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Urban Air Microbiome and its Impact on Asthma Severity in Pediatric Populations

Waage Maesano*

Department of Respiratory Medicine, Saint Camillus International University of Health Sciences, 00131 Rome, Italy

Introduction

Asthma is a prevalent chronic disease affecting over 300 million individuals worldwide, with pediatric populations being particularly vulnerable. Urban environments are consistently associated with higher asthma incidence and severity and while traditional environmental factors such as air pollution and allergens are well-documented, a growing body of evidence highlights the role of the urban air microbiome in influencing respiratory health. The urban air microbiome comprises microbial communities-bacteria, fungi, viruses and archaea-dispersed through the atmosphere, originating from soil, vegetation, animals, human activity and built environments. This airborne microbial diversity can shape immune development, modulate inflammation and contribute to respiratory pathophysiology. Urbanization reduces biodiversity and alters microbial exposures, often in ways that may predispose children to asthma or exacerbate its symptoms. This explores the composition of the urban air microbiome, mechanisms of interaction with the immune system and the epidemiological and clinical evidence linking air microbiota with asthma severity in children [1].

Airborne microbes are part of the broader aerobiome, which varies by geography, season, pollution levels, land use and human activities. In urban settings, microbial composition is often less diverse and dominated by taxa associated with human sources (e.g., skin, feces), industrial emissions and anthropogenic dust, whereas rural environments have a richer presence of soil- and plant-associated microbes. Several studies using 16S rRNA gene sequencing and metagenomics have demonstrated that urban air is enriched in potentially pro-inflammatory genera such as Acinetobacter, Pseudomonas and Staphylococcus, while lacking immunoregulatory genera like Lactobacillus and Mycobacterium commonly found in rural air [2].

Description

The “hygiene hypothesis” postulates that reduced exposure to microbes in early life due to urban lifestyles, sanitation and reduced contact with nature may impair immune system development, skewing it towards allergic Th2-type responses. This concept has evolved into the “biodiversity hypothesis,” which suggests that reduced environmental and microbial diversity associated with urbanization leads to inadequate stimulation of regulatory immune pathways, increasing the risk for asthma and other allergic conditions. Microbial exposures in early life influence the development of immune tolerance through interactions with epithelial and immune cells. Beneficial microbes promote the induction of regulatory T cells (Tregs) and anti-inflammatory cytokines such as IL-10 and TGF-β, while pathogenic or unbalanced microbiota can lead to chronic inflammation, eosinophilic infiltration and bronchial hyperreactivity. Children living in green environments with access to biodiverse natural areas demonstrate lower rates of asthma and their skin and gut microbiota tend to be more diverse and immunomodulatory than those of children in highly urbanized settings [3].

***Address for Correspondence:** Waage Maesano, Department of Respiratory Medicine, Saint Camillus International University of Health Sciences, 00131 Rome, Italy, E-mail: wmaesano@unicamillus.org*

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The respiratory tract is directly exposed to inhaled microbes, which interact with the mucosal immune system. Pattern recognition receptors such as Toll-like Receptors (TLRs) recognize Microbial-Associated Molecular Patterns (MAMPs), triggering immune cascades that can lead to tolerance or inflammation. Conversely, exposure to microbial diversity may lead to immune training, increased Treg activity and epithelial barrier reinforcement, thereby reducing asthma severity. In asthma, epithelial integrity is often compromised, allowing microbes and allergens deeper access into airway tissues, further amplifying inflammation. The urban air microbiome, particularly when combined with pollutants like PM2.5, can exacerbate oxidative stress and mitochondrial dysfunction in epithelial cells, promoting chronic airway remodeling, mucus overproduction and decreased lung function.

Multiple cohort studies have explored the association between air microbiome exposure and asthma risk or severity. The PARSIFAL and GABRIEL studies in Europe demonstrated that children raised in farming environments, rich in microbial exposures, had significantly lower rates of asthma and atopy. The CCAAPS (Cincinnati Childhood Allergy and Air Pollution Study) linked specific urban bioaerosol profiles to increased wheezing and asthma incidence in preschoolers. In Singapore, the GUSTO cohort found that indoor fungal richness was inversely associated with eczema and wheezing in early childhood. Moreover, a 2021 study using air filters and high-throughput sequencing in New York City identified a correlation between higher bacterial load in air and increased emergency room visits for pediatric asthma. These findings suggest that not only the quantity but the quality and diversity of microbial exposure are key in shaping respiratory outcomes [4].

Understanding the role of the urban air microbiome in asthma pathogenesis has implications for diagnostics, prevention and management. Microbial profiling of air in residential and school environments could inform risk assessments and early interventions. Probiotic-based interventions, though currently more common for gut-related disorders, may eventually extend to respiratory conditions, either via nasal sprays or environmental manipulation. However, significant research gaps remain. Most studies are cross-sectional and observational; longitudinal designs with mechanistic insights are needed. Standardization in air sampling, sequencing and data interpretation will improve comparability. Understanding the dose-response relationship and the role of synergistic factors such as pollutants, allergens and viruses is essential to develop comprehensive exposure models [5].

Conclusion

The urban air microbiome represents an underexplored but increasingly critical factor in pediatric asthma severity. The interplay between airborne microbial communities and the developing immune system can either protect or predispose children to chronic respiratory disease. Urbanization, with its impact on biodiversity, land use and built environments, has reshaped microbial exposures in ways that may partially explain rising asthma rates in cities. Protecting and enhancing microbial diversity-both outdoors and indoors-could become a valuable strategy in reducing asthma burden. Addressing this issue requires interdisciplinary collaboration among microbiologists, clinicians, urban planners and public health officials to create healthier environments for children. Ultimately, recognizing the air we breathe as not just a vector for pollutants but as a living ecosystem may transform our approach to respiratory health in the urban age.

Acknowledgement

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Conflict of Interest

None.

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