radiological and clinical survey of the northeastern Transvaal industry. At the time, the mine was owned by Cape Asbestos, a British company.

Schepers (1965) found deplorable working conditions at Penge:

> "Exposures were crude and unchecked. I found young children, completely included within large shipping bags, trampling down fluffy amosite asbestos, which all day long came cascading down over their heads. They were kept stepping lively by a burly supervisor with a hefty whip. I believe these children to have had the ultimate of asbestos exposure. X-ray revealed several to have radiologic asbestosis with cor pulmonale before the age of 12.
>
> ... During my 1949 survey I found that a number of employees of these asbestos mills [in the NE Transvaal] who had symptoms of marked pleural sclerosis. The majority were Negro [sic] labourers, but one was a white manager. In some instances the pleura was more than an inch thick and very radiodense. At that time we had not yet been alerted to the possibility of pleural mesotheliomatosis, and it was merely my suspicion that these thickened pleurae represented sequelae of empyema or pulmonary tuberculosis. After I saw my first example of pleural mesothelioma, I 'knew' what had occurred."[5]

Sluis-Cremer (1965, 1970) analyzed the post-mortem findings of black workers who had died while employed in the northern Cape and Penge mines. South African legislation requires that deceased miners receive a post-mortem examination of their cardiovascular organs, subject to permission of the miner's next of kin. In practice, most deceased white miners receive such post-mortems. However, there is no follow-up system to ensure compliance with post-mortem legislation for black miners (Frumkin et al., 1989). The result is that, generally, only those black workers who die while in employment receive these statutory post-mortems. The cause of death of most of the deceased black workers studied by Sluis-Cremer was thus injury (generally, occupational accidents) or infection (typically, pneumonia). Despite the limited occupational experience of these black miners in the two Sluis-Cremer studies, (4.1 and 6.6 years, respectively), the group still exhibited very high rates of asbestosis (80% and 60%), as shown in Table 2.

TABLE 2. Asbestosis at Routine Post-Mortem Examination of Deceased Black Miners from the Northern Cape and Penge Asbestos Mines*

Year of post-mortem	Total (n)	Asbestosis		Mean age (years)	Mean service (years)
Transvaal amphibole mines					
1959-1964	64	51	80%	43	4.1
1965-1969	139	89	60%	45	6.6
Cape crocidolite mines					
1959-1964	87	62	72%	43	3.5
1965-1969	124	44	36%	45	7.4

* Sluis-Cremer, 1965, 1970.

[5] See also the account by Schepers given to Flynn (1992: p. 193).

Regarding lung cancer, Oettle (1964), in his survey, could not find an increased risk of this disease in the Transvaal asbestos mining areas. However, when Webster (1965) analyzed the autopsy records of 69 deceased amosite mine workers who had died within the period circa 1959-64, he noted that two of nine whites and four of sixty black miners had died of carcinoma of the lung. Although lacking formal analysis, this finding seems to suggest an abnormally high proportional mortality for lung cancer among amosite miners of that period.

At the same time, an orthodoxy developed among establishment scientists in South Africa in the 1960s, that mesothelioma did not result from exposure to Transvaal amphibole fibers. The view was articulated most strongly in papers presented by Sluis-Cremer and Webster at the 1965 conference of the New York Academy of Sciences. Two factors contributed to the development of this position. First, the large number of mesotheliomas identified in South Africa came from the northern Cape — none from the Transvaal fields. In evaluating this argument, however, one has to take account of the crucial role played by the Cape-based Sleggs in identifying and following up cases and raising awareness of the disease among researchers. Sleggs was also a key field worker in the PRU study (Sluis-Cremer, 1965).

Second, it was presumed that almost all cases of mesothelioma in South Africa were being reported to the PRU. Sluis-Cremer (1965), for example, argued that "medical facilities for case finding are certainly adequate in the [Transvaal] area," while Webster (1965) stated that "there is an awareness of the diagnosis of mesothelioma in South Africa, and possible cases are usually brought to our notice."

