Response

To the Editor:

We thank Drs van den Bemt and Schermer for their thoughtful comments on our recent article in CHEST1 on the confounding influence of increased body mass on the diagnosis of COPD. Given the dramatic global increases in the prevalence of both obesity and COPD over the past decades, we can no longer neglect the impact of this combination on common pulmonary function measurements and their clinical interpretation. The interaction of COPD and obesity is complex and poorly understood, given the vast pathophysiologic heterogeneity of both conditions.

COPD essentially remains a clinical diagnosis based on the trial of smoking (or other noxious gas) exposure, the presence of persistent respiratory symptoms, and the objective demonstration of airflow obstruction that is not fully reversible. The definition of airflow obstruction based on postbronchodilator fixed FEV\textsubscript{1}/FVC ratio <0.7 has been criticized because of the risk of overdiagnosis in the elderly and underdiagnosis in the young.\textsuperscript{2,3} Less attention has been given to the risk of underdiagnosis of COPD in obese smokers by fixed ratio criteria, given the documented exponential decline in thoracic gas volumes (the denominator) with increasing BMI.\textsuperscript{1,4}

Diagnosis of COPD is further confounded in overweight individuals by uncertainty concerning the specific origin of their respiratory symptoms. Thus, activity-related dyspnea in the obese smoker could be explained by factors other than airflow obstruction: higher ventilatory demands as a result of higher metabolic weight to the proposed mechanism of treatment of atypical distress toxin in subjects with refractory asthma, provides a stronger rationale for use of macrolides in this setting, although it was not possible to eradicate this in all subjects. Could this toxin’s presence and its sometimes failed eradication potentially explain the observed, yet often unexplained and unpredictable, benefits of prolonged macrolide therapy in cases of refractory asthma when there is no apparent reflux or bronchiolitis? This would add further weight to the proposed mechanism of treatment of atypical infection\textsuperscript{2} in addition to the armamentarium of well-known prokinetic\textsuperscript{1} and antiinflammatory action\textsuperscript{1} of these drugs.

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References


Macrolides in Asthma

To the Editor:

The study by Peters et al\textsuperscript{1} (August 2011) demonstrating Mycoplasma pneumoniae community-acquired respiratory distress toxin in subjects with refractory asthma, provides a stronger rationale for use of macrolides in this setting, although it was not possible to eradicate this in all subjects. Could this toxin’s presence and its sometimes failed eradication potentially explain the observed, yet often unexplained and unpredictable, benefits of prolonged macrolide therapy in cases of refractory asthma when there is no apparent reflux or bronchiolitis? This would add further weight to the proposed mechanism of treatment of atypical infection\textsuperscript{2} in addition to the armamentarium of well-known prokinetic\textsuperscript{1} and antiinflammatory action\textsuperscript{1} of these drugs.

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Response

To the Editor:

We thank Dr Medford for his interest in our recent article in CHEST (August 2011).1 Prior studies by our group demonstrated that direct instillation of either Mycoplasma pneumoniae or recombinant M pneumoniae community-acquired respiratory distress syndrome toxin (rCARDSTx) causes a lymphocytic perivascular response and eventually induces a robust peribronchial inflammation in both rodents and baboons.2 More recent studies in our primate model found that rCARDSTx can initiate a T-helper 2 cell response and “asthma-like” lesions with mixed eosinophilic/lymphocytic infiltration of airways, mucous metaplasia, and focal mucous plugs (unpublished data). Other groups have shown that allergic airway inflammation impairs the innate host defenses of the lung and results in reduced clearance of M pneumoniae in animal models of asthma.3 There are increasing data that both M pneumoniae and CARDS Tx play some role in promoting airway inflammation that could contribute to the onset and clinical course of asthma.

The fact that macrolide antibiotics may be of therapeutic benefit in some patients with asthma is not surprising because macrolides belong to a family of compounds that possess both immunomodulatory and antimicrobial activity. The proven efficacy of macrolide antibiotics in other chronic respiratory conditions, such as diffuse pan-bronchiolitis, bronchiectasis, and cystic fibrosis,4 has led some physicians to use macrolides in patients with difficult to control asthma. However, whether macrolides “treat” occult atypical bacterial infections or reduce inflammatory processes is unclear. Thus, routine use of macrolide antibiotics in the management of chronic stable asthma cannot be recommended because of the lack of available evidence for their efficacy. Despite these facts, however, there is increasing evidence that some asthmatic patients may be chronically infected or colonized with atypical bacteria and may benefit from macrolide therapy. However, it remains unclear how to best identify this group of patients, as well as the appropriate dose, frequency, and duration of therapy required to eradicate these organisms. Clearly, more research is needed to better elucidate the role of atypical bacteria in the pathogenesis of asthma and to better define the antiinflammatory mechanisms of macrolide antibiotics. Only then will we be able to assess the therapeutic value of macrolides in chronic asthma.

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REFERENCES


Racial Differences Influence Health-Related Quality-of-Life Measurements

To the Editor:

I read with interest the article by Han et al1 in a recent issue of CHEST (November 2011) that shows African Americans with a prior history of COPD exacerbations reported worse health-related quality of life (HRQL) than their Caucasian counterparts despite having comparable lung functions. The data analyzed are huge and multi-centered in origin from the United States. In their statistical regression model, this racial factor remains independently associated with worse HRQL, along with several other variables such as dyspnea, age, smoking duration, and education level.

In a much smaller sample of local patients with persistent moderate-to-severe asthmatics, my colleagues and I have previously published that patients of Indian ethnicity, compared with those of Malays or Chinese ethnicity, reported worse HRQL based on the St. George Respiratory Questionnaire. These patients remained independently associated with lower HRQL after adjustment for age, sex, asthma duration, and inhaled corticosteroid dose.2 In our model of multiple regression using variables identified from factor analysis, education level stood together with Indian ethnicity as being independent associates. Although they studied different airway diseases, both these studies look at the influence of racial difference on HRQL of patients with chronic persistent airway diseases.

Han et al1 rightly discussed this from the perspective of disease exacerbations, and considered factors among African Americans like “experience” of breathlessness, education level, and socio-economic status with implications on health insurance as plausible explanations for worse HRQL. The obvious implication of this is how best to prevent and manage the exacerbations of African Americans. Here, disease exacerbations are perceived as the link to understanding why a particular race of people reports poorer HRQL.

Another important perspective when interpreting racial differences in HRQL findings is to consider HRQL as a measuring tool that lacks the sensitivity to discern influence of racial and cultural differences. Many such tools are developed primarily in...