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Study on the effects of upper respiratory tract infection, adenoid hypertrophy, and environmental factors on allergic rhinitis

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Abstract: To investigate the direct effects of Upper Respiratory Tract Infection and Adenoid Hypertrophy on Allergic Rhinitis, and to examine the mediating role of a harsh environment in these relationships, a quantitative cross-sectional study was conducted. Data were collected from 387 valid respondents using standardized questionnaires. Descriptive and correlation analyses were performed with SPSS 26.0, while AMOS 24.0 was used to construct a structural equation model to test both direct and mediating effects. Results showed that Upper Respiratory Tract Infection, Adenoid Hypertrophy, and a harsh environment each had significant direct positive effects on Allergic Rhinitis. Importantly, the harsh environment partially mediated the relationships between the two pathological factors (infection and hypertrophy) and Allergic Rhinitis. This suggests that these factors not only directly worsen the condition but also indirectly exacerbate it by increasing susceptibility or exposure in poor environmental conditions. The study confirms an interaction between individual pathological status and environmental factors in Allergic Rhinitis, establishing a combined pathway that amplifies disease severity. Prevention and treatment strategies should adopt an integrated medical and environmental approach. Clinically, proactive management of infections and hypertrophy is essential. At the public health level, improving environmental quality is fundamental to controlling the disease at its source.

Keywords: *Adenoid hypertrophy, Allergic Rhinitis, Harsh environment, Upper respiratory tract infection.*

1. Introduction

Allergic rhinitis (AR) is defined in modern medicine as a nonspecific chronic inflammation of the nasal mucosa mediated by IgE. Traditional Chinese medicine theory classifies it as a nasal obstruction based on clinical characteristics. With accelerating industrialization, the global prevalence of AR has risen to 10%-25%. Statistics indicate that approximately 1.2 billion people worldwide suffer from allergies, with an average of one in five individuals affected [1]. AR significantly diminishes patients' quality of life. Over half of sufferers experience reduced work efficiency due to the condition, while 38% find symptom flare-ups intolerable. Persistent nasal congestion and rhinorrhea not only disrupt sleep patterns but also hinder children's learning, memory, and growth development. Furthermore, AR frequently coexists with asthma, pharyngitis, sinusitis, and secretory otitis media. Consequently, nearly 40% of AR patients also have asthma, necessitating clinicians to adopt a holistic perspective when managing respiratory diseases.

Traditional Chinese medicine theory posits that, in the context of Upper Respiratory Tract Infection (URTI), external pathogenic factors, in combination with internal organ damage, jointly trigger nasal congestion. Deficiency in the lungs, spleen, and kidneys forms the internal foundation for the onset of the condition [2]. When lung qi is deficient, the protective function becomes impaired, and body fluids lose their cohesive control. Impaired spleen function allows dampness to ascend and invade the nasal passages. Kidney yang deficiency generates internal cold, leading to persistent sneezing.

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Against this backdrop of constitutional weakness, external factors such as wind, cold, and fire become key triggers of disease onset. Wind pathogens trigger rapid-onset nasal itching; cold pathogens impede lung qi dispersion; and post-infection stagnation readily transforms into fire, invading the nasal passages. This study posits that adenoid hypertrophy is a pathological product of organ deficiency intertwined with phlegm-stasis, while recurrent upper respiratory tract infections represent external pathogen invasion. Exploring these factors' influence on AR aligns with TCM's diagnostic logic of environmental and individual patient differentiation.

The adenoid occupies a central position within the pharyngeal lymphatic ring. AH exhibits complex pathological associations with AR. AH directly narrows the nasopharyngeal airway, compounded by AR-induced mucosal oedema, forcing patients to resort to mouth breathing [3]. This breathing pattern allows unfiltered air to reach the lower respiratory tract, increasing mucosal irritation from environmental allergens. From an immunological perspective, the inflammatory response of AR can spread along the mucosa to the nasopharynx, stimulating hyperplasia of the adenoid lymphoid tissue. The enlarged adenoids then become a harbor for bacteria and allergens, leading to recurrent respiratory infections. This persistent stimulation releases large amounts of inflammatory mediators, heightening the hyperreactivity of the nasal mucosa. At this point, the patient enters a vicious cycle of inflammatory stimulation, adenoid hypertrophy, drainage obstruction, and worsening infection.

Environmental factors are the primary external causes of AR onset. Among numerous pollution sources, severe pollution poses the greatest danger. Specifically, proteases released by dust mites can damage the respiratory epithelial barrier, increase mucosal permeability, and, in turn, induce sensitization [4]. Dust mite proliferation is highly dependent on indoor temperature and humidity. When indoor relative humidity exceeds 35%, mites begin to survive. Although modern buildings have improved thermal insulation, poor ventilation and moisture accumulation create ideal conditions for dust mite reproduction. Even in high-altitude regions like Kunming, dust mite accumulation in indoor microenvironments remains a primary cause of AR flare-ups. Additionally, physicochemical factors such as PM2.5 and passive smoking often synergize with biological allergens. Researchers must conduct thorough investigations of patients' living environments and analyze dust mite distribution patterns to develop effective prevention and control measures.

AR is a systemic pathology driven by multiple factors. Adenoid hypertrophy provides the physical pathological basis, recurrent infections offer immunological triggers, and environmental allergen exposure principles deliver sustained external stimuli. This study employs data mining and clinical observation to clarify the relative contributions of URTI, AH, and HE in AR pathogenesis. This integrated analytical approach facilitates the optimization of AR clinical diagnosis protocols. Findings will provide a scientific rationale for health maintenance interventions targeting rhinitis management, environmental mitigation, and multifactorial disease control, ultimately enhancing patients' overall quality of life.

2. Historical Documents

2.1. Study on the Correlation Between Upper Respiratory Tract Infection and Allergic Rhinitis

Clinical data confirm that URTI and AR share multiple pathological features [5]. Environmental pollution and smoke exposure directly damage the epithelial barrier of the nasal mucosa, providing a common physical pathway for virus and allergen invasion. Both acute infections and allergic reactions can disrupt the nasal cycle and elevate oxidative stress levels in the nasal cavity, demonstrating their consistency in local pathophysiological processes [6]. Human rhinoviruses and other pathogens can induce an excessive Th2-type immune response. This response directly amplifies the hyperreactivity of the nasal mucosa and significantly exacerbates AR symptoms [7]. Due to the widespread deficiency of key antimicrobial peptides such as β-defensin-2 and LL-37 in AR patients, individuals with allergies exhibit heightened susceptibility to viral infections. This immune deficiency triggers infections that exacerbate allergies, creating a vicious cycle in which allergies worsen susceptibility [8]. The successful implementation of allergen immunotherapy (AIT) and vaccination demonstrates that regulating

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immune balance or blocking infectious agents can effectively curb acute allergic rhinitis (AR) episodes. Clinicians should integrate recurrent upper respiratory tract infections (URTI) with AR into a unified chronic inflammatory chain for diagnosis and treatment. By shifting from a purely anti-infective approach to an anti-inflammatory and immunomodulatory model, healthcare providers can more effectively control disease progression and reduce recurrence rates.

