

The impact of particulate matter (PM) and nitric oxides (NO_x) on human health and an analysis of selected sources accounting for their emission in Poland

Jakub Krzeszowiak^{1 (a, b, c)}, Damian Stefanow^{2 (b, c)}, Krystyna Pawlas^{1 (a, d)}

¹ Department of Hygiene, Wrocław Medical University – Poland
Head of the Faculty and Department Hygiene - prof. K. Pawlas, PhD

² Faculty of Mechanical Engineering, Wrocław University of Technology – Poland
Head of the Department of Off-Road Machines and Vehicles Engineering - prof. P. Dudziński, Eng., PhD

(a) conception and design

(b) literature review

(c) drafting the article

(d) revising the text



mgr Jakub Krzeszowiak



prof. Krystyna Pawlas

ABSTRACT

Introduction and objective: This paper is concerned with the harmful impact of nitric oxides (NO_x) and particulate matter (PM) on humans. The objective was to determine which source of emission is the most urgent in terms of its reduction. Abbreviated description of the state of knowledge: In published epidemiological studies multiple notifications indicating the harmful impact of particulate matter on human health can be found. The harmful impact is underscored by the increase in the number of hospitalisations owing to diseases of respiratory and cardio-vascular systems, as well as by the rise in general fatality rate. The analysis of the PM impact on the human body is prompted by the fact that its detrimental effects

are not clearly defined. Additionally, nitric oxides contribute to the increased number of exacerbations of respiratory disease and are a factor increasing susceptibility to development of local inflammation.

Conclusions: The following study is meant to show that the air pollution which derives from vehicles (NO_x and PM) has a significant impact on human health. This applies particularly to residents of cities and big towns. This issue has gained special importance in Poland. According to the data from the Central Statistical Office, the increasing number of vehicles in use and their age lead to increased emission of the pollutants considered.

Key words: particulate matter, nitric oxides, air pollution, human health

INTRODUCTION

Polish cities are ranked among those with the highest level of air pollution in Europe [1]. This concerns particularly Cracow and cities in the Silesian agglomeration. Other big municipalities also wrestle

with the problem of air pollution caused, inter alia, by heavy traffic, unavoidable traffic jams, and low speed of driving in the city. Consideration must be given to the fact that dust can be created by industrial and business activity as well as by natural sources. Both in industrialized areas and in places without

well-developed industry (e.g. Wrocław), road transport is an important source of air pollution. Since at least 2001, road transport has been the largest emitter of nitrogen oxides (NOx). Its participation in general emission of NOx has reached 33% [2]. Road transport is also responsible for significant emission of particulate matter (PM) [2]. This is a result of increasing number of vehicles with a diesel engine. Therefore in cities characterized by significant traffic congestion, nitrogen oxide and particulate matter limits are often exceeded. That is due to the fact that exhaust gases from cars standing in “street canyons” rise over high-density housing at low rate. Furthermore, as will be shown further, most of the cars are over fifteen years old and emit sizeable amounts of NOx and PM.

Epidemiologic researches which show that an excessive increase in air pollution concentration impairs people’s health can be found in literature. This phenomenon is caused by affection mechanism of particulate matter and nitrogen on people. It also has to be emphasized that the impact of PM and NOx is synergistic and the most exposed people are children, which is attributable to their physiology.

MECHANISMS OF PARTICULATE MATTER TOXICITY

Dust is one of benchmarks for earth’s atmosphere pollution. In cities, we can observe increasing concentration of dust in the air, which may redound to the occurrence of negative health effects. Harmfulness of particulate matter depends on its amount, precisely on its diameter and composition. Atmospheric dust, otherwise named atmospheric aerosol, may be divided into two groups: with diameter of over 10 μm and below. Particles with diameter below 10 μm have a more negative impact on health, and they can be further divided into two fractions: PM 2,5 viz. particles <2,5 μm and PM 10 – particles <10 μm . These are called fine particles and have aerodynamic diameter. In literature, fraction PM 0.1 particle <100 nm can also be found and is commonly known as ultrafine particles (UFP) or nanoparticles (NP). Additionally, diesel exhaust particles (DEP) are specified. This is because diesel cars are the main emitter of particulate matter in road transport, as shown in Figure 1. DEP consists of both PM 10 and 2,5. It includes also UFP and bigger particles, called soot [3], as well as aromatic hydrocarbons. As will be shown further in the article, implemented EURO

