If you have a burning desire to respond to a paper published in Thorax, why not make use of our “rapid response” option? Log on to our website (www.thoraxjnl.com), find the paper that interests you, and send your response via email by clicking on the “eletters” option in the box at the top right hand corner. If it is timely and obscene, it will be posted within seven days. You can retrieve it by clicking on “read eletters” on our homepage.

The editors will decide as before whether to also publish it in a future paper issue.

Duplicate publication

We are writing to express our unease at what we believe is inappropriate censure imposed on our colleague Professor Corris concerning duplicate publications.1 2 Professor Corris was asked to write what was essentially a CME article for Clinical Medicine on a subject that he had previously reviewed in detail for Thorax. It was inevitable that there would be considerable duplication. The same papers and information were being discussed and there are limitations in the way complex arguments can be succinctly presented. It is universally accepted that a degree of duplication in review articles is completely different from trying to pass off as a new study previously published peer reviewed papers containing original data. It is commonplace for people with authoritative opinions to write similar articles in more than one journal as shown by the similarities between the Harveian oration by Warrell published in the same issue of Clinical Medicine and an earlier manuscript in the Lancet.3 4 We believe such duplication is entirely appropriate, as surely it is our duty as educators to disseminate information to as wide an audience as possible. Fraud in any shape or form in science is to be wholly deplored, but let us not be so zealous in its pursuit that we smear the essence is to be wholly deplored, but let us not be so zealous in its pursuit that we smear the

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.1 5 With the exception of the article in Thorax (for which the invitation to write came following a prompt from us), the remaining articles were all 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.

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References
1 Anon Notice of duplicate publication. Thorax 2002; 57: 1089–09.

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.5 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.6 7 Serum microimmunofluorescence (MIF) immunoglobulin G (IgG) titres for C pneumoniae were measured in 110 patients (FEV1% 39.8 (16.3)) were simultaneously sampled using nasopharyngeal aspiration and interleukin 6 (IL-6); 26% of the patients had IgG titres of ≥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 had 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% 39.8 (16.3) were simultaneously sampled using nasopharyngeal aspiration and interleukin 6 (IL-6) levels. In their paper Blasi et al did not report whether there was a relation between MIF titres and exacerbation frequency.

We found no C pneumoniae using a nested reverse transcriptase PCR adapted from Cunningham et al at stable baseline but nine patients (seven from induced sputum and another two in nasal aspirates)
IgG antibody inverse titres in 110 patients with stable COPD.

Induced sputum levels of IL-6 associated with smoking history, FEV1%, peak (28%) were positive for Chlamydia pneumoniae.

Distribution of serum C pneumoniae microimmunofluorescence (MIF) IgG titres in 110 patients with stable COPD.

We found no 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 are grateful to Seemungal et al for their comments regarding our recently published paper on Chlamydia pneumoniae and chronic bronchitis.

Our results on exacerbation frequency are based on the observation of 141 subjects for 2 years compared with 33 subjects in the study by Seemungal et al. The different number of subjects included in the two studies may explain some discrepancies. We do agree that caution is needed in interpreting the results of our study and stated that “our study indicates the possible role of C pneumoniae chronic infection in disease progression in COPD patients. Further confirmation based on large scale trials is needed”.

Figure 1 Distribution of serum C pneumoniae microimmunofluorescence (MIF) IgG titres.

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 (Mann-Whitney U test).

References

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 fibrinogen, and IL-6 levels. They found no correlation between high IgG titres and FEV1%, % predicted exacerbation frequency, plasma fibrinogen, and serum IL-6 levels. They also found no correlation between serological results and FEV1%, % predicted 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 further group of 33 patients who were simultaneously 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 respiratory 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 serum samples we found an acute infection in two of 34 exacerbations confirming our previous data of an overall incidence of 5–6%.

The reported discrepancy in PCR positivity on respiratory samples between our study and that of Seemungal et al may be related to different PCR techniques. In fact, we found 16/42 (38%) PCR positive patients with stable COPD, whereas they found 0/33 (28%) in stable COPD and during an exacerbation, respectively. Considering that the rate of positivity in our stable patients is comparable to that of patients with exacerbation in Seemungal’s series, we think that “our study indicate that PCR results may simply be related to PCR sensitivity, sputum quality/quantity, amount of DNA retrieved from the samples, and number of tested samples.” Seemungal et al tested a single induced sputum specimen for each stable patient whereas we analysed at least four spontaneous sputum samples for each stable patient. We defined any patient with at least two positive specimens as PCR positive whereas they defined a positive PCR as PCR positive on the basis of 43/69 (62%) PCR positive sputum samples. Twenty six patients were considered PCR negative; 24 had repeatedly negative PCR results on all 113 sputum samples and two patients had a single PCR positive sputum specimen (2/12 specimens).

Our results on exacerbation frequency are based on the observation of 141 subjects for 2 years compared with 33 subjects in the study by Seemungal et al. The different number of subjects included in the two studies may explain some discrepancies. We do agree that caution is needed in interpreting the results of our study and stated that “our study indicates the possible role of C pneumoniae chronic infection in disease progression in COPD patients. Further confirmation based on large scale trials is needed.” However, even a slight increase in exacerbation frequency may have a role in disease progression.

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References
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 the secondary outcome of mild asthma exacerbations differed by 2.5 per patient per 6 months while the difference in poorly controlled asthma days was 0.2 day 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 differences which were 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 naive 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.

In other 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.

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References


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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 £20,000 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, Llandough Hospital, Penarth, Vale of Glamorgan CF64 2XN, UK by 31 January 2003.