African Affairs, 108/431, 221–240 doi: 10.1093/afraf/adn085

Advance Access Publication 22 January 2009

COUNTING THE COST: GOLD MINING AND OCCUPATIONAL DISEASE IN CONTEMPORARY SOUTH AFRICA

JOCK MCCULLOCH

ABSTRACT

Gold mining has been central to the success of South Africa's economy. That labour intensive industry has relied heavily on migrant workers for its profitability. In the past decade, scientists in Johannesburg and Cape Town have identified a pandemic of the serious occupational disease silicosis among gold miners. Litigation currently before South African courts raises the possibility of a class action by hundred and thousands of miners against the major corporations such as Anglo American. If successful that litigation may well change work regimes in the mining industry. This article explores the role of migrant labour, state regulatory authorities and science in hiding a pandemic which probably spans the 20th century.

AFTER MORE THAN A CENTURY OF COMMERCIAL SUCCESS, gold mining in South Africa is a dying industry. Over the past decade more than 250,000 jobs have been lost. The corporate restructuring of the 1990s has left only four major players: AngloGold, AvGold, Goldfields, and RandGold. JCI, one of the original Rand companies, is bankrupt. In 1995 the Leon Commission into occupational health published its report, which found that dust levels on the mines have been unchanged for half a century. The implication is that many thousands of miners have died from uncompensated silicosis, a serious and incurable lung disease. Others are still suffering. Consider the case of Mankayi Mbini versus Anglo American, which was was filed in Pretoria in August 2004:

Mbini worked at the President Steyn mine in Welkom from 1958 to September 1997. Prior to his retirement due to silicosis he was earning R2,500 a month as a stope team leader.[1] Mbini has silicosis and tuberculosis and he is claiming damages of R885,994.60. That consists of the loss of past and future earnings of R397,494. Mbini's claim of R150,000 for pain,

Jock McCulloch PhD (Jock.mcculloch@rmit.edu.au) is Professor of History at RMIT University, Melbourne. He acknowledges funding from the Australian Research Council to investigate and report on silicosis issues.

1. See Mankayi Mbini and Anglo American Corporation of South Africa Ltd, High Court of South Africa (Witwatersrand Local Division), Case No. 04/18272 2, August 2004.

suffering and loss of amenities of life is very low compared with claims made routinely before United Kingdom, US and Australian courts. His condition is almost certain to worsen and he will need annual X-rays, lung function tests, and medication for chronic pain. Bacterial infections will require periodic hospitalization. For that reason he is claiming R338,500 for future hospital and medical expenses. Silicosis is a vile disease for which there is no effective treatment. It is bad enough for those who have access to modern medical care; in South Africa the burden of disease among gold miners and their families is much heavier.

Mbini's case is being led by the British lawyer Richard Meeran. It falls into three parts: the duty of the defendant to the plaintiff, the breach of that duty, and the level of compensation to be paid. Mbini claims that Anglo American knew or should have known that silica dust causes silicosis and tuberculosis. It knew that the risk could be reduced by preventing the release of dust during mining or by providing miners with respirators. It should have provided clean overalls, and adequate washing and laundry facilities. Finally, Anglo American should have removed all dust from the hostels in which Mbini lived, and funded testing for lung impairment.

In terms of a breach of statutory duty the most relevant legislation is the Mines and Works Act No. 27 of 1956, which stipulates that the air entering a mine shall be free from smoke or dust or other impurities; that the workplace shall be properly ventilated to maintain a safe environment; that every effort must be made to prevent the accumulation of dust in any part of a mine; and that the wearing of protective apparatus is required if the general concentration of dust exceeds the standards specified by the Government Mining Engineer. According to the civil rights lawyer Richard Spoor, the regulations were intended to place on employers a duty to provide a safe working environment, and responsibility for a failure to do so.[2] Such an approach is consistent with English and European law. Under the breach of care principle liability is generally not dependent on negligence or practicality and it is unaffected by delegation. According to Richard Meeran, the case has two aims: the establishment by the gold industry of a compensation fund for silicosis victims, and the creation of an effective system of medical monitoring for former miners.[3]

Anglo American has almost limitless resources with which to defend itself. Much of the evidence about dust levels and disease rates lies in the corporation's hands, which further disadvantages plaintiffs like Mbini. In addition, there are few experts in Johannesburg willing to testify on behalf of injured workers. But the major barriers to litigation are the Occupational Diseases in Mines and Works Act (ODMW) of 1993 and the Compensation

2. Richard Spoor, 'Notes on the case of Mankayi Mbini versus Anglo American' (unpublished paper, 2006).
3. 'Anglo being sued for R20m', *The Sunday Independent*, 30 September 2007.

for Occupational Injuries and Diseases Act (COIDA) Act. It has long been assumed by the Mine Workers Union (MWU) that miners' right to compensation from industry-based funds precluded its members from seeking redress in court for occupational injury.

The Mbini case is about the fate of one man but it also has a wide frame of reference. There is much undiagnosed and uncompensated silicosis among gold miners. As the Chief Inspector of Mines, Mavis Hermanus, has commented: 'The unchanged rate of silicosis is a current problem in the mining industry. Data from 1975 to 2001 indicate that we are not making progress against the disease.'[4] According to Eric Geilitshana, the National Union of Mineworkers (NUM) Secretary for Health and Safety, 40 percent of South African gold miners have silicosis, an estimate consistent with the recent epidemiology.[5] Mbini may well be the first step in a massive class action involving hundreds of thousands of miners from Lesotho, Zimbabwe, Malawi, Swaziland, Mozambique, and Botswana, all of which supplied labour to Anglo American. If those claims eventuate, the corporation's liability could be as high as R50 billion.[6] Even if unsuccessful, Mbini may well bring a change to work practices in South Africa's mines.

This article examines the history of silicosis and in particular the question as to how a pandemic of disease could have remained invisible for over half a century. The narrative focuses upon the relationship between capital, labour, and medical researchers, and in particular the collusion between state agencies and employers. It reviews the Mbini case in terms of the history of the gold mines, the medical knowledge of disease and the operation of workers' compensation schemes. The current context for Mbini is found in the joint International Labour Organization/World Health Organization International Programme on the Global Elimination of Silicosis. That initiative, to which South Africa is a signatory, is an indication of the importance of occupational dust diseases in both the developed and developing worlds. Historically, South African scientists have made a major contribution to the medical literature on silicosis. The current litigation may see its courts make an equally important contribution.