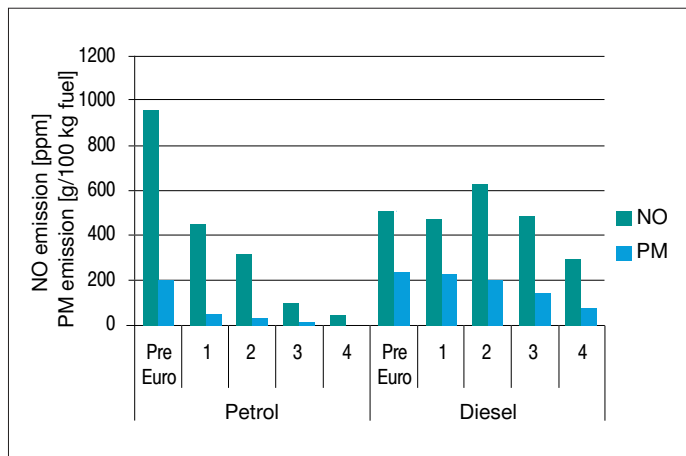


Fig. 1. Emissions of NO and PM relative to EURO standard and fuel type [48]

standards help reduce the amount of PM emission from diesel engines.

Atmospheric dust irrespective of the fraction negatively affects human health. “Large” particles 10 μm reside in the upper section of the respiratory system. They are captured and moved by the mucociliary system towards the nasal cavity. Excessive concentration of these particles can effect impairment of the ciliary epithelium, which is particularly visible in the case of smokers [4, 5]. Accordingly, aside from the harmfulness of smoking, smokers are exceptionally strongly affected by air pollution. This is the result of the fact that in the case of functional mucociliary system, 80–90% of particulate matter is removed from the respiratory tract [4]. In patients with dysfunctional ciliary epithelium, pollution penetrates much easier into the deeper sections of the respiratory system. It can also directly affect their mucous membrane for a longer time. The fine particles of fraction under 10 μm go easily into bronchial tubes and bronchioles up to alveoli, where they are subject to the phagocytosis process, mainly by macrophages. After absorbing particles, alveolar macrophages move into bronchioles lined with ciliary epithelium, which moves the engulfed particles in the direction of the pharyngo-nasal cavity [4]. However, as it was mentioned, the efficiency of this system can be constrained. The detrimental effect of PM10 was described for the first time by C. Arden Pope III. His main conclusion was that exposure to PM10 leads to higher prevalence rate and intensity of respiratory system disease [6]. Since that publication, much research has been published where authors point out the key impact of PM10 and PM2,5 on the number and course of respiratory and circulatory systems diseases [7].

Many mechanisms were suggested to explain how suspended fine particles impact health. It is stated that particulate matter of fraction $10\mu\text{m}$ leads to development of inflammation within the respiratory tract, affects the autonomic system, and contributes to increased generation of free radicals [8, 9]. Researchers' greatest attention is focused on the problem of intensified generation of free radicals where particles with active chemical groups, especially metal ions, affect the surface [8]. Metal ions in Fenton and Haber Weiss reaction cause increased generation of hydroxyl radical $\cdot\text{OH}$ [10]. It was proved that ions of transition metals reacting with PM may have pro-inflammatory effect as well as cause mentioned generation of reactive oxygen species. Pro-inflammatory action, in all probability initiated by ROS (reactive oxygen species), can be manifested by releasing pro-inflammatory cytokines IL-6, IL-8 (interleukins), TNF-alpha (tumor necrosis factor-alpha) from pulmonary macrophages [8, 11].

According to the above-named mechanism, protracted exposure to particulate matter can result in the development of persistent inflammation within the respiratory tract. That was verified in research on animals with the use of DEP. After inhalation, animals increased mucus secretion in the respiratory tract and inflammatory infiltration occurred. Also nitric oxide synthase rose and hyper responsiveness of bronchial tubes appeared [12–13]. All those elements create a condition closely related to asthma. It is not clear whether the exposure to PM can lead to the development of asthma in humans. However, it has been proved that the exposure to suspended particulate matter can result in more frequent occurrence of asthma. In epidemiological surveys, a correlation has been confirmed between its increased concentration in the air and an increase in the number of hospitalizations caused by asthma attack [7, 13]. This is in all probability due to the fact that particulate matter brings about intensification of already existing inflammation within the respiratory tract, acting as a triggering factor.

Nanoparticles prevalent in various branches of industry have increasing contribution in the air pollution. Their huge source is road transportation; they are a component of exhaust gases, but also come from abrasion of tires and braking pads. Nanoparticles or ultra-fine particles (UFP) with size $<100\text{ nm}$ after getting with air into the respiratory tract, act on cells of pulmonary alveoli. With high concentration of dust and its microscopic size, deep penetration is permitted, leading to their distributed influence; this constrains the macrophage chemotaxis by providing a signal for phagocytosis. Their size means also poorer accessibility for macrophages

[8, 14]. Experimental animal studies indicate that the basic mechanism of negative effect of nanoparticles in vivo is an extensive local production of superoxide anion radical O_2^- and also hydroxyl radical $\cdot\text{OH}$ [8, 15]. Nevertheless, as for now, no data is available on the impact of nanoparticles on human health; available data come from animal studies only.

