

Poultry meat as a source of foodborne *Campylobacter* and *Salmonella* infections in humans

Abstract

Foodborne infections caused by *Salmonella* and *Campylobacter* are very common worldwide, and these organisms are regarded as the most frequent cause of bacterial gastroenteritis. Consumption of improperly cooked poultry products or cross-contaminated foods is regarded as the most important risk factor for acquiring these diseases. In typical cases abdominal cramps, diarrhoea and fever are the symptoms observed. The infections are usually self-limiting and antibiotic therapy is rarely indicated. In small proportions of infected individuals, serious consequences like Guillian-Barre Syndrome (GBS) and reactive arthritis are seen.

Control of these bacteria in the poultry production is the only option to prevent infection in humans. Production of chicks free of *Salmonella* and *Campylobacter*, adoption of strict biosecurity measures are some alternatives. As level of subsequent carcass contamination is largely determined by the processing conditions in the abattoirs, strict hygienic processing operations should be followed. Besides, proper cooking and maintenance of hygienic conditions in the kitchen are the most important factors to eliminate the organism in the food.

TABLE OF CONTENTS

1	INTRODUCTION	2
2	THE ORGANISMS	3
2.1	GENERAL BACTERIOLOGY	3
2.1.1	<i>Campylobacter</i>	3
2.1.2	<i>Salmonella</i>	3
2.2	ISOLATION, CULTIVATION AND IDENTIFICATION	4
2.2.1	<i>Campylobacter</i>	4
2.2.2	<i>Salmonella</i>	6
3	POULTRY: THE INFECTION SOURCE	8
3.1	CAMPYLOBACTER IN POULTRY PRODUCTION	8
3.2	SALMONELLA IN POULTRY PRODUCTION	9
3.3	MODE OF CARCASS CONTAMINATION	11
4	THE DISEASE EPIDEMIOLOGY	12
4.1	CAMPYLOBACTERIOSIS	12
4.1.1	<i>Types causing the disease</i>	13
4.1.2	<i>Transmission/ Risk factors</i>	13
4.1.3	<i>Symptoms</i>	14
4.1.4	<i>Incidence and outbreaks</i>	14
4.1.5	<i>Pathogenesis and Sequelae</i>	18
4.1.6	<i>Treatment</i>	19
4.2	SALMONELLOSIS	19
4.2.1	<i>Types causing the disease</i>	20
4.2.2	<i>Transmission/Risk Factors</i>	21
4.2.3	<i>Symptoms</i>	21
4.2.4	<i>Incidence and outbreaks</i>	21
4.2.5	<i>Pathogenesis and consequences</i>	23
4.2.6	<i>Treatment</i>	24
5	CONTROL	24
5.1	ON FARM CONTROL	24
5.2	PROCESSING CONTROLS	26
5.3	FOOD HANDLING	27
6	CONCLUSIONS	28
	REFERENCES	30

1 Introduction

Food borne infections caused by the species of *Salmonella* and *Campylobacter* are the most common zoonoses occurring in the industrialized world today (Jorgensen et al., 2002). As a whole, the incidence of these food borne infections has increased worldwide in recent years (WHO, 2002a; Zweifel et al., 2004). Specifically, the incidence of *Salmonella* and *Campylobacter* infections has increased several folds in many countries (Ethelberg et al., 2004). In most of the developed countries, the incidence of *Campylobacter* infection in humans is higher than the incidence of *Salmonella* infection (WHO, 2002b). *Campylobacter*s are also generally regarded as the most common bacterial cause of enteritis worldwide (CDC, 2004a). Since these infections in developing countries are less well documented, the magnitude of the problem is not precisely known. However, high prevalence of diarrhoeal diseases in many developing countries suggests major underlying food safety problems (WHO, 2002b).

Among the foods of animal origin, poultry meat and eggs are the most common sources of *Campylobacter* and *Salmonella* infections in humans (Friedman et al., 2000; Jacobs-Reitsma, 2000; Baumler and Hargis, 2000; Heres et al., 2004). Poultry meat is the implicated source for 50 to 70% of human campylobacteriosis (Stern, 1992) and 67% of *Salmonella* outbreaks are attributable to it (Panisello et al., 2000). However, *Campylobacter* are found to be contaminating chicken carcass more often than *Salmonella* (Geilhausen et al., 1996).

Despite of various stringent control efforts, substantial reduction in the foodborne outbreaks or sporadic cases due to these organisms has not been successful. To reduce the cases of human infections, detail information of the recent outbreaks in terms of epidemiology, strains/serotypes of the organisms involved, and sources of infection is important. This essay reviews the significance of poultry meat as a source of food borne salmonellosis and campylobacteriosis. General bacteriology, outline of isolation and identification techniques, infection and colonization in birds and consequent carcass contamination during slaughter and processing, and the disease produced in humans following their consumption is discussed in detail in the light of latest outbreak information available. Control of the organisms beginning from rearing of the birds,

slaughtering, handling of carcass and processing, and prevention of infection during preparation/cooking and consumption is also discussed.

2 The organisms

2.1 General Bacteriology

2.1.1 *Campylobacter*

Campylobacters are Gram negative, non-spore forming, motile, bacilli or rods, curved or s-shaped or gull-wing shaped or spiral rods measuring 0.2 to 0.5 µm wide and 0.5 to 8 µm long. *C. jejuni* is a fastidious organism that requires complex medium and environment for growth. Campylobacters do not grow in air but require microaerobic atmosphere containing 5% O₂ 10% CO₂ and 85% N₂ for optimum recovery and growth (Doyle and Jones, 1992; Nachamkin, 1995). Being thermophilic, the optimum temperature for culturing *C. jejuni* is 42°C (Quinn and Markey, 2003). The organism is sensitive to acidic environment and does not grow at pH lower than 5 (Goossens and Butzler, 1992). It has been known that *C. jejuni* does not survive well on foods due to sensitivity to drying, temperature, ambient oxygen concentration and heat, however refrigeration conditions support survival and long-term survival is possible on frozen foods (Hunt and Abeyta, 2001; Donnison, 2003). Older and stressed organisms gradually become coccoidal (Nachamkin, 1995) and even more difficult to culture. They are described as “viable but non-culturable” (Corry et al., 1995; Takkinen and Ammon, 2003). Oxygen quenching agents in media such as hemin and charcoal, as well as a microaerobic atmosphere and pre-enrichment and enrichment can significantly improve recovery (Agulla et al., 1987; Ransom and Rose, 1998; Donnison, 2003).

2.1.2 *Salmonella*

Salmonella are gram-negative bacteria belonging to the family *Enterobacteriaceae*. They are nonsporeforming rods, up to 3 µm in length. Most *Salmonella* are motile via means of peritrichous flagella; however, *Salmonella* serotypes Gallinarum and Pullorum are notable exceptions. *Salmonella* are ubiquitous in environment and animals and birds are significant reservoirs.

Most *Salmonella* are mesophilic heterotrophs, needing only simple inorganic salts containing nitrogen, phosphorous, sulfur as source of divalent cations, and an organic substrate for carbon and energy to sustain life. While salmonellae grow best at moderate temperatures (35-37°C), they can grow over a much wider temperature range, with growth occurring at temperatures as low as 4°C and as high as 48°C. Growth is also best at moderate pH (6.5-7.5) in conditions of high water activity and low osmolarity (Anderson and Ziprin, 2001).

2.2 Isolation, cultivation and identification

2.2.1 Campylobacter

As there is no generally accepted standard method of isolating and identifying campylobacters, most workers use methods particular to their laboratories. Figure 1 outlines the sample preparation method for isolation of campylobacters from poultry meat samples, and figure 2 shows the flow diagram of procedures for isolation, identification and storage of thermophilic campylobacters. Table 1 shows the typical biochemical reactions of campylobacters.

Table 1. Typical biochemical reactions of commonly isolated species of thermophilic campylobacters (from Nachamkin, 1995; Donnison, 2003).

Characteristics	<i>C. jejuni</i>	<i>C. coli</i>	<i>C. lari</i>	<i>C. upsaliensis</i>
H ₂ S	-	Slightly +ve	-	-
Nalidixic acid	S	S	R	R
Cephalothin	R	R	R	S
Catalase	+	+	+	- ve or slight
Hippurate hydrolysis	+	-	-	-
Nitrate reduction	+	+	+	+
Indoxyl acetate hydrolysis	+	+	-	+
Oxidase	+	+	+	+

