



Coal Services Pty Limited

Final Report

Review of the Health Effects associated with Exposure to Respirable Crystalline Silica in Coal Dust

by
Martin Jennings & Martyn Flahive
Martin Jennings & Associates
PO Box 129, West Perth, WA 6872

mail@martinjennings.com.au

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Glossary of Abbreviations used in this Report

| | |
|-------------------|---|
| ACGIH | American Conference of Governmental Industrial Hygienists |
| ATS | American Thoracic Society |
| BMRC | British Medical Research Council |
| CHAN | Chemical Hazard Advisory Note |
| CI | Confidence Interval |
| COPD | Chronic obstructive pulmonary disease |
| COSHH | Control of Substances Hazardous to Health (Regulations) |
| CSPL | Coal Services Pty Ltd |
| CWP | Coal worker's pneumoconiosis |
| CXR | Chest X-ray |
| DFG | Deutsche Forschungsgemeinschaft |
| DOMEWA | Department of Minerals and Energy, Western Australia |
| FEV ₁ | Forced expiratory volume in one second |
| FVC | Forced vital capacity |
| HSC | (UK) Health and Safety Commission |
| HSE | (UK) Health and Safety Executive |
| IARC | International Agency for Research on Cancer |
| IOM | Institute of Occupational Medicine |
| mg/m ³ | milligrammes per cubic meter (of air) |
| MRE | Medical Research Establishment |
| MSDS | Material Safety Data Sheet |
| MSHA | Mine Safety and Health Administration |
| NCWAS | National Coal Workers' Autopsy Study |
| NOAEL | No observed adverse effect level |
| NIOSH | (US) National Institute of Occupational Safety and Health |
| NOHSC | National Occupational Health and Safety Commission |
| PEL | Permissible Exposure Limit (MSHA) |
| PFR | Pneumoconiosis Field Research (British Coal Industry) |
| PMF | Progressive Massive Fibrosis |
| RDS | Respirable Dust Standard (MSHA) |
| RCS | Respirable Crystalline Silica |
| REL | Recommended Exposure Limit (NIOSH) |
| ROS | Reactive oxygen species |
| RPE | Respiratory Protective Equipment |
| RSMR | Relative Standardised Mortality Ratio |
| SMR | Standardised Mortality Ratio |
| sCWP | Simple Coal Worker's (Miner's) Pneumoconiosis |
| TB | Tuberculosis |
| TLV [®] | Threshold Limit Value (ACGIH) |
| TWA | Time Weighted Average |
| UL | Upper Limit |
| VC | Vital capacity |

1. Executive Summary

Coal Services Pty Ltd (CSPL) has commissioned a study by the authors of respirable crystalline silica in coal dust, specifically asking the following questions:

- a) What are the adverse health effects from exposure to crystalline silica in respirable coal dust and are they different in any way to those from exposure to pure crystalline silica?
- b) Is the current NSW exposure standard of 0.15 mg/m³ respirable silica sufficient to prevent the onset of any disease outcome identified in (a)?

The authors viewed current available research and data provided by CSPL on coal dust and quartz exposures and on incidence of diseases in NSW coal miners. Where appropriate, personal contacts were initiated with persons identified as having expertise in the area.

There is considerable evidence to indicate that exposure to pure crystalline silica will result in a number of disease outcomes including silicosis, lung cancer, tuberculosis, chronic airways disease, auto-immune diseases and nephrotoxicity. Of these disease outcomes, silicosis and lung cancer were considered to be the most relevant to this study.

The literature indicates that the toxicity of quartz varies with exposure to freshly fractured surfaces; the presence of a coating of aluminium containing clay minerals; and particle size in relation to surface area. There is also evidence that lung macrophages remove the mineral coating from the surface of the quartz particles over time, leaving potentially pathogenic quartz particles in the lungs, which may cause delayed onset of silicosis or lung fibrosis.

It is well accepted that exposure to coal dust is responsible for disease outcomes such as simple coal worker's pneumoconiosis which may progress to progressive massive fibrosis. Silicosis in coal miners is not as well documented, although there is a notable study by Miller et al (1998) of coal miners at a Scottish colliery where the miners were exposed to high levels of quartz. They found a significant risk of silicosis that increased exponentially with increasing levels of airborne respirable crystalline silica.

Several studies in relation to lung cancer in coal miners were considered and the consensus of opinion amongst researchers is that there is no conclusive evidence that exposure to coal dust containing respirable crystalline silica is linked to lung cancer. There are reports of an association of gastric cancer and coal mining. The research is inconsistent and inconclusive. There does not however appear to be an association between respirable coal dust exposure, weakening any proposition of a link between respirable crystalline silica and gastric cancer.

In considering the NSW exposure standard of 0.15 mg/m^3 , comparison was made to other standards from Australia, the UK and US. The NSW standard for respirable crystalline silica is higher than most other standards with the notable exception of the standard in the WA Mines Safety and Inspection Regulations (1995) of 0.2 mg/m^3 . Despite an assertion that there have been no compensable cases of silicosis in nearly 20 years in WA, the authors do not feel this is a useful measure of effectiveness of control.

It was also noted that there have no reported cases of CWP or silicosis in 18 years among NSW coal miners. However, in the authors' opinion this warrants closer scrutiny as many miners are not subject to regular health surveillance when they retire or leave the industry. This is particularly relevant to silicosis as there is evidence of delayed onset of silicosis or fibrosis in individuals who are no longer exposed to coal dust.

On reviewing the exposure data provided by CSPL, the authors feel that a small number of coal miners may be exposed to levels of respirable crystalline silica at or above the exposure standard. These would include roof bolters and drillers. Where there are higher exposures, the exposures may be to more pathogenic silica particles with freshly fractured surfaces. These higher risk occupations should be subject to further investigation.

Although there is considerable evidence that the current exposure standard will reduce the incidence of CWP and silicosis, the authors are of the opinion that it is unlikely to prevent silicosis developing in all coal miners over their lifetime. Silicosis is an undesirable outcome, where there is no effective treatment.

In summary the authors were unable to find any evidence that CSPL should not adopt the proposed NOSH exposure standard of 0.1 mg/m^3 for respirable crystalline silica collected under the new Australian Standard for respirable dust (AS 2985-2004). At this stage the authors were unable to find strong evidence to support a more stringent standard for the coal industry.

What are the adverse health effects from exposure to crystalline silica in respirable coal dust and are they different in any way to those from exposure to pure crystalline silica?

2. Introduction

Following an extensive review of the scientific literature on the health effects of respirable crystalline silica by the Occupational and Respiratory Epidemiological Group at the Department of Public Health (University of Western Australia) (de Klerk et al., 2002a), the National Occupational Health and Safety Commission of Australia (NOHSC) has recommended a reduction of the Australian occupational exposure standard for respirable crystalline silica.

The proposed standard is 0.1 mg/m^3 specifically for quartz, cristobalite and tridymite (8hr TWA). This is a reduction from the previous exposure standard of 0.2 mg/m^3 for quartz and the previous standards for cristobalite were 0.1 mg/m^3 and tridymite 0.1 mg/m^3 .

de Klerk et al (2002a) recommended the exposure standard to respirable crystalline silica (RCS) be based upon the prevention of lung cancer with an exposure standard of 0.13 mg/m^3 .

de Klerk et al (2002a) identified that RCS was associated with a number of disease processes including silicosis, pulmonary tuberculosis, bronchiogenic carcinoma, industrial bronchitis with airflow limitation, and auto-immune diseases, including end-stage renal disease. The basis for the exposure standard would be to prevent against increased mortality from lung cancer. Lung cancer was seen as the least acceptable adverse effect from exposure to crystalline silica. Following the risk assessment guidelines set out by the UK Royal Society (Warner cited by de Klerk, 2002a). de Klerk et al 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 after 40 years of exposure and that it was likely to be around 1 per 100,000 or less.

After considering the change in sampling strategies with the new Australian Standard, AS2985-2004 (Standards Australia, 2004), NOHSC has recommended the 8 hour TWA standard of 0.1 mg/m^3 .

de Klerk et al (2002a) recognised that silica exposure in coal dust was a separate entity and required a separate review. They provided reasons that coal dust had different physico-chemical properties with some evidence that coal dust was capable of inhibiting the fibrogenic effect of crystalline silica, and its carcinogenicity. (IARC, 1997, Le Bouffant et al. 1982). The reduced

carcinogenicity of the coal dust has been reflected in a number of epidemiological studies, which will be reviewed further. It was also recognised that the pathologies associated with coal workers' pneumoconiosis (which is associated with the inhalation of coal dust containing RCS) is histologically different from silicosis (Gibbs, 1995). There also exists different exposure standards both in Australia and internationally for exposure to coal dust containing silica.

In the de Klerk et al (2002a) review, the various RCS related outcomes were again reviewed and for development of the exposure standard, de Klerk considered the pooled meta-analysis from ten cohorts of silica-exposed workers (Steenland et al., 2001). This pooled study included 65,980 subjects to expressly examine silica dust exposure and lung cancer risk and included over 1000 cancer cases. The relative risk was 1.064 (95%CI 1.003-1.096) per log (mg/m³-air) of cumulative respirable silica exposure.

An excess lifetime (to age 75) lung cancer risk of 1.8% to 2.8% for 45 years of exposure to a concentration of 0.1 mg/m³ of respirable silica was estimated.

There has been increased interest in the adverse outcomes of silica exposure since the International Agency for Research on Cancer (IARC, 1997) changed its classification of silica from a group 2A carcinogen (that is crystalline silica is probably carcinogenic to humans) to a group 1 classification¹. IARC found there was sufficient evidence that crystalline silica (inhaled in the form of quartz or cristobalite from occupational sources) is carcinogenic to humans. (IARC 1997).

The American Thoracic Society (ATS) subsequently produced a position statement outlining that the effects of exposure to crystalline silica and indicated lung cancer as an associated outcome of exposure. (ATS 1997).

The ATS concluded the following:

- The available data support the conclusion that silicosis produces increased risk for bronchiogenic carcinoma.
- However, 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.

¹ Group 1 classification is that there is sufficient evidence of carcinogenicity in humans.

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

There remains ongoing debate in the scientific community about the carcinogenicity of RCS. Hessel et al. (2000) were critical of the IARC monograph, believing the results of the studies to be inconsistent and, when positive, only weakly positive. Other methodologically strong negative studies have not been considered and several studies viewed as evidence supporting the evidence of carcinogenicity of silica have significant methodological weaknesses. It is noted that this research was facilitated by a grant from the Silica Coalition.

The issue of carcinogenicity has been further reviewed by the UK HSE who indicate that RCS is only weakly carcinogenic (HSE, 2003b).

Meldrum from the UK HSE in a personal communication states *“we think that for occupational risk management purposes, the primary aim should be to protect against silicosis. In the absence of silicosis development, any increased risk of lung cancer (over and above background rates) should be negligible. Of course there are uncertainties throughout the evidence-base, and we cannot be absolutely definitive that there will be no increased risk of lung cancer in the absence of silicosis because the evidence to prove this is impossible to obtain”*. (Meldrum 2004)

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

The ACGIH have based their exposure standard on the prevention of fibrosis and the UK HSE are indicating that they are following a similar approach.

The ACGIH have significantly reduced their exposure standard (TLV) by a factor of 2, from the previous value of 0.1 mg/m^3 to 0.05 mg/m^3 . They state that fibrosis undetected by chest x-ray probably does occur in workers exposed at levels near 0.1 mg/m^3 level. This is based on studies by Hnizdo (1993) who showed that a large percentage (up to 72% exposed to 0.5 mg/m^3 for twenty years) will have a moderate or greater degree of silicosis at autopsy that were not detected radiologically.

3. Silica exposure and coal dust.

Coal dust is a heterogeneous carbonaceous rock formed by the natural elevated temperature and pressure in the Earth's crust (IARC 1997).

Coal comes in various forms and can be classified in various ways such as bituminous coals and anthracite hard coals. The different types of coals vary in their carbon and water content and can be classified as low-rank and high-rank coals. Increasing rank refers to increasing 'coalification', low-rank coals such as lignite and sub-bituminous coals are typically softer, friable and have increased moisture levels, which a lower carbon content. High-rank coals are typically harder and stronger with a darker or black colour and have increased carbon and energy content. Anthracite is the highest ranked coal with the highest carbon content. The major NSW and Queensland deposits are bituminous and have carbon contents ranging from 78 to 91%. (Australian Coal Assn. 2004)

A wide range of other minerals are also found in coal including clays, carbonates, sulphide ores, oxide ores, quartz, phosphates and heavy minerals. (IARC,1997)

Silica exposure in coal dust primarily occurs in the form of quartz. Coal can contain varying levels of quartz. Therefore RCS and silica in coal mining dust is primarily quartz and the two terms will be considered interchangeable in this review.

Quartz exposure in coal mining may arise from exposure to the silica grains contained within the coal matrix, which is released as part of the coal winning processes or from mixed dust originating from fractured rock in the mine floor or roof; mining through stone rolls, dykes, and faults and from work processes including roof bolting.

Quartz levels tend to vary inversely with increasing coal rank. Quartz content is generally greatest in low-rank coal seams. Other minerals including kaolin and mica can constitute up to 14% of the overall dust levels and these also increase with lower rank coals. Quartz levels in the coalmine dust can range between 1.5% to 10.3%, (Crawford et al., 1982).

Tomb et al (1995) reported an extensive program of sampling for crystalline silica in underground mines in the US between 1985 and 1992. The average quartz percentage for the ten underground occupations was 4.7% (range 2.5 to 7%). Roof-bolters who drilled into the rock roof strata were found to have the highest exposure at 7%. In surface mines bulldozer operators and high wall drill operators had high quartz percentages at 12% and 14.9% respectively.

In relation to Australian coalmines, Cram and Glover (1995) reported on samples taken from underground mines in New South Wales between 1984 and 1995. 1.7% of these samples exceeded the respirable quartz content of 0.15 mg/m³.

These figures were thought to be unrepresentative as the only the samples considered to have high quartz levels were sent for analysis (IARC 1997).

Cram (2003) further reported analysis of respirable quartz samples from 1984 through 2001. Of the 46,000 samples taken, 956 (2%) of the samples exceeded the NSW quartz exposure standard at 0.15 mg/m^3 .

When specifically examining the mining types, Cram found that the long wall mines had 3.8% of the samples exceeding the exposure standard. In the Northern Coalfields (Newcastle) 6.3% of the samples exceeded the exposure standard. Cram comments that in the long walls of the Whybrow seam, the high quartz content in the seam requires mine operators to keep dust levels below half the respirable coal dust standard levels to avoid exceeding the quartz standard.