There are also a lot of studies into the adverse impact of suspended dust on the cardiovascular system (CV). There are no unequivocal reports regarding which fraction of dust is more harmful in that respect. In the epidemiological studies among a population of people exposed to PM, an increased number of hospitalizations caused by arrhythmias (ventricular arrhythmias, atrial fibrillation, transient ischaemic attacks – the stroke, the myocardial infarction) and hypertension was found [7].

Three potential mechanisms can be found in the literature. They are probably underlain by the impact of PM on CV. First of them involves PM impact on the autonomic system via the nervous system in response to inhalation of polluted air. The impact on the autonomic system particularly affects the stimulation of the sympathetic nervous system and the attenuation of impulsation from the sympathetic nervous system with all implications – vasoconstriction and cardiac disorders. The second mechanism includes direct release of pro-oxidative and pro-inflammatory mediators from the respiratory tract into circulatory system. After inhaling PM, higher concentration of IL-6, TNF-alpha, CRP and H_2O_2 (hydrogen peroxide) have been experienced. These factors can activate a chronic inflammatory condition, especially within blood vessels, that corroborates the thesis that arteriosclerosis occurs more often in people who are chronically exposed to particulate matter. Injury of blood vessels endothelium associated with an increased sympathetic nervous system activity (adrenaline) causes the imbalance between the shrinking factor (endothelin 1) and the vasodilating factor (nitrogen oxide), with prevalence of the former, which additionally contributes to the increase of resistance blood vessels. The third mechanism relates to nanoparticles' impact on CV. This pertains particularly to the fact that they probably initiate topical oxidative stress. The oxidative stress can lead to a lability of the atherosclerotic plaque and to a release of embolic material – this statement is a very contentious issue [9].

All the above mechanisms have coherent and causal connection, accordant with the pathophysiology of diseases such as hypertension, coronary attack or brain stroke. They also complement one another, creating a common chain, which is based on oxidative stress.

EXOGENOUS AND ENDOGENOUS NITRIC OXIDES – AFFECTION MECHANISM

In atmospheric air, a whole spectrum of nitric oxides known as NO_x is present. Amongst them, the most important and most often found in the highest concentrations are nitrogen monoxide and nitrogen dioxide, which are at the same time free radicals. The NO_x designation has also practical character, because in the environmental conditions there is no possibility of determination of the impact of just one among all nitrogen oxides (and also among the other polluting substances). That is caused by high reactivity of these chemical compounds. The NO_x resources in urban agglomerations are combustion processes, taking place in extremely high temperatures characteristic, among others, of internal-combustion engines. An additional source of NO_x for human health can also be tobacco smoke and gas cookers. Nitrogen oxides are absorbed from human respiratory tract in about 80–90%. After absorbing into the blood, the methemoglobin MetHb is created in the presence of oxygen and in the absence of NOHb [4]. High concentration of MetHb is akin to nitrogen oxides poisoning; however, this takes place only at high concentration of NO_x, typical of occupational exposure. In urban areas, people are subjected to persistent exposure to low concentrations of NO_x, with distinctive rise in these concentrations at peak time [16]; hence, it is difficult to explicitly define the specific mechanism of their toxic action. Now, it is necessary to underline that nitrogen monoxide occurs naturally in human body, performing important physiological function, especially acting as vasodilator as well as signalling molecule. It is formed in oxidation reaction of L-arginine with the participation of the specific synthase NOS (nitric oxide synthase) as well as co-factor NADPH. Two main nitric oxide synthases can be marked out: a constitutive form (cNOS) and an inducible form (iNOS). The difference between cNOS and iNOS is visible in the time of their activity. In physiological conditions, there is a few second process of releasing nitric oxide that is characteristic of cNOS activity. By contrast, prolonged release of nitric oxide, lasting several hours and leading in the end to a significantly larger amount of synthase (1000×), is characteristic of iNOS activity. Increasing the expression of inductive nitric oxide synthase form was shown in the inflammatory infiltration cells, i.e. in macrophages, eosinophiles, neutrophils, and mast cells [17].

