SORDSA

Surveillance of Occupational Respiratory Diseases in South Africa

ALERT

FEBRUARY 1999

Crystalline Silica

Health Hazards and Precautions

National Centre for Occupational Health (NCOH)
Department of Health
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NCOH/SORDSA

Crystalline Silica: Health Hazards and Precautions

SORDSA ALERT: February 1999

WARNING !!!
Workers exposed to airborne crystalline silica may develop serious or fatal respiratory disease.

1. INTRODUCTION

Occupational exposure to crystalline silica dust constitutes a serious health hazard. Although it is now well established that respiratory diseases associated with exposure to silica dust are preventable, they continue to occur and to cause disability or death. The primary health concerns in subjects exposed to silica dust are the fibrogenic capacity of the inhaled silica particles that can lead to the development of silicosis and an increased risk of tuberculosis. The occurrence of silicosis has been dramatically reduced in many developed and developing countries and currently the International Labour Organization (ILO) and the World Health Organization (WHO) have embarked on a 'Global Elimination of Silicosis Programme'.

2. WHERE DO YOU FIND CRYSTALLINE SILICA DUST?

Silica is the name which collectively describes various forms of silicon dioxide, including both the crystalline and non-crystalline (amorphous) forms of silica. While amorphous silica can be transformed into crystalline forms such as tridymite and cristobalite by heating to high temperatures it is generally only the crystalline forms of silica which are fibrogenic. Quartz is the most common form of crystalline silica found in workplaces.

Industries associated with exposure to airborne crystalline silica include abrasive blasting; brickworks; cement manufacturing; ceramics (pottery, sanitary ware and tiles); construction (sandblasting, rock drilling, masonry work, jack hammering, tunnelling); demolition; electronics; foundries (grinding, moulding, shake-out, core room); glassworks; manufacturing abrasives and paints; mining; railroads (setting and laying track); repair and maintenance of ladles, kilns and furnaces; shipyards (abrasive blasting) and steelworks. The potential exposure to crystalline silica in mining and tunnelling will vary depending on the geological formations worked.

In South Africa, silicosis frequently occurs in current and former miners, in foundry workers, ceramic, engineering and construction workers (Table 1). It is also often an

associated cause of death through its strong association with pulmonary tuberculosis.

There are no data as to how many cases of acute, accelerated, simple or complicated silicosis occur in South Africa, but the gold mining industry alone reports around 2 000 cases of pneumoconiosis per year.

Table 1 Non-mining cases of silicosis diagnosed by the NCOH Occupational Medicine Clinic 1991-1997

INDUSTRY	NUMBER	(%)
Foundry	65	(54)
Refractory	20	(17)
Pottery	15	(12)
Engineering	7	(6)
Construction	4	(3)
Chemical	4	(3)
Stone masonry	3	(2)
Stone crushing	2	(2)
Glass	1	(1)
TOTAL	121	(100)

3. HUMAN RESPIRATORY SYSTEM

Airborne silica dust enters the body primarily by inhalation. The inhaled air passes through the upper airways, the trachea, bronchi and smaller branches eventually reaching the bronchioles (or small airways) (Figure 1). The inhaled airborne dust particles are mainly exhaled or deposited in the upper airways and removed by the mucociliary escalator. Beyond the terminal (or respiratory) bronchioles are clusters of alveoli where oxygen and carbon dioxide exchanges occur. The walls of the alveolar air spaces are very thin and vulnerable to airborne substances. Small inhaled particles (<10 µm aerodynamic diameter) can be deposited in the area of respiratory bronchioles and in the alveoli. Within this region the dust particles are removed by the phagocytic cells called macrophages. The dust-laden macrophages can be removed from the lung by the mucociliary escalator or by the lymphatic system.