2.2. Study on the Correlation Between Adenoid Hypertrophy and Allergic Rhinitis

Modern medical research confirms that AH and AR frequently coexist in pediatric respiratory diseases, forming a bidirectional pathological relationship where each condition contributes to the other [9]. Epidemiological big data reveals a high degree of synchrony in the geographic and seasonal distribution of search popularity for both conditions. Clinical empirical studies also confirm that children with AH are far more likely to develop AR than healthy individuals [10]. The physical obstruction caused by AH, combined with the mucosal oedema induced by AR, creates a pathological overlap, forcing pediatric patients to resort to mouth breathing persistently. This breathing pattern directly increases exposure to lower respiratory allergens while simultaneously triggering craniofacial developmental abnormalities such as high palate arch deformity. The chronic allergic inflammation triggered by AR provides sustained stimulation for adenoid hyperplasia, with inflammatory mediators induced by postnasal drip triggering reactive proliferation of nasopharyngeal lymphoid tissue. Enlarged adenoids exacerbate nasal mucosal hyperreactivity by obstructing nasal drainage and prolonging allergen residence time, while also increasing the risk of local infection. This bidirectional model, driven by inflammation and influenced by the patient's anatomical structure, explains the fundamental cause of AR's chronic and difficult-to-treat nature [11]. Clinicians should integrate surgical resection, antiallergic therapy, and immunomodulatory regimens as part of comprehensive management to interrupt the chain of events in upper airway inflammation, ultimately improving patient prognosis and quality of life.

2.3. Research on the Correlation Between Harsh Environments and Allergic Rhinitis

Environmental factors play a multi-level critical role in the onset and progression of AR, with their impact mechanisms spanning multiple dimensions, including air pollution, indoor allergen exposure, climate variability, genetics, environmental interactions, and immune inflammation. Air pollution stands as a significant external risk factor; elevated PM2.5 levels not only diminish the quality of life for AR patients but also exacerbate symptoms through chronic inflammatory pathways. Regional modeling studies reveal a significant positive correlation between air pollution and AR incidence rates. Cross-national analyses further confirm that environmental variables, including sociodemographic factors, climatic conditions, pollution types, and urban structures, are closely associated with the epidemiological trends of AR and asthma [12].

Patient cohort studies indicate that the use of air purification devices can reduce the risk of developing respiratory allergic diseases, suggesting that improving indoor air quality could be an effective intervention strategy [13]. Regarding indoor environments, cross-sectional studies emphasize that home ventilation conditions, humidity levels, and dust mite exposure are closely associated with allergic rhinitis [14]. Research has found that Derp, the primary component of house dust mites, activates the NF-κB signaling pathway and inflammasomes in monocytes, thereby enhancing inflammatory responses. This provides molecular evidence for the immune pathogenesis of allergic rhinitis [15]. Among natural environmental and climatic factors, studies in high-pollen grassland regions indicate that AR symptoms during the grass pollen exposure season are closely associated with local airborne transmission characteristics [16]. The pattern of familial allergic diseases reflects the long-term effects of genetic-environmental interactions. At the genetic level, specific polymorphic variants in the GSDMB gene can modulate immune responses in response to environmental exposures, thereby significantly increasing the risk of allergic rhinitis [17].

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Nasal and peripheral blood eosinophil counts showed a significant positive correlation with serum IgE levels, confirming the dominant role of inflammatory cells in allergic mechanisms. Peripheral blood periostin may serve as a potential biomarker for assessing AR severity. At the same time, the high concentration and expression of IL-5 in nasal polyp tissue indicate the critical significance of eosinophilic inflammation in the chronic progression of AR [18]. Regarding environmental disease comorbidities, AR frequently coexists with asthma. Epidemiological studies indicate that AR patients with concomitant asthma exhibit significantly reduced lung function, supporting the notion that both conditions represent distinct manifestations within the same allergic reaction spectrum [19]. Research also indicates that air pollution and indoor ventilation conditions are closely associated with comorbidities such as sleep disorders, highlighting the prominence of respiratory sensitivity among patients [20]. In summary, environmental factors influence the onset and progression of allergic rhinitis through multiple pathways, including air pollution, climate change, exposure to indoor allergens, and infection-induced sensitization. Gene-environment interactions and immune regulatory mechanisms play a central role in this process.

3. Research Framework and Hypotheses

3.1. Research Framework

This study constructs a multifactorial synergistic theoretical model to analyze the pathogenesis of AR. As shown in Figure 1, this framework identifies AH and URTI as core independent variables. These two indicators directly reflect the patient's intrinsic biological pathology and immune status. Environmental factors serve as key mediating variables, linking intrinsic pathological characteristics with external sensitizing triggers. The model's logic indicates that anatomical abnormalities in the nasopharynx caused by AH, combined with a compromised mucosal barrier resulting from recurrent URTI, alter the body's interaction with the external environment. This alteration significantly increases the body's susceptibility and exposure to allergens, such as dust mites and pollutants. Environmental factors ultimately drive the pathological progression of allergic rhinitis through mediating effects. This model systematically elucidates the synergistic mechanisms between intrinsic pathological factors and external environmental influences. The findings provide a theoretical basis for implementing precision prevention and treatment strategies that integrate internal pathological management with external environmental control in clinical practice.

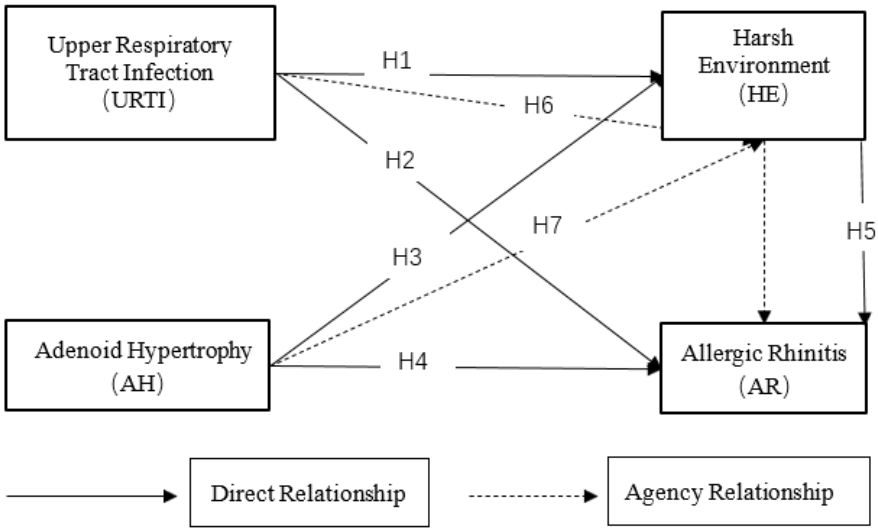

Figure 1.
Theoretical Framework.

