

## CHAPTER 2

### HEALTH IMPACTS OF WORKPLACE EXPOSURE TO TOXIC DUST

2.1 Workers may come into contact with many forms of toxic dust ranging from crystalline silica to wood dust and nanoparticles. This chapter provides an overview of the health impacts of exposure to respirable crystalline silica, beryllium, wood dust, alumina and textile dusts. The emerging issue of nanoparticle hazards is discussed in chapter 7.

#### **Crystalline silica**

2.2 Silica is a naturally occurring abundant mineral that forms the major component of most rocks and soil. Silica occurs in several crystalline forms and in amorphous non-crystalline forms. Amorphous non-crystalline forms of silica occur in nature, mainly as diatomaceous earth (the skeletons of marine organisms). The amorphous forms of silica are classified as nuisance dusts and do not induce pneumoconioses (respiratory diseases caused by inhalation of inorganic dusts). Among the crystalline forms, quartz is the most abundant, cristobalite and tridymite are less common.<sup>1</sup>

2.3 Exposure to respirable crystalline silica (RCS) occurs through cutting, chipping, drilling or grinding objects containing crystalline silica or through the use of materials that contain crystalline silica for abrasive blasting, for example sandblasting.

2.4 Workers in many occupations and industries use and come into contact with materials containing crystalline silica. Workers may come in contact with RCS through:

- excavation, where dust is created by drilling, chipping, jackhammering, etc;
- cutting to size of bricks, blocks, lightweight concrete panels, tiles, etc;
- sandblasting;
- grinding of floor slabs, granite for decorative purposes;
- concrete cutting and drilling;
- road building;
- glass manufacturing;
- refractory bricklaying;
- demolition; and

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1 *Submission 11*, pp.3-4 (DEWR).

- sweeping concrete floor slabs.<sup>2</sup>

2.5 The number of workers potentially exposed to silica in the course of their work was reported by the National Occupational Health and Safety Commission (NOHSC)<sup>3</sup> as nearly 294 000 in 2002. NOHSC noted that 'it should be kept in mind that workers in some of these industries have a different likelihood of exposure compared to those in others, that not all workers in the same industry will have the same likelihood of exposure, and the different exposed workers are likely to be exposed to different levels of silica'.<sup>4</sup>

2.6 A revised national exposure standard of 0.1 mg/m<sup>3</sup> (TWA, 8 hours) for quartz, cristobalite and tridymite came into effect on 1 January 2005.<sup>5</sup> Exposure standards are discussed in chapter 5.

### ***Exposure to crystalline silica***

2.7 Exposure to crystalline silica is known to cause a number of diseases and is linked to others.

#### *Silicosis*

2.8 Silicosis has long been known as a disease associated with mining and is caused by the inhalation of dust containing crystalline silica. Silicosis is characterised by a diffuse, nodular, interstitial pulmonary fibrosis.<sup>6</sup> Silicosis may cause breathing difficulties, chest pain, respiratory failure and lead to death. There are three main types of silicosis:

- Chronic/classic silicosis, which is the most common type, occurs after 15-20 years of moderate to low exposure. Symptoms associated with chronic silicosis may or may not be obvious in its early stages. As the disease progresses the worker may experience shortness of breath upon exercising. In the later stages the worker may experience extreme shortness of breath, chest pain or respiratory failure.
- Accelerated (subacute) silicosis, which can occur after 5-10 years of exposure to high levels of silica. Symptoms include severe shortness of breath, weakness and weight loss. The onset of symptoms takes longer than in acute silicosis. This is found in workers in occupations such as sandblasting, production of silica flour and stone masonry involving power tools.

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2 *Submission 13*, p.3 (CFMEU); NOHSC, *Regulation Impact Statement on the Proposed Amendments to the National Exposure Standards for Crystalline Silica*, October 2004, p.16.

3 In 2005, NOHSC became the Australian Safety and Compensation Council (ASCC).

4 NOHSC Regulatory Impact Statement, pp.18-19.

5 *Submission 11*, p.4 (DEWR).

6 *Submission 26*, p.3 (WHS).

