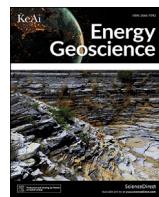




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Coal as an energy source and its impacts on human health

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ABSTRACT

Attempts to secure more energy, food, and infrastructure leave a trail of environmental contamination and human health hazards. Coal is a fossil fuel and nonrenewable energy source that is combusted and used to generate electricity. A coal-fired power plant is a prodigious generator of environmental pollution, releasing large quantities of particles as aerosols in the atmosphere. The inhalation of hazardous substances such as coal micro-particles, nanoparticles, and its by-products constitutes an invisible risk to human health. Although coal is predominantly composed of carbon, there are many other constituents including sulfur, nitrogen, organometallic compounds, and minerals, that contribute to the formation of extremely toxic secondary compounds that come in contact with the atmosphere. The continuous inhalation of these hazardous substances triggers many diseases such respiratory and cardiovascular disease, systemic inflammation, and neurodegeneration. Due to coal heterogeneity, it is extremely complex to establish all the effects of the molecules in the organism. Each cell can undergo different modifications depending on the stressing molecule. On that account, inhaling air contaminated with these particles can be highly dangerous and unpredictable. This review covers the impact of coal inhalation on the lungs, immune system, heart, reproductive system, brain, DNA, and, in general, the human health. For this review, Medline and Scopus databases were accessed, including human epidemiological, review studies, and coal characterization studies over the years. Coal as an energy source must be utilized with appropriate measures of environmental protection and to safeguard human health.

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1. Introduction

Coal is composed mostly of carbon and hydrocarbons, which have high energy density that is released through combustion (burning). In the course of the First Industrial Revolution, coal-burning was transformed into movement. From an economic point of view, this energy source was revolutionary. However, from an environmental standpoint, ambient air pollution is a threat

(Lozano et al., 2018; Oliveira et al., 2018b; Oberschelp et al., 2019).

The comparison between different forms of commercial power generation shows that the health burdens are higher in coal-, lignite-, and oil-fired power plants than most pollute outdoor air (Markandy and Wilkinson, 2007).

Coal is extensively used for energy generation in many countries (Zou et al., 2016). Coal, the second most important energy source worldwide, contributes to 40% of global primary energy consumption (Smil et al., 2016; World Energy Research World Energy Council, 2016). Many developing countries use coal as an energy source. In December 2015 in Paris, all nations agreed to invest and intensify combat against global warming for a sustainable, low-carbon future. The Paris Agreement intends to reduce greenhouse gas production (Paris Agreement, 2015). The goals are unlikely to be achieved, as most developing countries are more concerned with immediacy than with the future.

Coal combustion releases a mixture of gaseous components and a complex mixture of particles with distinct size, morphology, chemical, physical, and biological characteristics into the

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atmosphere. Power plants expose surrounding communities to pollutants that may cause negative impacts on health (Hendryx et al., 2020a). It is not just coal-burning that is a health issue. Significant amounts of coal dust are released during extraction, transport, and handling process, which reach workers and surrounding neighborhoods and cause xenobiotic effects (Espitia-Pérez et al., 2018; Oliveira et al., 2018a; Rovira et al., 2019).

Ninety-seven percent of cities with a population higher than 100,000 inhabitants in low- and middle-income countries do not meet WHO air quality guidelines (WHO, 2018). This is a complex problem and although coal combustion is one of the contaminants, it is important to note that the transportation of coal, point-source home heating and cooking sources, vehicle fuel burning, also aggravate environmental contamination. Good quality air is vital for the correct functioning of an organism. Chronic exposure to pollutants in the air is associated with several diseases that can lead to death. Attempts to reduce coal as an energy source are necessary to prevent severe climate change and to improve public health (Hendryx et al., 2020b).

From another perspective, coal is a rising source of critical elements (Dai and Finkelman, 2018). Coal contains rare earth elements and carbon nanoparticles that can be useful to high-technology (Nano- and Microtechnology) (Dai et al., 2016; Silva et al., 2020a, 2020b). Therefore, coal mining methods that do not harm the environment need to be employed.

1.1. Coal composition

Each coal is unique (Orem and Finkelman, 2003). Coal has been formed during the last 475 million years, with significant coal formation from the Carboniferous (Pennsylvanian) period 300 million years ago (Orem and Finkelman, 2003; Hower et al., 2019). Coal is a complex interplay between biological, geochemical, mineralogical, palynological, and petrological properties (Dai et al., 2020a).