The historical setting

The Rand mines are the largest and deepest in the world and historically they have been among the most dangerous. At the beginning and close of the twentieth century those mines faced a crisis over silicosis. The first crisis ran from 1896 to 1912; the second began in 1996. Both have threatened

4. See Mavis Hermanus, at <http://www.nosa-int.com/news/news0006asp> (12 August 2002).
5. See Martin Creamers, *Mining Weekly,* 7 May 2004.
6. Spoor, 'Notes on the case of Makaya Mbini'.

the industry's survival. The first crisis was focused upon disease among white miners, who represented less than 10 percent of the workforce: the second is about black migrant workers drawn from within and beyond South Africa's borders.[7] In 1912 South Africa's racialized labour system enabled the mining houses to reach an accommodation with white labour, thereby resolving the problem of silicosis in its favour. The second crisis is being played out in the High Court.

South Africa's mining industry has been dominated by a handful of corporations. That in turn has shaped the relationship between industry, labour, science, and the state. The mines have relied upon migrant labour. While migrancy has been common to industrial states, in South Africa it has been unique in the depth of its political foundations and in its negative impact upon labour-sending communities.[8] Migrant workers were defined by the mining houses and the state as temporary employees who worked for short periods, then returned home to recuperate. In practice, as Harold Wolpe noted long ago, under that system rural households became field hospitals for retrenched, medically repatriated, and ageing men.

Historically the four major causes of morbidity and mortality have been accidents, viral pneumonia, silicosis and tuberculosis. Accidents usually have a finite cause and an obvious remedy, and the Chamber of Mines has never been shy in talking publicly about such traumas. The same was true of pneumonia. After 1913 improved hygiene and a change in recruitment patterns saw a rapid fall in the calamitous death rate. In contrast, the threat of silicosis and its *sequella* tuberculosis among black migrant workers has never been addressed. The only way to prevent silicosis, and with it a propensity for tuberculosis, is to eradicate dust from the workplace. There is evidence from as early as 1926 that some experts in Johannesburg believed it was impossible to engineer dust out of the mines. That is not surprising as most underground work, including drilling, blasting, loading, and transporting ore, generates dust. Dust is also spread by the use of water sprays which are designed for its suppression. High velocity sprays quickly evaporate, thereby releasing fine particles into the air. It has been estimated that recycled water produces between 3 and 8 times more machine dust than clean water.[9]

7. On the first crisis see Elaine Katz, *The White Death: Silicosis on the Witwatersrand gold mines 1886–1910* (Witwatersrand University Press, Johannesburg, 1994) and Isidore Donsky, *A History of Silicosis on the Witwatersrand Gold Mines, 1910–1946* (Rand Afrikaans University, PhD thesis, 1993).

8. See Francis Wilson, *Labour in the South African Gold Mines 1911–1969* (Cambridge University Press, Cambridge, 1972).

9. Rina King, 'Silicosis in South African gold mines: a study of risk of disease for black mineworkers' (unpublished research paper, Department of Sociology, University of the Witwatersrand, 1985), p. 19.

During the first crisis, silicosis was, in the words of Jack Simons, a 'white disease'.[10] There were commissions and inquiries into its cause among white miners, dust suppression technologies were improved, and medical surveillance was introduced. Ordinances to promote safety and dust control were passed in 1903, 1905, 1907, and 1908. That flurry of activity saw a reduction in the dust levels, and transformed silicosis from an acute to a chronic disease. The Chamber of Mines and the state built sanatoria to treat white miners, and created a compensation scheme for their families. From 1910, the question of who should pay and at what level became a source of chronic conflict between the state, industry, and white labour.

In 1912 South Africa became the first state to recognize silicosis as a compensatable disease. Four years later tuberculosis was recognized as an occupational disease of gold miners. Benefits were based on wage levels, and so white miners received more generous compensation than did blacks.[11] In addition, black miners were migrant workers and few were aware of their rights. Consequently, in the three years to July 1916, the total compensation for whites was 30 times greater than that paid to black mine workers.[12] Over the period from 1912 to 1946, fifteen further silicosis acts were passed, most of which were designed to improve the benefits paid to white labour. The white Mine Workers' Union, founded in 1913, was so effective in promoting the interests of its members that each commission increased the compensation levels.[13] No effort was made to reduce the barriers facing black claimants. And there were no parallel commissions into viral pneumonia or meningitis, which killed more black miners than did silicosis.

Compensation was paid from a levy based on the number of men employed at each registered mine. Levies were set according to an estimate of dust levels. Sampling was done by the Chamber of Mines or the Government Mining Engineer every three years, and an index was calculated using a complex formula. From that index a levy was imposed per ton of ore. By 1925 more than 100,000 dust samples were being taken annually and it is likely that more dust samples were collected on the Rand than in the rest of the world.[14] Those samples were expensive to collect and yet the

10. See H. J. Simons, 'Occupational health in the South African mining industry, 1870–1956', in 'Migratory labour, migratory microbes' (unpublished manuscript, *circa* 1960), p. 27.

11. See Matthew John Smith, '*Working in the Grave': The development of a health and safety system on the Witwatersrand gold mines, 1900–1939* (Rhodes University, MA thesis, 1993), pp. 182–4, and Donsky, *A History of Silicosis,* pp. 179–201.

12. Donsky, *A History of Silicosis,* p. 196.

13. See Robert H. Davies, *Capital, State and White Labour in South Africa 1900–1960* (Humanities Press, Atlantic Highlands, NJ, 1979).

14. Dr Orenstein, in 'Silicosis: records of the International Conference held at Johannesburg 13–27th August 1930' (International Labour Organization, London, 1930), p. 32.

Chamber did not publish any studies from the data, nor did it attempt to match the data against the official disease rates. In theory, such an exercise could have identified an exposure level below which silicosis should not occur.