One of the causes of inflammatory infiltration can be exposure to atmospheric pollutions. The inflammatory infiltrations and escalated production

of endogenous nitric oxide associated with them constitute human defence mechanism against various pathogens. This is caused by nitric oxide characteristics, which in low concentration shows antioxidant properties [18]. Increased production of NO can lead to an increment of its concentration in expired air (FeNO), the distinctive feature of asthma [19].

The affection mechanisms of nitric oxide on system cells have been divided into two groups: direct and indirect effects. It is worth mentioning that NO half-life in biological systems, independent of whether it originates from breathing air or overly increased endogenous production, is 2–3 seconds and it reacts easily with O₂. This reaction leads to the generation of NO₂ that undergoes further chemical reactions in the water environment [17].

Direct effects include suppression of the constitutional form of nitric oxide synthase activity (cNOS); however, iNOS is less sensitive to nitric oxide inhibition [20]. Therefore, the endogenous homeostasis of nitric oxide synthase is disturbed. Nitric oxide inhibits the activity of cytochrome oxidase, forming part of complex IV in the respiratory chain, and with its higher concentration it inhibits also complex I and II, affecting the cellular respiration process. The inactivation of the above processes leads to the increased generation of reactive oxygen species (ROS) due to incomplete four-electron reduction of oxygen [10]. Nitric oxide, which is a free radical, reacts with proteins containing iron-sulphur centres, ions of transition metals and the heme groups, thereby inhibiting the activity of cytochrome P-450 or catalase [20], which is an antioxidant enzyme.

The negative indirect effects take place particularly at higher concentrations of NO. The main indirect effects of nitric oxide include nitrosation, the by-products of which (nitrosamines) cripple DNA, leading to the progress of the carcinogenesis process, and are responsible for chronic inflammation [10].

Another very significant indirect effect on human body is NO z [•]O₂⁻ reaction (superoxide radical anion), producing superoxide nitrate (ONOO⁻) [21]. Superoxide nitrate has oxidizing properties, it initiates lipid peroxidation, leading to damage of cells and tissues. Nitric oxide reacting with superoxide radical anion competes with superoxide dismutase (SOD), which results in increased superoxide nitrate production in the focal points of inflammation and supports the inflammatory process. The process of tissue damage by reactive nitrogen species is not limited to the activity of superoxide nitrate. High reactivity of nitrogen dioxide leads to the formation of superoxide radicals ROO[•] as well as alkoxy radicals RO[•]. As a result of this imbalance, a nitrate anion

NO_2^- and nitrate anion NO_3^- (also participating in the lipid peroxidation process) are generated [10].

The impact of all above reactive nitrogen species with the simultaneous increased generation of reactive oxygen species within the respiratory tract and lung parenchyma leads to the augmentation of pulmonary permeability, increased mucus secretion, damaged and fatty bronchial epithelium, and the damage of surfactant properties and type II pneumocytes, triggering and intensifying the inflammatory process [19]. Mild inflammation of tracheal mucosa is observed in chronic exposure. An irritated conjunctiva and ulceration of the nasal cavity can also be expected [4].

Epidemiological studies indicate a positive correlation between the increased concentration of nitric oxides in the atmospheric air and admissions to hospitals because of asthma as well as COPD (Chronic Obstructive Pulmonary Disease). Researchers' attention is focused also on the rising level of expired nitric oxide, particularly in children exposed to air condition, as a factor indicating the development of inflammation, which can later develop into bronchial hyperreactivity. It should not be forgotten that the increased concentration of nitric oxide in the atmospheric air combined with sun radiation results in the rise in ozone levels, which is secondary air pollution (it is not generated by combustion engines). Ozone as a reactive oxygen species oxidises other substances, both animated and unanimated. In humans it brings irritation of eye mucosa and causes lacrimation. After invading the respiratory tract, it leads to cell membrane damage, acting irritatingly [4, 22].

The presented literature review has shown that the influence of PM and NOx on human health is negative. Exposure to those factors results in various health outcomes, which the authors have listed in Table I, where the most important cardiovascular and respiratory diseases connected with long term exposure to air pollution are enumerated.

EXPOSURE TO PM AND NOX IN TERMS OF DIFFERENT SOURCES IN POLAND

The negative effect of PM and NOx is indisputable; therefore, reduction of the emissions of these pollutants should become a priority for the government and the society. However, to make any actions effective, it has to be estimated which pollution sources are the biggest threat to citizens. For that reason, an analysis of different sources of these pollutants was carried out. A brief description of the results is presented below.

