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Non-typhoid *Salmonella*: Changing Epidemiology and Resistance

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Antimicrobial resistance of non-typhoid *Salmonella* is a public health concern worldwide. *Salmonella* infections are primarily zoonotic in origin. When resistance is present, it has often been acquired prior to transmission of the organism through the food chain to humans. Since 1991 there has been an emergence in cattle and humans in many areas of the world of multidrug-resistant (MDR) strains of DT104 of R-type ACSSuT. In contrast to early predominant DTs 29 and 204/193/204c, the ACSSuT resistance genes in DT104 are chromosomally located. Although some variants have been identified, when studied by PFGE the majority of MDR *Salmonella enterica* serovar Typhimurium DT104 isolates are characterized by a distinctive *Xba*I-generated macrorestriction fingerprint. Over the last 15 years this particular clone has caused outbreaks of infection in food animals and humans in numerous European countries. In 1996 infections with MDR DT104 were recognized in cattle and humans in North America, both in Canada and United States. Since 1992 an increasing number of isolates of MDR *S. Typhimurium* have exhibited decreased susceptibility to ciprofloxacin. This property is chromosomally encoded as a result of mutations in the quinolone resistance determining region (QRDR). In MDR DT104 of R-type ACSSuT, resistance are contained in a 43-kb genomic island (salmonella genomic island 1[SGI]). Of note in recent years has been the identification of SGI1 in several different *Salmonella* serovars, including Agona, Albany, and Paratyphi B, Newport, and Derby. Such strains have caused infections both in humans and cattle. In fact, multidrug resistance in *Salmonella* in developed countries is not confined to *S. Typhimurium* DT104 and related strains. In the United States multidrug resistance has been reported in serovars Saintpaul, Heidelberg, Newport, and Typhimurium. Notably in 2000, the first case of domestically acquired ceftriaxone-resistant *Salmonella* in a case of human salmonellosis in the United States, involving the transmission of a resistant strain from cattle to the child of a veterinarian was reported. More recently MDR Newport with plasmid-encoded resistance to ceftriaxone has caused numerous infections in both cattle and humans in North America. This organism commonly shows R-type of ACSSuT, with additional resistance to third-generation cephalosporins mediated by the CMY-2 -lactamase gene (so called MDR-AmpC phenotype). Similarly there have been increasing reports of resistance to extended-spectrum-lactamases in *Salmonella* from humans and food animals in numerous countries worldwide. For example CTX-M-9, -15 and -17 to -18 enzymes have recently been reported in six different serovars isolated from humans in the United Kingdom, and CTX-M-like enzymes have been reported in serovar Virchow in Spain and in serovar Anatum in Taiwan. In Taiwan a particularly alarming development has been the emergence of a highly virulent strain of *S. Choleraesuis* displaying high-level resistance to ceftriaxone. It was confirmed that the ceftriaxone resistance was conferred by CMY-2 -lactamase gene located on a transferable 138-kb plasmid. *S. Choleraesuis* did not contain SGI1; however, the appearance of the plasmid-mediated MDR-AmpC phenotype is a cause for concern because the multidrug resistance could disseminate through the transfer of plasmid among different *Salmonella* serovars as well as other Enterobacteriaceae.