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REVIEW

Potential Mechanisms of Traditional Chinese Medicine for the Treatment of Allergic Rhinitis: Evidence from Molecular and Clinical Studies

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Abstract: Allergic rhinitis (AR) is a common chronic inflammatory disease of the upper respiratory tract and has become a significant global public health issue. With increasing globalization and environmental challenges, the factors contributing to AR pathogenesis have expanded, leading to a marked rise in its global incidence. Current clinical treatment guidelines recommend antihistamines, glucocorticoids, and leukotriene receptor antagonists for managing the clinical symptoms and pathological changes associated with AR. However, as patients' expectations for quality of life increase, concerns regarding the recurrent nature of AR and the long-term use of pharmacological treatments have grown, particularly concerning the potential side effects and sustained efficacy of these medications. Therefore, the pathogenesis of AR remains a key research focus and a critical entry point for therapeutic intervention. In this context, Traditional Chinese Medicine (TCM), with over 2500 years of clinical experience and a comprehensive theoretical framework, has long recognized the condition and offers therapeutic strategies to treat or delay its onset. TCM exerts its effects through multiple mechanisms, including immunomodulation, suppression of oxidative stress, regulation of gut microbiota, inhibition of inflammatory responses, and restoration of normal cellular autophagy and apoptosis. This paper reviews ex vivo and in vivo studies on the use of TCM in AR treatment, focusing on the modulation of various pathogenic mechanisms and the underlying theoretical basis. It aims to identify potential drug sources and expand therapeutic options for AR. In addition, this review analyzes the administration routes and treatment durations of various TCM interventions, offering insights into the development of safe and effective targeted therapies. It also provides guidance for future experimental and clinical research while highlighting the current challenges facing TCM in the management of AR.

Keywords: allergic rhinitis, Traditional Chinese Medicine, mechanism, herbal medicine, drug development

Introduction

Allergic rhinitis (AR) has emerged as a significant global public health concern. With ongoing globalization, industrialization, and changes in climate and the environment, the diversity and quantity of allergens have increased, contributing to the increasing prevalence and incidence of various forms of AR worldwide.[1] AR affects approximately 40% of the global population, with rising prevalence across all age groups. Studies indicate that the overall prevalence ranges from 10% to 30% in Asia, 15% to 25% in Europe, and up to 30% among children in Northern Europe. In Africa, the prevalence ranges from 5% to 15%, with notable regional disparities. In Northern Europe, children and adolescents constitute a large proportion of cases, while in Southern Europe, the total prevalence ranges from 10% to 25%. In urban areas, the overall prevalence reaches 20% to 35% in Oceania, with Australia reporting a prevalence of 35%.[2–5] AR is a common and heterogeneous chronic disease characterized by a Th2-dominant inflammatory response in the nasal mucosa. Symptoms of AR include nasal congestion, an itchy nose, sneezing, and rhinorrhea, with more severe cases potentially resulting in olfactory dysfunction. Historically, AR was regarded as a chronic inflammatory disorder limited to the nasal mucosa and airways. However, extensive basic and epidemiological studies have now established that AR is part of a broader systemic

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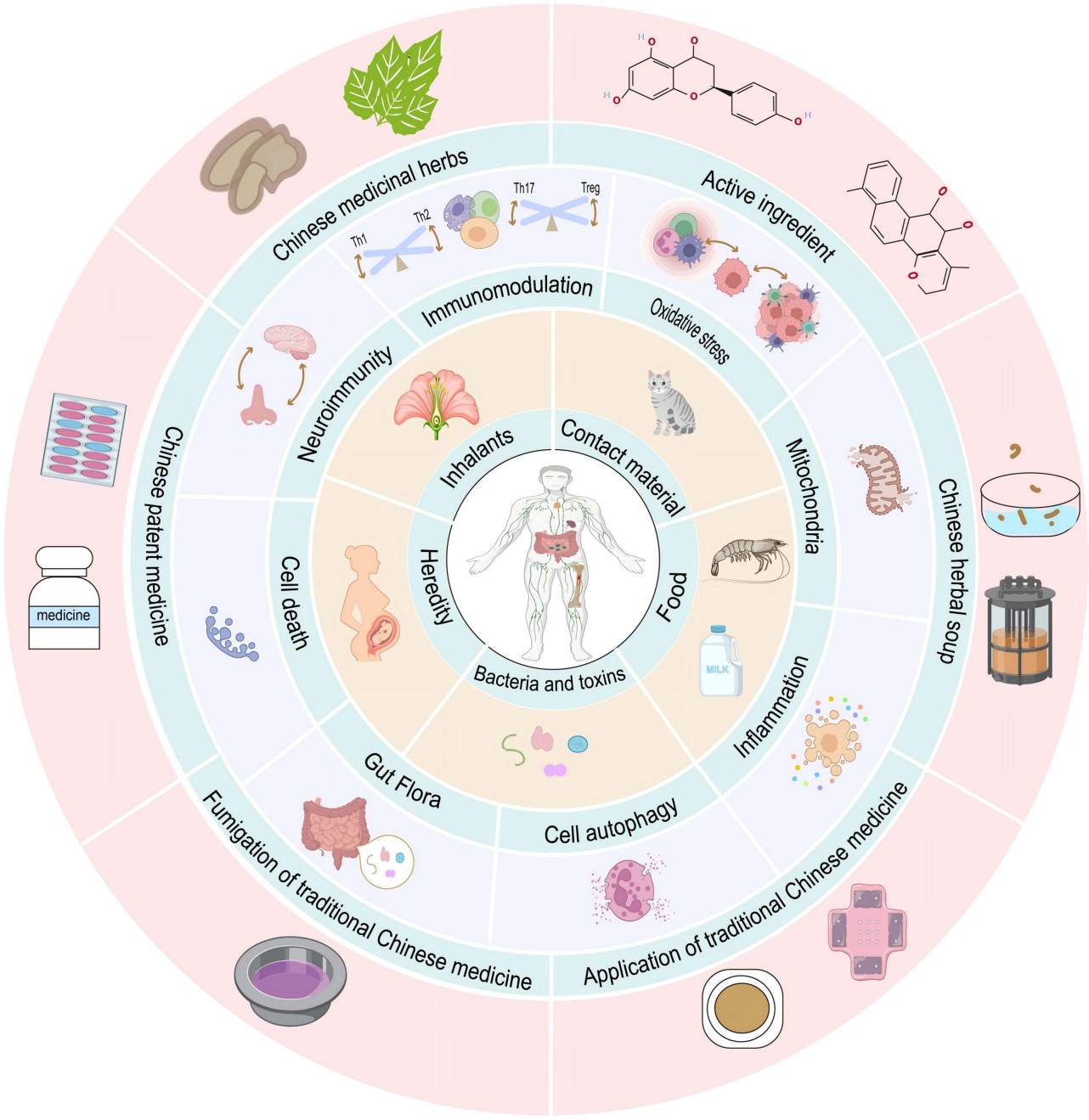
Graphical Abstract



inflammatory response and is closely linked to other mucosal inflammatory diseases, such as asthma, allergic conjunctivitis, atopic dermatitis, and sinusitis.[6] Characterized by a prolonged course, frequent recurrence, and complex pathophysiology, AR not only negatively affects patients' physical and mental health and quality of life, but also imposes a substantial socioeconomic burden. The etiology of AR remains incompletely understood in Western medicine, although key contributing factors are believed to include allergen exposure, inflammation, oxidative stress, and immune dysregulation.[7] Currently, clinical treatment options for AR are limited, with Western medicine primarily relying on pharmacological

therapies such as antihistamines, glucocorticoids, and leukotriene receptor antagonists. Commonly prescribed medications include loratadine, beclomethasone dipropionate, and montelukast. Despite their widespread use, long-term administration of these drugs may lead to serious adverse effects, including immunosuppression, osteoporosis, central nervous system depression, vasodilation, and other complications.[8] In recent years, immunotherapy has been introduced into the medical field, primarily targeting single allergens such as those derived from dust mites and Artemisia pollen. Currently, two major forms of immunotherapy are widely used in clinical practice: subcutaneous immunotherapy (SCIT) and sublingual immunotherapy (SLIT). These therapies typically require administration over a period of 3–5 years to achieve therapeutic efficacy, with a reported clinical response rate of approximately 45% to 50%.[9] Notably, although long-term immunotherapy can reduce reliance on medications such as antihistamines, it is associated with age-related limitations. Improper administration may exacerbate local and systemic allergic reactions, such as redness, swelling, and pruritus at the injection site, and, in severe cases, may lead to anaphylaxis.[10,11] At the same time, the economic burden of long-term immunotherapy remains significant, posing a considerable challenge for families with limited financial resources and in low-income regions. Therefore, questions remain regarding the widespread adoption of this treatment.[12] Traditional Chinese Medicine (TCM) has been practiced in China for thousands of years, giving rise to a comprehensive theoretical system and an extensive body of classical literature. Recently, there has been growing interest in treatments that are either assisted by or based primarily on Chinese medicine. As a well-established therapeutic approach, TCM adopts a holistic perspective, offering not only therapeutic effects for AR but also preventive and protective benefits. Herbal medicines are generally associated with low toxicity and a favorable safety profile. With the advancement of clinical practice, herbal treatments have undergone continuous refinement, enabling their application in a variety of dosage forms and administration routes.[13] Existing studies have confirmed that the total clinical effective rate of oral Chinese herbal medicine can reach 60%-90%, with significant variations observed among different herbal medicines or prescriptions. Compared with antihistamines, Chinese herbal medicine demonstrates a higher efficacy rate, fewer side effects, and a more pronounced improvement in clinical symptoms.[14] Notably, the recurrence rate of oral Chinese medicine is only 24.46%, whereas that of antihistamines ranges from 40% to 60%.[15] These findings indicate that TCM plays a valuable role in the long-term management of AR, effectively delaying disease progression. Moreover, TCM may address certain limitations of antihistamines or nasal corticosteroids. At present, there is growing global interest in holistic integrative medicine, which is a patient-centered approach that combines modern (Western) medicine with traditional and complementary therapies—such as TCM, acupuncture, nutritional support, and psychological interventions.[16] This integrative model has the potential to enhance therapeutic outcomes, reduce drug-related adverse effects, lower healthcare costs, improve patients' quality of life, and promote the evolution of the medical paradigm. In recent years, an increasing number of TCM treatment modalities have demonstrated efficacy in managing AR; however, these findings have not yet been systematically reviewed. This paper aims to summarize current research on the role of TCM in treating AR and to provide clinical insights and novel perspectives on its application in AR management. The mechanisms of AR treatment are analyzed from multiple perspectives, including Chinese medicinal herbs, active ingredients, compound prescriptions, Chinese patent medicines, and other auxiliary therapies. By summarizing both in vivo and in vitro studies, it is evident that TCM plays a significant role in regulating immunity, reducing inflammation, inhibiting oxidative stress, improving intestinal flora dysbiosis, and restoring cellular autophagy, apoptosis, and mitochondrial function—key pathological aspects of AR. Given the current limitations in selective treatment options and growing concerns regarding long-term drug efficacy and safety, this paper highlights the holistic concept of TCM and its integrated approach to prevention and treatment, while taking into account global economic disparities. Finally, the paper discusses the major opportunities and challenges in the current treatment of AR and identifies emerging areas for future research.

Pathogenesis of AR

Current research on the pathogenesis of AR focuses on seven key mechanisms: immune dysregulation, inflammatory responses, oxidative stress, alterations in the gut microbiota, impaired cellular autophagy, pyroptosis, and mitochondrial dysfunction.

Immunomodulation

Organismal Immunomodulation

Immunoregulatory mechanisms hold paramount importance in the progression of AR. Upon exposure to allergens, mucosal cells are damaged, allowing allergens to enter the body and trigger abnormal immune responses. When allergens interact with dendritic cells, antigen-presenting cells process the allergens and present them to CD4+ T cells. This process disrupts the balance between Th1/Th2 and Treg/Th17 cell populations, leading to the excessive secretion of cytokines such as IL-4, IL-13, and IL-5. These cytokines stimulate B cells to differentiate into memory B cells, which subsequently interact with dysregulated T cells to produce large quantities of allergen-specific IgE antibodies.[17] During the sensitization phase, re-exposure to the same allergen leads to the release of eosinophilic cationic protein (ECP), major basic protein (MBP), and the production of specific IgE antibodies. These IgE antibodies bind to high-affinity IgE receptors on mast cells and basophils, triggering degranulation and the release of mediators such as histamine, leukotrienes, and prostaglandins. These mediators induce vasodilation, vascular congestion of the nasal mucosa, and activation of nasal sensory nerves, resulting in early-phase clinical symptoms like sneezing and rhinorrhea.[18] Simultaneously, allergen-induced eosinophil infiltration generates oxidative stress and promotes the release of thymic stromal lymphopoietin (TSLP), which activates epithelial dendritic cells (DCs), prompting their migration to lymph nodes and initiating T cell differentiation. Additionally, TSLP—an epithelium-derived cytokine—upregulates OX40L expression on human myeloid dendritic cells (mDCs), thereby mediating Th2 cell immune responses and facilitating the recruitment of inflammatory cells.[19] Abnormal T-lymphocyte differentiation leads to the release of cytokines such as IL-4 and IL-13, which infiltrate macrophages and monocytes, triggering the aberrant secretion of inflammatory factors. Additionally, specific IgE antibodies bind to IgE receptors on macrophages, further enhancing IgE production, impairing immune cell function, intensifying the inflammatory response, and ultimately damaging the nasal mucosa and affecting the nervous system[20] (Figure 1).

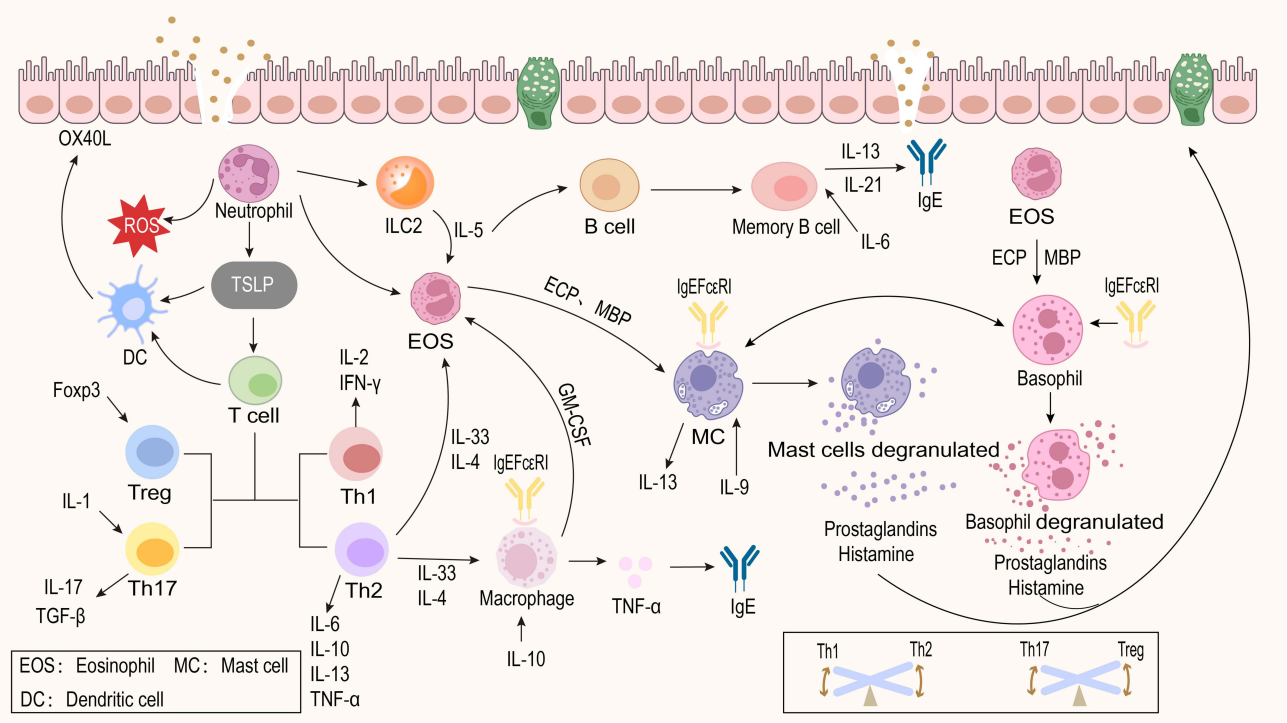


Figure 1 Nasal mucosa contact with allergens, destroying the original mucosal structure, when the violation of neutrophils, triggering oxidative stress, stimulating TSLP so that it affects the dendritic cells (DC) and T cells, resulting in the release of dendritic cells to destroy the mucosal immune function of OX40L; at the same time so that the T cells in the Th1, Th2, Treg and Th17 cells in the imbalance, the release of a large number of inflammatory factors, the counteracts the dendritic cells.Th2 cells release inflammatory factors (IL-33, IL4, IL-10) that act in conjunction with the IgE receptor on macrophages, causing them to release IgE and other inflammatory factors (TNF-α). GM-CSF released by macrophages, IL-5 released by ILC2 cells and Th2 cytokines together stimulate eosinophils. Factors in eosinophils (IL-5) stimulate B cells to become memory B cells with inflammatory factors to stimulate a large amount of IgE; ECP, MBP and inflammatory factors in eosinophils act on mast cells and basophils, causing them to degranulate and produce a large number of compounds, such as histamine, which stimulate the mucous membranes of the nasal cavity and aggravate the inflammatory response.

Neuroimmune Modulation

Currently, research on the bidirectional regulatory mechanisms between the central and peripheral systems, as well as the peripheral neural-immune networks in AR, is intensifying.[21] Studies have demonstrated that the nasal mucosa is innervated by sensory, parasympathetic, sympathetic, and nonadrenergic noncholinergic nerves. Additionally, transient receptor potential (TRP) ion channels are expressed on the surface of submucosal sensory nerve endings.[22] During an allergic inflammatory response, when the nasal mucosa is exposed to external stimuli, the parasympathetic nervous system becomes predominant and simultaneously activates sensory nerves. This results in the release of neuropeptides—such as substance P, neuropeptide Y, and vasoactive intestinal peptide—from peripheral neurons. These neuropeptides not only enhance the activation of eosinophils, key players in the immune response, but also promote the release of inflammatory mediators, facilitating the systemic transmission of inflammatory signals through the nervous system.[23,24] Advancing the "lung-brain axis" theory, the concept of the "nose-brain axis" has emerged. Sensory nerve endings in the nasal mucosa detect external stimuli and transmit signals to the central nervous system (CNS); the CNS subsequently processes this information and sends efferent signals to peripheral target organs, modulating the immune system.[25] Research focusing on the trigeminal nerve has revealed that neuropeptides released from nerve endings in AR patients, such as substance P and calcitonin gene-related peptide (CGRP), can act on the CNS. This interaction enhances central efferent signaling and lowers the threshold for external stimuli, thereby increasing the body's responsiveness to early allergic reactions and exacerbating the progression of these reactions. As a result, symptoms such as nasal congestion and rhinorrhea are exacerbated. Furthermore, the olfactory nerve may sustain damage, leading to olfactory dysfunction[26,27] (Figure 2).

Inflammatory Response

Inflammation is implicated in all phases of allergic disease pathogenesis, spanning onset, progression, and resolution. AR induces an imbalance in cytokine production. Following allergen exposure, T-lymphocytes undergo abnormal cytokine secretion, with Th2 cells releasing elevated levels of IL-4, IL-5, and IFN-γ, while Th17 cells secrete elevated levels of IL-17 and TGF-β. These

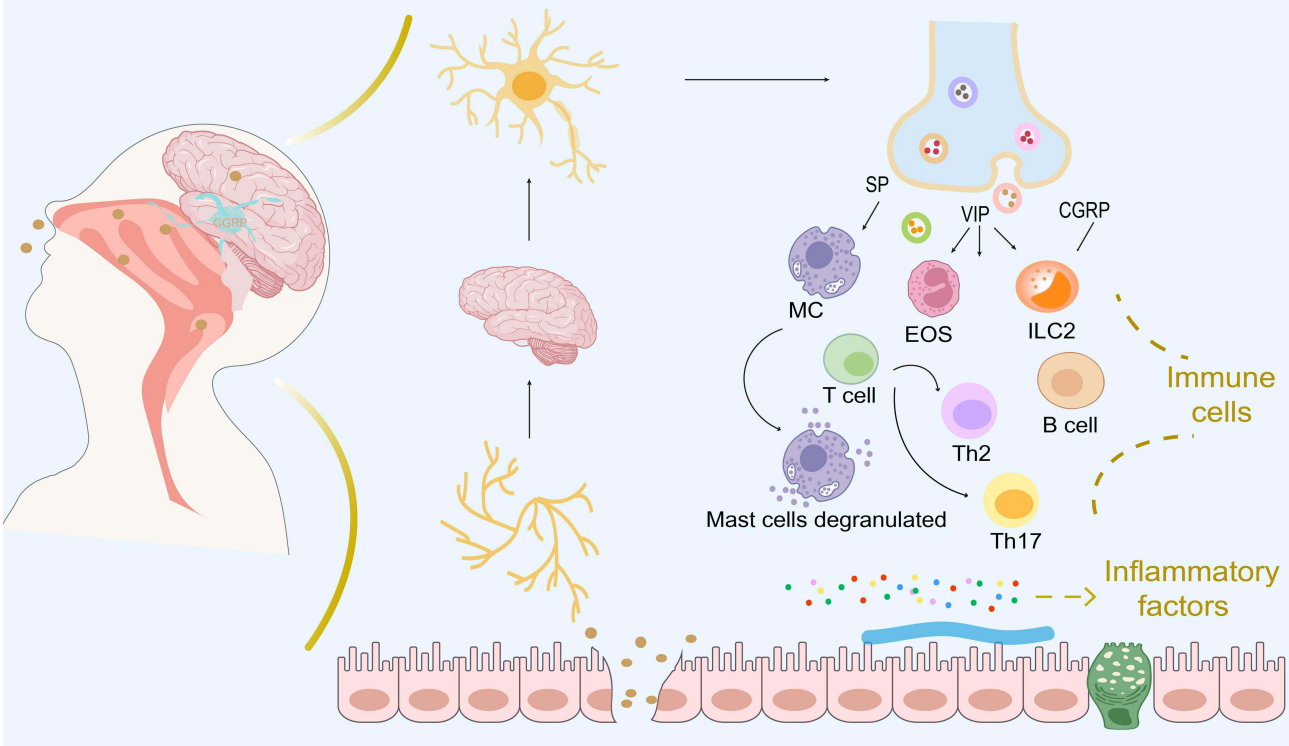


Figure 2 Allergens enter the nasal cavity, contact the nasal mucosa, stimulate the sensory nerves, parasympathetic nerves, and sympathetic nerve endings in the mucosa, and the signals from the nerve endings are uploaded to the ganglia of the brain, which cause the neurons to release a large number of neurotransmitters (SP, VIP, and VGRP), and the neurotransmitters activate the immune cells so that they can trigger inflammatory reactions, producing a large number of inflammatory factors that counteract the mucous membranes of the nasal cavity and damage the mucous membrane tissues, thus causing metamorphic reactions.

