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Effect of Dust Pollution of Coal and Coal Chemical Industries on the Risk of Developing Heart Diseases

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Abstract

This review exposes the issue of the relationship between exposure to dust particles from coal and coal processing industries with the risk of developing cardiovascular diseases (CVD). Typical features of exposure to dust particles of this type involves: 1) high levels and the chronic type of dust pollution, 2) a small aerodynamic diameter (AD) of dust particles (below 1.0 μm) that facilitates their inhalation and entry into teethridge, 3) their trend towards aggregating to clusters with an AD between 2.5 μm and 10 μm that set in the upper respiratory tract, and 4) specific chemical composition (carbon, silicon dioxide, alumina, sulphates, carbonates, nitrates, and volatile organic matter). In spite of the generally proven relationship of exposure to all fractions of dust particles with elevated morbidity and mortality caused by CVD, the proportion of cases triggered by dust particles regardless of respiratory system diseases that are characterised by comorbidity with CVD remains unclear. Furthermore, there is an obvious shortage of environmental epidemiology research with monitoring the particles with an aerodynamic diameter of less than 0.1 μm, exposure to which could most likely explain the relationship between dust pollution and CVD (the particles with so small AD are able to penetrate to the systemic circulation). At last, the majority of multicentre epidemiological investigations that substantiate relationships between dust particles and developing CVD have been performed in developed countries. Carrying out such research in developing countries, considering their geographical, climatic, and socioeconomic peculiarities, and also characteristics of the implementation of coal mining and processing, would facilitate the research on population interlinks of exposure to dust particles with other CVD risk factors and mechanisms of their pathogenic action. The ultimate objective of such research is to make the personified programme of CVD preventive care considering exposure level and duration to dust particles and co-occurring risk factors.

Key words: dust pollution, suspended dust particles, coal industry, cardiovascular diseases, ischemic heart disease

INTRODUCTION

Urbanization, industrialization, and globalization that are typical of the modern development of economy not only contribute to changing lifestyle but are also associated with the appearance of new risk factors of developing cardiovascular diseases (CVD), such as anthropogenic environmental pollution. According to the World Health Organization (WHO), atmospheric air pollution became the cause of 4.2 million cases of premature death in 2016 [1]. If cause-and-effect relationships of anthropogenic air pollution and respiratory system diseases have been explored for quite a long time [2], associative relationships between aeropollutants and the risk of developing CVD have become extensively investigated only in the two last decades.

Alongside with that, according to WHO assessments, the majority (~54 %) of cases of pre-

mature death linked to atmospheric air pollution took place resulting from ischemic heart disease (IHD) and acute cerebrovascular accident (ACA), and only 40 % as a result of chronic obstructive pulmonary disease (COPD) or lower respiratory tract infections [1]. The main cause of death rate is exposure to dust (also called aerosol) pollution indicated in state environmental standards as suspended dust particles [3–5]. The death rate of the Russian population because of aerosol air pollution is 75.6 per 100 000 people [6]. This problem is especially relevant for developing countries where the combustion of coal, fuel wood, or biomass to heat a house and cook food are common, which results in a substantial increase in the number of such particles both in the atmosphere (outdoor air pollution) as a whole and building spaces (indoor/household air pollution) [5, 7].

From the standpoint of geoecology and geochemistry, dust pollution (the term the atmospheric particulate matter or simply particulate matter (PM) is accepted in English literature) acts as one of the components that determine atmospheric chemical composition, climatic peculiarities, and biochemical cycles [7–11]. The term dust pollution, or aerosol pollution, describes PM (dust) found suspended in the atmosphere and liquid droplets formed upon vapour condensation or under the interaction of gas media or fallen into the air medium without changing phase composition [7–14].

Atmospheric dust particles are characterised by wide structure and shape variability, however, aerodynamic diameter (AD) has been accepted as their determining characteristic. A particle with an AD of 1 μm has the same inertial properties as a sphere with a diameter of 1 μm and a density of 1 g/cm^3 regardless of the actual size, shape, and density of the former [5, 7, 8, 11]. Dust particle distribution in the lower atmospheric layers usually has three peaks in AD ranges below 0.1 μm ($PM_{0.1}$), between 0.1 and 2.5 μm ($PM_{2.5}$), and also over 2.5 μm (PM_{10}). Among them, $PM_{0.1}$ is most common in the atmosphere, but PM_{10} has the highest mass [5, 7, 8, 11].

