Food Safety Risk Profile for *Salmonella* species in broiler (young) chickens

Compiled by the CCFH Working Group on Guidelines for control of *Campylobacter* and *Salmonella* spp. in broiler (young bird) chicken meat

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Some parts of this risk profile are in its present state not representative regarding the global situation due to lack of data from many countries, especially from Latin America and the Caribbean, Africa, the Near East and to a certain extent Asia. It is therefore requested that governments contribute with relevant data to the different sub-headings in the document.
# List of contents

1  Hazard-food commodity combinations of concern ............................................ 5
  1.1  Hazards of concern ....................................................................................... 5
  1.2  The food product of concern ....................................................................... 5
  1.3  Occurrence of the hazard in the food chain .................................................. 5

2  Description of the public health problem .......................................................... 5
  2.1  Description of the hazard ............................................................................... 5
    2.1.1  Taxonomy ................................................................................................. 5
    2.1.2  In vitro growth characteristics .................................................................. 5
    2.1.3  Temperature effects .................................................................................. 6
    2.1.4  Virulence ................................................................................................... 6
    2.1.5  Resistance to antimicrobial agents ............................................................. 7
  2.2  Characteristics of the disease in humans ....................................................... 8
    2.2.1  Acute illness .............................................................................................. 8
    2.2.2  Susceptible populations ............................................................................. 8
    2.2.3  Dose response ............................................................................................ 8
    2.2.4  Annual incidence rate in humans including differences between age and sex ................................................................................................................. 9
    2.2.5  Outcome of exposure .................................................................................. 11
    2.2.6  Availability and nature of treatment ........................................................ 12
    2.2.7  Percentage of annual cases attributable to foodborne transmission ....... 12
  2.3  Epidemiology of foodborne disease ............................................................... 13
    2.3.1  Implicated food and factors that influence transmission .......................... 13
    2.3.2  Frequency and characteristics of foodborne sporadic cases .................. 13
    2.3.3  Epidemiological data from outbreak investigations ............................... 13
  2.4  Regional and seasonal influences on the incidence of foodborne illness due to the hazard ................................................................. 14
  2.5  Economic impact of the disease ................................................................. 14
    2.5.1  Medical, hospital costs .............................................................................. 14
    2.5.2  Working days lost due to illness, etc. ......................................................... 14

3  Food Production, processing, distribution and consumption .................................. 15
  3.1  Characteristics of the commodity that is involved and that may impact on risk management ........................................................................................................... 15
  3.2  Description of the farm to table continuum .................................................. 15
    3.2.1  Primary production .................................................................................... 16
    3.2.2  Transport of live poultry ......................................................................... 18
    3.2.3  Primary processing .................................................................................... 19
    3.2.4  Secondary processing, distribution and retail sale of product ............... 21
    3.2.5  Consumer handling .................................................................................. 21
  3.3  Summary of current risk management practices .......................................... 22
    3.3.1  Primary production .................................................................................... 22
    3.3.2  Processing .................................................................................................. 23
    3.3.3  Food service and the home ....................................................................... 23

4  Other Risk Profile Elements .............................................................................. 23
  4.1  The extent of international trade of the food commodity ................................... 23
  4.2  Existence of regional/international trade agreements and how they may affect the public health impact with respect to the specific hazard/commodity combination ........................................................................... 23
  4.3  Public perceptions of the problem and the risk ............................................. 23

5  Available Information and Major Knowledge Gaps .......................................... 23
5.1 Existing national MRAs on the hazard/commodity combination..............23
5.2 Areas where major absences of information exist that could hamper
MRM activities including, if warranted, the conduct of an MRA ...............24
6 References...........................................................................................................24
1 Hazard-food commodity combinations of concern

1.1 Hazards of concern
Non-typhoidal Salmonella spp.

1.2 The food product of concern
Fresh broiler chicken meat (where fresh is defined according to CCMH); whole chicken carcasses and portions, excluding internal organs.

1.3 Occurrence of the hazard in the food chain
Salmonellosis is an important cause of enteric illness. A wide range of foods has been implicated in foodborne salmonellosis. However, as the disease is primarily zoonotic, foods of animal origin have been consistently implicated as the main sources of human salmonellosis (FAO/WHO, 2002).

Poultry and poultry products are often implicated in sporadic cases and in outbreaks of human salmonellosis (Bryan and Doyle, 1995; Humphrey, 2000).

2 Description of the public health problem

2.1 Description of the hazard

2.1.1 Taxonomy
The genus Salmonella belongs to the family Enterobacteriaceae and consists of two species, Salmonella enterica and Salmonella bongori. S. enterica is further divided into six subspecies: S. enterica subsp. enterica, S. enterica subsp. salamae, S. enterica subsp. arizonae, S. enterica subsp. diarizonae, S. enterica subsp. houtenae and S. enterica subsp. indica. More than 2400 Salmonella serotypes have been identified. A few of these are human–host-adapted serotypes, e.g. S. Typhi and S. Paratyphi. These serotypes are referred to as typhoidal salmonellae. This risk profile will discuss only non-typhoidal Salmonella belonging to the species enterica.

Salmonella typing is primarily performed using serological identification of somatic (O), flagella (H), and capsular (K) antigens. Further subtyping may be performed using susceptibility to bacteriophages. These types are denoted as phage type (PT) or definitive phage type (DT) numbers. These two terms are interchangeable and both are used in the literature.

2.1.2 In vitro growth characteristics
Salmonellae are gram-negative, oxidase negative, rod shaped, facultative anaerobic bacteria. They are motile (a few exceptions exist) due to the presence of flagella. Growth occurs in the range 5°C – 46°C with an optimum between 35°C and 43°C. The pH for optimum growth is between 6.6 and 8.2 with values greater than 9 or lower than 4 inhibiting growth. Depending on the acid used, minimum pH for growth may be as high as 5.5. Minimum water activity for growth is 0.94 in media with a neutral pH but higher values are required as pH decreases towards growth minimum (SCVPH, 2000).

Salmonella can grow in the presence or absence of air. The growth rate on beef muscle stored at 20°C under nitrogen is only slightly less than that obtained when
stored under air. At high concentrations of CO₂ (50-60%), growth is strongly inhibited on crab meat, beef steak and ground beef at a temperature of 10 to 11°C, but at 20°C there is little inhibition (AIFST, 2003).

### 2.1.3 Temperature effects

Heat resistance of salmonellae varies considerably between strains. The type of food involved and growth conditions such as pH and water activity also affect the heat resistance as well as other environmental factors. D-values (the time in minutes at a given temperature to get a 90% reduction in the number of viable bacteria) at 60°C usually vary between 2-6 minutes. At 70°C the D-value is usually 1 minute or less. (ICMSF, 1996; Doyle and Mazottta, 2000).

*Salmonella* can survive for long periods under refrigeration and freezing does not ensure the inactivation of salmonellae in foods. Survival for >10 weeks in butter stored at −23 and 25°C has been noted. *Salmonellae* can survive for 28 days on the surfaces of vegetables under refrigeration. Some foods, including meat, appear to be protective of *Salmonella* during freezing and frozen storage (Lake *et al*., 2004). AIFST (2003) report that rapid freezing promotes survival and that lower storage temperatures and less fluctuations in temperature give greater survival. Storage temperatures near the freezing point result in most death or injury. In minced chicken breast (pH 5.8), 60-83% of *Salmonella* cells survived storage at −20°C for 126 days, whereas at -2°C and -5°C only 1.3% to 5.8% were still viable after 5 days.

