Environmental Research 190 (2020) 109849

Contents lists available at ScienceDirect

Environmental Research

journal homepage: www.elsevier.com/locate/envres

Review article

The impacts of coal dust on miners' health: A review

Ting Liu [a,b], Shimin Liu [b,*]

[a] School of Safety Engineering, China University of Mining & Technology, Xuzhou, 221116, China
[b] Department of Energy and Mineral Engineering, G3 Center and EMS Energy Institute, The Pennsylvania State University, University Park, PA, USA

ARTICLE INFO

Keywords:
Coal dust
Nanoparticles
Coal workers' pneumoconiosis
Dust control

ABSTRACT

As one of the most important energy resources in the world, coal contributes a great deal to the world economy. Coal mining and processing involve multiple dust generation processes including coal cutting, transport, crushing and milling *etc*. Coal dust is one of the main sources of health hazard for the coal workers. Exposure of coal dusts can be prevented through administrative controls and engineering controls. Ineffective control of coal dust exposure can harm coal workers' health. Although many efforts have been made to eliminate these threats, recent years have seen an unexpected increase in coal workers' pneumoconiosis (CWP) in Appalachian basin in US. To explore the reasons for this phenomenon, in this review, we first reviewed the historical studies on coal mine dust including the regulation and engineering controls. Then, the effects of coal dust on human health was comprehensively reviewed. Next, the effects of nanoparticles on human health were reviewed, with an emphasis on toxicity of nanoparticles such as carbon nanotubes in other industries. From all this information, we hypothesize that nano-sized coal dust has contributed to the increase of CWP prevalence in recent years. As no research has been reported in this area, four directions which may need further investigation and future studies are recommended in this review. They include: 1) Systematic characterization of physicochemical properties of nano-size coal dust; 2) Toxicity and pathogenesis of nano-sized coal dust; 3) Development of real-time monitoring technology and equipment for nano-sized coal dust; 4) Development of exposure control technology and equipment. The intent of this review paper is to demonstrate the variation of coal dust properties and their impact on the mine worker's health. We suggest that the impact of nano-sized coal mine dust on miner's health has not yet been understood well and further improvements are necessary.

1. Introduction

Coal, a nonrenewable source of energy, has been an important component of the world's energy structure for a very long time. It can be seen from Fig. 1(a) and (b) that since 1971, total worldwide coal production and coal consumption has grown by more than 150%. According to the International Energy Agency, the proportion of coal in primary energy reached 29.03% in 2012, and it was predicted that by 2035, the proportion of coal will still be higher than 24% (Fig. 1(c)) (EIA, 2017). In the United States, coal was the second-largest energy source (30%) for electricity generation in 2017 (Fig. 1(d)). And in 2017, coal production was 702.49 million tons, which was 6.32% higher than it was in 2016 (U.S. Energy Information Administration, 2018; Alan et al., 2009). All these figures imply that coal is still the main energy source in the world in terms of promoting economic development.

Currently, surface mining and underground mining are the main methods of mining coal. The selection of mining method is mainly determined by the burial depth of coal seams, density and pressure of the overlying strata, and thickness of the coal seams. No matter which method is used, coal mining always raises many health issues.

One of the important issue involved in coal mining is coal dust, which can lead to a series of health problems (Yao et al., 2020). Nearly the whole mining processes is accompanied by coal dust generation (Shahan and Reed, 2019). Fig. 2 depicts the processes of coal dust generation during mining, showing that dust is generated during coal cutting, transportation and preparation processes (Fig. 3).

It is believed that coal dust is responsible for many human diseases, such as asthma, chronic obstructive pulmonary disease, stunted lung development, pneumoconiosis and lung cancer, cardiac arrhythmias, acute myocardial infarction, and so on (Petsonk et al., 2013). For coal miners, the most common and serious disease is pneumoconiosis (Finkelman et al., 2002). The most recent evaluation of 30 years' experience in the U.S. Coal Workers' Health Surveillance Program (CWHSP) showed that about 38% of coal miners with radiographic findings of

* Corresponding author.
E-mail address: szl3@psu.edu (S. Liu).

https://doi.org/10.1016/j.envres.2020.109849
Received 22 February 2020; Received in revised form 14 May 2020; Accepted 16 June 2020
Available online 5 August 2020

interstitial disease had primarily irregular opacities (Attfield, 2011). Since 2000, rates of CWP have constantly increased; most troubling is the increase in severity. On the other hand, increasing amounts evidence show that exposure to coal mine dust in coal workers is a high risk factor for lung cancer, which is causing more death than the next three major cancers combined in the U.S. Unfortunately, recent observations showing increased incidence of CWP or other lung disease, particularly among young miners in the Appalachian basin (of which the CWP prevalence is four times higher than the national average)—including Pennsylvania, West Virginia, Virginia, and Ohio—are alarming (Blackley et al., 2018a,b Potera, 2019).

Given the extent of coal mining globally and its potential for a large burden of disease, the coal dust generated in mining processes is a serious threat to coal miners' safety and health. In 2006, in order to address this health issue, the U.S. Federal Mine Safety and Health Administration (MSHA) enforced the dust rule for underground coal mines at a concentration level of less than 2 mg per cubic meter of ventilation air (2 mg/m^3). On August 1, 2016, MSHA even issued the new rule that lowered respirable coal dust concentration limits down to 1.5 mg/m^3 (MSHA, 2016).

2. Background and coal dust related health studies

2.1. Brief history of the coal dust related health regulation

Since the first case was reported by Gregory in 1831, coal workers' pneumoconiosis (CWP) has attracted great attention (Gregory, 1831; Ross and Murray, 2004). However, at the initial time, CWP was thought of as a variant of silicosis because of their similar chest radiographs, while coal dust was thought to be innocuous (Mo et al., 2014). In 1936,

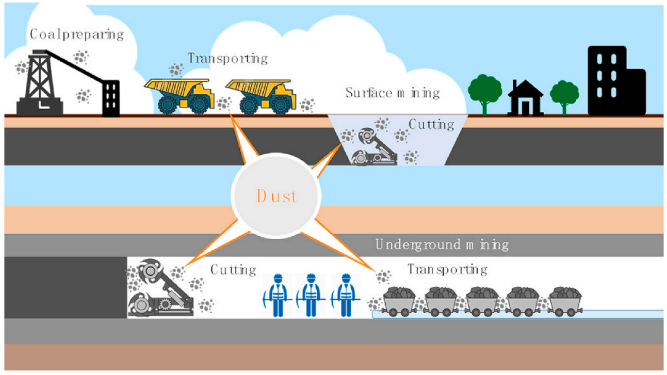

Fig. 2. Coal mine dust generation during mining processes.

the British Home Office and the Mines Department charged the Medical Research Council to conduct a survey on this topic, and the report showed a link between exposure to coal dust and respiratory disability (McGuire, 2019).

In 1969, the Federal Coal Mine Health and Safety Act, generally referred to as the Coal Act, was passed. In the Coal Act, specific procedures for the development of improved mandatory health and safety standards were included, and compensation for miners who were totally and permanently disabled by the progressive respiratory disease caused by the inhalation of fine coal dust pneumoconiosis, or "black lung," was also provided in this legislation. In addition, a permissible exposure limit of 2 mg/m^3 of respirable coal mine dust was mandated in the Coal Act (Attfield, 2011). This limit was derived from a British study that produced the only quantitative exposure-response relationship available at

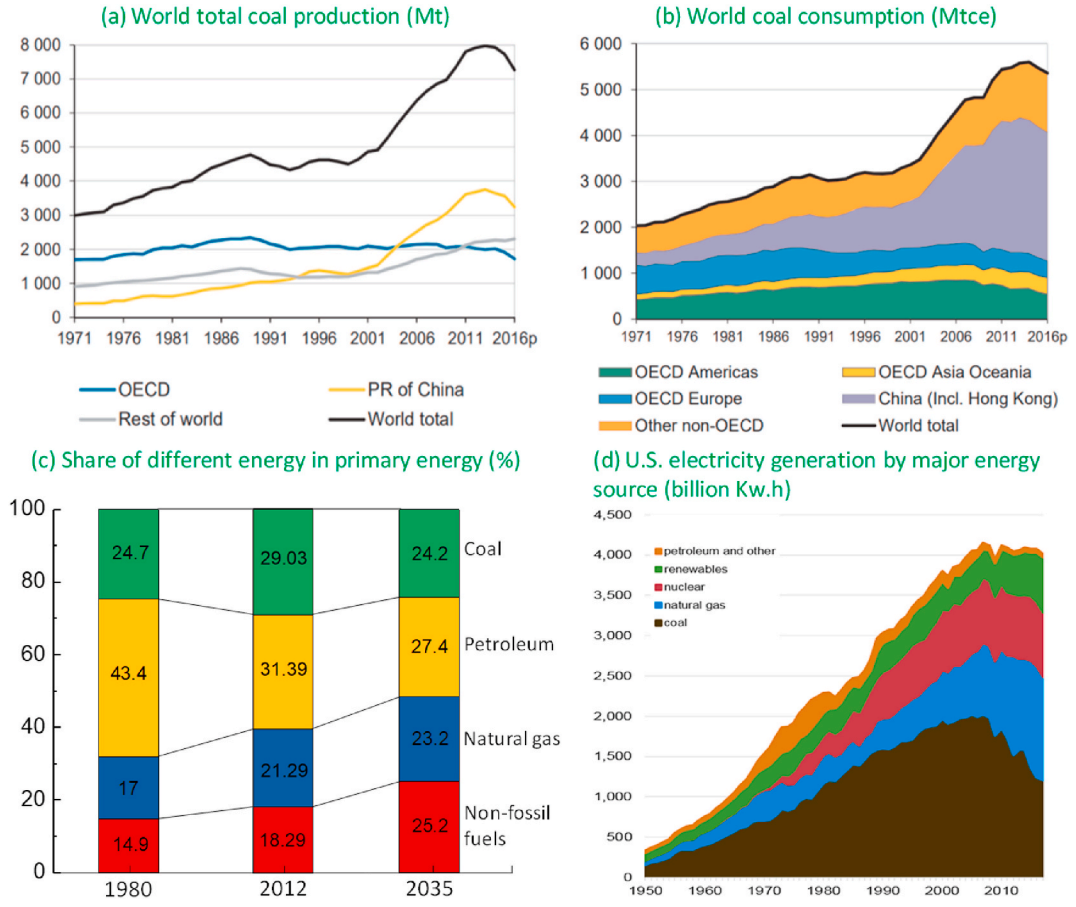

Fig. 1. Coal production, consumption, and proportion in primary energy and electricity generation (EIA, 2017).

the time, and which concluded that there would no cases as severe as category 2 on the International Labour Office classification system among miners who worked 35 years with respirable coal dust concentration of 2 mg/m^3 (Attfield, 2011; Jacobsen et al., 1971). However, research by Hurley and Maclaren (1987), and research on U.S. underground coal miners by the National Institute for Occupational Safety and Health (NIOSH) (Attfield, 2011) demonstrated that there was no threshold at 2 mg/m^3 under which the CWP cases would not occur. Further evidence indicated that simple coal workers' pneumoconiosis, progressive massive fibrosis, and chronic obstructive pulmonary disease may develop at the current permissible exposure limit of 2 mg/m^3. Therefore, in 1995, NIOSH recommended that the permissible exposure limit of coal dust should be reduced to 1 mg/m^3 as a time-weighted average concentration for up to 10 h a day during a 40-h work week (NIOSH, 1995).

Due to enforcement of the regulation regarding the permissible exposure limit of 2 mg/m^3, in the decades thereafter, we witnessed a significant decrease in the prevalence of CWP among active miners, as reported by the NIOSH Coal Workers' Health Surveillance Program (Blackley et al., 2015). For example, for underground miners with a 20- to 24-year tenure, the percentage of coal workers with pneumoconiosis (CWP) declined from 32.8% in 1970 to 1.9% in 2009 (Seaman et al., 2015). However, in spite of the regulations, a resurgence of the disease was observed in 2003 (Blackley et al., 2015) and a CDC/NIOSH report demonstrated that starting in 1995, the prevalence of CWP in U.S. underground coal miners was no longer decreasing but had again begun to increase (Attfield, 2011).

2.2. Statistics of coal dust related health issue

In case of heavy exposure to coal dust, coal workers' pneumoconiosis may develop into progressive massive fibrosis (PMF), which can be fatal to coal workers. There were 25,000 pneumoconiosis deaths worldwide in 2013 (Zosky et al., 2016; GBD 2013 Mortality and Causes of Death Collaborators, 2015). Currently, there are no specific medicines nor effective treatments for CWP. The only current treatments include measures to relieve symptoms, reduce complications, and slow disease progression (Napierska, 2010; Honma, 2004). Because of the progression and irreversibility of CWP, patients may gradually lose the ability to work; in addition, their quality of life will be reduced, and their life expectancy will be shortened. Additional physical, mental, and economic burdens may be put on patients and their families by the disease. Also, the normal production, survival, and development of the enterprise will be affected. In short, incalculable economic loss will be caused to society as a whole (Han et al., 2017a,b; Zhang, 2010).

Fig. 4(a) shows the prevalence of CWP in examined U.S. underground miners with different tenures during the period of 1970–2017. This data is shown as a 5-year moving average. It can be seen that at the initial time (1974), the prevalence of CWP was relatively high—nearly 35% of the examined workers with more than 25-year tenures were diagnosed with CWP; the prevalence of CWP of workers with 20–24 and 15-19-year tenures was more than 20% and 15%, respectively. Even the prevalence of CWP in workers with less than 10-year tenures was more than 3%. These figures demonstrate that CWP was very serious at that time. But, as we can see from this figure, from 1970 to 2000, the prevalence of CWP showed a significantly decreasing trend. This can mainly be explained by the implementation of the 1969 Federal Coal Mine Health and Safety Act and the advancement of the dust removal technology. And, the prevalence of the most severe form of CWP, PMF, reached a low of 0.08% of miners surveyed for the period 1995 to 2000 (Laney and Weissman, 2014). However, thereafter, the trends reversed. The national CWP prevalence in miners with 25 years or more of tenure now exceeds 10%; among miners with 20–24 years of tenure, the prevalence exceeds 5%. Things get worse in central Appalachia where 20.6% of long-tenured coal miners had radiographic evidence of CWP in the most recent 5-year period (Fig. 4(b)). This may be caused by the higher geometric mean concentration and the mean percent content of the respirable quartz in this zone (Doney et al., 2019). When miners from central Appalachia are removed from the calculation (Fig. 4(c)), the prevalence for the remainder of the United States is substantially lower, but the increasing trend is still evident (Blackley et al., 2019; Reed et al., 2019).

The upward trend seen in Fig. 4, which shows the all pneumoconiosis cases (category 1+), is even more evident for PMF depicted in Fig. 5, which shows that since 1994, the prevalence of PMF has begun to rise. The most troubling point is that the CWP severity is still increasing (Graber et al., 2017; Cohen et al., 2016). For example, in West Virginia alone, 138 miners with PMF were approved for compensation between

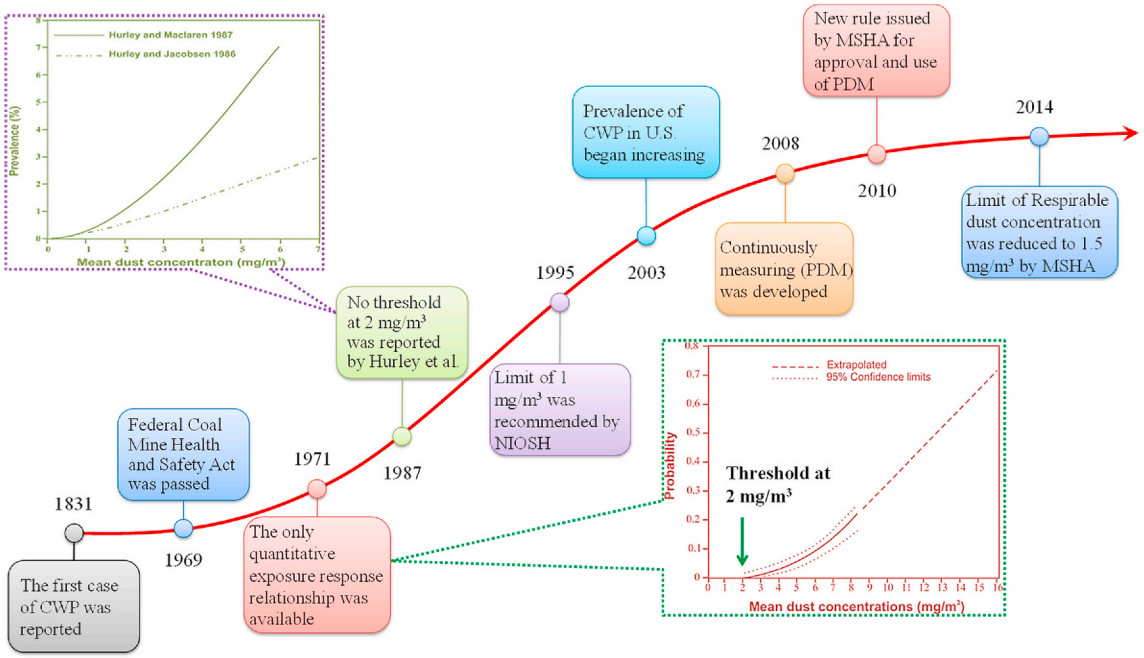

Fig. 3. Landmark events associated with coal dust (The curves in this figure are from (Attfield, 2011)).

