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MECHANISMS OF RESISTANCE

OXA-181-Producing Extraintestinal Pathogenic *Escherichia coli* Sequence Type 410 Isolated from a Dog in Portugal

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ABSTRACT Two multidrug-resistant and carbapenemase-producing *Escherichia coli* clones of sequence type 410 were isolated from fecal samples of a dog with skin infection on admission to an animal hospital in Portugal and 1 month after discharge. Whole-genome sequencing revealed a 126,409-bp Col156/IncFIA/IncFII multidrug resistance plasmid and a 51,479-bp IncX3 $bla_{OXA-181}$-containing plasmid. The chromosome and plasmids carried virulence genes characteristic for uropathogenic *E. coli*, indicating that dogs may carry multidrug-resistant *E. coli* isolates related to those causing urinary tract infections in humans.

KEYWORDS pets, carbapenemase, veterinary, ExPEC, companion animals, *Escherichia coli*

Carbapenemase-producing *Enterobacterales* (CPE) represent a major public health issue, and their detection in animals has been increasing worldwide (1). So far, a predominant proportion of carbapenemase-producing *Escherichia coli* (CPEc) isolates associated with infections in humans belong to specific clonal lineages (2). Among them, the high-risk multidrug-resistant (MDR) sequence type (ST) 410 shows strong potential for transmission between different hosts, including companion animals (CAs) and humans (1, 3). CPEc ST410 isolates were identified in the United Kingdom in 2016 (4), South Korea in 2017 (5), and Switzerland in 2018 (6), mostly associated with nosocomial carriage (6). Because of this CPE emergence in CAs and the potential transmission to humans (3, 7), we assessed CPE gut carriage in healthy CAs from the community, CAs with urinary tract infections (UTIs), and CAs suffering from skin and soft tissue infections (SSTIs) who attended the University Veterinary Teaching Hospital (UVTH) in Lisbon, Portugal.

Fecal samples of 71 healthy CAs (47 dogs and 24 cats) were collected at home, and samples of 15 CAs with UTIs (13 dogs and 2 cats) and 12 CAs with SSTIs (11 dogs and 1 cat) were taken on admission to UVTH for CPE screening between January 2016 and August 2019. Signed informed consent from the owners and ethical approval were obtained (CEBEA 027/2018). Samples were plated, with and without preenrichment in peptone water, onto MacConkey agar plates supplemented with antibiotic discs containing meropenem (10 μg), temocillin (30 μg), and CAT-ID (Mastdiscs ID for CPE screening). Isolates were identified using matrix-assisted laser desorption ionization–time of flight mass spectrometry (Brucker). Carbapenemase production was assessed using Blue-Carba (8). One dog from the SSTI group tested positive for CPEc (strain PT113) on admission in 2017 and was still positive after 1 month (strain PT109). Strains PT109 and PT113 were nonsusceptible to ampicillin (64 μg/ml), ceftazidime (>128 μg/ml), cefotaxime (>64 μg/ml), cefepime (>32 μg/ml), ciprofloxacin (>8 μg/ml), chloramphenicol (16 μg/ml), ertapenem (1 μg/ml), sulfamethoxazole (>1024 μg/ml), trimethoprim

