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Climate Change and Respiratory Allergy

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Abstract

Climate change, a global issue, directly and indirectly impacts human health while posing a significant threat to animals and ecosystems. As part of the exposome, it influences various factors like seasonality, production, and the distribution of airborne allergens due to rising carbon dioxide levels and temperatures. These environmental changes also contribute to increased epithelial permeability, microbial imbalances, and heightened allergic reactions to pollen and fungal spores. Earth's average surface temperature, currently around 14°C, has risen by 1°C over the past century, reflecting the growing urgency of this crisis. This has resulted in change in the environmental pattern and can have serious impact on vulnerable population, such as atopic individuals, elderly, children, or pregnant women. Climate change, air pollution, & reduced biodiversity are inter-related and has led to an increase in respiratory allergy diseases. This review emphasizes the important steps to mitigate the health disparities arising from global warming, climate change, and aeroallergen respiratory allergic diseases.

Keywords: Climate change, Green-house gases (GHGs), particulate matter (PM), volatile organic compounds (VOCs), sand & dust storm (SDS), ozone, carbon dioxide (CO_2)

INTRODUCTION

Climate change is a critical global challenge that drives significant environmental and weather disruptions, profoundly affecting human health, particularly by exacerbating allergies and asthma. It encompasses more than just global warming caused by fossil fuel combustion; it also includes extreme weather events, such as heatwaves, droughts, dust storms, wildfires, heavy rainfall, and flooding. Additionally, climate change contributes to a rise in airborne pollutants, further impacting environmental and public health. Air pollutants include gaseous pollutants (Sulphur compounds, nitrogen compounds, carbon oxides, hydrocarbon, and halogen compounds) and atmospheric particulates (total suspended particles, inhaled particles, PM10, fine particle matter, PM2.5, and ultra-fine particles). In urban areas, the sources of air pollution are industrial plants, motor vehicles and construction activities, while in rural areas biomass combustion (from cooking and burning of crop stubble). Natural sources of air pollutants that affect allergic and asthma diseases are pollen, fungal spores, and dust mites [1]. The main drivers of climate change are the increasing concentrations of greenhouse gases like carbon dioxide (CO_2), methane, and nitrous oxide, which significantly contribute to global warming. Moreover, when sunlight interacts with nitrogen oxides and volatile organic compounds (VOCs), it leads to higher ozone levels. Exposure to ozone poses serious health risks, particularly for vulnerable groups, such as individuals with atopic conditions, the elderly, children, and pregnant women. At the 2023 UN Climate Change Conference (COP28), a landmark agreement was reached, marking the "beginning of the end" for the fossil fuel era. The agreement highlights the urgent need for a fair and rapid transition to renewable energy sources like wind and solar, aiming to triple renewable energy

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capacity by 2030 and achieve carbon neutrality by 2050 (Figure 1) [2–5].

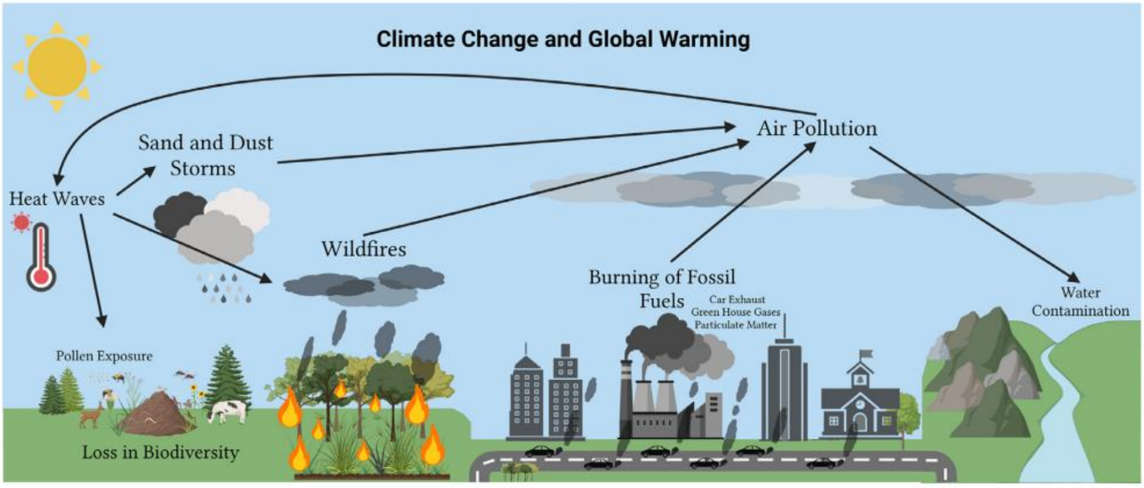

Figure 1. Factors contributing to climate change and global warming.