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3.2. Research Hypotheses

3.2.1. Relationship Between Upper Respiratory Tract Infection and Harsh Environment

Multiple studies have demonstrated the critical role of environmental factors in the development of AH. Researchers have found that exposure to secondhand smoke, crowded household conditions, and large family structures significantly increase the likelihood of developing AH [21]. Individuals with allergic constitutions face a significantly heightened risk of developing AR in the aforementioned environments [22]. Internet big data analysis reveals that Chinese public search trends for AR and AH are highly consistent, with search volume declining significantly during pandemic lockdowns. This phenomenon demonstrates that changes in environmental exposure levels directly influence the prevalence patterns of both conditions [23]. Nasal microbiome studies confirm that chronic inflammation triggered by AR and AH disrupts the nasal microbiome, leading to microbial imbalance [24]. Fluctuations in air quality and ecological environments thus alter the biological stability of the upper respiratory tract [25].

H_1: Environmental Factors show a significant positive correlation with Upper Respiratory Tract Infection.

3.2.2. Relationship Between Upper Respiratory Tract Infection and Allergic Rhinitis

Immunological experiments elucidated the intrinsic mechanism linking AH and AR. Research indicates that patients with AH complicated by AR exhibit elevated levels of eosinophils, basophils, and total IgE in their adenoid tissue. This finding confirms the presence of an active allergic immune response within the local tissue [26]. Researchers observed a significant increase in specific ILC2 cell populations in patients with AH complicated by AR through flow cytometry, a finding that directly reveals the pathological process of mucosal immunity [27]. Researchers have confirmed that regulating Treg and TGF-β1 levels not only aids in diagnosing AR with AH but also effectively predicts the risk of recurrence after surgery [28]. Clinical researchers further demonstrated the pivotal role of the inflammatory microenvironment in the comorbidity mechanism of AR and AH by measuring cytokines in nasal secretions [29].

H_2: Upper Respiratory Tract Infection and Allergic Rhinitis show a significant positive correlation.

3.2.3. Relationship Between Adenoid Hypertrophy and Harsh Environment

Multiple empirical studies have identified environmental exposure as a key contextual factor in the development of AH. Internet search data analysis confirms that the prevalence intensity of AH is highly synchronized with AR search volume. The decline in search activity during the pandemic further demonstrates that changes in environmental exposure patterns directly influence disease transmission patterns [30]. Air pollution and inhaled allergens constitute common pathogenic triggers for both AR and allergic rhinitis with AR-AH in patients [31]. Genomic analysis reveals that when AR-AH coexists, chronic inflammation induces severe disruption of the nasal core microbiota. Furthermore, chronic inflammation triggered by environmental allergens persistently stimulates the adenoids, leading to tissue hypertrophy [11]. The high prevalence of sinusitis coexisting with AH among asthma patients demonstrates that environmental factors exert a unified detrimental effect on overall respiratory health [32].

H_3: Adenoid Hypertrophy shows a significant positive correlation with Harsh Environment.

3.2.4. Relationship between Adenoid Hypertrophy and Allergic Rhinitis

Immunological mechanisms form a crucial basis for the association between AH and AR. Research has found that ILC2 cells are significantly increased in the adenoid tissue of children with AH and AR, suggesting the role of innate immune responses in these comorbid conditions [33]. Further immunophenotypic analysis revealed specific alterations in the immune characteristics of the adenoids in children with AR and AH who were sensitized to multiple allergens [34]. The study revealed immunological infiltration by eosinophils and basophils, and elevated IgE levels, in the adenoids of AH and AR patients, demonstrating the impact of the allergic inflammatory microenvironment on adenoid

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tissue [35]. Further research confirms that immunotherapy targeting specific allergens significantly reduces the risk of recurrence following AH surgery. Although no detailed summary of the cytokine profile analysis in nasal secretions was provided, the study's focus underscores the importance of the local immune environment in comorbid conditions.

H4: Adenoid Hypertrophy and Allergic Rhinitis Show a Significant Positive Correlation

3.2.5. Relationship Between Allergic Rhinitis and Harsh Environments

Environmental factors directly govern the onset and progression of AR. Air pollution is the primary driver of childhood illnesses. Particularly indoors, dust mites, mould, dander, and secondhand smoke continuously irritate the nasal mucosa, increasing the likelihood that patients will develop allergies [36]. Outdoor pathogens also pose a threat. Climate warming has extended the active periods of pollen and mould spores, while rising temperatures have lengthened the pollen season. This increases public exposure and exacerbates symptoms [37]. Populations in highly polluted areas often face greater disease burdens. Genetic factors provide a predisposition to disease, but environmental exposures ultimately determine the onset of clinical symptoms. Susceptible populations rapidly develop allergic reactions under environmental stressors. Environmental factors collectively shape disease prevalence through multiple pathways. Allergens trigger localized inflammation, while climate fluctuations alter disease distribution on a macro scale [38]. Medical interventions must prioritize air quality management and pollution reduction, as improving the environment is fundamental to alleviating the disease burden [39].

H5: Allergic Rhinitis shows a positive correlation with Harsh Environment

3.2.6. The Role of Harsh Environments in Upper Respiratory Tract Infections and Allergic Rhinitis

Harsh Environment serves as the critical intermediary between URTI and AR. Allergic conditions significantly exacerbate clinical symptoms following infection. When exposed to viral challenges, the immune systems of AR patients often mount more intense inflammatory responses, leading to worsening fever and cough [40]. Imbalances in the nasal microbiome amplify this susceptibility. Fluctuations in environmental temperature and humidity directly damage the nasal mucosal barrier, creating favorable conditions for pathogen invasion. Various pollutants accelerate this pathological process. Outdoor air pollution, indoor volatile organic compounds, and secondhand smoke collectively undermine the respiratory tract's defence system. High concentrations of mould spores directly increase the incidence of allergies and the severity of bronchial inflammation in the population [41].

Exposure to mould in humid environments triggers both allergic rhinitis and infection-like symptoms such as breathing difficulties. Chemicals such as perfluorooctane sulfonic acid (PFOS) interfere with immune recognition at the molecular level, leading to abnormal allergic reactions. Clinical intervention results confirm the subtle association between allergies and infections. While suppressing allergies, certain biologics unexpectedly increased the incidence of URTI [42]. This demonstrates that environmental factors and immune status jointly regulate disease progression. Harsh environments significantly diminish the efficacy of conventional medications.

While antihistamines alleviate nasal discomfort, they cannot eliminate the threat of infection posed by environmental factors. Relying solely on antipyretics or antibiotics for pediatric patients is insufficient to prevent recurrent illnesses. Prevention and control efforts must prioritize improving environmental quality. Only by reducing exposure to pollutants can we strengthen the foundation of immunity and truly break the chain of transmission between acute respiratory infections (ARIs) and URTIs.