There is also a need to cut into significant amounts of roof on the long wall faces of the Pike's Gully and Liddell seams. In comparison, the Southern Coalfield and Western Coalfield traditionally have higher quartz levels only when long wall faces are cutting roofs, stone rolls or floor stone.

The composition and structure of the surface layers of small but non-respirable silica particles ($10\text{-}20\mu\text{m}$) from the Whybrow seam have been characterized (Gong, Pigram, & Lamb 1999).

They found that silica particles investigated were typically encased in a multilayered shell, the composition of which differs from the average composition of both the silica and the bulk coal. The analysis directly demonstrates the presence of silanol-rich (Si-OH) interfacial layer 3 nm in thickness. This silanol-rich region separates the bulk silica and a complex non-silica layer encasing the particles.

The outer layer encasing the silica particles is 10 nm in thickness and is composed of clays and carbonates, and, in some cases, includes organic material. The elemental constituents of this layer include aluminum, lithium, sodium, potassium, magnesium, iron, and lesser amounts of titanium and copper.

The presence of a silanol rich surface may be important in the toxicity of silica (Fubini 1998). This research indicates that in some NSW coal the silica surface may be encased by a layer of minerals.

The research by Gong et al. (1999) needs to be conducted on smaller particles ($1\mu\text{m}\text{-}7\mu\text{m}$) as Reisner et al. (1988) found that almost all quartz particles greater than $>5\mu\text{m}$ were intergrown with coal or other minerals but with smaller particle size the percentage of non-intergrown particles increased.

4. Human Pathologic Reactions to Pure Crystalline Silica Exposure

The bioavailability of silica in the coal mine dust has been investigated and it has been found that with decreasing coal rank, there is increasing proportions of clay-occluded silica particles (IARC 1997). This would be consistent with the industry experience that even the lower-end coals have higher quartz content, but are not always found to have increasing risk of pneumoconiosis.

There is evidence that freshly fractured quartz dust such from processes such as tunnelling and cutting rock will result in more fibrogenic dust. (Fubini, 1998).

As noted by Fubini (1998), a 'native, uncontaminated quartz surface' i.e. a surface made up of real crystal faces, is very rarely in direct contact with biological matter.

The origin of quartz determines the characteristics of the quartz. Thus in mining, crystalline silica dusts of respirable size are usually generated by grinding macroscopic crystals of quartz. This results in irregularly shaped particles with sharp edges and spikes. The surface produced by mechanical cleavage of chemical bonds is usually very reactive and the state of the surface depends markedly on the grinding procedure and the components of the environment in which the grinding took place. A dry oxygen environment favours the formation of surface radicals and reactive oxygen species (ROS), while a wet one assists full surface hydration at broken bonds with virtually no yield in surface reactive forms (Fubini 1998). The modification of the surface of the quartz surface impacts upon its toxicity and this will be discussed further.

With respect to pure crystalline silica, Donaldson and Borm (1998) state that up to 80% of the animal studies on the quartz in the IARC monograph (1997) use either Min-U-Sil or DQ-12 quartz samples. DQ-12 is reportedly extracted from a Kaolinitic sand deposit in Dorentrüp, Germany and is typically around 87% pure quartz, with the remainder being amorphous silica with small contamination with Kaolinite (IARC 1997). In the case of Min-U-Sil, this is 99.0-99.9% pure silica, with trace contaminants, such as aluminium oxide, titanium oxide and iron oxide present at a total level of <1.0% (US Silica, 2000). Apparently, quartz in coal mine dust or coal fly ash (Borm 1997) is not as biologically active compared to equivalent doses of pure samples of quartz *in vivo* or *in vitro*.

Variability in the toxicity of silica

There is a growing body of evidence to suggest that the potency of silica in the causation of silicosis varies according to the circumstances of the exposure. This has implications for targeting regulatory activities to those industries and processes of most concern in relation to crystalline silica (Meldrum & Howden, 2002).

As noted by Donaldson and Borm (1998), that very few studies utilise quartz that is representative of the kinds of quartz used in the majority of industries where there is occupational exposure to quartz. Donaldson and Borm go on to say that it is well known that a vast amount of the exposures to quartz are to quartz in mixed dusts where it is relatively likely that modification of the surface could occur when substances such as iron or aluminium present.

Variability in fibrotic potency appears to depend on:

1. Polymorphic type of crystalline silica

Cristobalite and quartz appear to be more reactive and more cytotoxic than coesite, shishovite and tridymite. Cristobalite is only encountered in the conversion of quartz silica under high temperatures in kilns. The difference in the reactivity and toxicity have been attributed to the presence of silanol groups (SiOH) protruding from the surface of the crystal.

However, the weight of evidence from in vivo and in vitro studies shows no differences in the cytotoxic, inflammatory or fibrogenic properties of these polymorphs, i.e. quartz and cristobalite.

2. Presence of other minerals

There is experimental evidence, animal and human evidence, consistently indicating that the toxic effects of quartz are reduced in the presence of aluminium containing clays. In some coal mines, the quartz present may be coated with aluminium containing clay minerals such as kaolinite and illite. Meldrum & Howden (2002) suggest that as coal is formed, the surface of quartz grains in dirt bands associated with coal strata can become coated or intergrown with clay minerals. This reduces the amount of 'free' or unexposed quartz surface present. These quartz grains may be liberated and not fractured during mining activities and thereby retain their clay mineral coating. Hence, it may be the amount of 'free' quartz surface, rather than the total amount of quartz present in respirable coal dust, which is of relevance to the risk of coal worker's pneumoconiosis or silicosis.

Interestingly, Meldrum and Howden also suggest that the protective effect of the aluminium coating on the surface of the quartz may be lost over time, as aluminium is cleared more quickly from the lungs than is quartz. The retained 'clean' quartz then exerts a pathogenic effect. *This implies that when co-exposure to these minerals ceases, the possibility of delayed onset silicosis developing actually increases.*

This is extremely significant to coal workers, and points to the need to determine the time of onset of silicosis in former coal miners, as compared to miners still engaged in work. However, Meldrum & Howden state that there is a lack of clear human evidence on this point.

In a study by Rainey et. al (1996), dusts recovered from macrophages extracted by lavage from the lungs of three coal miners with long term exposure to coal mine dusts were compared with dusts obtained from typical mining environments, that is, respirable sized particles formed by grinding mineral samples from roof bolter boxes of coal mines. It was found that the composition and morphology of silica-based particles recovered from macrophages was clearly different from that of equivalent particles found in the roof bolter boxes. The macrophages were found to contain highly pure and crystalline particles of quartz not found in the mineral dust samples. The authors concluded there was likely to be differential dissolution of elements from the surface of quartz based particles, or more efficient removal from the lungs of aluminium containing particles, or that the results were an experimental artefact of the small number of miners sampled. Nevertheless, this study provides support for the notion that the chemical composition of the surface of inhaled quartz particles does not remain constant during long term residence in the lungs.

3. Particle number, size and surface area

As suggested above in the discussion regarding the amount of 'free' quartz surface, there is also some evidence to support the view that the total surface area of dust retained in the lungs is an important determinant of toxicity. The smaller the particle, the larger the surface area per unit mass when compared with larger sized particles. Hence, smaller particle size fractions (very fine dusts) of respirable crystalline silica would be expected to produce more lung damage than equal masses of larger respirable size fractions. From this, it may be inferred that there would be a greater risk of silicosis in workers exposed to very fine particles of crystalline silica, such as might be found in silica flours, than to those exposed to equal masses of larger sized respirable particles.

The implication of this for coal miners is that those occupations exposed to finer particles, such as drillers may be more at risk than those miners exposed to dust generated by other mining processes.

Fubini (1998) states that if ROS, as hypothesised, are implicated in some stages of the pathogenic mechanism, then mining and processing the same ore with different procedures may generate dusts differing in their pathogenic potential. Prolonged milling progressively converts the outer

parts of the particles from crystalline to amorphous, which lowers the dust toxicity.

With the introduction of an increased sampling pump flow rate of 2.2 l/min in the new respirable dust standard, AS 2985-2004 (Standards Australia 2004), to align the respirable sampling curve with the fraction described in ISO: 7708, there will be greater proportion of smaller particle sampled, than by samplers following the BMRC curve, as required in AS 2985-1987. The implication is that these samples may be more indicative of the cytotoxic risk. There may be some difference between the gravimetric results as they will be collecting smaller particles rather than larger particles. Grantham (de Klerk, 2002a) has discussed this at length and recommends a possible conversion factor of 1.4 from BMRC data to the ISO 7708 curve. However, there may be some specific issues in coal mining (higher ventilation rates to reduce dust levels, and different density of coal particles) (Cartwright, 1965) that makes a field study appropriate.

4. Variability between freshly fractured and 'aged' surfaces.

Freshly cleaved crystalline silica particles form smaller fragments, with freshly generated particles surfaces. These fragments have reactive free radical species on the freshly formed surfaces. This leads to an increase in cytotoxicity in short term *in vitro* tests.

Castranova et al. (1996) have demonstrated an enhanced pulmonary response in *in vivo* tests with freshly fractured silica surfaces. Male Fischer 344 rats were exposed for 2 weeks to 20 mg/m³, 5 hours/day, 5 days/week to freshly fractured α -quartz, as opposed to the control group which was exposed to aged dust, which had been milled and then stored for 2 months. The results showed that inhalation of fresh silica showed greater toxic and inflammatory pulmonary reaction than aged silica.

Overall, there are grounds to conclude that occupational exposures to freshly cut surfaces of crystalline silica will pose greater health risks than exposures to 'aged' surfaces. There is evidence that the activity of free radicals decays with time and that this decay process may be accelerated in the presence of water. Therefore, the use of wet processes will quench the formation of free radicals at the freshly cut surfaces, although it is not known to what extent the time between dust generation and inhalation or the effectiveness of the wetting will reduce the cytotoxic properties of the quartz.

There may be some relevance for this in mining in that dust generated from primary dust generating processes, such as drilling, blasting, digging, crushing and grinding is likely to be more active toxicologically than is dust generated from secondary sources, e.g. from resuspension of road dust,

or from dust packaging, distribution or transport of silica containing materials. It may be possible to differentiate between different exposure groups and determine whether one group is more at risk than another, although the quantification of the risk at present is unclear.

5. Reported Health Effects for Pure Crystalline Silica

Min-U-Sil is in essence, pure crystalline silica. This is used in the majority of animal studies assessing the health effects of pure crystalline silica.

The US Silica MSDS for Min-U-Sil adequately describes the potential health effects for pure crystalline silica as being:

Inhalation:

- a. Silicosis - Respirable crystalline silica (quartz) can cause silicosis, a fibrosis (scarring) of the lungs. Silicosis may be progressive; it may lead to disability and death.
- b. Cancer - Crystalline silica (quartz) inhaled from occupational sources is classified as carcinogenic to humans.
- c. Autoimmune Diseases - There are some studies that show excess numbers of cases of scleroderma and other connective tissue disorders in workers exposed to respirable crystalline silica.
- d. Tuberculosis - Silicosis increases the risk of tuberculosis.
- e. Nephrotoxicity - There are some studies that show an increased incidence of chronic kidney disease and end-stage renal disease in workers exposed to respirable crystalline silica.

Eye Contact: Crystalline silica (quartz) may cause abrasion of the cornea.

Skin Contact: Not applicable.

Ingestion: Not applicable.

Chronic Effects: The adverse health effects -- silicosis, cancer, autoimmune diseases, tuberculosis, and nephrotoxicity -- are chronic effects.

Signs and Symptoms of Exposure: Generally, there are no signs or symptoms of exposure to crystalline silica (quartz).

Medical Conditions Generally Aggravated by Exposure: The condition of individuals with lung disease (e.g., bronchitis, emphysema, chronic obstructive pulmonary disease) can be aggravated by exposure.

Exposure to crystalline silica can result in adverse pulmonary responses such as acute silicosis, accelerated silicosis, chronic silicosis, and conglomerate silicosis. In addition, silica exposure may also be associated with systemic and autoimmune diseases such as scleroderma, rheumatoid arthritis, systemic lupus erythematosus, nephropathy, and proliferative glomerulonephritis. Tuberculosis is a common complication of silicosis often seen in severe grades of the disease. A possible association between silicosis and lung cancer is being accepted on the basis of evidence for a role of silica exposure in increased lung tumour formation in experimental animals and exposed human populations (Castranova & Vallyathan 2000).

6. Silica Exposure and Cancer

The monograph published by IARC concluded that there is now sufficient evidence in humans for the carcinogenicity of inhaled crystalline silica in the form of quartz or cristobalite from occupational sources (IARC 1997). Several studies among the many reviewed by the IARC working group on the question of silica exposure and cancer risk in humans were negative or equivocal, and carcinogenicity of silica was not detected in all industrial operations. However, nine studies showed excessive risk for lung cancer. These included refractory brick workers, pottery workers, diatomaceous earth workers, foundry workers, granite workers, and mine workers, (although not coal-mine workers). It appears that the carcinogenic property of crystalline silica may be dependent on its biologic activity, polymorphic nature, or specific industrial processes such as heat treatment and mechanical grinding. The relationship between the ability of silica to generate ROS and carcinogenesis has recently been reviewed (Castranova & Vallyathan, 2000).

Animal Studies on Coal Dust and Silica

The IARC working group reviewed a small number of animal studies in their assessment of coal dust carcinogenicity. Coal dust was tested both separately and in combination with other toxic particles. The IARC working group concludes that one experiment was adequate for assessment and there was no increase in tumours compared to controls.

No lung tumours were reported in rats exposed to coal mine dust at 6.6 and 14.9 mg/m³ (six hours per day, five days a week) for up to 20 months (Karagianes, Palmer, & Busch 1981) despite large deposits of coal dust in the macrophages and interstitium.

In one study an increased number of lung tumours has been reported after very high doses (1000 mg) of coal dust for 24 months (five hours per day, five days per week). However, the incidence of tumours was not significant due to a small number of controls. (IARC, 1997)

Lung tumours have been found after repeated intratracheal instillations of five different coals of doses at 360 and 120 mg (Borm et al., 2000, Pott et al., 2000). But however, the quartz content was negatively associated with the tumour rate with the highest quartz content (16.7%) associated with the lowest tumour rate.

Another intratracheal instillation study (50 mg single instillation) of 30 different coal mine dusts with different quartz content (0.3 to 15.1% quartz) did not result in any lung tumours. (Reisner et al. 1982; Rosmanith et al. 1982)

Although there is some inconsistency with the higher-dose studies, there appears to be a consistent negative effect in relation to coal dust containing quartz in the animal studies. This is in contrast to the numerous animal studies conducted with silica in rats where the IARC working group (1997) found that several studies showed significant increases in the incidence of lung cancer through inhalation or intratracheal instillation of quartz and intrapleural or intrapeneal injection of quartz resulted in the thoracic malignant lymphomas. They concluded that there was sufficient evidence in experiments on animals for the carcinogenicity of quartz and cristobalite.