It is commonly known that higher concentration of pollutants is directly connected with the severity of health outcomes, especially in high sensitivity populations. For this reason, this section focuses on the quantitative analysis of air pollution emissions. Available data about emissions divide them into 11 groups, from which the highest amounts are produced definitely by: combustion processes in energy production and transformation sector, combustion processes in municipal and household sector, combustion processes in industrial sector, production processes and road transportation [2, 23]. All other sources contribute to about 15% of total emissions and are not considered in this work. From the major contributors, sources of highest emissions are not taken into account here, as their effect on the exposure level is not so direct as that of low emissions, so the energy production sector is not analysed. In the analysis, industrial sector is combined with production processes, as they usually appear together.

Graph in Figure 2 presents yearly emissions of PM and NOx in Poland from key sectors. It can be seen that emissions from the industrial and production sector are decreasing (average annual reduction ca. 1% PM and 9% NOx). This trend is promising, therefore the authors assessed this source as of secondary importance. Either way, reduction of PM's should proceed at a higher rate. The most important sources

Table I. Most important cardiovascular and respiratory diseases connected with long term exposure to air pollution

	PM	NOx
Respiratory diseases	<ul style="list-style-type: none"> • Asthma • COPD 	<ul style="list-style-type: none"> • Asthma • Irritation of the nose and throat
Cardiovascular diseases	<ul style="list-style-type: none"> • Hypertension • Atherosclerosis progression • Acute coronary syndrome • Ischemic stroke • cardiac arrhythmias 	<ul style="list-style-type: none"> • Formation of methemoglobin

in terms of exposure to considered pollutants, which are located nearest to places of peoples' residence, are household and mobile sources. This is justified by the fact that people spend most of the time in or in the neighbourhood of houses and cars. What is more, most of residential buildings and walkways are located in proximity to roads. It is important because NOx concentration within 50 m of roads can be even 100% higher than measured at nearby area-wide monitors [24]. The same applies to PM. Additionally, a very important factor that influences total PM exposure is in-vehicle PM 2.5 exposure. It can amount to as much as half of the total exposure for some people, depending on factors like employment status and the time spent in the vehicle [25].

An analysis of the emissions from household and mobile sources in 2005–2012 in Poland shows that PM emissions from mobile sources are increasing at a rate of about 5% per year, while from household sources at about 2% per year. Even though PM emissions from mobile sources are lower, they are growing 2.5 times faster. NOx emissions from both sources are growing at a similar percent rate. However, annual emissions from transportation are 3 times higher than from household sources (annual increase is 4 times higher than from households and much higher than from any other source). Average annual increase equals to almost 10% of annual emissions from households. It has to be mentioned that apart from the fact that NOx are reactive forms that damage cell membrane, they also transform into nitrates in the atmosphere and increase the overall amount of PM 2.5 [26]. This phenomenon is called secondary particle generation.

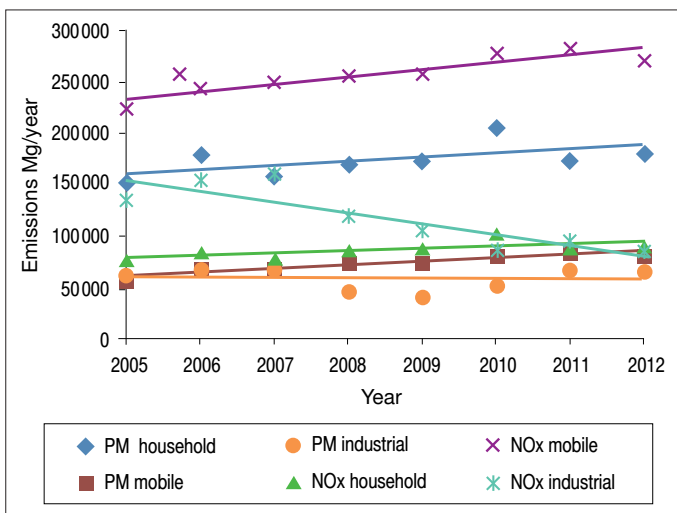


Fig. 2. Annual emissions of NOx and PM from mobile and household sources in the years 2005–2012 in Poland with trend lines predicting progress of the situation [2, 40, 41]

The above arguments lead to the conclusion that mobile sources of PM and NOx emissions should become the focal point of actions taken to improve air quality, especially in urban areas. In order to establish why the situation in Poland is so bad, the structure of the automobile market in Poland was analyzed. What follows is an analysis of probable reasons for this situation.

