# RISK PROFILE:
# *LISTERIA MONOCYTOGENES*
# IN SOFT CHEESES

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by

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# RISK PROFILE:
# *LISTERIA MONOCYTOGENES*
# IN SOFT CHEESES

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**SUMMARY**

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

This Risk Profile concerns *Listeria monocytogenes* in soft cheese. Soft cheeses are defined as having a moisture content of >50%, and both domestically produced and imported cheeses are considered. All soft cheese manufactured in or imported into New Zealand will be made from pasteurised milk.

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 rate of reported invasive listeriosis in New Zealand is similar to that found in other developed countries at approximately 0.5 per 100,000 population. As in other countries, most cases are sporadic, with outbreaks being rare. There is currently no evidence to link cases of *L. monocytogenes* infection in New Zealand with soft cheese consumption.

Data on the prevalence of *L. monocytogenes* in domestically produced soft cheeses indicate that contamination rates are very low. The mandatory pasteurisation of milk for making soft cheeses in New Zealand means that contamination from the environment after this step is the most likely source of *L. monocytogenes* in this food. There are risk management measures in place under dairy industry Product Safety Programmes to detect and control environmental contamination during cheese manufacture. However, the ubiquitous nature of *L. monocytogenes* in the environment means that contamination of soft cheeses may occur at any point post-pasteurisation through to retail sale and domestic handling. Validation and verification of the pasteurisation process and avoidance of post-pasteurisation contamination are key areas to control.

Contamination of soft cheese may occur during handling in ripening rooms, wrapping and packaging stages or at retail/domestic cutting stages. Surface ripened cheeses are especially at risk because consequent rises in pH and other factors at the external crust may allow *L. monocytogenes* to grow, in contrast to the core of the same cheese which will remain at a low pH.

As an organism widespread in the environment, the general population and susceptible populations are likely to be frequently exposed to *Listeria* spp. The available dose-response data indicate that for the general population the probability of invasive disease following exposure to even moderate levels of cells is very low.

New Zealand effectively has a zero tolerance for *L. monocytogenes* in dairy products (i.e. absence is required in a 25g sample). Certain countries overseas advocate a tolerance level of 100cfu per gram at point of consumption. The draft Codex guidelines for control of *L. monocytogenes* (Codex, 2002, Section 5.2) state that although limits are a responsibility of

individual governments, a 99% reduction in the number of illnesses will be obtained by setting a Food Safety Objective at <100 *L. monocytogenes* per gram of food at point of consumption.

Consumption of soft cheese in New Zealand is modest, particularly when compared to European countries. When this is considered alongside the mandatory nature of pasteurisation, and data indicating a very low prevalence of contamination, the current risk from this food/hazard combination to the general New Zealand population must be considered low, although the risk to susceptible populations (with reduced immunity) will be greater.

The potential for growth of *L. monocytogenes* in soft cheeses depends on a number of factors. Intrinsic factors include the pH level, water activity and use of preservatives and starter cultures. Extrinsic factors include the time and temperature combinations in ripening/storage, humidity levels under which the cheese is ripened and whether pasteurised or raw milk was used. Because *L. monocytogenes* is a psychrotroph, refrigeration cannot be relied upon to inhibit growth.

It is not possible to make definite predictions about the behaviour of *L. monocytogenes* in the many types of soft cheese, and all soft cheese types must be regarded as potentially allowing growth if post-pasteurisation contamination occurs. Consequently each soft cheese manufacturer would need to assess their process on a case-by-case basis.

There is considerable discussion as to whether the production and importation of cheeses made from unpasteurised milk should be permitted in New Zealand. This Risk Profile does not address the potential risk of such types of cheese for New Zealand. Overseas information indicates that contamination of raw milk by *L. monocytogenes* does occur, and at a high prevalence in some countries. Any risk assessment of cheese production from raw milk for New Zealand would require additional data, in particular the prevalence and concentration of *L. monocytogenes* in raw milk here.

The data gaps identified in this Risk Profile are:

- Prevalence and quantitative data on *L. monocytogenes* in soft cheeses sold in New Zealand. The 2004 NZFSA/ESR soft and semi-soft cheese *L. monocytogenes* prevalence survey did not detect any contamination, and provided few opportunities for determination of quantitative data, other than to infer that a negative result corresponds to <0.04 cfu/g (absence in 25g) (Wilson, 2004),
- Prevalence and concentration of *L. monocytogenes* in raw milk in New Zealand. Any survey conducted to determine such data should be combined with testing for other human pathogens, and
- Information on environmental *L. monocytogenes* contamination in New Zealand cheese production sites and associated areas.

# 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**

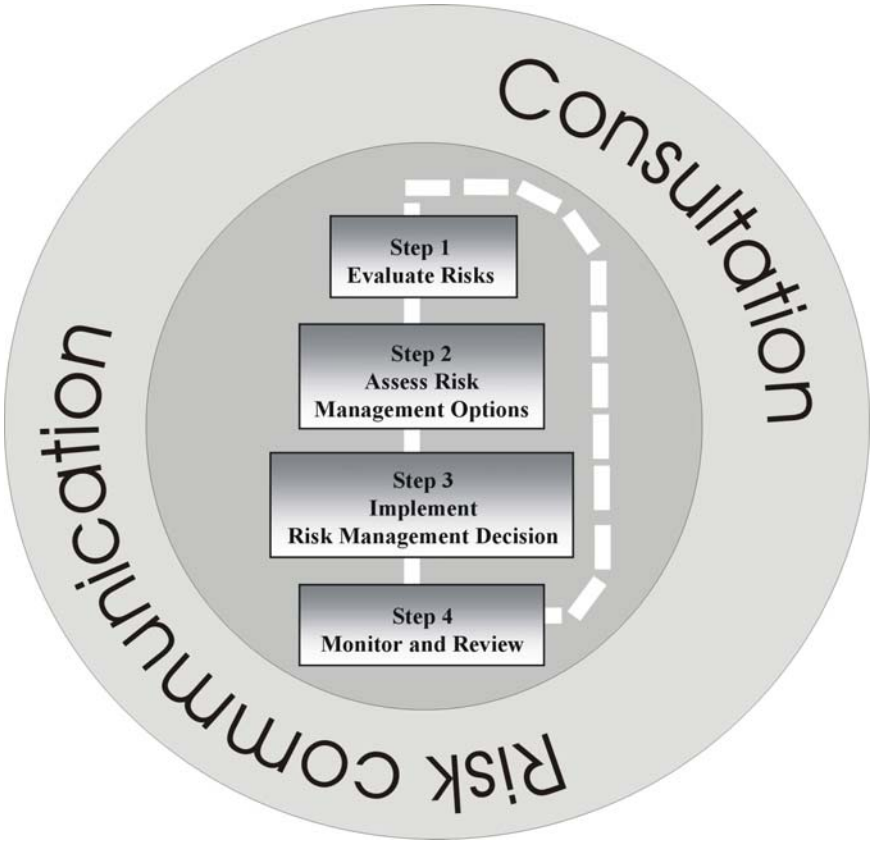



Figure reproduced from "Food Administration in New Zealand. A risk management framework for food safety" (Ministry of Health/Ministry of Agriculture and Forestry, 2000).

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, and
- 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 soft cheeses. This type of cheese is defined as containing >50% moisture, and includes Brie, Camembert, Feta, Ricotta, Gorgonzola, and the soft variety of Mozzarella.

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 (1999a).

*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:*

## 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

The bacterium is Gram-positive, non-sporulating and rod-shaped. Six species of *Listeria* bacteria have been recognised (ICMSF, 1996). Two are considered non-pathogenic; *L. innocua* and *L. murrayi*. (syn. *L. grayi*), while *L. seeligeri*, *L. ivanovii*, 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.

#### 2.1.2 Growth and survival

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

**Growth:**

Temperature: Optimum 37°C, range –1.5 to 45°C. Grows at refrigeration temperatures (4°C) (ICMSF, 1996).

pH: Optimum 7.0, range 4.4-9.4 (ICMSF, 1996).

Atmosphere: Grows optimally under microaerophilic conditions, but grows well both aerobically and anaerobically (anaerobic incubation has been shown to be more conducive to *Listeria* growth or survival than aerobic incubation). Can grow in food packaged under vacuum or nitrogen gas (AIFST, 2003). Growth of the organism was not retarded by a 5-10% $CO_2$ atmosphere and it can also grow in relatively high (e.g. 30%) $CO_2$, but growth is inhibited under 75% $CO_2$ (see survival below).

Water activity: The organism has a low $a_w$ limit for growth; 0.90 at 30°C in glycerol, 0.92 in sodium chloride (NaCl) and 0.92 in sucrose. The organism can grow in NaCl concentrations up to 10%, some laboratories report growth up to 12% NaCl (if pH is sufficiently high) (AIFST, 2003).

**Survival:**

Temperature: Survives freezing very well, but appears to depend on the isolate. In Feta cheese frozen at –38°C, survival of *L. monocytogenes* isolate Scott A was poor, while survival of *L. monocytogenes* isolate CA never dropped below 60% over 8 months storage. A similar pattern was observed at –18°C (Papageorgiou *et al*., 1997).

Atmosphere: The data suggest that modified atmospheres containing approximately 75% $CO_2$ and no oxygen will inhibit this organism (Hudson *et al*., 1993).

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

### 2.1.3 Inactivation (Critical Control Points 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) at a given pH. Inactivation proceeds faster at higher temperatures. The type of organic acid used can significantly affect results. In a study by Glass *et al.,* (1995), acetic acid reduced *L. monocytogenes* more effectively than malic or citric acids in a fresh soft cheese. Growth of *L. monocytogenes* has been found to occur at the surface of surface mould ripened soft cheese, due to increasing pH resulting from mould growth. Growth generally does not occur in the core of the cheese as pH values are inhibitory (see Section 3.2).

Water activity ($a_w$): Although growth does not occur at less than $a_w$ 0.90, the bacterium can survive for extended periods at lower $a_w$ values (AIFST, 2003).

Preservatives: Due to halotolerant nature of the organism, it is able to survive for long periods in salted foods (AIFST, 2003). It is 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). Nisin (a bacteriocin), has been shown to retard the growth of *L. monocytogenes* in Ricotta-style cheeses (Davies *et al*., 1997), but not in Camembert and with equivocal results in Feta where a nisin-producing lactic acid bacterium was added to the starter culture (Ramsaran *et al*., 1998). Cheeses produced with starter cultures would be unsuitable for nisin addition (due to its inhibitory action), although nisin-resistant starter cultures have potential for such applications (Davies *et al*., 1997).

The addition of enterococci to starter cultures has been shown to be effective in controlling *L. innocua* during the initial stages of the manufacture of Taleggio (a washed rind Italian soft cheese), due to the production of bacteriocins (Giraffa *et al*., 1994).

