Get to know members of the RSF Assembly

Is your research clinical, basic science or translational?
Translational

Tell us about your research?
Broadly, I am interested in identifying molecular determinants of chronic lung diseases – asthma and COPD, and the mechanisms by which they influence lung function and disease progression. My current focus is in investigating the role of autoimmune responses in COPD, particularly exploring common molecular mediators between COPD and its frequent comorbidity, cardiovascular disease.

Where do you see yourself in 5 years?
I hope to establish an independent research program that integrates classical bench techniques with cross-cutting computational methods, leveraging high-dimensional data from clinical and epidemiological cohorts to better understand shared molecular underpinnings of COPD and cardiovascular disease.

What do you find is the major benefit of RSF Assembly Membership?
The RSF assembly is a great scientific community for networking and collaboration. Membership also provides the opportunity to become more involved within the ATS.

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If you or someone you know would like to be featured as an ATS RSF ECP please email Carolyn Wang (carolyn.wang@hli.ubc.ca)
Objective: To investigate the role of antibodies to collagens in COPD.

Methods: TESAOD is a longitudinal, population-based epidemiological cohort with questionnaire and lung function data collected at baseline and multiple surveys, and mortality data from National Death Index searches. Baseline serum levels of autoantibodies to collagen types I, II, IV and VI (N=748) were measured using a multiplex bead-based autoimmune assay (Myriad-RBM Laboratory, Austin, TX). Subsequent to initial assessments by spearman correlations, a principal component analysis was performed for the four anti-collagens to create an “anti-collagen score” using the first principal component, after adjustment for age, sex and batch effect. Cross-sectional associations were tested between anti-collagen scores and percent-predicted (pp) lung function parameters FEV₁, FVC, FEV₁/FVC and FEF₂₅₋₇₅% by linear regression, and risk of an obstructive (defined as FEV₁/FVC<70) or restrictive (defined as FEV₁/FVC≥70 with ppFVC<80%) spirometric pattern, by multinomial logistic regression. All-cause mortality was analyzed using Cox proportional hazards regression. All analyses were adjusted for age, sex, body mass index, smoking, pack-years, and asthma.

Results: The first principal component explained ∼41% of the variance of the four anti-collagens and had strong positive loadings for anti-collagens II, IV and VI. The anti-collagen score was inversely associated with ppFEV₁/FVC (adjβ= -1.56, p<0.001) and ppFEF₂₅₋₇₅% (adjβ= -4.41, p=0.001). Consistent with these results, anti-collagen scores in the high tertile conferred increased risk of an obstructive (adjRRR=2.61, p=0.009), but not restrictive (adjRRR=0.90, p=0.725), spirometric pattern, as compared with scores in the low tertile. Anti-collagen scores in the high tertile were also associated with increased mortality risk (adjHR=1.26, p=0.051), particularly among ever-smokers (N=459, adjHR=1.62, p=0.002) and those with an obstructive spirometric pattern (N=70, adjHR=3.72, p=0.002) at baseline.

Conclusion: Taken together, these data provide evidence that levels of anti-collagens are associated with altered lung function and increased mortality risk in COPD.

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Elevated levels of anti-collagens are associated with increased risk for all-cause mortality, particularly in ever-smokers (top) and those with airflow limitation (bottom).