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Allergic rhinitis: the joint burden of indoor and outdoor environmental exposures

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Abstract. Allergic Rhinitis (AR) is a highly prevalent global upper-respiratory chronic disease that seriously impairs patients' quality of life and brings heavy public-health burdens. Its rising prevalence cannot be fully explained by stable genetic factors, implying environmental factors play a critical role. Nevertheless, most existing studies focus on single-factor effects, while evidence regarding combined and interactive impacts of multi-source environmental exposures remains insufficient. This narrative review synthesize indoor, outdoor and social-living environmental risk factors for allergic rhinitis, covering indoor biological allergens, indoor chemical pollutants, micro-environmental conditions, air pollution, pollen allergens, climate factors and factors related to social life. It concludes that the increased incidence of AR arises from synergistic interactions among multiple environmental risk factors. Limitations include the lack of original empirical data. Further multi-factor studies and targeted environmental interventions are required for AR prevention and control.

Keywords: allergic rhinitis, environmental factors, pathogenesis, allergens

1. Introduction

Allergic Rhinitis (AR), a non-communicable disease, is one of the most prevalent upper respiratory diseases worldwide. Its prevalence has reached approximately 10-30% of adult populations [1]. However, the true incidence of AR is likely higher, as undiagnosed individuals are not captured in routine surveillance data. Allergic rhinitis is characterized by rhinorrhea, nasal congestion, sneezing, itching, and even headache or facial pain. It affects millions of people's life quality, particularly sleep quality, productivity, and mental conditions. Hence, allergic rhinitis has become a global chronic public health issue of great concern, imposing a heavy burden on families and the public health system of society [2].

Research consistently shows that the onset of allergic rhinitis results from a combination of genetic susceptibility and external environmental factors [3]. In recent decades, the prevalence of allergic rhinitis has increased continuously. Nevertheless, genetic factors, which remain relatively stable, fail to account for the rapidly prevalence trend of allergic rhinitis. Thus, environmental factors should be regarded as key drivers behind this disease.

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Despite the large amount of existing research on allergic rhinitis and environmental factors, there is limited research focusing on the pathogenic effects resulting from the combined and interactive actions of multiple environmental factors. Additionally, most existing studies lack a systematic, holistic framework to support targeted environmental interventions and disease prevention. This creates a clear research gap that requires further investigation.

Therefore, this review systematically summarizes recent studies regarding indoor and outdoor environmental risk factors for allergic rhinitis, discusses current research limitations, and provides future perspectives for long-term good control.

2. Indoor environmental factors

The indoor environment is the living scene where people spend the longest time daily. Compared with the outdoor environment, the cumulative concentration of various pollutants and allergens indoors is higher, and the exposure is more stable and persistent. It is the core environmental setting that triggers and aggravates allergic rhinitis.

2.1. Indoor biological allergens

Indoor biological allergens are natural triggers of allergic rhinitis. The pathogenic association of house dust mites, molds, and pet dander with the disease has been confirmed by multiple studies.

House Dust Mite (HDM) is the leading allergen for Allergic Rhinitis (AR). HDM is widely present in home fabrics such as mattresses, sofas, and carpets. A warm and humid indoor environment significantly increases the reproduction rate and concentration of dust mites. In the study done by Zheng et al. [4], they concluded that the expression of CMPK2, which is increased by dust mite stimulation, STING and the NLRP3 inflammasome, was significantly higher in the nasal mucosa of AR patients compared with non-AR controls. Hence, this study suggests that long-term exposure to high concentrations of dust mites is a risk factor for persistent allergic rhinitis in both children and adults.

Mold is another indoor biological factor that is associated with allergic rhinitis. Issues like high indoor humidity and wall dampness easily induce mold reproduction. An analysis, based on thirty-one prior studies on rhinitis and allergic rhinitis, suggests that mold odor, visible mold, and exposure to dampness are all positively correlated with an increased overall risk of rhinitis [5]. Mold odor could continuously stimulate nasal mucosa through respiration, triggering chronic inflammation in susceptible people and causing rhinitis symptoms like nasal congestion and runny nose. The study also indicates that prevention of indoor dampness and mold can reduce the possibility of rhinitis.

