significant parametric pairwise t-test ($p \leq 0.05$) and ii) area under the receiver-operator characteristic curve (AUROC ≥ 0.59) were selected for inclusion in a logistic regression model.

Results The biomarkers that met criteria and were taken forward for further analysis are shown in Table 1. Of these CC16, CRP, MMP8 and NGAL combined had an AUROC 0.82 (95% confidence interval 0.74 to 0.90). The Youden's index gave a sensitivity and specificity of 78% and 82% respectively.

Conclusion COPD exacerbations can be identified by urinary biomarkers. The biomarker panel requires further validation in a prospective longitudinal study.

Abstract P185 Table 1 Candidate urine biomarkers of COPD exacerbation short listed from a list of 50 biomarkers

Biomarker	AUROC	p-value
Fibrinogen	0.6395	0.0029
Secretory leukocyte peptidase inhibitor (SLPI)	0.5917	0.0268
Interleukin - 6 (IL-6)	0.6025	0.0325
N-Formyl-methionyl-leucyl-phenylalanine (fMLP)	0.6132	0.0121
Desmosine	0.6210	0.0386
Clara Cell protein (CC16)	0.6405	0.0025
Tissue Inhibitor of Metalloproteinase 1 (TIMP 1)	0.6446	0.0325
Tissue Inhibitor of Metalloproteinase 2 (TIMP 2)	0.6271	0.0287
C-reactive Protein(CRP)	0.6463	0.0002
Chitinase 3 like 1 protein (CH3L1)	0.6172	0.0139
Alpha 1 Anti-Trypsin (A1AT)	0.7240	6.3 E-6
Creatinine	0.6460	0.0009
Beta -2 Microglobulin (B2M)	0.6972	0.0001
Cystatin C	0.6747	0.0098
Matrix Metalloproteinase 8 (MMP8)	0.6003	0.0170
Neutrophil Gelatinase-Associated Lipocalin (NGAL)	0.6169	0.0072

P186

DEMONSTRATING HIGHER DIFFUSION COEFFICIENTS IN PATIENTS WITH EOSINOPHILIC VS. NON-EOSINOPHILIC EXACERBATIONS OF COPD

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Background There is evidence that acute exacerbations of COPD (AECOPD) may be stratified by peripheral blood eosinophil count and that this may allow identification of a pathologically distinct phenotype with clinical value. We have compared lung function measures between patients with eosinophilic and non-eosinophilic AECOPD.

Methods All admissions to an inner London teaching hospital with AECOPD were recorded between 2004 and 2012. The eosinophilic phenotype is defined as a first blood count within 24 hours of admission showing peripheral blood eosinophil count $\geq 2\%$ of white blood cells. The first lung function test recorded, performed in a dedicated-laboratory between admissions, were used in analysis. We used non-parametric statistics (Mann-Witney U) for univariate comparisons, and linear regression for multivariate analyses.

Results There were 2793 admissions with AECOPD recorded, we used only first admissions (1,279). Of the first admissions 1104 (86.3%) have a recorded peripheral blood eosinophil phenotype ($n=331, n=773$) and 821 (64.1%) had a recorded FEV1. Of 821 the mean age was 69, 364 were female and 457 male. Proportion with eosinophilic AECOPD 31.1%. The mean FEV1 was 1.12 L (SD 0.55), mean FEV1% predicted 45.47 (SD 20.46) and Mean FEV1/VC (47.78 SD 15.88) The eosinophilic group had a higher FEV1 (mean 1.26 vs 1.05, $p < 0.001$) and higher percent predicted FEV1 (mean 48.75 vs. 44.00, $p = 0.01$) than those in the non-eosinophilic group. 565 patients had DLCO measured (44.2%). Eosinophilic phenotype was associated with a higher DLCO even after adjusting for FEV1% predicted ($p < 0.001$).

Figure 1 – A comparison of DLCO percent predicted measured in stable state, in patients with eosinophilic vs. non-eosinophilic AECOPD admissions.

Abstract P186 Figure 1

Discussion In this cohort, patients admitted to hospital with acute exacerbations of COPD associated with an eosinophilic phenotype had a higher FEV1 and higher DLCO% predicted. A lower DLCO is more associated with an “emphysematous” than “bronchial” form of COPD. Our Results suggest that the eosinophilic phenotype of AECOPD may be associated with less alveolar and pulmonary capillary damage.

