Coal Services Pty Limited

Review of Health Effects Associated with Exposure to Inhalable Coal Dust

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Table of Contents

List of Figures and Tables

Glossary of Abbreviations used in this Report

ACGIH	American Conference of Governmental Industrial Hygienists
AS	Australian Standard
ANOVA	Analysis of variance
BMRC	British Medical Research Council
CFR	Code of Federal Regulations (US)
CI	Confidence Interval
COPD	Chronic obstructive pulmonary disease
CPP	Coal Processing Plant
CSPL	Coal Services Pty Ltd
CWP	Coal worker's pneumoconiosis
FEV_1	Forced expiratory volume in one second
FVC	Forced vital capacity
FVC	Forced Vital Capacity
ghm^{-3}	Grammes
GI Tract	Gastro-intestinal tract
HEG	Homogeneous exposure group
HSE	(UK) Health and Safety Executive
IARC	International Agency for Research on Cancer
IOM	Institute of Occupational Medicine
ISO	International Organisation for Standardisation
mg/m^3	milligrammes per cubic meter (of air)
ml/lb/yr	milliliters per pound per year
mppcf	Millions of particles per cubic foot
MRE	Medical Research Establishment
NOHSC	National Occupational Health and Safety Commission
NSW	New South Wales
OES	Occupational Exposure Standard
OR	Odds ratio
OSHA	Occupational Safety and Health Administration (US)
PAHs	Polycyclic aromatic hydrocarbons
PFR	Pneumoconiosis Field Research (British Coal Industry)
PM_{10}	Particles with a mean aerodynamic diameter less than 10 microns.
PNOC/R	Particles not otherwise classified or regulated
RCS	Respirable Crystalline Silica
RPE	Respiratory Protective Equipment
RSM	Respirable mass exposure
sCWP	Simple Coal Worker's (Miner's) Pneumoconiosis
SIR	Standardised incidence ratio
SMR	Standardised Mortality Ratio
SPSS	Statistical Package for Social Sciences
STP	Standard thermal precipitator
TB	Tracheo-bronchial
TES	Technical and Environmental Services (Laboratory, HSE)

TLV®	Threshold Limit Value (ACGIH)
TOTM	Total mass (exposure)
TOTV	Total volume (exposure)
TWA	Time Weighted Average
US EPA	United States Environmental Protection Authority

Executive Summary

The authors were commissioned by Coal Services Pty Ltd (CSPL) to investigate the possible relationship between exposure to inhalable coal dust and any adverse health effects. The authors were asked to address the following specific questions:

- What (if any) are the adverse health effects known to arise from exposure to inhalable coal dust?
- If no effects are reported in the literature with respect to inhalable coal dust, are there any comparisons that can be made to other inhalable dusts?
- What is a suitable workplace exposure standard for inhalable coal dust and what degree of protection does it provide for specific health outcomes?

The authors conducted a literature review sourcing primary and secondary research papers. The authors also contacted certain prominent researchers in this field.

The health effects of respirable coal dust are well known (CWP, silicosis, emphysema, PMF). However, the effects of inhalable coal dust have not been well researched. The authors divided the possible effects into the upper airways and extra-thoracic region and the effects in the thoracic region separately.

In relation to the extra-thoracic region, the authors found limited reports of cancer in the upper airways (larynx, pharynx, buccal cavity). However, these reports were few in number with no significant research identifying a causal relationship.

A greater body of research assessed the possible relationship between coal mining and gastric cancer. Most studies considered coal mining as an occupation, with only a small number of papers assessing the relationship with exposure to coal dust. There was inconsistent evidence of an association with coal mining, but no conclusive evidence of a dose-response relationship with respirable, or inhalable coal dust. Although it is biologically plausible that coal dust exposure could result in gastric cancer, the relationship could be explained by a number of lifestyle and other confounding factors.

Therefore, the authors are in agreement with the IARC determination that there is insufficient evidence to classify coal dust as a carcinogen. Well-designed cohort studies carried out on NSW coal miners have not shown an excess of cancers.

In relation to the thoracic region, the potential of inhalable coal dust to cause COPD and chronic bronchitis was assessed. Although there is convincing evidence of an association between respirable coal dust and COPD and chronic

bronchitis, no research directly assessing inhalable coal and health outcomes was found.

The authors then examined the research assessing the relationship between respirable dust and inhalable dust exposures. There was reasonable evidence of a correlation between the two metrics in the published research, although the studies have not been replicated by any Australian researchers.

The authors were provided with data from over 400 paired air samples collected by McFadden (2004) at 3 NSW collieries and 2 coal processing plants. No correlation was observed between the samples suggesting different conditions may exist in Australian mines and further particle size analysis should be conducted. Further data provided by Coal Services (2005) was broadly supportive of the data supplied by McFadden.

The authors were also requested to assess whether comparisons could be made to any other inhalable dusts. It was concluded that as coal has such unique physico-chemical properties, no such comparisons could be made.

In order to provide comment on an inhalable dust standard, the authors examined the basis for the current proposed standard of 10 mg/m^3. This standard was developed some 30 years ago, primarily to reduce the effects of reduced visibility in the workplace, eye and nose irritation and was set at this level as it was considered reasonably achievable by industry. Subsequent changes in sampling methodologies for inhalable dust led to an effective 3-fold reduction in the inhalable dust limit in real terms.

On the basis that there was no research assessing inhalable dust, the authors are unable to suggest a suitable health based standard. The authors are aware that ongoing health surveillance is conducted on NSW coal miners and suggest that prior to the implementation of any standard, an epidemiological study is conducted (if feasible) using inhalable dust levels.

After considering Coal Services' data, which indicate that mean levels of respirable coal dust are 1.51 mg/m^3 and that particulate respirators are frequently used by coal miners, a suitable time frame for the application of any inhalable dust standard would be several years; a period of 5 years is suggested. It would also be likely that the NSW coal mining industry would find it difficult to meet a standard of 10 mg/m^3, if imposed immediately.

Based upon these findings, the authors have made recommendations for further work. It is recommended that:

- Further sampling of inhalable dust should be carried out;
- Particle size distributions should be characterised;

- The incidence and prevalence of COPD and upper airways disorders in NSW coal miners should be monitored for possible correlation with inhalable dust levels; and,
- There should be a phasing in period of 5 years allowed for the implementation of a legally enforceable standard of 10 mg/m^3. In the meantime, this figure should be regarded as a Best Practice Guideline.

What (if any) are the adverse health effects known to arise from exposure to inhalable coal dust?

Introduction

The fact that exposure to respirable coal dust causes emphysema and simple coal worker's pneumoconiosis (CWP) is well known. However, it is suspected that coal miners may be susceptible to a spectrum of dust related respiratory diseases, which may not necessarily be caused by the respirable fraction and this highlights the need to develop a method to assess coal dust exposures in the thoracic and inhalable particle size fractions to supplement the current method for the respirable size fraction.

Mining, especially underground operations can produce high respirable coal dust exposures, but inhalable and thoracic exposures have not previously been well characterized. It is hypothesized that these larger particle size fractions may well contribute to bronchitis, decreased FEV_1 and possibly elevated cancers of the sinuses, buccal cavity, throat, and GI tract in occupational cohorts of coal miners.

Methodology

The authors undertook a literature search using search terms such as "inhalable +coal +dust" initially, from which emerged a small body of primary literature. This was further refined by searching on specific health outcomes, "cancer", "bronchitis" etc., against "coal", and then by searching for organs or anatomical regions, e.g. "stomach", "nose", 'lungs", etc., against "coal" and/or "inhalable dust".

From this search, other secondary sources were located from relevant references in the primary sources. Finally, using citations indices, a search of more recent literature referencing the initial literature was carried out. In reviewing the literature, it soon became evident that there was a paucity of studies on health effects in miners, which made reference to airborne concentrations of coal dust.

In addition to the literature search, analyses of data provided by Coal Services Pty Ltd and from McFadden (2004) have been conducted.

Inhalable Dust, Adverse Health Effects and Size-Selective Sampling

Inhalable coal dust is the dust fraction of the airborne particles which are taken through the nose or mouth during breathing into the body. The inhalable dust fraction has been defined by ISO 7708 (AS3640-2004).

This dust will deposit in the upper and lower airways of the respiratory tract and through mucocilary clearance mechanisms in the gastrointestinal tract. It can then be absorbed into the blood or lymphatics if soluble, finely divided or has prolonged retention.

Inhalable dust is made up of all the dust sizes that can deposit throughout the respiratory tract. It can include the larger particles that deposit in the upper airways (nose and throat), the smaller particles that can penetrate the upper airways and deposit in the lungs (thoracic fraction) and finer particles that can penetrate alveolar region or gas exchange region (respirable fraction).

The thoracic fraction is defined in ISO 7708:1995 and can be broken down further into the tracheobronchial fraction and includes the respirable fraction. The various fractions of inhalable dust are illustrated in figure1.

These fractions fit closely to the observed deposition of these particles in each region of the respiratory tract in experiments and post-mortem studies.

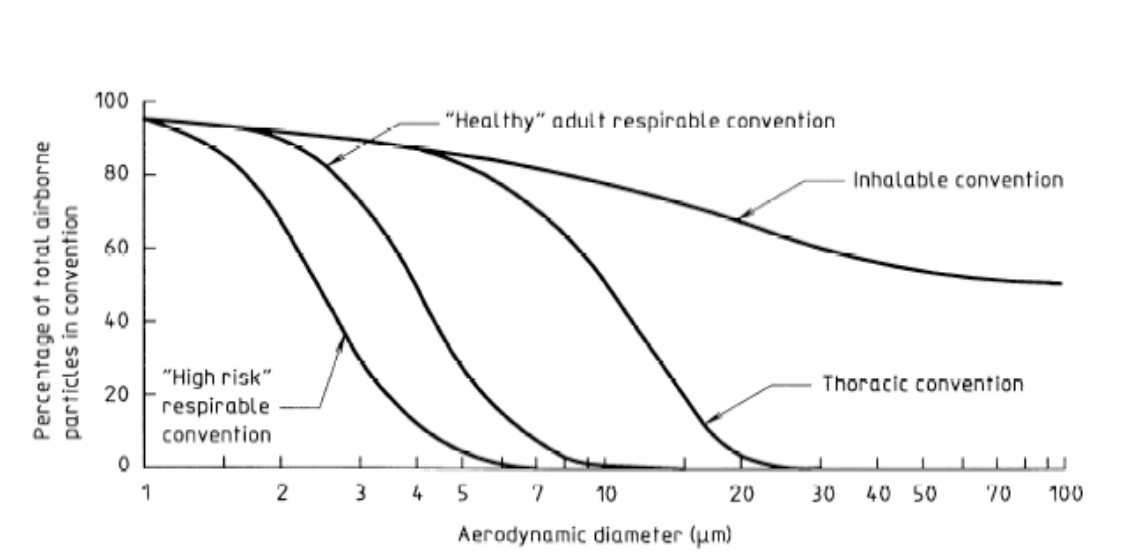

Figure 1 The inhalable, thoracic, respirable conventions as expressed as percentages of total airborne particles (ISO 7708:1995)

The effect of the particles depositing at the various sites of the respiratory tract will depend on the dose, the toxicity of the particles (local and systemic) and resident time at the zone of deposition and the clearance mechanism. To better assess the effect the various sampling conventions have been developed to assess the actual dose that will be occurring at the various zones. For example, only the fine particles (<10μm) are able to penetrate and deposit in the alveolar region. Therefore, for health outcomes occurring in this region the monitoring the fine particles will better predict outcomes, especially where gravimetric sampling is used and a few large particles of significant mass could bias the results if they made a significant portion of the inhaled dust.

A number of respirable dust conventions have been developed for the monitoring of respirable dust. These conventions were initially defined by the BMRC curve, US Atomic Energy Commission and ACGIH. In Australia, monitoring of respirable dust was initially in accordance with the BMRC curve, but with a move to standardisation, the ISO 7708 curve has recently been adopted (AS 2985:2004).

The other fractions are the thoracic dust fraction or the total inhalable dust and are defined further in table 1.

Inhalable dust is normally monitored in cases where there are upper respiratory effects or systemic effects. This will be discussed further. At this stage thoracic or tracheobronchial dust exposure is not routinely monitored in the occupational setting. However an indicator of the thoracic fraction (PM_{10}[a]) is monitored in environmental monitoring. This is not a measure of personal exposure and is normally to a mixture of chemicals in air pollution. The monitoring of the thoracic fraction could be useful in assessing outcomes for particles primarily exerting their effect in this region resulting in outcomes such as asthma and bronchitis (such as flour and grain dust).

Extra-thoracic Particle Deposition and Clearance

Airborne particles that deposit in the head (extra thoracic region) may be absorbed and/or swallowed, although some may be expelled directly from the body by bulk clearance mechanisms such as sneezing, spitting or nose blowing.

The respiratory surfaces of the nasal turbinates are in very close proximity and designed to warm and humidify the incoming air; consequently they can also function effectively as a diffusion deposition site for very small particles and an effective absorption site for water-soluble gases. The turbinates and nasal sinuses are lined with cilia which propel the overlying mucous layer posteriorly

[a]Particles with a mean diameter less than 10 microns. It is the particles in the air that can get past the upper respiratory tract (nose and mouth) into the thoracic region of the lungs. The US EPA defines a cut point and collection curve for PM_{10} monitors. The cut point is at 10microns but due to the curve slope it may a small number of collect particles up to 20 microns.

via the nasopharynx to the laryngeal region. Thus, the airways of the human head are major deposition sites for the largest inhalable particles (>10μm aerodynamic diameter) as well as the smallest particles (<0.1μm aerodynamic diameter). For the most part, the ET structures are lined with a squamous, non-ciliated mucous membrane. Collectively, the movement of upper airway mucus, whether transported by cilia or gravity, is mainly into the gastrointestinal (GI) tract.

Table 1 Site of Deposition in the Respiratory Tract (Phalen 1985).

Region	Anatomic Structure	Other Terminology
Extrathoracic (ET)	Nose Mouth Nasopharynx Oropharynx Laryngopharynx Larynx	Head airways region Nasopharynx (NP) Upper respiratory tract (URT) Naso-Oro-Pharyngo-Laryngeal (NOPL)
Tracheobronchial (TB)	Trachea Bronchi Bronchioles (including terminal bronchioles)	Lower conducting airways
Alveolar (A)	Respiratory bronchioles Alveolar ducts Alveolar sacs Alveoli	Gas exchange region Pulmonary region

Adverse effects of inhalable coal dust.

Possible adverse health effects associated with inhalable coal dust include:

Extrathoracic Region

- Nasal Cancer
- Sinusitis
- Buccal and lip cancer
- Pharyngeal and laryngeal cancer
- Gastric Cancer
- (Throat and Eye Irritation)

Tracheobronchial Region

- Chronic bronchitis
- Chronic obstructive airways disease and loss of FEV_1[b]

Alveolar Region

- Coal Workers Pneumoconiosis (CWP)
- Silicosis
- Emphysema
- Tuberculosis
- Caplan's Syndrome

In examining this topic, it was considered that larger (i.e. non-respirable) particles would deposit in the upper respiratory tract and upper airways and therefore, attention was paid to health impacts reported in the nose and nasal passages, the mouth (buccal cavity), pharynx, nasopharyngeal, oropharyngeal and laryngopharyngeal regions, the larynx, trachea, bronchi, bronchioles to the terminal bronchioles.

However, as respiratory dust is a component of inhalable dust, we will briefly discuss the effects in the alveolar region and the relevance of the respirable dust metric.

[b] FEV1 Forced expiratory volume in 1 second, a measure of obstructive airways disease and narrowing of the small airways.

