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Mineralogy and Malignant Mesothelioma: The South African Experience

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<http://dx.doi.org/10.5772/47974>

1. Introduction

South Africa is a uniquely mineral rich country. Of the six types of asbestiform minerals found in the country, three, namely crocidolite, amosite and chrysotile were mined and milled on a large commercial scale. Asbestos was used locally in South Africa, but the majority of its production was exported worldwide. In the 1970s, South Africa was the world's third largest producer of asbestos, behind Canada and the USSR. About 97% of the world's production of crocidolite and virtually all of the amosite came from South Africa.

The output from the South African asbestos mining industry peaked at 380,000 tonnes in 1977 and declined thereafter as export markets declined due to restrictive legislation in countries that imported asbestos (Virta, 2006; Kielkowski et al., 2011). Legislation in South Africa banning the use of all types of asbestos came into effect in 2008, well after the last asbestos mine ceased production in 2001 and closed in 2002. Although South Africa benefitted financially from the exploitation of its asbestos mineral reserves, the revenue from asbestos never accounted for more than 3% of the value of its total minerals output (McCulloch, 2003). There is however a high price to pay in terms of a legacy of disease and environmental contamination through mining activities and the transport of asbestos and asbestos containing products.

This account records, in the main, work done in Johannesburg at the National Institute for Occupational Health (NIOH) - formerly the Pneumoconiosis Research Unit (PRU), thereafter, the National Research Institute for Occupational Diseases and later the National Centre for Occupational Health - at the Medical Bureau for Occupational Diseases and its Division of Epidemiology Research. All the authors have spent the major part of their professional careers working at the NIOH.

All types of asbestos are crystalline silicates. Chrysotile, known locally in South Africa as white asbestos, occurs in ultramafic rock formations. It is a hydrated magnesium silicate and

differs from the other types of asbestos in that it has serpentine fibres and contains only the one cation: magnesium. The other types of asbestos have straight fibres and are called amphiboles. The amphiboles all contain iron and combinations of other cations – sodium, magnesium and calcium. Crocidolite, also known as Riebeckite and locally as blue asbestos, occurs in banded ironstone formations. It contains the cations sodium, magnesium and iron. Amosite (an acronym for Asbestos Mines of South Africa), also known as Grunerite and locally as brown asbestos, occurs in banded ironstone. It contains the cations magnesium and iron. Because of their different chemical composition and crystalline structure, the different types of asbestos have different physical properties. Commercially they were used for different purposes. Chrysotile was preferred for manufacturing friction linings, asbestos cement, textiles, ropes and yarns. Amosite was used where long fibres were required. It is much more resistant to acids and sea-water than chrysotile. In compacted form it was applied as a covering for marine turbines and jet engines. It was also used in blanket form for insulation in high temperature applications. Crocidolite has a high tensile strength and was used as insulation from very high temperatures. Long crocidolite fibres were used for boiler lagging, acid resistant packings and gaskets. Short crocidolite fibres were used in the manufacture of asbestos cement (Hart, 1988). It might be expected that different types of asbestos with different chemical and physical properties would have different potentials to cause disease.

The diseases most firmly attributed to exposure to asbestos are asbestosis, pleural effusions, diffuse pleural fibrosis, pleural plaques, lung cancer and mesothelioma (Figure 1). In South Africa, asbestosis was described as early as 1928 in lung tissue sent to the South African Institute for Medical Research in Johannesburg, for examination. The tissue was obtained at autopsy from asbestos miners who worked in Southern Rhodesia, now known as Zimbabwe (Simson, 1928). The association between mesothelioma and exposure to crocidolite asbestos was published by Wagner, Sleggs and Marchand in 1960 (Wagner et al., 1960). Wagner worked at the NIOH, which was known at that time as the PRU. He was recruited by his brother in law, Ian Webster, who encouraged him to research the adverse health effects of asbestos. Despite the knowledge that inhalation of asbestos fibres could cause disease, exposure levels for miners and millers were poorly controlled (Slade, 1931). Tens of thousands of formally employed miners and millers were exposed to asbestos (McCulloch, 2003). Many workers, including women and juveniles were employed informally by the mines and there are scant if any records of these employees (McCulloch, 2002). In addition, whole communities next to mines were environmentally exposed (Abratt et al., 2004) and in some instances this exposure continues. South Africans in the manufacturing industries were exposed along with artisans such as boiler makers. Asbestos and asbestos products were used on mines that mine other commodities such as gold. In a study of 18 cases of mesothelioma occurring in gold mine workers, 15 were artisans and included boiler makers, fitters, electricians, a plumber and a mason who lined furnaces with asbestos (Davies et al., 1987). Because of the extensive use of building materials that contain asbestos (Phillips et al., 2007) (Figure 2), workers in the construction, renovation and demolition sectors have a potential risk of exposure to asbestos through their work (Figure 3).

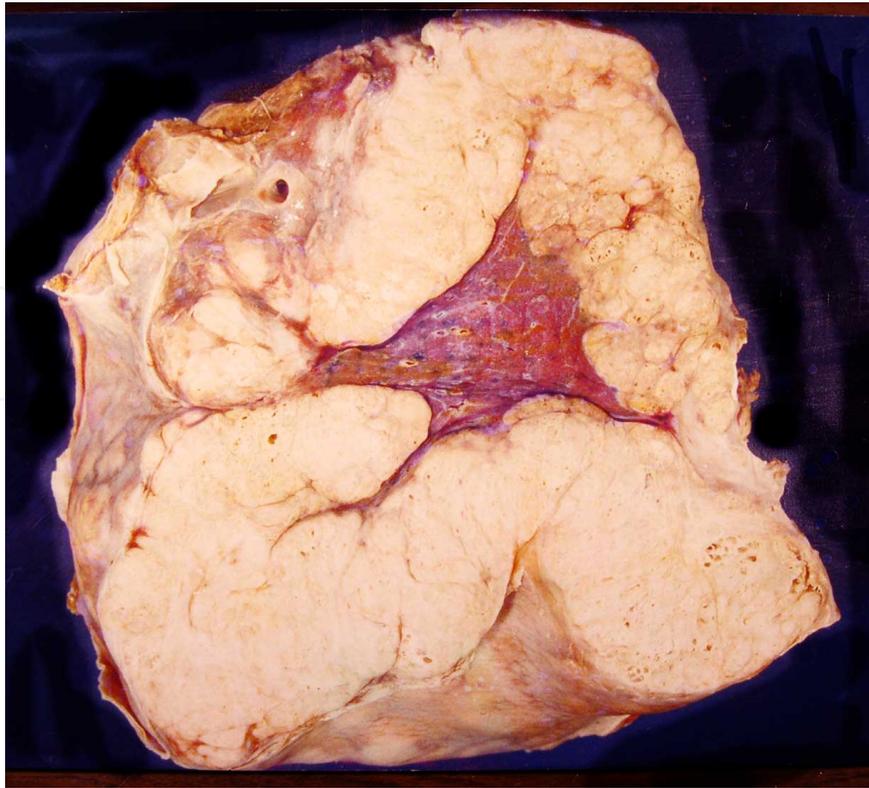


Figure 1. Gross specimen of malignant mesothelioma of the pleura. (Courtesy of NIOH archive).



Figure 2. Typical house in Soweto near Johannesburg built circa 1960 with an asbestos cement roof. (Courtesy of Professor JI Phillips).



Figure 3. Detail of asbestos roof sheet in Soweto, showing damage and exposed asbestos fibres. (Courtesy of Professor JI Phillips).

Although trends in mortality due to mesothelioma in the general population have been documented in other countries (Hodgson et al., 2005; Nishikawa et al., 2008), only two South African studies have quantified the burden of asbestos-related cancers in the general population. From 1976 to 1984, estimated incidence rates for mesothelioma averaged over this period were amongst the highest for a general population anywhere in the world. For white, mixed race and black men, the standardized incidence rates for mesothelioma per million population per year aged 15 and over were calculated to be 32.9, 24.8 and 7.6 respectively and 8.9, 13.9, and 3.0 for white, mixed race and black women respectively (Zwi et al., 1989). These figures reflect predominantly occupational exposure in men and predominantly environmental exposure in women.

Mesothelioma mortality rates in the general population of South Africa have been calculated from 1995 to 2007. The age adjusted mortality rates remained stable for the period and ranged from 11 to 16 per million per-year for men and 3 to 5 per million per year for women. The data for this study were not broken down into racial groups and are therefore not directly comparable to the 1976 to 1984 study. However, rates for the period 1995 to 2007 appear to be much lower than expected. The reasons for this are not entirely clear. It may be due to under reporting or to competing causes of death relating to the AIDS epidemic in the country (Kielkowski et al., 2011).

There has been a debate that apart from asbestos a virus may be involved in the development of mesothelioma. There has been research into the association of Simian Virus 40 (SV40) and mesothelioma. SV40 was a contaminant of poliomyelitis vaccine grown on cell lines derived from Macaques and was inadvertently administered with the vaccine to many people around the world. South Africa, however, produced its own poliomyelitis vaccine which was grown in vervet monkey kidney cell cultures. Unlike Macaques, vervet monkeys (*Cercopithecus aethiops*) are not a natural host for SV40 and South African vaccines were not contaminated (Malherbe, 1974). Studies on tissue from South African cases of mesothelioma showed no evidence for an etiologic role for SV40 (Manfredi et al., 2005).

The South African experience is of three commercially important asbestos types: crocidolite, amosite and chrysotile. These will be discussed separately in order to set out in detail what is known about the role of each of these fibres in the causation of mesothelioma in South Africa.

In South Africa, crocidolite mining began in 1893, near the town of Prieska, in what was called the North Western Cape and is now known as the Northern Cape Province (McCulloch, 2003) (Figure 4). The asbestos deposits occur along a 450 kilometre line between just south of Prieska to the town of Pomfret which is close to the Botswana border (McCulloch, 2002).

