J Antimicrob Chemother 2021; **76**: 653–658
doi:10.1093/jac/dkaa499 Advance Access publication 1 December 2020

Journal of Antimicrobial Chemotherapy

# Susceptibility of *Elizabethkingia* spp. to commonly tested and novel antibiotics and concordance between broth microdilution and automated testing methods

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*Received 5 May 2020; accepted 27 October 2020*

**Objectives:** We aimed to determine susceptibilities of *Elizabethkingia* spp. to 25 commonly tested and 8 novel antibiotics, and to compare the performance of different susceptibility testing methods.

**Methods:** Clinical isolates of *Elizabethkingia* spp., *Chryseobacterium* spp. and *Flavobacterium* spp. collected during 2002–18 (*n* = 210) in a nationwide surveillance programme in Taiwan were speciated by 16S rRNA sequencing. MICs were determined by broth microdilution. The broth microdilution results of 18 common antibiotics were compared with those obtained by the VITEK 2 automated system.

**Results:** Among the *Elizabethkingia* spp. identified (*n* = 108), *Elizabethkingia anophelis* was the most prevalent (*n* = 90), followed by *Elizabethkingia meningoseptica* (*n* = 7) and *Elizabethkingia miricola* cluster [*E. miricola* (*n* = 6), *Elizabethkingia bruuniana* (*n* = 3) and *Elizabethkingia ursingii* (*n* = 2)]. Most isolates were recovered from respiratory or blood specimens from hospitalized, elderly patients. PFGE showed two major and several minor *E. anophelis* clones. All isolates were resistant to nearly all the tested β-lactams. Doxycycline, minocycline and trimethoprim/sulfamethoxazole inhibited >90% of *Elizabethkingia* spp. Rifampin inhibited *E. meningoseptica* (100%) and *E. anophelis* (81.1%). Fluoroquinolones and tigecycline were active against *E. meningoseptica* and *E. miricola* cluster isolates. Novel antibiotics, including imipenem/relebactam, meropenem/vaborbactam, ceftazidime/avibactam, cefepime/zidebactam, delafloxacin, eravacycline and omadacycline were ineffective but lascufloxacin inhibited half of *Elizabethkingia* spp. The very major discrepancy rates of VITEK 2 were >1.5% for ciprofloxacin, moxifloxacin and vancomycin. Major discrepancy rates were >3% for amikacin, tigecycline, piperacillin/tazobactam and trimethoprim/sulfamethoxazole.

**Conclusions:** MDR, absence of standard interpretation criteria and poor intermethod concordance necessitate working guidelines to facilitate future research of emerging *Elizabethkingia* spp.

## Introduction

*Elizabethkingia* spp. are aerobic, non-motile, non-spore-forming Gram-negative bacilli that do not ferment glucose.[1] In addition to their natural reservoirs such as soil and water, *Elizabethkingia* spp. have also been recovered from hospital environments. At least six species have been classified in the genus since its designation as a novel taxon in 2005.[2] Three of the species are emerging opportunistic pathogens that cause serious infections, particularly in immunocompromised patients. *Elizabethkingia meningoseptica*, previously known as *Chryseobacterium meningosepticum*, is a well-known aetiological agent of nosocomial pneumonia, meningitis and sepsis.[3] *Elizabethkingia miricola* and *Elizabethkingia anophelis* were recently proposed in 2003 and 2011, respectively.[4,5]

While *E. miricola* causes sporadic cases, *E. anophelis* has caused moderate to large-scale nosocomial outbreaks.[6–14] The reported incidence of *Elizabethkingia* spp. infections is increasing in Asian countries, including Taiwan.[7–11] However, the unreliability of phenotypic methods to differentiate *Chryseobacterium* from *Elizabethkingia* and to speciate *Elizabethkingia* spp. isolates may confound epidemiological studies.[15] In addition, the numbers of longitudinal and nationwide surveillance studies are limited.

