Conclusion: We hypothesise that asbestos exposure is causally linked to many more cases of DPF than is currently accepted.

Chronic obstructive pulmonary disease: airways and systemic features

P152 SPUTUM IL-5 CONCENTRATION IS ASSOCIATED WITH A SPUTUM EOSINOPHILIA AND ATTENUATED BY CORTICOSTEROID THERAPY IN CHRONIC OBSTRUCTIVE PULMONARY DISEASE

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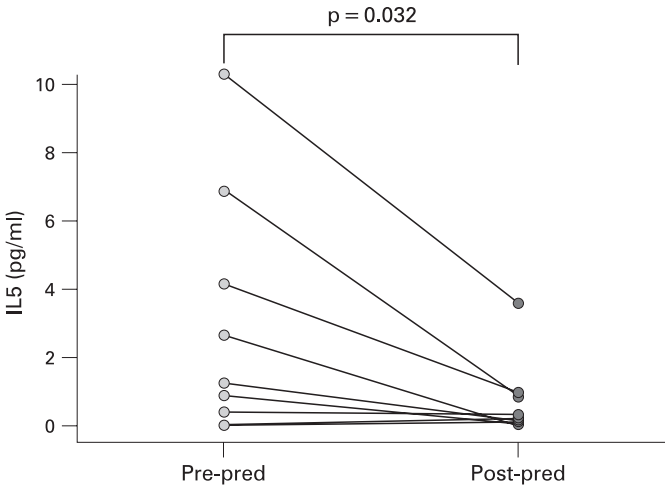
Background: Airway inflammation in chronic obstructive pulmonary disease (COPD) is predominantly neutrophilic, but some subjects demonstrate eosinophilic airway inflammation. Whether these inflammatory phenotypes have differential cytokine and chemokine expression is unknown.
Aims: To assess the sputum concentrations of cytokines and chemokines and their response to oral corticosteroid therapy in COPD subjects with or without a sputum eosinophilia.
Methods: Cytokine and chemokine concentrations were measured using the mesoscale device platform. To assess validity recovery of exogenous spikes was examined. The concentrations of the validated mediators were measured in COPD sputum from subjects with or without a sputum eosinophilia. In a subgroup with a sputum eosinophilia the response to prednisolone 10 mg for 1 month was examined.
Results: The recovery in sputum of exogenous spiked mediators was >80% in 17/26 cytokines and chemokines. In supernatants from eosinophilic (n = 39) versus non-eosinophilic (n = 59) sputa the geometric mean (95% CI) concentration was increased for IL-5 (9.0, 4.5 to 18 pg/ml vs 3.6, 2.7 to 6.3 pg/ml; p = 0.03) and CCL26 (23.4, 10.8 to 19.8 pg/ml vs 9.0, 5.4 to 16.2 pg/ml; p = 0.04). IL-5 correlated with sputum eosinophil counts (R^2 = 0.11, p = 0.001) and was attenuated following treatment with prednisolone (n = 9; mean difference 2.3 pg/ml (95% CI 0.2 to 4.3; p = 0.03).
Conclusion: We have validated the use of the mesoscale device platform for cytokine and chemokine measurements in the sputum supernatants in COPD. Sputum IL-5 was associated with a sputum eosinophilia. Whether this cytokine is important in the pathogenesis of COPD in a subgroup of patients warrants further investigation.



Abstract P152 Figure IL-5 attenuation following corticosteroid administration.

P153 CAN SYSTEMIC MARKERS OF INFLAMMATION HELP PREDICT MORTALITY AT 1 YEAR IN PATIENTS HOSPITALISED WITH EXACERBATIONS OF CHRONIC OBSTRUCTIVE PULMONARY DISEASE?

