Rodney Ehrlich trained in economics at UCT and Oxford before switching to Medicine which he completed at UCT in 1981. He subsequently worked for the National Centre for Occupational Health in Johannesburg where his interest in lung disease in miners was kindled. After training in occupational medicine and epidemiology at the Mount Sinai School of Medicine in New York, he qualified as a specialist in Community Health at UCT in 1992. He has worked in the Occupational Diseases Clinic at Groote Schuur Hospital since 1990, enabling him to combine clinical medicine with public health. His current research interests are in social epidemiology, workers’ health, tuberculosis and other chronic lung disease. He has held adjunct faculty appointments at the Mount Sinai School of Medicine and the Mailman School of Public Health, Columbia University, New York. In 2005 he was awarded a Fellowship by peer review of the College of Public Health Medicine (SA) (Occupational Medicine) and elected to the Collegium Ramazzini, an international college of occupational and environmental health scientists.

THE BODY AS HISTORY – ON LOOKING AT THE LUNGS OF MINERS

Rodney Ehrlich
Professor, School of Public Health and Family Medicine
University of Cape Town

(Editied version of Inaugural Professorial Lecture delivered, 26 September 2007, University of Cape Town)
I have chosen tonight to share with you an interest that was kindled earlier in my career and that has been interrupted but always resumed. In the next 35 minutes, I want to share with you what I have learned about the lung diseases suffered by miners in Southern Africa.

I have been examining former mineworkers for 23 years. This is not something that my otherwise outstanding training at UCT Medical School in the late 1970s prepared me for, and it was at a fine but much neglected institution in Johannesburg, the National Centre for Occupational Health, that I saw my first example of a diseased lung in a miner. This coincided with a realization, on moving from Cape Town to Johannesburg, that everything around me, particularly in central Johannesburg where I worked, and in the southwestern and then southeastern suburbs where I lived, bore the signs of a former mining camp, and that the close association between the wealth of this African city and the surrounding dumps told another, essential, tale of South Africa.

In telling this tale tonight, I want to pay tribute to three colleagues who were there before me, in other forums on other occasions. One is Prof. Tony Davies, my former boss and mentor at the National Centre for Occupational Health, who gave me my first insight into what the public health impulse means as a guide to professional calling. Another is Prof. Jonny Myers, who has kindly agreed to offer the vote to thanks tonight, who was one of the pioneers of critical health studies that inspired a number of our generation to go into occupational health, and who played a role in my return to Cape Town in 1990. The third is Prof. Neil White, who died before his time, and with whom I worked at the Occupational Diseases Clinic at Groote Schuur Hospital for 15 years. Any historical account of mineworker health in late 20th century in South Africa will find Neil’s name all over the collected body of research, advocacy and testimony.

This presentation is meant in a small way to signal the need continue this work, and hopefully to persuade some of my students of its importance, so as to prevent what I believe to be important truths about our society from fading from view once more under the powerful forces for amnesia.

The first part of the title of this talk, *The body as history* implies a number of narratives pertaining to miners, historical and personal. The second part of the title – *on looking at the lungs of miners* – roots the story visually in chest radiology, the medical science and it to this that we now turn.

**Radiology**

Radiography, the technology, followed the discovery of x-rays by Rontgen in 1895 and found immediate use in the South African mining industry. According to Elaine Katz in her seminal history of silicosis in the early years of the goldmining industry in South Africa, *The White Death*, QUOTE “In 1916 the Johannesburg medical fraternity laid the world-wide foundation for modern diagnostic practices. The Miners’ Phthisis Medical Bureau introduced routine radiography for miners…” END QUOTE. Further, mass miniature radiography was developed in South Africa by KGF Collender in the 1930s, while working for the Witwatersrand Native Labour Association, the primary mine labour recruiting organization across the subcontinent (a name now tellingly modernized to the The Employment Bureau of Africa).