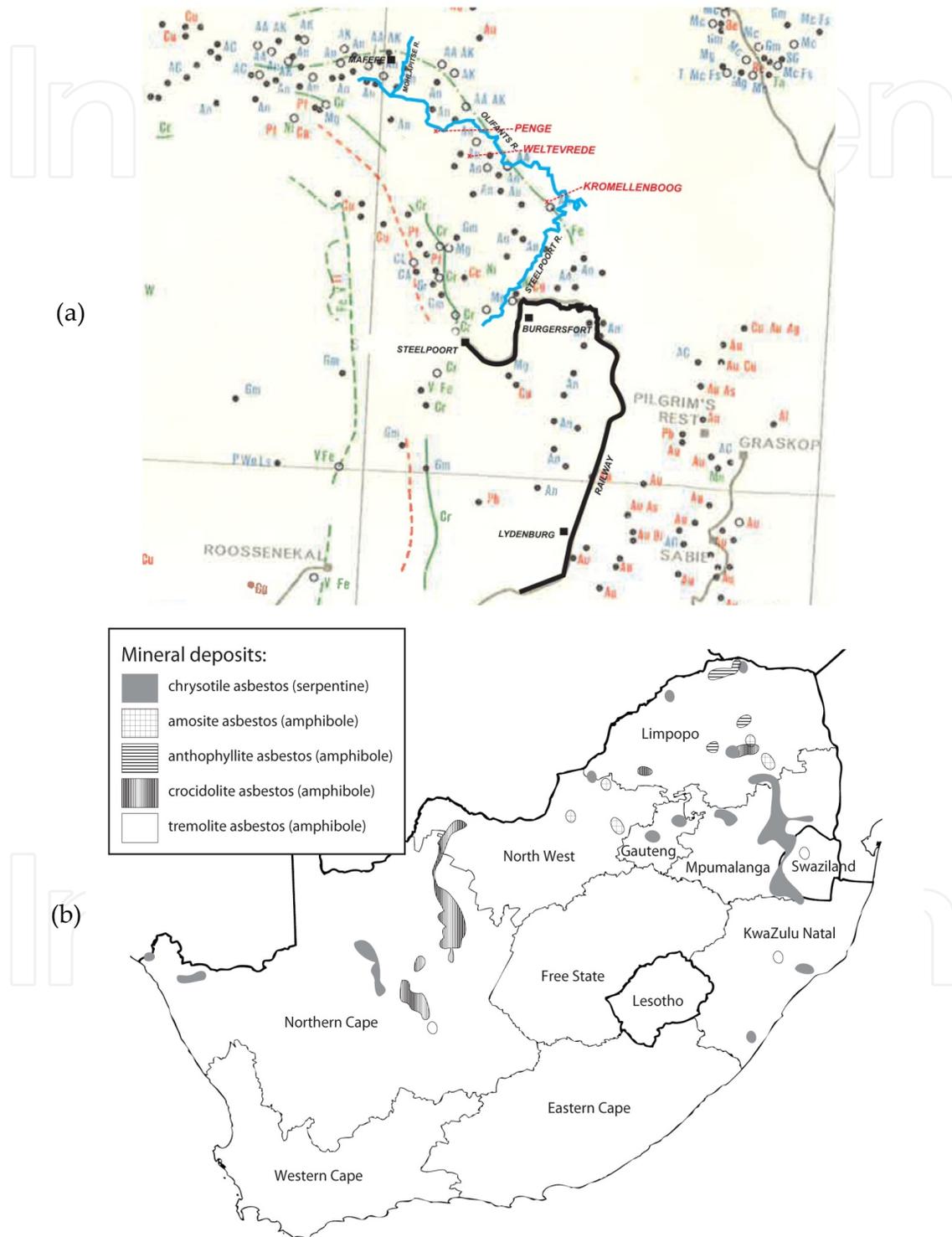


Figure 4. a. Map of the mineral deposits in the Transvaal Crocidolite-Amosite (Pietersburg) asbestos field located on the north-eastern border of the Bushveld Igneous Complex. b. Map of South Africa showing

asbestos deposits. (Courtesy of Dr Gillian Nelson). Key to mineral deposits: AK = Crocidolite, AA = Amosite, AC = Chrysotile, Cr = Chromite, Pt = Platinum, An = Andalusite, Mg = Magnesite, Cu = Copper, V = Vanadium, Pb = Lead, CA = Attapulgitite, Ni = Nickel. Modified from: Mineral Map of the Republic of South Africa, an accompaniment to the 1976 publication Mineral Resources of the Republic of South Africa, Department of Mines, Geological Survey, Government Printer, Pretoria ISBN0621034649.

The Pietersburg asbestos field is located in the Limpopo Province along the northern bank of the Olifants River at its western end and on the southern bank from Penge Mine eastwards to Kromellenboog Mine. It terminates on the northern bank of the Steelpoort River just short of its confluence with the Olifants River (29° 30' - 30° 30' E, 24° - 25° S). With the exception of Penge, all the mines are in rugged country and were served by gravel roads until recently. A tarred road links Penge to the railway at Burgersfort. The Pietersburg field is the most complex in geological terms, and seams of amosite and crocidolite are reported to overlap, and the field is sometimes referred to as the Transvaal Crocidolite-Amosite field (Coetzee et al., 1976). It is the source of almost all of the world's supply of amosite and some crocidolite. There is evidence that in the western portion of the field (west of the Mohlapiitse River) both crocidolite and amosite are found but that in the eastern part only amosite occurs. In addition to amosite and crocidolite, chrysotile deposits were also mined in the area (Coetzee et al., 1976). The occurrence and exploitation of three asbestos types in the region makes it clear that establishing occupational or environmental exposure to any particular fibre type, and especially to amosite fibre only is not easy or reliable. The only way to determine which fibres an individual has been exposed to is to examine and analyse the fibres retained in the lung and determine the lung fibre burden (Figure 5).

There are numerous deposits of chrysotile in the Mpumalanga, Limpopo and Kwa Zulu Natal Provinces. The most important are in the Barberton area of Mpumalanga where large scale chrysotile mining took place. It was in this region that South Africa's last asbestos mine closed in 2002.

Crocidolite

In South Africa, the first description in the medical literature of malignant mesothelioma of the pleura was the presentation of a single case by Dr Olaf Martiny to the February general meeting of the Transvaal Mine Medical Officers' Association, held at the Witwatersrand Native Labour Association Hospital, in Johannesburg, on the 16th February, 1956 (Martiny, 1956). This case presentation was of a 36 year old Botswana male who was admitted to one of the mine hospitals with pleural thickening and an effusion. Initially he was diagnosed with and treated for tuberculosis. The patient's condition deteriorated and he died. The autopsy and subsequent examination of the tissues was performed by Dr Christopher Wagner who was working at the NIOH (Figure 6). Professor B.J.P. Becker was also present at the autopsy examination. Becker and Wagner's diagnosis was a primary malignant mesothelioma of the pleura. The presentation of this case raised awareness of a hitherto rare pleural tumour presenting with some clinical features that were similar to and initially mistaken for tuberculosis.

Although it is not recorded in the original proceedings of this meeting of the Transvaal Mine Medical Officers Association, Wagner states in his thesis (Wagner, 1962) that in addition to the mesothelioma, histological examination of the lungs showed the presence of asbestosis, asbestos bodies and asbestos fibres. This finding indicates that the patient was significantly exposed to asbestos. Much later, Wagner credits his assistant, Mr D E Munday, with the

suggestion to take further sections of the lung which revealed the evidence of asbestos exposure (Wagner, 1991).

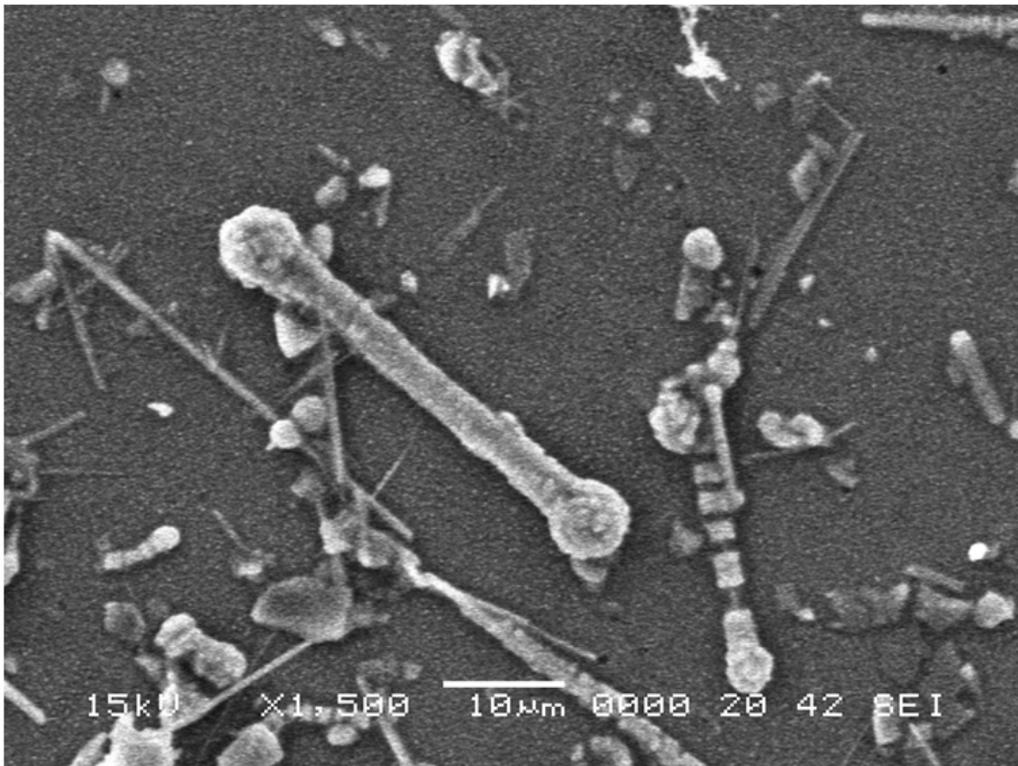


Figure 5. Crocidolite fibres and asbestos bodies from the lung of an asbestos miner. (Courtesy of Professor JI Phillips).

Dr Christopher Sleggs, the medical superintendent of the West End Tuberculosis Hospital, Kimberley, Northern Cape Province, had been concerned about patients with pleural disease who did not respond to the available tuberculosis treatment, and who died. He saw his first patient with what he called atypical tuberculosis in 1952 on a visit to St Konrad's Mission Hospital at Taung and St Michael's Hospital at Bathlaros. He found more cases at Kuruman and began keeping notes on these atypical cases. In 1954, he recorded the histories of two farmers who transported asbestos and were dying of massive pleural tumours. Sleggs observed that patients with pleural tuberculosis coming from areas to the east of Kimberley got better on anti-tuberculosis treatment, but some of those who came from the west, where the crocidolite asbestos fields were, did not respond to treatment and died. Since the early 1950s, Sleggs had referred 12 such patients with clinical features of atypical pleural tuberculosis to thoracic surgeons in Johannesburg, Pretoria, Durban and Cape Town. All were diagnosed as having metastatic carcinomata, not primary mesothelioma of the pleura (McCulloch, 2002).

