Abstract
Exposure to respirable particles of crystalline silica is associated with a number of human diseases (including cancer and lung disease). Silica related disease is preventable although once present it is incurable.

Disease risk is related to both the total dose and duration of silica exposure and the onset of the disease may occur years after the exposure.

Silicosis is regarded as an industrial disease although non-occupational exposures can lead to the development of fibrotic nodules, which characterise the current diagnosis of the disease, in exposed populations. The available literature suggests ambient crystalline silica levels can be significantly elevated downwind of sites that may release silica particles.

There is sufficient information on silicosis dose/response relationships and risk to develop an ambient air quality standard to protect the general population (including sensitive subgroups) from silica related disease downwind of industrial sources of silica.

This paper examines the disease risks associated with silica exposures and presents a methodology for establishing an air quality standard to protect exposed populations from silica related disease.

Introduction
Although there appears to be a low level of risk of contracting silica related disease from background levels of exposure (OEHHA 2005), there remain significant concerns over the risk of environmental exposures near peak sites (Roperto et al. 1994, Roperto et al. 1995, Baltais 2008 pers com., Somersby Action Committee 2006).

This paper examines the inhalation risk associated with non-occupational exposures to respirable crystalline silica near peak sites. In this context peak sites are sites which are likely to release respirable crystalline silica particles into the nearby airshed including quarrying (both hard rock and sand), mining, concrete batching, industrial sites (such as foundries and ceramic
works), concrete cutting, and demolition sites. Communities living immediately adjacent to these sites are potentially exposed to elevated concentrations of respirable crystalline silica.

This paper uses accepted risk paradigms including hazard characterisation, dose response and exposure (qualitative and quantitative) and environmental risk (qualitative and quantitative) (NH&MRC 2006) to examine the level of environmental risk near peak sites and the potential harm that respirable crystalline silica may cause to exposed populations.

Influence of mineralogy on silica hazard

Silica [or silicon dioxide (SiO$_2$)] is an abundantly occurring natural compound. WHO (2000) identifies that silica can be either crystalline or non-crystalline (amorphous). Crystalline silica has a distinct structure whereby the silicon and oxygen atoms are arranged in a three dimensional repeating pattern, whereas amorphous silica does not exhibit the repetition.

Of greatest concern to researchers, due to its effects on the health of exposed populations, is crystalline silica of which there are seven recognised forms or polymorphs i.e., quartz, cristobalite, trydimite, coesite, stishovite, moganite and melanophlogite (Republic of South Africa (RSA) 2006). Quartz is the most common polymorph of crystalline silica and is the second most commonly found mineral and is found in most terrestrial rocks and sedimentary deposits (ibid).

Silica disease

There is a strong body of evidence for a causal relationship between exposure to crystalline silica and disease (Rosner and Markowitz 2006). A number of papers (for example ATS 1997; Calvert et al. 2003; Goldsmith, 2006; Green and Vallyathan, 1996; IARC, 1997; McDonald et al. 2005; Rafnsson et al. 1998; and Steenland 2005) have identified that exposure to crystalline silica can result in physiological changes, disease and death in exposed populations caused by a range of diseases. These include silicosis, lung cancer, renal disease, kidney cancer, chronic obstructive pulmonary disease (COPD), scleroderma, rheumatoid arthritis, polyarthritis, mixed connective tissue disease, systemic lupus erythematosus, Sjögren's syndrome, polymyositis, fibrositis, cor pulmonale, lymphatic cancers (leukemia, lymphomas), stomach and/or gastrointestinal malignancies, dermatomyositis, glomerulonephritis.
There is a reliable relationship between cumulative silica dust exposure and increased mortality from lung cancer (Chen et al. 2006). This increase in lung cancer occurrence is independent of smoking (Berry et al. 2004). ATS 1997 report that silicotic patients have increased risk for lung cancers stating "...silicosis should be considered a condition that predisposes workers to an increased risk of lung cancer..."

Calvert et al. (2003) concluded that there is a link between crystalline silica exposure and rheumatoid arthritis. The link between silica and the autoimmune diseases requires further research to clarify the understanding of the dose response relationship and risk; particularly at low dose rates (Parks et al. 1999, Goldsmith 2006).

