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# Risk Profile: Salmonella (non-typhoidal) in Poultry (whole and pieces)

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Prepared for the Ministry for Primary Industries
by Nicola King, Dr Rob Lake, Peter Cressey

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# *SALMONELLA* (NON-TYPHOIDAL)
# IN POULTRY (WHOLE AND PIECES)

Prepared for the Ministry of Agriculture and Forestry
under project MRP/10/01, Microbiological Risk Profiles,
as part of overall contract for scientific services

Client report FW11044

by

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November 2011

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## CONTENTS

## LIST OF TABLES

## LIST OF FIGURES

## SUMMARY

The purpose of a Risk Profile is to provide information relevant to a food/hazard combination so that risk managers can make decisions and, if necessary, take further action. A Risk Profile includes elements of a qualitative risk assessment, as well as providing information relevant to risk management.

This Risk Profile concerns *Salmonella* species in poultry (chicken, turkey and duck) and poultry products. This is an update of a Risk Profile published in 2004.

This Risk Profile has been commissioned in order to address the following specific risk management questions:

- What is the public health risk from *Salmonella* in poultry (whole and portions) consumed in New Zealand?
- Has the risk of salmonellosis from consumption of poultry (whole and portions) changed since the 2004 Risk Profile?

The incidence of notified cases of salmonellosis has declined since a peak of 65 per 100,000 population in 2001, and has been stable in New Zealand since 2005 at 25-35 reported cases per 100,000 population. This rate is close to that in other developed countries, particularly those in Europe, and lower than in Australia. Throughout the 1980s and 1990s the rate fluctuated between 37 and 57 per 100,000 population, with no apparent trend.

National Microbiological Database (NMD) sampling of poultry for *Salmonella* only commenced in 2001, so it is not possible to consider trends before that year. The Poultry Industry Association of New Zealand (PIANZ) reported the prevalence of *Salmonella* on poultry carcasses during the 1990s as 17%. This prevalence figure appears to come from a retail survey of 137 unfrozen poultry samples.

NMD data represent approximately 1,800-2,000 carcass rinse samples per annum, taken at the end of primary processing. The previous Risk Profile reported that data received from PIANZ indicated the prevalence found by NMD testing was 1-2% for the period 2001 to 2003. New NMD data presented in this Risk Profile, covering 2005 to 2010, shows the prevalence declining from 3.5% to 0.2%.

The temporal pattern of a steady and considerable decline in prevalence of *Salmonella* in poultry samples from the 1990s to 2010 is different to the pattern of the incidence of notified salmonellosis cases, and suggests that they are not strongly linked.

There have been incidents of temporary increases in the numbers of salmonellosis cases or outbreaks involving particular serotypes in New Zealand. The incidence of the five serotypes causing the greatest number of cases from 2000 through 2009 (*S.* Typhimurium DT160, *S.* Typhimurium DT1, *S.* Brandenburg, *S.* Typhimurium DT135 and *S.* Typhimurium DT156) all peaked during 2000 through 2002. While these serotypes are still isolated frequently from salmonellosis cases (*S.* Typhimurium DT160 is still the most commonly isolated serotype), a variety of other serotypes have peaked in recent years, such as *S.* Infantis, *S.* Mbandaka and *S.* Stanley. The initial outbreaks of infection by some of these serotypes, such as *S.* Typhimurium DT160 and *S.* Brandenberg, were associated with animal contact, but the cause

of the fluctuating incidence of other serotypes, such as *S.* Infantis, is not known. These suggest that *Salmonella* contamination of transmission vehicles is sporadic and would be difficult to detect through routine monitoring of foods or other sources of infection.

Some outbreak investigations identify poultry as the probable cause of salmonellosis, but poultry and poultry products have not been demonstrated conclusively to be a vehicle in outbreaks or case-control studies. A temporary increase in the prevalence of *S.* Typhimurium DT1 in poultry in 2003 in Canterbury occurred at the same time as an increase in reported salmonellosis in that region. While poultry meat was identified as the likely source in a 2008 New Zealand *S.* Mbandaka outbreak, case interviews were equivocal and *S.* Mbandaka was not detected in any foods obtained from cases. A review of 204 salmonellosis outbreaks from 2000-2009 identified only one outbreak with strong evidence of a potential link to poultry, but in this outbreak the causative serotype (*S.* Thompson) was isolated from a mixed food containing chicken as an ingredient.

Despite a lack of robust epidemiological association, many foods including poultry might still be vehicles for infection for non-attributable small clusters and sporadic cases of salmonellosis. The NMD data indicate a very low prevalence of contamination in poultry carcasses at the end of primary processing, by international standards. This is consistent with the most recent retail surveys which also reported consistently low prevalences of salmonellae on poultry at retail (e.g. not detected on 163 broiler carcasses sampled in 2007, detected in 7/232 samples of minced or chopped raw chicken, 2003-2005). The low prevalence of *Salmonella* in New Zealand poultry suggests that, although poultry is a frequently consumed food by the New Zealand population, exposure to *Salmonella* will be infrequent. This appears to be at variance with the results of a modelling exercise, which attributed 21% of salmonellosis cases to poultry, and a review of scientific evidence that concluded that poultry was "very likely" (>90% probability) to be at least a moderate cause (between 10-30% or higher of all cases) of salmonellosis.

Conventional cooking (>60°C) would normally be expected to rapidly inactivate *Salmonella* in food (D value less than 2 minutes at 65°C and less than 30 seconds at 70°C). Therefore, thorough cooking of poultry will eliminate any *Salmonella* that might be present. This is supported by the results from overseas surveys of ready-to-eat chicken products in developed countries, which have been cooked by producers prior to reaching the public. The prevalences found in these surveys were <1%.

The low risk from this food/hazard combination, as assessed by the 2004 Risk Profile, does not appear to have changed. On the basis of the reduced prevalence in *Salmonella* found on poultry carcasses by the NMD testing programme from 2005 to 2010, it could be argued that the risk has declined.

The 2004 Risk Profile did not specifically identify data gaps, but since 2004 a number of surveys have been completed that provided new data on the prevalence of *Salmonella* on poultry from processing plants and retail outlets and on packaging. The data gaps identified in this Risk Profile are:

- Representative sampling and testing for *Salmonella* in broiler farm inputs (feed) and environment;

- Information on the impact of current processing practices in New Zealand on *Salmonella* prevalence and concentrations on poultry;
- Information on the concentration of salmonellae on poultry carcasses at the end of primary processing; and
- Transmission routes for the majority of salmonellosis cases in New Zealand.

# 1 STATEMENT OF PURPOSE

The purpose of a Risk Profile is to provide information relevant to a food/hazard combination so that risk managers can make decisions and, if necessary, take further action. Risk Profiles are part of the Risk Management Framework (RMF) approach taken by the Ministry of Agriculture and Forestry (MAF).[1] The Framework consists of a four step process, as shown in Figure 1.

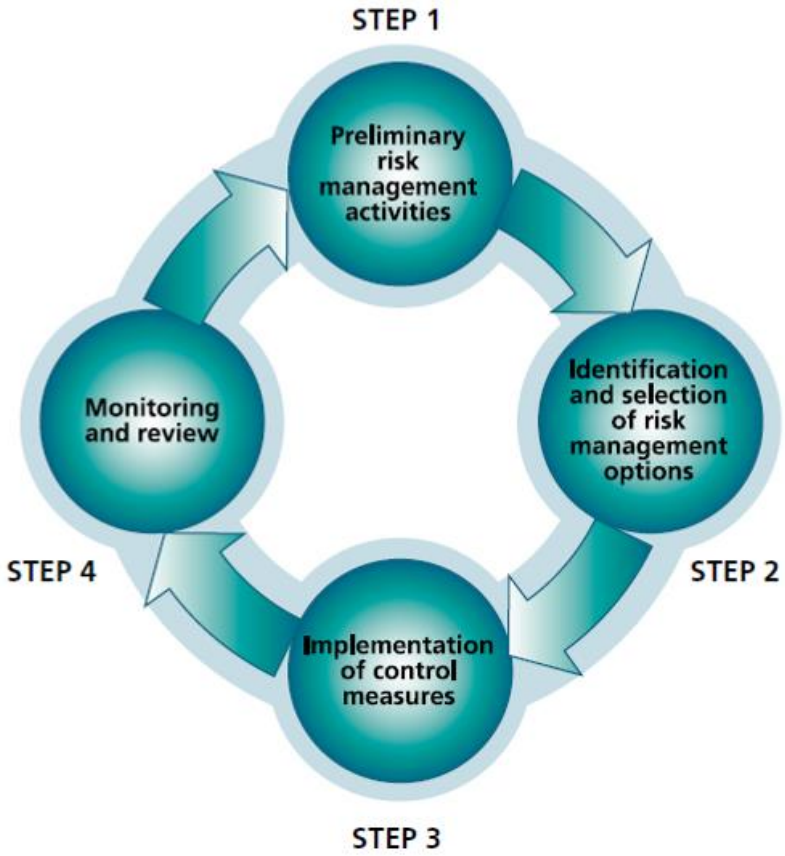



**Figure 1: The four steps of the Risk Management Framework**

This initial step in the RMF, Preliminary Risk Management Activities, includes a number of tasks:

- Identification of food safety issues
- Risk profiling
- Establishing broad risk management goals
- Deciding on the need for a risk assessment
- If needed, setting risk assessment policy and commissioning of the risk assessment
- Considering the results of the risk assessment

[1] http://www.foodsafety.govt.nz/elibrary/industry/RMF_full_document_-_11604_NZFSA_Risk_Management_Framework_3.1.pdf

- Ranking and prioritisation of the food safety issue for risk management action.

Risk profiling may be used directly by risk managers to guide identification and selection of risk management options, for example where:

- Rapid action is needed;
- There is sufficient scientific information for action;
- Embarking on a risk assessment is impractical.

## 1.1 Food/hazard Combination and Risk Management Questions

The food/hazard combination addressed by this Risk Profile is *Salmonella* (non-typhoidal) in poultry (whole and portions). This is an update of a Risk Profile published in 2004 (Lake *et al.*, 2004).

This Risk Profile has been commissioned in order to address the following specific risk management questions:

- What is the public health risk from *Salmonella* in poultry (whole and portions) consumed in New Zealand?
- Is the risk of salmonellosis from consumption of poultry (whole and portions) likely to have changed since the 2004 Risk Profile?

## 1.2 MAF Risk Management Strategy

In March 2010, MAF (then the New Zealand Food Safety Authority; NZFSA) released their *Salmonella* Risk Management Strategy 2009-2012. The Strategy aims to achieve a 30% reduction in the reported annual incidence of foodborne salmonellosis after five years. The strategy focuses on non-typhoid *Salmonella* and begins with a primary focus on intelligence gathering from a wide range of food sectors.

The objectives of the *Salmonella* risk management strategy are to:

- Quantify the proportion of foodborne cases attributable to:
  - specific foods
  - animal feeds
  - domestically produced versus imported foods
  - multi-resistant and virulent *Salmonella* genotypes associated with foods
- Identify sources of *Salmonella* contamination of specific foods and animal feeds
- Determine the relative value of different interventions throughout the food chain in reducing the risk of salmonellosis
- Make prioritised risk management decisions on appropriate *Salmonella* control measures across the food chain, and according to data availability
- Design and implement an effective monitoring and review programme to support strategic goals.

An updated version of the strategy was published in 2010 that covers 2010-2013.[2] This version records NZFSA's progress towards achieving the objectives. Those relevant to this Risk Profile are recorded in Table 1.

**Table 1: Poultry-specific outputs or results from the NZFSA *Salmonella* Risk Management Strategy**

| Work programme | Poultry-specific outputs/results | Ref* | See also (in this report) |
|---|---|---|---|
| Systematic review of the epidemiological evidence available within New Zealand of the aetiology of human *Salmonella* infection (completed) | Poultry is "very likely" (>90% probability) to be at least a moderate cause of salmonellosis (i.e. between 10-30% or higher of all cases). | 1 | Section 3.3.5.2 |
| Attribution of potentially foodborne enteric diseases: human salmonellosis. Enhanced surveillance including outbreaks (completed) | Poultry was commonly implicated as the vehicle of infection in sporadic cases and outbreaks between 2000 and 2009 but there was insufficient epidemiological or laboratory evidence to attribute salmonellosis cases to specific foods. | 2 | Section 3.3.5.2 |
| Code of Practice for Poultry Processors chapters: secondary processing, cleaning and sanitation (completed) | Additional chapters for the Code of Practice have been published that detail good manufacturing practice for secondary processing (April 2009), hygiene, cleaning and sanitation during poultry processing (October 2009), and repairs and maintenance for buildings, facilities and equipment related to the processing of poultry (August 2010). The Code of Practice applies to operators who are processing poultry products for human consumption (and animal consumption, where produced on the same premises). There are no specific requirements for *Salmonella* control. | 3 | Section 5.1.3.3 |
| Updated guidance material on safe handling of poultry meat and relevant time temperature applications (ongoing) | N/A | N/A | N/A |
| Voluntary audit of broiler grower farms (completed) | N/A | N/A | N/A |
| Compliance audit of poultry primary processors (completed) | N/A | N/A | N/A |
| Compliance audit of poultry primary processors to assess the application and effectiveness of the Poultry Processors Code of Practice | N/A | N/A | N/A |

[2] Available at http://www.foodsafety.govt.nz/industry/general/foodborne-illness/salmonella/strategy.htm (accessed 12 May 2011).

| Work programme | Poultry-specific outputs/results | Ref* | See also (in this report) |
|---|---|---|---|
| – new chapters 2009 plus NMD requirements (ongoing) | | | |
| NMD monitoring of poultry (ongoing) | The prevalence of *Salmonella* species on whole poultry carcasses was 0.2% in 2010, based on 1,876 samples. | N/A | Section 7.4.1 |
| Review spikes in NMD *Salmonella* results (ongoing) | N/A | N/A | N/A |

* References:

1. (Wilson and Baker, 2009)
2. (Adlam *et al.*, 2010; King *et al.*, 2011)
3. Available at http://www.foodsafety.govt.nz/elibrary/industry/processing-code-practice-poultry/index.htm (accessed March 2011).

N/A = not applicable

NMD = National Microbiological Database

# 2 HAZARD AND FOOD

## 2.1 *Salmonella* species

This group of bacteria is comprised of two species: *Salmonella enterica*, which is divided into six subspecies (*enterica, salamae, arizonae, diarizonae, houtenae* and *indica*), and *Salmonella bongori* (Grimont and Weill, 2007). Most pathogenic isolates from humans and other mammals belong to *S. enterica* subspecies *enterica*. Other *S. enterica* subspecies and *S. bongori* are more common in cold blooded animals and the environment, and are of lower pathogenicity to humans and livestock (Brenner *et al.*, 2000; Jay *et al.*, 2003).

*Salmonella* are primarily divided into types using serological identification of somatic (O), flagella (H), and capsular (K) antigens. There are more than 2,500 different *Salmonella* serotypes (also called serovars), and of these over 1,500 have been identified in the *S. enterica* subspecies *enterica* group (Grimont and Weill, 2007).

*S. enterica* subspecies *enterica* serotypes are given serotype names (Jay *et al.*, 2003). The full name and serotype name are normally abbreviated to a shortened form, where the serotype is capitalised and non-italicised, e.g. *Salmonella enterica* subsp. *enterica* serotype Enteritidis becomes *Salmonella* Enteritidis (or *S.* Enteritidis). In older publications this may be represented as a species name i.e. *Salmonella enteritidis*. The serotypes of other *S. enterica* subspecies and *S. bongori* are identified by their serotyping formula and are not given names (Grimont and Weill, 2007).

*Salmonella* species can be further subtyped by measuring susceptibility to a panel of bacteriophages. These types are denoted as provisional phage type (PT) or definitive phage type (DT) numbers. These two terms exist from the original two-step phage typing process between the 1950s and 1970s where a strain was originally given a PT number and later confirmed with a DT number. After the 1970s the methods were reasonably well established so the prefix PT was no longer required (Anderson *et al.*, 1977; Bell and Kyriakides, 2002). Both terms are still used in the literature.

Molecular methods are also used for *Salmonella* species typing in New Zealand, usually for salmonellosis outbreak or cluster investigations, and antimicrobial susceptibility is monitored. Further information on these methods, plus additional detail on serotyping and phage typing, is included in Appendix 1.

*Salmonella* Typhi and *Salmonella* Paratyphi are serotypes which cause serious enteric fever and are particularly well adapted to invasion and survival in human tissue. They have a particular antigen makeup and differing ecology to other serotypes of *Salmonella*. *Salmonella* Choleraesuis (SCS) is a typhi-like serotype that infects pigs. SCS is only found in a few countries, excluding New Zealand, and has a distinct pathogenic profile. This Risk Profile does not consider these human and porcine typhoidal serotypes.

## 2.2 Sources of *Salmonella* species

The information in this section represents a summary of a microbiological data sheet relevant to this Risk Profile. These data sheets are prepared by ESR for a number of different

foodborne pathogens as requested by MAF.[3] Additional information on the hazard and food is included in Appendix 1.

The primary sources of *Salmonella* are the gastrointestinal tracts of humans and animals and its widespread presence in the environment can be considered to be due to direct or indirect faecal contamination (Bell and Kyriakides, 2002).

Human: Person-to-person transmission of *Salmonella* is well recognised, and secondary transmission of *Salmonella* in outbreaks has been demonstrated (Loewenstein, 1975). Carriage in faeces in convalescent cases can be quite substantial with numbers approximating $10^6$-$10^7$ salmonellae/g persisting up to 10 days after initial diagnosis. Reduction in numbers with time is variable; most people will have counts of less than 100 salmonellae/g after 35 to 40 days, but a count of 6 x $10^3$/g has been recorded in one patient 48 days post-illness (Pether and Scott, 1982). In New Zealand, other gastrointestinal diseases such as cryptosporidiosis, giardiasis and shigellosis are more strongly associated with person-to-person transmission than salmonellosis, but person-to-person risk factors are commonly cited in outbreak reports (Adlam *et al.*, 2010). Asymptomatic carriage may also occur, and asymptomatic foodhandlers have been responsible for a British outbreak of hospital-acquired infection (Dryden, 1994), as well as an outbreak in a catering establishment in Jerusalem (Stein-Zamir *et al.*, 2009).

Animal: *Salmonella* can be found in mammals, fish, reptiles, amphibians, insects and birds. Most *Salmonella* colonisations in animals do not produce clinical signs. Some serotypes are largely confined to particular animal reservoirs causing both systemic and enteric disease, for example *S.* Choleraesuis is host restricted to pigs (Allison *et al.*, 1969) while other serotypes (for example *S.* Typhimurium) are frequently associated with intestinal infections in a wide range of phylogenetically unrelated species (Paulin *et al.*, 2002). Both plant and animal product-based animal feed ingredients may be contaminated with salmonellae. The *Salmonella* serotypes Brandenburg and Typhimurium DT9 are often associated with sporadic salmonellosis cases who have had contact with colonised animals in New Zealand (Adlam *et al.*, 2010).

Food: Red and white meats, meat products, milk, cheese and eggs are considered the major food sources of human salmonellosis, although a wide variety of other foods have been associated with outbreaks (Jay *et al.*, 2003). Other foods that have been contaminated by *Salmonella* include seafood (shellfish, salmon), nuts and nut products (desiccated coconut, peanut butter), cereal and cereal products (barley, cereal powder), spices (white and black pepper, paprika), oilseeds and oilseed products (cottonseed, soybean sauce, sesame seeds), vegetables (watercress, tomatoes, lettuce, potato and other salads, bean sprouts), fruit and fruit products (watermelon, melon, cider) and other miscellaneous products (chocolate, cocoa powder, dried yeast, candy). *Salmonella* contaminated tahini (a product made from crushed sesame seeds) has caused a number of outbreaks worldwide, including New Zealand and Australia (Unicomb *et al.*, 2005).

Environment: Salmonellae in sewage effluents or animal faeces can contaminate pasture, soil

[3] A full set of the data sheets can be found at: http://www.foodsafety.govt.nz/science-risk/hazard-data-sheets/pathogen-data-sheets.htm (accessed 12 May 2011).

and water. They do not usually multiply in soil and waters but may survive for long periods (Bell and Kyriakides, 2002). The organism may also be dispersed in dust and aerosols generated during the handling and processing of animals. Contamination in the environment can be spread by rodents or wild bird populations and act as a source of infection for other animals.

Transmission routes: Salmonellae may be transmitted to humans via person-to-person transmission, contaminated food or water, animal contact or from a contaminated environment. A review of non-typhoidal salmonellosis sporadic cases and outbreaks in New Zealand indicated that the important pathways for *Salmonella* infection are consumption of contaminated food, consumption of untreated drinking water and contact with sick animals (Adlam *et al.*, 2010).

## 2.3 The Food

### 2.3.1 Definitions

The specific foods considered by this Risk Profile are poultry and poultry products. Poultry includes chickens (*Gallus gallus*), turkeys and ducks that are commercially produced (i.e. not harvested for personal consumption).

Poultry products include:

- Whole poultry and poultry pieces/portions (such as wings, drumsticks, breasts), raw or cooked;
- Raw value-added poultry products, such as marinated or crumbed portions, stuffed whole birds, rolled breasts, frozen nuggets, sausages;
- Packaged ready-to-eat poultry products, such as cooked slices, smoked products;
- Ready-to-eat poultry products served by the food service industry.

The term "broiler" is often used for a chicken that is bred specifically for meat production (a "layer" has been bred specifically for egg production).

This Risk Profile excludes other types of poultry such as goose, pigeon and ostrich.

### 2.3.2 The Food Supply in New Zealand: Poultry and poultry products

The Poultry Industry Association of New Zealand Incorporated (PIANZ) represents the interests of poultry processing and breeding companies in New Zealand and has a role in developing poultry standards. Membership is voluntary, but the following 11 producers of almost all of this country's poultry meat choose to be represented by PIANZ.[4]

- Tegel Foods Ltd.;
- Inghams Enterprises (NZ) Pty Ltd.;
- PH van den Brink Ltd.;
- Turk's Poultry;
- A & J Heron Holdings Ltd.;

[4] As listed at http://www.pianz.org.nz (accessed 12 May 2011).

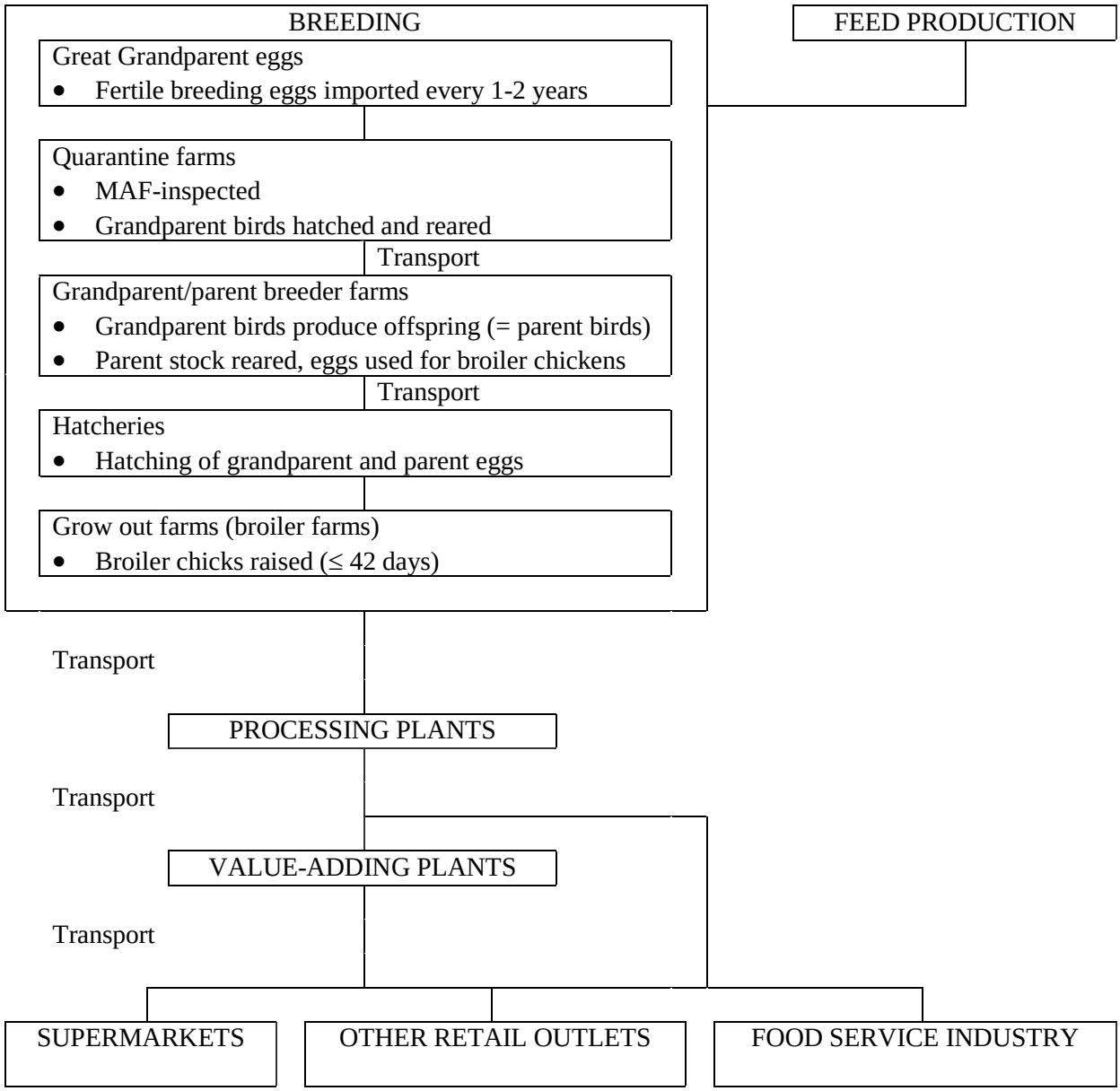
- Aviagen;
- Bromley Park Hatcheries;
- Canter Valley Processors;
- Crozier's Turkeys Ltd.;
- Eastherbrook Farm Ltd.; and
- Quack a duck.

Larger companies (such as Tegel Foods, Inghams Enterprises) are vertically integrated and manage all aspects of poultry meat production within their separate companies from feed production to breeding, processing and value-adding.

Tegel Foods Ltd., Inghams Enterprises (NZ) Pty Ltd. and PH van den Brink Ltd. together have the greatest market share in New Zealand. There are also a number of small or niche poultry producers who are not members of PIANZ (e.g. Heuvels, Mahurangi Ducklings).

Figure 2 outlines the product flow within the poultry industry in New Zealand.

**Figure 2: Generic flow of product within the poultry industry in New Zealand (Lake *et al.*, 2005)**




### *2.3.2.1 Production*

Data from PIANZ indicates that there were approximately 150,000 tonnes of poultry meat produced in 2010 from approximately 85 million birds.[5] Broiler chicken meat production has been steady at between 140,000 and 160,000 tonnes per year since 2003.

The majority of broilers produced in New Zealand (98%) are barn raised (Lake *et al.*, 2005). An overview of broiler farming in New Zealand assembled in 2006 using information from the four largest companies found that there were 130 farms with approximately 500 sheds

[5] See http://www.pianz.org.nz/industry-information/industry-statistics (accessed 25 June 2011).

(Hudson *et al.*, 2008). Most sheds held less than 50,000 birds (shed size 30,000 to 45,000), although individual farms may hold more than 200,000 birds per growing cycle.

A Risk Profile published in 2006 reported that approximately 40% of poultry was sold as whole carcasses (Lake *et al.*, 2006). In the year ending June 2006, approximately 98% of poultry consumption was chicken meat, with turkey, duck, and roasting fowl making up the remaining 2%. Most production (65%) was purchased and consumed by domestic households, while the remaining 35% entered the food service industry (including fast food outlets). Approximately 79% of chicken was sold as fresh chilled product and 21% frozen.

#### *2.3.2.2 New Zealand exports*

New Zealand exports only a small proportion of poultry production. The approximately 4,000 tonnes of chicken meat exported in the year ending March 2011 represents 2.7% of the approximately 150,000 tonnes total broiler chicken meat production for that period. Appendix 1 provides further details on exports.

#### *2.3.2.3 New Zealand imports*

Raw chicken is currently not permitted for import into New Zealand. There are import health standards in place for:

- Importing specified cooked poultry meat products for human consumption from Australia;[6] and
- Importing turkey meat and meat products from approved countries.[7]

These standards require the poultry products to be cooked, although raw turkey products may be imported if the importer can demonstrate disease-free status.

According to data released by Statistics New Zealand, in year ending March 2011, the three largest imported poultry products by weight were:

- Chicken preparations preserved in airtight containers or jars (not meat pastes or combined with vegetables or other substances): 367 tonnes;
- Poultry preparations preserved in airtight cans or jars (not turkey, livers or homogenised preparations and prepared without other food substances): 186 tonnes; and
- Chicken preparations preserved in airtight containers or jars (in combination with vegetables or other food substances) or meat pastes: 168 tonnes.

Other imported poultry products included sausages and liver products.

Thailand is the major source of imported poultry products, followed by the USA and Australia.

[6] http://www.biosecurity.govt.nz/files/ihs/meapouic.aus.pdf (accessed 25 June 2011).
[7] http://www.biosecurity.govt.nz/imports/animals/standards/pouturic.gen.htm (accessed 25 June 2011).

## 2.4 Behaviour of *Salmonella* species in Poultry and Poultry Products

The following information is a summary of activities that influence the introduction, growth or elimination of *Salmonella* species for poultry and poultry products. Appendix 1 contains additional information. Figure 3 shows a generic flow diagram of the main steps from poultry production to the consumer. There are many opportunities for salmonellae to enter this food chain, although other steps will prevent growth or inactivate the pathogen.

### 2.4.1 Poultry farming (primary production)

Risk factors reported in the literature to be associated with *Salmonella* contamination in broiler chickens have been reviewed in a retrospective study (Rose *et al.*, 1999). Important risk factors included:

- Contaminated chicks;
- Size of the farm (>3 poultry sheds – presumably related to increased human traffic between multiple sheds);
- Contaminated feed (the risk of *Salmonella* contamination of the flock was increased when feed trucks were parked near the entrance of the worker change room and when feed meal, instead of small pellets, was provided to day old chicks);
- Poor hygiene in the poultry house and *Salmonella* contamination in the previous flock;
- Summer (as well as wet conditions – greater environmental contamination in summer due to growth and survival of *Salmonella*); and
- Litter beetle infestation.

Transovarian transmission, a form of vertical transmission from parent to chick, is currently not of concern in New Zealand because the *S.* Enteritidis phage types (particularly PT4) able to infect egg contents are not endemic in New Zealand and have not been found in eggs (King *et al.*, 2011).

In a survey conducted in 2006, it was found that approximately 57% of broiler farming operations in New Zealand involved 3 or more sheds (Hudson *et al.*, 2008).

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. The contamination and survival of *Salmonella* in poultry feed, and its transmission to poultry, have been described in another Risk Profile concerning *Salmonella* species in animal feed (Cressey *et al.*, 2011). Application of Hazard Analysis Critical Control Point (HACCP) principles, including good manufacturing practices and general hygiene procedures are recognised as important measures for *Salmonella* control in feed production. Such measures are either in place or under development in the New Zealand feed industry, although it is uncertain what level of application of these principles is achieved outside the membership of the New Zealand Feed Manufacturers' Association. The members of the association are responsible for the production of more than 85% of the animal feed produced in New Zealand. It is recognised that the application of HACCP principles should ideally extend to rendering and crushing plants supplying ingredients to the animal feed industry.

**Figure 3: Process flow diagram for poultry: Primary production to the consumer***

| Stage | Step | Activity | | |
|---|---|---|---|---|
| Primary production | 1 | Manage grandparent flocks | | |
| | 2 | Transport eggs to hatchery | | |
| | 3 | Parent Hatchery | | |
| | 4 | Transport day-old chicks to parent farms | | |
| | 5 | Manage parent flocks | | |
| | 6 | Transport eggs to hatchery | | |
| | 7 | Hatchery | | |
| | 8 | Transport day-old chicks to grower sheds | | |
| | 9 | Manage chickens (broiler farm) | | |
| | 10 | Depopulate (full or partial) | | |
| | 11 | Transport live birds to slaughterhouse | | |
| Primary processing | 12 | Receive at slaughterhouse | | |
| | 13 | Ante-mortem inspection | | |
| | 14 | Slaughter | A. Hang<br>B. Electrical stun | A. Gas stun<br>B. Hang |
| | | | C. Neck cutting<br>D. Bleed Out | |
| | 15 | Dress<br>(may include wash steps) | A. Scalding<br>B. Defeathering<br>C. Head-pulling<br>D. Hock-cutting<br>E. Re-hanging (optional)<br>F. Venting<br>G. Evisceration<br>H. Crop removal<br>I. Neck-cracking/cutting of neck flap | |
| | 16 | Inside/outside wash | | |
| | 17 | On-line reprocessing | | |
| | 18 | Post-mortem inspection | | |
| | 19 | Chill carcass (air or immersion) | | |
| Secondary processing | 20 | Post-chill applications | | |
| | 21 | Portion or value adding (if applicable) | | |
| | 22 | Pack whole carcass, portions or products | | |
| | 23 | Chill or freeze | | |
| | 24 | Storage | | |
| Distribution channels | 25 | Transport | | |
| | 26 | Wholesale premises (if applicable) | | |
| | 27 | Transport | | |
| | 28 | Retail or food service | | |
| | 29 | Transport | | |
| | 30 | Consumer | | |

* Diagram has been recreated from (CCFH, 2010), with some modifications taken from (FSANZ, 2005)

*Salmonella* species colonise the intestinal tract of poultry where they can persist throughout the bird's lifespan in a poultry-producing environment and are shed with faeces (FAO/WHO, 2002; Gast, 2003). Faecal shedding allows salmonellae to be transmitted between birds in a flock. Poultry can become colonised by *Salmonella* species via horizontal transmission from litter, faeces, feed, water, fluff, dust, shavings straw, insects, equipment and other fomites, or by contact with other poultry or animals (e.g. rodents, wild birds), or contact with contaminated workers (CCFH, 2007; Poppe, 2000).

The risk of poultry becoming contaminated with salmonellae at farm level can be reduced by establishing strict biosecurity measures (including ensuring that poultry feed and water is *Salmonella*-free) (FSANZ, 2005), vaccination programmes (see Section 5.1.3.7) or the use of antibiotics (see Section 5.1.3.6). Biosecurity is particularly important to keep grandparent and parent flocks *Salmonella*-free. The New Zealand poultry industry has established a Broiler Growing Biosecurity Manual.[8] A survey of 60 New Zealand broiler farms found a good level of compliance with the procedures outlined in the Biosecurity Manual (Lake *et al*., 2008b).

Other strategies to prevent infection that are in development include encouraging immunity or resistance to infection in birds through the use of antibodies, feed additives or acidified food/water, and the use of bacteriophages.

Comparisons of *Salmonella* species contamination of free range or organic production systems with "conventional" systems have produced varied results and more statistically valid surveys are required to ascertain if differences do occur (Young *et al.*, 2009).

The transportation of poultry between farms and from the farm to the processing plant creates an environment where *Salmonella* species might be spread between birds (Mulder, 1995, Corry *et al*., 2002; Marin and Lainez, 2009). Increased shedding of pathogens in faecal material during transport is believed to be related to increased stress in birds (Mulder, 1995, Corry *et al*., 2002). The New Zealand Code of Practice for poultry processing recommends minimising bird stress and withholding feed (but not water) for 4-10 hours prior to slaughter (including catching and transportation time) to reduce contamination (NZFSA, 2009a).

Further information on *Salmonella* in poultry primary production is included in Appendix 1, Section 7.3.1.

### 2.4.2 Poultry primary processing

Most studies show the prevalence of *Salmonella* species to be higher on poultry carcasses at the end of primary processing than at the start (Lillard, 1990; Lake *et al.*, 2005), although the concentrations of organisms on carcasses tend to decrease (CCFH, 2007). There are two main sources of *Salmonella* contamination in the processing plant: the birds themselves and cross-contamination from other birds or the environment (FSANZ, 2005).

[8] http://www.pianz.org.nz/pianz/wp-content/uploads/2010/10/Broiler_Growing_Biosecurity_Manual.pdf (accessed 25 June 2011).

The steps in production most likely to increase the prevalence of *Salmonella* species on poultry are defeathering and evisceration, while high temperature scalding and spray washes are likely to decrease the prevalence (see Section 7.3.2) (FSANZ, 2005).

The New Zealand Code of Practice for poultry processing includes requirements for cleaning of defeathering equipment and recommends use of antimicrobials and physical separation of defeathering from later primary processing steps (NZFSA, 2009a). The Code also contains requirements for processors to define acceptable levels of visible faecal contamination following evisceration and monitoring requirements for faecal contamination. Continuous sprays must be used to rinse equipment and carcasses during evisceration and the use of an antimicrobial in the rinse water is recommended (NZFSA, 2009a). In New Zealand, a scald temperature of 56-58°C (high temperature) is standard (Lake *et al.*, 2007). Information on the effectiveness of different antimicrobial agents in spray rinses is included in Appendix 1, Section 7.3.2.2.

Further information on poultry primary processing and *Salmonella* control in poultry primary processing is included in Appendix 1, Section 7.3.2.

### 2.4.3 Poultry secondary processing

Poultry secondary processing includes portioning, and processing of carcasses or portions into value-added products. During secondary processing, *Salmonella* prevalence may increase due to cross-contamination, while concentrations of *Salmonella* may increase if temperature control is not properly maintained.

Further information on poultry secondary processing and *Salmonella* control in poultry secondary processing is included in Appendix 1, Section 7.3.3.

### 2.4.4 Retail and domestic handling

Both poultry muscle and skin are excellent substrates for a wide variety of microorganisms (ICMSF, 2005), but the potential shelf life of raw poultry is quite short (e.g. chicken samples had spoiled after 4 days at 9°C (Abu Ruwaida *et al.*, 1996)). Unless frozen, raw poultry has a rapid turnover at retail, often 24-48 hours with a best before date of 3-4 days (King and Wong, 2010).

