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LIVING IN THE SHADOW OF A DUST CLOUD

Occupational respiratory diseases in the South African mining industry, 1975 to 2009

Gillian Nelson

2012

A thesis submitted to the Faculty of Health Sciences,
University of the Witwatersrand,
in fulfilment of the requirements for the degree of
Doctor of Philosophy

Johannesburg, 2012

DECLARATION

I, Gillian Nelson, declare that this thesis is my own work. It is being submitted for the degree of Doctor of Philosophy in the University of the Witwatersrand, Johannesburg. It has not been submitted before for any degree or examination at this or any other university.



10th day of February 2012

DEDICATION

In memory of my parents

Gordon Kenneth Nelson
1929 – 1996

and

Joyce Marion Nelson
1931 - 1993

And to my children

Megan and Edward

PUBLICATIONS AND PRESENTATIONS ARISING FROM THE THESIS

Original papers

1. **Nelson G**, Girdler-Brown B, Ndlovu N, Murray J. Three Decades of Silicosis: Disease Trends at Autopsy in South African Gold Miners. *Environ Health Perspect* 2010; 118(3):421-6.
2. Rees D, Murray J, **Nelson G**, Sonnenberg P. Oscillating Migration and the Epidemics of Silicosis, Tuberculosis, and HIV Infection in South African Gold Miners. *Am J Ind Med* 2010; 53:398-404.
3. **Nelson G**, Murray J, Phillips J. Asbestos-related diseases in diamond mine workers The risk of asbestos exposure in South African diamond mine workers. Ann Occup Hyg 2011; 55(6):569–77.
4. **Nelson G**, Murray J. Silicosis at autopsy in platinum mine workers (provisionally accepted for publication by Occ Med, Jan 2012).

The publishers have given permission for reprinting of the three published papers.

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8. Murray J, Candy G, **Nelson G**, Ndlovu N. Pathology Division Surveillance Report: Demographic data and disease rates for January-December 2004. NIOH Report 7/2005. National Institute for Occupational Health, National Health Laboratory Service: Johannesburg, 2005. http://www.nioh.ac.za/assets/files/PATHAUT_Report_2004.pdf (accessed 21 Jul 2011).
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2. **Nelson G**, Murray J, Ross MH. Silicosis in South African platinum miners – an autopsy survey. 10th International Conference on Occupational Respiratory Diseases (10th ICORD), Beijing, China. 19 - 22 April 2005.
3. **Nelson G**, Ndlovu N, Candy G, Murray J. Thirty year silicosis trends at autopsy in South African gold miners. 3rd National Public Health Conference, Midrand, South Africa. 16 - 17 May 2006.
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7. **Nelson G**. Silicosis in South African miners: an autopsy study. Showcasing Postgraduate Research at Wits, Johannesburg, South Africa. 7 - 8 November 2008 (awarded joint-2nd oral presentation prize).

8. **Nelson G**, Ndlovu N, Murray J, Girdler-Brown B. Silicosis at autopsy in South African gold miners: 1975 to 2006. International Conference on Occupational Health (ICOH), Cape Town, South Africa. 23 – 27 March 2009.
9. **Nelson G**, Murray J, Girdler-Brown B, Ndlovu N. Silicosis trends in South African gold miners: 1975 – 2007. European Respiratory Society Annual Congress, Vienna, Austria. 12 – 16 September 2009 (e-presentation by J Murray).
10. **Nelson G**. Three decades of silicosis in the SA gold mining industry. Mine Medical Professionals' Association 12th Annual Congress, Magaliesburg, South Africa. 2 – 4 October 2009.
11. **Nelson G**. Diamond Mining: the inclusion of asbestos. University of the Witwatersrand Postgraduate Symposium, University of Witwatersrand, Johannesburg, South Africa. 20 – 21 October 2009.
12. **Nelson G**, Girdler-Brown B, Murray J, Ndlovu Z. Three decades of silicosis: disease trends at autopsy in South African gold miners. EPICOH-MEDICHEM Conference, Taipei, Taiwan. 20 - 25 April 2010 (poster presentation).
13. **Nelson G**. Accidental mining of asbestos: current concerns. Mine Medical Professionals' Association 13th Annual Congress, Rustenburg, South Africa. 20 – 22 May 2011.
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2. Case study: Investigating silicosis and tuberculosis in DBCM miners. De Beers Report to Society 2007, pg 66. http://www.debeersgroup.com/ImageVault/Images/id_1044/ImageVaultHandler.aspx (accessed 21 Jul 2011).
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4. Murray J, Sonnenberg P, **Nelson G**, Bester A, Shearer S, Glynn JR. Cause of death and presence of respiratory disease at autopsy in an HIV-1 seroconversion cohort of Southern African gold miners. AIDS 2007; 21(Suppl 6):S97-S104.

Asbestos

5. White N, **Nelson G**, Murray J. South African experience with asbestos related environmental mesothelioma: Is asbestos fiber type important? Regul Toxicol Pharmacol 2008; 52(1):S92-S96.
6. Murray J, **Nelson G**. Health effects of amosite mining and milling in South Africa. Regul Toxicol Pharmacol 2008; 52(1):S75-S81.

Occupational disease in mine workers

7. Murray J, **Nelson G**. Demographic data and disease rates in deceased South African miners. Occup Health Southern Africa 2004; 10(1):16.

ABSTRACT

Background

Silicosis rates in gold miners in South Africa are very high but there have been no analyses of long term trends. While much research has been conducted on occupational respiratory disease in gold, asbestos and coal miners, little is known about the respiratory health of miners of other commodities, such as diamonds and platinum, two of the most important minerals in South Africa. The ore bodies from which minerals are mined often contain other 'incidental' minerals and compounds that may cause disease.

Aims

The aims of this thesis were to conduct the first ever analysis of silicosis trends in black and white gold miners over a 33-year period; to discuss the role of oscillating migration in the high rates of silicosis; and to explore the potential for workers in the diamond and platinum mining sectors to develop occupational respiratory diseases.

Methods

Gold, diamond and platinum mine workers were identified from the PATHAUT autopsy database at the National Institute for Occupational Health. Trends in silicosis from 1975 to 2007 were calculated separately for black and white gold miners because of differences in exposure, patterns of employment and autopsy referral patterns. The role of oscillating migration in the silicosis epidemic was explored. Diamond mine workers with asbestos-related diseases at autopsy and platinum mine workers with silicosis and/or fibrotic nodules in the lymph nodes were identified. Supplementary data from other sources were reviewed to

exclude all those who might have been exposed to asbestos or silica, respectively, outside of the mining sector in which they worked. Asbestos lung burdens were calculated for the case series of diamond miners and mine tailings and soil samples were examined for asbestos fibres, using scanning electron microscopy.

Findings

The proportion of white miners with silicosis increased by 17% (from 18% to 22%) over the 33-year study period. That of black miners increased 10-fold (from 3% to 32%), primarily due to the aging workforce and increasing periods of employment. Adjusted odds ratios for silicosis increased with year of autopsy for black miners. Oscillating migration has also played a major role in the silicosis epidemic. Evidence indicates that diamond mine workers are at risk for developing asbestos-related diseases and that platinum mine workers are at risk for developing silicosis.

Conclusion

The gold mines have failed to control silica dust levels adequately and prevent disease in mine workers. The sparsity of available dust measurements and poorly documented work histories are major obstacles to conducting occupational health research in South Africa; attention and legislation needs to be focused urgently on these areas. The PATHAUT database is the only occupational respiratory disease database in South Africa that can be used for disease surveillance, trend analyses and research in all mining sectors.

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DEFINITIONS

Asbestosis a pneumoconiosis caused by exposure to asbestos fibres

Accidental mining the inadvertent mining of a mineral or compound that occurs during the process of extracting the mineral or compound of primary interest from the ore or country rock

Asbestos-related disease asbestosis, asbestos pleural plaques and mesothelioma (for the purposes of this thesis)

Asbestos plaque a deposit of fibrous tissue that develops in the chest cavity (usually on the parietal pleura) as a result of asbestos exposure

Coal workers' pneumoconiosis lung fibrosis caused by exposure to coal dust

Commodity a product that can be mined, processed and sold

Emphysema lung disease caused by the abnormal and permanent enlargement of the distal air spaces due to destruction of the alveolar walls

Department of Mineral Resources (DMR) the government agency responsible for the promotion and regulation of the South African minerals and mining sector (previously called the Department of Minerals and Energy)

Diamond miner a mine worker or ex-mine worker who was employed in no other mining sector than the diamond mining sector

Gold miner a mine worker or ex-mine worker who was employed in the gold mining industry, regardless of whether or not he was employed in any other industry or mining sector

Medical Bureau for Occupational Diseases (MBOD) the government agency that administers the Occupational Diseases in Mines and Works Act which provides compensation for occupational lung diseases in miners and ex-miners

Mesothelioma a malignant tumour of the pleural cavity of the lungs, associated with exposure to asbestos

Mine Health and Safety Council (MHSC) a national public entity, established in terms of the Mine Health and Safety Act, comprising a tripartite board represented by State, Employer, and Labour members under the chairmanship of the Chief Inspector of Mines; its main task is to advise the Minister of Mineral Resources on occupational health and safety legislation and research outcomes focused on improving and promoting occupational health and safety in South African mines

Mine Workers' Compensation (MWC) System database the electronic summary of work histories consolidated from the NIOH PATHAUT autopsy database, the MBOD files and other sources

Mine worker a person who has worked in a controlled mine or works, as defined by the DMR

National Institute for Occupational Health (NIOH) South Africa's major centre for occupational health development, training, service support and research

Naturally occurring asbestos asbestos deposits that have not been commercially mined

Oscillating migration a form of migration in which workers leave their rural partners to work in urban areas and return home periodically

Occupational Diseases in Mines and Works (ODMW) Act the Act that regulates compensation for occupational lung diseases in miners and ex-miners

PATHAUT database the electronic database comprising information on demographics, work histories and pathological findings at autopsy, for mine workers and ex-mine workers coming to autopsy at the NIOH

PATHAUT booklet the standardised, structured, numerically-coded document in which the macro- and microscopic pathology findings are recorded by the examining pathologist

Platinum miner a mine worker or ex-mine worker who was employed in no other mining sector other than the platinum mining sector

Pneumoconiosis a fibrotic lung disease caused by the inhalation of mineral particles

Pulmonary tuberculosis tuberculosis of the lung (see Tuberculosis)

Silicosis a pneumoconiosis caused by inhalation of crystalline silica dust, characterised by nodules of fibrosis, predominantly in the upper lobes of the lungs

Silicosis severity the degree of silicosis as determined by the number of nodules counted in the lung on macroscopic examination

Surveillance the ongoing and systematic collection, analysis and interpretation of data related to adverse health outcomes

Tuberculosis an infection caused by *Mycobacterium tuberculosis*

ABBREVIATIONS AND ACRONYMS

ARD	Asbestos-related disease
CCOD	Compensation Commissioner for Occupational Diseases
COPD	Chronic obstructive pulmonary disease
DBC	De Beers Consolidated mining company
DMR	Department of Mineral Resources
GPES	Global Program for the Elimination of Silicosis
HEG	homogeneous exposure group
HIV	Human immunodeficiency virus
ILO	International Labour Organisation
MBOD	Medical Bureau for Occupational Diseases
MHSC	Mine Health and Safety Council
MWC	Mine Workers' Compensation
NIOH	National Institute for Occupational Health
NOA	Naturally Occurring Asbestos
ODMWA	Occupational Disease in Mines and Works Act
PATHAUT	Pathology Automation System
PGM	Platinum Group Metals
PTB	Pulmonary Tuberculosis
SAMOSA	Surveillance of Upper Limb Musculoskeletal Disorders in South Africa
SEM	Scanning Electron Microscopy
SORDSA	Surveillance of Occupational Respiratory Disease in South Africa
TB	Tuberculosis
TEBA	The Employment Bureau of Africa
WHO	World Health Organisation

PREFACE

The thesis presented in the following pages covers two themes, viz. trends in silicosis in gold miners, and respiratory diseases in diamond and platinum mine workers caused by exposure to minerals that are incidental to those being mined, viz. asbestos and silica.

Research over the last two decades has shown that silicosis rates are high in both employed and ex- gold miners, especially black miners. However, these studies have been mostly cross-sectional and have used chest radiographs to diagnose disease. An analysis of trends in silicosis at autopsy in both black and white miners, published as part of this thesis, clearly demonstrates the failure of the gold mines to control dust and the long lasting effects of the migrant labour system on disease rates (comprehensively discussed in the first published paper of this thesis).

There have been no published data on respiratory diseases in mine workers with exclusive employment in the diamond or platinum mining sectors until now. There is mineralogical and geographical evidence to indicate that these mine workers are at risk of developing occupational respiratory disease. Two of the thesis papers describe the evidence for the risk of asbestos-related diseases in diamond mine workers and silicosis in platinum mine workers, respectively.

The primary data source used for the studies of disease in the three different mining sectors was the autopsy (PATHAUT) database at the National Institute for Occupational Health. The database is described in detail in the body of the thesis, together with its strengths and limitations.

The difficulties experienced in assessing individual dust exposures in the absence of valid dust measurements and incomplete work history records are discussed. The lack of these data makes it very difficult to identify and/or validate sources of dust exposure that may explain disease.

The four scientific papers that encompass the themes of the thesis can be found in appendices 1 to 4. Each includes a comprehensive description of the methodologies employed and the findings. The introduction and discussion of each paper have been expanded in the following pages, with an emphasis on obstacles to occupational respiratory health research in South Africa, recommendations emanating from this thesis, and suggestions for further research.

SECTION 1

BACKGROUND

Following a brief **INTRODUCTION**, this section provides a background to the two themes of the thesis in discussions on **SILICOSIS IN THE SOUTH AFRICAN MINING INDUSTRY** (both from an historical and a contemporary stance) and **THE POTENTIAL FOR RESPIRATORY DISEASE IN UNDER-RESEARCHED MINING SECTORS IN SOUTH AFRICA**, using the platinum and diamond sectors as examples. The section ends with the **AIMS AND OBJECTIVES** of the thesis.

1.1 INTRODUCTION

South Africa's economy was built on its mineral wealth which was dominated by gold for over a century. A variety of other minerals and commodities have been, and still are, mined in the country, with changing patterns of production over the decades. As well as revenue for the country, the occurrence and subsequent mining of minerals have provided direct and indirect employment for millions of people over the last century and more.

In 1985, the South African mining industry employed more than 800 000 workers (Table 1). By 2010, this number was less than 500 000, primarily due to the decrease in gold production. Platinum production now exceeds that of gold but both mining sectors employed more than 100 000 workers in 2010. In that year, platinum and gold mine workers comprised 65% of the mining workforce; most of the remaining workers were employed in the coal and diamond mining sectors.

Table 1. Change in employment patterns in South African major mining sectors from 1985 to 2010 [DMR, 2003; DMR, 2011a]

Commodity	Year		Difference	% change
	1985[1]	2010[2]		
	n	n	n	
PGMs*	73 880	168 591	94 711	128
Gold	527 947	143 750	-384 197	-73
Coal	120 959	71 588	-49 371	-41
Iron Ore	6 827	18 579	11 752	172
Chrome	7 347	13 719	6 372	87
Diamonds	18 352	11 159	-7 193	-39
Manganese	2 890	5 690	2 800	97
Copper	9 489	3 309	-6 180	-65
Asbestos	7 576	0	-7 576	-100
All mining	806 876	481 509	-325 367	-40

* Platinum Group Metals

The thesis is structured around two themes which are addressed in detail in four papers (Appendix 1 – 4). This section presents the themes, provides a background for them, including a discussion of the migrant labour system and some of its consequences, and contextualizes the issues that are discussed in the four thesis papers. The methods, which are comprehensively described in the four papers, are summarised in Section 2, and the major findings from each paper are described in Section 3. The discussion (Section 4) addresses the obstacles to occupational respiratory health research in South Africa, makes recommendations emanating from this thesis, and suggests directions for further research.

Thesis themes

The first theme addresses **trends in silicosis in the South African gold mining industry**. The results of this analysis, which covers a period of 33 years from 1975 to 2007, have been published, together with a discussion about factors contributing to high silicosis rates, such as migrancy, changing employment practices, and inadequate dust control. A second paper more comprehensively addresses the

issue of oscillating migration and the consequent effects on silicosis, tuberculosis and HIV.

The second theme explores the **potential for respiratory disease in under-researched mining sectors**. The platinum and diamond sectors were chosen because of their importance to the South African economy, the large numbers of workers employed in each, and the mineralogical evidence for the likelihood of the development of previously unidentified disease. This theme is addressed in the third and fourth thesis papers. The first describes the risk for asbestos-related disease in diamond mine workers and the second, silicosis in platinum mine workers.

The two themes are depicted in Figure 1, while the essence of the four papers and this integrating narrative is summarised in Table 2.

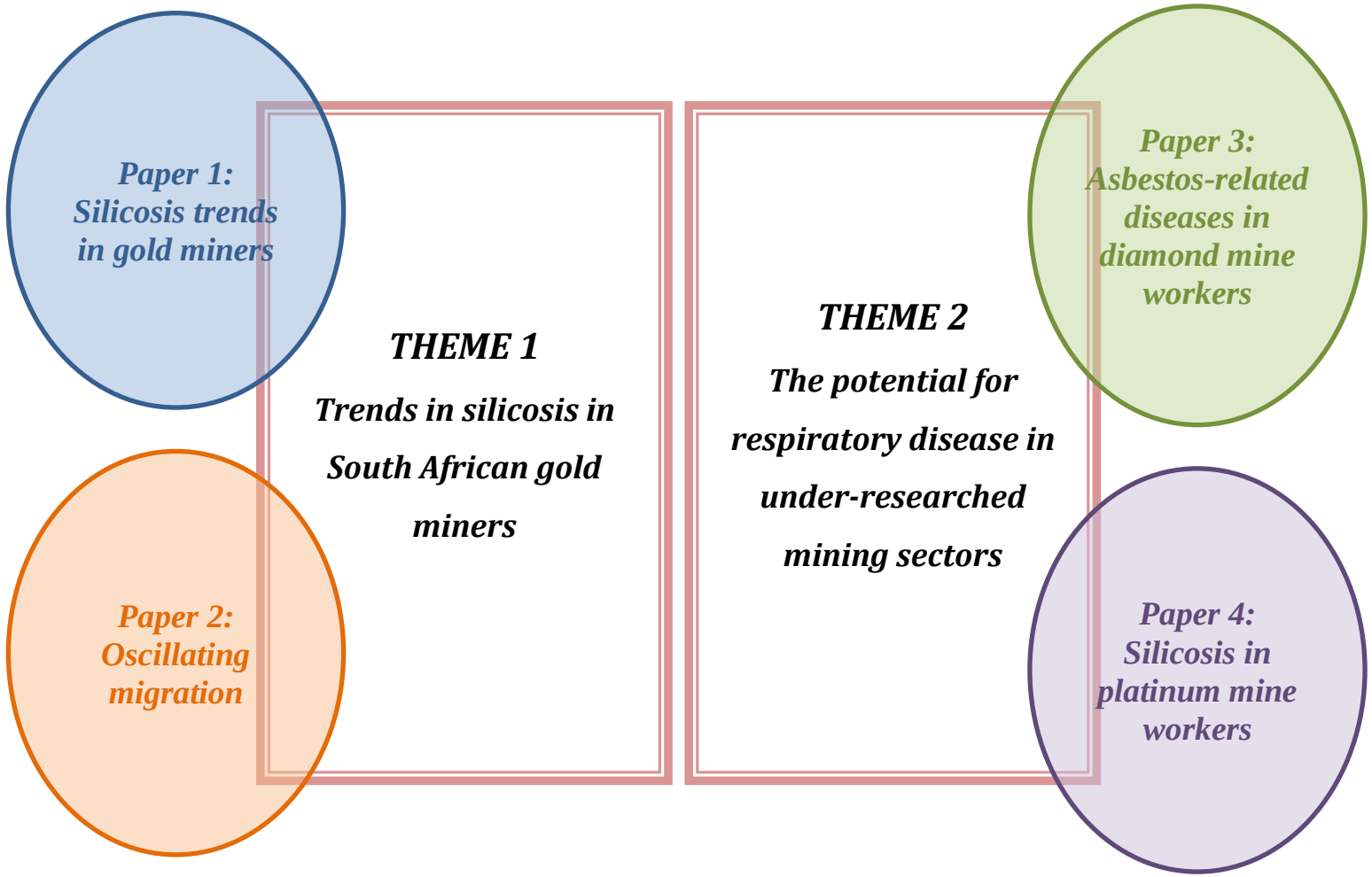


Figure 1. Themes and related scientific papers incorporated into the thesis

Table 2. Themes of the thesis addressed in the scientific papers

Themes	Paper 1 Silicosis in gold miners	Paper 2 Oscillating migration	Paper 3 Asbestos-related diseases in diamond miners	Paper 4 Silicosis in platinum mine workers	Integrating narrative
Trends in silicosis in South African gold miners	The proportion of black miners diagnosed with silicosis increased from 3% to 33% from 1975 to 2007; in white miners it increased from 18% to 22%.	Health care costs were externalised away from the mining companies as a direct result of oscillating migration, resulting in epidemics of diseases such as silicosis, tuberculosis and HIV.			Increasing trends are due to the migrant labour system, poor dust control, an inadequate occupational exposure limit, and an ageing workforce with increasing durations of employment.
The potential for respiratory disease in under-researched mining sectors			Diamond mine workers are at risk of developing asbestos-related diseases due to the composition of the rock.	Platinum mine workers are at risk of exposure to crystalline silica and thus of developing silicosis.	The PATHAUT database provides an opportunity for disease surveillance in miners of all commodities, including those in which risks of ill health are considered to be minimal.

1.2 SILICOSIS IN THE SOUTH AFRICAN GOLD MINING INDUSTRY

In this section of the Background, **DISEASES ASSOCIATED WITH SILICA DUST EXPOSURE**, particularly **silicosis and tuberculosis,** are described. The **onset and progression of silicosis** amongst both current and ex-miners are discussed, relevant to the ascertainment of disease and calculation of rates. A **HISTORY OF SILICOSIS IN THE SOUTH AFRICAN GOLD MINES** is provided, followed by a **LITERATURE REVIEW OF SOUTH AFRICAN SILICOSIS STUDIES** from 1978-2009. **FACTORS CONTRIBUTING TO THE HIGH RATES OF SILICOSIS IN GOLD MINERS IN SOUTH AFRICA** are then discussed in detail, viz. **oscillating migration and employment practices, the failure of the gold mines to reduce dust levels**, and **the South African occupational exposure limit for silica**. **THE AUTOPSY SURVEILLANCE DATABASE AT THE NATIONAL INSTITUTE FOR OCCUPATIONAL HEALTH** is described, with emphasis on its utilisation. The final sub-section describes **THE SOUTH AFRICAN SILICOSIS ELIMINATION PROGRAMME.**

1.2.1 DISEASES ASSOCIATED WITH SILICA DUST EXPOSURE

Exposure to silica dust (also known as crystalline silica or quartz dust) causes a broad spectrum of diseases, including silicosis, chronic obstructive pulmonary disease (COPD), lung cancer, and immune-related diseases (Murray et al., 2010). Both silica dust and silicosis are associated with tuberculosis (TB). Silicosis is an irreversible condition in which healthy lung becomes replaced with areas of fibrosis. Individuals with silicosis often have no signs or symptoms of the disease. In life, the disease is usually diagnosed using chest radiographs, but clinic-pathological studies have shown that silicosis may be missed using this diagnostic tool (Sun et al., 2008), and that diagnosis at autopsy is more sensitive (Hnizdo et al., 1993; Corbett et al., 1999).

Silicosis and tuberculosis

It is generally accepted that tuberculosis was introduced into the mines by the Cornish tin miners who flocked to South Africa in the hope of getting work on the Witwatersrand gold mines (Burke and Richardson, 1978). The bacillus soon spread throughout the mining community, facilitated by the dust in the mines, and the poor living conditions in the mine hostels where migrant workers were housed. When miners became sick, they were not allowed to work underground and were sent home with the bacillus which spread through the rural communities. South African gold miners now have one of the highest pulmonary tuberculosis rates in the world (Glynn et al., 2008).

Because of its close association with tuberculosis (Hnizdo and Murray, 1998), which was noted by Gorgas in a report to the Chamber of Mines in 1914 (Gorgas, 1914), silicosis is the occupational respiratory disease of major concern in the South African gold mining industry. Although HIV infection is the driving force behind the high rates of TB today (Corbett et al., 2004; Glynn et al., 2007; Murray et al., 2007; Glynn et al., 2008; Glynn et al., 2010), silicosis in the absence of HIV is still a risk factor for TB (Braun et al., 1991; Allen et al., 1992; Leroy et al., 1995). Together, silica dust, silicosis and HIV have a multiplicative effect on the development of tuberculosis on the South African gold mines. (Corbett et al., 2000; Sonnenberg et al., 2000). The effect of the HIV epidemic in a workforce that is already highly predisposed to TB from inadequately controlled silica dust exposure has been to drive the TB epidemic further out of control.

Onset and progression of silicosis

Silicosis has a long latency period of up to 20 years (Rees and Murray, 2007) and is progressive. Disease may develop only after dust exposure has ceased, long after the mine worker has returned home. Thus, rates and risks of silicosis in gold miners cannot be accurately calculated in studies that exclude ex-miners. The late-onset and progression of silicosis was illustrated by a cohort study on white ex- gold miners which showed that 52% had developed silicosis in 2004 (Murray and Hnizdo, 2005), compared to 14% some 13 years earlier (Hnizdo and Sluis-Cremer, 1993). In a more recent study on black ex- gold miners living in Lesotho, the prevalence of silicosis increased from 26.6% to 27% in only one year (Park et al., 2009).

1.2.2 HISTORY OF SILICOSIS IN THE SOUTH AFRICAN GOLD MINES

The history of silicosis in South Africa goes back as far as the gold mining industry. Silicosis was recognised as a problem in the early 1900s, not long after the discovery of gold. By 1912, silicosis rates had reached 48% in white drillers, as reported by the Miners' Phthisis Commission in 1912 (the Commission was appointed under the provisions of the Miners' Phthisis Allowance Act no. 34 of 1911).

A number of controls were put in place, including ventilation and watering to reduce dust. By the 1930s, dust levels in the mines had dropped dramatically (Hnizdo and Sluis-Cremer, 1993). Silicosis was thought to be under control as a reduction of silicosis rates in white gold miners followed; no attention was paid to disease in black miners.

The focus shifted away from dust in the mines until the 1970s when the mines were required to pay a contribution to a compensation fund based on the conditions underground (Hnizdo and Sluis-Cremer, 1993). Although Hnizdo and Sluis-Cremer stated that dust levels consequently decreased further by around 30%, the 1994 Commission of Inquiry into Safety and Health in the Mining Industry found no evidence that dust levels had decreased over the last 50 years (Leon et al., 1995).

Apartheid laws had a profound effect on disease rates. Black men were forced to live in compounds on the mine property without their families and in confined quarters. Exposure to dust caused chronic respiratory disease and, together with sub standard housing and sanitation in the mine hostels, provided ideal conditions for the spread of infectious diseases.

Labour patterns among white and black gold miners were very different until the 1980s. While white miners worked for long periods on the mines, often decades, black miners were employed on short-term contracts. Until 1983, black miners were not allowed to continue working underground if they contracted tuberculosis (Leger, 1992) and were sent home. Even if healthy, black miners worked on the mines for relatively short periods before returning home; because of their contractual conditions, they generally did not make a life-long career of mining. However, in the 1980s, the South African gold mining industry downsized its labour force, and the black workforce began to stabilise as employment contracts became permanent. Consequently, exposure to dust increased, in terms of both dose and time and, by the 1990s, silicosis rates in black workers on the mines were high (Churchyard et al., 2004).

In the mid-1990s, the South African political climate changed. Laws discriminating on the basis of race, including the payment of compensation for occupational respiratory disease, were replaced by new legislation. In 1994, a Commission of Inquiry into Safety and Health in the Mining Industry was led by Judge RN Leon (Leon et al., 1995), which was influential in renewing research into mining and health. The Commission raised a number of concerns, including high rates of tuberculosis, silicosis (and other pneumoconiosis), asbestos-related diseases, and noise-induced hearing loss. Tuberculosis rates were reportedly 58 per 1000 after 15 years of exposure, and the probability of developing silicosis, as high as 30%. While the Commission did not produce a list of research priorities, it recorded that "a great deal of epidemiological and biostatistical research remains to be done before effective control of occupational diseases can be assured (Vol. 1 para 4.8.7). One of the major recommendations of the report was to "conduct enhanced and properly targeted research, and to develop and acquire publications, including statistical publications."

1.2.3 LITERATURE REVIEW OF SOUTH AFRICAN SILICOSIS STUDIES, 1978-2009

Until the 1990s, there were very few studies on the extent of silicosis in South African gold miners. Studies published on the prevalence of silicosis in a variety of settings and populations from 1978 to 2009 are summarised in Table 3.

A cohort of more than 2 200 white gold miners employed for an average of 24 years from 1940 to the early1970s was established from 1968 to 1971. Three publications since then have reported on the increasing prevalence of silicosis in the cohort which was 7% in the first analysis reported in 1978 (Irwig and Rocks,

1978). By 1991, 14% of the cohort had radiological silicosis (Hnizdo et al., 1993). Thirteen years later, in 2004, more than 80% of the cohort had died and autopsy examinations revealed extensive progression: nearly 52% had silicosis with a mean duration of exposure of 40 years (Murray and Hnizdo, 2005).

This cohort provides a clear illustration of the progression of silicosis, long after retirement, and represents the only analysis of silicosis in white gold miners. No reports have been published on any other white gold miners subsequently, and no other long term cohort in gold miners has been established.

Little had been published on silicosis in black gold miners before Cowie and van Schalkwyk's 1987 study (Cowie and van Schalkwyk, 1987) which demonstrated much lower disease rates in black miners compared to Irwig and Rocks' report in white miners several years earlier (Irwig and Rocks, 1978). This is not surprising, given that black miners were employed on short term contracts before 1984 and white miners were permanent, long-term employees.

Cowie and van Schalkwyk's study was the first in a series of analyses of silicosis in black miners. Three more cross-sectional studies of radiological silicosis in living ex-miners from Lesotho, Botswana and the rural Eastern Cape followed in the 1990s. All reported high proportions of silicosis of up to 36% (Steen et al., 1997; Trapido et al., 1998a; Meel, 2002). Mean durations of employment ranged from 12 to 16 years.

By 2001 (Churchyard et al., 2004), many years after short-term contracts had been phased out, the proportion of black employed gold miners with silicosis was 14 times higher than that reported in 1984 (Cowie and van Schalkwyk, 1987).

Only one follow-up study has been conducted, in 779 black gold miners (Park et al., 2009), but the period between the two follow-up visits was very short at only a year. The proportions of miners with radiological silicosis were 27%, similar to that reported in the three previous cross-sectional studies.

One trend analysis in deceased gold miners showed a 3.5 fold increase in silicosis at autopsy in black employed miners who died from unnatural causes, from 1975 to 1991 (Murray et al., 1996). The analysis was limited to black gold miners who were employed at the time of death and died of external causes, and extended over a 17 year period.

All the other studies were cross-sectional, used X-rays (miniature or standard size) as the diagnostic tool, and the study populations were small, at less than 1000. Additional shortcomings of each, most of which would have resulted in an under-estimate of the prevalence of silicosis, are listed in Table 3.

Nevertheless, all the studies demonstrated a consistently high prevalence of silicosis in black gold miners after 1987. What is lacking, are studies to show if and how the prevalence of silicosis is changing. This can only be done by analysing long term trends in silicosis in large numbers of gold miners. Furthermore, in order to accurately calculate the extent of silicosis in the gold mining industry and associated risk factors, studies are required that include both current and ex-

miners from all population groups with a range of silica dust exposures (both in terms of dose and duration), across a range of age-groups.