However, in the most recent formal study of the subject, a cohort mortality analysis restricted to white miners, four mesotheliomas were reported among 3212 amosite miners, and two among 90 with mixed Transvaal amphibole exposure (Sluis-Cremer et al., 1992). Furthermore, recent pathology records from the northern Transvaal suggest that a higher incidence of mesothelioma (i.e., higher than has long been believed) may be attributable to previous asbestos mining in the area. In the 15 months from February 1989 to April 1990, 16 suspected mesotheliomas were noted at the Pietersburg branch of the South African Institute of Medical Research.[6] Eleven of these cases occurred among African persons, four of whom were women. These cases merit further investigation.

Penge's Marginal Neighbors: The Pietersburg Asbestos Fields

Mills in the Pietersburg fields (amosite and Transvaal crocidolite) were situated in valleys, often alongside streams and what later became the major roads of the

[6] H.J. Van Rensburg (1990), personal communication.

area. African miners settled close to the mills. Over the years, these remote, rural settlements grew into well established villages. In common with the conditions endured by such communities under apartheid, they had no piped water, no electricity, impoverished school and health care facilities, and were served by rutted gravel roads.

The marginal Pietersburg mines first received attention from state officials when Prof. S.F. Oosthuizen chaired a Department of Mines Committee which examined pulmonary disability and tuberculosis. The committee visited smaller mines such as those on the Pietersburg fields to study the relationship between pneumoconiosis and tuberculosis. Working conditions must have been dire for a committee appointed by the apartheid government to comment as critically as they did: "Although the Committee realizes that these small concerns are faced with innumerable practical difficulties, the Committee was shocked to learn of an[d] observe the very appalling conditions which prevail on these mines, particularly on the asbestos mines in the Pietersburg area. No effort is made to house and feed Native employees and disease and malnutrition is [are] rife" (Oosthuizen, 1955).

The Committee made several proposals to alleviate the problems noted. They recommended that thorough initial and final clinical examinations be made compulsory, and that "periodical visits to the mines by a Bureau mobile X-ray unit to conduct general surveys of all Native employees" be conducted (Oosthuizen, 1955).

Little action seems to have resulted from these recommendations. It was nearly 30 years later, in 1988, before a mobile x-ray unit was first utilized in conducting a survey of disease caused by environmental exposure to asbestos. The study was carried out at Mafefe, the most densely populated (population 12,000) district in the Pietersburg Fields (Felix et al., 1990).

At least nine mills operated in the Mafefe district, each creating a large asbestos waste dump. The communities in the region were never informed of the hazards of asbestos. After the mills closed, the abandoned dumps provided building material for the local people. Asbestos tailings were either mixed with clay to plaster mud homes or mixed with cement to make bricks. In 1987, half of all public buildings and 643 (36%) homes had been constructed with asbestos tailings. The grounds surrounding these houses were inadvertently contaminated with asbestos fibers during construction.

In a collaborative research project, Felix et al. (1990) studied the extent of disease. A census had been conducted to develop a universe from which a random sample of adults older than 19 years of age who had spent most of their life in Mafefe, was drawn. A total of 611 adults were x-rayed: 245 (44%) had pleural changes. Of the sample, 389 (64%) had environmental exposure alone and 222 (36%) had additional occupational exposure. In the occupationally exposed group, the prevalence of pleural plaques was 49%, significantly greater than those with environmental exposure only (34%). The prevalence of pleural plaques increased with age, reaching 54% among persons over 60 years of age.

In addition to lobbying for a comprehensive reclamation program, the researchers had fiber measurements taken using personal and strategic samplers. The mean of 213 samples was 10,302 fibers/m^3 (Rendall, 1990). This is below the South African Government Mining Engineer's recommended standard of 20,000 fibers/m^3 (1/100 of the South African mine standard of 2 fibers/ml). The high rate of pleural and parenchymal changes found in the study population from purely environmental exposure brings into question the safety of these standards.

