

Effect of short- and long-time exposure to ambient air pollution on blood pressure

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Abstract

It has been known for a number of years that air pollution significantly increases the morbidity and mortality of people exposed. More and more research and the last two meta-analyses also showed that even a short-time increase in air pollution can raise blood pressure and long-term air pollution leads to an increase in the incidence and prevalence of arterial hypertension. The elevation of blood pressure caused by air pollution may contribute to the increase in cardiovascular diseases observed in areas affected by air pollution. This increase in blood pressure caused by air pollution can be particularly dangerous for women, the elderly, obese people and those already burdened with cardiovascular diseases.

Key words: air pollution; hypertension

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In industrialized countries, the prevalence of hypertension increases. In the literature, it is postulated that the increase in the occurrence of hypertension is caused by the increasing body weight (especially in the middle-aged and elderly people), large intake of table salt, significant stress accompanying civilization today, more frequent premature births of which children are characterized by low birth weight and kidney hypoplasia [1]. In the last 15 years, attention was paid to yet another risk factor which is the growing pollution of the environment. This environmental pollution is especially high in our country. Current epidemiological data suggest that air pollution in Poland is the cause of nearly 50,000 deaths annually.

Primary pollution is directly emitted into atmosphere, principally during process of fossil fuel combustion, and comprises various gases and carbonaceous and non-carbonaceous particles. Secondary

pollution is formed within the atmosphere as a result of chemical reactions between primary pollution and natural atmospheric compounds and includes particles size from $< 0.1 \mu\text{m}$ to $10 \mu\text{m}$. These particles (PM, Particulate Matter) composed by both solid and liquid components are categorized from very small $\text{PM}_{1.0}$ ($< 0.1 \mu\text{m}$), through $\text{PM}_{2.5}$ ($< 2.5 \mu\text{m}$) to larger PM_{10} (between 2,5 and $10 \mu\text{m}$). Air pollutant also contain many gases like sulfur dioxide (SO_2), nitric oxide (NO), nitrogen dioxide (NO_2), carbon monoxide (CO) and ozone (O_3) [2]. The sources of outdoor PM vary depending upon specific location, particle sizes and commonly include traffic-related emissions, industrial processes, power generation (e.g. coal-fired plants), and wood-burning. Finally, especially in Poland, domestic stoves contribute many particles to the air pollution. Numerous epidemiological and clinical studies have clearly demonstrated that air pollution is responsible for the

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increased morbidity and mortality of people exposed to this environment [3]. Studies in the United States with over 500,000 people have shown that each increase in air pollution with particulates (PM_{2.5}) by 10 µg/m³ is associated with a 4% to 8% increase in mortality [4]. More than 3 billion people are exposed to high levels of these pollutants, causing them leading cause of morbidity and (very often, premature) mortality worldwide.

However, relatively recently it has been shown that this mortality is associated not only with respiratory diseases but also to a large extent with cardiovascular diseases. Epidemiological studies have shown that chronic air pollution exposure is associated with the degree of atherosclerosis and risk of cardiovascular events [5]. Acute exposure causes exacerbation of cardiorespiratory conditions, leading to an increase in hospital admissions and death. The effect of polluted air on the development of ischemic heart disease, heart failure and arrhythmias is discussed in more detail in the review by the authors Gluszek J. and Kosicka T. [6].

The mechanism of associations between air pollution and increase risk of morbidity and mortality are unclear, but recent controlled studies have demonstrated that air pollution generates reactive oxygen species, cause development of systemic inflammatory response and promotes endothelial dysfunction [7]. It is possible that air pollution, may contribute to cardiovascular disease through increasing blood pressure.

Initially, the studies focused on the influence of transient air pollution (and specially PM concentration) on blood pressure values. Later, an attempt was made to assess the association between long-term air pollution and the prevalence of arterial hypertension.

The Canada Health Measures Survey tested the association between cardiopulmonary parameters (blood pressure, spirometry and exercise capacity) and air pollutants. Each daily PM_{2.5} increase of 4.5 µg/m³ was significantly associated in 5011 individuals with elevations in both systolic and diastolic blood pressure by approximately 0.5 mm Hg [8]. The authors of this study also showed that increase by 12.6 ppb in NO₂ was associated with elevation of blood pressure (1 mm Hg).

