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Relative Prevalence and Antimicrobial Susceptibility of Clinical Isolates of *Elizabethkingia* Species Based on 16S rRNA Gene Sequencing

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ABSTRACT Some of the previously reported clinical isolates of *Elizabethkingia meningoseptica* may be later named species of *Elizabethkingia*. We determined the accuracy of species identification (with two matrix-assisted laser desorption ionization–time of flight mass spectrometry [MALDI-TOF MS] systems and the Vitek 2 GN card), relative prevalence of three *Elizabethkingia* spp. in clinical specimens, and antimicrobial susceptibility of the species identified by 16S rRNA gene sequencing. Specimens for culture were collected from patients in a university hospital in Seoul, South Korea, between 2009 and 2015. All 3 *Elizabethkingia* spp. were detected in patients; among the 86 isolates identified by 16S rRNA gene sequencing, 17 (19.8%) were *E. meningoseptica*, 18 (20.9%) were *Elizabethkingia miricola*, and 51 (59.3%) were *Elizabethkingia anophelis*. Only the MALDI-TOF Vitek MS system with an amended database correctly identified all of the isolates. The majority (76.7%) of the isolates were from the lower respiratory tract, and 8 (9.3%) were from blood. Over 90% of *E. meningoseptica* and *E. anophelis* isolates were susceptible to piperacillin-tazobactam and rifampin. In contrast, all *E. miricola* isolates were susceptible to fluoroquinolones except ciprofloxacin. Further studies are urgently needed to determine the optimal antimicrobial agents for the treatment of infections due to each individual *Elizabethkingia* species.

KEYWORDS *Elizabethkingia meningoseptica*, *Elizabethkingia miricola*, *Elizabethkingia anophelis*, antimicrobial susceptibility, 16S rRNA gene sequencing

Elizabethkingia species are aerobic, nonmotile, oxidase-positive, indole-positive, Gram-negative bacilli that do not ferment glucose. *Elizabethkingia* spp. can be found frequently in soil, freshwater, salt water, and in hospital environments (1). However, they do not normally exist in the human body. *Elizabethkingia meningoseptica* (formerly *Chryseobacterium meningosepticum*) has been a well-known human pathogen since its first description in a case of neonatal meningitis by Elizabeth O. King in 1959 (2). This organism was reported to cause various invasive infections in immunocompromised hosts and to be associated with nosocomial infections and outbreaks in intensive care units (ICUs) (3–5). It has been considered that the incidence of *E. meningoseptica* bacteremia has increased over the last decade (6). Two new species of *Elizabethkingia*, *Elizabethkingia miricola* and *Elizabethkingia anophelis*, were proposed in 2003 and 2011, respectively (7–9). Therefore, some of the previously reported clinical isolates of *E. meningoseptica* may be later named species of *Elizabethkingia*. The first

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TABLE 1 Comparison of species identified by 16S rRNA gene sequencing with those by the two MALDI-TOF systems and the Vitek 2 GN card system

16S rRNA gene sequencing (no. of isolates)	MALDI-TOF Vitek MS[a] (no. of isolates)	MALDI-TOF Bruker Biotyper (no. of isolates)	Vitek 2 with GN card (no. of isolates)
E. meningoseptica (17)	*E. meningoseptica* (17)	*E. meningoseptica* (16)	*E. meningoseptica* (16)
		Chryseobacterium indologenes (1)	*C. indologenes* (1)
E. miricola (18)	*E. miricola* (18)	*E. miricola* (17)	*E. meningoseptica* (16)
		C. indologenes (1)	*C. indologenes* (2)
E. anophelis (51)	*E. anophelis* (51)	*E. meningoseptica* (49)	*E. meningoseptica* (48)
			C. indologenes (1)
		E. meningoseptica/ E. miricola (1)	*E. meningoseptica* (2)
		E. miricola (1)	

[a]Identification was based on a SARAMIS database amended with *Elizabethkingia* spp. spectra provided to bioMérieux.

case of *E. miricola* sepsis was reported in 2008 (10), and a case of *E. anophelis* neonatal meningitis was reported in 2013 (11).