H6 The Role of Harsh Environments in Upper Respiratory Tract Infections and Allergic Rhinitis

3.2.7. Harsh Environment Mediates Between Adenoid Hypertrophy and Allergic Rhinitis

Harsh environmental conditions directly drive the pathological progression from adenoid hypertrophy to allergic rhinitis. While conventional medications can block local inflammatory

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pathways, persistent environmental stressors often counteract their therapeutic effects. Clinical data indicate that continuous exposure to the environment continuously activates the body's immune response. Air pollutants and chemical irritants systematically compromise the nasal defence system. These factors induce chronic mucosal inflammation, laying the groundwork for the progression from adenoid hypertrophy to allergic rhinitis. Modern lifestyles have fundamentally altered the patterns of human interaction with the environment [22].

The urbanization process modulates the immune characteristics of susceptible populations, leading to sequential lesions in respiratory tissues. Bacterial biofilms exert potent synergistic effects with physical factors, jointly activating local immunity. This persistent stimulation significantly accelerates the pathological transformation process of nasopharyngeal tissues. Exposure patterns exacerbate mucosal oedema, directly increasing the risk of adenoid hypertrophy. Environmental quality ultimately determines disease outcomes. Medical interventions must incorporate environmental management; otherwise, medication alone cannot break the vicious cycle of inflammation. Improving ecological exposure is the primary step in prevention and treatment, as this strategy effectively reduces the overall burden on the immune system.

H_7: Harsh Environment Mediates Between Adenoid Hypertrophy and Allergic Rhinitis

4. Methodology

4.1. Participants

This study employed purposive sampling techniques to select clinical subjects. The research team targeted patients with a combination of AH, URTI, and AR. This sample combination precisely targets core biological variables in the research model, providing an entry point for analyzing immune mechanisms in the context of multiple coexisting diseases. Subjects' ages are strictly restricted to 18 to 40 years. As the existing literature predominantly focuses on young and middle-aged adults, this approach enables more precise elucidation of the pathological continuity of AH across adulthood.

Environmental exposure intensity serves as the core threshold for sample selection. Between October and December 2025, the air quality index (AQI) in participants' residential areas must frequently exceed standards. Specifically, the cumulative number of days with an AQI exceeding 151 (moderate pollution) must reach 20 or more [43]. This high-concentration exposure to pollutants provides a real-world model for studying how environmental factors damage the mucosal barrier. Study participants must experience acute exacerbations of all three conditions during periods of heavy pollution. This temporal overlap helps isolate the combined effects of infection and environmental factors on allergic reactions.

Researchers continuously assessed participants' compliance and data integrity throughout the study. All finalized samples were submitted with comprehensive symptom questionnaires, environmental scales, and quality-of-life reports. The rigorous screening process yielded a database characterized by both high exposure and multiple disease manifestations. These participants formed natural physiological models of biological-environmental interactions. The raw data authentically documented how anatomical abnormalities and inflammation synergistically contribute to prolonged disease progression under severe stress conditions, providing foundational evidence for subsequent mechanistic studies.

4.2. Instruments

This study employs a structured questionnaire as its primary data collection tool. During the design phase, the research team strictly adhered to academic standards, drawing on established domestic and international scales while implementing localized revisions tailored to the specific contexts of URTI, AH, and HE. These adjustments ensure the measurement tool possesses excellent reliability and validity.

The questionnaire comprises two core sections. The first section collects respondents' basic information. This part primarily gathers demographic characteristics and medical history background, covering five items: gender, age, educational background, disease type, and duration of illness. These

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data clearly outline respondents' fundamental profiles and provide classification criteria for subsequent group differentiation analysis. Part Two comprises the core measurement scales for the construct. This section focuses on quantifying and evaluating four core variables: URTI, AH, HE, and AR. All scales uniformly employ a five-point Likert scale, ranging from 1 (Strongly Disagree) to 5 (Strongly Agree).

Respondents are required to rate each item independently based on their genuine perceptions. The research team established 12 measurement items to enhance data stability. Each core variable corresponds to three associated items, thereby comprehensively capturing the construct's essence. The multidimensional measurement approach effectively reduces random errors from reliance on single items, significantly strengthening internal consistency.

Table 1.
Construct Scale.

Indicator	Cod	Survey Content	References
Upper Respiratory Tract Infection (URTI)	URTI-1	I believe that every time I develop an upper respiratory tract infection, my allergic rhinitis symptoms, such as sneezing, a runny nose, and nasal itching, become more severe.	Jain et al. [44]
	URTI-2	I feel that the frequency of upper respiratory tract infections directly affects how often my allergic rhinitis flares up. The more frequent the infections, the more likely rhinitis is to flare.	Varricchio et al. [45]
	URTI-3	After recovering from an upper respiratory tract infection, my allergic rhinitis symptoms typically persist longer than before the infection, and my nose feels more susceptible to getting sick.	Jain et al. [44]
Adenoid Hypertrophy (AH)	AH-1	I believe my adenoid hypertrophy issues, such as persistent nasal congestion and mouth breathing, significantly exacerbate nasal discomfort during episodes of postnasal drip.	Althobaiti et al. [46]
	AH-2	I feel that due to nasal obstruction caused by adenoid hypertrophy, my allergic rhinitis symptoms are more severe than average when exposed to allergens.	Ameli et al. [47]
	AH-3	I believe that if my adenoid hypertrophy is effectively controlled, my allergic rhinitis symptoms will also be significantly alleviated.	Wei et al. [48]
Harsh Environment (HE)	HE-1	I clearly feel that during periods of poor air quality, such as smog or sandstorms, my allergic rhinitis symptoms worsen immediately or within a short time.	Rosario et al. [49]
	HE-2	I believe that prolonged exposure to environments with significant air pollution has made my nasal mucosa more sensitive, making it easier for even minor irritants to trigger allergic rhinitis.	Higgins and Reh [50]
	HE-3	During periods of poor air quality, I need to use my allergy medication more frequently to keep my symptoms within an acceptable range.	Bernstein et al. [51]
Allergic Rhinitis (AR)	AR-1	I believe harsh environments significantly impact upper respiratory tract infections, particularly by triggering allergic rhinitis.	Wu et al. [52]
	AR-2	I believe that harsh environments significantly impact adenoid hypertrophy, notably by triggering allergic rhinitis.	Wang [53]
	AR-3	I find that in cases of obvious infection and environmental pollution, my allergic rhinitis sometimes flares up suddenly and unexpectedly.	Wang [53]

4.3. Sample and Data Collection

The research team conducted data collection in Taiyuan City, Shanxi Province, China. The study population comprised relevant patients aged 10 to 40 years across major hospitals in the city. Team members executed a rigorous multi-stage process during data collection and cleaning. Researchers employed an oversampling method. A total of 534 questionnaires were distributed, which may have led some respondents to provide casual or incomplete answers. This oversampling strategy ensured a sufficient margin for subsequent screening of invalid samples.

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Researchers cleaned the raw data through a three-step procedure [54]. The team first discarded questionnaires with missing responses to core items. Subsequently, they excluded samples that did not meet the age criteria of 10 to 40 years. Finally, researchers removed low-quality questionnaires characterized by extremely short completion times or abnormal response patterns. This screening process ultimately yielded 387 valid samples. The effective response rate for this survey was 72.4%. This sample size met the requirements for complex model analysis. The high-quality data supported subsequent reliability and validity testing, structural equation modelling analysis, and scientific hypothesis verification.

