COPD and environmental risk factors other than smoking

1. Identifying relevant papers

Author : P N Lee
Date : 17th October 2007

1. Background and objectives

Though it is well known that the major risk factor for COPD is smoking, other relevant environmental risk factors may be important. The objective of the work is to identify a list of candidate risk factors likely to be relevant to COPD, and for each of them to identify relevant references to the studies providing useful evidence and (where available) to papers reviewing this evidence. For each risk factor, a summary of the available data would be prepared, the intent being to indicate the strength of the evidence linking the risk factor to COPD, rather than to carry out a full detailed review.

2. Identifying suitable risk factors

As a first step to deciding which risk factors other than smoking were prospective candidates for more detailed literature searching, a PubMed search was carried out of "(COPD or emphysema or chronic bronchitis) and risk factors" limited to studies of humans and to review papers. The search identified 559 papers and from their abstracts 24 references were obtained (from our RefMan database or by free download if possible, or from the British Library if not). Thirteen additional references of review papers were also obtained from Chapter 4 of the 2004 US Surgeon General Report[1] on the Health Consequences of Smoking, and in particular the section on Chronic Respiratory Diseases starting on p463. The actual section on COPD starting on p498 did not consider risk factors other than smoking, but the section on decline of lung function (see p475 particularly) provided useful material.

Including the US Surgeon General Report itself, these 38 papers[2-39] can be termed the "Step 1 papers". Many were general review papers[2-4,8-10,12,13,16,18-25,27,29,30,32-34,36,39], sometimes of the evidence in relation to specific

factors, but others[5-7,11,14,15,17,26,28,31,35,37,38] proved to be papers describing the results of a particular study (or studies).

The next step was to study each of the relevant papers and record which factors were referred to, and where appropriate the authors' belief as to the strength of the evidence. Based on this list the following risk factors were selected for more detailed consideration:

1. Race
2. Body mass index
3. Alcohol
4. Diet
5. Education/income/socioeconomic status
6. Occupational exposures
7. Air pollution
8. Childhood infections
9. Cooking and heating
10. Atopy, allergy and hyperresponsiveness
11. Adult infections
12. Eosinophilia.

Other factors that may be relevant to COPD, but were not pursued, were as follows (with reasons for not considering those given):

ETS exposure	-	There is already an up-to-date review here by myself and Barbara Forey[40].
FEV_1	-	The definition of COPD depends directly on FEV_1.
Sex, age and height	-	FEV_1 is standardized for sex, age and height.
Genetic factors	-	The review was intended to consider environmental factors, with genetic factors, such as α_1-antitrypsin deficiency, outside the area of interest.

Respiratory diseases	-	There is a problem distinguishing such diseases as asthma, cystic fibrosis, bronchiectasis and obliterative bronchiolitis, all diseases associated with airflow obstruction, from COPD, but these are not environmental risk factors.
Respiratory symptoms	-	While cough, dyspnoea, and mucous hypersecretion are all strongly associated with COPD, these can be considered part of the disease itself, and are also considered outside the scope of the current project.

3. <u>General approach for identifying relevant papers for each factor</u>

For many of the factors considered the approach used was as follows:

a) Carry out a PubMed search using the search term

"(COPD or emphysema or chronic bronchitis) FACTOR",

where "FACTOR" may itself be a combination such as

"(education or income or socioeconomic status)",

with restriction to studies in humans.

b) Print out abstracts for the identified references, and look at these to identify those which seem likely to be appropriate. Interest is in causes of COPD so, for example, papers describing studies of factors affecting survival of COPD patients, or uncontrolled studies of cases would not be relevant.

c) Go through relevant sections of the Step 1 papers which considered the risk factor and look for additional relevant references.

d) For each of the references identified under b) and c) obtain a copy of the relevant paper, from our own Reference Manager (RefMan) system if the paper was on it or, if not, from free downloads if available, or from the British Library if not. All the papers would be put on RefMan and given the keyword COPDRISKF and a keyword specific for the factor in question.

e) When all the papers collected under d) became available (or it became clear that some papers were unobtainable in a reasonable time) the papers were looked through to identify further relevant "secondary references," not previously identified. At this stage some papers obtained, which proved not to be relevant, were removed, and the keyword for the factor amended by adding REJ at the end of it.

f) These secondary references were then obtained using procedures similar to those described in section d).

g) Go through the papers identified in an earlier project as relevant for smoking and COPD, bronchitis and emphysema and see which provide data on risk of a relevant endpoint related to the factor of interest. This step was carried out separately by A J Thornton for all the identified risk factors simultaneously.

h) All additional papers identified under f) and g) were also put on RefMan (where not previously on it) and given the keyword COPDRISKF and the factor specific keyword.

i) As no attempt was made to identify any further papers from references lists in papers identified under f) and g), the set of papers was now complete, although again some were marked as rejected on RefMan, if appropriate.

