have relied on molecular methods which can be prolonged and expensive. The use of matrix-assisted laser desorption/ionisation-time of flight (MALDI-TOF) offers an efficient and economical approach to the evaluation of the airway microbiome, necessary to increase knowledge of the role of the microbial community in COPD.
Aims To identify the range and diversity of bacteria which form the airway microbiome in COPD To consider whether the MALDI-TOF is an appropriate method for this study.
Methods 44 patients from the London COPD cohort produced 65 sputum samples at baseline and exacerbation mean (±SD) age 73.7 years (±9.4); predicted FEV1 50.7% (±18.12); male gender 77.5%; exacerbation samples 47.7%. Sputum samples are initially sent to the NHS Royal Free Trust microbiology lab where traditional culture methods are performed. Once processed the agar plates were collected and different colonies are spotted onto target plates and loaded onto the MALDI-TOF MALDI-TOF profile spectrum is then generated allowing for data interpretation. Isolates that were defined as pathogenic were removed from the data with those classified as typical bacteria were then included separately. Results: Streptococcus, Staphylococcus, Rothia and Neisseria are the most dominant genera seen in baseline and exacerbation NRTF samples at the genus level contributing to 85, 28, 20 and 18 percent respectively. MALDI-TOF identified 25 NRTF groups at genus level in the COPD samples. In the typical pathogenic genus groups Haemophilus is the most dominant group identified by this method.
Conclusion The MALDI-TOF identifies a diverse bacterial range in COPD patients including pathogenic organisms and those defined as NRTF The MALDI-TOF is quick and inexpensive, as well as efficient which is an advantage over molecular assays.

S25 ARTERIAL STIFFNESS AND AIRWAY INFECTION AND INFLAMMATION DURING COPD EXACERBATIONS

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Introduction Cardiovascular risk is elevated following COPD exacerbations requiring systemic therapy (Donaldson *et al*, Chest 2010:137; 1091–7). We have recently shown that a potential mechanism is increased arterial stiffness during COPD exacerbations compared to the stable state (Patel *et al*, AJRCCM 2012:185; A5853). We hypothesised that arterial stiffness may be mediated by airway infection and inflammation during COPD exacerbations.
Methods We used aortic pulse wave velocity (aPWV) as a non-invasive validated measure of arterial stiffness using Vicorder apparatus (Skidmore Medical, UK) in outpatients from the London COPD cohort when they presented within seven days of exacerbation symptom onset and prior to any systemic therapy. These events were defined using our usual symptomatic criteria (Seemungal *et al*, AJRCCM 1998:157; 1418–22). We collected spontaneous sputum at the same clinic visit to identify typical bacterial (*S pneumoniae, H influenzae* and *M catarrhalis*) and rhinovirus infection using PCR. We also measured IL-6 and IL-8 in sputum supernatant using commercial ELISA kits (R&D Systems, USA).
Results 32 exacerbating COPD patients produced a spontaneous sputum sample during the same clinic visit as an aPWV measurement. Their stable clinical characteristics were: 69% male; 25% current smokers; median (IQR) 39 (21.74) pack year smoking history;

