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Nikky LaBranche  
*University of Queensland*

David Cliff  
*University of Queensland*

Kelly Johnstone  
*University of Queensland*

Carmel Bofinger  
*University of Queensland*

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RESPIRABLE COAL DUST AND SILICA EXPOSURE STANDARDS IN COAL MINING: SCIENCE OR BLACK MAGIC?

Nikky LaBranche¹, David Cliff², Kelly Johnstone³ and Carmel Bofinger⁴

ABSTRACT: The re-identification of mine dust lung diseases in coal mine workers has prompted much work to be done to improve exposure monitoring and health surveillance in Australia. It is now recognised to be inadequate to talk about respirable dust in general terms, because size, shape and chemical content can affect the adverse consequences of excessive exposure. The Minerals Industry Safety and Health Centre within the Sustainable Minerals Institute undertook a gap analysis to identify needs for further research into respirable dust exposure monitoring and control. One such project is characterising the dust present in different mining atmospheres to understand the contribution of the chemical components, particle sizes and shape to the incidence of mine dust lung diseases in Australian mine workers.

Exposure standards were first set in British Coal and have subsequently been adopted by the US and Australia. This paper starts with a discussion of the British Pneumoconiosis Field Research data collection methodology and assumptions on which those initial standards were based. From there the discussion moves into the application of these standards to the US and Australia, the history of revisions to the exposure standard and limitations in sampling equipment and radiological diagnosis of disease.

INTRODUCTION

This paper will discuss the history of the Workplace Exposure Standards (WES) for Respirable Coal Dust (RCD) and Respirable Crystalline Silica (RCS) in coal mining. The role of silica in the diagnosis of mine dust lung diseases, the limitations of radiological diagnosis and difference in monitoring equipment are also addressed. British Coal set initial standards with the British Pneumoconiosis Field Research (BPFR), which were then modified by the United States. Australian Standards are often based on US standards. Safe Work Australia recommended the same limits for RCS in 2019 that the National Institute for Occupational Safety and Health (NIOSH) and the American Conference of Governmental Industrial Hygienists (ACGIH) had proposed in 1974 and 1983, respectively (NIOSH, 1974, ACGIH, 2010). Safe Work Australia recommended the same limits for RCD in 2019 that NIOSH and ACGIH proposed in 1995 and 1996, respectively (ACGIH, 2001, NIOSH, 1995b).

While the exposure limit for RCD and RCS have gradually decreased, there continue to be incidences of mine dust lung disease (MDLD) occurring globally. For example, the number of miners being diagnosed with MDLDs is increasing in Central Appalachia in the USA (Blackley et al., 2018). Is simply continuing to lower the exposure standard enough? Or is there more to the exposure than meets the eye?

COAL MINE DUST COMPOSITION

Coal mine dust may contain a complex mixture of over 50 different elements and their oxides (IARC Working Group on the Evaluation of Carcinogenic Risk to Humans, 1997). Dust characteristics can vary widely between and even within mines and what is present in the respirable fraction may differ from the overall seam composition (Server et al., 2019). In a systematic review of the relationship

¹ Research Manager-OHS, Minerals Industry Safety and Health Centre, Sustainable Minerals Institute, The University of Queensland, Email: n.labranche@uq.edu.au, Tel: +61 4 0761 0108
² Professional Research Fellow, Minerals Industry Safety and Health Centre, Sustainable Minerals Institute, The University of Queensland, Email: d.cliff@uq.edu.au, Tel: +61 7 3346 4086
³ Senior Lecturer, School of Earth and Environmental Science, The University of Queensland, Email: k.johnstone2@uq.edu.au, Tel: +61 7 3346 7816
⁴ Principal Research Fellow, Minerals Industry Safety and Health Centre, Sustainable Minerals Institute, The University of Queensland, Email: c.bofinger@uq.edu.au, Tel: +61 7 3346 4082
between ‘pure coal’ (non-quartz) and interstitial lung disease, Beer et al., (2016) did not find any studies addressing the pure carbon part of coal dust.