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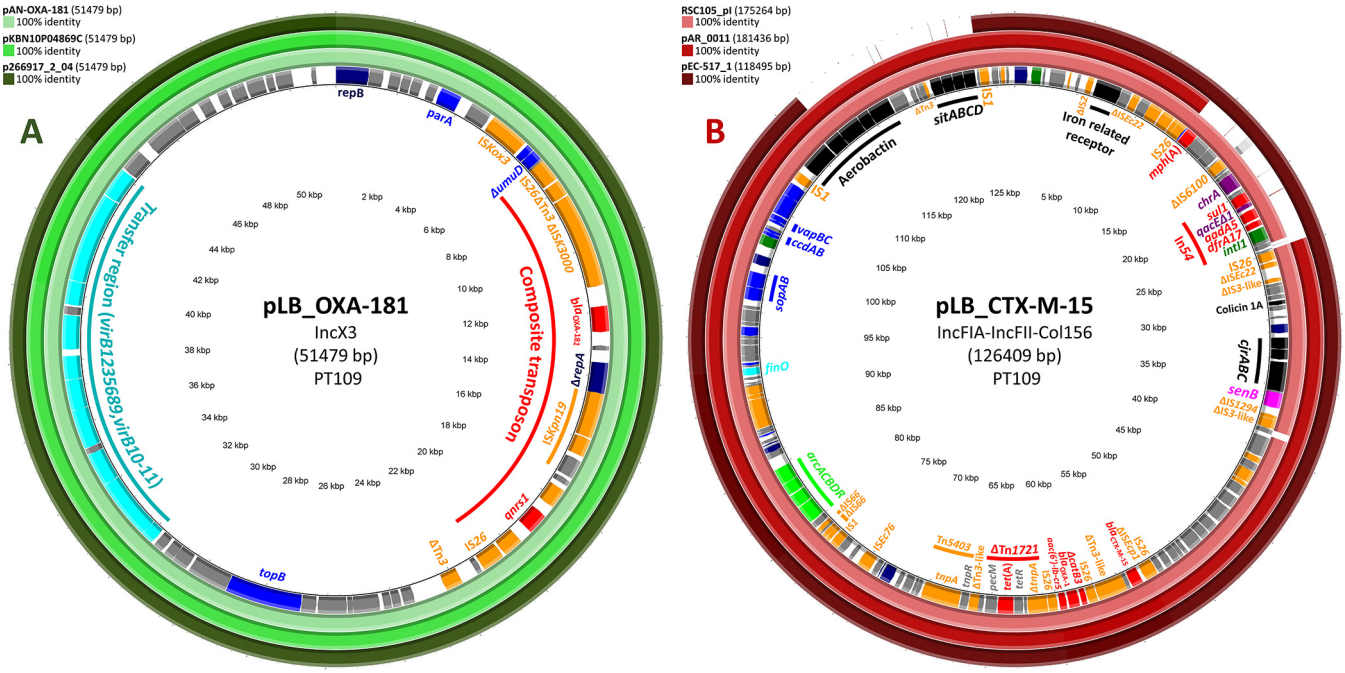

FIG 1 Circular map of plasmid pLB_OXA-181 (CP041033) (A) and plasmid pLB_CTX-M-15 (CP041032) (B) found in PT109. The colored outer rings represent regions of homology that both plasmids share with their three closest relative plasmids found in the GenBank database. Genes present in pLB_OXA-181 and pLB_CTX-M-15 are portrayed as arrows in the inner ring of each circular map and are colored according to their gene function classification. (A) First outer ring (light green), p_AN-OXA-181 (MK416154); second outer ring (green), pKBN10P04869C (CP026476); third outer ring (dark green), p266917_2_04 (CP026727). (B) First outer ring (pink), RSC105_pI (LO017737); second outer ring (red), pAR_0011 (CP024856); third outer ring (burgundy), pEC517_1 (CP018964). The scale circle shows the coordinates in kilobase pairs of the reference plasmid. Antibiotic resistance genes and their functions: *aadA*, streptomycin/spectinomycin adenyltransferase; *aac(6′)-Ib-cr*, aminoglycoside and quinolone acetyltransferase; bla_{OXA-1}, β-lactamase; $bla_{CTX-M-15}$, extended-spectrum β-lactamase; $bla_{OXA-181}$, carbapenemase; *dfrA17*, trimethoprim-insensitive dihydrofolate reductase; *mph*(A), macrolide phosphotransferase; *sul1*, sulfonamide-insensitive dihydropteroate synthase; *tet*(A), tetracycline efflux pump; *qnrS1*, DNA gyrase protection gene conferring low-level resistance to fluoroquinolones. Genes are represented by colored blocks: red, antibiotic resistance genes; lime, genes associated with higher fitness; purple, genes associated with resistance to disinfectants and heavy metals; orange, transposase genes, transposons (Tn), and insertion sequences (IS); green, integrase genes; light turquoise, conjugation-associated genes; blue, genes associated with partition, modification, and stability systems; dark blue, replication genes; black, virulence genes; pink, toxin genes; gray, other genes.