- Acute silicosis, which occurs after a few months or as long as two years following exposure to extremely high concentrations of respirable crystalline silica. Symptoms include severe disabling shortness of breath, weakness and weight loss, which often leads to death. 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 and workers in abrasive manufacturing.<sup>7</sup>

### *Latency*

2.9 There was extensive discussion in evidence on the latency of chronic silicosis. Cement Concrete and Aggregates Australia (CCAA) stated that chronic silicosis has a latency that may be up to seven years after cessation of exposure: 'that is, a worker may have no symptoms or signs of silicosis either clinical or on chest X-ray at the time of cessation of exposure and then be diagnosed with clinical silicosis up to about seven years later, with little or no clinical evidence of disease in the intervening period (and no ongoing exposure)'.<sup>8</sup>

2.10 CCAA went on to state that this delayed appearance or latency is rare and 'probably 95 per cent of all cases of silicosis are diagnosable within a year of cessation of exposure, if not at the time of exposure'.<sup>9</sup> CCAA commented:

The evidence from the literature is that nearly all workers who will eventually be diagnosed as having silicosis are diagnosable at the time their exposure ceases. Some who cease work because they are unwell, or leave work without having a recent X-ray, may not actually be diagnosed until they are investigated, but this usually occurs in a short period after they report illness to their doctor. If they have been under surveillance in compliance with the Hazardous Substances Regulations governing crystalline silica (in all Australian jurisdictions) they should have had an X-ray within 5 years of ceasing exposure. It can be expected that almost all who will eventually be diagnosed as having silicosis will have evidence on those X-rays.<sup>10</sup>

2.11 CCAA stated that silicosis does not have a long latency period, comparable with mesothelioma (which may occur up to 40 years after exposure has ceased) or some other occupational cancers. Those workers whose X-ray is classed as 'no opacities' when they cease exposure, will rarely develop opacities (with or without any signs of silicosis) in later years. CCAA concluded 'latency is not a major issue in relation to silicosis, and there will not be a wave of hidden cases occurring years

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7 *Submission* 13, p.2 (CFMEU); see also *Submissions* 14, Additional information, 2.12.05 (CCAA); 20, pp.8-9 (AIOH); 32, p.2 (Dust Diseases Board of NSW).

8 *Submission* 14, Additional information, 2.12.05, p.4 (CCAA).

9 *Committee Hansard* 30.9.05, p.5 (CCAA).

10 *Submission* 14, Additional information, 2.12.05, p.4 (CCAA).

ahead. The few who do will develop those opacities within a short time of ceasing work'.<sup>11</sup>

#### 2.12 CCAA concluded:

When considering individual and isolated cases, it is possible that a worker who has retired many years may have a chest X-ray for some reason and that a radiologist at that stage may detect a opacity on the X-ray which was not evident on X-rays done at the time of exposure. When coupled with the past history of exposure, the opacity may be queried or even diagnosed as due to silica exposure. Whether this could be regarded as silicosis in the absence of any clinical signs is debatable. Many workers with X-rays which have been queried in this way in Australia in recent years, are subsequently recognised as not having silicosis ie the opacity is an artefact, or due to some other cause. In a recent series of X-rays where five were queried, an opinion from Professor Paul Wheeler at Johns Hopkins in Baltimore a world-recognised expert, was that all were due to false opacities showing up, but really caused by obesity coupled with poor X-ray technique. Early signs of silicosis on X-ray can be confused with small opacities caused by many other medical conditions.<sup>12</sup>

2.13 In response to CCAA, Dr Faunce of the Australian Sandblasting Diseases Coalition stated that it did not agree that silicosis comes on long after exposure ceases without any initial evidence:

That is simply not supported, and we would disagree with that. We would certainly disagree that 95 per cent of all silicosis cases are diagnosable within a year of cessation of exposure.<sup>13</sup>

2.14 The US National Institute for Occupational Safety and Health (NIOSH) noted that chronic silicosis develops years after exposure to relatively low concentrations of respirable crystalline silica and that epidemiologic studies have found that chronic silicosis may develop or progress even after occupational exposure has ceased.<sup>14</sup> Researchers studying silicosis compensation in Western Australia stated:

Silicosis (except acute silicosis after intense exposure) usually takes many years to develop after silica exposure has begun and therefore, may not occur until long after a subject has left the industry where the relevant exposure occurred.<sup>15</sup>

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11 *Submission 14*, Additional information, 2.12.05, p.4 (CCAA).

