BMC Genomics

RESEARCH

Open Access

Genomic insights and epidemiology of *mcr-1*-Carrying *Escherichia albertii* isolated from agricultural soil in China

Yu Yang[1†], Wenhong Liu[1†], Zanzan Zhao[1], Kexin Guo[1], Xinrui Wang[2], Zhenghao Lou[1], Xiaolu Yang[1], Lu Gong[1], Kun Wang[1], Xiaojing Liu[3], Hao Xu[4], Qiyu Liu[1], Beiwen Zheng[4*] and Xiawei Jiang[1*]

Abstract

Background Polymyxins are critical in treating multidrug-resistant Gram-negative bacteria infections, yet their overuse has spurred the emergence of polymyxin-resistant pathogens globally. This study aims to analyze the genomic characteristics of the *Escherichia albertii* strain 6S-65-1 carrying the *mcr-1* gene and to investigate the global epidemiology of *mcr-1*-carrying *E. albertii* strains.

Results In this study, we identified and analyzed a polymyxin-resistant *Escherichia albertii* strain (6S-65-1) carrying the *mcr-1* gene, isolated from agricultural soil in China. Whole-genome sequencing and comparative genomic analyses revealed two chromosomal integrations of the *mcr-1* gene within Tn6330 transposon structures, indicating its capacity for horizontal gene transfer. Strain 6S-65-1 also harbors other antimicrobial resistance genes, including *tet(A), sul3,* and *aph (3')-Ia*, enhancing its resistance profile. Comparative genomic analysis of *E. albertii* genomes in the NCBI database revealed that *mcr-1*-carrying *E. albertii* strains are geographically restricted to China and Japan, and have been isolated from both animals and humans. Phylogenetic analysis revealed that strain 6S-65-1 was most closely related to a human-derived strain from Japan (SAMD00164101), with both strains carried virulence genes (*cdt*, *paa*, and *eae*) that enable them to form attaching and effacing (A/E) lesions. Among all publicly available ST4619 *E. albertii* genomes, strain 6S-65-1 is the first to carry the *mcr-1* gene.

Conclusion Our findings offer new insights into the epidemiology and genomic features of *mcr-1*-carrying *E. albertii*, underscoring the need for targeted management strategies to curb its spread. These findings underscore the importance of "One Health" approaches to antimicrobial resistance, which require coordinated efforts across human, animal and the environmental health sectors.

Keywords *Escherichia albertii*, Epidemiology, Whole-genome sequencing, *mcr-1*, Farm environment

[†]Yu Yang and Wenhong Liu contributed equally to this work.

*Correspondence:
Beiwen Zheng
zhengbw@zju.edu.cn
Xiawei Jiang
xiaweijiang@zcmu.edu.cn
Full list of author information is available at the end of the article

Background

Polymyxins are considered the last line of defense against serious infections caused by multi-resistant Gram-negative bacteria [1]. However, the overuse of polymyxins in veterinary and clinical settings has led to the global emergence of polymyxin-resistant pathogens [2]. The emergence and widespread dissemination of mobile colistin resistance genes (*mcr*) pose significant challenges to animal health, environment, and public health. Since *mcr-1* gene was first reported in *Escherichia coli* and *Klebsiella pneumoniae* from human and animal in China, it has been identified in other members of the Enterobacteriaceae family worldwide, such as *Pseudomonas aeruginosa*, *K. oxytoca*, and *Citrobacter freundii*, indicating that *mcr-1* gene has become prevalent globally [1, 3]. This issue has garnered extensive international attention.

E. albertii, identified in 2003, is a new member of genus *Escherichia* and an emerging zoonotic enteric pathogen [4]. Infections caused by *E. albertii* can lead to severe complications and pose a significant threat to public health. Historically, this bacterium was often misidentified due to detection and identification challenges, but advances in genomics now allow for accurate identification. Several outbreaks of foodborne *E. albertii* infections have been reported in Japan, with symptoms including diarrhea, abdominal pain, vomiting and fever [5–8]. Additionally, sporadic cases have been reported in countries such as Brazil, the United States and Nigeria [9–11]. To date, two cases of bacteremia caused by *E. albertii* infection have been reported [12, 13]. Furthermore, mass mortality events in other species, such as the common redpoll (*Carduelis flammea*) in the United States and Scotland has also been associated with this pathogen [14, 15]. *E. albertii* has been isolated from various sources, including migratory birds, raw meat, water, and raccoons [16–20]. These data demonstrate that the emergence and spread of *E. albertii* infections are global concerns. Although the transmission mechanism of *E. albertii* is not fully understood, its ability to exist in multiple hosts and environments suggests that it is highly adaptive and transmissible.

Despite these findings, there is still limited knowledge about the epidemiology, antimicrobial resistance and pathogenic potential of *E. albertii*. Recent studies have found the coexistence of genes encoding β-lactamase and *mcr-1* in *E. albertii* from animal and animal meat products in China [21]. Here, we performed whole-genome sequencing of an *E. albertii* strain 6S-65-1 isolated from farm soil using the Oxford Nanopore and Illumina NovaSeq platforms to analyze its antimicrobial resistance and genomic characteristics. In addition, we analyzed all the 651 genome sequences of *E. albertii* publicly available in NCBI database to determine the global distribution of *E. albertii* strains and the prevalence of *mcr-1*-carrying strains.

Methods

Bacterial isolation and identification, detection of *mcr-1* gene

In May 2019, a study aimed at detecting antimicrobial resistance in farm environments was conducted, collecting 976 samples from six randomly selected farms in Jiaxing City, Zhejiang Province, China. This included 12 stool samples from farmers, 769 stool samples from various animals (cattle, sheep, and pigs), and 195 swab samples from the environment (including dustbin, floor, table, door handle, soil, water, fodder and vegetables) as previously described [22]. All samples were kept at −20 ℃ during transportation and subsequently transferred to Mueller-Hinton broth (MHB) for enrichment at 37 ℃ within 72 h after sampling. The enriched samples were cultured on MacConkey agar (OXOID, Hampshire, United Kingdom) plates containing 2 μg/mL colistin and incubated at 37 ℃ for 24 h. Individual colonies with different morphologies were selected and further incubated at 37 ℃ for 24 h on Mueller-Hinton agar (MHA) to obtain pure strains. The bacterial species were identified using matrix-assisted laser desorption/ionization time-of-flight mass spectrometry (MALDI-TOF/MS) (Bruker Daltonik GmbH, Bremen, Germany), and further confirmed by Average nucleotide identity (ANI) and digital DNA-DNA hybridization (dDDH) comparisons. All purified strains were stored long-term in 20% glycerol cryogenic vials at -80 ℃. Additionally, all strains were subjected to polymerase chain reaction (PCR) and Sanger sequencing to detect the *mcr-1* gene, using primers *mcr-1*-F, CGCCATATG TGCAGCATACTTCTGTGTGG, and *mcr-1*-R, CCG CTCGAGGGTGCGGTCTTTGACTTTG.

