TITLE: RESISTANCE TO QUINOLONES IN Salmonella spp. ISOLATED FROM LIVE BROILERS AND CARCASES UNDER FEDERAL INSPECTION

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ABSTRACT:

The DNA-gyrase and Topoisomerase IV enzymes are involved in the bacterial DNA replication process, being essential to bacterial survival. Quinolones are antimicrobials whose main mechanism of action is the inhibition of these enzymes. These drugs are widely used in human and veterinary medicine for the treatment of bacterial infections. Resistance to these antimicrobials increases failure to treat these infections. Mutations in enzymes DNA-gyrase and Topoisomerase IV may develop bacterial resistance to guinolones. These mutations result in the substitution of aminoacids in the subunits encoded by the gyrA, gyrB, parC or parE genes, by altering the antimicrobial binding site. The aim of this study was to verify the resistance to guinolones, enrofloxacin (ENO), ciprofloxacin (CIP) and nalidixic acid (NAL), by mutation of gyrA, gyrB, parC and *parE* genes in *Salmonella* spp. strains isolated from live poultry and carcasses. In this study, 77 isolates of Salmonella spp. were used, being 20 of live broilers (cloacal swabs) and 57 of carcasses from slaughterhouses, under Federal Inspection. The following serotypes were identified: Salmonella Saint Paul (29/77), Salmonella Heidelberg (27/77), Salmonella Anatum (9/77), Salmonella Cerro (5/77), Salmonella Senftenberg (5/77), Salmonella enterica (O: 4,5) (1/77) e Salmonella enterica (O: 9.12) (1/77). Of the total strains studied, 19.5% (15/77) were resistant to ENO, 7.8% (6/77) to CIP and 33.8% (26/77) to NAL by Disk Diffusion Test. The 15 ENO resistant strains were selected for the detection of gyrA, gyrB, parC and parE genes by PCR and to genetic sequencing to identify mutations in these genes. Of 15 strains 33.3% (5/15) had point mutations in the gyrA gene and 6.7% (1/15) presented a point mutation in the parC gene. None of the 15 strains had mutations in the gyrB and parE genes. The existence of only point mutations in some genes of the analyzed strains is in agreement with the phenotypic resistance observed to NAL, but not with resistance to ENO and CIP. Other mechanisms may be related to the resistance found to ENO and CIP quinolones and additional studies are necessary to investigate the existence of those other mechanisms.

Keywords: DNA-gyrase, Topoisomerase, Mutation, Quinolones