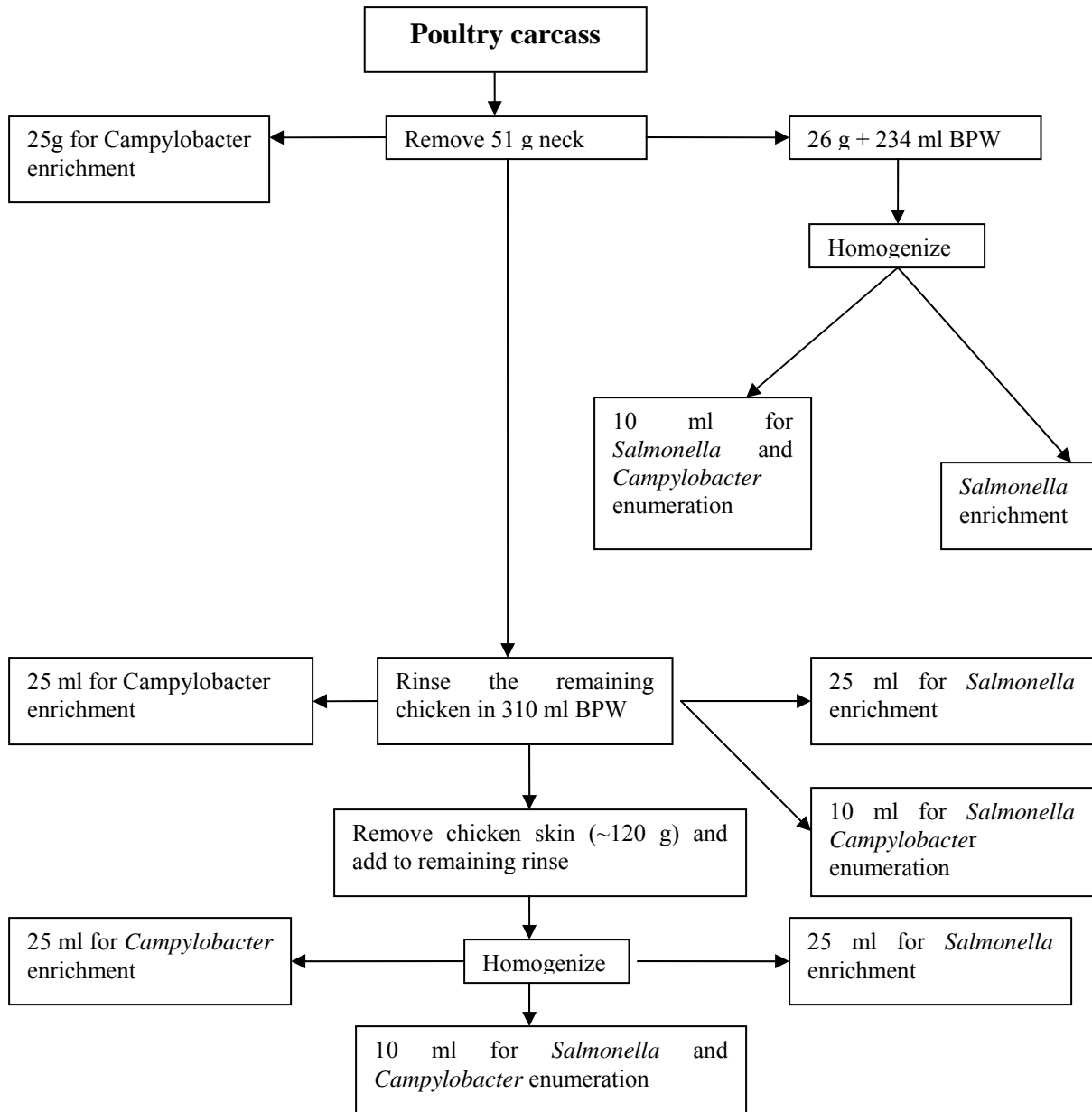


Figure 1. Sample preparation methods for examining raw retail chicken for *Salmonella* and *Campylobacter* spp. (from Jorgensen et al., 2002).

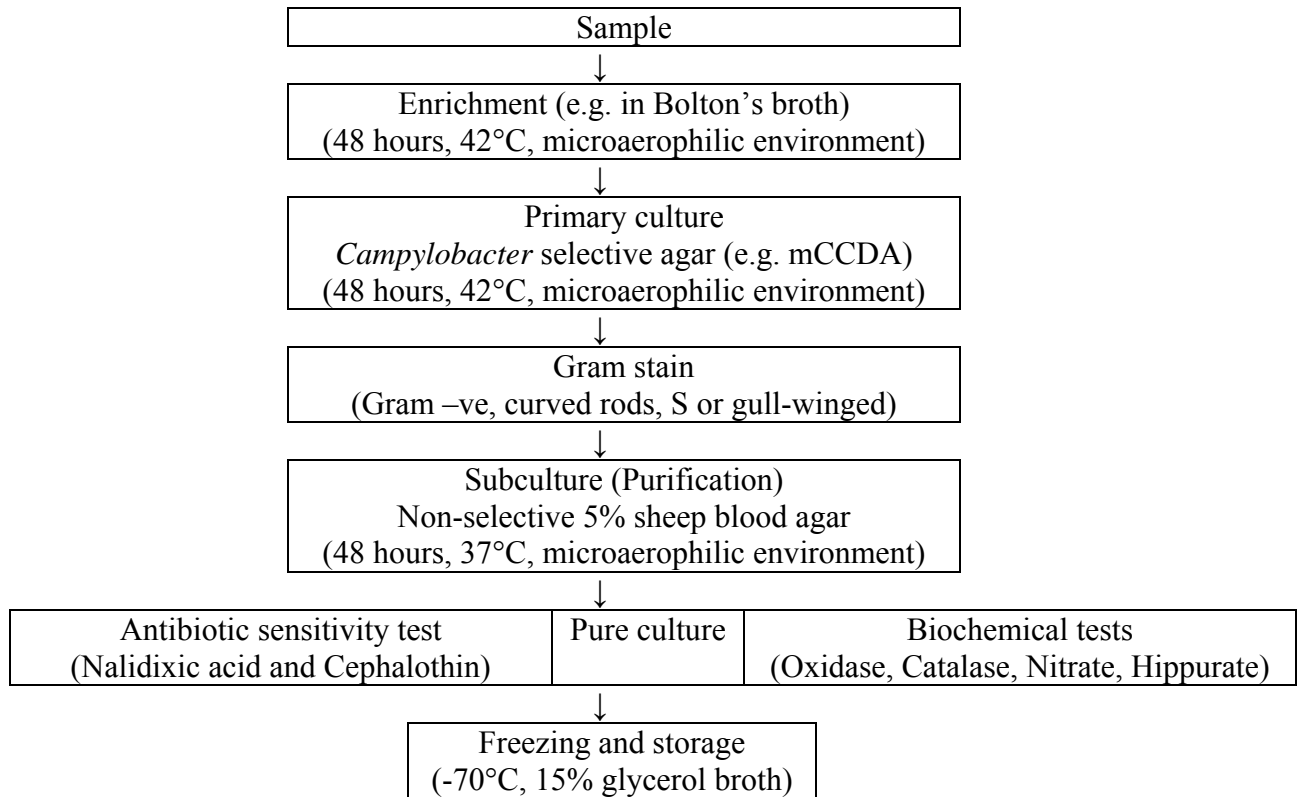


Figure 2. Flow diagram of procedures for isolation, identification and storage of thermophilic campylobacters (from Adhikari, 2003).

2.2.2 *Salmonella*

Although, techniques including nucleic acid and immunologically based methods have been developed to detect and/or isolate *Salmonella* from various specimens, bacteriological cultivation remains the most used method. Serotyping, using slide agglutination tests with antisera are used to detect somatic (O) and flagellar (H) antigens. Flow diagram of sample preparation for poultry meat was shown in figure 1 and that of isolation and identification in figure 3.

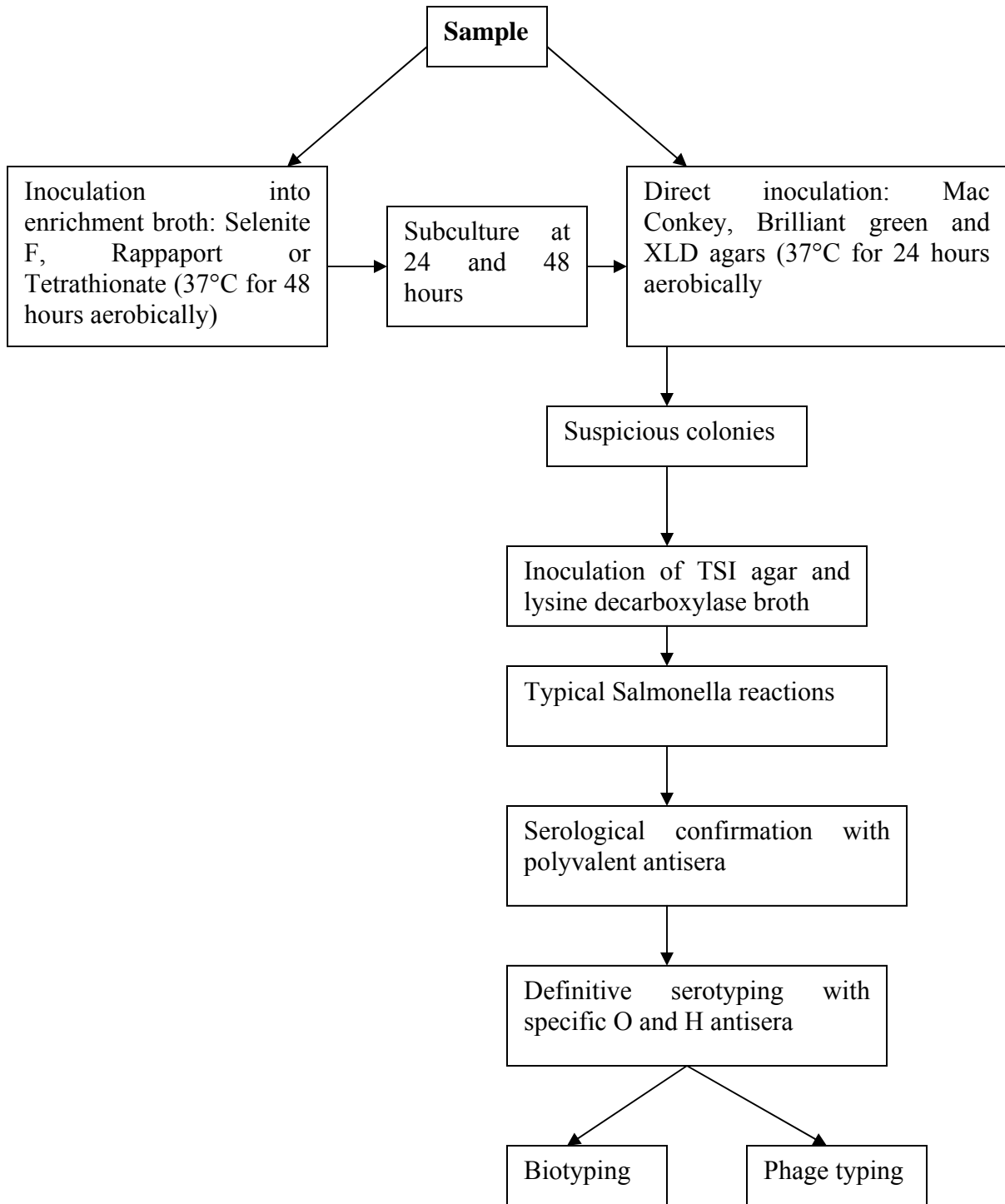


Figure 3. Flow diagram of procedures for the isolation and identification of *Salmonella* serotypes (from Quinn et al., 2002).

Biochemical characteristics: Salmonellae are oxidase-negative, facultative anaerobes. They do not produce indole, do not hydrolyze urea, and do not deaminate phenylalanine or tryptophan. Most reduce nitrate to nitrite, and most ferment a variety of carbohydrates with concomitant production of acid. D-Glucose is fermented via the mixed acid pathway, thus yielding a positive methyl red reaction; acetyl methyl carbinol is not produced by *Salmonella*, and they therefore yield a negative Voges-Proskauer reaction (Quinn and Markey, 2003).