5. Results

5.1. Sample Analysis

The core of this study lies in exploring how adenoid hypertrophy and upper respiratory tract infection influence allergic rhinitis through the harsh environment effect. Before formally testing the research hypotheses, we used SPSS 26.0 to assess the reliability and validity of the research instruments.

Reliability reflects the consistency and stability of measurement results [55]. This study employed Cronbach's alpha and composite reliability (CR) to assess the internal consistency of the core scales. Similarly, validity aims to ensure that measurement tools accurately reflect their intended constructs. This research focused on convergent validity, measured using the average variance extracted (AVE) metric. AVE values clearly reveal the extent to which measurement items jointly explain the corresponding construct.

It should be noted that the independent variables URTI and AH in this study were measured using objective data, such as clinical diagnoses or incidence rates. As they are not scale-based measures, they are not subject to the reliability and validity analysis conducted here. This comprehensive reliability and validity assessment ensures the quality of our measurement tools, laying a foundation for the reliability of subsequent model construction and hypothesis testing.

Table 2.
Reliability and Validity Test Results

Dimension	Variable	Factor loadings	Cronbach's	CR Value	AVE Value
Upper Respiratory Tract Infection	URTI -1	0.827			
	URTI -2	0.835	0.834	0.855	0.663
	URTI -3	0.780			
Adenoid Hypertrophy	AH-1	0.827			
	AH-2	0.803	0.818	0.858	0.668
	AH-3	0.821			
Harsh Environment	HE -1	0.824			
	HE -2	0.830	0.819	0.854	0.662
	HE -3	0.786			
Allergic Rhinitis	AR -1	0.799			
	AR -2	0.836	0.842	0.853	0.66
	AR -3	0.801			

Table 2 clearly demonstrates the reliability and validity of the scales developed in this study. Regarding validity, all measurement items exhibited factor loadings within the high range of 0.780 to 0.836, indicating that each item effectively reflected its corresponding latent construct. Simultaneously, the AVE values for all four dimensions exceeded 0.660, significantly surpassing the widely accepted benchmark of 0.5. This robust performance confirms the scale's ideal convergent validity. Regarding reliability, the scale demonstrated equally strong stability. Cronbach's α for each dimension exceeded 0.818, and CR values all surpassed 0.85. Collectively, these metrics indicate the scale's outstanding internal consistency and stability. Thus, this validation confirms the reliability and validity of the measurement tool, establishing a robust data foundation for our in-depth analysis of the complex relationships among AH, URTI, HE, and AR.

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5.2. Factor Analysis

To examine the construct validity of the core scales in this study, exploratory factor analysis (EFA) was employed. Before factor extraction, researchers must confirm that the data meet the analytical conditions [56]. To this end, two critical tests were conducted. First, the KMO test was used to assess the adequacy of the sample data, a metric reflecting the proportion of common variance among variables. Second, Bartlett's test of sphericity confirmed the presence of strong correlations among the variables, a prerequisite for factor analysis.

Table 3
Principal Component Analysis.

	Ingredients			
Variable	**URTI**	**AH**	**CA**	**PI**
URTI -1		0.827		
URTI -2		0.835		
URTI -3		0.780		
AH- 1			0.827	
AH -2			0.803	
AH -3			0.821	
HE -1				0.824
HE -2				0.830
HE -3				0.786
AR -1	0.799			
AR -2	0.836			
AR -3	0.801			
KMO	0.871			
χ^2	2076.578			
df	66			
Significance	0.000			
Eigenvalue (Math.)	5.088	1.465	1.264	1.166
Percentage of Variance （%）	42.4	12.206	10.532	9.716
Percentage of Cumulative Variance （%）	42.4	54.606	65.138	74.854

Table 3 primarily presents the results of the principal component analysis, which strongly validates the excellent construct validity of the scales developed in this study. The data fully met the prerequisites for factor analysis, with a KMO value of 0.871, well above the commonly accepted threshold of 0.7. Additionally, Bartlett's test of sphericity yielded a highly significant result ($p < 0.001$), indicating shared structure among variables and confirming the suitability of factor extraction. The analysis strictly adhered to the criterion of eigenvalues exceeding 1, ultimately extracting four distinct principal components. These four factors collectively explained 74.854% of the total variance, indicating that the model successfully captured the majority of critical information within the original data.

The factor loading matrix clearly demonstrated the attribution of each item. All items exhibited high loadings of 0.780-0.836 on their target factors, with no problematic cross-loadings. This outcome directly confirms that the four constructs measured in this study, URTI, AH, HE, and AR, are structurally distinct and independent. In summary, this analysis validates the scale's excellent construct validity, providing a robust and reliable measurement foundation for subsequent testing of the research model.

5.3. Correlation Analysis

Before formally testing the mediating effect, this study first employed Pearson correlation analysis to examine the relationships among the four core variables: URTI, AH, HE, and AR. Clarifying the

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direction and strength of associations between these variables is a necessary prerequisite for constructing path models and validating research hypotheses. Simultaneously, to ensure the accuracy of subsequent regression analysis results, this study also conducted multicollinearity diagnostics on the independent variables using the variance inflation factor (VIF) [57].

This examination aimed to confirm that correlations among independent variables were not sufficiently high to cause mutual interference, thereby ensuring model stability and reliability. The findings from this section provide preliminary yet critical data support for the study's subsequent exploration of HE's mediating role between independent variables and excessive AR.

Table 4.
Correlation Analysis Results Among Factors.

		URTI	AH	HE	AR
URTI	Pearson Correlation	1	0.423**	0.411**	0.475**
	Sig (two-tailed)		0.000	0.000	0.000
	Number of cases	387	387	387	387
AH	Pearson Correlation	0.423**	1	0.343**	0.427**
	Sig (two-tailed)	0.000		0.000	0.000
	Number of cases	387	387	387	387
HE	Pearson Correlation	0.411**	0.343**	1	0.461**
	Sig (two-tailed)	0.000	0.000		0.000
	Number of cases	387	387	387	387
AR	Pearson Correlation	0.475**	0.427**	0.461**	1
	Sig (two-tailed)	0.000	0.000	0.000	
	Number of cases	387	387	387	387

At the 0.01 level (two-tailed), the correlation is significant.

Table 4 presents the results of Pearson correlation analysis, providing preliminary yet robust support for the study's hypotheses. Findings indicate statistically significant positive correlations ($p < 0.01$) among all four core variables: URTI, AH, HE, and AR.

This association is particularly evident between independent and dependent variables: URTI (r=.475) and AH (r=.427), both of which exhibit moderately strong positive correlations with AR, directly validating our clinical expectations. Furthermore, HE, the predefined mediating variable in this study, played a pivotal role, as confirmed by the data. It was not only closely linked to both independent variables but also strongly correlated with the dependent variable, AR (r=.461).

