Abstract S28 Table 1 Change from baseline at 32 weeks

	Liraglutide 3.0 mg n = 180 Observed means (LOCF)	Placebo n = 179 Observed means (LOCF)	*p*-value
AHI[3] (events/h)	−12.2	−6.1	p = 0.0150[1]
Oxygen desaturation			
≥4% index (events/h)	−9.5	−5.1	p = 0.0608[1]
Total sleep time (min)	20.7	18.5	p = 0.1629[1]
Wake time after			
sleep onset (%)	−4.0	−3.7	p = 0.0994[1]
Body weight (%)	−5.7	−1.6	p < 0.0001[1]
≥5% body weight			
loss (%)	46.4	18.1	p < 0.0001[2]
>10% body weight			
loss (%)	22.4	1.5	p < 0l.0001[2]
HbA_{1c} (%)	−0.4	−0.2	p < 0.0001[1]
SBP (mmHg)	−3.4	0.4	p = 0.0003[1]

[1]ANCOVA model
[2]Logistic regression model
[3]Definitions of apnoea and hypopnoea from the 2007 AASM Manual for the Scoring of Sleep and Associated Events were used

3.0 mg produced significantly greater weight loss compared with placebo (Table) and enabled more individuals to reach ≥5% and >10% weight loss targets after 32 weeks (p < 0.0001, both). Oxygen saturation, polysomnographic measures, HbA_{1c} and systolic blood pressure (SBP) at 32 weeks are summarised (Table). Nausea and diarrhoea were the most common adverse events with liraglutide 3.0 mg (27% and 17% of individuals, respectively).

Discussion Liraglutide 3.0 mg produced significantly greater reductions than placebo in AHI, body weight, SBP and HbA_{1c} in obese individuals with moderate/severe OSA and was generally well tolerated.

'Blood and spit' – what to measure in AECOPD

S29 PROGNOSTIC VALUE OF PLATELET COUNT IN PATIENTS ADMITTED WITH AN ACUTE EXACERBATION OF COPD (AECOPD)

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Introduction In an observational cohort of patients admitted with AECOPD, thrombocytosis was associated with inpatient and 1-year mortality.[1] We aimed to validate, and explore mechanisms for, this association within our original DECAF cohort (n = 920).[2]

Methods Admission platelet counts were categorised as low (<150), normal (150–400), or high (>400) $x10^9$ cells/mm^3 and odds ratios assessed for inpatient and, among those surviving to discharge, 1-year mortality (normal platelet count=reference). For inpatient mortality, platelet category and DECAF indices were included in multivariate logistic regression. The areas under the ROC curves for DECAF and DECAF+Platelets were compared by the method of DeLong. Associations with thrombocytosis were analysed using Mann-Whitney or Fisher's exact test. Causes of death at 1-year due to respiratory, cardiac or malignant disease were recorded.

Results Thrombocytosis was associated with inpatient (OR 1.83, 95% CI 1.12–3.00, p = 0.016) and 1-year mortality (OR 1.62 95% CI 1.09–2.30, p = 0.017). Thrombocytopenia was associated with inpatient (OR 3.5, 95% CI 1.51–8.12, p = 0.004), but not 1-year mortality (OR 1.81, 95% CI 0.76–4.312.08, p = 0.181). On multivariate analysis, thrombocytosis (OR 1.85, 95% CI 1.03–3.33 p = 0.039) and thrombocytopenia (OR 3.00 95% CI 1.09–8.24 p = 0.033) independently predicted inpatient mortality, but did not improve predictive power of DECAF (AUROC: DECAF=0.86, DECAF+Platelets=0.86; p = 0.93).

Thrombocytosis was associated with a higher white cell count (p<0.001) and eMRCD score (i.e. more breathless when stable; p = 0.001), lower: albumin (p = 0.004), BMI (p = 0.002), FEV1 (p = 0.010), haemoglobin (p<0.001), and a lower proportion of women (p = 0.004), and patients with eosinopenia (<0.05 x 10^9/l) (p = 0.008), cardiac death (p = 0.044), current smoking (p = 0.046), AF (p = 0.029) and diabetes (p = 0.006). Thrombocytosis was not related to cardiovascular disease, prior exacerbation and readmission rates or LTOT use, admission PaO_2, pH or NIV, or length of stay.

Discussion Thrombocytosis was an independent predictor of both inpatient mortality and, amongst survivors to discharge, 1-year mortality. Thrombocytosis was not associated with cardiovascular disease and the higher 1-year mortality was not due excess cardiovascular or cancer deaths, suggesting that other mechanisms are responsible. Whilst thrombocytosis was not associated with LTOT use or PaO_2, it was associated with other indices of disease severity, including breathlessness and lower FEV1, BMI and albumin level.

REFERENCES

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2 Steer *Thorax* 2012

S30 RED CELL DISTRIBUTION WIDTH AS A PREDICTOR OF HOSPITAL MORTALITY IN ACUTE EXACERBATIONS OF COPD (AECOPD)

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Introduction An increased red cell distribution width (RDW), a routinely available index of the variability of erythrocyte size,

Abstract S29 Table 1 Platelet category and cause of death

Platelet count ($x10^9$ cells/mm^3)	Total patients	Inpatient deaths, n (% of total)	Deaths at 1 year, n (% of total)	Respiratory deaths, n (% of all deaths at 1 year)	Cardiovascular deaths, n (% of all deaths at 1 year)	Cancer deaths, n (% of all deaths at 1 year)
		8	16	13	2	1
<150	32	25.0	50.0	81.3	12.5	6.3
		62	203	153	24	15
150–400	713	8.7	28.5	75.4	11.8	7.4
		26	72	61	3	5
>400	175	14.9	41.1	84.7	4.2	6.9

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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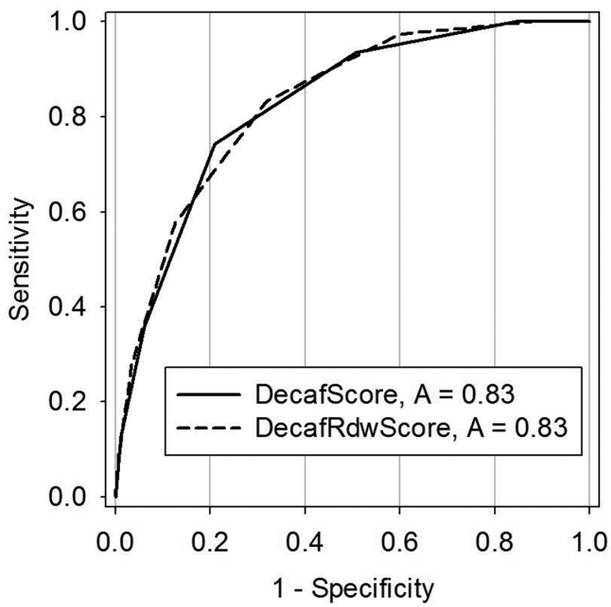

Abstract S30 Figure 1

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