



AIOH SUBMISSION

SENATE COMMUNITY AFFAIRS REFERENCES

COMMITTEE

WORKPLACE EXPOSURE TO TOXIC DUST

AUGUST 2005

Australian Institute of Occupational Hygienists Inc.
PO Box 1205
82 Carrick Drive
Tullamarine Vic 3043

PREPARED BY:
AIOH Senate Inquiry
Working Party

CONTACT:
Ms Anne Hewer,
AIOH Administrator:
Ph: (03) 9335 2577
admin@aioh.org.au

Table of Contents

Summary	6
AUTHORISATION	9
Australian Institute of Occupational Hygienist's (AIOH).....	9
AIOH Senate Inquiry Working Party.....	11
Twenty-fifth AIOH Council.....	12
1. The health impacts of workplace exposure to toxic dust including exposure to silica in sandblasting and other occupations	13
2. The adequacy and timeliness of regulation governing workplace exposure, safety precautions and the effectiveness of techniques used to assess airborne dust concentrations and toxicity	39
3. The extent to which employers and employees are informed of the risk of workplace dust inhalation.....	43
4. The availability of accurate diagnoses and medical services for those affected and the financial and social burdens of such conditions	46
5. The availability of accurate records on the nature and extent of illness, disability and death, diagnosis, morbidity and treatment	49
6. Access to compensation, limitations in seeking legal redress and alternative models of financial support for affected individuals and their families	49
7. The potential of emerging technologies, including nanoparticles, to result in workplace related harm	50
REFERENCES.....	58

Summary

The Australian Institute of Occupational Hygienists (AIOH) welcomes and supports the Senate Community Affairs References Committee Inquiry into Workplace Exposure to Toxic Dust. This submission attempts to address those items which fall within the Institute's sphere of competence.

The health impacts of silica and other toxic dusts are such that many workers in Australia, over a diverse range of industries, may be exposed to levels of airborne silica that can be injurious to health. The health impacts are discussed at some length. However, the true incidence and prevalence of silica related disease is uncertain due to factors such as incorrect diagnosis, the long latency period of the disease and those cases where workers may have signs of the disease, without exhibiting the symptoms. These factors, as well as the fact that diseases such as silica or lung cancer are irreversible, mean that there is no known cure or treatment, other than palliative care. Hence, the particular importance of prevention. This means limiting the exposure to the dust in the workplace to the lowest level that is reasonably achievable (the ALARA principle). Occupational hygienists are the key professionals involved in the recognition of the hazard, the assessment of the risk and then controlling the risk; i.e. ensuring that the workplace environment is free from hazards to health. Sadly, many workplaces do not have access to the services of occupational hygienists and as a consequence, many Australian workers continue to be exposed to unacceptably and unnecessarily high levels of toxic dust in their places of work.

Occupational safety and health legislation addresses the issue of toxic dusts in the workplace, but enforcement of this is somewhat weak, due to the shortage of occupational hygienists in government employment. This situation has deteriorated significantly over the last decade. It is probably true to say that there have been no prosecutions of companies for dust related disease in exposed workers. This is partly due to the long latency period of the disease and also due to the rules of evidence. Until the legislation is amended to address this weakness, this situation will continue.

It is considered that the extent to which employers and employees are informed of the risk of workplace dust inhalation is variable. Small to medium sized enterprises often lack the resources or capability to avail themselves of information that is available and as indicated above, a shortage of specialists in government service means that adequate information may not always be available from government sources. To a large extent, manufacturers and suppliers are responsible for ensuring that adequate information is provided to the end user of their product to ensure that they can use it in a safe and healthy manner. However, this responsibility is not always met and under these circumstances, third parties have been providing information such as material safety data sheets (MSDSs) which often carry misleading information. In this respect, there is an onus on manufacturers, importers and suppliers to improve the current standard of product stewardship.

No comment was offered on the availability of accurate diagnoses etc., as this is more properly left to other occupational health professionals such as physicians. However, it should be noted that occupational hygienists can often greatly assist in the diagnostic processes, by providing information on the nature or extent of the exposure to other professionals such as physicians or epidemiologists. At present, the services of occupational hygienists are under

utilised by those professionals engaged in the diagnosis of conditions and the provision of medical services.

The AIOH considers that great caution should be exercised by those responsible for developing new technologies. As mentioned elsewhere, product stewardship schemes which cover all safety, health and environmental aspects in the life cycle of product from cradle to grave should be mandatory, as the producer usually has the best knowledge on the potential for workplace harm. The risks to the workforce, the community and the environment should all be carefully assessed before any new technology is introduced into the country. The National Industrial Chemicals Notification and Assessment Scheme (NICNAS) may be a model to be considered for controlling new and emerging technologies.

AUTHORISATION

This response has been prepared by the AIOH Senate Inquiry Working Party and authorised by the AIOH Council.

Australian Institute of Occupational Hygienist's (AIOH)

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

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

Therefore the AIOH has a keen interest in workplace exposures to toxic dusts, as its members are the professionals most likely to be asked to identify the hazards associated with them and evaluate the risk of an adverse health effect due to exposure to the substance in question.

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

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

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

Consultation with AIOH Members

AIOH activities are managed through committees drawn from hygienists nationally. This submission has been prepared through a specially convened AIOH Senate Inquiry Working

Party with comment sought from AIOH members generally and active consultation with particular members selected for their known interest and expertise in this area. The Chair of the Working Party initiated, coordinated, reviewed and assembled comment into this submission, that was passed on to Council and the President for approval. Various AIOH Working Party and other members were contributors in the development of this submission.

AIOH Senate Inquiry Working Party

Current membership of the AIOH Senate Inquiry Working Party include:

Martin Jennings (WA) (Chair)
Alan Rogers (NSW)
Ian Firth (VIC)
Ross Di Corleto (QLD)
Janet Sowden (VIC)

Other contributors:

Russell Bond (VIC)
John Winters (NSW)

Twenty-fifth AIOH Council

President: Gerard Tiernan (QLD)
Secretary: Jennifer Hines (NSW)
Treasurer: Philip Hibbs (NSW)
President Elect: John Edwards (SA)
Councillors: Deborah Glass (Vic)
Bill Hanna (NSW)
Robert Golec (VIC)

1. The health impacts of workplace exposure to toxic dust including exposure to silica in sandblasting and other occupations

The AIOH has a keen interest in workplace exposure to toxic dusts, as its members are the ones most likely to be asked to identify the hazards associated with them, assess the risk of an adverse health effect due to exposure to the toxic dust in question and then advise on a suitable method of control to reduce the risk of exposure to an acceptable level. To all intents and purposes, an acceptable level is an airborne concentration of the toxic dust which is within the occupational exposure standard for that particular dust. In the case of silica, the exposure standard has recently been reduced to 0.1 mg/m³.