From a model-building perspective, these data similarly yield positive outcomes. All correlation coefficients between variables remain within the ideal range of 0.343 to 0.475, allowing this study to rule out multicollinearity as a confounding factor completely. Thus, the data clearly point to a potential mediating path, paving the way for subsequent in-depth testing.

5.4. Model Construction

To ultimately clarify the complex causal network among these four variables, this study employs Structural Equation Modeling (SEM) for in-depth analysis. Using AMOS 26.0, this study integrates all variables into a unified theoretical framework. The core task of this model is to examine the direct effects of URTI, AH, HE, and AR simultaneously, while precisely revealing HE's mediating role. This represents the most rigorous and comprehensive empirical test of the research hypotheses.

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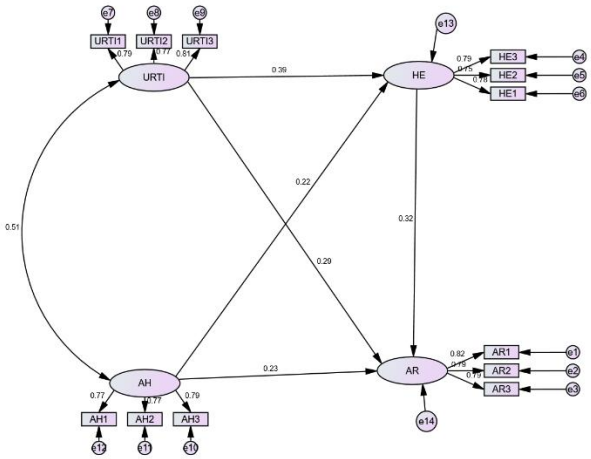

Figure 2.
Structural Equation Model Diagram.

The reliability of SEM hinges primarily on how well it fits the data in this study. To this end, we comprehensively examined the model's fit indices across multiple dimensions, focusing on core metrics such as the chi-square/degrees of freedom ratio (χ^2/df), RMSEA, CFI, and TLI. These indices must collectively meet the criteria, as this directly impacts the model's overall validity. Only after passing this initial threshold do subsequent discussions regarding path and mediating effects become meaningful, and only then can the conclusions drawn in this study be considered valid.

Table 5.
Structural Equation Model Fit Indices (n=387).

Measurement Model	X^2/df	GFI	AGFI	NFI	CFI	RMSEA
Evaluation Indicators	<5	>0.90	>0.90	>0.90	>0.90	<0.08
Outcome Modelling	0.957	0.980	0.968	0.978	1.000	0.001
Test Results	Fit	Fit	Fit	Fit	Fit	Fit

Table 5's data confirm that the model constructed in this study is successful. The model's chi-square-to-degrees-of-freedom ratio (χ^2/df) is 0.957, well below the academic threshold of 5, indicating near-perfect performance. Simultaneously, a series of key indicators measuring model excellence, including GFI and CFI, all fall within the top-tier range of 0.968 to 1.000. Even the Root Mean Square Error of Approximation (RMSEA), which measures model error, is remarkably low at 0.001, virtually negligible.

All indicators converge on the same conclusion: the theoretical framework aligns well with the empirical data. This fully demonstrates that the model developed in this study is ready for precise application in testing subsequent specific paths and mediating effects.

5.5. Path Analysis

The evaluation criteria for path analysis are transparent and rigorous. This study first examines path coefficients, which determine the direction and strength of influence. However, more crucially, path coefficients must pass a dual test: a P-value ($P \leq 0.05$) and a 95% confidence interval that does not include zero. Only when both conditions are met does this study deem the path effect genuine and significant. Otherwise, regardless of the magnitude of the path coefficient, it is considered invalid.

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Table 6
Main Effect Test Results.

Path			Path coefficient	95% confidence interval		P
URTI	→	HE	0.391	0.252	0.482	0.023
AH	→	HE	0.217	0.095	0.334	0.012
URTI	→	AR	0.294	0.149	0.431	0.012
AH	→	AR	0.230	0.117	0.348	0.015
HE	→	AR	0.316	0.213	0.488	0.005

Path analysis in Table 6 reveals two core pathogenic pathways for hypersensitivity rhinitis. URTI and AH directly exacerbate rhinitis without any mediators, with path coefficients of 0.294 and 0.230, respectively. More notably, HE plays a pivotal mediating role in this pathway. Upper Respiratory Tract Infection and Adenoid Hypertrophy first amplify the adverse effects of the environment. This significantly intensified environmental stress then acts back on the patient, powerfully triggering Allergic Rhinitis.

In summary, hypotheses H1, H2, H3, H4, and H5 are established. Statistical tests of all pathways indicate that the synergistic worsening effect between physiological susceptibility and environmental stress is the key mechanism driving disease progression.

Table 7.
Results of Mediating Effect Tests.

Path	Path coefficient	95% confidence interval		P	Conclusion
URTI→HE→AR	0.120	0.052	0.228	0.006	Fit
AH→HE→AR	0.096	0.043	0.168	0.010	Fit

The mediation effect analysis precisely pinpoints HE's central role. First, the model confirms that URTI has an indirect effect on AR via HE. Its extremely low p-value and a confidence interval that strictly excludes zero provide robust statistical validation of this causal pathway. Similarly, AH also exerts a clear, indirect impact on AR through HE as a mediator, with a level of statistical significance comparable to that of the direct effect. In summary, Hypotheses H6 and H7 are supported.

These two fully validated indirect pathways converge on a core mechanism: physiological pathology does not act in isolation on the human body. Still, it ultimately triggers or exacerbates AR through negative environmental influences. This reveals that, in clinical interventions, improving patients' ecological conditions is of equal strategic importance to treating their physiological pathologies.

6. Discussion

6.1. Mechanism of Action of Upper Respiratory Tract Infection on Allergic Rhinitis

The statistical model developed in this study reveals and quantifies a strong positive association between URTI and AR, a finding that strongly resonates with clinical observations. Our integrated analysis decodes the key pathophysiological pathways through which URTI contributes to AR across three core dimensions.

The study findings demonstrate that URTI directly triggers acute symptoms of AR, precisely corroborating the clinical reality of symptom exacerbation following infection. The underlying pathophysiological mechanism involves a dual impact. On one hand, pathogen invasion itself generates acute inflammation [58]. On the other hand, this infection-activated inflammatory environment drastically lowers the allergic response threshold of the nasal mucosa [59]. The immune system's crisis state during infection triggers an overreaction to subsequent allergens, disproportionately amplifying the immune response. Consequently, patients experience a severe exacerbation where even a common cold can push AR to the brink of uncontrollability.

The frequency of URTI occurrence is closely coupled with the frequency of AR flare-ups, providing pathological evidence for the phenomenon that more frequent infections lead to more frequent rhinitis.

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Each URTI represents a severe blow to the nasal barrier. Frequent infections force the nasal mucosal epithelium into an ineffective cycle of damage and repair, potentially leading to permanent structural defects in its barrier function. A dysfunctional barrier opens the door for allergen invasion [60]. At a deeper level, repeated inflammatory stimuli train and reshape the local immune microenvironment, cementing it into a persistent pro-inflammatory state. This state elevates the overall sensitivity baseline of the nasal cavity, making AR triggering no longer dependent on high allergen concentrations and significantly increasing the frequency of attacks.

