Journal of Antimicrobial Chemotherapy (2002) **49**, 697–702

JAC

Correspondence

In vitro antimicrobial susceptibility of clinical isolates of *Aeromonas caviae*, *Aeromonas hydrophila* and *Aeromonas veronii* biotype sobria

J Antimicrob Chemother 2002; **49:** 701–702

Jordi Vila[a]*, Francesc Marco[a], Lara Soler[b], Matilde Chacon[b] and Maria José Figueras[b]

[a]*Laboratorio de Microbiología, Institut d'Infeccions i Immunologia, Institut d'Investigació Biomèdica August Pi i Sunyer, Facultat de Medicina, Universitat de Barcelona, Villarroel 170, 08036 Barcelona;* [b]*Departament de Ciències Mèdiques Bàsiques, Facultat de Medicina i Ciències de la Salut, Universitat Rovira i Virgili, Sant Llorenç 21, 43201 Reus, Spain*

*Corresponding author. Tel: +34-93-227-55-22; Fax: +34-93-227-93-72; E-mail: vila@medicina.ub.es

Sir,

The genus *Aeromonas* comprises 14 species, although its taxonomy has not yet been resolved.[1] The main species considered to be pathogenic in humans are *Aeromonas hydrophila, Aeromonas caviae* and *Aeromonas veronii* biotype sobria.[1] These species can cause both gastrointestinal and extraintestinal infectious diseases. *Aeromona*s gastroenteritis is generally self-limiting and, except in immunocompromised patients, antibiotic treatment is unnecessary. However, for extraintestinal infections the susceptibility patterns should be known in order to impliment appropriate therapy.

The main objective of this study was to evaluate the activity of 24 antimicrobial agents against *A. caviae*, *A. hydrophila* and *A. veronii* biotype sobria clinical isolates.

Forty-three *Aeromonas* spp. clinical isolates were studied. Strains were distributed by species according to sample source as follows: 19 *A. caviae* strains (14 isolated from faeces, three from blood, one from an abscess, one from cellulitis), 14 *A. veronii* strains (12 from faeces, one from blood, one from an ulcer) and 10 *A. hydrophila* strains (seven isolated from faeces, two from joint fluids, one from a wound). All strains were identified to the species level by the 16S rDNA-RFLP method.[2]

Dade-microscan Combo Urine IS panels (Dade Behring, West Sacramento, CA, USA) were used for susceptibility testing. The panels were inoculated according to the manufacturer's guidelines and incubated overnight in the Walk-away System. *Escherichia coli* ATCC 25922 and *Pseudomonas aeruginosa* ATCC 27853 were used as quality control strains.

The results of testing the 43 *Aeromonas* spp. strains against the 24 antimicrobial agents are shown in the Table. Regarding the β-lactam antibiotics, the *Aeromonas* spp. strains analysed were uniformly resistant to ampicillin, as expected. Piperacillin was more active than ticarcillin against all species. The percentage of piperacillin-susceptible strains was >93%, whereas the percentage of ticarcillin-susceptible strains ranged from 14% to 74%, *A. caviae* being more susceptible than *A. veronii* and *A. hydrophila*. The combination of clavulanic acid and amoxicillin enhanced antibacterial activity, whereas tazobactam did not enhance the effect of piperacillin. All the cephalosporins tested except cefazolin showed very good activity against the different *Aeromonas* spp. tested. Susceptibility to cefazolin was higher for *A. veronii* (79%) than for *A. caviae* (53%) and *A. hydrophila* (40%), in agreement with previously described data.[1,4] As well as reporting a similar percentage of susceptibility to cefazolin for *A. veronii* (83%), Overman & Janda[4] also found similar results with *Aeromonas trota* (80%). All strains tested were susceptible to aztreonam and imipenem. In a previous study analysing 12 clinical isolates of *A. veronii*, 67% were resistant to imipenem.[4]

Among the aminoglycosides, gentamicin and amikacin were more active than tobramycin. This tobramycin-resistant, gentamicin-susceptible duality has also been observed in strains isolated in Australia, Taiwan and the USA.[1,4,5]

In previous studies,[1,4,5] fluoroquinolones showed good activity against all species of *Aeromonas*. We obtained similar results in which all the strains analysed were susceptible to ofloxacin and ciprofloxacin. However, 26% and 20% of the strains of *A. caviae* and *A. hydrophila* were resistant to nalidixic acid and pipemidic acid, whereas the resistance was higher in *A. veronii* strains, with 88% ($P < 0.05$ for the resistance to nalidixic acid of *A. veronii* compared with the other species) of the strains being resistant to nalidixic acid and pipemidic acid. Resistance in environment-isolated *Aeromonas* was found in 59% of strains analysed.[6]

