ciprofloxacin susceptibility and the presence of blaCTX-M are described in Table1. According to the serotypes, almost all of the K1 serotype isolates harbored five virulence genes. In K2 isolates, the prevalence of only rmpA and aerobactin was high. In K54 isolates, all of the five isolates had wcaG but the prevalence of other virulence genes were low (20–40%). For K57 isolates, all harbored rmpA and aerobactin, but none harbored other virulence genes. There was no difference in distribution of virulence genes (except rmpA) or serotypes according to the specimens.
Conclusion: The prevalence of virulence determinants was significantly higher in ciprofloxacin-susceptible isolates and it was different according to the serotypes. The K1 and K54 serotype was more frequent in ciprofloxacin-susceptible isolates and blaCTX-M-positive isolates, respectively.

Table 1. Prevalence of various virulence genes and serotypes according to the ciprofloxacin susceptibility and the presence of *bla*CTX-M

Virulence genes/ serotypes	CIP-R	CIP-S	*P* value	*bla*CTX-M-p	*bla*CTX-M-n	*P* value
wcaG	3/53	29/103	0.002	7/35	25/121	0.879
rmpA	5/53	34/103	0.003	5/35	34/121	0.150
allS	3/53	24/103	0.011	6/35	21/121	0.823
aerobactin	8/53	41/103	0.003	9/35	40/121	0.537
kfu	9/53	56/103	<0.001	11/35	54/121	0.230
K1	1/53	21/103	0.003	3/35	19/121	0.429
K2	2/53	8/103	0.536	1/35	9/121	0.560
K54	2/53	3/103	0.849	4/35	1/121	0.010
K57	1/53	4/103	0.880	0/35	4/121	0.629

P1357 *Escherichia coli* from human and avian origin: detection of fluoroquinolones-resistance associated clonal groups

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Objectives: Previous studies have suggested chicken as reservoirs for fluoroquinolones (FQ)-resistant extraintestinal pathogenic *E. coli* (ExPEC) strains isolated from humans. Our aim was to identify clones associated with FQ-resistance among human ExPEC strains and to investigate the possible source of these clones.
Methods: A total of 378 *E. coli* strains (277 from humans and 101 from avian species) were analyzed. Human ExPEC strains (142 FQ-susceptible and 135 resistant) were isolated from cases of urinary tract infections and sepsis. *E. coli* strains from avian source (68 FQ-susceptible and 33 resistant) were collected during routine control activities. Antimicrobial susceptibility was assessed by E-test. Phylogenetic groups were determined by PCR. MLST analysis was performed on a sample of *E. coli* strains from both human and avian sources. All phylogenetic group B2 strains were screened for the ST131 clone by PCR and then confirmed by MLST.
Results: Most FQ-resistant strains exhibited a multi-drug resistant (MDR) phenotype. Triple resistance to FQ, ampicillin and trimethoprim/sulfamethoxazole was the most frequent in both human and avian strains (30.5% and 60.6%, respectively). Overall, human ExPEC strains mostly fell into the phylogenetic group B2 (56.7%), followed by group D (17.7%). Avian strains mainly belonged to group A (34.6%) and group B1 (29.7%). A shift in phylogenetic distribution from group B2 to group A was observed associated to FQ-resistance in human strains. No shift associated with resistance was detected in avian strains. By MLST, human strains were distributed into 37 different sequence types (STs). Among FQ-resistant strains with a MDR phenotype, ST131 predominated (40%) followed by ST23 and ST10 complexes both belonging to the phylogenetic group A. FQ-susceptible strains from humans were widely dispersed among several STs and ST131 was found at low percentage (3.5%). Among avian *E. coli* strains, 27 different STs were identified. The clonal complexes ST 23, ST10 and ST156 were more common, each including FQ-resistant strains with a MDR phenotype. One ST131 FQ-susceptible strain was detected.
Conclusions: The major human and avian *E. coli* ST clones associated with FQ-resistance and MDR phenotype were identified. Although our results do not support the hypothesis of the possible avian origin of the major ST131 clone affecting humans, the sharing of the ST23 and ST10 clones across strains from human and avian sources poses a zoonotic risk.

