A HANDBOOK ON

OCCUPATIONAL HEALTH PRACTICE IN THE SOUTH AFRICAN MINING INDUSTRY

CHAPTER 8

Author
Prof Neil White

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Chapter 8

Occupational Lung Diseases

This chapter will assist readers to understand and manage occupational lung disease risks in the South African mining industry. The most important diseases are described, with an emphasis on clinical features and on the association between exposure and the occurrence of disease. Where possible, this is based on research conducted in South Africa. The relevant legal framework in terms of the Mine Health and Safety Act No. 29 of 1996 and the Occupational Diseases in Mines and Works Act No. 78 of 1973 is reviewed, and, as applicable, the Compensation for Occupational Injuries and Diseases Act No. No. 130 of 1993. Medical surveillance is discussed in detail. The use of questionnaires, chest X-rays, spirometry, and other investigations in screening for occupational disease is detailed, as well as how to manage the resulting information.

This Chapter was originally written by Prof Neil White

Pulmonologist

8.1 Introduction

Occupational lung diseases (OLDs) are a major preventable cause of disability, premature retirement and death among people working in the South African mining industry (SAMI). The Mine Health and Safety Act No. 29 of 1996 (MHSA), as amended (1), requires employers to take measures to assess and reduce the risk of these diseases, and establish medical surveillance programmes for the diagnosis, treatment, and fitness to work. While the employer has a responsibility to report some diseases and is levied to fund the compensation, the employee, submits the application for compensation to the Medical Bureau for Occupational Diseases (MBOD) under the Occupational Diseases in Mines and Works Act No. 78 of 1973 (ODMWA), as amended (2), or to the Compensation Commissioner under the Compensation for Occupational Injuries and Diseases Act No. 130 of 1993 (COIDA) (3) .

All OLDs are preventable.

Compensation mechanisms, such as the various occupational disease Trusts in South Africa, serve as lagging indicators of systemic failure to prevent OLDs. These mechanisms, while essential for justice and restitution, reflect harm that has already occurred, often decades after exposure. The establishment of the Asbestos Relief Trust, Q(h)ubeka Trust, Kgalagadi Trust, Tshiamiso Trust, along with the pending coal mine class action, illustrates the long latency between hazardous exposure, disease onset, and eventual redress. This underscores a critical shift that must occur: from reliance on compensation after disease has manifested, to effective dust control, innovative engineering, rigorous risk assessments, and dust prevention at source. Prevention must take precedence, because by the time compensation is due, the damage is already done.

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- **Initiatives addressing OLDs in the SAMI:**

  - ***Mine Health and Safety Council Milestones***

    The industry milestones for addressing silicosis in the SAMI were set by the Mine Health and Safety Council (MHSC), initially in 2003, and revised in 2014. They aimed to achieve a 95% exposure measurement result below 0.05 mg/m³ for respirable crystalline silica (RCS) by December 2024 and ensure that no new cases of silicosis occur among previously unexposed individuals, using current diagnostic techniques. However, the 2024 Milestones were not achieved.

    The MHSC has reset the milestones, committing the industry to eliminating all OLDs by 2034, by reducing exposure levels and preventing new cases of silicosis and coal workers' pneumoconiosis (CWP). According to these milestones, by 2034:

    - 95% of exposure measurements for respirable crystalline silica should be below 0.03 mg/m³
    - 95% of exposure measurements for respirable coal dust should be below 1.25 mg/m³
    - 95% of exposure measurements for respirable platinum mine dust should be below 1.0 mg/m³
    - No new cases of silicosis, coal workers' pneumoconiosis, or platinum dust pneumoconiosis should occur among individuals entering the industry after 2024

- ***Masoyise Health Programme***

  Led by the Minerals Council South Africa (MCSA), the Masoyise Health Programme was launched in 2015 as Masoyise iTB, an initiative for the testing of mineworkers for tuberculosis (TB) and HIV. Masoyise is a multistakeholder initiative that includes, among others, the Department(s) of Health (DoH) and Minerals and Petroleum Resources (DMPR), the five mining unions, and the United Nations (UN) organisations, the International Labour Organization (ILO), World Health Organization (WHO), and Joint United Nations Programme on HIV/AIDS (UNAIDS).

  Key interventions through Masoyise iTB and Masoyise Health Programme are:
  - Data reporting and monitoring system
  - Regular reporting to leadership forums and the MCSA Board
  - Analysis of the Masoyise data by the National Institute for Occupational Health (NIOH)
  - Dissemination of best practices, and knowledge transfer, through seminars and workshops
  - Contact tracing initiatives for all cases of TB are prioritised
  - Information, education, and communication materials are developed for the industry
  - TB in Gold Mines Working Group (TBGWG), established in 2023, specifically to advise on how TB can be reduced in the sector

  Since 2019, the Masoyise Health Programme has adopted a comprehensive approach to health, and moved beyond HIV and TB to incorporate OLDs, non-communicable diseases (NCDs), and mental health. This is in line with an aging workforce, where diseases of lifestyle are becoming more prevalent (5).

8.1.1 Definition of occupational lung disease

The MHSA defines an occupational disease as any condition contemplated in either the ODMWA or the COIDA. **Table 8.1** illustrates the compensable diseases under the ODMWA and Schedule 3 of COIDA.

Table 8.1. ODMWA and COIDA occupational lung diseases

ODMWA Section 1(a, b, c, d, eA) • Silicosis in miners and workers exposed to RCS dust • Silico-tuberculosis in miners and workers • Coal workers' pneumoconiosis in coal miners • Obstructive airways disease in miners • Tuberculosis, compensation applicable only if cardio-pulmonary organs involved, in miners or those exposed to dust on the surface, and exposed to > 200 risk shifts • Progressive systemic sclerosis or scleroderma, in miners exposed to RCS dust
ODMWA Section 1(e) – any other disease of the cardio-pulmonary organs, which experts consider attributable to risk work. In practice, this section covers: • Lung cancer (from mining hazards such as RCS, asbestos fibres, diesel particulates, radon, etc.) • Occupational asthma in platinum salt workers ('platinosis')

• Asbestosis (interstitial lung disease) in asbestos miners • Malignant mesothelioma in asbestos miners • Pleural plaques in asbestos miners • Asbestos-related lung cancer in asbestos miners • Bronchiolitis obliterans due to nitrous fumes in mineworkers • Hard metal pneumoconiosis, usually in drill shop workers; stannosis in tin miners
ODMWA Section 1(f) – any other disease which the Minister, Director (of the MBOD), and three or more medical practitioners designated by the Minister has declared to be a compensable disease, attributable to the performance of risk work at a mine
COIDA Schedule 3 – of particular relevance to mining, but not included in the ODMWA list, are: • Asphyxiation due to carbon monoxide, hydrogen cyanide fumes or its derivatives, and hydrogen sulphide fumes • Occupational asthma due to nickel, cobalt, vanadium, chromium salts, soldering or welding fumes, isocyanates, formaldehyde, anhydrides, amines and diamines, and hardening agents, including epoxy resins

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COIDA: Compensation for Occupational Injuries and Diseases Act No. 130 of 1993, MBOD: Medical Bureau for Occupational Diseases, ODMWA: Occupational Diseases in Mines and Works Act No. 78 of 1973, RCS: respirable crystalline silica

8.1.2 Cost of occupational lung diseases in the South African mining industry

Occupational lung disease may result in illness, premature retirement because of disability, or death. There are significant costs involved, experienced by individuals in loss of income and medical or related expenses, and by mining companies, through the loss of experienced employees and the expense of recruiting and training new employees, direct medical expenses, and compensation levies. For the 2023/2024 financial reporting year, the MBOD, through the Compensation Commissioner for Occupational Diseases (CCOD), reported having spent R34.3 billion in payments towards compensable diseases (6).

8.2 The Occupational Lung Disease

In this section, the characteristics of the more commonly assessed mining-related occupational lung diseases are described. These conditions are grouped into pneumoconioses, chronic obstructive pulmonary disease (COPD), occupational cancers, work-related asthma (WRA), and inhalational injuries, including those due to nitrogen dioxide and fire.

8.2.1 Pneumoconiosis (mineral dust disease)

The mineral dust diseases share the common feature of being caused by dust particles that are small enough to reach the alveoli or gas exchanging part of the lung. Such dust particles are termed ‘respirable dust’.

The relationship between particle size (‘aerodynamic diameter’) and the ability of particles to penetrate the lungs is illustrated in **Figure 8.1**.

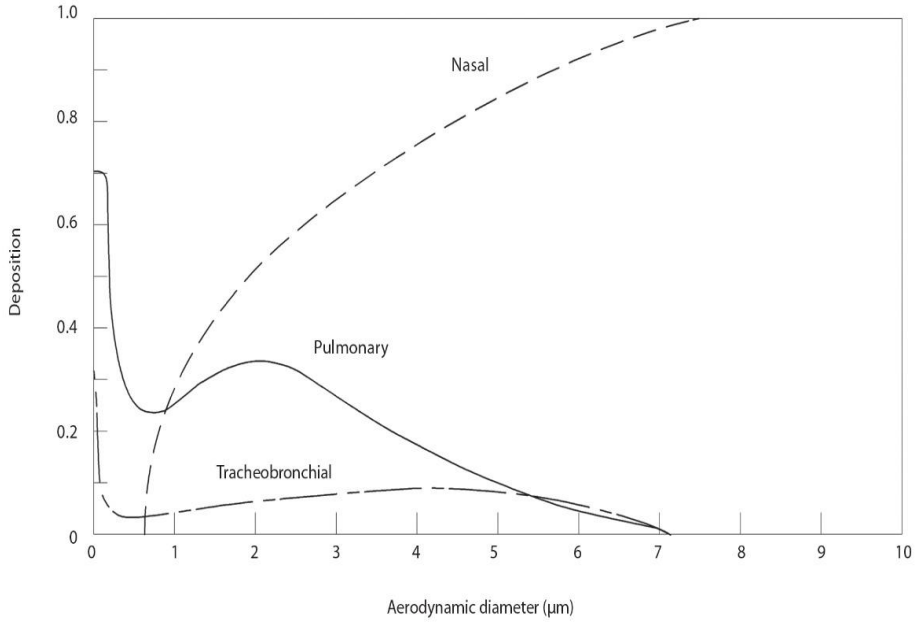

Figure 8.1. Deposition in the respiratory system as a function of particle size

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Source: Murray et al. (2001) (7)

The nasal passages are the first line of defense and will trap all particles with an aerodynamic diameter > 7 microns. The nose is less effective at preventing particles < 7 microns from entering the lungs, and below this limit, particles are defined as respirable. The lung's defenses can still remove particles reaching the airway walls in the tracheobronchial tree. However, approximately 30% of inhaled particles in the range 1–3 microns will be deposited in the lung tissue (**Figure 8.1**).

Dust particles in the respirable range that are deposited in the tiny airways, deep in the lung, will be engulfed by alveolar macrophages (defensive cells in the lung) and carried across the respiratory epithelium into the lung tissue itself. Crystalline silica (usually quartz) particles, especially when newly generated, are toxic to macrophages and result in cell death. A similar process occurs with coal and other mineral dusts. Death of the macrophage results in the release of the dust particles and attracts other macrophages that engulf the particles again. This recurring cycle of defensive cell death results in a low-grade inflammatory process in the lung. Gradually, the dust particles are sealed off in areas of fibrotic (scar) tissue that replaces normal lung tissue. This scar tissue is in the form of rounded opacities in the cases of silicosis and CWP, whereas in asbestosis or hard metal pneumoconiosis the scars are elongated or linear.

8.2.2 Silicosis

The dangers of silicosis from the inhalation of RCS dust produced by drilling, blasting, scraping, and other mining operations, have been recognised since the earliest days of gold mining operations in South Africa. The risk of silicosis occurs in all types of hard rock mining, tunnelling, quarrying, and crushing, where crystalline silica particles are liberated. Quartz is a pure form of silica crystal. Newly fractured quartz is the most toxic variety of crystalline silica.

Silica contained in an amorphous (non-crystalline) form appears to be less toxic than the crystalline variety, although amorphous silicas can cause silicosis. Amorphous silicas include diatomaceous earth, colloidal silica, fused silica, fumed silica, and thermally generated silica fume. Although there is a tendency to treat all amorphous silicas as a group, there is evidence that their toxicities do vary, depending on the particle size and content of crystalline silica.

8.2.2.1 Epidemiology of silicosis

The relationship between silica dust exposure and the occurrence of silicosis in South African gold mines was first investigated by Beadle in the 1960s, who believed that there was little evidence of any improvement in dust exposure levels in the years between 1938 and 1969 (9). This finding was still relevant later, when Hnizdo and Sluis-Cremer reported silicosis in a study on a cohort of white miners in the 1990s (10).

In 1995, the Leon Commission concluded that there had been little significant change in dust exposure in South African gold mines for fifty years (11). In 1999, the National Centre for Occupational Health published data based on 26 000 underground dust measurements, in 48 South African gold mines, from 1995 to 1997 (12). Only 8 (17%) of these mines had all of their estimated time-weighted average (TWA) measurements for respirable quartz below the DMPR's standard of 0.1 mg/m^3. Of the remainder, 21 mines (43%) had a high proportion of dust measurements in the range 0.1– 0.4 mg/m^3, whilst 19 (40%) had most of their measurements above 0.4 mg/m^3.

A further confirmation of the epidemiology of silicosis was through a cross-sectional study of black miners in the early 2000s by Churchyard et al., which showed a dose-response relationship of silica exposure and silicosis in the South African gold mining industry (13).

Risk assessment requires an understanding of cumulative exposure response curves. These curves illustrate what proportion of mineworkers exposed to a given cumulative dose of silica (the sum of all past exposures) will develop silicosis at some stage in their lives.

The exposure-response relationships from four studies of silicosis among miners, by Hnizdo and Sluis-Cremer, are shown in **Figure 8.2** (10). If the working population is exposed to 0.1 mg/m^3 of respirable crystalline silica for 20 years, then their cumulative exposure is 2 mg/m^3-years (0.1 times 20). It can be inferred from this study that less than 10% of such a population will ultimately develop radiological silicosis. If this group of workers spent the same time (20 years) working at 0.2 mg/m^3 (cumulative exposure of 4.0 mg/m^3-years), it can be expected that over 50% will ultimately develop silicosis.

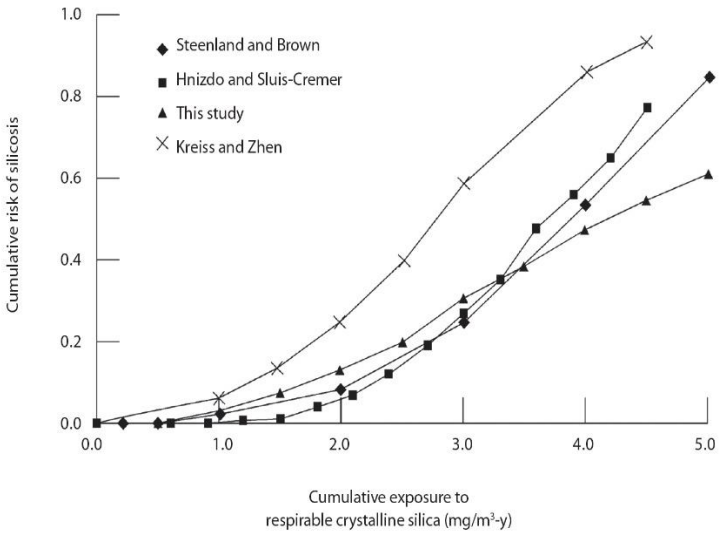

Figure 8.2. Cumulative risk of silicosis versus cumulative exposure to respirable crystalline silica

Source: Hnizdo and Sluis-Cremer (1993) (10)

Black workers comprise the vast majority of the mine labour force and do the jobs like drilling or ore removal in the dustiest areas of gold mines, such as stoping and development. Available information from recent studies indicates that black mineworkers have a working lifetime risk of between 220/1 000 and 360/1 000 of developing radiological silicosis from gold mining. These estimates, based on retired or retrenched workers, are much higher than estimates of the prevalence of silicosis in active black gold mineworkers.

Silicosis is a condition that develops slowly. As a group, active miners include members with a mix of various lengths of service. The workers with shorter lengths of service (< 10 years) are unlikely to have detectable silicosis, even if they are working in dust levels that are high enough to ultimately cause the condition. Silicosis continues to progress slowly with continued mining exposure. Cowie found that silicosis among Free State mineworkers, once established, progressed by approximately one ILO Classification subcategory (2/1 to 2/2) over a 4–5-year period. Even once exposure ceases, dust particles retained within the lung continue to be biologically active and the condition continues to develop.

The different estimates of the prevalence of silicosis in the active and retired workforce can probably all be reconciled on this basis. This natural history of silicosis has two major implications. First, the risk of silicosis can only be measured accurately by long-term follow up of cohorts of mineworkers, including ex- mineworkers. Second, estimates of occupational exposure limits (OELs) for silica dust exposure, as with other exposures, are usually based on studies of active workers, because this is the easiest group to study. Where studies of older and retired workers have been conducted, such as those included in **Figure 8.2**, they indicate that there is still a risk of pneumoconiosis from long working lifetime exposures below the DMPR OEL of 0.1 mg/m^3 for respirable quartz.

However, reduction of exposure to RCS dust remains an agenda, globally. In 2024, the Mine Safety and Health Administration (MSHA) in the USA issued a directive to reduce exposure to RCS dust to 0.05 mg/m^3 (14). In the same year, in South Africa, a new industry

milestone was adopted at an MHSC Summit to reduce exposure to RCS dust to 0.03 mg/m^3, respirable coal dust to 1.25 mg/m^3, and platinum mine dust to 1.0 mg/m^3 in the SAMI by 2029 (15), while the regulated OELs for these hazards remain unchanged in the MHSA Regulations.

Certification rates of compensable pneumoconiosis are also substantially lower than the actual rates of occurrence of pneumoconiosis (> 220/1 000) quoted for black workers above. There are several reasons for this. Firstly, epidemiological studies use sensitive radiological definitions of the presence of abnormalities consistent with pneumoconiosis. By contrast, certification of compensable pneumoconiosis, as detailed in Chapter 16 of this Handbook (Fitness to Work, Disability and Compensation), requires a more serious degree of abnormality consistent with at least a 10% impairment of cardio-respiratory function. The findings of two recent studies of retired black mineworkers confirm that compensation statistics substantially underestimate the occurrence of pneumoconiosis (16) (17). These studies both showed that there are substantial numbers of workers whose disease has either progressed since leaving the industry, or who were not referred to the MBOD for a benefit examination, as was their right, at the time of their exit from the industry.

Table 8.2 integrates DMPRs silicosis diagnoses from the database of mineworkers' pathology reports with the regulatory milestones in South Africa since 2001.

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Table 8.2. Timeline of silicosis diagnoses and regulatory milestones (2003-2025)

Yeat/Period	DMPR Findings (Silicosis Diagnoses)	Regulatory/Policy Milestones
2003-2007	DMPR records show high prevalence of silicosis in gold miners, especially long-service workers. Autopsy data confirm under-diagnosis during life.	MHSA medical surveillance strengthened. OEL for silica dust set at 0.1 mg/m³ (8h TWA).
2008	Continued diagnoses in current workers; latency effect evident	MHSC adopts milestone to reduce exposures, targeting 0.05 mg/m³
2010-2014	DMPR confirms persistent silicosis diagnoses, often with TB co-morbidity	2014 MHSC Summit milestone: 95% of exposures below 0.05 mg/m³ by Dec 2024
2015-2019	Diagnoses continue, though incidence begins to decline slightly. Compensation claims increase	Dust suppression programmes expanded; litigation and compensation schemes initiated for ex-miners
2020-2024	DMPR shows ongoing diagnoses in current workers, especially those with >15 years underground service.	MHSC "Elimination of Silicosis" target set for 2024. Medical surveillance triggered at >0.025 mg/m³ (50% of OEL).
2025	DMPR still records silicosis cases due to historical exposures. Disease burden persists despite improved controls.	Formal adoption of 0.05 mg/m³ OEL. Focus shifts to compensation, surveillance, and legacy disease management.

8.2.2.2 Prognosis of silicosis

Simple silicosis is a slowly progressive condition and has a latency of 10–15 years (18). Occupational medical practitioners (OMPs) typically diagnose silicosis radiographically. Unless simple silicosis is complicated by COPD, TB or progressive massive fibrosis (PMF), there is seldom any significant observable impairment of lung function.

8.2.2.2.1 Silicosis and loss of lung function

A progressive reduction of vital capacity of the lungs, with progression of X-ray changes, has been shown by a number of studies on silicosis cases (19) (20) (21).

A cross-sectional study of mineworkers by Ehrlich et al., the influence of silicosis on annual loss of forced expiratory volume in the first second (FEV1), with or without the effect of TB, as presented in **Figure 8.3**. The annual loss of FEV1 from silicosis without TB was less than 0 in ILO categories 1 and 2 but was visible in category 3. The FEV1 loss from silicosis with TB was more apparent, with the loss per milliliter increasing as the categories went higher.

Although there is evidence that simple silicosis without measurable lung function abnormality does not affect life expectancy in North America (22), there are no comparable data for South Africa. Complicated silicosis can be expected to reduce life expectancy. The most common complication of silicosis in South Africa is PTB, which has a significant adverse effect on lung functions and life expectancy, such as post-TB lung disease, which is underestimated, underdiagnosed and, therefore, not treated.

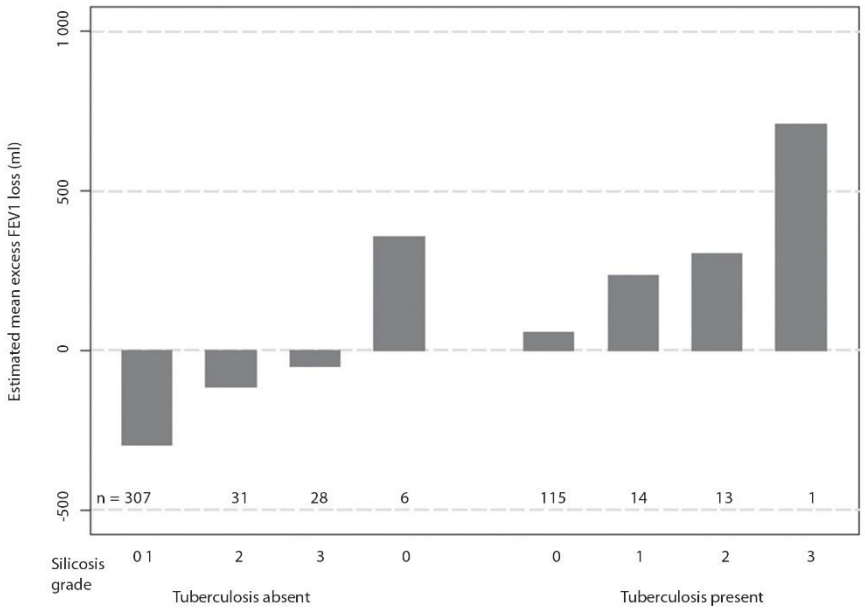

Figure 8.3. Excess FEV1 loss by major ILO categories of silicosis, with or without tuberculosis

Source: Ehrlich et al. (2011) (21)

8.2.3 Silico-tuberculosis

Both silica exposure and silicosis are risk factors for TB. Tuberculosis in a person with established silicosis is termed ‘silico-tuberculosis’. The risk of developing TB increases with duration of exposure to silica dust, even in the absence of silicosis (23). The presence of

radiological silicosis increases the risk of PTB approximately four-fold, with the risk rising as radiological silicosis becomes more severe (23). This increased risk of TB associated with silicosis is lifelong, continuing after silica exposure ceases.

The presence of silicosis in the lungs can modify the natural history of TB and may alter its radiological appearance. The interaction of TB and silicosis is very damaging to the lungs, unless the TB is diagnosed and treated early. With each episode of TB, lung function abnormality increases, as it does with each ILO category of profusion of silicosis (24).

Diagnosis of TB in the presence of silicosis should be according to the latest National TB Guidelines and revised guidelines for TB management in the SAMI (25). Silico-tuberculosis may be difficult to distinguish from PMF. Radiological suspicion of PTB in the presence of silicosis is greatly aided by comparisons with previous films, and should be prompted by:

- Rapid appearance of new infiltrates, especially in the upper third of the lung fields
- Coalescence of opacities, especially in the upper third of the lung fields
- Appearance of a cavity, especially with an irregularly shaped inner wall, within a conglomeration of opacities
- Appearance of any > 1 cm, non-retractile opacity limited by a fissure
- Development of pericardial or pleural effusion
- Development of segmental or lobar collapse, secondary to bronchial stenosis or occlusion

8.2.4 Non-silicosis silica dust-associated diseases

In addition to silicosis, exposure to RCS has been linked to several non-pulmonary diseases. These include autoimmune conditions such as systemic sclerosis, systemic lupus erythematosus (SLE), and rheumatoid arthritis. Chronic kidney disease has also been observed among silica-exposed workers, as well as an increased risk of cardiovascular disease. These associations underscore the systemic impact of silica exposure and the importance of comprehensive medical surveillance, beyond respiratory outcomes **(4)**.

