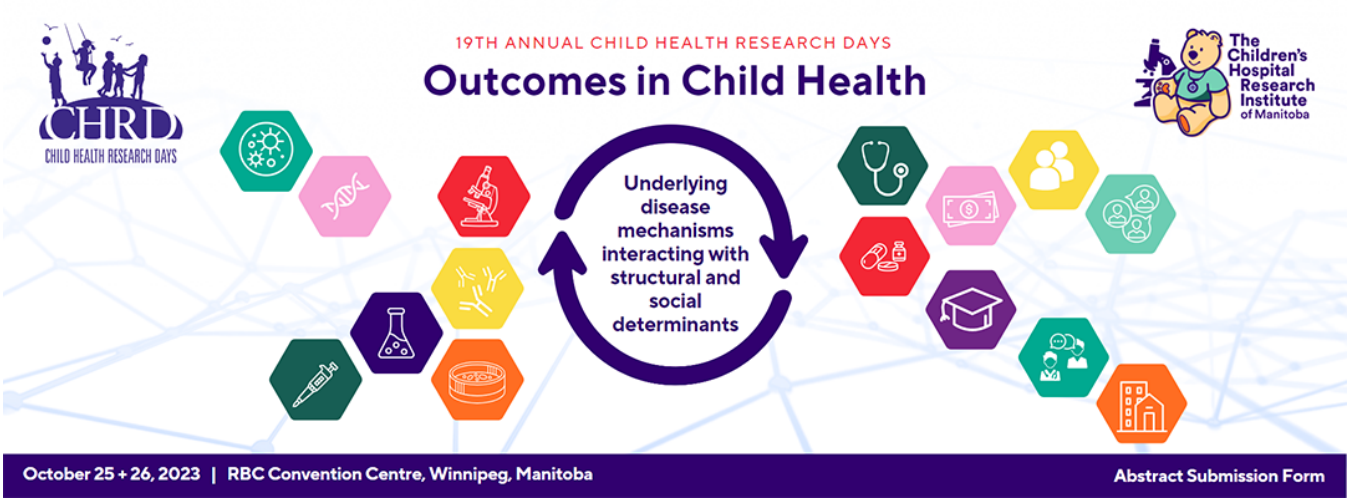

CHRD 2023: Abstract Submission Form

Submitter Name
Sina Taefehshokr

Presenter Name
Sina Taefehshokr

Presenter Status
Masters Student

Research Category
Basic Science

Role in the project
Design
Perform Experiments
Analyze Data
Write Abstract

Title
PTX3 deficiency exacerbates neutrophilic airway inflammation in house dust mite model of severe asthma.

Background
Pentraxin 3 (PTX3) is a member of the family of pattern recognition receptors known as pentraxins. Ptx3 deficiency exacerbates airway inflammation and AHR in an OVA mouse model of allergic inflammation. These events are due to an enhanced ability of pulmonary DC to produce IL-6 and IL-23, and thus the excessive secretion of IL-17A by lung CD4+T cells (Balhara J. et al., JACI, 2017, Editor Choice, Balhara J et al., Frontieres in Immunology, 2021). Furthermore, we showed that human airway smooth muscle cells as the primary source of PTX3 in vitro and ex vivo, and can inhibit ASM cell migration, suggesting a putative role of this protein in airway remodeling.

Objective
To determine the role of PTX3 in airway inflammation, remodelling and hyper-responsiveness in severe chronic asthma.

Methods
PTX3 knockout (KO) and wildtype mice were subjected to HDM chronic allergen protocol. Lung tissue and BALF were studied with flow cytometry. In addition, cytokines and serum immunoglobulins will be assessed using mesoscale and ELISA, respectively. Airway hyper-responsiveness (AHR) parameters were measured with FlexiVent ventilator. Collagen deposition and mucus production will be visualized by Sirus-red, and Periodic acid-Schiff (PAS) and associated genes will be investigated using real-time PCR.

Results
Our preliminary data showed a significant infiltration of neutrophil cells (CD11b+/Ly6G+) into the airway of PTX3-/- group compared to WT counterparts upon HDM- chronic challenge. Moreover, we had increased airway hyper-responsiveness in PTX3-/- group compared to WT counterparts.

Conclusion
PTX3 deficiency enhances neutrophil dominant inflammation as well as AHR in HDM model of severe chronic asthma.

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