guiding antibiotic prescribing. Our service evaluation showed that despite this, introducing it into a hospital setting is challenging. If the algorithm had been followed correctly 65% of patients would not have received antibiotics. Physicians often rely on clinical judgement and well-known markers of infections such as CRP and WCC. Education of physicians along with timely availability of the test play significant roles. We intend to persevere and report our results aiming to improve the use of PCT within our hospital.

P114 C-REACTIVE PROTEIN AT ONSET PREDICTS SYMPTOM RESOLUTION IN ACUTE EXACERBATIONS OF COPD

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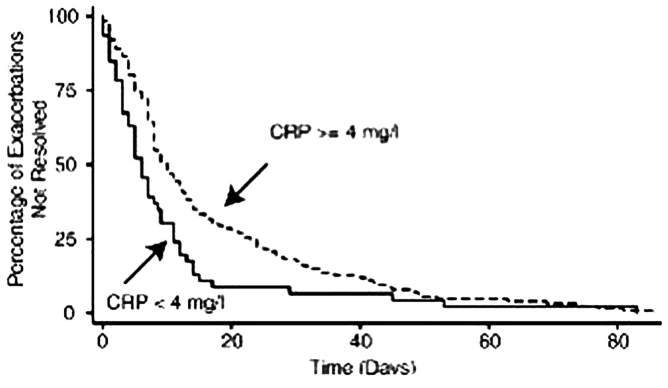
Introduction Patients with chronic obstructive pulmonary disease (COPD) may not recover swiftly from exacerbations. In 23% of patients, symptoms do not recover within 35 days.[1] It would be valuable for clinicians to have a systemic biomarker that taken at initial consultation for an exacerbation could predict exacerbation length. We hypothesised that inflammation, measured by serum C-reactive protein (CRP) predicts the time to symptomatic resolution.
Methods We analysed daily diary cards prospectively collected from the London COPD cohort between October 1995 and January 2010. All patients had an $FEV_1 \leq 80\%$ predicted and an FEV_1/FVC ratio $\leq 70\%$. An exacerbation was defined as an increase for 2 consecutive days in respiratory symptoms, with at least one major symptom (dyspnoea, sputum purulence or volume) plus another major or minor symptom (wheeze, cold sore throat and cough). Exacerbations were separated by five symptom free days. Patients were assessed and sampled at clinic visits within a median of 2 days of exacerbation onset. Exacerbation length was defined as the number of days from onset that increased airway symptoms were still recorded, and resolution time was defined as the time from clinic assessment to symptom cessation.
Results 268 patients with ≥365 days of diary card data experienced at least one exacerbation. Median baseline CRP was 4 mg/l. CRP at onset was available for 172 recovered exacerbations (Abstract P114 Figure 1). Mean age was 68.0 years (SD 8.3), with mean FEV_1 1.11 l (0.45), FEV_1 predicted 44.6% (16.2). Patients with a CRP<4 mg/l at initial clinic review had a significantly shorter resolution time than patients with CRP≥4 mg/l, median 6 (IQR 3-11; n=46) vs 10 (5–23; n=126) days, p=0.0017. Cox proportional hazard risk models with adjustment for repeated measures showed that CRP predicted both exacerbation length (p=0.039) and resolution time (p=0.029).
Conclusions COPD exacerbations can occur without an elevated systemic inflammatory response. These events are associated with a short resolution time. Thus, using CRP as a biomarker, it may be possible to identify patients that have a short recovery period and potentially may not require systemic treatment for their exacerbation.



Abstract P114 Figure 1 Kaplan–Meier plot of symptom resolution by high and low CRP at exacerbation onset.

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P115 VITAMIN D BINDING PROTEIN IN COPD EXACERBATIONS

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Introduction Vitamin D binding protein (DBP) plays a role in macrophage activation and neutrophil chemotaxis, both functions being relevant to COPD and airway infection. We have shown previously that DBP is present in the COPD lung and relates directly to macrophage activation.[1] Transcription of DBP is stimulated by inflammatory cytokines,[2] which tend to be raised in exacerbations. DBP is also cleaved from its binding site on neutrophils by the action of neutrophil elastase.[3] We reasoned that DBP would be elevated in the lung and systemic circulation during exacerbations of COPD compared to the stable state.
Methods 20 patients with a known diagnosis of COPD were studied in the stable state and at the start of an exacerbation. Circulating CRP, elastase, AAT and DBP were measured at each time point, together with sol phase DBP, AAT and CRP, where sufficient sample was available (n=9). All clinical parameters were studied in the stable state only. Relationships between exacerbation and stable state were sought using pair-wise tests for each individual, whilst group relationships between DBP, CRP and elastase were sought using Spearman's rank correlation.
Results No significant differences between stable state and exacerbation were seen for circulating or sol phase DBP (both p>0.4). Circulating CRP and AAT were significantly higher in exacerbation (mean difference 3.95, p<0.0001 and 137.39, p=0.03 respectively), as were sol phase CRP and AAT (mean difference 18.29, p=0.003 and 0.60, p=0.009 respectively). DBP did not relate to elastase in either stable state (p=0.61) or exacerbation (p=0.17), nor to CRP (p=0.71 and 0.98, respectively).
Conclusions The level of local and systemic inflammation, with consequent elastase release, during a COPD exacerbation is insufficient to alter DBP levels. As such it is unlikely that DBP-derived macrophage activation is important in their pathogenesis or resolution.

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P116 SERUM PARC (CCL18) AND EXACERBATION FREQUENCY IN COPD

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Introduction Patients with Chronic Obstructive Pulmonary Disease (COPD) experience intermittent exacerbations. The frequency of exacerbations varies amongst patients but the 'Frequent Exacerbator' appears to be an independent disease phenotype. As frequent exacerbations are associated with more rapid lung function decline, a validated biomarker is needed to identify those susceptible, prompting rigorous medical treatment and aiding selective