Review

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Climate Change and the Increasing Burden of Allergies in Children

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Abstract

Allergic diseases are increasing globally, particularly among children, who are highly vulnerable due to critical windows of immune development. This review examines climate change as a key environmental determinant driving the rising burden of pediatric allergic diseases, including asthma, allergic rhinitis (AR), atopic dermatitis (AD), and food allergy (FA). Climate change influences disease risk through interconnected pathways, such as increased air pollution, altered aeroallergen patterns, and more frequent extreme weather events. Elevated carbon dioxide (CO_2) levels and rising temperatures prolong pollen seasons and enhance allergenicity, while pollutants such as ozone (O_3) and particulate matter (PM) exacerbate airway inflammation and immune dysregulation. Emerging evidence emphasizes the role of early-life exposure, particularly during prenatal and early postnatal periods, when environmental insults can induce long-term effects via epigenetic modifications and immune reprogramming. These mechanisms may increase susceptibility to allergic sensitization and subsequent disease development. Epidemiological studies consistently link exposure to air pollution, including $PM_{2.5}$ (PM with aerodynamic diameter < 2.5 μm) and nitrogen dioxide (NO_2), with increased risk of allergic diseases in children. Additionally, climate change-related events such as wildfires, sand and dust storms, and thunderstorms further elevate exposure to allergens and pollutants, contributing to acute exacerbations and disease progression. Addressing this growing public health challenge requires integrated mitigation strategies to reduce greenhouse gas (GHG) emissions and improve air quality, alongside adaptive interventions to enhance resilience and reduce exposure. Understanding these mechanisms is essential for developing targeted prevention strategies and protecting child health in a changing climate.

Keywords: climate change; allergic diseases; children; asthma; air pollution; particulate matter; ozone; aeroallergens; epigenetics; early-life exposure

1. Introduction

Allergic diseases have demonstrated an increasing global prevalence, emerging as a major and growing public health concern, with children being recognized as a particularly vulnerable population [1–4]. Their etiology is multifactorial, with complex interactions between genetic predisposition and environmental exposures that ultimately result in the disease's phenotypic expression [5]. Among environmental determinants, climate change has profound impacts on the incidence, severity, and prevalence of allergic diseases, such as asthma, allergic rhinitis (AR), atopic dermatitis (AD), and food allergy (FA) [6]. Children are disproportionately affected, as environmental exposures frequently coincide with critical windows of development, including

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prenatal stages, early childhood, and adolescence, during which physiological systems are particularly sensitive, leading to potential lifelong adverse effects [7].

One of the key consequences of climate change is the increase in carbon dioxide (CO_2) levels and global temperatures [8]. Intergovernmental Panel on Climate Change (IPCC) assessments indicate that the mean global surface temperature has increased by approximately 1.19 °C above pre-industrial levels during 2014–2023, with the vast majority of this warming attributed to anthropogenic greenhouse gas (GHG) emissions and other human activities [9]. Rising CO_2 levels and temperatures prolong and intensify pollen seasons while enhancing pollen allergenicity, contributing to increased airway inflammation and a higher prevalence of respiratory diseases such as asthma and AR [8]. In addition, pollen acts not only as an allergen carrier but also releases bioactive lipid mediators that promote inflammation and modulate immune responses in allergic diseases [10]. Moreover, climate change increases ambient air pollution by raising pollutant concentrations and altering their spatial distribution. This leads to higher levels of ozone (O_3) and particulate matter (PM), which, in turn, worsen health outcomes [8].

Beyond increasing exposure, these climate- and pollution-driven stressors may also influence susceptibility at the molecular level through epigenetic reprogramming of immune pathways. Epigenetic regulation is increasingly recognized as a critical interface between environmental determinants and the pathogenesis of allergic and autoinflammatory diseases. Exposure to extreme temperatures, pollen, and polycyclic aromatic hydrocarbons (PAHs) can induce epigenetic changes (including DNA- and RNA-level modifications) that modulate key allergic pathways and promote inflammation through gene–environment interactions [11].

