

Understanding the Onset of Asthma and Strategies to Reduce Its Risk

Dr. Meenakshi Yadav

Associate Professor, Department of Chemistry, Meerut College, Meerut, Uttar Pradesh, INDIA.

Corresponding Author: meenu.meerut@gmail.com

ORCID

<https://orcid.org/0009-0001-6442-246X>



www.jrasb.com || Vol. 5 No. 1 (2026): February Issue

Received: 08-02-2026

Revised: 13-02-2026

Accepted: 17-02-2026

ABSTRACT

The symptoms of asthma, a long-term respiratory condition marked by inflammation and airway narrowing, include wheezing, coughing, tightness in the chest, and shortness of breath. Due to a number of lifestyle and environmental variables, the prevalence of asthma has dramatically increased globally in recent decades. Genetic vulnerability and environmental exposures, such as air pollution, allergens, tobacco smoke, and occupational irritants, all have a role in the development of asthma. Particularly in children and vulnerable groups, ongoing exposure to particulate matter, dust mites, pollen, and chemical pollutants can cause airway inflammation and hypersensitivity, which can result in the development of asthma.

This study examines the main causes of asthma onset and emphasizes doable tactics to lower the risk. Important preventive strategies include ensuring adequate ventilation, lowering exposure to external contaminants, improving indoor air quality, and implementing healthy lifestyle choices. Reducing the occurrence of asthma can also be greatly aided by raising public awareness and identifying triggers early. Improving respiratory health and lowering the prevalence of asthma worldwide can be greatly aided by comprehending the mechanisms underlying asthma development and putting effective preventative measures into practice.

Keywords- Asthma, inflammation, environment, chemical pollutants, air quality, external contaminants.

I. INTRODUCTION

Millions of individuals worldwide suffer with asthma, a chronic inflammatory illness of the respiratory system. It is typified by bronchial hyperresponsiveness, airflow obstruction, and airway inflammation, which cause symptoms like coughing, wheezing, shortness of breath, and tightness in the chest [1]. The prevalence of asthma has dramatically increased over the past few decades, especially in urban and industrialized areas where exposure to environmental contaminants is considerable, according to worldwide health surveys [2]. A complicated interplay between environmental and genetic variables influences the formation of asthma. People who have a family history of asthma or other allergies are more likely to get the illness. However, the

onset and exacerbation of asthma symptoms are largely caused by environmental factors. Air pollution, tobacco smoking, indoor allergens such dust mites and pet dander, pollen, mold, and exposure to chemicals at work are common causes [3]. Airway structural alterations such as epithelial hyperplasia and metaplasia, alterations in mucus-secreting cells, subepithelial fibrosis, muscle cell hyperplasia, and angiogenesis can result from chronic or recurrent inflammation. The composition, distribution, thickness, mass, volume, and/or quantity of structural elements seen in the airway wall may all change as a result of these pathological changes in the airways. This is thought to be a factor in airway remodeling, a progressive, irreversible decrease of lung function [3–5]. People may have mild, moderate, severe, or refractory asthma depending on the underlying

pathological alterations and the ensuing physiological effects on the airways. Additionally, some of these people may be at risk for permanent airflow restriction or irreversible airway remodeling. A fundamental feature of asthma is variability, characterised by inconsistency in the expression of the disease. While it is well recognised that variability in asthma may cause asthma attacks, it is less well understood that some people may spontaneously become asymptomatic with or without resolving their underlying pathological airway impairment and enter into a symptom-free state [6,7]. This is referred to as “asthma remission”. These individuals who remit or resolve the manifestations of asthma constitute a very interesting cohort who have recently gained much attention among the research community [8,9]. Understanding the mechanisms of remission may shed light on novel therapeutic approaches to asthma. In addition, a proportion of people who have achieved asthma remission or have “grown out of asthma” may relapse later in life. The reason for relapse is unclear, but it might be due to the ongoing dysfunction of underlying pathology. Consequently, these symptom-free patients have a certain risk of relapse, which may occur at any time, and this capricious phenomenon of diminution of disease is called remission [9-11]. Furthermore, exposure to dangerous airborne compounds has grown due to growing urbanization, industrial pollutants, and poor indoor air quality [1]. Developing successful preventative interventions requires an understanding of how asthma develops and the identification of the major risk factors. The chance of developing asthma can be considerably decreased by identifying asthma triggers early and implementing preventative interventions such as enhancing indoor air quality, lowering pollution exposure, and encouraging healthier lifestyles [2].