Additionally, there is an established link between silicosis and tuberculosis, as noted by ATS (1997 p761) “...the association between tuberculosis and silicosis has long been recognised... and a recent US survey documents a substantially higher tuberculosis mortality associated with silicosis in the US in the period 1979 – 1991...” and “…some data suggest that subjects without silicosis but with long exposures to silica dust have an excess risk of developing tuberculosis compared with the non-exposed population...” (ibid p763).

The factors of significance in the development of silica disease include: size of the particle; concentration of silica particles in the air, duration of exposure, particle surface characteristics including the age of the particle and the concentration of trace metals such as iron (Castranova et al. 1997, NIOSH 2002, Vallyathan et al. 1988, Vallyathan et al. 1995, ATS 1997, Steenland and Brown 1995).

The particle size affects the portion of the airway where particles are deposited. NIOSH (2002) identifies that silica related disease is caused by the inhalation and deposition [in the lung] of respirable crystalline silica particles. Respirable particles are defined as having a mean aerodynamic diameter in the size range less than 4μm (ibid p xvi). These particles are considered to be capable of penetrating through to the alveoli leading to the development of silica related disease (ibid).

The onset of silica disease is a function of the concentration of respirable silica particles and the duration of the exposure. Silicosis can arise from long duration exposures at low concentrations (either as simple chronic silicosis or complicated chronic silicosis), from shorter exposure at
higher concentrations (accelerated silicosis) and as acute silicosis (very short duration exposure to very high concentration) (NIOSH 2002).

RSA 2006 indicates that chronic silicosis is mainly the result of long term exposure; accelerated silicosis can develop after five to ten years exposure; and acute silicosis can occur very rapidly (from a few months to less than five years) following significant exposure. Other studies (such as ATS 1997; Hinzido and Sluis-Cremer 1993; Muir et al. 1989,) identified that silicosis can occur (or progress) some years after cessation of exposure. This phenomenon was subsequently demonstrated in rat experiments (Porter et al. 2004).

The risk of silicosis is a function of the cumulative dose, although the latency period between exposure and diagnosis appears to be independent of the dose (Hinzido and Sluis-Cremer 1993).

Rimal et al. 2005 provide further detail on the pathology of silicosis, stating (p169) “...There are several clinical and pathological varieties of silicosis including simple (nodular) silicosis, acute silicosis (silicoproteinosis), complicated pneumoconiosis (progressive massive fibrosis), and true diffuse interstitial fibrosis….”

The toxicity of silica appears to be related to the age of the inhaled particles (Vallyathan et al. 1988), a conclusion confirmed by Guidotti and Koehncke (1998) who showed that a relationship between freshly fractured particles and toxicity exists, with exposure to particles within six hours of fracturing having a higher biological activity. Freshly fractured silica particles are considered to have a greater potential to damage cells and stimulate reactive species production by alveolar macrophages (NIOSH 2002, Castranova et al. 1997).

The research suggests that freshly fractured silica particles generate silica based radicals on the fracture planes and hydroxyl radicals in aqueous media. Furthermore, freshly fractured silica with iron contamination results in enhanced generation of reactive radicals and is shown to be more inflammatory in the lung and cause significantly more damage to the alveolar air-blood barrier than equivalent exposure to crystalline silica particles associated with low iron contamination (Fubini 1998, Castranova et al. 1997).
The synergistic relationship between silica and iron reported by Castranova is in contrast with results reported elsewhere (such as Cullen et al. 1996). Castranova et al. 1997 investigated and resolved the apparent conflict through an analysis of the dose of iron that is required to neutralise the surface charge of freshly fractured silica, reporting (p1323) "...In conclusion the negative surface charge of crystalline quartz contributes to its unique toxicity. Gross quantities of Fe can coat the quartz surfaces and neutralize this surface charge to partially detoxify quartz. However, the required Fe:quartz particulate ratio of greater than 12:1 is not likely in occupational settings where silicosis is a concern. In contrast, trace contamination of quartz with Fe may occur in rock drilling or sand blasting. Such trace Fe could catalyze the generation of \(^{\cdot}\)OH by freshly fractured quartz leading to increased lung damage and inflammation..."

The development of silica related disease was shown to be a function of the free silica burden in the alveolar region, the silica burden on the alveolar macrophages, the burden in the interstitial region and the burden in the lymph nodes (Tran et al. 2005).

Silica deposited in the alveolar region is phagocytosed by the alveolar macrophages and are cleared upwards in the bronchial tree. The clearance is unimpaired while the silica burden remains below a critical level [suggesting the presence of an exposure level that makes the risk of silicosis negligible]. However, once this critical level is reached further clearance becomes impaired and an inflammatory reaction is initiated (ibid).

