High frequency of the combined presence of QRDR mutations and PMQR determinants in multidrug-resistant *Klebsiella pneumoniae* and *Escherichia coli* isolates from nosocomial and community-acquired infections

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**Abstract**

Plasmid-mediated quinolone resistance (PMQR) determinants combined with mutations in quinolone resistance-determining regions (QRDRs) and clonal dissemination were investigated in 40 fluoroquinolone-resistant *Klebsiella pneumoniae* and *Escherichia coli* isolates from nosocomial and community-acquired infections. We observed nucleotide substitutions in *gyrA* (Ser83Ile, Val37Leu, Lys154Arg, Ser171Ala, Ser19Asn, Ile198Val, Ser83Tyr, Ser83Leu, Asp87Asn and Asp87Gly) and *parC* genes (Ser80Ile, Glu84Lys, Ala129Ser, Val141Ala and Glu84Gly). Two novel substitutions were detected in the *gyrA* gene (Val37Leu and Ile198Val). The presence of PMQR genes predominated in community isolates (55.5 %). In addition to the frequent presence of the class 1 integron in isolates from community-acquired infections, the genetic similarity results obtained by PFGE showed high genomic diversity. This study suggests that management of multidrug-resistant *Enterobacteriaceae* isolates from the community are a possible source of genetic mobile elements that carry genes that confer resistance to fluoroquinolones. More attention should be paid to the surveillance of community-acquired infections.

*Klebsiella pneumoniae* and *Escherichia coli* are common opportunistic pathogens responsible for nosocomial and community-acquired infections [1, 2]. In both species, resistance to multiple classes of antimicrobials has been emerging as a global problem, probably due to mutations and other resistance determinants [3, 4].

Resistance to fluoroquinolone drugs among members of the family *Enterobacteriaceae* has increased in recent years, following the extensive and widespread use of antibiotics [5]. Resistance commonly occurs through mutations in the genes of antimicrobial target enzymes (DNA gyrase and topoisomerase IV), but can also be plasmid-mediated [6]. Among the plasmid-mediated quinolone resistance (PMQR) determinants, in the family *Enterobacteriaceae* the *qnrS*, *qnrB* and *aac(6’)-Ib-cr* genes have been implicated [1, 7]. Although the PMQR mechanism only confers low-level resistance to fluoroquinolones, its association with the occurrence of mutations in quinolone resistance-determining regions (QRDR) can lead to clinically relevant resistance levels [2].

The impact of antimicrobial resistance on clinical outcomes is the subject of ongoing investigation [8]. Inappropriate empirical therapy has been associated with increased mortality in multidrug-resistant (MDR) organism infections [9, 10]. Delays in starting appropriate therapy may contribute to the persistence of infection and increased length of hospital stay for patients [11].

In this study, we aimed to investigate the presence of PMQR determinants and QRDR mutations in hospital- and community-acquired infections with fluoroquinolone-resistant *K. pneumoniae* and *E. coli*, in addition to the clonal spread of these strains.

Active surveillance was conducted from April to October 2014 for the detection of nosocomial and community infections by fluoroquinolone-resistant *K. pneumoniae* and
E. coli strains in patients that attended the Clinical Hospital, Federal University of Uberlândia (UFU-HC), Brazil. To evaluate appropriate and inappropriate antimicrobial therapies and their impact on outcomes for patients with these infections, only the first episode of each infection was considered.

The criteria used to define the origin of infection (nosocomial or community), the MDR phenotype, previous antibiotic use, antimicrobial therapy, 30-day mortality, and the MIC50 and MIC90 were based on Bouchillon et al. [12], Magiorakos et al. [13], Gulen et al. [14], Gilbert et al. [15], Lodise et al. [16] and Schwarz et al. [17], respectively.

