against use of tigecycline to treat pulmonary exacerbations in CF patients. Besides, in poorly penetrated anatomic sites such as lung tissue, this may induce the development of resistance.

As Mycobacterium abscessus is increasingly involved in pulmonary infection in CF patients, we do agree with the authors that tigecycline offers exciting therapeutic potential for the rapidly growing mycobacteria (M. abscessus, Mycobacterium chelonae and Mycobacterium fortuitum).\(^1\) Although the clinical data are rather scarce, the susceptibility and PK–PD data seem promising. As the reported MIC\(_{90}\) for M. abscessus is 0.25 mg/L, the expected AUC\(_{24}/\text{MIC}_{90}\) for this pathogen would be 36 in lung tissue and 9.12 in epithelial lining fluid, when calculated as mentioned above.\(^1,6\)

However, we think that in order to make recommendations concerning tigecycline use in CF patients, human studies to define AUC\(_{24}/\text{MIC}_{90}\) for the colonizing/infectious pathogens are needed. We therefore currently agree with the FDA drug safety communication of January 2010 that warned not to use tigecycline in pulmonary infections, especially hospital-acquired and ventilator-associated pneumonia, because of increased mortality risk.

**Transparency declarations**

None to declare.

**References**


**J Antimicrob Chemother** 2011
doi:10.1093/jac/dkr036
Advance Access publication 3 March 2011

**Newer antibacterial agents and their potential role in cystic fibrosis pulmonary exacerbation management—authors’ response**

M. D. Parkins\(^1-3*\) and J. S. Elborn\(^3,4\)

\(^1\)Department of Medicine, University of Calgary, Calgary AB, Canada; \(^2\)Department of Microbiology and Infectious Disease, University of Calgary, Calgary AB, Canada; \(^3\)Northern Ireland Regional Adult Cystic Fibrosis Centre, Belfast City Hospital, Belfast, UK; \(^4\)Centre for Infection and Immunity, Queen’s University of Belfast, Belfast, UK

Corresponding author. Department of Medicine, Foothills Medical Center, 1403 29th Street NW, Calgary, AB, Canada. Tel: +1-403-220-5951; Fax: +1-403-270-2772; E-mail: mdparkin@ucalgary.ca

**Keywords:** tigecycline, pharmacokinetics, pneumonia

Sir,

In response to the comments of Cooreman and Jeurissen\(^1\) on our recent article\(^6\) we would like to make several clarifications. Although *Pseudomonas aeruginosa* remains the most commonly isolated pathogen in cystic fibrosis (CF), this pattern is changing. The practice of early eradication of *P. aeruginosa* with aerosolized antibiotics has become the standard of care.\(^3\) In clinics in which this practice has been aggressively adopted, *P. aeruginosa* prevalence rates have dwindled to <5% in those CF patients <18 years of age.\(^4\) According to Cooreman and Jeurissen,\(^1\) CF physicians are increasingly treating pulmonary exacerbations (PEx) caused by pathogens other than *P. aeruginosa* such as *Stenotrophomonas maltophilia*, *Achromobacter xylosoxidans*, *Staphylococcus aureus* and so on. It is PEx with these pathogens, and not *P. aeruginosa*, for which we have advocated the use of tigecycline, as clearly indicated in our review.

The FDA has recently released a drug safety communication cautioning practitioners on the use of tigecycline in severe infections based on the pooled analysis of 13 trials involving >7000 patients where all-cause mortality was observed to be increased by 0.6% [95% confidence interval (CI) 0.1–1.2] relative to comparator antibiotics.\(^5\) However, the FDA did not warn that tigecycline should not be used in pulmonary infections as asserted by Cooreman and Jeurissen.\(^1\) Furthermore, community-acquired pneumonia remains an approved indication for the use of tigecycline. Most of the increased mortality in tigecycline-treated patients was attributable to hospital-acquired pneumonia, in particular, ventilator-associated pneumonia (VAP). This observation may not be relevant to CF PEx, a disease for which no antibiotic has an FDA-approved indication. PEx in CF are overwhelmingly caused by chronically colonizing pathogens and not through the new acquisition of pathogens\(^6,7\) and as such the empirical provision of PEx antibacterials based on prior sputum results is commonplace and supported. The bacteriostatic nature of tigecycline, postulated to be a potential detractor in the management of respiratory infections, is not relevant in CF PEx as eradication of chronically infecting pathogens is generally not possible.\(^3\) The use of tigecycline in CF has been reported only rarely, but clinical outcomes have been favourable.\(^8\)

CF-specific pharmacokinetic data for tigecycline (as well as for many antibiotics) continue to be lacking and the lower AUC data observed in patients in tigecycline trials with VAP and associated lower clinical cure rates\(^9\) emphasize the importance of understanding the pharmacokinetics of antimicrobials in disease-specific settings. With the increasing burden of multidrug-resistant pathogens in CF and their associated increased risk of mortality,\(^10\) new therapeutic options are desperately required.
Sir,

In their recent article on spondylodiscitis, Gouliouris et al.\(^1\) seemed to imply that the haematogenous source of infection was mainly arterial. Strangely there was no mention of venous spread through Batson’s valveless venous plexus,\(^2\) which has been much cited as a route of spread of pelvic malignancies to non-contiguous sites\(^3\) and by ageing microbiologists as a route for coliform bacteria to cause lumbar vertebral spondylodiscitis.

Of course this hypothesis may be incorrect and may have been rejected by Gouliouris et al.\(^1\) as unproven. If I have misled numerous cycles of medical students over the decades I would welcome clarification by the authors who have reviewed the clinical literature.

I have repeated the Medline search indicated in the article by Gouliouris et al.\(^1\) and combined it with a search against Batson, limited to veins, spinal canal, low back pain and Batson appearing in the title or abstract. On limiting this to human and English language it yielded 183 references, none of which appeared to directly address the issue of venous spread.

So the question remains, are the 7%–33% quoted cases caused by Enterobacteriaceae delivered to their target by the venous or arterial route and, if the answer is unknown, how can this hypothesis be tested?

F. Z. Akcam*, O. Kaya and T. Ceylan

Department of Infectious Diseases, Suleyman Demirel University Medical School, Isparta, Turkey

*Corresponding author. E-mail: fzeynep@med.sdu.edu.tr

**Keywords:** laboratory methods, microbiology, microorganism

Sir,

We read with great interest the article by Gouliouris et al.\(^1\) regarding diagnosis and management of spondylodiscitis.

As interested physicians will know, spinal infections can be troublesome owing to the difficulties in their diagnosis and treatment. In this respect, the article has contributed to the literature.