Lung Cancer in Coal Miners

Lung cancer in coal miners occurs less frequently than in the general population after adjustment for age and smoking. Epidemiologic studies of British and U.S. coal miners reported a lower risk of lung cancer for miners compared to that in non-miners, and there was no apparent influence of mining tenure on the prevalence of lung tumours. There were also no changes in the histopathology of lung cancer cell types in coal miners, a point of view critically evaluated to assess the relationship of smoking. The tumours were mostly squamous cell (30%), adenocarcinoma (27%), and small cell (26%), again showing no influence of mining tenure on the frequency of these cell types. From these histopathologic studies, it is evident that there are no apparent cellular differences in lung cancer of coal miners who smoke and the cigarette-smoking general population.

IARC reviewed a number of cohort studies assessing the risks of cancer in coal miners. They included reviews by Goldman, (1965) who presented a mortality survey in miners and ex-miners and employed by National Coal Board in the United Kingdom. From men aged 20 to 65, the SMR for underground workers was 0.7 (95% CI, 0.61-0.8%) for lung cancer and 1.02 (CI 0.932-1.12) for all other neoplasms. For all other coal miners, the SMR was 0.74 (95% CI 0.65-0.83). As part of the same study, Goldman (1965) also assessed the risk of lung cancer mortality in 5096 male coal miners and ex-miners. A lower than expected lung cancer mortality risk was found and no association was seen with radiologic categories of pneumoconiosis and lung cancer. In relation to lung cancer miners with grade 0 the SMR was 0.87 (95 CI 0.5-1.4). The SMR for grades 1 to 3 pneumoconiosis was 0.57 (95 CI 0.21-1.24) and the SMR for miners with progressive mass or fibrosis was 1 (0.43-1.96). The relevance of this study is that it was conducted when dust exposures were high.

Other notable studies reviewed included a necropsy study on Lancashire coal miners published by Rooke et al. (1979). This study found that the prevalence of lung cancer in coal miners and ex-miners was no greater than the male population in the Northwest of England and there was no association between the presence of pneumoconiosis and lung cancer. A further large cohort reported by Liddell (1973) assessed 5362 deaths in 1961 amongst coal miners.

There was a lower percentage of death from lung cancer (8.8%) compared with men nationally (13.2%), the SMR for base workers was 0.49. The deficit in lung cancer rates was not counterbalanced by excesses in mortality from other neoplasms.

Ortmeyer et al. (1974) also conducted a mortality follow-up study of 2549 miners employed between 1963 and 1965 and 1177 ex-miners from the Appalachian region of the United States. The SMR was found to be elevated for ex-miners at 1.19 (95% CI 1.1-1.39) with a high SMR also measured for complicated pneumoconiosis. The lung cancer mortality SMR was less than the national rate at 0.67 (95% CI 0.43-0.99).

A large cohort mortality study of 23,232 coal miners was conducted by Rockette (1977). Follow-up was conducted for the years 1959 to 1971 and comparisons were again made to the United States mortality rates. There were excesses in the mortality rates for non-malignant respiratory diseases especially pneumoconiosis at 9.26 (95% CI 7.98-10.68). Mortality was also elevated to a certain extent amongst coal miners for stomach cancer of SMR 1.4 (95% CI 1.17-1.66) and lung cancer SMR 1.13 (95% CI 1.02-1.26).

Another mortality study considered was amongst the regions of the Rhondda Fach coal mining community in Wales, United Kingdom (Cochrane et al. 1979). The population of 6212 male miners and ex-miners and 2138 male non-miners. Amongst the miners and ex-miners, there was elevations of mortality from all causes with the SMRs ranging from 1.16 to 1.94, no excesses were found for lung cancer in either miners or non-miners.

In Western Australia, Armstrong et al. (1979) conducted a mortality study of 213 male coal miners during the years 1961 to 1975. 97% of the 213 coal miners worked underground and 99.5% had at least 10 years of mining experience. Compared to Western Australian mortality rates male miners, had an excess mortality from all causes with SMR of 1.25 (95% CI 0.93-1.62). There was again a deficit of mortality from lung cancer (SMR 0.2).

Miller & Jacobsen (1985) conducted a mortality follow-up study of 26,363 male coal miners from 20 collieries in England and Wales. The mortality was compared to national rates, which the IARC working group felt limited the interpretation of the results. However, it was found that the lung cancer mortality rate was again seen to be lower for men with simple pneumoconiosis and 26% lower for those with PMF.

A mortality study of pneumoconiosis cases in Netherlands during 1956 through 1960 (Meijers et al. 1991) found a small and non-significant of elevation for lung cancer with an SMR of 1.31 (95% CI 0.71-2.05) although there was an elevated SMR for all cause mortality (SMR 1.53).

Swaen, Meijers, & Slangen (1995) followed a cohort of 3790 coal miners in the Netherlands from the 1950s to 1992 and excess of mortality for all causes was found SMR 1.27 (95% CI 1.23-1.32) with an excess of mortality from non-malignant respiratory disease SMR 4.1 (95% CI 3.82-4.41) and small intestinal cancer SMR 1.47 (95% CI 1.22-1.76) were observed. No excess for lung cancer was observed with an SMR of 1.02 (95% CI 0.90-1.15).

Kuempel et al. (1995) reported a mortality follow-up on 8878 in United States coal miners that had been initially examined in 1969 to 1971 as part of a national study of coal workers' pneumoconiosis. Mortality from all causes was lower than expected from national rates with an SMR of 0.85 (95% CI 0.79-0.91) although there was an excess mortality from pneumoconiosis and other respiratory diseases with an SMR of 3.72 (95% CI 2.89-4.71). The mortality from lung cancer was lower than expected with an SMR of 0.77 (95% CI 0.6-0.99) a negative exposure response trend was found for lung cancer. No excess was seen for stomach cancer rates with an SMR of 0.91 (95% CI 0.39-1.8). A dose-response gradient was slightly positive for stomach cancer but not statistically significant.

The IARC working group concluded that the evidence from occupational cohort studies showed the association between coal mine dust and lung cancer had not been consistent with some studies revealing excess risk, whereas, other studies indicated a cohort-wide lung cancer deficit (IARC 1997).

Morfeld et al. (1997) conducted a study of a group of 4628 coal miners of the Saar in Germany, all of whom had worked for at least 5 years underground, with an average of 29 years. The authors focussed on the Saar region as the respirable coal dust in that region has a high quartz content (about 12%) and because that region possesses the only reliable population based cancer registry, which contained information on the mortality from each cause and morbidity for each cancer type in that area.

In the study period between January 1 1980 and December 31 1991, there were 317 deaths. It was found that of these, 104 were caused by cancer and 41 were from lung cancer. Overall mortality, cancer mortality and lung cancer mortality were significantly lower than expected. The SMR (with 0.95 confidence limits) was estimated as 0.63 (0.6 – 0.7) for overall mortality, 0.65 (0.5 – 0.8) for cancer mortality, 0.70 (0.5 – 1.0) for lung cancer mortality, 0.62 (0.2 – 1.4) for stomach cancer mortality and 1.27 (0.2 – 2.2) for malignancies of the lymphatic and hematopoietic tissue.

On reviewing these lung cancer studies, it appears that the larger cohort studies (that give the greater weight of evidence) suggest coal dust exposure is not associated with an excess of lung cancer mortality. This was confirmed in a meta-analysis of lung cancer in coal miners (Morfeld et al.1999).

In the meta-analysis of lung cancer from 14 studies in mortality on coal miners, as shown in Table 1, Morfeld et al. showed a pooled relative risk of 0.96 (0.92-1.00), but it was noted that all follow up studies were negative, whereas the odds-ratio case-control studies demonstrated an elevated lung cancer risk.

More recent case-control studies in France (Auburtin, 1998) and Germany (Morfeld et al., 1997, 1999) have shown a significant increase in lung cancer in miners with coal workers' pneumoconiosis and suggest that the effect of increased lung cancer may have been masked by the healthy workers' survivor effect. The study from German hard coal mines (Morfeld, 1999) concluded that they did not find evidence of a link between coal dust exposure and lung cancer.

Miller et al. (1997) analysed over 408 000 man-years of follow up to the end of 1992, assessing 18000 men and over 7000 deaths from British coal mines. The study included an extensive sampling program over a twenty-year program from the end of the 1950s.

The authors found a clear relationship between mortality from pneumoconiosis and exposure to respirable coal dust, and this was a better predictor than respirable quartz. They were not able to find a consistent relationship between quartz and lung cancer.

The authors concluded, *"these analyses were based on a large cohort with detailed occupational records and therefore had considerable power to identify occupational risks. This is exemplified by the strong relationship between dust exposure and pneumoconiosis. It is unlikely that a consistent association between quartz (present in relatively low concentrations in mixed coal dust) and lung cancer would have been missed."*

A further cohort study of 2579 miners followed up over a 20-year period (Isidro, I et al. 2004) did not identify any excess in the lung cancer rate. They defined two groups, one being all workers who were exposed to an average dust level of 2.5 mg/m³ with 17.5% silica and coal extractors exposed to an average of 3.4 mg/m³ of dust with 8% silica. Pneumoconiosis (category 1 or greater) was detected in 3.8% of the cohort.

Recent cohort studies over NSW coal miners have not revealed an excess lung cancer. (Christie et al. 1995). An inception cohort of 23 650 NSW coal miners were followed between 1973 and 1992. 491 deaths were reviewed and no cancer excess was identified (SMR, 0.78 CI 0.62-0.95). Follow up of the cohort up to 1997 again has revealed a deficit of lung cancer (SIR 0.65 CI 0.48-0.86) (Kirby et al. 2000).

There are some inconsistencies in some of the studies on coal mining and (predominately the case-control studies that are more subject to bias and error), however, the large epidemiological studies provide strong evidence that

exposure to coal dust (containing respirable crystalline silica) in coal mining is not associated with the development of lung cancer.

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A Review of Adverse Health Effects from Exposure to Crystalline Silica in Coal Dust

Table 1 Combined SMR and RSMR Estimates with 0.95 Confidence Intervals Based on the Sum of Observed and Expected Numbers from 14 SMR Studies on Coal Miners (Reproduced from Morfeld et. al., 1997)

| Reference Number | Study Details | Overall Mortality | | Cancer Mortality | | Lung Mortality | Cancer |
|----------------------------------|---|-------------------|------------------|------------------|------------------|----------------|------------------|
| | | Obs (exp) | SMR (0.95 CI) | Obs (exp) | SMR (0.95 CI) | Obs (exp) | SMR |
| 1. Enterline 1972 | All coal miners (20-64 years old) in US Public health Service 1950 (US) | 5793 (2966.0) | 1.95 (1.9-2.0) | 764 (428.0) | 1.79 (1.7-1.9) | 161 (84.0) | 1.92 (1.6-2.2) |
| 2. Ortmeyer et al. 1973 | 4004 coal miners comp. for CWP 1959-1961 (US) | 1455 (1118.0) | 1.30 (1.2-1.4) | - | - | - | - |
| 3. Costello et.al. 1974 | 3726 coal miners 1962-1971 (US) | - | - | - | - | 24 (36.0) | 0.67 (0.4-1.0) |
| 4. Ortmeyer et al. 1974 | 3577 coal miners (bituminous coal) 1963-1971 (US) | 533 (490.4) | 1.09 (1.0-1.2) | - | - | - | - |
| 5. Rockette 1977 | 23,232 coal miners in UMW Health and Retirement Fund 1959-1971 (US) | 7741 (7583.6) | 1.02 (1.0-1.04) | 1243 (1254.0) | 0.99 (.09-1.1) | 352 (310.9) | 1.13 (1.01-1.3) |
| 6. Armstrong et.al. 1977 | 213 coal miners (soft coal) 1961-1971 (Australia) | 54 (43.5) | 1.24 (0.9-1.6) | 18 (9.6) | 1.88 (1.1-3.0) | 1 (4.0) | 0.25 (0.01-1.4) |
| 7. Atuhaire et. al. 1985 | 5963 coal miners (20-74 years old) of Rhondda Fach 1950-1989 (UK) | 3865 (3002.5) | 1.29 (1.25-1.33) | 592 (604.1) | 0.98 (0.9-1.1) | 191 (246.1) | 0.78 (0.7-0.9) |
| 8. Maclaren 1992 | 12,361 coal miners 1960-1989 (UK) | 5852 (6092.2) | 0.96 (0.9-1.0) | - | - | 521 (599.7) | 0.87 (0.8-1.0) |
| 9. Christie et.al. 1995 | 23,630 coal miners 1973-1992 (Australia) | 491 (642.9) | 0.76 (0.7-0.8) | 92 (118.7) | 0.78 (0.6-1.0) | - | - |
| 10. Kuempel et.al. 1995 | 8878 coal miners 1969-1979 (US) | 793 (937.5) | 0.85 (0.8-0.9) | - | - | 65 (84.1) | 0.77 (0.6-1.0) |
| 11. Swaen et. al. 1995 | 3790 pneumoconiotic coal miners 1955-1992 (Netherlands) | 2941 (2314.3) | 1.27 (1.2-1.3) | 668 (690.5) | 0.97 (0.9-1.1) | 272 (266.7) | 1.02 (0.9-1.2) |
| 12. Une et. al. 1995 | 1976 coal miners 1987-1994 (Japan) | 169 (140.9) | 1.20 (1.0-1.4) | 88 (58.6) | 1.50 (1.2-1.9) | 19 (12.5) | 1.52 (0.9-2.4) |
| 13. Starzynski et. al. 1996 | 7065 pneum. Coal miners 1970-1991 (Poland) | 1995 (1830.0) | 1.09 (1.0-1.1) | 468 (463.4) | 1.01 (0.9-1.1) | 179 (167.3) | 1.07 (0.9-1.2) |
| 14. Morfeld et. al. 1997 (above) | 4578 coal miners of Saar area 1980-1991 (Germany) | 317 (506.3) | 0.63 (0.6-0.7) | 104 (160.9) | 0.65 (0.5-0.8) | 41 (58.6) | 0.70 (0.5-1.0) |
| Total No. 1-14 Combined SMR | | 31716 (27468.3) | 1.15 (1.14-1.16) | 3980 (3733.1) | 1.07 (1.04-1.10) | 1800 (1849.3) | 0.97 (0.93-1.02) |
| Combined RSMR | | - | - | 3980 (4310.4) | 0.92 (0.89-0.95) | 1800 (2135.3) | 0.84 (0.80-0.88) |
| Total No. 2-14 Combined SMR | | 25923 (24502.3) | 1.06 (1.05-1.07) | 3210 (3305.1) | 0.97 (0.94-1.00) | 1639 (1765.3) | 0.93 (0.89-0.98) |
| Combined RSMR | | - | - | 3210 (3496.7) | 0.92 (0.89-0.95) | 1639 (1867.7) | 0.88 (0.84-0.92) |

Gastric Cancer in Coal Miners

In contrast to lung cancer, epidemiologic studies have revealed a higher-than-normal incidence of mortality from gastric cancer in coal miners compared to that in non-miners. A significant relationship between cumulative dust exposure and increased mortality from cancers of the digestive system was also evident from some of these studies. The mechanism suggested was that nitrosation of ingested coal dust in the acidic gastric environment could result in the production of carcinogenic products, which may lead to the higher incidence of gastric cancer in coal miners. In support of this hypothesis, it was shown that upon nitrosation of coal dust extracts they become mutagenic and are able to induce neoplastic transformation of mammalian cells (Castranova and Vallyathan 2000).