The Rand system of dust sampling has a corollary in the so-called Threshold Limit Values (TLVs) which were used widely in the US to measure the risk of silicosis in foundries and mines. Such thresholds were based on the assumption that if exposure to toxins could be kept below a certain level, then workplaces would be safe. The idea of TLVs appealed to industry. Specific hazards could be negotiated between experts appointed by employers and government, thereby shifting debate about risk away from public scrutiny. Trade unions were perceived to have no scientific expertise and therefore no role to play in such discussions. From the mid-1930s TLVs were the accepted method for setting workplace standards in the US.[15] Thresholds were engineering rather than medical artefacts and 'good practice' was most often shorthand for what was commercially acceptable. In Western Europe, in contrast, the emphasis was upon general engineering controls, plant design, and work practices, rather than compliance with an abstract standard. The adoption of what were in effect TLVs in South Africa discouraged other types of data collection and in particular the use of follow-up studies to identify disease rates.

Arriving at a meaningful TLV is difficult for a number of reasons, not least because of the instrumentation. The Konimeter, which was invented by the Government Mining Engineer Robert Kotze in 1914, was used to measure dust in South Africa from 1920 until 1992. The instrument takes a snap reading lasting less than 1/4 second, which represents 1/100,000 of an 8-hour shift. Dust levels change from moment to moment and no single moment need be representative of a particular miner working a particular shift. Mines are huge workplaces and individual readings are unlikely to be typical of a mine as a whole. The Konimeter is inefficient when sampling high levels of fine dust but reliable with low levels of coarse dust. Unfortunately, fine dust is the most dangerous. Konimeter data were useful in setting levies but had little meaning as a measure of risk. The only true measure of the risk of silicosis was the actual disease rate. In the case of black miners little effort was made to research that question until the 1990s.

The compensation scheme for gold miners assuaged white labour, created the image of hygienic work environments, saved the industry the cost of dust suppression, and foreclosed on litigation. Even so, to compensate only 10 percent of the workforce proved expensive. The Legal Adviser

15. On the development of the US standards, see G. Markowitz, and D. Rosner, 'The limits of thresholds: silica and the politics of science', *American Journal of Public Health* 85 (1995), pp. 253–62.

to the Chamber of Mines, G. E. Barry, remarked at the 1930 Silicosis Conference in Johannesburg that the compensation scheme imposed such a heavy cost on employers that some mines had been forced to close.[16] H. W. Sampson, Minister of Posts and Telegraphs, told the delegates that compensation cost the gold mines almost one million pounds per year or 15 million pounds in the period from 1911 to 1929.[17] If adequate compensation had been paid to black miners the cost would have been many times greater.

The research community

South Africa's first silicosis crisis helped to make Johannesburg a world centre for research into dust disease. The leading scientists included I. G. Irving, A. Sutherland Strachan, F. W. Simson, W. Watkins-Pitchford, A. Mavrogordato, Spencer Lister, A. J. Orenstein, and Andrew Watt of the Rand Mutual Assurance Company, Ltd. The research community was close-knit and employed either by the state or the mining houses. Between 1930 and 1969 a series of international conferences in Johannesburg helped to define the scientific and regulatory agendas on pneumoconiosis, or occupational dust diseases of the lung. The 1930 Silicosis Conference was a pivotal moment in the global response to the disease. It was sponsored by the International Labour Organization and brought together scientists from the US, the UK, South Africa and Australia. The siting of the conference was testament to the importance of Johannesburg's researchers. As Professor Hall of Sheffield University commented at the time: 'The statistics of South Africa were so unique and of such importance to all countries which had to initiate legislation and compensation that every effort should be made to make them as clear as possible.'[18]

The South African Institute of Medical Research (SAIMR) was founded in 1912 jointly by the Witwatersrand Native Labour Association (WNLA), the recruiting arm of the mining industry, and by government. The Institute's first director was Wilfred Watkins-Pitchford and WNLA provided the bulk of the funding. Both WNLA and the state were concerned about the high mortality rates from pneumonia and silicosis, and the Institute's brief was to resolve those problems. Most of the key silicosis researchers were associated with the Institute's two divisions. The Research Division's primary focus was on silicosis and pneumonia, while the Routine Division carried out the diagnosis and treatment of miners, and conducted medico-legal investigations. The divisions' funding came from services to government

16. G. E. Barry, in 'Silicosis: records of the International Conference', p. 84.
17. H. W. Sampson, in *ibid.*, p. 14.
18. Professor Hall, in *ibid.*, p. 78.

departments and the mines. In 1919 the SAIMR had a staff of seven; it had grown to 75 by 1926. In that year the Institute carried out 66,000 diagnostic investigations.[19] The SAIMR's dual structure became the model after the Second World War for the Pneumoconiosis Research Unit (PRU) and the National Institute for Occupational Health (NIOH).

The intimate relationship between the Chamber of Mines, the Department of Mines and the research community is epitomized by the Institute. Those three groups shared the same research focus and a common source of funding through mine revenue. They also shared the same personnel, as key researchers moved between one sector and the other. The same men served on state commissions and departmental committees, and represented the Chamber at public inquiries. That made it difficult for an individual to confront the mining houses over the dust hazard.

There is evidence from the 1920s that at least one South African expert was concerned about silicosis among black miners. Anthony Mavrogordato, who had worked with J. S. Haldane in the UK, was appointed Fellow in Industrial Hygiene to the SAIMR in 1919. He worked closely with the Department of Mines and the Chamber on dust counting and ventilation. From his appointment until his retirement in 1939 Mavrogordato published only 12 papers.[20] That modest output is curious given the significance of his work.

