Pseudomonas aeruginosa. This is, in fact, the point we hoped to make. However, they go on to suggest that these relatively low concentrations of ertapenem will be ineffective against this strain. It is with this issue that we are concerned. In our article, we indicate that the emergence and spread of resistance are greatly enhanced at subinhibitory concentrations; however, resistance emergence is also a dynamic process of the progressive concentration of resistant strains within the clinical population, often by the same or related strains. In the individual case that Brink et al. describe, ertapenem did not select carbapenem-resistant P. aeruginosa; however, we are concerned with the much broader picture for, in the face of thousands or even millions of such challenges, we maintain that selection of resistant strains is likely to occur and progressively increases their concentration in the clinical setting.

With regard to their later comment, the widespread dissemination of extended-spectrum \(\beta\)-lactamases (ESBLs) and transferable \(ampC\) \(\beta\)-lactamases does demonstrate that we have not used cephalosporins optimally. We do, though, have the benefit of hindsight and different strategies; in particular, the use of cephalosporins in combination with a \(\beta\)-lactamase inhibitor might have suppressed the huge problem of cephalosporin resistance that we now face. It should be possible to learn from experience that we must not take individual treatment cases in isolation as a model for drug strategy because, unless we consider the broader potential impact of using weaker drugs, we may well face a new ‘ESBL’ problem, but this time it will be carbapenemases.

Transparency declarations

S. G. B. A. has received an educational grant from AstraZeneca.

References


Correspondence

Keywords: microbiological efficacy, Chlamydia pneumoniae, Mycoplasma pneumoniae

*Tel: +1-718-270-3097; Fax: +1-718-270-1985; E-mail: nhammerschlag@downstate.edu

Sir,

Assessing the microbiological efficacy of antibiotic treatment against respiratory infections caused by atypical organisms, specifically Chlamydia pneumoniae and Mycoplasma pneumoniae, is difficult. Culturing is difficult and not readily available. There are no validated, commercially available nucleic acid amplification tests for either organism. Recently, File et al. reported a study comparing 5 day versus 7 day treatment of community-acquired pneumonia with gemifloxacin. They used serology alone to determine whether patients were infected with C. pneumoniae and M. pneumoniae. The authors state in the Methods section that ‘because only serology was used for identification, bacteriological outcome was presumed on the basis of clinical response’. However, under ‘bacteriologic outcomes’, they state that C. pneumoniae and M. pneumoniae were ‘identified’ and refer to ‘eradication’ of these organisms in Table 3. Unfortunately, serology, especially for C. pneumoniae, is not standardized and correlates poorly with identification of the organism by culture or validated PCR. As many as 40% to 70% of patients with culture-documented C. pneumoniae infection will remain seronegative. Serology does not detect or identify an organism; it indicates possible exposure. We have previously demonstrated in two studies of community-acquired pneumonia in adults, utilizing cultures, that treatment with levofloxacin or moxifloxacin eradicated C. pneumoniae from 80% and 70% of infected patients, respectively. We also demonstrated poor correlation between serology, using the microimmunofluorescence assay, and culture. However, the patients who were microbiological failures were clinical cures, despite persistence of the organism. In vitro activity does predict \(in vivo\) efficacy. The MICs of moxifloxacin and gemifloxacin for C. pneumoniae are very similar.

Use of serology, at best, only allows a clinical endpoint. We need a consistent policy on acceptable criteria for determining microbiological efficacy for C. pneumoniae and M. pneumoniae infection.

Transparency declarations

None to declare.

References

Correspondence


Journal of Antimicrobial Chemotherapy
doi:10.1093/jac/dkm333
Advance Access publication 30 August 2007

Gemifloxacin once daily for 5 days versus 7 days for the treatment of community-acquired pneumonia: a randomized, multicentre, double-blind study—authors’ response

T. M. File1,2*, L. A. Mandell3 and G. Tillotson4†

1Northeastern Ohio Universities College of Medicine, Rootstown, OH, USA; 2Summa Health System, 75 Arch Street, Suite 105, Akron, OH 44304, USA; 3McMaster University School of Medicine, 711 Concession Street, Hamilton, Ontario L8V1C3, Canada; 4Oscient Pharmaceuticals Corporation, 1000 Winter Street, Suite 2200, Waltham, MA 02451, USA

Keywords: Chlamyphilia, microbiological efficacy, clinical cure

*Corresponding author. Tel: +1-330-375-3894; Fax: +1-330-375-6680; E-mail: filet@summa-health.org
†Present address. Replidyne Pharmaceuticals, Louisville, CO, USA.

