The draft genomes of *Elizabethkingia anophelis* of equine origin are genetically similar to three isolates from human clinical specimens

William L. Johnson¹, Akhilesh Ramachandran²*, Nathanial J. Torres¹, Ainsley C. Nicholson³, Anne M. Whitney³, Melissa Bell³, Aaron Villarma³, Ben W. Humrighouse³, Mili Sheth⁴, Scot E. Dowd⁵, John R. McQuiston³, John E. Gustafson¹*

¹ Department of Biochemistry and Molecular Biology, Oklahoma State University, Stillwater, Oklahoma, United States of America, ² Oklahoma Animal Disease Diagnostic Laboratory, Center for Veterinary Health Sciences, Oklahoma State University, Stillwater, Oklahoma, United States of America, ³ Special Bacteriology Reference Laboratory, Bacterial Special Pathogens Branch, Division of High-Consequence Pathogens and Pathology, Centers for Disease Control and Prevention, Atlanta, Georgia, United States of America, ⁴ Division of Scientific Resources, Centers for Disease Control and Prevention, Atlanta, Georgia, United States of America, ⁵ Molecular Research DNA Laboratory, Shallowater, Texas, United States of America

* rakhi@okstate.edu (AR); john.gustafson@okstate.edu (JEG)

**Abstract**

We report the isolation and characterization of two *Elizabethkingia anophelis* strains (OSUV-1 and OSUV-2) isolated from sources associated with horses in Oklahoma. Both strains appeared susceptible to fluoroquinolones and demonstrated high MICs to all cell wall active antimicrobials including vancomycin, along with aminoglycosides, fusidic acid, chloramphenicol, and tetracycline. Typical of the *Elizabethkingia*, both draft genomes contained multiple copies of β-lactamase genes as well as genes predicted to function in antimicrobial efflux. Phylogenetic analysis of the draft genomes revealed that OSUV-1 and OSUV-2 differ by only 6 SNPs and are in a clade with 3 strains of *Elizabethkingia anophelis* that were responsible for human infections. These findings therefore raise the possibility that *Elizabethkingia* might have the potential to move between humans and animals in a manner similar to known zoonotic pathogens.

**Introduction**

Organisms from the *Elizabethkingia* genus are ubiquitous and have been isolated from arthropods [1–5], lizards [6], fish [7], frogs [8], corn [9], hospital sinks and water spigots [10, 11], and the Mir space station [12]. Some *Elizabethkingia* spp. are considered opportunistic pathogens that can cause serious infections such as meningitis and bacteremia, primarily in neonates or immunocompromised individuals. In general, *Elizabethkingia* infections are associated with high mortality rates [13, 14], likely due in part to the intrinsic antibiotic resistance phenotype expressed by these organisms, with the majority of isolates showing resistance to broad spectrum β-lactams, tetracyclines, and aminoglycosides, both in vivo and in vitro, while more variability is found in resistance to vancomycin and ciprofloxacin [8, 15–42]. This variability
in vancomycin susceptibility is of interest as there appear to be discrepancies between laboratory reports for a variety of Elizabethkingia strains which were not susceptible to vancomycin in vitro based on CLSI standard antimicrobial susceptibility testing methods [19, 39], and clinical reports suggesting that vancomycin exhibits in vivo therapeutic efficacy [19, 20, 22, 26, 32, 35, 37, 43–45]. Recently, an unprecedented Elizabethkingia anophelis outbreak occurred in Wisconsin, Michigan, and Illinois, with 65 confirmed cases and 20 deaths reported [46, 47]. This outbreak is particularly notable because in addition to the high case count, this outbreak was primarily community-associated rather than healthcare-associated, and to date, no reservoir for this outbreak has been identified. *E. anophelis* is also the etiologic agent of disease in healthcare associated outbreaks that have occurred in Illinois [48], the Central African Republic [49], Hong Kong [14], Taiwan [25], Singapore [36], and other isolated cases [23, 28, 33, 40, 50, 51].

It has been well documented that both food and companion animals may serve as reservoirs for antibiotic-resistant bacterial pathogens [52–60]. The findings of *Elizabethkingia meningoseptica* isolated from a dog suffering from bacteremia [60] and contagious *Elizabethkingia miricola* among farmed frogs [8] suggest that farm and/or companion animals may also act as reservoirs for *Elizabethkingia* with the potential to cause human disease.

We report here the draft genomes and antibiotic susceptibility profiles of two *E. anophelis* strains isolated from horses. Whole genome sequence analysis suggests that these two strains are clonal and closely related to certain human clinical *E. anophelis* isolates.

**Materials and methods**

**Strains and growth conditions**

Strains OSUV-1 and OSUVM-2 were isolated in 2016 from diagnostic specimens associated with horses in Oklahoma that were submitted to the Oklahoma Animal Disease Diagnostic Laboratory. OSUVM-1 was cultured from a swab taken from an endoscope used at an equine hospital; and OSUVM-2 was isolated from a guttural pouch aspirate obtained from a 9-year-old intact female quarter horse that presented to Boren Veterinary Medical Teaching Hospital (BVMTH) with a previous history of strangles. In addition to OSUVM-1 and OSUVM-2, *Pseudomonas aeruginosa*, *Stenotrophomonas maltophilia* and *Chryseobacterium* spp. like bacteria were isolated from both specimens. All bacterial isolates were identified using MALDI-TOF MS. Working stocks of the *Elizabethkingia* isolates OSUVM-1 and OSUVM-2 were prepared from pure cultures grown on heart infusion agar (Remel, San Diego, CA, USA) supplemented with 5% defibrinated rabbit blood (Hemostat Laboratories, Dixon, CA, USA) that were incubated overnight at 37°C and subsequently stored at 4°C. Working cultures of each strain were prepared by inoculating a single colony into 3 ml of heart infusion (HIB) or Mueller Hinton broth (MHB) (Becton Dickinson and Company, Cockeysville, MD, USA) and incubated overnight (37°C, 200 rpm).

**Isolate identification using MALDI-TOF mass spectrometry**

For bacterial identification, fresh colonies grown on tryptic soy agar containing 5% sheep blood (Fisher Scientific, Hampton, NH, USA) were applied to a spot on the MALDI-TOF MS target plate and overlaid with freshly made matrix solution (Bruker Daltonics, Billerica, MA USA) containing 70% formic acid (Sigma-Aldrich, St Louis, MO, USA) and α-cyano-4-hydroxycinnamic acid following the manufacturer’s recommendations. Bacterial identification was carried out using a Microflex LT MALDI-TOF mass spectrometer (Bruker Daltonics) using default settings. Bacterial peptide spectra were collected using FlexControl software (version 3.4, Bruker Daltonics) in positive linear mode with a mass range from 2 to 20 kDa and a laser frequency of 60 Hz (IS1~20 kV; IS2~18 kV; lens—6 kV; extraction delay time of 100 ns) in
automatic mode by accumulating a maximum of 240 profiles (40 laser shots from six different positions of the target spot). Microbial peptide mass spectra were then analyzed using the Biotyper RTC software version 3.1 using the default settings and database version 4.0.0.1 (Bruker Daltonics). Both OSUV-1 and OSUVM-2 were identified by MALDI-TOF MS as *E. meningoseptica*. This is consistent with the known insufficiency of MALDI-TOF MS default databases to correctly identify certain *Flavobacteriaceae*, including species belonging to the *Chryseobacterium* and *Elizabethkingia* genera [61–63].

