

ATS 2019 Highlights

Respiratory Structure and Function Early Career Professionals

Get to know members of the RSF Assembly



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Is your research clinical, basic science or translational?

Basic Science

Tell us about your research?

Our laboratory is interested in identifying new pathways in chronic obstructive lung disease (COPD) and pulmonary fibrosis to improve therapeutic options for patients. We are studying the protective role of adenine nucleotide translocase (a mitochondrial ADP/ATP transporter) in the airway and alveolar epithelium in the context of cigarette smoking-related lung disease and lung fibrosis.

Where do you see yourself in 5 years?

My goal is to have a vibrant and productive lab team in academia. I hope to be able to expand our team size as I love to mentor students, graduate students and post-doctoral fellows as they are the future of science. Pulmonary is an exciting and open area for advances in biomedical research. I want to be able to contribute to bringing new therapies from the lab to the bedside.

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

Being an RSF member has been so helpful in building a scientific community and networking with people from institutions from around the world. As a member of the ECP Working Group, I have collaborated with so many amazing individuals and gained leadership experience that has helped my career to grow.

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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)



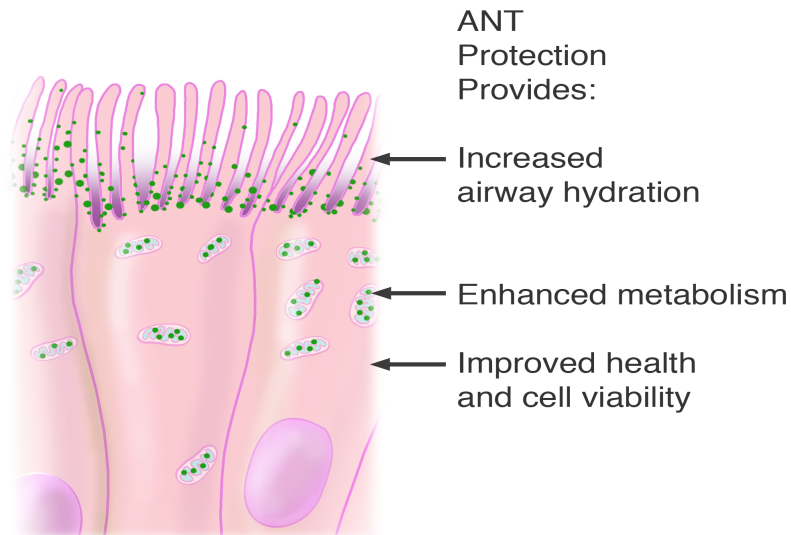
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Mitochondrial Adenine Nucleotide Translocase influences ciliary function and airway homeostasis

Airway hydration and ciliary function are biological processes that are critical to airway homeostasis and are dysregulated in chronic obstructive lung disease (COPD). To identify new relevant pathways, we utilized the amoeba *Dictyostelium discoideum* as a novel comparative discovery tool for lung biology and identified adenine nucleotide translocase (ANT), a mitochondrial ADP/ATP transporter, as being protective against cigarette smoke in *Dictyostelium* and human bronchial epithelial cells. In addition to mitochondria, populations of ANT1 and ANT2 may reside at the apical plasma membrane and motile cilia in airway epithelia. At the plasma membrane, ANT2 stimulates an increase in airway surface liquid hydration, mediated via ATP. ANT2 also protects against smoke-induced slowing of ciliary beat in primary human ciliated airway epithelial cultures. ANT2 gene expression is reduced in lung tissue from COPD patients and in a mouse model of COPD, suggesting ANT2 may be associated with COPD pathogenesis. These findings further highlight the potential of ANT2 modulation in protecting from metabolic defects, airway hydration, ATP regulation, and ciliary motility defects, thereby maintaining airway homeostasis. The unexpected role of ANT in airway function may provide the clue about a key missing link in the regulation of airway surface hydration, which is essential for many airway diseases including COPD.

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