Antimicrobial susceptibility testing

Antimicrobial susceptibility testing for strain 6S-65-1 was performed using both agar and broth microdilution. The results for tigecycline, omadacycline and eravacycline were defined according to FDA-defined breakpoints(https://www.fda.gov/drugs/development-resources/antibacterial-susceptibility-test-interpretive-criteria), while the remaining antimicrobials were interpreted according to Clinical and Laboratory Standards Institute (CLSI) guidelines(https://clsi.org/). To ensure the reliability of antimicrobial susceptibility testing, *E. coli* ATCC 25922 was used as a quality control standard.

Whole-genome sequencing and bioinformatics analysis

To characterize the genetic features of the *mcr-1*-positive strain 6S-65-1, whole-genome sequencing was conducted. Genomic DNA of strain 6S-65-1 was extracted using a Bacterial DNA Kit (QIAGEN, Hilden, Germany) and sequenced on Oxford Nanopore (Oxford Nanopore Technologies, Oxford, UK) and Illumina NovaSeq 6000 (Illumina, San Diego, CA, USA) platforms. The sequencing data was hybrid-assembled using Unicycler v0.4.7 [23], followed by genome annotation with Prokka v1.17(https://github.com/tseemann/prokka). The digital DNA-DNA hybridization(dDDH) was performed using the Genome-to-Genome Distance Calculator 3.0 online tool (https://ggdc.dsmz.de/ggdc.php/). Multilocus sequence typing (MLST) and antimicrobial resistance genes were identified using the Center for Genomic Epidemiology (CGE) platform (https://genomicepidemiology.org/services/). Virulence genes were identified using both the VFDB database(http://www.mgc.ac.cn/cgi-bin/VFs/v5/main.cgi) and CGE's virulenceFinder database(https://cge.food.dtu.dk/services/VirulenceFinder/). The genetic context surrounding the *mcr-1* gene was analyzed and visualized using Easyfig v2.2.5.

Phylogenetic Tree Construction and Comparative Genomic Analysis

Global Distribution of 652 E. albertii Strains

To evaluate the global distribution characteristics of *E. albertii* strains, a total of 651 publicly available genome sequences were retrieved and downloaded from the National Center for Biotechnology Information (NCBI) database(https://www.ncbi.nlm.nih.gov/pathogens/), accessed on March 24, 2024. The specific characteristics of these isolates are shown in Table S1. Genome sequence-based average nucleotide identity (ANI) analysis was performed for all 651 isolates, together with strain 6S-65-1 in this study using pyani v0.2.12(https://github.com/widdowquinn/pyani) to exclude confounding strains. Building on this, alignment files were generated using Prokka and Roary(https://github.com/sanger-pathogens/Roary). A maximum likelihood (ML) phylogenetic tree was constructed using FastTree and visualized using the online tool iTOL(https://itol.embl.de/). The geographic distribution and sources of the 652 *E. albertii* strains were analyzed using a customized world map dataset generated with the "ggplot2" package in R version 4.4.1.

Global Distribution of mcr-1-positive E. albertii Strains

To identify the presence of the *mcr-1* gene in these strains, the abricate v1.0.1 tool (https://github.com/tseemann/abricate) was used with the Resfinder database. For *mcr-1*-positive *E. albertii* strains, a phylogenetic tree was constructed. Additionally, the antimicrobial resistance genes and the virulence factors for each strain were visualized using heatmaps.

Global Distribution of ST4619 E. albertii Strains

To quickly obtain the ST types for all 652 strains, we used mlst v2.23.0 tool (https://github.com/tseemann/mlst) to analyze these genome data. The phylogenetic tree was then constructed for ST4619 *E. albertii* strains. The antimicrobial resistance genes were visualized using a heatmap. The geographic distribution of countries harboring *mcr-1* gene and ST4619 *E. albertii* strains was analyzed using world maps in R v4.4.1.

Serum Killing Assay

To evaluate the survival of strain 6S-65-1 in healthy human serum, a serum killing assay was performed with slight modifications based on the previously established protocol [24]. Blood samples were collected from healthy volunteers (research laboratory colleagues) after obtaining informed consent. The study was conducted in accordance with institutional ethical guidelines. No personal identifying information was included in the study, and all procedures were voluntary. The blood was centrifuged at 4000 rpm for 10 min to obtain serum. The serum was then divided into two portions: one was incubated at 56 °C for 30 min to inactivate complement, while the other remained untreated as normal serum. Bacterial cultures from overnight growth were adjusted to a turbidity of 0.5 McFarland standard (1×10^8 CFU/mL) and then diluted 1:100 with saline to reach 1×10^6 CFU/mL. 20 µL of the bacterial suspension was mixed with 180 µL of either inactivated or normal serum and incubated at 37 °C for 1 h. After incubation, 50 µL of the mixture was plated onto Mueller-Hinton agar, and colony-forming units (CFUs) were counted after 18-20 h of incubation. Statistical analysis was performed using the unpaired t-test with GraphPad Prism v8.0. Each experiment was conducted in triplicate. *E. coli* ATCC 25922 served as low-virulence control.

Results

Identification and antimicrobial resistance profile of *E. albertii* 6S-65-1

A total of 96 strains carrying *mcr-1* were detected through PCR screening in this study (Fig. 1). High-throughput ANI analysis was performed on these strains based on whole-genome sequencing results to confirm distinct species within the *Escherichia* family (Fig. S1). *E. coli* ATCC 25922 was used as the standard strain for comparison in this analysis. Among them, a colistin-resistant *E. albertii* strain was discovered and named

