



**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

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October 2002

Client Report
FW0186

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IN PROCESSED READY-TO-EAT MEATS**

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ACKNOWLEDGEMENTS

We would like to thank:

- The New Zealand Pork Industry Board for supplying information on pork production and processing.

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1 INTRODUCTION

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. The place of a risk profile in the risk management process is described in “Food Administration in New Zealand: A Risk Management Framework for Food Safety” (Ministry of Health/Ministry of Agriculture and Forestry, 2000). Figure 1 outlines the risk management process.

Figure 1: Risk Management Framework

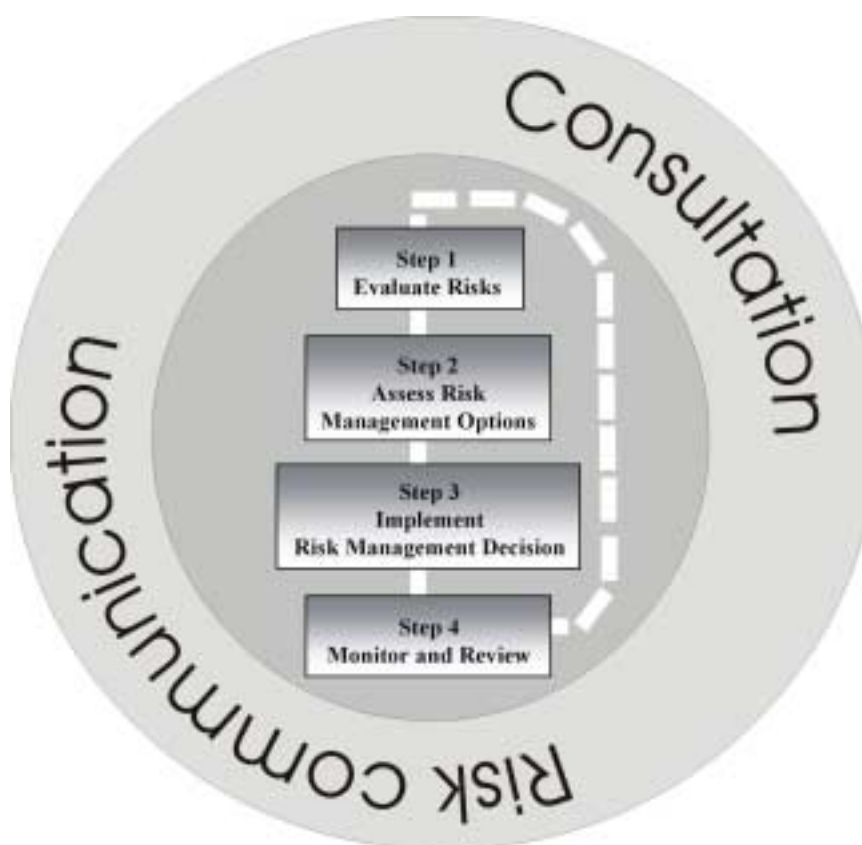


Figure reproduced from “Food Administration in New Zealand. A risk management framework for food safety” (Ministry of Health/Ministry of Agriculture and Forestry, 2000).

In more detail, the four step process is:

1. Risk evaluation

- identification of the food safety issue
- **establishment of a risk profile**
- ranking of the food safety issue for risk management
- establishment of risk assessment policy
- commissioning of a risk assessment
- consideration of the results of risk assessment

2. Risk management option assessment

- identification of available risk management options
- selection of preferred risk management option
- final risk management decision

3. Implementation of the risk management decision

4. Monitoring and review.

The Risk Profile informs the overall process, and provides an input into ranking the food safety issue for risk management. Risk Profiles include elements of a qualitative risk assessment. However, in most cases a full exposure estimate will not be possible, due to data gaps, particularly regarding the level of hazard in individual foods. Consequently the risk characterisation part of a risk assessment will usually rely on surveillance data.

The Risk Profiles also provide information relevant to risk management. Based on a Risk Profile, decisions are made regarding whether to conduct a quantitative risk assessment, or take action, in the form of gathering more data, or immediate risk management activity.

This Risk Profile concerns *Listeria monocytogenes* in processed ready-to-eat meats. This food/hazard combination was chosen for preparation of a detailed risk profile on the basis of the existence of notified cases of listeriosis in New Zealand, and the documentation of an outbreak of non-invasive listeriosis in February/March 2000 transmitted by corned silverside and ham (Whyte, 2000).

The sections in this Risk Profile are organised as much as possible as they would be for a conventional qualitative risk assessment, as defined by Codex (1999).

Hazard identification, including:

- A description of the organism
- A description of the food group

Hazard characterisation, including:

- A description of the adverse health effects caused by the organism.
- Dose-response information for the organism in humans, where available.

Exposure assessment, including:

- Data on the consumption of the food group by New Zealanders.
- Data on the occurrence of the hazard in the New Zealand food supply.
- Qualitative estimate of exposure to the organism (if possible).
- Overseas data relevant to dietary exposure to the organism

Risk characterisation:

- Information on the number of cases of adverse health effects resulting from exposure to the organism with particular reference to the food (based on surveillance data)
- Qualitative estimate of risk, including categorisation of the level of risk associated with the organism in the food (categories are described in Appendix 1).

Risk management information

- A description of the food industry sector, and relevant food safety controls.
- Information about risk management options.

Conclusions and recommendations for further action

Note: Earlier versions of this document were produced as part of a project undertaken by ESR and jointly directed by the Ministry of Health and the Ministry of Agriculture and Forestry. Ministry responsibilities for food safety were combined into the New Zealand Food Safety Authority (NZFSA) in July 2002.

The Australia New Zealand Food Authority (ANZFA) became Food Standards Australia New Zealand (FSANZ), also in July 2002.

Information and reports published by the older organisations have been referenced to those names.

2 HAZARD IDENTIFICATION: THE ORGANISM

The following information is taken from a data sheet prepared by ESR under a contract for the Ministry of Health. The data sheet is intended for use by regional public health units.

2.1 *Listeria monocytogenes*

2.1.1 The organism/toxin

Six species of *Listeria* bacteria have been recognised (ICMSF, 1996). Two are considered non-pathogenic, while *L. seeligeri*, *L. ivanovi*, and *L. welshimeri* rarely cause human infection. This leaves *L. monocytogenes* as 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.

Note that in microbiological terms "D" refers to a 90% (or decimal or 1 log cycle) reduction in the number of organisms.

2.1.2 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).

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.

2.1.3 Inactivation (CCPs and Hurdles)

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.

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

In other meats of around 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.

Radiation: D values depend on the food and temperature and range from 0.34 to 2 kGy. A dose of 3 kGy does not remove *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.

2.1.4 Sources

Human: *L. monocytogenes* is carried asymptotically in the faeces of 2-6% of the population. Person-to-person spread (other than mother to foetus) not often recorded but has been recognised. Up to 30% of case contacts may carry the organism. *L. monocytogenes* is shed in high numbers ($\geq 10^4$ /g) in the faeces of infected people.

Animal: Can cause disease in animals, and veterinarians were originally considered to be an at risk group. *Listeria* 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 regarding numbers exists, but is generally 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. Has been isolated from toothbrushes and other domestic environments.

Transmission routes: One study estimates that 1/3 of cases are foodborne. Other reports describe foodborne transmission as the primary source of human infections. Alternative routes include infections acquired in hospital and occupational exposure (e.g. veterinarians).

3 HAZARD IDENTIFICATION: THE FOOD

3.1 Relevant Characteristics of the Food: Processed Ready-to-Eat Meats

Ready-to-eat meats are products whose processing includes one or more pathogen reduction steps to render the products safe for consumption without further heating 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 may present hazards different to those of red meats, but ready-to-eat poultry products will usually be processed, sold and consumed in the same way as red meat products. Consequently the information included here will also be relevant to processed ready-to-eat poultry products.

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

- addition of flavouring (spices etc);
- addition of binders, extenders and emulsifiers (isolated soya protein or milk proteins, gums, etc);
- heating (pasteurising) (cooking, baking, boiling, steaming);
- curing;
- smoking;
- fermentation;
- drying;
- comminution;
- vacuum or modified atmosphere packaging; and,
- refrigerated or frozen storage.

The types of processed ready-to-eat meats are many. Examples are given below, grouped according to processing (ICMSF, 1998).