The quantitative assessment of dust pollution is carried out according to the mass concentration of the particles depending on their AD (as a rule, PM_{10} or $PM_{2.5}$) [7]. These AD values correspond to dust particles that fall into the upper respiratory tract (PM_{10}) and alveoles ($PM_{2.5}$) [7]. The number of PM_{10} generally varies in urbanized territories between dozens and hundreds of mg/m^3 [7].

In modern conditions dust pollution particles are mainly comprised of natural components of the earth's crust (carbonates and silicates), inorganic compounds (*e.g.*, sulphates, nitrates, chlorides, sodium, potassium, and ammonium salts), trace amounts of I and II group metals, other chemical elements (copper, arsenic, zinc, and vanadium), organic compounds (soot, carbon black, polycyclic aromatic hydrocarbons, and secondary organic aerosols formed resulting from oxidation followed by condensation of volatile organic matter) [7, 8, 11].

A high concentration of dust pollution particles in the atmospheric air reduces its quantity, visibility, and sunlight transmission, and also is able to result in changing the climate. Furthermore, these particles are of high importance in atmospheric chemistry providing the surface for various reactions probable only upon the presence of dust pollution. Dust transport is an important component of biochemical cycles for chemical elements of the earth's crust (*e.g.*, iron), as it ensures their ingress into aqueous ecosystems [5, 7].

It is worth noting that quality standards in the area of dust pollution vary significantly among various controlling organizations and countries. Thus, according to the guidelines of the Russian Federation (RF), the maximum permissible annual average concentration of PM_{10} is 40 mg/m^3, whereas that of PM_{25} is 25 mg/m^3 [15]. Maximum permissible average daily concentrations of the particles of these two types are 60 and 35 mg/m^3, respectively. Herewith, there should not be cases of exceeding both annual average and median diurnal maximum permissible concentrations [7].

The objective of this review was to critically analyse the effect of atmospheric air pollution with dust particles with various aerodynamic diameter and chemical composition (in particular, those typical for coal and coal chemical industries) on CVD morbidity and mortality in developed and developing countries.

CHARACTERISTICS OF PECULIARITIES OF DUST POLLUTION CAUSED BY COAL AND COAL CHEMICAL INDUSTRIES

When investigating the influence of dust pollution of different types on the environment and human health, it is important to consider the source of the effect. It is widely recognized that almost any branch of industry is characterised by its own peculiarities of environmental dust emissions; coal mining is also not an exception.

When the depth of mines is increased as a consequence of the violation of their ventilation and, accordingly, the removal of dust particles therefrom, both the measurement of dust pollu-

tion distribution and the appropriate assessment of dust particle exposure to mine workers are complicated [16, 17]. Coal mining equipment and appropriate transport generate significant quantities of particles, which directly fall into the environment and that mine workers are exposed to [16, 17]. Herewith, various processes used in coal mining (drilling, explosive works, coal loading, transporting, and unloading) are accompanied by emissions of various size dust [16, 17]. However, the common regularity of dust pollution caused by coal mining is the release of PM_{10} [16, 17].

The top layer is removed by open-cut mining at the first step of coal mining, which is accompanied with a relatively minor mechanical release of dust particles (1 % of the total amount released upon coal dust mining) out of rock layers over coal [16]. Large particle volumes by an order of magnitude higher (around 10 % of the total amount) are evolved when rock boring or blasting for the purpose of obtaining the access to coal seams. For example, around 660 kg of dust per day is released in India resulting from these processes [18]. Furthermore, coal seams also require boring in some cases or explosive works for better access to coal and therefore for the purpose of increasing the amount of coal produced [19]. Further seams weathering facilitates particles evolution into the environment and ensures 10–20 % of the total volume of dust pollution upon coal mining. Dust concentration is 15–30 mg/m^3 of atmospheric air upon rock boring or blasting [18]. Thereafter, slag rocks are loaded onto trucks, transported to the place of the disposal area, unloaded, and tamped by bulldozers. On the contrary, after loading onto trucks, coal is delivered to concentrating mills or its disposal sites or further transportation points where unloading takes place. The main release of dust particles into the environment (70–80 % of the total amount) proceeds precisely at the stage of loading, transporting, and unloading slag rocks [16–18, 20, 21].