8.2.5 Coal workers' pneumoconiosis

Coal workers' pneumoconiosis is also referred to as black lung disease. Coal mining in South Africa employed in excess of 90 000 people in 2022 (26), compared to 57 000 in 1998 (27). Most coal mining is either surface or underground, at comparatively shallow depths, not exceeding about 300 m. The output is predominantly bituminous coal, although a small amount of anthracite coal is also mined. The silica content of South African coal is generally low. Mpumalanga, Gauteng, and Free State coal has a quartz content of about 2%, whereas that of KwaZulu-Natal contains 3% quartz (28)

Coal miners are at risk of the following coal mine dust related diseases, according to Govender (2024) (4):

1. CWP
2. Rapidly progressive CWP
3. Complicated CWP
4. Rheumatoid CWP (Caplan's syndrome)
5. Mixed dust pneumoconiosis
6. Diffuse dust-related fibrosis (DDF)
7. Obstructive airways disease (emphysema, bronchitis)
8. Lung cancer

8.2.5.1 Clinical features of coal workers' pneumoconiosis

The clinical features of CWP are described in **Table 8.3**.

Table 8.3. Usual clinical features of coal workers' pneumoconiosis

CWP develops after 10–15 years of coal mine dust exposure.
In CWP, opacities < 1 mm in size become evident on the CXR, usually in the upper zones of the lung, but all zones can be affected with both rounded and irregular opacities.
According to the ILO International Classification of Radiographs of Pneumoconioses, radiological features resemble p,q,r,s,t,u opacities.
As the condition progresses, the opacities become more numerous.
CWP is frequently complicated by COPD (chronic bronchitis and emphysema).
CWP is usually slowly progressive over time.
The features of CWP resemble silicosis if there is appreciable silica content in the coal mine dust.
CWP differs from other pneumoconioses in that, even with large dust burdens, provided the silica content is low, fibrosis or scarring in the lungs is less severe.
With higher silica content of coal mine dust, high lung dust burden and/or infection with tuberculosis, CWP may evolve into a PMF (where smaller opacities coalesce into large opacities > 1 cm).

COPD: chronic obstructive pulmonary disease, CWP: coal workers' pneumoconiosis, ILO: International Labour Organization, PMF: progressive massive fibrosis

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8.2.5.2 Epidemiology of coal workers' pneumoconiosis

Some information relating to certification of CWP among coal miners is available from MBOD reports. Prior to 1980, certifications of pneumoconiosis (when expressed as a proxy rate of living cases certified per 1 000 miners currently employed) tended to be higher in coal mines than in gold mines (approximately 6 per 1 000 in 1980 versus 2 per 1 000 in gold mining). Coal workers' pneumoconiosis certification rates declined in the years 1980 to 1989, although in 1989 the rate was still over 4 per 1 000, a similar rate to the silicosis rate in gold mines. The MBOD Director's Report 2022/23 suggests a continuing decline in certifications of CWP, with less than 10% of the currently employed coal miners being diagnosed with CWP.

The cause of this decline is speculative in the absence of trend data on coal dust exposures in South Africa. Unlike gold mining, which remains a largely labour-intensive process, coal mining has become increasingly mechanised since the second half of the 20th century. On the one hand, mechanised mining greatly increases the potential for dust exposure in coal mining; on the other hand, mechanised mining means that fewer miners are exposed to these higher dust levels. However, mechanised mining also creates finer dust particles, which could potentially be more harmful to the fewer workers exposed.

It could be inferred that the MBOD certification rate for CWP may not be accurately reflected in some instances. This could be because cases are only submitted to the MBOD when they reach major category 2, which is the level at which compensation is awarded. This means that cases with less than major category 2 may not be submitted for compensation,

despite being diagnosed. The other reason could be that OMPs or radiologists not trained in ILO readings might miss cases that would otherwise qualify for compensation.

A study by White et al. documented radiological abnormalities among employees at several bituminous coal mines (29). A total of 188 ex-employees and 684 current employees, with more than five years' exposure at a mine, were included in the study. Of these ex- and current employees, 15.8% were thought to have CWP ILO subcategory of 1/0 or higher, including 7.6% of grade 1/1 or greater, and 1.1% (2 cases) grade 2/1 or greater. There was a positive association between presence of pneumoconiosis and length of service in a relatively young workforce (mean age 40.3 years). Findings of past or present TB were more common in those workers with CWP than those without.

Generally, the number of TB and silicosis cases in the SAMI comprise the largest representation for OLD, compared to CWP. In the 2021/2022 reporting period, the number of employees reported for CWP by the Mine Health and Safety Inspectorate (MHSI) was 2%, compared to 12% for silicosis and 39% for TB (26).

Exposure-response relationships for CWP and PMF have been well documented in epidemiological research in other coal mining regions of the world (30). The ability of coal dust to cause disease is generally highest for anthracite and higher-rank coals, such as those that have been formed under conditions of higher temperature and pressure and have higher carbon content. These dose response relationships may or may not be applicable to coal mining in South Africa, given that most South African coal is bituminous or low rank.

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Figure 8.4 shows a dose–response curve plotting the percentage of miners affected (on the y-axis) against exposure concentration to respirable coal mine dust in mg/m³ (on the x-axis).

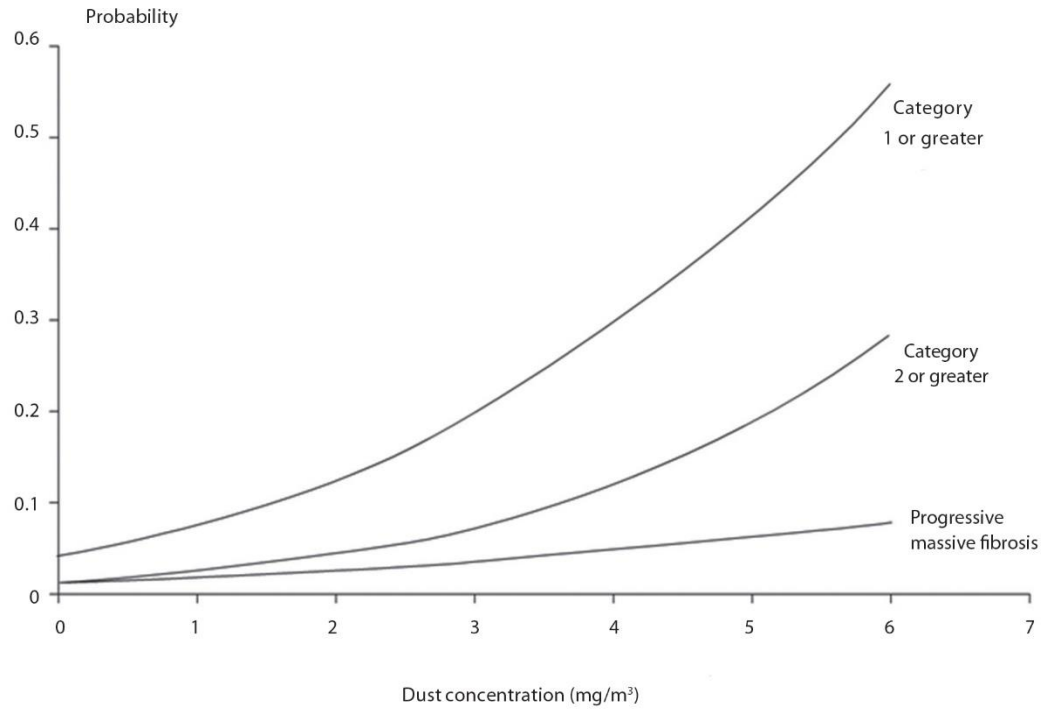

Figure 8.4. US coal miners' predicted risk of contracting irreversible pulmonary disease

Source: Reprinted from Rom (1992) **(31)**

However, the key assumption behind the figure, not explicitly shown on the graph itself, is that the epidemiological data from US coal miners were modeled to show disease risk after 40 years of full-shift occupational exposure at different dust concentrations (31).

In summary: at a dust concentration of 2 mg/m³, the estimated risk after 40 years is approximately:

- 12% for ILO category 1 or greater pneumoconiosis
- 2% for PMF

These curves are, thus, a function of both dose and time – specifically, the risk after 40 years of exposure at each concentration. This interpretation is critical to understanding the cumulative nature of risk and to ensuring appropriate occupational exposure limits are applied in risk management decisions. These estimates underscore the significance of maintaining exposures well below permissible limits and highlight the value of predictive modelling in setting occupational exposure standards and guiding medical surveillance.

8.2.6 Asbestos-related diseases

Asbestos is a family of crystalline hydrated silicates forming fibres, with a ratio of length to diameter, or aspect ratio, of more than 3:1. Asbestos fibres, thus, differ from silica or coal dust particles, which are spherical. This difference in the geometry of the particles is important for the causation of disease. The asbestos fibres that are retained in the lungs can be far larger and tend to be deposited in the lower zones of the lung. Macrophages are unable to engulf such large fibres, and many remain in the alveoli. Chemical reactions take place on the surface of the asbestos particles that are toxic to lung tissue, resulting in cell death and scar formation. Asbestos mining in South Africa commenced around the turn of the 19th century, and the country became the major global supplier of asbestos of the amphibole family, specifically, crocidolite and amosite. In addition, some chrysotile was mined. The asbestos minerals share the properties of heat resistance and tensile strength. They found many uses in heat insulation, construction, textiles, and engineering. Today, effective and acceptable substitutes are available for all these applications. McCulloch (32) traced the history of the mining of the three major commercial forms of asbestos in South Africa, namely chrysotile, crocidolite, and amosite, and detailed the epidemics of asbestos-related diseases that followed.

Although asbestos is no longer mined in South Africa and was officially banned in 2008, occupational health services are still likely to encounter asbestos-related diseases, in individuals who are recruited into mining operations with a history of asbestos mining or reside in an area where there has been environmental contamination with asbestos. The Asbestos Abatement Regulations of 2020 contain provisions appropriate to the prevention of asbestos-related diseases in this context (33).

8.2.6.1 Asbestos ban timeline in South Africa

1. *2001: Asbestos mining ban*
   Asbestos mining officially ended in 2001 in South Africa. This followed a decline in production and increasing evidence of the health risks associated with asbestos exposure (such as asbestosis, mesothelioma, and lung cancer).

2. *2008: Total ban on asbestos use*

In 2008, the Department of Labour enacted the Asbestos Regulations under the Occupational Health and Safety Act No. 85 of 1993 (OHSA) (33). These 2008 Regulations formally prohibited the import, sale, and use of asbestos and asbestos-containing materials (ACMs) in all sectors, including construction and manufacturing.

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3. *2020: Updated Regulations*
   Asbestos Abatement Regulations 2020: Replacing the 2001 and 2008 Regulations, the Asbestos Abatement Regulations (2020) came into effect on 10 November 2020. The Regulations focus on the identification, risk assessment, management plans, and safe removal of asbestos in existing structures and workplaces.

8.2.6.2 Clinical features of asbestos-related diseases

The general features of the asbestos-related diseases are summarised in **Table 8.4**. The asbestos-related diseases are well described in the medical literature, together with the ILO International Classification of Radiographs of Pneumoconioses (2022), which describes the radiological features for all asbestos-related diseases.

Table 8.4. General clinical features of the asbestos-related diseases

Disease	Features
Pleural plaques	Plaques are the most common manifestation of exposure to asbestos. They occur as hard, discrete, and often calcified, flat lesions on the surface lining of the inner chest wall. Plaques have been dubbed the 'visiting cards of asbestos', a term implying a minimal health effect, but a history of some exposure to asbestos. Plaques may appear where there has been only environmental exposure to asbestos. Plaques may signify an increased risk of mesothelioma, because they indicate significant asbestos exposure, but are not themselves premalignant.
Diffuse pleural thickening and pleural effusion	Asbestos-related diffuse pleural thickening is often the consequence of a pleural effusion that has become organised with fibrosis. When widespread, there may be significant functional impairment. Lung function testing is required to evaluate impairment.
Asbestosis	The term refers only to pneumoconiosis, such as fibrosis of the lung tissue. It varies in severity. When severe, the features include shortness of breath, cough, finger clubbing with cyanosis, basal crackles heard on auscultation of the chest, and ultimately right-sided heart failure and respiratory failure. Radiologically, the features are those of fine-to-coarse irregular and linear opacities that are initially basal. In severe cases, the whole lung may be involved. Lung function testing is required to evaluate impairment. According to the ILO International Classification of Radiographs of Pneumoconioses, radiological features resemble s,t,u opacities, predominantly in the lower lung zones.
Mesothelioma	Mesothelioma is a malignant cancer arising from the pleura or peritoneum (the membrane lining the organs and walls of the chest and abdominal cavities). Untreated, the disease has a poor prognosis, with less than 10% of sufferers surviving two years. Surgery, radiotherapy, or chemotherapy may reduce pain and discomfort.

Lung cancer	Lung cancer is a malignancy that arises in the lung tissue. Asbestos-related lung cancers usually occur in people who already have asbestosis. Asbestos exposure and tobacco smoking together multiply the risk of lung cancer. Thoracic surgery may be curative, but most lung cancers are inoperable at the time of diagnosis. Radiotherapy and chemotherapy may reduce pain and discomfort.

ILO: International Labour Organization

8.2.6.3 Epidemiology of asbestos-related diseases

At its peak in the 1970s, asbestos mining directly employed about 25 000 people. Employment in this sector has fallen to a negligible number, largely because of a collapse in demand for the mineral consequent to its replacement by safer materials.

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South Africa continues to pay the price of having mined this injurious mineral. As shown from MBOD reports, the numbers of workers certified as having asbestos-related diseases slowly climbed during the 1990s. In 1998/99, asbestosis accounted for 54.2% of all certifications for first-degree pneumoconiosis, and 19.6% of all second-degree certifications.

All types of asbestos are known to cause asbestosis, as well as pleural disorders. The consequences of asbestosis for life expectancy have not been evaluated fully in South Africa, but survival in Australian crocidolite mine and mill workers with asbestosis has been studied. Median survival, from claim for compensation, was 17 years in subjects with asbestosis ILO major category 1, 12 years in those with ILO major category 2, and three years in those with ILO major category 3 disease (34).

In 1960, the association between asbestos exposure and mesothelioma was established in a report of 33 asbestos workers with this rare disease, from the Northern Cape crocidolite fields (35). There is a dose response relationship between asbestos exposure and mesothelioma, such as the greater the exposure the greater the risk. However, the threshold is low – even modest exposures can cause this disease. A younger age of first exposure to asbestos is an additional risk factor, because there is a long lag between exposure and the appearance of the cancer. The potential of the various types of asbestos fibre to cause mesothelioma appears to be crocidolite > amosite > chrysotile.

An association between asbestos exposure, asbestosis, and lung cancer (as distinct from mesothelioma) has been recognised for many years. Evidence suggests that asbestos-related lung cancers occur more readily in lungs where there is existing fibrosis. However, this fibrosis may only be detectable at autopsy and may not be evident on the CXR. All types of asbestos have been associated with lung cancer.

Environmental contamination has resulted in many cases of fatal asbestos-related disease, primarily mesothelioma, an impact that continues in areas of the Northern Cape and Limpopo. Through a special programme, the South African Government, through the DMPR, has spent more than R40 million in the rehabilitation of abandoned and hazardous asbestos mines and works. The UK-based companies that mined in these areas disinvested in the 1970s, and have not made co-payments for this process, nor do they pay risk levies to the ODMWA Compensation Fund.

Historically, the cost of compensating such occupational diseases has had to be met from South African Government revenues. In 2000, the British House of Lords upheld the right of South African former employees of Cape Plc to sue the UK-based company for compensation. Cape Plc operated large crocidolite and amosite mines and mills in South Africa, mainly in the Northern Cape and Limpopo, for more than 70 years. More than 7 500 former employees were compensated through the resultant Trust funds, including the Asbestos Relief Trust and the Kgalagadi Relief Trust (36).

8.2.7 Chronic obstructive pulmonary disease

Chronic obstructive pulmonary disease is closely associated with cigarette smoking. However, COPD has been a compensable disease in the SAMI for nearly 40 years. Subsequent research in South Africa has confirmed the effect of underground exposures (such as dust, gases, and other particulates) on the risk of COPD (37).

Studies of black gold miners showed that the risk of chronic airflow limitation increases with duration of underground exposure, and is an effect that is independent of the presence of silicosis (21). The magnitude of this effect has been estimated as an additional decline of 8 ml per year in lung function. In the same study, the estimated direct effect of smoking 20 cigarettes a day was an excess decline of 7 ml per year. Differently expressed, the effect of gold mine air exposure on lung function is roughly equivalent to smoking twenty cigarettes a day.

Considerable attention has been paid elsewhere in the world to the relationship between dust exposure in the coal-mining environment and COPD. Dust exposure has been found to increase the prevalence of chronic bronchitis and chronic airflow limitation in coal miners, with similar effects observed in both smokers and non-smokers (9). Although measured lung function abnormalities are greater in smokers, the separate contributions of smoking and coal mine dust to COPD appear to be additive, rather than to be multiplicative of each other. An adverse effect of coal mine dust exposure on lung function has been observed even in the absence of radiographically detected CWP.

The usual clinical features of COPD are given in **Table 8.5**

Table 8.5. Usual clinical features of chronic obstructive pulmonary disease

COPD is the result of a combination of environmental exposures and genetic susceptibility. The term 'COPD' is used to describe three inter-related disorders that are often present together to a varying degree: • Chronic bronchitis, which is the consequence of airway inflammation and resultant mucus gland hyperplasia. Chronic bronchitis is defined by the presence of cough and sputum production on most days, for three or more months of the year, for two or more consecutive years. • Emphysema, which is the destruction of the gas exchanging tissue of the lung, consequent on a chronic inflammatory response to environmental exposures. • Chronic airflow limitation, which results from narrowing of the airways, as a consequence of both the inflammation occurring in chronic bronchitis and the loss of lung elastic recoil that occurs in emphysema. Lung function tests, bronchodilator responsiveness testing, and HRCT are required both to confirm the diagnosis of COPD and to assess its severity.

COPD: chronic obstructive pulmonary disease, HRCT: high-resolution computed tomography

In a study of coal miners, the average decline in FEV1, attributable to coal dust exposure, was 17 ml per mg/ml per year among active miners (23). This decline was of the same order as the adverse effect of smoking in this group.

Past MBOD reports indicate the long duration of exposure required for CWP. In 1989/90, the average period of risk work prior to certification for COPD in living miners was between 25 years (major category 1) and 30 years (major category 2) in gold mining, and somewhat shorter in coal mining.

8.2.8 Lung cancer

Lung cancer usually arises in the airway tissues, where exposure to noxious environmental agents is highest. Growth of the cancer may cause local effects such as obstruction of the airways, but death is usually caused by spread of the cancer to other sites in the body. Most lung cancers are diagnosed too late for curative surgery to be undertaken.

Tobacco smoking is the most common cause of lung cancer, globally, but a possible link has been established between lung cancer and mining exposures for the following, as documented by the International Agency for Research on Cancer (IARC) (38):

- Exposure to asbestos fibres
- Exposure to RCS dust
- Exposure to certain nickel compounds encountered in the smelting process
- Exposure to radon gas underground and diesel engine emissions

The occurrence of lung cancer in the SAMI has received less attention than it warrants. One of the barriers to proper assessment of risk is that lung cancer is uncommon among active miners, because most cases arise in the fifth or subsequent decades of life, after most miners have retired. Also, lung cancer is not listed as an ODMWA-compensable disease; as such, these cases were historically not submitted for compensation to the MBOD and, hence, not recorded. Recently, the practice has changed. All cases of lung cancer should be submitted to the MBOD for adjudication for compensation.

According to the NIOH's Pathaut Report 2022, 35 cases of primary lung cancer were identified among deceased miners who underwent autopsy in 2021. Most cases (60%) occurred in white male miners aged 60–79, with most having worked in the gold (60%) and asbestos sectors (23%). The lung cancer rate increased from approximately 28 per 1 000 autopsies (1975–2005) to about 75 per 1 000 by 2020, before declining to 64 per 1 000 in 2021.

The report may be accessed at: https://www.nioh.ac.za/wp-content/uploads/2025/04/PATHAUT-2022-FOR-FINAL-SUBMISSION.pdf

Due to limited smoking history data, it is unclear how much exposure to carcinogens like silica, radon, and diesel fumes alone, or in combination with smoking, contributed to these cases.

In addition to controlling exposures to these carcinogenic agents, an essential part of health promotion is to discourage smoking and assist with smoking cessation, and to discourage those who do not smoke to start smoking.

8.2.9 Scleroderma and progressive systemic sclerosis

Scleroderma, progressive systemic sclerosis (PSS), and rheumatoid arthritis are recognised as non- silicosis manifestations of RCS exposure (4) The usual clinical features of scleroderma and PSS are described in **Table 8.6** (39).

Table 8.6. Usual clinical features of scleroderma and progressive systemic sclerosis

Scleroderma: • Normal connective tissues are replaced by collagen, a dense fibrous tissue that affects normal functioning. • The process usually begins in the skin, which becomes hardened, depigmented, and contracts. • If this process remains localised to the skin, then it is called morphea – this is considered to be a different condition and is not compensable. Progressive systemic sclerosis: • Once internal organs are involved, the disease is considered to be systemic. • Over time, organs such as the lung, esophagus, and kidneys become affected by the disease process. This results in shortness of breath, difficulty in swallowing, and kidney failure. • Generally, the disease has a poor prognosis (< 40% 10-year survival). • D-penicillamine, colchicine and other agents may alleviate or slow progression of this disorder.

An association between PSS and exposure of Witwatersrand gold miners to dust containing a high fraction of silica was first suggested by Erasmus in 1957 (40). Sluis-Cremer later showed an association between PSS and lifetime silica exposure, rather than with silicosis. Cases appeared to have had higher intensity of exposure to silica during mining service, rather than longer duration of service.

It has been estimated that the annual incidence of PSS is 81.8 per million amongst black mineworkers in the group aged 33–57 years, compared with approximately 3.4 per million in a general population of similar age. A high incidence of TB was noted among workers suffering from PSS. It is likely that PSS is under-recognised and under-reported among miners.

8.2.10 Rheumatoid arthritis

Rheumatoid arthritis (RA), like scleroderma, is an autoimmune disease. When it occurs in conjunction with silicosis or CWP, it presents with distinctive radiological and histological features – a phenomenon known as Caplan's syndrome. In a case-control study of white gold miners attending the MBOD, Sluis-Cremer found that miners with RA had a higher likelihood of developing silicosis. Moreover, silicosis in these individuals tended to present with larger radiological opacities (classified as 'r'), and the disease progressed more rapidly compared to miners without RA.

8.2.11 Systematic lupus erythematosus

Systemic lupus erythematosus (SLE) is a chronic, systemic autoimmune disease, which is a long-term condition that can affect many parts of the body. In SLE, the body's immune system, which normally protects against foreign invaders, starts attacking its own tissues and organs, causing inflammation and damage. The clinical features include fatigue, fever, joint pain, and malar and discoid rash on the body, along with other skin, joint, and organ-related symptoms manifesting in the kidney, pulmonary, cardiovascular, neurological, and gastrointestinal systems.

Several studies have shown the association of exposure to RCS with SLE. A study by Parks et al. (41), recruited a total of 620 individuals from four university rheumatology

practices and 30 community-based rheumatologists in sixty contiguous countries. The aim of the study was to examine the association between occupational silica exposure and SLE in the south-eastern USA. The result showed more patients from the population with a history of medium-to-high level silica exposure from farming or trade, than the population with less exposure to silica across demographics. This study suggested that silica exposure may increase the risk of developing SLE.

A similar descriptive study to test the association between occupational silica exposure and SLE was conducted by Sanchez-Roman et al. (42), on a self-referred group of 50 workers from a factory producing scouring powder with a high silica content. The group underwent a prospective study, including clinical history and physical examination, an immunobiological study, human leukocyte antigen (HLA) typing, radiological and functional oesophageal and respiratory examination, ophthalmological examination, and isotopic testing of salivary glands. Of the result sample, 64% indicated symptoms of systemic illness. This study emphasised the risk of workers occupationally exposed to silica developing a multiple spectrum of clinical and serological autoimmune manifestations, especially SLE.

The pathology of SLE shows that it can affect multiple components of the immune system, including the complement system, T-suppressor cells, and cytokine production. A major player in the generation of autoantigens in SLE is the increase in apoptosis and/or the decrease in phagocytosis. This may result in the generation of autoantibodies, which may circulate for many years prior to the development of overt clinical SLE. The disease tends to have a relapsing and remitting course. The radiographical presentations manifest differently depending on the body system, which may include central nervous system, renal, musculoskeletal, gastrointestinal, and thoracic manifestations.

