A CENTURY OF MINERS’ PHTHISIS ON THE SOUTH AFRICAN GOLD MINES. ANY END IN SIGHT?

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ABSTRACT

Since its inception in 1886, high rates of occupational lung disease, particularly “miners’ phthisis” (silicosis and/or pulmonary tuberculosis) and accompanying racial disparities have been a feature of the South African gold mining industry. While mortality from silicosis and pulmonary tuberculosis fell among white miners during the first half of the 20th century, there was scanty information on silicosis among migrant black miners, it being widely held that high turnover and relatively short service protected them against silicosis. Data collected for statutory purposes on examinations of black workers showed an incidence of silicosis, with or without accompanying tuberculosis, of the order of 2 to 3 per 1,000 between 1916 and 1947.

While these compensation figures had risen to 5 or 6 per 1,000 by the late 1980s, prevalence studies on black miners were lacking. When such studies were eventually done, both independently of and from within the industry, a prevalence of silicosis among longer service workers of the order 20% to 25% emerged using the ILO profusion threshold of 1/1. Commentators have noted that there is no evidence that silica dust concentrations in the South African gold mines have been lowered substantially since the 1930s. The recent findings cited above suggest a rising prevalence of chronic silicosis among black miners, arguably to a level not recorded before in the history of the industry. Part of this rise may be due to historical factors that have served to obscure this burden, but the “stabilization” of the black workforce due to changes in the industry resulting in longer continuous service, is a more likely explanation. Combined with a high prevalence of HIV infection among miners (of the order of 20% to 30%), the pulmonary tuberculosis incidence rate has
risen to extraordinary levels of 3 000 per 100 000 per annum and higher despite intensive tuberculosis case finding and treatment in mine medical services. Given the large number of men throughout Southern Africa who have worked on the gold mines this epidemic of silicosis and related tuberculosis among black mineworkers is a public health crisis, but one hidden by the dispersion of former mineworkers. The statutory compensation system is grossly underfunded from levies on industry, while access by black mineworkers is poor, thus shifting the costs of ill health to poor rural areas of the subcontinent. While the industry has committed itself to eradicating silicosis in new entrants by 2013, a surveillance mechanism which takes into account the latency of silicosis and tuberculosis is still lacking.
INTRODUCTION

This paper seeks to provide an historical overview of silicosis among black workers on the South African mines and to pose the question of why 120 years after commencement of the industry, there remains a severe epidemic of silicosis and pulmonary tuberculosis in this population. It also seeks to call attention to the huge burden of undiagnosed and uncompensated silicosis and related lung disease among former mineworkers throughout Southern Africa. This exposition draws on the authors’ personal experience in examining former mineworkers for the past 23 years in an academic hospital clinic, as well as on epidemiologic and historical studies and reports of official inquiries and compensation agencies.

The South African gold mining industry has played an important role in the historical development of ideas about silicosis and pulmonary tuberculosis and in technologies for their control (Katz 1994; Packard 1989). The early years of the industry have been dramatically captured by Katz in her seminal work, *The White Death* (Katz 1994). For example, following the discovery of x-rays by Roentgen in 1895, radiography found immediate use in the South African mining industry, where the medical profession contributed to the development of modern diagnostic practices and routine radiography for miners in the early part of the 20th century (Katz 1994). In the 1930s, mass miniature radiography was developed in South Africa by Collender while working for the Witwatersrand Native Labour Association, the primary mine labour recruiting organization across the subcontinent (a name later modernized to the The Employment Bureau of Africa. (Marais et al. 2005).
However, it was another thirty years before routine radiography was applied to all African miners (Katz 1994). 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 on the mines between white labour and black labour (understood here as socioeconomic categories) prefigured many other separations in South African history. 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 (Burke et al. 1978). According to one of many official commissions of the time, in 1912 the average working life on the Witwatersrand gold mines of a rock driller, most of them white in that era, was seven years and the average age at death 33 years (Katz 1994). While the centrality of radiology in detecting miners’ lung disease has not changed, there has been a change in the conceptualization of these two diseases and accordingly in the legal and administrative means used to deal with them. The original term was miners’ phthisis – phthisis on its own 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, the pathogenesis of miners’ phthisis was at first poorly understood. With the discovery of the mycobacterium tuberculosis by Koch in 1882, the causal conception shifted to the infectious nature of miners’ phthisis, in which dust was seen as a contributor or vehicle but not as the primary cause (Katz 1994). In fact, black miners in South Africa
today still use the term phthisis to refer to tuberculosis, and many 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 gold miners. This distinction between silicosis and pulmonary tuberculosis is central to the South African statutory system of screening for and compensating miner’s lung disease. It is also the basis for historical attempts to limit the mining industry’s liability for tuberculosis by seeking to exclude the disease (in the absence of silicosis) from compensation legislation for occupational disease (Donsky 1993).

