ANTIMICROBIAL AGENTS AND CHEMOTHERAPY, Mar. 1996, p. 772–775 Vol. 40, No. 3
0066-4804/96/$04.00+0

NOTES

Susceptibilities of Non-*Pseudomonas aeruginosa* Gram-Negative Nonfermentative Rods to Ciprofloxacin, Ofloxacin, Levofloxacin, D-Ofloxacin, Sparfloxacin, Ceftazidime, Piperacillin, Piperacillin-Tazobactam, Trimethoprim-Sulfamethoxazole, and Imipenem

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Received 14 July 1995/Returned for modification 25 September 1995/Accepted 18 December 1995

Agar dilution MICs of 10 agents against 410 non-*Pseudomonas aeruginosa* gram-negative nonfermentative rods were determined. MICs at which 50 and 90% of the isolates were inhibited, respectively, were as follows (in micrograms per milliliter): sparfloxacin, 0.5 and 8.0; levofloxacin, 1.0 and 8.0; ciprofloxacin, 2.0 and 32.0; ofloxacin, 2.0 and 32.0; D-ofloxacin, 32.0 and >64.0; ceftazidime, 8.0 and 64.0; piperacillin with or without tazobactam, 16.0 and >64.0; trimethoprim-sulfamethoxazole, 0.5 and >64.0; imipenem, 2.0 and >64.0. With the exception of those for *Stenotrophomonas maltophilia*, *Burkholderia cepacia*, and *Alcaligenes faecalis-A. odorans*, agar dilution MICs for all strains tested were within 1 dilution of inhibitory (bacteriostatic) levels as determined by time-kill methodology.

Antimicrobial susceptibility patterns of nonfermenters differ from those of the members of the family *Enterobacteriaceae* in many respects, and many groups of nonfermenters have susceptibility spectra which differ from that of *Pseudomonas aeruginosa* (1–3, 9, 13, 33). In this work, we employed agar dilution to study in vitro susceptibilities of 410 non-*P. aeruginosa* gram-negative nonfermentative rods to ciprofloxacin, ofloxacin, levofloxacin, D-ofloxacin, sparfloxacin, ceftazidime, piperacillin with or without tazobactam, trimethoprim-sulfamethoxazole (SXT), and imipenem. Additionally, activities of these antimicrobial agents against 10 selected strains were studied by time-kill methodology.

Organisms (Table 1) were recent clinical isolates identified by standard methods (11, 32). Strains were all different isolates from different patients hospitalized at different times at Hershey Medical Center, Hershey, Pa.; University Hospitals of Cleveland, Cleveland, Ohio; Cleveland Clinic, Cleveland, Ohio; and Hôpital St. Louis, Paris, France, during the past 7 years. Twenty-five stock strains of less commonly isolated species were obtained from M. J. Pickett, University of California—Los Angeles, Los Angeles, Calif. Antibiotic powders were obtained from the respective manufacturers.

MICs were determined by the agar dilution method recommended by the National Committee for Clinical Laboratory Standards (19) by using Mueller-Hinton agar (BBL Microbiology Systems, Cockeysville, Md.), with 5% sheep blood for *Moraxella* strains. Time-kill experiments were performed as described previously (21), by using Mueller-Hinton broth (Difco Laboratories, Detroit, Mich.). Bacteriostatic and bactericidal values (21) were determined at 24 h. The problem of drug carryover was addressed as described previously (21).

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Results of MIC susceptibility testing are presented in Table 1. For all 410 strains tested, MICs at which 50% of the isolates were inhibited (MIC_{50}s) and MIC_{90}s, respectively (in micrograms per milliliter), were as follows: ciprofloxacin, 2.0 and 32.0; ofloxacin, 2.0 and 32.0; levofloxacin, 1.0 and 8.0; D-ofloxacin, 32.0 and >64.0; sparfloxacin, 0.5 and 8.0; ceftazidime, 8.0 and 64.0; piperacillin, 16.0 and >64.0; piperacillin-tazobactam, 16.0 and >64.0; SXT, 0.5 and >64.0; imipenem, 2.0 and >64.0. Levofloxacin yielded MICs which were 1 or 2 dilutions lower than those of ofloxacin. Levofloxacin and sparfloxacin were the most active of the four quinolones, followed by ciprofloxacin and ofloxacin, with D-ofloxacin yielding no appreciable activity. Of the 55 *Burkholderia* (*Pseudomonas*) *cepacia* strains tested, 26 were isolated from cystic fibrosis patients and 29 were isolated from non-cystic fibrosis patients. β-Lactams and SXT were more active in the non-cystic fibrosis group than in the cystic fibrosis group. MICs of imipenem and of piperacillin with or without tazobactam for *Stenotrophomonas* (*Xanthomonas*) *maltophilia* strains were uniformly high, but MICs of levofloxacin, sparfloxacin, and SXT were lower.

