has been associated with an adverse prognosis in cardiac and respiratory disease, including COPD.[1] We have assessed its value in AECOPD and whether adding RDW improves the predictive power of the DECAF score.[2]

Methods We studied 2 groups of patients with AECOPD, the "derivation cohort" (n = 920) in whom DECAF was derived[2] and the "internal validation cohort" (n = 880) in whom its prognostic value was confirmed.[3]

In the validation cohort RDW was collected prospectively and relationships to mortality assessed by univariate and multivariate logistic regression. RDW values were dichotomised by visual inspection of the receiver operator characteristic (ROC) curve which showed the optimal prognostic threshold for hospital mortality to be 15.5%, consistent with other studies.[1] "RDW score" (15.5% or less=0, greater than 15.5%=1) was added to the DECAF score and the areas under the ROC (AUROC) curves for the DECAF and DECAF-RDW scores were compared by the method of DeLong.

In the derivation cohort RDW was collected from laboratory records and the prognostic utility assessed separately by logistic regression.

Results In the validation cohort RDW >15.5% was a strong predictor of inpatient mortality in both univariate (OR 2.70, 95% CI 1.68–4.32, p < 0.001) and multivariate analysis (OR 2.16, 95% CI 1.28–3.6 4, p = 0.004). However, there was no difference between the AUROC curves for the DECAF and DECAF--RDW scores (Figure 1; p = 0.63).

In the derivation cohort RDW >15.5% showed a non-significant trend towards higher inpatient mortality on univariate analysis (OR 1.55, 95% CI 0.96–2.50, p = 0.07), but there was no association on multivariate analysis (OR 1.05, 95% CI 0.60–1.84, p = 0.86).

Discussion The significant association of RDW with inpatient mortality in AECOPD in one cohort but not the other suggests limited value in this population. When forced into the DECAF model, RDW did not improve its predictive power and is a weaker prognostic index than the component parts of DECAF.

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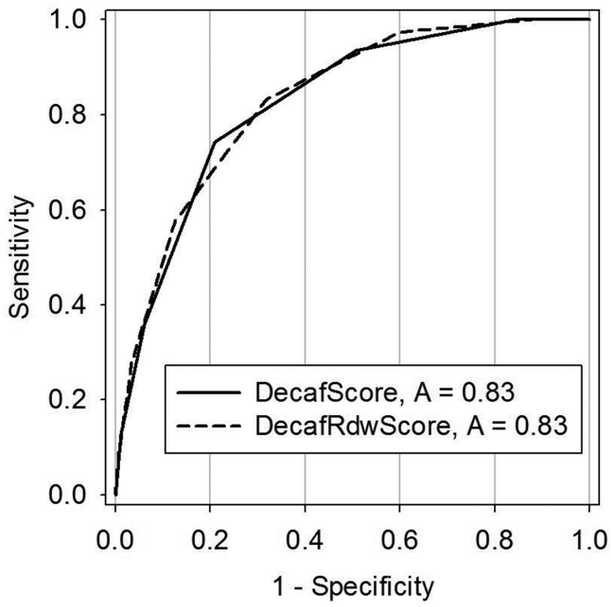
2 Steer *Thorax* 2012
3 Echevarria *Thorax* 2013(68:A138)



Abstract S30 Figure 1

S31 PREDICTING DEATH OR DETERIORATION IN PATIENTS ADMITTED WITH ACUTE EXACERBATION OF COPD USING PHYSIOLOGICAL AND BLOOD PARAMETERS

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Introduction and objectives A number of clinical prediction rules have been described to predict adverse outcomes in patients admitted to hospital with an acute exacerbation of COPD (AECOPD).[1] None are used routinely, perhaps because of limitations including setting (confined to intensive care), use of subjectively defined or difficult to access clinical measurements and lack of external validation. None have undergone impact assessment.

The National Early Warning Score (NEWS) in unselected medical admissions accurately predicts risk of in-patient mortality. The NEWS is less discriminating in patients with COPD. We hypothesised that patients admitted with an AECOPD could be more accurately risk stratified based on a combination of the NEWS and other parameters.

Methods This was a twin site observational cohort study, over a two-year period (March 2012 – February 2014). 2361 admissions with COPD were identified (J40–44).

Results 123 died during admission (5.2%) and a further 36 (1.5%) were escalated to Intensive Care (ICU) and survived to discharge. We analysed these 159 patients against a control group (n = 159) matched only for month of admission (to address seasonal fluctuations in disease severity).

Major results of the study are summarised in Table 1. Those who died or had care escalated were older, had a higher NEWS and respiratory rate. Neutrophils, lymphocyte count, neutrophil-lymphocyte ratio, urea, albumin and CRP were significantly different between the two groups studied. On multivariable analysis lymphocyte count, urea, NEWS and age were independent predictors of adverse outcome.

Abstract S31 Table 1 Admission parameters for patients with AECOPD - comparison between those who died or had care escalated versus those who remained on the ward and survived to discharge. Results given as Mean (Standard deviation). NLR = neutrophil-lymphocyte ratio, NEWS = National Early Warning Score

	Died / escalated (n=159)	Control group (n=159)	T-test or Mann-Whitney U test*
Neutrophils ($x10^9$/L)	12 (6.5)	9.9 (5.3)	0.001
Lymphocytes ($x10^9$/L)	1.0 (0.6)	1.5 (0.9)	<0.0001
NLR	17.2 (18.8)	9.7 (9.4)	<0.0001
Sodium (mmol/L)	136.6 (7.3)	136.7 (4.7)	0.913
Albumin (g/L)	35.0 (5.7)	36.7 (4.2)	0.003
Urea (mmol/L)	10.3 (6.8)	6.9 (4.2)	<0.001
CRP (mg/L)	84.0 (90.4)	53.4 (63.8)	0.001*
Age	76.1 (10.8)	71.8 (10.4)	<0.0001*
NEWS	6.2 (3.2)	4.1 (2.6)	<0.0001*
Respiratory Rate (per minute)	22.1 (5.3)	20.6 (4)	0.011

Conclusions Age, admission NEWS and blood parameters differed significantly between those who were managed on the ward with AECOPD and those who either died or whose care was escalated to ICU. This could form the basis for a prediction score, automatically calculable on admission to hospital using available technology to highlight those patients judged at greatest risk of deterioration.