**Genome sequencing, assembly, annotation, and phylogenetic analysis**

Genomic DNA was isolated from 3 ml overnight cultures of OSUV-1 and OSUVM-2 grown in HIB as described above using Qiagen Genomic-tip 100/g columns (Qiagen, Germantown, MD, USA) following the manufacturer’s protocol. The resulting DNA samples were sent to Molecular Research LP (Shallowater, TX, USA) where library preparation was performed using the Nextera DNA sample preparation kit (Illumina Inc., San Diego, CA, USA). Genomic DNA was then sequenced using PacBio SMRT sequencing and Illumina MiSeq systems and assembled using SeqMan NGen® version 12.0 (DNASTAR, Madison, WI, USA) with paired end sequencing parameters on the default settings. The resulting assemblies were annotated using the Rapid Annotations Using Subsystems Technology (RAST) server [64–66] and the Prokaryote Genome Annotation Pipeline [67]. Both genomes were further analyzed using the nucleotide and protein Basic Local Alignment Search Tool (BLAST) [68, 69]. The draft genome sequences can be found under bioproject PRJNA397081. OSUV-1 and OSUVM-2 are represented by biosamples SAMN08100548 and SAMN08100549 and nucleotide accession numbers PJMA00000000 and PJLZ00000000, respectively.

The OSUVM-1 and OSUVM-2 genomes were shared with the Special Bacteriology Reference Laboratory (SBRL) at CDC, where they were compared to the genomes of *E. anophelis* isolates derived from human clinical specimens which were obtained after the 2016 Wisconsin *Elizabethkingia* outbreak [30] in response to a general request from CDC to the various state public health departments for all *Elizabethkingia* isolates, which have been sequenced as a part of a larger project. Three isolates were found to be closely related to OSUVM-1 and OSUVM-2. These genomes had been sequenced from cultures grown at 35˚C on heart infusion agar (Difco) supplemented with 5% rabbit blood (Hemostat Laboratories). DNA was extracted using the Zymo ZR Fungal/Bacterial DNA Microprep kit (Zymo Research, Irvine, CA; strain 16–293), or the MasterPure™ Complete DNA and RNA Purification Kit (Epicentre, Madison, WI; strains 16–487 and 17–001), according to the manufacturer’s instructions. Libraries were prepared using the NEBNext Ultra DNA library prep kit (New England Biolabs, Inc., Ipswich, MA, USA), then sequencing was done with an Illumina MiSeq instrument using a 2x250 paired-end protocol as described previously [70]. The de Bruijn graph de novo assembler in CLC Genomics Workbench version 9.0. (CLCbio, Aarhus, Denmark) was used on reads trimmed with a quality limit of 0.02 to produce draft genomes. Ambiguous nucleotides (N’s) in the resulting contigs were resolved using read alignments, and contigs were split wherever N’s could not be resolved. The accession numbers of these strains are NWMM00000000, NWMI00000000, and NWMH00000000. Genomes were aligned and single nucleotide polymorphism (SNP) trees produced using HarvestTools [71], and exported Newick files were edited using MEGA v6 [72].

**Antibiotic susceptibility testing**

Minimum inhibitory concentrations (MIC) of antibiotics were determined using either standard CLSI protocols [73] for clindamycin, vancomycin, and fusidic acid, or the Sensititre
automated system (Thermo Scientific, Waltham, MA, USA) following the manufacturer’s protocol for equine samples.

**Results and discussion**

**Sequencing and mass spectrometry analysis**

The assembly of OSUVM-1 sequence data produced 7 contigs and a genome of 4,153,767 bp (%GC = 35.5). OSUVM-1 contained 3,850 putative coding sequences (CDS), of which 3,777 were protein CDS. RAST annotation assigned function to 2,421 (64%) predicted protein CDS and identified 75 rRNA and tRNA CDS.

OSUVM-2 sequences were assembled into 10 contigs to produce a genome of 4,109,384 bp (%GC = 35.5). OSUVM-2 contained 3,814 CDS, of which 3,750 were protein CDS. RAST annotation assigned function to 2,404 (64%) predicted protein CDS and identified 64 rRNA and tRNA CDS.

Bacterial identification using MALDI-TOF indicated that both OSUVM-1 and OSUVM-2 were members of the *Elizabethkingia* genus. The *Elizabethkingia* are nonmotile [42] and RAST analysis of the draft genomes of OSUVM-1 and OSUVM-2 revealed no features supporting motility and chemotaxis (S1 Table). The subsystem feature count in both strains were identical for 16 of 25 subsystems identified in the draft genomes (S1 Table). The two draft genomes differed in the feature count of the following subsystems: cell wall and capsule; virulence, disease, and defense; miscellaneous; membrane transport; iron acquisition and metabolism; protein metabolism; stress response; metabolism of aromatic compounds; and phages, prophages, and transposable elements (S1 Table). This last finding is consistent with our expectation that the loci carried by mobile genetic elements will be better represented in a complete genome than a draft genome, since a draft genome will contain a single copy of a transposon sequence (with coverage levels scaled to the number of copies of the transposon in the genome) while a complete genome will allow each gene in multiple copies to be identified.

**Core genome and phylogenetic analysis**

Nucleotide BLAST and phylogenetic analysis of the core genome of both isolates revealed that both strains were *E. anophelis*. It is of interest to note that OSUVM-1 and OSUVM-2 are part of a clade of strains resembling *E. anophelis* strain JM-87 [9, 74] (which was isolated from *Zea mays* stem tissue and initially described as the type strain of "*Elizabethkingia endophytica*" before whole genome sequence analysis revealed it to belonged to the *E. anophelis* species) rather than the clade containing *E. anophelis* type strain DSM_23781, which was isolated from the midgut of a mosquito (Fig 1) [9, 75].

Using the HarvestTools v1.1.2 module ParSNP, we determined that both OSUVM-1 and OSUVM-2 are closely related to *E. anophelis* isolates derived from human clinical specimens in Minnesota, Illinois, and Tennessee (Fig 1). A second analysis limited to OSUVM-1, OSUVM-2, and the three human clinical isolates, detected an 87% core genome among the five strains. Once ambiguous nucleotides were excluded only 198 SNP positions were located, scattered throughout the core genome of the five strains, and OSUVM-1 and OSUVM-2 differed by only 6 SNPs.

These results indicate that these five strains are highly related and that the two OSUVM isolates share commonalities with strains isolated from humans manifesting with disease caused by *Elizabethkingia*. Interestingly, Hu et al. [8] reported that an *Elizabethkingia miricola* strain responsible for a contagious disease resulting in black-spotted frog losses at farms in China was comparable to a human *E. miricola* isolate. Collectively these findings suggest that *Elizabethkingia* are not host-specific, which raises the possibility that *Elizabethkingia* might have the
potential to move between humans and animals in a similar manner to known zoonotic pathogens.