MICs for *Pseudomonas fluorescens-P. putida* strains (not differentiated from one another) with all quinolones except D-ofloxacin were low. MICs of all drugs except SXT for *Brevundimonas* (*Pseudomonas*) *diminuta* strains were high; *Pseudomonas stutzeri* and *Sphingobacterium* (*Flavobacterium*) *multivorum* organisms were inhibited by low concentrations of all quinolones except D-ofloxacin, as well as by SXT. *Alcaligenes faecalis-A. odorans* strains were more susceptible than were *Alcaligenes xylosoxidans* organisms, but MIC_{90}s for all *Alcaligenes* species were ≥8.0 μg/ml with all agents tested. MIC_{50}s for *Moraxella* species were low with all drugs tested. *Chryseobacterium meningosepticum* and *Flavobacterium odoratum* were the most resistant to all compounds; however, MICs for *Chryseobacterium indologenes-C. gleum* strains (formerly

TABLE 1. MICs for gram-negative nonfermentative strains tested

Strain (no. of isolates) and antimicrobial agent	MIC (μg/ml)		
	Range	50%	90%
Pseudomonas fluorescens-P. putida (15)			
Ciprofloxacin	<0.03–2.0	0.25	0.25
Ofloxacin	<0.03–8.0	0.25	1.0
Levofloxacin	<0.03–4.0	0.25	1.0
D-Ofloxacin	2.0–>64.0	16.0	32.0
Sparfloxacin	<0.03–2.0	0.25	1.0
Ceftazidime	0.5–16.0	2.0	8.0
Piperacillin	2.0–>64.0	16.0	>64.0
Piperacillin-tazobactam	2.0–>64.0	16.0	>64.0
SXT	1.0–>64.0	16.0	>64.0
Imipenem	0.125–>64.0	2.0	2.0
Pseudomonas stutzeri (7)			
Ciprofloxacin	<0.03–1.0	0.06	
Ofloxacin	<0.03–2.0	0.06	
Levofloxacin	<0.03–2.0	0.06	
D-Ofloxacin	2.0–32.0	16.0	
Sparfloxacin	<0.03–0.5	0.06	
Ceftazidime	0.5–8.0	2.0	
Piperacillin	0.5–4.0	2.0	
Piperacillin-tazobactam	0.25–4.0	2.0	
SXT	0.016–8.0	0.125	
Imipenem	0.125–2.0	0.5	
Brevundimonas (*Pseudomonas*) *diminuta* (11)			
Ciprofloxacin	8.0–>64.0	32.0	64.0
Ofloxacin	8.0–64.0	16.0	32.0
Levofloxacin	2.0–64.0	8.0	32.0
D-Ofloxacin	64.0–>64.0	>64.0	>64.0
Sparfloxacin	0.25–16.0	1.0	8.0
Ceftazidime	16.0–>640	64.0	64.0
Piperacillin	8.0–16.0	8.0	16.0
Piperacillin-tazobactam	1.0–8.0	2.0	8.0
SXT	0.06–1.0	0.5	1.0
Imipenem	1.0–8.0	1.0	8.0
Stenotrophomonas maltophilia (76)			
Ciprofloxacin	0.25–>64.0	4.0	8.0
Ofloxacin	0.06–16.0	2.0	8.0
Levofloxacin	0.06–8.0	1.0	4.0
D-Ofloxacin	0.125–>64.0	32.0	>64.0
Sparfloxacin	0.03–16.0	0.5	2.0
Ceftazidime	0.06–>64.0	16.0	64.0
Piperacillin	16.0–>64.0	64.0	>64.0
Piperacillin-tazobactam	8.0–>64.0	64.0	>64.0
SXT	0.06–>64.0	0.25	0.5
Imipenem	16.0–>64.0	>64.0	>64.0
Burkholderia cepacia cystic fibrosis (26)			
Ciprofloxacin	0.25–64.0	2.0	32.0
Ofloxacin	0.5–>64.0	2.0	32.0
Levofloxacin	0.25–64.0	2.0	16.0
