Original Article Pol J Public Health, Vol. 133 (2023): 11-15

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Air pollution – a factor of the cardiovascular system diseases

Abstract

Introduction. Air pollution is any chemical, physical or biological agent that alters the natural composition of the atmosphere. It is the cause of many respiratory, circulatory and nervous system diseases, as well as the occurrence of allergies, cancer and fertility problems.

Aim. The purpose of this study was to summarize the effects of polluted air on the cardiovascular system. This impact is associated with the whole spectrum of negative effects from additional hospitalizations to premature deaths.

Materials and methods. A review of the literature available in the PubMed database was conducted published by the World Health Organization (WHO) and European Environment Agency (EEA).

Basic results. Air pollution is a real threat to the cardiovascular system. The human population is constantly exposed to the toxins in the air we breathe from tobacco smoke and fireplace smoking to industrial emissions and volcanic eruptions. It is not just a problem for large metropolitan areas, but a huge global problem. It is worth noting that polluted air is not only outside, but also indoors where we live despite our supposed sense of security.

Conclusions. This is a huge problem that cannot be ignored, and a public health challenge. It is important to constantly raise awareness of the magnitude of the problem, take action on many levels and promote prevention to minimize exposure as much as possible and continue to improve air quality.

Keywords: air pollution, cardiovascular disease, particulate matter, myocardial infarction, arrhythmia.

DOI: 10.12923/2083-4829/2023-0003

INTRODUCTION

Ongoing efforts are being made to detect early risk factors for cardiovascular diseases. Exposure to toxins in the air has a significant impact on the circulatory system, yet this factor is often overlooked, despite being ubiquitous and affecting the majority of the global population [1]. Outdoor air pollution is the fifth largest risk factor for mortality from any cause [2]. It is the largest environmental health threat in Europe and has a significant impact on the health of the European population, particularly in urban areas [3]. The World Health Organization (WHO) estimates that it causes approximately 4.2 million deaths worldwide each year [4]. In 2020, exposure to concentrations of fine particulate matter exceeding the guidelines of the World Health Organization resulted in 238,000 premature deaths in Europe [3]. Between 40% and 80% of mortality caused by air pollution in Europe is attributed to cardiovascular events. Poor air quality shortens the average lifespan in Europe by about 2.2 years [5].

Composition and sources of polluted air

Atmospheric air pollution arises from emissions originating from industrial activities, households, passenger cars, and trucks, which constitute complex mixtures [4]. Emissions of pollutants include gases such as nitrogen oxide (NO), nitrogen dioxide (NO_2), carbon monoxide (CO), sulfur dioxide (SO_2), and particulate matter (PM) [6,7]. In Europe, NO_2 primarily contaminates the air due to road traffic and energy production through the combustion of fossil fuels [8]. The highest concentrations are found in certain large cities with significant traffic intensity [9]. Carbon monoxide in urban atmospheres is mainly produced by vehicles but also originates from industrial coal production, gas combustion, and cooking [10]. SO_2 is primarily emitted by industry [8]. Ozone (O_3) is a secondary gaseous pollutant that forms through photochemical reactions involving sunlight and gaseous precursors such as nitrogen oxides or volatile organic compounds [11]. Benzopyrene is a carcinogenic pollutant emitted primarily from coal and wood combustion for heating, as well as to a lesser extent from agricultural waste burning and industrial installations [9].

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Gaseous pollutants, such as sulfur dioxide, nitrogen oxides, carbon monoxide, and ozone, pose health risks, but it is PM that is the main culprit with the greatest impact on the cardiovascular system [6,7].

Particulate matter (PM) is a mixture of solid and liquid particles suspended in the air, resulting from both anthropogenic activities (road traffic, households, agriculture, industry, wood and fossil fuel burning, construction, and demolition) and natural sources (windblown dust, wildfires, volcanic eruptions) [8]. PM belongs to heterogeneous substances, having different sizes and varying chemical compositions [8].

PM can be divided into three categories based on particle size in the environment: coarse particles, also known as PM_{10}, with an aerodynamic diameter of <10 μm; fine particles, $PM_{2.5}$, with an aerodynamic diameter of <2.5 μm; and ultrafine particles (UFP) with a diameter <100 nm, also referred to as "nanoparticles" [11]. Among all these pollutants, fine particulate matter has the greatest impact on human health [4].