Raj et. al. (2003) briefly reviewed gastric cancer among coal miners. They refer to a study conducted in 1961 by Stocks, which analysed data from the Registrar General's Decennial Supplements of occupational Mortality. Gastric cancer was reported as being one of the six commonest causes of mortality in coal workers, in England and Wales between 1949-1953. Rockette is reported by Raj et al., to have concluded that gastric cancer is one of the three commonest causes of death in coal miners. Other studies have supported this view, but others have failed to support these findings. However, it appears that the coal component, rather than the silica in the dust mixture is generally regarded as being the responsible agent.

The large epidemiological study by Miller et al. (1997) demonstrated an increased risk of gastric cancer with an SMR of 124% (95% C110-141). No other cancer site showed increased risks. *No relationship was seen with dust exposure.* As the increased risk could not be explained by the inhalation and deposition of respirable coal dust or other direct occupational exposure, they felt that it may be secondary to social class. Cancer rates vary between social class and miners are categorised as social class III although their lifestyles are more typical of social class IV.

The SMR for Class III in 1971 were 109 and 125 for Class IV. They suggested that the risks were similar to those of the same social class but recommend further research in mining communities was warranted.

There have been other studies that have reported elevated mortality among coal miners from stomach cancer. Enterline, as cited in the ACGIH Documentation for Coal, (ACGIH 2001) studied mortality rates among U.S. coal miners using occupational coding of death certificates by the National Vital Statistics Division of the U.S. Public Health Service for the year 1950. A population of 391,620 coal miners, age 20 to 64, was studied. While death rates among coal miners were above those for all working males for every cause of death except diabetes, the greatest excess was for diseases of the respiratory system, cancer of the

stomach, and accidents. The SMR determined for stomach cancer was 275. Factors associated with stomach cancer such as diet, cigarette smoking, chewing tobacco, and coal dust exposure were not studied.

In summary there are reports of an association of gastric cancer and coal mining. The research is inconsistent and inconclusive. There does not however appear to be an association between respirable coal dust exposure, weakening any proposition of a link between respirable crystalline silica and gastric cancer

7. Silicosis and Coal Dust

Silicosis

Silicosis is the fibrotic lung disease caused by the inhalation of crystalline silica. It has been described in three forms, acute silicosis, subacute silicosis, and chronic silicosis.

Acute silicosis

Acute silicosis occurs when subjects are exposed to very high concentrations of silica over a period of usually a few weeks to four or five years. It is usually rapidly progressive with a presentation of progressive shortness of breath, fever, cough and weight loss. Death normally occurs from hypoxic respiratory failure and the fatal course of the disease is not influenced by treatment. This disease is primarily reported in occupations that can have very high exposures to fine silica dusts and include sandblasters, stone crushers, ceramic workers, silica flour workers, and workers in abrasive manufacturing. (Seaton 1995) Although high levels of silica dust is unlikely in coal mine dust exposure because of the low silica percentage in the dust, acute silicosis has been reported in surface coal mine drillers (Banks et al. 1983).

Subacute silicosis (accelerated silicosis)

In some occupations, exposure to high concentration silica over as little as five years result in a rapidly progressive form of silicosis. The principal feature is an early presentation of breathlessness followed by rapid deterioration to hypoxia with little in the way of physical signs. The accelerated silicosis primarily results from exposure to high concentrations of finely divided silica from transfer to sandblasting, production of silica flour, and stone masonry involving power tools.

Chronic silicosis

This is the most usual form of silicosis that normally occurs after many years of exposure to low levels of silica-containing dust.

Over time, a slowly nodular appearance is seen on the chest x-ray and this is not usually associated with any symptoms or physical signs. Initially it is indistinguishable from coal workers' pneumoconiosis. (Seaton 1995) There is frequently association with cough, sputum, and breathlessness, although these symptoms are more likely related to the associated disease of the airways.

The defining issue with simple radiological silicosis is that in comparison to coal workers' pneumoconiosis, it is a more progressive disease and even in the

absence of further dust exposure, increasing fibrosis can occur resulting in increasing disability. There is no effective cure and treatment is primarily supportive.

A condition called progressive massive fibrosis (PMF) can occur in silicosis or where there has been mixed dust exposures.

When progressive massive fibrosis occurs, the patient develops progressive respiratory symptoms from reduction in lung volumes, distortion of bronchi, and bullous emphysema. The main symptom is shortness of breath, which is progressive and ultimately disabling, potentially leading to cardiorespiratory failure.

Pathology of Silicosis

The pathology of silicosis has been described as the presence of discrete, rounded and whorled hyalinised fibrous nodules that are sharply separated from the surrounding lung tissue. These nodules are more frequently reported with exposure to dusts containing more than 18% silica (Gibbs, 1998).

In coal workers exposed to significant concentrations of quartz within the coal dust, lesions that are intermediate between the typical coal nodule and silicotic nodules have been described (mixed dust pneumoconiosis).

Microscopically the lungs are found to be thickened with fibrous tissue and the hilar lymph nodes are frequently enlarged, fixed, and frequently calcified. Cut sections of the nodes exhibit a whorled grey pattern. PMF where there has been predominantly silica exposure, lesions consist of fused whorled silicotic nodules and cavitation is not infrequent as a result of ischaemia or mycobacterial infection.

Classical silicosis is not generally reported as an issue in coal workers, as the exposure to silica is generally in a mixed dust containing less than 10% silica. It has been reported in coal miners exposed to dusts with high levels of silica i.e. rock bolters (Tomb et al. 1986). Most studies that have reported silicosis in coal miners involve exposure to freshly cut quartz and high proportions of quartz. (>10%). (Miller et al, 1998; Morfeld, 1998).

In 1998 in the USA there were 14 cases of silicosis (eight underground; six surface miners) on a background of 224 cases of CWP and PMF. (Mattos 1998, cited in Federal Register).

Coal Workers' Pneumoconiosis and Pathology

Simple coal workers' pneumoconiosis (CWP) is normally diagnosed through an abnormal chest x-ray. The severity of the disease is normally classified by the number of size and shape of small opacities on chest radiograph. The most

common abnormality seen is the presence of multiple small rounded opacities predominating in the upper zones. There also has reported the presence of small irregular opacities that may be associated with reduced lung function. (Cockcroft et al. 1983).

The pathology of coal workers' pneumoconiosis is recognised as a separate entity from silicosis. (Gibbs 1998).

Most miners with simple CWP have no symptoms or physical signs. Shortness of breath is normally not seen with the simple CWP. Studies of miners with CWP have demonstrated a reduction in FEV₁ (Seixas et al. 1993).

Complicated CWP or progressive massive fibrosis is associated with pulmonary disability and premature death. It normally develops in the background of severe simple CWP, categories 2 and 3. PMF may result in severe airways obstruction, restrictive lung defects, and congestive heart failure.

Pathology

The pneumoconiosis of coal workers is categorised into simple and complicated forms. Simple coal worker's pneumoconiosis (sCWP) corresponds to macular nodule lesions whereas complicated CWP corresponds with progressive massive fibrosis (PMF) and the relations of rheumatoid pneumoconiosis (Caplan's syndrome).

At autopsy, numerous coal macules measuring from 0.5 mm to 6 mm are seen throughout the lung, although the density is greatest in the upper lobes and in the upper portions of the middle lobes. Microscopically the macule is composed of coal dust-laden macrophages within the walls of respiratory bronchiole and adjacent alveoli. Surrounding the air spaces around the macula are usually enlarged (focal emphysema). This focal emphysema is especially common to be considered an integral part of coal workers' pneumoconiosis and is a form of centriacinar emphysema (Green 1998). Focal emphysema is seen in miners who have never smoked although smoking miners usually show more severe centriacinar emphysema.

In addition to the macules, firm nodules are also seen within the lung, primarily confined to the upper zones and can range in size from up to 7 mm to 20 mm. Histologically, the nodules are full of macrophages in a fibrotic stroma comprised of collagen and reticulin in situations of high quartz exposure, the collagen is arranged concentrically.

In the peribronchial hilar, paratracheal and occasionally abdominal lymph nodes, are found to be black and firm; microscopically the lymph nodes show large numbers of pigmented histocytes and variable degrees of fibrosis. The silicotic nodules are frequently observed in the tracheobronchial lymph nodes of coal workers usually in the absence of silicotic lesions within the pulmonary

parenchyma. This appears to be from a selective of silica enrichment of quartz within the pulmonary lymph nodes.

Lesions typical of silicosis have also been identified in autopsy studies. 12% of cases submitted to the National Coal Workers' Autopsy Study (NCWAS) demonstrated pathological lesions of silicosis (Green 1988).

In one post mortem study of 430 coal miners from West Virginia (Vallyathan et al. 1996) 15% had silicosis lesions identified.

In an Australian autopsy study 2% had moderate changes of silicosis, 11% had changes of mixed dust pneumoconiosis. (mixed silica and coal dust exposure) (Leigh et al. 1994). The relevance of this data to current exposures is unclear as the study involves miners who died between 1966 and 1983 and it is noted the possible bias of post mortem studies.

| Type of Lesion | Percent showing lesion |
|------------------------------|------------------------|
| Macules | 45.6 |
| Macules with focal emphysema | 35.8 |
| Nodules (CWP type) | 18.9 |
| PMF | 5.5 |
| Silicotic Nodules | |
| Lung Parenchyma | 12.6 |
| Tracheobronchial lymph nodes | 52.9 |

National Coal Workers' Autopsy Study NIOSH Analysis on 3365 cases.

Progressive Massive Fibrosis and Pathology

Microscopically, the PMF lesions appear as a black fibrotic mass that may be round, irregular or oblong shape. They are usually well demarcated from the surrounding lung.

Peripheral lesions are usually firmly tethered to the parietal pleura and some tracings contract towards the hilum.

The characteristic feature of PMF is the tendency of the lesion to transgress normal anatomical boundaries with fissures, bronchi, and vessels becoming obliterated as the lesion progresses.

The cut surfaces are typically rubbery and have a uniform black colour. Irregular areas of necrosis produce softening or cavitation. Some PMF lesions contain the conglomerated outlines of nodules. Some of nodules may have hyalinised whorled centres indicative of silicosis.

PMF is a destructive process and the remains are obliterated vessels and airways are apparent in the centres of the lesions.

Chemical extraction of the dust and other substances in massive lesions reveals: 20-50% coal, up to 30% collagen; lesser amounts of calcium phosphates, silica, silicates fibrinogen and immunoglobulins (Wagner et al. 1975).

The factors associated with development of progressive massive fibrosis are not fully understood, although several factors have been identified.

Primarily cumulative dust exposure has been the most significant factor implicated in addition to the prior identification of simple coal workers' pneumoconiosis.

The rate of PMF is closely related to the prior category of simple coal workers' pneumoconiosis. In a large study of over 100,000 working coal miners, the attack rate of PMF over a five year period with simple CWP categories 0, 1, 2 and 3 were 0%, 1%, 11% and 21% respectively (Hurley et al. 1987).

Other factors include immunological factors and infectious agents.

That quartz has been implicated will be described in the next section.

8. Silica, Coal Workers' Pneumoconiosis and Silicosis

The problem with epidemiological studies of coal workers is the inability of the chest x-rays to distinguish between silicosis and coal workers' pneumoconiosis. The typical eggshell calcification which is indicative of silica exposure is non-diagnostic and silicotic nodules are frequently observed in hilar lymph nodes in the absence of parenchymal disease (lung fibrosis). Consequently, the prevalence of silicosis in coal workers can only be reliably determined at autopsy (Green, 1998).

Silicosis is also infrequently encountered as an isolated form of pneumoconiosis in coal workers and usually it occurs against the background of simple nodular and macular coal workers' pneumoconiosis. Microscopically, silicotic nodules appear more uniform in size and have smoother borders and paler centres than the nodules of coal workers' pneumoconiosis. They have a similar distribution to the coal dust nodules. They are more common in the upper lung zones and found in the interstitial and peribronchial and subpleural locations. Involvement of the hilar lymph nodes is invariably present and these may be calcified. Coalescence of the silicotic nodules to produce complicated silicosis (PMF) can occur (Green 1998).

As discussed silicosis has been reported in underground miners especially rock bolters (Tomb 1986) and in surface coal mines in rock drillers in the USA. A survey of 1267 surface coal miners in Pennsylvania (1996-1997) revealed 6.7% showed radiological evidence of silicosis with the odds increasing with years of drilling (Tyson et al. 2000).

9. Influence of Silica on Coal Workers' Pneumoconiosis

Most cohort studies of coal miners *have not found that silica levels in dust have a significant effect on the development of coal workers' pneumoconiosis* and the most significant factor is cumulative coal dust exposure (Hurley et al. 1982; Jacobsen et al. 1970; Walton et al. 1975) in addition to other factors such as coal rank (Bennett et al. 1979; Reisner & Robock 1975) and other factors such as the age of miner, colliery and body build (Hurley et al. 1987). These cohort studies include very complete exposure data for coal miners in the United Kingdom involving medical examinations and individual exposure estimates for 50,000 miners for up to 30 years from the Pneumoconiosis Field Research (PFR) group.

Initial research from the PFR group demonstrated a strong correlation between the progression of simple CWP and the mean coalface respirable dust concentration on study of 4122 coalface workers from 20 collieries over a 10-year period. A poor correlation with quartz content of the dust was found with a strong dose-response relationship being identified for CWP and respirable coal dust.

Similar findings were noted in German coal mines (Reisner 1970). This led to the risk estimates and subsequent gravimetric coal dust standards (Jacobsen, Rae, Walton, & Rogan 1970).

The effect of quartz was further investigated by assessing the effect of cumulative dust and quartz exposures on 3154 face workers and CWP progression (Walton et al. 1975). They found the effect of quartz was paradoxical with, in some groups, the increasing percentage of quartz reduced the progression rate in some dust ranges and increased it in others. There was evidence that the high quartz concentrations were associated with higher clay mineral contents in lower rank coals and in the absence of clay the quartz may enhance the chances of developing simple CWP. Overall the effect of quartz was negative. The maximum quartz level was 10.5% and mainly less than 7.5%.

