CHRD 2024: Abstract Submission Form

Presenter Name
Huda Rajani

Presenter Status
Masters Student

Role in the project
Design
Perform Experiments
Analyze Data
Write Abstract

Research Category
Basic Science

Title
SEMAPHORIN 3E DOWNREGULATES MESENCHYMAL MARKERS GENE EXPRESSION IN HUMAN PRIMARY EPITHELIAL CELLS

Background
Asthma is more prevalent in children than adults, affecting airway development and function. While corticosteroids reduce inflammation, they do not reverse airway remodeling, particularly in epithelial-mesenchymal transition (EMT). Our previous studies have demonstrated reduced expression of Semaphorin 3E (Sema3E) in allergen-challenged mice and patients with severe asthma.

Objective
This study investigates the role of Sema3E in modulating EMT in primary human bronchial epithelial cells (BECs).

Methods
Primary BECs (N=3) from healthy individuals were cultured under an air-liquid interface (ALI) and in submerged conditions, while A549 cells were maintained in submerged culture. Cells were stimulated with house dust mite (HDM) extract, IL-13, TGF-β, and Sema3E. RNA expression of EMT markers, including E-cadherin (CDH1), TWIST, SNAI, vimentin, SMAD, and ZEB, was measured at various time points.

Results
TGF-β and HDM significantly upregulated Sema3E expression in A549 and primary BECs, $p<0.05$. Increased expression of EMT markers (SNAI, TWIST, Vimentin, ZEB2, and SMADII) was observed following stimulation with TGF-β, IL-13, and HDM, while Sema3E reduced the expression of these markers (fold change < -1). Conversely, Sema3E combined with TGF-β, HDM, or IL-13 upregulated CDH1 expression (fold change > 1).

Conclusion
The data suggest that Sema3E is a key regulator of EMT in the airway epithelium. TGF-β-induced Sema3E expression likely regulates EMT in BEC through a negative feedback loop.

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No

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