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## Chapter

# Salmonellosis and Campylobacteriosis, Emerging Zoonosis in the World and Current Situation in Mexico

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## Abstract

Salmonellosis and campylobacteriosis are the furthestmost common zoonotic infections around the world that are transferred. The spread of *Salmonella enterica* serotypes Enteritidis (SE) and Typhimurium (ST) has increased dramatically in the last 50 years due to the consumption of food contaminated and the emergence of SE and ST infections with multiple antibiotic resistance. Retrospective investigations imply an epidemiological link between people and poultry. It has been argued that farm modernization and global exports of progenitor birds have had a vital role in spreading SE and ST. On the other hand, campylobacteriosis is more common than salmonellosis in affluent countries. *Campylobacter jejuni* has been identified as the primary cause of acute diarrheal illnesses, frequently associated with animal-derived foods, particularly poultry meat. The current review examines immunological and molecular biological techniques that allow for the quick detection of asymptomatic animal carriers, as well as recent characterizations of relevant taxonomic and pathogenic characteristics of these organisms. We further urge epidemiological research to evaluate the incidence of human diseases arising from poultry eating, based on preliminary non-publisher findings implying a prevalence of salmonellosis and campylobacteriosis in Mexican poultry farms comparable to other nations.

**Keywords:** *Salmonella*, *Campylobacter*, poultry, epidemiology, zoonosis

## 1. Introduction

Animal protein is the source of a significant number of zoonosis in humans. Salmonellosis and campylobacteriosis are currently important zoonoses in industrialized countries [1].

Salmonellosis is predicted to cause about 18,000 sicknesses and 500 diseases in the USA annually [2]. In Denmark, the yearly cost of infection in humans is estimated to be USD 15.5 million. Denmark spent USD 14.1 million in a program to eradicate SE, which is considered low to USD 25.5 million estimated for losses

caused by non-work and medical treatment [3]. Unfortunately, information related to the cost of foodborne illness is generally not well reported or published in developing countries [3].

In some countries, salmonellosis problems have increased 20-fold between the eighties and nineties of the last century [4].

A retrospective analysis of salmonellosis cases carried out in Norway between 1966 and 1996 suggests an epidemiological relationship between birds and humans [5]. In the United Kingdom, salmonellosis and campylobacteriosis have been found to increase between June and August, which is attributed to the lack of timely refrigeration of food being stored in refrigeration just as the ambient temperature rises and also due to the habit of consuming barbecue since the meat is not adequately cooked during the summer [2].

Over the past decade, the number of salmonellosis cases recorded in Sweden has doubled due to more infections of SE, with four *salmonella enterica* serotypes were responsible for 60% of the cases detected in that country in 2001: ST (22.1%), SE (17.7%), S. Newport (10%), and S. Heidelberg (5.9%) [6].

In 2005, three serotypes of *Salmonella* were responsible for more than 70% of human cases in France: SE (33%), ST (32%), and S. Hadar (6%) [7].

Among the *Salmonella* serotypes that were most isolated in Mexico between 1972 and 1999 were SE, ST, S. Derby, S. Agona, and S. Anatum, in decreasing order [8].

The clinical form of SE infection usually manifests as an episode of self-limiting enterocolitis, with symptoms that resolve within five days. It takes 8–72 hours for the infection to manifest itself, with clinical signs of diarrhea and intestinal pain. Antibiotics are not usually required in most cases of recovery. Although rare, severe diarrhea can occur, and a person may become ill to the point where they require hospitalization. Age (Children and elderly) and immunocompromised individuals are more susceptible than the general population. The infection in these patients can move from the intestines into the bloodstream and then to other organs, potentially leading to death unless the patient receives quick treatment with antibiotics [9].

*Salmonella Enteritidis* is a bacterium that causes intestinal infection in various animal species, particularly birds, without showing any symptoms. A strain of SE enters the ovaries of otherwise healthy hens, infecting the eggs before the shell is formed and contaminating the eggs, causing high mortality rates in neonate chickens [7].

