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Climate change and allergic diseases in children and adolescents

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Abstract
Introduction: The Anthropocene is used to describe the most recent period where major disruptions in Earth's system processes have resulted from humanity's increasing ecological footprint. Climate change affects the social and environmental determinants of good health, such as clean air, stable ecosystems, safe drinking water, and sufficient and safe food, and they seem to be closely related to air pollution.
Objectives: This article aims to review the evidence of how extreme weather events and indoor and outdoor pollution are associated with insufficient lung growth in early life, changes in lung function, and the increase in respiratory infections, favoring the development of allergic respiratory diseases.
Material and Methods: Non-systematic review of English, Spanish, and Portuguese articles published in the last ten years in databases such as PubMed, EMBASE, and SciELO. The terms used were air pollution OR climate changes OR smoke, AND health OR allergic disease.
Results: Climate change and air pollution are the leading contributors to health emergencies around the world. On a global scale, those most at risk of adverse health effects associated with climate change include children, the elderly, and other vulnerable groups. Climate change and air pollution have adverse impacts on respiratory allergies, and the mechanisms are complex and interactive.
Conclusion: Health professionals must receive information and education necessary to establish effective mitigation and adaptation strategies to minimize the effects of climate changes on the respiratory health of their patients.

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Introduction

The Anthropocene period brought with it great acquisitions for humanity, and with these came humankind's significant impact on Earth's geology and ecosystems. In this period, before the end of World War II, there were atomic bomb explosions that covered the globe with radioactive debris that became embedded in sediment and glacial ice. Then, there was rapid population growth, swift acceleration of industrial production, and the use of agricultural products and other human activities that determine climate change[1] or anthropogenic global warming.

Disorderly population growth in large urban centers, the continuous burning of fossil fuels, air pollution, global warming, loss of biodiversity, heatwaves, droughts that facilitate wildfires, increased intensity of storms and floods, interference in the food crops, as well as the reduction of its nutritional value, and the change in the pattern of infectious vectors are all due to climate change. They induce stress; mental problems; food-, water-, or vector-borne diseases; respiratory diseases; malnutrition; and diseases secondary to excessive heat, poverty, and displacement.[2,3]

Climate change and air pollution have been associated with risks to individual health and well-being, especially for those most vulnerable, such as children. Studies have evaluated various routes of prenatal and postnatal exposure, and although we cannot predict what changes will be induced in future generations, they are likely to have a combined effect that will accelerate or worsen the morbidity and mortality rates of many health conditions, including allergic diseases.[4]

Early exposure, epigenetics, and microbiome

Evidence has been documented on the action of environmental pollution (household and outdoor) on human health,[5] associating with the increased prevalence of asthma and allergic diseases observed in recent decades.[6-8] The increase occurred so quickly that it cannot be explained only by variations in the genome but as a result of epigenetic mechanisms resulting from alterations exerted by environmental factors.[9,10]

Climate change associated with increased concentration of pollutants such as ozone (O_3), nitrogen dioxide (NO_2), and volatile compounds (VOCs) led to a greater interest in understanding the effects that the exposome has on the development of allergic diseases.[11,12]

Currently, more and more evidence that environmental exposures early on in life, including intrauterine exposure, can alter developmental trajectories and initiate the onset of allergic diseases is accumulating.[13] Furthermore, other evidence suggests that information from previous exposures can be transferred across generations, and experimental animal models suggest that such transmission can be through epigenetic mechanisms.[14-17]

Epigenetic changes modify the genotypic expression without changing the DNA sequence, allowing the body to adapt to those environmental changes without modifying the genome. The most common epigenetic mechanism is cytosine methylation at the cytosine-phosphate-guanine (CpG) site, which blocks the action of the gene promoter, suppressing gene expression.[18]

New evidence supports the impairment of epigenetic mechanisms (primarily methylation) in the relationship between environmental NO_2 and O_3 pollution exposure and neurodevelopmental, metabolic, inflammatory, and asthma disorders. A similar mechanism can be seen in the epigenetic effect of exposure to cigarette smoke in pregnant mothers.[19] A recent study of two cohorts showed evidence of specific alterations in eight CpG sites in eight genes related to lung morphogenesis that, when present at birth, can predict a 26-year trajectory of lung function.[20]