### 2.1.4 Virulence

After oral uptake *Salmonella* is successively exposed to low pH in the stomach, to the strong antimicrobial effect of bile, to decreasing oxygen supply, to the normal gut-flora and its metabolites, to intestinal peristalsis and cationic antimicrobial peptides present on the surface of epithelial cells (Rychlik and Barrow, 2005). The encounter with these stressful environments induces expression of a number of genes whose products are essential for *Salmonella* to invade the intestinal epithelium and infect the host.

The ability to cause disease relies on several virulence determinants. Some of these may be considered virulence determinants in the broad sense. Genes involved in nutrient biosynthesis/uptake, stress response (both in and outside the host) and repair of cell damage are among those. These genes may be considered housekeeping genes and are present in other closely related bacteria, such as *E. coli* (Bäumler *et al*., 2000). Another group of virulence genes specific for the genus *Salmonella* encode adaptations to overcome host defence mechanisms and may therefore be called true virulence determinants. The expression of both groups of virulence genes is regulated in response to environmental signals in the host. The regulatory genes mediating this control may also be considered virulence determinants (Bäumler *et al*., 2000).

The genetic control of *Salmonella* virulence is not fully elucidated. However both plasmid and chromosomal genes are involved.

Many of the virulence genes of *S. enterica* are chromosomal genes located on pathogenicity islands referred to as *Salmonella* Pathogenicity Islands (SPI). These genes are believed to have been acquired by *Salmonella* from other bacterial species through horizontal gene transfer (van Asten & van Dijk, 2005). This includes functions such as host cell invasion and intracellular pathogenesis. At present 12 different SPI have been described. The role in pathogenesis of some SPI is well
described but the function in virulence of many genes within SPI is yet not understood (Hensel, 2004).

At least six serotypes of Salmonella ( Abortusovis, Choleraesuis, Dublin, Enteritidis, Gallinarum/Pullorum and Typhimurium) harbour a virulence plasmid (although not all isolates of these serotypes). These plasmids vary in size between the serotypes. All these plasmids contain the salmonella plasmid virulence (spv) locus. This locus harbours five genes designated spvRABCD (van Asten & van Dijk, 2005). The first gene spvR encodes an activator of spvABCD, but the exact function of the encoded proteins is not fully known. These genes are induced by growth restriction, reduced nutrient supply or lowered pH and are involved in intra-macrophage survival of Salmonella (Rychlik et al., 2005). Other virulence factors of Salmonella include production of endotoxins and exotoxins, and presence of fimbrie and flagella. The role of these factors in the pathogenesis of Salmonella spp. is not fully established (van Asten & van Dijk, 2005).

2.1.5 Resistance to antimicrobial agents

Antimicrobial- resistant strains of Salmonella spp. are now widespread all over the world and are causing great concern not least due to the spread of multi-drug-resistant strains. In developed countries it is becoming more and more accepted that a majority of resistant strains are of zoonotic origin and have acquired their resistance in an animal host before being transmitted to humans through the food chain (Mølbak et al., 2002; ThriftFall, 2002; WHO 2004).

In animal production antimicrobial drugs are used for therapy, prophylaxis and growth promotion. The use of such drugs causes a selective pressure to be imposed on bacterial populations and antimicrobial resistances are selected. The pool of resistance genes is thus spread in the environment (WHO, 2004).

Antibiotic resistance determinants are usually encoded on plasmids but can also be present on the Salmonella chromosome. Resistance can be achieved through mutations and acquisition of resistance encoding genes. Cointegrates of resistance and virulence plasmids in Salmonella have been observed. This means that antibiotic pressure may select for these plasmids and that both resistance and virulence traits are obtained simultaneously. This may lead to more antibiotic-resistant and virulent Salmonella trains. The outcome of such a scenario is to a large extent dependent on the use of antibiotics (Fluit, 2005). Data suggesting that disease caused by resistant strains can be more severe than disease caused by susceptible strains have been published (Lee et al., 1994; Helms et al., 2004; Helms et al., 2002)

The prevalence of resistant isolates in different countries where intensive animal production is common is between 10-30 %. When concentrating on strains isolated from food-producing animals that are held under strong antibiotic selective pressures and are important to human health the prevalence of resistant strains can be very high, up to 60-90% (Helmuth, 2000).

In 1999, 8,508 Salmonella isolates of animal origin were tested against 17 antimicrobial drugs in the USA. The results are shown in Table 2.1 and indicate that many Salmonella serotypes are resistant to some of the antibiotics commonly used in human and animal health, and as growth promoters in the animal production industry (Headrick and Cray, 2001).
Table 2.1 Results from the USA of antimicrobial sensitivity assays for 8,508 *Salmonella* isolates

<table>
<thead>
<tr>
<th>Antimicrobial</th>
<th>Percent Sensitive</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amikacin</td>
<td>&gt;99.9</td>
</tr>
<tr>
<td>Amoxicillin/clavulanic acid</td>
<td>88.4</td>
</tr>
<tr>
<td>Ampicillin</td>
<td>81.9</td>
</tr>
<tr>
<td>Apramycin</td>
<td>98.9</td>
</tr>
<tr>
<td>Ceftiofur</td>
<td>96</td>
</tr>
<tr>
<td>Ceftriaxone</td>
<td>97.7</td>
</tr>
<tr>
<td>Cephalothin</td>
<td>92.3</td>
</tr>
<tr>
<td>Chloramphenicol</td>
<td>90.1</td>
</tr>
<tr>
<td>Ciprofloxacin</td>
<td>100</td>
</tr>
<tr>
<td>Gentamicin</td>
<td>90.8</td>
</tr>
<tr>
<td>Kanamycin</td>
<td>87.7</td>
</tr>
<tr>
<td>Nalidixic Acid</td>
<td>98.8</td>
</tr>
<tr>
<td>Streptomycin</td>
<td>69</td>
</tr>
<tr>
<td>Sulfamethoxazole</td>
<td>71.1</td>
</tr>
<tr>
<td>Tetracycline</td>
<td>64.8</td>
</tr>
<tr>
<td>Trimethoprim/sulfa</td>
<td>96.6</td>
</tr>
</tbody>
</table>

In 2004 in the EU, human isolates of the two dominating serotypes *S. Typhimurium* and *S. Enteritidis*, showed a considerable variation in the prevalence of resistant isolates between reporting countries. For *S. Enteritidis* the prevalence of resistant isolates was generally low but for *S. Typhimurium* resistance to commonly used antimicrobials was high in some countries. *S. Typhimurium* strains resistant to 2 or more antimicrobials varied from 7.8 to 56.4%. In the Netherlands 21% of human isolates of *S. Typhimurium* were resistant to more than 4 antimicrobials. In broiler meat the prevalence of resistant isolates of *Salmonella* spp. also showed great variation with a relatively high level of resistance to several antimicrobials reported from some countries. The percentage of strains resistant to 4 or more of the 11 tested antimicrobials varied between 0 and 36% among reporting countries (EFSA, 2005).