12 *Submission 14*, Additional information, 2.12.05, p.4 (CCAA).

13 *Committee Hansard* 10.11.05, p.36 (Dr Faunce).

14 Department of Health and Human Services, *NIOSH Hazard Review: Health Effects of Occupational Exposure to Crystalline Silica*, Publication No 2002-129, April 2002, [www.cdc.gov/niosh/pdfs/02-129.pdf](http://www.cdc.gov/niosh/pdfs/02-129.pdf), p.v.

15 de Klerk NH, Ambrosini GL, Pang SC and Musk AW, 'Silicosis Compensation in Western Australian Gold Miners Since the Introduction of an Occupational Exposure Standard for Crystalline Silica', *Annals of Occupational Hygiene*, Vol 46, No 8, pp.687-692, 2002.

2.15 Workplace Health and Safety Queensland (WHS) noted that 'there is general consensus amongst the researchers that the latency period of most cases of silicosis is in excess of twenty years from first exposure'.<sup>16</sup>

### *Risks to the community*

2.16 The Committee received submissions from members of communities living in the vicinity of quarries and smelters which raised concerns about the potential for members of the community to develop dust-related disease.

2.17 The Somersby Action Committee reported that due to extensive quarry activities on the Somersby Plateau NSW, residents experienced exposure to silica dust. Dust also affects schools and businesses in close proximity to quarries. There have been reports of the increased incidence of asthma and concerns that the community has been put at risk of silicosis. The Action Committee pointed to problems with policing sites to ensure that dust is minimised. Although fines have been imposed, these are considered to be too low to act as a deterrent. As there are proposals to expand quarries in the area, the Action Committee called for greater protection of populations living near quarries, more regular inspection of work sites, and independent environmental impact statements.<sup>17</sup>

2.18 CCAA stated that there have been no observances of silicosis arising from exposure to RCS in the community. This was not only the case in Australia, but overseas as well. Silicosis was seen as an industrial problem, not a community problem:

...any source of silica dust that is industrial is dissipated in terms of its intensity very rapidly by distance. So, although there is a theoretical possibility that somebody could be living next to a source of respirable silica dust, in practice nobody has ever found such a case.<sup>18</sup>

CCAA went on to note that there is also monitoring of exposures including around all industrial sites and around the perimeters. Controls are also in place to prevent dusts from escaping and organisations can and are prosecuted for failing to meet those standards.<sup>19</sup>

2.19 The Whyalla Red Dust Action Group voiced concern at the dust exposure of residents near the Onesteel Steelworks in South Australia. Fine iron ore dust is emitted from the works and the Action Group indicated that the dust seriously contaminates 1000 homes, public facilities, schools, businesses and sporting facilities. It noted that in July 2003, the South Australian Environment Protection Agency and the Department of Human Services 'issued a joint public statement which advised the

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16 *Submission 26*, p.3 (WHS).

17 *Committee Hansard 30.9.05*, pp.40, 43 (Somersby Action Committee).

18 *Committee Hansard 30.9.05*, pp.1, 4 (CCAA).

19 *Committee Hansard 30.9.05*, p.17 (CCAA).

exposed community that it may suffer adverse health effects from exposure to Onesteel's dust'.<sup>20</sup>

### *Airway disease*

2.20 While silicosis has long been identified as an occupational disease arising from inhalation of dust containing crystalline silica, there has been some dispute over the association of airway disease with crystalline silica. There was extensive discussion in evidence as to the incidence of airway disease related to toxic dust and in particular the compensation case of Mr Richard White arising from his exposure to RCS.

