In Vitro Activities of Tigecycline (GAR-936) and 12 Other Antimicrobial Agents against 90 Eikenella corrodens Clinical Isolates

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Eikenella corrodens is involved in a wide variety of oral and nonoral infections (4, 24). The increasing isolation of this microorganism in cases of periodontitis, parotitis, sinusitis, osteomyelitis, oral and nonoral abscesses, bacteremia, endocarditis, bite wound, intraabdominal, and pulmonary infections (1, 6–11, 13–18, 23–25) and its variable antimicrobial susceptibility make necessary the routine determination of susceptibility to antimicrobial agents. Tigecycline (GAR-936) is a new glycylcycline necessary the routine determination of susceptibility to anti-
bite wound, intraabdominal, and pulmonary infections (1,6–11,
croorganism in cases of periodontitis, parotitis, sinusitis, osteo-
nonoral infections (4, 24). The increasing isolation of this mi-

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The activity of tigecycline was compared with those of other antimicrobials against 90 isolates of Eikenella corrodens. The MIC at which 90% of the isolates were inhibited was 2 μg/ml for tigecycline and 1, ≤0.5/0.25, 0.5, ≤0.12, ≤2, and 0.5 μg/ml for ampicillin, amoxicillin-clavulanate, cefotaxime, imipenem, chloramphenicol, and ciprofloxacin, respectively.

Eikenella corrodens was provided by Wyeth-Ayerst Research Laboratories (Saint Davids, Pa.) and tested at twofold increasing concentrations within the following ranges: ampicillin, 0.25 to 16 μg/ml; amoxicillin-clavulanic acid, 0.5/0.25 to 8/4 μg/ml; cefuroxime, 0.5 to 8 μg/ml; cefotaxime, 0.06 to 4 μg/ml; imipenem, 0.12 to 4 μg/ml; erythromycin, 0.25 to 8 μg/ml; azithromycin, 0.5 to 4 μg/ml; ciprofloxacin, 0.12 to 4 μg/ml; gentamicin, 4 μg/ml; trimethoprim-sulfamethoxazole, 0.5/9.5 to 2/38 μg/ml; chloramphenicol, 2 to 8 μg/ml; tetracy-
cline, 2 to 8 μg/ml. Following inoculation with the same inoc-
ulum for the commercial and in-house panels (final inoculum concentration, ca. 5 × 10⁵ CFU/ml), MIC trays were incubated at 35°C in ambient air for 24 h before examination. Staphylo-
coccus aureus ATCC 29213, Enterococcus faecalis ATCC 29212, and Streptococcus pneumoniae ATCC 49619 were used as control strains.

Minimum bactericidal concentrations (MBCs) of tigecycline were defined as those causing a 99.9% reduction of the initial inoculum and were determined by subculturing 0.1 ml of every MIC test sample onto Columbia blood agar plates. Colonies were counted after 24 h of incubation at 35°C in ambient air.

Tigecycline inhibited all of the isolates tested at concentrations between ≤0.06 and 4 μg/ml (Table 1). The MIC of tigecycline at which 90% of the isolates were inhibited (MIC₉₀) was 2 μg/ml; 75% of the isolates were susceptible to ≤0.5 μg/ml of tigecycline per ml, 10 (11%) required 2 to 4 μg/ml for inhibition, and 4 were inhibited by ≤0.06 μg/ml. Nine strains resistant to macrolides (erythromycin and azithromycin MICS of >4 μg/ml) were susceptible to tigecycline, as were three beta-lactamase-producing and four tetracycline-resistant strains.

Tigecycline showed bacteriostatic activity against all of the isolates tested (MBC for 90% of the strains tested [MBC₉₀], 32 μg/ml; MBC range, 4 to >64 μg/ml). The MIC₉₀ of amoxicillin, amoxicillin-clavulanic acid, cefotaxime, chloramphenicol, ciprofloxacin, and tetracycline were 1, ≤0.5/0.25, 0.5, ≤2, 0.5, and ≤2 μg/ml, respectively. Imipenem showed the lowest MICs (MIC₉₀ of ≤0.12 μg/ml). The MICS (micrograms per milliliter) of tigecycline for the control organisms were as follows: for S. aureus ATCC 29213 (n = 9), 0.12 (n = 6) and 0.25 (n = 3); for E. faecalis ATCC 29212 (n = 9), ≤0.06 (n = 7) and 0.12 (n = 2); and for S. pneumoniae ATCC 49619 (n = 1), 0.03. All results were within quality control limits for all of the antimi-
crobials tested against all of the control strains, in the cases in which NCCLS-approved quality control ranges were available.

Tigecycline inhibits protein synthesis, including that of isolates resistant to tetracycline by either ribosomal protection or active efflux (22). We have only found one study in which the activity of tigecycline was tested against 18 isolates of *E. corrodens* (12), and all were inhibited by ≤0.06 μg/ml. We included many isolates of *E. corrodens*, 90% of which were inhibited by tigecycline at 2 μg/ml. Although penicillin-resistant strains have been isolated (15, 24), *E. corrodens* is usually susceptible to beta-lactam antibiotics, such as penicillin, ampicillin, cefuroxime, ceftoxitin, and cefotaxime, but resistant to cefazolin and cephalothin (1, 11, 17). We describe three strains (3%) producing beta-lactamase that were susceptible to amoxicillin-clavulanic acid. Since many of the infections produced by *E. corrodens* are treated with penicillins, in allergic patients, alternative antimicrobial agents are necessary. Tetracyclines can be used, but resistance to these compounds has been reported, ranging from 8 to 17.8% (13, 15, 17). We found four isolates (4%) resistant to tetracycline that were susceptible to minocycline. In our study, tigecycline inhibited beta-lactamase-producing and tetracycline-resistant isolates. *E. corrodens* has been reported to be usually resistant to erythromycin and clarithromycin but susceptible to azithromycin (9). Our results show that the MIC₉₀ of erythromycin and azithromycin were 4 and 2 μg/ml, respectively, and tigecycline inhibited isolates for which the macrolide MICs were >4 μg/ml. Previous studies have demonstrated good activity of the fluoroquinolones against *E. corrodens* (10), as we found. Gentamicin and chloramphenicol inhibited 90% of the isolates at ≤0.06 and ≤0.12 μg/ml, respectively, and the activity of trimethoprim-sulfamethoxazole against *E. corrodens* was poor (MIC₉₀ >2/38 μg/ml). We found an absence of bactericidal activity of tigecycline against *E. corrodens* (MBC₉₀ of >32 μg/ml).

This study contributes to the knowledge of the activities of different antimicrobial agents against *E. corrodens*, confirms the activity of tigecycline against this pathogen, and suggests a potential therapeutic role for tigecycline in the treatment of infections that involve this microorganism.

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### Notes

**REFERENCES**