Alveolar Region Health Effects, CWP and Silicosis

The association between Coal Worker Pneumococcosis. (CWP) and the exposure to inhalable coal dust has been widely established in the literature over many years. This has been the subject of a number of epidemiological investigations and the exposure–response relationship for respirable coal dust is well known. This has led the development of exposure limits and significant reductions in dust levels in coal mines and virtual elimination of these disease outcomes.

Early post mortem studies showed that particles ranging from 0.6μm to 2μm are primarily retained in the alveolar region with 90% of the particles being less than 5μm. (Leiteritz et al. 1972, Green 1998, Reisner & Robock 1975)

These studies show an increased retention or an enrichment of this finer fraction occurs with the 0.2-0.35μm making up 1.9% of the coal dust being inhaled but making up 6.1% of the lung dust retained in the lung. In contrast the 3.5-6.3μm fraction made up 47% of the coal dust but only 11.4% of the dust retained in the lungs (Leiteritz et al. 1972).

Hence when assessing dust exposure for the risk of developing CWP, it is logical having a sampler collecting primarily particles less than 3.5μm and larger particles with less efficiency. Additionally studies of the coal dust particles have found that silica particles, which are more pathogenic, have maximum concentrations in the very fine fraction (1.1μm and 2.0μm) and mean size 1.6μm (Leiteritz, Bauer, & Bruckmann 1970).

Subsequently a significant number of epidemiological studies have assessed the relationship between exposure to respirable coal dust, respirable crystalline silica (RCS) and CWP and silicosis. **The relationship has been firmly established with the respirable fraction of coal dust and silica and these disease outcomes. It is now well recognised that respirable coal dust and RCS is the correct metric for assessing exposure and risks of developing CWP and silicosis** (Attfield & Kuempel 2003, Kuempel et al. 1995, Kuempel et al. 2003, Soutar et al. 2004).

Extra-thoracic Region Health Effects

A review of the literature has been conducted to assess whether there are any consistent reports of adverse health outcomes in the upper airways and gastrointestinal tract. The possible outcomes will be discussed individually to assess whether they are possibly causal and whether specific dose response relationships have been established.

The most serious outcomes such as cancer have been given priority. In considering this, the Joint Coal Board Cancer Surveillance study by Kirby, Berry and Buckley (2000) data was also included.

Table 2 Results from NSW Cancer Surveillance in Coal Miners (Kirby, Berry, & Buckley 2000)

Cancer by Site	*1973 to 1992*					*1973 to 1997*				
	Obs	Exp	95% Confidence Interval			Obs	Exp	95%Confidence Interval		
			Lower	SIR	Upper			Lower	SIR	Upper
All Cancers	302	366.35	0.73	0.82	0.92	596	732	0.75	0.81	088
Lip	10	9.85	0.49	1.02	1.87	31	17.80	1.18	1.74	2.47
Other pharynx	9	18.15	0.23	0.5	0.94	18	32.33	0.33	0.56	0.88
Stomach	7	10.01	0.28	0.70	1.44	17	19.89	0.50	085	1.37
Colon	27	27.04	0.66	1.0	1.45	45	56.58	0.58	0.80	1.06
Rectum	15	18.13	0.46	0.83	1.36	39	39.88	0.70	0.98	1.34
Other gastrointestinal	9	15.71	0.26	0.57	1.09	26	31.93	0.53	0.81	1.19
Larynx	6	5.9	0.37	1.02	2.21	10	11.8	0.41	0.85	1.56
Lung	29	39.16	0.5	0.74	1.06	49	75.19	0.48	0.65	0.86
Pleura	2	1.9	0.13	1.05	3.80	6	6.45	0.34	0.93	2.03
Bone &Con Tissue	7	8.32	0.34	.8	1.73	7	25.35	0.11	0.28	0.57
Melanoma	85	75.52	0.90	1.13	1.39	138	128.7	0.90	1.07	1.27
Prostate	6	13.91	0.16	0.43	0.94	49	68.81	0.53	0.71	0.94
Testis	21	22.18	0.59	0.95	1.45	32	30.22	0.72	1.06	1.49
Bladder	10	12.45	0.39	0.80	1.48	16	22.49	0.41	0.71	1.16
Brain and Other CNS	14	13.36	0.57	1.05	1.76	23	24.29	0.60	0.95	1.42
Lymphomas	18	29.01	0.37	0.62	0.98	35	49.72	0.49	0.70	0.98
Leukaemia and Mult. Myelomas	6	15.55	0.14	0.39	0.84	21	30.07	0.43	0.7	1.07
Other Cancers	21	22.4	0.58	0.94	1.43	34	61.32	0.38	0.55	0.77

Nasal cancer

There have been reports of nasal cancer being associated with the occupation of coal mining, however these appear to be sparse. Acheson, Cowdell, & Rang (1981) found a statistically significant excess of nasal cancer incidence among miners and quarrymen from England and Wales during 1963–67. The standardised incidence ration (SIR) for coal miners was 1.60 (48 observed; [95% CI 1.18 – 2.21]) with the highest risk detected for coal face workers (22 observed; Standard Incidence Ratio (SIR) 4.30; [95% CI 2.69 – 6.5]) and a smaller non-significant excess among underground workers (30 observed, SIR 1.32; [95% CI 0.89–1.88]).

However, the authors conceded that in this study the results may be due to bias and no analysis of other confounders such as use of snuff and tobacco was undertaken. Subsequent cohort and mortality studies have not found such an association. In particular, a cohort of 18166 miners over 30 years (Miller et al. 1997) did not report such an association. No excess has been reported in NSW coal miners (Brown et al. 1997).

A pooled analysis of 12 case-control studies (627 cases, 3136 controls) did not find an excess of sino-nasal cancers in coal mining (Luce et al. 2002).

The authors conclude that there is insufficient evidence to support an association of inhalable coal dust and sino-nasal cancer.

Sinus Disease

Inhalable coal dust could result in inflammation of the para-nasal sinuses, but reported studies are extremely limited. Ozdemir et al. (2004) conducted a cross-sectional survey of 55 coal workers (26 with CWP) and with 20 controls.

The researchers conducted CT scans of the para-nasal sinuses and found that coal workers had greater prevalence of sinus disease (as measured by degree of mucosal thickening) in coal workers. All coal workers with CWP had some degree of sinus mucosal thickening on CT scanning with 35% having evidence of extensive disease, compared to 24% of coal workers without CWP and 15% of controls, but these differences were not statistically significant.

This study does indicate that further research into this area is appropriate. However, as this was a cross-sectional study, with the very small numbers, no conclusive statements can be made. No direct assessment of exposure was made and the results could well have been explained by selection bias.

The authors conclude that theoretically sinus disease could develop as a result of exposure to inhalable coal dust and this is supported by the survey of coal workers. However, no firm statements can be made on the published evidence about any association between sinus disease and coal dust.

Laryngeal cancer

In a case-control study 283 histologically confirmed cases of cancer in men treated during the first semester of 1983 at the Regional Cancer Institute in Northern France, Haguenoer (1990) found a threefold excess of laryngeal cancers among coal miners (OR 3.2, 95% CI 1.1 – 9.7). The authors comment that this may be related to a higher reported use of chewing tobacco by coal miner and exposure to mineral oils.

Laforest (2000) studied male patients with incident primary laryngeal and hypopharyngeal squamous cell cancers, diagnosed and histologically confirmed in 15 French hospitals between 1 January 1989 and 30 April 1991. The study was restricted to men and included 201 hypopharyngeal cancers, 296 laryngeal cancers and 296 controls (patients with other tumour sites). Exposure to coal dust (by report from patient) was associated with a non-significant increased risk of laryngeal cancer (OR 1.67, 95% CI 0.92 – 3.02), but no dose-response pattern was found. No association with silica dust exposure was found.

Hypopharyngeal cancer was found to be associated with exposure to coal dust (OR 2.31, 95% CI 1.21 – 4.40) with a significant rise in risk with probability ($p<0.005$ for trend) and level ($p<0.007$ for trend) of exposure. There was no increasing relationship with length of exposure. (Laforest et al. 2000). There is significant potential for recall bias in this study and no direct assessment of exposure was undertaken. The authors were unable to find any other consistent positive reports of laryngeal and pharyngeal cancer in the literature.

In contrast the mortality cohort studies have not been reported to show an association with this outcome despite reasonably large numbers of subjects (Miller et al. 1997). The cohort studies on NSW coal miners have not shown an excess of pharyngeal and hypopharyngeal cancer (Brown et al. 1997).

Therefore, it is possible that bias and confounders could explain the associations observed in the case-control studies. It remains plausible that long term exposure may result in such an association but there is at this stage, no evidence to establish a causal link between inhalable coal dusts and laryngeal cancer. The cohort studies provide reasonable evidence that there is no association.

Buccal cavity and pharynx

Using data from deaths in 1950 in US working men aged 20 – 64, Enterline (1964) estimated cause specific standardised mortality ratios (SMRs) among coal miners. The observed mortality excesses for several cancers, including of particular relevance to this report, the buccal cavity and pharynx (21 observed; SMR, 1.31; (95% CI, 0.81 – 2.01)).

In the study referred to above by Haguenoer (1990), coal miners showed a threefold excess risk for cancer of the lip (4 cases, no control) and of the buccal

cavity (OR 3.5, 95% CI 1.1 – 11.8). The authors acknowledged the frequent use of chewing tobacco in underground coal miners, a major risk for buccal cavity cancer.

An excess of lip cancer has also been observed in the NSW coal miners cancer-cohort study has also been observed (SIR 1.74 CI 1.18-2.47). Most studies have indicated that tobacco smoking and sun exposure are causally associated with lip cancer. With this small association observed in the NSW cohort, it is unlikely that this excess is related to inhalable dust and more likely to be related to other factors including non-occupational sun exposure (in Australian miners), cigarette and pipe smoking, and chewing tobacco.

Gastric cancer

There has been some evidence in the literature over the last fifty years or so indicating that coal miners may be more at risk from gastric cancer.

Coal dust contains silica, metal residuals, and polycyclic aromatic hydrocarbons (PAHs). It has been suggested that coal particles concentrated in the lung are eliminated by the ciliary cleaning function and then swallowed with saliva, thus reaching the stomach (Ames, 1982). The combustion of coal products releases PAHs, including benzo[a]pyrene, a known carcinogen. Hence, it is postulated that dust particles, essentially inhalable, are swallowed and thereby exert a carcinogenic effect upon the stomach.

A significant number of other studies on stomach cancer in coal miners have published, since the association was first identified by Stocks (1962). These are summarised in table 3. There are many studies showing increased gastric rates in coal miners, although the studies are not consistent and few have examined the relationship to coal dust exposure. Most have not established an exposure-response relationship.

Only one paper has been found that indicates a possible linkage to the exposure high levels of inhalable dust and gastric cancer. Swaen, Meijers and Slangen (1995) carried out a study to investigate the mortality patterns in a group of 3790 Dutch coal miners. The group had been selected from people with abnormal x-ray films, from routine medical examinations in the 1950s. Causes of death up to 1 January 1992 were traced and it was found that mortality from gastric cancer was significantly increased (SMR147.5, 95% CI 122.3 – 176.3).

Interestingly, this risk of mortality from gastric cancer was confined to workers with no pneumoconiosis or pneumoconiosis present in a mild form. They found the risk increased with duration of employment with the highest risk in those with no pneumoconiosis and greater than 30 years of employment underground. Workers with more severe manifestations of pneumoconiosis did not experience an excess in mortality from gastric cancer. This suggested to these researchers

that the impairment of the pulmonary clearance system as found in severe pneumoconiosis was such that inhaled dust did not reach the digestive tract.

The researchers commented that, "*In comparison with current exposure standards the coal miners were exposed to high dust concentrations. Several industrial hygiene surveys were carried out in the past. The average of the total gravimetric dust samples was 27 mg/m^3 with an average of 5% quartz. Although there were differences in industrial hygiene between pits it is likely that workers of all coal mines were exposed to coal dust concentrations of 20 mg/m^3 and over*." However, no direct measures of inhalable dust in the work groups were made.

The theory that miners with impaired clearance mechanisms have a lower risk of gastric cancer has not been supported by other studies. Ames and Gamble (1983) addressed this issue in their study of smoking and non-smoking coal miners. Coal miners with airways obstruction were shown to have a slightly elevated risk of stomach cancer, and this could not support the theory of increased gastric exposure through normal bronchial clearance mechanisms would increase gastric cancer rates.

Additionally, the mass fraction of the dust that in deposited is the alveolar region is very small compared to both respirable and total dust that is deposited in the whole respiratory tract (mouth, nose, upper and lower airways) and therefore, those miners with pneumoconiosis are likely to have been exposed to significantly more total dust than those without. The lack of dose-response seen with increasing severity of pneumoconiosis weakens any association between inhalable coal dust and gastric cancer.

A well designed cancer morbidity study of 23 630 NSW coal miners between 1973 and 1992 showed a SIR of 0.85 (CI 0.5-1.37) for gastric cancer (Brown et al. 1997).

In the large cohort study of 18 166 UK coal miners an excess of gastric cancer was observed (Miller et al. 1997). A total of 408 000 person years at risk was assessed in the follow-up period of thirty years to 1992.

The observed SMR for gastric cancer was 124 (CI 110-141). The study had comprehensive assessments of respirable dust and *was unable to show any relationship with cumulative dust exposure or even time spent in the industry*.

To explain this excess the study authors examined the expected rates for the social class and although miners are classified Social Class III (with an expected SMR for gastric cancer 109), by lifestyle they probably fit into Social Class IIIM (SMR 118) or Social Class IV (SMR 124) with Social Class V having an SMR of 147.

However, with the healthy worker effect taken in account, the relative SMR remains above that for a similar social class. The authors suggested that, although ingested coal from working underground may provide an explanation, the lack of relationship with time spent underground weakens this assertion. The authors suggest other possible causes that could explain the observed association including use of chewing tobacco underground, diet and alcohol consumption as well as other environmental factors from living in mining communities.

Given that only weak and inconsistent associations with gastric observed in the studies, it is probable that there is other factors leading to an excess of gastric cancer in coal miners.

The epidemiology of gastric cancer is complex, there is significant geographical variation (rate varying from 10/100,000 in North America to >40/100,000 in Asia) and even rates vary significantly within countries.

A survey of 2099 retired coal miners and referents from the general population found that 30.5% of miners took chewing tobacco either solely or in combination with smoking (Harrison, Morris, & Hardcastle 1993). They found dyspepsia a frequent symptom (20-30%) in the miners and significantly more showed acute on chronic gastritis on histology (22 *v* 11%, $p<0.01$) on gastroscopy and increased rates of intestinal metaplasia. Intestinal metaplasia is frequently associated with helicobacter infection (Huang et al. 1998).

Diet is a significant factor in the development of gastric cancer with salted meats, pickled foods being associated with excess rates and fruit and vegetables being associated with a protective effect. There is reasonable evidence that smoking is a causal factor. Other occupations associated with Social Class IV have been shown to have an excess risk of gastric cancer including work in agriculture and fishing. This may be an indication of diet and lifestyle.

There are many recent studies that show that helicobacter pylori infection is a significant cause of gastric cancer with high risk ratios from 2 to 18 (Parsonnet et al. 1991). A recent meta-analysis indicates the risk of gastric cancer with helicobacter infection is 1.98 (CI 1.32-2.78) (Huang et al. 1998). Helicobacter infection rates vary both with age and social class (Sitas et al. 1991). In summary, there is strong (odds ratio 2-3) and consistent evidence that helicobacter is associated with gastric cancer. No studies assessing the excess of gastric cancer in coal miners have controlled for this factor.

The authors conclude that, although it is biologically plausible that inhalable coal dust could result in gastric cancer, the inconsistent associations observed in the studies and consistent lack of dose-response relationship cumulative respirable dust exposure (and in most cases length of underground employment) suggests

that other lifestyle factors (smoking and diet) and probably helicobacter infection are likely to explain the observed gastric cancer excess in coal miners.