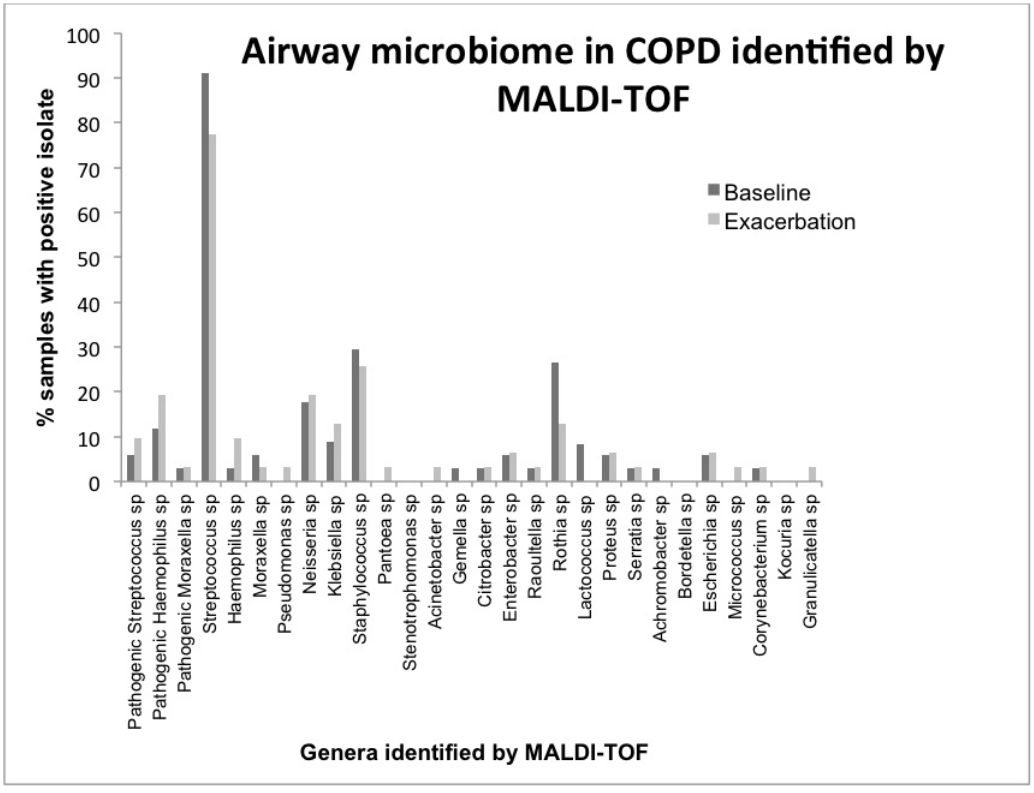


Figure 1, Genera identified by MALDI-TOF in baseline and exacerbation sputum samples from patients with COPD.

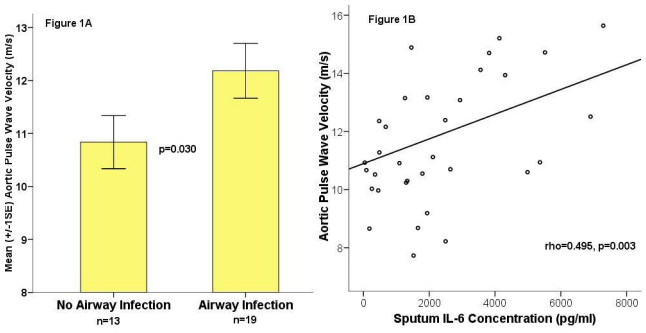
Abstract S24 Figure 1

mean±SD age 72.4±8.1 years; FEV1 1.18±0.41L and 46.8±18.0%predicted; FEV1/FVC ratio 0.44±0.15 and BMI 25.4±3.8 kg/m^2.

Arterial stiffness was higher in patients with airway infection at exacerbation presentation (mean ±SD aPWV 12.3±2.3 (n=19) vs 10.8±1.8 (n=13) m/s, p=0.030) (Figure 1A).

Arterial stiffness was strongly correlated with sputum IL-6 at exacerbation (n=32, rho=0.495, p=0.003) (Figure 1B) but not IL-8 (n=31, rho=0.100, p=0.591).

Conclusions Arterial stiffness is related to airway infection and inflammation during COPD exacerbations. Interventions to prevent and reduce airway infection and inflammation may lower cardiovascular risk during COPD exacerbations.



Abstract S25 Figure 1

S26 INDIVIDUALISING THE MORTALITY RISK FOR LUNG VOLUME REDUCTION SURGERY

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Background Despite the positive results of the NETT trial in favour of lung volume reduction surgery (LVRS) uptake of the technique has been limited largely due to an exaggerated fear of the associated mortality risk. We have analysed our 18 year experience of LVRS to provide a more sophisticated personalised risk profile based on individual patient data.