### 2.2 Characteristics of the disease in humans

#### 2.2.1 Acute illness

Exposure to *Salmonella* can cause symptoms from mild diarrhoea up to severe sepsis and death. Symptomless carriers are common.

#### 2.2.2 Susceptible populations

Epidemiologic information indicates that susceptibility is highest in infants, elderly people and immuno-compromised hosts (FAO/WHO, 2002).

#### 2.2.3 Dose response

The dose required to cause disease varies with many factors. Low attack rates have been observed in one outbreak where 4-45 cells were consumed, and another where
the dose was 6 cells/65g (Anonymous, 1996a). Different serotypes may have different
dose responses, and doses generally recognised to cause disease at high attack rates
are in the range of $10^5$ to $10^7$ cells. However, these observations simplify a situation
whereby there is no threshold dose for infection. The leading dose-response model
has been produced by the joint risk assessments of *Salmonella* in eggs and broiler

It has been repeatedly reported that the infectious dose is lower when the implicated
food has a high fat or protein content (Lake et al., 2004).

### 2.2.4 Annual incidence rate in humans including differences between
age and sex

Each year, approximately 40,000 *Salmonella* infections are culture-confirmed,
erotyped, and reported to the United States Centers for Disease Control and
Prevention (CDC), which estimates an annual rate of 1.4 million cases, 16,430
hospitalizations, and 582 deaths in the United States alone (Mead et al., 1999). Of
total cases, 95% are estimated to be caused by foods.

International data summarized by Thorns (2000) provides estimated incidences of
salmonellosis per 100,000 people for the year 1997: 14 in the USA, 38 in Australia,
and 73 in Japan.

In the European Union 192,703 cases of salmonellosis were reported in 2004 which
represents an incidence of 42.2 per 100,000 people. Incidence ranged from
6.6/100,000 people in Portugal to 300.9/100,000 in the Czech Republic (EFSA,
2005).

The greatest number of cases of salmonellosis in the EU in 2004 was in children aged
0-4 years. This age group represented 26% of all reported cases. A secondary peak
was reported for adults aged 25-44 years. The number of cases in the age group 65
years and older was the lowest reported of all age groups (EFSA, 2005). It should,
however, be pointed out that association with age may be spurious. Diseased children
are more likely to be given medical attention and are more likely to be tested than
adults. Elderly people with diarrhoea may also be expected to be more frequently
cultured than other age groups (Bananvala et al., 1999). Moreover, age association
may reflect behavioural characteristics. For example, in Norway an association
between eating snow, sand, or soil (most likely a childish behaviour) and infection
with *S. typhimurium* O:4-12 has been demonstrated (Kapperud et al., 1998a). Eating
and cooking habits may also be confounding factors.

The salmonellosis notification rate in Australia for 2002 was 40.3 cases per 100,000
Population. Children less than five years of age had by far the highest notification
rate, with a rate of 210.6 cases per 100,000 people reported for 2002 (Yohannes et al.,
2004). The higher rate of notified salmonellosis cases in this age group may reflect an
increased susceptibility upon first exposure, but may also be a result of other factors
such as an increased likelihood of exposure and increased likelihood to seek medical
care and be tested.

In terms of number of isolates, several studies indicate that men seem to be generally
more affected than are women. A male-to-female ratio of 1.1 has been reported on
various occasions (Blaser and Feldman, 1981; Le Bacq, et al, 1994; Wong et al.,
1994). However several factors may play an important role, such as proportion of the
two genders, as well as different age distributions for males and females within a
country or hospital catchment area. In the evaluation of a single study, it should be pointed out that the occurrence of other factors, e.g. pregnancy or use of antacids, relates to one gender more often or exclusively, and gender may thus have the effect of a confounder. Furthermore, differences in food handling practices and hygiene during food preparation, and amount of food consumed, may also be contributors to any apparent gender differences.

Travel abroad is a risk factor for *Salmonella* gastroenteritis that has been consistently demonstrated in both North America and Europe (FAO/WHO, 2002). For example, domestically acquired salmonellosis only accounts for 15% of human cases in Sweden (SVA, 2006).

### 2.2.4.1 Most common serotypes in humans

The isolation of different serotypes in humans varies geographically. The GSS Country Databank, managed by World Health Organisation Global Salm-Surv, lists the top 15 most commonly identified *Salmonella* serotypes among human and non-human sources in different countries or regions per year ([http://thor.dfvf.dk/portal/page?_pageid=53,1&_dad=portal&_schema=PORTAL](http://thor.dfvf.dk/portal/page?_pageid=53,1&_dad=portal&_schema=PORTAL)). An extract from this database is presented in table 2.2.

Table 2.2. The three most common *Salmonella* serotypes in human isolates in 2004 for different continents. Data from the WHO database Global Salmonella Survey.

<table>
<thead>
<tr>
<th>Continent</th>
<th>Rank 1</th>
<th>Rank 2</th>
<th>Rank 3</th>
<th>Total number of isolates</th>
<th>Average ranking in separate countries</th>
<th>Contributing countries</th>
</tr>
</thead>
<tbody>
<tr>
<td>Africa</td>
<td>Enteritidis (37%)</td>
<td>Anatum (21%)</td>
<td>Typhi (6%)</td>
<td>286</td>
<td>1.0 2.0 3.0</td>
<td>Tunisia</td>
</tr>
<tr>
<td>America</td>
<td>Typhimurium (26%)</td>
<td>Enteritidis (22%)</td>
<td>Newport (11%)</td>
<td>32,019</td>
<td>2.1 2.4 6.1</td>
<td>Argentina, Barbados, Bolivia, Brazil, Canada, Chile, Colombia, Costa Rica, Paraguay, United States of America, Venezuela</td>
</tr>
<tr>
<td>Asia</td>
<td>Enteritidis (28%)</td>
<td>Weltevreden (9%)</td>
<td>Stanley (9%)</td>
<td>5,177</td>
<td>2.3 3.2 6.5</td>
<td>Japan, Rep. of Korea, Malaysia, Oman, Philippines, Thailand</td>
</tr>
<tr>
<td>Europe</td>
<td>Enteritidis (78%)</td>
<td>Typhimurium (12%)</td>
<td>Virchow (2%)</td>
<td>93,651</td>
<td>1.0 2.1 6.0</td>
<td>Austria, Belgium, Bulgaria, Czech Republic, Denmark, Estonia, Finland, France, Greece, Iceland, Ireland, Israel, Italy, Latvia, Luxembourg, Malta, Netherlands, Norway, Portugal, Romania, Serbia and Montenegro, Slovenia, Spain, Sweden, Switzerland, United Kingdom</td>
</tr>
<tr>
<td>Oceania</td>
<td>Typhimurium (56%)</td>
<td>Enteritidis (9%)</td>
<td>Virchow (8%)</td>
<td>6,387</td>
<td>1.0 2.5 4.5</td>
<td>Australia, New Zealand</td>
</tr>
</tbody>
</table>

Of the approximately 2500 known serotypes, about 100 serotypes are annually identified in Finland in infections from abroad, but less than 50 serotypes in domestic
infections. An overall majority of all the salmonellae found belong to *Salmonella enterica* ssp. enterica, which is also the most common subspecies found in production animals. *Salmonellae* of other subspecies normally occur in cold-blooded animals, and these are also annually found in human infections. Of all the *Salmonella* infections detected in 1995 to 2004, about 75% annually were caused by only 10 serotypes and among these only two, *Salmonella Typhimurium* and *Salmonella Enteritidis*, caused most infections both in domestically acquired infections and in infections acquired abroad (FSCP, 2006).