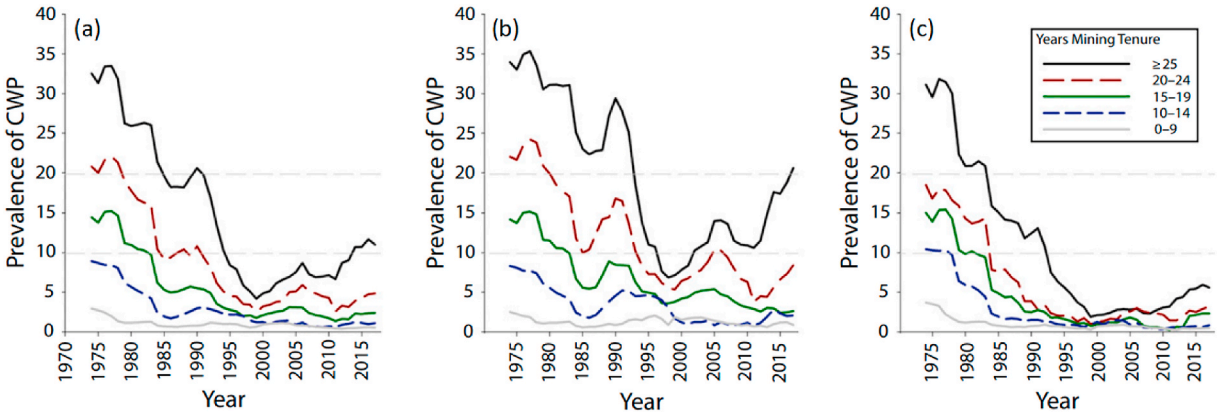

Fig. 4. Prevalence of Coal Workers' Pneumoconiosis (CWP) Among Those Working Underground in (a) the United States, (b) Central Appalachia, and (c) the United States Excluding Central Appalachia (Blackley et al., 2019).

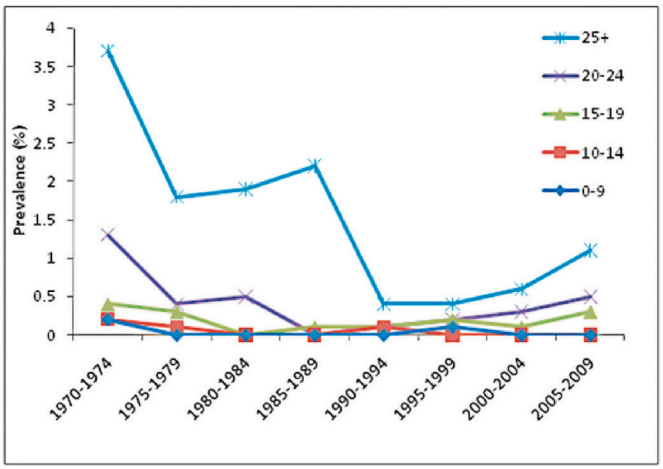

Fig. 5. Percentage of examined US underground miners with PMF from the NIOSH Coal Workers' X-ray Program from 1970 to 2009, by tenure in coal mining (Laney and Weissman, 2014).

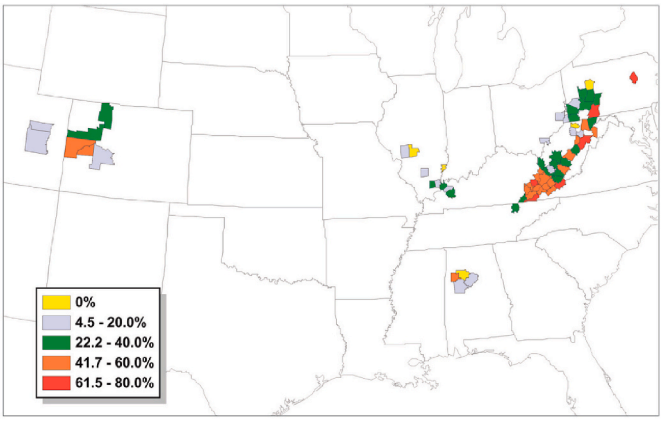

Fig. 6. Percentage of examined miners with rapidly progressive coal workers' pneumoconiosis by county (not shown are counties with fewer than 5 miners evaluated) (Attfield, 2011).

2000 and 2009. Their mean age was only 52.6 years (Laney and Weissman, 2014).

To understand the real causes for the upward trend of CWP prevalence, research was conducted by NIOSH through the CWHSP. The prevalence data obtained in this research was summarized by county and then plotted to get the 'hot spots' of the high incidence area of CWP. Fig. 6 shows the spatial distribution of CWP prevalence. It can be seen that the 'hot spots' tended to locate on the eastern edge of the Appalachian coal field but were particularly concentrated in the southern West Virginia (WV)/western Virginia (VA)/eastern Kentucky (KY) tri-state region (central Appalachian region) (Attfield, 2011; Antao et al., 2005).

CWP is not exclusive to the U.S. but is actually a worldwide problem. In Australia, it has been claimed that CWP has been absent for the past 30 years, but in 2016, more than 10 coal miners were diagnosed with the disease; also, it has been projected that up to 1000 coal miners in Queensland could have CWP among 150,000 unprocessed x-rays (Yates et al., 2016; Zosky et al., 2016; Xu et al., 2018). In Glass et al. (2018), 248 coal miners were sampled, and diagnostic results demonstrated that 7.3% of the sample has simple CWP, which is the early stage of CWP.

The prevalence of CWP is also high in China—it was 6.02% in the 2010s, and 13,955 and 13,846 miners were diagnosed with CWP in 2013 and 2014, respectively (Zosky et al., 2016; Han et al., 2017a,b; Harrison, 2015). Current statistics indicate that there are up to 720,000 pneumoconiosis patients in China, and about 62% of them come from the coal mining industry. To get a better understanding of CWP prevalence in a coal mining enterprise in China, a research study was conducted by Wang and Zhang (2016) from the Occupational Safety and Health Research Center of State Administration of Work Safety Supervision and Administration in China. In this research, 62,976 workers who were exposed to coal dust during 2002–2013 were examined. The results indicated that since 2002, CWP prevalence of this enterprise stabilized at around 10%. The prevalence in 2003 was the lowest with a value of 6.66%, while in 2013, it increased to 24.70% during this period. The mean age of onset was (52.6 ± 11.3), and the mean working age of onset was (18.4 ± 10.1) (Wang and Zhang, 2016).

The research results above demonstrate that the CWP remains a serious, worldwide occupational disease, which calls for further research to obtain a better understanding of its essential pathogenic mechanism.

3. Coal mine dust property and its effects on miners' health

3.1. Coal dust characterization

3.1.1. Size distribution

Particle size (particle size distribution) is an important parameter of coal dust, as it directly affects its reaction rate, sedimentation, solubility and human health (Marsalek and Sassikova, 2016). An accurate characterization of coal dust size distribution is of great importance to prevention of CWP.

Currently, there exist many methods to measure coal dust particle size, including sedimentation methods, mechanical particle sorting,

filtration, optical methods, dynamic light scattering, and laser diffraction (Marsalek and Sassikova, 2016; Sui et al., 2016; Tailakov et al., 2015). Among these methods, laser diffraction allows particle sizes to be measured from hundreds of nanometres to units of millimeters. This method is based on measurement of laser beam scattered by the particles. A position of maximum intensity of the diffraction light is proportional to particle size (Marsalek and Sassikova, 2016; Dalmora et al., 2016; Everard et al., 1997; Bäckström et al., 2015).

Xue (2016) characterized the pulverized coal particle size distribution with a Mastersizer 2000 particle size analyzer. Fig. 7 shows the size distribution of the pulverized coal. two peaks of size distribution can be observed at 60–70 and 830–900 μm. Nearly 90% of the coal particles are smaller than 200 μm, and 22.5% of pulverized coal is in the range of 90–200 μm on average.

3.1.2. Geometry

Geometry (shape, size) of the particles has a great influence on the physical and chemical actions occurring on its surface (Ulusoy et al., 2003). Recent studies indicated that the respirable particles with smaller size were more harmful to human health than the bigger ones (Mischler et al., 2016). It is also documented that the deposition mechanisms of dust in the lungs can be dependent on both size and shape (Watkins--Pitchford and Moir, 1916; WHO, 1997; Beeckmans, 1965). Currently, a lot of parameters have been put forward, among which, the length (L), width (W) and thickness (T) are the basic ones. With these directly measured parameters, some calculated parameters have been further proposed, such as circularity (Singh and Ramakrishnan, 1996), aspect ratio and elongation (Xia, 2017), shape factor, cross-sectional diameter, particle angularity, and so on (Sellaro et al., 2015; Ahmed, 2010).

Optical microscope (OM) was the first technology adopted to characterize the morphology of particles in micro-level. However, for its test range, it was difficult – using OM – to obtain the information at the nano-level. Since then, scanning electron microscopy (SEM) has become the most common method used to show enlarged pictures of micro/nano-mineral particles (Yekeler et al., 2004; Ulusoy et al., 2003; Johann-Essex et al., 2017a; Fisher et al., 1978).

Fig. 8 shows the cross-sectional diameter and aspect ratio of coal samples in central and northern Appalachia using computer-controlled scanning electron microscopy with energy dispersive X-ray. It indicated that samples from mid-central Appalachia had higher percentages of very small particles, while samples from south-central Appalachia had higher percentages of elongated particles (Johann-Essex et al., 2017b).

3.1.3. Composition and chemical properties

3.1.3.1. Element. It has been documented that there are about 76 elements in coal, among which, some elements such as arsenic, mercury, lead, etc., are potentially hazardous to human health (Flores, 2013). An accurate characterization of the occurrence of these elements in coal will greatly contribute to environmental protection and human health (Finkelman et al., 2018, 2019; Moreno et al., 2019).

According to differences in sample processing, available techniques for coal element determination can be divided into destructive and non-destructive methods. The destructive techniques generally require a sample preparation process to separate the elements from the coal matrix, and in most cases, the coal matrix will be destroyed. Commonly used destructive technique includes: ICP-OES, ICP-MS, AAS and IC (Swaine, 1994; Érico et al., 2007; Wagner and Hlatshwayo, 2005; Low and Zhang, 2012). Non-destructive techniques refer to the techniques requiring minimal sample preparation or no sample preparation at all. Techniques XRF, INAA, HR-CS-AAS and LA-ICP-MS/AES are non-destructive methods (Mketo et al., 2016).

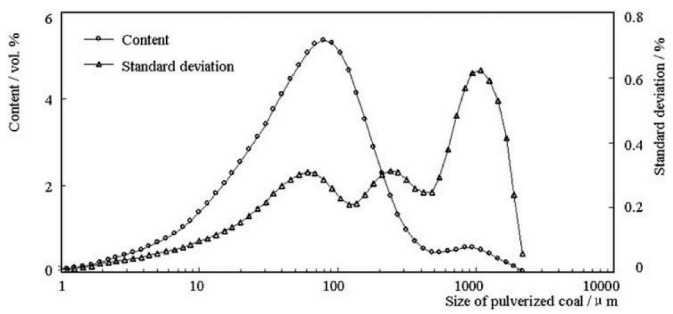

Fig. 7. Coal particle size distribution (Xue, 2016).

Yang et al. (2018) investigated the elemental composition of upper Pennsylvanian coal from the Hedong Coalfield in the eastern Ordos basin in China; a combination of X-ray fluorescence (XRF) spectrometry, inductively coupled plasma mass spectrometry, and collision/reaction cell technology for inductively coupled plasma mass spectrometry (ICP-CCT-MS) was adopted. The results shows that elements Cr, V, and Zn were close to the global average concentrations (Ketris and Yudovich, 2009), while As, Hg, and Mo were depleted. Elements Ba, Be, Co, U, Th, and F showed various enrichment degrees.

3.1.3.2. Mineral matter. It has been reported that there are about 120 minerals in coal (Ward, 2002, 2016; Flores, 2013). Particular minerals generated during mining process are thought to have great impact on human health (Finkelman et al., 2019). For example, silicosis is believed to be caused by long-term inhalation of dust with high content of quartz.

Currently, a range of techniques can be used for the qualitative and quantitative characterization of minerals in coal. Among these techniques, frequently used methods include low-temperature oxygen-plasma ashing, quantitative evaluation of X-ray diffraction data, observation using optical microscopy, chemical analysis, and, in some cases, mapping based on scanning electron microscopy and related methods (Ward, 2016).

Study on nine Australian bituminous coals from New South Wales and Queensland with X-ray diffraction analysis combined with mineral phase identification with SIROQUANT™ (Taylor, 1991) showed that the most abundant phase in coal was kaolinite, which was identified in all samples. Quartz was a major constituent of the minerals in most coals, while marcasite and rutile can only be found in individual samples (Grigore et al., 2008).

3.1.3.3. Functional group. The functional groups of coal have a great impact on chemical properties and adsorption for harmful or toxic gases. Currently, instrumental analysis methods, such as Fourier transform infrared spectroscopy (FTIR), nuclear magnetic resonance (NMR), and X-ray Photoelectron Spectroscopy (XPS) have been widely adopted to study the functional group of coal (Miao et al., 2015; Xin et al., 2014; Wang et al., 2011; Sun et al., 2018).

Infrared spectra are absorption spectra caused by vibration absorption of infrared light by chemical bonds or functional groups in molecules at a specific frequency. The functional group types and contents in samples can be determined according to the position and intensity of infrared absorption peaks (Ibarra et al., 1996).

Xu et al. (2017) characterized the surface chemical structure of different kinds of coal with a Nicolet 6700 FTIR spectrometer. The test results are shown in Fig. 9. The obvious peaks at 1610 cm^{-1} and 3046 cm^{-1} indicate the aromatic nucleus (C═C) structures and aromatic C–H structures, respectively. The peak at 2924 cm^{-1} means the aliphatic chains (CH3, CH2). The functions hydroxyl (OH), carboxyl (COOH) and carbonyl (C = O), appear at 3400 cm^{-1}, 1710 cm^{-1} and 1654 cm−1, respectively. And the quartz (Si═O) appears at 800 cm^{-1}.Fig. 10

3.1.3.4. Free radical. A great deal of research on the chemical structure of coal has revealed that free radicals exist in coal (Wang et al., 2018; Liu et al., 2014a,b), which has strong reactivity and may play an important role in coal utilization (Liu et al., 2015), and have great impact on

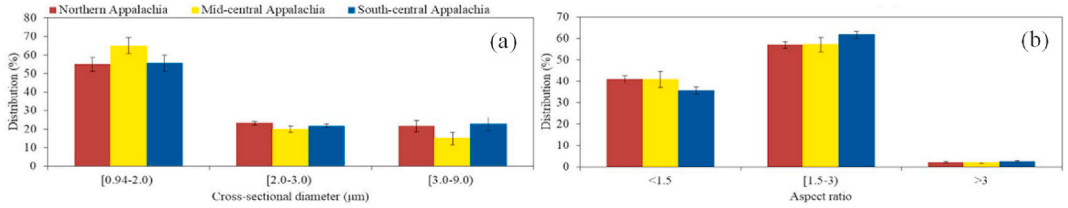

Fig. 8. Average dust properties. (a) Cross-sectional diameter of distinct mine regions; (b) aspect ratio of distinct mine regions (Johann-Essex et al., 2017b).

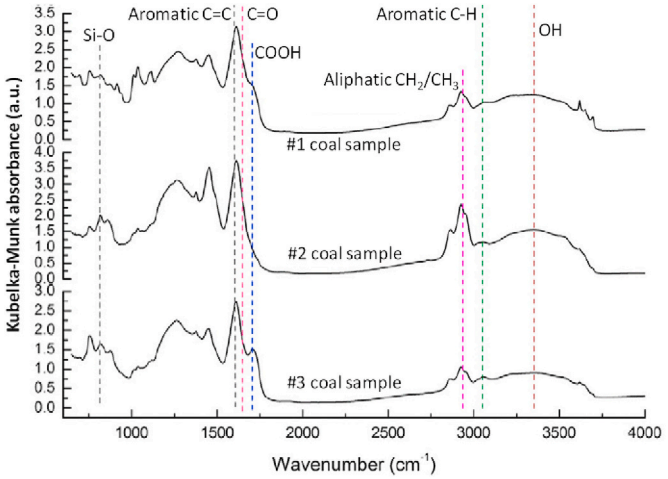

Fig. 9. FTIR spectra of different coal dust samples (Xu et al., 2017).

human health (Petsonk et al., 2013; Wang et al., 2018).

Ever since the electron spin resonance absorption in natural carbons was discovered (Uebersfeld et al., 1954; Ingram et al., 1954), a non-destructive technique of electron paramagnetic resonance (EPR) has been widely adopted to quantitatively characterize the free radicals in coal (Ingram et al., 1954; Liu et al., 2014a,b). With the EPR method, the EPR spectra of coal can be obtained.