A feature of silicosis, and one sometimes cited to diminish the importance of the condition, is that the sufferer may be asymptomatic at the time of examination. This has led to the use of terms such as chronic simple silicosis and benign silicosis, the latter particularly being an unfortunate misnomer in the setting of gold miners’ disease in South Africa. Radiologically, 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. In our experience, it is not infrequent for silicosis to be treated as pulmonary tuberculosis on the basis of radiological changes alone without any bacilli being isolated. Understandably then, the interpretation of what exactly constituted miners’ phthisis in the historical record is difficult.
THE PREVALENCE OF SILICOSIS AMONG BLACK MINERS IN SOUTHERN AFRICA

In the mid-1990s two groups of researchers ventured into labour-sending areas of rural Botswana (Steen et al. 1997) and rural Transkei (Trapido et al. 1998; Trapido 1999) in an effort to measure the true burden of silicosis, and lung disease more generally, among former gold miners. The Thamaga study in Botswana found that 25 percent of a random sample of former miners had silicosis (defined as a profusion ≥1/0 on the International Labour Organisation (ILO) scale (Steen et al. 1997, International Labour Organisation 1980). The other study, in the Libode district of the Transkei and recorded at length in Trapido’s doctoral thesis, found a prevalence of silicosis (using a stricter definition of an ILO profusion ≥ 1/1) of between 20 and 26 percent, depending on reader (Trapido et al. 1998). These were shocking figures and vindicated an earlier piece by Davies in the South African Medical Journal titled “Sound an alarm”, based on his observations of the neglect the statutory rights of former mineworkers in what was then the North Eastern Transvaal (Davies 1994). One of the criticisms of these rural village studies, however, was that they consisted of samples in which older or disabled miners are likely to have been concentrated (La Grange 1996).

However, a subsequent study of older in-service black mineworkers (range of mining service 6 – 34 years) confirmed a high prevalence of silicosis (profusion ≥ 1/1) of approximately 20 percent even in these active miners (Churchyard et al. 2004). The picture has been completed by a study undertaken of Basotho miners shortly after they were laid off from their mine of employment for economic reasons (Girdler-Brown et al. 2008). This
last study provides a view of an ex-mineworker cohort early in its natural history and is distinguished from the earlier studies in that these miners had been in employment on the same mine only one and a half years before the radiographs were taken and had not been selected out for health reasons. A prevalence of silicosis (profusion ≥ 1/1) of 23 percent has been found. All these lines of evidence are persuasive – among longer service black miners in active employment on the South African gold mines, as among ex-miners, between one in five and one in four has silicosis.

The official statistics of the Miners’ Phthisis Medical Bureau recorded an incidence of silicosis among black miners, with or without tuberculosis, of approximately 2 to 3 per 1,000 during the period 1917 to 1945 (Donsky 1993). [The numbers of black workers examined fluctuated between 190,000 and 360,000 during this period (Donsky 1993)]. By the late 1980 this compensation based incidence had risen to 5 to 6 per 1,000 (White 1995). What then is going on - a century after the first epidemic of miners’ phthisis in South Africa?