According to McCulloch, Mr Libero Fatti, the chief thoracic surgical consultant at the Johannesburg General Hospital was called to Kimberley in May 1955 to carry out an emergency operation on an accident victim – a case unrelated to pleural disease. While Fatti was in Kimberley, Sleggs took the opportunity to show him a series of X-rays of what he

called atypical tuberculosis. Fatti offered to investigate these cases and arranged for his partner, Mr Paul Marchand, to perform biopsies. Pleural needle biopsies and later open lung biopsies from patients at the West End Hospital were sent to Dr Ian Webster at what is now the NIOH in Johannesburg, who turned them over to his brother in law, Wagner.



Figure 6. Dr J.C. Wagner. (Courtesy of NIOH archive).

The cases from the West End Hospital formed the basis of the research which led to the publication of two papers (Wagner et al., 1960; Sleggs et al., 1961). The first paper in 1960 reported on 33 cases and the second on 30 of the original cases plus 4 additional cases. By the end of August 1961 Wagner had examined tissue from 78 cases which he collated along with their histories in a table in his thesis (Wagner, 1962). The 1960 paper was to become the most cited paper in the field of occupational health.

All the open lung biopsies from the West End Hospital showed histological features consistent with mesothelioma but it was felt that a definite diagnosis could not be given as "it was not possible to exclude other sites of primary origin"(Wagner, 1962). After

examining another 4 cases, the possibility of a common aetiological agent was considered. Because these patients came from the vicinity of the Northern Cape asbestos field, and because evidence of asbestosis and asbestos bodies were seen in the lung tissue of Martiny's case (Martiny, 1956), asbestos exposure was considered by Wagner to be a possible factor. This hypothesis however, could not be supported from the patients' histories, all of whom denied working with asbestos. Their occupations included housewives, domestic servants, cattle herders, farmers, a water bailiff, an assurance agent and an accountant. Subsequently it was discovered that working with asbestos carried a social stigma for all ethnic groups. In addition, many of the patients who had not worked with asbestos did not appreciate the significance of the asbestos mills and dumps in their vicinity (Wagner, 1962).

While the credit for making the association between exposure to asbestos and mesothelioma is generally ascribed to Wagner, several physicians, surgeons and pathologists played a role. The discovery depended on the biopsy material coming to the NIOH, so perhaps, the defining moment was when Sleggs approached Fatti with the X-rays of cases of atypical tuberculosis. The reason for Fatti's presence in Kimberley was serendipitous - he was there for his surgical expertise which was required by an accident victim - not to investigate pleural disease.

A field study, which was conducted by the NIOH in Prieska, Kuruman and Koegas in the Northern Cape from November 1960 to February 1962, concluded that people who were living or who had lived in proximity to asbestos mines or mills were in danger of contracting asbestosis, even though they had no industrial exposure to asbestos dust inhalation. As it was reported: "an alarmingly high number of cases with mesothelioma of the pleura had been discovered among people who have lived in the Northern Cape and that there is evidence that this condition is associated with exposure to asbestos dust inhalation which need not be industrial" (PRU, 1964).

Subsequent studies have shown that almost all cases of mesothelioma in South Africa are associated with exposure to crocidolite asbestos (Webster, 1973; Cochrane and Webster, 1978; Rees et al., 1999a; Nolan et al., 2006). Webster considered the association with mesothelioma to be a peculiar property of Cape crocidolite (Webster, 1973). In a study of 7317 white male employees in amosite and crocidolite mines, excluding miners of Transvaal crocidolite, it was shown that crocidolite miners were approximately 7 times more likely to develop mesothelioma than amosite miners (Sluis-Cremer et al., 1992).

A case control study of 123 South African cases of mesothelioma showed a preponderance of cases where the exposure was attributed to crocidolite (Rees et al., 1999a). In this study, 5 of the patients had no known history of exposure to asbestos. Of the remainder, 82% were occupationally exposed and 18% environmentally exposed. Of the environmentally exposed patients, 91% had contact with Cape crocidolite. There was a relative paucity of cases linked to amosite and none of the cases could be linked convincingly to chrysotile exposure. The conclusion of this study is that there is a fibre gradient of mesotheliomagenic potential for South African asbestos. The mesotheliomagenic potential for crocidolite is greater than that for amosite which is greater than that for chrysotile.

In an attempt to produce a definitive study of fibre type in cases of histologically proven mesothelioma, inorganic material was recovered from the lung parenchyma of 43 South African cases of mesothelioma. Using analytical transmission electron microscopy the types and concentrations of fibrous minerals were determined. Crocidolite was found to be the most frequently occurring fibre type. In 7 of the 9 cases with more than a million fibres per gram of dried lung tissue, at least 85% of the fibres were crocidolite. Crocidolite occurred alone in 12 of the 33 occupationally exposed cases and in 3 of the 4 environmentally exposed cases. In the fourth environmental case, 96% of the fibres were crocidolite. When the total asbestos concentration in the lung was less than 250,000 fibres per gram of dried lung tissue, crocidolite was the only fibre type identified. The mean concentration of crocidolite for all 43 cases was 270,000 fibres per gram of dried lung tissue. This fibre burden is substantially below the lung burden of chrysotile fibres in general populations without asbestos-related disease (Langer et al., 1971; Langer and Nolan, 1994). This study supports the hypothesis that mesothelioma can develop in individuals following exposures to crocidolite that may be brief or slight (Nolan et al., 2006).

Some types of mining work carried a high risk for developing mesothelioma; an example of this would be cobbing. Crocidolite and amosite occur in banded ironstone which is extremely hard. Cobbers removed adherent ironstone from the ends of cobs of fibre with a hammer in order to prevent the mills breaking down from the impact of the ironstone. They would work sitting all day long hammering at asbestos bearing rock less than half a metre from their breathing zone. A group of 53 women cobbers of crocidolite were examined at St Michael's Mission Hospital in the Northern Cape Province. Twelve of these 53 cobbers developed mesothelioma (Talent et al., 1978).

The mining of Transvaal crocidolite in the Pietersburg field came to an end in 1976 and the mining of crocidolite in the Northern Cape ceased in 1996. During the time the mines were active, exposure levels to asbestos fibres were high (McCulloch, 2002). Records of exposure levels are poor and sparse. A dust survey at the Dublin Consolidated blending plant in Pietersburg found an average concentration of 179 fibres/ml in 1966 and 40 fibres/ml in 1974. At eight small mines in the Limpopo Province around Mafefe the fibre levels varied from 1 to 89 fibres/ml (Felix, 1997).

Studies on data collected on mesothelioma occurring in specific geographical areas of South Africa showed high rates of mesothelioma in areas where Cape crocidolite was mined. A study conducted in five Cape crocidolite-mining magisterial districts was based on death registrations from 1968 to 1980 (Botha et al., 1986). The authors calculated standardised mortality ratios (SMRs) for asbestosis and/or mesothelioma, without distinguishing between the two diseases. Rates, compared to the control group, were significantly elevated for men and women of all races, with SMRs of 7.86 for white men, 10.3 for white women, and 8.43 and 8.72 for men and women of mixed race, respectively.

A birth cohort was established in one of the magisterial districts in which the above study was conducted (Reid et al., 1990; Kielkowski et al., 2000). The cohort comprised men and women whose births were registered from 1916 to 1936. By 1995, 74% of white men and

women had been traced compared to 13 to 22% of other race groups. Analysis was thus restricted to white cohort members. The crude mortality rates for mesothelioma were 366 and 172 per million person-years for men and women, respectively.

The evidence from studies of South African cases of mesothelioma is consistent in showing that the dominant fibre type responsible is Cape crocidolite. The data for Transvaal crocidolite is sparse. The Transvaal crocidolite mines were smaller operations and were often excluded from studies, or pooled with amosite mines (Sluis-Cremer et al., 1992; Rees et al., 1999a; Rees et al., 1999b). Part of the legacy of South Africa's exploitation of its asbestos mineral reserves is the large number of cases of mesothelioma caused by environmental exposure. These environmentally exposed cases, in particular, appear to be the result of exposure to crocidolite.

Amosite

An important feature of the South African experience in respect of malignant mesothelioma is the prominence of environmental exposure. This has been identified since the very first published paper (Wagner, 1960) on the association of blue fibre and malignant mesothelioma. The section on amosite makes it clear at the outset that determining exposure to a specific fibre only in the Pietersburg field is difficult. This is clear from detailed mapping, from west to east, of the geological transition from a succession of crocidolite and amosite seams at Malips River, to the dominance of amosite at the Mohlapiitse River, to amosite only at Penge mine (Figures 7 and 8) and at Kromellenboog mine (Coetzee, 1976).

The vast majority of occupational medicine studies are carried out and published without environmental measurements. In this respect the Pietersburg field is unusual as there are reliable measurements of both occupational and environmental exposure. The two studies summarized in this chapter are unique in their attention to detail and the spread of the findings. This account seeks to add new data to the inconclusive situation in the Pietersburg field and the studies of amosite miners, as opposed to insulators and ladders.

The health effects of amosite mining and milling in South Africa have been reviewed in detail (Murray and Nelson, 2008). There is sound evidence of occupational and environmental exposure to airborne amosite fibre in the Pietersburg asbestos field for more than fifty years. In her thesis, Felix cites the early description of 'unchecked' exposures of children at work in the mill at Penge Mine. It was then one of the largest asbestos mines in the world and the source of most of the supply of amosite. Schepers, an officer of the Medical Bureau for Occupational Diseases in Johannesburg, visited Penge Mine in 1949 and commented thus: "Exposures were crude and unchecked. I found children, completely included within large shipping bags, trampling down fluffy amosite asbestos, which all day came cascading down over their heads" (Felix, 1997).

Labour was drawn, in the main, from the rural areas surrounding the mines (Davies et al., 2004). The Penge group of mines (Penge, Weltevrede and Kromellenboog) operated a recruiting depot in the Eastern Cape, in what was formerly the Transkei, which was used to recruit additional labour when labour shortages occurred locally.

