elucidated. We hypothesised that Pellino1 would play a critical role in regulating the inflammatory response in the lung airway epithelium. Pellino1 was knocked down in the human bronchial airway epithelial cell line BEAS2B and primary human bronchial epithelial cells (PBECs) using targeted siRNA. Knockdown of Pellino1 at the transcriptional level was measured using qPCR. Pellino1 knockdown cells were stimulated with Toll-like receptor/IL-1R agonists or the natural pathogen rhinovirus 1B (RV1B) and cytokine release was measured using ELISA. Signalling pathways were explored by western blotting.Pellino1 was expressed in BEAS2B and primary bronchial epithelial cells. Pellino1 mRNA transcript level was knocked down by 78% in BEAS2B and 94% in PBECs. Pellino1 knockdown led to reduced IL-8 generation in response to IL-1ß and a viral mimic Poly (I:C) stimulation in BEAS2B, however RANTES production was unchanged. In contrast to the BEAS2B, the primary bronchial epithelial cells exhibited preserved IL-1 signalling. However, these cells also showed a reduction in IL-8 production in response to both Poly (I:C) stimulation and RV1B infection. Pellino1 knockdown had no effect on the interferon-stimulated gene RANTES production. Pellino1 knockdown leads to reduced IL-8 production in response to a viral mimic Poly (I:C) in lung airway epithelial cells and RV1B in PBECs. These data indicate that Pellino1 regulates proinflammatory responses in airway epithelium and may be a feasible target to downregulate neutro-phillic airway inflammation while retaining antiviral immunity, which would be highly beneficial in the treatment of chronic airway inflammatory disorders such as asthma and COPD. These studies were supported by an MRC/Asthma UK PhD studentship.

ILD: clinical studies

S133 THE ASSESSMENT OF HEALTH RELATED QUALITY OF LIFE IN INTERSTITIAL LUNG DISEASE WITH THE KING'S BRIEF INTERSTITIAL LUNG DISEASE QUESTIONNAIRE (K-BILD)

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Introduction The King's brief interstitial lung disease questionnaire (K-BILD) is a recently developed and validated 15 item HRQOL questionnaire comprising of 3 health domains (psychological, breathlessness and activities, and chest symptoms) and an overall HRQOL score. We set out to evaluate HRQOL in a large group of patients with interstitial lung diseases (ILD's) and determine the factors that influence it.

Methods 219 patients with ILD (60 idiopathic pulmonary fibrosis (IPF), 81 connective tissue associated ILD, 23 idiopathic non-specific interstitial pneumonitis (NSIP), 21 hypersensitivity pneumonitis, 10 organising pneumonia, 24 other) attending ILD clinics at King's College and Royal Brompton Hospitals completed the K-BILD. The K-BILD Scores range from 0 to 100, with a higher score representing a better HRQOL. Demographic data, immunosuppressant medication, long-term oxygen therapy use, multi-disciplinary team ILD diagnosis and lung function were recorded.

Results Patients had a mean (SEM) age of 60 (1) years, 75% of patients were Caucasian, 60% were females, mean (SEM) VC% predicted was 80 (24) % and TLCO % predicted was 47 (18)%. HRQOL was impaired in all domains, mean (SEM) scores: psychological 62 (2), breathlessness and activities 43 (2), chest symptoms 67 (2), total 59 (2). There were no significant associations between overall HRQOL and age (r=−0.007) or gender (p=0.13). There was a modest correlation between HRQOL and lung function (Abstract S133 table 1). HRQOL was significantly lower in IPF patients compared to other ILD's (total score 51 (3) vs 62 (2); p<0.01), those with UIP pattern vs NSIP (total score 51 (3) vs 62 (3); p<0.01) and those prescribed long-term oxygen therapy (total score 38 (4) vs 63 (2); p<0.01). IPF patients prescribed immunosuppressant medication had significantly worse overall HRQOL (48 (4) vs 74 (8); p=0.02). There was no significant difference between CTD-NSIP patients compared with idiopathic NSIP (total score 62 (3) vs 62 (5); p=0.91).

Conclusions HRQOL is impaired in patients with ILD. The type of ILD, immunosuppressant medication, and lung function all influence HRQOL. This study provides further clinical validation of the K-BILD.

S134 HIGH PREVALENCE OF ISCHAEMIC HEART DISEASE (IHD) IN PATIENTS MEETING CLINICO-RADIOLOGICAL PROFILE FOR IDIOPATHIC PULMONARY FIBROSIS (IPF)

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Introduction IPF and ischaemic cardiac disease may share pathological similarities including aberrant tissue remodelling. Given their recognised heterogeneity, we hypothesised that patterns of IPF (usual interstitial pneumonia, UIP; non-specific interstitial pneumonia, NSIP; indeterminate UIP-NSIP) may inter-relate differently with the prevalence of IHD and associated co-morbidities.

Methods Ethical approval was obtained to identify all cases of IPF undergoing high-resolution CT between 2003 and 2010. Details of cardiac disease and its management (risk factors, drug treatment, coronary angiography and revascularisation) were included. Consensus radiological diagnoses were reached by 2 chest physicians and 2 thoracic radiologists. Comparison of multiple variables was undertaken using logistic regression (STATA V.11).

Results Of 256 potentially suitable cases, 96 fulfilled ascertainment and were consented: 38 (40%) UIP, 44 (48%) NSIP and 14 (16%) indeterminate (INDET) pattern. An inter-observer k coefficient of 0.56 was calculated for radiological agreement. Regardless of morphological pattern, patients with IPF had a significant cardiovascular risk profile. Where present, IHD predated IPF in the majority of cases, with the highest occurrence (58%) in the UIP subgroup. UIP patients were of comparable age, gender frequency, smoking status and had similar rates of major cardiovascular risk factors as NSIP and INDET subjects. Crucially, they were 2.5 times more likely than NSIP patients to have IHD after adjustment for

Abstract S133 Table 1 The relationship between the HRQOL assessed with the K-BILD and lung function

	Psychological	Breathlessness and activities	Chest symptoms	Total score
FEV$_1$ % predicted	r=0.34	r=0.46	r=0.38	r=0.43
	p<0.01	p<0.01	p<0.01	p<0.01
FVC % predicted	r=0.32	r=0.45	r=0.38	r=0.42
	p<0.01	p<0.01	p<0.01	p<0.01
TLCO % predicted	r=0.41	r=0.48	r=0.35	r=0.46
	p<0.01	p<0.01	p<0.01	p<0.01
CPI	r=0.36	r=0.44	r=0.35	r=0.42
	p<0.01	p<0.01	p<0.01	p<0.01

Data shown are Spearman's correlation coefficients.
CPI, Composite physiologic index.