This document is to provide an answer to the question asked in the Transcript - Committee Inquiry - 2019 Review of the Dust Diseases Scheme - Hearing 20 September 2019 - Mr. Michael Shearer:

Question 1:

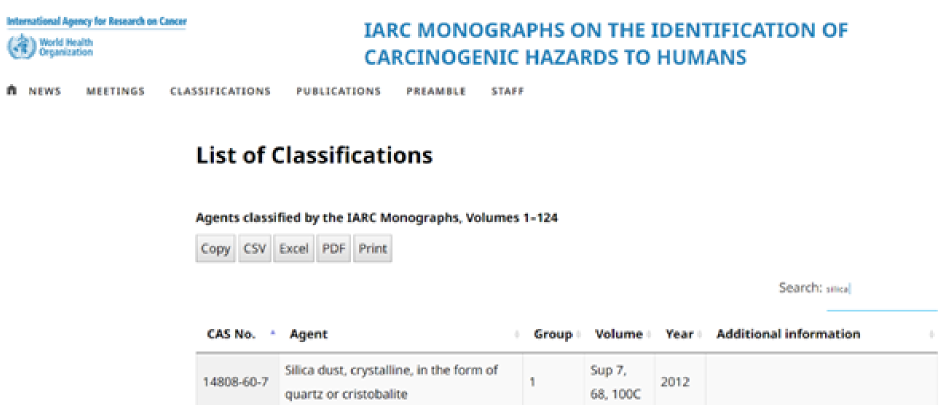
The Hon. ANTHONY D'ADAM: Thank you. In terms of substitution, which is the next recommendation you consider, you talk about the impact of silica—the prevalence of silica—and therefore the impossibility of it being eliminated. The question of silica is still the impact. We are dealing with the manufactured stone industry, which is the focus of this inquiry. On the evidence that we have received so far, it is not necessarily silica alone that is the driver of the silicosis arising from the manufactured stone industry. We have heard evidence that it may be something to do with the interaction between the silica and the polymers that are used in the manufacture of manufactured stone products. Do you have medical expertise? Are you able to provide some expert evidence on the impact of silica and its causative effects in terms of silicosis?

Answer 1:

Crystalline silica causes formation of scar tissue, thus reducing the lungs' ability to take in oxygen which is known as silicosis. There is no cure for silicosis.

To address the question of, "Are you able to provide some expert evidence on the impact of silica and its causative effects in terms of silicosis?" I provide the following response: The AIOH position paper *(Respirable Crystalline Silica and Occupational Health Issues | December 2018)* provides numerous references supporting the fact that breathing in respirable crystalline silica at concentrations above the Workplace Exposure Standard can cause silicosis.

There is an array of research material on the internet which provides evidence and explains causative effects. One such article and probably the most compelling is International Agency for Research on Cancer (IARC) Monograph 68. Please refer to page 210 section 5.5 paragraph 1 as evidence that exposure to crystalline silica in the form of quartz or cristobalite can cause adverse health effects. Below is a snippet of the group classification of silica dust in the form of quartz or cristobalite as per World Health Organisation (WHO).


International Agency for Research on Cancer
World Health Organization

IARC MONOGRAPHS ON THE IDENTIFICATION OF CARCINOGENIC HAZARDS TO HUMANS

NEWS MEETINGS CLASSIFICATIONS PUBLICATIONS PREAMBLE STAFF

List of Classifications

Agents classified by the IARC Monographs, Volumes 1–124

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Search: silica

CAS No.	Agent	Group	Volume	Year	Additional information
14808-60-7	Silica dust, crystalline, in the form of quartz or cristobalite	1	Sup 7, 68, 100C	2012	

Question 2:

Mr SHEARER: Personally I am not an occupational hygienist but I will take that on notice because one of my colleagues who helped put the submission together is an occupational hygienist—so he would be better equipped to answer that question for you. It is more around the potential for ill effects.

The Hon. ANTHONY D'ADAM: We have not had the same experience from the evidence so far, of the impact of cutting in the use of Sydney sandstone, for example. It seems to be something about the manufactured stone product that is driving a higher incidence of silicosis.

Mr SHEARER: That may also be driven by the lack of understanding or the lack of controls put in place in that particular industry, as well. That may be a catalyst. With those specific instances it could be other health and environmental issues, as well

Answer 2:

The committee should not assume that due to the lack of evidence of cases of silicosis from cutting into Sydney sandstone, that the components of manufactured stone are necessarily more toxic. Such evidence does not exist due to the lack of mandatory notification of diseases such as silicosis in addition to the fact that the current system relies on workers to come forward. This system is flawed, and is strongly recommended to be rectified by this committee by the establishment of mandatory health monitoring and disease notification in all at-risk industries.

Question 3:

The Hon. ANTHONY D'ADAM: My specific question is: Do you have knowledge of a specific engineered solution to that problem of cutting onsite? I am happy for you to take that on notice if it is not something you feel able to consider today.

Mr SHEARER: I will be able to provide you with more evidence. I will take it on notice. There are different types of equipment that are available out there in the marketplace. As I mentioned, it is all going to be task-specific and the controls around should be that people actually take the time to scope out exactly what they need to do, how they need to do it and what equipment they actually need to provide to ensure that everyone in that workspace is safe

Answer 3:

It is recommended that the committee refer to the recent publication by Workplace Health and Safety Queensland, *"Managing respirable crystalline silica dust exposure in the stone benchtop industry"* which contains references to numerous engineering solutions such as water suppression and on-tool extraction. Another useful resource is the *Managers Toolkit – Engineered Stone Silica Dust* available from www.breathefreelyaustralia.org.au/

General feedback:

Statement 1:

I want to refer to page 6 where;

The Hon. ANTHONY D'ADAM: In terms of manufactured stone there are other substitute products—you can use granite, you can use wooden benchtops—there are other viable substitutions for this product.

Feedback 1:

It must be noted that granite comprises of approximately 20% and 60% quartz by volume and would not necessarily eliminate the hazard. It could possibly reduce the level of exposure.

Statement 2:

The Hon. ANTHONY D'ADAM: Was the submission informed by economic analysis? Were there economists involved in the preparation of the submission?

Mr SHEARER: No, it was a collective submission from our society—from our committee.

The Hon. ANTHONY D'ADAM: In terms of expertise, you are not in a position to make comment about the economic impact of elimination as a strategy, are you?

Feedback 2:

To specifically answer the question related to economic feedback. There was no relative information given in the brief, so one was never going to be able to provide the relative answers with insufficient statistics.

I personally am not a practising economist, but however, I do have the skill set which allows me to provide an economical assessment on the potential or relative impact based on a specific business model based on a sensitivity analysis whether it be in real or nominal dollars.

I did study economics in high school, then I studied advanced accountancy, I have run my own small business for almost 20 years and have employed or sub-contracted employees. I studied as a mature student at UNSW whilst working 50+ hours per week. I hold a Master's Degree in Mine Engineering in which mineral economics and financial risk analysis were key features, this also included micro economics of working in developing countries. I also hold a Graduate Diploma in Underground Mine Ventilation in which I needed to learn about the mechanics to provide a healthy atmosphere for all underground workers that I have a duty of care too. This includes the controls required to lower exposures to the workforce whether it be by dust, gas, heat or emissions.

I currently look after the temporary ventilation systems for 3 major infrastructure projects under construction in Sydney worth approx. 10 billion dollars. I am required to make economic decisions constantly whilst ensuring that the safety and livelihood of our personnel is not compromised. This is the proactive approach that we adopt and safety is put first.

The collective pool of colleagues that make up the membership and committee of the MVSA are from many walks of life. These include Academia, Ventilation Practitioners, Occupational Hygienists, Government Inspectorates, Consultants and Suppliers who are from both Australia and Overseas. Due to our tight network of colleagues, we have solid relationships with other groups nationally and internationally which helps us to be aware of current standards or potential changes or improvements.

I believe that the regulatory bodies should rewrite their legislation to include the introduction of statutory appointments of both Ventilation Engineers and Occupational Hygienists similar to the Coal mining industry. This would allow an auditing process to commence and provide the leverage that is somewhat lacking at times. This would give organisations a distinct line that is required as a minimum to ensure that they are demonstrating their duty of care. Some of the current guidelines are suggestive, and industry best practice design criteria should be adopted as the minimum standard. This would be a positive step in the right direction to minimising the exposure to all workforces.