There was considerable variation between the collieries and the authors felt this was a result of a factor or factors that was not reflected in the environmental data and that cumulative coal dust exposure was the most suitable index of hazard in British coal mines where quartz does not exceed 7.5%.

A study of German coal miners also indicated the clay content was significant in preventing the progression of CWP. In a study of 9000 miners (Reisner, 1970) the issue of the clay content was assessed. They found that high dirt content delayed the occurrence of CWP changes over 7 years,

A further study on 2600 coalminers at 10 British coalmines in relation to their lifetime exposure of respirable coal mine dust (mean 33 years of dust exposure) did not show any significant effect of quartz. Estimates of exposure were based

on 20 years of observations at each colliery. Again there was no evidence that quartz (average 5% of mixed dust) had an influence on the rate of CWP. (Hurley et al. 1982)

The authors did note that some workers did have a *more rapid progression of the CWP with exposures to higher levels of quartz* and this was the subject of a case control study. Jacobsen and McLaren (1982). selected a total of 45 cases with unusual progression of CWP (greater than two steps on 12 point radiological scale) over ten years and compared to controls (with 0/0 profusion) at each colliery.

They found the average mean dust concentrations between the cases and controls were only marginally different but the exposures to quartz over the 10year period was significantly higher in cases with a mean case exposure of 6% and control exposure of 5% ($t=3.45$ $P < 0.00015$). They found that 11% of the cases had exposures at greater than 10% quartz (compared to 6% in controls). The attack rate of PMF was higher in cases (nine cases; three controls) but the authors concluded there was a suggestion that quartz dust may play a role in the aetiology of PMF, "*However that quartz is not the sole or even the most important factor as far as British coal miners are concerned*".

This research indicates that for most coal miners the primary risk factor for CWP is cumulative respirable coal dust exposure. There appears to be a small group of miners that are either reacting unfavourably to the quartz in the dust or the quartz component is a more significant factor in a proportion of coal dust exposures. This is then leading to a more rapid progression of the CWP or possibly silicosis.

10. Influence of Silica of Progressive Massive Fibrosis

Initial epidemiological studies in British coal mines did not find an association of quartz with PMF (McLintock, Rae, & Jacobsen 1970).

In a case control study involving 247 case of PMF, the factors associated with the development of PMF were assessed (Maclaren et al. 1989). The most significant factors were found to be age and prior category of CWP. Older men and higher starting point of the category of CWP were more likely to develop PMF. In relation to environmental factors, increased residence time of the dust in the lungs was associated with increasing risk.

In relation to quartz, cumulative exposure to quartz was found to have a negative association with the incidence of PMF although this was inconsistent with other reports (Seaton et al. 1981; Jacobsen and McLaren, 1982) it does add evidence that there may be other factors impacting on the effect of the quartz apart from the proportion of quartz in the dust in the dust.

11. Silicosis and Coal Mining, Quantitative Research

In one Scottish colliery, due to geological problems high levels of quartz concentrations were encountered and a number of miners showed rapid progression of changes of pneumoconiosis (Seaton et al. 1981). In a case control study (21 cases-control pairs) there was a clear relationship between progression of pneumoconiosis and dust levels, in particular the quartz levels. The radiological appearance of the cases had in many respects the appearance of silicosis.

One of the seams was quite narrow and the roof and floor were sandstone. The mining machinery in use made frequent incursions into the sandstone, resulting in abnormally high concentrations of quartz (up to 60%) in the respirable dust. Between 1971 and 1977, in one seam, 10% of the quarterly mean silica concentrations were greater than $1\text{mg}/\text{m}^3$ with two means were greater than $10\text{mg}/\text{m}^3$.

A follow up study was conducted on 551 men from this colliery in 1990. This comprised of 50% of the original cohort. A further CXR survey was conducted and average dust levels were estimated for each of the survey period for the periods between 1954 and 1978 (Miller et al. 1998).

The mean quartz percentage over the survey period (1954-1978) was 7.5% (1.3% min. to 18.3% max.) and over the last three survey periods (1970-1978) was 8.5% (1.3 % to 19.3% max). Individual quartz exposures were calculated by calculating the products of concentrations typical of the specific occupational groups and the time worked in those occupations.

They found most of the CXR progression took place after exposure ceased (that is after the colliery closed), this is more typical of silicosis.

Risk estimates were able to be calculated for radiological progression and they estimated an exposure to a mean concentration $0.1\text{ mg}/\text{m}^3$ over 15 years would give 5% risk of progression to radiological category 2/1+ (category II silicosis).

This was analysed further (Buchanan, Miller, & Soutar 2003), who assessed the risks by separating out the cases exposed to concentrations greater than $2\text{ mg}/\text{m}^3$ and below $2\text{ mg}/\text{m}^3$. They demonstrated that exposure to high concentration of quartz (greater than $2\text{ mg}/\text{m}^3$) for even a few months increased the risk of silicosis significantly.

Their model predicted a risk of category II silicosis of 2.5% for 15 years of exposure to $0.1\text{ mg}/\text{m}^3$ rises to 10.6% with the addition of a few months exposure to quartz at $2\text{ mg}/\text{m}^3$ and 72% with a year of exposure. It was estimated that 1

g.h.m^3 (a measure of cumulative dose²) of exposure at 2 mg/m^3 was equivalent to 3 g.h.m^3 at less than 2 mg/m^3 .

An exposure of 0.1 mg/m^3 over 15 years (with exposures less than 2 mg/m^3) is 2.5%; this rises to 20% at 0.3 mg/m^3 .

Although this study adds to the risk estimates for the risk of silicosis where there are high concentrations of silica, there are some difficulties in generalising the study to all coalmines. On average the miners spent almost 8000 hours with quartz levels above 0.1 mg/m^3 and in the category 2 group 18.5% of the hours was spent in dust exposures where quartz exceeded 10%. Overall 38% of dust exposures exceeded 5% quartz (46% in category II cases).

Therefore the primary relevance of the Scottish colliery research is to situations where silica concentrations exceed 10%.

This has been discussed further by Soutar et al. (2004). The lowest reported case in the cohort was as a result of exposure to average concentrations to 0.1 mg/m^3 over 12 years. The extrapolation below this gave a risk of 1% at 0.02 mg/m^3 and this was not significantly different to zero exposure (0.8% risk).

Concern has been expressed about the issue of selection bias in this Scottish colliery study as only about 50% of the original cohort was followed up (Finkelstein 2000) but this is not regarded as being a significant issue.

It is important to recognise that the miners were being exposed to coal dust and silica dust. The study of this Scottish colliery dust provides powerful evidence that exposure should be kept below an average of 0.1 mg/m^3 and where quartz concentrations are greater than 10% of the mine dust, high levels of quartz should be avoided even for short periods.

² g.h.m^3 is a measure of cumulative dose. If a miner works 1760 hours per year and is exposed to 0.1 mg/m^3 of quartz, the cumulative dose is $1760 \times 0.1/1000$. After ten years the cumulative dose will be 1.76 g.h.m^3 . After ten year of average exposures of 0.3 mg/m^3 the dose is 5.28 g.h.m^3 . Forty years at 0.05 mg/m^3 is 3.52 g.h.m^3 .

12. Chronic Bronchitis and Silica in Coal Miners

Chronic bronchitis has been associated with exposure to crystalline silica exposure in a number of industries including hard rock miners, gold miners, construction workers brick refractory workers and foundry workers (de Klerk et al., 2002a). Coalmine dust is also associated with the development of chronic bronchitis in a number of studies. However, the chronic bronchitis from coal dust or silica exposure can be difficult to distinguish from the effect of smoking (ATS, 1997)

In coal mines cumulative dust exposure has been associated with chronic bronchitis (Marine, Gurr, & Jacobsen 1988; Rogan et al. 1973; Wang, Li, & Zhao 1998). There has been little research on the effect of the silica concentration in the coal dust and the development of bronchitis. The presence of bronchitis is not always related to the presence of pneumoconiosis and therefore there may be other factors including the size of the particles involved as the cause.

In a recent autopsy study the prevalence rate was 86%. The most apparent features of chronic bronchitis in coal miners include dust deposition and dust cell infiltration on the walls of bronchi at all levels, and dust fibrosis around the bronchial walls, with the most severe pathological change of dust deposition on the respiratory bronchiole indicating that the deposition of dust is a significant factor in the development of bronchitis (Wang, Li, & Zhao 1998). The effect of quartz was not studied but there was an association with cumulative coal dust exposure

At this stage based on current evidence no comment can be made about the effect of silica in coal dust and chronic bronchitis. There is a relationship between cumulative respirable dust exposure and chronic bronchitis (Coggon & Newman 1998).

13. Silica Exposure, Coal Dust and Emphysema and Chronic Obstructive Airways Disease

Coal dust exposure has been associated with the development of emphysema and loss of lung volume. The pathology associated with coal dust exposure is that of focal or centriacinar emphysema. In a post mortem study of 503 men (Ruckley et al. 1984) has demonstrated clearly associated relationships between coal dust exposure and centriacinar emphysema. The pathogenesis of this is unknown; it has been suggested to result from mechanical forces from the coal macule or secretion of proteolytic enzymes from the coal dust activated macrophages. It appears to follow the formation of the coal macule (Green, 1998).

In a post mortem study of NSW coal miners (Leigh et al. 1994) 264 out of 376 coal miners were examined between 1966-83. The coal and quartz content of the lung was measured and compared to a number of factors including extent of fibrosis, lung function assessed prior to death and emphysema.

Silica dust was not significantly correlated to emphysema. Silica was found to be significantly correlated to fibrosis. Emphysema was negatively correlated to FEV₁%. The authors concluded the study provided strong support for the hypothesis that emphysema is causally associated with coal content and lifetime coal exposure.

Previous studies by the authors had found that in coal workers, emphysema is less likely with high lung silica content.

In silica exposure the evidence is less consistent (NIOSH 2002). Findings from post mortem examination shows that panacinar or centriacinar were the predominant types of emphysema found in the lungs of gold miners (Hnizdo et al 1994)

As there is more consistent research associating coal dust to emphysema it is apparent that coal dust is more of a concern then silica component.

Respirable crystalline silica exposure is also associated with loss of FEV₁ and chronic obstructive airways disease (NIOSH, 2002, de Klerk, 2002a). Coal dust is also associated with loss of lung function and chronic airways disease (Leigh, Wiles, & Glick 1986; Seixas et al. 1992; Marine, Gurr, & Jacobsen 1988). There has however been little research assessing the effect of the respirable crystalline silica in coal dust. Coggon & Newman (1998) suggest that the loss of FEV₁ is related to the centriacinar emphysema and there is primarily an effect of cumulative coal dust exposure. No further comment can be made on the basis of current research.

14. Auto immune Disorders, Silica and Coal Dust Exposure

A number of epidemiological studies have shown that RCS exposure is associated with increased mortality or cases from autoimmune or immunological diseases (Scleroderma, systemic lupus, erythemotosis, and sarcoidosis) and chronic renal diseases and sub-clinical renal diseases (NIOSH, 2002).

Systemic sclerosis is reported to occur in coal miners although it was unclear from the literature whether this was related to the silica exposure as a result of coal dust exposure (Seaton 1995).

Caplan's Syndrome is reported to occur in coal miners, although it was unclear from the literature whether this was related to the silica exposure as a result of coal dust exposure. Caplan's Syndrome (Rheumatoid pneumococcosis) is a condition of rheumatoid nodules developing in the lung. It has also been reported in workers exposed to RCS (Seaton 1995).

There does not appear to be any quantitative research assessing its the causation of autoimmune disorders in coal miners with respect RCS exposure.

15. Tuberculosis, Silica and Coal Dust Exposure

RCS exposure is associated with an increased risk of tuberculosis (ATS, 1997, NIOSH 2002). Subjects with silicosis have a three-fold risk of developing pulmonary tuberculosis (Hnizdo & Murray 1998).

Tuberculosis has long been associated with coal mining although initially considered to be due to the miner's poor working and living conditions. It is unclear whether the increases incidence relates to silica exposure or cumulative dust exposure and the presence of CWP and PMF.

No quantitative research was available that assessed the risk of silica and tuberculosis in coal mining. Tuberculosis is a condition that is becoming increasingly infrequent except in some areas where social and working conditions may be poorer.

Is the current NSW exposure standard of 0.15 mg/m³ respirable silica sufficient to prevent the onset of any disease outcome identified in (a)?

16. Introduction

In the Introduction to the Exposure Standards for Atmospheric Contaminants in the Occupational Environment, the National Occupational Health and Safety Commission (NOHSC, 1995) state the following:

"The exposure standards listed in [Exposure Standards for Atmospheric Contaminants in the Occupational Environment] represent airborne concentrations of individual chemical substances which, according to current knowledge, should neither impair the health of, nor cause undue discomfort to, nearly all workers.

"The exposure standards do not represent 'no-effect' levels which guarantee protection to every worker. Given the nature of biological variation and the range of individual susceptibility, it is inevitable that a very small proportion of workers who are exposed to concentrations around or below the exposure standard may suffer mild and transitory discomfort. An even smaller number may exhibit symptoms of illness.

"It follows from the foregoing that the exposure standards are not fine dividing lines between satisfactory and unsatisfactory working conditions, but rather that they are best used to assess the quality of the working environment and indicate where appropriate control measures are required."

The concept of exposure standards is one that causes considerable confusion in the minds of many employees and workers. Many people regard them as being indicators of safe/unsafe atmospheric concentrations, to the extent that workers will express extreme concern about their health and well being when they are informed that they have been exposed to an airborne contaminant at levels approaching the exposure standard. As implied above in the statements from NOHSC, this simply indicates that a process is poorly controlled and that some form of intervention is required.

This statement of the philosophy underlying the exposure standards can be traced back to the early experience of the ACGIH. This organisation found that by 1958, following confusion about the role of the Threshold Limit Values (TLVs), it was necessary to qualify the TLVs with a statement to the effect that,

TLVs, "... represent conditions under which it is believed that nearly all workers may be repeatedly exposed, day after day, without adverse effect"

The concept of protecting nearly all workers is somewhat nebulous. It recognises that in any working population, there will be a continuum of response to a given airborne contaminant. At the low end of the dose-response curve will lie those more sensitive individuals, who will react to relatively low concentrations, while at the high end of the curve will lie those more robust individuals, who are able to tolerate significantly higher concentrations.

