were no cannabis-only smokers and only 6/62 (9.7%) were never-smokers (tobacco&cannabis).

56/62 (90%) abnormal HRCTs were in ex/current tobacco smokers and 27/62 (44%) were in current tobacco&cannabis smokers. There was a higher prevalence of pneumothoraces and bullae with a cannabis and tobacco smoking history than for tobacco alone but this was not statistically significant different (chi-squared STATA) (Table1).

Conclusion More than half of tobacco smokers with abnormal HRCTs also had a history of previous and current cannabis smoking. Despite these findings 25% of patients with abnormal HRCTs had no documentation regarding cannabis smoking. This population of ≤50 years olds with abnormal HRCTs did not smoke cannabis without tobacco. While not statistically significant, bullae and pneumothoraces were more frequently observed in patients who smoked tobacco with cannabis compared to tobacco alone. Larger studies are needed to further understand the additive effect of cannabis smoking to tobacco-induced lung damage. These studies will require systematic recording of both tobacco and cannabis smoking histories.

Abstract P127 Table 1. The association between specific CT findings and tobacco smoking history (without a cannabis smoking history), compared to cannabis and tobacco smoking history.

	Emphysema (%)	Previous/current pneumothorax (%)	Bullae (%)	>1 abnormality (%)
Previous/current cannabis smoking history and previous/current tobacco smoking history (n = 43)	**29/43** (67.4)	**19/43** (44.1)	**25/43** (58.1)	**24/43** (55.8)
Previous/current tobacco smoking history with no previous/current cannabis smoking history (n = 13)	**10/13** (76.9)	**4/13** (30.8)	**5/13** (38.5)	**5/13** (38.5)

COPD exacerbations: the heart of the matter

P128 CARDIOVASCULAR EFFECTS OF STATIN THERAPY ON ARTERIAL STIFFNESS IN PATIENTS WITH COPD: A DOUBLE BLIND RANDOMISED CONTROLLED TRIAL

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Background As cardiovascular disease is a comorbidity and major cause of death in patients with COPD, primary preventative strategies are required. Arterial stiffness, as measured by aortic pulse wave velocity (PWV) is increased in patients with COPD[1], and is an independent predictor of cardiovascular risk[2], which is modifiable over the short term. We hypothesised that aortic PWV, would be reduced by six weeks treatment of simvastatin 20mg once daily compared to placebo in selected patients with COPD without concurrent heart disease, diabetes or hypercholesterolemia.

Methods Clinically stable patients with confirmed COPD were recruited and randomised to either simvastatin 20mg od (active) or placebo in double blinded fashion. Aortic PWV, blood pressure, spirometry, six minute walking distance, and lipids were measured pre- and post- 6 weeks treatment. Primary analysis compared PWV between groups. A predefined subgroup analysis compared those with a baseline PWV≥10m/s.

Results The patients were well matched for age, sex, smoking and lung function; active, n = 33 and placebo, n = 37. The recruitment target was met. Compliance was high with the active group achieving significantly lower total cholesterol - between arms mean (95% CI): -1.1 (-1.3, -0.8)mmol/L, p < 0.001. There was no significant change in aortic PWV after treatment in the active compared to placebo group: -0.7 (-1.8, 0.5)m/s, p = 0.24, In the subgroup with aortic PWV≥10m/s, n = 22, aortic PWV improved in the active arm compared to placebo: -2.8 (-5.2, -0.3)m/s, p = 0.03. This latter difference remained statistically significant after adjusting for age and sex. Blood pressure, lung function and six minute walking distance did not change.

Conclusions In this pilot study, despite a significant reduction in total cholesterol there was no improvement in aortic PWV in patients with COPD taking simvastatin 20mg compared to placebo over 6 weeks. The positive findings in the subgroup with a higher baseline aortic PWV warrants further studies in high risk patients to confirm the impact of statin use on the cardiovascular outcome of COPD.

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P129 SKELETAL MUSCLE WEAKNESS, NOT ARTERIAL STIFFNESS, DIFFERS ACCORDING TO GOLD GROUP IN COPD

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Introduction In the GOLD 2011 COPD classification, patients in Group D are considered to be at highest risk of mortality, due to airflow limitation and exacerbations and also to be most symptomatic. Skeletal muscle weakness and cardiovascular disease are associated with mortality in patients with COPD and elderly people *per se*. We hypothesised that GOLD D patients would have more severe extra-pulmonary manifestations of COPD than A-C.

Objectives To measure skeletal muscle weakness and arterial stiffness (an independent predictor of cardiovascular risk) in the ERICA (Evaluating the Role of Inflammation in Chronic Airways

Abstract P129 Table 1. Results are mean values (SD), except for gender. For 1-way ANOVA, p<0.05 is significant. Superscript letters indicate significant difference(s) between groups.

Variable	Group A (N=25)	Group B (N=130)	Group C (N=14)	Group D (N=226)	1-way ANOVA (p-value)
Gender Male (%)	19 (76)	79 (61)	9 (64)	129 (56)	0.300
Age (Mean years)	70 (7)	68 (8)	67 (7)	67 (8)	0.070
BMI (kg/m^2)	26.9 (4.1)	28.1 (5.1)^{C}	24.2 (3.6)^{B}	26.8 (5.7)	0.030
FEV_1% predicted	67.6 (8.7)^{C,D}	64.0 (8.5)^{C,D}	47.3 (12.8)^{A,B}	47.3 (15.5)^{A,B}	<0.001
FEV_1 (L)	1.7 (0.4)^{C,D}	1.6 (0.5)^{C,D}	1.3 (0.3)^{A,B}	1.3 (1.9)^{A,B}	<0.001
Smoking pack years	45 (24)	47 (25)	34 (16)	48 (27)	0.200
Fibrinogen (g/L)	3.4 (0.9)	3.33 (0.9)^{D}	3.51 (0.9)	3.66 (0.9)^{B}	0.008
QMVC (Kg)	37.1 (10.8)^{D}	32.4 (11.4)^{D}	36.4 (10.4)^{D}	29.2 (10.3)^{A,B,C}	<0.001
QMVC/BMI	1.4 (0.4)^{B,D}	1.2 (0.4)^{A,C}	1.5 (0.4)^{B,D}	1.1 (0.4)^{A,C}	<0.001
6MWD (m)	441(106)^{D}	379 (110)^{D}	440 (75)^{D}	313 (121)^{A,B,C}	<0.001
APWV (m/s)	10.6 (2.7)	10.2 (2.7)	9.4 (2.2)	10.3 (2.8)	0.600

