AIOH POSITION PAPER

Respirable Crystalline Silica and Occupational Health Issues

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AUTHORISATION

This position paper has been prepared by the AIOH Exposure Standards Committee and authorised by the AIOH Council.

President AIOH

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Australian Institute of Occupational Hygienists Inc (AIOH)

The Australian Institute of Occupational Hygienists Inc. (AIOH) is the association that represents professional occupational hygienists in Australia. Occupational hygiene is the science and art of anticipation, recognition, evaluation and control of hazards in the workplace and the environment. Occupational hygienists specialise in the assessment and control of:

- Chemical hazards (including dusts such as silica, carcinogens such as arsenic, fibrous dusts such as asbestos, gases such as chlorine, irritants such as ammonia and organic vapours such as petroleum hydrocarbons);
- Physical hazards (heat and cold, noise, vibration, ionising radiation, lasers, microwave radiation, radiofrequency radiation, ultra-violet light, visible light); and
- Biological hazards (bacteria, endotoxins, fungi, viruses, zoonoses).

Therefore the AIOH has a keen interest in the potential for workplace exposures to respirable crystalline silica (RCS), as its members are the professionals most likely to be asked to identify associated hazards and assess any exposure risks.

The Institute was formed in 1979 and incorporated in 1988. An elected governing Council, comprising the President, President Elect, Secretary, Treasurer and three Councillors, manages the affairs of the Institute. The AIOH is a member of the International Occupational Hygiene Association (IOHA).

The overall objective of the Institute is to help ensure that workplace health hazards are eliminated or controlled. It seeks to achieve this by:

- Promoting the profession of occupational hygiene in industry, government and the general community.
- Improving the practice of occupational hygiene and the knowledge, competence and standing of its practitioners.
- Providing a forum for the exchange of occupational hygiene information and ideas.
- Promoting the application of occupational hygiene principles to improve and maintain a safe and healthy working environment for all.
- Representing the profession nationally and internationally.

More information is available at our website – http://www.aioh.org.au

Consultation with AIOH Members

AIOH activities are managed through committees drawn from hygienists nationally. This position paper has been prepared by the Exposure Standards Committee, with comments sought from AIOH members generally and active consultation with particular members selected for their known interest and/or expertise in this area. Various AIOH members were contributors in the development of this position paper. Key contributors included: Gerard Tiernan and Kevin Hedges.

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List of Abbreviations and Acronyms

ACGIH	American Conference of Governmental Industrial Hygienists
AIOH	Australian Institute of Occupational Hygienists
ALARP	as low as reasonably practicable
AS/NZS	Australian New Zealand Standard
ASCC	Australian Safety and Compensation Council
BEI	biological exposure index
EPA	Environmental Protection Authority
ES	exposure standard
ES-TWA	exposure standard, time weighted average
HEPA	high efficiency particulate air filters
HSE	Health and Safety Executive (United Kingdom)
IARC	International Agency for Research on Cancer
IOM	Institute of Occupational Medicine
ISO	International Standards Organization
LEV	local exhaust ventilation
L	litre
LOAEL	lowest observed adverse effect level
m	metre
mg/m^3	milligrams (10^{-3} gm) per cubic metre
µ	micro-, (10^{-6}) as in micrometre
µg	microgram (10^{-6} gram)
mL	millilitre (10^{-3} litre)
NATA	National Association of Testing Authorities
NES	national exposure standard
NHMRC	National Health & Medical Research Council
NIOSH	National Institute for Occupational Safety and Health

NOAEL	no observable adverse effects level
NOHSC	National Occupational Health and Safety Commission
OHS	occupational health & safety
PAPR	powered air purifying respirator
PPE	personal protective equipment
ppm	parts per million (1 in 10^{-6})
ppb	parts per billion (1 in 10^{-9})
RCS	respirable crystalline silica
RPE	respiratory protection equipment
STEL	short term exposure limit
TLV	threshold limit value
TWA	time weighted average
WHO	World Health Organization

AIOH Position on Respirable Crystalline Silica and its Potential for Occupational Health Issues

Summary

Historically, respirable crystalline silica dust has been responsible for a large burden of occupational ill health, with countless deaths from silicosis, a disease which results in the formation of scar tissue in the lung. Media headlines often imply that silica is "the new asbestos". However examination of the data suggests otherwise. Silica has been under surveillance for many decades, and the morbidity and mortality of large populations of exposed individuals have also been studied over many decades. Worker's compensation statistics indicate there are very few new cases of silicosis arising from Australian industries.