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Mortality in the months after hospital admission for a chronic obstructive pulmonary disease (COPD) exacerbation is high. Many clinical and physiological markers such as FEV_1 and body mass index (BMI) are thought to predict the risk of dying after a COPD exacerbation. There are few data about potential COPD biomarkers in patients admitted to hospital. We hypothesised that markers of systemic inflammation measured at discharge would relate to the subsequent risk of death in the year post-hospitalisation.

We recruited patients who survived to hospital discharge after a COPD exacerbation to a case management study. As part of this study, blood samples were taken on the day the patients were discharged from hospital or the hospital at home service. Standard C-reactive protein (CRP), high sensitivity CRP (HSCRP), TNFα and IL-6 were measured as well as haemoglobin and creatinine.

107 patients were recruited, 55 female. Mean (SD) age 71 years (8), FEV_1 40% (16) predicted, BMI 24.0 (5.3) and inspiratory capacity median (interquartile range (IQR)) 53% (66–78) predicted. 18/107 (17%) of patients did not survive to 12 months.

Median (IQR) CRP was 4 mg/l (4–18), HSCRP 5.1 mg/l (1.8–17.6), 75 (70%) had CRP <10 mg/l, TNFα <1 pg/ml (<1, <1), IL-6 3.1 pg/ml (1.6–6.9), creatinine 85 mmol/l (73–97) and haemoglobin mean (SD) 13.3 g/dl (1.6).

FEV_1 (litres) died versus survived, respectively, 0.78 l (0.33) versus 0.97 l (0.33), p = 0.02; IC 57% (23) versus 67% (24), p = 0.01; body mass index 21.5 (5.1) versus 24.5 (5.3), p = 0.03, were all predictive of mortality at one year. Only creatinine was significantly associated with mortality, (died 77.5 (71–85) vs survived 87 (73.5–100), p = 0.04). There were no significant differences between the other biomarkers in relation to mortality.

There are still no clinically useful biomarkers that predict death following AECOPD. The observed difference in creatinine reflects low muscle mass, which is known to be associated with adverse outcome. In the clinical setting, this would probably only be useful in the context of very low levels.

P154 INFLAMMATION AND CENTRAL OBESITY IN CHRONIC OBSTRUCTIVE PULMONARY DISEASE WITH AND WITHOUT METABOLIC SYNDROME

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Background: Chronic obstructive pulmonary disease (COPD) is a chronic inflammatory disease of the airways that is characterised by partly reversible airways obstruction, which is predominantly caused by cigarette smoking.[1] It is known that excess body weight is associated with abnormal metabolic and inflammatory profiles that define the metabolic syndrome (MeS), which predisposes to cardiovascular disease.[2] The role of inflammation on features of MeS (central obesity) in smokers with and without COPD is less understood. The aim of this study is to understand the association of inflammation and central obesity in COPD.
Methods: 44 healthy controls (HC), 46 smokers without COPD (HS) and 41 with COPD were recruited for this study. They were matched for age and sex. Subjects were categorised according to the GOLD criteria for diagnosing COPD and International Diabetic Federation (IDF) criteria for defining MeS. All subjects underwent pulmonary function testing. Serum C-reactive protein (CRP) was measured. SPSS 14 was used as the statistical tool for the analysis.

Results: CRP and waist circumference were compared between HC, HS and COPD group. Univariate analysis showed significant difference in central obesity ($p<0.02$) and CRP ($p<0.001$) between the groups. Multivariate analysis showed r^2 adjusted of 0.35 between the groups. When it was analysed between HS and COPD with and without MeS it was observed that CRP was significant in the HS group ($p<0.01$) but not in the COPD group.
Conclusion: This study shows that there is increased central obesity both in smokers with and without COPD. This increase is associated with inflammation in HS but not in COPD. This suggests that there should be another mechanism in COPD subjects, which predisposes to features of MeS.

1. **COPD guidelines group of the standard of care committee of the BTS**. BTS guidelines for the management of COPD. *Thorax* 1997;(Suppl 5):S1–28. Ref type: Generic.
2. **Poulain M,** Doucet M, Drapeau V, *et al*. Metabolic and inflammatory profile in obese patients with chronic obstructive pulmonary disease. *Chronic Respir Dis* 2008;**5**:35–41.