Moreover, allergens produced by domestic pets have emerged as significant risk factors for allergic rhinitis worldwide. Pet allergens are protein molecules synthesized by glands and livers. These small molecules are primarily found on pet fur, on pet skin, and in their sweat. However, these allergens could also adhere to dust particles andremain airborne, substantially increasing the risk of inhalation exposure. Recent studies have identified effective interventions for allergic rhinitis caused by pet allergens. Pet removal is regarded as a direct strategy to prevent human exposure to these allergens. Another strategy is gene editing of pets, eliminating allergens [6]. At present, this strategy is still in the early research and development stage and has not been widely applied in practice.

2.2. Indoor chemical pollutants

With the widespread adoption of modern home decoration, indoor chemical pollution has gradually become a research focus regarding its role in causing allergic rhinitis. Indoor chemical pollutants mainly include formaldehyde and other volatile organic compounds generated during indoor renovation, as well as tobacco smoke and kitchen fumes from daily life. Although these pollutants are not traditional allergens, they can indirectly trigger or exacerbate allergic rhinitis by damaging the nasal mucosal epithelial barrier and weakening the nasal immune defense.

New home renovations, inferior furniture, and enclosed ventilation environments are the main causes of excessive indoor volatile pollutants. Multiple studies have found an adverse association between long-term exposure to formaldehyde and volatile organic compounds and allergic rhinitis [7]. Continuous irritation of respiratory mucosa causes damage to the mucosal barrier. The damage of mucosal barrier will further make external allergens more likely to invade the body and enhancing allergic reactions. For genetically susceptible individuals, long-term exposure to renovation pollution can significantly increase the incidence of allergic rhinitis.

Beyond the chemical pollutants stemming from renovation, the pathogenic effects of tobacco smoke are equally significant. Based on recent research, environmental tobacco smoke exposure does not actually raise the probability of allergic sensitization [8]. Evidence suggests no association between smoking exposure and mean IgE levels, an immunoglobulin responsible for triggering allergic reactions in humans, and this relationship may even be inversely correlated [9]. However, it does increase the incidence of rhinitis symptoms.

2.3. Indoor micro-environmental conditions

Although indoor temperature, humidity, and ventilation conditions are all micro-environmental factors that are not direct etiological factors for allergic rhinitis, they indirectly affect the occurrence and development of allergic rhinitis by regulating the reproduction of indoor allergens and altering the functional status of the nasal mucosa.

New research has confirmed that indoor temperature and humidity are crucial factors. As stated above, high indoor temperature and humidity provide ideal conditions for the reproduction of microorganisms such as dust mites and molds, thereby increasing the concentration of biological allergens. On the other hand, too low humidity can cause the nasal mucosa to become dry and the secretion function to decline, weakening the defense ability of the nasal mucosa and making the body more susceptible to allergic reactions.

Ventilation conditions also constitute a key factor in regulating the indoor environment. A long-term sealed living space may lead to the accumulation of biological allergens and chemical pollutants, since they cannot disperse effectively. High concentrations of biological allergens and pollutants will substantially increase human exposure levels. Conversely, adequate ventilation can dilute pollutant concentrations and lower the rate of microorganism proliferation. This represents one effective intervention method that reduces the risk of allergic rhinitis.

3. Outdoor environmental factors

Outdoor environmental exposure is an important external factor that induces allergic rhinitis. In recent years, the acceleration of urbanization, the intensification of air pollution and global climate change have made the pathogenic impact of outdoor environmental risk factors on allergic rhinitis increasingly prominent. Current

research mainly focuses on three aspects: atmospheric air pollution, airborne pollen exposure and meteorological and climatic factors.