Microbial identification and antimicrobial susceptibility tests were performed on the VITEK II system (bioMérieux, Brazil). The minimum inhibitory concentration (MIC) was determined for 40 randomly selected isolates by the broth microdilution method according to Capuano [18], with modifications, and interpreted according to CLSI [19].

Forty clinical fluoroquinolone-resistant isolates were selected randomly (20 K. pneumoniae and 20 E. coli). DNA extraction was performed using the PureYield Plasmid Miniprep system (Promega, Brazil). Amplification of the class 1 integron and PMQR markers [intI, qnrA, qnrB, qnrC, qnrD, qnrS, gfpA and aac(6')-Ib-cr] was performed using the primers listed in Table S1 (available in the online Supplementary Material). Amplifications were performed on a Mastercycler Personal (Eppendorf) using the following programme: initial denaturation at 95 °C for 2 min followed by 30 cycles of 30 s at 95 °C; 1 min at the annealing temperature (51 °C for qnrA, qnrB, qnrC, qnrS and qnrD; 52 °C for gfpA and aac(6')-Ib-cr; 56 °C for intI); 1 min at 72 °C; and a final extension step of 5 min at 72 °C. Multiplex PCR was performed for the genotypic characterization of qnrA, qnrB, qnrC and qnrS, while individual PCRs were carried out for the other genes [intI, qnrD, gfpA, and aac(6')-Ib-cr]. The amplified PCR products were visualized by electrophoresis on a 1.5 % agarose gel using the t-Pix EX photo documentation system (Loccus Biotechnology, Brazil). The PCR products of the target regions for QRDR mutations (gyrA and parC) and the aac(6')-Ib gene were sequenced for K. pneumoniae (13 and 10 isolates, respectively) and E. coli (4 and 7 isolates, respectively) (the primers are listed in Table S1), using an ABI-PRISM 3100 genetic analyzer automatic sequencer (Applied Biosystems, USA). Sequences were edited and alignment was carried out with Genaious version 9.1.3 software.

Isolates were typed according to the protocols described by Galetti [20] with modifications. DNA fragments were separated on 1 % (w/v) agarose gels in 0.5× Tris-borate-ethylenediamine tetra-acetic acid (EDTA) (TBE) buffer using the CHEF DR III apparatus (Bio-Rad, USA) at 6 V cm⁻¹, pulsed from 5 s to 40 s, for 21 h at 12 °C. Gels were stained with ethidium bromide and photographed under ultraviolet light. Computer-assisted analysis was performed using BioNumerics 5.01 software (Applied Maths, Belgium).

Comparison of the banding patterns was accomplished by the unweighted pair group method with arithmetic averages (UPGMA) using the Dice similarity coefficient.

The present study detected 129 episodes (48 K. pneumoniae and 81 E. coli) of nosocomial and community-acquired infections by fluoroquinolone-resistant K. pneumoniae and E. coli isolates. The distribution of the episodes and the number of strains analysed are given in Fig. S1. The clinical and demographic characteristics of the study population are shown in Table 1. The mean age of patients with nosocomial infection was 48.7 years (variation 0–87); most patients were males (58.8 %), and the mean length of stay was 50.7 days. Among those with community-acquired infection, the mean hospital stay was 5.3 days. Of the first group of patients, 21.6 % were admitted to the intensive care unit (ICU) at some point, and had more than one co-morbidity, in particular stroke and nephropathy. Similar frequencies were observed for these co-morbidities in patients with community-acquired infections. Urinary infections predominated in both the hospital and community groups, and of these, 11.8 % were using a urinary catheter. Surgical procedures and the use of invasive procedures were frequent in the population with nosocomial infections, especially central venous catheters, nasoenteral/nasogastric probes, tracheostomies and mechanical ventilation. Most patients with a nosocomial infection had previously used antimicrobials and had total and 30-day mortality rates of 29.4 and 19.6 %, respectively.

