**P135** A NOVEL COMPOSITE INDEX FOR PROGNOSTIC STAGING OF COPD PATIENTS

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**Introduction** Chronic Obstructive Pulmonary Disease (COPD) is characterised by high morbidity and mortality. Whether thorax computed tomography (CT)-derived parameters and lung function measurements carry more prognostic information individually or as a composite index has not yet been investigated.

**Aim** a) to compare the prognostic value of CT-determined emphysema and PAAo ratio versus various lung function parameters in a general COPD population and b) to construct a composite index for prognostic staging of COPD patients.

**Material and Methods** Predictors of mortality were assessed in a consecutive COPD outpatient population whose thorax CT, spirometry, lung volumes and gas transfer data were all collected prospectively in a clinical database. Univariate and multivariate Cox proportional Hazard analysis models were used and Hazard Ratios (HR) with corresponding 95% Confidence Intervals (CI) were the only independent predictors of mortality when ES was treated as continuous variable in the multivariate regression. No association was found between PAAo Ratio and survival. Further analysis was conducted using a composite ES-TLC index for the construction of the index. The final composite index separated patients in “high” risk (ES≥65% or TLC≥143% predicted for intermediate group) and “low” risk (ES<30% or TLC<143% predicted for intermediate group) (Figure) and was more discriminatory (HR = 2.751; 95% CI = 1.272–5.951) than any of its individual components.

**Conclusion** Although ES is better correlated with mortality than any pulmonary function parameter, a composite ES-TLC index carries the most prognostic information for COPD patients.

**P136** MULTIDIMENSIONAL PROGNOSTIC INDEX FOR EXACERBATIONS OF COPD

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**Introduction and Objectives** Prognostic assessment of COPD exacerbations is currently limited by models that only take into account short term prognostic factors. We developed a multidimensional tool for COPD exacerbations, predicting both short and long term outcome.

**Methods** A prospective multicentre, UK observational cohort of patients hospitalised with exacerbations of COPD 2009–2011. Cox-proportional hazards regression used to identify independent predictors of 30-day and 1 year mortality. Two independent risk scores based on exacerbation severity (acute score) and severity of COPD and co-morbidities (chronic score) were developed. The two scores were then used to generate a 4 class decision grid based on the GOLD 2011 criteria for stable COPD.

**Results** 1343 patients were included. 749 patients were readmitted or died during 1 year follow-up. Predictors of 30-day mortality (acute score) were new onset confusion HR 2.23 (95%CI 1.34–3.71)- 1 point, Urea >7mmol/L 2.64 (95%CI 1.51–4.61)- 1 point, acidosis 4.22 (95%CI 2.68–6.65)- 2 points, glucose >8mmol/L 1.56 95%CI (1.00–2.46)- 1 point and albumin <35g/L 2.23 (95%CI 1.42–3.5)- 1 point and heart rate >110bpm 2.37 (95%CI 1.50–3.73)- 1 point. The corresponding to ES optimal threshold, was further applied for the construction of the index. The final composite index separated patients in “high” risk (ES≥65% or TLC≥143% predicted for intermediate group) and “low” risk (ES<30% or TLC<143% predicted for intermediate group) (Figure) and was more discriminatory (HR = 2.751; 95% CI = 1.272–5.951) than any of its individual components.

Abstract P135 Figure 1. The Es-TLC composite index for the prognostic categorization of COPD patients.

Abstract P136 Figure 1.
INTERNAL VALIDATION OF THE DECAF SCORE AS A PREDICTOR OF INPATIENT MORTALITY IN ACUTE EXACERBATIONS OF COPD

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10.1136/thoraxjnl-2013-204457.287

Introduction In patients presenting with an acute exacerbation of COPD (AECOPD), accurate prediction of in-hospital mortality may help inform the most appropriate place and level of care. The DECAF score was developed for this purpose and designed to be simple to apply at the bedside using variables that are routinely collected on admission: Dyspnoea, Eosinopenia, Consolidation, Acidemia and atrial Fibrillation. Whilst the performance of the tool within the derivation cohort was strong,[1] before recommending use in clinical practice further validation is required.