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cytokines critically drive the subsequent inflammatory response.[28] IL-4 serves as a pivotal cytokine that induces Th2 cell differentiation. It demonstrates particular importance in vitro, stimulating proliferation of activated B and T cells, enhancing IgE secretion, and exacerbating inflammatory symptoms.[29] IL-5 promotes the proliferation, chemotaxis, and activation of eosinophils, triggering the release of cytotoxic and neurotoxic proteins—ECP and MBP. These proteins induce nasal mucosa damage and potentiate glandular secretion, clinically manifesting as nasal congestion, itchy nose, rhinorrhea, and sneezing.[30] Additionally, eosinophils themselves release IL-5, creating a positive feedback loop that prolongs inflammation and complicates the treatment of AR. IL-17, a pro-inflammatory cytokine produced by activated CD4+ T lymphocytes, initiates early inflammatory responses. IL-17 acts on various cell types and tissues, inducing mesenchymal stromal cells to secrete inflammatory and hematopoietic cytokines, thereby amplifying inflammation by promoting the release of neutrophil chemokines.[31,32] Conversely, TGF-β inhibits the differentiation of T cells into regulatory T (Treg) cells and promotes their differentiation toward the Th17 lineage. As potent pro-inflammatory effector cells, Th17 cells enhance the recruitment of inflammatory cells through the secretion of inflammatory mediators, thereby amplifying the inflammatory response and sustaining inflammation. Furthermore, TGF-β suppresses the maturation and differentiation of immune cells, leading to increased expression in the nasal mucosa, histopathological alterations, and even tissue remodeling of the nasal mucosa.[33,34] Accumulating evidence suggests that the key factor contributing to AR recurrence is the persistence of localized inflammation, termed minimal persistent inflammation (MPI).[35] Regardless of whether Th17, Th1/Th2, or ILC2 cells drive Th2 dominance during the minimal persistent inflammation phase, this Th2 bias may lead to a localized inflammatory cell infiltration in the nasal mucosa. Such Th2 dominance may cause the accumulation of AR-targeted cells at local sites, thereby triggering a more rapid and severe reaction upon re-exposure to the allergen.[36,37]

Oxidative Stress

Redox components are frequently implicated at multiple stages of disease progression, and AR arises from airway inflammation as well as an imbalance between oxidative and antioxidant systems. Upon allergen exposure, the immune system initiates an inflammatory response, leading to the excessive production of reactive oxygen species (ROS), which may exert toxic effects on various organs.[38] Under normal physiological conditions, the antioxidant defense system in a healthy individual can partially neutralize ROS. However, inflammatory processes can disturb the equilibrium between oxidants and antioxidants, leading to oxidative stress. This disruption subsequently activates inflammatory signaling pathways, promotes cell-mediated immune responses, and creates an oxidative milieu that further amplifies ROS generation, ultimately causing tissue injury and aberrant pathophysiological alterations.[39,40] In a reciprocal manner, oxidative stress activates inflammatory cells and mediators, thereby accelerating and intensifying the inflammatory cascade. The Nrf2/HO-1 signaling pathway is closely associated with oxidative stress. Nrf2, an intracellular transcription factor involved in the cellular response to oxidative stress, plays a key role in defending the antioxidant system; nevertheless, its excessive activation may disturb the homeostasis of oxidative stress.[41] Oxidative stress also activates the transcription factor NF-κB, which undergoes oxidation by H_2O_2, leading to the degradation of its inhibitory subunit (IκB). Consequently, NF-κB is released and translocates into the nucleus, where it acts as a transcription factor that regulates gene expression in inflammatory, immune, and acute-phase responses. Currently, malondialdehyde (MDA) levels, along with the activation of signaling pathways such as Nrf2 and NF-κB, are commonly used as biomarkers of oxidative stress to assess the extent of oxidative stress in AR.[42]

Intestinal Flora

The gut serves as the primary site of host-microbe interactions, where the host's immune system is continually shaped and regulated by the gut microbiota throughout life. The intestinal microbiota maintains a dynamic equilibrium over time, which is supported by the mucus layer, the epithelial barrier, and immune defenses within the gut. The immune barrier comprises gut-associated lymphoid tissues, the lamina propria, and intraepithelial lymphocytes. Key cellular components include intestinal epithelial cells (IECs), DCs, Treg cells, Th17 cells, group 2 innate lymphoid cells (ILC2), and other immune cell types.[43] Research has demonstrated that the gut can transport microbial antigens to the thymus through dendritic cells, facilitating microbe–T cell interactions within the thymus. This indicates that alterations in the gut microbiota can influence the balance between effector T cells and regulatory T (Treg) cells.[44] Delayed microbial colonization, decreased microbial diversity, and structural imbalances in the gut microbiota can lead to abnormal

metabolic processes, thereby compromising immune tolerance in the intestinal mucosa. Pathogenic microorganisms influence both the innate and adaptive immune responses through the "gut-organ axis" by modulating immune mediators and promoting the onset of allergic diseases. The interaction between T cells, B cells, and the gut microbiota supports the development of intestinal mucosal immunity, helping prevent the invasion of exogenous pathogens and contributing to the establishment of systemic immune tolerance and homeostasis. The symbiotic bacterium SFB, upon colonizing the ileal epithelial layer, induces ILC3 cells to produce IL-22. This cytokine subsequently stimulates the intestinal epithelium to secrete serum amyloid A1 and A2, promoting Th17 cell differentiation and resulting in an imbalance between Th17 and Treg cells.[45,46] Intestinal epithelial cells exposed to *Aspergillus* species induce the production of IL-25, IL-33, and TSLP, which enhance the secretion of ILC2 cells and TNF-α. Consequently, this promotes the activation of type 2 inflammatory mediators, facilitates the isotype switching from IgA to IgE, and stimulates eosinophil activity. These events collectively drive a Th2-polarized immune response, exacerbating allergic symptoms such as nasal congestion and rhinorrhea[47] (Figure 3).

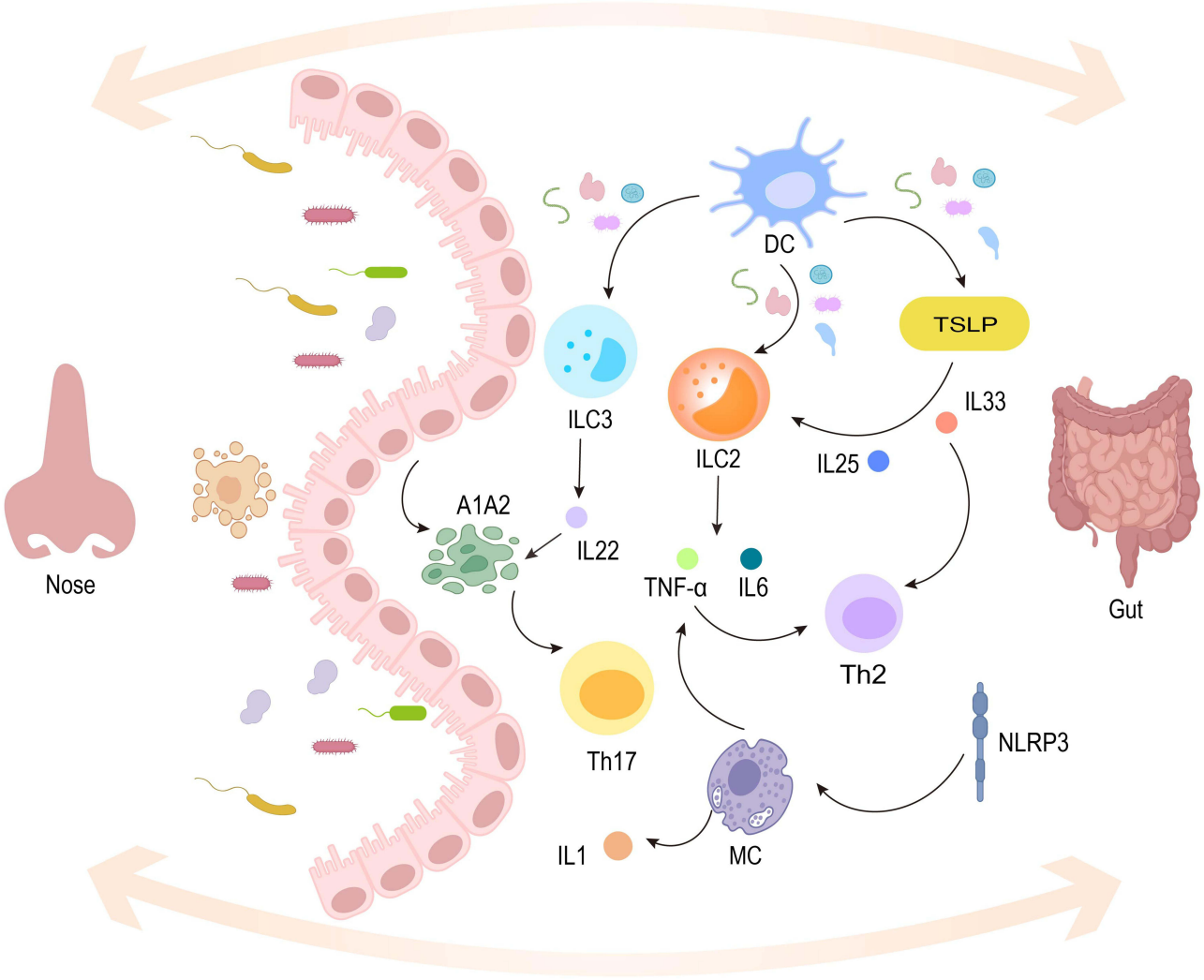

Figure 3 The nose is in an environment of metamorphosis and inflammatory reaction, immune cells are destroyed, and a large number of inflammatory factors are abnormally secreted, which downwardly stimulate the intestinal mucosa, destroying the original immune barrier and affecting the normal intestinal flora colonization, meanwhile the abnormal intestinal flora activate the dendritic cells (DC), and the commensal bacterium SFB induces the production of IL-22 from the ILC3, and the intestinal epithelium induces the production of the serum amyloid proteins, A1 and A2, by the IL-22, promotes the development of Th17 cells, leading to Th17/Treg imbalance. IL-25, IL-33, and thymic stromal lymphopoietin (TSLP) produced by the intestinal epithelial cells affected by Aspergillus phylum promote the secretion of large amounts of ILC2 cells and TNF-alpha, activate Th2 inflammatory mediators, and promote the isotypic conversion of IgA to IgE, while at the same time, the body is in the midst of a Th2 immune response due to the influence of NLRP3 on the eosinophil granulocytes in vivo.

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Cellular Autophagy

Cellular autophagy and its associated regulatory proteins critically regulate essential physiological processes, including inflammatory responses, immune function, cell proliferation, and programmed cell death.[48] Basal autophagy levels maintain cellular homeostasis, which is a prerequisite for cell survival. However, pathogenic stimuli—such as infection, oxidative stress, or nutrient starvation—can induce excessive autophagy, triggering uncontrolled degradation of cellular components. This culminates in autophagic cell death and promotes disease pathogenesis.[49] Autophagy directly contributes to AR development. Upon allergen exposure, antigen-presenting cells (APCs) utilize autophagy to phagocytize the antigens and subsequently deliver them to the lysosome for degradation, thereby facilitating antigen presentation. Notably, autophagosomes serve as the primary vehicles for transporting exogenous antigens to lysosomes and constitute key sources of intracellular MHC class II (MHCII) antigens. In naïve T cells, autophagy levels dictate T cell differentiation fates, indicating its essential role in T cell development and selection. Conversely, in mature T cells, autophagy is indispensable for maintaining homeostasis, survival, and effector functions. Crucially, autophagy modulates T cell responses both quantitatively and qualitatively.[50] For instance, T cell receptor (TCR) stimulation upregulates autophagic flux. Genetic impairment of autophagy-related genes reduces T cell production and blunts proliferation upon antigenic challenge. Consequently, this disrupts Th cell polarization and accelerates AR progression.[51] Autophagy further regulates B cell development and modulates antibody secretion. B cell activation induces the assembly and activation of the ATG14-Beclin-p150-PI3 kinase complex, which orchestrates autophagosome initiation and maturation. This autophagy-mediated regulation in eosinophils ultimately affects nasal mucosal epithelium, nasal vasculature, glandular secretion, and AR pathogenesis.[52,53]

Cellular Pyroptosis

In recent years, cellular pyroptosis has emerged as a key research focus across various disciplines, including allergic diseases, inflammatory disorders, and immune-related conditions. Accumulating evidence suggests that the activation of the NLRP3 inflammasome in nasal mucosal epithelial cells can trigger either classical or non-classical caspase (1/3/4/5/8/11)-mediated pyroptotic pathways, thereby activating GSDMD or GSDME. Subsequent pore formation in the plasma membrane triggers pyroptotic cell death and the release of inflammatory cytokines. This cascade amplifies the inflammatory response through a positive feedback loop. Moreover, caspase-3 and caspase-8 promote the progression of allergic diseases through induction of pyroptosis, while inflammatory mediators such as IL-1β further exacerbate the development of AR.[54,55]

Mitochondria

Mitochondria are double-membrane-bound organelles, consisting of the outer and inner membranes, that play a central role in cellular metabolism, redox regulation, calcium homeostasis, and cell signaling. They generate the majority of cellular energy in the form of adenosine triphosphate (ATP) through oxidative phosphorylation—a process that also contributes to the production of reactive ROS. Additionally, mitochondria act as key mediators of inflammatory responses. Under pathological conditions, mitochondrial dysfunction leads to the release of mitochondrial components into the extracellular environment, thus driving inflammatory reactions. This process involves excessive generation of mitochondrial ROS (mtROS), which results in the accumulation of oxidative free radicals and subsequent damage to both mitochondria and cells. These events promote the expression of pro-inflammatory cytokines, enhance cellular sensitivity to inflammatory signals, and activate inflammasomes, thereby contributing to the development of inflammatory diseases.[56] Under bidirectional regulation, oxidative stress can alter mitochondrial morphology and structure, leading to mitochondrial dysfunction. Emerging evidence indicates that ROS serve as key signaling molecules in the activation of the NLRP3 inflammasome. Elevated mtROS production from impaired mitochondria further facilitates NLRP3 inflammasome activation and aggravates AR progression. However, mitochondria can initiate mitophagy—a selective form of autophagy—to eliminate damaged mitochondria through the autophagosome-lysosome pathway. This process limits excessive ROS production and maintains mitochondrial homeostasis, ultimately suppressing the development of inflammatory diseases[57,58] (Figure 4).

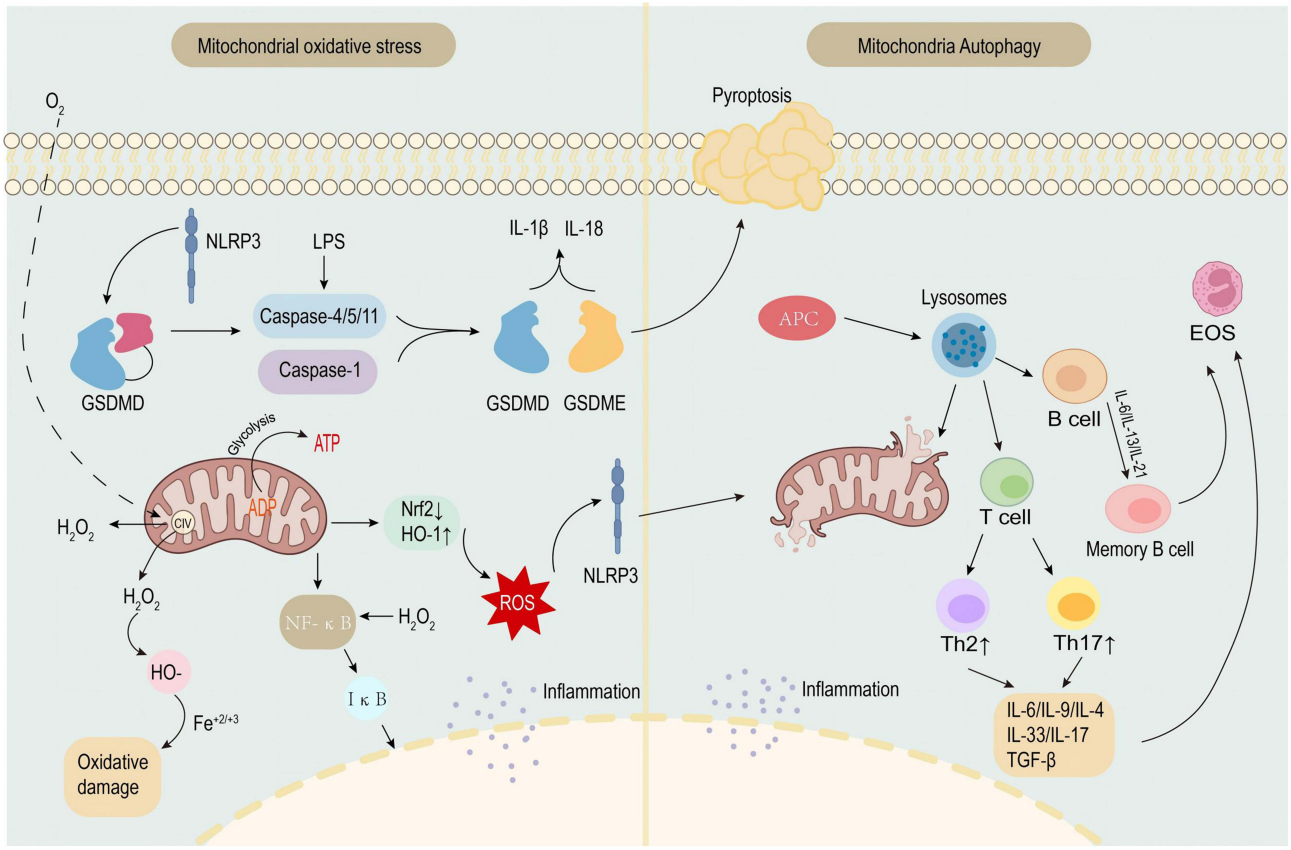

Figure 4 ADP in mitochondria is converted to ATP through glycolysis, while CIV releases H2O2 outward, and hydroxide ions in H2O2 combine with iron ions to form oxidative damage. H2O2 released by mitochondria affects the NF-κB pathway, causing it to separate IκB to invade the nucleus; mitochondria down-regulate Nrf2 and up-regulate HO-1, activating the oxidative stress response, and excess reactive oxygen species (ROS) stimulates NLRP3 inflammatory vesicles, which activate the GSDMD to initiate the caspase-1/3/4/5/8/11-mediated pyroptosis pathway, causing the GSDMD and GSDME to release inflammatory factors (IL-1β, IL-18), causing an inflammatory response into the nucleus. Antigen-presenting cells (APC) export to lysosomes, causing B cells to be transformed into memory B cells by inflammatory factors (IL-6, IL-13, IL-21), and lysosomes cause abnormal secretion of inflammatory factors by Th2 and Th17 cells in T cells, activating eosinophils and exacerbating metamorphic reactions.

Chinese Medicine Theory of AR

The treatment of AR is critically important, as untreated or inadequately managed cases frequently lead to complications including olfactory dysfunction, allergic conjunctivitis, allergic asthma, and enteritis. Although contemporary medical guidelines can ameliorate certain symptoms, they commonly induce adverse effects that significantly compromise patients' psychological well-being and may precipitate additional health complications. Currently, extensive domestic clinical studies have reported that an integrative medicine approach combining TCM with Western medicine delivers superior treatment outcomes for AR. TCM provides multi-pronged benefits in early-stage AR prevention and therapy. Crucially, it can effectively decelerate disease progression, reduce complication rates, alleviate core symptoms such as nasal obstruction and rhinorrhea, and significantly improve patients' quality of life.

Historical Progress of Chinese Medicine in the Treatment of AR

Although AR is classified as a modern disease entity, its earliest documentation in TCM theory appears as "nasal obstruction with sneezing" in *The Book of Rites: Monthly Ordinances*. Liu Wansu, a physician of the Jin Dynasty, further elucidated this condition in *Exploration of Original Diseases from Plain Questions*, characterizing it by watery nasal discharge accompanied by paroxysmal sneezing induced by nasal pruritus. These historical records demonstrate remarkably accurate clinical symptom identification of that time. During the Eastern Han Dynasty, Xu Shen's *Shuowen Jiezi* ascribed the disorder to nasal congestion caused by cold pathogen invasion, marking an initial

understanding of its pathomechanism. This conceptual shift advanced medical understanding beyond symptomatic observation toward foundational etiological exploration.[59]

The TCM Pathogenesis of AR

The core of Chinese medicine theory lies in its holistic perspective and pattern identification, which views the human body as an integrated whole in which the organs coordinate, support, interact, and influence one another.