The absence of occupational respiratory health surveillance databases is the primary reason that studies on silicosis have been largely limited to cross-sectional designs limited to selected populations. This is discussed in more detail in Section 4. One database that is suitable for more complex analyses is the autopsy database (PATHAUT) at the National Institute for Occupational Health (see Section 1.2.5 below).

Table 3. Studies of silicosis in South African gold miners, 1978 – 2009

Authors	Study design	Study period	Study Population	Study site	Sample size n	Mean/range of employment (years)	Diagnostic tool	Proportion with silicosis (%)	Limitations
White gold miners									
Irwig and Rocks 1978	Cross sectional	1968 to 1971	Employed white miners aged 45-54	All areas	1 973	> 10	Chest X rays	6.8	White miners only*†
Hnizdo and Sluis-Cremer 1993	Cohort	1968 to 1991	White ex-miners - living and dead	All areas	984	23.5	Chest X rays	14.0	White miners only†
Murray and Hnizdo 2005	Cohort	1968 to 2003	Deceased white gold miners	All areas	1 476	23.5	Autopsy	51.6	White miners only
Black gold miners									
Cowie and van Schalkwyk 1987	Cross sectional	1984	Employed black miners	Orange Free State, SA	132 765	Not stated	Chest X rays	1.4	Black miners only; no ex-miners; denominator included new recruits *†
Murray et al. 1996	Cross sectional trend analysis	1975 to 1991	Deceased black gold miners	All areas	16 454	4.4 - 6.9	Autopsy	9.3 -12.8	Black miners only; 62% of men employed for < 5 years 78% younger than 40*
Steen et al. 1997	Cross sectional	1994	Living black ex-miners	Botswana	304	15.5	Chest X rays	26.6 - 31.0	Black miners only; selection bias; no current miners *§†
Trapido et al. 1998a	Cross sectional	1996	Living black ex-miners	Eastern Cape, SA	238	12.2	Chest X rays	22.0 - 36.0	Black miners only; no current miners *§†
Meel 2002	Cross sectional	1997 to 1999	Living black ex-miners – hospital patients	Eastern Cape, SA	300	Not stated	Chest X rays	34.0	Black miners only; selection bias (hospital patients); no current miners *§†
Girdler-Brown et al. 2008	Cross sectional	1999	Living black ex-miners	Lesotho	624	25.6	Chest X rays	24.6	Black miners only; no current miners*§†

Churchyard et al. 2004	Cross sectional	2000 to 2001	Employed black miners older than 37	North West province, SA	520	21.8	Chest X rays	18.3 - 19.9	Black miners only; no ex-miners; healthy worker effect *§†
Park et al. 2009	Cohort	1999 to 2000	Living black ex-miners	Lesotho	553	26.1	Chest X rays	27.0	Black miners only; no current miners; short follow-up of 1 year *§†

* cross-sectional study design
§ small numbers
† insensitive diagnostic tool

1.2.4 FACTORS CONTRIBUTING TO THE HIGH RATES OF SILICOSIS IN GOLD MINERS IN SOUTH AFRICA

The migrant labour system, together with employment practices, played a major contributing role in the development of silicosis and the high rates that we see today. Dust levels in the gold mines have not decreased in several decades, and the South African current occupational exposure limit (OEL) of 0.1 mg/m^3 may not be low enough to prevent disease.

Oscillating migration and employment practices

Much has been written on oscillating migration in the South African gold mines (Crush and Wilmot, 1995; Harington et al., 2004; Crush et al., 2005), where it is still firmly entrenched, including the recent paper that forms part of this thesis (Rees et al., 2010).

History of the migrant labour system

The migrant labour system was firmly established in South Africa a decade before the discovery of gold. As early as the 1870s, farmers in the Western Cape solved their labour shortages by recruiting workers from rural areas in South Africa, from neighbouring countries and even further afield from the United Kingdom and Germany (Wilson, 1972).

The first mines to employ migrant labourers were the diamond mines, shortly followed by the gold mines. Only eight years after the discovery of diamonds in 1866, 10 000 black men were employed in Kimberley, most of whom were oscillating migrants (Wilson, 1972). They worked in the mines, picking, digging and shovelling, to earn money for various reasons, including cattle for lebola (payment

to the bride's family) and guns to protect themselves against white settlers (Turrell, 1986). Black men comprised almost 90% of the diamond mining labour complement, varying from 10 000 to 30 000; 50 000 to 100 000 travelled to and from Kimberley each year (Turrell, 1986).

The men usually worked from three to six months before returning home; some never returned to the mines (Turrell, 1986). This did not suit the mine owners who wanted a disciplined workforce and they tried to find ways to control the supply of labour and to keep the men in Kimberley. The pass system, introduced in 1872, went a long way to enabling this control. While the system inhibited desertion (leaving the mines before a contract had expired), it did not guarantee uninterrupted labour (Turrell, 1986), as the men, more suited to farming, were not dependent on mining for a livelihood. The pass laws established and maintained the forced oscillating migrant system as they effectively limited black urbanization (Wilson, 2001). In addition, hut taxes were introduced in the late 1800s (Massey, 1978), which forced men to seek employment in the mines.

Along with other factors, the development of underground mining was seen as a justifiable reason for introducing closed compounds in 1885. Underground mining was expensive and new and required workers to stay for longer than three months to increase labour efficiency (Turrell, 1986). The men were isolated for the period of their contracts and were no longer able to sell their labour for the highest wage (Turrell, 1986); the mines were thus successful in developing an experienced and cheap workforce.

The patterns of compounds and oscillating migration established in Kimberley were followed by the gold mines after the discovery of gold in 1886 (Wilson, 1972). The system became entrenched on mines of other commodities although information on historic figures for migrant workers on these mines is sparse (Wilson, 1972).

De Beers Diamond Mining Company ended the migrant labour system on its mines in 1973 (Wilson, 2001). The compounds were converted for families and single workers, and limited contracts were no longer issued. Unfortunately, the South African government allowed only those who had lived in Kimberly for at least 10 years to be employed as permanent workers, and many did not qualify (Wilson, 2001). Consequently, many miners, especially those from neighbouring countries, returned home.

Ten thousand diamond miners were working in Kimberly eight years after the first diamond was discovered in 1867, but it took less than 10 years for 100 000 men to be employed once the gold fields had opened (Wilson, 2001).

Wages were kept to a minimum by the Chamber of Mines and did not, in fact, increase for almost 60 years, from 1911 to 1969 (Wilson, 2001). Men were sent home to the rural areas after their contracts expired and laws prevented them from staying near the mines with their families (Wilson, 2001). Money earned on the mines was spent locally rather than being invested in the rural areas which gradually became impoverished labour reserves (Wilson, 2001).

Numbers of migrant labourers employed in the South African mines

In 1920 almost 100 000 migrant labourers were recorded as working on the mines (Figure 2). The number increased steadily, peaking in 1970 at 265 000. By 1990, the number had dropped slightly to 192 000 by (Crush et al., 2005). From 1990 to 2000, although the total number of men recruited to work on the mines decreased, the proportion from neighbouring countries increased. In 2000, 57% of all new mine recruits were foreigners, primarily from Lesotho (Crush et al., 2005).

In 1974 there was a drive by the Chamber of Mines to replace foreigners with black South Africans. By 1979, the gold mining work force comprised 16% foreigners, a significant reduction from 37% in 1966 (Harington et al., 2004). However, despite predictions that contract migrant labour was likely to become a thing of the past (Davies and Head, 1995), migrants remain a major labour source on all South African mines (Table 4) . Today, many men are employed on the mines as contract workers, a large proportion of whom are migrant labourers, including foreigners (Steinweg and de Haan, 2007).

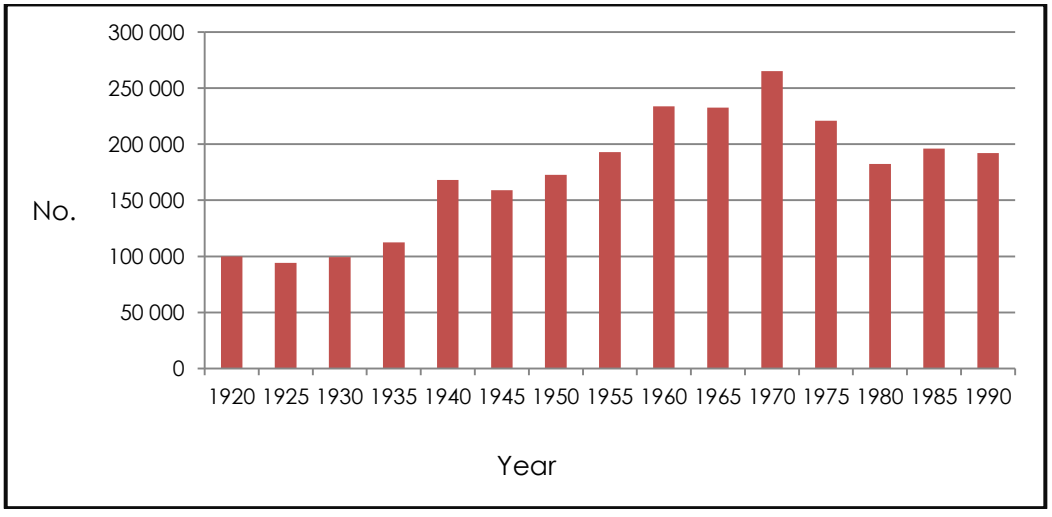


Figure 2. Contract labour migration to South African mines, 1920 – 1990 [adapted from Crush et al., 2005]

Table 4. Number of foreign workers employed by South African gold, platinum and coal mines, 1996-2010 [Rees et al., 2010, and updated from TEBA for 2008-2010]

	Country				
Year	**Swaziland**	**Mozambique**	**Lesotho**	**Botswana**	**Total**
1996	14 371	55 022	81 357	7 932	158 682
1997	12 960	55 027	76 360	7 536	151 883
1998	10 338	52 011	60 450	6 223	129 022
1999	9 307	46 890	52 436	5 130	113 763
2000	8 160	44 014	51 351	4 343	107 868
2001	7 794	45 254	49 599	3 651	106 298
2002	8 587	50 589	54 390	3 551	117 117
2003	7 885	52 205	54 202	4 246	118 538
2004	7 521	48 099	48 437	3 923	107 980
2005	6 878	46 256	43 693	3 257	100 084
2006	7 124	46 709	46 082	2 992	102 907
2007	7 099	44 879	45 608	2 845	100 431
2008	6 397	43 004	42 851	2 654	94 906
2009	5 855	39 090	38 559	2 357	85 861
2010	5 009	35 782	35 179	2 042	78 012

In 2010, an average of 32% of mine workers were reportedly contractors (Table 5).

Table 5. Number and proportions of contractors employed in different mining sectors in 2010 (DMR, 2011b)

Mining sector	**Total employees**	**Contractors**	
	n	**n**	**%**
Iron ore	18 578	10 963	59.0
Manganese	5 690	3 274	57.5
Coal	71 588	34 640	48.4
Chrome	13 719	5 697	41.5
Copper	3 309	1 171	35.4
PGMs	168 591	5 7591	34.2
Diamonds	11 159	2 694	24.1
Gold	143 750	22 338	15.5

The platinum mining sector is currently the largest employer in the mining industry. Until 1994, the platinum mines relied heavily on migrant labour (van Wyk, 2007). Today, the number of contract workers is still high and is increasing (Hamann, 2003; Steinweg and de Haan, 2007). In 2006/07, 42-46% of Anglo Platinum's workforce comprised contract workers (Steinweg and de Haan, 2007). Although

there is a move to phase out migrant labour and the mine hostels, many of the migrant workers are now working for sub-contractors and living in the hostels.

Migrancy and disease

Migrant workers worldwide are particularly vulnerable with regard to occupational health and safety. They are more represented in dangerous industries and hazardous jobs, and there is evidence that employment conditions and associated work organization of most migrant workers are dangerous to their health (Benach et al., 2010). Certainly, black migrant miners were employed in dustier jobs than white miners in the gold mines for many decades. Contractors may not have the same access to health care facilities and other mine services (such as training programmes) as permanent workers (van Wyk, 2007).

The migrant labour system caused poverty, resulted in social disintegration, and caused disease and its spread. Many hundreds of thousands of migrants came to work in the South African mines over the last century, and a high proportion returned home diseased. When migrant mine workers return home to the rural areas of South Africa (Figures 3 and 4) or to neighbouring countries, they take their disease with them. This resulting externalisation of health care to labour-sending areas (Trapido et al., 1998b) has resulted in high disease rates but poor accuracy in ascertainment of precise numbers of affected miners. Not only are the health services over-burdened, but so are their families who need to take care of them. Many of those who have left the mines before retirement are physically incapable of working and therefore cannot earn an income and contribute towards household costs. In many areas, health services are, at worst, unavailable or, at best, under-resourced. Migrant workers are thus unable to access health services,

including the autopsy service and, consequently, their disease remains undiagnosed and untreated, reducing the ascertainment of disease.

Studies that have reported high rates of silicosis in gold miners and ex-miners may have underestimated the burden of disease in these populations. Studies in current miners underestimate the extent of silicosis as many develop the disease only after they have left the mining industry due to the long latency period after exposure to silica dust. Cross-sectional studies of ex-miners also underestimate disease as ex-miners often represent survivor populations.

Foreign migrant workers are often invisible in national statistics (Wickramasekara, 2007). They do not appear in the population register of the Department of Home Affairs and their deaths are therefore not included in mortality statistics. Thus, the burden of silicosis (and other occupational diseases) is unlikely to be accurately reflected in general population disease and mortality rate calculations.



Figure 3. Photograph of a dwelling of an ex-gold miner in rural KwaZulu Natal (G Nelson)



Figure 4. Photograph of dwellings in rural KwaZulu Natal (G Nelson)

Failure of the gold mines to reduce dust levels

As the costs of treating, and often diagnosing, compensable disease have been largely externalised to the remote labour-sending areas, the mining companies are not overtly faced with the consequences of exposing their workers to unsafe dust levels. Most men do not stay in the vicinity of the mines once they have left mine employment and, consequently, there is limited pressure from civil society or organised labour to address the issue of these high rates of disease, and to reduce dust levels in the mines.

It seems that dust levels have not changed for many decades, a conclusion reached by the 1994/95 Commission of Inquiry into Safety and Health in the Mining Industry. The report stated that "dust levels have remained roughly the same over a period of about 50 years" (Leon et al., 1995). There is no reason to believe that they have decreased since the Commission (Churchyard et al., 2004). With no reduction in dust levels, one cannot expect silicosis rates in gold miners to have declined.

The South African occupational exposure limit for silica

There is evidence to suggest that the South African occupational exposure limit (OEL) of 0.1 mg/m^3 is not low enough to prevent silicosis. In the early to mid-1990s, three separate studies showed that different limits (called OELs, threshold limit values (TLVs), or permissible exposure limits (PELs), depending on the agency setting the standard) were not low enough to prevent silicosis. Hnizdo and Sluis Cremer (Hnizdo and Sluis-Cremer, 1993) calculated the risk of radiological silicosis (ILO profusion category of 1/1 or more) in a cohort of 2 235 white South African gold miners. Fourteen percent developed signs of silicosis at an average age of 55.9 years. At the highest exposure level of 15 mg/m^3-years, representing approximately 37 years of gold mining at an average respirable dust concentration of 0.4 mg/m^3, the cumulative risk for silicosis reached 77%. In the same year, Hnizdo et al. predicted, in a dose-response relationship model, that, after 10 years of exposure at a level of 0.5 mg/m^3, the prevalence of silicosis would be almost 90% (Hnizdo et al., 1993).

Steenland and Brown (Steenland and Brown, 1995) studied a cohort of 3 330 gold miners in a mine in the USA, who were exposed to a median silica level of 0.05 mg/m^3. They calculated that a 45-year exposure under the Occupational Safety and Health Administration (OSHA) standard of 0.09 mg/m^3 would lead to a lifetime risk of silicosis of 35% to 47%. The limit has since been reduced to 0.05 mg/m^3, in line with that of NIOSH (National Institute for Occupational Safety and Health).

A year later, Kreiss and Zhen (Kreiss and Zhen, 1996) published a report on the exposure-response relationship for silicosis among 134 men in a mining town in the USA. Average silica exposure was strongly associated with silicosis prevalence

rates: 13% had an average exposure of 0.025-0.05 mg/m^3, 34% had exposures of > 0.05-0.1 mg/m^3, and 75% had average exposures of more than 0.1 mg/m^3, suggesting that the relatively low PEL of 0.1 mg/m^3 for silica also does not protect against radiologic silicosis.

The results of these three studies were summarised by Greaves (see Figure 5), who concluded that even a limit of 0.05 mg/m^3, NIOSH's recommended exposure limit (REL), will not be sufficiently protective for a substantial proportion of workers (Greaves, 2000).

In 2005, Steenland et al. reported on the risk of developing silicosis, as well as dying from silicosis at age 75, after 45 years of exposure to silica at a level of 0.1mg^3/ml (Steenland, 2005). The risk was 47 to 77%, far higher than the OSHA acceptable risk of serious disease or death for workers of 0.1%. The absolute risk of death from silicosis was calculated as 1.9% (95% CI 0.8% to 2.9%).

Around the same time, Churchyard et al. reported a prevalence of silicosis of 18.3-19.9% in a cross-sectional study of 520 black gold miners, depending on the X-ray reader. The mean intensity of quartz was 0.053 mg/m^3 (range 0-0.095), well below the South African OEL, supporting the previous studies and Greaves' conclusion that an OEL of 0.1 mg/m^3 is not protective against silicosis (Churchyard et al., 2004). The limit set by the American Conference of Industrial Hygienists (ACGIH) in 2006 is 0.025 mg/m^3 but no data are available on silicosis prevalence rates at this standard.

The South African OEL for silica dust is a legislated limit. It is calculated as an 8-hour time weighted average (TWA) dust measurement and, as such, peak exposures are not recorded. Silica dust levels are reported by the mines themselves and there is no external validation of the measurements. Enforcement by the Department of Mineral Resources is also problematic. Many of the mines are noncompliant in reporting silica dust measurements. For example, in the period 1995 to 1997, only 48 gold mines reported data to the Department of Mineral Resources, many fewer than the number of gold mines operating in South Africa at that time. Only eight of the 48 mines (17%) reported silica dust levels below 0.1 mg/m^3 (Rees et al., 1999; Rees et al., 2001).

One of the targets of the Mine Health and Safety Council's (MHSC) Silicosis Elimination Programme (see Section 1.2.6 below) was for 95% of all crystalline silica measurements to be below the OEL of 0.10 mg/m^3 by December 2008. Data from the DMR for 2006 and 2007 indicate that the compliance rate for the country, overall, is above 90% but below the target of 95% (DOH, 2010). In 2006, the target was reached only by the Eastern Cape, Western Cape and Northern Cape provinces. In 2007, there was a decrease in compliance in most provinces, with only the Eastern Cape and North West province reaching 95%. Gauteng and Free State, the provinces in which most of the gold mines are located, did not achieve 95% compliance in either year. The data for 2008 were not available at the time that the report was written, so it is uncertain if the December 2008 target was reached.

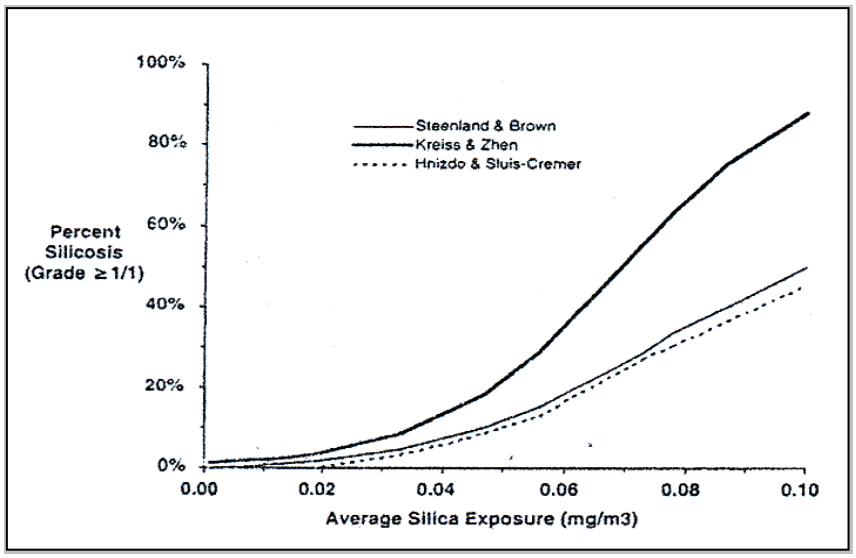


Figure 5. Comparison of silicosis risks in three studies of hard rock miners: average respirable silica levels derived from cumulative exposure data, assuming 45 years of continuous exposure (Greaves, 2000) *[permission to reproduce this figure was obtained from RightsLink: www.copyright.com]*.

1.2.5 THE AUTOPSY DATABASE AT THE NATIONAL INSTITUTE FOR OCCUPATIONAL HEALTH

In terms of the Occupational Diseases in Mines and Works (ODMW) Act, the Pathology Division of South Africa's National Institute for Occupational Health provides an autopsy service for deceased miners and ex-miners for the diagnosis of compensable disease (see Section 2.1.1 for more detail).

While autopsies have been performed for many decades, until 1975, all reports were paper-based. The electronic database has been fully functional for more than 30 years, and is part of the PATHAUT (Pathology Automation) System which was developed as a result of legislation providing compensation for miners who developed disease as a direct result of their working conditions. The development and structure of the database, which are described in detail in two earlier

publications (Soskolne et al., 1976; Hessel et al., 1987a) are discussed in more detail in Section 2.1.1.

Although it is essentially an administrative database for compensation, this is the only database that can be used for trend analyses of occupational respiratory diseases. The database provides for the recording of, not only disease data, but additional information related to demography and exposure. It contains the only data on occupational lung disease in the South African mining industry, diagnosed by standard pathological methods which are far more sensitive and accurate than chest radiography. It currently comprises more than 105 000 autopsy records of miners from all population groups from all mining sectors and all regions of South Africa (Ndlovu et al., 2010), dating back 37 years to 1975. An added advantage is that bias due to the healthy worker effect is minimised as many ex-miners also come to autopsy, regardless of the clinical cause of death or their disease status.

Around 80% of the autopsied mine workers are from the gold mining sector which has been, historically, the largest mining industry in South Africa. This high "yield" is also a consequence of the proximity of the gold mines to Johannesburg. Over the years, however, the proportion of gold miners has decreased (62.5% in 2010) (Ndlovu et al., 2011), in line with the decline in gold production in South Africa. With increasing platinum production, the number of platinum mine workers coming to autopsy is increasing each year. Most other mining commodities have some representation, e.g. coal, diamond and asbestos, as well as the less common commodities such as tin and vanadium. Employees of the Iron and Steel

Corporation of South Africa (ISCOR) are covered by the same compensation legislation and are also represented in the PATHAUT system.

Utilisation of the autopsy database

The PATHAUT database is an important resource for both surveillance and research on miners and ex-miners of all commodities. The inclusion of ex-miners in the database allows for research on diseases with long latency periods, such as silicosis and lung cancer. In addition, unlike databases on current miners, the healthy worker effect is minimised, particularly with regard to white miners. There is a high uptake of the autopsy service, regardless of the clinical cause of death, both when miners die while employed and after they have left the mining industry. The majority of black miners on the database are those who have died while employed; uptake of the autopsy service is high in this group, regardless of the clinical cause of death, i.e. disease or external causes. Black miners who have left the industry for any reason (e.g. disease, retirement or retrenchment) are under-represented on the PATHAUT database because they have difficulty in accessing the autopsy service.This and other biases are discussed in more detail in section 4.3.

While the PATHAUT database is underutilised for surveillance, it has been a major source of material for research over the last three decades as the data provide the opportunity to study respiratory diseases in all mine workers, including those from 'smaller' mining sectors. Long term disease trends can also be studied. In addition to some articles published in the 1980s, describing the earlier PATHAUT data (Soskolne et al., 1976; Hessel et al., 1987a; Hessel et al., 1987b), more than 100 scientific papers and over 30 reports have been published since 1976.

High rates of lung disease are found at autopsy (Murray et al., 2001; Ndlovu et al., 2011). The main occupational respiratory diseases diagnosed at autopsy are pulmonary tuberculosis, obstructive lung disease (including emphysema), pneumoconiosis (primarily silicosis), asbestosis, lung cancer, and mesothelioma.

Annual reports from 1998, using data on the PATHAUT database, and prepared by the Pathology Division of the NIOH, are available on the National Health Laboratory Service (NHLS) website (www.nioh.ac.za).

Numbers and rates of the main compensable respiratory diseases diagnosed at autopsy are reported by population group, commodity, age group and years of service. Diseases with the highest rates are, consistently, pulmonary tuberculosis, emphysema and silicosis, at 231, 250 and 233 per thousand, respectively, in 2010 (Ndlovu et al., 2011). However, the commodity-specific statistics that are described in the PATHAUT annual surveillance reports are calculated on the basis of where the worker was employed for most of his working life and do not take into account that he might have been employed in a number of different mining sectors. Many miners have worked in more than one mining sector and therefore have 'mixed exposures'.

1.2.6 THE SILICOSIS ELIMINATION PROGRAMME

The International Labour Organisation (ILO) and World Health Organisation (WHO) Global Program for the Elimination of Silicosis (GPES) was established following the recommendation of the 12th Session of Joint ILO/WHO Committee on Occupational Health in 1995. The Committee identified the global elimination of silicosis as a priority area for action in occupational health, obliging countries to

place it high on their agendas (GOHNET, 2007). The GPES targeted countries in which silicosis was an occupational health priority and, in June 2004, South Africa actively joined the Programme which is co-ordinated by the National Silicosis Working Group under the leadership of the Department of Labour. Other active South African agencies include the Department of Mineral Resources; the Department of Health; the Chamber of Mines (representing employers); three major trade unions, viz. Congress of South African Trade Unions (COSATU), National Council of Trade Unions (NACTU) and Federation of Unions of South Africa (FEDUSA); the NIOH; and academic and research institutions. The South Africa government is committed to reduce the prevalence of silicosis significantly by 2015 and to totally eliminate silicosis in workplaces by 2030.

The government identified four preventive actions on which to focus, viz. prevention of exposure through regulatory, enforcement and management systems; awareness raising, training and education at all levels; application of effective inspection and health surveillance systems and tools; and implementation of preventive health and safety measures (DOL, undated).

The MHSC is a partner in the GPES. Since its establishment in 1994, the MHSC has funded research into dust measurement and control, and understands the importance of evaluating existing practice in relation to control requirements, setting exposure reduction targets, and establishing best practice for targets for silica exposure to be met. The Council is therefore funding a project, which is being undertaken by the NIOH, in line with the GPES (NIOH, 2010). The project consists of three parts, viz. dust measurement and reporting, environmental

engineering / dust control, and human resources training and management. More details on each of these parts are available on the NIOH website (NIOH, 2010).

The overall success of the National Programme can only be measured by analysing disease data collected by surveillance systems such as the PATHAUT system which provides an opportunity to measure both historical and future silicosis trends; the PATHAUT database is described in detail in Section 2.1.1 below.

1.3 THE POTENTIAL FOR RESPIRATORY DISEASE IN UNDER-RESEARCHED MINING SECTORS

This section of the Background justifies the choice of the diamond and platinum mining sectors for the analysis of mine workers at risk of developing occupational respiratory diseases. Both mining sectors are under-represented in the health literature. Both are important in terms of their contributions to the South African economy, and **production and employment** in the two sectors are discussed. **DIAMOND MINE WORKERS** were selected, based on evidence pointing to a risk of developing **ASBESTOS-RELATED DISEASES**, including the **mineralogical content of kimberlite**, the **geographical location of asbestos deposits** relative to diamond deposits in South Arica, and previous reports of **asbestos in diamond mines**. Similarly, **PLATINUM MINE WORKERS** were selected, based on their risk of exposure to crystalline silica due to the **mineralogy of the Bushveld Complex** where platinum occurs, and reports on **silica dust in platinum mines**, which may result in the development of **SILICOSIS.**

Much of the research on occupational lung diseases in mine workers, including that from the PATHAUT database, has focused on gold miners and, to a lesser degree, asbestos and coal miners. Many of the other mining sectors have been overlooked in terms of health research, including the diamond and platinum mining industries. These two sectors were chosen as the focus of the second theme of the thesis because of their importance to the South African economy, as well as the objective evidence for the potential for exposure of workers to minerals that are secondary to that being mined, that may put them at risk of developing disease.

Pneumoconioses, such as silicosis and asbestosis, have been diagnosed in diamond and platinum mine workers since the early days of the autopsy service at the, now, National Institute for Occupational Health (Goldstein and Rendall, 1969; Goldstein and Webster, 1970), but these reports were not able to specify if the miners had been employed in other mining sectors or not. There has been no research on miners employed exclusively in one or the other mining sector.

There is ongoing exploration for untapped reserves of minerals and commodities in South Africa; and development of new technologies to extend the lifetimes of existing mines and to expand mining operations. Diamonds and the PGMs, in particular, are two of the dominant targets for exploration activity in South Africa (MEG, 2007; DMR, 2010). There is no information on the health effects of mining procedures on workers in these two economically important industries.

1.3.1 PRODUCTION AND EMPLOYMENT IN THE DIAMOND AND PLATINUM MINING SECTORS

The South African diamond mining sector

The diamond mining industry is the oldest large scale mining industry in South Africa. The first diamond was discovered in South Africa in 1867 near Hopetown; by 1873, the first mines were operational.

In the 1870s and 1880s the Kimberley Mines (kimberlite mines) were producing approximately 95% of the world's diamonds. South Africa remained the world's top producer in carat terms until 1933, when it was overtaken by Zaire. It continued to be the world's top producer by value until 1960 when Russia began producing diamonds (DMR, 2007).

The world's known diamond reserves are estimated to be at around 300 million carats (U.S. Geological Survey (USGS). In 2010, South Africa produced 8.8 million ct (DMR, 2011c). Ranked 7th in the world in terms of rough diamond production, South Africa currently produces in the region of 5% of global production (Mbendi, 2011). Until the recent economic recession, South Africa's diamond industry produced a stable 8 to 15 Mct annually, of which 90% was exported (DMR, 2011c), and employed 15 000 to 23 000 workers (Table 6). In 2010, Diamond miners comprised 2% of the total mining workforce.

Table 6. Diamond mining in South Africa: production and employment, 1980 - 2010 (DMR, 2003; DMR, 2011d)

Year	Diamond production (X10³ ct)				No. employed
	Alluvial mining	Kimberlite mining	Marine mining	Total	
1980	1 772	6 703	45	8 520	23 372
1985	1 298	8 916	48	10 263	18 352
1990	1 198	7 380	130	8 708	22 982
1995	960	8 625	98	9 684	15 548
2000	1 004	9 714	61	10 780	15 018
2005	1 381	14 340	55	15 776	22 033
2010	285	8 511	68	8 863	11 159

Figures 6 and 7 are photographs of an open cast and underground diamond mine, respectively.



Figure 6. Photograph of an open cast diamond mine [Venetia mine, Limpopo province]. Note generation of dust by truck in top right-hand corner (G Nelson)



Figure 7. Photograph of the big hole at Premier underground diamond mine, Cullinan, Gauteng (G Nelson)

The South African platinum mining sector

Platinum production in this country now exceeds that of gold; it has more than doubled since the late 1980s, from 128 000 kg in 1987 to 287 300 kg in 2010 (DMR, 2003; DMR, 2011c). Until 1996, all PGMs produced were exported. In 2010 South Africa exported 85% of the PGMs it produced, valued at R66 billion. The price of platinum also continues to rise: from US$ 291 per ounce in 1985, US$ 424 in 1995, US$ 897 in 2005, to a current price of more than US$1800 today (mid-September 2011).

