



Abstract S130 Figure 1 Kaplan–Meier curve showing *Pseudomonas*-free time following eradication therapy.

eradication ($p=0.002$). Mean number of hospital admissions per year were similar, at 0.39 pre-eradication and 0.29 post-eradication ($p=NS$). At first follow-up, 20 patients (66.7%) reported overall clinical improvement, with reduced cough in 12 (40.0%), reduced sputum volume in 14 (46.7%) and reduced sputum purulence in 11 (36.7%). Of the 21 patients followed up to 1 year, 6 (28.6%) had further overall improvement and 13 (61.9%) remained stable. Lung function was not affected by eradication therapy, with mean percentage predicted FEV₁ 62.1% pre and 64.1% post-eradication ($p=NS$).

Conclusion This study demonstrated that *Pseudomonas* eradication therapy can lead to prolonged clearance of this organism and highly significantly reduces exacerbation rate. This important outcome requires confirmation in a prospective study.

S131 FATIGUE IN BRONCHIECTASIS: ITS RELATIONSHIP TO PSEUDOMONAS COLONISATION, DYSPNOEA AND AIRFLOW OBSTRUCTION

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Introduction and objectives Fatigue is a complex and disabling symptom in non-CF bronchiectasis (nCF-Br) and can be formally measured using the Fatigue Impact Scale (FIS). FIS scores of >40 out of 120 are clinically significant. The FIS score has been shown to be prognostic of premature death in Primary Biliary cirrhosis but is not linked to markers of organ dysfunction (liver function tests). As poorer outcomes have been recently reported in *Pseudomonas* infected nCF-Br patients we aimed to measure the correlation between FIS scores and parameters of severity in nCF-Br, for example, *Pseudomonas* infection, degree of dyspnoea and airflow obstruction.

Methods FEV₁% predicted, MMRC dyspnoea score (MMRCD) and FIS were recorded in stable adult nCF-Br patients attending specialist clinic. All previous sputum cultures isolating *Pseudomonas aeruginosa* were reviewed. Two groups of patients were studied: those with *Pseudomonas* 'colonisation' (organism cultured ≥ 2 occasions, 3 months apart within 1-year period) and those with 'isolation' (organism cultured ≥ 1 occasion). Statistical comparison used χ^2 , Fisher's correlation and Mann–Whitney U tests.

Results 73F, 41M patients were included; average age 60 (range 24–90) with an average FEV₁ 66% predicted (SD ± 26). 54

(47%) patients had *Pseudomonas* isolation; 38 (33%) patients had colonisation. Fatigue levels were similar in patients with and without colonisation (median 48.5 vs 36.5, $p=0.31$). Significant fatigue (FIS>40) was more common in patients with *Pseudomonas* isolation (47%) than those with no previous isolates ($p=0.04$, OR=2.2) However, fatigue levels, although increased, were not significantly different (median FIS 50 vs 32; $p=0.064$). Fatigue correlated with MMRCD ($r=0.54$, $p<0.0001$) but less well with FEV₁% predicted ($r=0.2$, $p=0.04$). FEV₁% predicted was lower in patients with *Pseudomonas* colonisation (median FEV₁ 49% vs 74%, $p=0.0007$) and in patients with *Pseudomonas* isolation (median FEV₁ 52% vs 74%, $p=0.002$).

Conclusions *Pseudomonas* infection (past or present) appears to be associated with greater clinically significant fatigue scores and poorer lung function. Fatigue doesn't strongly correlate with FEV₁% predicted but is correlated with MMRCD. Further regression analysis of variables is underway to understand these inter relationships further. Systemic aspects of *Pseudomonas* infection may be different to other infections explaining the divergence.

Clinical and translational observations in asthma

S132 AIRWAY DYSFUNCTION AND INFLAMMATION IN POOL AND NON-POOL BASED ELITE ENDURANCE ATHLETES

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Introduction Previous studies have suggested that eosinophilic airway inflammation is common in elite swimmers; the chemical pool environment often blamed. We set out to address this question in a cross-sectional study of 109 international athletes from a variety of sporting backgrounds.