This review aims to comprehensively examine how climate change contributes to the increasing burden of allergic diseases in children by analyzing environmental drivers, critical developmental windows, and underlying biological and epigenetic mechanisms, while highlighting implications for prevention and public health action.

2. Climate Change and Allergic Diseases

Climate change is increasingly recognized as a systemic driver of allergic disease through its capacity to alter ecosystems and reshape environmental exposures that influence immune tolerance [12]. The interconnected pathways linking climate change, air pollution, aeroallergens, and immune dysregulation in the development of allergic diseases in children are summarized in Figure 1. Climate-related events, including thunderstorms, sand and dust storms, wildfires, and heat waves, modify air quality, allergen distribution, and biodiversity patterns, thereby increasing human exposure to pollutants, GHGs, pollen, and other sensitizing agents. These environmental pressures interact with biological susceptibility factors, such as epithelial barrier dysfunction, impaired immune maturation, and microbial dysbiosis, and disproportionately affect vulnerable populations, such as infants and children [13]. Infants and young children, for instance, are particularly susceptible to air pollution because their organs are still maturing and they inhale a greater volume of air per unit of body weight than adults [8]. The convergence of environmental exposures, host susceptibility, and social vulnerability increases the likelihood of allergic sensitization and progression to allergic disease [13].

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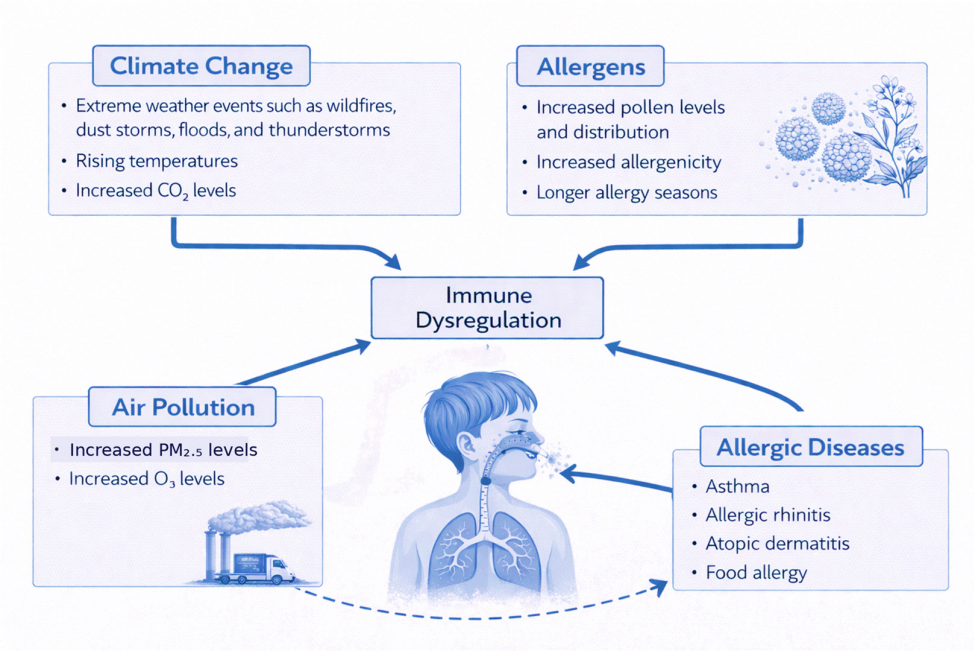

Figure 1. The interconnected pathways linking climate change, air pollution, aeroallergens, and immune dysregulation in the development of allergic diseases in children. Created with the assistance of ChatGPT (OpenAI), under the authors' supervision.

Thus, climate change should be regarded not only as an amplifier of allergen burden but also as a modifier of immune regulatory pathways through ecological disruption and microbiome alteration, eventually increasing susceptibility to allergic sensitization in children. Table 1 provides an integrated overview of the key climate change drivers, associated environmental exposures, underlying biological mechanisms, and their corresponding pediatric allergic health outcomes.

Table 1. Climate drivers, environmental exposures, biological mechanisms, and pediatric allergic outcomes.

