

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 $\leq 2\%$ (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 $\leq 2\%$ (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 (TNF α /IL1 β), 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 (GM-CSF) and interferon gamma (IFN γ). 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 TNF α /IL1 β (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 GM-CSF