Xu et al. (2017) tested the EPR spectra of coal during low-temperature oxidation from 30 to 230 °C. It showed that with the rise in oxidizing temperature from 30 °C to 230 °C, the Landes factor *g*-value increased slowly at first and was followed by a sharp decrease, and the maximum *g*-value occurred at 100 °C. For the free radical concentration N_g, it showed a constant increase with the increase of temperature from 30 °C to 230 °C.

3.1.4. Pore structure characterization

Pore structure, affecting the adsorption property, is one of the most important properties of coal. As to the effect of coal dust on human health, the adsorption property of coal is a critical factor because many toxic and harmful gases, elements, or minerals that may harm human health may be adsorbed in the coal pore.

According to the testing mechanism, methods for coal pore characterization can be divided into qualitative and quantitative methods. Among them, qualitative methods include Optical Microscope (OM, testing range, 10^3–10^5 nm), Scanning Electron Microscope (SEM, testing range, 10–10^5 nm), Transmission Electron Microscope (TEM, testing range, 10^0–10^3 nm), Atomic Force Microscope (AFM, testing range, 10^{-1}–10^3 nm), and Nano-CT (testing range, 10–10^5 nm). The quantitative methods contain Mercury Intrusion Porosimetry method (MIP, testing range, 10^2–10^5 nm), Low-pressure Nitrogen Gas Adsorption (LP-N_2GA, testing range, 2–100 nm), Synchrotron Small Angle X-ray Scattering/Small-Angle Neutron Scattering (SAXS/SANS, testing range, 1–100 nm), CO_2 Adsorption (testing range, 0.4–2 nm), and Nuclear Magnetic Resonance (NMR, testing range, 0–10^5 nm) (Qin et al., 2017).

Nie et al. (2015) tested the pore structure of eleven coal samples collected from a northern China mining area with low-pressure nitrogen gas adsorption (LP-N_2GA). Fig. 11 shows the pore size distribution of some of the coal samples. With the test results of LP-N_2GA, all the pore type, specific surface, volume and size distribution can be obtained.

3.2. Pathogenesis of coal dust-induced CWP

Coal pollutants affect all major human organ systems and contribute to four of the five leading causes of mortality in the U.S.: heart disease, cancer, stroke, and chronic lower respiratory diseases (Alan et al., 2009). For coal miners, the most common and major occupational health hazard is coal mine dust-related respiratory disease, which mainly includes coal workers' pneumoconiosis (CWP), progressive massive fibrosis (PMF), mixed-dust pneumoconiosis with coexistent silica exposure, chronic bronchitis, emphysema, and dust-related diffuse fibrosis (Perret et al., 2017), collectively termed as "coal mine dust lung disease" (CMDLD). Among these, the most common and classical CMDLD is CWP. In this section, we review the research on the pathogenesis of coal dust-induced CWP.

Coal workers' pneumoconiosis (CWP), also known as black lung

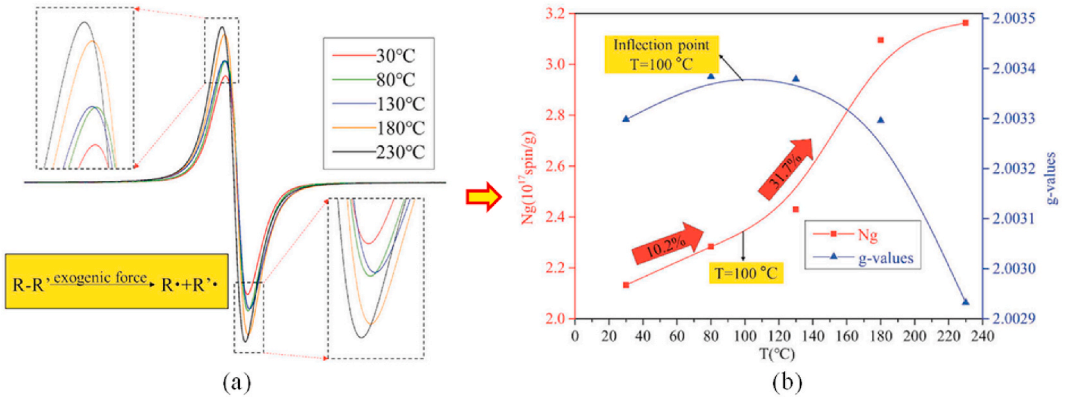

Fig. 10. EPR spectra and testing result of free radical. (a) EPR spectra; (b) testing result of free radical (C. Xu et al., 2017).

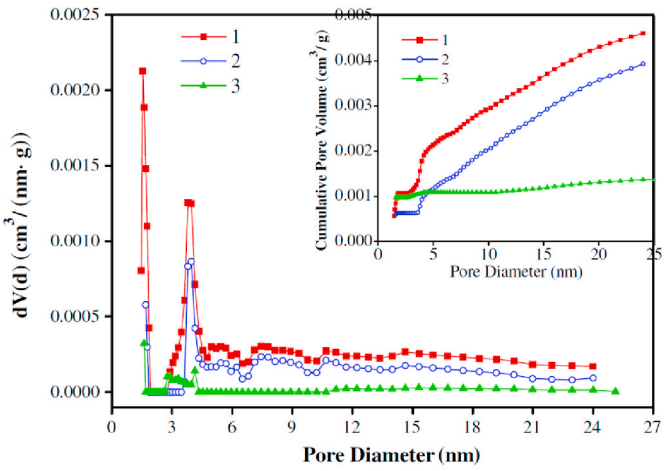

Fig. 11. Pore size distribution obtained by LP-N_2GA (Nie et al., 2015).

disease, is caused by long-term exposure to coal dust. It is a preventable but incurable lung disease that can be complicated by respiratory failure and death (Perret et al., 2017). After long-term exposure to coal dust, coal macules or opacities may appear on the lung; this consists of a collection of coal dust-laden macrophages that may be located in respiratory bronchioles walls or adjacent alveoli (Vallyathan et al., 1996).

Generally, CWP can be simple or complicated depending on the absence or presence of large opacities (Barber and Fishwick, 2016). Simple CWP is related to small nodules predominantly located at the upper zones of the lung, with the nodules in right part greater than in the left part (Barber and Fishwick, 2016; Seaman et al., 2015). In general, these coal macules can be visible on a chest X-ray. Fig. 12 shows imaging results of simple CWP. The radiograph with X-ray in Fig. 12(a) demonstrates that the nodules are predominantly located in the upper zone of the lung. However, these entities are radiographically indistinguishable. To identify these nodules more accurately, a coronal reformatted CT result [Fig. 12(b)] was obtained, and it confirmed that the coal macules mainly gather in the upper zone of the lung (Seaman et al., 2015). It was thought that without the presence of coexisting chronic obstructive pulmonary disease (COPD), simple CWP wouldn't be viewed as symptoms, physical signs, or abnormal physiology (Barber and Fishwick, 2016). And, the basis is that the profusion of small nodules on chest radiography in simple CWP has no significant relationship with the forced expiratory volume in 1 s (FEV_1) (Bauer et al., 2007). However, Blackley et al. (2015) argued that the all the FEV_1, FVC (forced vital capacity) and the FEV_1/FVC ratio showed decreasing trend with the profusion of opacities. According to the statistical results, they concluded that lung function progressively lowered across the range of small-opacity profusion. In regard to this point, further research should be conducted to get more conclusive results.

Unlike simple CWP, complicated CWP can be clinically disabling and potentially fatal (Seaman et al., 2015). Complicated CWP usually occurs on a background of simple CWP, but sometimes it also occurs de novo. Complicated CWP is often accompanied by cough, hemoptysis, pneumothorax, chronic interstitial pneumonia, etc., and in some cases, progression to hypoxia, right heart failure, and malignancy (Barber and Fishwick, 2016; Jun et al., 2013). Complicated CWP is generally defined by the presence of an opacity larger than 1 cm or larger, which can be found in Fig. 13 (the yellow arrows). Fig. 13(a) shows a background of upper-zone predominant small nodules. The arrows in the upper lung zones indicate the opacities which are larger than 1 cm. Fig. 13(b) and (c) show the coronal reformatted CT images, which confirm the background of upper-zone predominant centrilobular nodules (Seaman et al., 2015).

After the inhalation of coal dust, larger particles deposit on the large airways and the nose mucosa, and these deposited dusts are cleared through the transport of the mucociliary. Smaller coal particles are engulfed by alveolar macrophages and epithelium cells once they reach the alveoli. Because of particle migration and mediators released by primary target cells, other cells may also be influenced by the deposition of coal dust. Fig. 14 depicts the schematic map of major cellular pathways associated with the development of coal dust-induced lung disorders (Fig. 15).

The coal dust-laden macrophages will then migrate to the lymphatic, at which they will be eliminated. Because the particle clearance of the lymphatic in the upper posterior lung is the lowest, the coal dust will deposit there as much as possible. This may explain why the coal macules predominantly gather in the upper zone of the lung. With the agglomeration of coal dust, these mechanisms are overwhelmed rapidly. The inflammatory mediators released by alveolar macrophages generate extracellular matrix components, such as collagen, and stimulate fibroblasts, which lead to fibrosis. In addition, coal dust can directly result in epithelial damage, and lead to bronchitis and impaired ciliary clearance.

Mineral dust exposure is a complex interaction of reactive oxygen species, antioxidants, cytokines, growth factors, eicosanoids, proteases, and antiproteases, leading to lung dysfunction and pathology (Schins et al., 1999).

The above review of research on the pathogenicity mechanism of coal dust indicates that some issues needing further research remain. So far, research has mainly concentrated on pathological features caused by dust, and most research conclusions have been drawn from research into silica, asbestos, etc. However, the effects of physicochemical properties of coal particles—such as composition, functional group, particle size, particle shape, morphology, etc.—on CWP have been rarely reported. Besides, a great deal of literature has documented that nano-sized particles are more toxic due to their unique physicochemical properties and easier uptake by living organisms. And, one experimental study proved that when inhaled, specific sizes of nano-sized particles are efficiently deposited by diffusional mechanisms in all regions of the respiratory

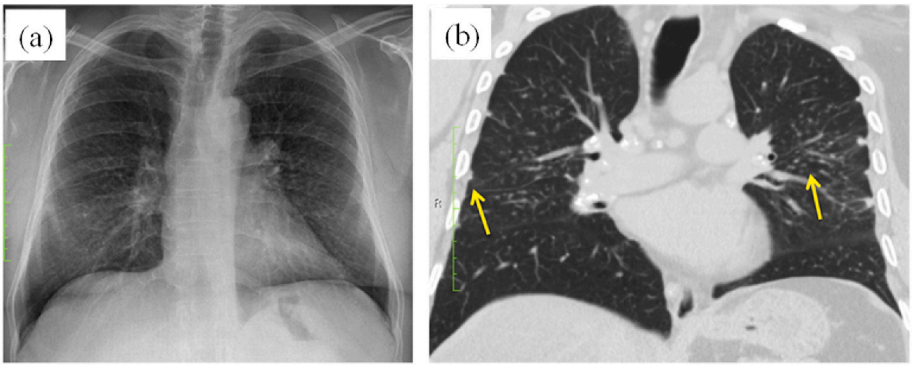

Fig. 12. Imaging results of simple CWP (Seaman et al., 2015).

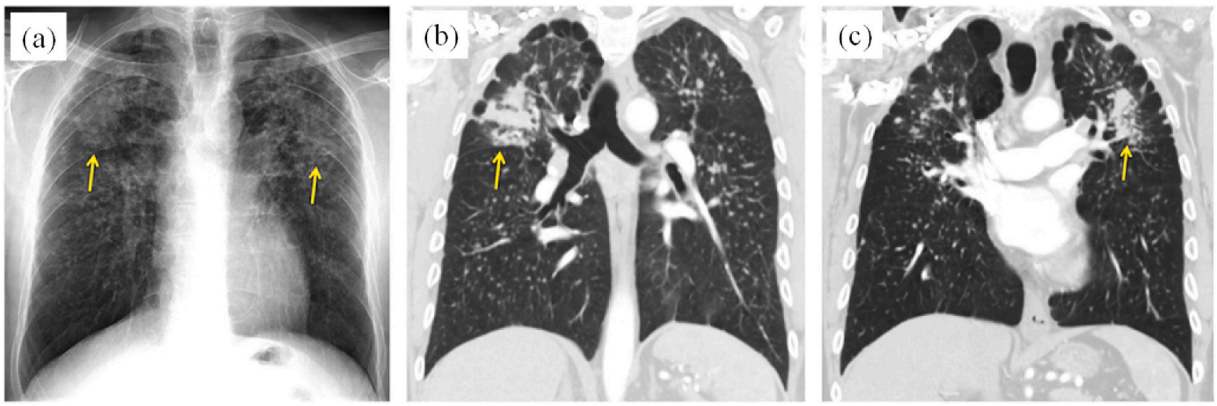

Fig. 13. Imaging results of complicated CWP (Seaman et al., 2015).

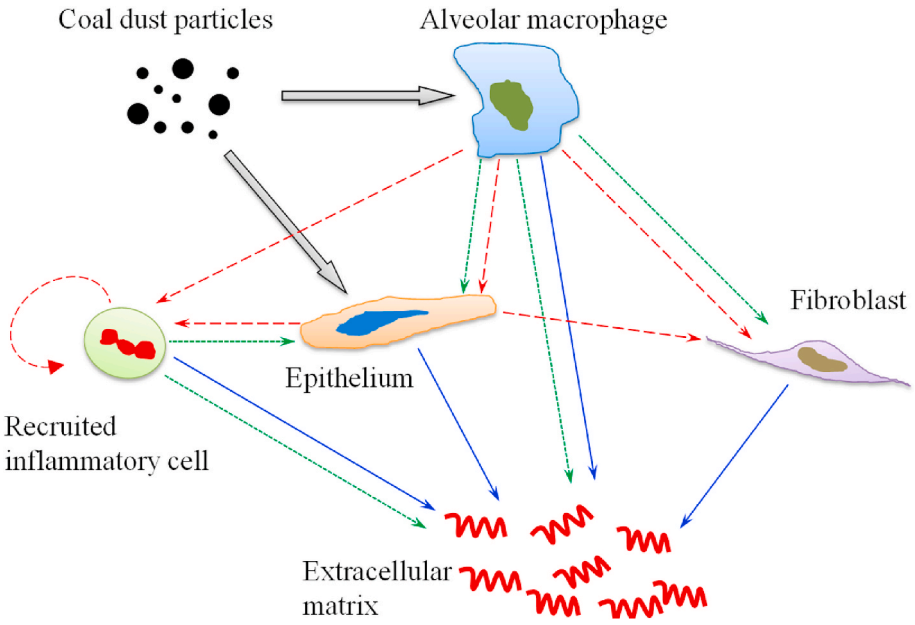

Fig. 14. Schematic map of major cellular pathways associated with the development of coal dust induced lung disorders (Schins et al., 1999).

tract. However, the toxicity of nano-sized coal particles to human tissues has rarely been reported. Therefore, it is reasonable to suspect that nano-sized particles may have contribute greatly to CWP.

4. Effects of nanoparticles on health

Much research has documented that human exposure to high level of airborne particle can lead to many diseases such as ischemic heart disease (IHD), cerebrovascular disease (stroke), chronic obstructive pulmonary disease (COPD) and lung cancer (LC), acute respiratory lung infection, etc. (Apte et al., 2015), and thus reduction of projected life expectancies by between 1 year and 5.5 years (Chen et al., 2013; WHO, 2003). Because of small size, fine particles can reach the lungs and alveolar regions, and ultrafine particles can enter the blood vessels (Song et al., 2016; Kampa and Castanas, 2008; Nemmar et al., 2002). Previous studies showed that there are positive and statistically significant relationships between exposure to $PM_{2.5}$ (Particulate matter with size <2.5 μm) and mobility and mortality (Pascal et al., 2014; Xing et al., 2016; Giannadaki et al., 2014; Weichenthal et al., 2014). In addition, much research has indicated that the adverse effect of particle matter is closely related to its size, and some researchers documented that because of its large surface area, nano-sized particles are more toxic than larger particles [122–125] (Maynard and Kuempel, 2005a,b; Dwivedi et al., 2012; Liu et al., 2013; Oberdörster et al., 2005). Since there are very few studies on the effects of nano-sized coal dust on human health, in this section, we will review the research into the adverse effects of particles with different sizes on human heath, and try to find the limitations of the current research, so as to provide a general direction for future research on the toxicity and pathogenesis of nano-sized coal dust.