*Campylobacter jejuni* is surpassing infections caused by *Salmonella* spp. and *Shigella* spp. in developed countries [10]. Most of the time, the origins of this infection are associated with animal feed, specifically poultry products [11]. In the United Kingdom and Denmark, outbreaks of campylobacteriosis are related to the consumption of undercook poultry products [12, 13]. Campylobacteriosis in animals destined for slaughter is rare in Mexico, and the disease's influence on human health is unknown.

## 2. Characterization of salmonellosis

*Salmonella* belongs to the Enterobacteriaceae family. They are Gram-negative bacilli that do not form spores. In this genus, there are three types of antigens: somatic O, flagellar H, and capsular Vi, which are used to distinguish more than 2500 serotypes based on their agglutination properties, which are used to determine more than 2500 serotypes. New serotypes are added to the Kauffmann-White list every year, which is updated with the latest information [14].

*Salmonella* is a genus that contains only two species: *salmonella bongori* and *S. enterica*, which is subdivided into six subspecies: *entericae*, *salamae*, *arizonae*, *diarizonae*, *houtenae*, and *indica*. Salmonellosis in humans and higher animals is caused

by serotypes of the subspecies *entericae*, which account for nearly 99% of all cases [15]. A serotype of *S. enterica*, subspecies *entericae*, serotype *enteritidis*, abbreviated SE is used for practical diagnostic and epidemiological purposes [16].

Serotypes can be further separated by developing biotypes and phagotypes, which can then be used for more detailed studies of taxonomy and pathogenesis. The biotype denotes the biochemical variance between organisms of the same serotype, whereas the phenotype states the varied vulnerability of organisms of the same serotype to bacteriophage lysis [16].

*Salmonella enterica* serotypes Typhi and Paratyphi produce severe infections in humans known as a septicemic syndrome and typhoid fever, respectively, but are not pathogenic to animals. *S. Gallinarum* and *S. Abortus-ovis* cause avian typhoid and abortions in sheep, respectively, but rarely cause mild or asymptomatic infections in people. However, there are serotypes of *S. Choleraesuis* that produce severe disease in its usual carrier, the pig, but can also be pathogenic in humans. SE and ST infect both people and animals, however, in the latter, primarily hens, they cause asymptomatic illnesses [15, 17]. Undercook poultry products and the increased antibiotic resistance are linked to the increased number of infections in humans [7].

The mechanisms causing the rise in SE infections in birds have not been fully identified, making it challenging to identify illness in otherwise healthy chickens. Infections with SE in many animals, particularly chickens, with no apparent clinical indications and no acute outbreaks with mortality have been identified [16]. However, these healthy carriers can spread infection by fecal contamination of meat and egg. It is challenging to detect SE when the number of bacteria present is less than 9% [18].

The mechanization of poultry production and the export of parent birds have both contributed to the global spread of SE. For example, in the USA, molting of laying hens is a common practice that reduces or eliminates feeding of the birds' weight loss in birds; this practice speeds up molting but renders chickens more susceptible to SE infection, and once infected, they excrete the microorganism in feces in significantly high concentrations, which in turn increases the risk of egg contamination [19]. In poultry, vertical transmission to the progeny is common [20]. According to research in the Netherlands, flocks of laying hens, are primarily infected through direct contact with contaminated farm environments [21]; however, the epidemic that occurred in the United Kingdom in the early 1980s is attributed to the introduction of lines of progenitor birds infected with the phage type 4 [22].

*S. Enteritidis* can be introduced into flocks by rodents, which are highly vulnerable to infection, to the point that purposeful infection was employed to eliminate mice [23, 24]. *S. Enteritidis*, which was employed as a pesticide in the United Kingdom in 1940, was a type 6 phage [24, 25]. However, it has been demonstrated that acquiring the IncX plasmid changes phage 4 to phage 6 strain [26].