*Elizabethkingia* spp. exhibit high-level MDR.[6–14] A variety of antibiotics have been tested *in vitro*. However, antibiotic susceptibilities

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obtained in different studies may not be comparable due to the use of different strains, testing methods and interpretative criteria. Broth microdilution methods were used in many studies as the gold standard to determine MICs of *Elizabethkingia* spp.[7,10,11] However, automated susceptibility testing systems are much more commonly used in clinical laboratories and discrepancies between broth microdilution and automated systems have been reported.[16]

The present study was conducted to investigate the epidemiology, clinical characteristics and antibiotic susceptibility profiles of *Elizabethkingia* isolates in Taiwan using 16S rRNA sequencing of isolates collected during 2002–18 by the Taiwan Surveillance of Antimicrobial Resistance (TSAR) programme. We assessed the antimicrobial susceptibilities of *Elizabethkingia* spp. to 25 antibiotics evaluated in previous studies[7–11] and used in clinical practice, and to 8 novel antibiotics that are either in clinical development or have been recently approved by the USA or EU.[17] Because of reported discrepancies between broth microdilution and automated systems,[16] we used both methods and compared results for 18 of the 25 commonly tested antibiotics.

## Materials and methods

### Bacterial isolates

The TSAR programme is a nationwide surveillance system that collects clinical isolates biennially from 11 medical centres and 15 regional hospitals in all four regions of Taiwan. The collection protocol has been described previously.[18] All isolates from participating hospitals are stored at −80°C and subcultured to ensure purity prior to subsequent testing. The study period was from 2002 to 2018 (corresponding to TSAR periods III to XI). Isolates identified as *Chryseobacterium* spp., *Elizabethkingia* spp. or *Flavobacterium* spp. by participating hospitals were selected and subjected to speciation.

### 16s rRNA gene sequencing

The species of all isolates in the present study were identified by 16S rRNA gene sequencing using Oxford Nanopore Technologies (ONT) MinION sequencing, modified from that published by Liou *et al.*[19] The 16S rRNA gene was amplified by PCR with 12 sets of barcodes attached to the universal primers (8F: AGAGTTTGATCCTGGCTCAG; 1492R: GGTTACCTTGTT ACGACTT). The 12 unique barcoded DNAs were purified and pooled for the second barcode ligation. The pooled DNAs were repaired and dA-tailed using NEBNext FFPE Repair Mix (New England BioLabs, NEB; M6630) and NEBNext Ultra II End Repair/dA-Tailing Module (NEB; E7546). The ONT Native Barcoding Kit (EXP-NBD103) was used to multiplex the eight pools of PCR-barcoded DNAs. A total of 96 (12 × 8) dual-barcoded DNAs were purified and pooled in equimolar amounts for downstream library construction using a 1D Ligation Sequencing Kit (ONT, SQK-LSK109). The sequencing adapter was ligated to the DNAs using NEBNext Quick Ligation Module (NEB; E6056). The library was loaded into a SpotON flowcell R9.4.1 (FLO-MIN106D) and sequencing was executed via MinKNOW (release v 19.05.0). Data processing was performed following the workflow published by Liou *et al.*[19] with basecalling and read-polishing applications changed to Guppy (v 3.0.3) and Medaka (v 0.7.0), respectively. These data were compared with those deposited in NCBI for species identification. We compared the results of ONT MinION sequencing of 16S rRNA areas with Sanger sequencing for five *E. anophelis*, five *E. meningoseptica* and five *E. miricola* cluster isolates. The overall accuracy was 99.87%. The number of discordant nucleotides ranged from 0 to 4 among the 1478 bp compared and the discordant nucleotides were not located in the area for speciation.

### Rapid identification by PCR

Two-step PCR reactions were performed for speciation (See Figure S1 and Table S1, available as Supplementary data at *JAC* Online). The first multiple-PCR step used two sets of primers targeting 23S rRNA to identify *E. anophelis* and *E. meningoseptica*. The amplification procedure consisted of initial denaturation at 95°C for 5 min, followed by 30 cycles of denaturation at 95°C for 30 s, annealing at 53°C for 30 s and elongation at 72°C for 1 min 10 s, then a final cycle of elongation at 72°C for 5 min. The second set of PCR primers was designed based on the findings of the previous study showing that *pheT* could be used for speciation.[20] For isolates other than *E. anophelis* and *E. meningoseptica* by the first PCR reaction, the second PCR was performed to identify *E. miricola* cluster isolates. Amplification conditions were the same as those used in the first PCR step except the annealing step was done at 50°C. A previously reported PCR assay by Chew *et al.*[7] was also tested in parallel to test the accuracy of our PCR typing scheme.