**Subsystem analysis**

**Beta-lactamases.** Genomic analysis of _Elizabethkingia_ spp. consistently identifies multiple β-lactamases, including three characterized β-lactamases [41, 76, 77], along with a varying number of putative β-lactamases [1, 2, 4, 5, 9, 11, 25, 29, 30, 70, 78]. The 19 putative β-lactamase CDS in both OSUVM-1 and OSUVM-2 included the previously characterized class A serine β-lactamase (SBL) _bla_CME_1 [76], and metallo-β-lactamases (MBL) class B1 _bla_B14_ [41] and class B3 _bla_GOB18_ [77]. Of the remaining 16 putative β-lactamases, one is similar to the previously

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**Fig 1.** Core genome single nucleotide polymorphism tree showing the position of OSUVM-1 and OSUVM-2 compared to the _Elizabethkingia anophelis_ strains reported by Nicholson et al. Type strains are denoted by a superscript T, and the location of the isolates from this study is denoted by a bracket.

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characterized class A SBL blaCIA-1 from *Chryseobacterium indologenes* (67% amino acid identity) [79]. 11 are similar to class C SBLs, and the remaining 7 are classified as putative MBLs.

**Multidrug efflux pumps.** Efflux pumps are a key component of the intrinsic antibiotic-resistance mechanism of many bacteria and function by transporting antibiotics from within the cell to the outside [80–82]. Efflux pumps are characterized as belonging to five families: ATP-binding cassette (ABC) [83], major facilitator superfamily (MFS) [84, 85], multidrug and toxic compound extrusion (MATE) [86], resistance-nodulation-cell division (RND) [87], and small multidrug resistance (SMR) [88]. Genomic annotation of all *Elizabethkingia* spp. reveals the presence of several drug efflux pumps, yet none of these transporters has been phenotypically characterized [1, 2, 4, 5, 9, 11, 25, 29, 30, 70, 78]. RAST annotation revealed 32 CDS related to antibiotic efflux in both OSUVM-1 and OSUVM-2: 18 of the 32 CDS (56%) were identified by RAST analysis as components of RND efflux operons, 12 CDS (38%) as components of MFS operons, while the remaining 2 CDS (6%) were identified as MATE efflux pumps. 

We are interested in the RND pumps in the draft genomes of OSUVM-1 and OSUVM-2 since RND efflux pumps can be a major factor contributing to clinically-relevant resistance to certain antibiotics in Gram-negative organisms [80]. Tripartite RND efflux pumps consist of an inner membrane pump attached to an outer membrane porin by way of a periplasmic adaptor protein [82, 87, 89, 90]. Although the arrangement of the genes that encode RND components varies among organisms, they can be found in a single operon in organisms such as *Pseudomonas aeruginosa* (e.g. *mexAB-oprM*) and *Campylobacter jejuni* (e.g. *cmeABC*) [87, 91]. When genes encoding the MexAB-OprM efflux pump in *P. aeruginosa* and the CmeABC efflux operon in *C. jejuni* are inactivated, a significant decrease in the MICs for various β-lactams, chloramphenicol, ciprofloxacin, erythromycin, nalidixic acid, and tetracycline is observed [90, 92–94].

The 18 CDS identified by RAST analysis as components of tripartite RND efflux pumps were all identical in OSUVM-1 and OSUVM-2 at the nucleotide level. These genes presented as six, three-gene operons, organized in the same manner as the *mexAB-oprM* and *cmeABC* operons. The OSUVM-1 and OSUVM-2 RND inner membrane pumps demonstrated 28–42% amino acid identity to MexB and CmeB, the periplasmic adaptor proteins demonstrated 24–27% amino acid identity to MexA and CmeA, while the outer membrane porins demonstrated 25–29% amino acid identity to OprM and CmeC. These homologies only suggest a relationship between these operons and characterized RND efflux systems. It should be noted that when Schindler et al. [95] cloned and expressed 21 genes putatively identified as encoding efflux proteins in *Staphylococcus aureus*, none resulted in increased MICs for any of the substrates tested, calling into question the function of these genes in drug efflux. As a result, it is important that the putative efflux genes from *Elizabethkingia* isolates be confirmed as drug resistance efflux pumps through biochemical analysis.

**Antimicrobial susceptibility testing**

Both OSUVM-1 and OSUVM-2 demonstrated high MICs for cefazolin, ceftazidime, ceftriaxone, ampicillin, penicillin, ticarcillin, ticarcillin + clavulanic acid, imipenem, amikacin, gentamicin, chloramphenicol, fusidic acid, and tetracycline (S2 Table). While the confirmed active β-lactamas in *Elizabethkingia* are known to contribute to resistance to a wide array of antibiotics that target penicillin-binding proteins [45–47], other mechanisms such as multidrug efflux, outer membrane alterations and penicillin-binding proteins that demonstrate reduced affinity for β-lactams can also contribute to β-lactam resistance, although these mechanisms remain untested in *Elizabethkingia* [81, 92, 93].

Interestingly OSUVM-1 demonstrated an oxacillin MIC of 0.25 mg/l, while OSUVM-2 showed a higher oxacillin MIC (≥ 4 mg/l), and overall OSUVM-2 displayed higher MICs for
11 of the antibiotics tested (S2 Table). Since the genes associated with resistances are identical in both strains, these MIC differences may be attributed to unidentified SNPs or specific gene content differences outside the core genome.

Both OSUVM-1 and OSUVM-2 demonstrated low MICs to ciprofloxacin and enrofloxacin, suggesting they are susceptible to these fluoroquinolones (S2 Table). Ciprofloxacin resistance in Gram-negative bacteria is driven primarily by mutations in the DNA gyrase A subunit (gyrA), and resistance is enhanced in both cases by mutations in gyrB, parC, and parE [96–101]. The E. anophelis gyrA encodes a predicted protein of 858 amino acids, and Perrin et al. [30] identified a Ser83Ile mutation in the gyrA of an E. anophelis strain isolated during the 2016 Wisconsin outbreak that displayed an increased ciprofloxacin MIC. Lin et al. [25] subsequently identified the same mutation in another E. anophelis strain which also demonstrated an elevated ciprofloxacin MIC. Thus, it is probable that the gyrA mutation Ser83Ile imparts ciprofloxacin resistance in E. anophelis, as it does for E. coli [102–107]. Both OSUVM-1 and OSUVM-2 contain the wild-type serine at position 83, along with two mutations, Val841Ala and Ala842Ile. Positions 841 and 842 lie outside of the region of gyrA thought to be responsible for fluoroquinolone resistance [96, 97, 102, 104] and the low fluoroquinolone MICs demonstrated by both strains are consistent with the expectation that these mutations would not convey fluoroquinolone resistance.

Vancomycin is used extensively for treating Gram-positive infections, in particular infections caused by methicillin-resistant S. aureus (MRSA) and Clostridium difficile [108, 109]. Gram-negative organisms are normally intrinsically refractory to the action of vancomycin and exhibit MICs > 64 mg/l [21], except Elizabethkingia, which have been reported to exhibit vancomycin MICs as low as 1 mg/l [16–19, 78, 110]. Vancomycin has been used singly or in combination therapies to treat Elizabethkingia infections with mixed success (reviewed in [110]). Furthermore, Hazuka et al. [24] reported that when an isolate of E. meningoseptica was exposed to vancomycin for 6 days, the MIC increased from 8 mg/l to 64 mg/l. Vancomycin dosing recommendations suggest that a serum trough concentration of between 15 to 20 mg/l should be reached and maintained to kill susceptible organisms, but this guidance requires that the target organism has a vancomycin MIC < 1 mg/l [108, 109, 111]. Using this standard, OSUVM-1 and OSUVM-2 (vancomycin MICs = 8 and 32 mg/l, respectively) would be resistant to vancomycin.