8.2.12 Miscellaneous mineral dust diseases

8.2.12.1 Stannosis and siderosis

Stannosis is the name given to the condition that results from the inhalation of tin, in the form of tin oxide (SnO_2) as fumes or dust. Tin oxide does not cause a fibrotic tissue reaction, and it is arguable whether stannosis should be called a pneumoconiosis. Iron oxides, which cause siderosis, appear to be similar in this respect. Since both tin and iron have high atomic weight and consequently absorb X-rays, chest radiographs depict these elements very clearly when they are deposited in lung tissue after inhalation.

Occurrence of stannosis has been described in South Africa. Stannosis in tin mines may be caused by the process of bagging, where a highly concentrated (70–80%) tin oxide is packaged prior to transport for smelting. This can be a very dusty occupation. In the smelter itself, there is exposure to tin oxide fumes, and most of the cases of stannosis seen at the MBOD have been from tin smelters. Silicaceous rock occurs where tin is mined, and silicosis may occur.

There are no reports on the respiratory health of iron ore miners in South Africa. Siderosis may, however, occur in jobs involving welding or metal cutting with high-temperature torches.

8.2.12.2 Pneumoconiosis – hard metals

Hard metal is an alloy of tungsten carbide and a matrix of cobalt, to which small amounts of titanium, nickel, chromium, and other metals may be added. The unique properties of this

compound are its extreme hardness (90–95% that of diamond). Because of this hardness and temperature resistance, hard metal is used in numerous mining applications, e.g. for the tips of drills. The cutting edges on drills must be reshaped at intervals by grinding. Workers who grind drill tips will be exposed to hard metal dust, unless this operation is carried out under water to prevent any respirable dust being produced.

Pneumoconiosis due to hard metal was first described in the 1940s. This pneumoconiosis takes the form of interstitial lung fibrosis. Cobalt has been identified as the most toxic component of the alloy. The prognosis of hard metal pneumoconiosis with pulmonary fibrosis is generally poor, since the disease is slowly progressive despite removal from exposure. A typical end stage with respiratory failure may occur within a few years. The clinical features of this end stage disease closely resemble cryptogenic fibrosing alveolitis. In 1987, Sluis-Cremer published the details of four cases (43). Since that time, there have been no further reports of the condition. The number of people at risk in the mining industry is unknown.

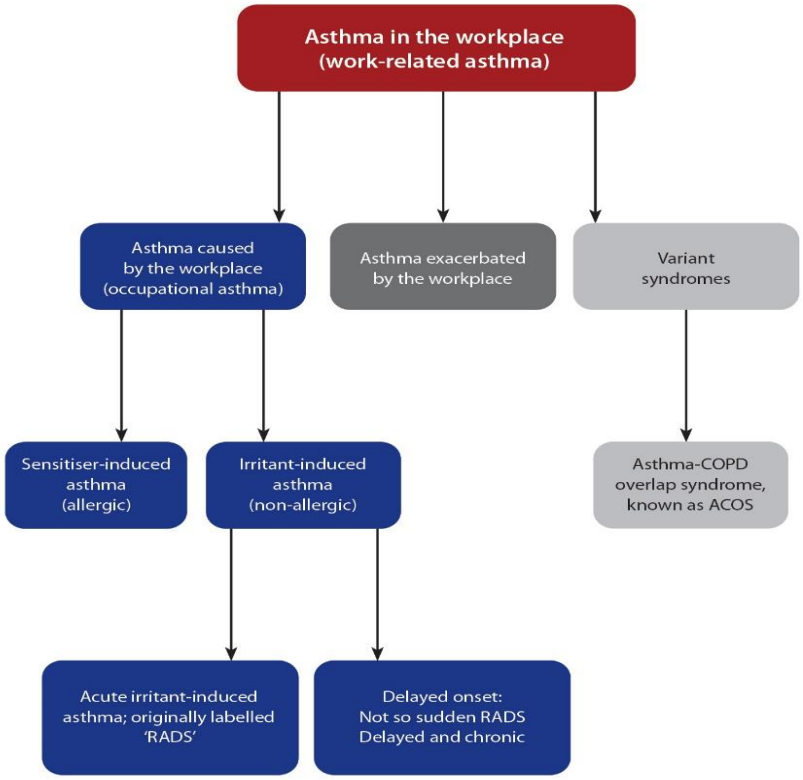
8.2.13 Work-related asthma

Asthma is a condition characterised by reversible narrowing of the airways of the lung, accompanied by airway inflammation. This inflammation is often caused by an allergic reaction, such as an adverse reaction of the body's immune system to a substance in the environment.

'Work-related asthma' is a broad term used to describe asthma triggered or worsened by exposures in the workplace. Work-related asthma is the most common occupational respiratory disorder in many countries, and early identification is crucial to prevent permanent airway damage.

The regulatory framework for workplace asthma is covered through circular instructions under the COIDA, for occupational asthma, work-exacerbated asthma, and irritant-induced asthma.

Work-related asthma includes three main types, as illustrated in **Figure 8.5** and described below:



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COPD: chronic obstructive pulmonary disease, RADs: reactive airway dysfunction syndrome

Figure 8.5. Phenotypic entities of asthma in the workplace

Source: Govender (2024) (4)

As illustrated in **Figure 8.5**, work-related asthma includes three main types:

1. Occupational asthma – asthma that is caused by specific substances in the workplace These substances include:
   - Sensitisers (e.g. flour, isocyanates, latex) that cause immune system reactions
   - Irritants (e.g. chlorine, ammonia) that inflame the airways

2. Work-aggravated (exacerbated) asthma – when a person already has asthma, and workplace exposures worsen their symptoms (e.g. dust, cold air, exertion)
   Key characteristics:
   - Symptoms include coughing, wheezing, chest tightness, and shortness of breath
   - Symptoms often improve when away from work (weekends, holidays)
   - Diagnosis involves history, lung function tests (LFTs) and, sometimes, workplace exposure assessments

3. Variant syndromes characterised by overlapping conditions of asthma and COPD, known as asthma -COPD overlap syndrome

8.2.13.1 Occupational asthma

Occupational asthma (OA) is asthma acquired as a result of sensitisation or reaction to a substance present in the workplace environment. A spirometry test can be used singly for evaluating lung function and confirming asthma, together with a bronchodilator responsiveness testing.

Mitigation of the risk of OA can be achieved by eliminating or substituting hazardous substances, implementing engineering controls like ventilation, and ensuring workers are trained and equipped with appropriate personal protective (PPE).

The usual clinical features of occupational asthma are described in **Table 8.7**.

Table 8.7. Usual clinical features of occupational asthma

Lung functioning is impacted by asthmatic narrowing of the bronchial airways, resulting in coughing, wheezing, or breathing difficulty.
This airway narrowing is episodic and can be reversed or prevented by the use of medication.
Sensitisation to a substance is a permanent condition and re-exposure to the causative substance, even in a relatively low dose, will predictably result in recurrence of asthma.
Exposure to the causative agent usually results in an asthmatic reaction within 30 minutes (the early response), but this reaction may even occur 18–24 hours following exposure (the late response).
Symptoms of allergic rhinitis (hay fever), conjunctivitis, and skin rashes may accompany occupational asthma, or may precede it.
Smokers and atopic individuals (people with a predisposition to allergic diseases) are generally at increased risk of developing occupational asthma.
Airways reversibility is demonstrated through the administration of a bronchodilator, and pre- and post-lung functions tests are conducted to determine the degree of reversibility.

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The smelting and processing of certain minerals can give rise to airborne exposures to specific metallic compounds, known to result in sensitisation and OA. This is certainly the case for platinum and nickel compounds, and probably also the case for vanadium.

8.2.13.2 Platinum salt sensitivity and platinum salt-related asthma

Platinum refining results in exposures to various salts of platinum that can cause sensitisation, allergic rhinitis (hay fever), and asthma. The most potent sensitising agent is ammonium hexachloroplatinate (ACP). Platinum is an important mineral mined in South Africa, and platinum sensitisation has been studied in some detail in the industry.

Respiratory manifestations: Chest tightness and wheezing are the most common symptoms that bring a platinum refinery worker to their doctor. Most sensitised workers have these symptoms immediately following exposure to an atmosphere with soluble platinum salts. They may also experience delayed symptoms, which occur when they are at home in the evening or asleep, commonly at about 2:00 am.

Dermatological tests: The existence of an allergy to platinum salts can be confirmed by skin prick tests in which a dilute solution of the offending salt results in a skin reaction. These tests often become positive a few months before symptoms develop but are occasionally negative even in the presence of work-related symptoms.

A study carried out at a local platinum refinery showed that most cases of OA developed early, usually in the first-or-second year following employment (44). Conversion to a positive skin prick test was most common in workers who were atopic, as defined by positive skin

prick tests to common aeroallergens such as pollens or house dust and was also more common in cigarette smokers.

Advances in platinum refining are capable of greatly reducing, but not altogether eliminating, exposure to platinum salts. Great care must be taken in dealing with chemical spills or other possible sources of accidental exposure. Even under optimal conditions, it has been shown that the incidence of sensitisation can range from 0.73–6.8 cases per 100 person-months worked, with symptomatic platinum salt sensitivity ranging from 0.59–2.4 cases per 100 person-months worked (45). The DMPR's OEL for soluble platinum salts is 0.002 mg/m^3. Even at exposures below this level, it is possible for susceptible individuals to become sensitised. Individuals who have become sensitised to platinum salts will experience symptoms at exposure levels below the OEL.

8.2.13.3 Reactive airways dysfunction syndrome

Irritant-induced asthma, also known as reactive airways dysfunction syndrome (RADS), can occur following single high-dose exposures to multiple irritant airborne exposures, such as sulphur dioxide, ammonia, and chlorine, as well as to the substances causing inhalation injuries as detailed in **Table 8.7**.

Reactive airways dysfunction syndrome should be suspected when a previously well person has persistent symptoms of airflow obstruction, three months after an accidental high dose exposure incident. Lung function tests should show airflow obstruction, or a histamine or methacholine challenge test should be positive, to make the diagnosis.

Reactive airways dysfunction syndrome is like OA, which is caused by sensitisation to a substance(s) in the workplace. It is a response to a specific workplace incident, like exposure to blasting fumes that causes direct injury to the airways, and the symptoms of airflow obstruction do not necessarily have any relationship to ongoing workplace exposure. However, like asthma, it may progress to a chronic condition.

8.2.13.4 Work-exacerbated asthma

Work-exacerbated asthma (WEA) is a condition that occurs when pre-existing asthma symptoms worsen due to workplace conditions or exposures, such as irritants, allergens, or environmental factors. The clinical features for WEA are like those of OA and RADS.

To mitigate risks of work-exacerbated asthma, employers should prioritise eliminating or reducing exposure to workplace triggers, through engineering controls, administrative controls, and PPE, while employees should actively participate in identifying triggers and seeking appropriate medical care.

8.2.13.5 Asthma-COPD overlap syndrome

Asthma-COPD overlap syndrome (ACOS) presents a more complex type of work-related asthma or respiratory disease, in that it is a condition where individuals experience symptoms and characteristics of both asthma and COPD. It presents with constrained lung functionality, whose clinical features include persistent airflow limitation, with symptoms of both asthma, such as wheezing, shortness of breath, and inflammation, and COPD, such as chronic cough, mucus production, and airflow obstruction.

Besides standard engineering, administrative, and PPE risk mitigation strategies, additional

measures can be implemented such as smoking cessation, vaccination, proper inhaler technique, and managing exacerbations with bronchodilators and corticosteroids to curb the risk of ACOS.

8.2.14 Inhalation injuries and asphyxia

Inhalation injuries in the mining industry may occur during underground fires with smoke inhalation, or from exposure to nitrous fumes from gelignite used in blasting operations. Many other agents can cause inhalation injuries, but only these two sources will be dealt with in any detail in this chapter. The usual clinical features of inhalation injuries are detailed in **Table 8.7** above.

Asphyxia among miners may result because of the development of a non-respirable atmosphere in the mine, secondary to an explosion or a fire. Carbon monoxide and other products of combustion may contribute to the tissue deprivation of oxygen that results in asphyxia. Self-contained, self-rescue apparatus and safety bays underground, where oxygen is available, are essential to prevent deaths from asphyxia. A second important cause of asphyxia occurs when miners trapped under rock falls experience chest compression. Relief of the compression using hydraulic jacks within the first 10 minutes may be lifesaving.

8.2.14.1 Nitrogen dioxide inhalation

Nitrogen dioxide is a heavy red/brown pungent gas produced by explosives. It is denser than air and, therefore, accumulates close to the ground in the workings following blasting. Adequate time is required for the gas to be cleared from the area after blasting is done.

This time requirement has set the pattern of work in the gold mines for more than half a century, in which all blasting is done at the end of the shift, and no one is allowed to enter the workings until the next morning when the dust has settled, and the nitrous fumes have dispersed. Very high concentrations of nitrogen dioxide may result if explosives burn during underground fires.

The DMPR OEL for nitrogen dioxide is 3 ppm. Because of the gas's low solubility, the conjunctivae and upper respiratory tract are not irritated at this, or higher levels, and 50 ppm can be breathed for some time before discomfort results. This lack of an irritative response at toxic levels of the gas can result in prolonged exposure and delayed presentation of the lung injury. Because of the gas's low solubility, damage is deep in the respiratory tract at the level of the small conducting airways and adjacent respiratory bronchioles.

Following high dose or prolonged exposure, the onset of shortness of breath and cough is delayed for about three hours but ranging from half an hour to 30 hours after exposure. Cases should be observed for at least 48 hours following exposure incidents, even if they are asymptomatic and with a normal CXR. Because of the marked variation in individual response to inhalation injury, follow-up LFTs should be performed after three months to ensure a return to normality, or to document the presence of a persistent airflow obstruction that could be a consequence of irritant-induced asthma or constrictive bronchiolitis.

8.2.14.2 Smoke inhalation injuries

Underground fires are an extremely serious occurrence and carry risks of asphyxia and smoke inhalation injuries. Fires underground occur usually in coal and gold mines, where

methane gas deposits initiate explosions that are then propagated as fires. Propagation of fires in coal mines is a special hazard, since the coal dust particles suspended by the original explosion are combustible. Underground fires burn any combustible material in the mine, making the resultant fire smoke a complex mixture of different ingredients, which vary not only from fire to fire, but also within the time course of a single fire.

Fires deplete atmospheric oxygen underground, creating a danger of asphyxia, but also increasing the rate of incomplete combustion with production of carbon monoxide and other toxic gases. Incomplete combustion of plastics and polyurethanes will emit both carbon monoxide and hydrogen cyanide and is, therefore, especially hazardous.

Inhalation injury causes 50–70% of the mortality of all burns patients (46). Simple fires usually result in pure thermal burns affecting only the upper respiratory tract, because of the low heat-carrying capacity of dry smoke. However, in patients with severe burns and upper airway injury, most also have involvement of the lower respiratory tract. Inhalation injury can occur in the absence of surface burns.

Patients assessed to be exposed to inhalation injury from blasts, explosions, fire, and/or smoke exposure should be observed in hospital for at least 24-48 hours, as symptoms are often delayed. Inspired air should be humidified and supplemental oxygen given. The possibility of carbon monoxide or cyanide poisoning should be considered, as applicable.

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Because of the marked variation in individual response, follow-up LFTs should be performed after three months to ensure a return to normality, or to document the presence of a persistent airflow obstruction that could be a consequence of irritant-induced asthma or constrictive bronchiolitis.

8.1.15 COVID-19 and its impact on occupational lung diseases in the South African mining industry

The South African Mining Industry (SAMI) experienced significant disruptions in the management of OLDs during the coronavirus disease-2019 (COVID-19) pandemic. Routine surveillance and screening for conditions like TB and silicosis were delayed or suspended, due to lockdowns and infection control protocols, including restricted access to spirometry and radiography services. Health system resources were diverted to manage COVID-19, overwhelming occupational health services and delaying diagnoses, referrals, and compensation claims. The overlapping respiratory symptoms of COVID-19 and OLDs created diagnostic confusion, potentially leading to misdiagnoses. Travel restrictions, mine closures, and reduced operations of certifying institutions further limited worker access to care, particularly for former mineworkers in remote areas. These delays may have worsened underlying lung conditions and increased vulnerability to severe COVID-19 outcomes. However, the crisis also spurred some positive developments, with a few mining companies adopting more integrated wellness strategies and exploring digital health tools to support worker care.

The COVID-19 experience in the SAMI highlights critical vulnerabilities in occupational health systems that must inform preparedness for future respiratory pandemics. It underscores the need for continuity plans that safeguard essential surveillance and care for OLDs, even during health emergencies. Integrated approaches – combining infectious disease control with ongoing OLD management – should become standard practice, ensuring that comorbidities are neither overlooked nor exacerbated. The use of digital

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health tools, mobile services, and decentralised care models proved promising, and should be scaled up to maintain service delivery during future disruptions.

Importantly, lessons learned from the COVID-19 pandemic reinforces the necessity for clear protocols to differentiate between pandemic-related illness and pre-existing occupational diseases, to prevent diagnostic delays and protect vulnerable worker populations.

8.3 Medical Surveillance

Medical surveillance is a programme of regular examinations, designed to detect disease for early treatment, referral, or appropriate placement of employees. The legal duties and rights of parties with respect to medical surveillance are summarised in **Appendix 8.1**.

A medical surveillance programme for OLD should be determined by the risk assessment and occupational risk exposure profile (OREP) for the individual worker.

The DMPR Guideline for the Compilation of a Mandatory Code of Practice for an Occupational Health Programme on Personal Exposure to Airborne Pollutants, 6 April 2018, guides mines in compiling and implementing a legally compliant Code of Practice (CoP) under section 9 of the MHSA. The purpose of the guideline is to ensure the effective management of health risks related to airborne pollutants, including respirable dust, silica, diesel particulate matter (DPM), and other airborne contaminants. It forms part of the broader effort to reduce OLDs, improve compliance, and protect mineworkers' health through structured, auditable programmes.

Specifically, the guideline aims to:

- Standardise occupational hygiene and medical surveillance programmes across the South African mining industry
- Ensure early identification and control of airborne exposure risks, in line with the MHSA
- Promote an integrated, risk-based approach linking exposure measurement (occupational hygiene) with health outcomes (medical surveillance)
- Provide a legal framework for enforcement by the DMPR, through inspections and audits

If the prevailing hazard for mineral dust exposure is less than 50% of the OEL, then a full programme of medical surveillance may not be required. Initial and exit medical examinations are appropriate to this level of risk.

Occupational exposure limits for, and levels at which, medical surveillance should be instituted for silica and coal mine dusts are illustrated in **Table 8.8**.

While the MHSA Regulations do not prescribe specific requirements for medical surveillance related to other airborne pollutants, such as DPM and radon, OMPs are encouraged to remain informed of the latest guidelines and international best practices, and to implement surveillance measures accordingly, where occupational risk justifies it.

Table 8.8 Occupational exposure limits for silica and coal mine dusts, as per MHSA Regulations

Airbourne pollutant	OEL	Medical surveillance required when exposures are > 50% of OEL
Crystalline silica (all forms)	0.05 mg/m^3 TWA (8 hours)	≥0.025 mg/m^3

OEL: occupational exposure limit, TWA: time-weighted average

8.3.1 Purpose of MHSA-aligned medical surveillance

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The MHSA gives clear guidance on the purpose of a medical surveillance programme, as summarised in **Table 8.9**. If there is a requirement for a medical surveillance programme, then the employer must draft a CoP for medical surveillance in accordance with the appropriate DMPR guidelines or Regulations, as required by section 9.2 of the MHSA.

The MHSA requires that there be clear communication between the OMP responsible for the medical surveillance programme and the health and safety committee(s) (HSC(s)) on the mine. A copy of the annual medical report, detailing OLD statistics for the most recent year, must be given to the HSC. This is the minimum requirement, but clearly the OMP can play an active role in health promotion, in conjunction with the HSC.

Table 8.9. Medical surveillance programme, in terms of the MHSA

A medical surveillance programme for employees should:

- Be appropriate to the health hazard
- Provide information that the employer can use in determining measures to:
  - Eliminate, control, or minimise the health risk and hazards
  - Prevent or detect and treat occupational diseases at an early stage
- Consist of an initial medical examination and other medical examinations, at appropriate intervals
- Establish a baseline against which subsequent changes in the health status of employees can be evaluated over time
- Be designed to identify medical conditions that may render employees temporarily or permanently unable to perform their occupations

If employees have previous exposure to mineral dust carrying a significant risk of disease, then they must also be included in a medical surveillance programme to monitor possible deterioration or development of disease.

- As far as reasonably practicable, ensure that:
  - Employees are fully informed of the health risk and hazards associated with their occupations and of the measures to eliminate, control, and minimise the health risk and hazards
  - The health status of employees does not place their health at increased risk in a particular working environment, nor place other employees or the public at increased risk

8.3.2 Employee education and medical surveillance

The training and education implied by the requirement that employees be informed about health risks needs to be linked to medical surveillance, wherever possible. Medical surveillance is itself an opportunity for education and is more likely to be acceptable to employees if they understand its purpose.

There are a variety of media that can be used for employee education. Such media are likely to have the most impact if they carry a simple message and are locally developed with professional assistance. Involving the HSC representatives in the design of the media and

programmes to disseminate the message will help to ensure relevance and impact. The various media include formal training sessions, peer education methods, videos, posters, and warning signs in the mine. As far as is practicable, all vernacular or mother-tongue languages in use by miners at a mine should be used.

Educational interventions need to be provided, in an appropriate form, for both newly engaged miners and more experienced miners. Such activities could be timed to coincide with medical surveillance (at three-year intervals) or more frequently, depending on the outcomes of the risk-based approach for assessing disease. Topics that are of particular relevance to lung disease include: explanations of relevant airborne pollutants, their health effects, and preventive measures in place at the mine; symptoms of PTB, to encourage appropriate self-presentation to health services; the hazards of smoking; and HIV/AIDS education.

The interaction of smoking and occupational risk factors for lung disease needs to be emphasised. The combined effect of occupational airborne pollutants and smoking may be more than the sum of the adverse effects of each risk factor alone. Investment in dust control, combined with smoking cessation programmes, contributes to the reduction of the risk of OLD.

8.3.3 Medical surveillance for pneumoconiosis and chronic obstructive pulmonary disease

The recommended current practice for tests and their frequency, in workers exposed to > 50% of the OEL of mineral dusts known to cause pneumoconiosis or COPD, is summarised in **Table 8.10**, as extracted from Chapter 11 of the Regulations of the MHSA. Each of the tests to be performed will be discussed separately, below.

Table 8.10 Medical surveillance for exposure to mineral dust known to cause pneumoconiosis

Examination	Tests to be performed
Initial – carried out on starting, or before undertaking, work	Respiratory questionnaire Cardiorespiratory examination CXR (large plate) Lung function test (spirometry)
Periodic – every three years	Cardiorespiratory examination CXR (large plate) Lung function test (spirometry)
Exit – when employment is terminated, for any reason	Cardiorespiratory examination CXR (large plate) (not necessary if done < 3 months before) Lung function test (spirometry) (not necessary if done < 1 year before)

CXR: chest X-ray

8.3.4 Data management and reporting responsibilities as part of medical surveillance

8.3.4.1 Record maintenance

The MHSA requires that employee records be kept for 40 years. Preservation of hard copy and software for such a long period requires special expertise. Within the company, there must be the allocation of responsibility to ensure that records are kept in the proper manner, to remain confidential, SECURE, legible, and accessible.

With the benefit of technological advances, globally, digital health records have become the norm at most large and small mines in the SAMI. It is advisable that occupational health and safety (OHS) service providers must have systems for archiving digital records and digitising old hard copies.

It is not a legal requirement that all records are kept as hard copy/paper, but there should always be at least partial (summary) paper records. If computerised records are kept, then it is essential that provision is made for all records to be backed up, and for transfer of old records onto new software systems when systems are changed, as compliant with the Protection of Personal Information Act No. 4 of 2013 (POPIA) and Promotion of Access to Information Act No.2 of 2000 (PAIA) legal frameworks.

The medical surveillance record system also must meet the responsibility towards the employees of contractors. A standard operating procedure should be in place on the mine with respect to employees of contractors, to identify those working in risk areas and avoid unnecessary repetition of medical surveillance of short-term employees.

8.3.4.2 Reporting

Section 13 (2) (b) of the MHSA explicitly states that a medical surveillance programme should be designed to provide information to assist with the prevention of occupational diseases. Medical surveillance data are consolidated and summarised into statistical data that are included in the annual medical report. The data relevant to OLD and required for such a report, as mandated by Regulations, are summarised in **Appendix 8.2**.

