

## ***Health Effects of Coal Dust in a Non-Occupational Context : Literature Review***

### **Introduction**

The transport, unloading, stockpiling and handling of coal in large quantities is conducted at Te Awaparahi Bay, Lyttelton, prior to loading of the coal onto ships moored at the nearby Cashin Quay berths for export to buyer countries.

Lyttelton Port Company (LPC) proposes to significantly increase the size of the stockpile area and associated facilities, to accommodate the extra quantities of coal expected to arise from new mine developments on the West Coast. The intention is to possibly double the annual tonnage of coal currently stockpiled and loaded at Lyttelton.

Whenever coal is stockpiled and, particularly, when it is transported, dumped or otherwise handled, there is the potential for release of small particles (i.e. dust) of coal in significant quantities. The range of sizes of these particles is considerable; visible coal dust (i.e. as a black cloud) is one extreme of the possible range of particle dimensions but within any such dust cloud there will be a proportion of particles which have dimensions of 20  $\mu\text{m}$  or less; this is generally referred to as Total Suspended Particulate (TSP). Further, within the TSP fraction of total airborne dust, there will be a proportion of particles which are of respirable size. While there is no precise cut-off point which delineates whether a particle is respirable or not (particle shape and associated aerodynamic factors are also relevant) the dimensional arbiter of particle respirability is taken to be 10  $\mu\text{m}$  or less (the so-called  $\text{PM}_{10}$  fraction).

The particle size distribution of this respirable fraction includes a proportion of particles less than 2.5  $\mu\text{m}$  in diameter (referred to as  $\text{PM}_{2.5}$ ). While all sub- $\text{PM}_{10}$  particles are considered respirable, those less than 2.5  $\mu\text{m}$  in diameter are likely to penetrate further into the lung and are generally considered therefore to present a greater risk to health.

Factors associated with the extent of coal dust release wherever coal is mined, transported, stockpiled, handled or loaded include wind speed, moisture content (especially of the stockpile surface) and the extent of mechanical handling of the coal. The latter factor contributes to abrasion of the coal material and thus to the generation of dust.

Consultation and inspection with local residents indicates that coal dust reaches some of the residential properties around the area of the Timeball Station. This has resulted in the deposition of visible coal dust on flat surfaces such as window sills. As noted earlier, wherever there are gross effects from visible coal dust deposition it is probable that there is at least some exposure to much finer dust particles, and some of these particles will be of respirable size. It is important therefore that the possible health effects of respirable coal dust at the concentrations likely to be experienced in a non-occupational context, as for residents living nearest to Te Awaparahi Bay, be investigated and this is the purpose of the following literature review.

## **Setting the Context**

There is a considerable body of international literature about the health effects of exposure to coal dust of respirable particle size (i.e. PM<sub>10</sub> and smaller). In almost every case the studies and associated reported health effects relate to coal miners and coal mine sites, either underground or open-cast. In many cases the actual extent of exposure, usually expressed as time in years but also on occasions as total body burden of inhaled dust, has been established. In turn, this has been used to draw conclusions on the dose-response relationship for the onset of adverse health effects from respirable coal dust exposure.

It is noteworthy, and of great importance to the consideration of non-occupational exposure, that symptoms of ill-health in coal miners are typically observable only after very lengthy exposure to high concentrations of coal dust of respirable particle size; in short it is (older) coal miners who are affected. Coal miners have typically been relatively grossly exposed to dusty conditions, particularly in the past.

## **Health Effects of Exposure to Coal Dust at Occupationally Encountered Levels**

A comprehensive recent review<sup>1</sup> gives an excellent overview of the conclusions of some 93 individual papers and research reports on the general subject of the health effects of inhalable coal dust.

While major improvements to mine working conditions with respect to coal dust have been made in the last four decades the typical levels of exposure of miners (say, 1-5 mg/m<sup>3</sup> respirable dust) are still relatively very high when compared to environmental exposure levels. Thus assessment of health effects occurring at levels of dust exposure encountered in mines, and keeping in mind the length of time before significant effects develop, can set parameters against which to judge the likely health risks posed by environmental exposure to coal dust.

