

off as unrecoverable. In Chapter nine, estimates of the size of this burden are calculated. Lewis and Jeebhay argue that to avoid having to write off the costs of occupational disease as unrecoverable, the compensation systems for mining and non-mining systems should be consolidated, so that sufficient resources can be provided to pay compensation that is owed to ex-mineworkers.¹⁶ Such consolidation would, however, violate the environmental principle that the polluter should pay. Cross subsidisation of mining would, furthermore, remove the financial incentives to correct the problem of hazardous mine environments. Erlich *et al* argue that the prevention of occupational disease is an implicit function of workers' compensation.¹⁷ An inefficient system of medical surveillance and compensation hinders disease prevention because:

"One of the most effective disincentives to hazardous working conditions is employer cost. Failure to compensate occupational disease means that the full costs are not borne by the employers ... The cost of pneumoconioses, noise induced hearing loss, occupational cancers and others are thus borne by the individual and frequently by public sector medical facilities. The economic link between hazard and disease is lost."

Inadequate reporting of occupational disease also threatens the actuarial foundation of the compensation fund, considering that it can lead to inadequate provision being made to cater for the true extent of the problem. This thesis will prove that there has been considerable under-reporting of occupational lung disease. Historical rates of compensation are, therefore, not an accurate basis from which to calculate the rate of drawdown on the compensation funds. White¹⁸ states that annual reports from the MBOD show that through the 1980s, between one and a half and four thousand men were certified as having silicosis compensable under the ODMWA. He notes that the prevalence of compensable pneumoconiosis from the Thamaga study is 52.6 per 1000 ex-mineworkers. White comments that the results from Thamaga suggest that if two million men¹⁹ have worked in the South African gold mines since 1973, it can be estimated that 96 200 compensable silicosis cases were not reported to the Director of the MBOD. If half of these men are still alive and each were to claim R30 000, this would represent a liability of R2.8 billion. The prevalence of pneumoconiosis in Libode and the potential unpaid liability as extrapolated from the results of this study will be

¹⁸ This denominator estimate was arrived at with the knowledge that 1353 000 novices have been recruited since 1973 and there have been 378 000 re-engagements since 1973.

examined later in this thesis.

In the preceding sections the concept of the burden of disease has been extended to include the impact thereof on the economic and social viability of households and communities. One mechanism for keeping down the cost of labour involves the externalising of the costs of reproducing labour, but by doing this, the mining industry violates the important ecological and economic principle that the polluter should pay. The next section will examine the economic and social effects at a household level of externalising the costs of occupational disease.

4.4 Costing the burden of occupational disease

Externalising occupational disease from the mining industry can be seen to have various short and long-term benefits and disadvantages for different sectors of society. There are levels on which the externalising of costs can be thought of as having been beneficial to South African society. Low expenditure on dust reduction and dust levies for the Compensation Fund, both contribute to a lower cost of gold production. The benefits of this lowered cost of production are passed on to shareholders who reinvest the funds. The benefits of a profitable mining industry are felt by the state in terms of incoming tax revenue. All members of South African society benefit from state expenditure made possible by taxation, although it must be noted in this context that many labour sending areas are not in South Africa and hence do not share in these benefits. The benefits need to be seen against both the historical and current cost of occupational disease compensation and also the effects of externalising the cost of disease, without adequate compensation, to the labour sending areas.

There is a growing body of literature which examines the cost of disease at various societal levels. This literature can be broadly divided into that dealing with the topic within a qualitative analytical framework and that examining the problem quantitatively.

4.4.1 Qualitative assessment of the cost of disease

Andreoni argues that society is made up of individuals linked together by bonds of family, village, tribe, etc.¹⁹ The economic viability of each worker is linked to that of other members of his community and the consequences of disease for the economic viability of the individual, have a series of down-stream effects through the household to the broader community and ultimately to state organisations financed through the fiscus. While there is

general recognition of the tremendous impact of disease on households and communities, there have been few successful attempts to document this impact. Moreover, the considerable amount of attention paid to the effects of disease at the level of the individual, may have led to neglect by health services of the worst-off sections of the community. Analysis of disease at the level of the household, rather than at the level of the individual, allows for insights into social processes and the larger picture of dynamics that lead to impoverishment of households and communities.

When a worker is diseased, the primary level at which this is felt, is within the family. This fact is recognised in the ODMWA, which provides for the compensation of widows and dependants of those found to have occupational disease at autopsy. In a socio-economic setting, such as the Transkei, where there are very limited job opportunities for women and where men are highly dependent on the mining industry as a source of employment, the consequences of mining disease on the welfare of all members of the household can be considerable. Rural health research in developing countries has tended to focus on studies of pregnant women and pre-school children, both having been identified as vulnerable groups. However, in labour sending areas with high rates of occupational disease, it might be more appropriate to focus on the health of breadwinners, since this impacts on the viability of the family as a whole. One of the key sources of vulnerability for women and children, is heavy dependence on male breadwinners who, in turn, are vulnerable to disease and injury in the workplace and then face a consequent loss of earnings.

As noted above, Ackles has argued that in a context of low levels of education and high levels of dependency on physical labour as a source of employment, even a relatively minor degree of disability can be a major impediment to employment. Chambers argues:

"the body is the poor person's greatest and uninsured asset. If this asset is devalued or ruined, from being an asset it becomes a liability that has to be fed, clothed, housed and treated. A livelihood is destroyed and a household is made permanently poorer."²⁰

It was noted in chapter two that there has been very little work on the social and economic impact of occupational disease in South African ex-mineworkers, but that some of the issues that need to be considered in order to understand the impact of this disease, have arisen out of studies on other diseases in other countries. Corbett comments that chronic disease in an economically active person can result in loss of income and often total loss of employment.

This loss of income includes both loss of the current wages and also loss of potential future earnings.²¹ Thus, chronic disease can adversely affect dependency ratios of a household. Disease in a breadwinner not only reduces income but also requires diversion of time and financial resources on the part of other family members into caring for the sick person. That portion of labour power devoted to nursing is lost to the family for subsistence or income-producing activity. In a peasant society, loss of labour power to care-giving can result in reduced self-sufficiency with regard to food. Foster argues that this loss of labour is highly deleterious where maize (the staple crop in the Transkei) is grown because maize production has peak periods demanding high labour input. A shortage of labour at these peak periods can adversely affect the harvest.²²

Corbett argues further that in addition to loss of current income, future earnings and agricultural labour, there are also other financial implications.²³ Older children are often taken out of school, either to care for the sick person or to reduce pressure on the domestic budget. This is often accompanied by a diversion of moneys into medical treatment, and transport costs to obtain such treatment, which leads to impoverishment and the sale of assets. Once assets are gone, the chronically incapacitated household becomes deeply indebted. Foster has argued, in relation to the households caring for people with AIDS in Zambia, that the sale of assets results in households being very vulnerable to future shocks such as drought. Therefore, the opportunity cost of disablement of a breadwinner is a factor greater than the sum of lost earnings.

Evans undertook a study of the impact of onchocerciasis on the work capacity of breadwinners and has shown that the subsequent negative impact of disease on the nutritional status, health and life expectancy of young, developing households, is considerable.²⁴ Pryer undertook a study of households with a chronically sick breadwinner in Bangladesh.²⁵ She concluded that children in such households were at three times greater risk of malnutrition than those without a sick breadwinner. Furthermore, Pryer has shown that in households with an incapacitated breadwinner not only the young children but most members of the household were malnourished. She advocates reorientation of emphasis by development agencies mandated to improve the health of women and children, towards services for all household members. Damage to members of households with a disabled breadwinner affects the present and the long-term future health of individuals because of social and biological processes. Barker and Osmond argue that poverty experienced in childhood can be manifested in the

causes and timing of deaths 60 years later.²⁶ Pryer is in favour of the development of community-based insurance and credit schemes to ease the financial burden incurred by sickness-induced loss of income. Pryer also highlights the dependency of women and children in her study group on male good health and earning capacity. She argues for an expansion of programmes aimed at promoting economic independence for women.