Table 7. Platinum mining in South Africa: production and sales of PGMs, and employment, 1980 – 2010 (DMR, 2003; DMR, 2011c; DMR, 2011d)

Year	Production X 10^3 kg	Mass exported X 10^3 kg	Export value X $R10^3$	Total value X $R10^3$	No. Employed
1980	114.3	112.1	851 083	851 195	77 404
1985	121.7	118.1	1 998 461	1 998 461	73 880
1990	141.9	135.6	5 164 216	5 164 216	97 373
1995	183.1	175.2	6 572 506	6 572 506	91 528
2000	206.8	198.9	24 645 761	27 094 627	96 273
2005	303.0	259.0	33 481 439	38 450 547	155 043
2010	287.3	244.4	65 894 341	73 786 910	168 591

With the reduction in gold production and the rapid expansion of the platinum industry at the beginning of the last decade (Platinum Today, 2003; MEG, 2007), many retrenched gold miners found employment in the platinum mines. The methods used in gold and platinum mining are similar – hard rock mining, requiring drilling and blasting – and experienced men were sought after. Employment has increased by 220% since 1980 (Table 7). In 2010, the South African platinum mining industry employed almost 170 000 workers (DMR, 2011b), comprising 35% of miners from all commodities. Gold and coal mine workers comprised 30% and 15%,

respectively, while the other mining commodities each comprised <5% of the workforce.

1.3.2 DIAMONDS MINE WORKERS AND ASBESTOS-RELATED DISEASES

Diamonds are formed from carbon over a very long period, under conditions of extreme heat (around 1 000 degrees) and very high pressures (above 30 kb). They can also be formed on impact and are thus sometimes found at meteorite sites. The rock formations in which diamonds may be found are known as kimberlite pipes which are pushed towards the surface of the earth by volcanic action. Most kimberlite is non-diamondiferous and only a few of those pipes that do contain diamonds are economically viable to mine. Alluvial diamond deposits result from weathering of the pipes and may wash into the sea from where they are 'mined'.

Processing of the kimberlite uses no toxic chemicals and produces no chemical pollutants (MiningTechnology.com, 2011) but the mining process generates large quantities of dust. Diamond mining is undertaken by a process of dry drilling, unlike underground mining operations such as gold and platinum mining, because water causes the kimberlite to decompose and the drill holes to close. During mining operations, large amounts of rock and materials are removed to expose the kimberlite and allow access to the diamonds. The ore is blasted and loaded into trucks, hauled to a crusher, reduced in size and conveyed to a primary stockpile. It is then conveyed to the treatment plant for processing where it is further crushed, washed and screened into different sizes. Each of these steps generates dust. Data on the mineral content of the dust, in terms of the concentrations of the different minerals and compounds are, however, not readily available.

As discussed below, there is strong evidence to suggest that diamond mine workers in South Africa are at risk of asbestos exposure, both in terms of mineralogical content of the kimberlite and geographical location of diamond and asbestos deposits in South Africa. In addition, asbestos has previously been identified in some diamond mines.

Mineralogical content of kimberlite

Diamonds are found in kimberlite which often contains fragments of ultramafic rock such as peridotite and eclogite. Ultramafic rocks have a low silica and high magnesium and iron content; these elements also form the basis of amphibole minerals. Eclogites are susceptible to metamorphism, by which they form both calcic amphibole-rock types, such as tremolite and actinolite; and glaucophane, a sodic amphibole with a chemical makeup similar to crocidolite (Leake et al., 1997). Both chrysotile and amphibole asbestos fibres (primarily tremolite) have been described in association with kimberlite, as early as 1914 (Wagner, 1914; Wagner and Reinecke, 1930; Aoki, 1972).

Not surprisingly, all reports on amphiboles in relation to kimberlite have been published in geology journals (Aoki, 1972; Dawson and Smith, 1977; Garanin et al., 1990). There are no reports of the association in the health literature, nor has any disease been described in diamond miners in the scientific literature.

Anecdotally, asbestos-related disease in the diamond, platinum and other mining sectors has been attributed to exposure outside the specific mining sector; it has also been reported in this way in some annual reports (Implats, 2006a).

Geographical location of diamond and asbestos deposits

An added risk of South African diamond mine workers being exposed to asbestos is the geographical location of the diamond mines. Diamond deposits are found in close proximity to both amphibole and chrysotile asbestos deposits in the Northern Cape province, the North West province, Limpopo province and Gauteng (see Figure 8).

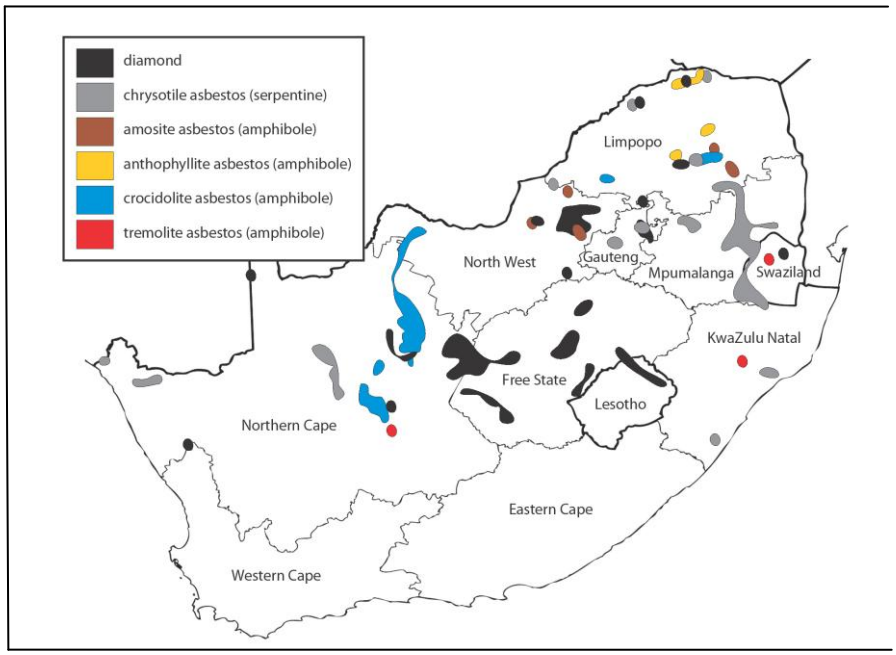


Figure 8. Map of South Africa showing geographical distribution of diamond and asbestos deposits [generated using ArcExplorer software (Geosciences, 2001)]

In addition to the asbestos deposits that were mined over several decades in South Africa, there are smaller non-commercially viable deposits of chrysotile, crocidolite, amosite, tremolite, actinolite and anthophyllite asbestos scattered around the country. These unexploited asbestos deposits are sometimes referred to as naturally occurring asbestos (NOA) in the literature.

There is some controversy in the literature with regard to this term, which was coined in the late 1990s (Gunter, 2009). As asbestos is a mineral, and all minerals are naturally occurring, preferable terminology may be "asbestos occurring in its natural setting" (as opposed to in mine tailings, road surfaces, etc.).

Nevertheless, NOA continues to be used to describe non-economic occurrences of asbestos that are not intentionally mined or used, but may cause disease if released into the environment through human activity or deflation (the term used for the detachment of mineral dust from the ground surface, through weathering and erosion (Derbyshire, 2007)), and is widely used in the scientific literature to differentiate natural sources of fibre from commercial or industrial sources (Hendrickx, 2009). NOA exposure differs from environmental exposure in that it is not related to any asbestos mining activities.

The issue of NOA has caught the attention of both scientists and activists (Orenstein and Schenker, 2000; Abraham, 2004; Berg, 2004; Cardile et al., 2007), and the US Environmental Protection Agency, in particular, has spent substantial resources developing appropriate measuring and risk assessment techniques (Troast, 2005).

In 2007, Derbyshire wrote:

> *" …there remains much to be learned about the physical, mineralogical, and geochemical characteristics of ambient mineral dust in the context of assessing the disease burden upon exposed human communities. …information on the disease burden arising from exposure to naturally occurring dusts in different populations around the world remains sparse… The pathological effects of prolonged exposure to natural mineral dust have been recognized in a general way since ancient times, but the number of modern studies of pneumoconiosis outside occupation-specific contexts remains small."*
>
> (Derbyshire, 2007)

Although the presence of NOA does not suggest that airborne fibre concentrations are substantially above background levels (Brodkin et al., 2006), aging, erosion and natural weathering of NOA may contribute to increased levels of asbestos in the environment (Orenstein and Schenker, 2000).

Exposure to NOA has been documented as the cause of disease since the late 1970s, primarily in individuals who used amphibole-containing soil as an additive to whitewash used to coat the walls of houses (Constantopoulos, 2008). Affected populations comprise villagers from Mediterranean countries, including Turkey (Boman et al., 1982; Metintas et al., 2005; Osman et al., 2007), Greece (Constantopoulos et al., 1991), Corsica (Rey et al., 1994), Italy (Bernardini et al., 2003), as well as New Caledonia in the South Pacific (Luce et al., 2000), where amphibole asbestos has been implicated as a significant health risk, likely due to its high ecopersistance (Favero-Longo et al., 2009). The 50 mesothelioma cases from the south of Turkey, studied by Zeren et al., are also likely to have been exposed to contaminated whitewash, although the authors do not specifically state this (Zeren et al., 2000). In all these populations, tremolite, sometimes together with actinolite, was present in the soil. In 2003, Luo et al. reported an

increase in pleural plaques and mesothelioma from exposure to crocidolite from a similar source in China (Luo et al., 2003). There have also been reports of amphibole exposure at construction sites where local materials, containing tremolite and/or actinolite have been used (Lee et al., 2008; Perkins et al., 2008).

There are few studies on the health effects of naturally occurring asbestos in settings where it is not used by local residents for domestic purposes (Orenstein and Schenker, 2000) but interest in this area has increased over the last few years (Pan et al., 2005; Cardile et al., 2007). The first such study was in Bulgaria in 1970 and showed an increase in the prevalence of pleural plaques in tobacco farmers in areas where the soil contained amphibole fibres (Burilkov and Michailova, 1970). Rey et al. attributed the high prevalence of pleural plaques to the surface deposits of tremolite asbestos in Murato in Corsica (41% of the inhabitants had pleural plaques compared to 7.5% in a control village where tremolite concentrations were 100 times lower) (Rey et al., 1994). In 2003, Bernadini et al. described mesothelioma in three Italian shepherds and identified naturally-occurring tremolite as the probable cause (Bernardini et al., 2003).

Exposure to naturally occurring amphibole asbestos fibres is thought to be a cause of malignant mesothelioma in several other areas of the world, including the USA(Pan et al., 2005) and Australia (Hendrickx, 2009). In 2003 Pan et al. published the results of a case control study on the association of residential distance from NOA with the incidence of mesothelioma in California (Pan et al., 2005). They calculated that the odds of having mesothelioma fell by 6.3% for every 10km further that a person lived from a NOA source, although other researchers have

pointed out the methodological limitations of the study, suggesting that the findings should be interpreted with caution (Brodkin et al., 2006; Kelsh et al., 2006).

Exposure to NOA when it is disturbed must be recognized as a health risk, no matter how small the size of the deposits. In a 1999 report on a review of the literature, Hillerdal concluded that there is no evidence of a threshold level below which there is no risk of mesothelioma (Hillerdal, 1999).

Evidence of asbestos in diamond mines

There have been two reports of asbestos in association with diamond mines in South Africa. The first was a report by the Environmental Control Department of the DMR, published in 1995. The report, cited by Unsted and Jansen van Vuuren in a 2001 MHSC report (Unsted and Jansen van Vuuren, 2001) showed that airborne fibres could be encountered in the workings of diamond mines. Unfortunately, the original DMR report is unobtainable and could thus not be reviewed for details about the fibres, either qualitatively or quantitatively. The second was the report by Unsted and Jansen van Vuuren, which described chrysotile, anthophyllite and tremolite-actinolite fibres, identified using scanning electron microscopy (SEM), in air samples in 11 different working environments of five diamond mines (Unsted and Jansen van Vuuren, 2001). Several types of asbestiform fibres were found in all the mines sampled, in both country rock and kimberlite excavations.

Crocidolite asbestos has been identified, by geologists, around the perimeter of the kimberlite pipe of Finsch diamond mine in the Asbestos Hills near the town of Lime Acres in the Northern Cape (PJ Jordaan, Finsch Diamond Mine, unpublished data). In 2009, a sample of chrysotile asbestos was sent to the NIOH electron

microscopy laboratory for analysis from Venetia diamond mine in Limpopo province (Figure 9).



Figure 9. Photograph of a chrysotile asbestos sample from a diamond mine [Venetia mine, Limpopo province] (Dr Jim Phillips, Head: Electron Microscopy Unit, Pathology Division, NIOH)

1.3.3 PLATINUM MINE WORKERS AND SILICOSIS

The PGMs, which include platinum, palladium, rhodium, iridium, ruthenium and osmium, are found in the Bushveld Complex in the north eastern part of South Africa (Figure 10), together with a range of other minerals. South Africa has around 75% and 50% of the world's platinum and palladium resources, respectively (Cawthorn, 1999). The Bushveld Complex, a layered igneous intrusion, is probably the most important of the South African mineral-bearing geological formations in terms of mineral content. It contains some of the richest ore deposits on earth and is one of the most extensively researched geological formations in South Africa.

As a volcanic intrusion through the earth's crust, it extends for 400 km into three of the nine provinces of South Africa, viz. the North West Province, Limpopo Province and Mpumalanga, falling roughly between the Pilansberg volcano in the west, Lydenburg in the east, Pretoria in the south, and Polokwane in the north. It covers an area of 65 000 km^2, making it the largest layered intrusion in the world, and reaches a depth of 7 km in some places (Cawthorn, 1999). Over billions of years, the intrusion tilted and eroded, with resulting outcrops of PGMs spread around the edge of a vast basin.

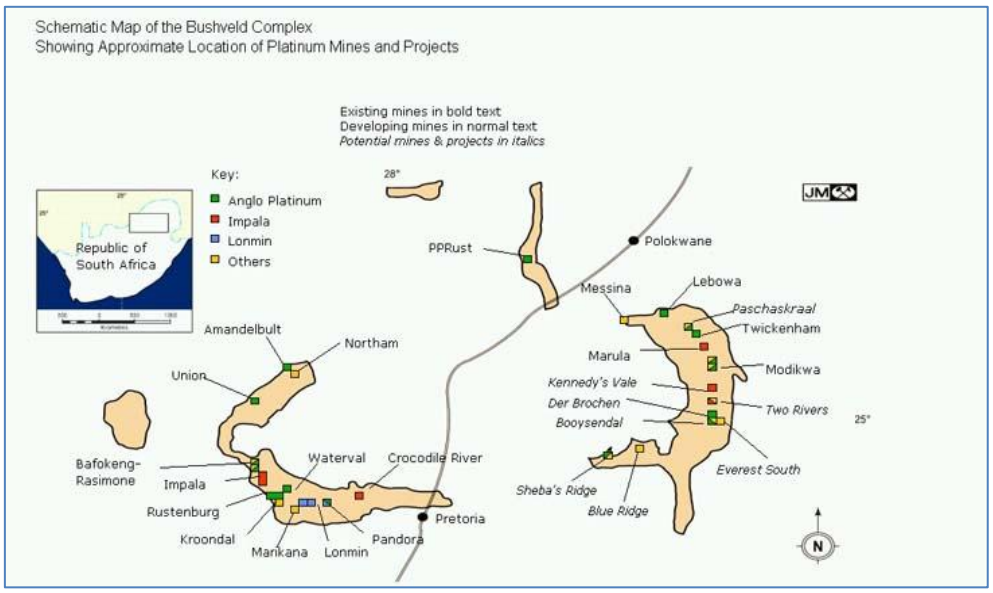


Figure 10. Map showing the platinum mines in the Bushveld Complex (Platinum Today, undated)

Figures 11 and 12 show the extensive size of the platinum mines and the tailings dumps.



Figure 11. Photograph of a platinum mine near Rustenburg [Aquarius platinum mine] (G Nelson)



Figure 12. Photograph of platinum tailings with a dump truck on top of a tailings dump [Aquarius platinum mine] (G Nelson)

Similarly to the diamond mining industry, health research in the platinum mining industry has been neglected. The mineral diversity of the Bushveld Complex and the extensive mining of these minerals greatly increase the potential for exposure of mine workers to some or all of the associated minerals. The methods used to mine platinum are the same to those used in gold mining. Hard rock mining methods are used whereby the rock surrounding the platinum is blasted and drilled (Figure 13). Both procedures produce dust which can be inhaled by mine workers.



Figure 13. Photograph of a mine worker drilling for platinum in a narrow, confined space [Impala platinum mine] (G Nelson)

Despite this, very little is known about the respiratory health of platinum mine workers. While it has been shown that *platinum refinery workers* have an increased risk of developing platinum salt sensitivity (Calverley et al., 1995; Taylor, 2001), associated with respiratory symptoms of asthma, rhinitis and urticaria, and

dermatitis, there is no research on disease in platinum miners published in the scientific literature.

Silicosis is perhaps the most researched occupational respiratory disease in South Africa but research in the mining industry has been limited to gold and coal mine workers. As in the case of asbestos-related diseases in diamond mine workers, silicosis in platinum miners has been attributed to prior exposure to silica in the gold mining industry. However, there is evidence to suggest that miners who have worked exclusively in the platinum mining sector are at risk of developing silicosis.

Mineralogy of the Bushveld Complex

Many minerals and compounds are associated with the PGMs. The three most abundant are pyrrhotite, chalcopyrite and pentlandite but exposure to these is associated with no ill health effects (Girdler-Brown et al., 2006). Chromite, gold, copper and nickel (Implats, 2006b), as well as smaller amounts of sulphur, arsenic, selenium, tellurium, iron, tin, cobalt, zinc, titaniferous magnetite and vanadium pentoxide also occur (Girdler-Brown et al., 2006) some of which cause adverse respiratory health effects. Interstitial fibrosis may result from exposure to copper or iron (Churg and Colby, 1998), pneumoconiosis from cobalt (Nordberg, 1994), stannosis from tin (Sluis-Cremer et al., 1989; Churg and Colby, 1998), adult respiratory distress syndrome from zinc or sulphur (Wright and Churg, 1998), and lung cancer from arsenic or nickel (Churg and Colby, 1998).

As the Bushveld Complex is an igneous intrusion, it also contains crystalline silica. The average crystalline silica level in igneous rocks is about 12% (Lujan and Ary, 1992) but the variation is wide. Miners of any of the minerals in the Bushveld

Complex are therefore potentially at risk of exposure to silica dust and may develop silicosis.

Silica dust in platinum mines

There are very few available silica dust measurements from the platinum mines as the silica content of the dust is not routinely measured (Girdler-Brown et al., 2006). However, two studies were commissioned by the MHSC in the early 2000s to measure silica dust levels in platinum mines; both are described in detail in Paper 4. In summary, in 2003, Biffi and Belle reported the crystalline silica content of crushed stope rock samples from two platinum mines to be 0.45%, compared to 9.9% and 39.1% in two gold mines (Biffi and Belle, 2003). The silica content of respirable dust samples was < 0.2%, compared to 4.5 to 57% in the gold mines. In 2007, Dekker *et al*., measured respirable silica dust concentrations, ranging from 0.018 mg/m^3 to 0.035 mg/m^3, much lower than the South African OEL of 0.1 mg/m^3 (Dekker et al., 2007), but higher than the American Conference of Governmental Industrial Hygienists (ACGIH) limit of 0.025 mg/m^3 .

However, little research has been done on health outcomes in the platinum mines. The only study on silicosis in mine workers was published as a Mine Health and Safety Council report in 2006 (Girdler-Brown et al., 2006). The authors conducted a cross-sectional survey on 969 platinum miners; silicosis was diagnosed in three, all of whom had a history of gold mining.

1.4 AIMS AND OBJECTIVES

The aim of this thesis was to, first, provide the only trend analysis of silicosis in gold miners over a 33-year period against which mine occupational hygiene practices may be assessed, and policy intervention programmes be evaluated; and, second, to explore the potential for the development of compensable respiratory disease in the diamond and platinum mining sectors.

Objectives

The specific objectives were:

1. To describe trends in silicosis at autopsy in South African gold miners from 1975 to 2007, and to quantify the contributions of age at autopsy and employment duration to these trends in black and white gold miners separately.
2. To discuss the contribution of the migrant labour system to the burden of silicosis in the gold mining industry.
3. To explore the possibility of asbestos exposure during the process of diamond mining by reviewing cases of asbestos-related respiratory disease diagnosed at autopsy in diamond miners from 1975 to 2008.
4. To explore the possibility of developing silicosis during the process of platinum mining by reviewing cases of silicosis diagnosed at autopsy in platinum miners from 1975 to 2009.

SECTION 2

METHODOLOGY

In this section, the **SOURCES OF DATA** are described in detail. The **primary data source**, the PATHAUT autopsy database, is discussed against the background of South African legislation regarding the examination of cardio-respiratory organs for compensable disease in the mining industry. The **supplementary data sources** are described in less detail. The subsequent sections of the Methodology address the choice of **STUDY DESIGN** and the **SELECTION OF THE STUDY SUBJECTS** for each aspect of the thesis, the **STATISTICAL METHODS** applied, and the **ETHICAL CONSIDERATIONS** of the thesis. More details of the methods can be found in the four thesis papers.

2.1 DATA SOURCES

2.1.1 PRIMARY DATA SOURCE

The electronic PATHAUT database was the primary and common data source for the analyses of disease in all three mining sectors (gold, diamond and platinum). This database was used to identify miners and ex-miners for inclusion in the analyses. All relevant data for each study subject, i.e. demographic data, exposure information and pathology findings were then copied from the PATHAUT database into a STATA IC 10 database for analysis, including all pathological information about the disease(s) of interest, and all demographic and exposure data.

The PATHAUT database was developed as a direct consequence of legislation regarding autopsy examination of miners and ex-miners for compensable respiratory disease, as described below.

South African legislation regarding the examination of cardio-respiratory organs for compensable disease in the mining industry

Compensation for respiratory disease contracted as a result of work dates back to 1911 (RSA, 1911) and, as early as 1916, legislation required that deceased miners' lungs be examined for disease.

Section 27 of the Miners' Phthisis Act 44 of 1916 reads as follows:

> *"It shall be the duty of every medical practitioner who carries out any post mortem examination at a Government mortuary, if he finds on such examination silicosis to be present in the lungs of the deceased, to send such lungs to the medical bureau, accompanied by his report thereon."*

This early legislation created a high level of awareness of occupational diseases among miners and their families who began submitting organs for pathological examination soon after the Act was promulgated (Hessel et al., 1987a). Over the following three decades, the Act was broadened, amended and replaced several times (Table 8) but the requirement for autopsy examination remained.

Table 8. Acts addressing the compensation of occupational respiratory disease in miners

YEAR	TITLE OF ACT
1911	Miners' Phthisis Allowances Act (No. 34 of 1911)
1912	Miners' Phthisis Act (No. 19 of 1912)
1916	Miners' Phthisis Act (No. 44 of 1916)
1925	Miners' Phthisis Acts Consolidation Act (No. 35 of 1925)
1946	Silicosis Act (No. 47 of 1946)
1956	Pneumoconiosis Act (No. 57 of 1956)
1962	Pneumoconiosis Compensation Act (No. 64 of 1962)
1973	Occupational Diseases in Mines and Works Act (No. 78 of 1973)
1994	Occupational Diseases in Mines and Works Amendment Act (No. 208 of 1993)

Although compensation was available to all racial groups from the implementation of the autopsy examinations, the Pneumoconiosis Act of 1956 introduced racial discrimination in terms of benefits. Differentiation between persons on the grounds of gender or population group ceased with the 1994 amendment to the current Act, the 1973 Occupational Diseases in Mines and Works (ODMW) Act (RSA, 1973).

The ODMW Act governs the lifelong monitoring and surveillance of both former miners and active miners for possible compensable occupational lung disease. It specifies that any attending physician who knows or suspects that a person was a miner is obliged to remove and submit the cardio-respiratory organs to the NIOH for examination for the presence of occupational disease by expert pathologists, providing the next of kin grants permission for this examination, and regardless of the cause of death (Goldstein and Webster, 1976).

Section 34 of the Occupational Diseases in Mines and Works Act of 1973 describes the post-mortem examination duties of a medical practitioner as follows:

1) The director may authorize or in writing direct any medical practitioner in the Republic to perform a post mortem examination or other post mortem service under this Act of a nature determined by the director, and a medical practitioner so authorized or directed …, shall forthwith submit to the director a detailed report on the result of the examination or service performed by him.

2) A medical practitioner in the Republic who attended a deceased person at the time of or immediately before his death, or has opened the body of a deceased person, and who knows or has reason to believe that the person worked at a mine or works, shall remove the cardiorespiratory organs and any other prescribed organs or parts of the body of the deceased and shall send such organs and parts of the body to the prescribed place or, if no place has been prescribed, to the bureau or to any other place specified by the director…

3) … a medical practitioner shall not perform a post mortem examination on any deceased person …, without the consent of his widow (if any) or an adult near relative of the deceased, if the widow or such relative can readily be consulted."

There have been several amendments to the ODMW Act, the last being in 2002, but section 34 has remained unchanged.

Compensable respiratory diseases

The following respiratory diseases are compensable under the ODMW Act:

- Pneumoconiosis (silicosis, asbestosis, coal workers pneumoconiosis and mixed dust fibrosis)
- Cardio-respiratory tuberculosis contracted during service or within 12 months of last risk shift
- Combined pneumoconiosis and pulmonary tuberculosis
- Permanent obstructive airways disease

- Progressive systemic sclerosis
- Asbestos-related lung diseases, including lung cancer and mesothelioma
- Platinum salt sensitivity (platinosis)

Before any of these diseases is considered for compensation, there must be documentation of occupational exposure in the form of employment records. Particular exposure requirements must be met for each disease and the disease must be of a particular severity.

The NIOH autopsy service

The post-mortem examinations specified by the ODMW Act are performed by highly skilled pathologists at a single designated centre, the National Institute for Occupational Health (NIOH) in Johannesburg, which is now part of the National Health Laboratory Service. The NIOH pathology laboratory is accredited by the South African National Accreditation System (SANAS) which is the single National Accreditation Body that gives formal recognition that laboratories and other facilities are competent to carry out specific tasks.

Examination of the cardio-respiratory organs

The cardio-respiratory organs are removed from the body, placed in formalin in specially provided buckets, and sent to the NIOH. In some cases, the organs are removed at the NIOH. Pathological examinations include macro- and microscopic examination of the lungs, heart, bronchi and bronchioles, regional glands, pulmonary vessel, and pleura. The Pneumoconiosis Act of 1956 (RSA, 1956) required a standardised method for the post mortem examination of the cardio-respiratory organs, which has been adhered to ever since.

Documentation of the pathological findings

All macro- and microscopic pathology findings are recorded by the attending pathologist in a standardised document which is in the form of a structured, numerically-coded booklet (see Appendix 5). Personal details and summarised information regarding employment in the mining industry are also recorded in the booklets which are archived at the NIOH (Figure 14).



Figure 14. Photograph of shelves holding the PATHAUT booklets in a filing room at the NIOH (J Mthombeni)

The PATHAUT database

Development of the electronic database

Initially, an edge-punched card system was developed for the systematic recording of the pathology findings. Approximately 40 000 autopsy examinations were recorded in this way until 1975 (Hessel et al., 1987a) when the system was replaced by the computerised PATHAUT system (Soskolne et al., 1976). The PATHAUT system and its development from the mid-1970s to the late 1980s have been described in detail elsewhere (Soskolne et al., 1976; Hessel et al., 1987a; Hessel et al., 1987b). The system underwent one major upgrade in 1996 (Murray et

al., 1998) and data recorded prior to this were incorporated into the upgraded system.

Structure of the database

The PATHAUT database comprises data from the autopsy records (booklets), demographic information (age, dates of birth and death, etc.), and occupational histories, including the commodities mined and the length of time spent in each mining sector. The data are stored in SAS (Statistical Analysis Software) format, and the database contains 292 variables.

2.1.2 SUPPLEMENTARY DATA SOURCES

Although the PATHAUT database was the primary (and only) source of data for the analysis of silicosis trends in gold miners, several other databases were identified and reviewed, in the course of this study, to supplement the PATHAUT data for the analysis of diamond and platinum mine workers with asbestos-related diseases and silicosis, respectively. The supplementary data sources used for each analysis are described below.

The Medical Bureau for Occupational Diseases (MBOD) paper-based files

More comprehensive mining employment histories are recorded in medical files kept at the Medical Bureau for Occupational Diseases (MBOD) in Johannesburg, originally established as the Miners 'Phthisis Board in 1912. Records are kept for all miners who apply for compensation for an occupational respiratory disease. These records can be linked to the PATHAUT data by a unique MBOD number that is recorded on the pathology booklet. The files contain paper-based information about specific occupations, employers and shifts worked, as well as results of the

routine annual medical examinations conducted during employment and special examinations conducted for compensation during life, and smoking information (Figure 15).

Until 1994, when the amendments to the ODMW Act were promulgated, examinations of white miners were conducted at the MBOD itself in central Johannesburg or at one of the sub-bureaus located in the vicinity of the gold mines. All records were kept at the MBOD. Black miners, on the other hand, were examined on the mines but their data were not routinely submitted to the MBOD. Nowadays, all examinations are conducted on the mines.



Figure 15. Photograph of MBOD files (J Mthombeni)

A detailed report of each case examined at the NIOH is sent to the MBOD. The MBOD keeps a database of all submissions for compensation, be they during life or after death. When a case is submitted for certification of a compensable disease, the MBOD files are reviewed together with autopsy findings. Certified cases are then referred to the Compensation Commissioner for Occupational Disease (CCOD), where payment of compensation is managed (Figure 16).

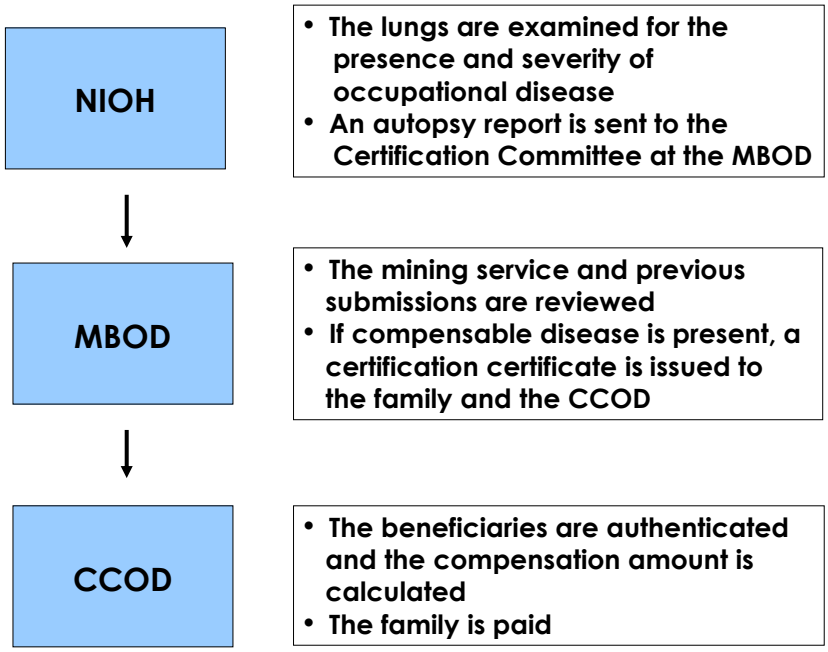


Figure 16. The flow of information from, and responsibilities of, each agency involved in the ascertainment and verification of compensable disease

Thirty-five MBOD files of diamond miners with asbestos-related disease and 90 files of platinum miners with silicosis were reviewed, specifically for employment history details such as dates and durations worked, commodities, names of companies and specific occupations (Figure 17). This was done to identify mine workers who might have worked in a mining sector other than the one of interest, thus excluding them from the respective studies as they would not be classified as 'exclusive' diamond or platinum mine workers. In addition, evidence of potential exposure to asbestos in the case of the diamond mine workers with asbestos-related diseases, and employment in the gold mines in the case of the platinum miners with silicosis was important to exclude the possibility of them having contracted the disease of interest in an industry or mining sector other than the diamond or platinum mines, respectively.