HEALTH EFFECTS OF CRYSTALLINE SILICA

Occupational exposure to quartz has occurred in its production and use; as a result of hard-rock mining; in the manufacture of ceramics; and in agriculture, foundries, and road construction and maintenance. The American Conference of Governmental Industrial Hygienists (ACGIH) note that use of silica sand for sandblasting continues despite the large number of acute and subacute cases of silicosis reported for this occupation (ACGIH, 2001). In Australia, the use or handling of a substance that consists of, or contains, crystalline silicon dioxide as an abrasive material in abrasive blasting, is now prohibited under occupational safety and health regulations.

Historical Considerations

The impacts of dust in the workplace have been known for centuries. As long ago as 1556, Agricola describes the effect of dust in mines upon the health of miners:

“On the other hand, some mines are so dry that they are entirely devoid of water, and this dryness causes the workmen even greater harm, for the dust which is stirred and beaten up by digging penetrates into the windpipe and lungs, and produces difficulty in breathing, and the disease which the Greeks call *ασθμα*. If the dust has corrosive properties, it eats away the lungs, and implants consumption in the body; hence in the mines of the Carpathian mountains women are found who have married seven husbands, all of whom this terrible consumption has carried off to a premature death.”

In 1713, Bernardino Ramazzini, sometimes referred to as the Father of occupational medicine, described how stone-cutters, sculptors, quarrymen and other such workers, were affected by exposure to dust in the course of their work:

“When they hew and cut marble underground or chisel it to make statues and other objects, they often breathe in the rough, sharp, jagged splinters that glance off; hence they are usually troubled with a cough, and some of them contract asthmatic affections and become consumptive...When the bodies of such workers are dissected, the lungs have been found to be stuffed with small stones. Diemerbroeck gives an interesting account of several stone-cutters who died of asthma; when he dissected their cadavers, he found, he says, piles of sand in the lungs, so much of it that in cutting with his knife he felt as though he were cutting a body of sand. He says too that he was told by a master stone-cutter that when he was chiselling stone a dust arose, so fine that it penetrated the ox-bladders hanging in the workshop; in fact in the course of one year he found that a handful of this dust had

accumulated inside a bladder. The man declared that that dust would gradually prove fatal to stone-cutters who took no precautions.”

Ramazzini’s advice on precautions to such workers was, “they must be warned to be as careful as possible not to breathe in these minute fragments by the mouth.”

In 1908, Edgar Collis was appointed as only the second medical inspector to the UK HM Medical Inspectorate of Factories. He developed an interest in the local silica brick industry in Stourbridge, where he was the local general practitioner and in 1915, he delivered the Milroy lectures, which were the first indication of the role of silica in silicosis (Raffle et. al., 1987).

In 1946-47, the ACGIH introduced a Threshold Limit Value (TLV[®]) for quartz, MAC-TWA, 5 mppcf — high (above 50% free silica); 20 mppcf — medium (5% to 50% free silica); 50 mppcf — low (below 5% free silica) (ACGIH, 2001). The TLV is defined as being the airborne concentration of individual chemical substances at which it is believed that nearly all workers may be repeatedly exposed, day after day, over a working lifetime, without adverse health effects (ACGIH, 2005). The TLV should not be considered to be a safe/unsafe threshold, but an indication of an airborne concentration at which controls must be used to reduce the risk to the exposed workers. These controls may include measures such as ventilation, or use of respirators, for example.

Health impacts associated with exposure to respirable crystalline silica

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).

Subacute silicosis (accelerated silicosis)

In some occupations, exposure to high concentrations of 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

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. In 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.

In 2003, the UK HSE published a report outlining how the toxicity of silica varied depending upon certain circumstances. Variability could be affected by:

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

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

The particle number, size and surface area – Current knowledge suggests that regardless of the type of dust, the total surface area of the dust retained in the lungs is an important determinant of toxicity. Surface area is related to particle size; smaller particles possess a much larger surface area than larger particles. Hence, smaller particle size fractions (very fine dusts) of respirable crystalline silica would be expected to produce more lung damage than equal masses of larger respirable size fractions. On this basis, it is suggested that there would be a greater risk of silicosis in workers exposed to very fine particles of crystalline silica, as

might be found in some silica flours, compared with equal masses of larger sized respirable particles.

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

Silica Exposure and Cancer

In 1997, a monograph published by the International Agency for Research on Cancer (IARC) concluded that there is now sufficient evidence in humans for the carcinogenicity of inhaled crystalline silica in the form of quartz or cristobalite from occupational sources (IARC 1997). Several studies among the many reviewed by the IARC working group on the question of silica exposure and cancer risk in humans were negative or equivocal, and carcinogenicity of silica was not detected in all industrial operations. However, nine studies showed 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 (reactive oxidative species) and carcinogenesis has recently been reviewed (Castranova & Vallyathan, 2000).

Silica and sand-blasting

Most jurisdictions in Australia have now prohibited the use of silica or silica containing material as an abrasive in sand-blasting. For example, in W.A. this is prohibited under regulation 5.14 of the OS&H (Hazardous Substances) Regulations 1996. Other States followed later, for example, WorkSafe Victoria proceeded with action to prohibit the use of materials containing greater than 1% crystalline silica in abrasive blasting with effect from 1 January 2002. The prohibition was given effect through the power of the Hazardous Substances Regulations to declare a substance to be a prohibited substance for specified uses (Workplace Relations Ministers Council).

To what extent this prohibition has been effective in completely eliminating the use of silica/silica containing materials in sand-blasting is unknown, but a report by Grantham and Groothof (2001) on the results of a blitz by the Department of Workplace Health and Safety on abrasive blasting operations throughout Queensland is informative. This survey found that of 49 operations audited, 2 (4%) were using dry sand. Other than the 2 (4%) using sand, they also found that garnet was used as a major blasting medium by 47 % of operators audited, 27 % used ilmenite, 34% employed different types of metal refinery slags and metal shot was used mainly by 9%. One operator used sodium bicarbonate. A small number were using glass. Use of quartz bearing sands is now low, but the 2 cases observed were found to contain silica between 58 – 78% free silica. These operations were issued with Prohibition Notices.

Silicosis – The Australian Situation

Health problems associated with exposure to crystalline silica dust have been under investigation and control in Australia for more than a century.

Members of the Australian Institute of Occupational Hygienists Inc. (AIOH) have been involved in assisting these various government enquiries, the setting of the exposure standards and also the ongoing monitoring of worker exposure and the implementation of various control procedures.

