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21 July, 2005

Mr Elton Humphery
Secretary
References Committee
Australian Senate
Parliament House
CANBERRA ACT 2600

Dear Mr Humphery

I am an academic respiratory physician presently appointed as Clinical Director, Department of Allergy, Immunology and Respiratory Medicine, The Alfred Hospital, Melbourne, 3004, and Clinical Associate Professor, Department of Medicine, Monash University. My main clinical interest is lung transplantation which has afforded me considerable experience and expertise in assessing patients with severe lung diseases. I am also a treating physician of Mr Richard White who has been central in the attempt to encourage the Australian senate to hold their enquiry into workplace exposure to toxic dust. I will make my comments under the specific headings of the terms of reference.

(a) *the health impacts of workplace exposure to toxic dust including exposure to silica in sandblasting and other occupations;"*

It has been known for over 100 years that inhalation of silica dust can cause serious lung disease. With the implementation of occupational health and safety controls as well as surveillance in "at risk" workplaces there has been a substantial reductions in classic silicosis. It has become apparent (somewhat parallelling what has been seen with asbestos exposure) that a new pattern of disease is emerging. People having previous silica exposure are presenting with diseases include obstructive lung diseases such as bronchitis, bronchiolitis and emphysema as well as the development of pulmonary fibrosis with a long lag-time from exposure to the development of lung disease. Also of great concern is the propensity for dust such as silica to increase the risk of the development of lung cancer and stomach cancer.

Classic silicosis is well known by most medical practitioners (indeed an Ovid search reveals 13,337 citations predominately dealing with this topic). There is far less information concerning the risk of COPD and emphysema as a complication of silica exposure (172 citations on Ovid search) or cancer risk (77 citations on Ovid search). I believe we are indebted to the work of Dr. Andrew Churg from Canada who in animal and human studies has shown a link and potential mechanisms by which

inhalation of silica causes bronchitis and emphysema. These effects seem to be synergistic (not just additional) to the effects of smoking. Almost none of the literature is reflective of the extent of injury due to silica dust exposure in Australians within Australia.

I am also concerned that many patients with so called idiopathic pulmonary fibrosis may have the genesis of their disease in exposure to fine dust such as silica and that causal link is not made because of a long delay from exposure to overt disease.

I don't believe we have sufficient information to even start to understand the extent of these problems in Australia and well designed studies are urgently needed.

(b) *"the adequacy and timeliness of regulation governing workplace exposure, safety precautions and the effectiveness of techniques used to assess airborne dust concentrations and toxicit:"*

I have no comments.

(c) *"the extent to which employers and employees are informed of the risk of workplace dust inhalation:"*

I think that with the reduction in incidence of classic silicosis coupled with a lack of focus on other potential health risks of exposure to fine dust such as silica may have led to a degree of complacency by both employers and employees. Nevertheless we have known that silica is a dangerous dust for more than a century.

(d) *"the availability of accurate diagnoses and medical services for those affected and the financial and social burden of such conditions:"*

As mentioned previously, classic silicosis is likely to be accurately diagnosed, however other consequences of silica exposure such as small airways disease, emphysema, stomach and lung cancer may be attributed to other causes such as asthma or exposure to cigarette smoke when in fact the predominate cause may be silica dust. Patients may also be erroneously diagnosed as idiopathic pulmonary fibrosis when their lung disease is due to fine particle dust exposure such as silica.

(e) *"the availability of accurate record on the nature and extent of illness, disability and death, diagnosis, morbidity and treatment:"*

It would appear to me that this needs a great deal more research for us to truly understand the extent of diseases caused by fine dust exposure such as silica in Australia.

(f) *"access to compensation, limitations in seeking legal redress and alternative models of financial support for affected individuals and their families:"*

The Richard White case I believe shows that the present model is not meeting the prime objectives, that is sanctioning those that allow their employees to work in a

hazardous environment and financially compensating individuals affected. From my observation is an expert witness in the case (but with no legal training) neither seems to have occurred in the White case. The complex and changing corporate entities, the inequity of legal resources, the time delay between exposure and symptoms, and an assumption (supported by plaintiff medical witnesses) that even a trivial smoking exposure was the cause Mr. White's lung disease, all appear significant factors in this.

(g) *"the potential of emerging technologies, including nanoparticles, to result in workplace related harm:"*

I have no comment.

Thank you for the opportunity to provide written submission.

Yours sincerely,

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