### Determination of clonality by PFGE

PFGE was performed on all *Elizabethkingia* spp. isolates after digestion with ApaI.[21] All electrophoresis runs were performed on 1% agarose gels in 0.5× Tris-borate-EDTA buffer at 14°C, in a Bio-Rad CHEF Mapper XA system operating with initial and final switch times of 5 and 35 s, respectively, for 22 h. Stained gels were photographed and analysed using BioNumerics software (v 5.1; Applied Maths, Saint-Martens-Latem, Belgium). ATCC® BAA-664™ was used as a standard for DNA pattern normalization. Dendrograms were generated to determine the relatedness of isolates. Isolates having >80% similarity were assigned a PFGE cluster name (pulsotype) if there were three or more isolates within the cluster.

### Antimicrobial susceptibility to commonly tested antibiotics

MICs of 25 common antimicrobial agents were determined by reference broth microdilution testing following the guidelines of CLSI.[22] In addition, the MICs of 18 of the 25 antimicrobial agents were determined by an automated system (VITEK 2). Broth microdilution testing was performed using the Sensititre GNX3F (Trek Diagnostics, West Sussex, England), except moxifloxacin, tetracycline, rifampin and vancomycin, for which in-house-prepared 96-well microtitre plates were used. The following quality control strains were included: *Escherichia coli* ATCC 25922 (rifampin, tetracycline and GNX3F), *Pseudomonas aeruginosa* ATCC 27853 (moxifloxacin, tetracycline and GNX3F), *Staphylococcus aureus* ATCC 29213 (vancomycin and GNX3F) and *Enterococcus faecalis* ATCC 29212 (moxifloxacin, rifampin and vancomycin).

For automated testing, VITEK 2 was used with AST-P605 and AST-N322 cards (bioMérieux, France). The quality control strains were *S. aureus* ATCC 29213 and *E. faecalis* ATCC 29212, as well as *E. coli* ATCC 25922 and *P. aeruginosa* ATCC 27853.

### Antimicrobial susceptibility to novel antibiotics

Novel antibiotics included imipenem/relebactam, meropenem/vaborbactam, ceftazidime/avibactam, cefepime/zidebactam, lascufloxacin, delafloxacin, eravacycline and omadacycline. Relebactam, vaborbactam, avibactam, eravacycline and omadacycline were obtained from MedChemExpress (USA), zidebactam and lascufloxacin were from MedKoo Biosciences (USA) and delafloxacin was from Sigma–Aldrich (USA). Broth microdilution testing was performed using in-house-prepared 96-well microtitre plates. *E. coli* ATCC 25922 was used as the quality control strain for eravacycline, lascufloxacin and omadacycline; *P. aeruginosa* ATCC 27853 was used for imipenem/relebactam, meropenem/vaborbactam, ceftazidime/avibactam, cefepime/zidebactam and delafloxacin.

### Data analysis

Susceptibilities were calculated using Whonet software (Stelling and O'Brien).[18] Due to the lack of specific interpretive criteria for *Elizabethkingia* spp., especially for novel antibiotics, CLSI or US FDA breakpoints for other species were adapted and specified according to previous protocols (Tables S2 and S3).[8,11] VITEK 2 results were compared with those of reference broth microdilution. Very major discrepancy (VMD) was defined as resistance in broth microdilution but susceptibility in VITEK 2; major discrepancy (MD) was defined as susceptibility in broth microdilution but resistance in VITEK 2. MD rates ≤3.0% and VMD rates ≤1.5% are considered as the minimum performance standard.

### Ethics

The TSAR bacterial isolates were recovered from clinical samples taken as part of standard care and the study was approved by the Research Ethics Committee of the National Health Research Institutes (EC1010602-E, EC1030406-E, EC1050606-E).

