presented as mean (SEM). Data collected included: demography, length of stay (LOS), vital status, initial treatment, full blood counts, renal function and CRP.

Results There were 549 patients, with 768 AECOPD events. The mean (SD) age was 71 years and 192 episodes were associated with an eosinophil count >2% (26.6%). There were 403 (56%) AECOPD episodes leading to admission; there was no difference in the eosinophil count between patients admitted or discharged from ED. Absolute and relative PBE levels were increased in patients re-attending ED (Absolute PBE mean difference 0.08, 95% CI: 0.02 to 0.13, p = 0.007; %PBE mean difference 0.6, 95% CI: 0.14 to 1.1, p = 0.001). Patients with a PBE > 2% were readmitted more often (p = 0.002, RR 1.16, 95% CI: 1.05 to 1.26). For patients admitted, mean LOS was reduced if admission %PBE levels were >2% (4.6 (0.5) vs. 5.8 (0.3) days, p = 0.012). In patients known to have received oral corticosteroids in ED, the reduction in LOS was greater still if the %PBE was >2% compared to ≤2% (mean (SD) 4.1 (1.0) vs. 6.5 (0.9), p = 0.046). In-patient mortality occurred in 35 patients and occurred more frequently in patients with a %PBE ≤ 2% (RR 1.16, 95% CI: 1.04 to 1.68, p = 0.012).

Conclusions This real-world data suggests that PBE levels may be a useful marker for predicting important clinical outcomes in AECOPD.

S69 TROPONIN LEVELS AND RISK OF DEATH FOLLOWING A MYOCARDIAL INFARCTION IN PEOPLE WITH AND WITHOUT COPD

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Introduction Myocardial Infarction (MI) is a common comorbidity and cause of death in people with COPD, and COPD is associated with increased risk of death following acute MI.[1,2] We aimed to: 1) compare levels of peak troponin following MI between people with and without COPD; and 2) investigate differences in the prognostic value of peak troponin between those with and without COPD.

Methods Patients from the Myocardial Ischaemic National Audit Project (MINAP) database who had linked Office of National Statistics (ONS) mortality data from 2003–2013 were included in the study. COPD was defined as the presence of obstructive airway disease and smoking history. We used linear regression to compare levels of peak troponin I and T between people with and without COPD followed by logistic regression to investigate the prognostic value of peak troponin level in predicting 180 day mortality separately for those with and without COPD. All models were adjusted for age, sex, smoking, peripheral vascular disease, cerebrovascular disease, chronic renal failure and previous angina.

Results We included 300,146 patients with a first MI, 34,027 (11.3%) with COPD. Peak troponin T & I was lower for those with COPD following both STEMI (troponin T: 0.51 ng/mL lower, adjusted% 17% lower (95% CI: 6–18%); troponin I: 5.49 ng/mL lower, adjusted% 12% lower (6–18%)) and non-STEMI (troponin T: 0.07 ng/mL lower, adjusted% 20% lower (95% CI: 16–25%); troponin I: 0.34 ng/mL lower, adjusted% 19% lower (15–23%)). The prognostic value of increased peak troponin was higher for COPD patients than those without COPD for troponin T, p-value for interaction = 0.02 (Table 1), but this was not apparent for troponin I p-value for interaction = 0.520.

Abstract S69 Table 1 Risk of death at 180 days after acute MI for those with and without COPD at differing troponin T levels.

Troponin level (ng/mL)	COPD adjusted OR (95% CI)	Non-COPD adjusted OR (95% CI)
<0.01	1 (reference)	1 (reference)
0.01–0.049	1.71 (0.84–3.47)	0.82 (0.62–1.08)
0.05–0.099	1.59 (0.79–3.18)	1.28 (1.00–1.65)
0.1–0.49	2.34 (1.20–4.55)	1.44 (1.34–1.82)
0.5–1.79	2.36 (1.21–4.60)	1.63 (1.29–2.06)
>1.8	2.55 (1.31–4.95)	1.94 (1.54–2.45)

Conclusion Cardiac Troponin T appears a better prognostic indicator for long-term outcome amongst COPD patients following an MI compared to Troponin I. Clinicians should not be reassured by relative lower troponin levels in COPD patients at the time of an MI.