P1358 Spread of plasmid-mediated quinolone resistance and pathogenic islands in *Escherichia coli* isolated from water

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Objectives: We report the search for plasmid-mediated quinolone resistance (PMQR) genes and virulence factors encoded in pathogenic islands (PAIs) among *Escherichia coli* strains isolated from waters of different origins.
Methods: Between May 2009 and February 2010, 37 isolates were recovered from drinking, recreational, waste, surface and ground waters. The antimicrobial susceptibilities were determined by the disc diffusion method by using CLSI guidelines. PMQR (qnr, aac(6′)-Ib-variant and qepA) determinants and PAIs markers were screened by PCR. Determination of the *E. coli* phylogenetic group was also performed by PCR.
Results: Mostly strains were isolated from surface waters (49%), and during the Summer 2009 (49%); 19% of the strains were resistant to nalidixic acid, 11% to ciprofloxacin and 5% to gentamicin. Molecular methods detected one type of PMQR: qnrA was detected among 16% of strains, all of which susceptible to quinolones. Pathogenicity islands (PAIs) IV536 was detected among surface (8%), drinking (5%) and ground waters (3%), while PAI IICFT073 was detected among drinking water (3%). According to the phylogenetic groups, we detected 41% and 27% of strains belonging to the less-virulent groups A and B1, while 30% and 3% were from groups D and B2, respectively. Our results showed 45% of strains from group D with the PAI IV536, a phylogenetic group also considered virulent. The PAI IV536 was also found in an isolate belonging to group B1 carrying a qnrA gene. All the qnrA genes were detected in group B1 strains collected in waters of different origin, geographical location and season.
Conclusions: The results showed the presence of potential pathogenic strains encoding resistance determinants and/or pathogenic islands among surface, ground and drinking waters. These strains can represent an increased risk for public health, as they were isolated from samples collected from waters with potential contact with humans and animals. Thus, it's of crucial importance a periodic monitoring of the water quality, not only for the presence of microorganisms, but also for screening antibiotic resistance and virulence factors.

P1359 Occurrence of plasmid-mediated quinolone resistance among bacteria isolated in animals in Portugal

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Background: Plasmid-mediated quinolone resistance (PMQR) is increasingly identified worldwide in Enterobacteriaceae. The aim of this study was to evaluate the extension of PMQR in isolates from animals in Portugal.
Methods: We screened 186 Enterobacteriaceae isolates for the presence of PMQR determinants, identified at National Laboratory of Veterinary Research (2008–2009). A total of 92 *Salmonella* isolates were isolated from broilers, layers and pigs, and 94 *Escherichia coli* were from farm animals, birds and mammals. Susceptibility testing of all isolates was performed by disk diffusion method, and MICs against nalidixic acid, ciprofloxacin, gatifloxacin, levofloxacin, ofloxacin, enrofloxacin, morbifloxacin and norfloxacin were determined by E-test for PMQR-positive isolates. PCR and nucleotide sequencing, by using specific primers, were used to screen for the presence of PMQR-encoding genes. The genetic context of PMQR genes was evaluated by using different molecular methods.

Results: We identified 5 qnrC-positive isolates: 2 *Salmonella enteritidis* collected in 2 layer chicken and in 3 *E. coli* from 2 broilers and one pig; 3 qnrS1 genes were detected in *E. coli* isolates from a broiler (co-expressing a qnr-C gene), a dog and a turtle-dove. The aac-(6′)-Ib-cr gene was detected in an *E. coli* isolated from a mammalian. Seven PMQR-positive isolates showed diminished susceptibility to at least one quinolone, and one was detected in the range of susceptibility against the seven (fluoro)quinolones tested. Three *E. coli* and one *S. enteritidis* were PMQR- and TEM-1 and/or CTX-M-15-producing isolates. An *E. coli* with qnrC, qnrS1, and blaTEM-1 genes and an *E. coli* with qnrC gene were positive for genes coding to class 1 integrons.
Conclusions: This survey showed that PMQR determinants are present in animals from different environments in Portugal, including food-producing animals, with a high frequency (3%) of QnrC-producing isolates. Susceptibility results demonstrate the difficulty to predict the PMQR mechanisms by phenotypic methods. Overall, the study suggests that PMQR genes are undergoing a dissemination process, which needs surveillance.