The adverse effects on health of significant occupational exposure to coal dust have been well known for many years and have been well characterised in recent decades<sup>1</sup>. Thus coal workers' pneumoconiosis (CWP) is a characteristic occupational disease involving chronic irritation of the lung resulting from coal dust inhalation. Associated with CWP, but also appearing in its absence, are conditions such as emphysema and chronic bronchitis which are also clearly linked, in terms of severity, with increasing exposure duration and/or airborne coal dust concentrations.

In addition, in about 1-2% of exposed persons with simple dust accumulation (simple CWP) solid, black masses develop which represent accumulations of coal dust in connective tissue. This condition is known as progressive massive fibrosis (PMF).

Many papers<sup>2-6</sup> have appeared in the literature in which correlations are made between extent of coal dust exposure (duration, concentration, coal type and age of miners) and onset of simple CWP and/or PMF. Thus in Britain<sup>2</sup> estimates of exposure based on 20 years of observations at 10 collieries showed that those miners with similar cumulative dust exposure but with longer exposure time had a higher prevalence of CWP. Those workers with simple CWP did not necessarily have significant clinical abnormalities such as impaired lung function but the risk of developing

complications such as PMF was higher for those miners showing clinical symptoms and thus with more advanced extents of simple CWP.

Models developed on the basis of a 1992 American study<sup>5</sup> indicate that between 2% and 12% of miners exposed to a constant dust burden of 2mg/m<sup>3</sup> in a bituminous coal mine would be expected to have simple CWP after a 40 year working life.

The crushing of coal with a component of quartz will generate dust containing silica. Long term inhalation of silica, typically after exposures in excess of 30 years and to levels of silica dust in the air within coal mines of 10% of the total dust burden or greater, may lead to the formation of scar tissue in the lungs and the development of a serious and irreversible condition known as silicosis. It is relevant to note that silica is present in New Zealand coals at levels of up to 2% as a maximum although, for most New Zealand coals, the proportion of silica is considerably lower, being around 0.1%. This is a very low silica content when compared to other world coal types.

It is universally acknowledged that silicosis is solely a workplace health issue. A recent Australian Government Senate Committee report<sup>7</sup> identified that there are no reports in the international literature of individuals developing silicosis as a result of exposure to non-occupational levels of silica dust. A United States Environment Protection Agency study<sup>8</sup>, which specifically addressed this issue, confirms this view.

It is clear from the above that the limited silica content of the coal being handled at Lyttelton is of negligible relevance to environmental health considerations with respect to both coal stockpile workers and also to neighbouring residents.

While studies<sup>8-10</sup> have shown that exposure to coal dust can result in emphysema and/or chronic bronchitis in coal miners it is noteworthy that these same investigations have shown that cigarette smoking is far more harmful than exposure to coal dust (at least under current working conditions in Britain) in causing either of these conditions.

Physical indicators of the onset of asthmatic symptoms, including forced vital capacity (FVC) and forced expiratory volume in 1 second (FEV<sub>1</sub>) have been assessed in a large sample of United States coal workers<sup>11</sup>. Consistent annual decreases in these lung function indicators were observed as cumulative exposure time to coal dust lengthened. The authors concluded that exposure to respirable coal dust at concentrations averaging around 2mg/m<sup>3</sup> would lead over time to decreases in measured criteria of lung function such as FVC and FEV<sub>1</sub>.

In summary it is clear that at substantial average exposure concentrations and over prolonged periods of time, inhalation of coal dust particles of respirable size gives rise to a variety of well-characterised conditions in coal miners. These include CWP and PMF which are uniquely associated with coal dust exposure and the more general conditions such as emphysema and chronic bronchitis. For all of these manifestations of ill-health in coal miners however the common prerequisite is gross dust exposure over lengthy time periods.

## **Studies on Health Effects of Particulate Air Pollution**

In terms of delineations in the international literature as to what particular component(s) of inhaled PM<sub>10</sub> particulates may instigate adverse health effects there is general agreement that typical contributing components of urban pollution are implicated and combustion emissions in particular are cited<sup>12,13</sup>.