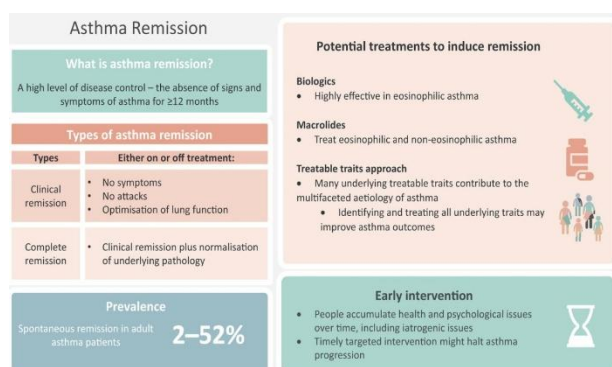


Figure 1. Overview of asthma remission mechanisms and treatment approaches [12].

Source: Adapted from Thomas et al., 2022.

Underlying Factors and Mechanisms of Asthma Development

Asthma develops due to multiple interacting factors rather than a single cause. Individuals with a genetic or biological predisposition are more vulnerable,

and repeated exposure to environmental triggers such as pollutants, allergens, and infections increases the risk of developing the disease. These continuous exposures lead to changes within the body, especially in the lungs, affecting how the respiratory system responds to external stimuli.

As a result, the airways become more sensitive and prone to inflammation. Over time, these changes create conditions that favor the onset and progression of asthma. Thus, asthma is a complex condition that arises from the combined effects of underlying susceptibility and environmental influences.

Genetics and individual susceptibility

It has long been known that asthma has a genetic component; children who have an asthmatic parent are more likely to develop asthma themselves [13]. Numerous genes have been linked to an increased risk of developing asthma. However, as asthma can occur in only one of two identical twins [14]. The results of twin cohort studies suggest that asthma is not solely a hereditary condition. Examining mechanistic pathways and how gene variants impact these pathways may better reflect individual susceptibility to exposures, which has not been adequately characterized by genome-wide association studies.

Changes in the environment or behaviour create epigenetic modifications, also known as epigenetic reprogramming, which modify gene expression without changing the genes themselves. It is becoming more widely acknowledged that these changes play a significant role in the pathophysiology of asthma [15]. These hereditary changes alter gene expression and coordinate subsequent processes including immunological and epithelial cell reactions. In addition to offering a possible target for therapies due to the epigenome's plasticity and potential reversibility, the discovery that epigenetic modifications are closely associated with early life events may help to explain why exposure timing is crucial [16].

Immune responses in asthma

Evidence from clinical, epidemiological, and experimental studies suggests that some insults and exposures can change immunological responses, which can worsen asthma in those who already have the condition [17,18]. Eosinophilic, non-eosinophilic, neutrophilic, and paucigranulocytic asthma are subtypes of asthma based on the inflammatory cell count in induced sputum, which reflects their numbers in the airways. These subtypes can be supported by a range of innate and adaptive immune responses, including those involving Th2, Th1, and Th17 [15]. The coordination, activation, and chemoattraction of immune cells from systemic locations, such as the bone marrow, depend on immunological responses in the lung microenvironment. According to recent research, local lung immune responses in asthmatics are similar to systemic immune responses, such as those observed in the blood, [19] suggesting that these responses should be taken into

consideration as potentially modifiable targets for therapies.

Oxidative stress

One of the main characteristics of asthma is oxidative stress, which is the local imbalance between oxidants (such as reactive oxygen species like hydrogen peroxide, superoxide anion, and hydroxyl radical) and antioxidants. This imbalance is linked to higher levels of proinflammatory cytokines, type 1 and type 2 immune responses, and increased inflammation. Genome-wide association studies have found mutations in genes linked to oxidative stress in asthma, such as the glutathione S-transferase genes GSTM1, GSTP1, and GSTT1. Additionally, oxidative stress is caused by numerous pertinent exposures and shocks [13]. It is unknown, therefore, to what extent an individual's susceptibility to oxidative stress explains asthma.

Altered lung structure

The airway epithelium is a crucial immune system modulator and the initial point of contact for chemicals breathed. Asthma has been linked to abnormalities in the airway epithelium, such as decreased barrier integrity, delayed healing, goblet cell hyperplasia, and changed inflammatory mediator production[20]. These flaws may make a person more susceptible to exposures and insults from the outside world. Furthermore, the epithelium of individuals with asthma does not react to viruses in the same way as healthy epithelial cells, which means that exposures may cause additional harm to the epithelium. Furthermore, the epithelium of individuals with asthma does not react to viruses in the same way as healthy epithelial cells, which means that exposures can cause additional harm to the epithelium. Epithelial remodeling may result from a vicious cycle of damage, altered responses, and healing.