P1360 Aminoglycoside resistance in *Escherichia coli* in western Norway

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Objective: There had been a steady increase in gentamicin resistance, from 1.2% in 2000 to 4% in 2009, in *E. coli* isolated from blood cultures in Norway. Aminoglycoside modifying enzymes (AME) are important in resistance development and are acquired by horizontal transfer of the respective genes located on plasmids. In *E. coli* the most prevalent AMEs are AAC(6′)-Ib and AAC(3)-II. Our objective was to find the prevalence of the genes encoding these AMEs among Norwegian *E. coli* isolates that were resistant to aminoglycosides (AGs) and to use their resistance profiles for different AGs to consider their value in predicting the responsible AME.
Methods: A total of 108 clinical isolates of *E. coli* from Western Norway were included, consisting of 42 from blood culture isolates that had reduced susceptibility to gentamicin/netilmicin, and 65 isolates from other sites that were non-susceptible to gentamicin/tobramycin, and also produced extended β-lactamases (ESBL). Minimum inhibitory concentrations (MIC) were determined by E-test/MIC test strips for gentamicin, netilmicin, tobramycin, kanamycin, amikacin and streptomycin. The isolates were screened by PCR for aac(6′)-Ib and aac(3)-IIa/c. aac(6′)-Ib was sequenced to identify its variant aac(6′)-Ib-cr, which confers resistance also to fluoroquinolones.
Results: The prevalence of aac(3)-IIa or -IIc was 79.6% (86/108) with no differences seen in the two groups. Resistance to gentamicin was seen in all isolates that had aac(3)-II, however 1–2 isolates were susceptible to netilmicin/tobramycin. aac(6′)-Ib was found in 35.2% of the isolates (38/108), of which 37 were identified as aac(6′)-Ib-cr, with a higher prevalence in ESBL isolates. The substrate specificity was as expected for kanamycin, netilmycin, tobramycin, but amikacin resistance was not found, although the MICs were marginally elevated in 9 of the 38 isolates. Isolates with aac(3)-II had higher MIC values for netilmicin than isolates with aac(6′)-Ib, and aac(6′)-Ib was associated with higher MICs for tobramycin. The prevalence (91.7%) and the degree of quinolone resistance could not be attributed solely to the presence of aac(6′)-Ib-cr.
Conclusion: aac(3)-II and aac(6′)-Ib-cr possibly contributed to the recent increase in gentamicin resistance. The resistance profile, MIC values for netilmicin and tobramycin and association with ESBL might be useful in predicting the AME.

P1361 Phenotypic and genotypic characterisation of aminoglycoside resistance in Norwegian clinical isolates of *Escherichia coli* and *Klebsiella* spp.