An important part of the survey was the training of a group of health workers who would educate the community about asbestos hazards. The committee refers people with possible asbestos-related disease to a clinic convened by the National Centre for Occupational Health. The clinic is held once a month at the nearest hospital, which is a one-and-a-half hour drive from Mafefe.

In the first year since the inception of the clinic, the cases of 200 former miners have been the subject of compensation determinations. Ninety-four of the miners (47%) have been certified with pneumoconiosis,[7] 30 (15%) with pneumoconiosis plus tuberculosis, and 36 (18%) with tuberculosis.[8] The large proportion of cases that have been certified illustrates a significant burden of occupational disease experienced by people residing in the area. Owing to limited clinic facilities, hundreds of former miners in Mafefe alone are waiting to be assessed.

The overwhelming local response to the initiative also highlights the historical neglect of the health of South Africa's rural and migrant workers (Davies, 1993). Previously, not a single case of occupational lung disease had been reported by the local hospital. Further, during the year of operation of the clinic no cases have been referred to the medical bureau of certification by the medical staff of the hospital. All the cases identified have followed from the work of community health groups.[7]

BEYOND MINING: URBAN MILLING AND TRANSPORT

Milling was not restricted to the mining localities. Collins, the principal government medical officer at the Durban Chest Clinic, traced the activities of an

[7] This could include other pneumoconioses, principally silicosis from gold mining work.

[8] Prof. J.C.A. Davies (1992). "Compensating mineworkers in Lebowa" (unpublished report).

asbestos "refinery" which operated between 1951 and 1964 near Durban, a major harbor. The plant milled crocidolite, chrysotile, and amosite. The investigation by Collins (1967) showed that despite the urban location of the plant, it had much in common with mills in remote areas:

> "The ventilation was hopelessly inadequate, and there was no dust extraction equipment. Semi-crude material was fed by hand into the mills, passed to cyclones which screened off most of the dust ... and finally emerged from chutes to be bagged by hand, being rammed tight with wooden clubs; or to be blended on the floor with shovels before bagging.
>
> The dust within the building resembled a dense fog, and could be seen escaping into the atmosphere through the entrance ... On one particularly gusty day, the adjacent railway siding was reminiscent of a desert sandstorm as the waste dust was tipped into open trucks, and the workers engaged in this task were blue figures with only eyes and mouth recognizable behind a dusty mask."

High turnover at the mill meant that only 22 men had more than three years of exposure. The mean exposure of this group was 7.6 years. Twelve had lung changes consistent with asbestosis. Only five had normal chest x-rays. Collins was the first to publish practical recommendations locally to minimize exposure. These included compulsory registration of all processes which utilized asbestos, measures to ensure that asbestos containers were dustproof, and the use of harmless substitutes wherever possible. The key to success, however, ultimately resulted from the forthright action of Collins to have the plant closed: no further exposure followed. Regulations directed at specific control of asbestos in the non-mining sector were promulgated belatedly in 1987 (Republic of South Africa, 1987)

The transportation of fiber to international markets created its own trail of disease. In 1985, Myers and colleagues examined a link in the transport chain: stevedores at the Port Elizabeth harbor who intermittently loaded about two cargoes per month of crocidolite onto ships. Prior to the 1970s, the crocidolite was transported in jute (hessian) bags. Thereafter, it was transported in impermeable plastic weave bags which were nevertheless frequently damaged through careless handling, dashed against the sides of ships, or punctured by forklifts. In their cross-sectional study, Myers (1985) found that 30% of the stevedores had x-ray abnormalities consistent with asbestosis. This result was in contrast to an initial review of the x-rays by local physicians and specialist radiologists (independent of the researchers) who did not identify a single case of possible asbestosis.