Katz points out, however, that it was another thirty years before routine radiography was applied to all African miners. We should note that this racialisation of access to medical examinations was part of the wider racialisation of the labour process on the mines and the accompanying emergence of attitudes and beliefs about African labour that not so much reflected what became known as apartheid...
South Africa but in many significant ways created it. The divergence between between white labour and black labour on the mines prefigured many other separations in our history, and it is mainly about black mine labour that I want to talk about tonight, understood as a socioeconomic category. This is not because white miners did not suffer – on the contrary, the first casualties of the dust of the Witwatersrand were heavily concentrated among migrant workers from Cornwall in Britain. According to one of many official commissions of the time, in 1912 the average working life of a rock driller on the Witwatersrand goldmines was seven years and the average age at death, 33 years.

Over the past century the centrality of radiology in miners’ lung disease has not changed. These diseases have, however, undergone a change in our understanding. The original term was *miner’s phthisis* – phthisis alone being an historical term for tuberculosis, from the Greek for “wasting”.

While understood in the context of mining to be a disease associated with dusty work, its pathogenesis, i.e. its natural biological history, was poorly understood. In fact it combined the two diseases that we today know as *silicosis*, fibrosis of the lung due to inhalation of silica dust, and *pulmonary tuberculosis*.

With the discovery of the mycobacterium tuberculosis by Koch in 1882, the causal conception shifted to the infectious nature of miner’s phthisis, in which dust was seen as a contributor or vehicle but not as the primary cause. (Interestingly, older black miners today still use the term phthisis to refer to tuberculosis, and often still have only a sketchy concept of silicosis as a separate and significant disease).

However, it soon became apparent that tuberculosis was not a necessary condition for this form of miners’ lung disease. Dust alone could indeed produce miners’ phthisis. Thus *silicosis* came to be understood as a distinct disease in these Rand goldminers.

This distinction is the basis of our system of screening for and compensating miner’s lung disease. It is, however, also the basis for attempts by the mining industry from time to time over the years to convert pulmonary tuberculosis in miners from an occupational disease into to a community acquired, non-occupational disease with all the shedding of responsibility that would go with such a transformation.

In keeping with our visual theme, we need to consider for a few moments, particularly for those in our audience unfamiliar with these images, what one sees when looking at plain radiograph.

(SLIDE: Chest Radiograph of normal lung)

A normal chest radiograph is an exercise in contrast: distinguishing structures which absorb x-rays such as the heart, blood vessels and ribs, and therefore appear white on the film, from those that allow the x-rays through and appear black, essentially the air filled lungs. Anything other than air that fills the lungs or replaces normal lung tissue, whether it be fluid, inflamed tissue, cancer or scar tissue will interfere with this pattern by showing up white. Silicosis and tuberculosis are two causes of these shadows – hence the importance of chest radiography in mining.

[SLIDE : Definitions of silicosis and silica (Text: Silicosis = fibrosis of the lung due to silica dust. Silica = silicon dioxide, component of the earth’s crust and concentrated in hard rock such a gold bearing ores). Electron micrograph of silica particle]
The silica dust that gives silicosis its name is *silicon dioxide*, a natural component of the earth’s crust and one that is unfortunately highly concentrated in the rock in which gold is found. In order to liberate the gold-bearing ore from the earth, large quantities of silica bearing dust have to be liberated as well, in the act of blasting, drilling, ore carrying and crushing.

(SLIDE: Chest radiograph of simple silicosis).

The characteristic features of the silicotic radiograph are the rounded white shadows which start in the upper half of each lung, extending over time to fill much of the lung.

SLIDE  (Whole lung section of gross pathology of silicosis; microscopic slide of lung tissue: silicosis)

When the whole lung is examined at post-mortem examination (as in the section on your left in this slide), a slice of the lung will be seen to contain a number of hard pigmented nodules which, when sufficiently numerous, produce the x-ray shadows on the radiograph. When a fragment of one of these nodules is examined under a light microscope after special staining, it reveals that the normal lace like open pattern of the lung (as illustrated in the section on your right in the slide) has been replaced by concentric rings of fibrosis.