It is difficult, if not impossible, to determine what is meant by the 'small number of workers who may exhibit signs of illness'. In commenting on the use of 'nearly all', by the ACGIH, Piney (2001) states,

Protection is not promised for all workers, as in the first Preface in 1953 but to “nearly” all workers. The one word, “nearly”, could be seen as an important shift in the TLV definition. This all depends upon how one defines the word “nearly”. It is suitably vague and, together with a tabular TLV list, probably did little at the time to change the message conveyed to most TLV users. After all, ‘nearly’ can mean ‘most’ and ‘most’ can soon come to mean ‘all’ apart from those that are, in some way, very “sensitive”. For the Committee itself, the phrase probably did not have any exact meaning at first except to say that ‘some people’ would be affected in some way when exposed at and below the TLV to some substances. As long as ‘nearly all’ was not defined, the Committee could rely upon most users interpreting the phrase as meaning small to vanishingly small and many did and still do so.

This is an important principle to grasp; as it clearly indicates that even rigid adherence to exposure standards is no guarantee that *some* worker's will not suffer adverse health consequences. In the context of this question therefore, i.e., is the current NSW exposure standard of 0.15 mg/m³ respirable silica sufficient *to prevent the onset of any disease outcome* identified in from exposure to crystalline silica in coal dust?, it appears that it has to be accepted that disease outcomes will still occur to *some* miners, even though their exposure may be well within the exposure standard of 0.15 mg/m³.

17. Comparable Standards

NSW

The NSW standard of 0.15 mg/m³ appears to be significantly higher than most other standards currently in force (see detailed below). On this basis alone, it is appropriate to question as to whether this is sufficient to prevent the disease outcomes identified above, or other disease outcomes.

It appears that by comparison with the ACGIH TLVs, there is no formal i.e. documented scientific basis for this standard. The standard was derived as being 5% of the coal dust standard of 3.0 mg/m³. This in turn was derived as a conversion from a former standard which was based upon a particle count (Hanson 2004). This being the case, the standard can almost be described as being arbitrary, as it could conceivably have been set at any level.

There is evidence, cited by NOHSC in the background information for the occupational exposure standard of 3 mg/m³ for coal dust containing < 5% quartz, the likelihood of quartz having an adverse effect on coal miners is low.

Queensland

Under the Queensland Mining and Quarrying Safety and Health Regulations, 2001 – Schedule 4, the general exposure limit for crystalline silica is defined as ‘the exposure standard stated for the contaminant in the ‘Adopted National Exposure Standards for Atmospheric Contaminants in the Occupational Environment’ and published in the Commonwealth of Australia Gazette No. C6 on 4 June 1996.’

Western Australia

Under the Mines Safety and Inspection Regulations (1996), Regulation 9.11, Exposure standards, the responsible person at a mine must ensure that in any workplace at a mine, the TWA concentration of respirable quartz does not exceed 0.2 milligrams per cubic metre of air.

Wan and Lee (1999) reviewed the incidence of silicosis in WA, comparing this with airborne concentrations of silica as sampled by the WA Department of Minerals and Energy. In WA, air sampling has been conducted by the Department of Minerals and Energy (DOMEWA) and the mining companies. This air sampling data has been used to estimate the levels of respirable silica in the mines from the 1920s through to the 1990s. The average measured atmospheric dust levels in the mines contained respirable silica of about 1 mg/m³ in the 1920s. No data were available for much of the 1930s. From the late 1930s through to 1950, measured respirable silica levels averaged 0.6 mg/m³.

From then onward, the average levels of respirable silica in the mines were:

- 1960s - 0.4mg/m³;
- 1980s - 0.2mg/m³; and
- 1990s - 0.1mg/m³.

Three of the cases had commenced exposure to silica in mining in the 5 year period 1965 to 1969 when exposure level for respirable crystalline silica was 0.4 mg/m³; and 2 cases had commenced in the five year period 1970 to 1974 when the exposure level was not monitored. There have been no cases which commenced mining work after 1974 when the exposure level has been 0.2 mg/m³. There have been no cases of silicosis in Western Australia since implementation of the current exposure standard of 0.2 mg/m³ for respirable crystalline silica. Wan states that of the 110 cases of silicosis seen in WA only 3 cases had commenced after 1968 and none after 1974. The absence of cases for the past 19 years corresponds to the implementation of the 0.2mg/m³ respirable crystalline silica exposure standard in Western Australia.

Wan and Lee (1999) assert that a more stringent exposure standard will have no impact on the decline of cases. The incidence of silicosis in Western Australia shows a progressively declining trend. When the new cases still arising as a legacy of the past have all been accounted for, new incidences of this disease will have been virtually eradicated. The gradient of this decline will not become steeper by lowering of the Exposure Standard because of compulsory medical examination for miners required by law in Western Australia and the absence of cases in the last 19 years.

This view is supported by Hewson (1996), who concludes that on the basis of prevention of new cases of silicosis, there is little support for a lowering of the (then) current Australian occupational exposure standard from 0.2 to 0.1 mg/m³.

However, Wan's findings are open to debate:

- When cases presented to the Ventilation Board, which are those which are counted, the individuals are suffering gross symptoms. The ACGIH refer to Hnizdo's findings that signs of silicosis are present with much lower exposure levels, which may not be determined by radiological examination;
- The accuracy of air samples collected during the earlier part of the twentieth century is questionable, as this was collected by counting devices such as konimeters, rather than gravimetric measurement;
- Samples may be grab samples or static samples, not necessarily personal;
- Later sampling was collected by the employers, and reported to the Department of Mines, the quality of which is open to question. In the US, there have been cases where companies' results have found to be significantly different to results collected by regulatory authorities.

de Klerk et. al. (2002b) reviewed this issue of silicosis and found that there were several potential inadequacies in Wan's assessment:

- Insufficient information concerning exposure;
- A lack of denominator data;
- No information on effects of rates of disease latency;
- Only compensated cases were considered; and,
- There was incomplete coverage of the exposed population.

de Klerk et.al. (2002b) recognised that the rates of silicosis have been dramatically reduced since the implementation of the 0.2 mg/m³ standard, however there may be an estimated rate of 4.8 cases/100,000 person-years expected using this data (UL CI for silicosis risk).

The authors of this report agree with this assessment by de Klerk, as the research primarily uses compensated cases of silicosis without systematic identification of cases and recognises that individuals may be developing significant fibrosis even where there is systematic health surveillance.

The authors also consider that the use of compensated cases of silicosis as a measure of the effectiveness of the 0.2 mg/m³ standard, suggests that the standard may be being interpreted as having an effect/no effect limit. However, the NOHSC Guidance Note (NOHSC 1995) clearly states that the exposure standards are not fine dividing lines between satisfactory and unsatisfactory working conditions, but rather that they are best used to assess the quality of the working environment and indicate where appropriate control measures are required. The authors consider that the use of data from cases of compensated silicosis is not a useful measure of successful control.

Australia

The standard for respirable crystalline silica as quartz in Australia, as determined by NOHSC, is 0.2 mg/m³ and is an interim standard. Following de Klerk et al (2002a) review a new standard for quartz, cristobalite, and tridymite 0.1mg/m³ has been proposed

USA

ACGIH

The ACGIH has set a TLV of 0.05 mg/m³ for crystalline silica as quartz, as respirable silica. The ACGIH has also assigned quartz an A2 classification, as a suspected human carcinogen.

This value is intended to minimise the potential for development of the progressive fibrotic lung disease, silicosis. ACGIH also believes there is

compelling evidence that many forms of pulmonary fibrosis constitute major risks for human lung cancer. The implications from this assessment are that control of worker exposure to avoid silicosis will also prevent silica associated lung cancer (ACGIH 2002).

The ACGIH reduced the standard to 0.05 m/m³ in 2000, from a previous TLV of 0.1 mg/m³. This was in recognition of the fact that fibrosis undetected by chest x-ray probably does occur in workers exposed at levels near the 0.1 mg/m³ level. This was based upon the work of Hnizdo (1993), who showed that a large percentage of subjects with moderate to marked silicosis at autopsy were not detected radiologically. It is the concern for fibrosis or silicosis and the role of fibrosis as a risk factor for lung cancer that persuaded the ACGIH to recommend the lowering of the TLV from 0.1 to 0.05 mg/m³.

The ACGIH documentation does not specifically refer to any potential mitigating effects of coal upon the cytotoxicity of silica. However, it does state that Donaldson and Borm (1998) reviewed the experimental literature on the toxicity of quartz and concluded that there were widely varying responses to quartz exposure depending on the presence of dust and minerals. They also noted the work of Fubini (1995) in describing the formation of Si· and SiO· radicals formed at freshly cleaved surfaces and their role in enhancing the toxicity of the silica.

NIOSH

NIOSH (2002) has recommended that until improved sampling and analytical methods are developed for respirable crystalline silica, they (NIOSH) will continue to recommend an exposure limit of 0.05 mg/m³ as a time-weighted average (TWA) for up to a 10-hr workday during a 40-hr workweek, to reduce the risk of developing silicosis, lung cancer, and other adverse health effects. NIOSH also recommends substituting less hazardous materials for crystalline silica when feasible, using appropriate respiratory protection when source controls cannot keep exposures below the REL, and making medical examinations available to exposed workers.

MSHA

| | | |
|------------------------------|---|--|
| MSHA [30 CFR 56, 57, 70, 71] | Respirable quartz in underground and surface metal and non-metal mines | PEL = 10 ÷ % quartz + 2 (8-hr TWA) |
| | Respirable crystalline silica present in concentrations >5% in surface and underground coal mines | RDS=2mg/m ³ or RDS = 10 ÷ % quartz (8-hr TWA)* or if quartz is less than 5% |

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* The intent of this formula is to maintain miner exposures to quartz below 0.1 mg/m³. The exposure standard for MSHA is based on a collection method to provide results comparable with the MRE gravimetric samples, that is, results that are comparable with UK data. Although using a different cyclone to the MRE sampler, a correction factor of 1.38 is applied (Department of Labour, Federal Register).

OSHA

| | | |
|-----------------------------------|---|--|
| OSHA [29 CFR 1910.1000–Table Z-3] | Respirable crystalline silica, quartz | PEL = 10 ÷ % quartz + 2 (8-hr TWA)* |
| | Respirable crystalline silica, cristobalite | PEL = half of the value calculated from the formula for quartz |
| | Respirable crystalline silica, tridymite | PEL = half of the value calculated from the formula for quartz |

* Based upon the ACGIH respirable sampling curve.

UK

Under the UK, COSHH Regulations there is a Maximum Exposure Limit (MEL) for respirable crystalline silica (RCS) of 0.3 mg/m³ (8-hour time weighted average [TWA]). Current evidence indicates that if workers are exposed regularly to 0.3 mg/m³ there is a much higher risk of lung damage than had been previously thought. The HSE advised in May 2003 in a chemical hazard advisory notice (CHAN) that employers should aim to control exposures to 0.1 mg/m³ (8-hour TWA) or below.

HSE is not proposing a formal change to the MEL because a new system of Occupational Exposure Limits (OELs) is planned to be in place by the end of 2004, which is likely to overtake any new MEL developments. HSE's intention is to consult on a more stringent OEL for RCS when the new OEL system comes into force.

The main health concern underlying the proposed MEL, originally published in 1992, was that of silicosis. It is noted that all forms of RCS of industrial relevance are capable of causing silicosis, but the potential to cause silicosis may be influenced by the type of industrial process and by the presence of surrounding minerals associated with RCS.

The CHAN was published in May 2003, and was obviously not available to de Klerk et al (de Klerk 2002a) for consideration when drafting their report (the UWA report). However, as the de Klerk (2002a) report recommended that the occupational exposure standard for respirable silica as quartz be reduced from 0.2 mg/m³ to 0.13 mg/m³, on the basis of the carcinogenicity of respirable silica, then this HSE Guidance Note is particularly relevant to any comments on the UWA report.

A consultative document for *Proposals for the control of inhalable dust in coalmines* was released by the HSC in early 2004. The proposed standard was for a reduction in the respirable coal dust standard to 3.0 mg/m³; and the implementation of a silica standard in coal mining to 0.3 mg/m³.

It has not been made clear by the HSE to the authors that the proposed crystalline respirable silica exposure limit in coal mining will be reduced once the new OEL for silica is implemented. Both the proposed coal mining regulations and proposed silica standards are currently going through the consultative process. It is expected that a new OEL for respirable silica will be introduced in March 2005.

In response to a question from the authors, as to decisions on the regulatory stance taken in relation to particular industries or processes involving exposure to RCS, Meldrum of the UK HSE commented, *"We could not have industry-specific OELs because we do not have industry specific dose-response data on which to set the OEL, nor do we believe that it is the industry that counts. It is the task. Drilling, cutting, grinding, fettling, milling, etc. are all high energy tasks that can generate freshly fractured quartz grains - and such tasks (or equivalent) may occur in all industries. So we are moving to a new OEL for respirable crystalline silica that will have to be complied with across all industry sectors"*. (Meldrum 2004)

18. NSW Coal Mine Quartz Data

The authors were provided with the quartz data for the NSW coal mines from 1984 to 2004. These results are not representative of the actual levels of quartz in the coal mine dust in NSW as only samples that were high or felt to contain quartz were sent for analysis. The authors had an opportunity to review 5100 results probably representing 10% of the total number of dust samples collected by the CSPL. The samples are collected 'crib room to crib room' which differs from the practice in many other countries where the collection is 'portal to portal'.

