

Antimicrobial resistance to fluoroquinolones in *Salmonella* Infantis isolates through complete genomic analysis

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Salmonella Infantis is associated with foodborne infections in humans worldwide, particularly in Europe and the United States. Its transmission is mainly related to chicken meat, and it can be isolated throughout the poultry production chain. The indiscriminate use of antimicrobial compounds in poultry farming contributes to the rise in antimicrobial resistance (AMR), posing a threat to the health of both animals and humans. Fluoroquinolone (CF) resistance is of particular concern, as this class of antimicrobials is on the World Health Organization's (WHO) list of greatest global health concerns. Whole Genome Sequencing (WGS), among numerous approaches, allows us to understand the mechanisms of AMR. This study aimed to describe, through WGS, the characteristics of antimicrobial resistance to fluoroquinolones in *Salmonella* Infantis isolates obtained from broiler carcasses. Six isolates of *S. infantis*, obtained from chickens slaughtered in a Brazilian export slaughterhouse, were subjected to WGS. The isolates were characterized by serotyping at the Adolfo Lutz Institute, according to the White-Kauffmann-Le Minor scheme, and submitted to the WGS, where they were confirmed by the analysis of their raw sequencing readings using the SeqSero pipeline. To identify the types of MLST (ST) sequences and antimicrobial resistance genes, the PubMLST and ResFinder v3.2 databases were used. MLST typing showed that all *S. infantis* isolates belonged to ST32, a sequence type frequently identified within this serovar. The genes *gyrA*, *gyrB*, *parC* and *parE*, responsible for genotypic resistance to quinolones and reduced susceptibility to antimicrobials such as ciprofloxacin in phenotypic tests, were found in all sequenced isolates. The results of this study demonstrate that *Salmonella* Infantis exhibits a strong mechanism of resistance to fluoroquinolones, mostly attributed to the accumulation of chromosomal mutations in the target topoisomerases, resulting in a highly resistant bacterium that is difficult to control.

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