Response

To the Editor:

We thank Dr Medford for his interest in our recent article in CHEST (August 2011). Prior studies by our group demonstrated that direct instillation of either Mycoplasma pneumoniae or recombinant M pneumoniae community-acquired respiratory distress syndrome toxin (rCARDS Tx) causes a lymphocytic perivascular response and eventually induces a robust peribronchial inflammation in both rodents and baboons. More recent studies in our primate model found that rCARDS Tx 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lesions with mixed eosinophilic/lymphocytic infiltration of airways, mucous metaplasia, and focal mucous plugs (unpublished data).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. 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, 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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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 large 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. 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
homogenous, well-educated patient populations that may not be universally interpretable when applied across different cultures and ethnicity. Findings like ours and those of Han et al lend support to such a notion, especially since different ethnic groups can report differing HRQL despite being comparable in lung function and disease severity. The implication from this is whether we can reliably interpret measurements of HRQL across different cultures and ethnicity, for example, in worldwide multi-center studies, and draw meaningful conclusions from them. Perhaps we should be mindful of these implications when interpreting HRQL measurements and appreciate that the racial influence exists and can be potent.14

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Response

To the Editor:

We thank Dr Loh for his interest in our recent article in CHEST.1 It is very interesting to see similar associations among race, education, and quality of life in asthma in a different patient population. As Dr Loh points out, cultural and socioeconomic factors do appear to influence the “experience” of this disease. He also raises a concern regarding the reliability and interpretability of quality-of-life assessments, such as the St. George Respiratory Questionnaire (SGRQ), across different cultures. The SGRQ has been used around the world in 100 languages. A recent study in nearly 5,000 patients from 28 countries2 The relationship between change in FEV₁ and SGRQ did not differ by world region, although patients in the Asia-Pacific region showed improvements in quality of life even in the placebo group. This may be due to a “trial effect,” in which patients in the study received better health care by joining a clinical trial. Hence, although there are currently no data clearly supporting the possibility that the SGRQ itself behaves differently in different races and cultures, differences in health-care systems certainly may influence scores.

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COPD and Ischemic Heart Disease

To the Editor:

We read with interest the article by Enriquez and colleagues3 in a recent issue of CHEST (September 2011) about the increased number of adverse events after percutaneous coronary intervention (PCI) in patients with COPD. Data from their study are consistent with those reported previously, indicating a higher incidence of adverse effects and mortality in patients with a previous diagnosis of COPD undergoing coronary catheterization for ischemic heart disease.4 However, diagnosis of COPD in both was based upon clinical criteria plus questionnaire criteria (COPD medication or a pre-bronchodilator (BD) FEV₁ < 75% predicted value) without full respiratory functional studies, while in the Konecny et al5 study, spirometry was available only in 60% of patients. In our opinion, this explains the

SGRQ data from TORCH (Towards a Revolution in COPD Health) in 2011;140(5):1169-1176. However, diagnosis of COPD in both was based upon clinical criteria plus questionnaire criteria (COPD medication or a pre-bronchodilator (BD) FEV₁ < 75% predicted value) without full respiratory functional studies, while in the Konecny et al5 study, spirometry was available only in 60% of patients. In our opinion, this explains the

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