A surprising feature of this type of silicosis, and one that is frequently cited by those seeking to minimize its importance, is that the sufferer may not be bothered by any of the breathlessness, fatigue, cough or phlegm that characterizes chronic lung disease in general. This has led medical texts and other commentators to refer to this form of silicosis as *simple silicosis* or *benign silicosis*, the latter being an unfortunate misnomer in the setting of goldminers’ disease in South Africa, as I hope to show.

SLIDE: (Chest radiograph of progressive massive silicosis)

For reasons that are not well understood, but must include more intense exposures to silica dust, in some silicosis sufferers the small nodules in the lung coalesce, to form large masses that replace much of the upper zones of the lung. I reproduce a particular gross example here for projection purposes. This is progressive massive fibrosis, a particularly disabling form of silicosis. The reduced life expectancy associated with this form of disease is undoubtedly one of the reasons for its rarity in surveys of active workers and in case series collected by compensation authorities.

SLIDE:  (Chest radiograph of healed pulmonary tuberculosis superimposed on silicosis)

Pulmonary tuberculosis is frequently superimposed on pre-existing silicosis. This may make it difficult to distinguish which of the two diseases predominates in the picture and whether the changes suggestive of tuberculosis signify old damage or active infection. Not infrequently we see at our clinic former mineworkers with silicosis who have been treated by the health services for tuberculosis without any bacilli being isolated. Understandably then, the interpretation of what exactly constituted miner’s phthisis in the historical record is difficult.

For goldminers, and particularly former goldminers, once active tuberculosis has been excluded as a diagnosis, the health care system loses interest in them. Unlike in the case of tuberculosis, there is no accepted treatment that can reverse the lung fibrosis, nor speed up the elimination of retained silica dust from the lung. The miner disappears from the screen. However, his lungs retain his story.

**Prevalence of silicosis**
In the mid-1990s two groups independent of the mining industry, under the guidance of Neil White, ventured into rural Botswana and rural Transkei to try to measure the true burden of silicosis, and lung disease more generally, among former goldminers. The Thamaga study in Botswana found that 25 percent of a random sample of former miners had silicosis. The other, in the Libode district of the Transkei, and recorded in Anna Trapido’s brilliant doctoral thesis, found a prevalence of the order of 30 percent. These were shocking figures and vindicated an earlier piece by Tony Davies in the South African Medical Journal titled “Sound an alarm”, based on his observations of the neglect of the statutory rights of former mineworkers in what was then the NE Transvaal. One of the criticisms of these studies, however, was that they consisted of samples in which disabled miners are likely to have been concentrated.

In 2000 while on sabbatical I examined a 600 chest radiographs as a part of study of former Basotho miners initiated by Gavin Churchyard of Aurum Health Research, an important source of mining epidemiology in South Africa, and Neil White. What distinguished these from the many other radiographs I had previously looked at was that they were from a single workforce and not sick patients referred to our clinic, and that these former miners had worked on the mines only one to two years before the radiographs were taken. They had also left the mines after being retrenched and not for health reasons, in other words they qualified for what is sometimes called a “healthy survivor cohort”. I was shocked by the extent of disease in these recently active miners – silicosis, much of it advanced, and lung destruction by tuberculosis.

We have subsequently confirmed a prevalence of silicosis of 23 percent in this group, and 26 percent with radiological evidence of past tuberculosis. This in turn confirmed a finding in a similar study of in service miners on a different mine, where we found a prevalence of silicosis of 20 percent among older long service miners. All these lines of evidence are persuasive – among longer service black miners in active service, as among ex-miners, about one in four has silicosis.

In 1916-17 the official statistics of the Miners’ Phtisis Medical Bureau recorded a prevalence of silicosis with or without tuberculosis of approx. 0.2% among 190 000 black workers ostensibly examined on mines. In 1945-1946 this prevalence remained at 0.25% among 290 000 black miners examined.

What then is going on - a century after the first epidemic of miner’s phthisis in South Africa?