It is acknowledged however that compensation statistics may not be a perfectly reliable source for determining the prevalence of silicosis in the workforce, or in industry sectors, as it is possible that many workers who suffer illness many years after exposure do not seek compensation.

There have been a number of occupational exposure studies that indicate crystalline silica is a potential human carcinogen. However these studies indicate there is little support for the hypothesis that occupational silica exposure is a direct acting cancer initiator, but there is compelling evidence that many forms of pulmonary fibrosis, including silicosis, constitute major risks for human lung cancer.

Therefore for occupational risk management purposes, the primary aim should be to protect against silicosis. In the absence of silicosis development, any increased risk of lung cancer, over and above background rates, should be negligible.

The AIOH supports the current ASCC (Australian Safety and Compensation Council) occupational exposure standard of 0.1 mg/m^3 for respirable crystalline silica. The principal reason for this position is the declining reported incidence of silicosis in Australia.

However it is becoming evident that there is not a substantiated "no observable adverse effects level" (NOAEL) at which it can be categorically stated that exposure to crystalline silica has no adverse health effects. The literature is demonstrating risks to health at levels previously considered as being acceptable. The determination of such a level may also be hampered by limitations in measurement technology which do not allow the measurement of very low level exposure.

There is an emerging trend within the occupational hygiene community to take a pragmatic approach to the measurement and control of exposures to toxic substances without attempting to define a dose response based exposure standard. Thus the AIOH acknowledges the importance of adhering to good control strategies so as to reduce exposures to "as low as reasonably practicable (ALARP)".

To this end the AIOH recommends that where there is a likelihood of 50% of the exposure standard being exceeded, control strategies and health surveillance should apply. To overcome limitations in analytical sensitivity for measurement of crystalline silica, near full shift monitoring and the use of a NATA registered laboratory is recommended.

What is Respirable Crystalline Silica?

Silica is silicon dioxide, one of the most abundant minerals in the earth's crust. It is present in almost all types of rock, sands, clays, shales and gravel. It is also a major constituent of construction materials such as bricks, tiles and concrete. Hence, silica is of great economic importance.

Silicon dioxide occurs in non-crystalline and in crystalline form. Crystalline silica is sometimes referred to as "free" silica. The main forms of crystalline silica are quartz, cristobalite and tridimite, the most prevalent of which is quartz. **Crystalline** silica is an aggressive, lung damaging dust when it is able to penetrate deep into the lung in sufficient quantity. The non-crystalline form of silica does not cause such lung damage. In order for the crystalline dust particles to reach the extremities of the lung where they have the potential to do damage, they must be particularly small (less than 10µm in diameter), and this size is defined as "**respirable**". Therefore we call the toxic form of this dust "**respirable crystalline silica**" or RCS.

How do we Measure it?

In order for RCS to present a risk to health it must be inhaled. Exposure is therefore assessed by measuring the air-borne concentration.

Air Monitoring

Currently the method used to sample airborne dust containing respirable crystalline silica is AS 2985 (2004) which follows ISO 7708:1995, Air quality – Particle size fraction definitions for health related sampling. According to AS 2985 (2004), respirable dust is the proportion of airborne particulate matter which, when inhaled, penetrates to the un-ciliated airways.

In Australia, analysis is typically carried out either by infrared spectroscopy or X-ray diffraction in accordance with the *NH&MRC method for the measurement of quartz in respirable dust by infrared spectroscopy and x-ray diffractometry (1984).*

Using competently operated modern analytical instruments and methodology, an 8-hour sampling period should provide an acceptable level of uncertainty at an RCS concentration of 0.05 mg/m^3. For a 4-hour air sample, results of 0.05 mg/m^3 may fall short of the standard required for legal proof if interference is present. For samples of 4 or more hours, the uncertainty is adequate for compliance monitoring and enforcement for concentrations of 0.1 mg/m^3 and above.

The AIOH strongly recommends near full shift sampling, that is an 8-hour sample period or 12-hour sample period for an 8 or 12 hour shift, respectively. The AIOH also strongly recommends use of a NATA accredited laboratory to do RCS analysis and that the results are reported on NATA test certificates.