Of the total number of *Salmonella* serovars reported to Australian health authorities during 2002, *S. Typhimurium* 135 was the most commonly reported. Distribution of *Salmonella* serovars varies geographically, with the most commonly reported serovars in Queensland, Tasmania and the Northern Territory being *S. Virchow* (10%), *S. Mississippian* (48%) and *S. Ball* (15%) respectively. Of the other States and Territories, *S. Typhimurium* was the most commonly reported serovar, representing 34% of cases in the Australian Capital Territory, 28% in New South Wales, 60% in South Australia, 66% in Victoria and 15% in Western Australia (FSANZ, 2005).

### 2.2.5 Outcome of exposure

#### 2.2.5.1 Severity of clinical manifestations

Non-typhoidal salmonellosis generally manifests as a self-limiting episode of enterocolitis characterised by diarrhea, abdominal pain, mild fever, chills, nausea and vomiting; prostration, anorexia, headaches and malaise may also occur. The incubation period is 5-72 hours. In general the course of disease is benign and clinical recovery takes place in 2-5 days. Occasionally, systemic infections can occur, particularly with *Salmonella Dublin* and *Salmonella Choleraesuis*, infections which exhibit a predilection toward septicaemia (D'Aoust, 1997).

In the USA it is estimated that in general, 93% of individuals with symptoms of salmonellosis recover fully without visiting a physician, 5% see a physician and recover fully, 1.1% of patients require hospital treatment and 0.04 –0.1% of patients will die (Buzby et al., 1996; Mead et al., 1999). A summary of data of cases with a known outcome from New Zealand in the years 1997-2002 shows a hospitalisation rate of 8.9 – 14.4% and a fatality rate of 0.05 – 0.4% (Lake et al., 2004).

#### 2.2.5.2 Nature and frequency of long-term complications

*Salmonella* has been implicated as a triggering organism for reactive arthritis (ReA) and Reiter's syndrome. Reactive arthritis is characterized by the development of synovitis (joint swelling and tenderness) within a few weeks after the occurrence of gastroenteritic symptoms. An incidence of reactive arthritis of 1.2- 7.3% (mean 3.5%) was estimated in a review of several outbreaks affecting 5525 patients with salmonellosis (Maki-Ikola and Granfors, 1992). Reiter's syndrome is defined as the occurrence of arthritis with one or more extra-articular symptoms typical of the disease such as conjunctivitis, iritis, urethritis and balanitis. The prognosis for ReA is usually favourable with symptoms lasting for <1 year in most persons, although 5 to 18% may have symptoms that last more than 1 year and 15 to 48% may experience multiple episodes of arthritis (FAO/WHO 2002).
### 2.2.6 Availability and nature of treatment

For uncomplicated enterocolitis in an otherwise healthy adult, no specific treatment other than rehydration and electrolyte replacement is usually prescribed. Antibiotic therapy is not routinely recommended (Hohmann, 2001).

### 2.2.7 Percentage of annual cases attributable to foodborne transmission

Although occasionally associated with exposure to pets, reptiles, and contaminated water, non-typhoidal salmonellosis is primarily a foodborne disease. Mead et al. (1999) estimated that 95% of non-typhoidal salmonellosis cases are foodborne in the US.

A study in New Zealand concluded that 63% of annual cases were the result of foodborne exposure (Table 2.3) (NZFSA, 2007).

#### Table 2.3: Summary of the contribution of all exposure pathways to total estimated human salmonellosis in New Zealand

<table>
<thead>
<tr>
<th>Pathway</th>
<th>Cases</th>
<th>% of Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Foodborne</td>
<td>5,670</td>
<td>63.0%</td>
</tr>
<tr>
<td>Animal to person</td>
<td>900</td>
<td>10.0%</td>
</tr>
<tr>
<td>Travel</td>
<td>585</td>
<td>6.5%</td>
</tr>
<tr>
<td>Person to Person</td>
<td>450</td>
<td>5.0%</td>
</tr>
<tr>
<td>Unknown</td>
<td>1,395</td>
<td>15.5%</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>9,000</td>
<td>100.0%</td>
</tr>
</tbody>
</table>

The Danish Zoonoses Centre (DZC) has applied a mathematical model to estimate the contribution of the major animal and food sources to human infections with *Salmonella*. This model is based on a comparison of the number of human cases caused by different *Salmonella* sero- and phage types with the prevalence of the *Salmonella* types isolated from the various animal-food sources. In 2005, the estimated mean number of human cases (per 100,000 inhabitants) that could be attributed to the various foods of animal origin, was as follows: table eggs: 3.9; broilers: 1.3; pork: 4.0; ducks: 0.3; beef: 0.5; imported poultry products: 4.0; imported beef: 1.2; imported pork: 0.8; cases related to outbreaks: 0.5; travel: 7.8; unknown sources: 8.3 (NFI, 2007).

Poultry and poultry products are often implicated in sporadic cases and in outbreaks of human salmonellosis (Bryan and Doyle, 1995; Humphrey, 2000).

In general, eggs, poultry meat and meat products are regarded as the most common food vehicles of salmonellosis to humans (Maijala and Ranta, 2003).

In the Netherlands chickens contributed to 20-22% of the occurrences of human salmonellosis in the period 1994-1998 (KvW and RIVM, 2002). *S.* Enteritidis and *S.* Typhimurium are by far the two dominating serotypes isolated from poultry and poultry products (Poppe, 2000; EFSA, 2004) and these two serotypes are also the most frequently isolated serotypes in humans (Herkstad et al., 2002).

In England and Wales, from 1996 to 2000, salmonellosis caused the most deaths (209) per year of an estimated 687 deaths from indigenous foodborne disease. The most important cause of indigenous foodborne disease was contaminated chicken.
Chicken consumption accounted for more disease, deaths, and healthcare usage than any other food type (Adak et al., 2005).

### 2.3 Epidemiology of foodborne disease

#### 2.3.1 Implicated food and factors that influence transmission

An association with salmonellosis and eating chicken at restaurants and from takeaway shops has been reported in some countries (Table 2.4) (Lake et al., 2004)

<table>
<thead>
<tr>
<th>Country</th>
<th>Risk/protective factors</th>
<th>Odds Ratios/P values</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Australia</td>
<td>(S. Bredeney only) Eating chicken (risk, not the only risk factor identified)</td>
<td>6.0, P=0.01</td>
<td>Baker et al., 1998</td>
</tr>
<tr>
<td>England and Wales</td>
<td>(multi-resistant S. Typhimurium DT104 only) Chicken from local restaurant/takeaway Chicken from local butcher</td>
<td>3.1, CI 1.3-7.6 6.3, CI 2.0-19.9</td>
<td>Wall et al., 1994</td>
</tr>
<tr>
<td>England and Wales</td>
<td>(S. Virchow only) Consumption of any chicken (risk) Consumption of chicken curry from restaurants and takeaways (risk) Other pre-prepared chicken (risk) Halal chicken (risk) Cooked whole chicken (protective)</td>
<td>2.5, CI 1.1-5.8 2.9, CI 1.4-6.1 3.8, CI 1.9-7.6 P=0.015 0.47, CI 0.23-0.95</td>
<td>Willocks et al., 1996</td>
</tr>
<tr>
<td>Norway</td>
<td>Consumption of poultry meat from abroad (risk)</td>
<td>7.6, P=&lt;0.01</td>
<td>Kapperud et al., 1998</td>
</tr>
</tbody>
</table>

#### 2.3.2 Frequency and characteristics of foodborne sporadic cases

There is a lack of knowledge regarding sporadic cases and salmonellosis.