In 1926 Mavrogordato wrote a 120-page review of the medical literature on silicosis reflecting upon the issues of risk and disease.[21] He identified three key problems on the Rand: the difficulties of diagnosis, the synergy between silicosis and tuberculosis, and the intractability of the dust burden. He noted that more cases of silicosis were picked up at autopsy than during routine X-rays. For example, a man killed in an accident after only three years underground would at autopsy show definite signs of fibrosis even though he was at the time of death in apparent good health.[22] That finding suggested that the disease rates might be higher than was officially recognized, an observation which subsequent research confirmed. Mavrogordato also believed that the minor changes to lung tissue found at autopsy, but invisible in X-rays, greatly increased the risk of tuberculosis.[23] That in turn

19. Marais Malan, *The Quest of Health: The South African Institute of Medical Research, 1912–1973* (Lowry Publishers, Johannesburg, 1988), p. 35.
20. *Ibid.*, pp. 30–2.
21. A. Mavrogordato, 'Contributions to the study of miners' phthisis' (typescript, Adler Medical Library, University of the Witwatersrand, Johannesburg), p. 56. An abridged version of the paper was published under the same title as a monograph by the South African Institute of Medical Research. See South African Institute for Medical Research, occasional publications, Vol. 3, No. 19 (December 1926).
22. Mavrogordato, 'Contributions', pp. 50–1.
23. *Ibid.*, p. 52.

suggested that tuberculosis was being exported to the labour-sending areas, a problem the industry has always denied.[24]

The most important aspect of Mavrogordato's paper deals with dust. He distinguishes between income dust, which is the dust generated by each day's work, and capital dust, which is the dust always in circulation underground.[25] Income dust had been greatly reduced by the use of water sprays but such sprays had no effect on capital dust, which Mavrogordato believed was sufficient in itself to produce silicosis. Capital dust also made it unsafe to use mine air for ventilation. Mavrogordato acknowledged that to improve air quality would greatly increase production costs.[26]

Mavrogordato's paper suggests it was virtually impossible to reduce dust to a level at which silicosis and tuberculosis would not occur. The use of water to lay dust increased the chances of tuberculosis. Even sub-clinical silicosis increased a miner's susceptibility to infection. His paper was not cited in the literature and Mavrogordato did not raise any of his concerns about capital dust and lung disease at the 1930 Silicosis Conference.

The accommodation between industry, white labour, and the state was fragile and it occasionally fractured. During the Second World War there was such pressure on production that work conditions on the mines deteriorated. The disease rates among white and black labour probably rose. After the war the opening of the Free State fields saw the size of the workforce increase along with output. White miners, led by the Mine Workers' Union (MWU) lobbied for better compensation. So too did a senior medical officer at the Silicosis Bureau, in Johannesburg, Dr G. W. H. Schepers. Those demands were played out through a series of commissions and resolved by the passing of four pieces of legislation. The underlying cause was the perennial problem of dust and disease.

All gold miners were required to undergo pre-employment and periodic medicals. White miners were assessed by interns at the Silicosis Bureau. Black miners were examined either at the WNLA depot or at individual mines. The Bureau interns were obliged to conduct an X-ray examination of each miner and complete a Form A which entailed a full medical and work history. Because of the volume of work such details were rarely recorded. In 1951 the Bureau carried out 54,772 medicals or 5,472 examinations per intern.[27] Allowing for an hour-long daily meeting to adjudicate on claims

24. See, for example, George A. Turner, 'Report on the prevalence of pulmonary tuberculosis and allied diseases in the kraals of the natives of Portuguese East African territory, south of latitude 22°' (Hayne and Gibson, Johannesburg, 1906) and 'Tuberculosis in South African natives with special reference to the disease amongst the mine labourers of the Witwatersrand' (South African Institute for Medical Research, Johannesburg, 1932).
25. Mavrogordato, 'Contributions', p. 99.
26. Mavrogordato 'Contributions', p. 109.
27. 'Report of the Commission of Enquiry into the functioning of the Silicosis Medical Bureau and the Silicosis Medical Board of Appeal' (Government Printer, Pretoria, 1952), p. 7.

and other duties, that meant each intern examined on average 5 patients an hour. At most they had 12 minutes for each examination. The process was even less rigorous with blacks.

Between 1944 and 1952 Dr G. W. H. Schepers was an intern with the Bureau and from 1952 till 1954 he was Director of the Pulmonary Disability Committee. The Bureau adjudications over compensation were based entirely on X-rays, although since the 1930s these had been recognized as inadequate for diagnosis, let alone for assessing disability.[28] Consequently it was common that a miner would be too sick to work but not qualify for assistance. Much to the anger of the MWU, many men were left destitute.[29] The examination of black miners was even less reliable and was influenced by racial prejudice and a cavalier attitude toward sick 'natives'.[30] Schepers lobbied the Bureau's director for change. When that failed, in frustration he testified before the Beyers Commission in January 1952.[31] Beyers found that because of crushing workloads and understaffing the MWU's complaints about the Bureau were fully justified.[32] Both the Beyers and the subsequent Oosthuizen commissions were highly critical of the Bureau's medical practices, which saw many legitimate claims for compensation refused.[33]

The new science

The Leon Commission of 1995 was held at a difficult time for the industry, which was not keen to see the issue of occupational disease debated at a public forum. The Chamber was reluctant to discuss silicosis and its initial submission on lung disease was two and a half pages in length.[34] The Commission found that silica dust levels on the gold mines had been unchanged for more than fifty years and suggested the same may be true of the incidence of silicosis.[35] This was a significant finding. In 1997 a joint initiative by the ILO, the WHO and the South African government to eliminate silicosis was established. It features a Tripartite Committee comprising workers (the NUM and the Mines Workers' Union), employers (Chamber

28. Memo from Dr Gerrit Schepers to Jock McCulloch regarding the Beyers Commission, 16 and 17 August, 2008, Great Falls, Virginia, USA.
29. *Ibid.*
30. *Ibid.*
31. See Dr G. W. H. Schepers, transcripts of evidence before the Beyers Commission (Johannesburg, 29 January 1952), pp. 492–537.
32. 'Report of the Commission of Enquiry into the fuctioning of the Silicosis Medical Bureau', pp. 7–8.
33. See 'Report of the Departmental Committee of Enquiry into the relationship between silicosis and pulmonary disability and the relationship between pneumoconiosis and tuberculosis' (Government Printer, Pretoria, 1955).
34. See 'Report of the Commission of Inquiry into Safety and Health in the Mining Industry'. (Department of Minerals and Energy Affairs, Pretoria, 1995), pp. 48–50.
35. *Ibid.*, pp. 514–55.

of Mines) and the government (Departments of Health and Minerals and Energy). The Committee's brief is to oversee the eradication of silicosis by the year 2013. That is going to be a difficult task.