Dust pollution upon coal combustion is characterised by the three modal particle size distribution with AD peaks in the 0.1 μm area, the 0.8–2 μm range, and around 10 μm [9, 10]. These particles are formed resulting from evaporation, nucleation, condensation, and aggregation [9, 10]. There is the first phase in the furnace when increasing temperature upon coal combustion when much inorganic mineral resources are evaporated [9, 10]. Vapours cool down in the course of their mechanical removal from the combustion source and therefore a decrease in temperature and form particles with a diameter of 1–10 nm. The latter are able to aggregate into larger ones [9, 10]. In addition, some vapours are condensed on the surface of the particles already formed even greater increasing their diameter [9, 10].

Especially large particles may be formed resulting from the fragmentation of microparticles due to their pore structure upon aerobic oxidation. In turn, microparticles appear upon high temperature resistance of minerals [9, 10]. Nevertheless, unlike coal mining, most dust particles evolved upon its combustion have an AD below 2.5 μm [9, 10].

The specificity of dust pollution caused by coal chemical enterprises consists in the fact that $PM_{2.5}$ and $PM_{0.1}$ are prevailing therein. The latter are comprised of compounds (carbon, silicon dioxide, alumina, iron (III) oxide, sulphur oxide, sodium oxide, and titanium dioxide) typical for side products of the coal industry, and also specific co-products from the chemical industry upon specific coal chemical processes [9, 10].

The elemental composition of dust pollution depends on both geological peculiarities of a specific coal pit or a mine and an AD of the particles [9, 10]. As shown, particles with an AD of 1–10 μm mainly contain silicon, aluminium, and iron, whereas the main components of dust particles with an AD below 1 μm – sodium, potassium, calcium, and sulphur [9, 10]. As a whole, compounds that are a part of dust pollution caused by the coal industry may be divided into three types: elements directly coupled with coal, those associated therewith, and species that form discrete inorganic mineral grains in coal, particles [9, 10]. Inorganic components of coal dust are mainly presented by alkali and alkali earth metal cations (Fe^{3+}, Mg^{2+}, Ca^{2+}, Na^{+}, and K^{+}) that are a part of clay minerals, carbides, carbonates, sulphates, chlorides, silicates, *etc*., and also trace elements, such as arsenic, beryllium, cadmium, cobalt, chromium, mercury, manganese, nickel, lead, antimony, and selenium [9, 10]. The major inorganic compounds of coal dust are carbon, silicon dioxide, aluminium oxide, iron (III) oxide, sulphur oxide, calcium oxide, magnesium oxide, potassium oxide, sodium oxide, and titanium dioxide [9, 10]. Volatile organic matter with a high carbon content is prevailing among inorganic components [9, 10].

Based on the above, one may conclude that dust pollution caused by opencast coal mines is characterised by the prevalence of PM_{10}, whereas that caused by coal and coal chemical enterprises – $PM_{2.5}$ and $PM_{0.1}$. Moreover, dust pollution caused by coal mining enterprises is characterised by the prevalence of carbon, silicon dioxide, and alumina, whereas that through coal processing plants – the high acidity due to significant

quantities of sulphates, nitrates, and carbonates, and also highly volatility because of low AD of dust particles in the presence of volatile organic matter. All this determines mechanisms of their injurious action on both human health and the environment.

PROOFS OF THE RELATIONSHIP BETWEEN DUST POLLUTION AND THE EVOLUTION OF CARDIOVASCULAR DISEASES

It is known that the localization of dust pollution particles in the human body after breathing in directly depends on their size: PM_{10} are sedimented in the upper respiratory airway, whereas $PM_{2.5}$ may penetrate into alveoles, and ПМ$_{0.1}$ into – the systemic circulation bypassing the aero-hematic barrier [4]. The relationship between dust pollution and the risk of developing chronic obstructive pulmonary disease (COPD) and bronchial asthma (BA) has been proven. The latter are characterised by risk factors (smoking, obesity, arterial hypertension, and metabolic syndrome) similar to cardiovascular diseases and represent important predictors of unfavourable prediction of CVD evolution [22, 23]. The relationship substantially complicates the assessment of development frequency of dust particles associated with exposure and thereat independent on respiratory system diseases, CVD, when exposed to common risk factors.