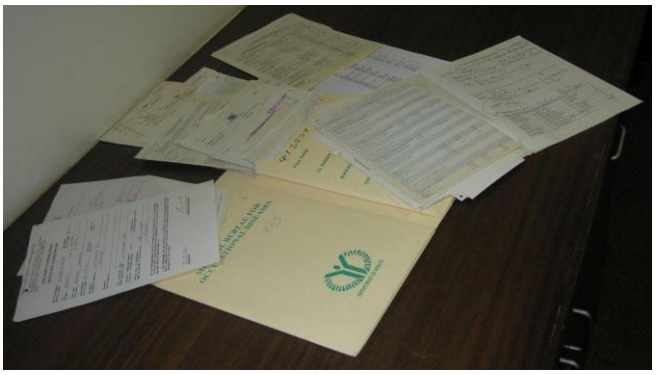


Figure 17. Photograph of the MBOD file of a deceased miner (J Mthombeni)

The MBOD files were not reviewed for the analysis of trends of silicosis in gold miners as more than 19 000 cases were included in this analysis. The main inclusion criterion for the study was a record of work in the gold mining industry, regardless of employment elsewhere, so cases were not excluded if they had mined a different commodity.

The Mine Workers' Compensation (MWC) system database

Until 2004, when the electronic MWC system was implemented, the MBOD was entirely paper-driven. Since then, all new applications to the MBOD have an electronic record in addition to a paper file. The MWC system consolidates and summarises paper-based information on work histories from the autopsy database, the MBOD files and other sources, for applications for occupational disease certification. The MWC employment record states the commodity, the date on which the individual started working at a particular mine and the date on which his or her employment was terminated, for whatever reason. The reason for termination of employment is, however, not recorded.

The MWC reports were reviewed to corroborate work history information obtained from other sources for diamond miners with asbestos-related disease and platinum mine workers with silicosis.

The Employment Bureau of Africa (TEBA)

TEBA Limited (www.teba.co.za) was established in 1902 to assist the growing gold mining industry to source and engage mine workers. TEBA stations were established in Malawi, Angola, Mozambique, Lesotho, Swaziland, Botswana and South Africa. TEBA still deals primarily, but not exclusively, with gold miners. On successful placement within a mining company, a man is assigned a unique mining industry number that remains his personal number for life, even if he is subsequently employed in another mining sector. The industry number is recorded on the PATHAUT database, allowing linkage of records.

Where available, the electronic TEBA service history records were reviewed for the diamond miners with asbestos-related diseases to confirm that they had not been employed in the asbestos or any other mining sector. Similarly, the records of platinum mine workers with silicosis were reviewed to confirm that they had not been employed in the gold or other mining sector.

Selected mines

Mining companies archive personnel data albeit for a limited period and very often in paper-based files. Where available, De Beers Consolidated mines' human resources records were reviewed for specific job descriptions of diamond mine workers to identify those who might have been employed in occupations that

utilised asbestos or asbestos-containing products (boiler maker, fitters and turner, etc.).

In the case of platinum mine workers, names and relevant unique identifying numbers were sent to Rustenburg Platinum Mining Company, from which most platinum miners' cardio-respiratory organs are received. The list was linked to their human resources database for identification of employees to determine if there were gaps in their employment histories during which time they might have worked in the gold mining sector.

For the study on the analysis of diamond miners with asbestos-related diseases, tailings and soil samples were collected from three different mines, with permission from De Beers Consolidated and the individual mines.

Relatives and friends

The telephone number(s) of the person who reported the death of a miner, as stated on the death notification form, is sometimes recorded in the MBOD file. The numbers were transcribed for platinum miners with silicosis. Where possible, that person was then contacted and asked if the deceased had ever worked in the gold mining industry.

2.2 STUDY DESIGNS

A number of different study designs were used in this thesis. The study on silicosis in gold miners was a time trend analysis over a 33-year period. Health outcomes in a population can only be fully understood if their frequency and distribution are examined in terms of person, place, and time (Rosenberg, 1997), and trend

analysis is one arm of this analytic triangle. It can be used for, amongst other things, public health surveillance and monitoring and programme evaluation, as for this study. Studies of time trends focus on the overall pattern of change over time, compare one population to another, compare one time period to another time period, or compare one geographic area to another (Rosenberg, 1997). The time trend analysis of silicosis in gold miners covered the first two topics comprehensively.

The paper on oscillating migration was a literature review and discussed the mine migrant labour system as a contributor to high rates of disease, in both historical and current contexts.

The studies on diamond miners with asbestos-related diseases and platinum mine workers with silicosis were both case series. The justification for the use of this study design is discussed in more detail in Section 4.1.

2.3 SELECTION OF STUDY SUBJECTS

The available data from the PATHAUT database were extracted for the relevant years for each analysis. The analysis of silicosis trends in gold miners encompassed the years 1975 to 2007 as this was the first of the three analyses, at which time the data were available only until 2007. Data for the analysis of the diamond mine workers with asbestos-related disease were available until 2008 as this analysis was performed subsequent to the silicosis trends analysis. The final analysis, viz. that on platinum mine workers with silicosis, was performed in 2010, at which time data were available until 2009.

All gold miners were included in the first analysis, regardless of whether they had worked in another mining sector or not. Diamond and platinum mine workers, on the other hand, were included in the respective analyses, only if they had not worked in another mining sector. The additional inclusion criteria for each analysis are described in detail in the three papers that address disease in gold, diamond and platinum mine workers.

2.4 STATISTICAL ANALYSES

The relevant data for each aspect of the thesis were extracted from the SAS PATHAUT database and transferred into a Stata IC 10 database. In each case, a repeat validation of the data was performed for the variables of interest, and the Stata dataset was amended with any additional data (e.g. employment histories) identified from the other sources. All statistical analyses were performed using Stata IC 10. All variables for all the cases included in a particular analysis were extracted and imported into Stata where they were analysed. The details of the statistical methods are described in each paper.

Stratified trend analyses were conducted for the study on silicosis in gold miners, using both simple linear regression and binary logistic regression models. The three explanatory variables were age, duration of employment and year of autopsy. Direct age and duration of employment standardization was used to compare proportions of black and white miners with silicosis.

No statistical analyses were performed on the diamond miners with asbestos-related diseases or the platinum mine workers with silicosis as these were both case series of diseased men.

2.5 ETHICAL CONSIDERATIONS

Consent for autopsy examination can be granted by the next-of-kin in terms of the Occupational Diseases in Mines and Works Act (ODMWA) of 1973 (RSA, 1973) and the Occupational Diseases in Mines and Works Amendment Act of 2002 (Act no. 60 of 2002). This PhD protocol was approved by the University of the Witwatersrand Ethics Committee (Ethics clearance number M050228 – Appendix 6). Permission was obtained from the mining companies for the collection of samples of tailings.

SECTION 3

RESULTS

The outcomes of the different aspects of the thesis are comprehensively described in each of the four papers but the major findings are highlighted in this section in the following order: **SILICOSIS TRENDS IN GOLD MINERS**, **OSCILLATING MIGRATION**, **ASBESTOS-RELATED DISEASES IN DIAMOND MINE WORKERS**, and **SILICOSIS IN PLATINUM MINE WORKERS**.

3.1 SILICOSIS TRENDS IN GOLD MINERS

The silicosis trend analysis was performed on 19 143 gold miners who died from external causes and worked for more than one year in the gold mining industry, 16 411 of whom were black and 2 732 white.

The difference in the changing proportions of black and gold miners with silicosis at autopsy was striking. From 1975 to 2007, the crude proportion of black miners with silicosis increased ten-fold from 3% to 32%; the increase in white men was slight, from 18% to 22% (Figure 18). More white miners had silicosis in 1975 (the proportion in white miners was six times higher than that in black miners) but, by 2007, this pattern was reversed (the proportion was 1.5 times higher in black miners). Overall, the proportion of black miners with silicosis was almost double that of white miners for the 33-year period.

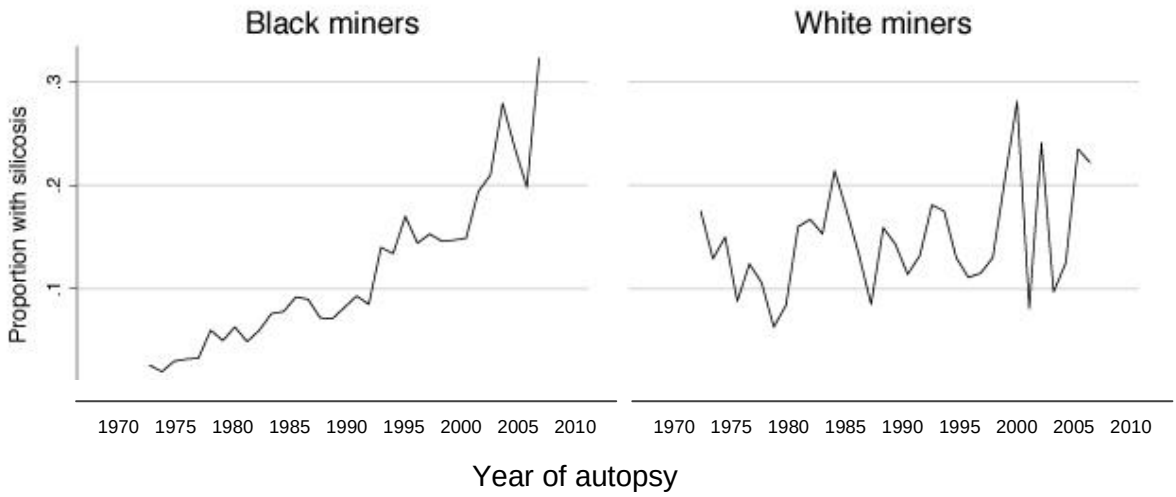


Figure 18. Crude population group-specific proportions of gold miners with silicosis, 1975 - 2007

Increasing age and duration of exposure explained much of the increase in silicosis. The mean age at death of both the black and white miners increased by more than 10 years over the study period, although the black miners were, on average, 11 years younger than the white miners at the beginning of the study period (33.0 and 44.1 years, respectively). The mean duration of employment increased by less than three years for the white miners (from 17.5 to 20.1 years), but by almost eight years, from 5.6 to 13.4 years for the black miners.

The standardized proportion ratio (SPR) was 1.70 (95% CI 1.45-1.99), showing that the proportion of black miners with silicosis was 70% higher than that for white miners. When the analysis was repeated for miners dying from natural causes, the SPR increased to 2.27 (95% CI 2.12-2.43), most likely due to the association of TB with silicosis.

The overall age- and employment duration- adjusted proportion of black miners with silicosis was higher than that of white miners, for all years of autopsy other

than 1975-1979, suggesting that a factor or factors other than increasing age and duration of employment was associated with the increase in disease. It is likely that black miners had higher intensities of exposure than white miners due to the dustier jobs in which they were (and still are) employed. This is supported by the fact that more black than white miners developed silicosis after relatively short periods of employment and at younger ages. The proportion of black miners with silicosis reached 2% after fewer years than it did for white miners, and the proportion of black miners with silicosis below the age of 50 years was more than double that of white miners.

3.2 OSCILLATING MIGRATION

This has been discussed in the background of the thesis (Section 1.2.4). In summary, oscillating migration is one of the reasons for the high silicosis rates in the gold mining industry. Miners from the rural areas of South Africa and neighbouring countries travelled long distances to find work on the mines, and still do. The migrant labour system reduced the ascertainment of compensable disease and externalised the costs of occupational disease to remote labour sending areas (Trapido et al., 1998b). If labour had been recruited from local communities, then these same communities would have large numbers of miners and ex-miners with silicosis which would heighten awareness of the extent of the disease. Ill workers would also be able to access the occupational mine health services. This has not happened because of oscillating migration.

Consequently, oscillating migration has both diluted community and organised-labour pressures on mine management to reduce dust levels and control disease,

and enabled mining companies to limit disease-associated costs, thereby reducing the financial incentive to control dust and disease.

3.3 ASBESTOS-RELATED DISEASES IN DIAMOND MINE WORKERS

The evidence for the risk of asbestos-related diseases in diamond miners is provided by the mineralogy of kimberlite, the diagnosis of asbestos-related disease in diamond mine workers, and the identification of asbestos fibres in both the lungs of diamond mine workers and diamond mine tailings.

Of the 559 exclusive diamond miners on the PATHAUT database from 1975 to 2008 who started working when they were younger than 26 years of age, 24 (4.3%) had one or more asbestos-related diseases at autopsy. After a comprehensive review of all data sources to ascertain possible exposure to asbestos outside of the diamond mining industry, six diamond miners with one or more asbestos-related diseases were identified. Four had asbestosis, one had pleural plaques, and one had malignant mesothelioma and pleural plaques.

The lungs of 11 other diamond mine workers were analysed under SEM. Tremolite asbestos fibres or fibres in the tremolite-actinolite series were identified in five of the 11. No chrysotile asbestos fibres were identified, although four of the five mine workers had worked in a diamond mine close to a chrysotile asbestos deposit. None had an asbestos-related disease.

Tailings samples from a diamond mine near a crocidolite asbestos deposit contained tremolite-actinolite series fibres. The tailings samples from the diamond mine near a chrysotile deposit contained both chrysotile fibres and tremolite-

actinolite series fibres. Tailings samples and dust collected from a crusher located above ground from a diamond mine near anthophyllite and chrysotile deposits contained tremolite-actinolite series fibres (Figure 19). A sample of pure chrysotile asbestos was also collected from between the kimberlite pipe and the country rock in this mine.



Figure 19. Photograph of dust on the surface of equipment near a diamond mine crushing plant: Venetia mine, Limpopo province. Amphibole asbestos fibres were identified in this dust by electron microscopy analysis (G Nelson).

3.4 SILICOSIS IN PLATINUM MINE WORKERS

A total of 6 490 platinum miners with no record of having worked in another mining sector were identified from the PATHAUT database. Eighty-five (2.2%) had been employed for more than one year and had silicosis at autopsy.

Fibrotic nodules were diagnosed in the lymph nodes of 490 (12.7%). After reviewing all available data sources, five platinum mine workers with pulmonary silicosis (four of whom also had fibrotic nodules in the lymph nodes), plus an additional 25 with fibrotic nodules in the lymph nodes but without silicosis were

identified. There was enough evidence to suggest that these pathological changes occurred in the course of the miners' employment on the platinum mines.

The diagnosis of silicosis and fibrotic nodules in the lymph nodes of platinum miners, as well as silica dust measurements in some of the mines point to a risk of silicosis in platinum mine workers.

SECTION 4

DISCUSSION

This section starts with a discussion of select **METHODOLOGICAL ISSUES** pertaining to the various analyses. **OBSTACLES TO OCCUPATIONAL RESPIRATORY HEALTH RESEARCH IN SOUTH AFRICA** are then addressed, including the **absence of occupational respiratory health surveillance databases**, the **inadequate documentation of work histories,** and the **lack of dust measurements**. The strengths and limitations of the **PATHAUT DATABASE** are described, followed by **policy**, **operational** and **research RECOMMENDATIONS** emanating from this thesis, and an overall **CONCLUSION**.

4.1 METHODOLOGICAL ISSUES

The methodologies employed in each of the analyses are described in detail in the papers in the appendices. In this section, select methodological issues are discussed, viz. the choice of study populations for the silicosis trend analysis and the analysis of the potential for silica exposure in platinum miners, the choice of a case series study design for the analyses of the risks of disease in diamond and platinum miners, and the stratified analysis of gold miners with silicosis.

Choice of study populations

Trends in silicosis in gold miners

Only gold miners who died from external causes were included in the trend analysis to prevent potential bias in the results. TB is a common cause of death in gold miners in South Africa and, because of its association with silicosis, miners dying from TB (natural causes) are more likely to have silicosis than those without.

This is clearly illustrated in Table 9 which shows that the proportions of miners with silicosis who died from natural causes from 1975 to 2007 were much higher than the proportions of those who died from external causes (almost three times higher in black miners and twice as high in white miners).

Table 9. Respiratory disease diagnosed in black and white gold miners who died from external and natural causes, 1975 - 2007

Disease diagnosed at death	Cause of death							
	External				Natural			
	Black miners		White miners		Black miners		White miners	
	N = 16 411		N = 2 732		N = 18 276		N = 19 842	
	n	(%)	n	(%)	n	(%)	n	(%)
Silicosis	1 304	7.9	391	14.3	3 818	20.9	5 365	27.0
TB and silicosis	113	0.7	12	0.4	1 148	6.3	249	1.3
Active PTB	482	2.9	43	1.6	3 922	21.5	500	2.5
COPD	1 175	7.2	781	28.6	2 614	14.3	11 634	58.6

Note: all miners had > 1 year duration of employment

Nonetheless, the results of an independent analysis of all black miners who died from either natural or external causes did not change the results of the trend analysis. The proportion of silicosis in all black miners increased from 3% to 34% over the study period, compared to the increase from 3% to 32% in those who died from external causes only.

Potential for silica exposure in platinum mine workers

The evidence for silica exposure in platinum miners was not restricted to those with pulmonary silicosis. The occurrence of fibrotic nodules in the hilar lymph nodes of silica-exposed individuals is well described (SSDC, 1988; Gibbs and Wagner, 1998). Mine workers with fibrotic nodules in the hilar lymph nodes were therefore also included in the case series. There is evidence that fibrosis in the lymph glands

precedes the development of overt pulmonary silicosis (Seaton and Cherrie, 1998). Baldwin and Lambert described five workers who exposed to silica who initially presented with bilateral hilar lymphadenopathy and no radiographic evidence of interstitial lung disease; one case progressed to silicosis (Baldwin et al., 1996).

Fibrosis of the lymph glands may be a response to low level silica dust as suggested by Murray et al. in 1991. They showed that a higher proportion of gold miners had fibrosed glands at autopsy than pulmonary silicosis after relatively short periods of employment (Murray et al., 1991). This was also the finding in a more recent study in which silica exposure was lower for uranium miners with lymph node fibrosis only than for those with both lymph node fibrosis and parenchymal silicosis (Cox-Ganser et al., 2009). The association of lymph node fibrosis with parenchymal silicosis remained after adjustment for silica exposure.

For these reasons, platinum mine workers with fibrotic nodules in the hilar lymph nodes, identified from the PATHAUT database, were included in the analysis, together with those who had pulmonary silicosis.

Choice of a case series study design

An analytical study design could not be used for the two studies on diamond and platinum mine workers because of the small numbers of diseased men who met the inclusion criteria. Such small numbers were not anticipated when the study was embarked upon, especially for platinum miners with silicosis. However, poorly documented employment histories resulted in the exclusion of many potential

study subjects and, as a result, case series study designs were adopted for the analysis of both groups of mine workers.

Initially, all miners in each of the two mining sectors were identified from the PATHAUT database. Those with evidence, from any of the data sources described in Section 2.1, of having worked in another mining industry were then excluded, reducing the numbers in both groups substantially. The next step was to exclude those who were employed in either the diamond or platinum mining sectors for less than a year as they were unlikely to have been exposed to dust levels sufficient to cause disease in such a short period.

One of the consequences of poorly documented employment histories is that many mine workers have 'gaps' in their employment records. Mine workers with such gaps were excluded to minimise the potential of bias due to misclassification.

After all exclusions, six diamond mine workers with asbestos-related diseases and 30 platinum mine workers with silicosis and/or fibrosis of the hilar lymph nodes remained in the respective study populations. It was a direct consequence of the mines' failure to record comprehensive work histories that very few diamond and platinum miners met the study inclusion criteria. This is discussed in more detail in Section 4.2.

This methodical 'data stripping' approach resulted in the exclusion of all mine workers who had worked in sectors other than the diamond and platinum mining sectors, respectively, and so the possibility of exposure to minerals and/or compounds that caused the disease in question, outside of that particular mining

sector (diamond or platinum), was excluded in every case. This resulted in cases that had, taking into account all available evidence, been exposed to the agent in question during mining of the commodity in the sector in which they were employed.

Although cases series are not ideal for examining exposure-disease associations, this was the only study design option available for the analysis of diamond mine workers with asbestos-related diseases and platinum mine workers with silicosis. It was not feasible to construct a cohort of either mining population, as discussed later in Section 4.4.3 of this thesis but, in the absence of cohorts, case series can be useful. They play an important role in the progress of medical science in that they permit the discovery of new diseases and unexpected effects, and play a role in the recognition of rare manifestations of disease (Vandenbroucke, 2001). The findings from a case series can be used to generate hypotheses for testing with the use of more rigorous study designs (Ho et al., 2008). They have a high sensitivity for detecting novelty and remain one of the cornerstones of medical progress.

Stratified analysis of gold miners with silicosis

The data for the trend analysis of silicosis in gold miners were stratified by population group because, when modelled as a single data set, the goodness-of-fit results were poor. It was reasonable to stratify the miners in this way because of differences in their historical employment patterns, as discussed earlier, their exposure experiences (black miners were employed in dustier jobs), and autopsy referral patterns (discussed in more detail in Section 4.3 below). The stratified analysis produced acceptable goodness-of-fit results.

4.2 OBSTACLES TO OCCUPATIONAL RESPIRATORY HEALTH RESEARCH IN SOUTH AFRICA

The major obstacles encountered during the course of this research apply to all occupational respiratory health research endeavours. First, there is a dearth of occupational respiratory health surveillance databases. Second, employment histories are poorly documented by the mining companies, which undermines the quality of research undertaken. Third, there is a lack of available and appropriate dust measurements which restricts attempts to calculate dose-response relationships, or to quantify risks from exposure to specific minerals.

There is a dearth of occupational respiratory health surveillance databases

As mentioned in Section 1, the absence of comprehensive occupational respiratory health surveillance databases is the primary reason that studies on silicosis (and other diseases) have been largely limited to cross-sectional designs on select populations. This is not unique to South Africa; there is no systematic surveillance of occupational disease in any African country (Loewenson, 1999).

Attempts have been made, in the past, to establish surveillance systems. In the early 2000s, the NIOH launched two such systems, based on voluntary case reporting by health practitioners, viz. the Surveillance of Occupational Respiratory Disease in South Africa (SORDSA) and the Surveillance of Upper Limb Musculoskeletal Disorders in South Africa (SAMOSA). Both surveillance systems failed because of incomplete reporting coverage, underreporting, reporter fatigue and competing demands on overworked health practitioners (NIOH, 2003; Sawry et al., 2006).

Given the dearth of consolidated occupational health related data, the launch, by the DMR, of the South African Mines Occupational Diseases Database (SAMODD) in September 2000 (Torres et al., 1998; HST, 2000) represented a significant milestone. Although SAMODD was anticipated to improve the monitoring of disease trends and the identification of priority issues and workplaces in the long term (Worku and Ohaju, 2004), no reports on the data have been published, and the system is non-functional.

There have been some cross-sectional studies on silicosis based on data collected by individual mines (see Table 3), primarily from routine occupational medical examinations of mine workers. Any data routinely and systematically collected by the mines over an extended period could be used for disease surveillance but this would be limited to those particular mines. Data from the various mines have not been collated into a larger database and analysed to provide an overall picture of disease in the gold (or other) mining sector.

Both the MBOD and the CCOD have data on occupational diseases in mine workers but these are broadly categorised. For example, silicosis and asbestosis are categorised together as first or second degree pneumoconiosis. In addition, neither agency has published an annual report since 2001 and 2003, respectively, despite the statutory requirement to do so. Although disease data are recorded on the MWC database, they are not accessible for analysis. There is, however, a move towards simplifying the MWC system to make the data more readily accessible.

Mine employment histories are poorly documented

The PATHAUT database is an administrative database that was designed for the diagnosis of compensable disease, as mentioned in Section 1.2.5 and discussed in more detail, later, in Section 4.3. Together with MWC data, the PATHAUT data are used to review a potential case for payment of compensation. Compensation is paid to the next of kin if the miner had an occupational disease that could be linked to exposure while employed in a particular mining sector. Comprehensive employment histories are not necessary for this, although missing employment records can be problematic if they pertain to the exposure in question.

Nevertheless, detailed work histories are essential for the conduct of meaningful analytical research, and the issue of poorly recorded employment histories, in all data sources reviewed, was a major limitation in the two case series analyses. This is problematic when trying to attribute disease to exposure to dust in a specific mining sector, such as silicosis in platinum miners, many of whom were recruited when gold production began to decrease. So, unless a platinum mine worker's record of service is complete, one cannot assume that his silicosis was contracted in the platinum mines if there are gaps in the service history, as he may have also worked in the gold mining sector.

The computerised employment data on the PATHAUT database are limited because the database makes provision for the recording of employment in a maximum of five different mining sectors. This information is recorded as 'gold, 'platinum', etc. with no additional details. Employment outside the mining industry may also be recorded in this section as 'other industry', with no further information. The only employment dates recorded are the first and last year of employment in

the mining industry overall. Additional information is sometimes recorded in the booklets as hand-written notes, but this is generally limited to the mining company in which the miner was most recently employed and is not entered into the electronic database.

Comprehensive mining work histories are not available from the MBOD files. First, not all miners are registered with, and have files at, the MBOD. Second, although the files contain documents specifically designed for the recording of mining work histories (including specific occupations and dates) that should be completed at every physical examination visit, this information is very seldom collected. Details can sometimes be obtained by scrutinising the hand-written notes in the individual files. Third, not all the files and/or data are accessible. Some files have been mislaid, lost or damaged over the years, and the data are not computerised; in many cases, only the autopsy report is in the file.

While TEBA has comprehensive electronic employment records for some mine workers, these are often incomplete. This is primarily due to the fact that records are kept only for mine workers that register with TEBA, and only from the date that they register. TEBA was initially set up for registering gold mine workers and this is the group with the largest representation on the database.

The mines themselves are often able to supply only limited information. This was highlighted when attempting to ascertain work histories from mine company records archived at the individual mines. None of the mines that was approached for records of deceased miners was able to provide information on every man and, in some instances, where data were available, they were

evidently incomplete. At face value, the mining companies appear to have little interest in maintaining complete and accurate records that do not have a direct bearing on profits.

There is a lack of available and appropriate dust measurements

Another obstacle to this and other occupational respiratory health research is the unavailability of dust measurements. In their absence, the less robust proxy measurements of age at autopsy and duration of employment are used to estimate cumulative dust exposures.

The DMR requires all mines to conduct occupational hygiene surveillance on a regular basis, according to prescribed guidelines. Regular reporting of levels of pollutants is required, based on full-shift measurements and the use of personal monitors. Occupational hygienists are also required to conduct periodic risk assessment exercises in the workplace during which areas are screened for potentially harmful airborne substances.

However, many of the mines are apathetic about measuring dust and do not follow the regulations. Measurements are sometimes made over a period of time shorter than a full shift, depending on convenience and the level of pollutants present. In some platinum mines, silica levels are believed to be too low to measure, and they are commonly reported as "below the detectable limit" (Girdler-Brown et al., 2006).

Silica dust measurements reported by the gold mines are estimated, rather than measured directly, and are based on a number of extrapolations. The procedure

followed when measuring respirable dust is to divide the mine workers into homogeneous exposure groups (HEGs) and then to collect respirable dust from a sample of mine workers in each HEG. The percentage of silica is measured in only a proportion of these respirable dust samples. The mean silica percentage is then calculated for each HEG and the silica concentration is estimated for each respirable dust sample by calculating the product of the mean silica percentage for each HEG and the respirable dust sample (Rees et al., 1999). The reliability of the data is affected by the fact that the dust measurements are collected for risk assessment in accordance with the ODMW Act, for purposes of calculations of levies that the mines pay. The measurements are not validated by the DMR or any other external agency.

Asbestos is not measured routinely in most mines although there are anecdotal reports of its presence in many mine environments.

4.3 THE PATHAUT DATABASE

Autopsy databases, such as the PATHAUT database, provide important public health information. With regard to occupational health, while it is essential to strengthen in-life systems to improve compensation mechanisms and strategies for early intervention, autopsies continue to play a crucial role in undiagnosed disease. This was demonstrated by Hnizdo et al. in 1993 in their study on the comparison of X-ray and autopsy diagnoses of silicosis: the sensitivity of X-rays was < 0.4 for all three independent X-ray readers in the study (Hnizdo et al., 1993). The rate of undiagnosed extensive active pulmonary tuberculosis is also high at around 60% (Murray et al., 2000).

The PATHAUT data have been widely used, by local government departments, and researchers from South Africa and other countries. The database was the primary source of data for the research described in this thesis, and it is appropriate to discuss both its strengths and its limitations more comprehensively. While some of these strengths and limitations were mentioned in Sections 1.2.5 and 2.1.1, they are repeated and expanded upon in this section. The usefulness of the PATHAUT database is also emphasised.

Strengths of the PATHAUT database

The PATHAUT database is the only database that contains comprehensive pathological data on the occupational respiratory health of mine workers and workers from related industries in South Africa. These pathological data are of high quality as autopsy examinations have been performed, over the years, by a limited number of skilled pathologists, all of whom have been trained in lung pathology, and the examinations are performed according to sensitive standardised methods.

The validity and reliability of the pathological diagnoses on the database were addressed a few years after the database had been established (Hessel et al., 1987b), and again almost 20 years later (Naidoo et al., 2005). The latter study specifically demonstrated that there was good agreement between the pathologists with respect to the diagnosis of the pneumoconioses.

In addition to pathological data, the PATHAUT database also contains basic demographic information, and some limited information on the mining sector(s) in which the worker was employed (see Section 2.1.1).

As discussed in Section 1.2.5, the database is large, containing data on over 105 000 miners, and includes records dating back 37 years to 1975. Current and ex-miners of all population groups are represented, as are all mining sectors in all nine provinces of South Africa (Ndlovu et al., 2010). The data are readily available in electronic form. As such, it is an ideal database for trend analyses of occupational respiratory diseases.

Because both miners and ex-miners come to autopsy, regardless of the clinical cause of death or their disease status, potential bias due to the healthy worker effect is reduced in analyses using these data.

Although the PATHAUT data are not double-entered into the electronic database, they are verified before the autopsy reports are signed by the pathologists and disseminated. Routine data validation checks and cleaning procedures are routinely performed. The quality of the data has improved over the years as the database has evolved and more quality checks have been built into the system.

Quality control has also resulted in very few records with missing information. The most commonly missing data are age at death and duration of employment in the mining industry but, in 2010, only 1.3% and 3.1% of cases had no age at death or employment duration, respectively.

Limitations of the PATHAUT database

As mentioned, the PATHAUT database is an administrative database, developed for the purpose of paying financial compensation to the families of deceased mine workers with respiratory disease caused by exposure to dust while working in

the mines. Although several changes have been made to it since its inception, data are still collected primarily for individual reporting purposes.

While the pathological data are of excellent quality, there are limitations in terms of the employment information on the database. As expected, only employment in the mining industry is recorded. Information regarding employment outside the mining industry would need to be obtained from other sources, such as pension records, and family and friends.

