

What impact of air pollution in pediatric respiratory allergic diseases

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Abstract

Respiratory allergies are known to affect people all over the world. Environmental factors related to pollution play a significant etiopathogenic role in this regard. Polluting sources are industrial activities and urban traffic, capable of generating various types of pollutants that trigger inflammatory, direct, and indirect damage to tissues, promoting allergic symptoms, even serious ones, and interfering with the pharmacologic response. They are also able to modify pollen, promoting allergic sensitization. Pollution could have played a significant predisposing role in the ongoing morbidity and mortality of SARS-CoV-2.

KEYWORDS

air pollution, asthma, oxidative stress, particulate matter, respiratory allergic disease, SARS-CoV-2

The problem of respiratory allergies concerns about 40% of the world population, with a significant impact on the quality of life for those who suffer from it. The most common clinical manifestations are allergic rhinoconjunctivitis and asthma, determined by genetic and environmental factors that can alter lung development in children and adolescents. Air pollution has a significant responsibility in this.¹ Among the main pollutants are certainly PM₁₀ (particulate matter) with an aerodynamic diameter of 10 μm and <2.5 μm (PM_{2.5}) and ultrafine particulate matter (UFP), which are particles <0.1 μm .² European legislation has set the "recommended" maximum concentration levels at 50 and 25 $\mu\text{g}/\text{m}^3$ for the annual average, respectively, for PM₁₀ and PM_{2.5}.

The particulates are produced from anthropogenic sources such as traffic, combustion processes, and industrial gas activities, and from natural sources, such as marine spray, crustal minerals, and forest fires. It also has a secondary component that is formed in the atmosphere and which, especially in the urban area, constitutes a significant fraction. Dimensions, surface, and composition of the polluting particle determine the potential risk for a patient who is exposed regularly.³⁻⁵ Early exposure, in gestational or neonatal times, for example, can trigger epigenetic mechanisms, such as DNA methylation and oxidative stress, which increase the risk of adverse outcomes at birth and the subsequent development of asthma and allergic rhinitis.⁶ Another powerful

oxidant is tropospheric ozone (O₃), a colorless gas that is created by photochemical reactions between nitrogen oxide and volatile organic compounds produced by the combustion of fuels and fossil fuels, associated with damage to the lungs tissue and the appearance of asthma. vehicular traffic, residual oil fly ash (ROFA), a complex mixture of sulfates and metals, in particular vanadium, adversely affects the immune system, causing bronchial hyper-reactivity and airway injury with increased asthma and allergies in childhood. It seems that air pollutants also can modify the allergenicity of some pollens, facilitating their dispersion in smaller fractions. In experimental models, ozone has produced a structural change in the coating layers of the pollen, influencing its interaction with human cells and enhancing the pollen's allergenic properties with the increase in new sensitizations.⁷ A Japanese study of 2506 children compared environmental nitric oxide (NO₂) levels and the incidence of asthma, monitoring them for 4 years. It has been observed that those who lived less than 50 meters from busy roads were more likely to develop asthma.⁸ The diesel exhaust particles (DEP) are made up of nanoparticles with a carbonaceous core, with a large surface area, to which other chemical products adhere, such as polycyclic aromatic hydrocarbons (PAH), heterocyclic, aldehydes, and aliphatic hydrocarbons. DEP can reach the cell surface and activate stimulation mechanisms for airway epithelial cells, inducing the production of cytokines, which lead to inflammation. In *in vitro* studies, these particles stimulate the epithelial and endothelial cells of the pulmonary microcirculation, with the release of IL-8, macrophages, granulocyte-stimulating factor, T cells, and intracellular adhesion molecules (ICAM and VCAM). It should be noted that PM has both seasonal and source-related variability, with inflammatory and pro-carcinogenic repercussions. PM₁₀ is also able to activate the Nlrp3 inflammasome, in response to extra- and intracellular warning signs, including mitochondrial oxidative stress, with significant production of IL-1B. All elements found significantly, together with eotaxin, are a chemokine produced by epithelial cells that stimulate the migration of eosinophils from small pulmonary vessels in patients with asthma. Particulate matter is also capable of damaging the DNA of lung cells.⁹ All this determines different influences on individual genotypes that lead to heterogeneous endotypes of asthma and increasingly severe clinical phenotypic expressions, impacting drug treatment's response. It should also be considered that more than half of the air inhaled during our life comes from an indoor environment. The sources of indoor contaminants mainly come from cigarette smoke, combustion products, and volatile organic compounds emitted by building materials, paints, products containing polyvinyl chloride (PVC), and food preparation. More than one study has shown that the concentrations of indoor contaminants (PM₁, PM_{2.5}, and PM₁₀) are higher than the outdoor values of PM₁₀ recommended by the European Union, and this can induce or worsen allergic symptoms. The average concentration of individual components of volatile organic compounds is constantly higher in the families of children diagnosed with asthma, rhinitis, and eczema, compared