**Establishing the true burden**

It is an extraordinary feature, in looking back at a century of mining lung disease in South Africa, that so little has been known until recently about silicosis among black miners. While the lethal form of accelerated silicosis was all too clearly recorded among white miners at the beginning of the century, very little was recorded among black miners. The conventional view was that the main risk to black workers was infection, namely tuberculosis and bacterial pneumonia, conditions to which, in the view of many commentators, they were racially susceptible. Furthermore, black miners were believed to be protected from silicosis by short contracts and breaks in service that were the central feature of the migrant labour system. Rural African life was also conveniently and frequently cited as having extraordinary recuperative powers.

At the same time, a number of technical improvements to reduce dust levels were introduced into underground goldmining by the industry, under pressure from the state, the white miners’ unions,
medical and engineering experts and a public opinion disturbed by the high death rates among white miners. As a result the epidemic of accelerated silicosis faded, to be replaced by more chronic forms of silicosis which took longer to acquire and had a lower rate of the more serious complications.

The historian Shula Marks in her recent article, *The Silent Scourge: Silicosis, Respiratory Disease and Goldmining in South Africa*, has argued that the risk of silicosis among black miners was systematically underestimated over most of the century. A number of factors conspired to produce this outcome, including *a priori* beliefs by medical examiners and the focus on tuberculosis, the adjustment of medical standards as convenient in response to the needs of a mine for labour, the generally cursory examinations of large numbers of black mineworkers – one historical image of a doctor with a stethoscope examining a few thousand recruits in one day stands out - and even the desire of medical examiners to protect mineworkers from service termination and loss of livelihood on the basis of what they considered a benign disease.

In addition, until 1985 the policy of the goldmining industry was one that has been euphemistically called “medical repatriation”, which meant that an active miner or one applying for a new contract found to have tuberculosis was sent back to his home area, “to recuperate” as it were, in the early years, and with a medical letter of referral to whatever treatment services there were, in later years. It is likely that a number of such miners were unrecorded silicotics.

In any case, all the data that were collected on silicosis in black miners effectively came from black miners in active employment on mines or applying for such employment. Almost nothing was known about the burden among the large and accumulating number of ex-miners outside the system during this period until the mid-1990s. To get some idea of how many such people we are referring to, the total number employed at the peak of employment of the industry 1986 was over 500,000, having declined to half that number of in the past two decades. Allowing for turnover, the number of former miners in Southern Africa must run into the millions. We must also note a substantial proportion, estimated at 30% of these, live in some of our poorest neighbours, particularly Lesotho, Mocambique and Malawi.

Given this history of neglect and obscuration, it is unlikely that we can reconstruct the true picture of silicosis among black goldminers from the first part of the century, other than to assert that it was no doubt worse than officially recorded.

However, it is highly plausible, as argued first by Jean Leger and later by others, that there has indeed been a significant rise in the incidence of silicosis in the latter part of the century owing to what has been called *labour stabilization*. This process includes the conversion of recurrent short term contracts into long-term arrangements, with the miner returning to the same mine year after year and accumulating long service. This process has been intensified by the continuous downsizing of the mining workforce with an increasing proportion of long service miners remaining. It is thus an irony that the historical introduction of stability to the employment contract of black miners should come at the cost of a much greater risk of silicosis a century after the commencement of the migrant labour system on the South African mines.

**Cohorts**

However, even the studies I have cited suffer a number of features likely to obscure our understanding of true risk of silicosis among black mineworkers. They are all what might be called snapshots - in epidemiologic terminology cross-sectional studies - of what we can think of as the movement of a
human population through time. Missing would be those miners who died early due to the lung disease. Workplace studies compound this selection effect by excluding those who failed fitness examinations or have not returned to the rigours of mining work because of ill health. Annual reports by compensation agencies, which are sometimes used, include only those who have managed to gain access to the system, notoriously impervious to black miners. Finally, all such studies suffer from cohort dilution, i.e. including people of different ages, different production eras and different service lengths, which are difficult to disentangle in a snapshot study.

