Elizabethkingia brunniana Infections in Humans, Taiwan, 2005–2017

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Using 16S rRNA and rpoB gene sequencing, we identified 6 patients infected with Elizabethkingia brunniana treated at E-Da Hospital (Kaohsiung, Taiwan) during 2005–2017. We describe patient characteristics and the molecular characteristics of the E. brunniana isolates, including their MICs. Larger-scale studies are needed for more robust characterization of this pathogen.

The Elizabethkingia genus comprises gram-negative, aerobic, nonmotile, nonspore-forming, nonfermenting rod-shaped bacteria (1). This genus previously comprised E. meningoseptica, E. miricola, and E. anophelis. In August 2017, Nicholson et al. proposed adding 3 new species, namely E. brunniana, E. ursingii, and E. occulta, to this genus (1). However, little information exists about these species. In this study, we report the clinical characteristics and demographics of a group of patients with E. brunniana infection in Taiwan and the molecular features of their E. brunniana isolates.

We conducted this study at E-Da Hospital, a 1,000-bed university-affiliated medical center in Kaohsiung, Taiwan; this study was approved by the institutional review board of the hospital (no. EMRP-106-105). We searched the hospital database to identify microbial cultures performed during January 2005–December 2017 that yielded Elizabethkingia. The isolates were initially identified by staff in the clinical microbiology laboratory using API/ID32 phenotyping kits or VITEK MS (both from bioMérieux, https://www.biomerieux.com). We reidentified these species as Elizabethkingia using both 16S rRNA and rpoB gene sequencing. The primers and methods we used for amplification and sequencing of the 16S rRNA and rpoB genes were described previously (1,2).

We compared the assembled 16S rRNA gene sequences with the nucleotide sequences of Elizabethkingia-type strains present in GenBank. We considered isolates with ≥99.5% similarity in the 16S rRNA gene sequence members of the same species, as recommended in a previous study (3). We constructed a phylogenetic tree using the rpoB genes of the isolates exhibiting ≥99.5% 16S rRNA gene sequence identity with the E. brunniana type strain G0146. We calculated the average nucleotide identity using OrthoANI (4) and computed in silico DNA–DNA hybridization (DDH) using the Genome-to-Genome Distance Calculator (5), using the average nucleotide identity value of ≥95% and the DDH value of ≥70% separately as criteria for species delineation (4,5). We sequenced the quinolone resistance–determining regions of DNA gyrase (gyrA and gyrB) and topoisomerase IV (parC and parE) to look for mutations associated with resistance (Appendix Table, https://wwwnc.cdc.gov/EID/article/25/7/18-0768-App1.pdf).

For the 13-year period, we found 103 nonduplicate Elizabethkingia isolates in the database of the clinical microbiology laboratory. Among these, 8 isolates shared ≥99.5% 16S rRNA gene sequence identity with E. brunniana G0146, and an rpoB gene–based phylogenetic analysis revealed that 6 of the 8 isolates were more closely related to E. brunniana G0146 (Appendix Figure 1). We previously published the complete whole-genome sequence of 1 of these 6 isolates, EM798-26 (GenBank accession no. CP023746) (6). Using 16S rRNA gene sequence analysis, we initially identified this isolate as E. miricola. Average nucleotide identity analysis demonstrated that EM798-26 and E. brunniana G0146 share 97.7% whole-genome similarity (Appendix Figure 2). Using in silico DDH analysis, we predicted a DDH value of 81.7% for EM798-26 and E. brunniana G0146 (Appendix Figure 3). These results support that EM798-26 and the other 5 isolates (EM20-50, EM455-89, EM828-05, EM863-68, and EM891-63) are E. brunniana.

These 6 isolates were collected from 6 (4 male and 2 female) patients (Table) with a mean age of 71.7 (SD ±11) years. The sources of isolation included bronchoalveolar lavage fluid (n = 2), blood (n = 2), urine (n = 1), and the tip of the central venous catheter (n = 1). All infections were healthcare associated. Two patients had septic shock, and all patients had ≥1 concurrent medical condition, such as hypertension, diabetes mellitus, or a malignancy. Antimicrobial therapy included piperacillin/tazobactam, trimethoprim/sulfamethoxazole, levofloxacin, or tigecycline, either singly or in combination. None of the patients died of E. brunniana infection.

Most E. brunniana isolates were resistant to β-lactams, β-lactam and lactamase inhibitors, carbapenems, aminoglycosides, and trimethoprim/sulfamethoxazole (Table). All isolates were susceptible to minocycline, 4 (67%) to tigecycline and levofloxacin, and 2 (33%) to ciprofloxacin. The antimicrobial susceptibility patterns we found are similar to those of other Elizabethkingia spp. identified...
in previous studies (7–10). For example, reports from the United States, Hong Kong, and South Korea have revealed that *E. anophelis* and *E. meningoseptica* were frequently resistant to most β-lactams, including ceftazidime, ceftriaxone, and imipenem, but showed variable susceptibility to piperacillin/tazobactam, cefepime, ciprofloxacin, and levofloxacin (7–10).

To investigate the association between target gene mutations and fluoroquinolone resistance, we examined the mutations present in quinolone resistance–determining regions in these 6 isolates. We did not find nonsynonymous substitutions in the quinolone resistance–determining regions of *gyrA*, *gyrB*, *parC*, and *parE*, which suggests that mutations in these genes are not the cause of fluoroquinolone resistance.

In summary, our study demonstrates the clinical manifestations of *E. bruuniana* infection and the molecular characteristics of the pathogen. Because cases in our study were limited in number, further large-scale studies are necessary to investigate the antimicrobial susceptibility patterns of *E. bruuniana* and elucidate the clinical characteristics and treatment of *E. bruuniana* infection.

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### References


Human Enterovirus C105, China, 2017

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