Microbiota

Environmental pollution and the loss of macro- and microbiodiversity due to human activities have been related to health problems, especially inflammatory and allergic diseases.[21] On the other hand, the balance of the intestinal microbiota (eubiosis) with adequate biodiversity becomes a central factor in epigenetic adaptation, preventing the development of allergies and allowing resilience to changes in environmental situations.[22] A clear example is the difference in microbiota between urban and rural environments, and within that, the protective effect of contact with farm animals, even when exposed to antibiotics in the first year of life.[23]

Studies have shown that changes in the composition of the maternal intestinal microbiota and its metabolites can generate epigenetic changes (e.g., reduced DNA methylation, inhibition of histone deacetylase or butyrate, a metabolite of the microbiota, which can increase the expression of the forkhead box P3 (FOXP3) protein that stimulates the generation of Treg progeny) that modify the predisposition to allergic diseases.[24] Adequate maternal exposure to a varied environmental microbiota, fiber consumption, low levels of stress and mental illnesses, and the absence of antibiotic use reduces the risk of allergic diseases in children due to the impact on the maternal microbiota and its products, leading to the establishment of a healthy microbiota in the baby, with a consequent tolerogenic attitude.[25]

In short, early exposure to environmental factors at critical periods, a product of climate change caused by Western and industrialized lifestyles, induces epigenetic changes that may be behind the increase in the prevalence of allergic diseases, in the same way that a balanced microbiota in the mother–child binomial is a protective factor for their future development.

Climate change, air pollutants, and allergic sensitization

The impacts of climate change on allergens and allergic diseases are complex, and multifactorial health effects of air pollutants depend on a wide range of exogenous and endogenous factors, including physical and chemical characteristics of the pollutants and the anatomical or physiological condition of the individual such as breathing patterns or level of activity.[26]

One of the mechanisms of air pollutants that cause adverse effects on respiratory health is due to oxidative stress, which damages the respiratory epithelium and enhances cellular inflammation. Components of air pollution may interact with airborne allergens and enhance the risk of atopic sensitization and exacerbation of symptoms in sensitized subjects. When airborne particles with strong oxidative activity such as O_3 and nitrogen oxides (NO_x), in suspension or gaseous form, are exposed to the respiratory epithelium, the formation of free radicals is induced, which triggers the release of proinflammatory cytokines and chemokines from effector cells and upregulation of adhesion molecules. Particle matter (PM), diesel exhaust particles, O_3, NO_2, and sulfur dioxide (SO_2) have been shown to have an inflammatory effect on the airways of susceptible subjects, increasing the release of soluble intercellular adhesion molecule 1 via a Toll-like receptor (TLR) 4 pattern recognition and/or TLR2 activation via activation of NFk-B signaling and NLRP3 inflammasome resulting in induced production of the proinflammatory mediators interleukin (IL)-1α, IL-1β, IL-6, IL-6, and CXCL8 and colony-stimulating factor (GM-CSF). All of these changes have a direct effect of the sensory cells that line the epithelium of the airways, which affects the smooth muscle and increases the permeability and facilitates the penetration of allergens into the mucous membranes and interaction with the cells of the immune system.[27-33]

Although the effect of each air pollutant may be unique, when combined with the effects of acute large exposure or chronic low-level exposure and a mixture of airborne pollutants, there is often an even greater pronounced effect on individuals with underlying allergic or nonallergic airway diseases.[26,34]

Increased pollen seasons and allergen exposure

PM refers to a mixture of solid particles and liquid droplets found in the atmosphere, and its main biological sources are marine spray, mineral, pollen, or organic substances emitted by natural sources and nonbiological sources are generated by human action mainly by the combustion of fossil fuels and industries. PM is categorized according to its aerodynamic diameter: PM_{10} (smaller than 10 μm), $PM_{2.5}$ (smaller than 2.5 μm), and ultrafine PM (smaller than 0.1 μm).[35]

Several studies have now confirmed that $PM_{2.5}$ airborne particulates in addition to other gaseous elements such as O_3, carbon (C), SO_2, and NO_2 can cause immunologic changes that favor development or/and an increase in asthma exacerbations resulting in increased frequency of emergency room visits and hospitalizations. Furthermore, some of these air pollutants such as $PM_{2.5}$ and ozone have been shown to work synergistically with specific allergens to enhance immunogenicity, which may also worsen asthma in susceptible individuals.[28,32,36,37]

