between 61.3% and 98.1%. Procalcitonin-based protocols decreased antibiotic prescription (RR 0.56 [0.43, 0.73]) and the total antibiotic exposure (MD -3.83 [-4.32, -3.35]), without affecting clinical outcomes such as rate of treatment failure (RR 0.81 [0.62, 1.06]), length of hospitalisation (MD -0.76 [-1.95, 0.43]), exacerbation recurrence rate (RR 0.96 [0.69, 1.35]) or mortality (RR 0.99 [0.57, 1.70]). However, the quality of the available evidence is low to moderate because of methodological limitations and small overall study population.

Conclusion Procalcitonin-based protocols to guide the administration of antibiotics in patients presenting with AECOPD appear safe and clinically effective. The quality of the available evidence is low-to-moderate because of methodological limitations and small overall population. Thus, additional appropriately designed and powered confirmatory randomised controlled trials are required.

P52 COPD AND PERIODONTITIS: CO-MORBIDITY YES OR NO?

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Introduction Chronic obstructive pulmonary disease (COPD) is an inflammatory disease associated with comorbidities including periodontitis.¹⁻² Periodontitis is characterised by plaque build-up, anaerobic bacterial overgrowth and gingival inflammation which promotes recruitment and activation of neutrophils leading to alveolar bone destruction and tooth loss. However, the characterisation of periodontitis varies between studies causing some uncertainty of any association.

Aim To determine whether clinical indices of periodontitis affects its prevalence in COPD patients with and without Alpha-1-antitrypsin deficiency (AATD) and any association with lung function.

Methods 108 COPD and 63 PiZ AATD patients underwent dental examinations and lung function testing as part of an EU FP7 cross sectional study.

Varying definitions of periodontitis used in previous publications were applied; including criteria from the Centres for Disease Control and Prevention in collaboration with the American Academy of Periodontology CDC-AAP (CDC-AAP) and 5th European Workshop in Periodontology.

Periodontal indices of probing depth (PD – depth from gingival margin to the base of periodontal pocket) and clinical attachment level (CAL – distance from the cemento-enamel junction to the gingival margin plus probing depth) were then compared to lung function parameters.

Results The prevalence of periodontitis varied depending on the definition used.

Prevalence ranged from 0.7–98.6% for the whole cohort, with the lowest prevalence for average probing depth >4 mm, but CDC-AAP criteria gave a prevalence of 84.2% and 98.6% with the 5th European workshop criteria.

Lung function was significantly correlated with indices of periodontitis for AATD patients; see Table.

Conclusions The prevalence of periodontitis depends on the definition used. PD is a marker of current status, whilst CAL represents cumulative disease activity, rather like current lung function parameters.

Abstract P52 Table 1 Relationships between clinical indices of periodontitis and lung function in COPD and AATD

	Spearman's Rho and p-value			
	Average Probing Depth (PD)		Average Clinical Attachment Level (CAL)	
	COPD	AATD	COPD	AATD
% predicted FEV1	0.085 p = NS	-0.42 p < 0.01	-0.03 p = NS	-0.52 p < 0.001
% predicted TLC	-0.06 p = NS	-0.34 p < 0.01	0.01 p = NS	-0.51 p < 0.001
% predicted KCO	-0.10 p = NS	-0.30 p < 0.05	-0.04 p = NS	-0.42 p < 0.01

Periodontal indices are correlated with lung function parameters in AATD patients which could reflect the inflammatory and predominantly neutrophilic pathophysiology leading to excessive tissue destruction in both diseases.

REFERENCES

- Shen TC, et al. Risk of Periodontal Diseases in Patients With Chronic Obstructive Pulmonary Disease: A Nationwide Population-based Cohort Study. *Medicine (Baltimore)* 2015; **94**(46):e2047.
- Chung JH, et al. *J Periodontol* 2016;1–11.

P53 PREDICTING POOR OUTCOMES IN COPD PATIENTS DEEMED 'LOW RISK' BY DOSE SCORE

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Introduction COPD continues to cause a substantial symptom, mortality and financial burden in the UK. Current treatment strategies are predominantly reactive as insufficient evidence exists to successfully target clinical resource into pre-emptive 'early interventions'. The DOSE (dyspnoea, obstruction, smoking status and exacerbation) score has been validated as a risk predictor for mortality, hospitalisation and poorer health status. However, only a small proportion of COPD patients with poor outcomes have high DOSE scores. We sought to establish if clinical characteristics can be used to pre-emptively identify those COPD patients vulnerable to future poor health status by using an electronic database of anonymised patient records-the Hampshire Health Record Analytical Database (HHRA).

Methods Within our HHRA database COPD cohort, we identified a cohort of 6890 patients who fell into the 'low risk' category by DOSE score (<4). Within this group, a subset met the criteria for poor COPD outcomes over the next four years, defined as; death (all cause), COPD related hospital admission, a DOSE score increase of ≥2 points or a subsequent DOSE score of ≥4 (high risk). We used logistic regression analysis to examine the association between demographic and clinical characteristics documented by Read code at baseline and those who subsequently fell into the poor outcomes subgroup.

