ATS 2019 Highlights

Respiratory Structure and Function Early Career Professionals

Get to know members of the RSF Assembly

Jade Jaffar, PhD, ATSF

Research Scientist/Manager; Alfred Lung Fibrosis Biobank
Depts. Respiratory Medicine and Immunology
The Alfred Hospital and Monash University

Jade's LinkedIn

Is your research clinical, basic science or translational?

Translational

Tell us about your research?

My research involves creating cell-based assays with which to accelerate development of pre-clinical candidates for idiopathic pulmonary fibrosis (IPF). I also am interested in identifying novel diagnostic and prognostic biomarkers for IPF and other interstitial lung diseases.

With my current supervisor Prof. Glen Westall, I established the Alfred Lung Fibrosis Biobank in 2014 to use as a resource for translational research that is shared with other Australian academic researchers and that works in tandem with industry.

Where do you see yourself in 5 years?

I hope that I can expand the Alfred Lung Fibrosis biobank to serve as a self-sustaining platform for translational lung research in Australia.

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

The best part of the RSF Assembly are the members themselves. I've met so many inspirational peers and mentors which give me new perspectives and amazing scientific (and social!) support.

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If you or someone you know would like to be featured as an ATS RSF ECP please email Katrina Tonga (katrina.tonga@sydney.edu.au)

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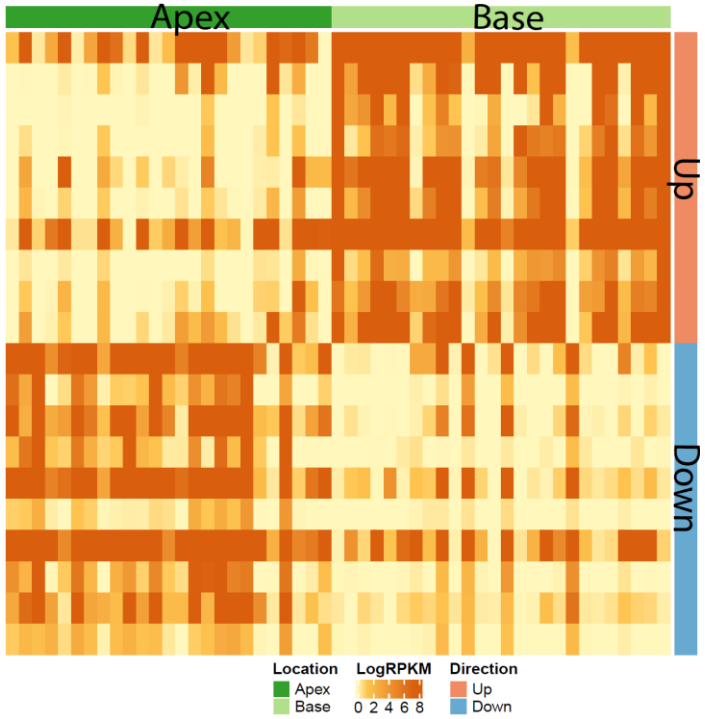

Figure 1. Heat-map showing the top 10 differentially expressed genes in matched lung tissue derived from the apex and base regions of 17 patients with idiopathic pulmonary fibrosis (IPF).
RPKM reads per kilobase million

Matrix metalloproteinases are increased in lung bases compared to apices in patients with idiopathic pulmonary fibrosis (IPF)

Introduction/Aim: Idiopathic pulmonary fibrosis (IPF) is a lethal fibrotic lung condition with an unpredictable disease course. The pathology of IPF starts in the lung bases such that a patient's lung apices are comparatively less fibrotic at the time of transplantation. Matrix metalloproteinases (MMPs) drive fibrogenesis. We hypothesized that RNA sequencing of the lung apices and bases may identify other differentially expressed MMPs to better understand disease progression in IPF.
Methods: High-quality mRNA was collected from resected lung apex and base tissue from 17 patients with IPF for a total of 52 samples. Single-end RNA sequencing using Illumina platforms and differential expression analysis was performed.
Results: A total of 691 genes were identified to be differentially expressed in IPF lung bases when compared to matched apices (Figure 1). Out of these, 563 genes were up-regulated and 128 genes were down-regulated. The expression of 16 MMPs (1, 2, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 19, 24, 25, 28) was detected in lung tissue. Five of these MMPs were up-regulated in lung bases compared to apices, namely MMP-1, MMP-7, MMP-10, MMP-12, and MMP-13.
Conclusions: Our results demonstrate that the expression profile in lung bases is vastly different to that in regions of the lung apices within the same patient and disease progression may be driven by MMPs. Five differentially expressed MMPs together encompass the spectrum of mechanisms thought to drive IPF; epithelial-to-mesenchymal transition (MMP-7), macrophage M1 to M2 switching (MMP-10), leukocyte migration (MMP-12), extracellular matrix dysregulation (MMP-13 and MMP-1). Targeting MMPs may provide clues to halting fibrosis.

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