



**RISK PROFILE:
LISTERIA MONOCYTOGENES
IN PROCESSED READY-TO-EAT MEATS**

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

by

Sue Gilbert
Rob Lake
Andrew Hudson
Peter Cressey

December 2009

Client Report
FW08021

RISK PROFILE:
LISTERIA MONOCYTOGENES
IN PROCESSED READY-TO-EAT MEATS

Dr Stephen On
Food Safety Programme Leader

Rob Lake
Project Leader

Maurice Wilson
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 food consumption information (24-hour dietary recall and qualitative food frequency questionnaire) from these surveys.

CONTENTS

EXECUTIVE SUMMARY	1
1 STATEMENT OF PURPOSE.....	3
1.1 Food/Hazard Combination and Risk Management Questions.....	4
2 HAZARD AND FOOD.....	5
2.1 <i>Listeria monocytogenes</i>	5
2.2 Sources of <i>Listeria monocytogenes</i>	5
2.3 Processed Ready-to-Eat Meats.....	6
2.4 The Food Supply in New Zealand	7
2.4.1 Production.....	7
2.4.2 Imported foods.....	8
2.4.3 Behaviour of <i>L. monocytogenes</i> on processed ready-to-eat meat	9
2.5 Exposure Assessment.....	11
2.5.1 <i>Listeria</i> in raw meat.....	11
2.5.2 <i>Listeria</i> in processed ready-to-eat meat products	13
2.5.3 Food consumption: Processed ready-to-eat meat products	15
2.6 Qualitative Estimate of Exposure	17
2.6.1 Number of servings of ready-to-eat meat and serving size	17
2.6.2 Contamination frequency	18
2.6.3 Predicted contamination level at retail	19
2.6.4 Growth rate during storage and most likely storage time.....	19
2.6.5 Heat treatment.....	20
2.6.6 Exposure summary	20
3 EVALUATION OF ADVERSE HEALTH EFFECTS	21
3.1 Listeriosis	21
3.2 Non-Invasive Febrile Gastroenteritis	21
3.3 Dose Response	22
3.4 Adverse Health Effects in New Zealand.....	22
3.4.1 Incidence.....	22
3.4.2 Clinical consequences of <i>Listeria</i> infection	23
3.4.3 Outbreaks.....	24
3.5 High Risk Groups in the New Zealand Population.....	25
3.5.1 Perinatal population.....	26
3.5.2 Elderly population	26
3.5.3 Immune compromised	26
3.6 Adverse Health Effects Overseas.....	27
3.7 Health Burden due to Invasive Listeriosis	27
4 EVALUATION OF RISK.....	28
4.1 Existing Risk Assessments.....	28
4.2 Estimation of Risk for New Zealand.....	28
4.2.1 Risks associated with processed ready-to-eat meats	28
4.2.2 Risks associated with other foods.....	29
4.2.3 Risk assessment options	30
4.3 Data Gaps.....	30
5 AVAILABILITY OF CONTROL MEASURES	31

5.1	Risk Management Strategy	31
5.2	Regulatory Controls	31
5.2.1	Australia New Zealand Food Standards Code.....	31
5.2.2	Food Act and Food Hygiene Regulations	32
5.2.3	Animal Products Act and Risk Management Plans.....	32
5.2.4	Codes of Practice	33
5.2.5	Microbiological criteria	33
5.2.6	Industry controls.....	34
5.3	Risk Communication.....	35
5.4	Control Options.....	36
5.5	Commentary on Control Options.....	37
6	REFERENCES	39
APPENDIX 1	HAZARD AND FOOD.....	50
1.1	<i>Listeria monocytogenes</i>	50
1.1.1	Growth and survival	50
1.1.2	Inactivation (CCPs and Hurdles).....	50
1.2	Prevalence of <i>Listeria</i> in Processed Ready-to-eat Meat Products Overseas.....	51
1.2.1	Handling and Packaging.....	62
APPENDIX 2	EVALUATION OF ADVERSE HEALTH EFFECTS.....	64
2.1	Dose Response	64
2.1.1	Listeriosis	64
2.1.2	Febrile gastroenteritis	65
2.2	Adverse Health Effects Overseas.....	66
2.2.1	Incidence.....	66
2.2.2	Contributions to outbreaks and incidents	66
2.2.3	Case control studies	67
APPENDIX 3	EVALUATION OF RISK	71
3.1	Risk Assessments.....	71
3.1.1	FAO/WHO	71
3.1.2	USA	72
3.1.3	Australia	74
3.1.4	Canada	78
APPENDIX 4	CONTROL MEASURES OVERSEAS	79
4.1	Codex	79
4.2	Australia.....	79
4.3	United States of America	80
4.4	Canada.....	81
4.5	England and Wales.....	81
4.6	Denmark.....	82

LIST OF TABLES

Table 1:	Types of processed ready-to-eat meats	7
Table 2:	New Zealand study; prevalence of <i>L. monocytogenes</i> in various red meat samples (1988)	12
Table 3:	Reported prevalence of <i>Listeria</i> in ready-to-eat (RTE) meat products in New Zealand.....	13
Table 4:	Reported prevalence of <i>Listeria</i> in ready-to-eat poultry products in New Zealand	15
Table 5:	Number of reported cases of invasive listeriosis and mortality from 1997 to 2008 (Williman <i>et al.</i> , 2009).....	22
Table 6:	Outcome data for listeriosis in New Zealand, 1997 - 2008	23
Table 7:	Relative susceptibility to <i>L. monocytogenes</i> for certain underlying health conditions.....	25
Table 8:	FSANZ Guidelines for <i>L. monocytogenes</i> in ready-to-eat foods.....	34
Table 9:	Results from a <i>Listeria</i> spp. knowledge questionnaire, Middlemore hospital.....	35
Table 10:	Reported prevalence of <i>Listeria</i> in overseas meat products	54
Table 11:	Prevalence of <i>L. monocytogenes</i> in ready-to-eat meat and poultry, 1990 – 1999 at 1800 production facilities across the USA.....	57
Table 12:	<i>L. monocytogenes</i> in ready-to eat meat products in the European Union, 2006..	58
Table 13:	Percentage of positive <i>L. monocytogenes</i> prevalence in ready-to-eat meat and poultry products in the European Union, 2004-2006.....	59
Table 14:	Reported prevalence of <i>L. monocytogenes</i> and <i>Listeria</i> species in overseas ready-to-eat poultry meat products	60
Table 15:	Quantitative data for <i>L. monocytogenes</i> in overseas ready-to-eat meats and poultry	61
Table 16:	Comparison of listeriosis incidence between countries	66
Table 17:	Overseas listeriosis outbreaks associated with ready-to-eat meat consumption..	68
Table 18:	Overseas listeriosis outbreaks associated with ready-to-eat poultry consumption	69
Table 19:	Contribution of <i>L. monocytogenes</i> to foodborne disease outbreaks and incidents overseas.....	69
Table 20:	Case control studies containing information on <i>L. monocytogenes</i> in ready-to-eat meats	69
Table 21:	Predicted relative risk rankings for listeriosis based on the North American sub-population using median estimates on a per serving basis	73
Table 22:	Risk Ranking of smallgoods and <i>L. monocytogenes</i> (South Australia).....	75
Table 23:	<i>L. monocytogenes</i> in processed meat products, Australia.....	76
Table 24:	Food categories and action levels (applicable in Australia only)	80
Table 25:	The microbiological criteria for <i>L. monocytogenes</i> for different categories of food and corresponding action levels in Canada	81
Table 26:	Food groups and tolerances for <i>L. monocytogenes</i> in Denmark.....	82

LIST OF FIGURES

Figure 1:	The four steps of the Risk Management Framework.....	3
Figure 2:	Invasive listeriosis notifications by year 1995 – 2008.....	23
Figure 3:	Number of listeriosis cases reported by month in New Zealand (January 2000 – January 2008).....	24
Figure 4:	Dose response models at median values for R for disease caused by <i>L. monocytogenes</i> *	64

EXECUTIVE SUMMARY

The NZFSA Statement of Intent 2008-2011 identifies control of *Listeria monocytogenes* as a strategic priority, with the performance indicator being no increase in cases of listeriosis after five years. This Risk Profile is intended to help inform the NZFSA *Listeria monocytogenes* Risk Management Strategy, and to provide a scientific underpinning for associated risk management measures. It is an update of a previous document completed in 2002, and describes the risk to New Zealand consumers from *Listeria monocytogenes* in processed ready-to-eat meats.

The number of invasive listeriosis cases reported every year is very small relative to other forms of potentially foodborne disease. The importance and high burden of the disease derives from the high proportion of serious outcomes for infants and fetuses.

The rate of reported invasive listeriosis in New Zealand has been static for many years, and is similar to that found in comparable countries. As in other countries, most cases are sporadic, with outbreaks being rare. There have been only two reported outbreaks involving *L. monocytogenes* in New Zealand; one associated with smoked mussels, and one of unknown source producing mainly non-perinatal cases. Both of these outbreaks involved the invasive form of listeriosis.

Analysis of Episurv data found 174 cases of listeriosis notified between 2000 and 2007. Foodstuffs implicated (but not confirmed) were noted for 16 cases (9%) which demonstrates the difficulty in determining the source of infection.

The incidence of non-invasive disease from *L. monocytogenes* infection in New Zealand is unknown. It is not normal practice for clinical laboratories to examine faecal specimens from cases of gastrointestinal disease for the presence of *L. monocytogenes* and it might be that more outbreaks will be reported as this form of the disease gains recognition. Two linked New Zealand outbreaks of non-invasive listeriosis have been reported and these involved cooked ready-to-eat meat products.

L. monocytogenes has been detected in a range of New Zealand ready-to-eat meats; the best data are for the most commonly consumed ready-to-eat meat i.e. ham, with a prevalence of approximately 3.5%.

The median daily consumption of ready-to-eat meats in New Zealand is similar to that for Australia, and somewhat lower than the amounts consumed in the USA. Although the data on imported processed meat products (mostly from Australia) do not clearly identify ready-to-eat meats as such, it appears that the large majority of ready-to-eat meats consumed in New Zealand are produced locally. Ham is the most commonly consumed type of ready-to-eat meat, followed by luncheon meat and corned beef. These would be included in the category of “deli meats” ranked first for relative risk of listeriosis in the USDA risk assessment. The US risk assessment has also attributed most of the risk from deli meats to those sliced or packaged at retail; this is consistent with the New Zealand surveys that found a higher prevalence of *L. monocytogenes* in this type of ham sample, compared to pre-packaged ham.

It is very difficult, if not impossible, to completely eliminate *Listeria* from food processing environments. Effective Food Control Plans for manufacturers of ready-to-eat meats will be

an essential part of risk management, and transition of this sector to requirements of the new domestic food legislation is anticipated in approximately 2011.

A quantitative risk assessment performed in Australia, where contamination and consumption prevalences are similar to New Zealand concluded that ready-to-eat meats were responsible for up to 40% of cases of listeriosis, based on a prevalence of contamination similar to that found in New Zealand. This attribution is in good agreement with the results of an expert elicitation for New Zealand, which estimated that 85% of listeriosis was foodborne and of this foodborne component 54% was due to transmission via processed ready-to-eat meats.

In their statement of intent the NZFSA have provided an indicator for listeriosis of “no increase in the foodborne component with increasing range of foods available to the consumer”. The data from New Zealand, and risk assessments from the USA and Australia indicate that maintained or improved risk management for *L. monocytogenes* in processed ready-to-eat meats would be an important contribution to achieving this objective.

The burden of illness analysis indicates that principal target for risk management would be pregnant women; a recent study in New Zealand indicates that although awareness of risk is high amongst this group, avoidance of high risk foods is less than ideal. This reinforces the need for preventive measures in the manufacturing sector.

The data gaps identified by this Risk Profile are:

- Incidence of the non-invasive form of listeriosis in New Zealand
- Data on degree of implementation and effectiveness of HACCP based food safety plans by the ready-to-eat meat food sector.
- Prevalence and quantitative data on a wider range of processed ready-to-eat meat products.
- Times and temperatures of storage (both at retail and domestically) for ready-to-eat meat products.
- More up-to-date food consumption information to support the perceived emergence of a wider range of processed ready-to-eat meat products. The results of the latest National Nutrition Survey, due in 2011, will go some way to addressing this 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.

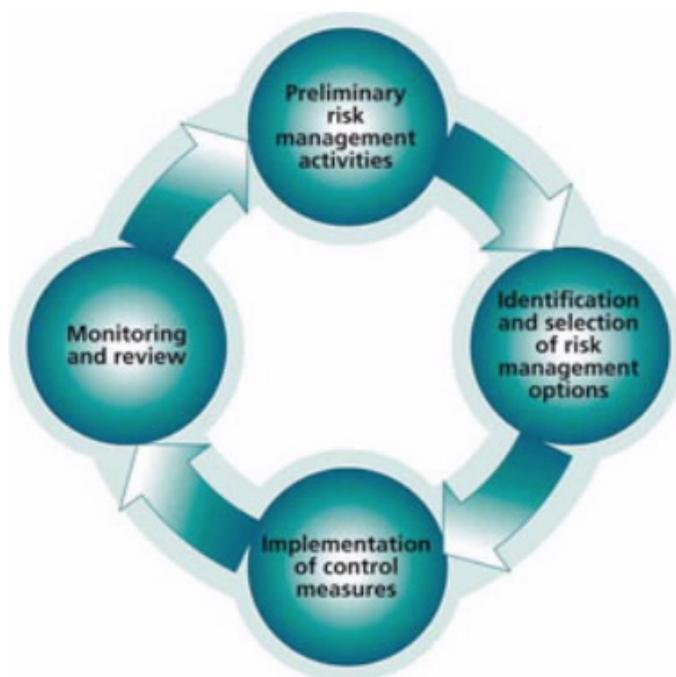


Figure 1: The four steps of the Risk Management Framework

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

NZFSA has recognised *Listeria monocytogenes* as one of the three most important foodborne pathogens in New Zealand and have developed a *Listeria monocytogenes* Risk Management Strategy. A number of Risk Profiles have been commissioned as part of the preliminary risk evaluation activities underpinning this strategy.

The food/hazard combination addressed by this Risk Profile is *Listeria monocytogenes* in processed ready-to-eat meats. It is an update of the existing Risk Profile completed in 2002.

There are many types of ready-to-eat meats considered in this Risk Profile. Based on processing they can be grouped as: raw cured shelf stable meats, dried meats, cooked perishable uncured meats, cooked perishable cured meats.

The NZFSA have commissioned this Risk Profile to address the following specific risk management questions:

- Has the level of risk attributable to *L. monocytogenes* in processed ready-to-eat meats changed since the 2002 Risk Profile was undertaken?
- Has the quality of information available for the profiling of risk attributable to this food/hazard combination changed?

2 HAZARD AND FOOD

2.1 *Listeria monocytogenes*

Six species comprise the genus *Listeria* (ICMSF, 1996). *L. grayi* and *L. innocua* are considered non-pathogenic, while *L. seeligeri*, *L. ivanovii*, and *L. welshimeri* are rarely causes of human infection. *L. monocytogenes* is the most important species with respect to human health.

Two forms of disease caused by this organism are now recognised; a serious invasive disease and a non-invasive gastroenteritis. While the invasive form of disease is uncommon, the clinical consequences are often serious. The organism's ability to grow at refrigeration temperatures is significant as chilling is often used as a control measure in the food industry.

2.2 Sources of *Listeria monocytogenes*

Human: *L. monocytogenes* is carried asymptotically in the faeces of 2-6% of the population. Person-to-person spread (other than mother to foetus) is infrequently reported but has been recognised. *L. monocytogenes* is shed in high numbers ($\geq 10^4$ /g) in the faeces of infected people.

Animal: Can cause disease in animals. Veterinarians were originally considered to be an "at risk" group, but the World Health Organization has stated that animals are not considered to be important as direct sources of human infection. Occasional incidents of cutaneous infection in livestock handlers have been reported. *Listeria* spp. present in animal faeces can contaminate milk or red meat. Improperly made silage can be a source of domestic animal infection.

Food: Should be considered as potentially present in all raw foods and ingredients. May be present in cooked foods as a result of post-cooking contamination. Risk posed is likely to be greatest in ready-to-eat cooked foods with long shelf lives on which *L. monocytogenes* can grow. Has been isolated from a wide variety of ready-to-eat and raw foods in NZ studies. Little information is available regarding numbers of cells; is considered to be present in low numbers (<10/g) on most foods, although it has been detected at numbers far in excess of this.

Environment: Is widespread in the environment including soil, vegetation, water and sewage. In domestic environments, 101/213 (47.4%) houses in the Netherlands had *Listeria* spp. present (Beumer *et al.*, 1996). Dishcloths (37%) and bathroom drains (27.2%) were most frequently contaminated. Enumeration of dishcloths and washing-up brushes found 10^4 CFU/object. From kitchen sinks, refrigerator vegetable compartments and tooth brushes, 10^3 CFU/object were obtained. Domestic refrigerators have been shown to harbour *L. monocytogenes* (Azevedo *et al.*, 2005; Sergelidis *et al.*, 1997).

Transmission routes: Transmission is mainly via food, according to a New Zealand expert elicitation process (Cressey and Lake, 2007). Alternative routes include infections acquired in hospital (nosocomial) and occupational exposure, for example through skin infections (e.g. veterinarians, farmers) (Cain and McCann, 1986).

2.3 Processed Ready-to-Eat Meats

Ready-to-eat meats are products whose processing includes one or more pathogen control steps to render the products safe for consumption without further processing or cooking by the consumer. The processed meats considered in this category principally include the red meats pork, beef, and lamb, or mixed species products. Poultry products are also included in this risk profile, as ready-to-eat poultry products will usually be processed, sold and consumed in the same way as red meat products.

Processing of ready-to-eat meats may involve the following steps, alone, or in combination.

- Comminution;
- Addition of binders, extenders and emulsifiers (isolated soya protein or milk proteins, gums, etc);
- Addition of flavouring (spices etc);
- Addition of antimicrobial preservatives (e.g. sodium nitrite)
- Heating (pasteurising) (cooking, baking, boiling, steaming);
- Curing;
- Smoking;
- Fermentation;
- Drying;
- Vacuum or modified atmosphere packaging; and,
- Refrigerated or frozen storage.

Of these steps, all but the first two (comminution and addition of binder, extenders and emulsifiers) have the potential to control pathogens.

Meat and poultry are cured by the addition of salt alone or in combination with one or more ingredients such as sodium nitrite, sugar, curing accelerators, and spices. These are used for partial preservation, flavouring, colour enhancement, tenderising and improving yield of meat products (ACT Health Protection Service, 1998). The process may include dry curing, immersion curing, direct addition, or injection of the curing ingredients. Several chemical preservatives are permitted to be added to cured meats, including nitrites (sodium and potassium salts) and nitrates (sodium and potassium salts) (FSANZ, 2009). Sodium nitrite is the most commonly used, due in part to its positive impact on meat colour. The maximum permitted concentration of sodium nitrite in cured meats is 125 mg/kg.

There are many types of processed ready-to-eat meats. Examples are given in Table 1, grouped according to processing (ICMSF, 1998).

Table 1: Types of processed ready-to-eat meats

Processed RTE meat type	Examples	Microbial control
Raw cured shelf stable meats	Raw ham, Chinese sausage (La Chang), salami, fermented sausages	Low water activity (curing and/or drying) or low pH and reduced water activity
Dried meats	Biltong, Rou Gan, beef jerky	Low water activity
Cooked perishable uncured meats	Roast beef and other cooked meats not reheated before consumption	Cooking
Cooked perishable cured meats	Cooked ham, pastrami, silverside, corned beef, luncheon meat, frankfurters, pâté	Cooking, preservatives (sodium nitrite), refrigeration

Ready-to-eat foods are vulnerable to recontamination with *L. monocytogenes* during handling following listericidal treatment. This may occur during further processing or packaging at the manufacturing facility, during processing (e.g. slicing) and packaging at the retail level, or in the domestic environment. The organism's ability to grow at low temperatures during any subsequent period of storage before consumption increases the risk. *L. monocytogenes* may be present in the environment of many food-processing and retail food facilities, and its complete elimination is extremely difficult.

A distinction can be made between ready-to-eat meats that are unlikely to support growth of *L. monocytogenes* following contamination (raw cured shelf stable meats, dried meats) and those which could allow growth (cooked perishable cured and uncured meats).

2.4 The Food Supply in New Zealand

While processed ready-to-eat meats may be based on any source meat (beef, sheep meat, pork, chicken, venison, etc.), available evidence suggests that the vast majority of these products consumed in New Zealand (see section 2.5.3) are derived from pork. It should be pointed out that this conclusion is based on data from the National Nutrition Surveys (Ministry of Health, 2003; Russell *et al.*, 1999), and the data are now quite old. The lack of an industry body with a focus on all processed ready-to-eat meats also means that production statistics are not readily available. For these reasons the following section concentrates on supply of the major known raw materials for processed ready-to-eat meat production.

2.4.1 Production

The largest component of processed ready-to-eat meat consumption in New Zealand is ham. The production of pig meat in New Zealand in 2007 was approximately 50,183 tonnes (bone-in equivalent weight), supplemented by approximately 40,434 tonnes of imported pigmeat (total 90,617 tonnes) (New Zealand Pork Industry Board, 2007).

In 1997-1998 approximately 11,000 tonnes of ham and 16,000 tonnes of other predominantly pork smallgoods were produced. Other pork production in 1997-1998 included bacon (approximately 12,000 tonnes) and sausages (approximately 17,000 tonnes), but these would not be considered ready-to-eat (<http://www.stats.govt.nz/datasets/manufacturing/bacon-and->

[ham-production.htm](#)). Collection of smallgoods production data by Statistics New Zealand was discontinued in 1999.

The New Zealand Pork Industry Board (NZPIB) provided an estimate of total uncooked comminuted fermented meat (UCFM) annual production in New Zealand in 2002 at 343,367 kg. No data concerning the overall production of other processed ready-to-eat meats in New Zealand have been located.

2.4.2 Imported foods

Approximately 41% of pork for domestic consumption was imported in 2006, increasing to a 45% share in 2007 (New Zealand Pork Industry Board, 2007). In terms of weight, Statistics New Zealand give the following information; for the year to September 2007 New Zealand imported 10,156 tonnes of pig meat from Australia, 9,488 tonnes from Canada, 4,152 tonnes from the United States, 1,757 tonnes from Finland, and 1,471 from Sweden. All were frozen meat carcasses and cuts, with the exception of 461 tonnes of fresh or chilled product imported from Australia.

Since 2001, sanitary measures have been applied to imported pork, to prevent introduction of Porcine Reproductive and Respiratory Syndrome (PRRS) virus (Biosecurity New Zealand, 2006). Provisional measures adopted at that time require imported pork is cooked, or else frozen and imported into a transitional facility where it is cooked, or subjected to certain pH levels (Biosecurity New Zealand, 2006). A risk analysis was completed in 2006 and concluded that “the risk of PRRS in imported pig meat is non-negligible, and the following sanitary measures are recommended to manage the identified risk:

Pig meat must be:

either

- From a country free from PRRS

or

- Treated prior to import or on arrival, in an officially approved facility, but approved cooking or pH change

or

- In the form of consumer-ready, high value cuts

or

- Further processed on arrival, in an officially approved facility, into consumer-ready high value cuts”

Completion of the risk analysis was followed by drafting of an import health standard (IHS) for pig meat and pig meat products for human consumption from Finland or Sweden in 2009 (Biosecurity New Zealand, 2009). Finland and Sweden were considered to be PRRS free, although an outbreak of PRRS occurred in Sweden in 2007.

New Zealand imports relatively small amounts of processed meats. For the year to September 2007, 1,901 tonnes of meat preparations in airtight cans or jars were imported. During the same period 3,421 tonnes of meat preparations of various types (sausages and similar products, pâté, hams and cuts) not in airtight cans or jars were imported. Of this total of 5,322 tonnes, 4,238 tonnes (80%) were imported from Australia. Only some of these imported meat products will be ready-to-eat, but for such products New Zealanders are reliant on the exporting country’s food safety assurance programmes.