The research to date has not adequately assessed these significant factors in coal miners, however, where an indicator of dust exposure has been assessed no exposure-response relationship has been observed.

Table 3 Summary of gastric effects in coal miners

Study	Population	Effect studied	Result	Authors
Cohort and Proportional Mortality				
Gastric carcinoma in a coal mining region	Residents of coal mining communities	Gastric Cancer	Estimated SMR for coal miners 158, non-miners 47, no actual assessment of occupation, no confidence interval assessed	Matolo et al. 1972
Mortality rates in coal miners	553 US male coal miners in 1937; follow up 1938-66	Digestive system cancers	SMR 2.10 (8 cases; 95% CI 0.91 – 4.25)	Enterline 1972
Mortality in Coal Miners	3239 deaths in 1961 among coal miners aged 20-64 identified by the NCB	All Cancers including Stomach Cancer	Stomach Cancer SMR Face Workers 1.01 Underground Workers 1.28 Surface Workers 0.32 (CI not calculated)	Liddel 1973
Mortality in UK Coal Miners	6212 miners and ex-miners aged > 20 yrs; follow up through 1950-1970	Cancer by radiographic category including stomach cancer	Stomach Ca by rad. cat: SMR (no;[CI]) Non-miners: 1.13(13;[0.60-1.93]) 0: 1.60 (52;[1.19-2.09]) 1-3: 1.08 (21;[0.67-1.66]) A-C: 1.84 (23;[1.17-2.76])	Cochrane et al (1979)
Mortality of men in the Rhondda Fach 1950 - 1980	30 yr follow up of 8526 men, aged >20	Mortality inc stomach cancer	Stomach Ca by cat: No; SMR (CI) Non-miners: 24; 1.31 (0.84-1.95) Cat 0: 69; 1.52 (1.18-1.92)* Cat 1,2,3: 33; 1.23 (0.85-1.73) Cat A: 14; 2.17 (1.18-3.64) Cat B, C: 13; 1.51 (0.81-2.59)	Atuhaire et al, 1985
Gastric cancer in a south Wales valley.	7939 men followed for 30 years	Gastric cancer	SMR -94 for non-miners, 125 for miners and ex-miners, not statistically significant.	Atuhaire, et al 1986
Cause specific mortality in US coal miners	23,232 US Coal miners covered by UMW Health & Retirement funds in 1959, follow up 1959-71	Cause specific mortality	129 Observed malignant neoplasms of the stomach, vs 92.4 expected, SMR = 140. CI 1.17-1.66 Elevated incidence seen only in miners aged 70 and over.	Rockette, 1977
Cause specific mortality in Western Aust Coal Miners	213 coal miners during 1961-1975	Cancer mortality inc stomach cancer	Gastric Cancer SMR 2.2 (CI 0.27-8.03)	Armstrong at al 1979
Cause specific mortality in US coal miners	Four cohorts of approx 20,000 coal miners provided cases of stomach and lung cancer	Cancer mortally including stomach cancer	Cancer: SMR Stomach Cancer: 1.55 (0.76-3.17) > 30 years smoking: 3.52 (1.11-11.7)	Ames and Gamble 1983

Study	Population	Effect studied	Result	Authors
Cohort and Proportional Mortality ctd.				
Dust exposure, pneumoconiosis & Mortality	26,363 British coal miners from 20 collieries over 22 yr periods ending in 1980.	General mortality	Excess of gastric cancer with cumulative dust exposure p = 0.05 ($\chi 2 = 4.07$)	Miller & Jacobsen 1985
Dust exposure, pneumoconiosis & Mortality	26,363 British coal miners from 20 collieries over 22 yr periods ending in 1980.	Cancer mortality	SMR 124 (CI 110-141). Excess of gastric cancer, no association with cumulative dust exposure	Miller et al 1997
Gastric cancer in pneumoconiotic coal miners	3790 Netherlands coal miners, who participated in lung study in 1950s, with radiological signs of disease, follow up to 1992	Gastric cancer	120 Deaths from gastric cancer observed, vs 81.4 expected; giving SMR of 147.5 (95% CI 122.5–76.3).	Swaen, Meijers & Slangen 1995
Mortality in US coal miners	8878 coal miners medically examined 1969-71;follow up through 1979	Gastric Cancer	SMR 0.91(0.39-1.80), slight exposure response gradient for gastric cancer	Kumpel et al 1995
Coal mine dust exposure and cancer mortality in German coal miners	4628 Saar coal miners, with >5 yrs work underground	Cancer Mortality	6 cases of stomach cancer, SMR of 0.62, 0.95 CI = 0.23 – 1.35.	Morfeld et al 1997
Mortality study in NSW Coal Miners	23,630 NSW coal miners followed between 1973 and 1997	Cancer Morbidity	SIR all cancers 0.81. (0.75-0.88) SIR Gastric cancer 0.85 (0.5-1.37)	Brown et al 1997
Case Control Studies				
Stomach cancer & work in dusty industries	95 incident cases & 190 age & sex matched community controls, in Stoke-on-Trent. 73 m, 22 f, aged 70 or less, of whom 26 were coal miners.	Stomach cancer	Relative risk of 1.2 (95% CI = 0.5 – 2.9) for employment in industry with high dust exposure, vs relative risk of 3.6 (95% CI = 1.1 – 12.2) for those manually employed in industry, but never in a job with high dust exposure.	Coggon, Barker & Cole, 1990.
Gastric cancer in coal miners: a case control study in a coal mining area	323 male cases of gastric cancer between 1/1/73 & 31/12/83 in the Netherlands & 323 controls matched by age	Gastric cancer	OR = 1.14 (95% CI = 0.34 – 1.73)	Swaen, Aerdts, et al 1985
Cancer in coal miners: final report	683 male cases of gastric cancer, with matched controls from Limburg, Netherlands.	Gastric cancer	OR = 1.15 (95% CI = 0.89 – 1.47) **No evidence of a dose response relationship**	Swaen, Collette, et al, 1987
Occupation & gastric cancer in	354 new cases of adenocarcinoma	Gastric cancer	For coal & coke mining, 11 cases & 3 referents were observed with a	Gonzalez et al. 1991

Spain	diagnosed between 11/87 & 12/89, matched with case referents.		high OR (11.8) which was statistically significant, but with a wide CI (95% CI 1.36 – 103)	
Other Studies				
Occupation and Gastric cancer	Review article	Gastric cancer	"Increased" risk of gastric cancer for the occupation of coal mining, based on 13 articles cited.	Raj, Maybery & Podas, 2003
Stomach cancer mortality in mining towns.	195,934 inhabitants of UK mining towns; 105,082 inhabitants of non-mining towns in 1971 census.	Stomach cancer	SMR for mining towns, 92 for men & 104 for women vs non-mining towns, SMR 91 & 86 respectively.	Davies 1980
Dyspepsia	670 North Nottinghamshire Coal miners vs 743 age & sex matched general population	Upper gastrointestinal symptoms	Anorexia p <0.001 Weight loss p<0.001 Dysphagia NS Epigastric pain NS Heartburn NS Vomiting NS	Harrison & Morris 1989

Inhalable Coal Dust and Cancer

In summary, on reviewing the reported literature, there is no conclusive evidence that inhalable coal dust is a causative factor in the development of cancer in the lung, stomach, or upper airways. There have been some reports of an excess of some cancers in coal miners, the most consistent being gastric cancer. However, quite powerful studies have not shown any exposure-response relationship with dust. Although ongoing research in this area is indicated, it is suggested that other lifestyle factors may be responsible for the observed effect. These have not been adequately taken into account in the research to date.

IARC have determined that there is inadequate evidence in humans for the carcinogenicity of coal dust; and that there is inadequate evidence in experimental animals for the carcinogenicity of coal dust.

Their overall evaluation is that coal dust cannot be classified as to its carcinogenicity to humans (Group 3).

This conclusion was based on both reviewing the epidemiological evidence and studies of animals exposed to coal dust. *The authors would support the IARC conclusion that there remains inadequate evidence for the carcinogenicity of inhalable coal dust.*

Inhalable Coal Dust, Bronchitis and COPD

The potential of coal dust to cause pneumoconiosis has long been recognised, but as pneumoconiosis is essentially linked to exposure to respirable dust it will not be considered in this paper. It is also important to note that severity of pneumoconiosis does not correlate well with severity of chronic bronchitis, airflow obstruction and chronic obstructive airways disease (Leigh et al. 1982, Rogan et al. 1973).

Chronic bronchitis is a clinical entity defined as the presence of a cough, sputum production (for more than two months on two consecutive years) and is associated with bronchial gland enlargement and mucous hypersecretion.

Airways obstruction is normally assessed as the loss of FEV_1, that is, the volume of air able to be expelled from the lungs in one second. As the FEV_1 decreases, there is increasing likelihood of shortness of breath, initially on exertion and then at rest, and increasing disability.

Chronic obstructive pulmonary disease (COPD) is typically defined as a slowly progressive disorder characterised by airflow obstruction, which does not vary over periods of observation and is irreversible. There is general agreement that COPD is the result of increased peripheral airways resistance, secondary to a mixture of small airways disease and emphysema (Calverley 2000). This can not be stated with certainty with some research showing that there is significant large airways obstruction in coal workers (Hankinson et al. 1975), and some research showing a correlation between chronic bronchitis pathology in large bronchi and symptoms and signs of obstructive airways disease (Glick, Outhred, & McKenzie 1971).

Chronic bronchitis is a frequent finding in coal miners with prevalence rates between 33-38%. A significant proportion of the reported prevalence would be attributable to smoking, making the investigation of causation difficult. In non smokers, the rates can be 5-15% in coal miners (Leigh et al 1986, Henneberger & Attfield 1996).

Emphysema is a pathological condition where there is enlargement of the air spaces (alveoli) distal to terminal airways or bronchioles accompanied by destruction of their walls.

It has also been widely published that coal dust exposure is associated with chronic bronchitis, chronic obstructive pulmonary disease and disability from COPD and loss of FEV_1. In 1992, this led the British Government to classify chronic bronchitis and emphysema in coal miners as an occupational disease for which industrial injuries benefit can be paid (Seaton 1998). This was on the primarily basis of the results from the Pneumoconiosis Field Research (PFR) unit studying the dust exposure, symptoms, chest x-rays and lung function of some

50 000 miners over 30 years. An initial criterion for benefits was evidence of dust exposure on chest x-ray (CWP).

The criterion was amended in 1996 and included evidence of employment in an underground mine for 20 years and a FEV_1 deficit of 1000ml. There was no need for radiographic evidence of pneumoconiosis or dust exposure. This was on the basis that in both smokers and non-smokers the cumulative exposure required to double the risk of disabling COPD was approximately 100-200ghm^{-3}[c] (Marine, Gurr, & Jacobsen 1988; Soutar et al. 1993). The 20 years of employment criteria was an estimate of the duration of employment that in many mine would have led to a cumulative exposure of 200ghm^{-3} (Newman-Taylor & Coggon 1999).

There is ongoing debate between experts in this field on whether coal mine dust can produce clinically relevant loss of lung function (Morgan 1999).

This matter was the subject of massive litigation in the UK between British Coal and a selected number of underground miners.

The decision is summarized in an editorial in Thorax (Rudd 1998). Much of the litigation focused on the interpretation of epidemiological data derived by the Coal Industry itself.

In general it was accepted by both parties that heavy dust exposure is associated with measurable decrements in spirometry (FEV_1). There are two main schools of thought. One associated largely with Dr WKC Morgan is that there is a small average fall in FEV_1 affecting most miners, and is due to sputum in the airways (Morgan 1986). Such sputum with associated small decrements in FEV_1 and is not likely to cause significant disability. The alternative view is that the small average decrement in FEV_1 in the population of miners includes a number of men who are more seriously affected and are, in fact significantly disabled as a result of their exposure to underground dust.

Justice Turner indicated in his judgement that the view of Morgan ignored the import of much of the available evidence.

The final judgment made the following 6 points:

1) Coal mine dust (coal or stone) is a cause of centri-acinar emphysema.
2) Such emphysema may, and usually does, lead to a loss of ventilatory capacity most easily demonstrated by the loss of FEV1.
3) Tobacco smoke has similar effects as (1) and (2) above.

[c] g.h.m^{-3} is a measure of cumulative dose. If a miner works 1760 hours per year and is exposed to 2mg/m^3 of respirable coal dust, the cumulative dose is 1760 x 2/1000. After 20 years the cumulative dose will be 70g.h.m^{-3}, after forty years it is 140g.h.m^{-3}. After 30 years of average exposures of 3mg/m^3 the dose is 158g.h.m^{-3}, after 40 years, 211g.h.m^{-3}.

4) It is probable, but not certain, that there is a common causal pathway to both cigarette and mine dust induced emphysema, which usually gives rise to breathlessness.
5) Whether (4) is established or not, the effects are generally the same in that there is a spectrum of effect which is not clinically detectable in the majority of cases but in the minority does produce a range of effects from simple impairment, frank disability and, occasionally death.
6) In the individual smoker it is not possible to attribute the cause of breathlessness either to the one insult or the other; this is so whether or not there is a common pathway.

The court recognised that dust exposure and cigarette smoking had similar effects and that in the individual it was possible to assign the proportion of causality according to the level of dust exposure and cigarette smoking.

Compensation was then awarded on a proportional basis. This approach emphasises the point that it is not valid to suggest that, in a dust exposed cigarette smoker, the disability can be treated as if it was due entirely to one cause or the other.

It makes no scientific sense to ask a physician which is "the most likely cause of the disability" or whether one or the other made a "significant contribution." The appropriate scientific method is to estimate, as far as possible, the contribution by each cause (Muir 1999).

Review of Evidence on the Association of Coal Dust and COPD

Coggon & Newman (1998) reviewed the scientific evidence for the association of coal mining and COPD. (It is worth noting that Professors Coggan and Rudd were expert witnesses for the plaintiff in the British Coal litigation.)

To summarise Coggon and Newman's review, the epidemiological investigations are of four main types:

1) Studies comparing lung function in miners and non-miners in other occupations
2) Patterns of symptoms and lung function in miners and non-miners
3) Analysis of mortality from COPD in relation to coal mining.
4) Assessment of emphysema in post-mortems on coal miners.

There are a number of studies that show that coal miners have a reduced lung function in comparison to non-miners. The weakness of these comparative studies is the inability to control for confounders and non-occupational influences in lung function.

Of more relevance are the studies that have assessed the patterns of symptoms and lung function in miners according to their exposure to coal dust. This research primarily related to the study of two cohorts, the first, of British coal

miners by the PFR program and second, of US coal miners in the US National Study of Coal Workers Pneumoconiosis (NSCWP).

The PFR researchers published a number of studies as part of the program, and will be summarised. Initial analysis with comparing those miners in 'dusty jobs' as estimated from the measurements of respirable dust showed a statistical significant relationship between bronchitic symptoms and increasing coal dust exposure in a study of 4122 miners (Rae, Walker, & Attfield 1970). This was seen in smokers and non-smokers. There was a clear trend in the younger workers but not in older workers.

Further analysis of this group of miners at the third survey period showed increasing loss of FEV_1 with increasing dust exposure and the loss of FEV_1 was greatest in those with the severest bronchitis symptoms (Rogan et al. 1973). The study importantly showed that there was loss of FEV_1 in miners with no bronchitic symptoms.

Analysis of 1677 miners who had participated in the 2nd to the 4th survey at five collieries, showed that after adjustment for height, smoking, age and difference between collieries, a cumulative exposure to 117ghm^{-3} prior to the survey was associated with a additional loss of FEV_1 of 42ml, but concurrent dust exposure was not significantly associated with a loss of FEV_1 over the 11 year survey period unless colliery differences were ignored (Love & Miller 1982).