(>32 μg/ml), and tetracycline (>64 μg/ml) as determined by broth microdilution (EUVSEC/EUVSEC2, Thermo Fisher Scientific) according to CLSI recommendations (9).

The genomic sequence of strain PT109 was obtained using MinION R.9.4.1 flow cell (Oxford Nanopore Technology) and Novaseq6000 (Illumina) and assembled with Unicycler (v0.4.4); whereas strain PT113 was sequenced with Novaseq6000, read mapped to contigs of strain PT109 (Geneious v10.1.3), and assembled using SPAdes (v3.12.0). The full assembly of strain PT109 resulted in the 4,815,030-bp chromosome (GenBank accession no. CP041031), the 126,409-bp Col156/IncFIA/IncFII plasmid pLB_CTX-M-15 (GenBank accession no. CP041032), and the 51,479-bp IncX3 plasmid pLB_OXA-181 (GenBank accession no. CP041033). PlasmidFinder 2.1, ResFinder 3.2, MLST 2.0, SerotypeFinder 2.0, VirulenceFinder 2.0, (http://www.genomicepidemiology.org/), virulence factor database (http://www.mgc.ac.cn/VFs/main.htm), ISfinder (https://isfinder.biotoul.fr/) and INTEGRALL (http://integrall.bio.ua.pt/) were used for *in silico* analyses.

Strains PT109 and PT113 belonged to ST410 and their serotypes to the recently described genotype OgN5 (10). The colanic acid operon was upstream of the O locus, which is crucial for biofilm production, withstanding desiccation (11), and for protection against complement-mediated killing in serum (12). The strains contained amino acid substitutions associated with fluoroquinolone resistance in GyrA (S83L and D87N) and ParC (S80I), as well as multiple antimicrobial resistance genes (ARGs) on plasmids pLB_CTX-M-15 and pLB_OXA-181 (Fig. 1). Plasmid pLB_OXA-181 was virtually identical to other OXA-181-containing IncX3 plasmids in the NCBI database (1 single nucleotide polymorphism difference) (5) (Fig. 1). The two plasmids sharing the closest similarity to pLB_CTX-M-15 lack the resistance integron In*54* or the iron transport systems (Fig. 1).