P155 SEASONAL VARIATION IN VITAMIN D STATUS IN PATIENTS WITH CHRONIC OBSTRUCTIVE PULMONARY DISEASE: A PILOT STUDY

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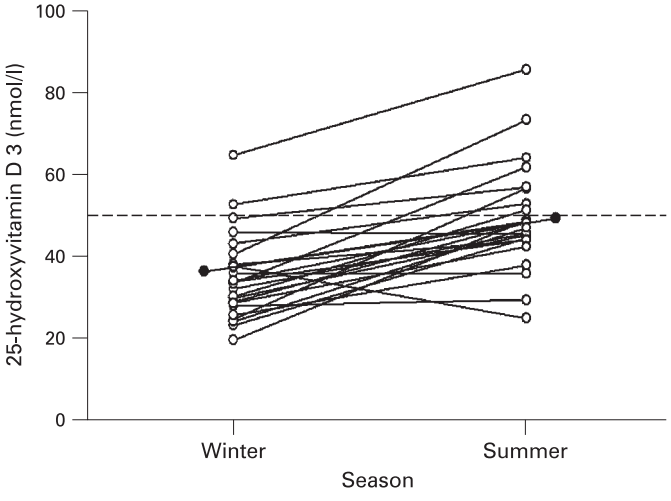
Introduction: Vitamin D has immunomodulatory effects and vitamin D status (assessed by serum 25-hydroxyvitamin D (25(OH)D) concentration) varies with season and correlates with lung function. Although vitamin D status is lower in winter than in summer in the general UK population, relationships between vitamin D status, season and pulmonary function in patients with chronic obstructive pulmonary disease (COPD) are not known.
Objective: To evaluate the seasonal variation in vitamin D status and the relationship to pulmonary function and inflammation in patients with COPD.
Methods: We evaluated 24 (17 male) patients with COPD, mean (SD) age of 69 years (5.8) and smoking history of 43 pack-years (15.8). Patients attended twice for assessment: March/April (end of winter) and August/September (end of summer). Measurements were made of serum 25(OH)D concentration, parathyroid hormone (PTH), calcium, tumour necrosis factor (TNF) α, spirometry (FEV_1, FVC) and exhaled nitric oxide (FE_{NO}). Vitamin D status was compared with estimates for the general population, matched for age, gender, latitude and date, from the National Diet and Nutrition Survey (NDNS).
Results: Vitamin D status was higher in August/September than April/March (see fig). Only one patient with COPD was vitamin D "replete" (25(OH)D concentration >75 nmol/l) in summer and none were "replete" in winter. There was significant correlation between serum 25(OH)D concentration and FEV_1 ($r = 0.486$, $p = 0.016$) but not for FVC ($r = 0.364$, $p = 0.081$), CRP ($r = -0.134$, $p = 0.533$), FE_{NO} ($r = 0.111$, $p = 0.605$) or TNFα ($r = -0.208$, $p = 0.328$). When compared with NDNS data, our patients had significantly ($p<0.001$) lower (winter: 11.9 nmol/l (95% CI 7.4 to 16.3 nmol/l) difference; summer: 20.9 nmol/l (95% CI 15.5 to 26.3 nmol/l) difference) serum 25(OH)D concentration.
Conclusion: Vitamin D status correlates significantly with pulmonary function, although not with inflammatory markers. Vitamin D status is lower in patients with COPD than predicted for healthy individuals in Great Britain. Interventional studies are required to determine whether these associations are causal.