Methods All had symptoms suggesting exercise-induced asthma and were either inhaled corticosteroid naïve or withdrew these for >4 weeks. β_2 -agonists, exercise and caffeine were withheld for 8 h prior to testing. Symptoms were assessed using the Juniper ACQ, airways dysfunction using the eucapnic voluntary hyperventilation (EVH) test and airways inflammation using exhaled nitric oxide (FE_{NO}) and induced sputum eosinophil % (eos). Athletes were classed as pool based if they exercised in an indoor pool environment for >5 h per week, and non-pool based if they exercised in a pool for <1/2 h per week.

Results Demographic details were similar. Mean (\pm SEM) % fall in FEV₁ post EVH was 18.96 \pm 1.701 (n=47) in pool and 11.39 \pm 1.249 (n=62) in non-pool athletes (mean difference 7.569; 95% CI 3.480 to 11.66; $p=0.0004$), 76% of pool and 39% of non-pool athletes had a positive test (>10% fall). The geometric mean (log SD) eos (pool 2.667 (0.797)), non-pool 3.060 (0.867), $p=0.802$), and FE_{NO} (pool 25.05 (1.570), non-pool 28.06 (1.475) ppb, $p=0.914$) was no different between groups; 14.9% of pool and 12.9% of non-pool athletes had eos >3%. % fall in FEV₁ had good correlation with log eos ($r=0.551$, $p<0.0001$); a 25% fall being the optimum (AUC 0.89, $p<0.0001$, sens 79%, spec 93%). Log FE_{NO} and log eos correlated strongly ($r=0.644$, $p<0.0001$); FE_{NO} of >47 being most predictive of eos >3% (AUC 0.912, $p<0.0001$, sens 78%, spec 92%). Symptoms correlated poorly with either airways dysfunction or inflammation.

Conclusions Individual athletes with symptoms vary markedly in the levels of airways dysfunction and inflammation expressed. There is more airway dysfunction in pool athletes but not more eosinophilic airways inflammation suggesting that the pool

environment if anything predisposes to airways dysfunction. Exhaled nitric oxide is a promising non-invasive means of assessing eosinophilic airways inflammation in all elite athletes.

S133 EOSINOPHILIC AIRWAY INFLAMMATION IS ASSOCIATED WITH FEV₁ DECLINE IN SEVERE ASTHMA

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Background Severe asthma is a multidimensional disease, with recent evidence supporting the notion that eosinophilic airway inflammation (EAI) is an important driver for exacerbations. In addition EAI has been shown to be associated with airflow limitation in cross sectional studies. However, it remains to be established whether EAI may drive FEV₁ decline.

Methods The severe asthma registry at Glenfield hospital, Leicester, was screened for patients with a physician diagnosis of asthma and at least 5 years of longitudinal data recording sputum eosinophils, pre- and post-bronchodilator spirometry, inhaled corticosteroid usage as well as standard demographic indices during stable scheduled follow-up visits. Linear mixed effects models were used to investigate the effect of log sputum eosinophils as a time varying covariate on decline of post bronchodilator FEV₁. Models were iteratively compared and refined using standard information criteria. Other fixed effects in the final model were, time and the interaction terms for time * log sputum eosinophils and time * daily dose of inhaled corticosteroids and pack years smoked. Individual variations in the slopes and intercepts of time and time*log sputum eosinophils were considered by adding them iteratively as random effects. A first-order autoregressive correlation structure was used to model covariance of random effects.