The Leon Commission coincided with a body of important science. Research by Anna Trapido, David Reese, Tony Davies, and Jill Murray at the National Institute of Occupational Health (NIOH) in Johannesburg and at the University of Cape Town by Neil White, T. W. Steen, Rodney Erlich and Jonny Myers has identified a pandemic of hitherto undiagnosed and uncompensated silicosis involving hundreds of thousands of miners. In 1997, T. W. Steen *et al.* published the first follow-up study of silicosis among migrant labour. Using a cohort of Botswana miners who had worked in South Africa, they found a rate of silicosis of between 27 and 31 percent; of that number, almost 7 percent had life-threatening fibrosis.[36] Anna Trapido's study on Libode, in the Eastern Cape, confirmed these data. Trapido estimates the prevalence of pneumoconiosis at between 22 and 36 percent.[37] Yet only 2 percent of miners at Libode with silicosis have received compensation. An industry-funded study by Churchyard *et al.* in the Free State found the prevalence of previously undiagnosed silicosis at between 18 and 23 percent.[38] Jill Murray estimates that up to 60 percent of miners will eventually develop silicosis.[39] The synergy between silicosis and a number of infectious diseases makes the problem all the greater. As the Chief Inspector of Mines, Mavis Hermanus, commented in 1998: 'Dust exposure drives a four fold increase in tuberculosis (TB), while TB accelerates HIV infection and Aids symptoms, which in turn accelerate the symptoms of TB. We have to break the vicious cycle that sustains a huge public health impact.'[40]

The under-reporting of silicosis raises the spectre of a backlog of compensation claims. Steen *et al.* found that the Medical Bureau for Occupational Diseases (MBOD) underestimates the rate of silicosis by a factor of between four and ten. That is in line with Trapido, who suggests there are 196,000

36. T. W. Steen, K. M. Gyi, N. W. White, *et al.*, 'Prevalence of occupational lung diseases among Botswana men formerly employed in the South African mining industry', *Occupational and Environmental Medicine,* 54 (1997), pp. 19–26.

37. Anna Trapido, *An Analysis of the Burden of Occupational Lung Disease in a Random Sample of Former Gold Mineworkers in the Libode District of the Eastern Cape* (University of the Witwatersrand, PhD thesis, 2000), p. v.

38. See G. Churchyard *et al.*, 'Silicosis prevalence and exposure response relationship in older black mineworkers on a South African gold mine' (Safety in Mines Research Advisory Committee, SimHealth, 15 May 2003).

39. Jill Murray, 'Development of radiological and autopsy silicosis in a cohort of gold miners followed up into retirement' (paper presented at the Research Forum, National Institute for Occupational Health, Johannesburg, 26 May 2005).

40. See Mavis Hermanus at <http://www.nosa-int.com/news/news0006asp>. On the synergy between HIV/Aids, tuberculosis, and silicosis, see Shula Marks, 'The silent scourge? Silicosis, respiratory disease and gold-mining in South Africa', *Journal of Ethnic and Migration Studies* **32**, 4 (2006), pp. 571–4.

former miners in South Africa and a further 84,000 in neighbouring states with compensatable silicosis.[41] Extrapolating from Churchyard, 80,000 men currently employed on the mines are eligible for compensation. Given the rate at which the workforce has replaced itself over the past twenty years, there should be a further 200,000 compensatable cases in the neighbouring states of Southern Africa. Neil White costs the shortfall in compensation at around R2.8 billion.[42] Under common law, to those figures must be added the costs of pain and suffering, the areas in which working-class plaintiffs usually have the strongest claims for compensation. For that to happen a number of legal barriers must first be negotiated.

The compensation schemes

The first legislation dealing with occupational diseases in the mining industry was passed in 1912. The Miners' Phthisis Act, with subsequent amendments, eventually became the Occupational Diseases in Mines and Works Act (ODMW) in 1973. In 1993 the Act was amended to remove the racially discriminatory clauses that had been one of its central features. The current ODMW Act deals solely with compensation of occupational lung diseases in miners. Lung diseases in non-miners and other occupational diseases and injuries sustained by workers in other industries are covered by the Compensation of Occupational Diseases (COIDA) Act of 1993. Each act creates funds to which employers contribute.

The acts are equivalent in terms of their definitions of dust-related disease and how a diagnosis is to be made. The eight other criteria used under ODMWA discriminate against miners and their dependants. For example, the criteria used for evaluating respiratory impairment and disability are distinct. Under ODWMA, disease may be present but because it is not judged disabling it will not warrant compensation. By contrast, within COIDA, any radiological abnormality will be compensatable irrespective of disability. Consequently, miners and non-miners with identical incomes and the same levels of occupational disease will receive differing amounts of compensation. As Neil White and Richard Spoor have pointed out, the differences between the two sets of legislation put miners at a major disadvantage to other workers.[43]

COIDA provides a pension whereas under ODMWA there are only lump sums. If a worker dies in a factory accident his dependants may receive a

41. Trapido, *An Analysis of the Burden of Occupational Lung Disease*, pp. 196–201.
42. Neil White, 'Is the ODMW Act fair? A comparison of the Occupational Diseases in Mines and Works Amendment Act, 1993 and the Compensation of Occupational Injuries and Diseases Act, 1993 with respect to compensation of pneumoconiosis' (unpublished paper, July 2004).
43. White, 'Is the ODMW Act fair?' and Spoor, 'Notes on the case of Mankayi Mbini'.

pension under the COIDA act, but if he dies from lung disease ODMWA precludes his wife from such an entitlement. Lump sum payments for physical impairment cannot be expected to support workers or their families for any length of time. For that reason they are discouraged by the ILO. Not surprisingly, South African miners and their dependants often seek social security support, thereby externalizing the costs of mining onto taxpayers.[44] The low levels of compensation under ODMWA, combined with the under-diagnosis of silicosis, mean that only a fraction of the real cost of occupational diseases on the mines has been borne by employers. That in turn has served as a financial disincentive for employers to address the problem of dust.

In its defence, Anglo American is arguing that ODMWA prevents any civil claims against the company. Section 100 of ODMWA states that in respect of the same disease no person is entitled to claim benefits from more than one source. Mine workers, for example, may not claim benefits from any scheme other than those set out under ODMWA. The meaning of that clause is, however, open to interpretation. Section 100 appears intended only to disallow additional claims by mine workers for COIDA benefits. If, as seems likely, the Act was designed to prevent the payment of double benefits, then section 100 of ODMWA is not a bar to miners with silicosis seeking damages in a civil action. That is the first issue the High Court is expected to adjudicate upon.