Over the years biopolymers of vascular and nonvascular plants, algae, microbiome, water, oxygen, resins, mosses, remains of prehistoric organisms and other organic and inorganic materials were piled up in layers slowly, retaining most of its carbon (peat deposit) (Hatcher and Clifford, 1997; Orem and Finkelman, 2003; Dai et al., 2020a, 2020b). Coal is formed from peat deposition during a complex metamorphism called coalification (Hower and Gayer, 2002; Orem and Finkelman, 2003; O'Keefe et al., 2013; Hower et al., 2019; Dai et al., 2020). The heterogeneous profile of coal is influenced by ecological depositional progressions, formed during syngenetic stages and subsequently altered during diagenesis and epigenesis (Silva et al., 2010; Oliveira et al., 2018a; Dai et al., 2020a). The peat composition is intrinsically related to elevated coal toxic components to human health, like sulfur, and, in some cases, As and Hg (Dai et al., 2012, 2020a).

The terminology in this work, "coal ashes" are for combustion products, and "coal dust" is for coal products before combustion. The major pollutants released by coal combustion are particulates, sulfur and nitrogen oxides, trace elements (As, Cs, F, Se, Hg, and the radionuclides U and Th), and organic compounds generated by incomplete coal combustion (Finkelman, 1999; Orem and Finkelman, 2003). The coal combustion also releases a large diversity of ultrafine, nanoparticles, and their aggregates (Silva et al., 2010; Silva and da Boit, 2011a; Dai et al., 2020b).

Coal-fired power plant processes induce chemical transformation in elements (Flores, 2014). This process releases significant amounts of gases such as carbon monoxide (CO), carbon dioxide (CO₂), sulfur dioxide (SO₂), sulfur trioxide (SO₃), nitric oxide (NO), and nitrogen dioxide (NO₂) (Orem and Finkelman, 2003; Munawer, 2018). The mixture of pollutants may trigger severe health impacts due to their capacity to generate varied chemical

reactions (Finkelman et al., 2002a, 2002b; Munawer, 2018).

The complex mixture of small particles found in coal combustion products is dominated by particulate matter (PM). The PM is classified according to particle size (aerodynamic equivalent diameter), for example, PM₁₀ and PM_{2.5}. PM_{2.5} can infiltrate in different respiratory system regions, triggering respiratory diseases (Anderson et al., 2012). The smaller the PM (nm) size, the deeper it can penetrate the lungs and possibly even pass to the systemic circulation, affecting other organs (Gomes and Florida-James, 2014) while the larger particles (PM₁₀) will be filtered by the nose or will be deposited in the more proximal conducting airways (Glencross et al., 2020). The airborne PM generated by coal combustion suspended with the gases may travel kilometers away, crossing continents (Verstraeten et al., 2015).

1.2. Coal composition and health

The composition of coal depends on the characteristics of the natural coal genesis site, which varies significantly over a few kilometers (Pires and Querol, 2004; Silva et al., 2009; Dai et al., 2020a; O'Keefe et al., 2013).

China and the United States are the main coal mining regions worldwide. However, Indonesia, Australia, Russia, South Africa, and Colombia also produce large amounts of coal (Oberschelp et al., 2019). In many countries burning coal is used to generate electricity, as in USA 23.5%, Germany 46%, India 69%, Australia 77%, China 79%, Poland 92%, South Africa 93% (Tarfafdar and Sinha, 2018; U.S. Energy Information Administration, 2020). In South America, Brazil is the largest consumer of coal (Oliveira et al., 2012, 2013; Cutruneo et al., 2014).

Impacts on health vary according to the composition of the coal. (Rauner et al., 2020). Each global localization has distinct coal composition according to peat depositional environments (Dai et al., 2020a). For example, Indian and Chinese coal-fired power plants release high Hg content to the atmosphere, (Oliveira et al., 2014; Liu et al., 2018); in addition, the negligence of SO₂ and NO_x control makes India one of the main responsible for global coal combustion health impacts (Oberschelp et al., 2019). High levels of Pb were measured around coal-fired power plants in South Africa (Okedeyi et al., 2014).

