decline of -8% (p = 0.03). In contrast, there were no significant differences in FEV_1 responses in volunteers with COPD, though there was a mean drop in FVC of -8% (p = 0.03) two hours after the start of exposure in Oxford street compared to Hyde Park. We recorded no changes in arterial stiffness in either group for either exposure site.

Conclusions These early findings suggest that the observations made in healthy volunteers from chamber studies can be replicated in ambient conditions. The apparent lack of any respiratory response in patients with COPD may reflect their up-regulated baseline inflammatory state, or systematic differences in the exposure differential between the two sites. We continue to recruit volunteers; further measurements including markers of oxidative stress in sputum, serum, and small airways impedance are under way.

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P121 COST-EFFECTIVENESS OF ALPHA-1 ANTITRYPSIN (A1AT) DEFICIENCY CASE-FINDING IN SECONDARY CARE

L Dron, S Whiting, D Thorburn, M Pinzani, B Gooptu, DA Lomas, JR Hurst; *UCL Medical School / Royal Free London NHS Foundation Trust, London, UK*

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Background Screening for A1AT-deficiency at our institution involves serum protein assay, then phenotyping where A1AT≤1.1g/L. RT-PCR genotyping for S/Z variants is available for clarification. We recently established a multi-disciplinary respiratory/hepatology London A1AT Deficiency Service and re-examined the cost-effectiveness of our diagnostic algorithm.

Method We studied all patients who had a serum A1AT request over the three years 1/1/2010–31/12/2012. We went on to examine all requests for phenotyping and/or genotyping over the ten years 1/1/2003–31/12/2012.

Results From 4460 requests over 2010–2012, 240(5.4%) had serum A1AT≤1.1g/l. Of these, phenotyping was not available in 33 and of the remainder the following phenotype-prevalence data were observed (n,%): MM (75,36%), MZ (89,43%), MS (29,14%), ZZ (6,3%), SZ (6,3%), SS (1,1%), GM (1,1%). In error, 28 patients had phenotyping with A1AT>1.1g/l: 27 were PiMM and 1 PiMS.

Over 2003–2012, 525 phenotyping tests were performed. In 41 the result was unclear or there was evidence of *in vivo* degradation. The prevalence of the respective phenotypes and mean (SD) A1AT concentrations are reported in the Table. Over the same period, 24 equivocal samples were sent for genotyping. This confirmed 13 = PiMM, 4 = PiZZ, 3 = PiMZ, 2 = PiMS and 2 = PiSZ.

The NHS costs of A1AT serum assay, phenotyping and genotyping are £2.45, £35.50 and £87.50. Using the 2010–2012 data, our mean annualised spend on A1AT serum, phenotyping and genotyping was £3,642 £3,170 and £376 respectively. The cost per ZZ/SZ case diagnosed was £1198.

The highest A1AT serum concentration recorded in the ZZ/SZ patients were 0.4 and 1.0g/l respectively suggesting that the ≤1.1g/l cut-off is appropriate. The specificity, sensitivity and positive predictive values are 26.5%, 100% and 6.5%.

Conclusion 5.4% of serum A1AT assays yielded results ≤1.1g/l. Of these, 6% were found to represent SZ/ZZ variants at risk of clinically relevant disease (0.27%, 1/372 requests). The ≤1.1g/l cut-off was 100% sensitive but only 26.5% specific for clinically relevant deficiency alleles. The ≤1.1g/l cut-off could be lowered with improved specificity and cost-saving if the desire was to detect only ZZ patients.

Abstract P121 TABLE 1. Prevalence of A1AT phenotypes with respective serum protein concentration in 484 analyses over ten years.

Phenotype	Prevalence n (%)	Serum A1AT mean (SD) g/l
MM	244 (50%)	1.18 (0.33)
MZ	150 (31%)	0.89 (0.12)
MS	62 (13%)	1.01 (0.13)
ZZ	12 (2%)	0.21 (0.11)
SZ	12 (2%)	0.64 (0.16)
SS	1 (<1%)	1.0
Other	3 (1%)	1.0

P122 THE PREVALENCE AND IMPACT OF OPIATE SMOKING ASSOCIATED COPD

[1]N Lewis-Burke, [1]O Wooding, [1]K McKinnell, [2]L Davies, [2]P P Walker; *[1]Sefton Primary Care Trust, Liverpool, UK; [2]University Hospital Aintree, Liverpool, UK*

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Within the last two decades there has been a shift from injection to inhalation of recreational opiates (heroin and crack cocaine) as this was perceived to be 'safer'. However, there are little data as to the effect on lung function and diagnosis and treatment of airway disease. We recruited 145 current and past opiate users from a local community drug service and they completed questionnaires on demographics, drug use, symptoms and health status as well as spirometry. Lower limit of normal was used to define airflow obstruction in this young population. Of the 145 subjects 10 failed to produce adequate pre and post-bronchodilator (BD) spirometry and 26 subjects had injected but not smoked opiates. Of the remaining 109 subjects 36 (33%) had post-BD airflow obstruction with minor or no reversibility and so consistent with COPD. A further 6 subjects had marked reversibility suggesting asthma and 2 subjects had normal spirometry but had a secondary care diagnosis of bronchiectasis meaning a prevalence of airway disease of 40%. The 36 COPD subjects had smoked heroin for a mean 24 (SD 9) years, 34/36 had also smoked crack for 13 (9) years and 33/36 had smoked cannabis for 22 (11) years. All had been cigarette smokers; 24 (21) pack years. Their mean age was 44.7 (6.3) years and 25/36 (69%) were male. Post-BD FEV1 was 2.62 (0.92) L; 74.6 (23.8)% predicted and FEV1/FVC was 0.6 (0.13). Most subjects had mild to moderate COPD–GOLD stage I = 17, II = 13, III = 5 and IV = 1. Only 5 had ever been given a diagnosis of COPD but 21 had been told they had asthma. Mean CAT score was 20 (10) with 50% (18/36) having CAT scores of 20 or greater. Mean MRC score was 3 (1.6) with 53% (19/36) having MRC scores of 3–5. Despite this only 56% (20/36) were on any form of therapy and only 25% (9/36) were prescribed any long-acting bronchodilator. COPD is very common in an opiate smoking population with a third having irreversible airflow obstruction despite their young age. However, few have been diagnosed with COPD and despite a high level of symptoms and poor health status, treatment is far from optimal. The frequency and early development of lung disease related to opiate smoking represents an important public health message and better and earlier recognition and treatment could reduce healthcare utilisation and costs.