TABLE 1 Virulence factors of *Escherichia coli* strains PT109 and PT113

Virulence factor	Function (ref)	Pathotype[b]	Virulence gene(s)	Genetic location in PT109, coordinates (accession no.)
F9 fimbriae	Adherence (18)	AIEC, EAEC, EPEC, EHEC, UPEC	*z2200–z2206*	Chromosome, 2,432,071–2,438,432 (CP041031)
Hemorrhagic *E. coli* pilus	Adherence (19)	EHEC, ETEC, EPEC	*hcpABC*	Chromosome, 3,881,323–3,884,350 (CP041031)
CFA/I fimbriae	Adherence (20)	ETEC	*cfaABCD*	Chromosome, 691,527–696,541 (CP041031)
Curli fibers	Adherence (13)	UPEC, SEPEC, APEC	*csgBAC, csgDEFG*	Chromosome, 2,883,283–2,887,724 (CP041031)
Intimin-like FdeC	Adherence (21)	ExPEC, UPEC, ETEC, EHEC, EPEC, AIEC, EAEC, STEC	*eaeH*	Chromosome, 3,690,607–3,694,863 (CP041031)
E. coli common pilus	Adherence (22)	EPEC and commensals	*ecpRABCDE*	Chromosome, 3,697,931–3,704,724 (CP041031)
Type 1 fimbriae	Adherence (13)	ExPEC, UPEC, NMEC, SEPEC, APEC	*fimBEAICDFGH*	Chromosome, 4,135,565–4,144,317 (CP041031)
Stg fimbriae	Adherence (23)	ExPEC, APEC	*stgABCD*	Chromosome, 4,783,515–4,788,497 (CP041031)
UpaG adhesin	Adherence (24)	ExPEC, UPEC	*upaG*	Chromosome, 135,019–130,169 (CP041031)
E. coli laminin-binding fimbriae	Adherence (25)	EPEC, STEC, EHEC, and commensals	*elfADCG*	Chromosome, 2,982,660–2,989,604 (CP041031)
NlpI lipoprotein	Adherence (26)	EPEC, AIEC	*nlpI*	Chromosome, 627,338–628,222 (CP041031)
Flagella	Adherence and motility (27)	EPEC, ExPEC	*fliRQPONMLKJLHGFE, fliSTDC, fliAZY, flhDC, motAB, cheAWMRBYZ, flhBAE, flgLKLIHGFEDCBAMN*	Chromosome, 1,987,639–1,976,661, 1,998,119–2,005,128, 2,025,740–2,041,357, 2,847,590–2,859,159 (CP041031)
ETT2 locus (degenerate)	Type III secretion system (28)	EPEC, STEC	*eivACI, eivJ12, epaOPQR, epaS12, eivH, eprHIJK*	Chromosome, 951,137–968,978 (CP041031)
Yersiniabactin	Siderophore (13)	ExPEC, UPEC	*fyuA, irp1, ipr2, ybtAEPQSTUX*	Chromosome, 1,904,355–1,933,185 (CP041031)
Enterobactin	Siderophore (13)	EPEC, ExPEC, and commensals	*entABCDEFH, entS, fepABCDEG, fes, ybdz*	Chromosome, 3,404,020–3,423,654 (CP041031)
Agn43	Autotransporter (13)	ExPEC, UPEC	*flu*	Chromosome, 52,072–49,228 (CP041031)
Aida-like protein	Autotransporter	Yet unspecified	Similar to *ehaB*	Chromosome, 3,615,037–3,617,682 (CP041031)
Colanic acid operon	Capsule production (12)	ExPEC, yet unspecified	*wzabc, wcaABCDEFJKLM, gmd, fcl, wcaI, cpsBG, wzxC*	Chromosome, 1,789,589–1,812,389 (CP041031)
Iron-related receptor[a]	Iron transport	Yet unspecified	*tonB-like*	pLB_CTX-M-15, 6,210–8,180 (CP041032)
Colicin A1 immunity protein	Colicin tolerance	Yet unspecified	Yet unspecified	pLB_CTX-M-15, 28,608–28,943 (CP041032)
CjrABC	Iron acquisition (14)	ExPEC, UPEC	*cjrABC*	pLB_CTX-M-15, 31,413–35,502 (CP041032)
SenB	Toxin (14)	ExPEC, UPEC	*senB*	pLB_CTX-M-15, 35,571–36,746 (CP041032)
Aerobactin	Siderophore (13)	UPEC, APEC	*iucABCD, iutA*	pLB_CTX-M-15, 108,676–116,656 (CP041032)
Sit operon	Iron acquisition (13)	UPEC, APEC	*sitABCD*	pLB_CTX-M-15, 119,979–123,428 (CP041032)
Arginine deiminase operon	Increased fitness (UTI) (15)	UPEC	*arcACBDR*	pLB_CTX-M-15, 79,550–84,792 (CP041032)

[a]The protein derived from the gene is related to a transferrin/lactoferrin family receptor.

[b]Groups of pathogenic *E. coli* with which the virulence factors are the most commonly associated: AIEC, adherent-invasive *E. coli*; APEC, avian pathogenic *E. coli*; EAEC, enteroaggregative *E. coli*; EHEC, enterohemorrhagic *E. coli*; EPEC, enteropathogenic *E. coli*; ETEC, enterotoxigenic *E. coli*; ExPEC, extraintestinal pathogenic *E. coli*; NMEC, neonatal meningitis-causing *E. coli*; SEPEC, sepsis-associated *E. coli*; UPEC, uropathogenic *E. coli*.

