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Discipline of Medicine

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

Dear Mr Humphery

Re: Inquiry into workplace exposure to toxic dust

Thank you for the opportunity of making a submission to this enquiry. I have a major professional and research interest in pathogenic processes within the lungs and the airways, and the etiology of both bronchial asthma and chronic obstructive disease of the airways. My particular interest in this issue arises from my previous professional interactions with Mr Richard White who, I believe, was the sentinel case which brought this area of interest to the attention of the Senate Committee. I will refer to that case in brief below.

In particular, I would like to make some comments pertinent to Term of Reference (a) the health impacts of workplace exposure to toxic dust including exposure to silica in sandblasting and other occupations and Reference (f) access to compensation, limitations in seeking legal redress etc.

I am currently Professor of Medicine at the University of Tasmania, with a strong research interest in respiratory disease. Between 1992 and 2001, I was Director of Respiratory Medicine at The Alfred Hospital in Melbourne, and foundation Professor of Respiratory Medicine at Monash University. It was in that role in 1999 that I had the opportunity to examine and write a report on Mr Richard White. Between 1985 and 1992, I was Senior Lecturer at the University of Newcastle-upon-Tyne in the UK and Associate Director of the North of England Occupational Lung Service where I became very familiar with a wide variety of occupational airway and lung diseases in an area of heavy industrial work, including asbestos-related disease from the ship building industry as well as traditional coal workers' diseases from the mining industry. There are also many more modern causes of occupational asthma in a number of settings. As an Advanced Trainee in Cardiff, South Wales between 1979 and 1984, I was responsible for running a 36 bed ward dedicated to pneumoconiosis from the Welsh mining and slate industries. I have over 250 research publications, many of them related to effects on the airways of environmental irritants and chemical toxins.

The case of Mr Richard White

Mr White has let it be known to me that he is prepared to have his case discussed publicly as a paradigm example of some of the difficulties currently faced by workers who may have been damaged by occupational lung exposures, in seeking adequate redress. As a late teenager between the ages of approximately 15 and 18, Mr White had substantial exposures to very fine silica dust in enclosed spaces while using sandblasting methods to clean the inside of barges, fuel tanks and other tanks while a teenager in Darwin Northern Territory. He was initially doing this work on a part time basis after school but for the last two years of employment he did this essentially full time. His exposures were made complex by the fact that he also sprayed the inside of tanks after cleaning them with an epoxy resin material. For this work he was essentially unprotected in terms of any sophisticated respiratory breathing apparatus or even a substantial mask.

From the age of approximately 30 Mr White experienced quite marked respiratory symptoms in the form of cough and sputum production and a great deal of irritancy in his airways limiting his ability to take good breaths because of paroxysms of cough which they would engender. During his 30's he gradually became more breathless so that by his early 40's a diagnosis of obstructive airway disease was made. The physiological components on formal lung function testing were somewhat complicated and have been open to a degree of debate, but essentially showed mainly fixed airflow obstruction particularly affecting his smaller airways. In the year 2000 he had a lung biopsy performed under general anaesthetic in an open procedure at The Alfred Hospital, Melbourne, because of concerns that he might need a lung transplant because of the rate of progression of his illness. The biopsy obtained showed a number features, but the most salient included evident silica particles within lung macrophages, and a degree of mild pulmonary fibrosis in relation to these, suggesting early silicosis. There was also a degree of related emphysema (emphysema is a condition in which the lung tissue itself breaks down to create holes within the lung tissue).

Mr White also smoked cigarettes but only relatively mildly to an extent of 5 – 10 cigarettes per day between the ages of approximately 17 and 37. It was this aspect of his background that ultimately seemed to make his case fail at law, on the basis that his disease was self-inflicted and related to the smoking habit. My opinion, and shared by some but not other medical experts called on the case, was that the illness was atypical for smoking-related disease in a number of ways: especially the early onset and the degree of cough and irritability of the airways experienced by Mr White. In addition, the biopsy proved the presence of silica within the lung and a degree of fibrosis of the lung seemed also to me to be very highly pertinent. My opinion was that intense exposure to silica in the context of a young man whose lungs and airways were still developing at the age of exposure had lead to an occupational respiratory disease particularly focused upon his smaller airways. It seemed to me that on the balance of probabilities this very unusual acute degree of exposure at a young age could not be written off as merely coincidental.