IMMUNE MECHANISMS OF CLIMATE CHANGE AND RESPIRATORY ALLERGY

Genetic factors contribute to allergic diseases, but the recent surge in their prevalence is too rapid to be explained by inherited genetic changes alone. This trend is now attributed to climate change and air pollution, which influence the body through epigenetic modifications [6–8]. Pollution and climate change events disrupt the epithelial barrier, cause microbial imbalances, and modify both innate and adaptive immune responses (see Figures 2 and 3). Events like air pollution, flooding, heatwaves, and water contamination significantly impact allergic diseases and asthma. Additionally, indirect effects, such as migration, displacement, and food and water insecurity further exacerbate these conditions [9, 10].

In healthy individuals, immune tolerance prevails as an active and essential process. Immune tolerance is linked to factors like exposure to pets and farm animals, breastfeeding, living in rural areas, consuming probiotics, maintaining a diverse diet, protecting the skin barrier, and eating fiber-rich foods, which enhance microbial exposure. Such exposures play a crucial role in training the immune system to build tolerance. Studies emphasize that the first 1,000 days of a child's life are vital for shaping immune tolerance. Both the innate and adaptive immune systems are essential for maintaining balance and defending against harmless substances while preventing allergic reactions to harmful foreign substances. During tolerance, T regulatory (Treg) cells are upregulated [11]. Dendritic cells process antigens and present them to naïve T cells, releasing co-stimulatory molecules, such as TGF-β, IL-10, retinoic acid, indoleamine 2, 3-dioxygenase, and retinal aldehyde dehydrogenase. These signals drive the differentiation of naïve T cells into CD4+ Treg cells, which then produce IL-10 and TGF-β. These cytokines further promote B cell class switching, leading to the production of IgG4 and IgA. IgG4 competes with IgE to reduce allergic responses, while IgA prevents antigen attachment to the epithelium. Immune tolerance involves mechanisms like Th2 cell suppression, increased Tregs, reduced IgE, elevated IgG4 and IgA, higher IL-10 and TGF-β levels, and suppression of basophils, eosinophils, and mast cells. Rising allergies are linked to reduced microbial exposure, pollutants, and allergens. Particulate matter (PM), VOCs, tobacco smoke, wildfire smoke, and heat stress disrupt the skin barrier, generate reactive oxygen species, and activate NF-κB and NLRP3 pathways, driving inflammation (e.g., IL-1, IL-6, TNF-α) and worsening allergic responses. Epigenetics further links pollution exposure to asthma severity, while firefighters exposed to wildfires show heightened inflammation [12, 13]. The aryl hydrocarbon receptor (AhR) has been linked to asthma. Particulate matter and air pollutants like polycyclic aromatic hydrocarbons activate AhR, leading to increased expression of Mucin-5AC (MUC5AC) and excessive mucus production [14]. Air pollution also impacts pollen by altering its morphology, protein content, and release. Pollen and fungal spores contain compounds that act as

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adjuvants, exacerbating allergic reactions. The surface of pollen is composed of pigments, waxes, lipids, aromatics, and proteins. Pollution elevates levels of pollen-associated lipid mediators (PALMs), particularly in areas with heavy traffic [15]. Studies show that PALMs from ragweed pollen extracts enhance Immunoglobulin E (IgE) production in Th2-primed B cells. Furthermore, bacteria and bacterial endotoxins have been associated with highly allergenic pollen. In vitro studies with bacterial isolates from hazel pollen triggered a concentration-dependent release of IL-8 and MCP-1, chemokines that recruit granulocytes. Ozone (O_3) worsens symptoms for those with pollen allergies during extended birch, grass, and ragweed seasons, which can last from mid-December to October [16–18].

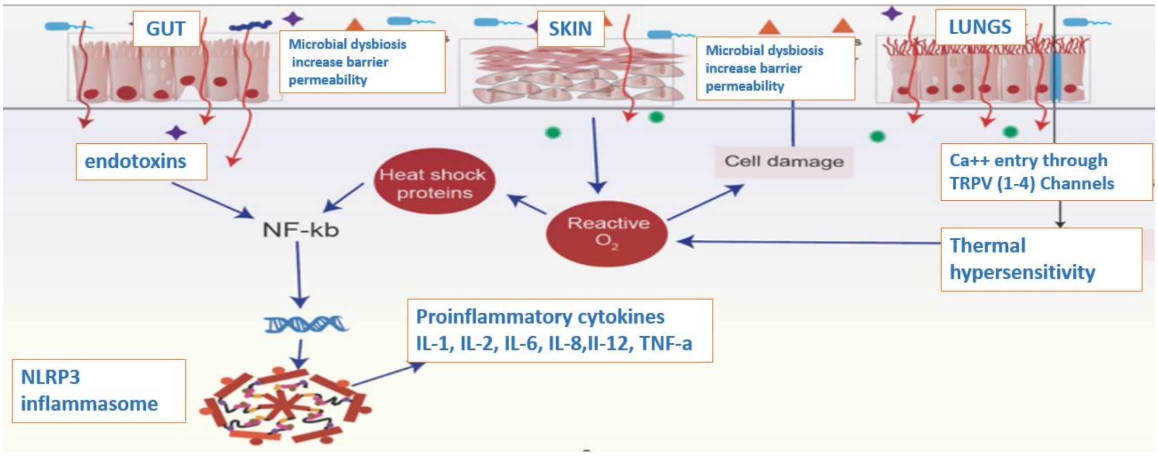

Figure 2. Epithelial barrier defect- Type V immunological reaction.

