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Abstract

The House dust mites (DPMs) are a major source of indoor aeroallergens and play a role in various allergic diseases, particularly allergic rhinitis, asthma, and atopic dermatitis. In Indonesia, sensitization to DPMs is common in children, and several studies in orphanages and urban areas have shown high levels of DPMs in mattresses and sleeping areas. Although the relationship between DPM density and disease incidence is inconsistent across studies, this article aims to review immune response to *Dermatophagoides* through a literature review. A search was conducted on scientific publications from 2022–2026 using Google Scholar with the keywords "immune response," "host and mite," and "*Dermatophagoides*," then selected and analyzed qualitatively. The review results indicate that the DPM life cycle is influenced by temperature and relative humidity >60%. Immunologically, TDR allergens—particularly Der p 1—have protease activity that disrupts the epithelial barrier, activates the innate immune response (alarmin IL-33, TSLP, IL-25) and induces a Th2 response dominance, increased IgE, mast cell activation, and the release of inflammatory mediators. This mechanism triggers type hypersensitivity reactions in allergic rhinitis and asthma (early and late phases), and exacerbates skin barrier dysfunction and chronic pruritus in atopic dermatitis through cytokine and neurosensory pathways.

INTRODUCTION

The House dust mites (*Dermatophagoides spp*) are a major source of indoor aeroallergens. Some allergic diseases associated with DPMs include allergic rhinitis, asthma, and eczema or atopic dermatitis (Aggarwal, 2023). Most house dust mite sufferers in Indonesia are school-aged children. House dust mite allergens are the main cause of DPMs in Indonesian children (Endaryanto, 2021). A study by Yolazenia et al. on house dust mite density as a trigger for allergic rhinitis in children at a Pekanbaru orphanage revealed that 81.2% of children experienced allergic rhinitis symptoms and found DPMs with an average density of 9.11/g of dust. The study found DPMs in all four orphanages studied, which had a high incidence of allergic rhinitis (Yolazenia, 2019). Meanwhile, another study by Nova et al. found no difference between DPM density and DPM incidence in two orphanages in Koto Tangah District (Nova, 2018).

A study states that indoor allergens produced by house dust mites, mold, and cats are triggers for asthma in childhood and adulthood, especially in people who are sensitive to these allergens (Dharmage, 2019). In a study, Pangadongan et al. (2017) concluded that there is a relationship between house dust mites and the incidence of bronchial asthma in children in the Lempake Community Health Center work area, Samarinda City. Meanwhile, Armunanto (2021) revealed no relationship between house dust mite density and the incidence of asthma in

pediatric patients in Semarang. A retrospective study by Nugroho et al. (2022) found that 63.3% of 60 atopic dermatitis patients had sensitization to DPM. Another study in Brunei conducted by Emran et al. (2019) showed a relationship between house dust mites and the incidence of atopic dermatitis.

Through a study in Pamulang (Tangerang) and Pasar Rebo (Jakarta), Subahar et al. (2016) found that 94.2% of respondents' homes tested positive for DPM and 63.6% in Pasar Rebo. The study also revealed that mattresses and bed floors are risk factors for HDM, and houses in these urban areas are often found with DPM. Based on previous research on environmental factors that play a role in houses found with dust mites in Manado City, Ikbal et al. (2015) concluded that the description of houses found with TDR involves the role of factors such as the use of mattresses, sofas, floors, pets, sheets, temperature, and humidity. The study also revealed that along with the increase in the number of room occupants, it is possible that the mite population in the room increases. This can be found in orphanages because children sleep together in one room (Yolazenia, 2019).

A study in China found that absolute humidity can be determined by subtracting indoor humidity from outdoor humidity. The study also found that reducing absolute humidity through increased air exchange rates is a likely cause of the high ventilation rate's reduction in house dust mite allergen concentrations (Sun, 2022). However, some article has explored the humoral and cellular mechanisms involved in *Dermatophagoides*. Therefore, the purpose of this article is to review the host immune response to house dust mites.

RESEARCH METHODS

This research is a literature review aimed at assessing and interpreting all findings related to the host immune response to house dust mites (HDM). This research was conducted by searching scientific publications from 2022 to 2026 through the Google Scholar database. The keywords used in the search were "immune response," "host and mite," and "Dermatophagoides." After going through a data filtering and extraction process, this study used qualitative data analysis techniques to combine the data that met the requirements (Ukratalo, 2023; Tuhumury & Ukratalo, 2023; Tofure et al., 2025)

Inclusion criteria is articles published between Januari 2022 and December 2026, studies investigating the host immune response to HDM, particularly *Dermatophagoides* spp, studies involving human or relevant experimental animal hosts exposed to or sensitized by HDM. Exclusion criteria is articles which focusing exclusively on mite taxonomy, morphology, ecology, environmental distribution or allergen characterization without evaluating the host immune response.