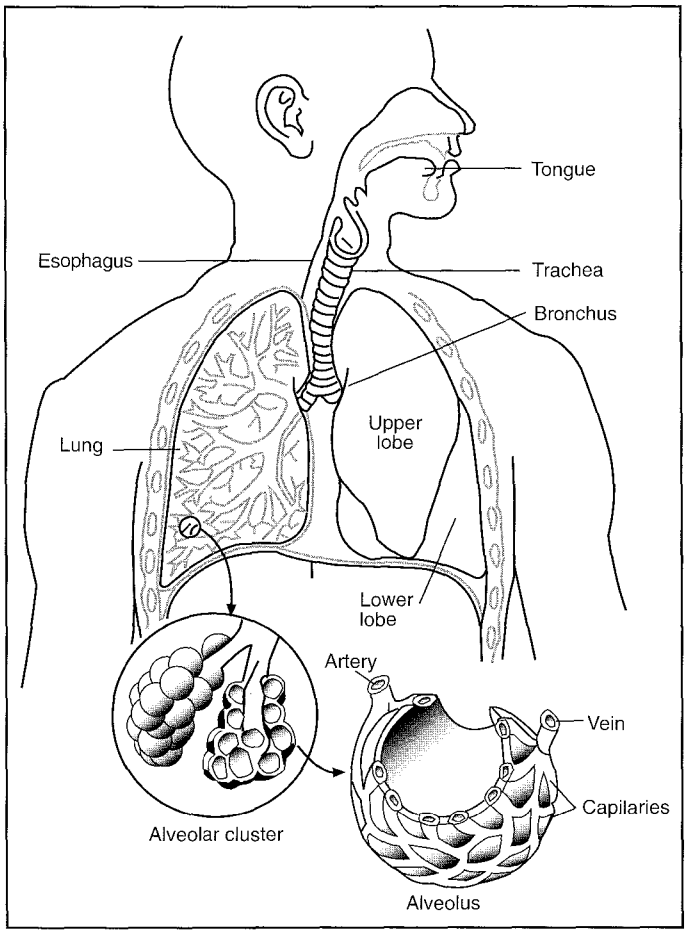

Figure 1 The Respiratory System

4. HEALTH EFFECTS ASSOCIATED WITH CRYSTALLINE SILICA DUST

The primary health concerns in subjects exposed to silica dust are the fibrogenic capacity of inhaled silica particles that can lead to the development of pneumoconiosis and the increased risk of tuberculosis. The term *pneumoconiosis* literally means 'dust in the lungs' and is defined by the ILO as 'the accumulation of dust in the lungs and the tissue reactions to its presence' [1]. The inhalation of silica dust over a long period of time and at sufficient concentrations can result in the formation of fibrotic lesions that form specific rounded fibrotic nodules. Three forms of silica-induced pneumoconiosis have been described - chronic, accelerated and acute.

4.1 Chronic silicosis. Chronic silicosis is the most common form of silicosis. In this form fibrotic changes in the lung occur after 10-30 years of inhalation of excessive levels of silica dust. Accumulated dust in the lungs can cause the fibrotic changes to continue to develop even after dust exposure has ceased. Chronic silicosis is further subdivided into simple and complicated silicosis.

Simple silicosis is the usual form of chronic silicosis and is characterised by the presence in the lung of discrete rounded fibrous nodules. Radiologically, these are seen as rounded opacities that are usually 3 - 6 mm in diameter. Workers with simple silicosis are usually without any respiratory symptoms or lung function impairment, unless they smoke or have coexistent disease. The fibrotic lesions in simple silicosis appear on the chest X-ray predominantly in the upper and middle lung zones as discrete small rounded opacities.

Complicated silicosis results when the silicotic nodules increase in size and coalesce into large lesions >1 cm in diameter. The large lesion is called progressive massive fibrosis. The conglomerate lesions may obliterate bronchi and vessels and cause marked distortion of lung structure and function. Symptoms of workers with complicated silicosis range from minimal complaints, which might include a chronic cough with phlegm production, to severe shortness of breath and rapidly occurring respiratory failure.