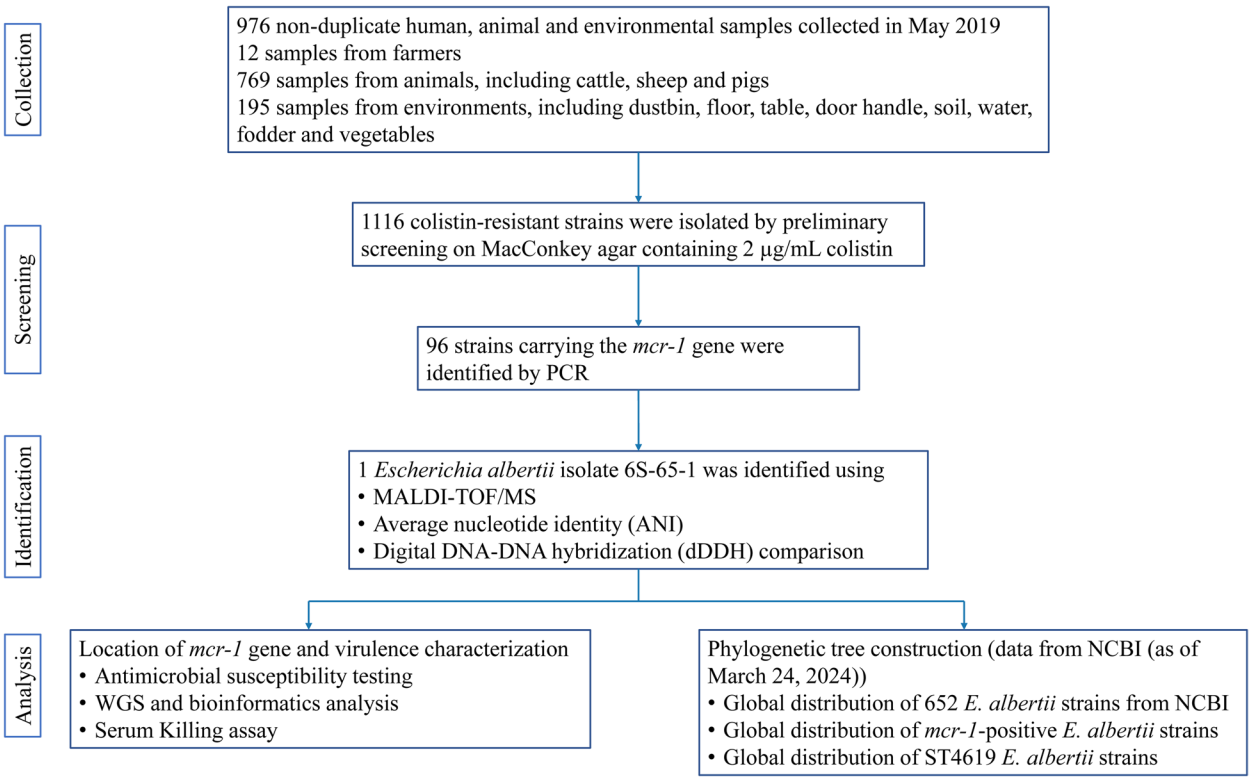


Fig. 1 Schematic representation of the workflow used in this study, which is divided into four main stages: sample collection, strain screening, strain identification, and data analysis

as 6S-65-1. To further confirm its species classification, digital DNA-DNA hybridization (dDDH) was conducted to compare strain 6S-65-1 with other members of the *Escherichia* genus, including *Escherichia fergusonii*, *E. coli*, *Escherichia marmotae*, *Escherichia ruysiae*, *Escherichia whittamii*, and *E. albertii*. The results revealed that the DDH value between strain 6S-65-1 and *E. albertii* was 88.50% [86–90.6%], exceeding the 70% threshold for species identification (Fig. 2). The antimicrobial resistance profile of strain 6S-65-1 was evaluated, and the minimum inhibitory concentration (MIC) values are listed in Table 1.

Comprehensive genome characterization of *E. albertii* 6S-65-1

The complete genomic characteristics of *E. albertii* 6S-65-1 are shown in Table 2. The genome consists of a circular chromosome of 4,889,721 bp and two plasmids with an average GC content of 49.7%. Multilocus sequence typing revealed that the strain belonged to ST4619 type and harbored eight different antimicrobial resistance genes, including *aph (3')-la*, *aadA1*, *aadA2b*, *mcr-1.1*, *cmlA1*, *sul3*, *tet (A)*, and *bleO*. Notably, all genes were located on the chromosome, except for *tet (A)* and *bleO*, which were located on plasmid1. Genetic environment analysis revealed that the *mcr-1* gene was integrated into two different segments of the chromosome by the composite transposon Tn6330, which consists of an IS*Apl1*-*mcr-1.1*-IS*Apl1* frame (Fig. 3).

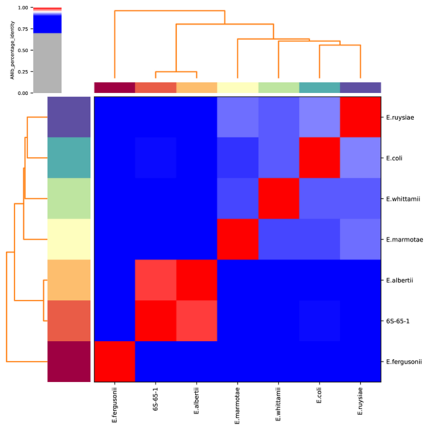
Phylogenetic and core-genome analysis

Global distribution of E. albertii strains

ANI analysis showed that genomic data were available for all 652 *E. albertii* strains (Fig. S2). Phylogenetic tree results showed that these strains were identified in 18 different countries, with a significant prevalence in Asia and Europe, especially in China and the UK. The geographic origin of 22 strains remains unclear. Regarding their sources, there were 152 isolates from humans, 382 from animals, 22 from the environment, and 74 from unknown sources. Among them, the largest number of human-derived strains were from the UK, totaling 109, while animal-derived and environment-derived strains were mainly identified in China, with 189 and 19 strains, respectively. For further details, refer to Fig. 4.

To further investigate the relationships within the clade containing strain 6S-65-1, we extracted all 124 strains from this cluster and constructed an independent phylogenetic tree (Fig. 5a). The strains in this cluster are widely distributed across six continents, only four

a



b

Query genome	Reference genome	DDH	Model C.I.
6S-65-1	*Escherichia fergusonii* (SAMN21218855)	34.2	[31.8 - 36.7%]
	Escherichia coli (SAMN01911278)	40.9	[38.5 - 43.5%]
	Escherichia marmotae (SAMN18536562)	38.8	[36.3 - 41.3%]
	Escherichia ruysiae (SAMN37199584)	39.9	[37.4 - 42.4%]
	Escherichia whittamii (SAMN15803250)	38.7	[36.2 - 41.2%]
	Escherichia albertii (SAMN33094113)	88.5	[86.0 - 90.6%]

All reference genome from NCBI

Fig. 2 **a** Heatmap generated by pyani. Red indicates high genome similarity, while blue represents lower genome similarity. **b** dDDH values of strain 6S-65-1 with other members of the *Escherichia* genus

of which are of environmental origin. Strain 6S-65-1 is closely related to two Chinese strains, one of human origin (GCA_032538345.1) and one of animal origin (GCA_032538045.1). Notably, only strain 6S-65-1, which is of environmental origin, carries the *mcr-1* gene. The most common ST types in this cluster are 4638, 4619, and 5431. Of these, ST4638 is predominantly identified in China, ST4619 is predominantly identified in Switzerland, and all of ST5431 is identified in the UK. Common resistance genes include aminoglycosides, sulfonamides, and tetracyclines, which are prevalent in animal sources. Notably, the virulence genes *iutA*, *iucA/B/C/D* are mainly found in the ST5431 strains from human sources and ST4619 strains from animal sources. This suggests that these virulence genes may be shared between different hosts, potentially affecting their pathogenicity.