The dramatic increase in infection with *Salmonella Enteritidis*, a type 4 phage in humans in Europe since 1980, suggests that the bacterium has recently acquired new virulence genes [27].

More microbial genomes have been sequenced and compared recently, allowing the frequency of mutations to be approximated. The recombination mechanisms implicit in the replication process through the acquisition or loss of gene-carrying areas are a significant source of evolution. Plasmids, genomic islands, bacteriophages, transposons, and insertion sequences are other mechanisms of transferring or acquiring virulence genes [27]. These mobile components provide advantages to microbes in adapting to infecting specific cells [28]. Pathogenicity islands, or genes associated with virulence, arise outside of bacteria as mobile elements. Acquired pathogenicity islands contribute to the aggressive nature of bacteria by containing clusters of genes that boost virulence and can change a benign organism into a pathogenic one. Twelve islands of pathogenicity for *Salmonella* spp. have been

described, some of which are shared by all serotypes of the species, while others are exclusive to individual serotypes [7].

*Salmonella Gallinarum* can induce flock immunity against serotype 09, indicating cross-immunity with *Salmonella Enteritidis*. As a result of this immunological feature, it has been proposed that to the extent that *Salmonella Gallinarum* has been removed by vaccination and slaughter of afflicted birds, its elimination may have allowed *Salmonella Enteritidis* to establish itself [7, 29]. Conversely, in Great Britain, the 50% decrease in *Salmonella Enteritidis* infection in birds since 1997 corresponds to the introduction of new live vaccines against serotype 09, in place of vaccines with bacteria killed in formalin [22]. For practical purposes, in terms of controlling transmissible zoonoses, vaccination of birds against *Salmonella Enteritidis* of serotype 09 may be indicated even in situations where *Salmonella Gallinarum* has been eradicated.

Integrations, which typically carry one or more antibiotic resistance genes, are another key source of microbial variety [30]. The growth and spread of antibiotic-resistant bacteria is an unavoidable side consequence of antibiotic treatment. Some *Salmonella* strains are resistant to most common antibiotics, including fluoroquinolones, with the latter resistance being related to point mutations in the *gyrA* gene [31].

Considering that integrations may spread antibiotic-resistant genes in addition to transposons, genomic islands, and plasmids, treatment with antimicrobial agents may contribute to the increase in the population of bacteria resistant to related antimicrobial agents, and therefore the use of antimicrobials in animal feed may have adverse effects on human health, for its selection effect on the resistant bacterial population [7].

### 3. Characterization of campylobacteriosis

The classification of the genus *Campylobacter* has been revised, and 16 species are now acknowledged [32]. These bacteria have spiral or curved rod shapes, are Gram-negative, have flagella that let them move, and are microaerophilic. The three species of medical and veterinary significance are as follows: *C. jejuni*, *Campylobacter coli*, and *C. lari*. *C. jejuni* is divided into two subspecies: *C. jejuni jejuni*, referred to simply as *C. jejuni*, which is associated with disease to humans, and *Campylobacter jejuni doylei*, which only sporadically affects humans [33].

*Campylobacter* spp. are oxidase-positive, reduce nitrates, are methyl red and Voges-Proskauer negative, and do not hydrolyze gelatin. Except for some strains of *Campylobacter lari*, most species are urea negative. Microorganisms that have been exposed to water for an extended period take the form of coconuts, which are more challenging to develop and may not even be cultivable. *C. jejuni*, *C. coli*, and *C. lari* are thermophilic, meaning they grow best at 42°C and 43°C and do not grow at temperatures lower than 25°C [34, 35]. Culture in selective media takes two days, and confirmatory testing on the species takes two more days [36].

Human infection is restricted to the gastrointestinal system and results in various forms of diarrhea. Infection can induce neurological abnormalities in rare cases [37]. The majority of illnesses are caused by the consumption of chicken and pork. In addition, *C. jejuni*, *C. coli*, and *C. lari* cause gastroenteritis in humans. Nevertheless, *C. lari* derived from pigs accounts for just 3% of the isolates [10].