**Conclusion**

Here we report the first two draft genomes from Elizabethkingia associated with horses, and that these two isolates are closely related to isolates derived from human infections, although to date no direct evidence for transmission of Elizabethkingia between humans and animals has been observed. We further demonstrated that both isolates display low MICs for ciprofloxacin and that both isolates display an elevated MIC for vancomycin. Clinical reports have shown potential efficacy for vancomycin in treating Elizabethkingia infections despite in vitro susceptibility results that would suggest otherwise [20, 22, 26, 32, 35, 37, 43–45], although treatment failure with vancomycin has also been reported [24, 27, 38]. We hope that this report of vancomycin-resistant E. anophelis isolates will stimulate discussion and further research to determine the efficacy (or lack thereof) of vancomycin in treating Elizabethkingia infections.

**Supporting information**

S1 Table. Distribution in coding sequence function as identified by RAST. Subsystems with differences in the number of coding sequences in the two strains are highlighted in bold. (PDF)
S2 Table. Minimum inhibitory concentrations for select antibiotics determined by the Sensititre system or broth microdilution method. Antibiotics displaying different MICs are highlighted in bold.

(PDF)

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Author Contributions
Conceptualization: Akhilesh Ramachandran, John E. Gustafson.
Data curation: William L. Johnson, Ainsley C. Nicholson, Scot E. Dowd.
Formal analysis: William L. Johnson, Ainsley C. Nicholson, Anne M. Whitney, Melissa Bell, Aaron Villarma, Ben W. Humrighouse, Mili Sheth, Scot E. Dowd.
Funding acquisition: Akhilesh Ramachandran, John R. McQuiston, John E. Gustafson.
Investigation: William L. Johnson, Akhilesh Ramachandran, Nathanial J. Torres, Ainsley C. Nicholson, Anne M. Whitney, Melissa Bell, Aaron Villarma, Ben W. Humrighouse, Mili Sheth, Scot E. Dowd.
Methodology: Akhilesh Ramachandran, Ainsley C. Nicholson, John R. McQuiston, John E. Gustafson.
Project administration: Akhilesh Ramachandran, Scot E. Dowd, John R. McQuiston, John E. Gustafson.
Resources: Akhilesh Ramachandran, Scot E. Dowd, John R. McQuiston, John E. Gustafson.
Supervision: Akhilesh Ramachandran, Ainsley C. Nicholson, Scot E. Dowd, John R. McQuiston, John E. Gustafson.
Validation: Scot E. Dowd.
Visualization: William L. Johnson, Ainsley C. Nicholson, Scot E. Dowd.
Writing – original draft: William L. Johnson, Akhilesh Ramachandran, Ainsley C. Nicholson, John E. Gustafson.
Writing – review & editing: Nathanial J. Torres, Anne M. Whitney, Melissa Bell, Aaron Villarma, Ben W. Humrighouse, Mili Sheth, Scot E. Dowd, John R. McQuiston.

References
1. Kukutla P, Lindberg BG, Pei D, Rayl M, Yu W, Steritz M, et al. Draft Genome Sequences of Elizabethkingia anophelis Strains R26T and Ag1 from the Midgut of the Malaria Mosquito Anopheles gambiae. Genome Announc. 2013; 1(6). Epub 2013/12/07. https://doi.org/10.1128/genomeA.01030-13 PMID: 24309745; PubMed Central PMCID: PMCPMC3853068.
2. Lee D, Kim YK, Kim YS, Kim TJ. Complete Genome Sequence of Elizabethkingia sp. BM10, a Symbiotic Bacterium of the Wood-Feeding Termite Reticulitermes speratus KMT1. Genome Announc. 2015; 3(5). Epub 2015/10/10. https://doi.org/10.1128/genomeA.01181-15 PMID: 26450743; PubMed Central PMCID: PMCPMC4599102.
3. Mee PT, Lynch SE, Walker PJ, Melville L, Duchemin JB. Detection of Elizabethkingia spp. in Culicoides Biting Midges, Australia. Emerg Infect Dis. 2017; 23(8):1409–10. Epub 2017/07/21. https://doi.org/10.3201/eid2308.161565 PMID: 28726605; PubMed Central PMCID: PMCPMC5547790.
4. Pei D, Nicholson AC, Jiang J, Chen H, Whitney AM, Villarma A, et al. Complete Circularized Genome Sequences of Four Strains of *Elizabethkingia anophelis*, Including Two Novel Strains Isolated from Wild-Caught *Anopheles sinensis*. Genome Announc. 2017; 5(47). Epub 2017/11/24. https://doi.org/10.1128/genomeA.01359-17 PMID: 29167265.

5. Raygoza Garay JA, Hughes GL, Koundal V, Rasgon JL, Mwangi MM. Genome Sequence of *Elizabethkingia anophelis* Strain EaAs1, Isolated from the Asian Malaria Mosquito *Anopheles stephensi*. Genome Announc. 2016; 4(2). Epub 2016/03/12. https://doi.org/10.1128/genomeA.00084-16 PMID: 26966196; PubMed Central PMCID: PMCPMC4786652.

6. Jiang HY, Ma JE, Li J, Zhang XJ, Li LM, He N, et al. Diets Alter the Gut Microbiome of Crocodile Lizards. Front Microbiol. 2017; 8:2073. Epub 2017/11/10. https://doi.org/10.3389/fmicb.2017.02073 PMID: 29118742; PubMed Central PMCID: PMCPMC5660983.

7. Jacobs A, Chenia HY. Biofilm formation and adherence characteristics of an *Elizabethkingia meningoseptica* isolate from *Oreochromis mossambicus*. Ann Clin Microbiol Antimicrob. 2011; 10:16. Epub 2011/05/07. https://doi.org/10.1186/1476-0711-10-16 PMID: 21545730; PubMed Central PMCID: PMCPMC3112384.

8. Djem O, Yuan J, Meng Y, Wang Z, Gu Z. Pathogenic *Elizabethkingia miricola* Infection in Cultured Black-Spotted Frogs, China. Emerg Infect Dis. 2017; 23(12):2055–9. Epub 2017/11/18. https://doi.org/10.3201/eid2312.170942 PMID: 29148374.

9. Kämper P, Busse HJ, McInroy JA, Glaeser SP. *Elizabethkingia endophytica* sp. nov., isolated from Zea mays and emended description of *Elizabethkingia anophelis* Kämper et al. 2011. Int J Syst Evol Microbiol. 2015; 65(7):2187–93. Epub 2015/04/11. https://doi.org/10.1099/ijsem.0.00239 PMID: 25858248.

10. Moore LS, Owens DS, Jepson A, Turton JF, Ashworth S, Donaldson H, et al. Waterborne *Elizabethkingia meningoseptica* In Adult Critical Care. Emerg Infect Dis. 2016; 22(1):9–17. Epub 2015/12/23. https://doi.org/10.3201/eid2201.150139 PMID: 26690562; PubMed Central PMCID: PMCPMC4696684.