3 Poultry: the infection source

Poultry meat is one of the most important sources of *Campylobacter* and *Salmonella* infection to humans. Besides, eggs are also significant source of nontyphoidal salmonellosis. *Campylobacter* are found to be contaminating chicken carcass more frequently than *Salmonella* (e.g. 28% vs. 14%; Geilhausen et al., 1996), but eggs are not usually contaminated with campylobacters (Jacobs-Reitsma, 2000).

3.1 Campylobacter in poultry production

Species detected: Poultry birds are found to be colonized primarily with *C. jejuni*, less often with *C. coli* and rarely with other species (Newell and Wagenaar, 2000; Zorman and Mozina, 2002). *C. lari* is also detected from the intestine of birds (Quinn et al., 2002). *C. upsaliensis* has been found in ducks (Ridsdale et al., cited by Corry and Atabay, 2001).

Colonization: *Campylobacter* has been found as normal commensal flora of intestinal tract in 30-100% of poultry (O'Sullivan et al., 2000). Occurrence of vertical transmission is controversial (Newell and Wagenaar, 2000; Corry and Atabay, 2001). Colonization of the organisms does not usually occur in birds younger than three weeks of age. However, at the time of slaughter, broiler flocks are commonly campylobacter-infected (Hald et al., 2001), and the colonization levels in the small intestines ranges from 10^5 to $>10^9$ CFU per gram (Berndtson et al., cited by Jacobs-Reitsma, 2000).

The infection occurs usually around 3-4 weeks of age since at this period the birds are exposed to various stressors, like change in feed and coccidiostats, and multiple vaccinations, and there will be no longer protection of maternal antibody (Humphrey,

2004). Horizontal transmission, directly from carrier flocks, or indirectly via contamination of poultry houses and transmission to the subsequent flock is a common route. The use of contaminated transport crates and catchers during depopulation is also reported to be a significant potential source of introduction of *Campylobacter* and *Salmonella* to the remainder of the flock (Slader et al., 2002). When birds are caught in batches for slaughter, intensive traffic to and from the rearing houses is believed to expose the premises to *Campylobacter* spp. and increase the rate of infection in birds (Hald et al., 2001). The spread of the organism is very rapid, especially in broiler flocks once *C. jejuni* is present, within 4 days all the birds can be carrying the bacteria (Humphrey, 2004).

Level of carcass contamination: There are several studies on level of *Campylobacter* contamination of chicken carcass, and the findings are largely inconsistent. The prevalence has been found to vary between 0 to 100%. These variations are believed to be due to difference on the type of samples studied (whole, portions, fresh, frozen, organic, free range etc), point of sampling in the processing chain, country, use of serological assays, carcass sampling and microbiological methods used in testing (Wilson, 2002). So, direct comparisons between different reports are rarely meaningful.

Stern (1992) reported the carcass contamination rate of chickens with *Campylobacter* to be 48-98%. A six year (1995-2000) survey of contamination rate of raw retail chicken with *Campylobacter* in the United Kingdom revealed the rate to be 47-81% (Wilson, 2002). The usual rate of contamination in New Zealand is 55-60% (Jutta, in study guide). In contrast to these findings, a recent New Zealand study reported a very low contamination rate of 27.5% (Baker et al., 2002).

Carcass contamination rate is also found to vary with season. In Danish poultry flocks, a higher contamination rate (50-80%) was seen during the summer (June to October), whereas a lower rate (13-40%) during the winter season (December to March) (Takkinen and Ammon, 2003).

3.2 *Salmonella* in poultry production

Species/serotypes detected: Table 2 lists the five most *Salmonella* serotypes occurring in animals (including poultry) worldwide. A large numbers of *Salmonella* serotypes

(species *Salmonella enterica* subspecies *enterica*) are detected in chicken carcasses. A recent Portuguese study recovered 10 different serotypes of *Salmonella* from 36 refrigerated poultry carcass. *Salmonella* Enteritidis was the predominant serotype accounting for 44% of the isolates, S. Hadar was found in 28% samples, S. Virchow in 8%. While one isolate each of S. Derby, S. Anatum, S. Heidelberg, S. Kingston, S. Saintpaul, S. Indiana and S. Blocky were detected in those samples (Antunes et al., 2003). In contrast, in one UK study (Wilson, 2002), the most dominant serotype contaminating the chicken carcass was found to be S. Bredeney (20%) followed by S. Enteritidis (18%). S. Enteritidis phage type 4 (PT4) is most commonly reported from poultry isolates (Poppe, 2001).

Table 2. The top five ranking serotypes of *Salmonella* isolated from animals worldwide (GSS, 2004).

Region	Year	Top five serotypes				
		1	2	3	4	5
Americas	2003	Enteritidis	Gallinarum	Kentucky	Typhimurium	Mbandaka
Europe	2003	Enteritidis	Typhimurium	Meleagridis	Dublin	Blockley
Oceania		Data not available				
Asia	2002	Enteritidis	Stanley	Typhimurium	Schwarzengrund	Virchow
Africa	2002	Hadar	Brancaster	Bredeney	Albany	Poona

The serotypes *Salmonella* Gallinarum-Pullorum, which produce Pullorum disease and Fowl typhoid in poultry, do not have human public health significance (Nisbet and Ziprin, 2001). And the serotypes, S. Typhimurium and S. Enteritidis, which produces illness in humans, usually remain subclinical in layer birds (Quinn et al., 2002). However, S. Enteritidis PT4 is reported to be highly virulent and causes significant mortality in young broilers (Humphrey, 1999; Wilson, 2002).

Colonization: Colonization of salmonellae in animals is believed to be favored by the intensive animal production systems (Antunes et al., 2003). Unlike *Campylobacter*, colonization of *Salmonella* occurs in young birds (Humphrey, 2004). The common route of infection for offspring of the commercial flocks from parentstocks is vertical, via transovarian route (Poppe, 1999). Other routes may be environmental, e.g. via contaminated feed, water, utensils, fomites, droppings of rodents or wild birds etc. Horizontal transmission may occur once few chicks are infected, right from various operations in the hatchery, during shipment or rearing. Older birds are usually

asymptomatic excretors that often carry their salmonellae to slaughter and are the source of widespread contamination of the processing plant environment and carcasses that enter the marketing chain (Williams, 1980). In laying hens, ovaries are the important sites of colonization, which facilitates egg contamination. It has been found that *Salmonella* can penetrate and enter the egg through an intact shell as well as through minor cracks in the eggshell (Nisbet and Ziprin, 2001).

Level of contamination: As with campylobacters, *Salmonella* contamination of chicken carcass varies widely depending on the nature of the survey and methods used. It has been seen that in a batch of slaughtered birds, proportion of *Salmonella* contaminated carcass may vary from 0 to 100% (Mead, 2000). In the United Kingdom, contamination of chicken carcass by *Salmonella* was found to be 11% on an average, during the period of 1995 to 2000 (Wilson, 2002). Nisbet and Ziprin (2001) have mentioned this rate to be more than 21% in the United States. But since 2000, this contamination rate has decreased by 66% in the United States (Anon, 2004). In Portugal, 60% of the chicken carcasses were found to be contaminated with *Salmonella* (Antunes et al., 2003). However, the contamination rate of eggs is found to be very low; e.g. 1 in 10 000 in the United States and 1 in 15 000 in the United Kingdom (Radkowski, 2001).

3.3 Mode of carcass contamination

Salmonella and *Campylobacter* colonizing in the gut, skin or feathers of the birds spread to carcass, mainly during the slaughtering process on the processing plants. Besides, environment of the processing plant may also serve as a source of contamination. In case of salmonellae, levels of intestinal carriage in the live bird are generally low at slaughter, which may be found to be 1-30 colony forming units (CFU) per carcass after slaughter and processing (Yang et al., 2001). However, in case of *C. jejuni*, both the proportion of contaminated carcasses and the numbers of viable cells present are consistently higher, so that finished carcasses may carry up to 10^6 CFU of this organism (Waldroup, cited in Mead, 2000).

Cross contamination at slaughterhouses: Contamination of carcass may not occur only from its own gut contents, but also cross-contamination occurs from the processing environment (contaminated equipment, working surfaces, process water, and air). This is

obvious from increased diversity of strains isolated from the processed carcass as compared to those in faecal samples (Allerberger et al., 2003). Scalding water is a significant site contributing to the cross-contamination of the carcasses, since the scald water remains heavily contaminated with faecal materials (Humphrey, 1999). Besides, leakage of gut contents during defeathering and evisceration and related processes leads to significant carcass contamination (Allerberger et al., 2003).

The skin and other carcass surfaces contain most of the microbes which contaminate the underlying tissues during subsequent portioning, skinning and boning. This may be the reason why whole chicken carcasses are less frequently contaminated than portions. Some studies have, however, indicated that slaughter processes have a minor influence on the risk for human campylobacteriosis (Takkinen and Ammon, 2003).

Salmonella: The septicaemic form of infection of PT4 in broiler chickens may facilitate localization of the organism in muscles (Humphrey, 1999). As *Salmonella* bacteria are present on the skin and feathers besides faeces (Nisbet and Ziprin, 2001), they are also the obvious sources for carcass contamination.

Campylobacter: Intestines, caeca and crop are the organs of broilers that are heavily contaminated with campylobacters at the time of entering the processing plants (Altekruse and Tollefson, 2003). Besides, feathers and skins also carry large number of campylobacters in colonized birds (Jacobs-Reitsma, 2000). Campylobacters appear to survive the scalding (up to 80°C), and the resistance to temperature increases as they are attached to chicken skin (Humphrey, 2004). This may be due to change in the skin surface as a result of high scalding temperature, facilitating the firm attachment of bacteria (Jacobs-Reitsma, 2000).

4 The disease epidemiology

4.1 Campylobacteriosis

Although *C. jejuni* and *C. coli* can exist as commensal organisms of the domestic poultry and livestock, they are potent human pathogens. Due to poorly developed and evaluated subspecies typing scheme for campylobacters, it is unclear whether or not the epidemiologies of human *Campylobacter* infections are related (Moore et al., 2000).

4.1.1 Types causing the disease

Four species recognized as major causes of human campylobacteriosis are: *C. jejuni*, *C. coli*, *C. lari* and *C. upsaliensis* (Lastovica and Skirrow, 2000). The most commonly isolated species of campylobacters from human cases are *C. jejuni* (Baker et al., 2002; CDC, 2004a; Vierikko et al., 2004) and *C. coli* (Nachamkin, 1995), accounting for more than 99% of the human isolates, and *C. jejuni* alone is isolated at the rate of about 90% to 95% (Hunt and Abeyta, 2001; Tam et al., 2003; Takkinen and Ammon, 2003; Gupta et al., 2004). *C. coli* is the second most common cause of human campylobacteriosis (Tam et al., 2003). Poultry meat is an important source of both *C. jejuni* and *C. coli* (Zorman and Mozina, 2002). Human and chicken isolates are genotypically and phenotypically very diverse, and it is believed that human isolates are more virulent than chicken isolates (Takkinen and Ammon, 2003).