Results 92 patients, 46% male with severe asthma were identified from a registry cohort of 686 between 2000 and 2009. The mean (sem) age was 54(12.9) years and age of onset 23 (2.1) years. The mean (range) duration of follow-up and number of visits were 6 years (4.6–10.5), 2.7/year. We found a significant interaction between sputum eosinophils, time and post bronchodilator FEV₁. Indicating a net decline (95% CI) of –16.8 mls(25.8–7.8 mls)/annum/log unit increase in sputum eosinophils (F(1, 43.4); p<0.0001). In contrast there was a net decline 95%CI of –0.015 mls (0.029 to 0.0014 mls)/annum/mcg of inhaled beclamethasonedipropionate daily (F(1,726); p=0.031).

Conclusion Eosinophilic airway inflammation is associated with a significant decline in FEV₁ in severe asthma.

S134 WITHDRAWN

S135 CAN YOUR MOBILE PHONE IMPROVE YOUR ASTHMA?

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Background It is recognised that some 45% of the population exhibit poor asthma control. Over 90% of the population possess a mobile phone (>70% over 60 years of age). Mobile technology potentially

Abstract S135 Table 1

ACQ6 score	Group 1(n=110)	Group 2(n=99)	Between group p value
Baseline, median (IQR)	2.17 (1.67–2.67)	2.33 (1.67–2.67)	0.441
6 months, median (IQR)	1.25 (0.67–1.83)	1.17 (0.67–1.83)	0.995
Median difference (IQR)	0.83 (0.17–1.50)	0.83 (0.33–1.33)	0.586
Within group p value (Wilcoxon)	<0.001	<0.001	
Mean difference (95% CI)	0.84 (0.67 to 1.02)	0.94 (0.77 to 1.11)	0.434
Within group p value (t-test)	<0.001	<0.001	
MID*, n (%)			0.652
Improvement = MID	74 (67.3)	73 (73.7)	
Improvement < MID	20 (18.2)	16 (16.2)	
Deterioration < MID	6 (5.5)	5 (5.5)	
Deterioration = MID	10 (9.1)	5 (5.1)	

addresses the barriers of low expectations and poor concordance which are factors in poor asthma control.

Hypothesis Using mobile phone recording of symptoms, lung function and medication use with instant feedback of asthma control, would improve control compared to using paper diaries.

Trial design A 6-month researcher-blinded randomised controlled trial

Setting UK primary care

Method Using central randomisation, we allocated patients ≥12 years of age with poorly controlled asthma (ACQ>1.5) to either mobile phone or paper-based monitoring. Clinical care was provided by practice asthma nurses in accordance with SIGN/BTS guidelines. Patients were reviewed monthly until control was achieved. A researcher, blinded to allocation assessed outcomes at 3 m and 6 m. Primary outcome measure: change in Asthma Control Questionnaire score between baseline and 6 months.

Results We randomised 288 patients from 32 practices (209 completed). Baseline characteristics of both groups were similar. Intention to treat analysis, before breaking the randomisation code, showed that control in both groups improved significantly and to a similar extent. ACQ: Group 1 (n=110) Baseline 2.17, 6m 1.25; Group 2 (n=99) Baseline 2.33, 6m 1.17. Mean (95% CI) improvement in ACQ: Group 1 (n=110) 0.84 (0.67, 1.02), Group 2 (n=99) 0.94 (0.77, 1.11) both p<0.001. Between group p=0.434 ns. Approximately 70% in each group improved by ≥0.5 (minimal clinically important difference).

Conclusion Both groups demonstrated significant improvement in asthma control from baseline. Use of mobile phone technology provided no additional benefit over paper diaries.

S136 FUNGAL SPUTUM CULTURE IN PATIENTS WITH SEVERE ASTHMA IS ASSOCIATED WITH A REDUCED POST BRONCHODILATOR FEV₁

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Introduction and objectives IgE sensitisation to fungal allergens is common in severe asthma, but the clinical relevance of this, and the relationship to airway colonisation with fungi, is not known. Many of the fungi that can grow at body temperature are filamentous moulds from the genera *Aspergillus* and *Penicillium*. We report here the