11. Teo J, Tan SY, Liu Y, Tay M, Ding Y, Li Y, et al. Comparative genomic analysis of malaria mosquito vector-associated novel pathogen *Elizabethkingia anophelis*. Genome Biol Evol. 2014; 6(5):1158–65. Epub 2014/05/08. https://doi.org/10.1093/gbe/evu094 PMID: 24803570; PubMed Central PMCID: PMCPMC4041001.

12. Kim KK, Kim MK, Lim JH, Park HY, Lee ST. Transfer of *Chryseobacterium meningosepticum* and *Chryseobacterium miricola* to *Elizabethkingia gen. nov.* as *Elizabethkingia meningoseptica comb. nov.* and *Elizabethkingia miricola comb. nov.* Int J Syst Evol Microbiol. 2005; 55(Pt 3):1287–93. Epub 2005/05/10. https://doi.org/10.1099/ijsem.0.06351-0 PMID: 15879269.

13. Jean SS, Lee WS, Chen FL, Ou TY, Hsueh PR. *Elizabethkingia meningoseptica*: an important emerging pathogen causing healthcare-associated infections. J Hosp Infect. 2014; 86(4):244–9. Epub 2014/04/01. https://doi.org/10.1016/j.jhin.2014.01.009 PMID: 24680187.

14. Lau SK, Chow WN, Foo CH, Curreem SO, Lo GC, Teng JL, et al. *Elizabethkingia anophelis* bacteremia is associated with clinically significant infections and high mortality. Sci Rep. 2016; 6:26045. Epub 2016/05/18. https://doi.org/10.1038/srep26045 PMID: 27185741; PubMed Central PMCID: PMCPMC4868968.

15. Aber RC, Wennersten C, Moellerling RC Jr. Antimicrobial susceptibility of *flavobacteria*. Antimicrob Agents Chemother. 1978; 14(3):483–7. Epub 1978/09/01. PMID: 708026; PubMed Central PMCID: PMCPMC352486.

16. Altmann G, Bogokovsky B. In-vitro sensitivity of *Flavobacterium meningosepticum* to antimicrobial agents. J Med Microbiol. 1971; 4(2):296–9. Epub 1971/05/01. https://doi.org/10.1099/00222615-4-2-296 PMID: 4105616.

17. Ceyhan M, Yildirim I, Tekeli A, Yurdakok M, Us E, Altun B, et al. A *Chryseobacterium meningosepticum* outbreak observed in 3 clusters involving both neonatal and non-neonatal pediatric patients. Am J Infect Control. 2008; 36(6):453–7. Epub 2008/08/05. https://doi.org/10.1016/j.ajic.2007.09.008 PMID: 18675153.

18. Chang JC, Hsueh PR, Wu JJ, Ho SW, Hsieh WC, Luh KT. Antimicrobial susceptibility of *flavobacteria* as determined by agar dilution and disk diffusion methods. Antimicrob Agents Chemother. 1997; 41 (6):1301–6. Epub 1997/06/01. PMID: 9174188; PubMed Central PMCID: PMCPMC163904.

19. Di Pentima MC, Mason EO Jr., Kaplan SL. *In vitro* antibiotic synergy against *Flavobacterium meningosepticum*: implications for therapeutic options. Clin Infect Dis. 1998; 26(5):1169–76. Epub 1998/05/23. PMID: 9597247.

20. Dias M, Prashant K, Pai R, Scaria B. *Chryseobacterium meningosepticum* bacteremia in diabetic nephropathy patient on hemodialysis. Indian J Nephrol. 2010; 20(4):203–4. Epub 2011/01/06. https://doi.org/10.4103/0971-4065.73460 PMID: 21206682; PubMed Central PMCID: PMCPMC3008949.
21. Fass RJ, Barnishan J. In vitro susceptibilities of nonfermentative gram-negative bacilli other than *Pseudomonas aeruginosa* to 32 antimicrobial agents. Rev Infect Dis. 1980; 2(6):S41–53. Epub 1980/11/01. PMID: 7012987.

22. Gunog S, Ozen M, Akiniz A, Durmaz R. A *Chryseobacterium meningosepticum* outbreak in a neonatal ward. Infect Control Hosp Epidemiol. 2003; 24(8):613–7. Epub 2003/08/28. doi:10.1086/522617 PMID: 12940584.

23. Gupta P, Zaman K, Mohan B, Taneja N. *Elizabthkingia miricola*: A rare non-fermenter causing urinary tract infection. World J Clin Cases. 2017; 5(5):187–90. doi:10.1099/wjcc.v5.i5.187 PMID: 28560237; PubMed Central PMCID: PMC5434319.

24. Hazuka BT, Dajani AS, Talbott K, Keen BM. Two outbreaks of *Flavobacterium meningosepticum* type E in a neonatal intensive care unit. J Clin Microbiol. 1977; 6(5):450–55. Epub 1977/11/01. PMID: 925147; PubMed Central PMCID: PMC274796.

25. Lin JN, Lai CH, Yang CH, Huang YH, Lin HH. Genomic features, phylogenetic relationships, and comparative genomics of *Elizabthkingia anophelis* strain EM361-97 isolated in Taiwan. Sci Rep. 2017; 7(1):14317. Epub 2017/11/01. doi:10.1038/s41598-017-14841-8 PMID: 29085032; PubMed Central PMCID: PMC5662595.

26. Montrucchio G, Corscione S, Vaj M, Zaccaria T, Costa C, Brazzi L, et al. First case of *Chryseobacterium meningosepticum* in neononetal patients. J Clin Microbiol. 2004; 42(7):3353–5. Epub 2004/07/10. doi:10.1128/JCM.42.7.3353-3355.2004 PMID: 15243115; PubMed Central PMCID: PMC446307.

27. Lothuvachai T, Likittanasombat K, Milindanakura S, Sakulsangprapha A, Kitiyakara C. *Chryseobacterium meningosepticum* infection and cardiac tamponade in a long-term hemodialysis patient. Am J Kidney Dis. 2006; 48(4):e49–53. Epub 2006/09/26. doi:10.1053/j.ajkd.2006.07.010 PMID: 16997045.

28. Plotkin SA, McKitrick JC. Nosocomial meningitis of the newborn caused by a *flavobacterium*. JAMA. 1966; 198(6):662–4. Epub 1966/11/07. PMID: 5953444.

29. Opota O, Diene SM, Bertelli C, Prod'hom P, Eckert P, Greub G. Genome of the carbapenemase-producing clinical isolate *Elizabethkingia miricola* EM CHUV and comparative genomics with *Elizabethkingia meningoseptica* and *Elizabethkingia anophelis*: evidence for intrinsic multidrug resistance trait of emerging pathogens. Int J Antimicrob Agents. 2017; 49(1):93–7. doi:10.1016/j.ijantimicag. 2016.09.031 PMID: 27913093.

30. Perrin A, Larsonneur E, Nicholson AC, Edwards DJ, Gundlach KM, Whitney AM, et al. Evolutionary dynamics and genomic features of the *Elizabthkingia anophelis* 2015 to 2016 Wisconsin outbreak strain. Nat Commun. 2017; 8:15483. Epub 2017/05/26. doi:10.1038/ncomms15483 PMID: 28537263; PubMed Central PMCID: PMC5458099.