Early investigations were carried out into ventilation conditions in the hard rock mining industry in Western Australia, (Royal Commission, 1905) and in the Victorian gold mines (Summons, 1906). In 1914, a Royal Commission was appointed to investigate, among other things, working temperatures, ventilation, compensation for industrial sickness or accident, and the best means of reducing accidents and ensuring the safe working of the mines at Broken Hill resulting in the first medically supervised on going surveillance of the workforce and a statutory workers compensation system. (George, 1947 and Bureau of Medical Inspection Annual Reports).

The coal industry in NSW and Queensland also had early and ongoing enquiries which led to the set up of statutory authorities to conduct exposure and disease surveillance in various states such as WA, Queensland and New South Wales (eg Joint Coal Board Annual Reports).

Surveillance by the NSW Silicosis Board (now the Dust Diseases Board) and the NSW Health Department resulted in the investigation and control of exposures for Sydney sandstone workers (Badham 1924, Francis 1968, Jones 1968).

The minutes of the Occupational Health Committee of the National Health and Medical Research Council bear testament to the various enquiries, the implementation of exposure standards and dust control measures and the subsequent reduction and virtual elimination of silicosis in Australia (minutes NHMRC, 19-1985).

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

For instance, in Western Australia, where there is a very good system of worker surveillance, such as with Kalgoorlie miners, the records show less than 5 new cases of silicosis per year across the state and that none of the cases commenced employment since 1974 when the exposure standard of 0.2 mg/m³ was introduced (Wan & Lee 1993). A review of the medical surveillance records from Broken Hill workers was presented to Worksafe Australia as proof that the implementation of the current level of 0.2 mg/ m³ respirable silica had proven to be more than adequate in preventing silicosis in the mine workforce (Submission by the Chamber of Mines, Metals & Extractive Industries NSW, 21 December 1988 and letter from Department of Mineral Resources 29 August 1991).

Further review of the statistics commissioned by Worksafe Australia in 2004 substantiated the small number of new cases arising from Australian industries (de Klerk, 2002a, de Klerk 2002b).

AIOH members have also been part of the continued State based dust disease surveillance programs particularly in Queensland and NSW. An examination of the silicosis and lung cancer risk has been carried out, based on NSW Dust Diseases Board data, by two of our members. The findings are presented in a research report and international scientific publication and essentially state that after allowing for tobacco smoking, there is nearly a doubling of lung cancer risk in compensable cases for silicosis (X-ray evidence, decreased lung function and disability) which is observed across most industries and occupations (Berry et al, 2002, and Berry et al 2004). The level of lung cancer risk is in line with that reported from other international studies.

Detailed examination of the various occupations and industries associated with the cases was carried out. (Berry, Rogers, Yeung, 2002, Rogers Yeung, Berry 2005) For instance of the 1447 silicosis cases (that were receiving compensation prior to 1970 and were still alive in 1970 and those who were awarded compensation from 1970 to 1994) there was only 17 (1.2% or less than 1 case per year) that indicated that they did sandblasting as part of their work. Most of these sand blasting cases received their exposure around 1970 or earlier.

Analysis of other industry types was also carried out by the above researchers, and the strong indications are that the historical reduction in silicosis numbers is due to a combination of regular medical surveillance, and reduction in exposures such as compliance with a regulatory exposure standard, the prohibition of specific tasks associated with high risk (such as sand blasting and the use of silica flour in foundry operations) and the use of adequate dust suppression systems such as ventilation and wetting down.

Long term exposure to high levels of crystalline silica has also been associated with increase in lung cancer (Berry et al 2002 & 2004). Although detailed examination of the various risk factors indicates that tobacco smoking contributes a higher risk component and hence the majority of the case numbers.

Airways disease (conditions such as reduced lung function, bronchitis, and asthma) has been statistically associated with some occupational groups such as some miners that have been exposed to long term high dust exposures. The findings are controversial as the associated disease symptoms are confounded due to lifestyle factors, particularly tobacco smoking. Similar to the findings with lung cancer outcomes, for airways disease detailed examination of the various risk factors indicates that tobacco smoking contributes a higher risk component and hence the majority of the case numbers.

Media headlines often imply that silica is “the new asbestos”. However examination of the data suggests otherwise. Silica has been under surveillance for many decades, and the morbidity and mortality of large populations of heavily exposed individuals have also been studied over many decades. Clinical silicosis is now a rarity, and elevated risk of lung cancer appears to be confined to cases where the silica exposure is of such a level that it results in clinical silicosis. Based on the number (say 10-30) of new cases of silicosis, this would amount to only 1 or 2 additional lung cancer cases per year across Australia. Removing the smoking component from airways disease and the reduced contemporary silica dust exposures would mean only a few additional cases of airways disease per year in Australia. Increased risk of renal disease has been implicated with elevated exposures to crystalline silica. A recent

US study found a doubling of risk of non-malignant renal disease but no increase in renal cancer (McDonald et al, 2005). This finding would imply only a small number of additional cases of non-malignant renal disease in Australia per year.

Occupational Exposure Standard – respirable crystalline silica

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) recommended a reduction of the Australian occupational exposure standard for respirable crystalline silica. The report provides a thorough review of the literature on health effects associated with occupational exposures to crystalline silica published up to February 2002. As quartz is the most widespread form of crystalline silica in the Australian context, mainly due to the magnitude of mining and construction industries, it is logical that emphasis be placed on it in the report.

The revised standard is 0.1 mg/m³ specifically for quartz, cristobalite and tridymite (8hr TWA). This is a reduction from the previous exposure standard of 0.2 mg/m³ for quartz and the previous standards for cristobalite were 0.1 mg/ m³ and tridymite 0.1 mg/m³. 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/ m³.

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 protect 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/m³ 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/m³.

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.

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.

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/m³ to 0.05 mg/m³. They state that fibrosis undetected by chest X-ray probably does occur in workers exposed at levels near 0.1mg/m³ level. This is based on studies by Hnizdo (1993) who showed that a large percentage (up to 72% exposed to 0.5mg/m³ for twenty years) will have a moderate or greater degree of silicosis at autopsy that were not detected radiologically.

HEALTH EFFECTS OF TIMBER DUST

In addition to silica, there are many other dusts which are toxic in the workplace. It would be impossible in the time available to compose this submission to consider all other toxic dusts, therefore, the example of wood dust is offered for consideration. While this might be considered an innocuous substance which every home handyman has probably encountered, it is quite toxic as the following review indicates. (material supplied by Janet Sowden).

A review of the literature

Trees are classified botanically into two types: gymnosperms (e.g. conifers – fir, cedar, cypress etc), which have exposed seeds, and angiosperms (e.g. most deciduous trees, eucalypts, and various tropical species), which have encapsulated seeds. Timber for commercial use is classified as softwood, from gymnosperms, and hardwood, from angiosperms. The two types differ both anatomically and chemically. As a general rule, hardwood is denser and contains less resin than softwood.