2.4.3 Behaviour of *L. monocytogenes* on processed ready-to-eat meat

2.4.3.1 Impact of processing

Farber *et al.* have followed the behaviour of *L. monocytogenes* on various styles of uncooked fermented sausages (Farber *et al.*, 1993). In German-style sausages prepared with starter-culture, levels of the organism decreased by 2-3 log₁₀ CFU/g after fermentation and smoking, and a further 1-2 log₁₀ CFU/g during drying. Similar results were found in American-style sausage: 5 log₁₀ CFU/g reductions after the fermentation and smoking process. In Italian-style sausage with no starter culture or smoke used, the numbers of *Listeria* increased during the 5 day fermentation, remained constant during the drying period, and decreased slightly during the 4 week holding period at 4°C. The trials demonstrated how inconsistent fermentation can be when sausages are made without a starter culture.

The behaviour of *L. monocytogenes* has been examined on heated (in marinade) and unheated meat strips followed by a drying phase to make beef jerky (Harrison and Harrison, 1996). On the unheated inoculated strips, the population decreased by 1.8 log₁₀ CFU/g during the first 3 hours of drying, and after 10 hours, the population had decreased by almost 6 log₁₀ CFU/g. In the heated inoculated meat, the cooking phase (71.1°C) decreased the population by 4.5 logs, and the micro-organism was undetectable after a further 10 hours of drying.

The potential for *L. monocytogenes* to survive the cooking process and grow on processed chicken has been investigated (Carpenter and Harrison, 1989). Chicken breasts were inoculated with *L. monocytogenes* (5-6 log₁₀ CFU/g), baked to five different internal temperatures (65.6, 71.1, 73.9, 76.7, 82.2°C), and then either vacuum-packaged or wrapped in oxygen-permeable film and stored for up to 4 weeks at 4°C or 10°C. At the three higher cooking temperatures the *L. monocytogenes* population was reduced 4-5 log₁₀ CFU/sample, while at 65.6 or 71.1°C the reduction was 1.8-2.8 log₁₀ CFU/sample.

At 10°C storage, *Listeria* numbers increased significantly regardless of the type of packaging. However, at 4°C storage the type of packaging affected growth. In the vacuum-packaged samples, significant growth occurred only in the sample heated to 65.6°C. The micro-aerophilic atmosphere created in the oxygen-permeable wrapped samples appeared to contribute to significant increases in the *L. monocytogenes* population by week 4 in all samples, except (for no apparent reason) in the samples heat treated to 71.1°C.

The ability of *L. monocytogenes* to grow on processed poultry has been attributed to the absence of nitrite (Duffy *et al.*, 1994).

2.4.3.2 Behaviour following post-processing contamination and storage

Ready-to-eat meats may become contaminated or recontaminated following processing. One possible source of contamination of ready-to-eat meats with *L. monocytogenes* is commercial mechanical slicers used at delicatessen counters. Two US studies have investigated cross-contamination dynamics (Lin *et al.*, 2006; Vorst *et al.*, 2006).

A comparative study of *L. monocytogenes* behaviour on a range of processed meat products during chilled stored has been published (Glass and Doyle, 1989). Meats included ham, bologna, weiners, sliced chicken, sliced turkey, fermented semi-dry sausage, bratwurst and

cooked roast beef. The meats were surface inoculated with $5 \log_{10}$ CFU/g, vacuum-packed and stored at 4.4°C. Products were kept for up to 12 weeks or until spoilage occurred.

The rate of growth was product and pH dependent. The most growth occurred when the pH was 5-6 or above, while little or no growth occurred near or below pH 5. The organism grew exceptionally well on chicken and turkey products (3-5 \log_{10} CFU/g increase within 4 weeks). These products had a pH of 6.3-6.5. Consistently high growth was observed within 6 weeks on all ham, bologna and bratwurst samples (3-4 \log_{10} CFU/g). Growth on weiners was variable, depending on the processor. Modest growth occurred on roast beef during the first two weeks of storage; growth then continued on one processor's product but numbers declined on another.

In slices of inoculated cooked beef, pork, chicken or turkey that were vacuum-packed and stored at either 0 or 5°C, decreased pH (6.0 – 5.0) and a_w (0.993 – 0.960 (NaCl)) increased the lag time and reduced the growth rate of *L. monocytogenes* (Duffy *et al.*, 1994). The type of meat had no effect on growth rate. Sodium nitrite also reduced the growth rate and increased the lag time, but sodium ascorbate had no significant effect on growth in the absence of nitrite.

Calculated growth rates from these experiments were compared to models derived from broth experiments. The times for a 3 \log_{10} CFU/g increase in numbers on vacuum packed meats and nitrite containing meats were up to 50% longer than those predicted from the model. Types of chilled vacuum packed processed meats were divided into risk categories: meat of a_w 0.968, pH 6.1-6.3, containing ascorbate and 50-70 mg/kg nitrite would belong to a relatively low risk category (a 3 log increase in numbers of *L. monocytogenes* would require storage at 5°C for at least 27 days) compared to a similar product of pH 6.5-6.7 containing no nitrite (8-10 days).

Two studies investigating the growth of *L. monocytogenes* on cooked beef were carried out in New Zealand in the early 1990s.

The first study examined cooked beef under refrigeration (5°C) and mild temperature abuse (10°C) conditions both aerobically and anaerobically (vacuum-packed) (Hudson and Mott, 1993). Growth rates were similar for *L. monocytogenes* under both atmospheres; at 5°C, it took 25 days to reach maximum numbers (7 \log_{10} CFU/g under aerobic and 6 \log_{10} CFU/g under anaerobic conditions). At 10°C, maximum numbers were reached after 8 days (aerobic 9 \log_{10} CFU/g and under vacuum 8 \log_{10} CFU/g). Other organisms growing with the *L. monocytogenes* were predominantly lactic acid bacteria. When these data were compared to the USDA predictive computer model, lag times were similar (within a few hours) but the model predicted growth rates that were faster than actual measurements, particularly under anaerobic conditions.

The second study examined the growth of *L. monocytogenes* on sliced roast beef (Hudson *et al.*, 1994). The beef was packaged under vacuum, or saturated carbon-dioxide (CO₂) controlled atmosphere conditions. At -1.5°C, the organism declined in numbers in the CO₂ packages but grew under vacuum conditions. At 3°C, growth occurred in both packaging systems, although the estimated lag time was longer and overall growth was retarded by about 1 \log_{10} CFU/g in the CO₂ package. The generation times were estimated to be about three times longer under the CO₂ conditions.

A survey of unpackaged ham in New Zealand (Cornelius *et al.*, 2008) produced representative data on the chemical composition of ham, such as the pH (mean 6.21, range 5.7-6.6), water activity (mean 0.994, standard deviation 0.006) and nitrite concentration (mean 29.6 mg/kg, standard deviation 27.6 mg/kg), which was entered into the Pathogen Modelling Programme 7.0 (PMP) (USDA, 2006). The model predicted that at 5°C, one generation of growth would take 13.2 hours. In contrast, the actual experiments found the three slowest growing *Listeria* spp. took between 72.3 and 150.5 hours to undergo one generation of growth. This indicates that the computer model overestimates growth rates.

L. monocytogenes has been described as a poor competitor in the presence of lactic acid bacteria; several studies have shown these micro-organisms can have an inhibiting effect. For example, in Norway a study found that indigenous lactic acid bacteria acted as a protective culture in cooked meat products that were sliced and either vacuum or gas packed (Bredholt *et al.*, 1999). Winkowski *et al.* describe the inhibitory effect of *Lactobacillus bavaricus* in three beef foods (Winkowski *et al.*, 1993). Production of bacteriocin rather than acidification was thought to cause the inhibition.

A study of unpackaged retail ham samples simulated domestic refrigerated storage (5°C for 7 days) and then tested the samples for *Listeria* spp. (Cornelius *et al.*, 2008). Thirteen samples (4.3%) contained *L. monocytogenes* and 13 other samples contained other *Listeria* spp. *Listeria* contaminated batches were further incubated at 5°C over approximately 3 weeks to assess the growth rate of natural contaminants. Growth occurred in 5 samples containing other *Listeria* spp. but the rate was slow (0.002 – 0.004 log₁₀ CFU/hour).

Thus while the growth of naturally occurring *L. monocytogenes* in foods has received comparatively little study; the data that does exist suggests that the growth rate is at times substantially slower than found from experiments with inoculated packs or forecast by predictive models (Cornelius *et al.*, 2008).

2.5 Exposure Assessment

2.5.1 Listeria in raw meat

While this Risk Profile is concerned with *L. monocytogenes* on processed ready-to-eat meat products, the microbial quality of the raw meat entering processing is important as:

- The higher the *Listeria* concentration on the raw material, the more effective the pathogen control processes need to be to reduce concentrations to acceptable levels; and
- High microbial loads on raw meat entering the process increase the potential for contamination of the processing environment and, if separation is not adequately maintained, the finished product.

Few studies have looked for *L. monocytogenes* in raw meat establishments in New Zealand. A survey of 100 bovine and 100 ovine carcasses in two North Island abattoirs used swab samples taken immediately after dressing (Hudson and Mott, 1994). Nearly every surface of the carcass, both exterior and cavity, were swabbed at the stage between the slaughterline and chiller. Only two samples contained *Listeria* spp.; *L. innocua* and *L. ivanovii*, both from ovine carcasses.

In a study of a South Island mutton slaughterhouse, seven *L. monocytogenes* isolates were obtained from 218 samples from ovine carcasses and the immediate environment (Pociecha *et al.*, 1991). No isolations of listeriae were made from freshly dressed carcasses (73 swab, 38 tissue cultures) or from meat contact surfaces (45). From 31 samples taken from cold rooms storing carcasses (at 5°C), four were positive for the organism. From 19 environmental samples (sheep faeces, hay, effluent mesh screen, soil, creek water and trough water) all were negative except for 1/4 hay samples and 2/7 soil samples. The survey confirmed that cold rooms, soil and fodder may be a source of contamination at the abattoir.

An earlier New Zealand study focused on cattle and sheep samples from slaughtering plants collected between January and May 1988 (Lowry and Tiong, 1988). Samples were obtained from whole carcasses, boned meat, cuts, offals, hides, pelts, viscera, equipment, work surfaces and effluent. Retail display meats of beef mince, pork cuts and poultry portions were also analysed. The results are shown in Table 2.

Table 2: New Zealand study; prevalence of *L. monocytogenes* in various red meat samples (1988)

Type of sample	No. of samples	No. (%) positive <i>L. monocytogenes</i>	No. (%) positive other <i>Listeria</i> spp. ¹
Beef/Lamb			
Beef (n=78)			
-boneless cuts	25	5 (20)	0
-offals	15	0	0
-hide pieces	23	4 (17)	2 (9)
-viscera	15*	0	0
Lamb (n=86)			
-boneless cuts	15	9 (60)	0
-carcass swabs	10	3 (30)	5 (50)
-offals	15	0	0
-pelt pieces	21	9 (43)	3 (14)
-viscera	25**	0	0
Environmental/effluent			
Beef cutting plant			
-work surfaces	15	4 (27)	2 (13)
-knives	5	2 (40)	1 (20)
Lamb cutting plant			
-work surfaces	15	11 (73)	8 (53)
-knives	5	2 (40)	
Retail display meats			
Beef mince	25	23 (92)	0
Pork cuts	25	17 (68)	0
Poultry portions	25	12 (48)	0

Source: Lowry and Tiong, 1988

¹ *L. ivanovii*, *L. innocua*, *L. welshimeri*

* 13 faecal contents, 2 mesenteric lymph nodes

** 20 faecal contents, 5 mesenteric lymph nodes

Failure to detect the organism from viscera contents or offals, but detection at almost the same incidence on hides/ pelts and on the meat samples led the authors to conclude that the animal hides/fleeces were a principal source of *L. monocytogenes* contamination of meat.

It is also clear that environmental contamination can be common in meat processing plants. However, the implications of this contamination for processed ready-to-eat meat products will depend to an extent on the microbial load that is transferred to the processed meat environment. The studies on raw meat in New Zealand did not include quantification of *Listeria*.

2.5.2 *Listeria* in processed ready-to-eat meat products

Ready-to-eat meat surveys of relatively small numbers of samples carried out in the early 1990s show a prevalence between 0 and 67% for *Listeria* spp., (mainly *L. innocua*) and between 0 and 50% for *L. monocytogenes*, depending on the type of meat (Table 3).

Table 3: Reported prevalence of *Listeria* in ready-to-eat (RTE) meat products in New Zealand

Meat product	Samples tested	No. of positive <i>L. monocytogenes</i> (%; 95 th percentile confidence interval)	No. of positive any <i>Listeria</i> species (%; 95 th percentile confidence interval)	Reference
RTE meat products cooked in packaging	54	NS	0.0	(Hudson <i>et al.</i> , 1991)
Fermented meats	39	NS	3 (7.7; 1.6-20.9)	(Hudson <i>et al.</i> , 1991)
Packaged RTE that had been handled (e.g. sliced)	36	NS	12 (33.0; 19-51)	(Hudson <i>et al.</i> , 1991)
Ready-to-eat meats from delicatessens	47	NS	18 (38.3; 25-54)	(Hudson <i>et al.</i> , 1991)
RTE pork products	34	1 (2.9; 0.1-15.3)	17 (50.0; 32-68)	(Hudson <i>et al.</i> , 1992)
RTE beef products	18	0	9 (50.0; 26-74)	(Hudson <i>et al.</i> , 1992)
RTE lamb products	3	0	2 (67.0; 9-99)	(Hudson <i>et al.</i> , 1992)
RTE mixed meat products	76	8 (10.5; 4.7-19.7)	17 (22.7; 13.6-33.4)	(Hudson <i>et al.</i> , 1992)
Jellied meats	6	3 (50.0; 12-89)	NS	(Ministry of Health, 1993)
Roast meats	6	2 (33.3; 4-78)	NS	(Ministry of Health, 1993)
Ham	32	12 (37.5; 21-56)	NS	(Ministry of Health, 1993)
Meat loaf	3	1 (33.3; 1-91)	NS	(Ministry of Health, 1993)
Corned beef/silverside	13	4 (30.8; 9-61)	NS	(Ministry of Health, 1993)
Luncheon	20	5 (25; 9-49)	NS	(Ministry of Health, 1993)
Pre cooked sausages	39	3 (7.7; 1.6-20.9)	NS	(Ministry of Health, 1993)

RTE = Ready-to-eat

NS = Not stated

The studies in Table 3 did not sample in proportion to consumption volumes in New Zealand. The two earlier surveys collected samples from supermarkets (Hudson *et al.*, 1991; Hudson *et al.*, 1992), while the 1993 survey collected up to five product types from 38 manufacturing premises in New Zealand (Ministry of Health, 1993).

Since the previous version of this Risk Profile (Lake *et al.*, 2002) surveys of New Zealand ham (pre-packaged and unpackaged) and pâté for *L. monocytogenes* have been reported.

According to food consumption information from the national nutrition surveys (Ministry of Health, 2003; Russell *et al.*, 1999), ham is the most commonly consumed processed ready-to-eat meat product in New Zealand, accounting for more than 50% of all servings consumed (see section 2.5.3.1). In a survey of pre-packaged ham samples, 104 samples from sixteen brands were tested (Wong *et al.*, 2005). All samples were held at 4°C and tested at the end of their shelf-life. Less than 7% of samples contained *Listeria*, and the single sample that contained *L. monocytogenes* was at a count of 50 CFU/g.

An unpackaged ham survey based the sampling on market share, i.e. 80% of samples were from supermarkets, 20% from other delicatessens (Cornelius *et al.*, 2008). Samples were collected in Auckland, Wellington and Christchurch over a period of two years. From the 301 samples tested, 10 (3.3%) were contaminated with *L. monocytogenes* only. Eight of the samples (2.7%) contained *L. innocua* only and 3 samples (1%) contained both *L. monocytogenes* and *L. innocua*. Overall, *L. monocytogenes* was present at a prevalence of 4.3% (95% CI 2.3% - 7.3%).

From the 13 samples positive for *L. monocytogenes*, counts were as follows;

- 1 sample: 1.6×10^3 CFU/g
- 1 sample: 1.5×10^2 CFU/g
- 3 samples: 50 CFU/g
- 8 samples: below level of enumeration (50 CFU/g) but positive in a presence/absence test (i.e. above 0.04 CFU/g (1 cell in 25g)).

Follow-up samples were collected from premises providing a positive *Listeria* result in the first test; “on most occasions” the second sample was negative. This may indicate sporadic rather than persistent contamination, heterogeneous distribution of *Listeria* in the product, or levels of *Listeria* at or near the detection limit of the analytical method.

An survey of pâté carried out in 2002 tested five samples from each of 60 lots of the nine brands that were on retail sale for *Listeria*, and quantified the positive samples (Wong *et al.*, 2005). The pâtés were sampled in Auckland and Christchurch (seven of the nine samples were of brands distributed in both cities). *Listeria* was detected in a single sample (0.3%, 95% confidence interval 0.0-1.8%). In this positive sample, the level of *L. monocytogenes* detected was 1700 CFU/g, and could have been even higher had not the high APC count of 10^8 CFU/g inhibited further growth. Six samples of pâté contained *Listeria* species other than *L. monocytogenes*. Two of these samples contained *L. welshimeri* (<50 and 450 CFU/g), and four had *L. innocua* (<50, 50, 200 and 400 CFU/g).

Available data of the prevalence of *Listeria* in ready-to-eat poultry products in New Zealand are summarised in Table 4.

Table 4: Reported prevalence of *Listeria* in ready-to-eat poultry products in New Zealand

Meat product	Samples tested	No. of positive <i>L. monocytogenes</i> (%; 95 th percentile confidence interval)	No. of positive any <i>Listeria</i> species (%; 95 th percentile confidence interval)	Reference
RTE Turkey products	6	0 (0.0, 0-46)	3 (50.0, 12-88)	(Hudson <i>et al.</i> , 1992)
RTE chicken products	16	2 (12.5, 2-38)	7 (43.8, 20-70)	(Hudson <i>et al.</i> , 1992)

RTE = Ready-to-eat

The data summarized in this section suggest that in ham, the most commonly consumed ready-to-eat meat covered by this Risk Profile, contamination with *L. monocytogenes* is common: 4% in unpackaged samples in the most recently reported (2008) survey. Results from earlier surveys reported in 1988 and 1993 have shown higher prevalences. The prevalence in pre-packaged ham was lower implying that contamination is occurring during post production handling. These data are consistent with the prevalence reported for ham in the UK, US and EU (see Appendix 1, Tables 16 and 17).

The survey of pâté involved 300 samples (which would have detected a prevalence of 1% or more with 95% confidence) only found one sample positive for *L. monocytogenes* (0.3%) suggesting a lower prevalence than for ham. The remaining data for other types of ready-to-eat meats are either more than ten years old or from surveys with insufficient numbers of samples to confidently estimate prevalence (although the finding that 2 of 16 ready-to-eat chicken products were positive is of concern).

There are insufficient data on the numbers of *L. monocytogenes* in positive samples to draw any conclusions.

2.5.3 Food consumption: Processed ready-to-eat meat products

The following information is taken from the 1997 New Zealand National Nutrition Survey (NNS) (Russell *et al.*, 1999) and the 2002 National Children's Nutrition Survey (CNS) (Ministry of Health, 2003). While the NNS and CNS are undoubtedly the best available source of data on daily levels of consumption of ready-to-eat meats, interpretation of some aspects of the data set can be problematic. Problems that arise include:

- Ready-to-eat meats, such as salami, may be eaten as purchased or may be included in a composite dish which is further heat processed.
- Some descriptors (roast beef, corned beef) may describe a ready-to-eat meat, or may describe a meat which undergoes or has undergone further heat processing in the domestic environment.
- Meats cooked in the home may, after a period of storage be eaten without further heat processing, for example, roast beef may be initially eaten as a hot roast, but may subsequently be eaten cold in sandwiches.

The NNS and CNS do not generally provide sufficient information to make clear judgment calls as to whether situations such as those described above may apply.

The following decision rules were applied to the current analysis:

- All instances of consumption of beef jerky, beef tongue, ham (including hoggett/mutton ham), luncheon, black pudding, brawn, lamb tongue, liverwurst, pâté, meat paste or salami were assumed to represent consumption of ready-to-eat meat.
- All consumption of meat as a component of sandwiches, filled rolls/croissants/bagels, or salads was assumed to represent consumption of ready-to-eat meat, unless otherwise specified.
- Consumption of corned meats, roasted meats or meatloaf when not associated with sandwiches, filled rolls/croissants/bagels, or salads was assumed **not** to represent consumption of ready-to-eat meat.

2.5.3.1 Proportion of population consuming processed ready-to-eat meat products

The qualitative food frequency questionnaire (QFFQ), delivered as part of the nutrition surveys, did not provide any insights on the frequency of consumption of processed ready-to-eat meat products. While respondents were asked how frequently they consumed bacon or ham, these foods were considered in aggregation.

Analysis of 24 hour dietary recall records from the nutrition surveys and using the decision rules outlined above revealed that 17.3% (95th percentile confidence interval 16.1-18.7%) of children (5-15 years) reported consumption of processed ready-to-eat meat products in the previous 24 hour period, while 18.0% (95th percentile confidence interval 16.9-19.1%) of adults (15+ years) reported consumption of this food type.

Servings of processed ready-to-eat meats are dominated by two product types, ham and luncheon meat, although the analysis is complicated by the use of the descriptor 'ham/bacon' for sandwich fillings. It has been assumed that these fillings are more likely to be ham. Ham constituted 53% of processed ready-to-eat meat product servings consumed by children and 63% of those consumed by adults, while luncheon made up 33% of child servings and 20% of adults. Salami was the next most consumed processed ready-to-eat meat product, constituting 4% of child servings and 8% of adult servings. This confirms that, despite the diversification in the range of ready-to-eat meat products on the market, those based on pig meat predominate.

2.5.3.2 Mean daily consumption of processed ready-to-eat meat products

Determination of consumption weights is complicated by the fact that processed ready-to-eat meats are often consumed as a component of a filled roll or sandwich and the nutrition surveys often only report the total weight of the sandwich. Based on analysis of representative dietary records, it was assumed that the meat component of a sandwich or filled roll would account for approximately 25% of the total weight.

The average daily consumption of processed ready-to-eat meat products was estimated to be 10.1 g/day for all children, taking into account both consumers and non-consumers of these products on the survey day. For only those respondents reporting consumption of processed

ready-to-eat meat products on the survey day (consumers) the mean was 58 g/day, the median 46 g/day and the 95th percentile 147 g/day.

Estimates are similar for adults, with the mean daily consumption for all respondents being 8.8 g/day, while for consumers only the mean is 49 g/day. The median and 95th percentile consumption levels for consumers only are 35 and 122 g/day, respectively.

2.5.3.3 *Serving sizes for processed ready-to-eat meat products*

Using the same assumptions outlined in the previous section for estimating the meat content of sandwiches and filled rolls, the following mean and percentile serving sizes were estimated for consumption of processed ready-to-eat meat products in New Zealand:

	Children	Adults
Mean (g)	45	43
Median (g)	29	28
95th Percentile (g)	112	109

Median daily consumption may represent one or more servings of ready-to-eat meats. The USDA risk assessment for *L. monocytogenes* determined median serving sizes (which may be equal to, or less than median daily intake) for four ready-to-eat meats (frankfurters, dry/semi-dry fermented sausages, deli meats, and pâté and meat spreads) as being in the range of 46-57 g/serving (USDA, 2003).

2.6 **Qualitative Estimate of Exposure**

2.6.1 Number of servings of ready-to-eat meat and serving size

2.6.1.1 *Total population*

From the NNS, 958 individual dietary records were deemed to represent consumption of a serving of RTE meat, while 736 records in the CNS related to consumption of ready-to-eat meat. The 2006 New Zealand census reported 3,096,273 people 16 years and older usually resident in New Zealand and 656,589 people aged 5-15 years (<http://www.stats.govt.nz/>). If it is assumed that children younger than one year will not eat ready-to-eat meats and that children 1-4 years (218,445 in 2006) will consume similar amounts of ready-to-eat meat to children 5-15 years and using survey populations of 4636 (NNS) and 3275 (CNS) :

$$\begin{aligned} \text{Annual number of servings (total population)} &= ((958 \times 3,096,273/4636) + (736 \times \\ &875,034/3275)) \times 365 \\ &= 3.05 \times 10^8 \text{ servings} \end{aligned}$$

This compares to 2.91×10^{10} servings of ready-to-eat meat calculated for the US population (USDA, 2001), based on a total population of 261,897,280 (1994-1996). These figures produce quite similar results for the number of servings per person per annum of 111 (US) and 87 (NZ).