Bennett et al., (1979) found a progressive and five-fold increase in the incidence of Coal Workers Pneumoconiosis (CWP) from UK collieries mining low rank coal to those mining high rank coal (Bennett et al., 1979). Beer et al., identified nine papers which evaluated the ‘pure coal effect’ which supported an independent effect of non-quartz coal dust on the development of Interstitial Lung Diseases. However, further evidence is needed to prove this theory due to methodological limitations of the existing evidence (Beer et al., 2016). Beer concluded “While the association between coal mine dust exposure and lung disease has been investigated for decades it is still not clear what components of the coal dust are actually responsible for disease development.”(Beer et al., 2016).

Correlations have been made between disease and the surrounding rock. However, minimal research has been carried out to measure the health effects of various concentrations of the other components of dust even though they may be more prevalent than coal dust in the respirable fraction. These other components may include calcite, muscovite, calcium silicate, kaolinite, apatite, chlorite, orthoclase, plagioclase, and amphiboles.

A study by Stocks (1962) of disease among coal miners in the UK made a connection between the surrounding soil and rock exposure and stomach cancer “...where mortality from stomach cancer is peculiarly high, farmers, quarry workers in slate and igneous rock and coal miners all showed pronounced excess in age adjusted death rates from stomach cancer compared with men in other occupations, and this suggested that direct contact with soil in areas with high mortality may be a factor of importance. It may be therefore that the notably large mortality excess in the South Wales miners is connected with the kind of rock and soil rather than, or in addition to, the kind of coal.” (Stocks, 1962). This study is included in the ACGIH document setting the WES for RCD (ACGIH, 2001).

**HISTORY OF OCCUPATIONAL DISEASE IN MINING**

Occupational diseases, including mine dust lung disease, have been recognised in mining for centuries. Agricola wrote in his posthumously published De Re Metallica circa 1556: “It remains for me to speak of the ailments and accidents of miners, and the methods by which they can guard against these, for we should always devote more care to maintaining our health, that we may freely perform our bodily functions, than to make profits. Of the illnesses, some affect the joints, others attack the lungs, some the eyes, and finally some are fatal to men.” (Agricola, 1556). The next few sections will step through the history of the RCD WES and to some extent the RCS WES.

**Early Identification in the UK**

The setting of a WES for coal dust began in the UK. In 1831, Dr James Craufurd Gregory first described black pigmentation and disease in the lungs of a deceased coal miner. He linked this to pulmonary accumulation of coal mine dust. Gregory hypothesised that the black material seen at autopsy in the collier’s lungs was inhaled coal dust and this was confirmed by chemical analysis carried out by Professor Sir Robert Christison (Donaldson et al., 2017).

Gregory suggested that coal dust was the cause of the disease and warned physicians in mining areas to be vigilant for the disease. This first description of what came to be known as ‘coal worker’s pneumoconiosis’ sparked a remarkable intellectual effort by physicians in Scotland, culminating in a large body of published work that led to the first understandings of this disease and its link to coal-blackened lungs (Donaldson et al., 2017). Pneumoconiosis in British coal miners had been identified before 1930, but relied upon miners voluntarily coming forward for medical examination (Fay and Rae, 1959).

**British Pneumoconiosis Field Research**

The Pneumoconiosis Research Unit (PRU) was established in 1945 and by 1952 researchers had determined that coal workers’ pneumoconiosis could be divided into simple pneumoconiosis and complicated pneumoconiosis (or Progressive Massive Fibrosis (PMF)). The dust alone was considered to be the cause of simple pneumoconiosis while complicated pneumoconiosis was thought to be caused by the addition of an infection, probably tuberculous onto lungs already affected by coal dust.
The Pneumoconiosis Field Research Unit Interim Standards Study (ISS) was established in 1952 due to lingering uncertainties of the true prevalence of pneumoconiosis throughout the UK as well as the attack and progression rates of the disease under various conditions. In this study, the National Coal Board (NCB) aimed to find a "safe" exposure standard in terms of dust quantity and quality plus the relationship between the disease and respiratory disability.