4.2 Accelerated silicosis. Accelerated silicosis results from the inhalation of very high concentrations of silica dust over a relatively short period, in the order of 5-10 years [2]. Although accelerated silicosis develops in a pattern similar to that of simple silicosis, with rounded nodular lesions in the upper lung zones, the time from initial exposure to the onset of disease is shorter and the progression to complicated silicosis is more rapid.

4.3 Acute silicosis. Acute silicosis develops from the inhalation of exceptionally high concentrations of crystalline silica over a short period (7 months to 5 years) [2]. The radiological appearance and the histopathological features are quite similar to those of pulmonary alveolar proteinosis. The radiological changes result from a filling of the air spaces by thick proteinaceous material (fluid and cells). Symptoms of acute silicosis include cough, weight loss, and fatigue. This may progress rapidly to respiratory failure over a period of several months. Death occurs after a few months. Acute silicosis has been reported among sand-blasters and drillers, and has historically been reported mainly among silica powder workers.

4.4 Silica dust and pulmonary tuberculosis. Silica particles can destroy or alter the metabolism of the pulmonary macrophage, thereby reducing its capacity for anti-bacterial defence. Occupational exposure to silica dust renders a subject susceptible to developing pulmonary tuberculosis. The risk of developing pulmonary tuberculosis while exposed, and also after exposure ends, depends on the amount of cumulative silica dust exposure [3, 4]. Furthermore, the presence of silicosis in the lung further increases the risk of developing pulmonary tuberculosis [5, 6]. The rate of tuberculosis in workers exposed to silica dust is also related to the rate of tuberculosis in the general population. The current HIV epidemic in South Africa will result in an increased incidence of tuberculosis in the general population, but more so in the silica dust exposed workers.

4.5 Silica dust and chronic obstructive lung disease. Destruction of alveolar walls in silica dust exposed subjects can lead to emphysema which is the main cause of chronic obstructive lung disease. Emphysema develops primarily in subjects who smoke, but silica dust exposure potentiates the damage done by smoking. Nonsmokers rarely develop emphysema due to the effect of silica dust only. Thus, smoking cessation is the most important preventive measure for chronic obstructive lung disease in silica dust exposed workers. Small airways disease specific to mineral dust, referred to as 'mineral dust airway disease' (MDAD) has also been described and results from fibrosis in the walls of small airways [7]. Patients with MDAD are reported to have impairment of lung function.

4.6 Silica dust and other health effects. In 1997, crystalline silica in the form of quartz or cristobalite was categorized as a human carcinogen by the International Agency for Research on Cancer (IARC) [8]. Lung cancer is the only cancer shown to be associated with silica dust exposure. Crystalline silica has been linked with cases of autoimmune diseases such as scleroderma, systemic lupus erythematosus (lupus), rheumatoid arthritis etc. Chronic renal disease, possibly due to immunological abnormalities, has also been linked with silica dust exposure.

5. MEDICAL SURVEILLANCE OF CRYSTALLINE SILICA EXPOSED WORKERS

All workers exposed to crystalline silica should participate in a respiratory surveillance programme, which should incorporate tuberculosis case-finding strategies. A description of a programme can be found in the *Guidelines: Monitoring Dust, Pneumoconiosis and Pulmonary Tuberculosis in South African Foundry Workers (NCOH, 1994).* Regulations for the surveillance of miners exposed to silica dust are pending in terms of the Mine Health and Safety Act, 1996, Department of Minerals and Energy. Silicosis and tuberculosis can manifest after exposure has ceased, therefore surveillance of significantly exposed workers should continue even after they have left the exposure industry.

The components of a surveillance programme and the frequency of surveillance should be determined by the level of risk (e.g. dustiness of the workplace, risk of tuberculosis) and the resources available. The basic components of a surveillance programme are:

- pre-placement exposure history and respiratory evaluation to establish baseline respiratory health. Respiratory symptoms and a full-sized chest radiograph is the minimum respiratory evaluation;

- training of workers in the symptoms of tuberculosis so that they can present themselves for medical evaluation when pertinent symptoms occur. Symptoms include a cough that lasts for two weeks or more, weight loss or coughing up blood;

- periodic full-sized chest x-rays classified into the *ILO Classification of Radiographs of the Pneumoconioses* (International Labour Office, 1981);

- spirometry (lung function testing) is generally recommended as part of a surveillance programme, but unless quality control is part of the programme, the results are likely to be uninterpretable;

- an exit chest radiograph, unless one has been done in the past 12 months.

