



**RISK PROFILE:  
SALMONELLA (NON TYPHOIDAL)  
IN  
PORK AND PORK PRODUCTS**

Prepared as part of a New Zealand Food Safety Authority  
contract for scientific services

by

Susan Gilbert  
Dr Rob Lake  
Peter Cressey  
Dr Andrew Hudson  
Nicola King

May 2010

Client Report  
FW08008

**RISK PROFILE:  
*SALMONELLA* (NON TYPHOIDAL)  
IN  
PORK AND PORK PRODUCTS**

Dr Stephen On  
Food Safety Programme Leader

Dr Rob Lake  
Project Leader

Dr Susan Paulin  
Peer Reviewer

## **DISCLAIMER**

This report or document (“the Report”) is given by the Institute of Environmental Science and Research Limited (“ESR”) solely for the benefit of the New Zealand Food Safety Authority (“NZFSA”), Public Health Services Providers and other Third Party Beneficiaries as defined in the Contract between ESR and the NZFSA, and is strictly subject to the conditions laid out in that Contract.

Neither ESR nor any of its employees makes any warranty, express or implied, or assumes any legal liability or responsibility for use of the Report or its contents by any other person or organisation.

## **ACKNOWLEDGEMENTS**

The authors wish to acknowledge the Ministry of Health as owner of the copyright and funders of the 1997 National Nutrition Survey and the 2002 National Children's Nutrition Survey and to thank them for access to these data.

We also acknowledge Grant Boston from the New Zealand Pork Industry Board and Vanessa Wintle from the New Zealand Feed Manufacturer's Association for providing valuable information.

## CONTENTS

<b>1</b>	<b>STATEMENT OF PURPOSE.....</b>	<b>3</b>
1.1	Food/Hazard Combination and Risk Management Questions.....	4
<b>2</b>	<b>HAZARD AND FOOD.....</b>	<b>5</b>
2.1	<i>Salmonella</i> .....	5
2.2	Sources of <i>Salmonella</i> .....	5
2.3	The Food Supply in New Zealand: Pork and Pork Products .....	6
2.3.1	Domestic pork production .....	6
2.3.2	Imported pork .....	7
2.3.3	<i>Salmonella</i> in the pork production chain.....	8
2.3.3.1	On farm .....	8
2.3.3.2	Primary processing.....	8
2.3.4	Behaviour of <i>Salmonella</i> on pork.....	9
2.3.4.1	Growth .....	9
2.3.4.2	Inactivation .....	10
2.4	Exposure Assessment.....	10
2.4.1	<i>Salmonella</i> in pigs .....	10
2.4.2	<i>Salmonella</i> in pork.....	10
2.4.3	Serotypes of <i>Salmonella</i> in pork and pork products.....	12
2.4.4	Food consumption: Pork.....	12
2.4.5	Evaluation of exposure .....	13
2.4.5.1	<i>Number of servings and serving sizes</i> .....	13
2.4.5.2	<i>Frequency of contamination</i> .....	14
2.4.5.3	<i>Predicted contamination level at retail</i> .....	14
2.4.5.4	<i>Growth rate during storage and most likely storage time</i> .....	14
2.4.5.5	<i>Heat treatment</i> .....	15
2.4.5.6	<i>Exposure summary</i> .....	15
2.5	Overseas Context .....	15
<b>3</b>	<b>EVALUATION OF ADVERSE HEALTH EFFECTS .....</b>	<b>16</b>
3.1	Disease Characteristics .....	16
3.2	Dose-Response.....	16
3.3	New Zealand Outbreak Information and Human Health Surveillance.....	17
3.3.1	Clinical Outcomes .....	18
3.3.2	Serotypes causing disease in New Zealand.....	19
3.3.3	Outbreaks.....	20
3.3.4	Case control studies and risk factors .....	21
3.4	Adverse Health Effects Overseas.....	22
3.5	Health Burden of Infection with <i>Salmonella</i> spp.....	22
3.6	Adverse Health Effects Summary.....	23
<b>4</b>	<b>EVALUATION OF RISK.....</b>	<b>24</b>
4.1	Existing Risk Assessments.....	24
4.2	Estimate of Risk for New Zealand.....	24
4.2.1	Risk associated with pork .....	24
4.2.2	Risk associated with other foods .....	25
4.3	Data Gaps.....	25

<b>5</b>	<b>AVAILABILITY OF CONTROL MEASURES .....</b>	<b>27</b>
5.1	Risk Management Strategy .....	27
5.2	Current Risk Management Measures .....	27
5.2.1	Relevant food controls .....	27
5.2.1.1	<i>The Animal Products Act</i> .....	27
5.2.1.2	<i>Import Health Standards</i> .....	28
5.2.1.3	<i>Regulation of antibiotic usage</i> .....	29
5.2.1.4	<i>NZ Pork activities in New Zealand</i> .....	29
5.3	Options for Risk Management .....	30
5.4	Commentary on Risk Management Options .....	31
<b>6</b>	<b>REFERENCES .....</b>	<b>32</b>
<b>APPENDIX 1:</b>	<b>HAZARD AND FOOD .....</b>	<b>45</b>
1.1	<i>Salmonella</i> .....	45
1.1.1	Growth and survival .....	45
1.1.2	Inactivation .....	46
1.2	The Food Supply: Pork and Pork Products .....	47
1.2.1	Pig production and processing with respect to <i>Salmonella</i> .....	47
	<i>Preharvest</i> .....	47
	<i>Post-harvest</i> .....	47
1.3	Prevalence of <i>Salmonella</i> in Pork and Pork Products Overseas .....	49
<b>APPENDIX 2:</b>	<b>EVALUATION OF ADVERSE HEALTH EFFECTS.....</b>	<b>56</b>
2.1	Adverse Health Effects Overseas.....	56
2.1.1	Contributions to outbreaks and incidents .....	57
2.1.2	Case control studies .....	60
2.1.3	Risk assessments and other activity overseas.....	61
<b>APPENDIX 3:</b>	<b>OVERSEAS CONTROL MEASURES.....</b>	<b>63</b>
3.1	Denmark and the Netherlands.....	63
3.2	USA.....	64

## LIST OF TABLES

Table 1:	Pork supply 2000 – 2008 (tonnes bone-in equivalent weight) .....	7
Table 2:	Presence of <i>Salmonella</i> in domestically produced and imported pork (Wong <i>et al.</i> , 2009) .....	11
Table 3:	National retail survey of <i>Salmonella</i> in raw minced/diced meat; August 2003 to May 2005 .....	11
Table 4:	<i>Salmonella</i> isolates derived from collective pork industry sources 2003 – 2008	12
Table 5:	New Zealand domestic meat consumption per capita 1985 to 2008 (kg/person/year) .....	13
Table 6:	Incidence data for salmonellosis in New Zealand .....	17
Table 7:	Outcome data for salmonellosis in New Zealand, 2003-2008 .....	19
Table 8:	Selected <i>Salmonella</i> serotypes and subtypes of laboratory-confirmed human isolates, 2005 – 2008 .....	19
Table 9:	Reported outbreak data for salmonellosis in New Zealand 2003 - 2008 .....	20
Table 10:	New Zealand outbreaks of salmonellosis with either epidemiological (suspected) links or laboratory confirmation linked with pork or pork product consumption 1999 – November 2009 .....	20
Table 11:	Prevalence of <i>Salmonella</i> in pigs and pork overseas .....	51
Table 12:	<i>Salmonella</i> in fresh pork samples in the EU (at cutting and retail level), 2007 ..	55
Table 13:	Reported incidence data for notified cases of salmonellosis overseas* .....	56
Table 14:	Ten most commonly confirmed human <i>Salmonella</i> serotypes in the EU, 2007 ..	57
Table 15:	Proportion of foodborne disease attributed to infection with <i>Salmonella</i> overseas .....	58
Table 16:	Examples of outbreaks of salmonellosis from consumption of pork overseas ....	59
Table 17:	Case control studies; Relevant risk/protective factors .....	60

## LIST OF FIGURES

Figure 1:	The four steps of the Risk Management Framework .....	3
Figure 2:	Incidence of notified salmonellosis in New Zealand 2000 - 2008 .....	18
Figure 3:	Process flowchart for pig slaughter .....	48

## EXECUTIVE SUMMARY

The purpose of a Risk Profile is to provide contextual and background information relevant to a food/hazard combination so that risk managers can make decisions and, if necessary, take further action. Risk Profiles include elements of a qualitative risk assessment, as well as providing information relevant to risk management. Risk profiling may result in a range of activities e.g. immediate risk management action, a decision to conduct a quantitative risk assessment, or a programme to gather more data. Risk Profiles also provide information for ranking of food safety issues.

NZFSA has recognised non-typhoidal *Salmonella* as one of the three most important foodborne pathogens in New Zealand. The organisation is taking a strategic approach to *Salmonella* Risk Management, with the ultimate aim of achieving a 30% reduction in foodborne salmonellosis after 5 years. Underpinning this strategy are a range of preliminary risk evaluation activities, including risk profiling to better understand the risk of *Salmonella* attributable to a range of food types.

This Risk Profile concerns pork (porcine muscle meat), and mechanically processed products such as mince and sausages. It does not cover uncooked, comminuted, fermented pork (UCFM), bacon or other fermented or cured pork products. Neither does it evaluate the risk from wild (feral) pork.

Approximately 45% of pork for domestic consumption was imported in 2007. Porcine Reproductive and Respiratory Syndrome (PRRS) is a viral disease of pigs. New Zealand has never had an outbreak of PRRS. Due to their PRRS-positive status, pork from some countries must be imported cooked or imported raw and cooked at a transitional facility, thereby inactivating any *Salmonella* if it is present.

The available data indicates a low prevalence of *Salmonella* contamination on pork in New Zealand; *Salmonella* was not found in small surveys of domestically produced carcasses (during and after primary processing), or in retail pork (minced or diced). A prevalence of 3.6% (95% CI 1.0 - 9.0%) was found in a small survey of imported pork.

Each of these surveys involved small numbers of samples, so the results must be treated with caution. Comparison with data from overseas surveys suggests that the prevalence of contamination in New Zealand is lower than overseas.

The rates of reported salmonellosis have fluctuated in the previous decade. Although rates (per 100,000 population) were generally higher between 1997 and 2002, since 2003 the rates have returned to below 40 per 100,000. Current rates of illness in New Zealand are similar to those in Australia and other developed countries.

The most common serotype of *Salmonella* derived from human cases in New Zealand is *S. Typhimurium*, with *S. Typhimurium* DT160 being the most common type. A case control study in 2001 did not indicate an elevated risk from pork products for *S. Typhimurium* DT160 human infection. However, consumption of pork steak was identified as a statistically significant risk factor for salmonellosis in another case control study conducted in 2002.



The serotype data from isolates submitted by the pork industry to the Enteric Reference Laboratory for typing are in such small numbers that comparison with human cases is statistically unsound and was not considered in this Risk Profile.

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.

Pork was implicated in nine outbreaks between the years 1999 to November 2009. Only one of the outbreaks had laboratory confirmation that the pathogen occurred in the suspected food (in this instance, pork cocktail sausages). In the other eight outbreaks, only epidemiological (suspected) linkage with pork was reported. However, the number of outbreak cases represents only a small proportion (approximately 10%) of the total reported cases, and the epidemiology of sporadic reported cases may be different.

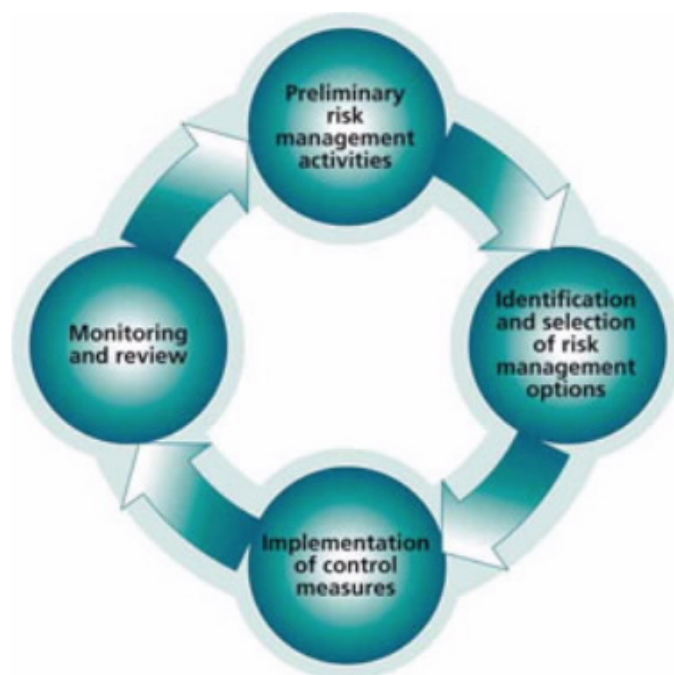
There are only limited data on the prevalence of *Salmonella* through the pork food chain, and none at all on concentrations of bacteria. Data on carriage of *Salmonella* by pigs prior to processing (and potential sources of infection e.g. pig feed) would seem to be the most pressing data gap, given the importance of this factor as an input into primary processing.

In relation to this data gap, a 12 month trial of porcine testing for *Salmonella* during primary processing was commenced in October 2009 under the testing programme of the National Microbiological Database.

Given the increasing proportion of New Zealand's pork supply that is imported, more detailed information on the prevalence and numbers of salmonellae in imported product is an important data gap.

## 1 STATEMENT OF PURPOSE

The purpose of a Risk Profile is to provide contextual and background 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) (<http://www.nzfsa.govt.nz/about-us/risk-management-framework/index.htm>) approach taken by the New Zealand Food Safety Authority (NZFSA). 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
- 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 pork and pork products.

NZFSA has recognised non-typhoidal *Salmonella* as one of the three most important foodborne pathogens in New Zealand. The organisation is taking a strategic approach to *Salmonella* Risk Management, with the ultimate aim of achieving a 30% reduction in foodborne salmonellosis after 5 years. Underpinning this strategy are a range of preliminary risk evaluation activities, including risk profiling to better understand the risk of *Salmonella* attributable to a range of food types.

## 2 HAZARD AND FOOD

### 2.1 *Salmonella*

This group of bacteria is comprised of two species: *Salmonella enterica*, which is divided into 6 subspecies (*enterica*, *salamae*, *arizonae*, *diarizonae*, *houtanae* and *indica*), and *Salmonella bongori* (Jay *et al.*, 1997). Most pathogenic isolates from humans and other mammals belong to subspecies I: *Salmonella enterica* subspecies *enterica*. Other *Salmonella enterica* subspecies and *Salmonella bongori* occur more commonly from cold blooded animals and the environment, and are of lower pathogenicity to humans and livestock.

*Salmonella* typing is primarily performed using serological identification of somatic (O), flagella (H), and capsular (K) antigens. There are more than 2400 different *Salmonella* serotypes.

*Salmonella enterica* serotypes are normally denoted in a shortened form that includes a non-italicised serotype name, e.g. *Salmonella enterica* subsp. *enterica* serotype Enteritidis becomes *Salmonella* Enteritidis. In older publications this may be represented as a full species name i.e. *Salmonella enteritidis*.

Further subtyping may be performed using susceptibility to bacteriophages. These types are denoted as phage type (PT) or definitive phage type (DT) numbers. These two terms are interchangeable and both are used in the literature.

*Salmonella* Typhi and *Salmonella* Paratyphi are serotypes which cause a 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 cholerae-suis* (SCS) is the equivalent porcine typhi-like serotype. SCS is not found in many countries but has a distinct pathogenic profile. These human and porcine typhoidal serotypes and the diseases they cause are not included in this Risk Profile.

Information on the behaviour of *Salmonella* in foods is given in Appendix 1.

### 2.2 Sources of *Salmonella*

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, numbers approximating  $10^6$ - $10^7$ /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  $6 \times 10^3$ /g has been recorded in one patient 48 days post-illness (Pether and Scott, 1982).

Animal: Most *Salmonella* infections in animals produce no clinical signs. Some serotypes are largely confined to particular animal reservoirs causing both systemic and enteric disease, for example *S. Cholerae-suis* 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). Animal feeds may be contaminated with salmonellae, although feeds that include animal products (e.g. meat and bone meal) should receive sufficient heat treatment to destroy the organism. *Salmonella* can also be found in mammals, fish, reptiles, amphibians, insects and birds.

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). Foods of non-animal origin which have been shown to be contaminated by *Salmonella* include coconut, barley, cereal powder, yeast, cottonseed, chocolate, soybean sauce, cider, watermelon, watercress, white and black pepper. The absence in New Zealand of *S. Enteritidis* types that can penetrate into eggs means that this food type is likely to be of lower risk here. Tahini, a product made from crushed sesame seeds, has been contaminated with *Salmonella* and 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 and water. They can remain viable for months in soil. The organism may also be dispersed in dust and aerosols generated during the handling and processing of animals. Contamination in the environment can act as a source of infection for other animals i.e. spreading by rodents or wild bird populations.

Transmission Routes: Salmonellae may be transmitted to humans via person to person transmission, contaminated food or water, animal contact, or from a contaminated environment. The faecal-oral route is the most common.

### **2.3 The Food Supply in New Zealand: Pork and Pork Products**

The term 'pork' concerns the uncured skeletal muscle meat of the pig. This Risk Profile concerns pork, and mechanically processed pork products such as mince, sausages and luncheon meat. It does not cover uncooked, comminuted, fermented pork (UCFM), bacon or other fermented or cured pork products, such as ham. Neither does it evaluate the risk from wild (feral) pork.

#### **2.3.1 Domestic pork production**

The New Zealand pork industry is relatively small and focussed on the domestic market. Statistics New Zealand reports 327 pig farms in New Zealand at 30 June 2007. A total of 743,805 pigs were slaughtered in New Zealand during the 2008 year (carcass weight total 50,115 tonnes), compared to 751,218 pigs slaughtered in the 2007 year (carcass weight total 51,041 tonnes) (New Zealand Pork, 2009). In 2009 there were 9 abattoirs in New Zealand that slaughter pigs. It has been estimated that approximately 15,000 pigs are slaughtered per week, with plant daily throughput being from 150 to over 1,000 animals (Titus, 2007).

The interests of the New Zealand pork industry are coordinated through the New Zealand Pork Industry Board (operational name: New Zealand Pork, or NZ Pork), a producer-funded body governed by the Pork Industry Board Act 1997. Commercial pork producers represent around 90% of pork production in New Zealand.

Table 1 shows data on the pork supply in New Zealand from 2000 to 2008. The trend is for increases in all areas of production, imports, and consumption. On-farm productivity gains have raised average kill weights. This means that, although there is a slight decline in pig numbers slaughtered, the average kill weight of 67.4 kg is maintaining production levels (New Zealand Pork, 2008). Consumer demand for fresh pork has been steadily increasing, with per

capita consumption of pork reaching 20.1 kg in 2005, 20.6 kg in 2006 and 21.4 kg during 2007, before falling back to 20.6 kg in 2008 (New Zealand Pork, 2008).