## Results and discussion

### Epidemiology

Among 210 isolates previously identified as *Elizabethkingia* spp., *Chryseobacterium* spp. or *Flavobacterium* spp. by hospital clinical laboratories during 2002–18, 16S rRNA sequencing showed that 108 were *Elizabethkingia* spp. and 90 were *Chryseobacterium* spp. The other 12 isolates were *Candidatus* spp., *Klebsiella aerogenes, Microbacterium arborescens, Pedobacter* spp., *Pseudomonas plecoglossicida, Pseudomonas putida, Rheinheimera* spp., *Sphingomonas* spp. and *Stenotrophomonas maltophilia*. The number of *Elizabethkingia* spp. isolates increased each year (Figure S2). Among the 108 *Elizabethkingia* spp. isolates, *E. anophelis* (n = 90) was the most common, followed by *E. meningoseptica* (n = 7). *E. miricola* (n = 6), *Elizabethkingia bruuniana* (n = 3) and *Elizabethkingia ursingii* (n = 2) isolates were grouped as '*E. miricola* cluster', according to previous studies.[11,20,23] *Elizabethkingia occulta*, the other member of the cluster, was not identified in this study.

Patient characteristics are listed in Table 1. Most isolates were recovered from respiratory or blood specimens in elderly (≥65-year-old) patients. The specimens were from patients not only in ICUs but also in wards and outpatient departments of regional hospitals and medical centres in different regions of Taiwan. Previous studies implied that clinical manifestations of *E. anophelis* infections may differ from those caused by other *Elizabethkingia* spp. For example, *Elizabethkingia* spp. other than *E. anophelis* are common colonizers of cystic fibrosis patients and are less pathogenic.[20,23] However, *E. meningoseptica* and *E. miricola* cluster isolates from blood cultures and from ICU patients were not uncommon in our study. Since the clinical outcomes were not collected in our study, we are not able to provide further clinical implications of different species.

**Table 1.** Patient characteristics in cases of *Elizabethkingia* spp. infection

| | *E. anophelis* | *E. meningoseptica* | *E. miricola* cluster[b] |
|---|---|---|---|
| Total (n) | 90 | 7 | 11 |
| Age, years, n (%)[a] | | | |
| <18 | 1 (1.1) | 0 (0) | 0 (0) |
| 18–64 | 20 (22.2) | 1 (14.3) | 2 (18.2) |
| ≥65 | 65 (72.2) | 6 (85.7) | 9 (81.8) |
| Hospital type, n (%) | | | |
| Medical centres | 63 (70) | 4 (57.1) | 5 (45.5) |
| Regional hospitals | 27 (30) | 3 (42.9) | 6 (54.5) |
| Region of hospitals, n (%) | | | |
| North | 42 (46.7) | 3 (42.9) | 7 (63.6) |
| Central | 22 (24.4) | 2 (28.6) | 1 (9.1) |
| South | 20 (22.2) | 2 (28.6) | 3 (27.3) |
| East | 6 (6.7) | 0 (0) | 0 (0) |
| Patient location, n (%)[a] | | | |
| ICU | 44 (48.9) | 4 (57.1) | 8 (72.7) |
| Non-ICU ward | 39 (43.3) | 1 (14.3) | 1 (9.1) |
| OPD/ER | 4 (4.4) | 0 (0) | 2 (18.2) |
| Specimen type, n (%) | | | |
| Respiratory | 46 (51.1) | 3 (42.9) | 3 (27.3) |
| Blood | 41 (45.6) | 3 (42.9) | 8 (72.7) |
| Pus/discharge | 1 (1.1) | 0 (0) | 0 (0) |
| Urine | 1 (1.1) | 0 (0) | 0 (0) |
| Other | 1 (1.1) | 1 (14.3) | 0 (0) |

OPD, outpatient department; ER, emergency room.
[a]Not all age and hospital location data were available.
[b]*E. miricola* cluster comprised *E. miricola* (6), *E. bruuniana* (3) and *E. ursingii* (2); no *E. occulta* isolates were identified.