Invisibility

The Mbini case raises the question as to how such a disease burden could have gone unrecognized for so long. There are parallels with the scandals over silicosis and black lung in the US, and more recently with miners' asthma and bronchitis in the UK. In both those cases recognition of occupational injury and then compensation took decades to achieve.[45] In South Africa the labour markets, the economic importance of gold mining and the political environment have made recognition even more difficult.

The system of medicals at the Bureau was never adequate to identify first stage silicosis among white miners. The Departments of Health and Mines in Pretoria enjoyed a monopoly over the data on disease, and took no interest in black workers once they had left the industry. The lack of biomedical care in rural South Africa and in the labour-sending states of Lesotho,

44. White, 'Is the ODMW Act fair?', p. 18.

45. See David Rosner and Gerald Markowitz, *Deadly Dust: Silicosis and the politics of occupational disease in twentieth-century America* (Princeton University Press, Princeton, NJ, 1991); Alan Derickson, *Black Lung: Anatomy of a public health disaster* (Cornell University Press, Ithaca, NY, 1998); Arthur McIvor and Ronald Johnston, *Miners' Lung: A history of dust diseases in British coal mining* (Ashgate, London, 2007).

Swaziland, and Mozambique further obscured the incidence of silicosis. Under-funded and under-resourced health systems were at their worst in dealing with a chronic disease for which there is no easy diagnosis and no treatment. Under minority rule the state was reluctant to give researchers access to labour-sending communities. When Marianne Felix from the NIOH began pioneering work among asbestos miners at Mafefe in the late 1980s, she encountered resistance from the Departments of Health and Mines.[46] The same happened five years later when Anna Trapido began work on silicosis in the Eastern Cape.

Under minority rule the recorded rates of silicosis among black miners were always far lower than for whites. In explaining that apparent difference, South African scientists have for decades referred to the short contracts worked by migrant labour. 'Observation had shown', Mavrogordato told the 1930 Silicosis Conference, 'that the smaller incidence of silicosis among natives, as compared with Europeans, was due to the intermittent employment; natives who were employed continuously developed silicosis more rapidly than Europeans.'[47] In fact the reason for the variation lay elsewhere. The official silicosis rates were based on successful claims made before the Miners' Phthisis Medical Bureau.[48] Blacks rarely lodged claims and so they were under-represented. At the time of the Weldon Commission into silicosis in 1904, a handful of researchers including J. S. Haldane, I. G. Irving, and D. McCaulay cautioned that Africans were contracting silicosis.[49] In 1906 health officials in the British South African Territories claimed that the industry was sending home mine workers with phthisis.[50] But as the dust levels on the mines were reduced and the first crisis was resolved, those protests petered out.

According to Dr Gerrit Schepers, the high rate of silicosis among African miners was common knowledge at the Bureau in the 1940s but that knowledge was tempered by a culture of 'keeping miners working, sick or not'.[51] The Chamber of Mines was keen to minimize the number of compensation awards. Through the Minister of Mines in Pretoria it 'ran the Bureau's head doctors' and through them the compensation system.[52]

Silicosis is not easy to diagnose. The absence of dramatic symptoms and the slow onset means that in its simple form the disease can go undetected for years. Furthermore, the degree of impairment will not

46. See Maria Anne Felix, *Environmental Asbestos and Respiratory Disease in South Africa* (University of the Witwatersrand, PhD thesis, 1997).
47. Mavrogordato, in 'Silicosis: records of the International Conference', p. 45.
48. Miners phthisis is the now antiquated term for the combined diseases of silicosis and pulmonary tuberculosis.
49. Katz, *The White Death*, p. 192.
50. See *Transvaal Medical Journal* (1 September 1905), pp. 25–6.
51. Memo from Dr Gerrit Schepers to Jock McCulloch, 23 August 2008.
52. *Ibid.*

necessarily coincide with a specific clinical profile.[53] At the beginning of the twentieth century South African doctors relied upon physical examinations which were laborious and often inconclusive. From 1916 Johannesburg physicians laid the foundation of modern practice when the Miners' Phthisis Medical Bureau introduced routine X-rays. That technology can detect tuberculosis and silicosis earlier than can a physical examination. In theory at least South African physicians were well equipped to diagnose silicosis. In practice, mini X-ray plates used for black miners since the 1960s were even less reliable than the full-sized plates used for whites. In 1963 Sluis-Cremer from the NIOH found that only 24 percent of slight/moderate cases of pneumoconiosis are picked up with mini-plates, in contrast to 48 percent with full-scaled plates.[54] Thirty years later the Leon Commission into occupational health found that the use of small X-ray plates was one of the major reasons for the under-diagnosis of silicosis.[55]

Even regular-sized chest X-rays may not reveal an abnormality, making lung function tests necessary. Computerized tomography is more accurate than X-rays but the technology is expensive and not widely available. Although there is no effective treatment for silicosis, symptoms can be relieved by cough-suppressants, while antibiotics may be necessary to combat respiratory infections. Since the disease is progressive, removal from a dusty workplace will not necessarily halt its advance. Under South African legislation a mine worker who has silicosis may be declared unfit for work and lose his job. That policy is a disincentive for a miner to seek medical care or to reveal his health status.

Having claimed for decades that there was no silicosis among migrant workers, by the 1980s the Chamber shifted its stance, albeit slightly. It now insisted that even if there was disease, it caused little disability. In 1982 Dr Schroder, of the Chamber's Air Pollution Division, commented:

> With the advent of specific anti-tuberculotic drugs... the likelihood of reactivation of tuberculosis and hence the lethal effects of the co-existing silico-tuberculosis, has become rare. This together with the dust control in South African mines had led to the statement that no one has died of silicosis during the past ten years.... [I]t can now be stated that in South African Gold Mines, no one today dies from silicosis, but many die with silicosis.[56]

In that year the Chamber's own returns had one black miner dying of silicosis.