CONTROL

From the turn of the century, dust control measures and compensation for pneumoconiosis were developed for the South African gold mines. In contrast, controls over asbestos mines were woefully inadequate. Most of the original mine owners (until they sold their assets in the 1980s) were based overseas. They included Cape Asbestos (United Kingdom), Turner and Newall (United Kingdom), and Eternit (Switzerland). These companies were subject to regulation in their European plants from 1930. Yet the first steps by government to control dust in South African asbestos mines and mills occurred only in the 1950s. This concluding section examines why controls on asbestos mines developed so much later than in the gold mines.

The Miners' Phthisis Commission of 1903 was appointed shortly after the Anglo-Boer War, when it was discovered that "of the 1177 rock drill [white] miners employed in the Witwatersrand mines prior to the War, 225 or 16.75% had died [of lung diseases] during the two and a half years" (Transvaal Government Mining Engineer, 1902). Over the next two decades, militant action by white mine trade unions led to numerous official investigations into silicosis and, accordingly, the introduction of control measures. Starting with a series of Acts between 1912 and 1916, the government legislated control and compensation measures for "miner's phthisis." Pre-employment and periodic medical examinations were introduced in 1916 with the establishment of the Miners' Phthisis Bureau (later, the Silicosis Medical Bureau, and currently, the Medical Bureau for Occupational Diseases). In the same year, a dust inspectorate was established (Burke et al., 1977).

Agitation by white miners over occupational disease culminated in the addition of compensation for tuberculosis in 1912 and chronic obstructive airways disease in 1973. On the asbestos mines, however, a far lower percentage of employees were white than on the gold mines, and of those who were employed, few were involved directly in blasting operations. The membership of the (white) Mine Workers' Union on the asbestos mines was accordingly low and the union was not especially active in demanding improved conditions on these mines.

Control measures on the gold mines focused on underground conditions where the majority of workers were employed. It was only in 1946 that surface gold mills were recognized by statute as dusty workplaces (du Toit, 1970b). This influenced the approach of state officials to the asbestos mines, in that underground workings received attention to the neglect of the mills. From the early work of Slade (1931) onward, it was clear that dust counts in the surface mills were high compared to the underground workings (see also Kuyper, 1970). Importantly, milling was a labor-intensive operation.

In South Africa, the Government Mining Engineer (GME) is the official who is responsible for control over dust in asbestos mines and mills, and is also in

charge of the mines inspectorate. Underground dust sampling on the asbestos mines by the inspectorate started only in 1940, while the first surface readings were taken in 1947 (du Toit, 1970a).

The Department of Mines survey of asbestos miners in 1949, to which Schepers contributed, has been mentioned. The results were so disturbing that asbestos mines were required to be registered from 1953, in order to facilitate medical assessments and make workers eligible for compensation.[9] The GME took the unprecedented step of temporarily halting production at certain mines until a commitment to reduce dust levels was received from their managements (Flynn, 1992). In 1956, the GME introduced the first dust standard, a maximum permissible concentration of 300 particles[10] per ml. These actions contributed to the refurbishing of 20 mills in the years up to 1969 (Kuyper, 1970).

During this period, mine management representatives strongly resisted the scientific evidence that asbestos was linked to cancer. Kuyper (1970) of Cape Asbestos argued at the 1969 Johannesburg Conference that "the industry's engineers are still reluctant to agree with the alleged relationship between asbestos dust (principally crocidolite dust) and mesothelioma or that a transient encounter with this dust could lead to the development of the disease 20-30 years later; or that in consequence of this there is likely to be an increase in the number of cases of mesothelioma in the foreseeable future."

Although the first asbestos dust standard had been introduced in 1956, Kuyper (1970) stated 13 years later that mines had not yet met this standard: "Our *current* objective ... is to lower dust levels at the mines to below an *arbitrary* [sic] standard ... and to eliminate the danger of unnecessarily exposing people *not engaged* in mining asbestos to a dust hazard."