Burning Brazilian coal releases high levels of As and Zn (Flues et al., 2013; Silva et al., 2010), and the waste derived from mining contributes to environmental contamination when it is not correctly disposed (Silva et al., 2011b, 2020c; Oliveira et al., 2013, 2018b; Cutruneo et al., 2014).

People living near or far from power plants may have health problems. The relationship between increasing human morbidity and mortality due to air pollution from all sources is evident (Saikia et al., 2017).

The pathogenesis and pathophysiology triggered by different components of coal-burning will be described throughout the article.

2. Methods

This review will cover the impact of coal inhalation on the human organism and how those particles affect the physiology of organs vital to the organism's proper functioning. Medline and Scopus databases were accessed. The database searches only included human epidemiological, review, and coal characterization studies published over the years.

3. Discussion

3.1. Lungs

The lungs are the first target of air pollutants (Marino et al., 2015). Inhalation of polluted air is the ninth leading risk factor for cardiopulmonary mortality (Kurt et al., 2016). According to the International Agency for Research on Cancer (IARC), air pollution is classified as a Group 1 carcinogen in humans (Shahadin et al., 2018). Gaseous components and particulate matter present in air trigger pulmonary disease — inducing at the first moment a pro-inflammatory condition (Glencross et al., 2020).

Chronic inhalation of coal dust affects the lungs directly. Studies carried out with coal miners show devastating data on lung health. Silicosis, mixed dust pneumoconiosis, chronic obstructive airway disease, including emphysema and chronic bronchitis, are a spectrum of diseases that, due to the high incidence of these diseases triggered by coal inhalation, are named coal mine-dust lung disease (CMDLD) (Laney and Weissman, 2014; Perret et al., 2017).

People who are indirectly exposed to coal may be affected by chronic obstructive pulmonary disease (COPD), asthma, lung cancer, and respiratory infection. These diseases may occur due to inhalation of a diversity of environmental air pollutants. Children and the elderly are particularly susceptible (Gao et al., 2013; Raaschou-Nielsen et al., 2013; Gauderman et al., 2015; Kurt et al., 2016).

Air pollution, even with low PM_{2.5} concentrations (according to European Union limit values), but with absorbed polycyclic aromatic hydrocarbons or toxic chemicals, is associated with increased lung cancer risk (Raaschou-Nielsen et al., 2013).

A longitudinal cohort study investigated the longitudinal effects of environmental air pollutants on lung function in adults. This study reported that long-term NO₂ and PM₁₀ exposure decreased lung function (Adam et al., 2015). Besides, nitrogen oxides are described as hazardous compounds to the lungs (Kurt et al., 2016).

Nitrogen dioxide, when inhaled chronically, may impair lung physiology. Nitrogen dioxide may change the lactate dehydrogenase activity or directly induce oxidative damage in lung structural proteins, such as elastin and collagen. The prolonged exposition to nitrogen dioxide causes irreversible damage to the lung structure, inducing emphysema-like lung disease. (Hueter and Fritzhand, 1971).

3.2. Immune system

Chronic coal inhalation alters the lung's interface between the immune cells and the airborne environment (Glencross et al., 2020). According to epidemiological studies, chronic exposure in utero and/or early childhood to the solid burning fuels causes immune modulation and predisposes infants to acute lower respiratory tract infections (Lee et al., 2015).

After inhalation, the contaminants reach the lungs, where they begin to damage the epithelium's cellular structure. First, the pulmonary microbiota alteration occurs; the mucus produced by it has drastically decreased, and contamination reaches epithelial cells. The epithelial cells that suffer oxidative stress generated by the heterogeneity of PM releases signaling molecules such as cytokines and chemokines addressing immune cells (Heal et al., 2012). The pro-inflammatory signaling between epithelial cells occurs via activating and inhibitory ligand-receptor interactions, which triggers free radicals and induce damage to alveoli and bronchioles (Müller and Jaspers, 2012).

Although there is an enzymatic and non-enzymatic defense system, the demand is greater than their capacity, so the cells of the immune system need to be recruited, sometimes they are also

insufficient, and end up inducing the release of several chemotactic molecules, recruiting more immune cells and forming an extremely pro-inflammatory environment. Most of the particles are phagocytosed by natural killer (NK) cells, macrophages, neutrophils, and dendritic cells. However, the coal components cannot be digested so that it can bioaccumulate in different cells, this perpetuates the pro-inflammatory signaling, which leads to cell death and distinct pathogenesis.