E. meningoseptica isolates are often resistant (R) to multiple β-lactam antibiotics due to intrinsic class A extended-spectrum β-lactamases (ESBLs) and inherent class B metallo-β-lactamases (MBLs) (12). The antimicrobial susceptibility of *Elizabethkingia* may vary depending on the species. However, there are scanty data on the susceptibility of the new species. The aims of this study were to determine the accuracy of species identification systems and the relative prevalence of three *Elizabethkingia* spp. in clinical specimens and to compare the antimicrobial susceptibility of the species identified by 16S rRNA gene sequencing.

RESULTS

***Elizabethkingia* spp. identified.** Among the total 86 *Elizabethkingia* isolates, the species identified using 16S rRNA gene sequencing were 17 isolates (19.8%) of *E. meningoseptica* (99.5% to 99.9% nucleotide identity to *E. meningoseptica* type strain ATCC 13253), 18 isolates (20.9%) of *E. miricola* (98.9% to 99.8% nucleotide identity to *E. miricola* type strain GTC862), and 51 isolates (59.3%) of *E. anophelis* (99.1% to 100.0% nucleotide identity to *E. anophelis* type strain FMS-007).

The matrix-assisted laser desorption ionization–time of flight (MALDI-TOF) Vitek MS system with an amended database correctly identified all of the 17, 18, and 51 isolates of *E. meningoseptica*, *E. miricola*, and *E. anophelis*, respectively. However, the Bruker Biotyper correctly identified 16 of 17 *E. meningoseptica* isolates and 17 of 18 *E. miricola* isolates but none of the *E. anophelis* isolates (Table 1). The Vitek 2 GN card system correctly identified 16 of 17 *E. meningoseptica* isolates but none of the other species.

Among the 86 isolates of *Elizabethkingia* spp., 66 (76.7%) were recovered from the lower respiratory tract, 8 (9.3%) from blood, 7 (8.1%) from urine, and 5 (5.8%) from other specimens (Table 2). Among the 8 isolates from blood, 2 isolates were identified as *E. miricola* and 6 isolates were identified as *E. anophelis*. There were no *E. meningoseptica* isolates from blood. Careful clinical evaluation suggested that the positive blood

TABLE 2 Source of detection of 86 isolates of *Elizabethkingia* spp. at a tertiary care hospital from 2009 to 2015

	No. (%) of isolates from:			
Species (no. of isolates)	Lower respiratory	Blood	Urine	Other[a]
E. meningoseptica (17)	14	0	3	0
E. miricola (18)	12	2	0	4
E. anophelis (51)	40	6	4	1
Total (%)	66 (76.7)	8 (9.3)	7 (8.1)	5 (5.8)

[a]Other sources includes eye, neck, head, pleural fluid, and external ear fluid.

cultures in 4 of the 8 patients were not significant. However, they may have been transient invaders from central arterial or venous lines or endotracheal tubes. Therefore, no antimicrobial agents were administered for *Elizabethkingia* infection (Table 3). All of the 8 patients had various underlying diseases and had indwelling catheters or endotracheal tubes. Four patients were admitted to the ICU. All of the patients, except for one, were treated with various antimicrobial agents during the 7 days prior to blood culture. Among the four patients, two were cured with tigecycline and trimethoprim-sulfamethoxazole, to which the isolates were susceptible (S). The remaining two patients were cured with trimethoprim-sulfamethoxazole, although the MICs for the isolates were 4 and 76 μg/ml (resistance breakpoints, $\geq$4 and 76 μg/ml), respectively.

In pulsed-field gel electrophoresis (PFGE) analysis, isolates of each *Elizabethkingia* species belonged to 5 to 10 different PFGE groups, while identical pulsotypes were found in 8 of 17 *E. meningoseptica* isolates, 6 of 18 *E. miricola* isolates, and 17 of 51 *E. anophelis* isolates (see Fig. S1 in the supplemental material).

Antimicrobial susceptibilities. The MICs of the antimicrobial agents and the susceptibilities of the isolates are shown in Table 4 (see Data Set S1 in the supplemental material). Among the *E. meningoseptica* isolates, 100% and 94% were susceptible to piperacillin-tazobactam and rifampin, respectively, but only 23% to 41% were susceptible to fluoroquinolones. Unlike *E. meningoseptica*, all *E. miricola* isolates were susceptible to fluoroquinolones, except for ciprofloxacin. Over 90% of *E. meningoseptica* and *E. anophelis* isolates were susceptible to piperacillin-tazobactam and rifampin. Although none of the species were susceptible to vancomycin, all three species exhibited at least 94% intermediate (I) reaction to this agent.

