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Focusing on its immunomodulatory properties, curcumin's therapeutic effectiveness for allergic disorders

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INTRODUCTION

The term "allergy," which is also known as "atopy," refers to a variety of immune-mediated diseases that affect almost 30% of the world's population. These diseases are brought on by the induction of immune responses against common environmental antigens, also known as "allergens," which are non-microbial and generally harmless. Urticaria, food allergy, allergic rhinitis (AR), allergic asthma, atopic dermatitis (AD), and anaphylaxis are the most common allergic diseases. According to an immunological perspective, patients with hypersensitive illnesses produce and advance the sort 2 resistant reaction intervened by a broad assortment of invulnerable cells including epithelial cells, pole cells, basophils, eosinophils, and B and T lymphocytes. These cells generate a variety of mediators, such as cytokines that are pro-inflammatory, chemokines that are anti-inflammatory, histamine, lipid mediators, and IgE antibodies, which aid in the development of allergic diseases. Several therapeutic strategies have been developed to alter the allergen-specific immune response in allergic patients by focusing on immune cells and their components in light of these harmful immune reactions mediated by innate and acquired immunity. Non-steroidal anti-inflammatory drugs (NSAIDs), topical and inhaled corticosteroids, anti-histamines, beta-adrenergic agonists, and biological agents (like monoclonal antibodies) are the most common of these medications. Even though these treatments have had some success in clinical trials, there is growing concern about their long-term use and side effects, which may result in infections (like oral candidiasis), glaucoma, dysphonia, damage to the liver, damage to the gastrointestinal tract, and heart failure [1].

DESCRIPTION

Additionally, the use of monoclonal antibodies has a significant financial impact on the sick. Lately, the tweak of resistant reactions utilizing regular beginning mixtures to control and converse key pathologic safe reactions in a few safe intervened illnesses holds guarantee gives another restorative procedure. Curcumin, which has high potency in modulating immune responses and therapeutic potential, is one of the most important natural compounds in this regard. Curcumin's therapeutic efficacy in treating or controlling a number of diseases, including immune-mediated inflammatory disorders like rheumatoid arthritis (RA), systemic lupus erythematosus (SLE), inflammatory bowel disease (IBD), multiple sclerosis (MS), and type-1 diabetes mellitus (T1DM), atherosclerosis, and cancers,

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has been demonstrated by extensive research conducted over the past thirty years. In order to investigate curcumin's immunomodulatory effects in allergic diseases, we combined a number of in-vitro and in-vivo studies in the present review. In addition, this review examines curcumin's therapeutic potential in relation to its immunomodulatory effects on allergic disease patients [2].

Immunopathogenesis of Allergies Innate immunity and Allergies Innate immune responses are crucial to the pathogenesis of allergies. As the first line of innate immunity, epithelial cells lining barrier sites secrete a wide range of chemokines and pro-inflammatory cytokines in response to airborne allergens and encourage innate immune responses in alveolar cavities. Several different kinds of cell surface receptors from the pattern recognition receptor family (PRR) are expressed by epithelial cells. These receptors bind to various allergens, initiate pro-inflammatory signaling pathways, and encourage innate immune responses that are involved in allergic diseases. In addition, DCs, which play a crucial role in innate immunity and express toll-like receptors (TLRs), one of the most important members of PRRs, are able to directly respond to foreign allergens by producing a variety of pro-inflammatory mediators like interleukin (IL) 8, IL-1, and tumor necrosis factor (TNF-).It is interesting to note that a number of factors, such as the type of cell, the allergens, and the method of exposure, play a significant role in the pathogenesis of allergy. Indeed, the mediating role of the TLR4 signaling cascade in uptake of intranasal allergens in allergic reactions and allergy development.Dectin2, which is located on the surface of DCs, is yet another significant receptor.Dectin2 and house dust mites can interact to promote allergic responses and TH2 cell-mediated type 2 immune responses. Another PRP, the Nod-Like Receptor (NLR), is linked to the severity of asthma and allergies. By inducing the production of thymic stromal lymphopoietin (TLSP) and IL-25, the NOD2 signaling pathway shifts immune responses toward the TH2 immune profile, promoting allergic reactions [3].

In allergic diseases, cytokines and chemokines play a crucial role in the innate immune system alongside PRPs.IL-33 is one of the most crucial cytokines in allergic reactions. When an aeroallergen activates PRRs, it causes the secretion of IL-33. This causes pro-inflammatory signaling cascades like mitogen-activated protein kinases (MAPKs) and nuclear factor-B (NF-B) pathways to be triggered, which in turn causes allergic responses.Additionally, eosinophils and mast cells—key components of allergic reactions—cannot survive without IL-33.Another important cytokine in innate immunity, TSLP, plays a pathogenic role in allergic diseases. A distinct phenotype of DCs that enhance TH2 immune responses can be activated and matured by both IL-33 and TSLP, resulting in allergic diseases. Through the promotion of inflammatory responses and the expression of adhesion molecules necessary for the infiltration of inflammatory cells into the site of allergic reactions, TNF-, a crucial pro-inflammatory mediator that is secreted by mast cells, plays pathologic roles in allergic reactions [4].