FACTORS INFLUENCING PM AND NOx EMISSIONS FROM MOBILE SOURCES IN POLAND

The main factors influencing emissions from vehicles are: the number of vehicles in exploitation, the average distance of travel by each vehicle and the standards which they meet. According to the data from the Central Statistical Office, the number of vehicles registered in Poland grows by over 1 million per year [27, 28]. In the years 2005–2010, the number of vehicles per 100 citizens increased from 403 to 576, while the number of passengers transported by means of public road transport decreased by 350 million (over 7%); the total number of registered vehicles increased by 37%. At the same time, the average annual distance covered by drivers decreased by 3721 km, which is about ¼ [29]. It has to be noticed that Poles do not travel less by private cars. The reduction of the average distance covered is a result of the fact that people who did not have and did not need a car before now have it and use it rarely. The overall effect was increased total fuel consumption from 10,901,000 tons in 2005 to 14,800,000 in 2010 (37% difference).

The authors recognize this phenomenon as the direct reason for the increase in emissions from vehicles. It seems that the most effective solution that could significantly help fighting pollutants is reduction of the number of vehicles in use on Polish roads. However, there are other methods of fighting emissions that do not affect directly vehicle owners.

One of the most important ways to reduce emissions, commonly used in the world, is the implementation of exhaust gas emission standards. The effect of this implementation is reduction of pollutants in vehicles' exhaust gases, which is well documented in [30]. The authors of the paper mentioned above have proved that emission of nitric oxide (NO) and particulate matter (PM) is growing according to the age of the car. It can thus be inferred that the implementation of the European EURO emission

standards contributes to their reduction. According to Figure 2, gasoline powered engines which satisfy the EURO 4 emission standard emit on average 19 times less NO than those produced before the introduction of the regulation. Similarly, emission reduction is visible when we focus on diesel engine, thence vehicles which satisfy the EURO 4 standards emit 4 times less PM than those manufactured before the regulation. Except the vehicles normalised to EURO 2 standards, NO reduction is also perceivable. However, analysing above data and EURO standards, it is noticeable that the reduction of PM emission in petrol engine declined over the years, despite the fact that the standards did not limit their emissions. It can be seen, therefore, that the reduction of PM emissions was most likely related to the improvement in engine operating parameters. In order to improve engine parameters, design and operational parameters of engines are modified, such as richness of the fuel mixture, setting valve timing, engine temperature and cooling system, ignition timing, pressure and temperature of the fuel, and temperature and volume of intake air [31]. Also the most high-tech exhaust gas after treatment methods are used, including Exhaust Gas Recirculation, Diesel Particulate Filter or Selective Catalytic Reduction, which lead to reduction of PM, NOx and other pollutants.

In the article [31] it was assumed that vehicles manufactured after the introduction of European emission standards meet these requirements, and thus demonstrate statistically significant correlation between the category of EURO standards and emission of pollutants.

Figure 3 presents the number of cars in different age groups registered in Poland. Important information that comes from the chart below is that Poles drive older cars every year. In the case of vehicles aged up to 2 years, the increase is negligible. Regarding cars 3–5 years old, a slight increase is noticeable, while in the case of cars aged 6–10 years, the drop in the number of registered vehicles is measurable. The situation changes when we take a look at the number of older cars, 11 to 31 years old, because their number is growing, particularly in the range of 11–20 years. Comparing statistical data from the Central Statistical Office, which are shown in Figure 3, with those presented in Table 1, it is possible to state that only several percent of registered vehicles in Poland comply with the require-

ments of EURO 5 standards. About half of the cars satisfy EURO 2, 3 and 4 standards and the rest – vehicles produced before 1995 – satisfy EURO 1 standards or none (data from 2011).

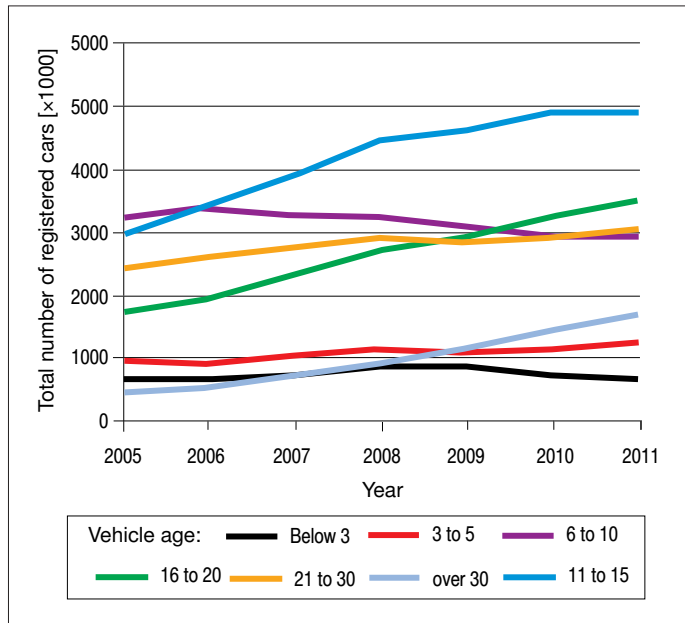


Fig. 3. The age structure of registered vehicles in Poland in 2005–2011 [45, 46]

The most important implication of the above data is that Poles use cars more often, but they do not change them to newer models. Therefore, emissions from mobile sources in Poland seem to be unstoppable. Chart in Figure 4 shows an increase in NOx and PM in the years 2005–2010. It provides some insight into the situation in Poland, which will lead inevitably to increased amount of complications caused by emission of those compounds.