Climate driver	Environmental Exposure	Biological Mechanisms	Pediatric Allergic Outcomes
Rising CO_2 and global temperature	Increased pollen production, longer pollen seasons, enhanced allergenicity [14,15]	Epithelial activation (IL-25, IL-33, TSLP); Th2 polarization; IgE sensitization; mast cell and eosinophil activation [16]	Asthma, AR [2,17,18]
Rising temperature & photochemical reactions	Ground-level O_3	Oxidative stress; airway epithelial injury; altered lung development; airway remodeling [19]	Asthma, AR, AD, conjunctivitis [20]
Fossil fuel combustion & industrial emissions	$PM_{2.5}$, PM_{10}, NO_2, SO_2	Oxidative stress; epithelial barrier dysfunction; Th2 skewing; immune dysregulation; epigenetic remodeling [5,21]	Asthma, AR, AD, FA [22–29]
Climate-amplified wildfires	Smoke-related $PM_{2.5}$, organic compounds	Oxidative stress; airway inflammation; impairment of the	Asthma exacerbations [31,32]

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		respiratory epithelial barrier [30]	
Climate change–related thunderstorms	Thunderstorm-induced fragmentation and dispersion of pollen and fungal spores; sudden spikes in respirable allergenic particles [33]	Allergen fragmentation → enhanced lower-airway penetration; interaction with underlying atopy; IgE-mediated mast cell activation; acute bronchospasm [33]	Acute asthma exacerbations/epidemic thunderstorm asthma events [34–36]
Drought & land degradation	Sand and dust storms (PM_{10}, bioaerosols)	Airway inflammation; increased FeNO; bronchial obstruction [37]	Asthma exacerbations [38,39]

CO_2: carbon dioxide, IL: interleukin, IgE: immunoglobulin E, O_3: ozone, $PM_{2.5}$: particulate matter with aerodynamic diameter < 2.5 μm, PM_{10}: particulate matter with aerodynamic diameter < 10 μm, NO_2: nitrogen dioxide, SO_2: sulfur dioxide, AR: allergic rhinitis, AD: atopic dermatitis, FA: food allergy, FeNO: fractional exhaled nitric oxide.

3. Prenatal Exposure and Epigenetics

The prenatal period represents a critical window of vulnerability [40]. During this stage, rapid immune differentiation and organ development occur, rendering the fetus particularly sensitive to environmental insults. Fetal immune development begins in utero, and environmental exposures during pregnancy can alter immune maturation through oxidative stress, systemic and placental inflammation, epigenetic modifications, and disruption of cytokine signaling pathways [41]. These early perturbations may influence long-term immune function by modifying developmental programming rather than inducing transient inflammatory responses alone.

Evidence indicates that air pollution exposure during pregnancy is associated with immune dysregulation, including alterations in T-cell polarization, Th2-skewing pathways, and cytokine profiles, with epigenetic mechanisms proposed as key mediators linking exposure to adverse maternal and neonatal outcomes [42]. At the molecular level, increases in ambient pollutants during pregnancy were associated with altered DNA methylation in key cytokine genes, including interleukin 4 (IL4), IL10, and IFNG, as well as changes in T-helper cell subset distributions. Given the central role of these cytokines in Th1/Th2 regulation, such epigenetic alterations may influence immune polarization and potentially contribute to downstream allergic susceptibility [43].

Beyond DNA methylation, prenatal $PM_{2.5}$ (particulate matter with aerodynamic diameter < 2.5 μm) exposure is associated not only with cytokine dysregulation but also with epigenetic changes in immune cells, including alterations in histone post-translational modifications. These chromatin-level modifications regulate gene transcription by altering chromatin accessibility, suggesting that pollutant exposure may induce sustained immune reprogramming through epigenetic remodeling rather than short-lived inflammatory activation [44].

Importantly, these molecular alterations appear to extend beyond isolated immune genes. A large cohort study has examined prenatal pollutant exposure (nitrogen dioxide - NO_2, O_3, and PM) in relation to DNA methylation patterns across asthma- and allergy-relevant genes and biospecimens, including placenta, cord blood, buccal, nasal mucosa, and lavage. It demonstrated that exposure-associated epigenetic variation may persist after birth and act upstream of later allergic phenotypes [45].