#### 2.3.3 Epidemiological data from outbreak investigations

In the US between 1993 and 1997, there were a total of 655 foodborne disease outbreaks involving 43,821 illnesses, attributable to bacterial pathogens. A total of 357 (54.5%) outbreaks involving 32,610 (74.4%) illnesses were due to Salmonella spp. (Mead et al., 1999).

In New Zealand the annual percentage of outbreaks that was caused by Salmonella varied between 10.0 and 15.0% during the years 1997 to 2002 (Lake et al., 2004).

In outbreaks in Europe between 1993 to 1998 Salmonella spp. were involved in 54.6% of cases. The most important foods where salmonellae caused the outbreak were: eggs and egg products 35%, cakes and ice-cream 28%, meat and meat products 8%, meat and eggs 7%, poultry and poultry products 4%, salads, dressings and mayonnaise 4% (WHO, 2001).
The food vehicles implicated in outbreaks from *Salmonella* spp., in the United States between 1993 and 1997 include eggs, beef, ice cream, chicken and pork (CDC, 2000).

In a review of reported foodborne disease outbreaks in Australia during 1995 – 2000, meats, in particular poultry meat, were associated with 33% of identified salmonellosis outbreaks. Ten out of 75 outbreaks were associated with chicken (FSANZ, 2005).

It can be concluded that non-typhoid *Salmonella* spp. are a major cause of foodborne infections and that amongst these chicken and chicken products are common vehicles in many countries.

### 2.4 Regional and seasonal influences on the incidence of foodborne illness due to the hazard

CDC data (1996) demonstrates that the foodborne disease outbreaks caused by *Salmonella* in the United States occur more frequently in the summer as compared to the winter months. This has also been shown in other parts of the world for example, in the EU, New Zealand and in Australia (Lake *et al*., 2004; EFSA, 2005, Yohannes *et al*., 2004). Temperature may be a major factor impacting the survival and proliferation of *Salmonella*, i.e, warm temperatures provide an environment in which *Salmonella* can grow during the processes of production, transport, and storage (Guthrie, 1992; Latimer, 1999).

### 2.5 Economic impact of the disease

#### 2.5.1 Medical, hospital costs

The actual cost of salmonellosis in various countries is difficult to calculate because of the existence of unreported cases. In USA it is estimated that for every verified human case there are approximately 50 unreported cases (Todd, 1993). In Sweden the number of unreported cases of human salmonellosis is believed to be low (Engvall *et al*., 1993). Estimates in other European countries usually indicate a frequency of unreported cases lying between the US and Swedish estimates.

Todd (1993) estimated the number of human cases of salmonellosis in USA to be 2 million annually. The cost for this was US$ 927 per case adding up to a total of 1.8 billion. In the Netherlands the cost per case in 1992 was estimated to be US$ 1700 for reported cases and US$ 343 for unreported cases (Notermans *et al*., 1992). In Sweden the corresponding costs in 1992 were US$ 2000 and US$ 600 respectively (Engvall *et al*., 1993). In 1998 the costs of foodborne salmonellosis for the United States population were estimated to be US $2,329 million annually for medical care and lost productivity (Frenzen *et al*., 1999).

Sundström (2007) estimated the “true” number of salmonellosis cases in Sweden and the associated costs. The estimated yearly 17,460 cases (based on 4,477 registered cases) would cost 11.7 million USD. Approximately 60% of the total costs could be attributed to production losses when employees are absent due to their own illness.

#### 2.5.2 Working days lost due to illness, etc.

Normally 1-3 working days are lost due to illness.
3 Food Production, processing, distribution and consumption

3.1 Characteristics of the commodity that is involved and that may impact on risk management

The term ‘broiler chicken meat’ principally concerns either whole carcases, or parts of the carcase or boned out meat of the species *Gallus gallus*.

The water activity \((a_w)\) of poultry meat is about 0.98 to 0.99 depending on if, and how long, the meat has been stored in dry air. The pH of chicken breast muscle is 5.7 to 5.9, while that of leg muscle is 6.4 to 6.7. Both poultry muscle and skin are excellent substrates for supporting the growth of a wide variety of microorganisms (ICMSF, 2005).

3.2 Description of the farm to table continuum

The farm to table continuum could be schematically be described as below.
### 3.2.1 Primary production

In the European Union-wide baseline study of *Salmonella* in commercial broiler flock, 2005-2006, the Community observed prevalence of positive flocks was 23.7% while the member-state specific rates varied from 0.0 to 68.2%. A total of 11.0% of the broiler flocks were estimated to be positive for *Salmonella* Enteritidis and/or *Salmonella* Typhimurium. The Member State-specific observed flock prevalence of *S.* Enteritidis and/or *S.* Typhimurium varied from 0% to 39.3% (EFSA, 2007).

**Table 3.1.** Prevalence rates of *Salmonella* in broiler chicken flocks in different countries

<table>
<thead>
<tr>
<th>Country</th>
<th>Prevalence (%)</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>S. spp</td>
<td>S. Enteritidis +S.Typhimurium</td>
</tr>
<tr>
<td>Austria</td>
<td>5.4</td>
<td>1.3</td>
</tr>
<tr>
<td>Belgium</td>
<td>12.4</td>
<td>2.0</td>
</tr>
<tr>
<td>Cyprus</td>
<td>9.1</td>
<td>1.7</td>
</tr>
<tr>
<td>Czech Republic</td>
<td>19.3</td>
<td>9.6</td>
</tr>
<tr>
<td>Denmark</td>
<td>1.6</td>
<td>0.3</td>
</tr>
<tr>
<td>Estonia</td>
<td>2.0</td>
<td>1.7</td>
</tr>
<tr>
<td><strong>EU</strong></td>
<td>23.7</td>
<td>11.0</td>
</tr>
<tr>
<td>Finland</td>
<td>0.1</td>
<td>0.0</td>
</tr>
<tr>
<td>France</td>
<td>6.2</td>
<td>0.5</td>
</tr>
<tr>
<td>Germany</td>
<td>15.0</td>
<td>1.6</td>
</tr>
<tr>
<td>Greece</td>
<td>24.0</td>
<td>3.2</td>
</tr>
<tr>
<td>Hungary</td>
<td>68.2</td>
<td>5.1</td>
</tr>
<tr>
<td>Ireland</td>
<td>27.6</td>
<td>0.0</td>
</tr>
<tr>
<td>Italy</td>
<td>28.3</td>
<td>2.3</td>
</tr>
<tr>
<td>Latvia</td>
<td>6.2</td>
<td>5.1</td>
</tr>
<tr>
<td>Lithuania</td>
<td>2.9</td>
<td>3.3</td>
</tr>
<tr>
<td>Norway</td>
<td>0.1</td>
<td>0.2</td>
</tr>
<tr>
<td>Poland</td>
<td>58.2</td>
<td>32.4</td>
</tr>
<tr>
<td>Portugal</td>
<td>43.5</td>
<td>39.3</td>
</tr>
<tr>
<td>Slovakia</td>
<td>5.7</td>
<td>3.3</td>
</tr>
<tr>
<td>Slovenia</td>
<td>1.6</td>
<td>1.6</td>
</tr>
<tr>
<td>Spain</td>
<td>41.2</td>
<td>28.4</td>
</tr>
<tr>
<td>Sweden</td>
<td>0.0</td>
<td>0.0</td>
</tr>
<tr>
<td>The Netherlands</td>
<td>7.5</td>
<td>1.0</td>
</tr>
<tr>
<td>Thailand</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>United Kingdom</td>
<td>8.2</td>
<td>0.2</td>
</tr>
<tr>
<td>USA</td>
<td>11.4</td>
<td></td>
</tr>
</tbody>
</table>