Rendall, working at the National Institute for Occupational Health, carried out a detailed survey of Penge mine in 1970 (Rendall and Davies, 2007). He collected 267 full-shift personal samples from underground and surface workers at Penge mine in 1971-72. The average of 94 personal samples collected from individuals working underground was 1.34 fibres(f)/ml (range 0.28-3.26) The median value for the individuals grouped by task or work station and averaged (22 groups) was 1.10 f/ml. This low level and narrow range of exposure is attributed to the fact that the underground workings at Penge mine are kept constantly wet by water dripping from the hanging walls and running down the side walls, acting as a dust suppressant. The ore leaving the underground workings is saturated. In stark contrast the average of 177 personal samples collected from individuals working above ground in the offices, workshops, mills and packing departments, where the material being processed is dry, was 25 f/ml (range 0.28-326.7). The median value for the individuals grouped by task or work station and averaged (24 groups) was 37.2 f/ml. The highest fibre level was associated with hand tamping in the packing process – exactly the situation described by Schepers (see Felix 1997) except for the fact that there were no children jumping up and down in the bags. Rendall also measured the total dust levels and calculated the number of fibres per milligram of dust, showing that the total dust level did not correlate well with the fibre exposure. In the assay laboratory the total dust level was 2.68 mg/m³ containing 24.1 million fibres/mg whereas in quality control the dust level of 0.55 mg/m³ contained 21.3 million f/mg. and in the bagging plant 21 million fibres. This illustrates the importance of the relationship between the process and the fibre content of the dust.

Simultaneously Cape Asbestos, the owners of Penge, operated a smaller mine or set of mines known as Egnep at Malipsdrift towards the western extremity of the Pietersburg field 60 kilometres west of Penge Mine. Officially Penge and Egnep were considered as one mine, and the product from these mines, well to the west of the Mohlapiitse River, was transported to Penge for shipment via the railway siding at Apiesdoring near Burgersfort.

The association of the mining operation at Malipsdrift with that at Penge and the uncertainty as to where deposits of Transvaal crocidolite end and pure amosite begins implies a possibility, if not of admixture in the product, at least of mixed exposure of workers. Production of amosite from Penge and the smaller mines along the northern bank of the Olifants River peaked in 1970 at 100,000 tonnes. At that stage 7,000 men were employed at Penge. Cape Asbestos had major crocidolite mining operations in the Northern Cape, and senior employees were interchanged or made visits to Cape's other mines. This adds a further complication to the accurate determination of exclusive fibre type exposure. Some experienced foremen were also moved from the crocidolite to the amosite mines and vice versa.

Environmental exposure was investigated in detail as part of the study of the villages round Mafefe. The results are recorded here to make the point that one would expect a significant number of environment only cases from Mafefe and the many similar groups of villages situated in close proximity to asbestos mines in the Pietersburg field. In 1990, as part of the study cited previously, Felix investigated the "current sources of environmental asbestos exposure in Mafefe" where a number of small mines worked deposits of amosite and



Figure 7. Derelict ore bins at Penge mine. (Courtesy of Dr Koichi Honma).

crocidolite, via surface workings, adits or less often from underground workings (Figure 8). Their tailings dumps were close to one or more of the 30 settlements which fall under the jurisdiction of the traditional ruler (kgosi) in the Mafefe area. The population of Mafefe in November 1987 was 11,119. Tailings were frequently dumped on the banks of the Mohlapiitse River or the streams draining into it. Deposits high in the Strydpoort Mountains were worked from adits and the waste rock and tailings tipped down the hillside – to this day the resulting environmental contamination cannot be abated in this rugged terrain. This doctoral thesis is a rich source of historical and contemporary information, and includes detailed maps and lists of fibre levels measured by government inspectors over the years – it would be instructive to reproduce more of the detail in the thesis but this would make the account unwieldy. Thorough investigations of asbestos exposure in communities living around asbestos mines, such as this one, are rare.

Ninety-two personal samples were collected by adults and children going about their usual tasks. The mean of the 92 samples was 12 fibres per litre or 12,000 f/m³ (0.6 – 90 f/l; S.D. 13.3 f/l). Fourteen samples showed levels above 20 f/l. The highest mean concentration of fibres was 20.3 f/l among children playing (13 samples). School attendance exposed children to a mean of 13.2 f/l (9 samples), and teachers were exposed to 12.5 f/l (5 samples). Usual activities such as building and gardening entailed exposure to 16.1f/l (8 samples) and 15 f/l (7 samples) respectively. Walking about the village exposed subjects to 12.0 f/l (9 samples) and housework entailed exposure to 8.6 f/l (16 samples).

The tailings dumps on the bank of streams feeding the Mohlapiitse River have been mentioned. The seasonally dry bed of this river is used as a source of building sand. Two

personal samples collected by individuals loading river sand onto a trailer revealed exposures to 4 and 12 f/l, indicating significant contamination of the river bed. This river joins the Olifants River which flows into the Indian Ocean through Mozambique. Needless to say nothing is known about asbestos-related diseases among the riparian population living downstream of the asbestos mines in the Pietersburg asbestos field.

The mean fibre concentration of 62 strategic samples was 11 f/l or 11,000 f/m³ (range 0.1 – 51.2 f/l; S.D.11.7 f/l). Two results were excluded; one taken alongside a children's playground (756.5 f/l) and another (50.4 f/l) taken inside a house in which there was no visible asbestos in the construction. These levels were judged to be aberrant and no explanation could be found. The mean of 44 outdoor strategic samples was 14.5 f/l (s.d. 13.9), and that of 18 taken indoors was 2.7 f/l (s.d. 1.8). On days when vehicles used the road, the mean of 12 strategic samples taken at the roadside was 20.7 f/l (s.d. 14.8) compared with 13.1 f/l (s.d. 14.5) on the 10 vehicle free days.

Finally, the extent and variability of environmental asbestos exposure is clearly established by two series of strategic measurements made in 7 villages in close proximity (less than 1 kilometre) to tailings dumps and 11 villages far (more than 1.5 kilometre) from dumps. The mean fibre concentration in ambient air collected by strategic sampling in villages close to the dumps (21 samples) was 18.6 f/l, and in the rest (23 samples) 10.8f/l. The difference is statistically significant ($p < 0.00001$). The unofficial limit for environmental exposure was set at 20 f/l. It is reasonable to conclude that residents of Mafefe were exposed intermittently to levels higher than this, and regularly to lower levels.



Figure 8. Amosite fibres contaminating the ground near Penge mine. (Courtesy of Dr Koichi Honma).

Given this degree of occupational and environmental exposure of the labour force, and the communities from which they were drawn, one might be justified in assuming that a situation comparable to that described in the study of the crocidolite-exposed Prieska birth cohort might be found in the area. This is not the case.

Felix (Felix, 1997) analysed the available data from two studies of autopsy findings in cardio-respiratory organs submitted from Penge Mine and from mines in the Northern Cape (Sluis-Cremer, 1965, 1970). The figures presented show a higher prevalence of asbestosis at Penge and, in addition, a much smaller improvement over time at Penge. The numbers of autopsies analysed, the age and length of service of the two groups do not differ significantly. There is a significant body of evidence of widespread benign pleural and parenchymal asbestos related respiratory disease among occupationally and environmentally exposed individuals. There are sporadic, unconfirmed reports of mesotheliomas.

In a random sample of 892 adults from a census of Mafefe, where a number of small mines worked deposits of amosite and crocidolite, 681 were examined. Pleural changes which could be confidently classified as asbestos-related pleural disease were present in 35% of women and 52% of men, with a significant upward trend with age (Felix, 1997).

In terms of the Occupational Diseases in Mines and Works Act 1973, doctors in South Africa are required to report occupational lung disease in current or former mine workers to the Medical Bureau for Occupational Diseases (MBOD) in Johannesburg. Reports from doctors working in the hospitals and clinics in and around the Pietersburg asbestos field were very uncommon prior to 1991. When a clinic was established at Groothoek Hospital to service the need for compensation examinations at Mafefe, no reports had ever been received from that hospital by the MBOD. Sustained attempts were made from November 1991 onwards to interview and examine former asbestos miners for compensation purposes, with the assistance of local activists. In the period 1991-1994, reports for 927 former asbestos miners were submitted to the MBOD. During an intensive case finding project in 1996 more than 2000 former miners were examined and reported (Davies et al., 2001). A number of publications resulted and these show a high prevalence of benign asbestos-related diseases, but did not identify any substantial number of proven malignant mesotheliomas or lung cancers (Davies et al., 2001; Davies et al., 2004).

The first report of a malignant mesothelioma from the area of the Pietersburg asbestos field, which has overlapping seams of amosite and crocidolite fibres, is contained in a review of 485 females and 53 males admitted to the medical wards of the Jane Furse Memorial Mission Hospital situated about 60 kilometres south-west of Penge Mine (Edginton et al., 1972). At the time the hospital was estimated to be responsible for the medical care of 100,000 people. Forty-eight per cent (231) of the diagnoses in women were respiratory disease - tuberculosis 98, other respiratory infections 130. Non-infective respiratory disorders were found in only three women – one autopsy proven malignant pleural mesothelioma, and one possible but unproven lung cancer, and one case of asthma. Despite the proximity of asbestos mining, asbestos-related disease is not mentioned in the discussion. The high incidence of

respiratory disease and a single mesothelioma in a series of fewer than 500 female patients is surely remarkable, and worthy of comment. However, we have no information about these patients' occupation or exposure history.

Further unverified evidence of mesotheliomas from the Pietersburg field is included in the Felix thesis. Felix's personal communication in 1990 with Dr van Rensburg, a pathologist working at the Pietersburg laboratory of the South African Institute for Medical Research (SAIMR) reads: "In 15 months from February 1989 to April 1990, 16 mesotheliomas were diagnosed at the SAIMR Pietersburg laboratory. Of the 16 cases, 11 occurred in black persons, 4 of whom were women". No details of occupation or exposure are given.

In 1990 a 44 year old migrant worker who worked for 2 years from 1963 to 1965 on an asbestos mine in the Pietersburg asbestos field, was diagnosed as having a mesothelioma at the Rand Mutual Hospital, Johannesburg (Felix, 1997). No details of the mine on which he was employed are available.

Serious reservations must be clearly stated before an attempt is made to make any conclusive statement about the relationship between occupational and/or environmental amosite exposure and the occurrence of malignant mesothelial tumours. The first proviso is that related to the overlapping fibre types in the Pietersburg field. Additional reservations include the inaccessible area in which amosite was mined, the rudimentary medical services in the area surrounding the amosite mines and the apparent neglect by the mine itself, all of which hamper us in getting conclusive data about amosite exposure.