After the contaminants successively affect the basement membrane, sub-epithelial connective tissue layer, and endothelial cells, and trigger oxidative stress, pro-inflammatory signaling reach the bloodstream (Zhang et al., 2018).

In the bloodstream, pro-inflammatory information will be available to all tissues in the body. In this situation, inflammatory cascades trigger blood coagulation processes. This conserved evolutionarily role of blood coagulation against invading bacteria prevents the organism from spreading infection (Loof et al., 2014). Coagulation leads to stroke, thrombosis, and atherosclerosis, intrinsically linked to cardiovascular morbidity and mortality.

3.3. Heart

Epidemiological studies, conducted in urban centers and rural areas, have provided evidence that environmental air pollution is strongly associated with increased risks of coronary artery disease, heart failure, stroke, and cardiovascular morbidity and mortality (Mehta et al., 2012; Hoek et al., 2013; Gomes and Florida-James, 2014; Adam et al., 2015; Liu et al., 2015; Aung et al., 2018; Hendryx et al., 2019).

Chronic exposition to PM_{2.5} is associated with ischemic heart disease/stroke and mortality (Hoek et al., 2013; Yu et al., 2018; Bassig et al., 2020). Other cardiovascular diseases have an association with air and water contaminated with toxicants found in coal and coal processing. (Hendryx and Zullig, 2009).

Exposure to specific air pollutants is associated with changes in the heart structure (Finch and Conklin, 2016). It is a severe problem in cardiovascular diseases; an increase in cardiac hypertrophy demands an increase in heart contraction number per minute for nutrients and oxygen to reach peripheral regions. This cardiac overload culminates in different cardiovascular diseases. The exposure to nitrogen dioxide, PM₁₀, and mainly PM_{2.5} particles are linked to an increase in the size of the left and right ventricle, leading to increased arterial blood pressures (Van Hee et al., 2009; Aung et al., 2018). A hypothesized mechanism is the development of oxidative stress, immune-mediated systemic inflammation, and endothelium dysfunction, resulting in an imbalance of vasoactive factors within the vasculature triggering hypercoagulation, cardiac hypertrophy, hypertension, atherosclerosis, thrombosis, myocardial ischemic damage, and cardiac remodeling (Kamath, 2012; Finch and Conklin, 2016; Aung et al., 2018).

3.4. Brain

The pollutants released by power plants reach their surrounding neighborhood causing a public health problem, and the brain is a target. Acute periods of exposition to high PM_{2.5} and black carbon levels are sufficient to influence children's cognitive development, including memory impairment (Alvarez-Pedrerol et al., 2017).

Neuropsychological development, brain structural changes, and air pollution have been associated. An epidemiological study showed that the exposition to polycyclic aromatic hydrocarbons, PM_{2.5}, and nitrogen oxides, results in alterations of gray and white matter volumes (Guxens et al., 2014; Suades-González et al., 2015; Erickson et al., 2020).

Among the many neurotoxic pollutants present in coal, As, Hg,

and Al are well described (Mukherjee et al., 2003; Mukherjee and Zevenhoven, 2006; Silva et al., 2010; Bilski et al., 2014; Schwartz et al., 2018). These components trigger oxidative stress and apoptosis in the central nervous system (Mochizuki, 2019). The As affects peripheral nerves, mainly sensory fibers causing painful and olfactory neuropathic diseases including anosmia and ageusia (Kawasaki et al., 2002; Ahamed et al., 2006). Axonal degeneration of peripheral nerve was observed in contaminated people by arsenic, the inhibition of thiamine uptake induces failure in myelination of nerves (Le Quesne and McLeod, 1977; Kawasaki et al., 2002).

In Neurodegenerative diseases, like Alzheimer's disease, the presence of mercury is the most substantial trace-element imbalance in the amygdala, hippocampus, and cerebral cortex (Thompson et al., 1988; Mutter et al., 2004). High levels of Hg were described in studies of brains and blood of Alzheimer's disease (AD) diagnosed people (Wallin et al., 2019). Significant amounts of Hg were evidenced in brain tissue from donors diagnosed with familial Alzheimer's disease (Mold et al., 2020).