People with asthma have essentially altered smooth muscle in their airways, which can lead to inflammation, remodeling, and narrowing of the airways. Hypertrophy or hyperplasia (remodeling), modifications to its intrinsic, structural, or mechanical characteristics, and changed interactions with the surrounding lung tissue are a few examples of these changes[21]. Although the fundamental mechanisms underpinning alterations in airway smooth muscle in asthma are recognized, it is less clear when these changes occur throughout life and whether they contribute to the pathophysiology of asthma or are its consequences.

The microbiome

People with asthma have a lower microbiome diversity and a higher bacterial load [22]. Early life microbial diversity is crucial for the immune system's development, and dysbiosis can tilt the scales between innate and adaptive immunological responses, resulting in inflammation and sensitization. Dysbiosis may increase the risk of asthma by creating conditions that make it easier for pathogenic germs to move from the upper to the lower airway [23]. Because it has a

significant impact on how the infant's immune system and microbiome grow, the mother's microbiome is also significant. Pregnancy-related exposures, such as those to antibiotics, can cause oxidative stress and inflammation, which can change the fetus's immunological primed [24]. The development of the infant's immune system is also significantly influenced by the transfer of gut or vaginal microorganisms from the mother to the child during childbirth.

II. MATERIAL AND METHODS

The assessment of earlier studies on asthma and related risk factors served as the foundation for this investigation. Information about asthma, its causes, and preventive measures was gathered from research articles, review papers, and standard reports. For the study, only pertinent and trustworthy sources were taken into account. The gathered data was thoroughly examined and categorized according to key elements such oxidative stress, immunological response, environmental exposure, genetic reasons, and features associated to the microbiome.

Common triggers, such as air pollution, allergies, and lifestyle-related factors, received special focus. To determine how these factors affect the development and course of asthma, a descriptive approach of analysis was employed. In order to detect recurring themes and significant conclusions from several studies, the available data was also compared. In order to comprehend their function in lowering the risk and severity of asthma, preventive interventions recommended in the literature were also examined.

III. RESULTS & DISCUSSION

The current study demonstrates that asthma is a complex illness impacted by both environmental and hereditary variables. Asthma is largely caused by environmental exposure, including indoor allergens, tobacco smoke, air pollution, and occupational irritants, particularly in metropolitan regions where pollution levels are higher [1,2].

Over time, these factors cause breathing problems by increasing airway sensitivity. Asthma is also influenced by genetics because people who have a family history of the condition are more likely to get it [13]. However, environmental exposure has an equally significant role in causing the illness, and genetics alone is not the cause.

The immune system has a significant role in the development of asthma. Airway inflammation and hypersensitivity have been found to result from an imbalance in immune responses, specifically involving Th1, Th2, and Th17 cells [15,16]. Oxidative stress, which results from an imbalance between the body's antioxidants and reactive oxygen species, exacerbates this inflammation[13].

Furthermore, long-term or irreversible breathing issues may result from airway remodeling, a structural alteration in the airways brought on by repeated exposure to detrimental environmental variables [3–5]. Because decreased microbial diversity, particularly in early life, is associated with an increased risk of asthma, the microbiome also plays a significant role [22,23]. Antibiotics and environmental contaminants are examples of maternal and early-life exposures that may impact immune system development and raise asthma susceptibility [24]. Overall, the results point to a combination of environmental and genetic variables in the development of asthma. Reducing exposure to triggers and identifying risk factors early on can help lessen the disease's incidence and severity.

IV. CONCLUSION

In conclusion, asthma is a long-term respiratory condition brought on by a confluence of environmental and genetic variables. Asthma risk is significantly increased by exposure to allergens, air pollution, and other irritants, especially in metropolitan populations. In addition, people who have a family history of asthma are more likely to develop the condition. The study also emphasizes how immunological reactions and oxidative stress contribute significantly to airway inflammation and exacerbate the illness. Long-term exposure to dangerous substances may cause structural alterations in the airways, which could lead to ongoing breathing issues. Additionally, the development of asthma later in life may be influenced by early-life factors such as environmental exposure and microbiome changes.

In general, there are several interrelated elements that contribute to the development of asthma, making it impossible to pinpoint a single cause. Therefore, reducing exposure to environmental triggers, improving living conditions, and increasing awareness about preventive measures can help in lowering the risk and burden of asthma.

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