In the dust rooms of the National Centre for Occupational Health non-human primates were exposed to specific dusts, including amosite, and kept in the laboratory for many years in order to determine the long term effects of the dusts to which they had been exposed. The non-human primates used in the experiments were locally captured baboons (*Papio ursinus*). At the time, there was no evidence that South African baboons were natural hosts for SV40 (Malherbe, 1974). In one study, 12 baboons were exposed to amosite for a period of between 242 days and 898 days. Exposures were high, ranging between 1100 and 1200 fibres per cc. Ten survived the exposure period and lived for a further 1.2 to 10.2 years. Five of these 10 baboons developed mesothelioma: 3 were peritoneal and 2 were pleural tumours (Webster et al., 1993). The amosite used in these exposure studies was the milled sample prepared at the NIOH for the UICC standard reference sample. This amosite standard reference sample has been used in asbestos-exposure related research studies worldwide (Timbrell and Rendall, 1971; Rendall, 1980). It is possible that the milling of the fibre may alter its physical properties thereby increasing its toxicity, in contrast to the freshly mined fibres. This may provide another possible explanation for the apparent rarity of malignant mesotheliomas among miners exposed to freshly mined amosite fibre.

Rees debates the role of amosite in the causation of malignant mesothelioma in the course of a case-control study of 123 cases and 119 cancer controls and 103 medical controls. The conclusion is carefully stated and is quoted in full: "The relative importance of Cape crocidolite should not mask the impact of identifying three cases in 16 months from a single

amosite mine (Penge). This, together with the contention by Felix (Felix et al., 1994) that mesothelioma is under-diagnosed in the North Eastern Transvaal (now Limpopo and Mpumalanga Provinces), provides motivation for case-finding strategies in the area. Cross-sectional surveys are inappropriate for a rare disease with short life expectancy following diagnosis, so alternatives are necessary. One approach would be to allocate the task to the regional health authority which would be in a position to identify cases by encouraging pathologists to submit suspect tissue for expert review. Cases could be identified by establishing diagnostic and compensation services for asbestos-related diseases in the major regional hospital and by providing information about the condition and the service to the community and local medical practitioners" (Rees et al., 1999b).

Since this was written many thousands of former asbestos workers have been examined for compensation purposes in the Pietersburg asbestos field (Davies et al., 2001; Davies et al., 2004). The Maandagshoek Project set up a network of clinics in co-operation with a number of community-based activists following five years of preliminary work at Groothoek Hospital. Workers who had been certified by the MBOD were subsequently denied compensation by an administrative decision making written records of mining employment obligatory. In an interesting report South Africa's Public Protector declared this to be unconstitutional and stated that there were 12,000 outstanding claims which should be reviewed (Public-Protector, 2008). Among the thousands of applicants for benefits interviewed and examined as part of the Maandagshoek Project there were no proven mesotheliomas.

The study of asbestos fibre type and mesothelioma carried out on cases autopsied at the NIOH demonstrates a residual burden of exclusively amosite fibres in only one of 43 cases examined. In five cases amosite was the majority fibre type (more than 50%) accompanied in all cases by crocidolite in proportions ranging from 7-33%. In six cases amosite was the second most common fibre (4-22%), crocidolite being the dominant fibre in each of these. The remainder of the cases were attributable to crocidolite (Nolan R P, 2006).

Occupational and environmental exposures to asbestos in the Pietersburg field were high and high rates of benign asbestos-related disease have been described (Murray and Nelson, 2008). But mesothelioma is rarely reported from the hospitals and clinics in and around the Pietersburg asbestos field. The reporting of asbestos-related disease is extremely poor in South Africa, particularly in the areas that supplied labour to the mines (Talent et al., 1978). There is an underlying problem in attributing cases of mesothelioma to amosite because of the geological relationship between deposits of amosite and Transvaal crocidolite. The single case from the Jane Furse Hospital and the unconfirmed series of cases reported from the SAIMR laboratory in Pietersburg have no occupational histories or exposure data. The nearest we have to definitive studies are the doctoral thesis by Rees (Rees, 1995) and the residual fibre study by Nolan (Nolan, 2006). Both of these suggest that amosite plays a minor role and that crocidolite is much more important. By comparison with the Northern Cape, where crocidolite is mined, environmental mesotheliomas in proximity to amosite mines and mills are rare.

Chrysotile

The role of chrysotile in the causation of mesothelioma has been debated for decades (Churg, 1988; Huncharek, 1994; Smith and Wright, 1996; Stayner et al., 1996; Egilman et al., 2003; McDonald, 2010). One view is that the amphiboles (crocidolite, amosite and tremolite) explain almost all cases of mesothelioma and that “chrysotile mesothelioma” is induced by contaminating tremolite (McDonald, 2010). A contrasting opinion is that chrysotile is the main cause of mesothelioma (Smith and Wright, 1996). The South African experience of mesothelioma and chrysotile is of interest in this debate because the country mined, milled, transported and used large quantities of the fibre.

Chrysotile mining started in South Africa in about 1920 (Felix et al., 1994). From 1975 to 1992 production was close to 100 000 US tons per annum. From 1992 output declined dramatically to about 20 000 tons in 2000 (Rees et al., 2001). The mining of chrysotile asbestos ceased when the last mine at Msauli closed in 2002. Exports of chrysotile overtook that of amosite in the mid-1970s and crocidolite in the early 1980s (Harington and McGlashan, 1998). From 1980 to 2003, 1,568,928 metric tonnes of chrysotile had been exported, making up 54% of South Africa’s total asbestos export over the period (Harington et al., 2010). Substantial numbers of miners produced the mineral: from the 1930s to mid-1980s roughly 1000- 2000 miners were employed at any time with a peak of about 2 600 in 1960 - 17% of all asbestos miners in that year (Rees et al., 1999a).

In South Africa, despite the substantial output and large numbers exposed, mesothelioma attributable to exposure to chrysotile-only has not been convincingly documented. The first paper to comment on this issue was published by Webster in 1973 (Webster, 1973), who reviewed the exposure histories of 232 cases of pleural mesothelioma referred to South Africa’s Asbestos Tumour Reference Panel. Seventy eight of the cases had been exposed during mining operations, 75 of them (96%) on crocidolite mines and three (4%) on Penge amosite mine. Webster reported that there were no cases in which there had been exposure to chrysotile only, but noted that relatively small numbers of miners were employed in chrysotile mining. Five years later, the exposure histories of 70 additional cases were published (Cochrane and Webster, 1978). Fibre-specific exposures were not reported except for 13 cases with non-occupational environmental exposure associated with mining: all 13 of them had been exposed in the Northern Cape crocidolite fields. In 1984, Solomons published a review of 80 cases of mesothelioma referred to the occupational medicine clinic of the NIOH, during 1977 to 1983 (Solomons, 1984). Fibre type was thought to be mixed in most cases. Documented exposure in 17 cases was to Cape crocidolite only and in four cases to amosite only (Penge mine). Although not explicitly stated, it can be assumed that no chrysotile-only case was found in this study.

In 1986, Wagner supported the view that mesothelioma was rare or non-existent in South African chrysotile miners by stating that the malignancy had not been recorded among these workers (Wagner, 1986). Surprisingly little research was conducted subsequently on mesothelioma in relation to this fibre type, but in 1999 the exposure histories of 123 consecutive incident cases diagnosed or treated in six South African cities were published (Rees et al., 1999a; Rees et al., 1999b). The cases were interviewed in life and details of

domestic, environmental and occupational asbestos exposure were obtained. No case had a history of chrysotile mining or environmental contact exclusively with this fibre type. Two cases (1.6%) reported contact with chrysotile and little if any contact with amphiboles, but neither was conclusive of chrysotile as the cause of the mesothelioma. One had been exposed to the mineral only four years prior to diagnosis. The other had spent over 30 years in chrysotile mining districts but had spent three months on an asbestos mine in the Northern Province - an amphibole mining area. In a biopsy of his pleural tissue, amphibole asbestos fibres were isolated (Rees et al., 1999a).

The most recent data are from two trusts which were established in 2003 and 2006 following litigation by claimants seeking compensation for asbestos-related diseases. The trusts are respectively the Asbestos Relief Trust (ART) and the Kgalagadi Relief Trust (KRT)(KRT, 2011). The ART provides for those exposed at or near 30 asbestos mines and related operations including all of the major chrysotile mines. The KRT provides only for claimants with exposure arising from two crocidolite mines. To date 275 cases of mesothelioma have been adjudicated, none from a chrysotile area (Mothemela, 2011). Additional information comes from the personal experience of pathologists at the NIOH. The NIOH Pathology Division conducts autopsies on former miners to ascertain eligibility for workers' compensation. All deceased miners are entitled to an autopsy. According to the Head of Pathology, no case of a mesothelioma has been recorded in a miner with service only in chrysotile mining (Personal communication Murray J, NIOH 2011). This is despite fairly large numbers of former miners with mesothelioma coming to autopsy: 111 cases from 2004 to 2007 (Phillips and Murray, 2009).

There are a number of possible explanations for the absence or paucity of documented chrysotile mesothelioma cases in South Africa. First, this may be a consequence of very small numbers of people having had exposure to chrysotile fibre. This explanation is unconvincing given the fairly large numbers employed in the industry over many decades. Second, it may well be that these cases are rare and have been missed. Based on cohort studies of workers predominantly exposed to chrysotile, it has been estimated that overall about 0.3% of these workers died of mesothelioma, although the percentage of deaths varied across the exposure settings and was influenced by time passed since first exposure and ascertainment of cohort vital status and cause of death (Stayner et al., 1996). Even at peak employment in South African chrysotile mining - 2 600 people - there would have been 7.8 mesothelioma cases in this cohort at an estimated risk of 0.3%. Thus, despite over 80 years of chrysotile mining, the number of cases may be relatively small, but it seems unlikely that all of them would have been unrecorded. A third explanation is that exposure was so well controlled that cases would not arise. Again, this is unlikely. Slade, a medical officer at a chrysotile mine, observed in 1931 that: "Several years of experience at 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." (Felix et al., 1994).

It should be noted, though, that management of African Chrysotile Asbestos (ACA), by far the largest chrysotile mine, has stated that fibre levels were consistently below 1 fibre/ml in

the 1980s and 90s (Rees et al., 2001). These measurements were not independently verified and the statement may have been based on average fibre concentrations, rather than the exposure levels of the most exposed miners and millers (Rees et al., 2001). Given the low credibility of the former explanations for the paucity of mesotheliomas attributable to chrysotile-only exposure, a relatively low level of contamination of South African chrysotile by tremolite deserves consideration. Unfortunately, data on this issue are scanty: only two small studies have been published (Rees et al., 1992; Rees et al., 2001). Additionally, there has been one small unpublished dust survey done by the NIOH (du Toit, 1992). In the first study, tremolite fibres were sought in the lungs of four subjects with service exceeding 20 years on chrysotile mines. Scanning electron microscopy demonstrated tremolite in two of the four, but the fibres were scanty: one fibre seen in about 20 fields at 1000 times magnification. In the second study, asbestos fibre concentrations were determined in the lungs of nine South African chrysotile mine workers. Despite long service in most (range: unknown - 32 years; median, 9 years), asbestos fibre concentrations were generally low (geometric mean 690,000 and 330,000 fibres/gram dried lung tissue for chrysotile and tremolite respectively). The tremolite:chrysotile ratio was greater than one in only a single case. Both these findings are in contrast to those observed for Canadian chrysotile miners who have been shown to have much higher lung fibre burdens and a general preponderance of tremolite over chrysotile (Becklake and Case, 1994; Churg, 1994; Rees et al., 2001). The unpublished study (du Toit, 1992) examined 20 rock samples, two bulk dust samples from the mill and seven samples of airborne dust from ACA mine. Tremolite was found in only one rock sample; the dust samples were inconclusive (possible tremolite); and one tremolite fibre was identified in each of two of the seven air samples. Taken together, these three pieces of information, although far from conclusive, are suggestive of relatively low tremolite contamination and offer a plausible explanation for the lack of mesotheliomas in South Africa attributable to chrysotile exposure alone.