As a general rule, evaluations should be done at pre-placement and then three yearly for workers with low dust exposure (respirable quartz < 0.1 mg/m^3), but yearly for workers with high dust exposure. Workers with exposure >10 years should be evaluated every two years, unless the programme does not detect cases of silicosis.
Workers with silicosis, whether exposed or not, should be evaluated yearly but a chest radiograph is not needed each year if respiratory symptoms are stable and features of tuberculosis are absent. At each evaluation the need to present on occurrence of symptoms suggestive of tuberculosis should be reinforced.

Recommendations on the frequency of evaluation of workers with past silica exposure but without silicosis are not well established, but an approach is to ensure that these workers know the symptoms of tuberculosis and to recommend chest radiography when convenient, or 5-yearly if symptoms are stable. (Note: there are specific provisions in the *Occupational Diseases in Mines and Works Act* for surveillance of former miners. The Director of the MBOD can advise on these). The programme should be audited for yield and adjusted accordingly. If no cases of silicosis or tuberculosis are detected through the programme, the frequency of evaluations can be reduced e.g. to 4 yearly for low exposure workers.

Table 2 Radiologic surveillance of workers exposed to silica dust

Category	Chest Radiography		
	Pre-placement	Periodic*	Exit [b]
1.Exposure conditions			
Low dust [a]	Yes	3-yearly	Yes
Low dust >10 years	Yes	Biennially	Yes
High dust	Yes	Annually	Yes
2. Workers with silicosis (assuming not exposed)	Yes	Biennially if symptoms stable (Medical evaluation annually)	Yes
3. Past exposure, but no current exposure and no silicosis	No, unless symptomatic or no radiograph within past 5 years	When convenient, or 5 yearly, or when symptoms change	No

* Frequency adjusted according to case yield.
[a] Respirable quartz below 0.1 mg/m^3.
[b] Exit radiograph necessary if none taken within the past 12 months.

6. OCCUPATIONAL EXPOSURE LIMITS FOR AIRBORNE CRYSTALLINE SILICA

6.1 Department of Labour Occupational Exposure Limits. *The Regulations for Hazardous Chemical Substances* under the Occupational Health and Safety Act, 1993, have assigned an 8-hour time weighted average (TWA) Occupational Exposure Limit - Control Limit (OEL-CL) for respirable crystalline silica of 0.4 mg/m^3. The Department of Labour OELs are largely based on the OELs published by the Health and Safety Executive in the U.K.

Under the Regulations for Hazardous Chemical Substances, those substances which have been assigned an OEL-CL are considered to be adequately controlled if exposures are as low as reasonably practicable below the OEL-CL. Excursions above the OEL-CL are only allowed if (i) they occur without a significant risk from exposure, (ii) the excursions are not indicative of a failure to maintain adequate control, (iii) during the excursions the area is temporarily demarcated as a respirator zone and (iv) the provisions of Regulation 11 regarding personal protective equipment and facilities are complied with.

Under Regulation 12 all control equipment and facilities provided are to be maintained in good working order and thorough examinations and tests of engineering control measures are required to be conducted by an Approved Inspection Authority (AIA) or by a person whose ability to do the measurements and tests is verified by an AIA. The examination/test intervals should not exceed 24 months.

Other forms of crystalline silica, tridymite and tripoli, are assigned an Occupational Exposure Limit - Recommended Limit (OEL-RL) of 0.4 mg/m^3 (as respirable dust).