**Table 1: Pork supply 2000 – 2008 (tonnes bone-in equivalent weight)**

Year to September end	Domestic production, Tonnes (%)	Import Volume, Tonnes (%)	Total supply (consumption), Tonnes
2000	44,984 (69)	20,521 (31)	65,505
2001	45,893 (72)	17,616 (28)	63,509
2002	45,134 (67)	22,386 (33)	67,520
2003	47,222 (64)	26,467 (36)	73,689
2004	51,861 (67)	25,366 (33)	77,227
2005	50,845 (62)	31,862 (38)	82,707
2006	50,650 (59)	34,852 (41)	85,502
2007	50,183 (55)	40,434 (45)	90,617
2008	51,399 (58)	36,657 (42)	88,056

Source: New Zealand Pork, 2008

### 2.3.2 Imported pork

Porcine Reproductive and Respiratory Syndrome (PRRS) is a viral disease of pigs. This disease causes reproductive failure in breeding stock and respiratory tract illness in young pigs. First detected in North America in 1987, PRRS has since been recorded in most pig-producing areas of the world; only Australia, New Zealand and Switzerland are reported to have never had an outbreak. The pig is the only species known to be naturally susceptible to the virus<sup>1</sup>. The causative virus is not a human pathogen and PRRS is not a food safety issue. However, control measures are likely to have benefits for food safety through an increased focus on biosecurity and hygiene.

Pork imported from countries that have PRRS needs to be heat-treated (not necessarily cooked) or pH adjusted before arrival, or imported into a transitional facility in New Zealand where it is similarly heat-treated or pH adjusted prior to release onto the market. There are several Import Health Standards (IHS) which contain requirements for the importation of processed pork products<sup>2</sup>. As a PRRS free country, Australian pork can be processed in the same areas as domestically produced pork and can be sold uncooked. Most of the Australian imported pork is processed into bacon (Wong *et al.*, 2009).

Approximately 42% of pork for domestic consumption was imported in 2008, down from 45% in 2007 (New Zealand Pork, 2008). The imported proportion of the total supply increased steadily up to 2007, as shown in Table 1. New Zealand has been viewed as a growth market by pork exporting countries for several years (FAS, 2001). Based on product weight, sources of imported pork in 2008 were Australia (36.5%), Canada (28.4%), USA (22.4%) and Finland (11.0%) (New Zealand Pork, 2008). Imports from Sweden dropped from 5.9% during 2006 to 0% in 2007 due to an outbreak of PRRS which started early June 2007 (New Zealand Pork, 2006; 2007). Finland meanwhile remains PRRS free and began exporting pork to New Zealand in 2007. Small amounts of pork started to be imported from Sweden again in 2008.

<sup>1</sup> [http://www.fao.org/docs/eims/upload/235243/Focus\\_ON\\_2\\_07.pdf](http://www.fao.org/docs/eims/upload/235243/Focus_ON_2_07.pdf)

<sup>2</sup> <http://www.maf.govt.nz/biosecurity/imports/animals/standards/index.htm>

The majority of pork imports in 2008 were frozen (94%), with the remainder chilled (1.3%), processed (3.0%), and cured (2.0%) (New Zealand Pork, 2008).

### 2.3.3 Salmonella in the pork production chain

#### 2.3.3.1 *On farm*

The types of *Salmonella* considered by this Risk Profile do not usually cause illness in pigs (Jay *et al.*, 2003). There are several transmission routes proposed for the infection of pigs: *Salmonella* in faeces, direct pig-to-pig spread, endemic flora in finishing sites, and breeding farms. Observed sources of contamination include rodents, insects, birds, other animals, humans and contaminated feed. Vertical transmission of *Salmonella* in pigs has been proposed by many researchers but has not been clearly established (Davies *et al.*, 1998).

Animal by-products used in animal feed are a recognised source of *Salmonella* spp. Heat applied during processing destroys salmonellae but re-contamination can occur post-processing and salmonellae can survive for long periods in dried meals (ICMSF, 1980).

Pigs may receive treatment for a variety of diseases from birth, although some treatments that are incidentally active against *Salmonella* are not introduced until the weaner stage (4-5 weeks). Because salmonellosis in pigs is rare in New Zealand, specific medications for treatment are used minimally.

Prior to slaughter animals are housed temporarily in lairage pens in the abattoir to allow pigs to recover from transport stress, and to serve as a reservoir of pigs for the slaughter facility. Cross infection during transport and lairage (a holding period without feed prior to primary processing) has been cited as a major problem. Minimising the time between farm and slaughter is believed to reduce this risk (Beloeil *et al.*, 2004). Research by Hurd *et al.*, (2002) found rapid infection rates during transport and during lairage. The *Salmonella* isolation rate in pigs slaughtered at the abattoir (39.9%) was seven times higher than for pigs killed on-farm (5.3%). A two hour lairage holding time is recommended which is the minimum time needed for pigs to recover from the stress of transport (Warriss *et al.*, 1992).

#### 2.3.3.2 *Primary processing*

Pig primary processing involves the following steps (summarised from Titus, 2007):

- Killing: Killing involves electrical stunning, shackling and elevation by the hind legs, and insertion of a knife resulting in death from ex-sanguination.
- Scalding: the carcass is immersed in a tank of water between 58 and 65°C for 6-10 minutes. This process results in the accumulation of dirt and faeces in the tank. In the three New Zealand processing plants visited for the PhD study (Titus, 2007), no water replacement or water flow was observed for the scalding tank.
- Dehairing: one or more carcasses are placed in a rotating drum with built in metal paddles that scrape the hair and outer layer of skin off the carcass. A water spray removes hair and debris.
- Singeing: Flames from a blow torch remove any remaining hair and tighten the skin. Skin temperatures of up to 100°C have been reported, although heat treatment of surfaces may be uneven, particularly in the groin, ears and fore-leg pits areas (ICMSF,

1998). Following singeing, polishing may be performed (with manual or mechanical brushes) to remove any black rind.

- Evisceration, trimming and halving: Removal of entrails (with associated potential for faecal contamination of the carcass from puncturing the intestine). Trimming is the manual removal of visibly contaminated or damaged regions on the carcass, while halving is achieved with a mechanical saw.
- Chilling/storage: The reduction of the temperature of carcasses to 4-7°C, using blast or conventional chillers (blast chillers appear to be standard in New Zealand). This controls bacterial numbers, but too rapid chilling can reduce meat quality.

High water temperatures in the scalding tank, and high skin temperatures during the singeing process reduce *Salmonella* numbers. Based on the USA scalding process (58.8°C for 6 minutes) (Dickson *et al.*, 2002) a 9 log reduction would be expected in the salmonellae population.

Evisceration is identified as the step having the greatest potential for *Salmonella* contamination. Berends *et al.* (1997) estimate 55-90% of contamination occurs at this point.

Supplemental information on pork processing and associated *Salmonella* contamination is given in Appendix 1.

#### 2.3.4 Behaviour of *Salmonella* on pork

The water activity ( $a_w$ ) of pork is approximately 0.99. The normal pH of pork reaches 5.6 - 6.0, 3 to 5 hours post slaughter.

##### 2.3.4.1 *Growth*

A study was undertaken to determine growth of *S. Typhimurium*, *S. Enteritidis* and background flora at various temperatures in minced and boneless pork. The inoculated meat was held at 5 different temperatures to simulate processing and holding (4.4, 7.2, 10°C) and ambient (22.2, 23.3°C) temperatures (Mann *et al.*, 2004). No significant growth of *Salmonella* in boneless chops or minced pork occurred at the refrigeration temperature for up to 72 hours. At room temperature, growth (2 log<sub>10</sub> CFU in 4 hours) was observed after 8 hours. The results indicate that processing pork at 4.4°C would not result in any *Salmonella* growth. Neither would processing at either 7.2°C or 10°C (for < 12 hours). Processing at ambient temperatures may result in significant *Salmonella* growth after 6-8 hours. It was therefore recommended that a processor operating at an ambient temperature should ensure that the product enters a refrigerated area within 6 hours of processing completion.

Similar results were noted in experiments with *Salmonella* (on beef), with no growth recorded at 7 to 8°C and a minimum generation time of 8.1 hours at 10°C. Generation times decreased as expected with increased temperature; 5.2 hours at 12.5°C and 2.9 hours at 15°C (Mackey *et al.*, 1980).

A predictive computer tool, "THERM" (temperature history evaluation for raw meats) has been developed for the growth of pathogens, including *Salmonella* in pork, during short term temperature abuse (Ingham *et al.*, 2007). The model was developed from experiments on raw minced pork samples inoculated with a five-strain cocktail and held at temperatures between 10°C and 43.3°C. Data that may be calculated include log<sub>10</sub> CFU values, lag-phase duration



and growth rate. Accuracy of the model was tested against 20 different inoculation experiments on a range of meat species under various temperature-abuse scenarios. THERM accurately predicted pathogen growth in 85% of the *Salmonella* experiments and made fail-safe predictions in the remaining 15%.

In New Zealand, based on Industry Standards 3 and 6 (NZFSA, 2004; 2006) any wet processing room that handles raw un-preserved product must be maintained at  $\leq 12^{\circ}\text{C}$  ( $10^{\circ}\text{C}$  is the norm). Slaughter facility areas and offal rooms are exempt from this requirement and can operate at ambient temperatures. In the case of warm boning (IS6: section 3.7.4), carcasses must be reduced to  $7^{\circ}\text{C}$  within 20 hours of the carcass leaving the slaughter floor.

#### 2.3.4.2 *Inactivation*

Heat resistance of salmonellae in foods is partly dependent on food composition; for example the presence of fat has been shown to exert a protective effect on *Salmonella* cells. A paper on beef (no papers on pork were found) experimented with varying fat contents and found that thermal inactivation of *S. Typhimurium* DT 104 did not begin until after a so-called “lag” period. The length of this period was proportional to the fat content; for example at  $58^{\circ}\text{C}$  the “lag” period was 4 minutes (at 7% fat) and 28 minutes (at 24% fat). However, once log linear death commenced D values tended to be lower at higher fat concentrations (Juneja and Eblen, 2000).

## 2.4 Exposure Assessment

### 2.4.1 *Salmonella* in pigs

There is little existing information on the carriage of *Salmonella* by New Zealand pigs (a 12 month trial introducing the testing of porcine carcasses at primary processing under the National Microbiological Database (NMD) commenced in October 2009). A limited survey of 134 swabs taken from faecal pats from pigs in lairage at three New Zealand plants in 2007 did not detect *Salmonella* (Titus, 2007).

### 2.4.2 *Salmonella* in pork

To examine the prevalence of *Salmonella* in New Zealand pork, a total of 100 New Zealand produced chilled pig carcass samples and 110 imported (Australia, USA, Canada) pork samples were obtained from processors between October 2004 and May 2005 (Wong *et al.*, 2009). The domestic pig carcasses originated from four New Zealand abattoirs. Ninety-five of the carcasses came from the South Island. The pig carcasses were swabbed with a sponge over a  $100\text{ cm}^2$  template. Swabs of pork from Canada and the USA were taken before the meat was cooked under PRRS requirements. The imported meat was either excised or swabbed as for domestic samples. *Salmonella* was tested using a presence/absence procedure.

*Salmonella* was not isolated from domestic pig carcasses or from pork imported from Canada and the USA; only samples of imported Australian pork were positive. Results with 95% confidence intervals are displayed in Table 2.

**Table 2: Presence of *Salmonella* in domestically produced and imported pork (Wong *et al.*, 2009)**

Country	Samples tested	No. positive for <i>Salmonella</i> spp.	% positive	Confidence interval (95%)
New Zealand	100	0	0	0 – 3.6
Australia	65	4	6.2	1.7 – 15.2
Canada	19	0	0	0 – 17.6
USA	26	0	0	0 – 13.2

These data are from surveys with small sample size. The four positive carcass swabs (of Australian origin) were all serotype *S. London* and the isolates were indistinguishable. This strain could be endemic in the Australian porcine population that supplies the New Zealand market. However, the number of salmonellosis cases in both Australia and New Zealand linked to this serotype is small (Wong *et al.*, 2009). The authors concluded that because this uncommon serotype had been isolated from raw imported pork, such importation was a potential route for the introduction of new serotypes into New Zealand.

Pig carcasses (n = 130) were swabbed (225 cm<sup>2</sup> representing 1.6% of the average total surface area) before and after abattoir procedures (scalding, dehairing, singeing, evisceration, trimming and halving) in two different abattoirs in New Zealand and the swabs tested for *Salmonella* (Titus, 2007). No salmonellae were found on the carcass swabs, either before or after processing. This prevalence result (0%, 95% CI 0.0 -2.8%) was from a survey representing only a small proportion of pig processing, but is consistent with the result above (Wong *et al.*, 2009).

A national quantitative survey of *Salmonella* in 25g samples of five types of uncooked retail meats, including pork, was conducted from August 2003 to May 2005 (Wong *et al.*, 2007). *Salmonella* was not detected in the pork samples, and the total prevalence of *Salmonella* in all 1108 meat samples was 1.1% (Table 3).

**Table 3: National retail survey of *Salmonella* in raw minced/diced meat; August 2003 to May 2005**

Meat (all minced/diced)	No. samples tested	Total number positive	% prevalence (95% CI)
Pork	231	0	0.0 (0-1.6)
Beef	232	1	0.4 (0-2.4)
Bobby veal	183	1	0.5 (0-3.0)
Lamb/mutton	230	3	1.3 (0.3-3.8)
Chicken	232	7	3.0 (1.2-6.1)
<b>All samples</b>	<b>1108</b>	<b>12</b>	<b>1.1 (0.6 – 1.9%)</b>

It was estimated that the overall number of samples (chicken, lamb/mutton, unweaned veal, beef and pork; n=1108) would give 99% confidence of detecting contamination at retail at a rate of 2% or greater per meat type. If the prevalence of *Salmonella* contamination in pork were the same as for all meats (1.1%), a sample size of 231 would have approximately 92% probability of detecting at least one positive sample.

### 2.4.3 Serotypes of *Salmonella* in pork and pork products

The ESR Enteric Reference Laboratory (ERL) undertakes typing of *Salmonella* isolates from human and non-human sources for New Zealand<sup>1</sup>. Isolate types from all porcine sources (which may be animal, environment, or meat) are collated in Table 4. Due to the small numbers of submitted isolates attempting comparisons with isolates from human salmonellosis cases is not possible.

**Table 4: *Salmonella* isolates derived from collective pork industry sources 2003 – 2008**

Serotype	2003	2004	2005	2006	2007	2008	Total
<i>S. Agona</i>			1				1
<i>S. Bovismorbificans</i>					2		
<i>S. Brandenburg</i>		3		1			4
<i>S. Hindmarsh</i>				1			1
<i>S. Saintpaul</i>		1					1
<i>S. Typhimurium</i> PT RDNC	1			1	1		9
156	1						
160	2		1				
193	1		1				
135			1				
8						1	
Group B 4,12:-:1,2				2			2
<b>Total</b>	<b>5</b>	<b>4</b>	<b>4</b>	<b>5</b>	<b>3</b>	<b>1</b>	<b>18</b>

RDNC Reacts but does not conform

Source: [http://www.surv.esr.cri.nz/enteric\\_reference/enteric\\_reference.php](http://www.surv.esr.cri.nz/enteric_reference/enteric_reference.php)

Unlike human isolates, *Salmonella* isolates from non-human sample sources are not consistently sent to a secondary laboratory for confirmatory testing and further typing. Only isolates obtained through NZFSA's National Microbiological Database testing programme are required to be sent to ERL for serotyping; there is no legislative requirement for other non-human isolates to be submitted. Overall, the proportion of isolates referred to ERL is unknown and likely to be only a small proportion of the isolates found by primary laboratories (Lake and Sexton, 2009). Data providing greater detail on the original source of the sample, and other valuable surveillance data such as geographic region of source, are largely lacking on the referral forms sent to ERL. In addition, the information given can be misleading, for example, an isolate labelled only as poultry may have come from a live animal, from poultry meat, from poultry litter or even poultry feed (Lake and Sexton, 2009).

### 2.4.4 Food consumption: Pork

A major shift in meat consumption patterns has taken place in New Zealand during the last 20 years, with major gains by the poultry and smaller gains in the pork industries (see Table 5).

<sup>1</sup> [http://www.surv.esr.cri.nz/enteric\\_reference/enteric\\_reference.php](http://www.surv.esr.cri.nz/enteric_reference/enteric_reference.php)

**Table 5: New Zealand domestic meat consumption per capita 1985 to 2008 (kg/person/year)**

Year	Pork	Mutton and Lamb	Beef and Veal	Poultry
1985 <sup>1</sup>	14.2	27.3	36.5	15.0
1995 <sup>1</sup>	15.7	23.2	34.6	26.2
1996 <sup>1</sup>	16.1	20.6	37.8	25.1
1999 <sup>1</sup>	17.1	14.3	31.2	26.8
2001 <sup>1</sup>	16.5	16.6	27.1	31.0
2006 <sup>2</sup>	19.6	13.0	34.2	36.5
2007 <sup>3</sup>	21.4	NA	NA	NA
2008 <sup>3</sup>	20.6	NA	NA	NA

NA = Not available

<sup>1</sup>From New Zealand Meat and Wool Board's Economic Service (MWBES) Annual Review of the Sheep and Beef Industry, 1999-2000. Available from: <http://www.beef.org.nz/statistics/sld002.asp>

<sup>2</sup>Meat & Wool New Zealand Compendium of Farm Facts. Available at: <http://www.meatnz.co.nz/main.cfm?id=261> (year ending September 2006)

<sup>3</sup>NZ Pork Industry Annual Report 2008 [http://www.pork.co.nz/nzpork/annual\\_report/2008\\_annual\\_report.pdf](http://www.pork.co.nz/nzpork/annual_report/2008_annual_report.pdf)

Other estimates of food consumption can be gained by reference to Food Balance Sheets (<http://faostat.fao.org/>). These consider the amount of food available for consumption in a country. Reference to Food Balance Sheets indicates that per capita pork consumption in New Zealand (22.0 kg/person/year) is similar to Australia (21.9 kg/person/year) and slightly less than the UK and USA (26.2 and 29.4 kg/person/year, respectively). These figures are for the 2005 year, the most recent year of data available through the FAO database.

The figures given above represent the meat available for consumption in New Zealand. Information on amounts of meat reported to be actually consumed can be abstracted from the 1997 National Nutrition Survey (NNS) (Russell *et al.*, 1999). FSANZ have carried out an analysis of this dataset (ANZFSA, 2001), including application of a set of standard recipes, to allow composite foods to be reduced to their component parts. Data from the 1997 NNS gave an estimate of pork consumption for New Zealand of 32.3 g/day (11.8 kg/person/year). The difference between this estimate of pork consumption and the estimate in Table 5 is presumably due to wastage (meat available for consumption, but not consumed), and under-reporting in the National Nutrition Survey (NNS).

Approximately 50% of pork in New Zealand is cut and marketed as fresh product, while the remainder is processed into ham, bacon and smallgoods (New Zealand Pork, 2004).