Serotyping of the organisms has been done in a very few studies. In one Danish study, a significant association was demonstrated between the infection with *Campylobacter jejuni* serotype O:6 and eating undercooked poultry (Takkinen and Ammon, 2003).

4.1.2 Transmission/ Risk factors

Although the modes of transmission are not well known (Vierikko et al., 2004), handling or consumption of undercooked or raw chicken has been regarded as the major risk factor for infection with *C. jejuni* (WHO, 2002b; Sopwith et al., 2003). Cross-contamination of other food items, especially those are eaten raw (e.g. salads) is also a significant risk factor. Commercial catering establishments like restaurants provide opportunities for outbreaks of foodborne disease because large quantities of different foods are handled in the same kitchen. This is the reason why eating chicken in the restaurants is associated with increased risk of infection (Altekruse and Tollefson, 2003).

Major risk factor for *C. coli* is, however, different than that of *C. jejuni*. It has been found to be more commonly associated with travel abroad than from poultry meat (Sopwith et al., 2003).

The morbidity and mortality of *Campylobacter* infection in HIV/AIDS patients is greater than in HIV-negative patients, and the people in developing countries, especially the infants are at high risk (Coker et al., 2002).

Campylobacter species are highly infective and the infective dose of *C. jejuni* varies from 500 to 10000 cells, depending on the strain, damage to cells from environmental stress, and susceptibility of the host (Tauxe, 1992; Hunt and Abeyta, 2001).

4.1.3 Symptoms

The clinical spectrum of *Campylobacter* enteritis ranges from a watery, nonbloody, noninflammatory diarrhoea to a severe inflammatory diarrhoea with abdominal pain and fever (Coker et al., 2002). Self-limiting acute enteritis is the most common syndrome. Prodromal symptoms are common and include headache, low fever, and myalgia lasting from a few hours to a few days. Symptoms of acute infection often begin with abdominal cramps followed by diarrhoea and high fever, peaking during the first day of illness (Altekruse and Tollefson, 2003). The incubation period for *Campylobacter* is normally two to four days, and is believed to be inversely proportional to infective dose (Gillespie, in press).

4.1.4 Incidence and outbreaks

The estimated annual incidence of *Campylobacter* human infections in the global population varies from country to country. Table 3 shows the incidence of *Campylobacter* cases in humans between countries. In most of the European countries, the reported cases of campylobacteriosis exceeds cases of *Salmonella* infection (Takkinen and Ammon, 2003) and in the United States, it is estimated to be nearly twice as high as *Salmonella* incidence (Nachamkin et al., 2000). In New Zealand, and many other countries around the world, campylobacteriosis is the most frequent notified disease (Baker et al., 2002). In the United States alone, 2.4 million persons are affected, and 124 persons are estimated to be dying of campylobacteriosis each year (CDC, 2004a). In this country, there has been a decline of approximately 27% in the incidence of campylobacteriosis between 1996 and 2001, yet *Campylobacter* spp. remain among the most common bacterial cause of foodborne infection (Altekruse and Tollefson, 2003). Because of the non existence of national surveillance programs for campylobacters in most of the developing countries, the details of outbreaks are rarely recorded. In these countries too, *Campylobacter* is one of the most frequently isolated bacteria from stool of

children with diarrhoea resulting from contaminated food and water (Oberhelman and Taylor, 2000).

Table 3. Comparison of campylobacteriosis incidence between countries (from Baker et al., 2002; Ethelberg et al., 2004).

Country	Period	Rate (per 100000)
New Zealand	2001	279.8
USA	2000	20.1
England & Wales	1998	111
Canada	1986-1998	39-54
Denmark	1999-2001	78-87
Australia	2000	107

Cases of *Campylobacter* infections are believed to be underreported. In spite of being the most common bacterial cause of gastroenteritis, only 4% of the food borne infection outbreaks is attributable to *Campylobacter* spp. (Panisello et al., 2000). While the majority of the human cases are sporadic in nature (Altekruse et al., 1999; Baker et al., 2002), which is believed to be up to 99% (Moore et al., 2000), outbreaks are occasionally detected. Because of the lack of simple, discriminatory, and cost-effective *Campylobacter* typing methods, outbreaks are difficult to identify (Ethelberg et al., 2004). Common source outbreaks occur, and most have been traced to unpasteurized milk (Altekruse and Tollefson, 2003) and contaminated drinking water (Tauxe, 1992; Merritt et al., 1999). Household outbreaks are common for *Campylobacter* infection (Ethelberg et al., 2004) but are usually recorded as sporadic cases. Regarding this, one interesting finding is mentioned in a Danish study. For the years 2000 and 2001, detail study of gastrointestinal disease outbreak database revealed 168 *Campylobacter* household outbreaks, in contrast to only eight outbreaks reported previously during this period (Ethelberg et al., 2004). In this connection, Slader et al. (2002) found that the actual number of *Campylobacter* infections in England is likely to be about eight times the published number.

Human cases show seasonality of infection with high infection rates during the summer and autumn (Sopwith et al., 2003; Takkinen and Ammon, 2003). Besides, a study done in England and Wales (Gillespie, in press) reported differences in the incidence of *C.*

jejuni infection throughout a week. It was found that disease incidence was greater on the days during or immediately following the weekend (figure 4). This was believed to be due to tendency of people eating out more at restaurants in their leisure time.

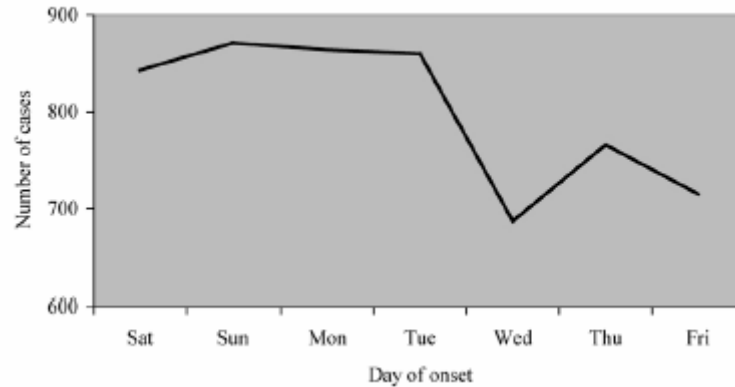


Figure 4. Weekly periodicity of indigenous *C. jejuni* infection in England and Wales (from Gillespie, in press).

However, the seasonality of human *Campylobacter* infections is not found to be associated with the rate of chicken carcass contamination with campylobacters (Wilson, 2002) as shown in figure 5. In the developing countries, however, *Campylobacter* enteritis has no seasonal changes in incidence (Coker et al., 2002).

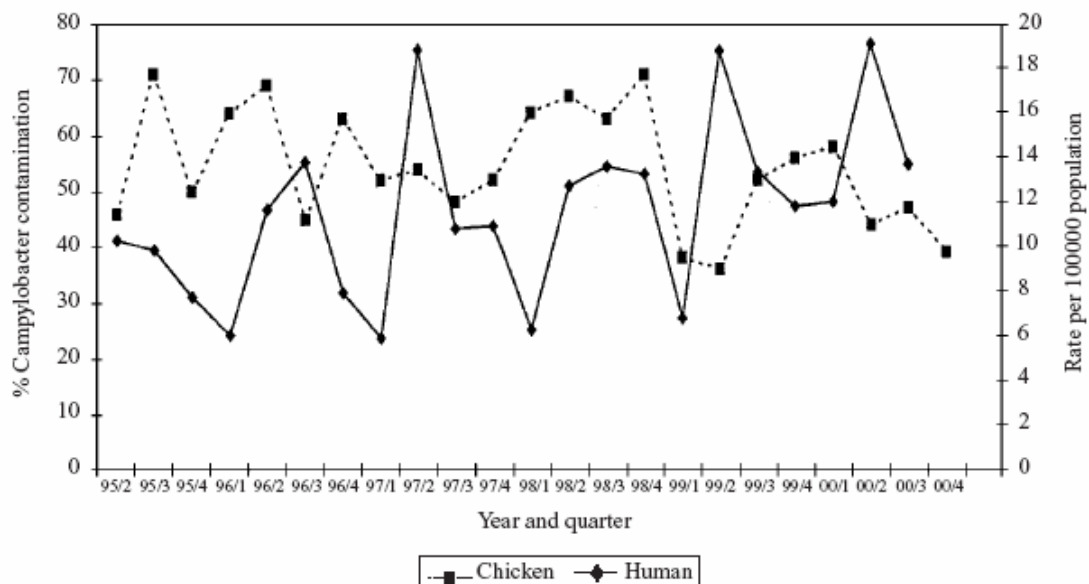


Figure 5. Relationship between *Campylobacter* contamination of chicken carcass and human infection during 1995 to 2000 in the UK (from Wilson, 2002).

An outbreak of *C. jejuni* infection among conference delegates was reported in South Australia during May 2001 (Raupach and Rebecca, 2003). Ten cases were identified among the cohort of 29 delegates. Other symptoms, apart from diarrhoea, included abdominal pain, fever/chills, headache, nausea and vomiting. Duration of illness ranged from one to seven days and one case was hospitalized for six days. Although a definitive source could not be determined, the most likely source was believed to be chicken dish as well as spring rolls and fried rice at a Chinese restaurant. Cross contamination of other dishes from chicken was suspected (Raupach and Rebecca, 2003).

A recent outbreak of *C. jejuni* in Europe, implicated to be associated with barbecued chicken, was documented by Allerberger et al. (2003). The outbreak involved five of six people, who had attended a barbeque party in Germany in March, 2001. The patients came down with diarrhoea 2-4 days after the party. Four of these five patients also suffered from severe headache and sore throat starting 24-48 h after the party. Three of the four patients submitting stool specimens had culture-confirmed *C. jejuni* infection. The farm of origin of chicken carcass was traced back and caecal swabs were obtained from 22 chickens, out of which 18 samples yielded *C. jejuni*. Also chicken carcasses were collected from incriminated processing plant which yielded *C. jejuni* in all six carcasses. The molecular subtyping of isolates indicated that the macrorestriction pattern of all the isolates from human cases and chicken carcass at the processing plant was same. However, only three isolates from caecal swabs showed the same pattern as human or chicken carcass isolates. It was concluded that the outbreak clone had been colonizing the processing plant and was cross contaminating the chicken carcasses there. One possible explanation for the diversity of strains in the caecal swab samples was late (7 weeks post detection of human infection) isolation being done, and changes in the fingerprinting pattern during this period (Allerberger et al., 2003).