Antimicrobial therapy was considered to be inappropriate in the majority of cases (56.9 %, corresponding to 60.0 % in infections caused by K. pneumoniae and 53.8 % by E. coli). The results of this study also showed that 15/51 (29.4 %) patients died during the hospitalization period, with 46.7 % of them receiving inappropriate therapy, especially in cases of sepsis (57.1 %) (data not shown).

The main aetiologial agent of community-acquired infections was E. coli (64.2 %), while K. pneumoniae predominated among nosocomial infections (60.4 %). In both groups, urinary tract infection was the most common type of infection (72.9 %; 94/129).

Regarding the resistance profile to antimicrobials, the frequency of resistant K. pneumoniae isolates of nosocomial origin was higher than 75 % for five of the antibiotics tested (ampicillin 100 %, cefepime 100 %, ceftriaxone 100 %, piperacillin/tazobactam 93.1 % and gentamicin 75.9 %), while E. coli resistance rates were 86.2 % for ampicillin, 44.8 % for amoxicillin/sulbactam and 41.4 % for cefepime and ceftriaxone. With regard to community-acquired K. pneumoniae strains, resistance was highest for ampicillin (100 %), nitrofurantoin (100 %), cephalexin (78.9 %), ceftriaxone and cefepime (73.6 %). For E. coli, resistance was highest for ampicillin (94.2 %) and sulfamethoxazole (75.0 %). Additionally, the analysis of antimicrobial susceptibility showed that most strains had an MDR profile (96.1 %); for isolates acquired in the hospital and the community the frequency was 93.1 and 98.6 % respectively.
Table 2 summarizes the results for MIC, presence of the class 1 integron and PMQR determinants, as well as the distribution of substitutions among *Enterobacteriaceae* species and different patterns of substitutions in GyrA and ParC in clinical fluoroquinolone-resistant strains included in this study.

The presence of PMQR genes was detected in 68.4% (13/19) of the nosocomial and community-acquired *K. pneumoniae* isolates tested, with the following frequency: *qnrA* (5%), *qnrB* (45%), *qnrD* (15%), *qnrS* (15%), *aac(6’)-Ib* (5.2%), *aac(6’)-Ib-cr* (31.6%) and *aac(6’)-Ib* (15.9%). Among the *E. coli* isolates, PMQR determinants were found in eight strains, with these being *qnrB* (15%), *qnrS* (15%), *aac(6’)-Ib-cr* (26.3%) and *aac(6’)-Ib* (5.3%). The coexistence of different PMQR genes was observed in 35% of *K. pneumoniae* (7/20) strains and 5% of *E. coli* isolates (1/20).

DNA sequencing of the QRDR of GyrA and ParC genes of the 17 PMQR-positive *Enterobacteriaceae* isolates (17/21) revealed point mutations involving amino acid (aa) substitutions. The substitutions were detected in 100% of isolates with ciprofloxacin MICs ≥4 µg l⁻¹. In total, 15 deduced aa substitutions in either the gyrA or parC genes were found in *K. pneumoniae* and *E. coli* isolates. In the case of gyrA, 10 substitutions were detected (Ser83Ile, Val37Leu, Lys154Arg, Ser171Ala, Ser19Asn, Ile198Val, Ser83Tyr, Ser83Leu, Asp87Asn and Asp87Gly), and 2 or more mutations were observed in all isolates. Furthermore, strains 172 and 177, from nosocomial and community-acquired infections, respectively, carried up to five mutations at the same position: 19 (Ser→Asn), 83 (Ser→Ile), 154 (Lys→Arg), 171 (Ser→Ala) and 198 (Ile→Val). In the case of parC, five aa substitutions were detected: Ser80Ile, Glu84Lys, Ala129Ser, Val141Ala and Glu84Gly. In only two strains were mutations in the parC not detected, and double mutations were observed in 33.3% of isolates (5/15). Two substitutions that had not been reported to date were observed, one at codon 37 (Val→Leu) of the gyrA gene from a community-acquired *K. pneumoniae* infection, and another at codon 198
The presence of the PMQR has been a growing and rapidly spreading problem, with 95% of Enterobacteriaceae species being resistant to fluoroquinolone. Furthermore, E. coli showed a higher number of mutations in the parC gene.