- Raw cured shelf stable meats (raw hams and some low-acid dry sausage and high acid fermented sausage where low a_w or a combination of low pH and reduced a_w provides microbial stability): dry cured hams, Chinese sausage, fermented high acid sausage, salami, pepperstick, biersticks, mettwurst, rockwurst. Any *L. monocytogenes* present are reduced during normal fermentation, drying and ripening and the small number of cells in the finished product cannot multiply. Fermented sausages rely on both a reduced pH (4.6–5.3) and a reduced water activity (a_w) of <0.95 for microbial stability. Chopped meat together with salt (2.5-3%), sodium nitrite, sugar (0.4-0.7%) and spices are fermented in moisture permeable casing. The sausages are then dried and may be smoked.
- Dried meats (microbiologically stable at ambient temperatures because of their low a_w): biltong, Rou Gan, beef jerky.
- Cooked perishable uncured meats (cooking process will effectively destroy vegetative bacterial pathogens): some roast beef and other cooked meats not reheated before consumption.
- Cooked perishable cured meats (contain up to 125 mg/kg of nitrite before heating, are cooked to about 65-75°C and require refrigerated storage): pressed ham, emulsion style sausages, pastrami, whole hams, silverside, corned beef, continental sausages, luncheon meats, saveloys, cocktail sausages etc., frankfurters, pâté, liverwurst.

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 creates the risk during any subsequent period of storage before consumption. *L. monocytogenes* bacteria are present in the environment of many food-processing and retail food facilities, and their 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).

3.1.1 Curing

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. The maximum residual sodium nitrite in the finished product is limited to 125 ppm in wet cures and 200 ppm for dry cured products in the New Zealand Food Regulations 1984. A sodium nitrite concentration of 120 ppm is usually sufficient for controlling bacterial growth.

Safety concerns have been expressed in the past with the use of nitrite/nitrate in foods. Nitrous acid, which is formed by the breakdown of nitrite, binds with amines in proteins to produce N-nitrosamines, which are known human carcinogens. N-nitrosamines have been isolated in small amounts from nearly all cured meat products. Research has been conducted into processing methods that would eliminate N-nitrosamines from cured meats, but still allow retention of nitrite because of its action in preventing the growth of *Clostridium botulinum* and other food poisoning organisms. It has generally been conceded that the risks from low levels of N-nitrosamines are less than those from botulism poisoning, which may occur if nitrites were removed from the cure. The addition of erythorbate and or ascorbate also reduces the formation of N-nitrosamines, so they are normally included (Pearson and Gillett, 1996).

3.2 **The Food Supply in New Zealand**

3.2.1 Production

The largest component of processed ready-to-eat meat consumption in New Zealand is ham (see Section 5.2). The production of pig meat in New Zealand in 2000 was approximately 46,000 tonnes, supplemented by approximately 21,000 tonnes of imported pigmeat (New Zealand Pork Industry Board, 2001). 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.

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

3.2.2 Imported foods

According to data from Statistics New Zealand, for the year to September 2001 New Zealand imported 2859 tonnes of pig meat from Australia, 8746 tonnes from Canada, 782 tonnes from Denmark and 284 tonnes from the United States. All were frozen meat carcasses and cuts. MAF Biosecurity requires that imported pork is cooked and frozen, or else frozen and imported into a transitional facility where it is cooked.

New Zealand imports relatively small amounts of processed meats. For the year to September 2001, 2287 tonnes of meat preparations in airtight cans or jars (mostly corned beef) were imported. During the same period 2483 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 4770 tonnes, 4110 tonnes 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.

4 HAZARD CHARACTERISATION: ADVERSE HEALTH EFFECTS

There are two types of disease associated with 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.

4.1 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 perinatal group includes foetuses or neonates, and infection can occur before or after birth. The symptoms experienced by the mother are usually only a mild fever.

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.

4.2 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.

4.3 Dose Response

4.3.1 Listeriosis

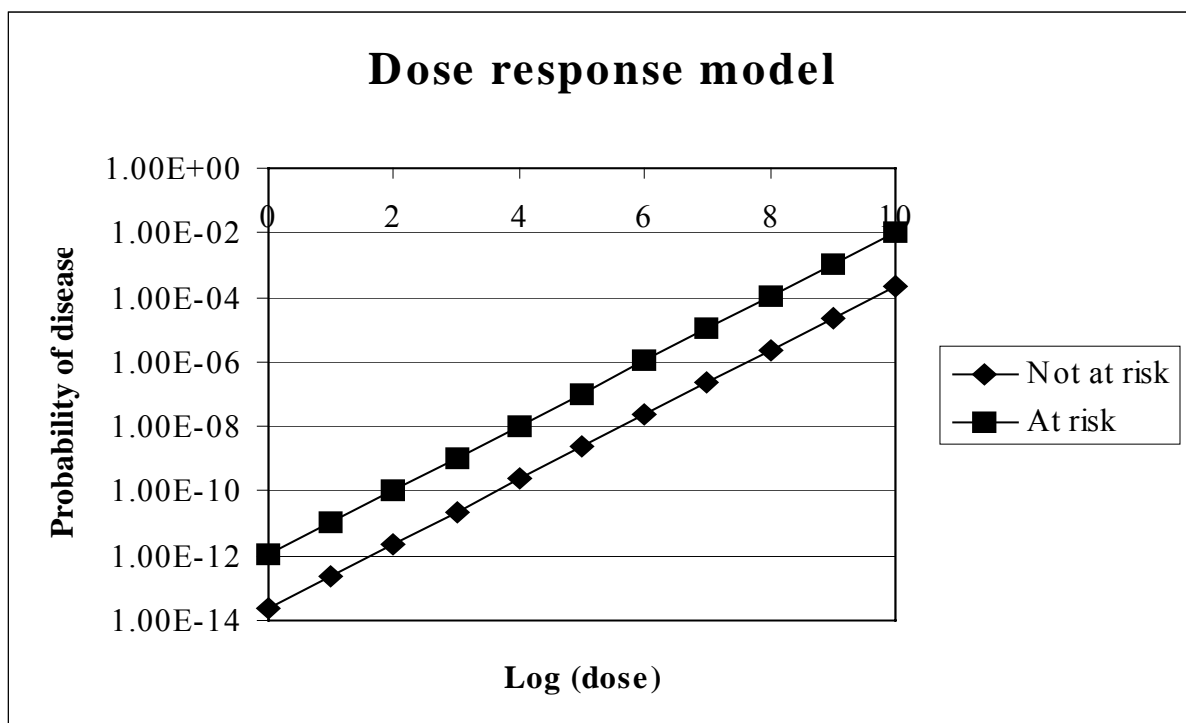
It is becoming increasingly realised that the only completely safe dose of *L. monocytogenes* is zero, even in healthy people. However the probability of invasive disease following exposure to even moderate levels of cells is very low.

The FAO/WHO risk assessment used a dose response model described by:

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

Where R is a variable that defines the dose/response relationship and N is the number of cells consumed. The values of R vary depending on population group (to reflect different susceptibilities) but are around the 10^{-12} - 10^{-14} level. 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 2 shows dose response curves for at risk and not at risk groups.

Figure 2: 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.

Given the median daily intake of ready-to-eat meats (34.5g) (see Section 5.2), a dose of 100/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 at risk consumers and 2.37×10^{-14} for those not at risk) results in a risk of:

- 3.66×10^{-9} per day for consumers in the at risk group
- 8.18×10^{-11} per day for consumers in not at risk at risk 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.

4.3.2 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*/g became ill with symptoms of febrile gastroenteritis (Sim *et al.* In Press). 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.* (1997) an attack rate of 75% was recorded where the median population consumed was estimated as being as high as 2.9×10^{11} cfu. 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 cause febrile non-invasive gastrointestinal disease, and because this organism is not routinely screened for in clinical laboratories many cases of non-invasive listeriosis may evade detection.

4.4 High Risk Groups in the New Zealand Population

Although there is increasing evidence that healthy individuals can become infected by *L. monocytogenes*, there are some high risk groups in the population (Sutherland and Porritt, 1997). The well categorised 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. The following sections provide information on the New Zealand population of these groups.

4.4.1 Perinatal population

Live births data for the 2001 Calendar year were 56,221 (<http://www.stats.govt.nz/>).

Births were spread evenly throughout the year, but were strongly weighted towards the Northern areas of New Zealand. This total compares well with the results of the 2001 Census, which reported 55,130 New Zealanders under the age of one year on Census night. Of these 51.3% were male and 48.7% female. This represents 1.4% of the total New Zealand population.

Given that there were seven perinatal cases in 2000, and there were (presumably) around 56,000 live births, this equates to an incidence of approximately 12 cases/100,000/year in the perinatal population.

4.4.2 Elderly population

According to the 2001 Census of New Zealand 615,580 New Zealanders were aged 60 years or over. This is 16.0% of the total population. The aged population is 45.2% male and 54.8% female. The population 80 years and over is 112,090 (2.6% of the population) and is made up of 34.3% males and 65.7% females (<http://www.stats.govt.nz/>).

