were performed after 0, 2, 4 and 6 h. A bactericidal effect was defined as a $\geq 3 \log_{10}$ (99.9% killing) reduction in CFU compared with the initial test inoculum. Colony counts were determined and averaged from each sample.

The results are presented in Fig. 1. Ofloxacin and ciprofloxacin showed bactericidal activity against E. coli J53 (pQR1) at concentrations two-, four- and eight-fold higher than the MICs, beginning after incubation for 2 h and 4 h for ofloxacin and ciprofloxacin, respectively. These results indicated that transfer of QnrA into E. coli J53 did not modify the bactericidal activity of ofloxacin and ciprofloxacin.

Martínez-Martínez et al. [2] reported that QnrA may enhance selection of higher levels of quinolone resistance [2]. The low level of resistance conferred by QnrA may allow the bacterial population to reach a concentration at which secondary chromosomal mutations for higher levels of quinolone resistance may occur. However, the bactericidal activity of fluoroquinolones remained unchanged. Our results indicate that, in the absence of additional chromosome-encoded quinolone resistance mechanisms, QnrA-positive enterobacterial isolates may remain susceptible to the bactericidal effect of fluoroquinolones. Animal infection models, as well as clinical studies, may provide further evidence to support these in-vitro results.

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Correct use of the term ‘pan-drug-resistant’ (PDR) Gram-negative bacteria

We congratulate Hsueh et al. for their recent publication in CMI regarding ‘pan-drug-resistant’ nosocomial Pseudomonas aeruginosa infections [1]. However, we would like to express our disagreement with the use of the term ‘pan-drug-resistant’ (PDR) in this article. We believe that this term should not be used for Gram-negative bacteria that are susceptible to polymyxins. This practice causes confusion among clinicians because it suggests an absence of antimicrobial agents for the management of infections caused by these bacteria, while a potential salvage option is available in the form of intravenous polymyxins.

An isolate of P. aeruginosa should be defined as ‘pan-drug-resistant’ if it is resistant to all seven available anti-pseudomonal classes of antimicrobial agents, namely anti-pseudomonal penicillins, cephalosporins, carbapenems, monobactams, quinolones, aminoglycosides and polymyxins [2]. Several recent studies have used the term ‘pan-drug-resistant’ despite the fact that the isolates had not been tested for their susceptibility to polymyxins [3–5]. For example, a mortality rate of 60% was reported in a study of patients with ‘pan-drug-resistant’ Acinetobacter baumannii infections from Taiwan; however, the isolates were not tested for their in-vitro susceptibility to polymyxins. Furthermore, no polymyxin was used to treat the patient population studied [3,4]. Several studies have now shown that intravenous polymyxins may be useful for the treatment of patients with infections caused by Gram-negative bacteria with in-vitro susceptibility to these antibiotics, even if
the isolates are resistant to antimicrobial agents of other classes.

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Serum procalcitonin levels in patients with mild community-acquired pneumonia

Serum procalcitonin (PCT) has been described as a useful parameter for distinguishing various types of inflammation [1]. A recent study in CMI [2] demonstrated the superiority of PCT for the differential diagnosis of febrile neutropenia in comparison with C-reactive protein. In order to elucidate the value of PCT in comparison with other parameters for diagnosis in patients with mild community-acquired pneumonia, we conducted a study in 116 consecutive patients with community-acquired pneumonia who were receiving oral antibiotic therapy. Patient demographics, medical history, signs and symptoms, X-ray characteristics and laboratory parameters were recorded. An aetiological diagnosis was attempted by blood and sputum culture, tests for Streptococcus pneumoniae (Binax, Portland, ME, USA) and Legionella pneumophila (Biotest AG, Dreieich, Germany) urinalysis, and antibody tests for Mycoplasma pneumoniae (ELISA; Serion Immundiagnostica, Würzburg, Germany), Chlamydia pneumoniae (microimmunofluorescence test; MRL Diagnostics, Cypress, CA, USA) and Legionella pneumophila (immunofluorescence; locally prepared L. pneumophila serogroup 1–14 and Legionella micdadei antigens) in paired samples. Statistical analysis was performed using SPSS v. 10.0 (SPSS Inc., Chicago, IL, USA) and Microsoft Excel 2000.

Complete data sets were available for 99 patients, and the aetiology was established for 62 patients. Patients with atypical (M. pneumoniae, C. pneumoniae and L. pneumophila), typical (S. pneumoniae, Haemophilus influenzae, Moraxella catarrhalis) and unknown aetiology differed in age (p 0.007), chills (p 0.042), dyspnoea (p 0.013), bronchial breathing (p 0.008), PCT (p 0.021) and log PCT (p < 0.0001). One of 42 patients with an atypical aetiology had a PCT level > 0.5 µg/L, compared with nine of 15 patients in the group with typical pneumonia. A substantial overlap was observed between patients with pneumonia of typical, atypical and unknown aetiology with respect to C-reactive protein values, white blood cell counts and the percentage of non-segmented neutrophils, but not for PCT and log PCT values (Fig. 1). In comparison with C-reactive protein, white blood cell and non-segmented neutrophil counts, only the area under the random operator characteristic curve for PCT was significantly different from the diagonal line of no predictive value (p 0.004). There was no difference in PCT levels for patients classified in different risk classes by Fine et al. [3]. The specificity of tests for PCT levels in distinguishing between typical and atypical pneumonia was highest with a cut-off level of 0.48 µg/L.

We conclude that measurement of PCT levels seems to be a useful tool to rule out an atypical aetiology of pneumonia and to limit the use of broad-spectrum antibiotics. The results outlined above support the use of PCT as a tool to help discriminate between patients with lower respiratory tract infections who need antibiotics and those who do not [4].