## 2.4.5 Evaluation of exposure

### 2.4.5.1 *Number of servings and serving sizes*

Analysis of 24 hour dietary recall records from the 1997 National Nutrition Survey (NNS; Adults 15+ years old) and the 2002 Children's National Nutrition Survey (CNS; children 5-15 years) revealed no major difference between the frequency of consumption of pork and pork products by the two population groups (Cressey *et al.*, 2006). On average, 37.8% of the population will consume pork or pork products on any given day. However, when pork products not covered by this risk profile (bacon, ham, salami) are excluded only 17.8% of adults and 21.1% of children reported eating pork in the previous 24 hour period. From the

NNS, 937 individual dietary records were deemed to represent consumption of a serving of pork, while 800 records in the CNS related to consumption of pork. The 2006 New Zealand census reported 3,096,273 people 16 years and older usually resident in New Zealand (76.9% of total population) and 656,589 people (16.3%) aged 5-15 years (<http://www.stats.govt.nz/>). It was assumed that children younger than one year will not eat pork, that children 1-4 years (218,445 in 2006; 5.4%) will consume similar amounts of pork to children 5-15 years and that the proportions of the age groups in the population are unchanged since 2006. Using the survey populations of 4636 (NNS) and 3275 (CNS) and a national population of 4,352,641 (<http://www.stats.govt.nz/> population clock, as of 28 January 2010):

$$\begin{aligned} \text{Annual number of servings (total population)} &= ((937 \times (4,352,641 \times 0.769)/4636) + \\ & (800 \times (4,352,641 \times 0.217)/3275)) \times 365 \\ &= 3.3 \times 10^8 \text{ servings} \end{aligned}$$

This represents a high number of servings, as would be expected from a commonly consumed food such as pork.

Based on the data in the NNS and CNS databases the 50, 75, 95, and 99<sup>th</sup> percentile serving sizes for pork, as defined in this risk profile, in New Zealand were:

Percentile	Serving size NNS (g)	Serving size CNS (g)
50	29	28
75	69	52
95	156	143
99	284	261

In other words, half of pork meals consumed by New Zealanders will result in consumption of 28-29 g or less of pork, while only 1% of pork meals will result in consumption of more than 261-284 g of pork. The modest median serving size for pork is due to the fact that pork is predominantly consumed as an ingredient of foods, such as sausage and luncheon, rather than as intact cuts (chops, roast, steak, etc.).

#### 2.4.5.2 Frequency of contamination

This parameter is uncertain for New Zealand, but based on available studies with low sample numbers, the indications are that the frequency of contamination of raw pork is reasonably low.

#### 2.4.5.3 Predicted contamination level at retail

Unknown. The available data from a 2004-2005 retail survey which did not find any contamination in 231 samples (95% CI 0 – 1.6%) suggests a low prevalence of contamination.

#### 2.4.5.4 Growth rate during storage and most likely storage time

Refrigerated trucks transport meat from abattoir to retail outlets. In the North Island, based on one supermarket chain obtaining pork from an Auckland based abattoir, transit times were between 0.5 to 10 hours (Titus, 2007). From receipt at the supermarket to sale, fresh pork has an average turnover of two days (based on a meat retailer questionnaire administered in

Palmerston North in connection with a PhD thesis, Titus 2007). No data are available for *Salmonella* counts in cooked pork.

#### 2.4.5.5 Heat treatment

The pork and pork products considered in this Risk Profile will be eaten cooked, which would be expected to reduce considerably the numbers of any *Salmonella* present.

#### 2.4.5.6 Exposure summary

The information presented above indicates that pork products are commonly eaten, but the probability of contamination by *Salmonella* is low.

## 2.5 Overseas Context

*Salmonella* spp. contamination in pigs and pork appears to be more prevalent overseas than in New Zealand, although this may be due to differences in sampling and testing regimes. Data from overseas are collated in Table 11, Appendix 1. However, estimates of the prevalence of *Salmonella* in New Zealand pigs and pork are currently based on relatively small surveys and estimates will be improved through the operation of the porcine NMD programme.

The European Food Safety Authority (EFSA, 2009) has collated data from fresh pork at the slaughterhouse, at cutting/processing plants and at retail. This is over a time period of 2004 to 2007 and not all member states are represented. In 2007, *Salmonella* was present in 0 - 8.9% of samples (25g) from cutting plants in 7 European countries (n=5913). At retail, this figure was 0 - 6.1%, (n= 2861) (EFSA, 2009).

### 3 EVALUATION OF ADVERSE HEALTH EFFECTS

#### 3.1 Disease Characteristics

*Incubation:* 6-48 hours (usually 12-36 hours).

*Symptoms:* Diarrhoea, abdominal pain, vomiting, nausea and fever lasting 1-7 days. Hospitalisation rate estimated at 22.1%, case fatality rate 0.8%.

*Condition:* Salmonellosis.

*Toxins:* Toxins are not produced in foods.

*People Affected:* The young, old, and immunocompromised are particularly at risk. In addition people of less privileged socioeconomic groups and those living in higher population densities are more at risk.

*Long Term Effects:* Septicaemia and subsequent extra-intestinal infections can occur. Reactive arthritis may occur 3-4 weeks after gastrointestinal symptoms. Approximately 2% of a population exposed to a triggering infection will develop reactive arthritis. The disease usually resolves within six months, but may persist for more than a year in some cases (Hannu *et al.*, 2006).

*Treatment:* The infection is usually self-limiting, uncomplicated gastroenteritis although fluid replacement may be required, especially in the elderly or young children. Less than 2% of clinical cases require antibiotic treatment. The site of infection and the immunity status of the case determines treatment choices. Cases of salmonellosis due to *S. Typhimurium* DT104 are of increasing concern in the UK due to the organism's resistance to ampicillin, chloramphenicol, streptomycin, sulphonamides, tetracycline, trimethoprim and ciprofloxacin. The result is that disease due to these strains is becoming more difficult to treat (USDA, 2005).

Supplemental information on adverse health effects is given in Appendix 2.

#### 3.2 Dose-Response

The dose required to cause disease varies and is multi-factorial. Low attack rates were observed in one outbreak where 4-45 cells were consumed, and another where the dose was 6 cells in 65g of food (Anonymous, 1996a). Different serotypes may have different dose responses, but *Salmonella* is generally understood to cause disease with high attack rates at doses of  $10^5$  to  $10^7$  cells.

The most commonly used dose-response model was produced by the joint risk assessments of *Salmonella* in eggs and broiler chickens by FAO/WHO (2002). Results from a number of human feeding trials of *Salmonella* serotypes have been analysed to develop dose-response models (most recently by Oscar (2004) using a three phase linear model). These feeding trials have a number of deficiencies, particularly at low doses, as described in the FAO/WHO report. Consequently the FAO/WHO model augmented the data with information from outbreak reports. These reports were screened and a final 20 outbreaks were used in the database (12 Enteritidis, 3 Typhimurium, Heidelberg, Cubana, Infantis, Newport and Oranienburg). Several vehicles of transmission were implicated including meat, eggs, dairy products and water. A

beta-Poisson model was used to develop the mathematical relationship, and a maximum likelihood technique used to generate the curve best fitting the data. The graph shows that for the ingestion of  $10^{10}$  cells there was a probability of around 0.9 of illness, while the ingestion of  $10^1$  cells resulted in a probability of around 0.02. Thus the probability of illness from exposure to small doses is low. For outbreaks where food has only a low concentration of contamination, but has been widely consumed, a small proportion of consumers will become ill.

It has been repeatedly reported that the probability of disease following ingestion of small numbers of cells is higher when the implicated food has a high fat or protein content. For example, chocolate or peanut butter may protect cells from gastric juices so permitting a lower dose than usual to cause infection. Experimentation has also shown this to be the case for high fat foods (minced beef) and high protein foods (egg white). It was concluded that the pH of the microenvironment of the organism in the food matrix is crucial in determining its resistance to stomach acids (Waterman and Small, 1998).

An outbreak of *S. Typhimurium* not used in the FAO/WHO model involved consumption of roast pork. The dose causing disease was calculated to be  $2.6 \times 10^5$  MPN/g. The outbreak occurred in a home for mentally disabled students in Kanagawa, Japan. The roast pork stored at the caterer's facility was found to contain  $4.3 \times 10^4$  MPN/g. From 140 people, 105 exhibited food poisoning symptoms, an attack rate of 75% (the FAO/WHO model predicts a probability of illness between 0.5-0.75) (Murase *et al.*, 2000).

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

The number of cases and incidence of notified salmonellosis since 2003 is shown in Table 6.

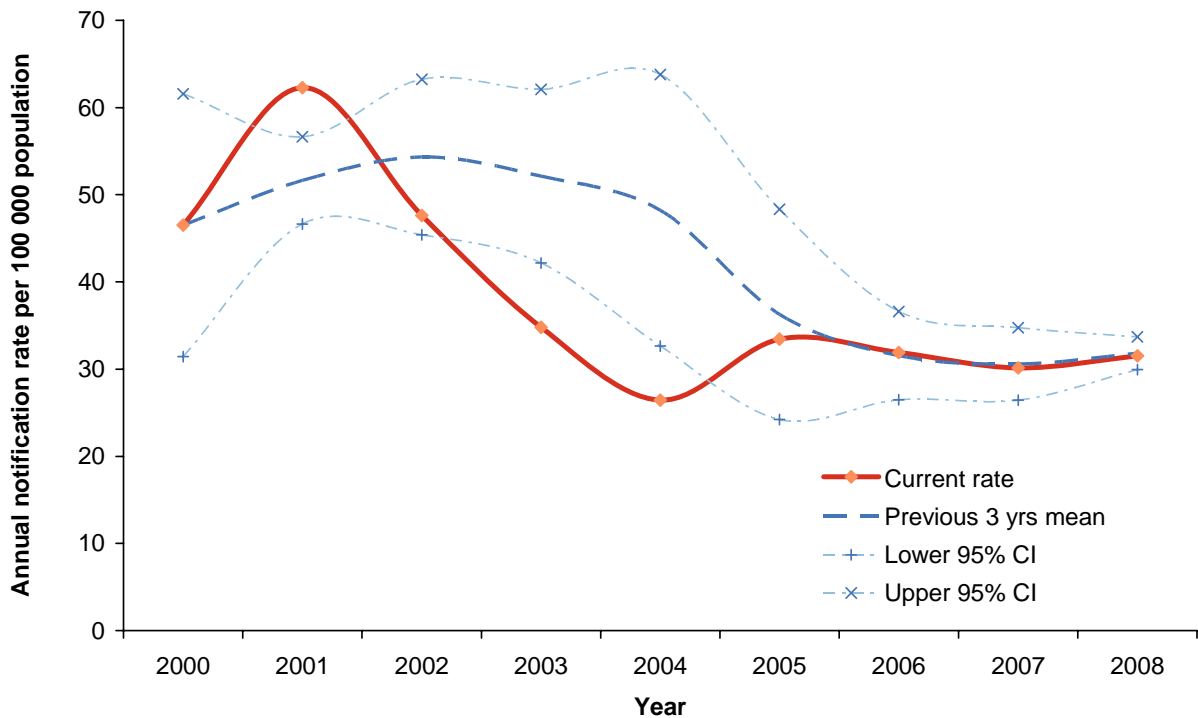
**Table 6: Incidence data for salmonellosis in New Zealand**

Year	Number of cases	Incidence (cases/100,000)
2003	1,401	37.5
2004	1,080	28.9
2005	1,383	37.0
2006	1,335	32.3
2007	1,274	30.1
2008	1,346	31.5

The notification rate per 100,000 population for cases of salmonellosis in New Zealand from 2000 – 2008 is shown in Figure 2. The rate has been stable since 2005 at approximately 30 per 100,000.



**Figure 2: Incidence of notified salmonellosis in New Zealand 2000 - 2008**



Reproduced from Williman *et al.* (2009)

The frequency of salmonellosis notifications is characterised by a late summer peak and a winter trough.

Highest rates are often reported from the lower South Island; in 2008 the highest rates were from South Canterbury (37 cases, 66.9/100,000) and Otago (129 cases, 68.9/100,000).

In terms of gender, the rates are similar for males (33.6/100,000 in 2008) and females (28.6/100,000 in 2008). Age specific rates are highest for the <1 year age group (135.8/100,000 in 2008), and 1 to 4 year olds (108.9/100,000 in 2008).

### 3.3.1 Clinical Outcomes

Hospitalisation and fatality rates for notified cases of salmonellosis in New Zealand are given in Table 7. These outcomes are not always reported for each case, so percentages are expressed in terms of the number of cases for which outcomes are reported. The hospitalisation rate and number of deaths has been stable over many years.

**Table 7: Outcome data for salmonellosis in New Zealand, 2003-2008**

Year	Hospitalised cases	Fatalities	Reference
2003	167/1118 (14.9%)	0/1401	ESR, 2004a
2004	109/871 (12.5%)	0/1080	ESR, 2005a
2005	142/1134 (12.5%)	1/1383 (0.07%)	ESR, 2006a
2006	148/1111 (13.3%)	1/1335 (0.07%)	ESR, 2007a
2007	110/833 (13.2%)	1/1274 (0.08%)	ESR, 2008a
2008	123/896 (13.7%)	1/1346 (0.07%)	ESR, 2009a

### 3.3.2 Serotypes causing disease in New Zealand

The principal serotypes of *Salmonella* identified from notified cases in New Zealand for the period 2005-2008 (Williman *et al.*, 2009) are *S. Typhimurium* (approximately 50% to all identified isolates, with the most frequent definitive phage type being DT160), and *S. Enteritidis* (approximately 10%).

Table 8 shows the trend for the number of human *Salmonella* isolates for selected serotypes or phage types during the period 2005-2008.

**Table 8: Selected *Salmonella* serotypes and subtypes of laboratory-confirmed human isolates, 2005 – 2008**

Subtype	2005	2006	2007	2008
<i>S. Typhimurium</i>	757	733	596	729
DT160	248	260	152	135
DT42	27	28	15	93
DT101	67	71	43	72
DT1	114	72	91	72
DT156	75	87	73	67
DT74	28	42	29	21
Other or unknown	198	173	193	269
<i>S. Enteritidis</i>	151	107	151	124
PT9a	73	53	60	45
PT1b	9	9	18	19
PT26	9	7	17	10
Other or unknown	60	38	56	50
<i>S. Infantis</i>	67	58	86	86
<i>S. Chester</i>	0	1	37	64
<i>S. Mbandaka</i>	8	22	14	39
<i>S. Saintpaul</i>	65	35	25	35
<i>S. Brandenburg</i>	68	55	47	33
<i>S. Virchow</i>	16	13	34	14
Other or unknown serotypes	274	319	277	215
<b>Total</b>	<b>1 406</b>	<b>1 343</b>	<b>1 267</b>	<b>1 339</b>

Reproduced from Williman *et al.* (2009)

### 3.3.3 Outbreaks

The number of reported outbreaks of salmonellosis in recent years in New Zealand is given in Table 9 (figures exclude *S. Typhi* and *S. Paratyphi*). The number of cases reported as outbreaks is approximately 10% of those reported as sporadic cases.

**Table 9: Reported outbreak data for salmonellosis in New Zealand 2003 - 2008**

Year	Salmonellosis outbreaks/ total enteric outbreaks	Cases/Total Enteric Cases*	Reference
2003	23/315 (7.3%)	59/2649 (2.2%)	ESR (2004b)
2004	5/313 (1.6%)	74/3971 (1.9%)	ESR (2005b)
2005	26/338 (7.7%)	120/2343 (5.1%)	ESR (2006b)
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 (2009b)

\* Includes both suspected and confirmed cases

A search of the Episurv outbreak database was carried out in November 2009 to identify outbreaks of salmonellosis where pork or pork product consumption had been reported. The time-frame analysed was 1999-2009, during which time there were 245 outbreaks of salmonellosis reported. Nine outbreaks were identified where the cases involved had consumed pork or pork products in the week prior to illness. In the majority of outbreaks, other risk factors were also present. The results are presented in Table 10.

**Table 10: New Zealand outbreaks of salmonellosis with either epidemiological (suspected) links or laboratory confirmation linked with pork or pork product consumption 1999 – November 2009**

Year	Food implicated	Setting	Number ill	Confirmation
1999	“Cheerios” pork cocktail sausages	Butchers	6C, 2P	3
2002	Ham roll	Bakery product	2C	1
2003	Shanghai style meal including salty pork and deep fried pork chops	Restaurant	3 C, 2P	1,4
2003	Cooked pork	Hangi	36C, 28P	1,5
2005	Home kill pork	Home	7C, 2P	1
2006	Number of risk factors including ham	Home	1C, 1P	None
2006	Honey chicken/BBQ pork and rice	Restaurant	11C	1,4,5
2006	Various foods implicated including pork buns	Market	11C, 4O	1
2007	BBQ chicken and bacon pizza implicated. Cross contamination suspected	Restaurant	1C, 1P	1

C=confirmed P=probable O=other

1 epidemiological (suspected) links– cases had history of exposure to implicated source

2 epidemiological (suspected) links– case control or cohort study showed elevated risk for cases exposed to implicated source

3 laboratory – pathogen suspected to have caused illness identified in implicated source

4 environmental investigation (suspected) links – identified critical control point failures linked to implicated source

5 pathogen identified in food handler

In the 1999 outbreak involving cold pork cocktail sausages, the serotype identified was *S. Typhimurium* PT135. The internal meat of the sausage was found to be highly contaminated ( $5.4 \times 10^4$  MPN/100g). It appeared that this contaminated batch was then inadequately cooked (due to floating in the cooking waterbath). Temperature abuse may have encouraged further growth of surviving cells. The investigating Health Protection Officers made several recommendations to the butchery including weighing down the product so that the sausages were underwater during cooking (Macleod, 2000).

### 3.3.4 Case control studies and risk factors

Two case-control studies of salmonellosis in New Zealand have been conducted to date. One concerned *S. Typhimurium* DT160 (Thornley *et al.*, 2002, Thornley *et al.*, 2003); the other *S. Brandenburg* (NZFSA, 2002).

The study of *S. Typhimurium* DT160 was prompted by a marked increase in the number of DT160 human isolates which began in May 2001. 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 organism was also isolated from poultry during 2001.

In addition to telephone interviews of cases (119, median age 8 years and 57% female) and controls (235), environmental sampling was carried out on roof-collected rainwater supplies from the homes of cases, and egg brands consumed by cases. 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). However, this high risk activity was associated with only a few cases. Questions regarding consumption of a number of pork products were asked, but none were statistically associated with increased risk.

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. The results of the study have now been reported (Baker *et al.*, 2003; 2007). The study recruited 182 cases of salmonellosis, including 43 cases of *S. Brandenburg* infection, with the same number of matched controls.

Factors occurring in the three days prior to illness (or interview) that were significantly associated with an elevated risk of salmonellosis in general were:

- Contact with bird faeces (OR 4.87, 95% CI 1.71, 17.17);
- Contact with other sick people (OR 8.73, 95% CI 2.08, 62.91);
- Consumption of pork steak (OR 5.60, 95% CI 1.11, 72.80);
- Overseas travel (OR 9.97, 95% CI 1.72, 167.46);
- Touching of pet puppies. (OR 6.79, 95% CI 1.33, 73.03); and,
- Use of a kitchen bench, table, or sink for chopping (OR 5.47, 95% CI 1.47, 31.42).

