

## Chronic Obstructive Pulmonary Disease (COPD): Epidemiological and Pan-airway Aspects

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### Abstract

*Background* – Chronic obstructive pulmonary disease (COPD) is a major cause of morbidity and mortality worldwide. However, there is little information on the prevalence and particularly on the incidence of COPD. Also, apart from the importance of tobacco smoking and  $\alpha$ 1-antitrypsin deficiency, information on contributing risk factors for the development of COPD is limited.

*Hypotheses* – 1. Spirometry can be used in primary health care settings to improve diagnosis of COPD. 2. Alcohol intake is associated with COPD. 3. Coronary heart disease (CHD) is common in patients with COPD. 4. A family history of COPD is an independent risk factor for development of COPD. 5. Specific nasal symptoms and nasal symptom-provoking exposures can predict the development of COPD. 6. The nasal mucosa in COPD may feature a specific pathology.

*Methods* – Studies focusing on spirometry assessments, analysis of a marker of alcohol intake (serum carbohydrate deficient transferrin: S-CDT), and register studies (the Swedish Survey on Living Conditions (ULF) linked with the Swedish Inpatient register), as well as respiratory questionnaire surveys were employed. Also, a nasal lavage study comprising histamine-challenges and analysis of markers of neutrophil activity (myeloperoxidase), plasma exudation and secretory/mucinous activity (fucose) was carried out.

*Results* – Spirometry detected many new cases of COPD. A majority of them were unaware of their condition. The prevalence of COPD was 11.5% and 4.5% as assessed by spirometry and questionnaires, respectively. The annual incidence of COPD as assessed by questionnaires (1992 and 2000, respectively) was 0.36%. S-CDT was elevated in COPD and inversely correlated to FEV<sub>1</sub>. Report of alcohol intake according to the ULF interview in 1988/89 predicted a higher risk of a hospitalisation for COPD from the time point of the ULF interview to the end of 2000. A hospitalisation for CHD during this time was also associated with an increased risk of COPD hospitalisation. A family history of COPD predicted the incidence of a self-reported physician's diagnosis of COPD. "Thick, yellow discharge" and "a blocked nose", as well as the nasal symptom provoking factors "damp/cold air" and "tobacco exposure", also predicted this incidence. Also, an association was observed between self-reported COPD and the incidence of a physician's diagnosis of nasal polyposis. The difference in myeloperoxidase between a histamine and saline lavage was greater in patients with COPD than in healthy subjects. Furthermore, COPD-patients reporting nasal symptoms presented increased levels of myeloperoxidase at histamine challenge (c.f. saline) and greater differences in myeloperoxidase and fucose, respectively, between the histamine and saline lavage (c.f. patients without symptoms).

*Conclusions* – The prevalence as well as the incidence of COPD in Sweden is high. Underdiagnosis of COPD is considerable and spirometry should be considered in all smokers. High alcohol consumption and CHD may be associated with an increased risk for being hospitalised due to COPD. A family history of COPD and distinct nasal features may be risk factors for the development of COPD. COPD is not associated with any marked nasal inflammation, but nasal pathology may need attention in this condition.