Kind Regards

Michael Shearer

President

Mine Ventilation Society of Australia

Respirable Crystalline Silica and Occupational Health Issues

Position Paper

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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.

EXPOSURE STANDARDS COMMITTEE MISSION STATEMENT

The AIOH established the Exposure Standards Committee to provide expert guidance and comment to the exposure standards setting process at a State and National level and internationally where appropriate, through development of AIOH Position Papers, AIOH guidance publications or comment on relevant Standards, Regulations and Codes of Practice. The Committee's remit is to confirm that the exposure standards numbers, and Standards and Codes of Practice, are changed for valid occupational hygiene and scientific reasons.

STATEMENT OF POSITION REGARDING AIOH POSITION PAPERS

The AIOH is not a standards setting body. Through its Position Papers, the AIOH seeks to provide relevant information on substances of interest where there is uncertainty about existing Australian exposure standards. This is done primarily through a review of the existing published, peer-reviewed scientific literature but may include anecdotal evidence based on the practical experience of certified AIOH members. The Position Papers attempt to recommend a health-based guidance exposure value that can be measured; that is, it is technically feasible to assess workplace exposures against the derived exposure value. It does not consider economic or engineering feasibility. As far as reasonably possible, the AIOH formulates a recommendation on the level of exposure that the typical worker can experience without significant risk of adverse health effects.

Any recommended guidance exposure value should not be viewed as a fine line between safe and unsafe exposures. They also do not represent quantitative estimates of risk at different exposure levels or by different routes of exposure. Any recommended exposure value should be used as a guideline by professionals trained in the practice of occupational hygiene to assist in the control of health hazards.

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 to this update included: Ian Firth, Alan Rogers, Rob Golec, Linda Apthorpe and Laurie Glossop.

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List of Abbreviations and Acronyms

ACGIH®	American Conference of Governmental Industrial Hygienists
ACTS	Advisory Committee on Toxic Substances (UK)
AIOH	Australian Institute of Occupational Hygienists
ALARP	As low as reasonably practicable
AS/NZS	Australian / New Zealand Standard
ATS	American Thoracic Society
CAL	Chronic airway limitation
CAO	Chronic airflow obstruction
CEN	European Committee for Standardization
cm	centimetre
COAD	Chronic obstructive airways disease
COH®	Certified Occupational Hygienist®
COPD	Chronic obstructive pulmonary disease
DNRME	Department of Natural Resources, Mining and Energy (QLD)
EAD	Equivalent aerodynamic diameter
FTIR	Fourier Transform Infrared
FVC	Forced vital capacity
FEV_1	Forced expiratory volume in 1 second
GM	Geometric mean
HSE	Health and Safety Executive (United Kingdom)
IARC	International Agency for Research on Cancer
ILO	International Labour Organization
IOM	Institute of Occupational Medicine
ISO	International Standards Organization
L	litre
LOD	Limit of detection
LOQ	Limit of quantitation
mm	millimetre
mg/m^3	milligrams (10^{-3} g) per cubic metre
µg	micro gram
µm	micro-, (10^{-6}) as in micrometre (µm)
NATA	National Association of Testing Authorities
nepsi.eu	The European network on Silica
NHMRC	National Health & Medical Research Council
NIOSH	National Institute for Occupational Safety and Health
NOAEL	No Observed Adverse Effect Level
NOHSC	National Occupational Health and Safety Commission
NSW	New South Wales
NTP	National Toxicology Program (US)
OEL	Occupational Exposure Limit

OSHA	Occupational Safety and Health Administration
Pa	pascal
PAHs	Polycyclic aromatic hydrocarbons
PAPR	Powered air purifying respirator
PEL	Permissible Exposure Limit
PMF	Progressive massive fibrosis
PPE	Personal protective equipment
%	Percent
QLD	Queensland
RCS	Respirable crystalline silica
RR	Relative risk
SABRE	Surveillance of Australian workplace Based Respiratory Events
SCOEL	Scientific Committee on Occupational Exposure Limits (EC)
SMR	Standardised mortality ratio
SWA	Safe Work Australia
TLV	Threshold Limit Value
TWA	Time Weighted Average
UK	United Kingdom
US / USA	United States of America
VIC	Victoria
WA	West Australia
WEL	Workplace Exposure Limit
WES	Workplace Exposure Standard
XRD	X-ray Diffraction

Definitions

- Crystalline: Refers to the orientation of silicon dioxide (SiO_2) molecules in a fixed pattern as opposed to a nonperiodic, random molecular arrangement, defined as amorphous.
- Equivalent aerodynamic diameter (EAD): The diameter of a spherical particle of density 1000 kg/m^3 which exhibits the same aerodynamic behaviour as the particle in question.
- Hazard: Means potential to cause harm.
- Limit of detection (LOD): The lowest concentration of a substance that can be feasibly determined to be statistically different (e.g. 3 x the standard deviation) from a sample that contains none of the substance (i.e. a blank sample).
- Limit of quantitation (LOQ): The lowest concentration of a substance that can be reliably and consistently detected and measured, taking into account bias and imprecision – it is the level above which quantitative results may be obtained with a specific degree of confidence.
- Respirable crystalline silica (RCS): The respirable dust fraction of the main forms of crystalline silica; that is, α-quartz, cristobalite and tridymite.
- Risk: Means the probability of harm actually occurring.

AIOH Position on respirable crystalline silica and its potential for Occupational Health Issues

Key messages

- Breathing in respirable crystalline silica (RCS) for many months or years at concentrations above the workplace exposure standard (WES) can cause silicosis, a disease which results in the formation of scar tissue in the lung resulting in loss of lung function, and is associated with an increased risk of lung cancer. RCS may also be related to the development of other diseases such as chronic obstructive pulmonary disease (COPD), kidney disease and autoimmune disorders.
- A number of factors are thought to affect the potential for RCS to cause silicosis and there are different types of silicosis (chronic, accelerated and acute) caused by different RCS levels and periods of exposure.
- Information on new cases of silicosis occurring in Australian industries is limited to results from medical surveillance in only a small number of industries along with determining the number of compensated cases in each state and territory, which does not provide the full extent of cases. The AIOH recommends the development of a centralised Australian register for the reporting of dust-related lung disease.
- It appears that reported new cases are mainly either due to historic poorly controlled long-term exposures or to contemporary acute uncontrolled exposure situations likely to have been above the current exposure limit. The AIOH is most concerned by the emergence of these new cases and the absence of widespread control measures in many industries, particularly with the use of hand-held high speed cutting and grinding tools on silica-containing materials.
- The AIOH believes that most exposures to RCS maybe adequately controlled by conventional means such as use of wet methods to suppress dusts, local exhaust ventilation, use of positive pressure cabins and segregation of workers from areas of high concentration, as published by various authorities. Controls must be maintained and verified that they are adequately reducing exposure to RCS.
- The AIOH recommends limiting worker exposure to RCS to as low as reasonably practicable (ALARP) to be at all times below an 8-hour time weighted average (TWA) guidance exposure value of 0.1 milligram (mg) respirable fraction in each cubic metre (m^3) of air. In addition, a TWA value of 0.05 mg/m^3 should be applied as an action level which triggers investigation of the sources of exposure and implementation of suitable control strategies as well as health surveillance. When assessing the degree of compliance to this guidance exposure value according to contemporary occupational hygiene assessment and statistical practice, the actual long-term average exposure is often less than 0.05 mg/m^3.
- There are limitations in measurement technology which restrict the accurate measurement of very low-level exposure below 0.05 mg/m^3. Therefore, the AIOH recommends near full-shift monitoring and sample analysis by a laboratory accredited by the National Association of Testing Authorities (NATA), applying standardised analysis and reporting methods.
- Where worker RCS exposure presents a significant risk to health, health surveillance is legally required. It should be regularly performed according to evidence-based standards to include detailed occupational history and task recording, respiratory function testing and radiological assessment.

Summary

This paper was compiled to give guidance on the assessment, evaluation and control of occupational exposure to RCS, with an emphasis on recommending a health-based occupational exposure guidance value. The current Safe Work Australia (SWA) workplace exposure standards (WES) and current international occupational exposure limits (OELs) are discussed and the possible health effects examined.

Historically, RCS dust has been responsible for a large burden of occupational ill health, with many deaths from silicosis, a disease which results in the formation of scar tissue in the lung, reduced oxygen transfer and associated loss of lung function. A number of factors are thought to affect the potential for RCS to cause silicosis and there are different types of silicosis (chronic, accelerated and acute) caused by different RCS levels and periods of exposure. 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.