Within TCM theory, AR is pathologically ascribed to deficiencies in the lung, spleen, and kidney organs, compounded by vital qi insufficiency and impaired Defensive Qi mechanisms. These impairments diminish the body's adaptive capacity to environmental challenges, manifesting clinically as nasal pruritus, paroxysmal sneezing, rhinorrhea, frequent olfactory decline, ocular irritation, or pharyngeal discomfort. A systematic analysis of historical and contemporary etiological frameworks reveals that TCM classifies AR pathogenesis under four primary syndromes: Lung Qi deficiency with cold syndrome, Spleen Qi deficiency syndrome, kidney qi deficiency syndrome, and Lung Meridian exogenous heat syndrome. Fundamentally, TCM anatomically designates the nose as the external orifice of the lungs, where the pulmonary system governs nasal discharge production. When Lung Qi deficiency and Wind-Cold invasion occur, the Defensive Qi barrier weakens, permitting penetration of exogenous wind and pathogens. This compromises pulmonary qi diffusion and distribution of bodily fluids, leading to the accumulation of pathogenic factors in nasal pathways. Concurrently, since the spleen constitutes the postnatal foundation of vitality, Spleen Qi deficiency impairs biosynthesis of qi and blood. Resultant nasal mucosal dehydration leads to inferior turbinate hypertrophy and persistent clear rhinorrhea. Furthermore, Kidney Yang deficiency attenuates the warming function, resulting in chronically cold nasal cavities that are vulnerable to pathogen invasion. This pathophysiological cascade leads to watery nasal discharge and refractory rhinorrhea.[60]

The core therapeutic philosophy in TCM is fundamentally rooted in the dual principles of "tonification and circulation", with nasal congestion pathogenesis specifically attributed to visceral organ deficiencies. Treatment strategies prioritize warming Lung Qi, fortifying the spleen and stomach systems, tonifying kidney yang, replenishing essence, dispelling exogenous pathogens, and eliminating pathogenic factors. This comprehensive approach aims to restore systemic physiological equilibrium. Guided by TCM syndrome differentiation principles, herbal medicines are selected based on pharmacodynamic actions: symptom palliation, bodily tonification, orifice-opening effects, and activation of blood circulation. Medicinal formulations—whether decoctions or finished products—strictly adhere to the herbal hierarchy of *jun* (monarch), *chen* (minister), *zuo* (assistant), and *shi* (envoy) roles. Critically, TCM's holistic, evidence-based methodology simultaneously achieves three therapeutic objectives: ameliorating localized symptoms, modulating systemic physiological functions, and addressing root etiologies while enhancing immunological competence.[61]

The TCM for AR

The pathogenesis of AR in TCM primarily originates from the lungs and is closely related to the functions of the spleen and kidneys, with pathological manifestations mainly occurring in the nose. Exogenous etiological factors predominantly involve wind, cold, and heat pathogens. Consequently, TCM therapeutic principles emphasize dispelling wind pathogens, regulating lung function, strengthening the spleen, nourishing kidney yin and yang, and clearing nasal passages by expelling cold pathogens. In addition to decoctions and herbal formulas, proprietary TCM preparations such as Tongqiao Biyan Capsules, Sanfeng Tongqiao Dropping Pills, and Wenfei Zhiliu Dan are commonly used. This systematic review examines contemporary clinical research on AR management through TCM, specifically analyzing two therapeutic approaches: internal interventions via herbal administration and external therapies utilizing medicinal fumigation and topical paste applications.

Chinese Medicinal Herbs

Table 1 summarizes the relevant herbs used in the treatment of AR, the mechanisms of which may involve the inhibition of inflammatory responses, the reduction of oxidative stress induced by oxidative products, and the modulation of immune cell activity.

Table 1 Summary of the Mechanism of Action of Chinese Herbs in the Treatment of AR

Chinese Medicinal Herbs (Latin Name/ English Name/Chinese Name)	Portion Used	Research Object and Model	Method of Administration	Dose	Medication Duration	Mechanism of Action	Reference
Centipeda minima (L.) A.Braun & Asch. (E bu shi cao)	Volatile oil emulsion	SD rats	Nasal drip	10μL/3times/d	15d	Inhibits the production of eosinophils and mast cells, reduces pathologic changes in nasal mucosal tissue	[62]
Astragalus mongholicus Bunge (Huangqi)	Granules	Children with allergic rhinitis	Oral	7.5g/3times/d	2months	Up-regulates Th1 cell function (IL-2, IFN-γ), down-regulates Th2 cell function to inhibit secretion of IL-4, IL-10	[63]
Xanthium strumarium L. (Cangerzi)	Water Extract	BALB/c mice	Gavage	High4g/kg/d, medium2g/kg/d, low1g/kg/d	14d	Lowering serum levels of IIL-4 and elevating levels of IFN-γ, thereby regulating Th1 /Th2 cell balance and lowering serum IgE levels	[64]
Angelica sinensis (Oliv). Diels (Baizhi)	Emulsion	C57BL/6mice	Gavage	30mg/kg/d	7d	Down-regulation of Th17 ratios (IL-6, IL-17) and up-regulation of Treg ratios (IL-10, TGF-β1) in serum and nasal lavage fluid restored Th17/Treg balance	[65]
Magnolia biondii Pamp. (Xinyi)	Essential oil	SD rats	Nasaldrip	1.0mL/d	On days 1,7, 14,21-27	Reduces Th2 cytokine concentrations (IL-4, IL-5), increases Th1 cytokine concentrations (IL-2, IFN-γ), improves Th1/Th2 cell balance, and reduces inflammatory response in allergic rhinitis	[66]
Saposhnikovia divaricata (Turcz). Schischk. (Fangfeng)	Water Extract	BALB/c mice	Gavage	Viscosity:5mg/mL,0.2mL/d	2weekends	Inhibits TLR4/TRAF6/IκBα/NF-κB p65 pathway activation, reduces mast cell infiltration and inflammatory cell number, reduces Treg/CD4+ T cell ratio, regulates body immunity, and improves allergic rhinitis symptoms	[67]
Prunus mume (Sieh). Sieb.et Zucc.(Wumei)	Water Extract	Mice	Gavage	10g/kg/d	On days22, 24, 26	Inhibition of mast cell exosomal secretion MMP9 regulates Treg/Th17 immune homeostasis	[68]
Mentha canadensis L. (Bohe)	Water Extract	SD rats	Oral	High300mg/kg/d, low100mg/kg/d	1hour before the experiment	Inhibits histamine release from mast cells and improves nasal clinical symptoms	[69]
Asarum heterotropoides F.Schmidt (Xixin)	volatile oil	FMMU guinea pig	Oral	0.45g·kg-1/d	10d	Reduces histamine release levels	[70]
Aconitum carmichaeli Debeaux (Fuzi)	Total epiphyllum alkali	Guinea pig	Gavage	Viscosity:2.6g/mL scope:36.0g/kg/d-109.8g/kg/d	10d	Reduces serum histamine levels and improves local infiltration of nasal mucosa inflammation	[71]
Schisandra chinensis (Turcz).Baill. (Wuweizi)	Schizandrin	Mice	Intraperitoneal injection	10mg/kg/d	8weekend	Reduces levels of IL-5, IL-6, soluble F-selectin and eosinophil chemokines, improves nasal function and relieves nasal symptoms	[69]
Bombyx mori Linnaeus (Jiangcan)	Water Extract	Mice	Gavage	High5g/10mL/d, low1g/10mL/d	10d	Regulates the levels of IL-4 and IFN-γ and restores Th1/Th2 balance	[72]
Cicadae Periostracum (Cantui)	Dry powder	KM mice	Intraperitoneal injection	Sodium hydroxide solution fusion ratio:1:10, 1:15, 1:20, 1:25, 1:30	5d	Altered free radical scavenging and inhibition of oxidative stress	[73]
Scorpion (Quanxie)	Soaking liquid	SD rats	Hypodermic injection	High1.04kg^{-1}/d, medium0.52kg^{-1}/d, low0.26kg^{-1}/d	28d	Modulation of inflammatory factors	[74]

Plant-Based Herbs

Centipeda minima (L.) A.Braun & Asch., a medicinal herb first documented during the Southern Tang Dynasty, is formally recorded in both the *Shiliao Bencao* (Food and Herbal Medicine) and the *Compendium of Materia Medica*. As described in the herbal section of the "*Compendium*", this plant exhibits pungent and warm properties, primarily acting on the Lung Meridian. The 2015 Edition of the Chinese Pharmacopoeia specifies its functions: dispelling Wind-Cold pathogens, unblocking nasal orifices, and suppressing cough. It is clinically applied for Wind-Cold headaches, productive cough, nasal congestion, impaired nasal ventilation, and rhinorrhea, serving as a key pungent-warm detoxifying agent.[75] With over one millennium of documented clinical application, this herb demonstrates significant efficacy in treating AR according to domestic and international research. Notably, it eliminates drug-resistant bacterial plasmids.[76] Pharmacological studies on *Centipeda minima*'s active constituents reveal that its extract inhibits AR pathogenesis by modulating the Th2-IgE-mast cell (basophil)-eosinophil immune axis, consequently reducing histopathological damage to nasal mucosa.[62]

Astragalus mongholicus Bunge is a fundamental herb in TCM used for replenishing vital energy (Qi). This species comprises two primary variants: Mongolian astragalus (*Astragalus membranaceus var. mongholicus*) and membranous milkvetch (*Astragalus membranaceus var. membranaceus*). Pharmacologically, it has a sweet flavor and a mildly warm property, with documented efficacy in Qi tonification, surface fortification, immune enhancement, and pathogenic factor elimination. Clinically, it is used to treat syndromes of Lung Qi deficiency and compromised immunity and serves as a core component in classical formulations including Yupingfeng San (Jade Screen Powder), Buzhong Yiqi Decoction, and Guizhi Jia Huangqi Decoction.[77] *Astragalus mongholicus* Bunge synthesizes bioactive constituents, primarily astragaloside IV and polysaccharides. Studies have shown that its standardized extracts modulate inflammatory mediators and regulate immune cell populations—particularly Th1, Th2, Treg, and Th17 cells—in the nasal mucosa and systemic circulation of AR patients.[63]

Xanthium strumarium L. is pharmacologically documented in classical Chinese medicine texts. This herb has a warm nature and exhibits pungent and bitter flavor properties. Its therapeutic actions encompass dispelling wind-cold, clearing nasal obstruction, expelling wind, and eliminating dampness, establishing its primary use in the management of nasal disorders.[78] The bioactive constituents of *Xanthium strumarium* L., particularly n-butanol-soluble compounds, modulate the LAT/PLCγ1/PKC signaling cascade. This molecular mechanism suppresses mast cell degranulation, reduces inflammatory mediator release, and enhances nasal vascular tone through dual pathways: α-adrenergic receptor-mediated vasoconstriction and nasal mucosal contraction. Collectively, these physiological responses alleviate rhinitis symptoms, including turbinate hypertrophy and nasal congestion.[64]

The *Shen Nong Ben Cao Jing* classically records that *Angelica sinensis* (Oliv). Diels has warm and pungent properties, functioning to dispel wind pathogens, eliminate dampness, reduce swellings and discharge pus, as well as alleviate pain through orifice-opening mechanisms. This herb has been historically used in clinical practice for nasal congestion, cephalalgia, and related conditions.[79] Pharmacologically, *Angelica sinensis* (Oliv). Diels emulsion inhibits the adherence of vascular endothelial cells and circulating leukocytes to organs and vascular walls. This action prevents inflammatory cell infiltration into nasal mucosa, thereby suppressing localized inflammatory responses.[65]

Magnolia biondii Pamp. possesses acrid and warm properties; its acridity disperses wind pathogens, while its warmth counteracts cold syndromes. This herb is clinically indicated for constitutionally deficient individuals who are predisposed to wind-related diseases. In TCM practice, synergistic herbal pairs—including *Magnolia biondii* Pamp. and *Xanthium strumarium* L., *Magnolia biondii* Pamp. and *Angelica sinensis* (Oliv). Diels, and *Magnolia biondii* Pamp., along with *Astragalus mongholicus* Bunge and *Saposhnikovia divaricata* (Turcz). Schischk.—effectively disperse Wind-Cold pathogens and unblock nasal orifices. These combinations demonstrate therapeutic efficacy against rhinitis through both oral and topical administration.[80] Advancements in phytochemical isolation have enabled the purification of volatile oils from *Magnolia biondii* Pamp., revealing that cinnamyl ester derivatives potently inhibit histamine release, attenuate inflammatory cascades, and enhance immunomodulatory functions. Critically, these bioactive components suppress the release of the neuropeptide substance P during inflammation and inhibit type I hypersensitivity reactions. This multi-target/multi-component synergistic mechanism provides a scientific basis for AR treatment.[66]

Saposhnikovia divaricata (Turcz). Schischk., primarily sourced from Hebei, Heilongjiang, and Inner Mongolia provinces, exhibits therapeutic actions including wind-dispelling, eliminating pathogens from the pulmonary system, alleviating Qi stagnation, and relieving nasal congestion. This makes it an essential herb for treating exogenous lung diseases, particularly those involving Lung Qi deficiency and cold syndromes caused by external pathogens. Given the TCM principle that "the lungs govern the nose", such pathological imbalances frequently manifest as rhinopathies.[81] Pharmacological studies have shown that *Saposhnikovia divaricata* polysaccharides inhibit cutaneous allergic reactions by 55.51% without inducing dermal irritation. Mechanistically, the herb's extract exerts anti-AR effects by modulating the TLR4/TRAF6/IκBα/NF-κB p65 signaling pathway in nasal mucosa. This pathway modulation critically enhances immunoregulatory responses through the suppression of pro-inflammatory nuclear translocation.[67]

Asarum heterotropoides F.Schmidt, initially documented as a superior-grade herb in the *Shen Nong Ben Cao Jing* during the Han Dynasty, exhibits dual therapeutic actions: dispersing exogenous Wind-Cold pathogens externally and eliminating endogenous yin-cold internally. This herb constitutes an essential component in classical formulations such as Mahuang Fuzi Xixin Decoction and Xiaoqinglong Decoction.[82] These pharmacological effects are principally mediated by methyl eugenol, a bioactive constituent of *Asarum heterotropoides* F. Schmidt. Mechanistically, methyl eugenol regulates the distribution of T-lymphocyte subsets, decreases the Th/Ts cell ratio, inhibits β-endorphin synthesis, and attenuates inflammatory cell infiltration in mucosal tissues.[70]

Insect Herbs

Bombyx mori Linnaeus, commonly known as the white silkworm, is a widely used insect-derived medicinal agent in China. This substance has a light aromatic odor and exhibits bidirectional therapeutic movement: ascending action targets the lungs to regulate and descend Lung Qi, disperse wind pathogens, and eliminate phlegm.[83] According to TCM principles, "the lungs govern the nose", and balanced Lung Qi along with strong Defensive Qi (Wei Qi) helps maintain nasal sensitivity and physiological responsiveness. *Bombyx mori* Linnaeus contains bioactive constituents such as proteins, enzymes, amino acids, and fatty acids. It stimulates adrenal cortical activity, increasing the secretion of adrenocorticotropic hormone. This endocrine modulation helps alleviate airway inflammation and allergic responses by restoring Th1/Th2 immune homeostasis, thereby significantly reducing the symptoms of AR.[72]

Cicadae Periostracum exhibits a sweet-salty flavor, a cool nature, and light ascending properties. Therapeutically, it effectively disperses wind pathogens, promotes rash eruption, and alleviates pruritus. Wind pathogens are characterized by rapid migration and transformative capacity. When exogenous wind invades the lungs and ascends to the nasal regions, it induces nasal discomfort.[84] This insect-derived material contains bioactive constituents, including chitin, proteins, inorganic salts, amino acids, and trace elements. These components enhance lipid metabolism and confer potent antioxidant activity, thereby attenuating oxidative stress damage to the nasal mucosa and ameliorating nasal symptoms such as itching.[73]

Active Ingredients of TCM

With the advancement of modern medicine, a considerable portion of research has increasingly focused on herbal monomers. Table 2 presents a summary of several effective active ingredients derived from TCMs that have been reported to exert therapeutic effects on AR in recent years.

Curcumin, a natural polyphenol and one of the primary bioactive constituents of turmeric, has been used in traditional medicine for over 4,000 years. Accumulating evidence confirms that curcumin possesses a wide range of pharmacological properties, including anti-inflammatory, antioxidant, free radical-scavenging, antiviral, hepatoprotective, nephroprotective, and anticancer effects, with minimal toxic or adverse effects.[114] Recent studies have shown that curcumin can inhibit histamine release, thereby exerting anti-allergic effects in animal models of both type I and type IV hypersensitivity reactions. Through immunomodulatory mechanisms, curcumin suppresses cell proliferation, mitigates the secretion of inflammatory cytokines, and reduces oxidative damage. It also inhibits the activation of nuclear factor-kappa B (NF-κB) and activator protein-1 (AP-1) transcription factors, which contributes to the suppression or delay of AR progression.[115] Research also indicates that curcumin enhances the activity of heme oxygenase-1 (HO-1) and superoxide dismutase 2 (SOD2) in hypertrophied inferior turbinate tissues of individuals with AR, thereby alleviating nasal mucosal injury caused by oxidative

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Table 2 Summary of the Mechanism of Action of Plant Compounds in the Treatment of AR

Plant Compounds	Compound Molecules	Source(Latin Name/ Chinese Name)	Research Object and Model	Method of Administration	Dose	Medication Duration	Mechanism of Action	Reference
Polyphenol	Curcumin	*Curcuma longa* L. (Jianghuang)	Inferior nasal concha	Intraperitoneal injection	5μm/d	7d	Activation of the Nrf2/HO-1 pathway reduces reactive oxygen species (ROS) and increases heme oxygenase (HO-1) and superoxide dismutase (SOD2) activity	[85]
	Resveratrol	*Reynoutria japonica* Houtt. (Huzhang)	Balb/c mice	Intranasal drip	High400μg/d, low200μg/d	7d	Inhibition of TXNIP - oxidative stress signaling pathway and reduction of IgE, PGD, LTC release to reduce ECP-stimulated mast cell degranulation to release histamine and downregulate inflammatory mediators (IL-4, IL-5, IL-6, IL-33)	[86]
	Gallic acid	*Rhus chinensis* Mill. (Wubeizi)	ICR mice	Intraperitoneal injection	High80mg·kg–[1]/d, medium40mg·kg–[1]/d, low20mg·kg–[1]/d	13d	Modulation of NF-κB/ p38 MAPK pathway activity, inhibition of mast cells, and decreased secretion of inflammatory factors (TNF-α, IL-6) in Th2 cells	[87]
	Catechin	*Callerya reticulata* (Benth). Schot (Jixueteng)	Balb/c mice	Intraperitoneal injection	High300mg/kg/d, medium150mg/kg/d, low70mg/kg/d	7d	Inhibition of NF-κB pathway activation attenuates IκBα protein degradation, affects upstream regulators of TSLP in epithelial cells, and suppresses inflammatory responses	[88]
	Magnolol	*Magnolia officinalis* Rehder&E. H.Wilson (Hopuo)	Balb/c mice	Intranasal drip	30μm/d	7d	Inhibits anti-CD3-induced cell proliferation, regulates ORAI1 and ANO1 channel-mediated ATP-induced electrolyte transport, reduces cellular infiltration and secretion of IL-22 and IL-4 inflammatory factors, and corrects Th1/Th2 imbalance	[89]
Flavonoid	Quercetin	*Panax ginseng* C.A. Mey. (Renshen)	Balb/c mice	Oral	High50mg/kg/d, medium35mg/kg/d, low20mg/kg/d	13d	Modulation of NF-κB pathway, inhibition of inflammatory cytokine production and induction of apoptosis	[90]
	Anthocyanin	*Lycium chinense* Miller (Goqi)	SD rat	Intranasal drip	0.05mL/per/3times/d	4weekends	Inhibition of IgE release from eosinophils,alteration of the ratio of Th1 and Th2 cells in the body	[91]
	Kaempferol	*Eucommia ulmoides* Oliv. (Duzhong)	Balb/c mice	Intraperitoneal injection	10mg/kg/d	7d	Inhibition of IL-32/TSLP/caspase-1 signaling pathway, down-regulation of histamine, inhibition of levels of inflammation-related proteins and infiltration of inflammatory cells	[92]
	Baicalein	*Scutellaria baicalensis* Georgi (Huangqin)	Guinea pig	Intraperitoneal injection	40mg/kg/d	10d	Blockade of JAK2-STAT5 and NF-κB signaling pathways in LPS-stimulated human mast cells to inhibit inflammatory factor release (IL-1, IL-6, IL-8, and TNF-α)	[93]
	Tanshinone	*Salvia miltiorrhiza* Bunge (Danshen)	Balb/c mice	Intraperitoneal injection	10mg/kg/d	10d	Inhibits dendritic cell maturation, increases the ratio of T-lymphocyte subsets CD3+, CD4+, and CD8+, decreases levels of inflammatory cytokines (IL-2, IL-4, INF-γ, TNF-α), and increases IL-10 levels	[94]
	Rhinocerosin	*Perilla frutescens* (L.) Britt. (Zisu)	SD rat	Intraperitoneal injection	1mg/kg/d	2weekends	Downregulation of PI3K/Akt/mTOR signaling pathway, reduction of Th1 levels and T-bet protein	[95]

	Naringenin	*Citrus aurantium* L. (Zhishi)	SD rat	Gavage	High2.0mg/mL/d, low1.0mg/mL/d	21d	Reduction of IgE, inhibition of TLR4/NF-κB/TNF-α pathway, modulation of Th17 factor, and effective improvement of nasal mucosal pathologic changes	[96]
Saponin	Hesperidin	*Citrus reticulata* Blanco (Chenpi)	Balb/c mice	Intraperitoneal injection	100mg/kg/d	28d	Inhibits TSLP/OX40L/OX40 signaling activation, reduces GATA3 expression, and modulates immune response	[97]
	Polydatin	*Reynoutria japonica* Houtt. (Huzhang)	Balb/c mice	Intraperitoneal injection	High45mg·kg−[1]/d, low30mg·Kg−[1]/d	10d	Activation of PINK1-Parkin signaling pathway promotes mitochondrial autophagy and ameliorates allergic rhinitis	[98]
	Ginsenoside	*Panax ginseng* C.A. Mey. (Renshen)	Balb/c mice	Intraperitoneal injection	High10mg/mL/d, medium1mg/mL/d, low0.1mg/mL/d	1h	Blockade of TSLP, modulation of NF-κB/RelA pathway, reduction of pro-inflammatory factors (IL-1β, IL-4), reduction of histamine release from mast cells, basophil degranulation	[99]
	Astragaloside	*Astragalus mongholicus* Bunge(Fisch). (Huangqi)	C57/BL6 mice	Intraperitoneal injection	40mg/kg/d	7d	Restoring the dynamic balance of mitochondria by regulating Drp1 activity and inhibiting the NK2 differentiation bias of NK cells, exerting an ameliorative effect on allergic rhinitis	[100]
Monoterpene	Glycyrrhizic acid	*Glycyrrhiza uralensis* Fisch. (Gancao)	SD rat	Intraperitoneal injection	High40mg.kg^{-1}/d, low20mg.kg^{-1}/d	4weekends	Inhibition of NF-κB and PI3K-Akt signaling pathway activity, regulation of Th1/Th2 balance in vivo	[101]
	Celastrol	*Tripterygium wilfordii* Hook. f. (Leigongteng)	Mast cell	Soak	2μm	1h	Increasing substance P to increase mast cell survival and altering key gene and protein expression of the PI3K/AKT/GSK3β signaling pathway in cells to weaken the immune response	[102]
	Paeoniflorin	*Paeonia lactiflora* Pall. (Shaoyao)	SD rat	Intraperitoneal injection	High150mg/kg/d, low50mg/kg/d	2weekends	Regulates cellular immune balance through IL-33/ST2 pathway, inhibits inflammation and reduces pathologic damage in allergic rhinitis	[103]
	Notoginsenoside	*Panax notoginseng* (Burkill) F.H. Chen (Sanqi)	Balb/c mice	Intraperitoneal injection	High30mg·kg −[1]/d, low 15mg·kg −[1]/d	4weekends	Inhibition of AMPK/DRP1 signaling axis and TXNIP/NLRP3 inflammatory vesicles prevents mitochondrial fission due to oxidative stress	[104]
Other	Saposhnikovia divaricate polysaccharide	*Saposhnikovia divaricata* (Turcz). Schischk. (Fangfeng)	SD rat	Intraperitoneal injection	High600mg·kg−[1]/d, medium300mg·kg −[1]/d, low150mg·kg −[1]/d	2weekends	Inhibits Th2 expression (IL-4, TNF-α, VCAM-1, IL-5), attenuates eosinophil activation, and reduces inflammatory factor release (IgE, HA, LTC4, PGD2)	[105]
	Andrographolide	*Andrographis paniculata* (Burm.f). Wall. ex Nees (Chuanxinlian)	Child with allergic rhinitis	Oral	0.6g/3times/d	2weekends	Reduction of serum IgE concentration and restoration of dysregulated Th1/Th2 ratio	[106]
	Lignans	Rheum palmatum L. (Dahuang)	ICR mice	Intraperitoneal injection	High50mg/kg/d, low25mg/kg/d	4weekends	Inhibition of receptor-proximal Syk-dependent signaling pathway suppresses mast cell activation and modulates allergic responses	[107]

(*Continued*)

Table 2 (Continued).