Abstract P53 Table 1 Associations between clinical characteristics and odds of future poor clinical outcome

Clinical characteristic*	OR	p
Age		
<50 (reference)		
50-59	1.28	0.272
60-69	1.52	0.043
70-79	2.00	0.001
80-89	2.70	<0.001
90+	5.21	<0.001
Deprivation decile**	0.95	<0.001
BMI		
18-24 (reference)		
<18	1.61	0.029
25-29	0.75	<0.001
30-34	0.67	<0.001
35+	0.87	0.195
History of pneumonia	5.10	<0.001
History of raised eosinophils (≥2%)	1.18	0.011
History of Heart Failure	1.44	0.007
History of Cerebrovascular Disease	1.49	<0.001
History of Peripheral Vascular Disease	1.53	0.001
History of Anxiety Or Depression	1.20	0.007
History of Chronic Kidney Disease	1.25	0.010
History of Rhinosinusitis	0.75	0.001
Prescriptions in preceding 12 months:		
Nebulised bronchodilator	2.24	<0.001
ICS/LABA combination inhaler	1.27	<0.001
Inhaled LABA or LAMA	1.71	<0.001
Inhaled SAMA	1.44	<0.001
Inhaled SABA	1.24	0.004
Constant	0.28	<0.001

*Variables excluded from model due to statistical insignificance: gender, IHD, hypertension, osteoporosis, GORD, diabetes, total number of comorbidities.

**Decile 10 represents least deprived.

years, 2211 (44.2%) of those 5000 patients fell into the poor outcomes subgroup. As shown in Table 1, poor future outcomes were significantly associated with age, high deprivation decile, low BMI, certain comorbidities, a raised eosinophil percentage (≥2%) and the prescription (in the preceding twelve months) of nebulised bronchodilators, inhaled bronchodilators and an ICS/LABA combination inhaler. A BMI of >25 and rhinosinusitis were associated with a lower risk of poor future outcomes.

Conclusions Poor future clinical outcomes appear to be associated with certain clinical characteristics in a COPD database cohort deemed low risk by DOSE score. These findings warrant further validation in a clinical cohort and investigation into the effect of pre-emptive optimisation of these characteristics on health outcomes.

P54

CAN MULTI-MORBID PHENOTYPES BE DESCRIBED IN PATIENTS WITH ADVANCED COPD USING CLUSTER ANALYSIS?

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Introduction and objectives Comorbidities have a negative effect upon outcomes in patients with COPD, and ‘phenotypes’ of comorbidity have been described.¹ International guidelines recommend that comorbidities “*should be looked for routinely*”.² We aimed to objectively assess comorbidities, and investigate whether comorbidity phenotypes could be described using cluster analysis, in a cohort of patients with advanced COPD.

Methods Patients with advanced COPD were prospectively recruited to undergo a ‘Comprehensive Respiratory Assessment’ (CRA), as previously described.³ 13 comorbidities were objectively assessed using validated definitions and their prevalence determined. K-means cluster analysis was applied with the objective measurements, and FEV₁%predicted. The clusters formed were compared with respect to demographic features, measures of health status, self-reported exacerbation frequency, and future cardiovascular risk.

Results In our ‘low risk’ cohort of 6890 COPD patients, 5000 held sufficient data to be included in the analysis. After four

Abstract P54 Table 1 Features of multi-morbid clusters

Cluster:	1 (n = 51)	2 (n = 50)	3 (n = 36)	4 (n = 36)	5 (n = 30)
Age (years)	62.3 ± 8.5*	72.4 ± 8.0†	64.2 ± 9.1	62.0 ± 9.0*	65.4 ± 7.4
Gender (% male)	52.9%	68.0%	52.8%	72.2%	53.3%
FEV ₁ % predicted	30.4% ± 10.5%	34.0% ± 11.2%†	25.6% ± 6.5%*	30.2% ± 9.9%	34.2% ± 9.8%
Participants using home oxygen (%)	35.3%	42.0%	30.6%	25.0%	53.3%†
COPD Assessment Test (CAT) score	30 ± 5†	24 ± 7	26 ± 6	21 ± 7*	27 ± 5
Eosinophilia (>0.3) in the past 3 years	45.1%	58.0%	44.4%	55.6%	53.3%
Antibiotic courses in past year	5.0 [2.0 – 7.0]	4.0 [2.0 – 6.0]	5.0 [2.0 – 7.0]	2.0 [1.0 – 5.0]*	3.0 [2.0 – 6.0]
Steroid courses in past year	4.0 [2.0 – 7.0]	4.0 [2.0 – 6.0]	4.0 [2.0 – 7.0]	2.0 [1.0 – 5.0]*	3.0 [2.0 – 6.0]
COPD hospitalisations in past year	1.0 [0.0 – 2.0]	0.5 [0.0 – 2.0]	1.0 [0.0 – 1.0]	0.0 [0.0 – 1.0]	0.0 [0.0 – 1.0]
Total hospitalisations in past year	1.0 [1.0 – 3.0]	1.0 [0.0 – 3.0]	1.0 [1.0 – 2.0]	1.0 [0.0 – 2.0]	1.0 [0.0 – 2.0]
Framingham risk (%)	7 ± 5*	12 ± 7†	7 ± 5*	11 ± 7	10 ± 7

Data presented as mean ± standard deviation and median [interquartile range].

* p<0.05, value of cluster is significantly lower than remainder of sample.

† p<0.05, value of cluster is significantly higher than remainder of sample.