Table 1 Antimicrobial resistance profile of *E. albertii* strain 6S-65-1

Antibiotics	MIC(μg/mL)	antimicrobial susceptibility
Aztreonam	0.125	S
Imipenem	0.25	S
Meropenem	0.03	S
Ceftriaxone	≤ 0.03	S
Cefotaxime	0.06	S
Ceftazidime	0.125	S
Levofloxacin	0.25	S
Ciprofloxacin	0.06	S
Amikacin	1	S
Gentamicin	0.5	S
Piperacillin-Tazobactam	4	S
Fosfomycin-D-glucose-6	1	S
Chloramphenicol	64	R
Trimethoprim-Sulfamethoxazole	0.25	S
Amoxicillin-Clavulanate	8	S
Cefepime	0.03	S
Ceftazidime-Avibactam	0.06	S
Tigecycline	0.125	S
Colistin	4	R
Polymyxin B	4	R
Omadacycline	0.25	S
Eravacycline	0.125	S

MIC minimum inhibitory concentration, *S* sensitive, *R* resistant

Global distribution of *mcr-1*-positive *E. albertii* strains

All these genomes were screened for the presence of the *mcr-1* gene, and only six strains were confirmed to carry it. A phylogenetic tree was constructed based on the core genome of the six strains (Fig. 5c). The results showed that all the *mcr-1*-positive *E. albertii* strains were located in China and Japan (Fig. 5b), while the strain 6S-65-1 in the present study was closely related to a human isolated strain in Japan (SAMD00164101). Additionally, all these six strains were not isolated from environment except strain 6S-65-1. Comparison of resistance gene among these strains revealed that the six *E. albertii* strains predominantly harbored *tet(A)* and *mdf(A)* gene (100%), followed by *floR* gene (83.3%). VFDB results showed that strain 6S-65-1 lacks type I fimbriae genes (*fimA/C/E/F/G/H/I*), compared to other strains.

Global distribution of ST4619 *E. albertii* strains

To characterize the geographic distribution patterns of *E. albertii* strains of same sequence type with strain 6S-65-1 (ST4619) worldwide, a comparative genome analysis of publicly available ST4619 *E. albertii* genomes (n = 16) was performed. The results showed that strains of ST4619 originated from six countries, primarily Switzerland

Table 2 Genomic features of *E. albertii* 6S-65-1

Feature	Chromosome	plasmid1	plasmid2
Total number of bases (bp)	4,889,721	180,516	3,293
GC content (%)	49.8	48.8	47.9
Circular	Yes	Yes	No
Number of coding sequences	4948	241	3
Number of RNAs	116	0	0
Plasmid replicon type	-	IncFIA/IncFIB	IncN
Resistance genes	*mcr-1.1, aph(3')-Ia, aadA1 aadA2b, cmlA1, sul3*	*tet(A), bleO*	
Accession numbers	CP141901	CP141902	CP141903

and China, with animals serving as the primary hosts (Fig. 5d). Notably, the *mcr-1* gene was absent in all other strains of ST4619, except strain 6S-65-1 in this study.

Virulence assessment of strain 6S-65-1

The serum complement system serves as a primary defense against bacterial invasion. In the serum killing assay, strain 6S-65-1 exhibited nearly 100% survival, indicating strong resistance to serum killing. In contrast, *E. coli* ATCC 25922 showed a lower survival rate of approximately 70-80%, consistent with its designation as low-virulence control strain. Statistical analysis revealed a significant difference in survival between the two strains ($P < 0.05$, see Fig. S3).

Discussion

Antimicrobial resistance remains a critical global health challenge, exacerbated by the overuse of polymyxins in clinical setting, which has led to the rise of colistin resistance through the *mcr* gene family [25, 26]. *E. albertii*, first isolated in 1991 from stool samples of children with diarrhea in Bangladesh [27], has seen a notable increase in infection cases worldwide [8, 12, 13, 28, 29]. However, knowledge regarding its epidemiology, antimicrobial resistance and genomic profiles remains limited. This study marks the first report of the *mcr-1* gene in ST4619 *E. albertii*, expanding our understanding of *mcr-1* in environmental isolates.

In this study, fecal and environmental samples from farmers and animals across various farms were collected and cultured following the "One Health" approach. An *E. albertii* strain, designated 6S-65-1 carrying the *mcr-1* gene, was isolated from a soil sample and underwent whole-genome sequencing. Multilocus sequence typing identified strain 6S-65-1 as belonging to ST4619. Interestingly, previously reported *E. albertii* strains carrying the *mcr-1* gene in China were associated to ST4479 [21]. Genetic environment analysis revealed that both the upstream and downstream regions of the *mcr-1* gene contained the insertion sequence IS*Apl1*, forming the composite transposon element Tn6330 [30, 31]. This finding suggests that Tn6330 serves as the primary vector for chromosome-mediated *mcr-1* gene transfer.

We performed a phylogenetic tree analysis of all available *E. albertii* strains in the NCBI database to examine their global geographic distribution. The results showed that each phylogenetic branch contained strains from multiple countries and sources, highlighting substantial geographic diversity. China and the UK emerged as the countries with the most extensive strain distribution. Most strains originated from animal sources, with only 22 environmental isolates identified. Of these, 19 were from China, while the remaining were from Poland, Bangladesh and Germany. These environmental isolates were distributed across various phylogenetic branches.

We constructed a separate phylogenetic tree for the clade containing strain 6S-65-1, revealing substantial genetic diversity among various ST types within this clade. These strains harbored multiple resistance genotypes, including those conferring resistance to polymyxins, aminoglycosides, beta-lactams, chloramphenicol, sulfonamides, trimethoprim, fosfomycin, macrolides, quinolones, and tetracyclines. Additionally, a diverse array of virulence genes were detected, encompassing genes associated with intestinal pathogenicity (*eae*, *escN*, *espA*, *tir*), iron uptake (*iucA/B/C/D*, *iutA*), toxins (*astA*, *cdtB*, *hlyA*, *pet*), adhesion (*fimH*, *ompA*, *paa*), and invasiveness (*ibeA*). Notably, the iron acquisition genes *iucA/B/C/D* and *iutA* were present not only in human- and animal-derived ST4619 strains but also in the environmentally derived strain 6S-65–1 identified in this study. This suggests that these strains are highly adaptable and may possess the potential for cross-species transmission. Compared to the other three environmental isolates in this clade, strain 6S-65-1 carried several additional antibiotic resistance genes, including those conferring