Epidemiological findings strongly support this mechanistic model. A meta-analysis of 38 studies reported that prenatal exposure to air pollutants, particularly $PM_{2.5}$ and NO_2, was associated with increased risks of several childhood allergic diseases. For every 10 $\mu g/m^3$ increase, risks rose by 34% for AD (odds ratio - OR = 1.34; 95% Confidence Interval - 95% CI: 1.10–1.63), 11% for asthma with $PM_{2.5}$ (OR = 1.11; 95% CI: 1.05–1.18), and 36% for AR with $PM_{2.5}$ (OR = 1.36; 95% CI: 1.17–1.58) [46].

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Similarly, a 14-year birth cohort study of 1439 mother–infant pairs demonstrated that higher prenatal exposure to NO_2, carbon monoxide (CO), and sulfur dioxide (SO_2) was associated with significantly elevated risks of AD and AR, with high-exposure clusters showing approximately threefold increased risk compared with lower-exposure groups [47]. A Danish cohort study found that higher prenatal exposure to NO_2, $PM_{2.5}$, and PM_{10} (particulate matter with aerodynamic diameter < 10 μm) was associated with altered early immune responses and increased risk of allergic sensitization (OR = 2.68; 95% CI: 1.58–4.62) and AR (OR = 2.63; 95% CI: 1.18–5.81) by age 6 years. Prenatal pollution-related inflammatory alterations in infancy were also linked to a higher risk of asthma at the age of 6 years (OR = 1.80; 95% CI: 1.18–2.76) [48]. Consistent associations were observed in a retrospective study of 3,177 preschool children, where higher prenatal and first-year NO_2 exposure was significantly associated with increased odds of asthma, AR, pneumonia, and AD. NO_2 exposure during pregnancy increased the odds of AR by approximately 59–68%, and exposure during the first year of life increased the odds by 57–73%, whereas SO_2 and PM_{10} showed no consistent associations [49].

Conversely, in the Pélagie mother–child cohort (n = 1,322 at 6 years; n = 1,118 at 12 years), median prenatal exposure levels were 15 μg/m^3 for $PM_{2.5}$ and 18 μg/m^3 for NO_2, and overall analyses did not show statistically significant associations with asthma, AR, or AD. However, at 12 years, there was a tendency toward increased asthma risk per interquartile range increase in $PM_{2.5}$ (OR =1.38; 95% CI: 0.98–1.93) and NO_2 (OR = 1.25; 95% CI: 0.98–1.59), and at 6 years, AD risk was significantly elevated in urban areas for $PM_{2.5}$ (OR = 1.49; 95% CI: 1.03–2.14) and NO_2 (OR = 1.40; 95% CI: 1.08–1.82) [50].

Taken together, the convergence of epigenetic evidence and longitudinal epidemiological data supports a coherent model in which prenatal air pollution exposure induces molecular and immune reprogramming during fetal development, thereby increasing susceptibility to allergic sensitization and allergic disease in childhood.

4. Early-Life Exposure

Growing evidence indicates that early life represents a critical window of susceptibility during which environmental exposures can shape immune maturation and long-term allergic risk. Early postnatal exposure to climate-sensitive pollutants has been increasingly linked to the development of allergic diseases in children. Recent studies suggest that these exposures may exert timing-specific effects, with certain developmental periods demonstrating heightened vulnerability to allergic outcomes [28,51,52].

A large multicenter study demonstrated that early infancy (30–38 weeks postnatal age; 7.5–9.5 months) constitutes a critical susceptibility window during which O_3 exposure increases the risk of AR in preschool children, following a threshold-free J-shaped exposure–response relationship [51]. A population-based cohort study reported that early-life exposure to NO_2 and O_3 increased the risk of childhood asthma by about 17% and AD by about 7% [27]. In a retrospective cohort of 182,387 children, wildfire smoke exposure during early postnatal life was associated with earlier use of upper respiratory medications, with each additional weekly smoke-day increasing risk by 9.4% in the first 12 weeks (hazardous ratio - HR = 1.094; 95% CI: 1.005–1.191) and 10.8% in weeks 13–24 (HR = 1.108; 95% CI: 1.016–1.209) [52]. In a cohort of 2,598 preschool children aged 3–6 years, 14.9% had doctor-diagnosed FA. Early-life exposure to both indoor and outdoor air pollution was significantly associated with FA. Prenatal exposure to mold or dampness nearly doubled the odds of FA (adjusted OR = 1.93; 95% CI: 1.35–2.75), while traffic-related NO_2 exposure increased risk both prenatally (OR = 1.24; 95% CI: 1.00–1.54) and postnatally (OR = 1.38; 95% CI: 1.03–1.85) per interquartile range increase [28].