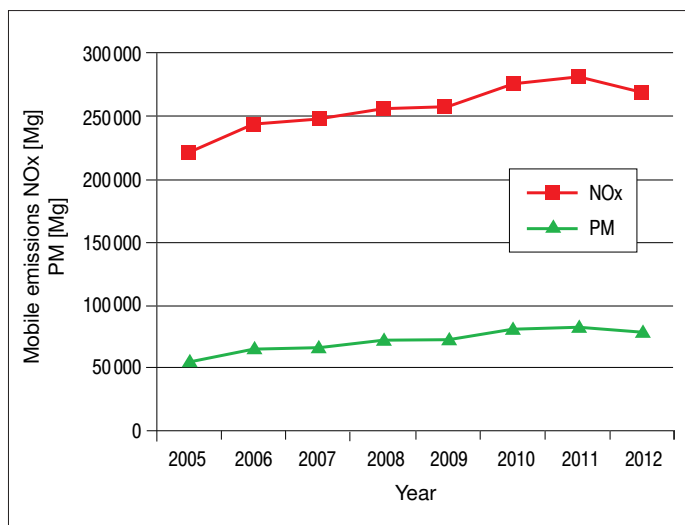


Fig. 4. Emissions of NOx and PM from vehicles in 2005–2010 [45, 46]

CONCLUSIONS

- The impact of particulate matter and nitric oxide is harmful to people.
- A summary of the quantitative analysis of different pollution sources was presented in order to assess which is the biggest threat to citizens. Among others, pollution from mobile sources was evaluated as one that should be treated by the government and society as a priority for further reduction.
- Further analysis has shown that the main reason for that situation is the structure of the vehicle market in Poland. It is suggested that the most efficient way to improve air quality, in terms of transportation, would be both actions leading to gradual reduction of the number of vehicles in use, as well as replacement of the oldest vehicles that do not meet modern EURO standards and promotion of more ecological forms of transportation, such as utility cycling, urban transportation or electrical vehicles.
- Further rapid increase in the number of cars in Poland will impair the quality of the environment, hence also the quality of human life, affecting health, especially in children and elders. Since the impact of motorization on human health is extensive, in the paper only two out of several issues have been explored. In particular, the important problems of noise and cancer-causing benzene, PAHs or heavy metals have not been considered.
- Mobile sources of PM contribute to less than 20% of total PM emissions. Still, it has to be emphasized that household emissions are the greatest contributors of PM in urban areas.