PFGE revealed one major *E. anophelis* cluster (>80% in similarity) in multiple hospitals and several minor clusters (Figure S3). The major cluster grouped into two subclusters, A1 and A2; the isolates of A1 and A2 were from multiple hospitals, indicating possible outbreaks. Other *Elizabethkingia* spp. were identified in sporadic cases unrelated to the *E. anophelis* cluster. Previous reports showed the ability of *E. anophelis* to cause outbreaks, in contrast to other *Elizabethkingia* spp.[12,13] Outbreaks of *E. meningoseptica* reported in previous studies may be attributable to *E. anophelis* due to the predominance of this species in the clinical settings and the lack of knowledge of this new species at the time of study.[24]

### Rapid identification by PCR

The 210 isolates described above were used to test the accuracies of our PCR scheme and that of Chew *et al.*[7] Using 16S rRNA sequencing as the reference gold standard, our first PCR reaction and the PCR assay by Chew *et al.*[7] both accurately differentiated all *E. anophelis* from *E. meningoseptica*. Additionally, our second PCR step was 100% accurate in identifying *E. miricola* cluster isolates. Ten isolates of *E. coli, K. pneumoniae, P. aeruginosa and Acinetobacter baumannii* randomly selected from the TSAR collection all tested negative in our PCR scheme (data not shown).

A recent study found that MALDI-TOF MS accurately identified all *E. anopheles, E. meningoseptica* and *E. miricola* when an amended database was used.[11] Since MALDI-TOF and 16S rRNA sequencing are not readily available in all clinical laboratories, the PCR scheme could be a rapid and simple alternative method to accurately differentiate these species. Our PCR scheme demonstrated good accuracy in the identification of *E. anophelis, E. meningoseptica* and *E. miricola* cluster isolates. In addition, one-tube testing for *E. anophelis* and *E. meningoseptica* and additional identification of *E. miricola* cluster isolates may be more practical than other PCR schemes.

### Susceptibility to 25 commonly tested and 8 novel antibiotics

Drug susceptibilities of the *Elizabethkingia* spp. isolates and MIC ranges for all tested antibiotics are shown in Figures 1 and 2 and Table S4. The commonly tested antibiotics for which <5% of all *Elizabethkingia* spp. were susceptible are shown in Table S4. All 108 isolates were resistant to all β-lactams, including carbapenems, due to intrinsic MBL genes $bla_B$ and $bla_{GOB}$.[10] Vancomycin, an agent with discordant *in vitro* testing results and varied treatment outcomes for *E. meningoseptica* reported in the literature,[25] was inactive against our *Elizabethkingia* spp. isolates (MIC ≥ 16 mg/L). Doxycycline, minocycline and trimethoprim/sulfamethoxazole inhibited >90% of all isolates. Previous studies showed consistently high rates of susceptibility to doxycycline and minocycline.[3,7–9,11] However, susceptibility to trimethoprim/sulfamethoxazole varied among these studies,[3,7–9,11] which may be attributed to differences in strains, methodology, definition of 80% reduction in growth[22] and interpretive criteria. In contrast, ciprofloxacin, levofloxacin, moxifloxacin and tigecycline were

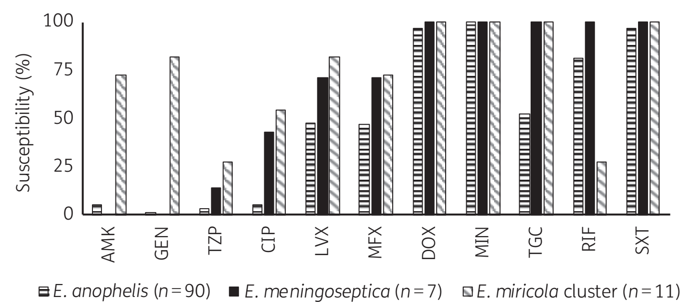


**Figure 1.** Susceptibility of *Elizabethkingia* spp. to commonly tested antibiotics. In the absence of CLSI breakpoints, susceptibility criteria were adapted from previous studies and are listed in Table S2. The commonly tested antibiotics for which <5% of all *Elizabethkingia* spp. were susceptible are not shown in Figure 1; refer to Table S4 for their susceptibility. They included cefepime, cefotaxime, ceftazidime, doripenem, imipenem, meropenem, ampicillin/sulbactam, ticarcillin/clavulanic acid, vancomycin, colistin, polymyxin B, tobramycin, tetracycline and aztreonam. AMK, amikacin; GEN, gentamicin; TZP, piperacillin/tazobactam; CIP, ciprofloxacin; LVX, levofloxacin; MFX, moxifloxacin; DOX, doxycycline; MIN, minocycline; TGC, tigecycline; RIF, rifampin; SXT, trimethoprim/sulfamethoxazole.