Sir,

We are in agreement with Dr Hammerschlag1 that assessment for Chlamydiaphila pneumoniae as an aetiology of respiratory infections is difficult; and we share her call for a consistent regulatory policy on acceptable criteria for determining microbiological efficacy. Since serological methods were the only means by which an atypical aetiology was assessed in our paper, we acknowledge this as a potential limitation, but felt that we should include this information to give the reader some impression of the pattern of aetiological agents found in our cases.

We would like to stress that the primary purpose of our paper was to demonstrate the clinical efficacy of a shorter 5 day course of gemifloxacin therapy for mild-to-moderate community-acquired pneumonia with the possible benefits which may accrue. We feel that we have demonstrated this in our paper.2

Transparency declarations

T. M. F. has received recent research funding from Abbott, Arpida AG, AstraZeneca, Bristol–Myers Squibb, Bayer, Binax Incorporated, Cubist, Genzyme, GlaxoSmithKline, Ortho-McNeil, Oscient, Pfizer, Sanofi-Aventis and Wyeth, is a consultant to Sanofi-Aventis, Bayer, GlaxoSmithKline, Ortho-McNeil, Merck, Oscient, Pfizer and Wyeth and is on the Speakers’ Bureau of Abbott, Sanofi-Aventis, GlaxoSmithKline, Merck, Oscient, Pfizer, Schering Plough and Wyeth. Further, he does not own stocks in any company that might be financially affected by the conclusions of this article. L. A. M. has received research funding from Bayer, Pfizer, Sanofi-Aventis and Wyeth, is a consultant to AstraZeneca, Aventis, Bayer, Pfizer, Sanofi-Aventis and Wyeth and is on the Speakers’ Bureau of Aventis, Bayer, Oscient, Pfizer and Sanofi-Aventis. G. S. T. was an employee of Oscent Pharmaceuticals at the time of the study and is currently an employee of Replidyne Pharmaceuticals, Louisville, CO, USA.

References


Journal of Antimicrobial Chemotherapy
doi:10.1093/jac/dkm283
Advance Access publication 25 July 2007

Complete nucleotide sequence of a small qnrS1-carrying plasmid from Salmonella enterica subsp. enterica Typhimurium DT193

Corinna Kehrenberg1, Katie L. Hopkins2, E. John Threlfall2 and Stefan Schwarz1*

1Institut für Tierzucht, Bundesforschungsanstalt für Landwirtschaft (FAL), Höhlystr. 10, 31535 Neustadt-Mariensee, Germany; 2Salmonella Reference Unit, Laboratory of Enteric Pathogens, Health Protection Agency Centre for Infections, 61 Colindale Avenue, London NW9 5EQ, UK

Keywords: quinolone resistance, mobilization, recombination, enteric pathogens

*Corresponding author. Tel: +49-5034-871-241; Fax: +49-5034-871-246; E-mail: stefan.schwarz@fal.de

Sir,

In a recent study on transferable quinolone resistance among Salmonella enterica strains isolated in the UK, qnrS1 genes were identified on plasmids in the four serotypes Typhimurium, Stanley, Virchow and Virginia.1 Since no complete sequence of a qnrS1-carrying plasmid has been available so far, we decided to sequence completely the smallest type of qnrS1-carrying plasmid to gain insight into the structure and putative origin of this plasmid. The plasmid chosen, TPqnrS-1a, was obtained from a multiresistant Salmonella Typhimurium DT193 strain and previously shown to mediate only decreased susceptibility to ciprofloxacin.1

903