Definitive evidence is widely available linking morbidity (illness) and mortality from respiratory and cardiovascular disease with exposure to particulate air pollution in the PM<sub>10</sub> and, particularly, the sub- 1 micron size range. While this general connection has been made by many scientists since air pollution episodes such as the London smog of 1952 a convincing explanation has been difficult to construct since the concentrations of air pollutant particles at which effects are observed to occur are very low by comparison with those to which many people are exposed in industrial workplaces without apparent harm.

Under the hypothesis of Seaton et al<sup>12</sup> the two key factors are that the urban pollution cloud comprises very small acidic particles, many of them in the nanometer (10<sup>-9</sup>m) size range, and which readily penetrate indoors and persist for long periods in the air.

Both the very small size of these particles and their ability to transport pollutant chemicals on the particle surface are postulated as being fundamentally important in lung inflammation at the microscopic level which promotes exacerbation of lung and cardiovascular disease.

In contrast industrial dust clouds consist mainly of much larger particles formed by the abrasion of rock or metal and these particles have no cohering urban pollutant additives but are instead “pure” particles. While they may have adverse health effects unique to themselves they are not capable of initiating the sorts of effects now accepted as being caused by the ultrafine air pollutant particles containing a substantial contribution from combustion processes (including automobile exhausts) and which are now recognised as the major constituents of typical urban air pollution.

A comprehensive publication by a World Health Organisation International Working Group<sup>14</sup> has broadly endorsed these conclusions of Seaton and co-workers; the WHO group’s conclusions are that the relationships between particle size, the nature of co-pollutants, exposure time frames and concentrations are highly complex but that the presence of, particularly, combustion-related contaminants adhering to particulate is of major significance in subsequent health effects arising in exposed persons.

In summary the issue of whether or not exposure to coal dust particles of respirable size can give rise to adverse health effects at environmentally encountered concentrations is not directly comparable with that of adverse health effects arising from exposure to PM<sub>10</sub> and finer particulates present in typical urban air pollution.

## **Environmental Health Effects of Coal Dust**

There are a limited number of papers and reports in the international literature dealing specifically with environmental exposure to coal dust. In one report<sup>15</sup> increases in asthma incidence in the community living “near” (the actual proximity is not specified) to an open cast coal mine site in South Wales were noted. This is a very brief report with a 1992 date and there does not appear to

have been any more extensive epidemiological study as a follow-up to the initial report. There are no data presented about dust levels, type of dust, possible presence of other factors predisposing towards asthmatic symptoms, and no information about diagnoses and other possible causes of the observed adverse health effects. Open cast mining uses heavy machinery on a large scale and creates very heavy airborne dust concentrations. It seems unlikely, although the matter is not discussed, that any dust suppression measures were in place at the South Wales open cast mining site.

A second paper<sup>16</sup> deals with respiratory ill-health in school children in an area of Merseyside, UK, who were exposed to coal dust and general industrial air pollutants. The research protocol is good and comparisons were made between an exposed (to coal dust) group of school children and two matched control groups. After statistical analysis of the results of a medical questionnaire presented to each group the authors concluded that there is a respiratory problem, in particular for a symptom labeled “recent cough”, in the children from the exposed area, but the problem could not be attributed to a specific cause. Symptoms and factors such as time off school were found to have increased in the exposed area but there was said to be “no evidence of serious damage”. The final comment of this paper is that ... “there is a child health problem but further studies are needed to determine if pollution is the cause”.

A study<sup>17</sup> by the University of Newcastle-upon-Tyne’s Department of Epidemiology and Public Health, published in December 1999, looked into possible links between open cast coal mining, associated coal stockpiling and respiratory health. The study approach was to use paired communities (five groups of such pairs) living either close to, or relatively distant from, open cast coal mine sites and conduct an examination of particulate concentrations and the incidence and seriousness of asthma and respiratory disease in children in these communities. The key findings showed that open cast activity was:

- associated with a small increase in the mean PM<sub>10</sub> particulate concentrations in areas close to open cast mines; and
- the respiratory health of children living close to the mine sites was very similar to that of children living in communities distant from such sites.