Another outbreak was reported from the United States, which was believed to be acquired from eating lettuce cross-contaminated with raw chicken (Graves et al., 1998). During mid-August of 1996, following lunch in a restaurant, 14 people developed diarrhoea. Other observed symptoms were fever (in 93%), abdominal cramps (in 93%), nausea (in 79%), vomiting (in 36%) and visible blood in the stool (in 21%). Stool specimens

collected from 10 of these people, all showed *C. jejuni*. Cultural examination of food samples was not done because of unavailability. Inspection of the restaurant revealed that cutting up raw chicken for meals was done before preparing salads. It was believed that lettuce salad was contaminated with *C. jejuni* from raw chicken through unwashed or inadequately washed hands, cooking utensils or the counterpart (Graves et al., 1998).

In 2000, similar outbreak was reported in the Northern Ireland among individuals attending a lunch (Moore et al., 2000), where the infection source was implicated to be contaminated seasonal leaves and tomato salad. Out of 40 people participating in the lunch, twelve cases were identified, out of which 11 had eaten the salad mentioned above. Nine faecal samples were found to be culture positive for *Campylobacter* species. The symptoms observed were diarrhoea and abdominal cramps in 11 individuals, nausea in eight, fever in seven, and two experienced vomiting. Although identification of the contamination source was not done, chicken meat was believed to be the most likely source of contamination, after examination of the kitchen.

4.1.5 Pathogenesis and Sequelae

Although not yet fully understood, cell invasion and cytotoxic protein production has been regarded as major pathogenic mechanism of *Campylobacter* infection, that may play a role in clinical courses of the disease (Altekruse and Tollefson, 2003; Takkinen and Ammon, 2003). Chronic sequelae of infection with *Campylobacter* spp. are recognized worldwide and include Guillian-Barre syndrome (GBS), reactive arthritis, Miller-Fisher syndrome, toxic megacolon, and Haemolytic uraemic syndrome. Complications such as bacteremia, hepatitis, pancreatitis, and abortion have all been reported with various degrees of frequency (WHO, 2002b). Infection can be severe in immunocompromised or elderly patients but are rarely fatal.

GBS is an autoimmune disorder of the peripheral nervous system, which causes acute flaccid paralysis. The frequency of GBS resulting from campylobacteriosis has been estimated to occur in 1 in every 1,000 patients (Altekruse et al., 1999). After 1 year, 70% of the patients with GBS make complete neurologic recovery, 22% partially recover, 8% remain unable to walk, and 2% remain bedridden or require ventilation (Altekruse and Tollefson, 2003). Reactive arthritis or Reiter's syndrome, may also be a sterile sequela to

acute gastrointestinal campylobacteriosis. Onset of reactive arthritis occurs 7 to 10 days after onset of diarrhoeal illness (Altekruse and Tollefson, 2003). The frequency of this illness has been estimated as 1-7% of all cases of campylobacteriosis (Altekruse et al., 1999; Altekruse and Tollefson, 2003).

Besides producing illness in humans, campylobacters can also establish a temporary asymptomatic carrier state (Hunt and Abeyta, 2001).

4.1.6 Treatment

Campylobacter enteritis is a self-limiting disease, and in the majority of the cases, antibiotic therapy is not necessary (Coker et al., 2002). In severe cases and to eliminate the carrier state, however, macrolides (e.g. erythromycin), and fluoroquinolones (e.g. ciprofloxacin) are the drugs of choice (WHO, 2002a). However, in the recent years, the increasing rate of resistance to fluoroquinolone, ciprofloxacin has limited its clinical usefulness. The resistance to ciprofloxacin has reached about 19% of the human *Campylobacter* isolates in the United States in the year 2001 (Altekruse and Tollefson, 2003; Gupta et al., 2004) and 30% in many European countries during the last few years (Takkinen and Ammon, 2003). In contrast, the resistance to erythromycin has not increased significantly, e.g. the rate is stable at 2% of the human isolates since 1997 to 2001 in the United States (Gupta et al., 2004). The increase in resistance has been associated with antibiotic use in food animals as well as inappropriate treatments in humans (Takkinen and Ammon, 2003).

4.2 Salmonellosis

Although it is not possible to obtain an accurate estimate of proportion of human salmonellosis attributable to poultry, Nisbet and Ziprin (2001) believed it to vary from 7.5 to 37% depending on the methodology used and assumptions underlying the methodology. However, Panisello et al. (2000) reported 67% of *Salmonella* outbreaks are attributable to poultry.

4.2.1 Types causing the disease

Approximately 2000 serotypes of *Salmonella* cause disease in humans (CDC, 2004b). Several serotypes of the species *Salmonella enterica* subspecies *enterica* can be transmitted from poultry meat and produce salmonellosis in humans. Enteritidis and Typhimurium are the most common serotype associated with foodborne illness worldwide (GSS, 2004), eggs and poultry meat being the important source (Quinn et al., 2002). The five most common serotypes of *Salmonella* isolated from humans infections reported to GSS (Global Salm Surv) are listed in table 4. *S. Enteritidis* phage type 4 (PT4) is the most frequently reported phage type from poultry isolates and from human infections (Poppe, 1999).

Table 4. The top five ranking serotypes of *Salmonella* isolated from human infections worldwide by region (GSS, 2004).

Region	Year	Top five serotypes				
		1	2	3	4	5
Americas	2003	Enteritidis	Typhimurium	Infantis	Agona	Saintpaul
Europe	2003	Enteritidis	Typhimurium	Infantis	Bovismorbificans	Agona
Oceania	2003	Typhimurium	Enteritidis	Infantis	Brandenburg	Montevideo
Asia	2002	Enteritidis	Rissen	Weltevreden	Anatum	Stanley
Africa	2002	Enteritidis	Typhi	Typhimurium	Virchow	Paratyphi B

It has also been reported that 50% of the salmonellosis cases are caused by the two serotypes: Enteritidis and Typhimurium (CDC, 2004b). *S. Senftenberg*, the serotype adapted to turkeys, is also capable of causing of severe illness in humans (Nisbet and Ziprin, 2001). However, the incidence is very rare.

Serotype Java was found to be responsible for outbreaks of human infections in Scotland (Brown et al., 2003). The source was implicated to be poultry meat imported from the Netherlands. In the Netherlands, however, a substantial increase (from 2% in 1996 to 60% in 2002) in this serotype infection in poultry was found to have no impact on public health, for the reasons not known (Van Pelt et al., 2003). Moore et al. (2003) have mentioned a rare serotype-Bredeney to be responsible for human outbreaks in the UK.

The serotypes *Salmonella Gallinarum*-Pullorum, which produce Pullorum disease and Fowl typhoid in poultry, do not have human public health significance (Nisbet and Ziprin, 2001).

4.2.2 Transmission/Risk Factors

As with *Campylobacter*, the presence of *Salmonella* in poultry products presents a risk when these products are undercooked, mishandled, or allowed to cross-contaminate other foods during food production or preparation. Improper handling of the product prior to cooking can lead to contamination of surfaces and utensils used during food preparation, and infections can occur due to contact with these surfaces or items. Besides, inadequate storage, infected food handlers, inadequate thawing, improper reheating, preparation too far in advance and inadequate facilities also pose risk to consumers (Panisello et al., 2000).

The very young, old, and immunocompromised individuals are at increased risk of infection, complications or death. The minimum infectious dose required to produce salmonellosis is often low and the nature of the food vehicle and health status of the host, to a large degree, influences the susceptibility to food-borne salmonellosis (Silliker and Gabis, 1986).

4.2.3 Symptoms

The common symptoms seen due to the nontyphoid *Salmonella* serotypes are: gastroenteritis and sometimes systemic infections. The initial symptoms are dramatic diarrhoea, which is sometimes accompanied by abdominal pain, nausea, vomiting, headaches, chills, myalgia, and low-grade fever. The onset of nontyphoid salmonellosis is dose dependent and is rapid, usually occurring within 12 hours to a few days after consumption of contaminated food (Ziprin and Hume, 2001).

4.2.4 Incidence and outbreaks

Salmonellosis was the most important zoonosis in developed countries during the 1970s (Williams, 1980), and still remains an important and common human illness in spite of well-developed food and water hygiene measures (Ziprin and Hume, 2001). Also in developing countries, salmonellosis is a common and important disease. However, it may be only one among a number of important causes of diarrhoea and one among several zoonoses (Williams, 1980). It is difficult to figure out the proportion of salmonellosis resulting from poultry meat source. However, in one study, it has been reported that 67%

of food borne disease outbreaks associated with poultry are attributable to *Salmonella* spp. (Panisello et al., 2000).

In the United States, salmonellosis caused by the serotypes *S. Enteritidis* and *S. Typhimurium* has decreased 22% and 24 % respectively, since 1996 (CDC, 2004). Across the European Union too, incidence of salmonellosis has decreased substantially from 100 267 in the peak year of 1997 to 73 006 in 2001 (O'Brien and de Valk, 2003). Although the incidence of reported *Salmonella* cases has decreased, the number of reported *Salmonella* outbreaks has consistently increased in the United States (Trevejo et al., 2002).

Recent outbreak of *S. Typhimurium* phage type U290 was reported from Australia (Tomaska et al., 2003). The outbreak was found epidemiologically to be linked with bakery products. Environmental investigation of the bakery provided evidence that the source of contamination were raw eggs.

Another outbreak of *S. Typhimurium* phage type 4 linked to cheesecakes was reported from South Australia (Fielding et al., 2003). Six cases were confirmed microbiologically. The symptoms observed were abdominal pain (in 100%), diarrhoea, nausea and vomiting (in 83%), and bloody diarrhoea (in 50%). The median incubation period and duration of illness were respectively 1 and 13 days. Although the source could not be confirmed, faecally contaminated eggs seen at the bakery were implicated as one of the potential sources.

An uncommon serotype; *S. Potsdam* outbreak was seen in New South Wales, Australia in February 2002 (Unicomb et al., 2003). The infection source was identified to be Caesar salad from a restaurant which was cross-contaminated from infected eggs and the organism was detected in salad dressings. Of the 17 individuals infected, 94% developed diarrhoea, 88% developed cramps, 65% developed nausea, 59% developed fever, 53% developed headache, 35% joint pain, 29% vomiting and lethargy was seen in 24% patients. No patients were seen with bloody diarrhoea.