For *S. Brandenburg* infection, two exposures were associated with a significant increase in disease risk:

- Occupational contact with live or dead sheep or lambs (OR 9.97, 95% CI 1.62, 196.29); and,
- Having a household member who had occupational contact with sheep or lamb (OR 4.28, 95% CI 1.23, 21.31).

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. Although a limited number of *Salmonella* isolates submitted to the Enteric Reference Laboratory have been typed as *S. Brandenburg* (Table 4), it is unknown whether pigs are a reservoir for this serotype.

### 3.4 Adverse Health Effects Overseas

The incidence of notified cases of salmonellosis in New Zealand is similar to rates in other developed countries, particularly Canada and Australia (see Appendix 2 Table 13). In contrast to New Zealand, in the EU the dominant serotype is *S. Enteritidis* (see Appendix 2 Table 14). In terms of outbreaks, salmonellosis appears to be a more significant cause of illness overseas (Appendix 2 Table 15) than in New Zealand, and several large outbreaks linked to pork have been reported (Appendix 2 Table 16).

### 3.5 Health Burden of Infection with *Salmonella* spp.

An estimate of the burden of foodborne disease 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 disease burden (after campylobacteriosis, norovirus infection, and perinatal listeriosis).

The burden of disease to the health system and society in general has also been considered, through a cost of illness estimate, based on the same incidence data (Cressey and Lake, 2008). This estimated the total cost for salmonellosis as \$4.8 million, with foodborne infections costing \$2.8 million.

A recent New Zealand study, using molecular sub-typing data and Bayesian techniques ('modified Hald model') estimated the attributable food source for human salmonellosis cases in New Zealand in 2003 (Mullner *et al.*, 2009). The authors urged caution in interpreting these results since molecular sub-typing data for pork were sparse and more biased than data for other food animal species (Mullner *et al.*, 2009). An estimated 60.2% (Bayesian credible interval 47-74%) of food sourced human salmonellosis was attributed to transmission by pork.

In the USA, foodborne salmonellosis cases are estimated to cost the economy \$US2.3 billion annually (1998 \$US) (Dickson *et al.*, 2002). Approximately 6-9% of human salmonellosis in USA is thought to be associated with the consumption of pork products (Frenzen *et al.*, 1999).

European estimates of the cost of salmonellosis are more in line with 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). The contribution of pork to human salmonellosis has been estimated by groups in Denmark and the Netherlands. A Dutch expert elicitation estimated that approximately 8% of human salmonellosis was due to

transmission via pork (Havelaar *et al.*, 2008), while estimates based on molecular subtyping estimated pigs to be the reservoir for approximately 20% of human salmonellosis cases in the Netherlands (2006) and 9-16% in Denmark (2005) (Panel on Biological Hazards, 2008).

### **3.6 Adverse Health Effects Summary**

The incidence of salmonellosis in New Zealand is comparable with the incidence in other developed countries. Human health surveillance data provides only limited evidence that pork is a vehicle for transmission of *Salmonella* in New Zealand. In only one of the outbreaks of salmonellosis where pork consumption was reported as a risk factor was *Salmonella* cultured from the implicated food; for the others the link with pork was based on epidemiological data, largely a history of exposure to a common source. Consumption of pork steak was identified as a statistically significant risk factor in the 2002 case control study (NZFSA, 2002), but not in the earlier study (Thornley *et al.*, 2002, Thornley *et al.*, 2003).

## 4 EVALUATION OF RISK

### 4.1 Existing Risk Assessments

Models to describe the behaviour of *Salmonella* at various stages during pork primary processing in New Zealand have been developed (Titus, 2007). These models are intended to contribute to a quantitative risk assessment for *Salmonella* in pork.

A summary of overseas risk assessments for *Salmonella* in pork is given in Appendix 2.

### 4.2 Estimate of Risk for New Zealand

#### 4.2.1 Risk associated with pork

The available data indicates a low prevalence of *Salmonella* contamination on domestically produced pork in New Zealand. The most recent data from two surveys did not find *Salmonella* on 230 domestic carcasses (Wong *et al.* (2009) swabbed 100 carcasses (area 100 cm<sup>2</sup>) (95% CI: 0-3.6), samples taken October 2004-May 2005; Titus (2007) swabbed 130 carcasses (area 225cm<sup>2</sup>) during primary processing (confidence intervals not stated), sampling dates not stated). Likewise, *Salmonella* was not detected in 231 retail samples of minced or diced pork sampled between August 2003 and May 2005 (95% CI: 0-1.6%) (Wong *et al.*, 2007).

A prevalence of 4/110 (3.6%) was found in a 2004-2005 survey of imported pork (all positive samples originated from Australia). The CI 95% was 1.0 - 9.0 (Wong *et al.*, 2009).

Each of these surveys involved small numbers of samples, so the results must be treated with caution. Comparison with data from overseas surveys suggests that the prevalence of contamination in New Zealand may be lower than overseas.

The rates of reported salmonellosis have fluctuated in the previous decade. Although rates (per 100,000 population) were generally higher between 1997 and 2002, since 2003 the rates have returned to below 40 per 100,000. Current rates of illness in New Zealand are similar to those in Australia and other developed countries.

The most common serotype of *Salmonella* isolated from human cases in New Zealand is *S.* Typhimurium, with *S.* Typhimurium DT160 being the most common type. A case control study in 2001 did not indicate an elevated risk from pork products for *S.* Typhimurium DT160 human infection (Thornley *et al.*, 2002, Thornley *et al.*, 2003). However, consumption of pork steak was identified as a statistically significant risk factor for salmonellosis in another case control study conducted in 2002 (Baker *et al.*, 2003; 2007).

The serotype data from isolates submitted by the pork industry to the ERL are in such small numbers that comparison with human cases is statistically unsound and was not considered in this Risk Profile.

Pork or pork products were included among risk factors reported for nine outbreaks between the years 1999 to 2009. Culture confirmation of *Salmonella* in the implicated food was possible only once (in this instance, pork cocktail sausages). For the other eight outbreaks, only epidemiological links with pork were reported. The number of outbreak cases represents

only a proportion (approximately 10%) of the total reported cases, and the epidemiology of sporadic reported cases may be different.

There are insufficient data available to assess the risk to New Zealanders from *Salmonella* in pork. Limited data suggest a low prevalence of contamination, and pork is rarely confirmed as a vehicle in reported salmonellosis outbreaks.

#### 4.2.2 Risk associated with other foods

Other food vehicles identified in outbreaks in New Zealand include eggs (surface contamination) and meat products (Wilson *et al.*, 2000). Transmission in poultry currently represents a minor component of salmonellosis etiology in New Zealand (Lake *et al.*, 2004a). A recent survey of eggs for *Salmonella* has been completed (Wilson, 2007) and this indicates an overall low prevalence of *Salmonella* contamination on eggs (9/514 = 1.8%, 95<sup>th</sup> percentile confidence interval 0.8-3.3%) in New Zealand, with no contamination within eggs being detected (95<sup>th</sup> percentile confidence interval 0.0-0.7%). Surveys of *Salmonella* on eggs conducted overseas have produced estimates of prevalence in the range 0.0-9.4% (Lake *et al.*, 2004b).

The potential for transmission of *S. Brandenburg* in sheep meat has been investigated, as part of a broad investigation into this pathogen (NZFSA, 2002). It was concluded that there was no evidence for foodborne transmission, and the majority of the human cases have been attributed to contact with infected farm animals. Takeaway foods were identified as an important risk factor in the *S. Typhimurium* DT160 case-control study (Thornley *et al.*, 2002, Thornley *et al.*, 2003). Two outbreaks related to umu functions have been reported; the foods involved were potato salad with egg mayonnaise which had been improperly stored (Callaghan and Simmons, 2001) and Palusami (umu cooked packs of taro in coconut milk wrapped in taro leaves) that had been privately imported from Samoa (Ng and Simmons, 2002).

A study of New Zealand outbreaks of salmonellosis between 1997 and 2006 (King and Lake, 2007) concluded that the etiology is hugely varied, although foodborne transmission is suggested for 40% of outbreaks, amongst which infected food handlers account for perhaps half.

### 4.3 Data Gaps

There are only limited data on the prevalence of *Salmonella* through the pork food chain, and none at all on concentrations of bacteria. Data on carriage of *Salmonella* by pigs prior to processing (and potential sources of infection e.g. pig feed) would seem to be the most pressing data gap, given the importance of this factor as an input into primary processing.

In relation to this data gap, a 12 month trial of testing of pork during primary processing for *Salmonella* was commenced in October 2009 under the testing programme of the National Microbiological Database (NMD).

Given the increasing proportion of New Zealand's pork supply that is imported, more detailed information on the prevalence and numbers of salmonellae in imported product is an important data gap.



While the ability of pigs to become colonised with salmonellae through consumption of contaminated feed has been demonstrated, there is insufficient information on the prevalence and concentration of *Salmonella* contamination of feed and the associated dose-response relationships for pigs to estimate the risk.

A report on options for a national *Salmonella* surveillance programme for New Zealand (Lake and Sexton, 2009) commented that for *Salmonella* isolates from non-human sources "...the proportion of isolates referred to ERL is unknown and likely to be only a small proportion of the isolates found by primary laboratories.". A more comprehensive system for sampling and testing for *Salmonella* in pigs and pork would assist with any risk assessment. The current trial of porcine *Salmonella* testing under the NMD and mandatory submission of any isolates to the ERL will improve this situation.

## 5 AVAILABILITY OF CONTROL MEASURES

### 5.1 Risk Management Strategy

In March 2009 NZFSA released their Salmonella Risk Management Strategy 2009-2012<sup>1</sup>. 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.

### 5.2 Current Risk Management Measures

#### 5.2.1 Relevant food controls

##### 5.2.1.1 *The Animal Products Act*

The [Animal Products Act 1999](#) reformed New Zealand law regulating the production and processing of animal material and animal products to:

- Manage associated risks; and
- Facilitate overseas market access.

The Animal Products Act requires all animal products traded and used to be "fit for intended purpose". This means they must meet New Zealand animal product standards. The New Zealand animal product standards are contained in Part 1 of the [Animal Product Regulations 2000](#). The Animal Products Act (except for Part 2) and the transitional Act commenced on 1 November 1999. Part 2 of the Animal Products Act commenced on 20 November 2000. Part 2 provides the requirements for risk management programmes.

The risk management system potentially applies anywhere in the value chain from production, through processing to the market. The risk management system comprises the following main types of controls:

- Risk management programmes;
- Regulated control schemes; and
- Controls relating to the export of animal material and animal products.

---

<sup>1</sup> <http://www.nzfsa.govt.nz/foodborne-illness/salmonella/strategy/salmonella-risk-management-strategy-2009-012.pdf>

A risk management programme is a documented programme to identify and manage biological, chemical and physical hazards. The programme is to be based on the principles of Hazard Analysis and Critical Control Point (HACCP): identifying the hazards, the systems of control, and demonstrating that the controls are effective. Risk management programmes are to be designed by individual businesses for the animal materials used, the processes performed and the product range produced.

Procedures for slaughtering and inspection are documented on the NZFSA website<sup>1</sup>. The NZFSA document has specific controls for pigs under part B, section 22.1 – 22.3. In summary, this stipulates that the area in which scalding and dehairing occurs must be physically separate from the area in which the carcasses are eviscerated and inspected. Scald water is stipulated to be above 59°C; however, water hotter than 64°C is recognised to cause skin surface damage.

Rapid chilling causes cold-shortening and toughening of meat, although pork is less likely than other meats to do this, so faster rates of chilling can be applied. Mesophiles such as *Salmonella* spp. are held in check by the drying surfaces and lowered water activity until the surface temperature falls below 7 to 8°C. Contact between carcasses encourages warm moist areas allowing *Salmonella* growth (ICMSF, 1998).

While there is some commercial hunting of feral pigs for sale on the New Zealand market, pork from feral pigs is outside the scope of this Risk Profile. Recreational hunters are permitted to kill wild pigs for their own consumption (including consumption by members of their hunting party, family or household). Trading of recreationally killed feral pork for human consumption is prohibited. Full details can be found in sections 68 and 69 of the Animal Products Act 1999. Further information on wild pigs and the policy on non-commercial wild food can be found at the NZFSA website<sup>2</sup>.

Processed pork products are required to be processed in premises operating under the *Animal Products Act 1999* or *Food Act 1981*. NZFSA and an industry working group are developing a code of practice for processed meats (expected publication April 2010).

#### 5.2.1.2 Import Health Standards

Pork imported from countries that have PRRS needs to be heat-treated (not necessarily cooked) or pH adjusted before arrival, or imported into a transitional facility in New Zealand where it is similarly heat-treated or pH adjusted prior to release onto the market. There are several Import Health Standards (IHS) which contain requirements for the importation of processed pork products<sup>3</sup>.

The Standards cover:

- Processed pork products from Canada, Denmark, Mexico, USA which are required to have been subjected to specified heat treatments or pH modification,

---

<sup>1</sup> <http://www.nzfsa.govt.nz/animalproducts/meat/meatman/is5/is5.pdf>

<sup>2</sup> <http://www.nzfsa.govt.nz/consumers/wild-foods-review/final-position-paper/index.htm>

<sup>3</sup> <http://www.maf.govt.nz/biosecurity/imports/animals/standards/index.htm>

- For pork products from Australia and Sweden, edible offals must be frozen to -18°C during transport,
- Unprocessed pork products from Canada, Denmark, the Mexican state of Sonora, USA which are required to receive specified heat treatments or pH modification after arrival, (due to PRRS),
- Specified pork products from Italy. These include prosciutto di Parma (Parma ham) or other ham that has undergone an equivalent 12 month curing process, and cooked pork products.

Up until 2001, pork was imported into New Zealand without sanitary controls for PRRS. A draft release assessment by MAF in 2001 using the results of a recent Australian feeding trial indicated the possibility of virus transmission by this route whereby the restrictions on pork from PRRS countries outlined above were put into place<sup>1</sup>. Because these measures were provisional, a full risk analysis has since been undertaken. The conclusion was that the likelihood of PRRS virus entering the country via pork and infecting a pig is extremely low. A move to allow high value consumer ready cut imports has been proposed, in the form of four draft IHS (covering Canada, USA, Mexico and ‘European Union’)<sup>2</sup>.

### 5.2.1.3 Regulation of antibiotic usage

Antibiotic use in animals in New Zealand is controlled by the NZFSA Agricultural Compounds and Veterinary Medicines (ACVM) Group. The use of antibiotics in animal feed and the potential promotion of antibiotic resistance in bacteria pathogenic to humans are subject to regular review<sup>3</sup>. In New Zealand there are two main uses for antibiotics in animal welfare:

- Therapeutic purposes; and,
- Prophylactic purposes.

Zinc bacitracin is used in the prevention and treatment of enteric pathogens in pigs and is usually administered in feed. It is active against Gram-positive bacteria and some Gram-negative bacteria but is not active against *Salmonella* spp. (Antibiotic Resistance Expert Panel, 1999; Wong and Gilbert, 2004).

A critical review of on-farm intervention options for *Salmonella* control reviewed eleven studies on antibiotic use and concluded that antimicrobials tend not to be useful and can be detrimental, due to selection of resistant serotypes (Friendship *et al.*, 2009).

### 5.2.1.4 NZ Pork activities in New Zealand

The New Zealand Pork Industry Board (NZ Pork) represents the New Zealand commercial pork processing industry. Financial activities of the board come from levies paid on all slaughtered pigs at premises operating registered risk management programmes. NZ Pork’s five key strategic areas (2009) are:

- Growing demand for New Zealand pork
- Increasing on-farm productivity
- Improving pork’s value

<sup>1</sup> [http://www.ipfsaph.org/cds\\_upload/kopool\\_data/WTOSPSNF\\_0/en\\_nnz1153.doc](http://www.ipfsaph.org/cds_upload/kopool_data/WTOSPSNF_0/en_nnz1153.doc)

<sup>2</sup> <http://www.biosecurity.govt.nz/media/12-11-07/pig-meat-ihs>

<sup>3</sup> <http://www.nzfsa.govt.nz/acvm/subject/antibiotic-resistance/2004arreport-final.htm>

- Securing industry sustainability
- Developing industry capability and profile.

NZ Pork encourages the following points as “best practice”. They are viewed as basic management strategies that reduce the incidence of most porcine diseases (Grant Boston, NZ Pork, personal communication 10 April 2008).

- Industry adoption of the “all-in, all-out” production system with age segregation, more common for farrowing and weaner rooms than grower and finishers, because of marketing of pigs in the final weeks. Most would be all-in/all-out by pen,
- Feed and water strategy. Feed is not fermented in New Zealand. Acidifiers are added to feeds 1-2 days post-weaning on most farms, primarily to manage *E. coli*. About 50% of feed in New Zealand is pelleted. Information from the New Zealand Feed Manufacturer’s Association states that pig feed going through the pelleting process is considered a critical control point for *Salmonella*. Manufacturers also place a priority on purchasing materials from approved suppliers who have a *Salmonella* testing programme in place. There are some meal pig diets that do not go through the pelleting process, in which case, manufacturers rely on inwards testing of raw materials or, purchasing from an approved supplier who has a *Salmonella* testing programme in place (Vanessa Wintle, NZFMA, personal communication, 21 April 2008). Most farmers cover their feed in closed silos to prevent pest access, although in outdoor housing, birds may have access to feeders within pens.
- Minimise number of suppliers of pigs to the farm, this strategy has been encouraged for many years. Most if not all registered commercial producers limit sources to one or two (e.g. gilts from one source, boars from another). Many take in semen but no live pigs to generate replacements. The smaller “backyard” sector may take in pigs from multiple and changing sources and is recognised as a biosecurity risk,
- Generally producers carry out a complete clean between batches, often at pen level. Intensity of cleaning/disinfection is generally very high for young pigs, decreasing with older animals. For example, farrowing rooms and weaner rooms would be water-blasted and /or disinfected, some using steam-cleaners. Most finisher pens would be pressure hosed but not water-blasted or disinfected,
- Policies for dealing with sick pigs,
- NZ Pork supports the appropriate use of antibiotics in pig production, under veterinary supervision, for the sole purpose of maintaining and enhancing pig health and welfare.

The New Zealand pork industry has previously utilised an industry developed total quality management “from farm to plate” programme known as PQIP (Pork Quality Improvement Process). During 2009, the Pork Processors Association and NZFSA developed a replacement for PQIP; the Processed Meat Code of Practice (CoP), which was published in March 2010.

The CoP provides guidance on good manufacturing practice, process control, and the application of HACCP principles for processed meats. The procedures given in the CoP are the accepted or industry agreed means of meeting the regulatory requirements Food Act 1981 or Animal Products Act 1999.