2.6.1.2 Elderly population

From the NNS, 241 individual dietary records were deemed to represent consumption of a serving of ready-to-eat meat for an individual aged 60 years or more. A total of 1087 people aged 60 years or more completed dietary recall questionnaires as part of the NNS. According to the 2006 Census 675,225 New Zealanders were aged 60 years or more.

$$\begin{aligned}\text{Annual number of servings (elderly population)} &= 241 \times 675,225 / 1087 \times 365 \\ &= 5.46 \times 10^7 \text{ servings}\end{aligned}$$

2.6.1.3 Perinatal population

The assumptions made by the USDA to calculate the perinatal population were used to calculate the number of perinatal servings for pregnant women in the New Zealand population. This was done by multiplying the number of servings for the total population (see above) by the annual birth rate (for New Zealand; 64,040 in 2007 as a percentage of the 2007 total population gives a birth rate of 1.5%, the same as that used for the USDA calculations) and dividing by 12, to estimate the number of women in the last month of pregnancy.

$$\begin{aligned}\text{Annual number of servings (perinatal population)} &= 3.05 \times 10^8 \times 0.015 / 12 \\ &= 3.82 \times 10^5 \text{ servings}\end{aligned}$$

2.6.1.4 Intermediate population

The annual number of servings consumed by the balance of the population is calculated by subtracting the value for the elderly and perinatal population from the total population.

$$\text{Annual number of servings (intermediate population)} = 2.50 \times 10^8 \text{ servings}$$

Based on the data in the CNS and NNS databases the 50, 75, 95, and 99th percentile serving sizes for ready-to-eat meats in New Zealand were:

Percentile	NNS Serving size (g)	CNS Serving size (g)
50	28	29
75	50	56
95	109	112
99	287	189

For comparison, the USDA risk assessment determined the following serving sizes for the same percentiles (figures are averages for four types of ready-to-eat meat); 54 g, 85 g, 143 g, 274 g. These figures suggest that a median New Zealand serving size is somewhat smaller than the US equivalent.

2.6.2 Contamination frequency

Ready-to-eat meat surveys from the early 1990s suggest a highly variable contamination prevalence between products, although the rates determined from a positive result in a very small sample set can be misleading. More recent surveys from 2005 of the dominant

processed ready-to-eat meat type (ham), suggests a prevalence of 3.5% (14/405, 95th percentile confidence interval 1.9-5.7%).

For retail pâté, 1/300 (0.3%) were positive for *L. monocytogenes*. For ready-to-eat poultry products, 2/22 (9%) were positive for *L. monocytogenes*.

International data on the prevalence of *Listeria* in processed ready-to-eat meats (see Appendix 1) suggests a very high level of variability, making it difficult to determine ‘typical’ levels of contamination for particular meat types. However, large (>100 samples), recent (post 2000) studies summarized in Appendix 1 suggest that the prevalence of *Listeria monocytogenes* in processed ready-to-eat meats rarely exceeds 10%.

2.6.3 Predicted contamination level at retail

In the retail prepackaged ham survey, the one *L. monocytogenes* result enumerated to 50 CFU g⁻¹. In the unpackaged ham survey, the 13 positive *L. monocytogenes* results were enumerated as follows:

- 1 sample, the highest count 1.6 x 10³ CFU g⁻¹,
- 1 sample 1.5 x 10² CFU g⁻¹,
- 3 samples at 50 CFU g⁻¹,
- remaining samples 0.04 (1 cell in 25g) to 50 CFU g⁻¹ (A positive result from presence/absence test, count below level of enumeration).

In terms of pâté, the one positive sample was enumerated and found to contain 1700 CFU g⁻¹.

While some information is available on levels of *Listeria* in ready-to-eat meats associated with suspected foodborne illness incidents, this is not appropriate for assessing population level exposure.

2.6.4 Growth rate during storage and most likely storage time

Vacuum packaged ready-to-eat meats generally have shelf lives measured in weeks. Supermarkets also buy vacuum-packed ready-to-eat meat, which they slice and sell in their delicatessens. Therefore, even product sold with a short shelf life may have come from meat that has been stored for 2-3 months. This was the experience of the non-invasive listeriosis outbreak in New Zealand in 2000 (Sim *et al.*, 2002).

In New Zealand, a recent survey of domestic refrigerators found one third (43/127;34%) to be operating at a mean temperature above 6°C (Gilbert *et al.*, 2007a). A questionnaire administered across New Zealand on domestic meat and poultry handling found that 304/308 (98.7%) respondents stored cooked meats and cooked poultry refrigerated for 7 days or less (Gilbert *et al.*, 2007b).

Growth of *L. monocytogenes* in ready-to-eat meats is possible at refrigeration temperatures but is slowed by a number of factors (as described in Section 2.4.3.2) e.g. natural populations grow more slowly than inoculated populations, nitrite inhibits growth (and is present in the most common ready-to-eat meats considered in this Risk Profile (Thomson *et al.*, 2007), as does modified atmosphere packaging that includes carbon dioxide. It appears that unless temperature abuse (e.g. storage at 10°C or above) occurs then several weeks would be required to achieve multiple log₁₀ CFU/g increases in numbers.

2.6.5 Heat treatment

Not applicable to ready-to-eat meat products.

2.6.6 Exposure summary

The most commonly consumed ready-to-eat meat, ham, has a prevalence of *L. monocytogenes* that suggests approximately 3% of servings might be contaminated. This is a particular issue for unpackaged ham; the market share of this type of ham compared to pre-packaged ham is unknown. The prevalence of contamination of the other common types of ready-to-eat meats (luncheon, salami) is unknown. As these products are also commonly sold unpackaged there is the potential for similar contamination.

Given that *L. monocytogenes* is a common environmental contaminant it is perhaps not surprising that some contamination of such meats does occur. To estimate the risk, it would also be necessary to have data on the numbers of *L. monocytogenes* present, and the frequency of storage times and temperatures that might permit growth. Such data are lacking.

3 EVALUATION OF ADVERSE HEALTH EFFECTS

There are two types of disease which can occur after infection by *L. monocytogenes*; invasive and non-invasive. The invasive disease is called listeriosis and normally occurs in people with weakened immune systems. The non-invasive disease is usually called febrile gastroenteritis i.e. gastroenteritis associated with mild 'flu-like' symptoms, and can occur in healthy people if large numbers of *L. monocytogenes* cells are consumed.

Listeriosis is a notifiable disease in New Zealand, and it is generally assumed that the severity of the disease means that there are no unreported cases. However, the non-invasive febrile gastroenteritis form of infection is not notifiable, and the only information on its incidence comes from an outbreak

3.1 Listeriosis

To cause this disease, ingested *L. monocytogenes* cells penetrate the intestinal tissue and become exposed to phagocytic cells of the immune system. A portion of the *L. monocytogenes* cells survive and multiply within the host phagocytes. They then move throughout the host via blood or the lymphatic system.

The populations most at risk from this disease are the elderly, the immunocompromised, and the perinatal. Perinatal infections occur primarily as a result of transplacental transmission to the foetus following infection of the mother. The symptoms experienced by the mother are usually only a mild fever with slight gastroenteritis or flu-like symptoms. The perinatal group includes foetuses or neonates, and infection can occur before or after birth. Late-onset listeriosis results from infection of the infant during birth or up to 10 days after birth.

Incubation: 1-90 days, mean 30 days.

Symptoms: Include 'flu'-like symptoms (e.g. fever, headache), diarrhoea, vomiting. In perinatal cases clinical outcomes for the foetus or newborn include general septicaemia, intrauterine death, premature birth, stillbirth. In non-perinatal cases symptoms commonly include bacteraemia and meningitis.

Long term effects: In one outbreak neurological problems (cranial nerve palsies) developed in 30% of the survivors of meningitis. Pre-term infants may suffer from excess fluid in the brain and partial paralysis.

Treatment: *L. monocytogenes* is susceptible to a number of antibiotics, but penicillin and ampicillin optionally with an aminoglycoside (e.g. gentamicin) is considered to be the combination of choice.

3.2 Non-Invasive Febrile Gastroenteritis

The non-invasive form of listeriosis was recognised during the 1990s.

Incubation: 11 hours to 7 days, median 18 hours.

Symptoms: Diarrhoea, fever, muscle pain, headache, and less frequently with abdominal cramps and vomiting. Attack rate reported to be upwards of 74%.

Toxins: No toxins are produced in foods.

3.3 Dose Response

Analysis of animal trial and outbreak data for the dose-response relationship of invasive listeriosis has produced models for both “at risk” and “not at risk” populations. The very low probability of disease at low doses has prompted analysis that shows that foods containing more than 100 CFU/g were responsible for more than 99% of listeriosis cases (Chen *et al.*, 2003). Ingestion of large numbers of cells (10^7 or more) is necessary for *L. monocytogenes* to cause the febrile gastroenteritis version of listeriosis.

Further details are given in Appendix 2.

3.4 Adverse Health Effects in New Zealand

3.4.1 Incidence

Notification and mortality data from the EpiSurv database for listeriosis for the years 1990 to 2008 are given in Table 5. It is important to note that these cases are not associated with any specific transmission vehicle.

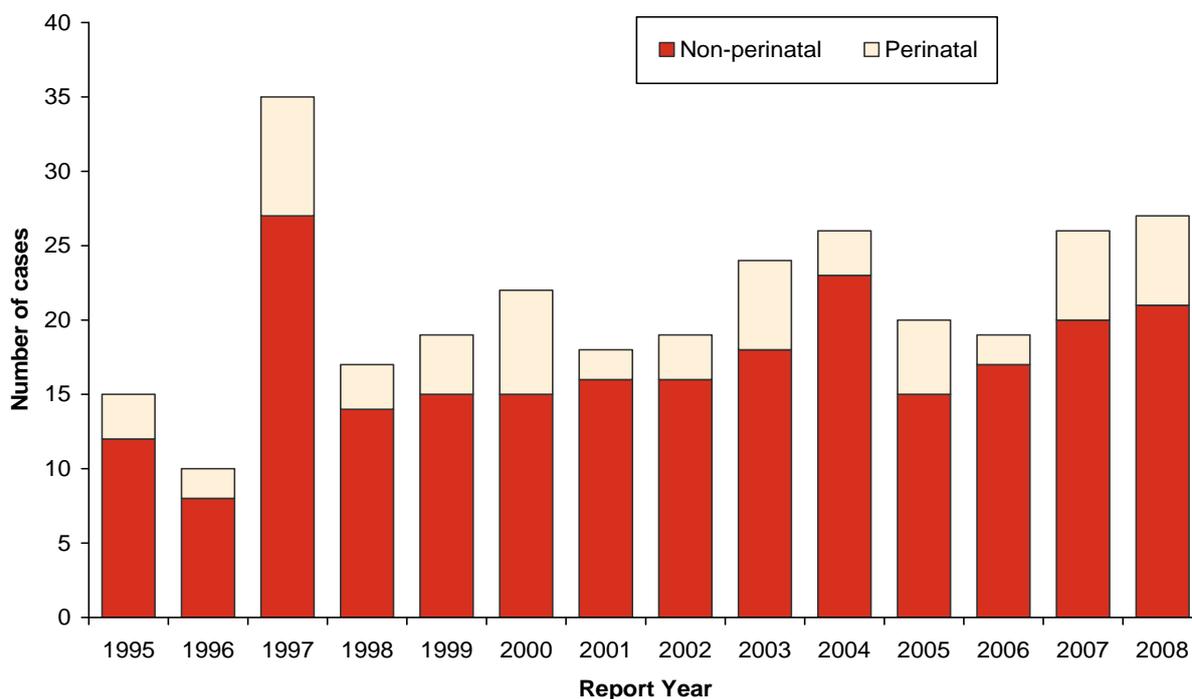
Table 5: Number of reported cases of invasive listeriosis and mortality from 1997 to 2008 (Williman *et al.*, 2009)

Year	Listeriosis cases	Deaths (perinatal)	Deaths (non-perinatal)	Rate / 100,000
1997	35	6	2	1.0
1998	17	0	0	0.5
1999	19	2	1	0.5
2000	22	4	2	0.6
2001	18	1	1	0.5
2002	19	3	0	0.5
2003	24	2	2	0.6
2004	26	2	3	0.7
2005	20	4	3	0.5
2006	19	1	0	0.5
2007	26	2	2	0.6
2008	27	2	3	0.6

NA = Not Available

Figure 2 shows a graphical representation of annual case numbers of reported invasive listeriosis with the proportions of perinatal and non-perinatal cases identified.

Figure 2: Invasive listeriosis notifications by year 1995 – 2008



Reproduced from (Williman *et al.*, 2009)

3.4.2 Clinical consequences of *Listeria* infection

Listeriosis has a high proportion of serious outcomes i.e. hospitalisation and death. Hospitalisation and fatality rates for notified cases of listeriosis in New Zealand during the period 1997-2008 are given in Table 6. These outcomes are not always reported for each case, so percentages are expressed in terms of the number of cases for which outcomes are known.

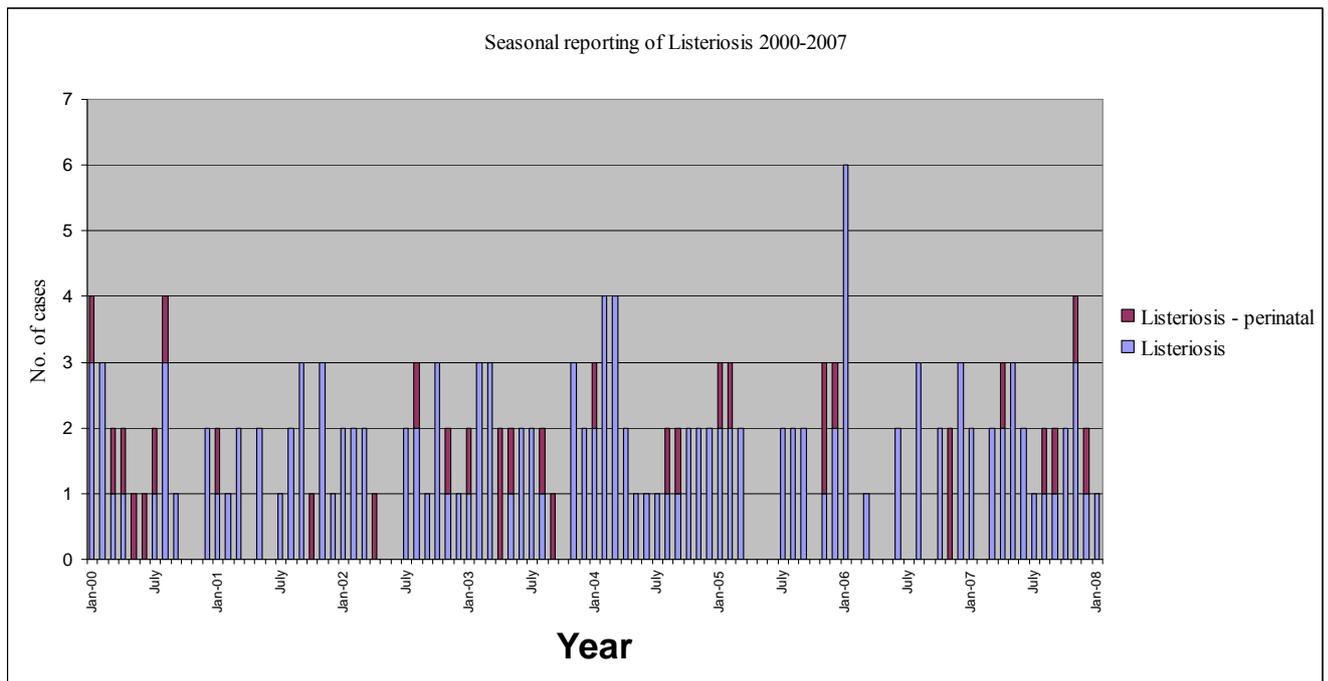
Table 6: Outcome data for listeriosis in New Zealand, 1997 - 2008

Year	Hospitalised cases	Fatalities	Reference
1997	33/33 (100%)	8/35 (22.9%)	(ESR, 1998)
1998	16/16 (100%)	0/17 (0.0%)	(Perks <i>et al.</i> , 1999)
1999	18/19 (94.7%)	3/19 (15.8%)	(Kieft <i>et al.</i> , 2000)
2000	22/22 (100%)	6/22 (27.3%)	(Lopez <i>et al.</i> , 2001)
2001	17/18 (94.4%)	2/18 (11.1%)	(Sneyd <i>et al.</i> , 2002)
2002	13/13 (100%)	3/19 (15.8%)	(Sneyd and Baker, 2003)
2003	22/22 (100%)	4/24 (16.7%)	(ESR, 2004)
2004	25/26 (96%)	5/26 (19.2%)	(ESR, 2005)
2005	13/15 (86.7%)	1/15 (6.7%)	(ESR, 2006)
2006	16/17 (94.1%)	1/17 (5.9%)	(ESR, 2007)
2007	19/19 (100%)	4/26 (15.3%)	(ESR, 2008)
2008	17/20 (85%)	5/27 (18.5%)	(ESR, 2009)

Over the period 1997-2008, the mean annual hospitalisation rate was 96.3% with a fatality rate of 15.8%. Estimates for the United States are similar to the New Zealand data, with 92% of cases hospitalised, and 20% of cases resulting in death (Mead *et al.*, 1999). However, part of the derivation of the US figures included a doubling of reported hospitalised cases and mortality figures, to account for under-reporting.

Figure 3 shows the number of cases reported by month from January 2000 – January 2008. This shows no seasonal trend in the notifications in New Zealand, although the numbers are small. In the USA, 2000 listeriosis cases were analysed by the CDC and no seasonal trends were detected (Gombas *et al.*, 2003).

Figure 3: Number of listeriosis cases reported by month in New Zealand (January 2000 – January 2008)



3.4.3 Outbreaks

Outbreaks of infection with *L. monocytogenes* in New Zealand are rare. From 1997 to 2006 only three have been reported. The first outbreak was caused by smoked mussels (Brett *et al.*, 1998) and is therefore outside the scope of this Risk Profile.

An outbreak of non-invasive febrile-gastroenteritis occurred in early 2000 (Sim *et al.*, 2002; Whyte, 2000). The outbreak involved 28 people in five separate incidents who became sick after eating corned silverside and ham from the same manufacturer. Numbers of *Listeria* cells were high with $1.8 \times 10^7/g$ being counted in the ham. The ham involved was produced in December but not eaten until the following March, representing a two to three month shelf life. All isolates were of the same serotype and pulsed field gel electrophoresis (PFGE) type (96/2).

This illustrates the risk from a contaminated product with a long shelf life, where the number of *L. monocytogenes* may increase greatly during the shelf life of the product even under correct refrigeration. If the product is temperature abused then the numbers may be higher.

The outbreak itself was recorded as one event of 7 people consuming corned silverside and another of 16 cases (now known to have been 21 cases) consuming ham. The other three cases were reported as individual cases under the suspect foodborne illness investigation programme.

In 1997, a record 35 cases of invasive listeriosis were notified. This was mainly due to an outbreak of a distinct strain (serotype O1/2a, phage type 1967 881, RFLP type 96/2). Between February and June 1997 there were 17 cases affected by this strain. No specific source was implicated in the outbreak (Anonymous, 1998a).

3.5 High Risk Groups in the New Zealand Population

While anyone can become infected by *L. monocytogenes*, it is generally recognised that there are some high risk groups in the population (Sutherland *et al.*, 2003). High risk groups for listeriosis include pregnant women and their foetuses, neonates, the elderly, and adults with a compromised immune system e.g. renal transplant patients, patients on corticosteroid treatment, and HIV/AIDS patients. New Zealand notification data supports the assertion that these groups are at greater risk, with the highest age specific listeriosis rates in the less than one year and the greater than 70 years age groups (Williman *et al.*, 2009). An underlying illness is the most commonly reported risk factor for invasive listeriosis in New Zealand (Williman *et al.*, 2009).

The potential impact of underlying conditions on the risk of invasive listeriosis is highlighted in Table 7, reproduced from the FAO/WHO risk assessment (FAO/WHO, 2004)

Table 7: Relative susceptibility to *L. monocytogenes* for certain underlying health conditions

Condition	Relative susceptibility
Transplant	2584
Cancer- blood	1364
AIDS	865
Dialysis	476
Cancer- pulmonary	229
Cancer- gastrointestinal and liver	211
Non-cancer liver disease	143
Cancer – bladder and prostate	112
Cancer - gynaecological	66
Diabetes, insulin dependent	30
Diabetes, non-insulin dependent	25
Alcoholism	18
Over 65 years old, no other condition	7.5
Less than 65 years, no other condition (reference population)	1

FAO/WHO (2004)

The following sections provide information on the New Zealand population of these groups.

3.5.1 Perinatal population

Live births data for the 2007 Calendar year were 64,040 (<http://www.stats.govt.nz/>) from a population of 4,228,300.

Births were spread evenly throughout the year, but were strongly weighted towards the Northern areas of New Zealand. This total shows an increase compared to the results of the 2006 Census, which reported 56,631 New Zealanders under the age of one year (usually resident) on Census night. The under 1-year olds represent 1.4% of the total New Zealand population.

In 2006, there were 19 cases of listeriosis of which 2 were perinatal. This equates to a rate of approximately 3.5 cases/100,000/year in the perinatal population.

The corresponding figures for 2007 are 25 cases of which 5 were perinatal, yielding a rate of 7.8 cases/100,000/year in the perinatal population.

3.5.2 Elderly population

According to the 2006 Census of New Zealand 675,225 New Zealander residents were aged 60 years or over. This is 16.8% of the total population. The population 80 years and over is 128,913 (3.2% of the population) (<http://www.stats.govt.nz/>).

3.5.3 Immune compromised

AIDS: At the end of 2006, 29 notifications of AIDS were reported. (<http://www.moh.govt.nz/aids.html>). The total number of people notified with AIDS to the end of June 2007 is 931 (842 males and 89 females).

HIV: Between 1985, when records began, and December 2005, 1608 men and 261 women have tested positive for HIV. The year 2005 saw the highest number of HIV diagnoses at 218. In 2006, the figure was 204 people in New Zealand newly diagnosed with HIV, the highest since 1985.

Cancer: The most recently available statistics on the incidence of cancer and cancer mortality in New Zealand are from 2003 (website accessed 20.02.08 [http://www.nzhis.govt.nz/moh.nsf/pagesns/32/\\$File/cancer2003.xls](http://www.nzhis.govt.nz/moh.nsf/pagesns/32/$File/cancer2003.xls)).

In 2003, 18,943 new cases of cancer were registered, made up of 9,856 males and 8,730 females. The overall cancer rate per 100,000 was 317.8. During the same period, mortality due to cancer was 8,027 made up of 4,292 males and 3,735 females. It is uncertain what proportion of the New Zealand population is suffering from cancer at any particular time.

Recipients of organ or tissue donations: The New Zealand Organ Donation website gives the following numbers for transplants performed in 2006; kidney (deceased donor) 41; kidney (living donor) 47, liver (deceased donor) 36, liver (living donor) 4, heart 8, lungs 10, pancreas 6 (<http://www.donor.co.nz> accessed 20.02.08). It appears likely that the total New Zealand

population of surviving major organ transplant recipients is less than 2,000 people (0.05% of the total population).

3.6 Adverse Health Effects Overseas

Overseas information on adverse health effects due to invasive listeriosis in general, outbreaks due to ready-to-eat meats and findings from case-control studies are included in Appendix 2, section 2.2.

3.7 Health Burden due to Invasive Listeriosis

The annual economic cost to New Zealand of cases of invasive listeriosis caused by foodborne transmission has been estimated as \$2.5 million, with \$2.3 million of this due to perinatal cases (Cressey and Lake, 2008). The number of cases and outcomes used for this estimate was based on an notification and hospitalisation data for 2005 (Cressey and Lake, 2008). The estimated value includes direct and indirect medical costs, and productivity losses due to cases and caregivers not attending work.