4.4.3 Immune compromised

AIDS: At the end of June 2001, 741 people in New Zealand were notified with AIDS. At the same date 1513 people in New Zealand were found to be infected with HIV (<http://www.moh.govt.nz/aids.html>). This represents 0.04% of the total New Zealand population.

Cancer: The most recently available statistics on the incidence of cancer and cancer mortality in New Zealand are from the 1998 year. In that year, 16,531 new cases of cancer were registered (311.9 cases per 100,000 population), made up of 8,842 males (357.0 cases per 100,000) and 7,689 females (279.6 cases per 100,000). During the same period mortality due to cancer was 7,582 (131.9 cases per 100,000) made up of 3911 males (152.4 per 100,000) and 3671 females (117.6 per 100,000). (<http://www.nzhis.govt.nz/stats/cancerstats.html>). It is uncertain what proportion of the New Zealand population are suffering from cancer at any particular time.

Recipients of organ or tissue donations: The NZHIS publication “Selected morbidity data for publicly funded hospitals 1997/98” lists only two patients under the category “V42 Organ or tissue replacement by transplant” and only five patients under the category “V43 Organ or tissue replacement by other means”. A similar document covering private hospital morbidity during 1995 reported 57 corneal transplants, 21 cases of transplantation of muscle and tendon of the hand, but no major organ transplants (<http://www.nzhis.govt.nz>).

However, this is an obvious underestimate as, presumably, a number of renal, heart and other transplants take place in New Zealand. Some information on major organ transplants can be obtained from diverse sources of information. An Australian summary indicates that the kidney is the most common organ transplanted, followed by liver, lung or heart-lung, heart and pancreas (<http://www.abs.gov.au/ausstats>).

In 2000, 106 kidney transplants were performed in New Zealand bringing the total number of surviving New Zealand kidney transplant recipients to 1014 (<http://www.anzdata.org.au>). In 2001, 36 liver transplants were performed at the Auckland liver transplant unit. The unit reported outcome statistics for 109 liver transplant recipients, but it is unclear whether this is the total surviving New Zealand population (<http://www.nzliver.org/outcomes>). The New Zealand Organ Donation website gives the following numbers for transplants performed in 2001; kidney (excluding living donor transplants) 67, liver 36, heart 15, lungs 12, pancreas 3 (<http://www.donor.co.nz>). It appears likely that the total New Zealand population of surviving major organ transplant recipients is less than 2000 people (0.05% of the total population).

5 EXPOSURE ASSESSMENT

5.1 The Hazard in the New Zealand Food Supply: *Listeria* in Processed Ready-to-Eat Meat Products

Available information on the presence of *Listeria* species in general, and *L. monocytogenes* in particular, in ready-to-eat meats in New Zealand has been summarised in Table 1. The same type of data for ready-to-eat poultry products are given in Table 2.

Data for New Zealand are comparable with foods from similar countries around the world (see Section 5.4). There has been a suggestion that heightened awareness of listeriosis could have resulted in a decrease in the prevalence of *Listeria* in foods since the 1991 and 1993 data. In some industries or companies this may be true, but is unlikely to be applicable in blanket terms. The incidence of notified cases of listeriosis in New Zealand (Table 10) does not indicate a decrease in incidence since 1993.

Published quantitative data for New Zealand ready-to-eat meats are not available and this represents a significant data gap.

Table 1: Reported prevalence of *Listeria* in meat products in New Zealand

Meat product	Samples tested	Positive <i>L. monocytogenes</i> (%)	Positive any <i>Listeria</i> species (%)	Year	Reference
RTE meat products cooked in packaging	54	NS	0.0	1991	Hudson <i>et al.</i> , 1991
Fermented meats	39	NS	7.7		Hudson <i>et al.</i> , 1991
Packaged RTE that had been handled (e.g. sliced)	36	NS	33.0		Hudson <i>et al.</i> , 1991
Ready-to-eat meats from delicatessens	47	NS	38.3		Hudson <i>et al.</i> , 1991
RTE pork products	34	2.9	50.0	1992	Hudson <i>et al.</i> , 1992
RTE beef products	18	0	50.0		Hudson <i>et al.</i> , 1992
RTE lamb products	3	0	67.0		Hudson <i>et al.</i> , 1992
RTE mixed meat products	76	10.5	22.7		Hudson <i>et al.</i> , 1992
Jellied meats	6	50.0	NS	1992	Ministry of Health, 1993
Roast meats	6	33.3	NS		Ministry of Health, 1993
Ham	32	37.5	NS		Ministry of Health, 1993
Meat loaf	3	33.3	NS		Ministry of Health, 1993
Corned beef/silverside	13	30.8	NS		Ministry of Health, 1993
Luncheon	20	25	NS		Ministry of Health, 1993
Pre cooked sausages	39	7.7	NS		Ministry of Health, 1993

RTE = Ready-to-eat

NS = Not stated

Table 2: Reported prevalence of *Listeria* in ready-to-eat poultry products in New Zealand

Meat product	Samples tested	Positive <i>L. monocytogenes</i> (%)	Positive any <i>Listeria</i> Species (%)	Year	Reference
RTE Turkey products	6	0	50.0	1992	Hudson <i>et al.</i> , 1992
RTE chicken products	16	12.5	43.8	1992	Hudson <i>et al.</i> , 1992

RTE = Ready-to-eat

5.2 Food Consumption: Processed Ready-to-Eat Meats

The following information is taken from the New Zealand National Nutrition Survey (NNS) conducted in 1997. While the 1997 NNS is 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 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 1997 NNS does not generally provide sufficient information to make clear judgement calls as to whether situations such as those described above may apply.

The following decision rules were applied to analysis of the 1997 NNS data:

- 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.
- 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.

Summary food consumption statistics can be expressed in terms of ‘consumer’ (just those people reporting to eat a particular food) or ‘persons’ (the whole population). Both will be presented here. The age groups used by the 1995 Australian National Nutrition Survey (Australian Bureau of Statistics, 1999) will initially be used so that an easy point of comparison can be made. These are 16-18 years, 19-24 years, 25-44 years, 45-64 years, 65 years and over. Ready-to-eat meat was assumed to be analogous to the Australian NNS

category of ‘Processed meat’. Table 3 gives the percentages of different age-sex groups within the population who reported consuming RTE meats during the 24 hour survey period. These figures are generally interpreted as the percentage of the population consuming RTE meats on any given day.

Table 3: Total ready-to-eat meats – percentage of respondents consuming

Age (years)	16-18	19-24	25-44	45-64	65+	Total
Male	21.1	15.9	30.3	23.3	22.1	25.2
Female	16.1	21.1	18.5	21.3	19.1	19.4
Total	18.3	18.9	23.1	22.2	20.3	21.8

These figures are generally higher than observed in the 1995 Australian NNS (Australian Bureau of Statistics, 1999) which reported 8.5-13.7% of respondents consuming processed meat, depending on the age group. The pattern of respondents with respect to age is similar to that seen in Australia, with typically higher percentages of people eating processed meat in the 25+ age groups, than in the younger age groups.

Table 4 summarises the median daily consumption of RTE meats by consumers only. If RTE meats were only eaten once during the day these figures would represent serving sizes, however, in some cases individuals may consume several servings of RTE meat during the day.

Table 4: Total ready-to-eat meats – median (50th percentile) consumption by consumers (g/day)

Age (years)	16-18	19-24	25-44	45-64	65+	Total
Male	56.0	42.0	50.0	37.5	31.6	44.0
Female	47.0	27.6	28.0	30.0	31.5	28.8
Total	50.0	34.5	34.6	33.9	31.6	34.5

The median amounts of ready-to-eat meats eaten by consumers are very similar to those reported for the 1995 Australian NNS (Australian Bureau of Statistics, 1999). The Australian study reported an overall median (males and females) for respondents aged 19 and over of 34.4 g/day, compared to an overall median of 34.5 g/day from the New Zealand NNS (1997). Overall figures for males and females are also very comparable (males; Australia 39.7 g/day, New Zealand 44.0 g/day, females; Australia 28.8 g/day, New Zealand 28.8 g/day).

Median daily consumption may represent one or more servings of ready-to-eat meats. The USDA risk assessment for *L. monocytogenes* (USDA, 2001) 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.

Table 5 summarises the 95th percentile levels of daily consumption of RTE meat for consumers only. These levels of consumption represent high level consumers of this food product and are often used when considering ‘worst-case scenarios’ for dietary exposure.