Non-malignant effects

Exposure to timber dust may cause simple irritation or, less frequently, immunologically mediated effects such as rhinitis, asthma, bronchitis and pneumonitis. Not all persons are allergic and not all timber species are allergenic. Asthma has been reported in workers using a variety of timbers, particularly certain softwoods (e.g. Western red cedar (notorious!), Californian redwood, spruce and some pine species) and a few hardwoods (e.g. blackwood, messmate, rosewood) .

Malignant effects

There is persuasive evidence dating from the 1960s that exposure to hardwood dust has been associated with adenocarcinoma of the nasal sinuses, especially in those industries requiring fine, accurate work such as furniture making and pattern making. Such work requires extensive sanding and shaping, and produces much fine hardwood dust. Sino-nasal cancers associated with hardwood dust, or with a mixture of hardwood and softwood dust, have been reported from many countries including Australia.

The International Agency for Research on Cancer (IARC) first reviewed the evidence for nasal cancer in the wood industries in 1981. At that time it stated that there was sufficient evidence that nasal cancers, particularly adenocarcinoma, had been caused by employment in the furniture-making industry; but that there was not sufficient evidence to assess the risk in other wood-working industries such as sawmilling.

The IARC again reviewed the evidence in 1987. At that time it was still evaluating the risk in certain woodworking industries rather than from particular wood species. It stated that there was sufficient evidence (as in 1981) for a carcinogenic risk in furniture-makers; that there was limited evidence for a carcinogenic risk connected with employment as a carpenter or joiner; and that there was inadequate evidence for a risk in sawmillers and loggers.

The evidence for softwood as a cause of nasal cancer is less convincing than that for hardwood. Softwoods are used more for construction and carpentry. Softwood dust appears not to be associated with adenocarcinoma, but it may be associated with squamous cell carcinoma of the nasal sinuses. It has been speculated that this difference in carcinogenic

effect between the two types of wood dust may be due to the smaller particle size and greater “dustiness” of hardwood (especially when seasoned), to its different chemical composition, to the differing trades in which it was used (e.g. precision woodwork as compared with sawmilling), to the fact that many of the softwood workers worked outdoors, and that they were handling fresh wood with a high moisture content. Fresh wood would produce coarser particles less readily inhaled and deposited in the nasal sinuses. The most comprehensive review of the topic in recent times was by IARC in 1995, which stated as follows: “Adenocarcinoma of the nasal sinuses is clearly associated with exposure to hardwood dust; in several series of cases of adenocarcinoma from different countries, a high proportion of cases had been exposed to hardwood, and these findings were confirmed in several case-control studies as well. There were too few studies of any type to evaluate cancer risks from softwood alone. In the few studies in which exposure was primarily to softwood, the risk for cancer of the nasal cavities and paranasal sinuses was elevated but considerably lower than that in studies of exposure to hardwood or to mixed wood types; furthermore, in the studies of exposure to softwood, exposure to hardwood could not be clearly ruled out.....” In 1995 the IARC classified both hardwood and softwood dust as carcinogenic to humans .

Latency

The average reported time between first exposure to wood dust and diagnosis is surprisingly consistent, at around 40 years. In individual cases the latency can be considerably longer or shorter than 40 years.

Health effects of MDF and other composite woods

Composite boards for building and cabinet making are made from wood fibres bonded together with a resin, usually urea-formaldehyde or phenol-formaldehyde. In Australia, the wood fibres appear to be softwood exclusively , but both softwood and hardwood are used in particle boards in other parts of the world . The fibre/resin mix is formed into layers and cured in a hot press. During this process, or if the resin is subsequently broken down by heat, or if it is improperly cured, a small amount of free formaldehyde may be released. There is thus a possibility that dust from the machining and working of these products may contain not only wood dust and cured resin, but also formaldehyde.

Formaldehyde is a pungent, intensely irritating gas. Atmospheres containing more than about 3 ppm formaldehyde are unbearable for more than a few minutes. Inhalation at levels above 3ppm causes cellular damage in the nasal tissues of experimental animals .

Exposure standards for timber dust and MDF

Wood dust: The recommended occupational exposure standards for timber dusts have changed over the years as more has become known about the hazards of particular species. Until the 1970s there was no standard specific to timber dust at all. In both Australia and the USA it was regarded as a nuisance dust. Such “dusts, unspecified” were controlled from 1955 in Victoria to a level of 50 million particles per cubic ft (mppcf).

From 1964, the Australian National Health and Medical Research Council (NHMRC) adopted the exposure standards published by the ACGIH in the USA, with a few local modifications for asbestos, silica and carbon monoxide. These standards were also used by the Victorian Health Department when assessing health hazards in industry, although they were never incorporated into legislation.

The ACGIH did not have a separate standard for wood dust until 1972 . In that year the nuisance dust standard became 10 mg/m³ and the standard for non-allergenic wood dusts became 5 mg/m³. No recommendation was made for allergenic dusts. From 1981 until 1998, the standard remained at 5 mg/m³ for softwoods; but for “certain hardwoods, e.g. beech, oak” the recommendation was 1 mg/m³ in recognition of the carcinogenic effects of these hardwoods.

Since 1998, the standard has become much more complex, not only because of the burgeoning literature on the carcinogenic and allergenic effects of a larger number of timber species, but also because of changes in dust sampling techniques and in the definition of inhalability. The timber dust exposure standards proposed in 2001 are as follows. All values are time-weighted average (TWA) values for inhalable particulate as defined by the European Standardization Committee:

- Wood dust, non-allergenic and non-carcinogenic: 2 mg/m³
- Western red cedar: 0.5 mg/ m³
- Other allergenic wood dust: 1 mg/m³
- Wood dust, confirmed or suspected to be carcinogenic: 1 mg/m³. (confirmed: beech and oak; suspected: birch, mahogany, teak and walnut)

In a draft 2003 document from the ACGIH there is a proposal to further reduce the exposure standards for both allergenic and carcinogenic species.

Formaldehyde: There has been a steady reduction in the exposure standard (TLV) for formaldehyde also. In recognition of its acute irritant effects it has had a short term exposure limit (STEL) as well as a TWA limit because of its suspected carcinogenicity. The ACGIH's TLV values for formaldehyde were under legal challenge in the USA for a number of years so their history is somewhat complicated. During the period 1977 – 1998 the TWA exposure standards varied from a value of 2ppm (1977 – 1984), 1 ppm (1985 – 1991) to 0.3 ppm (1992), and the STEL values varied from 2 ppm (1985 – 1992) to 0.3 ppm (1993 – 1997).