Abstract P155 Figure

P156 UNRECOGNISED CARDIOVASCULAR DISEASE IN PATIENTS WITH SEVERE CHRONIC OBSTRUCTIVE PULMONARY DISEASE

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Introduction and Objectives: The coexistence of undiagnosed cardiac disease in chronic obstructive pulmonary disease (COPD), other than cor pulmonale, is unclear. We investigated cardiac co-morbidity in patients with severe COPD under a chronic respiratory support (CRS) team (>2 COPD admissions/year; annual mortality 16–25%).
Methods: All patients under CRS were offered a consultant cardiologist assessment, investigations and treatment of co-existing cardiac disease.
Results: 33 patients (21 women; 12 men, mean (range) age 72 years (53–88)) with severe COPD (FEV_1 (mean ± SD) 0.66 + 0.26 litres, MRC dyspnoea score 4.4 + 0.7) were assessed. 25 patients (76%) were on long-term oxygen therapy and 30 (91%) used nebulised bronchodilators. Mean follow-up was 10 ± 5 months. Eight (24%) patients died, including three sudden deaths. 22/33 patients (67%) had co-existing cardiac disease, which was not previously identified in five (23%). 10 (30%) patients had ischaemic heart disease; identified from history in six (one ST-segment elevation myocardial infarction (MI), two troponin-positive acute coronary syndrome (ACS), one troponin-negative ACS, two exertional angina); ECG abnormalities in two (one anteroseptal MI, one anterolateral T-wave changes) and two patients had echocardiogram regional wall motion abnormalities with preserved systolic function. Six (18%) patients had hypertension. Six patients (18%) had rhythm abnormalities (five atrial fibrillation, one flutter). 11 (33%) had peripheral oedema. No patient had signs of left ventricular failure. 12/33 (36%) had abnormal echocardiograms: left ventricular systolic impairment (nine patients (27%)—three moderate, six mild; previously unrecognised in four and suboptimally treated in the other five), pulmonary hypertension (eight patients (23%)), dilated right ventricle/atrium (four patients (12%)) and diastolic dysfunction (two patients). 28/33 (85%) were on cardiac medications; diuretics (28), angiotensin-converting enzyme inhibitor/angiotensin receptor blocker (12), aspirin/other antiplatelet agent (13) and statins (five). No patients were taking beta-blockers. Seven/eight patients (88%) who died had co-existing cardiac disease. All three patients who died unexpectedly had evidence of ischaemic heart disease.
Conclusions: More than two-thirds of these patients with severe COPD and high mortality had co-existing cardiac disease not previously identified in a quarter of cases. Early formal cardiac

assessment in COPD would identify co-existing cardiac disease with a view to optimisation of treatment and may help identify patients at increased risk of sudden death.

P157 EXERCISE CAPACITY CORRELATES WITH SUBJECTIVE QUESTIONNAIRES, BUT NOT ACTUAL ACTIVITY IN CHRONIC OBSTRUCTIVE PULMONARY DISEASE PATIENTS

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Impaired exercise capacity (Celli *et al*. *N Engl J Med* 2004) and reduced self-reported physical activity (Yohannes *et al*. *Age Ageing* 2002) predict the risk of death in chronic obstructive pulmonary disease (COPD), whereas COPD patients are less active than their age matched cohort (Pitta *et al*. *AJRCCM* 2005). However, there are few data about how these different methods of assessing physical disability relate to each other.

We assessed 62 stable COPD patients, mean (SD) age 67.6 years (9.0), FEV_1 1.01 litres (0.38), 43.4% (16.6) predicted. Patients performed a 6-minute walk (6MW) to assess exercise capacity and completed the London chest (LCADL), Nottingham extended (NEADL), Aintree COPD (ACADL) and St George's respiratory questionnaire (SGRQ) to assess the impact of exercise intolerance in daily life. In addition, patients wore either the tri-axial DynaPort (n = 1) or uni-axial Actiwatch (n = 33) or both accelerometers (n = 28) for two consecutive days to measure objectively the intensity and duration of exercise at home. To allow for multiple statistical comparisons $p<0.01$ was considered significant.