Occupational medical practitioners have additional statutory reporting duties in terms of the ODMWA. They are required to produce exit medical certificates and to report diagnosed occupational diseases to the DMPR's South African Mining Occupational Diseases Database (SAMODD) and the Masoyise Health Programme under the MCSA. As is evident from **Appendix 8.2**, there is overlap in the fulfillment of data reporting requirements.

Accurate digital health records are essential to fulfilling these reporting requirements. Relational databases, which store data in tabular format with rows and columns, offer a powerful solution and are available commercially. By using unique identifiers, such as name or company number, individual records from different years can be linked and tracked over time, allowing for comprehensive longitudinal health surveillance. Using a relational database, new data captured in each year's surveillance can be linked to the results in previous years, using these identifiers. By using the same or compatible software, it is possible to import or use other data, including employee identifiers, past employment records on human resources (HR) databases, past exposure records, and compensation histories.

Most occupational health database systems include built-in validation checks, by pre-programming acceptable value ranges for data entry. These checks help prevent the entry of incorrect or implausible values and can automatically flag significant deviations from baseline measurements – for example, a ≥ 10% decline in lung function – prompting timely clinical review by the OMP. In addition to ensuring data integrity, such systems can generate

customised outputs or reports in formats aligned with regulatory or operational requirements. A key purpose of digital health record systems, in occupational settings, is to facilitate the integration of exposure and health outcome data. In line with section 12(3) of the MHSA, medical surveillance record systems should be capable of linking occupational hygiene data with individual medical surveillance results. This integrated approach enables more accurate medium- and long-term risk assessments of exposures, such as dust and other airborne pollutants, enhancing the ability to monitor trends, detect early adverse effects, and implement effective preventive strategies. The occupational hygiene database should allocate every person working in the mine to a particular sample area, for purposes of assigning them to an activity area and, within that, to a homogeneous exposure group (HEG). Within each HEG, the hygienist will be sampling the exposure of a 5% proportion of exposed individuals, according to a schedule that aligns with the objectives.

Performance objectives for medical surveillance of OLDs are based on the requirements for the national TB programme (47), the guidance note for OMPs on a tuberculosis control programme (48), and the requirements of the Masoyise Health Programme (49).

Some performance objectives of the Masoyise Health Programme include:

- **Tuberculosis** – increase annual screening of TB to 100%, and reduce incident rates of TB at mining operations to levels below the national average, year on year

- **HIV/AIDS** – 95% of employees should be offered HIV counselling and testing (HCT) annually, with all eligible employees linked and retained to an antiretroviral therapy treatment (ART) programme, as per the National Strategic Plan (NSP); 100% of employees should be offered annual HIV testing, conducted at mining operations

- **Non-communicable diseases** – 100% of employees presenting during the medical surveillance programme should be screened for NCD metabolic risk factors for hypertension and diabetes

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The data captured in **Appendices 8.2** to **8.4** can be used to derive a variety of useful measures of the occurrence of OLD on a mine. Cases should always be expressed per number of employees. These measures can be used to track trends over time, or to compare different parts of a mine characterised by exposure information. Such comparative rates will better serve the functions of risk assessment and prevention.

8.3.4.3 Incidence: annual new cases of pneumoconiosis or new certifications

The minimum requirement for the annual medical report and SAMODD is the crude incidence (number of new cases per 1 000 employees) of new ODMWA certifications. A more specific indicator of occurrence would be new certifications per 1 000 employees in a sample area, or new certifications per 1 000 employees in an HEG. The most sensitive available indicator would be new cases of pneumoconiosis diagnosed in the radiological surveillance programme for exposed employees, as detailed below.

Sample areas or HEGs can be ranked according to the measures of exposure made there, and an approximate dose-response relationship can be derived by examining the incidence of certifications at each level. In large mines, where there may be more than one HSC, certifications could be reported according to the activity area for which the committee is responsible.

Pneumoconiosis, COPD, and occupational cancers are diseases with long latency periods. In the instance of silicosis, it may take 15 years of underground gold mining before the radiographic changes become evident. This means that occurrence rates of these diseases are not particularly useful in indicating areas where engineering control of dust is required. Current environmental measurements are, thus, a better indicator of risk. In diseases of short latency, such as OA, the occurrence of cases in a particular area, for example in a section of the platinum refinery, would draw immediate attention to areas in need of control.

8.3.4.4 Cumulative incidence: all certifications over a period of years

Cumulative incidence of previously certified cases of OLDs over a given period, such as five years, is also a potentially useful statistic on disease occurrence. Cumulative incidence is the proportion or percentage of individuals in a population who develop a specific disease or event over a defined period, normally calculated by dividing the number of new cases by the total number of individuals at risk at the beginning of the observation period. In terms of ILO classifications, cumulative incidence should be all cases diagnosed from major category 1 and above, and progress to certified cases that are compensable.

8.3.5 Respiratory questionnaires

Respiratory questionnaires should be conducted on all persons who will be exposed to significant risk from mineral dust. An example of an abbreviated questionnaire is attached as **Appendix 8.3**. For screening purposes, any positive response to a question about symptoms should be followed up by additional questions.

Questionnaires should be administered by people who have been specifically trained for the task. The multilingual environment of mines makes use of such questionnaires challenging, since some southern African languages do not have direct translations for terms such as wheezing. One approach is to prepare mother-tongue language translations for all languages in use at the mine.

This is often feasible, since there may be only three or four languages in common usage. Usual practice in preparing the questionnaire is to have the questions translated by a person proficient in the target language, followed by independent back translation of the questions into English by another person proficient in both languages. Alternatively, where the interviewers have multilingual abilities, it is acceptable for the questionnaire to be available only in English, and the questions translated appropriately as the interview is conducted.

There can be considerable savings of time and effort if questionnaire administration is computer assisted. Entry of responses to computer-prompted questions results in immediate data capture, as well as guiding the interviewer through the process. The interviewer can be prompted if no response is entered, or if a response cannot be correct.

8.3.6 Lung function testing

A variety of LFTs – including spirometry, body plethysmography, and DLco – are used to assess respiratory physiology and function. In South Africa, spirometry is a regulatory requirement, and its use in the SAMI must align with the latest American Thoracic Society/European Respiratory Society (ATS/ERS) Standards. Spirometry is widely used across the SAMI due to its affordability, accessibility, and portability. It is an objective, non-invasive, and sensitive tool for detecting early pulmonary changes, and can be reliably performed by trained personnel in diverse settings. However, interpretation of results involves several complexities.

Clinicians must compare an individual's spirometry results to predicted normal values based on age, sex, height, and ethnicity, using validated reference equations. The choice of reference Standard, biological variability, and the longitudinal trajectory of lung function must be considered carefully. There is increasing recognition of the limitations and uncertainties related to race-based reference values.

To ensure accuracy and equity, the SAMI should adopt the latest international guidance – particularly the 2022 ERS/ATS technical Standards on interpretive strategies for routine lung function tests – and implement the Global Lung Function Initiative (GLI) 2012 reference equations **(4)**. These are considered the most appropriate for South Africa's diverse, multi-ethnic workforce. Furthermore, global best practices increasingly support longitudinal, race-neutral monitoring of individuals' lung function over time, to enhance early detection and intervention for OLDs.

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Guidelines are available for OMPs to assist in the performance and interpretation of LFTs that accord with best current practice for routine lung function testing (50).

Spirometry consists of a forced expiratory maneuver for measurement of FEV1 and forced vital capacity (FVC). There are several spirometers commercially available for use in the occupational setting.

Quality control of spirometry is essential to producing meaningful results. There are many factors such as subject understanding, tester competence, and equipment calibration, which affect the reliability of the test (50).

Lung function impairment in individuals with pneumoconioses occurs years after radiological changes become obvious.

In COPD and asthma, LFTs serve both as screening and diagnostic tests that, particularly in COPD, give an indication of the degree of severity. However, LFTs alone are not sufficient to make a diagnosis – occupational and medical history and full physical examination are also essential, and other additional tests such as HRCT may be required, e.g. to make a diagnosis of emphysema.

A variety of commercial software programmes, sometimes made available with equipment, are available for converting information depicting the full flow-volume curve into a database for storage.

The minimum data that must be recorded are age, height, FEV1, FVC, and the ratio of FEV1/FVC expressed as a percentage.

Spirometry is the most common pulmonary test used in the SAMI to assess lung function, and to provide objective information used in the diagnosis of lung disease and monitoring of lung health. The indications include measuring the physiological effects of the disease, describing the disease progression that affects lung function, and monitoring people for adverse effects of exposure to injurious agents.

Spirometry technicians properly trained in the performance of LFTs are essential, if reliable results are to be obtained.

8.3.7 Chest radiology

All the pneumoconioses (silicosis, asbestosis, CWP, and others) are best detected by full-size postero-anterior (PA) (35 cm x 43 cm, or 35 cm x 35 cm) plain CXRs of acceptable technical quality. A large PA film alone is acceptable, although lateral films should be taken if pathology other than pneumoconiosis is suspected. Digital X-rays are preferable.

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Chest X-rays are usually able to detect signs of pneumoconiosis well in advance of the onset of symptoms and abnormal LFTs. The most important limitations in the use of CXR in medical surveillance are the technical quality of the X-rays and the competence of the ILO reader of the films. **Table 8.11** shows the features of a technically adequate CXR.

It is the radiographer's responsibility to produce a film quality acceptable for detection of pneumoconiosis. The film reader's first decision, when looking at the film, is to judge whether it is acceptable using the ILO criteria (8). A close working relationship between radiographer and ILO reader will improve quality and reduce the cost and inconvenience of having to repeat films.

The ILO International Classification of Radiographs of Pneumoconioses, 2022, established universally acceptable criteria to assess the quality of chest radiographs.

The ILO offers online courses for readership on their website:
https://www.itcilo.org/courses/online-workshop-ilo-international-classification-radiographs-pneumoconioses

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Appendix 8.4 shows a reading form for use in radiological OLD surveillance programmes. The primary reading form lists seven categories of abnormality that will be encountered by the reader of X-rays in a medical surveillance programme. The significance of each finding is detailed, together with the recommended plan of action. Information generated by collecting information from the use of such a form will provide important information for outcome indicators, such as annual incidence of new cases of pneumoconiosis diagnosed.

Table 8.11. Features of a technically adequate chest X-ray for detecting pneumoconiosis

Labelled with name, date, and location
Image quality should be of grade 1 or 2, free of technical imperfections or artifacts likely to impair classification, according to the ILO International Classification of Radiographs of Pneumoconioses, 2022
Well centred with apices and costophrenic angles visible
Full inspiration (such that the sixth rib intersects the shadow of the diaphragm near its dome (under inspired films can result in over-reading of pneumoconiotic opacities)
Scapulae clear of the inner aspect of the chest wall where they may obscure other lesions
Reasonable contrast, uniform across the film, and allowing easy distinction between blood vessels and air-filled parenchyma (film fogging or poor film-screen contact during developing can result in poor contrast)
Acceptable exposure of the film, such that the major pulmonary vessels can be clearly seen behind the heart
Overexposure (film too dark) results in underreading of pneumoconiotic opacities and, conversely, under-exposure (film too light) can result in over-reading
Avoidance of artefacts such as mottle, which can be misread as small opacities visible throughout the film, including the soft tissues
For screening, high kV films (125 kV) are recommended

8.3.7.1 Other tests – high-resolution computed tomography and diffusing capacity of the lungs for carbon monoxide

High-resolution computed tomography of the chest is not routinely used in the SAMI, due to cost, accessibility, and regulatory frameworks that promote CXR. However, HRCT can enhance diagnostic precision where CXR is inconclusive, unclear, or inconsistent with

clinical findings in suspected interstitial lung diseases such as asbestosis, or emphysema, or follow up TB sequalae.

High-resolution computed tomography can be used effectively to correlate findings with the diffusing lung capacity for carbon monoxide (DLco). The DLco is an enhanced LFT, which evaluates the presence of possible parenchymal lung disease when spirometry tests and/or lung volume determinations suggest a reduced vital capacity.

HRCT offers superior detail of lung tissue, allowing for better identification of subtle parenchymal abnormalities like opacities, fibrosis, and ground-glass opacities, which may be missed on CXRs.

8.3.7.2 International Labour Organization International Classification of Radiographs of Pneumoconioses

The chest X-ray (CXR) appearance of the pneumoconiosis is described using the ILO's International Classification of Radiographs of Pneumoconioses (8). This method ensures **global standardisation**, enabling clinicians and surveillance systems to track disease progression and identify early cases reliably.

The ILO classification system is a descriptive tool and not diagnostic of pneumoconiosis. It must be interpreted in conjunction with clinical findings and occupational history to confirm a definitive diagnosis of OLD.

As described in Govender in 2024 (4), to confirm an OLD, three main criteria are typically required:

1. Documented history of sufficient exposure to the hazardous agent, considering both intensity and duration (not duration alone)

2. Radiological evidence, consistent with OLD, on CXR or high-resolution computed tomography (HRCT), such as:
   - For silicosis: ILO profusion ≥ 1/0 with small, rounded opacities (p, q, r)
   - For asbestosis: bilateral irregular opacities (s, t, u)

   Imaging should be reviewed in series to assess disease progression.

3. Exclusion of alternative diagnoses, through clinical assessment and laboratory testing, e.g. ruling out TB in settings with high TB, high HIV, and silica exposure

This system is based on comparison of the individual's CXR with a set of standard radiographs. The abnormalities in the lung fields of a CXR are classified according to the type of abnormality seen (rounded or irregular) and the optical density or profusion of the abnormalities (as an index of severity or concentration). The profusion is graded from 0 (no abnormality) to 3 (high profusion), with a series of intermediate grades on a 12-point scale.

The ILO major categories 1, 2, and 3 correspond to increasing severity of lung dust disease, while the ILO 12 subcategories provide finer detail, capturing the gradation and diagnostic confidence level, as follows:

- ILO major category 0: 0/-; 0/0; 0/1

- ILO major category 1: 1/0; 1/1; 1/2
- ILO major category 2: 2/1; 2/2; 2/3
- ILO major category 3: 3/2; 3/3; 3/+

The ILO International Classification of Radiographs of Pneumoconioses (2022) requires the ILO reader o grade the radiograph quality and categorise parenchymal opacities, according to the shape and size of profusion, to describe the pneumoconiosis (8).

ILO readers can be:

- 'A' reader – medical practitioner who has undergone the ILO training in the ILO Classification System, but does not necessarily hold a certificate as a National Institute for Occupational Safety and Health (NIOSH) B reader, having written the NIOSH examinations
- 'B' reader – candidate who has undergone the NIOSH certification examination and renews it annually
- ILO expert reader – medical practitioner trained in the ILO reading, BUT not necessarily by the ILO

Table 8.12 illustrates the two types of small parenchymal opacities and their ILO descriptions.

Table 8.12 International Labour Organization classification of small parenchymal opacities (≤ 10 mm)

Size and shape of small opacities			
Rounded		**Irregular**	
Denoting letter	**Diameter (mm)**	**Denoting letter**	**Width (mm)**
p	≤ 1.5	s	≤ 1.5
q	> 1.5 up to about 3	t	> 1.5 up to about 3
r	> 3 up to about 10	u	> 3 up to about 10

Source: Adapted from Govender (2024) (4)

The large opacities are classified as those with the longest dimensions exceeding 10 mm, as shown in **Table 8.13**.

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Table 8.13. International Labour Organization classification of large opacities (> 10 mm)

ILO category	**Description**	**Interpretation**
A	One opacity with the longest dimension > 10 mm and ≤ 50 mm, or several opacities with the sum of their longest dimensions ≤ 50 mm	Progressive massive fibrosis, given the appropriate exposure and clinical features, and exclusion of (at least) post-TB lung disease and lung cancer
B	One opacity with the longest dimension > 50 mm, but not exceeding the equivalent area of the right upper zone, or several large opacities with the sum of their longest dimensions > 50 mm, but not exceeding the equivalent area of the right upper zone	
C	One opacity exceeding the equivalent area of the	

	right upper zone, or several large opacities exceeding the equivalent area of the right upper zone	

Source: Adapted from Govender (2024) (4)

8.4 Tuberculosis and Associated Diseases

8.4.1 Introduction

- **The burden of TB on the mines:** When workers are both HIV-positive and exposed to RCS, particularly in gold mining, the risk for developing TB is multiplicative, not merely additive. This combination of biological (HIV) and occupational (RCS and silicosis) risk factors creates a perfect storm for TB, underscoring the need for integrated prevention strategies across workplace and clinical settings.

  The mining industry in South Africa, particularly the gold mining sector, has had some of the highest rates of TB in the world (2 500–3 000 per 100 1000 population) at the peak of the HIV epidemic. These rates exceeded the WHO's epidemic threshold by a factor of ten, underscoring the mining sector's critical role as a hotspot for TB transmission in the region **(4)**. Today. The SAMI can claim great achievements in the fight against HIV and has enhanced dust control, driving the TB rates to the lowest seen in decades. However, the gold sector still has challenges of higher TB rates than the national prevalence rates. This was confirmed through a study by Zungu et al., (2025) which assessed the pulmonary TB trends in the SAMI between 2015-2022 (73). That study revealed that although gold mines had a higher prevalence, the occurrence of TB had reduced significantly because of a multi-faceted intervention.

  Data trends for TB and HIV between 2003 and 2025 are presented in **Table 8.14** based on the DMPR Annual Inspectorate reports in the south African mining industry (74)

Table 8.14 Timeline of TB and HIV data trends in the SAMI (2003-2025)

Year/Period	TB Data (DMPR Findings)	HIV Data (DMPR Findings)	Regulatory/Policy Milestones
2003-2007	TB incidence in miners 3–7x higher than general population; strong link with silica exposure. Autopsy data confirm TB as a leading cause of morbidity	HIV prevalence among mineworkers >25%, significantly above national average. Migrant labour patterns drive spread	MHSA requires medical surveillance; HIV/TB programmes begin to be integrated into workplace health services
2008-2010	Persistent TB diagnoses; co-infection with silicosis common.	HIV prevalence stabilises but remains high; ART rollout begins in mining regions	DMRE introduces compulsory reporting of TB/HIV programmes; OEL for silica dust reduced to 0.1 mg/m³
2011-2014	TB remains dominant occupational disease; DMPR confirms under-diagnosis during employment	HIV counselling and testing scaled up; workplace ART programmes expand	2014 MHSC Summit milestone: 95% of silica exposures below 0.05 mg/m³ by Dec 2024; TB/HIV Advisory Committee established
2015-2019	TB incidence begins	HIV prevalence	DMR 164 reporting tool

	gradual decline but remains 2–3x national average; DMPR shows ongoing burden in gold mines	plateaus; ART coverage improves, reducing AIDS-related mortality	introduced for standardised TB/HIV data; litigation and compensation schemes initiated
2020-2024	TB diagnoses continue, especially in miners with >15 years service; DMPR confirms latency effect	HIV prevalence stabilises but continues to drive TB co-infection; workplace testing remains critical	MHSC "Elimination of Silicosis" target by 2024; DMRE guidance notes mandate integrated TB/HIV programmes in mines
2025	DMPR still records TB cases due to historical exposures; TB remains a major occupational health burden	HIV prevalence remains above national average; co-infection with TB persists	Formal adoption of 0.05 mg/m³ OEL; focus shifts to compensation, surveillance, and legacy disease management

Tuberculosis has been in existence for millennia, dating back to ancient civilizations, globally. It reached epidemic proportions in the 18th and 19th centuries, and its understanding and treatment have evolved significantly, from sanatoriums to antibiotics and the development of the Bacillus Calmette-Guérin (BCG) vaccine.

Tuberculosis was recognised as an important health hazard shortly after gold mining started in South Africa in the 1890s.The high mortality from TB in black miners in the early 1900s led to an inquiry by the Tuberculosis Commission in 1912 (51). New recruits appeared to be very susceptible, often presenting with TB in the first few months of service. The high mortality from TB was likely to have been underestimated, since mineworkers who developed TB were repatriated. Overcrowding, poor diet, and poor working conditions were identified as important factors contributing to the high rate of TB amongst miners. The practice of repatriating miners with active TB was discontinued in the early 1980s.

Tuberculosis was classified as a compensable disease in 1916. Compensation awards were racially biased for more than 80 years, with compensation awards to white miners many times the amounts paid to black miners. The ODMWA was amended in 1994 to remove overt racial discrimination for compensation, and to introduce a wage-based system.

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Under the ODMWA, TB as a compensable disease means that cardio-respiratory TB is active in a person who has worked at least 200 shifts where silica dust or any other injurious dust was present. For former mineworkers exposed to silica dust, TB contracted within 12 months of leaving the mines is considered to be occupational TB.

TB is caused by the bacterium *Mycobacterium tuberculosis*, primarily spread through airborne droplets when someone with active TB coughs, sneezes, or speaks, and can infect the lungs or other parts of the body.

Three factors determine the likelihood of transmission of TB:

- Number of organisms expelled into the air
- Concentration of organisms in the air, determined by the volume of the space and its ventilation
- Length of time an exposed person breathes the contaminated air

People with suppressed immunity are more likely to develop active TB than those with normal immunity; 50–60% of HIV-positive people infected with TB will go on to develop active disease. The annual risk of TB in an HIV-positive person is 10%, compared to a lifetime risk of 10% in a healthy individual. Immuno- suppressive conditions such as silicosis, diabetes mellitus, and prolonged use of corticosteroids and other immunosuppressive drugs also increase the risk of

progression to active TB.

- **TB policy framework in the South African mining industry:** In the mining industry, implementation of the National Tuberculosis Management Guidelines (NTBMG) and the NSP for HIV, TB and STIs 2023–2028 (47) is facilitated through three documents issued by the DMPR:
    - Guidance Note for a Management and Control Programme for Tuberculosis in the South African Mining Industry (48)
    - Guidelines for Tuberculosis Preventive Therapy Among People Living with HIV (PLHIV) and Silicosis in South Africa (isoniazid preventative therapy (IPT) policy) (52)
    - Guidance Note for the Management of Latent Tuberculosis Infection in the South African Mining Industry (53)

  All three of these documents should be consulted when compiling the employer's TB control programme.

8.4.1.1 Pathogenesis of tuberculosis

After inhalation, the droplet nuclei are carried down the trachea-bronchial tree and deposited in a respiratory bronchiole or alveolus, where they are ingested by alveolar macrophages that produce a nonspecific response to the bacillus. Infection depends both on the bacterial virulence and the inherent microbicidal ability of the alveolar macrophage that ingests it. If the bacillus can survive initial defenses, then it can multiply within the alveolar macrophage.

The tubercle bacillus grows slowly, dividing approximately every 25–32 hours within the macrophage. The mycobacterium has no known endotoxins or exotoxins, so there is no immediate host response to the infection. The organisms grow for 2–12 weeks and reach 10^3–10^4 in number, which is sufficient to elicit a cellular immune response that can be detected by a reaction to the tuberculin skin test. The destruction of macrophages and release of tubercle bacilli products and chemokines stimulates an immune response.

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Before the development of cellular immunity, tubercle bacilli spread via the lymphatics to the hilar lymph nodes, and from there, through the bloodstream to more distant sites. Certain organs and tissues are notably resistant to multiplication of these bacilli. The bone marrow, liver, and spleen are almost always seeded with mycobacteria, but uncontrolled multiplication of the bacteria in these sites is unusual. Organisms deposited in the upper lung zones, kidneys, bones, and brain find environments that favour their growth. Numerous bacterial divisions may occur before specific cellular immunity develops, limiting multiplication.

8.4.1.2 Primary infection

Primary infection occurs on first exposure to tubercle bacilli. This usually occurs in childhood, so primary TB is often thought of as childhood TB. However, it can occur at any age in a previously unexposed individual. Inhaled droplet nuclei containing bacilli lodge in the terminal alveoli of the lungs, usually in the lower part of the upper lobe or upper part of the lower lobe. The bacilli are phagocytosed by the alveolar macrophages; mycobacterial products inhibit the bactericidal activities of the alveolar macrophages, allowing the bacilli to replicate within the macrophages. Other macrophages and monocytes are attracted to the area and produce an immune response. This inflammatory area is known as the Ghon focus.

Primary infection is usually asymptomatic and a positive tuberculin skin test 4-6 weeks after infection is the only evidence of infection, which could manifest in outcomes shown in **Table 8.15**. In a few cases, the immune response is not strong enough to prevent multiplication of bacilli,

which may spread from the lymphatics into the bloodstream and throughout the body, causing disease within a few months. Primary progressive TB in the lungs leads to enlargement of the primary focus with spread throughout the airways or lymphatics. Multiple areas of caseation and cavitation are found, producing a clinical picture similar to post-primary TB.