In contrast to these snapshot studies is the study of cohorts. We are all members of cohorts, notably our birth cohort, each with a unique history. Epidemiology, the study of patterns and causes of disease, is starting to elucidate how our health prospects throughout our life course are influenced by circumstances during pregnancy and early childhood. These influences in turn are not random but the product of the particular social and historical arrangements of our society, which themselves require understanding. This synthesis of biological and social perspectives synthesis is encapsulated by the Krieger and Davey Smith in their concept of *embodiment*:

> OPEN QUOTE: “…bodies provide vivid evidence of how we literally embody the world in which we live, thereby producing population patterns of health, disease, disability and death. ….These aspects of our being not only are predictive of future health outcomes but also tell of our conjoined social and biologic origins and trajectories” END QUOTE

Our occupational cohort, shared with the people with whom we enter an occupation or industry, is another potentially powerful influence on health. However, despite the claimed ability of some people to guess other people’s occupations at dinner parties, most of us here tonight are remote from a world in which our occupations are clearly inscribed in our bodies. For a medical observer in the 18th century such as Bernadino Ramazzini, the founding figure of occupational medicine, such inscription was commonplace. The best known such characterization today is a figure of speech, “as mad as a hatter” – harking back to the neuropsychiatric disease due to mercury poisoning, mercury being used to stiffen the felt of the hat. Many of these diseases have faded into history such as *Weaver’s bottom*, *Phossy jaw*, the disfiguring erosion of the jawbone due to white phosphorus in match factory workers. One, however, has not – Potter’s Rot – the evocative name for silicosis among English ceramics workers.

The occupational cohort study is an attempt to examine the natural history of a working population so as to provide a true measure of average lifetime risk of disease, risk being defined here as the probability of suffering the disease at some point in one’s lifetime. The only modern cohort study of goldminers in South Africa is that of white goldminers, recorded by Gerhard Sluis-Cremer, Eva Hnizdo and others. Even among white mineworkers, who have for a long time experienced lower intensities of dust than black mineworkers because of the mining colour bar, these researchers found an accelerating proportion of the cohort developing silicosis with each dusty year in service. Extrapolating from their curves, at typical dust concentrations found on gold mines during the 1990s, half of a cohort doing continuous dusty work on the goldmines could be expected to develop silicosis after twenty years.

Equally significant for our current argument, more than half the cases of silicosis became apparent after the mining exposure ceased.

There is a reason for this latter finding - what has been called, in respect of another toxic mineral dust, asbestos, the long arm of latency. Silica is a biologically active dust. Once accumulated in sufficient quantities in the lung, it initiates a number of biological processes which do not stop because someone
has ceased to work with dust. In particular, silica particles are toxic to the immune cells which form one of the first lines of defence in the lungs – the pulmonary macrophage.

SLIDE: (Pulmonary macrophage attempting to engulf silica particles).

This fateful encounter produces a number of unfortunate consequences. One of these is the cascade of immunological and biochemical processes which results in fibrotic or scar tissue.

Another outcome of macrophage impairment has had even more serious consequences. The macrophage also plays an important role in the first line of defence against mycobacterium tuberculosis. For reasons that are still poorly understood, silica impairs this function.

It is common cause in the industry that goldminers with radiological silicosis are three to four times as likely to develop pulmonary tuberculosis than goldminers without silicosis. What is not as well known is that an elevated risk of tuberculosis is present even without silicosis on the radiograph, i.e. due to subclinical silicosis or to dust accumulation in the lung on its own. However, what Jill Murray and Eva Hnizdo have shown in the cohort of white miners described earlier, is that the elevated risk of tuberculosis accompanies miners after they have left the industry: and that most cases of tuberculosis in this cohort - 85% - occurred after the miners had ceased dust exposure.

With regard to pulmonary tuberculosis then, silica retained in the lung and silicosis are in effect a type of acquired immune deficiency. It should be noted that the compensation legislation for miners’ lung disease in South Africa limits any liability to pay compensation for tuberculosis alone (i.e. in the absence of silicosis) to the first 12 months after the end of mine service.

Since the commencement of the industry, there has never been a proper cohort study of black mineworkers in South Africa which has captured their full experience after they have left the mine. The difficulties of tracing contract miners has historically been cited as the reason for this. This should not longer be tenable. First, the regional recruiting agencies have a long established networks and routes of recruitment. Second, an industry that is able to mine 3 km underground is clearly not easily discouraged by difficult logistics.