*Salmonella* species can survive well at refrigeration temperatures and will grow on fresh poultry under warmer, more favourable, temperatures (e.g. during transportation from a retail outlet to a consumer's home). *Salmonella* species numbers are reduced under frozen storage but can survive, so freezing is not considered to be an adequate control step.

The times and temperatures of purchased poultry products during transportation by consumers have been examined in a New Zealand study (Gilbert *et al.*, 2006). A surface temperature of 30°C (optimal growth temperatures for *Salmonella* species are 35-37°C) was only recorded under the following three conditions (based on mean values of replicates), all during summer:

- Storage in a plastic bag in the car interior (30°C reached after approximately 1.5 hours);

- Storage in a plastic bag in the car boot (30°C reached after approximately 4 hours); and
- Storage in a chiller bag (no icepack) in the car interior (30°C reached after approximately 3.25 hours).

In a New Zealand consumer survey, the majority of poultry (62.9%) was purchased fresh (rather than frozen), and most consumers (94.4%) claimed that the time taken from food selection to reaching their home was one hour or less (Gilbert *et al.*, 2007). Approximately 64% of poultry purchased would be frozen once the consumer got it to their home.

This consumer survey also showed that thawing poultry at room temperature for up to 12 hours was a common practice (Gilbert *et al.*, 2007). Any salmonellae present on the surface of the poultry would be able to grow once the surface reached room temperature, but studies have shown that the time required for frozen poultry (-18°C) to reach minimum growth temperature (7°C) would be in the range 3-16 hours, depending on the freezer temperature and ambient (air) temperatures (McIntyre *et al.*, 2007). Ambient temperatures of up to 28ºC were included in this study. As growth is greatly reduced up to 15°C (requiring another 3 hours thawing), and not optimal until 35-37°C, normal thawing periods before cooking are unlikely to permit much growth, although situations involving warm freezer temperatures (-7°C) and high ambient temperatures may increase the amount of growth that occurs. Thawing experiments were conducted with chicken portions and whole chickens are likely to thaw more slowly.

Conventional cooking (>60°C) would normally be expected to rapidly inactivate *Salmonella* in food (D value less than 2 minutes at 65°C and less than 30 seconds at 70°C). This is the most important control step for eliminating any salmonellae that might be present on or in a poultry product.

Further information on domestic poultry handling practices and their impact on *Salmonella* survival and growth is included in Appendix 1, Section 7.3.5.

## 2.5 Exposure Assessment

### 2.5.1 Prevalence of *Salmonella* species in poultry and poultry products in New Zealand

#### *2.5.1.1 Testing programmes*

The National Microbiological Database (NMD) records results from the testing of poultry carcasses sampled at the end of primary processing (i.e. after the spin chiller). Data from the NMD indicates that, since 2007, the prevalence of *Salmonella* species has been less than 1% on these samples (see Section 7.4.1 for more information on the NMD programme and details of results for the years 2005-2010).

#### *2.5.1.2 Product surveys*

The results of seven product surveys have been reported in Appendix 1 (Section 7.4.2). Recent surveys have indicated that the prevalence and concentration of *Salmonella* species on retail poultry products is low, which is consistent with the NMD data. The most recent survey, in 2008, did not find *Salmonella* species in 163 whole carcass retail samples

(Chrystal *et al.*, 2008). A survey undertaken between 2003 and 2004 did not detect *Salmonella* in 300 retail samples of chicken portions, while one of 310 (0.3%; 95% CI 0.01-1.8%) samples of chicken portions provided by primary processors was positive (the serotype identified was *S.* Agona, present at <6 MPN/portion) (Wong, 2004).

A study between 2003 and 2005 found 3.0% (95% CI 1.2-6.1%) prevalence in 232 chicken samples purchased from retail outlets in Auckland, Hamilton, Wellington, Christchurch and Dunedin. The highest concentration recorded was 0.61 MPN/g (Wong *et al.*, 2007).

A 2003 Christchurch study (Wong, 2003) found a 7% (95% CI 3.9-11.5%) prevalence of *S.* Typhimurium in 200 retail poultry samples. This study was commissioned to assess the flow-on effects from a batch of *Salmonella* contaminated feed used in primary production and shows that sporadic contamination events can occur. The *S.* Typhimurium type isolated from the feed (DT1), was isolated from ten poultry samples, while a further three contained DT12a and a further one contained both types. However, for most positive samples the salmonellae were present in low numbers (<9 MPN/sample).

A 2005-2006 study detected a prevalence of 24.5% (95% CI 18.7-31.1%) of salmonellae in broiler chickens sampled from processing plants prior to scalding (Wong and Hudson, 2006). The highest concentration of *Salmonella* reported was $3 \times 10^3$ CFU/bird.

There have not been any recent surveys of salmonellae in ready-to-eat chicken products. A study published in 1995 reported that *Salmonella* species were not detected in 1,326 ready-to-eat chicken products (Campbell and Gilbert, 1995).

A 2002 study evaluated *Salmonella* species contamination on the outside of poultry packaging (Wong *et al.*, 2004). Of 300 packs of fresh chilled raw poultry purchased from retail outlets in Christchurch, *Salmonella* species were detected on one (0.3%; 95% CI 0.01-1.8%), at a concentration of <6 MPN/pack.

#### *2.5.1.3 Recalls*

Between 2001 and April 2011 there were no New Zealand recalls issued for contamination of poultry products with *Salmonella* species. Recalls will usually relate to ready-to-eat products. An investigation of the New Zealand poultry industry estimated that approximately 14% of poultry would reach the consumer in a pre-cooked form (Lake *et al.*, 2008a).

### 2.5.2 Poultry consumption

Consumption of poultry meat has increased steadily over the last 20 years, from an apparent consumption (poultry available for consumption per capita) of 14 kg/person/year in 1986 to 34.1 kg/person/year in 2006. This figure decreased to 30.4 kg/person/year in 2009, as part of a general 6.6% decrease in meat consumption compared with the previous year. In 2009, New Zealanders consumed 136,728 tonnes of poultry meat, which constitutes 35.8% of total meat consumption.[9]

[9] http://www.pianz.org.nz/industry-information/industry-statistics/meat-consumption/meat-consumption-percentages (accessed 25 June 2011).

Food Standards Australia New Zealand (FSANZ) carried out an analysis of the 1997 National Nutrition Survey dataset (Russell *et al.*, 1999), including application of a set of standard recipes, to allow composite foods to be reduced to their component parts (ANZFA, 2001). This analysis gave an estimate of the proportion of the population consuming poultry meat on any given day of 27.5%.

The following information is taken from the New Zealand National Nutrition Survey (NNS) conducted in 1997 (Russell *et al.*, 1999) and the 2002 Children's National Nutrition Survey (CNS) (MoH, 2003), unless otherwise stated. It should be noted that these two surveys are now quite old and general trends in poultry consumption suggest that figures from these surveys will probably under-estimate current poultry consumption.

This analysis refers only to chicken consumed as chicken meat or chicken portions and not to chicken consumed as a minor component of a recipe. Therefore, figures will differ from those of the FSANZ analysis described above.

#### *2.5.2.1 Proportion of population consuming poultry*

For the adult New Zealand population, 19.4% reported consuming chicken in the previous 24-hour period. Using data from the qualitative food frequency questionnaire (QFFQ), administered as part of the NNS, estimates of 12.2% consuming chicken (roasted, fried, steamed or barbecued) and 9.8% consuming chicken mixed dishes were obtained.

For children aged 5-15 years, 24.4% reported consuming chicken in the previous 24-hour period. The QFFQ, administered as part of the CNS suggests a much higher frequency of chicken consumption of approximately 34%.

Consumption of other poultry types was negligible.

A more recent survey of foods consumed by 12-24 month old New Zealand children found that 22% of respondents reported consuming chicken or turkey at least once on three randomly-selected non-consecutive days (Szymlek-Gay *et al.*, 2010).

#### *2.5.2.2 Mean daily consumption of poultry*

Consumers are defined as those who report consumption of a particular food within the survey timeframe. Analysis of poultry serving data from the 1997 NNS gave a mean daily intake for consumers of poultry of 136 g/person/day. The corresponding data for the child population (5-15 years) gave a mean daily consumption for consumers only of 114 g/person/day.

For 12-24 month old New Zealand consumers of chicken and turkey, the median daily intake was 22 g/person/day (Szymlek-Gay *et al.*, 2010).

#### *2.5.2.3 Types of poultry consumed and cooking method used*

The following section summarises information on portion types and cooking methods for chicken servings reported in the NNS and CNS.

For adult New Zealanders, the most commonly consumed portion type was breast (28% of servings), followed by drumstick (11.4%), light meat (11.4%), leg (9.8%), thigh (9.1%) and wing (8.2%). Overall, 10.2% of servings were described as 'Chicken, KFC' (Kentucky Fried Chicken™). The most common cooking method was baking/roasting (39.2% of servings), followed by frying (12.5%), stewing/braising (12.3%), and grilling/barbecuing (8.9%). The cooking method was not specified for 16.7% of servings.

For New Zealand children, the most commonly consumed portion type was drumstick (25.9%), followed by breast (19.9% of servings), wing (10.7%), light meat (8.8%), thigh (7.1%) and leg (6.7%). Only 4.2% reported consuming 'Chicken, KFC'. The most common cooking method was baking/roasting (44.4% of servings), followed by frying (15.7%), stewing/braising (10.1%) and grilling/barbecuing (10.1%).

These data on cooking methods are in broad agreement with the results of a postal survey of meat handling practices (Gilbert *et al.*, 2005). In this survey, 50% of respondents ($n$ = 257) reported that they would always or very frequently roast or bake chicken, while 31% of respondents ($n$ = 167) reported that they would always or very frequently pan fry chicken.

### 2.5.3 Evaluation of exposure

#### *2.5.3.1 Frequency of consumption and serving sizes*

Estimates of the proportion of the population consuming poultry meat on any given day ranged from 19.4% (adults) to 34% (children). The amount of poultry consumed is similar for adults and children (mean approximately 100 g).

#### *2.5.3.2 Frequency of contamination*

There are no recent data to indicate the prevalence of *Salmonella* species on cooked poultry in New Zealand, but NMD and survey data indicate that the prevalence of *Salmonella* species on raw poultry in New Zealand is low (<3% at retail; <1% at primary production).

#### *2.5.3.3 Growth rate during storage and most likely storage time*

At retail, poultry products are usually kept refrigerated or frozen and, provided the temperature is maintained at 7°C or below (ideally 4°C or below), salmonellae growth will be prevented. Unless frozen, raw poultry has a rapid turnover at retail, often 24-48 hours with a best before date of 3-4 days (King and Wong, 2010).

New Zealand studies of the transportation and refrigeration of raw poultry products by consumers showed that:

- Transportation of raw poultry by consumers was not likely to create warm enough conditions for enough time to cause any significant growth in *Salmonella* species, but refrigeration by consumers was not always adequate (Gilbert *et al.*, 2006). Simulated transport conditions included Summer and Winter conditions, transport times up to 6.5 hours and transport packaging ranging from a supermarket bag only to a cooler bag containing ice packs; and

- Records of the thawing practices of consumers ($n$ = 38) and thawing temperatures indicated that thawing at room temperature by consumers is not likely to encourage significant growth of *Salmonella* species (McIntyre *et al.*, 2007).

*Salmonella* is capable of growing to high numbers in inadequately refrigerated poultry products, and if a poultry product is contaminated with *Salmonella* species it is possible that the pathogen might be allowed to multiply while under the control of a retailer or consumer. A study of the growth of *Salmonella* species in minced chicken at 10°C demonstrated that salmonellae readily multiplied when the temperature was allowed to fluctuate briefly to 30°C (Bovill *et al.*, 2000). However, spoilage bacteria are also able to multiply during periods of inadequate refrigeration and spoilage of the product may prevent consumption.

#### *2.5.3.4 Heat treatment*

The studies summarised in Section 7.5.1 indicate that *Salmonella* are not unusually heat resistant when present in poultry-based foods. Normal cooking is therefore adequate to inactivate any organisms that might be present, although there may be greater risk of undercooking if poultry is cooked in a microwave oven, either due uneven surface heating (Göksoy *et al.*, 1999) or incorrect microwave cooking technique (Smith *et al.*, 2008). Experimental data indicate that fattier poultry products, or processed products such as chicken nuggets, require slightly longer cooking times to ensure any *Salmonella* species are inactivated.

In a New Zealand survey of domestic consumers, 35/128 (27.3%) respondents reported that they roasted chicken until it was "medium", and the remainder roasted chicken until it was "well done" or "very well done" (Gilbert *et al.*, 2007). In the same survey, the majority of participants (261/312; 83.7%) reheated leftover food until it was "steaming hot"; any *Salmonella* species present on the reheated food would most likely be inactivated. Fewer participants (34/312; 10.9%) reheated food until it was "warm", which suggests inadequate reheating for the purposes of removing pathogenic bacteria. However, this type of self reported data needs to be treated with caution as it may not match actual behaviours.

#### *2.5.3.5 Exposure summary*

The information presented here on exposure to *Salmonella* through consumption of poultry meat indicates that the food is commonly eaten, but that the probability of contaminated product (raw or purchased ready-to-eat) is low. The limited quantitative data has indicated that counts in contaminated retail samples are low (see Section 2.5.1.2). Normal domestic thawing of frozen poultry does not appear to provide much opportunity for growth. Cooking (>60°C) will readily destroy the organism.

## 2.6 Overseas Context

A summary of overseas studies of the prevalence of *Salmonella* on poultry or poultry products is provided in Appendix 1, Section 7.5.

Surveys of raw poultry are summarised in Table 14. The prevalence of *Salmonella* in Australian raw poultry is notably higher than in New Zealand, but a large proportion of the isolates are *S.* Sofia, which is found rarely in human infections, and the isolates of this

serotype found in Australia are considered "benign", or even non-pathogenic (Harrington et al., 1991; Sumner *et al.*, 2004a). In Europe and North America the prevalence is also high compared to New Zealand, while for some countries in Asia the prevalence can exceed 50%.

There are fewer studies of the prevalence of *Salmonella* in ready-to-eat poultry products but those summarised in Table 15, as well as the results of the United States Department of Agriculture Food Safety Inspection Service (USDA FSIS) surveys, indicate a very low or zero prevalence.

# 3 EVALUATION OF ADVERSE HEALTH EFFECTS

## 3.1 Disease Characteristics

Information regarding the disease characterisitics of non-typhoidal *Salmonella* outlined below is primarily from D'Aoust and Maurer, (2007), Jay *et al.* (2003), FAO/WHO, (2002) and the NZFSA datasheet, unless referenced elsewhere.[10]

*Incubation:* 8-72 hours, commonly 12-36 hours.

*Symptoms:* Non-bloody diarrhoea, abdominal pain, vomiting, nausea and fever lasting 2-7 days.

*Condition:* Salmonellosis, presents with symptoms of gastroenteritis or enterocolitis.

*Toxins:* Toxins are not produced in foods, but salmonellae may produce enterotoxins and cytotoxins within epithelial cells (Jay *et al*., 2003).

*People Affected:* Anyone can be infected, but rates of disease and the likelihood of more severe outcomes are higher amongst the young, old, and immunocompromised (FAO/WHO, 2002; Gorden 2008).

*Treatment:* The infection is usually self-limiting. Uncomplicated gastroenteritis may require supportive therapy such as fluid and electrolyte replacement, especially in the elderly or young children. The use of antibiotics is not recommended for mild or moderate cases because it prolongs the carriage and excretion of salmonellae.

*Long Term Effects:* Extra-intestinal infections have been reported to occur in approximately 7% of notified cases in the United States (Jones *et* al., 2008). Extra-intestinal infections usually require hospitalisation and treatment with antimicrobials. An increased risk of blood stream infections (bacteraemia) has been linked to patients with concurrent systemic lupus erythematosus, liver cirrhosis, solid organ cancers or immunodeficiency, and risk factors for atherosclerosis predisposed patients with blood stream infections to acquire endovascular infection (Hsu and Lin, 2005). Reactive arthritis may follow 3-4 weeks after onset of gastrointestinal symptoms and when it occurs can persist for 3-5 months, although long-term chronic conditions such as Reiter's Syndrome, septic arthritis or septicemia can also develop in some cases (Hannu *et al.*, 2006).

## 3.2 Dose-Response

The dose-response is the relationship between the number of microbial organisms ingested and a specific outcome such as infection, illness or death (Bollaerts *et al.*, 2008). Dose-response can be estimated from human feeding trials, animal trials, *in vivo* experiments, modelling or analysis of outbreak data. Calculation of dose-response can be difficult due to differences in host susceptibilities (e.g. individuals who are young, elderly, pregnant or immunocompromised are typically more susceptible to infection) and in *Salmonella* serotype infectivity (Bollaerts *et al.*, 2008).

[10] http://www.foodsafety.govt.nz/elibrary/industry/non-typhoid-salmonellae.pdf (accessed 7 November 2011).

For *Salmonella* the dose-response relationship can be estimated from either feeding trials with volunteers, or from outbreaks where the number of cells ingested can be estimated. Using outbreak data, the Food and Agriculture Organisation (FAO) and World Health Organization (WHO) produced a dose-response model as an output from the joint risk assessments of *Salmonella* in eggs and broiler chickens (FAO/WHO, 2002). The FAO/WHO model has been developed further to account for differences in host susceptibility, serotype infectivity and food matrix (Bollaerts *et al.*, 2008).

Most recently (Teunis *et al.*, 2010) used data from 35 salmonellosis outbreaks, three sporadic cases for which there was good dose information and two human volunteer feeding studies to estimate that the number of cells that need to be ingested to cause a 50% probability of illness was as low as 36.3, although the 95% percentiles were widespread (0.69-1.26 x $10^7$).

Further details are given in Appendix 2.

### 3.3 New Zealand Outbreak Information and Human Health Surveillance

Salmonellosis is a notifiable disease in New Zealand. The number of cases and incidence of notified (non-typhoidal) salmonellosis since 2003 is shown in Table 2. More historical data are given in Appendix 2.

**Table 2: Notification rates for salmonellosis in New Zealand**

| Year | Number of cases[1] | Incidence (cases/100,000) |
|---|---|---|
| 2003 | 1,401 | 37.5 |
| 2004 | 1,081 | 28.9 |
| 2005 | 1,383 | 37.0 |
| 2006 | 1,335 | 31.9 |
| 2007 | 1,274 | 30.1 |
| 2008 | 1,346 | 31.5 |
| 2009 | 1,129 | 26.2 |
| 2010 | 1,146 | 26.2 |

[1] Number of cases data taken from (ESR, 2010a), Population data for June each year taken from (http://www.stats.govt.nz/methods_and_services/access-data/tables/national-pop-estimates.aspx). Due to population adjustments by Statistics New Zealand rates may differ slightly from older Annual Surveillance Summary reports.

The notification rate per 100,000 population for cases of salmonellosis in New Zealand from 2000 – 2010 is shown in Figure 4. The rate has been stable since 2005 at approximately 30±4 per 100,000.

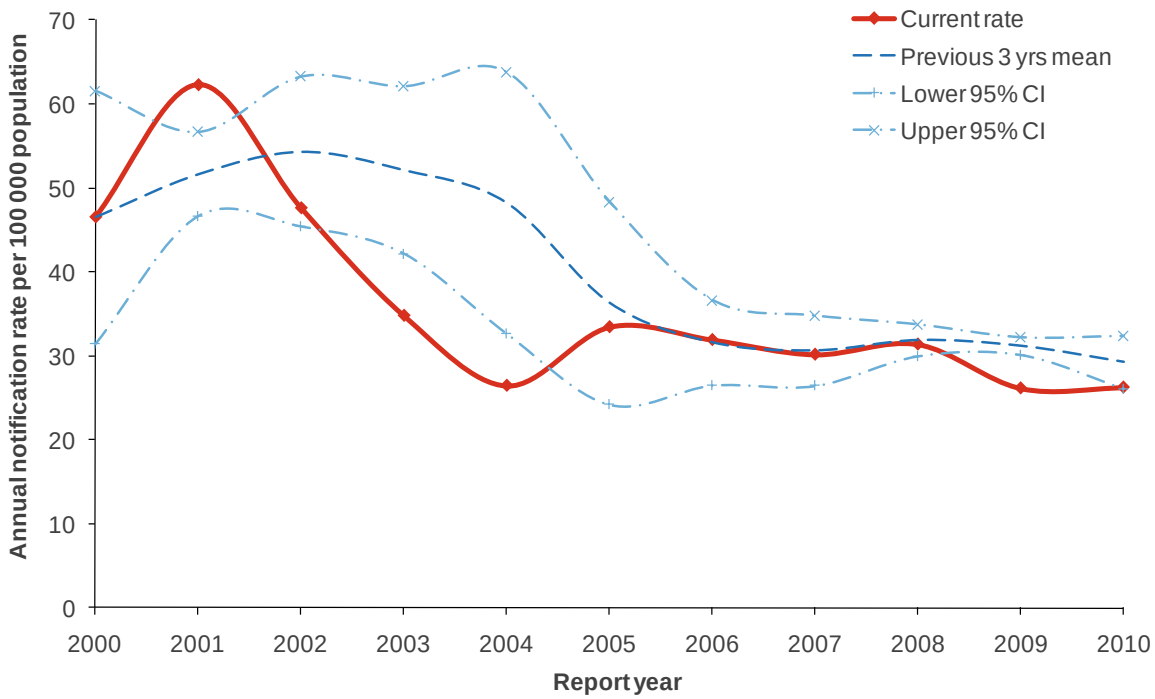



**Figure 4: Incidence of notified salmonellosis in New Zealand 2000 – 2010**

Reproduced from (Lim *et al.*, 2011)

The incidence of salmonellosis is characterised by a late summer peak and a winter trough. Rates of salmonellosis vary throughout the country but higher rates are often reported from the lower South Island, in particular South Canterbury DHB (2010 rate was 66.2 cases per 100,000, 37 cases) features in the highest quantile of salmonellosis notification rates between 2008 and 2010.

Reported rates are similar for males (26.2/100,000 in 2009) and females (25.7/100,000 in 2009). Age specific rates are highest for the <1 year age group (123.7/100,000 in 2009), and 1 to 4 year olds (89.9/100,000 in 2009).

### 3.3.1 Clinical outcomes: Salmonellosis in New Zealand

Hospitalisation and fatality rates for notified cases of salmonellosis in New Zealand are given in Table 3. These outcomes are not always reported for each case, so percentages are expressed in terms of the number of cases for which outcomes are known.

**Table 3: Outcome data for salmonellosis in New Zealand, 2005-2009**

| Year | Hospitalised cases | Fatalities | Reference |
|---|---|---|---|
| 2005 | 142/1134 (12.5%) | 1/1383 (0.07%) | (ESR, 2006b) |
| 2006 | 148/1111 (13.3%) | 1/1335 (0.07%) | (ESR, 2007a) |
| 2007 | 110/833 (13.2%) | 1/1274 (0.07%) | (ESR, 2008a) |
| 2008 | 123/896 (13.7%) | 1/1346 (0.07%) | (ESR, 2009b) |
| 2009 | 134/716 (18.7%) | 1/1129 (0.09%) | (ESR, 2010a) |
| 2010 | 136/763 (17.8%) | 0/1146 (0%) | (ESR, 2011) |

Chronic sequelae of *Salmonella* infections include reactive arthritis. A study carried out in the south of New Zealand found evidence of preceding *Salmonella* infection in two of 60 (3.3%; 95$^{th}$ percentile confidence interval 0.4-11.5%) cases of reactive arthritis (Highton and Priest, 1996). Studies from other countries have found the rates of *Salmonella*-associated reactive arthritis to vary from 4.2-18.7% (Townes 2010).

### 3.3.2 Serotypes causing disease in New Zealand

The Enteric Reference Laboratory (ERL) performs typing of *Salmonella* for the whole of New Zealand. From 2000 through 2009, *S.* Typhimurium was the most prevalent serotype reported for salmonellosis cases in New Zealand (Adlam *et al.*, 2010). This serotype caused 58.2% of 11,554 cases for which serotype information was available. The next most frequently reported serotype was *S.* Enteritidis (8.8% of cases). When considering serotype and phage type, *S.* Typhimurium DT160 was most frequently reported (19% of cases). There were 35 serotypes that caused 50 or more salmonellosis cases during this period, and together these serotypes caused 80% (9,290) of the 11,554 cases.

The incidence of the five serotypes causing the greatest number of cases from 2000 through 2009 (*S.* Typhimurium DT160, *S.* Typhimurium DT1, *S.* Brandenburg, *S.* Typhimurium DT135 and *S.* Typhimurium DT156) all peaked during 2000 through 2002 (Adlam *et al.*, 2010). While these serotypes are still isolated frequently from salmonellosis cases (*S.* Typhimurium DT160 is still the most commonly isolated serotype), a variety of other serotypes have peaked in recent years, such as *S.* Infantis, *S.* Mbandaka and *S.* Stanley.

The serotypes significantly associated with cases living in highly urban areas are *S.* Infantis ($p$<0.001) and *S.* Typhimurium DT160 ($p$<0.05) (Adlam *et al.*, 2010). The serotypes significantly associated with cases living in highly rural areas are *S.* Saintpaul ($p$<0.001), *S.* Brandenburg ($p$<0.01) and *S.* Typhimurium DT101 ($p$<0.05).

Appendix 2 contains more detail on *Salmonella* serotypes of human isolates in New Zealand.

### 3.3.3 Antimicrobial resistance of New Zealand *Salmonella* strains

ESR tests the antimicrobial resistance of approximately 20% of all human and non-human *Salmonella* isolates received for typing, along with all *S.* Typhimurium phage types that are internationally recognised as being multiresistant.[11] The results of this testing have been

[11] Data are available from the annual reports of antimicrobial susceptibility among *Salmonella*, produced by ESR and available at: http://www.surv.esr.cri.nz/antimicrobial/salmonella.php (accessed 1 December 2010).

compiled in Appendix 1 for the years 2005 through 2009. Rates of antibiotic non-susceptibility in *Salmonella* in New Zealand are increasing but still lower than in many international settings (Broughton *et al.*, 2010).

### 3.3.4 Outbreaks

The number of reported outbreaks of salmonellosis in recent years in New Zealand is given in Table 4 (figures exclude *S.* Typhi and *S.* Paratyphi). The number of salmonellosis outbreak cases is approximately 10% of those reported as sporadic cases. As a proportion of all enteric outbreaks or outbreak cases, salmonellosis makes a small contribution; the outbreak data are dominated by reported outbreaks of norovirus.

**Table 4: Reported outbreak data for salmonellosis in New Zealand 2005-2010 (as a proportion to total enteric bacterial, viral, parasitic and gastroenteritis outbreaks and cases)**

| Year | Salmonellosis outbreaks/ total enteric outbreaks | Cases/Total Enteric Cases[1] | Reference |
|---|---|---|---|
| 2005 | 26/338 (7.7%) | 120/2343 (5.1%) | (ESR, 2006a) |
| 2006 | 22/481 (4.6%) | 74/6162 (1.2%) | (ESR, 2007b) |
| 2007 | 8/477 (1.7%) | 141/7821 (1.8%) | (ESR, 2008b) |
| 2008 | 15/428 (3.5%) | 163/6295 (2.6%) | (ESR, 2009a) |
| 2009 | 12/586 (2.0%) | 76/10176 (0.7%) | (ESR, 2010c) |
| 2010 | 23/559 (4.1%) | 100/5929 (1.7%) | (ESR, 2011) |

[1] Includes both suspected and confirmed cases

A review of 204 salmonellosis outbreaks from 2000-2009 found that while non-typhoid salmonellosis was primarily a foodborne disease in New Zealand, there was insufficient information to identify important food vehicles (King *et al.*, 2011). Of the 70 outbreaks with at least some evidence of food as the sole route of transmission, 24 had moderate evidence, with a food or food type identified in 23 of these outbreaks.[12] For 9 (39%) of these 23 outbreaks, chicken or chicken as an ingredient was suspected, for 3 (13%) egg or egg as an ingredient was suspected, and for another 3 (13%) both chicken and eggs were suspected vehicles. Of the 22 outbreaks with strong evidence a contaminated food was identified in 7 outbreaks, with *S.* Thompson isolated from foods containing chicken from one outbreak.

Although it does not appear to have been reported as an outbreak, during January and February of 2003 contamination of broiler poultry feed with *Salmonella* Typhimurium DT1 was detected in the Canterbury region through industry testing (Cook *et al.*, 2006; Wong, 2003). The contamination was thought to have originated from wheat used in the feed formulation. Increases in the prevalence of *S.* Typhimurium DT1 on chicken at retail and in

[12] Strength of evidence classifications were: (i) weak, where affected persons had a history of exposure to the implicated source, (ii) moderate, where critical control point failures were linked to the implicated source (either specified in the free text fields or identified as part of the formal record) or a case control or cohort study revealed an elevated risk for persons exposed to the implicated source, and (iii) strong, where the same *Salmonella* serotype was isolated from one or more affected persons and the implicated source (including food handlers).

the number of notified human cases of salmonellosis, albeit small, were observed during this period.

Chicken, whole and in portions, from supermarkets, fast food outlets and restaurants were tested for *Salmonella* on eight occasions with sampling dates from 11 February 2003 to 7 March 2003. At the first sampling, 36% of samples (9/25) were *Salmonella* positive with 8/9 positive samples typed as *S.* Typhimurium DT1. At the second sampling (13 February 2003), 17% (4/24) of samples were *Salmonella* positive, with half of the isolates typed as *S.* Typhimurium DT1. One further *S.* Typhimurium DT1 positive sample was found at the third sampling, with no further *Salmonella* positive samples found on the subsequent five sampling occasions.

No human cases of *S.* Typhimurium DT1 were notified in Canterbury during October 2002-January 2003, but seven cases were notified during February 2003.

### 3.3.5 Case control studies and risk factors

#### *3.3.5.1 Case-control studies concerning Salmonella and poultry in New Zealand*

There have been two case-control studies performed to investigate outbreaks where exposure to poultry was identified as one of several possible risk factors for salmonellosis, but neither study was able to confirm poultry (or any other food) as the cause.

An outbreak of 24 cases of *S.* Enteritidis 9a infection in 2005 was associated with consumption of food purchased from a premises serving Middle Eastern dishes (OR = 10.2, 95% CI 2.4-49.9) (Anonymous, 2005). No single food item was identified as being associated with infection; consumption of chicken, hummus, flat bread, lettuce, tomato, onions and cabbage were all significantly associated with infection. Testing of food samples from the implicated premises identified *S.* Orion from tahini but *S.* Enteritidis 9a was not isolated.

An outbreak of 34 cases of *S.* Mbandaka infection in 2008 was epidemiologically linked with purchasing chicken breast from a supermarket that was supplied by a specific poultry processor (odds ratio in multivariate model = 9.24 or 5.83, depending on the model used), and eating eggs prepared away from home (odds ratio in multivariate model = 7.41 or 6.11, depending on the model used) (McCallum and Das, 2008). *Salmonella* was not isolated from food samples from case homes and implicated food premises, using swabs from bench tops, chopping boards, fridges and hand wash basins. While a specific poultry processor was the suspected source of the outbreak, no laboratory evidence was available to confirm this.

There have been five case-control studies in addition to those cited above and these are summarised in Appendix 2. Poultry or poultry products were not identified as being significantly associated with salmonellosis in any of these studies.

#### *3.3.5.2 New Zealand attribution studies*

In 2007, the NZFSA Science Group reported on modelling activities to support decision making on importing poultry products from the United Kingdom (NZFSA Science Group, 2007). The initial phase of this work involved estimating the number of salmonellosis cases

per year attributable to different exposure pathways. Expert opinion predicted an estimated 9,000 cases of human salmonellosis per annum in New Zealand, of which 63% (5,668) were estimated to be caused by foodborne transmission. Epidemiological reports of cases in New Zealand (1998-2003) and Australia (1995-2000) were used to estimate the contribution of different food categories. Domestic poultry was estimated to contribute 937/5,668 (17%) of foodborne cases, or 937/9,000 (10%) of all salmonellosis cases (taking into account non-foodborne pathways). Additional analyses, based on the prevalence of different *Salmonella* serotypes isolated from foodstuffs and from human cases from 2002 through 2004, estimated the relative contribution of selected food groups. The largest proportion of salmonellosis cases were attributed to chicken, and while this proportion declined each year, it remained significantly higher than the proportions of salmonellosis cases attributed to the other food groups (beef/veal, pork, lamb/mutton, eggs).

A New Zealand study using molecular sub-typing data and Bayesian techniques ('modified Hald model') estimated the food source attribution of human salmonellosis cases in New Zealand in 2003 (Müllner *et al.*, 2009). The risk model apportioned food sources to an estimated 981 cases based on 963 observed cases. The majority of cases were attributed to pork (60%), followed by poultry (21.2%). The authors advised caution in interpreting the results for pork because the data for pork were sparser and more biased than data for other sources.

A review of scientific evidence for salmonellosis aetiology in New Zealand concluded that poultry was "very likely" (>90% probability) to be at least a moderate cause (between 10-30% or higher of all cases) of salmonellosis (Wilson and Baker, 2009).

A later review of 204 New Zealand salmonellosis outbreaks from 2000 through 2009 was not able to quantify the proportions of salmonellosis cases attributable to specific foods (Adlam *et al.*, 2010; King *et al.*, 2011). There were only eight outbreaks where specific foods were identified by laboratory evidence as being contaminated with *Salmonella*; only one of these foods may have contained chicken but the record did not identify which of a variety of bakery products (chicken sandwich, bacon and egg pie, panini, fried chicken, chicken roll) was *Salmonella*-positive.

#### *3.3.5.3 Overseas attribution studies*

Overseas studies have also used typing data and outbreak reports to attribute salmonellosis. A number of Danish studies have used the extensive monitoring data available in that country in a Bayesian attribution model. The most recent publication (Pires *et al.*, 2010) attributed only a small proportion of cases (1.2-2.8%) to broilers over the period 2005-2007, while up to 10% was attributed to pork and layers (eggs) each. A considerably greater proportion of the salmonellosis burden (6.6-14.4%) was attributed to imported poultry (chicken, turkeys, ducks).

A study of foodborne illness in Latin America and the Caribbean during the period 1993-2010, using a probabilistic outbreak data model, attributed 9.7% of salmonellosis to poultry in the 1990s while only 4.8% was attributed in the 2000s (Pires *et al.*, 2011). In contrast attribution to eggs and pork increased over the same periods (from 13.5% to 37.3%, and from 3% to 7.8% respectively). It was noted that during the 1990s data on salmonellosis was provided almost exclusively by Cuba.

## 3.4 Adverse Health Effects Overseas

The incidence of notified cases of human salmonellosis in New Zealand is similar to rates in other developed countries and is almost identical to the overall rate for the European Union (EU) (see Appendix 2). In New Zealand the majority of human salmonellosis cases are caused by *S.* Typhimurium (52% in 2010; Lim *et al.*, 2011), with a lesser proportion due to *S.* Enteritidis (10% in 2010). In contrast, in the EU the dominant serotype causing human salmonellosis is *S.* Enteritidis (58% in 2008), followed by *S.* Typhimurium (22% in 2008) (see Appendix 2).

## 3.5 Health Burden of Infection with Pathogen

An estimate of the burden of foodborne illness for New Zealand (Cressey and Lake, 2007) includes an estimate for foodborne salmonellosis of 111 disability adjusted life years (DALYs). This represents 60.7% of the total 186 DALYs for salmonellosis, with the percentage foodborne being derived from an expert consultation process. This placed foodborne salmonellosis fourth on the list for foodborne illness burden (after campylobacteriosis, norovirus infection, and perinatal listeriosis).

This burden of disease estimate has been supplemented with a cost of illness estimate, based on the same incidence data (Cressey and Lake, 2008). The costs included were direct and indirect medical costs, as well as the value of lost production. This estimated the total cost for salmonellosis as $4.8 million, with foodborne infections costing $2.8 million. A more recent report estimated the cost of foodborne salmonellosis as $15.41 million (Gadiel and Abelson, 2010). This value included a monetisation of the burden of illness on individuals, previously measured as DALYs.

European estimates of the cost of salmonellosis are similar to New Zealand estimates (given population differences), with Kemmeren *et al*. estimating the cost of salmonellosis in the Netherlands to be 8.8 million Euros in 2004 (Kemmeren *et al.*, 2006).

A recent report from the United States ranked *Salmonella* as contributing the most to the total burden of foodborne illness, amongst 14 pathogens, in terms of quality adjusted life years (QALYs; 16,782) and in terms of cost of illness ($US 3.3 billion) (Batz *et al.*, 2011). *Salmonella* in poultry ranked as the fourth (of ten) highest pathogen-food pair in terms of QALYs (3,610 QALYs) and cost of illness ($US 712 million). The higher ranking pathogen-food pairs were *Campylobacter* in poultry, *Toxoplasma* in pork and *Listeria* in deli meats. No equivalent analysis of the burden of illness at the pathogen-food level is available for New Zealand.

## 3.6 Adverse Health Effects Summary

The incidence of reported salmonellosis has been stable in New Zealand since at least 2005. The rate since 2005 of 25-35 reported cases per 100,000 population is close to other developed countries, particularly those in Europe overall, and lower than in Australia.

Attribution models and systematic reviews have attributed 10-30% of domestically acquired salmonellosis to poultry. Results from outbreak investigations and case-control studies of infection with specific serotypes have found some epidemiological evidence for poultry as a

vehicle for infection, although these findings have rarely been supported by microbiological analyses. It should be noted that this is the case for all potential food vehicles for salmonellosis in New Zealand, with very few instances where human cases are linked to particular foods through microbiological evidence.

# 4 EVALUATION OF RISK

## 4.1 Existing Risk Assessments

### 4.1.1 New Zealand

The 2004 Risk Profile for non-typhoidal *Salmonella* in poultry (Lake *et al.*, 2004) commented that the low prevalence of contamination by *Salmonella* in poultry was evidence of good risk management by the New Zealand poultry industry, but that occasional spikes in *Salmonella* prevalence could occur. The authors concluded that transmission in poultry represented a minor component of salmonellosis aetiology in New Zealand.