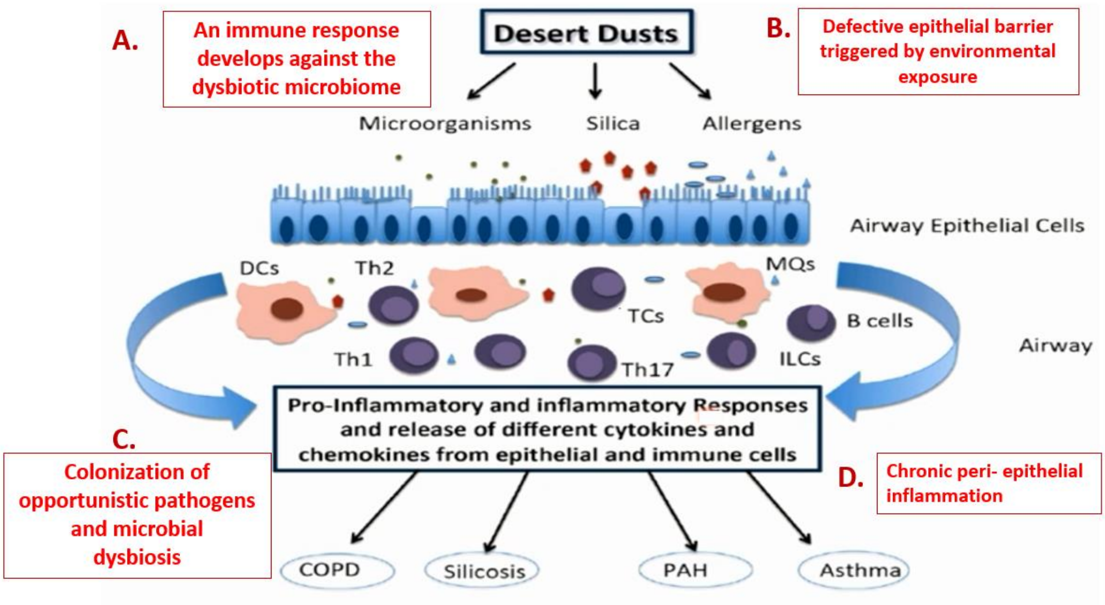

Figure 3. Climate change events and proposed mechanisms of epithelial barrier defects.

Environmental and lifestyle factors play a big role, both leading to poor development of the immune system and an increase in microbial dysbiosis. Immune dysregulation and epithelial barrier defect occur due to increased exposure not only due to the natural pollutants during wildfires, sand-and-dust storms (SDS), thunderstorms, and flooding but also due to synthetic air pollutants containing mixture of the chemicals. The common indoor pollutants are particulate matter (PM), various chemicals (VOCs from the cleaning supplies and building material) and allergens from pets, insects, dander, and mold. The outdoor pollutants are industrial, vehicle emissions (GHGs, PM, toxins), wildfires, SDS, and pollen. There is a link between climate change, increased prevalence, and severity of allergic asthma diseases

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by seven pathways and induces two immunological changes (Figure 4):

1. *Effect on the expression of β-expansin:* Environmental stressors like floods, salt, and drought can affect the expression of β-expansin, a key pollen allergen involved in cell wall modification and growth. Overexpression of β-expansin contributes to allergic reactions, particularly in grass pollen group 1 allergies [19].
2. *Dysregulation of immune system:* Air pollution has been linked to increased allergic sensitization and worsening of allergic diseases. A study by Liu et al. found that environmental factors affect ragweed pollen, causing longer and earlier pollen seasons, leading to a range of allergic diseases across different climate regions and times [20].

