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

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Also need to include Auckland City Hospital collabarators who provided the IPF data (any funding?)

**Introduction**

Idiopathic pulmonary fibrosis (IPF) is the most aggressive and frequently form of idiopathic interstitial pneumonias. It is a chronic and life-threatening disease of unknown cause, and occurs primarily in middle-aged and elderly adults. Progression of IPF is variable between individuals and unpredictable with few tissue-level biomarkers for progression identified. No established quantitative tools exist to assess rate of decline in IPF. The aim of this work is to integrate information from volume controlled imaging with standard pulmonary function tests using a predictive computational model under the hypothesis that tissue abnormalities analysed on volumetric CT are not on their own sufficient to explain increased lung stiffness and decreases in DLCO seen in IPF.

**Method**

Tissue regions in HRCT images from 8 patients with IPF were quantitatively analysed. IPF tissue was classified using CALIPER (Computer-Aided Lung Informatics for Pathology Evaluation and Ratings) software. Tissue density and lung volumes were analyzed from raw CT images combined with the disease locations. The classified data were also mapped to a statistical shape model, which allows consistent comparison between different patients or within one patient over time. Ventilation distribution and gas exchange were simulated to make a tredency prediction of V/Q matching and DLCO value in IPF lungs over time.

**Result**

Fibrosis usually has a consistently higher tissue density (0.3357, 0.4105 for reticular, ground-glass) compared to normal tissue (0.2752) over time, whereas emhysema has a lower value (0.0784). Most IPF patients experience a decrease of lung volume (11.85% off). Model predictions of ventilation distribution and DLCO show that V-Q mismatch occurs in IPF subjects even in CT evaluated ‘normal’ tissue and this impacts on lung function contributing to observed DLCO decreases.

**Conclusion**

A quantitative analysis of the spatial distribution of IPF disease coupled with computational models of function provide tools to improve assessment of contributors to decline in IPF patient status over time. Decreases in DLCO over time in IPF patients are a function of both regional lung stiffness due to disease location combined with a redistribution of ventilation to normal lung tissue.