Plant Compounds	Compound Molecules	Source(Latin Name/ Chinese Name)	Research Object and Model	Method of Administration	Dose	Medication Duration	Mechanism of Action	Reference
	Psoralen	*Cullen corylifolium* (L.) Medik. (Buguzhi)	Nasal epithelial cell	Soak	High20μm/d, medium10μm/d, low1μm/d	2h	Inhibition of the AP-1 pathway regulates CST1 expression, thereby inhibiting IL-13-induced inflammatory response and mucus production in nasal mucosal epithelial cells	[108]
	Sinomenine	*Sinomenium acutum* (Thunb). Rehder & E. H. Wilson (Qingteng)	Balb/c mice	Intraperitoneal injection	100mg/kg/d	5d	Reduces IgE levels, decreases eosinophil infiltration, decreases IL-4 in serum and nasal mucosa, increases TGF-β, exerts immunosuppressive effects	[109]
	Vitexin	*Crataegus pinnatifida* Bunge (Shanzha)	SD rat	Intraperitoneal injection	High12mg/kg/d, medium6mg/kg/d, low3mg/kg/d	14d	Activation of Sirt1/FoxO1 pathway in nasal mucosal tissue inhibits iron death of nasal mucosal epithelial cells and reverses nasal mucosal damage	[110]
	Ephedrine	*Ephedra* (Mahuang)	SD rat	Gavage	10mg/kg/d	7d	Inhibition of TSLP and OX40L in nasal mucosal tissues modulates Th2-type immune response	[111]
	Berberine	*Coptis chinensis* Franch. (Huanglian)	Balb/c mice	Intraperitoneal injection	High100mg/kg/d, low50mg/kg/d	28d	Down-regulates the levels of IL-6, TLR4, PTGS2 and IL-1β and inhibits the proliferation of inflammatory monocytes	[112]
	Lycium barbarum polysaccharide	*Lycium chinense* Miller (Goqi)	Balb/c mice	Gavage	High100mg/kg/d, medium50mg/kg/d, low25mg/kg/d	21d	Inhibiting the expression of TLR4/NF-κB signaling pathway-related proteins, correcting the imbalance of Th1/Th2 cytokines, and reducing the production of IgE, which effectively alleviated the nasal mucosal inflammation in mice with allergic rhinitis	[113]

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stress and supporting its protective role in oxidative stress-related AR pathogenesis.[85] Additionally, curcumin administration has been shown to normalize IgE and TNF-α levels in nasal mucosal epithelial cells and serum, leading to a reduction in allergy-related nasal symptoms, such as congestion, rhinorrhea, and nasal itching.

Quercetin, a flavonoid found in plants such as *Panax ginseng* C. A. Mey. and *Codonopsis pilosula* (Franch). Nannf., is primarily present in the form of glycosides and exhibits a broad spectrum of biological activities, including antioxidant and anti-allergic effects. These effects involve immune system modulation, antiviral activity, suppression of histamine release, and reduction of pro-inflammatory cytokines.[116] Research has demonstrated that quercetin inhibits the expression of Tim1 and Tim3 in the nasal mucosa, thereby regulating the Th1/Th2 cytokine balance. This helps correct immune imbalance, repair damaged nasal mucosa, and accelerate the recovery process in AR.[117] Quercetin upregulates ovalbumin-induced levels of IκBα and cytoplasmic p65, suppressing the NF-κB signaling pathway. It also downregulates COX-2 expression in ovarian cancer cells and modulates T-lymphocyte subpopulations, contributing to the treatment of AR.[90] Previous studies have reported that quercetin intervention at varying concentrations in model rats inhibits changes in nasal axon terminal calcium ion content. Additionally, it reduces the release of neuropeptides such as substance P, calcitonin gene-related peptide (CGRP), and nerve growth factor (NGF), while inhibiting eosinophil degranulation and chemokine production. These mechanisms alter allergic responses and mitigate the severity of AR.[118] Nanotechnology-based investigations have revealed that quercetin significantly inhibits IL-4-induced NO production in nasal mucosal epithelial cells. It regulates immune function in these cells by suppressing STAT6 activation and iNOS mRNA expression, thereby improving clinical symptoms and complications associated with AR.[119]

Baicalein, a bioactive flavonoid derived from the dried root of *Scutellaria baicalensis* Georgi, demonstrates multi-faceted pharmacological properties including antiviral, anti-inflammatory, and antiplatelet and anticoagulation effects. This compound exhibits significant therapeutic efficacy against various inflammation-mediated pathologies.[120] Mechanistically, baicalein suppresses ROS generation by upregulating Nrf2 protein expression. This antioxidant activity reduces oxidative stress damage and attenuates programmed neuronal apoptosis, collectively modulating inflammatory states in AR.[121] Experimental observations demonstrate that baicalein accelerates nasal mucosal tissue regeneration and alleviates disease progression in mouse models. These therapeutic outcomes arise from the inhibition of NF-κB/STAT3/ERK pathway activation, reduction in protein phosphorylation levels, and suppression of inflammatory cell activity.[122]

Lignans constitute potent anti-inflammatory and anti-allergic phytochemicals present in Chinese medicinal herbs such as *Chrysanthemum indicum* (wild chrysanthemum), *Lonicera japonica* (honeysuckle), and *Perilla frutescens*. These botanicals are clinically employed for chronic inflammatory airway disorders, with their therapeutic mechanisms potentially involving the modulation of NF-κB, MAPK/AP-1, and JAK-STAT signaling pathways.[123] Immunocytological analyses demonstrate that lignans inhibit lipopolysaccharide (LPS)-induced macrophage polarization toward the M2 phenotype. This process progressively downregulates M1 macrophage-derived p-STAT3 while upregulating p-STAT6, consequently reducing pro-inflammatory cytokine release—particularly IL-6 and TNF-α. These findings suggest that lignans modulate inflammation by repolarizing pro-inflammatory M1 macrophages toward anti-inflammatory M2 phenotypes. This phenotypic shift alters the inflammatory microenvironment, establishing novel therapeutic paradigms for AR management.[124]

Ginsenosides represent the most extensively researched and pharmacologically active constituents in *Panax ginseng*, renowned for their anti-inflammatory, immunomodulatory, and neuroprotective properties. Using systems biology techniques, both in vivo and in vitro experiments demonstrate that ginsenoside Rg3 upregulates Nrf2 and HO-1 protein expression in the nasal mucosa, thereby ameliorating oxidative stress microenvironments. Additionally, it activates 12 differentially expressed genes and modulates 8 key metabolites, conferring cytoprotective effects on nasal mucosa and contributing to systemic homeostasis in mouse models.[125] Notably, ginsenosides exhibit a bidirectional regulatory effect on immune cells, including mast cells and macrophages. These compounds demonstrate neuroprotective properties that can restore both sympathetic and parasympathetic nervous system functions, thereby alleviating sleep disorders, anxiety, and depression associated with AR.[126] Therefore, ginsenosides constitute natural, multi-pathway, multi-target therapeutic candidates for innovative drug development.

Polydatin is the primary active constituent of *Reynoutria japonica* Houtt. Pharmacological studies have demonstrated that polydatin exerts anti-inflammatory and antioxidant effects, among other therapeutic benefits. Previous research has

indicated that polydatin administration in AR mice enhances nasal mucosal barrier function by upregulating tight junction proteins, such as occludin and claudin-1. Moreover, polydatin increases the activities of superoxide dismutase (SOD) and catalase (CAT), reduces MDA levels, and modulates the pyroptosis pathway by regulating the ROS/NLRP3/Caspase-1 signaling cascade. These findings confirm polydatin's capacity to regulate oxidative stress, alleviate AR symptoms, and offer novel targeted therapeutic strategies for clinical applications.[127]

Glycyrrhizic acid, a pentacyclic triterpenoid derivative, constitutes a principal bioactive component in *Glycyrrhiza* licorice root. It demonstrates pharmacological properties analogous to glycyrrhizin, suppressing pro-inflammatory mediators and attenuating ROS release. 18*β*-glycyrrhetinic acid represents a common derivative of glycyrrhizic acid. Within Chinese herbal formulations, licorice frequently functions as a harmonizing agent and formula enabler. Early studies have shown that 18*β*-glycyrrhizinic acid enhances phagocyte-mediated nonspecific immune responses, restores ROS-induced oxidative damage to the mitochondrial membranes in nasal mucosal epithelial cells, and maintains intramitochondrial homeostasis. These mechanisms collectively inhibit inflammatory lesions while augmenting nasal mucosal resistance to allergens.[128] Aligned with the "lung-gut axis" theory, sodium glycyrrhizinate intervention in AR-modeled rats reduces eosinophilic infiltration in colorectal mucosa and alleviates intestinal metaplasia symptoms including diarrhea.[129]

Saposhnikovia divaricata (Turcz). Schischk. polysaccharides are currently considered the most effective bioactive components in research on *Saposhnikovia divaricata* (Turcz). Schischk. treatment. These polysaccharides play a pivotal role in modulating both specific and non-specific immune responses while exerting dual immunomodulatory and anti-inflammatory effects. These bioactive polysaccharides are integral components in classical Chinese medicinal formulations, including Fangfeng Tongsheng San (Ledebouriella Sage-Inspired Powder) and Yupingfeng San (Jade Windscreen Powder), with standardized nomenclature. Studies using OVA-sensitized mouse models have demonstrated that these polysaccharides can reduce IgE levels, suppress the expression of inflammatory mediators, and inhibit the activation of NF-κB and the phosphorylation of STAT3. Furthermore, they regulate the expression of adhesion molecules and chemokines, prevent excessive immune activation, and restore the balance between Th1 and Th2 lymphocyte subsets, thereby alleviating nasal mucosal edema and excessive fluid secretion.[130] These findings suggest that *Saposhnikovia divaricata* polysaccharides hold promise as effective immunomodulatory agents, and their further development may offer significant therapeutic benefits in enhancing immune function.

Anthraquinone derivatives, isolated from various herbs such as rhubarb and thuja, exhibit anti-inflammatory, antioxidant, and antitumor activities. Recent studies have shown that miRNAs, which are small non-coding RNAs, play regulatory roles in the progression of various diseases. Krüppel-like factors (KLFs) constitute a crucial family of transcription factors, among which KLF5 is notably implicated in inflammatory processes and has been identified as a target gene of miR-375.[131] Research into this regulatory mechanism remains in its early stages. However, existing evidence suggests that emodin may serve as a promising candidate for the development of novel anti-allergic therapeutic agents.

Chinese Herbal Decoction

In the management of AR, clinicians utilize a variety of treatment approaches guided by their clinical experience. These therapeutic strategies primarily aim to warm the meridians, dispel exterior pathogenic factors, and enhance lung function by dispersing cold pathogens. Accordingly, Table 3 presents a summary of commonly used clinical formulas, modified prescriptions, and self-developed regimens currently applied in the treatment of AR.

Xiaoqinglong Decoction

Xiaoqinglong Decoction, a classical formula documented in the *Treatise on Cold Damage and Miscellaneous Diseases*, is extensively used in TCM for respiratory and immune disorders. Modern pharmacological investigations have identified quercetin, kaempferol, and lignoceryl alcohol as the primary bioactive constituents responsible for its anti-AR efficacy. These compounds ameliorate nasal symptoms by modulating the expression of key immune genes, including interleukin-6 (IL-6), tumor necrosis factor (TNF), and interleukin-1β (IL-1β). In vivo studies demonstrate that Xiaoqinglong Decoction suppresses TSLP signaling, significantly reducing Th2 cytokine release (IL-4, IL-5, IL-13). This cascade

Table 3 Summary of the Mechanism of Action of Chinese Herbal Decoctions in the Treatment of AR

Chinese Herbal Decoctions	Main Ingredients (Latin Name)	Research Object and model	Method of Administration	Dose	Medication Duration	Mechanism of Action	Reference
Xiaoqinglong Decoction	*Ephedra, Cynanchum otophyllum* Schneid., *Asarum heterotropoides* F.Schmidt, *Zingiber officinale* Roscoe, *Glycyrrhiza uralensis Fisch., Schisandra chinensis* (Turcz).Baill., *Pinellia ternata* (Thunb). Makino	Balb/c mice	Intraperitoneal injection	High20.02g·kg−[1]/d, low10.01g·kg−[1]/d	10d	Modulation of the activation of cytokine-associated IL-33/ST2 pathway and co-stimulation of cytokine-associated JAK/STAT pathway in the ILC-2 regulatory network inhibits ILC-2 proliferation and maturation and alleviates allergic inflammation	[132]
Linggui Zhugan Decoction	*Astragalus mongholicus* Bunge (Fisch)., *Atractylodes macrocephala* Koidz., *Citrus reticulata* Blanco, *Actaea cimicifuga* L., *Bupleurum chinense* DC., *Panax ginseng* C.A. Mey., *Glycyrrhiza uralensis* Fisch., *Angelica sinensis* (Oliv). Diels	SD rat	Gavage	25mg/kg/d	10d	Inhibits SP expression activity (AOD) release and SPR expression, inhibits mast cell infiltration, and attenuates inflammatory response	[133]
Linggui Zhugan Decoction	*Poria, Cinnamomum cassia, Atractylodes macrocephala* Koidz., *Glycyrrhiza uralensis* Fisch.	SD rat	Gavage	High4.32mg/kg/2times/d, medium3.24mg/kg/2times/d, low2.16mg/kg/2times/d	14d	Down-regulates the expression of MUC5A and MUC5B, promotes the repair of nasal mucosal tissues, and restores the normal physiological function of nasal mucosal tissues	[134]
Mahuang Fuzi Xixin Decoction	*Ephedra, Aconitum carmichaeli* Debeaux, *Asarum heterotropoides* F.Schmidt	Wistarrat	Gavage	1mL/2times/d	14d	Regressing the α-diversity of intestinal flora, regulating the structure of intestinal flora, decreasing the expression of RORγt and elevating the expression of Foxp3 in the nasal mucosa, and maintaining the Th17/Treg dynamic balance	[135]
Xiaochaihu Decoction	*Bupleurum chinense* DC., *Pinellia ternata* (Thunb). Makino, *Panax ginseng* C.A. Mey., *Scutellaria baicalensis* Georgi, *Zingiber officinale* Roscoe, *Ziziphus jujuba* Mill., *Glycyrrhiza uralensis* Fisch.	Patients with allergic rhinitis	Oral	300mL/d	3month	Regulates the level of T lymphoid subgroups (CD3+, CD4+, CD8+) in the body, reduces the content of inflammatory mediators VCAM-1, HIF-1, MAPK, and improves the body's immune function	[136]
Yiqi Zhixiu Decoction	*Astragalus mongholicus Bunge* (Fisch)., *Bupleurum chinense* DC., *Scutellaria baicalensis* Georgi, *Magnolia biondii Pamp., Asarum heterotropoides* F.Schmidt, *Cynomorium songaricum* Rupr., *Moutan Cortex, Ligusticum sinense* Oliv., *Cynanchum otophyllum* Schneid., *Prunus mume* (Sieh). Sieb.et Zucc., *Schisandra chinensis* (Turcz).Baill., *Glycyrrhiza uralensis* Fisch.	Patients with allergic rhinitis	Oral	300mL/d	1month	Reducing the expression levels of CCL17, YKL-40, and ECP, reducing the degree of inflammatory response, and promoting disease regression	[137]

(*Continued*)

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Table 3 (Continued).

Chinese Herbal Decoctions	Main Ingredients (Latin Name)	Research Object and model	Method of Administration	Dose	Medication Duration	Mechanism of Action	Reference
Guizhi Decoction	*Cinnamomum* cassia, *Cynanchum otophyllum* Schneid., *Glycyrrhiza uralensis* Fisch., *Scutellaria baicalensis* Georgi, *Ziziphus jujuba* Mill.	SD rat	Gavage	High8.4g/kg/d, low4.2g/kg/d	14d	Affects the cAMP-PKA-CREB signaling pathway, increases AQP5 protein and mRNA expression, and attenuates nasal mucosal pathological damage	[138]
Guizhi plus Huangqi Decoction	*Cinnamomum* cassia, *Cynanchum otophyllum* Schneid., *Glycyrrhiza uralensis* Fisch., *Scutellaria baicalensis* Georgi, *Ziziphus jujuba* Mill., *Astragalus mongholicus* Bunge (Fisch)., *Saposhnikovia divaricata* (Turcz). Schischk., *Asarum heterotropoides* F.Schmidt, *Schisandra chinensis* (Turcz).Baill.	Patients with allergic rhinitis	Oral	200mL/d	1month	Inhibition of NF-κB signaling pathway, blocking downstream PTX3 expression, attenuating eosinophil infiltration of nasal mucosa, improving nasal ventilation and ciliary function of nasal mucosa	[139]
Mahuang Decoction	*Ephedra, Cinnamomum* cassia, *Semen Armeniacae* Amarum, *Glycyrrhiza uralensis* Fisch.	SD rat	Intraperitoneal injection	High5.6g/kg−[1]/d, low2.8g/kg−[1]/d	14d	Modulation of cAMP-PKA-CREB signaling pathway, upregulation of the AQP5 protein and mRNA expression, and attenuate the inflammatory response of nasal mucosa	[140]
Allergy Decoction	*Saposhnikovia divaricata* (Turcz). Schischk., *Prunus mume* (Sieh). Sieb.et Zucc., *Schisandra chinensis* (Turcz).Baill., *Bupleurum chinense* DC., *Glycyrrhiza uralensis* Fisch.	BALB/c mice	Intraperitoneal injection	6.5g/kg/d	7d	Up-regulation of epithelial tight junction proteins Claudin-1, JAM-M and ZO-1 restores epithelial barrier function	[141]
Bimin prescription	*Astragalus mongholicus Bunge* (Fisch)., *Atractylodes macrocephala* Koidz., *Saposhnikovia divaricata* (Turcz). Schischk., *Codonopsis pilosula* (Franch). Nannf., *Dioscoreae Rhizoma*, *Zingiber officinale* Roscoe, *Bupleurum chinense* DC., *Schisandra chinensis* (Turcz).Baill., *Magnolia biondii Pamp., Xanthium strumarium* L., *Asarum heterotropoides* F. Schmidt, *Glycyrrhiza uralensis* Fisch.	Child with allergic rhinitis	Oral	200mL/d	2weekends	Accelerates saccharin elimination time, nasal mucus cilia removal speed, and restores nasal mucosa cilia function	[142]
Binmin Decoction	*Astragalus mongholicus Bunge* (Fisch)., *Saposhnikovia divaricata* (Turcz). Schischk., *Atractylodes macrocephala* Koidz., *Cynanchum otophyllum* Schneid., *Prunus mume* (Sieh). Sieb. et Zucc., *Ephedra*	Patients with allergic rhinitis	Oral	200mL/d	30d	Increase the volume and minimum cross-sectional area of the nasal cavity to reduce the resistance to nasal ventilation and improve the symptoms of nasal congestion	[143]

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terminates the allergic response and facilitates inflammatory resolution.[144] Additionally, it mitigates AR-induced nasal mucosal damage by inhibiting the IL-33/ST2 pathway. IL-33 binding to the ST2 receptor recruits IL-1RAP, which triggers intracellular cascades that activate immune-inflammatory cells and upregulate Th2 inflammatory factors.[132] Metabolomic analyses further reveal that Xiaoqinglong Decoction restores ω-oxidation capacity, inhibits the synthesis of specific lipid metabolites, preserves epidermal permeability barrier integrity, and modulates Th2 polarization through metabolite-level reprogramming.[145]

Buzhong Yiqi Decoction

Modern pharmacological research has identified quercetin and kaempferol as key bioactive components in Buzhong Yiqi Decoction. These flavonoids interact with critical target proteins including IL-6, VEGFA, and the transcription factor Jun. Through these interactions and the modulation of essential signaling pathways such as the IL-17, Toll-like receptor (TLR), and nuclear factor-κB (NF-κB) pathways, the formula demonstrates multi-organ regulatory effects on the spleen, stomach, and intestinal functions. These mechanisms collectively contribute to immune modulation, suppression of allergic responses, and stress adaptation, ultimately ameliorating AR symptoms. Current pathophysiological understanding attributes AR development to a Th1/Th2 immune imbalance. Characteristically, Th1 cells exhibit impaired secretion of IFN-γ and IL-2, compromising their capacity for intracellular pathogen clearance and immunoregulation. In contrast, allergen exposure induces a significant increase in the production of IL-4, IL-8, and other cytokines by Th2 cells, activating inflammatory reactions, promoting immune cell degeneration, and exacerbating nasal mucosal damage. Buzhong Yiqi Decoction effectively rebalances this dysregulation by normalizing inflammatory mediator release and restoring Th1/Th2 homeostasis, leading to symptomatic relief and improved quality of life.[146] STAT6, a pivotal component of the JAK-STAT pathway, mediates Th2 cell activation. Experimental evidence demonstrates that Buzhong Yiqi Decoction treatment significantly downregulates STAT6 protein expression and mRNA transcription in the mouse nasal mucosa, concurrently modulating STAT6-mediated chemotaxis, attenuating airway hyperresponsiveness, and facilitating mucosal repair. Building upon these findings and integrating principles of syndrome differentiation, researchers developed Jiawei Buzhong Yiqi Decoction. Preclinical studies confirm its enhanced efficacy in reducing nasal mucosal mast cell infiltration, suppressing substance P (SP) neuropeptide release, and activating neuroimmune regulatory circuits. These actions collectively alleviate the hallmark symptoms of AR, including nasal obstruction, paroxysmal sneezing, and rhinorrhea.[133] Furthermore, mechanistic investigations reveal that Jiawei Buzhong Yiqi Decoction disrupts late-phase allergic responses through dual inhibition of IL-5 expression and splenic eosinophil recruitment.[147]

Linggui Zhugan Decoction

Linggui Zhugan Decoction, originally formulated from the classic Chinese medical text *Treatise on Typhoid Fever*, has been demonstrated by modern pharmacological studies to exhibit anti-allergic effects. It suppresses IgE-mediated mast cell degranulation and the consequent release of inflammatory mediators. In the context of AR, Linggui Zhugan Decoction alleviates symptoms such as nasal congestion and slows disease progression by reducing levels of inflammatory markers (IgE, CRP, and IL-4) in the body, with minimal adverse effects. Both in vitro and in vivo studies have shown that its mechanism of action involves the inhibition of serum TNF-α and VCAM-1 expression by suppressing TSLP expression in nasal epithelial cells of rats with spleen deficiency and dampness syndrome-type AR, thus alleviating allergic symptoms.[148] Moreover, further studies have revealed that Linggui Zhugan Decoction downregulates the protein expression of aquaporins (AQPs) in the nasal mucosa by modulating the NF-κB signaling pathway, thereby inhibiting glandular secretion and relieving hypersecretory symptoms such as excessive clear nasal discharge, nasal itching, and frequent sneezing in AR rats.[149] As research advances, it has also been found that Linggui Zhugan Decoction regulates mucin expression, suppresses excessive mucus production, reduces extracellular matrix accumulation, improves nasal mucosal hyperresponsiveness, controls eosinophil infiltration, and restores the structural integrity of the nasal mucosa.[134]

Mahuang Fuzi Xixin Decoction

Modern pharmacological investigations demonstrate that this herbal formulation modulates AR pathogenesis through bioactive compounds such as quercetin, lignoceryl alcohol, and kaempferol. It intervenes via hemodynamic regulation and multi-pathway modulation, encompassing atherosclerosis signaling, AGE-RAGE interactions, and NF-κB cascades.