The literature cited above shows that occupational disease in a breadwinner can have negative consequences for individuals, households and the broader community. Classical epidemiology has failed to adequately assess how women and children and the broader community can be affected by social and health stressors in potential breadwinners. Such factors are seldom taken into account when assessing health outcomes.

This study documents the impact of mining related lung disease on ex-mineworkers, their families and the Libode community. In previous chapters, it has been argued that Libode is a community in which there are few economic opportunities for women and there is a consequent dependence on male breadwinners. Male breadwinners in Libode have low levels of education and limited economic opportunities outside of the mining industry and as a result, are highly dependent on mining as a source of employment. Previous chapters have argued that mineworkers are very vulnerable to occupational disease and injury. Consequently, the viability of many households in Libode is precarious. Subsequent chapters will assess the social and economic consequences of the loss of labour power, the reduction in income, and the diversion of time and financial resources of other family members into caring for the ex-mineworkers who have developed occupational lung disease.

4.4.2 Quantitative assessment of the cost of disease

The impact of occupational disease in Transkeian breadwinners is largely hidden at the national level as mining wages are low relative to wages for other industrial workers and even in-service workers do not contribute a significant amount of personal income tax revenue. The reduction in life expectancy is not expressed either on the revenue side as lost contributions to the fiscus, or on the expenditure side as claims on the pension system, because many people die before they are eligible to claim. Where costs are evident is in the industry and national health budgets for, notably, disability grants and tuberculosis treatment facilities and resources (staff, laboratory, drugs, etc.).

Several studies in a number of countries have attempted to quantify the cost of occupational

causes and timing of deaths 60 years later.²⁶ Pryer is in favour of the development of community-based insurance and credit schemes to ease the financial burden incurred by sickness-induced loss of income. Pryer also highlights the dependency of women and children in her study group on male good health and earning capacity. She argues for an expansion of programmes aimed at promoting economic independence for women.

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Several studies in a number of countries have attempted to quantify the cost of occupational

disease and accidents in relation to Gross Domestic Product (GDP). Andreoni argues:

"Occupational accidents and disease are costly. For the community as a whole, they represent a burden which is constantly growing and which affects the standard of living of everyone, to the point that one can well ask whether the money spent paying for them would not be better used in financing the efforts of all those who throughout the world, are devoting their knowledge and their research efforts to prevention."²⁷

This is one good reason among many why we should know as accurately as possible the real cost of employment accidents and occupational diseases. The results of research and the statistical data published in many countries, especially industrialised ones, provide a certain amount of information which, however, shows how difficult it is to draw up a precise list of the numerous elements which make up this cost. While some of these elements can be isolated and quantified easily, others are much more difficult to perceive accurately.

Andreoni gives an indication of the ratio of costs of occupational disease and accidents to GDP for several developed countries but he does not distinguish costs of accidents and occupational disease. The cost ratio to GDP varies from 1.2 per cent for the USA (1977), to 2.0 per cent for Italy (1980) and 3.3 per cent for France (1977).²⁸

A 1994 study by the British Health and Safety Executive (HSE) has examined the cost of occupational disease and injury to the British economy.²⁹ In this study, the costs to employers, and also the costs to society of occupational disease, are listed and quantified. The key costs to employers are the payment of compensation levies; costs of medical treatment provided by the employer; administrative costs; medical and compensation services provided by the employer; loss of output (due to absence of sick workers); additional costs of maintaining output when workers are absent; costs of hiring and training new workers to replace those lost because of disease; penalties payable for delays in meeting contractual obligations; and loss of goodwill and reputation with the workforce, customers and the local community. The HSE study found that the total cost of occupational accidents and disease is far higher than had previously been thought because earlier studies had failed to take account of societal costs, which include the loss of resources (notably labour and capital), loss of future labour services from the victims, and loss of well-being due to pain, grief, and suffering caused by premature death, and disablement. The HSE report states:

"the overall cost to the British economy of all work accidents (including avoidable non-injury accidental events) and work-related ill health is estimated to be between 6 and 12 billion pounds. This is equivalent to between 1 per cent and 2 per cent of total Gross Domestic Product ... The total cost to society as a whole, including the estimate made for loss of welfare resulting from pain, grief, and suffering of individual victims and their families is estimated at between 11 billion and 16 billion pounds. This is equivalent to between 2 per cent and 3 per cent of total Gross Domestic Product, or a typical years economic growth. Illness accounts for about 4 to 5 billion pounds of this and accidents 6 to 11 billion pounds."³⁰

While one must be cautious about direct extrapolation from a British model to a South African economic and industrial health context, one can use this model to tentatively assess the effect on the national economy of occupational disease and accidents. South Africa's 1996 GDP was R483 billion, hence 2 per cent of GDP is R15 billion, and 3 per cent is R19 billion. This suggests that occupational health and safety costs have a major effect on the national economy. The HSE model provides a method of estimating figures for the cost of occupational disease. This model can be applied to occupational lung disease in the South African gold mining industry. Using the costs to the economy itemised in the HSE report and assuming a work force of 350 000, the values shown in Table 4.1 are obtained.³¹

Table 4.1 Direct, internalised costs of occupational lung disease to South African gold mining industry at current prices and assuming a total workforce of 350,000 men (following the HSE model)

| HSE Category | HSE Category components | Annual Value (Rands) |
|---|--|----------------------|
| Employers' contribution to compensation fund ¹ | Contribution of gold mine employers to Compensation Fund | 38, 009766 |
| Surveillance ² | Annual periodical examination | 140,000 000 |
| | Loss of output during periodical exam. ¹¹ | 131,250 000 |
| Medical treatment provided by employer | Tuberculosis ³ | |
| | Hospitalisation | 10,710 000 |
| | Costs of drugs for treatment ⁵ | 4,116 000 |
| | Costs of MDR Tb treatment ⁶ | 1,960 000 |
| | Costs of DOTS staff salaries ⁷ | 1,050 000 |
| | Pn and OAD | |
| | Case finding and reporting to MBOD ⁸ | 1,050 000 |
| Loss of output for men with Tb | Salary remuneration | 20,790 000 |
| | Production losses ¹⁰ | 120,960 000 |
| Total costs | | 469, 895 766 |
| Total costs per worker | | 1343 |

Notes

1. Based on contribution from April 1996 to March 1997
 2. Estimated cost R400 per person
 3. Assuming 2 per cent annual incidence
 4. Assuming 30 per cent of incident cases are hospitalised for an average of 17 days at R300 per day
 5. Assuming six months of treatment at R600 per course
 6. Assuming 2 per cent of Tb cases have multi-drug resistance costing R14,000 per course
 7. Staff salaries R2,000 per month; one person per 8,000 men
 8. Including X-rays, medical staff, claim administration at R150 per patient assuming 1 per cent incidence of Pn and OAD respectively
 9. Assuming 54 days off work on average at R55 per day in salary
 10. Assuming R320 per day for 54 days off work on average
 11. Salary costs at R55 per day and production loss at R320 per day
- (Source: personal communications D. Barnes)

The HSE model also provides for a calculation of total societal losses due to occupational disease and disability. There is insufficient South African data to apply the HSE method directly to calculate societal costs of occupational lung disease in gold mineworkers. Applying the relationship between cost to employers and cost to society found in the HSE model (which ranged between 0.54 and 0.66) the total cost would be between R723 million and R780 million per year. In 1996, gold mining contributed 4 per cent of total GDP which was approximately R20 billion, hence costs of occupational lung disease constitute between 3.6 per cent and 4.4 per cent of gold mining's contribution to GDP in 1996. South Africa's total GDP for 1996 was R482 billion, hence costs of occupational lung disease in gold mining represent between 0.15 per cent and 0.16 per cent of total GDP for 1996.