A further analysis involving 22 years of follow up on the 4059 miners (of which 2192 had left the industry) showed that in a group of miners the loss of FEV_1 was leading to disability (Soutar & Hurley 1986). Soutar and Hurley showed that cumulative dust exposure resulted in a parallel loss of FEV_1 and FVC in contrast to the normal smoking pattern suggesting a differing underlying pathology. They also showed that overall, the estimated loss of FEV_1 with exposure to respirable coal dust was 0.76ml per ghm^{-3}. This relationship held true for those with out evidence of CWP.

The effect was clearly seen in all groups i.e. smokers, ex-smokers and non-smokers. The effect was greatest in those that had left the industry with symptoms of chronic bronchitis and taken other jobs.

To further assess the risk of clinically significant lung function (i.e. FEV_1 less than 65% and 80% of predicted) an assessment of 3380 men who took part in the 3rd survey having worked for ten years underground were assessed (Marine, Gurr, & Jacobsen 1988). Comparisons were made between smokers and non-smokers. For the highest exposure (348ghm^{-3}) the risk of having FEV_1 less than 65% at age 47 was doubled. For all end points in both smokers and non-smokers there was a doubling of risk with the highest exposures.

Marine and colleagues' research was the subject of argument in the litigation, to a point that it was sent back to the IOM for reanalysis. The IOM confirmed the original findings even when taking into account the colliery differences.

The initial studies on the NSCWP cohort found decrements in FEV_1 when compared to years worked underground (Morgan et al. 1974). However, the trend was inconsistent and no assessment of dust exposures was made. Further study of the 1072 miners from cohort confirmed the loss of FEV_1 with estimated dust exposure but statistical significance was limited due to the size of the cohort (Attfield 1985).

A larger group of 7139 miners was studied in relation to their cumulative dust exposure and lung function. After adjustment for age, height, smoking and region, the estimate for loss of FEV_1 was 0.69ml per ghm^{-3} (Attfield & Hodous 1992).

Other studies have shown larger decrements in lung function (Seixas et al. 1992) although with smaller cohort. Some studies on new miners did not show any association (Seixas et al. 1993). Analysis of miners that had entered the industry prior to 1970 did have an accelerated loss of FEV_1 (Henneberger & Attfield 1997). Similar results have been seen in other groups of coal miners (Carta et al. 1996).

These studies provide consistent evidence that cumulative exposure to respirable coal dust is associated with loss of FEV_1, chronic bronchitis in both smokers and non-smokers and there is a dose response relationship. They also indicate that in a proportion of the group this exposure will result in clinically relevant lung deficits and disability. There are however sources of considerable error in these studies both on the assessment of exposure and health outcomes and assessment of smoking histories.

An analysis of 611 "Black Lung" claimants in the US found that nearly 40% of the ex-smokers had evidence that were continuing to smoke and 91% of those where a smoking history was known were smokers (Lapp, Morgan, & Zaldivar 1994). This could indicate that smoking is a considerable confounder in these results.

These studies have primarily focused on the loss of lung function and *respirable* coal dust. Coggon and Newman Taylor (1998) raised some concern that respirable coal dust may not be the most relevant index of exposure in relation to COPD. *In particular the risk of chronic bronchitis or loss of FEV_1 may be also influenced by larger particles depositing in the bronchi and the use of the wrong metric (i.e respirable dust) may weaken any observed association.*

Chronic Bronchitis and Inhalable Coal Dust

In respect to chronic bronchitis, there have been some studies that support the theory that respirable dust is the wrong metric (Rae, Walker, & Attfield 1970, (Leigh, Wiles, & Glick 1986)

Most studies have assessed dust exposure by counting particles in the 0.5 to 5µm range or used samplers use the BMRC curve with a cut of 7.1µm. It is probable that particles much larger than this range will settle on the bronchi and penetrate into the small airways. According to the ISO 7708 (1995) standard the thoracic convention, has a 50% cut point at 10µm with the cut off for the curve being 30µm. There is going to be a significant mass differential between the thoracic dust and respirable dust. If there is a poor correlation between the two components then misclassification may occur when assessing the outcomes.

Although, there is a good correlation between respirable dust and dust retained in the lungs, a significant portion of larger particles (>7µm) penetrate deep into the lung and are retained. A post mortem study of 60 miners showed that by mass, 55% of the coal particles retained in the lung were between 7-10µm in size and 12.5% were greater than 10µm in size (Xiang and Fu, 1988). By particle count only 4.5% were greater than 7µm.

A post-mortem study of 94 coal miners assessed the signs of bronchitis in relation to smoking and dust exposure. As expected, smoking was significantly correlated with signs of chronic bronchitis. They found a relationship with estimated dust exposure and chronic bronchitis (Douglas, Lamb, & Ruckley 1982).

No relationship was seen between pneumoconiosis or dust retained in the lungs and chronic bronchitis. Retained lung dust was correlated with respirable dust exposure.

The lack of association between retained lung dust and chronic bronchitis would support the hypothesis that chronic bronchitis is related to inhalable dust rather than respirable dust with small particles contributing to pneumoconiosis (and retained dust) and larger particles interacting with the airways mucosa and being rapidly cleared.

The morphological features of coal dust injury in the small airways are fibrotic thickening in the proximal membranous bronchioles and the more distal respiratory bronchioles with distortion and narrowing of the airway (Churg & Wright 1998).

However, emphysema is also correlated with loss of FEV_1 and lung dust content (Leigh et al. 1994). *This would suggest that a number of pathologies occur as a result of exposure to inhalable coal dust and result in airways obstruction*

including emphysema, small airway injury and inflammation and large airway inflammation.

Unfortunately the natural history of chronic bronchitis and COPD in coal miners is poorly understood and the level of disability is still debated (Morgan 1986; Morgan 1999).

The research suggests that chronic bronchitis and obstructive airways disease are separate disorders with the same aetiology (i.e. dust exposure or smoking) (Fletcher 1975).

The presence of airflow limitation is however clinically important, as the presence of airways obstruction as it is more frequently been reported to be associated with disability and increased mortality, whereas chronic bronchitis is not (Peto et al. 1983).

Unfortunately, the authors were unable to find any research assessing the relationship between *inhalable coal dust* and COPD or chronic bronchitis.

This has highlighted a deficit in the current knowledge and previous research about the actual component of the dust (respirable *vs* tracheobronchial *vs* inhalable) that is important in the development of chronic bronchitis, obstructive airways disease, or the clinically significant outcomes including dyspnea, disability and increased mortality.

An estimate of inhalable dust exposure could be determined by time spent in certain occupations, however the strongest associations were seen where direct estimates of respirable dust exposure were made in the studies assessed.

To assess this further analysis should be made between the relationship between respirable and inhalable dust. If there is poor correlation then the question could only be truly answered by an epidemiological study. (Coggon 2005)

The Effect of Reducing Respirable and Inhalable Coal Dust Levels

Further analysis has been conducted by IOM on to assess an appropriate exposure level for respirable coal dust. They re-analysed 7000 coal miners in the PFR surveys (Cowie et al. 1999).

This analysis showed was no clear level of FEV_1 which enabled separation of individuals who reported breathless from those who were not. Overall, however, levels of FEV_1 were lower in those reporting breathlessness. It was only possible, therefore, to define the probability of reporting breathlessness at any given FEV_1 (age and smoking) level.

Cumulative exposure to dust was examined in relation to a 'doubling of the risk' of having a clinically relevant deficit of FEV_1 that is doubling the odds of reporting breathlessness (loss of 993ml of FEV_1). There was a significant association between respirable dust exposure and level of FEV_1 with no evidence of a threshold. An increase in cumulative dust exposure of 50ghm^{-3} was associated with an increase in the order of 1 to 2% of men with clinically relevant deficits.

At a cumulative dust exposure of 120 ghm^{-3} (an average 2 mg/m^3 over 35 years) approximately 30% of 60-year old non-smokers would be expected to have a 'doubling of risk' compared to around 25% of 60-year old non-smokers not exposed to dust. **Reducing the average dust exposure to 1mg/m^3 the corresponding percentage would be about 27.5% of sixty year olds reporting breathlessness, thereby giving a reduction of 2.5% compared to that for an average concentration of 2mg/m^3[d].**

The IOM concluded that *reduction in the workplace standards for respirable coalmine dust would result in a small but significant decrease in the percentage of workers whose lung function was reduced to a level associated with a doubling of the odds of reporting symptoms of breathlessness*.

This research indicates that the dust exposure results in a shifting of the curve with miners losing lung function through other non-occupational factors including age. This shifting of the distribution curve is seen in figure 2. The greater the dust levels the greater the shift and greater numbers of miners reporting breathlessness. And in contrast, increasing controls on dust will have diminishing returns in preventing lung function loss.

[d] This small reduction in outcome with a significant reduction in dust levels highlights some of the weakness in our knowledge about the relationship between respirable coal dust, COPD and breathlessness.

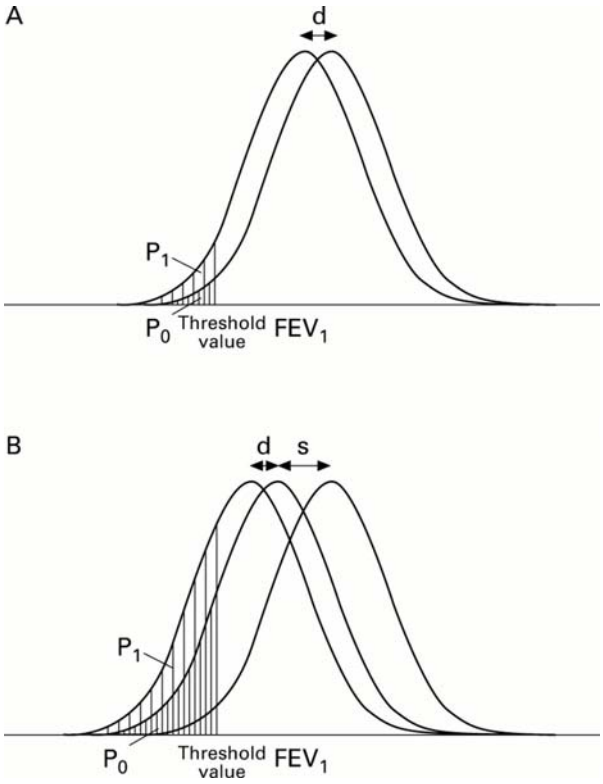

Figure 2 Combined effects of smoking and dust on the risk of FEV_1 From Coggan and Newmann Taylor 1998 [e]

The question remains that if inhalable dust exposure was assessed whether a greater relationship observed. There is some evidence to suggest that inhalable dust may show a stronger relationship (Heederik, de, & Endlich 1994). This study assessed exposure to inhalable and respirable dust (as measured by personal samples), symptoms of bronchitis and lung function in 263 dockworkers and longshoremen. At the dock the product being shipped was by mass 60% of was coal and cokes with 20% phosphate ore, 10% alumina, <5% sulphur and <2% vermiculite.

The study results showed stronger relationships with higher statistical significance between levels of inhalable dust and bronchitis symptoms and loss of lung function in comparison to respirable dust exposure. The differences were

[e] Combined effects of smoking and dust on the risk of FEV1 falling below a specified threshold. It is assumed that FEV1 is normally distributed in the population and that a given cumulative exposure to dust shifts the distribution downwards by a decrement "d". In the absence of smoking (A) the relative risk of having an FEV1 below a threshold value as a consequence of this dust exposure is given by the ratio of areas under the curves (P0 + P1)/P0. If it is assumed that a given cumulative exposure to tobacco smoke causes an additional downward shift in the distribution of FEV1 by a decrement "s", and that the effects of dust and smoking on FEV1 are additive, the position will be as depicted in B. The relative risk of having an FEV1 below the threshold is again given by (P0 + P1)/P0. As age increases, cumulative exposure to tobacco smoke among smokers also increases and so too does the decrement "s" attributable to smoking. The effect is a reduction in the ratio (P0 + P1)/P0. Thus, the relative risk of having an FEV1 below the threshold as a consequence of a given cumulative dust exposure would be expected to decline with age in smokers.

however small and where there was a relationship with inhalable dust, it was generally predicted by respirable dust exposure.

The other aspect for consideration would be the issue of particle size, site of deposition and pathology observed. The loss of FEV_1 is thought to result from the development of centri-acinar emphysema. This occurs in the very small airways and gas exchange region and therefore the particle will have to be at or near the respirable fraction. *For chronic bronchitis and large and small airways injury, the particles will have to be in the size fraction for tracheo-bronchial deposition.*

Therefore it would be appropriate to examine the relationship between the respirable and tracheo-bronchial fraction in coal mines. This is discussed in the next section.

Studies of Coal Dust Fractions and Health Outcomes

The issue of the importance of 'total dust' (as compared to the respirable fraction) in causing upper respiratory disease was addressed by Cowie et al (1981).

They used the dust sampling measurements that were available for the first ten year period of the British PFR research programme. They used direct measures of respirable dust with basic dust measures (particle size and particle number concentration). These were used to derive 'total' and respirable dust concentrations for various working occupations.

Cumulative dust exposures of individual coalface workers were calculated from this data and the time spent in each occupation.

Exposures were examined in relation to fall in FEV1 and prevalence of cough, sputum production recent chest illness and dyspnoea.

Age, smoking and physique were taken into account in the analysis.

During the first ten year period of the PFR research, a standard thermal precipitator (STP) was used initially, with the addition of the MRE Type 113 Gravimetric dust sampler in the later period. The use of both sampling methods enabled mass-number indices to be developed for the occupational groups. This enabled the estimate for equivalent respirable mass concentration between the two measurement methodologies.

Using the particle count with the application of the exponential size distribution, a collection of total dust samples for the STP and gravimetric analysis of the coarser dust collected on the MRE Gravimetric sampler elutriator plates, indices for 'total'/respirable dust were calculated.

This methodology is one of the significant weaknesses of this study as no direct measure of the 'total' or inhalable dust was used.

The researchers then used calculations for 'total' mass number and total volume number indices for exposure to 'total' dust.

The investigations found significant increases in each symptom of 'cough, phlegm, recent chest illness and dyspnoea with exposure to coal dust.

After allowing for the effect of smoking and age, FEV1 was negatively associated with all dust exposure indices.

They found that the best correlation between the health effects were between 'total' volume exposure (TOTV), but this was almost as good as 'total' mass exposure (TOTM) and respirable mass exposure (RSM) but poorer with particle number exposure.

The differences between TOTV, TOTM, and RSM **were so narrow that the authors concluded that 'total dust' offered no advantage, nor that including the coarser fraction gave no advantage in assessing upper respiratory diseases**.

However, as indicated, this study does not have any direct measure of personal inhalable dust exposure. The indices of total dust, total volumes and total respirable mass were highly correlated, therefore could be confounded by the respirable dust measurements.

As this matter had not been fully dealt with, Mark et al (1988) investigated the variability exposure to inhalable dust in the British coal mines. The initial component of their research was to assess the suitability of various sampling heads for measuring inspirable coal dust.

They found that in the conditions experienced in the coal mines (with high wind speeds), none of the available samplers had performed to the ACGIH curve in coal mine conditions. This led them to develop both a static and personal sampler. They also developed a cascade impactor to measure specific components of the inhalable dust.

They then used this equipment to assess personal exposures and compare the various respirable, inspirable and sub fractions of the dust at three collieries, with high rank and low rank coal, and using longwall retreating and longwall advancing methods.

They found that there was good correlation between inspirable coal dust and its sub-fractions including respirable dust and only small variation between the ratios of inspirable to respirable dust across the occupational groups. They found that respirable dust levels were a good predictor of inspirable levels and this could be used to predict levels for occupational groups. Some of their results are illustrated in Table 4 and 5.