31. Tay IC, Liu TP, Chen YJ, Lien RI, Lee CY, Huang YC. Outbreak of *Elizabethkingia meningoseptica* in an intensive care unit. New Microbiol. 2003; 26(1):57–63. Epub 2003/05/19. doi:10.1299/nm.2003.57-63 PMID: 12940584.

32. Sader HS, Jones RN, Pfaller MA. Relapse of catheter-related *Flavobacterium meningosepticum* bacteremia demonstrated by DNA macrorestriction analysis. Clin Infect Dis. 1995; 21(4):997–1000. Epub 1995/10/01. PMID: 8645856.

33. Sebastiampillai BS, Luke NV, Silva S, De Silva ST, Premaratna R. Septicaemia caused by *Elizabethkingia* in a ‘healthy’ Sri Lankan man. Trop Doct. 2017; 47:20135. Epub 2017/06/24. doi:10.1177/0049475517717135 PMID: 28641481.

34. Tai IC, Liu TP, Chen YJ, Lien RI, Lee CY, Huang YC. Outbreak of *Elizabethkingia meningoseptica* sepsis with meningitis in a well-baby nursery. J Hosp Infect. 2017; 96(2):168–71. Epub 2017/01/13. doi:10.1016/j.jhin.2016.11.018 PMID: 28077242.

35. Tekerekgolu MS, Durmaz R, Ayan M, Cizmeci Z, Akinci A. Analysis of an outbreak due to *Chryseobacterium meningosepticum* in a neonatal intensive care unit. New Microbiol. 2003; 26(1):57–63. Epub 2003/02/13. PMID: 12940584.

36. Teo J, Tan SY, Tay M, Ding Y, Kjelleberg S, Givskov M, et al. First case of *E anophelis* outbreak in an intensive-care unit. Lancet. 2013; 382(9985):855–6. Epub 2013/09/10. doi:10.1016/S0140-6736(13)61858-9 PMID: 24012265.

37. Tizer KB, Cervia JS, Dunn AM, Stavola JJ, Noel GJ. Successful combination vancomycin and rifampin therapy in a newborn with community-acquired *Flavobacterium meningosepticum* neonatal meningitis. Pediatr Infect Dis J. 1995; 14(10):916–7. Epub 1995/10/01. PMID: 8584328.

38. Tseng MH, Dang LK, Su YC, Lin SH. Catheter-related *Chryseobacterium meningosepticum* bacteremia in a haemodialysis patient. NDT Plus. 2009; 2(5):433–4. Epub 2009/10/01. doi:10.1093/ndtplus/sfp080 PMID: 25949372; PubMed Central PMCID: PMC4421377.
39. Fraser SL, Jorgensen JH. Reappraisal of the antimicrobial susceptibilities of Chryseobacterium and Flavobacterium species and methods for reliable susceptibility testing. Antimicrob Agents Chemother. 1997; 41(12):2738–41. Epub 1998/01/07. PMID: 9420049; PubMed Central PMCID: PMC164199.

40. Dziuban EJ, Franks J, So M, Peacock G, Blaney DD. Elizabethkingia in Children: A Comprehensive Review of Symptomatic Cases Reported from 1944–2017. Clin Infect Dis. 2017. Epub 2017/12/07. https://doi.org/10.1093/cid/cix152 PMID: 29211821.

41. Gonzalez LJ, Vila AJ. Carbapenem resistance in Elizabethkingia meningoseptica is mediated by metallo-beta-lactamase BlaB. Antimicrob Agents Chemother. 2012; 56(4):1686–92. Epub 2012/02/01. https://doi.org/10.1128/AAC.05835-11 PMID: 22290979; PubMed Central PMCID: PMCPMC3318372.

42. King EO. Studies on a group of previously unclassified bacteria associated with meningitis in infants. Am J Clin Pathol. 1959; 31(3):241–7. Epub 1959/03/01. PMID: 13637033.

43. Gump DW. Vancomycin for treatment of bacterial meningitis. Rev Infect Dis. 1981;3 suppl:S289–92. Epub 1981/11/01. PMID: 6896243.

44. Soman R, Agrawal U, Suthar M, Desai C, Shetty A. Successful Management of Elizabethkingia meningoseptica Meningitis with Intraventricular Vancomycin. J Assoc Physicians India. 2016; 64(10):98–9. Epub 2016/10/22. PMID: 27766817.

45. Neuner EA, Ahrens CL, Groszek JJ, Isada C, Vogelbaum MA, Fissell WH, et al. Use of therapeutic drug monitoring to treat Elizabethkingia meningoseptica meningitis and bacteremia in an adult. J Antimicrob Chemother. 2012; 67(6):1558–60. Epub 2012/02/24. https://doi.org/10.1093/jac/dks053 PMID: 22357803; PubMed Central PMCID: PMCPMC350328.

46. Centers for Disease Control and Prevention. Recent Outbreaks [Web Page]. 2016 [updated 6/16/2017; cited 2017 4/16]. Available from: https://www.cdc.gov/elizabethkingia/outbreaks/.

47. Wisconsin Department of Public Health Services. Elizabethkingia [Web Page]. 2015 [updated 5/2017; cited 2017 4/16]. Available from: https://www.dhs.wisconsin.gov/disease/elizabethkingia.htm.

48. Navon L, Clegg WJ, Morgan J, Austin C, McQuiston JR, Blaney DD, et al. Notes from the Field: Investigation of Elizabethkingia anophelis Cluster—Illinois, 2014–2016. MMWR Morb Mortal Wkly Rep. 2016; 65(48):1380–1. Epub 2016/12/10. https://doi.org/10.15585/mmwr.mm6548a6 PMID: 27932784.

49. Frank T, Gody JC, Nguyen LB, Berthet N, Le Fleche-Mateos A, Bata P, et al. First case of Elizabethkingia anophelis meningitis in the Central African Republic. Lancet. 2013; 381(9880):1876. Epub 2013/05/28. https://doi.org/10.1016/S0140-6736(13)60318-9 PMID: 23706804.

50. Kenna DTD, Fuller A, Martin K, Perry C, Pike R, Burns PJ, et al. ropB gene sequencing highlights the prevalence of an E. miricola cluster over other Elizabethkingia species among UK cystic fibrosis patients. Diagn Microbiol Infect Dis. 2017. Epub 2017/11/28. https://doi.org/10.1016/j.diagmicrobio.2017.10.014 PMID: 29174734.

51. Agarwal S, Kakati B, Khanduri S, Gupta S. Emergence of Carbapenem Resistant Non-Fermenting Gram-Negative Bacilli Isolated in an ICU of a Tertiary Care Hospital. J Clin Diagn Res. 2017; 11(1):DC04–DC7. Epub 2017/03/10. https://doi.org/10.7860/JCDR/2017/24023.9317 PMID: 28273965; PubMed Central PMCID: PMCPMC5324410.

52. Matyi SA, Dupre JM, Johnson WL, Hoyt PR, White DG, Brody T, et al. Isolation and characterization of Staphylococcus aureus strains from a Paso del Norte dairy. J Dairy Sci. 2013; 96(6):3535–42. Epub 2013/04/24. https://doi.org/10.3168/jds.2013-6590 PMID: 23608491; PubMed Central PMCID: PMCPMC5226338.

