4 ASBESTOS HEALTH HAZARDS

4.1 INTRODUCTION

The dangers associated with the inhalation of asbestos fibres have been known, although not necessarily very widely, for more than a hundred years[27]. The disease-causing capabilities of asbestos were unknown until the first case of asbestosis was recorded in England around 1900 and described by Dr Murray in 1907.[28] Since then, literally thousands of reports have been published. Figure 3 illustrates the pattern of disease awareness as it developed over a century and a half.

FIGURE 3: THE RELATIONSHIP BETWEEN ASBESTOS PRODUCTION AND ASBESTOS-RELATED DISEASES

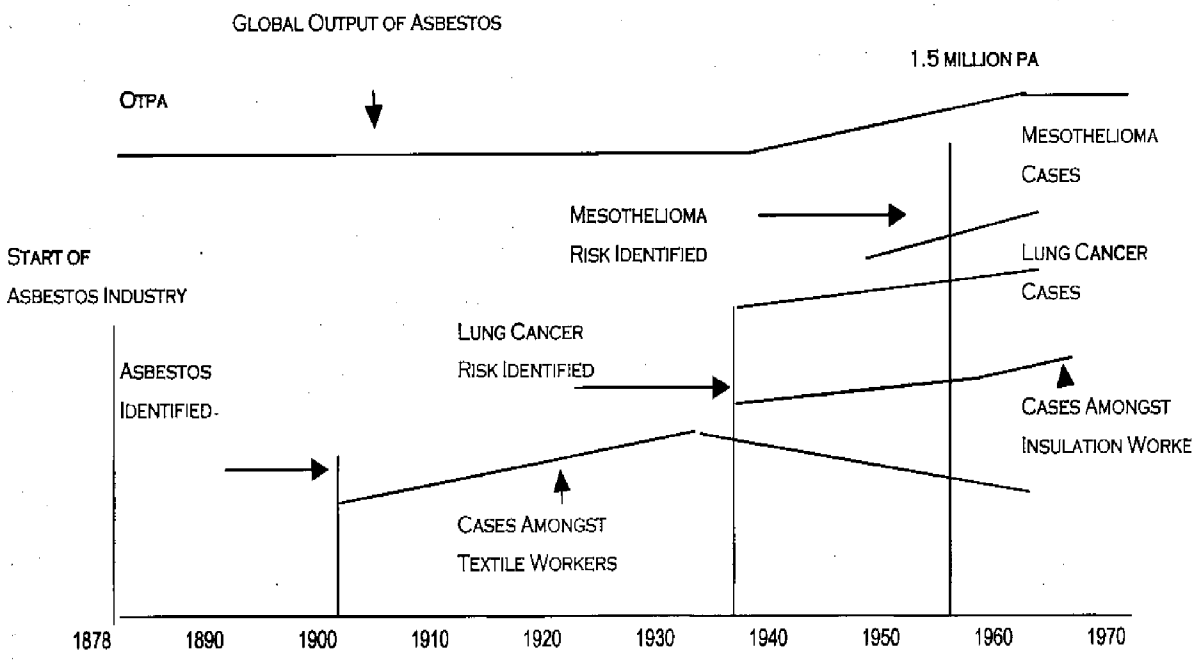

FIGURE 3: THE RELATIONSHIP BETWEEN ASBESTOS PRODUCTION AND ASBESTOS-RELATED DISEASES.
(SEE REFERENCE IN TEXT BELOW)

The relationship between asbestos exposure and disease was firmly established by 1930. In 1935, the link between asbestos exposure and lung cancer had been demonstrated and by 1955, it had been linked to several other types of cancers as well[29]. Asbestos has been declared a proven human carcinogen by the United States Environmental Protection Agency (EPA) and by the International Agency for Research on Cancer of the World Health Organization.

Clinical studies have established incontrovertibly that asbestos causes cancer of the lung, malignant mesothelioma of the pleura and peritoneum, cancer of the larynx, certain gastrointestinal cancers, as well as asbestosis, a progressive fibrotic disease of the lungs. The risk of contracting these diseases increases with increased exposure[30 31].

An important feature of most asbestos-related diseases is the latency period: it may take 20 to 40 years for the first symptoms to manifest. As such, many of those affected today were exposed to large quantities of fibres (sometimes more than 100 f/ml) up until the 1970s.