Table 4 Relationship between concentration of BMRC respirable sub-fraction and other dust fractions for face workers (Mark et al. 1988)

Colliery		No of Workers	Sub fractions			
			Thoracic	Tracheo-bronchial	Inspirable	Alveolar
Colliery Q- All Face workers	Correlation Slope	33	0.96 2.91	0.94 0.84	0.82 19.79	0.99 0.57
Colliery Y- All Face workers	Correlation Slope	21	0.96 2.85	0.96 0.84	0.83 16.11	0.99 0.57
Colliery F- All Face workers	Correlation Slope	22	0.90 3.12	0.89 0.99	0.63 13.74	0.99 0.59
P value for difference			0.225	0.01	0.024	0.185

Table 5 Relationship between concentration of BMRC respirable sub-fraction and other dust fractions for occupational groups at one colliery (Mark et al. 1988)

Colliery Q Occupational Group		No of Workers	Sub fractions			
			Thoracic	Tracheo-bronchial	Inspirable	Alveolar
Intake Rippers	Correlation Slope	7	0.48	0.02	0.45	0.22
Intake Stablemen	Correlation Slope	6	0.91 0.109	0.90 0.032	0.72 0.034	0.73 0.020
Face Team	Correlation Slope	8	0.95 0.154	0.91 0.044	0.94 0.048	0.92 0.028
Return Stablemen	Correlation Slope	5	0.22 -	0.73 -	--0.97 -	--0.91
Return Rippers	Correlation Slope	7	0.88 0.193	0.74 0.052	0.91 0.088	0.88 0.047
P value for difference			0.008	0.059	0.002	0.010

They concluded that when the collieries were considered separately any dust fraction could be used for correlation with upper airways disease. However, where the collieries were considered together, only one of the coarse fractions, the thoracic, had a relationship with the BMRC respirable dust fraction *and for the two other fractions, inspirable and tracheobronchial, there are significant differences between the collieries.*

The main limitation with this study is the small numbers of samples collected and with the variation between mines, there would be little ability to generalise to other mines or countries without further research.

Burkhart and colleagues (1987) measured particle mass vs aerodynamic diameter in 10 underground coal mines, using cascade impactors capable of operating with personal sampling pumps. These were located within 36 inches (914.4 mm) of the operator's breathing zone[f] as prescribed in the US dust sampling regulation 30 CFR 70. Their results showed a particle size distribution for all areas with the exception of the feeder/breaker, shared a primary size mode of about 17-20 µm. In addition, all areas, except for the continuous miner and the feeder/breaker showed a secondary size mode of about 5–8 µm. This bimodal distribution was maintained throughout the 10 mines studied and it was considered that this could be attributed to the action of the continuous miner.

Burkhart and his colleagues felt that this information would be useful in determining the most appropriate control techniques for reducing miner's dust exposures; they also suggested that the size distribution information is essential in determining the accuracy of instruments used to assess compliance with dust standards. Almost as a secondary consideration, they suggested that knowledge of the particle size distribution could provide information on tracheo-bronchial as well as respirable dust levels. This information, they suggested, together with pulmonary function data could be used to gain a better insight into the entire spectrum of coal mine dust-induced lung diseases.

Potts et al (1990) measured thoracic dust concentrations (ACGIH definition) on longwall and continuous mining sections. They found that Shearer cutting generated between 28 and 84 mg/m^3 of thoracic dust and accounted for 75% of the total thoracic dust generated on the sections employing unidirectional cuts. A bidirectional cut sequence and manual roof support advancement represented the worst case scenario for thoracic dust exposure because personnel are required to work downwind of the shearer. Supports were also a significant source of dust. Support advancement generated between 13 and 16 mg/m^3 of thoracic dust and accounted for 22% of the total thoracic dust generated on the sections employing unidirectional cuts. The mass median aerodynamic diameters of dusts contributed by the intake airway, beltway, crusher and shearer were 7.5, 9.1, 12 and 18 µm respectively. The mass median diameter of the support generated dust was greater than 21 µm. It was noted that in general, as the mass median diameter increased, the thoracic/respirable exposure ratio increased. They also observed that the mass median particle diameter of a dust cloud decreased as it moved further from the source, as the larger particles tend to settle out of the airstream at a much faster rate than smaller particles.

[f] It is worth noting that in contrast for Australian personal sampling, AS3640 defines Breathing Zone as A hemisphere of 300 mm radius extending in front of the face and measured from the midpoint of a line joining the ears.

Potts and his colleagues also calculated the thoracic/respirable dust exposure ratios. These are detailed in Table 5.

Table 6. Thoracic and Respirable Dust Ratios by location

Location in Longwall	Thoracic/respirable dust exposure ratio	Coefficient of variation
Intake airway	1.8	8
Beltway	2.7	17
Headgate	3.1	23
Downwind of support & panline advancement activities	6.7	16
Tailgate area	5.4	7

On this basis, Potts et al. concluded that compliance with the respirable dust standard would not equally limit the thoracic dust exposures of all longwall face workers.

It could be argued that a weakness of Potts et al.'s methodology is that only area sampling was used and the generalisation to personal sampling is difficult. The area sampling may increase the variability in the thoracic fraction and the ratio to respirable dust, due to larger particles settling out through deposition, the further the sampler is located from the face. Area samplers do not take into account personal dust clouds and dust generation from worker activities where coarse particles are re-suspended.

Coal Services (2005) undertook similar research in NSW coal mines in 2002-2003. This has been provided to and analysed by the authors.

A summary of the data is presented in table 7 and represents matched inhalable and respirable dust samples collected side by side. The samples were collected as static samples, rather than personal samples and therefore are representative of the workplace atmosphere, rather than individual exposures.

The coefficient of correlation for all samples is r = 0.83, which is surprisingly high, when considered against other examples in the literature. This compares with a correlation coefficient of 0.351 from McFadden's data, which is discussed further. This data is supportive of the work by Potts et al. although it does highlight some variability in the inhalable dust levels.

Table 7 Inspirable and Respirable Longwall Static Dust Sample Results (Coal Services 2005)

	Respirable #110 Chock	Inhal #110 Chock	Respirable #165 Chock	Inhal #165 Chock	Respirable #55 Chock	Inhal #55 Chock	Respirable Crusher	Inhal Crusher	Respirable Sivad Controller	Inhal Sivad Controller
n	11	11	11	11	12	12	12	12	12	12
Mean	3.027	18.082	3.991	26.118	1.633	14.133	0.533	3.925	0.375	0.983
Ratio of means	5.973		6.544		7.066		7.359		2.622	
Std Dev	1.585	12.951	4.956	18.358	0.701	12.957	0.215	3.649	0.122	0.434
Geo Mean	2.694	14.251	3.495	21.511	1.501	10.605	0.492	2.948	0.358	0.881
Correlation	0.830		0.797		0.475		0.354		0.301	
Min	1.4	3.7	0.7	9.6	0.7	5.4	0.3	1	0.2	0.3
Max	6.2	45.1	6.5	64.4	3	41.5	0.8	14.1	0.6	1.8

The important work by Potts led Seixas et al. (1995) to follow up with a study to better characterise the personal particle size distributions of exposures received by underground coal miners and if possible, to test the hypothesis that tracheo-bronchial exposures might be specifically associated with the development of chronic obstructive lung disease in miners. To do this, particle size distributions in underground mining jobs at four mines were assessed so that the job-specific respirable and tracheo-bronchial fractions could be estimated and used to convert the historical respirable dust measures into estimates of tracheo-bronchial dust exposures. The intention was to then examine the relationship between the newly estimated tracheo-bronchial dust exposures and respiratory outcomes in coal miners to see whether predictive power was increased over models using respirable dust estimates only.

Samples were collected using Andersen 8-stage personal cascade impactor samplers. Interestingly, non-face workers wore their samplers portal to portal, while face workers donned theirs in the mine, which suggests crib-to-crib monitoring for samplers which were expected to return higher dust loadings.
It was found that there were no significant differences when the samples were grouped by occupation, by proximity of work to the coal face, or by the type of mining technology in use. They concluded that although the tracheo-bronchial dust fraction may contribute to the development of obstructive lung disease, occupation specific tracheo-bronchial dust fractions are not likely to produce stronger exposure-response estimates than the historically collected respirable dust concentrations.

Seixas et al. compared their results to those of other workers, including Potts (1990). The results for longwall shearer, downwind of roof support movement, and continuous mine roof bolter in Potts' study showed similar distributions. The extreme values in the intake air and downwind of roof support were not supported and Sexias indicates these are not personal exposures.

Seixas et al. findings support the findings of Mark and his colleagues (1988) that there is high level of correlation between respirable dust and other particle sub-fractions of dust exposures and they concluded that respirable dust estimates should be equally predictive of obstructive lung disease as other sub-fractions of inhalable dust.

This authors are of the opinion that there is reasonable, but by no means conclusive evidence, that respirable dust is correlated to the tracheo-bronchial fraction. To the author's knowledge, there has not been any similar work done in Australian coal mines.

Inhalable and Respirable Dust Levels in NSW Coal Mines

Coal Services provided the authors a collection of 406 paired samples of respirable and inhalable dust data collected from 3 BHP Billiton coal mines (Appin, Elouera, and Westcliff) and 2 coal processing plants in NSW as part of a Coal Services Health & Safety Trust project (McFadden 2004). These are longwall mines with the Appin and Westcliff mines, mining the Bulli seams and the Elouera mine mining the Wongawilli seam.

This data was collected from 19 January 2003 until 30 May 2003. The data had been divided into 30 different HEG groups that had numbers of 5 to 23 in each group. The authors felt these groups were too small meaningful so the occupational groups for the mines were combined for most analyses. Using the occupational categories there were 21 occupational groups with numbers of 5 to 63. 5 cases were excluded due to missing data. The purpose of reviewing the data was to assess whether there was:

1. Significantly high levels of inhalable dust in the occupational groups or specific mines.
2. Assess whether there was any correlation between respirable dust levels and inhalable dusts levels, and if so could respirable dust levels be used to predict inhalable dust levels.
3. Assess whether there was significant variability between the mines in or the specific occupational groups.

Statistical Methods

The aim of reviewing the data provided was to assess the relationship and variability in the inhalable and respirable dust levels between the mines and occupational groups. The number, arithmetic mean, geometric mean, standard deviation and minimum and maximum values were determined for the occupational groups and mines. Box-and-whisker plots of the inhalable and respirable coal dust exposures for the occupational groups and mines to present mean values and quartiles.. The correlation statistics (Pearson's Coefficient) were calculated and the variance of the means was assessed with the one-way ANOVA test. To negate the effect of the skewed data natural logarithmic conversion was performed.

The data was imported from Excel worksheets and analysis was conducted with the SPSS 13.0 for Windows statistical package.

Results

The raw data has been presented in scatterplot in figure 3. As can be seen there is no correlation between the respirable dust and inhalable dust. (Pearson correlation coefficient 0.079) There are also a number of extreme variables with respirable dust levels of up to 16 mg/m^3 and inhalable levels of up to 490mg/m^3.

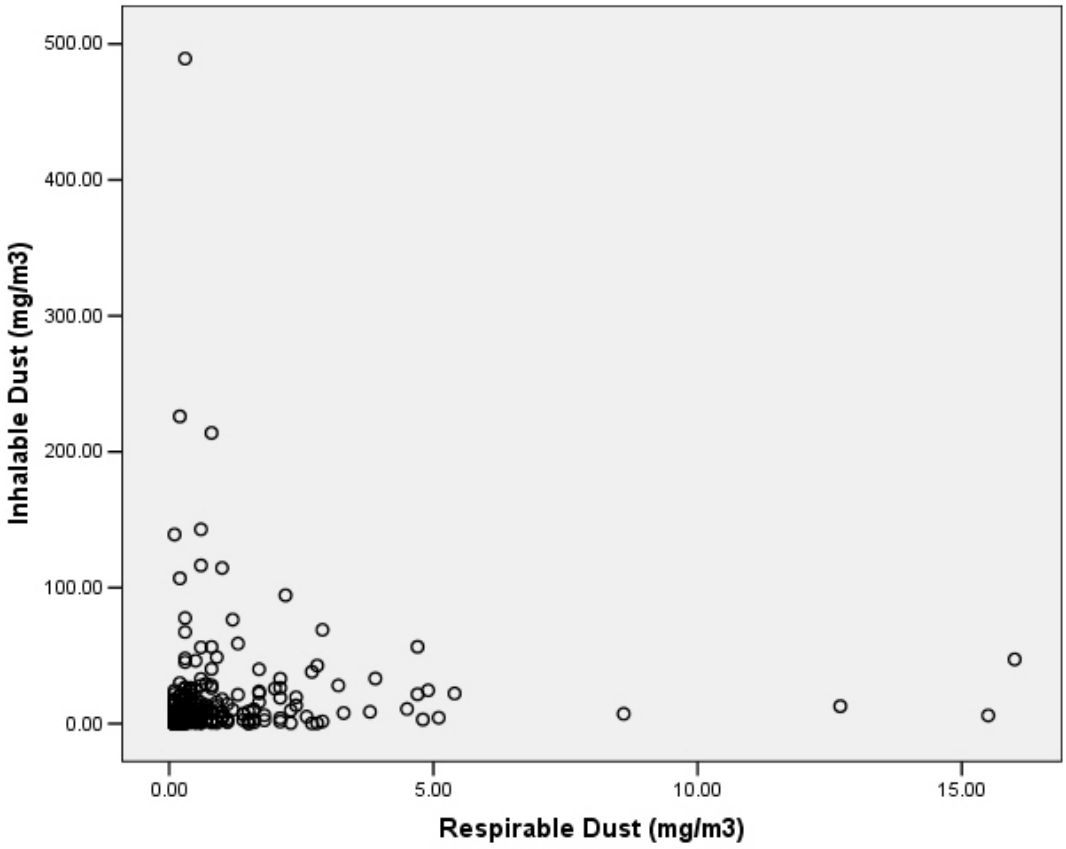

Figure 3 Scatterplot of Respirable Dust versus Inhalable Dust.

As there were only six respirable samples above 5mg/m^3 and eight inhalable samples above 100mg/m^3. It was decided to exclude these from the analysis as the authors considered the results quite high, with results less than 5mg/m^3 and 100mg/m^3 generally being reported in other studies. In addition to these, unpaired samples (i.e. where either a respirable or an inhalable sample was lost), were also excluded from the analysis. The results without the outliers are provided in the scatterplot graph provided figure 4.