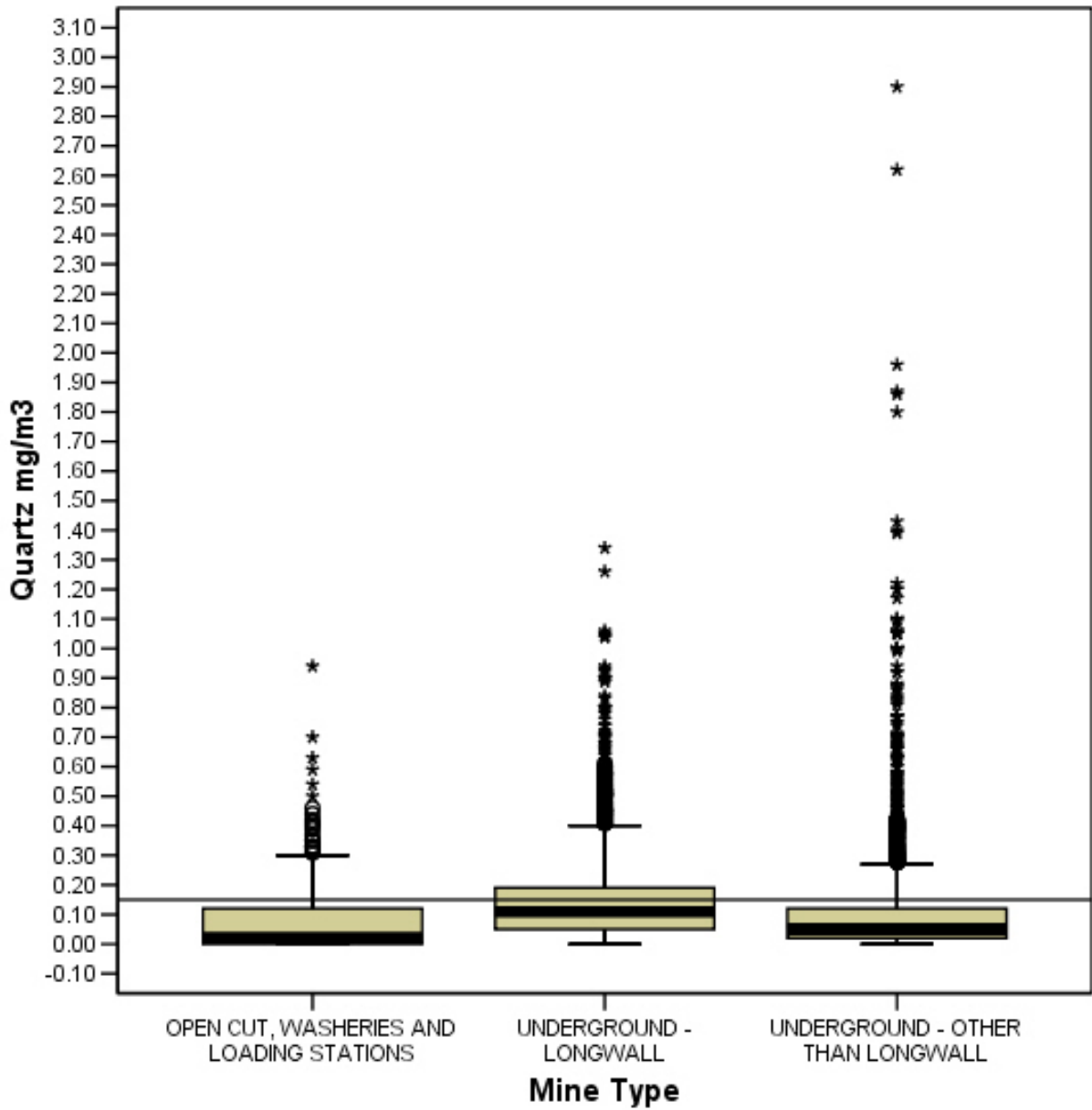
Of the samples analysed the long wall mines have the highest quartz levels. The mean quartz concentration was 0.149mg/m^3 (5% trimmed mean 0.13mg/m^3 , median, 0.11mg/m^3) with a SD of 0.154. The lowest was seen in the open cut mines with a mean of 0.07mg/m^3 (5% trimmed mean 0.06mg/m^3 , median 0.02g/m^3) and SD 0.12. The quartz results have been summarised in Figure 1.

Although these samples are non-random and are analysed when quartz exposure is suspected, assuming they represent 10% of all samples, the results could suggest close to 5% of long wall samples will be near or exceed the exposure standard of 0.15mg/m^3 .

The second analysis in figure 2 of specific occupational tasks also demonstrates that a number of work processes (including roof bolting, stemming and loading shot holes, drilling and roof scraping) are frequently associated with elevated quartz levels.

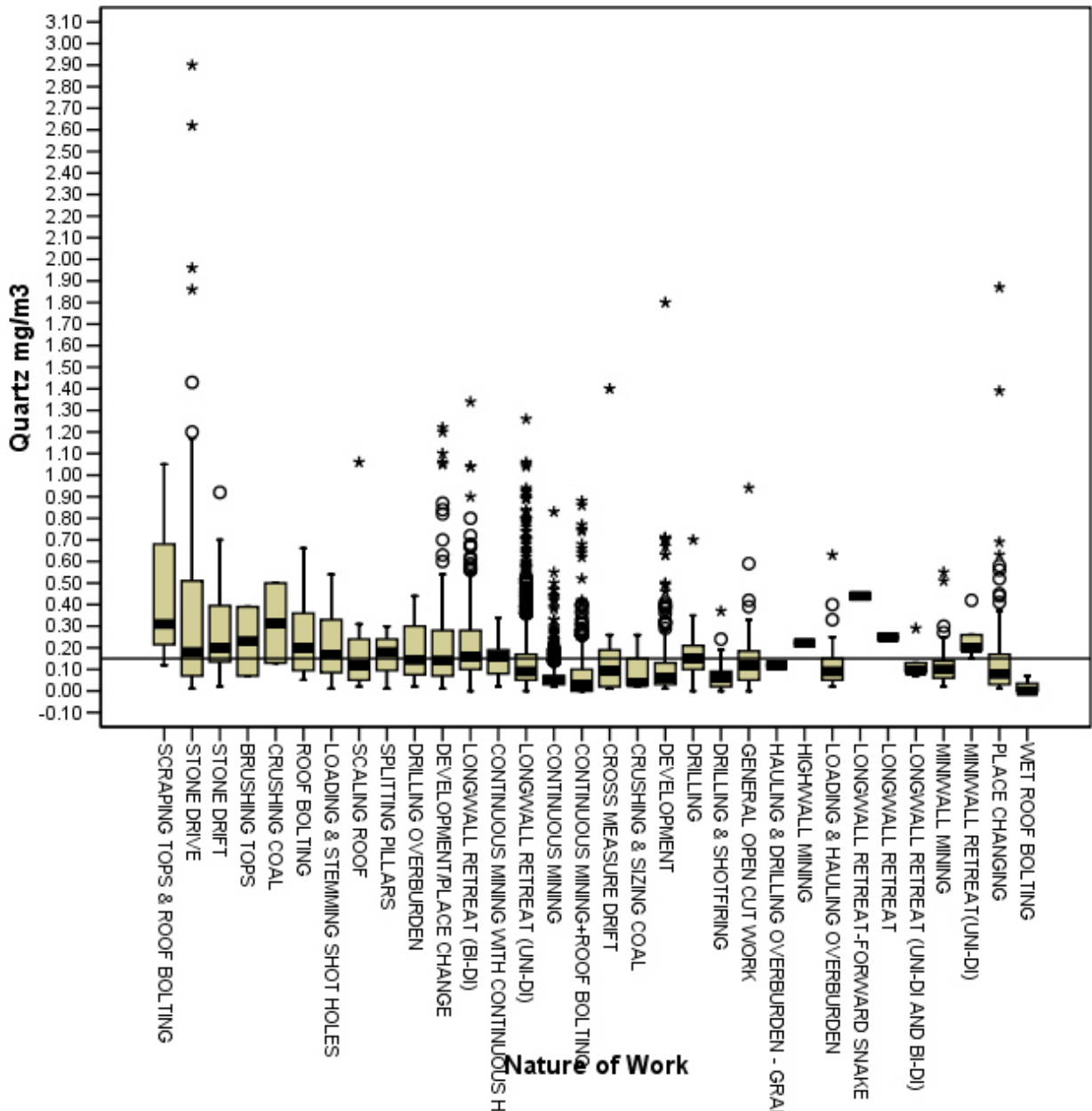
The authors recognise that these tasks that are associated with high quartz exposures can be described as high energy tasks that can generate freshly fractured quartz grains. These tasks may be associated with a greater risk of silicosis than other coal mining tasks.

Figure 1. Stem and Box Plot of Quartz Levels in NSW by Mine Type



Box plot indicating median, inter-quartile range and 95% interval.

Figure 2. Quartz Exposures In NSW Coal Mines by Task



Box plot indicating median, inter-quartile range and 95% interval.

The quartz data were analysed with SPSS 12 for Windows following importation of data from an Excel spreadsheet. As only samples that were thought to contain silica had been sent for analysis, and denominator data for each workgroup were not available, it was felt that the data would only give an insight into possible exposures rather than actual occupational exposures. Outliers were not excluded.

Coal Services Pty Limited

A Review of Adverse Health Effects from Exposure to Crystalline Silica in Coal Dust

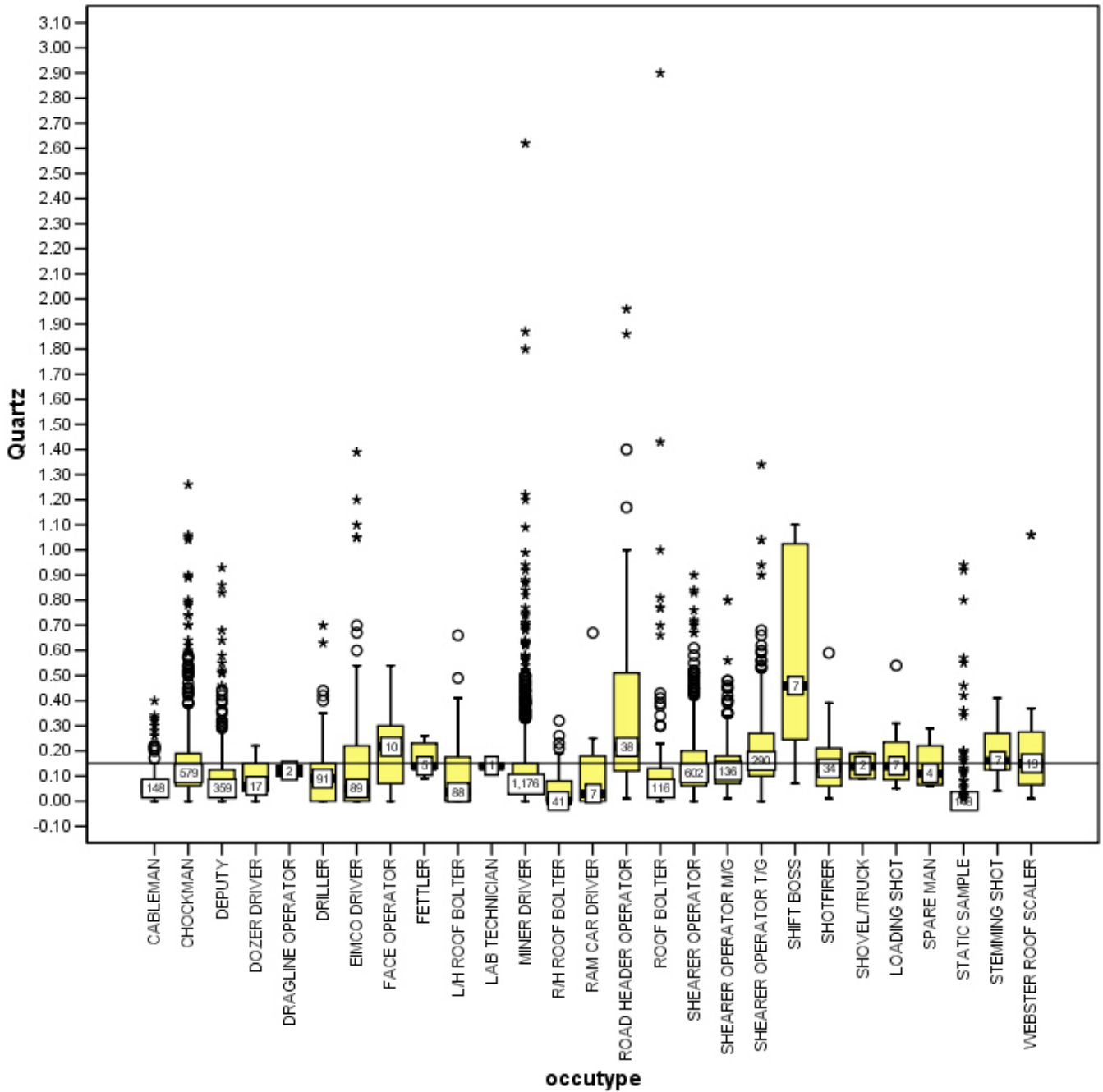
Specific tasks noted to have high levels were analysed. It was found that certain work processes had high mean levels of quartz exposure with scraping tops and roof bolting having a mean of 0.49 mg/m³ and the task of roof bolting having a mean exposure level of 0.26 mg/m³. It is important to recognise that these data are not reflective of actual average exposures to specific work groups or occupations. This was not able to be assessed from the data. It does indicate however that potentially for some tasks, high levels of probably freshly fractured quartz will be experienced and this may have a significant impact on average exposures in certain occupations.

As there was no denominator data, available it is difficult to comment on the exposures of each occupation. There were however reviewed and are detailed in Figure 3. The highest exposure groups include miner driver, shearer operator, chockman, roof bolter, driller, and face operator. The descriptive statistics for these groups are detailed in table 3. It is noted the shift boss has very high exposures but on the background of a small number of samples.

Table 3 Average and Median Exposure for high exposure groups.

| Occupation | Average Exposure mg/m³ | No of Samples | 5% Trimmed Mean | Median | Std Dev |
|-----------------------------|--|----------------------|------------------------|---------------|----------------|
| Miner Driver | 0.126 | 1176 | 0.10 | 0.07 | 0.17 |
| Shearer Operator | 0.15 | 602 | 0.139 | 0.11 | 0.13 |
| Shearer Operator T/G | 0.21 | 290 | 0.19 | 0.11 | 0.18 |
| Roof Bolter | 0.15 | 116 | 0.09 | 0.05 | 0.33 |
| Chockman | 0.157 | 579 | 0.13 | 0.11 | 0.16 |
| Driller | 0.11 | 91 | 0.09 | 0.095 | 0.13 |
| Face Operator | 0.22 | 10 | 0.22 | 0.21 | 0.17 |

Figure 3 Exposure for high exposure occupations



Box plot indicating median, inter-quartile range and 95% interval.

19. Incidence of Silicosis and CWP in NSW

A program of health surveillance is in place in the NSW Coal industry that includes a chest x-ray examination and lung function testing throughout a coal miners working life. Chest x-rays are conducted every five years. There has not been a reported case of CWP or silicosis in 18 years (Mace 2004). The incidence is illustrated in Figure 3 of cases reported to insurers.

The authors note that retirement in NSW Coalmines is at age 60 (Cram 2004). There is no follow up of miners beyond retirement apart from mortality cohort studies. It is noted however that any discovered outcome would be reported given the long known association with coal mining and lung disease.