The presence of the intI gene was observed in 85% of K. pneumoniae and 95% of E. coli strains. The minimum concentration to inhibit 50 and 90% of the 20 strains of each species was 128 µg ml⁻¹ and 512 µg ml⁻¹ for K. pneumoniae and E. coli, respectively. The genetic similarity results obtained by PFGE showed a polyclonal profile for both species, with 13 distinct genotypic profiles for K. pneumoniae (A–M) and 9 for E. coli (A–I), and none of the clones showing subtypes (Fig. 1a, b).

The common use of antibiotics in the hospital environment has led to the exponential increase in resistance in microorganisms [21, 22]. Among the microorganisms of the family Enterobacteriaceae, resistance to fluoroquinolones has been a growing and rapidly spreading problem, and is often associated with the accumulation of different determinants, such as QRDR mutations and, more recently, the presence of PMQR determinants [6]. Several surveillance studies have been conducted to identify the presence of QRDR mutations and PMQR determinants in Enterobacteriaceae isolates from nosocomial infections worldwide; however, less emphasis has been placed on community-acquired strains [1, 2, 23, 24].

PMQR is a phenomenon of increasing importance [24], and the most common PMQR genes found among Enterobacteriaceae isolates have been qnrS, qnrB and aac(6')-Ib-cr [1, 7]. Our data show that the frequency of PMQR genes was high (55.3%) in K. pneumoniae and E. coli strains, even among community-acquired strains (55.5%). According to the literature, high frequencies of PMQR genes have been observed in strains derived from the hospital, as shown in the study by Yang and collaborators [25] on K. pneumoniae and E. coli strains (79.4%). The relatively high prevalence of