Radiation: D values depend on the food and temperature and range from 0.34 to 2 kGy. The use of X-rays to control *L. monocytogenes* in soft and red smear cheeses has been shown to produce off flavours at doses exceeding 1 kGy, and therefore will only remove low doses of the pathogen in these cheeses. However, in the case of surface ripened soft cheeses, where *L. monocytogenes* grows particularly near the surface (pH is raised due to surface mould growth), a specific irradiation of the rind after ripening with a low energy electron beam could be used to administer a higher dose (up to 3.0 kGy) and reduce numbers in more heavily contaminated samples without noticeable organoleptic deterioration (Ennahar *et al*., 1994).

In the soft whey cheese Anthotyros, the calculated D value was 1.38 kGy and doses up to 4

kGy did not adversely affect the quality of the product, (Tsiotsias *et al*., 2002). A D value of 1.4 kGy has been reported in Mozzarella cheese at –78°C (Hashisaka *et al*., 1989). However no organoleptic assessment was conducted.

*L. monocytogenes* is more sensitive than other Gram positive bacteria to UV radiation.

2.1.4 Sources

Human: *L. monocytogenes* is carried asymptomatically in the faeces of 2-6% of the population. Person-to-person spread (other than mother to foetus) is 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* can be present in the faeces of healthy animals. Although *L. monocytogenes* does not readily invade the udder, it can also be excreted in milk of healthy cows (Vizcaino and Garcia, 1975) and goats (Løken *et al*., 1982) as well as milk from mastitis-infected animals - the organism can cause listerial mastitis (Back *et al.,* 1993). The organism can also be found on raw chicken and other raw meats. Improperly made silage can be a source of domestic animal infection. Griffiths (1989) found that milk obtained from cows fed on silage during the winter months was often contaminated with *L. monocytogenes*, and that brie cheese bought in winter was contaminated but cheese made during the summer months was not.

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. The organism grows readily in milk but is effectively controlled by pasteurisation (See Section 3.1.1.2). 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. In quantitative studies of food products, low levels are typically detected (<100 cfu/g), although it has been detected at numbers far in excess of this (Farber and Peterkin, 1991).

Environment: Is widespread in the environment including soil, vegetation, water and sewage. Has been isolated from dairy environments (e.g. water used to wash cheese prior to ripening, cheese ripening rooms) and in domestic environments.

Transmission routes: An estimate of the proportion of listeriosis cases that are foodborne in New Zealand has been made at 90% (Lake *et al*., 2000). Alternative routes include infections acquired in hospital and occupational exposure (e.g. farmers).

The sources of *L. monocytogenes* demonstrate its ubiquitous nature in the environment, animals, and humans and its potential to contaminate all raw foods and ingredients.

# 3 HAZARD IDENTIFICATION: THE FOOD

## 3.1 Relevant Characteristics of the Food: Soft Cheeses

As discussed further in Section 3.1.2, soft cheese for this Risk Profile is defined as cheese containing >50% moisture. This Risk Profile does not cover soft cheeses produced from raw i.e. unpasteurised milk. In New Zealand it is a requirement that soft cheeses be produced from pasteurised milk. This Risk Profile also does not consider processed cheese (this type of cheese is considered in the "*L. monocytogenes* in low moisture cheese" Risk Profile) or cheese spreads (where cheese is only one ingredient).

Cheese manufactured from milk is essentially a preservation technique because the dehydration turns a highly perishable product into a less perishable one. Cheese is defined by Codex (1999b) as; "the ripened or unripened soft or semi-hard, hard or extra hard product, which may be coated, and in which the whey protein/casein ratio does not exceed that of milk, obtained by:

(a) coagulating wholly or partly the following raw materials: milk and/or products obtained from milk, through the action of rennet or other suitable coagulating agents, and by partially draining the whey resulting from such coagulation; and/or
(b) processing techniques involving coagulation of milk and/or products obtained from milk which give an end-product with similar physical, chemical and organoleptic characteristics as the product defined under (a)".

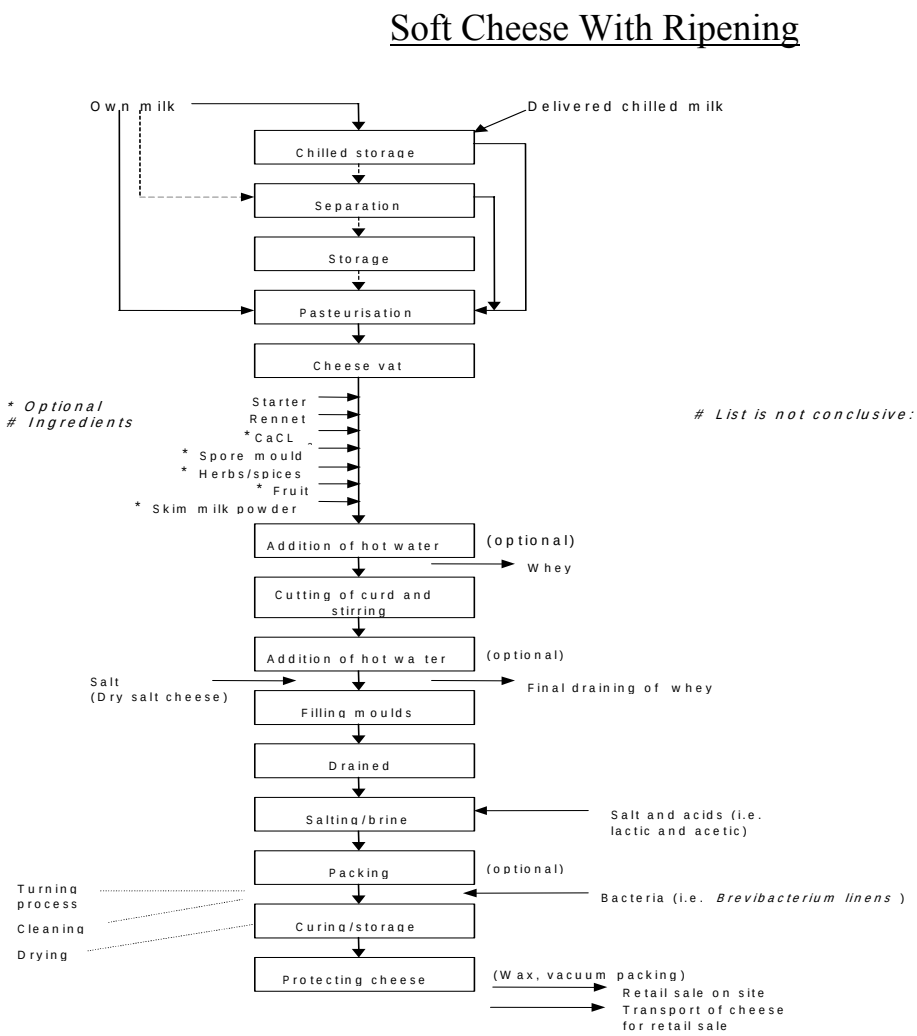
In simple terms, after milk treatment most cheese production takes the following steps; (ICMSF, 1998);

- Milk acidification,
- Coagulation (usually by the addition of rennet) to create curds,
- Dehydration through cutting of curds,
- Milling followed by salting (to stop starter culture activity),
- Pressing and shaping, and,
- Ripening.

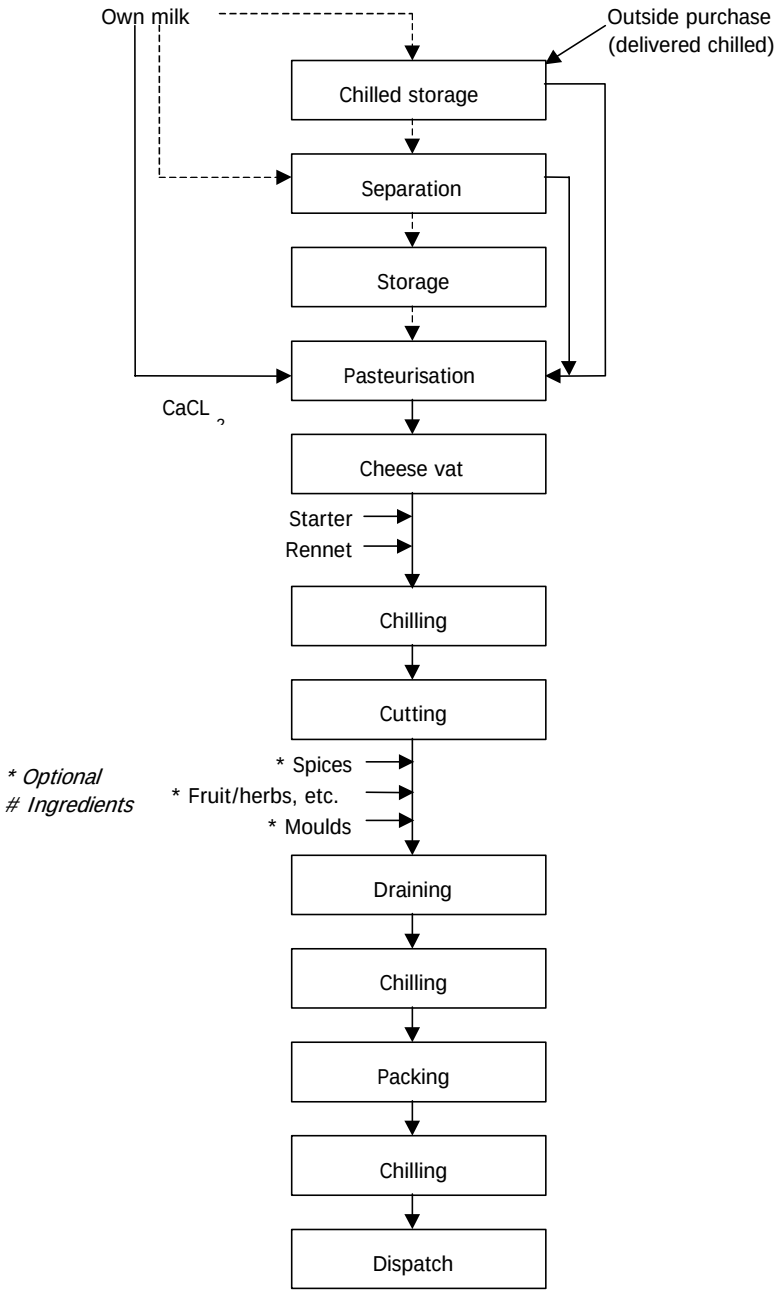
The two flowcharts in Figure 2 illustrate the production process of the two major types of soft cheese considered in this Risk Profile; with and without post-production ripening (also known as curing).

The flowcharts are reproduced with permission from the New Zealand Specialist Cheesemakers Association, Interim Code of Practice (NZSCA, 2002).

**Figure 2: The production of soft cheese with and without ripening.**



# Soft Cheese Without Ripening




** Optional*
*# Ingredients*

*# List is not conclusive*

The following sections provide an overview of the cheese making process in relation to *L. monocytogenes* prevalence and growth potential. Cheese production processes can vary considerably; only the main steps from farm to retail are covered here.

