significant reductions ($p < 0.01$) in IF and ACQ within TH. In VC, the ACQ improved at visit 2, but the IF did not.

Abstract P100 Table 1 Study groups and outcome measures

	Control (n = 12)	VC (n = 9)	TH (n = 9)
Sex (M/F)	7/5	4/5	7/2
Mean (SD) age, years	9.0 (2.0)	9.9 (3.3)	9.9 (1.3)
Mean (SD) FEV ₁ % predicted at visit 1	84.2 (19.6)	84.1 (13.9)	91.2 (14.6)
Median (quartiles) incorrect MDI steps at visit 1	2.0 (0; 4.75)	10 (6.5; 10)	6 (5; 9)
Median (quartiles) incorrect MDI steps at visit 2	0.5 (0; 2.75)	1.0 (0; 2.0)	0.0 (0; 0.5)
Mean (SD) peak IF pre-training at visit 1, l/min	46.7 (8.2)	99.1 (55.4)	115.8 (24.1)
Mean (SD) peak IF at visit 2, l/min	75.0 (34.2)	98.9 (65.8)	66.1 (19.0)
Mean (SD) ACQ at visit 1	1.14 (0.59)	2.43 (1.85)	2.39 (1.10)
Mean (SD) ACQ at visit 2	0.74 (0.93)	0.82 (0.64)	0.70 (0.97)

Conclusion VC and TH improved the children's MDI technique which was reflected on better asthma control. VC children could not, however, maintain the acceptable IF through their MDI which is critical for aerosol lung deposition. An inhaler training tool available to patients at any time can be helpful.

Best of science advances

P101 PERIPHERAL BLOOD TYPE 2 INNATE LYMPHOID CELL COUNT IN PATIENTS WITH SEVERE EOSINOPHILIC ASTHMA

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Background A subgroup of patients with severe asthma have persistent eosinophilic airway inflammation despite treatment with high intensity corticosteroid treatment. One possible explanation for this pattern of disease is involvement of a type 2 innate lymphoid cells (ILC2s) dependant and relatively corticosteroid resistant pathway generating type 2 cytokines such as IL-5 and IL-13. The presence of high numbers of ILC2s in the nasal polyps commonly associated with severe eosinophilic asthma supports this view. We have carried out a cross-sectional study testing the hypothesis that ILC2 counts are increased in peripheral blood of patients with severe eosinophilic asthma.

Methods Blood was taken from 9 controls and 33 patients with asthma, 23 of whom met the 2014 ERS/ATS guideline criteria for severe asthma and had historical evidence of eosinophilic airway inflammation as defined before (Pavord *et al.* Lancet 2012;380:651-9). ILC2 were measured as lineage-CD45⁺CD127⁺CRTH2⁺ by flow cytometry and numbers presented as total cell counts and % peripheral blood mononuclear cells.

Results ILC2 counts were repeatable within patients (ICC 0.97; $n = 6$). Mean $\pm$ SD ILC2 counts were 566 ± 379 , 323 ± 224 , 437 ± 628 and 429 ± 421 cells/mL (Figure 1A) and 0.034 ± 0.022 , 0.02 ± 0.017 , 0.020 ± 0.028 and $0.019 \pm 0.014\%$ of total lymphocytes (Figure 1B) in normal controls ($n = 9$), patients with mild to moderate asthma ($n = 10$), patients with

severe asthma at BTS step 4 ($n = 12$), and patients with severe asthma at BTS step 5 ($n = 11$) respectively.

Abstract P101 Figure 1 Comparison of ILC2 counts (A) and proportions in lymphocytes (B) in the blood from healthy control and different asthma patients. ($p = 0.7$ for A and $p = 0.28$ for B)

Conclusion Type 2 innate lymphoid cells are scarce in peripheral blood but can be measured consistently. We found no evidence of increased counts in peripheral blood from patients with severe eosinophilic asthma.

P102 DEVELOPMENT OF A NOVEL ASSAY FOR THE DETECTION OF ACTIVE NEUTROPHIL ELASTASE IN PATIENTS WITH CHRONIC OBSTRUCTIVE PULMONARY DISEASE

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Neutrophil elastase (NE), a biomarker of infection and inflammation, correlates with the severity of several respiratory diseases including chronic obstructive pulmonary disease (COPD). However, its detection and quantification in biological samples is confounded by a lack of reliable and robust methodologies. Standard assays using chromogenic or fluorogenic substrates are not specific when added to complex clinical samples containing multiple proteolytic and hydrolytic enzymes which have the ability to hydrolyse the substrate, thereby resulting in an over-estimation of the target protease. Furthermore, ELISA systems measure total protease levels which can be a mixture of latent, active and protease-inhibitor complexes. Therefore, we have developed a novel immunoassay (ProteaseTag™ Active NE Immunoassay) which is selective and specific for the capture of active NE in sputum and Bronchoalveolar Lavage (BAL) in patients with COPD.

The objective of this study was to clinically validate ProteaseTag™ Active NE Ultra Immunoassay for the detection of NE in sputum from COPD patients.

20 matched sputum sol samples were collected from 10 COPD patients ($M = 6$, $F = 4$; 73 ± 6 years) during stable and exacerbation phases. Samples were assayed for NE activity utilising both ProteaseTag™ Active NE Ultra Immunoassay and a fluorogenic substrate-based kinetic activity assay.

Both assays detected elevated levels of NE in the majority of patients ($n = 7$) during an exacerbation (mean = $217.2 \mu\text{g/ml} \pm 296.6$) compared to their stable phase (mean = $92.37 \mu\text{g/ml} \pm 259.8$). However, statistical analysis did not show this difference to be significant ($p = 0.07$, ProteaseTag™ Active NE Ultra Immunoassay; $p = 0.06$ kinetic assay), which is highly likely to be due to the low study number. A highly significant correlation was found between the 2 assay types ($p \leq 0.0001$, $r = 0.996$).