Detailed research was performed across the UK including both medical studies and environmental studies, which were then assembled and analysed together. In the medical studies, chest radiographs were taken of all miners who volunteered to participate at 24 test collieries.

To measure the dust in the environment a thermal precipitator was used to collect area samples of 1-5 micron diameter dust in the vicinity of the worker. The particles were then counted by hand using a microscope. It later became evident that this method significantly underestimated the count due to particle overlap and the data had to be adjusted after scientific experiments were carried out to estimate the required correction (Atfield and Kuempel, 2003). It was not possible to take an exposure sample of each worker due to the size of equipment and the fact there were 35,000 men participating in the study.

The population was divided into work groups, commonly referred to as Similar Exposure Groups (SEGs) in Australia. Samples were collected from each working group which reduced the stratification to 1,500 SEGs. A representative from each group was selected at random to have their working area monitored over a shift. Over 60,000 samples were collected across 14,000 shifts by September 1958 (Fay and Rae, 1959). Collieries included in this study were specifically chosen as examples of all of the major variations in mining conditions in Britain.

Occupational hygienists at mines assessed the dust exposures of all SEGs. Here dust concentrations were measured gravimetrically by placing samplers close to the participating men throughout their working shifts. From these samples a gravimetric area concentration was determined for all occupational groups and five year inter-medical-survey periods. These gravimetric area samples are in contrast to the particle counts performed by the standard thermal precipitator used in the first phase of the research. A series of side by side instrument comparisons were taken to relate the particle count to the gravimetric mass, and the earlier measurements were re-expressed in equivalent gravimetric terms (Hurley et al., 1987).

No direct monitoring (referred to as personal or occupational monitoring) of the workers exposure to dust concentrations took place either before the research began or subsequent to the study at non-research collieries. For those men not at research collieries, six categories of coal mining activity were used to assume the average concentrations to which they were exposed. It was assumed that dust concentrations in other mines in the region were similar to those at the research pit in the corresponding period. It was also assumed that concentrations before the research were similar to those experienced during the first 10 years of monitoring.

With the data collected and the numerous assumptions previously discussed, a series of curves were developed. They estimated the probabilities of radiological changes (CWP and or PMF) over a mines working tenure, for various combinations of cumulative dust exposure, age and carbon content of the coal, using logistic regression methods.

Jacobsen et al.'s 1971 curve, the heavy dashed line in Figure, modelled the probability of developing Category 2 simple CWP or greater over a 35 year working tenure to average dust concentrations. This curve was thought to give the incidence of Category 2 simple CWP or greater that would result from a certain level of dust exposure in a population of workers (Jacobsen et al., 1971). The Jacobsen 1971 curve was extrapolated using data from the 10-year ISS, which indicated that a miner would never be at high risk of developing PMF at an average exposure of 2mg/m³ or below, over their working life. Following this model, the focus of CWP control lay primarily in the simple reduction of respirable coal mine dust exposure levels. This curve supported the results of other research at that time, which indicated that PMF was very unlikely to develop from cases below Category 2 CWP. (Cochrane et al., 1961, McLintock et al., 1970). However, it was later identified that contrary to Cochrane's findings, PMF could develop from Category 0 or 1 CWP (Hurley et al., 1987, Hurley and Maclaren, 1988, Maclaren et al., 1989, Shennan et al., 1981).
The Hurley and MacLaren (1987) curve, the dash dot line labelled as “this report” in Figure 1 indicates the percentage of miners predicted to have Category 2 or greater simple CWP after 35 years of work at 1740 hours per year of mining at dust levels ranging from 1-8 mg/m³ at 86.2% carbon (Hurley and MacLaren, 1987). Dust concentrations were monitored close to the men throughout the working shift. Side-by-side comparisons were conducted to convert particle counts to gravimetric units and dust concentrations at other mines were assumed to be similar to those at the research pits where the measurements were taken (Hurley and MacLaren, 1987).