For some factors, alternative procedures were used. These will be described in 12 separate documents, one for each risk factor. These

documents will be numbered 2 to 13, as completed, not in the order listed on page 2.

4. References

1. *US Surgeon General. The health consequences of smoking. A report of the Surgeon General. Atlanta, Georgia: US Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health; 2004.* http://www.cdc.gov/tobacco/sgr/sgr_2004/index.htm

2. *Ameille J, Dalphin JC, Descatha A, Pairon JC. La bronchopneumopathie chronique obstructive professionnelle: une maladie méconnue (Occupational chronic obstructive pulmonary disease: a poorly understood disease). Rev Mal Respir 2006;**23**:13S119-30.*

3. *Becklake MR. Occupational exposures: evidence for causal association with chronic obstructive pulmonary disease. Am Rev Respir Dis 1989;**140**:S85-S91.*

4. *Blanchard AR. Treatment of COPD exacerbations. Pharmacologic options and modification of risk factors. Postgrad Med 2002;**111**:65-75.*

5. *Burchfiel CM, Marcus EB, Sharp DS, Enright PL, Rodriguez BL, Masaki KH, et al. Characteristics associated with rapid decline in forced expiratory volume. Ann Epidemiol 1996;**6**:217-27.*

6. *Burrows B, Knudson RJ, Camilli AE, Lyle SK, Lebowitz MD. The "horse-racing effect" and predicting decline in forced expiratory volume in one second from screening spirometry. Am Rev Respir Dis 1987;**135**:788-93.*

7. *Carey IM, Strachan DP, Cook DG. Effects of changes in fresh fruit consumption on ventilatory function in healthy British adults. Am J Respir Crit Care Med 1998;**158**:728-33.*

8. *Cazzola M, Donner CF, Hanania NA. One hundred years of chronic obstructive pulmonary disease (COPD). Respir Med 2007;**101**:1049-65.*

9. *Devereux G. ABC of chronic obstructive pulmonary disease. Definition, epidemiology, and risk factors. BMJ 2006;**332**:1142-4.*

10. *Doherty DE, Briggs DD, Jr. Chronic obstructive pulmonary disease: epidemiology, pathogenesis, disease course, and prognosis. Clin Cornerstone 2004;**Suppl 2**:S5-S16.*

11. *Frew AJ, Kennedy SM, Chan-Yeung M. Methacholine responsiveness, smoking, and atopy as risk factors for accelerated FEV_1 decline in male working populations. Am Rev Respir Dis 1992;**146**:878-83.*

12. *Higgins M. Risk factors associated with chronic obstructive lung disease. Ann N Y Acad Sci 1991;**624**:7-17.*

13. *Hnizdo E, Vallyathan V. Chronic obstructive pulmonary disease due to occupational exposure to silica dust: a review of epidemiological and pathological evidence. Occup Environ Med 2003;**60**:237-43.*

14. *Jaakkola MS, Ernst P, Jaakkola JJK, N'gan'ga LW, Becklake MR. Effect of cigarette smoking on evolution of ventilatory lung function in young adults: an eight year longitudinal study. Thorax 1991;**46**:907-13.*

15. *Jaakkola MS, Jaakkola JJK, Becklake MR. Ventilatory lung function in young cigarette smokers: a study of susceptibility. Eur Respir J 1991;**4**:643-50.*

16. *Jones A. Causes and effects of chronic obstructive pulmonary disease. Br J Nurs 2001;**10**:845-50.*

17. *Lange P, Parner J, Vestbo J, Schnohr P, Jensen G. A 15-year follow-up study of ventilatory function in adults with asthma. N Engl J Med 1998;**339**:1194-200.*

18. *Mannino DM. Chronic obstructive pulmonary disease: definition and epidemiology. Respir Care 2003;**48**:1185-91.*

19. *Mannino DM. Epidemiology and global impact of chronic obstructive pulmonary disease. Semin Respir Crit Care Med 2005;**26**:204-10.*