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Infections and the Impact on Childhood Respiratory Diseases

S70 NEONATAL AIRWAY EPITHELIAL CELL IL-8 RESPONSES TO INFECTION ARE REDUCED IN THOSE WHO GO ON TO WHEEZE

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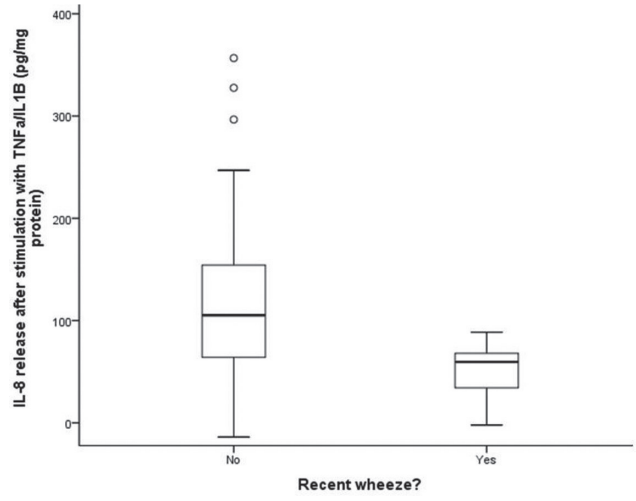
Introduction Airway epithelial cells (AEC) are important contributors to the innate immune system and AEC function in children with asthma differs to that of children without asthma. We recruited a birth cohort to establish whether AEC function was abnormal before the onset of asthma symptoms.

Methods Pregnant mothers were recruited and nasal AEC brushings were collected from neonates within 48 hours of birth. Cells were cultured in a submerged model and stimulated with tumour necrosis factor alpha/interleukin-1 beta (TNFa/IL1b), lipopolysaccharide (LPS) and house dust mite (HDM). The mediators in the culture supernatant were quantified by cytometric bead array or ELISA and included: interleukin (IL)−6, IL-8, granulocyte macrophage colony stimulating factor (GMCSF) and interferon gamma (IFNg). The primary outcome of interest was IL-8 expressed as median [interquartile range] in pg/mg protein. Parents returned a postal questionnaire when their child was four years old.

Results AEC were successfully cultured in 139 neonates of whom 120 were contacted and 85 questionnaires were returned. The mean age was 3.8 years, 42 were boys and 10 reported wheeze in the previous 12 months. Neonates who had recent wheeze at four years had reduced median AEC IL-8 release after exposure to TNFa/IL1b (60 [33, 71] versus 105 [64, 157], p = 0.002, see Figure 1) or to LPS (2.2 [0, 9.0] versus 10.5 [4.6, 24.0] p = 0.038) compared to those who did not wheeze. Neonatal AEC GMCSF

release was also reduced after exposure to TNFa/IL1b in those who later wheezed compared to non wheezers (0.09 [0.01, 0.72] versus 0.67 [0.32, 1.48] p = 0.014). Neonatal AEC release of IFNg and IL-6 were not associated with later wheeze. Maternal asthma was not associated with AEC IL-8 release.

Conclusions These results suggest that an abnormality which is present at birth in AEC response to infection is important to asthma causation.



Abstract S70 Figure 1

S71 DO CHILDREN WITH TROUBLESOME PRESCHOOL WHEEZE HAVE EVIDENCE OF FUNGAL SENSITISATION?

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Introduction Preschool wheeze is very common and can be troublesome necessitating frequent hospital admissions and trials of preventer medications. It has been suggested that fungal sensitisation is associated with severe childhood asthma (Vicencio 2014), however it is unclear when this sensitisation occurs and whether fungal sensitisation is associated with preschool wheeze. We aimed to assess whether children attending our clinic due to preschool wheeze were sensitised to fungi and whether fungal sensitisation is associated with troublesome wheezing.