ESTABLISHING THE TRUE BURDEN OF SILICOSIS AMONG BLACK GOLD MINING WORKERS

It is an extraordinary feature in looking back at a century of gold 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 noted 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 (Katz 1994, Packard 1989, Marks 2004).

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 cited as having recuperative powers (Katz 1994).

At the same time, a number of technical improvements to reduce dust levels were introduced into underground gold mining 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 (Katz 1994). 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.

Marks (Marks 2004) has recently argued that the risk of silicosis among black miners was systematically underestimated over most of the century. A number of factors have 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 in Marks’ account - and the practice of medical examiners seeking to protect mineworkers from service termination and loss of livelihood on the basis of what they considered a benign disease (Marks 2004).

In addition, until 1985 the practice in the gold mining industry, first as an industry practice and later under statutory regulations determining fitness for work, was one of “medical repatriation” (White 1995). This meant that an active miner, or one applying for a new
contract, found to have tuberculosis was sent home, to “recuperate” as it were in the pre-treatment era, or with some initial treatment and then referral to whatever local treatment services there were in the later years (White 1995). It is likely that a number of such miners were unrecorded silicotics.

Until the rural studies of the 1990s, data collected on silicosis in black miners for most of the century were derived from examination of miners in active employment or applying for such employment or were based on data collected by statutory compensation agencies, notoriously inaccessible to black miners. While analysis of the latter has enabled secular trends to be discerned (White 1995; Leger 1989; King unpublished), the true picture of silicosis among the large and accumulating population of former miners has remained obscure. The total number employed at the peak of employment of the gold mining industry in 1986 was over 500 000, having declined to half that number in the past two decades (Crush et al. 1999). Allowing for turnover, the number of former miners in Southern Africa still alive must run into the millions. We must also note a substantial proportion, between 30% and 50% of these, are from outside South Africa’s borders and live in some of South Africa’s poorest neighbours, particularly Lesotho and Mozambique (Crush et al. 1999).

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

The retention of mineworkers with treatable tuberculosis on the mines after 1985 is likely to have increased the prevalence of mineworkers with silicosis in active service (White
1995). However, it is highly plausible, as argued first by Leger (manuscript unpublished) 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 (Leger 1989). 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 greater 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.

However, even the recent cross-sectional studies suffer from a number of features likely to obscure our understanding of true lifetime risk of silicosis among black mineworkers. Missing would be those miners who died early due to their 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. 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.

The only modern cohort study of gold miners in South Africa is one of white gold miners, reported on by Sluis-Cremer, Hnizdo and others (Hnizdo et al. 1993, Hnizdo et al. 1998). Among the white mineworkers, who have for a long time experienced lower intensities of dust than black mineworkers because of the racial job hierarchy on the mines, these
researchers found a steep linear exposure response curve after a cumulative exposure to respirable silica of approximately 2.0 mg-years/m$^3$ (Hnizdo et al. 1993, White 2001). Unfortunately we do not have representative published dust exposure data on underground mineworkers over the relevant decades which would enable accurate construction of cumulative silica exposures and extrapolation from this exposure-response curve for different categories of miner. For any individual mineworker the percentage silica in the ore and his role in the underground production process are central to determining his cumulative exposure. However, for the industry as a whole the Commission of Enquiry into the Health and Safety of Miners in 1994, also known as the Leon Commission, concluded that there was no evidence of an improvement in dust levels on the mines during the preceding 70 years (Republic of South Africa 1995). A summary of 26 000 underground dust measurements on 48 gold mines from 1995 to 1997 found that only eight of the mines had all of their time weighted average respirable silica concentrations below 0.1 mg/m$^3$, the legal standard (National Centre for Occupational Health 1999). A more recent study of dust concentrations on two gold mines found average area respirable silica concentrations ranging between 0.045 and 1.8 mg/m$^3$, varying by mine, operation and silica concentration (Biffi et al. 2003).