This cost of illness figure used an estimate for the proportion of invasive listeriosis cases due to foodborne transmission of 85%, derived from an expert elicitation exercise carried out in 2005 (Lake *et al.*, 2009). In the USA, foodborne transmission of listeriosis has been estimated as 85-95% (Buzby *et al.*, 1996) and 99% (Mead *et al.*, 1999) of all cases, while in the Netherlands a lower proportion of foodborne cases has been estimated (69%) (Havelaar *et al.*, 2008).

An estimate of the burden of foodborne disease for New Zealand (Cressey and Lake, 2007) includes an estimate for foodborne invasive perinatal listeriosis of 195 disability adjusted life years (DALYs). This placed foodborne perinatal listeriosis third on the list for foodborne disease burden (after campylobacteriosis and norovirus infection). A separate estimate for foodborne acquired listeriosis (i.e. non-perinatal) was much smaller, at 22 DALYs. This reflects the importance of infant mortality on these estimates.

The burden of disease due to invasive listeriosis, estimated either in terms of cost of illness or DALYs, is characterised by the high proportion of fatalities. For most foodborne diseases the burden due to morbidity is the greater part of the burden of disease estimate. However, for perinatal invasive listeriosis mortality accounts for more than 99% of the DALY estimate. Similarly, for cost of illness estimates the largest component is due to the productivity losses due to permanent removal from the workforce through mortality.

These estimates cover all potential food vehicles. The expert elicitation process resulted in an estimate of 54% of foodborne invasive listeriosis cases resulting from transmission via processed ready-to-eat meat products (Cressey and Lake, 2005)

Health burden estimates for non-invasive listeriosis (febrile gastroenteritis) have not been made in New Zealand or overseas.

4 EVALUATION OF RISK

4.1 Existing Risk Assessments

Published risk assessments from FAO/WHO (ready-to-eat foods), USA (ready-to-eat foods), Australia (smallgoods and processed meat products), and Canada (pâté and semi-soft cheese) have been summarised in Appendix 3. The most relevant to this Risk Profile are those for the USA and Australia.

The USA risk assessment ranked 23 ready-to-eat foods according to the risk of invasive listeriosis, and deli meats, pâté and meat spread, and dry/semi dry fermented sausages were ranked 1, 3, and 15 respectively (the other ready-to-eat meats were frankfurters which are less common in New Zealand). More detailed examination of the risk from deli meats attributed more than 80% to deli meats sliced or packaged at retail rather than at processing plants.

In the Australian risk assessment, processed (deli) meats were estimated to cause approximately 40% of Australia's listeriosis cases annually. This was partly based on a prevalence of contamination of 4.77% in such meats.

4.2 Estimation of Risk for New Zealand

4.2.1 Risks associated with processed ready-to-eat meats

The number of invasive listeriosis cases reported every year is very small relative to other forms of potentially foodborne disease. The importance and high burden of the disease derives from the high proportion of serious outcomes for infants and foetuses.

The rate of reported invasive listeriosis in New Zealand has been static for many years, and is similar to that found in comparable countries. As in other countries, most cases are sporadic, with outbreaks being rare. There have been only two reported outbreaks involving *L. monocytogenes* in New Zealand; one associated with smoked mussels, and one of unknown source producing mainly non-perinatal cases. Both of these outbreaks involved the invasive form of listeriosis.

Analysis of Episurv data found 174 cases of listeriosis notified between 2000 and 2007. Foodstuffs implicated (but not confirmed) were noted for 16 cases (9%) which demonstrates the difficulty in determining the source of infection.

The incidence of non-invasive disease from *L. monocytogenes* infection in New Zealand is unknown. It is not normal practice for clinical laboratories to examine faecal specimens from cases of gastrointestinal disease for the presence of *L. monocytogenes* and it might be that more outbreaks will be reported as this form of the disease gains recognition. Two New Zealand outbreaks of non-invasive listeriosis have been reported (actually both were from the same incident) and involved cooked ready-to-eat meat products (Sim *et al.*, 2002).

L. monocytogenes has been detected in a range of New Zealand ready-to-eat meats; the best data are for the most commonly consumed ready-to-eat meat i.e. ham, with a prevalence of approximately 3.5%.

The median daily consumption of ready-to-eat meats in New Zealand is similar to that for Australia, and somewhat lower than the amounts consumed in the USA. Although the data on imported processed meat products (mostly from Australia) do not clearly identify ready-to-eat meats as such, it appears that the large majority of ready-to-eat meats consumed in New Zealand are produced locally. Ham is the most commonly consumed type of ready-to-eat meat, followed by luncheon meat and corned beef. These would be included in the category of “deli meats” ranked first for relative risk of listeriosis in the USDA risk assessment. The US risk assessment has also attributed most of the risk from deli meats to those sliced or packaged at retail; this is consistent with the New Zealand surveys that found a higher prevalence of *L. monocytogenes* in this type of ham sample, compared to pre-packaged ham.

It is very difficult, if not impossible, to completely eliminate *Listeria* from food processing environments (Sutherland *et al.*, 2003). Effective Food Control Plans for manufacturers of ready-to-eat meats will be an essential part of risk management, and transition of this sector to requirements of the new domestic food legislation is anticipated in approximately 2011.

A quantitative risk assessment performed in Australia, where contamination and consumption prevalences are similar to New Zealand concluded that ready-to-eat meats were responsible for up to 40% of cases of listeriosis (Ross *et al.*, 2009a), based on a prevalence of contamination similar to that found in New Zealand. This attribution is in good agreement with the results of an expert elicitation for New Zealand, which estimated that 85% of listeriosis was foodborne and of this foodborne component 54% was due to transmission via processed ready-to-eat meats (Cressey and Lake, 2005).

In their statement of intent the NZFSA have provided an indicator for listeriosis of “no increase in the foodborne component with increasing range of foods available to the consumer”. The data from New Zealand, and risk assessments from the USA and Australia indicate that maintained or improved risk management for *L. monocytogenes* in processed ready-to-eat meats would be an important contribution to achieving this objective.

The burden of illness analysis indicates that principal target for risk management would be pregnant women; a recent study in New Zealand (Rungan and Badkar, 2005) indicates that although awareness of risk is high amongst this group, avoidance of high risk foods is less than ideal. This reinforces the need for preventive measures in the manufacturing sector.

In response to the risk management questions stated in Section 1.1, it is not possible to determine whether the level of risk has changed since the previous Risk Profile on this food/hazard combination. However, the quality of information has certainly improved markedly.

4.2.2 Risks associated with other foods

Analysis of Episurv data found 174 cases of listeriosis notified between 2000 and 2007. Foodstuffs implicated (but not confirmed) were noted for 16 cases (9%) which demonstrates the difficulty in investigating the sources of infection. In particular, obtaining food histories and samples of suspected food for testing is challenging due to the long incubation period (1 to 90 days). Of these 16 cases, half were perinatal. Foods reported eaten by the mothers were raw seafood and salad, raw fish, mussels, Korean cabbage dish (kimji), cold beef satay and vegetable salad, Christmas ham, Chinese pork buns and fish. For the other eight non-

perinatal listeriosis cases, foodstuffs reported as eaten were unwashed vegetables, stir-fried meat chub, soft cheeses, yoghurt, processed meats, smoked chicken from a delicatessen, salad cross contaminated with raw chicken, homemade yoghurt and deli-products; corned beef and cold sliced meats.

Listeriosis is considered to be primarily a foodborne disease. Aside from ready-to-eat meats, the USDA risk assessment also listed high relative risks of listeriosis for the fresh soft cheese, smoked seafood, cooked ready-to-eat crustaceans and deli salads (USDA, 2000). Non-reheated frankfurters were also ranked highly for relative risk in the US; it is unlikely that this food is widely consumed in New Zealand, although saveloys and cocktail sausages may be eaten without reheating prior to consumption.

4.2.3 Risk assessment options

A quantitative risk assessment (QRA) for *L. monocytogenes* in ready-to-eat meats is now possible, given the additional prevalence data available, and an Australian prototype.

4.3 Data Gaps

The data gaps identified by this Risk Profile are:

- Incidence of the non-invasive form of listeriosis in New Zealand
- Data on degree of implementation and effectiveness of HACCP based food safety plans by the ready-to-eat meat food sector.
- Prevalence and quantitative data on a wider range of processed ready-to-eat meat products.
- Times and temperatures of storage (both at retail and domestically) for ready-to-eat meat products.
- More up-to-date food consumption information to support the perceived emergence of a wider range of processed ready-to-eat meat products. The results of the latest National Nutrition Survey, due in 2011, will go some way to addressing this data gap.

5 AVAILABILITY OF CONTROL MEASURES

5.1 Risk Management Strategy

In March 2009 NZFSA released their *Listeria monocytogenes* Risk Management Strategy 2008-2013:

<http://www.nzfsa.govt.nz/foodborne-illness/listeria/strategy.htm>

This document states that the strategy will:

- Ensure that risk management options for the control of *L. monocytogenes* are effective and applied consistently across all food businesses;
- Take account of international developments in *L. monocytogenes* risk management through involvement in international fora and collaborations;
- Provide enhanced and effective information to all stakeholders for reducing the potential for *L. monocytogenes* contamination of food and exposure of consumers to potentially contaminated food;
- Document a process that will monitor and review progress of the strategy to meet the SOI (Statement of Intent) performance target; and
- Identify and prioritise research needed to inform and support *L. monocytogenes* risk management options applied and proposed.

The SOI performance target is “no increase in reported incidence of foodborne listeriosis after five years”.

The objectives of the strategy are:

- To achieve no increase in human foodborne listeriosis cases;
- To engage with industry, other stakeholders and consumers in order to ensure that any outcomes developed are practical, feasible and cost effective;
- To effectively communicate the strategy and outcomes to all stakeholders (including consumers);
- To make well informed risk management decisions on appropriate control measures and their implementation; and
- To design and implement an ongoing monitoring and review programme to assess the effectiveness of risk management decisions.

5.2 Regulatory Controls

This section collates information on the regulatory regimes in place in New Zealand. Supplemental information on regulatory controls overseas is given in Appendix 4.

Shelf lives of ready-to-eat meats are determined by the food industry. A guide to calculating the shelf life of foods has been published by the NZFSA (NZFSA, 2005) but decisions on individual products are made by the manufacturer or retailer.

5.2.1 Australia New Zealand Food Standards Code

The joint Australia New Zealand Food Standards Code includes standards related to the composition of processed ready-to-eat meats, including microbiological limits (FSANZ, 2009).

Standard 1.3.1 specifies the food additives that are permitted in certain classes of meat products. Permitted additives are mainly antimicrobials/preservatives, specifically nitrite (sodium and potassium salts), nitrate (sodium and potassium salts), sorbic acid (sodium, potassium and calcium salts) and natamycin.

Standard 1.6.1 elaborates enforceable microbiological criteria for three categories of processed ready-to-eat meats; packaged cooked cured/salted meat, packaged heat treated meat paste and packed heat treated pâté, and all comminuted fermented meat which has not been cooked during the production process. Only the former two have microbiological criteria for *L. monocytogenes*, with a requirement that the organism not be detected in any of five 25 g samples (n = 5, c = 0, m = 0).

FSANZ have also produced a guide to accompany Standard 1.6.1 (FSANZ, 2001b). The guide includes additional material on sampling and testing methods.

Standard 2.2.1 contains compositional requirement for meat products, including required minimum meat contents. There are also two relevant labeling requirement for mandatory declaration of the presence of offal in meat products and mandatory labeling of fermented comminuted processed meat or fermented comminuted manufactured meat to indicate whether it is ‘not heat treated’, ‘heat treated’ or ‘cooked’.

5.2.2 Food Act and Food Hygiene Regulations

Historically, food premises have been inspected against the Food Hygiene Regulations 1974. Since 1996, there has been an option to develop a Food Safety Programme (FSP) – based on HACCP principles - which exempts the food business from the 1974 Regulations. A FSP is registered under the Food Act 1981. The process is applicable to any size of type of food business in New Zealand.

A long term review of the domestic food regulatory regime in New Zealand is underway by the NZFSA. Termed the Domestic Food Review (DFR), one of the proposals is the introduction of Food Control Plans (FCPs) to supercede the Food Safety Programme regime. Alternative arrangements would account for those businesses already with HACCP based systems in place such as Risk Management Plans (RMP).

Manufacturers of ready-to-eat meat products have been identified as a food sector for which custom made registered FCPS will be required in Year 3 of the transition to the new legislation (NZFSA, 2006).

5.2.3 Animal Products Act and Risk Management Plans

The [Animal Products Act 1999](#) reforms the New Zealand law that regulates 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](#).

All animal product primary processing businesses, except those exempt under the Act or under the [Animal Products \(Exemptions and Inclusions\) Order 2000](#), must have a Risk Management Programme (RMP).

An RMP is a documented programme to identify and manage biological, chemical and physical hazards and is based on the principles of Hazard Analysis and Critical Control Point (HACCP). RMPs are designed by individual businesses for the animal materials used, the processes performed and the product range produced.

Types of businesses that would have an RMP include primary processors of animal material, secondary processors of animal products (intended for human consumption) and retail butchers who are dual operator butchers (DOBs).

5.2.4 Codes of Practice

In addition to industry initiatives, NZFSA has developed a Code of Practice for production of processed meats, which was the subject of a consultation process during 2009 (<http://www.nzfsa.govt.nz/consultation/processed-meat-cop-part1-4/index.htm>). It is envisaged that this will be used by processors operating a Food Safety Plan under the Food Act 1981, those operating a Risk Management Plan under the Animal Products Act 1999, and those operating under the Food Hygiene Regulation 1974.

The draft Code includes provision for an environmental monitoring programme for *Listeria*. It also states that “Cooked cured/salted meat products must meet the microbiological limits given in the Food Standards Code, Standard 1.6.1.” (see below) and “When cooking is used to control pathogens in ready-to-eat (RTE) products, the cooking process must achieve a 6 decimal reduction of *Listeria monocytogenes* (a 6D process).” For such cooking, times and temperatures are recommended, although alternative approaches may be used provided they are validated by the processor and approved by the NZFSA. The HACCP plans included with the consultation documents specifically address the potential for *Listeria* contamination during processing.

5.2.5 Microbiological criteria

An important issue for food manufacturers and regulators is whether there should be a zero tolerance for the presence of *L. monocytogenes* in ready-to-eat foods, or whether a low level (usually 100 CFU/g) is tolerable in certain foods where growth of the bacterium is unlikely (Codex Alimentarius Commission, 2009).

A number of documents providing guidance around microbiological suitability of ready-to-eat foods have been published and are recommended for use by Regulators and Industry throughout New Zealand and Australia.

These include:

- The Guide to Standard 1.6.1 (FSANZ, 2001b)
- The Microbiological Reference Criteria for Food (Ministry of Health, 1995)

- Guidelines for the Microbiological Examination of Ready-to-eat Foods (FSANZ, 2001a)

The above documents provide thorough guidance for food businesses and “Regulators may also use the limits contained in these documents to interpret the results of microbiological testing and use these results” (NZFSA, 2008).

Broadly, there is a requirement that all ready-to-eat foods (including cooked meals, cooked meats and their products, cooked seafoods and their products, seafood products that are likely to be consumed in that state, prepared desserts and bakery products containing cream or other fillings of high water activity and dairy products including soft cheeses) and food produced by a step which is capable of achieving a *Listeria*-free product meet a zero tolerance, i.e.

L. monocytogenes /25g n=5, c=0, m=0

The 1995 Ministry of Health document does not require zero tolerance in the following foods:

- raw fruits, vegetables, meats and seafoods
- foods produced in accordance with Good Manufacturing Practice (GMP) that will not support the growth of *L. monocytogenes*, i.e. have a pH <4.6 or >9.0, and/or a_w <0.9, and/or are stored or displayed below 1°C
- other foods produced in accordance with good manufacturing practice recommended for consumption within four days of manufacture and clearly labelled as such.

Importantly, the Guidelines for the Microbiological Examination of Ready-to-eat Foods (FSANZ, 2001a) advise that detection of *L. monocytogenes* in foods prepared specifically for ‘at risk’ populations should be considered “potentially hazardous”. These guidelines apply only at the end of production or at the wholesale stage of distribution (Table 8).

Table 8: FSANZ Guidelines for *L. monocytogenes* in ready-to-eat foods

Test	Microbiological quality (CFU per gram unless other stated)			
	Satisfactory	Marginal	Unsatisfactory	Potentially hazardous
<i>L. monocytogenes</i>	Not detected in 25g	Detected but <10 ² *		>10 ²

* Foods with a long shelf life stored under refrigeration should have no *L. monocytogenes* detected in 25g
Source: Guidelines for the microbiological examination of ready-to-eat foods (December 2001:6)

5.2.6 Industry controls

5.2.6.1 *Pork Quality Improvement Process*

The New Zealand Pork Industry Board has implemented the Pork Quality Improvement Process (PQIP). This is a New Zealand developed tool to assist processed meat manufacturing plants to apply HACCP principles to their operation and has been approved as a Code of Practice (COP). The PQIP covers most of the ready-to-eat meat products currently on the market, apart from uncooked fermented comminuted meat (UCFM) products such as mettwurst and some salamis. However, an additional chapter covering UCFM issues was

added in Revision 1, on 1 July 2004 and is pending approval by the NZFSA as a COP, although the requirements for water activity and other controls have been approved already.

In 1999 a survey of the manufacturing practices of some of the larger salami processors in New Zealand was conducted on behalf of the Ministry of Health with the support of the Pork Industry Board (Hasell, 2000). It was found that while HACCP based food safety programmes were not in evidence in the major companies producing raw comminuted meat products in New Zealand, all the companies surveyed had much of their systems documented and were working towards the adoption of HACCP.

It was recommended that the industry should be supported in their initiative to develop food safety programmes. Once these are available, it was recommended that the Ministry of Health consider making HACCP based food safety programmes compulsory for the manufacturers of uncooked meat products.

It seems likely that HACCP based control programmes have been implemented across the meat processing industry; however, it would be useful to confirm this with a survey that covered not just salami manufacturers.

5.3 Risk Communication

Education is currently an actively used form of risk management, especially for pregnant women. Direct education campaigns by the NZFSA about the risk of listeriosis to pregnant women are already in place (<http://www.nzfsa.govt.nz/consumers/food-safety-topics/foodborne-illnesses/low-immunity/lowimmunity.pdf>).

The effectiveness of risk communication about *Listeria* was assessed in a survey of 100 pregnant and postnatal women attending antenatal clinics or resident at Middlemore hospital (Rungan and Badkar, 2005). A questionnaire was administered one-on-one with demographic information collected first. Then seven high-*Listeria* risk (HLR) and seven low-*Listeria* risk foods were randomly listed and respondents were asked to identify the high-risk foods. In addition, the women were asked whether they consumed such foods (it is not clear from the paper whether this means while pregnant). The results found 58% (95% CI 48.24-67.24) had been given information on *Listeria*. Table 9 summarises the results for five of the foodstuffs. The figures in brackets are results of a similar study in Australia (Bondarianzadeh *et al.*, 2007).

Table 9: Results from a *Listeria* spp. knowledge questionnaire, Middlemore hospital

Foodstuff	Percentage identified as HLR (Australia % in brackets)	Consumed
Raw seafood	92	36
Cold cooked meats	96 (64)	45 (56)
Soft cheese	67 (81)	12
Cold cooked fish and reheated takeaways	95	15-27
Coleslaw	72 (50)	62

In total, 26% of the respondents fully understood which foods to avoid whilst pregnant but up to 62% of women did not actually avoid eating HLR foods when pregnant.

Research in New South Wales regarding *Listeria* education in pregnancy highlighted some inadequacies in risk communication in Australia (Bondarianzadeh *et al.*, 2007). From 586 respondents at antenatal clinics, 74% indicated they were concerned about their food safety during pregnancy, although only 59% have received information on this topic. Women's social network was the most common source of *Listeria* information while 27% obtained advice from pamphlets. Most respondents (66%) knew contaminated food transmitted the infection with 86% knowing that food-borne illness could be potentially dangerous for the foetus. Overall, only 29% of respondents said they had enough information on listeriosis.

Given a list of food items to be avoided because of *Listeria*, 81% identified soft cheeses as a HLR. Chicken liver pâté and deli meats from a deli counter were chosen by 68% and 64% respondents respectively. Only half of respondents identified cold smoked salmon and coleslaw from a salad bar as being a *Listeria* risk. A food not associated with *Listeria* (hot takeaway chicken portions) was selected as a risk by 72% of respondents. Overall analysis found that 57% had incomplete knowledge of HLR foods. In terms of foods consumed during the pregnancy, 43% reporting eating cold deli meats regularly (once a fortnight or more) while 13% reported consumption frequently (daily to 2-3 times a week). For pâté/dips/spreads, 12% said they consumed these products regularly. Statistical analysis found women with apparent knowledge of listeriosis risk were 2.5 times more likely to report low risk consumption practices in relation to high-risk foods. The results of this research found that pregnant women did not receive appropriate advice about *Listeria* risk from their health-care practitioner, and there appears to be an under-use of government authorised information pamphlets.

Both these surveys identified a significant proportion of pregnant women did not have sufficient information or knowledge to enable them to make informed choices about the foods they consumed.

In 2002, a national survey of 403 pregnant women across the USA was conducted. A further survey in 2003 collated data from 286 pregnant women in Minnesota (Ogunmodede *et al.*, 2005). Just 18% (74/403) of the national sample set had ever read, heard or seen information regarding listeriosis. The figure in Minnesota was slightly less at 15% (43/286). For those who had some information, the sources were predominantly medical, followed by friends/family and television with government agencies last. The population reporting avoidance of delicatessen foods were 14% (national survey) and 18% in the Minnesota region.

While risk communication information is provided to other at risk groups, such as, those undergoing cancer treatment (<http://www.cancernz.org.nz/assets/files/EatingWell.pdf>), older people (Ministry of Health, 2009) and general advice for at risk groups (<http://www.foodstandards.gov.au/srcfiles/Listeria.pdf>), no assessments were located of the effectiveness of education in groups other than pregnant women.

5.4 Control Options

The main risk for foodborne transmission of listeriosis is from foods with high numbers of *L. monocytogenes*, and these are likely to be long shelf-life foods in which *L. monocytogenes*

can grow e.g. vacuum-packed ready-to-eat meats. Targeting these foods for application of zero tolerance, or at least to ensure a count of <100/g when consumed, could be the most effective way to reduce disease. The dose response model indicates that eliminating foods with high levels of *L. monocytogenes* present will have significantly greater effect than eliminating foods with only a few cells present (e.g. preventing one meal containing 10⁶ *L. monocytogenes* cells present from being eaten will result in the same reduction in risk as preventing the consumption of a million meals containing 10⁰ *L. monocytogenes* cells).

Chen *et al.* (2003) found in a recent analysis that foods containing more than 100 CFU/g were responsible for more than 99% of listeriosis cases. The authors conclude that an alternative to the zero-tolerance strategy is one focusing on numbers rather than presence alone, so that measures limiting maximum numbers of *L. monocytogenes* in foods may have a greater impact on improving public health than a zero-tolerance strategy.

Conditions likely to result in large numbers of organisms becoming present in a food will include:

- The presence of the pathogen in the first instance;
- A food that supports the growth of *L. monocytogenes*;
- A suitable storage period to allow growth (this might be either a long period of refrigerated storage or lesser periods of time/temperature abuse); and,
- The absence of a listericidal step prior to consumption.

Risk management steps could be targeted at any of these points.

The USDA FSIS risk assessment concluded that, for products receiving a treatment that inactivates *L. monocytogenes*, the risk of listeriosis is determined to a large extent by the potential for recontamination after that treatment. This may occur in production, retail or domestic environments. The risk assessment concluded that new strategies were needed to decrease rates of recontamination during the manufacturing and marketing of ready-to-eat foods.

Education is currently an actively used form of risk management, especially for pregnant women.