Most of the world's industrialized cities have a substantially high number of toxic gases in the atmosphere mixed with PM. All biological (such as pollen) and nonbiological matters (carbon, silicon, metals, ultrafine dust, and others) interact with each other in the atmosphere with direct effects on the health of the population, either due to their direct irritant effect or due to their interactions with this particulate matter.[35,37,38]

Evidence suggests that prevalence of pollen-related allergic respiratory diseases, for example, rhinitis and asthma, has increased in past decades. Data shows that climate change affects the production and protein composition of pollen and fungal spores and impacts aerobiological processes such as emission, dispersion, transport, and deposition associated with changing rainfall, winds, and other related meteorological factors. The composition and relative abundance of airborne pollen in urban and rural areas are strongly influenced by the climate. Environmental pollution and climate change has an impact on local flora, modifying their pollination season and/or pollen protein composition, and evidence shows that pollen in heavily polluted zones expresses a larger number of allergenic proteins, compared with areas characterized by less pollution. In cases of high levels of vehicular emissions, urbanization and Westernized lifestyles correlate with the increased frequency of pollen-induced respiratory allergies in urban areas as compared to that in rural areas.[28,36,39,40]

Pollen collected along high-traffic roads showed higher allergenicity and children living closer to high-traffic roads have been reported to have more severe respiratory allergies. In fact, air pollutants interact with allergen-carrying submicronic and paucimicronic particles derived from pollen and can reach peripheral airways inducing asthma in sensitized subjects.[41,42]

The increase in global temperature associated with climate change has modified the duration of pollinization seasons, affecting the duration and the usual periods during which plants release their pollen. Other effects associated with climate changes are related to several changes in rainfall, hurricanes, and stronger winds, which can further expand the reach of pollen species in the atmosphere and carry non-native pollen species from different regions to potentially sensitized susceptible populations in remotes areas.[37,43-46]

Climate change will also increase the frequency and intensity of floods; worsen storms; and thus increase production of fungal spores, a powerful asthma and rhinitis trigger; increased mold exposure has been linked to other lung diseases.[29]

Indoor air pollution

Indoor air exposures are a mixture of ambient air pollution brought into the home by ventilation and infiltration and pollution generated inside the home and emitted by the combustion of coal, kerosene, and biomass (wood, charcoal, crop waste, and animal manure); construction and furniture materials; and human behavior (smoking, cooking, and cleaning products).[47]

Pollutants in indoor air include inorganic gases such as carbon monoxide (CO), carbon dioxide (CO_2), reactive gases (O_3, NO_X), a wide range of VOCs and semivolatiles (SVOCs), and PM_{10}, $PM_{2.5,}$ and $PM_{1.0}$. Some compounds, such as polycyclic aromatic hydrocarbons (PAHs), perfluorinated compounds (PFCs), polychlorinated biphenyls (PCBs), and polybrominated diphenyl ethers (PBDEs), are found in the

gaseous and particulate phases, depending on the partition behavior and the emission source.[48]

In 2019, it was estimated that only 12% of families in countries with a low human development index (HDI) had a primary dependence on fuels and clean technologies for cooking. In rural families, in several countries with low and medium HDI, the average concentration of $PM_{2.5}$ in the main internal cooking area is estimated at more than 500 μg/m^3. In Ethiopia, the observed concentration exceeded 1,200 μg/m^3, 120 times the limit set by the World Health Organization (WHO).[49,50]

Energy poverty is a concern even in high-HDI countries, as around 7% of people in the European Union struggle to pay for enough heating for their homes. This puts them at risk for cold-related adverse health outcomes.[51]

It is estimated that approximately three billion people are exposed to indoor pollution (IDP) from the burning of biomass fuels worldwide, and of these, 2.31 million die annually from this exposure,[52] generating significant consequences for health, the economy, and society, particularly in low- and middle-income countries.[53-55]

According to different cultural patterns, household exposure to ambient air pollution is different for men and women (gender inequality). It is estimated that women are exposed 40% more than men,[54] being the second-largest risk factor for death among women and the fifth for men, after smoking, alcohol use, and high blood pressure.[53-56]