### 3.1.1 Ingredients and processing

The main ingredients of soft cheese are milk, starter culture, rennet and/or acid, and salt. Optional extras include herbs and spices, fresh or dried fruit, nuts and seeds, other derivatives of milk such as skimmed milk powder or cream, and preservatives such as sorbic acid.

#### *3.1.1.1 Milk*

Milk is the largest single ingredient in cheese making. Raw milk collection has the potential of delivering *Listeria monocytogenes* to the cheese making facility; through direct contamination from the lactating mammal (usually cow, sheep and goat) or environmental contamination from sources such as the milking environment and handler, equipment, water quality etc. In cows with mastitis, *L. monocytogenes* may be shed at 10,000-20,000 cells per ml of milk, with the appearance of the milk being normal and there being no inflammation of the affected quarter (Bunning *et al.* 1986).

Normal practice is to store raw milk at or below 7°C and use the milk within 36 hours of milking.

The psychrotrophic nature of *L. monocytogenes* means that high numbers could result when milk is stored for any significant length of time, even under refrigerated conditions. Growth of *L. monocytogenes* has been measured in whole milk at 4°C after a lag phase of approximately 48 hours, increasing approximately 1.5 $\log_{10}$ units in the following 48 hours (Donnelly and Briggs, 1986). At 10°C numbers increased approximately 6 $\log_{10}$ units in 48h. Growth in raw milk was slower than in pasteurised milk, probably due to the effects of competing bacteria (Northolt *et al*., 1988), but nevertheless occurred at both 4 and 7°C.

Enumeration of *L. monocytogenes* Type Scott A has been carried out where cows had been inoculated with the organism, including direct inoculation into the udder three weeks prior to the collection of milk (Doyle *et al.* 1987). Of twelve milk samples tested, four yielded *L. monocytogenes* on direct plating, with counts ranging from $3.0 \times 10^2$ /ml to $1.9 \times 10^4$ /ml. *L monocytogenes* was detected in two more samples following sonication. Sonicated samples yielded counts 2-5 times higher. In pooled milk from one cow which had been identified as shedding *L. monocytogenes*, counts in five replicate samples varied from $> 1.1 \times 10^3$ to $1.5 \times 10^4$ MPN/ml (Farber *et al.* 1988).

Few published data are available for raw milk in New Zealand. A study in 1987 (Stone, 1987) did not detect *L. monocytogenes* in 71 (50ml) raw milk samples, although 16 samples (23%) were *Listeria* positive. The species in positive samples were identified as *L. grayi* (10%), *L. innocua* (14%) and *L. welshimeri* (1.4%).

As summarised in Table 1, overseas studies have demonstrated the presence of *L. monocytogenes* and other *Listeria* spp. in milk. Most reports concern raw milk; there are few reports for pasteurised milk, although a large UK survey did not find any positive samples.

**Table 1: Prevalence of *L. monocytogenes* in raw milk samples overseas**

| Country/ region | Sample type | No. of samples tested | No.(%) positive for *L. monocytogenes* | Reference |
|---|---|---|---|---|
| **Asia** | | | | |
| Japan | Raw, farm bulk tank | 120 | 1 (0.8)* | Takai *et al.,* 1990 |
| **Europe** | | | | |
| France | Raw, bulk tanks | 1459 | (2.4) | Meyer-Broseta *et al.,* 2002 |
| Italy | Raw, goat | 60 | 0 (0) | Foschino *et al.,* 2002 |
| Netherlands | Raw | 137 | 6 (4.4) $<10^2$/ml | Beckers *et al.,* 1987 |
| Scotland | Raw, bulk tanks | 180<br>180<br>180 | 7 (3.8) summer<br>0 (0) autumn<br>2 (1.0) winter<br><1 cell/ml. | Fenlon and Wilson, 1989 |
| Switzerland | Raw | 340 | 2 (0.6) | Bachman and Spahr, 1994 |
| Turkey | Raw | 211 | 2 (0.9) | Uraz and Yücel, 1999 |
| UK | Raw<br>*Pasteurised* | 610<br>*1413* | 101 (16.5) <2 $\log_{10}$<br>*0* | Food Standards Agency, 2003 |
| **North America** | | | | |
| **Canada** | | | | |
| Ontario | Raw, from farm | 1720 | 47 (2.7) | Steele *et al.,* 1997 |
| | Raw, from bulk tanks | 455 | 6 (1.3) | Farber *et al.,* 1988a |
| Manitoba | Raw, farm<br>Raw, dairy | 192<br>64 | (1.0)<br>(3.1) | Davidson *et al.,* 1989 |
| **USA** | Raw<br>Raw<br>Raw | 124<br>121<br>650 | 15 (12.0)<br>15 (12.0)<br>27 (4.2) | Fleming *et al.,* 1985,<br>Hayes *et al.,* 1986<br>Lovett *et al.,* 1987 |
| Pacific Northwest | Raw | 474 | 23 (4.9) | Muraoka *et al.*, 2003 |
| Nebraska | Raw | 200 | 8 (4.0) | Liewen and Plautz, 1988 |
| South Dakota & Minnesota | Raw | 131 | 6 (4.6) | Jayarao and Henning, 2001 |
| Tennessee | Raw, bulk farm tanks | 292 | 12 (4.1) | Rohrbach *et al.,* 1992 |

**Listeria* spp. not *L. monocytogenes*

In the French study cited in Table 1 (Meyer-Broseta *et al.,* 2002) a seasonal pattern could be observed, with positive isolations tending to occur in the winter. Where enumeration was performed, eleven samples did not yield colonies when 2 ml of milk were enumerated. For the other three samples, counts of 210, 10 and 1 cfu/2 ml were recorded.

Before pasteurisation takes place, the milk may be pre-treated by (ICMSF, 1998);

- Filtration,
- Separation and clarification, and
- Bactofugation (a specialised clarification process).

Filtration (straining raw milk through filter cloths) is primarily aimed at removing physical contaminants, such as visible faecal matter and hair. It does not address the microbial loading of the raw milk and must be closely monitored to ensure cross contamination does not occur from a soiled filter. Microfiltration is a developmental process that promises raw, pathogen free milk (ICMSF, 1998) however it currently does not provide a safety equivalent process to pasteurisation.

Separation produces three fractions; skim, cream and sediment or 'slime'. Many micro-organisms including *Listeria* spp. can be physically removed in this manner although it is important that the sediment is isolated and removed hygienically.

Clarification removes suspended particulates and any adhering microorganisms by a centrifugal filtering process. Bactofugation, a specialised clarifier, reduces bacterial populations particularly spore forming bacteria, such as the *Clostridia* spp. This is more commonly used for low moisture cheese production, where certain *Clostridia* may produce an undesirable late blowing or 'gassiness' in the cheese texture.

*3.1.1.2 Pasteurisation*

Pasteurisation is a major critical control point and a legal requirement in New Zealand (see section 7.2), for controlling microbial contamination. Pasteurisation conditions are defined in the relevant New Zealand Standard as;

- Holding method (63-66ºC for not less than 30 minutes),
- High-temperature short-time method (>72ºC for not less than 15 seconds), and
- Any other heat treatment method that is as effective in terms of bacterial reduction as the methods above.

The high temperature short time method is the most commonly used in New Zealand.

Where the pasteurisation process is faulty through equipment defects or incorrect operation, *Listeria monocytogenes* and other pathogens may survive. It is therefore paramount that this critical control point is closely monitored for compliance.

It is generally considered that pasteurisation (high temperature, short time conditions) is effective in destroying *L. monocytogenes* in milk. The efficacy of HTST processing was demonstrated in four different experiments using up to $10^5$ *L. monocytogenes*/ml (Lovett *et al.,* 1990). Should any cells survive pasteurisation they will most likely be heat injured. It

has been shown that cells injured by pasteurisation cannot compete with surviving thermoduric organisms and do not grow in milk held under refrigerated storage (Crawford *et al.,* 1989).

However, this assurance has been the subject of considerable scientific debate (summarised in Hudson *et al.*, 2004). A high prevalence in pasteurised milk has been reported in Spain (3.2% fat milk treated at 78°C for 15 seconds), where 21.4% of samples from a single processing plant were positive for *L. monocytogenes* (Fernandez-Garayzabal *et al.,* 1986).

Following a serious outbreak of listeriosis associated with pasteurised milk in Massachusetts in 1983 (49 cases, 14 deaths) (Fleming *et al.*, 1985), investigations found no problems or contamination in the dairy plant. It was suggested that *L. monocytogenes* cells could survive pasteurisation if they were internalised by phagocytes in raw milk (Doyle *et al.*, 1987). However, no increased heat resistance for intracellular bacterial cells has been reported in other studies (Bunning *et al.*, 1988; Farber *et al.*, 1992).

It was suggested that the problem in Massachusetts occurred because whole milk was passed through a filter, rather than a centrifugal filtering process (clarification) (Fleming *et al.*, 1985). Clarifying the milk also removes leukocytes and is routine in major cheese manufacturers in New Zealand, but not for small producers. However, the pasteurisation conditions stipulated in Dairy Standard D121.1 are considered sufficient to control intracellular *Listeria*, as they take account of particle sizes in the milk.

Other than the "pasta filata" cheese process (section 3.2.2), there are no further heat treatments in the soft cheese making process that will inactivate *L. monocytogenes*. Because pasteurisation of soft cheese is a legal requirement in New Zealand, the two possible routes for contamination are ingredients added after pasteurisation, or (more likely) environmental contamination e.g. equipment, food handlers, pests etc. Post-pasteurisation hygiene is therefore a key area for cheesemakers to control and is discussed in more detail in Section 7.5.

At this point the hygroscopic salt, calcium chloride, may be added to assist in curd development.

#### *3.1.1.3 Acidification and starter cultures*

Acidification (normally lactic acid produced by 'starter' cultures of *Lactobacillus* or *Lactococcus* bacteria) is a key part of the early stages in cheesemaking. Pasteurisation kills most of the naturally present milk souring organisms; these are re-introduced into pasteurised milk by selected cultures, which 'ripen' the milk i.e. transform milk into cheese curd. *Listeria monocytogenes* is capable of growth if it is present in the starter culture, whether the starter is pH-controlled or not.

Specific starter cultures are used to give acidity, flavour and aroma to the cheese. Cultures can be in liquid, frozen and dried forms and are generally added to milk at the rate of 1 to 1.5%.

An alternative (and sometimes in addition to) the addition of starter culture is the direct addition of food grade acids, as in some cheeses such as Mozzarella and some cottage cheeses.

The milk must be at the correct temperature for the specific starter culture added, too high and the culture will be inactivated, too low and the culture will take too long, allowing other bacteria present to grow. Similarly the correct ratio of starter culture to milk is required; too much and a hard dry acidic cheese is the result. Too little and the acidity of the cheese will not develop quickly enough, allowing growth of potentially harmful bacteria.