Formaldehyde and timber dust in combination (MDF dust): Because both timber dust and formaldehyde have similar effects, the combined effect is additive. The formula for combining the two effects is:

$$\frac{C_1}{T_1} + \frac{C_2}{T_2}$$

where C is the measured concentration and T is the TLV of the contaminant in question, and if this sum exceeds 1, the TLV of the combination has been exceeded.

Dust levels during use of MDF

The concentration of airborne dust and formaldehyde during the use of composite boards such as MDF and pineboard will depend on the material used, the working practices, the tools used, the size and ventilation of the workspace, and whether or not the tool is fitted with local exhaust ventilation (LEV). Occupational hygienists have an important role in advising on these factors and ensuring the workers are exposed to dust levels that are as low as is

reasonably achievable. However, it is a fact that as many workers are employed in industries that do not have access to occupational hygienists. Typically, in small to medium enterprises, many workers will continue to be exposed to unacceptably high levels of wood dusts, with the attendant disease risks. This is a situation that can and should be rectified.

2. The adequacy and timeliness of regulation governing workplace exposure, safety precautions and the effectiveness of techniques used to assess airborne dust concentrations and toxicity

All Australian jurisdictions have occupational safety and health legislation governing workplace exposure to most toxic dusts. However, a major difficulty with the use of the duty of care obligation under OH&S legislation arises with the rules of evidence, which require that for a prosecution to proceed, it must be proven that an alleged breach of the Act or Regulations either has resulted, or could have resulted, in a fatality or serious injury. Prosecution will only be initiated where an inspector obtains sufficient evidence to establish a prima facie case and then can show that the evidence could lead to the conclusion, beyond reasonable doubt, that all the elements of the offence have been proved.

With diseases related to exposure to toxic dust in workplaces, it is practically impossible to prosecute an employer for a breach of his duty of care, because it requires proof, "beyond all reasonable doubt" that exposure to a given dust actually caused the related disease. Furthermore, a prosecution must be taken within 2 years after the offence was committed (at least in WA), and as occupational diseases have long latency periods of many years, it is highly unlikely that an employer will ever be prosecuted under any State or Territories' Occupational Safety and Health Act, for diseases such as silicosis.

As a consequence, prosecutions for toxic dusts in workplaces will usually be taken under occupational health and safety *regulations*, although it is interesting to note that in WA, no prosecutions have ever been taken under the Hazardous Substances Regulations in relation to airborne dusts. To some extent, this is due to the fact that regulators are more focussed on prevention of traumatic fatalities from electrocutions, falls from heights, crushing, etc., than on preventing diseases with long term outcomes.

A secondary factor in the failure of regulators to pursue more prosecutions for dust related diseases is the loss of expertise within their departments. In WorkSafe WA for example, the number of occupational hygienists employed has dropped from 7 in 1995 to 3 in 2005. Furthermore it is now difficult to find courses for training occupational hygienists, so future government occupational hygienists will have difficulty receiving the training required.

However, there has been some enforcement action taken on dust in workplaces. As indicated earlier Queensland had a blitz on abrasive blasting operations in 2000. The following information was provided to the AIOH Working Party in response to a query on the number of Improvement Notices issued by WorkSafe WA for 'dust' related breaches of the WA OS&H Act or Regulations:

Numbers of Notices Issued from 01 / 01 / 2000 to 28 / 07 / 2005 in Relation to "Dust":

- In total there have been 729 Notices issued by WorkSafe WA in relation to dust during this period.
- Not all notices deal with the subject of inhalation exposure or to the health hazards relating to dust exposure. The approximate percentages of notices issued under the various subject areas are given below.
- It should be noted that all of these 729 notices have the word "dust" contained somewhere within the text.
- Dust Notice Areas & the Estimated Corresponding Percentages of The Total "Dust" Notices Issued (based on a 50% sample of the total number of notices on dust issued during this period)
 - Notices dealing with dusty respirators, approximately 7% (52 notices out of the 729 in total)
 - Notices dealing with significant dust levels on Electrics & Switchboards, approximately 2% (8 Notices)
 - Notices dealing with dust & fire, approximately 1% (4 Notices)
 - Notices dealing with the control or assessment of dusts in relation to health or inhalation exposure, approximately 89% (648 notices)
 - Notices dealing with various other areas, approximately 1% (4 notices).
- It is interesting to observe that the number of notices requiring controls to be implemented far outweighed the number of notices requiring a risk assessment to be completed in relation to dust exposure.
- Notices dealt with various specific types of dust hazards including (but not limited to): Wood Dust & MDF dust; Asbestos; Nuisance / General Dust; Construction Work Dust; Silica, Marble Dust; Brick Dust; Lead Dust; Powder; Grain & Hay Dust; Brake Dust (from vehicles); Fly Ash; Aluminium Dust; Dust created when employees blow themselves down with compressed air; Abrasive Blasting Dust, Organic / Animal Dust (Information provided by David Torr, WorkSafe WA, July 2005).

Therefore, it is considered that although there is regulation of toxic dusts in workplaces and that there are adequate standards in place, the enforcement of the regulations is weak and mainly confined to issuing improvement notices, as it is very difficult to prosecute under the existing rules of evidence required under workplace health and safety legislation. This situation will not change until legislation can be amended to allow for prosecution of dust disease created by exposure to toxic dusts in the workplace.

3. The extent to which employers and employees are informed of the risk of workplace dust inhalation

Under Occupational Safety and Health legislation in all States and Territories, there is an obligation upon employers to provide information to employees to enable them to perform their work in such a manner so as not to expose them to hazards.

The Hazardous Substances Regulations, include requirements for risk assessment at least in theory. Historically OH&S inspectorates assisted industry in understanding how to control exposures however, due to downsizing and restructuring, such assistance is now severely limited or non-existent. While all jurisdictions publish guidance material or other sources of information, anecdotal evidence indicates that this is not filtering down into many small to medium enterprises.

As well as government publications, information on the risk of inhalation of toxic dusts in the workplace can be obtained from a number of sources. Manufacturers or suppliers of products containing toxic dusts, or which may generate toxic dust during handling, are required to provide information on the hazard associated with the product. Usually, this is provided in summary form by means of standardised risk phrases on the product label, e.g. R20: Harmful by inhalation, or in more detailed form by the product material safety data sheet (MSDS). Guidance on the provision of information on labels (NOHSC, 1994a) and MSDSs (NOHSC, 1994b) is given in National Codes of Practice, published by the National Occupational Health and Safety Commission.