Like all databases, the PATHAUT database does not contain a record of every deceased mine worker in the country. White miners and ex-miners are well represented on the databases as their families are generally more aware of the financial benefits than those of black miners, and are therefore more likely to submit the deceased's cardio-respiratory organs for examination. Black retired mine workers, on the other hand, are underrepresented. Many black miners who retire or leave the mining industry for other reasons return home to the rural areas of South Africa and neighbouring countries from where they were recruited, thereby missing the opportunity to have their hearts and lungs examined at autopsy because of the lack of health services. Black men with severe disease are especially likely to be underrepresented for this reason. There are very few autopsies in black miners older than 60 years of age or with more than 30 years of mining service. These biases may result in underestimates of disease in black miners. The service is utilized by around 80% of miners, black and white, who die while employed and these men represent a healthy workforce. A much higher proportion of black mine workers come to autopsy if they die while still employed.

Unfortunately, many of the smaller mines do not participate in the autopsy programme, to the financial detriment of the families of the deceased workers.

Usefulness of the PATHAUT database

Despite these inherent limitations and biases, the PATHAUT database provides the only information of this kind in South Africa. The large numbers of mine workers in the database go a long way in counteracting some of the common problems encountered when using administrative data for research. The data can, and have been, used successfully for large epidemiological studies, and lend themselves to disease trend analyses, such as that described in Paper 1 of this thesis.

In addition, smaller groups at risk can be identified from the PATHAUT database and studied, such as those in Paper 3 and Paper 4. However, studies such as these do require a review of data sources in addition to the PATHAUT database, such as those described in Section 2.1.2.

4.4 RECOMMENDATIONS

A number of recommendations emanate from the research results reported in this thesis. These are discussed under the broad headings of policy, operational and research recommendations. Policy recommendations include the need to establish effective disease surveillance programmes, document comprehensive work histories, measure and report silica dust and asbestos fibre levels, and address the problems that have resulted from the migrant labour system. Screening for asbestos- and silica-associated diseases, upgrading the PATHAUT database, and extending the NIOH outreach programme are operational

recommendations, while research recommendations are to quantify the risk of asbestos- and silica-associated diseases in diamond and platinum mine workers as well as those in other mining sectors, and to investigate respiratory disease in less well researched mining sectors.

4.4.1 POLICY RECOMMENDATIONS

Establish effective disease surveillance programmes

Monitoring of silicosis and other disease trends can be achieved by means of disease surveillance programmes. These programmes involve the evaluation of a defined population's health status through the periodic collection, analysis, and reporting of data for the purposes of disease prevention. Surveillance data are useful for evaluating the effectiveness of disease prevention and intervention programmes. The PATHAUT database is currently the only medical surveillance tool in South Africa that can be used to monitor disease in the mining industry. However, disease surveillance should not be confined to the PATHAUT database. The DMR has a national responsibility to institute a functional disease surveillance programme, and mining companies also need to establish such programmes.

The trends in silicosis rates reported here should be integrated into programme and policy issues as this is where trend data can be most useful (Rosenberg, 1997). Rates of silicosis in gold miners are unacceptably high, hence South Africa's participation in the WHO/ILO initiative to eliminate silicosis by 2030. Silicosis rates need to be monitored on an ongoing basis, in an effort to evaluate the success of the Programme. The PATHAUT database is one source of data for this, but other data sources need to be identified, such as disease surveillance programmes.

Earlier this year (2011) the Constitutional Court ruled in favour of an AngloGold Ashanti employee which allowed him to sue Anglo American for contracting silicosis. The ruling has opened the way for retired mineworkers to sue companies for contracting lung diseases on the mines and, in September, a group of 450 former gold mine workers lodged papers in the London High Court against Anglo American. More than 300 000 retired gold mine workers who contracted silicosis or TB during or after their employment in the industry, are pushing for the start of a multibillion-rand class action next year (Matomela, 2011). In view of this action, the gold mining industry needs to closely monitor silicosis rates amongst its workers.

Document comprehensive work histories

Complete work histories, including those outside of the mining industry, are important for disease surveillance systems to be effective and successful. Unfortunately, incomplete work histories are common throughout the mining industry, and this precludes accurate risk assessments. All mines and mining companies need to be aware of their obligations in terms of the Occupational Diseases in Mines and Works Act. Related to this is the collection and documentation of work histories.

All mining companies have a social responsibility to record comprehensive work histories, from the time the worker was first employed, including those outside of the mining industry, for the benefit of families seeking compensation for occupational disease. There are many opportunities to collect this information, including entry, annual and exit examinations. The information should be collated by the mine medical services and should be recorded in both the human resources and medical files. Details should be recorded with respect to dates,

specific companies and occupations, tasks, and geographical location of each employer. Information pertaining to the geographical location of birth and lifetime residence should also be noted.

Records should be kept for at least 60 years to ensure that evidence of employment (and exposure) exists should a mine worker be diagnosed with an occupational disease during his working life, after retirement or after death. Although the Health Professionals Council of South Africa recommends keeping records for 25 years in cases where "a patient has been exposed to conditions that might manifest in a slow-developing disease, such as asbestosis"(HPCSA, 2007), this time period is insufficient if a worker dies with an asbestos-related disease in his 80s, for example, and the family wants to claim compensation.

Comprehensive employment information will enable more rigorous analysis of disease associations related to different exposures during mining processes, as well as during employment in different industries unrelated to mining. It should be mandatory for all mines to keep comprehensive records.

In December 2010, de Beers Consolidated mining company conducted a pilot study on their medical surveillance data collection system and, in 2011, they introduced the collection of detailed past histories of environmental and work exposures at the routine medical surveillance examinations. This is a direct consequence of the outputs of this study, which have already raised awareness and resulted in attempts to rectify the highlighted problems.

Measure and report silica dust and asbestos fibre levels

Mining companies should conduct risk assessments, specifically for silica and asbestos. Both minerals occur throughout South Arica and it is likely that they will be present in many mines. Both have the potential to cause disease in mine workers, even at low concentrations, and all mining companies should incorporate the routine measurement of these dusts into their occupational hygiene programmes, regardless of whether or not there is a perceived risk. All fibres should be identified, characterised and measured, by an outside agency such as the NIOH, if necessary. The diamond mining companies, especially, need to take cognisance of the risk of asbestos exposure to their workers. Dust collection, measurement and recording procedures should be regularly audited by the DMR, and the dust measurements should be validated.

Address the problems that have resulted from the migrant labour system

Although much has been published on migrancy and health, the mining industry seems to lack awareness of the seriousness and long-term implications thereof. Occupational health professionals need to actively engage management on these issues; oscillating migration is a risk factor for work-related disease and thus falls within the ambit of occupational health services. Measures to prevent disease in migrant miners would include alternatives to single-gender hostels (thereby reducing HIV transmission), controlling exposure to silica dust, and isoniazid preventive treatment to reduce TB in miners with silicosis (Rees et al., 2010).

It would be unreasonable to recommend ending migrant labour as many men and women are dependent on employment away from their homes to support

themselves and their families. However, interventions are needed to ameliorate the negative consequences oscillating migration.

There is a need for both multi-level regional and national policies which acknowledge the economic value of migrant workers and provide for infrastructure, such as strengthening health services both at locations where migrant workers are employed or live, and in labour-sending areas (Rees et al., 2010). Health services in labour-sending areas are over-stretched by returning migrants (Clark et al., 2007) and need resources if they are to diagnose and manage occupationally-related diseases. In particular, it is important to establish cross-border referral systems to ensure continuity of treatment of TB (Rees et al., 2010).

4.4.2 OPERATIONAL RECOMMENDATIONS

Screen for asbestos- and silica-related diseases

While mines are required, by law, to establish and maintain disease surveillance programmes (RSA, 1996), occupational health practitioners need to be aware that disease other than that recognized as a risk in the mining sector in which the employee works, may also occur. All mine medical services need to have systems and resources in place for the diagnosis of asbestos- and silica-related diseases that might be caused by exposure to dust in previous work places, as well as in their current workplace. Regardless of the source of disease, mining companies have a responsibility to screen/test for, and treat workers with, any occupationally-related disease.

Upgrade the PATHAUT database

It is recommended that the section on work histories in the PATHAUT database be expanded to include fields for capturing data on all industries in which the person has been employed since he started working, as well as the specific occupations and dates of employment. A field should also be added for the age at which he/she started working. This information will identify those who worked outside of the mining industry at the beginning of their working lives where comprehensive employment information is unavailable. However, only comprehensive work histories will enable the identification of 'gaps' in mining employment. Information that cannot be obtained from work records should be requested from family members, preferably within the first few weeks of receipt of the cardio-respiratory organs.

Extend the NIOH outreach programme

In 2006, in an attempt to improve utilisation of the autopsy service, the Pathology Division of the NIOH initiated an outreach programme to addresses miners' rights with regard to autopsy examination for compensable disease ascertainment. The primary objective of the outreach programme is to increase the number of ex-mine workers that come to autopsy. Areas of the country that are underrepresented in the PATHAUT database have been targeted, primarily the rural areas that have poor health service resources. More recently, the programme has been extended into neighbouring countries, as ex-mine workers from these countries who worked in the mines in South Africa are also eligible for compensation. The outreach programme should be extended to individual mines to educate currently employed miners about their rights after death, as well as the mine medical personnel.

4.4.3 RESEARCH RECOMMENDATIONS

Quantify the risk of asbestos- and silica-related diseases in diamond and platinum mine workers

Now that risks of asbestos- and silica-associated diseases have been identified in diamond and platinum mine workers, respectively, more research is necessary to quantify the risks. While case series are useful in identifying unexpected disease, such as that reported in this thesis, analytical study designs are more robust when trying to establish stronger associations between exposure and disease outcome, and quantify risks.

Cohort studies of mine workers who could be followed up until death would enable the risk of disease due to the 'accidental mining' of minerals, such as asbestos and silica, to be quantified, as relative risks could be calculated. However, cohort studies in the mining industry are hindered by a number of factors. First, diseases such as silicosis and mesothelioma take many years to develop and it would be decades before the results of cohort study analyses were available. Second, migrant workers are difficult to follow up, as discussed earlier in this thesis, and the attrition rate of any mining cohort would be high.

These obstacles are not insurmountable but failure of the mining industry to adequately measure dust levels and record work histories needs to be resolved before further analytical research can be conducted. Neither the platinum nor the diamond mining industries acknowledge or are aware of the risks of ill health in their workforce due to dust exposure. Once the mining companies acknowledge that there is a risk, and start measuring dust levels and recording comprehensive employment histories, cohort studies can be implemented.

Research collaborations with neighbouring and other African countries where platinum and diamonds are mined, could be explored, viz. Zimbabwe (platinum and diamonds), and Angola, the Democratic Republic of Congo, Ghana, Tanzania, Lesotho and Botswana (diamonds).

Extend research on asbestos- and silica-associated diseases to other mining sectors

A similar approach to the identification of disease in diamond and platinum mine workers could be adopted in other mines where there is evidence of potential exposure to these minerals. Many mines, worldwide, are 'contaminated' with asbestos mineral tailings (Juntilla et al., 1996; Lee et al., 1999; Nolan et al., 1999; Gamble and Gibbs, 2008; Price, 2008; Loyola et al., 2010). Asbestos has been reported in a vermiculite mine in South Africa (Hessel and Sluis-Cremer, 1989) and there are anecdotal reports of asbestos in other mines, such as the iron and manganese mines in the Northern Cape province. Amphibole asbestos fibres have been identified from a South African chrome/platinum mine tailings dump (James I Phillips, NIOH, personal communication), and rocks which clearly contained tremolite were identified during a field trip to the platinum mining area.

Investigate respiratory disease in less well researched mining sectors

The mineral diversity of the Bushveld Complex and the extensive mining of these minerals greatly increase the potential for exposure of mine workers to some or all of the associated minerals. Thus, the risk of respiratory disease need to be investigated in other mining sectors that have been less well researched, such as tin, vanadium, feldspar, etc. Currently, very little is known about the health effects of exposure to dusts generated by the mining of some of these commodities.

The mineralogy of the surrounding areas should be studied for identification of potential hazards. The grey literature, in particular, needs to be reviewed for single case reports and anecdotal evidence of risks. The PATHAUT data can then be analysed to identify mine workers who were employed in a particular mining sector and developed the disease of interest. Analysis of the PATHAUT data is an inexpensive, quick, desk-top method but it will need to be supplemented by data from other sources.

4.5 CONCLUSION

The alarming increases in the proportion of gold miners with silicosis at autopsy reflects the failure of the gold mining companies to reduce silica dust to safe levels. As miners continue to age and work for longer periods, the burden of silicosis and its associated diseases will continue to rise. The high proportion of South African gold miners with silicosis, many of whom are already burdened with tuberculosis and/or HIV, has far reaching implications in terms of health services which need to be prepared for increasing morbidity and mortality rates in both current and ex-miners.

Much needs to be done to compensate for the far-reaching ill health effects of the migrant labour system, and appropriate and relevant policies need to be instituted to assure that ex-miners, in particular, have access to health care and compensation services.

Disease previously undocumented in diamond and platinum mine workers has been found to be associated with exposure to mineral dusts and fibres. This work

needs to be extended to quantify risks and identify potential risks in other mining sectors where health research has not been done.

The quality of future research on the health of mine workers hinges on the DMR putting legislation in place regarding the documenting of comprehensive work histories, and the measurement, reporting and validating of dust levels and dust measurement procedures. Such legislation will go a long way towards protecting the future health of mine workers.

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APPENDIX 1

Nelson G, Girdler-Brown B, Ndlovu N, Murray J. Three decades of silicosis: disease trends at autopsy in South African gold miners. *Environ Health Perspect* 2010; 118(3):421-6.

Three Decades of Silicosis: Disease Trends at Autopsy in South African Gold Miners

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Background: Eliminating silicosis is a priority of the International Labour Organization and the World Health Organization. Prevalence is particularly high in developing countries.

Objectives: We describe trends in silicosis among South African gold miners who had had an autopsy between 1975 and 2007 and quantify the contributions of age at autopsy and employment duration to these trends.

Methods: South African miners and ex-miners are eligible for autopsy examination for occupational lung disease, regardless of the clinical cause of death, and the families of deceased mine workers may receive compensation from the government of South Africa. Miners who died from external causes and who had been employed in the gold mines for > 1 year were stratified by population group because of differences in exposure, patterns of employment, and autopsy referral patterns. We extracted data from PATHAUT (Pathology Automation System) and used Stata 10 to estimate trends in relative proportions of silicosis that were standardized for age and employment duration.

Results: The crude proportion of silicosis for white miners was six times that of black miners in 1975. By 2007, it was 1.5 times higher for black miners. The proportion of miners with silicosis increased from 0.03 to 0.32 for black miners and from 0.18 to 0.22 for white miners. The increase can be explained by increasing age and employment duration for white miners. For black miners, it can be only partly explained by these two factors.

Conclusion: As miners continue to age and work for longer periods, the burden of silicosis will continue to rise. South Africa is committed to global efforts to eliminate silicosis by 2030. The autopsy database allows for disease surveillance, which is necessary to monitor the success of this initiative.

Key words: Africa, mining, PATHAUT, pneumoconiosis, silica. *Environ Health Perspect* 118:421–426 (2010). doi:10.1289/ehp.0900918 available via *http://dx.doi.org/* [Online 23 November 2009]

Silicosis is a major occupational health concern in both developed and developing countries. Although disease rates are reported to be decreasing in some developed countries (Bang et al. 2008; Gerhardsson 2002), silicosis is still very common in low- and middle-income countries (Lehtinen and Goldstein 2002), and mining countries have particularly high prevalences of silicosis (Rees and Murray 2007). South Africa joined the International Labour Organization and the World Health Organization (WHO) Global Program for the Elimination of Silicosis and has developed a national initiative under the leadership of the Department of Labour. In 2003, the Mine Health and Safety Council developed its own milestone, namely, that after December 2013, using present diagnostic techniques, no new cases of silicosis will occur among previously unexposed individuals (individuals unexposed before 2008) (Mine Health and Safety Council 2007).

South African legislation provides mineworkers who were ever employed in the mines of South Africa the right to have their cardiorespiratory organs examined and their families to be compensated for occupational lung disease, regardless of the cause of death (Occupational Diseases in Mines and Works Act of 1973). Although the autopsy service is used by approximately 80% of all miners who die while employed and by many white ex-miners, very few black ex-miners have had an autopsy. Autopsies are performed at the National Institute for Occupational Health (NIOH) in Johannesburg, and data on pathologic diagnoses, personal information (demographics), and work histories, including the commodities mined and the length of time spent in each, have been stored in an electronic database known as PATHAUT (Pathology Automation System; National Institute for Occupational Health, Johannesburg, South Africa) since 1975 (Hessel et al. 1987). The database currently contains more than 100,000 records and provides an ideal information source to analyze trends in respiratory disease over the past three decades. Of the miners who have come to autopsy, approximately 80% have worked in the gold mines, with employment ranging from 1 month to several decades.

During the past three decades, the gold mining industry has employed hundreds of thousands of workers, most of whom were black underground miners. Employment peaked in the mid-1980s at around 550,000. In the 1970s and 1980s, most black miners were migrant workers from rural areas of South Africa and from neighboring countries, employed on relatively short contract periods of around 18 months. When their contracts expired, they returned to their homes, after which they could apply for a new contract, although many men found employment elsewhere. On the other hand, white miners were employed as career miners. During the last two decades, this pattern has slowly changed as the contract system fell away and black miners became increasingly employed as career miners, although many are still migrants.

Historically, black miners have been exposed to higher dust levels than were white miners. Black men are employed underground in high-dust occupations such as drilling and stoping, whereas white men are largely employed in supervisory positions and maintenance jobs with lower dust exposure.

The only study on the prevalence of silicosis among white gold miners in South Africa is a large cohort of more than 2,200 men who were employed for an average of 24 years from 1940 to the early 1970s. By 1991, the prevalence of radiologically diagnosed silicosis was 14% (Hnizdo and Sluis-Cremer 1993). Murray and Hnizdo (unpublished data) analyzed the cohort autopsy data for an exposure–response relationship and found silicosis in almost 52% of the deceased mine workers who had been exposed to dust for 40 years.

Several cross-sectional studies have been conducted on black gold miners since the early 1990s, either on employed miners (Churchyard et al. 2004) or on ex-miners (Girdler-Brown et al. 2008; Steen et al. 1997; Trapido et al. 1998). Only one study has analyzed time trends in silicosis, in black gold miners who died of external causes while in mine employment (Murray et al. 1996).

In the present study we examined long-term trends in the proportions of both black

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and white South African gold miners with silicosis, who died from external causes. We used autopsy data from miners and ex-miners, which we standardized for age at death and duration of employment in the analysis.

Materials and Methods

Study population. Gold miners were defined as those who had ever been employed in the gold mining industry. The study population comprised all gold miners who had been employed for > 1 year and who died from an external cause of death. External causes of death included those coded in the *International Classification of Diseases* (WHO 1992) as injuries (e.g., as a result of mine or traffic accidents), burns, poisoning, drowning, intentional self-harm, and homicide. The following variables were extracted from the PATHAUT database for 1975–2007: population group, duration of gold mining employment, age at death, and whether silicosis was present at autopsy.

Miners have been significantly affected by the human immunodeficiency virus (HIV) epidemic during the past two decades, and tuberculosis is one of the most common causes of death in this HIV-affected population group (Murray et al. 2007). Miners with tuberculosis are more likely to have silicosis than those without (Corbett et al. 2000). Furthermore, the presence of silicosis increases the risk of mortality from tuberculosis, which is exacerbated by HIV infection (Churchyard et al. 2000). Thus, we felt that including those who died from causes other than external causes might bias the results of the trend analysis, and they were excluded from the study population.

Diagnosis of silicosis. Silicosis was defined as the presence of palpable silicotic nodules on macroscopic examination of the lungs, which was then confirmed on microscopic examination. The diagnoses were made by experienced pathologists.

Statistical analyses. Gold miners were stratified by population group (black and white) because of their differing employment, dust exposure, and autopsy referral patterns. Potential risk factors for the development of silicosis were considered to be age at death and duration of employment in the gold mining industry (as proxy measures of dust exposure in the absence of dust measurements), and year of autopsy.

All data analyses were carried out using Stata 10, (version 10; StataCorp LP, College Station, TX, USA) except 95% confidence intervals (CIs) for standardized proportion ratios (SPRs), which were calculated manually. Trends in the crude proportions of miners with silicosis by year were assessed by means of simple linear regression with weighting by the inverse of the variances of the single year proportions. Binary logistic regression modeling with silicosis (1 = present, 0 = absent) as a dichotomous outcome was used to estimate associations with the explanatory variables.

We compared the proportions of black and white miners with silicosis by 5-year intervals using direct standardization for age and duration of employment. Age categories that were selected for the standardization were < 40, 40–49, 50–59, and ≥ 60 years. Duration of employment categories were < 10, 10–14, 15–19, 20–24, 25–29, and ≥ 30 years. Age-group– and duration-group–specific proportions were then weighted by the total number of miners (black and white combined) in each age and duration category in the calculation of the standardized proportions with silicosis by year of autopsy interval. The variances for the year of autopsy interval black and white standardized proportions were calculated using the binomial approximation method without corrections for finite populations (Cochran 1977). The exact 95% CIs for the SPRs were calculated using the formula given by Curado et al. (2007).

We performed the same direct age and duration of employment standardized analysis of the proportions of miners with silicosis for those who died from nonexternal causes. The standardization was carried out using the same weightings that were used for those dying from external causes to enable comparisons to be made between the two groups.

The modeling process was carried out with the three explanatory variables (year of autopsy, age at death, and duration of employment) coded as categorical variables. Modeling was attempted with all possible combinations of coding options for these three variables (continuous vs. categorical), but none of the models that included one or more of the variables as a continuous variable had resulting acceptable goodness-of-fit test results. Interaction terms for year and age, year and duration, and age and duration were also included in the initial models for black and white miners.

Initially, age group categories were made up of 10-year intervals and duration group categories were made up of 5-year intervals. However, some categories were subsequently collapsed because of very small numbers of cases in some of the groups. In addition, we combined the first three age group categories for white miners because we found no statistically significant difference between them.

Postregression analysis for the binary logistic regression model included the Pearson's goodness-of-fit test and, as appropriate (if the number of covariate pattern cells with expected values < 5 exceeded 10% of all the cells), the Hosmer-Lemeshow goodness-of-fit test, as well as calculation of the area under the receiver operating characteristic (ROC) curve (Hosmer and Lemeshow 2000). Decisions to drop any explanatory variable from the binary logistic regression models were based on a significant Likelihood Ratio test with an alpha value of 0.05.

The study was approved by the University of the Witwatersrand Human Ethics Committee (protocol M050228).

Results

Of the 98,323 miners in the PATHAUT database, 76,231 (77.5%) had ever worked in the gold mining industry. Of these, 50,867 (66.7%) were black and 25,282 (33.2%) were white. The remaining 82 miners were classified as belonging to another population group, or information about their population group was missing or could not be validated.

A total of 19,143 miners died from external causes and worked for > 1 year in the gold mining industry. Table 1 lists the numbers of subjects excluded from the analysis and the reasons for their exclusion. The final study population comprised 16,411 black miners and 2,732 white miners.

The mean age at death of the black miners was 35.0 years (median, 34 years; range, 17–82 years), whereas the mean age at death of the white miners was 48.3 years (median, 47 years; range, 18–96 years). For the black miners, the mean age at death rose from 33.0 years in 1975 to 43.4 years in 2007, whereas the mean age at death for the white miners rose from 44.1 years in 1975 to 54.4 years during the same period.

Table 2 lists the age distributions of the study subjects, by population group, together with the age-specific proportions of silicosis found at autopsy. The proportion of black miners with silicosis had reached 0.18 by 40–49 years, more than double that of white miners in the same age group. The proportion of miners ≥ 60 years old with silicosis was higher among white miners than among black miners.

The average duration of employment in gold mining for the black miners was 7.7 years (median, 6.1 years; range, 1.1–48.0 years), and that for white miners was 17.1 years

Table 1. Numbers of gold miners on PATHAUT database and resons for exclusions.

Exclusion	No.	Remaining
Ever worked in gold mining industry		76,231
No age at death or birth date	2,727	73,504
No duration of gold mining employment	11,145	62,359
Race other than black or white	65	62,294
Validation of data not possible	21	62,273
Age at first employment < 15 years	194	62,079
Duration of employment ≤ 1 year	4,818	57,261
Cause of death not "external"	38,118	19,143[a]

[a]Includes 16,411 black miners and 2,732 white miners.

(median, 15.0 years; range, 1.1–54.0 years) for white miners. During the study period, the mean duration of employment increased from 5.6 to 13.4 years for the black miners, whereas that for the white miners increased from 17.5 to 20.1 years.

Table 3 illustrates the distribution of study subjects by duration of employment, by population group, together with the relevant proportions of miners with silicosis for each duration group. As the duration of employment increased among black and white miners, so did the proportion with silicosis. The proportion of black miners with silicosis reached 0.22 after 15–19 years of employment, but reached only 0.20 after 20–24 years in white miners. There was silicosis in both black and white miners who had been employed for < 10 years.

The results presented in Tables 2 and 3 show the crude proportions of miners with silicosis. The crude proportion, for the entire study period, was lower for black miners (0.08) than for white miners (0.14). However, after direct standardization for age and duration of employment, and applying the population group-specific proportions by age and duration of employment to this combined sample, the standardized proportions with silicosis changed to 0.09 for black miners and 0.05 for white miners (Table 4). This represents a change in the ratio of these proportions from 0.56 for the crude data to 1.70 for the proportions standardized for age and duration of employment. We calculated these ratios using estimates that were not rounded off; thus, they differ slightly from those based on the rounded-off estimates in the tables.

The differences between the population group standardized proportions for miners who died from external causes (Table 4) were not statistically significant for the initial period 1975–1979 or for 2000–2007. However, for the remaining 5-year time intervals, the proportions of black miners with silicosis were significantly higher than those of white miners, with SPRs of 2–3. The two periods for which the differences were not statistically significant are somewhat unusual, in that the crude proportion for black miners in the first period was lower than that for the other periods, whereas that for white miners in the last period was higher than for the other periods. It is apparent, however, from these results, that population group behaves as an effect modifier of the relationship between calendar interval and proportion with silicosis for the period 1980–1999.

Table 5 shows the age and duration of employment-standardized proportions of miners with silicosis for those miners who died from nonexternal causes. The differences between the population group standardized proportions were statistically significant for all periods other than 1975–1979. The proportions of black miners with silicosis were significantly higher than those for white miners for the remaining 5-year intervals.

In addition, although the year group-specific standardized proportions with silicosis were very similar for the white miners who died from nonexternal causes, we found an increasing trend in these proportions for black miners during the period of the study.

The standardized proportion of white miners with silicosis was very similar for those who died from external and nonexternal causes (0.06 and 0.05, respectively, with a ratio of 1.11; 95% CI, 0.95–1.29). However, for the black miners with silicosis, the proportion of those who died from nonexternal causes (0.13; 95% CI, 0.09–0.10) was much higher than for those who died from external causes (0.09; 95% CI, 0.12–0.13). The ratio of these two

Table 2. Crude proportions with silicosis at autopsy of miners, by age at death, stratified by population group: 1975–2007 (external causes of death).

	Black			White		
Age group (years)	*n*	Silicosis present	Proportion	*n*	Silicosis present	Proportion
< 30	5,285	27	0.00	386	1	0.00
30–39	6,234	328	0.05	529	17	0.03
40–49	3,561	635	0.18	587	45	0.08
50–59	1,184	283	0.24	535	95	0.18
60–69	133	28	0.21	374	120	0.32
> 69	14	3	0.21	321	113	0.35
Total	16,411	1,304	0.08	2,732	391	0.14

Table 3. Crude proportions with silicosis at autopsy of miners, by duration of employment, stratified by population group: 1975–2007 (external causes of death).

	Black			White		
Duration of employment (years)	*n*	Silicosis present	Proportion	*n*	Silicosis present	Proportion
< 10	11,683	443	0.04	978	19	0.02
10–14	2,815	377	0.13	362	28	0.08
15–19	1,284	286	0.22	320	40	0.13
20–24	400	119	0.30	306	60	0.20
25–29	161	61	0.38	265	74	0.28
> 30	68	18	0.26	501	170	0.34
Total	16,411	1,304	0.08	2,732	391	0.14

Table 4. Age- and duration-standardized proportions of miners with silicosis, by population group and time interval (external causes of death), with time interval-specific SPRs (blacks to whites).

	Black			White			
		Proportion with silicosis			Proportion with silicosis		
Year of autopsy	*n*	Crude	Standardized (95% CI)	*n*	Crude	Standardized (95% CI)	SPR[a] (95% CI)
1975–1979	3,167	0.03	0.04 (0.04–0.05)	487	0.13	0.05 (0.03–0.07)	0.90 (0.63–1.20)
1980–1984	3,795	0.06	0.08 (0.07–0.09)	487	0.11	0.04 (0.03–0.05)	2.19 (1.60–2.99)
1985–1989	4,205	0.08	0.11 (0.10–0.12)	669	0.16	0.04 (0.03–0.05)	2.69 (1.94–3.72)
1990–1994	2,968	0.09	0.09 (0.08–0.09)	549	0.15	0.04 (0.03–0.06)	1.98 (1.34–2.94)
1995–1999	1,321	0.14	0.10 (0.09–0.12)	258	0.13	0.03 (0.02–0.05)	2.98 (1.99–4.47)
2000–2007	955	0.20	0.12 (0.10–0.14)	282	0.18	0.13 (0.06–0.20)	0.96 (0.56–1.63)
All years	16,411	0.08	0.09 (0.09–0.10)	2,732	0.14	0.05 (0.04–0.06)	1.70 (1.45–1.99)

[a]SPRs indicate ratios of the standardized proportion of black miners with silicosis to the standardized proportion of white miners with silicosis.

Table 5. Age- and duration-standardized proportions of miners with silicosis, by population group and time interval (nonexternal causes of death), with time interval-specific SPRs (blacks to whites).

	Black			White			
		Proportion with silicosis			Proportion with silicosis		
Year of autopsy	*n*	Crude	Standardized (95% CI)	*n*	Crude	Standardized (95% CI)	SPR[a] (95% CI)
1975–1979	2,054	0.07	0.06 (0.05–0.07)	3,629	0.31	0.06 (0.05–0.07)	0.95 (0.72–1.27)
1980–1984	1,884	0.13	0.11 (0.09–0.12)	3,890	0.32	0.07 (0.04–0.09)	1.66 (1.18–2.34)
1985–1989	2,010	0.16	0.12 (0.10–0.13)	3,512	0.26	0.05 (0.03–0.07)	2.44 (1.55–3.82)
1990–1994	2,670	0.18	0.12 (0.10–0.13)	3,118	0.24	0.05 (0.03–0.06)	2.57 (1.70–3.89)
1995–1999	3,192	0.23	0.14 (0.12–0.15)	2,284	0.22	0.05 (0.04–0.06)	2.71 (2.01–3.67)
2000–2007	6,466	0.29	0.16 (0.15–0.17)	3,409	0.24	0.06 (0.04–0.09)	2.56 (1.66–3.95)
All years	18,276	0.21	0.13 (0.12–0.13)	19,842	0.27	0.06 (0.05–0.06)	2.27 (2.12–2.43)

[a]SPRs indicate ratios of the standardized proportion of black miners with silicosis to the standardized proportion of white miners with silicosis.

standardized proportions was statistically significant (1.48; 95% CI, 1.37–1.60). We calculated these ratios using nonrounded estimates; thus, they differ slightly from those calculated from the rounded estimates.

