capacity. The ISWT is one field exercise test which is often employed in the assessment of pulmonary and cardiac rehabilitation (PR, CR) patients and also to prescribe a walking speed. In PR and CR programmes the ISWT is employed to calculate walking speed set at a desired training threshold. The aim of the study was to establish reference values for the ISWT and an equation for its prediction in a healthy population. This will allow comparison between patients from PR and CR programmes and healthy age-matched controls.

Methods Subjects were aged between 40 and 90 years, had normal spirometry defined as FEV_1 % pred >80% and/or a FEV_1/FVC >70% and had no known co-morbidities affecting mobility. The best distance from two ISWT was recorded along with body mass index (BMI) and leg length. Quadriceps maximal voluntary contraction (QMVC: Kg) was measured using a strain gauge (Kern). Physical activity was assessed using the DUKE physical activity questionnaire and an activity monitor (SenseWear PRO^2 Armband). The number of steps and energy expenditure achieved over 2 days was recorded. Subjects also completed the Hospital Anxiety and Depression Scale.

Results 114 patients completed the study [mean (SD) age 60.48 (10.99) years, FEV_1 108.82% (15.13) predicted, 37 male]. Mean ISWT distance was 690 m (152.68). There were no significant differences in walking distance between males and females ($p>0.05$). ISWT distance showed significant correlations with age, BMI, FEV_1, QMVC, DUKE physical activity score and height ($p<0.01$). Stepwise multiple regression analysis showed that age, BMI, FEV_1, QMVC and DUKE physical activity score were independent contributors to the ISWT distance achieved by healthy subjects, explaining 50.4% of the variance. (Abstract S120 figure 1).

Conclusions Variance in the ISWT can be measured using a composite score, comprising of: age, BMI, FEV_1, QMVC and DUKE physical activity score. These findings would allow clinicians to express results of the ISWT as a percentage of the predicted values making results more meaningful for patients with chronic conditions.

Mast cells, smooth muscle and inflammation in asthma

S121 MEDIATOR PROFILING OF SEVERE ASTHMA PHENOTYPES

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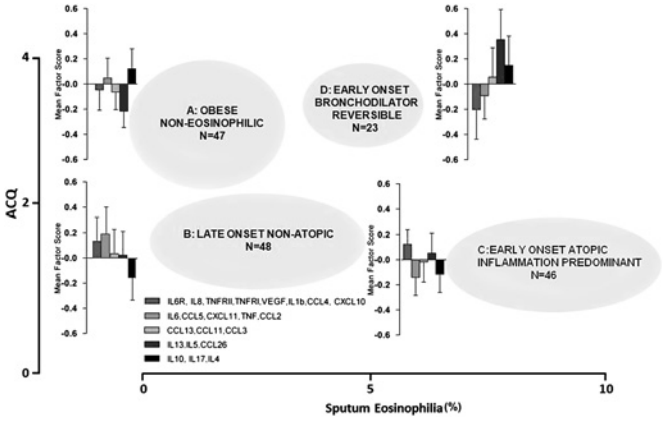
[1]D Desai, [1]C J Newby, [1]P Haldar, [1]S Shah, [1]S Gupta, [1]M Bafadhel, [1]A Singapuri, [1]S Siddiqui, [1]J Woods, [2]A Herath, [2]I K Anderson, [1]P Bradding, [1]R H Green, [1]A J Wardlaw, [1]I D Pavord, [2]R D May, [1]C E Brightling. *[1]Institute for Lung Health, Department of Infection, Immunity & Inflammation, University of Leicester, Leicester, UK; [2]Medimmune Ltd, Milstein Building, Granta Park, Cambridge, UK*

Background Severe asthma is a heterogeneous disease. Defining its phenotypic heterogeneity is likely to shed light upon its immunopathogenesis and direct therapy. We sought to determine the relationship between phenotypes of severe asthma and sputum mediator profiles.

Methods Subjects were recruited from a Difficult Asthma Clinic at a single centre (n=164) and assessments of lung function, atopic status, asthma control and sputum induction were undertaken. Sputum was obtained and supernatants were analysed for 23 mediators using the Meso-Scale Discovery platform. We performed k-means cluster analysis to determine clinical clusters using the baseline characteristics and sputum differential counts. The pattern of mediator expression was determined by factor analysis to identify biological factors. The biological factors were related to the clinical clusters and subjects stratified by asthma control, exacerbation frequency, treatment and sputum cell counts. The repeatability of the individual clinical characteristics and biological mediators was assessed in paired samples in 106 subjects and in three samples in 66 subjects.

Results We identified four clinical clusters and five biological factors. The biological factors were differentially expressed in subjects stratified by sputum cell counts, asthma control and exacerbation frequency, but were not significantly different across the clinical clusters. The within subject repeatability of mediators was moderate; biological factors were consistent and tracked with sputum cell counts for the repeated visits.

Conclusions Sputum mediator profiling of severe asthma revealed repeatable biological factors that were strongly associated with cellular profiles and inform our understanding of asthma phenotypes.



Abstract S121 Figure 1 Relationship between clinical clusters and biological factors. The clinical clusters are plotted in two dimensions with airway inflammation on the x axis and asthma control (ACQ) on the y axis. The size of the ellipsoid represents the number of subjects within each clinical cluster. The distribution of the five biological factors for each cluster is shown as the mean (SEM) factor scores.

S122 SPUTUM CYTOKINE PROFILES IN ASTHMA AND THE IMPACT OF SMOKING-A FACTOR ANALYSIS

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[1]M Spears, [2]C Dewet, [1]C McSharry, [1]R Chaudhuri, [1]I Donnelly, [1]L Jolly, [1]E Cameron, [1]N C Thomson. *[1]Institute of Infection, Immunity & Inflammation, University of Glasgow, Glasgow, UK; [2]NHS Education for Scotland, Glasgow, UK*

Introduction Cigarette smokers with asthma have a distinct clinical phenotype from non-smokers with asthma. This may reflect altered airway inflammation although how cigarette smoking directs this is unclear. We employed exploratory factor analysis to examine the impact of smoking on airway inflammation.

Abstract S122 Table 1 Factor loadings

Rotated component matrix			
	Factor		
	1	2	3
IFN-?	0.991		
IL-4	0.986		
IL-5	0.986		
IL-6		0.864	
CXCL9		0.964	
CXCL10		0.908	
CCL2			0.813
CCL3			0.889
CCL4			0.920