Asthma nervosum

To the Editors:

The name ''asthma nervosa'' [1] is linguistically incorrect. *Asthma* is a Greek neutral noun in a language that tries hard to keep its genders apart. *Nervosa* is a Latin adjective and this language also does not mix the genders: *nervosa* is feminine (as are *anorexia* and *bulimia*, two conditions for which *nervosa* is appropriate). Therefore, we should refer to *asthma* as *nervosum* (both neutral). This may sound slightly odd in English, and therefore, I would have no objection to ''nervous asthma''. However, if we want to pay tribute to the Classics, we should try to use them as correctly as possible.

A. Papagiannis
Pulmonary Dept, St Luke's Hospital, Thessaloniki, Greece.

Correspondence: A. Papagiannis, Gregoriou E' 42, GR 542 49 Thessaloniki, Greece. E-mail: antpap56@otenet.gr

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Respiratory *Chlamydophyla pneumoniae* resides primarily in the lower airway

To the Editors:

We read with interest the article by WANG *et al.* [1], which explored inflammatory phenotypes in adults and children with acute asthma. Their findings noted a striking paucity of *Chlamydophyla pneumoniae* organisms, even in patients with acute, neutrophilic asthma, and they concluded that: ''The aetiology of neutrophilic asthma is unknown and is not explained by the presence of current active *C. pneumoniae* infection.'' This is in direct contrast to recently published studies that used a similar cohort of patients [2]. A closer look at the results of the study by WANG *et al.* [1] revealed that while the authors used authentic quantitative PCR techniques to identify *C. pneumoniae* DNA, they utilised sputum samples for detection. It has been well established that *C. pneumoniae* resides primarily in the lower airway and is recovered much more reliably from bronchoalveolar lavage (BAL) fluid, as clearly demonstrated by MARTIN *et al.* [3].

Responding to a question about the future of PCR as a diagnostic tool for respiratory *Chlamydia* and *Mycoplasma* respiratory tract infection, M. Kraft, a co-author of the manuscript by MARTIN *et al.* [3] and an internationally recognised respiratory infectious disease expert, reiterated: ''As demonstrated by the present study, PCR remains the only reliable way of demonstrating chronic infection. But PCR requires collecting relevant samples. The lower airway samples we need for diagnosis require invasive bronchoscopy'' [4]. Indeed, viable *C. pneumoniae* have been successfully cultured from the BAL fluid of paediatric patients with chronic, severe asthma and the culture data correlated with the PCR (all culture-positive samples were also PCR-positive) [2]. Therefore, the absence of *C. pneumoniae* in the sputum samples used by WANG *et al.* [1] is not surprising. There is no question that under certain conditions of severe respiratory disease caused by *C. pneumoniae* the organism will be found in the sputum. However, in milder cases of acute infection, and in presumptive chronic infection, the chlamydial inclusions are most likely to be found in the macrophage or bronchial epithelial cells of the lower respiratory tract, making detection from induced sputum difficult and imprecise [3].

It may be that chronic, rather than acute, *C. pneumoniae* infection of only lung dendritic cells is a causative factor in asthma [5]. SCHRÖDER *et al*. [5] reported that adoptive transfer of dendritic cells from *C. pneumoniae*-infected mice was sufficient to induce an ''asthmatic'' phenotype in recipient animals. If this is found to be a relevant mechanism in human asthma, then direct microbiological diagnosis of *C. pneumoniae* by PCR, or by any method, will be even more difficult than has been anticipated, even by us. This, however, further strengthens the argument that asthma is a heterogeneous disease and this heterogeneity in inflammatory phenotype, as well as response to treatment, might be a direct result of the aetiology. While much work remains to be done in terms of deciphering the mechanisms underlying its involvement in the asthmatic process, the published data and ongoing work leave little doubt that *C. pneumoniae* plays an important role in asthma pathogenesis.

W.C. Webley* and D.L. Hahn#
*Dept of Microbiology, University of Massachusetts, Amherst, MA, and #Dept of Family Medicine, University of Wisconsin School of Medicine and Dean Medical Center, Madison, WI, USA.