3.1. Atmospheric particulate matter and air pollution

Air pollution is defined as the presence of noxious substances in the air at levels that impose a health hazard. Fine particulate matter (PM2.5), inhalable particulate matter (PM10), ozone, nitrogen dioxide and other pollutants in the air are the core outdoor risk factors that induce and exacerbate allergic rhinitis. Numerous epidemiological studies have confirmed a significant positive correlation between the concentration of air pollutants and the incidence of allergic rhinitis in the population. The higher the concentration of pollutants, the higher the risk of developing allergic rhinitis.

Among them, Diesel Exhaust Particulate matter (DEP) includes PM2.5 and PM10, along with various Traffic Related Air Pollutants (TRAP), which are key types of pollutants that affect nasal allergic immune responses. This type of pollutant can enhance the human body's Th2 targeted immune response and significantly increase the probability of sensitization to air allergens. Related mechanism studies have shown that pre-exposure of the human body to Diesel Exhaust Particulate matter (DEP) is an important prerequisite to produce antigen-specific IgE in the nasal mucosa. When the human body encounters allergens and DEP simultaneously, it will further stimulate the secretion of specific IgE, ultimately inducing allergic rhinitis [10].

Multiple controlled experiments have also fully verified the association and pathogenic mechanism between air pollution and allergic rhinitis. Experimental studies show that compared with exposure to allergens or air pollutants alone, concurrent co-exposure to both significantly exacerbates the clinical symptoms of allergic rhinitis, triggers specific changes in the allergic immune response, and further amplifies nasal allergic inflammation [1].

In addition, the degree of exposure to air pollution in the living environment of the population also directly affects the risk of developing allergic rhinitis. Related research has found that children living within 75 meters of urban main roads have a significantly increased risk of lifelong allergic rhinitis. At the same time, the straight-line distance between residential areas and main roads, the total length of surrounding main roads, and the proportion of land occupied by main roads are closely related to the risk of allergic rhinitis in children, further confirming that air pollution from transportation sources is an important risk factor for allergic rhinitis [10].

3.2. Airborne pollen allergens

Airborne pollen is a central driver of seasonal allergic rhinitis. Pollen concentration, vegetation type, and pollen season duration directly determine the incidence pattern of seasonal rhinitis. In recent years, against the background of global climate change, the rise in temperature has indirectly increased the risk of allergic rhinitis. Recent research has suggested that the risk increase in allergic rhinitis induced by heat is only observed under high-pollen conditions. The pathogenic effect of high temperatures is only observable under these specific environmental conditions. Joint exposure to high temperatures and pollen has shown a strong positive correlation with allergic rhinitis [11].

The differences in vegetation types in different regions can lead to regional differences in pollen sensitization characteristics. Herbal pollen and tree pollen are the main types of allergenic pollen in the northern and southern regions of China. Exposure to high concentrations of airborne pollen not only directly induces typical allergic symptoms such as nasal pruritus, sneezing, and rhinorrhea, but also interacts with

atmospheric pollutants to form composite allergenic particles, further augmenting the degree of sensitization and exacerbating patients' clinical symptoms.

3.3. Climate and meteorological factors

Meteorological factors such as temperature fluctuations, precipitation, and wind force are important triggers for allergic rhinitis. Research has shown that sudden drops in temperature and excessive temperature differences between day and night can cause constriction of nasal mucosal blood vessels and decreased defense function, reducing the body's tolerance to external allergens and easily triggering acute attacks of rhinitis. Cold dry air stimulation is an important meteorological trigger for the high incidence and frequency of allergic rhinitis in spring and autumn seasons.

Meanwhile, wind and precipitation can indirectly affect the diffusion and aggregation of pollen and particulate matter in the air. Thunderstorms and heavy rainfall break down pollen particles into tiny inhalable sub-particles [12]. In recent years, extreme weather has occurred frequently, and meteorological conditions have fluctuated dramatically, further exacerbating the instability of allergic rhinitis and increasing the difficulty of disease prevention and control.