Health Surveillance

Adverse health effects to personnel exposed to RCS can be assessed through health surveillance. Where there is long term potential for 50% of the exposure standard to be exceeded, health surveillance for crystalline silica should be carried out in accordance with "Guidelines for Health Surveillance" (NOHSC 1995).

Hazards Associated with Respirable Crystalline Silica

All forms of RCS of occupational relevance have the potential to cause silicosis, an irreversible and progressive condition in which healthy lung becomes replaced with areas of fibrosis. However human experience and experimental evidence both indicate that at specified levels of exposure, the potential to cause silicosis may be influenced by several factors. Occupational exposure to RCS also causes bronchogenic (lung) cancer but there is little support for the hypothesis that occupational silica exposure is a direct acting cancer initiator. There is however compelling evidence that many forms of pulmonary fibrosis, including silicosis, can lead to lung cancer. Silica exposure has also been associated with chronic obstructive pulmonary disease (COPD) and renal disease.

There are a number of factors which are thought to affect the potential for RCS to cause silicosis (HSE 2002). These are:

- *Polymorphic type of crystalline silica* – cristobalite, tridymite and quartz appear more reactive and more cytotoxic than coesite and shishovite.

- *The presence of other minerals* – Minerals containing aluminium may be found in close geological association with quartz. It has been found that the toxic effects of quartz are reduced in the presence of aluminium containing clay materials. However, there is evidence the protective effect of aluminium containing materials is not permanent, as the quartz dust may be "cleaned" in the lungs, and this eventually begins to express its pathogenic properties.

- *The particle number, size and surface area* – Current knowledge suggests that regardless of the type of dust, the total surface area of the dust retained in the lungs is an important determinant of toxicity. Surface area is related to particle size; smaller particles possess a much larger surface area than larger particles. Hence, smaller particle size fractions (very fine dusts) of RCS would be expected to produce more lung damage than equal masses of larger respirable size fractions.

- *Freshly fractured and "aged" surfaces* – Cleavage of crystalline silica particles into smaller fragments results in the formation of reactive radical species at the newly generated particle surfaces. This leads to an increase in cytotoxicity. Freshly generated surfaces may be generated in processes such as sand-blasting. However, the activity of the free radicals decays with time, a process known as 'aging'. This occurs slowly in air, but rapidly (within minutes) in water.

Silicosis

Silicosis is a fibrotic lung disease caused by the inhalation of RCS. It has been described as chronic silicosis, accelerated silicosis and acute silicosis.

Chronic silicosis (including simple and complicated silicosis), is the most common form, and results in fibrotic changes to the lungs after 10 to 30 years of exposure.

Simple silicosis, the usual form of chronic silicosis, is characterised by the presence of discrete rounded fibrous nodules in the lung. On the X-ray these are seen as 3 – 6 mm discrete rounded opacities that appear predominantly in the upper and middle lung zones. Respiratory symptoms or lung function impairment may not be observed unless the person smokes or has coexistent disease.

Complicated silicosis results when the silicotic nodules increase in size and coalesce into large lesions greater than 1 cm in diameter. The conglomerate lesions may obliterate bronchi and vessels and cause marked distortion of lung structure and function. The disease results in progressive massive fibrosis (PMF). When progressive massive fibrosis occurs, the patient develops progressive respiratory symptoms from reduction in lung volume, distortion of bronchi, and bullous emphysema. The main symptom is shortness of breath, which is progressive and ultimately disabling, potentially leading to cardio respiratory failure.

Accelerated silicosis results from the inhalation of very high concentrations of silica dust over a period typically in the order of 5 to 10 years. Although accelerated silicosis develops in a pattern similar to that of simple silicosis, the time from initial exposure to the onset of disease is shorter and the progression to complicated silicosis is more rapid.

Acute silicosis develops from the inhalation of high concentrations of RCS over a short period (7 months to 5 years). The air spaces fill with 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.

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 cumulative amount of silica dust exposure. Furthermore, the presence of silicosis in the lung further increases the risk of developing pulmonary tuberculosis. The rate of tuberculosis in workers exposed to silica dust is also related to the rate of tuberculosis in the general population (SORDSA, 1999).