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SEASONALITY OF EOSINOPHILIC AND NON-EOSINOPHILIC EXACERBATIONS OF COPD

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Background Patients presenting with an acute exacerbation of COPD (AECOPD) and a peripheral blood eosinophil count $\geq 2\%$ of the total white cell count have a better response to oral corticosteroids, suggesting that stratification by this biomarker identifies a pathologically distinct phenotype

Abstract P187 Figure 1

and has clinical value.¹ We have tested the hypothesis that the eosinophilic and non-eosinophilic exacerbations of COPD have different seasonality using a dataset of patents admitted to an inner London teaching hospital with AECOPD over 9 years.

Methods All admissions with AECOPD were recorded between 2004 and 2012. We recorded the first blood test result within 24 hours of admission. The month of admission was recorded. Seasons were defined as per figure 1 and the distribution of eosinophilic (blood eosinophil count $\geq 2\%$) and non-eosinophilic events compared.

Results 2793 admissions with AECOPD were recorded, of which 2416 (86.5%) had a FBC result available and 750 (31.0%) were eosinophilic. The mean age of the entire population was 70 with 44.3% female. Eosinophilic admissions had a median age 69.3 of which 40.5% were female. Non-eosinophilic admissions had a median age 70.9 of which 46.9% were female. There were no significant differences between the number of eosinophilic exacerbations across seasons. In contrast, non-eosinophilic exacerbations occurred more commonly in winter compared to summer. The proportion of eosinophilic events was 36.5 vs 28.1% in summer and winter respectively (mean difference 9%; 95% CI 4%–14%; $p=0.003$).

Figure 1 – Admissions via A and E for patients with Acute Exacerbations of COPD between 2004 and 2012 stratified by season and peripheral blood eosinophil percentage.

Discussion Exacerbations of COPD associated with a higher blood eosinophil count do not vary according to season whereas non-eosinophilic exacerbations occur more commonly in winter and account for a significantly higher proportion of winter events.

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P188

A FEASIBILITY STUDY OF SALIVARY PEPSIN MEASUREMENT TO ASSESS AIRWAYS REFLUX IN EXACERBATING COPD PATIENTS

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Introduction Chronic Obstructive Pulmonary Disease (COPD) patients experience dyspnoea and cough and are prone to episodes of rapid worsening, termed acute exacerbations (AECOPD). Infections, air pollution and gastroesophageal reflux (GOR) have all been implicated in AECOPD.¹ Identification of GOR during AECOPD may allow targeted treatment. However, confirmation of GOR requires invasive procedures (e.g., pH manometry). Salivary pepsin measurement using Peptest (RDBiomed Ltd) offers a non-invasive alternative. We investigated the feasibility of using Peptest to detect salivary pepsin as a biomarker in AECOPD.

Methods 30 consecutive patients admitted within the last 24 hours with AECOPD were recruited and saliva collected daily for 7 days using a standard method. Pepsin was measured using Peptest. GOR was considered present if pepsin was ≥ 75 ng/ml. Participants completed the following questionnaires on day 1: Frequency Scale for Symptoms of GORD, Hull Airways Reflux Questionnaire and COPD assessment test. Additional demographic and clinical data were collected. Data are presented as mean $\pm$ SD.

Results Thirty patients (Males: 18, Age: 64.1 ± 12.6 , current smokers: 18) were recruited over 30 days. 81% of eligible patients consented. 70% completed the study. Salivary pepsin levels over 7 days are presented in figure 1. 24/30 participants (80%) had salivary pepsin ≥ 75 ng/ml on day 1. Mean pepsin levels reduced significantly during the 7 days with highest levels observed on day 1 ($p < 0.01$). There was no correlation

Abstract P188 Figure 1 Mean salivary pepsin over 7 days with standard error bars.

between day 1 salivary pepsin and symptom questionnaires. Salivary pepsin measurement over 7 days identified four patterns: persistently low ($n=3$); persistently high ($n=3$); high on day 1 only ($n=6$); and variable ($n=13$).

Discussion Peptest was acceptable to patients. Salivary pepsin measurement appears to be able to categorise different population groups by pattern of pepsin concentration throughout the week. As a feasibility study, it was not powered to identify correlation with clinical outcomes. Our Results suggest that using salivary pepsin as a marker of GOR is feasible and is worthy of further study in a large prospective cohort to evaluate its relationship to outcomes.