5. Air Pollution

Climate change and air pollution are closely interrelated, with climate change significantly influencing air pollutant levels and overall air quality [26]. Air pollution consists of a complex

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mixture of gaseous pollutants and PM, including O_3, nitrogen oxides (NOx), CO, volatile organic compounds (VOCs), and heavy metals, which vary in their sources and biological effects. Human activities such as fossil fuel combustion, deforestation, land-use modification, livestock production, agricultural fertilization, and industrial processes have significantly elevated GHG emissions and enhanced the formation of ground-level O_3 [19].

Outdoor air pollution originates from multiple sources, including industrial and vehicular emissions, such as GHGs, PM, and other toxic pollutants, as well as natural events like wildfires and sand and dust storms [53]. Household air pollution is mainly generated from burning fuels such as wood, charcoal, coal, and kerosene for cooking or heating, and tobacco smoke [54,55]. About 2.1 billion people globally rely on cooking with open fires and inefficient stoves, which exposes them to significant air pollution [54].

Approximately 93% of children worldwide under the age of 15 years (around 1.8 billion) are exposed to air pollution, placing them at risk of harmful health effects during critical stages of development [56]. Air pollutants can affect children's respiratory system by inducing epithelial injury, oxidative stress, inflammation, and immune dysregulation, contributing to airway diseases such as asthma [57]. Numerous studies, including meta-analyses, have reported links between air pollutants and allergic diseases in children [23,24,46].

5.1. Ozone

Ground-level O_3 represents a major climate-sensitive air contaminant. Its concentrations tend to rise to hazardous levels during hot, sunny conditions [58]. Exposure to O_3 during critical periods of lung development can induce structural and morphological alterations, including reduced bronchial branching, decreased alveolar number, disruption of smooth muscle bundle orientation, and hyperinnervation of the pulmonary epithelium. This exposure during early life may increase susceptibility to allergic diseases later in life, including asthma, AD, and AR [19].

A multi-city study including 177,888 children found that long-term O_3 exposure was associated with significantly increased odds of allergic diseases, with each interquartile increase in O_3 concentration linked to 31% higher odds of asthma, 25% higher odds of AR, 19% higher odds of AD, and 28% higher odds of conjunctivitis [20].

5.2. Particular Matter

PM is a key air pollutant and known for its harmful effects on human health; PM is further classified by aerodynamic equivalent diameter into coarse particles (PM_{10}), fine particles ($PM_{2.5}$), and ultrafine particles [59]. PM, especially $PM_{2.5}$ and ultrafine particles, contribute to the pathophysiology of allergic disease by inducing oxidative stress and epithelial barrier dysfunction in the airways and skin, enhancing allergen penetration, and acting as immune adjuvants that promote Th2-skewed inflammation, IgE sensitization, and eosinophilic responses. It also alters the airway microbiome and epigenetic regulation during critical developmental windows [60].