A qualitative assessment of the risk to consumers of contracting salmonellosis from chicken nuggets was published in 2004 (Wong and Lake, 2004). These products are formed from raw ingredients and then flash fried. Following flash frying, the nugget does appear visually "cooked" but the core is still raw. Any *Salmonella*, if present internally, would survive this quick heat treatment step. The internal temperatures of the nugget at this stage were found to be in the range 12 to 26ºC. The authors assessed quality controls during manufacture, NMD data, *Salmonella* prevalence data for poultry, outbreak data and in-house testing data from a chicken nugget manufacturer and concluded that, as with any poultry product in New Zealand, there is a low (but non-zero) risk of *Salmonella* contamination in chicken nuggets. The risk of *Salmonella* infection from chicken nuggets cooked according to the manufacturer's instructions was extremely low. The manufacturing processes in the three plants visited in New Zealand were well controlled and the end products were snap frozen for ease of weighing, packaging and storage. The lack of drip from thawed nuggets would minimise cross-contamination on the kitchen surfaces and in handling food during preparation for cooking.

### 4.1.2 Other countries

The FAO and WHO have jointly carried out a quantitative risk assessment (QRA) of *Salmonella* species in eggs and broiler chickens (FAO/WHO, 2002). An exposure model was developed to estimate the probability of exposure to *Salmonella* in broiler chickens via an undercooked serving of chicken, and via cross-contamination resulting from preparation of that serving. The model began at the point of completion at the slaughterhouse and ended at consumption of a broiler that had been purchased fresh and whole from a retail outlet and prepared and consumed at home. For risk characterisation, the probability of illness was derived by combining the number of organisms ingested (from the exposure assessment) with dose-response information. The Expert Consultation commented that, using the current model, a reduction in the prevalence of *Salmonella*-contaminated chicken was associated with a one to one, or greater, reduction in the risk of illness, i.e. assuming everything else remained constant, a 50% reduction in the prevalence of contaminated poultry (e.g. 20% to 10%) produced a 50% reduction in the expected risk of illness per serving. A small reduction in the frequency of undercooking and the magnitude of the undercooking event resulted in a marked reduction in the expected risk of illness per serving. However, this reduction could be strongly affected by the risk of illness through the cross-contamination pathway. It was suggested that cross-contamination may in fact be the predominant source of risk of illness, and the nature of cross-contamination in the home is poorly understood.

As a result of this assessment, and parallel work on *Campylobacter* in poultry, the Codex Committee on Food Hygiene (CCFH) asked FAO and WHO to provide scientific advice concerning interventions to address the risks associated with these pathogens in broiler chicken meat at the point of consumption. The response to this request was published as a report of a 2009 meeting.[13] It was found that quantitative data on the effects of specific interventions applied during live animal production were available. Overall, the Good Hygiene Practices included in the CCFH guidelines for the control of *Campylobacter* and *Salmonella* in chicken meat (see Section 9.1) were endorsed.

FSANZ has quantified the risk of foodborne illness from the consumption of contaminated chicken through stochastic modelling (FSANZ, 2005). The model considered the food chain from the end of processing through transport (processing to retail), retail storage, transport (retail to food service/home), storage, cross-contamination, cooking and consumption. The effect of freezing poultry meat at the processing plant was also modelled. Due to the lack of suitable Australian data, the model was largely populated with data (non-Australian) from the scientific literature and the authors decided there was little scientific value in publishing the final risk estimate. However, the modelling did produce a list of variables that had the greatest influence on the probability of illness. The probability of illness was increased by (in order of most influential to least): *Salmonella* prevalence on carcasses at the end of processing, *Salmonella* concentration on carcasses at the end of processing, growth during thawing, using boards for other foods and not washing hands. The probability of illness was decreased by adequate cooking and *Salmonella* reduction due to freezing.

Additional information on overseas risk assessments is provided in Appendix 2.

## 4.2 Estimate of Risk for New Zealand

### 4.2.1 Risk associated with poultry or poultry products

The incidence of notified cases of salmonellosis has declined since a peak of 65 per 100,000 population in 2001, and has been stable in New Zealand since 2005 at 25-35 reported cases per 100,000 population. This rate is close to that in other developed countries, particularly those in Europe, and lower than in Australia. Throughout the 1980s and 1990s the rate fluctuated between 37 and 57 per 100,000 population, with no apparent trend.

NMD sampling of poultry for *Salmonella* only commenced in 2001, so it is not possible to consider trends before that year. The Poultry Industry Association of New Zealand (PIANZ) reported the prevalence of *Salmonella* on poultry carcasses during the 1990s as 17%.[14] While not stated on the PIANZ website, the prevalence figure for the 1990s appears to come from a retail survey of 137 unfrozen poultry samples (Campbell and Gilbert, 1995).

NMD data represent approximately 1,800-2,000 carcass rinse samples per annum, taken at the end of primary processing. The previous Risk Profile reported that data received from PIANZ indicated the prevalence found by NMD testing was 1-2% for the period 2001 to

[13] http://www.who.int/foodsafety/publications/micro/mra19/en/index.html (accessed 9 June 2011).
[14] http://www.pianz.org.nz/industry-issues/food-safety/safety-information/salmonella-in-new-zealand-broiler-chickens (accessed 9 June 2011).

2003 (Lake *et al.*, 2004). New NMD data presented in this Risk Profile, covering 2005 to 2010, shows the prevalence declining from 3.5% to 0.2% (see Section 7.4.1).

The temporal pattern of a steady and considerable decline in prevalence of *Salmonella* in poultry samples from the 1990s to 2010 is different to the pattern of the incidence of notified salmonellosis cases, and suggests that they are not strongly linked.

There have been incidents of temporary increases in the numbers of salmonellosis cases or outbreaks involving particular serotypes in New Zealand. The incidence of the five serotypes causing the greatest number of cases from 2000 through 2009 (*S.* Typhimurium DT160, *S.* Typhimurium DT1, *S.* Brandenburg, *S.* Typhimurium DT135 and *S.* Typhimurium DT156) all peaked during 2000 through 2002 (Adlam *et al.*, 2010). While these serotypes are still isolated frequently from salmonellosis cases (*S.* Typhimurium DT160 is still the most commonly isolated serotype), a variety of other serotypes have peaked in recent years, such as *S.* Infantis, *S.* Mbandaka and *S.* Stanley. The initial outbreaks of infection by some of these serotypes, such as *S.* Typhimurium DT160 and *S.* Brandenberg, were associated with animal contact, but the cause of the fluctuating incidence of other serotypes, such as *S.* Infantis, is not known. These suggest that *Salmonella* contamination of transmission vehicles is sporadic and would be difficult to detect through routine monitoring of foods or other sources of infection.

Some outbreak investigations identify poultry as the probable cause of salmonellosis, but poultry and poultry products have not been demonstrated conclusively to be a vehicle in outbreaks or case-control studies. A temporary increase in the prevalence of *S.* Typhimurium DT1 in poultry in 2003 in Canterbury occurred at the same time as an increase in reported salmonellosis in that region. Consumption of poultry was identified as a risk factor in a 2008 outbreak of *S.* Mbandaka but contamination was not confirmed by laboratory testing. A review of 204 salmonellosis outbreaks from 2000-2009 identified only one outbreak with strong evidence of a potential link to poultry, but in this outbreak the causative serotype (*S.* Thompson) was isolated from a mixed food containing chicken as an ingredient.

Despite a lack of robust epidemiological association, many foods including poultry might still be vehicles for infection for non-attributable small clusters and sporadic cases of salmonellosis. The NMD data indicate a very low prevalence of contamination in poultry carcasses at the end of primary processing, by international standards. This is consistent with the most recent retail surveys which also reported consistently low prevalences of salmonellae on poultry at retail (e.g. not detected on 163 broiler carcasses sampled in 2007, detected in 7/232 samples of minced or chopped raw chicken, 2003-2005). The low prevalence of *Salmonella* in New Zealand poultry suggests that, although poultry is a frequently consumed food by the New Zealand population, exposure to *Salmonella* will be infrequent. This appears to be at variance with the results of a modelling exercise, which attributed 21% of salmonellosis cases to poultry, and a review of scientific evidence that concluded that poultry was “very likely” (>90% probability) to be at least a moderate cause (between 10-30% or higher of all cases) of salmonellosis (Section 3.3.5.2).

The information in Section 7.3.5.6 indicates that conventional cooking (>60°C) would normally be expected to rapidly inactivate *Salmonella* in food (D value less than 2 minutes at 65°C and less than 30 seconds at 70°C). Therefore, thorough cooking of poultry will eliminate any *Salmonella* that might be present. This is supported by the results from

overseas surveys of ready-to-eat chicken products in developed countries, which have been cooked by producers prior to reaching the public. The prevalences found in these surveys were <1%.

The low risk from this food/hazard combination, as assessed by the 2004 Risk Profile, does not appear to have changed. On the basis of the reduced prevalence in *Salmonella* found on poultry carcasses by the NMD testing programme from 2005 to 2010, it could be argued that the risk has declined.

### 4.2.2 Risks associated with other foods

Risk Profiles with *Salmonella* as the hazard have been written for the most commonly suspected food transmission vehicles (other than poultry):

- Eggs (Lake *et al.*, 2004a) (currently being updated):
- Pork and pork products (Gilbert *et al.*, 2010a)
- High lipid foods made from sesame seeds, peanuts, and cocoa beans (Lake *et al.*, 2010)

The Profile concerning eggs concluded that there was "little evidence that transmission of *Salmonella* via eggs is a significant transmission route occurring in New Zealand". The Profile concerning pork concluded: "There are insufficient data available to assess the risk to New Zealanders from *Salmonella* in pork. The data that are available suggest a low prevalence of contamination, and pork is rarely identified as a vehicle in reported salmonellosis outbreaks."

The review of information concerning high lipid foods considered that contamination of these foods by *Salmonella* was likely to be sporadic, but when contamination did occur the potential for illness would be high, partly because ingestion of cells in high lipid foods protects them from the acid conditions in the stomach. The Profile concluded that such foods represented a minor component of the overall foodborne risk of this illness to New Zealanders.

In addition there is a Risk Profile concerning *Salmonella* in cereals (Gilbert *et al.*, 2010b), prompted by an outbreak from *Salmonella* in flour in New Zealand. This Profile stated: "Overall, the risk of human salmonellosis due to contaminated cereal grains must be classified as low. However, the outbreak linked to flour indicates that when cereal contamination occurs it has the potential to affect large numbers of people, even if potential exposures occur via specialised behaviours (e.g. ingestion of uncooked home baking materials) or less common foods (e.g. uncooked muesli ingredients)."

For poultry, feed is a potential route for introduction of *Salmonella* into livestock. A Risk Profile addressing *Salmonella* in animal feed (Cressey *et al.*, 2011), found that "The fact that the most common *Salmonella* serotype in finished animal feed in New Zealand in recent years (*S.* Tennessee), based on industry data, occurs infrequently amongst human cases argues against animal feed as a major source of human salmonellosis in New Zealand. However, the available information on the *Salmonella* status of feed and feed ingredients in New Zealand is not sufficiently comprehensive to assess animal feed as a source of human salmonellosis cases."

Thus, the important food vehicles for salmonellosis in New Zealand remain elusive.

### 4.3 Data gaps

The 2004 Risk Profile did not specifically identify data gaps, but since 2004 a number of surveys have been completed that provided new data on the prevalence of *Salmonella* on poultry from processing plants and retail outlets (Section 7.4.2) and on packaging (Section 7.3.4). The data gaps identified in this Risk Profile are:

- Representative sampling and testing for *Salmonella* in broiler farm inputs (feed) and environment;
- Information on the impact of current processing practices in New Zealand on *Salmonella* prevalence and concentrations on poultry;
- Information on the concentration of salmonellae on poultry carcasses at the end of primary processing; and
- Transmission routes for the majority of salmonellosis cases in New Zealand.

A report was commissioned by the New Zealand Food Safety Authority to investigate the feasibility of using microbial subtyping approaches for attribution of human salmonellosis. A study has also been designed to undertake phenotyping and genotyping of collections of *Salmonella* isolates originating from humans, cattle, sheep, pigs, chickens (poultry meat and eggs) and wild birds. The distribution of *Salmonella* subtypes among human and animal sources will be analysed using recently developed source attribution models to estimate, with uncertainty, the proportion of human cases attributable to cattle, sheep, pigs, poultry and wild birds in New Zealand.

The source attribution models will incorporate the typing data generated by the study in conjunction with outbreak data, epidemiological data and expert opinion, in order to help identify food safety interventions that would lead to the reduction of *Salmonella* infection in the human population. This is a collaborative project between ESR and mEpiLab, Massey University and will commence in mid-2011 (Dr Eve Pleydell, Massey University, pers. comm., July 2011). It is anticipated that these projects will gain further information regarding the distribution of *Salmonella* subtypes in New Zealand and insight to the sources of infection which is currently unclear and is a major gap for risk assessments.

# 5 AVAILABILITY OF CONTROL MEASURES

## 5.1 Current Risk Management Measures

### 5.1.1 Legislation

#### *5.1.1.1 The Animal Products Act*

The *Animal Products Act 1999* regulates the processing of animal material into products for use, trade, and export through managing associated risks and facilitating overseas market access.[15]

The Act requires all animal products traded and used to be "fit for intended purpose". The main means for ensuring that animal products are fit for their intended purpose is by requiring that the production and processing of animal materials and products occurs under a registered risk management programme. Poultry processors must operate under a risk management programme, and Part 2 of the Act provides for the registration and verification of these risk management programmes (Section 5.1.2.1).

Part 3 of the Act provides for the setting of regulated control schemes where risk factors cannot be managed under risk management programmes, or where special provision is required for overseas market access. The Animal Products (Regulated Control Scheme – Contaminant Monitoring and Surveillance) Regulations 2004 provides for the monitoring of agricultural compounds, veterinary medicines and environmental contaminants in poultry.

Part 4 of the Act provides for the setting of standards that must be met before an animal product can be considered fit for intended purpose, and for the setting of any specifications necessary to ensure the standards are met. The New Zealand animal product standards are contained in the Animal Products Regulations 2000 (Section 5.1.1.2) and the Australia New Zealand Food Standards Code (Section 5.1.1.3).

While the Animal Products legislation is unlikely to impact the *Salmonella* loading entering the processing facility it does require hazard analysis procedures that will highlight processing steps that may increase or decrease pathogen loading and encourage good manufacturing practice, including suitable hygiene procedures.

#### *5.1.1.2 Animal Products Regulations*

The Animal Products Regulations 2000 set out animal product standards and provide for the setting of specifications.[16] Section 6(1) requires that, taking into consideration its intended use, animal products must be free from biological, chemical, and physical hazards in amounts that may be directly or indirectly harmful to humans or animals. However, specifications can be set regarding the unacceptable hazards in relation to any type of animal product (e.g. raw or ready-to-eat poultry), and the acceptable or unacceptable levels of these hazards (Section 6(2) of the regulations). Specifications for *Salmonella* on poultry products are set out in the Australia New Zealand Food Standards Code.

[15] The Act may be viewed at http://www.legislation.govt.nz (accessed 9 March 2011).
[16] The Regulations may be viewed at http://www.legislation.govt.nz (accessed 9 March 2011).

### 5.1.1.3 Australia New Zealand Food Standards Code

Chapters 1 and 2 of the Australia New Zealand Food Standards Code contain many requirements that are applicable to the poultry industry (e.g. requirements for labelling (Part 1.2 and Standard 2.2.1) and substances added to food (Part 1.3), limits for fluid loss (Standard 2.2.1)).[17] Standard 1.6.1 sets out the microbiological limits for specific food products. Limits have not been set for raw poultry, as this product will be cooked before consumption. Limits for *Salmonella* have been set for poultry products prepared using the following methods:

- Packaged cooked cured/salted meat;
- Packaged heat treated meat paste and packaged heat treated pâté;
- All comminuted fermented meat which has not been cooked during the production process.

For all of these products, detection of *Salmonella* in any of five 25 g samples of food from the same lot would render that lot unacceptable.

### 5.1.1.4 Animal Products Notices

The *Animal Products Act 1999* provides for the issuing of notices.[18] The Animal Products (Specifications for Products intended for Human Consumption) Notice 2004 applies to risk management programme operators who are processing animal material or animal product intended for human consumption, i.e. poultry primary processors. The Notice (and subsequent amendments) sets out requirements for the way these facilities should be designed and maintained, and how they should operate, including detail such as the maximum chilling (7°C) or freezing (-12°C) temperatures, water quality monitoring and transportation.[19] While the direct impact of the Notices on the *Salmonella* status of poultry processed in relevant facilities will be limited, some aspects (separation of material to be processed from material for human consumption, hygiene requirements and water quality requirements) may contribute to reducing the *Salmonella* burden of product from the facility.

### 5.1.1.5 Code of Welfare for fully housed broilers

The Animal Welfare (Broiler Chickens: Fully Housed) Code of Welfare 2003 was issued under the *Animal Welfare Act 1999* by the National Animal Welfare Advisory Committee (NAWAC). Under this Act, codes of welfare set by the NZWAC are deemed to be regulations and can contain minimum standards that have legal effect. Codes of welfare may also contain recommended practice and recommended best practice that are not legally binding.

[17] The Australia New Zealand Food Standards Code is available at http://www.foodstandards.gov.au/foodstandards/foodstandardscode.cfm (accessed 9 March 2011).
[18] All Notices can be viewed at http://www.foodsafety.govt.nz/industry/general/animal-products/documents/specs.htm (accessed 9 March 2011).
[19] The 2004 Notice and amendments can be viewed at http://www.foodsafety.govt.nz/elibrary/industry/animal-products-specifications-asd/index.htm (accessed 9 March 2011).

The Animal Welfare (Broiler Chickens: Fully Housed) Code of Welfare 2003 (NAWAC, 2003) applies to all persons responsible for the welfare of broiler chickens in controlled environment broiler production systems, i.e. the chickens are kept in enclosed housing and are reliant on human management for all their daily requirements. There are no specific standards for *Salmonella* species, but many of the standards will help control salmonellae by improving general biosecurity and reducing the potential for entry of salmonellae into broiler houses (e.g. all hatcheries must have a documented cleaning, sanitising and hygiene programme, housing systems must be vermin-proof, and other than in some exceptional circumstances litter must be replaced after every growing cycle).

### 5.1.2 Mandatory requirements

#### *5.1.2.1 Risk management programmes*

The *Animal Products Act 1999* defines a risk management programme (RMP) as a programme designed to identify and control, manage, and eliminate or minimise hazards and other risk factors in relation to the production and processing of animal material and animal products in order to ensure that the resulting animal product is fit for intended purpose. RMPs must manage risks from hazards to human health, animal health, false or misleading labelling and risks to the wholesomeness of animal material or product (NZFSA, 2009c).

A RMP is based on the principles of HACCP: Identifying the hazards, the systems of control, and demonstrating that the controls are effective. The Act requires that RMPs are tailored for each animal product business according to the animal materials used, the processes performed and the product range produced. Operators must build any relevant regulatory limits (e.g. microbiological limits) into their RMP, but can also set their own measurable limits to ensure the food is safe and fit for purpose.

Primary processors of poultry must have a RMP in place, and so must secondary processors of poultry unless they are covered by a food safety programme under the *Food Act 1981* and its subsequent amendments. Poultry producers (i.e. broiler farms) and transporters of poultry to primary processing facilities are not required to have a RMP (NZFSA, 2009c).

The operator of the primary or secondary processing facility is responsible for developing and registering their RMP but the programmes are subject to independent verification. A generic RMP for the slaughter and dressing of broilers was issued in 2002 to support operators to develop their own RMPs (NZFSA/PIANZ, 2002).[20] In this document, *Salmonella* is frequently used as an example of an identified hazard that requires control.

#### *5.1.2.2 National microbiological database (NMD) programme*

The NMD Programme is an industry programme that monitors animal carcass hygiene after processing by an aerobic plate count and tests for *Salmonella* species, *Campylobacter* species and *E. coli*. Premises operating to process broiler chickens must have the NMD programme in place. Further details and recent results are presented in Section 7.4.1.

[20] The generic RMP is available at http://www.foodsafety.govt.nz/industry/general/rmp/documents/rmp-generic/ (accessed 9 March 2011).

### 5.1.3 Non-mandatory guidelines and codes of practice

#### *5.1.3.1 Ministry of Health criteria (1995)*

The New Zealand Ministry of Health has published microbiological criteria for foods intended as a guide for food producers where no mandatory standard exists (MoH, 1995). There are microbiological criteria for *Salmonella* in poultry products and for generic categories of foods that will include poultry products. The criteria for *Salmonella* are listed in Table 5.

**Table 5: Ministry of health microbiological reference criteria applicable to *Salmonella* in poultry products**

| **Product** | | ***Salmonella* per:** | **Criteria*** | | |
|---|---|---|---|---|---|
| | | | **n** | **c** | **m** |
| Poultry | Raw | 25 g or whole bird carcass rinse | 5 | 1 | 0 |
| | Nuggets, patties, etc requiring further cooking (> 70°C) | 25 g | 5 | 0 | 0 |
| | Cooked | 25 g | 5 | 0 | 0 |
| | Cured and/or smoked | 25 g | 5 | 0 | 0 |
| Meat and meat products | Chopped, minced or manufactured meat – uncooked | 25 g | 5 | 1 | 0 |
| | Corned, cured, pickled or salted – uncooked | 25 g | 5 | 1 | 0 |
| | Manufactured, cured or fermented meat - ready-to-eat | 25 g | 5 | 0 | 0 |
| | Meat paste or spread - including pâté | 25 g | 5 | 0 | 0 |
| | Hot smoked | 25 g | 5 | 0 | 0 |
| | Vacuum packed - semi-preserved but perishable products | 25 g | 5 | 0 | 0 |
| Foods – cooked, ready-to-eat (or with subsequent minimal heating < 70°C) | All components cooked in manufacturing process | 25 g | 5 | 0 | 0 |
| | Some components not cooked in manufacturing process (e.g. sandwiches) | 25 g | 5 | 0 | 0 |
| Foods – requiring further cooking (> 70°C) | | 25 g | 5 | 0 | 0 |

* n = the minimum number of sample units that must be examined from a lot of food; c = the maximum allowable number of defective sample units; m = the acceptable microbiological level in a sample unit (values above it are marginally acceptable or unacceptable).

### *5.1.3.2 FSANZ guidelines (2001)*

FSANZ has produced generic guidelines for the microbiological examination of ready-to-eat foods that apply to foods sampled at the point of sale or distribution to consumers (FSANZ, 2001). Under these guidelines, *Salmonella* species should not be detectable in any ready-to-eat food.

### *5.1.3.3 NZFSA/MAF Code of Practice for poultry processors*

MAF, in consultation with PIANZ, is developing a Code of Practice for poultry primary processors. The purpose of the Code of Practice is to help poultry processors meet the requirements of the Animal Products Act 1999 (Section 5.1.1.1) and risk management programmes (Section 5.1.2.1), and to produce poultry products for human and animal consumption that are safe and suitable for their purpose (NZFSA, 2007).

MAF (then NZFSA) began development of the Code of Practice in 2007 and has released four chapters.[21] Once complete, the code of practice will cover good manufacturing practice and process control, HACCP application, and the identification and control of risk factors related to wholesomeness and labelling. The code of practice will then replace the 1998 Poultry Industry Processing Standard 5 (PIPS 5), published by the Poultry Industry Standards Committee.[22] PIPS 5 sets the minimum standards for producers of poultry products for human consumption with the aim of minimising the potential food safety hazards associated with poultry, based on HACCP principals. PIPS 5 is still used as a guideline for aspects not yet covered by the Code of Practice (Michael Brooks (Executive Director, PIANZ), pers. comm., 23 May 2011).

The Code of Practice does not specifically address *Salmonella* in poultry products. However, many of the good manufacturing practices will help to reduce any *Salmonella* contamination on poultry carcasses, and prevent cross-contamination (e.g. temperature controls, wash steps, equipment cleaning and maintenance).

### *5.1.3.4 Broiler Growing Biosecurity Manual*

The Broiler Growing Biosecurity Manual describes the recommended minimum standards to be used in New Zealand's broiler production systems (PIANZ, 2007). One of the Manual's biosecurity objectives is to minimise the incidence and spread of organisms of public health concern, citing salmonellae as an example of such an organism. The Manual covers the set-up and operation of production facilities, management of personnel, and controls over inputs such as water and feed or potential routes of contamination such as vehicles and wildlife. These practices will help to control *Salmonella* species contamination during poultry production. Each poultry company has its own biosecurity manual and these incorporate aspects of the Broiler Growing Biosecurity Manual (Michael Brooks (Executive Director, PIANZ), pers. comm., 23 May 2011).

[21] The code of practice is available at http://www.foodsafety.govt.nz/elibrary/industry/processing-code-practice-poultry/index.htm (accessed 9 March 2011).
[22] PIPS5 is available from http://www.foodsafety.govt.nz/industry/sectors/meat-ostrich-emu-game/meatman/pips5/ (accessed 9 March 2011).

### *5.1.3.5 Poultry industry agreed standards and codes of practice*

The 1995 Poultry Industry Agreed Standards and Codes of Practice (PIANZ, 1995) have been superseded by the Broiler Growing Biosecurity Manual, the MAF Code of Practice and RMPs (Michael Brooks (Executive Director, PIANZ), pers. comm., 23 May 2011).

### *5.1.3.6 Antibiotic use in poultry production*

There are mainly three uses for antibiotics in animal welfare:

- Therapeutic purposes;
- Prophylactic purposes; and
- Growth promotion or growth/feed conversion.

Usually antibiotics are therapeutic in nature and are administered for a limited time to kill off the causative agent during disease manifestation. Prophylactic use of antibiotics is where the drug is administered for a limited period, when the risk of a specific disease is greatest. Growth promotion or growth/feed conversion is where an antibiotic is administered in low concentrations (lower than for prophylactic use) in feed to stimulate the animals' growth resulting in increased daily live weight gain and/or feed conversion efficiency. It is essentially a method of health maintenance in a population whereby if the animals or birds are healthy, then all feed consumed is efficiently converted to meat production.

The 2004 Risk Profile reported on two antibiotics commonly used during poultry production:

- Ionophores: Used prophylactically in feed or water to control coccidiosis.
- Zinc bacitracin: Used prophylactically in feed or water to control necrotic enteritis.

Neither of these antibiotics have an effect on *Salmonella* species or are used to treat humans in New Zealand (Lake *et al.*, 2004; Teirlynck *et al.*, 2009).

Antibiotic use in animals in New Zealand is controlled by the MAF Agricultural Compounds and Veterinary Medicines (ACVM) Group. The use of antibiotics and the potential promotion of antibiotic resistance in bacteria pathogenic to humans are subject to regular review. NZFSA established an antimicrobial resistance steering group in 2004. Two activities driven by the steering group are relevant to this Risk Profile:

- A year-long baseline survey for antimicrobial resistance of animal bacteria commenced in October 2009 as part of developing a national surveillance programme. The survey includes sampling from freshly dressed broiler carcasses and analysing for antimicrobial resistance among isolated salmonellae.
- MAF has developed a new database to provide an annual summary of statistics on sales of antimicrobial veterinary medicines that will include how the medicines were used. The latest available results are for 2006/07, and these showed that zinc bacitracin represented 36% of all antibiotics sold (by weight), and 94% of antibiotic usage in the pig and poultry category (mostly used for poultry and administered with feed).

PIANZ has published guidelines for the use of antibiotics in poultry (PIANZ, 2011). The guidelines advocate the use of antibiotics when there is evidence that:

- use is consistent with accepted veterinary practice;
- use is linked to a specific etiologic microbiological agent or disease syndrome;
- use is appropriately targeted in poultry;
- no reasonable alternatives for intervention exist.

The guidelines also advocate that antimicrobials of critical importance to human health are not used in poultry.

The New Zealand Veterinary Association has also published specific prescribing guidelines for veterinarians working the poultry industry (NZVA, 2006). The guidelines advocate that veterinarians must demonstrate the flocks are under his or her care before prescribing vaccines for disease prevention. The guidelines also advocate that livestock companies employing or contracting a veterinarian have a documented Whole Flock Health Scheme (as required by their RMPs) or a documented quality system that includes control of prescription animal remedies.

#### *5.1.3.7 Vaccination programme*

The New Zealand poultry industry routinely uses the vaccine "Megan®Vac-1" in layer and breeder flocks. The vaccine protects against *S.* Typhimurium and also reduces the likelihood of infection with *S.* Enteritidis, but is only sporadically used in broiler flocks.

#### *5.1.3.8 Labelling for chicken nuggets*

Consumers are may be exposed to *Salmonella* species if they consume chicken nuggets, or similar partially cooked poultry products, that have not been adequately cooked in the home. Consumers potentially perceive that these types of product are already cooked and may consume them without further cooking or after reheating (e.g. microwaved) rather than following the manufacturer's instructions for proper cooking. Pictures on the label showing a product that appears to be already cooked may reinforce this perception. Investigations of outbreaks of salmonellosis in the United States due to raw, ''flash fried,'' or ''par-fried'' chicken nuggets or strips indicated that inadequate labelling of the implicated chicken products, consumer responses to labelling and microwave cooking were primary factors contributing to these outbreaks (Smith *et al.*, 2008).

The 2004 risk assessment commented that current risk management arises from the labelling practices of some of the New Zealand manufacturers, including a clear statement that "this product is not cooked" in bold print on the label, as well as suitable cooking instructions (Wong and Lake, 2004).

Options to further reduce the risk to consumers would be for all manufacturers to:

- label their product as not cooked.
- cook their product fully (there appears to be the potential for confusion if only some manufacturers produce a fully cooked product).

In the absence of cases of salmonellosis linked to chicken nuggets in New Zealand, as well as the low level of *Salmonella* in New Zealand poultry generally, the first option would appear to be the most commensurate with the risk.

#### 5.1.4 Campylobacteriosis interventions

A range of regulator and industry interventions and activities were introduced from 2006 through 2008 with the aim of reducing poultry-associated foodborne campylobacteriosis in New Zealand (Sears *et al.*, 2011). Many of these interventions should also control *Salmonella* species in poultry including improving poultry transportation procedures, processing interventions to reduce levels of *Campylobacter* species on broilers at completion of primary processing, or using leak-proof packaging for consumer packs. However, the incidence of reported salmonellosis did not decline over the same period, and this was attributed to the low prevalence of contamination prior to the interventions.

#### 5.1.5 Control measures in other countries

These have been summarised in Appendix 3.

### 5.2 Options for Risk Management

New Zealand is fortunate in having a poultry industry in which types of *Salmonella* that have caused major problems overseas (*S.* Enteritidis PT4 and *S.* Typhimurium DT104) are not endemic. Import controls on poultry are partially designed to maintain this status. New Zealand cases of human illness caused by these types of bacterial infections appear to be principally acquired overseas.

The current low prevalence of contamination by *Salmonella* in poultry is evidence of good risk management by the New Zealand poultry industry. Despite this control, occasional spikes in *Salmonella* prevalence can occur, as shown by the incident in Canterbury in 2003 (Wong, 2003).

This event indicates that *Salmonella* control efforts should be maintained, supported by monitoring of feed, environmental, and processing line samples.

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# 7 APPENDIX 1: HAZARD AND FOOD

The information contained in this Risk Profile is current to the date of publication. Please be aware that new information on the subject may have arisen since the document was finalised.

## 7.1 *Salmonella* species

### 7.1.1 Typing methods

#### *7.1.1.1 Serotyping*

*Salmonella* serotypes are identified by observing the agglutination of a suite of *Salmonella*-specific antibodies with antigens on the bacterial surface. This is known as the Kauffmann-White scheme. The antigenic formulae of *Salmonella* serotypes are defined and maintained by the World Health Organization (WHO) Collaborating Centre for Reference and Research on *Salmonella*, at the Pasteur Institute in Paris (Brenner *et al.*, 2000; Grimont and Weill, 2007).

Somatic (O) antigens are present on the external surface of the bacterial outer membrane (D'Aoust and Maurer, 2007). The O-antigens can be described as smooth (S), where they are well developed and readily agglutinate with specific antibodies, or rough (R) if the antigens are incomplete and exhibit weak or no agglutination with the O-antibodies.

The flagellar (H) antigen is associated with the flagellin, which is a major component of the flagellar (EFSA Panel on Biological Hazards (BIOHAZ), 2010). *Salmonella* strains can have the ability to express two different compositions of the flagellar antigen, called phase 1 and phase 2, and these strains are described as diphasic (sometimes biphasic). Others only produce one composition (monophasic), and variants producing three (triphasic) or more compositions have been identified.

The Vi antigen is the only capsular (K) antigen detected in *Salmonella* serology, and is only produced by *S.* Typhi, *S.* Paratyphi C and *S.* Dublin (EFSA Panel on Biological Hazards (BIOHAZ), 2010).

The serology is expressed as an alphanumeric code that reads as: O-antigens: H-antigens of first phase: H-antigens of second phase (EFSA Panel on Biological Hazards (BIOHAZ), 2010). As an example, *S.* Typhimurium is denoted 1,4,[5],12:i:1,2. The O-antigens are 1, 4, 5 and 12. Both O-1 and O-5 may be present or absent in strains; underlining means that the factor was determined by a method called phage conversion and square brackets means that the antigen may be present or absent without any relation to phage conversion. The 'i' is a phase 1 H-antigen, and H-1 and H-2 are phase 2 H-antigens. A hyphen is used to indicate that an antigen is absent, for example several *S.* Typhimurium-like strains have been described which lack some of the H-antigens, e.g. 1,4,[5],12:i:- or 1,4,[5],12:-:1,2 (EFSA Panel on Biological Hazards (BIOHAZ), 2010).

The antigenic formula of some *Salmonella* serotypes that have been commonly isolated in New Zealand are as follows (Grimont and Weill, 2007):

- Enteritidis 1,9,12:g,m:-

- Brandenburg 4,[5]12:l,v:en,$z_{15}$
- Infantis 6,7,14:r:1,5
- Saintpaul 1,4,[5],12:e,h:1,2
- Heidelberg 1,4,[5],12:r:1,2
- Virchow 6,7,14:r:1,2

#### *7.1.1.2 Phage typing*

Once the serotype is identified, a *Salmonella* isolate can be further subtyped by measuring susceptibility to a panel of bacteriophages. Separate bacteriophage panels have been developed for different serotypes, and the ESR ERL in New Zealand routinely determines the phage types of any *S.* Typhimurium, *S.* Enteritidis or *S.* Typhi isolates they receive. The *S.* Typhimurium phage typing method involves testing the ability of 29 bacteriophages to lyse an isolate and is able to distinguish 235 phage types (Anderson *et al.*, 1977; Callow, 1959). Phage typing for *S.* Enteritidis uses 10 different bacteriophages (Ward *et al.*, 1987), and 33 bacteriophages are used to phage type *S.* Typhi isolates (Anderson and Williams, 1956).

#### *7.1.1.3 Molecular methods*

Pulsed-field gel electrophoresis (PFGE) is a common molecular method that is able to further distinguish *Salmonella* species on the basis of their DNA. In New Zealand this technique is usually only applied during cluster or outbreak investigations where it is used to determine whether salmonellosis cases had become ill with the same strain of *Salmonella* and to help link these cases with a source of infection. If PFGE does not adequately discriminate *S.* Typhimurium, another molecular-based test called multiple-locus variable-number tandem repeat analysis (MLVA) can be used.

#### *7.1.1.4 Antimicrobial susceptibility*

New Zealand hospital and community laboratories are requested to refer all *Salmonella* isolates from human salmonellosis cases to ESR for typing. ESR also receives *Salmonella* isolates from other sources, including food, animal and environmental sources. Approximately 20% of the non-typhoidal *Salmonella* isolates received by ESR are tested for antimicrobial susceptibility, along with all *S.* Typhimurium phage types that are internationally recognised as being multiresistant. These clones include *S.* Typhimurium phage types DT104, U302, DT12, DT120 and DT193 (ESR, 2010b).

ESR tests susceptibility to 12 antimicrobials: ampicillin, cephalothin, chloramphenicol, ciprofloxacin, co-amoxiclav, co-trimoxazole, gentamicin, nalidixic acid, streptomycin, sulphonamides, tetracycline and trimethoprim. All cephalothin-resistant isolates are further tested for the production of extended-spectrum β-lactamase (ESBL) and plasmid-mediated AmpC β-lactamase (ESR, 2010b). The 2005-2009 results from this antimicrobial testing programme are summarised in Section 8.2.5.

### 7.1.2 Growth and survival

The following information is taken from a number of different sources but, unless otherwise referenced, is primarily derived from a data sheet prepared by ESR under a contract for the

Ministry of Health in 2000-2001. The data sheets are located on the NZFSA website.[23] They are intended for use by regional public health units and will be updated from time to time.

#### *7.1.2.1 Growth*

Temperature: Minimum 7°C, growth greatly reduced at <15°C. Maximum 49.5°C. Optimum 35-37°C. Some evidence for growth at temperatures <7°C exists, but this is serotype specific, the data are still not universally accepted and doubts surrounding the experimentation exist.

pH: Minimum 3.8, optimum, 7-7.5, maximum 9.5. The minimum pH is influenced by other factors such as temperature, acid present, and the presence of nitrite etc.

Atmosphere: Can grow in the presence or absence of air as a facultative anaerobe. The growth rate on beef muscle stored at 20°C under nitrogen is only slightly less than that obtained when stored under air (Grau, 1983). At high concentrations of $CO_2$ (50-60%), growth is strongly inhibited on beef steak and minced beef at 10-11°C, but at 20°C there is little inhibition (Luiten *et al.*, 1982; Silliker and Wolfe, 1980).

Water activity: Minimum 0.94, optimum 0.99, maximum >0.99.

#### *7.1.2.2 Survival*

*Salmonella* are known to survive well in foods and on surfaces. Survival is particularly good in foods with low water activity e.g. flour.

Temperature: *Salmonella* can survive well in foods for long periods at low refrigeration temperatures. In frozen foods, although *Salmonella* numbers are considerably reduced, some survive for long periods. Some foods, including meat, ice-cream and butter, appear to be protective of *Salmonella* during freezing and frozen storage. Rapid freezing promotes survival with lower frozen storage temperatures and less fluctuation giving greater survival (Jay *et al.*, 2003).

Frozen storage temperatures near 0°C result in greater death or injury to bacterial cells. 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% of cells respectively were still viable after 5 days (Jay *et al.*, 2003).

pH: *Salmonella* appear to be significantly less tolerant of low pH (pH 2.5; hydrochloric acid) than *Shigella* species or *Escherichia coli*. These last two organisms possess additional acid survival systems that are not present in salmonellae (Gorden and Small, 1993; Lin *et al.*, 1995).

