Correspondence

Chlamydia pneumoniae antibodies and adult-onset asthma

To the Editor:

I would like to clarify a misunderstanding regarding previously published information cited by Routes et al,[1] who measured IgM and IgG antibodies to *Chlamydia pneumoniae* in 46 patients with adult-onset asthma and in age- and sex-matched control subjects by using serum obtained from inpatients, outpatients, and hospital workers. They reported that IgG seropositivity was equally high in both patients and control subjects and stated that it was not apparent why their findings contrasted with previously published results correlating seropositivity to *C pneumoniae* and asthma.[2]

In their introduction Routes et al[1] incorrectly stated that *C pneumoniae* IgG antibody was measured in the original study that reported a positive association between *C pneumoniae* serology and adult-onset asthma.[2] In fact a polyvalent antibody (mixture of IgM, IgG, and IgA) was measured in the original study, and subsequent research has revealed that the IgA, but not IgG, isotype is associated with asthma.[3,4] Thus lack of association of IgG antibody and asthma has been reported by others,[3] as well as by Routes et al.[1] Another possible contribution to the lack of association in their study was the use of hospital-based control subjects who could have higher levels of IgG antibody than control subjects from the general population.[3,4]

In any event, I agree with the authors' general conclusions that serologic testing alone will not be clinically useful in selecting asthmatic patients for antimicrobial therapy. Although *C pneumoniae* IgA antibody testing can be specific (if *C trachomatis* and *C psittaci* antigens are used as parallel controls), it is unlikely that IgA is sufficiently sensitive; furthermore, serologic testing cannot identify the location of infection. I agree with the authors that evidence based on organism identification should be sought in future studies. I believe that epidemiologic criteria will also need to be studied because identification of *C pneumoniae* in upper respiratory secretions is also insensitive, and it is not practical to diagnose all cases of infection by bronchoalveolar lavage and lung biopsy.

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Reply

To the Editor:

Dr Hahn is correct that the original study correlating *Chlamydia pneumoniae* serology and asthma measured polyvalent antibodies but not IgG.[1] However, we disagree with his assertion that the lack of association between IgG antibodies to *C pneumoniae* and asthma is widely accepted. Recent studies continue to indicate that such a correlation exists.[2] Moreover, his review on the subject cites several recent reports supporting this correlation, particularly in patients with extremely high IgG titers.[3] Dr Hahn cites his own recent study (published at the same time as our article) indicating that there is only a correlation between IgA antibodies (not IgG) to *C pneumoniae* and asthma.[4] In fact, this study found that IgG seroreactivity was higher in asthma associated with infection in comparison with atopic, occupational, or exercise-induced asthma. Furthermore, the studies that support the assertion that IgA serology is highly indicative of chronic *C pneumoniae* infection are extremely limited. Dr Hahn has used serology (polyvalent antibodies, IgG as well as IgA) as evidence for chronic *C pneumoniae* infection to justify the use of antibiotics in open-label trials.[5,6] Our data indicate that random titers of IgG or IgM to *C pneumoniae* should not be used in selecting asthmatic patients for antimicrobial therapy. Furthermore, we question the use of IgA seroreactivity because studies have not persuasively shown that IgA seroreactivity closely correlates with either culture or PCR evidence of *C pneumoniae* infection.

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