4.1. Fine/ultrafine particles and health hazards

4.1.1. Global and regional morbidity and mortality attributable to $PM_{2.5}$

Some research has been conducted to gain an overall understanding of the global and regional distribution of morbidity and mortality attributable to $PM_{2.5}$ (Song et al., 2016, 2017; Wang et al., 2017). It has been reported that about 3.2 million deaths were attributed to $PM_{2.5}$, which ranked as the sixth largest risk factor for global premature mortality (Wang et al., 2017). The number of deaths attributed to $PM_{2.5}$ is greater than that caused by malaria and HIV-AIDS combined (totally reported to be 2.7 million). Apte et al. (2015) analyzed high-resolution (~10 km) estimates of mortality attributable to ambient $PM_{2.5}$ with the assistance of their spatially resolved model and concluded that 3.24 million premature death could be attributed to $PM_{2.5}$ in 2010. Fig. 15 shows the spatial distribution of estimated premature mortality attributable to $PM_{2.5}$ in 2010. For the three domains (northern America,

Europe and northern Africa, and Asia) analyzed, 3.1 million deaths were attributed to $PM_{2.5}$, which accounts for 97% of the global total. The population-weighted mean concentration of $PM_{2.5}$ is 38 μg/m^3 in Asia, which is significantly higher than those of northern Americas (12 μg/m^3) and Europe and northern Africa (19 μg/m3). Accordingly, the total mortalities attributable to $PM_{2.5}$ in the northern America, Europe and northern Africa, and Asian domains were 150,000, 680,000 and 2, 300,000, respectively. This indicates that the Asian domain had the highest number of deaths, accounting for 72% of total mortality attributable to $PM_{2.5}$. The high spatial $PM_{2.5}$ mortality density in the Asian domain is due to the interaction of high population density, high disease prevalence, and high levels of $PM_{2.5}$ in these areas.

Some work has also been conducted on the regional distribution of $PM_{2.5}$ and its resultant mortality (Song et al., 2016, 2017). Aiming to provide a better understanding of long-term mortality effects of $PM_{2.5}$ pollution in China, Wang et al. (2017) estimated $PM_{2.5}$-related premature mortality for five endpoints across China in 2010, with four sets of satellite-derived $PM_{2.5}$ concentration data and an integrated exposure-response model. They concluded that the premature deaths attributable to $PM_{2.5}$ nationwide amounted to 1.27 million, which includes 119,167; 83,976; 390,266; and 670,906 for adult chronic obstructive pulmonary disease, lung cancer, ischemic heart disease, and stroke, respectively, and 3995 deaths of acute lower respiratory infections in children under the age of 5. Fig. 16 shows the concentration distribution of $PM_{2.5}$ and its related mortality. It can be seen that the spatial distribution of deaths is roughly consistent with that of the particle concentration, indicating that the concentration of $PM_{2.5}$ has a great impact on mortality.

4.1.2. Nano-sized particle and its toxicity

Besides $PM_{2.5}$, much recent research has focused on the toxicity of nano-sized particles and its impact on human health (Maynard and Kuempel, 2005a,b; Oberdörster et al., 2005; Lam et al., 2004; Mls et al., 2017a,b). Human beings have been exposed to airborne nano-sized particles since their evolutionary stages, but such exposure has increased significantly since the industrial revolution because of the use of combustion engines, power plants, etc. The sources of nano-sized particles can be divided into natural and anthropogenic sources. Naturally generated aerosol particles are estimated to account for 65% to about 95% of the global aerosol mass burden of all particle sizes (Baltensperger and Nyeki, 1998). These particles are created during processes such as forest fires, volcanic emissions, sea spray, biogenic magnetite, etc. (Oberdörster et al., 2005; Maynard and Kuempel, 2005a, b). Anthropogenic activities have also led to the production of large amounts of nano-sized particles, and this source can be divided into unintentional and intentional ones. Unintentional source include the use of internal combustion engines, power plants, electric motors, smelting, welting, frying, broiling, grilling, etc. Intentional sources include the production of specific materials such as nano-spheres, -wires, -needles, and -tubes for certain functions (Oberdörster et al., 2005; Cass et al., 2000; Harrison et al., 2000).

Nanotechnology, a generic term encompassing the manipulation of matter at atomic- and near-atomic-length scales to produce new materials, structures, and devices, is one of the most important intentional sources for the production of nano-sized particles, of which at least one dimension is in nanometer scale. Carbon nanotubes are one of the typical nanomaterials which have won enormous popularity in nanotechnology for their unique properties and applications (Maynard and Kuempel, 2005a,b; Liu et al., 2013; Magrez et al., 2006; Bottini et al., 2006). To lead the U.S. into the next industrial revolution, in 2000, President Clinton established the National Nanotechnology Initiative; that one of its major objectives is to develop materials 10 times stronger than steel, but a fraction of the weight, for making all kinds of land, sea, air, and space vehicles lighter and more fuel efficient (White House, 2000).

Although nanotechnology is of great importance to current and future industries, its development is accompanied by many potential adverse effects. The research results of Cui et al. (2005) and Monteiro-Riviere et al. (2005) indicated that because of the hydrophobicity and tendency to aggregate, carbon nanotubes are harmful to living cells in culture. Bottini et al. (2006) investigated the toxicity of pristine and oxidized multi-walled carbon nanotubes on human T cells and concluded that oxidized, multi-walled carbon nanotubes were more toxic than the former, and led to massive loss of cell viability through programmed cell death at doses of 400 μg/ml. Magrez et al. (2006) tested the toxicity of multiwalled carbon nanotubes, carbon nanofibers, and carbon nanoparticles on lung tumor cells, and results indicated that all these materials were toxic, and that their hazardous effects were size-dependent. Moreover, cytotoxicity would be enhanced when the surface of the particles was functionalized. In addition to nanotechnology-produced materials, in other industries, there also exist some nano-sized materials which will do harm to human health. Much research has implied that coal fly ash contains large amounts of nano-sized particles which would do great harm to human respiratory tract, alveolar, DNA, etc. (Yu et al., 2009; Dias et al., 2014; Silva and Boit, 2011; Sambandam et al., 2014; Dwivedi et al., 2012). For example, Sambandam et al. (2014) studied the cytotoxicity of coal fly ash nanoparticles (with the average size of 9–50 nm) collected from a coal-fired power plant. Their conclusions indicated that cellular metabolism is inhibited in a dose-dependent manner by coal fly ash concentrations varying from 13 to 800 μm/ml. Dwivedi et al. (2012) investigated the toxicity of coal fly ash on human peripheral blood mononuclear cells and its resultant damage to DNA. In the comet and cytokinesis-blocked micronucleus assays, substantial genomic DNA damage in peripheral blood mononuclear cells treated with coal fly ash nanoparticles was observed. And about 1.8 and 3.6 strand breaks per unit of DNA were

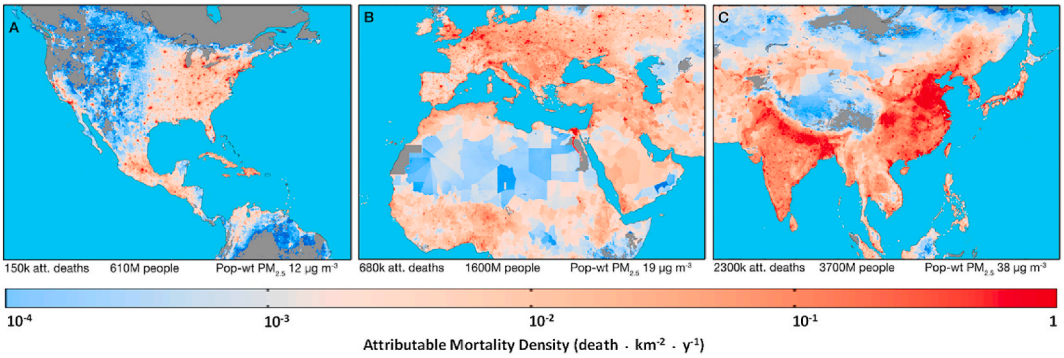

Fig. 15. Attributable premature mortality surfaces for $PM_{2.5}$ at 10 km resolution. (A) Northern Americas, (B) Europe and northern Africa, and (C) Asia; units for logarithmic color scale: premature deaths. Dark gray regions indicate areas without attributable mortality, due to ambient $PM_{2.5}$ below the theoretical minimum-risk concentration level or to unavailable input data (Apte et al., 2015).

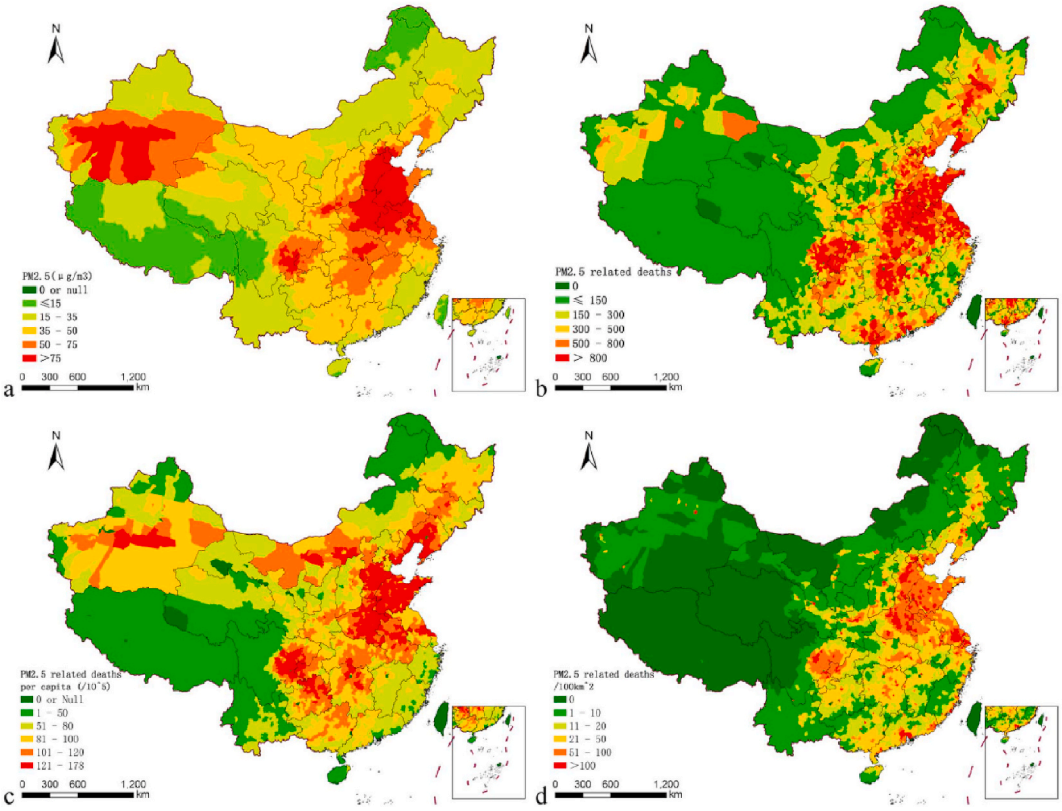

Fig. 16. PM 2.5 concentration and related mortality of China. (a) $PM_{2.5}$ concentration; (b) deaths attributed to $PM_{2.5}$; (c) deaths per capita ($/10^5$) attributed to $PM_{2.5}$; (d) deaths per 100 km^2 attributed to $PM_{2.5}$ (Wang et al., 2017).

estimated through alkaline unwinding assay at 1:100 DNA nucleotide/CFA ppm ratios with the aqueous and dimethyl sulfoxide extracts, respectively.

4.2. Physicochemical properties of nano-sized particles and its toxicity

Because of their specific physicochemical properties, ultrafine or nano-sized particles are thought to be more toxic and harmful to human beings. For example, for the small size, nano-size particles can more easily enter into the human body via the skin, digestive system, or respiratory tract, resulting in oxidative stress, inflammation, tissue damage, and diseases (Hochella et al., 2008; Fubini et al., 2007; Martinello et al., 2014; Alessandra et al., 2018). Also, due to the greater surface area of nano-sized particles, they are more environmentally active with respect to bio-uptake and associated health risks as compared to larger particles with the same chemical compositions [149]. And for greater surface areas, nano-sized particles carry considerable amounts of air toxins, which can harm human health [147, 149–150](Martinello et al., 2014; Binoy et al., 2018; Chow et al., 2005). Some research has indicated that the toxicity of particles is closely related to their size, surface area, surface chemistry, solubility, and shape (Maynard, A. D., & Kuempel, 2005a,b; Monteiroriviere et al., 2005; Sambandam et al., 2014; Dwivedi et al., 2012; Mls et al., 2017a,b). In this section, we reviewed the research on the effects of particle physicochemical properties on its toxicity.

4.2.1. Particle size

Many experiments on rats or human organ cells have verified that particle size not only influences its deposition, but also affects the biological responses (Maynard and Kuempel, 2005a,b; Oberdörster et al., 2005; Yu et al., 2009). This is most likely due, at least in part, to differences in surface and near-surface atomic structure, as well as crystal shape and surface topography as a function of particle size in the nanoscale (Hochella et al., 2008).

For particles with larger size, the inertial mechanism dominates its deposition behavior in the respiratory tract. In Fig. 17, when the size is greater than 1000 nm, most of the particles were deposited in the upper respiratory tract (head region). With the decrease of particle size, the deposition of the particles is controlled by the competition between inertia and Brownian diffusion, which leads to the particles penetrating deep into the lung. Competing deposition mechanisms lead to a minimum in deposition probability for particles between 100 nm and 1000 nm. For particles smaller than 100 nm, with the decrease of particle size, a higher deposition probability was observed, and deposition in Alveolar

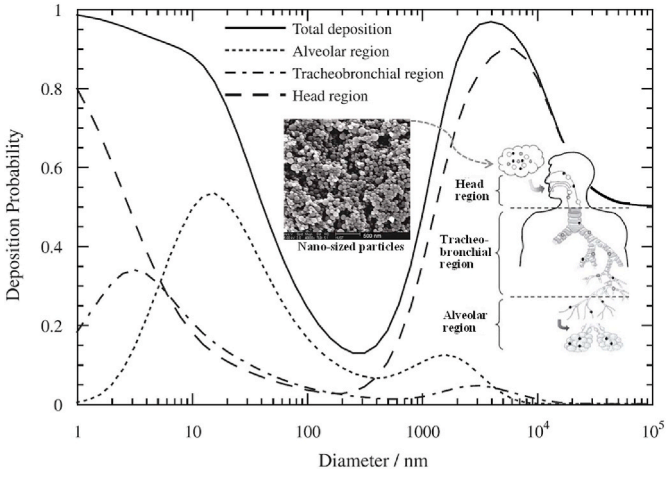

Fig. 17. Modeled total particle deposition probability in the respiratory tract, and deposition probability in the alveolar region. Modified from (Maynard and Kuempel, 2005a,b; Yu et al., 2009).

region dominated the whole deposition process, mainly due to the higher diffusion coefficient of smaller particles. In addition, this model also predicts up to 99% particle deposition within the respiratory tract at 1 nm (Maynard and Kuempel, 2005a,b; Oberdörster et al., 2005).

Since nano-sized particles can penetrate deep into the lungs, they have a higher inflammatory potential per given mass than do larger particles and can elicit severe acute lung injury, provided they have the same chemical composition (Oberdörster et al., 2005). The studies with ultrafine (~20 nm) and fine (~200 nm) titanium dioxide (TiO2) particles showed that ultrafine anatase TiO_2, when instilled intratracheally into rats and mice, penetrated the alveolar epithelial lining and entered the lung interstitium to a greater extent than an equal mass of larger respirable particles, and induced a much greater pulmonary-inflammatory neutrophil response than did fine anatase TiO_2 when both types of particles were instilled at the same mass dose (Oberdörster et al., 1992, 2005). In addition to nano-sized TiO_2 particles, other metals, such as aluminum oxide (Al_2O_3), gallium oxide (Ga_2O_3), cobalt, and nickel also presented a similar phenomenon (Ferin et al., 1991; Webb et al., 1986; Zhang et al., 2000a,b, 2003). Li et al. (2003)'s studies showed that in human bronchial epithelial cells and mouse alveolar macrophages, ultrafine particles less than 100 nm in size were observed to penetrate into cells, resulting in oxidative damage to mitochondrial membranes. Some studies also revealed that nano-sized particles can pass from the lungs into the bloodstream, which might lead to systemic health problems. For example, Takenaka et al. (2001)'s research results showed that inhaled ultrafine silver particles in rats were removed from the lungs to the blood circulation within 30 min, but if instilled into rat lungs, some agglomerates remained for at least 7 days.

4.2.2. Particle surface area

It was documented that with a reduction of particle size, the surface area of unit mass particle increases exponentially. Fig. 18 (a) shows the relationship between particle size and surface area. It shows that particles with a given size of 5000 μm corresponds to a surface area of 12 $\mu m^2/cm^3$, but when the particle size decreases to 5 μm, the corresponding surface area is 12000 $\mu m^2/cm^3$ (Oberdörster et al., 2005). With an increase in surface area, the ratio of surface to total atoms or molecules increases exponentially (Fig. 18 (b)).

According to results from Zhang et al. (2007), Silva and Boit (2011) inferred that nanoparticles can accommodate more adsorbates per unit of surface area than corresponding bulk material because the surfaces of the former are more structurally open for coordination of absorbates. Besides, adsorbates may bind with nanoparticles more strongly than with the corresponding bulk material because the binding energy for the former is higher than for the latter.'' If some toxic matters are absorbed to the surface of the nano-sized particles, then the toxicity of these particles may be enhanced. For example, 7-nm hematite shows a significantly increased sorption affinity for aqueous Cu2+ versus that of 25 nm and 88 nm hematite (Andrew et al., 2006).