These levels, mostly a result of workers installing extremely dusty, sprayed-on (friable) asbestos insulation materials, were 100 to 200 times higher than current statutory exposure limits in most countries. In addition, more hazardous amphibole varieties of asbestos were frequently used at the time. Workers in the shipbuilding and construction industries who installed friable asbestos insulation materials have been the most severely affected[32].

Despite standards and regulations that have been in existence since the 1970s, the number of asbestos-related deaths is still rising in some countries. This may suggest that the control measures introduced were ineffective.

4.2 POSSIBILITIES OF EXPOSURE

Before discussing the health risks of exposure to asbestos, one should first distinguish between the various possibilities of exposure. As stated in the introduction, most asbestos related diseases have a long latency period, often more than 30 years. Persons that were not occupationally exposed in the years immediately before diagnosis may too easily be classified under "neighbourhood exposure", although occupational exposure may have taken place 20 to 40 years previously[31].

It is also necessary to stress that it is not the presence of asbestos containing materials as such but the presence of asbestos fibres that are fine enough to be inhaled or ingested that constitutes a risk.

The following categories of exposure may be distinguished[33].

4.2.1 Direct Occupational Exposure

Examples include work in asbestos mines, asbestos textile weaving, manufacture of asbestos cement and asbestos insulation work. In addition to these classical exposures, there are a number of jobs in which asbestos containing materials have to be handled, i.e. workers in the building industry, cutters and layers of water pipes and blowing out of vehicle brake linings. Of the occupations exposed to asbestos, those most at risk are workers who are unknowingly exposed to high concentrations of respirable asbestos in buildings: plumbers, gas fitters, carpenters and electricians, whose job has involved drilling and sawing asbestos.

4.2.2 Indirect Occupational Exposure

In these situations, workers do not handle asbestos themselves, but are exposed as a result of the activities of others at nearby workplaces.

4.2.3 Para-Occupational Exposure

This category includes leisure time activities, sometimes serving as a second job. "Handyman", or Do-it-Yourself projects may involve cutting and working with asbestos containing products. During Do-it-Yourself projects, control measures such as those enforced in industry are more than often neglected, leading to potentially high exposures for the operator and others.

4.2.4 Para-Occupational Domestic Exposure

Women married to asbestos workers, or other members of the household may be exposed as a result of handling asbestos contaminated clothing. There is insufficient data to accurately estimate the population at risk, but there is some evidence of an increased prevalence of asbestos related disease in relatives of asbestos workers.

Today, most industries enforce strict personal hygiene measures, including workplace showers and laundries.

4.2.5 Neighborhood Exposure

This category includes people living in the vicinity of asbestos mines, asbestos processing factories and asbestos dumps.

4.2.6 True Environmental Exposure

True public health exposure includes the presence of asbestos in ambient air, food, beverages, water, as well as the wear and tear of asbestos products (i.e. building products, friction materials, etc.) In recent years, public concern has shifted from the workplace to exposure in the general environment. The environmental risks of asbestos have been studied by a number of major scientific bodies. For example, the WHO Conference on Mineral Fibres in the Non-Occupational Environment in September 1987 concluded that, for the general population, the risks of mesothelioma and lung cancer attributable to asbestos are probably undetectable low, and the risk of asbestosis is virtually zero.

A meeting on the Environmental Reduction of Asbestos convened by the WHO in 1988 calculated that high-density products (such as asbestos cement building products and friction materials) do not present an unacceptable risk to the general population, although care is needed to contain airborne dust during installation and repair. The meeting also recommended that the use of friable asbestos insulation materials should be discouraged. Diseases have developed where exposure has been only very brief, or where the quantity of airborne asbestos was very small.

4.3 ASBESTOS RELATED CONDITIONS AND DISEASES

In order to cause harm, asbestos fibres must be breathed into the lungs. Respirable asbestos fibres are defined as being less than 3 microns in diameter and more than 5 microns in length, with an aspect ratio of 3:1. Such a fibre would require 500 times magnification to be seen with the human eye.

Researchers have identified a number of conditions and diseases that are associated with the inhalation of the various types of asbestos fibre. A review of the literature is provided below.