More responsible manufacturers view the provision of information on labels and MSDSs as a minimum standard. Some companies have implemented voluntary product stewardship schemes, in which they exercise a cradle-to-grave responsibility for their products at every stage in their life cycle. Product Stewardship has been defined as a demonstrable process that places an ongoing responsibility on a company to identify, monitor, manage and continually improve the health, safety and environmental (HSE) performance of its products and packaging (PACIA). Thus, a product stewardship scheme embraces those workers engaged in the process of raw material production, transport, storage, manufacture, use and disposal. It is now interesting to note that the Commonwealth government is exploring a co-regulatory approach as one option for governments to support industry in working with other parts of the community to deliver better environmental outcomes through product stewardship (EPHC).

It is a matter of concern to some occupational hygiene professionals that information on the risk of exposure to toxic dusts often becomes clouded with emotion, so that scientific reason can be lost amid often outlandish claims. A good example of this phenomenon is the 'single fibre' theory of asbestos toxicity, where it is held that just one fibre of asbestos is enough to kill. The danger of bowing to emotional pressure is that the risk assessment process is negated, and resources are directed to the wrong priorities. Hence, when assessing and communicating risk it is important that it is based upon 'good' science.

There is also a concern in some quarters about the role of third party MSDS providers. These are enterprises which provide information on chemical substances, often promoting themselves as being more credible than the chemical manufacturers. As a consequence, these third-party MSDSs may overstate the actual risk of exposure to a substance, and as an example, this author is aware of a third party MSDS for a popular brand of household laundry powder, which refers to the risk of pneumoconiosis, asthma, cancer, nasal ulceration and

perforation of the nasal septum. When contacted, the actual manufacturers were unaware of this information being disseminated on their products, allegedly on their behalf. This indicates the problems that may arise when manufacturer's or suppliers fail to exercise all due diligence in ensuring that accurate information is provided information is provided about the risks associated with their products.

4. The availability of accurate diagnoses and medical services for those affected and the financial and social burdens of such conditions

The AIOH cannot comment on the availability of accurate diagnoses or medical services, as these questions are more appropriately left to other professionals such as occupational physicians and/or occupational health nurses and the organisations representing these professions. The Australian College of Occupational Medicine and the Australian College of Occupational Health Nurses should be able to provide relevant comment.

However, occupational hygienists can assist greatly in providing information to both physicians and nurses about the nature of the work environment, which will assist in the accurate diagnosis of a worker's condition. Occupational hygienists also provide data to epidemiologists on the nature of exposures to toxic dusts which enable information to be provided on the incidence or prevalence of occupational diseases in populations.

Occupational hygienists are able to assist by providing information on:

Identification of the nature of the hazard;

Determining the risk; in the case of toxic dusts, this would be by measuring the airborne concentration of the dust in the worker's breathing zone, measuring biological indicators of exposure, eg blood lead, and comparing these with the appropriate occupational exposure standard or biological exposure indices;

Advising on the effectiveness of controls;

Analysing data for trends or patterns, e.g. by comparing exposed groups of workers, it may be possible to establish that one particular group is especially at risk and then determine the factors that have contributed to this;

Advising medical practitioners if or when some form of health surveillance or biological monitoring may be warranted;

Compiling databases e.g. of air monitoring data, for interrogation by epidemiologists, etc.

A good example of a highly successful collaborative study involving physicians, hygienists and epidemiologists is the Health Watch study, of workers in the Australian oil industry. Since 1980, the Australian Institute of Petroleum (AIP) has commissioned the development and operation of an independent epidemiology program called Health Watch. Health Watch is a research program which studies people who have worked in the Australian petroleum (oil and gas) industry to find out what happens to them in terms of their health. Health Watch follows about 18,000 past and present employees in the petroleum industry during their time in the industry and after they leave or retire. Along the way, Health Watch records any occurrence of cancer and, eventually, the cause of death. The Health Watch study began at

the University of Melbourne and was relocated to the University of Adelaide in 1998 under the direction of Dr Richie Gun, a practicing occupational physician and Senior Lecturer in Occupational and Environmental Health in the Faculty of Medicine at the University of Adelaide ([Australian Institute of Petroleum website](#)).

While the Health Watch study could be adapted as a model to study the incidence of occupational disease as a consequence of exposure to toxic dusts in the workplace (such as exposure to silica in sandblasters), it may be difficult to attract participants. The oil industry in Australia is made up of just a few large, well-resourced companies, and an active industry association that are both able to draw upon occupational hygienists, occupational physicians and epidemiologists. Most Australians, however, are employed in small to medium sized enterprises, which do not have access to these sort of resources and as a consequence, it is difficult to characterise the precise incidence and prevalence of dust related disease in the general working population.

5. The availability of accurate records on the nature and extent of illness, disability and death, diagnosis, morbidity and treatment

Some states with a long history of dust diseases have a no cost medical examination system for claimants and provide a low cost medical surveillance system to industry, eg Dust Diseases Board (NSW). Other states have no such system.

6. Access to compensation, limitations in seeking legal redress and alternative models of financial support for affected individuals and their families

This matter is outside of the competence of most members of the AIOH, and therefore it is not really appropriate to provide a comment on this subject.

7. The potential of emerging technologies, including nanoparticles, to result in workplace related harm

Australian professionals and OH&S authorities do not exist in a technical vacuum, they have strong links to international bodies that report on emerging trends. The members of AIOH are in some instances also members of the American Conference of Governmental Industrial Hygienists (ACGIH), American Industrial Hygiene Association (AIHA), British Occupational Hygiene Society (BOHS) and International Occupational Hygiene Association (IOHA) where such potential developments are openly discussed. The AIOH is aware that the National Occupational Health and Safety Commission (NOHSC) have a watching brief on issues such as nanoparticles.

Emerging technologies, by definition, represent a great unknown. As well as nanotechnology, rapid advances are being made in other areas such as biotechnology and information technology. Unfortunately, the hazards associated with new technology are not usually

immediately apparent. Indeed, as seen with silica and many other toxic dusts in the workplace, the latency period between exposure and the onset of symptoms may be 20, 30 or even 40 years. Moreover, as long as the hazards associated with emerging technologies are not known, it is not possible to legislate for their safe use, as can be done for asbestos, silica, lead or other substances for which there is a well established exposure-response relationship.

Nanotechnology

Nanotechnology and consequent nanoparticles are an emerging issue. Nanotechnologies are poised to revolutionise medicine, manufacturing, energy production and other fundamental features of everyday life in the 21st Century. But they also pose important questions that stem from the unique nature of materials and processes at the nanometer scale. Nanoparticles or ultrafine particles are defined as having aerodynamic diameters < 100 nm (< 0.1 micron). Therefore, it is disturbing to note nanotechnologies are gaining in commercial application. Nanoparticles can comprise a range of different morphologies including nanotubes, nanowires, nanofibres, nanodots and a range of spherical or aggregated dendritic forms. These materials have seen application in a wide range of industries including electronics, pharmaceuticals, chemical-mechanical polishing, catalysis, magnetic, optoelectronic, biomedical, energy and materials applications. Areas producing the greatest revenue for nanoparticles are reportedly chemical-mechanical polishing, magnetic recording sunscreens, automotive catalyst supports, bio-labelling, electro-conductive coatings and optical fibres (Aitken et. al. 2004).