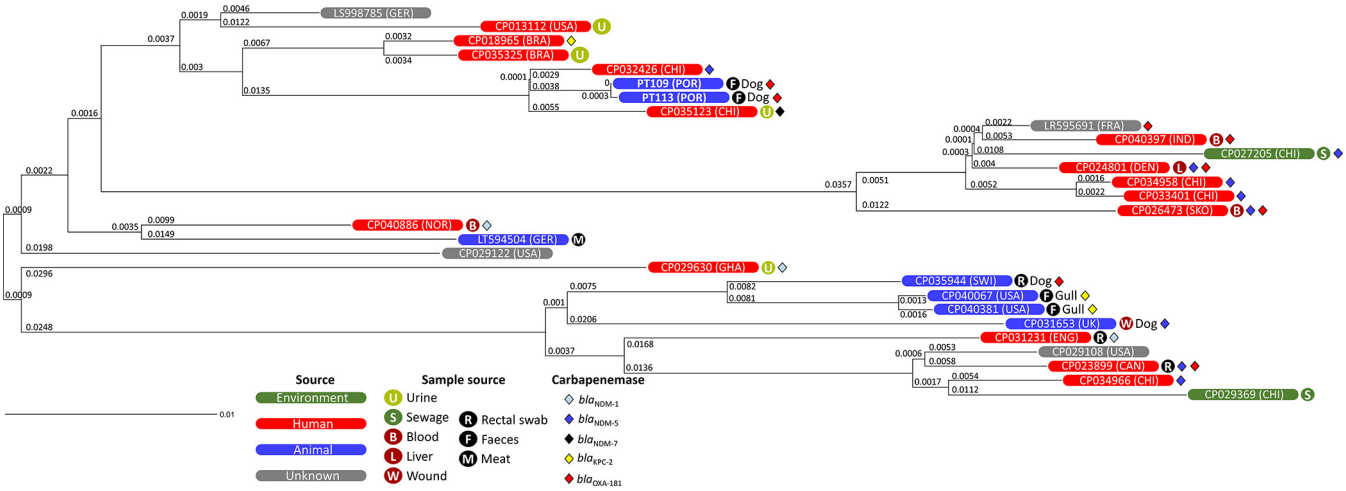

FIG 2 Phylogenetic neighbor-joining tree of all complete and/or circular genomes of *E. coli* ST410 strains available in the GenBank database ($n = 26$; accessed 5 August 2019), together with PT109 (GenBank accession no. CP041031) and PT113. The phylogenetic neighbor-joining tree was based on the parameters "pairwise ignoring missing values" and "percentage columns difference." The accession numbers of the sequences included in the tree are shown in the rounded rectangles, which are colored depending on their isolation source; when provided, details of the isolation source and carbapenemase production are shown by colored circles and rectangles, respectively. Accession numbers used were LS998785 (strain EC-TO75), CP013112 (YD786), CP018965 (Ecol_517), CP035325 (BR12-DEC), CP032426 (SCEC020001), CP041031 (PT109), CP035123 (EC25), LR595691 (EcMAD1), CP040397 (BA22372), CP027205 (WCHEC025943), CP024801 (AMA1167), CP034958 (SCEC020026), CP033401 (WCHEC020031), CP026473 (KBN10P04869), CP040886 (K71-77), LT594504 (RL465), CP029122 (AR434), CP029630 (ST410), CP035944 (AR24.2b), CP040067 (A1_181), CP040381 (A1_180), CP031653 (UK_Dog_Liverpool), CP031231 (Es_ST410_NW1_NDM_09_2017), CP029108 (AR437), CP023899 (FDAARGOS_433), CP034966 (WCHEC020032), and CP029369 (WCHEC035148).

The genome of PT109 and PT113 exhibited a repertoire of virulence factors ($n = 25$) that classified them as extraintestinal pathogenic *E. coli* (ExPEC) strains (13) (Table 1). Specifically, 12 of them were characteristic for uropathogenic *E. coli* (UPEC) (Table 1). UPEC virulence factors, such as the iron-related systems SitABCD, aerobactin (*iucABCD*, *iutA*), and CjrABC-SenB, and the arginine deiminase operon (ADO) (*arcACBDR*) were all located on pLB_CTX-M-15 (Fig. 1). The *cjrABC-senB* gene cluster is involved in the virulence of UPEC in humans (14), and ADO has enhanced the capacity of a wild-type strain of *E. coli* to infect kidneys in a mouse model (15), indicating that PT109 and PT113 isolates have strong potential for developing UTIs in humans (13–15).