Chronic obstructive pulmonary disease (COPD)

The literature is showing an increased weight of evidence regarding exposure to RCS causing COPD (Hnizdo & Vallyathan 2002). COPD is known by a number of names including chronic obstructive pulmonary disease (COPD), chronic obstructive airway disease (COAD), chronic airflow obstruction (CAO) and chronic airway limitation (CAL). It is also referred to as chronic bronchitis and emphysema. Chronic obstructive pulmonary disease does not include asthma in which the airflow obstruction is largely reversible. Destruction of alveolar walls in silica dust exposed subjects can lead to emphysema which is the main cause of chronic obstructive pulmonary disease (COPD).

Lung Cancer

In 1997, a monograph published by the International Agency for Research on Cancer (IARC) concluded that there is now sufficient evidence in humans for the carcinogenicity of inhaled crystalline silica in the form of quartz or cristobalite from occupational sources (IARC 1997). Several studies among the many reviewed by the IARC working group on the question of silica exposure and cancer risk in humans were negative or equivocal, and carcinogenicity of silica was not detected in all industrial operations. However, nine studies showed an excessive risk for lung cancer. These included refractory brick workers, pottery workers, diatomaceous earth workers, foundry workers, granite workers, and mine workers, (although not coal-mine workers). Increased lung cancer risk appears to be found only in those with silicosis.

Renal disease

Increased risk of renal disease has been implicated with elevated exposures to crystalline silica. A recent US study found a doubling of risk of non-malignant renal disease but no increase in renal cancer (McDonald *et al*, 2005).

Major Uses / Potential for Exposure

Workplaces where RCS is known to be present include:

- Mining
- Quarrying
- Exploration
- Foundries
- Ceramics
- Brick manufacture and heavy clay
- Industrial minerals and the production and use of silica sand and flour
- Construction
- Stonemasonry

In the past silica sand has been used for sandblasting however this resulted in a large number of acute and subacute cases of silicosis (ACGIH®, 2001). In Australia, the use or handling of a substance that consists of, or contains, crystalline silicon dioxide as an abrasive material in abrasive blasting, is prohibited under occupational safety and health regulations.

A significant proportion of the Australian working population are employed in these industries (NOHSC 1993). The Minerals Council of Australia has reported 127,000 people directly employed in the Mining Industry and 200,000 indirectly employed (Source: http://www.minerals.org.au/). According to the Department of Industry Tourism and Resources, between 2002 and 2003 there were approximately 730,000 people employed in the construction industry which is 7% of the workforce (Source: http://www.industry.gov.au/).

High RCS exposures can occur wherever crystalline silica-containing material is drilled, blasted, crushed, sieved or otherwise disturbed to release respirable dust particles into the atmosphere.

Levels of exposure in Australian industry have been estimated to range between 0.01 and 0.8 mg/m^3, the mean exposure level being 0.094 mg/m^3. In the working population at risk, 77% were estimated to be currently exposed to concentrations less than or equal to 0.1 mg/m^3, 90% to concentrations less than or equal to 0.2 mg/m^3, and 10% to concentrations exceeding 0.2 mg/m^3 (NOHSC, 1993). Actual measurements in NSW longwall underground coal mines (likely worst-case exposure group) suggest this to be an over estimate. A limit of 0.15 mg/m^3 was exceeded in only 1.4 to 9.3% of samples (Cram, 2003).

Risk of Health Effects

Australia

Silicosis

The National Occupational Health & Safety Commission (NOHSC) investigated the efficacy of the then current occupational exposure standard, legislative aspects and control strategies for silica (NOHSC, 1993). A review of the state by state silicosis records indicated probably less than 20-30 new cases per year and the generality that these cases arose from uncontrolled exposure situations (ie industries and occupations where there was minimal or negligible

adherence to the legislative exposure standard and control requirements). In Western Australia, worker surveillance, such as that for Kalgoorlie miners, showed less than 5 new cases of silicosis per year across the state. None of these cases commenced employment since 1974 when the exposure standard of 0.2 mg/m^3 was introduced (Wan & Lee, 1993). A review of the medical surveillance records from Broken Hill workers was presented to Worksafe Australia as proof that the implementation of the regulation level of 0.2 mg/m^3 RCS had proven to be more than adequate in preventing silicosis in the mine workforce (Submission by the Chamber of Mines, Metals & Extractive Industries NSW, 21 December 1988 and letter from Department of Mineral Resources 29 August 1991).

