Abstract

Salmonella are usually transmitted to human by the consumption of contaminated foods and water. The prevalence of *Salmonella* varies depending on the water supply, waste disposal, food preparation practices, and climate. The highest incidence rates occur in children younger than 5 years of age, particularly those younger than 1 year, and in individuals older than 70 years of age.

Human Salmonellosis caused by non-typhoidal *Salmonella* species (NTS) occurs with increasing frequencies in developed countries. The emergence of fluroquinolone resistant *Salmonella* poses a serious problem. Fluroquinolones are considered the treatment of choice in cases of acute salmonellosis. The increase in antimicrobial resistance has lead many countries to initiate surveillance program to monitor dissemination and detect the evolution of bacterial resistance.

In this study, we evaluated the antimicrobial susceptibility of (NTS) following the Clinical Laboratory Standard Institute recommendations. In addition, we determined the molecular mutations on *gyrA* that caused resistance to quinolones by PCR. To confirm and specify the changes that occurred on *gyrA,* representative samples were sequenced.

Our results revealed that *Salmonella* serogroups C and D were the most common among clinical isolates, while serogroups C and B were the most common among food isolates. Antimicrobial susceptibility testing revealed that all strains tested were susceptible to ceftriaxone, most of the strains were susceptible to chloramphenicol, gentamicin and trimethoprim sulfametaxazole. To evaluate resistance to quinolones, nalidixic acid and ciprofloxacin were used. NTS were divided into three main groups designated as: Susceptible to both quinolones (SS), resistant to nalidixic acid but susceptible to

ciprofloxacin (RS) and resistant to both (RR). Antimicrobial susceptibilities in the 3 groups in clinical and food isolates were 38% and 51% (SS), 30% and 30% (RS) and 30% and 15% (RR) respectively.

The *gyrA* gene was amplified and resolved by agarose gel electrophoresis. A sharp band of 630 bp was obtained and restricted by *Hin*f I enzyme. Subsequent agarose gel electrophoresis revealed various size bands depending on the pattern of susceptibility or resistance of *Salmonella* isolates to nalidixic acid and ciprofloxacin. The SS pattern revealed 3 major bands of 250 bp, 150 bp and 100 bp. A faintly appearing fourth band of about 130 bp was also seen. Identical results were obtained with the RS group, with the RR pattern; two major bands of 350 bp and 150 bp were evident. A faintly appearing third band of 130 bp was also seen.

The sequence results obtained for the SS pattern was normal with no mutations. The sequence obtained with the RS pattern revealed a mutation at position 87. A substitution of G to A (GAC became TAC). The sequence obtained with the RR pattern revealed two mutations at positions 83 and 87. At position 83, a substitution of C to T (TCC became TTC) was detected. At position 87, the same type of mutation occurred as in the RS pattern was detected, a substitution of G to A (GAC became TAC).

The level of resistance to quinolones in this study among NTS isolates is alarming. This warrants the prohibition of the use of quinolones in chicken feed and the restriction of administring of quinolones without a prescription.