Table 8.15 Possible outcomes of primary infection

NO CLINICAL DISEASE Positive tuberculin skin test (Usual 'outcome' in 90% of cases)	**HYPERSENSITIVITY REACTIONS** Such as: • Erythema nodosum • Phlyctenular conjunctivitis • Dactylitis
PULMONARY AND PLEURAL COMPLICATIONS Such as: • Tuberculous pneumonia • Lobar collapse (bronchial compression) • Pleural effusion	**DISSEMINATED DISEASE** Such as: • Lymphadenopathy (usually cervical) Meningitis • Pericarditis • military disease

8.4.1.3 Post-primary tuberculosis/secondary tuberculosis

Post-primary TB is the pattern of disease that occurs in a previously sensitised host, after a latent period of months or years after primary infection. It may occur either by reactivation of latent bacilli or by re-infection.

Reactivation occurs when dormant bacilli, persisting in tissues for months or years after primary infection, start to multiply. This may be in response to a trigger such as weakening of the immune system by HIV infection. Re-infection occurs when a person who previously had a primary infection is exposed to an infectious contact. In a small number of cases, it occurs as a progression of primary infection. Following primary infection, rapid progression to intra-thoracic disease is more common in children than in adults.

Chest X-rays may show intra-thoracic lymphadenopathy and lung infiltrates. Post-primary TB usually affects the lungs but can involve any part of the body. The characteristic features of post-primary PTB include upper lobe involvement with cavitation and extensive lung destruction. Sputum smears are usually positive, and there is usually no intrathoracic lymphadenopathy.

Pulmonary tuberculosis is the infectious and most common form of TB disease, occurring in over 80% of cases. Laryngeal tuberculosis, although uncommon, is also very infectious. Tuberculosis may, however, affect any part of the body. Extra-pulmonary tuberculosis is a result of the spread of mycobacteria to the organs, most commonly pleura, lymph nodes, spine, joints, genito-urinary tract, nervous system, or abdomen.

8.4.1.4 Latent tuberculosis

Latent TB (LTB) is when the body system is infected with *Mycobacterium tuberculosis*, but the bacteria are inactive and do not present any symptoms or active TB disease. Latent TB is a significant concern in the SAMI with high prevalence rates among mineworkers, particularly those with silicosis and/or those living with HIV. This can lead to increased risk of developing active TB disease. The guideline on management of LTB in the SAMI (53) outlines the rationale for SAMI employees, mining communities, and peri-mining communities who are exposed to TB, and may require tuberculosis preventive treatment (TPT) once the disease has been excluded through testing and the exclusion criteria have

been considered.

8.4.1.5 HIV and impact on tuberculosis

The immunosuppression that results from HIV infection leads to a greatly increased risk of TB disease following TB infection, both by increasing the rate of reactivation disease, and by increasing susceptibility to rapidly progressive disease following newly acquired infection. The increased susceptibility to TB is apparent from an early stage of HIV infection and becomes more pronounced with increasing degrees of immunosuppression. Because of the high rate of reactivation disease, latent TB infection (indicated by a positive purified protein derivative (PPD) skin test) is a strong risk factor for TB among HIV-positive individuals.

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The combined effect of silicosis and HIV infection, in particular, is an example of interaction or synergistic risk, in which the two risks do not merely add to each other but *multiply* each other (54). This is illustrated in **Figure 8.6**. The increasing TB incidence associated with increasing grades of silicosis, in the absence of HIV infection, is represented by the blue bars. The TB incidence among HIV-positive individuals in the absence of silicosis is represented by the horizontal dotted line. The TB incidence among HIV-positive individuals with silicosis is indicated by the pink bars and is clearly more than the sum of the individual incidence rates for all ILO major categories of silicosis.

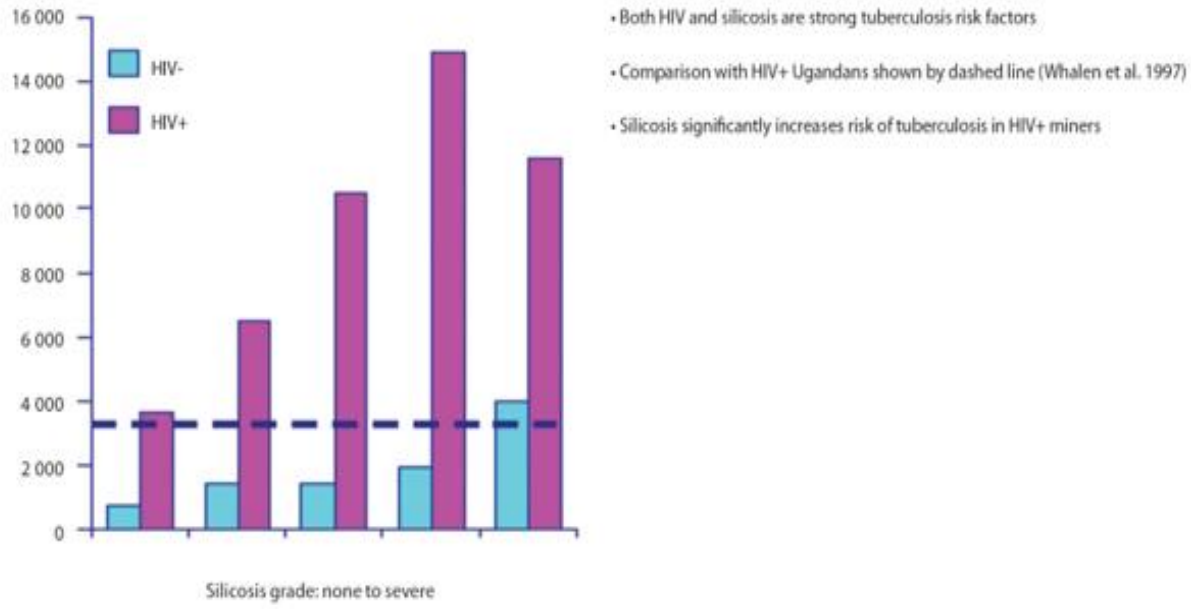


Figure 8.6. HIV, silicosis, and tuberculosis incidence (per 100 000 person years)

Source: Adapted from Whalen et al. (1997) (55)

Recent evidence underscores the multiplicative risk of developing PTB among individuals with concurrent HIV infection and occupational exposure to RCS. A landmark systematic review and meta-analysis by Ehrlich et al. confirmed that silicosis increases the risk of PTB fourfold (RR 4.01), while RCS exposure alone, controlling for silicosis, doubles the risk (RR 1.92) **(56)**.

In contrast, HIV infection confers an even greater risk: PLHIV are 20 to 30 times more likely to develop active TB compared to those without HIV, due to HIV-related immune suppression.

The implication of this interaction is that the incidence of HIV-related TB on the mines is strongly influenced by silicosis and RCS exposure, and control of both dust and HIV infection is required in order to reduce the incidence of TB.

To control the scourge of HIV, the SAMI rapidly rolled out ART for HIV-positive employees, starting around 2002, driven by the recognition of its economic benefits, including reduced healthcare costs and improved employee retention. Initially, there was resistance to rolling out HIV treatment in the mining sector, but the industry eventually understood the economic value of providing ART.

In the mid-to-late 1990s, mining companies began implementing ART programmes including anonymous HCT, often through their own clinics and hospitals, to provide care for employees, contractors, and their dependents. Additionally, guidance notes on the management and control of HIV in the SAMI provided information on referral forms, patient health records, counselling packages, and reporting requirements.

HIV ART provides significant benefits, including reduced absenteeism, improved worker retention, and cost savings for companies through lower healthcare expenses and benefit payments.

8.4.1.6 Post-tuberculosis lung disease

Post-tuberculosis lung disease (PTLD) refers to chronic respiratory abnormalities resulting, partly, from prior TB infection. It encompasses a spectrum of disorders affecting the airways, lung tissue, blood vessels, and pleura, and can be complicated by co-infections and haemoptysis. Individuals with PTLD often experience reduced lung function, lower exercise capacity, diminished quality of life, and a higher risk of recurrent TB and premature mortality, although long-term prognostic indicators remain unclear (4).

Studies of South African gold miners showed that TB is associated with accelerated loss of FEV1 and FVC (57). Although chronic TB adversely affects both FEV1 and FVC, the predominant effect appears to be obstructive. The implication is that any clinical evaluation of chronic TB, whether at the end of treatment or at any subsequent evaluation, such as for a medical benefit examination and compensation under the ODMWA, must include repeat chest radiography (HRCT, if clinically indicated) and LFTs.

Under the ODMWA, there are lump sum payments for first- or second-degree cardiorespiratory TB, where there is permanent impairment 12 months after TB treatment is completed, as per the COIDA Circular Instruction No.179 Regarding Compensation for Pulmonary Tuberculosis Associated with Silica Dust Exposure, published under GN 852 in GG 26585 of 16 July 2004.

8.4.1.7 COVID-19 pandemic and impact on tuberculosis

The severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) virus has potentiated underlying diseases, enhancing their progression at a much faster rate than usual due to the weakened immunity (58). The COVID-19 pandemic has put the TB gains at risk. Not only does SARS-CoV-2 pose an increased risk to people with TB, but it has severely disrupted services. An estimated 1.4 million fewer people received care for TB in 2024,

translating to 21% fewer than prior to the COVID-19 pandemic in 2019 (59).
In response, the SAMI implemented the CoP for managing the exposure to SARS-CoV-2 in the workplace (60), providing guidance to employers and employees in:

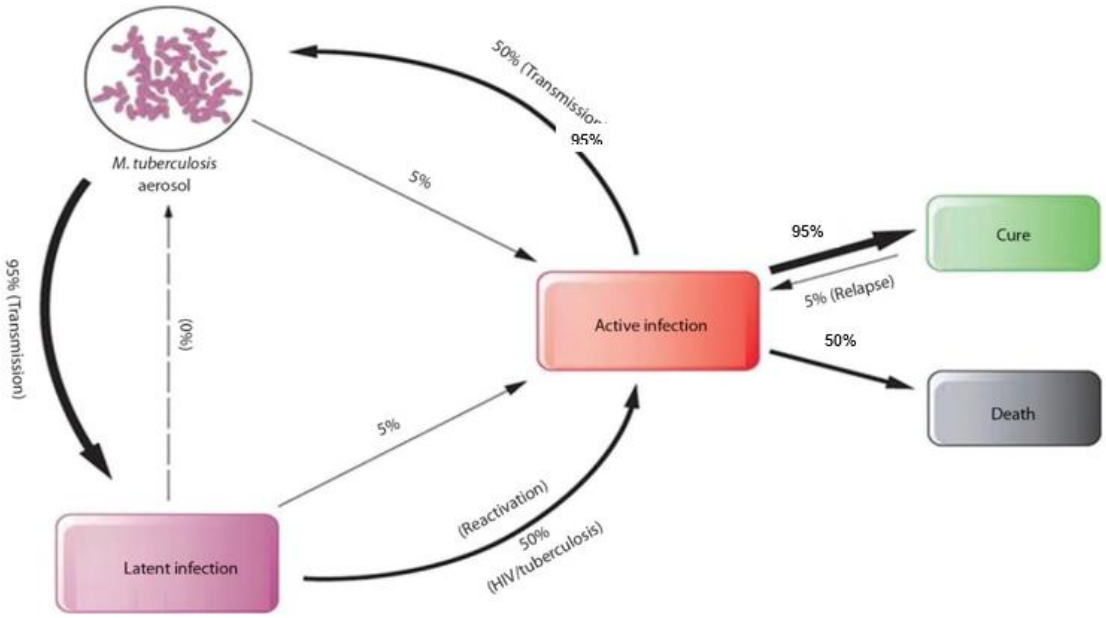
- Conducting or updating a risk assessment in terms of the Occupational Health and Safety Act No. 85 of 1993 (OHSA) and the Regulations for Hazardous Biological Agents in respect of SARS-CoV-2 exposure
- Developing a plan to limit infection and transmission, and mitigate the risks of serious illness or death based on that risk assessment
- Implementing the plan
- Managing absence from work due to infection, isolation, and adverse effects of vaccination
- Seeking to accommodate employees who refuse or fail to vaccinate against SARS-CoV-2

8.4.2 Epidemiology of tuberculosis

8.4.2.1 Tuberculosis infections, latency, and disease

Tuberculosis is caused by *Mycobacterium tuberculosis*, a widespread bacterial pathogen capable of prolonged survival within individuals in a state of latency (inactivity). This leads to an important distinction between latent TB infection, a state in which people are well, with normal medical investigations except for a positive skin test reaction to injected TB proteins (tuberculin test), and TB disease. Individuals with TB disease usually have symptoms such as a cough and weight loss, as well as CXR abnormalities and TB bacilli detectable at the site of tissue damage and disease.

The natural history of TB, as illustrated schematically in **Figure 8.7**, is the key to understanding the rationale of modern control strategies.



HIV: human immunodeficiency virus

Figure 8.7. Natural history of tuberculosis: infection and disease

Source: Adapted from McMillen (2015) **(51)**

Previous TB infection can be detected in most cases by a skin test reaction to injected purified protein derivatives. These skin tests are, currently, the only available method of diagnosing previous TB infection, which can also be positive as a response to previous BCG vaccination and infection with TB-like mycobacteria that are widespread in the environment (non-tuberculous mycobacteria).

The risk of latent TB is particularly high in individuals living in high TB transmission settings, as is the case with South African miners.

Latency complicates both the control and treatment of TB (see **Figure 8.7**). The risk of developing active TB is higher in latently infected individuals with HIV and has been identified as a major cause of recurrent TB. Latent TB bacilli are metabolically inactive and relatively insensitive to anti-tuberculous drugs, so that treatment of infection, as distinct from disease, requires prolonged therapy to avoid a high risk of developing active TB.

8.4.2.2 Relationship between tuberculosis infection and tuberculosis disease

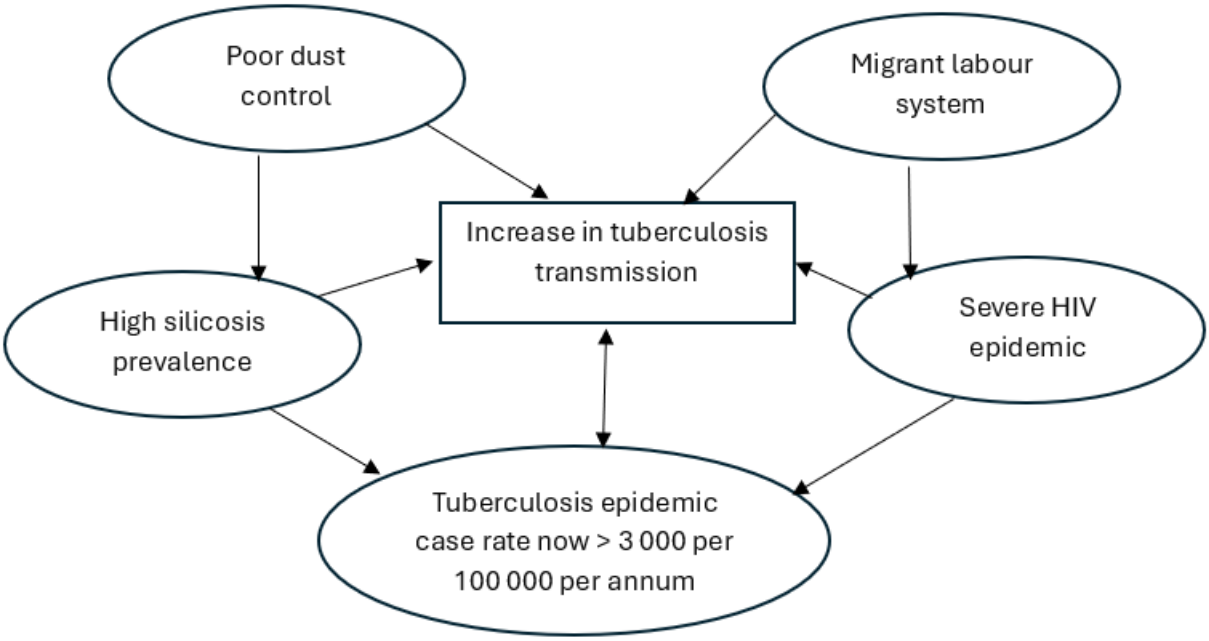
Studies among individuals who react to tuberculin skin testing and those with a recent 'conversion' to reactivity, indicate that most such otherwise fit individuals remain well and have no manifestation of disease. Of those who do go on to have disease, over 90% will do so within the first five years following infection (progressive primary disease). A small excess risk of reactivation of latent infection, however, persists for many years (see **Figure 8.7**).

Until recently, it was believed that once individuals had been infected with *M. tuberculosis*, they had lifetime protection against disease from reinfection. Any subsequent disease would, thus, be due to reactivation of that initial infecting strain. However, it has become apparent that, although previous infection may provide some protection against being re-infected, reinfection does occur. In the early 1990s, reinfection was identified as a cause of recurrent TB in HIV-positive patients, and more recently, as the major cause of recurrent TB in HIV-positive South African gold miners.

The distinction is important, because if most adult disease is in fact due to recently acquired infection, then reduction of TB transmission in a community will have a rapid and marked impact on the incidence of TB at all ages. A much slower response to improved control measures would be expected if most adult disease cases were the result of reactivation of latent TB.

8.4.2.3 Factors determining the incidence of tuberculosis in a community

The most important preventable factors in the mining industry influencing TB transmission, and progression to TB disease, are illustrated in **Figure 8.8**.



HIV: human immunodeficiency virus

Figure 8.8. Tuberculosis, silicosis, migrancy, and HIV

8.4.2.3.1 Exposure to tuberculosis

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The intensity of exposure to TB infection is by far the most important factor affecting TB incidence rates in a community. Estimated transmission rates are usually referred to as percentage, or 'annual risk of infection', representing the percentage age of the population that can be expected to be infected with TB during a one-year period.

8.4.2.3.2 Susceptibility to disease following infection

The other important factor determining TB incidence rates in a community is the percentage of infected individuals who progress to active TB. For otherwise healthy individuals, the lifetime risk of TB disease following TB infection has been estimated at 10%.

Several factors have been identified that greatly increase this risk, of which the most relevant for mining are age at infection (or reinfection), silica exposure and silicosis, and HIV infection. Alcohol is probably also an important risk factor but is poorly defined and will not be discussed further. The importance of smoking as a risk factor for TB among miners is unknown but is expected to be small in comparison to the other risk factors.

8.4.2.3.3 Age

Tuberculosis incidence in gold miners is strongly age-dependent, with a progressive increase in TB disease rates with increasing age. This is likely to be at least partly due to the effect of silica exposure, since age and length of service are closely related to one another and to cumulative silica exposure.

8.4.2.3.4 Silica dust, silicosis, and tuberculosis

Silicosis is dealt with in detail in this chapter. The strength of the relationship between

silicosis and TB was investigated in two cohort studies in gold miners in Welkom, Free State (54), as summarised in **Table 8.16**. Both Welkom studies provide estimates of the effect on TB of different grades of silicosis, based on individual CXR readings and follow up.

Table 8.16. Incidence of tuberculosis by silicosis grade among gold miners

Author/silicosis category	TB incidence rate ratio*	TB incidence rate (per 100 000 per annum)
Cowie 1994 – South Africa		
Older non-silicosis	1	1 000
ILO Category 1	2.2	2 200
ILO Category 2	2.9	2 900
ILO Category 3	6.3	6 600
Corbett 2000 – South Africa		
Miners with normal X-rays	*HIV-negative men only*	700
ILO Grade 0/1	1	1 400
ILO Grade 1/0 ILO Grade 1/1	1.8 1.8	1 400 1 900
ILO Grades 2/2 to 3/3	2.6 5.3	4 000

ILO: International Labour Organization

*Rate ratios quoted from Cowie and Corbett are unadjusted ILO International Classification of Radiographs of the Pneumoconioses

Another was a cross-sectional study of artisanal and small-scale miners in Zimbabwe by Moyo et al., in which a high burden of silicosis, TB, and HIV was found (61). It was shown that miners living with HIV were 1.25 times more likely to be diagnosed with silicosis, compared to those who were not. The risk of silicosis increased with both length of period as a miner and severity of exposure to silica dust. The risk of TB increased with increased years as a miner and extended exposure to RCS dust.

Overall, TB rates are approximately three times greater in silicosis than in non-silicosis. Workforce TB rates are about 3–4 times higher in silica-exposed gold miners than in non-silica exposed platinum miners (see Figure 8.12) (54)***.***

TB incidence in silica-exposed workers is high, even for those with normal chest radiographs without radiological silicosis. This is likely to be due to an increase in susceptibility to TB due to silica, even in the absence of radiographic silicosis. In addition, high silicosis prevalence will have a secondary impact of increasing TB prevalence and transmission, thus increasing TB incidence in the entire mining workforce.

8.4.3 Tuberculosis control in the mining industry: background and principles

Globally, the continued spread of TB has been driven by missed cases of TB, the HIV/AIDS pandemic, the emergence of drug-resistant TB strains, and weakened public health systems:

- **Missed TB** – Many of the populations most vulnerable to TB, including children, pregnant

women, PLHIV, and healthcare workers, are missed by health systems and do not get the care they need. Globally, more than 30% of all TB cases go undetected or unreported.

- **HIV/AIDS** – Worldwide, TB is the leading cause of death among people living with HIV/AIDS. People living with HIV have weakened immune systems, which put them at a much higher risk for developing TB disease.

- **Drug-resistance** – The emergence of multidrug-resistant TB (MDR-TB), including extensively drug-resistant TB (XDR-TB), is a major public health threat. Drug-resistant TB is very difficult, complicated, and expensive to treat.

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- **Weak heathcare systems** – Effective TB control efforts depend on political commitment and a strong public health infrastructure. Countries with limited resources often have weakened public health systems, making TB control efforts difficult to maintain.

South Africa faces a significant TB burden, but has made progress in TB control, with the Government committed to screening, accurate diagnosis, and treatment, while also focusing on preventing the spread of TB.

South Africa faces several challenges in the fight against TB. Notwithstanding the progress made towards controlling the disease, South Africa remains a country with a high burden of TB. This is exacerbated by co-infections, with a significant portion of TB cases and deaths occurring in PLHIV. Social determinants such as poverty, inequality, unemployment, undernutrition, and overcrowding play a major role in TB control. Drug-resistant TB poses another major challenge to control efforts.

Tuberculosis, HIV, and AIDS are the major causes of morbidity and mortality in South Africa. The country carries a triple burden of drug-susceptible TB (DS-TB), MDR-TB, and TB co-infected with HIV (TB/HIV), and is among the top 30 high-TB burden countries identified by the WHO ***(47)***. In 2019, the WHO estimated that 360 000 new people fell ill with TB in South Africa, of whom 58% were living with HIV. Tuberculosis is the leading cause of death in South Africa ***(47)***.

However, South Africa has implemented measures to control TB through the NSP, with the underpinning theme of *detect-treat-prevent-build (DTPB).* With measures such as contact tracing and community engagement, the focus on prevention and infection control has progressed TB control.

In the SAMI, TB control requires a multifaceted approach, including immediate treatment of active TB, latent TB treatment, HIV management, and improved living and working conditions to combat the disease's spread and impact on mineworkers. The industry faces a significant TB problem, with mineworkers experiencing a higher incidence of TB compared to the general population. The contributing factors include exposure to RCS, poor living and working conditions, high HIV prevalence, and migrant labour.