In summary, although fluoroquinolones have been reported as the first choice treatment for *Aeromonas* infections, microorganisms resistant to nalidixic acid and susceptible to ciprofloxacin are known to already have a

Table. Susceptibility of *Aeromonas* spp. to 24 antimicrobial agents

Antimicrobial agent	% susceptibility			Breakpoints[a]	
	A. veronii ($n = 14$)	*A. caviae* ($n = 19$)	*A. hydrophila* ($n = 10$)	S	R
Ampicillin	0	0	0	⩽8	⩾32
Co-amoxiclav	43	84	40	⩽8/4	⩾32/16
Piperacillin	93	99	100	⩽16	⩾128
Piperacillin + tazobactam	93	95	100	⩽16/4	⩾128/4
Ticarcillin	14	74	20	⩽16	⩾128
Cefazolin	79	53	40	⩽8	⩾32
Cefuroxime	100	100	100	⩽8	⩾32
Cefotaxime	100	100	100	⩽8	⩾32
Ceftazidime	100	100	100	⩽8	⩾32
Cefixime	100	100	100	⩽8	⩾32
Cefepime	100	100	100	⩽8	⩾32
Aztreonam	100	100	100	⩽8	⩾32
Imipenem	100	100	100	⩽4	⩾16
Gentamicin	93	100	100	⩽4	⩾16
Tobramycin	72	90	90	⩽4	⩾16
Amikacin	100	100	100	⩽16	⩾64
Nalidixic acid	22	74	80	⩽16	⩾32
Pipemidic acid	22	74	80	⩽4	⩾16
Ofloxacin	100	100	100	⩽2	⩾8
Ciprofloxacin	100	100	100	⩽1	⩾4
Fosfomycin	100	74	100	⩽64	⩾256
Nitrofurantoin	100	90	90	⩽32	⩾128
Trimethoprim/sulfamethoxazole	72	79	90	⩽2/8	⩾4/76

[a]NCCLS (2001).[3]

mutation in the *gyrA* gene, and can easily develop a second mutation of resistance, generating a MIC of ciprofloxacin above the breakpoint.[7] Therefore, fluoroquinolones should not be recommended for the treatment of infections produced by *Aeromonas* spp. resistant to nalidixic acid.

Acknowledgements

We would like to thank Drs R. Bartolome (Hospital Valle Hebrón, Barcelona) F. Soriano (Fundación Jiménez Diaz, Madrid), J. Reina (Hospital Son Dureta, Palma de Mallorca), and I. Pujol and F. Ballester (Hospital Universitari Sant Joan, Reus) for their collaboration. This work was supported by grants FIS 99/0944 and FIS 96/0579 from the Spanish Ministry of Health and from CIRIT (SGR 99/00103).

References

1. Janda, J. M., Abbott, S. L. & Morris, J. G., Jr (1995). *Aeromonas*, *Plesiomonas*, and *Edwardsiella*. In *Infections of the Gastrointestinal Tract*, (Blaser, M. J., Smith, P. D., Ravdin, J. I., Greenberg, H. B. & Guerrant, R. L., Eds), pp. 905–17. Raven Press, New York, NY.

2. Figueras, M. J., Soler, L., Chacón, M. R., Guarro, J. & Martínez-Murcia, A. J. (2000). Extended method for discrimination of *Aeromonas* spp. by 16S rDNA-RFLP analysis. *International Journal of Systematic Evolution and Microbiology* **50**, 2069–73.

3. National Committee for Clinical Laboratory Standards. (2001). *Performance Standards for Antimicrobial Susceptibility Testing. Eleventh Informational Supplement M100-S11*. NCCLS, Wayne, PA.

4. Overman, T. L. & Janda, J. M. (1999). Antimicrobial susceptibility patterns of *Aeromonas jandai*, *A. shubertii*, *A. trota*, and *A. veronii* biotype veronii. *Journal of Clinical Microbiology* **37**, 706–8.

5. Ko, W. C., Yu, K. W., Liu, C. Y., Huang, C. T., Leu, H. S. & Chuang, Y. C. (1996). Increasing antibiotic resistance in clinical isolates of *Aeromonas* strains in Taiwan. *Antimicrobial Agents and Chemotherapy* **40**, 1260–2.

6. Goñi-Urriza, M., Pineau, L., Capdepuy, M., Roques, C., Caumette, P. & Quentin, C. (2000). Antimicrobial resistance of mesophilic *Aeromonas* spp. isolated from two European rivers. *Journal of Antimicrobial Chemotherapy* **46**, 297–301.

7. Ruiz, J., Gómez, J., Navia, M. M., Ribera, A., Sierra, J. M., Marco, F. *et al.* (2002). High prevalence of nalidixic acid resistant, ciprofloxacin susceptible phenotype among clinical isolates of *Escherichia coli* and other Enterobacteriaceae. *Diagnostic Microbiology and Infectious Disease*, in press.