6MW distance correlated with activities of daily living questionnaire data (LCADL: $r = 0.54$, $p = 0.001$; NEADL: $r = 0.65$, $p = 0.000$; ACADL: $r = -0.54$, $p = 0.001$) but not with health status ($SGRQ_{activity}$: $r = -0.03$, $p = 0.86$; $SGRQ_{total}$: $r = -0.05$, $p = 0.77$). 6MW did not correlate with DynaPort %time moving ($r = -0.11$, $p = 0.79$), %time weight bearing ($r = 0.09$, $p = 0.83$) or movement intensity while moving ($r = -0.02$, $p = 0.96$), nor did 6MW correlate significantly with Actiwatch mean activity score ($r = 0.32$, $p = 0.08$), %time moving ($r = 0.33$, $p = 0.07$) or mean activity score while moving ($r = 0.23$, $p = 0.2$). No questionnaire correlated with any objective activity measure. There were weak correlations between ACADL and Actiwatch mean activity while moving ($r = -0.38$, $p = 0.005$) and Actiwatch intense activity ($r = -0.36$, $p = 0.007$).

What COPD patients say they can do correlates with what they can do, but these measures do not correlate well with what they actually do. Measuring physical activity with accelerometers provides additional information to that obtained from questionnaires and timed walking tests.

P158 AN INDEPENDENT SELF-MANAGEMENT PROGRAMME FOR CHRONIC OBSTRUCTIVE PULMONARY DISEASE: DOES IT WORK? A PILOT STUDY

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A recent Cochrane Review (2007) concluded that there is some evidence for self-management education but further research was required to provide clear guidelines about the use of self-management education. The aim of this study is to determine the effectiveness of a stand-alone self-management manual for chronic obstructive pulmonary disease (COPD) in primary care (SPACE for COPD—a self-management programme of activity, coping and education).

Method: Participants were recruited from primary care registers and randomly assigned to usual care or self-management. The primary outcome measure was health status (self-reported chronic respiratory questionnaire (CRQ-SR)) measured at baseline and 6 weeks. Exercise performance (incremental shuttle walking test (ISWT) and endurance shuttle walk test (ESWT)) was recorded as well as scores on the hospital anxiety and depression scale (HADS).

Results: 37 patients have been recruited to date and randomly assigned into usual care (n = 17; mean (SD) age 72.41 years (7.95); FEV_1 1.43 litres (0.46); 14 male) or self-management (n = 20; mean (SD) age 68.50 years (8.10); FEV_1 1.57 litres (0.71); 10 male). 25 patients have completed measures at 6 weeks (14 from usual care and 11 from self-management). Paired t tests were performed and are outlined in the table for all outcome measures. There were statistically significant improvements in the self-management group for CRQ-SR domains of dyspnoea, emotion and mastery, with mean changes being 0.81, 0.89 and 0.73, respectively. There were no significant changes in the usual care group in all CRQ-SR domains of dyspnoea, fatigue, emotion and mastery. Mean changes were 0.41, −0.04, 0.00 and −0.15, respectively. Statistically significant between-group differences were found for ISWT, ESWT and CRQ emotion domain ($p<0.05$). In addition CRQ domains of fatigue, mastery and emotion attained the minimal clinically important threshold of more than 0.5.

Conclusion: Participants receiving a self-management manual showed significant improvements in health status and exercise performance. This shows that a self-management package delivered as a stand-alone manual is feasible and effective.