It has been noted above that application of the HSE model is hampered by the very limited data available to calculate societal costs of occupational disease for South African mine labour sending areas. In an attempt to obtain information on the social and economic costs of occupational disease, a questionnaire was administered to those Libode random sample study subjects who were found to be eligible for compensation by the Medical Bureau for Occupational Diseases. These data will be presented in chapter nine.

4.5 Conclusion

This chapter has laid the theoretical groundwork upon for later chapters, in which quantitative models based on disease prevalence data for the study area, will be built. While the direct, internalised costs of occupational lung disease to the mining industry have been shown to be

considerable, it has also been demonstrated that much of the burden is being carried by the rural households. It has been argued that morbidity and premature mortality among breadwinners due to mining-related occupational lung disease has substantially reduced the welfare of affected households, and contributed to the overall impoverishment of labour sending communities such as Libode.

The large legacy of unpaid compensation has been touched upon and it has been noted that the failure to pay compensation has been both a contributory factor to and the result of an inefficient system of medical surveillance, because one of the most effective disincentives to hazardous working conditions is employer cost. In the South African gold mining industry, the economic link between hazard and disease has been weakened to the point of being useless. Internalising the costs of disease would almost certainly result in measures being taken to reduce dust levels, with a concomitant reduction in disease incidence and, therefore, a reduction in the costs to society of disabled ex-mineworkers.

¹ Marx K. *Capital : A critique of political economy*. Volume 1. London: Lawrence and Wishart, 1954.

² Wolpe H. Capitalism and cheap labour power; from segregation to apartheid. *Economy and Society* 1972; 1: 425-444.

³ Cohen R. *The new helots; migrants in the International division of labour*. London: Gower Publishing Company, 1987.

⁴ African Mineworkers Union. Submission to the 1943 Witwatersrand gold mines native wages commission. Quoted in; Allen VL. *The history of black mineworkers in South Africa*. volume 1. Johannesburg: National Union of Mineworkers Publication, 1992: 466.

⁵ Arkles RS. *The social consequences of industrial accidents: Disabled mineworkers in Lesotho*. (Dissertation Master of Arts). Johannesburg: University of the Witwatersrand, 1993.

⁶ Wilson F, Ramphela M. *Uprooting poverty: the South African challenge*. Cape Town: Heinemann, 1989.

⁷ Personal communication, Dr Graham Herbert, Anglo American Medical Division. June 1996.

⁸ Arkles RS. 1993 op.cit.

⁹ Wilson M. So truth be in the field. *The Alfred and Winifred Hoernle Memorial Lecture*: Johannesburg. South African Institute of Race Relations, 1975: 12.

¹⁰ Arkles RS. *The social consequences of industrial accidents: Disabled mineworkers in Lesotho*. (Dissertation Master of Arts). Johannesburg: University of the Witwatersrand, 1993.

¹¹ Phua KH. The economics of occupational health services. In; Jayaraman J, editor. *Occupational health in developing countries*. Oxford: Oxford University Press, 1992:125-135.

¹² Department of Mineral and Energy. *Report of the Commission of Inquiry into safety and health in the Mining Industry*. Leon RN, chairperson. Pretoria: Department of Mineral and Energy, 1995.

¹³ National Union of Mineworkers. Response by the National Union of Mineworkers to the Submission of the Chamber of Mines to the Commission of Inquiry into Safety and Health in the mining industry. Johannesburg:NUM, 1995:33.

¹⁴ Eckholm EP. The picture of health; environmental sources of disease. 2nd rev. ed. New York: Norton, 1977: 154-73.

¹⁵ Robbins D. New study shows high rate of illness among ex-mineworkers. The Star 1996 April 27.

¹⁶ Lewis P and Jeebhay M. The Mines Health and Safety Bill 1996 - A new era for health and safety in the mining industry. Industrial Law Journal 1996; 17(3):311-341.

¹⁷ Erlich R J, White N, Kerfoot W. Compensation for occupational disease: insult to injury. Occupational Health and Safety 1995; 1:4: 18-19.

¹⁸ White N. Dust Related diseases in former miners - the ODMWA legacy. Occupational Health South Africa 1997; 13(4): 20-24.

¹⁹ Andreoni D. The cost of occupational accidents and disease. Geneva: International Labour Organisation, 1986.

²⁰ Chambers R. Vulnerability, coping and policy. Institute of Development Studies Bulletin 1989; 20(2):1-8.

²¹ Corbett J. Poverty and sickness: The high costs of ill-health. Institute of Development Studies Bulletin 1989; 20(2):58-63.

²² Foster S. The economic impact of AIDS and HIV disease. [PhD thesis]. London: University of London, 1994.

²³ Corbett J. 1989 op. cit.

²⁴ Evans T. The impact of permanent disability on rural households: river blindness in Guinea. Institute of Development Studies Bulletin 1989; 20(2):41-49.

²⁵ Pryer J. When breadwinners fall ill: Preliminary findings from a case study in Bangladesh. Institute of Development Studies Bulletin 1989; 20(2):49-58.

²⁶ Barker DJP, Osmond C. Infant mortality, childhood nutrition and ischaemic heart disease in England and Wales. Lancet 1986; 1:1077-1078.

²⁷ Andreoni D. The cost of occupational accidents and disease. Geneva: International Labour Organisation, 1986.3.

²⁸ Andreoni D. 1986 op. cit.

²⁹ Davies NV, Teasdale P. The cost to the British economy of work accidents and work related ill health. London: Health and Safety Executive Publication, 1994.

³⁰ Davies NV, Teasdale P. 1994 op. cit. 27.

³¹ Information supplied by a mine medical officer, April 1998.

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- ¹³ National Union of Mineworkers. Response by the National Union of Mineworkers to the Submission of the Chamber of Mines to the Commission of Inquiry into Safety and Health in the mining industry. Johannesburg:NUM, 1995:33.
- ¹⁴ Eckholm EP. The picture of health; environmental sources of disease. 2nd rev. ed. New York: Northon, 1977: 154-73.
- ¹⁵ Robbins D. New study shows high rate of illness among ex-mineworkers. *The Star* 1996 April 27.
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- ¹⁷ Erlich R J, White N, Kerfoot W. Compensation for occupational disease: Insult to Injury. *Occupational Health and Safety* 1995; 1:4: 18-19.
- ¹⁸ White N. Dust Related diseases in former miners - the ODMWA legacy. *Occupational Health South Africa* 1997; 13(4): 20-24.
- ¹⁹ Andreoni D. The cost of occupational accidents and disease. Geneva: International Labour Organisation, 1986.
- ²⁰ Chambers R. Vulnerability, coping and policy. *Institute of Development Studies Bulletin* 1989; 20(2):1-8.
- ²¹ Corbett J. Poverty and sickness: The high costs of ill-health. *Institute of Development Studies Bulletin* 1989; 20(2):58-63.
- ²² Foster S. The economic impact of AIDS and HIV disease. [PhD thesis]. London: University of London, 1994.
- ²³ Corbett J. 1989 op. cit.
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- ²⁶ Barker DJP, Osmond C. Infant mortality, childhood nutrition and ischaemic heart disease in England and Wales. *Lancet* 1986;1:1077-1078.
- ²⁷ Andreoni D. The cost of occupational accidents and disease. Geneva: International Labour Organisation, 1986:3.
- ²⁸ Andreoni D. 1986 op. cit.
- ²⁹ Davies NV, Teasdale P. The cost to the British economy of work accidents and work related ill health. London: Health and Safety Executive Publication, 1994.
- ³⁰ Davies NV, Teasdale P. 1994 op. cit. 27.
- ³¹ Information supplied by a mine medical officer, April 1998.

