Antimicrobial resistance is a major hurdle impeding infection control and treatment worldwide. There is limited information on the antimicrobial susceptibility of *Escherichia albertii*, a recently discovered enteropathogen. *E. albertii* was first described in 2003 following isolation from human stool samples of Bangladeshi children in the early 1990s (1). *E. albertii* appears to infect both animals and humans, and it may be subclinically carried by wild and domestic birds (2). In Japan, *E. albertii* has increasingly been reported to be an enteropathogen infecting humans (2). In the Akita Prefecture of Japan, a number of *E. albertii* strains have been isolated or reidentified from stocks of laboratory *E. coli* strains (3,4). The purpose of this study was to investigate antimicrobial susceptibility of *E. albertii* strains.

A total of 28 *E. albertii* strains were used to investigate antimicrobial susceptibility in this study. These strains were isolated from human stool samples in the Akita Prefecture between 1995 and 2017. The susceptibility of *E. albertii* strains was determined using the BD-Sensi-Disc kit (Nippon Becton Dickinson, Tokyo, Japan), against 11 antimicrobial agents, namely: 10 μg ampicillin (ABPC), 30 μg ceftazidime (CAZ), 10 μg cefotaxime (CTX), 30 μg cefmetazole (CMZ), 10 μg imipenem (IPM), 30 μg kanamycin (KM), 10 μg streptomycin (SM), 50 μg fosfomycin (FOM), 30 μg tetracycline (TC), 30 μg nalidixic acid (NA), and 10 μg norfloxacin (NFLX), on Mueller Hinton agar (Oxoid, Basingstoke, UK). The results were interpreted according to the manufacturer’s instructions, based on the criteria outlined by the Clinical and Laboratory Standards Institute document M100-S23 (5). The minimum inhibitory concentration (MIC) of NFXL for NA-resistant strains was determined using the Ettest diagnostic kit (bioMérieux, Tokyo, Japan), according to the manufacturer’s instructions. Quality controls were performed using *E. coli* strain ATCC25922.

Resistance against quinolones is primarily due to amino acid substitutions in the topoisomerase enzymes, such as DNA gyrase and topoisomerase IV. To test for amino acid substitutions in quinolone-resistant strains, we examined the DNA sequences of *gyrA*, *gyrB*, *parC*, and *parE* amplicons encompassing the quinolone resistance determining regions (QRDRs) in DNA gyrase and topoisomerase IV. The QRDRs of *gyrA*, *parC*, and *parE* were amplified via PCR using previously reported primers (6) and Ex-taq DNA polymerase (TaKaRa Bio Inc., Shiga, Japan). *gyrB* was amplified using the primers 5'-CGG ACT GTC AGG ACG CGA C-3' and 5'- AGC CAA GCG CAG TGA TCA GC-3'. The reaction conditions were as follows: initial denaturation at 94 °C for 2 min, followed by 30 cycles of annealing and extension at 94 °C for 30 s, 55 °C for 30 s, and 72 °C for 45 s and final extension at 72 °C for 5 min. DNA sequencing was performed by Fasmac Co., Ltd. (Kanagawa, Japan). Predicted polypeptide products were analyzed for amino acid substitutions by comparison with *gyrA*, *gyrB*, *parC*, and *parE* of wild-type *E. coli* (accession number U00096) (6).

Three strains (10.7%) were resistant to at least one antimicrobial agent (Table 1). Among these, the strain EC15062 showed acquired resistance to SM, TC, and NA. The NFXL MIC for this strain was 1.0 μg/ml, although it was still within the range for susceptibility. DNA sequences of the amplicons of *gyrA*, *gyrB*, *parC*, and *parE* in EC15062 were determined (accession number LC481536-481539). The strain EC15062 harbored an amino acid substitution at position 83 in *gyrA* (Fig. 1).

Results of this study provide significant information regarding the antimicrobial susceptibility and epidemiology of *E. albertii*. Quinolones are commonly used antimicrobial agents against infectious human diseases and therefore, are one of the most important antimicrobials in public health. NA was the first quinolone described in the 1960s, and the family of quinolones emerged after the discovery of NA. NA-resistant strains are anticipated to show reduced susceptibility to other quinolones as well. *E. albertii* is naturally sensitive to quinolones, and the range of NFXL MIC is between 0.06 and 0.13 μg/ml (7). However,
NA-resistant strain EC15062 had a higher NFLX MIC, suggesting reduced susceptibility to other quinolones. The amino acid substitutions in the QRDR are likely to induce a local conformational change in DNA gyrase (8). In particular, the amino acid substitution S83L in GyrA is believed to impart reduced susceptibility to quinolones and is the most frequently detected substitution in quinolone-resistant E. coli strains (6, 8). Therefore, the amino acid substitution at position 83 in GyrA appears to be the preliminary step in the acquisition of fluoroquinolone resistance. Additional mutations in QRDRs might lead to fluoroquinolone resistance in E. albertii strains. Furthermore, emergence of multidrug-resistant E. albertii is a matter of concern since quinolone resistant strains are often more resistant to other antimicrobial agents than quinolone-susceptible strains (9). Thus, the emergence of antimicrobial resistant E. albertii should be considered during treatment. Meanwhile, the rate of quinolone resistance has been reported to increase in E. coli isolated from poultry (10). Quinolone-resistant E. albertii may be transmitted to humans via poultry consumption. Thus, further studies comparing antimicrobial susceptibility across strains derived from poultry and other sources are warranted to identify the specific source of E. albertii.

In conclusion, this study showed the emergence of quinolone-resistant E. albertii. The emergence and the spread of enhanced antimicrobial resistance, such as fluoroquinolone and multidrug resistance, in E. albertii are unsettling. Thus, careful monitoring of antimicrobial resistance in E. albertii is imperative for future control of infections.

Conflict of interest None to declare.

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