2.21 Chronic obstructive pulmonary disease (COPD) refers to a combination of cough and phlegm, breathlessness and airflow obstruction. Professor E Haydn Walters, University of Tasmania, stated that generally, 'it is likely that somebody will go from having some irritant cough and a bit of sputum to gradually developing some airflow obstruction to then becoming symptomatic and breathless on exercise perhaps over a 15- to 20-year period if they have moderate dust exposure which is continuing'.<sup>21</sup>

2.22 Professor Walters noted that COPD is a common problem in Australian society and is usually due to cigarette smoking. The Professor commented that it appeared that the case of Mr White 'suffered from legal and medical preconceptions' that 'airway disease is either classic "asthma"...or chronic obstructive pulmonary disease...caused by cigarette smoking'. Further it also appeared that there was a view that exposure to silica and other toxic dust causes lung parenchymal fibrosis or silicosis and not airway disease. The Professor went on to state:

The idea that occupational dusts and fumes can also give rise to airway disease and be a cause of fixed obstructive airway disease, but at doses to the lung insufficient to give clinically evident lung fibrosis, seems to have been slow to be accepted. However, I think the evidence is now becoming really quite strong and generally accepted that this is indeed the case.<sup>22</sup>

2.23 The Professor pointed to a study undertaken in Melbourne which showed that exposure to organic dusts was a significant cause of COPD in non-smokers rather than the general assumption it would all be due to cigarette smoking. The Professor concluded:

This does not relate of course directly to silica exposure, which is a non-organic mineral dust, but it does show in a general sense that occupational dusts are not insignificant in contributing to the burden of COPD in Australia. The population that we were dealing with in Melbourne would not have been significantly exposed to silica dusts but this does not mean

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20 *Submission 1*, p.2 (Whyalla Red Dust Action Group).

21 *Committee Hansard* 10.11.05, pp.26, 28 (Prof EH Walters).

22 *Submission 3*, p.2 (Prof EH Walters).

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that in relevant populations that this would not also be potentially of importance.<sup>23</sup>

2.24 The Professor informed the Committee that internationally there is now increasingly wide acceptance that non-organic dusts can also be a cause of fixed airflow obstruction and chronic bronchitis, and that this may be either additive to cigarette smoking or might be more evident in smokers. The Professor pointed to two recent papers. The first, a literature review by the UK Institute for Environment and Health concluded that the literature suggested that there are clearly elevated risks of developing COPD associated with several occupations including welding, flour mill work and cotton textile work.<sup>24</sup> Secondly, the US National Institute for Occupational Safety and Health (NIOSH) published a hazard review on RCS in 2002. The review concluded that silica is one of a number of occupational dusts associated with COPD. The review also noted that some studies suggest that these diseases may be less frequent or absent in non-smokers.<sup>25</sup>

2.25 In addition, in 1999 British miners were recognised as suffering a high incidence of COPD in relationship to mineral dust exposure, even in the absence of classic Coal Workers' Pneumoconiosis (CWP). Subsequently, the British Government assessed miners and ex-miners and provided compensation. Professor Walters noted that 'this has really been an extremely important development and a mind shift in terms of recognition that bronchitis and COPD are not just cigarette smoker diseases but also a disease of dust exposed workers'.<sup>26</sup>

2.26 The Australian Institute of Occupational Hygienists (AIOH) also commented on airway disease and noted that it has been statistically associated with some occupational groups such as miners who may have been exposed to long term high dust exposures. AIOH went on to comment that:

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.<sup>27</sup>

2.27 The Committee also notes that in its Regulation Impact Statement on the Proposed Amendment to the National Exposure Standards for Crystalline Silica in

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23 *Submission 3*, p.3 (Prof EH Walters).

24 Institute for Environment and Health, *Review of Literature on chronic Bronchitis and Emphysema and occupational Exposure*, January 2005, p.4.

25 *NIOSH Hazard Review*, p.vi.

26 *Submission 3*, p.3 (Prof EH Walters); see also *Committee Hansard* 10.11.05, pp.29, 30 (Prof EH Walters).

27 *Submission 20*, p.12 (AIOH).