Key Message

Environmental exposures related to airway pollution interplay with human host factors to promote the development and progression of allergic diseases.

to the control groups.¹⁰ All this leads us to consider the pollution aspect also about the ongoing epidemiologic analyses of the current SARS-CoV-2 pandemic. To reduce the spread of the disease, the drastic contraction in transport, work activities, and schools throughout the area concerned has led to a significant reduction in the concentrations of nitrogen dioxide (NO₂), an important tracer of the air pollution associated with morbidity and mortality. In central China, NO₂ emissions have been reduced by up to 30% (NASA, 2020). Similarly, CO₂ emissions, another common factor of air pollution, also decreased by 25% in China and by 6% in the rest of the world. The reduction in concentrations of PM_{2.5} and PM₁₀ was lower than that of NO₂, both because the weight of the different sources is different and because of the influence of the secondary component on the particulate matter. The effective role of pollution, as a cofactor of additional morbidity and lethality of SARS-CoV-2, in general, and in the regions of northern Italy, currently has no objective findings.¹¹ However, the Air Quality Index (AQI), an index recently introduced by the European Environment Agency (EEA), based on the concentration values of up to five key pollutants, PM₁₀, PM_{2.5}, O₃, SO₂, and NO₂S, which reflects the potential impact of air quality on health, highlights the area covered by Lombardy and Emilia Romagna as the most polluted in Italy.^{12,13}

1 | CONCLUSIONS

Air pollution is a determining factor in the development and increase in the clinical manifestations of allergic disease. Epigenetic, cellular, and molecular mechanisms that act, directly or indirectly, already in the prenatal period and how they contribute to the negative impact of patients' quality of life are still under investigation. Similarly, the role of pollution in the ongoing SARS-CoV-2 pandemic is still discussed and deserving to be explored even though a paradox can be affirmed: The decrease in air pollution following the lockdown linked to the SARS-CoV-2 pandemic could have reduced the total number of deaths during this period, reducing the number of victims caused by air pollution. It is useful to advise everyone to minimize prolonged exposure to pollutants, limiting outdoor activities on days with high levels of atmospheric pollution. We all have a moral duty to combine our efforts and scientific knowledge, supporting environmental policies to stem as much as possible the impact and consequences of this significant public health problem for the future.

CONFLICTS OF INTEREST

All authors declare that they have no conflict of interest.

PEER REVIEW

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REFERENCES

1. D'Amato G, Pawankar R, Vitale C, et al. Climate change and air pollution: effects on respiratory allergy. *Allergy Asthma Immunol Res*. 2016;8:391-395.
2. Clifford RL, Jones MJ, MacIsaac JL. Inhalation of diesel exhaust and allergen alters human bronchial epithelium DNA methylation. *J Allergy Clin Immunol*. 2017;139:112-121.
3. Riedl M, Diaz-Sanchez D. Biology of diesel exhaust effects on respiratory function. *J Allergy Clin Immunol*. 2005;115:221-228.
4. Cilluffo G, Ferrante G, Fasola S, et al. Associations of greenness, greyness and air pollution exposure with children's health: a cross-sectional study in Southern Italy. *Environ Health*. 2018;17:86.
5. Ruggieri S, Longo V, Perrino C, et al. Indoor air quality in schools of a highly polluted south Mediterranean area. *Indoor Air*. 2019;29:276-290.
6. Deng Q, Lu C, Li Y. Exposure to outdoor air pollution during trimesters of pregnancy and childhood asthma, allergic rhinitis and eczema. *Environ Res*. 2016;150:119-127.
7. Bowatte G, Lodge CJ, Knibbs LD. Traffic-related air pollution exposure is associated with allergic sensitization, asthma, and poor lung function in middle age. *J Allergy Clin Immunol*. 2017;139:122-129.
8. Shima M, Nitta Y, Ando M. Effects of air pollution on the prevalence and incidence of asthma in children. *Arch Environ Health*. 2002;57:529-535.
9. Bengalia R, Zerbonia A, Marchetti S, et al. In vitro pulmonary and vascular effects induced by different diesel exhaust particles. *Toxicol Lett*. 2019;306:13-24.
10. Cincinelli A, Martellini T. Indoor air quality and health. *Int J Environ Res Public Health*. 2017;14:1286.
11. Longhin EM, Mantecchia P, Gualtieri M. Fifteen years of airborne particulates in vitro toxicology in Milano: lessons and perspectives learned. *Int J Mol Sci*. 2020;21:2489.
12. Contini D, Costabile F. Does air pollution influence COVID-19 outbreaks? *Atmosphere*. 2020;11:377.
13. Conticini E, Frediani B, Caro D. Can atmospheric pollution be considered a co-factor in extremely high level of SARS-CoV-2 lethality in Northern Italy? *Environ Pollut*. 2020;261:114465.

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