ATS 2026 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 focuses on advancing our understanding of chronic obstructive pulmonary disease (COPD) through development of innovative imaging biomarkers. Specifically, I leverage low dose chest computed tomography (CT) images to quantify structural changes in the pulmonary vasculature within the lungs and develop novel biomarkers that can capture pulmonary vascular remodeling in COPD. These imaging derived metrics may have the potential to improve disease phenotyping, enable early detection of pulmonary vascular involvement in COPD. My research also has the potential to provide clinicians with more precise quantitative tools for monitoring disease progression and tailoring therapeutic interventions.

Where do you see yourself in 5 years?

My aim is to become a clinical medical physicist, integrating strong academic, clinical, research and community outreach. I envision myself as a clinical medical physicist, who contributes to novel innovations in the field of medicine, train next generation physicists and researchers.

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

Provides an opportunity to researchers and clinicians in respiratory research and patient care to collaborate, network, present their work and share ideas, thereby making a real impact on lung health.

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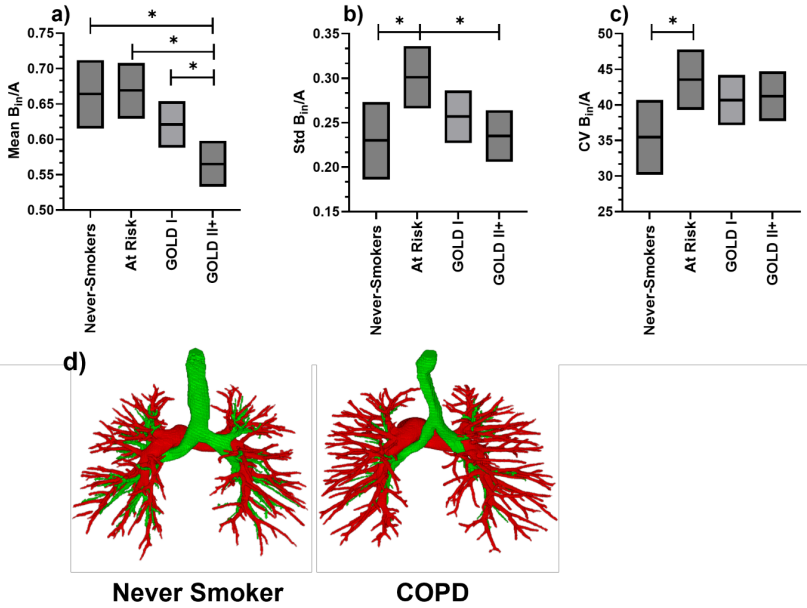

Figure 1: (Top) Comparison of **a)** mean Bin/A, **b)** Std Bin/A and **c)** CV Bin/A metrics between never-smokers, at-risk, GOLD I and GOLD II+. **d)** Comparison of 3D pulmonary arteries segmentation (red) overlayed on 3D airways segmentation (green) visualization between healthy never-smoker (66-year-old male; FEV_1: 3.2L; FEV_1/FVC: 73.2; mean Bin/A:0.71) and COPD (67-year-old male; FEV_1: 2.4L; FEV_1/FVC: 50.4; mean Bin/A:0.50) participant.

Bronchial-to-Artery (BA) Ratio: A Quantitative CT Marker of Airway–Vascular Coupling in COPD

Introduction: Chronic obstructive pulmonary disease (COPD) patients show remodeling and loss of distal airways and arteries, disrupting airway-vascular coupling even in the early stages. Previous studies have quantified this airway-vascular coupling on computed tomography (CT) images as the ratio of bronchial lumen diameters to adjacent arteries (BA) in individuals with COPD, and showed associations increased exacerbations and emphysema progression. However, previous studies have not quantified heterogeneity of the BA ratio in early/at risk COPD. We hypothesize the mean BA ratio will decline with increasing COPD severity, and that the variability in BA ratio will peak in the 'at-risk' group, reflecting early heterogeneous loss of coupling. Therefore, our objective was to develop a fully-automated artery-vein separation method pipeline, and to apply this across a large community-based cohort to quantify BA pair heterogeneity in individuals with and at risk of COPD.

Methods: Participants from CanCOLD with full-inspiration CT images were selected for analysis. Arteries were segmented using a deep learning segmentation pipeline, trained on 250 labelled chest CT images using 3D MedNeXt architecture. The arteries were spatially matched with the airways by determining centroids for each segment, ensuring parallel segment orientation and matching each airway segments to nearest arterial segments using minimum centroid-to-centroid distances. BA pairs were determined for five major airway segments (RB1, RB4, RB10, LB1 and LB10). For each of the segments, bronchial lumen diameter (Bin) and adjacent artery diameter (A) was calculated. Mean (mean Bin/A), standard deviation (Std Bin/A) and coefficient of variation (CV Bin/A) of the BA ratios were determined. Analysis of covariance was used for comparison of mean, standard deviation and coefficient of variation of BA ratios between never-smokers, at-risk, GOLD I and GOLD II+ COPD, adjusted for covariates (age; sex; pack-years; smoking status; body mass index; low attenuation area less than 910 Hounsfield units; CT lung volume and CT model).

Results: A total of 1082 CanCOLD participants having all BA pairs were evaluated: n= 221 never-smokers; n=306 at-risk; n=324 GOLD I; and n= 231 GOLD II+. Mean Bin/A was significantly reduced in GOLD II+ as compared to the other groups ($p<0.05$). Std Bin/A was increased in at-risk groups compared to never-smokers and GOLD II+ COPD ($p<0.05$). CV Bin/A was increased in at-risk groups compared to never-smokers ($p<0.05$).

Conclusion: These findings indicate that airway-to-artery ratio heterogeneity is an early marker of airway-vascular uncoupling and highlights its potential utility as a quantitative imaging endpoint for intervention studies.

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