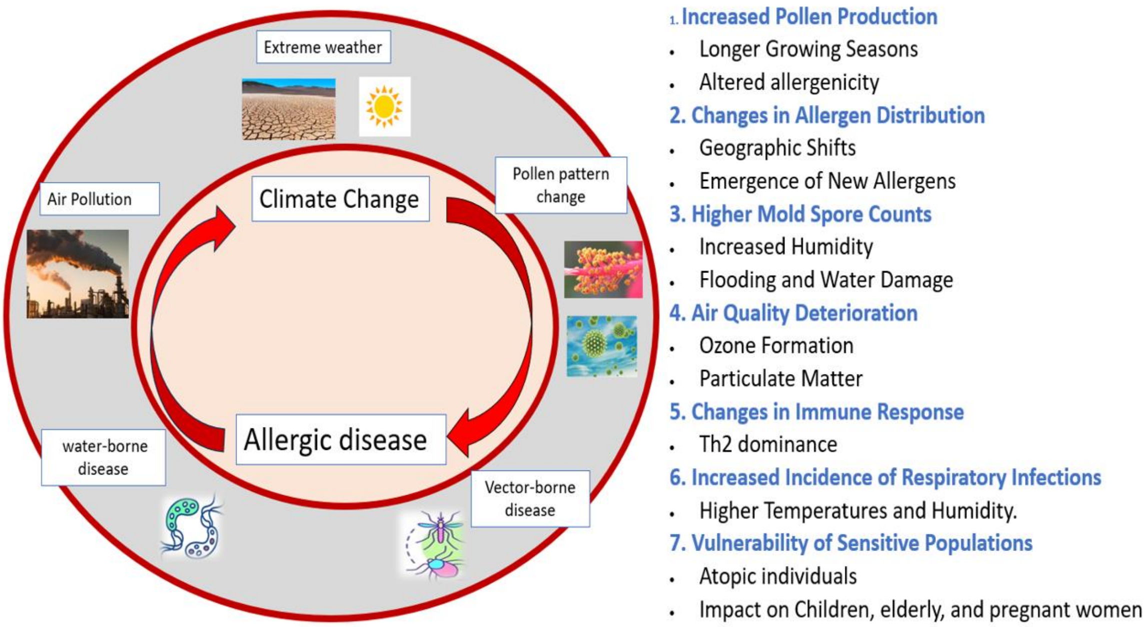

Figure 4. Inter-relation between climate change and allergic diseases.

EFFECT OF CLIMATE CHANGE AND AIRBORNE ALLERGEN [POLLEN FUNGUS, AND DUST MITE]

Climate change, particularly atmospheric CO_2 concentration and air temperature induces an impact on airborne allergens, such as pollen, fungal spores, and dust mites. Pollen season gets longer, pollen production, atmospheric concentration and allergenicity are increased (Figures 5 and 6) [21]. In North America, climate change is believed to account for roughly half of the extended pollen season. Rising temperatures and increased CO_2 levels enhance photosynthesis, promote plant growth, and drive higher pollen production. Additionally, air pollutants, such as ozone, nitrogen oxides, and particulate matter from traffic can amplify the production of allergenic proteins [22–25]. Evidence links climate change to increased allergic respiratory diseases, with higher pollen concentrations driving more atopic conditions. Pollen seasons vary by source, with tree pollen in spring and weed pollen in fall:

i. *Impact on seasonality:* Recent European studies highlight the complexity of climate change's impact on pollen seasonality, varying by species and location. The findings suggest that pollen seasons are becoming longer, with delayed start and end dates.

ii. *Impact on pollen & fungal spore production and atmospheric concentration:* Rising temperatures and CO_2 levels boost airborne allergen production. Studies show that increased CO_2 may double pollen production, with climate change potentially increasing emissions by up to 200% [26].

iii. *Impact on airborne allergen allergenicity and distribution:* pollen allergenicity is a temperature responsive trait. Pollen allergenicity increases with increasing temperature. Weed pollen will become more allergenic under condition of elevated CO_2, and drought stress.

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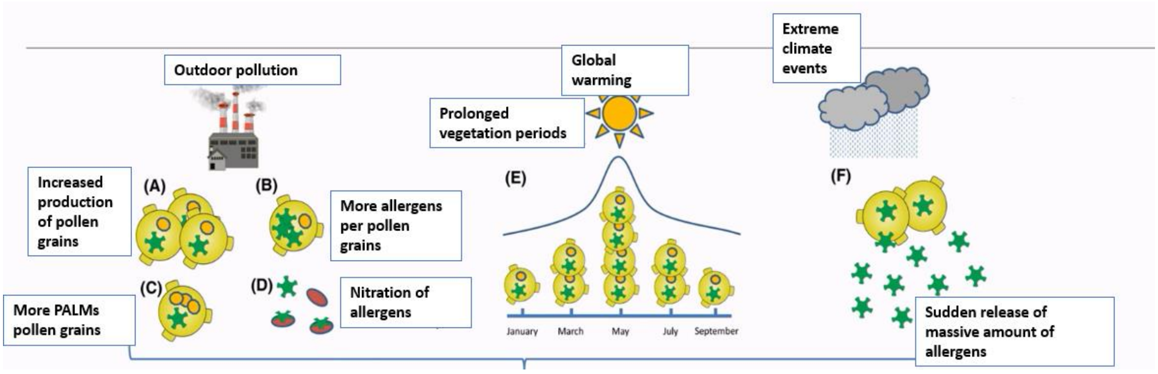

Figure 5. Effect of air pollution on pollen.

These changes, combined with the pro-inflammatory effects of pollen-associated lipid mediators (PALMs), are shaping the progression of allergic diseases [27, 28]. Pollen grains are swept up into the clouds at the formation of a thunderstorm (Figure 7). In the clouds, pollen grains are ruptured into small fragments less than 2.5 microns. These fragments are swept down to ground level with the help of dry, cold gust of the wind which precedes the rain. The fragments are small and can pass into the lower respiratory tract and induce bronchoconstriction.

