Abstract S129 Figure 1  Receiver operating characteristic (ROC) analysis for hydrogen cyanide and isoprene. Analysis of hydrogen cyanide and isoprene was performed in the exhaled breath of 21 patients with suspected community acquired pneumonia (CAP). A diagnosis of pneumonia was confirmed in 14 patients who met pre-defined criteria for this condition. For patients with suspected CAP and breath hydrogen cyanide <6.2 ppb sensitivity and specificity were 0.71 and 0.62 respectively (area under ROC curve, 0.71). For patients with suspected CAP and breath isoprene <89 ppb sensitivity and specificity were 0.86 and 0.77 respectively (area under ROC curve, 0.77).

Conclusions Results provide evidence that the levels of several prominent breath metabolites, which are potentially related to bacterial activity and lung injury, are significantly altered in patients with this disease. Breath analysis may therefore provide a novel, non-invasive and reliable strategy for the diagnosis of pneumonia.

S129 PREDICTING OUTCOME FROM HIV-ASSOCIATED PNEUMOCYSTIS PNEUMONIA

doi:10.1136/thx.2010.150946.30

Background The presentation of Pneumocystis jirovecii pneumonia (PCP) ranges from mild to severe. The former responds to anti-microbial therapy, the latter has a high mortality rate despite treatment. Several studies have described clinical and laboratory factors that are predictive of death from PCP. Our objectives were to create a prognostic scoring model to aid the clinician in predicting outcome from HIV-associated PCP.

Methods A prognostic scoring model was built using risk factors identifiable at/soon after hospitalisation, that is, ‘by the bedside’—which have previously been identified as being associated with mortality from PCP (a repeat episode of PCP, the patient’s age, their haemoglobin (Hb), PaO2 (breathing room air), both on admission, the presence of co-morbidity (Comorb), such as lymphoma or pregnancy, and the presence of pulmonary Kaposi sarcoma (PKS) (Walzer PD, et al CID 2008;46:625–33). The model was built from data concerning 592 consecutive episodes of PCP that had occurred among 540 patients presenting to a specialist inpatient HIV treatment centre.

Results The prognostic scoring model was: (25.5 + (age in years/10) + 2 (for a repeat episode of PCP) + 4 (if PKS detected) + 4 (if Comorb present) − PaO2 − Hb). The prognostic model produced scores ranging from 0 to 20; median (IQR) = 9 (7–11). Testing interactions between risk factors and time showed the model to be applicable across all time periods. Patients were divided into five groups according to prognostic score: 0–3.9 = group 1, 4–7.9 = group 2, 8–11.9 = group 3, 12–15.9 = group 4, 16 or greater = group 5. Abstract S129 Table 1 shows mortality rates among the 540 patients with PCP, grouped according to their prognostic scores.

Abstract S129 Table 1

<table>
<thead>
<tr>
<th>Group</th>
<th>Mortality (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>3</td>
<td>13</td>
</tr>
<tr>
<td>4</td>
<td>29</td>
</tr>
<tr>
<td>5</td>
<td>59</td>
</tr>
</tbody>
</table>

Conclusions While this prognostic scoring model requires further validation in patient cohorts from other healthcare institutions, it is potentially a simple ‘by the bedside’ method of identifying patients early in their hospital admission who are at high and low risk of in-hospital death from PCP and so may aid the clinician in assessing the severity of illness and in deciding on treatment strategies.

S130 OUTCOMES OF PSEUDOMONAS ERADICATION THERAPY IN PATIENTS WITH NON-CYSTIC FIBROSIS BRONCHIECTASIS

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Introduction and objectives In patients with non-cystic fibrosis (CF) bronchiectasis, Pseudomonas aeruginosa infection is associated with increased hospital admissions, poorer quality of life and accelerated lung function decline. The BTS guidelines recommend early Pseudomonas eradication therapy. However, despite clear evidence in CF, there is little evidence in non-CF bronchiectasis. Since 2004, we have initiated eradication therapy following initial Pseudomonas infection in non-CF bronchiectasis patients. This retrospective study assessed clinical and microbiological outcomes.

Methods All patients undergoing Pseudomonas eradication therapy from 2004 to 2010 were identified from our electronic database. Therapy included intravenous, oral and nebulised antibiotics. Patients were assessed for microbiological eradication, exacerbation frequency (measured by number of antibiotic courses), hospital admissions, clinical symptoms and lung function.

Results 30 patients were identified. 21 (67.7%) were females and mean age was 62.2 years. Median follow-up time was 26.4 months. Eradication therapy involved 2 weeks of intravenous antibiotics (12 patients), intravenous antibiotics followed by oral ciprofloxacin (13 patients) or oral ciprofloxacin alone (five patients), combined with 1–4 months of nebulised colomycin in 26 patients. Pseudomonas was initially eradicated from sputum in 24 patients (80.0%). 13 patients (43.3%) remained Pseudomona-free at latest follow-up. 11 patients (36.7%) were subsequently reinfected with median time to reinfection of 6.2 months (Abstract S130 Figure 1). 17 patients with chronic infection were maintained on regular nebulised antibiotics. Exacerbation frequency was significantly reduced, with mean number of antibiotic courses per year 3.93 pre-eradication, and 2.09 post-
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 one 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.

**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 naive or withdrew these for >4 weeks. β2-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 (FENO) 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 (±SEM) % fall in FEV1 post EVH was 13.96±1.701 (n=47) in pool and 11.39±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 59% 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=0.202), and FENO (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 92%). Log FENO and log eos correlated strongly (r=0.644, p<0.0001); FENO 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 airways dysfunction in pool athletes but not more eosinophilic airways inflammation suggesting that the pool