Reported new cases of silicosis occurring in Australian industries are concerning but appear to be either due to historic poorly controlled long-term exposures or to contemporary acute uncontrolled exposure situations above the current WES. It is acknowledged however, that not all workers who develop silicosis many years after exposure are accounted for in the statistics as they may not apply for or receive compensation. Information on new cases of silicosis occurring is limited to results from medical surveillance in only a small number of industries along with determining the number of compensated cases in each state and territory, a situation that could be remedied by development of a centralised register for the reporting of dust-related lung disease.

The International Agency for Research on Cancer (IARC, 2012) indicates that there is sufficient evidence that crystalline silica in the form of quartz or cristobalite is a human carcinogen and there is compelling evidence that many forms of pulmonary fibrosis, including silicosis, constitute major risks for developing human lung cancer. Most studies indicate that in the absence of silicosis development, any increased risk of lung cancer above background rates should be negligible. RCS may also be related to the development of other diseases such as chronic obstructive pulmonary disease (COPD), kidney disease and autoimmune disorders.

It is becoming evident that the extrapolated theoretical "no observed adverse effect level" (NOAEL) at which it can be categorically stated that exposure to crystalline silica has no adverse health effects is very low. Selective literature is predicting risks to health at levels previously considered as being acceptable, although we may have already reached a level of exposure where it is not possible to detect excess morbidity or mortality in a workforce above that experienced by the normal population. The determination of real-world exposures at such a level may be hampered by limitations in measurement technology which do not allow for the accurate measurement of very low-level exposure (< 0.05 mg/m^3).

The AIOH recognises that 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[1] (ALARP), particularly for carcinogens. For occupational risk management purposes, the primary aim should be to keep occupational exposures to RCS to ALARP.

The AIOH thus supports an 8-hour TWA WES of 0.1 mg/m^3 for RCS, as long as worker exposures are at all times limited to ALARP below this limit. The principal reason for this position is that current and historical evidence, including that from the Australian workforce, indicates that if enforced it appears to be protective of the incidence of silicosis, and it is consistent with published threshold levels of effect. It is also consistent with some other country OELs and is a measurable level that is conducive to encouraging industry to strive to determine compliance against the WES or action level.

To this end the AIOH recommends that where there is a likelihood of 50% of the exposure standard being exceeded, it should be applied as an action limit which triggers investigation of the sources of exposure and implementation of suitable control strategies as well as health surveillance. To overcome limitations in analytical sensitivity for measurement of crystalline silica, near full shift monitoring and the use of a NATA accredited laboratory applying standardised analysis and reporting methods is recommended. Health risk relative to the recommended 0.1 mg/m^3 guidance exposure value and 0.05 mg/m^3 action level relating to the need for controls and health surveillance should be determined by a Certified Occupational Hygienist (COH®) applying the approaches and compliance decision making process detailed in '*Occupational Hygiene Monitoring and Compliance Strategies*' published by the AIOH.

Control technologies have been developed and are available for successful implementation. The hierarchy of risk controls must be applied when determining the appropriate controls to be utilised. Most RCS exposures may be adequately controlled by conventional means such as use of wet methods to suppress dusts, local exhaust ventilation, use of positive pressure cabins and segregation of workers from areas of high concentration, as published by various authorities. There can be multiple sources of RCS dust and every workplace is different, hence more than one control strategy will likely be

[1] The meaning of 'reasonably practicable' is as set out in section 18 of the Model Work Health and Safety Act (2011), where the term "so far as is reasonably practicable" is used. Essentially, it means that all practical precautions need to be identified for controls followed by a process to determine reasonableness.

required to reduce worker exposures to acceptable levels. Whatever strategy is adopted it should be under-pinned by an effective maintenance program so that dust control effectiveness is sustained.

Where RCS exposure presents a significant risk to worker health there is a legal requirement to provide health surveillance, which should be regularly performed according to evidence-based standards to include detailed occupational history and task recording, respiratory function testing and radiological assessment.

1. Background

The re-emergence of cases of coal workers' pneumoconiosis in Queensland and the re-emergence of acute silicosis cases in stonemasons in a number of states has called into question not only the numerical value of the respirable coal dust exposure limit (AIOH, 2018) and that for RCS, but also the utmost importance of compliance with these limits.

This position paper is a revision of the 2009 AIOH paper on RCS. This current version of the RCS Position Paper is the outcome of review of an ever-increasing published literature on the topic, sometimes with opposing views, and AIOH member comment received on a draft version. There have been over 100 epidemiologic studies of RCS and lung cancer (Steenland & Ward, 2014). The literature on RCS is thus extensive and a complete review is beyond the scope of this Position Paper. However, more than 80 references are provided in this Paper.

2. What is respirable crystalline silica?

Crystalline silica, a form of silicon dioxide, is one of the most abundant minerals in the earth's crust, with quartz being the most common form. It is present as part of a mixture of minerals 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, and in natural and engineered or manufactured stone used to fabricate kitchen and bathroom benchtops and fit-outs for commercial premises. Hence, economically important activities carried out across numerous Australian industries inherently generate dust which contains RCS and therefore the potential for RCS exposure in the Australian working population is significant (refer section 4).

Silicon dioxide can occur in both crystalline and non-crystalline forms. Crystalline silica as quartz or other crystalline forms are sometimes referred to as 'free' silica (i.e. not combined with other elements such as other crystalline silicates like amphiboles). The main forms of crystalline silica are alpha-quartz (α-quartz), cristobalite and tridymite, the most prevalent of which is α-quartz (both in abundance and as a workplace RCS exposure). Other forms of crystalline silica include coesite, stishovite and morganite. The mineral 'chert' (a cryptocrystalline mineral), quartzite, tripoli and silica sand are also classified as crystalline silica under quartz (IARC, 2012).

Humans have been exposed only to quartz, cristobalite and tridymite, the other forms being very rare (IARC, 2012). Tridymite exposures are rarely if ever found in the workplace (NIOSH, 2002) and there is limited epidemiological research on its relative health effects.

Crystalline silica is an aggressive, lung damaging dust when it is able to penetrate deep into the lung in sufficient quantity. The greater the dose (cumulative exposure) the greater the degree of lung damage. The non-crystalline form of silica (i.e. amorphous 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 10 μm equivalent aerodynamic diameter - EAD), and this size is defined as 'respirable' (refer section 7). Therefore, we call the toxic form of this dust 'respirable crystalline silica' or RCS.

3. Hazards associated with respirable crystalline silica

RCS particles induce lung (bronchogenic) inflammation that persists even after cessation of exposure, with alveolar macrophages having reduced chemotactic responses and phagocytosis. Crystalline silica impairs macrophage-mediated clearance secondary to its cytotoxicity that allows these particles to accumulate and persist in the lungs (IARC, 1997). The National Institute for Occupational Safety and Health (NIOSH, 2002) reviewed the studies considered by the International Agency for Research on Cancer (IARC) and the American Thoracic Society (ATS) and agreed that RCS be considered a potential occupational carcinogen. They also noted that RCS exposure was associated with the development of silicosis, pulmonary tuberculosis and airways diseases, and may also be related to the development of autoimmune disorders (including scleroderma, systemic lupus erythematosus, rheumatoid arthritis, sarcoidosis), chronic renal disease and other adverse health effects.

In a study of 74,040 workers who worked at 29 metal mines and pottery factories in China for 1 year or more, Chen *et al* (2012) observed an increased risk not only for deaths due to respiratory diseases and lung cancer, but also for deaths due to cardiovascular disease for workers exposed to RCS. Gallagher *et al* (2015), on conducting an extended follow-up of lung cancer and non-malignant respiratory disease mortality among California diatomaceous earth workers (as used in the IARC assessment) found that the risk of lung cancer and non-malignant respiratory disease mortality remained elevated, although generally non-significant, and exposure-response trends with cumulative RCS persisted.

The following health effects have been attributed to RCS: silicosis, lung cancer, chronic obstructive pulmonary disease (COPD), kidney (renal) disease and development of autoimmune disorders.

Silicosis

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. 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 α- 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 thus 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 and odd-shaped particles possess a much larger surface area than larger particles for the same mass. 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 grit-blasting, crushing and grinding. However, the activity of the surface to produce free radicals' decays with time, a process known as 'aging'. This occurs slowly in air, but rapidly (within minutes) in water.

Silicosis has been described as chronic silicosis, accelerated silicosis and acute silicosis (Parker & Gregory, 2011).