2. Non-occupational mesothelioma in South Africa

South Africa has a uniquely high national burden of environmental mesothelioma (i.e. cases with only non-occupational asbestos exposure). Environmental mesothelioma and the role of fibre type has been reviewed (White et al., 2008). Table 1 shows a consistently high proportion of these cases – 8.8% to 33% - from a variety of South African studies.

The lowest proportion was derived from a series of cases referred to an occupational medicine clinic (Solomons, 1984), and the intention to claim workers' compensation for an occupational disease probably led to a referral bias of subjects exposed at work. At that time, occupationally-induced cases were compensable but not those that were environmentally-induced. The highest proportion of environmentally induced mesotheliomas (33%) was from a study of cases registered with the South African Asbestos Tumour Reference Panel (Webster, 1973). Exposure histories were obtained from an interview with the patient (percentage not stated) or from a proforma completed by the medical practitioner submitting the biopsy specimen. Exposure histories were missing in 9% and no asbestos exposure was recorded for a further 14%. It is likely that thorough interrogation for

Reference	Source of mesothelioma subjects	Proportion environmental	Definition of environmental	Comment
(Webster, 1973)	232 cases registered with the South African Asbestos Tumour Reference Panel, 1955-1970	33%	No occupational exposure	Exposure histories usually taken by submitting doctor
(Cochrane and Webster, 1978)	70 cases referred to the National Research Institute for Occupational Disease, Johannesburg, by local practitioners	18.6%	Minimum 3 years residence in a mining area with no occupational exposure	Exposure history taken by authors from patients in-life
(Solomons, 1984)	80 cases referred to National Centre for Occupational Health clinic, Johannesburg, 1977 to 1983	8.8%	No occupational exposure	Exposure histories taken by clinic doctors from patients. Referral to the clinic probably influenced by intention to claim workers' compensation
(Zwi et al., 1989)	1347 cases identified by South African practitioners, 1976-1984	Males: 10% Females: 35% Total: 16%	Lived in the north-western Cape asbestos belt, or had specified domestic exposure with no occupational exposure	Exposure data supplied by reporting practitioner. No information available on 33% of cases
(Rees et al., 1999a)	123 cases diagnosed or treated in 6 South African cities, late 1988-early 1990	17.9%	Spent time in asbestos mining area without occupational exposure. Two cases also lived or worked in asbestos cement structures	Detailed exposure history taken in-life from cases
(Kielkowski et al., 2000)	28 cases from a birth cohort of white South Africans born in Prieska district (a crocidolite mining area) 1916-1936	Estimated 29%	All women cases (8/28) considered environmental	No exposure data but white women rarely worked in asbestos industries
(White et al., 2008)	Review of 504 cases	23% in total	Histologically proven cases	Exposure data for 87% of cases
(Mothemela, 2011)	295 compensation claimants adjudicated by ART and KRT*, 2003-2010	29.2%	Claimant must have resided within 10 kms of a qualifying operation (e.g. mine or mill) without occupational asbestos contact	Exposure histories obtained from claimants files. Trusts provided compensation for environmental and occupational exposure

*ART = Asbestos Relief Trust; KRT = Kgalagadi Relief Trust.

Table 1. Proportions of South African mesothelioma cases considered to be environmental

occupational exposure was inconsistent as workers' compensation for mesothelioma was only provided for miners from 1962 and for non-mining workers from 1979 (Solomons, 1984), whereas the cases were collected from 1955-1970. The Kielkowski study (Kielkowski et al., 2000) did not have exposure data, but assumed environmental exposure in all women subjects; if even a small proportion had had occupational exposure the 29% proportion would be reduced. The Mothemela data (Mothemela, 2011) are not generalisable to the country as a whole as these data are from claimants who either lived near or worked at mining operations. The other studies also have methodological limitations, e.g. 33% of the subjects in the Zwi study did not have exposure histories (Zwi et al., 1989). In a review of 504 cases from four of the above studies (White et al., 2008), the exposure was attributed to the environment in 23% in total. The 1973 Webster and 1984 Solomons studies are probably the least reliable in terms of proportions of environmentally-induced cases; if these two are ignored the remaining studies are fairly consistent: in South African mining regions the proportion of environmental cases is in the order of 29% (Kielkowski et al., 2000; Mothemela, 2011) and between 16% and 19% for the country as a whole (Zwi et al., 1989; Rees et al., 1999a; Cochrane and Webster, 1978).

This high environmental burden is in sharp contrast to other settings, except for the Wittenoom crocidolite mining region of northwest Australia; Da-yao, southwestern China, a region with naturally scattered patches of crocidolite ore; and central Anatolia in Turkey, where soil is contaminated with tremolite or tremolite-actinolite-chrysotile mixtures and less so with anthophyllite-chrysotile mixtures. During 1979-1994, 176 Wittenoom mesothelioma cases were documented of whom 34 (19.3%) had not been employed in mining, milling or transport of asbestos, but had lived in or visited the area (Rogers and Nevill, 1995). The 34 cases arose over 16 years; about two non-occupational cases per year. The population of three villages of Da-yao with about 20% of the total ground surface covered by crocidolite ore has been about 68 000 and all residents are assumed to have been exposed (Luo et al., 2003). Additionally, the fibre was used in family-style production to manufacture asbestos products such as stoves, until banned in 1984. It is estimated that only about 50 people were involved in these activities (Lamb and Reid, 1968). The average number of mesothelioma cases diagnosed at a local county hospital was 6.6 per year from 1984-95, an incidence rate of 97 per million per year; and 22 per million per year from 1996 to 1999. The average annual mortality rates for mesothelioma determined from two cohort studies in the region was 85 per million per year during 1977-83 and 178 per million per year during 1987-95. The latter rate is lower but in the same order as the rate of 277 per million person-years (95% CI 170-384) found for mesothelioma in the mortality study of the birth cohort in the South African crocidolite mining district of Prieska (Kielkowski et al., 2000). Age-standardised mesothelioma incidence has been reported for past residents of Wittenoom without occupational exposure to asbestos (Hansen et al., 1998). At 260 per million person-years it is very similar to the Prieska rate. In central Anatolia, which had no occupational asbestos exposure, the standardised average annual mesothelioma rates were 114.8 per 100 000 (1148 per million) for men and 159.8 per 100 000 (1598 per million) for women (Metintas et al., 2002). These standardised Anatolia rates are considerably higher than those of Witternoom, and the highest environmental rates reported.

In contrast, studies of non-occupational mesothelioma in chrysotile mining regions are scanty and cases are rare in the few studies that have been published. For example, only seven deaths from pleural mesothelioma were identified in women living in the chrysotile mining areas of Thetford Mines and Asbestos, Quebec, Canada, during 1970 to 1989 (Camus et al., 1998). The combined population of the two towns was about 45 000 in 1981. At least three of the seven cases may have had occupational exposure to amphiboles (Churg, 1998). Balangero chrysotile mine in Italy was the largest in Europe and mined asbestos from 1917 to 1990 (Silvestri et al., 2001). The mine was surrounded by four municipalities which had a combined population of about 11 550 in 1991. Eight mesothelioma deaths were recorded in these residents during 1970-1988. Exposure histories were not reported. A further three cases were identified between 1990-1995 from one of the municipalities (unspecified) producing incidence rates of 1.86 per million person-years for men and 6.82 per million person-years for women. However, two of the cases probably had had occupational asbestos exposure (Silvestri et al., 2001). Asbest City in the Sverdlovsk region of the Russian Federation, contains the largest chrysotile mining and milling complex in the world (Scherbakov et al., 2001). Mining started in 1886. The city has had asbestos mills since the early 20th century and also had facilities for the manufacture of asbestos-containing goods. In 1999, Tomilova (see Scherbakov et al, 2001) reviewed 41 cases of mesothelioma that occurred in the Sverdlovsk region from 1981 to 1996. In 27% no history of occupational asbestos exposure was identified and in a further 34% no exposure data were available (Scherbakov et al., 2001). Five of the cases were from Asbest, producing a standardised incidence rate of 2.8 per million persons per year.

South Africa, like other countries that have mined and milled asbestos, has documented cases of mesothelioma attributable to environmental exposure. In South Africa the vast majority of these environmental cases can be attributed to exposure to Cape crocidolite. Large areas of the country particularly in the Northern Cape Province have been and remain heavily contaminated. Other areas of the country are also heavily contaminated with asbestos; for example, a report on the town of Penge suggests that the area has been found unfit for human habitation due to the ongoing dangers of asbestos pollution (Meintjes and Hermanus, 2008).

3. Conclusion

South Africa was a significant producer of crocidolite, amosite and chrysotile asbestos. From 1910 to 2002, a total of 10,099,568 tonnes of asbestos were mined. Local sales generated ZAR 1.746 billion and export sales ZAR 28.981 billion. In particular it supplied the world with crocidolite and amosite. The association between mesothelioma and asbestos was first described in South Africa, in individuals exposed occupationally and environmentally to Cape crocidolite. Cape crocidolite remains the most potent fibre type for the development of mesothelioma in South Africa for both occupationally and environmentally exposed people. The South African experience and the local research findings show that the association between Northern Cape crocidolite exposure and the development of mesothelioma is unequivocal.

While there have been cases of mesothelioma attributable to exposure to amosite in South Africa, the situation is clouded because of the possibility of mixed exposures to amosite and Transvaal crocidolite, both of which were mined in the Pietersburg asbestos field. There are very few studies of Transvaal crocidolite. The South African experience of a paucity of mesotheliomas attributable to pure amosite exposure appears to differ from that of countries which imported the fibre and used it industrially (Roggli et al., 1993; Gibbs and Berry, 2008). There is no satisfactory explanation for this difference. It is possible that the milled fibre behaves differently from the freshly mined fibre. Factors which add further uncertainty are under-ascertainment of cases due to the remoteness of the areas where mining took place, the poor quality of medical services in the labour-sending areas and widespread failure to report occupational disease.