Some control strategies implemented in the SAMI include:

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- **Comprehensive TB programmes** – including testing, treatment and prevention measures

- **Benefit medical examinations** (BMEs) – encouraging mineworkers to participate in BMEs regularly, for early detection of TB, is important

- **Improved working and living conditions** – addressing the underlying factors that contribute to TB transmission, such as crowded living conditions and poor ventilation

- **Monitoring and evaluation** – monitoring and evaluating TB control activities, regularly, to assess the effectiveness of interventions and identify areas for improvement (done by collecting and analysing data on TB incidence and prevalence)

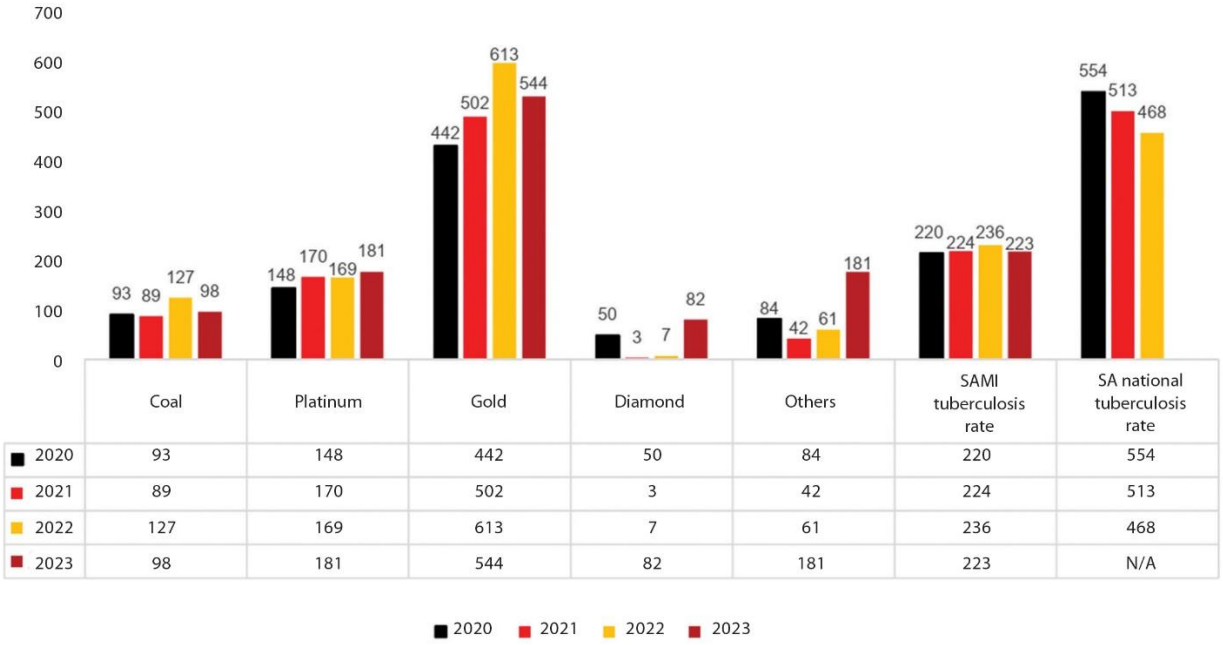
- **Social mobilisation** – raising awareness among mineworkers about TB, its prevention, and the importance of seeking timely treatment is vital

- **Guidance notes** – assisting in the management of TB in the SAMI, the MHSC issues guidance notes

The persistent prevalence of TB case rates may be the result of poorly run TB control programmes, or a failure of current TB control strategies to contain the TB epidemic. Apart from monitoring TB case rates, the effectiveness of a TB control programme can be determined by monitoring TB treatment outcomes, recurrence rates, and prevalence of drug resistance. Apart from a few recent publications on TB treatment outcomes among gold miners, there is a paucity of such information for the industry.

Tuberculosis case-fatality rates for miners are low in comparison to those reported for general populations. However, the TB case-fatality rate has increased significantly over the past decade, with the case-fatality rate for HIV-positive patients being at least 10-fold higher than HIV-negative TB patients (62).The prevalence of primary and acquired drug resistance has remained stable over the past two decades, suggesting that the TB control programmes have been effective in preventing drug resistance, even if failing to control TB incidence itself. There has been no association between drug resistance and HIV infection. While treatment outcomes seem promising, there are concerns regarding adherence to the treatment plan that warrant attention.

A comparison of TB prevalence across commodities over a period of four years, between 2020 and 2023, is shown in **Figure 8.9**. Gold mines had the highest TB prevalence compared to other commodities, and the SAMI incidents, overall, are lower than the national average.



	Coal	Platinum	Gold	Diamond	Others	SAMI tuberculosis rate	SA national tuberculosis rate
2020	93	148	442	50	84	220	554
2021	89	170	502	3	42	224	513
2022	127	169	613	7	61	236	468
2023	98	181	544	82	181	223	N/A

SA: South Africa, SAMI: South African mining industry

Figure 8.9 Tuberculosis incident rate per 100 000 population, by commodity

Source: Masoyise Health Programme (2023) (49)

8.4.3.1 Prompt diagnosis and treatment of infectious cases

Patients with PTB have the potential to infect others, until they are cured or die. Infectivity varies greatly: patients who are smear positive are considerably more infectious than those who are smear negative, and other factors, such as strain virulence, may also affect infectivity. Patients with chronic disease are an important source of transmission to other members of their community.

It has been estimated that, on average, 10–20 people are infected with TB by each smear positive reference (63)***.***

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Treatment of TB disease, caused by drug sensitive strains, results in a rapid decline in infectivity, but treatment has to be continued for prolonged periods to avoid a high risk of relapse to active disease and renewed infectivity.

8.4.3.2 Drug-resistant tuberculosis

Drug-resistant TB occurs when TB bacteria become resistant to at least one of the most effective TB medicines, leading to longer, more complex, and potentially less effective treatment. The categories of resistant TB (25) are:

- **Mono-resistant TB** – resistance to only one anti-TB drug, without resistance to other drugs

- **Poly-drug resistant TB** – resistance to more than one anti-TB drug, other than both isoniazid and rifampicin

- **Multidrug-resistant TB** – resistance to isoniazid and rifampicin, with or without resistance to other anti-TB drugs

- **Rifampicin-resistant TB** (RR-TB) – resistance to at least rifampicin, with or without resistance to other drugs (category includes MDR-TB, rifampicin mono-resistant TB, pre-XDR-TB, and XDR-TB)

- **Pre-extensively drug-resistant TB** (Pre-XDR-TB) – tuberculosis disease caused by strain of *M. tuberculosis* complex that is resistant to rifampicin (and may also be resistant to isoniazid), and that is also resistant to fluoroquinolones

- **Extensively drug-resistant TB** – tuberculosis disease caused by a strain of *M. tuberculosis* complex that is resistant to rifampicin (and may also be resistant to isoniazid), and that is also resistant to at least one fluoroquinolone (levofloxacin or moxifloxacin) and to at least one additional Group A drug, either bedaquiline or linezolid

- **Extensive (or advanced) pulmonary TB disease** – presence of bilateral cavitary disease or extensive parenchymal damage on chest radiography. In children aged below 15 years, severe disease defined by presence of cavities, parenchymal involvement of more or equal to one full lobe, miliary pattern, or mediastinal lymphadenopathy with airway compression on chest radiography

Drug resistance can be acquired during the course of TB if patients are treated with a single drug, or with combination therapy if the patient's strain already has resistance to all but one of the drugs in the combination. Acquired drug resistance can occur due to prescribing errors, patient non-adherence, or if the initial infecting strain is already resistant to some drugs.

Standard directly observed treatment, short course (DOTS) is unable to contain highly resistant strains of TB once they have become established at high prevalence, so that prevention of drug resistance is an essential component of DOTS and TB control programmes. Even in specialist units, treatment outcomes are poor for patients with TB caused by strains resistant to both rifampicin and isoniazid (the standard definition of multidrug-resistant TB [MDR TB]). Smear conversion from positive to negative may not be possible, and patients remain infectious to others for prolonged periods. Mortality and relapse rates are high even among HIV-negative patients, and mortality among HIV-positive patients during outbreaks of MDR-TB can be extremely high.

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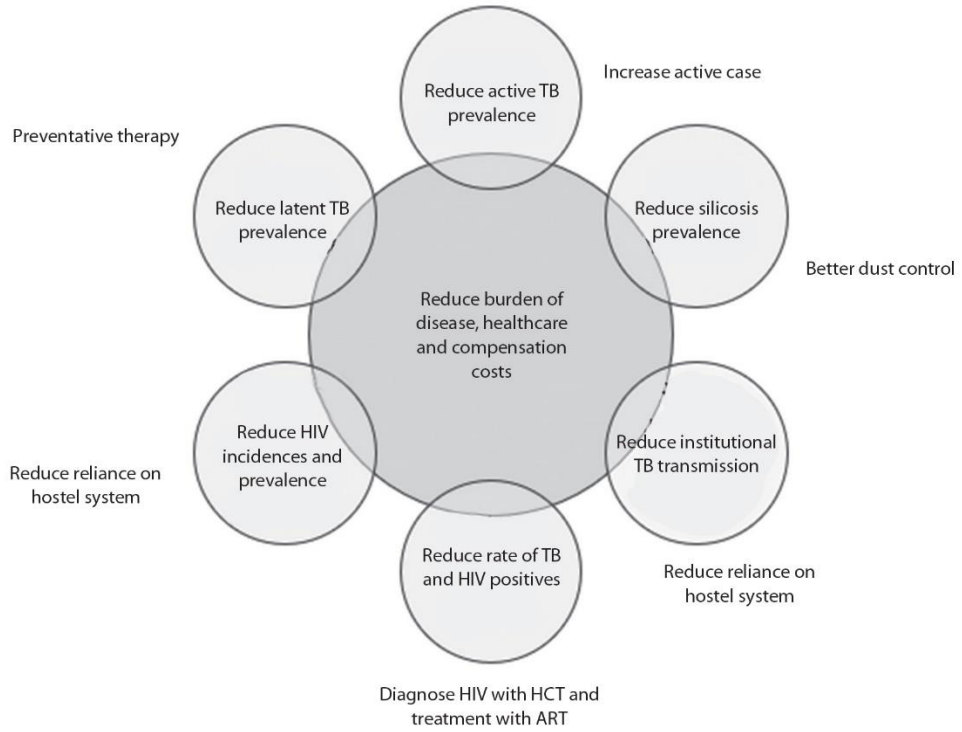
DOTS alone does not appear to be able to reduce, or even contain, TB incidence rates when drug resistance rates to rifampicin and isoniazid are already high, HIV/TB co-infection is prevalent, and in institutional settings where TB transmission rates are high. Under such circumstances, additional measures, such as increased case finding or use of preventive therapy, will be required to reduce TB incidence.

8.4.3.3 Comprehensive preventive strategy

The guidelines for a TB control programme in the mining industry are discussed in detail below. In addition to DOTS, there several other interventions that are needed to control TB

on the mines and are summarised in **Figure 8.10.** These include reduction in silicosis through dust control, reduction of transmission of TB by improvements in housing, preventive therapy against TB in high-risk miners, and reduction of the prevalence of HIV infection.



TB: tuberculosis, HIV: human immunodeficiency virus, HCT: HIV counselling and testing, ART: antiretroviral therapy

Figure 8.10. Potential interventions to reduce tuberculosis incidence

8.4.3.4 Tuberculosis control programme in the South African mining industry

The overall objectives of TB control programme at a mine should be to:

- Obtain over 90% treatment success rate for all TB cases
- Reduce defaulter rate to less than 5%
- Implement DOTS for 100% of TB cases on intensive and continuation treatment phases
- Report all TB cases to the national DoH and the DMPR, as per the national incident report
- Submit all reportable TB cases under the ODMWA and COIDA
- Promote access to HIV and AIDS prevention, treatment, care, and support services for all employees with TB, by:
  - Offering every TB patient with provider-initiated HIV counselling and testing
  - Putting all TB and HIV co-infected patients on ARV
  - Screening all HIV-positive patients for TB with increased frequency
- Reduce morbidity and mortality attributable to TB
- Prevent development of resistance to anti-TB drugs
- Ensure accurate and ongoing evaluation of programme performance

Specific objectives should include:

- Bacteriological evaluation of all PTB cases

- Directly observed therapy for all TB cases
- Cure of at least 85% of new smear-positive cases, using standard short course chemotherapy

8.4.3.5 Tuberculosis leading practices in the South African mining industry

Through its centre of excellence, the MHSC issued a compendium of the TB leading practices that should be implemented in the SAMI, including (64):

- **Preventative practices** – addressing the social determinants of health through conducive housing and living conditions, managing circular migration of workers, and mobilising and involving communities; practising infection control through prevention of nosocomial spread; and preventing progression of disease through chemoprophylaxis

- **Health promotion practices** – seeking care by increasing TB awareness through enhanced advocacy of health literacy and education, planning, managing and monitoring peer educator programmes, and passive case finding; positively influencing the social life of miners through promotion of healthy habits and behaviours, reducing TB stigma and fostering treatment; and seeking out those at higher risk through screening TB contacts and intensifying active case finding

- **Curative practices** – obtaining a timely accurate diagnosis; practising TB management, including improved referrals and addressing potential pre-disposing health conditions by treating HIV-TB co-morbidities; detecting and managing NCDs; preventing silicosis; and managing patient mobility to prevent MDR-TB

- **Rehabilitative practices** – tracking treatment outcomes and ensuring full recovery from TB

- **Workplace policy** – conducting pre-employment screening; addressing TB management during labour unrest and strikes; and incorporating TB management and risk awareness in policies for families and contractors

- **Monitoring and evaluation** – documenting outcomes accurately to learn from the process; monitoring and evaluating TB control effectiveness; and enhancing performance reviews

8.4.3.6 The company's tuberculosis policy and procedures

Management commitment is key to effective TB control in the workplace. Policies and procedures commensurate with the TB risk in the company, latest DMPR TB guiding documents, National TB Guidelines and the MHSC Tripartite agreed milestones for TB, should be in place.

The TB programme and its performance should be a standing item on the agenda of the HSC, and its statistics should be discussed regularly at management meetings.

8.4.3.7 Structure and staffing

A TB programme manager should be made responsible for the implementation, monitoring, and evaluation of the TB control programme. Ideally, the programme manager should be an OMP with competence in HIV/TB control. The level of staffing of the various services, such as medical, nursing, pharmacy, radiology, and laboratory, should be appropriate to the TB case load, to be able to meet the objectives of the TB control programme.

The programme manager should establish a TB control programme committee, responsible for programme evaluation and making the results available to all stakeholders, such as mine management and HSCs. The TB control programme committee should engage with the appropriate community TB control programmes – primarily to ensure continuity of treatment and good referral services, with follow up.

8.4.3.8 Contact tracing

Contact tracing involves identifying, assessing, and managing individuals who have recently been in contact with someone diagnosed with TB, to prevent further transmission and early detection of infection. The primary objective of contact tracing is to identify and treat individuals with active TB disease to prevent further spread, offer preventive treatment to individuals with latent TB infection (LTBI), and to prevent them from developing active TB as well as to identify and treat the source of an outbreak.

Contacts are regarded as people who have shared airspace with someone with infectious TB disease, such as household contacts, close friends, and work colleagues. Early detection and treatment of TB cases are crucial for preventing further transmission. Contact tracing helps to identify and treat individuals who may be at risk of developing TB, and it is a key tool in the control of TB and the prevention of secondary cases.

8.4.3.9 Passive case detection

Passive detection of TB cases is the reliance on self-presentation of individuals, with symptoms, to healthcare facilities. Delays in self-presentation with symptoms, particularly a cough, may occur, as coughing is a common symptom among miners. Significant delays may also occur in the diagnosis of TB if healthcare workers do not have a high index of suspicion for TB, and initiate investigations timeously. To combat this, it is recommended that:

- Self-presentation of employees should be promoted, through regular educational campaigns or use of wellness coordinators
- Healthcare workers, health and safety teams, and wellness coordinators should be trained in TB awareness, to minimise delays caused by the health service, and enhance prompt diagnosis and treatment of TB

8.4.3.10 Active case detection

Active case finding uses systematic screening, with radiography and/or sputum, to identify cases of active TB disease that have not been detected on symptoms. Active case finding may be conducted either to estimate TB prevalence, as part of contact tracing in close contacts of patients with active TB, or to intensify control measures in communities or individuals with a high risk of TB. Active case finding is expensive and, to be effective, requires access to an efficient TB treatment programme. Apart from prevalence estimation, the aims are to limit transmission to others and reduce morbidity, by detecting disease at a relatively early stage.

Because of the expense, active case finding, other than household contact screening, is not recommended as a core part of TB control in most settings. South African miners are one of the few communities in the world in which routine radiographic screening is conducted annually. Additionally, high-risk groups, such as silicosis, HIV-positive miners, and room contacts, may be considered for more frequent screening.

8.4.4 Diagnosis of tuberculosis

Tuberculosis diagnosis depends on symptom screening of all patients (including HIV-positive patients) presenting to the health facility and contacts of people with laboratory-confirmed PTB disease. All those who have symptoms of TB disease must be investigated for TB.

A GeneXpert XDR (Xpert MTB/XDR) cartridge should be used to detect fluoroquinolone and isoniazid resistance. This test also detects resistance to ethionamide and the second line injectables (amikacin, kanamycin, and capreomycin) in a single test.

Phenotypic testing for linezolid and bedaquiline should be used for all patients who have RR-TB. This test can also be done at the clinician's request. Pretomanid testing should be performed at the national TB reference laboratory for all isolates resistant to fluoroquinolones.

Algorithms for the management of both new and retreatment TB are presented in **Appendices 8.6** and **8.7.** Investigations for TB are initiated once a patient is suspected of having TB. Within the mining industry, further TB investigation is warranted if there is suspicion, because radiological diagnosis of TB in the presence of silicosis may be more difficult to assess, owing to similarities in radiological presentation. However, the microbiological diagnosis of TB remains unaltered in the presence of silicosis.

A diagnosis of TB should be based on strict criteria, and a presumptive diagnosis of TB, particularly pulmonary TB, is discouraged.

8.4.4.1 Symptoms

The presence of TB should be suspected in any individual who presents with one or more of the following symptoms:

- Persistent cough
- Sputum production, particularly if blood stained
- Chest pain, shortness of breath
- Loss of appetite and loss of weight
- Night sweats and fever
- Malaise (general fatigue and weakness)

Coughing is a common symptom among miners, and a cough alone is less likely to result in presentation to the health service. Miners are more likely to self-present to the health service if they have symptoms such as chest pain, shortness of breath, fever, loss of weight, and coughing up blood. Healthcare workers and wellness coordinators should be trained to recognise these symptoms and investigate promptly for TB.

Extrapulmonary TB (TB outside the lungs) occurs relatively commonly in miners (20). Tuberculosis may occur as TB pleurisy (infection of the lining of the lungs), pericarditis (infection of the outer heart lining), and lymphadenitis (infection of the lymph glands). Healthcare workers should, therefore, also be familiar with symptoms and signs suggestive of extrapulmonary TB.

Central chest pain that is relieved by sitting forward, tachycardia, and low blood pressure

suggests TB pericarditis. Pleuritic chest pain (sharp pain made worse by deep breathing) is suggestive of TB pleurisy. Lymphadenopathy on the CXR, particularly if asymmetrical in an HIV-positive individual, warrants investigations for TB.

8.4.4.2 Bacteriology

For miners with suspected TB, screening is conducted at mine clinics using the following techniques:

8.4.4.2.1 GeneXpert MTB/XDR test

The SAMI uses GeneXpert MTB test to detect XDR-TB, which involves collecting a sputum sample, mixing it with a provided reagent, loading it into a cartridge, and placing the cartridge into a GeneXpert instrument
for automated processing, using 10-colour panel technology.

8.4.4.2.2 Urine lipoarabinomannan

The other test used by the SAMI is the lipoarabinomannan (LAM), which is a rapid, point-of-care test that detects the presence of a mycobacterial LAM antigen in urine, assisting in the diagnosis of active TB, particularly in PLHIV who are severely ill or have advanced HIV disease.

8.4.4.2.3 Indications for tuberculosis culture

One sputum specimen should be collected for culture and organism identification. If the Ziehl-Neelsen method is used for smear microscopy, then a separate specimen will be required for culture. If the auramine method is used, then the same sputum sample can be used for culture. Available resources would determine the culture medium used. Culture using Lowenstein-Jensen medium is cheap and reliable but is rarely offered by private laboratories. Alternative culturing systems provide reliable results more rapidly but are more expensive. Each TB programme should review with their TB laboratory the most appropriate TB culture to use.

All positive culture should have the organism identified. The reason for this is that non-tuberculosis mycobacteria (NTM) disease occurs commonly among miners, is indistinguishable clinically from TB, and is treated differently from TB.

Failure to diagnose NTM disease would result in inappropriate therapy, progression of disease, and worse treatment outcomes. This is discussed further in the last section of the chapter.

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8.4.4.2.4 Tuberculosis drug susceptibility testing

Routine drug susceptibility testing is not recommended. The same indications, as in the National TB Guidelines, are used for drug susceptibility testing. These are:

- Failure of new smear-positive cases to convert their sputum smears from positive to negative after two months of TB treatment
- All retreatment cases at diagnosis (see section 8.4.5.1 below for definition of retreatment cases).

In addition, patients who have had previous TB preventive therapy, but who are diagnosed with TB, should have drug susceptibility testing requested at the time of TB diagnosis.

Ideally, drug susceptibility testing should be done for the primary anti-TB drugs, such as isoniazid, rifampicin, ethambutol, and streptomycin. Testing against pyrazinamide can be done using specific liquid culture techniques such as the BACTEC system. Some laboratories offer a testing algorithm, starting with tests against rifampicin, or rifampicin and isoniazid, and test against other drugs only if there is resistance to these two drugs, to reduce costs. Susceptibility testing against the second line drugs should be done only if there is multiple drug resistance, particularly against isoniazid and rifampicin.

8.4.4.2.5 Laboratory quality assurance

The programme manager should ensure that the TB testing has both internal and external quality assurance programmes linked to an accredited TB reference laboratory. The results of the quality assurance programme should be made available to the programme manager on a quarterly basis for review.

8.4.4.3 Chest radiography

A chest radiograph is indicated in patients with suspected PTB. The justification for this is:

- All PTB cases require confirmation with a large plate CXR and post-TB lung function testing, for assessment of compensability under the ODMWA and COIDA.
- Improvement in the chest radiograph, following TB treatment, provides supportive evidence of a diagnosis of TB in patients with smear-negative, pleural, and pericardial TB.
- Non-tuberculous chest pathology occurs commonly among miners.
- A chest radiograph is required at diagnosis and following completion of treatment, for notification and compensation assessment by the MBOD.
- Serial chest radiographs are essential for comparative purposes over time, to distinguish between TB and pneumoconiosis.

The diagnosis of PTB should not be based on the chest radiograph alone.

It should also be noted that the radiographic pictures of silicosis and TB may be similar. Tuberculosis should be suspected in silicosis patients who become symptomatic or develop new or changing radiological lesions (see section 8.2.3 above).

The chest radiographic picture of TB in HIV-positive patients is often atypical. A diagnosis of TB, therefore, should not be excluded in HIV-positive patients on the basis of the chest radiograph alone.

8.4.4.4 Criteria for diagnosis and classification of tuberculosis

8.4.4.4.1 Diagnostic criteria

The following criteria are recommended for diagnosis of pulmonary TB (65):

- Two positive sputum smears
- One positive sputum smear and a compatible clinical and/or radiological picture
- One positive sputum culture
- Three negative sputum smears, a compatible clinical and/or radiological picture, and no response to a course of broad-spectrum antibiotics. Response to TB treatment provides supportive evidence for the diagnosis of TB.

Patients who do not meet these criteria should not be treated for TB but should be investigated for other conditions.

8.4.4.4.2 Classification according to site of disease

Tuberculosis cases should be classified as pulmonary or extra-pulmonary TB. Patients who have both extra-pulmonary and pulmonary TB should always be classified as PTB.

Patients diagnosed with PTB should be classified as:

- Smear positive if one or more smears are positive, including scanty positives
- Smear negative if three sputum smears are negative

Classifying TB cases as smear positive or negative is required to create cohorts, to determine treatment outcomes.

8.4.4.5 Clinical and radiological features of HIV-associated tuberculosis

In addition to increasing the risk of disease, HIV infection alters the clinical and radiological presentation of TB. Most, but not all, case series have shown that a lower proportion of HIV-positive TB patients have smear-positive disease, and that a higher proportion have extra-pulmonary and disseminated disease than HIV-negative TB patients.

The radiological manifestations of PTB are also affected by HIV infection, so patients with HIV-associated TB more commonly have hilar adenopathy and/or pleural effusions and a lower prevalence of cavitation than HIV-negative TB patients. The impact of HIV infection on clinical and radiological features is related to the degree of immunosuppression, being more marked in those with low CD4+ T-lymphocyte counts (CD4 counts). The lobar distribution of radiological abnormalities also differs between HIV-positive and HIV-negative TB patients. Tuberculosis in HIV-negative adults usually has a ‘reactivation’ pattern, with the disease process starting either in one of the apical segments or in one of the apical segments of the lower lobes. A ‘progressive primary’ type of radiological appearance, with lobar consolidation and hilar adenopathy rather than cavitation, is much more common in HIV-positive than HIV-negative adults and is associated with lower CD4 counts and more advanced immunosuppression.

Mortality during TB treatment is higher in HIV-positive than HIV-negative patients, but this seems to be mainly due to a high incidence of other opportunistic infections in patients with HIV-associated TB. A diagnosis of HIV-associated TB has been associated with an increased mortality rate when TB patients are compared to other HIV-positive individuals, who have similar CD4 counts, but do not have TB. Among HIV-positive TB patients, survival is shorter in those with extra pulmonary and disseminated disease than those with pulmonary disease alone and is adversely affected by a low CD4 count.

In non-mining patients, both the risk of treatment failure and recurrence rates, after treatment with rifampicin-based regimens, are similar for HIV-positive and HIV-negative patients. In contrast, two studies in South African miners have shown a significantly increased rate of recurrence in HIV-positive compared to HIV-negative miners following treatment for TB. In such a high transmission setting, there is now good justification for using secondary isoniazid prophylaxis after TB treatment (see section 8.4.5.9.5).

