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P1341 Occurrence and commonalities of plasmid-mediated quinolone resistance in *Escherichia coli* isolates recovered from livestock and food in Germany

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Background: Quinolones are important antibiotics belonging to a family of synthetic broad-spectrum drugs. Resistance to quinolones can be chromosomally encoded or plasmid-mediated (PMQR). One PMQR mechanism is mediated by Qnr proteins. The horizontal gene transfer of this plasmid-mediated quinolone resistance increases the threat of fallible treatment with some antibiotics. To better understand the *qnr* PMQR pathway as well as the distribution of *qnr* genes, *Escherichia (E.) coli* isolates recovered in 2016 and 2017 from livestock and food were phenotypically and genotypically characterized.

Materials/methods: 6,817 *E. coli* isolates from the German National Reference Laboratory for Antimicrobial Resistance were investigated. The isolates were received in the German national monitoring program for antimicrobial resistance in zoonotic bacteria, recovered from livestock and food. Antimicrobial resistance was determined by broth microdilution according to CLSI guidelines. MIC values were evaluated using EUCAST epidemiological cut-off values. *E. coli* resistant to quinolones were subjected to *qnr*-PCR, XbaI-PFGE, S1-PFGE, WGS and bioinformatic analysis. Six different *qnr*-PCRs were conducted to identify the respective *qnr*-variants.

Results: Of 6,817 *E. coli* tested, more than 800 isolates were classified as quinolone-resistant (MIC_{NAL} ≥16 mg/L and/or MIC_{CIP} ≥0.06 mg/L). The most abundant *qnr*-variant was *qnrS*. With the exception of *qnrD,* other *qnr*-variants were found evenly distributed. In general, *E. coli* isolates with *qnr*-genes were found more frequently in the feces of animals than in the meat of the same animal species. PFGE with XbaI-digestion was performed to examine genetic relatedness of isolates. PFGE profiling demonstrated a rather high heterogeneity. The highly diverse PFGE pattern indicates that the screened isolates are not associated to a predominant *E. coli* clone spreading via vertical transmission. S1-PFGE plasmid profiling showed a variety of extrachromosomal elements of various sizes. Isolates, selected according their PFGE pattern were further screened for their genetic setting through short read whole genome sequencing (WGS). Sequencing of those isolates confirmed the high genetic diversity of the quinolone-resistant *E. coli* strains.

Conclusions: Quinolone-resistance could not be attributed to a specific lineage of *E. coli*. Further analysis is needed for better understanding of the plasmid diversity within *qnr*-harboring *E. coli* and the prerequisites of their spread.