The Jacobsen et al., (1971) and Hurley and MacLaren (1987) curves shown in Figure 1 were an average over a number of collieries. When the data is broken out into the individual mines as seen with the solid lines from Hurley et al., (1982), the slope of curve for the individual mines themselves varied and the average does not represent all cases. The results were based on the same set of data; however, the Hurley and MacLaren data includes percent carbon as a predictor variable. Hurley et al., (1982) shows the comparison of the mean dust concentrations at 10 British coalmines to the probability of developing CWP 2/1+ over 35 years. He found that for eight of the 10 collieries, there was minimal difference in risk associated with working in similar dust conditions for the same length of time. However, for the other two collieries, the results differed sharply from this pattern. It was thought that mineralogical characteristics of the coal may be influencing the dust-pneumoconiosis relationship. The coal rank indices did not explain the extreme variation, nor did the Quartz exposure explain the difference as Colliery Q had a quartz content of 6.4% while Colliery T was 5.0% (Hurley et al., 1982, Hurley and MacLaren, 1987).

It was clear to Hurley et al., (1982) that the risks are far higher or lower than the average values at some collieries. This implies that the “probability estimates will not necessarily reflect the risks to coalminers generally unless the dust concentrations and the sources of unexplained colliery-related variability occur in a pattern broadly similar to that observed in this study (Hurley et al., 1982).

![Diagram](image)

**Figure 2:** Estimation from various studies of the concentration-specific risks of showing category 2 or more CWP after 35 years work at 1740 hours per year.

**Figure 1:** Comparison of the Concentration Specific Risk of Category 2+ CWP after 35 years from Jacobsen et al., (1974), Hurley et al., (1982) and Hurley and MacLaren (1987).

While coals may be similar, mining methods may present important differences in the dust exposures. In 1992, Attfield stated that “because the various methods may give rise to different particle-size distributions and involve cutting into roof, floor, or dirt band rock to differing degrees, the resulting dust clouds may vary considerably in type and composition and thus may have differing fibrogenic potential” (Attfield, 1992). In summary, the British system was based on particle counts a number of
research collieries which were later correlated to gravimetric sample. It was at first thought that PMF only resulted from Category 2+ CWP, which was not possible at an exposure of 2mg/m³ or below over a working life.

**US Workplace Exposure Standard History**

The United States Mine Safety and Health Administration (MSHA) changed the coal dust exposure limit from 3.0 to 2.0 mg/m³ in 1972. And then on 1 August 2016, the concentration limit of respirable coal dust reduced from 2.0 mg/m³ to 1.5 mg/m³. The 2 mg/m³ exposure standard, which came into effect in the US in December 1972, was based on the UK data from the Pneumoconiosis Field Research study. Attfield (1992) commented on the validity of extrapolating the results from previous studies of British mines to the US situation stating it may not be possible “given that such an evaluation would require knowledge that is now unavailable (such as that particle-size distributions or composition for mines that are now closed)” (Attfield, 1992). He also noted that the British studies were based on x-ray readings from international classification standards for pneumoconiosis that were no longer current (Attfield, 1992).

The US has well documented regional variations in the prevalence of coal worker’s pneumoconiosis. In Figure 2, Graph A shows the prevalence of CWP for all of the US, while Graph B breaks out central Appalachia into its own category. In this instance, central Appalachia is defined as Kentucky, Virginia and West Virginia. As can be seen, the rates of CWP have been increasing in central Appalachia. The third graph shows the US without the effects of the central Appalachia region where the increase is only slight for this cohort. The CWP prevalence is four times higher for Central Appalachia underground miners than it is for long tenured underground miners elsewhere in the US. One in 20 long tenured miners in central Appalachia has CWP that has progressed to PMF. This is even with the 1.5 mg/m³ exposure standard and the use of continuous personal dust monitoring technology underground (Blackley et al., 2018).