When PM_{10} level is increased by 10 μg/m^3 or upon a drastic increase in $PM_{2.5}$ level, a COPD hospitalization risk is elevated by 0.9–2.5 % [24, 25]. It is believed that mortality risk run is increased by 0.4–1.5 % and 0.6–1.2 % in the short upon an increase in PM_{10} and $PM_{2.5}$ levels by 20 μg/m^3 and 10 μg/m^3, respectively [26]. As shown, a 10 μg/m^3 increase in $PM_{2.5}$ level increases mortality risk by 10–14 % in the long run [27, 28]. According to encouraging assessments by experts, a 20 % reduction in PM_{10} level may decrease the number of death cases related to this by 30 % in the short run [29].

Upon a 10 μg/m^3 increase in $PM_{2.5}$, CVD morbidity is enhanced by about 21 % [30]. Even a short-time increase in dust particle concentration by 7.1 μg per 1 m^3 of atmospheric air causes increasing risk development for myocardial infarction (MI) by about 18 % [31]. Furthermore, chronic dust exposure to the particles also increases the risk of developing CVD [4, 5]. For example, when PM_{10} level is increased by 10 μg/m^3, the risk of developing coronary artery disease (CAD) growth by 12 %, and $PM_{2.5}$ per 5 μg/m^3 – by 13 % [32]. A similar relationship was also detected for chronic heart failure (CHF) [33]. The risk of developing fatal and non-fatal acute cerebrovascular events (ACE) is increased by 83 and 35 %, respectively, upon an increase in $PM_{2.5}$ level by 10 μg/m^3 [30]. Meta-analysis of 34 investigations proves the relationship between emission outbreaks of $PM_{2.5}$ and an elevated risk of developing MI [34]. As demonstrated by another meta-analysis of 195 investigations, even a short-term increase in the degree of dust particles increases the risk of hospitalizations and death caused by chronic heart failure (CHF) [35]. Herewith, it is important to note the negative synergetic effect of PM_{25} when tobacco products are consumed, which ultimately results in a cumulative increase in the risk of emerging CVD [36].

It has been proven that even the short time exposure of significant amounts of dust particles is associated with an increased death number caused by circulatory diseases (CD) (approximate numbers are 0.6–1.8 % when PM_{10} is increased by 20 μg/m^3) and 0.6–1.3 % upon a 10 μg/m^3 increase in $PM_{2.5}$ [27]. According to meta-analysis involving 14 cohort investigations on assessing the accumulative action of $PM_{2.5}$, it has been found that a 10 μg/m^3 increase in $PM_{2.5}$ content in the air leads to increased mortality caused by CVD by 12–14 % [37]. At the same time, only one of these investigations has been carried out in a developing country [37]. The risk of developing ACE varies between 12 and 83 % upon a similar increase in $PM_{2.5}$ content. Nevertheless, only six similar cohort investigations have been performed in the whole world, moreover, all of them – in developing countries [37]. Thus, almost all epidemiological investigations have been carried out in developed countries where dust particle concentrations in the atmosphere are relatively low compared to developing countries. Furthermore, data in relation to unfavorable cardiovascular, respiratory, and other event are limited by a concentration range of $PM_{0.5}$ within 25–1000 μg/m^3. For that reason, the research on the effect of the accumulative impact of dust particles on developing CVD is relevant; the issue of identifying high-risk groups remains open.