4.3.1 Asbestos bodies in the Sputum and Lung

Asbestos bodies are elongated structures between 20 μm and 200 μm[34] and usually have bulbous ends. An asbestos body consists of an iron-mucoprotein complex surrounding, or coating the asbestos fibres after inhalation and retention in the lung. However, it has been demonstrated that any inorganic fibre of the appropriate size[35] may form the core.

It seems as if the formation of asbestos bodies is a defence mechanism of the lungs and the mere presence thereof in the sputum or lung is not indicative of disease. Coated fibres are at the most indicative of exposure to asbestos.

4.3.2 Pleural Plaques and Diffuse Pleural Fibrosis

Pleural plaques are circumscribed areas composed of very firm, white hyaline material that may be focally calcified. These plaques may be extensive, are leaf shaped, often bilateral and have an irregular shape[35].

In some cases there is generalised pleural fibrosis, leaving the lungs completely sheathed in a thick layer of fibrous tissue. Unlike plaques, generalised pleural thickening can restrict the expansion of the lungs, causing breathlessness[35].

Pleural plaques are frequently found in asbestosis cases and in persons casually exposed to asbestos dust. Low exposures from work-related, domestic and natural sources may induce pleural plaques. The duration since first exposure is an important determinant in the chance of getting the disease. For diffuse pleural thickening, high exposure levels may be required[36].

Pleural plaques are generally not found in less than10 years, and seldom in less than 20 years after first exposure to asbestos. Pleural calcification rarely occurs less than 20years after first exposure to asbestos. Most cases of pleural effusion occur later in persons with prolonged exposure, but in a small number it has been seen after an interval of less than 10 years[36].

4.3.3 Asbestosis

Asbestosis is a form of pneumoconiosis, specially associated with exposure to asbestos dust. The disease is characterised by a massive interstitial fibrosis starting at the base of the lungs and progressing slowly upwards[34 35 37]. During the development of the disease, the lung tissue is gradually changed into a diffuse collagenous fibrotic mass. The active lung tissue is thereby replaced by inactive scar tissue, resulting in dyspnoea[34].

Asbestosis is usually associated with the presence of large numbers of asbestos bodies and fibres in the tissue. Pleural plaques may be present[36]. A dose-effect relationship has been establishment for asbestosis. The degree of the disease will therefore depend on the dose, i.e.

Dose = Concentration x Duration of exposure.

Asbestosis is generally associated with relatively high exposure levels and there is no evidence that casual exposure, such as occurs in the general population, causes asbestosis. According to Rees[5], asbestosis presents after a minimum of 10 to 20 years of exposure, with a latent period between first exposure and disease discovery a minimum of 15 years, and often considerably longer.

As a general guideline, asbestosis will not progress to clinical manifestation at or below a lifetime occupational exposure of 25 fibre/ml years, i.e. concentrations of 5 fibres/ml for 5 years, or 1 fibre/ml for 25 years, etc[36]. In this respect, it is important to refer to the section of this report dealing with South African regulations, as the Asbestos Regulations introduced in 1987 in RSA specified an Occupational Exposure Limit of 1 fibre/ml (assuming 24-hour exposure during a working lifetime of 25 years) and was primarily aimed at preventing asbestosis.

Asbestosis does not occur as a result of the occasional cutting of asbestos but rather in workmen regularly exposed to the dust. The disease, however, has been demonstratedin wives that came into continuous contact with the clothes of workmen and in persons living in the vicinity of asbestos mines, factories and dumps[36].

Asbestosis may be well established before it is diagnosed by radiological or physiological examination and cigarette smoking often obscures the symptoms[36]. Sufficient exposure may cause the disease to progress after the worker has left the industry.

4.3.4 Cancer of the Lung

In 1955 Doll concluded that asbestos workers faced a lung cancer risk ten times as great as that in the general population. It must be added that the group of affected workers had had 20 years or more exposure before the implementation of the Asbestos

Regulations in England in 1931.

A more recent study indicated that the risk of developing lung cancer varied between 1,5 to 5,6 times or higher than the normal risk[29]. Most studies suggest a dose–response relationship with lung cancers more common in those groups most heavily exposed.

The Helsinki Criteria offers practical guidelines as to whether a lung cancer should be attributed to asbestos exposure. According to these criteria, a minimum lag time of 10 years from first exposure to asbestos is required to attribute lung cancer to asbestos.

The risk of contracting a lung cancer seems to run parallel with the asbestosis risk, i.e. asbestosis sometimes leads to cancer of the lung[29 34 35].