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Objectives: Resistance to gentamicin among both *Escherichia coli* and *Klebsiella* spp. in Norway have increased over the last 5 years. The aim of this study was to investigate the susceptibility to different aminoglycosides and the prevalence of aminoglycoside modifying enzymes (AME) among clinical isolates of *Escherichia coli* and *Klebsiella* spp. collected through the Norwegian Surveillance Program for Antimicrobial Resistance (NORM).
Materials and Methods: Collection I included 137 isolates of *E. coli* (blood n = 61, urine n = 44) and *Klebsiella* spp. (blood n = 17, urine n = 15) with reduced susceptibility to gentamicin and/or tobramycin selected from NORM 2009. NORM 2009 included a total of 2,510 *E. coli* isolates (blood n = 1,381, urine n = 1,129) and 1,578 *Klebsiella* spp. isolates (blood n = 571, urine n = 1,007). In addition, 68 ESBL-positive *E. coli* (CTX-M n = 56, SHV n = 4) and *Klebsiella* spp. (CTX-M n = 3, SHV n = 5) isolates from NORM 2007–08 was included as collection II. The susceptibility patterns for gentamicin, tobramycin and amikacin were investigated using Etest. PCR was used for the detection of the following AMEs: aac(6′)-Ib, aac(3)-Ia, aac(3)-II, ant(2″)-Ia and ant(4′)-IIb.
Results: The prevalence's of resistance among *E. coli* isolates in collection I (NORM 2009) were 3,2%, 3,4%, and 0,4% to gentamicin, tobramycin and amikacin, respectively. Among *Klebsiella* spp., 1%, 1,3%, and 0,3% were resistant to gentamicin, tobramycin and amikacin, respectively. In collection II; 45%, 57%, and 0,2% of the ESBL-positive *E. coli* and 38%, 50% and 0% of the ESBL-positive *Klebsiella* spp. were resistant to gentamicin, tobramycin and amikacin, respectively.
aac(3)-II was the most prevalent AME detected in 123 isolates (collection I n = 84, collection II n = 39), followed by aac(6′)-Ib where 53 isolates were positive (collection I n = 30, collection II n = 23). ant(2″)-1a was detected in 3 isolates from collection I. aac(3)-Ia and ant(4′)-IIb were not detected.
Conclusion: The prevalence of aminoglycoside resistance is still low among Norwegian *E. coli* and *Klebsiella* spp. isolates. In contrast, ESBL-positive isolates showed a high prevalence of resistance to gentamicin and tobramycin. Amikacin resistance was low and comparable in both collections. aac(3)-II, which confers resistance to both gentamicin and tobramycin, was the predominant AME in both collections.

P1362 Carbapenemase-producing *Acinetobacter* spp. in Kyoto University Hospital, Japan: the occurrence of metallo-β-lactamases

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Objectives: In recent years, carbapenemase-producing *Acinetobacter* spp. has increased substantially. The carbapenem-hydrolyzing β-lactamases in *Acinetobacter* spp. are either metallo-β-lactamases (MBLs) or oxacillinases (carbapenem-hydrolyzing class D β-lactamases [CHDLs]). Although CHDLs were reported as more major carbapenemase than MBLs, blaIMP-2 detection in *Acinetobacter* spp. was previously reported in Japan. The aim of this study was to evaluate the occurrence of CHDL- and MBL-encoding genes among *Acinetobacter* spp. isolates recovered from Kyoto University Hospital, Japan.
Methods: During 2004–2009, 435 *Acinetobacter* spp. were recovered and 28 (6.4%) isolates were carbapenem-non-susceptible. Only the first isolate was included in this study. 16 of 28 carbapenem-non-susceptible *Acinetobacter* spp. isolates were preserved in our hospital. Carbapenem-non-susceptibility, MIC >4mcg/ml for imipenem or meropenem was determined by the broth microdilution method according to CLSI M100-S20 guideline. These isolates were all screened for CHDL-encoding genes (OXA-23, OXA-24, OXA-51, OXA-58) and MBL-encoding genes (IMP1/2,VIM1/2, SPM-1, SIM-1, GIM-1), and confirmed by sequencing. Clonality of *A. calcoaceticus-A. baumannii* complex was analyzed by MLST.
Results: 16 carbapenem-non-susceptible *Acinetobacter* spp. included 8 A. genomic sp.3, followed by 2 *A. baumannii*, 2 *A. lwoffii*, and 4 other *Acinetobacter* spp. CLDL- or MBL-encoding genes were detected in 14 (87.5%) isolates. All 8 of carbapenem-non-susceptible A. gen. sp. 3 harboured MBL-encoding gene (1 IMP-1 and 7 IMP-2), and 1 isolate