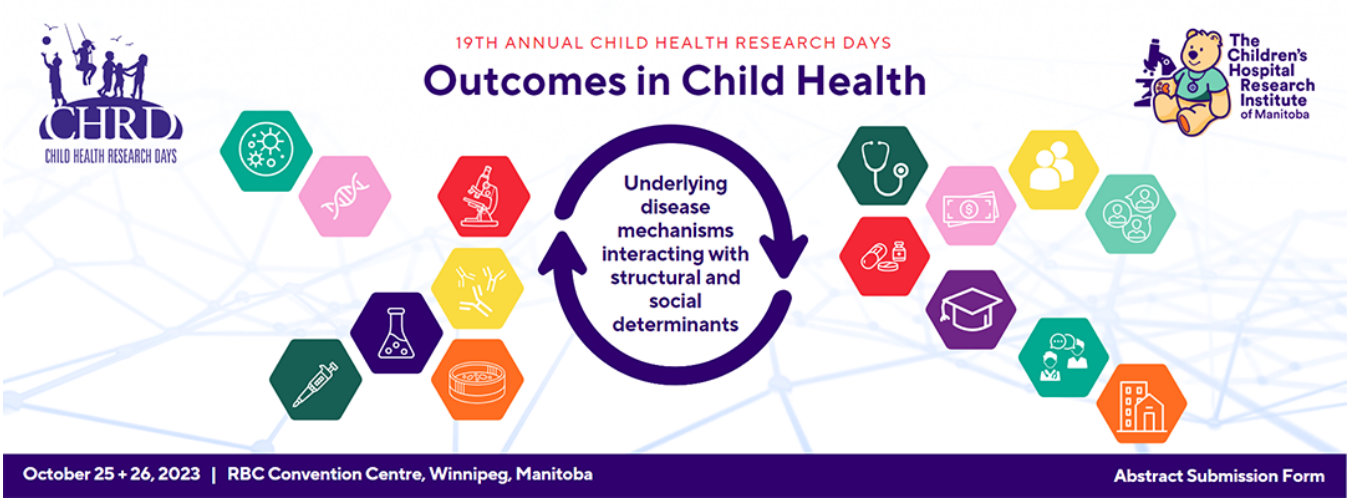

CHRD 2023: Abstract Submission Form

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Presenter Status
Masters Student

Research Category
Basic Science

Role in the project
Design
Perform Experiments
Analyze Data
Write Abstract

Title
SEMA3E IS A NOVEL REGULATOR OF IGE PRODUCTION THROUGH GERMINAL CENTER FORMATION

Background
Semaphorin3E (Sema3E) is a protein, critical in immune diseases, including asthma. Sema3E expression is significantly reduced in the airways of human asthmatics and mouse models of allergic asthma. House Dust Mite (HDM) challenged Sema3E deficient mice showed increased airway inflammation, hyperresponsiveness, and remodeling associated with a significantly higher total and allergen-specific Immunoglobulin E (IgE) level. These results suggest that Sema3E regulates IgE production in airway allergic inflammation.

Objective
Sema3E regulates IgE levels through germinal center (GC) reactions in HDM-induced allergic inflammation.

Methods
Sema3E KO mice and WT were subjected to HDM acute challenge protocol. Flow cytometry was performed to investigate the level of GC B cells, T follicular helper (Tfh), eosinophils, and neutrophils. The antibodies and cytokines levels were detected using ELISA and mesoscale, respectively. GC formation was blocked by intraperitoneal injection of anti-ICOS-L. The control group received rat IgG.

Results

Sema3E-/- mice presented a significant amount of GC B cells, Tfh cells, and IgE+ B cells compared to WT counterparts. Furthermore, compared to WT mice, a substantial reduction in GC B cells, Tfh, IgE+ B cells, and plasmablasts were observed in Sema3E-/- upon the ICOS/ICOS-L neutralization. Similarly, disruption of the ICOS/ICOS-L leads to a decrease in the levels of IgE, IgG, Th2, Th1, and Th17 cytokines. Moreover, inflammatory cells such as eosinophils and neutrophils significantly decreased in the lung and Bronchoalveolar lavage fluid following anti-ICOS-L treatment in Sema3E KO compared to the WT group.

Conclusion
: Sema3E regulates IgE levels through GC formation. Our data also suggest that Sema 3E influences the outcome of allergic inflammatory response in asthma by influencing the GC environment.

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