CHAPTER 5

Dust and Occupational Disease in the South African Gold Mining Industry

5.1 Introduction

Dust is defined by the International Labour Organisation as "an aerosol composed of solid inanimate particles".¹ In mining, the mechanical disintegration of matter that results in dust is generated predominantly during the breaking up and transportation of rock. The first section of this chapter will analyse the four factors which determine the nature of the health risk arising from exposure to dust, assess available evidence concerning to dust levels on South African gold mines and set out the legal framework pertaining to dust control. The second section of this chapter will discuss the pathogenesis, clinical manifestations and epidemiology of the key occupational diseases arising from silica dust exposure on the South African gold mines.

5.2 The health risk posed by dust

The key dust-generating activities in mining are drilling, blasting and mechanical loading. The extent to which dust inhaled during minework poses a health risk, is determined by four interlinked factors: particle size, mineralogical composition, concentration, and duration of exposure.²

In order for dust to create a risk in terms of occupational lung disease, it must be deposited in the lung alveoli. There are a range of pulmonary defence mechanisms which prevent this from happening and as a result, not all dust that is inhaled is hazardous to health. Particles of varying sizes lodge in different regions of the lungs according to their aerodynamic size. Large particles are captured by the mucous-covered surfaces of the nose and are expelled rapidly. On the other hand, dust particles which are small enough to penetrate the alveolar region of the lungs, pose a health risk. Such particles are associated with pneumoconiosis. It would seem that the important size range which results in deposition in the alveolar region of the lungs is between 0.4 and 7.0 μm and this is referred to as the respirable dust range. Particles smaller than 0.4 μm are expired and are not deposited.³ It is thought that the exact

distribution of particle sizes deposited in alveoli in relation to total inhaled dust, will correspond with the curves initially suggested by the British Medical Research Council in 1952. This standard was subsequently adopted by the International Conference on Pneumoconiosis held in Johannesburg in 1959.⁴ In terms of this curve, dust-measuring equipment has been developed to collect particles within the range 1µm to 7µm.

The mineral composition of dust particles varies by mine type and not all respirable dust causes reaction in lung tissue. In gold mines, the key health hazard in dust is biologically active, free crystalline silica, a compound of silicon and oxygen.⁵ Silica is the most abundant mineral on Earth, comprising 28 per cent of the Earth's crust. It occurs in a tetrahedron form made up of a central atom of silicon connected to four oxygen atoms, each of which is, in turn, connected to a silicon atom. The spatial arrangement of the silicon atoms determines the different forms of silica. The commonest form of crystalline silica is quartz. The high rate of silicosis peculiar to the Witwatersrand gold mines is in part due to the exceptionally high silica content of the reef formations. Hnizdo reports that in South African gold mines, about 60-80 per cent of virgin rock is silica and approximately 30 per cent of respirable mine dust is free silica.⁶ It should be noted that there can be variations in quartz concentration of respirable dust as there are variable concentrations of quartz in the ore body.⁷ The implications of this variability for occupational medicine and occupational hygiene surveillance on South African gold mines, will be discussed later in this chapter.

The concentration of dust particles is an important determinant of health hazard. Threshold Limit Values (TLV) have been set for the safe concentration of airborne dust and are a time-weighted average concentration for an 8-hour day and a 40-hour week and to which workers can be exposed indefinitely with an adverse health-effects risk of less than one per 1000. TLVs are published by the American Conference of Government Industrial Hygienists and are based on knowledge acquired from animal experimentation and through industrial epidemiology. The current TLV for respirable silica is 0.1 mg/m³.⁸ The South African limit for respirable silica dust in the mining industry is legislated to be 0.1 mg/m³.

It is difficult to establish precise relationships between exposure to silica dust and the subsequent development of silicosis. Some studies on in-service workers support the existing TLV standard of 0.1mg/m³ but other studies suggest that the long latency period between exposure and disease may have lead to an over-estimate of safe TLV levels.^{9,10} There is a growing body of evidence to suggest that silicosis progresses for many years after the

cessation of silica exposure. A study of retired, white South African gold miners found that the risk of developing silicosis increased from 0.5 per cent at a cumulative exposure of $0.1\text{mg}/\text{m}^3$ years of silica to 27 per cent at $3\text{mg}/\text{m}^3$ years and 82 per cent at $15\text{mg}/\text{m}^3$ years, assuming that the dust contains 30 per cent silica.¹¹ A study of death certificates for South Dakota gold miners predicted that a cumulative exposure of $4\text{mg}/\text{m}^3$ years would result in a lifetime risk of silicosis of 47%.¹² A survey of retired Colorado miners found that the cumulative risk of developing silicosis increased from about 25 per cent at a cumulative exposure of $1\text{mg}/\text{m}^3$ years to 58 per cent at $3\text{mg}/\text{m}^3$ years.¹³ The data provided by Hnizdo *et al.* indicate that there is a substantially lower risk of silicosis at dust levels below $2\text{mg}/\text{m}^3$ than was found in the Colorado and South Dakota studies. One possible explanation for these differences and also for differences involving the Libode data from the present investigation, will be discussed in chapter seven. These surveys reveal the need for lifetime follow-up to assess the full health burden of silica dust exposure. As a result of these studies, the National Institute for Occupational Safety and Health (NIOSH) has recommended that TLV should be adjusted to $0.05\text{mg}/\text{m}^3$.¹⁴

All of the above work was based on the assumption that if one was exposed to $0.1\text{mg}/\text{m}^3$ for a standard working year then the cumulative exposure would be $0.1\text{mg}/\text{m}^3$ years. However, the concept of a standard working year is problematic, given that we know that there is wide variability, with very high peak concentrations of dust within a shift.¹⁵ There are also variations in the quartz concentration of respirable dust because of the variation in the concentration of quartz in the ore body.¹⁶ Furthermore, studies undertaken in experimental animals suggest that lung clearance mechanisms are less effective when the lung burden (i.e. the amount of a particular substance in the lung) exceeds a given threshold and that the rate of clearance associated with such an overload is slower than that of normal pulmonary clearance.^{17,18,19} Hence, the notion of a standard working year may not provide useful data when one is trying to assess the relationship between silica dust exposure and the subsequent development of silicosis.

TLV standards have been criticised for several reasons. First, section 4.14.1 of the regulations of the Mine Health and Safety Act, Act 29, 1996, which was brought forward from the Minerals Act, Act 50, 1991, which was, in turn, brought forward from the Mines and Works Act, Act 27, 1956 (variously amended), restricts underground risk work to 48 hours per week plus 60 minutes daily travelling time rather than the 40 hours on which the TLV is based.

Secondly, TLV international standards are based on a normal respiration rate rather than on the increased respiration rate that is associated with extremes of depth (and associated heat) of the South gold African mines. The depth and heat is likely to cause an increase in the ventilation requirements of South African gold mineworkers for any given work load. And places them at a further disadvantage vis a vis other hard rock miners working at lesser depth. Consequently, the TLV formula fails to adequately protect South African mineworkers.²⁰ Thirdly, Castleman has argued that there has been undue corporate employer influence over the setting of the TLVs.²¹

Duration of exposure has been shown to be a key factor in determining the degree of health hazard. Beadle correlated dust samples, work histories and medical data in a cohort of white South African workers who started employment between 1934 and 1938.²² There was no known history of occupational dust exposure outside the gold mines and the mineworkers had worked at least 3000 shifts. A correlation was found between a radiological diagnosis of silicosis and duration of exposure. These data suggest that after after 3000 shifts (approximately 10 years), there is a risk of silicosis of 2 per cent and after 9000 shifts (i.e. approximately 30 years), there is a cumulative risk of silicosis of 40 per cent.²³ Hnizdo has subsequently used Beadle's dust measurements to correlate dust exposure to a set of x-rays taken from a more extensive cohort of workers with a longer follow up period.²⁴ Beadle's data and Hnizdo's data are consistent with each other (as would be expected given that they use the same dust data). However, the Hnizdo study shows a higher cumulative risk of disease. Hnizdo suggests that at $0.5\text{mg}/\text{m}^3$, the cumulative risk of silicosis after 10 years is 5 per cent and after 30 years, it is 80 per cent. The higher cumulative risk in Hnizdo's surveys is probably due to the longer latency period from first exposure to manifestation of radiologically evident disease (this cohort having been followed for a longer time). Hnizdo reports an average latency period of 36 years, whereas Beadle followed up for 30 years, on average, suggesting that Beadle's cohort might have subsequently developed disease that was not radiologically apparent in 1969.