With variations in surface and near-surface atomic structure as a function of size, one can anticipate a concomitant change in the chemical interactions of nano-sized particles with their environment (Hochella et al., 2008). Examples for the enhanced reactivity of nano-sized particles is that with 7-nm hematite nanocrystals as the catalyzer, the oxidation rate of aqueous Mn^{2+} can be improved for one to two orders of magnitude than with 37 nm hematite crystals (Madden and Jr, 2005); hydroquinone-driven reductive dissolution reactions of 5 × 64 nm goethite (a-FeOOH) are twice as fast compared with those of 22 × 367 nm goethite (Hochella et al., 2008).

The large surface area of nano-sized particles also confers themselves

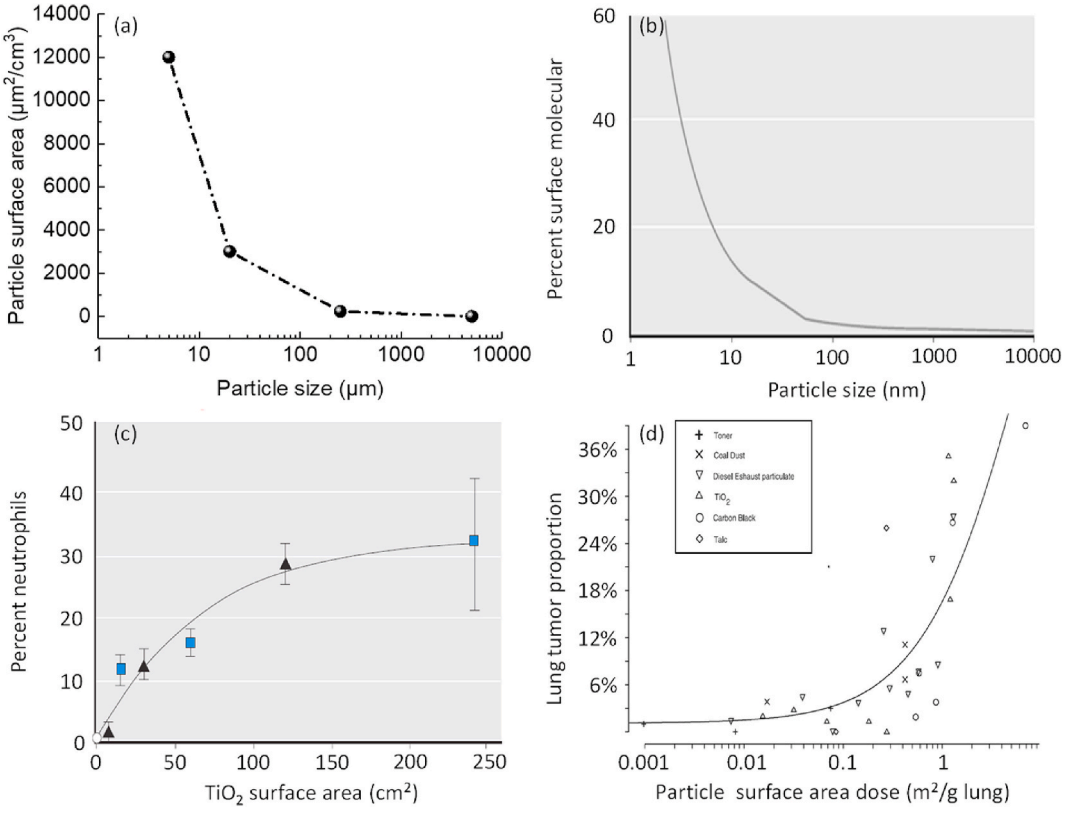

Fig. 18. Surface area of particles to its toxicity. (a) relationship between particle size and surface area; (b) variation of percent surface molecular with particle size; (c) percentage of neutrophils in lung lavage of rats as indicators of inflammation 24 h after intratracheal instillation of different mass doses of 20-nm and 250-nm TiO_2 particles in rats; (d) relationship between TiO_2 surface area dose in the lungs of rats after chronic inhalation to various types of poorly soluble low toxicity particles and tumor proportion (Maynard and Kuempel, 2005a,b; Oberdörster et al., 2005).

a greater biologic activity. Both Fig. 18 (c) and (d) indicate that particles with higher surface area will do more harm to organisms (Maynard and Kuempel, 2005a,b; Oberdörster et al., 2005), which may lead to negative and toxicity, induction of oxidative stress or of cellular dysfunction.

4.2.3. Particle solubility

Particle solubility is a crucial property in predicting the fate of particles and dissolved species in the environment, so as to determine the pathogenicity of the particle matters (Hochella et al., 2008; Binoy et al., 2018; Xing et al., 2016). With a decrease of particle size to nanoscale, the solubility of particles is generally expected to increase (Maynard and Kuempel, 2005a,b). A modified version of the Kelvin equation predicted that particle solubility depends on size, and with particles getting smaller in nanosize, solubility increases exponentially. This implies that nano-sized particles in a dissolution setting would have short lifespans (Hochella et al., 2008). However, in some cases, particle solubility reduces with the decrease of particle size (Tang et al., 2004). For example, mineral nanoparticles of lepidocrocite and FePO4·nH20 have been formed at lower Fe/P ratios in solution than predicted by their bulk solubilities (Sahai et al., 2007).

Poorly soluble particles, when retained in the lungs, can result in oxidative stress, which may either be caused by free radicals on the surface or by triggering the influx of defensive cells into the lungs, leading to inflammation, fibrosis, or cancer (Maynard and Kuempel, 2005a,b; Donaldson, 1998; Vincent, 1998; Stone et al., 2000; Zhang et al., 2000a,b). In a laboratory experiment, a single dose of solubilized arsenic trioxide was intratracheally instilled in rats, and a transient increase in total protein and albumin was observed; a persistent and progressive effect of crystalline silica was seen in this test (Lasfargues et al., 1995). In a chronic inhalation study, poorly soluble crystalline nickel subsulfide and nickel oxide was reported to cause an exposure-related increase in alveolar/bronchiolar neoplasms and adrenal medulla neoplasms in rats, while no excess neoplasms were observed in the rats exposed to the water-soluble compound nickel sulfate hexahydrate (Dunnick et al., 1995). These results imply that poorly soluble nano-sized particles may be more toxic and can do more harm to human health.

4.2.4. Particle shape

Engineered nano-sized materials may have very different shapes, such as tubes, fibers, spheres, rings, and planes. Fig. 19 (a)~(c) shows the SEM images of multiwalled carbon nanotubes, carbon nanofibers, and carbon nanoparticles. Much research has indicated that particle shape may have great impact on its toxicity. Toxicologic studies of spherical and fibrous particles have well established that natural and manmade fibers are associated with increased risks of pulmonary fibrosis and cancer after prolonged exposures (Oberdörster et al., 2005; Greim et al., 2001). Aspect ratio, for its simplicity, is generally adopted for describing the shape of nano-sized particles. With this parameter, fibers are generally defined as elongated structures with an aspect ratio of 1:3 or greater; while carbon nanotubes have aspect ratios of up to ≥100 (Oberdörster et al., 2005).

Kinloch (2008) documented that when introduced into the abdominal cavity of mice, carbon nanotubes would have asbestos-like pathogenicity because of their needle-like fiber shape, including inflammation and the formation of granulomas. Sato et al. (2005) indicated that 825 nm-long CNTs can cause more serious inflammation than 220 nm-long CNTs in vivo because macrophages could envelop 220 nm-long CNTs more readily. Fig. 19 (d) shows that when the nano-sized particles are short, they can be readily removed by macrophages, but when nano-sized particles are too long, the macrophages cannot completely engulf them, which may lead to cell death (Magrez et al., 2006). In Lam et al. (2004)'s experiment, three types of nanomaterials—namely carbon nanotubes, carbon black, and quartz—were intratracheally instilled into mice, and the results showed that mice lungs treated with carbon black were normal, those treated with high-dose quartz revealed mild to moderate inflammation, while lungs treated with carbon nanotubes revealed peribronchial inflammation and necrosis that had extended

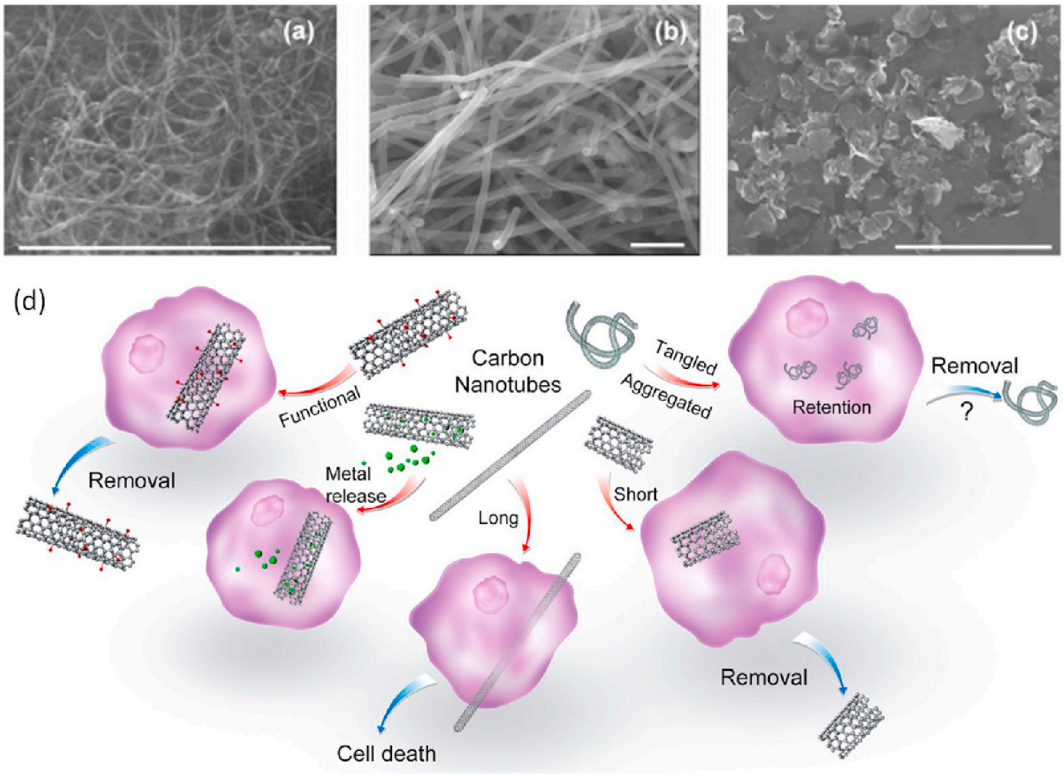

Fig. 19. Shape of different types of nano-sized materials and its pathogenesis. (a) multiwalled carbon nanotubes; (b) carbon nanofibers; (c) carbon nanoparticles; (d) particle aspect ratio affects its phagocytosis and cytotoxicity (Liu et al., 2013; Magrez et al., 2006).

into the veolar septa. This conclusion is analogous to that of Bottini et al. (2006) (see Fig. 20). All of these results show that nano-sized particles with larger aspect ratio can be more toxic to biosome. However, the research results of Magrez et al. (2006) showed that the toxicity of multiwalled carbon nanotubes (an average diameter of 20 nm and aspect ratios ranging from 80 to 90), carbon nanofibers (a mean diameter of 150 nm and aspect ratios of 30–40) and carbon black with an aspect ratio about 1 presented a toxicity order of carbon black > CNFs > MWCNTs, which is diametrically opposed to the above conclusion. This difference may be resulted from impurities, chemical characteristics, or surrounding microenvironment and analytical methods (Liu et al., 2013).

4.2.5. Particle surface chemistry

The surface chemistry properties of nano-sized particles have a close correlation with their potential toxic effects. Coal-derived nano-sized particle is a common type of airborne particle matters in the atmosphere and working space. The most common hazardous elements observed in these particles are Al, Cr, Fe, K, Mg, Ni, Si, Ti, and Zr although other toxic trace elements such as As, Be, Cd, Co, Cu, Hg, Li,Mn,Mo, Pb, Se, U, Th, V, and Zn may also be present (Martinello et al., 2014). These elements may have complex toxicity. The element S often exists on the surface of nano-sized particles in the form of sulfuric acid, sulfurous acid or sulfates, which have a high biologic activity, and can lead to depression of pulmonary particle clearance and asthma (Amdur, 1989). Wise et al. (2008) demonstrated that cells treated with Cr(VI) exhibit a series of DNA damage containing base modification, single-strand breaks, double-strand breaks, Cr–DNA adducts, DNA–Cr–DNA adducts, and protein–Cr–DNA adducts. Other transition metals such as V, Cu, Fe, Mn and Pt act as catalysts in the formation of reactive oxygen species (ROS) which are associated with direct molecule impairment and lead to atherosclerosis, cancer and neurodegenerative diseases (Lighty et al., 2000; Aruoma, 1998). Pyrite in coals has been documented to spontaneously generate ROS, such as hydrogen peroxide (H_2O_2) and hydroxyl radicals (•OH) when placed in water (Cohn et al., 2004, 2005), which explain that aqueous pyrite slurries degrade yeast RNA, ribosomal RNA and DNA (Cohn et al., 2006).

Laboratory studies demonstrated that pulmonary inflammation was much higher in rats exposed to crystalline silica than in those exposed to fine or ultrafine TiO2 particles (Oberdörster et al., 2016; Duffin et al., 2002). This is because that crystalline silica is highly reactive with biological membranes through hydroxyl radical-mediated lipid peroxidation, which can result in cell death (Vincent, 1998). The differences in pulmonary inflammation and cytotoxicity caused by different particles is related to the particle surface free radical activity (Zhang et al., 1998), for example, in a rat instillation study, the inflammatory response to four different types of ultrafine particles (carbon black, cobalt, nickel, and titanium dioxide) was consistent with their measured free radical generation (Dick et al., 2003).

Surface modification was able to change the interaction process of CNTs with the cellular lipid bilayer, which further changed the cell uptake viability (Liu et al., 2013). Bottini et al. (2006) compared the toxicity of pristine and oxidized multi-walled carbon nanotubes on human T cells and the results are depicted in Fig. 20. It can be seen that the oxidized CNTs were shorter and straighter than the pristine CNTs. And at 400 μg/ml, oxidized CNTs caused a loss of >80% of the cells within 5 days, while pristine CNTs killed less than half. This is because that the oxidized CNTs are more hydrophilic and are better dispersed in aqueous solution and therefore reach higher concentrations of free CNTs. In addition, Duffin et al. (2002) showed that by modifying the surface of the quartz, the inflammation response was reduced to that observed for the particles with relatively low toxicity. To investigate the effect of surface chemical properties on toxicity, Magrez et al. (2006) decorated the surface of multiwalled nanotubes by acid treatment. This process introduced carbonyl (CdO), carboxyl (COOH), and/or hydroxyl (OH) groups onto the nanotube and nanofiber surfaces. The results showed that the toxicity increases with the chemical surface treatment.

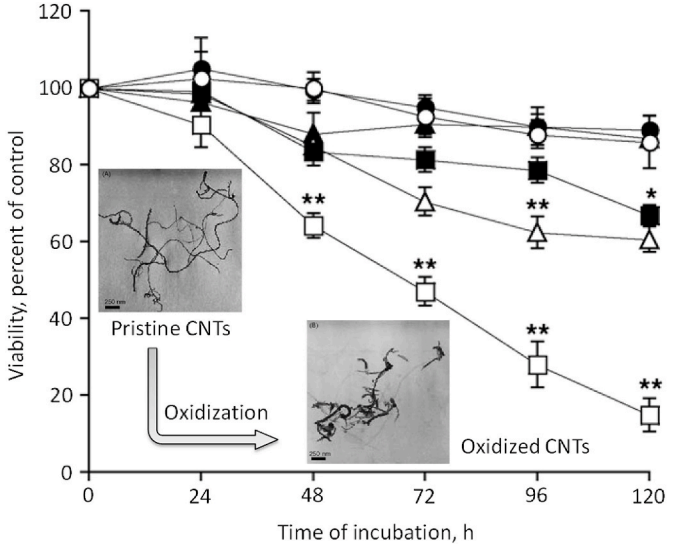

Fig. 20. Dose- and time-dependent reduction in human T-cell viability by CNTs. The graph shows the viability of T-cells incubated with 1 ng/cell (closed symbols) or 10 ng/cell (open symbols) of CB (circles), pristine CNTs (triangles) or oxidized CNTs (squares) for the indicated periods of time (Bottini et al., 2006).

4.3. Pathogenic mechanism of nano-sized particles

Up to date, most of the toxicity research on NSP carried out in mammalian systems has focused on respiratory system exposures for testing the hypothesis that airborne ultra-fine particles cause significant health effects. However, to nano-sized particles, other exposure routes, such as skin and GI tract, should also be considered as potential portals of entry (Oberdörster et al., 2005).