Lung Cancer

An examination of silicosis and lung cancer risk was carried out, based on NSW Dust Diseases Board data (Berry *et al*, 2002 & 2004). Detailed examination of the various occupations and industries associated with 1447 silicosis cases was undertaken. Long term exposure to high levels of RCS was associated with increase in lung cancer risk. A detailed examination of the various risk factors indicates that tobacco smoking contributes a higher risk component and hence the majority of the case numbers. However after allowing for tobacco smoking, there is nearly a doubling of lung cancer risk in compensable cases for silicosis (X-ray evidence, decreased lung function and disability) which is observed across most industries and occupations. The level of lung cancer risk is in line with that reported from other international studies.

Significant risk of lung cancer (SMR 1.6) was found in WA gold miners who had developed silicosis, however no evidence was found of an increased lung cancer risk due to silica exposure in the absence of silicosis (de Klerk et al 1998).

Clinical silicosis is now a rarity, and elevated risk of lung cancer appears to be confined to cases where the RCS exposure is of such a level that it results in clinical silicosis. Based on the number (say 10-30) of new cases of silicosis, this would amount to 1 or 2 additional lung cancer cases per year across Australia. Removing the smoking component from airways disease and the reduced contemporary silica dust exposures would mean few additional cases of airways disease per year in Australia.

In a report to NOHSC *de Klerk et al* (2002*)* proposed that an exposure standard of 0.13 mg/m^3 of RCS would keep the risk of excess annual lung cancer below 1 per 10,000 per year after 40 years of exposure and that it was likely to be around 1 per 100,000 per year or less. A risk level of higher than 1 per 10,000 per year is considered unacceptable and a risk level of lower than 1 per 100,000 per year is considered acceptable.

USA

The American Thoracic Society (ATS) produced a position statement outlining the effects of exposure to RCS and indicated lung cancer as an associated outcome of exposure (ATS, 1997). The ATS concluded the following:

- The available data support the conclusion that silicosis produces increased risk for bronchiogenic carcinoma.
- Less information is available for lung cancer risk among silicotics who never smoked and workers who were exposed to silica but did not have silicosis.
- Whether silica exposure is associated with lung cancer in the absence of silicosis is less clear.

NIOSH also reviewed the studies considered by IARC and ATS and concurred with the conclusions and recommended that RCS be considered a potential occupational carcinogen (NIOSH, 2002).

There remains ongoing debate in the scientific community about the carcinogenicity of RCS. Hessel *et al* (2000) were critical of the IARC monograph, believing the results of the studies to be inconsistent and, when positive, only weakly positive. Other methodologically strong negative studies have not been considered and several studies viewed as evidence supporting carcinogenicity of silica have significant methodological weaknesses.

ACGIH (2001) classified crystalline quartz silica as an A2 suspected human carcinogen. This was on the basis that although there was little support for the hypothesis that occupational silica exposure is a direct acting initiator, there was compelling evidence that many forms of pulmonary fibrosis constitute major risks for human lung cancer. They concluded from their assessment that control of worker exposure to avoid silicosis would also prevent silica associated lung cancer.

The ACGIH have based their exposure standard on the prevention of fibrosis and the UK HSE has followed a similar approach. The ACGIH have significantly reduced their exposure standard (TLV) by a factor of 4, from the previous value of 0.1 mg/m^3 to 0.025 mg/m^3. They state that fibrosis undetected by chest X-ray probably does occur in workers exposed at levels near the 0.1 mg/m^3 level.

United Kingdom (UK)

In the UK, a review (HSE, 2002) by the Health and Safety Executive (HSE) revealed unacceptable silicosis risks for workers exposed to RCS at the workplace exposure limit (WEL) which, at that time, was 0.3 mg/m^3. In fact, the HSE cited a study that indicated a 20% risk for developing silicosis at this limit. The review concluded that RCS is only weakly carcinogenic (HSE, 2002).

In 2002 the European Scientific Committee on Occupational Exposure Limits (SCOEL), made a recommendation to the European Commission. SCOEL noted that to reduce the incidence of silicosis, the Occupational Exposure Limit (OEL), would have to be set below 0.05 mg/m^3 (SCOEL 2002). This recommendation challenged the adequacy of the UK WEL and the HSE therefore considered it prudent that they develop a more stringent regulatory position on RCS. The HSE risk estimates were influenced by a study involving hundreds of workers from a Scottish coalmine that indicate that there is some risk of developing silicosis when exposed at levels of RCS of 0.02mg/m^3 (0.25% risk), 0.04 mg/m^3 (0.5% risk) and 0.1 mg/m^3 (2.5% risk). A regulatory impact assessment was carried out in 2005, looking at a cost benefit analysis of four potential WEL values: 0.3 mg/m^3 (the UK Maximum Exposure Limit in 2005), 0.1 mg/m^3, 0.05 mg/m^3 and 0.01 mg/m^3. This analysis resulted in the revised UK WEL for RCS being set, in 2006, as 0.1 mg/m^3 (ACTS, 2006).

