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An Investigation into the Relationship between Type II Epithelial Cells and Alveolar Macrophages in Chronic Obstructive Pulmonary Disease (COPD)

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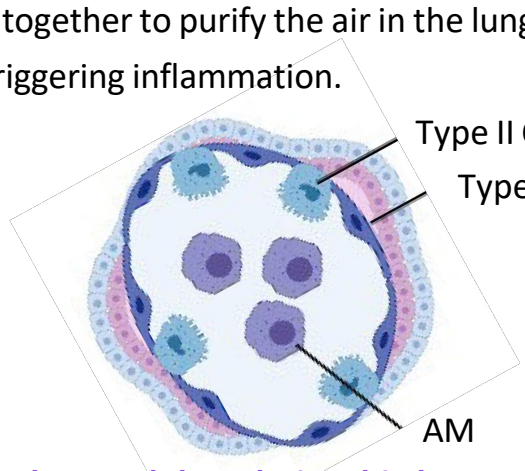
1. Introduction

- COPD is the 3rd leading cause of death worldwide, contributing to 3.2 million deaths in 2017 [1].
- Symptoms: breathing difficulty, a chronic cough and inflammation of the lungs.
- Causes: smoking & exposure to air pollution.
- It is a terminal disease with very few treatments available as it is severely underfunded and under researched.

I studied the interactions of 2 cells found in the alveoli of the lungs:

1. Type II cells = epithelial cells (line the alveoli & secrete mucous etc.).
2. AMs = alveolar macrophages (immune cells that engulf pathogens).

These cells work together to purify the air in the lungs. In COPD, they are overactive, triggering inflammation.



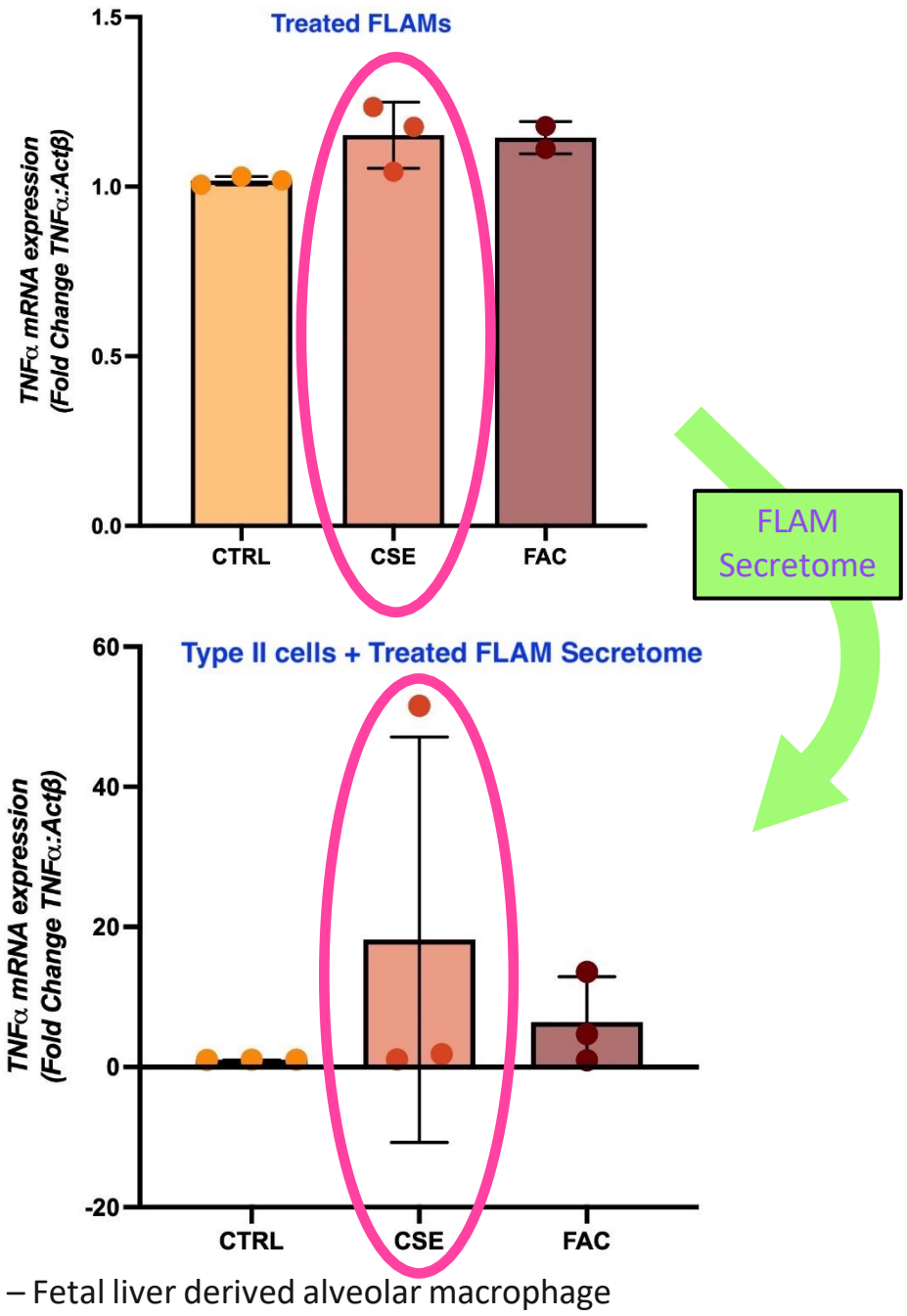
Project aim: Understand the relationship between these cells to discover a treatment

2. Methodology

a) Treatment Stage – cell culturing
- CSE - cigarette smoke extract
- FAC – ferric ammonium citrate (iron)

b) Analysis Stage
- Inflammatory markers were measured using RT-qPCR

3. Results



*FLAM – Fetal liver derived alveolar macrophage
* Secretome – the substances released by cells during treatment such as inflammatory markers

4. Discussion & Conclusions

- FLAMs were treated with CSE and FAC in order to mimic the conditions of COPD. They responded as expected, by secreting pro-inflammatory markers such as TNFα.
- The secretome of these AMs was transferred onto healthy type II cells in order to observe their response. This step mimicked the alveolar conditions where both cell types are in close association.
- Upon analysis, it was found that the dysfunctional FLAMs induced a similar reaction in the type II cells releasing the same cytokines (seen in the graph above with an increase in TNFα production).
- This information suggests that the overactivity of one cell type in the alveoli can trigger the other to react similarly, therefore exacerbating the inflammatory response in the lungs and contributing to the symptoms of COPD.

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