A recent meta-analysis showed that exposure to PM was significantly associated with increased asthma morbidity in children and adolescents, with a pooled OR of 1.04 (CI: 1.03–1.06) for PM_{10} and 1.05 (95% CI: 1.04–1.07) for $PM_{2.5}$ across 47 observational studies including 417,874 participants. PM exposure was also linked to wheezing, with a pooled OR of 1.06 (95% CI: 1.05–1.07) for PM_{10} and a stronger association for $PM_{2.5}$ (OR = 1.15, 95% CI: 1.10–1.20), indicating greater toxicity of finer particles [23]. Another recent meta-analysis included 21 studies with 120,454 children and found that each interquartile range increase in $PM_{2.5}$ was associated with a 20% higher risk of FA (OR = 1.20; 95% CI 1.01–1.42), while mold or damp exposure increased risk by 53% (OR = 1.53; 95% CI 1.21–1.93) [61]. Likewise, a meta-analysis of 21 studies and 217,396 children demonstrated that each 10 $\mu g/m^3$ increase in $PM_{2.5}$ was associated with a 9% increase in the risk of AR in children (OR = 1.09, 95% CI: 1.01–1.17), while each 10 $\mu g/m^3$ increase in PM_{10} was associated with a 6% increase in risk (OR = 1.06, 95% CI: 1.02–1.11) [24].

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A cross-sectional study showed that $PM_{2.5}$ exposure was positively associated with AR in adolescents, with each 1 μg/m³ increase in annual mean $PM_{2.5}$ raising the risk by 11% (OR = 1.110, 95% CI: 1.054–1.169). The overall prevalence of AR in the study population was 17.5% [62]. A population-based study identified that an interquartile-range increase in PM_{10} was associated with higher odds of AD in children, including increased AD symptoms (OR = 1.06, 95% CI: 1.01–1.12) and physician-diagnosed AD (OR = 1.07, 95% CI: 1.01–1.13) [25]. In addition, indoor $PM_{2.5}$ exposure was linked to worsening AD symptoms in children, with each 10 μg/m³ increase in $PM_{2.5}$ raising AD symptom scores by 16.5% in spring and 12.6% in winter. The harmful effects were stronger in children with inhalant allergen sensitization and severe disease, reaching a 15.7% increase in symptoms per 10 μg/m³ rise in $PM_{2.5}$ [63].

6. Effect of Climate Change on Aeroallergens

Evidence has shown that both rising CO_2, O_3, and NOx concentrations and global temperatures, as a result of climate change, cause alterations in aeroallergen patterns, particularly pollen, marked by longer seasons, wider spread, higher levels, and increased allergenicity [14,15]. These climate-driven changes in pollen patterns may contribute to more frequent asthma exacerbations and a greater burden of allergic diseases [8,64].

A meta-analysis of 155 included studies, mainly conducted in Europe, examined 11 major allergenic pollen taxa, including Poaceae, Betula, Olea, Cupressaceae, Fraxinus, and Platanus. It found an overall increase in pollen production between 1974 and 2020, with positive trends reported in 100% of Alnus studies, 92% of Fraxinus, 82% of Olea and Betula, 78% of Platanus, 76% of Cupressaceae, and 72% of Poaceae. Conversely, decreases were observed in 70% of Artemisia and 85% of Urticaceae studies, with higher temperatures and rainfall showing species-dependent associations with increased pollen production [65]. Paudel et al. found that climate-related increases in temperature and precipitation were linked to significant changes in pollen activity, including an increase in tree pollen exposure duration by approximately 0.47 weeks and an increase in tree pollen season length by about 0.38 weeks between 2002 and 2019, along with a significant rise in the number of active pollen weeks, in San Francisco Bay Area (California, USA). Peak pollen concentrations were positively associated with temperature changes at time lags of 0–1, 0–4, and 0–12 weeks and with precipitation changes at time lags of 0–4, 0–12, and 0–24 weeks [14]. In a study of 11,895 participants (adults and children) in northern Taiwan, 7.8% demonstrated pollen-specific IgE sensitization, with the annual prevalence increasing significantly from 6.7% in 2014 to 9.9% in 2020. They also developed sensitization to several pollens (e.g., Bermuda grass, ragweed mix, black willow), showing strong positive correlations with rising winter temperatures (r_s – Spearman's rank correlation coefficient = 0.821–0.893, $p \leq 0.023$) and inverse correlations with winter rainfall [66].

Pollen allergens penetrate epithelial barriers and stimulate epithelial cells to release cytokines such as IL-25, IL-33, and thymic stromal lymphopoietin (TSLP), which activate dendritic cells and innate lymphoid cells, promoting Th2 differentiation and the production of IL-4, IL-5, IL-9, and IL-13. This Th2-skewed immune response induces allergen-specific IgE production and sensitization of mast cells and basophils, leading to mediator release, eosinophilic inflammation, and clinical allergic disease [16].