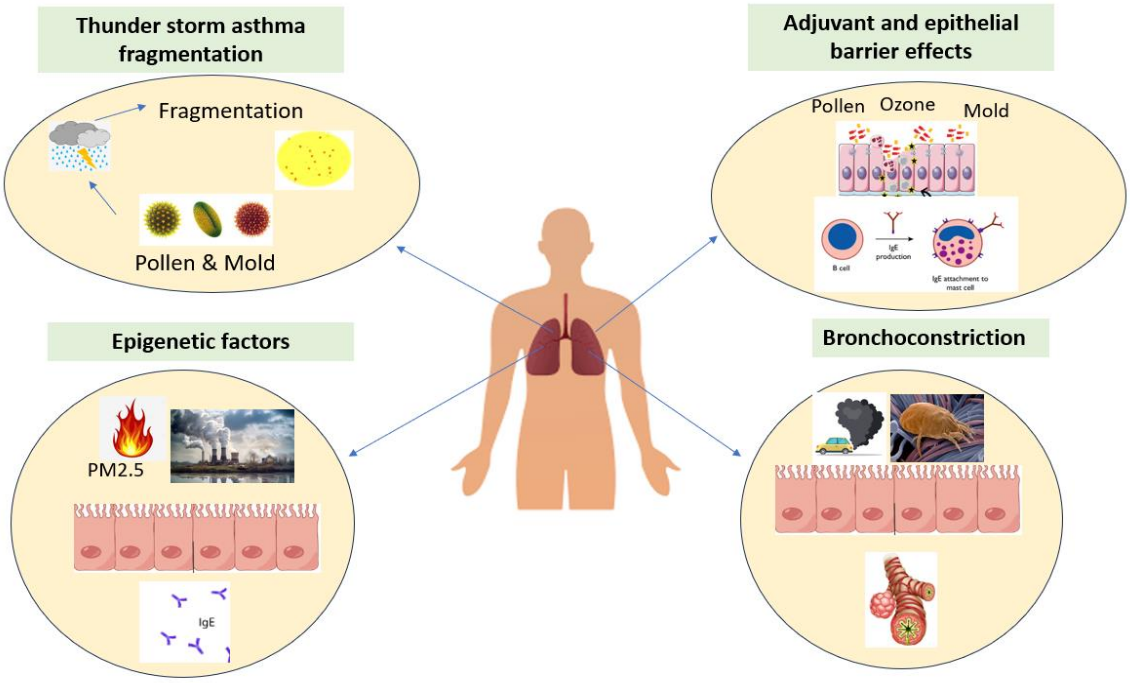

Figure 6. Effect of climate change and airborne allergens (pollens, fungus, and dust mite) including thunderstorms and epigenetic effects.

House Dust Mites

Climate change can enhance the growth and survival of house dust mites (HDMs) (Figure 8) due to the increased frequency and severity of hydrological natural disasters [29]. Increased humidity and dampness create ideal conditions for house dust mites (HDMs) to thrive. Sensitization to HDMs has become more prevalent over time, particularly in subtropical and tropical regions. Children with early sensitization to HDMs are at a higher risk of developing asthma, allergic rhinitis (AR), and impaired lung function. HDMs are commonly found in bedding, upholstered furniture, carpets, curtains, and blankets, and 40–85% of children with asthma and allergies in the Americas, Europe, Southeast Asia, and Australia are allergic to them [30, 31].

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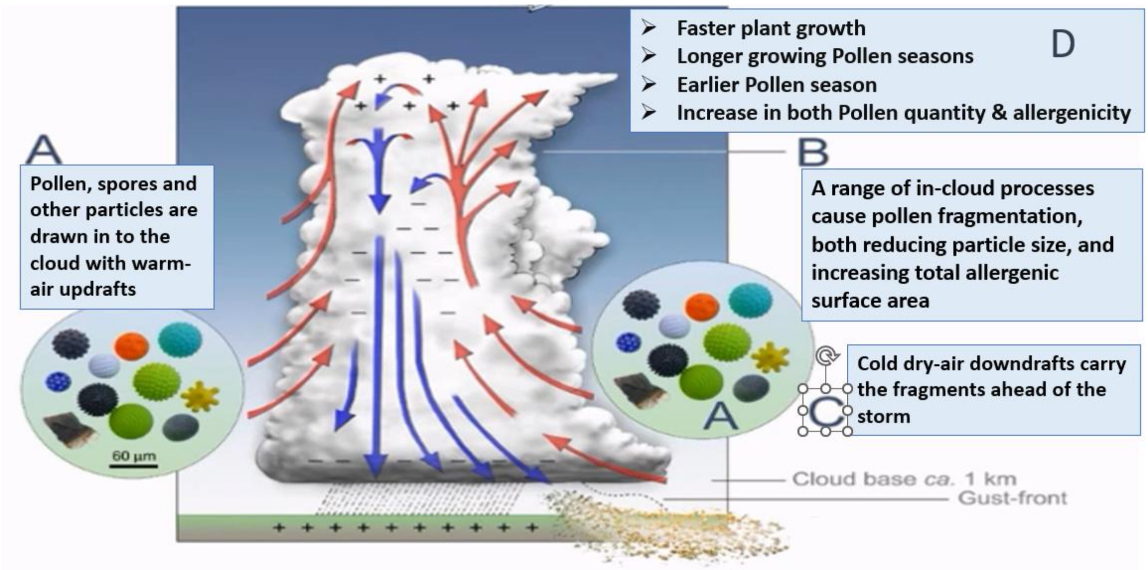

Figure 7. Impact of climate change on pollen seasons and thunderstorms.