REFERENCES

- [1] Air quality in Europe – 2013 report. European Environment Agency. 2013;No. 9
- [2] Krajowy bilans emisji SO₂, NO_x, CO, NH₃, NMLZO, pyłów, metali ciężkich i TZO za lata 2010-2011 w układzie klasyfikacji SNAP. Raport Syntetyczny. Krajowy Ośrodek Bilansowania i Zarządzania Emisjami 2013.
- [3] Wichmann HE. Diesel exhaust particles. *Inhal Toxicol.* 2007;19 Suppl., 1:241-4.
- [4] Seńczuk W. Niemetale i ich połączenia nieorganiczne. In: Seńczuk W. Toksykologia. Wydanie IV. Wydawnictwo Lekarskie PZWL. Warszawa 2002 (in Polish).
- [5] Florek E. Skład chemiczny i kancerogeny dymu tytoniowego. *Alkoholizm i Narkomania*, 1999;36(3):333-345.
- [6] Arden Pope III C. Respiratory Hospital Admissions Associated with PM₁₀ Pollution in Utah, Salt Lake, and Cache Valleys. *Arch. Environ. Health Journal.* 1991;46:90-97.
- [7] Anderson JA, Thundiyil JG, Andrew Stolbach A. Clearing the Air: A Review of the Effects of Particulate Matter Air Pollution on Human Health. *J. Med. Toxicol.* 2012;8:166-175.
- [8] Li N, Xia T, Nel AE: The Role of Oxidative Stress in Ambient Particulate Matter induced Lung Diseases and Its Implications in the Toxicity of Engineered Nanoparticles. *Free Radic Biol Med.* 2008;44(9):1689-1699.
- [9] Brook RD.: Cardiovascular effects of air pollution. *Clinical Science.* 2008;115:175-187.
- [10] Bartosz G. Druga twarz tlenu. Wolne rodniki w przyrodzie. Wydawnictwo Naukowe PWN, Warszawa 2008 (in Polish).
- [11] Becker S, Dailey LA, Soukup JM et al.: Seasonal variations in air pollution particle-induced inflammatory mediator release and oxidative stress. *Environ Health Perspect.* 2005; 113:1032-1038.
- [12] Sagai M, Furuyama A, Ichinose T. Biological effects of diesel exhaust particles (DEP). III. Pathogenesis of asthma like symptoms in mice. *Free Rad Biol Med.* 1996;21(2):199-209.
- [13] Manzo ND, LaGier AJ, Slade R et al. Nitric oxide and superoxide mediate diesel particle effects in cytokine-treated mice and murine lung epithelial cells – implications for susceptibility to traffic-related air pollution. *Particle and Fibre Toxicology.* 2012;9(43):1-15.
- [14] Kreyling WG, Semmler-Behnke M, Moller W.: Ultrafine particle-lung interactions: does size matter? *J Aerosol Med* 2006; 19:74-83.
- [15] Nel A, Xia T, Madler L et al. Toxic potential of materials at the nanolevel. *Science* 2006;311:622-627.
- [16] Ziętkowski Z, Ziętkowska E, Bodzenta-Ekaszkyk A. Kliniczne znaczenie pomiarów stężenia tlenu azotu w powietrzu wydychanym w chorobach układu oddechowego. *Alergia Astma Immunologia* 2009;14(4):215-222 (in Polish)
- [17] Kowalczyk E, Kopff A, Kopff M et al.: Metabolizm tlenu azotu. *Wiad Lek* 2006;59(11-12):889-893.
- [18] Grisham MB, Jourd'Heuil D, Wink DA. Physiological chemistry of nitric oxide and its metabolites: implications in inflammation. *Am J Physiol Gastrointest Liver Physiol* 1999; 276:315-321.
- [19] Peter J. Barnes.: Nitric Oxide and Airway Disease. *Ann Med.* 1995;27(3):389-393.
- [20] Perrone LA, Belser JA, Wadford DA et al. Inducible nitric oxide contributes to viral pathogenesis following highly pathogenic influenza virus infection in mice. *J Infect Dis* 2013; 207(10):1576-84.
- [21] Fusco D, Forastiere F, Michelozzi P et al. Air pollution and hospital admissions for respiratory conditions in Rome, Italy. *Eur Respir J* 2001;17:1143-1150.
- [22] Michel T, Feron O: Nitric Oxide Synthases: Which, Where, How, and Why? In: Perspective Series: Nitric Oxide and Nitric Oxide Synthases. *J Clin Invest* 1997;100(10):2146-2152.
- [23] Krajowy bilans emisji SO₂, NO_x, CO, NH₃, nmlzo, pyłów, metali ciężkich i tzo za lata 2011 - 2012 w układzie klasyfikacji snap, Raport syntetyczny, March 2014.
- [24] strona internetowa: <http://www.epa.gov/air/nitroge...>, acces: 2014-05-13.
- [25] Geiss O, Barrero-Moreno J, Tirendi S et al. Exposure to Particulate Matter in Vehicle Cabins of Private Cars, Aerosoll and Air Quality Research, Volume 10, No. 6, December 2010, p. 581-588
- [26] Risk assessment of selected pollutants, in: Air Quality Guidelines. Global update 2005. WHO Europe 2005.
- [27] Transport drogowy w Polsce w latach 2005-2009, Główny Urząd Statystyczny, Warszawa 2011.

- [28] Transport drogowy w Polsce w latach 2010-2011, Główny Urząd Statystyczny, Warszawa 2013.
- [29] Pasy bezpieczeństwa, Badanie zrealizowane przez PBS DGA na zlecenie Krajowej Rady Bezpieczeństwa Ruchu Drogowego, Maj 2011.
- [30] Rhys-Tyler GA, Legassick W, Bell MC. The significance of vehicle emissions standards for levels of exhaust pollution from light vehicles in an urban area, Atmospheric Environment. 2011;45:3286-3293.
- [31] Piock W, Hoffmann G, Berndorfer AP et al. Strategies Towards Meeting Future Particulate Matter Emission Require-

ments in Homogeneous Gasoline Direct Injection Engines, SAE International, 2011.

Corresponding author:

*Jakub Krzeszowiak
Department of Hygiene
Wrocław Medical University
ul. Mikulicza Radeckiego 7
50-435 Wrocław*