

frequency and airway inflammation have been suggested. There is however limited data describing the reproducibility of sputum microbiology results in clinically stable COPD patients.

**Aims** Examine repeatability of sputum microbiology in subjects with stable COPD over time.

**Methods** Subjects with COPD were enrolled into an observational study and seen at baseline and at stable visits after 3 and 6 months. Sputum was obtained and samples were divided and analysed over time using standard culture, semi-quantitative bacterial count (colony forming units, CFU), PCR for potentially pathogenic organisms [*Haemophilus influenzae* (HI), *Streptococcus pneumoniae* (SP), *Staphylococcus aureus* (SA), *Moraxella catarrhalis* (MC)] and quantitative bacterial 16S analysis.

**Results** 63 subjects provided paired sputum samples; 52 were male with a mean (SD) FEV1 (L) and FEV1/FVC ratio (%) of 1.48(0.54) and 53% (12.8) respectively. 40% were current smokers with an exacerbation frequency of 3 in the preceding year.

**Results** for standard culture were divided into two groups (culture positive or negative). Results are expressed as Kappa values (95% CI). There was moderate agreement after 3 months, Kappa = 0.48 (0.24 to 0.71); and after 6 months, Kappa = 0.50 (0.25 to 0.76). Individual PCR revealed fair agreement after both time intervals. After 3 months, HI=0.17(−0.08 to 0.43), SA=0.27(−0.03 to 0.56), SP=0.30(0.06 to 0.53), MC=0.19(−0.04 to 0.43). After 6 months, HI=0.09(−0.18 to 0.35), SA=0.10(−0.22 to 0.43), SP=0.37(0.13 to 0.62) and MC= −0.14(−0.4 to 0.11).

Quantitative bacterial analysis demonstrated no differences (mean difference; 95% CI) at 3 or 6 months in bacterial load measured by CFU (−0.18; −0.41 to 0.04,  $p=0.11$  and −0.06; −0.32 to 0.2,  $p=0.65$  respectively) or 16S (−0.03; −0.28 to 0.33,  $p=0.86$  and −0.1; −0.42 to 0.22,  $p=0.54$  respectively).

**Discussions** These results demonstrate that sputum microbiological assessment in stable COPD is complex. Further longitudinal assessments of sputum microbiology and associations with clinical features are needed.

## P213 THE PREVALENCE AND IMPACT OF GASTRO-OESOPHAGEAL REFLUX SYMPTOMS IN STABLE COPD PATIENTS

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**Introduction** Gastro-oesophageal reflux disease (GORD) has been associated with increased COPD exacerbation frequency (Terada et al, Thorax 2008) and was independently associated with the frequent exacerbator phenotype in the ECLIPSE study (Hurst et al, NEJM 2010). We aimed to quantify the prevalence and impact of GORD in stable COPD in terms of airflow limitation, dyspnoea, health status and exacerbation frequency in a well-characterised cohort.

**Methods** Stable outpatients from the London COPD cohort completed the Frequency Scale for the Symptoms of Gastro-oesophageal reflux (FSSG), Hull Airway Reflux Questionnaire (HARQ), MRC dyspnoea score, and St George's Respiratory Questionnaire (SGRQ) during clinic visits. Spirometry was performed in accordance with ATS/ERS guidance. Comorbidities including GOR and all medications were recorded by clinical research staff. Exacerbations were defined using our usual symptomatic criteria from daily diary cards (Seemungal et al, AJRCCM 1998).

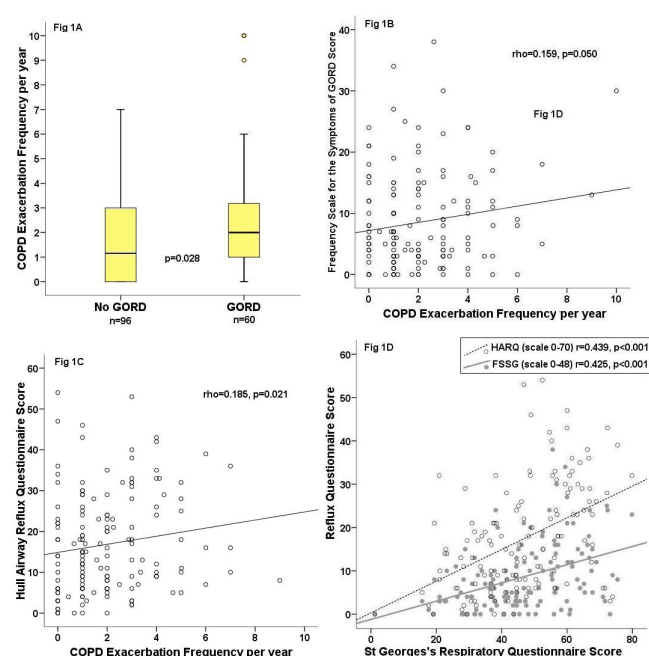
**Results** 156 stable COPD patients had a mean±SD age of 72.2±9.7 years, 63% male, 29% current smokers, median (IQR) 46 (25,59) pack years, mean±SD FEV1 1.34±0.57L and 54.4±20.2% predicted, BMI 26.5±5.7kg/m<sup>2</sup>.

60 (38%) patients had a diagnosis of GORD, of whom 45 (75%) were taking regular acid suppression therapy (42 proton pump inhibitors, 3 H<sub>2</sub> antagonists).

COPD patients with GORD had a higher exacerbation frequency than those without (median (IQR) 2.0 (1.0,3.4) vs 1.2 (0.0, 3.0) per year,  $p=0.028$ ). In those with GORD, the use of acid suppression therapy did not impact exacerbation frequency (median (IQR) 2.0 (1.0,4.0) (n=45) vs 2.0 (1.0, 3.0) (n=15) per year,  $p=0.431$ ).

FSSG and HARQ scores were both related to COPD exacerbation frequency ( $\rho=0.159$ ,  $p=0.050$  and  $\rho=0.185$ ,  $p=0.021$  respectively) and more strongly to SGRQ ( $r=0.425$ ,  $p<0.001$  and  $r=0.439$ ,  $p<0.001$  respectively). They were not related to MRC dyspnoea score ( $\rho=0.079$ ,  $p=0.367$  and  $\rho=0.126$ ,  $p=0.148$  respectively) or FEV1% predicted ( $r=-0.063$ ,  $p=0.452$  and  $r=-0.067$ ,  $p=0.416$  respectively).

**Conclusions** GORD is common in COPD and is associated with higher exacerbation frequency, although acid suppression therapy does not appear to affect this. Higher GORD symptom scores relate to worse health status and higher exacerbation frequency but not to airflow limitation or dyspnoea. Understanding the mechanisms may lead to novel effective interventions in COPD.



Abstract P213 Figure 1

## P214 GASTRO-OESOPHAGEAL REFLUX SYMPTOMS DURING COPD EXACERBATIONS

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**Introduction** Gastro-oesophageal reflux disease (GORD) has been associated with increased COPD exacerbation frequency (Terada et al, Thorax 2008) and was independently associated with the frequent exacerbator phenotype in the ECLIPSE study (Hurst et al, NEJM 2010). We aimed to quantify any changes in GORD symptoms during COPD exacerbations.

**Methods** Outpatients from the London COPD cohort completed the Frequency Scale for the Symptoms of Gastro-oesophageal reflux (FSSG) and Hull Airway Reflux Questionnaire (HARQ) during stable-state clinic visits and at exacerbation, within a week of symptom-onset, and prior to systemic therapy. FSSG and HARQ scores range from 0–48 and 0–70 respectively, with significant reflux thought to be associated with scores of ≥8 and ≥13 respectively.