### 5.3 Options for Risk Management

There is a wealth of literature available to assess risk management interventions throughout the food chain, although it is widely recognised that prevention of animal infection is the most

effective option. Details of risk management programmes in Denmark, the Netherlands, and the USA are given in Appendix 3. A review of approaches to *Salmonella* control (Ojha and Kostrznska, 2007) examined enhancement of indigenous microflora (competitive exclusion, probiotics, prebiotics), targeting of pathogens (bacteriophages, bacteriocins, antibiotics, mineral supplementation, vaccination) as well as farm management practices.

The Danish Swine Salmonellosis Control Programme established in 1993 has been successful in controlling infection in pigs (Wegener *et al.*, 2003). This has reduced the estimated number of cases attributed to domestically produced pork from 22 to 2.6 cases per 100,000 population over the period 1993 to 2004. This was achieved through extensive serological testing of animals from breeder herds and slaughter pigs on the primary processing line. *Salmonella*-positive herds are logistically slaughtered (slaughtered at the end of the processing day) with application of extra hygiene measures. The programme is now focusing on improving the cost-effectiveness of the testing regime (Benschop *et al.*, 2008)

Organic acid washes are another option for management of bacteria on meat carcasses<sup>1</sup>.

#### **5.4 Commentary on Risk Management Options**

Due to their PRRS-positive status, pork from all countries except Australia must be imported cooked or imported raw and cooked at a transitional facility, thereby inactivating any *Salmonella* if it is present.

Should further investigation provide evidence for transmission of *Salmonella* in pork in New Zealand that requires risk management, a number of successful overseas measures, detailed in Appendix 3, can be considered as effective options. These options include systems for the reduction of *Salmonella* in herds and during processing.

---

<sup>1</sup> <http://www.meatupdate.csiro.au/new/Organic%20Acids.pdf>

## 6 REFERENCES

Advisory Committee on the Microbiological Safety of Food (1993). In: Report on *Salmonella* in eggs, ACMSF. London: HMSO.

Alford JA, Palumbo SA. (1969) Interaction of salt, pH and temperature on the growth and survival of salmonellae in ground pork. *Applied Microbiology*; 17: 528-532.

Allison, MJ, Dalton HP, Escobar MR, Martin CJ. (1969). *Salmonella choleraesuis* infections in man: a report of 19 cases and a critical literature review. *Southern Medical Journal*; 62: 593-596.

Angkitittrakul S, Chomvarin C, Chaita T, Kanistanon K, Waethewutajarn S. (2005) Epidemiology of antimicrobial resistance in *Salmonella* isolated from pork, chicken meat and humans in Thailand. *Southeast Asian Journal of Tropical Medicine and Public Health*; 36: 1510-1515.

Anonymous. (1996a) Extremely low infectious dose of *Salmonella* can cause disease in humans; adequacy of current QA is questioned. *Food Chemical News*; 38: 23-24.

Anonymous (1996b) Nationwide pork microbiological baseline data collection program: market hogs April 1995-March 1996. U.S. Department of Agriculture and Food Safety and Inspection Service, Microbiology Division, June 1996 Available at: <http://www.fsis.usda.gov/OPHS/baseline/markhog1.pdf>. Accessed: 30 November 2007.

Anonymous (1998) Nationwide sponge microbiological baseline data collection program: swine. June 1997-May 1998. U. S. Department of Agriculture and Food Safety and Inspection Service, Microbiology Division, Available at: [http://www.fsis.usda.gov/PDF/Baseline\\_Data\\_Swine.pdf](http://www.fsis.usda.gov/PDF/Baseline_Data_Swine.pdf). Accessed: 30 November 2007.

Antibiotic Resistance Expert Panel. (1999) Antibiotic resistance and in-feed use of antibiotics in New Zealand. Expert Panel Review, 31 July 1999. Available at: <http://www.nzfsa.govt.nz/acvm/publications/information-papers/antibiotic.pdf>. Accessed: 21 January 2010.

ANZFA. (2001) Raw commodity consumption figures. Canberra: ANZFA.

Bahnsen PB, Snyder C, Omran LM. (2006) *Salmonella enterica* in superficial cervical (prescapular) and ileocecal lymph nodes of slaughtered pigs. *Journal of Food Protection*; 69: 925-927.

Baker M, Thornley C, Lopez L, Garrett N, Nicol C. (2003) Quantifying the sources of *Salmonella* and *S. Brandenburg* infection in New Zealand. ESR Client Report FW0341. ESR: Kenepuru.

Baker MG, Thornley CN, Lopez LD, Garrett NK, Nicol CM. (2007) A recurring salmonellosis epidemic in New Zealand linked to contact with sheep. *Epidemiology and Infection*; 135: 76-83.

Beloel PA, Chauvin C, Proux K, Madec F, Fravalo P, Alioum A. (2004) Impact of the *Salmonella* status of market-age pigs and the pre-slaughter process on *Salmonella* caecal contamination at slaughter. *Veterinary Research*; 35: 513-530.

Benschop J, Stevenson MA, Dahl J, Morris RS, French NP. (2008) Temporal and longitudinal analysis of Danish Swine Salmonellosis Control Programme data: implications for surveillance. *Epidemiology and Infection*; 136: 1511-1520.

Berends BR, Van Kanpen F, Snijders JM, Mossel DA. (1997) Identification and quantification of risk factors regarding *Salmonella* spp. on pork carcasses. *International Journal of Food Microbiology*; 36: 199-206.

Berends BR, van Knapen F, Mossel DAA, Burt SA, Snijders JMA. (1998) *Salmonella* spp. on pork at cutting plants and at the retail level and the influence of particular risk factors. *International Journal of Food Microbiology*; 44: 207-217.

Bollaerts KE, Messens W, Delhalle L, Aerts M, van der Stede Y, Dewulf J, Quoilin S, Maes D, Mintiens K, Grijspeerdt K. (2009) Development of a quantitative microbial risk assessment for human salmonellosis through household consumption of fresh minced pork meat in Belgium. *Risk Analysis*; 29: 820-840.

Botteldoorn N, Heyndrickx, M, Rijpens N, Grijspeerdt K, Herman L. (2003) *Salmonella* on pig carcasses: positive pigs and cross contamination in the slaughterhouse. *Journal of Applied Microbiology*; 95: 891-903.

Boughton C, Leonard FC, Egan J, Kelly G, O'Mahony P, Markey BK, Griffin M. (2004) Prevalence and number of *Salmonella* in Irish retail pork sausages. *Journal of Food Protection*; 67: 1834-1839.

Bruun T, Sørensen G, Forshell LP, Jensen T *et al.* (2009) An outbreak of *Salmonella* Typhimurium infections in Denmark, Norway and Sweden, 2008. *Eurosurveillance*; 14: 1-6.

Buchholz U, Brodhun B, Brockmann SO, Dreweck CM, Prager R, Tschape H, Ammon A. (2005) An outbreak of *Salmonella* München in Germany associated with raw pork meat. *Journal of Food Protection*; 68: 273-276.

Callaghan M, Simmons G. (2001) Outbreak of *Salmonella* Typhimurium type 160 in Auckland linked to an umu function. *New Zealand Public Health Report*; 8: 44-45.

Childers AB, Keahey EE, Kotula AW. (1977) Reduction of *Salmonella* and faecal contamination of pork during swine slaughter. *Journal of American Veterinary Medicine Association*; 171: 1161-1164.

Chiou A, Chen L-H, Chen S-K. (1991) Foodborne illness in Taiwan, 1981-1989. *Food Australia*; 43: 70-71.

Cressey P, King N, Lake R. (2006) Food consumption data for risk assessments. ESR Client Report FW0602. Christchurch: ESR.



Cressey P, Lake R. (2007) Risk ranking: estimates of the burden of foodborne disease for New Zealand. Client Report FW0724. Available from: [http://www.nzfsa.govt.nz/science/research-projects/FW0724\\_DALY\\_estimates\\_August\\_2007\\_final.pdf](http://www.nzfsa.govt.nz/science/research-projects/FW0724_DALY_estimates_August_2007_final.pdf). Accessed: 25 April 2008.

Cressey P, Lake R. (2008) Risk ranking: Estimates of the cost of foodborne disease for New Zealand. A report for the New Zealand Food Safety Authority. ESR Client Report FW07102. Christchurch: ESR. Available from: [http://www.nzfsa.govt.nz/science/risk-ranking/FW07102\\_COI\\_estimates.pdf](http://www.nzfsa.govt.nz/science/risk-ranking/FW07102_COI_estimates.pdf). Accessed: 14 January 2010.

Davies PR, Bovee FGE, Funk JA, Morrow WEM, Jones FT, Deen J. (1998) Isolation of *Salmonella* serotypes from feces of pigs raised in multiple site production systems. Journal of the American Veterinary Medical Association; 212: 1925-1929.

Delpech V, McAnulty, Morgan K. (1998) A salmonellosis outbreak linked to internally contaminated pork meat. Australian and New Zealand Journal of Public Health; 22: 243-246.

Dickson JS, Hurd HS, Rostagno MH. (2002) *Salmonella* in the pork production chain. Factsheet – Pork Information Gateway. PIG 13-05-02: pp1-15. Available at: <http://www.pork.org/PorkScience/Documents/Pork%20Production%20Chain.pdf>

Doyle ME, Mazzotta AS. (2000) Review of studies on the thermal resistance of salmonellae. Journal of Food Protection; 63: 779-795.

Duffy EA, Belk KE, Sofos JN, Bellinger GR, Pape A, Smith GC. (2001) Extent of microbial contamination in United States pork retail products. Journal of Food Protection; 64: 172-178.

ECDC. (2009) Annual epidemiological report on communicable diseases in Europe 2009. Stockholm: European Centre for Disease Prevention and Control.

EFSA (2007a) Food safety aspects of different pig housing and husbandry systems. Scientific Opinion of the Panel on Biological Hazards. EFSA Journal (2007) 613: 1-20.

EFSA (2007b) Report of the Task Force on zoonoses data collection on a proposal for technical specifications of a baseline survey on the prevalence of *Salmonella* in breeding pigs. EFSA Journal; 99: 1-28.

EFSA (2009) Community Summary Report on Trends and Sources of Zoonoses and Zoonotic Agents in the European Union in 2007. EFSA Journal; 223: 1-312.

Escartín EF, Lozano JS, García OR. (2000) Quantitative survival of native *Salmonella* serovars during storage of frozen raw pork. International Journal of Food Microbiology; 54(1-2): 19-25.

ESR. (2004a) Notifiable and Other Diseases in New Zealand. Annual Report 2003. ESR Client Report FW0426. April 2004. Porirua: ESR.

ESR. (2004b) Annual Summary of Outbreaks in New Zealand. Annual Report 2003. ESR Client Report FW0419. Porirua: ESR.

ESR. (2005a) Notifiable and Other Diseases in New Zealand. Annual Report 2004. ESR Client Report FW0532. Porirua: ESR.

ESR. (2005b) Annual Summary of outbreaks in New Zealand, 2004. ESR Client Report FW0543. Porirua: ESR.

ESR. (2006a) Notifiable and Other Diseases in New Zealand. Annual Report 2005. ESR Client Report FW0621. Porirua: ESR.

ESR. (2006b) Annual Summary of outbreaks in New Zealand, 2005. ESR Client Report FW0623. Porirua: ESR.

ESR. (2007a) Notifiable and Other Diseases in New Zealand. Annual Report 2006. ESR Client Report FW0717. Porirua: ESR.

ESR. (2007b) Annual Summary of outbreaks in New Zealand, 2006. ESR Client Report. FW0714. Porirua: ESR.

ESR. (2008a) Notifiable and Other Diseases in New Zealand. Annual Report 2007. ESR Client Report FW08034. Porirua: ESR.

ESR. (2008b) Annual Summary of outbreaks in New Zealand, 2007. ESR Client Report. FW08035. Porirua: ESR.

ESR. (2009a) Notifiable and Other Diseases in New Zealand. Annual Surveillance Report 2008. ESR Client Report FW09074. Porirua: ESR.

ESR (2009b) Annual Summary of outbreaks in New Zealand, 2008. ESR Client Report. FW09036. Porirua: ESR.

Evans HS, Madden P, Douglas C, Adak GK, O'Brien SJ, Djuretic T, Wall PG, Stanwell-Smith R. (1998) General outbreaks of infectious intestinal disease in England and Wales: 1995 and 1996. *Communicable Disease and Public Health*; 1: 165-171.

FAO/WHO. (2002) Risk assessments of *Salmonella* in eggs and broiler chickens. Microbiological Risk Assessment Series No. 2. Geneva: World Health Organization.

FAS (United States Department of Agriculture Foreign Agricultural Service). (2001) New Zealand: New pork market? Available at: [http://www.fas.usda.gov/info/agexporter/1998/nz\\_pork.html](http://www.fas.usda.gov/info/agexporter/1998/nz_pork.html)

Fedoraka-Cray PJ, Whipp SC, Isaacson RE, Nord N, Lager K. (1994) Transmission of *Salmonella* Typhimurium to swine. *Veterinary Microbiology*; 41: 333-344.

Fedoraka-Cray PJ, Kelley LC, Stabel TJ, Gray JT, Laufer JA. (1995) Alternate routes of invasion may affect pathogenesis of *Salmonella typhimurium* in swine. *Infection and Immunity*; 63: 2658-2664.

Frenzen PD, Buzby JC, Roberts T. (1999) An updated estimate of the economic costs of human illness due to foodborne *Salmonella* in the United States. In: Proceedings of the 3<sup>rd</sup> International Symposium on the Epidemiology and Control of *Salmonella* in Pork, Washington, DC.: 215-218.

Friendship RM, Mouchili A, McEwen S, Rajić A. (2009) Critical review of on-farm intervention strategies against *Salmonella*. Available at: <http://www.bpex.org/downloads/298614/292327/Critical%20review%20of%20onfarm%20intervention%20strategies%20against%20Salmonella.pdf>. Accessed: 20 January 2010.

Gessner BD, Beller M. (1994) Protective effect of conventional cooking versus use of microwave ovens in an outbreak of salmonellosis. *American Journal of Epidemiology*; 139: 903-909.

Giovannini A, Prencipe V, Conte A, Marino L, Petrini A, Pomilio F, Rizzi V, Migliorati G. (2004) Quantitative risk assessment of *Salmonella* spp. infection for the consumer of pork products in an Italian region. *Food Control*; 15: 139-144.

Goldbach SG, Alban L. (2006) A cost-benefit analysis of *Salmonella*-control strategies in Danish pork production. *Preventive Veterinary Medicine*; 77: 1-14.

Gorden J, Small PLC. (1993) Acid resistance in enteric bacteria. *Infection and Immunity*; 61: 364-367.

Grau FH. (1983) Growth of *Escherichia coli* and *Salmonella typhimurium* on beef tissue at 25°C. *Journal of Food Science*; 48: 1700-1704.

Hald T, Andersen JS. (2001) Trends and seasonal variations in the occurrence of *Salmonella* in pigs, pork and humans in Denmark, 1995-2000. *Berliner und Münchener tierärztliche Wochenschrift*; 114:346-349.

Hald T, Wingstrand A, Swanenburg M, von Altrock A, Thorberg BM. (2003) The occurrence and epidemiology of *Salmonella* in European pig slaughterhouses. *Epidemiology and Infection*; 131: 1187-1203.

Hannu T, Inman R, Granfors K, Leirisalo-Repo M. (2006) Reactive arthritis or post-infectious arthritis? *Best Practice & Research Clinical Rheumatology*; 20(3): 49-433.

Haraga A, Ohlson MB, Miller SI. (2008) Salmonellae interplay with host cells. *Nature Reviews*; 6: 53-66

Havelaar AH, Vargas Galindo A, Kurowicka D, Cooke RM. (2008) Attribution of foodborne pathogens using structured expert elicitation. *Foodborne Pathogens and Disease*; 5(5): 649-659.

Hurd HS, Gailey JK, McKean JD, Rostagno MH. (2001a) Experimental rapid infection in market swine following exposure to a *Salmonella* contaminated environment. *Berliner und Münchener tierärztliche Wochenschrift*; 114:382-384.

Hurd HS, Gailey JK, Rostagno MH. (2001b) Rapid infection in market swine occurs following exposure to a *Salmonella* contaminated environment. *American Journal of Veterinary Research*; 62: 114-119.

Hurd HS, McKean JD, Griffith RW, Wesley IV, Rostagno MH. (2002) *Salmonella enterica* infections in market swine with and without transport and holding. *Applied and Environmental Microbiology*; 68: 2376-2381.

ICMSF. (1980) *Microbial ecology of foods. Volume 2. Food Commodities.* New York: Academic Press.

ICMSF. (1998) *Micro-organisms in foods 6. Microbial ecology of food commodities.* International Commission on Microbiological Specifications for Foods (ICMSF). London: Blackie Academic and Professional.

Ingham SC, Fanslau MA, Burnham GM, Ingham BH, Norback JP, Schaffner DW. (2007) Predicting pathogen growth during short-term temperature abuse of raw pork, beef and poultry products: use of an isothermal-based predictive tool. *Journal of Food Protection*; 70: 1445-1456.

Jansen A, Frank C, Prager R, Oppermann H, Stark K. (2005) Nation-wide outbreak of *Salmonella* Gibe in Germany, 2004. *Zeitschrift für Gastroenterologie*; 43: 707-713 [Article in German].

Jay S, Grau FH, Smith K, Lightfoot D, Murray C, Davey GR. (1997) *Salmonella*. In: Hocking AD, Arnold G, Jenson I, Newton K, Sutherland P (eds). *Foodborne Microorganisms of Public Health Significance. Fifth Edition.* Sydney: Australian Institute of Food Science and Technology.

Jay LS, Davos D, Dundas M, Frankish E, Lightfoot D. (2003) *Salmonella*. In: Hocking AD (eds). *Foodborne Microorganisms of Public Health Significance. Sixth edition.* Waterloo, NSW: Australian Institute of Food Science and Technology Incorporated, NSW Branch, Food Biology Group.

Juneja VK, Eblen BS. (2000) Heat inactivation of *Salmonella* Typhimurium DT 104 in beef as affected by fat content. *Letters in Applied Microbiology*; 30: 461-467.

Kemmeren JM, Manges J-MJ, van Duynhoven YTHP, Havelaar AH. (2006) Priority setting of foodborne pathogens. Disease burden and cost of selected enteric pathogens. RIVM Report 330080001/2006. Bilthoven, the Netherlands: RIVM.

King N, Lake R. (2007) Review of notified salmonellosis outbreak data as a source of information for attribution. ESR Client Report FW06114. Christchurch: ESR.

Lake RJ, Sexton K. (2009) Options for a national *Salmonella* surveillance programme for New Zealand. ESR Client Report FW09044. Christchurch: ESR.

Lake R, Hudson A, Cressey P, Wong TL, Gilbert SE. (2004a) Risk profile: *Salmonella* (non-typhoidal) in poultry (whole and pieces). ESR Client Report FW0425, Christchurch, ESR.

Lake R, Hudson A, Cressey P, Gilbert SE. (2004b) Risk profile: *Salmonella* (non-typhoidal) in and on eggs. ESR Client Report FW0420, Christchurch, ESR.