53. Voss A, Loeffen F, Bakker J, Klaassen C, Wulf M. Methicillin-resistant Staphylococcus aureus in pig farming. Emerg Infect Dis. 2005; 11(12):1965–6. Epub 2006/02/21. https://doi.org/10.3201/eid1112.050428 PMID: 16685492; PubMed Central PMCID: PMCPMC3367632.

54. Lozano C, Aspiroz C, Ara M, Gomez-Sanz E, Zarazaga M, Torres C. Methicillin-resistant Staphylococcus aureus (MRSA) ST398 in a farmer with skin lesions and in pigs of his farm: clonal relationship and detection of lnu(A) gene. Clin Microbiol Infect. 2011; 17(6):923–7. Epub 2011/06/21. https://doi.org/10.1111/j.1469-0691.2010.03437.x PMID: 21662806.

55. Bates J, Jordens JZ, Griffiths DT. Farm animals as a putative reservoir for vancomycin-resistant enterococcal infection in man. J Antimicrob Chemother. 1994; 34(4):507–14. Epub 1994/10/01. PMID: 7868403.

56. Guardabassi L, Loeber ME, Jacobson A. Transmission of multiple antimicrobial-resistant Staphylococcus intermedius between dogs affected by deep pyoderma and their owners. Vet Microbiol. 2004; 98(1):23–7. Epub 2004/01/24. PMID: 14738778.
57. Guardabassi L, Schwarz S, Lloyd DH. Pet animals as reservoirs of antimicrobial-resistant bacteria. J Antimicrob Chemother. 2004; 54(2):321–32. Epub 2004/07/16. https://doi.org/10.1093/jac/dkh332 PMID: 15254022.

58. van den Bogard AE, Stoberingham EE. Epidemiology of resistance to antibiotics. Links between animals and humans. Int J Antimicrob Agents. 2000; 14(4):327–35. Epub 2000/05/05. PMID: 10794955.

59. Damborg P, Olsen KE, Moller Nielsen E, Guardabassi L. Occurrence of Campylobacter jejuni in pets living with human patients infected with C. jejuni. J Clin Microbiol. 2004; 42(3):1363–4. Epub 2004/03/09. https://doi.org/10.1128/JCM.42.3.1363-1364.2004 PMID: 15004120; PubMed Central PMCID: PMC356901.

60. Bordelo J, Viegas C, Coelho C, Poeta P. First report of bacteremia caused by Elizabethkingia meningoseptica in a dog. Can Vet J. 2016; 57(9):994. Epub 2016/09/03. PMID: 27587896; PubMed Central PMCID: PMC4982576.

61. de Carvalho Filho EB, Marson FAL, Levy CE. Challenges in the identification of Chryseobacterium indologenes and Elizabethkingia meningoseptica in cases of nosocomial infections and patients with cystic fibrosis. New Microbes New Infect. 2017; 20:27–33. https://doi.org/10.1016/j.nmni.2017.09.002 PMID: 29062487; PubMed Central PMCID: PMC5643076.

62. Mirza HC, Tuncer O, Olmez S, Sener B, Tugcu GD, Ozcelik U, et al. Clinical Strains of Chryseobacterium and Elizabethkingia spp. Isolated from Pediatric Patients in a University Hospital: Performance of MALDI-TOF MS-Based Identification, Antimicrobial Susceptibilities, and Baseline Patient Characteristics. Microb Drug Resist. 2017. https://doi.org/10.1089/mdr.2017.0206 PMID: 29227188.

63. Nicholson AC, Gulvik CA, Whitney AM, Humrighouse BW, Graziano J, Emery B, et al. Revisiting the taxonomy of the genus Elizabethkingia using whole-genome sequencing, optical mapping, and MALDI-TOF, along with proposal of three novel Elizabethkingia species: Elizabethkingia bruuniana sp. nov., Elizabethkingia urasingi sp. nov., and Elizabethkingia occulta sp. nov. Antonie Van Leeuwenhoek. 2018; 111(1):55–72. https://doi.org/10.1007/s10482-017-0926-3 PMID: 28856455.

64. Aziz RK, Bartels D, Best AA, DeLongh M, Disz T, Edwards RA, et al. The RAST Server: rapid annotation of microbial genomes using Subsystems technology. BMC Genomics. 2008; 9:75. Epub 2008/02/12. https://doi.org/10.1186/1471-2164-9-75 PMID: 18261238; PubMed Central PMCID: PMC2265698.

65. Brettin T, Davis JJ, Disz T, Edwards RA, Gerdes S, Olsen GJ, et al. RASTk: a modular and extensible implementation of the RAST algorithm for building custom annotation pipelines and annotating batches of genomes. Sci Rep. 2015; 5:8365. Epub 2015/02/11. https://doi.org/10.1038/srep08365 PMID: 25666585; PubMed Central PMCID: PMC4322359.

66. Overbeek R, Olson R, Pusch GD, Olsen GJ, Davis JJ, Disz T, et al. The SEED and the Rapid Annotation of microbial genomes using Subsystems Technology (RAST). Nucleic Acids Res. 2014; 42(Database issue):D206–14. Epub 2013/12/03. https://doi.org/10.1093/nar/gkt1226 PMID: 24293654; PubMed Central PMCID: PMC3965101.

67. Angiuoli SV, Gussman A, Klimke W, Cochrane G, Field D, Garrity G, et al. Toward an online repository of Standard Operating Procedures (SOPs) for (meta)genomic annotation. OMICS. 2008; 12(2):137–41. Epub 2008/04/18. https://doi.org/10.1089/omi.2008.0017 PMID: 18416670; PubMed Central PMCID: PMC3196215.

68. Altschul SF, Gish W, Miller W, Myers EW, Lipman DJ. Basic local alignment search tool. J Mol Biol. 1990; 215(3):403–10. Epub 1990/10/05. https://doi.org/10.1016/S0022-2836(05)80360-2 PMID: 2231712.

69. Altschul SF, Madden TL, Schaffer AA, Zhang J, Zhang Z, Miller W, et al. Gapped BLAST and PSI-BLAST: a new generation of protein database search programs. Nucleic Acids Res. 1997; 25(17):3389–402. Epub 1997/09/01. PMID: 9254694; PubMed Central PMCID: PMC149617.

70. Nicholson AC, Humrighouse BW, Graziano JC, Emery B, McQuiston JR. Draft Genome Sequences of Strains Representing Each of the Elizabethkingia Genospecies Previously Determined by DNA-DNA Hybridization. Genome Announc. 2016; 4(2). https://doi.org/10.1128/genomeA.00045-16 PMID: 26966213; PubMed Central PMCID: PMC4786648.

71. Treangen TJ, Ondov BD, Koren S, Phillippy AM. The Harvest suite for rapid core-genome alignment and visualization of thousands of intraspecific microbial genomes. Genome Biol. 2014; 15(11):524. Epub 2014/11/21. https://doi.org/10.1186/s13059-014-0524-x PMID: 25410596; PubMed Central PMCID: PMC4262987.