<table>
<thead>
<tr>
<th>Strains</th>
<th>Origin</th>
<th>Source</th>
<th>β-lactam* and aminoglycoside resistance</th>
<th>PMQR genes</th>
<th>QRDR mutations</th>
<th>Class I integron</th>
<th>MIC* (µg ml⁻¹) CIP</th>
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</thead>
<tbody>
<tr>
<td>K. pneumoniae</td>
<td>79 Community Urine</td>
<td>CMP, CTR, GEN, AMP, CEF, TZP</td>
<td>qnrB</td>
<td>SerR3Ile, Val53Leu, Lys154Arg, Ser171Ala</td>
<td>SerR3Ile –</td>
<td>–</td>
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<tr>
<td>112 Community Urine</td>
<td>CMP, CTR, AMP, CEF, TZP</td>
<td>qnrR, aac(6')-Ib-cr</td>
<td>SerR3Ile, Lys154Arg, Ser171Ala</td>
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<tr>
<td>129 Community Urine</td>
<td>CMP, CTR, GEN, AMP, CEF, TZP</td>
<td>qnrA, aac(6')-Ib-cr</td>
<td>Ser19Asn, Ser33Ile, Ser171Ala</td>
<td>Ser19Asn –</td>
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<tr>
<td>164 Community Urine</td>
<td>CMP, CTR, GEN, AMP, CEF, TZP</td>
<td>qnrS</td>
<td>Lys154Arg, Ser171Ala</td>
<td>Ser19Asn, Ser33Ile, Lys154Arg, Ser171Ala, Ile198Val</td>
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<td>–</td>
<td>128</td>
</tr>
<tr>
<td>177 Community Urine</td>
<td>CMP, CTR, GEN, AMP, CEF, TZP</td>
<td>–</td>
<td>–</td>
<td>SerR3Ile, Lys154Arg, Ser171Ala</td>
<td>Ser80Ile –</td>
<td>–</td>
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<tr>
<td>04 Nosocomial Blood</td>
<td>CMP, CTR, GEN, AMP, TZP, SAM</td>
<td>qnrB, qnrS</td>
<td>SerR3Ile, Ser19Asn, Lys154Arg, Ser171Ala</td>
<td>Ser19Asn, Lys154Arg, Ser171Ala, Ile198Val</td>
<td>SerR3Ile, Ile198Val</td>
<td>–</td>
<td>128</td>
</tr>
<tr>
<td>33 Nosocomial Tracheal aspirate</td>
<td>CMP, CTR, GEN, AMP, TZP, SAM</td>
<td>qnrB, qnrD</td>
<td>SerR3Ile, Ser19Asn, Lys154Arg, Ser171Ala</td>
<td>Ser19Asn, Lys154Arg, Ser171Ala, Ile198Val</td>
<td>SerR3Ile, Ile198Val</td>
<td>–</td>
<td>128</td>
</tr>
<tr>
<td>55 Nosocomial Abdominal secretion</td>
<td>CMP, CTR, GEN, AMP, TZP, SAM</td>
<td>qnrB, qnrD</td>
<td>SerR3Ile, Ser19Asn, Lys154Arg, Ser171Ala</td>
<td>Ser19Asn, Lys154Arg, Ser171Ala, Ile198Val</td>
<td>SerR3Ile, Ile198Val</td>
<td>–</td>
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<tr>
<td>67 Nosocomial Blood</td>
<td>CMP, CTR, GEN, AMP, TZP, SAM</td>
<td>qnrB, qnrS</td>
<td>SerR3Ile, Ser19Asn, Lys154Arg, Ser171Ala</td>
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<td>SerR3Ile, Ile198Val</td>
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<td>114 Community Urine</td>
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<td>Ser19Asn, Lys154Arg, Ser171Ala, Ile198Val</td>
<td>SerR3Ile, Ile198Val</td>
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<tr>
<td>126 Nosocomial Urine</td>
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<td>qnrB, qnrS</td>
<td>SerR3Ile, Ser19Asn, Lys154Arg, Ser171Ala</td>
<td>Ser19Asn, Lys154Arg, Ser171Ala, Ile198Val</td>
<td>SerR3Ile, Ile198Val</td>
<td>–</td>
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<td>172 Nosocomial Blood</td>
<td>CMP, CTR, GEN, AMP, TZP, SAM</td>
<td>qnrB, qnrS</td>
<td>SerR3Ile, Ser19Asn, Lys154Arg, Ser171Ala</td>
<td>Ser19Asn, Lys154Arg, Ser171Ala, Ile198Val</td>
<td>SerR3Ile, Ile198Val</td>
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<td>E. coli</td>
<td>59 Community Urine</td>
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<td>qnrS</td>
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<td>Ser80Ile, Ala129Ser, Val414Ala</td>
<td>Ser80Ile</td>
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<td>72 Community Urine</td>
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<td>ND</td>
<td>Ser80Ile, Ala129Ser, Val414Ala</td>
<td>Ser80Ile</td>
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<td>102 Community Urine</td>
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<td>qnrS</td>
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<td>Ser80Ile</td>
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<tr>
<td>131 Community Urine</td>
<td>CMP, CTR, AMP, CEF</td>
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<td>Ser80Ile</td>
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<td>152 Community Urine</td>
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<td>46 Nosocomial Urine</td>
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<td>06 Nosocomial Urine</td>
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<td>128</td>
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<tr>
<td>75 Nosocomial Tracheal aspirate</td>
<td>CMP, CTR, AMP, SAM</td>
<td>qnrB, qnrS</td>
<td>Ser80Ile, Ala129Ser, Val414Ala</td>
<td>Ser80Ile</td>
<td>128</td>
<td></td>
<td></td>
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</tbody>
</table>

*Cephalosporin (third and fourth generation).