The formula transcriptionally activates T-bet and GATA3 factors while reducing pro-inflammatory cytokines (ie, IFN-γ, IL-4), thereby countering Th1/Th2 imbalance through JAK/STAT-1 pathway activation. Concomitantly, it attenuates nasal mucosal inflammation by suppressing IgE synthesis, inhibiting immune cell proliferation and histamine release, while repairing pathomorphological alterations in nasal and lung tissues. Moreover, it stabilizes the internal milieu, decreasing nasal mucosal hypersensitivity to allergens and noxious stimuli.[150] Notably, the therapeutic scope extends to the "intestinal-nasal-inflammatory-neural-immune" axis. The formula regulates neuropeptide release, diminishes multiple pro-inflammatory mediators, and restores the alpha diversity of intestinal flora in AR rats. It additionally repairs damaged intestinal epithelium and enhances mucosal immunity in both the nasal and gastrointestinal tracts. Subsequent studies have corroborated these AR-modulating benefits.[151] Guided by TCM theory, the formula was augmented with *Epimedium brevicornum, Lycium barbarum, Cuscuta chinensis*, and Os Draconis (Long Gu). In vitro analyses indicate that its mechanism involves downregulating Th2-associated inflammatory factors and inhibiting Th2 polarization via TSLP-OX40L pathway modulation, establishing a novel therapeutic paradigm forAR.[152]

Xiaochaihu Decoction

Xiaochaihu Decoction, a classical TCM formulation, demonstrates significant anti-inflammatory and antibacterial effects. It is clinically employed for managing allergic inflammatory conditions including AR. Studies have demonstrated that this decoction reduces nasal mucosal inflammation, regulates T lymphocyte subpopulations, promotes immune homeostasis, and alleviates clinical manifestations such as xeromycteria (nasal dryness), pharyngoxerosis (pharyngeal dryness), and olfactory hypersensitivity.[136] Notably, Xiaochaihu Decoction modulates eosinophil degranulation abnormalities and enhances pulmonary function, thereby supporting the "lung-nose axis" concept. This aligns with foundational TCM theory wherein the lungs govern nasal orifices, with nasal ventilation and olfactory functions dependent on lung regulation.[153] Complementarily, incorporating Xiaoqinglong Decoction into therapeutic regimens restores the Th17/Treg cell equilibrium. This synergistic approach significantly improves patients' quality of life indices through multidimensional symptom control.

Yiqi Zhixiu Decoction

Yiqi Zhixiu Decoction, developed through extensive clinical experience, has undergone modern pharmacological investigation. Research reveals that its chemical constituents—including astragalus polysaccharides, chromone derivatives, and total peony glucosides—modulate IgE and non-IgE antibody secretion. These components effectively suppress mast cell-mediated cellular immunity and allergic mediator-triggered humoral immunity during the acute phase of AR, thereby controlling disease progression. Empirical studies have demonstrated that Yiqi Zhixiu Decoction significantly alleviates AR-induced nasal symptoms, such as congestion and pruritus. Concurrently, it ameliorates systemic and localized inflammatory responses, enhances immunological competence, and regulates somatic constitution and internal homeostasis.[154] In vivo analyses further indicate that its anti-inflammatory mechanism involves dual modulation: enhancing Treg-mediated immune tolerance while inhibiting Th17-driven inflammatory cascades, thereby restoring Treg/Th17 immune equilibrium. Recent investigations have confirmed that Yiqi Zhixiu Decoction inhibits chemokine aggregation, such as C-C motif chemokine ligand 17 (CCL17) and chitinase-3-like protein 1 (YKL-40), in the nasal mucosa; blocks the secretion of ECP; downregulates adhesion molecule expression; attenuates inflammation; and reduces AR recurrence rates.[137]

Guizhi Decoction

Guizhi Decoction, a classical formula for treating Wind-Cold exterior deficiency syndrome in TCM, demonstrates multifaceted pharmacological properties. Modern research confirms its dual immunomodulatory effects encompassing anti-hypersensitivity, antibacterial, antiviral, and microcirculatory regulatory activities. This formula is clinically applied in the treatment of AR, influenza, arthritis, and diabetes mellitus. Early studies have indicated that Guizhi Decoction regulates cyclic adenosine monophosphate/guanosine monophosphate (cAMP/cGMP) levels to suppress hypersensitivity reactions, prevent the release of inflammatory mediators, and attenuate localized inflammation. In vitro studies demonstrate its capacity to restore nasal mucosal histopathology, alleviate nasal congestion, and upregulate aquaporin-5 (AQP5) protein and mRNA expression in nasal epithelial cells. Mechanistically, these effects are mediated through the cAMP/

protein kinase A-cAMP response element-binding protein (PKA-CREB) signaling cascade. This pathway modulation significantly reduces pruritic behaviors and paroxysmal sneezing in OVA-induced AR rat models, ultimately ameliorating the severity of AR.[138]

Guizhi Jia Huangqi Decoction

Guizhi Jia Huangqi Decoction, an enhanced formulation derived from Gui Zhi Decoction in *Essentials of the Golden Chamber*, incorporates *Astragalus mongholicus* into the classical cinnamon twig base. Guided by the TCM principle of "syndrome differentiation and treatment", this formula is clinically adapted for the management of AR symptoms. Modern pharmacological studies confirm that it contains bioactive polyphenols, flavonoids, and sesquiterpene lactones, which confer anti-inflammatory, antibacterial, antiviral, hypoglycemic, immunomodulatory, and anti-hypersensitivity properties. Immunological studies demonstrate that Guizhi Jia Huangqi Decoction attenuates chronic antigen exposure-induced stress responses that elevate immunoglobulin levels (IgA, IgG, and IgM), while neutralizing the pathological conversion of these immune factors. Furthermore, it regulates adaptive immunity by promoting dendritic cell maturation and stimulating T-lymphocyte proliferation and differentiation, collectively contributing to the treatment of AR. In vivo evidence indicates that this formula enhances immune competence, improves systemic health, and reduces AR recurrence.[155] Mechanistically, it modulates inflammatory mediators through NF-κB pathway activation while suppressing downstream pentraxin 3 (PTX3) expression. Concurrently, it inhibits eosinophil infiltration into the nasal mucosa, restoring nasal aeration and mucociliary clearance. This action alleviates pathological changes, including capillary dilation, glandular hypersecretion, and smooth muscle contraction.[139]

Mahuang Decoction

Mahuang Decoction is widely used as a foundational formula in the treatment of exogenous febrile diseases (such as common colds or influenza) in TCM. It is also recognized for its anti-inflammatory, analgesic, and anti-allergic properties, especially in managing allergic inflammation. Studies have shown that Mahuang Decoction can inhibit nasal mucosal capillary dilation in mice by suppressing the degranulation of pre-existing mast cells.[156] In vitro research further indicates that Mahuang Decoction reduces eosinophilic infiltration into nasal epithelial cells and glandular expansion, leading to decreased peripheral blood levels of IgE and IL-4, while promoting an increase in IFN-γ levels. By regulating the Th1/Th2 immune balance via the cAMP-PKA signaling pathway, Mahuang Decoction facilitates the binding of nuclear components to CBP, thereby upregulating AQP5 protein expression. This immunomodulatory effect contributes to the alleviation of OVA-induced allergic symptoms, such as sneezing, nose scratching, and rhinorrhea, in animal models of AR.[140]

Allergy Decoction

Allergy Decoction, characterized by its simplified yet efficacious formulation, is extensively employed in the management of allergic disorders. Modern pharmacological studies have identified bioactive compounds such as schisandrin, quercetin, kaempferol, isorhamnetin, and carboline alkaloids. These constituents counteract histamine-induced pruritus, inhibit mast cell degranulation, and enhance immunological competence. Experimental studies show that Allergy Decoction facilitates dendritic cell-mediated allergen uptake and processing. It transcriptionally upregulates T-bet expression while downregulating GATA-3 gene expression, concurrently reducing specific CD4+ T-lymphocyte subsets. This combined action prevents Th0-to-Th2 cell polarization, thereby increasing IFN-γ production and decreasing IL-4 levels. Consequently, it inhibits pro-inflammatory mediators, alleviating nasal mucosal congestion, edema, and glandular hyperplasia-associated rhinorrhea.[157] Histopathological analysis of nasal mucosa reveals that the decoction activates the expression of epithelial tight junction proteins—specifically claudin-1, JAM-A, and ZO-1. This mechanism restores epithelial integrity and repairs barrier dysfunction in damaged nasal epithelium. Additionally, it enhances the nasal mucosal resistance to allergens and ameliorates symptoms such as pruritic behaviors and rhinorrhea in OVA-sensitized AR mouse models.[141] Clinical reports further indicate that the pediatric application of Allergy Decoction is associated with reduced adverse effects and enhanced immune functionality.

Bimin Prescription

Bimin Prescription, an empirically derived formula based on clinical insights into AR, demonstrates multifaceted pharmacological actions. Modern research reveals that Qi-tonifying phytocompounds—particularly astragalosides and ginsenosides—enhance systemic immune defense while suppressing excessive immune responses and attenuating severe hypersensitivity reactions. This collectively improves the allergic microenvironment. Given the established roles of immune dysregulation and inflammation in AR pathogenesis, Bimin Prescription primarily targets the Th17/IL-17A signaling pathway. It modulates inflammatory CD4+ T lymphocytes, downregulates IL-23 and IL-6 expression, and reduces eosinophilic infiltration. These actions help restore autoimmune-damaged nasal mucosa and confer protection against allergen exposure.[158] Studies confirm the formula accelerates saccharin clearance time, enhances mucociliary transport efficiency, restores nasal epithelial ciliary function, promotes mucosal secretion absorption, and alleviates rhinorrhea.[142] Mechanistically, the Bimin Prescription inhibits NF-κB activation in cytoplasmic and nuclear compartments, downregulates AQP5 expression, regulates glandular fluid secretion, and suppresses downstream pro-inflammatory cytokines (IL-1β, IL-6, and TNF-α). These effects ameliorate Lung-Spleen Qi Deficiency (LSQD)-associated AR in animal models.[159] Notably, it inhibits nasal epithelial hypersecretion through the TMEM16A/NF-κB pathway modulation and MUC5AC mucin downregulation. This mechanism correlates with improved nasal symptoms and enhanced systemic well-being—including locomotor activity, defecation patterns, and behavioral responses—in OVA-sensitized AR rats.[160]

Binmin Decoction

Binmin Decoction is specifically formulated to address the clinical manifestations of AR. Modern pharmacological investigations demonstrate its capacity to modulate immune responses and exert potent anti-allergic effects. Research indicates that the formula regulates the differentiation and plasticity of Th1/Th2 lymphocyte subsets while inhibiting Th2-mediated immune cascades, thereby effectively managing and preventing AR. Further mechanistic studies reveal that Binmin Decoction suppresses γδ T-cell differentiation and attenuates their pro-inflammatory activity. Additionally, it modulates nasal mucosal T-lymphocyte subpopulation ratios and downregulates the expression of key transcriptional receptors, including Foxp3 and RORγt, in peripheral circulation. These actions collectively alleviate mouse AR symptoms, such as nasal obstruction, paroxysmal sneezing, and nasal pruritus.[161] Employing acoustic rhinometry for objective nasal cavity assessment, researchers documented that Binmin Decoction increases nasal cavity volume and the minimal cross-sectional area. This reduces nasal airflow resistance, accelerates gas exchange, and improves symptoms of congestion and olfactory dysfunction.[143]

Proprietary Chinese Medicine Products

Advancements in modern pharmaceutical technology have enabled the development of diverse drug formulations, enhancing patient convenience while improving the clinical utility and acceptance of TCM. Table 4 comprehensively summarizes clinically validated proprietary Chinese medicines commonly used for the management of AR.

Yupingfeng San (Jade Screen Powder)

Yupingfeng San (Jade Screen Powder) is a classic TCM formulation designed for integrated treatment and prevention. Modern pharmacological studies confirm that bioactive compounds such as astragalus polysaccharides and volatile oil polysaccharides from *Saposhnikovia divaricata* (Turcz). Schischk. extract, enhance cellular and humoral immunity while mitigating free radical-induced tissue damage. Early research established AR as an airway disease that affects comorbid respiratory conditions. Yupingfeng San reduces eosinophil granulocyte concentrations in nasal septal and bronchopulmonary mucosa, redirecting these cells from inflammatory sites. This decreases chemokine expression, improves allergic reactions, and reduces airway hyperresponsiveness. Additionally, cGMP and cAMP levels correlate significantly with AR progression. By reducing these levels, Yupingfeng San enhances allergen recognition and defense capabilities, promoting balanced immune responses. These responses help regulate inflammation, cellular proliferation, and differentiation. Consequently, it alleviates symptoms such as sneezing, rhinorrhea, nasal congestion, and pruritus.[170] Furthermore, beyond nasal symptom improvement, Yupingfeng San modulates mast cell activity by inhibiting IL-13-mediated

Table 4 Summary of the Mechanism of Action of Proprietary Chinese Medicine Products for the Treatment of AR

Drug Names	Main ingredients (Latin Name)	Research Object and Model	Method of Administration	Dose	Medication Duration	Mechanism of Action	Reference
Yupingfeng Powder	*Astragalus mongholicus* Bunge (Fisch)., *Atractylodes macrocephala* Koidz., *Saposhnikovia divaricata* (Turcz). Schischk.	Mice	Gavage	High24mg·kg⁻[1]/d, medium12mg·kg⁻[1]/d, low6mg·kg⁻[1]/d	2 weekends	Affects the ROS/NLRP3/Caspase-1 signaling pathway, induces cellular pyroptosis, and inhibits the development of allergic rhinitis	[162]
Tongqiao rhinitis capsules	*Astragalus mongholicus* Bunge (Fisch)., *Atractylodes macrocephala* Koidz., *Xanthium strumarium* L., *Angelica sinensis* (Oliv). Diels, *Saposhnikovia divaricata* (Turcz). Schischk., *Magnolia biondii Pamp., Mentha canadensis* L.	Patients with allergic rhinitis	Oral	2g/3times/d	6 weekends	Regulates Th1 /Th2 balance in patients with allergic rhinitis, reduces oxidative stress and inflammation and improves symptoms	[163]
Sanfeng Tongqiao Dropping Pill	*Scutellaria baicalensis* Georgi, *Nepeta cataria* L., *Hansenia weberbaueriana, Asarum heterotropoides* F.Schmidt	Patients with allergic rhinitis	Oral	15.2g/3times/d	7d	Inhibit the phosphorylation of STAT3, prevent the proliferation of cuprocytes, play an immunomodulatory role, and reduce the secretion of nasal mucus	[164]
Biyuan Tongqiao Granule	*Magnolia biondii Pamp., Xanthium strumarium* L., *Ephedrae, Angelica sinensis (Oliv). Diels, Mentha canadensis* L., *Ligusticum sinense* Oliv., *Scutellaria baicalensis* Georgi	Patients with allergic rhinitis	Oral	15g/3times/d	14d	Improves trace elements in the body, regulates immune function, and improves nasal ventilation	[165]
Cangerzi Powder	*Xanthium strumarium* L., *Magnolia biondii* Pamp., *Angelica sinensis* (Oliv). Diels, Mentha canadensis L.	KM mice	Gavage	High12g/kg/d, medium6g/kg/d, low3g/kg/d	14d	Reduced serum inflammatory factor levels, regulated cytokine secretion by Th cells, and increased AQP5 protein expression	[166]
Wenfei Zhiliu Dan	*Panax ginseng* C.A. Mey., *Nepeta cataria* L., *Asarum heterotropoides* F.*Schmidt, Terminalia chebula* Retz., *Platycodon grandiflorus, Heartleaf Houttuynia*	Patients with allergic rhinitis	Oral	400mL/d	14d	Reduced IgE and IgG levels, attenuated pro-inflammatory factors, eosinophilic acid cell cation (ECP) levels, increased CD4+ /CD8 + ratio, reducing symptoms	[167]
Xinqin Granule	*Asarum heterotropoides* F.Schmidt, *Scutellaria baicalensis Georgi, Nepeta cataria* L., *Saposhnikovia divaricata* (Turcz). Schischk., *Angelica sinensis* (Oliv). Diels, *Xanthium strumarium* L., *Astragalus mongholicus* Bunge(Fisch)., *Atractylodes macrocephala* Koidz., *Acorus gramineu*	Mice	Gavage	5.0g/kg/d	7d	Decrease in the number of SP-positive cells and decrease in SP-R mRNA expression in the nasal mucosa	[168]
Xiangju Capsule	*Platycarya strobilacea* Sieb, Saposhnikovia divaricata (Turcz). Schischk., *Chrysanthemum, Magnolia biondii Pamp., Astragalus mongholicus Bunge* (Fisch)., *Prunella vulgaris* L.	Patients with allergic rhinitis	Oral	1.2g/times/d	14d	Reduces LTB4, IgG, IgE levels, improves allergic status; increases CD3+, CD4+, CD4+/CD8+ levels, reduces inflammatory factors IL-4, IL-8, IL 17 levels, improves immune function, reduces inflammatory response	[169]

activation and suppressing GM-CSF expression. This stabilizes mast cells and reduces histamine release. Significantly, it modulates the TLR2-HO-1/CO pathway, upregulates claudin-1 and ZO-1 tight junction gene expression, and repairs nasal mucosal epithelial barrier integrity. Research on pyroptosis demonstrates that Yupingfeng San inhibits NLRP3 inflammasome assembly, blocks caspase-1/GSDMD protease activation in epithelial cells, and reduces pro-inflammatory cytokine secretion (IL-4, IL-1β, IL-18), thereby preventing aberrant mucosal epithelial pyroptosis both in vivo and in vitro. Simultaneously, it elevates IFN-γ levels, suppressing mitochondrial oxidative stress—a key trigger for nasal pruritus and olfactory dysfunction.[162] Additionally, Yupingfeng San modulates the NF-κB and p38 MAPK signaling pathways, therapeutically intervening in AR pathogenesis and attenuating histamine-driven inflammatory responses.[171]

Tongqiao Biyan Capsule

Tongqiao Biyan Capsule is a clinically established treatment for AR. Relevant studies demonstrate that this formulation improves nasal tissue architecture, reduces turbinate volume, decreases nasal airflow resistance, and alleviates congestion symptoms. By lowering ECP levels, it attenuates toxin-induced nasal mucosal damage. Additionally, it blocks the binding of the ST2 receptor to sIgE, consequently reducing the release of inflammatory factors in AR, diminishing nasal hyperresponsiveness, and effectively controlling disease progression.[172] Pathogenetic analyses reveal that Tongqiao Biyan Capsule activates T lymphocyte proliferation and modulates lymphocyte function. It regulates Th1/Th2/Th17/Treg cell subset balance, improving the imbalance between Th1/Th2 and Th17/Treg. This immunomodulation maintains immune homeostasis and improves clinical manifestations such as nasal obstruction and sneezing.[173] To enhance patient compliance, the original capsule formulation has been innovatively adapted into a granule form. In vivo studies confirm that Tongqiao Biyan Granule regulates inflammatory cytokine release (TNF-α, IL-4, IL-6, IL-12), suppresses inflammatory responses, and reduces nasal mucosal edema. It concurrently decreases nasal resistance (NR), mucociliary transport time (MTT), and mucociliary clearance rate (MCR) indices, restores ciliary function, increases turbinate volume, and ameliorates rhinorrhea and congestion.[174] Research verifies that while AR exacerbates pulmonary ventilation dysfunction, Tongqiao Biyan Granule reduces nasal eosinophil infiltration, lowers sIgE and eotaxin-2 levels, and improves clinical symptoms with minimal side effects.[163] Notably, it reduces the levels of SOD and MDA, which are elevated during oxidative stress. This action accelerates cellular metabolism and mitigates oxidative damage.