An international outbreak of diarrhoeal illness was reported from Mexico, among the conference attendees in November, 1996 (Shane et al., 2002). Among the 454 conference participants, 83 individuals, residing in seven different countries, reported illness, who had attended a banquet. The banquet food item "chili rellenos" was found to

be associated with illness as its ingredients, among shelled eggs and cheese, the latter was found to be contaminated with *Salmonella* (serotype unknown). The incubation period, nature and duration of illness were consistent with *Salmonella* gastroenteritis. *S. Enteritidis* phage type 4 was found in the stool specimen of ill individuals who had eaten “chili rellenos”. Although the not examined for the organisms, eggs were suspected to be the most likely source of infection.

Moore et al. (2003) have described an outbreak of Salmonellosis involving 10 people in Northern Ireland, associated with consumption of contaminated cooked chicken. Diarrhoea, fever, abdominal pain, vomiting and headache were the exhibited symptoms. A rare serotype-Bredeney was found to be the causative organism. The organism was isolated and confirmed from all of the ten patient’s faeces and a raw chicken carcass sampled.

4.2.5 Pathogenesis and consequences

The usual portal of entry is oral and invasion occurs at the ileum from where the organisms travel and infect other sites. Invasion of the intestinal mucosa and a concomitant inflammatory response leads to secretory diarrhoea mediated by activation of cyclic adenosine monophosphate (Ziprin and Hume, 2001). However, the infection can occur at any site of the body even at very unusual locations like the extradural space (Ziprin and Hume, 2001). Severe clinical consequences can include septicemia, bacteruria, arthritis, meningitis, and pneumonia (Trevejo et al., 2001). A case of *S. Enteritidis* mycotic aneurysm in a 65-year-old man has also been reported (Cited in Ziprin and Hume, 2001).

Reactive arthritis results from an inappropriate immunological response to *Salmonella* proteins. In general, the illness starts a few days after a period of enterocolitis, usually within less than 3 weeks from the onset of the gastrointestinal disturbance, and is common in those patients in whom the diarrhoeal symptoms persisted longer than usual (Ziprin and Hume, 2001).

4.2.6 Treatment

As infection with nontyphoidal *Salmonella* usually results in a self-limiting gastroenteritis, antibiotic therapy is not indicated. Fluid and electrolyte replacement therapy are generally recommended. However, when invasive disease is suspected, antibiotic therapy with chloramphenicol, ampicillin, trimethoprim-sulfamethoxazole, gentamicin, fluoroquinolones and cephalosporins are indicated. Ciprofloxacin is considered to be the drug of choice for the treatment of systemic infections with *Salmonella* spp. (Ziprin and Hume, 2001).

The efficacy of antibiotic therapy has, however, been reduced as there is an ever-increasing incidence of multidrug-resistant strains throughout the world, in both developing and developed countries (O'Brien and de Valk, 2003; Threlfall, et al., 2003; Ziprin and Hume, 2001).

5 Control

Implementation of hazard analysis and critical points (HACCP) programs is important in the control of all foodborne pathogens, including campylobacters and salmonellae. There are three practical levels at which infection control could be targeted and implemented: at the poultry farm, during poultry meat processing, and during meat handling and cooking by the end consumer.

5.1 On farm control

As control of contamination of poultry meat at abattoirs is not practical, the aim must be to produce birds free from infection at the time of slaughter. Hygienic farming practices are a strategy to prevent introduction of pathogens into a flock. Biosecurity, vaccination of the birds, use of feed supplements like probiotics and acidifiers, are the common on farm control measures.

Biosecurity and feed hygiene: Biosecurity control option is feasible only where birds are kept in closed housing conditions. Measures include enhanced biosecurity to avoid horizontal transmission of the organisms from the environment to the flock of birds.

In one study, biosecurity measures like restriction in the entry of personnel and vehicles, use of disinfectant footbaths, daily water disinfection, and location of ventilation units in

the poultry production room were associated with 50% reduction in colonization rate of *Campylobacter* in the flocks (Gibbens et al., 2001). One of the most important practices interfering with biosecurity is depopulation or thinning of the birds, mainly broilers. As depopulation in batches have been reported to increase the rate of colonization (Hald et al., 2001), the birds should be depopulated as quickly as possible and in one batch only. “All in all out” system should be practiced. Avoiding the use of *Salmonella* and *Campylobacter* contaminated crates and catcher during flock thinning is also a likely option of preventing the infection in birds (Slader et al., 2002).

Legislation regarding compulsory “slaughter and compensation” for the breeder flock, aimed at producing *Salmonella*-free chickens has worked well in control of *Salmonella* in some European countries (Wilson, 2002). In the UK, contamination rate of chicken carcass with *Salmonella* has fallen from 80% in the 1980 to 5% in 2001, as a result of removal of breeding flock infected with *S. Enteritidis* and improvements in on-farm biosecurity and feed hygiene (Humphrey, 2004).

However, biosecurity measures appropriate for one type of organism may be ineffective for another. Wilson (2002) suggested that the biosecurity which contributed to reducing *Salmonella* infection did not impact on *Campylobacter* as the epidemiology of *Campylobacter* in poultry meat production is quite different from that of *Salmonella*. This may also be due to the fact that campylobacters are more ubiquitous than salmonellae and also have low infective dose in birds (Humphrey, 2004).

Competitive exclusion, probiotics and feed acidifiers: As the use of antibiotic in feed to prevent the colonization of *Campylobacter/Salmonella* has been banned in most of the developed countries, use of probiotics appears to be a promising alternative. Recently there has been considerable research interest in improving the efficacy of competitive exclusion. One recent *in vitro* study indicated that *Lactobacillus* (P93) strain isolated from chicken gut had inhibitory effect in *Campylobacter* growth, whereas the probiotic bacteria *Enterococcus* spp. and colicin-producing *E. coli* had no effect (Chaveerach et al., 2004).

One recent study in the Netherlands (Heres et al., 2004) reported that acidification of feed by organic acids (lactic acid; 5.7%, acetic acid 0.7%) had limited effects on preventing the colonization of *Campylobacter* in broiler chickens, however, no effect was seen in

case of *Salmonella*. Also the various sugars (lactose, arabinose, galactose etc.) provided to chickens either in feed or water are believed to avoid the adherence of *Salmonella* Typhimurium to the chick cecal surfaces (Nisbet and Ziprin, 2001). Research on the effect of administration of bacteriophage on reduction of the numbers of Campylobacters on chickens is underway with promising results (Conner, cited in Humphrey, 2004).

Vaccination: Although not clearly established, vaccination in the broiler breeders against salmonellosis was thought to reduce salmonellosis in broilers and the consequent carcass contamination with these organisms (Wilson, 2002). Recently, a live avirulent *Salmonella* vaccine was shown to induce excellent protection against intestinal, visceral and reproductive tract, and egg colonization, invasion and/or contamination by *Salmonella* (Nisbet and Ziprin, 2001). In the UK, the introduction of vaccination of commercial egg laying flock, since 1997, is thought to be the main reason for marked fall in human infection with *S. Enteritidis* (Humphrey, 2004). Vaccines against campylobacters in poultry are yet to develop.

5.2 Processing controls

Processing is an important site of reduction of pathogens from the carcass, as well as an ideal site of cross-contamination. The number and incidence of *Salmonella* and *Campylobacter* in raw poultry carcass are greatly affected by the operating conditions of scalding and defeathering, evisceration, washing, skinning, boning, portioning, chilling, decontaminating (irradiation) and freezing.

In commercial scalding, temperature is set in a range of 50 to 60°C for 2 to 2.5 min. One study found that increasing of scalding water temperature from 50 to 60°C reduced the number of *C. jejuni* and *S. Typhimurium* by approximately 6 log CFU/ ml (Yang et al., 2001). In contrast, Humphrey (2004) found that hot water (up to 80°C) treatments were not much more effective in removing campylobacters as compared to cold water. So, he suggested that heat treatment is not a significant control measure, particularly when chicken carcasses are to be sold whole. He also concluded that, when attached to chicken skin, campylobacters can resist high temperatures.

Treatment of wash water has been found to be a potential processing control to reduce contamination. Alternatives are treatment with chlorine water, electrolyzed water,

trisodium phosphate, cetylpyridinium chloride, hydrogen peroxide etc. Chlorine water (up to 50 ppm) is generally used for washing the chicken carcass to reduce bacterial load on the surface (Yang et al., 2001). A recent study demonstrated that electrolyzed water was as effective as chlorinated water in reducing *C. jejuni* from chicken carcass by 3 log₁₀ CFU per gram (Park et al., 2002). In addition, it could also prevent cross-contamination of processing environments (Park et al., 2002).

Air chilling of the carcass has been suggested to be more effective in eliminating the contamination as compared to water chilling (Jacobs-Reitsma, 2000).

Freezing of the carcasses is another way of reducing the bacterial load. It was recently demonstrated in a study from Ireland that avoiding consumption of fresh chicken (eating only the frozen chickens) reduced the 72% cases (of 158 per 100000 inhabitants) of domestic campylobacteriosis (Takkinen and Ammon, 2003).

Irradiation can eliminate the potentially pathogenic non-spore forming bacteria including *Salmonella* and *Campylobacter* from suspected food products like poultry without affecting the quality of the product (Farkas, 1998).

Education in hygienic handling of foods for abattoir workers and those involved in the production of raw meat is essential to keep microbiological contamination to minimum.

5.3 Food handling

Food handling is the last control point in the farm-to-fork food safety continuum for preventing foodborne campylobacteriosis and salmonellosis. As eating of poorly cooked chicken is the most important risk factor, the public should thoroughly cook chicken. Proper cooking of poultry will certainly ensure that *Salmonella* and *Campylobacter* infection does not occur due to ingestion of the cooked product. Poultry should be heated to an internal temperature of 82°C to kill campylobacters (Graves et al., 1998). Meat thermometers are available that can be used to measure the internal temperature of meat and poultry.