5.5 Commentary on Control Options

Some classes of processed ready-to-eat meats are required to comply with a zero tolerance for *L. monocytogenes* under the Australia New Zealand Food Standards Code (FSANZ, 2009). However, the regulations regarding tolerances for *L. monocytogenes* are probably less important than the degree of compliance and during 2009 NZFSA consulted on a Code of Practice (CoP) for processed meat manufacturers.

Many manufacturers of ready-to-eat meats will already have in place HACCP based hazard management systems, and the implementation of the PQIP resource and the NZFSA CoP should expand the coverage.

Prevention of post-processing contamination is important, particularly for activities such as slicing. Long storage periods even at correct storage temperatures may allow *L. monocytogenes* to grow and reach high numbers by the end of their shelf life.

Education of pregnant women with regard to the dangers of listeriosis occurs currently although small scale surveys have revealed that these messages are not always being received and/or being put into practice. Enhancing the scheme to inform other at risk groups (particularly the over-60s) could reduce exposure of the susceptible population to the organism.

Typing of isolates from clinical cases and foods would assist in confirming the foodborne route of disease, and allow the identification of foods containing types associated with disease.

The low incidence of reported invasive listeriosis and historical fluctuations would make it difficult to use notification data as an indicator of trends in disease burden or the effects of risk management.

6 REFERENCES

- ACT Health Protection Service. (1998) Microbiological and chemical quality of cured and salted meats. January - March 1998. Accessed at: <http://www.health.act.gov.au/publications/foodsurvey/1997-98/curedmeat.html>. Accessed: 30 March 2008.
- Angelidis AS, Koutsoumanis K. (2006) Prevalence and concentration of *Listeria monocytogenes* in sliced ready-to-eat meat products in the Hellenic retail market. *Journal of Food Protection*; 69(4): 938-942.
- Anonymous. (1998a) 1997 a record year for listeriosis. *New Zealand Public Health Report*; 5: 53.
- Anonymous. (1998b) Multistate outbreak of listeriosis--United States, 1998. *Morbidity and Mortality Weekly Report*; 47(50): 1085-1086.
- Arnold GJ, Coble J. (1995) Incidence of *Listeria* species in foods in NSW. *Food Australia*; 47: 71-75.
- Art D, Andre P. (1991) Clinical and epidemiological aspects of Listeriosis in Belgium, 1985-1990. *Zentralblatt fur Bakteriologie*; 275(4): 549-556.
- Azevedo I, Regalo M, Mena C, Almeida G, Carneiro L, Teixeira P, Hogg T, Gibbs PA. (2005) Incidence of *Listeria* spp. in domestic refrigerators in Portugal. *Food Control*; 16(2): 121-124.
- Baek SY, Lim SY, Lee DH, Min KH, Kim CM. (2000) Incidence and characterization of *Listeria monocytogenes* from domestic and imported foods in Korea. *Journal of Food Protection*; 63(2): 186-9.
- Bean NH, Goulding JS, Lao C, Angulo FJ. (1996) Surveillance for foodborne-disease outbreaks--United States, 1988-1992. *MMWR CDC Surveillance Summaries*; 45(5): 1-66.
- Beumer RR, te Giffel MC, Spoorenberg E, Rombouts FM. (1996) *Listeria* species in domestic environments. *Epidemiology and Infection*; 117(3): 437-442.
- Biosecurity New Zealand. (2006) Import risk analysis: Porcine reproductive and respiratory syndrome (PRRS) virus in pig meat. Wellington: Ministry of Agriculture and Forestry.
- Biosecurity New Zealand. (2009) Draft IHS for Pig Meat and Pig Meat Products for Human Consumption from Finland or Sweden. Accessed at: <http://www.biosecurity.govt.nz/biosec/consult/draft-ihs-meaporic.spe>. Accessed: 30 November 2009.
- Bohaychuk VM, Gensler GE, King RK, Manninen KI, Sorensen O, Wu JT, Stiles ME, McMullen LM. (2006) Occurrence of pathogens in raw and ready-to-eat meat and poultry products collected from the retail marketplace in Edmonton, Alberta, Canada. *Journal of Food Protection*; 69(9): 2176-2182.

Bondarianzadeh D, Yeatman H, Condon-Paoloni D. (2007) *Listeria* education in pregnancy: Lost opportunity for health professionals. Australian and New Zealand Journal of Public Health; 31(5): 468-474.

Bredholt S, Nesbakken T, Holck A. (1999) Protective cultures inhibit growth of *Listeria monocytogenes* and *Escherichia coli* O157:H7 in cooked, sliced, vacuum- and gas-packaged meat. International Journal of Food Microbiology; 53(1): 43-52.

Brett MS, Short P, McLauchlin J. (1998) A small outbreak of listeriosis associated with smoked mussels. International Journal of Food Microbiology; 43(3): 223-9.

Buchanan RL, Damert WG, Whiting RC, Van Schothorst M. (1997) Use of epidemiologic and food survey data to estimate a purposefully conservative dose-response relationship for *Listeria monocytogenes* levels and incidence of listeriosis. Journal of Food Protection; 60(8): 918-922.

Buzby JC, Roberts T, Lin C-TJ, MacDonald JM. (1996) Bacterial foodborne disease: Medical costs and productivity losses. Agricultural Economic Report Number 741. Washington DC: United States Department of Agriculture Economic Research Service.

Cain DB, McCann VL. (1986) An unusual case of cutaneous listeriosis. Journal of Clinical Microbiology; 23(5): 976-7.

Carpenter SL, Harrison MA. (1989) Survival of *Listeria monocytogenes* on processed poultry. Journal of Food Science; 54: 556-557.

Chen Y, Ross WH, Scott VN, Gombas DE. (2003) *Listeria monocytogenes*: low levels equal low risk. Journal of Food Protection; 66(4): 570-577.

Codex Alimentarius Commission. (2009) Guidelines on the application of general principles of food hygiene to the control of *Listeria monocytogenes* in foods. CAC/GL 61-2007. Accessed at:

http://www.codexalimentarius.net/web/more_info.jsp?id_sta=10740. Accessed: 3 December 2009.

Cornelius AJ, Hudson JA, Wong TL. (2008) Enumeration and growth of naturally occurring *Listeria* spp. in unpackaged ham. Food Microbiology; 25(2): 407-412.

Cressey P, Lake R. (2005) Ranking food safety risks: Development of NZFSA policy 2004-2005. ESR Client Report FW0563. Christchurch: ESR.

Cressey P, Lake R. (2007) Risk ranking: Estimates of the burden of foodborne disease for New Zealand. ESR Client Report FW0724. Christchurch: ESR.

Cressey P, Lake R. (2008) Risk ranking: Estimates of the cost of foodborne disease for New Zealand. ESR Client Report FW07102. Christchurch: ESR.

Dalton CB, Austin CC, Sobel J, Hayes PS, Bibb WF, Graves LM, Swaminathan B, Proctor ME, Griffin PM. (1997) An outbreak of gastroenteritis and fever due to *Listeria monocytogenes* in milk. New England Journal of Medicine; 336(2): 100-5.

De Valk H, Vaillant H, Pierre V, Rocourt J, Jacquet C, Lequerrec F, Thomas J-C, Goulet V. (1998) Risk factors for sporadic listeriosis in France. Accessed at: <http://www.invs.sante.fr/epiet/seminar/1998/valk.html>. Accessed: 12 November 2008.

Dorozynski A. (2000) Seven die in French *Listeria* outbreak. *British Medical Journal*; 320(7235): 601.

Duffy LL, Vanderlinde PB, Grau FH. (1994) Growth of *Listeria monocytogenes* on vacuum-packed cooked meats: effects of pH, aw, nitrite and ascorbate. *International Journal of Food Microbiology*; 23(3-4): 377-390.

EFSA. (2005) The Community Summary Report on Trends and Sources of Zoonoses, Zoonotic Agents and Antimicrobial resistance in the European Union in 2004. *EFSA Journal*; 310: 1-275.

EFSA. (2006) The Community Summary Report on Trends and Sources of Zoonoses, Zoonotic Agents and Antimicrobial resistance and Foodborne Outbreaks in the European Union in 2005. *EFSA Journal*; 94: 1-288.

EFSA. (2007) The Community Summary Report on Trends and Sources of Zoonoses, Zoonotic Agents and Antimicrobial resistance and Foodborne Outbreaks in the European Union in 2006. *EFSA Journal*; 130: 1-352.

Elson R, Burgess F, Little CL, Mitchell RT. (2004) Microbiological examination of ready-to-eat cold sliced meats and pate from catering and retail premises in the UK. *Journal of Applied Microbiology*; 96(3): 499-509.

ESR. (1998) Annual surveillance summary 1997. Porirua: ESR.

ESR. (2004) Notifiable and other Diseases in New Zealand. Annual Report 2003. ESR Client Report FW0426. Kenepuru: ESR.

ESR. (2005) Notifiable and other Diseases in New Zealand. Annual Report 2004. ESR Client Report FW0532. Kenepuru: ESR.

ESR. (2006) Notifiable and other Diseases in New Zealand. Annual Report 2005. ESR Client Report FW0621. Kenepuru: ESR.

ESR. (2007) Notifiable and other Diseases in New Zealand. Annual Report 2006. ESR Client Report FW0717. Kenepuru: ESR.

ESR. (2008) Notifiable and other Diseases in New Zealand. Annual Report 2007. ESR Client Report FW08034. Kenepuru: ESR.

ESR. (2009) Notifiable and other Diseases in New Zealand. 2008 Annual Surveillance Report ESR Client Report FW09074. Kenepuru: ESR.

FAO/WHO. (2004) Risk assessment of *Listeria monocytogenes* in ready-to-eat foods: interpretative summary. Microbiological Risk Assessment Series No. 4. Geneva: World Health Organization.

Farber JM, Daley E, Coates F, Beausoleil N, Fournier J. (1991) Feeding trials of *Listeria monocytogenes* with a nonhuman primate model. *Journal of Clinical Microbiology*; 29(11): 2606-8.

Farber JM, Daley E, Holley R, Osborne WR. (1993) Survival of *Listeria monocytogenes* during the production of uncooked German, American and Italian-style fermented sausages. *Food Microbiology*; 10: 123-132.

Farber JM, Peterkin PI. (1991) *Listeria monocytogenes*, a food-borne pathogen. *Microbiological Reviews*; 55(3): 476-511.

Farber JM, Ross WH, Harwig J. (1996) Health risk assessment of *Listeria monocytogenes* in Canada. *International Journal of Food Microbiology*; 30(1-2): 145-156.

FSANZ. (2001a) Guidelines for the microbiological examination of ready-to-eat foods. Accessed at:
http://www.foodstandards.gov.au/_srcfiles/Guidelines%20for%20Micro%20exam.pdf.
Accessed: 30 March 2008.

FSANZ. (2001b) User guide to Standard 1.6.1 – Microbiological Limits for Food with additional guideline criteria. Accessed at:
http://www.foodstandards.gov.au/_srcfiles/Micro_limits_edit0702.pdf. Accessed: 3
December 2009.

FSANZ. (2009) Australia New Zealand Food Standards Code. Accessed at:
http://www.foodstandards.gov.au/_srcfiles/Standard_1_3_1_Additives_Part_1_v111.pdf.
Accessed: 30 March 2008.

Gianfranceschi M, Gattuso A, Fiore A, D'Ottavio MC, Casale M, Palumbo A, Aureli P. (2006) Survival of *Listeria monocytogenes* in uncooked Italian dry sausage (salami). *Journal of Food Protection*; 69(7): 1533-1538.

Gilbert RJ, de Louvois J, Donovan T, Little C, Nye K, Ribeiro CD, Richards J, Roberts D, Bolton FJ. (2000) Guidelines for the microbiological quality of some ready-to-eat foods sampled at the point of sale. PHLS Advisory Committee for Food and Dairy Products. *Communicable Disease and Public Health*; 3(3): 163-167.

Gilbert SE, Whyte R, Bayne G, Lake RJ, Van Der Logt P. (2007a) Survey of internal temperatures of New Zealand domestic refrigerators. *British Food Journal*; 109(4): 323-329.

Gilbert SE, Whyte R, Bayne G, Paulin SM, Lake RJ, van der Logt P. (2007b) Survey of domestic food handling practices in New Zealand. *International Journal of Food Microbiology*; 117(3): 306-311.

Gillespie I, Little C, Mitchell R. (2000) Microbiological examination of cold ready-to-eat sliced meats from catering establishments in the United Kingdom. *Journal of Applied Microbiology*; 88(3): 467-474.

Glass KA, Doyle MP. (1989) Fate of *Listeria monocytogenes* in processed meat products during refrigerated storage. *Applied and Environmental Microbiology*; 55(6): 1565-1569.

Gombas DE, Chen Y, Clavero RS, Scott VN. (2003) Survey of *Listeria monocytogenes* in ready-to-eat foods. *Journal of Food Protection*; 66(4): 559-569.

Gottlieb SL, Newbern EC, Griffin PM, Graves LM, Hoekstra RM, Baker NL, Hunter SB, Holt KG, Ramsey F, Head M, Levine P, Johnson G, Schoonmaker-Bopp D, Reddy V, Kornstein L, Gerwel M, Nsubuga J, Edwards L, Stonecipher S, Hurd S, Austin D, Jefferson MA, Young SD, Hise K, Chernak ED, Sobel J. (2006) Multistate outbreak of Listeriosis linked to turkey deli meat and subsequent changes in US regulatory policy. *Clinical Infectious Disease*; 42(1): 29-36.

Goulet V, Rocourt J, Rebiere I, Jacquet C, Moyse C, Dehaumont P, Salvat G, Veit P. (1998) Listeriosis outbreak associated with the consumption of rillettes in France in 1993. *Journal of Infectious Diseases*; 177(1): 155-160.

Grau F, Vanderlinde P. (1992) Occurrence, numbers, and growth of *Listeria monocytogenes* on some vacuum-packaged processed meats. *Journal of Food Protection*; 55: 4-7.

Grau FH. (1996) Smallgoods and *Listeria*. *Food Australia*; 48: 81-83.

Hall R, Shaw D, Lim I, Murphey F, Davos D, Lanser J, Delroy B, Tribe I, Holland R, Carman J. (1996) A cluster of listeriosis cases in South Australia. *Communicable Disease Intelligence*; 20: 465.

Harrison JA, Harrison MA. (1996) Fate of *Escherichia coli* O157:H7, *Listeria monocytogenes*, and *Salmonella typhimurium* during preparation and storage of beef jerky. *Journal of Food Protection*; 59: 1336-1338.

Harvey J, Gilmour A. (1993) Occurrence and characteristics of *Listeria* in foods produced in Northern Ireland. *International Journal of Food Microbiology*; 19(3): 193-205.

Hasell S. (2000) Hazard management guidelines for fermented meat products. ESR Client Report FW0092. Christchurch: ESR.

Havelaar AH, Galindo AV, Kurowicka D, Cooke RM. (2008) Attribution of foodborne pathogens using structured expert elicitation. *Foodborne Pathogens and Disease*; 5(5): 649-59.

Health Canada. (2008) Notifiable Diseases On-line. Laboratory Centre for Disease Control of Health Canada. Accessed at:

http://dsol-smed.phac-aspc.gc.ca/dsol-smed/cgi-bin/ndischart2?DATA_TYPE=R&YEAR_FROM=89&YEAR_TO=04&CAUSE=066&ARE_A=00&AGE=0&SEX=3&CTIME1=View+Chart. Accessed: 25 June 2008.

- Hitchins AD. (1996) Assessment of alimentary exposure to *Listeria monocytogenes*. *International Journal of Food Microbiology*; 30(1-2): 71-85.
- Hudson JA. (1992) Efficacy of high sodium chloride concentrations for the destruction of *Listeria monocytogenes*. *Letters in Applied Microbiology*; 14: 178-180.
- Hudson JA, Lowry PD, Boerema J, Delacy KM. (1991) Prevalence of *Listeria* spp. in retail meat products. *Communicable Disease New Zealand*; 92: 56-57.
- Hudson JA, Mott SJ. (1993) Growth of *Listeria monocytogenes*, *Aeromonas hydrophila* and *Yersinia enterocolitica* on cooked beef under refrigeration and mild temperature abuse. *Food Microbiology*; 10: 429-437.
- Hudson JA, Mott SJ. (1994) A survey for *Listeria* species, motile aeromonads and *Yersinia enterocolitica* on bovine and ovine carcasses. *New Zealand Veterinary Journal*; 42(1): 33-34.
- Hudson JA, Mott SJ, Delacy KM, Edridge AL. (1992) Incidence and coincidence of *Listeria* spp., motile aeromonads and *Yersinia enterocolitica* on ready-to-eat fleshfoods. *International Journal of Food Microbiology*; 16(2): 99-108.
- Hudson JA, Mott SJ, Penney N. (1994) Growth of *Listeria monocytogenes*, *Aeromonas hydrophila*, and *Yersinia enterocolitica* on vacuum and saturated carbon dioxide controlled atmosphere-packaged sliced roast beef. *Journal of Food Protection*; 57: 204-208.
- ICMSF. (1996) Micro-organisms in foods 5. Microbiological specifications of food pathogens. International Commission on Microbiological Specifications for Foods (ICMSF). London: Blackie Academic and Professional.
- 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.
- Institute de Veille Sanitaire. (2000) Outbreak of listeriosis linked to the consumption of rilletes in France. *Eurosurveillance*; 4(3): 1674.
- Jacquet C, Catimel B, Brosch R, Buchrieser C, Dehaumont P, Goulet V, Lepoutre A, Veit P, Rocourt J. (1995) Investigations related to the epidemic strain involved in the French listeriosis outbreak in 1992. *Applied and Environmental Microbiology*; 61(6): 2242-2246.
- Jeffers GT, Bruce JL, McDonough PL, Scarlett J, Boor KJ, Wiedmann M. (2001) Comparative genetic characterization of *Listeria monocytogenes* isolates from human and animal listeriosis cases. *Microbiology*; 147(Pt 5): 1095-104.
- Jensen A, Frederiksen W, Gerner-Smidt P. (1994) Risk factors for listeriosis in Denmark, 1989-1990. *Scandinavian Journal of Infectious Diseases*; 26(2): 171-178.
- Johansson T. (1998) Enhanced detection and enumeration of *Listeria monocytogenes* from foodstuffs and food-processing environments. *International Journal of Food Microbiology*; 40(1-2): 77-85.

Kieft C, Perks M, Baker M, Galloway Y, Sutherland H. (2000) Annual Surveillance Summary 1999. ESR Client Report FW0059. Porirua: ESR.

Lake RJ, Cressey PJ, Campbell DM, Oakley E. (2009) Risk ranking for foodborne microbial hazards in New Zealand: Burden of disease estimates. Risk Analysis; Published online 23 July 2009; DOI: 10.1111/j.1539-6924.2009.01269.x.

Lake RJ, Hudson JA, Cressey P, Nortje G. (2002) Risk Profile: *Listeria monocytogenes* in processed ready-to-eat meats. ESR Client Report FW0186. Christchurch: ESR.

Levine P, Rose B, Green S, Ransom G, Hill W. (2001) Pathogen testing of ready-to-eat meat and poultry products collected at federally inspected establishments in the United States, 1990 to 1999. Journal of Food Protection; 64: 1188-1193.

Lin CM, Takeuchi K, Zhang L, Dohm CB, Meyer JD, Hall PA, Doyle MP. (2006) Cross-contamination between processing equipment and deli meats by *Listeria monocytogenes*. Journal of Food Protection; 69(1): 71-79.

Lindqvist R, Westoo A. (2000) Quantitative risk assessment for *Listeria monocytogenes* in smoked or gravad salmon and rainbow trout in Sweden. International Journal of Food Microbiology; 58(3): 181-196.

Little CL, Barrett NJ, Grant K, McLauchlin J. (2008) Microbiological safety of sandwiches from hospitals and other health care establishments in the United Kingdom with a focus on *Listeria monocytogenes* and other *Listeria* species. Journal of Food Protection; 71(2): 309-318.

Loncarevic S, Danielsson-Tham ML, Martensson L, Ringner A, Runehagen A, Tham W. (1997) A case of foodborne listeriosis in Sweden. Letters in Applied Microbiology; 24(1): 65-68.

Lopez L, Baker M, Perks M. (2001) Annual surveillance summary 2000. ESR Client Report FW0156. Kenepuru: ESR.

Lowry PD, Tiong I. (1988) The incidence of *Listeria monocytogenes* in meat and in meat products- factors affecting distribution. In: (eds). Proceedings of 34th International Congress of Meat Science and Technology, Brisbane, Australia. pp: Part B pp 528-530.

Lund BM. (1990) The prevention of foodborne listeriosis. British Food Journal; 92: 13-22.

MacGowan AP, Bowker K, McLauchlin J, Bennett PM, Reeves DS. (1994) The occurrence and seasonal changes in the isolation of *Listeria* spp. in shop bought food stuffs, human faeces, sewage and soil from urban sources. International Journal of Food Microbiology; 21(4): 325-34.

MAFF. (1997) Microbiological survey – ready to eat meats and meat products. MAFF Food safety Information Bulletin No. 85. Accessed at: www.foodstandards.gov.uk/maff/archive/food/bulletin/1997/no85/rtemeat.htm. Accessed: 24 June 2008.

Mead PS, Slutsker L, Dietz V, McCaig LF, Bresee JS, Shapiro C, Griffin PM, Tauxe RV. (1999) Food-related illness and death in the United States. *Emerging Infectious Diseases*; 5(5): 607-625.

Miettinen MK, Bjorkroth KJ, Korkeala HJ. (1999) Characterization of *Listeria monocytogenes* from an ice cream plant by serotyping and pulsed-field gel electrophoresis. *International Journal of Food Microbiology*; 46(3): 187-92.

Ministry of Health. (1993) National cooked meat smallgoods survey. Wellington: Ministry of Health.

Ministry of Health. (1995) Microbiological Reference Criteria for Food. Wellington: Ministry of Health.

Ministry of Health. (2003) NZ Food NZ Children. Key results of the 2002 National Children's Nutrition Survey. Wellington: Ministry of Health.

Ministry of Health. (2009) Food and nutrition guidelines for healthy older people. A background paper. Draft for consultation. Wellington: Ministry of Health.

Morris IJ, Ribeiro CD. (1991) The occurrence of *Listeria* species in pate: the Cardiff experience 1989. *Epidemiology and Infection*; 107(1): 111-7.

New Zealand Pork Industry Board. (2007) Annual Report and Pork production in New Zealand. Accessed at: http://www.pork.co.nz/nzpork/technical_papers/introduction.asp. Accessed: 28 March 2008.

Nichols G, McLauchlin J, de Louvais J. (1998) The contamination of pâté with *Listeria monocytogenes*-results from the 1994 European community-coordinated food control programme for England and Wales. *Journal of Food Protection*; 61: 1299-1304.

Nørrung B, Andersen JK. (2000) Variations in virulence between different electrophoretic types of *Listeria monocytogenes*. *Letters in Applied Microbiology*; 30(3): 228-232.

Nørrung B, Andersen JK, Schlundt J. (1999) Incidence and control of *Listeria monocytogenes* in foods in Denmark. *International Journal of Food Microbiology*; 53(2-3): 195-203.

Notermans S, Dufrenne J, Teunis P, Chackraborty T. (1998) Studies on the risk assessment of *Listeria monocytogenes*. *Journal of Food Protection*; 61(2): 244-248.

NZFSA. (2005) A guide to calculating the shelf life of foods, an information booklet for the food industry. Wellington: New Zealand Food Safety Authority.

NZFSA. (2006) Domestic Food Review Transition Policy and Related Implementation. Discussion Document NZFSA Paper No 06/06 October 2006. Accessed at: <http://www.nzfsa.govt.nz/policy-law/projects/domestic-food-review/consultation/index.htm>. Accessed: 30 March 2008.

NZFSA. (2008) Which microbiological limits do I use?. Factsheet. Accessed at:

<http://www.nzfsa.govt.nz/processed-food-retail-sale/fact-sheets/which-micro.htm>. Accessed: 24 June 2008.

Ogunmodede F, Jones JL, Scheftel J, Kirkland E, Schulkin J, Lynfield R. (2005) Listeriosis prevention knowledge among pregnant women in the USA. *Infectious Diseases in Obstetrics and Gynecology*; 13(1): 11-15.