October 2004, NOHSC stated emphysema, the main cause of chronic obstructive lung disease, can be caused by inhalation of crystalline silica and that silica dust can worsen the damage done by smoking.<sup>28</sup>

### *Lung cancer*

2.28 Since 1997 silica has been listed as a Class One carcinogen by the International Agency for Research on Cancer (IARC).<sup>29</sup> In 2002 NIOSH commented that 'the carcinogenicity of crystalline silica in humans has been strongly debated in the scientific community'.<sup>30</sup> The NOHSC Regulation Impact Statement (2004) stated that 'the balance of evidence suggests that RCS exposure causes lung cancer' but that 'there is dispute as to whether RCS exposure causes lung cancer directly, or whether RCS exposure causes lung cancer indirectly, i.e., whether the development of silicosis increases the risk of lung cancer'.<sup>31</sup>

2.29 The Regulation Impact Statement provided the following comparison of carcinogen classifications of crystalline silica.

**Table 2.1: International carcinogen classification of crystalline silica**

| <b>International Body</b>  | <b>Carcinogen Classification</b>                                   |
|--|--|
| International Agency for Research on Cancer (IARC)                     | Crystalline silica – human carcinogen                              |
| National Institute of Occupational Safety and Health (NIOSH, USA)      | Crystalline silica – <i>potential</i> occupational carcinogen      |
| National Toxicology Program (NTP, USA)                                 | RCS – known to be a human carcinogen                               |
| British Health & Safety Executive                                      | RCS – causes lung cancer, but is probably a <i>weak carcinogen</i> |
| American Conference of Governmental Industrial Hygienists (ACGIH, USA) | Crystalline silica – <i>suspected</i> human carcinogen             |

*Source: NOHSC, Regulation Impact Statement on the Proposed Amendment to the National Exposure Standards for Crystalline Silica, October 2004, p.14.*

2.30 In evidence differing views on the link between RCS and lung cancer were also expressed. AIOH stated that:

28 NOHSC, Regulatory Impact Statement, p.13.

29 *Submission 13*, p.2 (CFMEU).

30 *NIOSH Hazard Review*, p.v.

31 NOHSC, Regulatory Impact Statement, p.14.



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Several studies among the many reviewed by the [International Agency for Research on Cancer] 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.<sup>32</sup>

2.31 AIOH went on to note that an Australian study indicated that 'long term exposure to high levels of crystalline silica has also been associated with increase in lung cancer. 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.'<sup>33</sup>

2.32 Mr Lindsay Fraser of the Construction, Forestry, Mining and Energy Union (CFMEU) noted that both IARC and NIOSH accept that exposure to crystalline silica is a carcinogen and stated 'so I dispute the evidence that there is now equivocation on that. It is accepted by the world's medical and scientific professions that it is a carcinogen and that it can lead to a horrid death.'<sup>34</sup>

2.33 The Minerals Council of Australia (MCA) stated that in the review of the silica standard it had put the view that there was 'uncertainty in the epidemiological evidence linking exposure to silica to lung cancer, especially in those workers where there was no evidence of silicosis'.<sup>35</sup> The CCAA went further and called the IARC's listing of silica a 'controversial decision' and went on to state that the original decision has been disputed by members of the original IARC panel since that time. In addition, CCAA commented that the IARC panel only considered epidemiological evidence up to 1994 and the more current research on workers, for example in the UK sand industry, indicated no excess risk of lung cancer or other cancers. CCAA quoted from recent reviews undertaken for the American Chemical Society:

...the literature does not support the view that silica dust causes lung cancer, nor does it suggest that silicosis is a cause of lung cancer. Further, the data indicate that the current (and probably the former) TLV-TWA for silica dust is protective for silicosis with an adequate margin of safety.<sup>36</sup>

2.34 CCAA also provided the Committee with a paper on research conducted into the British sand industry carried out by the Institute for Environment and Health.

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32 *Submission 20*, p.10 (AIOH).

33 *Submission 20*, p.12 (AIOH).

34 *Committee Hansard 30.9.05*, p.22 (CFMEU).

35 *Committee Hansard 10.11.05*, p.15 (MCA).

36 *Submission 14*, Additional information, 2.12.05, p.5 (CCAA).