In South Africa there are very few cases of mesothelioma that can be attributed to chrysotile. This is despite the fact that the commercial mining of chrysotile continued after the mining of crocidolite and amosite ceased. A possible explanation for this is that the amount of contamination of South African chrysotile with tremolite asbestos is very low (Rees et al., 2001). The mesotheliomagenic potential of South African chrysotile is certainly much less than that of Cape crocidolite and amosite.

Despite knowing about the adverse health effects of asbestos since 1928 (Simson, 1928) and its association with mesothelioma since 1960 (Wagner et al., 1960), asbestos mines continued to operate in South Africa until 2002. The closure of mines was more about the global market for asbestos than concerns for the health of the mine workers. Because of South Africa's past economic and political situation the mining companies had a great deal of influence (McCulloch, 2002). Pressure from the industry sought to limit research into the adverse health effects of asbestos and delayed the publication of reports and scientific studies, such as the PRU report and the Botha paper (PRU, 1964; Botha et al., 1986).

Data on exposure levels at South African mines and mills are sparse. Poor record keeping, variable criteria for measurement and changes in the instruments used for measuring dust and fibre levels contributed to this situation (Sluis-Cremer et al., 1992). Where data is available the exposure levels for workers were shown to be extremely high (Rendall and Davies, 2007). As a consequence, levels of benign pleural and parenchymal asbestos-related disease are high (Davies et al., 2001; Davies et al., 2004).

When the mines closed they left a legacy of disease and a contaminated environment. Litigation, class actions and eventually an out of court settlement resulted in the founding of the ART and KRT funds (KRT, 2011). These funds offered restitution in the form of compensation money to individuals and their families who suffered because of asbestos-related diseases. In terms of this settlement, compensation became available for the first time for environmentally exposed individuals. A small amount of money was also made available for rehabilitation of the environment. The money for the funds came from a number of companies that mined asbestos. Access to these funds and compensation was limited to ex-employees of these companies and individuals who lived in the vicinity of these mines. Not all mines were part of this settlement. A significant number of mines, mills, tailings dumps and

surface workings are persistent sources of environmental contamination. Former workers at these operations are not covered by the trusts and cannot claim from them.

While the ART and KRT have done an excellent job in tracing, examining and compensating claimants, huge social and environmental problems persist. The contamination is not confined to the areas in the vicinity of mines. The transportation of asbestos contaminated other areas including railways, roads, marshalling yards, warehouses and docks (Braun and Kisting, 2006). Asbestos containing building materials pose an ongoing hazard for construction and demolition workers (Phillips et al., 2006; Phillips et al., 2007; Phillips et al., 2009). The disposal of asbestos containing material and the maintenance of asbestos dump sites is an ongoing problem. There is a shortage of housing and building materials in South Africa and the recycling of asbestos cement building materials, although banned in legislation, continues.

Despite being the world's largest producer of crocidolite and amosite, South African mesothelioma mortality rates for the period 1995 to 2007 are much lower than expected. In 1984, South Africa had one of the highest mesothelioma rates in the world. Unlike Australia where the rate of mesothelioma is still rising, the rate in South Africa appears to have peaked. The reasons for this are not entirely clear. It may be due to under reporting or competing causes of death related to the AIDS epidemic in the country (Kielkowski et al., 2011). In the 1950s, cases of mesothelioma were being mistaken for atypical pleural tuberculosis. Currently, tuberculosis is the most common AIDS defining illness in South Africa and co-infection is present in up to 80% of cases of tuberculosis. Given this background of a high mortality rate due to HIV/AIDS and tuberculosis, it is possible that cases of mesothelioma are being missed.

Although asbestos mining has ceased and the manufacture, import and export of asbestos containing products is banned, there is still a legacy of existing durable asbestos containing products in the environment (Braun and Kisting, 2006). Furthermore the asbestos deposits remain in the ground. Mining of minerals continues to be important to the economy of the country and asbestos deposits occur in association with other mineral deposits. In order to exploit the mineral wealth of South Africa, there is evidence of accidental or incidental mining of asbestos (Figure 9). In diamond mines the kimberlite ore body is drilled dry. Asbestos is known to occur in association with kimberlite pipes and the incidental mining of asbestos can occur. A risk of exposure to asbestos has recently been shown in a study of South African diamond miners. Tremolite-actinolite asbestos fibres have been identified in the lungs of miners and in the tailings from diamond mines. In this retrospective autopsy-based study of diamond miners, asbestosis, pleural plaques, a lung cancer and a case of mesothelioma were identified (Nelson et al., 2011).

Looking back on the South African experience with asbestos, it is clear that there were lessons that should have been learned sooner. The collection of data about exposure in the workplace and in the environment is vital. The analysis of these data leads to conclusions that should be incorporated into policy and implemented (Murray et al., 2011). On the whole, this has not been the case in the South African mining industry.

The question needs to be asked: on balance, was it worth mining asbestos? The commercial advantage never amounted to more than 3% of the total value of mineral-based revenues



Figure 9. Tremolite asbestos fibres from the tailings dump of South African platinum mine. (Courtesy of Professor JI Phillips).

generated by the mining industry. It is impossible to put a value on the pain and suffering due to asbestos-related disease. Money has been spent on health care and compensation, and more will need to be spent. Similarly, money has been spent on rehabilitation of the environment but much more needs to be spent on cleaning up vast tracks of land, including roads and railways as well as the remediation of old mining areas and the maintenance of asbestos waste dumps.

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Acknowledgement

The authors would like to acknowledge the many members of the staff of the NIOH, both past and present, who contributed to this work. The contribution of Prof JI Phillips is based on research supported by the National Research Foundation.

4. References

- Abratt RP, Vorobiof DA, White N. 2004. Asbestos and mesothelioma in South Africa. *Lung Cancer* 45 Suppl 1:S3-6.
- Becklake MR, Case BW. 1994. Fiber burden and asbestos-related lung disease: determinants of dose-response relationships. *Am J Respir Crit Care Med* 150:1488-1492.
- Botha JL, Irwig LM, Strelbel PM. 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.
- Braun L, Kisting S. 2006. Asbestos-related disease in South Africa: the social production of an invisible epidemic. *Am J Public Health* 96:1386-1396.

- Camus M, Siemiatycki J, Meek B. 1998. Nonoccupational exposure to chrysotile asbestos and the risk of lung cancer. *N Engl J Med* 338:1565-1571.
- Churg A. 1988. Chrysotile, tremolite, and malignant mesothelioma in man. *Chest* 93:621-628.
- Churg A. 1994. Deposition and clearance of chrysotile asbestos. *Ann Occup Hyg* 38:625-633, 424-625.
- Churg A. 1998. Nonoccupational exposure to chrysotile asbestos and the risk of lung cancer. *N Engl J Med* 339:999; author reply 1001-1002.
- Cochrane JC, Webster I. 1978. Mesothelioma in relation to asbestos fibre exposure. A review of 70 serial cases. *S Afr Med J* 54:279-281.
- Coetzee CB, Brabers AJM, Malherbe SJ, van Biljon WJ. 1976. Asbestos: Mineral Resources of the Republic of South Africa. Department of Mines, Geological Survey. Government Printer, Pretoria. ISBN 0 621 03464 9.
- Davies JC, Kielkowski D, Phillips JI, Govuzela M, Solomon A, Makofane MR, Sekgobela ML, Garton E. 2004. Asbestos in the sputum, crackles in the lungs, and radiologic changes in women exposed to asbestos. *Int J Occup Environ Health* 10:220-225.
- Davies JC, Williams BG, Debeila MA, Davies DA. 2001. Asbestos related lung disease among women in the Northern Province of South Africa. *S Afr J Sci* 97:87-92.
- Davies JCA, Landau SP, Goldsmith C, Langton ME. 1987. Mesothelioma risk among gold miners in South Africa. *South African Journal of Science* 83:184-185.
- du Toit RS. 1992. Dust survey. Internal report. Rdt 4.13.16.
- Edginton ME, Hodgkinson J, Seftel HC. 1972. Disease patterns in a South African rural Bantu population, including a commentary on comparisons with the pattern in urbanized Johannesburg Bantu. *S Afr Med J* 46:968-976.
- Egilman D, Fehnel C, Bohme SR. 2003. Exposing the "myth" of ABC, "anything but chrysotile": a critique of the Canadian asbestos mining industry and McGill University chrysotile studies. *Am J Ind Med* 44:540-557.
- Felix MA. 1997. Environmental asbestos and respiratory disease in South Africa. Ph.D Thesis, Faculty of Medicine. University of the Witwatersrand, Johannesburg. p 281.
- Felix MA, Leger J, Ehrlich RI. 1994. Three minerals, three epidemics - asbestos mining and disease in South Africa. *Advances in Modern Environmental Toxicology* 21.
- Gibbs GW, Berry G. 2008. Mesothelioma and asbestos. *Regul Toxicol Pharmacol* 52:S223-231.
- Hansen J, de Klerk NH, Musk AW, Hobbs MS. 1998. Environmental exposure to crocidolite and mesothelioma: exposure-response relationships. *Am J Respir Crit Care Med* 157:69-75.
- Harington JS, McGlashan ND. 1998. South African asbestos: production, exports, and destinations, 1959-1993. *Am J Ind Med* 33:321-326.
- Harington JS, McGlashan ND, Chelkowska EZ. 2010. South Africa's export trade in asbestos: demise of an industry. *Am J Ind Med* 53:524-534.
- Hart HP. 1988. Asbestos in South Africa. *J. S. Afr. Inst. Min. Metall.* 88:185-198.
- Hodgson JT, McElvenny DM, Darnton AJ, Price MJ, Peto J. 2005. The expected burden of mesothelioma mortality in Great Britain from 2002 to 2050. *Br J Cancer* 92:587-593.
- Huncharek M. 1994. Asbestos and cancer: epidemiological and public health controversies. *Cancer Invest* 12:214-222.
- Kielkowski D, Nelson G, Bello B, Kgalamono S, Phillips JI. 2011. Trends in mesothelioma mortality rates in South Africa: 1995-2007. *Occup Environ Med* 68:547-549.