Comparative analysis of the virulence and ARGs found in strains PT109 and PT113 with those of other ST410 strains from NCBI showed that they all contained different ARGs but shared equally 12 of the virulence factors (see Tables S1 and S2 in the supplemental material). However, presence of the *sit* operon (40% of strains) and aerobactin was less common (35%); whereas the colanic acid operon (25%), yersiniabactin (21%), and CjrABC-SenB (4%) were rarely detected (see Table S2). In fact, none of these other strains possessed the same virulence and resistance traits as PT109 and PT113, emphasizing the genetic plasticity and the variable pathogenic potential of the ST410 lineage. These different factors might have contributed to the colonization success and persistence capacity of strains PT109 and PT113, which cannot be explained by antibiotic selective pressure alone.

The genetic relationship among PT109, PT113, and other ST410 strains from the GenBank database ($n = 26$) was also determined using an *ad hoc* core genome MLST analysis comparing 3,375 genes common to all strains (cgMLST Target Definer, Seqsphere+ v6.0.2). The resulting phylogenetic analysis dissociated PT109 and PT113 from the other CPEc isolates of animal origin but connected them to human strains isolated from urine, which supports the assumption that both strains have potential for developing UTIs in humans (Fig. 2). Strains PT109 and PT113 were highly genetically related, differing by only one allele, whereas they differed from the other ST410 strains by at least 23 and up to 262 different alleles. Strains from the same clade of PT109 and PT113 (CP018965, CP035325, CP032426, and CP035123) shared most virulence factors ($n = 24/25$) and contained the same genotype OgN5, suggesting that specific ST410 lineages are more likely to be successful in colonization and/or infection.

This study provided in-depth characterization of the first OXA-181-producing ExPEC obtained in a veterinary environment and comparison with other ST410 strains. Detection of the same clone within a 1-month period indicated that such MDR and carbapenemase-producing pathogenic *E. coli* isolates can temporarily persist in dogs and disseminate into the environment, other animals, and humans, therefore posing a major One Health concern (16, 17).

Data availability. Newly determined sequences have been deposited in GenBank under accession no. CP041031 to CP041033.

SUPPLEMENTAL MATERIAL

Supplemental material is available online only.

SUPPLEMENTAL FILE 1, PDF file, 0.1 MB.