PM_{10} is mainly emitted from the combustion of solid fuels for residential heating, but it also originates from industry, agriculture, and road transport [9]. It also comes from natural sources such as forest fires and bioaerosols (endotoxins, fungal spores, pollen) [6,7]. Concentrations exceeding the daily allowable limit in the European Union for PM_{10} are primarily observed in Italy and some Eastern European countries [9].

$PM_{2.5}$ particles are smaller, multiple times smaller than the diameter of a human hair. $PM_{2.5}$ is a heterogeneous combination of various compounds formed through combustion or secondary atmospheric reactions (nitrates, sulfates). $PM_{2.5}$ can remain suspended in the air for longer periods, even several days, and can be transported over distances of hundreds of kilometers [12]. The highest concentrations of $PM_{2.5}$ have been observed in northern Italy and some Eastern European countries [9].

UFP particles are generally short-lived, remaining airborne for many hours. They originate from sources such as road traffic, diesel engine exhaust particles, and smokestack emissions from factories [6,12]. UFP particles quickly aggregate, absorbing water, organic material, and other gases, reaching particle sizes within the $PM_{2.5}$ range [6].

The concentration of particulate matter (PM) is measured in micrograms per cubic meter (μg/m³), and the World Health Organization (WHO) has established guidelines for air quality (AQG) with a level of <10 μg/m³ for the annual average concentration. These are the lowest levels above which an increase in total mortality, cardiovascular and lung diseases, and lung cancer has been shown with over 95% certainty in response to long-term exposure to $PM_{2.5}$ [12].

In 2021, 97% of urban residents were exposed to concentrations of fine particulate matter above the recommended health level set by the WHO [9]. Short-term mortality is associated with PM_{10}, while long-term mortality with exposure to $PM_{2.5}$ [6]. Even short-term increases in $PM_{2.5}$ concentrations raise the relative risk of acute cardiovascular events by 1-3% within a few days. Prolonged exposure over several years increases this risk by approximately 10%, which can be partially attributed to the development of cardiometabolic diseases [12].

In Europe, the highest burden of $PM_{2.5}$-related mortality has been estimated for cities in northern Italy, Poland, and the Czech Republic, while the highest NO_2-related mortality has been estimated for large cities and capitals in western and southern Europe. The high mortality associated with $PM_{2.5}$ in these countries is related to a higher degree of anthropogenic emissions and unfavorable climatic conditions in these areas. Polish cities such as Jastrzębie-Zdrój and Rybnik have one of the highest estimated mortality rates in Europe [13].

Impact on the cardiovascular system – pathophysiology

Among the cardiovascular diseases associated with air pollution, myocardial infarction, heart failure, cardiac arrhythmias, atherosclerosis, and even cardiac arrest can be mentioned [6].

Polluted air exerts a prothrombotic effect through the activation of multiple pathophysiological processes. It has been shown that exposure to air pollution affects each stage of homeostasis, and increasing evidence suggests that the overall hemostatic balance is shifted towards a procoagulant and antifibrinolytic state. Platelet activation, oxidative stress, the interaction between interleukin-6 and tissue factor, along with the emerging role of circulating microparticles and epigenetic changes, appear to be potentially important mechanisms increasing the thrombotic risk induced by pollution. Acute exposure to $PM_{2.5}$ induces a shift in the hemostatic balance towards a prothrombotic state [14,15]. Oxidative stress induced by air pollutants can contribute to the development of many diseases. It is defined as a process in which an imbalance between the oxidative and antioxidative systems, caused by metabolic disorders or an excess of reactive oxygen species (ROS) along with harmful factors, induces gene mutations, protein denaturation, and lipid peroxidation, leading to physiological, biochemical, and metabolic dysfunction of cells. The surface of ultrafine particles (UFP) and $PM_{2.5}$ contains transition metals (including iron, copper, zinc, manganese), polycyclic aromatic hydrocarbons, and lipopolysaccharides, which possess properties of active free radicals and stimulate the production of ROS in the body [16].

Stroke, heart attack – anything else?

In Shanghai, a study was conducted on healthy young adults who were exposed to air pollution associated with traffic-related emissions, including fine and ultrafine particulate matter, black carbon, nitrogen dioxide, and carbon monoxide. Dozens of molecular changes in metabolites, lipids, and proteins were found, including systemic inflammation, oxidative stress, endothelial dysfunction, coagulation, and lipid metabolism. Furthermore, elevated blood pressure and reduced heart rate variability were observed after exposure to traffic-related air pollution [17]. Diesel engine exhaust particles increase the level of interleukin-8, thereby triggering an inflammatory cascade. Increased levels of SO_2 elevate the level of fibrinogen. Ozone can induce direct oxidation in both pulmonary and systemic vessels, leading to inflammation and arterial constriction [6].

