CEMENT CONCRETE & AGGREGATES AUSTRALIA

2 December 2005

Mr Elton Humphrey
Secretary
Australian Senate Community Affairs References Committee
Parliament House
CANBERRA ACT 2600
Email: community.affairs.sen@aph.gov.au

Dear Mr Humphrey

INQUIRY INTO WORKPLACE EXPOSURE TO TOXIC DUST

At the 30 September 2005 public hearings of the Senate Community Affairs References Committee Inquiry into Workplace Exposure to Toxic Dust, our organisation was invited to provide further information on a number of points relating to our submission and those submitted by other parties.

We are pleased to have the opportunity to do this and trust that this information will be taken into account by the Committee.

We note that there are a number of errors of fact contained in a number of the submissions, particularly the verbal submissions provided during the Public Hearings. We do not intend to comment on them all but rather highlight those that may be relevant to the outcomes of the Inquiry.

As a general comment we would strongly urge the Committee to rely on supported facts rather than the unqualified and unsupported opinions of those providing evidence.

There are three matters on which we would like to provide comment as follows:

1. AIOH Submission

We note that in the verbal component of the AIOH submission presented on 29 September in Melbourne, Mr Jennings stated that the NOHSC Occupational Exposure Standard for Respirable Crystalline Silica (RCS) of 0.1 mg/m3 which was introduced on 1 January 2005 was introduced on the basis of preventing lung cancer.

Mr Jennings further suggested that at exposure at the current standard levels of 0.1mg/m3 there was in his view a likelihood that there would be further incidence of silicosis and that a further reduction in the OES was warranted to prevent this from occurring.

This is not correct. Reference to NOHSC standard will indicate that the OES was specifically introduced to prevent the incidence of Silicosis. We would refer the committee back to the AIOH submission which on pp11-12 states as follows:

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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 0.2mg/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.

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.

Mr Jennings went on to advise the Committee that he was of the view that a further reduction in the standard to prevent silicosis was warranted. This appears contrary to the written submission and further, we have been made aware that AIOH have written to the Secretary of the Committee distancing itself from Mr Jennings opinions on this matter and explicitly stating that AIOH support the standard at its current level.

We would ask that the Committee consider the views of AIOH as described in its submission rather than any unsupported personal views.

2. The Relationship between Exposure and the Incidence of Silicosis.

The Committee will recall that a number of submissions to the Inquiry indicated that silicosis had a long period of latency. This was the subject of some questioning of our Dr John Bisby by Sen. Allison on the wording of part of the NSW Dust Diseases Board submission.

There was an apparent implication drawn from the NSW Dust Diseases Board(DDB) submission that the **onset** of silicosis can be from 2 - 40 years after (cessation of) exposure.

We believe that confusion has arisen from ambiguity in the wording of the DDB written submission around the concept of latency and levels of exposure.

The concept of latency is useful in epidemiology, but can only be fully understood if several other concepts are also included, such as the concept of cumulative exposure being causative. In the individual, one can never know if one "time of exposure" or another time period "caused" the disease.

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The DDB submission states:

Silicosis:

Silicosis is caused by exposure to crystalline silica dust. There are three types of silica, quartz, tridymite and cristobalite. Silica is deposited in the air sacs resulting in fibrosis of the lung. Symptoms include breathlessness and a dry cough. There are three types of silicosis:

1. *Chronic - this is the most common form occurring over 20-40 years after long-term exposure to low and moderate levels;*
2. *Accelerated - occurring 5-10 years following high exposure levels;*
3. *Acute silicosis - occurring up to 2 years after short-term high exposure levels. It is often fatal, sometimes within a few months.*

Comment

This section followed a section in the DDB submission dealing with Asbestos, which used the term "latency" – particularly in relation to the asbestos cancer mesothelioma.

The term "latency" was not used by the DDB in relation to silica and silicosis. The ambiguity in the use of the word "after" in types 1 and 3 in the DDB submission seems to have caused some confusion in the hearings. The wording has been interpreted *as if referring to latency* whereas it actually refers to period of exposure. The two concepts are quite different:

- one dealing with the necessary length of time (usually years) of exposure which leads to the disease, this being the "exposure period" - this can be 2-40 years of exposure before silicosis occurs and is diagnosed,
- the other dealing with the average length of time (years) between the cessation of the exposure and the date of diagnosis - the "latency period".

When discussing a disease, eg. sinus cancer in woodworkers, both concepts are based on epidemiological estimates from groups of workers who have been diagnosed. They are thus normally expressed as average times, usually expressed in years and often given as a range eg:

Nasal sinus cancer in woodworkers has a latency period of 5-20 years.