PMQR, plasmid-mediated quinolone resistance; QRDR, quinolone-resistance determining region; MIC, minimum inhibitory concentration to ciprofloxacin; CMP, cefepime; CTR, ceftaxime; GEN, gentamicin; AMP, ampicillin; CEF, cephaplatin; TZP, piperacillin–tazobactam; SAM, ampicillin–sulbactam; –, negative; ND, not determined.
aac(6′)-Ib-cr is not surprising, since the literature has shown that this gene is often present on the same plasmid as the \( \text{bla}^{\text{ESBL}} \) gene [26]. In a previous study carried out by our team, this association also showed a high frequency of \( \text{bla}^{\text{ESBL}} \)-producing family Enterobacteriaceae isolates from UFU-HC [27].

With respect to the target-site mutations in fluoroquinolone-resistant \( K. \) pneumoniae and \( E. \) coli isolates, we observed that most mutations were consistent with those previously published in the literature [2, 28, 29]. Ser to Ala at codon position 171 in \( \text{gyrA} \) gene was the most frequent (70.6%) substitution detected in our isolates, while Ser to Ile at position 80 was the main substitution in the \( \text{parC} \) gene (76.4%). A double \( \text{gyrA}/\text{parC} \) mutation was also common, confirming the accumulation of mutations and the mechanisms associated with different PMQR genes [2, 30]. In addition, our study showed mutations corresponding to a

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Fig. 1. (a) UPGMA dendrogram of the PFGE profiles of 15 Klebsiella pneumoniae isolates used in this study using the Dice coefficient under 1% tolerance and 1% optimization. (b) UPGMA dendrogram of the PFGE profiles of nine Escherichia coli isolates used in this study using the Dice coefficient under 1% tolerance and 1% optimization. A similarity coefficient of 80% was chosen for cluster definition.
Val to Leu substitution at position 37 in the gyrA gene in a community-acquired MDR K. pneumoniae strain, and Val to Val at codon 198 in one nosocomial and two community-acquired strains, that, to the best of our knowledge, have not yet been described in the literature.

In fact, we describe fluoroquinolone-resistant strains with high MICs, particularly in those from the community, and independent of species. Although we did not find an association between MIC and PMQR determinants, even with the diversity of changes that were found in the gyrA and parC genes, we suggest that the determinants associated with QRDR mutations are relevant and may increase resistance levels to fluoroquinolones, besides suggesting the existence of other mechanisms of resistance in these strains [24, 31, 32].

In addition to the high frequency of PMQR genes, the class 1 integron sequence was also present at high frequency in these strains, regardless of their origin. PMQR determinants have been found inserted into the integron cassette [24], especially qnrA, qnrB, and aac(6’)-Ib-cr [6, 33]. In our study, PMQR genes and the class 1 integron sequence were identified in most fluoroquinolone-resistant strains. However, the presence of PMQR determinants inserted into the integron cassette was not investigated.

A genetic study of bacterial isolates harbouring PMQR determinants revealed great genomic diversity in the Enterobacteriaceae species analysed (13 and 9 pulsotypes for K. pneumoniae and E. coli, respectively), and closely related clones were only observed for K. pneumoniae (A and B). Our data clearly suggest the dissemination and coexistence of different clones. In the hospital investigated in this study, the reasons for the unbridled spread were unknown, although inappropriate use of antibiotics and poor adherence to infection control policies are probably among the reasons.

In conclusion, a high frequency of the combined presence of PMQR, class 1 integrons and QRDR mutations in highly resistant K. pneumoniae and E. coli strains was observed in infections, regardless of whether the infection was hospital- or community-acquired. This study suggests that the community may be a source of MDR Enterobacteriaceae isolates for the hospital and that these isolates may be in turn be sources of genetic mobile elements (plasmids and/or class 1 integrons) that carry genes that confer resistance to fluoroquinolones. More attention should be paid to the surveillance of community-acquired isolates.

References

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