DISCUSSION

The accuracy of species identification was low with the Vitek 2 GN card system. Although our Bruker Biotyper without an amended database failed to identify *E. anophelis* isolates, the addition of a database for *E. anophelis* was recently reported (13). This result indicates that a MALDI-TOF mass spectrometry (MS) system can be a reliable species identification system for the genus *Elizabethkingia*.

Although *E. meningoseptica* infections are well known, *E. miricola* sepsis in a lymphoma patient was reported in the United States after the proposal of new species (10). Several *E. anophelis* infections have been reported from tropical or subtropical regions, the Central African Republic (11), Singapore (14), and Hong Kong (15). A recent study in Hong Kong urged researchers to consider the clinically significant morbidity and mortality of patients with *E. anophelis* bacteremia (13). An *E. anophelis* outbreak in Wisconsin in 2016 resulted in the deaths of at least 18 patients (16).

In our study, which took place in the temperate country of South Korea, all three species of *Elizabethkingia* were detected in patients, and *E. anophelis* was the most prevalent. It is interesting that *E. meningoseptica* was not present while *E. anophelis* was the most common among blood isolates (Table 2). These findings suggest that *E. anophelis* plays a significant role as a human pathogen. In general, the majority of blood isolates are clinically significant (5, 13, 16). However, in our study, only 4 of the 8 patients with positive *Elizabethkingia* blood cultures were clinically significant, although all of the patients had risk factors for infection (Table 3). The majority of the *Elizabethkingia* isolates were detected in lower respiratory tract specimens, but it was difficult to distinguish infection from colonization as reported in other studies (4, 17).

Our PFGE analysis of each species showed that certain pulsotypes were more prevalent than others, suggesting that these types are either more prevalent in the hospital environment or that they have a higher capability to infect or colonize.

E. meningoseptica has been known to be resistant to multiple antimicrobial agents (18, 19). However, as mentioned above, the susceptibility of *E. meningoseptica* in the previous study may include those of the other 2 species. To the best of our knowledge, our study is the first one to compare the susceptibilities of all *Elizabethkingia* species (Table 4). The organisms are typically resistant to β-lactams (15, 18). In a previous study from our group (19), all 31 isolates of *E. meningoseptica* (which may include other *Elizabethkingia* species)

TABLE 3 Case summary of eight patients with *Elizabethkingia* spp. isolation from blood

No.	Age/sex[a]	Initial identification[b]	Final identification[c]	Underlying disease[d]	Indwelling device	Ward[e]	No. positive/total pairs, bottles (culture date)	Infection sign[f]		Previous antibiotic therapy (1 week before culture)	Antimicrobial therapy for *Elizabethkingia* infection	Outcome	
								BT (°C)	WBC (per μl)			Microbiologic eradication[g]	Clinical response[h]
1	79/M	*E. meningoseptica*	*E. miricola*	Bladder cancer, hypoxic brain damage	Endotracheal tube, central venous line	ICU	1/3 (Aug. 8, 2011) 1/1 (Aug. 9, 2011)	38.0	23,990	None	None	NA[i]	NA
2	69/M	*E. meningoseptica*	*E. miricola*	COPD, CRF, cerebral infarction, fungal pneumonia	Endotracheal tube	GW	1/4 (Aug. 26, 2011)	35.9	9,990	Meropenem, teicoplanin, levofloxacin	None	NA	NA
3	69/M	*E. meningoseptica*	*E. anophelis*	Rectal cancer	Endotracheal tube, arterial line, central venous line	ICU	1/3 (Sep. 25, 2010)	38.0	8,320	Imipenem, metronidazole	None	NA	NA
4	72/F	*E. meningoseptica*	*E. anophelis*	Klatskin tumor, DM	Endotracheal tube, arterial line, central venous line	GW	2/3 (Oct. 30, 2010)	36.2	7,420	Tigecycline	Tigecycline, ciprofloxacin	Cured	Failed
5	72/M	*E. meningoseptica*	*E. anophelis*	Alcoholic LC	Endotracheal tube, central venous line	GW	3/3 (Nov. 13, 2012) 2/2 (Nov. 15, 2012)	37.8	2,400	Imipenem, colistin, teicoplanin, minocycline	Trimethoprim-sulfamethoxazole	Cured	Cured
6	67/F	*E. meningoseptica*	*E. anophelis*	Myelofibrosis, splenomegaly, thrombotic endocarditis	Central venous line	GW	4/4 (Nov. 22, 2012) 1/3 (Nov. 23, 2012 1/1 (Nov. 24, 2012) 2/4 (Nov. 26, 2012)	37.5	11,180	Ciprofloxacin, cefazolin	Trimethoprim-sulfamethoxazole	Cured	Cured
7	49/M	*E. meningoseptica*	*E. anophelis*	Mitral valve replacement, valvular heart failure	Endotracheal tube, arterial line	ICU	1/5 (Mar. 20, 2013)	38.6	16,630	Piperacillin-tazobactam, cefepime, teicoplanin	None	NA	NA
8	63/M	*E. meningoseptica*	*E. anophelis*	Pneumonectomy, lung transplantation, RA	Endotracheal tube, chest tube, arterial line, central venous line	ICU	2/3 (Sep. 20, 2013) 2/3 (Sep. 22, 2013) 4/4 (Sep. 24, 2013)	36.4	2,890	Meropenem, colistin	Trimethoprim-sulfamethoxazole, tigecycline	Cured	Cured