The meta-analysis by Shrestha et al. found that increases in ambient pollen concentrations were significantly associated with higher pediatric asthma hospitalizations, with each 10 grains/m³ increase in grass pollen linked to approximately a 3% increase in asthma admissions (OR = 1.03; 95% CI: 1.01–1.04). Similarly, birch pollen increases of 10 grains/m³ were linked to a significant rise in childhood asthma admissions, supporting a consistent relationship between elevated pollen exposure and pediatric asthma exacerbations [17]. In a longitudinal study of 8,295 children and adolescents in Seoul, the duration of the tree pollen season increased from 98 days to 140 days, coinciding with rising temperatures. Over the same period, sensitization rates to major tree pollens (oak, hazel, and alder) increased by approximately 0.28% per year, demonstrating an association between prolonged pollen exposure and increasing allergic sensitization in the pediatric population

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[18]. In another longitudinal study of children with asthma, higher pollen severity in the preceding week was significantly associated with poorer asthma control, with each interquartile range increase in pollen linked to a 0.35-point increase in asthma control score ($\beta = 0.35$, $p < 0.05$). This association remained significant after adjustment for $PM_{2.5}$ and O_3 exposure, with an even stronger effect size ($\beta = 0.44$, $p < 0.05$), indicating that pollen independently worsened asthma control in children [67].

7. Extreme Weather Conditions

Climate change directly leads to higher temperatures, including more frequent heatwaves and rapid temperature fluctuations, along with shifts in rainfall patterns that contribute to increased wildfires, floods, drought, sand and dust storms, and thunderstorms [64,68].

7.1. Wildfires

Wildfire smoke is composed of numerous chemical components and produces multiple air pollutants, including PM, O_3, and various organic compounds [64]. The respiratory effects of wildfire smoke include oxidative stress, immune dysregulation with heightened inflammation, and impairment of the respiratory epithelial barrier [30].

A recent scoping review of 44 studies found that wildfire smoke exposure was consistently associated with worsening pediatric asthma outcomes, with 42 studies reporting increased exacerbations, including higher medication use (9 studies), increased emergency department visits (20 studies), hospitalizations (8 studies), and reduced lung function (9 studies) [69]. A cross-sectional study that examined the impact of wildfire smoke on pediatric asthma control, in the Northeastern United States, included 1,217 pediatric asthma encounters and found significantly worse asthma control in the wildfire-smoke–affected summer of 2023 compared with 2022, but no significant differences between 2023 and 2024 [31]. In a cohort study of 57,375 children with asthma, on wildfire smoke days ($PM_{2.5} \geq 20$ μg/m^3), emergency department visits increased to 6.5 per day versus 5.8 per day on good air quality days, corresponding to a 13% higher rate of asthma exacerbations (incidence ratio rate – IRR = 1.13; 95% CI: 1.02–1.24), whereas non–wildfire-related air pollution did not show a significant increase [32].

7.2. Sand and Dust Storms

Sand and dust storms pose a significant obstacle to sustainable development, especially in arid and semi-arid regions, where environmental vulnerability and land degradation amplify their frequency and impact. Approximately 2 billion tonnes of dust are released into the atmosphere annually, with the Asia-Pacific region accounting for about 27% of those emissions [70]. Sandstorm dust carries a complex mixture of environmental pollutants and microorganisms, such as bacteria, fungi, and viruses. Moreover, sandstorms significantly elevate ambient air pollutant concentrations, particularly $PM_{2.5}$, CO, and O_3, leading to substantial deterioration of air quality. These events increase environmental contamination and pose notable respiratory health risks [71].