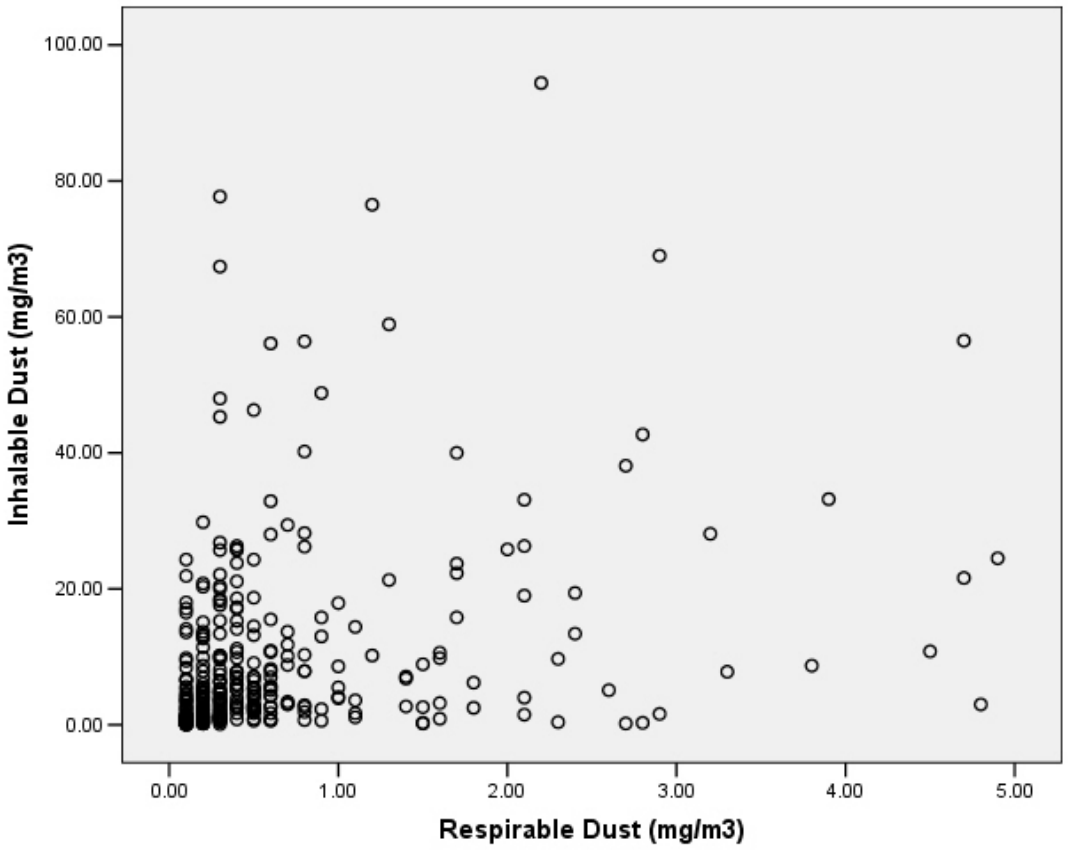

Figure 4. Scatterplot graph of Respirable Dust versus Inhalable Dust without the Outliers.

As can be seen there is still little correlation between respirable dust and inhalable dust with the correlation coefficient being 0.351. (Significant at $p<0.01$).

The low correlation differs from that reported by Mark et al. (1988). This is difficult to explain, but may be due to a number of factors. These possibly include higher air velocities and ventilation rates (i.e due to methane) in Australian mines, differing temperatures and humidity, and differing cutting tools and mining machinery that could result in varying levels of coarse particulate.

There was large variation seen in the occupational groups and between the underground mines and coal processing plants. The mean levels for the occupational groups are presented in Table 6. The respirable and inhalable dust results for the occupational groups are presented in a box and whisker plot in figures 5 and 6.

Table 8 Mean Inhalable and Respirable Dust Levels by Occupation

HEG Composition	Statistic	Respirable Dust (mg/m3)	Inhalable Dust (mg/m3)	Ratio inhalable/Respirable
Belts	Mean	.30	4.53	26.70
N= 17	Geometric Mean	0.23	3.01	13.21
	Std. Deviation	.27	4.42	42.99
Control Room	Mean	.16	.34	2.57
N= 5	Geometric Mean	0.14	1.26	1.80
	Std. Deviation	.089	.25	1.76
Day Maintenance	Mean	.30	1.86	13.03
N=9	Geometric Mean	0.18	1.19	6.68
	Std. Deviation	.45	2.10	12.80
Dyke	Mean	.58	13.62	52.54
N=5	Geometric Mean	0.44	10.68	24.52
	Std. Deviation	.50	10.11	59.96
Gen. Underground	Mean	.40	5.72	24.98
N=17	Geometric Mean	0.33	4.22	12.83
	Std. Deviation	.308	4.68	33.91
Haulage Operators	Mean	.11	.35	3.23
N=21	Geometric Mean	0.11	0.32	2.86
	Std. Deviation	.035	.188	1.86
Longwall	Mean	1.07	14.39	26.54
N=50	Geometric Mean	0.68	11.18	16.33
	Std. Deviation	1.15	11.97	38.78
Management	Mean	.116	1.38	13.25
N=6	Geometric Mean	0.11	0.66	5.90
	Std. Deviation	.041	2.10	21.26
Materials Handling	Mean	.1571	6.79	23.95
N=7	Geometric Mean	0.14	0.66	4.79
	Std. Deviation	.098	16.98	56.03
Methane Drillers	Mean	.42	3.96	14.99
N=23	Geometric Mean	0.32	2.93	9.29
	Std. Deviation	.35	3.13	17.90
Mobile Equipment Op	Mean	.2962	.8192	4.65
N=26	Geometric Mean	0.19	0.56	2.98
	Std. Deviation	.51	1.00	4.36
Operators	Mean	.29	4.34	17.70
N=9	Geometric Mean	0.22	1.64	7.31
	Std. Deviation	.231	6.90	22.98
Outbye Workers	Mean	.85	9.79	30.41
N=36	Geometric Mean	0.59	7.20	12.21
	Std. Deviation	.94	10.39	47.77

HEG Composition	Statistic	Respirable Dust (mg/m3)	Inhalable Dust (mg/m3)	Ratio inhalable/Respirable
Panel	Mean	.71	12.41	25.38
N=62	Geometric Mean	0.50	9.09	18.13
	Std. Deviation	.82	10.01	21.96
Shift Operators	Mean	.4471	3.22	24.05
N=17	Geometric Mean	0.22	1.45	6.57
	Std. Deviation	.69	4.23	43.35
Special Groups	Mean	1.19	8.23	20.27
N=8	Geometric Mean	0.72	6.37	8.76
	Std. Deviation	1.20	6.58	34.42
Surface Workers	Mean	.42	1.1	6.63
N=28	Geometric Mean	0.22	0.83	3.81
	Std. Deviation	.625	1.073	7.73
Tailgate Workers	Mean	.34	8.12	26.90
N=5	Geometric Mean	0.33	3.53	10.62
N=6	Std. Deviation	.089	10.55	35.30
Transport Operators	Mean	.30	5.26	17.65
	Geometric Mean	0.27	2.65	9.95
	Std. Deviation	.14	7.82	19.37
Workshop Personnel	Mean	.14	1.90	18.13
N=14	Geometric Mean	0.11	0.76	6.44
	Std. Deviation	.134	3.01	30.43
Yard Personnel	Mean	.10	4.37	43.67
N=6	Geometric Mean	0.10	0.83	8.27
	Std. Deviation	.00	6.60	66.03
Total	Mean	.54	6.88	20.47
	Geometric Mean	0.32	2.87	9.03
N=377	Std. Deviation	.76	9.19	32.55

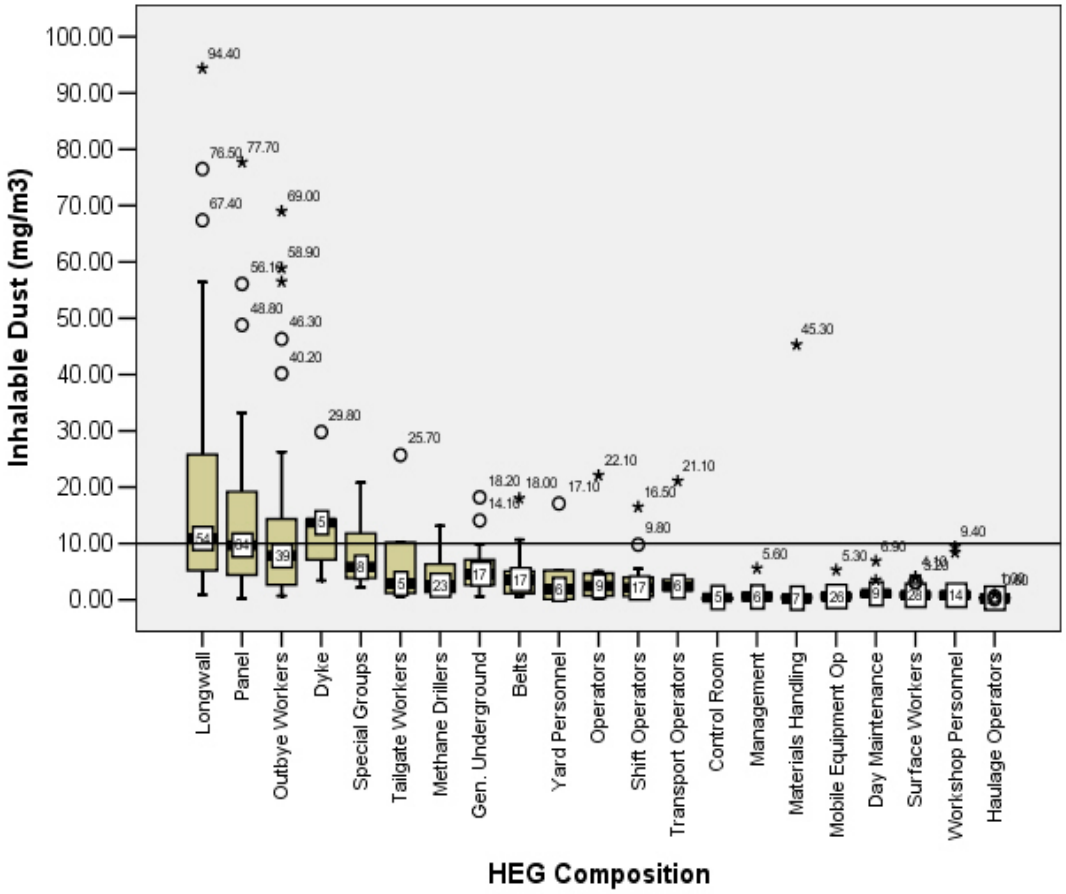

Figure 5. Box and Whisker Plot for Inhalable Dust

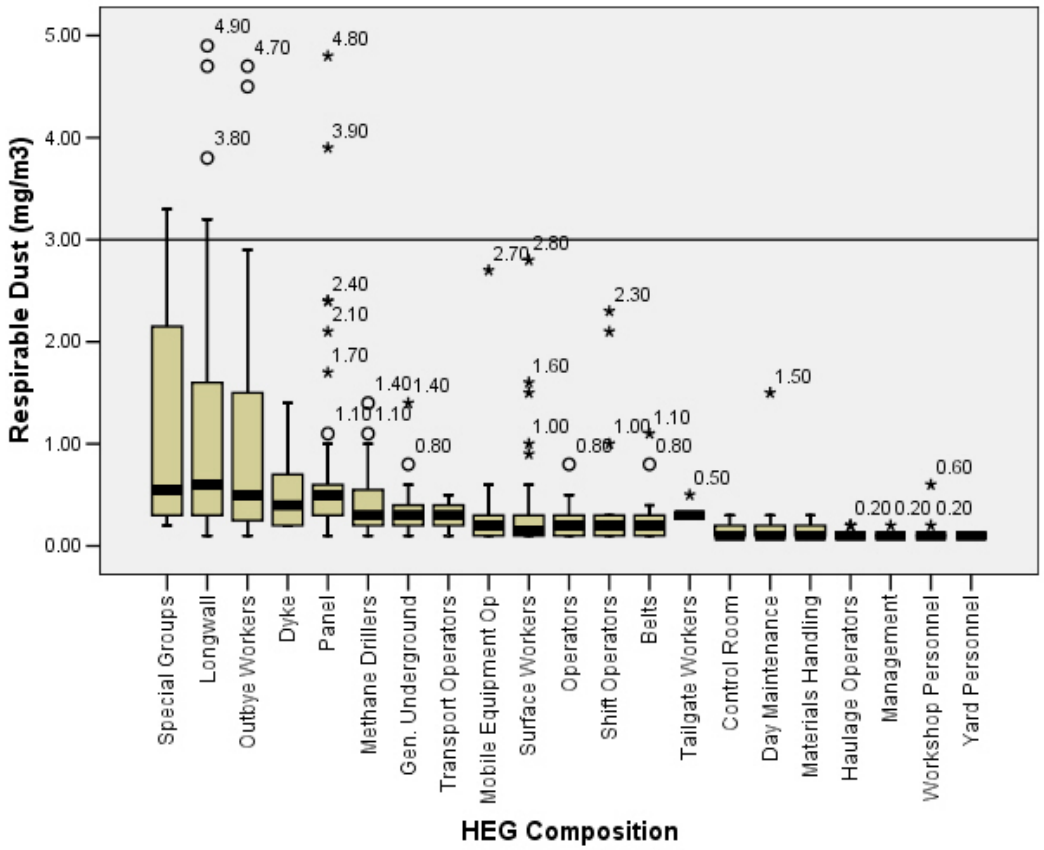

Figure 6. Box and Whisker Plot for Respirable Dust

As can be seen in figure 5 there is considerable variability in some of occupational groups for inhalable dust. Similar variability is seen in the respirable dust levels in these groups, however most of the respirable dust results are well below the TWA exposure standard of 3mg/m^3 and for all occupational groups the geometric means were all below 1mg/m^3.

In the longwall, panel and outbye occupational groups a significant portion of the inhalable results are above the level of 10mg/m^3. In comparison there is less variation between the data for respirable dust with only a small proportion of the results are above the respirable dust standard of 3mg/m^3 the geometric means are 0.6-0.7mg/m^3.

For the Coal Processing Plant occupations, the results indicate that the inhalable dust levels are low and respirable dust levels are very low.

The results for each mine and coal processing plant (CPP) are presented in figure 5 (inhalable dust) and figure 6 (respirable dust). The highest geometric mean levels for inhalable dust at the CPPs were 1.45 mg/m^3 for the Dendrobium CPP operators and 1.65 mg/m^3 for the West Cliff CPP. Therefore the results for the CPPs were removed from further analysis.

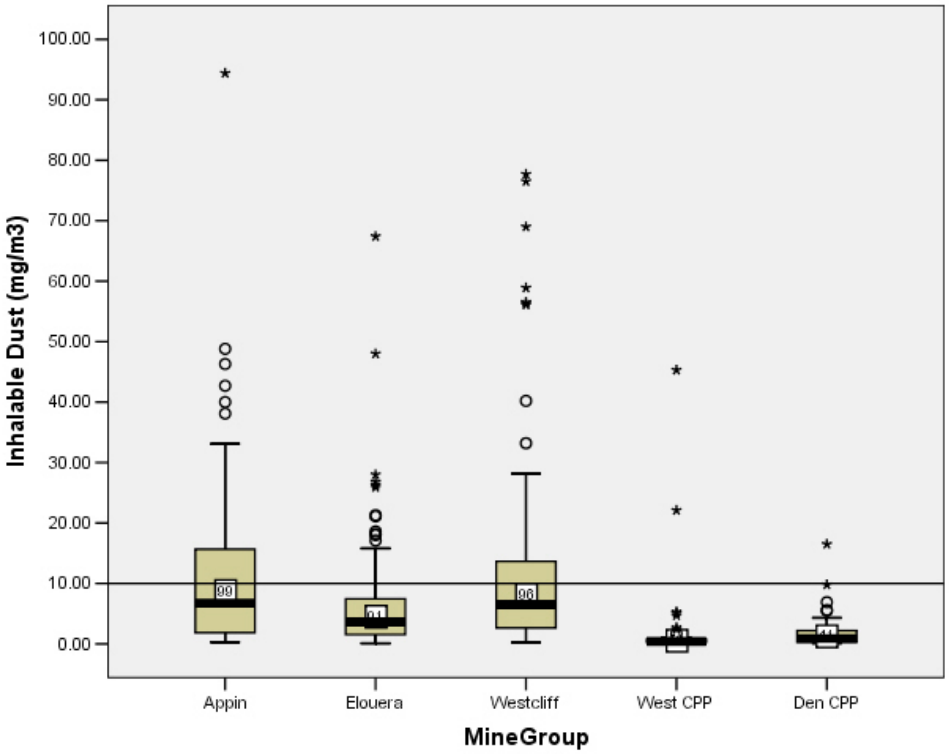

Figure 7. Box and Whisker Plot for Underground Mines and Coal Processing Plants -Inhalable Dust