After the initial milk-ripening period (typically 45 minutes), the acidity of the milk has risen due to the lactic acid production. It is at this stage that rennet is added.

*3.1.1.4 Rennet*

Calf rennet contains milk-clotting enzymes, the most important of which is chymosin. Vegetarian rennet can also be used which is extracted from fungal fermentations. Genetically modified microorganisms (with DNA coding for calf rennet) have also been used to produce pure chymosin. The rennet causes casein (milk protein) to coagulate into curd. Curd formation can take anyway between 5 minutes (for Swiss cheese) to 16 hours (for a long set cottage cheese) (ICMSF, 1998).

*Listeria monocytogenes* is inactivated at varying rates in pepsin-rennet of microbial and animal origin, but may survive long enough to be present in microbial rennet to contaminate batches of cheese (Ryser, 1999).

*3.1.1.5 Curd processing*

Once the cheese has coagulated to the desired consistency, the curd is cut into cubes. The size of these determines expulsion of whey, with larger cubes producing higher moisture cheese. The curd is then 'cooked' or 'scalded' (<40°C up to 54°C dependent on type of cheese being produced) to arrest the starter culture, further remove whey and alter the texture of the curd particles, (the curd cooking stage does not reach temperature inactivation levels for *L. monocytogenes*).

In several studies, higher concentrations of *L. monocytogenes* have been observed in curd than in whey. *Listeria* appears to be trapped within the casein of the milk and concentrated into the curd (Dominguez, 1987).

Various additional ingredients (e.g. fruit, vegetables, moulds, herbs, nuts and seeds) may be added at the renneting or curd cutting stage. Stabilisers and/or skimmed milk powder may also be added. Doyle *et al.*, (1985) cited in ICMSF (1998) observed a reduction of *L. monocytogenes* during the drying and ambient storage of milk powder but some samples remained positive for up to 12 weeks. The ICMSF (1998) goes on to state that proper pasteurisation and prevention of recontamination should preclude *Listeria* spp. in dry dairy products. There have been no reported incidents of *Listeria* spp. contamination in cheese due to skimmed milk powder as an ingredient.

*3.1.1.6 Salt*

Salt has a number of roles in cheese besides adding a salty flavor. It preserves cheese from spoiling, draws moisture from the curd, and firms up a cheese's texture in an interaction with

its proteins. Some cheeses are salted from the outside with dry salt or brine washes. Most cheeses have the salt mixed directly into the curds.

Although salt has a general prohibitive effect on bacteria, it does not have a strong bacteriocidal effect on *L. monocytogenes* until relatively high concentrations are reached. *L. monocytogenes* can grow in salt concentrations up to 10%. Research carried out by Hudson, (1992) on the effect of various salt concentrations (up to 26% (saturation point)) and temperatures on the organism found all the salt concentrations tested were ineffective in reducing numbers over six hours incubation at low temperatures. However, taken in conjunction with low pH and other synergistic factors, the addition of salt in cheese does have several important functions. These include control of microbial growth and metabolism, control of enzymatic activity and texture differences (ICMSF, 1998). Where cheese is brined, the solution of the brine should be at least 50% of saturation point and changed or heat-treated frequently. A saturated brine solution contains 26.4% salt by weight at 15.5°C, saturation point can vary slightly according to temperature. A 50% saturated solution is therefore at least 13% salt by weight.

*3.1.1.7 Storage*

A newly created cheese is usually salty yet bland in flavor and, for harder varieties, rubbery in texture. These qualities may be desirable, but usually cheeses are left to rest under carefully controlled conditions. This aging period (also called ripening) can last from a few days to several years. As a cheese ages, microbes and enzymes transform its texture and intensify its flavor. This transformation is largely a result of the breakdown of casein proteins and milkfat into a complex mix of amino acids, amines, and fatty acids.

The pH changes that take place during production and ripening (and consequent effect on *L. monocytogenes*) are discussed in more detail in Sections 3.1.5 and 3.2.

Cheeses are normally kept in the chill chain from production to retail.

*3.1.1.8 Summary of controls in cheese making*

Key process controls in cheesemaking are (Food Standards Agency, 2001);

- Good animal health and veterinary care: to minimise *Listeria* contamination in the raw milk,
- Clean milking, handling and cooling: to avoid contamination and restrict bacterial growth,
- Avoid inhibitory substances in raw milk (e.g. antibiotics) to ensure correct acid development,
- Milk pasteurisation (if applied): to destroy pathogens,
- Correct acidification: to inhibit pathogens, and
- Correct salt addition: to inhibit pathogens.

In addition, the temperature and humidity conditions specific for the cheese need to be correct for ripening.

### 3.1.2 Definition of soft cheeses

There are about 2000 cheese varieties worldwide and they can be classified from a number of viewpoints (Belitz *et al.*, 2004):

- Milk used (cow, goat, or sheep),
- Curd formation (using acids, rennet extract or a combination of both),
- Texture or consistency or water content,
- Fat content or percentage dry matter.

In this report soft cheeses are defined as a group based on the moisture content, while further categorisation is done on the basis of post-production ripening (also known as curing).

There are many definitions of cheese types based on moisture content and none seem to be universally accepted.

For this Risk Profile, it was agreed with the NZFSA that the Food Standards Australia New Zealand (FSANZ) definition of soft cheese would be used: i.e. "soft" cheese contains ≥50% moisture. Attachment 2 of the User Guide to Standard 1.6.1 Microbiological Limits for Food published in July 2001 by FSANZ gives the classification scheme for cheeses and is presented in Table 2. This soft cheese definition also corresponds with the FDA/FSIS (2003) risk assessment work carried out in the USA (http://www.cfsan.fda.gov/~dms/lmr2-5.html), thus enabling direct comparison with that assessment.

**Table 2: FSANZ cheese classification, according to moisture content & ripening methods**

| Moisture content (%) | Cheese Type |
|---|---|
| 50-85% | *Soft Cheeses*<br>• Unripened e.g. Cottage, Quark, Cream, Mozzarella (soft variety)<br>• Ripened e.g. Camembert, Brie, Neufchatel, Caciotta<br>• Salt-cured or pickled e.g. Feta, Domiata |
| 39-50% | *Semi soft*<br>• Ripened principally by internal mould growth e.g. Stilton, Roquefort, Gorgonzola, Danish Blue<br>• Ripened by bacteria and surface micro-organisms, e.g. Limburger, Brick, Trappist, Port Salut<br>• Ripened primarily by bacteria e.g. Bel Paesa, Pasta Filata, Provolone, Brick, Gouda, Edam |
| <39% | *Hard*<br>• Without eyes, ripened by bacteria e.g. Cheddar, Caciocavallo<br>• With eyes, ripened by bacteria e.g. Emmental, Gruyere |
| <34% | *Very hard*<br>e.g. Asiago old, Parmesan, Romano, Grana. |

The Codex General Standard for Cheese (Codex 1999b – see website: ftp://ftp.fao.org/codex/standard/en/CXS_A06_2003e.pdf), classifies cheese on the basis of moisture content and ripening method. Moisture is represented on a percentage Moisture Fat Free Basis (%MFFB = weight of moisture of cheese x 100/ (total weight of cheese – weight of fat in cheese)). The classifications are as follows;

| **MFFB%** | **Designation** |
|---|---|
| > 67 | Soft |
| 54-69 | Firm/semi-hard |
| 49-56 | Hard |
| < 51 | Extra hard |

The NZFSA separates cheese into two types; "soft and semi-soft" and "firm and hard cheese", primarily for export categorisation. Soft and semi-soft cheeses are defined as having >60% MFFB. Soft cheese is further classified as having >67% moisture which is the same as the international Codex definition. Firm and hard cheese is defined as ≤ 60% MFFB. The following website contains the definitions on pages 19 and 20 of the register; http://www.nzfsa.govt.nz/dairy/registers-lists/prod-descr-20040914.xls.

A moisture content of 50% in a cheese with a typical fat content of 40%, would represent an 83% MFFB. Thus the definition of soft cheeses for this Risk Profile (>50% moisture) is well within the range specified by Codex and NZFSA for soft cheese.

Specific definitions of individual cheeses can be found in Codex Report of the 6th session on Milk and Milk Products; http://www.codexalimentarius.net/reports.asp.

Sections 3.1.3 to 3.1.7 provide an overview of the wide variety of types of soft cheeses, with some detail on their manufacture. Section 3.2 then provides data on the behaviour of *L. monocytogenes* in these types of soft cheese, drawn from the scientific literature.

### 3.1.3 Types of soft cheeses

Soft cheeses have high moisture content (although the exact figures vary between information sources) and so the range of water activity will permit microbial growth.

Soft cheeses can be broadly sub-divided into the following groups;

- unripened, (includes fresh),
- ripened,
- salt cured or pickled, and
- whey cheese.

In the US "fresh cheese" may describe two types of cheese. The first is cheese produced by acid coagulation (e.g. by addition of lactic acid in the form of glucono-delta-lactone) with little or no added rennet. These cheese types (cottage, quark, and cream cheese) are included in this report as part of the soft cheese unripened group.

The second type of "fresh cheese" is rennet coagulated and produced with little or no culture. Without acid production, pH remains high. This type of fresh cheese includes the "queso" and Mexican style soft cheese varieties (see

http://www.foodsci.uoguelph.ca/cheese/sectiona.htm). This type of cheese is of particular importance in the USA due to a large outbreak of listeriosis associated with its consumption (Bolton and Frank, 1999), but is less likely to be consumed in New Zealand. In this Risk Profile the term "fresh cheese" is reserved for this second category that is specifically considered in the FDA/FSIS *L. monocytogenes* risk assessment.

### 3.1.4 Unripened soft cheeses (e.g. Cottage, Quark, Cream, Mozzarella)

Characterised by a slightly acidic, mild flavour and spreadable texture, unripened cheeses are ready to consume shortly after manufacture. They are sometimes referred to as fresh, lactic or curd cheeses. There are a variety of ways in which unripened soft cheeses can be made, although a general characteristic is that the curds are not pressed. Direct addition of food grade acids (i.e. citric, acetic, malic) can be used to produce the curds.

Acidification to make cottage cheese can also be achieved by the addition of starter cultures but a distinguishing characteristic of this type of cheese is that coagulation involves little or no coagulating enzyme. Once the correct texture has been obtained, the curd is cut at around pH4.6 – 4.8, and then cooked or "scalded" to 52°C, inactivating the starter culture which prevents further acid development. After the scalding process, up to 50% of the whey is drained off and the curd washed up to 3 times in chilled water, further reducing acidity, removing whey and lactose. The dry curd is then blended with a salted cream dressing. Stabilisers may be added at this stage.

The fat content of fresh cheese can vary considerably. Cottage cheese is made from skimmed milk whereas cream cheeses are prepared from cream.