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 to 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 (typically 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 workers in the engineered stone industry, and has historically been reported mainly among silica powder workers.

Crystalline 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 (Rees & Murray, 2007).

Susceptibility to silicosis is in part genetically determined. Polymorphisms in the promoter region of tumour necrosis factor, a cytokine with a central role in the pathophysiology of silicosis, have been associated with predisposition to several infectious and inflammatory diseases (Yucesoy *et al*, 2001; Corbett *et al*, 2002).

Lung cancer

In 1997, a monograph published by IARC concluded that there was sufficient evidence in humans for the carcinogenicity of inhaled RCS in the form of quartz or cristobalite from occupational sources (IARC, 1997). The 2012 monograph confirmed that crystalline silica in the form of quartz or cristobalite dust is a Group 1 carcinogen without assigning potency (IARC, 2012). Several studies among the many reviewed by the IARC working group in 1997 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, in the 2012 monograph, findings of relevance to lung cancer and crystalline silica exposure arose from five main industrial settings: ceramics, diatomaceous earth, ore mining, quarries, and sand and gravel. Of these, the industries with the least potential for confounding are sand and gravel operations, quarries, and diatomaceous earth facilities. Among those industry segments, most studies with quantitative exposures report associations between crystalline silica exposure and lung cancer risk. Results from the other industry segments generally added support although some studies had potential confounding from arsenic, radon, or PAHs. The strongest evidence supporting the carcinogenicity of RCS in the lung comes from the pooled and meta-analyses of selected epidemiological studies.

Liu *et al* (2013) investigated a cohort in China (1960–2003) of 34,018 workers without exposure to other occupational carcinogens. Categorical analyses by quartiles of cumulative RCS exposure (using a 25-year lag) yielded hazard ratios of 1.26, 1.54, 1.68, and 1.70, respectively, compared with the unexposed group. The joint effect of silica and smoking was more than additive and close to a multiplicative effect.

The National Toxicology Program (NTP, 2016) concluded that exposure of workers to RCS is associated with elevated rates of lung cancer, this link being strongest in studies of quarry and granite workers and workers involved in ceramic, pottery, refractory brick and diatomaceous earth industries. They also noted that silicosis was associated with elevated lung cancer rates.

Gamble (2011) critically assessed the IARC cancer classification based on exposure-response analyses in 18 studies from eight countries with about 2,000 lung cancer cases and the same database used by IARC. Strength of association was found to be consistently weak in the majority of studies. At the highest exposure level, the mean relative risk (RR) was 1.5; four studies had strong associations (RRs > 2), three had moderate strong associations (RRs 1.5-2.0), six had weak-negligible associations (RRs 1-1.5), and five had no associations (RRs ≤1.0). Biological gradients were an inconsistent finding. Three studies had clear positive exposure-response trends; 3 had suggestive trends; and 12 had no exposure-response trends, 9 of which were flat or negative. There was a negative exposure-response slope using RRs at the highest exposure of each study. It was concluded that the weight of evidence from occupational epidemiology does not support a causal association of lung cancer and silica exposure, contrary to the IARC conclusion.

In contrast, OSHA (2010) concluded that the human studies that IARC reviewed provided ample evidence that exposure to RCS increased the risk of lung cancer among exposed workers. Their conclusion was based on different studies of the same cohort used by Gamble. OSHA and Gamble had differing opinions on which study was most appropriate.

The UK HSE (2003) concluded that the balance of evidence suggests that heavy and prolonged occupational exposures to RCS can cause an increased risk of lung cancer. However, they noted that, of the very many studies available, most of which clearly demonstrate excess mortality and morbidity from silicosis, there are few studies that, taken in isolation, provide reasonably convincing evidence for an increase in lung cancer that can be attributed to RCS. They suggest this appears to support the view that RCS is a relatively weak carcinogen, otherwise the evidence for lung cancer would be far clearer and convincing than is the case.

Quantitative estimates of the relationship between exposure to RCS and lung cancer risk have varied widely, even among studies conducted in the same industry. Vacek and Callas (2017) re-analysed two previous studies relating crystalline silica exposure to lung-cancer mortality in Vermont granite workers which yielded conflicting results. Differing results from the two studies were partly attributable to incomplete vital status and work history information used in the earlier study, as well as differences in cohort inclusion criteria. However, differences in length of follow-up and other factors likely played a larger role. The results demonstrate some of the difficulties in using epidemiological data, particularly limited, extrapolated or derived exposure data, to estimate exposure-response and determine appropriate exposure limits.

Brown (2009) also noted that the latent period for cancer development can make it difficult to establish a definite exposure-response relationship. The picture is further complicated by variable job histories, concomitant exposure to other carcinogens and other factors such as genetic susceptibility and poor nutrition. Brown concluded that further research is needed in order to understand the complex pattern of interactions leading to lung cancer among silica-exposed workers (and cancers and workplace exposures in general) and to understand whether and to what extent other workplace lung carcinogens (e.g. smoking), total respirable dust and total surface size and age of silica particles affect the carcinogenic potential of silica.

Lung cancer with or without silicosis debate

Brown (2009) noted that if silicosis were the necessary step leading to lung cancer, enforcing the current RCS standards would also protect workers against lung cancer risk. Alternatively, a direct silica-lung cancer association implies that regulatory standards should be revised.

The ATS (1997) concluded the following:

- The available data support the conclusion that silicosis produces increased risk for bronchogenic 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.

The hypothesis that occupational RCS exposure is a direct acting cancer initiator is still debated producing no clarity in conclusion.

The UK HSE (2003) concluded that the weight of evidence suggests that exposures to RCS insufficient to cause silicosis would be unlikely to lead to an increased risk of lung cancer, although the evidence for this is not definitive. Parker and Gregory (2011) noted that uncertainty over the pathogenic mechanisms for the development of lung cancer in silica-exposed populations exists, and the possible relationship between silicosis and cancer in exposed workers continues to be studied. There is however compelling evidence that many forms of pulmonary fibrosis, including silicosis, can lead to lung cancer (HSE, 2003).

SCOEL (2003) concluded that there is evidence that the incidence of lung cancer increases with increasing cumulative exposure to RCS dust and that the relative lung cancer risk is increased for persons with silicosis. It is not clear from which exposure value the relative lung cancer risk is increased. The studies differ with respect to exposure levels and durations, with respect to the type of crystalline silica and also the occupational confounders such as simultaneous exposure to radon.

ACGIH® (2010) classifies 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 RCS 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.

Mundt *et al* (2011) quantified silicosis and lung cancer risks among porcelain workers occupationally exposed to RCS and found that exposure to more than 0.15 mg/m^3 (average) were strongly associated with silicosis, but unrelated to lung cancer risks. Vacek *et al* (2011) found that RCS exposure in Vermont granite workers was associated with increased mortality from silicosis and other non-malignant respiratory disease, but there was no evidence that increased lung cancer mortality in the cohort was due to exposure. Erren *et al* (2011) followed up on a previous meta-analysis of lung cancer risk in individuals without silicosis to answer the question "does silica cause lung cancer in the absence of silicosis"? They substantiated evidence of a strong association between silicosis and lung cancer. However, questions remained regarding lung cancer caused by silica in non-silicotics.

Regarding the IARC (2012) classification of RCS as a human carcinogen, Guha *et al* (2011) note that the strongest supportive evidence comes from pooled and meta-analyses that employed quantitative exposure assessment, focused on silicotics. Borm *et al* (2011) after reviewing literature concluded that the mechanism of RCS genotoxicity is via inflammation-driven secondary genotoxicity; that is due to “persistent inflammation”, as concluded by IARC (2012). The role of inflammation driven by quartz surface in genotoxic and carcinogenic effects after inhalation have since been confirmed in an updated review of the genotoxicity of RCS, and the findings support a practical threshold (Borm *et al*, 2018).

Steenland & Ward (2014) suggest that recent epidemiological studies have provided new information about RCS and lung cancer, in particular that excess lung mortality occurs in silica- exposed workers who do not have silicosis and who do not smoke, citing the Chinese study by Liu *et al* (2013).

Greaves (2000) noted that “The causal pathways between silica, silicosis, and lung cancer will continue to be debated, but these are futile discussions when it comes to protecting silica-exposed individuals from developing lung cancer. Fortunately, regardless of the actual mechanism, the information linking silica, silicosis, and lung cancer leads to a common conclusion: individuals who inhale silica dust in sufficient amounts to cause fibrosis (silicosis) are at increased risk of lung cancer.”