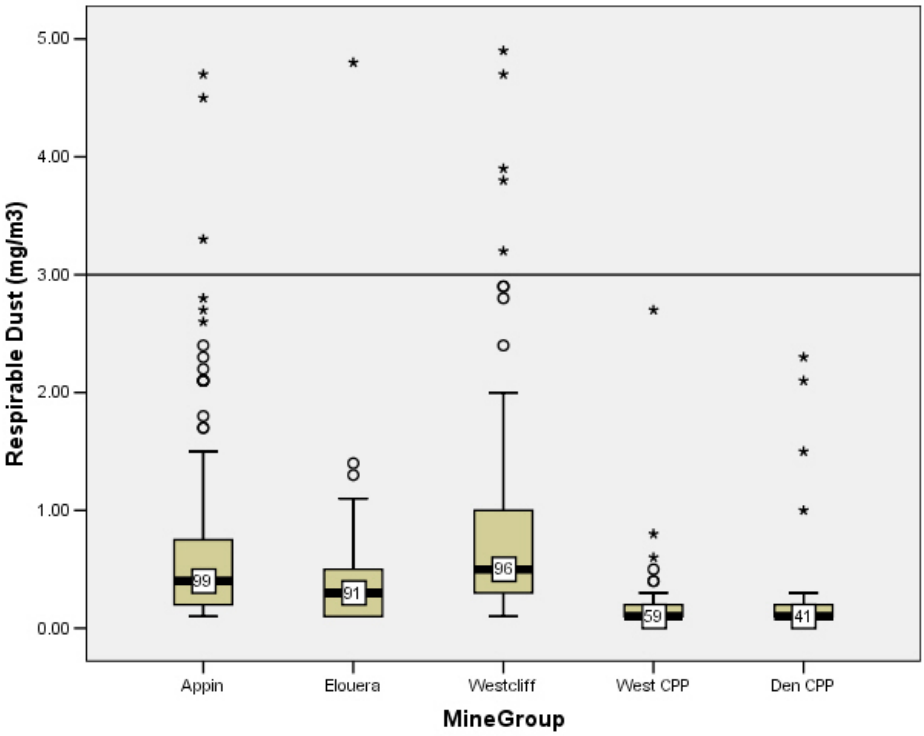

Figure 8 Respirable Dust Levels at the Coal Mines and Coal Processing Plants

This left the results for the three underground mines Appin, Elouera and Westcliff mines. These results were analysed to assess whether there was any correlation between the inhalable and respirable dust results both in each mine and with each occupational group.

Again further analysis found no strong correlation was found between respirable and inhalable dust in the mines (0.344, sig at P<0.05 for Appin, 0.097 for Elouera and 0.354, sig at P<0.05 for Westcliff) or occupational groups.

The best correlation for the occupational groups was at WestCliff for outbye workers at 0.53 and for longwall miners at Appin at 0.43. All other results showed a poor correlation.

Additionally the differences were assessed for mean respirable and inhalable dust exposures between the mines. Overall there was a significant differences in mean dust levels for respirable dust and inhalable levels.

Table 9. Respirable and Inhalable Dust Levels at the three underground mines

Mine	Statistic	Respirable Dust (mg/m3)	Inhalable Dust (mg/m3)
Appin	Mean	0.75	11.57
N=99	Std. Deviation	0.92	14.09
	Geometric Mean	0.44	5.64
Elouera	Mean	0.39	7.15
N=91	Geometric Mean	0.27	3.14
	Std. Deviation	0.54	10.3
Westcliff	Mean	0.88	12.66
N=96	Geometric Mean	0.54	5.90
	Std. Deviation	0.99	16.93
Total	Mean	0.68	10.53
N=286	Geometric Mean	0.40	4.75
	Std. Deviation	0.87	14.24

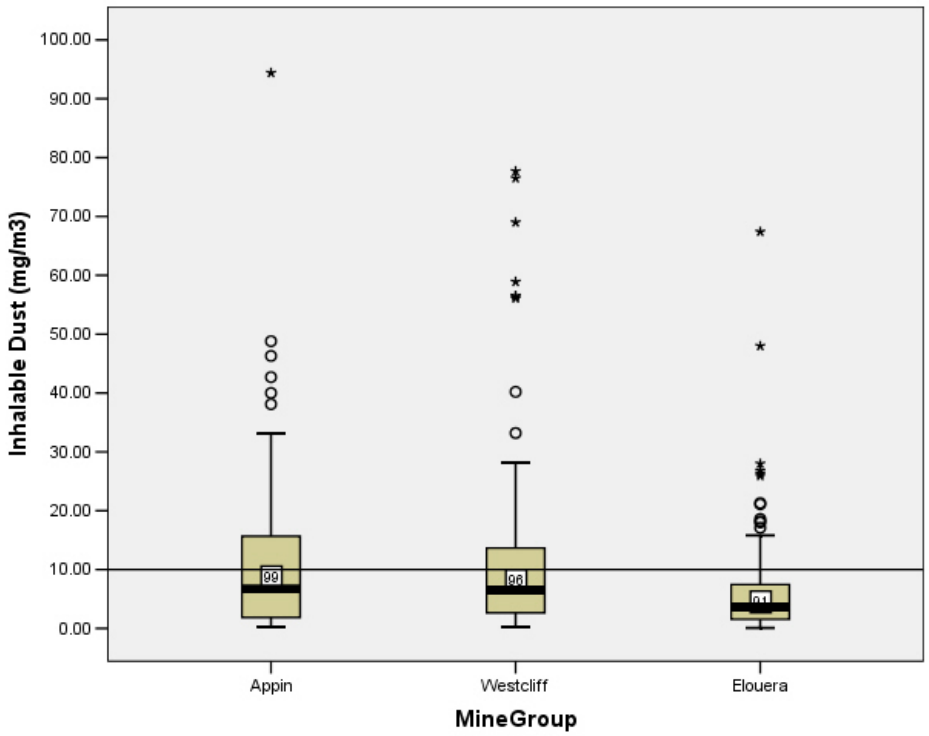

Figure 9. Box and Whisker Plot for Inhalable Dust at Underground Longwall Mines without outliers.

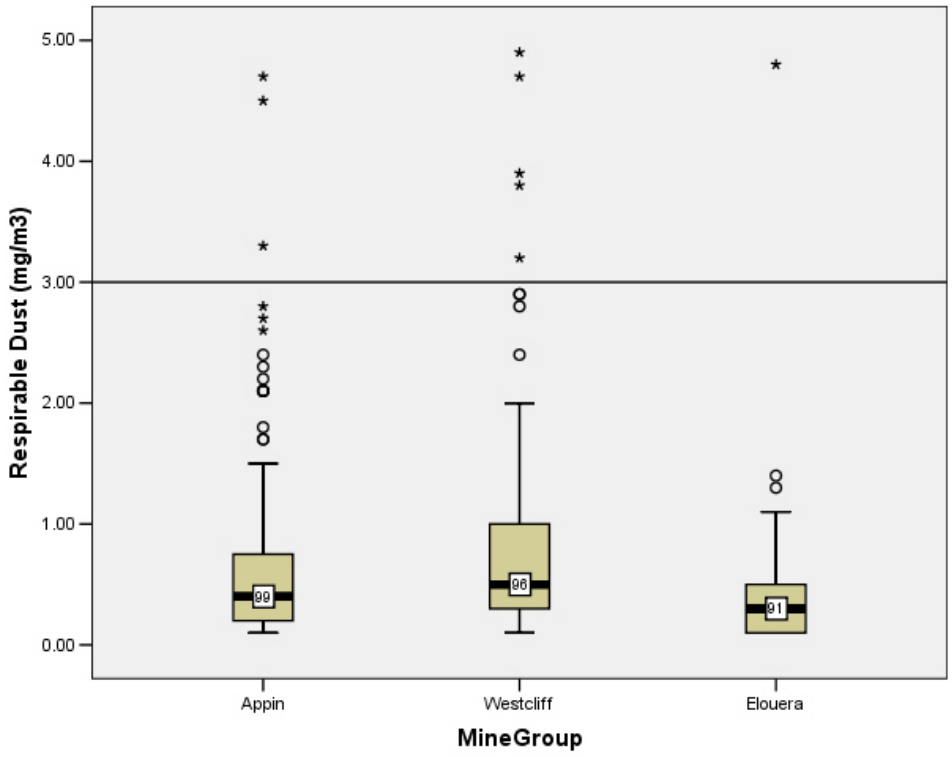

Figure 10. Box and Whisker Plot for Respirable Dust at Underground Longwall Mines without outliers.

As the difference in mean inhalable and respirable dust levels could be explained by having different tasks and occupational groups, the two occupational groups common to all three mines (longwall and panel workers) were analysed separately with comparison between the two groups.

Overall, again there was no significant correlation between inhalable and respirable dust levels in the panel and longwall miners when analysed separately or together.

Table 10 Correlation between inhalable and respirable coal dust in occupational group - longwall miner.

		Respirable Dust (mg/m3)
Inhalable Dust (mg/m3)	Pearson Correlation	.270(*)
	Sig. (1-tailed)	.024
	N	54

* Correlation is significant at the 0.05 level (1-tailed).

Table 11 Correlation between inhalable and respirable coal dust in occupational group - panel miner.

		Respirable Dust (mg/m3)
Inhalable Dust (mg/m3)	Pearson Correlation	.110
	Sig. (1-tailed)	.193
	N	64

There was a significant difference in the mean inhalable dust levels for the panel workers at Elouera when compared to the Westcliff Panel workers (mean difference 12mg/m^3 significant at $P<0.05$).

This may be due to a number of reasons including positioning of the panel miners at each mine in relation to the face, mining equipment or different dust suppression methods. It does suggest that there can be significant variability in inhalable dust levels in similar occupations between mines. This however would have to be clarified further.

The relevance of the ratio between the inhalable and respirable dust levels was investigated further. However, as there was such poor correlation between the two variables and wide rages of the ratio, the usefulness of the ratio was found to be poor in predicting inhalable dust levels.

Assessment of the Inhalable and Respirable Dust Data for NSW.

Given that there was no correlation between the respirable and inhalable dust levels both within the mines and occupational groups, the authors conclude that the respirable dust levels will not predict the inhalable dust exposure, either at the mines or within occupational groups.

The data suggest that inhalable dust levels are unlikely to be relevant in coal processing plants where dust levels are low. The inhalable dust levels are unlikely to add any further useful data in these plants.

In underground longwall mines, inhalable dust levels are significant. In particular a majority of face-occupations will experience inhalable dust levels of greater than 5mg/m^3 and a significant number (~30%) have levels greater than 10mg/m^3.

There is considerable variability in the inhalable dust levels with levels ranging between 0.1 mg/m^3 to 489 mg/m^3. Greatest levels are seen in longwall, panels and outbye workers. Geometric mean levels in the occupational groups range from 7 to 11mg/m^3 of inhalable coal dust.

The relevance or significance of these high levels is open to question. No doubt high levels will reduce visibility, potentially produce mechanical irritation and reduce job satisfaction.

Furthermore, the potential health effects from these levels is unclear, if the respirable levels are well below the acceptable standard.

This issue requires further research, to assess whether these occupations that are experiencing high levels are at increased risk. With these occupations being close to the face, it is possible that the samplers are collecting very coarse particles of high mass, (> 50μm) that will make the gravimetric result very high but not result is the same health effect as a large number of small particles that deposit in the tracheo-bronchial region.

To assess this further, initial investigations should include laser particle sizing of the inhalable dust samples from workers at the face away from the face. Further analysis of the mass fractions could be done with a cascade impactor or through assessing the aerodynamic diameter of each mass fraction.

Analysis of other factors such as humidity, air velocity, dust suppression methods, production rates, effect of shield advance and visibility is necessary.

The question remains whether inhalable dust is a suitable metric for coal mining.

However, at present, the authors must also consider whether the lack of any inhalable dust standard will result in any adverse health outcomes. Certainly, it

can be asserted that inhalable coal dust (or the tracheo-bronchial fraction) will be associated with chronic bronchitis and probably COPD.

The authors believe however, that it is best practice to keep any atmospheric contaminants at the lowest reasonably practicably achievable levels.

Where dust levels are high and at a level that could result in possible adverse effects the mine should implement appropriate controls. The authors understand that is common practice that most coal miners wear disposable particulate respirators.

If no effects are reported in the literature with respect to inhalable coal dust, are there any comparisons that can be made to other inhalable dusts?

Coal is a heterogeneous carbonaceous rock formed by elevated temperature and pressure in the earth's crust, and includes a wide range of heavy minerals, including carbon, clays, carbonates, sulphide ores, oxide ores, quartz, phosphates, metals and a range of organic chemicals, such as methane, benzene, phenols, naphthalenes, acenaphthalenes, and 3,4 and 5 poly-cyclic compounds.

The authors considered a number of substances that may have prove useful comparisons for inhalable coal dust. These included talc, mica, graphite, carbon black, wood dusts.

The authors excluded many substances have exposure standards as they have differing toxicology to coal dust and generally can be placed in the following three classes:

- Highly soluble materials which can quickly enter the blood and exhibit their toxicity; e.g. nicotine and soluble salts;
- Materials which can exhibit toxicity after dissolving in the gastro-intestinal tract , e.g. toxic metals; and,
- Materials which can exhibit toxicity at the deposition site, e.g. acids and nasal carcinogens. (AS 3640:2004)

The authors assessed potentially similar substances in relation to their typical particle size, physio-chemical properties, toxicity, presence of other contaminants (silica, PAHs) and reported health effects.

However, given the unique physio-chemical composition of coal, and observed health effects in relation to respirable coal dust to date the authors have not identified any relevant comparisons and conclude that no useful comparisons could be made.

In most circumstances, the effect of other mineral dusts is impacted by the significant component of silica.

The toxicity of coal dust (cytotoxicity, induction of inflammatory mediators, presence of stable radicals and induction of reactive oxygen species) cannot be explained by the presence of silica alone. Coal dust is not a benign or nuisance dust, it is a complex mixture of non-organic and organic minerals.

Its toxicity has been comprehensively reviewed by Schins and Borm (1999). They summarised that it is well recognised that inhaled coal dust particles are important non-cellular and cellular sources of reactive oxygen species (ROS) in the lung, and may be significantly involved in the damage of lung target cells as well as important macromolecules including α1-antitrypsin and DNA.

In vitro and in vivo studies with coal dusts showed the up-regulation of important leukocyte recruiting factors, e.g. Leukotriene-B4 (LTB4), Platelet Derived Growth Factor (PDGF), Monocyte Chemotactic Protein-1 (MCP-1), and Tumor Necrosis Factor-(TNF), as well as the neutrophil adhesion factor Intercellular Adhesion Molecule-1(ICAM-1).

Coal dust particles are also known to stimulate the (macrophage) production of various factors with potential capacity to modulate lung cells and/or extracellular matrix, including O_2^-, HO_2^-, and $NO^-\cdot$, fibroblast chemoattractants (e.g. Transforming Growth Factor-ß (TGFß), PDGF, and fibronectin) and a number of factors that have been shown to stimulate and/or inhibit fibroblast growth or collagen production such as (TNF, TGFß, PDGF, Insulin Like Growth Factor, and Prostaglandin-E2.)

AS 3640 states that for dusts which have a toxic effects if absorbed in the nasopharyngeal region, or which may exert toxic effects if ingested after deposition in the nasopharyngeal region, it is appropriate to measure inhalable dust. This applies whether or not these dusts also have a toxic effect on the lungs or are toxic if absorbed in the lungs or are swallowed.

Coal dust does not meet these criteria.

What is a suitable workplace exposure standard for inhalable coal dust and what degree of protection does it provide for specific health outcomes?