Using the exposure response curve developed among white miners, a cumulative incidence of silicosis of 25 percent would be reached after fifteen years of respirable silica concentration at 0.2 mg/m$^3$, which is well within the range of published dust concentrations (National Centre for Occupational Health 1999; Biffi et al. 2003).
What is equally significant is that more than half the cases of silicosis in the 
avovementioned cohort became apparent after the mining exposure ceased (Hnzido et al. 1993). However, this long arm of latency, so central to our understanding of the effects of asbestos inhalation, does not appear to have received the same emphasis in relation to silica inhalation. Silica is a biologically active dust (Gulumian et al. 2006) and the biological processes which are initiated do not cease once silica exposure ceases. The fateful encounter between silica particles and the pulmonary macrophage produces a cascade of immunological and biochemical processes resulting in ongoing fibrosis (Gulumian et al. 2006). The macrophage also plays an important role in the first line of defence against mycobacterium tuberculosis. Through mechanisms that are still poorly understood, silica impairs this function.

There are a number of recent studies showing that South African gold miners with radiological silicosis are three to four times more likely to develop pulmonary tuberculosis than gold miners without silicosis (Hnzido et al. 1993; Cowie 1994; Corbett et al. 1999; Corbett et al. 2000). What has been less well appreciated is that this elevated risk of tuberculosis is present even without silicosis on the radiograph, i.e. due either to subradiological silicosis or to dust accumulation in the lung alone (Hnzido et al. 1993; te Water Naude et al. 2006). Furthermore, in the cohort of white miners described earlier, the elevated risk of tuberculosis accompanied miners after they had left the industry: and that most cases of tuberculosis in this cohort - 85% - occurred after the miners had ceased dust exposure (Hnzido et al. 1998). With regard to pulmonary tuberculosis then, silica retained in the lung and silicosis represent 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 (Republic of South Africa 1973).

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 in an industry with the resources of that of the gold mining industry and against a background of a vastly improved national registration system for all South Africans following the coming of democracy to South Africa in 1994. Even in neighbouring countries with more formidable logistical problems of tracking individuals, the existence of regional recruiting organizations with retained records and well-established networks and agents provides a real opportunity for tracking cohort members were the necessary resources provided.

HIV

This grim convergence of silica and tuberculosis among miners has been greatly worsened by the advent of HIV. HIV seroprevalences among gold miner workforces of the order of 27% have been reported (Corbett et al. 2004). The epidemiology of HIV infection is complex. 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 (Kark 1949; Hargrove 2007).
Tuberculosis is the face of HIV infection for many South Africans and HIV infection increases the risk of tuberculosis among gold miners five to six-fold compared to miners not infected with HIV (Corbett et al. 1999, Corbett et al. 2000). However, among gold miners who are both silicotic and HIV positive, the risk is increased 12 fold i.e. 1200 percent, compared to gold miners who have neither silicosis nor HIV infection (Corbett et al. 2000).

A completely unanswered question is the risk of lung cancer among these heavily silica exposed cohorts of black gold miners. “Cancer of the respiratory system” had become the most common site of cancer in the black gold mining population by the early 1990s (McGlashan 2003). However, only studies of white miners have specifically looked at the association between lung cancer and silica exposure and these have differed in their findings. In the latest study with the longest follow up, an increased risk of lung cancer was found in relation to both cumulative dust exposure and silicosis (Hnzido et al. 1999, Hnzido et al. 1997). There are some reasons why the risk of lung cancer among black miners might be lower than among white miners. Tobacco smoking rates have historically been much lower among black miners than white miners (Churchyard et al. 2004; Kleinschmidt 1997). Black miners have also had more competing causes of death at younger ages than white miners, notably trauma, tuberculosis and more recently AIDS.