Water Activity: Survival in dry environments is a characteristic of these organisms. For example, they can survive in bitter chocolate ($a_w$ 0.3-0.5) for months. Exposure to low $a_w$ environments can greatly increase the heat resistance of these organisms.

[23] See http://www.foodsafety.govt.nz/science/other-documents/data-sheets/ (accessed January 2011). ESR originally prepared the data sheets for the Ministry of Health in 2001.

### 7.1.3 Inactivation

Note that in microbiological terms "D" refers to a 90% (a decimal or 1 $\log_{10}$ cycle) reduction in the number of organisms.

Temperature: Inactivation is greater during the freezing process rather than subsequent frozen storage, but those cells that survive remain viable. Freezing does not ensure the inactivation of salmonellae in foods.

D times with heat treatment: At 60°C usually 2-6 min; at 70°C usually 1 min or less. Some rare serotypes (e.g. *S.* Senftenberg) are significantly more heat resistant than the others, but this organism is not considered to be important as a food pathogen (Doyle and Mazzotta, 2000).

D times for *Salmonella* species can depend on the type of food involved. Long D times have been reported for experiments with *Salmonella* Typhimurium in milk chocolate. Values reported were up to 1,050 min at 70°C, 222 min at 80°C and 78 min at 90°C (Goepfert and Biggie, 1968).

pH: Low pH values and the nature of the acidulant determines the rate of death. Temperature is also a factor.

In the studies by Alford and Palumbo, the authors demonstrated how decreasing temperature increases the inhibitory effects of pH and NaCl. In broth, at 10°C, growth of 22/23 strains were inhibited by pH 5 and 2% NaCl (Alford and Palumbo, 1969). At pH 5.8 (more representative of meat), 5% NaCl at 10°C was required to inhibit growth. Increasing the salt concentration slightly decreased survival time at 10°C.

Water activity: At $a_w$ levels below those allowing growth, salmonellae die slowly. The rate of death decreases as the $a_w$ is lowered and also decreases as the temperature is reduced (Troller and Christian, 1978).

Radiation: A mixture of six strains of *Salmonella* Typhimurium DT104 was inoculated into three ground pork products (of varying fat content) (Rajkowski *et al.*, 2006). The amount of beta radiation to achieve a 90% reduction was around 0.43 kGy regardless of fat content.

Disinfectants: A number of disinfectants have been shown to reduce the prevalence or concentration of *Salmonella* on poultry (see Section 7.3.2.2).

## 7.2 Poultry Exports

In the year to March 2011 New Zealand exported 3,255 tonnes of chicken products (including offals), 8 tonnes of turkeys and products, and 0.1 tonnes of ducks and duck products.[24] The following seven countries received over 100 tonnes of poultry products:

- Australia: 1,265 tonnes;

[24] Data are sourced from Statistics New Zealand's Infoshare resource (http://www.stats.govt.nz/infoshare/) using the Harmonised Trade codes.

- Fiji: 1,212 tonnes;
- Papua New Guinea: 522 tonnes;
- Cook Islands: 247 tonnes;
- Vanuatu: 208 tonnes;
- Hong Kong: 170 tonnes; and
- French Polynesia: 144 tonnes.

Other important export markets during 2011 included Samoa, Tonga, Niue, Solomon Islands, Norfolk Island, Tuvalu and New Caledonia (all received >1 tonne of poultry products). Prior to 2011, Mozambique and the Philippines were also important export markets.

## 7.3 Behaviour of *Salmonella* on Poultry

### 7.3.1 Poultry farming (primary production)

*Salmonella* species initially colonise the intestinal tract of poultry where they can persist throughout the bird's lifespan in a poultry production environment (Gast, 2003). Colonisation of poultry is usually asymptomatic, but colonisation can lead to illness and death in young birds. Systemic infection (infection of internal organs) has been well studied for *S.* Enteritidis (which invades the ovary and oviduct), but *S.* Typhimurium, *S.* Infantis and *S.* Heidelberg are also known to be invasive for poultry. Avian serotypes such as *S.* Pullorum and *S.* Gallinarum are serious infections for poultry and, while some rare human cases have been reported, these serotypes are usually host-specific and of no major concern for human salmonellosis (Shivaprasad, 2003). Neither of these poultry serotypes have been detected in New Zealand since 1985 (Davidson, 2022).

Poultry shed salmonellae with faeces, and the concentration of these bacteria can be up to $10^4$ CFU/g of gut content or faeces (FAO/WHO, 2002).

#### *7.3.1.1 Organic or free range vs. conventional primary production*

Free range production means that poultry have access to areas of the farm outside the poultry house. Organic production means that poultry are raised according to the principles of organic production. Some aspects of free range or organic production could be expected to increase the risk of poultry becoming contaminated with *Salmonella* species, such as access to the outdoors and mixed farming with other animals. However, some aspects of organic or free range production might help to reduce *Salmonella* prevalence in poultry. In a survey of 60 flocks from 34 free-range broiler farms in Northern Spain (Esteban *et al.*, 2008), *Salmonella* species were only isolated from one flock on one farm. The authors speculated that lack of stress could contribute to a reduction in shedding rates, lower bird densities could hamper faecal-oral transmission, and that the higher age of birds at slaughter would enable the birds to develop a mucosal immune response reducing *Salmonella* infection.

Some researchers have investigated whether *Salmonella* contamination is different for poultry produced under organic or "conventional" (not organic) conditions. After a systematic literature review and meta-analysis, seven studies were identified that compared the prevalence of *Salmonella* species from conventionally or organically produced poultry on farms or at slaughter using faecal, caecal and environmental samples, or from retail samples (Young *et al.*, 2009). Only one of these studies reported a statistically significant difference

between the production methods; these authors found that *Salmonella* prevalence was significantly higher in conventionally-raised broilers when compared with pasture-raised broilers (Siemon *et al.*, 2007). Young *et al*. (2009) also identified two studies comparing the antimicrobial resistance of *Salmonella* species isolated from poultry samples from conventional production systems with isolates from organic production systems. Only one of these studies (also Siemon *et al*., 2007) reported a statistically significant difference; isolates from conventional broiler chickens had a significantly higher multidrug resistance (resistance to three or more classes of antimicrobials). Young *et al.* (2009) concluded that the research available was inconsistent and limited, and there was a need for future research of sufficient quality in this area.

In another study, researchers tested 141 conventional and 53 organic chicken carcasses purchased from retail stores in Louisiana, USA, during 2006-07 (Lestari *et al.*, 2009). *Salmonella* species were isolated from 22.0% of conventional and from 20.8% of organic chicken samples. The predominant *Salmonella* serotypes recovered from both conventional and organic chickens were Kentucky, Hadar, and Enteritidis. The researchers also found that *Salmonella* isolates from both sample types demonstrated antibiotic resistance, and concluded that organic production did not guarantee an absence of antimicrobial resistance (e.g. *S.* Kentucky isolates from organic chicken samples were susceptible to 11 of the antimicrobials tested, whereas those from conventional chickens were only susceptible to four).

#### *7.3.1.2 Salmonella reduction during primary production*

The contamination and transmission of *Salmonella* to poultry can be reduced by a variety of on farm measures, and there is a large volume of literature evaluating different intervention methods.

The preventative and curative strategies for reducing the incidence of *Salmonella* colonisation in broiler chickens at farm level include (Vandeplas *et al.*, 2010):

- Biosecurity: Minimising the risk of introducing *Salmonella* into a flock by ensuring the flock, house, feed, litter and water are kept *Salmonella*-free;
- Vaccination with dead or attenuated live *Salmonella* strains; and
- Antibiotics such as fluoroquinolones, trimethoprim and polymyxin B (NB: antibiotic-use for growth promotion has been banned in the EU since January 2006).

Emerging strategies include (Vandeplas *et al.*, 2010):

- Passive immunity of birds that have been fed specific antibodies produced from eggs of hyperimmunised hens;
- Feed additives or modification of feed to reduce host susceptibility to colonisation, e.g. whole wheat, β-glucans, alfalfa, enzymes, probiotics, prebiotics;
- Genetically resistant chicken lines;
- Acidification of feed and drinking water with short- and medium-chain fatty acids (e.g. lactic acid, formic acid, caprylic acid) added to the matrix or produced or by fermentation; and,
- Bacteriophages.

#### *7.3.1.3 Transportation prior to slaughter*

Several studies have shown transportation of poultry increases *Salmonella* prevalence among poultry and that transport cages are an important source of cross-contamination (CCFH, 2007). During transport, birds are often stored in open crates that are placed on top of each other and the stress of transport increases faecal excretion, so the possibility of cross-contamination is increased (FAO/WHO, 2002). Minimising bird stress and withholding feed (but not water) for 4-10 hours prior to slaughter (including catching and transportation time) reduces contamination without significantly affecting carcass weight (NZFSA, 2009a). Washing the transport cages with water and leaving them to dry for 48 hours reduces the levels of residual *Salmonella* species found in transport cages (CCFH, 2007).

Birds being held prior to slaughter may have fans to cool them. In experiments with turkeys, *Salmonella* was transmitted from caged contaminated to caged uncontaminated birds within two hours when a fan was introduced (Harbaugh *et al.*, 2006). The authors hypothesised that dust was an important route of infection.

### 7.3.2 Poultry killing and processing (primary processing)

There are two main sources of *Salmonella* contamination in the processing plant: the birds themselves and cross-contamination from other birds or the environment (FSANZ, 2005). FSANZ has summarised the effect of processing on *Salmonella* contamination on chicken carcasses (FSANZ, 2005), drawing from the FAO/WHO risk assessment of *Salmonella* on broiler chickens (FAO/WHO, 2002) (Table 6).

The stun and slaughter step is unlikely to change the levels of contamination (FSANZ, 2005).

Scalding facilitates the removal of feathers but scalding temperatures differ for different poultry species depending on the difficulty in removing feathers and the end market. For example, in Australia, the temperature of the scald water is 50-52°C for birds for the fresh poultry meat market and 58°C for birds for the frozen poultry meat market (FSANZ, 2005). In New Zealand, a scald temperature of 56-58°C is standard (Lake *et al.*, 2007b). Scald water washes salmonellae from the external surfaces of birds and can transfer these bacteria to other birds. Most studies (summarised by FAO/WHO, 2002) show little reduction in *Salmonella* species prevalence after scalding, and it has been proposed that the acid conditions that can develop in scald water may act to increase the heat resistance of *Salmonella* (Humphrey and Lanning, 1987).

Defeathering is carried out by machines that remove the loosened feathers from the carcass and this step is considered to be a major source of cross-contamination. *Salmonella* species can become trapped in cracks and/or joins of the rubber fingers of the machines and cross-contamination can also occur via aerosols (FSANZ, 2005).

During evisceration the crop, gut and other internal organs are removed manually or mechanically. Poorly controlled evisceration (e.g. untrained workers, poor equipment maintenance and calibration, line speed too fast) will result in contamination of the carcass and equipment via rupture of the intestines (FSANZ, 2005). Most studies show a 2-5 fold increase in the prevalence of *Salmonella* species after evisceration, although one study in the US showed little effect of evisceration (summarised by FAO/WHO, 2002).

**Table 6: Effect of processing stage on *Salmonella* contamination (from FSANZ, 2005)**

| Process stage | Comments | Effect on *Salmonella* contamination | | |
|---|---|---|---|---|
| | | Reduce | Minimal | Increase |
| Stun/kill | | | ✓ | |
| Scald (low temperature)[1] | Survival of *Salmonella* in scald water (cross contamination) | | | ✓ |
| Scald (high temperature)[1] | Kill step (depending on temperature) | ✓ | | |
| De-feathering | Cross-contamination | | | ✓ |
| Effective washing | Physical removal of bacteria | ✓ | | |
| Evisceration | Contamination with faeces, main source of carcass contamination | | | ✓ |
| Effective washing | Physical removal of bacteria | ✓ | | |
| Chilling – immersion (suboptimal operation) | Cross-contamination | | | ✓ |
| Chilling – immersion (effective operation) | Requires constant monitoring of water temperature, flow rates and chlorine levels | | ✓ | |
| Chilling – air | Slight reduction due to desiccation of the carcass surface | | ✓ | |
| Portioning | Possible growth/cross contamination | | | ✓ |

[1] Low temperature scalding is carried out at 50-52ºC.High temperature scalding refers to processes performed at about 58ºC

Carcasses are washed after defeathering and evisceration. While washing will remove some salmonellae from the carcass, these bacteria can be trapped within the skin and feather follicles and cross-contamination can occur via the wash water. In Australia, the wash water temperature must be no more than 18°C, and for immersion washing, carcasses cannot remain in the tank for more than 15 min, unless the water temperature is <4°C (FSANZ, 2005).

Chilling the carcasses to <4°C as quickly as possible limits the opportunity for microorganisms to grow. Chilling methods include air-chilling, water immersion (e.g. spin chiller) and spray chilling (FSANZ, 2005). Immersion chilling is common in Australia and standard in New Zealand. Cross-contamination may occur where immersion chilling is used.

The portioning and packaging steps present the opportunity for cross-contamination from knives, surfaces and hands. There is also the opportunity for *Salmonella* species to grow if the carcass or air temperature becomes favourable for long enough (see Section 7.3.3).

Aerosols are also a source of contamination throughout the processing line. In a study of the microbial composition of the air in various areas of a high-throughput chicken slaughtering facility, researchers found the highest counts of microorganisms (including *Salmonella*

species) in the initial stages of processing (the receiving/killing and defeathering areas) (Lues *et al.*, 2007).

The prevalence of *Salmonella* species may be higher on poultry carcasses at the end of primary processing than at the start. For example, the prevalence of *Salmonella* species in six turkey flocks prior to processing was 0.6% (1/160 birds sampled) but after final chilling the prevalence was 36.3% (58/160 birds) (Trampel *et al.*, 2000). *Salmonella* was detected in the scald water, chill water and equipment in this slaughterhouse.

#### *7.3.2.1 Salmonella reduction during primary processing*

The contamination and transmission of *Salmonella* to poultry during primary production can be reduced by a variety of measures, and there is a large volume of literature evaluating different intervention methods. MAF recommends good manufacturing practices as part of their code of practice for the processing of poultry (NZFSA, 2009a):

- Scalding: Temperature control of the scald tank is important as low temperatures result in inadequate removal of feathers and increased survival of bacteria, whereas high temperatures damage the epidermis and may result in undesirable appearance. Sufficient contact time is also important for good feather removal. There are two commonly used scalding procedures: Hard scald (sub-scald), 55-60°C, and soft scald (semi-scald), 50-54.5°C. Scalding tanks should be set up as a counterflow system.
- Plucking: Recommend that an antimicrobial is used in at least the last half of the plucker. Collection and removal of feathers from the defeathering and scalding areas must be carried out at a frequency and in a manner that minimises build-up of feathers and contamination of the produce or processing areas. Defeathering is considered a "dirty" activity and should be physically separated as much as possible from later primary processing activities.
- After defeathering: All birds must be rinsed by a constant spray of potable water before any incision is made.
- Vent opening: There must be continuous sprays to rinse the equipment, vent area and rear of the bird and an antimicrobial chemical should be added to the rinse water. Knives or equipment used for venting must not be used for cutting any other part of the carcass.
- Evisceration: There must be continuous sprays to rinse equipment and the bird – an antimicrobial chemical should be added to the rinse water.
- Carcass rinse: After evisceration, all carcasses must be rinsed in running potable water with at least 0.5 L per bird and/or a processing aid to remove any remaining visible contamination (sprays should ensure thorough rinsing of inside and outside of carcass). Excess water should be removed prior to any air chilling.
- Chilling: Immersion and/or air chilling must deliver a product at 10°C or less before the product leaves primary processing (a pre-chill tank using ambient pH corrected water with a maximum of 200 ppm total chlorine is sometimes used). For immersion chillers, operators should aim for 3-5 ppm free available chlorine where the water exits the final tank. Excess water should be removed.

Where offal is to be recovered for human consumption, it is recommended that the offal is removed and handled in a way that minimises contamination and is rinsed using potable

water with an antimicrobial before or during chilling, with continuous cooling to 7°C or colder within four hours of removal (NZFSA, 2009a).

Another strategy to reduce the risk of contaminated poultry meat is slaughtering *Salmonella*-positive flocks at the end of the week or day, followed by intensified cleaning and disinfection (CCFH, 2007). Meat from infected flocks could also be channelled into food pathways that will involve a bactericidal treatment (e.g. cooking) prior to reaching the consumer.

#### *7.3.2.2 The use of disinfectants during primary processing*

A number of disinfectants have been shown to reduce the prevalence of *Salmonella*-positive chicken carcasses during primary processing (CCFH, 2010). For example, spray applications of 20-50 ppm chlorinated water or immersion in trisodium phosphate (TSP) following defeathering and carcass evisceration have been shown to reduce *Salmonella* prevalence. Acidified sodium chlorite (ASC) has also been shown to reduce *Salmonella* prevalence on chicken carcasses, e.g. spraying carcasses with ASC (250ppm, pH 2.5) reduced the prevalence from 50% to levels below detection.

The effectiveness of various physical and chemical decontamination treatments for poultry carcasses have been recently summarised (Loretz *et al.*, 2010). Some of the data specific to *Salmonella* have been summarised in Table 7. Steam and TSP appear to be the most effective, but due to different methods and reporting the results are difficult to compare. Modest reductions in the concentration of *Salmonella* on poultry have also been achieved through applying pressurised water, ozonated water, ultrasound, air chilling, sodium hydroxide, chlorine-based treatments (e.g. chlorine dioxide, sodium hypochlorite), phosphate-based treatments (e.g. sodium acid pyrophosphate, monosodium phosphate) and grapefruit seed extract (Loretz *et al.*, 2010). Chlorine is often added to the water to control pathogens, although it is rapidly inactivated by organic material. Chlorine is most commonly used in New Zealand. A number of researchers have also reported the effectiveness of multiple hurdles (combining two or more different treatments) (see Loretz *et al.*, 2010).

**Table 7: Studies of the effectiveness of hot water, steam and chemical treatments in reducing *Salmonella* inoculated onto poultry samples, as summarised by Loretz *et al*. (2010)***

| Treatment | Temperature, time | Sample | Inoculation | Reduction |
|---|---|---|---|---|
| Hot water spray | 21-54°C, 0.1 min | Carcass | *Salmonella* spp. | 0.7-1.2 log CFU/ml |
| Steam | 100°C, 1 min | Breast (retail) | *S.* Typhimurium | 6.2 log CFU/cm$^2$ |
| Acetic acid spray (2.5%) | 55°C, 0.5 min | Breast | *S.* Hadar | 1.8-2.0 log CFU/10 cm$^2$ |
| Acetic acid spray (20 ppm) | 0.3 min | Carcass | *S.* Typhimurium | 0.8 log CFU/ml |
| Acetic acid immersion (20 ppm) | 4°C, 45 min | Carcass | *S.* Typhimurium | 1.4 log CFU/ml |

| Treatment | Temperature, time | Sample | Inoculation | Reduction |
|---|---|---|---|---|
| Lactic acid immersion (1%) | 25°C, 30 min | Breast | *Salmonella* spp. | 2.0 log CFU/cm$^2$ |
| Lactic acid immersion (0.5-2%) | 25°C, 10-30 min | Breast (retail) | *S.* Enteritidis | 0.8-1.7 log CFU/g |
| Lactic acid spray (1-2%) | 20°C, 0.5 min | Breast | *S.* Typhimurium | 2.2 log CFU/ml |
| Lactic acid spray (2%) | 35°C, 0.3 min | Carcass | *S.* Typhimurium | 1.8 log CFU/carcass |
| Chlorine spray (55 ppm) | 21-54°C, 0.1 min | Carcass | *Salmonella* spp. | 0.9-1.1 log CFU/ml |
| Cetylpyridinium chloride spray (0.5%) | 35°C, 0.28 min | Carcass | *S.* Typhimurium | 2.0 log CFU/carcass |
| Cetylpyridinium chloride spray (0.1%) | 10-60°C, 0.5 min | Breast | *S.* Typhimurium | 1.5-2.5 log CFU/38.5 cm$^2$ |
| Cetylpyridinium chloride spray (0.1%) | 15 or 50°C, 1 min | Breast | *S.* Typhimurium | 0.9-1.7 log CFU/cm$^2$ |
| Cetylpyridinium chloride spray (0.1-0.5%) | 20°C, 0.5 min | Breast | *S.* Typhimurium | 1.5-1.9 log CFU/ml |
| Cetylpyridinium chloride immersion (0.1%) | 1-3 min | Breast | *S.* Typhimurium | 1.0-1.6 log CFU/cm$^2$ |
| TSP immersion (1%) | 25°C, 25 min | Breast | *Salmonella* spp. | 1.7 log CFU/cm$^2$ |
| TSP immersion (10%) | 20°C, 15 min | Carcass | *Salmonella* spp. | 1.4 log CFU/g |
| TSP immersion (210 mM) | 37°C, 10 min | Leg | *S.* Typhimurium | 2.3 log CFU/ml |
| TSP immersion (10%) | 10°C, 0.3 min | Leg (retail) | *S.* Typhimurium | >2.2 log CFU/cm$^2$ |
| TSP immersion (10 ppm) | 4°C, 45 min | Carcass | *S.* Typhimurium | 1.4 log CFU/ml |
| TSP immersion (1%) | 23°C, 10 min | Carcass | *S.* Typhimurium | 0.6-0.9 log CFU/cm$^2$ |
| TSP spray (5-10%) | 20°C, 0.5 min | Breast | *S.* Typhimurium | 2.1-2.2 log CFU/ml |
| TSP spray (10%) | 35°C, 0.3 min | Carcass | *S.* Typhimurium | 1.8 log CFU/carcass |
| TSP spray (10%) | 10-60°C, 0.5 min | Breast | *S.* Typhimurium | 1.5-2.1 log CFU/38.5 cm$^2$ |
| TSP spray (10 ppm) | 0.3 min | Carcass | *S.* Typhimurium | 0.9 log CFU/ml |

* Data are extracted from tables presented by Loretz *et al.* (2010) that summarise studies from other researchers. See Loretz *et al.* (2010) for references to the original studies.

Acidic electrolysed water (AEW, pH 2-3) and neutral electrolysed water (NEW, pH 7-8) effectively inactivate *Salmonella* species in suspension (Al-Haq *et al.*, 2005; Hricova *et al.*, 2008). For example, *S.* Enteritidis was reduced by >7 log when exposed to AEW (pH 2.4) for 5 minutes at 23°C, and by >6 log when exposed to NEW (pH 8.2) for the same time and temperature (Deza *et al.*, 2003; Venkitanarayanan *et al.*, 1999). Immersion of broiler carcasses inoculated with *S.* Typhimurium into AEW (pH 2.6) for 45 minutes at 4°C reduced the salmonellae concentration by 0.86 $\log_{10}$ CFU/ml of rinsate (although acetic acid and trisodium phosphate were more effective, reducing the concentration by 1.41 $\log_{10}$ CFU/ml rinsate) (Fabrizio *et al.*, 2002). Spray washing carcasses (85 psi, 15 seconds, 25°C) with AEW was as effective (reduction of 0.59 $\log_{10}$ CFU/ml of rinsate) as spraying with distilled

water. It is not clear whether pH, the concentration of active chlorine or the oxidation-reduction potential (or combinations of these factors) are responsible for the antimicrobial activity of AEW (Hricova *et al.*, 2008).

Pretreatment with basic electrolysed water (BEW; pH >11.3) seems to sensitize bacterial cell surfaces to any follow-up disinfecting agents (Hricova *et al.*, 2008). Spray washing chicken carcasses with BEW (pH 11.6) followed by immersion in AEW caused a larger reduction in *S.* Typhimurium concentration (reduction of 2.11 $\log_{10}$ CFU/ml of rinsate) than immersion in AEW alone (Fabrizio *et al.*, 2002).

In a study analysing the prevalence of *Salmonella* on broilers in 20 USA processing plants during 2005 (Berrang *et al.*, 2009), the prevalence was significantly higher at rehang (pre-evisceration; 574/800, 71.8%) than post-chill (161/798, 20.2%). The authors compared the chemical processing aids in use by the processing plants between rehang and post-chill. While the *Salmonella* prevalence was significantly lowered under all treatment conditions (including processing without any chemical aids), the prevalences on post-chill carcasses were lower when Sanova (acidified sodium chlorite, Ecolab Inc., St. Paul, MN), FreshFX (blend of food-grade acids, SteriFX Inc., Shreveport, LA) or Tom$CO_2$ (hypochlorous acid, Tomco Equipment Co., Loganville, GA) were in use. However, when comparing the *Salmonella* prevalences recorded for rehang with post-chill carcasses, the largest difference was recorded for processing plants using Inspexx (peroxyacetic acid–based antimicrobial, Ecolab Inc.; from 93% *Salmonella* positive at rehang to 20% *Salmonella*-positive post-chill, n=100). The difference using Tom$CO_2$ (42.5% to 12.5%, n=40) was similar to that observed with no chemical aids (62.5% to 32.3%, n=80).

### 7.3.3 Secondary processing

Cross-contamination is the main cause of *Salmonella* contamination of poultry products during secondary processing. Time/temperature controls are important to prevent growth.

#### *7.3.3.1 Salmonella reduction during secondary processing*

NZFSA recommends good manufacturing practices as part of their code of practice for the processing of poultry (NZFSA, 2009b):

- Secondary processors must process meat types with relatively high microbial counts (e.g. offal) separately from that with relatively low (e.g. whole birds), or process those with low counts first. Processing of different meat types (e.g. beef, lamb, poultry) must also be kept separate.
- Raw poultry should be kept at 10°C or below during processing (e.g. maintain air temperature at 10°C or less).
- Maturing should be less than 72 hours (usually it is 4-6 hours at 4°C).
- Comminution can raise the temperature of the meat – reducing the temperature prior to comminution should ensure that cooling time after comminution minimises the opportunity of microbial growth.

#### *7.3.3.2 Packaging*

Whole or individual parts of birds may be packaged raw for direct sale. A major poultry producer in New Zealand uses a system called 'Leakguard' in its packaging, whereby two bags are used and the second bag is double sealed.

Whole or individual parts of birds may be packaged raw for direct sale. Poultry producers in New Zealand have introduced the use of leak proof packaging, intended to prevent chicken juice leakage and potential cross contamination from the exterior of the package onto other foods. Where the birds are portioned, they are generally cut into a number of pieces, which are placed on "PLIX" porous food trays (open cell, expanded polystyrene) and covered with a plastic film.
Most frozen poultry is vacuum-packed in plastic bags and then frozen in high-velocity freezers. Before freezing, poultry may be injected with various salts, flavourings, and oils in order to increase the juiciness of the meat.

### 7.3.4 Retail poultry products and poultry handling

#### *7.3.4.1 Raw whole poultry or poultry pieces*

The water activity ($a_w$) of poultry meat is about 0.98 to 0.99. 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 a wide variety of microorganisms (ICMSF, 2005).

The shelf life of raw poultry is quite short in comparison with other meats. Shelf lives of 7, 5 and 4 days at 4, 7 and 9°C respectively were determined using an end point of approximately 7.2 $\log_{10}$ CFU spoilage bacteria/ml of half-carcass rinse (Abu Ruwaida *et al.*, 1996). This end point was accompanied by changes in organoleptic characteristics, which would make the chicken unacceptable to consumers. This is likely to limit the amount of *Salmonella* that might grow in the food prior to cooking (N.B. in one *Salmonella* outbreak involving turkey, the turkey was washed prior to serving to negate its poor organoleptic quality). Unless frozen, raw poultry has a rapid turnover at retail, often 24-48 hours with a best before date of 3-4 days (King and Wong, 2010).

At moderate temperatures *Salmonella* will grow rapidly on chicken. In an analysis of growth at 30°C, a lag time of 3 hours and a generation time of 0.74 hour (44 minutes) was found for *S.* Typhimurium growing on sterile, raw, skinless chicken breast (McKay *et al.*, 1997). In an older study, the number of *S.* Typhimurium inoculated onto chicken muscle increased from 4.7 $\log_{10}$ to 7.2 $\log_{10}$ in 21 hours at 20°C (Mattila and Frost, 1988).

A more recent study has modelled the growth of *Salmonella* species on raw poultry under aerobic conditions at various temperatures, using chicken tenderloins (non-sterilised) inoculated with antimicrobial resistant strains of *S.* Typhimurium or *S.* Kentucky, and strains of the same serotypes that were not resistant to antimicrobials (Dominguez and Schaffner, 2008). Neither antibiotic resistance nor inoculum size affected *Salmonella* growth rates, and the presence of spoilage microflora did not appear to slow the growth of the *Salmonella* species The authors' model predicted growth rates of:

- 0.0252 log CFU/h at 10°C;

- 0.1837 log CFU/h at 20°C;
- 0.4880 log CFU/h at 30°C; and
- 0.7878 log CFU/h at 37°C.

A clearly defined lag phase was not observed in the experimental work and was subsequently not modelled.

#### *7.3.4.2 Raw value-added products*

Additional processing such as marination, crumbing or adding other ingredients (e.g. stuffed poultry, filled poultry such as cordon bleu) can increase the risk of *Salmonella* being introduced to poultry products through cross-contamination between products, from equipment, workers or the environment, or from the added ingredients. Processing that involves the addition of preservatives (e.g. salt) or partial cooking (e.g. chicken nuggets) can reduce any *Salmonella* that might be present on the product or prevent further growth. Raw value-added products can be sold to the consumer fresh or frozen.

**Marinated poultry**

Marinades are used to improve tenderness and flavour. The migration of marinades into meat can also encourage pathogens on the surface of poultry or in the marinade to penetrate into the meat interior (Warsow *et al.*, 2008). Irradiated whole chicken breasts were marinated in a solution of 90% water, 7% NaCl and 3% mixed phosphate (wt/wt) which was inoculated with a cocktail of eight *Salmonella* serotypes. The chicken breasts were marinated for 2, 10 or 20 minutes at 4°C, either standing in the marinade under normal atmospheric pressure or under vacuum (a commercial method of marinating meat that might also involve tumbling). Core samples from the chicken breasts showed that the salmonellae migrated into the meat and were still detectable 4 cm deep, with or without vacuum. The authors did not present information on the rate of migration with time.

Some marinades might have an antibacterial effect. The effect of lemon pepper and teriyaki marinades on strains of *S.* Typhimurium, *S.* Heidelberg and *S.* Senftenberg inoculated onto chicken skin has been investigated (Pathania *et al.*, 2010). The samples were marinated for up to 36 hours, at 4 or 25°C. After 36 hours, the *Salmonella* concentration reduced under both marinades in samples held at 4°C, but the change in concentration was not significant. The only significant reduction was observed in samples marinated in teriyaki at 25°C. In separate experiments examining changes in the prevalence of each *Salmonella* serotype, the same researchers found that teriyaki marinade effectively reduced the prevalence regardless of storage temperature or *Salmonella* serotype. The teriyaki marinade's antimicrobial effect was attributed to an acidic pH (pH 4) and antimicrobial ingredients such as garlic, soy sauce, phosphates and salt.

**Chicken nuggets**

Most chicken nuggets and strips sold in stores are not fully cooked but can appear so, and an increased risk of contracting salmonellosis from these products has been attributed to uncertainty as to whether they are cooked (Bucher *et al.*, 2007). Chicken nuggets are also a reconstituted product so *Salmonella* can be present throughout the meat matrix.

The risk from chicken nuggets, and risk management measures, were discussed in a specific review conducted by ESR in 2004 (Wong and Lake, 2004) (see Section 4.1.1). A chicken nugget can be defined as a small piece of reconstituted raw chicken product encased in flash-fried batter or crumb. It contains between 33% and 56% chicken derived material (skinless breast and thigh meats, and skin), as well as flour and starch binders, water, soy protein, milk solids, mineral salts and flavourings. The ingredients are blended together and machine moulded into specific shapes.

The time taken from mould stamping, coating, battering and frying is roughly 5 minutes, too short for a significant increase in pathogen numbers. Flash frying in hot oil hardens the batter or crumb coating and holds the chicken product together thus giving it the characteristic nugget shape. Flash-frying is performed in hot oil (>180ºC) for no more than 30 seconds. This step is too short for the core temperature to reach a bactericidal treatment, i.e. 74ºC for 15 seconds for minced poultry. Following flash frying, the nugget does appear visually "cooked" but the core is still raw. *Salmonella*, if present internally, would survive this quick-heat treatment step. The internal temperatures of nuggets at this stage were measured during a visit by ESR to nugget manufacturers during 2004, and ranged from 12 to 26ºC.

The raw nuggets are then immediately chilled and frozen in a spiral freezer or liquid nitrogen tunnel to a temperature of -18ºC or lower. The hard-frozen nuggets are weighed and bagged along an automated conveyer belt and the bags packed into cartons by hand for frozen storage and distribution. During weighing and packing, the nuggets or related products are frozen solid. The survival of a cocktail of *Salmonella* serotypes inoculated into breaded (crumbed) cooked chicken nuggets or breaded raw chicken strips and stored frozen has been studied (Dominguez and Schaffner, 2009). The researchers found that storage at -20°C for 16 weeks sublethally injured *Salmonella* species but the bacteria were able to recover and the concentration did not reduce over the study period. In a more recent study in Canada (Bucher *et al.*, 2007), *Salmonella* was present in 95% of 20 chicken nugget meat samples taken from a package of *Salmonella*-positive chicken nuggets, but the pathogen was not detected in the nugget coatings, suggesting that the source of the contamination was the meat.

#### *7.3.4.3 Packaged ready-to-eat products*

The growth of *Salmonella* species in cooked chicken breast patties was suppressed in the presence of carbon dioxide (Murphy *et al.*, 2001b). Hot water pasteurisation of cooked poultry can reduce or eliminate *Salmonella* species, but effectiveness is reduced with thicker packaging film (Murphy *et al.*, 2002b), and larger or thicker portions of meat (Murphy and Berrang, 2002; Murphy *et al.*, 2003b).

#### *7.3.4.4 Transportation and refrigeration at retail*

Transportation of poultry products under chilled (4°C or lower) or frozen conditions will prevent the growth of any *Salmonella* species present on the product. Poultry meat surface temperatures were measured in a new Zealand study during distribution from slaughterhouse to retail premises during winter, 2004 (Whyte, 2005). Poultry transported on seven occasions did not exceed 7°C. During summer 2005, the surface temperature of meat upon arrival at retail premises was measured 193 times; 15 (8%) of the readings exceeded the 7°C.

The surface temperatures of meat held in New Zealand retail premises under refrigeration were measured during 2004/05 (Whyte, 2005). Of the 1,368 temperature readings recorded for meat surfaces on display, only 13 (1%) readings exceeded 13°C.

### 7.3.5 Domestic poultry handling

#### *7.3.5.1 Consumer transportation*

The times and temperatures of purchased poultry products during transportation by consumers have been examined in a New Zealand study (Gilbert *et al.*, 2006). Packets of fresh chicken drumsticks were stored in various packaging conditions (supermarket bag, or a cooler bag with or without an icepack) and placed either in a car boot or car interior to simulate the period between purchase and storage of these products in the home. The internal and surface temperatures of the products were monitored over several hours during three experiments each in summer and winter. The initial surface temperature of the drumsticks was between 9 and 13°C in summer (internal temperature 6-10°C), and between 7 and 13°C in winter (internal temperature 6-11°C). In summer, the drumsticks reached 15°C in less than two hours, even when stored in a chiller bag with an icepack in the boot of the car (Table 8). *Salmonella* species could start to grow at 15°C, albeit slowly. A surface temperature of 30°C (closer to optimal growth conditions for *Salmonella* species) was only recorded under the following three conditions (based on mean values of replicates), all during summer:

- Storage in a plastic bag in the car interior (30°C reached after approximately 1.5 h);
- Storage in a plastic bag in the car boot (30°C reached after approximately 4 h); and
- Storage in a chiller bag (no icepack) in the car interior (30°C reached after approximately 3.25 h).

**Table 8: Transport temperature – approximate time for chicken drumsticks to reach 15°C after being purchased and stored in a car (Gilbert et al., 2006)**

| Season | Position in car | Packaging | Approximate time to reach 15°C* | |
|---|---|---|---|---|
| | | | Internal | Surface |
| Summer | Interior | Plastic bag | 0.75 | 0.5 |
| | | Cooler bag | 1.0 | 1.0 |
| | | Cooler bag + icepack | 1.75 | 1.25 |
| | Boot | Plastic bag | 0.75 | 0.5 |
| | | Cooler bag | 0.75 | 0.75 |
| | | Cooler bag + icepack | 2.0 | 1.25 |
| Winter | Interior | Plastic bag | 2.5 | 2.0 |
| | | Cooler bag | 3.0 | 2.5 |
| | | Cooler bag + icepack | Not reached | 4.0 |
| | Boot | Plastic bag | 4.5 | 4.5 |
| | | Cooler bag | Not reached | Not reached |
| | | Cooler bag + icepack | Not reached | Not reached |

* Estimated to nearest 15 minute (0.25 h) from graphs presented in (Gilbert *et al.*, 2006) which show mean values of three replicates.

In a New Zealand consumer survey, the majority of poultry (62.9%) was purchased fresh (rather than frozen), and most consumers (94.4%) claimed that the time taken from food selection to reaching their home was one hour or less (Gilbert *et al.*, 2007). Approximately 64% of poultry purchased would be frozen once the consumer got it to their home.

#### *7.3.5.2 Domestic thawing*

In a New Zealand consumer survey, respondents were asked about the method and time they would take to thaw a representative piece of poultry (e.g. a small chicken) (Gilbert *et al.*, 2007). Of 318 respondents to this question, 46.2% thawed at room temperature, most of whom thawed at this temperature for up to 12 hours. A sink of cold water was used by 4.1% of the respondents, 18.2% used a microwave and 5.7% cooked the product from frozen. Refrigerated thawing was reported by the remaining 25.8% of respondents, most of whom thawed under refrigerated conditions for up to 12 hours.

A 2007 study collected data on the freezing and thawing of chicken breast samples with and without skin (McIntyre *et al.*, 2007). The time taken for chicken portions to reduce from 1 to -5°C in a freezer was between 186 and 659 minutes (approximately 3-11 hours). Chicken samples thawed at room temperature took on average 686 minutes (approximately 11 hours) to reach ambient temperatures. Thawing at refrigeration temperatures took considerably longer (18 hours to nearly 3 days).

