Molecular Analysis of Quinolones and Fluoroquinolones Resistance in Uropathogenic *Escherichia coli*


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Introduction: Urinary tract infections (UTI) have a high incidence in the population and in most cases, are caused by Enterobacteriaceae members, especially *Escherichia coli*. The main choice in the treatment of UTIs is quinolones and fluoroquinolones. These agents target two enzymes involved in DNA supercoiling that leads bacteria to death, DNA Gyrase, consisting of two GyrA and two GyrB subunits, and Topoisomerase IV, composed of two ParC and two ParE subunits. Mutations in these genes can confer high levels of resistance. Other mechanism of resistance involves genes carried by plasmids such as *qnr*. These genes encode proteins that protect the target enzymes of quinolones. Another gene (*aac(6')-Ib-cr*) also present in plasmids can change the chemical structure of certain fluoroquinolones, interfering with their efficiency. The aim of this study was to evaluate the patterns of resistance to quinolones and fluoroquinolones in samples of uropathogenic *E. coli*. Material and Methods: This study analyzed 55 samples of *E. coli* isolated from patients with UTI collected in Curitiba, PR, Brazil. The phenotypical resistance profile was performed using the Kirby Bauer method. The resistance mechanisms were determined by multiplex PCR, followed by sequencing of the amplified fragments. Results and Discussion: The resistance to nalidixic acid, norfloxacin, ciprofloxacin and ofloxacin was observed in 69.1% of the samples. Molecular analysis showed mutations in *gyrA* (codons 83 and 87) and *parC* (codon 80). The presence of genes *qnr's* and *aac(6')-Ib-cr* was also investigated. Two samples were positive for *aac(6')-Ib-cr*. The presence of *aac(6')-Ib-cr* in Brazil was first reported in 2012 in Belo Horizonte, MG. There were no positive samples for the *qnr's* genes. Conclusions: These results show a high incidence of resistance to quinolones and the ability to horizontally transfer acquired resistance between the bacterial strains.

Keywords: Resistance, quinolones, mutation, *aac (6')-Ib-cr*

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