Sanfeng Tongqiao Dropping Pills

Sanfeng Tongqiao Dropping Pills are a compound Chinese medicinal formulation clinically employed for AR management. Contemporary pharmacological investigations demonstrate that this preparation utilizes bioactive constituents, including flavonoids, sterols, and lignans, to regulate gene expression such as JUN, MAPK14, and IL6. These constituents may target signaling pathways such as AGE-RAGE, TNF, and PI3K-Akt to modulate AR pathogenesis. In vitro studies using OVA-sensitized mouse models revealed inhibition of Th2 cell differentiation through disrupted Gata-3 binding to target DNA sequences. Additionally, it upregulates T-bet, a transcription factor expression in Th1 cells, exerts bidirectional immunomodulation, and activates Foxp3, a key transcriptional regulator in Treg cells. These mechanisms collectively suppress allergen-induced Th2 responses, alter nasal mucosal inflammation in vivo, and alleviate the characteristic symptoms of AR in mice, including sneezing and nasal pruritus. Complementary in vivo analyses indicate suppression of STAT3 phosphorylation, inhibition of goblet cell hyperplasia, reduced nasal mucus secretion, and immunoregulatory activity.[164]

Biyuan Tongqiao Granules

Biyuan Tongqiao Granules represent a specialized anti-AR formulation developed through modern pharmaceutical technology. Contemporary pharmacological studies have demonstrated that this preparation contains bioactive compounds such as quercetin, apigenin, kaempferol, ursolic acid, and rosmarinic acid. These compounds target PTGS2, CHRM2, and NOS2 proteins. These agents mediate the MAPK, NF-κB, and HIF1 signaling pathways, collectively contributing to antioxidative and anti-inflammatory effects in AR management. Studies have shown that Biyuan Tongqiao Granules inhibit production of inflammatory mediators (AQP5, IL-4, TNF-α), thereby reducing mast cell activation and limiting the release of sensitizing factors. This suppression decreases AQP5 expression in the nasal mucosa, alleviating glandular epithelial congestion and edema. Concurrently, it inhibits excessive mucus secretion,

downregulates intercellular adhesion molecule-1 (ICAM-1) expression, and prevents eosinophil infiltration into nasal tissues, significantly improving AR pathology.[175] Moreover, AR frequently coexists with adenoid hypertrophy in pathogenesis. Biyuan Tongqiao Granules modulate serum inflammatory mediators (IL-4, IL-8, and IFN-γ), altering the inflammatory milieu and reducing adenoid volume. This dual action improves sleep quality, enhances immune competence, and accelerates clinical recovery.[176] Furthermore, this formulation restores nasal ventilation function by increasing peak expiratory flow (PEF) while decreasing diurnal PEF variation rates. It also counteracts immune dysregulation by normalizing micronutrient profiles. In vitro studies have found that Biyuan Tongqiao Granules upregulate surfactant protein A (SP-A) expression in the nasal mucosa of rats, strengthening allergen resistance. Additionally, they elevate Th1 and T-bet+ Th1 cell proportions, rebalancing Th1/Th2/Th17 subsets to mitigate inflammation.[165]

Cangerzi Powder

Cangerzi Powder is a classic formulation for the management of nasal disorders. Modern pharmacological research has identified its active constituents including naringin, sitosterol, and luteolin. These compounds primarily target PTGS2, PIK3CG, and AKT1 proteins and modulate calcium signaling, HIF-1, TLR, and AMPK pathways during the treatment of AR. Notably, this formulation restores Th1/Th2 immune balance and mitigates histamine-mediated pathological damage to nasal mucosa by regulating the secretion of Th1/Th2 cytokines (IL-5, IL-10, IFN-γ). Concurrently, it regulates AQP5 distribution in nasal epithelium, reduces glandular hypersecretion, and alleviates rhinorrhea in AR mouse models.[166] Additionally, Cangerzi Powder significantly alters NR, total nasal volume (TNV), and minimal cross-sectional area (MCA), thereby enhancing nasal airflow dynamics and ameliorating congestion symptoms. Mechanistic studies reveal that it elevates IFN-γ levels in the nasal mucosa, which inhibits JNK phosphorylation and consequently blocks c-Jun dimer formation. This accelerates remodeling of the damaged nasal mucosa. Simultaneously, the formulation reduces the levels of TNF-α, TGF-β1, and IL-4, suppresses IgE production, and attenuates the severity of allergic inflammatory responses.[177]

Wenfei Zhiliu Dan

Wenfei Zhiliu Dan constitutes a specialized AR treatment grounded in TCM theory. Contemporary pharmacological investigations confirm its anti-inflammatory, antipruritic, and immunomodulatory properties. Research demonstrates that this formulation regulates cytokine release, such as IL-5, IL-10, IL-17, IL-35, and TNF-α, inhibits IgE production and Th2 cell differentiation, promotes eosinophil apoptosis, and prevents ECP-induced mast cell degranulation. This immunoregulatory activity elevates the CD4+/CD8+ T-cell ratio, activates systemic immunoregulatory functions, and effectively alleviates AR symptoms such as nasal congestion, rhinorrhea, and paroxysmal sneezing.[167] Beyond nasal symptom management, Wenfei Zhiliu Dan also mitigates ocular symptoms such as allergic pruritus. In vivo cytological analyses reveal reduced serum IL-33 expression in AR patients, suppressed Th2 cell and eosinophil infiltration in nasal mucosa, and decreased chemokine/cytokine production. Critically, this modulation attenuates allergy-induced nasal congestion by modulating nasal vascular permeability.[178]

Xin Qin Granules

Xin Qin Granules is a specialized nasal medication targeting symptoms including nasal pruritus, paroxysmal sneezing, rhinorrhea, and rhinovirus-induced colds. Contemporary pharmacological studies confirm its anti-allergic properties, with both Phase I and II biotransformation pathways potently inhibiting mast cell activation and the release of allergic mediators. These inhibitory effects manifest more prominently within target tissues due to localized concentration gradients. In vivo investigations demonstrate that Xin Qin Granules reduce serum IL-4 concentrations, modulate YKL-40 levels, promote Th0 cell proliferation and differentiation toward Th1 phenotypes, and correct systemic immune deviation. They also suppress histamine and LTC-4 production in the nasal cavity, stabilize plasma membranes, and reduce tissue edema—collectively enabling effective AR management.[179] Notably, this formulation bidirectionally regulates inflammatory factors (IL-4, IL-6, and IL-10), synergistically improving nasal function while enhancing allergen recognition and immune resistance capabilities. Clinical stability studies confirm sustained efficacy with low relapse rates and no withdrawal reactions observed 30 days after treatment discontinuation. In vitro analyses reveal that therapeutic effects involve regulation of the SP → SP-R ← → SP-R mRNA positive feedback cascade.[168]

Xiang Ju Capsule

Xiang Ju Capsule is a commonly prescribed medication in Chinese otorhinolaryngology clinics for the management of AR. Its active components, derived from *Chimonanthus praecox* inflorescence and *Chrysanthemum indicum*, exert potent anti-inflammatory, antibacterial, and antiviral effects. Pharmacologically, this formulation reduces IgE and IgG levels, which mediate immediate and delayed-type allergic responses, respectively. Additionally, it inhibits LTB4 overexpression, modulates nasal mucosal vascular permeability, and alleviates localized mucosal edema.[180] It is noteworthy that Xiang Ju Capsule can regulate T-lymphocyte immunoreactive cells (CD3+, CD4+, CD8+) toward immune homeostasis while concurrently modulating the levels of IL-4, IL-8, and IL-17. This triple-action mechanism—reducing inflammatory responses, protecting the nasal mucosa through enhanced microcirculation, and boosting mucosal immunity—collectively enhances therapeutic efficacy.[169]

Chinese Herbal Fumigation

TCM integrates internal and external therapeutic modalities. Herbal fumigation therapy, perpetuated throughout China's historical development for millennia, synthesizes pharmacological actions with moist-heat physiotherapy to prevent diseases, treat pathologies, and fortify physiological resilience. The nasal cavity serves as a critical interface for material exchange between the body and the external environment. Its mucosal surface area (about 150cm^2) must be maintained at body temperature with saturated humidity state, in order to ensure normal ciliary motility, physiological mucus secretion, suppression of pathogen proliferation, reduced inflammatory responses. Nasopharyngeal mucosal lesions primarily arise from viral infections and allergic sensitization. Research has shown that rhinovirus is most effectively inactivated at temperatures between 42°C and 43°C. The smoke generated from the complete combustion of TCM or the steam produced after decoction can be controlled at an appropriate temperature and inhaled through the nasal cavity. The medicine is locally absorbed and enters the bloodstream through the "nasal-pulmonary" circulation, thereby bypassing the intestinal and hepatic circulatory pathways. In addition, drug molecules stimulate local neurons, triggering processes such as phagocytosis, pinocytosis, intracellular transport, and lateral transport within neurons. Through neural reflexes and via the olfactory nerve pathway as well as the olfactory mucosal epithelial pathway, drugs can effectively bypass the blood-brain barrier, regulate the higher central nervous system, achieve therapeutic effects, and promote blood circulation. These mechanisms help alleviate symptoms such as pruritus, rhinorrhea, paroxysmal sneezing, and nasal congestion, while also reducing nasal mucosal edema and hyperemia. The steam and active molecular components in Chinese herbal fumigation can regulate glandular secretion, restoring it to a normal physiological state. TCM fumigation avoids gastrointestinal irritation and maximizes the anti-inflammatory effects of the herbs. It also allows for individualized adjustments based on the patient's specific needs, reducing discomfort and intolerance associated with the taste of oral decoctions. Herbal fumigation demonstrates a favorable synergistic effect, which can be combined with other medications to enhance therapeutic outcomes and improve immunity. Existing studies have shown that fumigation significantly improves nasal clinical symptoms and alleviates ocular symptoms of AR, such as itchy eyes, foreign body sensation, conjunctival erythema, and epiphora.

Chinese Herbal Application

Chinese herbal compresses represent an ancient and distinctive external therapeutic modality in TCM. Based on TCM diagnostic and treatment theories, this therapy involves selecting appropriate medicinal substances, which are formulated into medicinal cakes or creams and then directly applied to the skin for the treatment of specific diseases or skin conditions. Through acupoint application, the medicinal ingredients directly stimulate acupoints and are transmitted to corresponding internal organs via the meridian system. This process can improve local microcirculation, enhance the phagocytic ability of leukocytes, and help restore immune homeostasis. At the same time, it can avoid the side effects of orally administered drugs and the liver's inactivation of the drugs. The application of TCM herbal preparations can stimulate acupoints, activate the meridians, warm the channels to dispel cold, regulate the balance of yin and yang, enhance immunity, and treat diseases, thereby embodying the TCM concept of "preventing disease before it occurs". Sanfutie (a traditional Chinese herbal patch used during the hottest summer days for treating winter illnesses) can dredge

the meridians, promote blood circulation, regulate the functions of the lungs and spleen, restore normal physiological activities, enhance immune function, significantly alleviate patient symptoms, reduce the recurrence rate of rhinitis, and improve overall quality of life. Herbal compresses act directly on the nasal area, stimulating the body's defense mechanisms and thereby enhancing immunity. In vivo studies have shown that the herbal patch modulates immune protein levels in the body, increases serum IgA levels, decreases serum IgE levels, and inhibits type I hypersensitivity reactions.

Chinese Herbal Sachet

Chinese herbal sachets, also known as aromatherapy pouches, are made by crushing aromatic Chinese medicinal herbs into fine powder, mixing them evenly, and then using the mixture to create sachets or infuse clothing items (such as undershirts, knee pads, and pillows). They are worn on the body or placed on the chest, pillow, or in front of the nose to strengthen the body and resist disease. This traditional method is a distinctive therapeutic approach in TCM. According to TCM theory, the nose and lungs are interconnected. The medicinal substances used in these scented pouches contain volatile compounds that enter the body through the nasal mucosa, respiratory tract, or skin. They then circulate through the bloodstream and meridian system, spreading throughout the body to achieve disease prevention and control. Modern research also supports this practice, indicating that the volatile components in the sachets enter the body through the mouth, nose, and skin, enhancing respiratory immunity and exerting direct or indirect inhibitory effects on bacteria and viruses, thereby contributing to disease prevention and treatment.

Signaling Pathways

Regulation of the NF-κB Signaling Pathway

The formation of AR is closely associated with the inflammatory factors secreted by Th1 and Th2 cells, which are in turn regulated by the NF-κB signaling pathway. Under normal physiological conditions, NF-κB is bound to IκB, forming a stable dimeric complex. When the NF-κB signaling pathway is activated by external stimuli, the IκB kinase (IKK) phosphorylates IκB, leading to its ubiquitin-mediated degradation. As a result, free NF-κB (primarily composed of the p65 subunit) translocates into the nucleus, where it promotes the transcription and secretion of downstream inflammatory cytokines such as IL-1β, IL-6, IL-8, and TNF-α, which are known to contribute to the inflammatory response observed in AR. Furthermore, activation of the NF-κB pathway also stimulates the expression of inducible nitric oxide synthase (iNOS) and cyclooxygenase-2 (COX-2). Inhibition of COX-2 expression has been shown to effectively modulate the immune response. Lycium barbarum, a widely used medicinal herb in TCM, is well known for its health benefits and therapeutic effects. Lycium barbarum polysaccharides have been found to suppress the expression of TLR4/NF-κB signaling pathway-related proteins, restore the balance of Th1/Th2 cytokines, and reduce IgE production, thereby significantly alleviating nasal mucosal inflammation in AR mouse models.

Regulation of the p38 MAPK Signaling Pathway

Signal transduction mediated by key MAPK family signaling molecules can influence various cellular functions, including the mechanisms underlying cell death, apoptosis, and inflammation. The MAPK signaling cascade plays a crucial role in the activation of multiple immune cell types. Specifically, the p38 MAPK signaling pathway is activated at the interaction sites of the TCR in T cells, the B-cell receptor (BCR) in B cells, and the FcεRI in mast cells. Upon activation, these pathways can trigger cellular responses such as proliferation, differentiation, cytokine production, and degranulation. The p38 MAPK pathway is also essential for the activation of chemokine receptors and IL-5 cytokine receptors in eosinophils, contributing to eosinophil survival, activation, degranulation, and chemotaxis. These findings indicate that the p38 MAPK signaling pathway is deeply involved in inflammatory responses and immunomodulatory processes, promoting the release of inflammatory mediators. Notably, the traditional herbal formula Yupingfeng San has been shown to inhibit the expression of both p38 MAPK and phosphorylated p38 MAPK proteins, interfere with the transcription of inflammatory cytokine genes, reduce histamine release, and alleviate histamine-induced pathological changes in the nasal mucosa.

Regulation of the PI3K/AKT Signaling Pathway

Phosphatidylinositol-3-kinase (PI3K) is a downstream signaling effector of G-protein-coupled receptors, and serine/threonine kinase AKT is a critical downstream signaling molecule of PI3K. Together, they constitute an important intracellular signaling pathway. PI3K is involved in allergic airway inflammatory responses by promoting the activation of inflammatory mediators, facilitating eosinophil recruitment, and mediating Th1/Th2 immune imbalances, particularly favoring Th2-type inflammatory responses. In addition, intracellular PI3K plays a crucial role in eosinophil chemotaxis and also mediates the activation and secretion of mast cells, influencing their degranulation and subsequent histamine release. The therapeutic effect of Cangerzi Powder, a formula commonly used for treating AR, in alleviating AR progression may be associated with the phosphorylation of the Syk/PI3K/AKT pathway phosphorylation, which in turn regulates the functions of Th1/Th2 cells, B cells, mast cells, eosinophils, and goblet cells, thereby exerting anti-inflammatory and anti-allergic effects.

Conclusions and Future Research

With the improvement of people's quality of life and increased global awareness of disease education, public attention to AR has significantly increased. Moreover, continuous changes in climate, environmental conditions, and lifestyle factors have contributed to a rise in disease-causing agents, greater complexity in disease progression, a decline in the body's immune defenses, earlier onset ages for AR, and shorter intervals between disease episodes. Given China's large population base, vast geographical expanse, and regional disparities in economic development and healthcare resources, the number of AR patients is substantial. In clinical practice, although long-term use of Western medicine can alleviate certain symptoms, its long-term efficacy remains limited. Additionally, prolonged medication use often leads to complications, imposing significant physical, economic, and psychological burdens on patients. This situation exacerbates doctor-patient tensions in China. Therefore, the development of a long-lasting treatment modality with minimal side effects that also addresses disease complications is of critical importance for the effective management of AR in China.

TCM, with millennia of historical evolution and abundant medicinal resources, presents a comprehensive therapeutic approach that spans disease prevention, treatment, and peri-/post-operative management. Rooted in evidence-based syndrome differentiation and holistic therapeutic principles, TCM's potential in AR management extends beyond monotherapy to encompass individualized interventions. These include tailored internal decoctions, standardized herbal preparations, medicinal fumigation, topical applications, and specialized TCM modalities. Evidence-based TCM practice emphasizes systematic collation and targeted analysis, enabling precise disease classification and patient-specific diagnosis. This approach facilitates dynamic formula modification based on concurrent symptoms through the addition or subtraction of herbs. Crucially, TCM's "preventive treatment of disease" philosophy—documented since antiquity in classical texts such as the *Yellow Emperor's Canon*—epitomizes the strategic significance of prophylactic medicine in disease control.

The complexity of the components found in Chinese medicine monomers and compounds, along with their synergistic and multi-target effects, can help overcome the inevitable limitations associated with the use of chemical drugs. Numerous clinical trials have shown that, in the treatment of post-AR, the efficacy of either Chinese herbal compound formulations or adjunctive therapies such as fumigation or combined medication is superior to that of chemical drugs used alone. Some data indicate that the therapeutic efficiency of the Chinese medicine group is significantly higher than that of the western medicine group, with notable improvements in accompanying symptoms such as nasal congestion, sneezing, and runny nose. With the advancement of modern pharmaceutical technology, research on herbal medicines has become more refined. For example, the purification of Fritillaria has led to the development of a Fritillaria extract used in nasal irrigation therapy, which helps alleviate symptoms in patients with AR. This paper confirms that the therapeutic effects of TCM are primarily attributed to its ability to regulate immune cells, thereby reducing the release of inflammatory factors and inhibiting oxidative stress generation. This mechanism contributes to delaying disease onset and alleviating symptoms.

Regrettably, despite TCM having demonstrated efficacy in AR management through holistic advantages and evidence-based approaches—alongside promising application prospects—several limitations persist: (1) Current studies exhibit inconsistent methodological rigor, lacking large-scale randomized controlled trials. Original literature may contain inherent bias and methodological constraints. (2) Most TCM interventions for AR lack quantifiable biomarker changes and standardized efficacy evaluation protocols. (3) AR management requires further standardization regarding dosage forms, therapeutic doses, and treatment duration. (4) Fumigation and similar adjuvant therapies primarily provide symptomatic relief. Acupoint fumigation protocols remain predominantly experience-based, lacking evidence-driven positioning. (5) Advancing TCM integration with modern medicine necessitates strengthened policy support and international cooperative frameworks.

In conclusion, integrating TCM with modern medicine—such as combining antihistamines (the recommended use time is less than half a month), which provide acute-phase control in modern medicine, with long-acting Chinese herbal formulations currently used in clinical practice (the recommended use time is more than half a month)—can enhance both therapeutic efficacy and safety. By extensively and deeply utilizing the core principles of TCM, novel therapeutic strategies and targets can be developed, offering more practical and effective treatment options for AR. The holistic and individualized approach of TCM provides promising potential for improving patient prognosis. As our understanding of TCM and its underlying mechanisms continues to expand, so too does its global potential in combating AR.

Data Sharing Statement

All data are available in the paper and they are shown in the figures and tables.

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors declare no conflict of interest.

References

1. Czech EJ, Overholser A, Schultz P. Allergic Rhinitis. *Prim Care*. 2023;50(2):159–178. doi:10.1016/j.pop.2023.01.003
2. Mims JW. Epidemiology of allergic rhinitis. *Int Forum Allergy Rhinol*. 2014;4(Suppl 2):S18–20. doi:10.1002/alr.21385
3. Cingi C, Gevaert P, Mösges R, et al. Multi-morbidities of allergic rhinitis in adults: European Academy of Allergy and Clinical Immunology Task Force report. *Clin Transl Allergy*. 2017;7:17. doi:10.1186/s13601-017-0153-z
4. Wise SK, Lin SY, Toskala E, et al. International consensus statement on allergy and rhinology: allergic rhinitis. *Int Forum Allergy Rhinol*. 2018;8(2):108–352. doi:10.1002/alr.22073
5. Ramsey AC, Deane PM. Early-life risk factors and allergic rhinitis: comparing European and US data. *J Allergy Clin Immunol*. 2011;128(4):824–825. doi:10.1016/j.jaci.2011.07.037
6. Hoyte FCL, Nelson HS. Recent advances in allergic rhinitis. *F1000Res*. 2018;7:1333. doi:10.12688/f1000research.15367.1
7. Czech EJ, Overholser A, Schultz P. Allergic rhinitis. *Med Clin North Am*. 2024;108(4):609–628. doi:10.1016/j.mcna.2023.08.013
8. Tosca MA, Trincianti C, Naso M, Nosratian V, Ciprandi G. Treatment of allergic rhinitis in clinical practice. *Curr Pediatr Rev*. 2024;20(3):271–277. doi:10.2174/1573396320666230912103108
9. Creticos PS, Gunaydin FE, Nolte H, Damask C, Durham SR. Allergen immunotherapy: the evidence supporting the efficacy and safety of subcutaneous immunotherapy and sublingual forms of immunotherapy for allergic rhinitis/conjunctivitis and asthma. *J Allergy Clin Immunol Pract*. 2024;12(6):1415–1427. doi:10.1016/j.jaip.2024.04.034
10. Olivier CE, Argentão DG, RP DSL, da Silva MD, Dos Santos RA, Fabbri N. Assessment of allergen-induced respiratory hyperresponsiveness before the prescription of a specific immunotherapy. *Allergy Rhinol*. 2015;6(2):89–93. doi:10.2500/ar.2015.6.0122