53. See Katz, *The White Death,* 'Appendix: the development of silicosis', pp. 215–20.
54. King, 'Silicosis in South African Gold mines', p. 13.
55. See 'Report of the Commission of Inquiry into Safety and Health', pp. 16–17.
56. H. H. Schroder, 'The properties and effects of dust. Environmental engineering in South African mines' (Report, Mine Ventilation Society of South Africa, 1982), quoted in King, 'Silicosis in South African Gold mines', p. 12.

Breaking the barriers

Anglo American had good reason to believe that the various mines acts stretching back to 1911 and the compensation system which has operated since 1913 meant that it would never face litigation over silicosis. The work by Steen *et al.* and Trapido brought no immediate change to work practices or to state regulation. What did bring change was a new political environment. The election of the ANC government in 1994 coincided with a dramatic shift in the legal possibilities for the victims of occupational injury. That shift has its origins in successful litigation by asbestos miners.

Asbestos mining by the British companies Cape Plc and Turner & Newall was carried out in South Africa for almost a century. The work conditions on the mines were hazardous and when they closed in the mid-1980s they left behind large numbers of men and women in the Northern Cape and the Northern Province with occupational disease.[57] Extensive lobbying by community groups led eventually to a claim against Cape Plc, which began in a London court in February 1997. The plaintiffs, represented by the British lawyer Richard Meeran, sought to hold Cape liable for its failure to provide a duty of care for its employees. Over the next three years the legal process was devoted to the question of jurisdiction. Cape Plc wanted the case heard in South Africa, where the injuries had taken place and where the plaintiffs lived. The defence argued for the UK, where Cape's assets are held. On appeal, the Law Lords finally ruled unanimously in favour of the plaintiffs. The issue of a duty of care was then scheduled to be heard. While the Cape case was running, a second set of claims was lodged in South Africa in August 2002 against the corporate giant Gencor. Those claims were made by the civil rights lawyer Richard Spoor on behalf of more than 4,000 men and women who had worked for Gefco, a fully owned subsidiary. It was the first time that such a claim had been brought in South Africa for injuries sustained in the mining industry.

In March 2003 Cape Plc and Gencor reached out-of-court settlements in London and Johannesburg.[58] The settlements were notable for a number of reasons. They were class actions involving thousands of claimants. The Gencor case was the first time that South African miners had won a civil action for injuries sustained in the workplace. The London plaintiffs had breached the corporate veil which Cape's lawyers assumed would protect the company from its South African litigants. Documents spilled into the public domain reveal that the Departments of Mines and Health were aware

57. See J. E. Roberts, *What Is the Price of 80 KGs: The failure of the detection of, and compensation for, asbestos-related disease: social exclusion in Sekhukhuneland* (University of Natal, MA thesis, December 2000).

58. See Jock McCulloch, 'Beating the odds: the quest for justice by South African asbestos mining communities', *The Review of African Political Economy* **32**, 103 (2005), pp. 63–77.

for decades of the hazardous conditions on the mines but did nothing.[59] The degree of collusion between state agencies and industry was so extreme that it threw into question the integrity of the regulatory system on South African mines since the 1930s. The day after the R450 million Gencor agreement was signed, Richard Spoor announced he would turn his attention to the plight of gold miners suffering from silicosis. Mbini is the first of those claimants.

The asbestos and silicosis stories share much common ground. They are connected by the careers of the leading medical researchers in the UK, the US, and South Africa who from the 1930s worked on silicosis and asbestosis. They are connected by the US corporations such as Johns Manville and Union Carbide, which over a period of decades have faced litigation from employees suffering from those diseases. Within South Africa the connections are more intimate. The Cape/Gencor and the Anglo American cases have involved the same lawyers: Richard Meeran and Richard Spoor. Meeran has remarked on the parallels:

> The chronology of the hazard is also very similar. In this case, the mines and the government had specific knowledge of the dangers of dust and the fact that it caused silicosis. While the asbestos issue was subjected to litigation for years – since the 1960s – the gold mining industry has managed to escape justice.[60]

Of particular importance has been the role played by the mining company Charter Consolidated.

The Anglo American Corporation and its associate De Beers are vast enterprises. By 1957 they controlled 40 percent of South Africa's gold production, 80 percent of the word's diamonds, and a sixth of its copper. They also produced most of South Africa's coal. From the 1930s Anglo American developed a maze of interlocking directorships, mutual agreements, and restrictive trade practices. That complex structure, which is characteristic of the South African mining houses, makes identifying the ownership and control of subsidiaries difficult.[61] Cape was a British firm with its head office in London, yet from its foundation it was linked with De Beers. It was also related to Anglo American. During the Second World War, the Oppenheimer company Central Mining & Investment Corporation became the major shareholder in Cape. Central was one of four holding companies within the Oppenheimer group: the others being Anglo American, De Beers, and Rand Selection. By 1949 Central held the majority of seats on the Cape board. In 1969 the Central holding in Cape was superseded by Charter Consolidated, a British mining company, also controlled

59. Jock McCulloch, 'Asbestos, lies and the state: occupational disease and South African science', *African Studies* **64**, 2 (2005), pp. 201–16.
60. 'Anglo being sued for R20m', *The Sunday Independent*, 30 September 2007.
61. See Duncan Innes, *Anglo American and the Rise of Modern South Africa* (Monthly Review Press, London, 1984).

by Oppenheimer interests. Charter held 63 percent of Cape's shares.[62] In 1979 Cape's mines were sold for £15.5 million to Barlow Rand, an Anglo American company. They were then sold on again to General Mining, yet another Oppenheimer enterprise. Charter's controlling share in Cape gave Anglo American a commercial interest in the asbestos industry. It also gave Anglo American's board a reason to monitor the Cape litigation.

Conclusion

The first crisis saw the state, industry, and white labour achieve an accommodation which was maintained for almost a century. That fragile agreement was constantly renegotiated through a plethora of acts and commissions. Underwriting the whole system was the lack of awards for black miners, who bore the burden of occupational disease. The current silicosis crisis has destroyed that precarious balance.