Disease) consortium (work package 1) cohort categorised by GOLD Group (ABCD).

Methods ERICA is a multicentre UK study investigating the role of inflammation and the prevalence and significance of cardiovascular and skeletal muscle manifestations in COPD. This interim analysis was conducted on 395 (49%) of 800 planned participants. Measurements include aortic pulse wave velocity (APWV) to measure arterial stiffness, quadriceps maximal voluntary contraction force (QMVC), plasma fibrinogen and 6-minute walk distance (6MWD). We defined arterial stiffness as APWV >10m/s, and skeletal muscle weakness as QMVC/BMI (Body Mass Index) ratio >1.2.

Results 395 subjects were classified according to GOLD groups using the mMRC (modified Medical Research Council) dyspnoea scale to evaluate symptoms, (**Table 1**). The majority of subjects were in groups D (57%) and B (33%), with low numbers observed in groups A (6%) and C (4%). Higher levels of airflow limitation were observed in groups C & D (p = 0.012). Fibrinogen was higher in groups C & D (p = 0.001), consistent with COPD severity.

Group D had reduced 6MWD and quadriceps strength consistent with the hypothesis that they have more severe extra-pulmonary manifestations of COPD and increased risk of mortality. Of note, Group B ('high symptom, low risk') also had reduced quadriceps strength and 6MWD, although for 6MWD this reduction (p = 0.06 compared to group A) was not as profound as Group D (p < 0.001). No difference between groups was observed for APWV.

Conclusion The GOLD group classification captures risk related to skeletal muscle weakness but not arterial stiffness and indicates groups B & D may benefit from intensive exercise therapy.

P130 IMPACT OF LEFT VENTRICULAR HYPERTROPHY ON MORTALITY IN COPD

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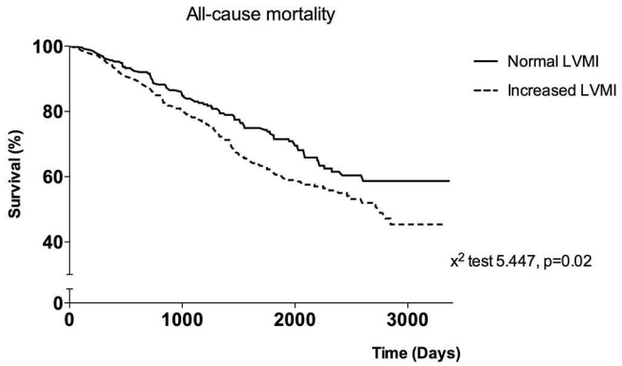
Introduction and Objectives Left ventricular hypertrophy (LVH) is a significant risk factor of cardiovascular disease and is associated with increased mortality. Previous studies have shown an increased prevalence of LVH in normoxaemic COPD patients. The impact of LVH on mortality in COPD is yet to be established.

We evaluated the impact of LVH on mortality in COPD patients by measurement of left ventricular dimensions by echocardiography.

Methods We performed a retrospective cohort study utilising a NHS database of COPD patients (TARDIS) in Tayside, Scotland between 2001 and 2010 that was linked with NHS Tayside databases regarding echocardiograms, pharmacy prescription and the General Register Office for Scotland death registry. Left ventricular internal diastolic diameter (LVIDd) and left ventricular mass index (LVMI) were measured. Increased LVIDd was defined as > 5.3 cm (female) and > 5.9 cm (male). LVMI was obtained by correcting the left ventricular mass to body surface area. LVH was defined as an LVMI of >95 g/m^2 (female) and > 115 g/m^2 (male). Patients with aortic valve disease were excluded from the analysis. The impact of increased LVIDd and LVMI on mortality were evaluated by Kaplan Meir testing and Cox Regression analyses after inclusion of covariates (age, FEV_1%, SaO_2%, history of diabetes and cardiovascular disease, medication (aspirin, ACE-inhibitor, statin, beta blocker).

Results 617 patients were included for analysis. Mean (SD) age at diagnosis, 70 (9); mean FEV_1% (SD), 60.6 (19.3); mean resting SaO_2% (SD), 92.7 (10). Mean follow up 4.5 years. Increased LVIDd was not associated with increased mortality, X^2 = 0.767, p = 0.381. Increased LVMI was associated with a significant increased risk of mortality, X^2 = 5.447, p = 0.02. with an adjusted HR (95%CI) of 1.542 (1.068–2.228), p = 0.021. (see graph below).

Conclusion The presence of left ventricular hypertrophy, demonstrated by elevated left ventricular mass index is associated with a significantly increased risk of mortality in COPD patients. Therapeutic interventions are required to address this important modifiable risk factor in COPD patients.



Abstract P130 Figure1 Kaplan _meier estimate of probability of survival dependent on LVMI. Increased LVMI; (male > 115g/m^2 or female > 95g/m^2)