Methods Both external and internal validation of the DECAF score are currently in progress; for each, a cohort of 840 patients consecutively admitted with AECOPD is being recruited. The resulting 4 stage model identifies different outcomes within each subgroup (See Figure 1). Furthermore the 4 stage model predicted 30-day mortality AUC 0.76 (95%CI 0.72–0.79), 1 year mortality 0.72 (95%CI 0.70–0.74) and readmissions 0.74 (95%CI 0.72–0.76) better than GOLD 2011 criteria.

Conclusion A multidimensional prognostic index can predict both short and long term outcomes after COPD exacerbations, and divides patients into clinically useful subgroups based on exacerbation severity and chronic health status.

Abstract P137 Table 1.

<table>
<thead>
<tr>
<th>DECAF AUROC</th>
<th>Derivation DECAF AUROC</th>
<th>Internal validation DECAF AUROC</th>
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<tbody>
<tr>
<td>0.82 (95% CI 0.76 to 0.87)</td>
<td>0.86 (95% CI 0.82 to 0.89)</td>
<td>0.82 (95% CI 0.76 to 0.87)</td>
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Discussion As in the derivation study, DECAF is a good predictor of inpatient mortality (AUROC = 0.82), with a stepwise increase in mortality with DECAF score. The DECAF score accurately identifies low risk (DECAF score 0-1) and high risk patients (3 or greater) admitted with an exacerbation of COPD, potentially helping select patients for early supported discharge schemes, or for intensified medical treatment or early palliation.

REFERENCES

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MODERATE-INTENSITY ACTIVITY IS ASSOCIATED WITH REDUCED CARDIOVASCULAR RISK FACTORS IN COPD

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10.1136/thoraxjnl-2013-204457.288

Background Steps measurement is a common measure of daily physical activity, but cannot define the intensity of physical activity. Moderate intensity activity is recommended type to maintain a basic level of health (1), but in patients with COPD, the effect of moderate intensity activity on systemic manifestations has not been studied. We hypothesised that patients with higher moderate activity time would have lower frailty, inflammation and cardiovascular risk.

Methods As part of a longitudinal study in COPD (ARCade), daily physical activity was recorded over seven days using a multisensor armband (SenseWear Pro armband) in 75 stable patients with COPD. Spirometry, body composition, aortic stiffness, comprehensive geriatric assessment, C-reactive protein (CRP) and fibrinogen were also determined. Moderate-intensity activity was determined by the monitor for activity between 3 to 6 METs.

Results Patients (42 males) mean (SD) age was 66 (7) years, BMI 27.5 (5.2) Kg/m², FEV1 predicted 55 (17)% and moderate activity time 1.46 (1.23) hours. The time spent on moderate activities was not related to age or FEV1pred. The moderate activity time related to BMI (r = -0.41), fat mass (r = -0.38) and fat percentage (r = -0.37), all p < 0.001, but not with fat free mass. However, none of these parameters related to the number of steps. Moderate activity time was inversely associated with aortic stiffness, r = -0.31, p < 0.01, CRP, fibrinogen (r = -0.26; r = -0.24, respectively) and frailty score all p < 0.05.

However, the number of steps only related to the inflammatory markers.

Conclusion The changes in frailty and cardiovascular risk factors including adipose tissue, aortic stiffness and inflammatory markers are linked to the proportion of time in moderate activities as their predominant form of activity.

REFERENCES

Mechanisms of lung injury

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PULMONARY SURFACTANT PROTECTS AGAINST SILVER NANOPARTICLE-INDUCED INFLAMMATION IN THE PERIPHERAL HUMAN LUNG

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10.1136/thoraxjnl-2013-204457.289

Background The concentration of silver nanoparticles (AgNPs) is increasing in the environment. We examined whether pulmonary surfactant protects against AgNPs-induced inflammation.

Methods Human tracheobronchial epithelial cells (HTECs) and peripheral blood mononuclear cells (PBMCs) were exposed to 1.25, 2.5, 5.0 or 10 μg/mL AgNPs or 1 μg/mL AgNO3. Cell viability was assessed with a trypan blue exclusion assay. PBMCs were also treated with recombinant surfactant in the presence and absence of AgNPs.

Results AgNPs induced dose-dependent increases in IL-6, IL-1β, and TNF-α production in HTECs, and IL-1β and TNF-α in PBMCs. Addition of recombinant surfactant significantly reduced AgNPs-induced IL-1β and TNF-α production in PBMCs.

Discussion Surfactant may play a protective role against AgNPs-induced inflammation in the lungs.