URTI leaves behind long-term pathological scarring, characterized by nasal hyperreactivity, which underlies the essence of post-infection nasal fragility. The perceived vulnerability experienced by patients directly reflects this post-infection nasal hypersensitivity. Even after the immune system eliminates pathogens, nerve endings remain sensitized, and mucosal damage incurred during infection cannot be immediately reversed. Exposed nerve endings become hyper-sensitive to nonspecific stimuli, while the slowly repairing mucosal barrier remains at a persistently low level of defense. Consequently, patients face prolonged AR symptoms and reduced overall nasal tolerance, a permanent imprint left by the infection.

This discussion confirms that the association between URTI and AR is far from a simple linear cause-and-effect relationship. URTI initiates a self-reinforcing pathological cycle that systematically disrupts nasal homeostasis through three mechanisms: immediate amplification of inflammation, cumulative barrier damage, and long-term nerve sensitization. It is not only an independent risk factor for AR but also a key engine driving progressive disease deterioration. Therefore, in comprehensive AR management strategies, we must elevate proactive URTI intervention to a core strategic position, recognizing it as the critical intervention point to break the vicious cycle of disease progression.

6.2. Mechanism of Action of Adenoid Hypertrophy on Allergic Rhinitis

The structural equation model in this study precisely identified AH as a key physical driver of AR, with its significant positive effect size providing robust quantitative evidence for the complex relationship between the two. This finding not only validates long-standing clinical intuition but also, by integrating patient reports, enables us to systematically decode how AH establishes a pathological platform for the onset and progression of AR across three dimensions.

Research reveals that AH creates a symptomatic synergy with AR's inflammatory oedema through mechanical obstruction. This perfectly explains the clinical observation that adenoid hypertrophy exacerbates rhinitis discomfort. At the pathophysiological level, during AR episodes, the nasal mucosa becomes congested and edematous due to allergic reactions, thereby narrowing the airway. At this point, the enlarged adenoids act as a constant physical obstruction, perched atop the nasopharynx, further compressing the already restricted respiratory passage [61]. The superimposition of these two distinct types of obstruction collectively produces severe nasal congestion and respiratory distress far exceeding what either condition alone could cause. Consequently, patients experience not merely the simple sum of symptoms but an exponentially amplified suffering, with accompanying worsening of mouth breathing and sleep disturbances.

Allergic rhinitis disrupts the normal physiological function of the nasal cavity, creating a closed inflammatory reaction chamber. This provides a core mechanism explaining why allergic rhinitis leads to more intense inflammatory responses. The healthy nasal cavity relies on continuous mucociliary clearance to remove inhaled allergens, pathogens, and inflammatory metabolites. However, the substantial volume of AH severely impedes drainage in the posterior nasal cavity, preventing the smooth evacuation of nasal secretions. This results in significantly prolonged retention of allergens within the nasal cavity, leading to cumulative increases in their exposure to the nasal mucosa and triggering more intense and protracted immune responses [62]. More critically, inflammatory mediators, such as histamine and leukotrienes, produced during allergic reactions, become trapped in this poorly drained environment, forming a high-concentration inflammatory lesion. This lesion

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continuously irritates the nasal mucosa, not only amplifying the intensity of each allergic episode but also maintaining the nasal cavity in a state of heightened sensitivity.

Effective intervention targeting AH significantly alleviates AR symptoms, providing the most direct clinical evidence for the causal link between the two conditions. The resolution of rhinitis following control of Adenoid Hypertrophy serves as compelling counterevidence to both mechanisms described above. When enlarged adenoids are reduced through surgery or medication, the positive effects are immediate. Removing mechanical obstruction directly restores nasal ventilation, instantly improving nasal congestion and breathing patterns. Allergens and inflammatory mediators are promptly cleared, preventing the nasal mucosa from remaining passively in an inflammatory environment. Its reaction threshold gradually returns to normal. This fundamentally removes the physical platform upon which AR relies to intensify, representing a root-cause treatment approach.

Therefore, this study reveals a profound pathological symbiosis between AH and AR. AH is not merely a complication of AR; for patients with AR complicated by AH, any treatment targeting allergies alone is likely to yield minimal results. Clinicians must regard the assessment and management of adenoid issues as the logical starting point and key breakthrough in treating this complex form of rhinitis. Only then can the vicious cycle of the disease be truly broken, achieving long-term symptom control.

6.3. Mechanism of Action of Harsh Environments on Allergic Rhinitis

The multifactorial regression model in this study clearly demonstrates that HE factors are strong independent predictors of AR prevalence and severity. This data-driven conclusion precisely corroborates patients' real-world experiences.

First, air pollutants act as acute irritants and immune adjuvants, directly triggering and amplifying the immediate response of AR. This provides a direct pathophysiological explanation for the widespread phenomenon of environmental influences on rhinitis [63]. When patients are exposed to high-concentration polluted environments such as smog or sandstorms, their nasal mucosa faces a dual assault. On one hand, pollutants such as particulate matter, sulfur dioxide, and ozone are potent chemical irritants. They directly damage epithelial cells, induce nonspecific inflammation, and produce rhinitis-like symptoms.

On the other hand, these particulates adsorb large quantities of allergens onto their surfaces, delivering them deeper and more effectively to immune cells beneath the mucosa. Simultaneously, the pollutants themselves exert potent mediating effects. They activate the immune system, amplifying Th2 responses and thereby significantly enhancing specific reactions to allergens. The synergistic action of these two mechanisms results in patients experiencing a chemically catalyzed, intensified allergic phenomenon during polluted weather.

Secondly, prolonged exposure to polluted environments causes persistent damage to the nasal mucosal barrier, leading to a progressive decline in its defensive function and a permanent reduction in sensitivity thresholds. The nasal mucosal barrier serves as the patient's primary physical and immune defense against external invasions. However, chronic oxidative stress and low-grade inflammation triggered by continuous pollutant exposure disrupt tight junctions between epithelial cells and impair ciliary clearance function. Over time, this barrier exhibits significantly increased permeability. The heightened nasal sensitivity perceived by patients is fundamentally a physiological manifestation of the weakened nasal defense system resulting from prolonged environmental stress.

Ultimately, environmental stressors directly translate into heightened medication requirements, providing the most objective clinical validation of these mechanisms. This study quantifies the direct impact of environmental factors on disease control. When the nasal mucosa remains chronically inflamed and hypersensitive due to prolonged exposure to pollution, its baseline inflammatory levels are elevated. Against this backdrop, any new allergen or pollutant stimulus more readily pushes inflammation levels toward uncontrollable thresholds. Conventional doses of antihistamines or nasal corticosteroids become insufficient [64]. Consequently, patients must increase medication frequency or

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dosage, not due to psychological effects, but as a direct consequence of environmental factors substantially elevating disease severity. This reflects an environmentally induced form of treatment resistance.