Chronic obstructive pulmonary disease (COPD)

The literature is showing an increased weight of evidence regarding exposure to RCS (and also other non-siliceous mineral dusts) causing COPD (Hnizdo & Vallyathan, 2003; Heederick, 2004; Brüske *et al*, 2014). COPD is known by a number of other names including chronic obstructive airway disease (COAD), chronic airflow obstruction (CAO) and chronic airway limitation (CAL). It is also referred to as chronic bronchitis and emphysema. COPD 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 contributor of COPD.

Heederick (2004) concluded that evidence from various sources (toxicological, epidemiological and pathological studies) all indicate that RCS dust exposure can lead to COPD, independent from smoking and in the absence of silicosis. The available evidence also suggests that chronic exposure to RCS dust, even below levels associated with silicosis, may cause COPD. Rego *et al* (2008) evaluated RCS exposure and respiratory disease in granite workers. They found that RCS induced respiratory function (FEV_1) alteration regardless of silicosis and, in all likelihood, synergistically with tobacco.

Brüske *et al* (2014) conducted a systematic review and meta-analysis of cross-sectional studies to obtain an overall estimate of forced expiratory volume in 1 second (FEV_1) and FEV_1/forced vital capacity (FVC) reduction due to RCS exposure. The meta-analysis showed a statistically significant reduction in the mean ratio FEV_1 to FVC and the FEV_1 of workers exposed to RCS dust, which was 4.6% less than predicted compared with workers with no/low exposure.

Renal disease

IARC (2012) also concluded that cancers other than that of the lung have not been as thoroughly researched.

Increased risk of renal disease has been implicated with elevated exposures to crystalline silica. A US study found a doubling of risk of non-malignant renal disease but no increase in renal cancer in sand and gravel workers (McDonald *et al*, 2005), although Attfield & Costello (2004) did find an increased risk of kidney cancer in granite workers. Vacek *et al* (2011) found that mortality from malignant and non-malignant kidney disease was not significantly increased or associated with RCS exposure in Vermont granite workers. Möhner *et al* (2017) conducted a review of the association between occupational exposure to RCS and chronic non-malignant renal disease. They concluded that while the studies of cohorts exposed to RCS found elevated standard mortality ratios (SMRs) for renal disease, there was no clear evidence of a dose–response relationship, the elevated risk perhaps being attributed to diagnostic and methodological issues.

Both direct effects and an autoimmune response have been postulated as mechanisms for silica related renal disease (Cherry, 2004).

Autoimmune disorders

Cooper *et al* (2002) reviewed the epidemiologic and experimental literature on the association between autoimmune diseases and occupational exposure to silica, solvents, pesticides and ultraviolet radiation. They found that the strongest associations (i.e. relative risks of 3.0 and higher) were documented in investigations of RCS dust and rheumatoid arthritis, lupus, scleroderma and glomerulonephritis. Parks *et al* (1999) suggested a 'possible association' between RCS exposure and scleroderma, the evidence being strongest in relation to dust exposures in the mining industry, and lesser or even absent in other industries where RCS is known to be present. An Australian population-based study investigating male systemic sclerosis sufferers in Sydney and occupational RCS exposure found a relative risk of between two and four-fold when compared to age and gender matched controls without systemic sclerosis (Englert *et al*, 2000). The study also found that the latency from first onset of systemic sclerosis symptoms and first exposure in the study group was approximately 23 years (8 to 38 years). No difference was found in the characteristics between RCS exposed and non-silica exposed systemic sclerosis group, indicating that systemic sclerosis in RCS exposed individuals is largely indistinguishable from idiopathic systemic sclerosis; a point highlighted in the review by Parks *et al* (1999).

A meta-analysis of RCS as a risk factor for scleroderma (McCormic *et al*, 2010) concluded that RCS exposure may be a significant risk factor for developing systemic sclerosis (again, a relative risk of 3.0), particularly in males. There was significant heterogeneity of the data analysed, there being some studies that found no significant increased risk while others had significantly higher relative risk than three, hence the evidence to date is not sufficient to conclude that RCS is a causative factor for systemic sclerosis. A more recent and larger meta-analysis (Rudio-Rivas *et al*, 2017) found a significant increased overall risk of scleroderma after exposure to RCS, solvents, silicone breast implants, epoxy resins, pesticides, and welding fumes, with RCS and solvent exposures being the two most likely substances related to the pathogenesis of systemic sclerosis. There is no exposure-response data available for RCS exposure and systemic sclerosis, with a small number of studies indicating high acute exposures may produce a slightly higher level of risk than indicated by long-term cumulative exposures (Martin *et al*, 1999; Sluis-Cremer *et al*, 1985).

The systematic review of data from cases of silicosis due to artificial (i.e. engineered) stone dust identified nine out of 40 silicosis cases (23%) with findings consistent with various autoimmune diseases (Shtraichman *et al*, 2015). Among these nine cases, three also had findings consistent with pulmonary alveolar proteinosis. Based on an expected autoimmune disease prevalence of 3% (based on the upper-end estimate for this group of diseases in European international data), the proportion of disease in this group represented a greater than 7-fold excess.

4. Major uses / potential for exposure (in Australia)

Industries where RCS is known to be present include:

- Mining and exploration
- Quarrying
- Foundries
- Ceramics
- Brick manufacture and heavy clay
- Refractories manufacturing
- Industrial minerals and the production and use of silica sand and flour
- Construction
- Tunnelling

- Stonemasonry (either using natural stone, i.e. sandstone, or engineered stone, i.e. kitchen bench tops)

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®, 2010). In Australia, the use or handling of a substance that consists of, or contains, crystalline silica as an abrasive material in abrasive blasting, is prohibited under work safety and health regulations.

A significant proportion of the Australian working population are employed in the above industries, and local monitoring data indicates exposure varies considerably between the various industries and trades (NOHSC 1993). Mining / quarrying and construction / tunnelling are the largest industries with exposure to RCS in Australia. Mine maintenance workers can be some of the highest exposed. Miners, especially iron ore miners in Western Australia, can be exposed to the mineral chert. The Minerals Council of Australia has reported 484,114 people directly employed in the Mining Industry and 655,654 indirectly employed, amounting to approximately 10% of total employment. However, the Parliament of Australia website notes only around 220,000 people employed in mining during 2017 and 1.16 million employed in construction.

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. The cutting of bricks, concrete and tiles is also a source of high RCS exposure. In more recent times, the cutting of engineered stone in kitchen and bathroom renovations has become a source of high RCS exposure with up to 44.6 mg/m^3 occurring during dry cutting over a 30-minute sampling period (Cooper *et al*, 2015). In a plant making natural and engineered stone countertops, RCS exposures for employees using pneumatic wet grinders with diamond cup wheels were highest, with 8-hour TWA exposures ranging from 0.093 to 0.14 mg/m^3 for four personal samples (Zwack *et al*, 2016). Queensland WorkSafe (2018) also reported that RCS was not adequately controlled even when wet methods of fabrication were used (2018). RCS exposures (8-hour TWA) for restoration stonemasons working on Sydney sandstone were found to exceed 0.1 mg/m^3, with some exposures calculated at 40, 60 and 120 times this value (Alamango *et al*, 2015).

A cross-sectional survey of the Australian working population (18-65 years old) conducted using telephone interview found that overall, 6.4% of respondents were deemed exposed to RCS at work in 2012 (3.3% were exposed at a high level) (Si *et al*, 2016). The exposure varied with sex, state of residence and socioeconomic status. Miners and construction workers were most likely to be highly exposed to RCS when performing tasks with concrete or cement or when working near crushers that create RCS-containing dusts. Workers involved in tunnelling work for roads and infrastructure projects are also at risk of high RCS exposures unless well controlled. When extrapolated to the entire Australian working population, 6.6% of Australian workers were exposed to RCS and 3.7% were highly exposed when carrying out tasks at work.

The New South Wales (NSW) Standing Dust Committee (2018) reported on 2,526 respirable coal dust samples taken in NSW during 2017. RCS samples were taken in 966 incidences, with 5.3% underground and 2.0% surface operators exceeding the OEL of 0.1 mg/m^3. The NSW data indicate declining exposures to RCS since 2015 with high RCS exposure occurring for underground mining processes that involve driving drifts through stone, mining through rock intrusions, drilling or bolting into a stone roof during development and secondary support activities.