It seems to me that Mr White was unfortunate in that the court took the opinion that since Mr White had been a smoker all airway disease must therefore be related to the smoking and that the silica was merely coincidental and couldn't either have produced an additive effect to the smoking or a coincidental independent one. At the time that Mr White was taking legal proceedings, research and medical clinical opinions were already beginning to harden in the direction of accepting that silica and other toxic dust exposures may be having a significant effect upon workers' airways, but this was obviously not sufficiently developed at that stage to persuade the legal system or indeed some of the medical practitioners who gave evidence, that this was the case. However, things have developed and moved on since the late 1990's and I would like to make reference and make available to the Committee two recent documents which are highly relevant and to which I refer below.

Traditional Concepts on Dust Related Lung Disease

It seems to me, that Mr White suffered from legal and medical preconceptions that 1) airway disease is either classic "asthma" i.e. an episodic and reversible disease of the airways mainly due to bronchospasm and airway inflammation, or chronic obstructive pulmonary disease (bronchitis and emphysema) caused by cigarette smoking; and 2) dust diseases cause lung parenchymal fibrosis giving rise to classic co-workers pneumoconiosis or silicosis as paradigm illnesses and not airway disease. 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.

In recent epidemiological studies in Melbourne, in which I have been a senior investigator, we have surveyed middle age and elderly populations and shown that "only" approximately two thirds of chronic obstructive pulmonary disease (COPD) can be attributed to cigarette smoking, rather against the general assumption it would all be due to cigarette smoking. Peer-reviewed papers containing these data will be published later this year in Thorax (the journal of the British Thoracic Society) and the Internal Medicine Journal (the journal of the Australian and New Zealand College of Physicians), and I would be happy to provide copies of these papers to you either in pre-print form or hopefully when they are formally published shortly. What we also found was that exposure to organic dusts in this population was a significant cause of obstructive lung disease in non-smokers.

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 that in relevant populations that this would not also be potentially of importance.

Current Concepts of Mineral Dusts and Chronic Obstructive Airway Disease (COPD)

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 indeed might be more evident in smokers. The literature has recently been reviewed in detail by the Institute of Environment and Health under the auspice of the British Medical Research Council. I have a copy of this document which has been kindly provided by colleagues in the UK, although it was not prepared for general circulation. However I think the literature review might be of great use to the Committee. In addition, there is another British document prepared by the National Institute for Occupational Safety and Health. and published in 2002 which also comes to the conclusion that silica is one of a number of occupational dusts which do cause airway disease leading to fixed airflow obstruction.

The Committee will undoubtedly become aware, if not already, that after a well publicized court case in the UK in 1999 that British miners were recognized 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 has been spending a great deal of money in a very large exercise to assess miners and ex-miners and to provide substantial amounts of compensation. 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. I think this is of great relevance to the case of Mr White, but probably also to many other Australian workers with premature ageing of the lungs and development of fixed airflow obstruction at relatively young ages who may or may not be smokers but in whom the degree of disability would be more than can be accounted for by their smoking behaviour.

As in the UK, I think it is encumbent upon the Australian community, especially given the substantial contribution of the mining industry to its wellbeing and tax base, to take this issue seriously, and to formulate laws and regulations which are sympathetic to mining and quarrying workers. Those workers who have been exposed to dusts and fumes and where there is significant evidence of damage to the airways in excess of what would be expected from smoking, should be compensated. This also means that much more attention should be paid in Australia to performing quality lung function tests, especially spirometry, on vulnerable groups including workers in dusty industries. This should be performed on a regular basis, so that they get picked up early if they begin to develop COPD.

Yours sincerely

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