There is a definite relationship between cancer of the lung and cigarette smoking in asbestos workers. The risk of a cigarette-smoking worker heavily exposed to asbestos developing lung cancer is 25 to 50 times greater than an age-matched non-smoker who has never worked with asbestos[35]. The reason for this increased risk associated with smoking seems to be that the two carcinogenic factors act in a multiplicative fashion rather than an additive fashion.

4.3.5 Mesothelioma

Mesothelioma is a rare diffuse cancer that spreads over the surface of the lung (pleura) and the abdominal organs (peritoneum). Wagner et al first suggested an association between this disease and asbestos exposure in 1960, when he found an abundance of cases in the vicinity of asbestos mines in the Northern Cape Province[38]. Today this association is clear, although discussions continue on whether some types of asbestos fibre and level of exposure have greater potential in causing the disease than others.

Patients with mesothelioma often have a history of only transient exposure to asbestos, amounting to a few weeks only, many years before diagnosis[34]. Mesothelioma may be present in the absence of any radiological or pathological evidence of fibrosis. Some cases show amounts of asbestos bodies in the lung that are the same as that observed in city dwellers[34]. This raises the question of whether minor degrees of asbestos pollution are capable of giving rise to mesothelioma.

According to Rees, city dwellers are not at risk from the general background pollution produced by widespread asbestos use[36].

Lag periods of between 25 years and 50 years with an average of 33 years between first exposure and the onset of symptoms have been recorded[34]. Rees gives a latency period of 30 to 40 years as the norm.

4.3.6 Cancer of the Gastro-Intestinal Tract

Several studies have reported a significant increase in cancer of the gastro-intestinal (GI) tract as a result of asbestos exposure[34 35]. A GI cancer risk of 1 in 10 workers exposed to asbestos is mentioned.

Many everyday commodities, such as water, beer and other drinks, may present a health hazard as a result of asbestos contamination[34].

4.3.7 Asbestos Warts

These wart-like formations in the skin develop around asbestos fibres that have penetrated into the skin. They are found in exposed parts of the skin of asbestos workers. These warts are harmless[34].

4.4 THE INCIDENCE OF ASBESTOS RELATED DISEASES

Studies regarding the incidence of asbestos related diseases (ARD's) in South Africa and elsewhere in the world have been conducted since the mid 1920's. The studies have proven the causal effect of asbestos in causing certain diseases such as asbestosis, cancer of the lung and mesothelioma.

The earliest South African reference to asbestos related disease was a report of the histological findings in the lung of a chrysotile miner who died in 1926[39].

A systematic epidemiological study of South African chrysotile miners by Slade between 1926 and 1930 revealed that: "with continued exposure to high concentration of asbestos dust, workers may show symptoms and physical signs of pulmonary fibrosis within a year"[40].

Autopsy findings of 64 Pietersburg asbestos field miners, who died in active service between July 1959 and June 1964, revealed the presence of asbestosis in 51 out of 64 autopsies (80%)[41].

Mesothelioma is considered to be a rare tumour. An association between this disease and asbestos exposure was suggested by Wagner et al in 1960, who found an abundance of cases in the vicinity of asbestos mines in the Cape Province (now referred to as Northern Cape)[42].

Mesothelioma's incidence has been reported as between 1 and 2,2 per million-per-year in Canada and the USA respectively[43]. Research conducted by McDonald & McDonald revealed that mortality rates for mesothelioma seemed to be the highest in insulation workers[44]:

- Ranging from 5,2% to 8,8% in insulation workers
- 0,8 to 6% in asbestos factory workers
- 0% to 0,19% in anthophyllite or chrysotile miners and millers; and
- 0,03% to 0,06% in the general population

These figures are, however, representative of the situation in countries such as Canada and the USA and are, therefore, not entirely applicable to South Africa.

The South African incidence of mesothelioma has been found to be more than 3 times higher than in the USA. The explanation given is that asbestos is not mined or milled in any quantity in the USA, whereas these activities are central to the South African asbestos industry[45]. The Canadian incidence rate was also found to be much lower than in South Africa, presumably because only chrysotile is mined and milled in Canada. South Africa mined crocidolite and amosite, which are considered to be more carcinogenic in man than chrysotile[46].