The above data are somewhat compromised by the fact that information on current dust levels for underground air that is representative of South African gold mines, as well as details concerning exposure levels of workers, are unsatisfactory in a range of ways. The report of the Leon Commission points out that data relating to dust levels:

"still relies heavily on the original studies carried out by Beadle and amplified and commented on

Such data must be treated with caution, because they rely on a study that is almost 30 years old and on an untested assumption that dust levels have not changed in this time period. Furthermore, the measurements for the Beadle study were made on white miners. Earlier chapters have shown that legally enforced job reservation excluded black workers from all but the dustiest jobs, but in using Beadle's data, there is an untested assumption that exposure to dust did not and does not differ by race; that there is no genetic variability in the ways in which black and white workers respond to silica; and that there is no variability pertaining to the prevalence of other diseases that may interact with pneumoconiosis (e.g. tuberculosis) among the two groups. Furthermore, the jobs done by mineworkers in South Africa, have been determined largely by race. Different jobs will result in exposure to different processes. This may result in exposure to differing levels of toxicity. Castranova *et al.*²⁶ have shown that freshly generated dust containing crystalline silica will exhibit increased toxicity in lung cells compared with aged dust. It is thought that this increased toxicity is due to the recent creation and production of surface free radicals. Thus, workers engaged in different processes, who will come into contact with dust of more or less recent creation, may be at different risk of disease. In later chapters, these issues will be discussed in relation to disease prevalence data pertaining to the Libode study group and comparisons of this group with earlier studies of white South African mineworkers will be made.

It can be argued that the key reason that there is such a heavy reliance on Beadle's data is that all subsequent measurements of dust levels have been carried out for the purpose of determining a Risk Rating in terms of the Occupational Diseases in Mines and Works Act (ODMWA). The Risk Rating is calculated by averaging gravimetric samples collected off statistically selected personnel.²⁷ This is an averaging process which cannot detect any peaks in dust concentrations. Such a method cannot be effective in identifying specific, high, personal, occupational or workplace dust levels. Furthermore, these measurements have been carried out under the supervision of mine management and are used only to calculate an air quality index for the whole mine which is, in turn, used to determine the average risk for the entire mine. The raw data are not in the public domain and it is difficult to evaluate the significance of these measurements because a standardised sampling method is not followed. A recent report in the *Journal of the Mine Ventilation Society of South Africa* states that the results of the dust sampling programme:

"have not yielded results that are meaningful for risk calculations nor for equitable risks. Furthermore, the results are not meaningful for dust control in the workplace, for evaluating control measures, nor for identifying individuals, workplaces or operations with unsatisfactory exposure or dust levels. The reports submitted by the mines to the DME are also unsatisfactory for epidemiological research or studies, mainly due to the fact that they are generated with respect to various mining activities and not for specific occupations."²⁸

A recent actuarial report on the ODMWA Compensation Fund sought to assess the link between cumulative exposure to dust (i.e. the theoretical basis of the risk levy system) and the actual claim patterns of mineworkers.²⁹ The average risk index (weighted by the number of shifts worked) for each mineral group within the South African mining industry for the period 1979 to 1993 and also the average cost of occupational disease claims (weighted by the number of risk shifts worked) was calculated. This study found that there was no statistically significant link between compensable disease benefit payments (even after excluding tuberculosis) and average risk indices. The authors of the report stated that:

"although cumulative exposure to harmful concentrations of specific airborne particulates is a significant cause of non tuberculosis compensable diseases, the current system of gravimetric sampling does not accurately reflect the relative level of risk. The measurement process must be reinvestigated and redefined in such a way that it accurately reflects relative risk levels."³⁰

This report supports the conclusion reached in chapter 2 that the current system of dust measurement, risk assessment and levy implementation does not provide an incentive for mining companies to improve working conditions.

Given the lack of alternative sources of data (and in view of their limitations discussed above) the Risk Rating data kept by the South African Government Mining Engineer merit consideration.

Because of the relative lack of alternative data, this thesis will also use measures based on Beadle's work. Hnizdo amplifies and comments on the data provided by Beadle and indicates that underground air representative of deep-level South African gold mines, contains 0.33-1.38mg/m³ total dust and 0.23-0.97 mg/m³ respirable dust, with the silica content of respirable dust ranging from 20-62 per cent i.e. 0.05-0.6 mg/m³. The average respirable dust concentration to which the miners (white) are exposed in high dust occupations, is 0.48

mg/m³; in medium dust occupations, 0.30mg/m³; and low dust occupations, 0.20mg/m³.

Trends in dust levels determine the extent to which the health hazard is increasing or decreasing. White notes that dust control measures resulted in a significant reduction of respirable dust in South African gold mines in the early 20th Century and that by 1940, as a result of this reduction, the number of deaths per thousand miners had declined markedly.³¹

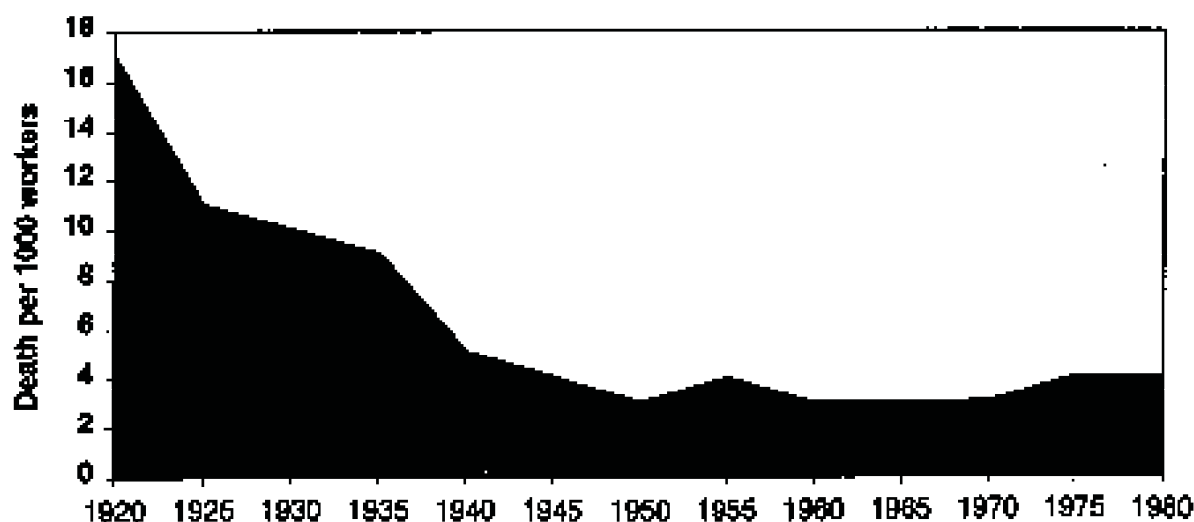


Figure 5.1 Deaths due to occupational lung disease per 1000 South African gold mineworkers, 1920-1980

Source: Leger, JP: 1995.³²

The debate as to whether dust levels have risen, fallen or have remained static over the past 30 years, is characterised largely by argument by assertion rather than by presentation of scientifically provable data. In 1969, Beadle stated that there was no evidence for a reduction in underground dust levels on South African gold mines in the period since 1938.³³ This may still be the case, because a recent autopsy survey, based on data for the period 1975-1991, shows no statistically significant trend in the prevalence of silicosis due to factors other than increasing age and years of service. This stasis indicates that the dust levels on South African gold mines have remained unchanged during the 17 years of the study.³⁴ Hnizdo considers that dust levels have dropped slightly since 1970 and attributes this to the establishment of a compensation fund into which each mine contributes a levy based on dust levels.³⁵ Conversely, the Chamber of Mines Research Organisation (COMRO) analysed dust submissions compiled in terms of the risk assessment requirements of the ODMWA for the period 1964 - 1982.³⁶ It was concluded that since 1964, dust levels have dropped on South

African mines but that in the late 1970s, conditions underground deteriorated. The report singles out tips, loading boxes and stopes as the key areas where deterioration in dust conditions has taken place. The paper is somewhat unhelpful as it does not state how the dust control was implemented or what the reasons for the subsequent deterioration were.