- Kielkowski D, Nelson G, Rees D. 2000. Risk of mesothelioma from exposure to crocidolite asbestos: a 1995 update of a South African mortality study. *Occup Environ Med* 57:563-567.
- KRT 2011. Annual Report. Asbestos Relief Trust. URL <http://www.asbestostrust.co.za/> (accessed 21/6/2012).
- Lamb D, Reid L. 1968. Mitotic rates, goblet cell increase and histochemical changes in mucus in rat bronchial epithelium during exposure to sulphur dioxide. *J Pathol Bacteriol* 96:97-111.
- Langer AM, Nolan RP. 1994. Chrysotile biopersistence in the lungs of persons in the general population and exposed workers. *Environ Health Perspect* 102 Suppl 5:235-239.
- Langer AM, Selikoff IJ, Sastre A. 1971. Chrysotile asbestos in the lungs of persons in New York City. *Arch Environ Health* 22:348-361.
- Luo S, Liu X, Mu S, Tsai SP, Wen CP. 2003. Asbestos related diseases from environmental exposure to crocidolite in Da-yao, China. I. Review of exposure and epidemiological data. *Occup Environ Med* 60:35-41; discussion 41-32.
- Malherbe HH. 1974. The viruses of vervet monkeys and of baboons in South Africa. M.D. Thesis, Faculty of Medicine. University of the Witwatersrand, Johannesburg. p 65.
- Manfredi JJ, Dong J, Liu WJ, Resnick-Silverman L, Qiao R, Chahinian P, Saric M, Gibbs AR, Phillips JI, Murray J, Axten CW, Nolan RP, Aaronson SA. 2005. Evidence against a role for SV40 in human mesothelioma. *Cancer Res* 65:2602-2609.
- Martiny O. 1956. Primary mesothelioma of the pleura. *Proc. Transvaal Mine Medical Officers' Assoc.* 35:63-64.
- McCulloch J. 2002. *Asbestos Blues*. Oxford and Indianapolis: James Currey and Indiana University Press.
- McCulloch J. 2003. Asbestos mining in Southern Africa, 1893-2002. *Int J Occup Environ Health* 9:230-235.
- McDonald JC. 2010. Epidemiology of malignant mesothelioma--an outline. *Ann Occup Hyg* 54:851-857.
- Meintjes S, Hermanus M. 2008. "Unfit for human habitation: The Penge report". In: Phiroshaw C, editor. *National Asbestos Conference Report*, ISBN: 978-0-620-46691-2 ed. Johannesburg: KRT and ART. p 55-59.
- Metintas S, Metintas M, Ucgun I, Oner U. 2002. Malignant mesothelioma due to environmental exposure to asbestos: follow-up of a Turkish cohort living in a rural area. *Chest* 122:2224-2229.
- Mothemela M. 2011. Mesothelioma in South Africa three decades post peak of asbestos production: an analysis of claims database of asbestos ex-miners. In: African regional association of occupational health congress: Care for the occupational health needs of the worker. Birchwood Hotel, Boksburg, South Africa. URL <http://www.asbestostrust.co.za/documents/Abstracts-2011.pdf> (accessed 21/6/2012)
- Murray J, Davies JCA, Rees D. 2011. Occupational lung disease in the South African mining industry: Research and policy implementation. *Journal of Public Health Policy* 32:S65-S79.
- Murray J, Nelson G. 2008. Health effects of amosite mining and milling in South Africa. *Regul Toxicol Pharmacol* 52:S75-81.
- Nelson G, Murray J, Phillips JI. 2011. The risk of asbestos exposure in South African diamond mine workers. *Ann Occup Hyg* 55:569-577.

- Nishikawa K, Takahashi K, Karjalainen A, Wen CP, Furuya S, Hoshuyama T, Todoroki M, Kiyomoto Y, Wilson D, Higashi T, Ohtaki M, Pan G, Wagner G. 2008. Recent mortality from pleural mesothelioma, historical patterns of asbestos use, and adoption of bans: a global assessment. *Environ Health Perspect* 116:1675-1680.
- Nolan RP, Ross M, Nord GL, Raskina M, Phillips JL, Murray J, Gibbs GW. 2006. Asbestos Fibre-type and Mesothelioma Risk in the Republic of South Africa. *Clay Science* 12:223 - 227.
- Phillips JL, Murray J. 2009. South African data on malignant mesothelioma. *Ind Health* 47:198-199.
- Phillips JL, Norman G, Renton K. 2009. Asbestos in soil around dwellings in Soweto. *Occupational Health Southern Africa* 15:24-27.
- Phillips JL, Renton K, Badenhorst C. 2006. Potential health hazard from cleaning asbestos cement roofs: A case report. *Occup Health Southern Africa* 12:20-22.
- Phillips JL, Renton K, Murray J, Garton E, Tylee BE, Rees D. 2007. Asbestos in and around Soweto dwellings with asbestos roofs. *Occupational Health Southern Africa* 13:3 - 7.
- PRU. 1964. Field survey in the North Western Cape and at Penge in the Transvaal. In. Johannesburg: Pneumoconiosis Research Unit. p 64.
- Public-Protector SA. 2008. Report on an investigation into allegations of an improper insistence on the provision of documentary proof of service relating to claims for benefits and an improper refusal to accept affidavits by the Compensation Commissioner for Occupational Diseases. In. p 29.
- Rees D, du Toit RSJ, Rendall REG, van Sittert GCH, Rama DBK. 1992. Tremolite in South African chrysotile. *S Afr J Sci* 88:468-469.
- Rees D, Goodman K, Fourie E, Chapman R, Blignaut C, Bachmann MO, Myers J. 1999a. Asbestos exposure and mesothelioma in South Africa. *S Afr Med J* 89:627-634.
- Rees D, Myers JE, Goodman K, Fourie E, Blignaut C, Chapman R, Bachmann MO. 1999b. Case-control study of mesothelioma in South Africa. *Am J Ind Med* 35:213-222.
- Rees D, Phillips JL, Garton E, Pooley FD. 2001. Asbestos lung fibre concentrations in South African chrysotile mine workers. *Ann Occup Hyg* 45:473-477.
- Rees DJ. 1995. A case-control study of mesothelioma in South Africa. Ph.D. Thesis, Faculty of Medicine. University of Cape Town. p 192.
- Reid G, Kielkowski D, Steyn SD, Botha K. 1990. Mortality of an asbestos-exposed birth cohort. A pilot study. *S Afr Med J* 78:584-586.
- Rendall RE. 1980. Physical and chemical characteristics of UICC reference samples. *IARC Sci Publ*: 30:87-96
- Rendall REG, Davies JCA. 2007. Dust and fibre levels at Penge amosite mine 1970 to 1971. *Adler Mus Bull* 33:26-30.
- Rogers A, Nevill M. 1995. Occupational and environmental mesotheliomas due to crocidolite mining activities in Wittenoom, Western Australia. *Scand J Work Environ Health* 21:259-264.
- Roggli VL, Pratt PC, Brody AR. 1993. Asbestos fiber type in malignant mesothelioma: an analytical scanning electron microscopic study of 94 cases. *Am J Ind Med* 23:605-614.
- Scherbakov SV, Kashansky S, Domnin SG, Koslov V, Kochelayev VA, Nolan RP. 2001. The health effects of mining and milling chrysotile: The Russian experience.: Mineralogical Association of Canada.

- Silvestri S, Magnani C, Calisti R, Bruno C. 2001. The experience of the Balangero chrysotile mine in Italy: Health effects among workers mining and milling asbestos and the health experience of persons living nearby.: Mineralogical Association of Canada.
- Simson FW. 1928. Pulmonary asbestosis in South Africa. *BMJ* 1:885-887.
- Slade GF. 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. In. Johannesburg: University of the Witwatersrand. p 150.
- Sleggs CA, Marchand P, Wagner C. 1961. Diffuse pleural mesothelioma in South Africa. *South African Medical journal* 35:28 - 34.
- Sluis-Cremer GK. 1965. Asbestosis in South Africa--certain geographical and environmental considerations. *Ann N Y Acad Sci* 132:215-234.
- Sluis-Cremer GK. 1970. Asbestosis in South African asbestos miners. *Environ Res* 3:310-319.
- Sluis-Cremer GK, Liddell FD, Logan WP, Bezuidenhout BN. 1992. The mortality of amphibole miners in South Africa, 1946-80. *Br J Ind Med* 49:566-575.
- Smith AH, Wright CC. 1996. Chrysotile asbestos is the main cause of pleural mesothelioma. *Am J Ind Med* 30:252-266.
- Solomons K. 1984. Malignant mesothelioma--clinical and epidemiological features. A report of 80 cases. *S Afr Med J* 66:407-412.
- Stayner LT, Dankovic DA, Lemen RA. 1996. Occupational exposure to chrysotile asbestos and cancer risk: a review of the amphibole hypothesis. *Am J Public Health* 86:179-186.
- Talent JM, Harrison WO, Webster I. 1978. The prevalence of asbestosis, pleural changes and malignant neoplasia in black ex-miners and female cobblers. In: Annual Report. Johannesburg: National Research Institute for Occupational Diseases. p 36 -37.
- Timbrell V, Rendall RE. 1971. Preparation of the UICC Standard Reference Samples of asbestos. *Powder Technology* 5:279-287.
- Virta RL. 2006. Worldwide asbestos supply and consumption trends from 1900 through 2003: US Geological Survey circular 1298, 80p. In.
- Wagner JC. 1962. The pathology of asbestosis in South Africa. M.D. Thesis, School of Pathology, University of the Witwatersrand, Johannesburg. p 150.
- Wagner JC. 1986. Mesothelioma and mineral fibers. *Cancer* 57:1905-1911.
- Wagner JC. 1991. The discovery of the association between blue asbestos and mesotheliomas and the aftermath. *Br J Ind Med* 48:399-403.
- Wagner JC, Sleggs CA, Marchand P. 1960. Diffuse pleural mesothelioma and asbestos exposure in the North Western Cape Province. *Br J Ind Med* 17:260-271.
- Webster I. 1973. Asbestos and malignancy. *S Afr Med J* 47:165-171.
- Webster I, Goldstein B, Coetzee FS, van Sittert GC. 1993. Malignant mesothelioma induced in baboons by inhalation of amosite asbestos. *Am J Ind Med* 24:659-666.
- White N, Nelson G, Murray J. 2008. South African experience with asbestos related environmental mesothelioma: is asbestos fiber type important? *Regul Toxicol Pharmacol* 52:S92-96.
- Zwi AB, Reid G, Landau SP, Kielkowski D, Sitas P, Becklake MR. 1989. Mesothelioma in South Africa, 1976-84: incidence and case characteristics. *Int J Epidemiol* 18:320-329.