VIP/VPAC Axis plays an immunomodulatory role in a murine model of *Aspergillus fumigatus*-induced allergic asthma

Amali E Samarasinghe¹, Scott A Hoselton² and Jane M Schuh²

¹University of Tennessee Health Science Center, USA
²North Dakota State University, USA

Allergic asthma is a disease of the airways that affects over 25 million people in the United States incurring over $50 billion in direct and indirect costs per year. Although we understand the symptoms associated with allergic asthma, the exact causes and sequence of events that follow allergen exposure warrants further investigation. Animal models that effectively recapitulate the hallmarks of human disease are important to delineate the pathways that lead to the immunological, architectural and physiological changes that occur. We have identified that the neuroimmunological axis plays a role in the development of allergic asthma using a novel murine model induced by *Aspergillus fumigatus* conidia. Vasoactive intestinal peptide (VIP) is a neuropeptide with cytokine properties which has been demonstrated to have an anti-inflammatory role in the lungs when acting through its receptor VPAC2. We showed that VIP localization in the columnar epithelium of the airways was dynamically regulated following allergen provocation with levels decreasing early during the allergic cascade and increasing after the influx of lymphocytes into the airways. The VIP/VPAC₂ axis, previously shown to promote T₄₁² immunity, was demonstrated herein not to play a role in the development or maintenance of allergic asthma. VPAC₂ null mice have elevated levels of IgG₂a and IgA in response to allergen challenge, indicating its novel role in regulating the humoral immune response.

Biography

Amali E Samarasinghe completed her PhD in 2010 with a Presidential Fellowship from North Dakota State University during which she developed and characterized a novel murine model of allergic asthma. She moved to St. Jude Children’s Research Hospital for Postdoctoral training in Infectious Diseases wherein she developed a new model to study asthma and influenza comorbidity which has clinical relevance. She moved to University of Tennessee Health Science Center in 2012 as junior faculty and continues to work on the impact of respiratory infections in allergic asthma.

asamaras@uthsc.edu

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