Multiplication of a cocktail of *Salmonella* serotypes inoculated onto whole chickens has been monitored as chickens were thawed at 22 or 30°C for 9 hours (Ingham *et al.*, 2005). After 9 hours of thawing at 30°C, the exterior of the whole chickens had reached a temperature of 20°C but *Salmonella* growth was not observed. Interior temperatures were not monitored.

#### *7.3.5.3 Cooking*

The results from a number of heat inactivation studies of *Salmonella* in minced poultry products have been reviewed (O'Bryan *et al.*, 2006). All of the experiments used mixtures of *Salmonella* strains. While it should be noted that these studies are not directly comparable since the methods and poultry substrates varied, the compiled results show that *Salmonella* are quickly inactivated at temperatures above 60ºC. At 60ºC the D value range was 4-8 minutes in raw, inoculated products. The upper 95$^{th}$ percentile D value, based on regression analysis, has been estimated to be approximately 13 minutes for poultry (McInytre and Hudson, 2011). At 65ºC, the D times were <1 minute (upper 95$^{th}$ percentile 1.9 minutes), and at 70ºC the D times were <10 seconds (upper 95$^{th}$ percentile 27 seconds). The unusually heat resistant serotype *S.* Senftenberg was reported to have D values of approximately 220, 14 and 3 minutes at 55, 60 and 65ºC respectively in ground turkey thigh meat (Veeramuthu *et al.*, 1998), and was still detected in ground chicken patties cooked to an internal temperature of 80°C (Murphy *et al.*, 2001c). However, many of the studies cited in O'Brian *et al.* (2006) included *S.* Senftenberg in their inoculum mixes.

The death kinetics of *Salmonella* in poultry can depend on the type of food being cooked. Slightly longer cooking times are required to inactivate salmonellae in poultry products with higher fat content. In experiments with minced turkey and chicken with various fat contents, the inactivation of salmonellae took longer in samples containing higher concentrations of fat (Juneja *et al.*, 2001). For example, in chicken meat treated at 60°C a 7 $\log_{10}$ reduction of

salmonellae took approximately 34 minutes with 2% fat but just over 40 minutes with 12% fat. Additionally, at higher temperatures (e.g. 65°C) a lag period was apparent for samples with higher fat concentrations before linear bacterial death commenced.

The death kinetics of *Salmonella* in poultry is also affected by the level of moisture in the cooking environment. Studies of chicken patties inoculated with *Salmonella* species and cooked in an air convection oven showed that the thermal lethality increased with increasing product temperature and wet bulb temperature (Murphy *et al.*, 2001a; Murphy *et al.*, 2001b). A higher wet bulb temperature correlates with higher humidity.

It appears that slightly longer cooking times are required to inactivate salmonellae inoculated into processed poultry products such as chicken nuggets. At 60°C the D times for a cocktail of six *Salmonella* serotypes inoculated into commercially formulated chicken patties and chicken tenders were 8.1 and 8.5 minutes, respectively (Murphy *et al.*, 2002a). At 65°C the D times were 1.4 and 1.3 minutes, and at 70°C the D time for both products was about 20 seconds.

In a comparison of whole and ground turkey breasts inoculated with a mixture of eight *Salmonella* serotypes, the rate of *Salmonella* inactivation was significantly greater in ground meat than in whole meat samples when heated at 55, 60 or 62.5°C (Tuntivanich *et al.*, 2008). The minced meat was formed from the same batch of whole turkey breasts so the samples were similar in composition. The authors offered some suggestions for these results, which included bacterial protection in whole muscle meat through attachment to fibres or internalisation, and increased susceptibility to heat in minced meat due to higher water availability.

A review of the thermal resistance of salmonellae (Doyle and Mazzotta, 2000) reported that deep fat frying of chicken parts coated with batter from room temperature, chilled, or frozen initial temperatures, was an effective means of destroying the pathogen when cooked to a temperature of 73.9°C.

The main concern with microwave cooking is that does not reliably destroy salmonellae inoculated onto chickens or turkeys, even though recommended internal temperatures may have been reached. This may be because heating is uneven resulting in parts of the food not reaching lethal temperatures (Heddleson *et al.*, 1994). The varied composition of poultry products (e.g. fat, protein, salt and moisture) can also affect the effectiveness of microwave cooking. Under commercial conditions, vapour inserted in the oven cavity to distribute the heat or the use of packaging with valves can help with more even heating (Aymerich *et al.*, 2008).

Studies of microwave cooking are subject to the microwave technologies available at the time and older studies may have used microwave ovens of lower power and without rotating turntables. In an experiment published in 2009 (Jamshidi *et al.*, 2009), chicken “drumettes” (the small fleshy part of a chicken wing) were soaked in a broth containing *S.* Typhimurium then microwaved for up to 35 seconds in a domestic oven at full power. *S.* Typhimurium were not detected on the surface of the chicken after 25 seconds at which time the surface temperature was >60°C.

In a 2005 study (Pucciarelli and Benassi, 2005), chicken thighs were coated with an inoculum of *S.* Enteritidis and cooked at different power levels in a domestic microwave oven. The temperature under the skin and inside the thigh of each portion was recorded after cooking. The temperature increase was not smooth and the temperature under the skin was always lower than inside the thigh, sometimes by as much as 11°C (the thermometers were inserted after different cooking times which may account for both of these findings). At high power, the reduction in *S.* Enteritidis was not linear; reduction was slow initially, followed by a period of faster reduction, which then tailed off to a slower rate. The reduction was more linear when the samples were cooked under medium power, but a longer cooking time was required before *S.* Enteritidis was no longer detectable.

Sausages manufactured from mechanically recovered poultry meat (8.6% fat) were inoculated with *S.* Enteritidis and cooked in a waterbath for 10 or 30 minutes at 50, 60 or 70oC, or 30 minutes at 75°C (Yuste *et al.*, 2000). The background concentration of *Salmonella* before inoculation was 1.36 CFU/g. No significant reduction in *Salmonella* was observed at 50°C. After treatment for 10 minutes at 60°C the concentration of *Salmonella* had reduced by approximately 3.6 log, and cooking for any period at 70°C or above reduced the concentration by 6-7 log.

#### *7.3.5.4 Cooked poultry*

The D- and z-values of *Salmonella* species in fully cooked chicken breast meat fillets (75% moisture, 20% protein, < 2% fat), turkey breast meat (78% moisture, 15% protein, < 2% fat and roast duck halves (deboned; skin and muscle meat tested separately) have been determined (Murphy *et al.*, 2003a). The cooked poultry products were ground and inoculated with a cocktail of six *Salmonella* serotypes and cooked in sealed bags in a waterbath. D-values were recorded at 2.5°C intervals from 55 to 70°C, and some of these are presented in Table 9. The z-values were calculated as follows:

- Chicken 6.3°C;
- Turkey 6.2°C; and
- Duck meat 5.8°C.

**Table 9: D-values of *Salmonella* species in fully cooked poultry products**

| Product | D-values[1] (minutes) | | | |
|---|---|---|---|---|
| | 55°C | 60°C | 65°C | 70°C |
| Chicken | 24.1 | 3.8 | 0.6 | 0.1 |
| Turkey | 24.7 | 5.2 | 0.6 | 0.1 |
| Duck meat | 28.6 | 6.8 | 0.6 | 0.1 |

[1] (Murphy *et al.*, 2003a) (mean of three replicates)

Chicken breast patties inoculated with cocktail of *Salmonella* serotypes (including *S.* Senftenberg) were cooked in an air convection oven to an internal temperature of 70°C, after which no salmonellae were detected (limit of detection was <1 $\log_{10}$ CFU/g) (Murphy *et al.*, 2001b). However, when the patties were subsequently stored at 15°C for six days, the researchers detected the salmonellae at a concentration 5.96 $\log_{10}$ CFU/g after only one day. The result suggested that heat-damaged cells were able to recover and multiply.

## 7.4 Prevalence of *Salmonella* species in Poultry Products in New Zealand

### 7.4.1 National Microbiological Database-

Schedule 1 of the Animal Products (National Microbiological Database Specifications) Notice 2011 sets out the requirements of the National Microbiological Database (NMD) Programme.[25] Premises operating to process broiler chickens must have the NMD programme in place. The NMD programme requires processors to sample fresh broiler carcasses every processing week, with the number and frequency of sampling depending on the size of the operation:

- Very Low Throughput (VLT) premises must sample at least five carcasses on one processing day of each processing week. VLT premises are those that slaughter one million or less birds per annum.[26]
- All other processors must sample three carcasses per processing day.

The samples must be taken after chilling, prior to bagging or further processing, and tested for *E. coli*, *Salmonella* species and *Campylobacter* species Schedule 1 contains specifications for how samples are to be taken and tested; the samples are tested using the whole rinse carcass method such that:

- All carcasses are tested for *Campylobacter* species;
- One carcass is tested for *Salmonella* species and *E. coli*; and
- For non-VLT premises, an additional carcass is tested for *E. coli*.

Thus VLT premises will submit *Salmonella* results to the NMD for one carcass per processing week and all other premises will submit *Salmonella* results for one carcass per processing day.

The PIANZ website states that in the mid-1990s the prevalence of *Salmonella* on carcasses was 17%. Testing of poultry by the NMD commenced in 2001. The previous Risk Profile (Lake *et al.*, 2004) reported that data received from PIANZ showed the rate of *Salmonella* isolation from whole poultry rinses was 2.0% in 2003. In 2002, the rate was 1.0%, whereas the last two quarters of 2001 it was 2.1%. The NMD results since 2005 for the detection of *Salmonella* species on poultry are presented in Table 11.

[25] Available at http://www.foodsafety.govt.nz/elibrary/industry/animal-products-national-nmd/schedule-2011.pdf (accessed 3 June 2011).
[26] The requirements for VLT plants are currently under review. See: http://www.foodsafety.govt.nz/elibrary/industry/consultation-proposed-changes-to-campylobacter-performance-target/index.htm (accessed 3 June 2011).

**Table 10: NMD results for *Salmonella* species on whole poultry carcasses, 2005-2010***

| Year | Number of samples tested | Number of *Salmonella*-positive samples (%) |
|---|---|---|
| 2005 | 1,930 | 68 (3.5) |
| 2006 | 1,885 | 40 (2.1) |
| 2007 | 1,918 | 15 (0.8) |
| 2008 | 1,980 | 13 (0.7) |
| 2009 | 1,906 | 2 (0.1) |
| 2010 | 1,876 | 3 (0.2) |

* Data provided by PIANZ, http://www.pianz.org.nz/food-safety/safety-information/salmonella-in-new-zealand-broiler-chickens

### 7.4.2 Product surveys

#### *7.4.2.1 Retail survey 2007*

During October and November 2007, 163 whole broiler carcasses were purchased from retail outlets in Auckland, Wellington, and Christchurch and tested for the presence of *Salmonella* species using enrichment (Chrystal *et al.*, 2008). The sampling ensured carcasses from each of the seven major New Zealand poultry processing plants were tested. *Salmonella* species were not detected in 163 carcasses using a whole carcass rinse.

#### *7.4.2.2 Retail survey 2003-2005*

From August 2003 to May 2005, 232 chicken samples were purchased from butchers and supermarkets in Auckland, Hamilton, Wellington, Christchurch and Dunedin (Wong *et al.*, 2007). All samples were raw and minced, diced or cut into strips. The samples were tested for prevalence of *Salmonella* per 25g, followed by enumeration of an additional 10 g of *Salmonella*-positive samples. It was estimated that the sampling programme would give 99% confidence of detected contamination if present at a rate of 2% or greater.

*Salmonella* was detected in 7/232 chicken samples (3.0%, 95% CI 1.2-6.1). The concentrations of *Salmonella* in these positive samples were low and comprised the following serotypes:

- *S.* Typhimurium PT160 — 0.61 MPN/g (95% CI 0.29-3.84)
- *S.* Typhimurium PT1 — 0.30 MPN/g (95% CI 0.09-2.08)
- *S.* Enteritidis PT9a — <0.30 MPN/g
- *Salmonella* sp. 4,12:-:- — <0.30 MPN/g
- *Salmonella* sp. 4,12:-:- variant — <0.30 MPN/g
- *Salmonella* sp. 4,5,12:-:- — <0.30 MPN/g
- *Salmonella* sp. 6,7:k:- — <0.30 MPN/g

A comparison of the *S.* Typhimurium PT1 strain (isolated from diced chicken from a Dunedin store) and the three serotypes 4,12:-:-, 4,12:-:- variant and 4,5,12:-:- (all isolated from minced chicken from stores in Dunedin) showed that all strains were indistinguishable or 96% similar by pulsed field gel electrophoresis to the MeganVac1 strain of *Salmonella*.

This vaccine strain is an attenuated strain of *S.* Typhimurium PT1 that is predominantly used to vaccinate breeding stock and laying hens. The authors suggested that the mince samples (all from the same processing plant) became contaminated by edible meats recovered from spent layers, breeding stock or broiler birds.

#### *7.4.2.3 Christchurch retail survey 2003*

In February 2003, NZFSA was notified by the management of a poultry processing plant that a batch of stock feed used to feed broilers in the South Island was contaminated with *S.* Typhimurium DT1. An increased prevalence of *Salmonella* was noted in whole bird rinses in the processing plant and there was also an increase in salmonellosis cases in Canterbury during January/February who were infected with *S.* Typhimurium DT1. To assess the flow-on effect of the contaminated batch of poultry, 200 samples of poultry meat (100 whole birds and 100 portions) were purchased from supermarkets, restaurants and a fast food outlet in Christchurch over four-week period in February/March 2003 (Wong, 2003; Cook et al., 2006).

*S.* Typhimurium was detected in 14/200 samples (7%; 9 whole birds and 5 portions) and most of the positive samples were detected in the first week of sampling (all samples were negative in weeks three and four). The positive samples comprised the following phage types:

- DT1: 10 samples;
- DT12a: 3 samples; and
- DT1 and DT12a: 1 sample.

For most positive samples the salmonellae were present in low numbers (<9 MPN/sample). The concentration on one whole bird was 720 MPN.

#### *7.4.2.4 Vertical chain survey 2004*

Between September 2003 and June 2004, a total of 610 chilled chicken portions (breasts, thighs, drums and wings/nibbles) were tested for *Salmonella* (Wong, 2004). The samples covered seven chicken processors in New Zealand, which represented all major processing companies and brands. Of the 610 samples, 310 were sampled at the end of primary processing and 300 were purchased from retail outlets. To investigate changes in the prevalence and levels of *Salmonella* vertically through the supply chain, the samples from retail outlets were collected on the same day or one day after dispatch from the processors to retail outlets.

All 300 retail samples of chicken portions were negative for *Salmonella.* At the same time, 1/310 (0.3%) samples of chicken portions provided by the primary processors was positive (the serotype identified was *S.* Agona, present at <6 MPN/portion).

#### *7.4.2.5 Poultry processing survey 2005-2006*

Between April 2005 and February 2006, 200 broiler chickens were sampled immediately post-stunning and ex-sanguination, but prior to scalding, from four commercial processing plants (two in the South Island and two in the North Island) (Wong and Hudson, 2006). The chickens were obtained in batches of five, where all five were from the same flock, and each

processor provided 50 birds. The sampling programme captured 39 flocks supplied by 30 farms.

All of the birds were tested for *Salmonella* by caecal swab (presence/absence) and whole bird rinsate (enumeration). The caecal content of only one bird was positive for *Salmonella*. *Salmonella* species were isolated from the rinsates of 49 chickens (24.5%), representing 12 (30.8%) flocks, and all of these samples were from two of the four processors. The concentration of *Salmonella* species was generally low, but the authors reported some difficulties in attaining counts. The highest concentration of *Salmonella* reported was $3 \times 10^3$ CFU $bird^{-1}$. The most common serotypes isolated were *S.* Typhimurium DT101, *S.* Tennessee and *S.* Infantis.

*7.4.2.6 Earlier surveys*

A study published in 1995 reported that 13/137 (17%) unfrozen poultry samples and 2/17 (12%) frozen raw chicken samples were contaminated with *Salmonella* (Campbell and Gilbert, 1995). The serotypes that were identifiable comprised *S.* Infantis (36% of salmonellae detected), *S.* Hadar (28%), *S.* Typhimurium PT13 (12%), *S.* Thompson (8%), *S.* Tennessee (4%), *S.* Brandenburg (4%) and *S.* Typhimurium PT8 (4%). All of the *S.* Typhimurium PT13 isolates were from ducklings. In the same study, salmonellae were not detected in 1,326 ready-to-eat chicken products.

A study published by *Consumer* magazine reported that salmonellae were detected in 17/50 (34%) whole, fresh raw chickens (Anonymous, 1999). The chickens were purchased from supermarkets and butcheries in Christchurch and Auckland. Serotyping was not carried out.

7.4.3 Packaging surveys

A 2002 study evaluated *Salmonella* species contamination on the outside of poultry packaging (Wong *et al.*, 2004). Three hundred packs of fresh chilled raw poultry were purchased from retail outlets in Christchurch, consisting of 50 whole chickens in bags, and 200 packs of portions and 50 packs of chicken offals on plastic-wrapped trays. The surface area of each tray sample was measured and the leakage from all packs graded from 1 to 3, where 3 represented visible leakage of close to a teaspoon inside the bag in which the sample pack was collected. The outside of each package was tested for the presence and enumeration of *Salmonella* species by a whole rinse method.

*Salmonella* species were detected on one (0.3%) sample, which was serotyped as *S.* Tennessee. The sample was a whole chicken and the concentration of *S.* Tennessee was <6 MPN/pack. The leakage from this pack was graded as 2, which meant that there were large visible droplets in the transportation bag.

A 2007 study evaluated *Salmonella* species contamination on the outside of the packaging of 163 whole poultry carcasses purchased from retail outlets in Auckland, Wellington and Christchurch (Chrystal *et al.*, 2008). The researchers swabbed a 25 $cm^2$ area of each package but did not detect *Salmonella* species on any sample.

### 7.4.4 Common serotypes

Data on the serotypes of *Salmonella* isolated by the poultry industry are available annually from two sources:

- MAF Biosecurity publishes data generated by the New Zealand poultry industry and other poultry sample testing laboratories in their journal *Surveillance*. The *Salmonella* isolates are cultured from poultry feed and broiler samples. The broiler samples from 2005 to 2007 were neck flaps, caecal swabs and environmental swabs, and were environmental swabs and whole-carcass-rinse birds from 2008 to 2009.[27]
- ESR publishes data generated by ERL. The *Salmonella* isolates are submitted to the ERL by poultry producers and are cultured from samples of poultry (neck flaps, "product"), feed and environmental samples. It should be noted that not all isolates are submitted to the ERL (although most *S.* Typhimurium isolates are likely to be submitted for phage typing), so the information may not be completely representative of those cultured on a day-to-day basis in laboratories servicing the poultry industry.[28]

There is potential for these two data sets to overlap, so they are presented separately in Table 11 and Table 12. These tables do not list all of the serotype names but show those identified more often (the cut-off values of five-or-more or ten-or-more are arbitrary values reflecting the size of the data sets). MAF publishes the broiler sample results as aggregate values from all sample types each year, so the ERL results are presented similarly. It is important to note that the data sets only indicate the possible prevalence of different *Salmonella* serotypes in poultry and poultry feed; they are not the results of specific studies of serotype prevalence.

From 2005 through 2009 there were 35 different serotypes identified by the poultry industry and 92 different serotypes identified by the ERL. All of the serotypes identified by the poultry industry were also identified by the ERL, except for *S.* Cubana (four isolates from feed in 2004) and *S.* Sandiego (one isolate from feed in 2009).

The 2004 Risk Profile presented data from 1998 through 2003. The most commonly isolated serotypes were *S.* Agona, *S.* Typhimurium DT101, *S.* Typhimurium DT135, *S.* Infantis, and *S.* Brandenburg. The 2005-2009 data shows that *S.* Agona, *S.* Typhimurium DT101 and *S.* Infantis are still commonly isolated, although the ERL broiler sample data show a decline in *S.* Typhimurium DT101 (from 51% of isolates in 2005 to 4% in 2009). *S.* Tennessee was also among those most commonly identified in the latter time period, but both data sets indicate a decline in prevalence.

The 1998-2003 data set registered the emergence of *S.* Typhimurium DT160 in 2000, although the case-control study investigating this serotype did not identify poultry as a significant risk factor (Thornley *et al.*, 2003) (Table 21, Appendix 2). For the period 2005-2009, *S.* Typhimurium DT160 represented only 1% (8/603) of the serotyped isolates from the poultry industry and 5% (83/1,634) of the serotyped ERL isolates.

[27] *Surveillance* is available at: http://www.biosecurity.govt.nz/publications/surveillance/index.htm (accessed January 2010).
[28] The annual reports of non-human *Salmonella* serotypes submitted to the ERL are available at: http://www.surv.esr.cri.nz/enteric_reference/nonhuman_salmonella.php (accessed January 2010).

The 1998-2003 data set also showed the emergence of *S.* Derby in 2003 (0.6% of MAF isolates and 3.5% of ERL isolates). This serotype is among the most common serotypes identified in the latter data set, although the prevalence has not markedly increased (4.3% of serotyped MAF isolates and 7.6% of ERL isolates).

Some of the serotypes appeared to peak over one or two years during the 2005-2009 period. For example, *S.* Give 15+ was frequently identified in broiler and feed samples in 2007, but rarely identified in other years. *S.* Kentucky was identified more often in 2006 than other years.

When comparing isolation of serotypes in feed or broiler samples as proportions of the total serotyped isolates for each sample type, there is some suggestion that *S.* Derby is more frequently isolated from feed samples (MAF = 6.0% of feed isolates, ERL = 17.1%) than broiler samples (MAF = 3.6% of broiler isolates, ERL = 2.8%). In contrast, it also appears that *S.* Typhimurium DT101 is isolated more often from broiler samples (MAF = 17.3% of broiler isolates, ERL = 41.1%) than from feed samples (MAF = 2.2% of feed isolates, ERL = 2.4%). However, these data are not results from a prevalence study so there is no certainty that a true difference exists.

#### *7.4.4.1 Antimicrobial susceptibility*

The antibiotic susceptibility of 1,560 human and 1,505 non-human *Salmonella* isolates from human and non-human sources in New Zealand has been evaluated (Broughton *et al.*, 2010). The isolates were obtained between 2002 and 2007. Overall, more isolates were resistant to streptomycin than to any other antibiotic, but almost all isolates were susceptible to ciprofloxacin and gentamicin. Of 436 poultry isolates, 92 (21.1%) were not susceptible to streptomycin, 11 (2.5%) were not susceptible to sulfonamides and 5 (1.1%) were not susceptible to tetracycline. All were susceptible to ampicillin and trimethoprim. Ampicillin, streptomycin and sulfonamides are generally not used to treat clinical salmonellosis cases.

**Table 11: *Salmonella* serotypes identified five or more times (2005-2009) from poultry samples tested by the poultry industry**

| ***Salmonella* serotype** | **Poultry feed** | | | | | | **Broiler samples: Neck flaps, caecal, environmental, carcass** | | | | | | **Grand total** |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | **2005** | **2006** | **2007** | **2008** | **2009** | **Total** | **2005** | **2006** | **2007** | **2008** | **2009** | **Total** | |
| Agona | 0 | 0 | 44 | 0 | 0 | 44 | 41 | 46 | 0 | 15 | 5 | 107 | **151** |
| Typhimurium DT101 | 0 | 0 | 3 | 1 | 0 | 4 | 37 | 32 | 2 | 1 | 1 | 73 | **77** |
| Tennessee | 12 | 25 | 1 | 1 | 0 | 39 | 19 | 6 | 12 | 0 | 0 | 37 | **76** |
| Typhimurium | 0 | 1 | 0 | 0 | 0 | 1 | 24 | 33 | 0 | 0 | 0 | 57 | **58** |
| Infantis | 6 | 2 | 10 | 0 | 0 | 18 | 6 | 1 | 14 | 2 | 1 | 24 | **42** |
| Give 15+ | 0 | 1 | 0 | 0 | 0 | 1 | 0 | 0 | 40 | 0 | 0 | 40 | **41** |
| Derby | 6 | 1 | 2 | 0 | 2 | 11 | 4 | 8 | 1 | 2 | 0 | 15 | **26** |
| Mbandaka | 2 | 3 | 0 | 0 | 0 | 5 | 0 | 0 | 14 | 0 | 0 | 14 | **19** |
| Anatum | 3 | 10 | 0 | 0 | 0 | 13 | 0 | 0 | 2 | 0 | 0 | 2 | **15** |
| Typhimurium RDNC | 0 | 2 | 0 | 0 | 0 | 2 | 0 | 0 | 12 | 1 | 0 | 13 | **15** |
| Oranienburg | 1 | 7 | 0 | 0 | 0 | 8 | 0 | 0 | 3 | 0 | 0 | 3 | **11** |
| Anatum 15+ | 5 | 1 | 0 | 0 | 0 | 6 | 0 | 0 | 3 | 0 | 0 | 3 | **9** |
| Senftenberg | 2 | 0 | 0 | 0 | 2 | 4 | 3 | 0 | 2 | 0 | 0 | 5 | **9** |
| Kentucky | 0 | 3 | 0 | 0 | 0 | 3 | 0 | 4 | 1 | 0 | 0 | 5 | **8** |
| Typhimurium DT160 | 3 | 4 | 0 | 0 | 0 | 7 | 0 | 0 | 1 | 0 | 0 | 1 | **8** |
| Brandenburg | 1 | 1 | 0 | 0 | 1 | 3 | 0 | 1 | 2 | 0 | 0 | 3 | **6** |
| Rissen | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 4 | 0 | 0 | 4 | **5** |
| **Total poultry isolates serotyped[1]** | **48** | **63** | **62** | **3** | **6** | **182** | **135** | **133** | **123** | **22** | **8** | **421** | **603** |
| Total serotypes identified | 14 | 15 | 7 | 3 | 4 | 25 | 8 | 10 | 23 | 6 | 4 | 29 | **35** |
| No. isolates with undetermined serotype | 2 | 0 | 0 | 0 | 0 | 2 | 37 | 74 | 1 | 5 | 0 | 117 | **119** |

1. Including serotypes that were identified four times or less from 2005 through 2009.
Source: *Surveillance* (the quarterly MAF Biosecurity magazine, available at http://www.biosecurity.govt.nz/publications/surveillance/index.htm).

**Table 12: *Salmonella* serotypes identified 10 or more times (2005-2009) from poultry isolates submitted to the ERL**

| *Salmonella* serotype | Poultry feed | | | | | | Broiler samples: Neck flaps, product, environmental | | | | | | Grand total |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | 2005 | 2006 | 2007 | 2008 | 2009 | Total | 2005 | 2006 | 2007 | 2008 | 2009 | Total | |
| Typhimurium DT101 | 2 | 5 | 2 | 4 | 0 | 13 | 177 | 158 | 27 | 80 | 4 | 446 | **459** |
| Derby | 64 | 19 | 4 | 4 | 3 | 94 | 18 | 7 | 1 | 3 | 1 | 30 | **124** |
| Agona | 4 | 5 | 0 | 4 | 3 | 16 | 23 | 25 | 12 | 7 | 23 | 90 | **106** |
| Infantis | 3 | 0 | 6 | 16 | 0 | 25 | 20 | 18 | 24 | 14 | 4 | 80 | **105** |
| Typhimurium DT160 | 9 | 7 | 1 | 9 | 11 | 37 | 21 | 11 | 4 | 8 | 2 | 46 | **83** |
| Typhimurium RDNC | 0 | 1 | 3 | 9 | 2 | 15 | 2 | 10 | 13 | 16 | 14 | 55 | **70** |
| Tennessee | 9 | 4 | 1 | 7 | 1 | 22 | 23 | 8 | 1 | 4 | 0 | 36 | **58** |
| Brandenburg | 14 | 7 | 4 | 2 | 3 | 30 | 7 | 11 | 1 | 3 | 1 | 23 | **53** |
| Mbandaka | 4 | 3 | 9 | 13 | 2 | 31 | 2 | 1 | 3 | 13 | 2 | 21 | **52** |
| Senftenberg | 10 | 5 | 3 | 12 | 6 | 36 | 9 | 2 | 4 | 1 | 0 | 16 | **52** |
| Give 15+ | 0 | 1 | 30 | 0 | 0 | 31 | 0 | 0 | 1 | 1 | 0 | 2 | **33** |
| Anatum 15+ | 6 | 9 | 3 | 0 | 0 | 18 | 4 | 2 | 0 | 0 | 6 | 12 | **30** |
| Anatum | 1 | 2 | 4 | 7 | 1 | 15 | 7 | 3 | 4 | 0 | 0 | 14 | **29** |
| Typhimurium DT42 | 0 | 0 | 1 | 9 | 2 | 12 | 2 | 10 | 2 | 2 | 0 | 16 | **28** |
| Typhimurium DT89 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 24 | 24 | **24** |
| Kentucky | 0 | 0 | 1 | 1 | 0 | 2 | 1 | 17 | 2 | 0 | 0 | 20 | **22** |
| Havana | 0 | 2 | 4 | 1 | 2 | 9 | 4 | 2 | 3 | 3 | 0 | 12 | **21** |
| Group C 6,7 : k : - | 0 | 0 | 4 | 1 | 1 | 6 | 0 | 1 | 1 | 9 | 1 | 12 | **18** |
| Montevideo | 0 | 0 | 2 | 2 | 6 | 10 | 2 | 0 | 1 | 2 | 2 | 7 | **17** |
| Group E 3,19 : - : - | 0 | 0 | 0 | 12 | 0 | 12 | 0 | 0 | 0 | 1 | 0 | 1 | **13** |
| Heidelberg | 2 | 9 | 0 | 0 | 0 | 11 | 0 | 0 | 0 | 0 | 1 | 1 | **12** |
| Rissen | 0 | 0 | 4 | 3 | 3 | 10 | 0 | 0 | 0 | 1 | 0 | 1 | **11** |
| Thompson | 0 | 0 | 0 | 0 | 4 | 4 | 2 | 1 | 0 | 2 | 2 | 7 | **11** |
| Typhimurium DT1 | 0 | 0 | 0 | 0 | 0 | 0 | 2 | 6 | 2 | 1 | 0 | 11 | **11** |
| Oranienburg | 0 | 2 | 2 | 1 | 0 | 5 | 0 | 0 | 3 | 0 | 2 | 5 | **10** |
| Typhimurium DT12a | 1 | 1 | 0 | 0 | 0 | 2 | 3 | 3 | 0 | 2 | 0 | 8 | **10** |
| **Total isolates serotyped[1]** | **142** | **100** | **100** | **143** | **66** | **551** | **345** | **315** | **129** | **200** | **94** | **1,083** | **1,634** |
| Total serotypes identified | 20 | 29 | 31 | 34 | 28 | 65 | 28 | 32 | 30 | 35 | 20 | 64 | **92** |

1. From poultry only, including serotypes that were identified nine times or less from 2005 through 2009.
Source: ERL annual reports of non-human *Salmonella* serotypes (available at: http://www.surv.esr.cri.nz/enteric_reference/nonhuman_salmonella.php)

## 7.5 Prevalence of *Salmonella* species in Poultry Products in Other Countries

### 7.5.1 Product surveys

Data from surveys undertaken in other countries are presented in the following tables. These tables only include studies where samples were collected during the year 2000 or later, or in the absence of this information, where the report was published during 2000 or later. Earlier studies have been presented in the 2004 Risk Profile (Lake *et al.*, 2004b), and have also been summarised by Simmons *et al*. (2003). The data presented in these earlier studies are less relevant to a current exposure assessment because the prevalence in many countries has been lowered over the last decade through efforts to control *Salmonella* in poultry.

The prevalence and concentration values will be influenced by the sampling method, for example swab sampling is likely to lead to lower numbers being recorded than whole carcass rinsing (Logue and Nde, 2007).

Prevalence of *Salmonella* in raw poultry products (Table 13).

Prevalence of *Salmonella* in ready-to-eat poultry products (Table 14).

Concentration of *Salmonella* on raw poultry products (Table 15).

No studies were identified that enumerated *Salmonella* species in ready-to-eat poultry products.

**Table 13: Prevalence of *Salmonella* species in raw poultry products (studies published or conducted during 2000 or afterwards)**

| Year(s)[1] | Country | Samples tested | Number positive/ total (%) | *Salmonella* serotypes (%) | Reference |
|---|---|---|---|---|---|
| **Chicken: Australia** | | | | | |
| 1999-2000 | Australia: Australian Capital Territory | Retail: Whole carcasses, pieces, offal, comminuted, raw ready-made (e.g. kiev) | 109/266 (41.0) | (n=93 isolates)<br>Sofia subsp. II (58.1)<br>Kiambu (19.4)<br>Typhimurium DT135 (6.5)<br>Typhimurium DT64 (5.4)<br>subsp. II rough (2.2)<br>Typhimurium untypable (2.2)<br>Typhimurium DT9 (2.2)<br>Typhimurium RDNC (1.1)<br>Typhimurium DT135a (1.1)<br>Typhimurium DT193 (1.1)<br>Zanzibar (1.1) | (Millard and Rockliff, 2000) |
| 2002 | Australia: South Australia | 17 poultry processing plants: Whole carcasses, skinless breasts, liver | 140/260 (53.7)<br>- whole 68/120 (57)<br>- breast 66/120 (55)<br>- liver 6/20 (30) | (n=145 isolates)<br>Sofia (90.3)<br>Infantis (5.5)<br>Zanzibar (1.4)<br>Anatum (0.7)<br>Chester (0.7)<br>Mbandaka (0.7)<br>Typhimurium PT8 (0.7) | (Sumner *et al.*, 2004a) |
| 2002 | Australia: South Australia | Retail: Chicken fillet, mince, livers | 39/112 (34.8) | Sofia (74)<br>Typhimurium (15.4)<br>Infantis (7.7)<br>Zanzibar (2.6) | Pers. comm. reported in (FSANZ, 2005) |
| 1996-2003 | Australia: Western Australia | Carcasses | 47/369 (12.7) | Typhimurium (55.0)<br>Singapore (13.7)<br>Kiambu (7.8)<br>Bovismorbificans (3.9)<br>Bredeney (3.9)<br>Derby (3.9)<br>Infantis (2.0)<br>Adelaide (2.0)<br>Tennessee (2.0) | Pers. comm. reported in (FSANZ, 2005) |

| Year(s)[1] | Country | Samples tested | Number positive/ total (%) | *Salmonella* serotypes (%) | Reference |
|---|---|---|---|---|---|
| | | | | Livingston (2.0) | |
| 2008 | Australia: South Australia | Chicken meat (skin on or off) | 138/356 (38.8%) | (n=138 positive samples)<br>Infantis (20.3)<br>Typhimurium DT135a (14.5)<br>Sofia (13.8)<br>Typhimurium DT6 (11.6)<br>Kiambu (8.7)<br>Agona (6.5)<br>Reading (5.8)<br>*Salmonella* 16:1,v:- (2.9)<br>Typhimurium DT29 (2.9)<br>Adelaide (2.2) | (Fearnley *et al.*, 2011) |
| **Chicken: Europe (including the UK)** | | | | | |
| 2008 | EU Member States (MS) | Fresh broiler meat | 5.1% of 15,355 samples[2]<br>- at slaughter (9 MS) 0.6-23.4%<br>- at processing/cutting plant (9 MS) 0-15.6%<br>- at retail (12 MS) 0.3-16.2% | N/R | (EFSA, 2010a) |
| 2009 | EU Member States (MS) | Fresh broiler meat | 5.4% of 26,591 samples[2]<br>- at slaughter (13 MS) 0-60.8%<br>- at processing/cutting plant (13 MS) 0-31.1%<br>- at retail (17 MS) 0-36.1% | N/R | (European Food Safety Authority and European Centre for Disease Prevention and Control, 2011) |
| 2008 | EU Member States (MS) | Non-ready-to-eat broiler minced meat, meat preparation and meat products | 2.0% of 12,938 samples[2]<br>- at processing plant (8 MS) 0-10.8%<br>- at retail (10 MS) 0-17.8% | N/R | (EFSA, 2010a) |
| 2008 | 26 EU Member States, 2 non-Member States | Neck skin and breast skin from carcass after chilling at processing plant (561 slaughterhouses sampled) | 1,225/10,035 (12.2%) | (n=1,225 positive samples)<br>Infantis (29.2)<br>Enteritidis (13.6)<br>Kentucky (6.2)<br>Typhimurium (4.4)<br>Bredeney (4.3)<br>Virchow (4.1)<br>Hardar (3.8) | (EFSA, 2010b; European Food Safety Authority and European Centre for Disease Prevention and Control, 2011) |

| Year(s)[1] | Country | Samples tested | Number positive/ total (%) | *Salmonella* serotypes (%) | Reference |
|---|---|---|---|---|---|
| | | | | Paratyphi B var. Java (3.8)<br>Agona (3.0)<br>Indiana (2.9)<br>Other serotypes and non-typable (27.3) | |
| 2001-2002 | Italy | Laboratories: Routine testing of poultry samples collected from retailers and processors | 291/2,953 (9.9) | (n=199 isolates)<br>Blockley (11.6)<br>Hadar (10.1)<br>Typhimurium (8.5)<br>Infantis (2.0)<br>Enteritidis (1.0)<br>Bredeney (0.5)<br>Others (66.3) | (Busani *et al.*, 2005) |
| 2002-2004 | Republic of Ireland | Industry testing programme: Chicken | 528/18,782 (2.8) | N/R[3] | (Jordan *et al.*, 2006) |
| 1999-2000 | Spain | Chicken | 0/40 (0) | N/A | (Soriano *et al.*, 2001) |
| N/R | Spain | Retail: Carcasses, wings, legs, giblets, red sausages, white sausages, hamburgers | All samples 34/70 (48.6)<br>- carcasses 22/40 (55)<br>- wings 2/5 (40)<br>- legs 2/5 (40)<br>- giblets 2/5 (40)<br>- red sausages 2/5 (40)<br>- white sausages 3/5 (60)<br>- hamburgers 1/5 (20) | (n=34 positive samples)<br>Enteritidis (70.6)<br>Poona (23.5)<br>Worthington (2.9) | (Capita *et al.*, 2003) |
| N/R | Spain | Processor: Whole carcasses, portions (wings, breasts, legs) | Carcasses 1/30 (3.3)<br>Fresh portions 4/90 (4.4)<br>Frozen portions 4/45 (8.9) | N/R | (Reiter *et al.*, 2007) |
| N/R | Spain | Four processors: Whole carcasses | 56/150 (37.3) | (n=142 isolates)<br>Blockley (73.2)<br>Paratyphi B (16.9)<br>Bredeney (6.3)<br>Neftenbach (1.4)<br>Hadar (1.4)<br>Thompson (0.7) | (Sakaridis *et al.*, 2011) |
| 1998-2000 | Switzerland | Processor: Fresh chicken | 188/3,462 (5.4) | N/R | (Sauli *et al.*, 2003) |

