It is a matter of some concern to us that you felt obliged to print a notice of duplicate publication for Professor Corris.1 While we all deplore dual publication of original scientific data, the purpose of review articles is to provide a form of CME for practising physicians. It is therefore inevitable that, when an authority in a field is asked to give their current view on a subject, there will be considerable overlap with their previous thoughts on the subject. This does not make the article uninteresting to read, nor—as we are sure the Editors are aware—does it stop such articles being frequently referenced.

It is our belief that it is generally understood within the community that review articles by a given author are likely to contain significant overlap with previously published reviews by the same author and that, in this situation, it is rather “missing the point” to call this a duplicate publication.

To illustrate the point we enclose a list of review articles which all contain overlapping material concerning the assessment of respiratory muscle strength.2 3 With the exception of the article in Thorax (for which the invitation to write came following a prompt from us), the remaining articles were written as a result of unsolicited requests by the editorial team of the journal concerned. Like Professor Corris’s articles, they serve a useful function because these journals reach widely differing audiences and in each case the text of the article has been aligned to fit the interests of the readership of the journal concerned.

Our belief is that reviews of this sort do serve a useful role in postgraduate medical education and, because writing them is not recognised by the University Research Assessment Exercise, it is becoming increasingly hard to find experts in their fields who are prepared to do so. Publicly identifying this type of “duplicate publication” serves no useful purpose.

M Polkey
Consultant Physician, Royal Brompton Hospital, London, UK

J Moxham
Professor of Respiratory Medicine, Guy’s Kings and St Thomas’ School of Medicine, London, UK

Correspondence to: Dr M Polkey, Respiratory Muscle Laboratory, Royal Brompton Hospital, London SW3 6HP, UK; m.polkey@rbh.nhames.nhs.uk

Chlamydia pneumoniae and COPD exacerbation

We read with interest the recent paper by Blasi et al which showed that Chlamydia pneumoniae infection is associated with higher rates of exacerbation and airway microbial colonisation in patients with COPD.4 We have prospectively studied patients in the East London COPD study with daily monitoring using diary cards to detect COPD exacerbation defined using the same criteria.4,5 Serum microimmunofluorescence (MIF) immunoglobulin G (IgG) titres for C pneumoniae were measured in 110 patients (FEV1 % RT 18.4) with stable COPD during 1 year with simultaneous estimation of plasma fibrinogen and serum interleukin 6 (IL-6); 26% of the patients had IgG titres of 1:1 in 16 (fig 1). High C pneumoniae IgG titres were not related to FEV1 % predicted, exacerbation frequency, plasma fibrinogen, or serum IL-6 levels. In their paper Blasi et al did not report whether there was a relation between MIF titres and exacerbation frequency.

Blasi and colleagues found that 43% of patients when stable were positive for C pneumoniae by DNA polymerase chain reaction (PCR) using peripheral blood mononuclear cells (PBMCs). At exacerbation they have only shown data for the 34 (of 61) who consented to the antibiotic trial and all 34 were positive for C pneumoniae. In our study a further 33 patients (FEV1 % RT 16.3) were simultaneously sampled using nasopharyngeal swabs. We found no C pneumoniae using a nested reverse transcription PCR adapted from Cunningham et al at stable baseline but nine patients (seven from induced sputum and another two in nasal aspirates)
were 

(28%) were positive for 

C pneumoniae at 

exacerbation. The presence of 

C pneumoniae was not 

associated with smoking history, FEV1%, peak 

flow change at or peak flow recovery from 

COPD exacerbation, rate of peak flow recov-

ery, IL-6 (fig 2) or IL-8 levels, or total and dif-

ferential cell counts in induced sputum. The lack of relationship between 

C pneumoniae detection and inflammatory markers at exacerbation 

suggests to us that 

C pneumoniae exacerbations are no different from exacerbations 

not associated with 

C pneumoniae.

We found no relationship between 

C pneumoniae detection in the airway at exacerbation 

and exacerbation frequency (p=0.504), but 

Blasi et al found that 

C pneumoniae positive patients (stable COPD) had a greater 
tendency towards frequent exacerbation. However, the difference in exacerbation frequency 

between the two groups was small (6 exacerbations per year), and the authors 

need to be cautious about concluding that this 
difference could affect disease progression.

The main difference between the data of Blasi et al and ours is that in their study 16 of 

42 patients (38%) enrolled in study 1 had sputum positive for 

C pneumoniae by DNA PCR and a similar number (61/141, 43%) in study 2 in 

PBMCs, both during stable COPD. We sampled only once in stable COPD and found none, despite finding 28% at exacerbation. Blasi and colleagues sampled subjects repeat-

edly (at least four times), but it is not clear how many times they had to be positive to be 
defined as "respiratory samples positive for 

C pneumoniae by DNA PCR". The 16 positive patients provided 69 sputum samples; were all sputum samples positive on all occasions examined in these patients? Similarly, were all 125 sputum samples from the 26 patients who were 

C pneumoniae negative always negative?

It would be helpful if the authors could give the data on the chronic nature of infection in their sputum samples.

References

1 Blasi F, Damato S, Casentini R, et al. 


Authors’ reply

We are grateful to Seemungal et al for their comments regarding our recently published paper on Chlamydia pneumoniae and chronic bronchitis.