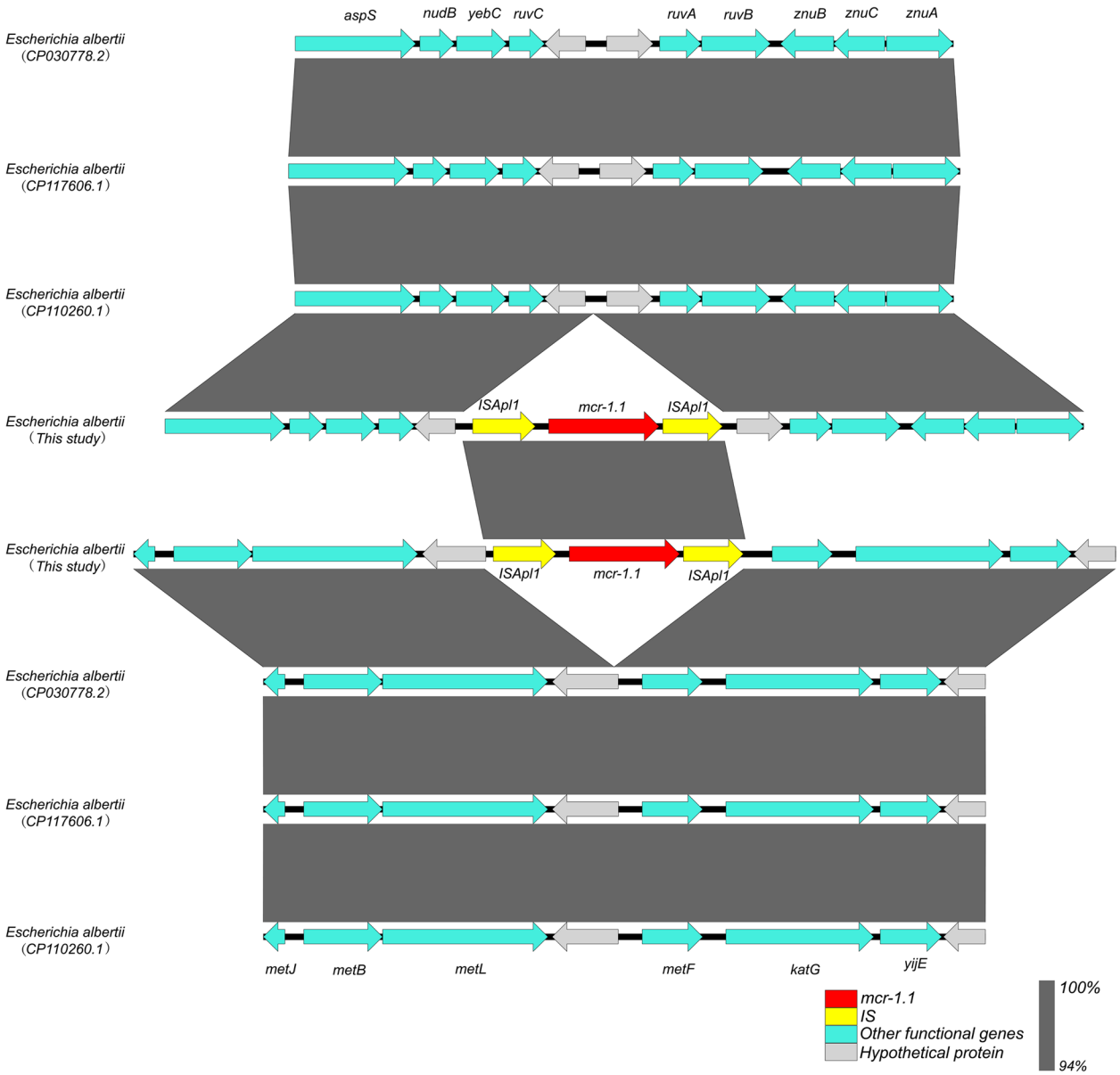


Fig. 3 Genetic environment of two *mcr-1.1* genes located in different chromosomal fragments of strain 6S-65-1. Red arrows represent the *mcr-1* gene, yellow arrows represent mobile genetic elements, and blue and gray arrows represent other functional genes and hypothetical proteins, respectively

resistance to aminoglycosides, tetracyclines and sulfonamides, in addition to the *mcr-1* gene. Moreover, strain 6S-65-1 carried a wider range of virulence genes, such as the iron acquisition genes (*iucA/B/C/D*, *iutA*), which were not found in the other environmental isolates. These additional genetic features highlight the potential for strain 6S-65-1 to be more virulent and resistant than other environmental strains, reinforcing the possibility that it could serve as a source of cross-species transmission with implications for both animal and human health.

Phylogenetic analysis of the genome sequences of the six *E. albertii* strains carrying the *mcr-1* gene, publicly available in NCBI database, showed that *mcr-1*-carrying *E. albertii* strains have only been reported in China and Japan. Strain 6S-65-1 was closely related to a human-derived strain from human in Japan (SAMD00164101). In addition to the *mcr-1* gene, these strains carry genes conferring resistance to aminoglycosides (*aac (3)-IId*, *aac (3)-Iva*, *aadA2*, *ant (3")-Ia*, *aph (3")-Ib*, *aph (3')-Iia*, *aph(3')-Ia*, *aph(4)-Ia*, *aph(6)-Id*), extended-spectrum

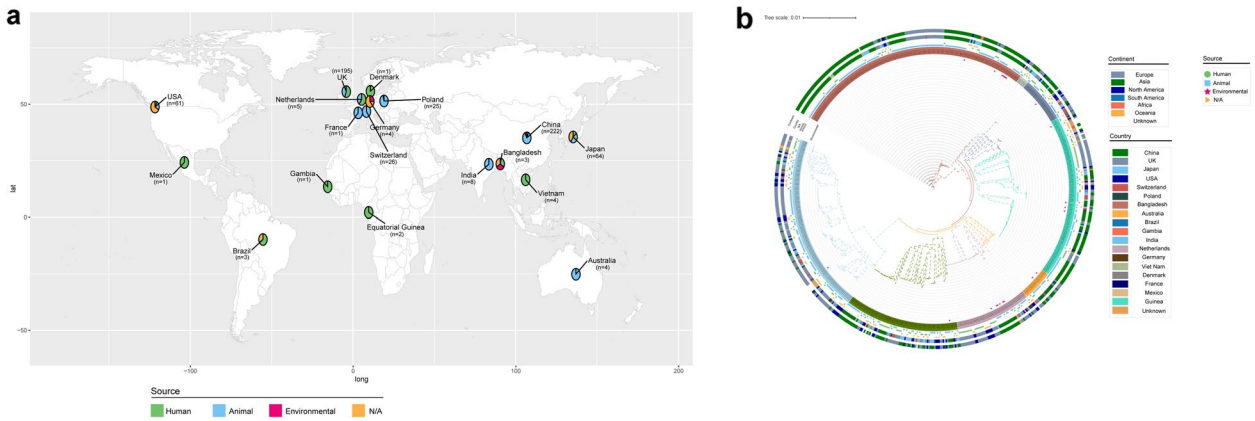


Fig. 4 Overview of the geographical distribution and source types of the 652 *E. albertii* strains. **a** The map shows pie charts for 18 countries, illustrating the distribution of human-derived(green), animal-derived(blue), environment-derived(pink), and unknown source(orange) strains. **b** The phylogenetic tree shows clustering patterns and geographical distribution of these strains. The shaded colors represent different branches, with different shapes from inner to outer layers indicating different sources: pink pentagons represent environment sources, blue squares represent animal sources, green circles represent human sources, and orange triangles represent unknown sources. The concentric rings, from inner to outer layers, represent different countries and states

