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DOI: 10.1183/09031936.00116807

From the authors:

The authors would like to thank P. Charles for his interest in their recent article [1]. His major point of criticism is that the finding of an association between pre-hospital treatment and outcome may lead to unnecessary antimicrobial usage in the ambulatory setting. P. Charles emphasises the role of host factors on outcome of pneumonia and cites a recent study evaluating community-acquired pneumonia (CAP) treatment failure [2]. Host factors, namely neoplasia and neurological disease, were associated with CAP outcome [2]. However, patients with previous antimicrobial treatment were excluded in the study by GENNE *et al.* [2], thus it is impossible to know the impact of pre-hospital antibiotic therapy on outcome from the study. In our study, a less severe course of pneumococcal disease in patients with prior ambulatory treatment was found [1]. As a randomised trial was not performed, data are clearly observational and open to confounding. Accordingly we did not claim that a causal relationship is proven by these findings. However, we think it worthwhile to discuss these seemingly provocative data as, in the pre-hospital phase of CAP, the duration and impact of treatment delay may be even larger than in the hospital setting, where most guidelines now recommend institution of treatment within 4–8 h after admission or ''as soon as possible'' [3].

Since the mortality of pneumococcal disease has not changed much over the past decades, new options for increasing survival are necessary. Several data, including ours and on the timing of in-hospital treatment [4], suggest a benefit from institution of antimicrobial therapy early in the course of disease. This has to be weighed against the potential of antibiotic misuse as outlined by P. Charles. Obviously more data, especially from randomised trials are needed to draw firm conclusions.

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STATEMENT OF INTEREST

None declared.

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β_2-Adrenoceptor polymorphisms and asthma phenotypes: interactions with passive smoking

To the Editors:

The article of ZHANG *et al.* [1] was of particular interest to us for two reasons. First, this article reported reduced lung function at age 11 yrs in children with arginine 16, compared with those homozygous for glycine 16, among those exposed to tobacco smoke but not in unexposed children. The present authors' report [2] of a similar association of reduced lung function with the presence of the arginine 16 allele was not mentioned in their discussion. Unlike ZHANG *et al.* [1], we also found lung function to be reduced in children with any glutamine 27 alleles. However, in our study of a smaller birth cohort, the association of