Clearance of the silica particles is impaired by the toxic effects of silica destroying the macrophages, resulting in a re-release of the silica particle back into a free state (Rimal et al. 2005) This has the effect of compounding the dust burden in the alveolar region further stimulating the inflammatory response (Tran et al. 2005). As indicated above, the surface chemistry of freshly fractured silica has a greater toxic effect on the alveolar macrophages which may result in reduced clearance of these particles.

Silica-induced inflammatory response has been implicated in the pathogenesis of fibrosis (Rao et al. 2004). Rimal et al. (2005) report that quartz treated human macrophages release growth factors which stimulate collagen synthesis of fibroblasts.

The initiation of silicosis occurs once the silica burden exceeds the ability of the alveolar macrophages to clear the deposited dose of silica in the alveoli. Furthermore, Tran et al. 2005
states (p31) "... it is correct that in temporal order the inflammation starts before the fibrosis. However once the retained silica dose has reached the point of initiating inflammation, clearance has stopped, and the progression to fibrosis is inevitable..." 

Using data obtained from rat experiments Tran et al. (2005) modelled the formation of fibrosis, reporting (p18): "...However, it associates the formation of fibrosis with a second wave of inflammatory reaction... The identification of a second threshold dose associated with fibrosis is still in need of validation by further experimentation. However, it is worth noting that clearance ceases once the first threshold is reached. If exposure continues, it will become difficult to avoid reaching the second threshold [leading to the development of fibrosis]..."

**Quantitative Dose Response**

Occupational Exposure limits are available for crystalline silica. In Australian workplaces the Time Weighted Average (TWA) is 0.1 mg/m$^3$. The TWA is set for a standard (8 hr day) working day (Commonwealth of Australia 2004). The USA guidelines and limits include: NIOSH recommended exposure limit 0.05 mg/m$^3$ (for a 10 hour workday during a 40 hour week); ACGIH (American Conference of Governmental Industrial Hygienists) TLV 0.05 mg/m$^3$ (8 hr TWA) (NIOSH 2002).

These occupational levels appear to retain an associated degree of residual risk of developing silicosis. OEHHA (2005) reported (p21): "...Silicosis is still being diagnosed at death in workers who were supposed to be exposed to occupational levels of 50-100 μg/m$^3$ [0.05 - 0.1 mg/m$^3$]..." Steenland and Brown (1995) reported a lifetime (75 years) silicosis risk for someone exposed for a working life (estimated to be 45 years) at 0.09 mg.m$^3$ to be 35 to 45% (i.e. for every 100 workers exposed during their 45 year working life at 0.09 mg/m$^3$ (90μg/m$^3$) there would be 35 to 45 cases of silicosis by age 75). Estimated mortality risk from pooled occupational cohort studies was presented by Mannetje et al. (2002). This study reported that the mortality risk from 45 years exposure is 13 deaths per 1000 workers at an occupational exposure rate of 0.1 mg/m$^3$ (100 μg/m$^3$) and 6 deaths per 1000 workers at 0.05 mg/m$^3$ (50 μg/m$^3$).

Goldsmith (2006) predicted lifetime risks of mortality from lung cancer (assuming 45 years exposure to respirable crystalline silica (estimates are for 1000 workers) (refer Table 1).
Table 1: Lifetime risks of mortality from lung cancer from respirable crystalline silica (Goldsmith 2006)

<table>
<thead>
<tr>
<th>Crystalline silica concentration (mg/m³)</th>
<th>Predicted Lung Cancer Deaths per 1000 workers (a)(b)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.001</td>
<td>0.3</td>
</tr>
<tr>
<td>0.005</td>
<td>1.5</td>
</tr>
<tr>
<td>0.01</td>
<td>2.9</td>
</tr>
<tr>
<td>0.02</td>
<td>5.9</td>
</tr>
<tr>
<td>0.03</td>
<td>8.8</td>
</tr>
<tr>
<td>0.04</td>
<td>12</td>
</tr>
<tr>
<td>0.05</td>
<td>15</td>
</tr>
<tr>
<td>0.06</td>
<td>18</td>
</tr>
<tr>
<td>0.07</td>
<td>20</td>
</tr>
<tr>
<td>0.08</td>
<td>23</td>
</tr>
<tr>
<td>0.09</td>
<td>26</td>
</tr>
<tr>
<td>0.1</td>
<td>29</td>
</tr>
</tbody>
</table>

a: Assumes constant 45 years exposure between age 20 and 65 and thereafter accumulating annual risks to 85 years; and

b: Excess risk estimates/1000 workers exposed (i.e. the excess lifetime risk for lung cancer at 0.1 mg/m³ silica is 29 deaths/1000 workers).