Solomons stated that the reporting of mesothelioma cases in South Africa is unreliable[45]. Considering the dose-response relationship, it was further stated that it is reasonable to assume that employees with shorter durations of exposure have a lower relative risk of contracting mesothelioma, compared to those with longer exposures[47]. Most South African asbestos miners have been exposed for relatively longer periods and would therefore have a significantly higher risk (an exposure of greater than 2-3 fibre-years/ml) for the development of asbestos disease. Solomons stated that, in spite of the relatively large numbers of miners who have mesothelioma, significant numbers of cases are not recognised or diagnosed. From the mean figure of 96,3 new cases of mesothelioma per year at the time of the study, Solomons derived a mesothelioma incidence rate of 7,2 per million of population (Age-adjusted to US population (1980) for annual best-available South African mesothelioma incidence rate).

A further study conducted by Solomons reviewed the clinical and epidemiological features of 80 cases of malignant mesothelioma (as proven by examination of biopsy specimens) referred to the clinic at the NCOH between January 1977 and June 1983. This study revealed that there was a positive history of asbestos exposure in 71 out of the 80 subjects (89%). Similar studies in the past have found between 18% and 97% positive asbestos related exposure histories[46]. Exposures included Cape crocidolite and amosite asbestos, predominantly in mixed combinations. In 17 out of the 80 subjects (21%), the exposure was solely to Cape crocidolite asbestos. A mean exposure period of 13,6 years, and a mean lag period of time from first exposure to diagnosis of 34,4 years, was reported[45].

Felix, in a study of the Mafefe community, drew attention to the potential numbers of previously undetected cases of occupational lung disease among the men and women living around old asbestos mines[48]. The study also drew specific attention to the exposure of women to asbestos. In a random sample of adults living in Mafefe in 1998, Felix found that 126/247 (51%) men and 137/432 (32%) women had worked in mines. 60% of the men and 44% of the women were found to have radiological evidence of asbestos related pleural disease.

The above-mentioned findings led to the first survey of occupational lung disease in women. A study by Davies et al of a sample of 770 women, who had worked in asbestos mines in the Northern Province between 1929 and 1992, determined the prevalence of asbestos related disease[49]. A clinical diagnosis of pleural and/or parenchymal asbestosis was made in 741/770 (96,2%) of the women. The reasons for the high prevalence of asbestosis may be attributed to the high levels of amphibole dust exposure and the long residence time of the dust in the lungs. This study therefore confirmed that there is a significant unrecognised burden of asbestos related diseases among woman in South Africa who have worked on asbestos mines.

A broad study was conducted by Rees et al covering the major centres in South Africa, to describe the exposure experiences of South African mesothelioma cases, with emphasis on the contribution made to the caseload by different fibres during exposure[50]. A sample of 123 subjects, diagnosed with mesothelioma, was interviewed in various centres in South Africa.

The results revealed a convincing history of asbestos exposure in 118/123 (96%) of the subjects:

- Strong evidence of exposure to crocidolite asbestos was reported in 68/123 (55,3%) of the mesothelioma cases
- None of the 123 mesothelioma subjects had previously worked on chrysotile asbestos mines
- Environmental exposure accounted for 22/123 (18%) of the mesothelioma cases, of which 20/22 (91%) had contact with Cape crocidolite asbestos.
- There was a relative scarcity of cases linked to amosite and no convincing chrysotile asbestos case.

From the results of this study, and taking note of the high percentage of asbestos exposure histories in the mesothelioma subjects, it can be deducted that non-asbestos cases of mesothelioma occur rarely in South Africa. The study further establishes that there is fibre gradient of mesotheliomagenic potential for South African asbestos: crocidolite > amosite > chrysotile.

Sluis-Cremer et al suggested that, according to South African data, amosite is less dangerous than Cape Crocidolite, at least as far as mesothelioma is concerned[51]. These researchers found the incidence of mesothelioma for crocidolite exposed workers to be 446 per million person-years, and for amosite exposed workers to be 78 per million person-years. A significant difference can be detected for the mesothelioma incidence between workers exposed to crocidolite and amosite fibres respectively

A birth cohort mortality study was carried out by Kielkowsky et al to establish the risk of developing mesothelioma in a group born between 1916 and 1936, in Prieska, Northern Province. Kieslowsky referred to several studies that have shown an increased risk of developing mesothelioma or respiratory cancer after occupational or environmental exposure to crocidolite asbestos.