Methods Since 1994 we have performed 250 lung volume reduction procedures on 220 patients: 153M:97F, age 61 (39–74) years. The initially approach was through median sternotomy (20 patients), with the subsequent 230 procedures performed by video-assisted thoracoscopic surgery (VATS), 3 of which required conversion to open thoracotomy. All patients underwent standard physiological and anatomical selection techniques with 51 (20%) falling outside recognised safety limits (FEV1 or DLCO <20% predicted). All patients were offered surgery after discussion in our LVRS MDT panel and counselled on risk on their basis of their physiological status. We analysed data collected prospectively using logistic regression to identify the factors predicting early postoperative mortality.

Results Open surgery significantly increased the risk of 30 day mortality 22% vs VATS 3.6% (p=0.005). Bilateral vs unilateral VATS had no influence. At 30 days mortality was associated with low BMI, DLCO and KCO At 90 days, mortality was also associated with FEV1 and RV:TLC DLCO was the only significant independent predictor of 30 day (OR 0.88, CI 0.80–0.97) and 90 day (OR 0.92, CI 0.88–0.98) mortality after VATS (table 1).

The causes of death after 30 days in the VATS group were mainly due to pneumonia (5 cases) with cardiac complications (2); tension pneumothorax (1) and fatal pulmonary haemorrhage (1) in the remainder.

Conclusion LVRS is primarily a procedure to improve health status so accurately informed consent is imperative. Careful consideration of preoperative physiological characteristics and operative technique allows estimation of an individualised mortality risk for LVRS which may be lower than the commonly perceived overall figure.

Abstract S26 Table 1

DLCO (%pred)	30 day mortality VATS	90 day mortality VATS
<20%pred (n=19)	16%	32%
20–40%pred (n=102)	5%	11%
40–60%pred (n=76)	0%	0%
>60%pred (n=16)	0%	6%

S27 CARDIOVASCULAR EVENTS FOLLOWING CLARITHROMYCIN USE IN LOWER RESPIRATORY TRACT INFECTIONS: ANALYSIS OF TWO PROSPECTIVE COHORT STUDIES

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Background Previous studies have suggested that use of macrolide antibiotics may increase cardiovascular risk.

Objective To study the effects of clarithromycin on cardiovascular events in the setting of acute exacerbations of chronic obstructive pulmonary disease (AECOPD) and community acquired pneumonia (CAP).

Design Cohort study of two prospectively collected datasets; a multicentre observational study of patients hospitalised with AECOPD and the Edinburgh pneumonia study cohort.

Setting COPD dataset includes patients admitted to one of 12 hospitals around the United Kingdom between 2009–2011. The Edinburgh pneumonia study cohort includes patients admitted to NHS Lothian Hospitals between 2005–2009.

Main outcome measures Hazard ratios (HR) for cardiovascular events at 1 year (defined as hospitalised with acute coronary syndrome (ACS), decompensated cardiac failure, serious arrhythmia or sudden cardiac death) and hospitalisation for acute coronary syndrome (acute ST elevation myocardial infarction, non-ST elevation MI and unstable angina). Secondary outcomes were all cause and cardiovascular mortality at 1 year. Cox proportional hazard regression was used to calculate hazard ratio's and 95% confidence intervals after adjusting for significant covariates.

Results There were 1323 and 1631 patients in the AECOPD and CAP cohorts with 268 and 171 cardiovascular events respectively over 1 year. Macrolide use in AECOPD was associated with increased risk of cardiovascular events HR 1.60 (1.17–2.20) and ACS HR 1.88 (1.16–3.01). Macrolide use in CAP was associated with increased risk of cardiovascular events HR 1.81 (1.28–2.55) and ACS HR 1.90 (1.07–3.33). There was a significant association between macrolide use and cardiovascular but not all cause mortality in AECOPD HR 1.67 (1.11–2.51) and 1.24 (0.96–1.61). Macrolide use in CAP was associated with a trend towards increased risk of all cause and cardiovascular mortality HR 1.13 (0.85–1.51) and 1.57 (0.84–2.82). These relationships persisted after propensity matching. Statins and other cardiac drugs attenuated this increased risk. Longer durations of macrolide use were associated with more cardiovascular