In order to figure out the pathogenic mechanism, it is crucial to know the deposition and the subsequent fate of the airborne particles, which is particle size-dependent, and has been discussed in Fig. 17. In this part, we focus on the deposition of the nano-sized particles. As demonstrated above, for the nano-sized particles, diffusion is the main mechanism dominate the particle deposition, and other mechanisms which are critical to large particles do not contribute to nano-sized particle deposition. Once deposited, NSPs appear to translocate readily to extrapulmonary sites and reach other target organs by different transfer routes and mechanisms. One involves transcytosis across epithelia of the respiratory tract into the interstitium and access to the blood circulation directly or via lymphatics, resulting in distribution throughout the body. The other involves their uptake by sensory nerve endings embedded in airway epithelia (Oberdörster et al., 2005).

Because there has been no research on the pathogenic mechanism of nano-sized coal dust, in this section, we will review the studies on the pathogenic mechanism of other nano-sized particles, such as carbon nanotubes, metal nanoparticles, fibers et al., which may provide a reference for the study of nano-sized coal dust.

After carbon nanotubes enter the body, the underlying mechanisms of toxicity are manifested as oxidative stress, inflammatory responses, malignant transformation, DNA damage and mutation, formation of granuloma, and interstitial fibrosis (Oberdörster et al., 2005). Oxidative stress caused by carbon nanotubes has been regarded as the most acceptable mechanism. Oxidative stress from particles and inflammatory cells also can cause the guanine to adduct 8-hydroxy deoxy guanosine (8-OH-dG) which was thought to be important in carcinogenesis by particles (Schins, 2002; Schins et al., 2002; Ken et al., 2006). According to Kane (1996), Fibers such as asbestos caused fibrosis and

cancer could be also attributed to oxidative stress from the fibers or the inflammatory response causing 8-OH-dG adducts leading to mutation. In addition, the increased intracellar ROS which either induced by transition metals released from carbon nanotubes or the high surface area of the carbon nanotubes, can react with cellular macromolecules including DNA, proteins, and lipids and disturb the homeostasis of the intracellular milieu [186]. Besides, Maricica et al. (2008) documented that carbon nanotubes could activate the specific molecular signaling associated with oxidative stress, including activator protein-1 (AP-1) and nuclear factor κB (NF-κB) and MAPK. These molecular signaling led to the release of proinflammatory cytokines together with the depletion of antioxidant defenses, poly (ADP-ribose) polymerase1 (PARP-1), protein p38, and protein serine-threonine kinase (Akt). Fig. 21 shows the mechanisms of cell damage and inflammation induced by carbon nanotubes.

Apoptosis has been viewed as a major mechanism of cell death caused by nano-sized particles induced oxidative stress (Hsin et al., 2008; Borm et al., 2006). Among these different apoptotic pathways, the intrinsic mitochondrial apoptotic pathway played a major role in metal oxide nano-particles induced cell death since mitochondria are one of the major target organelles for NP-induced oxidative stress (Xia et al., 2006). High levels of ROS in the mitochondria can cause damage to membrane phospholipids (Lenaz, 2010). Various metal oxide nano-particles including Zn, Cu, Ti, and Si elicit ROS-mediated cell death via mitochondrial dysfunction (Manke et al., 2013). DNA is one of the major targets for oxidative stress and represents the first step involved in mutagenesis, carcinogenesis, and aging. ROS/RNS (reactive oxygen species/reactive nitrogen species) damage DNA through DNA strand breaks, DNA protein cross-links, and alkali-labile sites (Kawanishi et al., 2002; Shi et al., 2004), and given their characteristic nature, free radicals appear as one of the likely carcinogens (Wiseman and Halliwell, 1996).

By activating the MAPK and NF–B signaling pathways which control transcription of inflammatory genes such as IL-1, IL-8, and TNF-, uncontrolled generation of ROS can trigger a cascade of proinflammatory cytokines and mediators (Thannickal and Fanburg, 2000). Moon et al.'s (2010) study showed that a coexposure of metal oxide NP with a bacterial endotoxin could lead to exaggerated lung inflammation and pulmonary edema. In addition, studies with various metal oxide nano-sized particles have indicated ROS-mediated inflammatory response. For example, SiO2 and TiO2 nanoparticles cause an elevated inflammatory response through ROS generation (Park et al., 2008, 2009).

5. Discussion

Dust generated during coal mining operations includes particles small enough to be deposited in the airways and the gas-exchange region of a miner's lung, when inhaled. Chronic exposure to those particles, referred to as respirable coal mine dust, puts miners at risk for various lung diseases, including coal workers' pneumoconiosis, emphysema, silicosis, and chronic bronchitis (National Academies of Sciences, Engineering, and Medicine, 2018). Up to now, there is no effective medical treatment can be adopted to reverse these coal dust-induced lung diseases or control their progression. The only way to prevent these diseases is to minimize the coal dust exposure along with medical surveillance for early disease detection. Based on this consideration, a series of efforts have been made by U.S. federal regulations over the years. In 1969, a limit of 3.0 mg/m^3 of respirable coal mine dust was established. And in 1972, this limit was lowered to 2.0 mg/m^3. In 2014, the Mine Safety and Health Administration (MSHA) issued a dust rule, which went into effect in August 1, 2016, that reduced the limit to 1.5 mg/m^3 (National Academies of Sciences, Engineering, and Medicine, 2018). The implementation of these regulatory requirements contributed to several decades of decreased prevalence of CWP in underground coal miners. The recognized prevalence of disease in underground coal miners with more than 25 years of work tenure decreased from more than 30 percent in 1970 to about 5 percent in 2000. However, since around 2000 there has been an unexpected increase in the proportion of CWP in coal miners with 25 or more years of work tenure, and with an increase or plateau of disease prevalence in those with shorter mining tenure (Laney and Weissman, 2014). Moreover, recent studies have described rapidly progressive, severe and fatal forms of disease including progressive massive fibrosis occurring mainly in the central geographic region of Appalachia (Blackley et al., 2018a,b; National Academies of Sciences, Engineering, and Medicine, 2018).

There are likely a number of factors that have contributed to an increase in the prevalence and severity of coal dust related lung diseases. Determining the causes of that increase and eliminating occupational lung disease in coal miners is a complex scientific, engineering, medical, regulatory, social, political, economic and legal problem [204].

The NIOSH study demonstrates that the prevalence of the CWP is coal-rank dependent following high-rank coal > low-rank coal > medium-rank coal (Attfield, 2011). The reason why CWP is highly rank dependent has not been studied. The high rank coals termed as anthracites are more brittle than medium and low rank coals, which may produce more nano-sized coal dusts. To our best knowledge, the toxicity of the nano-sized coal dusts has never been studied. However, recent studies have confirmed that nano-sized particles are more toxic due to their unique physicochemical properties and easier uptake by living organisms (Magrez et al., 2006; Jean-Pierre et al., 2004). In addition, some studies have proved that nano-sized coal dusts do exist in the respirable coal dust produced during the coal production [93, 111, 206] (Barone et al., 2017; Johann-Essex et al., 2017b; Bäckström et al., 2015). Therefore, it is reasonable to deduce that the nano-sized coal dust contributes to the coal dust-induced lung diseases.

However, to our best knowledge, there is no research on how the nano-sized coal dusts affect the human health and how there induce CWP and other coal dust-related lung diseases. In this section, we try to put forward the problems corresponded to this topic which calls for further exploration.

5.1. Physicochemical properties of nano-size coal dust

As discussed above, the physicochemical properties of nano-sized particles have great effects on their toxicity. The NIOSH study demonstrates that the prevalence of the CWP is coal-rank dependent following high-rank coal > low-rank coal > medium-rank coal (Attfield, 2011). A probable reason for this phenomenon is that different ranks of coal have different physicochemical properties, including coal dust size distribution after crushing, specific surface area, composition (minerals, elements), surface functional group, solubility, particle shape, particle stiffness, hydrophility, surface charge, aggregation, and so on. For nano-sized coal dusts, the distribution of these properties and their evolution with particle size are still unclear. For example, a modified version of the Kelvin equation predicts solubility dependence on size, stating that as particles get smaller through the nanorange of sizes, their

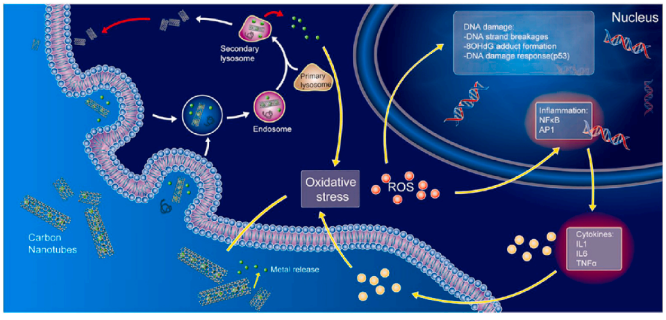

Fig. 21. The mechanisms by which carbon nanotunes induce cell damage and inflammation (Oberdörster et al., 2005).

solubilities increase exponentially, but certain minerals are known to become less soluble as they get smaller (Hochella et al., 2008). Then for nano-sized coal dusts, how will the solubility change with its size? And for nano-sized coal dusts with different ranks, will the regularity keep consistent? All of these need further investigations.

Therefore, advanced techniques such as XRD, FTIR and so on and so forth, should be adopted to get a systematic and comprehensive understanding of the physicochemical properties of nano-sized coal dusts.

5.2. Toxicity and pathogenesis of nano-sized coal dust

Although toxicity of some nanoparticles such as carbon nanotubes, carbon fibers has been well studied, and several pathogenesis of these particles have been put forward, unfortunately, no related research on nano-sized coal dust has been reported so far. However, these research is of great importance to understand the relationship between the nano-sized coal dust and the coal dust-related lung diseases.

Therefore, studies about the toxicity of nano-sized coal dusts should be conducted on levels of cells, tissues, organs and living animals. Based on these studies, a reliable pathogenesis needs to be proposed. To study bio-effect of nano-sized mine dust, we can treat normal lung epithelial cells (BEAS-2B, ATCC) and lung carcinoma cells (NCI–H441, ATCC) in cell culture with both organic and inorganic constituents at a different dosage for each nano-sized coal dusts. The uptake of the nano-sized coal dusts will be observed using optical microscopy and transmission electron microscopy. The current knowledge in nanomedicine predicts nanoparticles of the same materials of microparticles will be more readily taken up by cells through endocytosis. It will be of high interest to investigate if coal mine dusts follow the same rule. We will further measure the cell viability using LIVE/DEAD and caspase activity assay (ThermoFisher). The tumorigenic, migration, and invasion capability of the tumor cells before and after treatment will be measured by colony forming assay, scratch assay, and transwell assay, respectively. To study the effect of nano-sized coal dust with lung epithelium at the organ level, we will adopt a lung-on-chip model and measure surfactant production and alveolar barrier permeability to gauge potential change of the lung function.

5.3. Real-time monitoring technology and equipment for nano-sized coal dust

Another challenge for the mining industry is the lack of a real-time dust monitoring system at the mine environment. One commercial personal dust monitor (PDM) was developed by NIOSH to measure respirable coal mine dust mass to provide accurate exposure data for each shift (Page et al., 2008). However, weighted at ~7.5 lbs, the current PDM is bulky and heavy. According to feedback from miners, they feel uncomfortable and some of them do not want to wear due to this. Another aspect that hinders further in-situ dust detection is that PDM can only analyze overall weight concentration of dust (Halterman et al., 2017), but it is impossible to characterize dust composition and their respective proportions. Thus, a miniaturized real-time dust monitoring system that is capable of characterizing dust size and composition is highly desirable. Coal fluorescence is a property reflecting the characteristic of the conjugated double bond in its macromolecular structure (Crelling, 2011). The main components of the coal dust include carbonaceous, silicate, quartz, carbonate, heavy mineral. These components can significantly influence the fluorescence spectra, meaning that fluorescence spectroscopy can be an effective tool to interrogate the compositions of coal.

Therefore, developments of new instruments and devices that can simultaneously quantify dust concentration and composition based on fluorescence spectroscopy will be a step forward to monitor the dust concentration for future mining application. The overall size and weight of the developed device should be much smaller than the NIOSH PDM which will promote coal mine workers' willingness to wear.

5.4. Exposure control technology and equipment

Considering the adverse effects of coal dusts on coal workers' health, considerable research has been carried out to develop coal dust control technologies. In underground mines, the most common and traditional methods are ventilation and water spray with plain water. However, with the introduction of fully mechanized longwall mining method to coal mining industry, these traditional methods were thought to be inadequate to meet the coal dust control standard. Therefore, increasing attention was paid to research related to surfactant aided water spray coal dust suppression technology (Xu et al., 2018). These technologies are thought to be effective in removing large particles from the working environment and contribute a lot to coal mine workers' health. However, with a decrease of the coal dust size, these technologies become less effective.

To create a safe and comfortable working environment for coal mine workers, new technologies which can be used to effectively remove the nano-sized coal dust from the working environment should be developed. When addressing a potentially harmful exposure within a workplace, a hierarchical approach should be considered to reduce risk. This includes the prevention or containment of hazardous workplace emissions at their source, the removal of emissions from the pathway between the source and the worker, and lastly, control of worker exposure with barriers between the worker and the hazardous work environment (Maynard and Kuempel, 2005a,b). This means that all methods used at the working face, in the roadway, and on the workers should be developed.

5.5. Development strategy and policy guarantee

As the second largest energy source in the world, coal plays an important role in the global energy profile. In 2017, coal accounted for 27% of primary energy, and the coal power generation accounted for 38% of the whole generation capacity of the world. Currently, the major coal-producing countries includes China, India, USA, Australia, Indonesia, Russia, South Africa, Germany, Poland and Kazakhstan. For these countries, the issue that the impact of coal dust on miners' health is a universal and urgent problem that each nation is dealing with. In response to this challenge, in terms of research, the international collaboration is suggested to be a good model to allow all partners share the resources toward a common goal of mitigating this challenge.

In addition, the prevention and control of health effects of coal dust belongs to the public welfare entity, therefore, the various government agencies from various coal production countries should consider to provide dedicated funds for future scientific studies through the implementation of major research projects, formulation relevant policies and regulations. In addition to the governmental support, the coal production enterprises should adopt the best field practices to mitigate the respirable hazard for coal mines.

Finally, the issue that the effect of coal dust on human health involves a series of process, including coal dust production, migration, interaction between the coal dust and human organs, and is a multidisciplinary subject intersected by Mining, Nanoscience, the Science of Occupational Health and Medical Science, and Engineering Design. For solving this problem, a multidisciplinary research approach should be adopted to advance the understanding of the issue which will be the future basis of providing the effective engineering solution for future mining practice.

Overall, the prevention and control of health effects of coal dust is a complex system engineering problem which is on the cross-edge of many disciplines, and it calls for joint efforts from international society, governments and researchers.

6. Summary

We reviewed the coal dust related health studies, coal mine dust

property and its effects on miners' health and safety, and the effects of nanoparticles on human health. Although a series of regulations have been formulated, and a lot of measures have been taken to lower the exposure of coal mine workers to coal dust, recent years have witnessed an unexpected increase of the CWP prevalence in U.S.. The reasons for this phenomenon involve many aspects and may be complicated. Considering the toxicity of nanoparticles such as carbon nanotubes and carbon nanofibers in other industries, it is reasonable to deduce that the nano-sized coal dust contributes to the increase of the CWP prevalence observed in recent years. However, research on relationship between the nano-sized coal dust and the coal dust-induced lung diseases has not been reported. To address these problems, in the future studies, the following issues should be further investigated. They are: 1) Systematic characterization of physicochemical properties of nano-size coal dust; 2) Toxicity and pathogenesis of nano-sized coal dust; 3) Development of real-time monitoring technology and equipment for nano-sized coal dust; 4) Development of exposure control technology and equipment; and 5) adoption of new technology for real time dust monitoring to effectively reduce the dust exposure for future miners. In addition, in response to this challenge, the international collaboration is suggested to be a good model to allow all partners share the resources toward a common goal of mitigating this challenge.

Declaration of competing interest

On behalf of all authors, the corresponding author states that there is no conflict of interest.

Acknowledgement

This work is financially supported by The National Institute of Occupational Safety and Health (NIOSH) under the contract No. NIOSH-75D30119C05128.