The primary production is the most important reservoir of *Salmonella* spp. entering the food chain (EFSA, 2004). Due to lack of data the effect of different on-farm interventions could not be evaluated in the FAO/WHO risk assessment (FAO/WHO, 2002). Nevertheless the importance of reducing *Salmonella* infections in the various levels in primary production is obvious.

The efficient control of *Salmonella* spp. in all parent flocks reduces the prevalence at the broiler production stage (SCVPH, 2000). This has been well illustrated in Denmark where a control programme based on top–down eradication, has reduced the proportion of *Salmonella*-infected broiler flocks from >65% in 1989 to <5% in 2000.
(Wegener et al., 2003). This reduction has also been shown to have a reducing effect on human salmonellosis (Hald et al., 2004).

In Finland, Sweden and Norway control programmes have documented that the prevalence of Salmonella spp. in broiler flocks since 1996 has been < 1% (EFSA 2004). The situation in the broiler flocks is reflected in the prevalence in broiler meat (EFSA 2005).

Controlling Salmonella spp. in the primary production relies heavily on biosecurity measures including supply of Salmonella-free feed and water. The use of competitive exclusion and pro- and prebiotics are examples of complementary interventions (Wierup et al., 1992; Fuller, 1989; Bailey et al., 1991).

Due to lack of quantitative data on the efficacy it is impossible to prioritise between different intervention strategies. A combination of different interventions is no doubt beneficial in achieving substantial reductions in the frequency of Salmonella-contaminated broilers sent to slaughter.

The sources of salmonella infection for domestic fowls are numerous. Infection can occur via horizontal transmission by litter, faeces, feed, water, fluff, dust, shavings, straw, insects, equipment and other fomites contaminated with Salmonella and by contact with other chicks or poults, rodents, pets, wild birds, other domestic and wild animals and personnel contaminated with Salmonella (Poppe, 2000).

Contaminated feed is often a significant source of salmonellae on the farm, enhanced by the pathogen’s ability to survive prolonged periods in dry environments. Contaminated feed is often attributed to animal derived ingredients such as rendered animal by-products, and fishmeal. The risk of oral infection has been demonstrated by feeding chicks with contaminated feedstuffs (Williams, 1981). A study by Henken et al. (1992) concluded that farms supplied with contaminated feed were 5.3 times more likely to produce Salmonella-positive flocks than farms supplied with microbiologically safe feed. Also vegetative feed has been found positive for Salmonella, such as soybean products and rapeseed meal.

Rose et al. (1999) reviewed some of the risk factors reported in the literature to be associated with Salmonella contamination in broiler-chickens. They included:

- Poor hygiene and Salmonella contamination in the previous flock (vertical)
- Contaminated chicks (vertical)
- Contaminated feed (horizontal)
- Size of the farm (>3 houses) (horizontal-presumably related to increased human traffic between multiple sheds)
- Summer (as well as wet conditions) (horizontal-greater environmental contamination in summer due to growth and survival of Salmonella)
- Litter beetle infestation

Based on domestic and international data, the major risk factors and their relative importance for Salmonella contamination on-farm are shown in the Table 3.2. Significant variability and uncertainty is associated with the transmission of Salmonella on-farm and the list should not be considered exhaustive nor the importance of each factor absolute (FSANZ, 2005).
Table 3.2 Left to right: Increasing importance

<table>
<thead>
<tr>
<th>Biosecurity</th>
<th>Salmonella</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vertical transmission from breeder flocks</td>
<td>Salmonella</td>
</tr>
<tr>
<td>Positive chicks</td>
<td>Salmonella</td>
</tr>
<tr>
<td>Previously positive flocks</td>
<td>Salmonella</td>
</tr>
<tr>
<td>Litter/Insects</td>
<td>Salmonella</td>
</tr>
<tr>
<td>Contaminated Feed</td>
<td>Salmonella</td>
</tr>
<tr>
<td>Age of birds</td>
<td>Salmonella</td>
</tr>
</tbody>
</table>

Controlling subsurface moisture in grow-out houses is a significant best management practice. It reduces levels of *Salmonella* in the environment and reduces cross contamination within flocks. Drying litter is recommended as a good strategy to lower *Salmonella* on the farms (USDA-FSIS, 2007).

Feed withdrawal is recommended to reduce food and fecal contamination on the carcasses (NCC, 1992). Removing feed too late may result in carcass contamination because the gut may rupture during processing.

Vaccines for specific serotypes (for example *S*. Enteritidis and *S*. Typhimurium) are available. *Salmonella* live vaccines may interfere with bacteriological testing whereas killed vaccines may interfere with serological testing. The use of vaccination depends on the epidemiological situation. Vaccines have very little chance of eradicating *Salmonella* from an infected flock, but may decrease the infectious burden. The positive effects of vaccines against *S*. Enteritidis and *S*. Typhimurium have been demonstrated by, amongst others, Feberwee *et al*. (2000) and Clifton-Hadley *et al*. (2002).

Heuzenroeder *et al*. (2001) undertook a series of challenge studies to determine if *S*. Sofia (low virulence for humans) could act to competitively exclude more pathogenic *Salmonella* serotypes. The results of the research concluded that *S*. Sofia does not exclude the virulent serovar *S*. Typhimurium as both serotypes could co-colonise regardless of initial colonisation status.

3.2.2 Transport of live poultry

Generally, there is a tendency for the numbers of contaminated birds to increase during transport from farm to processing plants (FSANZ, 2005).

The feathers, skin, crop, and cloaca of birds brought to slaughter are often highly contaminated with *Salmonella* (Kotula and Pandya, 1995). Cross-contamination of both birds and cages is frequently made worse when the birds are moved to the plants. There can be a 20-40% increase in *Salmonella* both inside and outside the birds during movement. Moving the birds causes them to pass more fecal material. If the birds have *Salmonella*, the cages have *Salmonella* as well. Transport cages are important sources of cross contamination (Berrang *et al*., 2003; Slader *et al*., 2002; Bailey *et al*., 2001; Corry *et al*., 2002; Humphrey and Allen, 2002). A recent study
found that 5% of the cages sampled were positive for *Salmonella* before use and 10% after use. Additional research showed that the presence of *Salmonella* and *Campylobacter* on birds at receiving was linked to dirty cages (Corry *et al.*, 2002; Slader *et al.*, 2002). Research shows that washing the transport cages with water and leaving them to dry for 48 hours greatly lowers the levels of *Salmonella* found in the cages.