Tran et al. (2005) estimated a No Observed Adverse Effect Level (NOAEL) from animal studies and epidemiological reports from occupational studies (Buchanan et al. 2003 cited in Tran et al. 2005). These risks were presented in terms of developing an ILO classification 2/1+ silicosis (refer Table 2) from 15 years exposure and 15 years post exposure (refer Table 3).
Table 2. International Labor Office (ILO) categorisation of silicosis (ILO 1980).

<table>
<thead>
<tr>
<th>ILO Category</th>
<th>Qualitative Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0/0</td>
<td>No small (up to 1 cm) silicotic opacities (nodules) are present</td>
</tr>
<tr>
<td>0/1</td>
<td>Probably no nodules, but some areas of radiograph are suspect [possible silicosis]</td>
</tr>
<tr>
<td>1/0</td>
<td>Small silicotic nodules are most likely present, but not certainly [probable silicosis]</td>
</tr>
<tr>
<td>1/1</td>
<td>Small silicotic nodules are definitely present</td>
</tr>
<tr>
<td>1/2</td>
<td>Small silicotic nodules are definitely present; other areas of the radiograph may indicate more advanced lesions including large opacities (&gt; 1 cm), pleural thickening, etc.</td>
</tr>
<tr>
<td>2/1, 2/2, 2/3, 3/2, 3/3</td>
<td>More advanced stages of silicosis/increasing certainty of the presence of lung abnormalities</td>
</tr>
</tbody>
</table>

Table 3: Silicosis risk (Tran et al. 2005)

<table>
<thead>
<tr>
<th>Exposure Concentration (mg/m³)</th>
<th>Duration</th>
<th>Exposure Concentration (mg.yrs.m⁻³)</th>
<th>Estimated risk (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.0</td>
<td>15 years + 15 years post-exp</td>
<td>0</td>
<td>0.79</td>
</tr>
<tr>
<td>0.01</td>
<td>15 years + 15 years post-exp</td>
<td>0.15</td>
<td>0.89</td>
</tr>
<tr>
<td>0.025</td>
<td>15 years + 15 years post-exp</td>
<td>0.375</td>
<td>1.06</td>
</tr>
<tr>
<td>0.03</td>
<td>15 years + 15 years post-exp</td>
<td>0.45</td>
<td>1.12</td>
</tr>
<tr>
<td>0.1</td>
<td>15 years + 15 years post-exp</td>
<td>1.5</td>
<td>2.48</td>
</tr>
</tbody>
</table>

a: Calculated as the risks of reaching ILO classification grade 2/1+

Table 3 suggests that for every 1000 workers exposed for 15 years at the Australian TWA limit there are predicted to be 248 cases of silicosis at a grade 2/1+ after a further 15 years post exposure.
Tran et al. (2005) report that the occupational NOAEL (less than 1 case per 1000 workers) is 0.01 mg/m$^3$ (i.e. 10μg/m$^3$). From the animal studies in their report the Human NOAEL (extrapolated from the animal-based NOAEL and inclusive of intra-human variation) is 0.0011 mg/m$^3$ (i.e. 1.1 μg/m$^3$). It is noted, by Tran et al. 2005, that the reported human NOAEL may be overly conservative and this is attributed to the application of currently acceptable uncertainty factors.

**Exposure outcomes (Diagnosis of silicosis)**

Silicosis is currently diagnosed on the basis of exposure history and the presence of fibrotic nodules on a chest radiograph or CT scan (Muir et al. 1989, NIOSH 2002, WHO 2000). Although lung function, sputum and lung biopsies can be used (RSA 2006).

In simple silicosis the nodules are up to one centimetre in size. Larger nodules can develop through prolonged exposures (complicated chronic silicosis) or where the nodules coalesce and form progressive fibrosis and reduction of lung volume or sometimes form large conglomerate masses (progressive massive fibrosis). Acute silicosis is characterised by high exposures and pulmonary edema, interstitial inflammation and accumulation in the alveoli of proteinaceous fluid (Rimal et al. 2005).