RCS exposures to tunnel construction workers were reported in 2010 to be above the current WES for most tunnel workers, rising to more than 50 times that standard for those workers operating open-cabin road headers (Queensland WorkSafe, 2010), which are still used in Australia. Exposures to tunnellers working on tunnel boring machines in 2016 were reported to be double the WES (Cole, 2016). The tunnelling industry has significantly expanded since those two studies to include many more major projects, hence RCS exposures in this industry are of increasing concern.

International data for construction worker exposure to RCS suggest that trade-specific geometric mean (GM) varied from 0.01 (plumber) to 0.30 mg/m^3 (tunnel construction skilled labour), while tasks vary from 0.01 (six categories, including sanding and electrical maintenance) to 1.59 mg/m^3 (abrasive blasting) (Beaudry *et al*, 2012). Sauvé *et al* (2013) found that of the 27 construction tasks they analysed, abrasive blasting, masonry chipping, scrabbling concrete, tuck pointing and tunnel boring had estimated GMs above 0.1 mg/m^3 based on the exposure scenarios they developed.

Data from Alberta Canada (Radnoff *et al*, 2014) demonstrated that the industries with the highest potential for RCS over-exposures occurred in sand and mineral processing (GM 0.090 mg/m^3), followed by new commercial building construction (GM 0.055 mg/m^3), aggregate mining and crushing (GM 0.048 mg/m^3), abrasive blasting (GM 0.027 mg/m^3) and demolition (GM 0.027 mg/m^3). For worker occupations, GM exposure ranged from 0.105 mg/m^3 (brick layer / mason / concrete cutting) to 0.008 mg/m^3 (dispatcher / shipping, administration). Potential for GM exposure at levels exceeding the OEL was identified in a number of occupations where it was not expected, such as electricians, carpenters and painters. These exposures were generally related to the specific task the worker was doing, or arose from incidental exposure from other activities at the work site.

5. Risk of health effects

Quantification of the risks of silicosis should take account of variations in RCS exposure intensity, particularly for exposure to concentrations of greater than 1 or 2 mg/m^3, even if exposure is for relatively short periods. The risks of silicosis over a working lifetime can rise dramatically with even brief exposure to such high quartz concentrations (Buchanan *et al*, 2003). There are varying views on the risk of health effects at RCS exposure concentrations of 0.1 mg/m^3 and below.

Finkelstein (2000), upon investigating exposure-response relationships for RCS, silicosis and lung cancer, concluded that the lifetime risk of silicosis and lung cancer at an exposure level of 0.1 mg/m^3 is high. The available data suggested that 30 years daily average exposure at 0.1 mg/m^3 might lead to a lifetime silicosis risk of about 25%, whereas reduction of the exposure to 0.05 mg/m^3 might reduce the risk to under 5%.

While a cohort study of long-term exposure to RCS and risk of mortality in Chinese workers found significant positive exposure-response relationships from all causes (SMR 1.06), ischemic heart disease (SMR 1.65) and pneumoconiosis (SMR 11.01) among workers exposed to RCS concentrations equal to or lower than 0.1 mg/m^3, there was possible underestimation of the level of silica dust exposure for individuals who worked at the mines/factories before 1950 (Chen *et al*, 2012).

Cox (2011) modelled the exposure-response relation between RCS and risk of lung pathologies such as chronic inflammation, silicosis, fibrosis, and lung cancer using an inflammatory mode of action. The mechanism derived implied a "tipping point" threshold for the exposure-response relation. Applying this new model to epidemiological data, Cox concluded that current exposure levels, on the order of 0.1 mg/m³, are probably below the threshold for triggering lung diseases in humans.

Morfeld *et al* (2013) concluded that a concentration threshold for silicosis is plausible and estimated a threshold value for the RCS dust concentration and silicosis incidence (1/1, ILO 1980/2000) in the German porcelain worker cohort. They also concluded that a threshold Cox model fitted the data significantly better than a non-threshold model, summarised the cohort information without a loss in extracted information and more simply than restricted cubic splines and fractional polynomials. They calculated a best threshold estimate was 0.25mg/m^3 (95% confidence interval: 0.15 to 0.30 mg/m^3). Taking into account various uncertainties, this study indicated an RCS dust exposure (8-hour TWA) concentration threshold greater than 0.1 mg/m^3 and possibly as high as 0.25 mg/m^3.

Liu *et al* (2013) determined that for Chinese workers exposed to RCS from ages 20 to 65 years at 0.1 mg/m^3, the estimated excess lifetime risk (through age 75 years) was 0.51%.

A study on risk estimates for silicosis undertaken in 2005 by the Institute of Occupational Medicine (IOM) in Edinburgh applied bio-mathematical modelling to animal studies to estimate the human "no observed adverse effect level" (NOAEL) at 0.001 mg/m^3 (Tran *et al*, 2005). The study noted that the average exposure limits implied by risk estimates from epidemiological studies ranged from 0.01 to about 0.05 mg/m^3, some 9 to 45 times higher than the limits derived from the animal studies, thus the conventional uncertainty factors applied in the animal-based risk estimates may be over-precautionary.

Australia

Numerous state and commonwealth investigations into RCS exposure have resulted in repeat recommendations for the establishment of a centralised occupational disease(s) register along with the mandatory reporting and recording of disease cases (Faunce *et al*, 2006). No such register has been established, therefore in the absence of such, quantifying the incidence of silicosis in the Australian population is limited to determining the number by reference to a small number of medical surveillance programs such as is conducted by the previous NSW Dust Diseases Board and compensated cases recorded in each state and territory. This makes it difficult to determine the true extent of lung disease in Australia, as such statistics only reflect the number of cases that have been successful in their application for compensation and are not representative of the actual incidence of disease(s) associated with occupational RCS exposure, including disease outside the scope of the respiratory system. This situation could be remedied by development of a centralised occupational disease(s) register for the reporting of dust-related lung disease.

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 (i.e. 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).

The Surveillance of Australian workplace Based Respiratory Events (SABRE) NSW scheme, a voluntary notification scheme established to determine the incidence of occupational lung diseases in NSW, was analysed by Hannaford-Turner *et al* (2010) for June 2001 to December 2008. They found that while asbestos-related diseases were the most frequently reported conditions to SABRE NSW, silicosis cases were the next most reported, with 90 (2%) new cases; an estimated average annual incidence of 9 new cases. Construction workers and labourers employed to perform 'in site' preparation (demolition and excavation) in the building industry were most frequently reported to have RCS exposure and to have developed silica-related disease. It should be noted that the SABRE scheme was discontinued by the former NSW Dust Diseases Board as a result of “low participation, under diagnosis and under reporting resulting in incidence rates of new diagnoses being underestimated” (Parliament of NSW, 2017).

Hoy *et al* (2018) reported on seven male silicosis patients, all of who were employed in Australian small kitchen and bathroom benchtop fabrication businesses with an average of eight employees (range 2–20). All workplaces primarily used engineered stone, and dust control measures were poor with all patients being involved in dry cutting of the stone. The median duration of exposure prior to symptoms was 7 years (range 4–10). Six patients demonstrated radiological features of progressive massive fibrosis. Based on initial exposure studies on the industry conducted by state regulators, exposures to RCS were likely many times over the current WES of 0.1 mg/m^3.

Current reported cases of silicosis in Australia appear to be either due to historic poorly controlled long-term exposures or to contemporary acute uncontrolled exposure situations above the current WES.

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 & Music, 1998).

In a report to NOHSC de Klerk *et al* (2002a) 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. It should be noted that the above RCS value would be equivalent to about 0.1 mg/m^3 using the CEN/ISO/ACGIH sampling efficiency curve (Isabella *et al*, 2005) (refer Section 7).

USA

The Occupational Safety and Health Administration (OSHA, 2010) in conducting a comprehensive review of RCS health effects presented lifetime silicosis risk estimates associated with occupational exposure at varying levels of exposure. For RCS generated using high-energy processes (i.e. freshly cut or fractured), silicosis morbidity risk associated with exposure to 0.1 mg/m^3 over 45 years was estimated to range between 12 and 77 cases per 100 workers, based on studies that were judged to have sufficient follow-up of retired workers. For similar exposure to 0.05 mg/m^3 the estimated risk ranged between 2 and 17 cases per 100 workers. OSHA estimated the most reliable risks to be 30 cases per 100 workers for exposure to 0.1 mg/m^3 and 5.5 cases per 100 workers for exposure to 0.05 mg/m^3. However, those estimates reflected the risk of developing more advanced stages of silicosis than did the other studies, and thus underestimated the actual risk of radiological silicosis.

