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Detection of qnr genes and gyrA mutation to quinolone phenotypic resistance of UTI pathogens in Bangladesh and the implications

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Abstract

Background: Plasmid-mediated quinolone-resistant (PMQR) genes and mutations within the quinolone resistance-determining regions (QRDRs) resulted in the advent of quinolone-resistant pathogenic microbes. This research was designed to assess the roles of three PMQR genes, *qnrA*, *qnrB*, and *qnrS*, and any mutation in the *gyrA* gene in the QRDR as a process of quinolone/fluoroquinolone resistance to urinary tract infection (UTI) bacteria in Bangladesh to guide future management of UTIs. **Methods:** Pathogens from UTIs were isolated and identified, and their phenotypic antibiotic susceptibilities were tested for lomefloxacin, ofloxacin, ciprofloxacin, and nalidixic acid. Polymerase chain reaction (PCR) detected the *qnrA*, *qnrB*, and *qnrS* genes. PCR and sequencing were performed to evaluate any mutation within the QRDRs of the *gyrA* gene. **Results:** Of 100 UTI bacteria, phenotypic resistance was observed in 95.0%, 89.0%, 83.0%, and 71.0% against lomefloxacin, nalidixic acid, ofloxacin, and ciprofloxacin, respectively. PMQR genes *qnrS*, *qnrA*, and *qnrB* genes were found in 54.0%, 1.0%, and 4.0% of isolates, respectively. Sequencing the *gyrA* gene revealed single mutation (Ser-83 to Leu) and double mutations (Ser-83 to Leu and Asp-87 to Asn). PMQR genes showed a statistically nonsignificant association with phenotypic resistance. **Conclusions:** This study confirms the presence of QRDR mutations that were independent of PMQR genes. Consequently, high resistance against quinolones among uropathogens is evident, and their future use needs to be moderated. © 2022. Tanjum Ara Haque et al. All Rights Reserved.

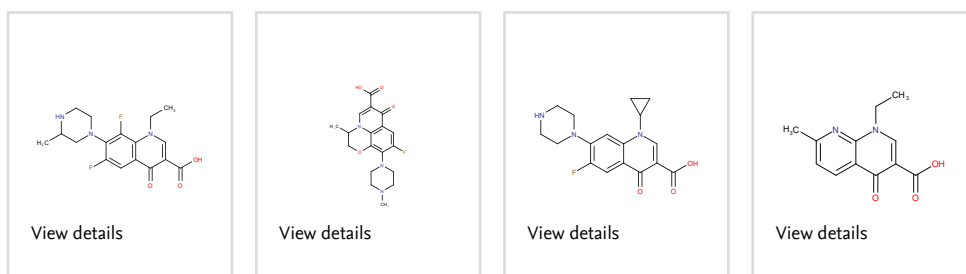
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Bangladesh; PMQR; *qnr*; QRDR; Quinolone; UTI

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