D-Ofloxacin	16.0–>64.0	>64.0	>64.0
Sparfloxacin	<0.03–64.0	2.0	8.0
Ceftazidime	2.0–>64.0	8.0	>64.0
Piperacillin	4.0–>64.0	8.0	>64.0
Piperacillin-tazobactam	4.0–>64.0	8.0	>64.0
SXT	4.0–>64.0	32.0	>64.0
Imipenem	1.0–>64.0	16.0	>64.0
Burkholderia cepacia non-cystic fibrosis (29)			
Ciprofloxacin	<0.03–64.0	2.0	16.0
Ofloxacin	<0.03–32.0	4.0	32.0
Levofloxacin	<0.03–32.0	4.0	16.0
D-Ofloxacin	<0.03–>64.0	>64.0	>64.0
Sparfloxacin	<0.03–16.0	1.0	8.0
Ceftazidime	<0.03–16.0	8.0	16.0
Piperacillin	2.0–>64.0	8.0	32.0
Piperacillin-tazobactam	2.0–>64.0	8.0	32.0
SXT	0.25–>64.0	4.0	16.0
Imipenem	<0.03–>64.0	8.0	32.0
Acinetobacter genospecies (72)			
Ciprofloxacin	<0.03–>64.0	1.0	64.0
Ofloxacin	0.06–64.0	1.0	16.0
Levofloxacin	0.06–32.0	0.25	8.0
D-Ofloxacin	0.25–>64.0	16.0	>64.0
Sparfloxacin	0.03–16.0	0.125	8.0
Ceftazidime	0.125–>64.0	8.0	>64.0
Piperacillin	4.0–>64.0	32.0	>64.0
Piperacillin-tazobactam	0.016–>64.0	16.0	64.0
SXT	0.06–>64.0	0.25	16.0
Imipenem	<0.03–16.0	1.0	2.0
Alcaligenes xylosoxidans (40)[a]			
Ciprofloxacin	1.0–64.0	16.0	32.0
Ofloxacin	0.5–64.0	32.0	32.0
Levofloxacin	0.5–64.0	8.0	32.0
D-Ofloxacin	16.0–>64.0	>64.0	>64.0
Sparfloxacin	0.125–64.0	4.0	16.0
Ceftazidime	2.0–>64.0	16.0	>64.0
Piperacillin	0.5–>64.0	1.0	32.0
Piperacillin-tazobactam	0.25–>64.0	1.0	16.0
SXT	0.125–>64.0	16.0	>64.0
Imipenem	0.5–>64.0	4.0	>64.0
Alcaligenes faecalis-A. odorans (24)			
Ciprofloxacin	<0.03–32.0	2.0	16.0
Ofloxacin	0.06–32.0	4.0	16.0
Levofloxacin	<0.03–16.0	1.0	8.0
D-Ofloxacin	0.5–>64.0	64.0	>64.0
Sparfloxacin	<0.03–16.0	1.0	8.0
Ceftazidime	0.5–>64.0	16.0	64.0
Piperacillin	0.25–32.0	1.0	32.0
Piperacillin-tazobactam	0.25–32.0	1.0	16.0
SXT	1.0–>64.0	8.0	>64.0
Imipenem	<0.03–>64.0	1.0	32.0
Moraxella spp.[b] (9)			
Ciprofloxacin	<0.03–4.0	0.06	
Ofloxacin	0.06–4.0	0.125	
Levofloxacin	<0.03–1.0	0.06	
D-Ofloxacin	1.0–>64.0	4.0	
Sparfloxacin	<0.03–0.5	0.03	
Ceftazidime	0.125–64.0	8.0	
Piperacillin	1.0–32.0	1.0	
Piperacillin-tazobactam	0.016–32.0	0.016	
SXT	0.06–0.5	0.25	
Imipenem	<0.03–2.0	0.125	
Flavobacterium odoratum (13)			
Ciprofloxacin	0.125–64.0	4.0	64.0
Ofloxacin	0.5–>64.0	2.0	64.0
Levofloxacin	0.125–>64.0	1.0	32.0
D-Ofloxacin	8.0–>64.0	>64.0	>64.0
Sparfloxacin	0.06–32.0	1.0	32.0
Ceftazidime	8.0–>64.0	32.0	>64.0
Piperacillin	8.0–>64.0	16.0	>64.0
Piperacillin-tazobactam	4.0–64.0	8.0	32.0
SXT	1.0–>64.0	4.0	>64.0
Imipenem	1.0–32.0	8.0	32.0
Chryseobacterium meningosepticum (13)			
Ciprofloxacin	0.125–16.0	4.0	16.0
Ofloxacin	0.25–64.0	4.0	32.0
Levofloxacin	0.125–32.0	2.0	16.0
D-Ofloxacin	8.0–>64.0	32.0	>64.0
Sparfloxacin	0.03–16.0	1.0	8.0

