

# ATS 2020 Highlights

## Respiratory Structure and Function Early Career Professionals

### ***Get to know members of the RSF Assembly***

#### ***Is your research clinical, basic science or translational?***

Translational

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

My lab applies a multidisciplinary approach to design and develop bio-inspired technologies that enable us to elucidate cellular and molecular mechanisms that govern tissue pathology or offer protection during lung injury. Our research lies at the intersection of respiratory medicine, immuno-microbiology, tissue engineering, and systems and synthetic biology. Ultimately, my lab's goal is to discover novel druggable targets and personalized diagnostics using microengineered systems that recreate complex human organ pathophysiology *in vitro*.

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

I see myself expanding my research program and taking discoveries and technologies that my team is developing to clinic to enable a positive impact on healthcare.

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

Great opportunity for networking, building collaboration and learning to ask clinically relevant scientific questions!

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@ATS\_RSf



**Kambez H. Benam, D.Phil.**

*Assistant Professor of Medicine  
and Bioengineering  
University of Colorado Denver*

[https://benamlab.net/;](https://benamlab.net/)  
[@KambezBenam;](#) [@BenamLab](#)

*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](mailto:katrina.tonga@sydney.edu.au))*

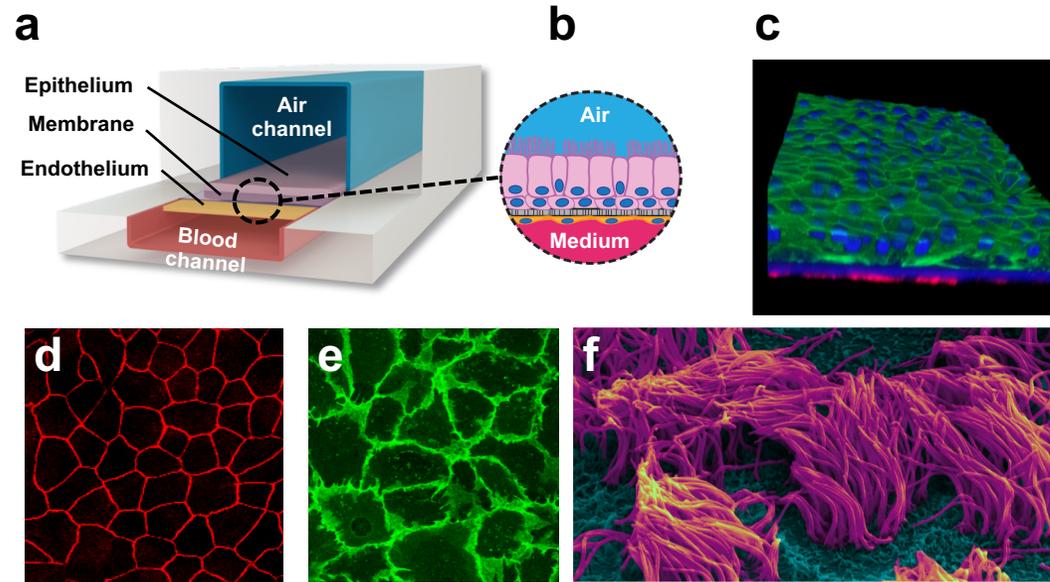


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**Primary Human Lung Airway-on-a-Chip.** (a, b) Chip design schematics, (c) Confocal vertical cross-section through pseudostratified epithelium (green), membrane (blue) and endothelium (red). (d) ZO-1 tight-junction staining of epithelium. (e) VE-cadherin cell boundary immunostaining in endothelium. (f) Cilia (purple) & mucin (green) on surface of epithelium in a pseudo-colored scanning electron micrograph. Reproduced from Benam et al. *Nature Methods* 2016 & *Cell Systems* 2016.

### **Small airway-on-a-chip enables analysis of human lung inflammation and drug responses *in vitro***

Here we describe the development of a human lung 'small airway-on-a-chip' containing a differentiated, mucociliary bronchiolar epithelium and an underlying microvascular endothelium that experiences fluid flow, which allows for analysis of organ-level lung pathophysiology *in vitro*. Exposure of the epithelium to interleukin-13 (IL-13) reconstituted the goblet cell hyperplasia, cytokine hypersecretion and decreased ciliary function of asthmatics. Small airway chips lined with epithelial cells from individuals with chronic obstructive pulmonary disease recapitulated features of the disease such as selective cytokine hypersecretion, increased neutrophil recruitment and clinical exacerbation by exposure to viral and bacterial infections. With this robust *in vitro* method for modelling human lung inflammatory disorders, it is possible to detect synergistic effects of lung endothelium and epithelium on cytokine secretion, identify new biomarkers of disease exacerbation and measure responses to anti-inflammatory compounds that inhibit cytokine-induced recruitment of circulating neutrophils under flow.

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