Lammerding AM, Garcia MM, Mann ED, Robinson Y, Dourward WJ, Truscott RB, Tittiger F. (1988) Prevalence of *Salmonella* and thermophilic *Campylobacter* in fresh pork, veal and poultry in Canada. *Journal of Food Protection*; 51: 47-52.

Lee W-C, Lee M-J, Kim J-S, Park S-Y. (2001) Foodborne illness in Korea and Japan studied retrospectively. *Journal of Food Protection*; 64: 899-902.

Letellier A, Messier S, Quessy S. (1999) Prevalence of *Salmonella* spp. and *Yersinia enterocolitica* in finishing swine at Canadian abattoirs. *Journal of Food Protection*; 62: 22-25.

Lin J, Lee IS, Frey J, Slonczewski JL, Foster JW. (1995) Comparative analysis of extreme acid survival in *Salmonella typhimurium*, *Shigella flexneri*, and *Escherichia coli*. *Journal of Bacteriology*; 177: 4097-4104.

Lindqvist R, Andersson Y, de Jong B, Norberg P. (2000) A summary of foodborne disease incidents in Sweden, 1992 to 1997. *Journal of Food Protection*; 63: 1315-1320.

Little CL, Richardson JF, Owen R, Ward LR, de Pinna E, Threlfall J. (2006) Report on the two year monitoring study of pathogens in raw meats, 2003-2005. Available to subscribers of the Local Authorities Co-ordinators of Regulatory Services. LACORS/HPA.

Loewenstein MS. (1975) An outbreak of salmonellosis propagated by person-to-person transmission on an Indian reservation. *American Journal of Epidemiology*; 102: 257-262.

Lo Fo Wong DMA, Hald T. (2000) *Salmonella* in Pork (Salinpork). Preharvest and harvest control options based on epidemiologic, diagnostic and economic research, Final Report. Contract no. FAIR1 CT95-0400. Copenhagen, Denmark. [www.vet.dtu.dk/Admin/Public/DWSDownload.aspx?File=Files%2FFiler%2FZoonosecentret%2FPublikationer%2FSalinp](http://www.vet.dtu.dk/Admin/Public/DWSDownload.aspx?File=Files%2FFiler%2FZoonosecentret%2FPublikationer%2FSalinp)

Loynachan AT, Nugent JM, Erdman MM, Harris DL. (2004) Acute infection of swine by various *Salmonella* serovars. *Journal of Food Protection*; 67: 1484-1488.

Loynachan AT, Harris DL. (2005) Dose determination for acute *Salmonella* infection in pigs. *Applied and Environmental Microbiology*; 71: 2753-2755.

Luiten LS, Marchello JA, Dryden FD. (1982) Growth of *Salmonella typhimurium* and mesophilic organisms on beef steaks as influenced by type of packaging. *Journal of Food Protection*; 45: 263-267.

Luzzi I, Galetta P, Massari M, Rizzo C, Dionisi AM, Filetici E, Cawthorne A, Tozzi A, Argentieri M, Bilei S, Busani L, Gnesivo C, Pendenza A, Piccoli A, Napoli P, Loffredo L, Trinito MO, Santarelli E, Ciofi degli Atti ML. (2007) An Easter outbreak of *Salmonella* Typhimurium DT104A associated with traditional pork salami in Italy. *Eurosurveillance*; 12: E11-12.

Mackey BM, Roberts TA, Mansfield J, Farkas G. (1980) Growth of *Salmonella* on chilled meat. *Journal of Hygiene (London)*; 85: 115-124.

Macleod K. (2000) A butchery salmonellosis outbreak. *New Zealand Journal of Environmental Health*; 1: 27-30.

Maguire HC, Codd AA, Mackay VE, Rowe B, Mitchell E. (1993) A large outbreak of human salmonellosis traced to a local pig farm. *Epidemiology and Infection*; 110: 239-246.

Mann JE, Smith L, Brashears MM. (2004) Validation of time and temperature values as critical limits for *Salmonella* and background flora growth during the production of fresh ground and boneless pork products. *Journal of Food Protection*; 67: 1389-1393.

McDowell SWJ, Porter R, Madden R, Cooper B, Neill SD. (2007) *Salmonella* in slaughter pigs in Northern Ireland: Prevalence and use of statistical modeling to investigate sample and abattoir effects. *International Journal of Food Microbiology*; 118: 116-125.

Miller GY, Liu X, McNamara PE, Barber DA. (2005) Influence of *Salmonella* in pigs preharvest and during pork processing on human health costs and risks from pork. *Journal of Food Protection*; 68: 1788-1798.

Mølbak K, Baggensen DL, Aarestrup FM, Ebbesen JM, Engberg J, Frydendahl K, Gerner-Smidt P, Petersen AM, Wegener HC. (2000) An outbreak of multidrug-resistant, quinolone-resistant *Salmonella* enterica serotype typhimurium DT104. *New England Journal of Medicine*; 341:1420-1425.

Morgan IR, Krautil FL, Craven JA. (1987a) Bacterial populations on dressed pig carcasses. *Epidemiology and Infection*; 98: 15-24.

Mossong J, Marques P, Ragimbeau C, Huberty-Krau P, Losch S, Meyer G, Moris G, Strottner C, Rabsch W, Schneider F. (2007) Outbreaks of monophasic *Salmonella enterica* serovar 4,[5],12:i:- in Luxembourg, 2006. *Eurosurveillance*; 12(6): E11-12.

Mullner P, Jones G, Noble A, Spencer SEF, Hathaway S, French NP. (2009) Source attribution of food-borne zoonoses in New Zealand: A modified Hald model. *Risk Analysis*; 29(7): 970-984.

Murase T, Yamada M, Muto T, Matsushima A, Yamai S. (2000) Fecal excretion of *Salmonella enterica* Serovar Typhimurium following a food-borne outbreak. *Journal of Clinical Microbiology*; 38: 3495-3497.

Narain JP, Lofgren JP. (1989) Epidemic of restaurant-associated illness due to *Salmonella* Newport. *The Southern Medical Journal*; 7:837-840.

Naugle AL, Barlow KE, Eblen DR, Teter V, Umholtz R. (2006) US Food Safety and Inspection Service testing for *Salmonella* in selected raw meat and poultry products in the United States, 1998 through 2003: analysis of set results. *Journal of Food Protection*; 69: 2607-2614.

New Zealand Pork. (2008) Annual report 2008. Accessed at: <http://www.nzpork.co.nz/LinkClick.aspx?fileticket=sVnMTbgJtW8%3d&tabid=137&mid=684>. Accessed on: 24 December 2009.



New Zealand Pork. (2009) Pork Outlook. January 2009. Accessed at: <http://www.nzpork.co.nz/LinkClick.aspx?fileticket=NpskB95gF0s%3d&tabid=128&mid=648>  
Accessed on: 23 December 2009.

New Zealand Pork. (2004) Pork production in New Zealand. [http://www.pork.co.nz/nzpork/technical\\_papers/introduction.asp](http://www.pork.co.nz/nzpork/technical_papers/introduction.asp). Accessed on 18 February 2008. Wellington: New Zealand Pork.

New Zealand Pork. (2006) Annual Report and Pork production in New Zealand. [http://www.pork.co.nz/nzpork/technical\\_papers/introduction.asp](http://www.pork.co.nz/nzpork/technical_papers/introduction.asp). Accessed on 18 February 2008. Wellington: New Zealand Pork.

New Zealand Pork. (2007) Annual Report and Pork production in New Zealand. [http://www.pork.co.nz/nzpork/technical\\_papers/introduction.asp](http://www.pork.co.nz/nzpork/technical_papers/introduction.asp). Accessed on 28 March 2008. Wellington: New Zealand Pork.

Ng L, Simmons G. (2002) An outbreak of salmonellosis linked to consumption of umu food from the Pacific Islands, Auckland, February 2002. Auckland: Public Health Protection.

Noël H, Dominguez M, Weill FX, Brisabois A, Duchazeaubeneix C, Kerouanton A, Delmas G, Pihier N, Couturier E. (2006) Outbreak of *Salmonella Enterica* serotype Manhattan infection associated with meat products, France, 2005. *Eurosurveillance*; 11: 270-273.

NZFSA. (2002) Project Report: Quantitative risk assessment of *Salmonella* in sheep meat produced in New Zealand. Available at: <http://www.nzfsa.govt.nz/science-technology/research-reports/salmonella-in-sheep.pdf>

NZFSA. (2004) Industry Standard 6 / Industry Agreed Standard 6 (IS6/IAS6) Processing of Edible Product. Amendment 5, July 2004.

NZFSA. (2006) Industry Standard 3 / Industry Agreed Standard 3 (IS3/IAS3) Hygiene and Sanitation. Amendment 4, April 2006.

Ojha S, Kostrzynska M. (2007) Review: Approaches for reducing *Salmonella* in pork production. *Journal of Food Protection*; 70: 2676-2694.

Olsen SJ, MacKinnon LC, Goulding JS, Bean NH, Slutsker L. (2000) Surveillance for foodborne disease outbreaks-United States, 1993-1997. *Morbidity and Mortality Weekly Reports*; 49: 1-51.

Oosterom J, Dekker R, de Wilde GJ, van Kempen-deTroye F, Engels GB. (1985) Prevalence of *Campylobacter jejuni* and *Salmonella* during pig slaughtering. *Veterinary quarterly*; 7: 31-34.

Oscar, T. (2004) Dose-response model for 13 strains of *Salmonella*. *Risk Analysis*; 24: 41-49.

OzFoodNet (2008) Monitoring the incidence and causes of diseases potentially transmitted by food in Australia: Annual Report of the OzFoodNet network, 2007. *Communicable Disease Intelligence*; 32(4): 400-424.

Palá TR, Sevilla A. (2004) Microbial contamination of carcasses, meat, and equipment from an Iberian pork cutting plant. *Journal of Food Protection*; 67:1624-1629.

Panel on Biological Hazards. (2008) A quantitative microbiological risk assessment on *Salmonella* in meat: Source attribution for human salmonellosis from meat. *EFSA Journal*; 625: 1-32.

Paulin SM, Watson PR, Benmore AR, Stevens MP, Jones PW, Villarreal-Ramos B, Wallis TS. (2002) Analysis of *Salmonella enterica* serotype-host specificity in calves: avirulence of *S. enterica* serotype gallinarum correlates with bacterial dissemination from mesenteric lymph nodes and persistence in vivo. *Infection and Immunity*; 70:6788-6797.

Pether JVS, Scott RJD. (1982) *Salmonella* carriers: are they dangerous? A study to identify finger contamination with *Salmonella* by convalescent carriers. *Journal of Infection*; 5: 81-88.

Rajkowski KT, Niebuhr SE, Dickson J. (2006) Effect of gamma or beta radiation on *Salmonella* DT104 in ground pork. *Journal of Food Protection*; 69: 1430-1433.

Russell DG, Parcel WR, Wilson NC *et al.* (1999) NZ Food: NZ People. Key results of the 1997 National Nutrition Survey. Wellington: Ministry of Health.

Saide-Albornoz JJ, Knipe LC, Murano EA, Beran GW. (1995) Contamination of pork carcasses during slaughter, fabrication, and chilled storage. *Journal of Food Protection*; 58: 993-997.

Sandberg M, Hopp P, Jarp J, Skjerve E. (2002) An evaluation of the Norwegian *Salmonella* surveillance and control program in live pig and pork. *International Journal of Food Microbiology*; 72: 1-11.

Silas JC, Carpenter JA, Reagan JO. (1984) Update: prevalence of *Salmonella* in pork sausage. *Journal of Animal Science*; 59: 122-124.

Silliker JH, Wolfe SK. (1980) Microbiological safety considerations in controlled-atmosphere storage of meats. *Food Technology*; 74: 59-63.

Simone E, Goosen M, Notermans SHW, Borgdorff MW. (1997) Investigations of foodborne disease by food inspection services in the Netherlands, 1991 to 1994. *Journal of Food Protection*; 60: 442-446.

Swanenburg M, van der Wolf PJ, Urlings HAP, Snijders JMA, van Kanpen F. (2001a) *Salmonella* in slaughter pigs: the effect of logistic slaughter procedures of pigs on the prevalence of *Salmonella* in pork. *International Journal of Food Microbiology*; 70: 231-242.

Swanenburg M, Berends BR, Urlings HA, Snijders JM, van Knapen F. (2001b) Epidemiological investigations into the sources of *Salmonella* contamination of pork. *Berliner und Münchener tierärztliche Wochenschrift*; 114: 356-359.

Tamplin ML, Feder I, Palumbo SA, Oser A, Yoder L, Luchansky JB. (2001) *Salmonella* spp. and *Escherichia coli* biotype I on swine carcasses processed under the Hazard Analysis and



Critical Control point-based inspection models project. *Journal of Food Protection*; 64: 1305-1308.

Thorberg BM, Engvall A. (2001) Incidence of *Salmonella* in five Swedish slaughterhouses. *Journal of Food Protection*; 64: 542-545.

Thornley C, Simmons G, Nicol C, Callaghan M, Baker M, Gilmore K, Garrett N, McLean M, Brieseman M. (2002) Investigation of an outbreak of *Salmonella* Typhimurium DT160. ESR Client Report FW0209. Porirua: ESR.

Thornley C, , Simmons G, Callaghan M, Nicol C, Baker M, Gilmore K, Garrett N, (2003) First Incursion of *Salmonella enterica* Serotype Typhimurium DT160 into New Zealand. *Emerging Infectious Diseases*; 9: 493-495

Titus SM. (2007) A novel model developed for Quantitative Microbial Risk Assessment in the pork food chain. Massey University PhD thesis. Available at: <http://epicentre.massey.ac.nz/Default.aspx?tabid=132&pid=0>

Todd ECD. (1992) Foodborne disease in Canada-a 10-year summary from 1975 to 1984. *Journal of Food Protection*; 55: 123-132.

Tokumaru M, Konuma H, Umesako M, Konno S, Shinagawa K. (1991) Rates of detection of *Salmonella* and *Campylobacter* in meats in response to the sample size and the infection level of each species. *International Journal of Food Microbiology*; 13: 41-46.

Torpdahl M, Sørensen G, Ethelberg S, Sandø G, Gammelgård K, Porsbo LJ. (2006) A regional outbreak of *S. Typhimurium* in Denmark and identification of the source using MLVA typing. *Eurosurveillance*; 11(5): 134-136.

Troller JA, Christian JHB. (1978) *Water Activity and Food*. New York: Academic Press.

Unicomb LE, Simmons G, Merritt T, Gregory J, Nicol C, Jelfs P, Kirk M, Tan A, Thomson R, Adamopoulos J, Little CL, Currie A, Dalton CB. (2005) Sesame seed products contaminated with *Salmonella*: three outbreaks associated with tahini. *Epidemiology and Infection*; 133(6): 1065-1072.

USDA/FSIS (1996) Pathogen reduction; hazard analysis and critical control point (HACCP) systems; final rule. <http://www.fsis.usda.gov/OPPDE/rdad/FRPubs/93-016F.pdf> Accessed 4.12.07

USDA/FSIS (1999) Generic HACCP Model for Pork Slaughter. United States Department of Agriculture, Food Safety and Inspection Service. September 1999, HACCP-14, Washington DC, USA.

USDA (2005) A focus on *Salmonella*. Food Safety Research Information Office Available at: [http://fsrio.nal.usda.gov/document\\_fsheetsheet.php?product\\_id=58](http://fsrio.nal.usda.gov/document_fsheetsheet.php?product_id=58). Accessed: 30 April 2008.

USDA/FSIS (2008) HACCP for raw processes. 28/06/08. Available at: [http://www.fsis.usda.gov/PDF/FSRE-HACCP-Raw\\_haccp1.pdf](http://www.fsis.usda.gov/PDF/FSRE-HACCP-Raw_haccp1.pdf). Accessed: 14 October 2009.

van der Gaag MA (2004) Epidemiological and economic simulation of *Salmonella* in the pork supply chain. PhD thesis ISBN 90-5808-973-8. Available at: <http://library.wur.nl/wda/>

van Netten P, Mossel D, Huis in't Veld. J. (1995) Lactic acid decontamination of fresh pork carcasses: a pilot plant study. *International Journal of Food Microbiology*; 25:1-9.

Vieiro-Pinto M, Temudo P, Martins C. (2005) Occurrence of *Salmonella* in the ileum, ileocolic lymph nodes, tonsils, mandibular lymph nodes and carcasses of pigs slaughtered for consumption. *Journal of Veterinary Medicine*; 52: 476-481.

Warriss PD, Brown SN, Edwards JE, Anil MH, Fordham DP. (1992) Time in lairage needed by pigs to recover from the stress of transport. *Veterinary Record*; 131:194-196.

Waterman SR, Small PLC (1998) Acid-sensitive pathogens are protected from killing under extremely acidic conditions of pH 2.5 when they are inoculated onto certain solid food sources. *Applied and Environmental Microbiology*; 64: 3882-3886.

Wegener HC, Hald T, LoFo Wong D, Madsen M, Korsgaard H, Bager F, Gerner-Smidt P, Mølbak K. (2003) *Salmonella* control programs in Denmark. *Emerging Infectious Diseases*; 9: 774-780

Williman J, Lim E, Pirie R, Cressey P, Lake R. (2009) Annual report concerning foodborne disease in New Zealand 2008. ESR Client Report FW09062. Christchurch: ESR.

Wilson MW, Lake RJ, Kieft CJ. (2000) Background for risk assessment of imported poultry products. FW0078. A report for the Ministry of Health. Auckland: ESR.

Wilson MW. (2007) Survey of retail eggs for *Salmonella*. ESR Client Report FW0779. Auckland: ESR.

Wingstrand A, Nielsen J, Heisel D, Dahl J. (1996) Experimental subclinical *Salmonella* infection in pigs, in 14<sup>th</sup> IPVS Congress, Bologna, Italy, p. 175.

Wong TL, Gilbert SE. (2004) Effect on *Salmonella* in poultry meats of removal of antibiotics from poultry feed in New Zealand. Client Report FW0430. Christchurch: ESR.

Wong TL, Nicol C, Cook R, McDiarmid S. (2007) *Salmonella* in uncooked retail meats in New Zealand. *Journal of Food Protection*; 70: 1360-1365.

Wong TL, MacDiarmid S, Cook R. (2009) *Salmonella*, *Escherichia coli* O157:H7 and *E. coli* biotype 1 in a pilot survey of imported and New Zealand pig meats. *Food Microbiology*; 26: 177-182.

Yeh KS, Chen SP, Lin JH. (2005) One-year nationwide pork carcass microbiological baseline data survey in Taiwan. *Journal of Food Protection*; 68: 458-461.

Zaidi MB, McDermott PF, Fedorka-Cray P, Leon V, Canche C, Hubert SK, Abbott J, Leon M, Zhao S, Headrick M, Tollefson L. (2006) Non-typhoidal *Salmonella* from human clinical

cases, asymptomatic children, and raw retail meats in Yucatan, Mexico. *Clinical Infectious Diseases*; 42: 21-28.