Although smoking is the leading cause of chronic obstructive pulmonary disease (COPD) in the developed world, exposure to IDP is a critical factor to be prevented in low- and middle-income countries, especially in women.[57,58] The greatest burden of IDP-related premature deaths is pneumonia in children, resulting from exposure to biomass smoke. Among adults, the greatest estimated burden is cardiovascular diseases, COPD, and lung cancer, which are significant causes of disability and premature death in developing countries.[59] It is essential to mention that current or past exposure to IDP determines an increased risk of respiratory diseases or neoplasms and obliges us to perform earlier screenings on these individuals.[60]

Human behavior and domestic practices

Biomass (wood, manure, and charcoal) is the primary source of domestic energy for 40% of households and 90% of rural households in low-income countries that use it for cooking and heating, often burned in inefficient and poorly ventilated combustion devices (bonfires and traditional stoves).[61]

Although the impact is more pronounced in low-to-middle-income countries (LMICs), significant health effects are observed among those exposed to lower concentrations of pollutants. The WHO estimated in 2016 that around 400,000 deaths in high-income countries were attributed to indoor air pollution from the continued use of solid fuels.[62] Even for households that have transitioned to cleaner fuels such as electricity, liquefied petroleum gas, or ethanol, the current economic slowdown caused by the Covid-19 pandemic could mean a necessary return to fuelwood consumption or other cooking methods that cause pollution.[63]

NO_2 is of particular interest to households that use gas stoves for cooking.[64] For every hour of gas stove or furnace use, there is an associated increase of 18 parts per billion (ppb) in NO_2 concentrations in 24 hours. In poorly ventilated houses, and depending on how frequently the stoves are used, domestic concentrations of $PM_{2.5}$ can increase significantly.[65] A recent study showed that gas stoves leak methane even when appliances are not in use.[66]

Indoor cooking is a known source of aerosols. Depending on the cooking method used (steaming, boiling, frying), anything that uses oil generates more aerosols.[67] Olive oil and peanut oil were associated with the highest $PM_{2.5}$ emission compared to coconut, soy, corn, and canola oils.[68]

Burning candles, used for esthetic and religious purposes (meditation, memorials, and ceremonies), usually in internal settings, is a source of particulate emissions.[69] The amount of ultrafine particles generated by burning candles is greater than that produced by smoking, frying meat, cooking with an electric stove, and other sources of particulate emissions.[70] These ultrafine particles are mostly deposited in the alveolar region.[71]

"Mosquito-killing" spirals are often burned as insect repellent in summertime. They are generally burned slowly indoors and generate high concentrations of PM.[72] The same happens when burning incense indoors, which is a significant source of PM_{10} and $PM_{2.5}$.[73]

Usage of household cleaning products, especially the more powerful disinfectants used to reduce viral infection rates, has been further encouraged, causing overexposure to the chemical agents they release and leading to unintended risks to human health.[74]

Having several internal sources that can emit quantities of fine and ultrafine particles that can remain suspended and accumulate in the air, and the widespread adoption of sedentary lifestyles by the population in recent years, significantly increases the probability of exposure to internal aerosols, especially in closed spaces with inadequate ventilation.[75,76]

Tobacco Exposure

Tobacco smoke is another significant source of IDP, particularly indoors and in buildings.[77] It accounts for 50%–90% of indoor PM concentrations in high-income countries. $PM_{2.5}$ concentrations were observed to be 10 times higher in smokers' homes when compared to nonsmokers homes, especially if ventilation is inadequate.[78,79]

Ambient tobacco smoke is a primary source of not only $PM_{2.5}$ but also NO_2 and several VOCs.[80] Much of the developed world has adopted national regulations prohibiting smoking in public spaces with the primary aim of protecting the health of nonsmokers,[81] but homes have not yet been incorporated into this legal framework and therefore remain a source of exposure to many adolescents and young children.[82]