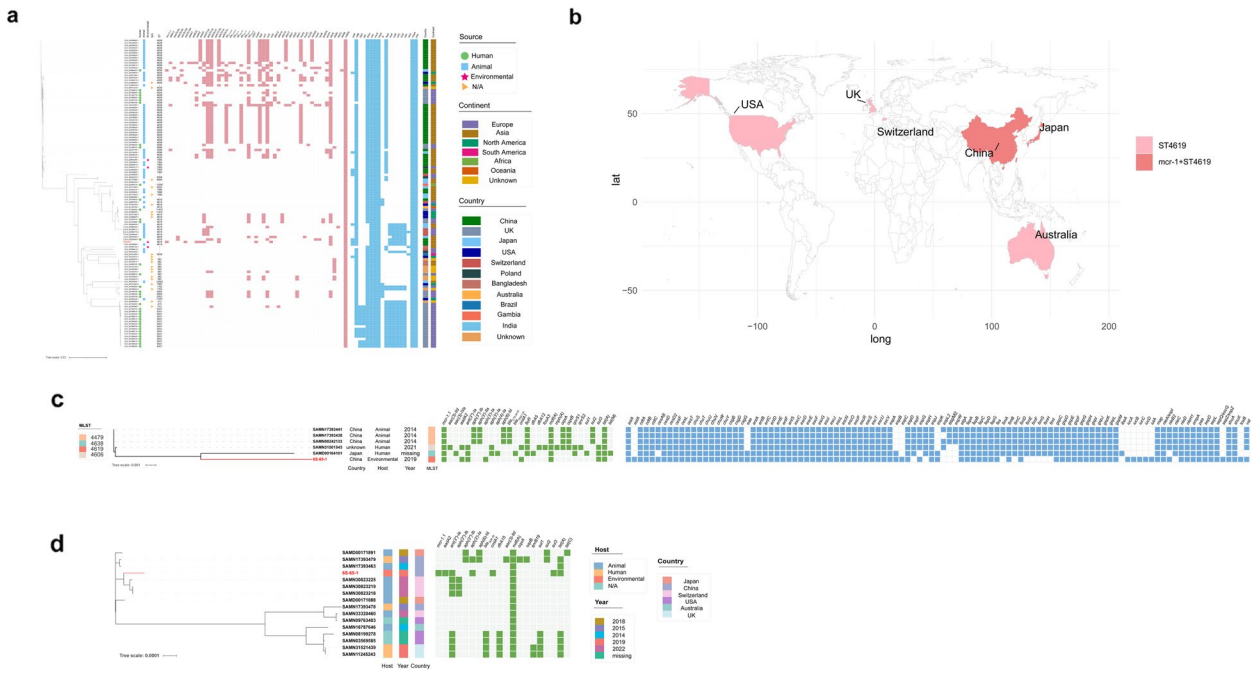


Fig. 5 **a** A phylogenetic analysis of a distinct clade containing 6S-65-1 strain, extracted from the 652 *E. albertii* strains. Red indicates the presence of resistance genes, and blue indicates the presence of virulence genes. **b** Geographic distribution of countries harboring the *mcr-1* gene and ST4619 *E. albertii* strains. Deep red represent ST4619 strains carrying the *mcr-1* gene, while pink represent ST4619 strains without the *mcr-1* gene. **c** Phylogenetic tree and distribution *mcr-1*-positive *E. albertii* strains. Green indicates the presence of resistance genes and blue indicates the presence of virulence genes. **d** Phylogenetic tree and distribution of *E. albertii* strains with the same ST type

beta-lactams ($bla_{\text{CTX-M-55}}$, $bla_{\text{TEM-135}}$), choramphenicols (*cmlA1*, *floR*), quinolones (*oqxA*, *oqxB*, *qnrS1*, *qnrS2*), sulfonamides (*sul1*, *sul2*, *sul3*), and tetracyclines (*tet(A)*, *tet(M)*), indicated their multi-drug resistance potential. Additionally, all strains possessed genes encoding cytolethal distending toxin (*cdt*), composed of *cdtA*, *cdtB*, and *cdtC*, associated with long-term bacterial colonization, invasion and increased disease severity [19], and the porcine attaching and effacing-associated toxin (*paa*), involved in early stages of virulence

[32]. Both 6S-65-1 and SAMD00164101 possessed the *eae* gene which encods intimin, a protein essential for forming A/E lesions. Together, these genes (*cdt*, *paa*, and *eae*) play critical roles in the pathogenic potential of *E. albertii* strain 6S-65-1. Furthermore, unlike the other five *mcr-1*-carrying *E. albertii* strains, 6S-65-1 lacks type I fimbriae genes, which are typically associated with bacterial adhesion and may affect its colonization and infection ability. However, 6S-65-1 also carries other virulence genes related to pathogenicity (*chuU/V/S/W/Y*, *iutA*, and *iucA/B/C/D*), which are associated with extraintestinal infections, suggesting that the strain may maintain its pathogenicity through different mechanisms. Serum killing assays indicated that 6S-65-1 has a higher resistance, suggesting an enhanced ability to evade immune responses. This immune evasion capability may contribute to its persistence in animal or human hosts. Overall, these findings indicate that strain 6S-65-1 has notable pathogenic potential for both animals and humans.

Comparative genomic analysis of all the fifteen publicly available ST4619 *E. albertii* strains in the NCBI database and strain 6S-65-1 revealed that ST4619 strains have been identified in both animal and human hosts. Strain 6S-65-1 showed close genetic relatedness to three animal-derived strains (SAMN30823225, SAMN30823219 and SAMN30823218) from Switzerland. However, none of these fifteen ST4619 strains carried the *mcr-1* gene, making this study the first to identify the *mcr-1* gene in an ST4619 *E. albertii* strain. Previous research has shown that *E. albertii* can colonize healthy wild birds and other animals [16, 33, 34], suggesting that strain 6S-65-1, isolated from farm soil, may spread through the soil, water, or food chain, potentially becoming a source of infection for humans and animals. These findings highlight the importance of environmental surveillance in tracking antimicrobial resistance (AMR), as environmental reservoirs may serve as sources for the spread of resistant pathogens. Regular environmental monitoring, particularly in agricultural and animal-associated environments, could be crucial for early detection of AMR threats.