![Figure 2: Prevalence of coal workers pneumoconiosis in the US by region a: All US b: Central Appalachia only c: All US less Central Appalachia (Blackley et al., 2018)](image)

NIOSH recommended a WES of 1.0 mg/m³ in 1995 (equivalent to 0.9 mg/m³ measured according to ISO/CEN/ACGIH criteria) (NIOSH, 1995a). There was pushback from industry and MSHA ultimately adopted a WES for RCD of 1.5 mg/m³. The US measures quartz content as opposed to RCS. These samples are collected gravimetrically and a reduction factor is applied to the RCD limit measured via real-time monitoring, if necessary. In summary, the US adopted the UK exposure standard without redoing the epidemiology to account for the different coal geology and mining conditions, but then revised the standard downward given the high incidence of CWP.

**Australian Workplace Exposure Standard History**

The two major coal mining states, Queensland (QLD) and New South Wales (NSW) both adopted the 3.0 mg/m³ standard from the US. Australian coal mining legislation is state based. Each state therefore has their own legislation and systems for setting and monitoring the RCD WES. In 2004, the pump flow rate changed to 2.2 L/min in order for the cyclone elutriators to better conform to the ISO
curve. At that point, NSW lowered the WES to 2.5 mg/m³. QLD remained at 3.0 mg/m³ with shift adjustment.

Queensland reduced the RCD WES from 3.0 mg/m³ to 2.5 mg/m³ on 1 November 2018 and then applied a further reduction to 1.5 mg/m³, which commenced 1 September 2020 (Queensland Government, 2018). Queensland also reduced the WES for RCS to 0.5 mg/m³ on 1 September 2020 (Queensland Government, 2018). In QLD, only about a third of the MDLD cases being diagnosed are CWP and a growing number are silicosis and COPD (Queensland Government, 2020). NSW reduced the RCS WES from 1.0 mg/m³ to 0.5 mg/m³ on 1 July 2020. The reduction of the RCD exposure standard from 2.5 mg/m³ to 1.5 mg/m³ will take place on 1 February 2021.

Silicosis in Coal Miners

The exposure limit for silica is independent of the limit for respirable dust and much of the literature used to set the ACGIH TLV-TWA is for non-coal mining applications (ACGIH, 2010). The ACGIH recommends a WES of 0.025 mg/m³ to protect against silicosis and lung cancer. There were no studies referenced confirming a protective effect at 0.025, rather studies were cited indicating that 0.05 mg/m³ “would probably not be sufficiently protective of workers’ health” (ACGIH, 2010). This recommendation comes from the findings of several epidemiological studies that a WES of 0.05 mg/m³ has not shown a change in longevity or lung function even though a percentage were found to have 1/0 or 1/1 ILO profusion rating (ACGIH, 2010). The risk of silicosis and lung cancer was found to significantly increase at levels greater than 0.06 and 0.65 mg/m³ (Graham et al., 2004, Steenland and Sanderson, 2001). There is also evidence that Silicosis can progress even after miners leave the industry and the exposure to silica dust has ceased (Hnizdo and Sluis-Cremer, 1993).

For the UK data, in the Hurley et al., (1982) study, silica did not explain the variation in predicted incidence of CWP by colliery. However, there was evidence that some miners show unusual radiological changes when exposed to coal mine dust with a relatively high quartz content (Hurley et al., 1982). Hurley hypothesised that a slight overall quartz effect may remain hidden as a miners estimated lifetime exposure to quartz is less accurate than his corresponding mixed dust exposure estimate (Hurley et al., 1982).

The US has also found similar confounding factors between coal dust and silica. The comparison between countries did not take into account the differences in methodology used to estimate quartz levels (Attfield, 1992). Since 1980, the prevalence of r-type opacities (associated with silica) have increased sixfold among underground coal miners in Central Appalachia, while remaining static for the rest of the US. A 2016 case study in the US found that miners with rapidly progressive pneumoconiosis had lung pathology consistent with accelerated silicosis, mixed dust pneumoconiosis and these miners were exposed to silica and silicate minerals contained in respirable coal dust during their mining careers. Jobs associated with higher exposures to silica more frequently have severe and rapidly progressing disease than jobs associated with low silica exposure (Hall et al., 2019).