[a]M, male; F, female.
[b]Identified by Vitek 2 with GN card for no. 1 to 7 and by MALDI-TOF Bruker Biotyper for No. 8.
[c]Final identification was done by 16S rRNA gene sequencing.
[d]COPD, chronic obstructive pulmonary disease; CRF, chronic renal failure; LC, liver cirrhosis; RA, rheumatic arthritis; DM, diabetes mellitus.
[e]GW, general ward.
[f]BT, body temperature; WBC, white blood cell.
[g]Microbiologic eradication was the absence of the original pathogens detected from blood (7 days after the first positive blood culture).
[h]Clinical response: a favorable clinical response was defined as the resolution of fever (defined as ≥38.0°C), leukocytosis (WBC, ≥11 × 10^6/μl), and hypotension (mean arterial pressure of <65 mm Hg), in addition to no longer requiring support from vasoactive agents. Patients who had persistence or deterioration in clinical parameters or who died were classified as treatment failures.
[i]NA, not applicable. There was no follow up data due to transfer of the patient within 7 days.

TABLE 4 Antimicrobial susceptibilities of *Elizabethkingia* isolates determined by the agar dilution method

Species (no. of isolates) and antimicrobial agents	Breakpoint (μg/ml)[a]		MIC (μg/ml)			Susceptibility (%)		
	S	R	Range	50%	90%	S	I	R
E. meningoseptica (17)								
Piperacillin	≤16	≥128	16–32	16	32	65	35	0
Piperacillin-tazobactam[b]	≤16	≥128	8–16	8	16	100	0	0
Ceftazidime	≤8	≥32	64 to >128	>128	>128	0	0	100
Imipenem	≤4	≥16	16–32	32	32	0	0	100
Ciprofloxacin	≤1	≥4	1 to >64	64	>64	23	6	71
Levofloxacin	≤2	≥8	0.5–128	16	64	35	0	65
Moxifloxacin	≤2	≥8	0.12–64	4	32	41	12	47
Gatifloxacin	≤2	≥8	0.5–128	8	64	35	12	53
Trimethoprim-sulfamethoxazole[b]	≤2	≥4	2–8	4	4	6	0	94
Gentamicin	≤4	≥16	4 to >128	32	64	6	0	94
Vancomycin	≤4	≥32	8–64	8	16	0	94	6
Rifampin	≤1	≥4	0.25–2	0.5	1	94	6	0
E. miricola (18)								
Piperacillin	≤16	≥128	4–32	16	32	83	17	0
Piperacillin-tazobactam	≤16	≥128	4–32	8	16	94	6	0
Ceftazidime	≤8	≥32	64 to ≥128	>128	>128	0	0	100
Imipenem	≤4	≥16	16 to ≥64	64	64	0	0	100
Ciprofloxacin	≤1	≥4	0.5–4	1	4	56	22	22
Levofloxacin	≤2	≥8	0.25–2	0.5	2	100	0	0
Moxifloxacin	≤2	≥8	≤0.06–1	0.25	1	100	0	0
Gatifloxacin	≤2	≥8	0.12–2	0.5	2	100	0	0
Trimethoprim-sulfamethoxazole	≤2	≥4	1–8	4	8	28	0	72
Gentamicin	≤4	≥16	4 to >128	8	>128	45	22	33
Vancomycin	≤4	≥32	8–16	16	16	0	100	0
Rifampin	≤1	≥4	0.25 to >128	1	16	66	17	17
E. anophelis (51)								
Piperacillin	≤16	≥128	8–64	16	32	82	18	0
Piperacillin-tazobactam	≤16	≥128	≤0.12–32	8	8	92	8	0
Ceftazidime	≤8	≥32	64 to >128	>128	>128	0	0	100
Imipenem	≤4	≥16	16 to >64	64	>64	0	0	100
Ciprofloxacin	≤1	≥4	1 to >64	64	>64	22	6	72
Levofloxacin	≤2	≥8	0.5 to >128	32	64	29	6	65
Moxifloxacin	≤2	≥8	0.12–64	4	32	41	10	49
Gatifloxacin	≤2	≥8	0.25–128	8	32	33	12	55
Trimethoprim-sulfamethoxazole	≤2	≥4	2–16	4	8	22	0	78
Gentamicin	≤4	≥16	1 to >128	32	64	22	23	55
Vancomycin	≤4	≥32	8–64	16	16	0	94	6
Rifampin	≤1	≥4	≤0.06–16	1	1	96	2	2