Our study has several limitations. First, the sample size is small, with only one strain included, and the research is primarily focused on a specific geographic region, which may limit the validity of the data in China. Secondly, there is a lack of additional samples to support direct associations between different transmission routes (such as water, soil or the food chain) and *mcr-1*-carrying *E. albertii* strains. Therefore, future studies should aim to increase sample sizes and explore broader geographic regions and diverse sample sources to better assess potential environmental transmission pathways and the global prevalence of *mcr-1*-carrying *E. albertii* strains.

Conclusions

In conclusion, this study identified an *E. albertii* strain (6S-65-1) carrying the *mcr-1* gene, and conducted whole-genome sequencing and comprehensively analysis of its resistance genes and virulence factors. Additionally, we investigated the epidemiology of *E. albertii* strains to better understand the prevalence and distribution of this pathogen. Our findings indicate that farm environments play an important role in the transmission of *E. albertii*, providing new insights into its epidemiologic and genomic characteristics. The presence of the *mcr-1* gene in this ST4619 strain suggests potential transmission to animals and humans via water or the food chain, highlighting the need for prevention and control measures by health and agricultural authorities. To manage the spread of the *mcr-1* gene, it is critical to implement regular environmental surveillance in agricultural and animal-associated environments, strengthen antimicrobial stewardship programs, and foster global collaboration to monitor and control the spread of resistant strains effectively.

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12864-025-11493-1.

Supplementary Material 1.

Supplementary Material 2.

Supplementary Material 3.

Supplementary Material 4.

Acknowledgements

Not applicable.

Authors' contributions

YY was the main contributor to writing the manuscript. YY, WL, and ZZ were responsible for figure creation and data analysis. KG, XW, QL, and ZL contributed to data visualization. XY, LG, KW, XL, and HX provided technical assistance. BZ oversaw project administration and funding acquisition. XJ revised the manuscript.

Funding

We gratefully acknowledge the financial support from the National Key R&D Program of China (2020YFE0204300); National Natural Science Foundation of China (82072314); Shandong Provincial Laboratory Project (SYS202202); Fundamental Research Funds for the Central Universities (2022ZFJH003).

Data availability

The complete genome sequence of strain 6S-65-1 is available at GenBank accession number SAMN39248548.

Declarations

Ethics approval and consent to participate

This study was approved by the Clinical Research Ethics Committee of the First Affiliated Hospital, Zhejiang University School of Medicine [number 2020-IIT-591] for human fecal sample collection. Informed consent was obtained from all participants prior to sample collection. All methods were performed in accordance with the relevant guidelines and regulations, including the Declaration of Helsinki. For animal fecal samples, only naturally excreted fecal samples from animals were collected, without direct interaction or

intervention. Informed consent was obtained from all animal owners prior to sample collection. As the sample collection was non-invasive and did not involve animal experimentation, additional ethical approval was not required.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

Author details
[1]School of Basic Medical Sciences, Zhejiang Chinese Medical University, Hangzhou, China. [2]College of Veterinary Medicine, China Agricultural University, Beijing 100193, People's Republic of China. [3]Shandong First Medical University and Shandong Academy of Medical Sciences, Jinan, China. [4]State Key Laboratory for Diagnosis and treatment of Infectious Diseases, Collaborative Innovation Center for Diagnosis and Treatment of Infectious Diseases, National Clinical Research Center for Infectious Diseases, National Medical Center for Infectious Diseases, the First Affiliated Hospital, Zhejiang University School of Medicine, Hangzhou, China.

Received: 24 December 2024 Accepted: 17 March 2025
Published online: 05 April 2025