References

Ahmed, M.M., 2010. Effect of comminution on particle shape and surface roughness and their relation to flotation process. Int. J. Miner. Process. 94 (3-4), 180–191.
Amdur, M.O., 1989. Health effects of air pollutants: sulfuric acid, the old and the new. Environ. Health Perspect. 81 (2), 109–113.
Andrew, S. Madden, Hochella Jr., Michael F., Luxton, Todd P., 2006. Insights for size-dependent reactivity of hematite nanomineral surfaces through Cu^{2+} sorption. Geochem. Cosmochim. Acta 70, 4095–4104.
Antao, V.C.D.S., Petsonk, E., Sokolow, L., Wolfe, A., Pinheiro, G., Hale, J., et al., 2005. Rapidly progressive coal workers' pneumoconiosis in the United States: geographic clustering and other factors. Occup. Environ. Med. 62 (10), 670–674.
Apte, J.S., Marshall, J.D., Cohen, A.J., Brauer, M., 2015. Addressing global mortality from ambient pm2.5. Environ. Sci. Technol. 49 (13), 8057–8066.
Aruoma, O.I., 1998. Free radicals, oxidative stress, and antioxidants in human health and disease. J. Am. Oil Chem. Soc. 75 (2), 199–212.
Attfield, M., 2011. Coal Mine Dust Exposures and Associated Health Outcomes.
Bäckström, D., Dan, G., Pushp, M., et al., 2015. Particle composition and size distribution in coal flames – the influence on radiative heat transfer. Exp. Therm. Fluid Sci. 64, 70–80.
Baltensperger, U., Nyeki, S., 1998. Atmospheric aerosols. In: Colbeck, I. (Ed.), Physical and Chemical Properties of Aerosols. I. Colbeck. Blackie Academic & Professional, London, pp. 280–330.
Barber, C., Fishwick, D., 2016. Pneumoconiosis. Medicine 44 (6), 355–358.
Barone, T., Hesse, E., Seaman, C.E., et al., 2017. Real-time Sizing of Airborne Coarse Coal Dust. North American Mine Ventilation Symposium.
Bauer, T.T., Heyer, C.M., Duchna, H.W., et al., 2007. Radiological findings, pulmonary function and dyspnea in underground coal miners. Respiration 74 (1), 80–87.
Beeckmans, J.M., 1965. The deposition of aerosols in the respiratory tract. I. mathematical analysis and comparison with experimental data. Can. J. Physiol. Pharmacol. 43 (3), 157–172.
Binoy, K., Saikia, Jyotilima Saikia, Rabha, Shahadev, Silva, Luis F.O., Finkelman, Robert, 2018. Ambient nanoparticles/nanominerals and hazardous elements from coal combustion activity: implications on energy challenges and health hazards. Geoscience Frontiers 9, 863–875.
Blackley, D.J., Laney, A.S., Halldin, C.N., Cohen, R.A., 2015. Profusion of opacities in simple coal worker's pneumoconiosis is associated with reduced lung function. Chest 148 (5), 1293–1299.
Blackley, D.J., Reynolds, L.E., Short, C., et al., 2018a. Progressive massive fibrosis in coal miners from 3 clinics in Virginia. J. Am. Med. Assoc. 319, 500.
Blackley, D.J., Halldin, C.N., Scott, L.A., 2018b. Continued increase in prevalence of coal workers' pneumoconiosis in the United States, 1970–2017. Am. J. Publ. Health e1–e3.
Borm, P.J., Robbins, D., Haubold, S., et al., 2006. The potential risks of nanomaterials: a review carried out for ecetoc. Part. Fibre Toxicol. 3 (1), 11.
Bottini, M., Bruckner, S., Nika, K., et al., 2006. Multi-walled carbon nanotubes induce t lymphocyte apoptosis. Toxicol. Lett. 160 (2), 121–126.
Cass, G.R., Hughes, L.A., Bhave, P., Kleeman, M.J., Allen, J.O., Salmon, L.G., 2000. The chemical composition of atmospheric ultrafine particles. Phil. Trans. Biol. Sci. 358 (1775), 2581–2592.
Chen, Y., Ebenstein, A., Greenstone, M., Li, H., 2013. Evidence on the impact of sustained exposure to air pollution on life expectancy from China's huai river policy. Proc. Natl. Acad. Sci. Unit. States Am. 110 (32), 12936–12941.
Chow, J., Watson, J., NoraSavage, Jacksolomon, C., Cheng, Yung-Sung, Mcmurry, P., et al., 2005. Nanoparticles and the environment. J. Air Waste Manag. Assoc. 55 (10), 1411–1417.
Cohen, R.A., Petsonk, E.L., Rose, C., et al., 2016. Lung pathology in U.S. coal workers with rapidly progressive pneumoconiosis implicates silica and silicates. Am. J. Respir. Crit. Care Med. 193 (6), 673–680.
Cohn, C.A., Borda, M.J., Schoonen, M.A., 2004. RNA decomposition by pyrite-induced radicals and possible role of lipids during the emergence of life. Earth Planet Sci. Lett. 225 (3), 271–278.
Cohn, C.A., Pak, A., Strongin, D., Schoonen, M.A., 2005. Quantifying hydrogen peroxide in iron-containing solutions using leuco crystal violet. Geochem. Trans. 6 (3), 1–5.
Cohn, C.A., Mueller, S., Wimmer, E., et al., 2006. Pyrite-induced hydroxyl radical formation and its effect on nucleic acids. Geochem. Trans. 7 (1), 1–11.
Crelling, J.C., 2011. Current uses of fluorescence microscopy in coal petrology. J. Microsc. 132 (3), 251–266.
Cui, D., Tian, F., Ozkan, C.S., Wang, M., Gao, H., 2005. Effect of single wall carbon nanotubes on human hek293 cells. Toxicol. Lett. 155 (1), 73–85.
Dalmora, A.C., Ramos, C.G., Oliveira, M.L.S., et al., 2016. Chemical characterization, nano-particle mineralogy and particle size distribution of basalt dust wastes. Sci. Total Environ. 539, 560–565.
Dias, C.L., Oliveira, M., Hower, J.C., 2014. Nanominerals and ultrafine particles from coal fires from Santa Catarina, south Brazil. Int. J. Coal Geol. 122 (1), 50–60.
Donaldson, K., 1998. Ultrafine (nanometer) particles mediated lung injury. J Aersole Sci 29 (5–6), 553–560.
Doney, Brent C., Blackley, David, Hale, Janet M., et al., 2019. Respirable coal mine dust in underground mines, United States, 1982-2017. Am. J. Ind. Med. 62 (6), 478–485.
Duffin, R., Tran, C.L., Clouter, A., et al., 2002. The importance of surface area and specific reactivity in the acute pulmonary inflammatory response to particles. Ann. Occup. Hyg. 46 (Suppl. 1), 242–245.
Dunnick, J.K., Elwell, M.R., Radovsky, A.E., et al., 1995. Comparative carcinogenic effects of nickel subsulfide, nickel oxide, or nickel sulfate hexahydrate chronic exposures in the lung. Canc. Res. 55 (22), 5251–5256.
Dwivedi, S., Saquib, Q., Al-Khedhairy, A.A., Ali, A.Y.S., Musarrat, J., 2012. Characterization of coal fly ash nanoparticles and induced oxidative DNA damage in human peripheral blood mononuclear cells. Sci. Total Environ. 437 (6), 331–338.
Érico, M.M.F., Juliano, S.B., Márcia, F.M., Günter, K., 2007. Sample preparation techniques based on combustion reactions in closed vessels-A brief overview and recent applications. Spectrochim. Acta, Part B 62, 1051–1064.
Everard, M.L., Devadason, S.G., Le, S., 1997. Flow early in the inspiratory manoeuvre affects the aerosol particle size distribution from a turbuhaler. Respir. Med. 91 (10), 624–628.
Ferin, J., Oberdörster, G., Soderholm, S.C., Gelein, R., 1991. Pulmonary tissue access of ultrafine particles. J. Aerosol Sci. 4 (1), 57–68.
Finkelman, R.B., Orem, W., Castranova, V., et al., 2002. Health impacts of coal and coal use: possible solutions. Int. J. Coal Geol. 50, 425–443.
Finkelman, Robert B., Palmer, Curtis A., Wang, Peipei, 2018. Quantification of the modes of occurrence of 42 elements in coal. Int. J. Coal Geol. 185, 138–160.
Finkelman, Robert B., Dai, Shifeng, French, David, 2019. The importance of minerals in coal as the hosts of chemical elements: a review. Int. J. Coal Geol. 212, 103251.
Fisher, G.L., Prentice, B.A., Silberman, D., et al., 1978. Physical and morphological studies of size-classified coal fly ash. Environ.sci. & Tech 12 (4), 447–451.
Flores, R.M., 2013. Coal and Coalbed Gas: Fueling the Future. Elsevier Science & Technology Books.
Fubini, B., Fenoglio, I., 2007. Toxic potential of mineral dusts. Mineral. Soc. Am. 3, 407–414.
GBD 2013 Mortality and Causes of Death Collaborators, 2015. Global, regional, and national age-sex specific all-cause and cause specific mortality for 240 causes of death, 1990-2013: a systematic analysis for the Global Burden of Disease Study 2013. Lancet 385, 117–171.
Giannadaki, D., Pozzer, A., Lelieveld, J., 2014. Modeled global effects of airborne desert dust on air quality and premature mortality. Atmos. Chem. Phys. 14 (2), 24023–24050.
Glass, D., Cohen, R., Roberts, M., et al., 2018. 1497 Review of the Respiratory Component of the Queensland Coal Mine Workers' Health Scheme, vol. 75. Occupational and Environmental Medicine, p. A438.
Graber, J.M., Harris, G., Almberg, K.S., Rose, C.S., Cohen, R.A., 2017. Increasing severity of pneumoconiosis among younger former us coal miners working exclusively under modern dust-control regulations. J. Occup. Environ. Med. 59 (6), e105–e111.
Gregory, J.C., 1831. Case of particular black infiltration of the whole lungs resembling melanosis. Edinburgh. Med. Surg. 36, 389–394.
Greim, H., Borm, P., Schins, R., et al., 2001. Toxicity of fibers and particles. report of the workshop held in Munich, Germany. Inhal. Toxicol. 13 (9), 737–754, 26-27 October 2000.

Grigore, M., Sakurovs, R., French, D., Sahajwalla, V., 2008. Mineral matter in coals and their reactions during coking. Int. J. Coal Geol. 76 (4), 301–308.
Halterman, A., Sousan, S., Peters, T.M., 2017. Comparison of respirable mass concentrations measured by a personal dust monitor and a personal dataram to gravimetric measurements. Ann Work Expo Health 62 (1).
Han, L., Gao, Q., Yang, J., et al., 2017a. Survival analysis of coal workers' pneumoconiosis (CWP) patients in a state-owned mine in the east of China from 1963 to 2014. Int. J. Environ. Res. Publ. Health 14 (5), 489.
Han, L., Gao, Q., Yang, J., 2017b. Survival analysis of coal workers' pneumoconiosis (CWP) patients in a state-owned mine in the east of China from 1963 to 2014. Int. J. Environ. Res. Publ. Health 14 (5), 489–499.
Harrison, P., 2015. Commissioner for Mine Safety and Health, Queensland Mines Inspectorate Annual Performance Report 2014–2015, pp. 1–34.
Harrison, R.M., Shi, J.P., Khan, A., et al., 2000. Measurement of number, mass and size distribution of particles in the atmosphere. Philosophical Transactions Mathematical Physical & Engineering Sciences 358 (1775), 2567–2580.
Hochella, M.F., Lower, S.K., Maurice, P.A., et al., 2008. Nanominerals, mineral nanoparticles, and earth systems. Science 319 (5870), 1631–1635.
Honma, K., 2004. Proposed criteria for mixed-dust pneumoconiosis: definition, descriptions, and guidelines for pathologic diagnosis and clinical correlation. Hum. Pathol. 35, 1515–1523.
Hsin, Y.H., Chen, C.F., Huang, S., et al., 2008. The apoptotic effect of nanosilver is mediated by a ros- and jnk-dependent mechanism involving the mitochondrial pathway in nih3t3 cells. Toxicol. Lett. 179 (3), 130–139.
Hurley, J.F., Maclaren, W.M., 1987. Dust Related Risks of Radiological Changes in Coalminers over a 40-year Working Life: Report on Work Commissioned by NIOSH. Institute of Occupational Medicine, Edinburgh, Scotland. TM/79/09.
Ibarra, J., Muñoz, E., Moliner, R., 1996. FTIR study of the evolution of coal structure during the coalification process. Org. Geochem. 24 (6), 725–735.
Ingram, D.J.E., Tapley, J.G., Jackson, R., et al., 1954. Paramagnetic resonance in carbonaceous solids. Nature 174 (4434), 797–798.
International Energy Agency (EIA), 2017. Coal Information: Overview.
Jacobsen, M., Rae, S., Walton, W.H., Rogan, J.M., 1971. The relation between pneumoconiosis and dust exposure in British coal mines. In: Walton, W.H. (Ed.), Inhaled Particles III. Old Woking. Unwin Brothers, England, pp. 903–919.
Jean-Pierre, Jolivet, Cédric, Froidefond, Agnès, Pottier, et al., 2004. Size tailoring of oxide nanoparticles by precipitation in aqueous medium. a semi-quantitative modelling. J. Mater. Chem. 14 (21), 3281–3288.
Johann-Essex, V., Keles, C., Sarver, E., 2017a. A computer-controlled sem-edx routine for characterizing respirable coal mine dust. Minerals 7 (1), 15.
Johann-Essex, V., Keles, C., Rezaee, M., et al., 2017b. Respirable coal mine dust characteristics in samples collected in central and northern Appalachia. Int. J. Coal Geol. 182, 85–93.
Jun, J.S., Jung, J.I., Kim, H.R., et al., 2013. Complications of pneumoconiosis: radiologic overview. Eur. J. Radiol. 82 (10), 1819–1830.
Kampa, M., Castanas, E., 2008. Human health effects of air pollution. Environ. Pollut. 151 (2), 362–367.
Kane, A.B., 1996. Mechanisms of mineral fibre carcinogenesis. IARC (Int. Agency Res. Cancer) Sci. Publ. 140 (140), 11–34.
Kawanishi, S., Hiraku, Y., Murata, M., Oikawa, S., 2002. The role of metals in site-specific DNA damage with reference to carcinogenesis. Free Radic. Biol. Med. 23 (9), 822–832.
Ken, Donaldson, Robert, Aitken, Lang, Tran, 2006. Carbon nanotubes: a review of their properties in relation to pulmonary toxicology and workplace safety. Toxicol. Sci. 92 (1), 5–22.
Ketris, M.P., Yudovich, Y.E., 2009. Estimations of clarkes for carbonaceous biolithes: world averages for trace element contents in black shales and coals. Int. J. Coal Geol. 78 (2), 135–148.
Kinloch, I., 2008. Carbon nanotubes introduced into the abdominal cavity of mice show asbestos-like pathogenicity in a pilot study. Nat. Nanotechnol. 3 (7), 423–428.
Lam, C.W., James, J.T., Mccluskey, R., et al., 2004. Pulmonary toxicity of single-wall carbon nanotubes in mice 7 and 90 days after intratracheal instillation. Toxicol. Sci. 77 (1), 126–134.
Laney, A.S., Weissman, D.N., 2014. Respiratory diseases caused by coal mine dust. J. Occup. Environ. Med. 56 (Suppl. 10), S18.
Lasfargues, G., Lardot, C., Delos, M., 1995. The delayed lung responses to single and repeated intratracheal administration of pure cobalt and hard metal powder in the rat. Environ. Res. 69 (2), 108–121.
Lenaz, G., 2010. The mitochondrial production of reactive oxygen species: mechanisms and implications in human pathology. IUBMB Life 52 (3-5), 159–164.
Li, N., Constantinos, S., Arthur, C., et al., 2003. Ultrafine particulate pollutants induce oxidative stress and mitochondrial damage. Environ. Health Perspect. 111 (4), 455–460.
Lighty, J.S., Veranth, J.M., Sarofim, A.F., 2000. Combustion aerosols: factors governing their size and composition and implications to human health. Air Repair 50 (9), 1565–1618.
Liu, Y., Zhao, Y., Sun, B., et al., 2013. Understanding the toxicity of carbon nanotubes. Acc. Chem. Res. 46 (3), 702–713.
Liu, J., Jiang, X., Shen, J., Hai, Z., 2014a. Chemical properties of superfine pulverized coal particles. part 1. electron paramagnetic resonance analysis of free radical characteristics. Adv. Powder Technol. 25 (3), 916–925.
Liu, J., Jiang, X., Han, X., 2014b. Chemical properties of superfine pulverized coals. part 2. demineralization effects on free radical characteristics. Fuel 115 (12), 685–696.
Liu, J., Jiang, X., Shen, J., Hai, Z., 2015. Influences of particle size, ultraviolet irradiation and pyrolysis temperature on stable free radicals in coal. Powder Technol. 272, 64–74.
Low, F., Zhang, L., 2012. Microwave digestion for the quantification of inorganic elements in coal and coal ash using ICP-OES. Talanta 101, 346–352.
Madden, A.S., Jr, M.F.H., 2005. A test of geochemical reactivity as a function of mineral size: manganese oxidation promoted by hematite nanoparticles. Geochem. Cosmochim. Acta 69 (2), 389–398.
Magrez, A., Kasas, S., Salicio, V., et al., 2006. Cellular toxicity of carbon-based nanomaterials. Nano Lett. 6 (6), 1121–1125.
Manke, A., Wang, L., Rojanasakul, Y., 2013. Mechanisms of nanoparticle-induced oxidative stress and toxicity. BioMed Res. Int. 1, 1–15.
Marsalek, R., Sassikova, M., 2016. Characterization of the size distribution of subbituminous coal by laser diffraction. Instrum. Sci. Technol. 44 (3), 233–240.
Martinello, K., Oliveira, M.L., Molossi, F.A., et al., 2014. Direct identification of hazardous elements in ultra-fine and nanominerals from coal fly ash produced during diesel co-firing. Sci. Total Environ. 470–471 (2), 444–452.
Maynard, A.D., Kuempel, E.D., 2005a. Airborne nanostructured particles and occupational health. J. Nanoparticle Res. 7 (6), 587–614.
Maynard, A.D., Kuempel, E.D., 2005b. Airborne nanostructured particles and occupational health. J. Nanoparticle Res. 7 (6), 587–614.
McGuire, Coreen, 2019. Dust to dust. The Lancet Respiratory Medicine 7 (5), 383–384.
Miao, Yulong, Yao, Nan, Li, Xiaonian, 2015. Overview of coal functional groups characterization methods. Zhejiang Chemical Industry 46, 43–48.
Mischler, S.E., Cauda, E.G., Di, G.M., et al., 2016. Differential activation of raw 264.7 macrophages by size-segregated crystalline silica. J. Occup. Med. Toxicol. 11 (1), 57.
Mketo, N., Nomngongo, P.N., Ngila, J.C., 2016. An overview on analytical methods for quantitative determination of multi-element in coal samples. Trends Anal. Chem. 85, 107–116.
Mls, O., Navarro, O.G., Crissien, T.J., et al., 2017a. Coal emissions adverse human health effects associated with ultrafine/nano-particles role and resultant engineering controls. Environ. Res. 158, 450–455.
Mls, O., Navarro, O.G., Crissien, T.J., et al., 2017b. Coal emissions adverse human health effects associated with ultrafine/nano-particles role and resultant engineering controls. Environ. Res. 158, 450–455.
Mo, J., Wang, L., Au, W., et al., 2014. Prevalence of coal workers' pneumoconiosis in China: a systematic analysis of 2001–2011 studies. Int. J. Hyg Environ. Health 217 (1), 46–51.
Monteiroriviere, N.A., Nemanich, R.J., Inman, A.O., 2005. Multi-walled carbon nanotube interactions with human epidermal keratinocytes. Toxicol. Lett. 155 (3), 377–384.
Moreno, Teresa, Pedro, Trechera, Xavier, Querol, et al., 2019. Trace element fractionation between PM10 and PM2.5 in coal mine dust: implications for occupational respiratory health. Int. J. Coal Geol. 203, 52–59.
MSHA, 2016. Respirable Coal Mine Dust Rule Enters Final Phase of Implementation.
Napierska, D., 2010. The nanosilica hazard: another variable entity. Part. Fibre Toxicol 7, 39.
National Academies of Sciences, Engineering, and Medicine, 2018. Monitoring and Sampling Approaches to Assess Underground Coal Mine Dust Exposures. The National Academies Press, Washington, DC. https://doi.org/10.17226/25111.
National Institute for Occupational Safety and Health (NIOSH), 1995. Criteria for a Recommended Standard: Occupational Exposure to Coal Mine Dust. DHHS (NIOSH) Publication No. 95–106. National Institute for Occupational Safety and Health, Washington, DC.
Nemmar, A., Hoet, P.H., Vanquickenborne, B., et al., 2002. Passage of inhaled particles into the blood circulation in humans. Circulation 105 (4), 411–414.
Nie, B., Liu, X., Yang, L., et al., 2015. Pore structure characterization of different rank coals using gas adsorption and scanning electron microscopy. Fuel 158, 908–917.
Oberdörster, G., Ferin, J., Gelein, R., et al., 1992. Role of the alveolar macrophage in lung injury: studies with ultrafine particles. Environ. Health Perspect. 97 (3), 193–199.
Oberdörster, G., Oberdörster, E., Oberdörster, J., 2005. Nanotoxicology: an emerging discipline evolving from studies of ultrafine particles. Environ. Health Perspect. 113 (7), 823–839.
Oberdörster, G., Ferin, J., Soderholm, S., et al., 2016. Increased pulmonary toxicity of inhaled ultrafine particles: due to lung overload alone? Ann. Occup. Hyg. 38, 295–302.
Page, S.J., Volkwein, J.C., Vinson, R.P., et al., 2008. Equivalency of a personal dust monitor to the current United States coal mine respirable dust sampler. J. Environ. Monit. 10, 96–101.
Park, E., Choi, J., Park, Y., 2008. Oxidative stress induced by cerium oxide nanoparticles in cultured BEAS-2B cells. Toxicology 245 (1-2), 90–100.
Park, E., Yoon, J., Choi, K., et al., 2009. Induction of chronic inflammation in mice treated with titanium dioxide nanoparticles by intratracheal instillation. Toxicology 260 (1-3), 37–46.
Pascal, M., Falq, G., Wagner, V., 2014. Short-term impacts of particulate matter (PM_{10}, $PM_{10\text{-}2.5}$, $PM_{2.5}$) on mortality in nine French cities. Atmos. Environ. 95, 175–184.
Perret, J.L., Plush, B., Lachapelle, P., 2017. Coal mine dust lung disease in the modern era. Respirology 22 (4), 662–670.
Petsonk, E.L., Rose, C., Cohen, R., 2013. Coal mine dust lung disease. New lessons from an old exposure. Am. J. Respir. Crit. Care Med. 187 (11), 1178–1185.
Potera, Carol, 2019. Black lung disease resurges in Appalachian coal miners. Am. J. Nurs. 119 (4), 14.
Qin, L., Zhai, C., Liu, S., et al., 2017. Changes in the petrophysical properties of coal subjected to liquid nitrogen freeze-thaw-a nuclear magnetic resonance investigation. Fuel 194, 102–114.
Reed, W.R., Joy, G.J., Shahan, M., et al., 2019. Laboratory results of a 3rd generation roof bolter canopy air curtain for respirable coal mine dust control. International Journal of Coal Science & Technology 6 (1), 15–26.
Ross, M.H., Murray, J., 2004. Occupational respiratory disease in mining. Occup. Med. 54 (5), 304–310.