As demonstrated by experimental data, the trend towards developing atherosclerosis in ApoE knockout mice upon two-month exposure to a mixture of dust fractions with different sizes, an increase in the level of cholesterol and oxidised lipoproteins of low density (LPLD) has been found in blood serum [38]. It has also been illustrated in one of the epidemiological investigations that chronic exposition of $PM_{2.5}$ to young men is associated with an increased level of LPLD in blood serum [39]. Moreover, exposure of PM_{10} for 24 h

resulted in an increase in the level of products of the reaction with thiobarbituric acid in patients with type 2 diabetes [40]. In general, in spite of the presence of fragmented evidence of an increase in the oxidation level of lipids typical for developing atherosclerosis upon exposure to dust particles and causing endothelium dysfunction, that issue has not received adequate coverage and requires scale epidemiological analysis, as clinical observations are in need of a pathophysiological validation.

It is required to consider that depending on chemical composition, dust particles may be different according to the degree of toxicity for various systems of organs. In addition, dust particles are able to adsorb on their surface numerous chemical compounds with various degrees of toxicity [41]. For example, the chemical composition of dust particles caused by coal mining (carbon, silicon dioxide, and alumina) and coal processing industries (sulphates, carbonates, and volatile organic matter) is associated with an increase in the probability of myocardial infarction (MI) [42]. Hence, research in regions with various chemical compositions of particles may also be different according to the expressiveness of their relationship with development risk and mortality caused by CVD.

It is worth noting that apart from size, chemical composition has a substantial effect on the toxicity of dust particles. In one of the first works devoted to this problem, it was demonstrated that exposure of $PM_{2.5}$ caused by transport facilities was higher than the former of $PM_{2.5}$ through coal dust (3.4 and 1.1 %, respectively, upon an increase in 10 $\mu g/m^3$) according to the contribution to mortality [43]. Other research has shown the relationship between high contents of a carbon component and the level of nitrates in the dust caused by the chemical industry linked to aluminum, nickel, chromium, bromine, silicon, manganese, selenium, sodium, sulphates, and arsenic also increase the risk of hospitalizations by reason of circulatory system diseases (CSD) and that of myocardial infarction and death for the exposed population [45–47].

PECULIARITIES OF THE EFFECT OF DUST POLLUTION CAUSED BY COAL AND COAL CHEMICAL INDUSTRIES ON DEVELOPING CARDIOVASCULAR DISEASES AND PATHOLOGIES CAUSING THEM

Currently, it has been widely recognized that dust pollution caused by coal-mining and coal processing enterprises has a wide range of a negative impact on the human organism. Furthermore, this has been proven by a number of epidemiological and pathophysiological investigations [48, 49]. Reducing this effect requires either a reduction in coal mining and processing volumes or an increase in the environmental friendliness of the chain of coal recovery and further use (removing the upper layer of soil – coal drilling – blasting – loading – transporting – processing) at the expense of developing technologies. Both approaches are associated with significant costs and hence with a decrease in the earning power of coal mining and processing, which complicates the introduction of the former.

The effects of dust pollution caused by coal and coal chemical industries on the cardiovascular system become apparent not only after emission outbreaks of a large volume of dust particles but also resulting from the chronic exposure of relatively small but constant amounts of dust particles [4, 5]. In this case, risk groups are children and elderly people, pregnant, and patients with several pathologies that exert synergizing negative effect (the so-called effect of comorbidity) [4, 5].

Exposure to dust particles caused by coal enterprises is associated with diminished lung function, COPD, acute infectional and allergic diseases of the respiratory tract, and also lung cancer. In particular, it has been demonstrated that an increase in the level of PM_{10} caused by coal industry by 10 mg/m^3 increases the rate of hospitalizations because of COPD by 2.5 %, and $PM_{2.5}$ particles through the coal industry – by 0.9 % [5]. The direct relationship between the level of dust pollution and mortality caused by COPD has also been shown [5]. Moreover, it has been found that an increase in the level of PM_{10} caused by the coal industry by 10 $\mu g/m^3$ increases the risk of developing lung cancer by 20 % [5]. Extreme outbreaks of emission of dust particles through coal enterprises to atmospheric air are associated with enhanced population mortality by 2–8 % per each 50 g/m^3 of dust [50]. At the same time, the improvement of lung function has been demonstrated under big industrial city conditions when reducing the extent of dust pollution [5]. Inhalation of silicon-containing particles is associated with developing lung cancer, pneumoconiosis, and silicosis; whereas silico-tuberculosis is developed upon accompanying tuberculosis [51, 52]. Apart from exposure to coal dust particles in the open air, the former indoors (*e.g.*, at the workplace) in association with such professional diseases, as lung fibrosis, histiocytosis, desquamative interstitial pneumonia and respiratory bronchiolitis associated with interstitial lung disease, may also be hazardous [5].