OSHA (2010) estimated the lifetime lung cancer risk associated with 45 years of exposure to RCS at 0.1 mg/m^3 to range from 13 to 60 deaths per 1,000 workers. For exposure to 0.05 mg/m^3, the calculated lifetime risk estimates were in the range of 6 to 26 deaths per 1,000. OSHA has thus issued two new RCS standards: one for construction, and the other for general industry and maritime, both using an 8-hour TWA permissible exposure limit (PEL) of 0.05 mg/m^3 to trigger worker protection.

The ACGIH® (2010) 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. While there is a lack of toxicological and industrial hygiene data to recommend a short-term exposure limit, the ACGIH® note that high exposures of short duration to freshly fragmented crystalline particles produce an acute and rapidly progressive form of silicosis. It should be noted that, in deriving the 0.025 mg/m^3 TLV®, the ACGIH state that a TLV-TWA of 0.05 mg/m^3 would probably not be sufficiently protective of workers' health as there was a significant increase in mortality risk from lung cancer at average exposure levels greater than 0.065 mg/m^3 in the Steenland and Sanderson (2001) study. This study had a geometric standard deviation of 10.9 overall, which indicates highly variable exposures, and more than half of the exposure results exceeded 0.05 mg/m^3. In addition, the highest SMR was for workers with less than 6 months tenure, so the risk data was re-analysed without them.

United Kingdom (UK)

In the UK, a review by the Health and Safety Executive (HSE, 2002) 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. Phase 2 of the review concluded that RCS is only weakly carcinogenic (HSE, 2003).

In 2003 the European Scientific Committee on Occupational Exposure Limits (SCOEL), made a recommendation to the European Commission. SCOEL noted that in humans the main effect of the exposure to RCS dust is silicosis. Other non-neoplastic pulmonary effects in humans were inflammation, lymph node fibrosis, chronic air flow limitation, emphysema and extrapulmonary silicosis. They also noted that there was an association between exposure to RCS dust and an increased probability of developing lung cancer, with the incidence of lung cancer increasing especially in workers with silicosis.

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, 2003). 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).

6. Current applicable legislation and standards

According to the GESTIS International Limit Values website, the 8-hour TWA OEL for RCS ranges from 0.03 to 0.15 mg/m^3 in different countries, with 0.05 and 0.1 mg/m^3 being most common. The current Safe Work Australia (SWA) workplace exposure standard (WES) for RCS is 0.1 mg/m^3. All Australian states have adopted this into their regulations.

The SWA (2016) *Model Work Health and Safety Regulations* require that risks to health and safety must either be eliminated or minimised so far as is reasonably practicable, following the hierarchy of risk control. The Model Regulations also require that no person in a workplace is exposed to RCS at a concentration above the WES and requires the conduct of air monitoring to determine worker's RCS exposure concentration(s) where:

- there is uncertainty as to whether the RCS concentration exceeds the WES; or
- monitoring will inform the determination of risk to workers health.

In addition, where workplace exposure to RCS could adversely affect worker health, then health monitoring (surveillance) must be provided.

7. How do we measure it?

In order for RCS to present a risk to health it must be inhaled deeply into the lungs. Exposure is therefore assessed by measuring the airborne concentration of a particular size fraction. It should be noted that there are state guidelines available that detail how to manage the monitoring of workers' exposure to RCS and how to manage their health surveillance (e.g. the Queensland Guideline for Management of Respirable Crystalline Silica in Queensland Mineral Mines and Quarries - QGL02 (Queensland DNRME, 2018)).

SWA (2013a) note that air monitoring must not be used as an alternative to controlling exposure and is best done after control measures have been put in place. Air monitoring has both a sampling and an analysis component that can influence the accuracy and precision of the RCS exposure measurement.

Air monitoring – sampling

Currently the method used to sample respirable dust is AS 2985 (2009) which follows ISO 7708:1995, *Air quality – Particle size fraction definitions for health-related sampling*. According to AS 2985 (2009), respirable dust is the proportion of airborne particulate matter which, when inhaled, penetrates to the unciliated airways (deep in the lung). Respirable dust satisfies a sampling efficiency curve that passes through the points of 100% efficiency at 0 µm EAD and below, 50% efficiency at 4 µm EAD and 1% efficiency for particles of 10 µm EAD and upwards. This is also known as the CEN/ISO/ACGIH defined particulate size range.

Parallel particulate impactor samplers have been promoted as respirable dust samplers to measure levels of RCS below 0.05 mg/m^3 but they require a larger pump to achieve the higher flow rate required (e.g. 4 L/minute) and use a 37 mm rather than 25 mm filter. Although they may collect more dust, the dust is spread over a larger surface area, hence may not provide an improvement in the limit of detection (LOD) when analysing direct on the filter.

It is important that any sampler used to collect respirable dust meets the requirements of the ISO/CEN/ACGIH sampling efficiency curve. Issues with sampling such as poor control of flow rate and sample duration can introduce uncertainties into the exposure estimate,

Air monitoring – analysis

In Australia, analysis of respirable dust on filters for crystalline silica is typically carried out either by fourier transform infrared spectroscopy (FTIR) or X-ray diffraction (XRD) in accordance with the *NH&MRC method for the measurement of quartz in respirable dust by infrared spectroscopy and X-ray diffractometry* (1984), calibrated against a known national quartz standard. Analysing direct on the filter provides a much better LOD and limit of quantitation (LOQ) than analysing an ashed sample or other re-deposition samples, which introduce problems of potential sample loss in treatment and transfer. It should be noted that if both α-quartz and appreciable levels of cristobalite or other interfering minerals are present on the same sample filter, XRD analysis should be undertaken, as it is the more robust technique with less interferences than those encountered in using FTIR.[2]

Stacey (2007) noted that the LODs reported for RCS are theoretical, not applicable to all matrices and often optimistic of what can be achieved. Measurements at 0.025 mg/m^3 and short-term measurement (~4 hours) at 0.05 mg/m^3 are at the limit of what can be reliably measured using the existing methods and techniques.

Both analytical techniques do remarkably well on the analysis of pure α-quartz standards. The HSE (2014) method for determining RCS in airborne dusts presents data on detection limits and uncertainty. This method is suitable for the determination of quartz and cristobalite at a concentration of 20 µg to 1 mg on a 25 mm filter for both FTIR and XRD. The qualitative and quantitative FTIR LODs for crystalline silica, defined as three times and ten times the standard deviation of a blank determination, are typically around 3 µg and 10 µg per sample respectively. For a 500 L air sample (~4 hours at 2.2 L/minute), these figures correspond to qualitative and quantitative detection limits of 0.006 mg/m^3 and 0.02 mg/m^3 respectively. For XRD, the estimation of LODs for quartz and cristobalite is problematic because they are dependent on the sample matrix, instrumental parameters and performance of the respirable sampler. Using the strongest diffraction peaks, LODs would be 0.02 mg/m^3 quartz and 0.04 mg/m^3 cristobalite for a 500 L air sample. Using the weakest diffraction peaks, LODs could be between 0.03 and 0.08 mg/m^3.

The LODs and LOQs reported for the HSE (2014) method are similar to those reported in other methods such as the NIOSH Methods 7500, 7602 and 7603. The NHMRC (1984) method used by most laboratories in Australia quotes LODs of 20 µg per filter for infrared spectroscopy (0.04 mg/m^3 for a 500 L sample) and 10 µg per filter for XRD (0.02 mg/m^3 for a 500 L sample) for samples which do not have substantial quantities of interfering substances present.

However, many workplace samples have other constituents in the sample that can interfere with the accuracy of the analysis. In the case of FTIR, a reported 30% of workplace samples revealed interference of the infra-red absorbances and did not comply with the 1.0 to 1.4 peak ratio criteria as a quality parameter for acceptability of the acquired signal (Ichikawa *et al*, 2018). The variation of the background signal of the PVC filter has a significant influence on the FTIR result and correction must be performed, enlarging the uncertainty of the FTIR analysis. FTIR cannot be performed on filters that are loaded with more than 1 mg of dust. XRD can analyse up to 2 mg of dust loading, above which a silver background filter can be used to correct for the overload. XRD does not require background correction for the PVC filter, has fewer interferences, however, a 5% reduction in α-quartz response can be observed for samples containing 100 µg of iron oxide.