4. Confounding factors related to social life and living environment

In addition to typical physical, chemical, and biological environmental risk factors, broader contextual factors such as urbanization, living conditions, and lifestyle also exert a significant impact on the incidence of allergic rhinitis, representing an important addition to research on the environmental etiology of the disease in recent years. The differences in living environment between urban and rural areas, the degree of overcrowding, outdoor activity duration, and hygiene conditions can all affect the occurrence of allergic diseases by altering the human immune exposure environment.

The hygiene hypothesis represents a foundational framework for accounting for the rising burden of allergic diseases in contemporary populations. Recent work has further expanded and refined the original formulation of the hypothesis. Moderate exposure to microorganisms during childhood can effectively promote the development of the immune system and establish a good immune tolerance mechanism [13], However, excessive cleaning, insufficient exposure to microorganisms, dirty living environments, and accumulation of harmful microorganisms can all cause immune developmental disorders, disrupt immune balance, and significantly increase the risk of allergic rhinitis.

The environmental changes brought about by urbanization, such as residential confinement, reduced outdoor activities, and changes in urban greening structure, can also reduce the level of exposure to the natural environment of the human body, weaken immune adaptability, and are important social environmental factors contributing to the continuous increase in the incidence of allergic rhinitis in contemporary populations. The problems of living conditions such as overcrowding, low floors, and insufficient lighting and ventilation can combine various environmental risk factors to further increase the probability of disease occurrence.

5. Conclusion

This review systematically sorts out environmental risk factors associated with allergic rhinitis from indoor, outdoor and social-living dimensions. The high incidence and prevalence of allergic rhinitis are the result of multiple environmental risk factors working together. Within the indoor environment, biological allergens, chemical pollutants, and microenvironmental imbalances act together to trigger and aggravate the disease.

House dust mites, mold and pet dander are confirmed biological triggers. Formaldehyde, volatile organic compounds and tobacco smoke damage the nasal mucosal barrier and weaken immune defense, while indoor temperature, humidity and ventilation indirectly regulate allergen concentration and nasal mucosal state. For outdoor exposure, air pollution, airborne pollen, meteorological and climate changes jointly induce or worsen rhinitis symptoms. Fine particulate matter, diesel exhaust particles and traffic-related pollutants can enhance Th2 immune responses and promote specific IgE production. Pollen concentration, vegetation type and pollen season duration determine the seasonal pattern of rhinitis And sudden temperature drops, cold dry air and thunderstorm events further destabilize disease attacks. Moreover, changes in living environments related to urbanization, such as residential confinement, reduced outdoor exposure and overcrowded housing, interfere with immune development and tolerance under the extended hygiene hypothesis. Taken together, biological allergens, chemical pollutants, and microenvironmental imbalances in indoor environments, as well as air pollution, airborne pollen, meteorological and climate changes in outdoor environments, and changes in living environments related to urbanization, are all core risk factors for inducing and exacerbating allergic rhinitis. The overlapping and synergistic effects of various environmental factors are the key reasons for the increasing incidence and younger development of allergic rhinitis in recent years.

Nevertheless, several limitations of the present review should be acknowledged. This paper is a narrative literature review and does not adopt original empirical or experimental research. All conclusions are drawn from previously published studies. In addition, the number and scope of included literature are limited, and no quantitative meta-analysis has been performed to pool effect sizes, so the exact magnitude of each environmental risk factor cannot be quantitatively estimated. In view of these drawbacks, future research should break through the limitations of single factor studies and focus on exploring the synergistic pathogenic mechanisms of multiple environmental factors combined exposure. Large sample, long-term dynamic cohort studies should be conducted to clarify the causal and dose-response relationships between environmental exposure and disease occurrence. At the clinical and public health prevention and control level, attention should be paid to environmental intervention measures such as home environment management, air quality improvement, and lifestyle optimization. By accurately regulating environmental risk factors, early prevention and disease control of allergic rhinitis can be achieved, providing new theoretical basis and practical ideas for the public health prevention and control of this disease.

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