Sahai, N., Lee, Y.J., Xu, H., et al., 2007. Role of Fe(ii) and phosphate in arsenic uptake by coprecipitation. Geochem. Cosmochim. Acta 71 (13), 3193–3210.
Sambandam, B., Palanisami, E., Abbugounder, R., Prakhya, B., Thiyagarajan, D., 2014. Characterizations of coal fly ash nanoparticles and induced in vitro toxicity in cell lines. J. Nanoparticle Res. 16 (2), 2217.
Sato, Y., Yokoyama, A., Shibata, K., et al., 2005. Influence of length on cytotoxicity of multi-walled carbon nanotubes against human acute monocytic leukemia cell line thp-1 in vitro and subcutaneous tissue of rats in vivo. Mol. Biosyst. 1 (2), 176–182.
Schins, R.P., 2002. Mechanisms of genotoxicity of particles and fibers. Inhal. Toxicol. 14 (1), 57–78.
Schins, R.P., Knaapen, A.M., Cakmak, G.D., et al., 2002. Oxidant-induced DNA damage by quartz in alveolar epithelial cells. Mutat. Res. 517 (1), 77–86.
Seaman, D.M., Meyer, C.A., Kanne, J.P., 2015. Occupational and environmental lung disease. Clin. Chest Med. 36 (2), 249–268.
Sellaro, R., Sarver, E., Baxter, D., 2015. A standard characterization methodology for respirable coal mine dust using sem-edx. Resources 4 (4), 939–957.
Shahan, Michael R., Reed, William Randolph, 2019. The design of a laboratory apparatus to simulate the dust generated by longwall shield advances. International Journal of Coal Science & Technology 6, 577–585.
Shi, H., Hudson, L.G., Liu, K.J., 2004. Oxidative stress and apoptosis in metal ion-induced carcinogenesis. Free Radic. Biol. Med. 5 (37), 582–593.
Silva, L.F.O., Boit, K.M.D., 2011. Nanominerals and nanoparticles in feed coal and bottom ash: implications for human health effects. Environ. Monit. Assess. 174 (1-4), 187–197.
Singh, P., Ramakrishnan, P., 1996. Powder characterization by particle shape assessment. Kona 14, 16–30.
Song, Y., Wang, X., Maher, B.A., et al., 2016. The spatial-temporal characteristics and health impacts of ambient fine particulate matter in China. J. Clean. Prod. 112, 1312–1318.
Song, C., He, J., Wu, L., et al., 2017. Health burden attributable to ambient pm2.5 in China. Environ. Pollut. 223, 575–586.
Stone, V., Brown, D.M., Watt, N., et al., 2000. Ultrafine particle-mediated activation of macrophages: intracellular calcium signaling and oxidative stress. Inhal. Toxicol. 12 (Suppl. 3), 345–351.
Sui, Z., Zhang, Y., Yao, J., et al., 2016. The influence of Nacl and Na_2Co_3, on fine particulate emission and size distribution during coal combustion. Fuel 184, 718–724.
Sun, Y., Wu, M., Zheng, L., et al., 2018. Uranium speciation in coal bottom ash investigated via, x-ray absorption fine structure and x-ray photoelectron spectra. J. Environ. Sci. 74, 88–94.
Swaine, D.J., 1994. Trace elements in coal and their dispersal during combustion. Fuel Process. Technol. 39, 121–131.
Tailakov, O.V., Makeev, M.P., Kormin, A.N., et al., 2015. Development of method of rapid analysis of particle size distribution of the coal charge based on digital image processing. Appl. Mech. Mater. 770, 512–517.
Takenaka, S., Karg, E., Roth, C., et al., 2001. Pulmonary and systemic distribution of inhaled ultrafine silver particles in rats. Environ. Health Perspect. 109 (Suppl. 4), 547–551.
Tang, R., Wang, L., Nancollas, G.H., 2004. Size-effects in the dissolution of hydroxyapatite: an understanding of biological demineralization. J. Mater. Chem. 14 (14), 2341–2346.
Taylor, J.C., 1991. Computer programs for standardless quantitative analysis of minerals using the full powder diffraction profile. Powder Diffr. 6 (1), 2–9.
Thannickal, V.J., Fanburg, B.L., 2000. Reactive oxygen species in cell signaling. Am. J. Physiol. 279 (6), L1005–L1028.
Uebersfeld, J., Tienne É, A., Combrisson, J., 1954. Paramagnetic resonance, a new property of coal-like materials. Nature 174 (4430), 614-614.
Ulusoy, U., Yekeler, M., Hiçyılmaz, C., 2003. Determination of the shape, morphological and wettability properties of quartz and their correlations. Miner. Eng. 16 (10), 951–964.
U.S. Energy Information Administration, 2018. Short-Term Energy Outlook (STEO).
Vallyathan, V., Brower, P.S., Green, F.H., Attfield, M.D., 1996. Radiographic and pathologic correlation of coal workers' pneumoconiosis. Am. J. Respir. Crit. Care Med. 154 (1), 741–748.
Vincent, Castranova, 1998. Particulates and the airways: basic biological mechanisms of pulmonary pathogenicity. Appl. Occup. Environ. Hyg 13 (8), 613–616.
Wagner, N.J., Hlatshwayo, B., 2005. The occurrence of potentially hazardous trace elements in five Highveld coals, South Africa. Int. J. Coal Geol. 63, 228–246.
Wang, X.T., Zhang, G., 2016. Prevalence of pneumoconiosis in a coal enterprise from 2002 to 2013. Chin. J. Ind. Hyg. Occup. Dis. 34, 681–682 (In Chinese).
Wang, S., Tang, Y., Schobert, H.H., et al., 2011. FTIR and 13c NMR investigation of coal component of late permian coals from southern China. Energy Fuels 25 (12), 5672–5677.
Wang, Q., Wang, J., He, M.Z., et al., 2017. A county-level estimate of pm2.5 related chronic mortality risk in China based on multi-model exposure data. Environ. Int. 110, 105–112.
Wang, C., Yang, X., Li, Q., et al., 2018. Free radicals, apparent activation energy, and functional groups during low-temperature oxidation of Jurassic coal in northern Shaanxi. International Journal of Mining Science and Technology 28 (3), 469–475.
Ward, C.R., 2002. Analysis and significance of mineral matter in coal seams. Int. J. Coal Geol. 50 (1), 135–168.
Ward, C.R., 2016. Analysis, origin and significance of mineral matter in coal: an updated review. Int. J. Coal Geol. 165, 1–27.
Watkins-Pitchford, W., Moir, J., 1916. On the nature of the doubly refracting particles seen in microscopic sections of silicotic lungs. Rep. S. Afr. Inst. Med. Res. 1, 000.
Webb, D.R., Wilson, S.E., Carter, D.E., 1986. Comparative pulmonary toxicity of gallium arsenide, gallium(iii) oxide, or arsenic(iii) oxide intratracheally instilled into rats. Toxicol. Appl. Pharmacol. 82 (3), 405–416.
Weichenthal, S., Villeneuve, P.J., Burnett, R.T., et al., 2014. Long-term exposure to fine particulate matter: association with nonaccidental and cardiovascular mortality in the agricultural health study cohort. Environ. Health Perspect. 122 (6), 609–615.
WHO, 1997. Determination of Airborne Fibre Number Concentrations: a Recommended Method, by Phase-Contrast Optical Microscopy (Membrane Filter Method). World Health Organization, Geneva.
WHO, 2003. Health Aspects of Air Pollution with Particulate Matter, Ozone and Nitrogen Dioxide. Report 2003 on a WHO Working Group, Bonn.
Wise, S.S., Holmes, A.L., Sr, W.J., 2008. Hexavalent chromium-induced DNA damage and repair mechanisms. Rev. Environ. Health 23 (1), 39–58.
Wiseman, H., Halliwell, B., 1996. Damage to DNA by reactive oxygen and nitrogen species: role in inflammatory disease and progression to cancer. Biochem. J. 313, 17–29.
Xia, W., 2017. Role of particle shape in the floatability of mineral particle: an overview of recent advances. Powder Technol. 317, 104–116.
Xia, T., Kovochich, Michael, Brant, Jonathan, et al., 2006. Comparison of the abilities of ambient and manufactured nanoparticles to induce cellular toxicity according to an oxidative stress paradigm. Nano Lett. 6 (8), 1794–1807.
Xin, H.H., Wang, D.M., Qi, X.Y., et al., 2014. Structural characteristics of coal functional groups using quantum chemistry for quantification of infrared spectra. Fuel Process. Technol. 118 (2), 287–295.
Xing, Y.F., Xu, Y.H., Shi, M.H., et al., 2016. The impact of $PM_{2.5}$ on the human respiratory system. J. Thorac. Dis. 8 (1), E69–E74.
Xu, C., Wang, D., Wang, H., et al., 2017. Effects of chemical properties of coal dust on its wettability. Powder Technol. 318, 33–39.
Xu, Guang, Chen, Yinping, Jacques, Eksteen, Xu, Jialin, 2018. Surfactant-aided coal dust suppression: a review of evaluation methods and influencing factors. Sci. Total Environ. 639, 1060–1076.
Xue, X., 2016. Prediction for the burnout of pulverized coal in a coal-fired power plant by combining proximate analysis, coal petrography, and pulverized-coal size distribution. Energy Sources 38 (1), 69–74.
Yang, N., Tang, S.H., Zhang, S.H., et al., 2018. In seam variation of element-oxides and trace elements in coal from the eastern Ordos Basin, China. Int. J. Coal Geol. 197, 31–41.
Yao, Haifei, Wang, Haiyan, Li, Yanchuan, et al., 2020. Three-dimensional spatial and temporal distributions of dust in roadway tunneling. International Journal of Coal Science & Technology 7 (1), 88–96.
Yates, D.H., Gibson, P.G., Hoy, R., et al., 2016. Down under in the coal mines. Am. J. Respir. Crit. Care Med. 194 (6), 772–773.
Yekeler, M., Ulusoy, U., Hiçyılmaz, C., 2004. Effect of particle shape and roughness of talc mineral ground by different mills on the wettability and floatability. Powder Technol. 140 (1), 68–78.
Yu, D.X., Xu, M.H., Yao, H., et al., 2009. Physicochemical properties and potential health effects of nanoparticles from pulverized coal combustion. Chin. Sci. Bull. 54 (7), 1243–1250.
Zhang, M., 2010. Silicosis in automobile foundry workers: a 29-year cohort study. Biomed. Environ. Sci. 23, 121–129.
Zhang, Q., Kusaka, Y., Sato, K., et al., 1998. Differences in the extent of inflammation caused by intratracheal exposure to three ultrafine metals: role of free radicals. J. Toxicol. Environ. Health 53 (6), 423–438.
Zhang, Q., Kusaka, Y., Donaldson, K., 2000a. Comparative pulmonary responses caused by exposure to standard cobalt and ultrafine cobalt. J. Occup. Health 42 (4), 179–184.
Zhang, Q., Kusaka, Y., Donaldson, K., 2000b. Comparative injurious and proinflammatory effects of three ultrafine metals in macrophages from young and old rats. Inhal. Toxicol. 12 (Suppl. 3), 267–273.
Zhang, Q., Kusaka, Y., Zhu, X., et al., 2003. Comparative toxicity of standard nickel and ultrafine nickel in lung after intratracheal instillation. J. Occup. Health 45, 23–30.
Zhang, H., Rustad, J.R., Banfield, J.F., 2007. Interaction between water molecules and zinc sulfide nanoparticles studied by temperature-programmed desorption and molecular dynamics simulations. J. Phys. Chem. 111 (23), 5008.
Zosky, G.R., Hoy, R.F., Silverstone, E.J., et al., 2016. Coal workers' pneumoconiosis: an Australian perspective. Med. J. Aust. 204 (11), 414–418.