CHRD 2024: Abstract Submission Form

Presenter Name
Sina Taefehshokr

Presenter Status
Masters Student

Role in the project
Perform Experiments
Analyze Data
Write Abstract

Research Category
Basic Science

Title
THE ROLE OF RECOMBINANT PTX3 IN MURINE MODEL OF NEUTROPHILC ASTHMA

Background
Neutrophilic asthma, a severe subtype resistant to standard treatments like inhaled corticosteroids, remains the most understudied type in children. Its resistance to therapy and unclear mechanisms creates a significant economic and social burden. We have previously showed deficiency of Pentraxin 3 (PTX3), a protein expressed by immune cells, exacerbates airway inflammation and hyperresponsiveness in allergic asthma models.

Objective
This study aimed to assess PTX3's potential to reduce neutrophilic inflammation, and hyperresponsiveness in a murine model of neutrophilic asthma.

Methods
C57BL/6 mice were subjected to HDM+c-di-GMP to induce neutrophilic asthma. Asthmatic mice were intranasally administered recombinant PTX3 (rPTX3). hyperresponsiveness parameters were measured with FlexiVent ventilator. Lung tissue and BALF immunophenotyping were studied with flow cytometry. In addition, cytokines and serum immunoglobulins were assessed by mesoscale and ELISA, respectively.

Results
C57BL/6 mice, subjected to rPTX3, exhibited reduced airway and tissue resistance. However, total lung resistance and tissue remained unchanged. Totoal number of BALF cells remained unchanged after rPTX3 administration. Surprisingly, rPTX3 enhanced cells influx in lung. In mice exposed to HDM+c-di-GMP, rPTX3 significantly increased absolute number and percentage of eosinophils in both lung and BALF. Moreover, the percentage of neutrophils were also significantly higher in BALF. rPTX3 treatment led to a non-significant increase in macrophage numbers and percentage in the BALF. The absolute number and percentage of CD4+ and CD8+ T cells and B cells remained unchanged in the lung after rPTX3 treatment. Additionally, mice received rPTX3 demonstrated elevated total and HDM-specific IgE, while displaying comparable levels total and HDM-specific IgG1, IgG2a, IgG2b.

Conclusion
Although recombinant PTX3 reduced airway and tissue resistance, it also led to increased inflammatory cell recruitment. Further research is needed to evaluate its effectiveness in addressing other clinical asthma features (e.g. airway remodeling) and its potential as a therapeutic agent.

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No

Authors

Name	Email	Role	Profession
Sina Taefehshokr	taefehss@myumanitoba.ca	Presenting Author	Graduate
Lianyu Shan	lianyu.shan@umanitoba.ca	Co Author	Graduate
Mojdeh Matloubi	matloubim@myumanitoba.ca	Co Author	Graduate
Sujata Basu	sujata.basu@umanitoba.ca	Co Author	Graduate
Abdelilah Soussi Gounni	abdel.gounni@umanitoba.ca	Co Author	Full Professor