Finally, even if there were an elevated risk of lung cancer in surviving cohorts, the access of black miners in rural areas to an accurate cancer diagnosis is limited. Radiological detection would be further complicated by radiological overlap of the features of silicosis, tuberculosis and carcinoma.
Whatever the situation regarding lung cancer, after 100 years of industrial development, for black gold miners in Southern Africa at the turn of the 21st century, “miners’ 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, moreover, should be grateful. Rather we should see it as the product of South Africa’s particular history, what Fassin in the context of competing explanations of AIDS, has called the *embodiment of inequality*, an attempt to explain “how history becomes physical reality and how biological facts become social facts…” (Fassin 2003).

**COMPENSATION**

General workers’ compensation legislation, which dates from the second decade of the 20th century in South Africa, 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 miners’ lung disease (Republic of South Africa 1973). The argument that employer indemnity from direct civil action by mineworkers did not carry over to this separate legislation was recently rejected in a test case in South African courts (Pringle 2008).
When I started examining miners in 1984, black miners were awarded a once off payment of R1200\(^1\) for tuberculosis and R2000 for silicosis (Myers et al. 1987). These were on average one tenth of the amounts awarded to white miners. Racial differentials were replaced in 1994 by waged based differentials such that miners now receive approximately R27 000 for silicosis alone and R70 000 for silicosis plus tuberculosis. These are not large amounts for middle aged miners often with large families. As Trapido (1998), quoting Chambers (1989), has written: “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…”

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 clinics such as at our hospital, it is not unusual for them to wait up to five years for their payment. Trapido et al. (Trapido et al. 1998), using the Transkei findings and an assumption of two million former mineworkers throughout Southern Africa in the late 1990s, extrapolated a notional figure of the order of R10 billion in unpaid statutory compensation liability, of which they estimated only R2 billion would realistically be claimed were a concerted effort to be mounted to track, examine and compensate this population for occupational lung disease. The bulk of such payments would flow to the poorest regions of the subcontinent. However, it has been observed that the system of compensation has developed and survived historically on the basis that black mineworkers would have de jure but not de facto access to its benefits (Trapido 1999).

\(^1\) One Euro = approx. 12 South African Rand (R) in August 2008.
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\(^2\). 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 (Morris 2005). In the same year the auditor-general referred to the financial controls of the Fund as a “shambles” (Morris 2005).

A QUESTION OF IDENTITY

Even should a former mineworker find his way into the system a further hurdle awaits – to prove his gold mining service. Some record of mine service has always been required, but until recent years the miner’s own recall was accepted together with a fingerprint record. In response to some incidents of fraud, the Compensation authority is now insisting on an original record of service. Our experience is that many older mineworkers are unable to produce such evidence. While those with more recent service and longer contracts may have retained their personnel card and have an extant record at the recruiting organization, in a number of cases service is long past and consisted of a variety of contracts at different mines. A surprising number of mineworkers have lost their documents in fires which frequently ravage informal housing 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.

\(^2\) Office of the Compensation Commissioner for Occupational Diseases, Johannesburg. Personal communication.
The history of South Africa reveals the double edged nature of registration and identification of its black citizens. After 1994 the hated “pass-book” that was central to apartheid’s workings was fully 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 gold miners, 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**

At the Commission of Enquiry into the Health and Safety of Miners in the 1994, the submission of the industry association (Chamber of Mines) paid little attention to occupational disease while emphasizing the socioeconomic benefits of mining (Chamber of Mines of South Africa 1994). In general, 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, for example, through the industry’s support of tertiary education, we are all beneficiaries. 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 South African mining industry has recently committed itself to eliminating any new cases of silicosis among the entrant cohorts of miners from 2008 (Chamber of Mines of South Africa 2007). Whether this is credible or feasible remains to be seen. However, it will clearly be insufficient to use only cases detected while in active employment as the target while not putting in place some means throughout the subcontinent for detecting cases after workers have left the industry.

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, 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.
REFERENCES