Mite fecal particles are a significant source of dust allergens, though airborne mite allergens are typically detected only when dust is disturbed and settle quickly within minutes [32]. During gentle breathing, only 5–10% of larger particles (10–20 µm) reach the bronchial tree. Particle deposition in the lungs depends on their aerodynamic diameter, with fibers and flakes increasing the likelihood of lung deposition. In humid conditions, the Der p 1 allergen can transfer from fecal particles to smaller nearby particles. Inhalation of approximately 100 fecal particles per hour (each containing 0.2 ng of Der p 1) could result in 5–10 particles entering the lungs, assuming airborne levels of 20 ng/m^3. Most detectable Der p 1 particles are larger than 10 µm, but smaller particles under 5 µm can reach the lower airways, potentially contributing to bronchial hyperreactivity with chronic exposure [33].

HDMs produce potent protein allergens, proteases, and components like bacterial lipopolysaccharides (LPS) and fungal β-D-glucan, which can compromise epithelial barrier integrity, increasing allergen recognition and causing respiratory inflammation. For children sensitized to dust mite allergens, HDM exposure has been linked to asthma exacerbations [34, 35].

Figure 8. House dust mite.

CLIMATE CHANGE AND AIR POLLUTANTS

The rise in allergic and respiratory diseases has been driven by air pollution, climate change, and declining biodiversity. Globally, people are increasingly exposed to outdoor pollutants like PM2.5, PM10, suspended particulate matter (SPM), carbon monoxide (CO), ozone (O_3), nitrogen dioxide

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(NO_2), sulfur dioxide (SO_2), and nitric oxide (NO), as well as indoor pollutants, such as biomass smoke and tobacco [36, 37]. These pollutants aggravate respiratory conditions through four primary mechanisms: oxidative stress, airway remodeling, inflammation, and heightened respiratory sensitization. In atopic dermatitis (AD), pollutants contribute to skin damage, increased water loss, and alterations in the skin's microflora. Common indoor pollutants are listed in Table 1.

Table 1. Common sources of indoor pollutants Indoor pollutants.

1. Volatile organic compound
Use low VOC compounds as they induce nausea, headache, vomiting
2. Paints
Use low VOC paints
3. Cooking
Cooking with nonstick cookware may release toxic fumes at high temperatures. Use cast iron or stainless-steel pots and pans.
4. Chemicals for crafts
Markers, glues, other art supplies, polymer clays
5. Cleaning products
Which contains ammonia and chlorine. Clean with hot water, bake soda and microfiber cloth
6. Dry-clean
Contains perchloroethylene. First, the air dry clean for days before use.
7. Secondhand smoke and thirdhand smoke
The toxic residue that lingers in clothes and, cushions and carpets
8. Stove problem
Contain low level CO & NO2 so vent the stove with fan that blows outside.
9. Chimney & furnace gas
In a control heating & air conditioning system
10. Radon
When uranium naturally decays in soil, rocks, or water, it can enter our homes through cracks or holes in foundation walls or around pipes.
11. Air freshener
Induce headache and breathing difficult as they contain phthalates chemicals linked to child development and hormones so use natural mint or basil.
12. Formaldehyde furniture
Processed wood or drapes

Particulate Matter

Particulate matter (PM) comes from sources like traffic, industry, power plants, construction, waste burning, wildfires, SDS, and earthquakes. The primary components consist of sulfates, nitrates, ammonia, sodium chloride, black carbon, mineral dust, and water. They are defined by aero-dynamic diameter with PM2.5 and PM10. Particles more than 10 um in diameter are generally not respirable, the deleterious impacts of very coarse particles are by irritating the skin and eyes. PM10 particles <10 um can be inhaled and are associated with respiratory disease of the upper respiratory tract [38].

PM1 particles have a larger surface area compared to other fine particles, making them more likely to carry heavy metals, chemicals, and VOCs, which can cause more harm when inhaled. Ultrafine particles, even smaller than 0.1 microns – down to 0.003 microns – are considered the most dangerous. They can enter the lung tissue and pass into the bloodstream through pulmonary vessels. PM levels rise in winter due to increased car traffic and home heating [39, 40].

Ozone

The ozone (O_3) standard is 70 ppb (8-hour daily maximum). A review found that long-term O_3 exposure in children is linked to reduced FEV1. Ozone, a key component of smog, is produced when volatile organic compounds and nitrogen oxides react in the presence of sunlight, particularly in warmer

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regions during the summer. Ground-level O3 is a greenhouse gas and potent oxidant, associated with respiratory irritation, inflammation, oxidative stress, reduced lung function, and increased epithelial permeability. It stimulates an inflammatory reaction in the airways by raising the levels of proinflammatory mediators, chemokines, and neutrophils [41, 42].

