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 

1. To compare the prognostic value of CT-determined emphysema and PAAo ratio versus various lung function parameters in a general COPD population and 2. 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 calculated. Survival data were available until April, 2013.

Results 

169 patients were included (59.8% male, 61.1 years old). During the follow-up 20.1% died; mean survival was 115.4 months. Age (HR = 1.077; 95% CI = 1.032–1.121) and emphysema score (ES) (HR = 1.033; 95% CI = 1.010–1.057) 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 indicated that the 55% threshold of ES could be used as a composite index for COPD patients.

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.

Abstract P135 Figure 1. The Ex-TLC composite index for the prognostic categorization of COPD patients.
AUC for 30 day mortality for the acute score was 0.84 (95% CI 0.80–0.88).

The strongest predictors of 1 year mortality were age >80 years HR 1.25 (95% CI 1.00–1.44)–1 point, neoplastic disease 1.91 (95% CI 1.37–2.65)–1 point, MRC dyspnoea grade 4 or 5 (3.82 95% CI 2.61–5.58)–2 points, FEV1 <30% predicted 2.61 (95% CI 1.99–3.43)–1 point, long term oxygen therapy 1.60 (95% CI 1.11–2.29)–1 point and a history of myocardial infarction or heart failure 2.76 (95% CI 1.70–4.47)–1 point. The AUC for 1 year mortality of the chronic score was 0.76 (95% CI 0.73–0.80).

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.

## P137

**INTERNAL VALIDATION OF THE DECAF SCORE AS A PREDICTOR OF INPATIENT MORTALITY IN ACUTE EXACERBATIONS OF COPD**

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**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 on 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. To optimise data capture, patients are identified by screening admissions units and coding records. Indices that comprise the DECAF score and other independent predictors of mortality and readmission are collected. Dyspnoea is assessed using the extended MRC dyspnoea score.[1] Inclusion criteria are: primary diagnosis of AECOPD, age 35 or older, 10 pack years or more smoking history, and obstructive spirometry. Exclusion criteria are: other illness likely to limit survival to less than one year (principally metastatic malignancy) and previous inclusion in the validation study. We present an analysis of the performance of DECAF in the first 623 patients recruited to the internal validation cohort.

**Results**

**Abstract P137 Table 1.**

<table>
<thead>
<tr>
<th>Predictor</th>
<th>DECAF (AUC)</th>
<th>Derivation DECAF (AUC)</th>
</tr>
</thead>
<tbody>
<tr>
<td>FEV1 % predicted</td>
<td>0.82 (95% CI 0.76 to 0.87)</td>
<td>0.86 (95% CI 0.82 to 0.89)</td>
</tr>
<tr>
<td>Dyspnoea grade 4 or 5</td>
<td>0.76 (95% CI 0.70 to 0.74)</td>
<td>0.72 (95% CI 0.69 to 0.75)</td>
</tr>
<tr>
<td>MRC dyspnoea grade 4 or 5</td>
<td>0.85 (95% CI 0.81 to 0.89)</td>
<td>0.82 (95% CI 0.78 to 0.86)</td>
</tr>
</tbody>
</table>

*p value using Fisher’s exact test Internal validation DECAF AUC 0.82 (95% CI 0.76 to 0.87) Derivation DECAF AUC 0.86 (95% CI 0.82 to 0.89)*

**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**


## P138

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

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**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

P139 **PULMONARY SURFACTANT PROTECTS AGAINST SILVER NANOPARTICLE-INDUCED INFLAMMATION IN THE PERIPHERAL HUMAN LUNG**

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**Background** The effects of inhaled silver nanoparticles (AgNPs) on the lung are largely unknown. However, the response of pulmonary surfactant to other irritants is well documented.

**Aims** To investigate the protective role of pulmonary surfactant against AgNP-induced inflammation.

**Methods** Human lung fragments were exposed to AgNPs for up to 24 hours, followed by uptake of the inflamed lung slice. The lung slices were stained with anti-cytokeratin-18 and DAPI. Analysis was performed using confocal microscopy.

**Results** AgNP exposure resulted in increased cytokine production and inflammation in lung slices. Surfactant pre-treatment significantly reduced AgNP-induced inflammation.

**Conclusion** Pulmonary surfactant plays a protective role against AgNP-induced inflammation in the peripheral lung.