Residence time of the dust in the lungs may also be a factor for disease progression (Hurley et al., 1982, Graham et al., 2004). The ACGIH TLV documentation cites the 2004 work of Graham et al., on granite miners that found that when retirees whose RCS workplace exposure concentrations average 0.06 mg/m³ were studied, the risk of silicosis was significantly greater (7.1% versus 1.2%) when compared to employees examined at or before retirement. (Graham et al., 2004).

Hnizdo and colleagues (1993) investigated a cohort where 313 South African miners developed 1/1 silicosis at an average age of 55.9 years. They found that in 57% of the silicotics studied, the radiological signs developed on average 7.4 years after mining exposure ceased. This means the risk of silicosis was strongly dose-dependent while the latency period was largely independent of the cumulative dust exposure (Hnizdo and Sluis-Cremer, 1993).

RADIOLOGICAL DIAGNOSIS OF CWP AND OTHER MDLDS

Diagnosis of CWP has historically been based on chest radiographs, also known as x-rays, which add an additional set of confounding factors to the estimation of the exposure standard. The historically reported CWP levels may be an underrepresentation of actual disease prevalence for several reasons. In the UK, ISS miners needed to have been working at the colliery for 10 years (Hurley et al., 1982). It
is likely that miners may be self-selecting themselves out of the cohort if they start to experience symptoms before this threshold.

It is unknown what the false negative rate was for CWP in chest radiographs, so there may be cases that exist that were not diagnosed by x-ray. Most surveillance programs stop when a miner retires or leaves the industry. The few studies that have been performed on retired miners have found that MLDs, especially silicosis, can continue to develop after exposure has stopped. Baseline prevalence also increases with age. While age was included in some UK analysis, the model chosen does not allow for this variable (Attfield, 1992).

Interpretation of x-rays also differed between the UK and US. US readers tended to report more abnormalities that past UK readers. Based on this trend, the CWP estimates for the US would be greater than those of the UK for the same expected exposure curve. The classification standards for x-rays changed over time and were different during the ISS than they were for more recent US studies (Attfield, 1992).

Comparisons of X-rays to CT scans show that x-rays tend to significantly underestimate disease. In a study by Remy-Jardin 1990, 48 patients were diagnosed as Category 0 by radiograph, but CT revealed that only 36 were in fact Category 0, while seven were Category 1 and five were Category 2. For the 65 patients diagnosed at Category 1 by radiograph, 31 patients were Category 0, 29 maintained their Category 1 status and five were Category 2 (Remy-Jardin et al., 1990).

Hnzido compared x-ray diagnosis of silicosis to autopsies in 984 miners and found that where silicosis was positively identified in the autopsy it was not diagnosed by the most accurate x-ray reader in 75%, 54% and 26% of cases of slight, moderate and marked silicosis cases, respectively. The 326 cases diagnosed/confirmed at autopsy averaged of 63% of silicotics not diagnosed as positive using x-ray analysis alone with a 1/1 cut-off point (ACGIH, 2010, Hnzido and Sluis-Cremer, 1993).

Past studies have predominately only looked at cumulative dust in coal mines and not addressed the pure carbon part or other components of coal dust, with a small exception for bioavailable iron (Huang et al., 2005). In many cases, the exposure to silica dust was not treated separately to the exposure to coal dust. There are most likely other contaminants within the coal seam that are leading to differences in prevalence between mines and seams, thus necessitating further work to identify the components.

**MONITORING EQUIPMENT**

The UK, USA and Australia use different monitoring equipment to measure respirable dust. Cyclone elutriators have biases in sampling and different models may have different biases. These biases may be due to both the sampler design and manufacture as well as the different particle size distributions being sampled. Sampling performed with different samplers may not be directly comparable. For instance, the US uses Dorr-Oliver cyclones with a 1.7 L/min flowrate while Australia uses Higgins-Dewell type cyclones at 2.2 L/min. A direct comparison of dust cannot be made between the two without side by side testing as different coal seams have different particle size distributions and therefore different sampling bias. Further error in sampling can be introduced based on pump pulsation.