RESULTS AND DISCUSSION

House Dust Mite Life Cycle and Identification

The life cycle of house dust mites depends on temperature and relative humidity above 60%. At 23°C, the life cycles of *D. pteronyssinus* and *D. farinae* last 36 and 34 days, respectively. At this temperature, female house dust mites produce up to four relatively large eggs per day, resulting in a total of between 80 and 300 eggs being laid during the female's lifetime.

Meanwhile, at temperatures between 35°C and 16°C, *D. pteronyssinus* require 15 to 23 days to fully develop. *D. farinae*, on the other hand, cannot develop well at temperatures between 35°C and 16°C (Bergmann, 2022).

Males can only successfully mate with mature females. After mating, the females lay eggs (approximately 80 × 170 µm) singly in the soil. The TDR has six developmental stages, each separated by a molt: egg, prelarva, larva, protonymphs, deutonymphs, and tritonymphs (juvenile stages), and adulti (adult males/females). Eggs contain the prelarval stage. The hatched larvae and nymphs (young stages) have active and resting developmental phases. The transition to the next developmental stage is characterized by a resting phase of 2-3 days each, after which the skin is molted to the next developmental stage. The life cycle from egg to adult is approximately one month, and adult mites can live another one to three months. The average life cycle of house dust mites is 65–100 days. One study found that males of *D. pteronyssinus* live between 60 and 100 days, females up to 150 days, and *D. farinae* only about 60 days (Sarwar, 2020).

The presence of house dust mites can be confirmed microscopically, requiring the collection of samples from mattresses, sofas, or carpets. Furthermore, in general practice, a minimum of 10x magnification is required for proper identification. One study used a vacuum cleaner to collect dust samples. The dust samples were then placed in prepared petri dishes and labeled with the room number and name of the room where the dust originated. The study detected the presence of mites by placing a quantity of dust on the surface of water and examining it under 20x magnification to observe the mites floating on the surface (Vatandoost, 2022).

Immune Responses Triggering Allergic Rhinitis

The pathophysiology of AR associated with TDR involves TDR protease activity, which leads to excessive IgE production. This is caused by the activity of the protease Der p 1 in TDR, which can directly disrupt the epithelial barrier, or by other allergens, such as Der p 2 in TDR, which can activate pattern recognition receptors (PRRs). The nasal epithelium can then initiate an innate immune response through the release of alarmins such as IL-33, thymic stromal lymphopoietin (TSLP), or IL-25. Ultimately, alarmins can activate group 2 innate lymphoid cells (ILC2s), which immediately produce type 2 cytokines (IL-5, IL-13, and IL-4) in situ. This then initiates an adaptive immune response, resulting in IgE class switching and mucosal inflammation. Thus, in AR patients sensitized to TDR, Der p 1 disrupts the IgE feedback mechanism through selective cleavage of CD23, resulting in excessive IgE production by B cells (Nur, 2022).

Type I hypersensitivity reactions occur rapidly, typically within 20 minutes of allergen exposure. This reaction involves activation of mast cells and inflammatory cells, leading to tissue infiltration. Naive CD4+ T cells then differentiate, transforming into allergen-specific Th2 cells. Th2 cells release cytokines such as IL-4 and IL-13, which are then activated and interact with B cells to produce allergen-specific IgE. This allergen-specific IgE binds to high-affinity FcεRI receptors on mast cells, activating them. These mediators induce mucosal edema and watery rhinorrhea through vascular leakage. Histamine, the primary mediator of AR, activates H1 receptors on sensory nerve endings, resulting in sneezing, pruritus, and a reflex secretory

response. It also interacts with H1 and H2 receptors in mucosal blood vessels, leading to nasal congestion due to vascular engorgement and plasma leakage (Bousquet, 2020).

After 4-6 hours of allergen exposure, the final phase of the allergic response begins. Activation of structural cells in the nasal mucosa, such as epithelial cells and fibroblasts, can induce the release of additional chemokines (e.g., eotaxin, RANTES, and TARC), which facilitate the influx of cells from the peripheral blood. This inflammatory cell recruitment is triggered by cytokines such as IL-4 and IL-5, which upregulate the expression of adhesion molecules, such as vascular cell adhesion molecule 1 (VCAM-1), on endothelial cells, facilitating inflammatory cell influx (Nur, 2022).