Zhao C, Ge B, De Villena J, Sudler R, Yeh E, Zhao S, White DG, Wagner D, Meng J. (2001) Prevalence of *Campylobacter* spp., *Escherichia coli*, and *Salmonella* serovars in retail chicken, turkey, pork, and beef from the greater Washington area. *Applied and Environmental Microbiology*; 67: 5431-5436.

## APPENDIX 1: HAZARD AND FOOD

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 and are intended for use by regional public health units. The datasheets will be updated from time to time, and placed on this website: <http://www.nzfsa.govt.nz/science/data-sheets/index.htm>. Please be aware that new information on the subject may have arisen since this document was finalised.

### 1.1 *Salmonella*

#### 1.1.1 Growth and survival

##### **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.

Studies of multiple *Salmonella* strains in broth and minced pork medium (in competition with natural flora) by Alford and Palumbo (1969) found that in minced pork, five *Salmonella* serotypes grew well at 10°C. Even where the salmonellae accounted for <10% of total flora, they were able to grow competitively at 10°C.

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 or other additives.

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<sub>2</sub> (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.

##### **Survival:**

*Salmonella* are known to survive well in foods and on surfaces.

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 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.

Escartín *et al.*, (2000) carried out quantitative survival studies of *Salmonella* on frozen raw pork and found that reductions in bacterial numbers increased with storage time from 7-11 to 1.6 MPN/g over 22 weeks; from 1,500-9,000 to 2.5 MPN/g over 42 weeks and from 2,000-20,000 to 20 MPN/g over 78 weeks storage.

pH: *Salmonella* appear to be significantly less tolerant of low pH (pH 2.5; hydrochloric acid) than *Shigella* spp. 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 subsequent heat resistance of these organisms.

### 1.1.2 Inactivation

Note that in microbiological terms “D” refers to a 90% (a decimal or 1 log<sub>10</sub> 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: at 60°C usually 2-6 min; at 70°C usually 1 minutes 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* 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 1050 minutes at 70°C, 222 minutes at 80°C and 78 minutes at 90°C.

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

Decreasing temperature increases the inhibitory effects of pH and NaCl (Alfred and Palumbo, 1969). In broth, at 10°C, growth of 22/23 strains were inhibited by pH 5 and 2% NaCl. 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: The effect of gamma or beta radiation on *Salmonella* DT104 in ground pork has been researched (Rajkowski *et al.*, 2006). A mixture of six strains were used to inoculate three ground pork products (of varying fat content). The amount of beta radiation to achieve a 90% reduction was around 0.43 kGy regardless of fat content.

Disinfectants: Sanitisers appear to have some effectiveness against *Salmonella* during pork primary processing (Childers *et al.*, 1977). For example, the wearing of plastic gloves and

disinfecting the knife in 82°C water before each carcass reduced contamination of the carcass by 50%. Dipping the knife into 500 ppm chlorine solution (pH 6) or in 25 ppm iodine solution reduced contamination by 75%.

## 1.2 The Food Supply: Pork and Pork Products

### 1.2.1 Pig production and processing with respect to *Salmonella*

Most *Salmonella* infections in pigs are asymptomatic. Carriage occurs primarily in the gastrointestinal tract, although mesenteric and hepatic lymph nodes and sometimes the gallbladder can contain *Salmonella* even when the organism is not detected in the intestinal contents (ICMSF, 1998). The organism has also been isolated from the pharynx, tongue, tonsils, ileum, liver, and stomach contents (Hurd *et al.*, 2001a, 2001b; Swanenburg *et al.*, 2001b; Fedorka-Cray *et al.*, 1994). The Swanenburg study found predominantly *S. Infantis* (100%) on carcasses, *S. Typhimurium* in livers (33%) and tongues (62%), *S. Typhimurium* (30%) and *S. Brandenburg* (26%) in rectal contents, others (47%) in mesenteric lymph nodes and others (42%) in tonsils.

Exceptions to asymptomatic infections are *S. Cholerae-suis* (a porcine-host restricted serotype that causes severe systemic illness, but rarely infects humans) and some strains of *S. Typhimurium* that typically cause enteric disease post-weaning.

Traditionally, the faecal-oral route of *Salmonella* transmission in pigs is recognised as the major route of infection, with invasion through the intestinal wall (Fedorka-Cray *et al.*, 1995). Another transmission route of significance is by aerosols infecting tonsils and lungs. A dose determination for intranasal or contaminated environment transmission found that a minimum of 10<sup>3</sup> salmonellae per animal were required to infect both alimentary and non-alimentary tissues (Loynachan *et al.*, 2004; Loynachan and Harris, 2005). This is relevant to the transmission of *Salmonella* from subclinically infected pigs to naïve animals during transportation and lairage (holding before slaughter), a period in which higher animal stress promotes shedding. Such events immediately prior to slaughter have been shown to correlate with an increased rate of *Salmonella* isolation from porcine carcasses and pork products.

#### *Preharvest*

The production of pigs on commercial farms in New Zealand is based on two main areas;

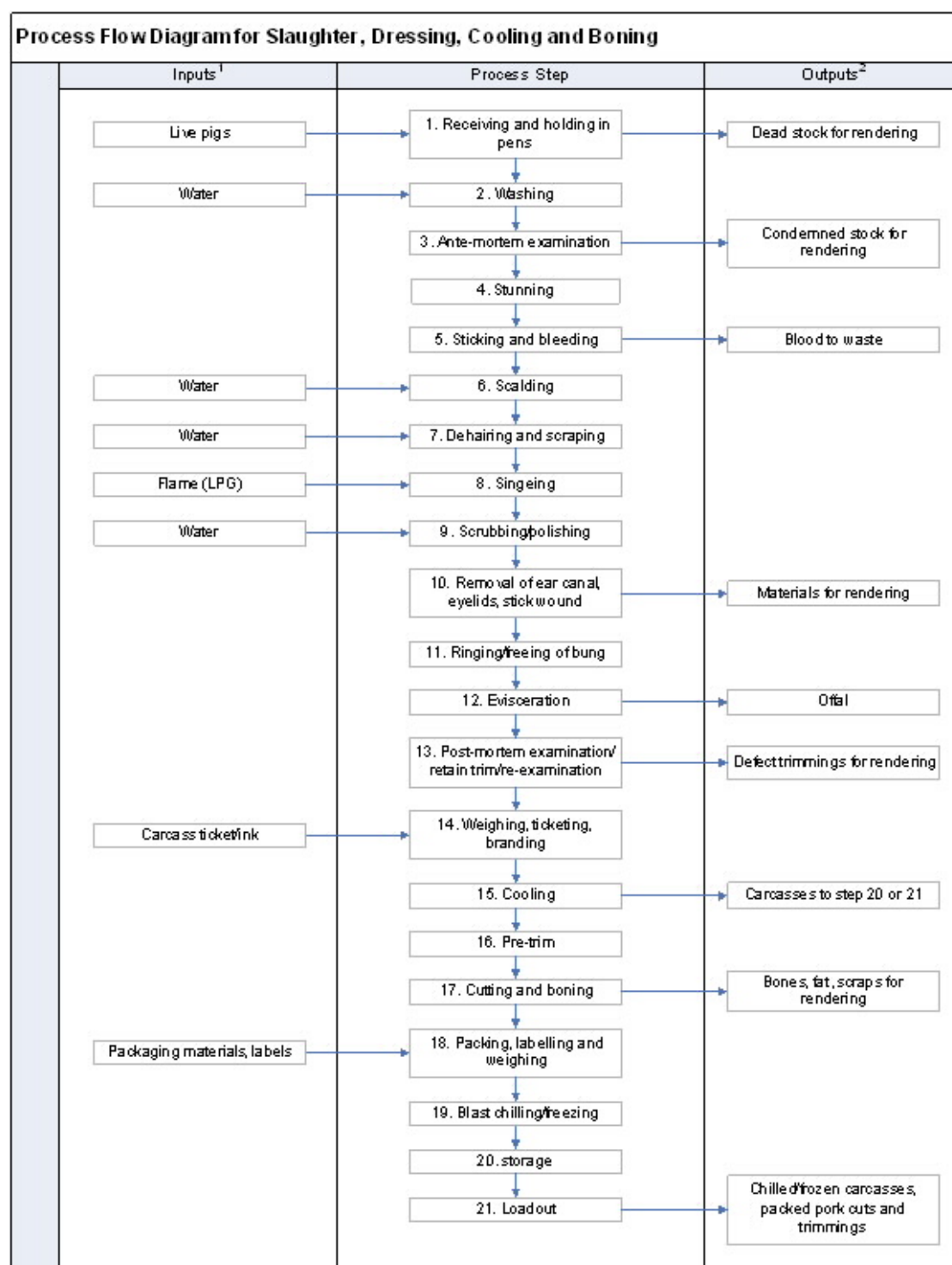
- The 'breeding herd' producing piglets (suckers) to weaning age (about 4-5 weeks old) by breeding sows.
- The 'grower herd' of weaned piglets to slaughter weight.

Breeding stages are mating, gestating and farrowing. Growing stages are weaner, grower and finishing. Each stage tends to be housed in distinct areas of the farm. This involves moving herds and the stress of mixing and moving facilitates transmission of pathogens including *Salmonella*.

#### *Post-harvest*

The flowchart in Figure 3 illustrates an example process flow from receipt of live animals through to despatch.

**Figure 3: Process flowchart for pig slaughter**



Excerpt from: NZFSA Generic RMP Model for the Slaughter and Dressing of Pigs  
[http://www.nzfsa.govt.nz/industry/general/rmp/documents/generic-rmp-models/pigs/rmp-model/page-01.htm#P180\\_7653](http://www.nzfsa.govt.nz/industry/general/rmp/documents/generic-rmp-models/pigs/rmp-model/page-01.htm#P180_7653)

<sup>1</sup> Only those inputs that become part of the final product have been identified in this generic RMP. The operator may wish to include

<sup>2</sup> All outputs for human or animal consumption must be identified in the process flow.

Experimental studies of pork primary processing (Berends *et al.*, 1997) have examined the changes in numbers of Enterobacteriaceae on the skin of pigs. The results show a reduction in numbers by approximately  $2 \log_{10}$  cfu/cm<sup>2</sup> during scalding, and a larger reduction ( $3 \log_{10}$  cfu/cm<sup>2</sup>) during singeing. Increases in bacterial numbers were observed during dehairing, polishing and evisceration, presumably due to contamination from faecal material. Salmonellae will be part of the skin population of Enterobacteriaceae.

A major study in Europe known as “Salinpork” (Lo Fo Wong and Hald, 2000) explored veterinary, epidemiological and economic aspects of *Salmonella* in pork. The research covered four main areas; pre-harvest, harvest, diagnostic procedures and surveillance. Nine centres in six countries were involved; UK, Netherlands, Germany, Denmark, Sweden and Greece.

As part of the Salinpork project, data were collected from 12 slaughterhouses in five countries (Hald *et al.*, 2003). Sweden recorded no isolations (from 5 slaughterhouses). For the remaining 7 slaughterhouses in four countries, *Salmonella* was isolated from 5.3% of 3485 samples from carcasses, livers and tongues (range 2.5% - 8.5%). Of the 1,623 carcasses tested, 62 (3.8%) were positive (range 1% - 8%). 13.8% of 3576 environmental samples were positive (range 6.3% - 28.3%). Prevalence became significantly higher during the warmer months (possibly due to increased ambient temperature encouraging pathogen growth). Environmental contamination also increased significantly during the day of slaughter. The last sample of the day being 4 times more probable to be positive than the first sample, suggesting a build up of bacteria during the slaughterhouse hours of operation. Temperatures of 62°C for scald water and appropriate cleaning/disinfection of polishing equipment at least once a day were recommended.

### 1.3 Prevalence of *Salmonella* in Pork and Pork Products Overseas

Overseas prevalence data for *Salmonella* in pork from individual countries is collated in Table 11, while Table 12 summarises an EU collation of data by the European Food Safety Authority.

In the Northern Ireland study (McDowell *et al.*, 2007) cited in Table 11, there were significant differences in carcass contamination reported between the seasons, with highest prevalences occurring in Spring/Summer and lowest prevalences occurring in Autumn/Winter. Significant differences were also observed in the day of sampling with highest contamination prevalences at the end of the week (Fridays) as opposed to Mondays.

In the Belgium study, (Botteldoorn *et al.*, 2003) study, cross contamination was estimated to account for 29% of positive carcasses with the slaughterhouse environment found to be highly contaminated, even before the start of slaughtering activities. The high number of positive carcasses was attributed to the delivery of *Salmonella*-positive pigs and cross-contamination during slaughtering.

Most prevalent serotypes from the environment and colon were *S. Typhimurium*, *S. Livingstone* and *S. Derby*. *S. Typhimurium* was most prevalent (71% of isolations) on the carcasses.

The results from the Mexican study (Zaidi *et al.*, 2006) were part of a wider surveillance project. Retail poultry and beef were also tested and positive samples were serotyped. Retail



pork (58.1%) and beef (54.0%) were more commonly contaminated with *Salmonella* when compared to poultry (39.7%).

**Table 11: Prevalence of *Salmonella* in pigs and pork overseas**

Country	Year	Samples tested	Number (%) positive	Reference
<b>Australasia</b>				
Australia	NS	50	6 (12)	Morgan <i>et al.</i> , 1987
		50	15 (30)	
<b>Europe</b>				
England	2003-2005	1440 pork samples (from food businesses). consisting of: 477 whole muscle cut 83 joint 729 chops 131 offal (liver, heart, kidney, tripe) 20 other (diced)	56 (3.9) 7 (1.5) 4 (4.8) 14 (1.9) 31 (23.6) 0	Little <i>et al.</i> , 2006
Belgium	NS	370 carcasses 345 colon contents	138 (37) 65 (19)	Botteldoorn <i>et al.</i> , 2003
Ireland	2001 2002	Branded prepacked and loose sausages at retail	(4.4) (1.7) 5 phage types detected, Among the <i>S.</i> <i>Typhimurium</i> isolates, DT104 was predominant.	Boughton <i>et al.</i> , 2004
Netherlands	NS	210 Intestinal tract of apparently healthy pigs 210 carcass swab (after evisceration) 210 lymph nodes 210 carcass swabs (after cooling) 248 minced pork (10g)	44 (21) mean 10 CFU/g 27 (12.9) 7 (3.3) 12 (5.7) 33 (13.3)	Oosterom <i>et al.</i> , 1985
Netherlands	Based on research data	Primal cuts and retail-ready pork (butchers' shops) Minced pork/pork sausages	(5-40) (50-55)	Berends <i>et al.</i> , 1998

Country	Year	Samples tested	Number (%) positive	Reference
Northern Ireland	2002	4 abattoirs 513 caecal contents 507 carcass surface swab 513 meat juice (serological)	161 (31.4) * 203 (40) 111 (21.6) +ve or suspect	McDowell <i>et al.</i> , 2007
Spain	NS	Iberian pork (mainly dry-cured sausages/hams) ## 76 carcasses 71 meat pieces 66 meat for dry-cured sausages 158 equipment surfaces Total 290 samples	3 (3.9) ### 3 (4.5) 9 (13.6) 20 (7)	Palá and Sevilla, 2004
Sweden	Aug. 1997- Aug. 1998	5 slaughterhouses (65% of Swedish pork production) 1060 carcass 359 liver 359 tongue 1610 various environmental samples Total of 3388 samples	(0) (0) (0) (0) (0)	Thorberg and Engvall, 2001
<b>North America</b>				
Canada	June 1995 – April 1996	After evisceration/inspection, caecal material from 1420 apparently healthy 5 month pigs (223 producers)	# 12 (5.2) 95% CI 4.0-6.4%	Letellier <i>et al.</i> , 1999
Canada	NS	596 pork (neck muscle excised before chilling)	67 (11.2)	Lammerding <i>et al.</i> , 1988
Georgia, USA	1983	175 samples of fresh pork sausage Store A 30 Store B 30 Store C 35 Store D 20 Store E 15 Store F 45	Mean 47 (27) 0 15 (50) 15 (43) 5 (25) 5 (33) 10 (22)	Silas <i>et al.</i> , 1984
Mexico	2000-2002	339 retail pork	197 (58.1)	Zaidi <i>et al.</i> , 2006

Country	Year	Samples tested	Number (%) positive	Reference
USA	1997-1998	Sponge samples from 2,127 carcass halves (ham, belly and jowl)**	147 (6.9) SE 0.6	Anonymous 1998
USA	1995-1996	Sponge samples from 2,112 carcass halves (ham, belly and jowl)**	184 (8.7) SE 0.6	Anonymous, 1996b
USA	1999-2000	209 retail	7 (3.3)	Zhao <i>et al.</i> , 2001
Midwest USA	1993	3 slaughtering plants Carcass surfaces; 270 after singeing 270 after final wash 270 after 24 h chilled storage 135 boneless loins before packaging 45 vacuum-packaged loins after 36 days @ 2°C	12 (4.4) 3 (1.1) 1 (0.4) 1 (0.7) 0	Saide-Albornoz <i>et al.</i> , 1995
USA	2000	100 carcasses following bleed-out Faeces from 60 of the 100 bled-out carcasses 122 carcasses after wash, evisceration and overnight chilling	73 (73) 20 (33.3) 1 (0.7)	Tamplin <i>et al.</i> , 2001
LA, Denver, Dallas, Memphis, Sioux Falls, Baltimore, USA	NS	Retail (6 cities) 96 whole-muscle 96 whole-muscle enhanced 96 store-ground fresh/sausage 96 prepackaged ground pork/sausage Total 384  40 Hot-boning plant 40 Slaughter/processing 40 Further-processing Total	8 (8.3) 10 (10.4) 7 (7.3) 12 (12.5) 37 (9.6)  4 (10.0) 3 (7.5) 0 7 (5.8)	Duffy <i>et al.</i> , 2001

Country	Year	Samples tested	Number (%) positive	Reference
USA	1998-2003	560 market hog carcasses	73 (13)	Naugle <i>et al.</i> , 2006
	1998	17	6 (35.3)	
	1999	44	7 (15.9)	
	2000	80	18 (22.5)	
	2001	109	20 (18.3)	
	2002	174	15 (8.6)	
	2003	136	7 (5.2)***	
USA	NS	300 pre-scapular lymph nodes – normal slaughter	0	Bahnson <i>et al.</i> , 2006
		75 pre-scap. lymph nodes (10 herds)	0	
		60 (5 pigs per pool) Ileo-caecal lymph nodes	30 (50)	
<b>Asia</b>				
Thailand	2003	40 pork retail markets	26 (65)****	Angkititrakul <i>et al.</i> , 2005
Japan	NS	94 pork samples from slaughter houses and butchers	3 (3.2)	Tokumaru <i>et al.</i> , 1991
Taiwan	2003	1038 carcass samples	18 (1.7)	Yeh <i>et al.</i> , 2005

# 12 serotypes identified, most frequently isolated were *S. Brandenburg*, *S. Infantis*, *S. Derby*, *S. Typhimurium*, *S. Schwartzengrund* and *S. Urbana*.