Seemungal et al prospectively studied 110 patients with COPD for 1 year, evaluating serum microimmunofluorescence IgG titres, plasma fi-

brinogen, and IL-6 levels. They found no correla-
tion between high IgG titres and FEV1%, predicted 
exacerbation frequency, plasma fibrinogen, and 

IL-6 levels. We also found no correlation be-
tween serological results and FEV1%, predicted 
average exacerbation frequency. In fact, as in previous reports, we found a low degree of correlation 
between 

C pneumoniae serology and peripheral blood mononuclear cell (PBMC) PCR. A greater degree of correlation was observed when IgG and 

IgA titres were combined but, unfortunately, no comparison is possible as Seemungal et al only 

performed IgG titre determinations. In any case, our findings are not truly comparable with 
those of Seemungal et al as serology is known to be less specific than PCR for the identification of chronic infection with 

C pneumoniae.

In the second part of their letter Seemungal 
et al report the results of an analysis on a fur-
ther group of 33 patients who were simultane-
ously sampled for nasal aspirates and induced sputum when stable and during exacerbation. They found no PCR positivity in stable patients, whereas in nine of 43 exacerbations 

C pneumoniae was detected by PCR in respira-
tory specimens. The authors infer that DNA 

positivity in the sputum is a marker of 

C pneumoniae acute infection; this would mean that around 30% of all acute exacerbations are 
sustained by 

C pneumoniae. However, the gold standard for acute infection is still considered

serology on paired samples. Applying both PCR and serology on paired sputum samples we 

found an acute infection in two of 34 exacerbations confirming our previous data of an 
overall incidence of 5–6%. Their definition of acute 

C pneumoniae infection may explain, at least in part, why the authors could not detect any 
difference between exacerbations associated or not associated with 

C pneumoniae in terms of 
inflammatory response.

The reported discrepancy in PCR positivity on 

respiratory samples between our study and 

that of Seemungal et al may be related to dif-

ferent PCR techniques. In fact, we found 16/42 (38%) PCR positive patients with stable 

COPD, whereas they found 0/33 and 9/33 (28%) in stable COPD and during an 
exacerbation, respectively. Considering that the rate of positivity in our stable patients is compar-

table to that of patients with exacerbation in 

Seemungal et al’s series, we think that “our study indicates 

that the different PCR results may simply be related to PCR 
sensitivity, sputum quantity/quality, amount of DNA retrieved from the samples, and 
test positivity in the sputum is a marker of 

C pneumoniae exacerbations in 33 patients with 43 
exacerbations. Outliers are shown; p=0.187 
(Wann-Whitney U test).

Figure 1 Distribution of serum 

C pneumoniae microimmunofluorescence (MIF) IgG antibody inverse titres in 110 patients with stable COPD.

Figure 2 Induced sputum levels of IL-6 during Chlamydia (+) and non-Chlamydia (–) 

COPD exacerbations in 33 patients with 43 exacerbations. Outliers are shown; p=0.187 
(Wann-Whitney U test).
Marginal benefits of adding formoterol

Price and colleagues' conclusion that adding formoterol confers a therapeutic advantage to inhaled steroid in patients with mild to moderate asthma. During the 6 month follow up in part II of the study the frequency of secondary end points of mild asthma exacerbations differed by 2.5 per patient per 6 months while the difference in poorly controlled asthma days was 4.2 days per patient per 6 months. These differences, while statistically significant, are unlikely to be of real clinical relevance. Indeed, during the same period the difference in quality of life was neither significant nor clinically relevant. The main difference which was significant were in bronchodilator sensitive outcomes such as peak flow and reliever use, which are to be expected when patients are taking a 24/7 bronchodilator. These data are little different from those in steroid naïve patients in the OPTIMA trial over 12 months where the addition of formoterol to low dose budesonide improved lung function but not exacerbations, while in the same trial the addition of formoterol conferred only a small but significant reduction in exacerbations in patients previously treated with corticosteroids.

Pointedly, neither of these studies evaluated any inflammatory surrogates. We would therefore suggest that these trials indicate that most patients with mild to moderate asthma can be adequately controlled on low to medium doses of inhaled budesonide alone, and that there is only a marginal advantage conferred by adding formoterol. Moreover, combination inhalers are considerably more expensive than inhaled steroid alone and their routine use is not warranted in primary care.

Scadding-Morriston Davies Joint Fellowship in Respiratory Medicine 2003

This fellowship is available to support visits to medical centres in the UK or abroad for the purpose of undertaking studies related to respiratory medicine. Applications are invited from medical graduates practising in the UK, including consultants and irrespective of the number of years in that grade. There is no application form but a curriculum vitae should be submitted together with a detailed account of the duration and nature of the work and the centres to be visited, confirming that these have agreed to provide the facilities required. Please state the sum of money needed for travel and subsistence. A sum of up to £20000 can be awarded to the successful candidate, or the sum may be divided to support two or more applications. Applications should be sent to Dr I A Campbell, Secretary to the Scadding-Morriston Davies Fellowship, Landlouse Hospital, Penarth, Vale of Glamorgan CF64 2XX, UK by 31 January 2003.

Department of Respiratory Medicine, Belfast City Hospital, Belfast BT9 7AB, UK, tim@dsl.pipex.com

T J Warke

www.thoraxjn.com
T J Warke

Thorax 2002 57: 1089
doi: 10.1136/thorax.57.12.1089-b

Updated information and services can be found at:
http://thorax.bmj.com/content/57/12/1089.3

These include:

Email alerting service
Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

Notes

To request permissions go to:
http://group.bmj.com/group/rights-licensing/permissions

To order reprints go to:
http://journals.bmj.com/cgi/reprintform

To subscribe to BMJ go to:
http://group.bmj.com/subscribe/