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. 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 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. 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 innocent to the detriment of us all.

At risk of another duplicate publication, we have also sent this letter to the editor of Clinical Medicine.

D P Pavord, M D L Morgan, A J Wardlaw
Institute for Lung Health, Department of Respiratory Medicine and Thoracic Surgery, Glenfield Hospital, Groveby Road, Leicester LE3 9QP, UK; trina.rathery@uhl.nhs.uk

References

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 microvascular colloisa in patients with COPD. 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. Serum microimmunofluorescence (MIF) immunoglobulin G (IgG) titres for C pneumoniae were measured in 110 patients (FEV1% 41.7 (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 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 (PMNCs). At exacerbation they have only studied patients in the East London COPD study with daily monitoring using diary cards to detect COPD exacerbation defined using the same criteria. Serum microimmunofluorescence (MIF) immunoglobulin G (IgG) titres for C pneumoniae were measured in 110 patients (FEV1% 41.7 (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 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.

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It is a matter of some concern to us that you felt obliged to print a notice of duplicate publication for Professor Corris. 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 his/her 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.1 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.

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.nhms.nhs.uk

Editors’ reply

We published the statement on duplicate publication, as did the other journal concerned, in response to a correspondent who pointed out the similarity between the two articles. When we looked through the article published in Clinical Medicine it was evident that large parts of the article in Thorax were reproduced verbatim.

We appreciate that review articles by the same author in different journals often contain overlapping information, but that was not the point on this occasion. The point was that much of the material was exactly the same, and Professor Corris had not explicitly acknowledged this or the contribution of other authors to it.

We have taken a firm line on duplicate publication and non-disclosure of related publications in the past and, although we accept that some degree of duplicate reporting is acceptable and common in review or opinion articles, having received a formal complaint about the article we did not feel able to dismiss it. This was particularly the case since Professor Corris was until very recently an Associate Editor of Thorax, and we were in danger of being open to accusations of special privilege for people who have been associated with the journal.

J Britton
Executive Editor

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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.

T A R Seemungal, J A Wedzicha
Academic Unit of Respiratory Medicine, St Bartholomew's and Royal London School of Medicine and Dentistry, London

P K MacCallum
Medical Research Council Epidemiology and Medical Care Unit, Wolfson Institute of Preventive Medicine, St Bartholomew’s and Royal London School of Medicine and Dentistry, London

S L Johnston
Imperial College at St Mary’s Hospital, London

P A Lambert
Aston University, Birmingham

Correspondence to: Professor J A Wedzicha, Academic Unit of Respiratory Medicine, Dominon House, St Bartholomew’s Hospital, London EC1A 7BE, UK; J.A.Wedzicha@mdrl.ac.uk

References

Authors’ reply
We are grateful to Seemungal et al for their comments regarding our recently published paper on Chlamydia pneumoniae and chronic bronchitis.1 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 or exacerbation frequency, plasma fibrinogen, and serum IL-6 levels. We also found no correlation between serological results and FEV1 % predicted or exacerbation frequency. In fact, as in previous reports,2 we found a low degree of correlation between C. pneumoniae serology and peripheral blood mononuclear cells (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.3

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%.4 Their definition of acute C. pneumoniae infection may explain, at least in part, why they 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 different PCR techniques. In fact, we found 16/42 (38%) PCR positive patients with stable COPD, whereas they found 9/33 (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 et al’s series, we think that our indications for a positive PCR results may simply be related to PCR sensitivity, sputum quality/quantity, amount of DNA retrieved from the samples, and number of tested specimens.5 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 COPD, whereas they found 9/33 (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 et al’s series, we think that our indications for a positive PCR results may simply be related to PCR sensitivity, sputum quality/quantity, amount of DNA retrieved from the samples, and number of tested specimens.6

Our results on exacerbation frequency are based on the observation of 141 subjects for 2 years compared with 33 subjects in the study of 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 that exacerbation frequency between the two groups was small and a similar number (61/141, 43%) in study 2 suggests to us that C. pneumoniae exacerbations not associated with C. pneumoniae.”

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 exacerbations differed by 2.5 per patient per 6 months while the difference in poorly controlled asthma days was 2.2 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 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.1

Pointedly, neither of these studies evaluated any inflammatory surrogates. We 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.

Reference


L Howard

Department of Respiratory Medicine, Norfolk & Norwich University Hospital, Norwich NR4 7UY, UK, luke.howard@tinyworld.co.uk

References


BOOK REVIEW

Respiratory Medicine Specialist Handbook


This is the first in a new series of specialist handbooks that aims to fill the niche between the comprehensive textbook and the pocket handbook. There are the obvious pitfalls of trying to squeeze in too much detail at the expense of accessibility or reducing the subject to little more than a series of disjointed notes. However, this book—for the most part—steers clear of both of these errors and has produced a very readable, yet reasonably detailed, summary of specialist respiratory medicine. The 31 chapters cover a wide variety of topics and the authors’ list is like a “Who’s Who?” of UK respiratory medicine.

It is possible to pick up this book, read a chapter in less than half an hour, and come away with an increased knowledge of the pathophysiology of the condition under study and, perhaps more usefully, the intricacies of practical management which is the focus of the book. It will therefore cater to the specialist registrar undergoing higher specialist training in providing a broad understanding in reasonable detail of most facets of respiratory medicine, but it could also be of use to the experienced physician in reaffirming, refreshing, and refining the basics, and perhaps updating knowledge with regard to more recent developments.

The book is attractively presented with short paragraphs of text interspersed with helpful tables and figures. For those who are stimulated to seek more information on any subject, each chapter has a selection of references for further reading. Whilst we all might aspire to read, study and inwardly digest a weighty, comprehensive tome of respiratory medicine, for most of us in busy clinical practice this proves difficult. This reviewer would therefore encourage reading and studying this excellent book as an alternative which is more attainable and possibly of more practical relevance.

T J Warke

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

ICU outcomes in acute respiratory failure secondary to COPD

The data presented by Breen et al regarding the outcomes of patients with chronic obstructive pulmonary disease (COPD) are encouraging and lend support to the respiratory physician often faced with nihilistic attitudes towards ventilating these patients in acute respiratory failure. However, despite the proposition by the authors that certain patients with likely poor outcomes might have been excluded, the ICU stays for both groups (intubated and non-intubated) are strikingly short.

This is that the threshold for intubation as opposed to non-invasive ventilation (NIV) may have been lower before 1994 than in current practice. Although the authors explain the reason for the high levels of PaO2 on admission to the ICU, it could be that the severity of respiratory acidosis may have reflected excess oxygen therapy rather than the severity of the underlying mechanical respiratory failure, thus being more readily reversible and requiring a shorter period of ventilatory support. Although the decision to intubate is not solely based upon blood gases, with the increasing availability of NIV it might be the subgroup of these patients would now be managed using controlled oxygen therapy, respiratory stimulants, and NIV. As a result, I suspect that the physiological state of the patient that we offer to the ICU in our current practice may be worse than in this study with commensurate outcomes (longer stays and higher mortality). Despite this, many patients still do well and studies of this type need to continue to assess predictors of unfavourable end points.

J Howard

Department of Respiratory Medicine, Norwich & Norwich University Hospital, Norwich NR4 7UY, UK, luke.howard@tinyworld.co.uk