Ojeniyi B, Christensen J, Bisgaard M. (2000) Comparative investigations of *Listeria monocytogenes* isolated from a turkey processing plant, turkey products, and from human cases of listeriosis in Denmark. *Epidemiology and Infection*; 125(2): 303-308.

Olsen SJ, MacKinnon LC, Goulding JS, Bean NH, Slutsker L. (2000) Surveillance for foodborne-disease outbreaks--United States, 1993-1997. *MMWR CDC Surveillance Summaries*; 49(1): 1-62.

OzFoodNet. (2006) Burden and causes of foodborne disease in Australia: Annual report of the OzFoodNet network, 2005. *Communicable Disease Intelligence*; 30: 278-300.

OzFoodNet. (2007) Monitoring the incidence and causes of diseases potentially transmitted by food in Australia: Annual report of the OzFoodNet network, 2006. *Communicable Disease Intelligence*; 31: 345-365.

Perks M, Galloway Y, Baker M. (1999) Annual Surveillance Summary 1998. Porirua: ESR.

Pociecha JZ, Smith KR, Manderson GJ. (1991) Incidence of *Listeria monocytogenes* in meat production environments of a South Island (New Zealand) mutton slaughterhouse. *International Journal of Food Microbiology*; 13(4): 321-327.

Ross T, Rasmussen S, Fazil A, Paoli G, Sumner J. (2009a) Quantitative risk assessment of *Listeria monocytogenes* in ready-to-eat meats in Australia. *International Journal of Food Microbiology*; 131(2-3): 128-137.

Ross T, Rasmussen S, Sumner J. (2009b) Using a quantitative risk assessment to mitigate risk of *Listeria monocytogenes* in ready-to-eat meats in Australia. *Food Control*; 20(11): 1058-1062.

Rungan S, Badkar G. (2005) *Listeria*--how much do pregnant women really understand about it? *New Zealand Medical Journal*; 118(1225): U1745.

Russell DG, Parnell WR, Wilson NC, Faed J, Ferguson E, Herbison P, Horwath C, Nye T, Reid P, Walker R, Wilson B, Tukuitonga C. (1999) *NZ Food: NZ People*. Wellington: Ministry of Health.

Sagoo SK, Little CL, Allen G, Williamson K, Grant KA. (2007) Microbiological safety of retail vacuum-packed and modified-atmosphere-packed cooked meats at end of shelf life. *Journal of Food Protection*; 70(4): 943-951.

Schuchat A, Deaver KA, Wenger JD, Plikaytis BD, Mascola L, Pinner RW, Reingold AL, Broome CV. (1992) Role of foods in sporadic listeriosis. I. Case-control study of dietary risk

factors. The Listeria Study Group. Journal of the American Medical Association; 267(15): 2041-2045.

Schwartz B, Ciesielski CA, Broome CV, Gaventa S, Brown GR, Gellin BG, Hightower AW, Mascola L. (1988) Association of sporadic listeriosis with consumption of uncooked hot dogs and undercooked chicken. Lancet; 2(8614): 779-782.

Sergelidis D, Abraham A, Sarimvei A, Panoulis C, Karaioannoglou P, Genigeorgis C. (1997) Temperature distribution and prevalence of *Listeria* spp. in domestic, retail and industrial refrigerators in Greece. International Journal of Food Microbiology; 34(2): 171-177.

Sim J, Hood D, Finnie L, Wilson M, Graham C, Brett M, Hudson JA. (2002) Series of incidents of *Listeria monocytogenes* non-invasive febrile gastroenteritis involving ready-to-eat meats. Letters in Applied Microbiology; 35(5): 409-413.

Sneyd E, Baker M. (2003) Infectious diseases in New Zealand: 2002 Annual Surveillance Summary. ESR Client Report FW0332. Porirua: ESR.

Sneyd E, Lopez L, Eglinton M, McDowell R, Margolin T. (2002) Annual surveillance summary 2001. ESR Client Report FW0156. Kenepuru: ESR.

Sorrells KM, Enigl DC. (1990) Effect of pH, acidulant, sodium chloride and temperature on the growth of *Listeria monocytogenes*. Journal of Food Safety; 11: 31-37.

Sumner J. (2002) Food safety risk profile for primary industries in South Australia. Final Report. Adelaide: Department of Primary Resources SA.

Sumner J, Ross T, Jenson I, Pointon A. (2005) A risk microbiological profile of the Australian red meat industry: risk ratings of hazard-product pairings. International Journal of Food Microbiology; 105(2): 221-232.

Sutherland PS, Miles DW, Laboyrie DA. (2003) *Listeria monocytogenes*. In: Hocking AD (eds). Foodborne Microorganisms of Public Health Significance. Sixth edition. Sydney: Australian Institute of Food Science and Technology Inc.

Thomson BM, Nokes CJ, Cressey PJ. (2007) Intake and risk assessment of nitrate and nitrite from New Zealand foods and drinking water. Food Additives and Contaminants, Part A; 24(2): 113-21.

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

Trott D, Seneviratna P, Robertson J. (1991) *Listeria* in cooked chicken, pate and mixed smallgoods. Australian Veterinary Journal; 68(7): 249-250.

USDA. (2000) Food Safety and Inspection Service. Revised action plan for control of *Listeria monocytogenes* for the prevention of foodborne listeriosis. Accessed at: www.fsis.usda.gov/OA/topics/lm_action.htm. Accessed: 30 March 2008.

USDA. (2001) Reducing the risk of *Listeria monocytogenes*. Joint response to the President. Accessed at: www.foodsafety.gov/~dms/lmriplan.html. Accessed: 30 March 2008.

USDA. (2003) FSIS Risk assessment for *Listeria monocytogenes* in deli meats. Accessed at: <http://www.fsis.usda.gov/OPPDE/rdad/FRPubs/97-013F/ListeriaReport.pdf>. Accessed: 30 March 2008.

USDA. (2006) Pathogen Modeling Program (PMP). Accessed at: <http://ars.usda.gov/Services/docs.htm?docid=6786>. Accessed: 30 March 2008.

Uyttendaele M, De Troy P, Debevere J. (1999) Incidence of *Listeria monocytogenes* in different types of meat products on the Belgian retail market. *International Journal of Food Microbiology*; 53(1): 75-80.

Van de Weyer A, Devleeschouwer MJ, Dony J. (1993) Bactericidal activity of disinfectants on *Listeria*. *Journal of Applied Bacteriology*; 74(4): 480-483.

Varabioff Y. (1992) Incidence of *Listeria* in smallgoods. *Letters in Applied Microbiology*; 14(4): 167-169.

Vasseur C, Rigaud N, Hebraud M, Labadie J. (2001) Combined effects of NaCl, NaOH, and biocides (monolaurin or lauric acid) on inactivation of *Listeria monocytogenes* and *Pseudomonas* spp. *Journal of Food Protection*; 64(9): 1442-1445.

Vorst KL, Todd EC, Rysert ET. (2006) Transfer of *Listeria monocytogenes* during mechanical slicing of turkey breast, bologna, and salami. *Journal of Food Protection*; 69(3): 619-626.

Wang C, Muriana PM. (1994) Incidence of *Listeria monocytogenes* in retail franks. *Journal of Food Protection*; 57: 382-386.

Whyte R. (2000) ESR's foodborne illness case study. *Food Technology in New Zealand*; 35: 32.

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 IG. (1995) Occurrence of *Listeria* species in ready to eat foods. *Epidemiology and Infection*; 115(3): 519-526.

Winkowski K, Crandall AD, Montville TJ. (1993) Inhibition of *Listeria monocytogenes* by *Lactobacillus bavaricus* MN in beef systems at refrigeration temperatures. *Applied and Environmental Microbiology*; 59(8): 2552-2557.

Wong TL, Carey-Smith GV, Hollis L, Hudson JA. (2005) Microbiological survey of prepackaged pâté and ham in New Zealand. *Letters in Applied Microbiology*; 41: 106-111.

APPENDIX 1 HAZARD AND FOOD

1.1 *Listeria monocytogenes*

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

The following information is adapted from a data sheet <http://www.nzfsa.govt.nz/science/data-sheets/listeria-monocytogenes.pdf> prepared by ESR under a contract for the Ministry of Health (and now kept on the NZFSA website). The data sheet is intended for use by Regional Public Health Units.

1.1.1 Growth and survival

Growth:

Temperature: Optimum 37°C, range -1.5 to 45°C. Grows at refrigeration temperatures (4°C).

pH: Optimum 7.0, range 4.4-9.4.

Atmosphere: Grows optimally under microaerophilic conditions but grows well both aerobically and anaerobically. Can grow in relatively high (e.g. 30%) CO₂, but is inhibited under 100% CO₂. Growth was not retarded by a 5-10% CO₂ atmosphere.

Water activity: Minimum a_w permitting growth = 0.92 (≡11.5 % NaCl). Can grow in 10% NaCl at 35°C and 12% NaCl at 25°C and 10°C.

Survival:

Temperature: Survives freezing very well.

Atmosphere: Not influenced by atmosphere.

Viable but non-culturable (VNC) cells: There is some recent evidence that *L. monocytogenes* may become VNC.

L. monocytogenes can persist and tolerate a combination of low pH, high salt and low temperatures (Sorrells and Enigl, 1990).

1.1.2 Inactivation (CCPs and Hurdles)

Note that in microbiological terms “D” refers to a 90% (or decimal or 1 log cycle) reduction in the number of organisms. CFU = colony forming units.

Temperature: Rapidly inactivated at temperatures above 70°C. D time at 50°C can be in the order of hours, at 60°C 5-10 minutes, 70°C approximately 10 seconds.

pH: Inactivated at pH values less than 4.4 at rates depending on the acidulant and temperature. Organic acids, such as acetic, are more effective than mineral acids (e.g.

hydrochloric). Inactivation proceeds faster at higher temperatures.

Water activity (a_w): Can remain viable in dry environments for long periods. The effect of NaCl concentrations (6, 16 and 26% weight/volume NaCl) on the survival *L. monocytogenes* at low temperatures (10°C, refrigeration (average temperature 2°C) and freezing (-18°C)) has been studied (Hudson, 1992). All salt concentrations tested were ineffective in reducing numbers over 6 hours incubation at low temperatures. However, over 33 days, the organism grew in 6% NaCl, numbers remained the same in 16% NaCl and declined in 26% NaCl. Although *L. monocytogenes* was destroyed in 26% NaCl, numbers declined too slowly for immersion in cold brine to be a useful bacteriocidal treatment.

Preservatives: Inactivated on vegetables by lysozyme (100 mg/kg), 0.2% sodium benzoate at pH 5, 0.25-0.3% sodium propionate (pH 5, and less effective at lower temperatures), and 0.2-0.3% potassium sorbate (pH 5.0).

The use of appropriate starter cultures results in the elimination of the organism from salami via pH reduction. The addition of nitrite to salami-type meat batter had minimal effect on survival of the organism at 37°C (pH was the primary factor).

In other meats, with pH 6-6.3, nitrite (70-140 ppm) did retard growth, and sodium ascorbate (0.042%) in combination with the nitrite retarded growth further. Ascorbate had no effect in the absence of nitrite (Duffy *et al.*, 1994).

A commercial phage-based control for *L. monocytogenes* is approved by the US Food and Drug Administration as generally regarded as safe (GRAS) for use on all food products. It has not yet been approved by FSANZ for use in New Zealand and Australia.

Radiation: D values depend on the food and temperature and range from 0.34 to 2 kGy. A dose of 3 kGy does not eliminate *L. monocytogenes* from vacuum-packed pork. When present on fish the D values are lower (0.2-0.3 kGy). Is more sensitive than other Gram positive bacteria to UV radiation.

Disinfectant: Nine chemical disinfectants including phenolic compounds, alcohols, and quaternary ammonium compounds were tested against *L. monocytogenes* serotypes 1/2a, 1/2b, 1/2c and 4b (plus *L. innocua* and *L. welshimeri*). None of these strains displayed significant resistance; reductions of more than 5 log₁₀ CFU were achieved within 5 minutes contact time. However, the surface-active agents, aldehydes, dichlorine and quaternary ammonium compounds had diminished activity in the presence of organic matter (Van de Weyer *et al.*, 1993).

Vasseur *et al.* observed that pH shock (an alkaline treatment (pH 10.5) followed by an acid treatment (pH 5.4)) was effective against *L. monocytogenes* (3 log₁₀ CFU reduction) (Vasseur *et al.*, 2001).

1.2 Prevalence of Listeria in Processed Ready-to-eat Meat Products Overseas

Information from the scientific literature on the prevalence of *Listeria* species in general, and *L. monocytogenes* in particular, has been summarised for ready-to-eat meat products in Table 10.

Most of the prevalence values given fall within the range of 0-20% positive, although in a few cases the prevalence is up to almost 80%. Overall the reported prevalence appears to have declined in more recent surveys.

The prevalences given in the table will be underestimates. This is because the detection limit usually applied (presence in 25g) has a theoretical detection limit of one cell per 25g ($\approx 0.04/g$). Samples tested early in the shelf life may not yet have numbers sufficient for this detection level to be exceeded. Even if the “five unit sampling plan” is adhered to, the probability of detecting a contaminated batch is low. The proportion of contaminated units within a batch, as measured by a presence/absence test, will therefore increase with time during storage. The practice of testing one sample immediately after manufacture for the presence of *L. monocytogenes* will only detect the batches with highest numbers of cells.

A major survey of ready-to-eat meat and poultry products for *L. monocytogenes* from 1800 production facilities was conducted in the USA from 1990 to 1999. The results are presented in Table 11.

Table 12 focuses on the prevalence and enumeration of *L. monocytogenes* in ready-to-eat meat products across the European Union in 2006. Note that the practice of consuming raw minced beef in Belgium means that this product falls into the ready-to-eat category. Table 13 compares the percentage of positive *L. monocytogenes* samples in ready-to-eat meat and poultry products over three years (2004-2006), note that Belgium and Luxembourg were reporting positive results in raw meat intended to be eaten raw, with concentrations above 100 CFU/g. The percentage of positive *L. monocytogenes* results in poultry products in 2005 was acknowledged as low.

Information from the literature on the prevalence of *Listeria* species in general, and *L. monocytogenes* in particular, in ready-to-eat poultry has been summarised in Table 14. The prevalences reported for these foods are about the same as for other ready-to-eat meat products. This should not be considered surprising as these products are manufactured, distributed and retailed in a similar manner to other ready-to-eat meats.

Reports that provided quantitative data on levels of *L. monocytogenes* in ready-to-eat meats and poultry have been summarised in Table 15. In general, samples which are contaminated by *L. monocytogenes* usually contain low numbers (regarded here as being <10 CFU/g). Generally, very few samples contain numbers of *Listeria* in excess of this number.

Some caution must be observed in interpreting these data as most surveys test foods purchased from retail outlets. More realistic measures of numbers of pathogens in foods that people might eat need to take into account the fact that consumers will have products at home for some time before consumption. This extra time may allow numbers to increase further depending on the temperatures of consumers’ fridges, and the ability of the organism to grow in foods.

The distribution of *L. monocytogenes* in ready-to-eat foods can best be summarised as moderately frequent, but usually at low levels. The consequences of this observation depend a great deal on the nature of the food that is contaminated. Most fermented salami will not support the growth of *L. monocytogenes*, and so a low number of contaminants on the food at any point after manufacture is extremely unlikely to present a problem. However, this is not

the case for foods that are of a formulation that will allow *L. monocytogenes* to grow, such as roast beef.

Table 10: Reported prevalence of *Listeria* in overseas meat products

Country	Meat	Samples tested	Percentage positive <i>L. monocytogenes</i>	Percentage positive any <i>Listeria</i> species	Year	Reference
USA, Maryland and North California	8 categories of RTE foods - all categories - luncheon meats (mostly ham and bologna)	31,705 9,199	1.82 0.89	NS	2000/2001	(Gombas <i>et al.</i> , 2003)
Canada	Fermented sausage	100	4	NS	2001	(Bohaychuk <i>et al.</i> , 2006)
	Roast beef	101	0	NS	2001	(Bohaychuk <i>et al.</i> , 2006)
	Beef wieners	100	5.0	NS	2001	(Bohaychuk <i>et al.</i> , 2006)
Australia	Mixed small goods	20	0	0	1991	(Trott <i>et al.</i> , 1991)
Australia	Corned beef	72	72.2	83.3	1992	(Grau and Vanderlinde, 1992)
Australia	Ham	71	33.8	40.8	1992	(Grau and Vanderlinde, 1992)
Australia	Luncheon	13	23.1	15.4	1992	(Grau and Vanderlinde, 1992)
Australia	Salami	19	0	5.3	1992	(Grau and Vanderlinde, 1992)
Australia	Smallgoods	342	13.2	NS	1992	(Varabioff, 1992)
Australia (NSW)	Smallgoods	130	17.5	33.0	1995	(Arnold and Coble, 1995)
Australia (NSW)	Pâté	156	5.1	7.7	1995	(Arnold and Coble, 1995)
Australia	Vacuum packed sliced meats	175	45.0	NS	1996	(Grau, 1996)
Belgium	Cooked meat products	886	6.9	NS	1985-1990	(Art and Andre, 1991)
Belgium	Cooked meats Raw cured meats	824 3405	13.71 4.9	NS	1997-1998	(Uyttendaele <i>et al.</i> , 1999)
Canada	Salami	96	5.0	NS	1996	(Grau, 1996)
Denmark	Heat-treated meat products	45	5	NS	1994-1995	(Nørrung <i>et al.</i> , 1999)
Finland	Sausages and ham	24	79.2	NS	1998	(Johansson, 1998)
Finland	Frankfurters and pâtés	44	11.4	NS	1998	(Johansson, 1998)
Germany	Frankfurter	NS	17.0	NS	1996	(Grau, 1996)
Greece	Sliced RTE meat products	209	8.1, particularly	64.7%* 11/17	2004	(Angelidis and Koutsoumanis,

Country	Meat	Samples tested	Percentage positive <i>L. monocytogenes</i>	Percentage positive any <i>Listeria</i> species	Year	Reference
			cubed cooked ham and bacon	samples		2006)
Italy	Salami, pressed pork	243	0.2	NS	1996	(Grau, 1996)
Italy	Salami (from 17 production plants)	1020	22.7	NS	2002/2003	(Gianfranceschi <i>et al.</i> , 2006)
Korea	Ham	50	0	NS	1993-1997	(Baek <i>et al.</i> , 2000)
Switzerland	Dried beef, salami, mettwurst	99	4.0	NS	1996	(Grau, 1996)
UK	Pâté	216	35.0	NS	1989	(Morris and Ribeiro, 1991)
UK	Meat pâté	31	0	0	1991	(MacGowan <i>et al.</i> , 1994)
UK	Cooked meat	68	8.8	19.1	1991	(MacGowan <i>et al.</i> , 1994)
UK	Cured/cooked meat	39	0	0	1993	(Harvey and Gilmour, 1993)
UK	Salami etc	67	16.0	NS	1996	(Hitchins, 1996)
UK	Processed meat	29	7.0	NS	1996	(Hitchins, 1996)
UK	Ready-to-eat meat	2041	5.7	NS	1996	(Hitchins, 1996)
UK	Meat pâté	239	7.1	NS	1996	(Hitchins, 1996)
UK	Ready-to-eat meat products (mostly salami)	455	3.3	5.3	1997	(MAFF, 1997)
UK	Meat based pâté	1804	2.0	NS	1998	(Nichols <i>et al.</i> , 1998)
UK	Cold meats	2874	2.1	6.3	2002	(Elson <i>et al.</i> , 2004)
(catering & retail)	Meat pâté	639	1	NS	2002	
UK	Cold RTE sliced meats (catering)	3494	0.1	0.3	1998	(Gillespie <i>et al.</i> , 2000)
UK	Meat sandwiches (main filling) (hospital)	1141	3.1	7.8	2005-2006	(Little <i>et al.</i> , 2008)
UK	RTE meat samples, vacuum packed and MAP. At end of shelf life	2980	6.4	8.8	2003	(Sagoo <i>et al.</i> , 2007)

Country	Meat	Samples tested	Percentage positive <i>L. monocytogenes</i>	Percentage positive any <i>Listeria</i> species	Year	Reference
Northern Ireland	Bacon	20	NS	0	1994	(Wilson, 1995)
	Beef	1295	NS	3		
	Lamb	37	NS	3		
	Ham	1141	NS	6		
	Pork	794	NS	4		
	Fermented sausage	53	NS	2		
	Pâté	222	NS	1		
USA	Frankfurters (19 brands)	93	7.5	9.7	1994	(Wang and Muriana, 1994)
USA	Frankfurters (1 brand)	24	71	81	1994	(Wang and Muriana, 1994)
USA	Cooked beef	844	2.7	NS	1996	(Grau, 1996)
USA	Sliced ham	205	1.5	NS	1996	(Grau, 1996)
USA	Sliced ham/pork	NS	4.6	NS	1999	(USDA, 2000)
USA	Cooked/roast/corned/beef	NS	2.7	NS	1999	(USDA, 2000)
USA	Fermented sausage	NS	2.1	NS	1999	(USDA, 2000)
USA	Jerky	NS	0.0	NS	1999	(USDA, 2000)
Yugoslavia	Salami	21	19.0	NS	1996	(Grau, 1996)
Yugoslavia	Cooked sausage	14	21.0	NS	1996	(Grau, 1996)
Not specified	Cooked cured/smoked meat	29	7.0	NS	1990	(Lund, 1990)
Not specified	Salami and continental sausages	67	16.0	NS	1990	(Lund, 1990)
Not specified	Fermented sausages	30	20.0	NS	1990	(Lund, 1990)

NS = Not stated

* Other *Listeria* spp. assayed and enumerated only in samples positive for *L. monocytogenes*

The European Union have collated results from *L. monocytogenes* in ready-to-eat meat products, the results are presented in Table 10.

Table 11: Prevalence of *L. monocytogenes* in ready-to-eat meat and poultry, 1990 – 1999 at 1800 production facilities across the USA

Year	Cooked roast beef	Ham and luncheon meat	Small cooked sausages	Large cooked sausages	Jerky	Cooked poultry	Salads, spreads, pâtés	Fermented sausages
1990	22/345 (6.38)*	1/13 (7.69)	13/309 (4.21)	5/94 (5.32)	0/25 (0)	12/430 (2.79)	5.48 (19/347)	N/A
1991	20/498 (4.02)	4/73 (5.48)	28/387 (7.24)	12/261 (4.6)	0/39 (0)	17/649 (2.62)	15/473 (3.17)	N/A
1992	19/492 (3.86)	9/114 (7.89)	21/348 (6.03)	1/239 (0.42)	0/19 (0)	7/349 (2.01)	8/241 (3.32)	N/A
1993	13/428 (3.04)	12/149 (8.05)	25/472 (5.3)	7/328 (2.13)	0/39 (0)	6/314 (1.91)	6/274 (2.19)	N/A
1994	10/479 (2.09)	13/238 (5.46)	29/603 (4.81)	5/438 (1.14)	1/45 (2.22)	13/549 (2.37)	14/580 (2.41)	N/A
1995	15/560 (2.68)	5/100 (5.0)	25/611 (4.09)	5/438 (1.14)	0/50 (0)	20/889 (2.25)	28/597 (4.69)	N/A
1996	17/507 (3.35)	7/91 (7.69)	21/561 (3.74)	4/420 (0.95)	0/43 (0)	28/883 (3.17)	12/554 (2.17)	N/A
1997	11/530 (2.08)	12/286 (4.2)	17/621 (2.74)	6/371 (1.62)	0/40 (0)	9/946 (0.95)	5/206 (2.43)	10/108 (9.26)
1998	11/511 (2.15)	11/263 (4.18)	26/746 (3.49)	6/506 (1.19)	3/192 (1.56)	19/857 (2.22)	7/225 (3.11)	7/244 (2.87)
1999	25/922 (2.71)	44/960 (4.58)	38/2162 (1.76)	5/1167 (0.43)	0/278 (0)	14/970 (1.44)	5/435 (1.15)	10/478 (2.09)
Cumulative	163/5272 (3.09)	118/2287 (5.16)	243/6820 (3.56)	56/4262 (1.31)	4/770 (0.52)	145/6836 (2.12)	119/3932 (3.03)	27/830 (3.25)

Source: (Levine *et al.*, 2001)

N/A not applicable

No. positive/no. of samples (%)

Sample size more than 25g.