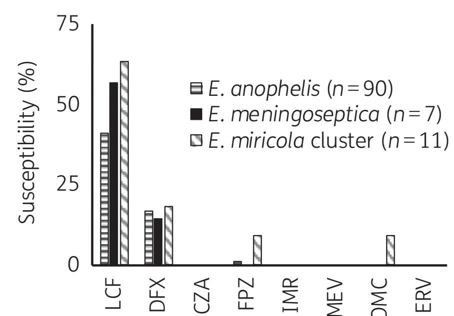


**Figure 2.** Susceptibility of *Elizabethkingia* spp. to novel antibiotics. Due to the lack of CLSI breakpoints, susceptibility criteria were adapted from previous studies and are listed in Table S3. LCF, lascufloxacin; DFX, delafloxacin; CZA, ceftazidime/avibactam; FPZ, cefepime/zidebactam; IMR, imipenem/relebactam; MEV, meropenem/vaborbactam; OMC, omadacycline; ERV, eravacycline.

more active against *E. meningoseptica*/*E. miricola* cluster isolates, with respective susceptibilities of 42.9%/54.5%, 71.4%/81.8%, 71.4%/72.7% and 100%/100%, compared with 5.6%, 47.8%, 46.7% and 52.2% for *E. anophelis*. *E. miricola* cluster isolates were more susceptible to aminoglycosides, including amikacin and gentamicin, but not tobramycin. Rifampin inhibited 81.1% of *E. anophelis* and 100% of *E. meningoseptica*.

Our *Elizabethkingia* spp. isolates were also highly resistant to seven of the eight novel antibiotics tested, which further underscores the clinical challenges posed by *Elizabethkingia* spp. The inability of new β-lactamase inhibitors to enhance the activity of β-lactams was not unexpected because these inhibitors are known to have low activity against MBLs,[24] which are intrinsically present in *Elizabethkingia* spp. Lascufloxacin inhibited 41.1%, 57.1% and 63.6% of *E. anophelis, E. meningoseptica* and *E. miricola*, respectively. However, its activity was not better than that of levofloxacin,

**Table 2.** Discrepancy rates of VITEK 2 compared with broth microdilution for *Elizabethkingia* spp.[a,b]

| Agent | Total (n = 108) | | *E. anophelis* (n = 90) | | *E. meningoseptica* (n = 7) | | *E. miricola* cluster (n = 11)[c] | |
|---|---|---|---|---|---|---|---|---|
| | MD | VMD | MD | VMD | MD | VMD | MD | VMD |
| Amikacin | 12.1 | 0 | 5.6 | 0 | 0 | 0 | 72.7 | 0 |
| Ampicillin/sulbactam | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Cefepime | 0.9 | 0 | 1.1 | 0 | 0 | 0 | 0 | 0 |
| Cefotaxime | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Ceftazidime | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Ciprofloxacin | 0 | 8.3 | 0 | 8.9 | 0 | 0 | 0 | 9.1 |
| Colistin | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Gentamicin | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Imipenem | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Levofloxacin | 0 | 0.9 | 0 | 1.1 | 0 | 0 | 0 | 0 |
| Meropenem | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Minocycline | 0.9 | 0 | 0 | 0 | 0 | 0 | 9.1 | 0 |
| Moxifloxacin | 0.9 | 5.6 | 0 | 6.7 | 0 | 0 | 9.1 | 0 |
| Piperacillin/tazobactam | 6.5 | 0 | 3.3 | 0 | 14.3 | 0 | 27.3 | 0 |
| Rifampin | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Tigecycline | 11.1 | 0 | 7.8 | 0 | 42.9 | 0 | 18.2 | 0 |
| Trimethoprim/sulfamethoxazole | 45.4 | 0 | 50 | 0 | 57.1 | 0 | 0 | 0 |
| Vancomycin | 0 | 3.7 | 0 | 1.1 | 0 | 14.3 | 0 | 18.2 |