Particulate matter can have a dual toxic effect on the cardiovascular system. It can directly affect the circulation by soluble components of $PM_{2.5}$ passing through the respiratory epithelium into the systemic bloodstream. However, more commonly, the impact is indirect and involves the induction of pulmonary and systemic oxidative stress, leading to inflammation. Inflammation can serve as an initiator of a cascade of events, resulting in changes in blood rheology and prothrombotic effects (increased levels of fibrinogen, enhanced platelet aggregation), as well as alterations in the cardiac autonomic system leading to rhythm disturbances [6,7]. It is also a potential factor in inducing hypertension, mainly due to the imbalance of the autonomic nervous system, vasoconstriction, and endothelial dysfunction [18].

In a global study conducted in 2015 different cities worldwide, the short-term association between $PM_{2.5-10}$ and total, cardiovascular, and respiratory mortality was assessed. An increase of 10 μg/m^3 in $PM_{2.5-10}$ concentration was associated with a 0.51% increase in total mortality, 0.43% in cardiovascular mortality, and 0.41% in respiratory mortality, depending on the country and region [19]. It has been found that chemical components of $PM_{2.5}$ can be a leading cause of short-term cardiovascular effects in healthy adults in the context of traffic-related air pollution. Circulatory system biomarkers such as TNF-α (tumor necrosis factor), fibrinogen, PAI-1 (plasminogen activator inhibitor type 1), t-PA (tissue plasminogen activator), vWF (von Willebrand factor), and P-selectin were more likely to change in response to metal content in $PM_{2.5}$, and several transitional metals (Zn, Co, and Mn) as well as NO_3 – were found to have the strongest correlations with these biomarkers. Although the observed changes in biomarkers in study participants were small, they pose a minimal risk to healthy adults. However, adverse reactions in the cardiovascular system may likely occur in patients with pre-existing cardiovascular disorders, which may contribute to unfavorable health events [15,20]. $PM_{2.5}$ is associated with an increased incidence of arrhythmias. The nature of the arrhythmias depends on the source of pollution, although anthropogenic sources are primarily responsible [21]. A Chinese study showed that short-term exposure to a 10 μg/m^3 increase in both $PM_{2.5}$ and PM_{10} was associated with a respective 3.8% and 2.7% increase in the risk of atrial fibrillation [22]. Long-term exposure to $PM_{2.5}$ is linked to endothelial dysfunction, leading to chronic functional changes in blood vessels [23]. PM affects the development of vascular inflammation and, in the long term, has atherosclerotic effects [24]. Some studies have also shown that both leukocyte and red blood cell counts decrease with increasing $PM_{2.5}$ concentrations [16]. A Dutch study involving nearly 40,000 residents found that long-term exposure to UFP (ultrafine particles) is associated with an increased risk of cardiovascular diseases, heart attacks, and heart failure [25].

Exposure to air pollution in residential areas contributes to the occurrence of chronic coronary syndrome and arterial hypertension. It also appears to be a significant factor increasing the frequency of ventricular rhythm disorders, conduction disturbances, and ST segment depression episodes in Holter ECG monitoring [26]. The risk of ST-segment elevation myocardial infarction (STEMI) increases within a few hours of exposure to air pollution, with nitrogen dioxide (NO_2) having the strongest impact. However, no associations were observed between STEMI and sulfur dioxide (SO_2), ozone, or particulate matter (PM_{10}) [27].

A study conducted in 26 Chinese cities revealed that short-term exposure to air pollution was significantly associated with an increased risk of hospitalization due to congestive heart failure, particularly among patients with diabetes or arterial hypertension [28]. It also leads to impaired high-density lipoprotein (HDL) function and elevated levels of circulating atherogenic lipids (oxidized LDL), confirming the atherosclerotic nature of air pollution, which can ultimately lead to coronary artery disease [29].