Table 12: *L. monocytogenes* in ready-to eat meat products in the European Union, 2006

Country	Product	N (for presence)	Presence in 25g (% pos.)	No. tested for enumeration	> detection but ≤100 CFU g ⁻¹ (%)	L. m. > 100 CFU g ⁻¹
2006	Bovine					
Single sample						
Belgium	Meat prep. eaten raw at retail	-	-	117	0.9	0
	Minced meat, eaten raw, at processing	67	14.9	-	-	-
	Minced meat, eaten raw, at retail	-	-	36	0	0
France	Meat products, cooked	-	-	57	29.8	7.0
Ireland	Meat products, cooked, at retail	44	15.9	44	2.3	0
	Meat products, cooked, at retail	-	-	208	0	0
		-				
Italy	Meat products, cooked	350	0			
Netherlands	Meat products, cooked	951	0.8	951	0.8	0
Batch						
Belgium	Meat products, cooked	122	27.0	-	-	-
Czech Republic	Meat products, cooked	373	0	-	-	-
Italy	Meat products, cooked	96	7.3	-	-	-
Poland	Meat products, cooked	79	10.1	8	12.5	0
EU total		2,082	3.5	1,421	2.0	0.3

Source: (EFSA, 2007)

Table 13: Percentage of positive *L. monocytogenes* prevalence in ready-to-eat meat and poultry products in the European Union, 2004-2006

Food item	% pos. 2004	% pos. 2005	% pos 2006
Bovine meat products, RTE	0 – 48.6	0.7 – 5.3	0 – 27.0
Pig meat products, RTE	0 – 27.6	0 – 26.5	0 – 34.0
Poultry meat products, RTE	0 – 40.0	0 – 3.1	0 – 36.5
Other meat, RTE	0 – 29.1	0 – 39.1	0 – 21.9

Source (EFSA, 2005;2006;2007)

Table 14: Reported prevalence of *L. monocytogenes* and *Listeria* species in overseas ready-to-eat poultry meat products

Country	Poultry meat	Samples tested	Positive <i>L. monocytogenes</i> (%)	Positive any <i>Listeria</i> species (%)	Year	Reference
Australia	Cooked chicken	50	16	24	1991	(Trott <i>et al.</i> , 1991)
Australia	Chicken liver pâté	30	16.6	16.6	1991	(Trott <i>et al.</i> , 1991)
Belgium	Cooked chicken products	53	16.9	NS	1985-1990	(Art and Andre, 1991)
Canada	Turkey breast	100	3.0	NS	2001	(Bohaychuk <i>et al.</i> , 2006)
Canada	Chicken wieners	101	3.0	NS	2001	(Bohaychuk <i>et al.</i> , 2006)
Denmark	Ready-to-eat turkey products	55	7.3	NS	2000	(Ojeniyi <i>et al.</i> , 2000)
Northern Ireland	Retail RTE Chicken Turkey	949 509	NS NS	11 5	1994	(Wilson, 1995)
UK	Ready-to-eat chilled chickens and portions	758	6.0	16.0	1997	(MAFF, 1997)
UK	Poultry pâté	268	2	NS	2002	(Elson <i>et al.</i> , 2004)
UK	Poultry sandwiches (main filling) (hospital)	376	5.9	12.2	2005-2006	(Little <i>et al.</i> , 2008)
UK/US	Ready-to-eat poultry	527	12	NS	1996	(Hitchins, 1996)

NS Not stated

Table 15: Quantitative data for *L. monocytogenes* in overseas ready-to-eat meats and poultry

Country	Food	Count breakdown (CFU/g)		Number positive	Reference
Belgium	Pâté	96.5%	<0.04(-ve)	376	(Art and Andre, 1991)
		2.9%	<10		
		0.3%	10-10 ²		
		0%	10 ² -10 ³		
		0.3%	10 ³ -10 ⁴		
Belgium	Sausages (ready-to-eat)	95.4%	<0.04(-ve)	241	(Art and Andre, 1991)
		3.3%	<10		
		0.8%	10-10 ²		
		0%	10 ² -10 ³		
		1.6%	10 ³ -10 ⁴		
Belgium	Hams	83.6%	<0.04(-ve)	61	(Art and Andre, 1991)
		9.8%	<10		
		1.6%	10-10 ²		
		1.6%	10 ² -10 ³		
		3.2%	10 ³ -10 ⁴		
Belgium	Steakburgers	94.7%	<0.04(-ve)	38	(Art and Andre, 1991)
		5.3%	<10		
Belgium	Other meat products	83.5%	<0.04(-ve)	121	(Art and Andre, 1991)
		14.9%	<10		
		0%	10-10 ²		
		0.8%	10 ² -10 ³		
		0.8%	10 ³ -10 ⁴		
Belgium	Chicken products	83.0%	<0.04(-ve)	53	(Art and Andre, 1991)
		5.7%	<10		
		1.9%	10-10 ²		
		3.8%	10 ² -10 ³		
		1.9%	10 ³ -10 ⁴		
		3.8%	10 ³ -10 ⁴		
Denmark	Heat-treated meat products	5% samples(+ve in 25g)		45	(Nørrung <i>et al.</i> , 1999)
		1.5%	10-100		
		1.4%	>100		
England	Ready-to-eat meat products (mostly salami)	96.7%	<0.04(-ve)	455	(MAFF, 1997)
		3.3%	<100		
England	Ready-to-eat chicken and chicken portions	84.2%	<0.04(-ve)	758	(MAFF, 1997)
		15.1%	<100		
		0.7%	>100		
England and Wales	Pâté	98%	<0.04 (-ve)	1,804	(Nichols <i>et al.</i> , 1998)
		1.5%	<200		
		0.06%	200-10 ³		
		0.12%	10 ³ -10 ⁴		
		0.12%	10 ⁴ -10 ⁵		

Country	Food	Count breakdown (CFU/g)		Number positive	Reference
		0.12%	>10 ⁶		
UK	Cold RTE sliced meats (catering)	61.5%	<10 ²	13	(Gillespie <i>et al.</i> , 2000)
		38.5%	10 ² -<10 ³		
UK	Cold meats	2.1%	<20	61	(Elson <i>et al.</i> , 2004)
		0.03%	10 ⁴ -<10 ⁵		
UK	Meat sandwiches (main filling) (hospital)	96.9%	<0.04(-ve)	35	(Little <i>et al.</i> , 2008)
		2.9%	0.04-1		
		0.2%	10-100	22	
		5.6%	0.04 -1		
	Poultry sandwiches (main filling) (hospital)				
Germany	Ready-to-eat meat products	13.7%	0.04-1	NS	(Notermans <i>et al.</i> , 1998)
		7.8%	1-10 ²		
		1.4%	10 ² -10 ⁴		
		0.2%	>10 ⁴		
Italy	Salami	100%	<10	232	(Gianfranceschi <i>et al.</i> , 2006)
California – USA	Luncheon meat	64.3%	0.4-10	28	(Gombas <i>et al.</i> , 2003)
		7.1%	>10 ² -10 ³		
		3.6%	>10 ³ -10 ⁴		
Maryland, USA	Luncheon meat	81.5%	0.04-11	54	
		9.3%	>10 ² -10 ³		
Wales	Pâté	65%	<0.04(-ve)	216	(Morris and Ribeiro, 1991)
		30%	samples+ve		
		5%	>10 ⁴		

+ve in 25g is equivalent to > 0.04/g, < count for next highest group.

1.2.1 Handling and Packaging

The Gombas *et al.* (2003) survey compared the packaging location and prevalence of *L. monocytogenes*:

- 77% of the luncheon meats were prepackaged by the manufacturer, of which 0.4% were positive
- 23% of samples were in-store packaged luncheon meats of which 2.7% were positive.

Several reasons have been proposed for the difference: additional handling at retail and differences in refrigerated displays. However, this does not mean that in-store retail packaging is worse because concentrations of *L. monocytogenes* tended to be higher in

manufacturer-packaged ready-to-eat foods, possibly because the organisms had a longer time to grow.

The differences in *Listeria* prevalence between whole, sliced or minced products was examined in a Belgian survey (Uyttendaele *et al.*, 1999).

Whole cooked product (before slicing)	1.56%
After slicing	6.65%
Whole meat product	3.96%
Cooked minced meat products	6.14%
Whole cured raw hams e.g. Prosciutto	14.92%
Minced cured meat products	11.69%

Overall cured meat products were more frequently contaminated with *L. monocytogenes* compared with cooked meat products.

APPENDIX 2 EVALUATION OF ADVERSE HEALTH EFFECTS

2.1 Dose Response

2.1.1 Listeriosis

Human feeding trials have not been undertaken due to ethical concerns. Monkey feeding trials and data from outbreaks have been used to model dose response interactions (Farber *et al.*, 1991).

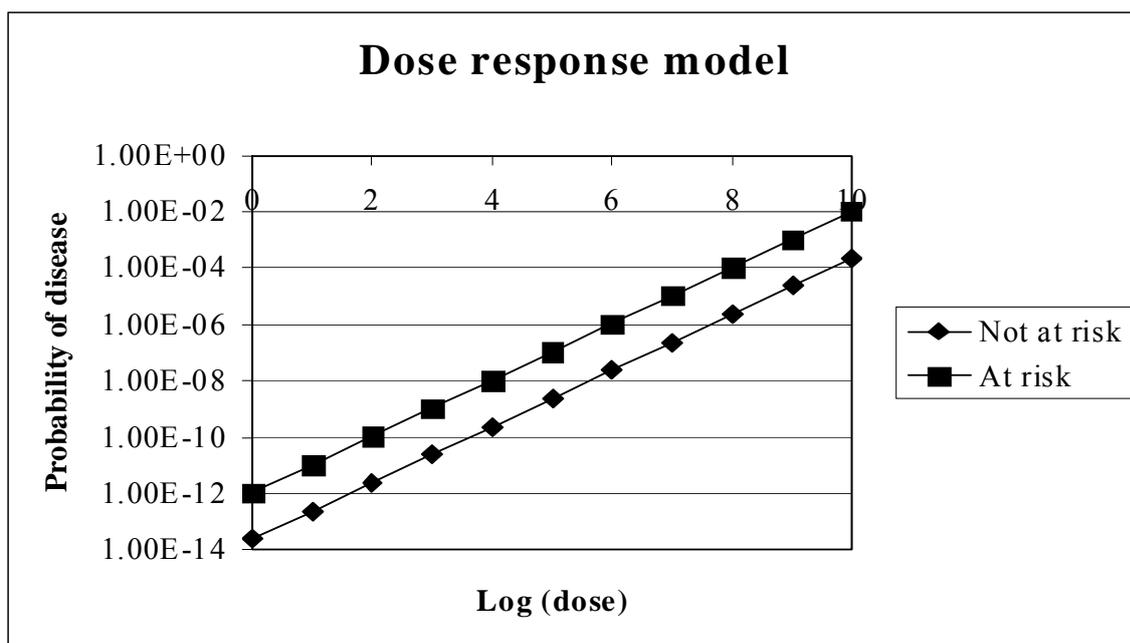
It is becoming increasingly realised that the only completely safe dose of *L. monocytogenes* is zero, even in healthy people. However the probability of invasive disease following exposure to even moderate levels of cells is very low.

The FAO/WHO risk assessment used a dose response model described by (FAO/WHO, 2004):

$$P_{\text{health outcome}} = 1 - \exp^{-R \cdot N}$$

where R is a variable that defines the dose/response relationship and N is the number of cells consumed. The value of R depends on the population group (to reflect different susceptibilities) but are approximately 10^{-12} - 10^{-14} . The model is a single hit model which means that there is a probability of illness associated with each cell consumed. It is therefore total consumption of cells that dictates risk; there is no “infectious dose”, and there is no difference to risk if a small number of cells are eaten frequently or many cells eaten at the same time as long as the total eaten is the same. Figure 4 shows dose response curves for high and low susceptibility groups.

Figure 4: Dose response models at median values for R for disease caused by *L. monocytogenes**



* Information provided by Dr. Tom Ross, University of Tasmania, and is that used in the FAO/WHO Listeria quantitative risk assessment.

There has been considerable discussion about the potential for a relaxation of the zero tolerance approach for *L. monocytogenes* contamination of food, to a tolerance of up to 100 CFU/g. Given the median daily intake of ready-to-eat meats (34.5g) (see Section 5.2), a dose of 100 CFU/g gives a median intake per contaminated meal of 3,450 *L. monocytogenes* cells. This exposure, taken with median values for R (1.06×10^{-12} for high susceptibility consumers and 2.37×10^{-14} for those at low susceptibility) results in a risk of infection of:

- 3.66×10^{-9} per day for consumers in the high susceptibility group
- 8.18×10^{-11} per day for consumers in the low susceptibility group

The FDA/FSIS modelled value of R accounts for variation of virulence in the types of *L. monocytogenes* extant in the population. It is known that certain serotypes of *L. monocytogenes* appear to be associated with human disease, but there is no certainty that any one isolate will be pathogenic to humans just because it belongs to a particular serotype. A recent study has grouped *L. monocytogenes* into three distinct lineages (Jeffers *et al.*, 2001), and there did appear to be some differences between the contributions that the lineages made to human disease. However, these lineages are not based on serotyping. The conservative approach is to treat all isolates as potentially capable of causing disease, but modelling of variability will be a more accurate reflection of real life. Virulence between different electrophoretic types of *L. monocytogenes* is discussed in Nørrung and Andersen (Nørrung and Andersen, 2000).

Analysis of the zero tolerance policy in the USA led Chen *et al.* to assess contamination levels and the associated risk with *L. monocytogenes* in food (Chen *et al.*, 2003). A survey of over 31,000 ready-to-eat retail foods, representing eight product categories found the overall prevalence to be 1.82% (Gombas *et al.*, 2003). Food survey data together with consumption and other population data were collated to derive a dose-response model. An exponential dose-response model calculates an R value of 1.76×10^{-10} (the probability of a single cell causing illness – for the population at highest risk). Chen *et al.* found in a recent analysis that foods containing more than 100 CFU/g were responsible for more than 99% of listeriosis cases (Chen *et al.*, 2003).

2.1.2 Febrile gastroenteritis

Dose response data for febrile gastroenteritis are limited. In a New Zealand outbreak involving ham, 21 of 24 (87.5%) people consuming the food contaminated with 1.8×10^7 *L. monocytogenes* CFU g⁻¹ became ill with symptoms of febrile gastroenteritis (Sim *et al.*, 2002). Assuming approximately 100g of ham was eaten by each person at the meal, then the dose ingested to produce this response was of the order of 10^9 CFU. In the outbreak described by Dalton *et al.* an attack rate of 75% was recorded where the median population consumed was estimated as being as high as 2.9×10^{11} CFU (Dalton *et al.*, 1997). In other outbreaks it is difficult to estimate dose responses as portion sizes are not detailed or the number of cells present not accurately known. However, of all of the other outbreaks, the lowest number in food that has been shown to cause febrile non-invasive listeriosis is 1.9×10^5 CFU g⁻¹ (Miettinen *et al.*, 1999), although the serving sizes were not detailed. In this incident all five people eating the contaminated fish became ill with gastroenteritis, nausea, abdominal cramps and diarrhoea.

Therefore consumption of more than, perhaps, 10^7 cells appears to be sufficient to cause *L. monocytogenes* febrile gastroenteritis at a high infection rate in some circumstances. It is possible that foods contaminated with lower numbers of *L. monocytogenes* may also infrequently cause febrile non-invasive gastrointestinal disease.

2.2 Adverse Health Effects Overseas

2.2.1 Incidence

Comparisons of listeriosis rates between countries must be made cautiously, as reporting practices may differ. However, the data in Table 16 indicate that New Zealand's rate is similar to that of other developed countries. EFSA lists the reported cases of human listeriosis cases for the years 2002-2006 for 24 European Member State countries and 5 Non-Member States, of which 11 countries are presented in Table 16 for the year 2006. The total reported listeriosis cases in the European Union in 2006 were 1,583 confirmed, equating to 0.3/100,000.

Table 16: Comparison of listeriosis incidence between countries

Country	Period	Rate /100,000	Reference
New Zealand	2008	0.6	(ESR, 2009)
Australia	2006	0.29	(OzFoodNet, 2007)
USA	2005	0.31	www.cdc.gov/mmwr
Canada	1999	0.25	(Health Canada, 2008)*
Europe	2006	0.3	(EFSA, 2007)
Belgium	2006	0.6	(EFSA, 2007)
Czech Rep.	2006	0.8	(EFSA, 2007)
England & Wales	2005	0.3	www.hpa.org.uk
Denmark	2006	1.0	(EFSA, 2007)
Germany	2006	0.6	(EFSA, 2007)
Finland	2006	0.9	(EFSA, 2007)
France	2006	0.5	(EFSA, 2007)
Luxembourg	2006	0.9	(EFSA, 2007)
Netherlands	2006	0.4	(EFSA, 2007)
Sweden	2006	0.5	(EFSA, 2007)
Spain	2006	0.2	(EFSA, 2007)
UK	2006	0.3	(EFSA, 2007)

* all types of listeriosis, (removed from Canadian national surveillance as of January 2000).

2.2.2 Contributions to outbreaks and incidents

Information on outbreaks associated with transmission of *L. monocytogenes* via ready-to-eat meats and ready-to-eat poultry are summarised in Tables 17 and 18 respectively. Data on the contribution of *L. monocytogenes* to overall foodborne disease outbreaks and incidents overseas are given in Table 19.

When outbreaks of listeriosis occur, they often involve a large number of cases. For example three of the ten meat product related outbreaks in Table 17 involved more than 100 people.

This is because they are associated with products that have extremely wide distributions (e.g. hot dogs/deli meats outbreak), and/or are associated with contaminated products distributed over a long period (e.g. pork tongue in jelly outbreak). The outbreaks are often dispersed over time and geography, and may be detected only because of a good bacterial typing system (e.g. hot dogs/deli meats outbreak) or because of a rise in the number of cases above that expected (e.g. pork tongue in jelly outbreak). The use of typing as a tool for outbreak recognition allows interventions to be relatively rapid, whereas outbreak recognition relying on a rise in the number of reported cases mean that the outbreak had been occurring for some months before detection.

Fortunately outbreaks of listeriosis are rare (at least with respect to other foodborne pathogens), and often do not feature in summaries of foodborne disease. The small amount of information reflects the low contribution that listeriosis makes to foodborne disease each year. Where data are available (Table 19) a very few percent of outbreaks and a fraction of a percent of cases are caused by *L. monocytogenes*.

2.2.3 Case control studies

The case control studies (Table 20) reflect information that is consistent with observed outbreaks and distributions of the organism in foods. The USA has in recent years experienced a large multi-state outbreak due to the consumption of hot dogs. This product should probably be cooked before consumption, but by custom seems not to be in the USA, at least with some consumers. Pâté was apparently responsible for a large rise in case numbers in the UK, which was halted through notifying the public about the risk associated with pâté consumption. The outbreak of non-invasive infection with *L. monocytogenes* in New Zealand (Sim *et al.*, 2002) confirms the risk from deli counter food as noted in a US study.

Case-control studies for sporadic listeriosis in France (De Valk *et al.*, 1998) and the USA study (Schuchat *et al.*, 1992) identified soft cheese consumption as the principal risk factor.

Table 17: Overseas listeriosis outbreaks associated with ready-to-eat meat consumption

Country	Food implicated	No. ill	No. deaths	Evidence for food implicated	Reference, year
Australia	Pâté	11	NS	NS	(Grau, 1996)
France	Pork tongue in jelly	279	85 (including 22 abortions)	Case control study, isolate typing	(Jacquet <i>et al.</i> , 1995)
France	Pork rillettes	38	NS	Epidemiological, typing of case and food isolates	(Goulet <i>et al.</i> , 1998)
France	Pork rillettes	6	2	Same serotype, phage type and DNA macrorestriction pattern, food history	(Institute de Veille Sanitaire, 2000)
France	Pork tongue in aspic	26	7	NS	(Dorozynski, 2000)
Italy	Pork sausage	1	NS	NS	(Grau, 1996)
Sweden	Medwurst	1	NS	Epidemiological, isolate typing	(Loncarevic <i>et al.</i> , 1997)
UK	Pâté	>300	NS	Epidemiology	(Farber and Peterkin, 1991)
USA	Pork and rice sausage	1	NS	NS	(Grau, 1996)
USA	Hot dogs/deli meats	101	21	Epidemiological, strain typing in food and case isolates	(USDA, 2001)

NS = Not Stated

Table 18: Overseas listeriosis outbreaks associated with ready-to-eat poultry consumption

Country	Food implicated	No. ill	No. deaths	Evidence for food implicated	Reference
Australia	Diced chicken	5	1	Isolation from chicken and preparation area. DNA typing.	(Hall <i>et al.</i> , 1996)
USA	Deli turkey meat	29	4 deaths, 3 miscarriages/ stillbirths	Epidemiological	(Anonymous, 1998b)

Table 19: Contribution of *L. monocytogenes* to foodborne disease outbreaks and incidents overseas

Country	Year	No. (%) Outbreaks	No. (%) incidents or cases	Reference
Canada	1981	NS	1 (0.2) incidents 41 (0.0) cases	(Todd, 1992)
USA	1989	1 (0.2)	2 (0.0) cases	(Bean <i>et al.</i> , 1996)
USA	1993-1997	3 (0.1)	100 (0.1) cases	(Olsen <i>et al.</i> , 2000)
Australia	2005	1/102 (1.0)	3/1975 (0.15)	(OzFoodNet, 2006)
Europe*	2006	9*/5807** (0.15)	120/55029 (0.2)	(EFSA, 2007)

* Belgium, Czech Republic, Finland, Germany, Spain and Switzerland

** Europe except Cyprus, Luxembourg and Malta. Figure includes Norway, Romania and Switzerland

Table 20: Case control studies containing information on *L. monocytogenes* in ready-to-eat meats

Country	Risk/Protective factor	Odds ratio (95% CI)	Reference
Denmark	Pâté (risk)	>8.1 (0.6 - 117)	(Jensen <i>et al.</i> , 1994)
USA	Uncooked hot dogs (risk) Undercooked chicken (risk)	12.3 (1.6 - 97.3) 20.5 (1.2 - 343)	(Schwartz <i>et al.</i> , 1988)
USA	Food purchased from store delicatessen counters* (risk) Eating undercooked chicken (risk, among immunosuppressed patients)	1 (1.0 - 2.5) 3.3 (1.2 - 9.2)	(Schuchat <i>et al.</i> , 1992)
USA	Consumed turkey deli meat (risk) – 4 weeks before illness	4.5 (1.3 – 17.1)	(Gottlieb <i>et al.</i> , 2006)

*Includes cold meats, sandwiches, cheese and salads. 26 of 31 volunteering information bought “some” ready-to-eat meats from this source

CI = Confidence interval

The Centers for Disease Control in the USA have a website containing information on case-control studies and listeriosis, (see website: http://www.cdc.gov/foodnet/studies_pages/case.htm, accessed 16.04.08). Started in February 2000, the project has 8 FoodNet sites participating.

APPENDIX 3 EVALUATION OF RISK

3.1 Risk Assessments

3.1.1 FAO/WHO

The FAO/WHO *Listeria monocytogenes* in ready-to-eat foods risk assessment reflects the state of knowledge as at 2002 (the Series 4 booklet contains an interpretative summary while Series 5 contains the technical report) (FAO/WHO, 2004). Four foods were selected for the risk assessment, each different in terms of contamination, storage and consumption patterns: milk, ice cream, cold-smoked fish and fermented meat products.

The risk assessment aimed to answer three specific questions:

1. Estimate the risk of serious illness from *L. monocytogenes* in food when there is a range of absence in 25g through to a contamination rate of 1000 CFU/g;
2. Estimate the risk of serious illness for consumers in different susceptible population groups, relative to the general population; and,
3. Estimate the risk of serious illness from *L. monocytogenes* in foods that support its growth and foods that do not under specific storage and shelf-life conditions.