The use of a radiograph as the diagnostic tool in determining the commencement of silicosis is considered, by some researchers, to be subjective and unreliable and subject to variability between readers Muir et al. (1989). NIOSH 2002 identifies the potential for false negatives in radiological readings.

The use of radiographs to diagnose silicosis on the basis of fibrotic nodules does not lead to prevention of the disease, moreover it suggests that a level of permanent damage to the lung has already occurred (WHO 2007). The formation of radiographically visible nodules indicates that clearance of the silica particles has stopped and the progression of the disease may be independent of further exposure (ATS 1997; Hitzdo and Sluis-Cremer 1993; Muir et al. 1989; Tran et al. 2005; Rimal et al. 2005).

As Tran et al. (2005) observed (p 19) "...Silicosis is a progressive damage process, and there is no clear diagnostic point past which disease is discriminated from absence of disease, except by arbitrary convention..."
Qualitative Environmental risk

Safa and Machado (2003) stated “... A hazard to health should always be suspected whenever dust containing free silica is likely to be liberated into the air…”

The literature search conducted for this paper was unsuccessful at locating papers where community exposures or disease incidence at peak sites were examined using accepted epidemiological methodologies.

There appears to be a general consensus amongst researchers that at ambient levels there is little risk of silicosis in the general population (OEHHA 2005), whilst at the occupational level there is an extreme level of risk and recorded mortality and morbidity (for example NIOSH 2002, Rosner and Markowitz 2006, WHO 2000). OEHHA 2005 note, however, that silicosis is not the subject of medical attention in the general population, and the possibility that it does occur cannot entirely be dismissed.

The question of non-occupational risk associated with communities living adjacent to peak sites requires examination. Community exposures to respirable crystalline silica (including freshly fractured, iron contaminated crystalline silica) in the near field of a quarry (for example) are likely to lie between ambient concentration and occupational concentrations in the ambient air.

While epidemiological studies of exposed communities are not present in the literature there are some limited references that provide qualitative insight to the level of environmental risk including:

- Epidemiological and animal inhalation studies which demonstrate that the risk of silicosis is related to both concentration and duration of exposure (Steenland and Brown 1995, Steenland 2005, Tran et al. 2005, NIOSH 2002, RSA 2006);
- Identification of non-occupational silicosis due to elevated ambient exposures to silica particulate (Bar-Ziv and Goldsmith 1974; Sayed et al. 1991; Safa and Machado 2003; Nouh 1989; Guo et al. 1997);
- Epidemiological and other studies that may be used to develop benchmark doses for silica exposure (Hnizdo and Sluis-Cremer 1993, OEHHA 2005);
• Investigations into the vertical and downwind extent of plumes from peak anthropogenic sources of respirable crystalline silica (Holmen and Shiraki 2001); and
• Studies which identify silicosis in exposed animals (pigs and water buffalo) downwind of peak sites (Roperto et al. 1994, Roperto et al. 1995).

Of particular interest are the studies of silica disease in farm animals downwind of peak sources of respirable crystalline silica. These studies demonstrate that environmental exposures near peak sites could result in silicosis. Roperto et al. (1994) states (p233):

"...Thus, domestic animals living in an environment in which there is daily exposure to significant concentrations of environmental inorganic dust could be used as an important biological model for similar conditions in man..."

Roperto et al. (1994) examined lung and tracheobronchial lymph tissue samples from each of four one-year old pigs that were raised near two cement-works and several chalk quarries. Analysis of the tissue samples confirmed that each of these one-year old pigs had silicate pneumoconiosis. Additionally, Roperto et al. (1995) investigated the case of two aged water buffalo which were suffering from silico-tuberculosis and were raised on a farm with high particulate pollution due to the proximity of a quartz quarry nearby. Tests of tissue samples confirmed the presence of silicosis and silica fibrosis in these animals.

The study by Holmen and Shiraki (2001) examined particulate concentrations downwind from a sand and gravel plant, including LIDAR measurement of plume height and downwind extent as well as particulate monitoring including analysis for crystalline silica. The technique used included collection of particulate on a cellulose substrate and subsequent laboratory analysis using scanning electron microscopy and X-ray diffraction. The findings of this study were that crystalline silica concentrations were significantly elevated downwind of the site as were both PM$_{10}$ and PM$_{2.5}$.