The maximum permitted dust standard was more clearly defined in 1970 as 45 fibers/ml and 255 particles/ml. The standard was lowered in 1973 to 12 fibers/ml and 200 particles/ml. In August 1976, separate standards were introduced for underground and surface workings: 5 fibers/ml for underground workings and 12 fibers/ml for surface. A few months later, in December 1976, the surface standard was further decreased to 10 fibers/ml. At the end of 1981 the standard was reduced to 2 fibers/ml underground and 5 fibers/ml on surface. Only in 1984 was a uniform underground and surface standard of 2 fibers/ml introduced, which

---

9 J.M.O. Van Sittert (1992), personal communication.

10 Particles were classified into two categories, 'fiber needles' and 'particles'. A fiber needle was defined as a rod-like particle with a width of less than 5 microns and a length between 5 and 100 microns. A 'particle' was defined as a more or less spherical particle of respirable dust with a diameter between 0.5 and 5 microns (Kuyper, 1970).

remains the current level. No distinction was ever made with regard to commercial fiber type.

The South African mining limits were not onerous by international standards: in the United Kingdom and the United States, the 2 fibers/ml standard had been required by 1969 and 1976, respectively. Even so, it is clear that individual mines did not comply with the limits set by the GME. Dust counts taken in the Penge mill, for example, remained well above the 1976 standard of 12 fibers/ml throughout the second half of the 1970s. Control measures finally brought the counts down to around 2 to 3 fibers/ml by 1983 (Dempster, 1984).

Mine management states that currently 80% of workplaces are in compliance with the standard of less than 1 fiber/ml (see footnote 1). The risk of exposure is described as being greatest in packaging, a dry, labor-intensive process in which workers handle asbestos in pure, fiberized form.

In South Africa, control of atmospheric pollution caused by mining operations is provided for by the Atmospheric Pollution Prevention Act of 1965. As with the pneumoconiosis legislation, only gold mining areas were scheduled under this statute; the waste produced by asbestos mines was neglected. No substantial environmental control of abandoned asbestos tailings dumps — which had been "forgotten" by GME officials — occurred until 1984 (Felix, 1991). In that year, extensive media coverage highlighted the pollution caused by waste dumps and the attendant risk to nearby communities, particularly communities surrounding the Pietersburg asbestos fields. As the chairman of the Msauli (chrysotile) mine noted in the company's *Annual Report* (Msauli, 1985): "In view of the unrelenting negative publicity given this year to the health issue, a special section of this report has been devoted to the subject." The media coverage resulted in pressure on the Department of Mineral and Energy Affairs (formerly Mines) and the mining companies to undertake an abatement program of waste dumps.

CONCLUSION

The mining and milling of asbestos in South Africa led to three local epidemics in which thousands of persons suffered ill health or premature death following occupational or environmental exposure. The gaps in our epidemiological knowledge mean that the gravity of these asbestos epidemics will never be fully recorded. What is clear is that where scientists conducted sound studies, high proportions of the populations surveyed were found with asbestos-related disease. This contrasted with the relatively small number of cases of asbestos-related disease diagnosed by standard medical services serving these populations.

Comparison of the state controls over the asbestos industry with those on the gold mines shows the crucial role of worker involvement in ensuring precautions against hazards. Both mining sectors faced similar disease problems, but controls in the asbestos industry lagged decades behind the gold mines. The force behind reforms on the gold mines was the politically powerful Mineworkers' Union (MWU). It gained representation on the state institutions that oversaw compensation and research into lung diseases in the gold mines. Black workers were never represented on these institutions, although they benefited from some of the reforms that were introduced. In the case of the asbestos mines, however, the MWU never had a large constituency and, therefore, did not apply the same pressures for control measures.