Dvonch et al. assessed the effect of exposure to air pollution on arterial blood pressure in 347 people living in three Detroit districts [9]. Concentration of 10 PM_{2.5}/m³ caused an increase in blood pressure from 3.2 to 8.6 mm Hg, depending on the location of the subjects and degree of air pollution. These authors also noted that people younger than 55 years and not taking antihypertensive drugs more

often showed a significant increase in systolic pressure after inhalation of contaminated air. Huang and colleagues compared the short-term effects of air pollution in Michigan (9.1 + 1.8 PM_{2.5}) and Beijing (86.7 + 52 < 1 PM_{2.5}) on the arterial blood pressure [10]. The increased outdoor-ambient exposure (per 10 µg/m³) was associated with significant elevation in diastolic BP (0.16 mm Hg). In overweight adults significant increase in systolic (0.4 mm Hg) and diastolic (0.4 mm Hg) BP levels were observed.

The impact of the two-week exposure to air pollution inside the home on heart rate variability and arterial pressure was the subject of research by Liu et al. [11]. The crossover study covered 35 non-smokers of the elderly residents of Beijing. Portable air filtration units were randomly allocated to active filtration for two weeks and sham filtration for two weeks in the households. Blood pressure was measured using the ABPM method. Authors observed decreases of 34.8% in indoor PM_{2.5} and 35.3% in indoor black carbon concentrations during active filtration. Each 1 µg/m³ increase in indoor black carbon was associated with a significant increase of 2.4% in systolic blood pressure and each 10 µg/m³ increase in PM_{2.5} was associated with significant reduction of 1.34% in heart rate variability compared with active filtration.

One of the most polluted cities in the world is Beijing with the concentration of PM_{2.5} dust ranging from 86 to 140 µg/m³. In the study of Langrish, 15 healthy volunteers at the average age of 28 years walked the main streets of this city for a minimum of 2 hours [12]. Blood pressure was measured before and after exposure to contaminated air. At the end of the study, the mean systolic arterial pressure in the ABPM study was 121 mmHg. Then, the subjects were asked to wear masks in the daytime hours on the day before the pressure measurement and on the next day with the obligatory 2-hour walk on the same city streets as before and the mean systolic arterial pressure in the ABPM study was 114 mm Hg.

The impact of long-term exposure to air pollution in healthy young people was studied by Lenters et al. [13]. These authors have demonstrated the relationship between air pollution and blood pressure values; however, this did not translate into the thickness of the intima-media of the carotid artery and the speed of the pulse wave. The authors showed a 37% increase in augmentation index associated with inhalation of air contaminated with nitrogen dioxide.

An increase in blood pressure is particularly dangerous in the elderly and with concomitant chronic ischemic heart disease. Delfino et al. followed the values of blood pressure in such people during the varying intensity of contaminants of the inhaled

air [14]. At the same time physical activity of patients was measured using actiograph. The authors showed that the highest increase in blood pressure is caused by inhalation of air contaminated with organic carbon compounds (exhaust fumes of fossil fuels). Increased concentrations of these compounds by $5.2 \mu\text{g}/\text{m}^3$ lead to an increase in systolic blood pressure by an average of 8.2 mm Hg and diastolic pressure by 5.8 mm Hg. The relationship between increased pressure and air pollution was greater during physical exercise and in obese patients.

Zanobetti et al. investigated a correlation between arterial pressure values and the degree of air pollution with $\text{PM}_{2.5}$ at rest and during controlled physical exercise in rehabilitated patients after a cardiac incident [15]. At rest, the systolic pressure was higher by 2.8 mm Hg and the diastolic blood pressure by 2.7 mm Hg on days with high air pollution than on control days. In car traffic, diesel engines pollute the air the most. Cosselman and colleagues decided to check how the exhaust of this car can affect blood pressure [16]. The study of these authors was conducted in a group of 45 non-smoking, healthy volunteers aged from 18 to 49. Each participant of this test for 2 hours inhaled through a special mask exhaust engine diesel containing up to $200 \mu\text{g}/\text{m}^3$ $\text{PM}_{2.5}$ and after 2 weeks through the same mask clean air. The order of breathing in exhaust fumes or clean air was randomized. The arterial pressure was measured with an automatic oscillometric apparatus at 5, 30, 60 minutes after the time of mask placement and then after 3, 5, 7 and 24 hours. Breathing in diesel motor fumes caused a statistically significant increase in systolic arterial pressure by 3.8 mm Hg after 30 minutes and by 5.1 mm Hg after 60 minutes of the experiment. During 24 hours after the end of the experiment, systolic blood pressure increased by 2 mm Hg. This precise random test showed that the high concentration of exhaust gases causes the largest increase in systolic pressure after several minutes of inhalation of exhaust gases, but the increase by 2 mm is maintained for at least 24 hours. Air pollution in large US cities reaches $130 \mu\text{g}/\text{m}^3$ and lasts much longer than 2 hours.