In addition, mast cells produce IL-4, which is necessary for the differentiation of TH2 cells and the induction of allergic reactions. TNF-a is the essential factor for the production of allergen-specific IgE. Recently, it has been reported that patients with AR have nasal mucosa that produces more IL-32.IL-32 expression is triggered when granulocyte macrophage-colony stimulating factor (GM-CSF) activates caspase-1 in allergic reactions. In allergic reactions, IL-32 is closely linked to the activation of the NF-B pathway, the release of IL-1, IL-6, and TNF-, and the elevation of IgE. In conclusion, there is growing evidence that the innate immune system is crucial to the development of allergic diseases. Hence, focusing on natural safe cells and their parts could be an important procedure in treating or controlling hypersensitive issues.

Since the discovery of TH1 and TH2 cells, it has been well established that these cells play important physiologic roles in the defense against numerous harmful pathogens. TH1/TH2-mediated immune responses and allergic diseases. However, these cells also play a pathological role in a number of diseases caused by the immune system. The immunopathogenesis of allergic disorders is now better understood thanks to the rapid development of cellular and molecular techniques. In this regard, studies conducted on human cells, tissues, and animal models suggested that immune responses mediated by TH1 and TH2 might play important roles in allergic reactions.

The imbalance between TH1 and TH2 was found to be a major feature of allergy, according to increasing evidence. TH2 cells produce a significant quantity of IL-4, IL-5, and IL-13.IgE production is then stimulated when TH2 cells interact with B cells. Histamine, leukotrienes (LTs), and prostaglandins (PGs) are among the allergic mediators that are secreted when mast cells and basophils are activated and IgE is bound to FcRI, which are specific receptors on the surface of these cells. Allergy is characterized by the production of IgE and its subsequent effects, which are dependent on the activation of IL-4–producing helper T cells. Additionally, activation of eosinophils by IL-5 is required for the promotion of inflammatory responses to allergens within tissues, which in allergic asthma results in respiratory dysfunction. When epithelial cells in the airways are activated by IL-13, they produce a lot of mucus, which is also a common symptom of allergic reactions.

In contrast to the TH2 subtype, immune responses mediated by TH1 cells suppress TH2-related profiles by inhibiting IgE production and producing interferon-gamma (IFN-), thereby protecting against allergic disorders.

It has been hypothesized that an imbalance between the subsets of TH1 and TH2 cells plays a role in the development of allergic diseases. As a result, it may be possible to lessen the severity of allergic diseases by addressing this imbalance [5].

CONCLUSION

Immune responses mediated by TH17/Treg and allergic diseases The TH17 subtype is a distinct lineage of CD4+ T cells that has a high capacity to produce important cytokines, such as IL-17, IL-22, and IL-25, in order to

carry out inflammatory functions. In addition to the fundamental functions that TH17 performs in the etiology and progression of autoimmune diseases, additional evidence demonstrates that these inflammatory effector cells play important roles in allergic reactions. The severity of allergic asthma has been linked to the prevalence of TH17 cells and the cytokines that are associated with them. Through the activation and recruitment of neutrophils, IL-17A, IL-17B, and IL-17F cause inflammation in the airway, where neutrophils cause pathological changes in lung tissues by producing several enzymes that degrade tissue. Additionally, the TH2 immune responses triggered by TH17 cells' production of IL-25 eventually increase allergic reactions.

REFERENCES

1. **Hilty M, Burke C, Pedro H et al.** Disordered microbial communities in asthmatic airways. *PloS one*. 2010;5(1):8578.
2. **Insel M, Kraft M.** Bacteria in asthma pathogenesis. *Immunol Allergy Clin*. 2019;39(3):377-89.
3. **Teo SM, Mok D, Pham K et al.** The infant nasopharyngeal microbiome impacts severity of lower respiratory infection and risk of asthma development. *Cell host microbe*. 2015;17(5):704-15.
4. **Larsen JM, Brix S, Thysen AH et al.** Children with asthma by school age display aberrant immune responses to pathogenic airway bacteria as infants. *J Allergy Clin Immunol*. 2014;133(4):1008-13.
5. **Marri PR, Stern DA, Wright AL et al.** Asthma-associated differences in microbial composition of induced sputum. *J Allergy Clin. Immunol*. 2013;131(2):346-52.