[a]The interpretive criteria applied were those of the CLSI for non-*Enterobacteriaceae*; the criteria for vancomycin and rifampin were those for *Staphylococcus* or *Enterococcus* spp. The criterion of gatifloxacin was that for moxifloxacin.

[b]In the combinations, the concentration of tazobactam was 4 μg/ml constant, and the ratio of trimethoprim to sulfamethoxazole was 1 to 19.

had both bla_{BlaB} and bla_{GOB} genes. The GenBank database shows that chromosomes of *E. anophelis* (accession numbers CP006576 and CP007547) and *E. miricola* (accession number CP011059) possess bla_{BlaB}, bla_{GOB}, and bla_{CME} genes and an AmpC β-lactamase gene. In our study, over 90% of the isolates of 3 *Elizabethkingia* spp. were susceptible to a piperacillin-tazobactam combination. This has also been shown by other studies (5, 20). However, it is necessary to evaluate clinical efficacy, given that all *Elizabethkingia* spp. appear to be inherent MBL producers.

Antimicrobial resistance may vary depending on the species as well as the region and time of bacterial isolation. As mentioned above, limited data are currently available on the susceptibility patterns of *Elizabethkingia* spp. Although three previous studies were performed using relatively significant numbers of isolates, the specimens were from unspecified sources of patients, blood, or hospital environments (5, 21, 22). Furthermore, all three reports stated that the species were *E. meningoseptica*, but the isolates were identified before the proposal of new species or unreliable phenotypic methods were used for identification. However, by comparing our results, based on

identification by 16S rRNA sequencing, to those of other studies, the following generalization can be made: *Elizabethkingia* spp. are nonsusceptible to ceftazidime, imipenem, and vancomycin (high vancomycin susceptibility in a study may be due to the use of a higher breakpoint, 16 μg/ml [21]); the susceptibility rates of *E. miricola* to fluoroquinolones are higher than those of the other species; and the susceptibility of *Elizabethkingia* spp. to other antimicrobial agents are difficult to predict.

Several reports have shown that the incidence of *Elizabethkingia* bacteremia increased and the mortality rate was high (5, 20, 23, 24). Indwelling devices and inappropriate antimicrobial therapy were independent risk factors for poor outcomes with *Elizabethkingia* bacteremia (5, 24, 25). In our study, all 4 bacteremic patients were microbiologically cured with trimethoprim-sulfamethoxazole alone or with a combination of tigecycline plus trimethoprim-sulfamethoxazole or ciprofloxacin. Anecdotal reports have indicated that some cases of *E. meningoseptica* infection respond only to combinations of piperacillin-tazobactam plus rifampin, vancomycin plus rifampin, or a fluoroquinolone plus vancomycin and rifampin (6). In our study, none of the *Elizabethkingia* isolates were susceptible to vancomycin, and the majority were intermediate, indicating similar susceptibility with those of the worldwide collection from 1999 to 2001 (22). Therefore, it seems that vancomycin alone is ineffective in the treatment of *Elizabethkingia* infection.