6.2 Department of Minerals and Energy Occupational Exposure Limits. The Threshold Limit Values (TLVs) of the American Conference of Governmental Industrial Hygienists (ACGIH) are utilised as guidelines in the mining industry in South Africa. The ACGIH TLV-TWA for respirable quartz of 0.1 mg/m^3 and TLV-TWA for respirable cristobalite and tridymite of 0.05 mg/m^3 are incorporated in the Department of Minerals and Energy List of Threshold Limit Values *(Supporting Document No 2, Guidelines for the gravimetric sampling of airborne particulates for risk assessment in terms of the Occupational Diseases in Mines and Works Act No 78 of 1973).* Under the Mine Health and Safety Act, 1996, regulations are being developed which will include a Schedule containing occupational exposure limits.

The 1998 ACGIH TLVs Booklet lists the following TLV-TWA values for respirable crystalline silica:

Quartz	0.1 mg/m^3	(Adopted by ACGIH 1986)
Cristobalite and Tridymite	0.05 mg/m^3	(As above)
Tripoli (quartz content)	0.1 mg/m^3	(Adopted by ACGIH 1985)

7. MEASUREMENT OF EXPOSURE TO AIRBORNE CRYSTALLINE SILICA

Personal respirable dust sampling is required to measure worker exposures to airborne crystalline silica. A cyclone is generally used to separate the respirable fraction of the airborne particulate from the non-respirable fraction. By virtue of the cyclone design, respirable particles are carried onto the filter medium contained in the filter holder, whilst the larger particles drop into a grit pot. The crystalline silica content of the respirable dust collected can be determined by X-ray diffraction or infra red analysis.

In 1993 the international and European standards organisations, ISO and CEN, together with the ACGIH agreed on a new respirable dust convention to replace the diversity of sampling conventions then in place. A sampling flowrate of 2.2 litres per minute instead of 1.9 litres per minute is required with the Higgins-Dewell cyclone sampler for personal sampling when sampling to meet the ISO/CEN respirable dust convention (10 microns and below). At this time, no change is recommended by the ACGIH in the flow rate of 1.7 litres per minute utilised with the 10 mm nylon cyclone.

8. DUST CONTROL

The WHO, through their Prevention And Control Exchange Programme (PACE), are developing a number of documents on occupational hazard prevention and control. The WHO consider that there is a vast knowledge on hazard prevention and control which, if applied, could avoid most hazardous exposures. This is the case with silicosis which is a disease which is 100% preventable. The first document to be published under the PACE programme *Hazard Prevention and Control in the Work Environment: Dust Control* should be available in 1999. Videos to illustrate preventive principles applicable to dust control will also be available under the PACE programme.

Quotation from Alice Hamilton, the pioneer American physician and hygienist:

".....**obviously, the way to attack silicosis is to prevent the formation and escape of dust,**....."

A vast amount of literature exists on how to prevent silicosis and in recent years information has also become available through the Internet.

The US Occupational Safety and Health Administration (OSHA) *Silica Technical Advisor* provides on-line training on silica and silicosis prevention (Section 10. Internet Resources on Silica and Silicosis Prevention).

OSHA, the US Mine Safety and Health Administration (MSHA), and the US National Institute for Occupational Safety and Health (NIOSH) provide Internet sites with extensive information on Silica and Silicosis prevention. The document *A Guide to Working Safely with Silica* and NIOSH Alerts on silicosis including: *Preventing Silicosis and Deaths in Rock Drillers*, *Preventing Silicosis and Deaths from Sandblasting*, and *Preventing Silicosis and Deaths in Construction Workers*, can be down-loaded from the NIOSH web site.

8.1 The main control solutions for crystalline silica are:

- Use a silica substitute.
- Use appropriate engineering controls.
- Improve work practices.
- Use appropriate personal protective equipment.