In summary, this study confirms that environmental factors play a highly active and aggressive role in the pathophysiology of allergic rhinitis (AR). It is recommended that ecological risk management be elevated to a core therapeutic pillar, equal in importance to drug therapy and allergen avoidance. Educating patients about environmental risks and guiding them in adopting effective protective measures should become a key component in controlling AR, particularly severe and refractory cases.

6.4. Mediating Role of Harsh Environment

The mediating-effect analysis in this study reveals a deeper, pathological dynamic model than simple superposition. HE acts as a key Pathological Catalytic Mediator between URTI, AH, and AR. This finding provides a robust statistical and pathophysiological foundation for clinically observed complex interactions, systematically explaining why specific physical conditions disproportionately amplify the harm of environmental factors.

The study found that URTI compromises the nasal barrier, creating preconditions for harsh environment invasion and catalytic action. Viral or bacterial infections directly cause epithelial cell damage, ciliary dysfunction, and altered mucus composition. This synergistic effect not only exacerbates the inflammation caused by the disease but also drastically lowers the threshold for triggering allergic reactions. This triggers latent AR or causes existing AR symptoms to deteriorate exponentially.

Allergic rhinitis creates a high-concentration inflammatory zone by forming physical barriers, thereby amplifying the effects of harsh environmental factors. This significantly prolongs the contact time between harmful substances and the nasal mucosa while elevating their local concentration [65]. Consequently, even under moderate external pollution levels, the microenvironment within the nasal passages of AH patients may already be severely polluted. Within this high concentration of inflammatory substances, the mediating role of environmental pollutants reaches its peak, continuously and intensely stimulating the immune system. This leads to AR reactions that are exceptionally stubborn and severe.

When infection coexists with structural abnormalities, environmental factors can trigger a catastrophic pathological resonance. This reveals the mechanism behind the sudden onset of rhinitis following infection, compounded by pollution. Such a scenario represents a complete collapse of the nasal defense system. The immune system must respond to assaults from diverse agents, viruses, bacteria, physical irritants, chemicals, allergens, a process that destabilizes regulatory mechanisms. This extreme scenario explains why patients experience sudden, severe AR flare-ups resistant to conventional medications. At this juncture, the environment ceases to be merely a mediator; it becomes the final straw that breaks the camel's back, the ultimate trigger igniting the entire pathological network.

Thus, the conclusions of this study transcend the conventional understanding that treats URTI, AH, and environmental factors as independent risk factors for AR.

7. Research Contributions, Impact, and Limitations

7.1. Research Contributions

The core contribution of this study lies in its fundamental innovation in research perspective and in the interdisciplinary integration of methodologies, offering a novel dimension for understanding AR as a complex, chronic disease.

Theoretically, this study pioneers the use of patients' subjective experiences and perceptions as core data sources for exploring pathological mechanisms. Traditional clinical research often relies on objective physiological indicators while overlooking patients' complex, real-world feelings and interactive experiences. Through systematic investigation and analysis, this study confirms that patients' intuitive feelings are genuine reflections of universal, verifiable pathophysiological patterns.

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This perspective not only lends rigorous scientific value to patient narratives but also bridges the gap between macro-level clinical data and individual disease experiences. It significantly enhances the ecological validity and clinical translation potential of research conclusions. Crucially, it conveys a vital message to the clinical community: patient perception is indispensable, evidence-based grounding for diagnostic and treatment decisions.

Methodologically, this study employed innovative sociological methods to analyze complex clinical interactions. Confronted with the dynamic interactions among three significant factors, adenoid hypertrophy, upper respiratory tract infection, and a harsh environment, traditional single-variable or laboratory studies struggle to capture their synergistic effects fully. The sociological approach adopted in this study effectively captures and quantifies the synergistic dynamic impact among these three factors within authentic, non-interventional living environments. This ultimately led to the successful construction of a pathological catalytic mediation model for Harsh Environment. This achievement not only provides a highly explanatory theoretical framework for the current research topic but also pioneers an efficient, low-cost, and reality-based research pathway for addressing similar multifactorial, cross-disciplinary complex medical issues in the future.

7.2. Research Impact

The profound impact of this study lies in providing clear, actionable scientific guidance for public AR prevention while offering a powerful personal health incentive to enhance nationwide environmental health awareness.

This study intuitively reveals the intrinsic logic of multifactorial interactions in AR, thereby reshaping traditional prevention concepts. Previously, public AR prevention efforts primarily focused on avoiding single allergens. The findings clearly indicate that for many patients, timely treatment of URTI and effective management of AH are equally crucial as controlling environmental exposures. This discovery offers residents a more comprehensive prevention strategy: on days with poor air quality, they should not only implement physical protection but also vigilantly treat respiratory infections, such as colds, to prevent them from becoming primary triggers of AR. For households with AH, this study further establishes environmental management as central, guiding patients to view avoiding ecological pollution as a key intervention for controlling AR symptoms and preventing disease progression.

This study provides robust, critical evidence to advance environmental protection and promote healthy lifestyles. By scientifically demonstrating HE's mediating role in the development of AR, it directly and closely links macro-level ecological protection issues with micro-level individual health and well-being. It conveys a clear message to the public: caring for the environment is not a distant slogan but a concrete practice for safeguarding respiratory health for oneself and one's family. When residents recognize that their community's air quality directly determines the severity of their respiratory symptoms, this awareness will significantly boost their intrinsic motivation to support and participate in environmental protection actions. Therefore, the findings of this study serve not only as clinical guidelines but also as effective public health education materials. They translate abstract environmental responsibility into concrete health self-defense, providing a valuable scientific basis and a strong foundation for social mobilization to build healthy communities and promote green development.

7.3. Research Limitations

This study offers valuable insights through its perspective and methodology, yet objective constraints have resulted in certain limitations that also point to directions for future research.

Regarding sample selection, the study's sample size remains relatively limited, primarily due to resource constraints. While the current sample adequately supports the construction and validation of the core model, sample size directly impacts the external validity and generalizability of research conclusions. A larger sample size would better reflect universal patterns across diverse populations and improve the reliability of statistical results. Therefore, future research urgently requires expanding sample coverage to include participants from more varied geographic regions, age groups, and

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socioeconomic backgrounds. This would test the stability and broad applicability of the findings, thereby strengthening the persuasiveness of the pathological catalysis mediation model.

Regarding variable control, this study focused on the interactions among three core variables and did not comprehensively control for other potential confounding factors. For instance, individual genetic susceptibility, the family microenvironment, specific dietary habits, and other coexisting health conditions may all influence the final manifestation of AR. These factors were not incorporated into the model. Future research should design more sophisticated study protocols that include a broader range of influencing factors as control variables or covariates in the analysis. This approach would enable more precise isolation of the independent and interactive effects of each variable within the model, thereby enhancing the scientific rigor of the research and the reliability of its conclusions.

Transparency:

The authors confirm that the manuscript is an honest, accurate, and transparent account of the study; that no vital features of the study have been omitted; and that any discrepancies from the study as planned have been explained. This study followed all ethical practices during writing.

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