Table 5: Total ready-to-eat meats – 95th percentile consumption by consumers (g/day)

Age (years)	16-18	19-24	25-44	45-64	65+	Total
Male	141.5	214.1	223.7	138.0	116.2	175.6
Female	72.9	100.0	106.4	111.4	121.8	108.4
Total	106.4	142.0	172.6	125.3	120.0	138.3

Table 6 gives the mean level of daily consumption of RTE meats for the whole population.

Table 6: Total ready-to-eat meats – mean consumption by persons (g/day)

Age (years)	16-18	19-24	25-44	45-64	65+	Total
Male	14.2	10.9	22.1	12.3	9.4	15.7
Female	7.1	8.9	7.5	8.9	8.4	8.1
Total	10.3	9.7	13.2	10.5	8.8	11.2

The higher percentage of New Zealanders who reported consuming ready-to-eat meats than Australians consuming processed meat produces a large discrepancy in mean consumption values by person (Australia 5.4 g/day compared to New Zealand 11.2 g/day).

The major types of ready-to-eat meat contributing to this total are:

- Ham, 35% of total
- Luncheon meat, 16% of total
- Beef, corned (silverside, brisket, canned, etc), 15% of total
- Beef, roast, 5% of total
- Salami, 5% of total

5.3 Qualitative Estimate of Exposure

5.3.1 Number of servings of RTE meat and serving size

5.3.1.1 Total population

From the NNS, 1136 individual dietary records were deemed to represent consumption of a serving of RTE meat. Using a total survey population of 4636 and a total New Zealand population of 3,850,100 (<http://www.stats.govt.nz/>):

$$\begin{aligned} \text{Annual number of servings (total population)} &= 1136 \times 3,850,100 / 4636 \times 365 \\ &= 3.44 \times 10^8 \text{ servings} \end{aligned}$$

This compares to 2.91×10^{10} servings of RTE 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 90 (NZ).

5.3.1.2 Elderly population

From the NNS, 241 individual dietary records were deemed to represent consumption of a serving of RTE 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 2001 Census 615,580 New Zealanders were aged 60 years or more.

$$\begin{aligned}\text{Annual number of servings (elderly population)} &= 241 \times 615,580 / 1087 \times 365 \\ &= 4.98 \times 10^7 \text{ servings}\end{aligned}$$

5.3.1.3 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; 56,221 in 2001 as a percentage of the 2001 total population gives a birth rate of 1.46% compared to the US rate of 1.5%) and dividing by 12, to estimate the number of women in the last month of pregnancy.

$$\begin{aligned}\text{Annual number of servings (perinatal population)} &= 3.44 \times 10^8 \times 0.0146/12 \\ &= 4.19 \times 10^5 \text{ servings}\end{aligned}$$

5.3.1.4 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.

$$\text{Annual number of servings (intermediate population)} = 2.94 \times 10^8 \text{ servings}$$

Based on the data in the NNS database the 50, 75, 95, and 99th percentile serving sizes for RTE meats in New Zealand were:

Percentile	Serving size (g)
50	30.0
75	56.0
95	125.0
99	275.0

For comparison, the USDA risk assessment determined the following serving sizes for the same percentiles (figures are averages for four types of RTE 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.

5.3.2 Contamination frequency

There are no recent data that would allow the assignment of an overall level of contamination to RTE meat products in New Zealand. However, historical data and more recent information from overseas would suggest a moderate level of contamination (1-10% of samples).

5.3.3 Predicted contamination level at retail

No New Zealand data are available on levels of *L. monocytogenes* in RTE meats at retail. While some information is available on levels of *Listeria* in RTE meats associated with suspected foodborne illness incidents, this is not appropriate for assessing population level exposure.

5.3.4 Growth rate during storage and most likely storage time

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

5.3.5 Heat treatment

Not applicable to RTE meat products.

5.3.6 Exposure summary

Consumption of RTE meats in New Zealand appears to be similar to the frequency of consumption determined for the US population. These foods constitute a commonly eaten food in New Zealand. The very limited amount of New Zealand information available suggests that the frequency of *Listeria* contamination of RTE meat products is similar to that observed in a number of overseas studies.

While some information is available on the frequency with which *Listeria* is present in New Zealand RTE meats, no information is available on the levels which may be present. The storage characteristics of these foods suggest that there is potential for the organism to multiply during storage. This potential reinforces the need for quantitative information on *Listeria* levels in RTE meats.

5.4 Overseas Context

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 7.

Most of the prevalence values given fall within the range of 0-20% positive, although in a few cases the prevalences are up to almost 80%.

The prevalences given in the table are also underestimates of the true rates of contamination. 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 reached. Even if the “five unit sampling plan” is adhered to, the chances of detection of a contaminated batch are poor. The proportion

of contaminated units within a batch, as measured in 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 most seriously contaminated batches, allowing other contaminated batches on to the market.

Information from the scientific literature on the prevalence of *Listeria* species in general, and *L. monocytogenes* in particular, in ready-to-eat poultry has been summarised in Table 8. 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 have been summarised in Table 9.

Summarising this information is difficult as in some cases only the data for positive samples were given. However, it is the general trend that samples which are contaminated by *L. monocytogenes* are usually contaminated at quite low numbers (regarded here as being <10/g). Generally, only a few percent or a fraction of a percent of samples are at levels 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, e.g. *L. monocytogenes* is unlikely to grow on fermented salami, but is likely to grow on roast beef.

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. If a product with a long shelf life is contaminated prior to vacuum packaging, then 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 which may be attained will be higher. Recent evidence from the outbreak in New Zealand in 2000 support this observation (Sim *et al.*, in press). Here the ham involved was produced in December, yet was eaten in March. This represents a two to three month shelf life. This ham caused 21 people out of a total of 24 consumers to become sick with febrile non-invasive gastroenteritis and the number of *L. monocytogenes* present on the ham was $>10^7$ /g.

Table 7: Reported prevalence of *Listeria* in overseas meat products

Country	Meat	Samples tested	Positive <i>L. monocytogenes</i> (%)	Positive any <i>Listeria</i> species (%)	Year	Reference
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 André, 1991
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
Italy	Salami, pressed pork	243	0.2	NS	1996	Grau, 1996
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, 1989
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

Country	Meat	Samples tested	Positive <i>L. monocytogenes</i> (%)	Positive any <i>Listeria</i> species (%)	Year	Reference
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
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

Table 8: Reported prevalence of *L. monocytogenes* and *Listeria* species in overseas ready-to-eat poultry meat products

Country	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 André, 1991
Denmark	Ready-to-eat turkey products	55	7.3	NS	2000	Ojeniyi <i>et al.</i> , 2000
UK	Ready-to-eat chilled chickens and portions	758	6.0	16.0	1997	MAFF, 1997
UK/US	Ready-to-eat poultry	527	12	NS	1996	Hitchins, 1996

Table 9: Quantitative data for *L. monocytogenes* in overseas ready-to-eat meats

Country	Food	Count breakdown	No. sampled or number positive	Reference
Belgium	Pâté	96.5% <0.04/g (-ve) 2.9% <10/g 0.3% 10-10 ² /g 0% 10 ² -10 ³ /g 0.3% 10 ³ -10 ⁴ /g	376	Arts and André, 1991
Belgium	Sausages (ready-to-eat)	95.4% <0.04/g (-ve) 3.3% <10/g 0.8% 10-10 ² /g 0% 10 ² -10 ³ /g 1.6% 10 ³ -10 ⁴ /g	241	Arts and André, 1991
Belgium	Hams	83.6% <0.04/g (-ve) 9.8% <10/g 1.6% 10-10 ² /g 1.6% 10 ² -10 ³ /g 3.2% 10 ³ -10 ⁴ /g	61	Arts and André, 1991
Belgium	Steakburgers	94.7% <0.04/g (-ve) 5.3% <10/g	38	Arts and André, 1991
Belgium	Other meat products	83.5 <0.04/g (-ve) 14.9% <10/g 0% 10-10 ² /g 0.8% 10 ² -10 ³ /g 0.8% 10 ³ -10 ⁴ /g	121	Arts and André, 1991
Belgium	Chicken products	83.0% <0.04/g (-ve) 5.7% <10/g 1.9% 10-10 ² /g 3.8% 10 ² -10 ³ /g	53	Arts and André, 1991

Country	Food	Count breakdown	No. sampled or number positive	Reference
		1.9% 10^3 - 10^4 /g 3.8% 10^3 - 10^4 /g		
Denmark	Heat-treated meat products	5% samples +ve in 25g 1.5% 10-100/g 1.4% >100/g	45	Nørrung <i>et al.</i> , 1999
England	Ready-to-eat meat products (mostly salami)	96.7% <0.04/g (-ve) 3.3% <100/g	455	MAFF, 1997
England	Ready-to-eat chicken and chicken portions	84.2% <0.04/g (-ve) 15.1% <100/g 0.7% >100/g	758	MAFF, 1997
England and Wales	Pâté	98% <0.04/g (-ve) 1.5% <200/g 0.06% 200 - 10^3 /g 0.12% 10^3 - 10^4 /g 0.12% 10^4 - 10^5 /g 0.12% > 10^6 /g	1,804	Nichols <i>et al.</i> , 1998
Germany	Ready-to-eat meat products	13.7% 0.04-1 7.8% 1 - 10^2 /g 1.4% 10^2 - 10^4 /g 0.2% > 10^4 /g	NS	Notermans <i>et al.</i> , 1998
Wales	Pâté	65% <0.04/g (-ve) 30% samples +ve 5% > 10^4 /g	216	Morris and Ribeiro, 1989

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

6 RISK CHARACTERISATION

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. Consequently this section is principally concerned with invasive listeriosis.