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REFERENCES

1. Köck R, Daniels-Haardt I, Becker K, Mellmann A, Friedrich AW, Mevius D, Schwarz S, Jurke A. 2018. Carbapenem-resistant *Enterobacteriaceae* in wildlife, food-producing, and companion animals: a systematic review. Clin Microbiol Infect 24:1241–1250. https://doi.org/10.1016/j.cmi.2018.04.004.
2. Manges AR, Geum HM, Guo A, Edens TJ, Fibke CD, Pitout J. 2019. Global extraintestinal pathogenic *Escherichia coli* (ExPEC) lineages. Clin Microbiol Rev 32:e00135-18. https://doi.org/10.1128/CMR.00135-18.
3. Endimiani A, Brilhante M, Bernasconi OJ, Perreten V, Schmidt JS, Dazio V, Nigg A, Gobeli Brawand S, Kuster SP, Schuller S, Willi B. 2019. Employees of Swiss veterinary clinics colonized with epidemic clones of carbapenemase-producing *Escherichia coli*. J Antimicrob Chemother Dec 9:dkz470. https://doi.org/10.1093/jac/dkz470.
4. Reynolds ME, Phan HTT, George S, Hubbard ATM, Stoesser N, Maciuca IE, Crook DW, Timofte D. 2019. Occurrence and characterization of *Escherichia coli* ST410 co-harbouring bla_{NDM-5}, bla_{CMY-42} and $bla_{TEM-190}$ in a dog from the UK. J Antimicrob Chemother 74:1207–1211. https://doi.org/10.1093/jac/dkz017.
5. Hong JS, Song W, Park HM, Oh JY, Chae JC, Han JI, Jeong SH. 2019. First detection of New Delhi metallo-β-lactamase-5-producing *Escherichia coli* from companion animals in Korea. Microb Drug Resist 25:344–349. https://doi.org/10.1089/mdr.2018.0237.
6. Nigg A, Brilhante M, Dazio V, Clément M, Collaud A, Gobeli Brawand S, Willi B, Endimiani A, Schuller S, Perreten V. 2019. Shedding of OXA-181 carbapenemase-producing *Escherichia coli* from companion animals after hospitalisation in Switzerland: an outbreak in 2018. Euro Surveill 24:1900071. https://doi.org/10.2807/1560-7917.ES.2019.24.39.1900071.
7. Grönthal T, Österblad M, Eklund M, Jalava J, Nykäsenoja S, Pekkanen K, Rantala M. 2018. Sharing more than friendship - transmission of NDM-5 ST167 and CTX-M-9 ST69 *Escherichia coli* between dogs and humans in a family, Finland, 2015. Euro Surveill 23:1700497. https://doi.org/10.2807/1560-7917.ES.2018.23.27.1700497.
8. Pires J, Novais A, Peixe L. 2013. Blue-Carba, an easy biochemical test for detection of diverse carbapenemase producers directly from bacterial cultures. J Clin Microbiol 51:4281–4283. https://doi.org/10.1128/JCM.01634-13.
9. Clinical and Laboratory Standards Institute. 2018. Performance standards for antimicrobial susceptibility testing—28th ed. CLSI supplement M100. Clinical and Laboratory Standards Institute Wayne, PA.
10. Iguchi A, von Mentzer A, Kikuchi T, Thomson NR. 2017. An untypeable enterotoxigenic *Escherichia coli* represents one of the dominant types causing human disease. Microb Genom 3:e000121. https://doi.org/10.1099/mgen.0.000121.
11. Whitfield C. 2006. Biosynthesis and assembly of capsular polysaccharides in *Escherichia coli*. Annu Rev Biochem 75:39–68. https://doi.org/10.1146/annurev.biochem.75.103004.142545.
12. Miajlovic H, Cooke NM, Moran GP, Rogers TR, Smith SG. 2014. Response of extraintestinal pathogenic *Escherichia coli* to human serum reveals a protective role for Rcs-regulated exopolysaccharide colanic acid. Infect Immun 82:298–305. https://doi.org/10.1128/IAI.00800-13.
13. Sarowska J, Futoma-Koloch B, Jama-Kmiecik A, Frej-Madrzak M, Ksiazczyk M, Bugla-Ploskonska G, Choroszy-Krol I. 2019. Virulence factors, prevalence and potential transmission of extraintestinal pathogenic *Escherichia coli* isolated from different sources: recent reports. Gut Pathog 11:10. https://doi.org/10.1186/s13099-019-0290-0.
14. Mao BH, Chang YF, Scaria J, Chang CC, Chou LW, Tien N, Wu JJ, Tseng CC, Wang MC, Chang CC, Hsu YM, Teng CH. 2012. Identification of *Escherichia coli* genes associated with urinary tract infections. J Clin Microbiol 50:449–456. https://doi.org/10.1128/JCM.00640-11.
15. Billard-Pomares T, Clermont O, Castellanos M, Magdoud F, Royer G, Condamine B, Fouteau S, Barbe V, Roche D, Cruveiller S, Médigue C, Pognard D, Glodt J, Dion S, Rigal O, Picard B, Denamur E, Branger C. 2019. The arginine deiminase operon is responsible for a fitness trade-off in extended-spectrum-β-lactamase-producing strains of *Escherichia coli*. Antimicrob Agents Chemother 63:e00635-19. https://doi.org/10.1128/AAC.00635-19.
16. Pulss S, Stolle I, Stamm I, Leidner U, Heydel C, Semmler T, Prenger-Berninghoff E, Ewers C. 2018. Multispecies and clonal dissemination of OXA-48 carbapenemase in *Enterobacteriaceae* from companion animals