There are two varieties of mozzarella. The soft variety (traditionally made from water buffalo milk), can be sold vacuum packed dry or in a salted/unsalted liquid known as "latte". The other variety comes under the low moisture category (less than 50% moisture, therefore will not be covered by this Risk Profile). Both varieties are unripened cheeses made by the "pasta filata" process which involves heating curd of a suitable pH in a water bath, where the curd is kneaded and stretched, ensuring the curd is smooth and free from lumps.

The shelf life of unripened soft cheeses may be lengthened by the addition of the preservative sorbic acid (soluble form, potassium sorbate). The high moisture content can permit undesirable surface yeast and mould growth. Bacteriological spoilage can occur mainly due to psychrotrophs such as *Pseudonomas.* The main change during storage of unripened soft cheeses is the conversion of lactose (in the whey) to lactic acid (National Dairy Council, 1996).

### 3.1.5 Ripened cheeses

This type of cheese is not ready for consumption shortly after manufacture but must be held under specific time, temperature, and other conditions as necessary for biochemical and physical changes characteristic of that cheese to take place (Codex, 1999b). As the cheese matures, proteolysis takes place, water is lost and a rind can develop.

Mould ripening of cheese is accomplished primarily by the development of characteristic mould growth throughout the interior (blue veined cheese) and/or on the surface of the cheese (both mould ripening processes are used in Blue Brie). At the end of the ripening period the

dominant organisms on the surface are moulds such as *Penicillium camemberti*. The surface pH of surface ripened cheeses is related to the quantity of ammonia produced by filamentous fungi, as well as the consumption of lactic acid. In an experimental surface ripened soft cheese (50.3% moisture) the free fatty acid content of the exterior of the cheese was much higher than in the centre, reflecting lipolytic activity (Furtado and Chandan, 1985). Two examples of surface mould ripened cheeses are Camembert and Brie.

An alternative to mould surface ripening is to use a bacterial/yeast mixture that produces the "smear" cheeses. In these cheeses, yeasts and moulds dominate the early microflora since they are acid and salt-tolerant. Toward the end of the ripening period, the bacteria become the dominant organisms. These include the characteristic orange pigmented bacterium *Brevibacterium linens* giving the cheese surface a distinctive red colour (Corsetti *et al*., 2001). Smear organisms produce proteolytic and lipolytic enzymes, and produce alkaline compounds such as ammonia that penetrate the cheese. In some cases the microbial flora present on smear cheeses can inhibit the growth of *L. monocytogenes* (Eppert *et al*., 1997) although the basis to this effect is unknown.

The result of the microbial activity at the cheese surface is to raise the pH. This surface effect on pH is found in a number of other surface ripened cheeses (e.g. Back *et al*., 1993).

Surface ripening of cheese is performed at high humidity for some weeks to encourage the growth of surface micro-organisms. This ripening process takes place from the surface to the centre (as opposed to low humidity ripening for most hard cheeses). Most of the ripened soft cheeses discussed in this Risk Profile belong to the surface mould ripened type or microflora surface red smear type.

### 3.1.6 Salt cured or pickled

This type of soft cheese is also known as white brined cheese. These cheeses have salt added to the milk or curd, or the cheese can be stored in a brine solution. One example of the latter is feta production (Papageorgiou and Marth, 1989). Pickled cheese manufactured from sheep milk has been shown to contain between 2.3 and 5.3% salt. Domiati cheese contains 4.5% increasing to 4.9% with storage.

### 3.1.7 Whey cheese

An alternative to producing cheese from curd is to use the by-product whey. There are two types of whey;

- Acid whey: produced from acid coagulation of fresh cheeses, and
- Sweet whey: produced from rennet type enzyme coagulations.

Whey cheeses were traditionally produced in Greece and Norway and are prepared by one of two methods;

- Concentration of (acid or sweet) whey; by heat evaporation. This heating process, to temperatures 70$^{o}$C and above, evaporates the water. The high lactose content gives the cheese a yellowish/brown colour and a sweet, caramelized flavour. In Norway, this basic whey cheese is known as Mysost. Added milk/cream to the mix and/or

leaving more water content produces different brown cheeses such as Gjetost, which has a distinct fudge-like texture and caramel flavour,

- Coagulation of sweet whey, produced by heat precipitation of whey with or without the addition of acid. The low lactose gives a white to yellow colour. The cheese may be either ripened or unripened. Ricotta, (Italian for "recooked") is the best known whey cheese of this type. Inoculated bacteria in the whey ferments the remaining sugars producing lactic acid and lowering the pH (or the whey can be directly acidified). The heat then denatures the protein, which precipitates out.

## 3.2 Survival and Growth of *L. monocytogenes* in or on Soft Cheeses

### 3.2.1 Summary of FDA/FSIS information

Information on the behaviour of *Listeria monocytogenes* in "fresh soft", "soft unripened", and "soft ripened" cheese has been collated by the FDA/FSIS (2003) and summarised in Appendix 8 of their document for the purposes of a Quantitative Risk Assessment (see section 6.2.4). A summary of this information, adapted from the FDA/FSIS document, is presented below;

- For "fresh soft" cheese, 3 studies provided 10 data sets of growth data. Eight of the data sets show levels increasing; the other two demonstrated a decline (-2.0 logs in 30 days in Queso fresco and –0.8 logs in 10 days in Queso Ranchero). Exponential growth rates modelled at 5°C ranged from –0.080 to 0.285 $\log_{10}$ cfu/day. The average growth rate modelled at 5°C was 0.08 $\log_{10}$ cfu/day.

The individual studies are listed below.

| Type | Temp. (°C) | Growth rate | Reference |
|---|---|---|---|
| **Fresh soft cheese** | | | |
| Queso blanco | 4 | 1.4 log in 14 days | Glass *et al.*, 1995 |
| Queso fresco | 3 | 0.13 log in 1 day | Mendoza-Yepes *et al.*, 1999 |
| | 7 | 0.5 log in 1 day | |
| Queso fresco | 4 | 2.0 log **decr**. in 30 days | Genigeorgis *et al.*, 1991 |
| Queso Ranchero | 4 | 0.3 log **decr**. in 18 days | |
| Queso Panella | 4 | 2.13 log in 10 days | |
| | 4 | 0.21 log in 30 days | |
| | 4 | 0.44 log in 36 days | |

- In soft unripened cheese, six studies provided 29 data points. Growth or decline appears largely dependent on pH. Nine data sets indicated a decline while the other 20 data sets saw an increase. Exponential growth rates modelled at 5°C ranged from –0.333 to 1.423 $\log_{10}$ cfu/day. The average growth rate modelled at 5°C was 0.09 $\log_{10}$ cfu/day.

| Type | Temp. (°C) | Growth rate | Reference |
|---|---|---|---|
| **Soft unripened cheese** | | | |
| Cottage (multiple brands) | 8 | 0.59 log in 18 days | Genigeorgis *et al.*, 1991 |
| | | 1.87 log **decr**. in 36 days | |
| | | 0.42 log in 24 days | |
| | | 1.13 log in 8 days | |
| | | 1.87 log **decr.** in 8 days | |
| | 4 | 0.39 log in 24 days | |
| | | 0.34 log in 24 days | |
| | | 0.41 log in 16 days | |
| | | 0.94 log in 36 days | |
| | | 1.87 log **decr**. in 8 days | |
| Teleme | 8 | 2.2 log in 36 days | |
| | 4 | 0.42 log **decr**. in 36 days | |
| Ricotta (3 brands) | 8 | 2.11 log in 8 days | |
| | | 1.75 log in 6 days | |
| | | 1.88 log in 8 days | |
| | 4 | 1.53 log in 30 days | |
| | | 3.58 log in 36 days | |
| | | 1.97 log in 22 days | |
| Cream | 8 | 2.0 log **decr**. in 30 days | |
| | 4 | 2.0 log **decr**. in 36 days | |
| | | >2.0 log **decr**. in 36 days | |
| Cream | 4 | 2 log in 2 days | Cottin *et al*., 1990 |
| Ricotta (whey) | 5 | 16.2 – 20.2 hours GT | Papageorgiou *et al*., 1996 |
| | 12 | 5.1 – 5.8 hours GT (generation time) | |
| Cottage | 4 | 2.0 log in 40 days | Chen and Hotchkiss, 1993 |
| | 7 | 2.4 log in 10 days | |
| Cottage | 5 | 2 log in 22 days | Fedio *et al*., 1994 |
| Cottage | 'refrigerated' | 0.5 – 1.5 log **decr**. in 1 to 5 weeks | El-Shenawy and Marth, 1990 |
| | 6 | 1 log **decr**. in 21 days | |

- In soft ripened cheese, eight studies provided 17 data points. Seven data sets showed a decline, one survival only and 9 indicated growth. Exponential growth rates modelled at 5°C ranged from –0.250 to 0.197 $\log_{10}$ cfu/day. The average growth rate modelled at 5°C was a slow decline rate at –0.013 $\log_{10}$ cfu/day.

| Type | Temp. (°C) | Growth rate | Reference |
|---|---|---|---|
| **Soft ripened cheese** | | | |
| Feta | 4 | Survival > 90 days<br>Scott A 1.28 log **decr**.<br>3.07 log in 90 days | Papageorgiou and Marth, (1989) |
| mozzarella | 5 | 4 log in 21 days | Stecchini *et al*., 1995 |
| Brie | 4 | 0.6 log in 30 days<br>0.6 log in 14 days | Genigeorgis *et al*., 1991 |
| Feta | 4 | >2.0 log **decr**. in 8 days<br>>2.0 log **decr**. in 8 days<br>>2.0 log **decr**. in 8 days | |
| Camembert | 6 ripening | 4 log in 45 days | Ryser and Marth, 1987 |
| Camembert | 4 | 2 to 3 log **decr.** in 365 days | Farber *et al*., 1987 |
| Camembert | 3<br>6<br>10 | 0.9 log in 10 days<br>1.5 log in 15 days<br>2.4 log in 15 days | Back *et al*., 1993 |
| Blue cheese | 5 | Decr. during storage<br>3 log in 56 days | Papageorgiou and Marth, 1989 |
| Camembert | 14<br>7 | 4.5 log in 34 days<br>- | Sulzer and Busse, 1993 |
| surface growth | 4 | - | |
| Blue cheese | 4 | >2.0 log **decr.** in 36 days | Genigeorgis *et al*., 1991 |
| Camembert | 4 | 0.64 log in 36 days | |

The above summary of the studies carried out indicates that it is difficult to be definitive about the growth potential of *L. monocytogenes* in soft cheeses. For a single cheese type, studies collated by the FDA/FSIS may report either a decline or growth of the bacterium.

Further information on factors affecting survival and growth is presented in the sections below.

3.2.2 Fresh and soft unripened cheeses

Growth of *L. monocytogenes* occurs in Spanish unripened soft cheese (Queso fresco) of pH 6.5 at 7°C, reaching final numbers of around $10^7$ cfu/g after 10 days in the absence of starter culture. Growth also occurred at 3°C after a 10 day lag period (Mendoza-Yepes *et al*., 1999). When starter culture (*Lactococcus lactis* subsp. *diacetylactis*) was used no growth occurred at

these temperatures after 22 days storage. Predictive models are available concerning the growth of *L. monocytogenes* in Mexican-style soft cheese based on pH, salt and moisture content (Bolton and Frank, 1999).