CCAA stated that the 'paper shows beyond reasonable doubt that there is no cancer risk in that industry, and also indicates that any risk of silicosis is extremely low (although that issue was not the primary focus for the research)'.<sup>37</sup>

2.35 WHS also commented that 'crystalline silica has been found to be a carcinogen in animals (rats) but it is yet to be proven in humans from epidemiology studies' and noted that:

It was this revelation that led the International Agency for Cancer Research (IARC) to classify crystalline silica as a group 1 human carcinogen. (Brown and Rushton, 2005; Verma, Purdham and Roels, 2002). Brown's research (2005) did not find any consistent correlation between respirable crystalline silica and the development of lung cancer.<sup>38</sup>

WHS went on to state that in studies of NSW Dust Diseases Board compensation cases the excess lung cancer risk amongst compensated silicotics corrected for smoking, was found to be 1.90 (confidence interval 1.54 to 2.33). WHS commented that this 'is highly significant so lung cancer remains an issue. Whether it remains to be so in the absence of silicosis is not yet fully clear and is expected to become a battleground for plaintiff lawyers and compensation bodies during the next 10 to 20 years'.<sup>39</sup>

2.36 The Dust Diseases Board of NSW has also commented that there is much debate as to whether silicosis is a pre-requisite to the development of lung cancer. In addition, 'smoking is believed to increase the risk, possibly 2-fold more than in non-smokers'. Lung cancer developing in patients with silicosis is accepted for compensation by the Board, 'even if the person has smoked or is smoking'. Approximately 8 per cent of all compensated lung cancer cases are in association with silicosis.<sup>40</sup>

#### *Other diseases related to respirable crystalline silica*

2.37 Occupational exposures to respirable crystalline silica can also have heart effects. In severe cases, fibrosis in the lungs can lead to prolonged increase in the blood pressure in the arteries and veins of the lungs (pulmonary hypertension).<sup>41</sup> Exposure may also be related to the development of autoimmune disorders (such as scleroderma, systemic lupus erythematosus and rheumatoid arthritis), chronic renal disease and other adverse health effects.<sup>42</sup>

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37 *Submission 14*, Additional information, 2.12.05, p.5 (CCAA).

38 *Submission 26*, pp.3-4 (WHS).

39 *Submission 26*, p.7 (WHS).

40 Dust Diseases Board, *A Guide to Compensated Occupational Lung Disease in NSW*, p.6.

41 NOHSC Regulatory Impact Statement, p.13.

42 *NIOSH Hazard Review*; see also *Submission 26*, p.5 (WHS).

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## Beryllium

2.38 Beryllium copper alloy or copper-beryllium is exceptionally strong and hard and is an excellent electrical and thermal conductor, nonmagnetic and resistant to corrosion and fatigue. Beryllium oxide is an outstanding conductor of heat. These metals are used in the automotive industry, mining, glass manufacturing, smelters, foundries, ship manufacture, dental laboratories (crown and bridge), bicycle frames, aerospace, nuclear power, aviation and electrical instruments. They are used extensively in aircraft: engines, auxiliary power units (APUs), aircraft main and nose landing gear brushes, wheels and brakes, in airframe structures, in helicopters and in some jet engine igniter plug firing tips, electrical wiring, instruments, communications systems including radios, radar, computers, and weaponry systems.<sup>43</sup>

2.39 Inhaled beryllium dust particles causes lymphocytes in the lungs to become sensitised and then proliferate. As the cells react to the particles they form clumps that rob the lungs of their elasticity and make it difficult to breath. Exposure to high concentrations of beryllium dust results in acute beryllium disease (ABD). Symptoms of ABD include shortness of breath, cough, chest pain, and rapid heart beat. Workers generally recover from ABD but some will develop chronic beryllium disease (CBD). CBD is incurable, although when caught early, symptoms can be suppressed with steroids. CBD can damage the lungs, liver and spleen. It can also cause skin ulcers and other rashes. CBD has a long latency, appearing up to 40 or more years after initial exposure. It occurs in as much as 17 per cent of workers in particularly risky occupations, such as those who work in machine shops or in construction where beryllium is used.<sup>44</sup>

2.40 Mr John Edwards stated that he and others considered CBD to be far worse than asbestos-related lung diseases as CBD can affect every major organ of the human body: lungs, heart, eyes, kidneys, liver and joints; and cause fibrosis.<sup>45</sup>

## Timber dust

2.41 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, for example Western red cedar, Californian redwood, spruce and some pine species, and a few hardwoods, for example blackwood, messmate and rosewood.