In Australia, NOHSC (1995) recommends that where no specific exposure standard has been assigned and the substance is both of inherently low toxicity and free from toxic impurities, the recommended exposure standard for dust in general should be 10 mg/m^3, measured as inspirable dust. The proposal in NSW to adopt a standard of 10 mg/m^3 appears to follow this convention.

To examine the relevancy of a standard for inhalable dust and the applicability of the limit of 10mg/m^3, the authors have examined the basis for this limit of 10mg/m^3. This limit is generally applied to 'particulates not otherwise classified or regulated' (PNOC/R) as recommended by NOHSC. (NOHSC:3008, 1995). NOHSC goes onto comment that compliance with this limit (provided that the airborne particulate does not contain any other hazardous components) should prevent against any impairment of respiratory function even over many years of exposure.

The NOHSC guideline indicates that this should not be used if there is greater then 1% silica or other toxic substances present.

The view that exposure to PNOC/R for many years will not result in significant impairment of respiratory function, is generally supported by Guidotti (1998) in his review of the issue. Guidotti cautions, however, that PNOC/R can be made up of three more pathogenic sub groups –

-Antigenic dusts
-Toxic dusts
-Biological inert dusts.

Antigenic dusts are those with a potential to provoke an immune response. Dusts of plant and animal origin, but because they are PNOC/R they are unlikely to be potently antigenic or they would be identified by now. Toxic dusts that may be in the PNOC/R if they are of low toxicity but can exert a more toxic effect if delivered in a mode that would increase toxicity such as ultra fine dusts.

Biologically inert dusts are those without specific pulmonary toxicity and antigenicity. Nevertheless they will act as foreign bodies provoking a non-specific pulmonary response such as macrophage overload. The reactions to these dusts will be generally in response to particle number, size and surface area.

With these biologically inert particles, preventing overload in the alveolar region should prevent against the development of sustained inflammation and adverse health outcomes.

To support his view, Guidotti (1998) draws attention to a study of steel workers exposed to dusty jobs (Wang et al. 1996). The investigators found that a decline in FEV_1 was closely associated to age, smoking and weight gain. Dust exposure was associated with a 9.3ml loss of FEV_1, 6.4ml loss of FVC and 0.1% of FEV_1/FVC per year of employment. In comparison to the average national decline with age this is very small and is unlikely to account by itself for any clinically significant airflow obstruction.

Wang et al (1996) also found obesity was associated with loss of FEV_1 (4.7ml/lb/yr) and FVC(6.3ml/lb/yr). This adds another factor to be considered when assessing the literature on dust exposure and lung function and may have been a source of confounding in previous research.

It is noted that high levels of a nuisance can cause irreversible changes to the airways from overload, and consequently present more than an irritant effect (Soutar et al. 1997).

The history of the PNOC/R standard has been comprehensively reviewed and by Hearl (1998).

The PNOC/R (originally termed nuisances dust) standard was developed in the 1940s by the ACGIH as part of a list of Threshold Limit Values (TLV)®. It was based on Public Health Service studies from the 1920s and 1930s and the TLV for nuisance dust was set at 50mppcf until 1962. As the techniques for the analysis of silica improved the ACGIH introduced a new formula for dust containing silica –

$$TLV = \frac{250\text{mppcf}}{\%\text{quartz} + 5},$$

providing a smooth transition of the range of dusts from nuisance dust with a limit of 50mppcf.

Following the Johannesburg Pneumoconiosis Conference in 1959, effort was made to introduce size selective mass based standards. In 1964 there was a transition arrangement where a TLV of 15mg/m^3 or 50mppcf was introduced. During the transactions of the ACGIH in 1970, it was noted the TLV for nuisance dust had not been reduced for some years and a new standard of 10mg/m^3 was recommended

The justification for the new standard for the PNOC/R limit was based on reduction of visibility and the irritant effect on skin, ears, and mucous membranes of the eye and nose.

These limits remained unchanged for some years, although new standards in relation to respirable dust and respirable quartz were added. In 1994, the ACGIH again modified the limit to 10mg/m^3 *inhalable* (insoluble) particulate for dusts containing no asbestos and <1%silica.

The change to *inhalable dust* had implications as to the actual dust concentrations with the new inhalable samplers collecting more than three times as much as the 37mm closed face cassette sampler. This would result in a three-fold reduction in the dust standard in (in some circumstances) in real terms.

OSHA in their 1989 review kept the PNOC/R dust limit at 15mg/m^3 citing the physical irritation as the basis of the standard. The current collection method remains the 37mm closed faced cassette sampler, collecting less particulate than the inhalable dust samplers.

In Australia, the methodology for collecting inhalable dust (AS 3640:1995) conforms to ISO:7708 standard and is likely to collect even more dust than the ACGIH method. Therefore, the NOHSC standard is even more stringent.

The authors have not found any specific epidemiology assessing the PNOC/R limit of 10mg/m^3. As seen in its development, this standard is based on what is seen to be achievable and reasonable.

In the situation of exposure to coal dust, adverse outcomes of significant concern. (CWP or Silicosis) are being regulated by the respirable coal dust and respirable silica dust exposure limits.

The concern is whether exposures to excess levels of dust in the tracheo–bronchial region will occur with adverse outcomes, while these Respirable Coal Dust limits are being met. As can be seen from Potts et al (1990) and the NSW data (McFadden 2004, Coal Services 2005), the ratio of inhalable to respirable dusts can vary significantly. However, the research would indicate that on average, there is a reasonable correlation between respirable dust and the tracheo–bronchial fraction (Mark et al. 1988, Seixas et al. 1995).

Further study of this ratio of respirable to inhalable dust levels is indicated however, given that the studies addressing this matter have been small and all done overseas.

The authors note that the ratio of respirable to tracheo-bronchial to inhalable dust can vary within and between mines, with varying mining methods, ventilation rates, coal rank, and dust suppression methods.

Therefore, the presence of uncontrolled inhalable dust levels may be resulting in adverse health outcomes.

There is no doubt uncontrolled and unregulated inhalable dust levels will result in adverse health outcomes, through problems of eye irritation, reduced visibility, and reduced job satisfaction. Additionally from the review of the literature controlling respirable dust will not potentially protect against the development of chronic bronchitis and potentially all cases of COPD.

Comparison with Overseas Exposure Standards

As stated in the scope of work, the NSW Department of Mineral Resources desires to introduce a workplace exposure standard for inhalable dust and has suggested a value of 10 mg/m^3, *but as no authority currently invokes a respirable and inhalable coal dust standard,* the basis for such action remains unclear.

It should be noted that under mining regulations, which have been in force since September 1994, the Polish authorities apply TLVs for both respirable and inhalable airborne dusts in coal mines (Marek & Lebecki, 1999). No basis for the standards is given in this paper, although the subject matter is the occurrence of pneumoconiosis. See Table 8 below for TLVs:

Table 12 TLV for Airborne Dusts (mg/m^3) in Polish Coal mines.

Dust containing free silica above 50%		
	Inhalable dust	10
	Respirable dust	0.3
Dust containing free silica 10% - 50%		
	Inhalable dust	2.0
	Respirable dust	1.0
Dust containing free silica 2% - 10%		
	Inhalable dust	4.0
	Respirable dust	2.0
Dust containing free silica below 2%		
	Inhalable dust	10.0
	Respirable dust	-

NOHSC currently applies a OES-TWA of 3 mg/m^3 (respirable dust) for use in Australia. In the USA, the ACGIH has a TLV of 0.4 mg/m^3 (respirable) for anthracite and 0.9 mg/m^3 (respirable) for bituminous coal. **Both figures are based on lung fibrosis and lung function (ACGIH, 2004).**

As part of a review reported in the Safety and Health in Mines Research Advisory Board Annual Review 1998 (HSE 1999), the Institute of Occupational Medicine (IOM) assessed dust concentrations and exposures to ensure that the review of standards included an assessment of what can reasonably be achieved in mines. The main review, reported in the previous year's report, was limited by a lack of

up-to-date data on the size distribution and mineral composition of airborne dust in coal mines. A sampling exercise to fill these gaps was commissioned and IOM reported their findings in the 1998 annual report.

The Technical and Environmental Services Laboratory (TES) at Bretby collected 100 samples from 33 representative intakes, returns and headings at 9 collieries. Samples were collected by three types of personal samplers alongside the standard MRE 113A instrument. This allowed the measurement of concentrations of dust in various size fractions - respirable, thoracic and inhalable.

Concentrations of inhalable dust ranged from 2.3 mg/m^3 to 57.7 mg/m^3; generally two to three times greater, but up to 10 times greater, than the equivalent respirable dust concentration. This limited survey suggested the coal mining industry would find it very difficult to meet the current standard for inhalable dust which applies to surface industries.

IOM concluded that an occupational exposure standard for inhalable coal dust was not practicable. IOM was then asked to consider what level of exposure could reasonably be achieved, and recommend a regime for monitoring exposure to respirable coal mine dust and quartz.

HSE commissioned IOM to carry out a project involving further analysis of data for 7000 coalminers who were working at nine collieries in the late 1970s, gathered as part of the long term Pneumoconiosis Field Research programme. Three questions were posed:

- What deficits of forced expired volume in one second (FEV_1) influence detectably the level of exertional breathlessness in miners?
- What definitions of clinically relevant deficits of FEV_1 does this suggest?
- What are the quantitative relationships between cumulative exposure to respirable coalmine dust, and risks of these clinically relevant deficits, overall, and specifically over the range of low exposures likely to be encountered in modern British mines?

FEV_1 is an objective measure of lung function, which may be reduced in a range of respiratory conditions including both pneumoconiosis and chronic bronchitis/emphysema (chronic airflow limitation). Definition of what constitutes a clinically important degree of functional impairment is relevant to the assessment of risk in a regulatory context. The aims of this study were, therefore, firstly to relate the objective measure of lung function to subjective reports of breathlessness in order to identify clinically relevant deficits of FEV_1 and, secondly, to define the levels of exposure to coalmine dust which would give rise to these clinically relevant deficits of FEV_1.

The study has been completed and the final report (Cowie et al. 1999) published by IOM, indicated that there was no clear level of FEV_1 which enabled separation of individuals who were breathless from those who were not. Overall, however, levels of FEV_1 were lower in those reporting breathlessness. It was only possible, therefore, to define the probability of reporting breathlessness at any given FEV_1 (age and smoking) level.

Cumulative exposure to dust was examined in relation to a 'doubling of the risk' of having a clinically relevant deficit of FEV_1 that is doubling the odds of reporting breathlessness. There was a significant association between dust exposure and level of FEV_1 with no evidence of a threshold. An increase in cumulative dust exposure of 50ghm^3 was associated with an increase in the order of 1 to 2% of men with clinically relevant deficits.

At a cumulative dust exposure of 120 ghm^3 (an average 2 ghm^3 over 35 years) approximately 30% of 60-year old non-smokers would be expected to have a 'doubling of risk' compared to around 25% of 60-year old non-smokers not exposed to dust. Reducing the average dust exposure to 1mg/m^3 the corresponding percentage would be about 27.5% thereby giving a reduction of 2.5% compared to that for an average concentration of 2mg/m^3.

The IOM conclude that a reduction in the workplace standards for respirable coalmine dust would result in a small but significant decrease in the percentage of workers whose lung function was reduced to a level associated with a doubling of the odds of reporting symptoms of breathlessness.

Following this review the HSE has proposed new Inhalable Dust Regulations with a new standard of 3mg/m^3 for respirable dust. No inhalable dust limit has been included in these regulations.

The loss of FEV_1 in NSW coal mines has been reviewed by Kizil and Donahue (2002). They found that in longwall mines, average respirable dust levels are 1.51mg/m^3. They have suggested that although at these levels of exposure, the coal miners will develop some loss of FEV_1 as a result of their dust exposure, the loss will be small and probably not clinically relevant. On considering the work from IOM, a very small percentage will benefit for an improvement of dust levels. Therefore continuing reduction of dust levels is indicated.

On considering the NSW data provided on inhalable dust, it is seen that controlling dust levels to concentrations within the standard for respirable dust may not necessarily guarantee compliance with a standard for inhalable dust. It also suggests that the technical feasibility of achieving compliance will also have to be considered as an issue.

The metric used to measure and describe exposure to inhalable particles needs to be carefully considered, before deciding upon any particular value. Mass can be measured and is always concentrated in larger particles, which have a smaller surface area and therefore less available contact with body tissues. Total surface area must be estimated using very difficult calculations, but it may be more useful in understanding dose, at least for acute responses. Particle number may be more useful in understanding non-specific effects, but its measurement assumes a cut-off point at the lower limit (Guidotti, 1998).

The authors noted on their review of the coal services data that particulate respirator use in common in the NSW coal industry and this reduces the risk further from inhalable coal dust.

Conclusions

On the basis of this study, it appears that there is evidence to support the assertion that exposure to inhalable coal dust may be linked to certain adverse health outcomes. In particular, there is particularly strong evidence linking chronic obstructive pulmonary disease, such as bronchitis to prolonged exposure to high levels of inhalable dust.

There is also a significant body of literature which has investigated the relationship between coal mining and/or exposure to coal dust with cancer conditions, especially gastric cancer. There is evidence that the occupation of coal mining is associated with gastric cancer. The assertion that inhalable coal dust may cause gastric cancer is plausible as larger inhalable particles are swallowed from the nose-pharynx-larynx region and after transfer from the tracheobronchial region by means of the mucociliary cleansing system into the GI tract.

However, the published research does not provide any consistent evidence to support any association between *inhalable coal dust* and gastric cancer. It is probable that other lifestyle and infective causes have resulted in the observed associations, although further research would be necessary to make any firm statements.

As coal is a unique material, possessed of physical and chemical properties quite dissimilar to any other mineral, the authors did not feel it was possible to draw any comparisons with other inhalable dusts.

The question of a suitable workplace exposure standard for inhalable coal dust and the degree of protection it might provide for specific health outcomes is a vexed one. The research in this paper has indicated that virtually all research into dust related health outcomes in coal miners is based upon respirable dust. Furthermore, it appears that respirable dust levels cannot be used to predict inhalable dust levels, due to the enormous variability. This is a concern, given the findings of McFadden (2004) into inhalable coal dust concentrations in NSW Mines. It suggests that as in the UK, the NSW coal mining industry would find it very difficult to meet the proposed standard of 10 mg/m^3 for inhalable dust, although without any such standard adverse health outcomes will be observed including eye, nose and upper airway irritation and chronic bronchitis. There remains also a potential for the development of chronic obstructive airways disease.

Recommendations

The authors offer the following recommendations for the consideration of the Dust Committee:

1. More work is required on sampling of inhalable dust. The work commenced by McFadden in collecting his inhalable dust samples with respirable dust samples should be continued.

2. Further information is required on the data gathered by McFadden. In particular, it is recommended that the particle size distribution of coal dust should be characterised for different mines and for different occupations within the mines.

3. It is recommended that if further investigation confirms the poor relationship between respirable, inhalable coal dust and its subfractions, a study should be conducted to determine the incidence or prevalence of chronic obstructive pulmonary disease in NSW coal miners, and whether this can be correlated with levels of inhalable dust observed in NSW mines.

4. Further studies should be conducted on the prevalence and incidence of upper airways disorders and their relationship with exposure to inhalable coal dust.

5. Given that is unlikely to be practicable for the coal industry to meet the proposed standard of 10 mg/m^3 for inhalable coal dust, and the lack of any compelling rationale on current evidence for such a standard, it is suggested that the proposed standard should be adopted as a 'Best Practice Guideline' with a 5 year phasing in period until adoption as a legally enforceable standard. During this period the results of the study list in points 3 and 4 could be concluded. The authors were of the opinion that immediate imposition of a standard was not necessary after considering the literature to date and the assessment by Kizil and Donaghue (2002) indicating that current levels of respirable coal were not significantly high in the NSW coal mines to result in clinically relevant losses in FEV_1.

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