The population-group–specific trends in the proportions of miners with silicosis who died from external causes are illustrated in Figure 1. These are crude proportions for the period 1975–2007. The proportion of white miners with silicosis was six times higher than that of black miners in 1975. By 2007, the proportion was 1.5 times higher for black miners. The proportion of black gold miners with silicosis increased steadily from 0.03 in 1975 to 0.32 in 2007 (trend assessment using the variance weighted least squares method: $p < 0.001$). The proportion of white gold miners with silicosis increased from 0.18 to 0.22 ($p = 0.16$) during the same period.

We first modeled the data (using logistic regression modeling) as a single data set with the inclusion of a dichotomous variable for population group. The resulting model proved to have poor goodness-of-fit statistics ($p < 0.01$). We then stratified the data by population group and developed two separate logistic regression models.

Tables 6 and 7 present the results of the binary logistic regression analysis. None of the interaction terms was statistically significant in the models for the black or the white miners. Consequently, we dropped the interaction terms from both models. The results show increasing adjusted odds ratios (ORs) for disease (relative to the baseline groups) as age groups increase for both the black and the white miners. In addition, the adjusted ORs for disease increase (relative to the baseline groups) for both population groups as the durations of exposure increase, apart from white miners with more than 34 years of exposure, where the OR falls to 6.71.

For black miners, all year-of-autopsy groups have raised adjusted ORs, relative to the period 1975–1979, of 1.90–2.94. For the white miners, however, all the year-of-autopsy category–adjusted ORs were not statistically significant (data not shown). This result indicates a lack of evidence that the year-to-year risks have changed for these miners during the study period after adjusting for age and duration of employment. Hence, we omitted the variable for year of autopsy from the model in Table 7.

Silicosis is classified by severity in the PATHUAT database. We analyzed these data but found no trends with regard to severity during the study period.

Discussion

Our results show that the overall proportion of gold miners with silicosis at autopsy is currently high at 0.32 for black miners and 0.22 for white miners (2007 data). In the late 1990s, two groups reported similarly high prevalences in two cross-sectional studies of living black ex-miners, ranging from 22% to 36%, depending on the X-ray reader (Trapido et al. 1998; Steen et al. 1997). More recently, Churchyard et al. reported prevalences of 18.3% to 19.9% in employed black miners older than 37 years (Churchyard et al. 2004). Girdler-Brown et al. found a prevalence of 24.6% in black ex-miners with a mean employment duration of 26 years (Girdler-Brown et al. 2008). The high prevalence of 51.6% in white miners, calculated by Murray and Hnizdo, was for a cohort of deceased miners employed for an average of 40 years (Murray and Hnizdo, unpublished data).

The frequency distributions of the miners in the different age categories and duration of employment categories were very different in the two population groups. This may be why the attempt at modeling the data as a single data set with an added dichotomous variable for population group was unsuccessful. Having two separate regression models for the two population groups, each with somewhat different age groups and duration of employment groups, means that the effect of population group on the proportion with silicosis is difficult to discern from the regression results.

We investigated this issue by performing age group and duration group direct standardization of the proportions of black and white miners with silicosis. After standardizing for these factors, the proportion of black miners with silicosis was almost double (1.7-fold) that of white miners for the overall study period and for those who died from external causes. The difference was even greater for those who died from nonexternal causes (2.27-fold).

These findings lend support to our belief that the inclusion of data from the miners who died from nonexternal causes would have biased our findings. The HIV epidemic has resulted in an increase in tuberculosis and an associated increase in mortality; those with tuberculosis are more likely to have silicosis than those without (Corbett et al. 2000).

The higher proportions of black miners with silicosis, after adjusting for age and duration of employment, suggest that black miners had higher intensities of exposure to silica than white miners. Generally, black miners are employed as drillers and stopers, and these are the processes that generate the most dust. White men, on the other hand, are exposed to less dust in their jobs as supervisors and maintenance workers. This is supported by the fact that the proportion of black miners with silicosis reached 0.2 after shorter durations of employment than for white miners. In addition, a much higher proportion of black miners had silicosis at younger ages than did white miners. The proportion of black miners younger than 50 years with silicosis was more than double that of white miners in the same age group.

In addition to the high proportion of miners with silicosis, this study shows that the proportions in black miners have been rising since the mid-1970s. The proportion of black miners with silicosis increased more than 10-fold during the study period. This increase can be partly attributed to

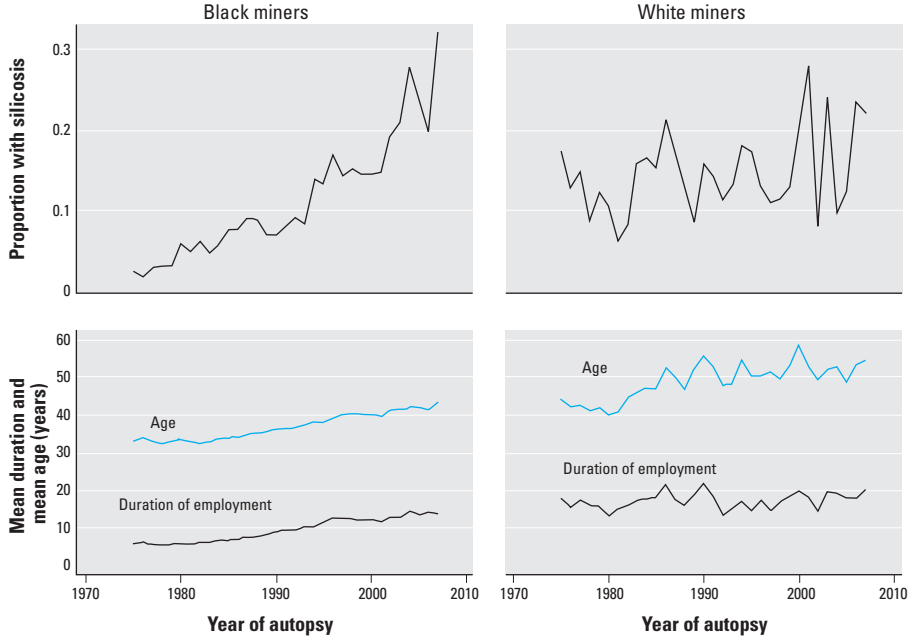


Figure 1. Crude population group-specific proportions of gold miners with silicosis (external causes of death): mean ages and mean employment durations from 1975 to 2007 (n = 16,411 for black miners and n = 2,732 for white miners).

increasing age at autopsy and longer employment periods, both of which may be direct consequences of workforce stabilization, a socioeconomic phenomenon that started in the 1980s. In the 1970s, when the computerized autopsy database was established, the system of employing miners on short contracts was in force. Gradually, economic changes led to increased poverty in the rural areas and fewer opportunities for alternative employment. Stabilization of the workforce increased (Cowie and van Schalkwyk 1987; Leger 1992), and more black miners became career miners. Before the mid-1980s, black miners with tuberculosis were discharged and repatriated to the rural areas and neighboring countries after being started on treatment, to reduce the number of ill miners in the workforce. When short-course chemotherapy for tuberculosis was introduced into the gold mines, they were allowed to continue working (Cowie 1989), and this also resulted in longer employment periods. In this study, the mean age at autopsy among black miners increased by > 10 years, from 33 to 43.4 years, whereas mean duration of employment increased by almost 8 years, from 5.6 to 13.4 years.

Year of autopsy is the year in which the pathologic examination was performed and correlates with the year of death. Because year of autopsy remained a significant predictor of silicosis among black miners when we adjusted the data for duration of employment and age at death, it is likely that there are other factors that we cannot measure that contribute toward this increase. As technology advanced over the years, and higher speed rock drills were developed, it is possible that the dust particles that were generated became smaller. It is also possible that, as mine shafts became deeper, the surface properties and hence the toxicity of the dust increased, or there may have been an increase in the proportion of freshly fractured silica dust. The gradual introduction of production bonuses may also have resulted in men working for longer shifts, or for more shifts each year, which would not be reflected in the duration variable that measures exposure in calendar months.

Limiting the study population to those who died from external causes did not change the results with regard to the trends during the study period. When we included all causes of death, the crude proportions of black miners increased from 0.03 in 1975 to 0.34 in 2007. In those dying of external causes, these proportions were very similar (0.03 and 0.32, respectively).

Some miners in both population groups developed silicosis within 10 years of employment. This is likely to be accelerated silicosis, which usually occurs within 10 years of dust exposure. It is not acute silicosis (silica-associated alveolar proteinosis); we have seen only a single case in the last 25 years, and this was in a nonminer.

The increase in ORs was more strongly associated with age than with duration of employment. It is well established that silicosis progresses even after cessation of dust exposure (Hessel et al. 1988). The high OR in older white men, in particular, may be due to longer residence time of dust in the lungs.

The strengths of the present study include that it is population-based and that the study population comprises around 19,000 gold miners for whom data were collected for a period of more than 30 years. In addition, the diagnosis of silicosis was made at autopsy, rather than radiologically, by experienced pathologists, using standardized methods. Autopsies are useful for diagnosing diseases such as silicosis that may be undetected on X rays. Corbett et al. (1999) found radiology to be insensitive for detecting early silicosis, using miniature X rays. Hnizdo et al. (1993), using standard sized films, also showed lower radiologic sensitivity compared with autopsy findings, even for advanced silicosis.

There are several potential biases in the data. One is that there is a difference between black and white miners with regard to the use of the autopsy service. Most white men who die while employed or after having left the mining industry come to autopsy. The proportion of white gold miners coming to autopsy is high, at more than 80%. In 1986, Hessel et al. (1986) estimated that 86% of deceased white gold miners undergo autopsy. This is supported by a cohort study in which around 1,700 gold miners who had started working in the 1940s were followed up for several decades (Murray and Hnizdo, unpublished data). By 2003, 83% of the deceased men had had an autopsy.

The rate is also high for black miners who died while employed. Corbett et al. (1999) reported that 94% of gold miners, employed by a single company, who died in the period January 1996 to June 1997 came to autopsy. In a more recent study on causes of death in a large cohort of black gold miners, "70% of the deaths occurring in the mine were followed by autopsy" (Murray et al. 2007). Very

Table 6. Adjusted ORs for black miners (external causes of death).

Variable	*n*	OR	SE	*z*-Value	*p*-Value	95% CI
Age group (years)						
< 30	6,113	Reference				
30–39	5,921	7.40	1.26	11.74	< 0.001	5.30–10.34
40–49	3,315	20.25	3.45	17.65	< 0.001	14.50–28.29
≥ 50	1,062	22.08	4.03	16.94	< 0.001	15.43–31.58
Duration of employment (years)						
< 10	11,683	Reference				
10–14	2,815	1.93	0.15	8.37	< 0.001	1.65–2.25
15–19	1,284	2.54	0.23	10.18	< 0.001	2.12–3.03
≥ 20	629	3.06	0.34	10.18	< 0.001	2.47–3.80
Year of autopsy						
1975–1979	3,167	Reference				
1980–1984	3,795	2.02	0.27	5.35	< 0.001	1.56–2.61
1985–1989	4,205	2.43	0.30	7.12	< 0.001	1.90–3.10
1990–1994	2,968	1.90	0.25	4.98	< 0.001	1.48–2.45
1995–1999	1,321	2.27	0.32	5.88	< 0.001	1.73–2.99
2000–2007	955	2.94	0.42	7.60	< 0.001	2.22–3.88

Area under the ROC curve = 0.82. Hosmer-Lemeshow goodness-of-fit test *p*-value = 0.36 (10 groups), 0.13 (8 groups), and 0.35 (12 groups). Pearson's goodness-of-fit test *p*-value = 0.01; when performing the Pearson goodness-of-fit test, 25 of 168 (14.9%) of the expected cell values were < 5, so the Hosmer-Lemeshow test result is preferred.

Table 7. Adjusted ORs for white miners (external causes of death).

Variable	*n*	OR	SE	*z*-Value	*p*-Value	95% CI
Age group (years)						
< 50	1,555	Reference				
50–59	519	2.62	0.48	5.27	< 0.001	1.83–3.75
60–69	378	5.41	1.00	9.13	< 0.001	3.76–7.77
≥ 70	280	6.07	1.21	9.04	< 0.001	4.11–8.97
Duration of employment (years)						
< 10	978	Reference				
10–14	362	3.62	1.12	4.17	< 0.001	1.98–6.62
15–19	320	5.30	1.55	5.69	< 0.001	2.98–9.42
20–24	306	8.13	2.28	7.47	< 0.001	4.69–14.08
25–29	265	10.37	2.91	8.35	< 0.001	5.99–17.97
30–34	260	11.98	3.37	8.84	< 0.001	6.91–20.78
≥ 35	241	6.71	1.95	6.55	< 0.001	3.79–11.85

Year of autopsy groups is not shown because all ORs were not statistically significant. Area under the ROC curve = 0.82. Pearson goodness-of-fit test *p*-value = 0.38; Hosmer-Lemeshow goodness-of-fit test *p*-value = 0.79 (10 groups), 0.62 (8 groups), and 0.46 (12 groups); when performing the Pearson goodness-of-fit test, 5 of 54 (9.3%) of the expected cell values were < 5.

few men in this study who died elsewhere came to autopsy (7%).

Often, black miners who retire or leave the mining industry for other reasons return home to the rural areas of South Africa and neighboring countries from where they were recruited, often far from medical services that are able to remove the cardiorespiratory organs for autopsy. Miners who develop disabling silicosis while employed are not allowed to continue to work in dusty jobs, and so they leave the mines. Thus, the missing black miners may have had more disease (as a proportion) as a result of heavier or longer exposures (healthy worker effect). Because elderly, retired black miners seldom come to autopsy, the black miners are generally younger at autopsy than are the white miners. In this study, few autopsies had been conducted on black miners who were older than 59 years or who had worked for > 24 years.

These biases will underestimate the proportion of black miners with silicosis. The NIOH has an outreach program that addresses miners' rights regarding autopsy examination and ascertaining compensation for occupational lung disease. This program has been extended to the rural areas of South Africa and neighboring countries.

The main limitation in this study was the unavailability of dust measurements and the use of proxy measurements of age at autopsy and duration of employment to estimate cumulative dust exposures.

There is no evidence that silica dust levels have decreased in the mines over the past few decades. In 1994, the Commission of Inquiry into Safety and Health in the Mining Industry, led by Judge R.N. Leon, concluded that "dust levels have remained roughly the same over a period of about 50 years" (South African OHS Commissions 1995). There is no reason to believe that they have decreased since the commission's inquiry (Churchyard et al. 2004).

The increasing proportion of black gold miners with silicosis at autopsy has several implications. Prevalences of diseases associated with silicosis and silica dust exposure, such as tuberculosis, chronic obstructive airway disease (COAD), and lung cancer, are also likely to rise. No data exists on trends in lung cancer or in COAD for South African gold miners, but the burden of COAD in gold miners is high (Girdler-Brown et al. 2008). The most important impact, however, is the prevalence of tuberculosis among black miners, which is increasing (Sonnenberg et al. 2000). The HIV epidemic has played a major role in the rising prevalence of tuberculosis but has been exacerbated by the presence of silicosis, because silicosis and HIV have a multiplicative effect on tuberculosis (Corbett et al. 2000).

Conclusion

Our analyses show that, during the 33-year study period, there has been no reduction in the proportion of miners (with external causes of death) coming to autopsy with pathologic evidence of silicosis after adjustment for age group and duration of employment. Furthermore, the standardized proportion of black miners with silicosis is almost double that of white miners during this period. The large proportion of men with silicosis reflects the inability of gold mining companies to reduce silica dust to safe levels. As miners age and work for longer periods, the burden of silicosis and its associated diseases will continue to rise. HIV, tuberculosis, and silicosis have a multiplicative interaction. Thus, the high proportion of South African gold miners with silicosis, many of whom are already burdened with tuberculosis and/or HIV, has far reaching implications in terms of health services that need to be prepared for increasing morbidity and mortality rates in both current and ex-miners. South Africa is committed to global efforts to eliminate silicosis by 2030, but this will require extraordinary efforts. The recording of valid and reliable dust measurements linked to ongoing medical surveillance is essential for success.

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APPENDIX 2

Rees D, Murray J, Nelson G, Sonnenberg P. Oscillating Migration and the Epidemics of Silicosis, Tuberculosis, and HIV Infection in South African Gold Miners. *Am J Ind Med* 2010; 53:398-404.

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Oscillating Migration and the Epidemics of Silicosis, Tuberculosis, and HIV Infection in South African Gold Miners

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Background *Hundreds of thousands of men from rural areas of South Africa and neighboring countries have come to seek work in the gold mines. They are not immigrants in the usual sense as they work for periods in the mines, go home, and then return. This is termed oscillating or circular migration. Today we have serious interrelated epidemics of silicosis, tuberculosis, and HIV infection in the gold mining industry.*

Methods *This article discusses the role of oscillating migration in fuelling these epidemics, by examining the historical, political, social, and economic contexts of these diseases.*

Results *The impact of silicosis, tuberculosis, and HIV infection extends beyond individual miners to their families and communities.*

Conclusion *Failure to control dust and tuberculosis has resulted in serious consequences decades later. The economic and political migrant labor system provided the foundations for the epidemics seen in southern Africa today.* Am. J. Ind. Med. 53:398–404, 2010.
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KEY WORDS: circulatory migration; gold mining; migrant labor; South Africa

INTRODUCTION

Each year, for well over a century, hundreds of thousands of people have come to South Africa from neighboring countries seeking work in mines, farms, and factories [Crush et al., 2005]. Although these people serve a large variety of industries, gold mining has historically been a major recruiter of these migrant workers and is the industrial sector with the most data on employment and disease rates. Thus, while gold mining is by no means unique, it is a good setting to examine the impact of this type of employment on aspects of occupational health.

The migrant labor system in southern Africa was born with the discovery of gold on the Witwatersrand in 1886, leading to a large demand for cheap, unskilled labor. Gold mining is labor-intensive and, consequently, recruitment has been on a massive scale. Close to 200,000 people were employed in the industry in the 1920s and about half a million in the mid-1980s and early 1990s [Harington et al., 2004]. Thereafter, employment declined fairly dramatically to approximately 160,000 by 2006 [Chamber of Mines of South Africa, 2006]. Large proportions of these mineworkers have been foreign migrants; 40–60% of gold miners were from neighboring countries from 1911 to the 1990s [Harington et al., 2004]. Even in modern times, a large number of gold miners are from outside South Africa, as shown in Table I.

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TABLE I. Number of Foreign Workers Employed by South African Gold,* Platinum, and Coal Mines 1996–2007

	Country				
Year	**Swaziland**	**Mozambique**	**Lesotho**	**Botswana**	**Total**
1996	14,371	55,022	81,357	7,932	158,682
1997	12,960	55,027	76,360	7,536	151,883
1998	10,338	52,011	60,450	6,223	129,022
1999	9,307	46,890	52,436	5,130	113,763
2000	8,160	44,014	51,351	4,343	107,868
2001	7,794	45,254	49,599	3,651	106,298
2002	8,587	50,589	54,390	3,551	117,117
2003	7,885	52,205	54,202	4,246	118,538
2004	7,521	48,099	48,437	3,923	107,980
2005	6,878	46,256	43,693	3,257	100,084
2006	7,124	46,709	46,082	2,992	102,907
2007	7,099	44,879	45,608	2,845	100,431

Source: TEBA (The Employment Bureau of Africa Limited), May 2008.
*Roughly 80% employed by gold mines.

These workers have not been immigrants in the usual sense: the apartheid political system and, latterly, immigration policies, made permanent immigration illegal or very difficult. The 1913 Natives' Land Act which limited access to land and the imposition of taxes ensured that men would come to urban areas to earn a cash wage. Restriction of movement, legislated under Apartheid, meant that workers could obtain contracts (lasting around 9 months) but their families had to remain at home in the rural areas of South Africa or in their countries of origin. At the end of their contracts the men were made to return home, which they did for variable periods before returning to the mines to renew their contracts. However, they often worked on the gold mines for only a few years in total. More recently, employment has become more permanent but still workers oscillate between work on the mine and periods at home. This kind of immigration has been termed oscillating or circular migration and is not limited to mining [Collinson et al., 2006].

Oscillating migration produces particular social circumstances. Housing while at work is usually in single-sex compounds or hostels. The vast majority of miners are men and, although miners may be married and have families at home, they may live, in effect, as single men for much of their working lives, or they may have an informal family near their place of work [Lurie, 2000]. Thus, men live strangely distinct dual lives. Hostels were the norm for gold miners (98% of black miners on Anglo American mines lived in hostels in 1987 and 89% in 1993) [Crush, 1997] and, even now, this type of accommodation is common. At the end of 2007, 51% of South African employees of AngloGold Ashanti West Wits gold mines were housed in residences (communal hostels) compared to 62% in 2003 [AngloGold Ashanti, 2007].

In this article, we discuss the role of oscillating migration on three interrelated epidemics of occupationally related diseases in gold miners: silicosis, tuberculosis (TB), and HIV infection. The evidence that migration has increased disease rates in gold miners is empiric, intuitive, and by inference from lower rates in other industries with less reliance on migrant labor. In order to appreciate the trends in the incidence of silicosis, TB, and HIV infection, and the role of oscillating migration, it is important to look at the historical, political, social, and economic contexts of these diseases.

SILICOSIS

Silicosis is still very common on South African gold mines. The prevalence of silicosis in a recent cross-sectional study of 520 gold miners (with mean length of service of 21.8 years) was found to be 18.3–19.9%, according to two X-ray readers [Churchyard et al., 2004]. The prevalence shows good concordance with the 24.6% found in 624 gold miners examined 18 months after cessation of employment with mean length of service of 25.6 years [Girdler-Brown et al., 2008].

Many reasons may be posited for the high levels of silicosis in gold miners. The silica (quartz) is freshly fractured by drilling, blasting, and moving rock, and the particles are probably very small [Phillips, 2007] and largely uncontaminated by associated clays: all factors thought to increase the fibrogenicity of silica [Health and Safety Executive, 2002]. Deep-level mining in constantly changing workplaces introduces difficulties for dust control.

The high level of migrancy has also played a role in escalating disease rates. Migrancy has reduced the ascertainment of compensable disease, externalized the costs of attending to occupational disease to remote labor-sending areas, and diluted community and organized-labor pressures on management that one would expect with such high levels of occupational disease. If gold mines recruited labor from local communities, the surrounding towns and villages would have substantial numbers of miners with occupational disease. Local community awareness would be high, there would be ready access to mine occupational health services, and mine management and trade union officials would live among the diseased employees and their families. Oscillating migration means that this is not the case. On the contrary, the problem is far away. A quote from a mine manager made during an interview for a research project on mine managers' attitudes to silicosis is illustrative of the lack of urgency brought about by distance: ''Actually, by the time you [the miner] are retired and are now graded [compensated] first, second or third degree . . ., it doesn't matter because you

TABLE II. Silicosis Prevalences and Compensation Data in Former Gold Miners on South African Mines

Sources	Location	Silicosis prevalence by reader[a]	Workers' compensation
Trapido [1998a, 1998b]	Eastern Cape, South Africa	22–37%	62% of those eligible not compensated
			35% compensated partially (disease had progressed)
			2.5% fully compensated
Steen [1997]	Thamaga, Botswana	26–31%	Many previously undiagnosed[b]
			Very few had been compensated[b]

[a]Two X-ray readers in study.
[b]Actual percentages not provided.

are not in my face anymore'' [Page-Shipp et al., 2006]. Separation from affected individuals, communities, and their problems surely reduces pressures to control dust.

Two studies, in Botswana [Steen et al., 1997] and the Eastern Cape province of South Africa [Trapido et al., 1998a], demonstrate the high rates of silicosis in former gold miners living in remote labor-sending areas and significant failure to provide workers compensation (Table II). Assuming similar disease and compensation failure percentages in the total population of former miners as in the Eastern Cape sample results in an estimated 288,000 cases of compensable pneumoconiosis and an estimated 10 billion Rand (ZAR) in unpaid compensation liability at 1998 values; much more in today's terms [Trapido et al., 1998b]. White [1997] estimated that 96,200 compensable silicosis cases in gold miners were not reported from 1973 to the late 1990s. Trapido et al. [1998a] summarized this well: ''A combination of natural disease latency and social, political and economic factors associated with labor migrancy have resulted in an externalizing of occupational disease costs away from the mining industry.'' The consequence of this unpaid cost—and all the medical costs associated with occupational disease that are not borne by employers because ill workers are in other countries or far away—is reduced financial incentive to control dust.

The rate of silicosis has been affected by changes in the nature of the mine labor system since 1975, a process termed ''stabilization'' [Leger, 1992]. Socio-economic factors, such as the decline in the southern African economy, deteriorating conditions in rural areas, prolonged drought, and contraction of the mining industry, led to a decreased turnover of the workforce. In addition, exposure, in terms of the total number of years spent on the mines, has increased, leading to a rising trend in rates of silicosis in the industry [Murray et al., 1996]. In South Africa, deceased miners are entitled to an autopsy examination to determine eligibility for compensation, regardless of the clinical cause of death. These autopsies are done at the Pathology Division, National Institute for Occupational Health (NIOH) in Johannesburg, and the findings constitute an important database, allowing examination of trends in disease rates over time. The total number of years spent on the gold mines has increased from an average of 6–16 years since 1975 for black men coming to autopsy at the NIOH, with a corresponding increase in rates of silicosis from 3% to 34%.

TUBERCULOSIS

The gold mines in South Africa have among the highest TB incidence rates in the world. TB prevalence has increased steadily, from 806/100,000 in 1991, to 1914/100,000 in 1998, to 3,821/100,000 in 2004 [Glynn et al., 2008]. TB acquired in the mines has the potential to fuel TB transmission in home regions through oscillating migration. Once TB was introduced onto the gold mines it spread rapidly to communities through the migrant labor system.

It has been known for a very long time that silicosis increases the risk of TB, and increased rates of the disease have been found in South African gold miners with pneumoconiosis [Cowie, 1994] and, importantly, in those exposed to silica without silicosis [Hnizdo and Murray, 1998]. The Miners' Phthisis Act of 1912 recognized TB (as well as silicosis) as an occupational disease because of its association with silica dust exposure. HIV is also a strong risk factor for TB as discussed later in this article.

Prior to the advent of gold mining in South Africa, TB was rare among black southern Africans [Collins, 1982]. The miners from Europe came from communities where TB was common and many arrived in South Africa already infected, albeit latently, with *Mycobacterium tuberculosis*. The mine living conditions and high dust exposure resulted in many of these men developing active TB and transmitting it to black miners. A study by Maynard in 1912 found a higher prevalence of infection in men who had previously been employed in the mines (18%) than in new recruits (2.4%) (cited in a 1932 report of the Tuberculosis Research Committee) [South African Institute for Medical Research, 1932].

Black mineworkers who developed TB had their contracts terminated and were repatriated to the neighboring countries and rural areas from whence they came, thus transmitting TB to the rural areas of southern Africa [Collins, 1982]. Even when anti-TB chemotherapy was discovered in the 1950s, government regulations still did not permit black miners to work underground if they had TB and they continued to be sent home where they were unable to complete the required 18 months of therapy. Health services in rural areas were, and continue to be, rudimentary and under-resourced. These infected men, now unemployed, thus added the risk of TB to the poverty of their families and communities [Cowie et al., 1989]. In the 21st century, TB continues to be transmitted within the mines as demonstrated by a recent study using DNA fingerprinting, where at least 50% of the TB could be attributed to ongoing transmission within the mining community and, in 85% of patients in clusters, transmission could be explained by current or past mine employment [Godfrey-Faussett et al., 2000].

In 1977, a study showed that men with silicosis responded as well to short-course anti-TB chemotherapy as those without silicosis [Escreet et al., 1984]. This led to a change in practice and, since 1983, black miners with TB have been permitted to continue to work underground. Although this provided continued employment, it had the unforeseen consequence of increasing the rate of TB, in the same way that stabilization increased the rate of silicosis. Murray et al. [1996] reported an increase in the prevalence of pulmonary TB at autopsy in men who died of trauma, from 0.9% in 1975 to 3.9% in 1991, before the HIV epidemic.

HIV INFECTION

The 1990s saw a dramatic increase in HIV prevalence in South African gold miners. In 1987, 0.03% of mineworkers from areas other than Malawi (seroprevalence 4%) tested HIV positive [Brink and Clausen, 1987]. This increased to 1.3% in 1990 [Petschel et al., 1993], rising to an estimated prevalence of 24% in 1999 [Charalambous et al., 2001] and reaching 27% in 2000 [Corbett et al., 2004].

There is a large body of literature linking HIV, mobility, and migration in southern Africa [Lurie, 2000; Williams et al., 2002; Lurie et al., 2003; Clark et al., 2007; Jochelson et al., 1991; Hargrove, 2008].

The South African gold mining community, with its migrant population and single-sex hostels, was the ideal environment for any sexually transmitted pathogen to find a niche. HIV was introduced into a population that already had high rates of other sexually transmitted diseases [Jochelson et al., 1991], which enhance HIV transmission. Miners spend long periods separated from their wives and families, and the mine setting provides few opportunities for stable intimate social relationships [Campbell, 1997]. Conditions in the mines, including working under dangerous conditions, make it more likely that men will engage in high-risk sexual behavior [Campbell, 1997]. In addition, the migrant labor system creates a market in which vulnerable women, out of economic necessity, need to support themselves and their children through commercial sex work [Jochelson et al., 1991]. There is a complex geographic network of social and sexual relationships, which extends from the heterosexual mining community, to the surrounding communities, to nearby urban areas, and to rural areas [Jochelson et al., 1991]. Oscillating migration influences HIV spread by creating a social and economic system that encourages multiple partners and concurrent partnerships. Oscillating migration, involving these complex sexual networks, would inevitably result in an HIV epidemic extending to the entire region [Chirwa, 1997].

The impact of oscillating migration to gold mines on HIV infection has been shown in a study in two adjacent districts of KwaZulu Natal province in South Africa [Lurie et al., 2003]. This cross-sectional study of 196 migrant and 64 non-migrant men and their partners found that 25.9% of migrant men and 12.7% of non-migrant men were HIV positive (OR 2.4), a significant difference. Multivariate analysis showed migration to be an independent risk factor for HIV infection. The migrant men worked in two locations: gold mines in Carletonville (700 km from their homes) and Richards Bay (less than 100 km away). Almost all the gold miners lived in single sex hostels. HIV prevalence among Carletonville miners was 28.7% compared to 22.4% in Richards Bay [Coffee et al., 2007].

The gold mining industry is by no means unique in having high HIV prevalence rates. Industries that rely on migrant labor (mining, heavy engineering, and metal processing) and the transport sector appear to have the highest burdens (Table III), above the national average of 18.8% in the adult population (15–49 years) [Department of Health, 2006]. About 5.54 million people were estimated to be living with HIV in South Africa in 2005 [Department of Health, 2006].

Many factors influence the HIV burden in individual workforces: background rates in the population supplying the workers and around the workplace; year of ascertainment of the HIV status; socio-economic circumstances; and age, sex, and race profiles, being some. Therefore, comparing HIV prevalences among workforces is problematic. HIV cannot be modeled for a particular company purely on the basis of knowing the demographic structure of the workforce [Colvin et al., 2007]. However, in concluding his inaugural address on the determinants of the HIV pandemic in southern Africa—Migration, Mines and Mores—Hargrove [2008] stated that it is tempting to suggest that the high incidence of HIV infection has an underlying source: the breakdown of family structure associated with oscillating migration.

TABLE III. HIV Prevalence in Selected Workforces in South Africa

Source	Sector	HIV prevalence[a]	Number of workforces
Colvin et al. [2007]	Public	7.4–9.7	2
	Utility/parastatal[b]	7.4–15.7	4
	Private	4.9–20.0[c]	13
Corbett et al. [2004]	Gold mining	27%	1
Stevens et al. [2006]	Platinum mining	24.6%	N/A
Evian et al. [2004]	Mining	15.5%	
	Metal processing	17.8%	
	Manufacturing	13.0%	
	Other	11.6%	
Shisana [2005]	Educators	12%	
Ramjee and Gouws [2002]	Truck drivers	56%	N/A

[a]Colvin et al. [2007] adjusted prevalences.
[b]Excludes transport workers at 24.3%.
[c]20% in heavy engineering.