Certain industrial by-products can be considered to contain nanoparticles - eg. from combustion engines (eg. diesel particulate material), furnaces and welding. There are bulk synthetic nanoparticulates - eg. titanium dioxide (TiO₂ - used in cosmetics); carbon blacks (used in pigments, tires, toner); amorphous silica (used in paints & fillers); and iron oxides. There is already work being conducted to address these industrial exposures. It should also be noted that there are natural nanoparticles in sand storms and forest fires.

The ubiquitous occurrence of airborne ultra fine particles results in significant human exposures under environmental and certain occupational conditions. Several epidemiological studies have found associations between exposure to ambient ultra fine particles and adverse respiratory and cardiovascular effects, an impetus for the Air Toxics NEPM work on PM_{2.5} particulates. Significant research has identified that inflammation is a primary health effect and oxidative stress can be identified as a dominant mechanism in the production of this inflammation. Studies have indicated that low solubility ultrafine particles are more toxic than larger particles on a mass for mass basis. There are strong indications that particle surface area and surface chemistry are primarily responsible for observed responses in cell cultures and animals.

In the realm of occupational health, much is unknown about the ways in which people may be exposed to nanomaterials through their manufacture and use in the workplace, and the potential health implications of such exposure. There are indications that ultrafine particles can penetrate through the skin, or translocate from the respiratory system to other organs. The likely adverse effects of ultra fine particles will depend on their chemical composition, their bioavailability, and their toxic effects on mucosal and neuronal cells as well as other tissue sites they enter from the general circulation. The likely health impact of ultra fine particles may include alveolar inflammation, the blood coagulation pathway and cardiovascular function. Modifying factors for these effects may include age, pre-existing disease

susceptibility and other co-pollutants. Research is continuing to understand how these unique modes of biological interaction may lead to specific health effects.

There is no technique yet available for assessing exposure, nor are there any exposure standards yet promulgated.

Three preliminary studies have been funded by the UK HSE as part of its horizon scanning activities to look at the potential hazards and risk of nanotechnology. These 'snapshot' reviews considered:

- Fire and explosion;
- Occupational hygiene; and
- Toxicological hazard.

These all reported how limited the data available was and the difficulty of reading across from existing data and that the hazards from nanoparticles and fibres are sufficiently different from bulk materials to require further careful study. They also indicated that the areas of initial occupational health concern should be:

- potential for enhanced toxicity;
- potential to cross the skin barrier;
- existing control measures are unproven; and
- possible persistence in the workplace.

These reports are available from the HSE website

(<http://www.hse.gov.uk/horizons/nanotech/>).

The First International Symposium on Nanotechnology and Occupational Health was held in October 2004 in the British town of Buxton. It brought researchers, decision makers, occupational health professionals and other stakeholders together to discuss what is known and what we still need to discover about the occupational impact of nanotechnology. Workshops at the end of the symposium considered how best to address the key information gaps that exist in order to protect workers. An informal summary from the symposium is available at www.cdc.gov/niosh/topics/nanotech/confsumm-04.html. A new initiative based in the United Kingdom, the Safety of Nanomaterials Interdisciplinary Collaboration (SnIRC), has recently commenced. The goals of the collaboration are to raise awareness about issues of nanotechnology, health and the environment; generate new research; and integrate UK research with corresponding studies in other European countries and the US. A web site describing the goals, composition and activities of the collaboration is posted at <http://www.snirc.org/index.html>. This new collaboration responds to a recommendation in the 2004 report of the Royal Society and Royal Academy of Engineering, "Nanoscience and Nanotechnologies: opportunities and uncertainties"

(<http://www.nanotec.org.uk/finalReport.htm>). Much of this information can be accessed at <http://www.cdc.gov/niosh/topics/nanotech/>

The US NTP is planning to focus its studies on the potential toxicity of nanomaterials, beginning with titanium dioxide and a limited number of manufactured nanomaterials. The first studies will be of the distribution and uptake by the skin. The US EPA is funding research at universities to examine the toxicity of manufactured nanomaterials and titanium dioxide. The agency is also providing information on the effects of nanoparticles on human health through its current and past work in ultrafine particulates. The issue of nanoparticles in aluminium smelting fume is beginning to be addressed by the work of the Norwegians, in particular Yngvar Thomassen and also by work being done in New Zealand at Auckland University by Dr Margaret Hyland. The main issue being identified is that there are significant numbers of these particles with very high surface areas with gases and the like absorbed onto them. They are occurring independently and also attached to other particles in the working environment. Most of the studies identify their presence and their potential impact on the health but as in the Thoassen report end up with a conclusion such as "the data on particle size and number concentration are not sufficient for toxicological assessment". The other issue is the general reliance on gravimetric assessments and this one relies on particle numbers and surface area more than weight. The work of Vincent, Thomassen & Hyland are indicating that ultrafines and nanoparticles may well have health impacts and will need to be included in some way, at least as a flag initially, until we see some more information on the work being done in industry.

This lack of knowledge about the possible health impacts of nanotechnology places a great responsibility on those involved in the development, import, marketing and distribution of emerging technology products to ensure that workers will not suffer any adverse response from being exposed to these products in their workplaces. Those involved in the development of products must ensure that adequate information is provided to users to ensure they are able to use the technology safely. This may for example, require them to test the product to determine its potential toxicity to humans and the environment. In this regard, there are parallels with existing schemes for controlling the introduction of new chemicals, such as the National Industrial Chemicals Notification and Assessment Scheme (NICNAS).

Changing Patterns of Employment

A further area that should be explored is the significance of any changing patterns of employment and likely dust exposed populations into the future. Will work populations in dust-exposed industries be exposed for 10, 20 or 40 years? If there is a growth towards greater long-term employment stability (eg. encouraged by factors such as portable long service and superannuation opportunities in construction industries), or market monopolies in mineral commodity suppliers, then there may be a shift towards longer dust exposed working lives (not to mention some possibilities in altered [extended] shift exposures), placing a greater importance on recommendations for reduced standards.