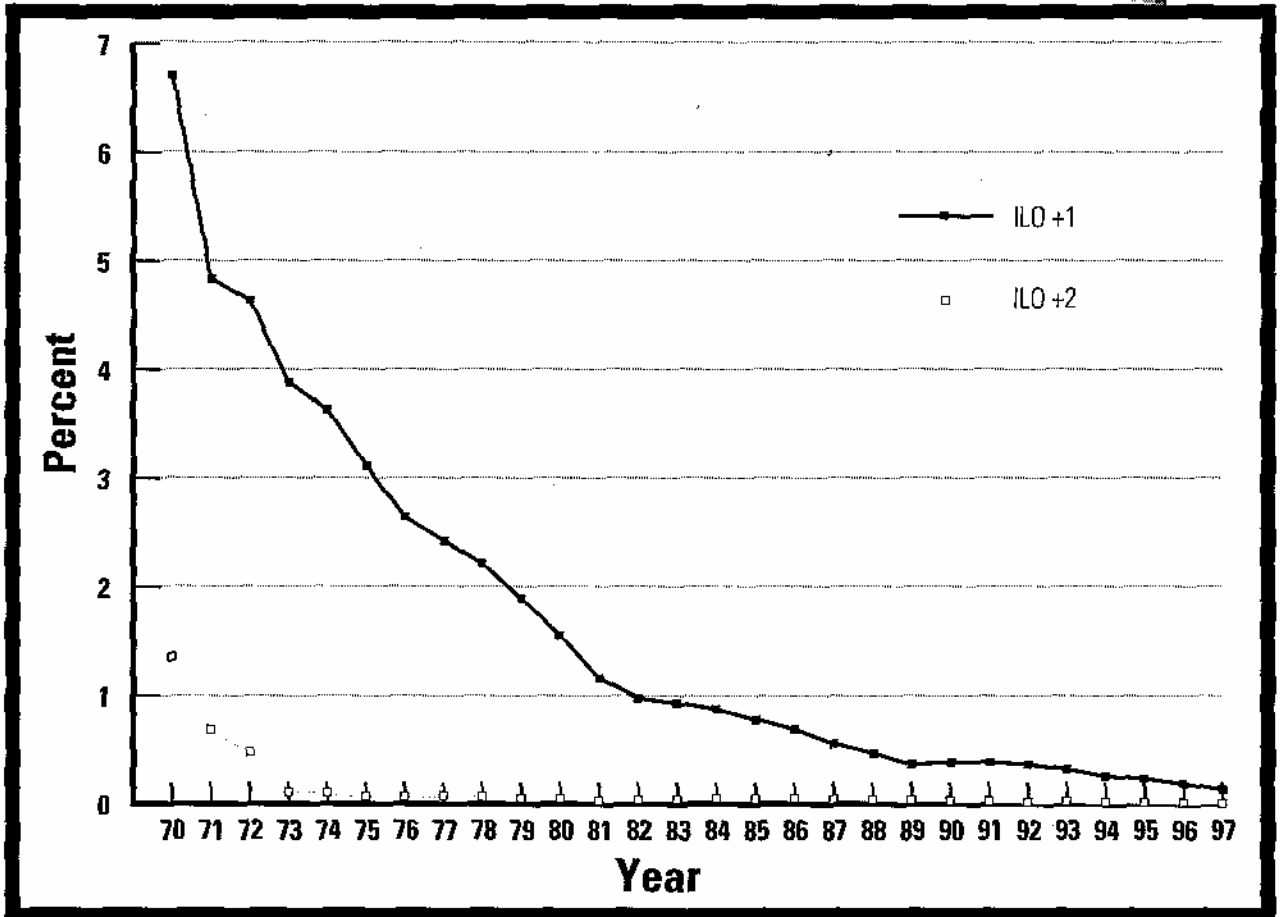
Although reassuring, there may be some deficit in the data through lack of follow up and in light of comments by Meldrum & Howden (2002), there may be significant latency in the development of silicosis and with cessation of exposure to coalmining dust the possibility of delayed onset of silicosis developing actually increases.

There are a smaller number of coal miners who are exposed to high levels of RCS and the expected number of cases may be small and overlooked by the current surveillance process.

There continues to be cases reported of CWP and silicosis in the UK and USA where there are large numbers of coalminers (>90,000 in USA). In NSW, approximately 4650 coalminers work in underground mines (Mace 2004). Therefore any expected number of cases of both in NSW would be low. The authors cannot say with confidence that silicosis or CWP is not occurring or will be detected under the current surveillance system. Recent experience in the UK (HSC, 2004) has indicated that although PMF was thought to be eradicated by the mid 1990's, recently there a number of cases of CWP have been reported including two cases of PMF in 2000.

The authors did assess the quartz data in relation to usage of respiratory protective equipment. We did find that where there were high levels of quartz RPE was almost always used. However, there were a number of cases of high levels of quartz where no RPE was used. Additionally RPE is the lowest level of protection on the hierarchy of controls and compliance can become an issue with increasing complacency in an industry.

Figure 3 Rates of ILO category 1 and 2 CWP & silicosis in the NSW Coal Industry 1970-1997



20. Conclusions

The primary identified health risk from exposure to respirable crystalline silica in coal mining will be silicosis (or progression to CWP). The authors were unable to find any convincing or consistent epidemiological evidence that RCS in coal mining is associated with lung cancer or any other malignancy.

There may be situations in the coal mining industry where workers are exposed to high levels of freshly fractured quartz and may develop silicosis. These workers may be at increased risk of lung cancer. There is quantitative research assessing this risk, including a number of large epidemiological studies that would indicate that the risk is low.

In relation to silicosis, there is good evidence from the health surveillance data that with current work practices in NSW, including the exposure standard of 0.15 mg/m³ that the risk is very low, i.e. consistently less than 0.5% during the period 1990–2000. The authors however, are concerned that the current health surveillance data may be deficient in that screening does not follow beyond retirement age of 60 and, with the effect of coal dust, the latency of silicosis may be greater. There remains the issue that after leaving the industry, the aluminium coated particles of silica may be cleaned by lung macrophages and subsequent delayed onset fibrosis may occur. Autopsy studies indicated that silicosis can occur in up to 2-12 % of coal workers examined. There may be selection bias with this type of research given that consent for autopsy may result in compensation for families, however, this research does indicate that fibrosis and silicosis can result from coal dust exposure. There are no recent autopsy studies available for NSW.

Silicosis remains an issue of concern in the US coal mines, where similar standards are in place although their exposure monitoring is conducted 'portal to portal' as opposed to 'crib room to crib room' in NSW. Some research indicates that 'crib room to crib room' samples are higher than 'portal to portal' samples by a factor of 1.35 (Tomb, cited by de Klerk, 2002a). The authors are unable to comment on an actual conversion factor as this is going to vary widely with travelling times in the mine from the portal to the crib. In addition, this will only apply to underground workers.

The authors recognise that the research in the UK coal mines provides reasonably powerful evidence that in situations where silica in the coal mine dust does not exceed 5%, the silica exposure will not exert a dominant role in the health outcome. The authors recognise that there will be situations where the quartz component of coal dust will exceed 5% and there will be a need to regulate against increased levels of respirable silica through a workplace exposure standard. The strongest epidemiological research assessing this risk in coal miners relates to the studies of one Scottish colliery, however, the authors have some concerns in relation to this for the implementation of an exposure

standard as exposures to respirable quartz were at times extremely high. It does however raise some concerns as one of the cases in the study developed category 2 silicosis after a mean exposure of 0.1 mg/m^3 , over 12 years. Outside this epidemiological research, it will be difficult to comment on a risk level for silica exposure in coal mining.

Coal Services Pty Ltd will need to consider the research from other industries for assessment of risk. It is recognised that the risk estimates for silicosis with exposure to respirable crystalline silica are varied. There is some consistency that with exposures above 0.3 mg/m^3 , the risk rises exponentially and at or below 0.1 mg/m^3 the risk is reduced or may be within in the acceptable range for many regulatory authorities.

With respect to NSW the exposure standard of 0.15 mg/m^3 , the authors are of the opinion that this is unlikely to be sufficient to prevent the onset of any disease outcome. Specifically the risk of silicosis, which is an undesirable health outcome, may not be prevented over the lifetime of all coal workers.

This is on the basis that there is a potential for delayed onset of silicosis, that some workers have relatively high levels of quartz with some work processes, and the current health data do not include long term follow up. It is recognised from the research to date that the risk is low in NSW coal mines and any reduction to the standard need only be small.

The authors have appended some relevant risk assessments in coalmines for coal miners.

There will be a need for reduction in the standard with the implementation of the new Australian Standard (AS 2985-2004). The implementation of AS2985-2004, will result in a change in the collection curve. There have been some conversion factors recommended by Grantham in de Klerk et al, (2002a). The authors were unable to find any significant research on this issue and suggest either adopting a conservative conversion factor (as suggested by Grantham) or conducting a field study. There are differing densities between coal dust and rock dust in addition to differing environmental conditions in coal mines that may affect the sampling results.

The authors recommend that to assess the risk further there should be a systematic study of the of the quartz exposures in high risk work groups eg miner drivers, shearer operators, chockmen, roof bolters and drillers.

In addition, the authors recommend the assessment the impact of 'crib room to crib' room' sampling for risk assessment purposes and to determine whether there needs to be an adjustment for regulatory purposes.

Health surveillance on high risk work groups following retirement should be considered.

In summary the authors were unable to find any evidence that CSPL should not adopt the proposed NOSH exposure standard of $0.1\text{mg}/\text{m}^3$ for respirable crystalline silica collected under the new Australian Standard for respirable dust (AS 2985-2004), which has adopted the ISO7708 respirable dust curve. Preliminary data provided by Coal Services Pty Limited suggests that $0.1\text{ mg}/\text{m}^3$ under the 2004 standard equates to approximately $0.12\text{ mg}/\text{m}^3$, as measured under AS 2985-1987, which used the BMRC curve³.

At this stage the authors were unable to find strong evidence to support a more stringent standard for the coal industry.

³ $y = 0.9056x + 0.0038$, where x = BMRC curve and y = ISO7708 curve points.

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22. Appendix One

Risk Estimates from study in Scottish Coalminers

In one Scottish colliery, exposure to unusually high levels of silica occurred in the 1970s, due to the incursion of coal getting machinery into the sandstone seam roof and floor. Rapid radiological changes were observed in some men within several years. Data from the PFR showed a clear relation between this progression and exposure to quartz.

Miller et al. (1998) described the radiographic changes in workers from this colliery. It was estimated that they were exposed to unusually high concentrations ($> 1 \text{ mg/m}^3$) of respirable quartz during the 1970s. Of the 1416 men who had worked at one colliery during that time, at least 200 were dead and another 156 could not be contacted. Of the remaining, 876 were asked to be in a health survey, and 711 agreed. Of these 551 were surveyed and chest radiographs were taken. Classifications of the films under the ILO scheme were related by logistic regression to existing data on an individual's exposures to respirable dust and to quartz. From the median of the results of the three readers, 203 men (38%) showed progression of at least one profusion category on the ILO scale (for example from 2/1 to 2/2) from the various 1970s surveys to the follow up in 1990-1991. A total of 158 men (29%) had a profusion of at least 1/0, 103 (19%) had a profusion of at least 1/1, and 47 (8.6%) of at least 2/1 at the follow up survey. Large opacities were recorded as present by at least two readers for 14 (2.6%) of the men. Profusion of small opacities was firmly related to exposures in the 1970s; it was more strongly related with quartz than with the non-quartz fraction of the dust. Estimates of risk of silicosis by logistic regression over the range of quartz exposures are tabulated below in the Summary Table 13. The silica exposures of some men at this mine were associated with considerable progression of X-ray abnormalities after exposure ended which illustrates the problems caused by incomplete follow-up. In addition only 39% (551/1416) of the original cohort was surveyed.

Buchanan et al. (2003) determined quantitative relationships between silica exposure and silicosis in this cohort. They estimated that a miner exposed for 15 years to $100 \text{ } \mu\text{g/m}^3$ respirable quartz ($1.5 \text{ mg/m}^3\text{-year}$) would have a risk of silicosis (in this case an ILO reading $> 2/1$) of 0.0248 fifteen years after exposure ended. Workers who also had several months exposure to high concentrations ($2000 \text{ } \mu\text{g/m}^3$) would have much greater risk of silicosis. Note that the ILO cut-off criterion of 2/1 applied to this cohort is higher than in all the other studies.

The data from these studies indicate a 2.5% risk of eventually developing silicosis with long terms exposure to 0.1 mg/m^3 and above 0.1 mg/m^3 the risk rises steeply, to a risk of 20% at 0.3 mg/m^3 . These risks pertain to a situation where there was exposure to freshly fractured quartz grains, relatively

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uncontaminated by other minerals. The relative risk of developing silicosis, based upon the study in Scottish coal miners, is shown in the table below:

| 15 years exposure to RCS (8hour TWA) (mg/m ³) | Equivalent cumulative exposure (mg/m ³ .years) | Risk of developing silicosis 15 years post-exposure indicated by ILO score (Cat 2/1+) |
|---|---|---|
| 0.02 | 0.3 | 0.25% |
| 0.04 | 0.6 | 0.5% |
| 0.1 | 1.5 | 2.5% |
| 0.3 | 4.5 | 20% |

↑ Mathematical Projections
 ↓ Match Observed Cases

23. Appendix 2

Calculation of a Safe Exposure Limit from Lung Burdens

Borm and Tran (2002) hypothesised that lung burden data on quartz in coal miner's lungs after a lifetime exposure could be used to set a non-carcinogenic lung burden of quartz. Lung burden was assessed by reviewing a number of post-mortem studies, twelve in all, in which the lungs were either digested or ashed and the total or specific dust in the lung is measured. These studies showed that in coal workers, up to 30g of total dust may be found in the lungs, with an accumulation rate of 0.4-1.7g of dust retained each year. The free silica load is usually a reflection of its content in respirable dust, but is 'concentrated' in lymph nodes compared with the lung tissue.

Of the twelve studies, Borm and Tran selected one by Davis (1977), of measured total dust, coal content and quartz in UK coal miners (n=74), as this was the only study to include lungs without fibrosis. Davis showed that a mean lung burden of 1.0 g (range 0.7-1.7g) was associated with macules only and this figure was used in the calculations (below):

$$1. \text{ Lifetime burden (mg)} = \text{conc}^n \cdot (\text{mg/m}^3) \times \text{deposited fraction} \times \text{ventilation} \\ (\text{m}^3/\text{day}) \times \text{workdays (days/year)} \times \text{duration (yr)}$$

The deposited fraction for respirable particles (< 5µm) is taken as 0.12 and the lung ventilation is 13.5 m³/day for a Caucasian male undergoing the heavy exercise associated with underground coal mining. Assuming 200 working days in a year, and total time underground of 30 years, the concentration of quartz (C) is derived from:

$$2. C = (\text{lifetime burden}) / (\text{deposited fraction} \times \text{ventilation} \times \text{workdays} \times \text{duration}) \\ = 1.0 \text{ g} \times 1000 / (0.12 \times 13.5 \times 200 \times 30) \\ = 0.10 \text{ mg/m}^3.$$

Taking the above quartz loads acquired over 30 years as a no effect burden for fibrosis, substitution in equation 2 leads to a NOAEL of 0.1 mg/m³ of respirable quartz when exposed for 30 years underground.