TABLE 1—*Continued*

Strain (no. of isolates) and antimicrobial agent	MIC (μg/ml)		
	Range	50%	90%
Ceftazidime	4.0–>64.0	16.0	64.0
Piperacillin	1.0–>64.0	16.0	>64.0
Piperacillin-tazobactam	0.5–64.0	4.0	64.0
SXT	0.5–>64.0	>64.0	>64.0
Imipenem	0.25–64.0	4.0	64.0
Chryseobacterium indologenes-C. gleum (11)			
Ciprofloxacin	0.125–64.0	2.0	8.0
Ofloxacin	0.25–64.0	2.0	8.0
Levofloxacin	0.125–16.0	1.0	2.0
D-Ofloxacin	8.0–>64.0	32.0	64.0
Sparfloxacin	0.03–8.0	0.125	2.0
Ceftazidime	0.25–64.0	8.0	8.0
Piperacillin	0.5–>64.0	8.0	>64.0
Piperacillin-tazobactam	0.016–>64.0	4.0	>64.0
SXT	0.06–>64.0	0.25	>64.0
Imipenem	1.0–32.0	4.0	32.0
Sphingobacterium multivorum (12)			
Ciprofloxacin	0.03–2.0	0.25	1.0
Ofloxacin	0.125–4.0	0.25	2.0
Levofloxacin	<0.03–0.5	0.125	0.25
D-Ofloxacin	2.0–32.0	16.0	32.0
Sparfloxacin	<0.03–1.0	0.06	0.25
Ceftazidime	0.5–64.0	8.0	32.0
Piperacillin	32.0–>64.0	64.0	>64.0
Piperacillin-tazobactam	32.0–>64.0	64.0	>64.0
SXT	0.06–0.125	0.06	0.125
Imipenem	0.5–32.0	16.0	32.0
Miscellaneous spp.[c] (52)			
Ciprofloxacin	<0.03–64.0	0.5	4.0
Ofloxacin	<0.03–>64.0	0.5	4.0
Levofloxacin	<0.03–64.0	0.5	2.0
D-Ofloxacin	0.125–>64.0	32.0	>64.0
Sparfloxacin	<0.03–32.0	0.25	2.0
Ceftazidime	<0.03–>64.0	4.0	64.0
Piperacillin	0.06–>64.0	2.0	>64.0
Piperacillin-tazobactam	0.016–>64.0	2.0	>64.0
SXT	0.03–1.0	0.06	1.0
Imipenem	<0.03–>64.0	1.0	8.0
All strains (410)			
Ciprofloxacin	<0.03–>64.0	2.0	32.0
Ofloxacin	<0.03–>64.0	2.0	32.0
Levofloxacin	<0.03–>64.0	1.0	8.0
D-Ofloxacin	0.125–>64.0	32.0	>64.0
Sparfloxacin	<0.03–64.0	0.5	8.0
Ceftazidime	<0.03–>64.0	8.0	64.0
Piperacillin	0.06–>64.0	16.0	>64.0
Piperacillin-tazobactam	0.016–>64.0	16.0	>64.0
SXT	0.016–>64.0	0.5	>64.0
Imipenem	<0.03–>64.0	2.0	>64.0

[a] Biovar xylosoxidans (20 isolates) and biovar denitrificans (20).
[b] *Moraxella osloensis* (7 isolates) and *Moraxella nonliquefaciens* (2).
[c] *Shewanella putrefaciens* (4 isolates), *Pseudomonas alcaligenes* (4), *Pseudomonas pseudoalcaligenes* (1), *Pseudomonas mendocina* (3), *Pseudomonas testosteroni* (2), *Pseudomonas* group 2 (2), *Oligella ureolytica* (1), *Sphingomonas paucimobilis* (3), *Burkholderia* (*Pseudomonas*) *thomasii-B. pickettii* (6), *Brevundimonas* (*Pseudomonas*) *vesicularis* (3), *F. oryzihabitans* (4), *Chromobacterium violaceum* (2), a *Methylobacterium* sp. (1), *Comamonas acidovorans* (3), *Bordetella bronchiseptica* (4), *Weeksella virosa* (1), *Ochrobactrum anthropi* (2), CDC IV C-2 (2), CDC EO-2 (2), CDC M-6 (1), and CDC EF-a (1).

Flavobacterium indologenes-F. gleum) were lower, especially those of levofloxacin and sparfloxacin.

With the exception of those for *Stenotrophomonas maltophilia*, *Burkholderia cepacia*, and *Alcaligenes faecalis-A. odorans*, agar dilution MICs were all within 1 dilution of concentrations required to inhibit growth in the broth dilution system. In the other cases, bacteriostatic levels in broth were 2 to 3 dilutions higher than agar dilution MICs. For many nonfermenter strains, the MBCs were >1 dilution higher than MICs. This was particularly the case for *Stenotrophomonas maltophilia*, *Burkholderia cepacia*, and *Alcaligenes faecalis-A. odorans*, where regrowth occurred after 24 h for all quinolones tested. Higher concentrations of ceftazidime were required for a bactericidal versus a bacteriostatic effect.