72. Tamura K, Stecher G, Peterson D, Filipski A, Kumar S. MEGA6: Molecular Evolutionary Genetics Analysis version 6.0. Mol Biol Evol. 2013; 30(12):2725–9. Epub 2013/10/18. https://doi.org/10.1093/molbev/ms3197 PMID: 24132122; PubMed Central PMCID: PMC3840312.

73. Clinical and Laboratory Standards Institute. Clinical and Laboratory Standards Institute: [document]. Wayne, Pa.: Clinical and Laboratory Standards Institute; 2005. p. volumes.
74. Dojlad S, Ghosh H, Glaeser S, Kampfer P, Chakraborty T. Taxonomic reassessment of the genus Elizabethkingia using whole-genome sequencing. *Elizabethkingia endophytica* Kampfer et al. 2015 is a later subjective synonym of *Elizabethkingia anophelis* Kampfer et al. 2011. Int J Syst Evol Microbiol. 2016; 66(11):4555–9. Epub 2016/08/09. https://doi.org/10.1099/ijsem.0.01390 PMID: 27498788.

75. Kampfer P, Matthews H, Glaeser SP, Martin K, Lodders N, Faye I. *Elizabethkingia anophelis* sp. nov., isolated from the midgut of the mosquito *Anopheles gambiae*. Int J Syst Evol Microbiol. 2011; 61(Pt 11):2670–9. Epub 2010/12/21. https://doi.org/10.1099/ijsem.0.02639-0 PMID: 21169462.

76. Rossolini GM, Franceschini N, Lauretti L, Caravelli B, Riccio ML, Galleni M, et al. Cloning of a *Chryseobacterium (Flavobacterium) meningosepticum* chromosomal gene (*blaA(CME)*) encoding an extended-spectrum class A beta-lactamase related to the Bacteroides cephalospinorinas and the VEB-1 and PER beta-lactamases. Antimicrob Agents Chemother. 1999; 43(9):2193–9. Epub 1999/09/03. PMID: 10471563; PubMed Central PMCID: PMCPMC89445.

77. Moran-Barrio J, Lisa MN, Larrieux N, Drusin SI, Viale AM, Moreno DM, et al. Crystal Structure of the Metallo-beta-Lactamase GOb in the Periplasmic Dizinc Form Reveals an Unusual Metal Site. *Antimicrob Agents Chemother*. 2015; 59(11):5471–82. Epub 2015/08/13. https://doi.org/10.1128/AAC.01096-16 PMID: 26282926.

78. Lin XH, Sun XH, Huang Y, Li JB. Genetic diversity analyses of antimicrobial resistance genes in clinical *Chryseobacterium meningosepticum* isolated from Hefei, China. J Antimicrob Agents. 2012; 40(2):186–8. Epub 2012/05/23. https://doi.org/10.1016/j.jamia.2012.03.020 PMID: 22612901.

79. Matsumoto T, Nagata M, Ishimine N, Kawasaki K, Yamauchi K, Hidaka E, et al. Characterization of CIA-1, an Ambler class A extended-spectrum beta-lactamase from *Chryseobacterium indologenes*. Antimicrob Agents Chemother. 2012; 56(1):588–90. Epub 2011/11/16. https://doi.org/10.1128/AAC.05165-11 PMID: 22083470; PubMed Central PMCID: PMCPMC3256067.

80. Li XZ, Plesiat P, Nikaido H. The challenge of efflux-mediated antibiotic resistance in Gram-negative bacteria. Clin Microbiol Rev. 2015; 28(2):337–418. Epub 2015/03/20. https://doi.org/10.1128/CMR.00117-14 PMID: 25788514; PubMed Central PMCID: PMCPMC4402952.

81. Poole K. Efflux-mediated antimicrobial resistance. J Antimicrob Chemother. 2005; 56(1):20–51. Epub 2005/05/26. https://doi.org/10.1093/jac/dki171 PMID: 15914491.

82. Du D, van Veen HW, Murakami S, Pos KM, Luisi BF. Structure, mechanism and cooperation of bacterial multidrug transporters. Curr Opin Struct Biol. 2015; 33:76–91. Epub 2015/08/19. https://doi.org/10.1016/j.sbi.2015.07.015 PMID: 26282926.

83. Lubelski J, Konings WN, Driessen AJ. Distribution and physiology of ABC-type transporters contributing to multidrug resistance in bacteria. Microbiol Mol Biol Rev. 2007; 71(3):463–76. Epub 2007/09/07. https://doi.org/10.1128/MMBR.00001-07 PMID: 17804667; PubMed Central PMCID: PMCPMC2186643.

84. Pao SS, Paulsen IT, Saier MH Jr. Major facilitator superfamily. Microbiol Mol Biol Rev. 1998; 62(1):1–34. Epub 1998/04/08. PMID: 9529885; PubMed Central PMCID: PMCPMC89004.

85. Saier M.H.Jr., Beatty JT, Goffaeu A, Harley KT, Heijne WH, Huang SC, et al. The major facilitator superfamily. J Mol Microbiol Biotechnol. 1999; 1(2):257–79. Epub 2000/08/16. PMID: 10943556.

86. Kuroda T, Tsuchiya T. Multidrug efflux transporters in the MATE family. Biochim Biophys Acta. 2009; 1794(5):763–8. Epub 2008/12/23. https://doi.org/10.1016/j.bbabap.2008.11.012 PMID: 19100867.

87. Nikaido H. Structure and mechanism of RND-type multidrug efflux pumps. Adv Enzymol Relat Areas Mol Biol. 2011; 77:1–60. Epub 2011/06/23. PMID: 21692366; PubMed Central PMCID: PMCPMC3122131.

88. Chung YJ, Saier MH Jr. SMR-type multidrug resistance pumps. Curr Opin Drug Discov Devel. 2001; 4(2):237–45. Epub 2001/05/31. PMID: 11379893.

89. Fralick JA. Evidence that TolC is required for functioning of the Mar/ArCAB efflux pump of *Escherichia coli*. J Bacteriol. 1996; 178(19):5803–5. Epub 1996/10/01. PMID: 8824631; PubMed Central PMCID: PMCPMC178425.

90. Lin J, Michel LO, Zhang Q. CmeABC functions as a multidrug efflux system in *Campylobacter jejuni*. Antimicrob Agents Chemother. 2002; 46(7):2124–31. Epub 2002/06/19. https://doi.org/10.1128/AAC.46.7.2124-2131.2002 PMID: 12069964; PubMed Central PMCID: PMCPMC127319.

91. Okusu H, Ma D, Nikaido H. AcrAB efflux pump plays a major role in the antibiotic resistance phenotype of *Escherichia coli* multiple-antibiotic-resistance (Mar) mutants. J Bacteriol. 1996; 178(1):306–8. Epub 1996/01/01. PMID: 8504539; PubMed Central PMCID: PMCPMC177656.

92. Poole K, Krebes K, McNally C, Neshat S. Multiple antibiotic resistance in *Pseudomonas aeruginosa*: evidence for involvement of an efflux operon. J Bacteriol. 1993; 175(22):7363–72. Epub 1993/11/01. PMID: 8226684; PubMed Central PMCID: PMCPMC2068881.
Similarity of Elizabethkingia anophelis isolated from horses to three human clinical isolates