ATS 2023 Highlights

Respiratory Structure and Function Early Career Professionals

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Get to know members of the RSF Assembly

Is your research clinical, basic science or translational?

Translational & Clinical

Tell us about your research?

Under Dr. Don Sin's expert guidance, I am studying airway mucosal changes in patients with chronic obstructive pulmonary disease (COPD) and those experiencing persistent pulmonary symptoms from long COVID using single-cell RNA sequencing. My research objectives include understanding the molecular basis of COPD and pulmonary long COVID, discovering biomarkers, and deep phenotyping of these patients. This work aims to enhance our understanding of these conditions and advance personalized diagnostic and therapeutic approaches.

Where do you see yourself in 5 years?

In five years, my aim is to continue my research journey, working to uncover the genetic underpinnings of respiratory conditions and their associated risks. Whether in academia or industry, my aspiration is to play a role in improving patient care through innovative diagnostic and therapeutic approaches. I'm dedicated to making a meaningful impact in the field, helping enhance the lives of those affected by these conditions.

What do you find is the major benefit of RSF Assembly Membership?

The RSF Assembly fosters an incredible sense of fellowship. It connects early career professionals with leading respiratory researchers, inspiring collaborative opportunities and international ties. This supportive community is ideal for networking and scientific partnerships, making it a valuable resource for emerging researchers.

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Figure 1:

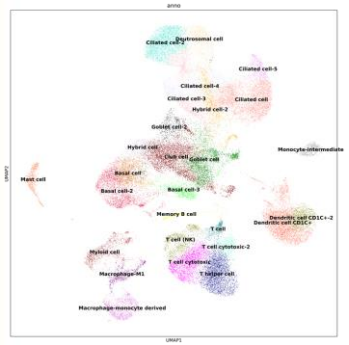


Figure 2a:

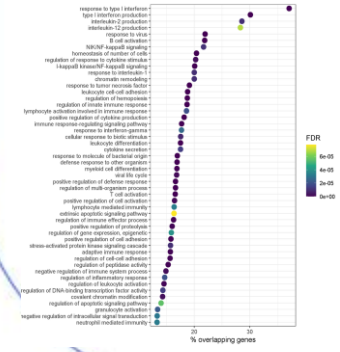
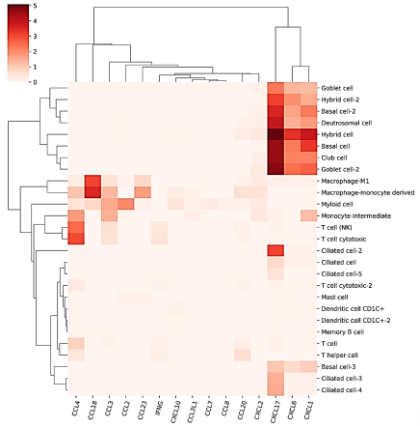


Figure 2b:

Figure 2c:



Airway mucosal abnormalities in post-COVID-19 patients with persistent pulmonary symptoms: results from single-cell RNA-sequencing

Objective: Long COVID is a significant emerging public health challenge. The symptoms of long COVID are highly variable, but persistent pulmonary symptoms identified to linger longer. We suspect that changes in the airway mucosa may explain why some long-COVID patients experience persistent pulmonary symptoms. Therefore, we profiled the cellular composition and transcriptome of the airway mucosa in post-COVID-19 individuals, with and without persistent pulmonary symptoms, using single-cell RNA-sequencing (scRNAseq) technology.

Methods: We recruited volunteers who were post-COVID-19 with persistent pulmonary symptoms ('PLC'), defined as new or worsening pulmonary symptoms at >12 weeks post-infection. Our control group ('HC') consist of both individuals with no history of COVID-19 infection, as well as those who were > 12 weeks post COVID-19 infection without onset of persistent pulmonary symptoms. Samples were collected from 6th-8th generation airways using cytological brushing via bronchoscopy. A single cell suspension was created, and cells were sequenced using the Chromium X (reagents v3.1, 10X Genomics, Pleasanton, CA) and Illumina NextSeq2000 (Illumina, San Diego, CA) platforms. Cell Ranger, SoupX, and Pegasuspy software were used for downstream analysis.

Results:. We sequenced a total of 44,736 cells from n=15 individuals (Figure 1a). In basal cells, there was a shift in distribution from a classical basal cell (KRT5high/TP63high) towards a more differentiated phenotype (KRT5low/TP63– with upregulation of secretory markers SCGB1A1/MUC5AC/MUC5B) in PLC compared to HC. Among immune cells, PLC airways showed a shift in T cells toward a more cytotoxic phenotype, where further pathway analysis was indicative of upregulation of various cytokines and chemokines, alongside antiviral response genes (Figure 2a,b,c).

Conclusion:
Pulmonary phenotype of long COVID is characterized by activated airway epithelial cells & inflammation. These include altered distribution of basal cell sub-types, upregulation of cytokines, chemokines and type I interferon genes across various cell types, and more presence of cytotoxic T cells. These data indicate perturbations in the airway mucosa following COVID-19, which may explain the persistent pulmonary symptoms Long COVID.

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