strongly with sputum and blood eosinophil counts, and are most useful when a moving average is taken over approximately 9 days. Further studies are required to determine if daily FeNO measurements may have a role in predicting loss of asthma control or exacerbations.

P70 BLOOD EOSINOPHIL COUNTS IN NORMAL CONTROLS WITH NO HISTORY OF ALLERGIC DISEASE

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Background The beneficial effect of corticosteroids and anti-interleukin (IL)-5 on exacerbations of airway disease becomes apparent at blood eosinophil counts above 0.15×10^9/L, well within the normal range. One potential explanation is that the upper limit of the normal range is artificially high because studies have included patients with allergic disease and eosinophilic inflammation. We have assessed the normal range for blood eosinophil counts in volunteers with no self-reported history of allergic disease and compared this with the findings from a more traditional control population.

Methods We recruited 78 volunteers (15 male) with a mean age of 38.8 years. Volunteers with a self-reported history of asthma, allergic rhinitis and/or eczema were excluded. The differential cell count was carried out using sysmex XN analyser and serum IgE measured using automated enzyme immunoassay by Phadia Immunocap equipment. Result were compared with an unscreened population (n = 120) used to calculate our local normal ranges.

Results One outlier value of 0.79×10^9/L, (>5 SD above the mean) was excluded from further analysis. In the remainder the mean blood eosinophil count was 0.15×10^9/L with an upper limit of normal range of 0.27×10^9/L. Volunteers with no self-reported history of allergic disease but an IgE >120 iu/L and/or positive specific IgE to house dust mites or grass were not statistically different. The mean blood eosinophil count in the laboratory population was 0.19×10^9/L (p 0.018 vs our population) and the upper limit of normal range 0.42×10^9/L.

Conclusions The upper limit of the normal range for blood eosinophil count is lower in a population who have no clinical history of allergic disease.

P71 THE RELATIONSHIP BETWEEN THE LEICESTER COUGH QUESTIONNAIRE, EOSINOPHILIC AIRWAY INFLAMMATION AND ASTHMA PATIENT RELATED OUTCOME MEASURES IN SEVERE ASTHMA

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Background Severe asthma is characterised by a variety of symptoms which include chronic cough, although the mechanisms responsible are poorly understood. Modulation of cough reflex sensitivity by eosinophilic airway inflammation is considered likely but has not been well studied. Likewise the impact of chronic cough on patients health status is not well known as existing asthma patient related outcome instruments such as the Juniper Asthma Control Score (ACQ-6), Asthma Quality of Life Questionnaire (AQLQ) were not primarily designed to capture cough and its related morbidity in asthma. We sought to evaluate (i) the Leicester Cough Questionnaire (LCQ) in a severe asthma population, (ii) the relationship between the Leicester Cough Questionnaire (LCQ) and the ACQ-6, AQLQ and (iii) airway inflammation in sputum in severe asthma patients.

Methods 312 patients [mean (SD) age of 60.4(16.2) years, median (IQR) GINA treatment score 5[4–5] and median (IQR) sputum eosinophil percentage of 3.0(0.5–16.6)] attending the Leicester difficult asthma service were evaluated with the LCQ, ACQ-6 and AQLQ at a single clinical visit. Induced sputum samples were also acquired at the same visit for differential cell count.

Results The LCQ demonstrated the following distribution properties: mean 15.30, standard deviation 4.49, range 4.04–21 and 10^{th} percentile point of 8.52. Domain specific scores were LCQ (physical) 4.8(1.45), LCQ (psychological) 5.30 (1.55) and LCQ (social) 5.19(1.67). There were modest correlations between LCQ and ACQ-6 ($r = -0.60$; $p < 0.001$) but not with AQLQ ($r = -0.067$). There was no correlation between LCQ and sputum eosinophilis/neutrophils.

Discussion Severe asthma is associated with a high degree of cough related morbidity that appears to be independent of eosinophilic airway inflammation and not captured fully by available patient reported outcome instruments. Further research is required to determine the validity of the LCQ and its responsiveness in severe asthma populations.

P72 T2 BIOMARKERS RELATE TO EXACERBATIONS AND CONTROL IN REFRACTORY ASTHMA

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Background T2 biomarkers have been shown to predict responsiveness to corticosteroids and possibly relate to asthma exacerbations and control. T2 biomarkers in the form of fraction exhaled nitric oxide (FeNO), peripheral blood eosinophils (PBE), and serum periostin are easier to measure and probable surrogate markers for induced sputum eosinophilia (ISE). The relationships between PBE, FeNO and periostin particularly in refractory asthma have been conflicting. A composite score of T2 biomarkers has also been postulated to predict exacerbations and may be more sensitive.[1]

Aim To explore the relationship between the T2 biomarkers individually, and in the form of composite score to asthma exacerbations and control.

Methods Unselected consecutive patients with confirmed diagnosis of refractory asthma (ATS) attending a tertiary severe asthma centre were recruited following an informed consent. Participants were evaluated for the followings: demographics, exacerbations requiring corticosteroids in the preceding 12 months, asthma control questionnaire (Juniper ACQ7), lung function, FeNO, PBE, and periostin measurement. The composite T2 score of all the 3 biomarkers was calculated as previously reported (reference). Statistical analyses were conducted using MedCalc software.

Results One-hundred and fifteen patients were recruited with mean age 45 yrs, 88 (69.8%) females, mean inhaled corticosteroids (BDP equivalent) = 1,647 μg/day, on maintenance OCS =