Abstract P158 Table Mean baseline scores and mean changes for HADS, CRQ-SR, ISWT and ESWT for usual care and self-management groups

	Self-management		Usual GP care	
Measure	Baseline	Mean change (95% CI)	Baseline	Mean change (95% CI)
Anxiety	8.00	−2.45 (0.17 to 4.82)*	4.64	−0.57 (0.48 to 1.63)
Depression	5.73	−0.91 (1.03 to 2.84)	3.85	−0.29 (1.23 to 0.66)
Dyspnoea	3.39	0.81 (1.59 to 0.03)*	3.31	0.41 (0.94 to 0.11)
Fatigue	3.96	0.54 (1.55 to 0.47)	4.54	0.04 (0.74 to 0.82)
Emotion	4.58	0.89 (1.65 to 0.12)*	5.27	0.15 (0.14 to 0.45)
Mastery	5.60	0.73 (1.39 to 0.06)*	5.77	0.00 (0.45 to 0.45)
ISWT (m)	298.18	30.91(55.77 to 6.05)*	332.86	−21.43 (9.36 to 52.22)
ESWT (s)	241.36	409.73 (623.35 to 196.11)**	402.93	46.50 (133.25 to 40.25)

*p>0.05; **p>0.01. CRQ-SR, self-reported chronic respiratory questionnaire; ESWT, endurance shuttle walk test; HADS, hospital anxiety and depression scale; ISWT, incremental shuttle walking test.

P159 THE IMPLICATIONS OF EMPHYSEMA ZONE FOR DIAGNOSIS OF ALPHA-1-ANTITRYPSIN DEFICIENCY

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Introduction: Patients with alpha-1-antitrypsin deficiency (AATD) classically exhibit lower zone predominant emphysema, unlike individuals with usual chronic obstructive pulmonary disease (COPD). However, phenotypic variation is recognised[1] and with the advent of specific treatment for AATD[2] it is more critical to identify deficient individuals. In the presence of atypical disease a diagnosis of AATD may not be considered.

Methods: All PiZZ subjects from the UK AATD registry with a quantitative chest computed tomography (CT) scan were studied (n = 369). Emphysema was quantified at −910 HU with slices representing the upper zone and lower zone, respectively. Subjects with a normal voxel index in either zone[3] were excluded. Emphysema zone was determined by the difference in voxel index between lower and upper zone slices (LZVI–UZVI). Subjects diagnosed due to lung disease (index cases) were compared with subjects diagnosed through family screening or for other reasons (non-index cases).

Results: Index cases were younger, had smoked more and had lower zone dominant emphysema (LZDE) in over 88% of cases (see table). Almost 30% of non-index subjects had a CT scan diagnostic of upper zone dominant emphysema (UZDE). Index status remained a significant predictor of emphysema zone after correction for age, smoking and gender ($p = 0.005$, $B = 4.90$), such that it accounted for 15% of variability in emphysema zone score, compared with 6.9% for smoke exposure.

Conclusions: There are several interpretations of these results. Non-index cases may be at an earlier disease stage, suggested by their age, and implying that upper zone disease occurs first, consistent with previous evidence.[4] Second, there may be ascertainment bias, such that subjects with UZDE are less likely to be tested for AATD. Third, UZDE may be a different disease entity, influenced by other genetic factors.[5] Testing for AATD should therefore be offered to all subjects with COPD to ensure optimum management.

1. **Needham,** Stockley. *Thorax* 2004;**59**:441–5.
2. **Dirksen,** *et al*. *AJRCCM* 2008;**177**:A419.
3. **Soejima,** *et al*. *AJRCCM* 2000;**161**:1264–73.
4. **Holme,** *et al*. *Thorax* 2007;**62**:A140.
5. **DeMeo,** *et al*. *AJRCCM* 2007;**176**:42–8.

Abstract P159 Table

	Index cases	Non-index cases	p Value
LZDE (%)	88.55	70.89	<0.001
LZVI–UZVI	15.52 (0.86)	8.71 (1.39)	<0.001
Age (years)	51.74 (0.63)	48.33 (1.11)	0.009
Gender (%male)	64.62	53.85	0.085
Pack years smoked	18.00 (7.50–25.50)	5.25 (0–18.25)	<0.001
FEV_1 (% predicted)	35.41 (1.20)	75.14 (4.13)	<0.001
KCO (% predicted)	63.74 (1.41)	81.15 (2.54)	<0.001

LZDE, lower zone dominant emphysema; LZVI–UZVI, lower and upper zone voxel index.