## Iberian pigs are extensively reared and undergo different cutting operations, producing different primal cuts from all other intensively reared “white” pigs

### Swabbed at perianal zone

\* Predominantly *S. Typhimurium* (52%) and *S. Derby* (35%)

\*\* 100cm<sup>2</sup> each, 300 cm<sup>2</sup> in total

\*\*\* Under the Pathogen Reduction-HACCP scheme (USDA/FSIS, 1996) the final rule was defined as 8.7% or less of market hog carcasses testing positive for *Salmonella*

\*\*\*\* Most prevalent, *S. Rissen* (61.5%) followed by *S. Stanley* and *S. Lexington* (11.5%). Isolates from humans *S. Rissen* (20.4%) and *S. Stanley* (18.5%).

NS Not stated

**Table 12: *Salmonella* in fresh pork samples in the EU (at cutting and retail level), 2007**

<b>Country</b>	<b>Sample size</b>	<b>n</b>	<b>% pos</b>
<b>At cutting/processing</b>			
Belgium	25g	537	4.1
Estonia	25g	520	0.4
Finland	25g	2329	<0.1
Germany	25g	304	8.9
Ireland	25g	1992	2.9
Slovenia	25g	168	0
Spain	25g	63	7.9
<b>At retail</b>			
Austria	10g/25g	400	1.0
Germany	25g	1664	2.8
Greece	25g/200g	30	0
Luxembourg	25g	39	5.1
Netherlands	25g	277	3.2
Slovenia	25g	385	0.3
Spain	25g	66	6.1

Source: EFSA (2009: p.44)

## 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 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 (39 isolates since 1992, including a small 3 case outbreak in 1997). Of the 39 human isolates 37 were multi-resistant. During the period since 1997 this serotype has only been isolated on 7 occasions from non-human sources (4 bovine, 1 environmental, 1 poultry feed and 1 poultry environment) (Wilson *et al.*, 2000). Three of the non-human isolates have been multi-resistant strains (Carolyn Nicol, ERL, personal communication).

### 2.1 Adverse Health Effects Overseas

Table 13 shows the reported incidence of salmonellosis in several countries.

**Table 13: Reported incidence data for notified cases of salmonellosis overseas\***

Country	Incidence (cases/100,000)	No. of cases	Year	Reference
Australia	45	9,484	2007	1
<b>North America</b>				
USA	16.0	47,995	2007	2
Canada	29.4	9,619	2006	3
<b>Europe</b>				
Belgium	37	3,915	2007	4
Denmark	30	1,648	2007	4
United Kingdom	22	13,557	2007	4
France	8.4	5,313	2007	4
Germany	67	55,400	2007	4

Country	Incidence (cases/100,000)	No. of cases	Year	Reference
Ireland	10	457	2007	4
Netherlands	-	1,224	2007	4
Sweden	43	3,930	2007	4
Spain	-	3,842	2007	4

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

1 OzFoodNet (2008)

2 <http://www.cdc.gov/mmwr/PDF/wk/mm5653.pdf> Summary of notifiable diseases, United States - 2007

3 [www.dsol-smed.phac-aspc.gc.ca](http://www.dsol-smed.phac-aspc.gc.ca) Public Health Agency of Canada: Centre for Infectious Disease Prevention and Control. Does not include *S. Paratyphi*.

4 ECDC (2009)

In terms of the serotypes causing disease overseas, the European Union have collated information on the ten most frequently reported serotypes in 2007 (according to The European Surveillance System “TESSy” for infectious diseases), see Table 14. TESSy represents uploaded case-based and aggregated data that have been approved by each member state and is preferred over the Enter-net method that relies directly on Reference Laboratories or epidemiologists reports.

**Table 14: Ten most commonly confirmed human *Salmonella* serotypes in the EU, 2007**

Serotype	N	%
Enteritidis	81,472	64.5
Typhimurium	20,781	16.5
Infantis	1,310	1.0
Virchow	1,068	0.8
Newport	733	0.6
Hadar	479	0.4
Stanley	589	0.5
Derby	469	0.4
Agona	387	0.3
Kentucky	431	0.3
Other	18,562	14.7
<b>Total</b>	<b>126,281</b>	

Source; EFSA (2009)

### 2.1.1 Contributions to outbreaks and incidents

Salmonellosis is a significant contributor to infectious intestinal disease incidents and outbreaks in many countries as shown by the data summarised in Table 15. The lowest proportion of outbreaks caused by *Salmonella* (3.7%) was in Taiwan (Chiou *et al.*, 1991). Foodborne illness in Taiwan is dominated by outbreaks of infection with *Vibrio parahaemolyticus*, probably due to high consumption of seafood.

It is clear from these overseas data that not only is salmonellosis a significant contributor to foodborne disease, but pork may not be such a significant vehicle as poultry meat and eggs.



**Table 15: Proportion of foodborne disease attributed to infection with *Salmonella* overseas**

Country	Incidents	Outbreaks	Year(s)	Reference
Canada	25.7% of incidents of known cause (78.0% were of unknown cause)	NS	1975-1984 (mean)	Todd, 1992
England and Wales	NS	19.8% outbreaks of known cause, 14.3% of outbreak cases (22.2% were of unknown cause)	1996	Evans <i>et al.</i> , 1998
Japan	NS	17.2% of cases of known cause, 23.8% of outbreak cases (16.2% were of unknown cause)	1981-1995	Lee <i>et al.</i> , 2001
Korea	NS	28.3% of outbreaks of known cause, 31.2% of outbreak cases (26.6% were of unknown cause)	1981-1995	Lee <i>et al.</i> , 2001
Netherlands	14.2% of incidents with known cause (91.7% were of unknown cause)	15.5% of outbreaks of known cause (90.4% were of unknown cause)	1991-1994	Simone <i>et al.</i> , 1997
Sweden	17.6% of incidents of known cause, 14.5% incident cases (66% incidents were of unknown cause)	17.8% of outbreaks of known cause, 14.5% of outbreak cases (61% of outbreaks were of unknown cause)	1992-1997	Lindqvist <i>et al.</i> , 2000
Taiwan	NS	3.7% of outbreaks of known cause (51.4% were of unknown cause)	1981-1989	Chiou <i>et al.</i> , 1991
USA	NS	13.3% of outbreaks of known cause, 37.9% of outbreak cases, and 46.4% of outbreak deaths (68.1% of outbreaks were of unknown cause).	1993-1997	Olsen <i>et al.</i> , 2000

NS = Not Stated

Table 16 gives some examples of salmonellosis outbreaks associated with pork that have been reported in the literature.

**Table 16: Examples of outbreaks of salmonellosis from consumption of pork overseas**

Country	Number involved	Implicated Food and serotype	Year(s)	Reference
<b>Europe</b>				
England	206	<i>S. Typhimurium</i> DT193 Cold roast pork from butcher ( $p < 0.05$ ). Same serotype isolated in pig faeces on farm (inadequate processing and cross contamination)	1989	Maguire <i>et al.</i> , (1993)
England	58	Roast pork <i>S. Typhimurium</i> PT193	1989	Jay <i>et al.</i> , 2003
Denmark	25, (2 fatal)	Danish pork <i>S. Typhimurium</i> DT104	1998	Mølbak <i>et al.</i> , 2000
Denmark	26	Danish pork from same geographic location as cases <i>S. Typhimurium</i> DT12	2005	Torpdahl <i>et al.</i> , 2006
Denmark, Norway and Sweden	47	Danish pork <i>S. Typhimurium</i> U288, RDNC, and U302 (MLVA types)	2008	Bruun <i>et al.</i> , 2009
France	69	Pork sausages <i>S. Manhattan</i>	2005/2006	Noël <i>et al.</i> , 2006
Germany	21	Raw pork meat <i>S. München</i>	2001	Buchholz <i>et al.</i> , 2005
Germany	115 High virulence (55% of cases over 60 years were hospitalised)	Raw minced pork <i>S. Give</i>	2004	Jansen <i>et al.</i> , 2005
Italy	63	Pork salami <i>S. Typhimurium</i> DT104A	2004	Luzzi <i>et al.</i> , 2007
Luxembourg	133, (24 hospitalisations, 1 death)	<i>Salmonella enterica</i> serotype 4,[5],12:i:-DT193 2 outbreaks Locally produced pork (poor hygiene at abattoir)	2006	Mossong <i>et al.</i> , 2007

Country	Number involved	Implicated Food and serotype	Year(s)	Reference
Scotland	472	Raw pork <i>S. Typhimurium</i> PT32	1968	Jay <i>et al.</i> , 2003
<b>USA</b>				
Alaska, USA	21	Roast pork <i>S. Typhimurium</i>	1992	Gessner and Beller, 1994
Arkansas, USA	120	Ham or pork sandwiches, <i>S. Newport</i>	1982	Narain and Lofgren, 1989
<b>Australasia</b>				
Australia	22	Roast pork (internal contamination during deboning, pork served rare) <i>S. Typhimurium</i> PT9	1995	Delpech <i>et al.</i> , 1998
<b>Asia</b>				
Kanagawa, Japan	100+	<i>S. Typhimurium</i> Roast pork	1993	Murase <i>et al.</i> , (2000)

### 2.1.2 Case control studies

Case control studies investigating the causes of infection with *Salmonella* where pork related foods represented an elevated risk are summarised in Table 17.

In the case control study of *S. Typhimurium* in Alaska, the roast pork was consumed at a picnic and leftovers taken home. All ten people who reheated the pork in a microwave oven became ill while there was no illness for twenty people using conventional oven/skillet methods. This was partly attributed to the pork being un-refrigerated for 17-20 hours after cooking.

**Table 17: Case control studies; Relevant risk/protective factors**

Country	Risk/protective factors	Odds Ratios	Reference
Italy	Eating pork salami (risk)	OR 25.5; CI 95% 1.6-416.8	Luzzi <i>et al.</i> , 2007
Alaska, USA	Eating reheated roast pork (risk) Microwave reheating, no protective effect.	RR 8.3* CI 95% 1.2-57.0	Gessner and Beller 1994
SE France	Eating pork sausages (risk)	OR = 5.9 CI 95% 5.9 (1.3:26.9), p=0.05	Noël <i>et al.</i> , 2006
Germany	Eating raw minced pork (risk)	OR 8.0, CI 95% 2.3-27.7, p=0.001	Jansen <i>et al.</i> , 2005

\* Relative Risk

### 2.1.3 Risk assessments and other activity overseas

A quantitative risk assessment for *Salmonella* in pork (RMIS 5426) in both the Republic of Ireland and Northern Ireland, is currently underway at the Irish Agriculture and Food Development Authority (Teagasc) (Deirdre Prendergast, Teagasc, personal communication, December 2007). These jurisdictions have two different approaches to the control of *Salmonella* in pig herds. The outputs of the model are intended to assess the public health risk and to determine the effectiveness of the two different control programmes.

In the USA, a pork food chain model up to the chilled porcine carcass stage, has been developed (Miller *et al.*, 2005). The model predicts annual human salmonellosis cases associated with pork at a mean of 99,430 (90% CI 20,970 – 245,560). Corresponding social costs are estimated as \$US 81.53 million (90% CI \$18.75 - \$197.44 million). Changes in *Salmonella* status during processing were found to be more important in terms of human health risk than on-farm strategies.

In Norway, Sandberg *et al.* (2002) used population data, prevalence data from *Salmonella* surveillance and information from control programmes for pigs (termed NSSCP) (years 1998-1999) in a simulation model. The model was in three parts, individual prevalence at abattoir, sampling strategy and within herd prevalence. The model predicted that the NSSCP controls did not have any significant consumer protection effect.

An epidemiological and economic simulation model has been produced by van der Gaag (2004) in the Netherlands. In terms of the economic model, for each stage a package of feasible measures to control *Salmonella* were simulated. The net costs for the control package per pig were 2.99 Euros (transportation stage 0.65 Euros, lairage 0.40 Euros and slaughtering 1.47 Euros).

A quantitative risk assessment has been undertaken for consumers of pork products (especially fresh sausage) in an Italian region (Giovannini *et al.*, 2004). The authors concluded that the sausages may be an important source of infection.

In February 2008 the European Food Safety Authority (EFSA) published “A quantitative microbiological risk assessment on *Salmonella* in meat: source attribution for human salmonellosis from meat<sup>1</sup>. The report notes that there are many approaches to source attribution in use amongst Member States, and data gaps impede full analyses in many instances. “In the EU, among the foodborne cases of human salmonellosis, egg and egg products are still the most frequently implicated sources. Meat is also an important source of human salmonellosis, with poultry and pork implicated more often than beef and lamb. More specific conclusions about the relative importance of specific meat categories brought into the kitchen raw, for example fresh meat and products thereof, minced meat and meat preparations, cannot be made at present.” A data request concerning *Salmonella* in pork was issued<sup>2</sup> as a contribution to the consortium of European researchers who have been charged with developing a quantitative microbiological risk assessment.

The METZOON model is a quantitative microbial risk assessment for human salmonellosis in Belgium from consumption of fresh minced pork meat (Bollaerts *et al.*, 2009). It includes six

---

<sup>1</sup> [http://www.efsa.europa.eu/EFSA/efsa\\_locale-1178620753812\\_1178686062644.htm](http://www.efsa.europa.eu/EFSA/efsa_locale-1178620753812_1178686062644.htm)

<sup>2</sup> [http://www.efsa.europa.eu/EFSA/efsa\\_locale-1178620753812\\_1178696473049.htm](http://www.efsa.europa.eu/EFSA/efsa_locale-1178620753812_1178696473049.htm)

consecutive modules: primary production, transport and lairage, slaughterhouse, post-processing, distribution and storage, and preparation and consumption. The model predicts that the risk from undercooking and cross contamination will result in 9,000 – 38,000 cases (90% CI) per year, which was considered within the range based on surveillance (1,000 – 70,000).

## APPENDIX 3: OVERSEAS CONTROL MEASURES

A European Commission Scientific Opinion on the food safety aspects of pig housing and husbandry systems was published in 2007 (EFSA, 2007a). It appeared that practices considered beneficial in terms of pig welfare were having a negative effect on the exposure to, and spread of, food-borne pathogens including *Salmonella*. Examples of pig welfare practices included holding in groups, use of bedding, use of non-slippery floors (difficult to sanitise) and access to outdoor spaces. Further research programmes that achieve a synergism between the two were recommended.

Another EFSA Opinion published in 2006 concerned “Risk assessment and mitigation options of *Salmonella* in pig production”<sup>1</sup>. It was noted that the most common serotype from pork causing human illness was *S. Typhimurium*. It was recommended that measures should address (i) the prevention of introduction of *Salmonella* into the herd (ii) the prevention of in-herd transmission and (iii) increase of resistance to the infection. No universal mitigation option was identified.

Following research into the occurrence of *Salmonella* in the lymph nodes, tonsils and carcasses of pork, Vieiro-Pinto *et al.* (2005) have suggested that to control contamination during the slaughter process better, improved evisceration techniques, and extraction of the tonsils and mandibular lymph nodes would be beneficial.

In Europe, EC Regulation 2160/2003 lays down provisions for the control for *Salmonella* and other zoonotic agents. However, it is recognised that a Community target needs to be established for reduction of the organism in breeding pigs in the EU before these Regulations can be implemented. A proposal by EFSA focuses on the *Salmonella* prevalence and serotyping in pig breeding and pig ‘production holding’ establishments across the EU. Work is now progressing on collection of these data (EFSA, 2007b).

### 3.1 Denmark and the Netherlands

The official policy in Denmark is to target *Salmonella* at its source. During the early 1990s, a rising burden of illness was attributed to *Salmonella* in pork. A number of initiatives were set up and, since 1995, the number of *Salmonella*-infected pigs has been in decline. A seasonal variation and trends analysis of *Salmonella* in pigs, pork and humans undertaken between January 1995 and July 2000 found a double peaked annual cycle in the disease. Prevalence in pork and humans followed a very similar course, whereby a peak of prevalence in pork was found 4 – 5 weeks before a peak of case registration (Hald and Andersen, 2001).

The control programme is based on two monitoring programmes: herd monitoring based on serology and bacteriological follow-up, and post-chill monitoring of carcasses (Goldbach and Alban, 2006). Herds are assigned to one of three levels based on the proportion of reactor positive results, and herds at the highest level are slaughtered logistically at the end of the day with additional sanitary precautions, such as no head splitting and offals being condemned or heat treated.

---

<sup>1</sup> [http://www.efsa.europa.eu/EFSA/efsa\\_locale-1178620753812\\_1178620776028.htm](http://www.efsa.europa.eu/EFSA/efsa_locale-1178620753812_1178620776028.htm)

Four additional control strategies for Denmark have been assessed in a cost-benefit analysis (Goldbach and Alban, 2006). The strategies were;

- 1) Hot-water decontamination, (showered with 80°C water for 14-16 seconds);
- 2) Sanitary slaughter where herds with high prevalence of *Salmonella* were encountered (logistic slaughter);
- 3) Use of home-mixed feeds; and
- 4) Acidified feeds for slaughter pigs.

Only hot-water decontamination was considered economically viable with an expected 2-log<sub>10</sub> reduction of *Salmonella*. A treatment with organic acid has a similar effect (van Netten *et al.*, 1995) but decontamination with organic acids is not permissible in the European Union or New Zealand (Titus, 2007).

In terms of the cost of *Salmonella* controls in poultry and pigs in Denmark, Wegener *et al.* (2003) determined that the initial phase costs were in the region of \$US14.1 million per annum (2001 figures). Operational costs are lower to approximately US\$8.5 million per annum (based on 21,000 producers of 21-22 million slaughter pigs a year). For pork, control costs equate to \$US0.075/kg. But the control measures are estimated to have saved Denmark direct health costs and lost productivity days. For 2001, foodborne salmonellosis (note: not confined to pork) cost the Danish \$US15.5 million – based on 54.6 cases per 100,000 (and 10% laboratory confirmed).

Logistical slaughter (processing of infected pigs before uninfected pigs) has been investigated in the Netherlands (Swanenburg *et al.*, 2001a) and has been useful in decreasing the prevalence of *Salmonella*-contaminated pork in sero-negative pig herds.

## 3.2 USA

In 1997 the US Federal Government introduced a new food safety regulation for meat and poultry slaughter and processing plants. The regulation was called the Pathogen Reduction; Hazard Analysis and Critical Control Point (PR/HACCP) rule, and is administered by the Food Safety and Inspection Service (FSIS) of the United States Department of Agriculture (USDA). The components of this programme include:

- 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;

The system became effective in large establishments in January 1998, small establishments in January 1999 and for very small establishments in January 2000. Baseline levels were measured by the USDA from April 1995 to March 1996 before the introduction of PR/HACCP. The baseline was determined at 8.7% positive for *Salmonella* based on 2112 carcass swab samples. Further testing by USDA up to 2001 demonstrated a reduction in overall *Salmonella* contamination in market pigs of 5.4%, with reductions seen across all sizes of processing plants. Overall the major reductions have been attributed to the initial implementation of HACCP programmes.