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 < 0I.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

1 Harrison *Thorax* 2014
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