It is acknowledged that plume propagation and dispersion is site specific and therefore these results should be regarded as illustrative of the potential for elevated concentrations of crystalline silica downwind of an industrial source of these particles. Holmen and Shiraki 1991, confirm that crystalline silica in the respirable range downwind of a peak source can be significantly elevated above ambient concentrations.
The atmospheric residence time of fine particles in the lower troposphere can be three to four days (Husar 2003) and may travel several tens of kilometres downwind. The particles tend to be removed by wet deposition. Additionally, crystalline silica particles tend to be inert and can re-suspend following deposition (Husar 2003).

The available evidence suggests that it is possible that environmental exposures could lead to silica related disease in exposed communities near peak sites.

**Selection of an appropriate environmental risk endpoint**

As reported by NHMRC (2006) (p7): "...air pollution has the potential to affect everyone in the community, and that individuals cannot readily control the extent to which they may be exposed to air-borne pollutants, there is a reliance on governments to ensure that appropriate levels of public health protection are enacted through air quality standards..."

Whilst there is sufficient evidence to support the classification of crystalline silica as a human carcinogen (ATS 1997, Guidotti and Koehncke 1998), the available data do not provide a definitive dose response relationship for lung cancer in the absence of silicosis (Pelucchi et al. 2006). The critical factor in the onset of the silica disease appears to be cessation of clearance of silica particles and the onset of inflammation (Tran 2005). This inflammation culminates in the development of nodules which characterise the diagnosis of silicosis (Rao et al. 2004).

The objective of an environmental health based end point should be the prevention of radiographically diagnosed fibrotic nodules in the general population as this is the point which would protect community members from silica related disease. Therefore this objective was selected as the most appropriate endpoint for the quantitative risk assessment for environmental exposure.

**Setting the Standard: Quantitative environmental risk**

WHO (2007) stated “…it should be kept in mind that when silicosis is detected by a chest X-ray, it is already too late; that lung will never be normal again...”

A number of papers produced by Government agencies identify that while silicosis is a preventable disease, once contracted it is an incurable one (WHO 2007, NIOSH 2002, RSA...
Activities undertaken by Government agencies are focused on establishing strategies to prevent exposure to crystalline silica particles (NIOSH 2002).

OEHHA (2005) report that the quantitative data associated with the dose response relationship for silicosis risk is sufficient to develop an ambient air quality standard for respirable crystalline silica that will protect the general population. Of particular relevance are the studies that enable determination of silica doses that would prevent the development of silicotic nodules in the lung (ibid).

A health based environmental exposure standard should incorporate protection of sensitive subgroups within the human population (such as children) (WHO 2006, NHMRC 2006, DEH 2007). WHO 2006 state: "... new data and methodologies have emerged, indicating that children are a vulnerable population subgroup with special susceptibilities and unique exposures to environmental factors that have important implications for public health practices and risk assessment approaches...." NHMRC (2006) provides further support for the inclusion of children as a specific subgroup for protection within an ambient air quality standard, stating (p45): "...For example children may be considered a sensitive sub-population because any irreversible effects may influence their health throughout their entire life..." Furthermore, a review of ambient air quality and children's health conducted by the DEH (2007) identified that infants and children inhale and retain larger amounts of air pollutants than adults, have developing immune systems and therefore cannot detoxify and excrete toxins as well as do adults.

The approach of developing an ambient air quality standard for crystalline silica that prevents the development of silicotic nodules in the general population is consistent with the approach adopted by the USEPA for non-cancer health endpoints (Castrorina and Woodruff 2003). This approach is based on the development of reference doses (RfD) and reference concentrations (RfC) (USEPA 1995). These are defined as "an estimate of daily or continuous exposure to the human population (including sensitive subgroups) that is likely to be without appreciable risk of deleterious risks during a lifetime..." (Castrorina and Woodruff 2003 p 1318).

The USEPA has developed a Benchmark Dose technique for fitting a model to the available dose response data. The Benchmark Dose is the dose of a compound that corresponds to a specified level of response (for example the silica BMC05 corresponds to the lower bound
estimate of a dose in which five percent of the population would develop silicosis). This technique is regarded as being more robust than the NOAEL approach and provides superior assessment of the actual risks pertaining to exposures (USEPA 2000b, Castrorina and Woodruff 2003).