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8.4.5 Treatment of tuberculosis

8.4.5.1 Case definitions

Treatment regimens vary according to treatment category and site of disease. The following case definitions should be used for treatment category:

- New cases are patients who have not previously been treated for TB for more than one month.
- Retreatment cases are patients who have previously had more than one month of TB treatment and now require retreatment. Retreatment for TB may be required for relapse following previous cure, interruption of treatment, or treatment failure.

8.4.5.2 Short (six-month) tuberculosis regimen

The six-month BPaL-L regimen, comprising bedaquiline, pretomanid, linezolid (600 mg), and levofloxacin, is used programmatically in place of nine-month or longer (> 18 months) regimens, in non-pregnant patients (aged ≥ 15 years) with MDR/RR-TB and Pre-XDR-TB, who have not had previous exposure to bedaquiline, pretomanid, and linezolid (defined as > 1 month exposure).

Individuals who had more than one month exposure of second line drugs are started on bedaquiline, pretomanid, linezolid, and levofloxacin (BPaL-L), but resistance to bedaquiline and linezolid must be excluded. Treatment initiation must not be delayed pending results of resistance tests. This regimen may be used without levofloxacin (BPaL) in the case of documented resistance to fluoroquinolones.

Drug susceptibility testing to fluoroquinolones should be done using the GeneXpert XDR cartridge, but this should not delay treatment initiation. All persons who are not responding to therapy should be identified early (i.e. based in part on month 3 culture status), and should be reviewed by the Provincial Clinical Advisory Committee (PCAC) or National Clinical Advisory Committee (NCAC) for a possible rescue regimen.

8.4.5.3 Long individualised regimen

This treatment regimen is offered to persons with documented resistance to pretomanid and/or bedaquiline and/or linezolid. Prior exposure to pretomanid, linezolid, and bedaquiline for more than a month is not an indication for long individualised regimen. Extended drug susceptibility testing is done on request by clinicians only to support long individualised regimen. The composition of the regimen depends on the drug resistance pattern, prior drug exposure, and toxicity. The long individualised regimen can be offered to persons with RR-TB, who have a high risk of resistance to bedaquiline and/or linezolid.

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8.4.5.4 In-patient and ambulatory care

All patients who are infectious (smear positive) or clinically ill should be admitted to a TB facility for initial care. Once patients are clinically well and no longer infectious, (converted from sputum smear positive to negative), they may be discharged from the in-patient facility.

Patients may return to work on treatment if clinically fit to do so.

These individuals are no longer infectious and would not pose a threat to other employees. Cure rates of miners who return to work while taking TB treatment are good. The WHO

supports the return to work of TB patients as soon as possible. This also applies to underground mining work.

8.4.5.5 Adherence to therapy

- All treatment must be administered under direct observation.
- Trained treatment supporters, other than healthcare workers, may be used.
- Adherence to therapy can further be improved if the directly observed therapy is provided in a patient-centred manner, by providing TB treatment at a time and place convenient to the patient, by treatment supporters who are sympathetic to the cultural and economic needs of the patient.
- Treatment dispensed under directly observed therapy should be recorded on a TB treatment record card.
- The TB record cards should be reviewed at least weekly. Patients who have missed treatment should be traced and encouraged to continue treatment.

8.4.5.6 Missed treatment

During the intensive phase: if no more than 10 doses were missed, then these should be added onto the end of the intensive phase. If more than 10 doses were missed, then the patient should be registered and managed as a standard retreatment case.

During the continuation phase: if more than 40 doses were missed and the patient is sputum smear negative, then the continuation phase should be completed. If the patient is sputum smear positive, then the patient should be commenced on a retreatment regimen.

8.4.5.7 Monitoring response to treatment

Monitoring response to treatment among patients with new and retreatment TB is outlined in **Appendices 8.6** and **8.7** respectively.

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8.4.5.7.1 New cases

Two sputum smears should be examined at the end of the intensive phase (after two months) and in the last month of treatment. If smears are negative at the end of two months of treatment, then the patient can be changed to the continuation phase of treatment. If one or more smears are positive, then an additional month of intensive phase treatment should be given. Sputum smears should then be repeated at the end of the third month of treatment, and the patient changed to the continuation phase of treatment. If smears are positive, then a specimen should be obtained for drug susceptibility testing. Treatment should not be altered, unless resistance to anti-TB drugs is demonstrated.

8.4.5.7.2 Retreatment cases

Two sputum smears should be examined at the end of the intensive phase (after three months) and in the last month of therapy. If the sputum smears are positive at the end of the intensive phase, and if drug susceptibility testing was not done at diagnosis or the culture was contaminated, then a repeat sputum specimen should be obtained for drug susceptibility testing. Treatment should only be altered according to drug susceptibility testing.

8.4.5.8 Treatment outcomes

8.4.5.8.1 Case definitions

The case definitions shown in **Table 8.17** are used for treatment outcomes, in accordance with the National TB Control Programme guidelines.

Table 8.17 Case definitions for tuberculosis treatment outcomes

Cure	Patient is smear negative within the last month of treatment and on at least one previous occasion
Treatment completion	Patient completed treatment without bacteriological confirmation of cure
Treatment failure	Patient has positive sputum smears or cultures within the last month of treatment
Died	Patient died for any reason during the course of treatment
Treatment interrupted	Patient interrupted treatment after a month, for more than 10 days during the intensive phase of treatment or more than 40 days during the continuation phase of treatment
Transfer out	Patient was transferred to another clinic for treatment

8.4.5.8.2 Cohort analysis

Response to treatment is evaluated by creating cohorts of smear-positive PTB cases, over a specified period. Treatment outcomes are defined according to the above case definitions, and expressed as a percentage of total known outcomes. Confirmed cases of NTM disease should be excluded from the cohort of patients when determining treatment outcomes.

The National TB Control Programme register should be used for reporting treatment outcomes. Evaluation of treatment outcomes should be done separately for new cases, and retreatment smear-positive PTB cases. If a significant proportion of patients are transferred out on treatment due to retrenchment, then the cohort analysis should be repeated after excluding these patients, to obtain an accurate cure rate of miners treated in service.

The following treatment outcome targets are recommended for smear-positive pulmonary TB cases that were not transferred out while on treatment:

- Cure > 85%, in accordance with WHO guidelines
- Treatment failure < 5%
- Treatment interruption < 5%

8.4.5.9 Special treatment situations

8.4.5.9.1 Smear-negative disease

The priority of any TB control programme is to stop the transmission of TB by curing new infectious smear-positive patients. However, smear-negative disease occurs commonly among miners, owing to the earlier detection of TB by the radiological screening programme.

The treatment regimens are the same as for smear-positive cases. The National TB Control

Programme does not require documentation of treatment outcomes for smear-negative PTB cases. However, the mining industry is encouraged to determine treatment outcomes for smear-negative TB cases. The monitoring of response to therapy by repeating sputum smear examination, at the end of the intensive phase and end of treatment, is not required. For these cases, cure can be defined as a good clinical response to treatment and the completion of treatment.

8.4.5.9.2 HIV-associated tuberculosis

8.4.5.9.2.1 HIV testing

HIV counselling testing should be undertaken where individual pre- and post-test counselling is provided by trained counsellors, with informed consent and confidentiality of results assured.

HIV counselling and testing has several benefits for the HIV-positive and -negative patient. The HIV-positive patient has the opportunity to receive emotional support, plan for the future, and protect sexual partners. For the HIV-negative patient, healthcare providers can offer preventive therapy, enabling the reduction of opportunistic infections while the patient is on TB treatment, and of recurrence of TB once completed. HIV-negative patients should be encouraged and supported to maintain their negative status.

8.4.5.9.2.2 Treatment of HIV-positive cases of tuberculosis

The same treatment regimens are used for HIV-negative individuals. The case fatality rate is higher among HIV-positive patients, compared to HIV-negative patients. Cure rates among HIV-positive TB patients, who survive to the end of treatment, are like those in HIV-negative TB patients.

Morbidity and mortality due to opportunistic infections occur commonly among HIV-positive patients. Use of cotrimoxazole in HIV-positive TB patients has been associated with reduced morbidity and mortality from opportunistic infections, particularly due to bacterial infections. Cotrimoxazole chemoprophylaxis may be of limited value among HIV-positive miners, owing to a high level of cotrimoxazole resistance among commonly occurring bacterial pathogens, and a high prevalence of opportunistic infections that are not preventable with cotrimoxazole, such as cryptococcal disease.

8.4.5.9.3 Silico-tuberculosis

The same treatment regimens as for TB are used to treat silico-tuberculosis, and similar outcomes can be expected among those who survive to completion of treatment. However, silico-tuberculosis may be associated with an increased mortality compared to TB patients without silicosis (9).

8.4.5.9.4 Extrapulmonary tuberculosis

Pleural and pericardial TB (both compensable) are the most common forms of extrapulmonary TB among miners. The diagnosis of pleural TB should be confirmed by histology or bacteriology. Sputum microscopy and culture should be requested, as a significant number of cases may have positive smears. Culture of the pleural fluid or tissue has a high diagnostic yield, particularly in HIV-positive individuals.

A diagnosis of TB pericarditis should be presumptive, based on the presence of a fibrinous

pericardial effusion on ultrasonography and/or echocardiography in the absence of another cause. Sputum microscopy and culture should also be requested. Response to therapy provides supportive evidence of the diagnosis. The treatment of pleural and pericardial TB is the same as for other TB cases.

8.4.5.9.5 Multidrug-resistant tuberculosis

Multidrug-resistant TB is defined as resistance of *M. tuberculosis* to isoniazid and rifampicin, with or without resistance to other TB drugs.

To minimise the risk of drug resistance, initial therapy should follow established guidelines, and should be with four drugs, preferably given as combination tablets, or five drugs if the patient has had TB treatment before.

The treatment of MDR-TB should only be undertaken in specialised centres by experienced clinicians. It is, therefore, strongly recommended that unless facilities for in-patient isolation exist and experienced clinicians supervise treatment, that these patients be referred to an appropriate centre.

Once MDR-TB is diagnosed, the patient should be isolated and the regimen changed to include at least two, and preferably three or four, drugs to which the isolate is sensitive. Single new drugs should never be added to a failing regimen: it is better to wait for the laboratory sensitivity patterns before making changes. Surgery to remove or close cavities should be considered if second line medical therapy fails.

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For further information, consult the DoH guidelines for the management of MDR-TB (25).

Notification of TB cases at diagnosis (new and retreatment), or death from confirmed or suspected TB, is required to be sent to the DoH (Form GW17/5).

Quarterly reports for case rates and treatment outcomes should be submitted to the local health authority (GW 10/16).

8.4.6 Occupational issues

8.4.6.1 Reporting

All cases of suspected or confirmed cardio-respiratory TB (CR-TB) must be reported to the MBOD when suspected. Cardio-respiratory TB is compensable under the ODMWA and, according to the definitions under the ODMWA, CR-TB includes disease to the larynx, trachea, bronchial tree, lung parenchyma, pleurae, lymphatic system of the lungs, vascular system of the lungs, nerve supply of the lungs, diaphragm and nerve supply to the diaphragm, heart, pericardium, and large intrathoracic blood vessels (2).

It is recommended that the following be reported to mine managers and mine HSCs, on a quarterly basis:

- Number and proportion of employees with the inability to perform customary duties due to a disease or injury, impacting on an employee's ability to find or retain employment (medical incapacitation), and fatalities due to CR-TB
- Number and proportion of lost shifts due to CR-TB
- Number of cases of CR-TB submitted to the MBOD, alone or in combination with other OLDs

- Number of cases of CR-TB, alone or in combination with other OLDs, certified by the MBOD, and the degree of impairment, i.e. first- or second-degree certification

Data from these reports can be incorporated into the annual medical report required under the MHSA.

8.4.6.2 Sick leave and compensation

Miners on sick leave for TB receive their normal salary for 42 days if they have worked for the company for one year, and up to 82 days if they have worked for more than two years. (Certain MCSA member companies have had agreements with employee groups for employees to receive 100% payment for six months, while on sick leave for an occupational disease or injury.)

Under the ODMWA, employees who suffer loss of earnings, as a result of being off work with CR-TB, are eligible for compensation for loss of earnings to the value of 75% of the earnings lost, for a maximum of six months. These monies are payable to the employee retrospectively by the Compensation Commissioner for Occupational Diseases. However, miners with CR-TB will be awarded with loss of earnings only if they have exceeded their sick leave allowance.

It is the OMP's duty to ensure that all cases of CR-TB are reported to the MBOD, even if they have previously been certified and compensated as a first-degree OLD. See Chapter 16 of this Handbook (Fitness to Work, Disability, and Compensation). Once a second-degree award has been paid, no further compensation in any form is due.

Miners who successfully complete treatment for CR-TB must undergo a benefit medical examination, under the ODMWA, one year after completion of TB treatment. Those patients considered to fall into a compensable category, due to lung function impairment, should have a full benefit examination, which includes a medical report, a current full-sized chest radiograph (preferably in digital format), and LFTs, and the case should then be forwarded to the MBOD for assessment for compensation. The responsibility for notification and assessment of impairment rests with the OMP. See Chapter 16 of this Handbook (Fitness to Work, Disability, and Compensation) for further details on compensation.

8.4.6.3 Termination of employment contract

Where possible, termination of the contract of employment of miners while on the intensive phase of treatment should be avoided.

Miners whose contract of employment is terminated, for whatever reason, while on TB treatment should undergo an exit examination prior to leaving. The patient should be classified as a transfer out in the TB register, and a referral letter given to the patient detailing the TB diagnosis made and the current treatment.

Cardio-respiratory TB is considered an occupational disease if diagnosed within 12 months after leaving risk work. Former miners who develop CR-TB during this time, and who have worked more than 200 risk shifts, are eligible for a benefit examination and submission for compensation See Chapter 16 of this Handbook (Fitness to Work, Disability, and Compensation). If underlying silicosis or any other OLD is present, then CR-TB diagnosed any time after the diagnosis of such OLD will be considered for compensation by the MBOD.

8.4.6.4 Nosocomial (hospital-acquired) tuberculosis

Transmission of TB may occur within healthcare facilities to patients and healthcare workers alike. Policies and procedures that ensure a safe environment for healthcare workers and patients should be in place. Measures to reduce the nosocomial transmission of TB include administrative and environmental controls and personal respiratory protection.

Administrative controls should take precedence over all other measures to reduce nosocomial transmission of TB. Without effective administrative controls, environmental and personal respiratory protection are of limited value. Administrative controls should include policies and procedures for the identification and isolation of patients with suspected infectious TB, both as out- and in-patients, and early diagnosis and treatment. Known infectious TB patients leaving isolation areas for further investigations should be required to wear a disposable surgical mask.

Environmental controls aim to reduce the concentration of infectious droplet nuclei in high-risk areas in which healthcare workers or patients may be exposed. Natural ventilation should be maximised by keeping windows open and doors closed. Exhaust ventilation systems may be used, in isolation rooms or wards, to produce negative pressure that minimises the escape of contaminated air into hallways and surrounding areas. Patients should be encouraged to cough into tissues. Ultraviolet irradiation, although effective, requires proper installation and careful ongoing maintenance.

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Personal respiratory protection masks are expensive and not generally available. The use of such masks by healthcare workers may be considered when performing cough-inducing procedures, or when caring for MDR-TB patients.

8.4.6.5 Tuberculosis in healthcare workers

In a comprehensive guideline for protecting health workers from occupational TB, Ehrlich et al. (2020), (66) highlight that health workers remain at high occupational risk of TB, despite the existence of numerous infection prevention and control (IPC) guidelines over the past 25 years. Implementation of these guidelines has been inadequate and inconsistent**,** leading to continued risk. They conclude that adopting a system-wide, worker-centred occupational health strategy would not only reduce TB risk for health workers, but also enhance the resilience of health systems to other infectious disease threats.

Refer to the COIDA circular instruction for TB in healthcare workers which covers the definition, diagnosis, impairment, benefits, reporting, and compensation claims processing. The latest guidelines for TB preventive therapy in the NSP 2023–2028 should be adhered to.

8.4.7 Tuberculosis control programme evaluation

Each mining company should evaluate its TB control programme performance, annually. The TB control programme can be evaluated by monitoring trends for case rates, treatment outcomes, relapse rates, and prevalence of drug resistance.

If treatment outcome targets are not being met, then the TB control programme should be audited to determine possible reasons for not meeting these targets.

Process measures that should be audited include:

- Proportion of TB cases treated under DOTS
- Adherence to treatment, determined by reviewing treatment record cards

- Bacteriological coverage of pulmonary cases started on treatment
- Proportion of smear-positive cases that have sputum collected at the end of the intensive phase and end of treatment

The effectiveness of the TB control programme can be determined, further, by the relapse rate among cohorts of patients with new smear-positive pulmonary TB. The relapse rate should be < 5% over two years for HIV-negative patients.

The prevalence of drug resistance, particularly primary drug resistance, is a good indicator of the overall success of the TB control programme. Surveillance of drug resistance should be conducted every three to five years.

8.4.8 Other interventions to reduce incidence of tuberculosis

8.4.8.1 Dust control

As most of the workforce is occupationally exposed to dust and is at risk of developing silicosis, improved dust control is of critical importance in improving TB control.

Strategies to improve dust control are discussed in Chapter 4 of this Handbook (Occupational Health Management in the Mining Environment). However, it should be borne in mind that even if sustained dust exposures well below the OEL were achieved with immediate effect, the prevalence of silicosis would decline only over decades, as employees leave the industry. As a result, the impact of improved dust control on TB case rates would become evident only in the long term.

8.4.8.2 Housing

The 1995 Report of the Commission of Enquiry into Safety and Health in the Mining Industry, known as the Leon Commission, identified unacceptable housing conditions on some mines (11). The report noted the long historical association between mining, the migrant labour system, and single-sex hostels, and the likely negative effects on health of this type of mine accommodation.

Modelling of the factors associated with TB incidence, now being done for SIMRAC, indicates a lower risk for TB among miners in non-hostel single partner or family accommodation.

The conditions needed for adequate respiratory health, generally, including reduction of the risk of TB transmission, include uncrowded sleeping and living quarters, good ventilation, and the minimum of pollution from indoor fuels. Therefore, the upgrading of accommodation on mines, as recommended by the Leon Commission, should be regarded as part of a TB control strategy.

The SAMI responded to this recommendation by converting or upgrading single-sex hostels or compounds into family units, extending the space to attain a one-person-per-room occupancy rate, and facilitating home ownership options for all mine employees.

8.4.8.3 Tuberculosis preventive treatment

Tuberculosis preventive therapy is an underutilised, but highly effective, method of reducing

an individual's risk of TB, by eradicating recently acquired or latent TB infection.

Isoniazid has been used since the 1950s to reduce the risk of active TB in asymptomatic individuals, shown to be infected with *M. tuberculosis* based on skin test reactivity.

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When taken for between 6–12 months, isoniazid treatment has been shown to reduce significantly the subsequent incidence of TB in a variety of high-risk groups, including children with recent skin test conversions, adults living in, or emigrating from, high TB prevalence areas, individuals with radiological evidence of self-healed TB, silicosis, and HIV-positive individuals.

Six months of isoniazid treatment has been the recommended standard, considered to maximise cost-effectiveness. This recommendation has recently been changed to nine months in the USA (67).

8.4.8.3.1 Tuberculosis preventive therapy and HIV infection

A clear benefit in reduced TB incidence, in the short term, has been shown when six months of isoniazid treatment is taken by tuberculin skin test or PPD-positive individuals, who are also HIV infected. The long-term effectiveness is less certain, since incidence begins to rise again soon after the end of treatment. It is also not yet clear to what extent medium-term mortality is reduced. The benefit of treating individuals who are PPD-negative, or too immunosuppressed to make any skin response (anergic), is smaller and was not significant in a recent meta-analysis.

8.4.8.3.2 Preventive therapy for silicosis

There have been two placebo-controlled trials of preventive therapy in presumed HIV-negative silicosis; one in South African miners and one in granite workers living in Hong Kong.

The Hong Kong study compared placebo with six months of isoniazid, three months of rifampicin alone, and three months of isoniazid plus rifampicin. Each of the three active regimens led to a significant reduction in TB incidence, compared to placebo, and was of equivalent efficacy (estimated reduction in TB incidence of 48%, 63%, and 41%, respectively, at five years). The South African trial compared three months of rifampicin, isoniazid, and pyrazinamide with placebo and found no significant difference, with an annual TB incidence in the order of 1.5 per 100 person-years during the four years of follow up in both groups. The lack of efficacy was probably the result of outside reinfection.

The current American Thoracic Society (ATS) guidelines recommend that workers with silicosis be offered 12 months of isoniazid therapy.

Useful guidelines on TPT are available at The Aurum Institute website: https://www.auruminstitute.org/component/edocman/clinician-tools-tb.

8.4.8.3.3 Isoniazid toxicity and resistance

The serious side effects of isoniazid are peripheral neuropathy (nerve damage), which can be treated or prevented by the vitamin pyridoxine, and hepatitis (liver damage), which is occasionally severe and fatal.

Risk factors for isoniazid-induced hepatitis include older age and heavy alcohol consumption. The risk of mild biochemical hepatitis is under 1%. The fatality rate has been estimated to be about 1 in 10 000, or even below.

A further potential hazard of isoniazid treatment is that subjects with minimally symptomatic active TB may inadvertently be given isoniazid monotherapy, creating a risk of inducing isoniazid resistance. This appears to be a rare event in practice, provided that patients are screened to exclude active TB before starting isoniazid treatment. Screening can be based on history, examination, and chest radiography, with bacteriological examination of sputum only when one or more abnormalities are found.

8.4.8.3.4 Adherence and alternative short course combination regimens

Adherence to preventive therapy has been shown to be poor in all studies where this question has been specifically addressed. Due to better adherence with shorter regimens, alternative short-course preventive therapies lasting two-to-three months, based on rifampicin combined with one or two additional drugs, have been tested. These regimens have generally shown short-term efficacy, equivalent to the traditional six-month isoniazid course. However, most studies have not been large enough to detect anything but significant differences in effectiveness.

Alternative regimens are discussed in the ATS/Centers for Disease Control and Prevention recommendations (see 'Targeted tuberculin testing and treatment of latent tuberculosis infection', under Guide to resources).

8.4.8.3.5 Recommendations for preventive therapy in miners

High-risk HIV-negative miners include those with radiological silicosis. Others with intermediate risk include older or heavily dust-exposed miners, and those with lesser risk factors such as diabetics or patients taking corticosteroid therapy.

Isoniazid preventive therapy should be used for at least the six – preferably nine – months' duration considered standard care for miners in a high-risk group.

However, tuberculin testing should be conducted on HIV-negative individuals at intermediate risk. Isoniazid preventative therapy should be limited to those with reactions of 10 mm or more, using the standard intradermal Mantoux technique with five units of PPD equivalent.

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Patients receiving IPT should receive counselling on side-effects, and then be followed up monthly with clinical assessment for symptoms or signs of hepatotoxicity, peripheral neuropathy, or TB. At each visit, patients should be instructed to interrupt treatment, and present to the healthcare service immediately, following the onset of symptoms of potential toxicity, such as nausea, vomiting, dark urine, or jaundice.

Pyridoxine should be offered to those with symptoms of neuropathy. Isoniazid preventive therapy should be terminated if the patients develop hepatitis or active TB (68).

8.4.8.3.6 3HP tuberculosis preventive therapy

Another TPT strategy is the use of 3HP treatment, which is a short-course treatment regimen that combines two antibiotics, isoniazid and rifapentine, for the treatment of latent

TB. The high doses of isoniazid and rifapentine are taken once weekly for three months.

8.4.8.3.7 Tuberculosis preventive treatment for workers with silicosis

For workers with silicosis, TPT is recommended to prevent TB, especially after ruling out active TB. Such treatment can be administered using regimens like 3HP or 6H, which is a six-month daily dosage of isoniazid. This therapy aims to prevent TB in silica dust-exposed workers, by targeting LTBI before it progresses to TB.

8.4.8.4 Vaccination

Bacillus Calmette-Guérin remains the only TB vaccine available, and the world's most widely used vaccine, despite widely discrepant results from studies of efficacy. While protection against progressive primary forms of TB, such as childhood meningitis, is good, protection against reactivation disease appears to be incomplete, varies between populations, and wanes with passing time since vaccination. HIV infection, silica exposure, and COVID-19 are considered to be relative contraindications to BCG vaccination.

However, the WHO is actively promoting the development of more effective TB vaccines. The TB vaccine candidate pipeline includes various vaccine platforms such as whole cell vaccines, adjuvanted proteins, and recombinant subunit vector vaccines. The candidate vaccines being developed are for prevention of TB in adolescents and adults, for early life immunisation as a BCG replacement, as BCG boosters, for vaccination of TB patients after treatment to prevent disease recurrence, or as immunotherapeutic adjuncts to drug therapy intended to reduce treatment duration.