Growth following a four day lag phase has been shown in Ricotta-type cheese produced without fermentation (direct acidification with acetic acid) stored at 6-8$^{o}$C where the pH of the cheese was close to 6 (Davies *et al*., 1997).

In cottage cheese inoculated after production, *L. monocytogenes* declined in numbers during storage at 4, 8 and 12$^{o}$C, with the rate of decline dependent on the pH, which varied from 5.06 to 4.69 among the three batches tested (Hicks and Lund, 1991). Similar results were shown by Piccinin and Shelef (1995). Cottage cheese production involves a moderate cooking step which has been shown to decrease numbers of *L. monocytogenes* by >100 fold (Ryser *et al*., 1985). However, other work has shown growth of *L. innocua* on cottage cheese of pH 5.0 at 5°C incubated under air or nitrogen following a 7 day lag phase, but not under carbon dioxide or 50%$N_2$:50%$CO_2$ (Fedio *et al*., 1994).

The use of rennet, gluconic acid or HCl to coagulate the curd during cottage cheese production has been compared (El-Shenawy and Marth, 1990). *L. monocytogenes* was added at high numbers to the milk used for cheese making. The organism was detected in cheese produced by rennet and HCl coagulation methods, but was not detected in curd or whey when gluconic acid was used as the coagulant.

In a study by Buazzi *et al*. (1992), the "stretching" process in mozzarella production was carried out for 3-4 minutes in 77$^{o}$C water. The curd reached 58-65$^{o}$C and this process was found to eliminate *L. monocytogenes* present at inoculated levels of 6.2 x $10^4$ /g from this particular kind of soft cheese.

### 3.2.3 Surface ripened cheeses

Many studies indicate that in surface mould ripened cheeses, whatever the type of milk (raw or pasteurised) conditions are more favourable for the growth of *L. monocytogenes* at the surface than at the centre. It is thought that this is due to higher pH values at the surface after the initial ripening period because of the proteolysis of casein, releasing amino acids and peptides (and ammonia) associated with the white mould ripening process. The amino acids and vitamins released may stimulate the growth of *L. monocytogenes* (Back *et al.,* 1993). For example, growth of *L. monocytogenes* has been demonstrated in laboratory-produced Camembert cheeses (Back *et al*., 1993; Ryser and Marth, 1987). While the numbers of *L. monocytogenes* declined during the initial ripening period (14 days), growth was recorded at the surface in the subsequent storage period at temperatures between 3-15$^{o}$C, with growth being faster and to higher final numbers with increasing temperature. At the centre of the cheese, the numbers declined at all temperatures except at 15$^{o}$C. This was reflected in the measured pH; where the surface pH raised 1-3 units over the ripening and storage period, while the centre remained at around pH 5. After 25 days at 15$^{o}$C, a yellow-green mould grew on the surface of some cheeses, and an odour of rotten cabbage was noted. Affected areas of the cheese became soft indicating extensive proteolysis. The centre and surface pH rose to >7.0 after 40 days with corresponding growth of *Listeria monocytogenes* at the centre and surface.

In the case of red smear cheese, the microbial activity at the surface of the cheese also has the effect of raising pH allowing growth of *L. monocytogenes* to occur at the surface, if present (Back *et al.,* 1993).

Differences between raw and pasteurised milk cheeses affecting the growth of *Listeria monocytogenes* have been described following analysis of 240 cheeses (Ennahar *et al.,* 1994). In cheese made from raw milk, the bacterium appears in the rind when the pH becomes >6.3 (after 9 days ripening). In cheese made from pasteurised milk, the pH of the rind increases more slowly with the bacterium appearing in the rind after 16 days ripening. *Listeria monocytogenes* did not grow in the centre of the raw or pasteurised milk cheeses in these experiments.

Back *et al*. (1993) reported the growth of *L. monocytogenes* when inoculated onto the surface of a number of soft cheeses, including Brie and Lymeswold.

Pini and Gilbert (1988) found that the *L. monocytogenes* count of a French soft cheese was 400 times higher at the surface (pH 6.5) than at the centre (pH 5.5).

In work with soft cheese made from goats' milk by a purely fermentative process (i.e. no involvement of rennet), *L. monocytogenes* inoculated into the raw milk was detectable in the cheese at all stages of ripening and storage (Morgan *et al*., 2001). In this cheese, the curd had a low pH (4.3) but this rose during storage. In contrast, *L. monocytogenes* grew but then declined in numbers quickly in Spanish Afuega'l Pitu cheese, which is made using a starter culture and rennet (Margolles *et al*., 1997). In this cheese however the pH fell to around 4.5 in less than a day and reached pH 4 after 2 days.

In contrast to most of the information above, numbers of *L. monocytogenes* reduced in Italico soft cheese during storage at 4$^{o}$C (Comi *et al.,* 1990) although it was detectable by enrichment at the end of the ripening period. The pH of this cheese started at approximately 5.2 and rose close to 6.0 in some cases, but the water activity decreased marginally during ripening.

### 3.2.4 Interior mould cheese ripening processes

Blue vein cheese undergoes an internal ripening method after being inoculated with *Penicillium* spores. Blue vein cheeses are usually in the semi-soft cheese category; however, some surface ripened soft cheeses can also be inoculated producing cheese that has both mould ripening characteristics (e.g. Blue brie).

Comparisons have been made between interior and surface methods of ripening. Kinderlerer *et al*., (1996) carried out a study comparing (internally ripened) blue veined cheese to soft surface mould ripened cheese, made from unpasteurised milk. *L. monocytogenes* was isolated only from the surface mould ripened cheese. Higher concentrations of free medium chain fatty acids (MCFA) were found in the veins of blue mould ripened cheese, and this was suggested to be due to lipolytic enzymes produced by the mould. The study concluded that the higher concentrations of MCFA present in the blue veins of internally mould-ripened cheeses (as opposed to surface ripened cheeses) could act as a natural preservative and inhibit the growth of *Listeria* in conditions where they might be expected to grow.

### 3.2.5 Salt cured or pickled cheeses

In Feta cheese *L. monocytogenes* was shown to increase in numbers during manufacture and the early stages of brine immersion. The pH fell to around 4.5, but *L. monocytogenes* survived well in stored cheese for up to 3 months, although one of two isolates declined around a thousand fold over this time period (Papageorgiou and Marth, 1989). A study of traditional Feta production in Greece failed to detect *L. monocytogenes* in samples taken at various stages of production in three different dairies (Manolopoulou *et al*., 2003).

Growth in other pickled cheeses has been reported (Abdalla *et al*., 1993). Growth occurred because the salt concentration (around 5%), was not high enough to inhibit growth of *L. monocytogenes*, but was high enough to prevent lactic acid production and so the pH remained high (6-7).

### 3.2.6 Whey cheeses

Whey cheese produced by the concentration method involves a heating process to above 70°C that would be sufficient to inactivate any *L. monocytogenes* in the whey. *L. monocytogenes* was shown to grow in inoculated fresh heat evaporated whey cheeses (such as Myzithra, Anthotyros and Manouri cheese) when incubated at 5, 12 and 22$^{o}$C (Papageorgiou *et al*., 1996). The pH of these cheeses was initially around 6 and only reduced to pH 5 under some circumstances. The moisture of the cheeses was up to 70%. Growth of the organism was therefore not unexpected.

Growth following a four day lag phase has been shown in Ricotta-type cheese produced without fermentation (direct acidification with acetic acid) stored at 6-8$^{o}$C where the pH of the cheese was close to 6 (Davies *et al*., 1997).

### 3.2.7 Summary

One of the major papers used in the FDA Risk Assessment providing many of the data sets was a comparative study of the ability of *L. monocytogenes* to grow on the surface of 24 types of cheese available in the USA (Genigeorgis *et al*., 1991). Overall, a highly significant correlation of *Listeria* growth with cheese pH values >5.5 and/or the absence of lactic acid starter cultures during cheese manufacture were observed. Ricotta, produced by direct acidification and with a high pH, was the best growth substrate since it supported growth at 4 to 30$^{o}$C despite the presence of acetic acid and the preservative, potassium sorbate.

For soft ripened cheeses, given that water activities present in these cheeses are, by definition, not inhibitory to the pathogen (with a possible exception noted for Italico cheese (Comi *et al.* 1990)), higher pH conditions occurring at the surface can result in growth of *L. monocytogenes*.

The organism may be introduced at many points in production, from being present in raw milk or whey to surface contamination of product prior to packaging or after purchase in the home of the consumer. The fate of the pathogen when introduced into the cheese at various points in the process will be very much dependent on the specifics of that process. For example *L. monocytogenes* introduced into Mozzarella production in the raw milk is unlikely to survive as there is an extra heating step in the production of this kind of cheese (the

"stretching" step), while in white pickled cheese *L. monocytogenes* added to the pasteurised milk after cooling and prior to the addition of rennet grew in the product (Abdalla *et al*., 1993).

Given that soft cheese in New Zealand will be made from pasteurised milk, the ability of *L. monocytogenes* to grow on cheese when introduced post-production is likely to be of more relevance than its ability to grow during manufacture. Most contamination is likely to occur at the surface, and it appears that surface conditions (especially pH) in most types of soft cheese (unripened and ripened) are not inhibitory for *L. monocytogenes* growth.

Despite these indications, the variability in processing of soft cheeses, the potential for small process changes by individual manufacturers, and the variable results from scientific studies, means that it is not possible to make a fully reliable prediction about the behaviour of *L. monocytogenes* in types of soft cheese.

## 3.3 The Food Supply in New Zealand

### 3.3.1 Production

Total cheese production in New Zealand in 2002 was 311,000 tonnes (MAF, 2002), and approximately 90% of this production was exported (277,000 tonnes). The balance (40,000 tonnes) equates to a per capita consumption of approximately 27 g/person/day, which is in good agreement with consumption estimates derived from other sources (see Section 5.2). Total cheese production decreased to 275,000 tonnes in 2003 (MAF, 2003). Exports were actually higher than production (293,000 tonnes; MAF, 2003) due to Fonterra exporting a large amount of product from its inventory.

While New Zealand's level of cheese production is modest compared to countries such as the United States, France and Germany, New Zealand is one of the largest exporters of cheese in the world market (http://www.fas.usda.gov/psd/complete_tables/DA-table2-136.htm). Major markets for New Zealand cheese include Japan, the United States and Australia (MAF, 2003).

Two large companies; New Zealand Dairy Foods (NZDF) and Mainland dominate the domestic cheese market in New Zealand.. Both of these companies sell cheese in New Zealand under a range of brand names. The market also includes two significant medium-size producers; Puhoi Valley Cheese Company (now owned by NZDF) and Kapiti. Other companies producing cheese in New Zealand are very small by comparison.