It must be noted that all of the South African dust data described above were collected using konimetric sampling techniques. Konimetric sampling involves taking small, non-representative air samples by way of hand-held devices loaded with a glass slide which has been pre-treated with vaseline xylol. A plunger is released and air is sucked at high velocity and impinged onto the slide. Ignition and emersion treatment is used to remove water-borne salts and carbon particles from the slide, which is then examined under a microscope and the number of particles per millimetre are counted. Gravimetric sampling involves a filter inserted into a cartridge which has a separator cyclone in order to ensure that only respirable particles can reach the filter. Air is then sucked through the filter at a rate of 1.9 litres per minute. Gravimetric sampling measures dust exposure over an eight-hour working shift. The filter is weighed before and after the shift and dust is measured in terms of mg per cubic metre.³⁷

Konimetric sampling was abandoned internationally in the 1970s in favour of gravimetric sampling, which provides a shift-long representative evaluation of dust levels. Konimetric sampling was abandoned in South Africa in 1992. Early versions of gravimetric devices were unable to withstand the harshness of South African mining conditions, in terms of the underground temperature and pressure variations. Gravimetric pumps have now been designed to withstand the South African mining environment.

No reliable conversion exists for changing konimetric measuring units into gravimetric measuring units.³⁸ Konimetric data are measured in terms of the number of particles per mm and thus cannot be converted into mg/m³. Furthermore, the sampling strategies are not comparable (konimetric sampling strategies are based on point sampling and gravimetric strategies are based on shift-long exposures). The continued use of the konimeter in South Africa until 1992, makes it difficult to compare South African conditions with those in other mining countries. It also makes it difficult to compare current conditions with those in earlier reports.

Konimetric techniques have been criticised for several reasons. Beadle comments that:

"when sampling a high concentration of fine dust the konimeter will have a low efficiency and the

recorded results will be significantly lower than the corresponding thermal precipitator count. When sampling a low concentration of coarse particles the konimeter will appear to have a high efficiency and will sometimes give a higher count than that given by the thermal precipitator."³⁹

Thus, a konimeter may not measure the concentration of small respirable particles accurately. Beadle notes that "results obtained with the konimeter may therefore be very misleading, and a more exact way of determining dust concentration is desirable."⁴⁰ Kerrich⁴¹ showed that if two konimeters were used simultaneously, side by side, the results would differ significantly. He also found that the ratio between the two konimeters varied in an unpredictable way. Felix has noted that in Department of Mines dust surveys, four spot samples were taken in each work place and the average recorded.⁴²

The above data show that South Africa (the world's largest gold producer in the 1970s, employing the largest number of gold mineworkers) continued to use unscientific and outdated konimetric dust sampling methods long after the rest of the world had moved to gravimetric techniques.

Given the above, in order to identify high-risk groups and to evaluate the efficacy of current dust control measures, there is a need for a new 'Beadle study', namely, an industry-wide survey of incidence and prevalence of occupational lung disease in relation to dust exposure. This could either be for individuals in specific jobs, or at least for occupational categories of workers using gravimetric dust-measuring tools.

Earlier in this chapter, several studies which analysed the relationship between exposure to silica and the development of silicosis, were quoted and evaluated. Hnizdo's survey of retired white South African gold miners, which indicated that the cumulative risk of developing silicosis was 0.5 per cent at a cumulative exposure of $1\text{mg}/\text{m}^3$, was compared with a study of retired workers from Colorado, USA, in which the cumulative risk of silicosis was 25 per cent at a cumulative exposure of $1\text{mg}/\text{m}^3$. Given that the South African data were collected using konimetric methods and the American data were obtained by means of gravimetric techniques, it is not clear how comparable such studies really are.

5.3 Legislation

Dust levels in the mining industry were until recently, regulated by the Minerals Act (Act 50, 1991) and the ODMWA. The Minerals Act was intended to control dust and the purpose of

the ODMWA was to protect the health of mineworkers through medical surveillance, to set risk ratings based on dust measurements, and to legislate for the collection of a dust levy on the basis of the risk rating. These two Acts represented an artificial separation of medical surveillance from dust control. I would argue that this separation of occupational medicine from occupational hygiene has resulted in a failure to implement effective engineering solutions aimed at dust reduction.

The Mine Health and Safety Act (MHSA), Act 29 of 1996, is a recent piece of legislation (resulting from the recommendations of the 1995 Commission of Inquiry into Safety and Health in the Mining Industry) which is intended to provide tripartite systems and institutions to promote employer, employee and state participation in health and safety issues, so as to provide for effective dust monitoring, inspection and control systems. The tripartite body that drafted the MHSA envisaged that it would considerably reduce the scope of the Minerals Act (which would then regulate only prospecting and rehabilitation of mines) and the ODMWA (which would then regulate only occupational disease surveillance and compensation services for former mineworkers).

The MHSA represents a major departure from the Minerals Act and the ODMWA. Lewis and Jeebhay argue that:

"up until now, the mining industry and previous governments have paid only lip service to the health, welfare, and working conditions of this enormous and vital group of workers. It is therefore absolutely appropriate that the MHSA should virtually coincide with the adoption of the new South African constitution - it can be regarded as a new constitution for the mining industry. As such it is a major achievement in legislative terms".⁴³

The MHSA has the potential to substantially alter the culture and politics of health and safety in the mining industry. However, by late 1998, new regulations for the MHSA were still being drafted, in a lengthy tripartite process, and as such the regulations that were in place for the Minerals Act, were still in force. As has been mentioned earlier in this chapter the regulations from the Minerals Act which have been brought forward to the MHSA, were themselves brought forward from the Mines and Works Act of 1956. Regulations set out in practical detail how the purposes of an Act are to be achieved. Without new regulations, much of the spirit of the MHSA remains legislative intention without a mechanism to stipulate performance and sanction non-performance. The MHSA is an ambitious attempt to develop a

new culture of health and safety and which places onerous duties on employer, employee and state bodies to achieve this worthwhile end. However, it is a matter of concern that this is a major challenge to the parties to give effect to its intention. The strengths and weaknesses of the ODMWA have been discussed in detail in a previous chapter. This chapter will deal largely with the Minerals Act and the MHSA.

The MHSA ends an 85-year-old legal regime governing health and safety in the South African mining industry which has for many years lagged far behind comparable regimes in Europe, North America, and Australasia. The MHSA seeks for the first time in the history of South African mining disease legislation, to link medical records to exposure records and in so doing, to ensure that environmental modifications are made in response to the medical and occupational hygiene surveillance. In Chapter 1, Section 12, of the MHSA, it is noted that qualified occupational hygienists must be engaged by each mine in order to measure exposure to hazards at the mine and that all occupational hygiene measurements must be designed so "that they provide information that the manager can use in determining measures to eliminate, control and minimise the health risks and hazards to which employees are or may be exposed".