in Germany, 2009–2016. Front Microbiol 9:1265. https://doi.org/10.3389/fmicb.2018.01265.
17. Pitout JDD, Peirano G, Kock MM, Strydom KA, Matsumura Y. 2019. The global ascendency of OXA-48-type carbapenemases. Clin Microbiol Rev 33:e00102-19. https://doi.org/10.1128/CMR.00102-19.
18. Wurpel DJ, Totsika M, Allsopp LP, Hartley-Tassell LE, Day CJ, Peters KM, Sarkar S, Ulett GC, Yang J, Tiralongo J, Strugnell RA, Jennings MP, Schembri MA. 2014. F9 fimbriae of uropathogenic *Escherichia coli* are expressed at low temperature and recognise Galβ1-3GlcNAc-containing glycans. PLoS One 9:e93177. https://doi.org/10.1371/journal.pone.0093177.
19. Xicohtencatl-Cortes J, Monteiro-Neto V, Saldaña Z, Ledesma MA, Puente JL, Girón JA. 2009. The type 4 pili of enterohemorrhagic *Escherichia coli* O157:H7 are multipurpose structures with pathogenic attributes. J Bacteriol 191:411–421. https://doi.org/10.1128/JB.01306-08.
20. Li YF, Poole S, Nishio K, Jang K, Rasulova F, McVeigh A, Savarino SJ, Xia D, Bullitt E. 2009. Structure of CFA/I fimbriae from enterotoxigenic *Escherichia coli*. Proc Natl Acad Sci U S A 106:10793–10798. https://doi.org/10.1073/pnas.0812843106.
21. Nesta B, Spraggon G, Alteri C, Moriel DG, Rosini R, Veggi D, Smith S, Bertoldi I, Pastorello I, Ferlenghi I, Fontana MR, Frankel G, Mobley HL, Rappuoli R, Pizza M, Serino L, Soriani M. 2012. FdeC, a novel broadly conserved *Escherichia coli* adhesin eliciting protection against urinary tract infections. mBio 3:e00010-12. https://doi.org/10.1128/mBio.00010-12.
22. Rendón MA, Saldaña Z, Erdem AL, Monteiro-Neto V, Vázquez A, Kaper JB, Puente JL, Girón JA. 2007. Commensal and pathogenic *Escherichia coli* use a common pilus adherence factor for epithelial cell colonization. Proc Natl Acad Sci U S A 104:10637–10642. https://doi.org/10.1073/pnas.0704104104.
23. Lymberopoulos MH, Houle S, Daigle F, Léveillé S, Brée A, Moulin-Schouleur M, Johnson JR, Dozois CM. 2006. Characterization of Stg fimbriae from an avian pathogenic *Escherichia coli* O78:K80 strain and assessment of their contribution to colonization of the chicken respiratory tract. J Bacteriol 188:6449–6459. https://doi.org/10.1128/JB.00453-06.
24. Valle J, Mabbett AN, Ulett GC, Toledo-Arana A, Wecker K, Totsika M, Schembri MA, Ghigo JM, Beloin C. 2008. UpaG, a new member of the trimeric autotransporter family of adhesins in uropathogenic *Escherichia coli*. J Bacteriol 190:4147–4161. https://doi.org/10.1128/JB.00122-08.
25. Samadder P, Xicohtencatl-Cortes J, Saldaña Z, Jordan D, Tarr PI, Kaper JB, Girón JA. 2009. The *Escherichia coli* ycbQRST operon encodes fimbriae with laminin-binding and epithelial cell adherence properties in Shiga-toxigenic *E. coli* O157:H7. Environ Microbiol 11:1815–1826. https://doi.org/10.1111/j.1462-2920.2009.01906.x.
26. Barnich N, Bringer MA, Claret L, Darfeuille-Michaud A. 2004. Involvement of lipoprotein NlpI in the virulence of adherent invasive *Escherichia coli* strain LF82 isolated from a patient with Crohn's disease. Infect Immun 72:2484–2493. https://doi.org/10.1128/iai.72.5.2484-2493.2004.
27. Haiko J, Westerlund-Wikström B. 2013. The role of the bacterial flagellum in adhesion and virulence. Biology (Basel) 2:1242–1267. https://doi.org/10.3390/biology2041242.
28. Zhou M, Guo Z, Duan Q, Hardwidge PR, Zhu G. 2014. *Escherichia coli* type III secretion system 2: a new kind of T3SS? Vet Res 45:32. https://doi.org/10.1186/1297-9716-45-32.