Avoidance of cross-contamination in kitchen is another important control option. Failure to wash hands, utensils, or countertops can lead to contamination of foods that will not be cooked. Careful kitchen sanitation involving cleaning and disinfection of food contact surfaces, hands, and utensils following contact with raw poultry is recommended. In

addition, storing of raw poultry should be done separately from foods that will be served without subsequent cooking. Food handlers should be attentive during preparation of food and should observe hygienic rules of food preparation. They should be aware that pathogens can be present on raw poultry and that foodborne disease can be prevented by adhering to the following measures(Graves et al., 1998): 1) raw poultry and meat should be prepared on a separate countertop or cutting board from other food items; 2) all utensils, cutting boards, and countertops should be cleaned with hot water and soap after preparing raw poultry or meat and before preparing other foods; 3) hands should be washed thoroughly with soap and running water after handling raw poultry or meat; and 4) poultry should be cooked thoroughly to an internal temperature of 82°C or until the meat is no longer pink and juiciness run clear.

6 Conclusions

Although the advances in the 20th century such the application HACCP have contributed to improvements in microbiological safety of most foods, food borne diseases remain a significant cause of morbidity and mortality throughout the world. Foodborne salmonellosis and campylobacteriosis continue to remain the most important cause of bacterial gastroenteritis worldwide. Even in the most developed countries of the world like the United States, despite of the stringent control efforts, considerable reduction in the cases of human infections has not been achieved yet. Intensive animal production system, complex chain of meat handling, processing, marketing and eating of raw or undercooked meat may be the explanation for this (Williams, 1980). Other factors making the controls difficult are like difficulty in tracing back the origin of infection and antimicrobial resistance developed by the organisms (O'Brien and de Valk, 2003). Control is difficult in developing countries also because of the substantial gap in the knowledge about the epidemiology of these organisms in these countries.

Some success, however, has been achieved in control of foodborne salmonellosis. The reduction in number of salmonellosis is believed to be to some extent contributed by the reduction of salmonellosis in poultry, either by enhanced biosecurity or “slaughter and compensation” campaign (Humphrey, 2004). These measures, however, proved to be largely ineffective against campylobacters (Wilson, 2002), and new interventions are

needed. Identification of the events that predisposes the colonization of campylobacters in chickens can give very important clues for control of these organisms (Humphrey, 2004).

While many such human infections could be avoided through correct hygiene procedures in the handling of foods, only coordinated farm to fork approach is likely to achieve permanent and significant reductions in foodborne bacterial zoonoses in the future. This underlines the urgency of improved coordination between the veterinary and public health authorities. One can be hopeful regarding control of these foodborne infections as enhanced surveillance of the organisms, serotyping and antimicrobial susceptibility testing are being conducted by various surveillance networks like Global Salm Surv (GSS, 2004), and Enter-net (Fisher, 1999) to devise effective control strategies.

References

- Anonymous.** *Salmonella* in meat and poultry products decline drastically, American Veterinary Medical Association, 2004. Retrieved: September 16, 2004 from www.electronicipc.com/jornalez/mo/detail/.cfm?CODE=04290010650302&cfid
- Adhikari B.** Sparrows, flies, and rodents as reservoirs of *Campylobacter* spp. on a dairy farm. MVSc thesis, Massey University, Palmerston North, New Zealand, 2003
- Agulla A, Merino FJ, Villasante PA, Saz JV, Diaz A, Velasco AC.** Evaluation of Four Enrichment Media for Isolation of *Campylobacter jejuni*. *Journal of Clinical Microbiology* 25, 174-5, 1987
- Allerberger F, Al-Jazrawi N, Kreidl P, Dierichm MP, Feierl G, Hein I, Wagner M.** Barbecued Chicken Causing a Multi-State Outbreak of *Campylobacter jejuni* Enteritis. *Infection* 31, 19-23, 2003
- Altekruse SF, Stern NJ, Fields PI, Swerdlow DL.** *Campylobacter jejuni*—An Emerging Foodborne Pathogen. *Emerging infectious diseases* 5, 1999
- Altekruse SF, Tollefson LK.** Human campylobacteriosis: a challenge for the veterinary profession. *Journal of the American Veterinary Medical Association* 223, 445-52, 2003
- Andersen RC, Ziprin RL.** Bacteriology of *Salmonella*. In: Hui YH, Pierson MD, Gorham RJ (eds). *Foodborne Disease Handbook*. Pp 247-63. Marcel Dekker, Inc., New York, 2001
- Antunes P, Reu C, Sousa JC, Peixe L, Pestana N.** Incidence of *Salmonella* from Poultry Products and their Susceptibility to Antimicrobial Agents. *International Journal of Food Microbiology* 82, 97-103, 2003
- Baker M, Ball A, Devane M, Garrett N, Gilpin B, Hudson A, Klena J, C. N, Savill M, Scholes P, Williams D.** Potential Transmission Route of *Campylobacter* from Environment to Humans. Ministry of Health, New Zealand, 2002
- Brown DJ, Mather H, Browing LM, Cola JE.** Investigation of human infections with *Salmonella* enterica serovar Java in Scotland and possible association with imported poultry. *Euro Surveillance* 8, 35-40, 2003
- CDC.** *Campylobacter* Infections, Centre for Disease Control and Prevention, 2004a. Retrieved: September 1, 2004 from: www.cdc.gov/ncidod/diseaseinfo/campylobacter_t.htm

CDC. Salmonellosis-Technical Information, Centre for Disease Control and Prevention, 2004b. Retrieved: September 1, 2004 from: http://www.cdc.gov/ncidod/dbmd/diseaseinfo/salmonellosis_t.htm

Chaveerach P, Lipman LJA, van Knapen F. Antagonistic activities of several bacteria on in vitro growth of 10 strains of *Campylobacter jejuni/coli*. *International Journal of Food Microbiology* 90, 43-50, 2004

Coker AO, Isokpehi RD, Thomas BN, Amisu KO, Obi CL. Human Campylobacteriosis in Developing Countries. *Emerging infectious diseases* 8, 237-43, 2002

Corry JIE, Post DE, Colin P, Laisney MJ. Culture media for the isolation of campylobacters. *International Journal of Food Microbiology* 26, 43-76, 1995

Corry JEL, Atabay HI. Poultry as a source of *Campylobacter* and related organisms. *Journal of Applied Microbiology* 90, 96S-114S, 2001

Donnison A. Isolation of Thermotolerant *Campylobacter*-Review and Methods for New Zealand Laboratories. Ministry of Health, 2003

Doyle MP, Jones DM. Food-Borne Transmission and Antibiotic Resistance of *Campylobacter jejuni*. In: Nachamkin I, Blaser JM, Thompkins LS (eds). *Campylobacter jejuni: Current status and Future Trends*. Pp 45-8. American Society for Microbiology, Washington, DC, 1992

Ethelberg S, Olsen KEP, Gerner-Smidt P, Molbak K. Household Outbreaks among Culture-confirmed Cases of Bacterial Gastrointestinal Disease. *American Journal of Epidemiology* 159, 406-12, 2004

Farkas J. Irradiation as a method for decontaminating food: A review. *International Journal of Food Microbiology* 44, 189-204, 1998

Fielding JE, Snell P, Adriana M, Fabbro LD, Raupach J. An outbreak of *Salmonella* Typhimurium phage type 4 linked to cold set cheesecake. *Communicable Diseases Intelligence* 27, 2003. Retrieved: August 30, 2004 from: www.cda.gov.au/pubs/cdi/2003/cdi2704/htm/cdi2704m.htm

Fisher IST. The Enter-net international surveillance network-how it works. *Euro Surveillance* 4, 52-5, 1999

Friedman C, Neimann J, Wegener H, Tauxe R. Epidemiology of *Campylobacter jejuni* Infections in the United States and Other Industrialized Nations. In: Nachamkin I, Blaser M (eds). *Campylobacter*. American Society for Microbiology, Washington, DC, 2000

Geilhausen B, Schutt-Gerowitt H, Aleksic S, Koenen R, Mauff G, Pulverer G. *Campylobacter* and *Salmonella* contamination of fresh chicken meat. In: Newell DG, Ketley JM, Feldman RA (eds). *Campylobacters, Helicobacters, and Related Organisms*. Pp 105-8. Plenum Press, New York, 1996

Gibbens JC, Pascoe SJ, Evans SJ, Davies RH, Sayers AR. A trial of biosecurity as a means to control *Campylobacter* infection of broiler chickens. *Preventive Veterinary Medicine* 48, 85-99, 2001

Gillespie A. Is *Campylobacter jejuni* enteritis a weekend disease? *Journal of Infection*, In Press.

Goossens H, Butzler J-P. Isolation and Identification of *Campylobacter* Spp. In: Nachamkin I, Blaser JM, Thompkins LS (eds). *Campylobacter jejuni: current status and future trends*. Pp 93-109. American Society for Microbiology, Washington, D.C., 1992

Graves TK, Bradley KK, Crutcher JM. Outbreak of *Campylobacter* Enteritis Associated with Cross-Contamination of Food. *Morbidity and Mortality Weekly Report* 47, 129-31, 1998

GSS. Top 15 *Salmonella* serotype list. 2004. Retrieved: September 10, 2004 from: http://sherlock.dzc.dk/pls/portal30/ARJ.ALL_SERO_SUMMARIES_REP.SHOW_P_ARMS

Gupta A, Nelson JM, T.J. B, Tauxe R, Tossiter SP, Friedman CR, Joyce KW, Smith KE, Jones TF, Hawkins MA, Shiferaw B, Beebe JL, Vugia DJ, Rabatsky-Ehr T, Benson JA, Root TP, Angulo FJ. Antimicrobial Resistance among *Campylobacter* Strains, United States, 1997-2001. *Emerging infectious diseases* 10, 1102-9, 2004

Hald B, Rattenborg E, Madsen M. Role of batch depletion of broiler houses on the occurrence of *Campylobacter* spp. in chicken flocks. *Letters in Applied Microbiology* 32, 253-6, 2001

Heres L, Engel B, Urlings HAP, Wagenaar JA, van Knapen F. Effect of acidified feed on susceptibility of broiler chickens to intestinal infection by *Campylobacter* and *Salmonella*. *Veterinary Microbiology* 99, 259-67, 2004

Humphrey TJ. Contamination of Eggs and Poultry meat with *Salmonella enterica* Serovar Enteritidis. In: *Salmonella enterica Serovar Enteritidis in Humans and Animals*. Pp 183-92. Iowa State University, Ames, Iowa, 1999

Humphrey T. Control of *Campylobacter* spp. in the food chain: a far from simple task. *Culture* 25, 6-9, 2004