Recently, the M72/AS01$_E$ candidate vaccine was found to be significantly protective against TB disease in a phase 2b trial conducted in Kenya, South Africa, and Zambia, in individuals with evidence of latent TB infection (69). The point estimate of vaccine efficacy was 50% (90% CI, 12–71), over approximately three years of follow up.

However, the M72/AS01$_E$ candidate vaccine is still in phase 3 clinical trials, and has not yet been approved for general use (70).

BCG revaccination has no place in the prevention of endemic TB in adult populations such as South African miners.

8.4.9 Impact of tuberculosis medication on persons

Tuberculosis medicines can have side effects. Patients should be encouraged to report any concerning symptoms to their nurse or doctor immediately.

Most of the adverse effects are mild, and they disappear when medication is continued or there is a way to ease them. In average, one out of ten persons who get treatment for TB has more severe adverse effects. In such cases, medication might have to be temporarily interrupted.

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Use of TB medicines and certain other medicines at the same time must be taken into consideration by the doctor. Therefore, it is crucial for the doctor to be informed about all the medications the patient is taking, such as those for diabetes, birth control, or preventing

blood clots.

Mild adverse effects are common and can be bothersome, but the patient should not stop medication. Patients are encouraged to discuss these side effects with their healthcare provider to find ways to alleviate them. Here are some typical mild side effects and suggestions for managing them:

- **Orange-coloured urine, saliva, or tears** are caused by rifampicin. Contact lenses may also be stained, so it is best to avoid them during treatment.
- **Redness and itching of the skin in sunlight** are a result of increased sun sensitivity. Using sunscreen and wearing protective clothing is recommended.
- **Nausea, diarrhea, and loss of appetite** are very common side effects, and usually resolve after a few weeks of continuing TB medication. Persistent nausea may be treated with medication.
- **Mild pain in the upper abdomen** can be managed with medication.
- **Mild joint aches** can be alleviated with pain killers and physical therapy.
- **Mild headaches** can be treated with pain relievers.
- **Sleep disorders and depression** can also be managed with appropriate medication.

Serious adverse effects are rare, but if they occur, patients should temporarily stop their medication and immediately contact their healthcare provider. Here are symptoms that require prompt attention:

- **Vomiting and severe abdominal pain** could indicate a serious issue.
- **Yellowing of the skin or eyes** may suggest hepatitis caused by TB drugs. Liver function is monitored regularly with blood tests during treatment.
- **Fever and skin rash** are potential signs of a severe allergic reaction.
- **Severe joint or muscle pain** that hinders movement needs urgent evaluation.
- **Dizziness** may indicate an imbalance or other complications.
- **Tinnitus or hearing issues** that deteriorate should be reported.
- **Decreased urine output or dark urine** could signal kidney problems.
- **Vision deterioration** requires immediate assessment.
- **Seizures** are a critical sign needing urgent medical attention.
- **Visual or auditory hallucinations** should be addressed swiftly.
- **Suicidal thoughts or severe mood changes** (mental health) concerns are as urgent as physical symptoms.
- **Bleeding gums or nose** can indicate an underlying issue.

Some adverse effects of TB medications can be mitigated. For instance, isoniazid often leads to peripheral neuropathy, which manifests as numbness, aching, or tingling in the hands and feet. These side effects can be prevented with the use of vitamin B6 supplements.

Additionally, alcohol consumption during TB treatment significantly raises the risk of hepatitis. Therefore, it is crucial to advise patients to abstain from alcohol throughout their treatment period.

8

8.5 References

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Additional guidelines and resources for OLDs:

1. Govender, VG (4)

2. COIDA circular instructions:

   a) Circular Instruction No. 174 Regarding Compensation for Occupational Lung Cancer, published under GoN 1324 in GG 23969 of 23 October 2002

   b) Circular Instruction No. 173 Regarding Compensation for Mesothelioma Due to Occupational Asbestos Exposure, published under GoN 1323 in GG 23969 of 23 October 2002

   c) Circular Instruction No. 176 Regarding Compensation for Occupational Asthma, published under GoN 82 in GG 24231 of 10 January 2003

   d) Circular Instruction No. 177 Regarding Compensation for Irritant Induced Asthma, published under GoN 598 in GG 24782 of 25 April 2003

   e) Circular Instruction No. 184 Regarding the Compensation for Work-Aggravated Asthma, published under GoN 336 in GG 27329 of 25 February 2005

   f) Circular Instruction No. 178 Regarding Compensation for Pulmonary Tuberculosis in Health Care Workers, published under GoN 81 in GG 24231 of 10 January 2003

   g) Circular Instruction No. 179 Regarding Compensation for Pulmonary Tuberculosis Associated with Silica Dust Exposure, published under GoN 852 in GG 26585 of 16 July 2004

   h) Circular Instruction No. 187 Regarding Compensation for Work-Related Upper Respiratory Tract Disorders, published under GoN 119 in GG 27216 of 28 January 2005

Appendix 8.1. Legal Duties and Rights with Respect to Occupational Diseases and Medical Surveillance Programmes, in Terms of the Mine Health and Safety Act No. 29 of 1996

Employer must:

- 13 (3): engage the services of a full-time or part-time OMP, together with, insofar as is necessary, other appropriately qualified occupational health practitioners
- 13 (3)(b): provide OMPs with the means necessary to perform their functions
- 18: pay the costs of all clinical examinations and medical tests
- 13 (3)(c), 8 (a) and (b), 15 (2) (a) and (b): keep a record of medical surveillance for each employee exposed to a health hazard, store it safely and not dispose of it for 40 years or until the mine closes. When the mine closes, the records of medical surveillance to be delivered to the Medical Inspector
- 12 (3): keep occupational hygiene records in a manner that can be linked, as far as practical, to each employee's record of medical surveillance
- 17 (1): if the services of an employee who was subject to medical surveillance are terminated for any reason, arrange for an exit medical examination of the employee
- 13 (6): investigate in terms of section 11 (5) if any employee is declared unfit to perform work as a result of an occupational disease

OMP must:

- 13 (5): take every measure that is reasonably practicable to promote the health and safety of employees at the mine and assist employees in matters relating to occupational medicine
- 16 (1) and (2): compile an annual medical report covering employees at that mine, giving an analysis of the employees' health based on employees' records of medical surveillance, without disclosing the names of the employees. Copies of the report to be provided to the employer, the health and safety committee and the DMPR Medical Inspector
- (15) and 13 (2)(a): maintain confidentiality of medical surveillance records, to be stored in a safe place and made available only in accordance with the ethics of medical practice, or if required by law or court order, or if the employee has in writing consented to the release of the information
- 13 (7): inform the employer and employee in the event of the employee being temporarily unfit to perform work, as a result of an occupational disease
- 14 (4) (a and b): when conducting an exit medical examination, ensure that an exit certificate is produced, indicating the results of all medical surveillance and the presence or absence of any occupational disease. A copy of the exit certificate to be entered into the employees' record of medical surveillance
- 19 (2): provide the employee with a copy of the exit medical certificate
- 20 (4): in the instance of an appeal against a finding that an employee is unfit to perform any particular category of work, or a finding contained in an exit medical certificate, provide a report to the Medical Inspector

Employee may/must:

- 19 (1) (a) and (b): request and be provided with a copy of any medical surveillance record or part of it that relates to themself
- 17 (1) and (3): attend the arranged exit medical examination
- 20 (1) and (2): appeal against a decision that they are unfit to perform any category of work, or any finding contained in an exit medical certificate. The appeal must be lodged with the Medical

Inspector within 30 days of the relevant decision

	Annual medical report	SAMODD input form	MBOD benefit application	Exit medical examination
Name and surname of individual	No	Yes	Yes	Yes
Individual's signature	No	No	Yes	Yes
Gender	No	Yes	No	No
National ID number/passport number	No	Yes[1]	Yes[1]	Yes[1]
Industry employee number [1]	No	Yes	No	Yes[1]
Employee number [1]	No	Yes	Yes[2]	Yes[2]
Date of birth	No	Yes	Yes	Yes
Date of death	No	Yes	Yes	No
Individual's address	No	No	Yes	No
Name of mine/employer	Yes	Yes	Yes	Yes
Employee of contractor	Yes	Yes	No	No
Type of mine or commodity mined	Yes	No	Yes[2]	Yes
Total number of employees in the mine	Yes[3]	Yes[4]	No	No
Employee job title	No	No	Yes[2]	Yes[2]
Years of exposure in current job	No	Yes	Yes[2]	Yes[2]
Hazard exposed in current job	No	Yes	Yes[2]	Yes[2]
Previous job titles, years of exposure with current and former employers	No	No	Yes[2]	Yes
Name, address, telephone number of OMP or other person completing form	Yes	Yes	Yes[5]	Yes[5]
Submitting party's case reference number	No	Yes	No	No
Date of initial medical examination	No[6]	No	No	Yes
Date of most recent medical examination	No	Yes	Yes	Yes
Clinical findings at most recent medical examination	No	No	Yes[7]	No
Results of lung function test	Yes[8]	Yes	Yes	Yes
Results of chest radiograph	Yes[8]	Yes	Yes[10]	Yes[9]
Results of other tests[11]	Yes[8]	Yes	Yes	Yes
Diagnosis	Yes[8]	Yes[12]	Yes	Yes[13]
ODMWA benefit application made	No	Yes	-	Yes
ODMWA compensable history	Yes	Yes	-	Yes
Previous compensation history	No	No	Yes	Yes
MBOD reference number	No	No	Yes	No

MBOD: Medical Bureau for Occupational Diseases. ODMWA: Occupational Diseases in Mines and Works Act No. 78 of 1973, SAMODD: South African Mines Occupational Diseases Database

1. National ID number, passport number, DMPR ID number, industry number, company number, TEBA number, and MBOD number are optional for routine reporting as a minimum data requirement.

2. The MBOD and exit medical report require a full occupational exposure history, as well as employee ID numbers with previous employers in the mining industry.

3. The annual report requires that number of employees (including contract workers) subject to medical surveillance in terms of section 13 be reported, together with the total number of hours worked in the reporting period.

4. The SAMODD requires reporting of the employer's workforce for the previous calendar year, classified into surface, underground, open cast, or at sea.

5. The signature is also required.

6. The annual medical report requires a summary of the number of initial, periodical, and exit examinations performed.

7. The MBOD requires past and present medical history, findings of height, weight, blood pressure, general, and chest examination.

8. Results are required to conduct an analysis of employees' health, based on the employees' records of medical surveillance.

9. Use of the ILO Radiological Classification of the Pneumoconioses is mentioned.

10. The MBOD radiologists will interpret the films using the ILO Radiological Classification of the pneumoconioses.

[11.] Tests include audiometry and other biological monitoring.

[12.] Diagnoses are entered by disease group (pneumoconiosis, cardiorespiratory tuberculosis, COPD, NIHL, heat disorders, and other).

[13.] This must state occupational diseases, past or present, including severity.

(Also consider which of the data in **Appendix 8.2** you require to collect directly from employees.)

Appendix 8.3. An Abbreviated Respiratory Symptoms Questionnaire

I am going to ask you some questions, mainly about your chest. I should like you to answer YES or NO, whenever possible. Please think carefully before answering.				
No	**Question**	**Yes**	**No**	**Don't know**
1	**COUGH**: Do you usually have a cough on most days? (Count a cough with first smoke or on first going outdoors) (Exclude clearing of throat)			
2	**PHLEGM**: Do you usually bring up phlegm from your chest? (Count phlegm with the first smoke or on first going outdoors. Exclude phlegm from the nose. Count swallowed phlegm)			
3	**WHEEZING:** Does your chest ever sound like wheezing or whistling?			
4	**CHEST ILLNESS**: During the past three years, have you had any chest illness that has kept you away from work for as much as a week?			
	Has a doctor ever told you that you have? 4.1 Heart trouble 4.2 Bronchitis 4.3 Asthma 4.4 Pneumonia 4.5 Silicosis 4.6 Other chest trouble 4.7 If yes to 4.6, please specify other chest trouble (was it confirmed by a doctor and age at start?)			
5	**TUBERCULOSIS SCREENING SYMPTOMS**: Do you have? • Persistent cough of two weeks or more, OR of any duration if HIV positive • Persistent fever of > two weeks • Documented weight loss > 1.5 kg in a month • Drenching night sweats • Sputum production, particularly if blood stained Chest pain, shortness of breath			
6	**MEDICATION**: Do you take any medication? 6.1 If yes to above, specify condition/disease and medication			
7	**TOBACCO SMOKING**: Have you ever smoked? (Yes = more than 20 packs of tobacco in your life, or more than 1 cigarette a day for a year) 7.1 If yes, do you smoke now? (Yes = present smoker; No = ex-smoker)			
8	**Present smoker**			
	8.1 What was your age in years when you started smoking? 8.2 How much do you smoke per day? (Commercial cigarettes, hand-rolled cigarettes, pipes)			
9	**Ex-smoker**			
	9.1 What was your age in years when you started smoking? 9.2 In the past, on average, how much did you smoke per day? (No. of commercial cigarettes, hand-rolled cigarettes, pipes?) 9.3 How old were you when you stopped smoking?			

Appendix 8.4. Reading Forms for Use in Occupational Lungs Disease Surveillance, Including a Short Form for the ILO Radiological Classification of the Pneumoconioses (8)

Symbols in the short ILO Classification

0/0	Pn not present
0/1	Possibility that Pn present considered, but rejected
p	Small, rounded opacities < 1.5 mm in diameter
q	Small, rounded opacities 1.5–3 mm in diameter
r	Small, rounded opacities 3–10 mm in diameter
s	Small, irregular opacities < 1.5 mm in width
t	Small, irregular opacities 1.5–3 mm in width
u	Small, irregular opacities 3–10 mm in width
1/0	Pn present – insufficient small opacities to classify as category 1, earliest sign of Pn
1, 2 or 3	Small opacities present (refer to standard films)
A	Large opacity presents with greatest diameter between 1–5 cm
B	Large opacity or opacities present with combined area < right upper lobe
C	One or more large opacities with a combined area > right upper lobe

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Abbreviations in the short ILO Classification

cv	Cavitation
ef	Effusion
em	Emphysema
es	Egg-shell calcification of glands
hi	Enlargement of hilar or mediastinal glands
hv	Heart and vessels
oth	Any other pathology or abnormality not represented in the table, which should be described in words
pla	Costophrenic angle obliteration
plc	Pleural calcification (plaque)
plw	Pleural thickening, latent wall
tba	Active tuberculosis, probably
tbu	Tuberculosis, activity uncertain

Appendix 8.5. TB Treatment Comparison Between the National Strategic Plan for HIV/tuberculosis and Those Recommended in this Chapter for the Mining Industry

	National	Mining industry
Case finding		
Active	Yes	Yes
Passive	Yes	Yes
Contact tracing	No (except for children)	No
Diagnosis		
Sputum smear	Yes (two smears)	Yes (three smears)
Sputum culture	One sputum for culture if smear negative at diagnosis and unresponsive to a course of antibiotics	All patients with suspected pulmonary TB should have one sputum specimen submitted for culture
	Failure to convert smear	Failure to convert smear
	Retreatment TB	Retreatment TB
Identification	No	Yes (NTM speciated)
Drug susceptibility testing	Failure to convert smears and for all retreatment cases at diagnosis	Same
Admission criteria		
Smear positive	No	Yes
Clinically indicated	Yes	Yes
Retreatment TB	Yes (initial phase)	Yes
Treatments		
New TB	2RHZE/4HR	Same
Retreatment TB	2RHZES/ 1RHZE/ 5RHE	Same
Treatment frequency	five days/week (initial)	Five days/week (initial)
	five or three days/week (continuation)	Five days/week (continuation)
Smear-negative TB	Yes, if no response to broad-spectrum antibiotics and CXR compatible	Same
DOTS (intensive phase)	Yes	Yes
DOTS (continuation phase)	If possible	Yes
Multidrug-resistant TB	Individualised/standard	Same

	National	Mining industry
CXR At diagnosis Follow up	 Yes, if ≥ 1 smear is negative No	 Yes, on all patients Yes
Treatment outcomes	As per definition	Same

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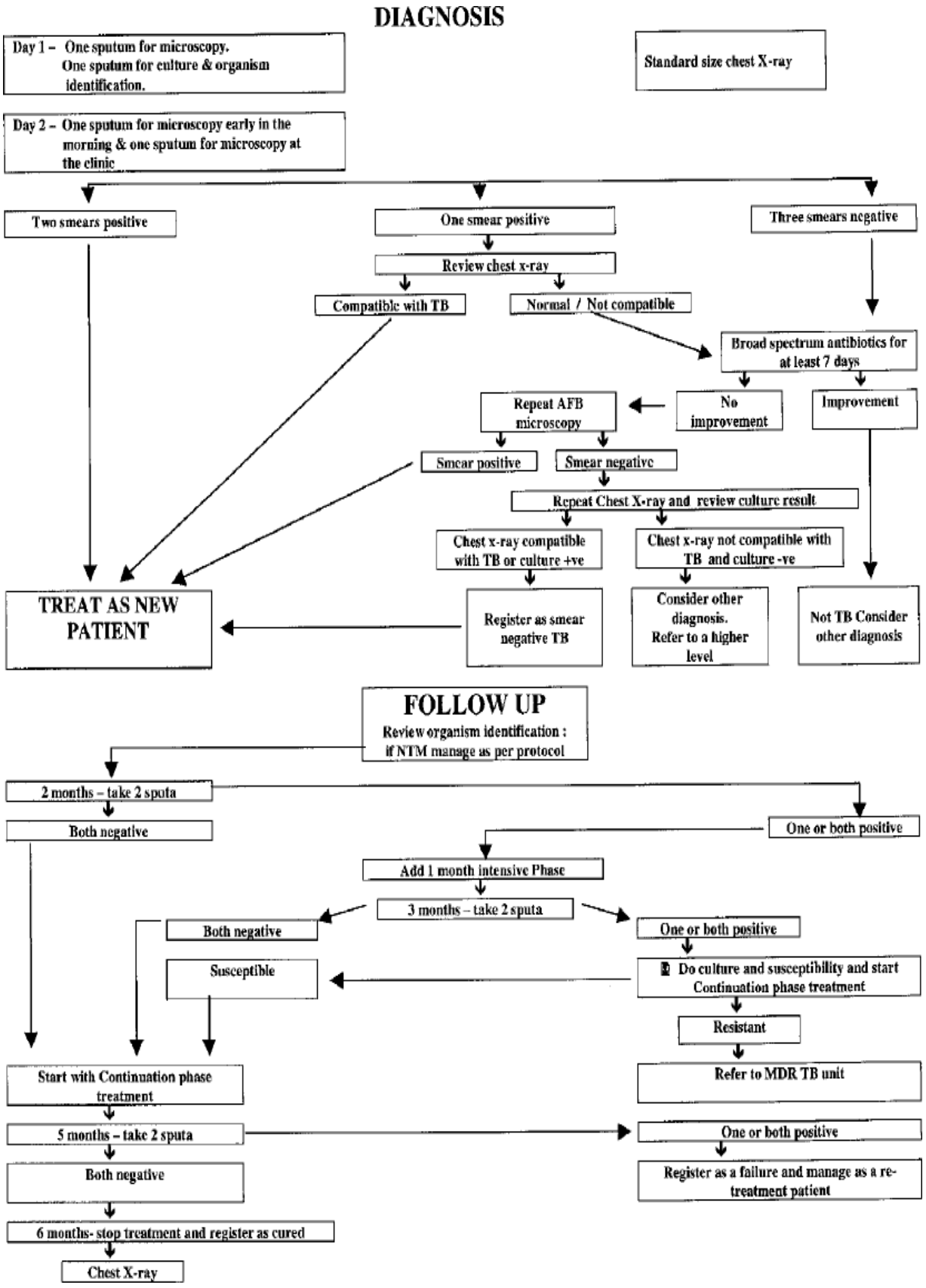
CXR: chest X-ray, E: ethambutol, DOTS: directly observed therapy, short course, H: isoniazid, NTM: Nontuberculosis mycobacteria, R: rifampicin, RSP: radiological screening programme, S: streptomycin, S-/C- = smear and TB culture, TB: tuberculosis, Z: pyrazinamide

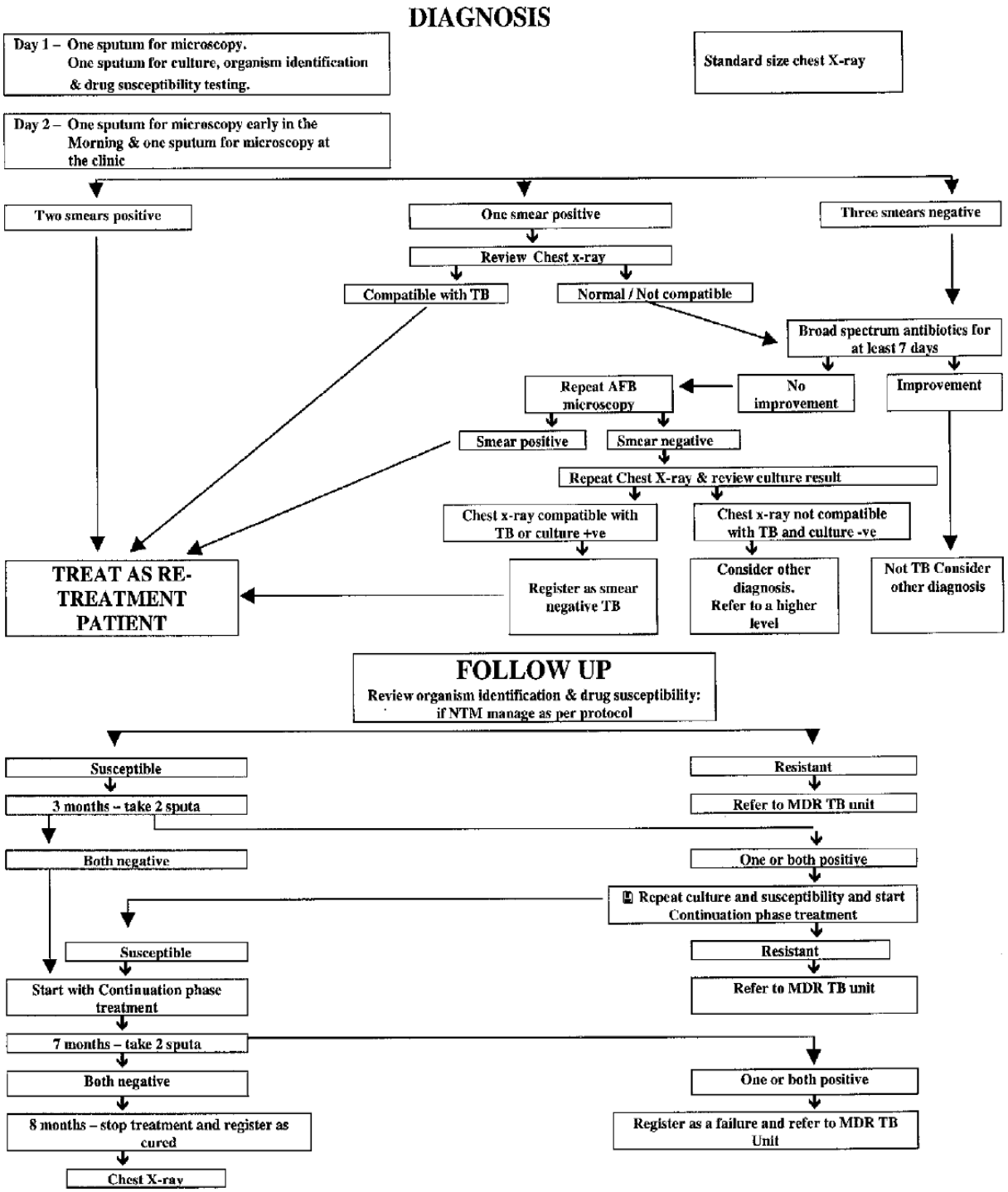
Fixed dose combination tablets should be used for RHZE, RHE, and RH.

Appendix 8.6: Management of New Pulmonary Tuberculosis Patients (Never had more than Four Weeks of Previous Tuberculosis Treatment.



Source: Official American Thoracic Society/Infectious Diseases Society of America/Center for Disease Control and Prevention Clinical Practice Guidelines: Diagnosis of Tuberculosis in Adults and Children (2017) (72)

Appendix 8.7: Management of Retreatment Pulmonary Tuberculosis Patients (Have had more than Four Weeks of Previous Tuberculosis Treatment)



Source: Official American Thoracic Society/Infectious Diseases Society of America/Center for Disease Control and Prevention Clinical Practice Guidelines: Diagnosis of Tuberculosis in Adults and Children (2017) (72)