THE INTERACTION OF SILICOSIS, TUBERCULOSIS, AND HIV INFECTION

It is well known that HIV infection increases the incidence of TB, both through the increased risk of reactivation of latent *M. tuberculosis* infection and through more rapid progression from infection to disease. Combining HIV and silicosis has an even larger effect on TB risk because there is a multiplicative interaction [Corbett et al., 2000]. So, TB remains an occupational disease in the era of HIV. Results from autopsy examinations at the NIOH (Fig. 1) show the extraordinary increase in active TB from 1975 to 2007.

An added dimension is that the increase in TB in settings with high rates of HIV infection results in an increase in the infectious pool, thereby increasing the risk of infection in HIV-negative people. This has been supported by longitudinal studies in the gold mines which have shown that, in addition to dramatic increases in TB in HIV-positive miners, there has been increased incidence in HIV-negative miners [Sonnenberg et al., 2005; Glynn et al., 2008]. Particularly at risk from this ongoing transmission of TB are spouses and families of miners, who are most likely to be co-infected with both TB and HIV.

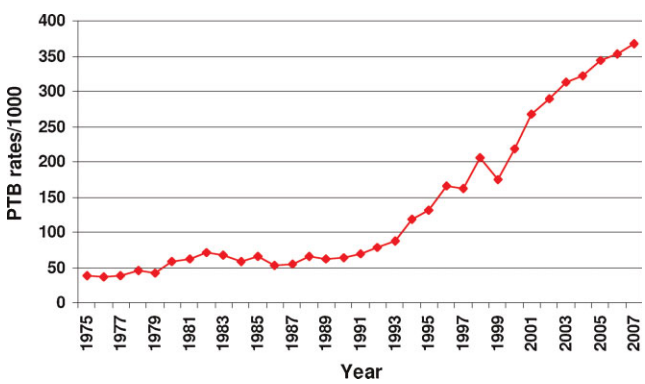


FIGURE 1. Active pulmonary tuberculosis at autopsy in black miners, all commodities, 1975–2007. Source: PATHAUT database, March 22, 2007, Pathology Division, National Institute for Occupational Health, Johannesburg.

CONCLUSION

Cross-border and rural-urban migration are international phenomena. Southern Africa is characterized by high levels of job-seeking migrant workers as mines and other large-scale industries are generally not located close to towns or cities from which large numbers of workers can be recruited. Migrant labor is a significant source of income and even survival for many southern Africans [Crush et al., 2005]. Recommending a sudden end to migration ignores substantial logistical and political impediments and is likely to result in both predictable and unforeseen consequences. Notwithstanding this reality, interventions are needed to ameliorate the negative aspects of migration.

Migration patterns in southern Africa include both cross-border and internal migration. At a macro level, multi-level regional and national policies are therefore needed which acknowledge the economic value of these individuals and which provide for infrastructure, such as strengthening health services both at locations where migrant workers are employed or live, and in labor-sending areas. General health services in labor-sending areas are over-stretched by returning migrants [Clark et al., 2007] and need resources if they are to diagnose and manage occupationally related diseases. These services would benefit from support by companies employing people from local communities, and

there are some initiatives in this regard [Chamber of Mines, 2008]. In particular, it is important to establish cross-border referral systems to ensure continuity of treatment of TB. Public and health-care provider awareness regarding TB should be promoted throughout the SADC region through the development of educational materials, and media campaigns.

Although much has been published on the linkages between migrancy and health, the mining industry itself seems to lack awareness of the seriousness and long-term implications of the matter. Occupational health professionals need to actively engage management on these issues; oscillating migration is a risk factor for work-related disease and thus falls within the ambit of occupational health services. Preventing disease in migrant miners would include alternatives to single-sex hostels, and thus the reduction of HIV infected transmission, controlling exposure to silica dust, and isoniazid preventive treatment to reduce TB in miners with silicosis.

Access to workers' compensation by cross-border migrants who have returned to their home countries is subject to many hurdles and both case-finding surveys and compensation advice and support offices in these areas may assist former miners with occupational disease to get workers' compensation.

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APPENDIX 3

Nelson G, Murray J, Phillips J. Asbestos-related diseases in diamond mine workers The risk of asbestos exposure in South African diamond mine workers. Ann Occup Hyg 2011; 55(6):569–77.

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The Risk of Asbestos Exposure in South African Diamond Mine Workers

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Objectives: Asbestos is associated with South African diamond mines due to the nature of kimberlite and the location of the diamond mines in relation to asbestos deposits. Very little is known about the health risks in the diamond mining industry. The objective of this study was to explore the possibility of asbestos exposure during the process of diamond mining.

Methods: Scanning electron microscopy and energy-dispersive X-ray spectroscopy analysis were used to identify asbestos fibres in the lungs of diamond mine workers who had an autopsy for compensation purposes and in the tailings and soils from three South African diamond mines located close to asbestos deposits. The asbestos lung fibre burdens were calculated. We also documented asbestos-related patholological findings in diamond mine workers at autopsy.

Results: Tremolite–actinolite asbestos fibres were identified in the lungs of five men working on diamond mines. Tremolite–actinolite and/or chrysotile asbestos were present in the mine tailings of all three mines. Mesothelioma, asbestosis, and/or pleural plaques were diagnosed in six diamond mine workers at autopsy.

Conclusions: These findings indicate that diamond mine workers are at risk of asbestos exposure and, thus, of developing asbestos-related diseases. South Africa is a mineral-rich country and, when mining one commodity, it is likely that other minerals, including asbestos, will be accidentally mined. Even at low concentrations, asbestos has the potential to cause disease, and mining companies should be aware of the health risk of accidentally mining it. Recording of comprehensive work histories should be mandatory to enable the risk to be quantified in future studies.

Keywords: asbestiform; autopsy; lung fibre burden; naturally occurring asbestos; PATHAUT

INTRODUCTION

South Africa is a major producer of diamonds and gold and the leading producer of other commodities such as platinum, chrome, manganese, and vanadium. It was the third largest producer of asbestos, which was mined from the late 1800s to 2002. While much is known about the health effects of exposure to dust generated by the mining of gold, asbestos, and coal, little research has been done in other commodities, including diamonds. Unlike many other types of mining, diamond mining uses no toxic chemicals and produces no chemical pollutants; there are no documented ill-health effects of diamond mining.

Diamond miners work in mineral complex environments. Kimberlite, in which diamonds are found, also contains olivine, phlogopite, calcite, serpentine, diopside, monticellite, apatite, perovskite, and ilemenite (Wilson and Anhaeusser, 1998). Kimberlite also often contains fragments of ultramafic rocks, such as peridotite and eclogite, which are

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formed under very high pressure; and xenocrysts such as pyrope garnet, picro-ilmenite, chromium spinel, and chrome diopside (Wilson and Anhaeusser, 1998). Eclogites are susceptible to metamorphism, by which they form both calcic amphibole rock types, such as tremolite and actinolite; and glaucophane, a sodic amphibole with a chemical makeup similar to crocidolite (Leake *et al.*, 1997). Both chrysotile and amphibole asbestos fibres (primarily tremolite) have been described in association with kimberlite (Wagner and Reinecke, 1930; Wagner, 1971; Aoki, 1972). [P. A. Wagner was the father of J. C. (Chris) Wagner who wrote the seminal 1960 paper (Wagner *et al.*, 1960) on the association of mesothelioma and exposure to crocidolite asbestos.]

Diamond mine workers in South Africa are thus at risk of asbestos exposure for two reasons. Firstly, diamonds are found in kimberlite, an ultramafic rock (Wilson *et al.*, 2007) in which amphibole minerals also occur. Ultramafic rocks have a low silica and high magnesium and iron content and often contain asbestos (Anhaeusser, 1976; Pan *et al.*, 2005; Perkins *et al.*, 2008). Secondly, many of the diamond mines are in close proximity to asbestos deposits, both those that have been mined and those that were not commercially viable.

Health research has focused on the three main commercial types of asbestos (chrysotile, crocidolite, and amosite), all of which were mined in South Africa. The health effects of other asbestiform amphiboles, including tremolite, actinolite, richterite, winchite, and anthophyllite, have been less researched, although there is strong evidence that they, too, cause disease (Price, 2008; Phillips and Murray, 2010).

Tremolite and/or actinolite asbestos fibres have been described in talc mines in various countries (Gamble and Gibbs, 2008; Loyola *et al.*, 2010) and limestone and dolomite mines in Finland (Juntilla *et al.*, 1996). Amosite asbestos was reported in an iron ore mine in the USA (Nolan *et al.*, 1999). The most well-known occurrence of asbestos-contaminated ore and subsequent health effects is the vermiculite mine in Libby, Montana in the USA, which contained tremolite–actinolite, richterite, and winchite asbestos fibres (Price, 2008; Whitehouse *et al.*, 2008). Tremolite asbestos has also been reported in a South African vermiculite mine (Hessel and Sluis-Cremer, 1989).

There are diamond deposits in Limpopo province, close to chrysotile, anthophyllite, and crocidolite asbestos deposits (Fig. 1); in Gauteng near chrysotile deposits; in North West province near amosite deposits; and in the Northern Cape near crocidolite deposits. Of these, only the Northern Cape asbestos deposits were mined. The only operational kimberlite diamond mines are in Limpopo, Gauteng, and Northern Cape. There are no kimberlite diamond mines located near the amosite asbestos deposits in the North West province or near the tremolite deposits.

One of the larger South African diamond mines is located in the Asbestos Hills in the Northern Cape, and crocidolite asbestos was previously identified around the perimeter of the kimberlite pipe (P. J. Jordaan, unpublished data). Asbestos fibres (chrysotile, anthophyllite, and tremolite–actinolite) have also been previously identified in air samples from five unnamed South African diamond mines (Unsted and Jansen van Vuuren, 2001).

In this paper, we explore the possibility of asbestos exposure during the process of diamond mining by examining the lungs of deceased diamond mine workers for asbestos fibres, identifying asbestos fibres in the tailings and soils of diamond mines and documenting respiratory disease in diamond mine workers.

Analysis of asbestos fibres in lungs is a way to estimate lifelong exposure to asbestos, especially amphibole fibres which are retained in the lungs to a greater degree than chrysotile fibres. Although the correlation between lifetime cumulative exposure and fibre concentration in the lungs is not exact, the findings from lungs may give a better estimation of exposure than even a careful retrospective analysis of the patient's history, at least in low-grade exposure (Hillerdal, 1999).

METHODS

Data sources

Anyone who has ever been employed in a South African mine has the right to have his/her cardiorespiratory organs examined for compensable disease, regardless of the cause of death and provided that the next-of-kin agrees (Republic of South Africa, 1973). The autopsies are performed at the National Institute for Occupational Health (NIOH) in Johannesburg. The findings of these examinations, as well as the commodities in which the person worked and the number of years for which he was employed in each, and demographic data, are recorded in the PATHAUT (Pathology Automation) database (Hessel *et al.*, 1987a,b). All histological material is archived, and whole lungs from miners of specific commodities or with specific diseases have been stored since 2001. This database was used to identify, and collate information on, diamond mine workers.

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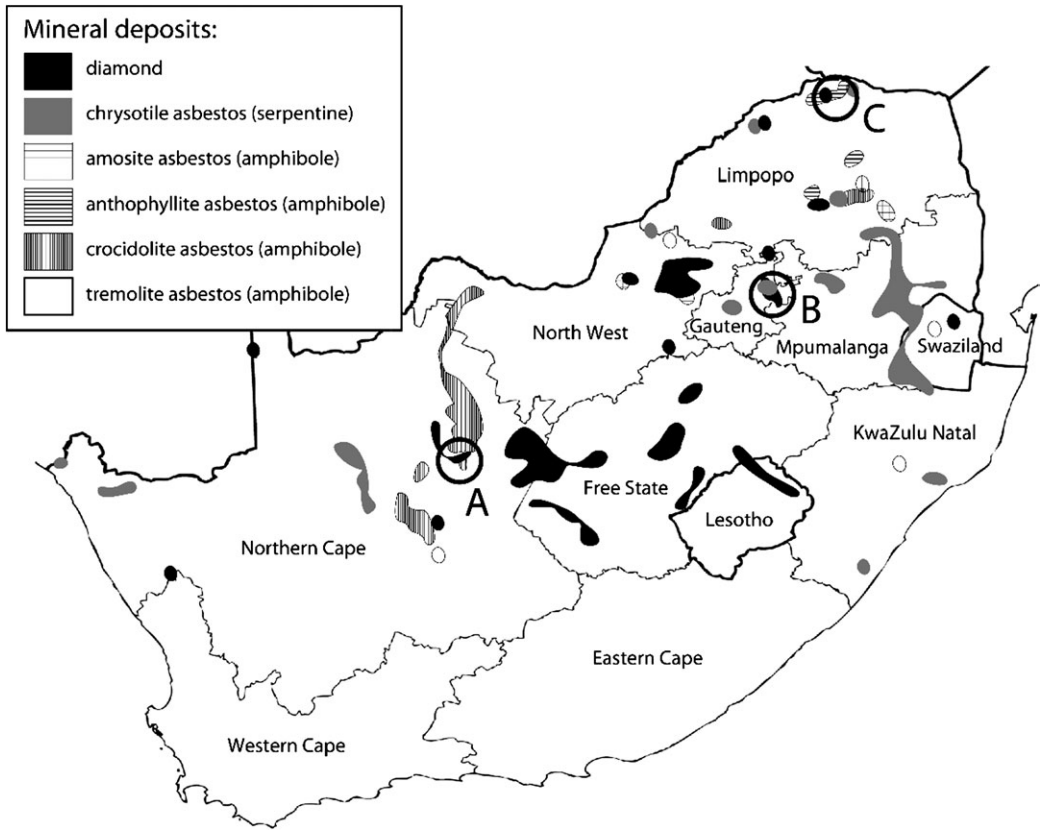


Fig. 1. Diamond and asbestos deposits in South Africa, with location of study mines marked (A, B, and C). Map not to scale, generated using ArcExplorer software (Council for Geoscience, 2001).

The results of annual physical medical examinations of mine workers are housed at the Medical Bureau of Occupational Diseases (MBOD). These results, together with chest X-rays, spirometry test results, compensation information, work histories, and other relevant documents, are stored in files, identified by a number linked to the PATHAUT database. The files of the study subjects were reviewed for possible asbestos exposure.

Mining companies' human resource departments keep paper-based and/or electronic records of all employees, including information on job descriptions, periods employed, etc. Where available, these records were reviewed for possible sources of asbestos exposure.

Burden of asbestos-related disease at autopsy

Diamond mine workers with complete work histories, identified on the PATHAUT database as having never mined a different commodity, and for whom the calculated age at which they started working was no >25 years were selected for the period January 1975 to December 2008. Those with pathological findings indicative of asbestos exposure, viz. asbestosis, pleural plaques, and/or malignant mesothelioma, were identified and all available documents were reviewed for possible sources of asbestos exposure, including company human resources records.

Asbestosis was diagnosed, microscopically, by the presence of diffuse interstitial fibrosis and two or more associated asbestos bodies. Historically, asbestosis on the PATHAUT database has been graded into three categories, conforming to those described by Roggli *et al.* (2010). Slight asbestosis comprises fibrosis involving the bronchiolar wall and adjacent alveoli. Moderate asbestosis is diagnosed when the fibrosis extends to more distant alveolar walls but excludes some alveoli between adjacent bronchioles (Fig. 2). Severe asbestosis comprises fibrosis involving all alveoli between adjacent bronchioles. Malignant mesothelioma is diagnosed according to the World Health Organization Classification of Tumours (Travis *et al.*, 2004). Pleural plaques were defined as circumscribed areas of dense acellular

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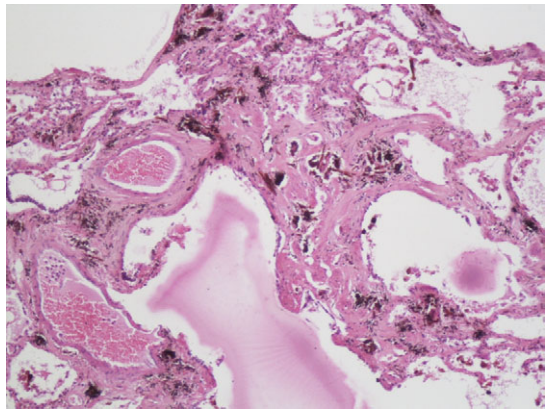


Fig. 2. Photograph of a section of a diamond mine worker's lung (Case C) showing moderate asbestosis (×40 magnification).

collagenization with a basket weave appearance, involving the parietal, diaphragmatic, and/or visceral pleura, and with a minimum diameter of 5 cm.

Asbestos fibres in lungs

Stored lungs from all diamond mine workers (defined as above) were selected for the period January 2001 to February 2009 as lungs were not stored prior to this. The MBOD files were reviewed for work histories.

Tissue samples of ∼2 cm^3 were cut from the upper, middle, and lower zones of one of the lungs of each mine worker. The tissue was weighed, digested using potassium hydroxide, rinsed to extract the residual minerals, and ashed at 400°C. The residue was mixed with 50 ml of distilled water, 1 ml of which was passed through a 0.2-μm polycarbonate filter. After drying, the filter was coated with gold and examined in a Jeol JSM 5600 scanning electron microscope (SEM) equipped with Thermo Noran energy-dispersive X-ray spectroscopy (EDS) capabilities. Two hundred fields were examined with an accelerating voltage of 15 kV and at a magnification of ×2000. Fibres defined as having an aspect ratio >3:1 were counted.

Fibres were categorized as asbestos or non-asbestos by identifying their chemical elements and the proportions in which these elements occurred, using EDS analysis. The asbestos fibres were classified as serpentine (chrysotile) or amphibole by comparing their spectral peaks against the Union Internationale Contre le Cancer standard asbestos samples and asbestos reference standards prepared and validated by the Institute of Occupational Medicine on behalf of the Health and Safety Executive (HSE), UK.

The naming of the specific amphiboles is problematic (Sokolova *et al.*, 2001) as they often occur on a continuum, making the precise identification of a fibre difficult. For example, although actinolite is an intermediate member between magnesium-rich tremolite and iron-rich ferro-actinolite, in the magnesium–iron-rich amphibole series, all three amphiboles are often simply referred to as tremolite–actinolite and we followed this convention.

Asbestos fibres in mine tailings and soils

Three diamond mines were selected, based on their geographical locations. Mine A is in the vicinity of crocidolite asbestos deposits, Mine B is located near chrysotile asbestos deposits, and there are both anthophyllite and chrysotile asbestos deposits near Mine C (Fig. 1). Grab samples of tailings were collected from all three mines, and samples of surface soil were collected from Mines A and C.

Tailings samples were placed onto a pure carbon disc and gold coated in preparation for SEM examination. The method used for the soil samples was based on that used by the HSE in the UK (Davies *et al.*, 1996). A 0.2 g sample of each soil was suspended in 50 ml of distilled water. The suspension was shaken and the particles were allowed to settle for 30 s. A 1 ml aliquot was then passed through a 25 mm diameter 0.8-μm Millipore polycarbonate filter. When the filter was dry, it was coated with gold and 420 fields were examined, as described above. For both tailings and soils, only qualitative analyses were performed.

Ethical considerations

Consent for autopsy examination was granted by the next-of-kin in terms of the Occupational Disease in Mines and Works Act of 1973 (Republic of South Africa, 1973). Approval for all studies utilizing retrospective data from PATHAUT was obtained from the University of the Witwatersrand Human Research Ethics Committee (clearance number 40421).

RESULTS

Burden of asbestos-related disease at autopsy

Less than 2% (1887 of 100 124) of all deceased miners on the PATHAUT database had a record of having worked in the diamond mining industry from 1975 to 2008. Of these, 29.6% (559) had no record of having mined any other commodity. The numbers were fairly consistent over the study period, with an average of 16 cases $year^{-1}$. In the last 4 years, however, these numbers decreased to <10 $year^{-1}$. Twenty-four of the 559 mine workers (4.3%) started working at the age of ≤25 years and had one or more

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asbestos-related diseases at autopsy: 21 had asbestosis, six had pleural plaques, and four had malignant mesothelioma (six had more than one asbestos-related disease).

The PATHAUT data alone were insufficient to determine if work histories were complete for many of the mine workers. Information from additional sources showed that four of the 24 diamond mine workers had worked as boilermakers and five had worked in asbestos mines. For nine of the mine workers, there was insufficient information to determine if they had worked exclusively in the diamond mining industry. The remaining six had no evidence of having worked elsewhere. Four of the six had asbestosis, one had pleural plaques, and one had malignant mesothelioma and pleural plaques (Table 1). Unfortunately, lungs for these mine workers were not stored as they all died prior to 2001.

Asbestos fibres in lungs

Twenty mine workers in the PATHAUT database fulfilled the selection criteria for lung fibre analysis. Nine mine workers were subsequently excluded: seven on the basis of asbestos exposure (one asbestos miner, three fitters and turners, and three boilermakers) and two who were diamond cutters who did not work on or near a diamond mine. None had an asbestos-related disease. Asbestos fibres were identified in five of the 11 lung samples (Table 2); all fibres were tremolite or fibres in the tremolite–actinolite series (Fig. 3). No chrysotile asbestos fibres were identified, although four of the five mine workers had worked in Mine B (close to a chrysotile asbestos deposit). The fifth mine worker worked in an area in which there are no asbestos deposits.

Asbestos fibres in mine tailings and soils

Tailings samples from Mine A (near crocidolite deposit) contained tremolite–actinolite series fibres (Table 3). No fibres were identified in the soil samples. The tailings samples from Mine B (near chrysotile deposit) contained both chrysotile fibres and tremolite–actinolite series. While the soil samples from Mine C (near anthophyllite and chrysotile deposits) contained no asbestos fibres, both the tailings samples and the dust collected from a crusher located above ground contained tremolite–actinolite series fibres. During the study, a sample of pure chrysotile asbestos was also collected from between the kimberlite pipe and the country rock in this mine.

DISCUSSION

Chrysotile and asbestiform amphiboles are associated with disease. This is the first report of asbestos fibres in the lungs of diamond mine workers. Tremolite–actinolite fibres were identified in the lungs of mine workers, as well the diamond mine tailings, supporting the hypothesis that diamond mine workers are at risk of exposure to non-commercial asbestos.

Two of the mine workers with asbestosis worked for very short periods. Both these men worked underground in the 1970s when dust control was known to be poor and may have been exposed to high asbestos levels. Although asbestosis is generally associated with relatively high exposure levels, it is possible that

Table 1. Asbestos-related pathological findings in six diamond mine workers

Case number	Age at death (years)	Total number of years employed	Years worked	Clinical cause of death	Pathological findings indicative of asbestos exposure	Severity of asbestosis	Asbestos bodies	Occupation	Mine
A	24	3	1972–1975	Gastric ulcer	Asbestosis	Slight	Yes	Underground labourer	B
B	69	35	1936–1971	Lung cancer	Asbestosis Lung cancer	Slight	Yes	Mason, miner, carpenter, and timberman	Other[a]
C	30	5	1974–1979	Tuberculosis Pneumonia	Asbestosis	Moderate	Yes	Underground first aid attendant	B
D	40	20	1962–1982	Multiple injuries	Asbestosis	Slight	Yes	Labourer	Other[a]
E	43	25	1972–1997	Empyema	Mesothelioma Pleural plaques		No	Labourer	B
F	50	27	1971–1998	Atrial fibrillation	Pleural plaques		No	Driver Machine operator	B

[a]In vicinity of crocidolite asbestos deposits; mine no longer operational.

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Table 2. Asbestos fibre content of lung tissue of diamond mine workers

Case number	Age at death (years)	Total numbers of years employed	Years worked	Asbestos fibre type	No. asbestos fibres[a]	Asbestos fibre count[b]	Asbestos bodies	Clinical cause of death[c]	Occupation	Mine at which employed
1	48	30	1973–2003	Tremolite–actinolite	13	629 662	No	Intra-cerebral bleeding	Labourer	B
				Chrysotile	0	<48 435				
2	50	27	1974–2004	Tremolite–actinolite	16	794 448	No	Not stated	Underground labourer	B
				Chrysotile	0	<49 635				
3	28	8	1994–2002	Tremolite–actinolite	8	264 506	No	Not stated	Mechanic	B
				Chrysotile	0	<33 063				
4	50	31	1972–2003	Tremolite–actinolite	11	443 092	Yes	Cardio-myopathy	Sampling helper	B
				Chrysotile	0	<40 281				
5	39	21	1980–2001	Tremolite–actinolite	4	227 047	Yes	Tuberculosis	Underground labourer	Other[d]
				Chrysotile	0	<56 761				
6	26	5	1995–2001	Tremolite–actinolite	0	<98 092	Yes	Pneumonia Tuberculosis	Driller	Other[d]
				Chrysotile	0	<98 092				
7	64	28	1961–1989	Tremolite–actinolite	0	<75 172	No	Cerebro-vascular accident	Labourer Driver	B
				Chrysotile	0	<75 172				
8	34	14	1988–2002	Tremolite–actinolite	0	<53 553	No	Tuberculosis Pneumonia	Trammer Lasher	Other[d]
				Chrysotile	0	<53 553				
9	75	33	1952–1985	Tremolite–actinolite	0	<58 867	No	Not stated	Miner	B
				Chrysotile	0	<58 867				
10	54	27	1967–1994	Tremolite–actinolite	0	<89 579	No	Myocardial infarction	Miner	B
				Chrysotile	0	<89 579				
11	78	39	1949–1988	Tremolite–actinolite	0	<34 931	No	Respiratory failure	Diamond grader	B
				Chrysotile	0	<34 931				

[a]Counting criteria: aspect ratio >1:3 and EDS analysis for chemistry.
[b]Calculated per gram dry weight; in our laboratory, ≥125 000 fibres g^{-1} dry weight is considered indicative of significant exposure; calculations based on number of fibres and dry weight of lung tissue; where no fibres were identified, calculated detectable limit was based on the count of one fibre.
[c]As stated on the death certificate accompanying the cardiorespiratory organs.
[d]Mine other than one of the study mines, A, B, or C.

fibrosis may occur at lower exposure levels (Roggli *et al.*, 2010). Alternatively, or in addition, they may have been exposed to asbestos in the environment, which would not have been recorded. We acknowledge that missing information on environmental exposure is a limitation of this study.

While the amphibole concentrations in the lung appear to be not particularly high, given the long duration of employment, they are all >125 000 fibres g^{-1} of dry lung tissue, the level considered in our laboratory to be indicative of significant exposure.

Chrysotile fibres break up into very fine fibrils which are not always visible using SEM. This may be the reason that we did not detect chrysotile fibres in the lungs, in addition to the rapid clearance of chrysotile fibres from the lungs.

The type of fibre found in the lung tissue appears to match that found in the tailings. None of the commercial types of asbestos mined in the adjacent asbestos mining areas were found in the lung tissue. The difference in risk from the two sources is not measurable, but the amphiboles identified in this

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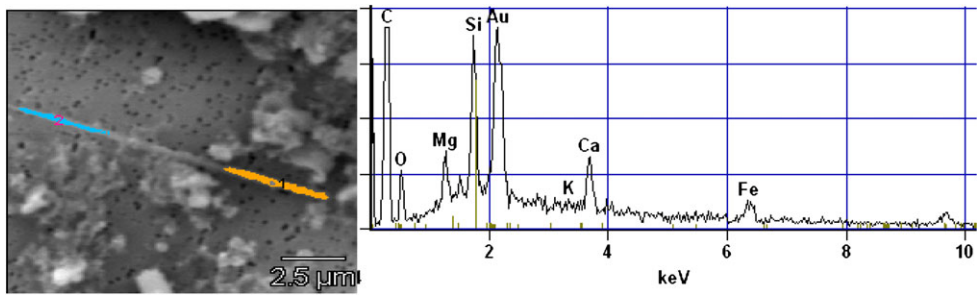


Fig. 3. Fibre identified in the lung of a diamond worker using SEM and corresponding elemental trace using EDS analysis.

Table 3. Asbestos fibres identified from different sources

Mine	Associated asbestos deposit	Source	Asbestos fibres identified[a]
A	Crocidolite	Soil	None
		Tailings	Tremolite–actinolite
B	Chrysotile	Tailings	Tremolite–actinolite
			Chrysotile
C	Anthophyllite	Soil	None
		Tailings	Tremolite–actinolite
	Chrysotile	Dust	Tremolite–actinolite
		Pipe perimeter	Chrysotile

[a]Counting criteria: aspect ratio >1:3 and EDS analysis for chemistry.

study are probably associated with the kimberlite. However, tremolite asbestos fibres have previously been identified at low levels in South African chrysotile (Rees *et al.*, 1992) and in the lungs of chrysotile asbestos miners (Rees *et al.*, 1992, 2001) and it is possible that the chrysotile deposits in the area of the asbestos mines are contaminated with amphibole asbestos. It is unlikely that the contamination is as high as that in the mines in Quebec where high prevalences of mesothelioma have been reported (McDonald, 2010). No mesotheliomas associated with chrysotile exposure have been reported in South Africa (Rees *et al.*, 1992).

We identified only one diamond mine worker (a labourer) with mesothelioma in this study. Mesothelioma is an indicator of exposure to asbestos and there is no known threshold below which the tumour does not occur. The high prevalence of HIV in the past two decades has reduced the longevity of South Africans, especially black men who comprise the majority of mine labourers. Black miners coming to autopsy are generally younger than white miners (Ndlovu *et al.*, 2010) and have a much lower life expectancy.

Although the mining and use of asbestos are now banned in many countries, including South Africa (Department of Environmental Affairs and Tourism, 2007), the risk of exposure continues, both in the workplace and in the environment, and it is important to identify these sources. Fibres can be liberated from asbestos-containing products during demolition and renovation processes, as well as during weathering of asbestos-containing products (Phillips *et al.*, 2009). Asbestos tailings dumps that have not been rehabilitated, and those where rehabilitation has not been successful in the long term, also generate fibres. A further source of asbestos exposure is deflation [the detachment of mineral dust from the ground surface, through weathering and erosion (Derbyshire, 2007)] of asbestos outcrops that were never mined (sometimes referred to as naturally occurring asbestos).

One of the limitations of this study is the quality of the work histories. Details of employment are recorded on the PATHAUT database from information received from the most recent mining company that employed the worker and, in most cases, is limited to that company. This was highlighted when we attempted to ascertain complete work histories from mine company records archived at the individual mines.

South Africa is a uniquely mineral-rich country, and in many areas, such as the Bushveld Complex, there are concentrations of a variety of minerals. When mining one commodity, it is very likely that other minerals will be accidentally mined at the same time. Asbestos is a major concern as it causes severe health effects. Mining geologists and industrial hygienists should routinely look for asbestos. All mining companies need to conduct risk assessments and institute medical surveillance programmes for asbestos-related diseases.

Even at low concentrations in the ore, amphibole asbestos has the potential to cause disease in mine workers, as demonstrated in the Libby vermiculite workers where the concentration of tremolite in the ore was ~1% (Nolan *et al.*, 1999). When reviewing cases with asbestos-related disease for compensation, exposure to asbestos, as a mineral associated with the commodity that was mined, should be considered.

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It is important to identify specific areas of risk for exposure to asbestos. The PATHAUT database is the only medical surveillance tool in South Africa that might serve this purpose in the mining industry. The database currently comprises >100 000 records from 1975 and provides an ideal platform for the surveillance of asbestos risk groups and areas as asbestos-related diseases continue to be diagnosed at autopsy. Extrapolation from the findings might also identify a risk to the spouse and children of mine workers or a general environmental risk to the surrounding community.

