home for use in the event of an exacerbation. Whilst this approach is established with a strong evidence base in asthma, evidence suggestive that this is effective in reducing hospital admissions and readmissions in COPD is inconsistent.¹

**Aims and Objectives** This study aims to assess the impact of implementing a unified self management strategy, consisting of self management plan, education and rescue medications, in reducing hospital inpatient readmissions at 30 and 90 days.

**Methods** The study was carried out over six months, across three acute hospitals, between November 2010 and April 2011. All patients admitted with a primary diagnosis of COPD exacerbation were included and given the following unless there were any contra-indications for providing this:
- A unified written self management plan
- Rescue medication of a 7 day course of prednisolone and 5 day course of antibiotic
- Education on self management and how to use their rescue medication

To assess the impact of the self management strategy, data was collected for both patients who did and did not receive this intervention. For the purpose of accuracy, 10% of data was cross-checked by an independent person.

**Results** During the 6 month audit period, 457 patients with acute hospital admission for COPD exacerbation were recruited, with 68%, 58.6% and 24.5% (mean 40.1%) of patients at each of the 3 sites receiving a self management plan and rescue medication. Main reasons for not receiving included patients not speaking English, couldn’t understand self management advice or refused to self manage.

**Conclusions** Self management and rescue medication is associated with a reduction in 30 and 90 day readmission rates by 12.5% and 4.3% respectively. A high proportion of patients did not receive these for practical reasons which need addressing for future evaluations.

**References**


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**Mechanisms of chronic lung disease**

**P249**  **COULD AN INTRONIC SNP IN THE ALPHA-1-ANTITRYPSIN GENE CONFER PROTECTION TO CHRONIC OBSTRUCTIVE PULMONARY DISEASE?**

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Chronic obstructive pulmonary disease (COPD) results from complex interactions between both environmental and genetic factors. This is evidenced by the considerable variation found in the risk of developing COPD despite the established dose-response relationship from the biggest known risk factor, tobacco smoking. Thus, genetic susceptibility remains poorly understood given the best-characterised genetic determinant of COPD, severe alpha-1-antitrypsin (AAT) deficiency, only affects 1–2% of all COPD patients.

A genome-wide association study implicated an intronic single nucleotide polymorphism (SNP) rs5748312 within AAT gene as the strongest locus associated with lung function (a heritable surrogate predictor of COPD). Thus, this was investigated as part of a larger research project aimed at identifying rare sequence variants of the AAT gene that may be associated with COPD.

A sample of 230 COPD patients of European descent either predicted to carry one of six haplotypes conferring COPD risk, or who presented with severe early-onset COPD were genotyped for SNP rs5748312 within the AAT gene utilising TaqMan® assay with >5% of samples sequenced for concordance. The data was compared against control data of 60 patients of European ancestry from dbSNP.

In examining the allelic distribution ($p=0.049$, OR 0.57 95% CI 0.323–1.003) borderline significance was noted, however no significant difference between cases and controls was found in the genotype distribution ($p=0.096$, OR 0.583, 95% CI 0.308–1.106).

This preliminary study suggests the SNP merits further work in a more adequately powered investigation with adjustment for covariates such as age, smoking history and lung function given the borderline nature of the findings indicative of a protective effect for developing COPD with the minorallele (A). It is feasible that associated functional SNPs in linkage disequilibrium reflect the true association.