Botha et al reported an annual death rate due to asbestosis or mesothelioma for white people of 542 per million for a high exposure crocidolite area (which included Prieska), 87 for a low exposure crocidolite area, and 24 in a control area[52]. By comparison the rates for coloured people were 573 per million for high exposure areas and 215 per million for low exposure areas, respectively. Sluis-Cremer et al showed a mesothelioma rate of 446 per million person-years for white men employed in crocidolite mining[41]. By comparison, a recently published study conducted in Australia estimated the incidence of mesothelioma due to environmental asbestos exposure in Witenoom as 270 per million person-years[53]. From the above it may be deducted that there are consistently observed, increased rates of asbestos related diseases, such as mesothelioma, especially in areas where crocidolite asbestos was mined.

The study conducted by Kielkowsky et al in the Prieska area revealed high figures for mesothelioma mortality, and for lung cancer mortality[54]. The mortality rates for men and women were 366 and 172 per million person-years respectively. The mortality for lung cancer was 287 per million person-years. The study found that all cancer mortality ratios were increased. The reasons given for the higher mortality rates in men, was that men were more likely to be exposed to both occupational and environmental exposure. The mortality rate for woman was still considered to be high and may be attributed to the high level of environmental exposure in the Prieska area. Kielkowsky further stated that the cause-specific mortality rates could be expected to increase over time due to the long latency periods from the first exposure to diagnosis of the neoplasms.

4.5 OCCUPATIONAL HEALTH RISKS

There are no valid, reliable incidence statistics of asbestos related diseases (ARD's) for South African mining and industrial environments. There are a number of sources from which it is possible to obtain individual case information. However, no one has as yet attempted to produce statistics that would indicate the percentage of people that are exposed to asbestos and who then subsequently develop an ARD.

This makes projecting the occupational health benefits of phasing out of asbestos in South Africa extremely difficult. Unlike the economic costs and benefits (i.e. disinvestments/investment, employment, cost/price changes, etc.), it is impossible to project with any degree of reliability the likely reduction in diseases and deaths, nor the medical and compensation cost savings that would be made from a complete phasing out of asbestos in South Africa.

4.6 COMPENSATION STATISTICS

One of the few organisations that do have data on ARD's is the Workers Compensation Fund. This organization processes claims for compensation from South African workers who have develop occupational diseases. As such, the Fund has case information for workers that have submitted claims for ARD's.

Table 8 reflects a breakdown of the 277 ARD cases processed by the Workers Compensation Fund for the 1994 to 1997 period. The table reflects the numbers of claims submitted and paid out by industry sector, ranked by number of claims submitted.