To estimate the risk from a range of contamination rates, two approaches were taken. Firstly, based on the predicted risk per serving, the number of cases annually was predicted using a worst-case scenario in that all servings had maximum contamination of 0.04, 0.1, 1, 10, 100 and 1000 CFU/g. The second approach was a more realistic but complex model using a distribution for the numbers of *L. monocytogenes* consumed in food rather than absolute values, giving a risk per serving and a much lower predicted number of cases annually.

Two “what-if” scenarios were developed based on the current 0.04 CFU/g standard in the USA and the 100 CFU/g standard used in many other countries. Defect rates as a percentage of servings exceeding these standards were then modeled. With up to 1% “defective” servings, the difference in predicted numbers of cases between 0.04 and 100 CFU/g standards was very small.

The probability of becoming ill from ingesting *L. monocytogenes* is higher for susceptible populations compared to the general population. Based upon the US data, people aged 60+ years were 2.6 times more susceptible relative to the general healthy population. Perinatal neonates were 14 times more susceptible.

The same analysis has been carried out on other susceptible sub-populations.

Key findings from this risk assessment are:

- Probability of illness from consuming a specified number of bacterial cells is based on a disease triangle of food matrix, virulence of strain and susceptibility of the consumer;
- The model predicts that nearly all listeriosis cases are the result of eating high numbers of *L. monocytogenes*;

- Based on available data, there doesn't appear to be any dose-response variations between populations in different countries, but differences in manufacturing and handling practices can affect contamination rates and therefore risk per serving;
- Control measures that reduce frequencies of contamination have proportional reductions in rate of illness. Control measures that prevent high levels of contamination, at point of consumption would be expected to have the greatest reduction impact;
- Better temperature control or limiting length of storage periods will mitigate the increased risk in foods that support growth;
- The vast majority of cases are associated with consumptions of foods that do not meet current standards, whether that be zero-tolerance or 100 CFU/g

The quantitative data on *L. monocytogenes* contamination of food was based on primarily European foods, the consumption data were based on Canada or the USA. The dose-response function is based upon elements of the FDA risk assessment, see website: ftp://ftp.fao.org/codex/alinorm04/al04_13e.pdf

In terms of the fermented meat products, it was noted that serving sizes and rates of consumption are usually moderate in most countries. Processing and composition differ world-wide but salami and pepperoni represents the vast majority of products. Traditionally, production does not include a thermal inactivation step although some manufacturers do include this step between fermentation and drying. Hurdles such as salt, lactic acid and nitrites prevent *Listeria* growth, so that although storage times can be lengthy, growth does not occur and inactivation is likely. Contamination of raw meat ingredients can lead to moderate contamination at retail but generally the global number of annual cases per 100,000 was the lowest of the four foods modeled.

3.1.2 USA

The United State's joint risk assessment conducted by the Food and Drug Administration (FDA) and the United States Department of Agriculture (USDA) Food Safety and Inspection Service (FSIS) was published in September 2003 (<http://www.foodsafety.gov/~dms/lmr2-toc.html>) and is very much a North American risk assessment, using an exposure assessment particular to that part of the world (even though data from all over the world were used to calculate prevalences in food). We might assume that the hazard characterization (essentially dose response) would be the same in New Zealand as North America, but the derived risk characterisation will be different because of the different exposure assessments.

The relative risks predicted for the various ready-to-eat food categories in the FDA/FSIS risk assessment are given in Table 21 for various at-risk population groups, and also as an overall ranking. One food, frankfurters, may or may not be reheated prior to consumption so is considered as two separate food categories. It is recognised that additional foods or cross-contamination may contribute further cases. Note that the rankings in this table have changed from those given in the draft version of this risk assessment.

Given the caveats regarding the data, it can be noted that several meat products have a high relative risk in the North American population, with the pâté/meat spread, deli meats and non-reheated frankfurter categories ranking in the top five predictive relative risks for listeriosis. Frankfurters are a food that is meant to be eaten only after heating, but in the US it is customary for some people not to cook them prior to consumption. Since they are pre-

cooked then they can be considered as analogous to pre-cooked sausages available in New Zealand. This food receives a very high rank only when it is treated as a food that is not subject to further cooking prior to consumption. The overall frankfurter ranking assumes 1-14% of frankfurters are consumed without cooking, and they reach a moderate ranking because of the large volumes that are consumed.

Table 21: Predicted relative risk rankings for listeriosis based on the North American sub-population using median estimates on a per serving basis

Food Categories ^a	Sub-Population			
	Intermediate Age ^b	Elderly ^b	Perinatal ^b	Total ^{b,c}
Relative Rank (1- 23)				
SEAFOOD				
Smoked seafood	6	5	5	5b
Raw seafood	12	12	12	13d
Preserved fish	13	13	13	12d,e
Cooked ready-to-eat crustaceans	5	6	6	6b
FRUIT AND VEGETABLES				
Vegetables	18	18	18	18
Fruits	15	15	15	14e
DAIRY PRODUCTS				
Fresh soft cheese (e.g. queso fresco)	10	10	10	10
Soft ripened cheese, >50% moisture	17	17	17	17f
Soft unripened cheese, >50% moisture	8	8	8	8c
Semi-soft Cheese, 39-50% moisture	16	16	16	16f
Processed cheese	20	20	20	21g
Hard cheese <39% moisture	23	23	23	23
Fluid milk, pasteurised	9	9	9	9c
Fluid milk unpasteurised	4	4	4	4b
Ice cream and frozen dairy products	21	21	21	20g
Cultured Milk Products	22	22	22	22g
High Fat and Other Dairy Products	7	7	7	7
MEATS				
Reheated frankfurters	11	11	11	11
Non-reheated frankfurters	2	2	2	2a
Dry/semi dry fermented sausages	14	14	14	15d
Deli meats	1	1	1	1a
Pâté and meat spread	3	3	3	3
COMBINATION FOODS				
Deli salads	19	19	19	19

^a Food categories are grouped by type of food but are not in any particular order.

^b A ranking of 1 indicates the food category with the greatest predicted relative risk per serving of causing listeriosis and a ranking of 23 indicates the lowest predicted relative risk of causing listeriosis.

^cRanks with the same letter are not significantly different based on the Bonferroni Multiple Comparison Test (alpha = 0.05).

Source: FDA/FSIS 2003 (<http://www.cfsan.fda.gov/~dms/lmr2-5.html>)

Another risk assessment was conducted by the USDA FSIS in parallel with that by the FDA/FSIS above. This concerned the risk from *Listeria monocytogenes* in deli meats (USDA, 2003) and considered in particular the risk from cross contamination via food contact surfaces. The intention was to provide a scientific basis for a proposed rule on testing and sanitation for food contact surfaces in processing plants.

To supplement the FDA/FSIS risk assessment, an “in-plant” dynamic model was constructed to describe the behaviour of *L. monocytogenes*, and the effect of testing and sanitation options.

The 2003 risk assessment of deli meats estimated the risk of illness from meat sliced and packaged at federally inspected processing establishment and those sliced at retail facilities. The results indicated that approximately 80% of listeriosis cases related to deli meats were associated with those sliced at retail. These preliminary results have now been supplemented by a more detailed re-analysis released for public comment in early 2009. This indicated that approximately 83% of listeriosis cases and deaths attributed to deli meat consumption are from deli meat sliced and packaged at retail.

For links to the deli meats and related risk assessments see:

http://www.fsis.usda.gov/Science/Risk_Assessments/index.asp

3.1.3 Australia

Two relevant studies have been completed in Australia. The first was a risk profile for *L. monocytogenes* in cooked and cured smallgoods including whole hams, sliced vacuum packed meat and pâté (Sumner, 2002). Risk ratings were prepared for these three hazard-product pairings on a scale of 0-100 (0 =no risk, 100 = everybody eating a meal containing a lethal dose of the hazard every day). A “low” risk equated to <25, “medium” 26-40, and “high” >40. Because the scale is logarithmic, an increment of 6 in the ranking relates to a 10-fold increase in risk. The risk ranking for cooked, cured ham was 45 (high), sliced vacuum packed meat 51 (high) and pâté 39 (medium). Details of the risk rating and associated assumptions are presented in Table 22. Assumptions for the assessment are;

- 2% raw meat contamination levels based on Meat and Livestock Australia (MLA) data;
- recontamination levels based on MLA risk assessment of *L. monocytogenes* in small goods;
- higher levels in sliced product (to reflect increased surface area and handling); and
- increase to infective dose based on FAO/WHO risk assessment – probability response around 10^7 cells.

Table 22: Risk Ranking of smallgoods and *L. monocytogenes* (South Australia)

Risk Criteria	Whole hams	Sliced, vacuum packed meat	Pâté
Dose and severity			
Hazard severity	Moderate	Moderate	Moderate
Susceptibility	General	General	General
Probability of exposure			
Frequency of consumption	Monthly	Monthly	Few times
Proportion consuming (%)	Most (75)	Most (75)	Some (25)
Size of population	S. Australia 1.5 million	S. Australia 1.5 million	S. Australia 1.5 million
Probability of contamination			
Probability of raw meat contaminated	2%	2%	2%
Effect of processing	Reliably eliminates	Reliably eliminates	Reliably eliminates
Possibility of recontamination	Minor (1%)	10%	Minor (1%)
Post-process control	Well controlled – cold chain	Well controlled – cold chain	Well controlled – cold chain
Further cooking before eating	Not effective in reducing hazard	Well controlled – cold chain	Well controlled – cold chain
Predicted illnesses p.a. in selected population	1	14	0.1
Risk Rating (0 – 100)	33	33	44

The assessment predicted one illness per annum in the general and very susceptible populations providing that the raw material contamination was low (0.1/g or 10 cells per serving). However, where the raw material was contaminated to the extent of 1000 cells per serving and eaten by susceptible individuals, the prediction rises significantly to 114 annual illnesses.

In the second study, the authors collated information on a range of red meat products and microbiological hazard combinations including ready-to-eat meats with extended shelf life and *L. monocytogenes* (Sumner *et al.*, 2005). Details of the risk ranking for *L. monocytogenes* in a range of smallgoods products can be found in Table 23. Infectious dose estimates were based upon $ID_{50} \sim 10^{12}$ CFU by FAO/WHO (2004).

Table 23: *L. monocytogenes* in processed meat products, Australia

Risk Criteria	Cooked sausages	Salami	Deli meats	Pâté /terrines	Fresh sausage
Hazard severity	Moderate	Moderate	Moderate	Moderate	Moderate
Population susceptibility	General	General	General	General	General
Frequency of consumption	Weekly	Weekly	Weekly	Monthly	Weekly
Proportion consuming (%)	75%	25%	100%	25%	75%
Total population	19.7 million	19.7 million	19.7 million	19.7 million	19.7 million
Proportion of raw product contaminated (%)	10%	10%	10%	10%	10%
Effect of processing on hazard	100% reduction	99% reduction	100% reduction	100% reduction	No effect
Post processing contamination rate (%)	15	12	9	10.5	5
Post-processing control	1000 x increase	No increase	1000 x increase	1000 x increase	3 x increase
Increase required to cause infection	5×10^{10}	5×10^{10}	5×10^{10}	5×10^{10}	5×10^{10}
Effects of preparation before eating on hazard	99% reduction	No effect	No effect	No effect	99% reduction
Predicted cases per annum	0.04	0.0003	5	0.7	0.0001
Risk Rating (0 – 100)	25 low	12 low	36 medium	32 medium	11 low

The two medium ratings for deli-meats and pâtés stem from the likelihood of post-process contamination and a long shelf life (up to 8 weeks in distribution, retail and consumer chain). The low score of 11 for fresh sausages reflects the possibility that some cells may survive the cooking process where sausages are undercooked. Cooked sausages scored low because of the 5-log reduction cooking process.

Despite a reduction in contamination rates of Australian smallgoods over the past decade, Australia has not seen a corresponding decline in listeriosis incidence. The risk ranking work was therefore taken to the next stage and a quantitative risk assessment of *L. monocytogenes* in ready-to-eat meats undertaken. Two papers on the research have recently been published, one detailing the risk assessment (Ross *et al.*, 2009a), the second on how the model can be used to mitigate the risk (Ross *et al.*, 2009b).

The quantitative risk assessment studied luncheon meat, cooked sausages and pâtés to cover the various meat formulations (Ross *et al.*, 2009a). Fermented meats were also considered but due to the risk being considered negligible, were not further developed. A negligible risk conclusion was also made in the FAO/WHO (2004) risk assessment described above.

A stochastic simulation model was constructed that predicts numbers of the organism likely to be consumed in RTE meats under a wide range of scenarios. If the model predicted that the product would be spoiled before consumption, the product was assumed to be discarded and not a contributor to the illness burden. The purpose was to improve the estimate of risk and more specifically to identify where critical data were lacking. The model is based on initial contamination levels, product formulation, times, temperatures, storage and consumption patterns. The prevalence of contamination was based on unpublished industry data from 1997 – 2003 and had average prevalences for processed (deli) meats (4.77%), pâtés (1.20%), and cooked sausages/frankfurters (2.77%). The initial microbial counts on cooked meat products were typically in the range $10^2 - 10^3$ CFU/g. On any day, between 20 to 50 % of Australians consume RTE meats, with various serving sizes depending on the nature of the product.

Swedish data estimate approximately 20% of their population are at increased risk of listeriosis (including >65 years old) (Lindqvist and Westoo, 2000). USA data estimate the figure also to be approximately 20% of their population (Buchanan *et al.*, 1997). The largest contributor to the at-risk group will be those over 60 years of age due to their diminishing immunity. In Australia, the YOPI (Young, Old, Pregnant or Immuno-compromised) group accounts for between 15-18.7% of the population (based on neonates, over 65s/over 60s, pregnant women and their foetuses, alcoholics, HIV and AIDS patients). The probable number of listeriosis cases due to processed meats was predicted to be 43 per year, with pâtés/liverwursts contributing an additional 0.36 cases and cooked sausages 0.24 cases, making the overall predicted number of cases 44 per year due to RTE meat products. This equates to approximately 40% of Australia’s listeriosis cases annually and is in agreement with available epidemiological data.

Inputs to the model can be changed to investigate different management strategies and it was these mitigation strategies that formed the basis for the second paper (Ross *et al.*, 2009b). The authors explored potential risk management options, by identifying and manipulating factors that contribute most significantly to the risk. These mitigations were;

- 1) reducing prevalence of *L. monocytogenes* at the plant, (by 90% and 67%)
- 2) reducing growth rate of *L. monocytogenes* on processed meats, (to reflect the addition of a listeristatic compound), growth rate reduced by 50% and 30%, and
- 3) reducing *L. monocytogenes* levels ‘in-pack’ (such as heat treatment or high pressure processing) (effectively a 3-4 log CFU/g reduction but a 1-2 log reduction was also modelled).

The magnitude of the risk reductions using each scenario is listed below.

Simulated risk reduction strategy	Predicted risk relative to <i>status quo</i> (%)
90 % reduction in prevalence	20.9
66% reduction in prevalence	46.2
50% reduction in growth rate	13.6
30% reduction in growth rate (+ incr. relative lag time)	14.9
3 - 4 log reduction in initial contamination levels	0.16
1 - 2 log reduction in initial contamination levels	0.67

The model predicted the most effective means of reducing risk of listeriosis (from Australian processed meats) is to reduce initial contamination levels ‘in-pack’ by methods such as pasteurisation. The authors comment however that ‘in-pack’ technology is unlikely to be available to the vast majority of small producers. The primary control therefore would involve use of antimicrobials to extend lag phase and prevent growth. Salts of organic acids can reduce risk but their benefit could be lost where the shelf-life is further extended.

3.1.4 Canada

A risk assessment concerning *L. monocytogenes* in Canada concentrated on pâté and semi-soft cheese and gave predicted human infection rates for these foods under a variety of scenarios in Canada (Farber *et al.*, 1996). Risk modelling to estimate numbers of cases was considered to predict reasonably well the actual numbers of cases, if it was assumed that 10-20% of cases were attributable to cheese consumption and the under-reporting rate for listeriosis was 10-100.

APPENDIX 4 CONTROL MEASURES OVERSEAS

4.1 Codex

The Codex “Guidelines on the Application of General Principles of Food Hygiene to the Control of *Listeria monocytogenes* in Foods” (CAC/GL 61-2007, http://www.codexalimentarius.net/download/standards/10740/CXG_061e.pdf) includes discussion of microbiological criteria for *L. monocytogenes* in ready-to-eat foods (Annex II). These criteria differentiate between foods in which growth will and will not occur. The document describes some food characteristics which will not permit growth of *L. monocytogenes*, but ultimately this needs to be determined and validated by food operators.

For foods in which growth will not occur the criterion is that 0 of 5 samples tested exceed 100 CFU/g. For foods in which growth will occur the criterion is that 0 of 5 samples tested show “Absence in 25 g (< 0.04 CFU/g)”.

These microbiological criteria were adopted by the Codex Alimentarius Commission at their Thirty Second Session meeting in mid-2009.

4.2 Australia

Australian State and Territory Food Acts have adopted the Australia New Zealand Food Standards Code in its entirety (see section 5.2.1). Controls and criteria fall under these legislative instruments.

Recall guidelines for Australia only are provided on the FSANZ website under the title ‘Recall Guidelines for Packaged Ready-to-eat foods found to contain *L. monocytogenes* at point of sale, April 2001’. For further information on the Australian recall system refer to; <http://www.foodstandards.gov.au/foodmatters/listeria/listeriarecallguidel1321.cfm>

This differentiation between ready-to-eat foods for which there is a zero tolerance for *L. monocytogenes*, and ready-to-eat foods which have no listericidal step, is also reflected in the recall guidelines (see Australian recall guide website cited above). Ready-to-eat salad would fall into category 2 below and would necessitate a recall where ≥ 100 CFU g⁻¹ were detected (Table 24).

Table 24: Food categories and action levels (applicable in Australia only)

Category of food	Level of <i>L. monocytogenes</i>	Action
<p>Category 1 ready-to-eat foods requiring refrigerated storage and able to support the growth of <i>L. monocytogenes</i>*;</p> <p>ready-to-eat foods that have been implicated in human listeriosis (e.g. soft & semi soft cheeses, pate, cooked cold chicken, cold-smoked fish**) and/or which may be consumed by at risk groups, especially infants</p>	<p>Detected in 25g[#] (Method: AS/NZS 1766.2.16.1-1998 for the detection of <i>L. monocytogenes</i>***)</p>	Recall
<p>Category 2 - all other packaged ready-to-eat foods</p>	<p>Equal to or greater than 100 CFU per gram (Method: No AS/NZS enumeration method;)</p>	Recall

* Factors such as freezing, pH, water activity, lactates and organic acids may inhibit the growth of *L. monocytogenes*. When it is difficult to predict whether a given food is supportive of growth for *L. monocytogenes* within the stated shelf-life, the authorities may take a conservative approach and regard growth as possible, unless there is documented evidence provided by the manufacturer that the product does not support growth of *L. monocytogenes*.

** The Joint Australia New Zealand *Food Standards Code* has a sampling plan for cold-smoked fish that allows one out of five samples to contain *L. monocytogenes* up to 100 CFU g-1

*** Equivalent methods may be used AS/NZS 4659.

10 or >10/g if an enumeration method is used.

4.3 United States of America

The United States of America has a zero tolerance for *L. monocytogenes* in ready-to-eat foods, including ready-to-eat meats (absence in 25g, which equates to < 0.04 CFU/g). This means that if the organism is detected within 25g of ready-to-eat food, the product is deemed to be adulterated. The zero tolerance policy adopted in the 1980s makes no distinction between foods contaminated at high or low levels, contamination at a detectable level is enough to deem the food as unfit. This current regulatory approach has been challenged because it concentrates on further reducing prevalence of the organism in ready-to-eat foods and continues zero-tolerance for all ready-to-eat foods.

A recently published draft consultation paper by the USFDA proposes to raise the zero tolerance limit to 100 CFU/g for foods that do not support the growth of the pathogen, see website, accessed 8 December 2009: <http://www.fda.gov/Food/GuidanceComplianceRegulatoryInformation/GuidanceDocuments/FoodProcessingHACCP/ucm073110.htm>

The US National Health Objectives for 2010 have specified goals for four foodborne pathogens, the target for *L. monocytogenes* is to reduce its incidence to 0.25 /100,000 by 2010 (baseline 1997 of 0.5/100,000 cases). An interim action plan in November 2003 was published by USFDA in order to facilitate this goal, see website; <http://www.cfsan.fda.gov/~dms/lmr2plan.html>.

4.4 Canada

Canada has implemented a three category system for *L. monocytogenes* in ready-to-eat foods (Farber *et al.*, 1996). This categorisation system is summarised in Table 25.

Table 25: The microbiological criteria for *L. monocytogenes* for different categories of food and corresponding action levels in Canada

Category	Foods	Microbiological criteria for <i>L. monocytogenes</i>	Action level
1	Foods causally linked to listeriosis, with a shelf-life >10 days.	absence in 50g	>0 CFU/50g Immediate action-Class I recall to retail level.
2	All other ready-to-eat foods capable of supporting growth, refrigerated shelf-life of >10 days.	absence in 25g	>0 CFU/25g Immediate action-Class II recall to retail level.
3 (two types of foods)	<ul style="list-style-type: none"> • supports growth with refrigerated shelf-life of <10 days • all other ready-to-eat foods not supporting growth; <ul style="list-style-type: none"> ➢ pH 5.0 – 5.5 and $a_w < 0.95$ ➢ pH <5.0 regardless of a_w ➢ $a_w \leq 0.92$ regardless of pH ➢ frozen foods. 	<p>$\leq 100 \text{ CFU g}^{-1}$ with adequate GMP</p> <p>$\leq 100 \text{ CFU g}^{-1}$ with inadequate or no GMP</p> <p>$> 100 \text{ CFU g}^{-1}$</p>	<p>Immediate action-allow sale. -follow up at plant level.</p> <p>Immediate action-consider class II recall or stop sale.-follow up at plant level.</p> <p>Class II recall or stop sale - follow up at plant level.</p>

4.5 England and Wales

A working group of the Public Health Laboratory Service (PHLS) in the UK produced revised guidelines for the microbiological quality of ready-to-eat foods (Gilbert *et al.*, 2000). These were developed as guidelines for the interpretation of laboratory results. For all ready-to-eat foods sampled at the point of sale, there are three guideline categories of results for *L. monocytogenes*:

- <20/g Satisfactory (some products with long refrigerated shelf lives have absence in 25g as satisfactory)
- 20-<100/g Acceptable
- ≥ 100 /g Unacceptable/potentially hazardous

Processed ready-to-eat meats specifically identified in the list of ready-to-eat foods included brawn, ham, sliced meats and salami and fermented meat products.

4.6 Denmark

Nørrung *et al.* describe the control of *L. monocytogenes* in Denmark (Nørrung *et al.*, 1999). The regulatory policy is based on HACCP and a health risk assessment approach. Ready-to-eat foods are categorised into six subsets with the following tolerances (Table 26).

Table 26: Food groups and tolerances for *L. monocytogenes* in Denmark

Category	Food groups	No. of samples (<i>n</i>)	Absence in 25g (<i>c</i>)	<i>m</i>	<i>M</i>
I	Foods heat treated in final package	5	0	0	-
II	Heat treated foods, handled after treatment. Shelf life > 1 week, food supports growth	5	0	0	-
III	Lightly preserved, not heat treated, shelf life > 3 weeks	5	0	0	-
IV	Heat-treated foods, handled after treatment. Stabilised against growth within shelf life	5	1	10*	100*
V	Lightly preserved, not heat treated, stabilised against growth during shelf life	5	1	10*	100*
VI	Raw, ready-to-eat foods	5	2	10*	100*

* denotes *L. monocytogenes* per g.

n represents the number of samples from each batch/lot required for examination,

c represents the maximum allowable number of defective samples after which whole batch/lot is rejected,

m represents the acceptable level above which samples are marginally acceptable,

M represents the values above this level are unacceptable. One or more sample to exceed this level causes rejection of whole batch/lot.

Levels above 100 CFU/g of *L. monocytogenes* are regarded as posing a health risk to consumers (Food Act s.12), control activities include prohibition of sale and recalls (Nørrung *et al.*, 1999).