Registered dairy factories as at 22/11/04 are listed on the following website; http://www.nzfsa.govt.nz/dairy/registers-lists/reg-fac.htm. An extract is reproduced in Table 3 showing the cheesemaking companies registered factories, their registration number, cheese type produced (soft, cream, cottage and/or processed) along with overseas markets information. Cheesemakers who make hard cheese only are not included in Table 3. Production volumes and type of milk used (cow, ewe, goat) are not given in the information on this website.

**Table 3: Registered cheesemaking premises in New Zealand**

| Reg. No. | Name of premises | Type of Cheese | Markets |
|---|---|---|---|
| 137 | Art of Cheese Ltd. | Soft | |
| 94 | Barry's Bay Cheese | Soft | |
| 595 | Blue River Dairy Products Ltd. | Soft | BR |
| 905 | Canaan Cheeses | Soft | |
| 1630 | Delago Limited | Soft,Cottage,Cream | |
| 32 | Evansdale Cheese Factory | Soft | |
| 1203 | Hautapu Cheese Dev. Fonterra Ltd. | Soft, Cream | SL |
| 1573 | Clandeboye Cheese, Fonterra Ltd. | Soft | BR, EU, NC, PA, SL |
| 2573 | Lichfield Cheese 1 & 2, Fonterra Ltd. | Soft | BR, EU, SL |
| 3673 | Edendale cheese, Fonterra Ltd. | Soft | BR, EU, SL, PA |
| 4103 | Edgecumbe Butter Dev. Fonterra Ltd. | Soft | BR, EU, SL |
| 4773 | Whareroa Cheese 1 & 2, Fonterra Ltd. | Soft | BR, EU, NC, PA, SL |
| 6073 | Te Rapa, Fonterra Ltd. | Cream, Soft | BR, EU, CR, SL |
| 7373 | Waitoa Cheese, Fonterra Ltd. | Soft | BR, EU, SL |
| 7473 | Stirling Cheese, Fonterra Ltd. | Soft | BR, EU, PA, SL |
| 967 | Fromage du Nord Ltd. | Soft | |
| 168 | Kapiti Fine Foods Ltd. | Soft | |
| 715 | Kingsmeade Partnership Ltd. | Soft | |
| 31 | Natural Pak, Mainland Products Ltd. | Soft | BR |
| 35 | Christchurch Cultured Foods, Mainland Products Ltd. | Cream | |
| 6 | Mainland Products Ltd. | Cottage, Cream | EU |
| 38 | Mainland Products Ltd. – Grated Cheese Division | Soft | BR |
| 1450 | Matatoki Farm Cheese | Soft | |
| 41 | New Zealand Dairy Foods Ltd. | Cottage, Cream | |
| 530 | South Island Beverages Plant (NZDF) | Soft | |
| 4 | Puhoi Valley Cheese Co. Ltd. (Div. of NZDF) | Soft | |
| 785 | Talbot Forest Cheese Ltd. | Soft | |
| 60 | White Stone Cheese Ltd. | Soft | |
| 750 | Zany Zeus | Soft | |

BR=Brazil EU=European Union
SL=Sri Lanka CR=Costa Rica
PA=Panama NC=Nicaragua

3.3.2 Imported foods

It is a legal requirement that soft cheese imported into New Zealand must be produced from pasteurised milk. Imported cheese (of all types) is reported to make up 15% of the cheese consumed in New Zealand (MAF, 2003). Information on the production systems of individual imported cheeses is not readily available and therefore it is difficult to assess

whether production systems for domestic and imported soft cheese are comparable in terms of microbial safety.

Import statistics for the year ending March 2003 record a total of 1,900 tonnes of cheese being imported into New Zealand. Of this, the majority comes from Australia (82%), followed by Denmark (7%), France (3.0%) and Italy (2.2%). It is uncertain what proportion of this is soft cheese, as the bulk of the cheese imports are classified as 'cheese, (other than in tins, not grated, powdered or processed), not elsewhere specified'.

There is a category of fresh cheese (unripened or uncured, including whey cheese) that of blue vein cheese (no moisture content stated) is listed at Australia 769kg, Denmark 34,403kg, France 602kg and the UK 7,186 kg.

Border surveillance exists in New Zealand for high risk foods. All soft cheese and grated/powdered cheese including low moisture cheese such as Cheddar, Colby, Cheshire, Egmont and Gouda are included, principally because of the risk of *Listeria monocytogenes* contamination. The cheeses are monitored by sampling and testing for the organism, sampling regimes are outlined on the following website: http://www.nzfsa.govt.nz/imported-food/high-risk/01softcheesenf.htm. A nil or "zero-tolerance" for *Listeria monocytogenes* per 25g grated and soft cheeses is the criteria when deciding if the consignment is safe for release.

# 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 (Invasive) 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 immuno-compromised, 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 and 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

It is generally accepted that if pathogens such as *Listeria* are present in high fat content milk products such as cheese, the fat micelles can protect the pathogens against human gastric acids (D'Aoust, 1985).

### 4.3.1 Listeriosis

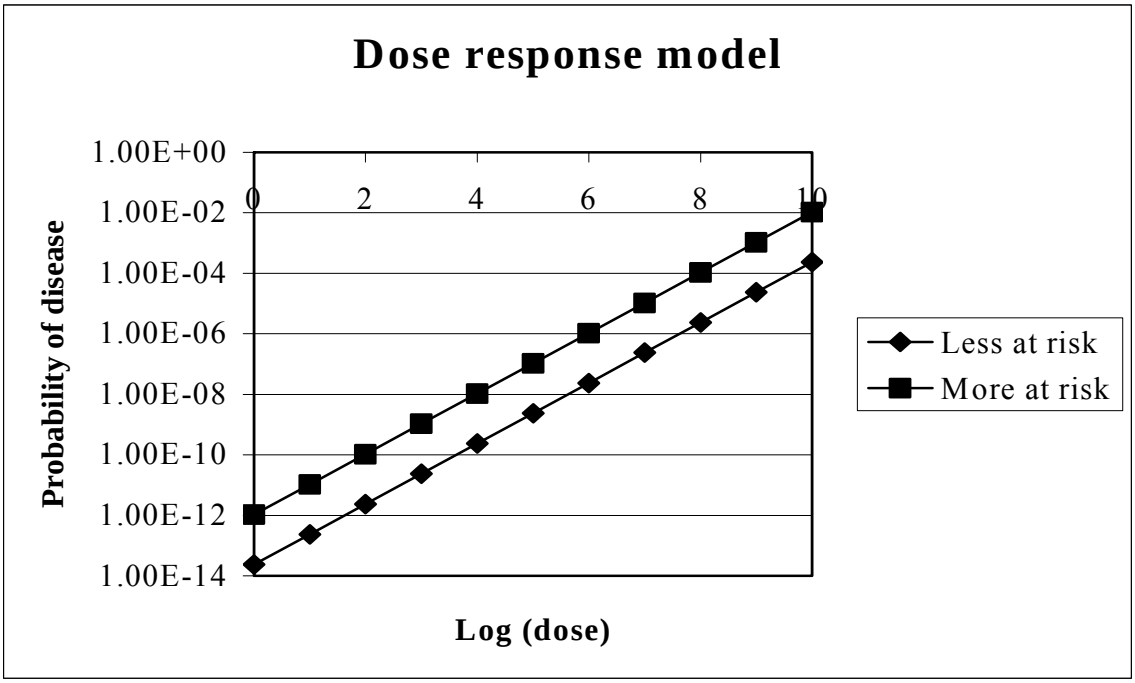
It is becoming increasingly realised that the only completely safe dose of *L. monocytogenes* is zero, even in healthy people. However the probability of invasive disease following exposure to even moderate levels of cells is very low.

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

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

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 that 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 3 shows dose response curves for at risk and not at risk groups. Information provided by Dr. Tom Ross, University of Tasmania, and is that used in the FAO/WHO *Listeria* quantitative risk assessment.

**Figure 3: Dose response models at median values for R for invasive disease caused by *L. monocytogenes.***



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. 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 $1.8 \times 10^7$ *L. monocytogenes* cells/g became ill with symptoms of febrile gastroenteritis (Sim *et al*., 2002). Assuming approximately 100g of ham was eaten by each person at the meal, then the dose ingested to produce this response was of the order of $10^9$ cfu. In the outbreak described by Dalton *et al.* (1997), an attack rate of 75% was recorded where the median number of cells consumed was estimated as being as high as $2.9 \times 10^{11}$ 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 $1.9 \times 10^5$ 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 2003 calendar year were 56,130 (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.

Based on a figure of approximately 56,000 live births per annum and the number of perinatal cases of listeriosis in 2003 (6), this equates to an incidence of approximately 11 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 2003, 788 people in New Zealand were notified with AIDS. At the same date 1,974 people in New Zealand were infected with HIV (http://www.moh.govt.nz/aids.html). This represents 0.05% 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 is 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).

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 2002, 117 kidney transplants were performed in New Zealand bringing the total number of surviving New Zealand kidney transplant recipients to 1114 (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 2003; kidney (excluding living donor transplants) 66, liver 38, heart 22, lungs 14, pancreas 6 (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).

## 4.5 Serotypes Isolated from Soft Cheese and Human Cases

The terms "*subtyping*" or "*typing*" describe a test or assay, that is able to distinguish isolates of a microbial species from each other. There are a variety of typing methods, including reaction with antibodies (serotyping), interaction with bacterial viruses called "phage", and analysis of bacterial DNA by a number of different techniques. Subtyping tools can be valuable for:

- Outbreak identification
- Population studies, and,
- Further characterisation of the pathogen.

In outbreak identification and investigation, subtyping allows investigators to identify outbreaks out of the general dispersion of sporadic cases, provide tight specific case-definitions for outbreak investigations, link "unrelated" outbreaks, link cases to known outbreaks, provide clues about possible sources of an outbreak, and confirm epidemiological associations with a particular source. Studies of pathogen reservoirs and transmission routes benefit through ability of subtyping to follow strains from suspected sources. Additional levels of subtyping allow determinations of potential virulence, survival, antibiotic resistance etc.

There are various typing schemes for *L. monocytogenes* (ICMSF, 1996):

- Serotyping distinguishes 13 serovars, of which three account for most of the human cases of invasive listeriosis: serotype 4b is most common, while infections with serotypes 1/2a and 1/2b occur less frequently,
- Phage-typing can distinguish about 70% of strains,
- Multilocus enzyme electrophoresis; and,
- Nucleic acid fingerprinting.

While these typing schemes are useful in epidemiological outbreak investigations, they are of limited use in distinguishing pathogenic from non-pathogenic strains (ICMSF, 1996), and currently the majority opinion is that all strains should be regarded as potentially pathogenic.

