expression (MFI) of CD25+ cells was then monitored after two days by flow cytometry.

**Results** 1,25(OH)2D3 significantly increased CTLA4 MFI in both healthy and latent populations following stimulation with SEB (p≤0.01) or PPD (P=0.026, 0.008). 25(OH)D3 also enhanced CTLA-4 expression in SEB cultures (p≤0.01). Induction of CTLA-4 was however reduced in PPD cultures (median 121) compared to SEB (median 360). Interestingly, the magnitude of CTLA-4 induction by 1,25(OH)2D3 or 25(OH)D3 also differed for healthy and latent populations in response to SEB (1,25(OH)2D3 (p=0.01) and 25(OH)D3 (p=0.006), with a similar trend in PPD stimulated cells (p=0.092).

**Conclusion** The shift towards a Treg population as a result of vitamin D is blunted in latent TB compared to health. Differential response of memory cells in latent disease could account for this.

Methods We performed a randomised, double-blind trial in 21 mild-to-moderate persistent asthmatics receiving ICS with elevated FeNO (>30ppb) that increased further (>10ppb) after ICS washout. Patients were randomised to 2 weeks of either fluticasone propionate 50µg twice-daily (FP100) or 250µg twice-daily (FP500). The primary outcome was response in diurnal domiciliary FeNO levels. Secondary outcomes included: mannitol challenge; serum eosinophilic cationic protein (ECP); blood eosinophil count; and asthma control questionnaire (ACQ).

Results We found significant dose-related reductions of diurnal FeNO compared to baseline - morning FeNO: baseline=71ppb (95%CI:61–83ppb); FP100=34ppb (95%CI:29–40ppb), p<0.001; FP500=27ppb (95%CI:22–33ppb), p<0.001; and significant dose separation for morning, p<0.05, and evening, p<0.001. Time series FeNO displayed exponential decay (Figure 1): FP100 R²=0.913, half-life=69hrs (95%CI:50–114hrs); FP500 R²=0.966, half-life=55hrs (95%CI:45–69hrs); as well as diurnal variation. ACO showed significant improvements exceeding the minimal important difference (>0.5) with values in keeping with controlled asthma (<0.75) after each dose: FP100=0.48 (95%CI:0.24–0.71), p=0.004; FP500=0.37 (95%CI:0.18–0.57), p=0.001. All other secondary inflammatory related outcomes (mannitol, ECP and eosinophils) showed significant improvements from baseline but no dose separation.

Conclusions There is a significant dose-response of diurnal FeNO to ICS in asthmatics with an elevated FeNO phenotype, which translates into well-controlled asthma. Further interventional studies are warranted using domiciliary FeNO in this specific phenotype.

Abstract S6 Figure 1

Time series morning and evening exhaled tidal nitric oxide (FeNO) values and one-phase exponential decay curves. FeNO values displayed as geometric means at each sequential time point for each group. R² = coefficient of determination (goodness of fit) of exponential decay curves to each data set. t½ = half-life of exponential decay. ppb = parts per billion.

CAN EOSINOPHIL AND NEUTROPHIL MIGRATION BE THE KEY TO PHENOTYPING ASTHMA?

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Introduction To date, our knowledge of in vivo migration of neutrophils and eosinophils in homeostasis and disease states is based on granulocytes. Here we present a pilot study using purified human eosinophils or neutrophils and demonstrate their differential in vivo kinetics in asthmatic and healthy volunteers. Methods: On two separate occasions 100 ml of blood was obtained from eight human