Controls

Given the ubiquitous nature of crystalline silica, elimination or substitution as control measures are generally impractical due to its ubiquitous nature. For similar reasons the option of a legislative ban on crystalline silica, as has been done with asbestos, is not practical and in addition is not warranted by the health risk. This leaves engineering controls, administrative controls and the use of PPE as the means of reducing exposures. The practicality and costs of implementing such controls needs to be more rigorously determined and balanced against the expected gain in health benefits. Research indicates that these control measures have been effective. The historical reduction in silicosis numbers is due to a combination of regular

medical surveillance, reduction in exposures such as compliance with a regulatory exposure standard, the prohibition of specific tasks associated with high risk (such as sand blasting and the use of silica flour in foundry operations) and the use of adequate dust suppression systems such as ventilation and wetting down.

Wherever the bulk material contains crystalline silica **and** there is potential for RCS to be generated good practice guidance should be followed including air monitoring and health surveillance.

The control principles that apply to RCS are similar to those that apply to all mechanically generated dust exposures.

- Design and operate processes and activities to minimize emission, release and spread of dust;
- Position personnel so they are out of the dust either in enclosed and filtered cabins or so they are working upwind of dust emission;
- Use sharp cutting tools that minimise the generation of large quantities of fine dust;
- Use wet processes to prevent dust generation;
- Use water suppression to prevent dust spread;
- Ensure ore passes are not emptied below the brow point and crusher chutes are kept full;
- Use water curtains and rubber curtains to prevent dust release, particularly at conveyor transfer points and chute draw points;
- Use ventilation, either dilution or extraction, to control dust spread and dust release;
- Ensure suppressed dust is captured by scrubbing or filtering so it cannot be re-entrained in workplace air;
- Apply good house-keeping practices to prevent dust build-up;
- Provide training in the health effects of dust and its control;
- Where adequate control of exposure cannot be achieved by other means, provide, in combination with other control measures, suitable PPE. For most exposures to RCS this will be a P1 or P2 efficiency half face respirator. Ensure training is provided in the use and limitations of respiratory protective equipment (eg have a clean shaven policy). Face fit testing is also recommended, as per AS 1715 (1994).

There is a Good Practices Guide available from http://www.nepsi.eu/ containing more than 50 different task sheets that include controls for respirable crystalline silica generation.

Current Applicable Legislation and Standards

The current Australian Safety and Compensation Council (ASCC) exposure standard for RCS is 0.1 mg/m^3. The majority of Australian states have adopted this into their regulations.

An exception is the NSW Coal Mining Regulation 2006, which has prescribed the limit for quartz-containing dust at 0.12 mg/m^3 of respirable quartz for underground coal mines. Open cut coal mines are required to meet 0.1 mg/m^3. The different standard relates to different sampling regimes used in NSW underground coal mines.

The AIOH consider it prudent to apply the ASCC occupational exposure standard (TWA) of 0.1 mg/m^3 to all industries until the research demonstrates otherwise.

AIOH Recommendations

The profile of Australia's silica exposed populations is not well documented. Some workplaces may be far from compliant either through a lack of regulatory enforcement or

simply through a lack of awareness. Industry and government monitoring resources are probably too few to readily reveal the extent of exposure. The lack of exposure data is probably more important for workers such as those in construction. It is in such industries that a combination of small amounts of increased education and enforcement may produce greatly enhanced benefits.

There is a degree of uncertainty about exposure and potential long term health effects, and therefore it is prudent that Australia continues to monitor and reduce RCS exposures. Hence the AIOH maintains that it is important to adhere to good control strategies so as to reduce exposures to "as low as reasonably practicable (ALARP)".

The AIOH recommends that where there is a continued likelihood of 50% of the exposure standard being exceeded, exposure monitoring and health surveillance should apply. To overcome limitations in analytical sensitivity, full shift monitoring and the use of a NATA registered laboratory is recommended.

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