ATS 2018 Highlights

Respiratory Structure and Function Early Career Professionals

Get to know members of the RSF Assembly

Jacelyn Emily Peabody, MD/PhD Trainee

University of Alabama at Birmingham
Medical Scientist Training Program (MSTP) – 3rd Year, GS-1 [Contact Jacelyn]

Is your research clinical, basic science or translational?

Basic and translational science.

Tell us about your research?

I am developing a novel bleomycin-induced pulmonary fibrosis ferret model to evaluate mucociliary physiology in idiopathic pulmonary fibrosis (IPF). Unlike rodent models, ferret airways contain submucosal glands, the major source of MUC5B in humans. I have begun to elucidate defects in the mucociliary transport apparatus via micron-optical coherence tomography and micro-CT imaging.

Where do you see yourself in 5 years?

I aim to be finishing up my intern year at an academic medical center although I am trying to decide between internal medicine or surgery. I also want to be involved with medical education to help teach and mentor the next generation of physician scientists. The potential impact of my work and career path is what inspires me day in and day out! By treating patients and advancing scientific knowledge, I will be able to help patients both directly and indirectly.

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

Identifying near-peer mentors and being able to network with leaders in the field! These contacts have bolstered my scientific knowledge and are helping shape my career trajectory. This is a supportive, collegial group of people who I look forward to continue interacting with throughout my career!

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If you or someone you know would like to be featured as an ATS RSF ECP please email Jade Jaffar (jade.jaffar@monash.edu)

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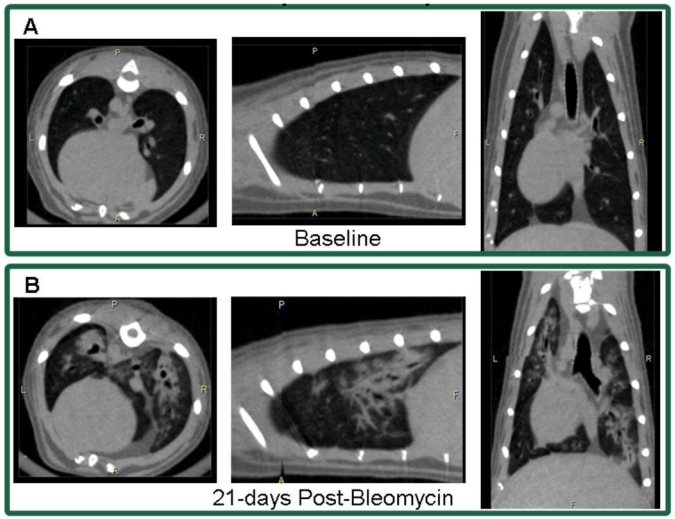

Bleomycin Induces Patch Airway-centric and Peripheral Ground Glass Opacities on µCT in Ferret

A. Baseline axial, sagittal, and coronal µCT images from a female ferret thorax prior to treatment with bleomycin. **B.** Axial, sagittal, and coronal µCT images from the same female ferret, 21-days post-bleomycin treatment.

Matters of Mucus: Mucociliary Physiology in a Bleomycin-Induced Pulmonary Fibrosis Ferret.

Introduction: Idiopathic pulmonary fibrosis (IPF) is a progressive fibrotic lung disease with median-survival ranging from 3-5 years after diagnosis. Although the greatest risk factor for developing IPF is a variant in the MUC5B gene, the role of MUC5B in IPF pathogenesis is unknown. We are developing a novel bleomycin-induced pulmonary fibrosis ferret model to evaluate the hypothesis that mucociliary physiology may alter pro-fibrotic mechanisms.

Methods: A single-dose of bleomycin-sulfate solution (2.5-5U/kg) or saline-vehicle was administered intratracheally via microaerosolization to normal ferrets. Fibrosis was assessed with µCT scans, histology, and second harmonic imaging. Muc5B expression was assessed with immunohistochemistry.

Results: All ferrets (N=16) survived to euthanasia at 3 or 6 weeks post-bleomycin administration. µCT scans demonstrated evidence of patchy airway-centric and peripheral ground glass opacities that was worse in the dependent lung, evident at 2 weeks, and persistent through 6 weeks. Threshold-based volumetric µCT analysis revealed that bleomycin-treated lungs showed 38.2% fibrosis and a significant increase from baseline compared to controls (mean increase 18.1±2.2% bleomycin compared to -0.8±0.8% control, $P<0.001$) (Figure).

Conclusions: Bleomycin treatment induced pulmonary fibrosis in ferrets, with inflammation, fibrotic lesions, and remodeling changes analogous to IPF in humans. Contemporaneous analysis of fibrosis development and the mucociliary transport apparatus suggests a strong relationship and indicates that the altered airway surface microenvironment may affect the pathogenesis of fibrosis.

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