The emphasis on environmental modification in the MHSA contrasts with what was contained in the Minerals Act, which was extensively criticised for its emphasis on personal protective equipment rather than on engineering achieved means of controlling the amount of dust present in mines.⁴⁴

The MHSA is also innovative in that it decrees that a record of hazardous work must be kept by the manager of each mine and that annual health and safety reports are also to be compiled by each mine. These reports are to include statistics compiled by an occupational health practitioner and an occupational hygienist, giving an analysis of employee health data based on the records of medical surveillance, exit medicals and exposure hazards. This contrasts with the provisions of the Minerals Act, which required only the submission of dust measurements (which were converted into a risk rating and a financial levy) and no associated health statistics. This change is to be welcomed in view of the fact that South Africa has had a gold mining industry for over a century; and even though dust management mechanisms and regulations have been in place for most of that time, the prevalence of occupational lung disease indicates that these measures have been ineffective.

Furthermore, the MHSA establishes representative, tripartite institutions to promote health and to provide for monitoring systems to improve mine health and safety. The worker participation that is enshrined within the MHSA represents a significant change in attitudes. It was noted in Chapter One that until 1992, there was no employee representative on the, now moribund, Risk Committee, despite the Chamber of Mines representation on this committee since its inception in 1973. Altered employer-employee power relations are reflected in the legislated worker access to medical and occupational hygiene records and an explicit right of workers to leave a dangerous workplace. It is to be hoped that greater access to information will allow employees and their representatives to participate in a more meaningful way in processes to improve the health and safety of the working environment.

Later in this chapter, the burden of occupational lung disease will be evaluated on the basis of existing research. The burden of pneumoconiosis and tuberculosis in the gold mining workforce will be shown to be high. It will be argued that the prevalence and incidence of occupational lung disease shows that current practices are not safe and that this is indicative of the need to monitor disease prevalence as well as exposure levels, in order to ensure that there is adequate occupational health control.

What follows is an overview of the pathogenesis, clinical manifestations, radiology, spirometry and epidemiology of the diseases primarily associated with gold mining in South Africa. The conditions considered below are all scheduled diseases in terms of the ODMWA.

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5.4 Pneumoconiosis

Pneumoconiosis can be defined as the accumulation of dust in the lungs and the tissue reaction to its presence.⁴⁶ The name pneumoconiosis means "dust-containing lung" and is derived from the German word 'pneumonokontosis', a term coined by the pathologist Friedrich von Zenker in 1866.⁴⁷

All the pneumoconioses result from the inhalation of mineral dust. This can cause a breakdown in the defence mechanisms of the lung, penetration of lung tissue by particles and resultant deposition of fibrotic tissue. Pulmonary fibrosis is accompanied by a decrease in the lung's aerating surface area and the clinical outcome is dyspnoea (shortness of breath), cough and, ultimately, heart failure. Inhalation of dust from silica, coal, asbestos and other silicates, leads to the development of silicosis, coal worker's pneumoconiosis, asbestosis, and

silicatoses respectively.

5.4.1 Identification of pneumoconiosis

Pneumoconiosis is conveniently detected on X-ray film in accordance with the International Classification of Radiographs of Pneumoconioses, developed by the International Labour Organisation (ILO). In the USA, radiographs are officially interpreted by 'B readers' who have been certified by the National Institute of Occupational Safety and Health (NIOSH). Standard films published by the ILO are compared with films being read.

This interpretative system initially requires a reader to evaluate whether the film is normal or abnormal. If the film is abnormal, the reader must state whether the changes are consistent with pneumoconiosis. The reader then rates the abnormal opacities by size, shape and profusion. Rounded opacities are classified as *p*, *q*, or *r*. The *p* opacities are those of up to 1.5 mm, *q* opacities are those of up to 1.5-3 mm and *r* are those from 3-10 mm. Profusion is recorded as 0,1,2, or 3. Progressive Massive Fibrosis (PMF) lesions appear as confluent masses of greater than 10 mm in diameter. These are classified by the ILO as A (10-50mm), B (opacities larger and more numerous than A, but not exceeding the equivalent of one third of one lung) and C (the combined area of the opacity mass exceeds the equivalent of one third of one lung). Pleural changes are graded as to thickness, extent and the presence of calcification. Finally, there are 22 designations which allow the interpreter to indicate the presence of other abnormalities, such as distortion of the hilar structures or diseases such as cancer and emphysema.³⁸ Simple silicosis manifests as discrete, rounded nodules, located predominantly in the upper lung zones. As the disease progresses, the nodules can form conglomerate masses, resulting in gross distortion and displacement of the mediastinal and hilar structures.

The ILO reference standards are a valuable epidemiological tool because they allow for standardised epidemiological research and as such, have been important in establishing exposure-response relationships in the workplace. However, the ILO reference standards are not without problems in certain contexts. Indeed, they are difficult to use in countries such as South Africa, where there is a very high prevalence of tuberculosis, and where many silicotics have both diseases. The problem is that the reference standards deal with pneumoconiosis in the absence of tuberculosis. This issue will be returned to later in this thesis. Furthermore, Epler has shown that while there is a relatively good correlation between lung pathology findings and ILO readings of chest radiographs at the higher profusion levels, X-ray films

may not reveal early pulmonary changes resulting from dust exposure.⁴⁸ A study of the correlation between radiological and pathological diagnosis of silicosis in a cohort of white South African gold mineworkers revealed that many study subjects had silicotic nodules found at necropsy which had not been detected radiologically.⁴⁹ Subsequent research on the same cohort has shown that only 34 per cent of 719 study subjects found to have silicosis at necropsy had been diagnosed radiologically (although this cohort had been for annual radiological examinations while in mine service and for regular occupational health surveillance examinations in retirement).⁵⁰ It can be argued that imaging techniques such as high resolution chest tomography scans provide more information than traditional chest films. There is not yet a widely accepted, standardised method of classifying CT scans although several suggestions have been made.^{51,52} These imaging techniques are currently prohibitively expensive for hospitals based in rural mine labour sending communities but in the future, they may play a crucial role in distinguishing pneumoconiosis from tuberculosis.

5.4.2 Lung function tests for pneumoconiosis

In addition to radiography, lung function tests can be used to help diagnose lung diseases, including pneumoconiosis, tuberculosis and emphysema. Lung function tests can be used to obtain several measures of lung function including the vital capacity (VC) during a slow vital capacity movement, the forced vital capacity (FVC) during forced, rapid expiration and the forced expiratory volume in one second (FEV₁). The ratio of FEV₁ to FVC can be an important index of airflow. Lung function abnormalities can be either obstructive, restrictive or both. Conditions which limit airflow cause a reduction of FEV₁ relative to the FVC, such a reduction being an indicator of an obstructive disease pattern. In the case of a person with emphysema, the Total Lung Capacity (TLC) is well preserved but FEV₁ is decreased because of a loss of elasticity in the lungs, whereas the VC is better preserved. Conditions in which there is shrinkage of the lungs (i.e. a reduction in TLC) cause a reduction in the FEV₁ and the FVC in equal proportions (as is the case with both tuberculosis and silicosis). This is indicative of a restrictive disease pattern. Simple silicosis in the lower grades of profusion produces little, if any, change in FEV₁ or FVC. Major lung function changes emerge as large opacities develop.

5.4.3 Silicosis

Silicosis is the commonest form of pneumoconiosis in South Africa.⁵³ The high prevalence of

silicosis relative to the other pneumoconioses reflects both the large numbers of people employed in risk work within the South African gold mining industry and the toxicity of free crystalline silica. The incidence of silicosis is directly related to the amount of exposure to silica dust and prevention of silicosis depends entirely on preventing people from inhaling dust.⁵⁴ Unlike some other occupational diseases, such as obstructive airways disease, there is no evidence to suggest that tobacco smoking or environmental pollutants are associated with the presence of silicosis in the lung. Prevention is particularly important in relation to silicosis, since there is no effective treatment for silicotic lesions.⁵⁵

The pathogenesis of silicosis is complex and incompletely understood. Nevertheless, there has been much progress in recent years in identifying the events which occur in lung cells following the inhalation of silica. These advances in knowledge have come from research in which cell cultures were exposed to quartz, and also from animal experimentation. However, it must be recognised that both cell culture work research using animal models, rely on a process of exposure that is somewhat different to those which occur when humans are exposed in a dusty workplace.