Immune Responses That Trigger Asthma

One significant factor influencing asthma control is exposure to environmental allergens, particularly indoor allergens. Various studies indicate that allergens such as house dust mites, animal dander, cockroaches, rodents, and molds play a significant role in triggering asthma symptoms and exacerbations, especially in sensitized individuals. The home environment represents the setting where individuals experience the longest duration of exposure, thereby contributing substantially to the course of the disease. Exposure to allergens in the home environment is known to be associated with increased asthma symptoms, a higher frequency of exacerbations, and reduced levels of disease control (Trasia, 2026).

The pathophysiology of asthma associated with DPM involves mucosal immune cells, molecules, and pathways targeted by house dust mite proteins to trigger allergic asthma. The primary TDR allergen is Der p 1, a cysteine protease. Not only is it an antigen, Der p 1 also contributes to the development of allergic asthma through its effects on a number of immune cells and molecules. Therefore, Der p 1 can cleave epithelial cell tight junction proteins and access immune cells, including naive dendritic cells. Der p 1 can also induce activation of protease-sensitive receptors (PARs) and TLR4, or cause injury to epithelial cells (Abu, 2020).

This, in turn, leads to activation of the NLRP3 inflammasome and the secretion of cytokines and chemokines that recruit myeloid cells, including dendritic cells, eosinophils, and ILC2s. Cleavage of CD40 limits the production of IL-12 and IFN γ , thus promoting Th2 cell polarization. Through cleavage of the CD23 receptor, Der p 1 increases IgE production, which in turn disrupts the negative feedback loop regulating IgE production. Furthermore, Der p 1-specific IgE cross-linking to mast cells by Der p 1 mediates mast cell degranulation and the release of histamine and other mediators associated with allergic asthma symptoms (Saglan, 2019).

The two phases of asthma exacerbation in the pathophysiological mechanism of asthma are the early and late phases. The early phase begins with plasma cells releasing sensitized IgE antibodies. These antibodies respond to specific triggers from environmental risk factors. IgE antibodies then bind with high affinity to mast cells and basophils. When pollutants or substances from risk factors are inhaled, cytokines are released by mast cells, resulting in degranulation. Mast cells release histamine, prostaglandins, and leukotrienes. These cells then contract smooth muscle, narrowing the airways. Th2 lymphocytes play an integral role in producing various interleukins (IL-4, IL-5, IL-13) and GM-CSF, which act as intermediaries with

other cells and cause inflammation. IL-13 plays a role in remodeling, fibrosis, and hyperplasia. Meanwhile, IL-3 and IL-5 are involved in the survival of eosinophils and basophils (Sinyor, 2023).

Immune Responses Triggering Atopic Dermatitis

Airborne allergens can be specific triggers that play a key role in the pathogenesis of atopic dermatitis (AD). For example, AD exacerbations can occur in patients exposed to airborne allergens derived from DPM. Through their enzymatic activity, allergens can damage tight junctions and impair skin barrier function in patients with AD. This disruption of the skin barrier allows proteins from airborne allergens to penetrate the epidermis, reaching allergen-presenting dendritic cells, thereby triggering a systemic Th2 allergic inflammatory response and exacerbating AD severity (Bumbacea, 2020).

The pathogenesis of AD is complex and multifactorial. Genetic factors are involved through deficiencies in genes encoding stratum corneum structural proteins such as filaggrin. Furthermore, upregulation of genes encoding Th2 cytokines, such as IL-4 and IL-13, and polymorphisms in genes encoding their receptors play a role. Overexpression of the Th2 axis results in skin barrier dysfunction with downregulation of key proteins for stratum corneum stability, such as filaggrin, locrin, involucrin, and corneodesmosin. Decreased production of the antimicrobial peptide LL-37 and β -defensins increases the risk of *S. aureus* superinfection (Lobefaro, 2022).

Pruritus contributes significantly to the disease burden of AD, significantly impairing the quality of life of atopic patients by limiting productivity and daily activities. Itch in AD patients occurs through the role of histamine-independent signaling pathways. Skin barrier disruption results in the release of inflammatory cytokines, including CXCL1, which recruits neutrophils. Neutrophils trigger the release of CXCL10- and CXCR3-expressing sensory neurons, which transmit itch signals. Chronic itch is characterized by infiltration of TSLP-receptor-expressing basophils, which release IL-4 and CD4⁺ T cells, which then release IL-4, IL-13, and IL-31. These three interleukins contribute to the development of pruritus, primarily through their involvement in stimulating specific sensory neurons through a JAK-dependent mechanism. IL-31 triggers the elongation of sensory nerve fibers. Binding of IL-31 to its receptor expressed on sensory nerve fibers induces pruritus via TRPV1 and/or TRPA1 channels (Nutten, 2015).