References

1. Xiaomin S, Yiming L, Yuying Y, Zhangqi S, Yongning W, Shaolin W. Global impact of mcr-1-positive Enterobacteriaceae bacteria on "one health." Crit Rev Microbiol. 2020;46(5):565–77. https://doi.org/10.1080/1040841x.2020.1812510.
2. Xu L, Wan F, Fu H, Tang B, Ruan Z, Xiao Y, et al. Emergence of colistin resistance gene mcr-10 in enterobacterales isolates recovered from fecal samples of chickens, slaughterhouse workers, and a nearby resident. Microbiol Spectr. 2022;10(2): e0041822. https://doi.org/10.1128/spectrum.00418-22.
3. Liu YY, Wang Y, Walsh TR, Yi LX, Zhang R, Spencer J, et al. Emergence of plasmid-mediated colistin resistance mechanism MCR-1 in animals and human beings in China: a microbiological and molecular biological study. Lancet Infect Dis. 2016;16(2):161–8. https://doi.org/10.1016/s1473-3099(15)00424-7.
4. Bengtsson RJ, Baker KS, Cunningham AA, Greig DR, John SK, Macgregor SK, et al. The genomic epidemiology of Escherichia albertii infecting humans and birds in Great Britain. Nat Commun. 2023;14(1):1707. https://doi.org/10.1038/s41467-023-37312-3.
5. Asoshima N, Matsuda M, Shigemura K, Honda M, Yoshida H, Hiwaki H, et al. Identification of Escherichia albertii as a causative agent of a foodborne outbreak occurred in 2003. Jpn J Infect Dis. 2014;67(2):139–40. https://doi.org/10.7883/yoken.67.139.
6. Konno T, Yatsuyanagi J, Takahashi S, Kumagai Y, Wada E, Chiba M, et al. Isolation and identification of Escherichia albertii from a patient in an outbreak of gastroenteritis. Jpn J Infect Dis. 2012;65(3):203–7. https://doi.org/10.7883/yoken.65.203.
7. Masuda K, Ooka T, Akita H, Hiratsuka T, Takao S, Fukada M, et al. Epidemiological aspects of Escherichia albertii outbreaks in Japan and genetic characteristics of the causative pathogen. Foodborne Pathog Dis. 2020;17(2):144–50. https://doi.org/10.1089/fpd.2019.2654.
8. Ooka T, Tokuoka E, Furukawa M, Nagamura T, Ogura Y, Arisawa K, et al. Human gastroenteritis outbreak associated with Escherichia albertii. Japan Emerg Infect Dis. 2013;19(1):144–6. https://doi.org/10.3201/eid1901.120646.
9. Lima MP, Yamamoto D, Santos ACM, Ooka T, Hernandes RT, Vieira MAM, et al. Phenotypic characterization and virulence-related properties of Escherichia albertii strains isolated from children with diarrhea in Brazil. Pathog Dis. 2019;77(2); https://doi.org/10.1093/femspd/ftz014.
10. Lindsey RL, Rowe LA, Batra D, Smith P, Strockbine NA. PacBio genome sequences of eight Escherichia albertii strains isolated from humans in the United States. Microbiol Resour Announc. 2019;8(9): e01663-18. https://doi.org/10.1128/mra.01663-18.
11. Sulaiman MA, Aminu M, Ella EE, Abdullahi IO. Prevalence and risks factors of the novel Escherichia albertii among gastroenteritis patients in Kano State, Nigeria. J Med Trop. 2021;23(1):39–45. https://doi.org/10.4103/jomt.jomt_34_20.
12. Inglis TJ, Merritt AJ, Bzdyl N, Lansley S, Urosevic MN. First bacteraemic human infection with Escherichia albertii. New Microbes New Infect. 2015;8:171–3. https://doi.org/10.1016/j.nmni.2015.07.003.
13. Liu Q, Wang H, Zhang S, Yan G, Yang X, Bai X, et al. Escherichia albertii isolated from the bloodstream of a patient with liver cirrhosis in China: a case report. Heliyon. 2023;9(11): e22298. https://doi.org/10.1016/j.heliyon.2023.e22298.
14. Foster G, Ross HM, Pennycott TW, Hopkins GF, McLaren IM. Isolation of Escherichia coli O86:K61 producing cyto-lethal distending toxin from wild birds of the finch family. Lett Appl Microbiol. 1998;26(6):395–8. https://doi.org/10.1046/j.1472-765x.1998.00359.x.
15. Oaks JL, Besser TE, Walk ST, Gordon DM, Beckmen KB, Burek KA, et al. Escherichia albertii in wild and domestic birds. Emerg Infect Dis. 2010;16(4):638–46. https://doi.org/10.3201/eid1604.090695.
16. Liu Q, Bai X, Yang X, Fan G, Wu K, Song W, et al. Identification and genomic characterization of Escherichia albertii in migratory birds from Poyang Lake, China. Pathogens. 2022;12(1): 9. https://doi.org/10.3390/pathogens12010009.
17. Wang H, Zhang L, Cao L, Zeng X, Gillespie B, Lin J. Isolation and characterization of Escherichia albertii originated from the broiler farms in Mississippi and Alabama. Vet Microbiol. 2022;267: 109379. https://doi.org/10.1016/j.vetmic.2022.109379.
18. Hinenoya A, Nagano K, Awasthi SP, Hatanaka N, Yamasaki S. Prevalence of Escherichia albertii in Raccoons (Procyon lotor). Japan Emerg Infect Dis. 2020;26(6):1304–7. https://doi.org/10.3201/eid2606.191436.
19. Muchaamba F, Barmettler K, Treier A, Houf K, Stephan R. Microbiology and epidemiology of Escherichia albertii-An emerging elusive foodborne pathogen. Microorganisms. 2022;10(5): 875. https://doi.org/10.3390/microorganisms10050875.
20. Hinenoya A, Wang H, Patrick EM, Zeng X, Cao L, Li XP, et al. Longitudinal surveillance and comparative characterization of Escherichia albertii in wild raccoons in the United States. Microbiol Res. 2022;262: 127109. https://doi.org/10.1016/j.micres.2022.127109.
21. Li Q, Wang H, Xu Y, Bai X, Wang J, Zhang Z, et al. Multidrug-resistant Escherichia albertii: co-occurrence of β-lactamase and MCR-1 encoding genes. Front Microbiol. 2018;9: 258. https://doi.org/10.3389/fmicb.2018.00258.
22. Hu X, Chen Y, Xu H, Qiao J, Ge H, Liu R, et al. Genomic epidemiology and transmission characteristics of mcr1-positive colistin-resistant Escherichia coli strains circulating at natural environment. Sci Total Environ. 2023;882: 163600. https://doi.org/10.1016/j.scitotenv.2023.163600.
23. Wick RR, Judd LM, Gorrie CL, Holt KE. Unicycler: resolving bacterial genome assemblies from short and long sequencing reads. PLoS Comput Biol. 2017;13(6): e1005595. https://doi.org/10.1371/journal.pcbi.1005595.
24. Liu R, Xu H, Zhao J, Hu X, Wu L, Qiao J, et al. Emergence of mcr-8.2-harboring hypervirulent ST412 Klebsiella pneumoniae strain from pediatric sepsis: A comparative genomic survey. Virulence. 2023;14(1):233–45. https://doi.org/10.1080/21505594.2022.2158980.
25. Djordjevic SP, Jarocki VM, Seemann T, Cummins ML, Watt AE, Drigo B, et al. Genomic surveillance for antimicrobial resistance - a One Health perspective. Nat Rev Genet. 2024;25(2):142–57. https://doi.org/10.1038/s41576-023-00649-y.
26. Hussein NH, Al-Kadmy IMS, Taha BM, Hussein JD. Mobilized colistin resistance (mcr) genes from 1 to 10: a comprehensive review. Mol Biol Rep. 2021;48(3):2897–907. https://doi.org/10.1007/s11033-021-06307-y.
27. Albert MJ, Alam K, Islam M, Montanaro J, Rahaman AS, Haider K, et al. Hafnia alvei, a probable cause of diarrhea in humans. Infect Immun. 1991;59(4):1507–13. https://doi.org/10.1128/iai.59.4.1507-1513.1991.
28. Foroughi A, Namdari A, Rahimian-Zarif B. Detection of Escherichia albertii in urinary and gastrointestinal infections in Kermanshah, Iran. Int J Enteric Pathog. 2021;9(1):37–42. https://doi.org/10.34172/ijep.2021.08.
29. Zaki MES, Eid AE, El-Kazzaz SS, El-Sabbagh AM. Molecular study of Escherichia albertii in pediatric urinary tract infections. Open Microbiol J. 2021;15(1):139–44. https://doi.org/10.2174/1874285802115010139.
30. Poirel L, Kieffer N, Nordmann P. In vitro study of ISApl1-mediated mobilization of the colistin resistance gene mcr-1. Antimicrob Agents Chemother. 2017;61(7): e00127-17. https://doi.org/10.1128/aac.00127-17.

31. Snesrud E, He S, Chandler M, Dekker JP, Hickman AB, McGann P, et al. A model for transposition of the colistin resistance gene mcr-1 by ISApl1. Antimicrob Agents Chemother. 2016;60(11):6973–6. https://doi.org/10.1128/aac.01457-16.
32. Batisson I, Guimond MP, Girard F, An H, Zhu C, Oswald E, et al. Characterization of the novel factor paa involved in the early steps of the adhesion mechanism of attaching and effacing Escherichia coli. Infect Immun. 2003;71(8):4516–25. https://doi.org/10.1128/iai.71.8.4516-4525.2003.
33. Gomes TAT, Ooka T, Hernandes RT, Yamamoto D, Hayashi T. Escherichia albertii Pathogenesis. EcoSal Plus. 2020;9(1). https://doi.org/10.1128/ecosalplus.ESP-0015-2019.
34. Naka A, Hinenoya A, Awasthi SP, Yamasaki S. Isolation and characterization of Escherichia albertii from wild and safeguarded animals in Okayama Prefecture and its prefectural borders. Japan J Vet Med Sci. 2022;84(9):1299–306. https://doi.org/10.1292/jvms.22-0213.

Publisher's Note