Air monitoring – conclusions

Taking into account the LOQ[3] plus analytical measurement uncertainty (e.g. $\pm$2 to 5 µg) and including unpredictable uncertainties associated with interfering minerals (independent of FTIR or XRD, which both have different levels of sensitivity to different interfering materials), and considering the uncertainties associated with sampling (e.g. flow rate & sample duration), the reliable determination of RCS levels less than 0.05 mg/m^3 in real world occupational exposure situations is fraught with difficulties.

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 to determine compliance against the WES or action level. 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:

- Conducting near full shift sampling (i.e. an 8-hour sample period or 12-hour sample period for an 8 or 12-hour shift, respectively) under the guidance of a COH® and in accordance with AS 2985.
- Due consideration of the limitations associated with both sampling and analysis for RCS. Caution is needed with regard to the flow rate used and to not overload the cyclone sampler in high dust situations, which makes analysis difficult or even impossible.
- Use of a NATA accredited laboratory to conduct RCS analysis and that the results are reported on NATA endorsed test certificates.

[2] It should be noted that when using FTIR to analyse for α-quartz, the presence of cristobalite or amorphous silica (e.g. uncalcined diatomaceous earth) and some silicates (e.g. kaolinite) on the same sample filter may result in interference or masking. Therefore, to minimise interferences, XRD should be used whenever there are mixtures of different types of RCS and amorphous silica present. However, some other minerals cause interference with XRD also (NHMRC, 1984).

[3] The LOQ should be at least 10% of the exposure standard to best facilitate compliance monitoring.

- Conducting personal sampling for determining the degree of health risk and compliance with the WES, applying '*Occupational Hygiene Monitoring and Compliance Strategies*' published by the AIOH (Grantham & Firth, 2014). Fixed position monitoring may also be used as a means of ongoing monitoring of the effectiveness of controls.

Health surveillance

Adverse health effects to personnel exposed to RCS can be assessed through health surveillance, comparing results to a baseline medical assessment.[4] 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 '*Health Monitoring for Exposure to Hazardous Chemicals – Guide for Medical Practitioners*' (SWA, 2013b) and '*Crystalline silica health monitoring*' (SWA, 2013c). State guidance on health surveillance, such as that in the Queensland WorkSafe (2018) '*Guide to safe bench top fabrication and installation'*, should also be adhered to.

Health surveillance should be regularly performed according to evidence-based standards to include respiratory function testing and radiological assessment. Nicol *et al* (2015) note that it is important to have a low threshold for early radiological screening to promote early and effective detection of silicosis. They reported on six cases of silicosis in young, asymptomatic construction workers who were exposed to RCS between 7 and 20 years (mean 13 years). All had a low apparent predicted probability of pneumoconiosis based on health questionnaires, spirometry and duration of silica exposure. However, the initial chest X-ray was abnormal in all six cases with radiological evidence of silicosis.

The review of the respiratory component of the health assessment performed under the Queensland *Coal Mine Workers' Health Scheme* demonstrates that health surveillance programs require considerable commitment to be effective at detecting disease at an early stage of exposure (Monash University & University of Illinois, 2016). Health surveillance medical examinations need to be systematic and comprehensive.

8. Available controls

Given the ubiquitous nature of crystalline silica, elimination or substitution as control measures are generally impractical. 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. Research indicates that these control measures can be effective. Historical reduction in silicosis numbers has been 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.

Whenever 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.

Any controls for both inhalable and respirable dust exposures will also impact RCS levels in air. The control principles that apply to RCS are thus similar to those that apply to all mechanically generated mineral dust exposures. The hierarchy of risk controls must be applied when determining the appropriate controls to be utilised:

- Design and operate processes and activities to minimise emission, release and spread of dust;
- Position personnel so they are out of the dust either in enclosed and filtered cabins with positive pressure (at least 50 Pa pressure differential) 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, particularly when the process involves freshly cut quartz surfaces;
- Use water (or water with additive) suppression to prevent dust spread;
- Minimise the fall distance of dust generating materials (e.g. 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 suitable ventilation, either dilution or preferably local 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 (especially important inside vehicle dust-proof cabins);
- Use a vacuum cleaner with appropriate filter to clean-up dust spills;
- Provide training in the health effects of RCS dust, its sources and its control, and communicate the results of airborne monitoring and the assessed risk of exposure at the workplace;
- 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 type filtering half face respirator, although a P3 type filter, powered air purifying respirator (PAPR) or even an air-supplied respirator may be required for high RCS exposures (e.g. where a protection factor of more than 50 or 100 is required). Ensure training is

[4] Health surveillance is referred to as 'health monitoring' in the SWA *Model Work Health and Safety Regulations* at Part 7.1, Division 6, which provides detailed requirements.

provided in the use and limitations of respiratory protective equipment (e.g. have a clean-shaven policy). Face fit testing is also required, as per AS/NZS 1715 (2009).

A general observation is that respiratory protection programs are often not well implemented. The New South Wales (NSW) Standing Dust Committee (2018) reported that while respiratory protection was well utilised by coal mining longwall face operators, road header operators, outbye supplies operators, gas drainage operators and ventilation crew operators, this was not the case for continuous miner operators, shuttle car operators, development and outbye deputies and secondary support workers.

Sauvé *et al* (2013) found that, in construction work, the use of water-fed tools and local exhaust ventilation were associated with a reduction of 71 and 69% in exposure levels compared with no controls, respectively. Cooper *et al* (2015) found that for engineered stone countertop cutting, the mean RCS exposure for the baseline wetted-blade-only condition was an order of magnitude lower than the 'dry blade' concentration. The mean RCS concentration for the wetted blade plus local exhaust ventilation was 92% lower than the mean concentration for the wetted-blade-only scenario.

Queensland WorkSafe (2013) provide a technical guide to managing RCS exposure in the workplace and also a guide to safe bench top fabrication and installation with regard to protecting workers from RCS exposure (Queensland WorkSafe, 2018). There is a *Good Practices Guide* available from http://www.nepsi.eu/ containing more than 50 different task sheets that include controls for RCS generation. The UK HSE also provides guidance documentation for controlling RCS exposures at http://www.hse.gov.uk/pubns/silicaindex.htm, as does the US NIOSH at https://www.cdc.gov/niosh/topics/silica/default.html.

There can be multiple sources of RCS dust and every workplace is different, hence more than one control strategy will likely be required to reduce worker exposures to acceptable levels. Whatever strategy is adopted it should be under-pinned by an effective maintenance program so that dust control effectiveness is sustained. It is critical that the effectiveness of controls be determined, as evidenced through reductions in exposure concentrations. It must be said that past implementation of RCS exposure controls has been varied in effectiveness.

9. AIOH recommendation

Some workplaces may be far from compliant with the current WES 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, except for perhaps the mining industry which is subject to specific regulatory requirements. The lack of exposure data is probably more important for workers such as those in construction and manufacturing. It is in such industries that a combination of increased education and enforcement may produce greatly enhanced benefits.

It should be noted that with current sampling and analytical methods (see section 7), the reliable determination of RCS levels less than 0.05 mg/m^3 is possible, but with large analytical and statistical uncertainty, and determining compliance with an action level of 0.025 mg/m^3 is at the very limits of available techniques.

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

Based on the available information, the AIOH recommends that RCS exposure should be controlled to an ALARP**Error! Bookmark not defined.** level to be at all times below an 8-hour TWA guidance value of 0.1 mg/m^3, with the approach of applying a TWA value of 0.05 mg/m^3 as an action level which triggers investigation of the sources of exposure and implementation of suitable control strategies as well as health surveillance. To overcome limitations in analytical sensitivity, full shift monitoring and the use of a NATA accredited laboratory applying specific national analytical methods and reporting requirements is recommended.

Health risk relative to the recommended 0.1 mg/m^3 guidance exposure value and 0.05 mg/m^3 action level relating to the need for controls and health surveillance should be determined by a COH® applying the approaches and compliance decision making process detailed in '*Occupational Hygiene Monitoring and Compliance Strategies*' published by the AIOH (Grantham & Firth, 2014). This systematic assessment of degree of compliance to the above guidance exposure value would ensure that the long-term average exposure of the workforce was less than 0.05 mg/m^3 and the likelihood of detectable silicosis and excess lung cancers should be negligible.

Health surveillance should be regularly performed according to evidence-based standards to include detailed occupational history and task recording, respiratory function testing and radiological assessment, as per SWA (2013b, 2013c) and state (e.g. Queensland WorkSafe, 2018) documentation.

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