The problem of incomplete work histories is not confined to the diamond mines. All mining companies have a social responsibility to record comprehensive work histories, from the time the worker was first employed, for the benefit of families seeking compensation for occupational disease. This should be done by the mine medical services at entry, annual, and exit examinations and should be recorded in the human resources and medical files. Details should be recorded with respect to dates, specific companies and occupations, tasks, and geographical location of each employer. Companies should also record where the mine worker was born and where he lived.

CONCLUSIONS

Our findings show that diamond mine workers are at risk of developing asbestos-related diseases. Asbestos is associated with the South African diamond mining industry, primarily due to the nature of kimberlite but also the location of the diamond mines in relation to asbestos deposits. South Africa is a uniquely mineral-rich country and, when mining one commodity, it is likely that other minerals, including asbestos, will be accidentally mined. Even at low concentrations, asbestos has the potential to cause disease. Although the use and mining of asbestos are banned, all mining companies should be aware of the continuing risk of asbestos exposure due to the accidental mining of asbestos, as well as task-related activities and environmental exposure. Recording of comprehensive lifelong work histories should be mandatory to enable the risk to be quantified in future studies.

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APPENDIX 4

Nelson G, Murray J. Silicosis at autopsy in platinum mine workers (provisionally accepted for publication by Occ Med, Jan 2012)

Silicosis at autopsy in platinum mine workers

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Background South Africa is the largest producer of platinum group metals in the world. Platinum is found in the Bushveld Complex in the north-east of the country. This volcanic intrusion contains many other minerals, including crystalline silica. Little is known about the health risks in the platinum mining industry.

Aim The aim was to explore the potential for platinum mine workers to develop silicosis.

Methods Autopsies are performed at the National Institute for Occupational Health, for compensation purposes. Platinum mine workers, who had worked for more than a year and had silicosis and/or fibrotic nodules in the lymph nodes, were identified from the autopsy database. An exhaustive search of other available data sources was undertaken to exclude exposure to silica dust in the gold mining industry.

Results Eighty-five of 3 863 (2.2%) platinum mine workers employed for more than a year had silicosis at autopsy; an additional 490 (12.7%) had fibrotic nodules in the lymph nodes. After reviewing all data sources, five mine workers with silicosis and 25 with fibrotic nodules in the lymph nodes fulfilled the study inclusion criteria.

Conclusion This case series supports the suggestion that there is a risk of silica exposure in platinum mine workers, as demonstrated by the few silica dust measurements taken in the platinum mines. The mines should be cognisant of this risk and should measure silica dust levels routinely. Recording of comprehensive work histories and silica dust measurements should be mandatory to enable risks of disease to be quantified in future studies.

Keywords silica, autopsy, PATHAUT, miners

Introduction

The platinum group metals (PGMs) have surpassed gold in value, production and employment in South Africa. Production has more than doubled from 128 000 kg in 1987 to 275 000 kg in 2008 [1]. Employment has increased by 240% since 1987; in 2008, the platinum mining industry employed almost 200 000 workers, comprising 39% of miners in all commodities [1].

A number of minerals and compounds are associated with the PGMs which occur in the mineral-rich Bushveld Complex in South Africa. The three most abundant are being pyrrhotite, chalcopyrite and pentlandite, but no health effects of exposure to these are described in the literature [2]. Others include chromite, gold, copper and nickel [3], as well as smaller amounts of sulphur, arsenic, selenium, tellurium, iron, tin, cobalt, zinc, titaniferous magnetite and vanadium pentoxide [2]. Some produce adverse respiratory health effects: interstitial fibrosis may result from exposure to copper or iron [4], pneumoconiosis from cobalt [5], stannosis from tin [4,6], adult respiratory distress syndrome from zinc or sulphur [7], and lung cancer from arsenic or nickel [4]. The Bushveld Complex, as an igneous intrusion, also contains crystalline silica, one of the most commonly occurring harmful associated minerals to which miners in South Africa are likely to be exposed. Although the average crystalline silica level in igneous rocks is about 12% [8], the variation is wide.

Very little is known about the health of platinum miners. Studies in South Africa have focussed on the gold and asbestos mining industries and, to a lesser extent, the coal mining industry. Health in miners of other commodities has not been well researched. Although it has been shown that platinum refinery workers are at an increased risk of developing platinum salt sensitivity [9,10] associated with respiratory symptoms of asthma, rhinitis and urticaria, and dermatitis, there is almost no published research on diseases in platinum miners.

Silicosis has been reported in platinum miners but is ascribed to previous exposure to silica dust in the gold mining industry in which many platinum miners worked. With the reduction in gold production and the rapid expansion of platinum mining, many retrenched gold miners find employment in the platinum mines as the methods used in gold and platinum mining are similar, i.e. hard rock mining which requires drilling and blasting.

It is possible, however, that platinum miners are exposed to crystalline silica through accidental mining (the unintentional mining of a mineral other than the one of primary interest). The ore bodies in which minerals are found often contain other minerals, either in juxtaposition to the mineral of interest or scattered within the surrounding country rock. Miners drill through rock containing these associated minerals, creating dust to which they are exposed. Accidental mining of other minerals, such as asbestos, has been described in the literature [11-13].

Crystalline silica is not routinely measured in the platinum mines. Two studies reported low silica dust levels in some mines. In 2003, Biffi and Belle measured the crystalline silica content of crushed stope rock samples from two platinum mines in the Bushveld Complex, one of which mined the Merensky platinum reef and the other, the UG2 reef [14]. The silica content of the rock was 0.45% in both, compared to 9.9% and 39.1% in the two gold mines in the study. Seven static airborne gravimetric respirable dust samples from the one platinum mine and three from the other were performed over full shifts: the silica content, measured by xray diffraction (XRD) analysis, was less than 0.2%, compared to 4.5 to 57% in the gold mines. In 2007, Dekker *et al*., measured respirable dust in one underground platinum mine [15]. They collected and analysed 113 personal dust samples over full shifts. Using XRD analysis, they measured respirable silica dust concentrations ranging from 0.018 mg/m^3 to 0.035 mg/m^3, equivalent to 18% and 35% of the South African legislated OEL of 0.1 mg/m^3. However, even these levels may not be low enough to prevent disease.

In a paper published in 2000, Greaves concluded that the 0.05 mg/m^3 recommended exposure limit (REL) of the National Institute for Occupational Safety and Health (NIOSH) might not be sufficiently protective for a substantial proportion of workers [16]. Subsequently, the American Conference of Governmental Industrial Hygienists (ACGIH) set a limit of 0.025 mg/m3 in 2006, slightly lower than the 0.035 mg/m3reported by Dekker *et al*. [15].

The only study of silicosis in platinum miners was a cross-sectional survey of 969 platinum miners seen for routine occupational health surveillance, at a single platinum mine. Silicosis was diagnosed in three, all of whom had a history of gold mining. The diagnosis, made by two experienced readers, was based on the International Labour Organisation (ILO) chest radiograph score for silicosis of at least 1/1. Two were given an ILO score of 1/1 and one, 1/2. Radiographs are, however, limited in terms of their specificity and sensitivity for diagnosing silicosis [17,18].

We discuss the potential for platinum miners to develop silicosis by describing a case series of deceased mine workers from the platinum mining industry with pulmonary silicosis and/or fibrotic nodules in the lymph nodes.

Methods

Histological examination of lung tissue is the gold standard for the diagnosis of silicosis. Any person who has worked on a South African mine has a legal right to have his/her cardio-respiratory organs examined for compensable disease, regardless of the cause of death, and provided that the next-of-kin agrees [19] . Autopsies are performed at the National Institute for Occupational Health (NIOH) in Johannesburg. The pathological findings, the commodities and the number of years for which he was employed in each, and demographic data, are recorded in the PATHAUT (Pathology Automation) database [20]. All histological material is archived, and lungs from miners of specific commodities or with specific diseases have been stored since 2001. Around 2 000 autopsy examinations are performed annually; platinum mine workers (defined as those who worked for longer in the platinum mining industry than in any other mining industry) currently comprise around 20% of all miners and ex-miners coming to autopsy [21]. The proportion has more than doubled since 1998 [22].

The evidence for silica exposure in platinum miners was not restricted to pulmonary silicosis. Mine workers with fibrotic nodules in the hilar lymph nodes were also included in the case series. The occurrence of fibrotic nodules in the lymph nodes of silica-exposed individuals is well described [23,24], and their presence may be a sensitive indicator for the potential to develop silicosis. These fibrotic nodules have the same histological characteristics as silicotic nodules in the lung parenchyma but, when limited to the lymph nodes, they are not called silicosis [24].

There is evidence that fibrosis in the lymph glands precedes the development of overt pulmonary silicosis [25]. Baldwin and Lambert described five workers exposed to silica who initially presented with bilateral hilar lymphadenopathy and no radiographic evidence of interstitial lung disease; one progressed to silicosis [26]. Fibrosis of the lymph glands may be a response to low level silica dust as suggested by Murray *et al*. in 1991. They showed that a higher proportion of gold miners had fibrosed glands at autopsy than pulmonary silicosis after relatively short periods of employment [27]. This was also the finding in a more recent study in which silica exposure was lower for uranium miners with lymph node fibrosis only than for those with both lymph node fibrosis and parenchymal silicosis [28]. The association of lymph node fibrosis with parenchymal silicosis remained after adjustment for silica exposure.

Thus, workers with silicosis and/or fibrotic nodules in the lymph nodes, with PATHAUT records indicating that they were employed exclusively in the platinum mining industry, for more than one year, were selected for the period January 1975 to December 2009. The records of those who started working before the age of 25 were comprehensively reviewed. The age of 25 was chosen to minimise the possibility of the inclusion of workers who had previously worked in the gold mines.

Mine workers undergo annual physical examinations, the results of which are centralised at the Medical Bureau of Occupational Diseases (MBOD). These include chest x-rays, spirometry test results, compensation information, work histories and other relevant documents, all of which are kept in individual paper-based files. Each mine worker is allocated a 'bureau' number which is linked to the PATHAUT database. The files of all the study subjects were reviewed for evidence of employment in the gold mining industry.

The PATHAUT database is linked to the electronic Mineworkers Compensation (MWC) database which was also searched for work histories. The MWC links various databases and the system and supports the functions of the MBOD, the NIOH and the Compensation Commissioner for Occupational Diseases.

If a miner was recruited by The Employment Bureau of Africa (TEBA), he is assigned an industry number which he keeps for life, no matter for which mining company he works, and his work history is recorded in an electronic database. TEBA was provided with a list of industry numbers of all platinum mine workers with silicosis and/or fibrotic nodules in the lymph glands at autopsy to identify those with gold mining histories.

Where telephone numbers of relatives were recorded (in either the PATHAUT or the MBOD files), the relative was contacted and asked if the person had ever worked in the gold mining industry. Mining companies' human resource departments were also asked to provide work histories.

Pulmonary silicosis is diagnosed at autopsy by experienced pathologists, and is defined as the presence of palpable silicotic nodules on macroscopic examination of the lungs, which is then confirmed on microscopic examination. The degree of severity of silicosis is based on the number of silicotic nodules on macroscopic examination, and is categorised as occasional (one to four nodules), few (5 to fourteen nodules), moderate (15 to 30) or large (more than 30). Sections of the silicotic nodules are routinely examined microscopically, and exhibit foci of concentric fibrosis with a whorled gray pattern [24]. Hilar lymph nodes from the right and left lungs are also routinely removed and examined histologically. All cases were reviewed to confirm the diagnosis of pulmonary silicosis and/or the presence of fibrotic nodules in the lymph nodes.

Consent for autopsy examination was granted by the next-of-kin in terms of the Occupational Disease in Mines and Works Act of 1973 (Republic of South Africa, 1973). Approval for all studies utilizing retrospective data from the PATHAUT database was obtained from the University of the Witwatersrand Human Research Ethics Committee (clearance number 40421).

Results

A total of 12 241 men on the PATHAUT database had worked in the platinum mining industry from 1975 to 2009. Of these, 6 490 (53.0%) had no record of having worked in another mining sector. Almost 60% (n = 3863; 59.5%) had been employed for more than one year. Eighty-five (2.2%) had silicosis at autopsy. Fibrotic nodules were diagnosed in the lymph nodes of 490 (12.7%).

After reviewing all the data sources, five mine workers with pulmonary silicosis (four of whom also had fibrotic nodules in the lymph nodes) remained. All had occasional (one to four) palpable nodules and were therefore classified as mildly diseased. An additional 25 mine workers with fibrotic nodules in the lymph nodes but without silicosis fulfilled the study inclusion criteria (Table 1).

There was enough evidence to suggest that all five mine workers with silicosis had developed the disease in the course of their employment on the platinum mines. Four of the five also had fibrotic nodules in the lymph nodes. The 25 additional men with fibrotic nodules in the lymph nodes only had worked in the platinum mining industry for an average of 23 years and 6 months (range 6 years and 4 months to 30 years; median 25 years and 6 months.

Discussion

Silicosis rates in gold miners in South Africa are amongst the highest in the world: in 2007, 22% and 32% black and white miners, respectively, were diagnosed with silicosis at autopsy [29]. The case series presented here is the first comprehensive report on silicosis in platinum mine workers. While some studies have reported on the hazard of silica dust exposure in platinum mines, little research has been done on health outcomes. The only previous study on silicosis looked at fewer than 1000 platinum miners in a single mine; silicosis was diagnosed in three, all with a gold mining history [2].

Together with the analysis of the silica content of the platinum-bearing ore and the ambient air in the platinum mines, this case series completes the triangulation of evidence of silica and silicosis in the platinum mining industry, all of which indicate that there is a risk for platinum miners to develop silicosis. First, there is crystalline silica in the platinum-containing ore, as measured by Biffi and Belle [14], albeit at much lower levels than the gold mine rock. Second, measured respirable silica dust concentrations range from 0.018 mg/m3 to 0.035 mg/m3 [15], higher than the ACGIH threshold limit value (TLV) of 0.025 mg/m3. Third, as shown in this study, silicosis is diagnosed at autopsy in platinum miners with no history of exposure to silica dust elsewhere, such as in the gold mining industry.

The PATHAUT database is an administrative database. Its primary purpose was to record pathological diagnoses of occupational respiratory disease which, together with work histories, could be reviewed for the payment of compensation to the families of deceased miners. It is the only database available for the analysis of compensable disease in the mining industry, comprising more than 100 000 records of deceased miners that have been collected since 1975. The database contains records of almost 6 500 platinum mine workers from all of the major platinum mines in the Bushveld Complex and is the most comprehensive database of disease in platinum miners at autopsy in the world.

While the data in the PATHAUT database are continuously validated, information is sometimes incorrectly recorded and not all data entries can be easily checked and validated, giving rise to misclassification. Some men might have been categorised as exclusive platinum mine workers although they had undisclosed histories of having worked in the gold mining industry. However, the methodology employed in this study, viz. the review of the MDOD, TEBA and mining company records minimised any such biases.

The main limitation of this study is the incomplete work histories. Gaps in employment histories exist in the records of many of the mine workers that come to autopsy. It is possible that some of the miners were incorrectly classified as exclusive platinum mine workers. Many miners start working from the age of 18, so some of the miners may have worked in the gold mines before

moving to the platinum mines. We tried to minimise this bias by excluding all those who started working after the age of 24.

The PATHAUT database, together with the MWC system, contains as much information on work and exposure histories as is available. Often, this information is supplied by the last company where the mine worker was employed. It is recommended that mine human resource departments record comprehensive work histories of all employees. This information can be recorded at entry and exit interviews as well as during annual physical examination visits to the mine medical services. A complete life history, including geographical location from birth, should ideally be recorded to document possible exposures to dusts with known adverse health effects.

Mining sectors that perceive little or no health risks from exposure to dust do not routinely measure dust, including silica. The availability of comprehensive data on work histories and the recording of silica and other dust measurements in all mining sectors will facilitate studies such as the one reported here, as well as studies on the health effects of mining lesser researched commodities with smaller work forces, such as lime, vanadium, chromite, etc. Currently, very little is known about the health effects of exposure to dusts and fumes generated by the mining of some of these commodities.

In conclusion, there are very few recorded silica dust measurements from any of the platinum mines, but those that are available provide evidence that the silica dust levels to which platinum mine workers are exposed are high enough to cause disease. Platinum mine workers are diagnosed at autopsy with fibrotic nodules in the lymph nodes, indicative of silica dust exposure, and/or silicosis. All mine medical services need to have systems in place for the diagnosis of silicosis (and other diseases) that might be caused by exposure to dust in previous work places, as well as dust not perceived to pose a risk in the particular mining sector in which the worker is currently employed. The recording of life-long work histories, including those prior to current employment, should be a mandatory requirement of all mining companies. This should be in conjunction with improved occupational hygiene services, incorporating the routine measurement of dust levels.

Key points

- Little research has been conducted on the health of platinum mine workers, unlike workers in other mining sectors.
- Silica dust levels in South Africa platinum mine workers may not be low enough to prevent disease.
- Platinum mine workers have a risk of developing silicosis.

Acknowledgments

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Table 1. Demographic details of platinum mine workers with silicosis and/or fibrotic nodules in the lymph nodes at autopsy

Case no.	Pulmonary silicosis (severity)	Fibrotic nodules in the lymph nodes*	Year of death	Age at death	Age at start of employment	Length of employment	Years worked	Cause of death	Occupation
1	Yes (mild)	No	1986	25	18	7y 0m	unknown	External cause	Blasting assistant, mine stoper, jack hammer operator
2	Yes (mild)	Yes	2002	43	19	23y 9m	1978 - 2002	Tuberculosis	Pipes, tracks and ventilation worker
3	Yes (mild)	Yes	2004	47	22	23y 9m	1979 - 2004	Myocardial infarction	Loader driver
4	Yes (mild)	Yes	2006	45	20	25y 2m	1981 - 2006	Acute myeloid leukaemia	Artisan
5	Yes (mild)	Yes	2008	44	23	20y 6m	1987 - 2008	Multi-organ failure	Miner
6	No	Yes	2009	52	24	28y 6m	1981 - 2009	Broncho-pneumonia	Pipe track labourer
7	No	Yes	2008	27	22	6y 4m	2003 - 2008	Broncho-pneumonia	Locomotive driver
8	No	Yes	2007	49	21	28y 3m	1979 - 2007	Interstitial pneumonitis	Underground miner
9	No	Yes	2008	43	19	21y 9m	1983 - 2008	Tuberculosis	Underground battery attendant
10	No	Yes	2005	48	20	25y 3m	1977 - 2005	Tuberculosis	Underground miner
11	No	Yes	2007	42	20	21y 5m	1985 - 2007	Tuberculosis	Underground pump attendant
12	No	Yes	2008	52	24	28y 0m	1980 - 2008	Tuberculosis	Locomotive driver
13	No	Yes	2008	47	21	23y 9m	1982 - 2008	Tuberculosis	Shift supervisor
14	No	Yes	2007	52	22	26y 1m	1977 - 2007	Tuberculosis	Boilermaker
15	No	Yes	2008	36	24	12y 1m	1996 - 2008	Tuberculosis	Winch driver
16	No	Yes	2004	50	21	30y 0m	1974 - 2004	Tuberculosis	Jack hammer operator
17	No	Yes	2006	47	22	25y 5m	1980 - 2006	Cardiac failure	Shaft timberman

18	No	Yes	2009	48	18	30y 1m	1979 - 2009	AIDS	Underground stoper
19	No	Yes	2005	45	21	22y 3m	1981 - 2005	Pneumocystic pneumonia	Winch driver
20	No	Yes	2009	48	22	26y 7m	1983 - 2009	Pneumocystic pneumonia	Machine operator
21	No	Yes	2009	45	21	24y 7m	1985 - 2009	Pneumocystic pneumonia	Locomotive operator
22	No	Yes	2008	33	21	11y 8m	1996 - 2008	AIDS	Stope lasher
23	No	Yes	2005	40	22	16y 7m	1987 - 2005	AIDS	Underground miner
24	No	Yes	2009	46	19	28y 4m	1981 - 2008	AIDS	Scraper winch operator
25	No	Yes	2008	52	24	27y 6m	1980 - 2008	AIDS	Stope timberman
26	No	Yes	2008	49	22	27y 11m	1980 - 2008	Unknown	Scraper winch driver
27	No	Yes	2000	66	19	17y 2m	1953 - 1970	Pneumonia	Plumber/rigger
28	No	Yes	2005	47	21	25y 1m	1979 - 2005	Mine accident	Locomotive driver
29	No	Yes	2007	61	24	29y 6m	1970 - 1999	Unknown	Ventilation officer
30	No	Yes	2008	45	20	25y 7m	1982 - 2007	Tuberculosis	Jack hammer operator

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APPENDIX 5

Standardised booklet in which pathology findings are recorded

NIOH: Pathology Examination Booklet

Ex-miner: Yes ☐ No ☐

P number

Bureau no

Pathologist

Applic ID

Red Box No

Examination Report Status Code

cardio-respiratory organs only ☐

cardio-respiratory organs with limited autopsy see appended report ☐

cardio-respiratory organs with full autopsy see appended report ☐

Cause of death: death certificate _____

Cause of death: NIOH Pathologist _____

Surname

First name plus initials

Industry no

National ID no

Population group ☐

Gender ☐

	dd	mm	year
received			
examined			
report			
birth			
death			
age			

Exposure:

	Type	Year
most		
2nd most		
3rd most		
4th most		
Other		

Calender period: year started

year ended

Last mine worked _____

block no.	no.	tissue	remarks

Slight case ☐

Additional comments_____

Macroscopic report

Regional glands

1		could not be examined
2		show no gross change
3		are autolysed
4		are pigmented
5		are enlarged
6		are fibrosed
7		are calcified
8		are tuberculous
9		are tumour infiltrated
10		additional comments _____

Pulmonary vessels

1			could not be examined
2			show no gross change
3			thrombo embolism is present
	1		on left
	2		on right
4			additional comments _____

Pleura

1			plaques are absent
2			occasional
3			few
4			moderate
5			numerous
6			a chronic non-specific plurisy present
	1		on left
	2		on right
7			an acute pleurisy is present
	1		on left
	2		on right
8			asbestotic plaque formation
	1		on left
	2		on right
9			this involves the
	1		parietal pleura
	2		visceral pleura
	3		diaphragm
10			pleural shows malignant infiltration
	1		on left
	2		on right
11			additional comments _____

Bronchi

1			could not be examined
2			show no gross change
3			bronchiectasis is present
	1		on left
	2		on right
4			additional comments _____

Heart

1			not submitted
2			poorly preserved
3			shows no gross change
4			total weight ☐☐☐☐
5			ventricular mass (N<250) ☐☐☐☐
6			mass of L V + septum (N190) ☐☐☐☐
7			mass of R V (N<65) ☐☐☐☐
8			L V + septum: R V ☐☐
9			is enlarged due to
	1		left ventricular hypertrophy
	2		right ventricular hypertrophy
10			atherosclerosis is
	1		absent
	2		slight
	3		moderate
	4		marked
11			disease is present in the
	1		mitral valve
	2		aortic valve
	3		tricuspid valve
	4		pulmonary valve
12			the pericardium shows a
	1		chronic non-specific pericarditis
	2		fibrinous pericarditis
	3		tuberculous pericarditis
13			ante mortem thrombus is present
14			bypass grafts are present
15			there is ischaemic fibrosis
16			a valve replacement is present
17			there is a wound
18			additional comments _____

Lungs

1 ☐ inflated: not weighted
2 ☐ poorly preserved
3 ☐ a whole lung section was taken
4 ☐
  1 ☐ left lung ☐☐☐☐ g
  2 ☐ incomplete not weighed
5 ☐
  1 ☐ right lung ☐☐☐☐ g
  2 ☐ incomplete not weighed
6 ☐ show(s) an increase in pigment
7 ☐ there is no evidence of a pneumoconiosis
8 ☐ the pigment is aggregated in
  1 ☐ occasional
  2 ☐ a few
  3 ☐ a moderate number
  4 ☐ a large number ☐ islets
9 ☐ the pigment is aggregated in
  1 ☐ occasional
  2 ☐ a few
  3 ☐ a moderate number
  4 ☐ a large number ☐ nodules
10 ☐ the pigment is aggregated in
  1 ☐ occasional
  2 ☐ a few
  3 ☐ a moderate number
  4 ☐ a large number ☐ macules
11 ☐ emphysema is
  1 ☐ absent
  2 ☐ insignificant
  3 ☐ moderate
  4 ☐ marked
12 ☐ the type of emphysema is
  1 ☐ panacinar
  2 ☐ centrilobular
  3 ☐ focal
  4 ☐ irregular
13 ☐ emphysema score on the W.L.S. ☐☐☐
14 ☐ the W.L.S is not suitable
15 ☐ marginal bullae are present
16 ☐ there is a primary neoplasm in
  1 ☐ the left lung
  2 ☐ the right lung
17 ☐ the tumour is
  1 ☐ central
  2 ☐ peripheral
18 ☐ the tumour measures ☐☐☐ mm
19 ☐ metastatic neoplastic deposits are
  1 ☐ present in the left lung
  2 ☐ present in the right lung
20 ☐ consolidation is
  1 ☐ absent
  2 ☐ present in the left lung
  3 ☐ present in the right lung
21 ☐ the parenchyma shows
  1 ☐ necrosis
  2 ☐ fibrosis
  3 ☐ encapsulated necrosis
  4 ☐ cavitation
  5 ☐ milliary TB
22 ☐ tuberculosis is
  1 ☐ absent
  2 ☐ insignificant
  3 ☐ moderate
  4 ☐ marked
23 ☐ there is a lacerated wound
24 ☐ haemorrhage is present
25 ☐ palpable foci are present
26 ☐ additional comments _____

Oesophagus

1		there is a neoplasm
2		the tumour measures ☐☐ mm
3		additional comments _____

_____

_____

Microscopic report

Regional glands

1		too poorly preserved for assessment
2		dust deposition but no fibrosis
3		diffuse non-specific fibrosis
4		foci of concentric fibrosis
5		foci of calcification
6		encapsulated necrosis consistent with with inactive TB
7		granulomatous inflammation consistent with active TB
8		sarcoidosis cannot be excluded
9		foci of malignancy
10		additional comments _____

_____

_____

_____

Lungs

1		show	
	1		slight
	2		moderate — dust deposition but no pneumoconiosis
	3		marked
2		foci of dust reticulation	
	1		with insignificant collagenisation
	2		with significant collagenisation
3		silicotic islets	
4		silicotic pleural plaques	
5		ferruginous bodies	
	1		are not present
	2		are present but no associated fibrosis
	3		with slight interstitial fibrosis
	4		with moderate interstitial fibrosis
	5		with marked interstitial fibrosis

6		the macules show
	1	insignificant collgenisation and focal emphysema
	2	significant collgenisation and focal emphysema
	3	significant collgenisation without focal emphysema
7		nodules - mixed dust pneumoconiosis
8		show massive fibrosis
9		the necrosis shows granulomas consistent with an active
	1	fibrocaseous tuberculosis
	2	tuberculoma
	3	tuberculous bronchopneumonia
	4	milliary tuberculosis
	5	tuberculous cavity
10		stains for acid fast bacilli were
	1	positive
	2	negative
11		necrosis is encapsulated in keeping with inactive TB
12		the consolidation shows
	1	an acute broncho-pneumonia
	2	an acute lobar pneumonia
	3	an organising pneumonia
	4	a suppurative pneumonia
	5	a viral pneumonia
	6	diffuse alveolar damage (shock lung)
	7	a fungal infection
	8	an aspiration pneumonia
	9	other (specify) _____

_____

13		foci of broncho-pneumonia are present in some sections
14		the neoplasm is a:
	1	small lung carcinoma
	2	large cell lung carcinoma
	3	squamous lung carcinoma
	4	lung adenocarcinoma
	5	broncho-alveolar carcinoma
	6	metastatic neoplasm
	7	chondroid hamartoma
	8	other lung tumour _____

_____

15		the pleura shows a mesothelioma
	1	epithelial
	2	sarcomatous
16		the cavity is:
	1	an inactive tuberculous cavity
	2	a bronchiectatic cavity
	3	an abscess cavity
	4	other (specify) _____

17		the palpable foci shows:
	1	non-specific fibrosis
	2	non-specific fibrosis with elastosis
	3	nodular muscular hyperplasia
	4	parenchymal lymph nodes
18		the pleura shows:
	1	an active tuberculous pleurisy
	2	a chronic non-specific pleurisy
	3	a fibrinous pleurisy
	4	an empyema
	5	a fibrino-haemorrhagic pleurisy
	6	fibrosis with elastosis consistent with a Medlar's cap

19		necrosis shows features of an infract
20		bilharzia is present
21		there is intra-alveolar
	1	haemorrhage
	2	oedema
22		tissue has been taken for asb fibre count
23		additional comments _____

Heart

the sections show:

1		a tuberculous pericarditis
2		an acute myocardial infarct
3		additional comments _____

Bronchi and bronchioles

1		chronic bronchiolitis
	1	cannot be assessed due to poor preservation
	2	is absent
	3	insignificant
	4	moderate
	5	marked
2		bronchial gland hyperplasia
	1	cannot be assessed due to poor preservation
	2	cannot be determined, main bronchi are absent
	3	absent
	4	insignificant
	5	moderate
	6	marked
3		The Reid Index is ☐ , ☐☐
4		additional comments _____

Oesophagus

1		sections show a squamous carcinoma
2		additional comments

I am of the opinion that

there is

1		silicosis
2		asbestos related disease
3		coal workers' pneumoconiosis
4		mixed dust fibrosis
5		other (specify)

2		additional comments

Comment

1		the service has been noted
2		additional comments

1		Date of review
2		Reason for review

23		Pathologist 1

2

3

4

5

23		the case has been reviewed and
1		the original findings are confirmed
2		the findings have been amended as follows (specify)

Bureau number

P number

Year	Mine	Occupation	Remarks

Dust exposure totals:

High dust _____
Low dust _____
Surface dust _____
Non-dust _____
Gaps _____
Grand total _____

Calendar period: 19__ to 19__/___ years ___ months

Certification

_____
_____
_____
_____
_____
_____
_____
_____
_____
_____
_____

APPENDIX 6

University of the Witwatersrand Human Research Ethics clearance certificate

UNIVERSITY OF THE WITWATERSRAND, JOHANNESBURG

RECEIVED 2005

Division of the Deputy Registrar (Research)

HUMAN RESEARCH ETHICS COMMITTEE (MEDICAL)
R14/49 Nelson

CLEARANCE CERTIFICATE	**PROTOCOL NUMBER M050228**
PROJECT	Occupational Respiratory Diseases: Rates Trends and Risks in Platinum & Diamond Miners Coming to Autopsy 1975-2005
INVESTIGATORS	Ms G Nelson
DEPARTMENT	School of Public Health
DATE CONSIDERED	05.02.25
DECISION OF THE COMMITTEE*	Approved subject to submitting written permission from the mine

Unless otherwise specified this ethical clearance is valid for 5 years and may be renewed upon application.

DATE 05.03.01 **CHAIRPERSON** .....
(Professor PE Cleaton-Jones)

*Guidelines for written 'informed consent' attached where applicable

cc: Supervisor : Dr G Candy

DECLARATION OF INVESTIGATOR(S)

To be completed in duplicate and **ONE COPY** returned to the Secretary at Room 10005, 10th Floor, Senate House, University.

I/~~We~~ fully understand the conditions under which I am/~~we are~~ authorized to carry out the abovementioned research and I/~~we~~ guarantee to ensure compliance with these conditions. Should any departure to be contemplated from the research procedure as approved I/~~we~~ undertake to resubmit the protocol to the Committee. **I agree to a completion of a yearly progress report.**

PLEASE QUOTE THE PROTOCOL NUMBER IN ALL ENQUIRIES