Hunt J, Abeyta C. *Campylobacter*. In: Bacteriological Analytical Manual online, U.S. Food and Drug Administration Centre for Food Safety & Applied Nutrition, International Gaithersburg, MD,, 2001. Retrieved: August 21, 2004 from: www.cfsan.fda.gov/~ebam/bam-7.html

Jacobs-Reitsma W. *Campylobacter* in the food supply. In: Nachamkin I, Blaser M (eds). *Campylobacter*. Pp 467-81. American Society for Microbiology, Washington, DC, 2000

Jorgensen F, Bailey R, Williams S, Henderson P, Wareing DRA, Bolton FJ, Frost JA, Ward L, Humphrey TJ. Prevalence and numbers of *Salmonella* and *Campylobacter* spp. on raw, whole chickens in relation to sampling methods. *International Journal of Food Microbiology* 76, 151-64, 2002

Mead GC. HACCP in Primary Processing: Poultry. In: Brown M (ed) *HACCP in The Meat Industry*. Pp 123-53. Woodhead Publishing Limited, Cambridge, England, 2000

Merritt A, Miles R, Bates J. An outbreak of *Campylobacter* enteritis on an island resort, north Queensland. *Communicable Disease Intelligence* 23, 215-9, 1999

Moore JE, Stanley T, Smithson R, O'Malley H, Murphy PG. Outbreak of *Campylobacter* food-poisoning in Northern Ireland. *Clinical Microbiology & Infection* 6, 397-8, 2000

Moore JE, O'Riordan L, Wareing DRA, Doyle R, Lanser J, Stanley T, Matsuda M, Matsui T, Murphy PG. Phenotypic and genotypic relationship between *Campylobacter* spp isolated from humans and chickens in Northern Ireland a comparison of three phenotyping and two genotyping schemes. *International Journal of Hygiene and Environmental Health* 206, 211-6, 2003

Nachamkin I. *Campylobacter* and Arcobacter. In: Murray PR, Baron EJ, Pfaller MA, Tenover FC, Tenover RH (eds). *Manual of Clinical Microbiology*. Pp 483-91. American Society for Microbiology, Washington, D.C., 1995

Nachamkin I, Engberg J, Aarestrup FM. Diagnosis and antimicrobial susceptibility of *Campylobacter* species. In: Nachamkin I, Blaser M (eds). *Campylobacter*. American Society for Microbiology, Washington, D.C., 2000

Newell DG, Wagenaar JA. Poultry infections and their control at farm level. In: Nachamkin I, Blaser MJ (eds). *Campylobacter*. Pp 497-509. American Society for Microbiology, Washington, DC, 2000

Nisbet DJ, Ziprin RL. Salmonellosis in Animals. In: Hui YH, Pierson MD, Gorham RJ (eds). *Foodborne Disease Handbook*. Pp 265-84. Marcel Dekker, Inc., New York, 2001

O'Brien B, de Valk H. *Salmonella*- "old" organism, continued challenges! *Euro Surveillance* 8, 29-31, 2003

O'Sullivan NA, Fallon R, Carroll C, Smith T, Maher M. Detection and differentiation of *Campylobacter jejuni* and *Campylobacter coli* in broiler chicken samples using a PCR/DNA probe membrane based colorimetric detection assay. *Molecular and Cellular Probes* 14, 7-16, 2000

Oberhelman RA, Taylor DN. *Campylobacter* infections in developing countries. In: Nachamkin I, Blaser JM (eds). *Campylobacter*. Pp 139-53. American Society for Microbiology, Washington, D.C., 2000

Panisello PJ, Rooney R, Quantick PC, Stanwell-Smith R. Application of foodborne disease outbreak data in the development and maintenance of HACCP systems. *International Journal of Food Microbiology* 59, 221-34, 2000

Park H, Hung YC, Brackett RE. Antimicrobial effect of electrolyzed water for inactivating *Campylobacter jejuni* during poultry washing. *International Journal of Food Microbiology* 72, 77-83, 2002

Poppe C. Epidemiology of *Salmonella enterica* Serovar Enteritidis. In: Saeed AM, Gast RK, Potter ME, Wall PG (eds). *Salmonella enterica Serovar Enteritidis in Humans and Animals*. Pp 3-18. Iowa State University Press, Ames, Iowa, 1999

Quinn PJ, Markey BK, Cater ME, Donnelly WJC, Leonard FC. *Veterinary Microbiology and Microbial Diseases*, First edn. Blackwell Science, Inc., Oxford, 2002

Quinn PJ, Markey BK. *Concise Review of Veterinary Microbiology*, First edn. Blackwell Publishing Limited, Oxford, UK, 2003

Radkowski M. Occurrence of *Salmonella* spp. in consumption eggs in Poland. *International Journal of Food Microbiology* 64, 189-91, 2001

Ransom GM, Rose BE. Isolation, Identification, and Enumeration of *Campylobacter jejuni/coli* from Meat and Poultry products. In: *Microbiology Laboratory Guidebook*. Pp 6-1-6-10. USDA/FSIS, 1998

Raupach JCA, Hundy RL. An outbreak of *Campylobacter jejuni* infection among conference delegates. *Communicable Disease Intelligence* 27, 380-3, 2003

Shane AL, Roels TH, Goldoft M, Herikstad H, Hedberg C, Angulo FJ. Foodborne disease in our global village: a multinational investigation of an outbreak of *Salmonella* serotype Enteritidis phage type 4 infection in Puerto Vallarta, Mexico. *International Journal of Infectious Diseases* 6, 98-102, 2002

Silliker JH, Gabis DA. *Salmonella*. In: Pearson AM, Dutson TR (eds). *Advances in Meat Research: Meat and Poultry Microbiology*. Pp 209-29. AVI Publishing, Connecticut, 1986

Slader J, Domingue G, Jorgensen F, McAlpine K, Owen RJ, Bolton FJ, Humphrey TJ. Impact of Transport Crates Reuse and of Catching and Processing on *Campylobacter* and *Salmonella* Contamination of Broiler Chickens. *Applied and Environmental Microbiology* 68, 713-9, 2002

Sopwith W, Ashton M, Frost JA, Tocque K, O'Brien S, Regan M, Syed Q. Enhanced Surveillance of *Campylobacter* Infection in the North West of England 1997-1999. *Journal of Infection* 46, 35-45, 2003

Stern NJ. Reservoirs for *Campylobacter jejuni* and Approaches for Intervention in Poultry. In: Nachamkin I, Blaser JM, Thompkins LS (eds). *Campylobacter jejuni: Current Status and Future Trends*. Pp 49-60. American Society for Microbiology, Washington, D.C., 1992

Takkinen J, Ammon A. The 11th International Workshop on *Campylobacter*, *Helicobacter* and related Organisms. *Euro Surveillance* 8, 219-22, 2003

Tam CC, O'Brien SJ, Adak GK, Meakins SM, Frost JA. *Campylobacter coli*-an important foodborne pathogen. *Journal of Infection* 47, 28-32, 2003

Tauxe R. Epidemiology of *Campylobacter jejuni* infections in the United States and other industrial nations. In: Nachamkin I, Blaser MJ, Thompkins LS (eds). *Campylobacter jejuni: current status and future trends*. Pp 9-19. American Society for Microbiology, Washington, D.C., 1992

Threlfall EJ, Fisher IST, Berghold C, Gemet-Smith P, Tschape H, Cormican N, Luzzi F, Schrieder F, Wannell W, Machado J, Edwards G. Antimicrobial drug resistance in isolates of *Salmonella enterica* from cases of salmonellosis in humans in Europe in 2000: results of international multi-centre surveillance. *Euro Surveillance* 8, 41-5, 2003

Tomaska NA, Lalor K, Gregory JE, O'Donnell HJ, Dawood F, Williams MC. *Salmonella* Typhimurium U290 outbreak linked to bakery. *Communicable Diseases Intelligence* 27, 2003. Retrieved: August 29, 2004 from: www.cda.gov.au/pubs/cdi/2003/cdi2703/htm/cdi2703n.htm

Trevejo RT, Courtney JG, Starr M, Vugia DJ. Epidemiology of Salmonellosis in California, 1990-1999: Morbidity, Mortality and Hospitalization Costs. *American Journal of Epidemiology* 157, 48-57, 2002

Unicomb L, Bird P, Dalton C. Outbreak of *Salmonella* Potsdam associated with salad

dressing at a restaurant. *Communicable Diseases Intelligence* 27, 2003. Retrieved: September 5, 2004 from: www.cda.gov.au/pubs/cdi/2003/cdi2704/htm/cdi27041.htm

Van Pelt W, Mather M, Browing LM, Cola JE. Explosive increase of *Salmonella* java in poultry in the Netherlands: Consequences for public health. *Euro Surveillance* 8, 31-5, 2003

Vierikko A, Haarmann M, Siitonen A, Ruutu P, Rautelin H. Domestically Acquired *Campylobacter* Infections in Finland. *Emerging infectious diseases* 10, 127-30, 2004

WHO. Fact Sheets: *Campylobacter*. WHO, 2002a. Retrieved: August 25, 2004 from: www.who.int/inf-fs/en/fact255.html

WHO. Food safety and foodborne illness. World Health Organization, 2002b. Retrieved: September 1, 2004 from: www.who.int/mediacentre/factsheets/fs237/en/print.html

Williams L. Salmonellosis. In: Steele J, Stoenner H, William K (eds). *CRC Handbook series in Zoonoses*. Pp 11-37. CRC Press, Florida, 1980

Wilson IG. *Salmonella* and *Campylobacter* contamination of raw retail chickens from different procedures: a six year survey. *Epidemiology and Infection* 129, 635-45, 2002

Yang H, Li Y, Johnson MG. Survival and Death of *Salmonella* Typhimurium and *Campylobacter jejuni* in Processing Water and on Chicken Skin during Poultry Scalding and Chilling. *Journal of Food Protection* 64, 770-6, 2001

Ziprin RL, Hume MH. Human Salmonellosis: General Medical Aspects. In: Hui YH, Pierson MD, Gorham RJ (eds). *Foodborne Disease Handbook: Bacterial Pathogens*. Pp 285-321. Marcel Dekker, Inc., New York, 2001

Zorman T, Mozina SS. Classical and molecular identification of thermotolerant campylobacters from poultry meat. *Food Technology and Biotechnology* 40, 177-84, 2002

Zweifel C, Zychowska MA, Stephan R. Prevalence and characteristics of Shiga toxin-producing *Escherichia coli*, *Salmonella* spp. and *Campylobacter* spp. isolated from slaughtered sheep in Switzerland. *International Journal of Food Microbiology* 92, 45-53, 2004