REFERENCE

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S32 THE RELATIONSHIP BETWEEN EXERCISE CAPACITY AND INFLAMMATORY MARKERS AT COPD EXACERBATION

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Introduction Chronic obstructive pulmonary disease (COPD) is characterised by breathlessness, fatigue and reduced daily activity which worsens acutely at exacerbation. A three year observational study has shown a reduction in 6MWT over time that correlates with increase over the same period in plasma Interleukin-6 and C-reactive protein (CRP) levels (Ferrari, Tanni *et al*. 2013). We therefore investigated whether acute changes in 6MWT at exacerbation were associated with changes in systemic inflammatory markers and the perception of fatigue.

Methods Forty four patients from the London COPD cohort who had a mean age of (±SD) 71(±7) years; FEV_1 52(±17)% predicted; male gender 72% and still smoking 30% were asked to performed a 6MWT and completed a FACIT-F questionnaire when stable (baseline) and 3 days after first presenting with the exacerbation. Blood was drawn for assay of CRP and fibrinogen. 6MWT was performed according to ATS protocols. Exacerbations were defined by our usual symptomatic criteria (Seemungal, Donaldson *et al*. 1998). High scores in the FACIT-F questionnaire indicate low fatigue. Stable COPD was defined as having no exacerbations in the preceding six weeks or subsequent two weeks. Data was analysed by paired t-test, Wilcoxon sign rank test and Spearman correlation.

Results The 6MWT was significantly lower at 3 day post exacerbation compared to baseline measurements [414(SD±111) vs 359(SD±1222) metres; $p \leq 0.001$] and fatigue was worse [37 (9.3) vs 35(9.1); $p = 0.037$]. Inflammatory markers were significantly higher at the exacerbation recovery visit compared to stable state, CRP [median (IQR)] [3.0 (1–8) vs 8.0(3–37) mg/L; $p < 0.001$] and fibrinogen [3.5 (3–4) vs 4.3 (3–5) g/l; $p = 0.003$)].

The fall in exercise capacity from baseline to exacerbation recovery visit was positively correlated with greater increases in CRP [rho= -0.41; $p = 0.021$] (Figure 1A) and in fibrinogen [rho= -0.42, $p = 0.025$] (Figure 1B). Also, the falls in exercise capacity between baseline and exacerbation were associated with increased in fatigue levels [$r = 0.44$; $p = 0.013$] (Figure 1C).

Conclusions These findings suggest that changes in inflammatory markers and other metabolites in the body at exacerbation altering the perception of fatigue and reducing the patient exercise capacity.

S33 SPUTUM COLOUR IN THE LIGHT OF THE HEALTH RELATED QUALITY OF LIFE, AIRWAYS AND SYSTEMIC BIOMARKERS IN EXACERBATIONS OF COPD

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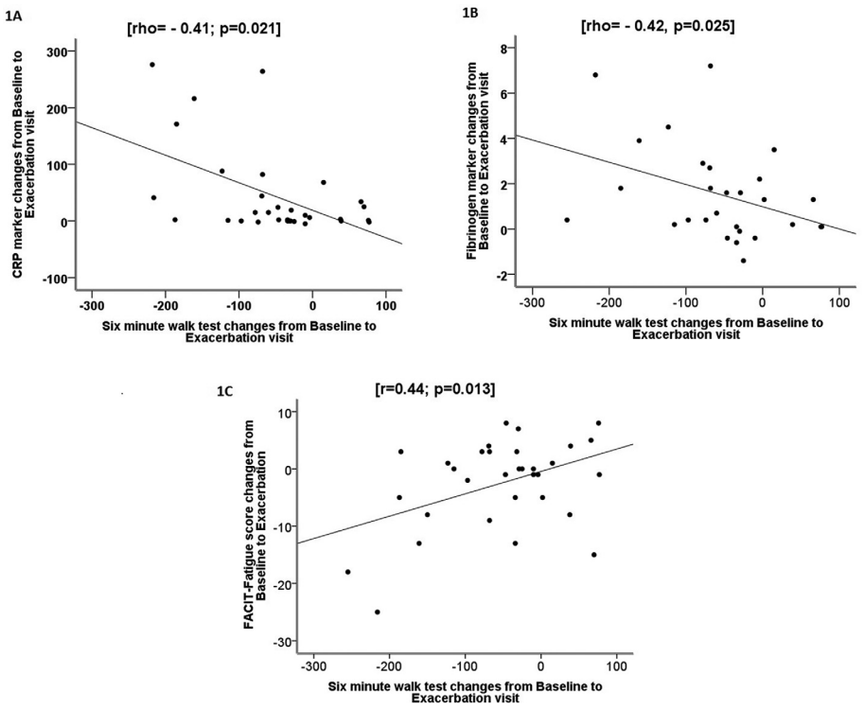
10.1136/thoraxjnl-2014-206260.39



Abstract S32 Figure 1 The correlation between six minute walk test (6MWT) and inflammatory markers (1A) CRP, (1B) fibrinogen and (1C) fatigue