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Editorial

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Aerobiology in Asian airway allergic diseases

Bernard Yu-Hor Thong*

Department of Rheumatology, Allergy and Immunology, Tan Tock Seng Hospital, Singapore 308433, Singapore

House dust mite allergy is present in up to 90% of Asian atopic patients, with increasing incidence and prevalence of sensitization and clinical allergy from childhood through to adulthood. This far exceeds the reported prevalence of 50%–70% in Western populations [1]. House dust mite allergy is particularly common in the tropical areas of Southeast Asia due to the warm, humid climate [2]. In contrast, allergy to grass and tree pollen and animal dander affect less than 10% of Asian patients compared to 40%–70% of individuals with asthma and allergic rhinitis living in the West. It is only in certain parts of Asia and Australasia where grass and tree pollen allergy is more prevalent than house dust mite allergy e.g., Japanese cedar, subtropical Bahia grass, and Bermuda grass pollens [3]. Distinct regional differences in aeroallergen sensitization reflects the different urban versus rural, environmental, lifestyle, geographical and climatic differences within the Asia-Pacific region. Time trend changes in the patterns of aeroallergen sensitization from a diverse aerobiology to increasing house dust mite sensitization may also be a result of climate change. Similarly, a study from Seoul and Incheon showed time trend and diversity differences in pollen allergy in 2015 and 2016, where pollen seasons became longer and peak concentrations possibly potentiated by climate change [4].

In 2015, the World Allergy Organization issued a position statement on the potential short and long-term effects of climate change on the prevalence of allergic airway diseases, in particular asthma and rhinitis [5]. Global warming and the increasing concentration of greenhouse gases, especially carbon dioxide; severe and prolonged heat waves, air pollution, forest fires, desert storms, droughts, and floods have the potential to increase the prevalence, severity, morbidity, and mortality from respiratory allergy. Global warming may also affect the start, duration, and intensity of the pollen season; or the rate of asthma exacerbations due to air pollution, respiratory infections, and/or cold air inhalation. Air pollution is associated with decrease in lung function, and increased emergency room visits and hospitalizations in asthma. Occupational exposure to both allergens and nonallergens may also exacerbate asthma [6]. At the cellular level, the influence of environmental air pollutants on bronchial hyperresponsiveness is elegantly demonstrated in a study using a murine asthma model, which showed enhanced oxygen toxicity and antioxidant activity in response to ozone in asthmatic but not normal mice [7]. Such translational research will help improve our understanding of the interaction between environment pollutants and asthma control, and hence the effectiveness of environmental interventions in asthma management, particularly in children [8].

*Correspondence: Bernard Yu-Hor Thong
Department of Rheumatology, Allergy and Immunology, Tan Tock Seng Hospital, 11 Jalan Tan Tock Seng, Singapore 308433, Singapore
Tel: +65-63577-7822
Fax: +65-6357-2686
E-mail: bernard_thong@ttsh.com.sg

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Other than house dust mites, animal dander, grass and tree pollen, fungi may also cause a wide spectrum of fungal diseases of the upper and lower airways, of which *Aspergillus fumigatus* is clinically the most important [9]. Allergic fungal rhinosinusitis causes chronic rhinosinusitis (CRS) symptoms for which surgical intervention and systemic corticosteroids are the recommended treatments. Allergic bronchopulmonary aspergillosis (ABPA) is most commonly diagnosed in patients with asthma or cystic fibrosis, where long term systemic corticosteroids with the addition of an antifungal medication is the mainstay of treatment. Fungal sensitization or exposure increases the risk of developing severe asthma, and this has also been termed severe asthma associated with fungal sensitivity. A series of childhood ABPA not associated with cystic fibrosis from India is reported in this issue, highlighting the need to consider this diagnosis in difficult childhood asthma in order to prevent irreversible lung damage in adulthood [10].

Aerobiology is but one of the many drivers of allergic sensitization in airway allergic disease. The understanding of disease endotypes based on immunological and pathophysiological principles, and their validation across clinically meaningful outcomes is crucial for the success of precision medicine as a new targeted approach to patient management [11]. For instance, in the eosinophilic asthma endotype, treatments targeting immunoglobulin E and the type 2-cytokines interleukin (IL)-4, IL-5, and IL-13 have been shown to improve asthma-related clinical outcomes and/or have steroid-sparing properties. Similarly among east Asians with CRS, subclassification into CRS in the presence/absence of tissue eosinophilia and nasal polyposis may also improve risk-stratification, prediction of pharmacological responses and outcomes [12].

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