### 3.2.3 Primary processing

Poultry processing does not reduce carcass contamination *per se*, the proportion of contaminated carcasses may even increase during slaughter. Cross-contamination occurs especially at scalding, defeathering, evisceration and giblet operations (Bryan and Doyle, 1995). At present no effective barriers that might control *Salmonella* during processing exist (Fries, 2002) However, differences in hygiene practices between slaughterhouses with resulting differences in carcass contamination have been demonstrated indicating that improved hygiene management could significantly reduce the risk of *Salmonella* contamination of broiler meat (Heyndrickx *et al*., 2002).

Specific strategies to reduce the risk of contaminated poultry meat include slaughter of positive flocks at the end of the week or at the end of the day followed by intensified cleaning and disinfection and channelling of meat from infected flocks to heat-treatment or other bactericidal treatments.

Scalding is an important step that can reduce levels of *Salmonella* on the carcass. Much of the dirt, litter, and feces on carcasses are removed here. One researcher reported a 38% decrease in the number of *Salmonella* positive poultry carcasses post scalding (Geornaras, *et al*., 1997). Chemical decontamination of the carcasses, mainly by using chlorinated water for washing and chilling have been widely used. The effect is a matter of discussion. Some studies have shown a reduction of $1\text{-}2 \log_{10}$ while other have found a reduction of cross-contamination between carcasses but no effect on bacteria entrapped or otherwise attached to skin and muscle surfaces (Lillard 1989; Yang *et al*., 2001). In UK the Food Standards Agency (FSA) concluded that the use of chlorine offers only limited public health benefits (FSA, 2001).

Decontamination with irradiation and ionisation are very effective methods, however, public resistance against these methods has hindered their application in many countries even though scientific experts agree on the safety of these techniques.

During further processing contamination of equipment and workers will occur from positive carcasses and the final cut-up products will be contaminated to a greater extent than the carcasses from which they were prepared (Bryan and Doyle, 1995).

Processing converts live birds into poultry carcasses and poultry meat and in doing so exposes the meat to contamination from the outside of the bird, potentially the intestinal contents of the bird and the processing environment. Processing can be divided into a number of stages. Published studies on the effect of these stages on both the level and prevalence of *Salmonella* on chicken carcasses are often conflicting, indicating a large amount of variability associated with each process. Table 3.3 highlights the typical effect of processing factors on the numbers of *Salmonella* on chicken carcasses. It is recognised that individual plants or companies may perform these tasks differently and to different levels of hygiene.
Table 3.3. Effect on contamination by *Salmonella* (FSANZ 2005)

<table>
<thead>
<tr>
<th>Process stage</th>
<th>Comments</th>
<th>Reduce</th>
<th>Minimal</th>
<th>Increase</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stun/Skill</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Scald – low temperature</td>
<td>Survival of salmonella in scald water- cross contamination</td>
<td></td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>Scald – high temperature</td>
<td>Kill step</td>
<td>✓</td>
<td></td>
<td></td>
</tr>
<tr>
<td>De-feathering</td>
<td>Cross-contamination</td>
<td></td>
<td>✓</td>
<td></td>
</tr>
<tr>
<td>Effective Washing</td>
<td>Physical removal of bacteria</td>
<td>✓</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Evisceration</td>
<td>Contamination with faeces, main source of carcass contamination</td>
<td></td>
<td>✓</td>
<td></td>
</tr>
<tr>
<td>Effective Washing</td>
<td>Physical removal of bacteria</td>
<td>✓</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chilling – immersion suboptimal</td>
<td>Cross-contamination</td>
<td></td>
<td>✓</td>
<td></td>
</tr>
<tr>
<td>Chilling – immersion effective</td>
<td>Requires constant monitoring of water temp., flow rates and chlorine levels</td>
<td></td>
<td>✓</td>
<td></td>
</tr>
<tr>
<td>Chilling – air</td>
<td>Slight reduction due to desiccation of the carcass surface</td>
<td></td>
<td>✓</td>
<td></td>
</tr>
<tr>
<td>Portioning</td>
<td>Possible growth/cross contamination</td>
<td></td>
<td>✓</td>
<td></td>
</tr>
</tbody>
</table>

The levels of *Salmonella* on poultry carcasses fall during processing, although prevalence (ie proportion of contaminated birds) tends to increase, especially after evisceration. Chilling, under effective operation, usually results in a decrease in both numbers and prevalence (FSANZ, 2005).

Methods for carcass chilling include air-chilling, water immersion and spray chilling. Due to the risk of cross contamination in immersion chilling, European countries have generally moved to air chilling of carcasses, whereas in Australia and the US immersion chilling is common (FSANZ, 2005).

Freezing will reduce the populations of *Salmonella*. There is usually a rapid reduction in bacterial numbers on poultry when frozen, followed by a gradual reduction over time (FSANZ, 2005).

In the USA the percent positive of *Salmonella* sets in broiler establishments increased between 2002 and 2005 from 11.5% to 16.3%, but was in 2006 decreased to 11.4%, presumably as a result of the USDA-FSIS’s redirection of the sampling programme and new initiatives to encourage establishments to reassess their food safety systems to achieve and maintain consistent process control (USDA-FSIS, 2007).

Lake *et al.* (2004) compiled studies of the prevalence for *Salmonella* in poultry and raw poultry products from 12 countries worldwide showing a variation from 0 to >50%.
In USA the Pathogen Reduction/HACCP Programme for broiler establishments was launched in 1988. In 2002 this programme could demonstrate a reduction of contaminated ground chicken from a baseline of 44.6% to 14.4% (FDA, 2002).

In northern Thailand, the prevalence of Salmonella in chickens at the slaughterhouse was 9% (Padungtod and Kaneene, 2006).

In New Zealand 1.2% of 1318 samples taken on chicken carcasses after chilling and draining dripping were positive for Salmonella (Lake et al., 2004).

In Vietnam 24 (7.9%) of 302 faecal or intestinal samples from chicken were positive for Salmonella (Tran et al., 2004).

In the EU in 2004 Salmonella was detected in 0.1 – 26.3% in fresh broiler meat taken at processing/cutting plant (10 member states) (EFSA, 2005).

### 3.2.4 Secondary processing, distribution and retail sale of product

A sensitivity analysis of a risk assessment model of salmonellosis in humans inputs indicates that the probability of illness was highly sensitive to the growth of Salmonella during distribution and storage. Improper thawing was also a significant factor (FSANZ, 2005).

D’Aoust (2000) reported the prevalence of Salmonella spp. in retail poultry: chicken 6.9 – 81.5% (13 reports from Denmark, France, Germany, India, Italy, Japan, Malaysia, Mexico, Northern Ireland, Thailand, the Netherlands, Turkey and the UK); minced chicken 42% (1 report from the USA); chicken liver 11.1 – 90.2% (3 reports, Malaysia, Mexico and Thailand) and chicken gizzards 44-88% (2 reports, Malaysia and Thailand).

In a retail survey undertaken in South Australia (2002) on the microbiological quality of chicken fillet, mince and liver Salmonella spp were isolated from 30%, 20% and 59% of samples respectively. When S. Sofia was excluded, however, the isolation rate of Salmonella spp. from chicken fillet, mince and liver was 8%, 10% and 9% respectively (FSANZ, 2005).