HIV

This grim convergence of silica and tuberculosis among miners has been greatly worsened by the advent of HIV. Goldmining industry estimates cite HIV seroprevalences among the workforce of between 20 and 30 percent. The epidemiology of HIV infection is complex and not fully understood. However, migrant labour and transactional sex associated with single sex mine compounds are surely part of the story of the spread of HIV infection in Southern Africa, joining its historical precursor syphilis in this regard.

Tuberculosis is the face of HIV infection for many South Africans and HIV infection increases the risk of tuberculosis among goldminers four fold compared to miners not infected with HIV. However, among goldminers who are both silicotic and HIV positive, the risk is increased 12 fold i.e. 1 200 percent, compared to goldminers who have neither silicosis nor HIV infection.

After 100 years of industrial development, for black goldminers in Southern Africa at the turn of the 21st century, “miner’s phthisis” is back with a vengeance. We should resist the temptation to view this as somehow regrettable but inevitable, an unavoidable consequence of earning a living in an economy
with few opportunities for unskilled men, a living for which mineworkers should moreover be grateful, but rather try to see it as the product of South Africa’s particular history, what Didier Fassin in the context of competing explanations of AIDS has called the *embodiment of inequality*, an attempt to explain *QUOTE* “how history becomes physical reality and how biological facts become social facts.”*END QUOTE*

**Compensation**

What of compensation? Workers’ compensation legislation, which dates from the second decade of the 20th century in South Africa, effectively monetises loss of employment opportunities and permanent impairment suffered as a result of an occupational injury or disease in the form of a system of no-fault insurance. In return, such legislation removes the right of civil action for damages by workers against employers. The latter has been referred to as the “historic compromise”, the argument being that workers were seldom successful in such civil suits. South Africa, as in other mining countries, established separate legislation to compensate mineworkers’ lung disease. However, the legal ability of mineworkers to sue their employers outside of this legislation is still being tested in South African courts. If finally accepted by the courts, this principle would have momentous consequences for the industry and mineworkers with occupational lung disease.

When I started examining miners, black miners were awarded a once off payment of R1200 for tuberculosis and R2000 for silicosis. They were on average one tenth of the amounts awarded to white miners, reflecting what was once called the “civilized wage ratio”. Racial differentials were replaced in 1994 by waged based differentials such that miners now receive approx. R27 000 for silicosis alone and R70 000 for silicosis plus tuberculosis. If you think the latter is a considerable sum, consider a 45 year old miner with four dependents and significantly reduced employment prospects. As Anna Trapido, quoting Chambers has written: *QUOTE* “the body is the poor person’s greatest and uninsured asset. If this asset is devalued or ruined, far from being an asset it becomes a liability that has to be fed, clothed housed and treated….” *END QUOTE*

Notwithstanding the paltriness of the payouts, black former mineworkers have had very little access to this system. Even when they do have access, as via our clinic, it is not unusual for them to wait up to five years for their payment. Neil White and Anna Trapido, using the Transkei findings and an assumption of two million former mineworkers throughout Southern Africa in the late 1990s, extrapolated a figure of the order of R10 billion in unpaid statutory compensation liability at that time, most of which, if paid out, would flow to the poorest regions of the subcontinent. However, as has been noted, the system has developed and survived historically on the basis that black mineworkers would have de jure but not de facto access to its benefits.

The miners’ compensation Fund is financed by levies on the mining industry although at various points the state has had to supplement this fund from general taxation and in fact pays for the administration of the system. In 2004 the Fund paid out approximately R110 million. A 2004 actuarial study confirmed, however, that the Fund was technically insolvent even on the basis of current claims, and that the levies paid by the mining industry needed to be raised 100 fold over a period of 15 year period. In the same year the auditor-general referred to the financial controls of the Fund as a shambles.

**A question of identity**

Even should a former mineworker find his way into the system a further hurdle awaits – to prove his goldmining service. Some record of mine service has always been required, but until recent years the
miner’s own recall was accepted. In response to some incidents of fraud, the Compensation authority is now insisting on some original record of service.