8.2 Workers need to take the following steps to protect themselves from exposure to crystalline silica:

- Be aware of the health effects of respirable crystalline silica.
- Participate in any medical examinations, air monitoring or training programmes offered by your employer.
- Substitute less hazardous abrasive-blasting materials (e.g. steel grit or shot, aluminium oxide etc.) for those containing crystalline silica.
- Use engineering controls such as blast-cleaning machines, cabinets, dust collectors, wet methods and local exhaust ventilation to minimize exposure to silica dust.
- Always use the dust control systems and keep them well maintained.
- Tell your employer when the control measures are not working properly.
- Be aware that the highest silica dust concentrations may occur inside enclosed spaces during, for example: concrete sawing, masonry sawing or abrasive blasting.
- When abrasive blasting use a supplied-air helmet/hood respirator operated in continuous flow mode. Full face airline supplied respirators should be operated in pressure-demand or positive pressure mode. Use breathing quality air supply.
- Change into disposable or washable work clothes at the worksite or workplace.
- Do not eat, drink or use tobacco products in dusty areas.
- Wash hands and face before eating, drinking or smoking outside dusty areas.
- Shower and change into clean clothes before leaving the worksite or workplace.
- Ask for the results of any airborne dust monitoring conducted in your workplace.
- Talk to your employer, health and safety representative, employee representative or union if you are concerned about the dust in your workplace.

8.3 Key Questions to be asked to improve dust control include:

- Why do we do it this way?
- Do we really need to use this particular harmful substance? Can we eliminate the use of the substance? Is there a less dusty or toxic alternative?
- If we must use this substance, can we change its form so that it no longer produces as much dust? Can our suppliers provide raw materials in a less friable or dusty form?
- What options are available for controlling these releases by engineering methods?
- If dust is produced, can we enclose or automate the process?
- If release is inevitable, can we prevent release into the operator's breathing zone?
- Does the operator need to be close to the process? Can we move the operator away from the source of emission?
- Are control rooms or enclosed equipment cabs utilised to minimise exposures?
- Do other workers need to be in the area?
- Can we segregate the process?
- Are the existing controls working effectively?
- Do we need a dust control committee?
- How effective is our ventilation system?
- How effective is our personal protective equipment (PPE) programme, what does it cost, and are we supplying the correct respiratory protection?
- Are proper warning signs and labels utilised?
- Are the Material Safety Data Sheets (MSDS) adequate ?
- Is the workforce properly trained, involved and committed to dust control?
- Is the work area kept clean to reduce exposure by re-entrainment of settled dust?
- Is the required air monitoring and medical surveillance conducted?
- Is there proper managerial control over the workforce?
- Are senior management and the organisation committed to effective dust control?

9. COMPENSATION

Two Acts make provision for compensation for those workers with diseases arising from occupational exposure.

9.1 Occupational Diseases in Mines and Works Act (Act 78 of 1973) (ODMWA Amendment Act 1993). This Act provides for compensation for cardiorespiratory diseases, of which silicosis is one, arising from occupational exposure in mines and scheduled works. Compensation for employees is guaranteed without regard to apportionment of blame. The amendment of 1993 has authorised owners of mines and works to issue certificates of fitness to employees. The Certification Committee, based at the Medical Bureau for Occupational Diseases (MBOD), remains responsible for certifying people as being first or second degree disabled. Payment remains a lump sum payment, influenced by the job category of the miner or ex-miner. A spouse is eligible for lump sum payment of disease proven on post mortem irrespective of the cause of death.

9.1.1 Duties of the medical practitioner. Whenever a medical practitioner suspects an occupational cardiorespiratory disease in a person who has worked in a mine or works, the practitioner must communicate the findings to the Director of the MBOD. The Director may then request a full benefit examination by a medical practitioner of choice, not necessarily the reporting practitioner.

The cardiorespiratory organs of a deceased person who has ever worked in a mine or works, should be removed by the attending medical practitioner and sent to the NCOH in a red box (Details are available from the Pathology Directorate, NCOH).

9.2 Compensation for Occupational Injuries and Diseases Act (Act 130 of 1993) (COIDA). This Act provides for compensation for disablement caused by occupational diseases contracted by employees in the course of their employment, or for death resulting from such injuries or diseases; and to provide for matters connected with the above.

According to Scedule 3 of the Act, pneumonoconiosis-fibrosis of the parenchyma of the lung resulting from exposure to organic or inorganic fibrogenic dust, e.g. silicosis, is compensable.