REFERENCES

- ACGIH (2001) Documentation of the TLVs – Silica, Crystalline Quartz, Cincinnati, OH.
- ACGIH (2005) Threshold Limit Values for Chemical Substances and Physical Agents & Biological Exposure Indices, Cincinnati, OH.
- Agricola, Georgius (1556) 'De Re Metallica', translated by Herbert Clark Hoover and Lou Henry Hoover, Dover Publications Inc, New York, 1950.
- Aitken RJ, Creely KS, Tran CL (2004), HSE Nanoparticles: An occupational hygiene review, research report 274, Health & Safety Executive, HMSO, UK.
- Australian Institute of Petroleum, Occupational Health in the Oil Industry – The Health Watch Study, <http://www.aip.com.au/health/ohs.htm>
- Badham C. (1924a) Studies in Industrial Hygiene, No 2, An investigation concerning Ventilation and the Sandstone Dust present in the Air of certain Sewer Tunnels under Construction at North Shore, and in other Sandstone Workings; in Report of the Director-General of Public Health, New South Wales, for the Year ended 31 December 1924, 52-64.
- Badham C. (1924b) Studies in Industrial Hygiene No 4, An Investigation concerning the Working Conditions and the Health of Quarrymen in Certain Government Quarries in New South Wales; *ibid*, 69-75.
- Badham C. (1924c) Studies in Industrial Hygiene No 5 An Investigation into the Sandstone Dust Hazard among Miners, Quarrymen, and Stonemasons in New South Wales; *ibid*, 76-89.
- Berry G, Rogers A, Yeung P, (2002) Lung Cancer Mortality of Compensated Silicotics in NSW, Report for NSW Dust Diseases Board Research Grants Scheme, Stages 1-5.
- Berry G, Rogers A, Yeung P. (2004) Silicosis and lung cancer: a mortality study of men compensated with silicosis in New South Wales, Australia. *J Occup Med*; 54 387-394.
- de Klerk, Ambrosini, Musk, (2002a) A review of the Australian Occupational Exposure Standard for Crystalline Silica, December 2002.
- de Klerk Ambrosini, Pang, Musk (2002b) Silicosis Compensation in Western Australian Gold Miners since the Introduction of an Occupational Exposure standard for Crystalline Silica, *Ann Occ Hyg*, 46, 687-692.
- DDB (2004) NSW Workers' Compensation Dust Diseases Board, 2004 Annual Report 2003-2004; www.ddb.nsw.gov.au
- Demers et al: (1997), What to do about softwood? A review of respiratory effects and recommendations regarding exposure limits. *Am. J. Ind. Med* 31, 385 – 398.
- Elwood, JM (1981) Wood exposure and smoking: association with cancer of the nasal cancer and paranasal sinuses in British Columbia. *CMA Journal* 124:1573 – 1577,

EPHC (2004), Industry Discussion Paper on Co-regulatory Frameworks for Product Stewardship, Environment Protection and Heritage Council, December 2004.

Francis E. (1973) Dust Concentrations in City Excavations. In Longley EO, Proceedings of the Symposium on Dust Control in Building and Excavation Work, Workers Compensation (Dust Diseases Board). p. 18-27.

George, (1947) Notes on the Incidence and Prevention of Silicosis at Broken Hill, NSW; in Silicosis Pneumoconiosis and Dust Suppression in Mines, pp 101-103).

Grantham, D.L. and Groothof, B. (2001), Report of a Blitz on Abrasive Blasting Operations throughout Queensland, DETIR.

Hernberg et al (1985) Nasal and sinonasal cancer. Scand. J. Work Environ. Health 9, 315 – 326.

HSE (2003) Respirable Crystalline Silica Phase 1 – Variability in Fibrogenic Potency and Exposure Response Relationships for Silicosis, Hazard Assessment Document, EH75/4.

IARC Monographs on the evaluation of carcinogenic risks to humans: Vol. 25 – Wood, leather and some associated industries. IARC Lyon, 1981

IARC Monographs on the evaluation of carcinogenic risks to humans: Overall evaluations of carcinogenicity, an updating of IARC Monographs volumes 1 – 42, suppl. 7. IARC Lyon, 1987.

IARC Monographs on the evaluation of carcinogenic risks to humans: Vol. 62 – Wood dust and formaldehyde. IARC Lyon 1995

Jones AT. (1968) The Control of Dust in Tunnelling and Excavation Work. In Proceedings of First Australian Pneumoconiosis Conference, Joint Coal Board. p. 351-361.

NOHSC (1993), Draft Technical Report on Crystalline Silica, September 1993.

NOHSC (1995) Exposure Standards for Atmospheric Contaminants.

NOHSC (1994a) National Code of Practice for the Labelling of Workplace Substances [NOHSC:2012(1994)], AGPS, Canberra.

NOHSC (1994b) National Code of Practice for the Preparation of Material Safety Data Sheets [NOHSC:2011(1994)], AGPS, Canberra.

PACIA, Responsible Care – Product Stewardship, (undated pamphlet), http://www.pacia.org.au/_uploaditems/docs/5.rcpsbroc.pdf

Ramazzini, Bernardino (1713), “De Morbis Artificum (Diseases of Workers), translated by Wilmer Cave Wright, University of Chicago Press, 1940.

Report of the Royal Commission on the Ventilation and Sanitation of Mines, Perth, February 1905

Rogers A, Yeung P, Berry G. (2005) Silicosis in New South Wales –An Historical Review of Occupation-Industry Sources with application to Contemporary Australian Industry. To be presented at the AIOH Annual Conference, December 2005 (in preparation).

Summons (1906), Miners phthisis: Report on the Ventilation of Bendigo Mines, extracts contained in de Silva P, Science at Work A History of Occupational Health in Victoria, Penfolk Publishing, March 2000, p27-35.

Voss et al (1985) Sinonasal cancer and exposure to softwood. Acta Laryngol (Stockh) 99, 172 – 178.

Yngvar Thomassen, Nils-Petter Skaugset, Dag Ellingsen, Lars Jordbekken, Wolfgang Koch, Wilhelm Dunkhorst (undated) Final Report Characterization of the exposure to ultrafine particles at workplaces of a primary aluminium smelter. Fraunhofer ITEM Project No: 112 062, Fraunhofer Institute of Toxicology and Experimental Medicine [Fraunhofer ITEM], Nikolai-Fuchs-Str. 1 30625 Hannover, Germany

Wan KC, and Lee E, (1993) Silicosis in Western Australia 1984-1993, presented at NOHSC National Scientific Forum on Crystalline Silica, November 1993.

Workmen's Compensation (Broken Hill) Act 1920.

Report of the Bureau of Medical Inspection Broken Hill various years to 2002

Workplace Relations Minister's Council (2002), Comparative Performance Monitoring Comparison of Occupational Health and Safety Arrangements in Australia and New Zealand, August 2002.