The efficiency of a cyclone elutriator is affected by the airflow draw from the pump and flow pulsations may invalidate sample collection. Sampling requirements in BS EN ISO 13137:2013 Workplace atmospheres- Pumps for personal sampling of chemical and biological agents - Requirements and test methods requires that pump pulsation not exceed 10% of the flowrate (ISO (International Organisation for Standardization), 2013). Lee tested 13 widely available sampling pumps and found that ~80% of the pump models tested generated pulse magnitudes ≥10% with a wide variety of pulse shapes (Lee et al., 2014). Cornelissen (2008) tested pumps from three different manufacturers and found that all the pumps tested failed to maintain ≤ 10% pump pulsation. The flowrate also exceeded the allowable limits of flowrate variation in 77% of the readings (Cornelissen, 2008).

The respirable fraction of coal mine dust and the components within it have differing particle size distributions. Two mines in different seams may measure the same gravimetric mass. However, the number of particles present and the surface area of those particles may vary considerably. Cyclone elutriators measure aerodynamic equivalent diameter and not simply particle diameter. Particles with the same size and shape may have different densities due to different chemical compositions.
SHIFT ADJUSTMENT

The workplace exposure standards for RCD and RCS are based on 8-hour days, five days a week. Most Australian mines work longer shifts, for instance 12 hours a day, seven days on and seven days off. To account for these longer shifts, the WES needs to be adjusted.

The Brief and Scala model for adjusting WESs for non-traditional work weeks, is one of the more conservative models and is recommended by Safe Work Australia. It reduces exposure standards proportionally for increased exposure and reduced recovery time. The Brief and Scala model was developed for the petroleum industry and has not been validated for dust exposures. The adjustment process has no consideration for the agents' activity on the body, the process by which the body removes the chemical or the biological half-life of the chemical (AIOH (Australian Institute of Occupational Hygienists), 2016, Teirnan and VanZanten, 1998).

The Occupational Safety and Health Administration (OSHA), Quebec and Pharmacokinetic models are based on the residence time for the particles in the lungs and assume that a long biological half-life for a substance is 1000 hours. This assumption needs to be analysed and verified for coal dust as measurements of the half-life of coal dust yield significantly longer timeframes. Morrow (1982) cited several mammal studies measuring half-lives of coal including, Heppleston et al., (1971), which found rats with chronic high level exposure had 380 and 430 day half-lives for high and low rank coals and Stober et al., (1967), which found a 4.9 year biological half-life in a post-mortem investigation of miners. Morrow (1982) found coal retention half-lives to be 767 and 637 days for anthracite and high rank bituminous coals in dogs lungs (Morrow et al., 1982).

OTHER LIMITS OF DATA

The epidemiological data only considers papers from English-speaking countries. Historic study populations consisted solely of males. There is therefore no information about the particular risks of female susceptibility or any potential impact of ethnicity. Smoking was only taken into account in a few studies and it is suggested that smoking is a true risk factor as well as an effect modifier. Most studies were not adjusted for smoking status and may, in general, overestimate the effect of coal dust (Beer et al., 2016).

CONCLUSIONS

In conclusion, there are a number of factors to be considered when setting exposure standards. A WES cannot simply be copied across from one country to another without consideration of the differences in monitoring methodology, mining conditions, coal geology and health surveillance. Australia uses different sampling equipment than the US at a different flow rate, which has a different bias to the ISO sampling curve. The cyclone elutriators are measuring aerodynamic equivalent diameter and not strictly particle diameter, which means particles of similar sizes but different densities may be treated differently by the cyclone. Australia should perform its own studies to verify that overseas WESs are fit for purpose in the Australian mining environment including shift lengths.

The UK and US standards are based on averages of dust concentrations for all mines. The data shows disease prevalence can vary significantly in different mines and regions. Consideration should be given to these differences in Australia. More research is needed into the chemical composition and particle size distribution of these coals and what is causing these differences in disease prevalence. Future WESs may need to include mine or region specific limits based on these factors. In Queensland, CWP accounts for only a third of MDLD cases. The WES for RCD also needs to consider not just CWP, but the host of other MDLDs that are being diagnosed. This may include the need to understand the contributions of the inhalable and submicron fractions of dust in addition to the respirable fraction.

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