



The Role of Human Rhinovirus Infections in the Pathogenesis of Airway Remodelling in Asthma Diana Pham (University of Calgary)

Supervisor: Dr. Richard Leigh, Professor and Department Head, Department of Medicine

Asthma Canada / AllerGen Goran-Enhorning Graduate Student Research Award

\$10,000 to support investigations into early-onset asthma by an MSc student

Diana Pham is a Master of Science student at the University of Calgary with Dr. Richard Leigh. She is conducting research to expand our knowledge on the molecular mechanisms contributing to the development of asthma. Her findings will help determine an appropriate pharmaceutical target or preventative measure for the airway remodelling that occurs in early childhood asthmatics.

The Role of Human Rhinovirus Infections in the Pathogenesis of Airway Remodelling in Asthma

Asthma is a chronic inflammatory disease that affects the lives of over 3 million Canadians. Shortness of breath, chest tightness, and wheezing are just some of the many symptoms that asthmatics experience on a daily basis. Asthma is such a debilitating disease, yet the mechanisms responsible for its onset are still not fully understood.

This is where Diana Pham, a Master of Science student at the University of Calgary, steps in. This 23-year-old emerging researcher is studying the mechanisms of airway remodelling – a prominent feature in asthma that leads to thickened airway walls and the narrowing of the airway. The phenomenon can present in early childhood before a formal diagnosis of asthma, suggesting that airway remodelling is not hereditary, but rather developed in response to exposures in early childhood.

Pham and her lab are conducting research on the role of human rhinovirus (HRV) infections on airway remodelling in asthma. Her lab has shown that upon infection, the epithelium (the primary site of

infection) releases growth factors strongly involved in the airway remodelling process and induces structural cells, called fibroblasts, to migrate.

Given that fibroblasts migrate in response to HRV infection, Pham questions whether these fibroblasts can now transform into myofibroblasts. Myofibroblasts, another structural cell responsible for airway wall thickening, are highly increased in asthmatic airways, yet their origin is unclear. Her research project focuses on fibroblast-to-myofibroblast transformation by which she will expose fibroblasts to growth factors released from HRV-infected epithelial cells and see if they express myofibroblast characteristics.

These new findings from Pham will expand our knowledge on the mechanisms contributing to the development of asthma. She hopes her research will help determine an appropriate pharmaceutical target or preventative measure that can be used to mediate the airway remodelling process in childhood asthmatics.

About Diana Pham

Diana Pham's interest in the field of asthma first stemmed from working at the Ottawa Allergy Research Corporation as a research assistant looking at omalizumab, a biologic therapy for asthma. She investigated the effects of omalizumab in patients with various diseases such as chronic spontaneous urticaria, pediatric asthma, cystic fibrosis, and allergic bronchopulmonary aspergillosis.

Pham's preliminary data was accepted at the 2017 Canadian Society of Allergy and Clinical Immunology (CSACI) Conference in Toronto, ON, for a poster presentation with Pham as first author. Since the start of her graduate studies, she has also been awarded The Lung Association – Alberta and NWT Studentship Award, ranking first overall.

After completing her master's degree, Pham hopes to attend medical school and specialize in allergy and immunology. With her clinical and research background, she aspires to become a principal investigator for clinical trials and labs conducting asthma research.

A Message to the Asthma Community – From: Diana Pham

"I want to tell the asthma community that I empathize with them. There are so many people who care and want to help them. I think it is so great that this community sticks together and wants to know more about what we researchers do. Keep fighting!"