The OEHHA (2005) applied the Benchmark Dose technique to available occupational studies into silicosis using the $BMC_{01}$ as the appropriate response level. The $BMC_{01}$ levels were corrected for continuous environmental exposure against occupational exposure to develop an equivalent exposure which was subsequently corrected for intrahuman uncertainty ($UF_h$). The results reported by OEHHA 2005 are summarised in Table 3. The Environmental Equivalent Concentration (EEC) is calculated by:

$$EEC = BMC_{01} \times \left( \frac{RV_{oc}}{RV_{day}} \times \frac{OCC_{yr}}{Yr} \right) + t$$

Where:

- $RV_{oc}$ is the Respiratory Volume during the work period (assumed to be $10m^3$)
- $RV_{day}$ is the Respiratory Volume during the day (assumed to be $20m^3$)
- $OCC_{yr}$ is the number of days per annum spent at work (assumed to be 270 days)
- $Yr$ is the number of days in the year (i.e. 365).
- $t$ is the duration (in years) of exposure within each study

The Reference Exposure Level (REL) is calculated by:

$$REL = \frac{EEC}{UF_h}$$

Where:

- $EEC$ is the Environmental Equivalent Concentration (above); and
- $UF_h$ is the intrahuman uncertainty factor to protect sensitive individuals within the general population (OEHHA 2005 p34).
Table 4: Reference Exposure Levels (OEHHA 2005)

<table>
<thead>
<tr>
<th>Study</th>
<th>$\text{BMC}_{01}$ (mg/m$^3$/yr)</th>
<th>Exposure Duration (yrs)</th>
<th>Environmental Equivalent Concentration (µg/m$^3$)</th>
<th>Cumulative Uncertainty Factor (UFH)</th>
<th>Reference Exposure Level (µg/m$^3$)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hnzdido and Sluis Cremer (1993)</td>
<td>0.636</td>
<td>24</td>
<td>9.8</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>Steenland and Brown 1995</td>
<td>0.34</td>
<td>9</td>
<td>12.4</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>Hughes et al. 1998</td>
<td>N/A</td>
<td>11.5</td>
<td>29 ($^a$)</td>
<td>10</td>
<td>3</td>
</tr>
<tr>
<td>Chen et al. 2001</td>
<td>0.132</td>
<td>2.2</td>
<td>18</td>
<td>3</td>
<td>6</td>
</tr>
<tr>
<td>Churchyard et al. 2004</td>
<td>0.673 ($^b$)</td>
<td>21.8</td>
<td>11.4</td>
<td>3</td>
<td>4</td>
</tr>
</tbody>
</table>

*a: Calculated from Lowest Observed Adverse Effect Level  
*b: reported as BMC$_{05}$

The OEHHA study resulted in the recommendation of 3µg/m$^3$ as an inhalation reference level for ambient exposure to respirable crystalline silica (OEHHA 2005). This is broadly consistent with a recent protocol developed by the Victorian EPA for a PM$_{2.5}$ 3µg/m$^3$ limit for crystalline silica in the environment surrounding mines and extractive industries (Vic EPA 2008).

Conclusions
Silica is abundant within the earth's crust and is released as a fine particulate whenever the rock is disturbed. Furthermore silica is an important industrial mineral with broad applications in manufacturing processes.

There is a causal link between exposure to respirable crystalline silica and a number of human diseases (including cancer and lung disease). The risks associated with silica exposure are...
related to cumulative dose and the onset of the disease may occur several years after the exposure has ended.

Although the risk of silicosis is regarded as negligible in the ambient environment, cases of non-occupational silicosis are recorded within the literature. The risk of silica related disease is a function of the cumulative dose, and environmental exposures to respirable crystalline silica can be significantly elevated downwind of industrial sources including industry, quarries and sand mining. Increasing the concentration of respirable particles containing crystalline silica near industrial sources increases the level of risk of silica disease in exposed populations.

The current occupational exposure levels are shown to result in morbidity and mortality in workers and do not offer protection to communities (and associated sensitive sub-groups) exposed near peak sites.

The risk associated with peak sites is exacerbated in situations where the community (including sensitive sub-groups) would be exposed to freshly fractured silica particles especially in the presence of iron. A typical example of this situation would be downwind of a quarry where rock is blasted and crushed.

A limit of 3µg/m$^3$ (respirable crystalline silica) would protect communities, who are potentially exposed, in the vicinity of these peak sources. This would be a total exposure limit and include background ambient levels, and the cumulative exposure due to all industrial sources near the affected community.

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