11. Živković Z, Djurić-Filipović I, Živanović S. Current issues on sublingual allergen-specific immunotherapy in children with asthma and allergic rhinitis. *Srp Arh Celok Lek*. 2016;144(5–6):345–350. doi:10.2298/SARH1606345Z
12. Endaryanto A, Nugraha RA. Safety profile and issues of subcutaneous immunotherapy in the treatment of children with allergic rhinitis. *Cells*. 2022;11(9):1584. doi:10.3390/cells11091584
13. Chan HHL, Ng T. Traditional Chinese Medicine (TCM) and allergic diseases. *Curr Allergy Asthma Rep*. 2020;20(11):67. doi:10.1007/s11882-020-00959-9
14. Li H, Kreiner JM, Wong AR, et al. Oral application of Chinese herbal medicine for allergic rhinitis: a systematic review and meta-analysis of randomized controlled trials. *Phytother Res*. 2021;35(6):3113–3129. doi:10.1002/ptr.7037
15. Zhang Y, Qi L, Wang R. Meta-analysis: reducing the recurrence rate of allergic rhinitis through oral administration of traditional Chinese medicine. *Eur Rev Med Pharmacol Sci*. 2023;27(17):7924–7934. doi:10.26355/eurrev_202309_33551
16. Soffer GK, Myers C, Weinstock M, Hand M. Alternative, complementary, and integrative medicine approaches to allergic rhinitis. *Ann Allergy Asthma Immunol*. 2025;135(1):5–6. doi:10.1016/j.anai.2025.01.021
17. Zhang Y, Lan F, Zhang L. Update on pathomechanisms and treatments in allergic rhinitis. *Allergy*. 2022;77(11):3309–3319. doi:10.1111/all.15454
18. Eifan AO, Durham SR. Pathogenesis of rhinitis. *Clin Exp Allergy*. 2016;46(9):1139–1151. doi:10.1111/cea.12780
19. Han X, Krempski JW, Nadeau K. Advances and novel developments in mechanisms of allergic inflammation. *Allergy*. 2020;75(12):3100–3111. doi:10.1111/all.14632
20. Kaminuma O, Nishimura T, Saeki M, Mori A, Hiroi T. T cell-mediated nasal hyperresponsiveness in allergic rhinitis. *Biol Pharm Bull*. 2020;43(1):36–40. doi:10.1248/bpb.b18-01021
21. Klimek L, Werminghaus P, Bergmann C, et al. Neuroimmunologie der allergischen Rhinitis: teil 1: zelluläre und humorale Grundlagen. [Neuroimmunology of allergic rhinitis: part 1: cellular and humoral basic principles]. *Hno*. 2023;71(5):337–346. doi:10.1007/s00106-023-01292-z
22. Ruysseveldt E, Steelant B, Wils T, et al. The nasal basal cell population shifts toward a diseased phenotype with impaired barrier formation capacity in allergic rhinitis. *J Allergy Clin Immunol*. 2024;154(3):631–643. doi:10.1016/j.jaci.2024.04.021
23. Klimek L, Werminghaus P, Bergmann C, et al. Neuroimmunologie der allergischen Rhinitis Teil 2: interaktionen von Neuronen und Immunzellen und neuroimmunologische Einheiten. [Neuroimmunology of allergic rhinitis part 2: interactions of neurons and immune cells and neuroimmunological units]. *Hno*. 2023;71(6):413–421. doi:10.1007/s00106-023-01304-y
24. Lan YA, Guo JX, Yao MH, Kang YT, Liao ZR, Jing YH. The role of neuro-immune interactions in the pathology and pathogenesis of allergic rhinitis. *Immunol Invest*. 2024;53(7):1013–1029. doi:10.1080/08820139.2024.2382792
25. Wang Y, Song XY, Wei SZ, et al. Brain response in allergic rhinitis: profile and proposal. *J Neurosci Res*. 2023;101(4):480–491. doi:10.1002/jnr.25159
26. Cai S, Lou H. [Neuroimmunomodulation in allergic rhinitis]. *Lin Chuang Er Bi Yan Hou Tou Jing Wai Ke Za Zhi*. 2021;35(9):859–864. doi:10.13201/j.issn.2096-7993.2021.09.021
27. Konstantinou GN, Konstantinou GN, Koulias C, Petalas K, Makris M. Further understanding of neuro-immune interactions in allergy: implications in pathophysiology and role in disease progression. *J Asthma Allergy*. 2022;15:1273–1291. doi:10.2147/jaa.S282039
28. Xiao C, Feng L, Yang W. Inhibition of dendritic cell autophagy alleviates the progression of allergic rhinitis by inhibiting Th1/Th2/Th17 immune imbalance and inflammation. *Histol Histopathol*. 2025;40(2):237–247. doi:10.14670/hh-18-769
29. Jiang F, Yan A. IL-4 rs2243250 polymorphism associated with susceptibility to allergic rhinitis: a meta-analysis. *Biosci Rep*. 2021;41(4). doi:10.1042/bsr20210522
30. Nagase H, Ueki S, Fujieda S. The roles of IL-5 and anti-IL-5 treatment in eosinophilic diseases: asthma, eosinophilic granulomatosis with polyangiitis, and eosinophilic chronic rhinosinusitis. *Allergol Int*. 2020;69(2):178–186. doi:10.1016/j.alit.2020.02.002
31. Amin K, Issa SM, Ali KM, et al. Evidence for eosinophil and IL-17 mediated inflammation in allergic rhinitis. *Clin Mol Allergy*. 2020;18:6. doi:10.1186/s12948-020-00117-6
32. Gupta RK, Gupta K, Dwivedi PD. Pathophysiology of IL-33 and IL-17 in allergic disorders. *Cytokine Growth Factor Rev*. 2017;38:22–36. doi:10.1016/j.cytogfr.2017.09.005
33. Bayrak Degirmenci P, Aksun S, Altin Z, et al. Allergic rhinitis and its relationship with IL-10, IL-17, TGF-β, IFN-γ, IL 22, and IL-35. *Dis Markers*. 2018;2018:9131432. doi:10.1155/2018/9131432
34. Chen RX, Lu WM, Lu MP, et al. Polymorphisms in microRNA target sites of TGF-β signaling pathway genes and susceptibility to allergic rhinitis. *Int Arch Allergy Immunol*. 2021;182(5):399–407. doi:10.1159/000511975
35. Lei F, Zhu D, Sun J, Dong Z. Effects of minimal persistent inflammation on nasal mucosa of experimental allergic rhinitis. *Am J Rhinol Allergy*. 2010;24(1):e23–8. doi:10.2500/ajra.2010.24.3414
36. Tomazic PV, Darnhofer B, Birner-Gruenberger R. Nasal mucus proteome and its involvement in allergic rhinitis. *Expert Rev Proteomics*. 2020;17(3):191–199. doi:10.1080/14789450.2020.1748502
37. Nur Husna SM, Tan HT, Md Shukri N, Mohd Ashari NS, Wong KK. Nasal epithelial barrier integrity and tight junctions disruption in allergic rhinitis: overview and pathogenic insights. *Front Immunol*. 2021;12:663626. doi:10.3389/fimmu.2021.663626
38. Koksal ZG, Uysal P, Erdogan O, Cevik O. The association between allergic rhinitis and airway dysfunction and nasal endothelial damage and oxidative stress. *Rhinology*. 2023;61(3):272–282. doi:10.4193/Rhin22.484
39. Qin Z, Xie L, Li W, Wang C, Li Y. New insights into mechanisms Traditional Chinese Medicine for allergic rhinitis by regulating inflammatory and oxidative stress pathways. *J Asthma Allergy*. 2024;17:97–112. doi:10.2147/jaa.S444923
40. Han M, Lee D, Lee SH, Kim TH. Oxidative stress and antioxidant pathway in allergic rhinitis. *Antioxidants*. 2021;10(8). doi:10.3390/antiox10081266
41. Piao CH, Fan Y, Nguyen TV, et al. PM(2.5) Exacerbates Oxidative Stress and Inflammatory Response through the Nrf2/NF-κB Signaling Pathway in OVA-Induced Allergic Rhinitis Mouse Model. *Int J Mol Sci*. 2021;22(15):8173. doi:10.3390/ijms22158173
42. Kang C, Li X, Liu P, et al. Tolerogenic dendritic cells and TLR4/IRAK4/NF-κB signaling pathway in allergic rhinitis. *Front Immunol*. 2023;14:1276512. doi:10.3389/fimmu.2023.1276512

43. Lin X, Hu X, Zhang J, Luo J, Qin G, Jiang L. Gut microbiota, allergic rhinitis, vasomotor rhinitis, Mendelian randomization, causal association. *Braz J Otorhinolaryngol*. 2024;90(6):101491. doi:10.1016/j.bjorl.2024.101491
44. Pang W, Jiang Y, Li A, et al. Bacteroides thetaiotaomicron Ameliorates Experimental Allergic Airway Inflammation via Activation of ICOS(+) Tregs and Inhibition of Th2 Response. *Front Immunol*. 2021;12:620943. doi:10.3389/fimmu.2021.620943
45. Panpan Z, Jinli H, Qiuhong L, et al. Changes in respiratory tract and gut microbiota in AR mice and their relationship with Th1/Th2/Treg. *Microb Pathog*. 2024;195:106881. doi:10.1016/j.micpath.2024.106881
46. Lin Y, Rui X, Li Y. Role of gut microbiota in children with allergic rhinitis with high serum total IgE level. *Lin Chuang Er Bi Yan Hou Tou Jing Wai Ke Za Zhi*. 2020;34(12):1123–1128. doi:10.13201/j.issn.2096-7993.2020.12.016
47. Jin Q, Ren F, Dai D, Sun N, Qian Y, Song P. The causality between intestinal flora and allergic diseases: insights from a bi-directional two-sample Mendelian randomization analysis. *Front Immunol*. 2023;14:1121273. doi:10.3389/fimmu.2023.1121273
48. Li Y, Sun L, Zhang Y. Programmed cell death in the epithelial cells of the nasal mucosa in allergic rhinitis. *Int Immunopharmacol*. 2022;112:109252. doi:10.1016/j.intimp.2022.109252
49. Racanelli AC, Kikkers SA, Choi AMK, Cloonan SM. Autophagy and inflammation in chronic respiratory disease. *Autophagy*. 2018;14 (2):221–232. doi:10.1080/15548627.2017.1389823
50. He YQ, Qiao YL, Xu S, et al. Allergen induces CD11c(+) dendritic cell autophagy to aggravate allergic rhinitis through promoting immune imbalance. *Int Immunopharmacol*. 2022;106:108611. doi:10.1016/j.intimp.2022.108611
51. Yu Mm Y, Yan Mm J. Study on the mechanism of allergic rhinitis based on the expression of FIB, PCT, hs-CRP, and Th17/Treg-IL10/IL-17 axis balance. *Am J Rhinol Allergy*. 2023;37(4):429–437. doi:10.1177/19458924231162737
52. Li J, Li Y. Autophagy is involved in allergic rhinitis by inducing airway remodeling. *Int Forum Allergy Rhinol*. 2019;9(11):1346–1351. doi:10.1002/alr.22424
53. Nian JB, Zeng M, Zheng J, et al. Epithelial cells expressed IL-33 to promote degranulation of mast cells through inhibition on ST2/PI3K/mTOR-mediated autophagy in allergic rhinitis. *Cell Cycle*. 2020;19(10):1132–1142. doi:10.1080/15384101.2020.1749402
54. Zhou H, Zhang W, Qin D, et al. Activation of NLRP3 inflammasome contributes to the inflammatory response to allergic rhinitis via macrophage pyroptosis. *Int Immunopharmacol*. 2022;110:109012. doi:10.1016/j.intimp.2022.109012
55. Cheng N, Wang Y, Gu Z. Understanding the role of NLRP3-mediated pyroptosis in allergic rhinitis: a review. *Biomed Pharmacother*. 2023;165:115203. doi:10.1016/j.biopha.2023.115203
56. Qi S, Barnig C, Charles AL, et al. Effect of nasal allergen challenge in allergic rhinitis on mitochondrial function of peripheral blood mononuclear cells. *Ann Allergy Asthma Immunol*. 2017;118(3):367–369. doi:10.1016/j.anai.2016.11.026
57. Ding H, Lu X, Wang H, Chen W, Niu B. NLRP3 inflammasome deficiency alleviates inflammation and oxidative stress by promoting PINK1/Parkin-mediated mitophagy in allergic rhinitis mice and nasal epithelial cells. *J Asthma Allergy*. 2024;17:717–731. doi:10.2147/jaa.S467774
58. Lin Q, Li S, Jiang N, et al. Inhibiting NLRP3 inflammasome attenuates apoptosis in contrast-induced acute kidney injury through the upregulation of HIF1A and BNIP3-mediated mitophagy. *Autophagy*. 2021;17(10):2975–2990. doi:10.1080/15548627.2020.1848971
59. Cheng L, Chen J, Fu Q, et al. Chinese Society of Allergy Guidelines for Diagnosis and Treatment of Allergic Rhinitis. *Allergy Asthma Immunol Res*. 2018;10(4):300–353. doi:10.4168/aair.2018.10.4.300
60. Liu X, Shufang L, Xiang W, Guoqing F. Correlation study of Th17/Treg expression in allergic rhinitis in children with Chinese medicine patterns and conditions. *World Sci Technol*. 2023;25(04):1481–1487.
61. Tiantian L, Jiaxin L, Tian F, Yanchun X. A study on the correlation between traditional Chinese medicine constitution and clinical characteristics of 215 adult patients with allergic rhinitis. *J Guangzhou Univ Chin Med*. 2025;42(04):803–812. doi:10.13359/j.cnki.gzxbtcm.2025.04.002
62. Zhigang L, Hongmeng S, Sanli W, Yulin L. Histopathological study on allergic rhinitis treated with Centipeda minima. *Chinese Journal of Traditional Chinese Medicine*. 2005;(04):53–55.
63. Wei Z, Fei Z. Effect of supplementary therapy with astragalus on serum Th1/Th2 in children with allergic rhinitis. *Chin Remedies Clinics*. 2006;(09):680–683.
64. Xiaocao S, Qiang Z, Xiumei Z, Congshan L. Effect of aqueous extract of Aesculus vulgaris on nasal mucosal lesions and serum inflammatory cytokines in Guinea pigs with allergic rhinitis. *J Anhui Univ Trad Chin Med*. 2019;38(06):44–48.
65. Tong L, Peng S, Shan L. Effect of Angelica dahurica emulsion on serum cytokines and Th17/Treg balance in nasal mucosa of mice with allergic rhinitis. *Modern Immunol*. 2020;40(05):402–407.
66. Hong X, Wenqiang L. The influence of Xinyi on Th1/Th2 cytokines in mice with allergic rhinitis. *Clin J Chin Med*. 2023;15(28):90–93.
67. Yanchun C, Shiying X, Hangyu Y, Zhiling C. Study on the improvement effect of Fangfeng water extract on mice with aggravated allergic rhinitis by antibiotics. *Zhejiang Med Sci*. 2023;45(05):453–459+561.
68. Zhaolan S, Xia L, Tingting H, Meiling X. Study on molecular mechanism of Wumei-Fangfeng drug in regulating Treg/Th17 immune balance in allergic rhinitis. *Chin J Immunol*. 2024;40(09):1908–1913.
69. Jieting H, Zengxin X, Beido Z. Analysis of the main effects of mint based on network pharmacology and molecular docking technology. *J Ningde Normal University*. 2024;36(03):273–282. doi:10.15911/j.cnki.35-1311/n.2024.03.002
70. Shaoyu L, Xiaomei T, Yongchang Z, Jie G. Preliminary study on effects of Xixin Oil on blood histamine and pathomorphological change in nasal mucosa in Guinea Pigs with allergic rhinitis. *Chin J Exp Trad Med Formulae*. 2011;17(02):149–151. doi:10.13422/j.cnki.syfjx.2011.02.044
71. Shaoyu L, Xiaomei T, Jie G, Yuanli H. Study on acute toxicity of total alkaloids of processed Radix Aconiti Lateralis and its effects on blood histamine contents and pathomorphological changes in nasal mucosa in allergic rhinitis Guinea pigs. *Chin J Trad Chin Med*. 2011;26 (12):2986–2989.
72. Zeqing H, Xiaojing C, Jianxin Z. Effects of Bombyx Batryticatus on IL-4 and IFN-γ of asthmatic model in Guinea pigs. *Clin J Chin Med*. 2012;4(15):30–31.
73. Yujie L, Jingjing C, Cheng T, Yan M. Optimization of extraction process of chitin from Periostracum Cicadae and evaluation of antioxidant activity and hemostatic effect. *Nat Prod Res Dev*. 2024;36(02):268–278. doi:10.16333/j.1001-6880.2024.2.010
74. Yusong G, Yuqing Q, Shuhang J, Xin L. Study on the anti-inflammatory effect of whole scorpion from Xinjiang on rats with adjuvant arthritis. *Asia-Pacific Trad Med*. 2024;20(06):16–20.

75. Hao L, Mingrui S, Peng Z, Weimin Z. Introduction to the 2025 edition of the Chinese Pharmacopoeia, a major addition and revision of content. *China Pharm Standards*. 2025;26(01):17–22. doi:10.19778/j.chp.2025.01.003
76. Tan J, Qiao Z, Meng M, et al. Centipeda minima: an update on its phytochemistry, pharmacology and safety. *J Ethnopharmacol*. 2022;292:115027. doi:10.1016/j.jep.2022.115027
77. Hua Y, Tan X, Zhang J, et al. Deciphering the pharmacological mechanism of Radix astragali for allergic rhinitis through network pharmacology and experimental validation. *Sci Rep*. 2024;14(1):29873. doi:10.1038/s41598-024-80101-1
78. Jing Z, Li W, Liao W, et al. Fructus Xanthii and Magnolia liliiflora volatile oils liposomes-loaded thermosensitive in situ gel for allergic rhinitis management. *Int J Nanomed*. 2024;19:1557–1570. doi:10.2147/ijn.S445240
79. Li D, Wu L. Coumarins from the roots of Angelica dahurica cause anti-allergic inflammation. *Exp Ther Med*. 2017;14(1):874–880. doi:10.3892/etm.2017.4569
80. Lu T, Yang Y, Yang Z, et al. Mechanisms of the Compound of Magnoliae Flos and Xanthii Fructus essential oils for the treatment of allergic rhinitis based on the integration of network pharmacology, molecular docking, and animal experiment. *Comb Chem High Throughput Screen*. 2025;28. doi:10.2174/0113862073333657241216040227
81. Yusi L, Hui X, Yan L, Qingyun L. Study on the treatment of fangfeng-xinyi drug pair based on network pharmacology mechanism of allergic rhinitis. *J Chin Eye Ear Nose Throat*. 2020;10(04):185–190.
82. Duo T, Shaoyu L, Yuanxin T, Feilong C. Predicting effective components and targets of essential oil of Asari Radix et Rhizoma on allergic rhinitis. *Chin J Exp Trad Med Formulae*. 2015;21(24):126–131. doi:10.13422/j.cnki.syfjx.2015240126
83. Qi D, Kangxi L, Qiaobo Y, Yongsheng W. Reaserch progress on chemical constituents, pharmacological effects and toxicology of Bombyx Batryticatus. *China Drug Eval*. 2023;40(05):402–408.
84. Shan Z, Fang P, Zhiwei Z, Lan Z. Analysis of the molecular mechanism of Magnoliae flos-Cicada slough in the treatment of allergic rhinitis in children based on data mining and network pharmacology. *J Yunnan Univ Chin Med*. 2023;46(03):58–64. doi:10.19288/j.cnki.issn.1000-2723.2023.03.011
85. Kim JS, Oh JM, Choi H, et al. Activation of the Nrf2/HO-1 pathway by curcumin inhibits oxidative stress in human nasal fibroblasts exposed to urban particulate matter. *BMC Complement Med Ther*. 2020;20(1):101. doi:10.1186/s12906-020-02886-8
86. Zhang W, Tang R, Ba G, Li M, Lin H. Anti-allergic and anti-inflammatory effects of resveratrol via inhibiting TXNIP-oxidative stress pathway in a mouse model of allergic rhinitis. *World Allergy Organ J*. 2020;13(10):100473. doi:10.1016/j.waojou.2020.100473
87. Kim SH, Jun CD, Suk K, et al. Gallic acid inhibits histamine release and pro-inflammatory cytokine production in mast cells. *Toxicol Sci*. 2006;91(1):123–131. doi:10.1093/toxsci/kfj063
88. Pan Z, Zhou Y, Luo X, et al. Against NF-κB/thymic stromal lymphopoietin signaling pathway, catechin alleviates the inflammation in allergic rhinitis. *Int Immunopharmacol*. 2018;61:241–248. doi:10.1016/j.intimp.2018.06.011
89. Phan HTL, Nam YR, Kim HJ, et al. In-vitro and in-vivo anti-allergic effects of magnolol on allergic rhinitis via inhibition of ORAI1 and ANO1 channels. *J Ethnopharmacol*. 2022;289:115061. doi:10.1016/j.jep.2022.115061
90. Ke X, Chen Z, Wang X, Kang H, Hong S. Quercetin improves the imbalance of Th1/Th2 cells and Treg/Th17 cells to attenuate allergic rhinitis. *Autoimmunity*. 2023;56(1):2189133. doi:10.1080/08916934.2023.2189133
91. Hua H, Li F, Xi Y, Jiao W, Wen S, Tao Z. To explore the therapeutic effect of myrtle oil, anthocyanin and hyaluronic acid in combination with topical application on allergic rhinitis in rats exposed to PM2.5. *Lin Chuang Er Bi Yan Hou Tou Jing Wai Ke Za Zhi*. 2020;34(8):719–725. doi:10.13201/j.issn.2096-7993.2020.08.010
92. Oh HA, Han NR, Kim MJ, Kim HM, Jeong HJ. Evaluation of the effect of kaempferol in a murine allergic rhinitis model. *Eur J Pharmacol*. 2013;718(1–3):48–56. doi:10.1016/j.ejphar.2013.08.045
93. Zhou YJ, Wang H, Sui HH, Li L, Zhou CL, Huang JJ. Inhibitory effect of baicalin on allergic response in ovalbumin-induced allergic rhinitis Guinea pigs and lipopolysaccharide-stimulated human mast cells. *Inflamm Res*. 2016;65(8):603–612. doi:10.1007/s00011-016-0943-0
94. Chen Q, Shao L, Li Y, et al. Tanshinone IIA alleviates ovalbumin-induced allergic rhinitis symptoms by inhibiting Th2 cytokine production and mast cell histamine release in mice. *Pharm Biol*. 2022;60(1):326–333. doi:10.1080/13880209.2022.2034894
95. Shuo H, Shuang L, Zhe C, Tao P. Tanshinone IIA affects mast cells by modulating the NF-κB pathway in mast cell mediated allergic rhinitis. *J Wuhan University*. 2018;39(02):223–227. doi:10.14188/j.1671-8852.2018.0242
96. Niu Y, She Z, Su C, Zhao Q, Wang S, Xiao B. The effects and the mechanisms of naringenin from Artemisia ordosica Krasch on allergic rhinitis based on mast cell degranulation model and network pharmacology. *J Pharm Pharmacol*. 2022;74(3):397–408. doi:10.1093/jpp/rgab166
97. Kilic K, Sakat MS, Yildirim S, et al. The amendatory effect of hesperidin and thymol in allergic rhinitis: an ovalbumin-induced rat model. *Eur Arch Otorhinolaryngol*. 2019;276(2):407–415. doi:10.1007/s00405-018-5222-y
98. Liu S, Wang C, Zhang Y, et al. Polydatin inhibits mitochondrial damage and mitochondrial ROS by promoting PINK1-Parkin-mediated mitophagy in allergic rhinitis. *FASEB J*. 2023;37(4):e22852. doi:10.1096/fj.202201231RR
99. Oh HA, Seo JY, Jeong HJ, Kim HM. Ginsenoside Rg1 inhibits the TSLP production in allergic rhinitis mice. *Immunopharmacol Immunotoxicol*. 2013;35(6):678–686. doi:10.3109/08923973.2013.837061
100. Xuqing C, Longyun Z, Huaan M, Shufen L. Preliminary study on the improvement effects of Astragaloside IV on allergic rhinitis model mice. *Chin J Immunol*. 2022;38(07):801–807.
101. Yushui F, Yuanzheng F, Hui Y, Lihua Z. Study on 18β glycyrrhetinic acid sodium in improving allergic rhinitis, nasal mucosal tissue disease and th1/th2 balance mechanism in young rats. *Western J Med*. 2021;33(11):1596–1601+1607.
102. Ciyu Y, Tao W, Yunlin J, Xin Z. Inhibitory effect of triptolide on sensitized mast cells and its effect on PI3K/AKT/gsk3-β signaling pathway. *Fujian J Med*. 2017;39(01):61–64. doi:10.20148/j.fmj.2017.01.022
103. Feng L, Wu Y, Peng J, Fei X. Effects of paeoniflorin on immune homeostasis in rats with allergic rhinitis based on IL-33 / ST2 pathway. *Chin J Gerontol*. 2024;44(12):3034–3038.
104. Yalin Z, Chongyang W, Siqi L, Hainan J. Panax notoginseng saponin alleviates allergic rhinitis via ampkdrp1-mediated mitochondrial fission. *Chin Pharmacol Bull*. 2023;39(03):512–519.
105. Yumei G, Zhenwei Z, Lei S. Effect of Polysaccharides from Radix Saplshnikoviae on the immune factors in rats with allergic rhinitis. *Chin Pharm*. 2017;20(07):1188–1191.