| Year(s)[1] | Country | Samples tested | Number positive/ total (%) | *Salmonella* serotypes (%) | Reference |
|---|---|---|---|---|---|
| N/R | UK | Retail: Whole carcasses, breasts, pieces | 87/300 (29%)<br>- whole 50/95 (52.6)<br>- breast 31/95 (32.6)<br>- pieces 14.5% (n=110)[2] | N/R | (Harrison *et al.*, 2001) |
| 1998-2000 | UK: England | Retail: Whole carcasses | 60/241 (25%) | (n=60 samples)<br>Hadar (28)<br>Enteritidis PT4 (16)<br>Indiana (16)<br>Thomson (6.7)<br>Virchow (6.7)<br>Heidelberg (4.9)<br>Agona (3.3)<br>Anatum (3.3)<br>Bredeney (3.3)<br>Typhimurium DT104 (3.3)<br>Infantis (1.6)<br>Kentucky (1.6)<br>Livingstone (1.6)<br>Newport (1.6)<br>Worthington (1.6) | (Jørgensen *et al.*, 2002) |
| 1995-2000 | UK: Northern Ireland | Retail: Whole carcasses | 91/803 (11.3) | (n=130 isolates)<br>Most common serotypes only:<br>Bredeney (20)<br>Enteritidis (17.7)<br>Kentucky (12.3)<br>Bareilly (11.5) | (Wilson, 2002) |
| 2002 | UK: Northern Ireland | Retail: Legs, breasts | 3/205 (1.5) | Infantis, Tennessee, unknown serotype in group C1+C4 | (Soultos *et al.*, 2003) |
| 2005 | UK: Wales, Northern Ireland | Retail: Whole carcasses | 35/877 (4.0) | N/R | (Meldrum and Wilson, 2007) |
| **Chicken: North America** | | | | | |
| 2001 | Canada | Retail: Legs<br>Retail: Processed breast<br>Retail: Chicken wieners | 30/100 (30)<br>0/100<br>0/101 | (n=27 isolates)<br>Heidelberg (59.3)<br>Braenderup (11.1)<br>Enteritidis (7.4)<br>Kentucky (7.4) | (Bohaychuk *et al.*, 2006) |

| Year(s)[1] | Country | Samples tested | Number positive/ total (%) | *Salmonella* serotypes (%) | Reference |
|---|---|---|---|---|---|
| | | | | Schwarzengrund (3.7)<br>Thompson (3.7)<br>Mbandaka (3.7)<br>Typhimurium (3.7) | |
| 2004-2005 | Canada | Processors: Whole carcasses | 37.5% (n=1,295)[2] | N/R | (Bohaychuk *et al.*, 2009) |
| N/R | Canada | Retail: Raw, frozen nuggets and strips | 25/92 (27.2) | (n=33 isolates)<br>Heidelberg (51.5)<br>Kentucky (18.2)<br>Enteritidis (9.1)<br>Hadar (6.1)<br>Serotype 6,8:-e,n,x (6.1)<br>Indiana (3.0)<br>Infantis (3.0)<br>Mbandaka (3.0) | (Bucher *et al.*, 2007) |
| 2008 | Canada<br>(sentinel site) | Retail: Breasts | 60/185 (32.4) | (n=60 samples)<br>Kentucky (36.7)<br>Heidelberg (23.3)<br>Enteritidis PT8 (8.3)<br>Hadar (8.3)<br>Enteritidis PT13a (3.3)<br>Infantis (3.3)<br>Typhimurium DT135 (3.3)<br>Kiambu (1.7)<br>Mbandaka (1.7)<br>Montevideo (1.7)<br>Senftenburg (1.7)<br>Thompson (1.7)<br>Typhimurium DT104 (1.7)<br>Typhimurium DT108 (1.7)<br>Typhimurium DT208 (1.7) | (Cook *et al.*, 2009) |
| 1999-2000 | USA: Washington D.C. | Retail: Whole carcasses | 9/212 (4.2) | N/R | (Zhao *et al.*, 2001) |
| 2000 | USA | Randomly selected processors: Whole carcasses, mince | - carcasses 9.1% (n=10,057)[3]<br>- mince 57/414 (13.8) | N/R | (Rose *et al.*, 2002) |

| Year(s)[1] | Country | Samples tested | Number positive/ total (%) | *Salmonella* serotypes (%) | Reference |
|---|---|---|---|---|---|
| N/R | USA | Retail: Whole carcasses | 85/251 (33.9) | N/R | (Simmons *et al.*, 2003) |
| 2004-2005 | USA | Processor: Whole carcasses | 202/240 (84.2) | (n=202 positive samples)<br>Most often isolated:<br>Kentucky (67.3)<br>Typhimurium (15.3)<br>Mbandaka (5.9) | (Parveen *et al.*, 2007) |
| 2005 | USA | 20 processors: Whole carcasses (post chill) | 161/798 (20.2) | (n=161 positive samples)<br>Two most commonly identified serotypes:<br>Kentucky (43.5)<br>Heidelberg (18.0) | (Berrang *et al.*, 2009) |
| 2007-2008 | USA (FSIS baseline survey) | 182 Processors: Whole carcasses (re-hang and post-chill) | 1,500/3,275 (45.8) Re-hang<br>267/3,275 (8.2) Post-chill | (n=1,174 isolates)<br>Kentucky (59.9)<br>Heidelberg (18.4)<br>Typhimurium (11.8)<br>Typhimurium (Copenhagen) (10.0) | (FSIS, 2008) |
| **Chicken: Other countries** | | | | | |
| 2004 | Brazil | Processors: Whole carcasses | 25/260 (9.6) | (n=20 isolates)<br>Most often isolated:<br>Enteritidis (25) | (Duarte *et al.*, 2009) |
| N/R | Brazil | Retail: Whole carcasses | 0/127 | N/A | (de Freitas *et al.*, 2010) |
| 2006-2007 | Cambodia | Retail: Whole carcasses | 134/152 (88.2) | (n=201 isolates)<br>Most often isolated:<br>Anatum (6.5)<br>Typhimurium (6.5)<br>Corvallis (6.0)<br>Stanley (5.5)<br>Enteritidis (5.0)<br><br>Different quantitative contaminations of *Salmonella* were displayed by 34 samples (22.4%) at 3-4 log10CFU/g, 56 samples (36.8%) at 2-3 log10CFU/g, 32 samples (21.1%) at 1-2 | (Lay *et al.*, 2010) |

| Year(s)[1] | Country | Samples tested | Number positive/ total (%) | *Salmonella* serotypes (%) | Reference |
|---|---|---|---|---|---|
| | | | | log10CFU/g, and 12 samples (7.9%) at 0-1 log10CFU/g | |
| 2005 | China | Retail: Chicken | 19/120 (15.8) | N/R[4] | (Yan *et al.*, 2010) |
| 2007-2008 | China | Retail: Chicken | 276/515 (53.6) | (n=292 isolates)<br>Most often isolated:<br>Enteritidis (35.6)<br>Typhimurium (13.0)<br>Shubra (11.6)<br>Indiana (11.0)<br>Djugu (7.2)<br>Derby (5.1) | (Yang *et al.*, 2010) |
| 2006-2007 | Iran | Retail: Chicken | 86/190 (45.3) | (n=86 isolates)<br>Thompson (75.6)<br>Hadar (7.0)<br>Enteritidis (5.8)<br>Virginia (3.5)<br>Paratyphi C (2.3)<br>Typhimurium (1.2)<br>Untypable (4.7) | (Dallal *et al.*, 2010) |
| 2006-2008 | Japan | Retail: Chicken | 164/821 (20.0) | (n=452 isolates)<br>Most often isolated:<br>Infantis (17.9)<br>Kalamu (12.4)<br>Schwarzengrund (9.5) | (Iwabuchi *et al.*, 2011) |
| 2005-2006 | Morocco | Retail: breasts, legs, gizzards, livers | 57/576 (9.9)<br>- breasts 9/144 (6.3)<br>- legs 12/144 (8.3)<br>- gizzards 16/144 (11.1)<br>- livers 20/144 (13.9) | (n=57 samples)<br>Typhimurium (40.4)<br>Newport (26.3)<br>Montevideo (17.5)<br>Heidelberg (15.8) | (Abdellah *et al.*, 2009) |
| 2002-2004 | Saudi Arabia (Kingdom of) | Retail: Whole carcasses | 74/422 (17.5) | N/R[4] | (Saad *et al.*, 2007) |
| N/R | South Africa | Retail: Whole carcasses | 19/99 (19.2)<br>- fresh 11/66 (16.7)<br>- frozen 8/33 (24.2) | (n=19 isolates)<br>Hadar (31.6)<br>Blockley (10.5)<br>Irumu (10.5)<br>plus 9 additional serotypes | (van Nierop *et al.*, 2005) |

| Year(s)[1] | Country | Samples tested | Number positive/ total (%) | *Salmonella* serotypes (%) | Reference |
|---|---|---|---|---|---|
| 2000-2006 | Taiwan | Retail: Pieces ("buttocks", necks, wings, drumsticks) from marketplaces | 59% (n=508)[2] | Most common serotypes:<br>Albany, Schwarzengrund, Istanbul | (Chen *et al.*, 2010) |
| 2001 | Thailand | Retail: Thighs | 41/72 (56.9) | (n=20 isolates)<br>Weltevreden (55)<br>Emek (25)<br>Hadar (10) | (Padungtod and Kaneene, 2006) |
| N/R | Turkey | Retail: Poultry meat | 22/75 (29.3) | (n=22 positive samples)<br>Typhimurium (90.9) | (Arslan and Eyi, 2010) |
| N/R | Turkey | Retail: Whole carcasses, legs, wings, breasts, giblets | All samples 23/125 (18.4)<br>- carcasses 4/25 (16)<br>- legs 2/25 (8)<br>- wings 3/25 (12)<br>- breasts12/25 (48)<br>- giblets 2/25 (8) | N/R | (Vural *et al.*, 2006) |
| **Turkey: All countries** | | | | | |
| 2008 | EU Member States (MS) | Non-ready-to-eat turkey products | 5.6% of 3,134 samples[2]<br>- at slaughter (2 MS) 2.8-4.0%<br>- at processing/cutting plant (5 MS) 0-17.0%<br>- at retail (6 MS) 2.6-17.9% | N/R | (EFSA, 2010a) |
| 2009 | EU Member States (MS) | Non-ready-to-eat turkey products | 8.7% of 3,953 samples[2]<br>- at slaughter (4 MS) 0-20.7%<br>- at processing/cutting plant (6 MS) 0-19.2%<br>- at retail (5 MS) 0-11.8% | N/R | (European Food Safety Authority and European Centre for Disease Prevention and Control, 2011) |
| 2005-2008 | Morocco | Retail: Mince | 39/192 (20.3) | Kentucky (20.5)<br>Corvallis (15.3)<br>Muenster (12.8)<br>Newport (12.8)<br>Typhimurium (5.1)<br>10 other serotypes (each 1%) | (Karraouan *et al.*, 2010) |
| 2002-2004 | Republic of Ireland | Industry testing programme: Turkey | 26/832 (3.1) | N/R[3] | (Jordan *et al.*, 2006) |

| Year(s)[1] | Country | Samples tested | Number positive/ total (%) | *Salmonella* serotypes (%) | Reference |
|---|---|---|---|---|---|
| 1999-2000 | USA: Washington D.C. | Retail: Breasts | 5/194 (2.6) | N/R | (Zhao *et al.*, 2001) |
| 2000 | USA | Randomly selected processors: Mince | 25.7% (n=1,551)[2] | N/R | (Rose *et al.*, 2002) |
| 2008-2009 | USA (FSIS baseline survey) | 58 Processors: Whole carcasses (post-chill) | 24/1,442 (1.7) | (n=17 isolates)<br>Hadar (76.5)<br>Albany (11.8)<br>Heidelberg (11.8) | (FSIS, 2009) |
| **Duck: All countries** | | | | | |
| 2002-2004 | Republic of Ireland | Industry testing programme: Duck | 4/281 (1.4) | N/R[4] | (Jordan *et al.*, 2006) |

N/R, Not Reported (includes studies where the serotypes were not analysed)
N/A, Not Applicable
FSIS, Food Safety Inspection Service of the United States Department of Agriculture
[1] Year or years survey was done, or if this information is unavailable, the year of publication.
[2] Only the total number of samples tested and the percentage positive for *Salmonella* is reported. The number of samples positive was not able to be calculated accurately from these data.
[3] The chicken, turkey and duck serotypes are not reported separately. The *Salmonella* serotypes most often isolated were Bredeney, Enteritidis, Infantis, Kentucky, Livingstone, Mbandaka and Typhimurium (mostly DT104).
[4] The serotypes isolated from chicken samples were not reported separately from those isolated from other sample types.

## Table 14: Prevalence of *Salmonella* species in ready-to-eat poultry products

| Year(s)[1] | Country | Samples tested | Number positive/total (%) | *Salmonella* serotypes (%) | Reference |
|---|---|---|---|---|---|
| **Chicken** | | | | | |
| 2002 | Australia: Australian Capital Territory | Whole kebabs containing chicken cooked on a vertical spit | 0/36 (0) | N/A | (Rockliff and Khan, 2002) |
| 2001 | Canada | Chicken wieners | 0/101 | N/A | (Bohaychuk *et al.*, 2006) |
| 2008 | EU Member States (MS) | Ready-to-eat broiler meat product samples | 1.1% of 3,402 samples[2]<br>- at processing plant (5 MS) 0-2.8%<br>- at retail (10 MS) 0-5.6% | N/R | (EFSA, 2010a) |
| 2009 | EU Member States (MS) | Ready-to-eat broiler meat product samples | 0.2% of 3,284 samples[2]<br>- at processing plant (7 MS) 0-0.1%<br>- at retail (10 MS) 0-3.5% | N/R | (European Food Safety Authority and European Centre for Disease Prevention and Control, 2011) |
| N/R | Senegal | Pooled sample from three whole servings of chicken meat from each of 42 restaurants | 6/42 (14.3) | Most commonly isolated: Istanbul, Kentucky | (Dione *et al.*, 2009) |
| 1999-2000 | Spain | Chicken | 0/40 (0) | N/A | (Soriano *et al.*, 2001) |
| 1998-2004 | Spain | Frozen chicken croquettes<br>Duck liver pate<br>Cooked turkey breast | 1/65 (1.5)<br>0/23<br>0/15 | N/R | (Cabedo *et al.*, 2008) |
| 2003-2005 | UK: Wales | Rotisserie chicken | Detected in 25g:<br>0/117 | N/A | (Meldrum *et al.*, 2006) |
| **Turkey** | | | | | |
| 2001 | Canada | Processed breast | 0/100 | N/A | (Bohaychuk *et al.*, 2006) |
| 2008 | EU Member States (MS) | Ready-to-eat turkey products | 3/675 (0.4)<br>- at processing/cutting plant (3 MS) 0-1.2%<br>- at retail (4 MS) 0-1.6% | N/R | (EFSA, 2010a) |
| 2009 | EU Member States (MS) | Ready-to-eat turkey products | 0.8% of 2,171 samples[2]<br>- at processing/cutting plant (4 | N/R | (European Food Safety Authority |

| Year(s)[1] | Country | Samples tested | Number positive/total (%) | *Salmonella* serotypes (%) | Reference |
|---|---|---|---|---|---|
| | | | MS) 0-6.7%<br>- at retail (4 MS) 0-1.2% | | and European Centre for Disease Prevention and Control, 2011) |
| 1998-2004 | Spain | Cooked breast | 0/15 | N/R | (Cabedo *et al.*, 2008) |
| **Duck** | | | | | |
| 1998-2004 | Spain | Duck liver pate | 0/23 | N/R | (Cabedo *et al.*, 2008) |

N/R, Not Reported
N/A, Not Applicable
[1] Year or years survey was done, or if this information is unavailable, the year of publication.
[2] Only the total number of samples tested and the percentage positive for *Salmonella* is reported. The number of samples positive was not able to be calculated accurately from these data.

## Table 15: Quantitative data for *Salmonella* species in raw poultry products

| Year(s)[1] | Country | Samples tested | Counts (percent of samples) CFU/g (unless otherwise stated) | Reference |
|---|---|---|---|---|
| **Chicken** | | | | |
| N/R | N/R | N/R | Typically 1-30 cells with occasionally up to 104 CFU per 100g of broiler skin (paraphrased) | (Bryan and Doyle, 1995) |
| 1993-1996 | Belgium | Distribution centre: Whole carcasses, pieces (legs, wings, breast, fillets), processed (sausages, hamburgers, sliced on a skewer, coated with spices) | Whole[2]<br>- broiler >1/100cm$^2$ (22.3)<br>>1/cm$^2$ (15.0)<br>- broiling hen >1/100cm$^2$ (39.0)<br>>1/cm$^2$ (25.0)<br>- spring chicken >1/100cm$^2$ (17.7)<br>>1/cm$^2$ (4.8)<br>- guinea fowl >1/100cm$^2$ (18.7)<br>>1/cm$^2$ (20.0)<br><br>Pieces:[2] >1/25cm$^2$ or g (41.8)<br>- broiler >1/cm$^2$ or g (20.3)<br><br>Processed:[2] >1/25g (35.2)<br>- broiler >1/g (23.0) | (Uyttendaele *et al.*, 1998) |
| 2006-2007 | Cambodia | Retail: Whole carcasses | (n=152)<br>Not detected (11.8)<br>0-1 $\log_{10}$ CFU/g (7.9)<br>1-2 $\log_{10}$ CFU/g (21.1)<br>2-3 $\log_{10}$ CFU/g (36.8)<br>3-4 $\log_{10}$ CFU/g (22.4) | (Lay *et al.*, 2010) |
| 1998-2000 | England | Retail: Whole carcasses (chilled and frozen) | 1998-99 Carcass rinse + whole skin (n=101)<br><800 CFU/sample (100)<br>4.5 $\log_{10}$ CFU/sample (1)<br><br>1999-00 Carcass rinse + neck skin (n=140)<br><300 $\log_{10}$ CFU/sample (140) | (Jørgensen *et al.*, 2002) |
| N/R | France | Processors: Chicken skin | Traditional MPN:<br>- mean 5.7 MPN/cm$^2$<br>- range 0.2-95,300 MPN/cm$^2$<br>Miniature MPN :<br>- mean 12 MPN/cm$^2$<br>- range 0.9-5,556 MPN/cm$^2$ | (Humbert *et al.*, 1997) |
| 1996 | Ireland | Retail: Chicken | (n=106)<br><0.7 $\log_{10}$ (73.6)<br>0.7-1.0 $\log_{10}$ (18.9)<br>1.0-1.5 $\log_{10}$ (1.9)<br>1.5-2.0 $\log_{10}$ (4.7)<br>2.0-2.5 $\log_{10}$ (0.9) | (Duffy *et al.*, 1999) |

| Year(s)[1] | Country | Samples tested | Counts (percent of samples) CFU/g (unless otherwise stated) | Reference |
|---|---|---|---|---|
| N/R | The Netherlands | Retail: Whole carcasses (fresh and frozen), portions | Fresh (n=45; 38 were whole)<br>0-10 MPN/carcass (89)<br>11-100 MPN/carcass (9)<br>101-1,100 MPN/carcass (0)<br>>1,100 MPN/carcass (2)<br><br>Frozen (n=44)<br>0-10 MPN/carcass (68)<br>11-100 MPN/carcass (23)<br>101-1,100 MPN/carcass (4)<br>>1,100 MPN/carcass (2) | (Dufrenne *et al.*, 2001) |
| 2005 | The Netherlands | Retail: Chilled filets | (n=220)<br><1 log MPN/filet (91.4)<br>1.00 log MPN/filet (3.2)<br>1.41 log MPN/filet (1.4)<br>1.65 log MPN/filet (0.5)<br>1.81 log MPN/filet (0.9)<br>2.00 log MPN/filet (0.5)<br>2.08 log MPN/filet (0.5)<br>2.83 log MPN/filet (0.9)<br>3.81 log MPN/filet (0.9) | (Straver *et al.*, 2007) |
| 2007-2008 | USA (FSIS baseline survey) | 182 Processors: Whole carcasses (re-hang and post-chill) | Re-hang (n=1,333)<br>0.0301-0.3 MPN/ml (41.5)<br>0.301-3.0 MPN/ml (33.7)<br>3.01-30.0 MPN/ml (12.9)<br>30.01-300.0 MPN/ml (0.47)<br>Undetermined (0.27)<br><br>Post-chill (n=170)<br>0.0301-0.3 MPN/ml (46.1)<br>0.301-3.0 MPN/ml (14.2)<br>3.01-30.0 MPN/ml (3.4) | (FSIS, 2008) |
| **Turkey** | | | | |
| 1993-1996 | Belgium | Distribution centre: Pieces (legs, wings, breast, fillets) | >1/25cm$^2$ or g (10.8)<br>>1/cm$^2$ or g (7.5) | (Uyttendaele *et al.*, 1998) |
| 2008-2009 | USA (FSIS baseline survey) | 58 Processors: Whole carcasses (re-hang, post-chill) | Re-hang (n=72)<br>0.075-0.750 MPN/cm$^2$ (75.0)<br>0.751 - 7.50 MPN/cm$^2$ (15.3)<br>7.51 - 75.0 MPN/cm$^2$ (8.3)<br>Undetermined (1.4)<br><br>Post-chill (n=5)<br>0.075-0.750 MPN/cm$^2$ (80.0)<br>0.751 - 7.50 MPN/cm$^2$ (20.0) | (FSIS, 2009) |
| **Duck** | | | | |
| No studies identified. | | | | |

N/R, Not Reported

FSIS, Food Safety Inspection Service of the United States Department of Agriculture

[1] Year or years survey was done, or if this information is unavailable, the year of publication.

[2] Broiler = 6-8 weeks old if produced conventionally, 12-13 weeks old if free-range; broiling hen = ≤2 years old; spring chicken = 4-6 weeks old; guinea fowl = ≤13 weeks old.

#### *7.5.1.1 Australian survey data from poultry flocks*

FSANZ led a national survey during 2007 and 2008 to collect baseline data on the prevalence and concentration of *Salmonella* and *Campylobacter* on poultry and poultry meat at various stages along the supply chain in Australia (FSANZ, 2010a). The baseline data will be used as a comparison for follow-up surveys to be undertaken after the new FSANZ poultry standard comes into effect in 2012 (for further details on the standard, see Section 9.2, Appendix 3). The survey measured the prevalence and, where appropriate, concentration, of *Salmonella* at three points along the chicken meat supply chain: on-farm, just prior to processing, and at the end of primary processing.

Researchers tested pooled faecal samples from 233 sheds, from 39 farms. *Salmonella* was detected on 33/39 farms (84.6%, 95% CI 69.5-94.1) and in 109/233 faecal samples (46.8%, 95% CI 40.2-53.4). Two samples were positive for *S.* Sofia, a serotype commonly isolated from poultry in Australia but thought to be of low virulence to humans, but all 109 positive samples were positive for non-Sofia serotypes (most commonly *S.* Mbandaka, *S.* Livingstone and *S.* Havana).

After evisceration at a poultry processing plant, 636 caecal samples were collected from poultry and their contents analysed. The *Salmonella* prevalence was 81/636 (12.7%, 95% CI 10.2-15.6). The mean concentration of *Salmonella* detected from positive samples was 1.02 $\log_{10}$ MPN/g (approximately 10 MPN/cm$^2$). Non-Sofia serotypes (commonly *S.* Infantis and *S.* Typhimurium) were detected in 48/81 (59.3%) positive samples and *S.* Sofia was detected in 33/81 (40.7%) positive samples.

After spin-chilling at the end of processing, 1,112 carcass rinse samples were tested. *Salmonella* was detected from 408/1,112 (36.7%, 95% CI 33.9-39.6) samples. The mean concentration of *Salmonella* detected in positive samples was -1.99 $\log_{10}$ MPN/cm$^2$ (approximately 0.01 MPN/cm$^2$).[29] Non-Sofia serotypes (commonly *S.* Typhimurium, *S.* Infantis, *S.* Kiambu, *S.* Muenster and *S.* Agona) were detected in 246/408 (60.3%) positive samples and *S.* Sofia was isolated from 168/408 (41.2%) positive samples.

#### *7.5.1.2 European survey data from poultry flocks and carcasses*

The European Food Safety Authority (EFSA) collates data on compliance with EU regulations set out for testing *Salmonella* in different food product groups during their shelf life, and different foods during processing (see Section 9.3, Appendix 3, for further details on these regulations). The 2009 compliance data relevant for poultry products during their shelf life are presented in Table 17. Of all the food product groups monitored by the EFSA, mechanically separated meat and meat products from poultry intended to be eaten cooked had the highest levels of non-compliance at batch level (1.2% and 1.0%, respectively) (European Food Safety Authority and European Centre for Disease Prevention and Control, 2011).

[29] The post processing (rinse) data for *Salmonella* was converted to log10 MPN/cm2, according to the formulae for carcasses in the Australian Standard, AS 5013.20–2004 *Method 20: Preparation of test samples for microbiological examination-Poultry and poultry products*.

**Table 16: Compliance of EU member states with EU Regulations (EC) No 2073/2005 and No 1441/2007 applicable to *Salmonella* in poultry, 2008**

| Food category | Total single samples | | | Total batches | | |
|---|---|---|---|---|---|---|
| | Sample weight | N | % non-compliant | Sample weight | N | % non-compliant |
| Minced meat and meat preparations from poultry to be eaten cooked | 10g or 25g or not stated | 1,870 | 8.7 | 10g or 25g or 200g or not stated | 11,949 | 1.0 |
| Meat products from poultry meat intended to be eaten cooked | 10g or 25g or not stated | 3,781 | 0.8 | 10g or 25g or not stated | 9,269 | 0.5 |
| Meat products intended to be eaten raw | 25g | 1,263 | 1.7 | 25g | 159 | 0.6 |
| Minced meat and meat preparations to be eaten raw | 25g | 3,043 | 1.2 | 10g or 25g or 200g or not stated | 7,132 | 0.6 |
| Mechanically separated meat | 25g or 250g | 156 | 0 | 10g or 25g or 100g or 200g or not stated | 2,516 | 1.2 |

Source: (European Food Safety Authority and European Centre for Disease Prevention and Control, 2011)

EU Member States (MS) must also implement a *Salmonella* control programme for flocks (see Section 9.3, Appendix 3) and report surveillance data annually. These data can be compared with baseline surveys conducted prior to setting the new EU targets (EFSA, 2007, 2008).

Twenty MS and one non-MS reported *Salmonella* surveillance data for parent breeding flocks of chickens used for meat production during 2009 (European Food Safety Authority and European Centre for Disease Prevention and Control, 2011). Eight countries did not report any positive flocks, whereas the other countries reported *Salmonella* prevalences of 0.8% to 10.6%. Five countries reported prevalences higher than the 1% target set for five serotypes. In the same year, 18 MS and two non-MS reported *Salmonella* surveillance data for broiler flocks. Two countries reported no positive broiler flocks, and the other countries reported prevalences of <0.1 to 32.4%. Overall, a total of 182,271 broiler flocks were tested from the MS, of which 5.0% were *Salmonella*-positive (0.7% were positive for *S.* Enteritidis and/or *S.* Typhimurium).

These results appear favourably against the 2005-06 baseline study on broiler flocks, which was based on 6,325 holdings corresponding to 7,440 flocks (EFSA, 2007). At EU Community level 23.7% of flocks tested positive for *Salmonella*, i.e. one in four broiler flocks raised over the one year period of the baseline survey was *Salmonella*-positive. The *Salmonella* prevalence varied amongst the Member States, from 0% to 68.2%. A total of 11.0% of the broiler flocks was estimated to be positive for *Salmonella* Enteritidis and/or *Salmonella* Typhimurium (MS range 0-39.3%). The five most frequently isolated *Salmonella* serotypes were *S.* Enteritidis, *S.* Infantis, S. Mbandaka, *S.* Typhimurium and *S.* Hadar, but the distribution of these types varied amongst the Member States.

The 2006-07 baseline study on turkey flocks included every MS, including Bulgaria and Romania (EFSA, 2008). A total of 539 breeding turkey flocks and 3,769 fattening turkey flocks were included in the survey analyses. The Community observed prevalence of *Salmonella*-positive flocks was 13.6% in breeding turkeys, although 8/14 MSs did not isolate *Salmonella* in their breeding flocks. The *Salmonella* prevalence in these flocks varied amongst the MSs, from 0% to 82.9%. The Community observed prevalence of *Salmonella*-positive fattening flocks was 30.7%, i.e. one in three fattening turkey flocks raised over the one year period of the baseline survey was *Salmonella*-positive. The *Salmonella* prevalence in these flocks ranged amongst the MSs from 0% to 78.5%.

Nine EU countries reported information from the routine monitoring of turkey breeding flocks in 2009 (European Food Safety Authority and European Centre for Disease Prevention and Control, 2011). *Salmonella* was only detected in two countries (Czech Republic and Poland). Eight MSs and one non-MS provided data on *Salmonella* surveillance of turkey production flocks; the reported prevalence range was 0-11.2%. Overall, a total of 2,707 turkey production flocks were tested from MS, of which 7.1% were *Salmonella*-positive (1.8% were positive for *S.* Enteritidis or *S.* Typhimurium).

*Salmonella* surveillance data for duck breeding flocks in 2008 were also reported by five EU countries; three countries detected positive flocks (Ireland, Poland, Slovakia) (European Food Safety Authority and European Centre for Disease Prevention and Control, 2011). *Salmonella* prevalence data from duck production flocks, as reported by four MS, ranged from 4.2% to 63.5% (this high value was reported by Denmark and none of the serotypes isolated were *S.* Typhimurium or *S.* Enteritidis). Overall, a total of 358 duck production flocks were tested of which 22.1% were *Salmonella*-positive (5.6% were positive for *S.* Enteritidis or *S.* Typhimurium).

Ten EU MSs reported data on *Salmonella* serotypes identified from broiler meat during 2009 (European Food Safety Authority and European Centre for Disease Prevention and Control, 2011). As in 2008 (EFSA, 2010a), of the isolates that were serotyped (2,585 in 2008, 1,349 in 2009), *S.* Infantis was most often identified (40.1% in 2008, 50.9% in 2009). Fifteen EU MSs provided data on *Salmonella* serotypes identified in chicken flocks (breeders, layers and broilers) for 2009 (European Food Safety Authority and European Centre for Disease Prevention and Control, 2011). Of 10,531 isolates, 24.5% were *S.* Infantis and 18.5% were *S.* Enteritidis. Other relevant EU data on *Salmonella* in poultry has been captured in Table 13 and Table 15. Data from the 2008 baseline survey on *Salmonella* on broiler carcasses (EFSA, 2010b) appears in Table 13.

#### *7.5.1.3 USA baseline data and surveys*

The USDA FSIS collects data on the prevalence of *Salmonella* in raw poultry products as part of monitoring against the *Salmonella* performance standards (see Section 9.5, Appendix 3).[30] The results from the last three years (2008-2010) are presented in Table 17. For each of these years, only the very small establishments (as a combined group) exceeded the performance standard for broiler carcasses. Together, all establishments met all performance

[30] The results from many of FSIS's data collection activities are available from http://www.fsis.usda.gov/Science/Microbiology/index.asp (accessed 20 October 2011).

standards each year. The serotypes Kentucky, Enteritidis, Heidelberg and Typhimurium were most commonly identified in each of these years (FSIS, 2010b).

**Table 17: Prevalence of *Salmonella* in the USDA/FSIS PR/HACCP verification testing programme (2008-2010)**

| Year | Performance standard (% positive) | Percentage of samples positive for *Salmonella* (number of samples analysed)[1] | | | |
|---|---|---|---|---|---|
| | | Large establishments | Small establishments | Very small establishments | All size establishments |
| **Broiler carcasses** | | | | | |
| 2008 | 20.0 | 5.9 (4,694) | 10.0 (1,644) | 21.6 (125) | 7.3 (6,514)[2] |
| 2009 | 20.0 | 5.0 (4,605) | 11.7 (1,653) | 21.0 (181) | 7.2 (6,439) |
| 2010 | 20.0 | 4.3 (4,753) | 11.5 (1,956) | 25.8 (120) | 6.7 (6,829) |
| **Ground chicken** | | | | | |
| 2008 | 44.6 | 32.4 (145) | 19.2 (213) | 32.1 (53) | 25.5 (411) |
| 2009 | 44.6 | 30.4 (46) | 11.6 (276) | 42.3 (52) | 18.2 (374) |
| 2010 | 44.6 | 22.5 (89) | 16.3 (312) | 36.0 (25) | 18.8 (426) |
| **Ground turkey** | | | | | |
| 2008 | 49.9 | 16.9 (764) | 7.1 (84) | 0.0 (28) | 15.4 (876) |
| 2009 | 49.9 | 11.8 (423) | 9.4 (128) | 5.3 (57) | 10.7 (608) |
| 2010 | 49.9 | 11.6 (658) | 7.8 (154) | 1.6 (61) | 10.2 (873) |
| **Turkey carcasses** | | | | | |
| 2008 | 19.6[3] | 3.89 (77) | 9.6 (52) | 0 (0) | 6.2 (129) |
| 2009 | 19.6[3] | 3.5 (931) | 4.2 (501) | 0 (0) | 3.8 (1,432) |
| 2010 | 19.6[3] | 4.9 (1,049) | 3.8 (395) | 0 (0) | 4.6 (1,444) |

Source: (FSIS, 2011)

1. Large establishment: 500 or more employees on January 26, 1998; small establishment: 10-499 employees on January 25, 1999; very small establishment: <10 employees *or* annual sales of <$2.5 million on January 25, 2000 (FSIS, 1996).
2. Includes 51 samples from processing premises of unknown size.
3. Baseline guidance only (FSIS, 2006).

FSIS also conducts baseline surveys to estimate the national prevalence of *Salmonella* on the poultry products covered in Table 17, and to inform performance standards for industry. FSIS have done these since the early 1990's, and the latest results available for young chickens were collected 2007/08, and data on young turkeys was collected 2008/09 (these data are incorporated into Table 13 and Table 15).

FSIS also has two microbiological testing programs in place for Ready-To-Eat (RTE) poultry products (Mamber, 2010):

- ALLRTE: Sampling of all RTE poultry (and meat) products, initiated in January of 2004. Establishments and products are sampled at random.
- RTE001: Sampling of RTE poultry based on the risk characteristics of the producing establishment, initiated in January 2005. The selection of establishments for sample

collection and testing is made each month using a risk-ranking multivariate equation or algorithm based on control of *Listeria monocytogenes*.

From 2005 through 2008, 11,822 RTE samples were tested for *Salmonella* species under the ALLRTE sampling program and 33,276 samples were tested under the RTE001 sampling program. A subset of these samples contain poultry (Mamber, 2010). Overall, 8 (0.07%) of the ALLRTE samples and 14 (0.04%) of the RTE001 samples tested positive for *Salmonella* species The positive samples that included poultry were:

- Patties (sausage and chicken) (2 samples);
- Chicken and cheese burrito (1 sample);
- Chicken casserole (1 sample);
- Breaded chicken (1 sample); and
- Smoked chicken (1 sample).

### 7.5.2 Packaging surveys

The packaging of 140 whole chickens sampled from English retail stores in 1999/2000 was tested for *Salmonella* species (Jørgensen *et al.*, 2002). The researchers swabbed the outside of each chicken pack then removed the chicken and rinsed entire packaging, and enriched the swab and packaging rinsates. They also enumerated a portion of the whole package rinsate. No *Salmonella* were detected in the enumerated samples (<250 CFU/chicken), but 9/140 (6.4%) of the swab samples and 25/140 (17.9%) of the whole package rinses were *Salmonella*-positive.

In another UK study, retail packs of fresh raw meat were collected in 2002, and swabbed to detect the presence of *Salmonella* species (Burgess *et al.*, 2005). *Salmonella* species were detected on only 2/895 (0.2%) packs of chicken. *Salmonella* species were not detected on 129 packs of turkey and 28 packs containing game fowl.

### 7.5.3 Recalls

This section provides a summary of food recalls from Australia, Canada, the EU, the UK and the USA, where poultry products have been recalled because they may be contaminated with *Salmonella*. Recalls are not necessarily linked to human illness. These data indicate how often recalls have been issued for poultry products that were potentially contaminated with *Salmonella*. Poultry or poultry products have also been recalled because of possible contamination with other contaminants or hazards, or non-compliant labelling, but these data are not relevant to this Risk Profile and are excluded.

It was necessary to take different approaches with each recall database since these operate in different ways. Searches were restricted to the period January 2006 to the most up-to-date information available (the searches were conducted in May 2010), except for Australia, where records back to 2000 were examined. The sources and methods used to retrieve the recall data were as follows:

Australia: Food recalls recorded by FSANZ from 2000 to April 2010 were scanned for relevant records.[31]

Canada: All recalls reported by the Canadian Food Inspection Agency from January 2006 to April 2011 were scanned for relevant records.
(Source: http://www.inspection.gc.ca/english/corpaffr/recarapp/recal2e.shtml).

EU: A search function (portal) was used to retrieve records from the Rapid Alert System for Food and Feed, from January 2006. There are 31 countries that participate in this system (including the UK).[32]
(Source: https://webgate.ec.europa.eu/rasff-window/portal/)

UK: All recalls reported by the UK Food Standards Agency from January 2006 to April 2011 were examined for relevant records.
(Source: http://www.food.gov.uk/enforcement/alerts/)

USA: All recalls reported by the US Food and Drug Administration from January 2006 to April 2011 were scanned for relevant records.
(Source: http://www.fda.gov/Safety/Recalls/default.htm)

Only one relevant Australian recall was identified from the FSANZ data. The recall was issued in November 2001 and was for possible *Salmonella* contamination of a chicken and vegetable pie product. Relevant recalls from Canada, and the UK and USA are listed in Table 18.