Methods Preschoolers attending our clinic due to wheezing investigated for allergic sensitisation to a panel of aeroallergens (*D. pteronyssinus*, cat, dog, timothy grass and tree pollen) and fungi were identified. Evidence of sensitisation was determined by a raised specific IgE (sIgE) > 35 kU/L detected by fluorescent enzyme immunoassay. Sensitisation to fungi was determined either by raised sIgE to a 'mould screen' (comprising *Penicillium notatum*, *Cladosporium herbarum*, *Aspergillus fumigatus*, *Candida albicans*, *Alternaria alternata and Helminthosporium halodes*) or to specific fungi including those in the mould screen and *Malassezia spp*. Data on the number of hospital-recorded admissions due to an exacerbation of wheezing within the preceding 12 months and currently prescribed medications, hence BTS treatment step, were collected.

Results Table 1 displays the demographic features of the 51 children identified, the number of children with a raised total IgE and evidence of sensitisation to aeroallergens and fungi. Six (24%) children with a raised total IgE and 4 (31%) sensitised to aeroallergens were sensitised to fungi. Whilst we found a positive correlation between age and serum total IgE (0.364, $r^2 = 0.13$, $p < 0.01$ – Spearman's rank) there was no significant correlation between age and sIgE to the mould screen or specific fungi. We found no significant differences in the age, number of hospital-recorded admissions due to wheeze or BTS treatment step between those preschoolers sensitised and not-sensitised to fungi.

Discussion Our preliminary data suggests that a) few children with preschool wheeze attending clinic have evidence of fungal sensitisation and b) fungal sensitisation does not appear to be associated with hospitalisation frequency or required medications. Further exploration is required to determine, if any, the relationship between fungal sensitisation and preschool wheeze.

Abstract S71 Table 1 Demographic data, evidence of sensitisation and clinical parameters

Male, *n* (%)	30 (59)
Median (range) age in months	39 (8–71)
Serum makers of allergic sensitisation	
Raised total IgE (> 52 kU/L), *n* (%)	25 (49)
Evidence of sensitisation to aeroallergens, *n* (% of those with raised total IgE)	13 (52)
Evidence of sensitisation to fungi, *n* (% of those with raised total IgE)	6 (24)
Evidence of sensitisation to fungi, *n* (% of those sensitised to aeroallergens)	4 (31)*
Number of hospital-recorded admissions due to wheezing in one year	
≥2 admissions (n = 14)	2 (14) †
Number sensitised to fungi, *n* (%)	
<2 admissions (n = 37)	4 (11) †
Number sensitised to fungi, *n* (%)	
BTS treatment step#	
BTS step 3 or above (n = 11)	2 (18) †
Number sensitised to fungi, *n* (%)	
BTS step below 3 (n = 40)	4 (10) †
Number sensitised to fungi, *n* (%)	

*Those children sensitised to aeroallergens were more likely to be sensitised to fungi than those not sensitised to aeroallergens, p<0.05 (Pearson chi-square), †There was no statistically significant difference between the number of children sensitised and not-sensitised to fungi and the number of hospital-recorded admissions or required asthma medications, p>0.05 (Pearson chi-square), #BTS treatment step (BTS/SIGN 2014 guidelines on the management of asthma).

S72 IMMUNOMODULATORY EFFECTS OF CARBON PARTICULATES ON MACROPHAGE HANDLING OF STREPTOCOCCAL INFECTIONS

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Background and objective Airway macrophages are key to a successful immune response to pulmonary infections. Smoke exposure has been previously shown to result in particulate loading of alveolar macrophages and subsequent impaired macrophage responses to infection with *Streptococcus pneumoniae*.[1] Rheumatic heart disease (RHD) is an autoimmune disorder that follows an airway infection with *Streptococcus pyogenes* in some individuals. Little is understood about the aetiological factors predisposing the development of RHD but smoke exposure from combustion of solid fuels has recently been epidemiologically linked to RHD.

In the context of this epidemiological observation, we hypothesised that smoke exposure would affect macrophage handling of