As elsewhere, scientific knowledge was a necessary, but by no means sufficient, condition for control of the asbestos hazard. The companies that mined and milled asbestos in South Africa were predominantly British and West European. Studies conducted in their metropolitan plants highlighted the risk of asbestosis from the beginning of the twentieth century, and the cancer hazard from at least the 1950s. Yet in their South African operations, this medical knowledge was either not applied, or was applied sporadically. Such was the disregard of the hazards that, as late as 1949, one British-owned company employed children inside jute sacks to trample and compress asbestos prior to shipment.

The double standards applied by these first world-owned companies gives the lie to the efficacy of self-regulation. The belated controls over asbestos mining that were introduced in the 1950s resulted from government initiatives. It was also pressure from community groups and from press coverage in the 1980s that prompted steps to abate the hazards caused by abandoned waste tailings.

ACKNOWLEDGMENTS

We are grateful to the following persons who gave of their time and knowledge: Prof. J.C.A. Davies, Mr. H.P. Hart, Mr. P.A. Van Zijl, Mr. J.M.O. Van Sittert, Dr. R.S.J. du Toit, Ms. G. Reid, Mr. R.E.G. Rendall, Ms. L. Porter, Prof. I. Webster, and Dr. S. Candy.

REFERENCES

BOTHA, J.M., IRWIG, L.M., and STREBEL, P.M. (1986). "Excess mortality from stomach cancer, lung cancer, and asbestosis and/or mesothelioma in crocidolite mining districts in South Africa." Am. J. Epidemiol. 123: 30-40.

BURKE, G. and RICHARDSON, P. (1979). "The migration of miners' pthisis between Cornwall and the Transvaal, 1876-1918." In: Labour, Townships and Protest (B. Bozzoli, ed.). pp. 219-247. Ravan Press, Johannesburg.

COLLINS, T.F.B. (1967). "Asbestos - the lethal dust." S. Afr. Med. J. 41: 639-646.

COUNCIL FOR SCIENTIFIC AND INDUSTRIAL RESEARCH (CSIR). Pneumoconiosis Research Unit (PRU). (1964). Field Survey in the North Western Cape and at Penge in the Transvaal (Asbestosis and Mesothelioma). Report No. 1/64, Pneumoconiosis Research Unit, Johannesburg.

DAVIES, J.C.A. (1993). "Occupational lung disease "(letter). S. Afr. Med. J. 83: 64.

DE KUN, N. (1965). The Mineral Resources of Africa. Elsevier, Amsterdam.

DEMPSTER, C. (1984). "Penge not charged for illegal fibre levels." The Star (Johannesburg). 17 August: 4.