6.1 Adverse Health Effects in New Zealand

6.1.1 Incidence

Notification and mortality data from the EpiSurv database for listeriosis for the years 1990 to 2000 are given in Table 10. It is important to note that these cases are not associated with any specific transmission vehicle.

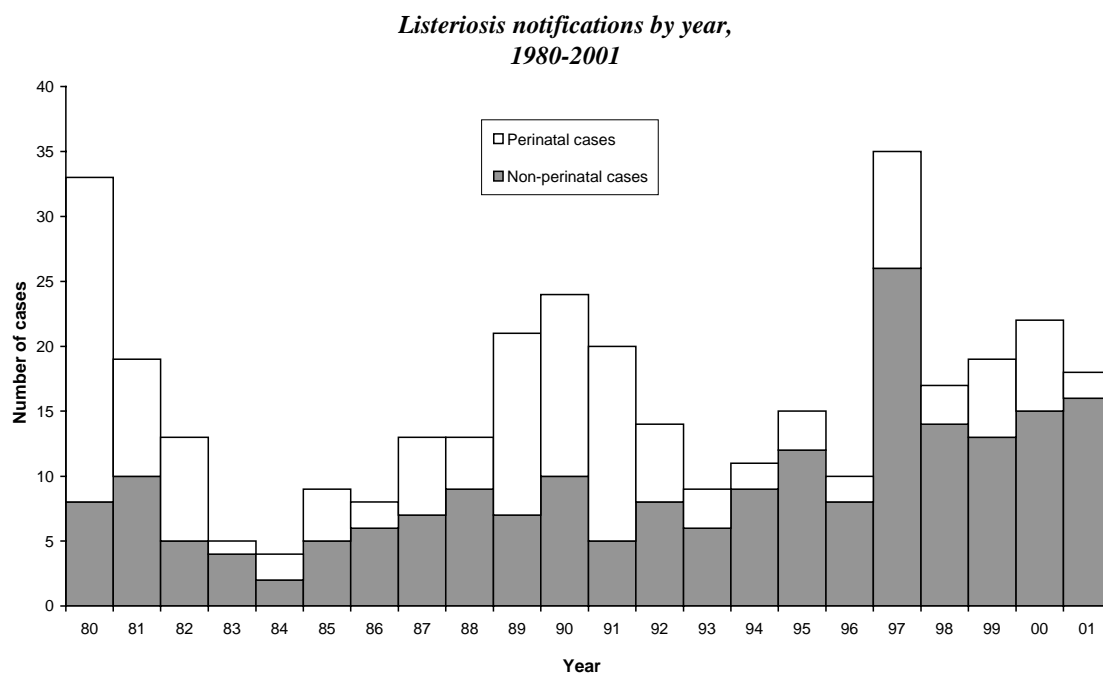
Table 10: Number of reported cases of invasive listeriosis and mortality from 1990 to 2001 (Kieft *et al.*, 2000; Lopez *et al.*, 2001; Sneyd *et al.* 2002)

Year	Listeriosis cases	Deaths (perinatal)	Deaths (non-perinatal)
1990	16	2	NA
1991	26	1	NA
1992	16	0	NA
1993	11	2	NA
1994	8	0	NA
1995	13	1	0
1996	10	1	0
1997	35	6	2
1998	17	0	0
1999	19	2	1
2000	22	4	2
2001	18	1	1

NA = Not Available

Figure 3 shows a graphical representation of annual case numbers of listeriosis with the proportions of perinatal and non-perinatal cases identified.

Figure 3: Listeriosis notifications by year 1980 – 2001



Reproduced from Sneyd et al. (2002)

6.1.2 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 1997-2001 are given in Table 11. 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 11: Outcome data for listeriosis in New Zealand

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

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.

6.1.3 Listeria in ready-to-eat meats – information from Ministry of Health’s suspect foodborne illness investigation programme

The Ministry of Health’s Suspect Foodborne Illness Investigation Programme provides investigative analyses to Public Health Units and provides a means of collating such investigations. The programme is funded by the Ministry of Health and provided by ESR. It contains information relating particular foods to episodes of suspected foodborne illness. This may be due to the fact that it is a genuine risk factor related to the symptoms presented, or may be due to preconceptions of the person experiencing the illness or the investigating officer. If the laboratory investigation identifies a known food pathogen in the suspect food at levels sufficient to cause illness and the symptoms known to be caused as a result of infection by the organism are consistent with the case details then the food may be identified as confirmed. Less compelling evidence may be provided in cases where a known pathogen is identified in faecal specimens associated with the suspected foodborne illness episode but not from the food samples provided (in some cases food samples may not have been provided, but a food may still be suspected).

Details of suspect foodborne illness episodes in which ready-to-eat meats were implicated during the 1997/98, 1998/99 and 1999/00 years are summarised in Table 12. In this period six episodes of listeriosis associated with ready-to-eat meats were identified. These included three of the cases involved in the outbreak described below (Sim *et al.*, in press), as well as one case each associated with corned beef, ham and saveloys/ham. Relatively few incidents are confirmed by testing of food and faeces. Given the low numbers of episodes involved, *L. monocytogenes* was involved in a significant proportion, 2 of 10 episodes confirmed from faecal samples and 6 from 8 episodes confirmed from food samples. This may be a reflection of the distinctive nature of the disease, especially in cases of non-invasive febrile gastroenteritis (the same argument might be applied to *Staphylococcus aureus*, which was detected in 3 of 10 faecal samples).

6.1.4 Outbreaks

Outbreaks of infection with *L. monocytogenes* in New Zealand are rare. From 1997 to 2001 only three have been reported. An earlier small outbreak, in 1992, was linked to smoked mussels (Brett *et al.*, 1998).

An outbreak of non-invasive gastroenteritis commenced in early 2000 (Sim *et al.*, in press; Whyte, 2000). The outbreak concerned 26 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 1.8×10^7 /g being counted in the ham. All isolates were of the same serotype and pulsed field gel electrophoresis (PFGE) type. This is only the second outbreak of illness caused by *L. monocytogenes* in New Zealand where a food has been identified with certainty (the first was caused by smoked mussels (Brett *et al.*, 1998) and is therefore outside the scope of this profile).

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 (see above).

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

Table 12: Summary of results from the Ministry of Health's suspect foodborne illness investigation programme, 1997-2000: Episodes associated with ready-to-eat meats

Ready-to-eat meat	Number of episodes	Number of cases	Number of confirmed episodes (faeces)	Number of confirmed episodes (food)
Ham (including ham rolls)	39	90	3 (1 x <i>Staphylococcus aureus</i> , 1 x <i>Clostridium perfringens</i> , 1 x <i>Campylobacter</i>)	3 (All <i>Listeria</i>)
Saveloys/ frankfurters/ cocktail sausages	11	15	1 (<i>Salmonella</i>)	2 (1 x <i>Salmonella</i> , 1 x <i>Listeria</i>)
Luncheon	7	12	1 (<i>Staphylococcus aureus</i>)	0
Salami	6	8	1 (<i>Staphylococcus aureus</i>)	0
Corned beef/silverside	5	17	2 (Both <i>Listeria</i>)	3 (All <i>Listeria</i>)
Pork, lamb ready-to-eat	4	5	2 (Both <i>Clostridium perfringens</i>)	0
Black pudding	3	5	0	0
Meatloaf	3	3	0	0
Ready-to-eat meats, unspecified	3	7	0	0
Pepperoni	2	2	0	0
Other (pastrami, beef jerky)	2	2	0	0

6.2 Adverse Health Effects Overseas

6.2.1 Incidence

Comparisons of listeriosis rates between countries must be made cautiously, as reporting practices may differ. However, the data in Table 13 indicate that New Zealand's rate is similar to that of other developed countries.