Exposure to electronic cigarettes

Exposure to electronic cigarettes (ECs) during pregnancy can compromise placental function, resulting in fetal structural abnormalities, and it has deleterious effects on the health of the exposed offspring.[83] Gestational exposure

to EC aerosols can impact crucial biological processes, with significant alterations in the amino acid profile in the maternal and fetal compartments, including the fetal lungs. Studies demonstrate a targeted disruption of the NO pathway, branched-chain amino acid metabolism, fetal protein synthesis, and the urea cycle,[84] leading to pathophysiological changes in developing lungs, which in turn can impair respiratory health later in life.[83]

Experimental studies in a murine model suggest that exposures to EC emissions during prenatal and early life may impair postnatal lung growth and Wnt signaling in the lung. WnT signaling is one of the major signaling pathways involved in regulating embryonic and fetal lung development.[85]

As ECs do not generate secondary smoke like conventional cigarettes, the EC exhaled aerosol is the only source of environmental exposure.[86] This generates a low perception in parents about the risks associated with exposing children to ECs, increasing their susceptibility to the harmful effects of passive vaporization,[83] especially in closed environments.

The consumption of ECs can cause short- and long-term respiratory problems in the pediatric population and a great concern for young people developing nicotine dependence in the future. Although second- and third-hand exposures are likely to be lower than those associated with conventional smoking, ECs and new generations of heated tobacco products (IQOS) are significant sources of indoor pollution.[87]

It should be noted that smoking inside cars is of particular concern because concentrations of potentially harmful substances can become very high in such small spaces. Tobacco cigarettes, ECs, and heated tobacco products are avoidable sources of indoor pollutants. To protect the health of other nonsmoking passengers, primarily sensitive people such as children and pregnant women, these products should not be used in automobiles.[88]

Indoor Pollution and Health Impacts

Pre- and postnatal exposure to PM_{10} and maternal smoking during pregnancy have been associated with reduced lung function, especially in children with a genetic predisposition to asthma.[89] A baby's altered lung function can increase the risk of pneumonia and respiratory infections in the first few years of life.[90] Lee *et al.* evaluated the association between maternal exposure to CO from wood stove sources as the dominant fuel and infant lung function at 30 days, demonstrating an increased risk of physician-diagnosed pneumonia and severe pneumonia in the first year of life.[91]

Women and children are particularly susceptible to the toxic effects of pollution and are exposed to the highest concentrations. Interventions must target these high-risk groups and be of sufficient quality to make the air clean.[5,92] Thus, to ensure that the air in homes and their surroundings is healthy, the new WHO guidelines on air quality and fuel use in homes contain recommendations on types of fuels and technology to protect health, as well as strategies for disseminating these domestic fuels.[49]

Reduction targets in the burden of IDP-related diseases are necessary to assess progress toward Sustainable Development Goal 3 (SDG 3). Ensuring universal access to clean fuels and technology is also a Development Goal for Affordable and Clean Energy (SDG 7). Meeting this goal would prevent millions of deaths and improve the health and well-being of billions of people who depend on fuels and technology that pollute for cooking, heating, and lighting.[93]

Wildfire and noxious action on health

The increase in the frequency and intensity of wildfires over the past 40 years has been largely driven by the impact of climate change, the combination of hot, dry, and windy conditions, deforestation, and human intervention.[94] While wildfires in high-income countries attract a lot of attention, low- and middle-income countries have the highest arson activity by far.[95] Wildfires have a very high cost, but prescribed fires, although on a smaller scale, have the highest costs per hectare, probably due to their closer proximity to where urban and rural environments meet. Therefore, they usually occur closer to populated areas.[96]

While wildfires cause widespread damage and threaten the lives of those who fight and flee from them, the PM in the smoke caused by the burning poses the most significant risk to public health.[94] Exposure to $PM_{2.5}$ from wildfire smoke is estimated to account for 680,000 annual deaths, 270,000 of which occur in children under five years old.[95]

Wildfires substantially increase the environmental risks to the health of people living in the affected areas, especially those who are disadvantaged either by their socioeconomic status, racial/ethnic origin, geographic location, age, and those with pre-existing health and working conditions.[97,98]

Exposure to PM from wildfires is associated with an increased risk of respiratory events, including impaired lung function, hospitalizations, and emergency room visits, and the use of medications for asthma, COPD, and respiratory infections.[92]