In particular, the levels of asthma, wheeze and bronchitis in the children were broadly similar. In four out of five of the community pairs there were slightly increased GP consultation rates for general respiratory, skin and eye complaints but the differences were small between the close and distant pair component communities.

A recent paper<sup>18</sup> describes a study of public health records data for nearly 16,500 persons in West Virginia which investigates relationships between health indicators and residential proximity to coal mining activities. The results are claimed to indicate that “high levels of coal production are associated with worse adjusted health status” and with higher rates of cardiopulmonary disease, chronic obstructive pulmonary disease, hypertension, lung disease and kidney disease. No data are presented on relative proximity of residents to coal mining activity, whether the cohort contained actual coal miners, or about airborne dust concentrations to which persons were exposed.

## **Conclusions**

There are few studies in the literature of respirable dust concentrations and/or health outcomes for non-occupational exposure to coal dust. What studies have been conducted show, at most, a very small difference between exposed persons and controls. In addition, these studies have typically involved communities living near open cast coal mining activities where it may be realistically expected that evolved dust concentrations will be much higher than from relatively passive coal stockpiling activities.

Conversely, the health effects of respirable dust on coal miners is an intensively researched area and the adverse health impacts are well documented. Those studies show that both relatively extremely heavy and lengthy exposure to respirable coal dust is necessary before adverse health impacts occur.

## REFERENCES

1. M. Jennings and M. Flahive, **Review of Health Effects Associated With Exposure to Inhalable Coal Dust**, Coal Services Pty Ltd, October 2005.
2. J.F. Hurley, J. Burns, L. Copland, J. Dodgson and M. Jacobsen, **Br J Ind Med**, 1982, 39, 120
3. **World Health Organisation Technical Report Series**, 734, Geneva, 1984.
4. **"Induced Disease - Drug, Irradiation, Occupation"**, Grune and Stratton, New York, 1964.
5. M.D. Attfield and K. Moring, **Am.Ind Hyg Assoc.J** 53, 486, 1992.
6. R.N. Naidoo, T.G. Robins, N. Seixas, U.G. Laloo and M. Becklake, **Respirable Coal Dust Exposure and Respiratory symptoms in South African Coal Miners; a Comparison of Current and Ex-Miners**, J. Occup. Environ. Med., 48, 581-590, 2006.
7. Commonwealth of Australia, Senate Community Affairs References Committee, **Workplace Exposure to Toxic Dust**, Canberra, 2005.
8. United States Environment Protection Authority, **Ambient Levels and Non-cancer Health Effects of Inhaled Crystalline and Amorphous Silica: Health Issue Assessment**, USEPA, Washington, 1996.
9. **Am J Ind Med** 6, 401, 1984.
10. **European J Resp Diseases** 63, 53, 1982
11. N.S. Seixas, T.G. Robins, M.D. Attfield and L.H. Moulton, **Br J Med** 50, 929 1993
12. A. Seaton, W. MacNee, K. Donaldson and D. Godden, **Particulate Air Pollution and Acute Health Effects**, The Lancet 345, 176, 1995.
13. **New Scientist**, # 1916, March 1994, pg 12 - 13.
14. **Health Aspects of Air Pollution with Particulate Matter, Ozone and Nitrogen Dioxide**, Report of a WHO Working Group, Bonn, Germany January 2003.
15. J.M. Temple and A.M. Sykes, **Asthma and Open Cast Mining**, British Medical Journal 305, 396, 1992.
16. B. Brabin, M. Smith, P. Milligan, C. Benjamin, E. Dunne and M. Pearson, **Respiratory Morbidity in Merseyside School Children Exposed to Coal Dust and Air Pollution**, Arch Diseases in Childhood 70, 305, 1994.

17. **Do Particulates from Opencast Coal Mining Impair Children's Respiratory Health?**, Department of Epidemiology and Public Health, University of Newcastle-upon-Tyne, December 1999.
18. M. Hendryx and M.M. Ahern, **Relations Between Health Indicators and Residential Proximity to Coal Mining in West Virginia**, American J Public Health, 98, 669-671, 2008.