An analysis of the short-term effects of air pollution in China indicated that increased concentrations of particulate matter, including $PM_{2.5}$ and PM_{10}, were significantly associated with an increased risk of hospitalization for stroke and myocardial infarction, with $PM_{2.5}$ showing a stronger adverse effect on these diseases compared to PM_{10}. Increased concentrations of gaseous pollutants such as sulfur dioxide (SO_2), nitrogen dioxide (NO_2), and ozone (O_3) were also significantly associated with an increased risk of hospitalization for stroke and myocardial infarction [30]. The risk of ischemic stroke rises following short- or long-term exposure to air pollution, with short-term exposure increasing the risk of intracerebral hemorrhage, while the effects of long-term exposure are less pronounced [31]. A study conducted in Germany found that long-term exposure, especially to benzene, O_3, NO, SO_2, and $PM_{2.5}$, is associated with increased mortality in hospitalized patients with ischemic stroke [32]. It is estimated that air pollution levels have been responsible for 14% of all stroke-related deaths in recent decades [31].

Both nitrogen oxides and carbon monoxide have a negative impact on the functioning of implantable cardioverter-defibrillators (ICDs) [6]. Studies conducted in Canada suggest significant associations between outdoor air pollution exposure and adverse cardiovascular measurements in patients with ICDs. This is associated with an increase in diastolic blood pressure after exertion in response to elevated levels of air quality index, $PM_{2.5}$, NO_2, and CO, as well as an increased heart rate after exertion in response to elevated $PM_{2.5}$ levels [33]. There is also an increased risk of ventricular arrhythmias in patients with implanted ICDs, suggesting an acute cardiac response to increased ambient air pollution [34]. The occurrence of venous thromboembolic disease, which includes deep vein thrombosis and pulmonary embolism, is highly likely [35].

Studies also suggest that maternal exposure to SO_2, NO_2, PM_{10}, and $PM_{2.5}$ during pregnancy is associated with a higher incidence of congenital heart defects in children, with the most vulnerable periods of exposure occurring primarily in the second and third trimesters of pregnancy [36].

CONCLUSION

Air pollution is a major health issue for Europeans, causing not only mortality but also morbidity, resulting in personal suffering and significant healthcare costs [3]. A significant portion of premature deaths in European cities can be prevented by reducing air pollution levels, especially below the WHO guidelines. This reduction could prevent 51,213 deaths annually due to exposure to $PM_{2.5}$ particulate matter and 900 deaths annually due to exposure to NO_2 [13].

We can largely prevent the health effects of polluted air. Reducing pollution at the source can have a rapid and significant impact. Within a few weeks, symptoms not only from the respiratory system (such as breathlessness, coughing) disappear, but there is also a significant decrease in the number of visits to clinics, hospitalizations, and cardiovascular-related illnesses and deaths [37]. The reduction of air pollution emissions can be pursued on three different levels: governmental, industrial, and individual [8]. Urban development, ongoing industrialization, global warming, and new knowledge about the harmful effects of air pollution are factors that increase the urgency of pollution control and emphasize the consequences of inaction at every level [37].

Reducing emissions from vehicles, considering their identified toxicity, is likely to improve the functioning of the cardiovascular system, and this battle should be a priority for air quality strategies [11]. Reducing air pollution can also be achieved through industrial modernization, alternative energy

sources, improving public transportation, city development programs to reduce transportation and increase pedestrian areas, and systematic health education [8]. Replacing fossil fuels with renewable energy sources would be an effective intervention to reduce mortality rates in Europe, potentially increasing the average lifespan by about 1.2 years [5].

Plant leaves act as biological filters, removing significant amounts of particles from the atmosphere in urban areas. Green infrastructure contributes to better air quality. Not only trees but also urban meadows can be a key strategy for air purification in polluted and densely populated cities. Various plant species provide abundant resources for reducing PM pollutants, such as European ash (Fraxinus excelsior), European linden (Tilia europaea), mountain pine (Pinus mugo), and English ivy (Hedera helix) [38].

Awareness of air pollution levels is key to taking individual actions. The risk of air pollution is greatest for patients with pre-existing chronic cardiovascular or pulmonary diseases, the elderly, and children [6]. By educating patients, doctors should encourage raising awareness of daily air quality, which can be found in weather reports, websites, or through email notifications or applications [8]. It is worthwhile to take steps such as staying indoors on days with high air pollution, using air purifiers indoors, and limiting outdoor physical activities near sources of air pollution [39]. Reducing exposure on days when air quality standards are exceeded can help mitigate the adverse effects on cardiovascular measurements in patients with ICDs [33]. The use of face masks for protection against air pollution may seem simple and intuitive, but maximizing effectiveness and minimizing potential harm depends on complex interactions among various factors, starting from assessing when and where to use a face mask, choosing the appropriate mask, and using it correctly [40]. Groups of people in socially disadvantaged situations and with lower education levels may be more vulnerable, and it is crucial to address their needs [8].

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