Table 13: Comparison of listeriosis incidence between countries

Country	Period	Rate /100,000	Reference
New Zealand	1999	0.5	Kieft <i>et al.</i> , 2000
New Zealand	2000	0.6	Lopez <i>et al.</i> , 2001
New Zealand	2001	0.5	Sneyd <i>et al.</i> , 2002
Australia (WA)	1993-1998	0.1-1.1	Health Department of Western Australia, 1998
Canada	1990-1998	0.1-0.3	Health Canada, 2000
Denmark	1999	0.8	Dansk Zoonosecenter, 2000
France	1997	0.4	De Valk <i>et al.</i> , 1998
UK	1983-2000	Approx. 0.2 - 0.5	PHLS, 2001
USA	1998	0.6	Anonymous, 2000a

Rates are compared with the legislative situation in various countries in Section 7.1.3.

6.2.2 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 14 and 15 respectively. Data on the contribution of *L. monocytogenes* to overall foodborne disease outbreaks and incidents overseas is given in Table 16, while Table 17 summarises case control studies on *L. monocytogenes* in ready-to-eat meats.

When outbreaks of listeriosis occur, they often involve a large number of cases. For example three of the nine meat product related outbreaks in Table 14 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 what is 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 that relies on a rise in the number of reported cases meant that the outbreak had been occurring for some months before it was detected.

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 16) a very few percent of outbreaks and a fraction of a percent of cases are caused by *L. monocytogenes*.

6.2.3 Case control studies

The case control studies (Table 17) 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 recent outbreak of non-invasive infection with *L. monocytogenes* in New Zealand (Sim *et al.*, in press) 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 in Table 17 (Schuchat *et al.*, 1992) identified soft cheese as the principal risk factor.

Table 14: 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 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 15: 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, 2000b

Table 16: 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

Table 17: Case control studies containing information on *L. monocytogenes* in ready-to-eat meats

Country	Risk/Protective factor	Odds ratio (CI)	Reference, year
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

*Includes cold meats, sandwiches, cheese and salads. 26 of 31 volunteering information bought "some" ready-to-eat meats from this source
CI = Confidence interval

6.2.4 Risk assessments

A number of risk assessments have now been published concerning *L. monocytogenes*. Farber *et al.* (1996) concentrated on two foods, pâté and semi-soft cheese and gave predicted human infection rates for these foods under a variety of scenarios in Canada.

More recently, two draft risk assessments for *L. monocytogenes* in ready-to-eat foods have been produced, one by the USFDA/FSIS (<http://vm.cfsan.fda.gov/~dms/lmrisk1.html>) in January 2001, and the other by FAO/WHO (http://www.who.int/fsf/mbriskassess/Scientific_documents/mra001.pdf). After the most recent round of revisions the FAO/WHO model has combined aspects of the FDA/FSIS one and almost merged the two. However, since the latest draft of the FAO/WHO assessment is not yet publicly available only the FDA/FSIS assessment will be discussed here.

It should be noted that this is very much a North American risk assessment and so used an exposure assessment which is particular to that part of the world (even though data from anywhere in 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 characterization 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 18. These risk rankings are quite consistent with results from case control studies.

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 and meat spread, and deli meats categories ranking in the top five causes of 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 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 18: Predicted relative risks of listeriosis based on median values for the North American population (1 represents the highest ranked risk and 20 the lowest)

Food Categories	Sub-Population		
	Intermediate Age	Elderly	Perinatal
	Relative Rank (1-20)		
SEAFOOD			
Smoked seafood	3	3	3
Raw seafood	14	14	14
Preserved fish	7	7	6
Cooked ready-to-eat crustaceans	6	5	5
FRUIT AND VEGETABLES			
Vegetables	17	17	17
Fruits	18	18	18
DAIRY PRODUCTS			
Soft mould ripened & blue vein cheese	9	9	9
Goat, sheep and feta cheese	16	16	16
Fresh soft cheese (e.g. queso fresco)	2	1	1
Heat-treated natural/process cheese	15	15	15
Aged cheese	19	19	19
Fluid milk, pasteurised	10	10	10
Fluid milk unpasteurised	11	11	11
Ice cream and frozen dairy products	20	20	20
Miscellaneous	12	13	13
MEATS			
Frankfurters*			
All frankfurters	8	8	7
Only reheated frankfurters	[15]	[15]	[15]
Only non-reheated frankfurters	[1]	[2]	[2]
Dry/semi dry fermented sausages	13	12	12
Deli meats	4	4	4
Pâté and meat spread	1	2	2
COMBINATION FOODS			
Deli salads	5	6	8

*Numbers in square brackets are for sub frankfurters either eaten raw or cooked.

Source: USDA/FSIS (<http://vm.cfsan.fda.gov/~dms/lmrisk1.html>)

6.3 Qualitative Estimate of Risk

The information summarised above leads to the conclusion that the transmission of *L. monocytogenes* by meat products is likely to contribute a significant proportion of invasive listeriosis cases in New Zealand. Evidence for this conclusion comes from:

- food surveys indicating a prevalence of contamination in ready-to-eat meat products similar to that found overseas;
- the high level of consumption of these foods in terms of numbers of servings and serving sizes;
- a report of a New Zealand outbreak of non-invasive infection by *L. monocytogenes* where ready-to-eat meats were identified as the vehicle; and,
- the identification of ready-to-eat meats as a transmission vehicle in episodes reported from the Investigation of Foodborne Illness Project (F12).

The limited prevalence data for New Zealand suggests that contamination occurs across all types of ready-to-eat meats. The risk assessment by the USDA/FSIS suggests that pâté and meat spreads, along with deli meats represent the highest relative risks in the ready-to-eat meat group, while fermented meat products have a lower relative risk.

6.4 Risk Categorisation

The rationale for categorisation of food/hazard combinations is presented in Appendix 1.

The invasive form of listeriosis causes a high (>5%) proportion of serious outcomes (hospitalisation, long term illness, and death). Although there are no data to identify the proportion of listeriosis transmitted by ready-to-eat meats compared to other food groups, the incidence will be in the lowest category because the overall incidence is below 1 per 100,000.

The non-invasive form of the disease is presumed to cause few serious outcomes, but data on incidence of this form are not available.

6.5 Summary

Food/hazard combination	Severity	Incidence	Trade importance	Other considerations
<i>L. monocytogenes</i> in processed ready-to-eat meats	1 (>5% serious outcomes)	4 (<1 per 100,000)	High (control essential)	Incidents attract adverse media attention

7 RISK MANAGEMENT INFORMATION

7.1 Relevant Food Controls

7.1.1 Status of Food Safety Programmes in the food industry sector

Risk Management Programmes (RMPs) are part of the emerging food assurance system in New Zealand. They form part of the Animal Products Act of 1999. These will eventually be integrated with the Food Safety Programmes (FSPs) and Product Safety Programmes (PSPs) required by the Food Act and Dairy Industry Act (<http://www.maf.govt.nz/Food/key-concepts.htm#risk>).

In New Zealand Hazard Analysis and Critical Control Point (HACCP) based food safety programmes are compulsory for all the NZ meat plants and seafood companies listed for exporting to the US. From 29 November 2000 all new entrants to the animal product industries governed by the Animal Products Act will have to have risk management plans in place based on HACCP principles. The local companies already in existence, but not exporting, will have two years to comply with this requirement.

The New Zealand Pork Industry Board is currently implementing 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. The plan will cover most of the ready-to-eat meat products currently on the market, apart from uncooked fermented comminuted meat products such as mettwurst and some salamis. A new module for the PQIP programme (PQIP07) to cover these products is in development.

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.

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 Ministry of Health (Ministry of Health, 1995a) but decisions on individual products are made by the manufacturer or retailer.

7.1.2 Legislative environment in New Zealand and overseas with respect to *Listeria monocytogenes*

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 bacteria is unlikely. This section collates information on the regulatory regimes in place in New Zealand and overseas.

7.1.2.1 New Zealand

Criteria regarding *L. monocytogenes* in foods are contained in the Microbiological Reference Criteria for Food (Ministry of Health, 1995b). 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

Foods exempt from these criteria include:

- 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.

7.1.2.2 Australia

The Food Standards Code contains a small number of categories that require zero tolerance. Such categories include meat paste and pâté, smoked fish products, marinated smoked mussels and cheeses with a moisture content equal to or greater than 40% and a pH ≥ 5.0 . These regulations apply only at the end of production or at the wholesale stage of distribution.