However, when reporting an individual case, the exposure period and the latency may be expressed by calculating from the individual's history eg. in the case of a man presenting with sinus cancer who has been a woodworker:

- *Mr A developed the disease following x years of exposure (to the causative agent). His exposure period was x years.*
- *Mr B developed the cancer y years after exposure ceased (thus the latency period was y years). Mr B would also have an exposure period, in this case x years.*

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In the Senate Inquiry, the reference was to the disease of Silicosis. Thus the wording regarding silica might have been better explained in the DDB submission as:

1. Chronic - this is the most common form, following 20-40 years of long-term exposure to low and moderate levels;

2. Accelerated - occurring after only 5-10 years of exposure to high levels;

3. Acute silicosis - occurring after only 2 years or less of high exposure levels. It is often fatal, sometimes within a few months.

The evidence regarding latency in chronic silicosis is that latency 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).

However, this delayed appearance or latency is rare. **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.

Silicosis does not have a long latency period, comparable say with mesothelioma or some other occupational cancers. Almost no workers whose X-ray is classed as “no opacities” when they cease exposure, will subsequently develop opacities (with or without any signs of silicosis) in later years. Latency is not a major issue in relation to silicosis, and there will not be a wave of hidden cases occurring years ahead. The few who do will develop those opacities within a short time of ceasing work.

The concept of latency in Accelerated and Acute silicosis is not applicable, as these conditions almost always occur in workers with ongoing exposure, and result in clinical and obvious disease.

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 recognized 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.

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Every person has some silica present in their lungs, because silica dust is ubiquitous. Thus any examination of lung tissue from any person (a common diagnostic procedure in any severe lung disease) may reveal presence of silica. This is not evidence that the disease is silicosis, nor that silica dust has contributed to any clinical disease. In particular persons with smoking-caused disease are more prone to retaining any dust from any source in their lungs due to impaired clearance mechanisms.

3. Relationship between Silica Exposure and Lung Cancer

In 1996, in a controversial decision crystalline silica (inhaled in the form of quartz or Cristobalite from occupational sources) was classified by The International Agency for Research on Cancer (IARC) as carcinogenic to humans (Group 1).

However that original decision has been disputed by members of that original IARC panel since that time (McDonald reference pdf file). In addition, the IARC panel only considered epidemiological evidence up to 1994 and the more current research on workers eg. in the UK sand industry indicates no excess risk of lung cancer or other cancers. Recent reviews of the evidence eg by Patrick Hessel for the American Chemical Society confirm that (quote):

"Considered as a whole, 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." (end quote)

Being forwarded separately to this supplementary submission by facsimile on today's date is a copy of the UK sand industry research, carried out by the Medical Research Council of the UK (OEM 2005 paper). That 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).

In closing we would like to restate a number of facts regarding the management of Silicosis in the cement, concrete and quarries industries. We are strongly of the view that the cement concrete and quarries industries are well aware of the risks and consequences of overexposure to RCS and members have taken and continue to take their obligations to provide a safe and healthy workplace very seriously. This is evidenced by the very low level of incidence of silica related disease in our industry.

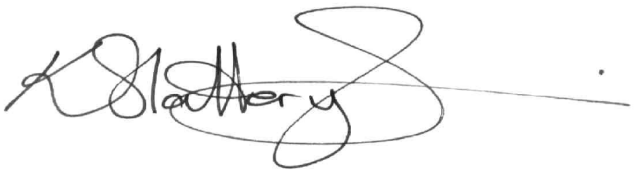
The Senate Committee has heard views expressed that in some sectors of industry, risks may still remain. We submit that silicosis really only arises from exposures above the nationally accepted and adopted occupational exposure standards. In uncontrolled environments, such as may have been seen in the sand blasting industry when silica sand was used as an abrasive medium, exposures several hundred times greater than the occupational exposures may well have been encountered and may have lead to debilitating disease. But in well managed and controlled work environments, such as those operated by our industry, the risks are effectively managed with the results speaking for themselves.

…/6

We remain strongly of the view that any further reduction in the exposure standard for RCS below the current level of 0.1mg/m3 would not be productive as the incidence of the disease in our industries is already very low, even without the full impact of the most recent change in standards at the beginning of this year having been realised.

Again we would like to thank the Committee for the opportunity to provide further input to this Inquiry. As always we would be pleased to provide whatever further information that the Committee sees relevant.

Yours sincerely



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