20. *Mayer AS, Newman LS. Genetic and environmental modulation of chronic obstructive pulmonary disease. Respir Physiol 2001;**128**:3-11.*

21. *O'Connor GT, Sparrow D, Weiss ST. The role of allergy and nonspecific airway hyperresponsiveness in the pathogenesis of chronic obstructive pulmonary disease. Am Rev Respir Dis 1989;**140**:225-52.*

22. *Pauwels RA, Rabe KF. Burden and clinical features of chronic obstructive pulmonary disease (COPD). Lancet 2004;**364**:613-20.*

23. *Petty TL. Definitions, causes, course, and prognosis of chronic obstructive pulmonary disease. Respir Care Clin N Am 1998;**4**:345-58.*

24. *Pistelli R, Baldari F, Sammarro S. Fattori di rischio per broncopneumopatia cronica ostruttiva (Risk factors of chronic obstructive pulmonary disease). Ann Ist Super Sanita 2003;**39**:485-93.*

25. *Romieu I, Trenga C. Diet and obstructive lung diseases. Epidemiol Rev 2001;**23**:268-87.*

26. *Scanlon PD, Connett JE, Waller LA, Altose MD, Bailey WC, Buist AS, et al. Smoking cessation and lung function in mild-to-moderate chronic obstructive pulmonary disease. The Lung Health Study. Am J Respir Crit Care Med 2000;**161**:381-90.*

27. *Sethi JM, Rochester CL. Smoking and chronic obstructive pulmonary disease. Clin Chest Med 2000;**21**:67-86, viii.*

28. *Sherman CB, Xu X, Speizer FE, Ferris BG, Jr., Weiss ST, Dockery DW. Longitudinal lung function decline in subjects with respiratory symptoms. Am Rev Respir Dis 1992;**146**:855-9.*

29. *Silverman EK, Speizer FE. Risk factors for the development of chronic obstructive pulmonary disease. Med Clin North Am 1996;**80**:501-22.*

30. *Snider GL. Chronic obstructive pulmonary disease: risk factors, pathophysiology and pathogenesis. Annu Rev Med 1989;**40**:411-29.*

31. *Tashkin DP, Altose MD, Connett JE, Kanner RE, Lee WW, Wise RA. Methacholine reactivity predicts changes in lung function over time in smokers with early chronic obstructive pulmonary disease. Am J Respir Crit Care Med 1996;**153**:1802-11.*

32. *Viegi G, Scognamiglio A, Baldacci S, Pistelli F, Carrozzi L. Epidemiology of chronic obstructive pulmonary disease (COPD). Respiration 2001;**68**:4-19.*

33. *Viegi G, Di Pede C. Chronic obstructive lung diseases and occupational exposure. Curr Opin Allergy Clin Immunol 2002;**2**:115-21.*

34. *Viegi G, Maio S, Pistelli F, Baldacci S, Carrozzi L. Epidemiology of chronic obstructive pulmonary disease: health effects of air pollution. Respirology 2006;**11**:523-32.*

35. *Vollmer WM, Enright PL, Pedula KL, Speizer F, Kuller LH, Kiley J, et al. Race and gender differences in the effects of smoking on lung function. Chest 2000;**117**:764-72.*

36. *Weiss ST. Atopy as a risk factor for chronic obstructive pulmonary disease: epidemiological evidence. Am J Respir Crit Care Med 2000;**162**:S134-S136.*

37. *Xu X, Weiss ST, Rijcken B, Schouten JP. Smoking, changes in smoking habits, and rate of decline in FEV_1: new insight into gender differences. Eur Respir J 1994;**7**:1056-61.*

38. *Xu X, Wang L. Synergistic effects of air pollution and personal smoking on adult pulmonary function. Arch Environ Health 1998;**53**:44-53.*

39. *US Surgeon General. The health consequences of smoking. Chronic obstructive lung disease. A report of the Surgeon General. Rockville, Maryland: US Department of Health and Human Services; Public Health Service; 1984. DHHS (PHS) 84-50205. http://www.cdc.gov/tobacco/sgr/index.htm*

40. *Lee PN, Forey BA. Epidemiological evidence on environmental tobacco smoke and COPD. Sutton, Surrey: P N Lee Statistics and Computing Ltd; 2007. www.pnlee.co.uk/reflist.htm [Download LEE2007C]*