DU TOIT, R.S.J. (1970a). "Dust in South African asbestos mines and fiberizing plants." In: Pneumoconiosis, Proceedings of the International Conference, Johannesburg 1969 (H.A. Shapiro, ed.). pp. 13-17. Oxford University Press, Cape Town.
DU TOIT, R.S.J. (1970b). "Period of service before certification for pneumoconiosis on South African gold mines." In: Pneumoconiosis, Proceedings of the International Conference, Johannesburg 1969 (H.A. Shapiro, ed.). pp. 470-472. Oxford University Press, Cape Town.
FELIX, M.A. (1991). "Risking their lives in ignorance - the story of an asbestos polluted community." In: Going Green: People, Politics and the Environment in South Africa. (J. Cock and E. Koch. eds.). pp. 33-43. Oxford University Press, Cape Town.
FELIX, M.A., MABILETJA, Z.M., ROODT, L., CARLIN, A.W.J., and STEINBERG, M. (1990). "Aftermath of asbestos mining - health effects of fibres in the environment." In: Proceedings of the First International Union of Air Pollution Prevention Associations (IUAPPA) Regional Conference on Air Pollution, Johannesburg 1990. Vol. 2, paper 81. National Association for Clean Air, Pretoria.
FLYNN, L. (1982). "South Africa blacks out blue asbestos risk." New Scientist. 22 April: 237-239.
FLYNN, L. (1992). Studded with Diamonds and Paved with Gold: Miners, Mining Companies and Human Rights in Southern Africa. Bloomsbury, London.
FRUMKIN, H., MYERS, J.E., and BACHMANN, O.M. (1989). "Periodic examination of South African mine workers" (letter). J. Occ. Med. 31: 563-564.
GILSON, J.C. (1970). "Asbestos exposure in South Africa." In: Pneumoconiosis, Proceedings of the International Conference, Johannesburg 1969 (H.A. Shapiro, ed.). pp. 213-214. Oxford University Press, Cape Town.
HALL, A.L. (1930). Asbestos in the Union of South Africa. Geological Survey Memoir No. 12. Union of South Africa, Department of Mines and Industries, Pretoria.
HART, H.P. (1988). "Asbestos in South Africa." J. S. Afr. Inst. Min. Metall. 88: 185-198.
HART, H.P. (1992). "Mines move to combat inflation." Engineering News Mining Supplement. 27 November: 5.
IRWIG, L.M., DU TOIT, R.S.J., SLUIS-CREMER G.K., SOLOMON, A., GLYN THOMAS, R., HAMEL, P.P.H., WEBSTER, I., and HASTIE, T. (1979). "Risk of asbestosis in crocidolite and amosite mines in South Africa." In: Health Hazards of Asbestos Exposure (I.J. Selikoff, E.C. Hammond, eds.). Ann. N.Y. Acad. Sci. 330: 35-52.
KUYPER, I.N. (1970). "Dust problems in the mining, milling and packaging of asbestos." In: Pneumoconiosis, Proceedings of the International Conference, Johannesburg 1969 (H.A. Shapiro, ed.). pp. 42-50. Oxford University Press, Cape Town.
MARTINY, O. (1956). "Primary mesothelioma of the pleura." Proc. Transvaal Mine Medical Officers' Assoc. 35: 63-64.
MAY, T.C. (1965). "Statistical tables - asbestos." Ann. N. Y. Acad. Sci. 132: 745-765.
McDERMOTT, M., BEVAN, M.M., ELMES, P.C., ALLARDICE, J.T., and BRADLEY, A.C. (1982). "Lung function and radiographic changes in chrysotile workers in Swaziland." Br. J. Ind. Med. 39: 338-343.
MEREWETHER, E.R.A. and PRICE, C.W. (1930). Report on the Effects of Asbestos on the Lungs and Dust Suppression in the Asbestos Industry. HM Stationery Office, London.
MSAULI ASBES BEPERK. (1985). 33rd Annual Report. Johannesburg.
MYERS, J. (1980). "Asbestos and asbestos related disease in South Africa." Working Paper No. 28. South African Labour and Development Research Unit, University of Cape Town, Cape Town.
MYERS, J.E., GARISCH, D., MYERS, H.S., CORNELL, J.E., and RWEXU, R.D. (1985). "A respiratory epidemiological study of stevedores intermittently exposed to asbestos in a South African port." Am. J. Ind. Med. 7: 273-283.
