**High resolution CT-based characterization analysis of idiopathic pulmonary fibrosis**

Zhang, Y.1, Clark, A.R.1, Kumar, H.1, Milne, D.2, Wilsher, M.2, Bartholmai, B.J.3, Tawhai, M.H.1

1Auckland Bioengineering Institute, University of Auckland, Auckland, NZ, 2Auckland City Hospital, Auckland, New Zealand, 3 Department of Radiology, Mayo Clinic, Minnesota, US.

**Introduction**

Idiopathic pulmonary fibrosis (IPF) is the most aggressive and frequent form of idiopathic interstitial pneumonias. Progression of IPF is variable between individuals and no established quantitative tools exist to assess its development. This work aims to integrate information from volumetric imaging with pulmonary function tests using a predictive computational model under the hypothesis that abnormalities on volumetric are not sufficient to explain increased lung stiffness and decreases in DLCO.

**Method**

Tissue regions in HRCT images from 8 patients with IPF were quantitatively analysed. Tissue abnormalities were classified using CALIPER (Computer-Aided Lung Informatics for Pathology Evaluation and Ratings) software. Tissue density (TD) distribution and volume of tissue (classified as reticular, ground-glass, normal and emphysema in each lung) were calculated. The classified data were mapped to a statistical shape model, which allows consistent comparison of regional tissue properties between patients or within one patient at different time points. Ventilation distribution and gas exchange were simulated in a computational model that was parameterised to each subject’s lung tissue characterisation, to predict the relationship between V-Q matching, DLCO and disease distribution.

**Result**

Fibrosis usually has a consistently higher TD (0.34/0.41 for reticular/ground-glass) compared to normal tissue (0.28), and presents predominantly in lower lobes (72%, 58%, 65% for honeycomb, reticular, ground-glass). In contrast, emphysema has lower density (0.08) and appears predominantly in upper lobes (73%). Model predictions of ventilation distribution and DLCO show that V-Q mismatch occurs due to redistribution of ventilation away from diseased regions, contributing to observed DLCO decreases.

**Conclusion**

A quantitative analysis of the spatial distribution of IPF disease coupled with functional models provides a potential tool to improve assessment of the contributors to decline in IPF patient status over time. Decline in DLCO is a function of regional lung stiffness due to disease location combined with a redistribution of ventilation to ‘normal’ lung tissue.