7.1.2.3 Joint Australia New Zealand Food Standards Code

The joint Australia New Zealand Food Standards Code microbiological standards for *Listeria* apply to foods sampled at any point during their stated shelf life. Zero tolerance (n=5, c=0, m=0) applies to;

- butter made from unpasteurised milk and/or unpasteurised milk products
- soft and semi-soft cheeses (moisture content >39%) with pH>5.0
- all raw milk cheeses (cheese made from milk not pasteurised or thermised)
- unpasteurised milk
- packaged cooked cured/salted meat
- packaged heat treated paste and packaged heat treated pâté
- cooked crustacea
- molluscs that have undergone processing other than depuration

For ready-to-eat finfish, other than fully retorted fish, the standard is n=5, c=1, m=0, M=100.

7.1.2.4 United States of America

The United States of America has a zero tolerance for *L. monocytogenes* in ready-to-eat foods.

7.1.2.5 Canada

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

Category 1: Foods causally linked to listeriosis. Includes soft cheese, liver pâté, coleslaw mix with a shelf-life >10 days, and jellied pork tongue.

Action level >0 cfu/50g. Immediate action-Class I recall to retail level.

Category 2: All other ready-to-eat foods supporting the growth of *L. monocytogenes* with a refrigerated shelf-life of >10 days.

Action level >0 cfu/25g. Immediate action-Class II recall to retail level.

Category 3: Ready-to-eat foods supporting the growth of *L. monocytogenes* with refrigerated shelf-life of <10 days and all other ready-to-eat foods not supporting growth.

Action level ≤100 cfu/g with adequate GMP. Immediate action-allow sale. Follow up action-follow up at plant level.

Action level ≤100 cfu/g with inadequate or no GMP. Immediate action-consider class II recall or stop sale. Follow up action-follow up at plant level.

Action level >100 cfu/g. Class II recall or stop sale. Follow up action-follow up at plant level.

Ready-to-eat foods not supporting growth of *L. monocytogenes* include the following:

- 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

7.1.2.6 England and Wales

For the ready-to-eat food categories listed by Gilbert *et al.* (2000), when sampled at the point of sale, there are three 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

7.1.2.7 Denmark

Danish criteria for *L. monocytogenes* in foods are rather more complicated (Nørrung *et al.*, 1999). They are intended to be applied at any point during the shelf life of the product.

The criteria are:

Group I. Foods heat-treated in the final package. n=5, c=0, m=0 (in 25g)

Group II. Heat treated foods that are handled after treatment. The product supports the growth of *L. monocytogenes* within the shelf life, and typically the shelf life is > 1 week. n=5, c=0, m=0 (in 25g)

Group III. Lightly preserved, not heat-treated, ready-to-eat products. The product supports the growth of *L. monocytogenes* within the shelf life, and typically the shelf life is > 3 weeks. n=5, c=0, m=0 (in 25g)

Group IV. Heat treated foods that are handled after treatment. These products are “stabilized” against the growth of *L. monocytogenes*, and foods with a shelf life of < 1 week are regarded as “stabilized”. n=5, c=1, m=10, M=100 (in 1g)

Group V. Lightly preserved not heat-treated products. These products are “stabilized” against the growth of *L. monocytogenes*, and foods with a shelf life of < 3 weeks are regarded as “stabilized”. n=5, c=2, m=10, M=100 (in 1 g).

7.1.2.8 European Union

The European Union has a zero tolerance for *L. monocytogenes* in soft cheese and pasteurised milk (absence in 25g) and for all other dairy products (absence in 1g).

7.1.3 Correlation between regulatory situation and incidence of listeriosis

A comparison between the regulatory status with respect to *L. monocytogenes* and the incidence of reported listeriosis in a variety of countries is given in Table 19.

Table 19: Correlation between standards/guidelines and incidence of listeriosis

Country	Regulatory policy	Incidence of listeriosis (cases per million population)	Year
New Zealand	Zero tolerance with exceptions	5	2000
Australia	Few foods with zero tolerance	1-11 (Western Australia)	1993-1998
ANZ Food Code	Few foods with zero tolerance, one with a tolerance	See above, code not fully implemented.	See above
Canada	Three categories, some tolerance of <100/g in some foods.	1-3	1990-1998
Denmark	Five categories, some tolerance of <100/g in some foods.	8	2000
England and Wales	Tolerates low levels in many RTE foods	2-5	1983-2000
EU	Dairy foods only.	Individual country data given above	
France	Some tolerance of <100 cfu/g at point of sale	4	1997
Germany	Some tolerance of <100 cfu/g at point of sale	2.5	Late 1990s
Italy	Essentially zero tolerance	3.5	1991-1992
Netherlands	Some tolerance of <100 cfu/g at point of sale	1.3-1.9	1996-1999
USA	Zero tolerance	6	1998

The data in Table 19 would suggest that the guidelines or standards applied by different countries have little effect on the incidence of disease. The main problem with this kind of analysis is that, while countries may set different standards, the crucial element is actually the degree of compliance with those standards. For example New Zealand has a zero tolerance for *L. monocytogenes* in cooked meat products, but there is every reason to believe from the information presented in this document that transgressions occur on a regular basis. There are no survey data to suggest that any country with zero tolerance has a ready-to-eat food supply that is free of *L. monocytogenes*.

7.2 Adverse Economic Effects from Infection with *Listeria monocytogenes*

The annual economic cost to New Zealand of cases of invasive listeriosis caused by foodborne transmission has been estimated as \$818,000, which represents 1.5% of the estimated total cost of foodborne infectious intestinal disease (Scott *et al.*, 2000). The number of cases and outcomes used for this estimate was based on an average of notification and hospitalisation data from 1991 to 1998 (Lake *et al.*, 2000). The estimated value includes

direct and indirect medical costs, the value of productive days lost, and the statistical value of mortality, but not the value of lost quality of life.

This estimate was based on several assumptions, the most important of which was that 90% of all cases of listeriosis were caused by foodborne transmission. This proportion was derived from proportions cited in the US. In that country, foodborne transmission of listeriosis has been estimated as 85-95% (Buzby *et al.*, 1996) and 99% (Mead *et al.*, 1999) of all cases.

This economic estimate covers all potential food vehicles. No data are available on the proportion of transmission due to ready-to-eat meats alone.

7.3 Risk Management Options

The main risk for foodborne transmission of listeriosis is from foods with high numbers of *L. monocytogenes*, and these are likely to be foods in which *L. monocytogenes* can grow, e.g. ready-to-eat meats with a long shelf life. 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 10^6 *L. monocytogenes* cells present from being eaten will result in the same reduction in risk as preventing the consumption of a million meals containing 10^0 *L. monocytogenes* cells).

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 that receive 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. Direct education campaigns by the Ministry of Health about the risk of listeriosis to pregnant women are already in place.

8 CONCLUSIONS

8.1 Description of Risks to New Zealand Consumers

8.1.1 Risks associated with red meat and meat products

The rate of reported invasive listeriosis in New Zealand is similar to that found in like countries (Table 13). As in other countries, most cases are sporadic, with outbreaks being rare. 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. *L. monocytogenes* in ready-to-eat meats has also been linked with several incidents of foodborne illness investigated under the F12 programme.

There have been only two other 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.

The number of invasive listeriosis cases reported every year is very small relative to other forms of foodborne disease. If rates were calculated specifically for “at risk” groups the numbers would be higher e.g. 12/100,000/year for the perinatal population in 2000. The importance of the disease derives from the high proportion of serious outcomes.

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.

L. monocytogenes has been detected in New Zealand ready-to-eat meats with a prevalence consistent with that observed overseas, although the New Zealand data are now almost 10 years old. No quantitative data are available for New Zealand, but it is likely that the level of contamination in these foods is also similar to that reported overseas as production and storage practices are not obviously different i.e. the majority of *L. monocytogenes* positive samples are contaminated at a very low level, with a very small percentage of samples containing more than 100 cells per gram.

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 fourth for relative risk of listeriosis in the USDA risk assessment. Pâté and meat spreads were ranked higher in this risk assessment, but these appear to be less frequently consumed in New Zealand. Salami represents only 5% of ready-to-eat meat servings, and is less likely to be a significant source of exposure as properly prepared fermented salami will not support the growth of *L. monocytogenes*.

It is likely therefore that ready-to-eat meats are a source of exposure to *L. monocytogenes* for the New Zealand population, but the degree of exposure cannot be estimated without

quantitative data on contamination levels. This conclusion is reinforced by the incidents and outbreak in which ready-to-eat meats have been identified as a vehicle for transmission.

8.1.2 Risks associated with other foods

Listeriosis is considered to be primarily a foodborne disease. Other potential vehicles of infection have been identified but most assessments assign the majority of cases to the foodborne route. It is likely that other ready-to-eat foods also contribute to foodborne listeriosis but foods on which it cannot grow, e.g. cheddar cheese, or which have a short shelf life, e.g. pre-prepared salads, are less likely to contribute to the disease burden significantly as the organism should not reach high numbers.