Over 100 relevant recalls from the EU were identified for the period January 2006 - April 2011, often for product recalled in multiple countries. Most recalls were for processed poultry products.

**Table 18: Recalls of poultry or poultry products due to the possibility of *Salmonella* contamination: Canada, UK and the USA (January 2006-April 2011)**

| Country/countries where product recalled | Date of recall notice (month, year) | Product | Product country of origin |
|---|---|---|---|
| Canada | March 2007 | Cooked seasoned sliced turkey breast | Canada |
| Canada | April 2010 | Chicken soup mix, chicken noodle soup mix | Canada |
| UK | December 2008 | Pre-packed sliced turkey salami | Not stated |
| UK | August 2008 | Chicken and bacon sandwich filler[1] | Ireland |
| USA | January 2009 | Indonesian chicken with coconut rice, and chicken satay & Bangkok peanut sauce with jasmine rice meals[2] | USA |
| USA | January 2009 | Chicken pad Thai and spicy kung pao chicken meals[2] | Not stated |
| USA | February 2009 | Chicken pad Thai[2] | USA |

[1] The bacon used in this product was potentially contaminated with *Salmonella*.
[2] The peanut butter used in these meals was potentially contaminated with *Salmonella*.

[31] The FSANZ website (http://www.foodstandards.gov.au) only displays recalls from the previous 12 months. The full dataset from 2000 through September 2010 was kindly provided by FSANZ in September 2010.
[32] Search function parameters entered: Notified between 01/01/2006 and 30/04/2011; Type = Food; Classification = alert; Category = poultry meat and poultry meat products; Category = pathogenic micro-organisms. This search retrieved 135 records (119 relevant).

### 7.5.4 Risk assessments overseas

There have been several models published that estimate the probability of salmonellosis through exposure to poultry products.[33]

A quantitative risk assessment model for *Salmonella* and whole chickens was published in 2004 (Oscar, 2004b). The model covered the retail-to-table pathway, calculating the risk of salmonellosis through consumption of chicken from data on contamination at retail, growth during consumer transport, thermal inactivation during cooking, cross-contamination during serving and dose response after consumption. The model predicted a mean of 17.8 salmonellosis cases per 1,000,000 chickens consumed, or 0.44 cases of salmonellosis per 100,000 consumers of chicken.

For 766 outbreaks from 1996-2000 in England and Wales a single vehicle of infection was identified by epidemiologic or microbiologic investigations, and the causative agent was identified. Of these, 478 were caused by salmonellae. 108 salmonellosis outbreaks were linked to poultry. The food-specific risk was then calculated using food consumption data (Adak *et al.*, 2005). The disease risk from poultry was calculated as 140 cases/1 million servings (chicken = 111, turkey = 157 and mixed/unspecified poultry = 24). The hospitalisation risk was calculated as 2,063 per 1 billion servings (chicken = 2,518, turkey = 645 and mixed/unspecified poultry = 852). Risks were not calculated specifically for salmonellosis linked to poultry, and although the authors recognised the relative prominence of *Salmonella* and *Campylobacter* in illness attributed to chicken, they postulated that the greatest challenge to protect the population from foodborne infection was the develop effective programs to control *Campylobacter* in chicken production.

The Codex Committee on Food Hygiene (CCFH) Working Group on Guidelines for control of *Campylobacter* and *Salmonella* species in broiler (young bird) chicken meat has published a Risk Profile for *Salmonella* species in broiler (young) chickens (CCFH, 2007). The Risk Profile concerns non-typhoidal *Salmonella* species and fresh broiler chicken meat (whole chicken carcasses and portions, excluding internal organs). The document compiles useful information on the hazard/risk combination and identified several data gaps, but does not attempt to estimate risk.

Data on the concentration of *Salmonella* on chicken filets sampled from retail outlets in The Netherlands has been used in a model to estimate the probability of illness from consuming lettuce that had been contaminated from a filet via a cutting board (Straver *et al.*, 2007). The researchers found that prevalence of low-contaminated servings (i.e. 0-10 *Salmonella*/filet) was high, but the risk of illness was small because of the low probability of illness due to this dose level. The risk increased as dose levels increased, but at high dose levels, the risk decreased again because of the rare occurrence of these servings. Their model indicated that approximately 66% of the annual predicted illnesses were caused by only 0.8% of filets that carried a concentration of *Salmonella* of 1,000 cells or more at retail. The researchers concluded that it is important to consider not only *Salmonella* prevalence, but also the number of *Salmonella* present to assess risks properly. Additionally, their results suggested that only a small number of poorly performing poultry producers may be responsible for a disproportionate public health risk.

[33] http://foodrisk.org/ is a website that collates risk assessment models.

# 8 APPENDIX 2: EVALUATION OF ADVERSE HEALTH EFFECTS

Salmonellae possess virulence determinants that enable them to adhere to small intestinal epithelial cells, provided they survive the low pH of the stomach and other innate immune host defence mechanisms (Jay *et al.*, 2003). After entering epithelial cells, pathogenic salmonellae may multiply within a protective vacuole. Disruption of cellular tight junctions, leading to paracellular passage of ions, water and immune cells together with induction of host inflammatory cells is likely to contribute to the production of diarrhoea (Haraga *et al.*, 2008).

Two serotypes that have caused major problems overseas are *S.* Enteritidis which is capable of transovarian transmission into eggs (especially phage type 4 (PT4)) and the antibiotic resistant *S.* Typhimurium definitive phage type 104 (DT104).

*S.* Enteritidis PT4 became the most prevalent *Salmonella* causing human infection in the United Kingdom during the 1980s and 1990s. This was, in part, due to the fact that chicken eggs can be infected with *S.* Enteritidis PT4 internally or externally by the time they are laid, or can subsequently become contaminated after lay (Advisory Committee on the Microbiological Safety of Food, 1993). Similar problems occurred in the USA, but involved a wider range of phage types.

New Zealand does not appear to have a reservoir of the phage types associated with transovarian egg contamination. The notified human cases of salmonellosis infected with *S.* Enteritidis PT4 have usually recently travelled overseas.

Antibiotic resistant *S.* Typhimurium DT104 is infrequently isolated from humans in New Zealand (52 isolates since 1992, including a small 3 case outbreak in 1997). Of the 52 human isolates 50 were multi-resistant. Since 1992, this serotype has only been isolated on 7 occasions from non-human sources (1 environmental, 1 poultry feed, 1 poultry environment, 3 canine and 1 feline). Three of the non-human isolates have been multi-resistant strains (Carolyn Nicol, ERL, personal communication June 2011).

## 8.1 Dose-Response

Gastric hydrochloric acid is an important barrier to preventing *Salmonella* species ingested with food or water from surviving to invade the cells of the small intestine (Smith, 2003). However, the survival of salmonellae in the stomach is enhanced when they are ingested with fatty or proteinaceous foods, or the pH of the stomach acid is increased (e.g. by antacids, or by medical conditions or interventions) (Kothary and Babu, 2001; Smith, 2003). In a stimulated stomach, one strain of *S.* Typhimurium survived longer when digested with scrambled egg than with lettuce (Koseki *et al.*, 2010). The authors postulated that ingested bacteria in the stomach would barely be inactivated in the real digestive process. Furthermore, if previously exposed to acidic pH, salmonellae can develop acid resistance that helps them survive exposure to gastric acid (a response which also enhances their survival in low acid foods) (Smith, 2003).

### 8.1.1 Dose-response from feeding trials

Results obtained through feeding studies usually indicate that consumption of a large number of organisms causes gastrointestinal disease. However, these studies are usually conducted by feeding the pathogens to healthy adult volunteers in liquids such as milk or sodium bicarbonate, and do not consider low doses (Bollaerts *et al.*, 2008; Kothary and Babu, 2001). Therefore the results do not necessarily indicate the true infective dose for the variety of individuals found in a normal population, or for foodborne transmission.

A set of volunteer feeding studies reported in 1951 (McCullough and Eisele, 1951a, 1951b, 1951c, 1951d), while criticised for various deficiencies (Oscar, 2004a; FAO/WHO, 2002), are commonly used to predict dose-response for salmonellosis. These studies observed illness after healthy young men ingested different doses of six *Salmonella* serotypes. In a review of these studies, Kothary and Babu (2001) reported that the infective dose ranged between $10^5$ and $10^{10}$ organisms depending on the *Salmonella* serotype. The attack rate was also serotype-dependant, and ranged from 16-50%.

A number of studies have used the McCullough and Eisele data to model dose-response. One model, combining data only from cases who had not previously participated in the experiments, predicted that a dose of $2.4 \times 10^4$ salmonellae would infect 50% of the population (FAO/WHO, 2002). Another approach investigated variability between the serotypes (Oscar, 2004a). These researchers predicted minimum illness doses that ranged from $6.0 \times 10^4$ of a *S.* Bareilly strain to $2.1 \times 10^9$ of a *S.* Pullorum strain. The minimum illness doses of the *S.* Derby and *S.* Newport strains were $7.6 \times 10^6$ and $1.7 \times 10^7$ organisms, respectively.

### 8.1.2 Dose-response from outbreak data

In contrast to human feeding trials, data from outbreaks suggest that the infective dose could be as high as $10^7$-$10^9$ salmonellae, or less than 100 salmonellae (Kothary and Babu, 2001). The high fat or protein content of the food vehicle (e.g. ice cream, chocolate, cheese, beef, egg) can help protect salmonellae from gastric acidity, and salmonellosis outbreaks often involve young children and the elderly who are more susceptible to *Salmonella* infection (Kothary and Babu, 2001; Waterman and Small, 1998). In a salmonellosis outbreak in the USA caused by contaminated ice cream, the infective dose may have been as low as 6 cells in 65 g of ice cream (Hennessy *et al.*, 1996). Similarly, ingestion of as few as 10 *S.* Typhimurium cells may have been sufficient to cause symptomatic disease in a USA outbreak caused by contaminated chocolate (Wilson and Baker, 2009). Across 31 outbreaks in Japan, the calculated dose ranged from 11 to $7.5 \times 10^9$ CFU/person (or from 31 to $3.8 \times 10^6$ CFU/person for the 13 outbreaks where food samples were definitely frozen before testing) (Kasuga *et al.*, 2004). An analysis of nine outbreaks in Japan suggested an inverse relationship between the dose ingested and the incubation period before symptoms become evident (Abe *et al.*, 2004).

Using outbreak data, FAO/WHO produced a dose-response model as an output from the joint risk assessments of *Salmonella* in eggs and broiler chickens (FAO/WHO, 2002). The model was based on 20 outbreaks in North America and Japan (12 *S.* Enteritidis, 3 *S.* Typhimurium, *S.* Heidelberg, *S.* Cubana, *S.* Infantis, *S.* Newport and *S.* Oranienburg) with vehicles of transmission that included meat, eggs, dairy products, cake, vegetables and water. The graph

shows that for the ingestion of $10^{10}$ cells there was in a probability of around 0.9 (90%) of illness, while the ingestion of $10^{1}$ cells resulted in a probability of around 0.02 (2%). The model also predicts that a dose of $10^{4}$ cells has a probability of illness of 50%. Thus the probability of illness from exposure to small doses is low. For outbreaks where food contains only low numbers of organisms but has been widely consumed, a small proportion of consumers are likely to become ill.

The FAO/WHO model has been developed further to account for differences in host susceptibility, serotype infectivity and food matrix (Bollaerts *et al.*, 2008). The FAO/WHO model separated the cases in each outbreak into normal and susceptible populations (e.g. children) where possible, but concluded that there was insufficient evidence to show that some segments of the population have a higher probability of illness. However, Bollaerts *et al*. (2008) (using the same data set) concluded that a susceptible population has a higher probability of illness at low dose levels when the combination of the pathogen and food matrix is extremely virulent (e.g. fatty foods), and at high dose levels when this combination is less virulent. The authors' analyses also suggested that there is some immunity in the normal population but not in the susceptible population.

A recent study (Teunis *et al.*, 2010) used data from 35 salmonellosis outbreaks, three sporadic cases for which there was good dose information and two human volunteer feeding studies (the doses ranged from <10 to $10^{11}$ organisms). From this wider data set, the researchers predicted that the number of cells that need to be ingested to cause a 50% probability of illness was as low as 36.3, although the 95% percentiles were widespread ($0.69\text{-}1.26 \times 10^{7}$). A 1% probability of illness was associated with 0.4 cells (0.01-89.7).

## 8.2 New Zealand Epidemiological Data

### 8.2.1 Incidence

Historical data for the incidence of notified salmonellosis in New Zealand is given in Table 20.

**Table 19: Incidence data for salmonellosis in New Zealand**

| Year | Number of cases | Incidence (cases/100,000) |
|---|---|---|
| 1985 | 1,234 | 38.9 |
| 1986 | 1,335 | 40.4 |
| 1987 | 1,140 | 34.5 |
| 1988 | 1,128 | 34.1 |
| 1989 | 1,860 | 56.2 |
| 1990 | 1,619 | 50.0 |
| 1991 | 1,244 | 36.9 |
| 1992 | 1,239 | 36.7 |
| 1993 | 1,340 | 39.7 |
| 1994 | 1,522 | 45.1 |
| 1995 | 1,334 | 39.5 |
| 1996 | 1,141 | 31.5 |
| 1997 | 1,177 | 35.3 |
| 1998 | 2,069 | 57.2 |

| Year | Number of cases | Incidence (cases/100,000) |
|---|---|---|
| 1999 | 2,077 | 57.5 |
| 2000 | 1,795 | 48.1 |
| 2001 | 2,417 | 64.7 |
| 2002 | 1,880 | 50.3 |
| 2003 | 1,401 | 37.5 |
| 2004 | 1,081 | 28.9 |
| 2005 | 1,382 | 37.0 |
| 2006 | 1,335 | 31.9 |
| 2007 | 1,275 | 30.1 |
| 2008 | 1,339 | 31.5 |
| 2009 | 1,128 | 26.2 |
| 2010 | 1,146 | 26.2 |

Number of cases data taken from ESR, 2010a, Population data for June each year taken from Statistics New Zealand (http://www.stats.govt.nz/methods_and_services/access-data/tables/national-pop-estimates.aspx). Due to population adjustments by Statistics New Zealand rates may differ slightly from older Annual Surveillance Summary reports.

### 8.2.2 Outbreaks where poultry or poultry products were listed as a suspected food

Relevant outbreaks, extracted from EpiSurv, are summarised in Table 20.

**Table 20: New Zealand non-typhoid salmonellosis outbreaks where poultry or poultry products were a suspected or confirmed source of infection, 2000-2010**

| Year[1] | *Salmonella* serotype | Food(s) reported | Setting | Number of cases[2] | Evidence[3] |
|---|---|---|---|---|---|
| 2000 | (not identified) | chicken burritos | Restaurant/café | 2P | History |
| 2000 | Typhimurium 135 | country fried chicken, chicken rolls and sandwiches | Bakery | 11C | History |
| 2000 | (not identified) | hot spicy chicken nibbles | Supermarket | 1C, 1P | No evidence |
| 2000 | (not identified) | Feral shellfish, feral kina, farm kill turkey | Tangi | 2C | History |
| 2000 | Enteritidis 9a | "Blowing" free range eggs, organic produce, pre-cooked chicken purchased from supermarket | Home | 3C | No evidence |
| 2000 | Typhimurium 135 | Cross contamination between chicken and apple pie | Home | 7C | History |
| 2000 | Typhimurium 135 | Chicken | Restaurant/café | 1C, 1P | History & CCP |
| 2000 | Montevideo | Chicken and lamb kebabs | Takeaway | 11C | Handler |
| 2000 | Typhimurium 135 | honey chicken, barbequed pork and rice | Restaurant/café | 11C | Handler |
| 2000 | Typhimurium | Chicken breast in stir fry | Home | 1C, 2P | History |

| Year[1] | *Salmonella* serotype | Food(s) reported | Setting | Number of cases[2] | Evidence[3] |
|---|---|---|---|---|---|
| | | chicken | | | |
| 2001 | Typhimurium | Chicken panini | Restaurant/café | 2C, 1P | History |
| 2001 | Typhimurium 160 | Butter chicken | Restaurant/café | 2C | History & CCP |
| 2001 | Typhimurium | Chicken nuggets | Restaurant/café | 1C, 1P | History |
| 2001 | Typhimurium 23 | Satay chicken | Restaurant/café | 2C | History & CCP |
| 2001 | Typhimurium 160 | Smoked chicken, luncheon, steak | Home | 2C, 1P | History |
| 2001 | Typhimurium 160 | Rotisserre chicken, hollandaise sauce with raw egg | Restaurant/café | 4C, 5P | Environ. |
| 2001 | Infantis | egg fu yong, curry beef, chicken fried rice | Takeaway | 1C, 1P | Environ. |
| 2001 | Typhimurium 160 | undercooked turkey, chicken avocado salad | Home | 1C, 4P | Environ. |
| 2002 | Typhimurium 160 | Barbecued chicken | Home | 2C, 7P | History |
| 2002 | Typhimurium 160 | Undercooked chicken | Home | 4C | Environ. |
| 2002 | Typhimurium RDNC Aug 01 | Handling of raw chicken giblets | Home | 2C | History & CCP |
| 2003 | (not identified) | Roast chicken | Home | 1C, 2P | History |
| 2003 | Typhimurium 160 | Chicken meal | Home | 3C | History & CCP |
| 2003 | Heidelberg | Shanghai style sliced chicken, braised gluten, salty pork and winter melon soup, Shanghai style rice with vegetables in soup, deep fried pork chops | Restaurant/café | 3C, 2P | Environ. |
| 2003 | Infantis | Chicken broth | Restaurant/café | 1C, 1P | History |
| 2005 | Typhimurium 160 | Shredded chicken noodle salad, chocolate cake | Unknown | 2C | History |
| 2005 | Enteritidis 9a, Heidelberg | Middle Eastern food: Chicken, hummus, flat bread, lettuce, tomato, onions, cabbage. | Takeaway | 25C | Elev. risk |
| 2005 | Typhimurium 1 | Chicken drumsticks | Takeaway | 3C, 1P | History & CCP |
| 2005 | Thompson | Chicken sandwich, bacon and egg pie, panini, fried chicken, chicken roll | Restaurant/café | 9C, 4P | Source |
| 2005 | (not identified) | Chicken satay | Home | 3C, 2P | History |
| 2005 | Typhimurium 160 | Undercooked chicken | Hangi | 3C, 5P | History |

| Year[1] | *Salmonella* serotype | Food(s) reported | Setting | Number of cases[2] | Evidence[3] |
|---|---|---|---|---|---|
| 2005 | Typhimurium 160 | Roast chicken | Home | 2C, 2P | History & CCP |
| 2005 | (not identified) | Smoked chicken, lettuce and tomato sandwich | Restaurant/café | 2C | History & CCP |
| 2007 | Typhimurium 156 | Chicken, taro, chop suey, sweet and sour mince, egg fu yong | Fundraising event | 11C, 8P | History |
| 2007 | Typhimurium 160 | BBQ chicken bacon pizza | Takeaway | 1C, 1P | History & CCP |
| 2007 | Montevideo | Chicken kebabs, lamb kebabs, vegetarian falafels | Takeaway | 10C | History & CCP |
| 2007 | Mbandaka | Chicken, eggs | Home | 34C | Elev. risk |

[1] Based on the date of onset of symptoms in the index case in the outbreak.
[2] C, confirmed cases; P, probable cases.
[3] No evidence: No evidence was reported.
History (epidemiological evidence): Cases had history of exposure to implicated source.
History & CCP: History, but investigation also found critical control point failures.
Environ: Environmental investigation identified critical control point failures linked to the implicated source.
Elev. risk (epidemiological evidence): Case control or cohort study showed elevated risk for cases exposed to implicated source.
Source (laboratory evidence): The same *Salmonella* serotype was identified in the implicated source, e.g. food, water, animal or environmental source.
Handler (laboratory evidence): The same *Salmonella* serotype was identified from clinical samples provided by one or more food handlers responsible for the implicated food(s).

### 8.2.3 Case control studies

Table 21 lists seven case control studies of salmonellosis in New Zealand.

Two case control studies have linked increased incidence of salmonellosis to contact with infected animals. The study of *S.* Typhimurium DT160 was prompted by a marked increase in the number of DT160 human isolates which began in July 2000 (Thornley *et al.*, 2003; Thornley *et al.*, 2002). The epidemic of *S.* Typhimurium DT160 infection among humans occurred in parallel with illness due to the same pathogen in wild birds, particularly sparrows. The case control study identified several exposures, but the strongest finding was the association between *S.* Typhimurium DT160 infection and direct handling of dead wild birds. However, this high risk activity was associated with only a few cases and the authors acknowledged that the case control study did not investigate exposure to environments contaminated by wild bird faeces, such as parks and play areas.

In addition to the *S.* Typhimurium DT160 case control study, environmental sampling was carried out on roof-collected drinking water supplies from the homes of cases, and egg brands consumed by cases. *S.* Typhimurium DT160 was isolated from four drinking water sources that were used by five cases. The authors suggested that consumption of water that had not been disinfected was not confirmed in the case control study because cases and controls were matched by neighbourhood, which usually have similar water sources. Six different brands of eggs were identified by four patients who had eaten them raw. *S.* Thompson was isolated

from the shell surface in samples of two of these brands, but *S.* Typhimurium DT160 was not isolated.

The strongest finding was that there was an association between infection with S. Typhimurium and direct contact with wild birds (mOR = 12.3, CI: 2.8-54.6). This high risk activity was however associated with only a few cases. Consumption of takeaway food had a weakly positive association with infection (mOR = 1.7, CI: 1.04-2.8), but consumption of whole chicken was less common amongst cases than controls (mOR = 0.4, CII: 0.2-0.6). Contact with another individual with diarrhoea and vomiting was also associated with *S.* Typhimurium DT160 infection (mOR = 3.1, CI: 1.7-5.7). Population attributable ratios (PAR) were calculated and the largest PAR% was demonstrated for consumption of takeaway food (26.1%). However, no single type of takeaway outlet was significantly associated with illness.

The second case control study was conducted by ESR in late January 2002 as a component of the NZFSA quantitative risk assessment of *Salmonella* in New Zealand sheep meat (NZFSA, 2002). The aim of the study was to quantify the incidence of human infection with *Salmonella* species, in particular *S.* Brandenburg, and to estimate the contribution of New Zealand sheep meat consumption to this incidence. Table 21 only shows the results of the case control study that specifically addresses *S.* Brandenburg infection (Baker *et al.*, 2007). The case control study also investigated general salmonellosis, and the full number of salmonellosis cases recruited was 182, including the 43 cases of *S.* Brandenburg infection. There were also 182 matched controls (Baker *et al.*, 2003). Factors occurring in the three days prior to illness (or interview) that were significantly associated with an elevated risk of salmonellosis in general were (95% confidence intervals are in parentheses):

- Contact with bird faeces, OR=4.87 (1.71-17.17);
- Contact with other sick people, OR=8.73 (2.08-62.91);
- Consumption of pork steak, OR=5.60 (1.11-72.80);
- Overseas travel, OR=9.97 (1.72-167.46);
- Touching of pet puppies, OR=6.79 (1.33-73.03); and,
- Use of a kitchen bench, table, or sink for chopping, OR=5.47 (1.47-31.42).

All other foods included in the questionnaire (whole chicken, imported food, uncooked vegetables, unpeeled fruit, pies, bacon, small goods, eggs, dairy products) were protective (OR < 1). *S.* Brandenburg infection was associated with contact with live or dead sheep or lambs, or contact with a household member who had occupational contact with sheep or lambs (Baker *et al.*, 2007). Overall the study indicated that infection with *S.* Brandenburg had not become a foodborne disease, and instead was an important zoonotic disease representing a risk to farmers and others with direct occupational contact with infected sheep.

In the remaining five case control studies, salmonellosis infection by the serotype of interest was associated with consumption of raw carrots, food from a premises serving Middle Eastern dishes, chicken or eggs or lettuce, watermelon, or flour.

One suspected cause of the 2005 outbreak of *S.* Saintpaul infection was the washing of raw carrots with untreated stream water (Neuwelt *et al.*, 2006). Samples of the stream water contained a high coliform count (460 to 2400 per 100 ml) and *E. coli* (9.8 to 88 per 100 ml), but *Salmonella* was not isolated.

The results of the case control study for the 2005 outbreak of *S.* Enteritidis 9a infection associated consumption of food purchased for a premises serving Middle Eastern dishes with illness (Anonymous, 2005). However, no single food item was identified as being associated with infection; consumption of chicken, hummus, flat bread, lettuce, tomato, onions and cabbage were all significant. When logistic regression was used to control for confounding between food items, no food item was identified as a significant independent risk factor. *S.* Enteritidis 9a was not isolated from any food samples taken from the implicated premises, although *S.* Orion was isolated from tahini. However, consumption of chicken remained significantly associated with illness after excluding people who had consumed food from the implicated premises.

The case control study for the 2008 outbreak of *S.* Mbandaka infection did not conclusively identify a causative food (McCallum and Das, 2008). There were increased risks of infection associated with purchasing chicken breast from a supermarket that was supplied by a specific poultry processor, and eating eggs prepared away from home. The results suggested that there was also an association between lettuces and chicken purchased from the supermarket, however the authors reasoned that this outcome may be due to consumers being more likely to purchase both items. An environmental investigation tested food samples from cases homes and implicated food premises, plus swabs from bench tops, chopping boards, fridges and hand wash basins. *Salmonella* was not isolated from any food or environmental samples. During the outbreak, *S.* Mbandaka was isolated from samples taken as part of routine monitoring of poultry feed, poultry products and the poultry processing environment. *S.* Mbandaka had been isolated from these types of samples in previous years, but at lower incidence.

The 2009 outbreak of *S.* Typhimurium DT1 infection was associated with consumption of watermelon purchased from a roadside stall from a grower in Gisborne (McCallum *et al.*, 2009). An environmental investigation revealed unhygienic conditions in the watermelon packhouse and a septic tank located near the watermelon growing area. *Salmonella* was not isolated from watermelon samples. Cases also had increased odds of exposure to ham, in particular ham purchased from a specific supermarket, but this association was not significant.

An outbreak of *S.* Typhimurium DT42 infection in 2008-09 was caused by contaminated flour (Lisa McCallum, ESR, personal communication). Twelve cases were hospitalised (no fatalities) and the majority of the cases resided in Canterbury (22/75) and Otago (17/75). An elevated significant OR was also found for a specific supermarket and brand of flour. Flour samples were collected and tested for *Salmonella* from open packets in the homes of cases (4/26 positive), unopened packets that had been on sale in retail outlets prior to withdrawal (2/41 positive) and retrieved/withdrawn flour (3/23 batches of flour positive). Contamination levels were estimated for 3 of the positive samples. *Salmonella* counts ranged from 1 per 300g to 1 per 50g.

The same outbreak strain had been previously isolated from poultry feed produced by an animal feed mill from a by-product of flour milling called “broll”. Broll is the husk of the wheat kernel removed during milling of flour. The broll had been produced by the same flour mill that produced the contaminated flour, during the same time period. Environmental

swabs were taken at the implicated flour mill as part of the flour outbreak investigation, but the outbreak strain was not isolated.

The flour company that produced the flour has two mills located in North and South Islands. Only the flour from the South Island mill was found to be contaminated which is consistent with the majority of cases being from the South Island. The South Island flour mill receives wheat from more than 400 New Zealand growers as well as imported wheat. Testing of withdrawn flour was undertaken to narrow down the search for a particular wheat source. Although further positive batches of flour were identified, the source of the contaminated wheat could not be established.

## Table 21: Case control studies of salmonellosis in New Zealand

| Year | *Salmonella* serotype | No. cases[1] | No. cases and controls[2] | Exposures associated with increased disease risk OR/mOR (95% confidence interval)[3] | Reference |
|---|---|---|---|---|---|
| 2001 | Typhimurium DT160 | 45 | 119 cases<br>235 controls | • Direct handling of dead wild birds, mOR=12.28 (2.76-54.63)<br>• Exposure to person with diarrhoea and vomiting (D&V) in household in 3 days before illness, mOR=4.67 (1.21-18.05)<br>• Exposure to person with D&V in any setting in 3 days before illness, mOR=3.81 (1.53-9.49)<br>• Exposure to person with D&V in household in 28 days before illness, mOR=3.11 (1.13-8.54)<br>• Exposure to person with D&V in any setting in 28 days before illness, mOR=3.05 (1.64-5.69)<br>• Consumption of food at a large gathering, mOR=2.44 (1.27-4.68)<br>• Consumption of any fast food, mOR=1.69 (1.04-2.75)<br>After step-wise regression:<br>• Direct handling of dead wild birds, aOR=10.5 (2.3-47.5)<br>• Exposure to person with D&V 28 days before illness, aOR=2.8 (1.4-5.4)<br>• Consumption of any fast food, aOR=1.7 (1.0-2.9) | (Sneyd *et al.*, 2002; Thornley *et al.*, 2003) |
| 2002-2003 | Brandenburg | 85 | 43 cases<br>43 controls | After multivariate analysis:<br>• Occupational contact with live or dead sheep or lambs during the 3 days prior to illness, OR=9.79 (1.69-190.38)<br>• Having a household member who had occupational contact with sheep or lambs in the 3 days prior to illness or interview, OR=4.31 (1.26-21.33) | (Baker *et al.*, 2007) |
| 2005 | Saintpaul | 19 | 19 cases<br>57 controls | • Eaten raw carrots during the 3 day period prior to illness or interview, OR=4.0 (1.35-12.01); mOR=7.3 (1.8-30.6)<br>After controlling for age and matching telephone number, aOR=2.86 (0.66-12.3), i.e. not significant. | (Neuwelt *et al.*, 2006) |
| 2005 | Enteritidis 9a | 24 | 24 cases<br>72 controls | • Eaten food from a Middle Eastern restaurant prior to illness, OR=10.2 (2.4-49.9) | (Anonymous, 2005) |
| 2008 | Mbandaka | 34 | 21 cases<br>63 controls | • Chicken breast prepared at home from a specific processor, OR= 10.71 (1.50-118.52)<br>• Eat chicken prepared away from home, OR=5.41 (1.67-18.38)<br>• Eat eggs prepared away from home, OR=4.58 (1.10-19.07)<br>• Eat eggs prepared away from home prepared using other method (not scrambled, omelette, fried, boiled, poached), OR=14.75 (1.28-728.09)<br>After multivariate logistic regression of all exposures where p-value was ≤0.05:<br>• Chicken breast prepared at home, purchased from premise supplied by a specific processor, | (McCallum and Das, 2008) |

| Year | *Salmonella* serotype | No. cases[1] | No. cases and controls[2] | Exposures associated with increased disease risk OR/mOR (95% confidence interval)[3] | Reference |
|---|---|---|---|---|---|
| | | | | OR=9.24 (1.23-69.48)<br>• Eggs prepared away from home, OR=7.41 (1.67-32.99)<br>• Iceberg lettuce purchased from a specific supermarket, OR=6.25 (1.33-29.38)<br>After multivariate logistic regression of all exposures where p-value was ≤0.05, excluding 'chicken breast prepared at home, purchased from premise supplied by a specific processor':<br>• Chicken prepared away from home, OR=5.83 (1.83-18.52)<br>• Eggs prepared away from home, OR=6.11 (1.43-26.06) | |
| 2009 | Typhimurium DT1 | 19 | 15 cases<br>40 controls | • Watermelon eaten at home, OR=5.17 (1.22-22.42)<br>• Watermelon purchased from roadside stall, OR=9.5 (1.08-114.8)<br>• Ate any water melon (at home or away from home), OR=6.00 (1.4-27.28)<br>After controlling for age group and sex:<br>• Watermelon eaten at home, aOR=6.78 (1.26-36.59)<br>• Ate any water melon (at home or away from home), aOR=7.33 (1.36-39.61)<br>After excluding cases that had contact with symptomatic people and controlling for age group and sex:<br>• Watermelon eaten at home, aOR=9.87 (1.46-66.79)<br>• Ate any water melon (at home or away from home), aOR=6.22 (1.12-34.68) | (McCallum *et al.*, 2009) |
| 2008-2009 | Typhimurium DT42 | 75 | 33 cases<br>66 controls | • Eating, licking or tasting uncooked baking mixture, OR=3.6 (1.2-10.7)<br>After adjusting for eggs in individual baking ingredients:<br>• Flour, aOR=5.7 (1.1-29.1)<br>After adjusting for flour in individual baking ingredients:<br>• Eggs, OR=0.8 (0.2-3.4), i.e. not significant | Lisa McCallum, ESR, personal communication |

[1] Number of cases initially identified in the outbreak or cluster.
[2] Number of cases and controls included in the case control study.
[3] OR, odds ratio mOR, matched odds ratio aOR, adjusted odds ratio: Controlling for factors such as age, sex or other exposures.

### 8.2.4 Serotypes causing disease in New Zealand

There were 11,554 New Zealand cases of salmonellosis reported for the period 2000 to 2009 for which the *Salmonella* serotype was available (Adlam *et al.*, 2010). *S.* Typhimurium was the reported cause of 58.2% of these cases and the next most frequently reported serotype was *S.* Enteritidis (8.8% of cases). When considering serotype and phage type, *S.* Typhimurium DT160 was most frequently reported (19% of cases). Table 22 displays the peak years and total number of cases for serotypes that have caused 50 or more salmonellosis cases between 2000 and 2009. Together these 35 serotypes caused 80% (9,290) of the 11,554 cases.

**Table 22: *Salmonella* serotypes that caused 50 or more cases over the years 2000 to 2009 – peak occurrence and total cases (Adlam *et al.*, 2010)**

| ***Salmonella* serotype** | **Peak occurrence[1]** | | | | | | | | | | **Total cases[2]** |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | **2000** | **2001** | **2002** | **2003** | **2004** | **2005** | **2006** | **2007** | **2008** | **2009** | |
| Typhimurium DT160 | | + | | | | | | | | | **2,147** |
| Typhimurium DT1 | + | + | + | | | | | | | | **729** |
| Brandenburg | + | + | | | | | | | | | **700** |
| Typhimurium DT135 | + | + | | | | | | | | | **698** |
| Typhimurium DT156 | + | + | | | | | | | | | **562** |
| Infantis | | | | | | | | | + | + | **523** |
| Typhimurium DT101 | + | | | | | | | | | | **505** |
| Enteritidis PT9a | + | + | | | | | | | | | **432** |
| Typhimurium DT42 | + | | | | | | | | | | **257** |
| Saintpaul | | | | | | + | | | | | **249** |
| Typhimurium DT12a | | | | | | | + | | | | **237** |
| Typhimurium DT9 | + | | | | | | | | | | **182** |
| Typhimurium RDNC-May 06[3] | | | | | | | | | + | | **154** |
| Heidelberg | | + | | | | | | | | | **150** |
| Virchow | | | | | + | | | + | | | **141** |
| Typhimurium DT74 | | | | | | | + | | | | **139** |
| Typhimurium DT23 | | + | | | | | | | | | **138** |
| Typhimurium RDNC[3] | | | | + | | | | | | + | **137** |
| Mississippi | | | | | | + | | | | | **95** |
| Enteritidis PT4 | + | | + | | | | | | | | **95** |
| Thompson | | | | | | | + | | | | **92** |
| Agona | | | | | | | + | | | | **92** |
| Weltevreden | | + | | | | | + | | | | **88** |
| Montevideo | | | + | + | | | | | | | **79** |
| Mbandaka | | | | | | | + | | + | | **76** |
| Newport | | + | | | | | + | | | | **68** |
| Stanley | | | | | | | | | + | | **65** |
| Enteritidis PT6a | | | | | | | | | + | | **62** |

| *Salmonella* serotype | Peak occurrence[1] | | | | | | | | | | Total cases[2] |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | 2000 | 2001 | 2002 | 2003 | 2004 | 2005 | 2006 | 2007 | 2008 | 2009 | |
| Corvallis | | | | | | | + | | | | **61** |
| *Salmonella* sp. 4,5,12:d :- | | | | | + | | | + | | | **59** |
| Typhimurium DT8 | | | | | | | | + | | | **58** |
| Enteritidis PT1 | | + | | | | | | | | | **58** |
| Enteritidis PT1b | | | | | | | | | + | | **57** |
| Hadar | + | + | + | | | | | | | | **55** |
| Typhimurium RDNC Aug-01[3] | | | | + | | | | | | | **50** |

[1] + denotes where number of cases exceeds ten year mean plus one standard deviation for a given serotype.
[2] There were 232 cases caused by S. Typhimurium that did not have phage typing data available. These cases are excluded from this table.
[3] Typhimurium RDNC is not a single serotype, but a grouping of serotypes. RDNC stands for 'reaction does not conform' and indicates that the isolate does not match any recognised serotypes. RDNC can sometimes be followed by the month and year of isolation.

Of the 11,554 salmonellosis cases between 2000 and 2009 for which the *Salmonella* serotype is known, 77.3% lived in highly urban areas and 10.2% lived in highly rural areas (Adlam *et al.*, 2010). The serotypes significantly associated with cases living in highly urban areas were *S.* Infantis ($p<0.001$) and *S.* Typhimurium DT160 ($p<0.05$). The serotypes significantly associated with cases living in highly rural areas were *S.* Saintpaul ($p<0.001$), *S.* Brandenburg ($p<0.01$) and *S.* Typhimurium DT101 ($p<0.05$).