Copper, Fe, Mn, Pb, and Zn ions interact with many enzymes and molecules in the brain. Due to their charges, these ions trigger high levels of oxygen-reactive species and cellular injury, which can result in different neurodegenerative diseases, such as Alzheimer's, Parkinson's diseases and Huntington's disease (Wang et al., 2001; Xiao et al., 2013).

Nano magnetite ($\text{PM} < 200 \text{ nm}$) formed by combustion can penetrate the brain through the olfactory nerve leading to permanent olfactory neuropathy and chronically targeting brain cells (Maher et al., 2016). The biological accumulation of magnetite in brain structures may be associate with neurodegenerative disorders (Maher et al., 2016). The nano magnetite particle accumulation was observed in the frontal cortex of 37 subjects who lived in Mexico City and Manchester, UK. It is important to note that magnetite nanoparticles match exactly with the high-temperature magnetite nanospheres generated by combustion (Maher et al., 2016).

3.5. Reproductive system

Air quality has an impact on the reproductive function of humans. Environmental pollutants induce a deficiency in gametogenesis resulting in a drop in reproductive capacities of exposed populations (Carré et al., 2017). Infertility is a multifaceted disorder comprised of male and female issues (Mahalingaiah et al., 2016).

Air contamination can alter women's reproductive health, precisely menstrual cycle characteristics. According to a cross-sectional study evaluating air pollution and human fertility, there was an association between the reduction in fertility rates and increasing levels of oxides of nitrogen and PM from traffic air contamination. The relationship was strongest for $\text{PM}_{2.5} - 10$ (Nieuwenhuijsen et al., 2014).

Air pollution exposures have been associated with low birth weight and prematurity (Mahalingaiah et al., 2016). The exposition to PM_{10} is associate with this effect, especially exposure during the first trimester of pregnancy (Merklinger-Gruchala and Kapiszewska, 2015).

The association between women exposed to air pollution and their reproductive health is demonstrated by changes in the menstrual cycle and the length of its phases (follicular and luteal) besides oocyte quality, and risk of miscarriage. Air pollution affected luteal phase shortening is a possible manifestation of luteal phase deficiency, specifically by the toxicants released by fossil fuel combustion PM_{10} and SO_2 (Merklinger-Gruchala et al., 2017).

Over the past 50 years, there has been a decline in sperm count; this effect seems to be associated with air pollution among other

factors (Hallak et al., 2018). As male fertility is, to some extent, correlated with sperm count, the results may reproduce a general decrease in male fertility (Carlsen et al., 1992).

Coal miners have a significant decline in semen volume and seminal viscosity. It can be hypothesized that coal dust exposure might be responsible for its result since the high level of Pb and Cd was found in blood and semen of coal mine workers after a long term (10–25 years) exposure. The presence of Pb and Cd in both these biological fluids suggests that they might cross blood testis-barrier at the present exposure level and subsequently produce detrimental effects on sperm structure and sperm parameters (Mohanty and Mahananda, 2015).

Smoking and alcohol consumption have been associated with decreased sperm counts and abnormal sperm morphologically (Joo et al., 2012). Coal mine workers smoke and consume more alcohol than non-occupationally exposed subjects, increasing risk factors for infertility (Mohanty and Mahananda, 2015).

3.6. DNA damage

Since the air pollutants can trigger pro-inflammatory modulation in the lung or immune cells or even the presence of nano or microparticles themselves in the circulatory system, this mixture containing heavy metals and organic/inorganic material can trigger damage to important macromolecules like DNA.

Genetic parameters were investigated in peripheral lymphocytes of workers exposed to coal mining residues. These workers had high concentrations of Al and Si in their blood (Leon-Mejia et al., 2014). Besides occupational exposure to coal dust result in cytogenetic damage (Donbak et al., 2005; Leon-Mejia et al., 2011; León-Mejía et al., 2014).

The disorders related to coal inhalation can be originated in genetic damage (Leon-Mejia et al., 2014). The interaction of particles with immune cells and epithelial cells can generate reactive oxygen species (Schins and Born, 1999; Leon-Mejia et al., 2014; Walton et al., 2016). High levels of reactive oxygen species within the cells induce signaling for many metabolism pathways, resulting in translocation/transcription of pro-inflammatory genes resulting in an exacerbated increase in cytokines and proteins associated with cellular damage. Under these conditions, lipoperoxidation, membrane disruption, protein modification, apoptosis, mitochondrial damage, and DNA damage are just a matter of time. Telomeres length increase with enhancing annual exposure to NO_x , $\text{PM}_{2.5}$, and PM_{10} , and this effect have an association with age-related diseases (Barth et al., 2017; Carré et al., 2017).