Among the quinolones studied, sparfloxacin and levofloxacin yielded the greatest activity, followed by ciprofloxacin and ofloxacin. D-Ofloxacin had essentially no appreciable activity. Most previous reports have documented lower sparfloxacin MICs in comparison with those of ciprofloxacin and ofloxacin for gram-negative nonfermenters (6, 14, 16, 17, 30, 31). However, Rolston and coworkers have reported MICs of ciprofloxacin which were a few dilutions below those of sparfloxacin against some gram-negative nonfermenters isolated from cancer patients (25). Although MICs of ciprofloxacin and ofloxacin were similar for these organisms, levofloxacin MICs for all organism groups tested have been reported to be 1 or 2 dilutions lower than those for ofloxacin (8, 10, 20, 29). Our results mirror the latter findings. Ceftazidime, while being very active against *P. aeruginosa*, was less active against most species in the current study. Resistance to imipenem (MICs of ≥16.0 μg/ml) and piperacillin with or without tazobactam (MICs of ≥64.0 μg/ml) was observed with all commonly encountered species.

Stenotrophomonas maltophilia exhibited the antimicrobial susceptibility pattern typical of this organism, including susceptibility to quinolones and SXT and high-level resistance to imipenem (1–3, 6, 8, 10, 13, 15–17, 30, 31, 33). β-Lactam MICs for *Burkholderia cepacia* from cystic fibrosis patients were higher than those for the same organism from non-cystic fibrosis patients, but quinolone resistance patterns did not differ significantly. High carbapenem and SXT MICs for this species have been described (9, 17, 33). Regrowth was also observed for *Burkholderia cepacia* in time-kill experiments.

The genus *Acinetobacter* currently comprises 19 genospecies (32). No attempt was made in the current study to delineate the isolates' exact genospecies identification. Comparison of *Acinetobacter* susceptibility patterns has been complicated by recent changes in classification (32). Previous studies (1–4, 6, 10, 20, 23, 29) have shown this group to be more susceptible to quinolones, ceftazidime, and imipenem than we found in the present study. However, other workers have documented similarly high MICs of some or all compounds (8, 13, 27, 31, 33), and resistance of *Acinetobacter* species to quinolones and nonquinolones has been described (9, 14). Seifert and coworkers have described increased resistance among isolates of *Acinetobacter baumannii* biotype 9 (their commonest isolate) and biotype 6, compared with resistance among nine other *Acinetobacter* genospecies (26).

MICs of quinolones, ceftazidime, and imipenem for *Alcaligenes* species were higher than reported previously (1, 3, 5, 13, 18, 33). However, more-recent publications have documented high quinolone MICs, β-lactamase production, and higher β-lactam MICs for some strains (7, 12). Regrowth in time-kill experiments may also play a role in antimicrobial resistance of these organisms.

Resistance of *Flavobacterium* and *Chryseobacterium* spp. to multiple agents, including SXT, has been described (1, 3, 9, 13, 22, 33). Susceptibility patterns obtained in this study were similar to those previously reported for *P. fluorescens-P. putida* (1–3, 13, 33), *Moraxella* spp., *P. stutzeri*, *Flavimonas oryzihabi-*

tans, *Sphingomonas paucimobilis*, and *Sphingobacterium multivorum* (1–3, 13, 17, 24, 28).

Widespread resistance to SXT, ceftazidime, imipenem, and piperacillin with or without tazobactam, together with significantly increased bactericidal compared with bacteriostatic levels for most strains with ceftazidime, limits use of these compounds in treatment of infections caused by non-*P. aeruginosa* nonfermenters. SXT is active against *Stenotrophomonas maltophilia* and many less commonly encountered species. This study highlights resistance of non-*P. aeruginosa* gram-negative nonfermenters to SXT, fluoroquinolones, and β-lactams and demonstrates the need for development of agents with activity against this group of organisms. Clinical studies are necessary to test the relevance of the increased activity of sparfloxacin and levofloxacin against these strains.

This study was supported by a grant from Hoechst-Marion-Roussel, Clinical Pharmacology and Anti-Infectives, Romainville, France.

We thank G. Hall (Cleveland Clinic), M. J. Pickett (University of California—Los Angeles), and A. Philippon (Hôpital St. Louis, Paris, France) for provision of cultures.

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