Last year, Tsai tried to find a relationship between the degree of air pollution with $\text{PM}_{2.5}$ dust and the frequency of admission to hospitals in Kaohsiung in Taiwan due to hypertension in the period from 2009 to 2013 [17]. Such dependence was not demonstrated in the summer months, while on days with the temperature below 25 degrees Celsius in days with a high degree of air pollution the number of admissions to the hospital due to hypertension increased by 12%. This year, Song et al. based on observations

of 650,000 hospital records showed a weak but statistically significant relationship between concentration of $\text{PM}_{2.5}$, PM_{10} , NO_2 , O_3 , CO and the risk of admission of patients with hypertension to hospitals [18]. This study suggests that patients with hypertension had an increased risk of hospital admission when exposed to air pollution; moreover, there were non-significant differences in the associations between air pollution and sex or age group.

Air pollution can cause not only a temporary increase in blood pressure, but in the long-term can also lead to the occurrence of arterial hypertension.

Chan and colleagues evaluated the long-term impact of breathing contaminated air by 433 women at the age of 35 years [19]. In this study, it was shown that long-term breathing in air contaminated with dust with a diameter smaller than $2.5 \mu\text{m}$ is correlated with a small but statistically significant increase in blood pressure. An increase of $10 \mu\text{g}/\text{m}^3$ in this dust leads to an increase in systolic blood pressure by 1.4 mm Hg, without significant changes in diastolic pressure. The increase in the concentration of nitrogen dioxide in the inhaled air caused a slight but statistically significant increase in the pulse pressure.

A large prospective European ESCAPE study evaluated the relationship between air pollution with PM, nitrogen oxides and noise and traffic noise and the incidence of hypertension [20]. The study involved over 40,000 participants, and within 5–8 years 6,200 people (15.1%) experienced hypertension. The authors showed a statistically significant relationship ($R = 1.22$) between air pollution and incidence of hypertension. Traffic noise caused only slight influence on the appearance. These estimates decreased slightly upon adjustment for road traffic noise.

As part of the Aging Project, Honda et al. evaluated the impact of air pollution on values of blood pressure in 4,121 Americans over 57 years of age [21]. The researchers observed an increase in the incidence of hypertension by 24% among people who have been exposed to inhaled air pollution for a long time. The increase in inhalation of $\text{PM}_{2.5}$ dusts for one year leads to an increase in blood pressure. These dependencies are also correlated with the degree of air pollution.

In a large Chinese epidemiological study Xi, Wang and colleagues assessed the relationship between long-lasting impact air pollution on the incidence of hypertension in the population of people of reproductive age (20–49 years). According to these authors, 2.3% of the hypertension cases could be attributed to $\text{PM}_{2.5}$ exposures in reproductive-age adult populations [22].

Air pollution, besides leading to increased blood pressure, increases the risk of hypertriglyceridemia. Shamy et al. [23] observed in 2025 people the relationship between the increase in dusts ($PM_{2.5}$) and the incidence of metabolic syndrome with hypertriglyceridemia and elevated blood pressure.

The results of one of the largest studies were published last year by the Chinese [24]. They observed the 3-year influence of air pollution on more than 16,000 people, residents of 33 towns. In this study, statistically significant associations between concentrations of PM_{10} , SO_2 and O_3 and the values of blood pressure were demonstrated. All these interactions were stronger among overweight adults, in women, older people and participants living in areas with lower income levels.

The vast majority of research concerned the relationship between polluted air and blood pressure in out-door conditions. Only a few authors described the relationship between the increase in blood pressure and air pollution in the biomass-fired farmers' houses. Polluted air of big cities can also penetrate into apartments. Last year, Rumchev and colleagues showed an increase in blood pressure values of 12/10 mm Hg in people exposed to inhalation of contaminated air due to indoor air pollution in the large metropolis of Perth, Australia [25].

However, not all studies have shown a direct relationship between air pollution and the size of the arterial pressure increase [26]. Researchers in Serbia evaluated the effect of short-term air pollution on systolic and diastolic blood pressure in 98 healthy non-smoking female volunteers. The authors of this study did not show any statistically significant impact of black smoke and sulfur dioxide (SO_2) on the heart rate and blood pressure values [2].

The influence of air pollution on blood pressure has already been the subject of several meta-analyses. In all of these studies, a significant but slight increase in blood pressure was demonstrated. In the meta-analysis of Liang et al., each increase in $PM_{2.5}$ concentration by $10 \mu\text{g}/\text{m}^3$ caused an increase in systolic pressure of 1.39 mm Hg and diastolic pressure by 0.87 mm Hg. A longer exposure to polluted air caused a greater increase in blood pressure [28].

Literature review by Giorgini and al. from 2016 demonstrated that particulate pollution causes significant increase in blood pressure in relation to both short- and long-term exposures [2]. Moreover, epidemiological studies suggest a positive association between residence within regions with higher levels of ambient PM and an increased incidence in and prevalence of overt hypertension [2, 28–30].