Water Pollution

Detergents, allergens, and pollutants can harm the immune system by disrupting the epithelial barrier and triggering inflammation. Toxic metals, such as cadmium, chromium, copper, nickel, zinc, lead, and mercury have contaminated water sources. Additionally, exposure to arsenic is linked to higher levels of T helper 2 cell mediators (IL-4, IL-6, IL-13), increasing susceptibility to allergies and asthma [43–45].

Wildfires

Wildfires contribute to global warming and release PM, O_3, and CO. During the 2020 California wildfire, PM2.5 increased by 220%, O_3 by 20%, and CO by 151%. These pollutants are linked to higher levels of pro-inflammatory markers like CRP and IL-1B, which worsen asthma and allergy symptoms [46].

Floods

Increased dampness has led to more mold-spore growth, exposing residents to indoor allergens. Key factors driving the rise in allergic diseases include: (1) lifestyle changes, such as greater hygiene and less exposure to diverse plants, animals, and microbes, which reduce immune tolerance and increase microbial dysbiosis; and (2) higher exposure to environmental pollutants, which disrupt the epithelial barrier and immune function. These pollutants, both natural (GHGs, PM, pollen, mold) and synthetic, have worsened due to climate change events like wildfires, SDS, thunderstorms, and flooding [47].

Sand and Dust Storms (SDS)

It has become more common due to higher temperatures, droughts, and deforestation. Sand and dust storms (SDS) can worsen air pollution by increasing levels of particulate matter (PM), carbon dioxide (CO_2), and ozone (O_3). Desert dust is composed of clay, silicates, minerals, quartz, silicon dioxide, iron oxides, aluminum, titanium, magnesium, sodium, evaporated minerals, etc. It also contains pathogen, microorganism, fungi and spores. Desert storms are increasing in frequency and intensity due to climate change. Desert dust has the capacity to cause damage to the alveolar wall and bronchial epithelial cells through a direct physical effect (alarmins, innate and adaptive immunity), it influences oxidative stress and release of pro-inflammatory cytokines in respiratory epithelial cells. it causes damage to DNA and cause deterioration in the pulmonary function. Pollution due to desert dust has a clear effect on exacerbation and severity of allergic diseases.

Thunderstorm

Thunderstorm asthma (TA)- grass pollen concentration was extremely high (>100 grains cumm) during Melbourne, Australia thunderstorm 2016. The high humidity during this period caused pollen grains to rupture into sub pollen allergenic particles by osmotic shock which results in release of less than 5 microns allergenic components in the environment inducing exacerbation of asthma in lower airways.

Global warming increases evaporation, raising air moisture levels. During thunderstorms, warm updrafts lift pollen and mold spores, causing osmotic shock and breaking them into smaller, more allergenic fragments. These smaller fragments are easily inhaled, triggering bronchial hyperresponsiveness and worsening asthma and allergic rhinitis. Even non-asthmatic individuals with seasonal rhinitis are at higher risk of asthma attacks during such storms [47–49].

Climate-change-driven thunderstorm asthma events cause increased release of allergenic mediators. Warm updrafts lift pollen to cloud bases, where it is fragmented into smaller particles by electrical

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forces or moisture absorption. For example, rye grass pollen breaks into fine fragments that bypass nasal filtration and are inhaled into the lungs. High humidity in thunderstorms further fragments pollen and concentrates aeroallergens, triggering asthma symptoms even in non-asthmatic individuals and worsening symptoms in those with asthma, as seen in Australia's 2016 epidemic [50, 51].

Heat Stress

It refers to prolonged periods of ambient temperatures exceeding the average, with temperatures above 105°F (41°C) being harmful to human health. Heat stress contributes to asthma and allergies through several mechanisms. During heat stress, blood flow is shifted from central organs to the periphery, increasing membrane fluidity and aiding in heat dissipation. Most studies on heat stress in humans have focused on exertional heat stress [52]. Studies in humans and animals during heat stress show that high temperatures disrupt intestinal integrity, leading to increased intestinal permeability (leaky gut). Heat causes the breakdown of key proteins like claudins, occludins, and junctional adhesion molecules in tight junctions, allowing antigens, such as lipopolysaccharides (LPS) to enter the bloodstream. LPS activates toll-like receptor 4 (TLR4), triggering the nuclear factor κB (NF-κB) pathway. Additionally, reactive oxygen species (ROS) released during heat stress activate NF-κB, which then triggers the inflammasome NLRP3 and releases pro-inflammatory mediators like PGE2, TNF-α, IL-1β, IL-6, IFN-γ, and CRP [53–56].