106. Li H, Jianying C, Mingbin H. Effects of andrographolide Driping Pills combined with Danxi Yupingfeng granules on immunologic func tion of children with allergic rhinitis. *Chin Pharm*. 2017;28(05):643–645.
107. Lu Y, Yang JH, Li X, et al. Emodin, a naturally occurring anthraquinone derivative, suppresses IgE-mediated anaphylactic reaction and mast cell activation. *Biochem Pharmacol*. 2011;82(11):1700–1708. doi:10.1016/j.bcp.2011.08.022
108. Gao W, Jin Z, Zheng Y, Xu Y. Psoralen inhibits the inflammatory response and mucus production in allergic rhinitis by inhibiting the activator protein 1 pathway and the downstream expression of cystatin-SN. *Mol Med Rep*. 2021;24(3). doi:10.3892/mmr.2021.12291
109. Chen Z, Tao ZZ, Zhou XH, Wu TT, Ye LF. Immunosuppressive effect of sinomenine in an allergic rhinitis mouse model. *Exp Ther Med*. 2017;13(5):2405–2410. doi:10.3892/etm.2017.4237
110. Yanfeng L, Zhenmin L. Protective effect of vitexin on ferroptosis mediated by the Sirt1/FoxO1 pathway on nasal mucosal damage with allergic rhinitis in rats. *Modern Drugs Clinics*. 2024;39(01):14–22.
111. Ling Y, Jie L, Jiangping L, He L. Effect of ephedrine mediated TSLP/OX40L pathway in regulating Th2 type immune response in rats with allergic rhinitis. *Chin J Immunol*. 2022;38(03):319–323.
112. Luo G, Gao M, Lin Q. Integration of bioinformatics analysis, molecular docking and animal experiments to study the therapeutic mechanisms of berberine against allergic rhinitis. *Sci Rep*. 2024;14(1):11999. doi:10.1038/s41598-024-60871-4
113. Shaoli W, Zhi F, Xiangming W. Effects of Lycium barbarum polysaccharides on TH1TH2 cytokines in allergic rhinitis mice based on NF-κb signaling pathway. *J Modern Integr Chin Western Med*. 2024;33(07):928–932.
114. Acar M, Muluk NB, Yigitaslan S, et al. Can curcumin modulate allergic rhinitis in rats? *J Laryngol Otol*. 2016;130(12):1103–1109. doi:10.1017/s0022215116008999
115. Wu S, Xiao D. Effect of curcumin on nasal symptoms and airflow in patients with perennial allergic rhinitis. *Ann Allergy Asthma Immunol*. 2016;117(6):697–702.e1. doi:10.1016/j.anai.2016.09.427
116. Jafarinia M, Sadat Hosseini M, Kasiri N, et al. Quercetin with the potential effect on allergic diseases. *Allergy Asthma Clin Immunol*. 2020;16:36. doi:10.1186/s13223-020-00434-0
117. Tanaka Y, Furuta A, Asano K, Kobayashi H. Modulation of Th1/Th2 cytokine balance by quercetin in vitro. *Medicines*. 2020;7(8). doi:10.3390/medicines7080046
118. Kashiwabara M, Asano K, Mizuyoshi T, Kobayashi H. Suppression of neuropeptide production by quercetin in allergic rhinitis model rats. *BMC Complement Altern Med*. 2016;16:132. doi:10.1186/s12906-016-1123-z
119. Ebihara N, Takahashi K, Takemura H, Akanuma Y, Asano K, Sunagawa M. Suppressive effect of quercetin on nitric oxide production from nasal epithelial cells in vitro. *Evid Based Complement Alternat Med*. 2018;2018:6097625. doi:10.1155/2018/6097625
120. Liu T, Xu J, Wu Y, et al. Beneficial effects of baicalein on a model of allergic rhinitis. *Acta Pharm*. 2020;70(1):35–47. doi:10.2478/acph-2020-0009
121. Xu Y, Xu L, Jian X, Wang Q, Li Z, Ge H. Baicalein attenuates ovalbumin-induced allergic rhinitis through the activation of nuclear receptor subfamily 4 group a member 1. *Immunol Res*. 2025;73(1):32. doi:10.1007/s12026-024-09590-6
122. Qing H, Weiwei Y, Wenguang W, Lingyan P. Baicalein ameliorates allergic rhinitis by inhibiting the NF-κB / STAT3 / ERK signaling pathway. *Chin J Gerontol*. 2024;44(01):165–170.
123. Osmakov DI, Kalinovskii AP, Belozerova OA, Andreev YA, Kozlov SA. Lignans as pharmacological agents in disorders related to oxidative stress and inflammation: chemical synthesis approaches and biological activities. *Int J Mol Sci*. 2022;23(11):6031. doi:10.3390/ijms23116031
124. Lu J, Zhang H, Pan J, et al. Fargesin ameliorates osteoarthritis via macrophage reprogramming by downregulating MAPK and NF-κB pathways. *Arthritis Res Ther*. 2021;23(1):142. doi:10.1186/s13075-021-02512-z
125. Liu J, Yang N, Yi X, et al. Integration of transcriptomics and metabolomics to reveal the effect of ginsenoside Rg3 on allergic rhinitis in mice. *Food Funct*. 2023;14(5):2416–2431. doi:10.1039/d2fo03885d
126. Piazzetta GL, Lobello N, Pelaia C, Preianò M, Lombardo N, Chiarella E. Modulating nasal barrier function and tissue remodeling in inflammatory diseases: the role of ginseng and its bioactive compounds. *Tissue Barriers*. 2025;2470477. doi:10.1080/21688370.2025.2470477
127. Qiong G, Di N. Effect and mechanism of polydatin on inflammatory response in mice with allergic rhinitis. *Chin J Clin Pharmacol*. 2023;39(20):2951–2955. doi:10.13699/j.cnki.1001-6821.2023.20.014
128. Xuefeng B, Zhengxiang W, Zhengju H. Effect of 18β–glycyrrhetinic acid on nasal mucosal remodeling in rats with allergic rhinitis. *Chin J Clin Pharmacol*. 2020;36(14):2103–2106. doi:10.13699/j.cnki.1001-6821.2020.14.054
129. Liuqing Z, Jing H, Kang P, Zhiyong Z. Observation of intestinal allergy reaction of sodium glycyrrhetinate in rats with allergic rhinitis. *Ningxia J Med*. 2019;41(08):728–729. doi:10.13621/j.1001-5949.2019.08.0728
130. Junxiu R, Yan H, Zhenxia Z, Anna R. Theeffectsofpolysaccharidesfromradixsandifoliaejaponicaeonbehavior, AQP5andnasalmucosatissuesofallergicrhinitisratsbasedonNF-KB/STAT3signalingpathway. *Western J Med*. 2023;35(01):39–45.
131. Xing W, Lifu D, Bincheng Y, Renqiang M. Impact of emodin on inflammatory response in mice with allergic rhinitis by regulating microRNA-375/Kruppel-like factor 5 axis. *J Yunnan Minzu Univ*. 1–14.
132. Jiajun Z, Xuecheng H, Qindong L, Weizhen X. Effect of Xiao Qinglongtang on IL-33/ST2 signaling pathway in mice with allergic rhinitis. *Chin J Exp Trad Med Formulae*. 2022;28(14):13–19. doi:10.13422/j.cnki.syfjx.20220806
133. Hongjian S, Yu S, Yingchun H, Daofa T. Effects of Jiawei Buzhong Yiqi decoction on the expression activity of SP and SPR innasal mucosa of rats with allergic rhinitis. *Chin J Trad Chin Med*. 2015;30(03):918–920.
134. Qixue D, Xiaoxiao W. Effects of Linggui Zhugan decoction on nasal hypersecretion in allergic rhinitis rats. *Asia-Pacific Tradit Med*. 2024;20(06):12–15.
135. Liang X. *Study on the mechanism of Ephedra fuzi xixin decoction in the treatment of yang-deficiency allergic rhinitis based on intestinal flora and network pharmacology* [Doctor]. Southern Medical University; 2020. https://link.cnki.net/doi/10.27003/d.cnki.gojyu.2020.000267.
136. Jing L, Hao L, Huangxing L. Effects of modified Xiaochaihu decoction on TCM symptoms and immune function in allergic rhinitis. *World Chin Med*. 2023;18(24):3547–3550+3555.
137. Ying G, Jun X, Zhimei Q, Hong M. Yiqi Zhiqiu decoction assisted mometasone furoate nasal spray study on the therapeutic efficacy of treating allergic rhinitis and the changes of CCL17 and YKL-40. *Chin J Trad Chin Med*. 2024;42(05):33–36. doi:10.13193/j.issn.1673-7717.2024.05.007
138. Weihua Z, Zho L, Junyi W, Junjun C. Effects of Guizhi decoction on the expression of AQP5 and cAMP/PKA-CREB signaling pathway in the epithelial cells of nasal mucosa of allergic rhinitis rats. *Chin J Trad Chin Med*. 2016;31(01):283–287.

139. Fei Y, Peng L, Bentao W, Qiang Z. Clinical observation on guizhi plus huangqi decoction combined with acupuncture for treatment of lung-qi deficiency and cold syndrome in allergic rhinitis. *Tianjin J Trad Chin Med*. 2024;41(03):349–353.
140. Wenna F, Weihua Z, Zho L, Junjun C. Effect of Mahuang decoction on the inflammation, AQP5 and cAMP /PKA-CREB in rats with allergic rhinitis. *Acta Chin Med*. 2017;32(09):1603–1608. doi:10.16368/j.issn.1674-8999.2017.09.423
141. Xiaodan W. *Effect of Kangminjian on expression of Claudin-1, JAM-A and Zo-1 in nasal epithelium of Ar mice* [Master]. 2023. https://link.cnki.net/doi/10.26987/d.cnki.gcdtc.2023.000146.
142. Wenjing P, Peiming Y, Junhui H, Hong L. Effect and safety of Biminfang combined with montelukast sodium on children with allergic rhinitis. *J China Prescript Drug*. 2019;17(12):91–93.
143. Jingli X, Xinyu Y, Hong Z, Jingyi Z. Evaluation of biminjian in treating perennial allergic rhinitis of lung-qi deficiency and cold type. *Chin J Clin*. 2022;50(07):874–877.
144. Liu HL, Chen HF, Wu YD, et al. Xiaoqinglong decoction mitigates nasal inflammation and modulates gut microbiota in allergic rhinitis mice. *Front Microbiol*. 2024;15:1290985. doi:10.3389/fmicb.2024.1290985
145. Liu QD, Zhou M, Zhang JJ, et al. Metabolomics of nasal lavage fluid in patients with allergic rhinitis treated by Xiaoqinglong decoction. *Zhongguo Zhong Yao Za Zhi*. 2023;48(22):6164–6172. doi:10.19540/j.cnki.cjcmm.20230718.501
146. Aihua T, Jiabao N, Yun C. Curative effect of Buzhong Yiqi decoction combined with budesonide nasal spray on allergic rhinitis and its influenceson helper T cell 1/helper T cell 2 immune balance. *Hebei J Trad Chin Med*. 2023;45(11):1829–1832+1836.
147. Xiang L, Lan H, Yingchun H, Wei S. Inhibitory effects of modified center-supplementing Qi-boosting decoction on the Ex pression of IL-5 in the Spleen tissue of rats with experimental allergic rhinitis in the late-phase. *Lishizhen Med Materia Medica Res*. 2012;23(07):1619–1621.
148. Xu Y. Effect of Linggui Zhugan decoction combined with Piduo Mowder on the expression of TSLP, TNF-α, VCAM-1 and IL-4 in allergic rhinitis rats with spleen deficiency and dampness excess [Master]. 2020. https://link.cnki.net/doi/10.27231/d.cnki.gnmyc.2020.000190.
149. Qixue D, Xiaoxiao W, Hui W. Effects of Lingguizhugan Tang on mucin level in allergic rhinitis rats. *Liaoning J Trad Chin Med*. 1–7.
150. Wei X, Ding M, Liang X, Zhang B, Tan X, Zheng Z. Mahuang Fuzi Xixin decoction ameliorates allergic rhinitis and repairs the airway epithelial barrier by modulating the lung microbiota dysbiosis. *Front Microbiol*. 2023;14:1206454. doi:10.3389/fmicb.2023.1206454
151. Liang X, Liu CS, Wei XH, et al. Mahuang Fuzi Xixin decoction Ameliorates allergic rhinitis in rats by regulating the gut microbiota and Th17/Treg balance. *J Immunol Res*. 2020;2020:6841078. doi:10.1155/2020/6841078
152. Xiujuan D, Le H, Haiyan L, Lainge L. Exploring the effects and mechanisms of Mahuang Fuzi Xixin decoction plus Shen Siwei decoction on allergic rhinitis in rats based on TSLP-OX40L pathway. *Modern J Integr Trad Chin Western Med*. 2023;32(23):3219–3224+3320.
153. Gemin X, Wenhai G, Min D, Yuewu Y. Efficacy of Erxiao decoction on allergic rhinitis in patients with pulmonary qi deficiency and cold pattern and its effect on regulatory Treg cell-related factors:a randomized controlled trial. *J Pract Med*. 2021;37(17):2287–2291.
154. Jun S, Yu L. Clinical research on allergic rhinitis treated by yiqi zhiqiu decoction combined with sublingual Hapten. *Chin J Emerg Trad Chin Med*. 2017;26(10):1693–1695+1699.
155. Jingran C, Zhen Y, Fangfei X, Lanying F. Clinical study on the treatment of allergic rhinitis with lung qi deficiency and cold syndrome by Gui Zhi plus Huang Qi Decoction. *Chin J Clin Physicians*. 2022;50(11):1379–1380.
156. Ruiquan Y, Yuhang Y. Comparative study of modified Mahuang decoction and Western medicine in the treatment of allergic rhinitis. *Trad Chin Med Inner Mongolia*. 2017;36(19):34–35. doi:10.16040/j.cnki.cn15-1101.2017.19.029
157. Jinmei W, Zhen W. Clinical observation on treatment of infantile allergic rhinitis with deficiency of lung and spleen qi by Guominjian combined with natural moxibustion. *Inner Mongolia J Trad Chin Med*. 2024;43(10):2–4. doi:10.16040/j.cnki.cn15-1101.2024.10.046
158. Yongming Z, Jie L, Duo W. Observation on the therapeutic effect of Bi Min Fang on allergic rhinitis of lung qi deficiency and cold. *J Modern Integr Chin Western Med*. 2022;31(08):1092–1095.
159. Xiaocong F, Wei H, Shiqing Z, Xi T. Effect of Bimin decoction on Aquaporin 5 in Nasal Mucosa of allergic rhinitis rats with lung and Spleen Qi deficiency syndrome via NF-κB signaling pathway. *Chin J Exp Trad Med Formulae*. 2020;26(08):81–88. doi:10.13422/j.cnki.syfjx.20200837
160. Ningcong X, Yiwei H, Xi T, Jinhan W. Effect of bimin recipe on TMEM16A/NF-KB/MUC5AC signaling pathway in nasal mucosa of rats with lung and spleen qi deficiency syndrome of allergic rhinitis. *J Trad Chin Med*. 2024;65(08):842–848. doi:10.13288/j.11-2166/r.2024.08.012
161. Jingyi Z, Xinyu Y, Jianwei A, Man W. Effect of biminjian on lymphocyte subcohorte in allergic rhinitis model mice. *J Trad Chin Med*. 2021;62(13):1164–1168+1178. doi:10.13288/j.11-2166/r.2021.13.013
162. Zilu W, Jingbo L, Junjie W, Jitang C. Mechanism of Yupingfeng San against allergic rhinitis through ROS/NLRP3/Caspase-1Signaling pathway. *Chin J Exp Trad Med Formulae*. 2023;29(24):1–10. doi:10.13422/j.cnki.syfjx.20230904
163. Chunjie Z, Jijun S. The Effects of Tongqiao Biyan Granules combined with Azelastine hydrochloride tablets on the inflammatory response, oxidative stress level and Th1 /Th2 balance in patients with allergic rhinitis. *Chinese Journal of Rational Drug Use*. 2020;17(12):55–59.
164. Weiwei W, Xuan C, Hongcheng G. Therapeutic effect of Sanfengtongqiao dripping pills on allergic rhinitis with deficiency of lung and kidney and its regulation on IL-35 and STAT3 pathways. *Liaoning J Trad Chin Med*. 2024;51(07):76–80. doi:10.13192/j.issn.1000-1719.2024.07.022
165. Caifeng H, Xing C, Yong T, Mingxing W. Analysis of the effect of Nasobuchongtongtong granules combined with lupatadine fumarate on micronutrients and ventilatory function in patients with allergic rhinitis. *Chin J Integr Otolaryngol Western Med*. 2021;29(01):46–49. doi:10.16542/j.cnki.issn.1007-4856.2021.01.012
166. Ming S, Xiaoqiang H. Effects of Xanthium powder on inflammation and AQP5 protein expression in allergic rhinitis mice. *Chin J Ethnomed Ethnopharm*. 2021;30(21):19–24.
167. Wenming C, Jingbo L, Huimin W, Junjie W. Effect of Wenfei Zhiliudan in treating Lung Qi deficiency cold type allergic rhinitis and its mechanism. *Chin J Exp Trad Med Formulae*. 2019;25(22):55–59. doi:10.13422/j.cnki.syfjx.20191424
168. Lijuann P, Guangming S, Xuegu X, Peihua L. Experimental study of Xinqin granule associated with H 1 receptor antagonist on allergic rhinitis in Guinea pigs. *Chin J Otorhinolaryngol*. 2010;16(05):330–334+340.179.
169. Quansheng F, Jie Q. Clinical study on Xiangju Capsules combined with cetirizine in treatment of allergic rhinitis in children. *Modern Drugs Clin*. 2023;38(05):1165–1169.
170. Makino T, Ito Y, Sasaki SY, Fujimura Y, Kano Y. Preventive and curative effects of Gyokuheifu-san, a formula of traditional Chinese medicine, on allergic rhinitis induced with Japanese cedar pollens in Guinea pig. *Biol Pharm Bull*. 2004;27(4):554–558. doi:10.1248/bpb.27.554

171. Cheong PK, Ho TM, Chan KL, et al. The efficacy and safety of Yupingfeng Powder with variation in the treatment of allergic rhinitis: study protocol for a randomized, double-blind, placebo-controlled trial. *Front Pharmacol*. 2022;13:1058176. doi:10.3389/fphar.2022.1058176
172. Yu X, Zhang L. The effect of montelukast sodium combined with Tongqiao rhinitis capsule in the treatment of allergic rhinitis. *Minerva Surg*. 2024;79(3):361–363. doi:10.23736/s2724-5691.23.10038-4
173. Zanfeng L, Guang X, Wei W, Xiujuan Z. Effect of Tongqiao Biyan capsule combined with budesonide on allergic rhinitis and its effect on Th17/Treg balance. *Anhui Med Pharm J*. 2024;28(02):399–402.
174. Wang R, Yang T, Feng Q, et al. Integration of network pharmacology and proteomics to elucidate the mechanism and targets of traditional Chinese medicine Biyuan Tongqiao granule against allergic rhinitis in an ovalbumin-induced mice model. *J Ethnopharmacol*. 2024;318(Pt A):116816. doi:10.1016/j.jep.2023.116816
175. Chai K, Wang W, Wang H, et al. Efficacy and safety of biyuan tongqiao granules in the treatment of allergic rhinitis: a meta-analysis of randomized controlled trials. *J Ethnopharmacol*. 2025;344:119572. doi:10.1016/j.jep.2025.119572
176. Xing W, Dongni W, Jing Z. Effect of biyuantongqiao granule combined with nasal lavage on adenoidal hypertrophy and allergic rhinitis in children and its influence on sleep quality. *World J Sleep Med*. 2023;10(08):1808–1811.
177. Mengjuan G, Zhiqiang Z. Study on the regulation of cGAS-STING signaling pathway by Cangerzi powder on enhancing immune and anti-allergic rhinitis effect. *J Liaoning Univ TCM*. 2024;26(08):145–149. doi:10.13194/j.issn.1673-842x.2024.08.030
178. Jing Y, Qingli X. Clinical study on Wenfei Zhiliu Pellets combined with Western Medicine for allergic rhinitis of Lung Qi deficiency-cold type. *New Chin Med*. 2023;55(11):72–76. doi:10.13457/j.cnki.jncm.2023.11.012
179. Jiali B, Lin L, Nan Z, Xinlei Z. Application of Xinqin Granule in treatment of patients with allergic rhinitis and its effect on changes of inflammatory indicators. *Liaoning J Chin Med*. 2024;51(05):123–126. doi:10.13192/j.issn.1000-1719.2024.05.034
180. Liu H, Cui X, Xie YM, Li YY. Pharmacoeconomic evaluation of Xiangju Capsules in treatment of chronic rhinosinusitis. *Zhongguo Zhong Yao Za Zhi*. 2022;47(14):3950–3955. doi:10.19540/j.cnki.cjcmm.20220415.501