Meta-analysis of Yang et al. from 2018 showed a significant association of long-term exposures to $PM_{2.5}$ with hypertension [30]. The authors of this meta-analysis note, however, that in the vast majority of work assessing the impact of air pollution on arterial pressure, traditional pressure measurement was used. However, Yang and al. refer to 5 works using the ABPM method to assess the relationship between exposure to air pollution and the values of blood pressure. In most of these works, a small but statistically significant increase in blood pressure was found. According to Yang et al., the ambulatory BP monitoring may have provided a more accurate evaluation within a very short time period of exposure to air pollution.

The risk of hypertension is high in pregnant women. Therefore, air pollution could aggravate and/or trigger adverse hemodynamic response in this vulnerable group of subjects. More than 10 studies linking air pollution with blood pressure among pregnant women have been recently published [2]. The relationship between $PM_{2.5}$ and higher systolic blood pressure was found in all trimester of pregnancy [2]. According to the meta-analysis of Pedersen et al., an increase in $PM_{2.5}$ by $5 \mu\text{g}/\text{m}^3$ leads to a significant increase in gestational hypertension (OR 1.47) and preeclampsia (OR 1.3) [31]. A smaller, but also statistically significant, increase in blood pressure in pregnancy is caused by air contamination with gases like NO_2 and O_3 [31].

So far, few papers have been published regarding the impact of air pollution on the value of arterial blood pressure in children. In two schools in Pakistan, the average arterial pressure of children was compared. In a school exposed to high air pollution, the average pressure was 115.9/70.9 mm Hg, and in a school with low air pollution — 108.2/66.4 mm Hg [32]. The PIAMA study showed a small but statistically significant relationship between PM_{10} and iron and diastolic BP values in children aged 12 years. According to the authors of the study, possible association with iron may indicate the health relevance of non-exhaust emission of traffic [33].

The pathomechanism of the increase in blood pressure following exposure to air pollution is not completely clear. It is suspected that air pollution may cause autonomic nervous system imbalance favoring sympathetic over parasympathetic tone. Air pollution breathing could mediate the generation and release of systemic oxidative stress and inflammation. Inhalation of polluted air causes a decrease in heart rate variability, a manifestation of an imbalance of autonomic nervous system [34, 35]. It has been demonstrated that 2-hour exposure on

high concentration of PM was associated with rapid elevation in blood pressure and heart rate probably due to autonomic imbalance. In experimental studies, a 6-month exposure to contaminated air in mice resulted in an increase in blood pressure and elevation of urinary norepinephrine excretion [36]. Several reports have shown association of air pollution exposure with change of concentration acute-phase proteins such C-reactive protein (CRP), interleukins 1b and 6 (IL-1b, IL-6), tumor necrosis factor- α (TNF- α), fibrinogen [37]. These pulmonary and systemic responses may activate vascular oxidative stress and stimulate superoxide generation. Chronic inflammation is characteristic of hypertension. Some authors suggest damage to the endothelial function following exposure to air pollution. Langrish et al. suggest that the unfavorable effect of exposure to air pollution is caused in large part by the impairment of nitric oxide bioavailability [38]. Other authors postulate increased secretion of endothelin in people exposed to the action of air pollution. Pediatricians from Mexico studied serum endothelin concentration in blood of 81 children with mean age of 7.9 ± 1.3 years (from 6 to 13 years). Forty children lived in the southern district of Mexico in which high concentrations of ozone are found, 19 children lived in the northern districts of Mexico in which air pollution with dust of various sizes is particularly high [39]. Still another 22 children lived in Polotilan, characterized by the lack of air pollution. In the blood serum of children living in Mexico, there was statistically significant (relative to control children) increase in endothelin serum concentration. The increase in endothelin concentration correlated ($r = 0.31$, $p = 0.012$) with the number of hours spent by children outside and correlated with the accumulative amount of dust with a diameter below $2.5 \mu\text{m}$ ($r = 0.23$, $p = 0.03$). The authors of this study also showed a significant correlation between the number of hours spent outside by children and the pressure in the pulmonary artery [39]. Previously, in experimental studies in rats, a rapid increase in the concentration of endothelin in the blood during inhalation of ozone and other air pollutants has also been demonstrated [40]. Other studies, however, have found no change in endothelin system related to air pollution exposure or no alternation in the functional outcomes related to endothelin-1-induced vasoconstriction [41]. The role of endothelin in the course of air pollution is discussed in detail in the work of Finch and Conklin [42].

The consequences of ubiquity air pollution are underestimated. Even small effect on raising blood pres-

sure and/or prevalence of hypertension would have enormous global public health implications and can be the major risk factor for mortality and morbidity worldwide. We need energetic actions to reduce air pollution in our country.

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