The population that resides near the coal mines presents cytogenetic instability. There is a spatial relationship between $\text{PM}_{2.5}$ air concentration and micronuclei frequency in binucleated and mononucleated cells (Espitia-Pérez et al., 2018). The exposition to $\text{PM}_{2.5}$ characterizes the most significant health risk for residents near open-pit mines. Sulfur, Cu, and Cr were highly enriched in $\text{PM}_{2.5}$ from coal mining areas (Espitia-Pérez et al., 2018). Topography and wind in mining areas are significant contributors to amplify the spread of $\text{PM}_{2.5}$. (Espitia-Pérez et al., 2018).

Air pollution may induce epigenetic modifications, which affect the genomic structure, like DNA methylation, regulating gene expression without affecting the DNA sequence. The epigenetic modifications have been implicated in the effect of air pollution on carcinogenesis, and lung function (Holloway et al., 2012; Lepeule et al., 2014; Cao, 2015; Carré et al., 2017).

3.7. Comorbidities

Comorbidity can be defined as the occurrence of additional diseases in relation to an index disease in one person. It is linked

with worse health consequences and more complex clinical management (Valderas et al., 2009). Hypertension, obesity, and diabetes are common comorbidities for hospitalization (Fraze et al., 2010).

Air pollution triggers pathologies on individuals exposed to pollution; however, some people are more susceptible to hazardous elements. Characteristics of vulnerability include socioeconomic factors; genetic predisposition; life stage; and preexisting diseases, such as asthma, chronic obstructive pulmonary, respiratory system disease, cystic fibrosis, hypertension, cardiovascular disease, or diabetes (Camiciottoli et al., 2016; Hooper and Kaufman, 2018).

The prevalence of chronic diseases associated with environmental contamination is increasing annually. Obese people are more susceptible to the harmful properties of air pollution (Limaye and Salvi, 2014). A cohort study showed that overweight children were more susceptible to the pulmonary effects of indoor PM_{2.5} and NO₂ than lean children (Lu et al., 2013). Higher exposure to air contamination, obesity, and nonatopic status are associated with poorer asthma control among older asthmatic patients (Epstein et al., 2012). On the other hand, exposition to high concentrations of ambient air pollution is associated with overweight and obesity among children (2–13 years old) (Dong et al., 2012). PM_{2.5} is associated with many diseases and contributed to about 4.2 million premature deaths in 2015 (Cohen et al., 2017; Bowe et al., 2018).

According to epidemiological studies, smoking and cumulative total dust exposure led to a cumulative abnormal rate of pulmonary function in coal-miner workers (Kuempel et al., 2009; Taeger et al., 2015; Qian et al., 2016). Therefore, air contamination is an important environmental risk factor for lung diseases, and the smoking association may increase lung damage (Yu et al., 2015; van der Zee et al., 2016).

4. Conclusion

In comparison, coal is the energy source that emits more nitrogen oxides, sulfur dioxide, carbon dioxide, heavy metals, and particulate matter per unit of energy than other fuel sources. For each ton of oil equivalent (TOE) of coal consumed in electricity generation, close to four tons of CO₂ are emitted. In the case of oil, CO₂ emissions are close to three tons per TOE consumed. Efforts to implementation of clean energy must be prioritized in governmental initiatives around the world to reduce air pollutant emissions concerning life.

The coal combustion-based power generation emits large quantities of massive pollutants impacting fauna and flora. Thousands of people around the world get sick or die from reasons related to inhaling coal/pollution. The substances in coal react chemically with two essential elements for life: water and air. These chemical changes are perennial, so the use of coal as a generating source is a setback; it depletes the natural sources of human survival. Uncontrolled emission of gases and particles is unacceptable nowadays.

Smoking, poor or excessive diets, lack of exercise, and comorbidities are factors that can aggravate the effects caused by air pollution. When coal hazardous elements interact with vulnerable human cells, pathogenesis is likely to be inevitable. Thus, the utilization of coal as an energy source need to ensure that adequate measures are in place for environmental protection and to safeguard human health.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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