


From the authors:

We would like to thank S. Teramoto and co-workers for the important issues they raised. While appraising their comments, it is important to make a distinction between the use of severity rules inside and outside hospital settings. Looking at the available literature, we think that the pneumonia severity index (PSI) and CURB-65 (Confusion, Urea >7 mmol L⁻¹, Respiratory rate ≥30 breaths min⁻¹, Blood pressure (systolic value <90 mmHg or diastolic value ≤60 mmHg)) are both valid and useful in hospital settings. However, it is an interesting suggestion to improve CURB-65 by introducing more detailed age groups in the score. In primary care, PSI and CURB-65 are less useful for various reasons. Regarding the predictive value of age, the results of our study [1] showed that age >80 yrs was a better predictor of outcome than age categories between 65–80 yrs. Probably as there are a lot of healthy individuals aged 65–80 yrs in primary care who have a low risk for poor outcome.

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STATEMENT OF INTEREST
None declared.

REFERENCES


Oral antibiotics prior to hospitalisation for community-acquired pneumonia

To the Editor:

SCHAFF et al. [1] postulate that antibiotics prior to hospitalisation with community-acquired pneumonia may be protective because of a slightly lower death rate and lower C-reactive protein concentration, leucocyte count and acute physiology score in the 13 out of 105 patients that received them. Since AUSTRIAN and GOLD [2] demonstrated a reduction in mortality from 80 to 17% in bacteraemic pneumococcal infections treated with penicillin, the death rate for this condition has changed little. A 2006 study has suggested that deaths in patients with community-acquired pneumonia are far more likely to be due to host factors rather than antibiotic choices [3].

It is possible that such host factors could lead to some patients having better outcomes, subacute presentations and more time before hospitalisation in which to receive oral antibiotics. Conversely, those patients with worse outcomes may show more acute presentations, removing the option of pre-hospitalisation antibiotics. Information on the number of days that patients were unwell prior to admission may help to answer this in part. Given the inaccuracy with which doctors make the diagnosis of community-acquired pneumonia, this is an important point [4–6], since pharmaceutical companies might be predicted to use potentially misleading conclusions such as this to encourage primary care physicians to prescribe antibiotics to anyone who might have community-acquired pneumonia, with potential for increased levels of antibiotic resistance, unnecessary costs and potential side-effects.

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REFERENCES:


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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
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REFERENCES


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**β₂-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