IL-31 then binds to IL-31RA, a combination expressed on keratinocytes, and stimulates the secretion of LTB₄. LTB₄ binds to the BTL1 receptor, which is localized on sensory nerve fibers. Sensory neurons are then activated, driving the sensation of itch and scratching behavior. An update of a study that included the finding that AD inflammation triggers early phenotypic changes in patients with circulating basophils suggests that a specific basophil subset exhibits increased expression of Fc ϵ RI and CD203c. This IgE-R⁺ basophil subset releases LTC₄ upon antigen-specific IgE stimulation. LTC₄ binds to its receptor CysLTR2 expressed on sensory nerve fibers, resulting in the activation of sensory neurons that transmit itch signals that ultimately drive scratching and pruritus behavior during relapses or flare-ups (Fleming, 2020).

Recent Findings on House Dust Mite Prevalence in Indonesia

According to Arsusanti et al. (2025), carpet and mattress dust samples that tested positive for HDMs had a low average density. The growth and development of HDMs are influenced by environmental factors such as temperature, humidity, and high occupancy rates. Furthermore, this study found a significant relationship between hygiene practices, such as sunning mattresses/carpets and washing bed linens, and HDMs density.

Research in Padang City found that all dust samples (100%) tested positive, and identification revealed that the most common HDMs species was *Dermatophagoides sp.*, followed by *Acarus sp.*, *Cheyletus sp.*, *Carpoglyphus sp.*, *Glycyphagus sp.*, *Thyrophagus sp.*, and *Blomia tropicalis*. Most houses' HDMs densities were low, with an average HDMs density of 72.85 mites per gram of dust. The prevalence of allergic rhinitis in children in orphanages is 18.75%, most frequently occurring in the 13-18 age group, with the highest number of cases found in girls. This study showed no significant association between HDMs density and a history of allergic rhinitis. This study concluded that other factors, such as genetic predisposition, environmental conditions, and immune sensitization status, may play a role in the development of allergic rhinitis (Agusman, 2025).

A study by Habibi (2025) found the prevalence of urticaria and/or angioedema was 25.6%, relatively balanced by age group (5 children aged 6–12 years; 5 children aged 13–17 years). Based on the statistical analysis, it was concluded that there was no significant association between DPM exposure and urticaria and/or angioedema in children in Pauh District, Padang City. In this study, it was resumed that there are other factors such as genetic predisposition, environmental conditions, and immune sensitization status, which may play a role in the development of urticaria and/or angioedema.

CONCLUSION

DPM particularly *Dermatophagoides pteronyssinus* and *Dermatophagoides farinae*, have a life cycle strongly influenced by temperature and humidity, with a high reproductive capacity that allows their populations to grow rapidly in domestic environments. The presence of DPMs can be identified through microscopic examination of house dust samples, confirming their role as major environmental allergens. Pathophysiologically, DPM allergens—particularly Der p 1—have protease activity capable of disrupting the epithelial barrier and activating both innate and adaptive immune responses. This activation triggers a dominant Th2-type immune response, increased IgE production, mast cell activation, and the release of inflammatory mediators such as histamine, cytokines, and chemokines. These mechanisms play a key role in the onset and progression of allergic diseases, including allergic rhinitis, allergic asthma, and atopic dermatitis.

In allergic rhinitis and asthma, DPM trigger a type I hypersensitivity reaction with early and late phases characterized by mucosal inflammation, bronchoconstriction, and tissue remodeling. Meanwhile, in atopic dermatitis, DPM exacerbates skin barrier dysfunction, enhances the Th2 inflammatory response, increases the risk of secondary infections, and contributes to chronic pruritus through complex, largely histamine-independent signaling pathways. Thus, DPM plays a central role in the pathogenesis of allergic diseases through a

close interaction between environmental factors, epithelial barrier disruption, and immune response dysregulation. A comprehensive understanding of the DPM life cycle and the immunological mechanisms involved provides an important basis for strategies for prevention, environmental control, and clinical management of DPM-related allergic diseases.

DECLARATIONS

A.A.S contributed to designing the study. R.F.T contributed to preparing and proofreading the manuscript. This research did not receive any funding from public, commercial or any external funding. The authors declare no relevant conflicts of interest related to this study. The data used in this are available upon reasonable request and in accordance with established procedures.

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