### 8.2.5 Antimicrobial resistance of New Zealand *Salmonella* strains

ESR tests the antimicrobial resistance of approximately 20% of all human and non-human *Salmonella* isolates received for typing, along with all *S.* Typhimurium phage types that are internationally recognised as being multiresistant.[34]

Resistance to each of the 12 antimicrobials tested and multiresistance to three or more of these is shown in Table 24 for human isolates, and Table 24 for non-human isolates (isolates from animal or environmental samples), for the years 2005 to 2009

**Table 23: Antimicrobial resistance of a sample of New Zealand *Salmonella* isolates from humans, 2005-2009[1]**

| Antimicrobial | Percent of isolates resistant each year (n=number tested) | | | | |
|---|---|---|---|---|---|
| | 2005 (n=318) | 2006 (n=276) | 2007 (n=267) | 2008 (n=277) | 2009 (n=235) |
| Ampicillin | 4.1 | 4.4 | 6.7 | 5.1 | 5.5 |
| Cephalothin | 0.6 | 0.4 | 0.8 | 0.4 | 1.7 |
| Chloramphenicol | 1.9 | 2.9 | 1.5 | 0.7 | 3.0 |
| Ciprofloxacin | 0.3 | 0 | 0 | 0 | 0.9 |
| Co-amoxiclav | 0.3 | 0 | 0 | 0.4 | 1.7 |
| Co-trimoxazole | 1.9 | 2.2 | 1.5 | 1.4 | 2.1 |
| Gentamicin | 0.6 | 0 | 0 | 0.4 | 1.7 |
| Nalidixic acid | 5.7 | 4.7 | 5.2 | 6.1 | 3.8 |

[34] Data are available from the annual reports of antimicrobial susceptibility among *Salmonella*, produced by ESR and available at: http://www.surv.esr.cri.nz/antimicrobial/salmonella.php (accessed 1 December 2010).

| Antimicrobial | Percent of isolates resistant each year (n=number tested) | | | | |
|---|---|---|---|---|---|
| | 2005 (n=318) | 2006 (n=276) | 2007 (n=267) | 2008 (n=277) | 2009 (n=235) |
| Streptomycin | 3.1 | 4.7 | 8.6 | 5.1 | 5.1 |
| Sulphonamides | 4.1 | 5.1 | 6.4 | 5.8 | 6.0 |
| Tetracycline | 5.0 | 5.8 | 9.0 | 6.9 | 4.7 |
| Trimethoprim | 1.9 | 2.2 | 1.5 | 1.8 | 2.1 |
| Multiresistant to ≥3 antimicrobials[2] | 4.1 | 4.7 | 6.4 | 5.8 | 5.5 |

[1] Data are from the annual reports of antimicrobial susceptibility among *Salmonella*, produced by ESR and available at: http://www.surv.esr.cri.nz/antimicrobial/salmonella.php (accessed 1 December 2010).
[2] For all years, co-trimoxazole and trimethoprim resistance were counted as one resistance for the estimates of multiresistance. In 2009, ciprofloxacin and nalidixic acid resistance was counted as one resistance.

**Table 24: Antimicrobial resistance of a sample of New Zealand *Salmonella* isolates from animal and environmental samples, 2005-2009[1]**

| Antimicrobial | Percent of isolates resistant each year (n=number tested) | | | | |
|---|---|---|---|---|---|
| | 2005 (n=298) | 2006 (n=298) | 2007 (n=206) | 2008 (n=277) | 2009 (n=180) |
| Ampicillin | 0.3 | 0.3 | 1.9 | 0.7 | 0 |
| Cephalothin | 0 | 0 | 0.5 | 0.7 | 0 |
| Chloramphenicol | 0 | 0 | 0 | 0.4 | 0 |
| Ciprofloxacin | 0 | 0 | 0 | 0 | 0 |
| Co-amoxiclav | 0 | 0 | 0.5 | 0.7 | 0 |
| Co-trimoxazole | 0.3 | 0.3 | 1.0 | 0 | 0 |
| Gentamicin | 0.3 | 0 | 0 | 0 | 0 |
| Nalidixic acid | 0 | 0 | 0.5 | 0 | 0 |
| Streptomycin | 3.0 | 1.7 | 6.3 | 5.4 | 5.0 |
| Sulphonamides | 1.7 | 1.7 | 7.8 | 2.5 | 4.4 |
| Tetracycline | 2.7 | 2.4 | 3.9 | 1.8 | 3.3 |
| Trimethoprim | 0.3 | 0.3 | 1.0 | 0 | 0 |
| Multiresistant to ≥3 antimicrobials[1] | 1.3 | 1.3 | 4.4 | 2.2 | 2.8 |

[1] For all years, co-trimoxazole and trimethoprim resistance were counted as one resistance for the estimates of multiresistance. In 2009, ciprofloxacin and nalidixic acid resistance was counted as one resistance.

## 8.3 Adverse health effects in other countries

The global burden of non-typhoid salmonellosis (circa 2006) was recently estimated at 93.8 million cases, with 155,000 deaths (Majowicz *et al.*, 2010). An estimated 80.3 million of these cases were from foodborne infection. The incidence was estimated as 1,140 per 100,000 person-years.

Table 25 shows the reported incidence of salmonellosis in several countries.

**Table 25: Reported incidence data for notified cases of salmonellosis in other countries***

| Country | Incidence (cases/100,000) | Year | Data source |
|---|---|---|---|
| Australia | 43.6 | 2009 | 1 |
| Canada | 18.0 | 2006 | 2 |
| EU (27 member states) | 23.7 | 2009 | 3 |
| USA | 15.2 | 2009 | 4 |
| USA | 16.9 | 2008 | 5 |
| Fiji | 5.1 | 2004-05 | 6 |

* Does not include *S.* Typhi or *S.* Paratyphi

Data sources:

1. (National Notifiable Diseases Surveillance System, 2011)
2. (Public Health Agency of Canada, 2007)
3. (European Food Safety Authority and European Centre for Disease Prevention and Control, 2011). Range 2.1 in Portugal to 100.1 in the Czech Republic, Germany and the United Kingdom.
4. (Matyas *et al.*, 2010). Data is based on ten USA states.
5. (Hall-Baker *et al.*, 2010). Data is from health departments in the 50 states, five territories, New York City, and the District of Columbia.
6. (Dunn *et al.*, 2005)

Estimates of foodborne diseases acquired in the USA have recently been reported for 31 major pathogens, including *Salmonella* species (Scallan *et al.*, 2011). The authors used data from a number of active and passive surveillance systems for the period 2000-2008, and based all estimates on the 2006 population. An estimated 9.4 million (90% credible interval 6.6-12.7 million) illnesses per year were domestically-acquired foodborne infections. Non-typhoidal *Salmonella* was the causative pathogen of an estimated 1 million (0.6-1.7 million), or 11%, of these infections, second only to norovirus (5.5 million, 58%). Non-typhoidal salmonellosis was estimated to be the leading cause of hospitalisation due to domestically-acquired foodborne infection (35% of hospitalised cases) and deaths (28%).

Surveillance data from 1996-2000 have also been used to estimate the impact of foodborne disease in England and Wales (Adak *et al.*, 2005). Non-typhoidal salmonellosis was the estimated cause of 73,193/1,724,315 (4.2%) cases of domestically-acquired foodborne disease per annum, only exceeded by campylobacteriosis (19.6%), *Clostridium perfringens* infection (9.8%) and yersiniosis (7.5%). Salmonellosis was also estimated to be the leading cause of death (30%), and second only to campylobacteriosis in causing hospitalisation (12%). Poultry was also reported to cause an estimated 0.5 million (29%) cases of domestically-acquired foodborne disease, and a case fatality rate of 38 per 100,000 cases.

In Australia, an estimated 81,000 (95% credibility interval (CrI) 23,000-138,000) cases of gastroenteritis per annum were caused by foodborne *Salmonella* infection (based on a typical year circa 2000; incidence 422.9 per 100,000 people[35]) (Hall *et al.*, 2005). These cases represented 5.5% of the total estimated cases of foodborne gastroenteritis caused by 16 known pathogens. An estimated 14,700 cases were hospitalised with foodborne

[35] Calculated from a population of 19,153,400 as reported for June 2000, by the Australian Bureau of Statistics (http://www.abs.gov.au/AUSSTATS/abs@.nsf/DetailsPage/3101.0Jun%202010?OpenDocument, see Table 1; accessed 8 February 2011)

gastroenteritis per year (including an estimated 11,000 cases infected with an unidentified pathogen), and of these, an estimated 1,060 (900-1,240; 7.2%) were caused by non-typhoidal *Salmonella* infection. In another study based on data from 2000-2004, the annual community incidence of salmonellosis in Australia was estimated as 49,843 (95% CrI 28,466-118,518) cases, and the salmonellosis rate as 262 (95% credible interval 150-624) per 100,000 people (Hall *et al.*, 2008).

### 8.3.1 *Salmonella* serotypes causing disease in other countries

The ten most frequently reported serotypes isolated from humans in 2008 have been reported for 26 EU member states (EFSA, 2010a) (Table 26). *S.* Enteritidis and *S.* Typhimurium were the serovars most frequently associated with human illness, and this trend continued in 2009 (European Food Safety Authority and European Centre for Disease Prevention and Control, 2011). *S.* Enteritidis cases were most commonly associated with the consumption of contaminated eggs and poultry meat, while *S.* Typhimurium cases were mostly associated with the consumption of contaminated pig, poultry and bovine meat. The proportion of *S.* Enteritidis and *S.* Typhimurium cases with phage type data was very low (12.2% and 20.2%, respectively). The available data showed the most commonly identified *S.* Enteritidis phage types to be PT4 and PT8. *S.* Typhimurium U292 was the most commonly identified *S.* Typhimurium phage type (all were from Denmark). There are reports that salmonellosis caused by *S.* Enteritidis infection are declining as a result of vaccination programmes to immunise layers against *S.* Enteritidis (e.g. (Cogan and Humphrey, 2003; Collard *et al.*, 2008; Kornschober *et al.*, 2009)).

**Table 26: Ten most commonly confirmed human salmonellosis serotypes in the EU, 2008**

| ***Salmonella* serotype** | **N** | **%** |
|---|---|---|
| Enteritidis | 70,091 | 58.0 |
| Typhimurium | 26,423 | 21.9 |
| Infantis | 1,317 | 1.1 |
| Virchow | 860 | 0.7 |
| Newport | 787 | 0.7 |
| Agona | 636 | 0.5 |
| Derby | 624 | 0.5 |
| Stanley | 529 | 0.4 |
| Bovismorbificans | 501 | 0.4 |
| Kentucky | 497 | 0.4 |
| Other | 18,495 | 15.3 |
| **Total** | **120,760** | - |

Source: (EFSA, 2010a); data submitted from 26 EU member states.

In Australia during 2009, the most commonly notified *Salmonella* serotype was *S.* Typhimurium, which was responsible for approximately 41% of all notified infections (serotype information was available for 6,983/7,464 (94%) *Salmonella* notifications) (OzFoodNet Working Group, 2010). Commonly isolated phage types during this year were *S.* Typhimurium DT170/DT108 and *S.* Typhimurium DT135/DT135a. *S.* Enteritidis is not endemic in Australian egg layer flocks and during 2009, 508 of the 587 (87%) cases of *S.* Enteritidis infection had reported overseas travel (travel histories were not available for 40 of the 587 cases).

Preliminary data for 2009 released by the Foodborne Diseases Active Surveillance Network (FoodNet) (based on data from ten USA states) showed that among 6,371 *Salmonella* isolates serotyped, 10 serotypes accounted for 73.1% of infections (Matyas *et al.*, 2010). *S.* Enteritidis was most often identified (1,226, 19.2%, followed by *S.* Typhimurium (1,024, 16.1%), *S.* Newport (772, 12.1%) and *S.* Javiana (544, 8.5%).

The most frequently isolated serotypes in Canada during 2006 were *S.* Enteritidis (1,344/5,870 notifications where serotyping information was available, or 23%), *S.* Typhimurium (1,005, 17%) and *S.* Heidelberg (707, 12%) (Public Health Agency of Canada, 2007).

### 8.3.2 Salmonellosis outbreaks in other countries

Salmonellosis is a significant contributor to infectious intestinal disease outbreaks in many countries as shown by the data summarised in Table 27.

**Table 27: Foodborne outbreaks in other countries: Proportion attributed to *Salmonella* infection**

| Country | Year(s) | Foodborne outbreaks attributed to *Salmonella* infection | Data source |
|---|---|---|---|
| Australia | 2009 | 59/163 (36%) | (OzFoodNet Working Group, 2010) |
| England and Wales | 1992-2008 | 1,135/2,429 (47%) | (Gormley *et al.*, 2010) |
| European Union | 2009 | 324/977 (33%) Verified (55% in 2008)<br>1,722/5,550 (31%) All | (EFSA, 2010a; European Food Safety Authority and European Centre for Disease Prevention and Control, 2011) |
| Japan | 1981-95 | 17.2% of cases of known cause, 23.8% of outbreak cases (16.2% were of unknown cause) | (Lee *et al.*, 2001) |
| Korea | 1981-95 | 28.3% of outbreaks of known cause, 31.2% of outbreak cases (26.6% were of unknown cause) | (Lee *et al.*, 2001) |
| Netherlands | 1991-94 | 15.5% of outbreaks of known cause (90.4% were of unknown cause) | (Simone *et al.*, 1997) |
| Sweden | 1992-97 | 17.8% of outbreaks of known cause, 14.5% of outbreak cases (61% of outbreaks were of unknown cause) | (Lindqvist *et al.*, 2000) |
| USA | 2007 | 142/1097 (12.9) | (Boore *et al.*, 2010) |

Table 28 gives some examples of salmonellosis outbreaks associated with poultry meat or foods containing poultry that have been reported in the literature. Contact with live poultry has also caused salmonellosis outbreaks (e.g. (Bidol *et al.*, 2007; Hedican *et al.*, 2009)), as has consumption of foods contaminated with raw poultry (e.g. (Kistemann *et al.*, 2000; Moffatt *et al.*, 2006)) but these have not been included here.

**Table 28: Examples of outbreaks of salmonellosis from consumption of poultry or foods containing poultry in other countries**

| Country | Year(s) | *Salmonella* serotype | No. cases | Suspected food(s) | Probable cause | Reference |
|---|---|---|---|---|---|---|
| **UK and Europe** | | | | | | |
| England | 1995-6 | Montevideo | 4 | Roast chicken purchased hot from a local supermarket (confirmed for 1 case) | Cross-contamination from supermarket knife and cutting board | (Threlfall *et al.*, 1999) |
| England | 1996 | Agona PT15 | 9 | Precooked turkey meat (confirmed) | Undercooking | (Synnott *et al.*, 1998) |
| Ireland | 1996 | Typhimurium DT104 (multiresistant) | 58 | Turkey | Temperature abuse, cross-contamination | (Grein *et al.*, 1999) |
| Northern Ireland | 1997 | Bredeney | 10 | Cooked chicken prepared by butcher and retailed through bakeries (confirmed) | Undercooking | (Moore *et al.*, 2003) |
| Spain | 2005 | Hadar | >2,000 | One widely distributed brand of precooked, vacuum-packed roast chicken (confirmed) | Contaminated product | (Lenglet, 2005) |
| Scotland | 2000 | Enteritidis PT5c and PT6a | 70 | Chicken dishes from Chinese takeaway | Not identified | (Cowden *et al.*, 2003) |
| Estonia | 2008 | Enteritidis | 94 | Chicken soup | Not identified | (Dontšenko *et al.*, 2008) |
| Hungary | 2007 | Enteritidis PT8 | 31 | Chicken dishes in a buffet | Undercooking, temperature abuse | (Krisztalovics *et al.*, 2007) |
| **North and South America** | | | | | | |
| USA | 1990 | N/R | 824 | Turkey | Temperature abuse | (Luby *et al.*, 1993) |
| USA | 2007 | I 4,5,12:i:- | 401 | Frozen, not ready-to-eat pot pies (uneaten pies containing turkey tested positive) | Undercooking: Consumer confusion over microwave instructions | (Meyer *et al.*, 2008) |
| Brazil | 2005 | Enteritidis | 5 | Chicken (confirmed; $1.1x10^5$ MPN/g *Salmonella*) | N/R | (Mürmann *et al.*, 2008) |
| Brazil | 2005 | Enteritidis | 9 | Chicken, potatoes with mayonnaise, sausage (confirmed, all $10^6$-$10^7$ MPN/g *Salmonella*) | N/R | (Mürmann *et al.*, 2008) |

| Country | Year(s) | *Salmonella* serotype | No. cases | Suspected food(s) | Probable cause | Reference |
|---|---|---|---|---|---|---|
| USA | 2008 | Montevideo | >60 | Chicken, cilantro | Undercooking, cross-contamination | (Patel *et al.*, 2010) |
| USA | 1998<br>2005<br>2005-06<br>2006 | Typhimurium<br>Heidelberg<br>Enteritidis<br>Typhimurium | 33<br>4<br>27<br>3 | Raw, frozen, stuffed, breaded, prebrowned chicken products (confirmed) | Undercooking | (Smith *et al.*, 2008) |
| **Australasia/Pacific** | | | | | | |
| Australia | 1998 | Typhimurium PT12 | 10 | Chicken nuggets (confirmed) | Flash fried only, but assumed cooked by consumers | (Kenny *et al.*, 1999) |
| Australia | 1998 | Typhimurium RDNC A045 | 38 | Spatchcock[2], scampi | Temperature abuse, cross-contamination | (Brennan *et al.*, 1999) |
| **Asia** | | | | | | |
| Japan[1] | N/R | Enteritidis | 53 | Chicken and eggs on rice | N/R | (Kasuga *et al.*, 2004) |

N/R – not reported

[1] 133 people were exposed to the food at a day care centre, of which 3 adults and 50 children became ill (attack rate 18.75% for adults and 42.74% for children). The food contained 27 CFU/g *Salmonella*, and based on 150 g of food being consumed the dose was estimated as 4,050 CFU/person.

[2] A spatchcock is a particular method of poultry preparation. These spatchcocks were broiler chickens.

### 8.3.3 Case control studies in other countries

Case control studies that have implicated poultry as a probable cause of *Salmonella* infection in a number of countries are summarised in Table 29.

In the case control study of *S.* Typhimurium DT104 in England and Wales, in addition to the risk factors associated with chicken from local takeaways, restaurants and butchers, it was found that chicken eaten at home was associated with a reduced risk of infection. This was attributed to either better cooking practices, or that the chickens purchased from supermarkets for home consumption came from plants with good microbiological standards (Wall *et al.*, 1994).

The finding in Norway that consumption of poultry overall was not a risk factor, but consumption of poultry purchased from outside Norway was, is of interest. The Norwegian food chain has historically had a low level of *Salmonella* in comparison to other countries and this has been reinforced by a programme of surveillance and control initiated in 1995 (Kapperud *et al.*, 1998).

### 8.3.4 Secondary transmission

Secondary transmission of *Salmonella* in outbreaks is a recognised phenomenon. Carriage in faeces in convalescent excreters can be quite substantial, numbers approximating $10^6$-$10^7$/g persisting up to 10 days after initial diagnosis. Reduction in numbers with time is variable although the authors suggest that most people will have counts of less than 100 salmonellae/g after 35 to 40 days. (see section 2.2) but a count of 6 x $10^3$/g has been recorded in one patient 48 days post illness (Pether and Scott, 1982).

## Table 29: Case control studies in other countries which identified consumption of poultry as a risk factor

| Country | Year | *Salmonella* serotype | No. cases[1] | No. cases and controls[2] | Exposures associated with increased disease risk OR/mOR (95% confidence interval, or *p*-value if OR not provided)[3] | Reference |
|---|---|---|---|---|---|---|
| England and Wales | 1994 | Virchow PT26 | N/A | 88 cases<br>182 controls | Consumption of any chicken, OR=2.5 (1.1-5.8)<br>Consumption of chicken curry, OR=2.9 (1.4-6.1)<br>Consumption of other pre-prepared chicken, OR=3.8 (1.9-7.6)<br>Also possibly consumption of Halal chicken (p=0.015) | (Willocks *et al.*, 1996) |
| England and Wales | 1993 | Multi-resistant Typhimurium DT104 | N/A | 83 cases<br>235 controls | Pork sausages from restaurant/takeaways, OR=>1000 (1.9-indeterminate)<br>Chicken from restaurant/takeaway, OR=3.1 (1.3-7.6)<br>Chicken from local butcher, OR=6.3 (2.0-19.9)<br>"Brand Y" meat paste, OR=11.2 (1.2-10.5) | (Wall *et al.*, 1994) |
| Norway | 1993-4 | All | N/A | 94 cases<br>226 controls | Consumption of poultry purchased abroad, OR=7.6 (2.1-27.0) | (Kapperud *et al.*, 1998) |
| England | 1988 | Enteritidis PT4 | N/A | 160 cases<br>196 controls | Consumption of raw eggs (p=0.02)<br>Consumption of brought sandwiches containing mayonnaise (p=0.00004)<br>Consumption of bought sandwiches containing eggs (p=0.02)<br>Consumption of lightly cooked eggs (p=0.02)<br>Consumption of ready prepared hot chicken (p=0.006) | (Cowden *et al.*, 1989) |
| Australia | 1995 | Bredeney | 157 | 30 cases<br>60 controls | After removal of probable secondary cases:<br>Consumption of chicken, OR=6.0<br>Consumption of ground pepper, OR=3.75<br>Consumption of cold meat(s), OR=2.8 | (Baker *et al.*, 1998) |
| Australia | 2005 | Typhimurium DT135a | N/A | 61 cases<br>173 controls | Consumption of chicken from a fast food outlet, aOR=2.8 (1.0-7.7)<br>Consumption of chicken purchased from "supermarket A", aOR=3.2 (1.2-9.0)<br>(samples from supermarket A tested positive) | (McPherson *et al.*, 2006) |
| Canada | 2003 | Heidelberg | N/A | 95 matched pairs plus 16 unmatched cases | Chicken nuggets, OR=3.2 (1.5-6.8)<br>Chicken nuggets prepared at home, OR=3.5 (1.6-7.7)<br>Chicken strips, OR=6.8 (1.9-38.1)<br>Chicken strips prepared at home, OR=21.3 (3.0-947.3)<br>Chicken nuggets and/or strips prepared at home, OR=3.8 (1.7-8.2)<br>Chicken wings, OR=3.8 (1.2-15.5) | (Currie *et al.*, 2005) |

| Country | Year | *Salmonella* serotype | No. cases[1] | No. cases and controls[2] | Exposures associated with increased disease risk OR/mOR (95% confidence interval, or *p*-value if OR not provided)[3] | Reference |
|---|---|---|---|---|---|---|
| | | | | | Undercooked eggs, OR=5.5 (1.2-51.1)<br>Deli chicken, OR=6.9 (1.8-41.4)<br>Roast beef, OR=2.2 (1.0-4.9) | |

N/A, not applicable as investigation based on a national rise in prevalence rather than an identified outbreak or cluster.

[1] Number of cases initially identified in the outbreak or cluster.
[2] Number of cases and controls included in the case control study.
[3] OR, odds ratio mOR, matched odds ratio aOR, adjusted odds ratio p, probability

# 9 APPENDIX 3: CONTROL MEASURES IN OTHER COUNTRIES

## 9.1 FAO/WHO

In 2007 the Codex Alimentarius Commission (CAC) agreed that the development of guidelines for the control of *Salmonella* and *Campylobacter* in poultry was a priority. The Codex Committee on Food Hygiene (CCFH) determined that the guidelines would consist of three sections, addressing good hygiene practices (GHP), hazard-based control measures and risk-based control measures (FAO/WHO, 2009). The CCFH In their most recent meeting in November-December 2010, the CCFH agreed that the draft guidelines, titled "Proposed Draft Guidelines for the Control of *Campylobacter* and *Salmonella* in Chicken Meat", were ready to be forwarded to the Commission for adoption at Step 5/8 (CAC, 2010).[36]

The proposed draft guidelines set out potential GHP-based and hazard-based control measures for *Salmonella* and *Campylobacter* on chicken meat from broilers, for each step in the food chain. They do not set quantitative limits for these pathogens. The intention is for Government and industry to use the guidelines to inform decisions on critical control points when applying HACCP principles and to set quantitative limits if they choose to.[37]

The CCFH envisaged that the third part of the guidelines, the risk-based control measures, should be used in conjunction with an internet-based risk-management decision-support tool. The draft guidelines recommend that national-level competent authorities should develop risk-based control measures for *Campylobacter* and *Salmonella* where possible and practical, and lists some key requirements for doing this. The Joint FAO/WHO Expert Meetings on Microbiological Risk Assessment (JEMRA) has developed a prototype internet-based tool, and expect to pilot a fully functional tool in 2011. The tool will allow a risk manager to input data specific to their production and processing system and evaluate measures that might be most effective for risk reduction in those particular conditions (FAO/WHO, 2009).

## 9.2 Australia

In May 2010 the FSANZ Australia and New Zealand Food Regulation Ministerial Council approved a draft Primary Production and Processing Standard for Poultry Meat (Poultry Standard). The poultry standard will become part of the Australia New Zealand Food Standards Code and will commence on 20 May 2012. The poultry standard only applies in Australia. As a result of the new standard, other changes to the Food Standards Code will be introduced at the same time, some of which apply in New Zealand (Table 30).

[36] Delegations from New Zealand (NZFSA) and Sweden jointly lead the guideline's development.
[37] The draft guidelines are available in Appendix III of the minutes for the 42nd session (Alinorm 11) of the Codex Committee on Food Hygiene (http://www.codexalimentarius.net/web/archives.jsp?lang=en (accessed January 2011).

**Table 30: Changes to the Australia New Zealand Food Standards Code from 20 May 2012***

| Standard | Deletions | Insertions | Applies to |
|---|---|---|---|
| 1.6.2 | Delete clause 4 (eviscerated poultry) | Deleted | Australia |
| 2.2.1 | Delete clause 2 (limit on fluid loss from thawed poultry) | Deleted | Australia, New Zealand |
| 2.2.1 | Delete schedule (determination of fluid in a package of frozen poultry carcass) | (none) | Australia, New Zealand |
| 4.4.1 | (none) | New standard (primary production and processing standards – preliminary provisions) | Australia |
| 4.4.2 | Delete standard | New standard (primary production and processing standard for poultry meat) | Australia |

* The Australia New Zealand Food Standards Code, including these changes, can be accessed at http://www.foodstandards.gov.au/foodstandards/foodstandardscode/ (accessed February 2010).

The poultry standard aims to reduce the incidence of foodborne illness from *Campylobacter* and *Salmonella* by minimising the prevalence and concentration of these two pathogens in poultry (FSANZ, 2010b). There are currently no regulatory measures in place for poultry growers to minimise the likelihood of poultry being contaminated with *Salmonella* and *Campylobacter* on-farm, although it has been reported that the majority of chicken and turkey growers comply with the *National Biosecurity Manual for Contract Meat Chicken Farming* developed by the Australian Chicken Meat Federation (FSANZ, 2010b).[38] The poultry standard requires poultry growers to identify and control the food safety hazards associated with the growing of poultry. In 2009 the Australian Government Department of Agriculture, Fisheries and Forestry published a new *National Biosecurity Manual for Poultry Production* that will, in part, assist poultry growers to meet their legal obligations under the poultry standard.[39]

Poultry processors will continue to be required to identify and control the food safety hazards associated with the processing of poultry (which includes the slaughtering process) and verify the effectiveness of the control measures (FSANZ, 2010b). Poultry processors also work to the *Australian Standard for Construction of Premises and Hygienic Production of Poultry Meat for Human Consumption* (AS 4465-2005), which requires poultry processors to develop and implement HACCP programs and also includes specific requirements relating to the design and construction of the premises, the processing of poultry, health and hygiene requirements and cleaning and sanitising.[40]

The Australian poultry sector began installing HACCP systems in the mid-1980s and whole bird rinse testing at the processing plants began in 1981 (Sumner *et al.*, 2004b). Researchers used published and unpublished data to identify whether the regulatory changes had affected

[38] The manual is available from http://www.chicken.org.au/ (accessed February 2011).
[39] The manual is available from http://www.daff.gov.au/animal-plant-health/pests-diseases-weeds/biosecurity/animal_biosecurity/bird-owners/poultry_biosecurity_manual (accessed February 2011)
[40] AS 4465-2005 is available from http://www.publish.csiro.au/pid/5203.htm (accessed February 2011).

the prevalence of salmonellosis. They found some evidence that the microbiological status of poultry had improved, but this improvement had not lead to any apparent reduction in case rates for salmonellosis.

## 9.3 European Union

### 9.3.1 Controls in flocks

Commission Regulation (EC) No 2160/2003 (control of *Salmonella* and other specified food-borne zoonotic agents) provides for the setting of targets for reduction of the prevalence of zoonoses and zoonotic agents, and requires Member States to establish national control programmes that cover feed production, primary production of animals, and processing and preparation of food of animal origin.[41] The Regulation requires targets to be set for all *Salmonella* serotypes with public health significance for the primary production of breeding flocks of *Gallus gallus* (domestic chickens), broilers and turkeys.

The targets are presented in Table 31. These targets were set after a set of baseline studies on *Salmonella* prevalence were completed (see Appendix 1). The regulations referred to in Table 31 also set out the sampling and testing requirements for Member States to demonstrate progress towards the targets.
In 2009, Regulation 1003/2005 was expanded to cover flocks of breeding turkeys via Commission Regulation (EC) No 213/2009.[42] A further Regulation released in March 2010, Regulation (EC) No 200/2010,[43] repealed Regulation 1003/2005 since the period over which the target applied finished on 31 December 2009 (see Table 31). Regulation 200/2010 required Member States to maintain the same breeding flock target from 1 January 2010.

The targets for broilers and turkey set under Regulations 646/2007 and 584/2008 are interim targets for *S.* Enteritidis and *S.* Typhimurium only. These Regulations allow for other serotypes with public health significance to be considered after a three-year transitional period, which will end 31 December 2011 for broilers and 31 December 2012 for turkeys.

[41] All EC regulations are available at: http://eur-lex.europa.eu/en/index.htm (accessed January 2011)
[42] Commission Regulation (EC) No 213/2009 of 18 March 2009 amending Regulation (EC) No 2160/2003 of the European Parliament and of the Council and Regulation (EC) No 1003/2005 as regards the control and testing of *Salmonella* in breeding flocks of *Gallus gallus* and turkeys.
[43] Commission Regulation (EU) No 200/2010 of 10 March 2010 implementing Regulation (EC) No 2160/2003 of the European Parliament and of the Council as regards a Union target for the reduction of the prevalence of *Salmonella* serotypes in adult breeding flocks of *Gallus gallus*.

**Table 31: *Salmonella* targets for EU Member States in chicken breeding flocks and flocks of broilers and turkeys**

| Target set for: | *Salmonella* serotypes included in target | Target | Regulation |
|---|---|---|---|
| Breeding flocks of *Gallus gallus* | *S.* Enteritidis<br>*S.* Hadar<br>*S.* Infantis<br>*S.* Typhimurium<br>*S.* Virchow | Reduction of the maximum percentage of adult breeding flocks comprising at least 250 birds remaining positive to 1 % or less by 31 December 2009.<br><br>For Member States with fewer than 100 breeding flocks, not more than one adult breeding flock shall remain positive. | 1003/2005[1] |
| Broilers | *S.* Enteritidis<br>*S.* Typhimurium | Reduction of the maximum percentage of flocks of broilers remaining positive of *S.* Enteritidis and *S.* Typhimurium to 1% or less by 31 December 2011. | 646/2007[2] |
| Turkeys | *S.* Enteritidis<br>*S.* Typhimurium | (a) reduction of the maximum percentage of fattening turkey flocks remaining positive of *S.* Enteritidis and *S.* Typhimurium to 1% or less by 31 December 2012; and<br>(b) reduction of the maximum percentage of adult breeding turkey flocks remaining positive of *S.* Enteritidis and *S.* Typhimurium to 1% or less by 31 December 2012.<br><br>For Member States with less than 100 flocks of adult breeding or fattening turkeys: No more than one flock of adult breeding or fattening turkeys may remain positive by 31 December 2012. | 584/2008[3] |

All regulations are available from http://eur-lex.europa.eu/en/index.htm (accessed January 2011)

[1] Commission Regulation (EC) No 1003/2005 of 30 June 2005 implementing Regulation (EC) No 2160/2003 as regards a Community target for the reduction of the prevalence of certain *Salmonella* serotypes in breeding flocks of *Gallus gallus* and amending Regulation (EC) No 2160/2003.

[2] Commission Regulation (EC) No 646/2007 of 12 June 2007 implementing Regulation (EC) No 2160/2003 of the European Parliament and of the Council as regards a Community target for the reduction of the prevalence of *Salmonella* Typhimurium and *Salmonella* Enteritidis in broilers and repealing Regulation (EC) No 1091/2005.

[3] Commission Regulation (EC) No 584/2008 of 20 June 2008 implementing Regulation (EC) No 2160/2003 of the European Parliament and of the Council as regards a Community target for the reduction of the prevalence of *Salmonella* Enteritidis and *Salmonella* Typhimurium in turkeys).

### 9.3.2 Controls in food

Commission Regulation (EC) No 2073/2005 (microbiological criteria for foodstuffs) and its amendment, No 1441/2007, were applied from 1 January 2006. These regulations set limits for the presence of *Salmonella* for 19 specific food categories and for three food products during processing, and prescribe rules for sampling and testing.

The Regulations require that *Salmonella* must be absent in poultry products placed on the market and during their shelf life according to the following testing regime:

- Minced meat and meat preparations made from poultry meat intended to be eaten cooked: n=5, c=0, m/M=absence in 10g from 1 January 2006 and absence in 25g from 1 January 2010.[44]
- Meat products made from poultry meat intended to be eaten cooked: n=5, c=0, m/M=absence in 10g from 1 January 2006 and absence in 25g from 1 January 2010.

*Salmonella* must also be monitored during poultry processing:

- Poultry carcasses of broilers and turkeys (after chilling): n=50, c=7, m/M=absence in 25g of a pooled sample of neck skin.[45]

In addition to the criteria above, poultry products might also fit into the following categories and be subject to their criteria:

- Minced meat and meat preparations intended to be eaten raw: n=5, c=0, m/M=absence in 25g.
- Mechanically separated meat (MSM): n=5, c=0, m/M=absence in 10g.

Compliance with these regulations is monitored by the EFSA (2008 data are presented in Appendix 1).

### 9.4 Scandinavia

In 1989, the Danish Poultry Council initiated voluntary control measures for poultry production after a decline in the *Salmonella* control of broilers in Denmark and problems with trade (Bisgaard, 1992). Initially the programme focussed on eradication of *Salmonella* from breeding and parent stock, and then controls were introduced for broiler farms and slaughterhouses. *Salmonella* monitoring programmes were established to measure the effectiveness of the controls. In 1992 the Ministry for Agriculture and Fisheries implemented the official control of *Salmonella* in broilers and since then the controls on broilers and layers have increased, primarily through the Danish National *Salmonella* Control Programme launched in December 1996 (CCFH, 2007; Helwigh, 2009). The programme was designed to be a top-down control effort based on the compulsory destruction or slaughter of infected flocks. *Salmonella* in turkey flocks was monitored from 1992, and ducks were monitored from 1999, however from March 2004 turkeys were no longer slaughtered in Denmark.

In 1989, the prevalence of *Salmonella* in broiler flocks 2-3 weeks prior to slaughter was >65%. This had reduced to <5% by the year 2000, and this level has been maintained (Danish Zoonosis Centre, 2010a; Wegener *et al.*, 2003). The incidence of human salmonellosis cases attributed to broilers has also declined, from approximately 30 per 100,000 in 1988 to <1 per 100,000 in 2009 (Danish Zoonosis Centre, 2010b). The major sources of human salmonellosis in 2009, excluding cases where the source was unknown, were travel, table eggs and pork. Ducks, imported ducks, imported chicken and imported turkey were the source of relatively small numbers of cases.

[44] N=number of units comprising the sample; c=number of sample units giving values between m and M (*Salmonella* should be absent in all raw poultry samples).
[45] c=the number of samples where the presence of *Salmonella* is detected. This c value is subject to review and EU Member States or regions having low *Salmonella* prevalence may use lower c values.

Similar *Salmonella* control programmes have been in place in Sweden and Finland, which are based on a zero-tolerance strategy including all *Salmonella* serotypes (CCFH, 2007). The prevalence of *Salmonella* contaminated flocks in these countries has been consistently low since the late 1990s and positive samples found after slaughter and in cutting plants have been very few.

## 9.5 USA

In 1996, the USDA FSIS published a final rule, ''Pathogen Reduction; Hazard Analysis and Critical Control Point (HACCP) Systems'' (61 FR 38806) (FSIS, 1996).[46] This final rule established a new food safety regulation for meat and poultry slaughter and processing plants (FSIS, 2005). The components of this programme were:

- Adoption of Sanitation Standard Operating Procedures (SSOPs) by every slaughter and processing plant (a written plan describing the daily procedures used to ensure sanitation during production);
- *Salmonella* performance standards for slaughter and ground product plants;
- Generic *E. coli* performance standards for slaughter plants.

FSIS is responsible for conducting the *Salmonella* sampling program for carcasses and raw product and developed the *Salmonella* performance standards by conducting nationwide baseline programs. Baseline programmes and microbiological surveys have continued to monitor trends. The performance standards for *Salmonella* in poultry are presented in Table 32.

**Table 32: USDA FSIS pathogen reduction performance standards for *Salmonella* in poultry**

| Product | Percent positive for *Salmonella* (%) | Number of samples tested per sample set | Maximum number of positive samples permitted |
|---|---|---|---|
| Broiler carcasses | 20.0 | 51 | 12 |
| Ground turkey | 49.9 | 53 | 29 |
| Ground chicken | 44.6 | 53 | 26 |
| Young turkey carcasses* | 19.6 | 56 | 13 |
| Goose carcasses* | 13.7 | 54 | 9 |

* Baseline guidance only (FSIS, 2006).

In 2010 the FSIS announced revised performance standards for *Salmonella* in young chickens and turkeys that will take effect from July 2011.[47] The standards were revised according to the results of baseline studies. The new performance standards will be much lower than the current standards (FSIS, 2010a):

- Broiler carcasses (post-chill): 7.5% prevalence, based on a 51-sample set (where a maximum of five positive samples are allowed to achieve the standard).

[46] Available at http://www.federalregister.gov/articles/1996/07/25/96-17837/pathogen-reduction-hazard-analysis-and-critical-control-point-haccp-systems (accessed January 2011).
[47] Details available at http://www.fsis.usda.gov/OPPDE/rdad/FRPubs/2009-0029.pdf (accessed January 2011).

- Turkey carcasses (post-chill): 1.7% prevalence, based on a 56-sample set (where a maximum of four positive samples are allowed to achieve the standard).