In northern Thailand, the prevalence of Salmonella in chicken meat at the market was 57% (Padongtod and Kaneene, 2006).

The prevalence of Salmonella in chickens (262 samples) at retail markets in Hanoi, Vietnam, was 48.9% (Luu et al., 2006).

In the EU in 2004, Salmonella was found in 0.1 – 6.9% in samples of broiler meat products (EFSA, 2005).

In a survey by the UK Food Standards Agency the overall frequency of Salmonella contamination in retail chicken in the UK was 29% (FSA, 2005). In that study Salmonella was isolated from the inside of 11% of the packages.

### 3.2.5 Consumer handling

During normal cooking (roasting, frying, grilling) surfaces of poultry will reach temperatures at which Salmonella are killed. A risk for the consumer to be infected exists when eating undercooked products. In UK (FSA, 2005) undercooking of chicken was observed during consumer preparation (BBQ and stir-fry meals)

Cross-contamination from raw products to cooked products or to ready-to-eat products via contaminated cutting-boards, kitchen utensils, dishcloths, hands etc are
also well known. However, in a study in UK no cross-contamination of *Salmonella* was detected when consumers prepared chicken in the home (FSA, 2005)

Educating and informing the consumer about basic food hygiene and about the correct handling and cooking of broilers are means to reduce the incidence of human salmonellosis caused by contaminated broilers and broiler products.

### 3.3 Summary of current risk management practices

#### 3.3.1 Primary production

The risk assessment of *Salmonella* in broiler chickens (FAO/WHO, 2002) contained limited information concerning the effects of various risk reduction options. It was acknowledged that destruction of *Salmonella* positive flocks will have a public health effect but due to lack of specific information on how this would translate to fewer infected birds or fewer *Salmonella* cells per infected bird at the completion of processing, the magnitude of risk reduction was not estimated. Nevertheless it was estimated that a reduction in the concentration of *Salmonella* cells on carcasses leaving processing would reduce the risk of illness per serving at least proportionally.

Many interventions aimed at reducing the prevalence of infected broilers and broiler products reaching the consumer have been described. In experimental as well as under natural circumstances these interventions have been shown to be more or less effective. Even though there is a lack of quantitative data on the effects of these interventions on the prevalence of *Salmonella* contaminated carcasses and the concentration of *Salmonella* on contaminated broilers under full-scale broiler production, the results from various control programmes show that well focused strategies based on combinations of risk management interventions, implemented along the “stable to table”- continuum, can be very effective.

In many countries *Salmonella* control programmes have been or will be implemented. In the EU, all member states will have to implement control programmes and a Community target for the prevalence of *Salmonella* serotypes with public health significance in broiler flocks will be set according to regulation EC No 2160/2003.

In Finland, Norway and Sweden national *Salmonella* control programmes have been effectuated for many years. These programmes include all steps from breeder production to final processing and are based on a zero-tolerance strategy including all *Salmonella* serotypes. Whenever *Salmonella* is found immediate actions are taken.

The prevalence of *Salmonella* contaminated flocks in these countries has consistently been very low for the last ten years and positive samples found after slaughter and in cutting plants have been very few, if any (e.g. EFSA, 2005). The effects of these programmes on public health have been elucidated and found to be very good (Engvall *et al*., 1993; Maijala *et al*., 2005).

The Danish National *Salmonella* Control Programme for the production of table eggs and broilers was launched in December 1996. The programme was designed to be a ‘top-down’ control effort based on an elimination strategy, whereby the infected flocks were eradicated by means of compulsory destruction or slaughter. The percentage of positive broiler-production flocks at ante mortem (AM) inspection has declined from 12.9% in 1997 to 1.5% in 2002 (NFI, 2007).
3.3.2 Processing

In USA the Pathogen Reduction/HACCP Programme for broiler establishments was launched in 1988. In 2002 this programme could demonstrate a reduction of contaminated broilers from a baseline of 20% to 10.2% and from a baseline of 44.6% in ground chicken to 14.4% (FDA, 2002).

3.3.3 Food service and the home

Consumer education campaigns exist in many countries, however little information is available regarding their efficiency.

4 Other Risk Profile Elements

4.1 The extent of international trade of the food commodity

In 2005 approximately 71 million tonnes of chicken meat were produced globally (see http://faostat.fao.org/site/343/DesktopDefault.aspx?PageID=343). Out of this, 11.1 million tonnes were exported. This compares with 12.2 tonnes of beef and 11.6 tonnes of pig meat. The ten leading exporters of chicken meat were Brazil (26%), the USA (24%), the Netherlands (9%), China (8%), France (7%), Belgium (4%), United Kingdom (3%), Germany (3%), Thailand (3%) and Poland (2%).

4.2 Existence of regional/international trade agreements and how they may affect the public health impact with respect to the specific hazard/commodity combination(s)

Many countries have trade restrictions for Salmonella and poultry trade between countries have been interrupted by Salmonella contaminated consignments (Mathews et al., 2003).

4.3 Public perceptions of the problem and the risk

Consumers in Sweden have in three different surveys performed during the last seven years repeatedly identified Salmonella-free products as the most important quality aspect of broiler chicken meat (Ståhle, 2007).

Based on a questionnaire sent to 2000 people in Finland (response rate 55%) consumers are prepared to pay 5.8 euros per household per month for running the Finnish Salmonella Control Programme (Peltola et al., 2001).

5 Available Information and Major Knowledge Gaps

5.1 Existing national MRAs on the hazard/commodity combination

A Quantitative Risk Assessment on Salmonella in Broiler Production in Finland, Riitta Maijala & Jukka Ranta, National Veterinary and Food Research Institute, EELA, Finland.

Food standards Australia New Zealand, Scientific Assessment of the Public Health and Safety of Poultry Meat in Australia.

5.2 **Areas where major absences of information exist that could hamper MRM activities including, if warranted, the conduct of an MRA**

The risk assessment of *Salmonella* in broiler chickens (FAO/WHO 2002) contained limited information concerning the effects of various risk reduction options. The expert group found the available data on the importance of various routes for introduction of *Salmonella* spp. into flocks to be inconclusive. It was therefore not possible to evaluate the importance of on-farm routes of introduction of *Salmonella* spp.

The main data gaps for primary production are:

- *Salmonella* prevalence information is available for some countries worldwide, however many of these studies give limited details of study design.
- Data are limited or missing from most countries in Africa, Asia and Latin America.
- The effect on *Salmonella* prevalence of specific risk reduction interventions.
- Data on the impact of on-farm factors on the prevalence of contaminated flocks and/or birds.
- Identification of contamination sources for *Salmonella* during primary production.
- Data on the prevalence and, in particular, levels of *Salmonella* on birds/carcasses at all stages of the exposure pathway.

The main data gaps for processing are:

- Quantitative data are limited for several steps of processing.
- There is limited information on processing practices used in different countries.
- Data on cross-contamination between birds pre-harvest, and during transport and processing.
- Data on the magnitude of cross-contamination and improper cooking of poultry meat in different countries.
- Many studies are old, more recent information on changes in prevalence and numbers would be beneficial.

6 **References**


FSA (Food Standards Agency, UK). (2001) Achieving the agency’s Salmonella in chicken target. Paper FSA 01/03/02b.