Environmental diversity plays a key role in allergen exposure and immune development in children. Urban areas with high pollution often trigger a T-helper (Th) 2 immune response, promoting allergies. On the other hand, rural areas with greater microbial diversity may protect against allergies by modulating the immune system. This supports the "hygiene hypothesis," which suggests that early exposure can shift immune response, reducing the risk of allergies. For instance, children living near green spaces, with more actinobacteria diversity, show less allergic sensitization. Children living near coasts may be exposed to higher levels of mold, while those in industrial areas are more prone to chemical pollutants. Moreover, increased rainfall and humidity are associated with a rise in bacterial and viral respiratory infections among children. These differences emphasize the importance of tailored approaches to managing and preventing allergies and respiratory infections in different regions [57–62].

EFFECTIVE STRATEGIES FOR MANAGING AND CONTROLLING CLIMATE CHANGE

Climate change affects the health of human beings, animals, plants, and environmental health which are interconnected to each other and need specific strategies at individual, committee, national and global level [63]. Healthcare professionals should raise awareness about the risks of climate change and advocate for policy reforms. Below are some effective approaches to tackling climate change:

Mitigation

- *Reducing Greenhouse Gas Emissions:* Transitioning to renewable energy sources like solar, wind, and hydro, while enhancing energy efficiency in industries, buildings, and transportation.
- *Carbon Sequestration:* Strengthening natural processes like reforestation and soil management, along with developing technologies to capture and store carbon dioxide.
- *Sustainable Agriculture:* Adopting farming practices that minimize emissions, including no-till farming, crop rotation, and agroforestry.

Adaptation

- *Infrastructure Resilience:* Strengthening buildings, roads, and other infrastructure to better endure extreme weather events.
- *Water Management:* Developing systems to conserve water and manage supplies more efficiently, especially in drought-prone areas.
- *Disaster Preparedness:* Developing early warning systems and emergency response strategies for communities at risk from climate-related impacts.

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Policy and Regulation
- *International Agreements:* Participating in global efforts like the Paris Agreement to set emission reduction targets.
- *Local and National Policies:* Implementing laws and regulations that promote sustainability, such as carbon pricing or incentives for green technology.
- *Public Engagement:* Increasing awareness and educating communities about the effects of climate change and the need for sustainability.

Research and Innovation
- *Investing in Technology:* Supporting research into new technologies that can help reduce emissions or improve energy efficiency.
- *Climate Science:* Funding studies to better understand climate impacts and develop predictive models for future changes.

Community Involvement
- *Grassroots Initiatives:* Promoting local efforts like community gardens, clean-up drives, and renewable energy projects.
- *Partnerships:* Working with businesses, NGOs, and other organizations to exchange knowledge and resources.

Economic Transformation
- *Green Economy:* Encouraging the growth of industries and job opportunities in renewable energy, sustainable agriculture, and conservation.
- *Investment in Sustainability:* Allocating funds to projects that foster environmental sustainability and lower carbon footprints.

Addressing climate change requires collective efforts from all parts of society, including governments, businesses, and individuals. While the challenge is large, with proactive strategies and creative solutions, a more sustainable future is achievable (Table 2).

Table 2. Actions and Long-term Objectives for Environmental and Health Improvement.

Short Term Interventions	Long Term Goals
Urban planning • Reduction in fossil fuels • Decrease in greenhouse gases • Traffic management • Green space • Location of residential areas and key community buildings like schools, hospitals, etc. • Public transport infrastructure	*One health approaches* • Regulation for air quality • E-mobility incentives • Investment in innovative resources and technologies protecting the environment • Providing safe housing with improved weatherization, reducing dampness, improving ventilation
Educational programs on clean environment • Healthcare providers • Patients & families • General public	*Improving diets and agricultural practices* • Access to healthy foods • Sustainable agricultural practices

To prevent severe climate change impacts, we need systemic changes and collective action from healthcare professionals, communities, and individuals. Personal choices, such as using public transportation, embracing a plant-based diet, and improving home energy efficiency, can have a significant impact. Local efforts, including collaboration with community organizations and creating climate-resilient neighborhoods, strengthen social bonds for better climate adaptation. In healthcare, advocating for reduced hospital emissions, advising patients on minimizing air pollution exposure, and addressing knowledge gaps through research are critical steps. Ultimately, national and international policy changes and cooperation are vital for mitigating climate change. According to the

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Intergovernmental Panel on Climate Change, reducing our reliance on fossil fuels and transitioning to renewable energy sources is key to limiting the temperature rise to below 1.5°C [64, 65].

CONCLUSIONS

Global warming and climate change have a major impact on both the environment and human health, especially concerning allergic and atopic diseases. We have discussed how climate change is connected to the growing frequency and intensity of asthma and other allergic conditions. Rising air pollution can influence pollen production on local and regional levels. Vulnerable populations, such as the elderly, children, and pregnant women, are particularly at risk. It's crucial to take action to minimize the health impacts of climate change. Proactive steps like reducing carbon emissions, developing strategies for managing allergies in a changing climate, and raising awareness about the health risks of climate change are essential. Supporting policies that mitigate climate change's effects is key.

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