Compensation is paid to successful claimants from a Compensation Fund that is financed by a levy paid by industry. The worker is compensated for the injury or disease, NOT for the loss of the job or the inability to continue a particular job.

9.2.1 Duties of the medical practitioner. Section 74(1) imposes a duty on a medical practitioner to furnish a medical report to the employer concerned, in the prescribed manner within 14 days of having diagnosed an occupational disease. At the request of the employee or the dependent of the employee, the practitioner must furnish a copy of the report. If the commissioner or the employer requires further medical reports regarding the employee, the medical practitioner who has, or is treating the employee must furnish the desired reports.

10. INTERNET RESOURCES ON SILICA AND SILICOSIS PREVENTION

Occupational Safety & Health Administration (OSHA), U.S. Department of Labour
Crystalline Silica
http://www.osha-slc.gov/SLTC/silicacrystalline/index.html
Silica Technical Advisor (On-line training and information)
http://www.osha-slc.gov/SLTC/silica_advisor/mainpage.html

Mine Safety and Health Administration (MSHA), U.S. Department of Labour
Silicosis Prevention
http://www.msha.gov/S&HINFO/SILICO/SILICO.HTM

National Institute for Occupational Safety and Health (NIOSH), U.S. Dept. of Health & Human Services
NIOSH Silicosis Prevention and Hotlinks to Silicosis Prevention
http://www.cdc.gov/niosh/silicpag.html
A Guide to Working Safely with Silica - If It's Silica, It's Not Just Dust
http://www.cdc.gov/niosh/pdfs/silicax.pdf

Canadian Centre for Occupational Health & Safety (CCOHS)
Quartz Silica
http://www.ccohs.ca/oshanswers/chemicals/chem_profiles/quartz_silica/quartz_silica.htm

Steel Structures Painting Council (SSPC), USA
Protecting Workers from Crystalline Silica Exposure
http://www.sspc.org/site/compliance/96_4/Silica.html

Workplace Safety and Health Division, Manitoba Department of Labour, Canada
Safe Sandblast Cleaning
http://www.gov.mb.ca/labour/safety/bulletins/bltn153.html

Michigan State University, USA
Abrasive Blasting Training Manuals (Training Manual and Instructors Manual)
http://www.chm.msu.edu/oem/index.htm

For further links visit The Best Sites page at ASOSH.ORG
http://www.asosh.org/WorldLinks/best_sites.htm

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APPENDIX

CONTACT DETAILS FOR FURTHER INFORMATION

Approved Inspection Authorities for the Monitoring of Chemical Stress Factors

Occupational Health and Safety, Department of Labour
Tel: (012) 309 4000
Fax: (012) 497 3000

General Information

National Centre for Occupational Health (NCOH), Department of Health
Tel: (011) 720 5734
Fax: (011) 720 6608
E-mail: info@ncoh.pwv.gov.za

Medical Bureau for Occupational Diseases (MBOD), Department of Health
Tel: (011) 403 6322
Fax: (011) 403 1346
E-mail: banyini@hltrsa2.pwv.gov.za

Compensation Commissioner, Department of Labour
Tel: (012) 319 9111
Fax: (012) 323 6986 / 326 7889
Web: http://www.wcomp.gov.za

Department of Minerals and Energy
Tel: (012) 317 9000
Fax: (012) 322 3416

Surveillance and Disease Reporting

To obtain comprehensive information on the extent of silicosis occurrence and the industries causing silicosis, SORDSA encourages the reporting of silicosis. To enhance the uniformity of reporting, SORDSA has issued guidelines which can be used by reporting physicians and other health care providers. For information about SORDSA contact:

Mrs Tonya Esterhuizen, Epidemiology and Surveillance Section
National Centre for Occupational Health (NCOH)
PO Box 4788, Johannesburg 2000
Tel/Fax: (011) 725-5978 E-mail: sordsa@ncoh.pwv.gov.za
Web: http://www.asosh.org/Programmes/SORDSA/Sordsa.htm

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