

# ATS 2022 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

***Tell us about your research?***

My research is focused on the intersection between imaging and 'omics to study the heterogeneity of lung disease and identify novel pathological features with a primary focus on COPD and IPF.

***Where do you see yourself in 5 years?***

I hope that my research will help open new avenues for the development of therapeutics in treating lung disease. Personally, I look forward to leading my own team, either in academia or industry, to further the study of lung pathology.

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

RSF has helped me to develop a network comprised of like-minded scientists who are focused on the structure and function of the lung. This is especially important as a PhD scientist in a clinical department.



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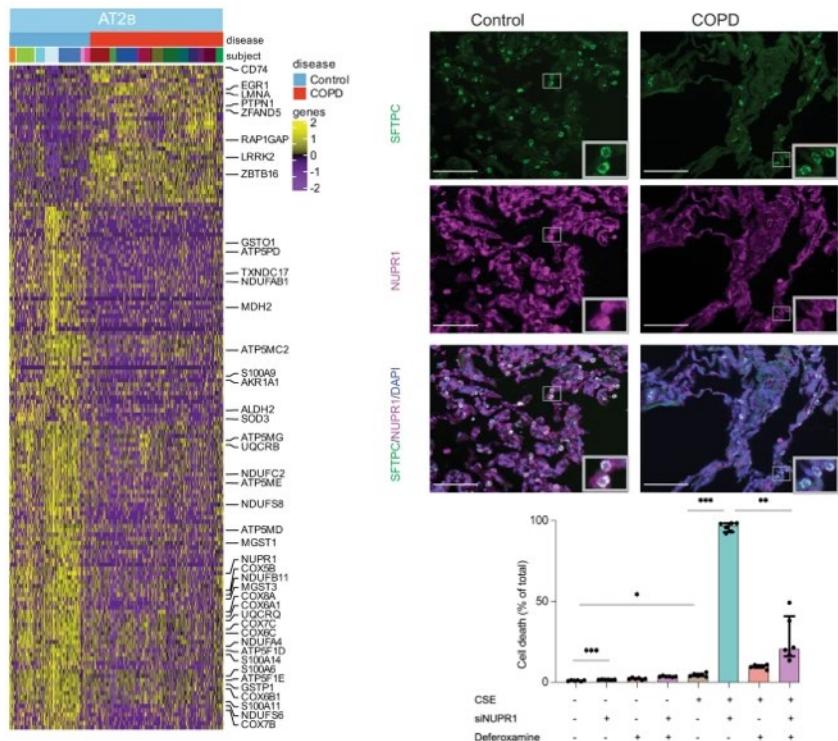
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### Characterization of the COPD alveolar niche using single-cell RNA sequencing

**Objective:** Chronic obstructive pulmonary disease (COPD) is a leading cause of death worldwide, however our understanding of cell specific mechanisms underlying COPD pathobiology remains incomplete.

**Methods:** We analysed single-cell RNA sequencing profiles of explanted lung tissue from subjects with advanced COPD or control lungs, and validated findings using single-cell RNA sequencing of lungs from mice exposed to 10 months of cigarette smoke, RNA sequencing of isolated human alveolar epithelial cells, functional *in vitro* models, and *in situ* hybridization and immunostaining of human lung tissue samples.

**Results:** We identified a subpopulation of alveolar epithelial type II cells with transcriptional evidence for aberrant cellular metabolism and reduced cellular stress tolerance in COPD. Using transcriptomic network analyses, we predict capillary endothelial cells are inflamed in COPD, particularly through increased CXCL-motif chemokine signaling. Finally, we detect a high-metallothionein expressing macrophage subpopulation enriched in advanced COPD.

**Conclusion:** Collectively, these findings highlight cell-specific mechanisms involved in the pathobiology of advanced COPD.



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