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P189

REDUCING READMISSION IN HIGH RISK COPD PATIENTS

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Introduction and Objectives COPD patients with acute exacerbations make up a significant proportion of the inpatient hospital population. This patient group is at risk of recurrent readmission to hospital, which not only reduces patient quality of life but also has financial implications. The aim of this study was to correctly identify COPD patients at high risk of readmission and reduce this by addressing reversible factors and/or creating an anticipatory care plan (ACP).

Methods 200 consecutive admissions with an acute exacerbation of COPD were reviewed to identify risk factors for readmission at 28 days or mortality within 6 months. Patients who were identified as high risk of readmission were reviewed using an algorithm to identify potential reversible factors and if ACP would be appropriate. This review was carried out either during admission or at the point of discharge.

Results The following factors were shown to predict readmission or death, recurrent admissions (≥ 2 in the last year) (readmission: $p=0.002$, $OR=3.56$, death: $p<0.001$, $OR=11.64$), NIV during admission (readmission: $p=0.073$, $OR=4.06$, death: $p=0.023$, $OR=7.17$) and long term oxygen therapy (readmission: $p=0.034$, $OR=3.23$, death: $p<0.001$, $OR=9.22$). 72 subjects were identified as having a high risk of readmission. Of these 72 subjects, 14 had the algorithm applied during admission (prospectively) and 58 at the point of discharge (retrospectively). 53% of the study population was readmitted within 28 days. 21% of the prospective group was readmitted compared to 60% of the retrospective group.

55% of the prospective group had an ACP in place compared to 32% of the retrospective group.

Conclusions By identifying characteristics associated with high risk of readmission in patients with COPD, we were able to apply an algorithm in the hope of reducing readmission. Although current numbers are small, initial Results indicate that the use of the algorithm during a hospital admission could significantly reduce readmission to hospital. Undoubtedly more data is required. In the future routine use of this proactive approach could potentially improve patient care as well as reduce the financial burden for healthcare providers.

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SPECIALIST EMERGENCY CARE AND COPD OUTCOMES

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Introduction On 16/06/15 the Northumbria Specialist Emergency Care Hospital (NSECH) opened, introducing 24/7 speciality consultant on-call, direct transfer from the emergency department to speciality wards and 7 day consultant review. A Respiratory support unit opened for non-invasive ventilation (NIV), with enhanced staffing ratios. Pre-NSECH the NIV service included mandated training and competency assessment, 24/7 single point of access, initiation of ventilation in the Emergency Department, a door-to-mask time target, early titration of pressures, and structured weaning. Pneumonia or hypercapnic coma complicating ECOPD is not considered a contra-indication to NIV. Post-NSECH staff-patient ratios increased, the NIV pathway was streamlined and structured review introduced. The NCEPOD 2015 enquiry and 2013 BTS NIV audit showed $\geq 34\%$ of patients receiving acute NIV died.

Methods Patients hospitalised with ECOPD between 1/1/13 and 31/12/16 were identified using ICD10 J44 codes. Ventilation status was confirmed from rolling audit data, combined with a coding search (J96) and verification from patient records. Age, gender, admission from nursing home, consolidation, Charlson index, key comorbidities, length of stay and inpatient and 30 day mortality were captured. Population characteristics and outcomes were compared pre- and post-NSECH. Independent predictors of mortality were identified by logistic regression. Inpatient and 30 day mortality, adjusted for baseline performance and prognostic indices, was plotted (VLAD: Variable Life Adjusted Display).

Results 6291 patients were identified. Pre- and post-NSECH, demographic and clinical indices were similar. Among ventilated patients, 96.5% and 98% received NIV respectively. Inpatient plus 30 day mortality was lower post-NSECH for the whole cohort, and for ventilated and non-ventilated sub-groups. Independent predictors of mortality in a) the whole cohort were: NSECH [Beta=0.64; $p=0.0001$], age, admission from nursing home and Charlson Index; and b) in ventilated patients were: NSECH [Beta=0.51; $p=0.0016$], age and male gender. The VLAD plot showed sustained improvement in observed/expected mortality post-NSECH. Post-NSECH median length of stay fell by one day in both sub-groups.

Conclusions Introduction of 24/7 specialist emergency care was associated with a substantial fall in ECOPD mortality from strong baseline performance. Improved outcome was not

Correction: *P187 Seasonality of eosinophilic and non-eosinophilic exacerbations of copd*

Barker R, Shrimanker R, Russell R, *et al.* P187 Seasonality of eosinophilic and non-eosinophilic exacerbations of copd. *Thorax* 2017;72:A183-A185. doi:10.1136/thoraxjnl-2017-210983.329

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