[a]Data are presented as percentages (%).
[b]MD rates of >3.0% or VMD rates of >1.5% are underlined.
[c]*E. miricola* cluster comprised *E. miricola* (6), *E. bruuniana* (3) and *E. ursingii* (2); no *E. occulta* isolates were identified.

to which *E. anophelis*, *E. meningoseptica* and *E. miricola* exhibited susceptibility rates of 47.8%, 71.4% and 81.8%, respectively.

### *Performance of VITEK 2 for 18 commonly tested antibiotics*

VMD rates were >1.5% for ciprofloxacin, moxifloxacin and vancomycin; the MD rates were >3% for amikacin, piperacillin/tazobactam, tigecycline and trimethoprim/sulfamethoxazole (Table 2). Similar discrepancy rates were also observed within each species with some variations. Vancomycin was inactive against all *Elizabethkingia* spp., but VITEK 2 gave false-positive results of vancomycin susceptibility in 3.7% of isolates. Figure S4 illustrates the poor correlation between the two testing methods for many antibiotics. Whether results obtained by the automated susceptibility testing system could help determine the treatment of *Elizabethkingia* spp. warrants further investigation. Notably, modest susceptibilities to fluoroquinolones, relatively high discrepancy rates and emerging resistance to other fluoroquinolones in *Elizabethkingia* spp.[9,10] indicate that this drug class should be used cautiously.

In conclusion, *Elizabethkingia* spp., especially *E. anophelis*, emerged in multiple healthcare settings in Taiwan, causing both sporadic cases and outbreaks. Isolates were resistant to commonly tested and newly developed antibiotics. The absence of interpretive criteria specific to these organisms and low concordance between testing methods further confound therapeutic decision-making. In view of the increasing threat of *Elizabethkingia* spp. as emerging opportunistic pathogens, working guidelines and consensus statements on MIC testing and interpretation will be essential to guide clinical practice and to facilitate future clinical and basic research.

## Acknowledgements

We express our sincere appreciation to the following hospitals for their participation in the TSAR: Buddhist Tzu Chi General Hospital; Cathay General Hospital; Changhua Christian Hospital; Cheng-Ching Hospital; Chung Shan Medical University Hospital; Da Chien General Hospital; Ditmanson Medical Foundation Chia-Yi Christian Hospital; Far Eastern Memorial Hospital; Hua-Lien Hospital; Jen-Ai Hospital; Kaohsiung Armed Forces General Hospital; Kaohsiung Chang Gung Memorial Hospital of the C.G.M.F.; Kaohsiung Medical University Chung-Ho Memorial Hospital; Kaohsiung Veterans General Hospital; Kuang Tien General Hospital; Lo-Hsu Foundation, Inc. Lotung Poh-Ai Hospital; Mennonite Christian Hospital; Min-Sheng Healthcare; National Cheng Kung University Hospital; Saint Mary's Hospital Luodong; Show Chwan Memorial Hospital; Tungs' Taichung MetroHarbor Hospital; Taichung Veterans General Hospital; Tainan Sin-Lau Hospital, the Presbyterian Church in Taiwan; Taipei City Hospital Heping Fuyou Branch; Taipei City Hospital Zhongxiao Branch; Taipei Veterans General Hospital; and Tri-Service General Hospital.

We also thank Shu-Man Shih, Institute of Population Health Sciences, National Health Research Institutes for drawing Figure S4.

## Funding

This project was supported by an intramural grant from the National Health Research Institutes (IV-108-PP-09, IV-107-PP-09, IV-107-SP-01) and Ministry of Science and Technology (107-2320-B-400-010-MY3 and 109-2321-B-415-004-).

## Transparency declarations

None to declare.

## Supplementary data

Tables S1 and S2 are available as Supplementary data at *JAC* Online.

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