Senzeni Zokwana, who appeared before the Leon Commission in 1994, worked at President Steyn mine in Welkom during the same period as Mankayi Mbini. Zokwana, a shop steward for the NUM, told the Commission that sometimes miners at President Steyn were compelled to work in unsafe conditions. He described a contradictory work setting which was highly centralized in terms of decision making but in which there was a lack of control over risk. 'They (the miners) are under the constant control of management,' he said. 'They do not have the right to decide for themselves. They are controlled underground by the mine bosses and again on the surface, and even get dismissed because of breaking hostel rules.'[63] There was no full-time shop steward and the only reporting on underground safety was to the mine captain. His primary role was with production and production bonuses rather than with safety and occupational health.[64] Zokwana told the Commission that only the right to refuse dangerous work and access to safety training would reduce the number of deaths and injuries and the rate of lung disease. Zokwana is from the Transkei and he presented a picture of hardship among ex-miners, many of whom suffer from lung disease. The roads are poor and men die because they cannot get medical care.[65] The nearest hospital is more than 60 kilometres from Zokwana's village. It has an X-ray plant but it does not report tuberculosis or silicosis cases to the authorities in Johannesburg.

62. See Geoffrey Tweedale and Laurie Flynn, 'Piercing the corporate veil: Cape Industries and multinational corporate liability for a toxic hazard, 1950–2005', *Enterprise and Society* 8 (June 2007), p. 7.
63. Senzeni Zokwana, National Union of Mineworkers, transcript of evidence given before Commission of Inquiry into Safety and Health in the Mining Industry (held at Braamfontein, Johannesburg, 17 August 1994), p. 209.
64. *Ibid.*, p. 205.
65. *Ibid.*, p. 205.

The testimony of Senzeni Zokwana, like the case of Mankayi Mbini, raises many questions. How could the mining houses, with their knowledge of work conditions and their intimate links with the state, have been unaware of the silicosis pandemic? How could the Department of Mines and the SAIMR not have known of the disease burden, given the number of commissions, the millions of dust samples and X-rays, and the hundreds of thousands of annual medical examinations? Were T. W. Steen *et al.* (1997) and Anna Trapido (2000) really the first to recognize the silicosis problem? While those questions may be answered in Pretoria's High Court, the consequences of a century of neglect are obvious. As Neil White has written; 'This occupational diseases epidemic is directly fueled by the transference of health costs from the employer to the state and the individual, which removes the incentive for the employer to effectively limit exposure to silica dust.'[66] The industry has long argued that the unique character of South Africa's deep gold mines makes it inappropriate to judge working conditions against international standards. While that claim is open to question it is almost certain that the degree of collusion between the state and industry in South Africa has been unique.

A number of factors helped to render the silicosis pandemic invisible. The racialized labour process and its supporting compensation system were influential. So too was the insidious character of the disease. One of the most important barriers to knowledge was the nature of the research community in Johannesburg. There was no employment for researchers outside of the Chamber or the state, and it was the Chamber which largely set the research agenda and decided upon the models of data collection. Such data were expensive to process but, as the British Medical Inspector of Factories, Dr Middleton, commented at the 1930 Silicosis Conference, dust counts in themselves mean little.[67] There are so many variables such as the size of the particles, their silica content, and the limits of the instrumentation. The free silica content on the Rand, for example, varies between 35 and 80 percent.[68] As the Johannesburg conferences of 1930, 1959, and 1969 demonstrate, such questions preoccupied researchers at the SAIMR and PRU for decades. There was more and more data but after Mavrogordato important questions were not asked. The Leon Commission commented on a general failure to collect and use data effectively.[69] Leon's comments suggest that the research community was itself one of the barriers to the recognition of silicosis. The work of Steen *et al.* and Trapido shows that if

66. White, 'Is the ODMW Act fair?', p. 20.
67. Dr Middleton, in 'Silicosis: records of the International Conference', p. 30.
68. Dr I. G. Irvine, Dr A. Mavrogordato, and Hans Pirow, 'A review of the history of silicosis on the Witwatersrand goldfields' in 'Silicosis: records of the International Conference', p. 178.
69. See 'Report of the Commission of Inquiry into Safety and Health', p. 41.

Anglo American had wanted to identify the disease burden it could have commissioned follow-up studies of the labour-sending populations.

In terms of scale, the Anglo American case is many times greater than the asbestos litigation and politically far more important. Gold mining may be a declining industry but it still employs hundreds of thousands of men and women. The claim against Cape was supported by both the ANC and the NUM, and by a number of social movements. In contrast, Mbini is not supported by the South African government, nor by the NUM. The reason has much to do with the threat to jobs at a time of economic hardship. The mining industry now employs far fewer men and women than it did twenty years ago, but it remains a major industry which directly or indirectly employs hundreds of thousands of people. In addition, many gold miners are migrant workers and that gives Mbini's claim significance throughout the SADC region. The case may eventually draw the governments of Lesotho, Mozambique, and Swaziland into conflict with the South African government.

The Cape Plc/Gencor claims led directly to Mbini and it seems likely that without that litigation there would be no current action against Anglo American. The accommodation established after 1912 was sufficiently resilient to weather the high rates of silicosis identified by Leon. It took the Cape plc case and majority rule to breach that system. The links between the asbestos and silicosis scandals may also help to explain why the Department of Mines and the Chamber fought so hard during the 1970s and 1980s to save the asbestos mines from their own occupational health crisis. Asbestos was an insignificant industry which at its peak in 1977 accounted for only 3 percent of the value of South Africa's minerals production and employed less than 40,000 men and women. Despite the public health costs the Department of Mines and even the South African Cabinet gave the industry their full support.[70] One possibility is that the Department feared a recognition of asbestos disease would lead investigators directly to the silicosis pandemic, which is exactly what has happened.

Over the past two decades the policy of labour stabilization has seen men staying for longer periods on the gold mines, thereby ending the short-term contracts characteristic of the migrant labour system. The shift to sub-contracting underground work and the use of production bonuses have resulted in worsening conditions.[71] These, coupled with an increase in the annual hours worked, have led to higher rates of noise-induced hearing loss as well as an elevated synergy between silicosis and tuberculosis. Current research suggests that the disease rates are rising. That possibility gives Mbini's claim all the more urgency.

70. See McCulloch, 'Asbestos, lies and the state'.

71. See J. Crush *et al.*, 'Undermining labour: the rise of sub-contracting in South African gold mines', *Journal of Southern African Studies* **27**, 1 (2001), pp. 5–31.