Pregnant women exposed to wildfire smoke are at an increased risk of adverse effects on their pregnancy and fetal consequences, such as low birth weight, premature birth, and infant mortality.[95,98] If the expectant mother is a smoker, the toxic impacts can increase, and the mental stress can lead to increased tobacco consumption.[99]

Changes in DNA methylation are postulated as potential epigenetic mechanisms that link exposure to PM from smoke and prenatal stress to poor respiratory health outcomes during childhood.[100]

$PM_{2.5}$ related to wildfires has long-range transport capabilities and contributes to poor air quality, even after fire seasons.[101,102] The greatest toxicity potential of $PM_{2.5}$ resulting from wildfires is attributed to its small particle composition (submicrometric and ultrafine) and more oxidative and pro-inflammatory components, such as PAHs and aldehydes. Other pollutants, such as O_3 and NO_2, add to the effects of $PM_{2.5}$, which by association can amplify its health risks.[98,101]

Short-term exposures to high concentrations of $PM_{2.5}$ from wildfires can lead to many health outcomes, including coughing, asthma, heart attacks, stroke, cardiorespiratory disease, acute and chronic effects on respiratory health,

decreased lung function, hospital stays, and premature death.[94,102]

$PM_{2.5}$ from wildfires has been identified as at least 10 times more harmful to children's respiratory health than other sources.[103] A pediatric study documented that children under five years of age exposed to $PM_{2.5}$ from wildfires made twice as many emergency room visits for respiratory problems per day compared to 6- to 12-year olds.[18]

Individuals born prematurely or with low birth weight may be at greater risk of adverse respiratory health effects after exposure to wildfire smoke in early adulthood, possibly related to having worsened expiratory flow rates.[104]

A follow-up study on exposure to pollution from wildfires documented a significant reduction in lung function (ratio between forced expiratory volume in one second and forced vital capacity) after one year of the event and which persisted until two years later (33.9%). These data support the idea that long-term exposure to smoke induces obstructive pulmonary abnormalities.[105]

A recent study evaluated the relationship between short-term exposure to wildfire smoke and emergency hospital admissions in Brazil and identified significant associations between $PM_{2.5}$ concentration and hospitalizations for respiratory and cardiovascular causes, with children (0–9 years) and elderly individuals (≥80 years) being the most vulnerable.[103]

Acute exposures at 10 μg/m^3 $PM_{2.5}$ from wildfires were associated with approximately 2% increases in mortality rates from cardiovascular, respiratory, and all causes of illness. It is thought that this greater toxicity is due to a synergy between the wildfire smoke and the combination with other sources of indoor air pollution.[101,106]

A North American study verified a 1% increase in the mortality rate from nontraumatic causes in all age groups on days when the population was exposed to wildfire smoke compared to days with exposure to smoke without wildfire pollutants. The per-case stratification of these deaths on the same day revealed that respiratory problems increased by 9% and COPD increased by 14%.[107]

On the other hand, Kiser *et al*. evaluated the number of COVID-19 cases in Reno, United States of America. They documented a positive association between exposure to an increase of 10 μg/m^3 in the average concentration of $PM_{2.5}$ from smoke from wildfires for 7 days and an increase in the number of cases. The authors verified a 6.3% increase in the positivity rate for SARS-CoV-2 and a 17.7% increase in the number of cases estimated during the most intense period of wildfires.[108] An important caveat made by Navarro *et al*. regarded the higher risk of infection by SARS-CoV-2 and severity among the firefighters who worked on controlling the wildfires.[109]

The exact mechanisms by which air pollutants increase the frequency of SARS-CoV-2 infections are not fully understood. They are possibly related to the oxidation and inflammation of the lungs and other tissues resulting from the direct or indirect action of pollutants or even to the alternation of expression of the angiotensin-II converting enzyme in the respiratory system and other cells.[110]

Recently, the Intergovernmental Panel on Climate Change (IPCC) Working Group issued a warning stating that wildfires are likely to become even more common due to human influences and as the climate continues to change.[111]

Conclusions

Since climate change and pollution can cause many unexpected and persistent effects on health, especially in allergic respiratory diseases,[6,8] healthcare professionals must receive adequate and sufficient information and education to establish effective mitigation and adaptation strategies, aiming to minimize these effects on the respiratory health of individuals.[112,113]

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