TABLE 8: WORKERS COMPENSATION FUND ARD CASES

	NUMBER OF CASES		NUMBER OF PAYOUTS	TOTAL PAY OUT	AVERAGE PAY OUT
Underground Mining	92	33.2%	84	R 13,946,323	R 166,027.65
Transnet Operations	34	12.3%	29	R 4,054,316	R 139,804.00
Asbestos Product Manufacturing	27	9.7%	26	R 3,082,740	R 118,566.92
Metal Tube Manufacturing	17	6.1%	15	R 2,483,750	R 165,583.33
Electrical Power Generation	11	4.0%	11	R 2,071,200	R 188,290.91
Foundry Manufacturing	10	3.6%	10	R 776,494	R 77,649.40
Cement/Coal Merchanting	9	3.2%	9	R 1,393,685	R 154,853.89
Chemical Manufacturing	8	2.9%	8	R 1,370,600	R 171,325.00
Building & Construction - General	7	2.5%	7	R 502,823	R 71,831.86
Professional Services	5	1.8%	5	R 828,480	R 165,696.00
Textile Manufacturing	5	1.8%	5	R 341,552	R 68,310.40
Auto Maintenance	4	1.4%	4	R 267,609	R 66,902.25
Brick Manufacturing	4	1.4%	4	R 1,448,193	R 362,048.25
Concrete Manufacturing	4	1.4%	4	R 834,573	R 208,643.25
Iron and Steel Manufacturing	4	1.4%	4	R 181,483	R 45,370.75
Glass Manufacturing	3	1.1%	3	R 380,967	R 126,989.00
Oil Distribution	3	1.1%	3	R 81,896	R 27,298.67
Steel Erection	3	1.1%	3	R 277,428	R 92,476.00
Banking, Finance and Insurance	2	0.7%	2	R 223,015	R 111,507.50
Bonded Warehousing	2	0.7%	2	R 29,788	R 14,894.00
Farming	2	0.7%	2	R 301,656	R 150,828.00
Food Retailing	2	0.7%	2	R 421,636	R 210,818.00
Building & Construction - Installation	1	0.4%	1	R 20,783	R 20,783.00
Building Merchanting	1	0.4%	1	R 15,032	R 15,032.00
Candle Manufacturing	1	0.4%	1	R 17,637	R 17,637.00
Cement Manufacturing	1	0.4%	1	R 105,542	R 105,542.00
City Council Administration	1	0.4%	1	R 17,345	R 17,345.00
Educational Services	1	0.4%	1	R 349,398	R 349,398.00
Electronics Manufacturing	1	0.4%	1	R 11,776	R 11,776.00
Fruit Canning	1	0.4%	1	R 19,015	R 19,015.00
Gas Manufacturing	1	0.4%	0	R 0	#DIV/0!
Grain Milling	1	0.4%	1	R 242,485	R 242,485.00
Granite Manufacturing	1	0.4%	1	R 54,864	R 54,864.00
Hospitals	1	0.4%	1	R 67,379	R 67,379.00
Hotels	1	0.4%	1	R 28,181	R 28,181.00
Panel Beating	1	0.4%	1	R 498,916	R 498,916.00
Printing	1	0.4%	1	R 25,700	R 25,700.00
Shipping	1	0.4%	1	R 31,707	R 31,707.00
Tobacco Manufacturing	1	0.4%	1	R 465,052	R 465,052.00

	NUMBER OF CASES		NUMBER OF PAYOUTS	TOTAL PAY OUT	AVERAGE PAY OUT
Transport	1	0.4%	1	R 18,030	R 18,030.00
Vulcanising	1	0.4%	1	R 906	R 906.00
TOTALS	**277**		**260**	**R 37,289,955**	**R 143,422.90**

Although these statistics do not reflect the incidence of ARD's in these sectors, a comparison of the absolute number of claims submitted and paid per sector may give some indication of the relative prevalence of ARD's across industries.

Mining, with 33%, has the highest number of claims. Relative to mining all of the other industries have small numbers of claims. This seems to suggest that the incidence of ARD's in these industries may be quite low. However, without proper epidemiology and incidence statistics, this may in fact be an invalid assumption.

4.7 SUMMARY AND CONCLUSION

The relationship between asbestos exposure and diseases such as asbestosis, cancer and mesothelioma has been demonstrated in numerous studies conducted over the greater part of the last century. There are a number of possibilities where workers or members of the general public may be exposed to asbestos, ranging from occupational to environmental exposure.

With regard to the ability of the various forms of asbestos fibres to cause disease, it was proven that a fibre gradient could be observed. Rees et al concluded that there is a fibre gradient of mesotheliomagenic potential for South African asbestos. This fibre gradient can be summarised as follows: crocidolite > amosite > chrysotile. Studies by Sluis-Cremer et al support this view.

Various researchers have expressed the opinion that current estimates of asbestosis and mesothelioma, and other asbestos related lung diseases, are an underestimate of the true situation. It was stated by Rees that comprehensive information on occupational lung disease is not available for South Africa and, what is available underestimates disease burdens markedly[55].

Despite the fact that there are no valid reliable epidemiological studies of the incidence of asbestos related diseases in South Africa, there is a significant body of case related evidence that suggests that ARD's are a significant public health issue.

More research, surveillance and case-finding activities are needed and the data that is gathered by state agencies needs to be put to better use. Compensation and enforcement agencies need to appreciate the value of the data they create, and thus publish it more quickly and comprehensively, and make data sets available to researchers as a matter of course[55].

Useful data is contained in compensation statistics (i.e. Workmens Compensation Commission, the Medical Bureau for Occupational Disease – MBOD), surveillance programmes (i.e. Surveillance of Work-Related and Occupational Respiratory Diseases in South Africa – SORDSA, the Pathology Division Report) and national registers (i.e. the National Cancer Register). However, because of differences in recording, great care should be exercised when making comparisons and extrapolations from the available information[55].