OETTLE, A.G. (1964) "Cancer in Africa, especially in regions south of the Sahara." J. Nat. Cancer. Inst. 33: 383-439.
OOSTHUIZEN, S.F. (1955). Report of the Departmental Committee of Enquiry into the Relationship between Silicosis and Pulmonary Disability and the Relationship between Pneumoconiosis and Tuberculosis. Government Printer, Pretoria.
REID, G., KIELKOWSKI, D., STEYN, S.D., and BOTHA, K. (1990). "Mortality of an asbestos-exposed birth cohort - a pilot study." S. Afr. Med. J. 78: 584-565.
RENDALL, R.E.G., FELIX M.A., MOGOMOTSI, E., and MABELITJA. Z. (1990). "Asbestos in the air around abandoned mines in north eastern Transvaal." In: Proceedings of the First International Union of Air Pollution Prevention Associations (IUAPPA) Regional Conference on Air Pollution, Johannesburg 1990. Vol. 2, paper 80. National Association for Clean Air, Pretoria.
REPUBLIC OF SOUTH AFRICA. (1983). Machinery and Occupational Safety Act. No. 6 of 1983. Government Gazette 213 (8572): 1-42.
REPUBLIC OF SOUTH AFRICA. (1987). Department of Manpower. Asbestos Regulations. No. R. 773. Government Gazette 262 (10700): 1-10.
REPUBLIC OF SOUTH AFRICA. (1991). Minerals Act. No. 50 of 1991. Government Gazette 311 (13253): 1-80.
SCHEPERS, G.W.H. (1965). "Asbestosis in South Africa - certain geographical and environmental considerations." Ann. N.Y. Acad. Sci. 132: 246-247.
SIMSON, F.W. (1928). "Pulmonary asbestosis in South Africa." Brit. Med. J. Part I: 885-887.
SLADE, G.F. (1931). The Incidence of Respiratory Disability in Workers Employed in Asbestos Mining, with Special Reference to the Type of Disability Caused by the Inhalation of Asbestos Dust. M.D. thesis. University of the Witwatersrand, Johannesburg.
SLEGGS, C.A. (1960). "Clinical aspects of asbestosis in the Northern Cape." In: Proceedings of the Pneumoconiosis Conference, Johannesburg 1959 (A.J. Orenstein, ed.). pp. 383-390. J. & A. Churchill, London.
SLEGGS, C.A., MARCHAND, P., and WAGNER, J.C. (1961). "Diffuse pleural mesotheliomas in South Africa." S. Afr. Med. J. 35: 28-34.
SLUIS-CREMER, G.K. (1965). "Asbestosis in South Africa - certain geographical and environmental considerations." Ann. N.Y. Acad. Sci. 132: 215-234.
SLUIS-CREMER, G.K. (1970). "Asbestosis in South African asbestos miners." Environ. Res. 3: 310-319.
SLUIS-CREMER, G.K., LIDDELL, F.D.K., LOGAN, W.P.D., and BEZUIDENHOUT, B.N. (1992). "The mortality of amphibole miners in South Africa, 1946-1980." Br. J. Ind. Med. 49: 566-575.
SOUTH AFRICAN INSTITUTE FOR MEDICAL RESEARCH (SAIMR). (1930). Annual Report for the Year 1929. Johannesburg.
TALENT, J.M., HARRISON, W.O., SOLOMON, A., and WEBSTER, I.A. (1980). "A survey of black mineworkers of the Cape crocidolite mines." In: Biological Effects of Mineral Fibres (J. C. Wagner, ed.). Vol. 2. pp. 723-729. International Agency for Research on Cancer, Lyon.
TRANSVAAL GOVERNMENT MINING ENGINEER. (1902). Half Yearly Report. Mines Department. Government Printing Works, Pretoria.

WAGNER, J.C. (1960). "Some pathological aspects of asbestosis in the Union of South Africa." In: Proceedings of the Pneumoconiosis Conference, Johannesburg 1959 (A.J. Orenstein, ed.). pp. 373-382. J. & A. Churchill, London.

WAGNER J.C., SLEGGS, C.A., and MARCHAND, P. (1960). "Diffuse pleural mesothelioma and asbestos exposure in the northwestern Cape Province." Brit. J. Indust. Med. 17: 260-271.

WEBSTER, I. (1965). "Mesotheliomatous tumours in South Africa: pathology and experimental pathology." Ann. N.Y. Acad. Sci. 132: 623-646.

ZWI, A.B., REID, G., LANDAU, S.P., KIELKOWSKI, D., SITAS, F., and BECKLAKE, M.R. (1989). "Mesothelioma in South Africa, 1976-84: incidence and case characteristics." Int. J. Epid. 18: 320-329.