2.42 AIOH also noted that hardwood dust has been associated with adenocarcinoma of the nasal sinuses, especially in those industries requiring fine,

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43 *Submission 45*, p.2 (Mr J Edwards).

44 Newman LS, 'Toxic Beryllium: New Solutions for a Chronic Problem', *Environmental Health Perspectives*, Vol 109, No 2, Feb 2001; see also *Submission 24*, p.5 (Dr G Pain).

45 *Submission 45*, p.1 (Mr J Edwards).

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. Softwood dust may be associated with squamous cell carcinoma of the nasal sinuses. The average reported time between first exposure to wood dust and diagnosis is around 40 years.<sup>46</sup>

2.43 Fibreboard dust is another hazard. Fibreboard is made from wood fibres bonded together with a resin. Construction workers doing flooring and wall panelling handle large amounts of particleboard or fibreboard. These custom woods contain formaldehyde, which can possibly cause cancer in humans. Machining operations such as sawing, drilling and sanding can generate large amounts of airborne wood dust. Inhaling formaldehyde can cause burning sensations in the eyes, nose and throat and a range of other symptoms if higher levels are in the air.<sup>47</sup>

2.44 The CFMEU stated:

Since [medium density fibreboard] has been introduced, there have been all kinds of problems associated with it. There is a formaldehyde problem that has been dealt with here in Australia. We have the lowest formaldehyde emission of anywhere in the world – that is by agreement between the manufacturers and ourselves...But wood dust is a known carcinogen. Nobody has ever argued it is not. The employers know that. The manufacturers know that. There are all kinds of regimes set up for when you are cutting hardwoods, about the extractors and the cutting rooms, so we do not see that as quite the same problem as silica.<sup>48</sup>

2.45 Exposure standards for timber dusts have changed over the years as more has become known about the hazards of particular timber species. AIOH commented that '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'. Proposals have been made in the US to reduce further the exposure standards for both allergenic and carcinogenic species.<sup>49</sup> The exposure standards for formaldehyde have also reduced over time.

2.46 Dr John Bisby for CCAA commented on the high risks associated with wood dust:

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46 *Submission 20*, p.16 (AIOH).

47 *Submission 28*, p.4 (ACTU).

48 *Committee Hansard 30.9.05*, p.33 (CFMEU).

49 *Submission 20*, p.17 (AIOH).

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It is killing Australians today. The incidence of certain cancers in wood workers exposed to wood dust is 50 times or more. Not 50 per cent; 50 times. And that is today. In our group we have seen about 30 [cases].<sup>50</sup>

Dr Bisby went on to state that 'wood dust is a bigger issue [than silicosis] because nobody is aware of it'.<sup>51</sup>

2.47 AIOH concluded that '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.'<sup>52</sup>

## **Alumina**

2.48 Aluminosis is the occupational lung diseases seen in workers exposed to the fine aluminium powder or dust. The disease is characterised by a scarring of lung tissue after prolonged inhalation. The degree of scarring is related to the duration of a worker's exposure to the dust, the concentration of the dust in the air and the fineness of the particles.<sup>53</sup>

## **Textile dusts**

2.49 Byssinosis is an occupational airways disease seen in textile workers due to the inhalation of certain textile dusts. The symptoms include chest tightness, wheezing and shortness of breath. Initial symptoms appear several hours after arriving at work on the first day of the working week or the first day back from a holiday. They generally improve over the course of the week and do not recur until the beginning of the following week after the individual has had at least two days of no exposure to textile dust. With prolonged and intense exposure the individual's symptoms may progress to become continuous throughout the week, both at work and home. This continuous irritation of the airways can lead to permanent irreversible impairment of a worker's lung function. This condition is now rare.<sup>54</sup>

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50 *Committee Hansard* 30.9.05, p.7 (Dr Bisby).

51 *Committee Hansard* 30.9.05, p.15 (Dr Bisby).

52 *Submission* 20, p.18 (AIOH).

53 *Submission* 32, p.4 (Dust Diseases Board of NSW).

54 *Submission* 32, p.3 (Dust Diseases Board of NSW).