### 4. Relevant aspects of pathogenicity mechanisms

*Salmonella Enteritidis* causes infection by attaching the intestinal mucosa and then invading the enterocytes. *Salmonella Enteritidis* adheres to the surface of

enterocytes via the fimbriae and flagella. *Salmonella Enteritidis*' primary fimbriae are SEF14, SEF17, and SEF21. *Salmonella Enteritidis* colonization in birds occurs primarily in the cecum. Glycosphingolipid (GSL) GlcCer (N-1) and ganglioside GM3 (G-1) from chicken intestinal mucosa, found in ileum and cecum, have been studied as *Salmonella Enteritidis* SEF21 fimbria receptors [38].

When *Salmonella Enteritidis* crosses the epithelium and reaches the intestinal lamina propria, it invades the macrophages, and as it is generally resistant to the action of these, these cells serve as a vehicle to invade other organs [39].

*C. jejuni* and *C. coli* are commensals that reside in the intestines of numerous animals, including poultry, and survive in temperatures as low as 4°C for several weeks [40].

*Campylobacter jejuni*, produces enterotoxins and cytotoxins, which are the main cause of digestive symptoms in humans [33].

Until recently, the primary source of *Campylobacter* transmission in birds was thought to be horizontal from garbage, water, insects, equipment, and wildlife. Given the failures in attempts to grow *Campylobacter* from incubators or newborn chicks [41, 42]. However, considering that *C. jejuni* has been isolated in the reproductive organs of hens and the sperm of parent roosters, vertical transmission through the egg must be considered as a probable route of infection [43].

## 5. The problem of diagnosis and detection

The main problem is that *Salmonella* and *Campylobacter* are inhabitants of the intestine of birds, and in that environment, many bacterial organisms grow. Therefore, when trying to isolate a pathogenic species, it may not be possible to detect if the number is proportionally deficient and hidden by other organisms' growth. For this reason, the use of immunological and molecular biology techniques has been recommended to detect the existence of carrier animals in a short time. Isolation and identification of *Campylobacter* are problematic since it is slow-growing and easily confused with bacteria of the genus *Arcobacter*. The biggest drawback is that these are inert organisms that do not metabolize the sugars that are traditionally used to differentiate enterobacteria. That different environmental conditions, temperature, and antimicrobial sensitivity, as well as the hydrolysis of hippurate and indoxyl acetate, are used during isolation of *Campylobacter* [44].

*Salmonella* detection techniques based on polymerase chain reaction (PCR) have been developed for naturally and artificially contaminated food [45]. Several sensitive and precise PCR approaches for the detection of *Campylobacter spp.* are also available, which reduce diagnosis time to 48 hours and can detect up to one CFU (colony forming unit)/gram of sample [33, 46].

## 6. Foresight in Mexico

Unpublished results (isolation and identification) from a recent study in a commercial poultry company in Mexico revealed that from 30 broiler chickens, 8 chickens were positive for SE, and 2 were positive for ST. Without intending to conclude a single farm's sampling, given the homogeneous conditions under which modern poultry farming is carried out, these preliminary findings suggest that the prevalence of *Salmonella* in poultry farms in Mexico may be comparable to that of other countries with technical poultry farming. In the same study, 9/30 chickens were *C. jejuni* positives, and 2/30 were *C. coli* positives. According to these preliminary findings, *C. jejuni* and *C. coli* are likely to live as commensals in the intestinal

tracts of broiler chicken in commercial flocks in Mexico, implying that studies involving representative segments of national poultry farming will be helpful soon. Campylobacteriosis in people must be expected in Mexico; hence, epidemiological research is a top priority. Given its sensitivity and specificity, the PCR approach is recommended to detect salmonellosis and campylobacteriosis in animals, food, and the environment to strengthen the foundations for the development of relevant epidemiological markers in Mexico.

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