In order to explain, at first glance, the counter-intuitive relationship between dust pollution caused by coal enterprises and cardiovascular system diseases, the whole range of pathogenetic mechanisms, including the activation of routes of systemic inflammation after inhalation of dust particles, the synthesis of reactive oxygen species by various cells in response to reduced lung function and disrupted blood clotting ability, have been suggested [5]. In fact, the relationship between exposure to coal dust particles and an enhanced risk of developing CVD has been demonstrated in a series of cohort investigations. It is believed that a 10 $\mu g/m^3$ increase in the level of $PM_{2.5}$ caused by the coal industry gives rise to a 21 % increase CVD level [30]. According to other assessments, a 10 $\mu g/m^3$ increase in the level of PM_{10} caused by the coal industry increases the risk of developing ischemic heart disease (IHD) by 12 % and that of $PM_{2.5}$ by 5 $\mu g/m^3$ – of 13 % [32]. A similar relationship has also been found with various types of IHD, such as unstable stenocardia and congestive heart failure (CHF) [33]. It has been demonstrated in one of the last systematic reviews and meta-analyses that even a short-time (before 5 years) increase in the level of dust particles caused by coal enterprises enhances the risk of hospitalization and CHF mortality [35]. Furthermore, in addition to IHD, exposure to coal dust particles is associated with acute cerebrovascular accident (ACA) [30]. The risk of developing lethal and non-lethal ACA is increased by 83 and 35 %, respectively, with a 10 g/m^3 increase in $PM_{2.5}$ level [39].

Apart from arterial diseases, exposure to dust pollution caused by coal industry enterprises causes enhanced blood clotting and vein thrombosis. For example, the relationship between $PM_{2.5}$ caused by the coal industry with both vein thrombosis and pulmonary embolism has been demonstrated in one of the recent papers [53]. However, another research work on hospitalizations concerning vein thrombosis linked exposure to $PM_{2.5}$ dust particles but not to PM_{10} ones caused by the coal industry [54]. It is believed that a 10 $\mu g/m^3$ increase in the level of PM_{10} caused by coal dust increases the risk of developing vein thrombosis by 70 % [55, 56].

CONCLUSION

All fractions of dust particles evolved in the atmosphere upon coal mining and processing significantly increase the risk of developing cardiovascular diseases (CVD) and respiratory system diseases causing them. Apart from the direct toxic action of dust particles caused by coal enterprises, chemical matter by itself and elements comprising thereof are also hazardous. They possess their own toxic effects not related to the corpuscular nature of particles. Coal pollution caused by both the coal industry as a whole, and also by coal mining and coal processing enterprises is characterised by high degrees of the former, small aerodynamic diameter (AD) of dust particles (below 1.0 μm) that facilitates their breathing in and penetration into alveoles, the trend of dust particles towards aggregation into clusters with an AD between 2.5 and 10 μm, and also by specific chemical composition (carbon, silicon dioxide, alumina, sulphates, carbonates, nitrates, and volatile organic matter). All this determines the high toxicity of dust particles caused by coal and coal processing industries for the cardiovascular system. However, the issue of whether PM_{10} and $PM_{2.5}$ are directly responsible for developing CVD or only for respiratory system diseases potentiating them remains open in this case. In turn, analysis of the relationship between $PM_{0.1}$ that enter the systemic circulation and CVD is complicated because of lack or inaccuracy (depending on the region) of monitoring their degree in atmospheric air. Furthermore, the overwhelming majority of epidemiological investigations that substantiate relationships between dust particles and developing CVD have been carried out in developed countries. The extension of such multi-centre investigations to a number of developing countries with different geographical, climatic, and socio-economic peculiarities and various characteristics in organizing coal mining and processing would certainly help decoding both population interlinks of exposure to dust particles with other CVD risk factors and mechanisms of their action at tissular, subcellular, and molecular levels. This is likely to assist in developing the personified programme of the primary and secondary prevention of CVD in the long run.

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