Clinical aspects of pleural disease

P160 ABSENCE OF *PNEUMOCYSTIS JIROVECII* IN PLEURAL INFECTION: A GENETIC AMPLIFICATION STUDY

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Introduction: Up to 26% of pleural infections have no pathogens identified using standard microbiological and genetic amplification techniques. Possible explanations for this phenomenon include previous use of antibiotics, microbiologically different pleural fluid locules, or difficulty isolating organisms using conventional techniques. *Pneumocystis jirovecii* (previously *carinii*) causes pneumonia in immunocompromised hosts. Previous studies have demonstrated an asymptomatic carrier state in immunocompetent individuals—*P jirovecii* DNA was detected in the bronchoalveolar lavage fluid of 18% of patients with lung disease, 4.4% with bacterial pneumonia (both without HIV) and in the oropharynx of 20% of healthy individuals. We hypothesised that *P jirovecii* may account for some microbiologically obscure pleural infections and that co-infection with *P jirovecii* and bacteria may occur in pleural infection. The aim of this study was to assess the prevalence of *P jirovecii* in pleural infection, using highly sensitive and specific genetic detection techniques.

Methods: Pleural fluid was obtained from participants in the MIST1 trial (a double-blind, placebo-controlled trial of intrapleural streptokinase in pleural infection). Criteria for pleural infection were purulence, culture positivity, Gram staining positivity, or pH below 7.2, with clinical evidence of infection. Samples from 100 patients (randomly selected from 454 patients recruited) were analysed using highly sensitive real-time PCR targeting part of the *P jirovecii* heat shock 70 (HSP70) gene sequence. Potential inhibition of *P jirovecii* HSP70 PCR was assessed by the use of the SPUD assay.

Results: There was no evidence of *P jirovecii* DNA in any sample. Samples with possible inhibition of PCR were diluted and retested; these samples remained negative for *P jirovecii* DNA despite recovery of the SPUD amplicon.

Conclusions: Having given consideration to the possible inhibition of PCR, there was no evidence of *P jirovecii* DNA in any sample using the highly sensitive real-time HSP70 PCR technique. Therefore, *P jirovecii* is neither a common pathogen nor co-pathogen in those with pleural infection, an important negative finding given its prevalence as a co-pathogen in pneumonia. There is no need to investigate for *P jirovecii*, nor for its blind treatment, in pleural infection unless a patient is severely immunocompromised.

P161 PLEURAL EFFUSION IN PRIMARY LUNG CANCER: PREVALENCE AND RISK FACTORS

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Introduction: In patients presenting with lung cancer, pleural involvement has significant implications on disease staging, prognosis and treatment. Computerised tomography (CT) is significantly more sensitive than plain radiographs in identifying pleural effusions but the prevalence of pleural effusion detected by CT in patients presenting with lung cancer is unknown.

Aims: To determine the prevalence of pleural effusion on CT in patients presenting with lung cancer and during the subsequent disease course. To identify factors associated with the presence of pleural effusions.

Method: Retrospective review of 200 consecutive patients presenting to two UK hospitals.

Results: Pleural effusions were common. CT scan was more sensitive for detecting pleural effusions than chest *x* ray (CXR), detecting an effusion in 50 (25%) versus 27 patients (13.5%), respectively ($p<0.001$, McNemar test). 16 (32%) were cytologically confirmed to be malignant. A further 11 (5.5%) patients developed an effusion as their disease progressed, giving an overall prevalence of 30.5%. Patients with small-cell lung cancer (SCLC) were more likely (relative risk 1.8, $p = 0.026$) to develop an effusion. Age, gender, and location of the tumours were not predictive of the presence of pleural effusions (see table). All patients with SCLC and a pleural effusion had extensive disease.