As has been stated above, the physical features of inhaled silica determine the tissue response in that the particles must be small enough to reach the alveoli and also because the potency of the particle is greater if it has recently been fractured. It has been proposed that the inhalation of silicates causes oxidant generation. The silanol groups which are common to all silicates are thought to attract ferric ions and, together, result in the formation of a silicato-iron complex. Iron in the silicato-iron complex is thought to mediate an electron exchange when it is reduced to the ferrous state. This electron exchange results in the production of an hydroxyl radical which is a potent oxidant and this, in turn, is thought to cause lipid peroxidation of cell membranes. The generation of oxidants initiated by inhalation of the silica sets off a cascade of events which leads to alteration of macrophage function, activation of humoral and immune systems and, ultimately, the collagen deposition and pulmonary parenchymal damage known as silicosis.⁵⁶

The key diagnostic features of silicosis are a history of silica exposure in conjunction with a chest radiograph that reveals silicotic nodules and an absence of other conditions which might mimic silicosis.⁵⁷

There are different types of silicosis: classic, accelerated and acute. These can be

distinguished in terms of the degree of airborne dust concentration and length of exposure to the crystalline silica required to induce them, the speed with which they manifest and the degree to which lung tissue is replaced by fibrotic nodules.

5.4.4 Classic silicosis

Classic silicosis is usually the result of low to moderate silica dust exposure over a considerable number of years. There are two key forms of classic silicosis: simple silicosis and progressive massive fibrosis (PMF).

In simple silicosis, fibrotic scar tissue develops slowly around the inhaled silica dust in the lungs. These fibrotic nodules gradually replace lung tissue and can cause permanent lung damage. The nodules are seen in greatest profusion in the apical and posterior regions of the upper and lower lobes of the lungs. The degree of pulmonary damage is determined by the extent to which the nodules have replaced lung tissue.

Most of the medical literature indicates that patients with simple silicosis are generally thought to have little or no impairment of lung function,⁵⁸ although some studies^{59,60} have shown a high prevalence of a restrictive pattern (FVC < 80 per cent of predicted). Obstructive impairment is rare in patients with simple silicosis. Dyspnoea on physical exertion is the most common feature of silicosis but is found more often in association with large radiographic opacities rather than with simple silicosis.⁶¹ Complicated silicosis (presence of large opacities) has been associated with obstruction, mixed obstruction and restriction or pure restriction.

The major risk in relation to simple silicosis is increased susceptibility to mycobacterial infections, especially tuberculosis.^{62,63} A study of silicotic and non-silicotic copper miners in the then Northern Rhodesia in 1960, found that the incidence of tuberculosis was 30 times higher in the silicotic miners than in the non-silicotic miners.⁶⁴ Two recent surveys, one amongst Danish factory workers⁶⁵ and the other in South African gold miners,⁶⁶ have revealed that workers with chronic silicosis have a three-fold increase in the incidence of tuberculosis when compared with a group without silicosis, matched for exposure and age. The incidence of active tuberculosis increases in direct proportion to the severity of the silicosis and the risk ratio for those with an ILO small opacity profusion code of 3/3 is comparable with the increased risk for HIV-infected subjects.⁶⁷ However, even the earliest silicotic changes carry an increased risk of tuberculosis.⁶⁸ Hnizdo and Murray undertook a study of the risk of

tuberculosis relative to silicosis and silica exposure and found that even a negligible degree of silicosis (< 5 nodules at necropsy) was associated with an increased risk when compared with mineworkers without silicosis. This result seems to suggest that the effect of silica dust rather than those few silicotic nodules, is causing the increased risk. This topic will be returned to later in this chapter.

Progressive massive fibrosis (PMF) is a rapid proliferation of nodules which form a stony mass of confluent lesions. PMF develops in 20-30 per cent of subjects with simple silicosis.⁶⁹ The factors known to cause the progression of simple silicosis to PMF are higher cumulative dust exposure, younger age at onset of pneumoconiosis, and continued dust exposure in the presence of simple pneumoconiosis. In advanced PMF, the fibrous masses may distort the shape of the lungs. Several studies have shown that PMF causes significant respiratory impairment (restricted lung volume, decreased pulmonary compliance and a decrease in gas transfer).⁷⁰ PMF is characterised by dyspnoea, sputum production and cough and increases the risk of tuberculosis which causes a rapid worsening of symptoms and accelerates the decline of lung function. Evidence from a Canadian study of compensated silicotics suggests that simple silicosis does not affect life expectancy but that PMF may do so.⁷¹ One cannot extrapolate directly from the Canadian context to that of South Africa, given the paucity of South African data on life expectancy and silicosis and the very high prevalence of tuberculosis in South Africa, which will may reduce life expectancy for all silicotics.

5.4.5 Accelerated silicosis

Accelerated silicosis is the result of exposure to high concentrations of crystalline silica and it develops 5-10 years after initial exposure. Clinical autoimmune conditions such as scleroderma (persistent hardening and contraction of the body's connective tissue) are also associated with accelerated silicosis,⁷² although it occurs in mineworkers without the presence of silicosis.

5.4.6 Acute silicosis

Acute silicosis is the result of exposure to very high concentrations of silica dust and symptoms can develop within a period of 1-5 years after initial exposure. The lung is consolidated, grey-white in colour and does not contain the nodules and lesions that are found in classic silicosis. The alveolar spaces are filled with acidophilic fluid containing macrophages. Acute silicosis results in rapid death by respiratory failure. The key symptoms

of accelerated and acute silicosis are dyspnoea, restriction of lung volume and impairment of the diffusing capacity of the lungs. Accelerated and acute silicosis were common on the Witwatersrand in the early 20th Century. However, the prevalence of both conditions has dropped markedly due to the improved working conditions and lower dust levels in many industrial countries.⁷³ As has previously been noted, the incidence of tuberculosis is higher in those with silicosis as compared with similarly aged, silica-exposed workers without silicosis. However, this increased incidence of tuberculosis is especially marked in those with acute and accelerated silicosis.⁷⁴

As was discussed in Chapter one, there is no effective way of reversing the course of silicosis and prevention is through the elimination of hazardous conditions. The clinical approach to people with silicosis is aimed at the amelioration of symptoms and the reduction of risk of related disorders.

5.4.7 Transkei silicosis

Transkei silicosis is a domestically-acquired pneumoconiosis. Palmer noted that this condition is found predominantly in women and is thought to be associated with inhalation of silica generated from grinding stones during the grinding of corn and the use of biomass fuels (cow dung) in poorly ventilated homes.⁷⁵ On the basis of the observation that grinding of corn is not an activity undertaken by men in the Eastern Cape Province, that all persons in rural Transkei (whether migrant mineworkers or not) are subject to the same exposure to biomass fuels and also after perusal of the case studies that Palmer presents, it seems unlikely that this condition contributes to silicosis prevalence in former mineworkers. Grobbelaar coined the name "Hut Lung" to describe this condition.⁷⁶ In a survey based on the radiological and pathophysiological changes in the lungs of 25 cases, Grobbelaar found that most women were asymptomatic but that radiological findings ranged from a miliary tuberculosis to extensive fibrosis.

Given the cultural aversion to corn grinding in men from the Eastern Cape, Transkei silicosis can be excluded as a possible cause of disease in the study group used for this thesis.

5.4.8 Tuberculosis

Tuberculosis is an infectious disease contracted primarily through inhalation of airborne droplets containing tubercle bacilli. The micro-organisms are exhaled by people with active