In a cohort study, among 131 children with asthma (77 atopic, 54 non-atopic), atopic children had 113.71% higher fractional exhaled nitric oxide (FeNO) concentrations (95% CI: 68.08%–171.73%) and a 2.62% lower Forced Expiratory Volume in 1 second/Forced Vital Capacity (FEV_1/FVC) ratio (95% CI: −5.00, −0.24) compared to non-atopic peers during dust and pollen periods [37]. In a time-series analysis of 42,920 children, mild dust storms (PM_{10} >71 μg/m^3) were linked to a 5% increase in asthma reliever medication purchases on the same day and a 15% increase in asthma hospitalizations one day after exposure [38]. In a case-crossover study conducted in Toyama, Japan, involving 620 hospitalizations of children aged 1–15 years, heavy desert dust events (> 0.1 mg/m^3) were significantly associated with increased asthma admissions, with a crude OR of 1.88 (95% CI: 1.04–3.41) on the same day and 1.83 (95% CI: 1.31–2.56) over the previous week [39].

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7.3. Thunderstorm Asthma

Thunderstorms are localized weather events driven by atmospheric convection and are marked by thunder, lightning, powerful winds, and intense precipitation. Thunderstorm asthma occurs when pollen or fungal spores are fragmented and dispersed during storm activity, interacting with underlying atopy to trigger acute, localized surges in pediatric asthma exacerbations. As climate change increases the frequency and unpredictability of severe thunderstorms, susceptible children may face greater exposure to sudden spikes in airborne allergens [33].

Following a thunderstorm asthma outbreak in Yulin, a northwest city of China, 391 children presented with respiratory symptoms, with daily emergency visits increasing 2.7-fold and hospital admissions increasing 16-fold. Among 51 hospitalized children, 56% had no prior asthma, 67% had AR, and 94% were sensitized to mugwort pollen [34]. In addition, during the 2023 London thunderstorm asthma event, 50 children (28%) presented with wheeze on 12 June and 18 (19%) on 13 June, with 57% having no prior asthma, 65% presenting with severe or life-threatening symptoms, and 11% requiring hospital admission, with no deaths or intubations reported [35].

8. Adaptation and Mitigation Strategies

Confronting the effects of climate change on allergic diseases requires a comprehensive, systems-based approach that simultaneously targets the mitigation of climate change and integrates evidence-informed adaptation strategies to minimize associated health burdens (Figure 2).

Mitigation	- Phase down fossil fuels - Reduce greenhouses gases emissions (agriculture, cooling, waste) - Improve air quality - Environmental monitoring - Early warning systems - Real-time exposure surveillance
Adaptation	- Strengthen immune resilience - Improve diet & food systems - Safe housing (ventilation, mold control) - Expand urban biodiversity - Planetary health integration

Figure 2. Climate drivers, environmental exposures, biological mechanisms, and pediatric allergic outcomes.

Mitigation approaches should focus on reducing emissions to improve air quality by decreasing fossil fuel use across household, industrial, and transportation sectors, as well as reducing GHG emissions from agricultural activities, cooling and refrigeration systems, and waste management [13,16]. Additionally, continuous monitoring and the implementation of early warning systems for weather-related events, such as sand and dust storms and wildfires, should be integrated into health information systems. Access to real-time data enables health authorities to anticipate high-risk periods and implement targeted measures to mitigate the health effects of air pollution and aeroallergen exposure [64,72].

Adaptation should emphasize strengthening immune tolerance and resilience through health-promoting environmental and societal interventions, by improving diets and agricultural practices

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to enhance food security and access to diverse, healthy foods; providing safe housing through improved weatherization, ventilation, and mold remediation; and increasing biodiversity, particularly by expanding urban green spaces and promoting planetary health [13,16].

9. Conclusions

Climate change represents an escalating and multifactorial driver of allergic diseases in children, acting through increased air pollution, altered aeroallergen patterns, extreme weather events, and early-life immune reprogramming. Converging epidemiological and mechanistic evidence underscores heightened vulnerability during prenatal and early postnatal windows, with potential long-term consequences for immune tolerance and disease susceptibility. Addressing this growing burden requires coordinated mitigation to reduce emissions and environmental degradation, alongside adaptive strategies that strengthen resilience and health system preparedness. Future research should prioritize longitudinal, mechanistic, and intervention-based studies to inform evidence-driven policies and protect pediatric populations in a changing climate.

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