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Do Genes Play a Role in Allergic Rhinitis?

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Editorial
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Abbreviations: AR: Allergic Rhinitis; HLA: Human Leukocyte Antigen; MHC: Major Histocompatibility Complex.

Editorial

Allergic rhinitis is a common condition affecting millions of people worldwide. While the causes of this condition are complex and multifactorial, recent research has shed light on the role of genetics in modulating the development and severity of allergic rhinitis. Anyone can becomes allergic when their body develops antigens against an allergen, and repeated exposures can lead to the severity of the allergic process. Allergies affect people of all ages, races, genders and socioeconomic statuses [1]. Confirmed diagnostic tests for AR include skin prick test and IgE test.

Allergic rhinitis, also known as hay fever, is a common allergic disorder characterized by exposure to allergens like pollen, dust mites, and animal dander and inducing inflammation of the nasal mucosa. The development of allergic rhinitis is influenced by a complex interplay of both, genetic and environmental factors. It is a type of allergic reaction that affects the nasal passages and causes symptoms such as sneezing, runny nose, and itchy eyes. It is a complex disease that results from environmental factors which can influence the expression of genetic parameters associated with allergic rhinitis in several ways: firstly, genetic factors have been shown to play a role in the development of allergic rhinitis, by modulating the expression of these genes which is often influenced by environmental factors. Secondly, environmental factors can also modulate the severity of the disease, as exposure to air pollution can exacerbate allergic rhinitis symptoms, and studies have shown that genetic variations in genes involved in inflammation and immune responses can modify the effects of air pollution on allergy symptoms. Lastly, environmental factors can also influence the effectiveness of treatments for allergic rhinitis, because tobacco smoke has been shown to reduce the effectiveness of corticosteroid nasal sprays in treating allergic rhinitis. Thus, environmental factors interact with genetic parameters in complex ways, influencing the onset, severity, and treatment of the condition [2]. An example of statistically significant association of smoking and Rhinitis comes from a study which demonstrated that adolescents in European countries developed allergic rhino-conjunctivitis [3]. Severity of the symptoms can be divided into mild with no interference with quality of life or it can be severe with symptoms impacting sleep, and work performance in all daily activities [4]. Several genes and gene variants have been associated with the development or modulation of allergic rhinitis. Some of the genetic factors which play a key role in the development and progression of allergic rhinitis (AR) are included in this article.

Most important are the Human Leukocyte Antigen (HLA) genes, the HLA gene complex is a crucial component of the immune system, responsible for presenting antigens to T-cells and regulating immune responses. Specific HLA gene variants have been associated with an increased risk of developing allergic rhinitis, such as HLA-DQB1, HLA-DRB1, and HLA-DRA. Interleukins are a group of cytokines that play a significant role in immune system function, including the regulation of allergic responses. Variants in several interleukin genes have been linked to allergic rhinitis such as: IL4 and IL4R, IL4 is a cytokine that promotes the

differentiation of T-helper 2 (Th2) cells, which are involved in allergic responses. Variants in the IL4 gene and its receptor gene**,** IL4R, have been associated with increased susceptibility to allergic rhinitis. IL13 is another cytokine involved in Th2 cell differentiation and has similar functions to IL4. Variants in the IL13 gene have been linked to allergic rhinitis. IL10 is an anti-inflammatory cytokine that helps regulate immune responses. Certain variants in the IL10 gene may contribute to a heightened risk of developing allergic rhinitis by modulating the immune response.

To know the difference between the smokers and nonsmokers [5] one study reported that they had evaluated Immunological markers in serum and nasal washes (IgE, IL-4, IL 5, IL 13, IL 17, IL 33). Subsequently after analyzing statistically using Student T test, x2, Mann Whitney U (Anova 2-way), and Kruskal Wallis for the groups they showed that the P value (< 0.05) was significant.

In allergic rhinitis, the inflammatory response has always been described as being mediated by immune response which is a fact as it is mediated by interaction of several immune cells (monocytes, lymphocytes, and polymorphonuclear cells) and cellular chemical mediators. A few of the immune parameters include - Immunoglobulin E (IgE) Receptor gene: IgE is an antibody that plays a central role in allergic reactions [6]. Variants in genes encoding IgE receptors, such as FCER1A, FCER1B, and MS4A2, have been implicated in the development of Allergic rhinitis. Repeated exposure to allergens over a period to time induces the production of large amount of IgE antibodies thus generating allergic reaction.

Nur Husna, et al. [7] and Sani MM, et al. [8] have beautifully described the complete process of IgE production as a response to the allergens. These authors report that within 20 minutes of exposure to an harmful allergen the dendritic cells in the nasal mucosa process the allergens to make peptides on the major histocompatibility complex (MHC) class II molecules which serve as ligands for T-cell receptors which then differentiate into allergen specific Th2 cells. Followed by release of IL-4 and IL-13 cytokines from the activated Th2 cells to interact with B cells to produce allergen specific IgE. The IgE then binds to the Fc receptor leading to mast cell activation. T-cell receptor genes are essential for T-cell activation and regulation of immune responses. Some studies have suggested that variants in T-cell receptor genes, like TCRα and TCRβ, may influence the risk of AR. Toll-like receptor genes (TLRs) are critical for recognizing pathogens and initiating immune responses. Variations in TLR genes, such as TLR2, TLR4, and TLR9, have been associated with allergic rhinitis susceptibility. Symptoms of an allergic reactions vary with the type and amount of allergen encountered and the manner in which the body's immune system reacts to that allergen.

Adhesion molecule genes are involved in the recruitment and migration of immune cells to sites of inflammation. Variants in genes encoding adhesion molecules, such as ICAM1 and VCAM1, have been associated with allergic rhinitis susceptibility [9]. The biochemical response that follow after IgE production and the release of mediators categories the disease as mild, moderate or severe. Mucosal edema or watery rhinorrhea of AR and the leak in the blood vessels is caused by Histamine a major chemical mediator of AR. These mediators cause vascular engorgement (nasal congestion) by interacting with H1 and H2 receptors on the blood vessels [10].

Other Genes of concern in AR that have been implicated in the development or modulation of allergic rhinitis, including genes involved in inflammation, immune regulation, and the function of the airway epithelium are STAT6, NPSR1, ADAM33 and GSDMB. It is essential to note that the genetics of allergic rhinitis is complex, and no single gene can determine the development or severity of the condition. The genes listed above have been identified through various studies and may contribute to allergic rhinitis susceptibility to varying degrees. Further research is needed to fully understand the genetic factors involved in allergic rhinitis and how they interact with environmental factors to influence disease development research is needed to fully understand the genetic factors involved in allergic rhinitis and how they interact with environmental factors to influence disease development and research is needed to fully understand the genetic factors involved in allergic rhinitis and how they interact with environmental factors to influence disease development and progress.

Previous research has identified several genes that may be involved in the pathogenesis of allergic rhinitis. For example, the IL-13 gene has been shown to be associated with increased risk for allergic rhinitis, while the HLA gene has been shown to be protective against the condition. More recently, a genome-wide association study identified several new genetic variants that may play a role in allergic rhinitis. This latest research on genetic factors modulating allergic rhinitis has important clinical implications. By better understanding the genetic basis of the condition, we may be able to develop more targeted treatments and improve patient outcomes. For example, we may be able to develop personalized treatments based on a patient's genetic profile.

In conclusion, the latest research on genetic factors modulating allergic rhinitis is an exciting development in the field of allergy and immunology. Continued research in this area has the potential to greatly improve our understanding of the condition and lead to better outcomes for patients.

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