Aside from ready-to-eat meats, the USDA risk assessment also listed high (5 or above) relative risks of listeriosis for the following food groups (Table 18): fresh soft cheese, smoked seafood, cooked ready to eat crustaceans, deli salads. 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.

There are no data to estimate the relative risks of these other foods compared to ready-to-eat meats for New Zealand, although an outbreak of invasive listeriosis was attributed to smoked mussels.

8.1.3 Quantitative risk assessment

A quantitative risk assessment (QRA) for *L. monocytogenes* in ready-to-eat meats is an option that could be considered since there are now a number of others available that could be used to help build one (i.e. the resources required may not be too onerous). Published quantitative data for *L. monocytogenes* in New Zealand ready-to-eat meats are not available and this represents the most significant data gap, as it prevents use of the dose response relationship to produce a risk characterisation.

8.2 **Commentary on Risk Management Options**

The dose response and epidemiological data indicate that the consumption of ~100 cells/g in the serving sizes reported poses a very low risk of disease. Many overseas countries with tolerances of less than 100 cells/g for foods in which *L. monocytogenes* cannot grow do not have higher rates of disease. The zero tolerance for *L. monocytogenes* in New Zealand and Australia is currently being reviewed by a group led by FSANZ with respect to smoked fish, but no changes are planned for ready-to-eat meats.

As commented in Section 7.1.3, the regulations regarding tolerances for *L. monocytogenes* are probably less important than the degree of compliance.

Many manufacturers of ready-to-eat meats will already have in place HACCP based hazard management systems, and the implementation of the PQIP resource should expand the coverage. Universal adoption and successful implementation of these systems should result in a reduction in exposure to this pathogen. Estimates of the exposure of the population to *L. monocytogenes* in ready-to-eat meats are now old and could be revised to assess whether the

continued implementation of HACCP based systems is resulting in decreased exposure. A survey of ready-to-eat meats to determine quantitative levels of *L. monocytogenes* would provide both data for a risk assessment and some indication of the effectiveness of the HACCP based management systems.

Shelf lives given to ready-to-eat meats on which *L. monocytogenes* can grow could be investigated in the light of potential growth of *L. monocytogenes* on those foods. Long storage periods even at correct storage temperatures may allow *L. monocytogenes* to grow and reach high numbers. Reduction of shelf lives will have an economic affect on the manufacturers, but initially the basis on which shelf lives are allocated could be audited.

Education of pregnant women with regard to the dangers of listeriosis occurs currently and this should assist in minimising cases. Enhancing the scheme to inform other at risk groups could reduce exposure of the 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 disease, although serious, is quite rare in comparison to other foodborne diseases, and this is reflected in the low overall economic impact of the disease. Given the increased adoption of HACCP based plans, the rate of disease would be anticipated to decline if no additional actions are implemented. However, the low rate of the disease would make it difficult to observe changes. A more useful indicator would be the level of contamination in products at retail.

The current low incidence of invasive listeriosis, along with the small proportion of the overall economic cost of foodborne infectious intestinal disease attributable to listeriosis, may seem to mitigate against it being important in New Zealand. However, two factors make this disease important in terms of risk ranking. Firstly listeriosis affects very young children with long term morbidity and occasional deaths, and these outcomes have occurred in New Zealand.

The second point is that *L. monocytogenes* is still very important in terms of international trade. While ready-to-eat meat products are not exported at present, good control of *L. monocytogenes* contamination is likely to affect perceptions and markets for other ready-to-eat foods which are exported, such as some types of seafood. Continued implementation of HACCP based programmes, together with data that shows they are being effective, would be valuable.

As stated earlier, the incidence of the non-invasive form of listeriosis in New Zealand is unknown. A targeted surveillance project would provide some estimation of the incidence of this disease. One possible approach would be to add testing for *L. monocytogenes* on clinical samples submitted for testing by GPs to the range of standard pathogen tests, over a limited period.

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APPENDIX 1: CATEGORIES FOR RISK PROFILES

The assignment of a category for a food/hazard combination uses two criteria: incidence and severity.

1. Incidence

The incidence is an estimate of the proportion of the foodborne disease rate due to an individual hazard, that is transmitted by a single food or food group.

The overall rate of foodborne disease caused by individual hazards can be derived from information in the published estimate of foodborne disease (Lake *et al.*, 2000). This estimate has been updated to reflect more recent notifications rates for the 12 months to June 2001, but still using 1996 census figures (3,681,546 population). Rates include estimates for unreported cases who do not present to a GP.

Disease/organism	Food rate (/100,000 population) Calculated for 12 months to June 2001	Food rate (/100,000 population) Calculated for 12 months to December 1998
Campylobacteriosis	1320	2047
Listeriosis	0.4	0.4
VTEC/STEC	1.9	1.4
Salmonellosis	176	230
Yersiniosis	38	62
Shigellosis	7	7
NLV*	478	478
Toxins*	414	414
Typhoid*	0.3	0.3
Hepatitis A*	0.4	0.4

* not recalculated.

These are **total** foodborne rates, so it is probably safe to assume that in most cases the rates associated with a particular food are likely to be an order of magnitude lower. For instance, a category of “>1000” would only be assigned if it was decided that all campylobacteriosis was due to a single food/food type.

The following categories are proposed for the rates attributable to a single hazard/food (or food group) combination:

Category	Rate range	Comments/examples
1	>100	Significant contributor to foodborne campylobacteriosis Major contributor to foodborne NLV
2	10-100	Major contributor to foodborne salmonellosis Significant contributor to foodborne NLV
3	1-10	Major contributor to foodborne yersiniosis, shigellosis
4	<1	Major contributor to foodborne listeriosis

A further category, of “no evidence for foodborne disease in New Zealand” is desirable, but it was considered more appropriate to make this separate from the others. Also separate is

another category, of “no information to determine level of foodborne disease in New Zealand”.

The estimation of the proportion of the total foodborne disease rate contributed by a single food or food group will require information from a variety of sources including:

- exposure estimates
- results from epidemiological studies (case control risk factors)
- overseas estimates

For illnesses where the rate is <1 per 100,000 the ability to assign a proportion is unlikely to be sensible. For such illnesses it may be more useful to consider a Risk Profile across the range of all high risk foods, rather than individual foods or food groups.

2. Severity

Severity is related to the probability of severe outcomes from infection with the hazard.

The outcomes of infectious intestinal disease are defined in the estimate of the incidence (Lake *et al.*, 2000) as:

- death
- hospitalised and long term illness (GBS, reactive arthritis, HUS)
- hospitalised and recover
- visit a GP but not hospitalised
- do not visit a GP

The first three categories of cases were classed as severe outcomes. Some hospitalisations will result from dehydration etc. caused by gastrointestinal disease. However, for infections with *Listeria* and STEC hospitalisation will result from more severe illness, even if recovery is achieved.

The proportion of severe outcomes resulting from infection with the hazards can be estimated from the proportion of cases hospitalised and recover, hospitalised and long term illness, and deaths (Lake *et al.*, 2000).

Disease/organism	Percentage of outcomes involving death or long term illness from foodborne cases
Campylobacteriosis	0.3
Listeriosis	60.0
VTEC/STEC	10.4
Salmonellosis	1.0
Yersiniosis	0.4
Shigellosis	2.7
NLV	Assumed to be <0.5%
Hepatitis A	15.4
Typhoid	83.3
Toxins	Assumed to be <0.5%

Categories for the probability of severe outcomes are suggested as follows:

Severity Category	Percentage of cases that experience severe outcomes	Examples
1	>5%	listeriosis, STEC, hepatitis A, typhoid
2	0.5 – 5%	salmonellosis, shigellosis
3	<0.5%	campylobacteriosis, yersiniosis, NLV, toxins

There are a number of hazards for which the incidence of foodborne disease is uncertain. These have been assigned to the above severity categories as follows:

Severity category 1:

Bacteria

Clostridium botulinum

Protozoa

Toxoplasma

Severity category 3:

Bacteria

Aeromonas/Plesiomonas

Arcobacter

E. coli (pathogenic, other than STEC)

Pseudomonas

Streptococcus

Vibrio parahaemolyticus

Viruses

Others (e.g. rotavirus)

Protozoa

Giardia

Cryptosporidium

Cyclospora

Others (e.g. *Entamoeba*)

Proposed Category Matrix

Incidence	>100	10-100	1-10	<1
Severity 1				
Severity 2				
Severity 3				

Alternatives:

No evidence for foodborne disease in New Zealand

No information to determine level of foodborne disease in New Zealand