In our experience only one in two and perhaps as little as one in three mineworkers can produce such evidence. A few, particularly those with more recent service and longer contracts, have retained their personnel card and may have an extant record at the mine. Frequently, however, service is long past and consisted of a number of contracts at different mines. A surprising number of mineworkers have lost their documents in fires which frequently ravage informal settlements and the remaining scraps of documents tying them to their past. As far as the system is concerned, their mine service has been expunged from the record.

Simon Szreter, the English public health historian, has narrated a story of the English rural poor in the 17th and 18th centuries in England and Wales. With large numbers of such poor forced by economic forces to seek work in the towns, an early universal social security system was provided by the so-called Poor Laws. However, personal identification was required to render yourself eligible for Poor Law assistance. This identification was enabled by a compulsory system of recording births, marriages and deaths at parish level. Szreter goes on to argue that together with a system of Justices of the Peace to enforce these laws, this right to personal registration was central to the operation of the Poor Laws, and that these factors were the reasons the British Isles achieved sustained economic growth and effectively abolished famine related mortality by the late 18th century while other successful trading nations fell behind.

The history of South Africa reveals the double edged nature of registration and identification, through the dompas and its centrality to apartheid’s design. In 1994 it was replaced by a system appropriate to a democratic citizenry. It is a terrible historic irony that many mineworkers thereby lost the record of their past service recorded in those documents. The statutory compensation to which miners with occupational lung disease are entitled is by no means a universal system of social insurance. It has many faults which have been well documented. However, for many former mineworkers it represents a small cushion against dire poverty.

For South African goldminers, there is one historical record which ties them irrevocably to a mining past, which while not entirely reliable, certainly serves what might be called prima facie evidence of service. That historical record is their lungs and the stamp of silicosis or silicotuberculosis to be found there.

Conclusion

We come to the end of our tale. At the Commission of Enquiry into the Health and Safety of Miners in the 1994, also known as the Leon Commission, the Chamber of Mines’ submission relied heavily on a discourse on the difficult economic circumstances of mining. The threats to the industry of rising costs and declining ore grades are central to narratives in defence of the mining industry’s actions. What is almost always missing from such narratives are the voices of black mineworkers and an attempt at an honest accounting of the toll in health and life against the socioeconomic benefits the industry has brought to Southern Africa. It is undoubtedly true that the mining industry laid the basis of industrialization and of modern South Africa and that, if in no other way than through the industry’s support of tertiary education, we are all beneficiaries. I do not pretend to be able to draw up a final balance sheet. However, we do need to ask ourselves why there is such little overlap between the modern beneficiaries and casualties of the system.
We need to pose the questions as to why 120 years after the commencement of this powerful industry there is such a large and even rising toll of miners’ lung disease, why it continues to be hidden from view, why there is no proper accounting of the costs being borne in the most remote regions of the subcontinent and why the compensation system is a shambles.

The Leon Commission concluded in 1994 that there was no evidence of an improvement in dust levels on the mines during the preceding 70 years. The industry has recently committed itself to eliminating any new cases of silicosis among the entrant cohorts of miners from 2008. Whether this is credible or feasible remains to be seen. However, it will clearly be insufficient to use only cases detected in service as the target while not putting in place some means throughout the subcontinent for detecting cases (and I should add supporting the treatment of such cases for tuberculosis) after workers have left the industry.

It has been my privilege, with colleagues, some of whom I regret are not here tonight, to be an observer of the depredations of minework on the lungs and health of miners, to discern a history that extends beyond the often constricted circumstances of their current lives, to try to record with the scientific method the extent of damage done, and to assist with the amelioration that even the small amounts of compensation brings.

The path to socioeconomic justice in this matter lies in the hands of the mineworkers and former mineworkers themselves. However, in service of such efforts to achieve justice, I believe that medical and epidemiological witness are needed more than ever to prevent the historical experience of succeeding generations of mineworkers, as inscribed in their lungs, from fading from our view.

I thank you.

(References available on request: Rodney.Ehrlich@uct.ac.za)