In conclusion, *E. anophelis* was the most frequently detected species in clinical specimens. Over 90% of 3 *Elizabethkingia* spp. were susceptible to piperacillin-tazobactam. The majority of *E. meningoseptica* and *E. anophelis* isolates were susceptible to rifampin, and all isolates of *E. miricola* only were susceptible to levofloxacin, moxifloxacin, and gatifloxacin. Therefore, further studies are urgently needed to determine the optimal antimicrobial agents for treatment of infections caused by each individual *Elizabethkingia* species.

MATERIALS AND METHODS

Clinical specimens and identification of *Elizabethkingia* spp. Clinical specimens for bacterial culture were collected from patients at a tertiary care university hospital in Seoul, South Korea between January 2009 and February 2015. The species were initially identified using the Vitek 2 GN card system (bioMérieux, Mercy l'Etoile, France). Isolates identified as either *Elizabethkingia* spp. or *Chryseobacterium* spp. were kept frozen until used in this study.

16S rRNA gene sequencing and MALDI-TOF MS analysis. The 16S rRNA gene was amplified and sequenced using the universal primers 8F (5′-AGA GTT TGA TCC TGG CTC AG-3′) and 1541R (5′-AAG GAG GTG ATC CAG CCG CA-3′). The following additional primers were used to analyze the sequence: 310R (5′-AGT ACC AGT GTG GGG GAT CA-3′) and 1170F (5′-CAA ATC ATC ACG GCC CTT AC-3′). The species were identified by comparing the sequences using the EzTaxon server (http://www.ezbiocloud.net/).

All clinical isolates were identified by two MALDI-TOF systems, the Bruker Biotyper (Bruker Daltonics, Bremen, Germany) and the Vitek MS (bioMérieux). There were no reference data for the identification of *E. anophelis* in either system. However, the Vitek MS research use only (RUO) (Saramis) database was amended for our study by providing the spectra data of 20 isolates of three *Elizabethkingia* spp. identified by 16S rRNA gene sequencing to the bioMérieux. These were used to compute species-specific SuperSpectra for automated identification with SARAMIS (details to be published elsewhere). The accuracy of species identification using the MALDI-TOF and Vitek 2 GN card systems was determined by comparing the results of the 16S rRNA gene sequence as a reference.

Pulsed-field gel electrophoresis. Chromosomal DNA of *Elizabethkingia* isolates were digested with XbaI and analyzed for PFGE patterns using the CHEF DR II system (Bio-Rad, Hercules, CA, USA) as described previously (15).

Antimicrobial susceptibility testing. The MICs of the antimicrobial agents were determined using an agar dilution method (26). The antimicrobial agents used were piperacillin and tazobactam (Wyeth, Pearl River, NY, USA); ceftazidime, gentamicin, rifampin, and vancomycin (Sigma Chemical, St. Louis, MO, USA); imipenem (Choongwae, Seoul, South Korea); ciprofloxacin and moxifloxacin (Bayer Korea, Seoul, South Korea); levofloxacin (Daiichi, Tokyo, Japan); gatifloxacin (Bristol-Myers Squibb, Princeton, NJ, USA); and trimethoprim and sulfamethoxazole (Dong Wha, Seoul, South Korea).

The MICs were interpreted based on the Clinical and Laboratory Standards Institute (CLSI) criteria for other non-*Enterobacteriaceae* (27). The breakpoints used for vancomycin (S, ≤4 μg/ml; R, ≥32 μg/ml) and rifampin (S, ≤1 μg/ml; R, ≥4 μg/ml) were those for *Staphylococcus* spp. The moxifloxacin breakpoint was used for gatifloxacin. *Escherichia coli* ATCC 25922, *Pseudomonas aeruginosa* ATCC 27853, and *Staphylococcus aureus* ATCC 29213 were used as controls.

Accession number(s). The GenBank accession numbers of 16S rRNA sequence are as follows: KP836318 and KP836320 for *E. meningoseptica*; KP836321 and KP844567 for *E. miricola*; and KT768343, KT768344, KT768345, KP836317, and KP836319 for *E. anophelis*.

SUPPLEMENTAL MATERIAL

Supplemental material for this article may be found at https://doi.org/10.1128/JCM.01637-16.

DATASET S1, XLSX file, 0.02 MB.

TEXT S1, PDF file, 0.1 MB.

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We declare no conflicts of interest.

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