Of 200 isolates from 19 cheeses purchased in Sweden, 97% (194/200) were serogroup 1/2 (33.5% were 1/2a and 58.5% 1/2b) (Loncarevic *et al*., 1998). The remaining isolates were serogroup 4b. At the time of the study, 41% of Swedish human cases were of serotype 1/2 and 59% serogroup 4.

Schönberg *et al*., (1989) tested 89 selected ripened soft European cheeses and 8 (9%) were positive for *L. monocytogenes.* Five isolates were serotype 1/2a and four 1/2b (one sample yielded both serovars). No isolates were identified as serotype 4b despite two thirds of human cases being recorded as being caused by this serotype. It has been noted that since 1989, the proportion of human cases caused by serotype 1/2, at least in Europe, appears to be reaching a similar proportion as those caused by serotype 4 (Loncarevic *et al*., 1998).

Pak *et al*., (2002) typed 3722 isolates from Swiss dairy products and dairy processing environments. The most common serotypes were 1/2b (38.6%), 1/2a (33.0%) and 4b (21.1%). Serotype 1/2b was more frequently isolated from hard and semi-hard cheese, while serotype 1/2a was more common in soft cheeses.

Differences in the serotypes and esterase types of *L. monocytogenes* from cheese and human cases have been noted in Belgium (Gilot *et al*., 1996). Esterase Type 1B-serotype 1/2a accounted for 44.2% of the cheese isolates but only 4.2% of the human isolates. However, when tested for pathogenicity in immuno-compromised mice, all isolates of this type were similar in their $LD_{50}$.

These studies show that serotypes of *L. monocytogenes* isolated from cheeses overseas are predominantly 1/2a and 1/2b, while those from human cases in the same countries include a high proportion (if not the majority) of serotype 4. It is possible that some strains of *L. monocytogenes* are adapted to the dairy environment and so are isolated more frequently from dairy products.

In New Zealand clinical isolates of *L. monocytogenes* for the period 1999 to 2003 were approximately evenly split between the 1/2 and 4 serotypes (Pat Short, ESR Enteric Reference Laboratory, Kenepuru Science Centre, personal communication, December 2003). Genotyping data are routinely produced for clinical isolates but, until very recently, this has not been the case for food isolates.

# 5 EXPOSURE ASSESSMENT

## 5.1 The Hazard in the New Zealand Food Supply: *Listeria* in Soft Cheeses

The ESR laboratory database contains results for testing of approximately 1,000 soft cheeses for *L. monocytogenes* (exact numbers cannot be determined, as the actual cheese type was not always recorded). *L. monocytogenes* was only isolated from one cheese sample, a Mozzarella – the sample had been rejected in Taiwan and was being re-imported into New Zealand (reference number P950276 on database). It is not recorded what the moisture content of the Mozzarella was. The majority of soft cheeses tested were imported cheese being tested before release onto the New Zealand market.

During 2003/2004 ESR carried out a survey of 307 soft and semi-soft cheeses (approximately 50 samples were of semi-soft blue cheese) for the presence of *L. monocytogenes*. Samples were stored till the end of shelf life before testing. Results from this survey found that no soft cheese samples tested positive for *L. monocytogenes,* although *L. welshimeri* was detected in a semi-soft blue cheese (Wilson, 2004). The survey included soft cheese types Camembert, Ricotta, Brie, Mozzarella and a range of blue cheeses, but not cottage or cream cheese. Wide ranges of large and small manufacturers were included.

## 5.2 Food Consumption: Total Cheese & Soft Cheese Consumption

### 5.2.1 Total cheese consumption

Analysis of data from the 1997 National Nutrition Survey (Russell *et al*., 1999) gives an estimate for the total per capita consumption of cheese by New Zealanders aged 15 years and over of 16.6 g/day. This estimate was derived by applying a standard set of recipes to cheese-containing foods such as cheesecake, pizza, cheese sauce, quiche, savoury muffins and scones, etc. to determine the amount of cheese contributed to the diet by these recipes. This estimate is similar to that derived in the 1991 Life in New Zealand Survey of 18 g/day (LINZ, 1992) and slightly lower than amount used for simulated typical diets in the 1997/98 New Zealand Total Diet Survey (adult males; 20 g/day, adult females; 18.9 g/day; Brinsdon *et al.*, 1999).

The 1995 Australian National Nutrition Survey gives a slightly lower estimate of cheese consumption for the Australian population aged 19 and over of 14.6 g/day, with males, on average, consuming more (16.2 g/day) than females (13.0 g/day) (Australian Bureau of Statistics, 1999).

The US Environmental Protection Agency gives consumption estimates for the total US population in the range 14-17 g/day (EPA, 1997). Similar estimates of 15.7 g/day have been made for the United Kingdom population; (http://statistics.defra.gov.uk/esg/publications/nfs/2000/default.asp).

European cheese consumption can be found at; http://www.cheeseboard.co.uk/new/trade/cheeseCon.htm. This source gives a value of 9.8 kg/head/year for the UK population or 26.8 g/day. Consumption in European countries is reported as ranging from 22.7 g/day (Portugal) to 66.0 g/day (France).

Information summarised in the GEMS/Food Regional diets indicates that cheese consumption is significantly greater in European style diets (28.0 g/person/day) than any other, followed by the Middle Eastern diet (8.5 g/person/day) and the Latin American diet (4.5 g/person/day). Cheese is not a significant food in the Far Eastern or African diets (http://www.who.int/foodsafety/publications/chem/regional_diets/en/).

### 5.2.2 Soft cheese consumption

The Qualitative Food Frequency Questionnaire (QFFQ) administered as part of the 1997 National Nutrition Survey asked questions of New Zealanders concerning the types and frequency of consumption of various types of cheese. Results for the total population aged 15 years and over are summarised in Table 4.

**Table 4: Frequency of consumption of various cheese types by the New Zealand population aged 15 years and over**

| Cheese type | Percentage of survey population consuming cheese type | | | |
|---|---|---|---|---|
| | Never | Rarely | 1-6 times/week | At least 1 time/day |
| Cream cheese | 56 | 41 | 3 | 0 |
| Cottage/Ricotta | 63 | 32 | 5 | 0 |
| Mozzarella/Feta/ Camembert | 54 | 40 | 5 | 0 |
| Edam/Gouda | 54 | 26 | 18 | 2 |
| Cheddar (Colby/Mild/Tasty) | 9 | 26 | 57 | 8 |
| Specialty | 62 | 33 | 5 | 0 |

The first three categories (Cream cheese, Cottage/Ricotta, Mozzarella/Feta/Camembert) cover the most common soft cheese types available in New Zealand. Women are more likely to consume all classes of soft cheese and more frequently (7.1% of respondents) than men (4.5% of respondents), while Maori and Pacific Islanders are less likely to consume soft cheeses than European and other New Zealanders. This translates into a higher per capita intake of soft cheese for women (2.0 g /day) compared to men (1.3 g /day). Those aged 25 to 64 (either gender) are more likely to consume soft cheese than the young or the old.

Table 5 gives an analysis of data from the 24-hour dietary record records in the 1997 National Nutrition Survey, giving the proportion of various cheese types to total cheese, on the basis of numbers of servings and on the basis of weight.

**Table 5: Proportions of different soft cheese types consumed in New Zealand (1997 National Nutrition Survey)**

| Cheese type | Percentage of total cheese consumed by number of servings | Percentage of total cheese consumed by weight | Estimated per capita consumption (g/day) |
|---|---|---|---|
| Brie | 1.4 | 0.9 | 0.15 |
| Camembert | 1.4 | 1.2 | 0.20 |
| Cottage | 2.5 | 3.0 | 0.49 |
| Cream | 4.2 | 4.4 | 0.73 |
| Feta | 0.6 | 0.5 | 0.09 |
| Mozzarella/bocconcini* | 0.4 | 0.4 | 0.06 |
| Quark/quarg/kwark | <0.1 | <0.1 | <0.01 |
| **Total (soft cheese)** | **10.5** | **10.4** | **1.72** |

* There are two varieties of Mozzarella, low moisture and soft. The variety of Mozzarella commonly used in pizzas is low moisture cheese and therefore not covered by this Risk Profile. Therefore it will be difficult to determine what proportion of Mozzarella is low moisture or soft in the figure given in this table. Bocconcini is a high moisture mozzarella cheese shaped into small balls.

## 5.3 Qualitative Estimate of Exposure

### 5.3.1 Number of servings of soft cheese and serving size

#### *5.3.1.1 Total population*

From the National Nutrition Survey (NNS), 279 individual dietary records were deemed to represent consumption of a serving of soft cheese. Using a total survey population of 4636 and a total New Zealand population of 4,054,200 (at 31 March 2004) (http://www.stats.govt.nz/):

Annual number of servings (total population) = 279 x 4,054,200/4636 x 365
= $8.9 \times 10^7$ servings

The FDA/FSIS (2003) risk assessment for *L. monocytogenes* in ready-to-eat foods does not use 'soft cheese' as a category descriptor, however, three of the categories appear to cover the majority of soft cheese types. These are listed below with their calculated annual number of servings;

- Soft ripened, $1.9 \times 10^9$;
- Fresh soft, $7.1 \times 10^7$; and,
- Soft unripened cheese, $4.4 \times 10^9$.

For these cheese types, approximately 1 ounce (30g) was representative of a typical serving size and the total calculated per annum number of servings for the total population is calculated at $63.7 \times 10^{8.}$ Based on a total population of 293,494,282 (at 14 June 2004) (http://www.census.gov/cgi-bin/popclock), these figures produce remarkably similar results for the number of servings per person per annum of 21.7 for the USA and 21.9 for New Zealand.

*5.3.1.2 Elderly population*

From the NNS, 50 individual dietary records were deemed to represent consumption of a serving of soft cheese 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.

Annual number of servings (elderly population) = 50 x 615,580 /1087 x 365
= $1.03 \times 10^7$ servings

*5.3.1.3 Perinatal population*

The assumptions made by the FDA/FSIS to calculate the perinatal population were used to calculate the number of perinatal servings for pregnant women in the New Zealand population. This approach has recently (September 2003) been altered (http://www.foodsafety.gov/~dms/lmr2-toc.html). This was done by multiplying the number of servings for the intermediate population (see below) by the annual pregnancy rate and by 0.25 (3/12) to estimate the number of pregnant women in the last trimester – the period of greatest susceptibility for perinatal listeriosis. A pregnancy rate for New Zealand could not be located and the US figure of 2.77% was used, however, trial calculations for the New Zealand population (live births plus abortions x 1.33, to account for the difference between gestation period and year length, as a percentage of the intermediate age population) gave a similar figure.

Annual number of servings (perinatal population) = $7.46 \times 10^7$ x 0.0277 x 0.25
= $5.17 \times 10^5$ servings

*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 population from the total population.

Annual number of servings (intermediate population) = $7.46 \times 10^7$ servings

5.3.2 Serving sizes

Based on the data in the NNS database the 50, 75, 95, and 99$^{th}$ percentile serving sizes for various soft cheese types are given in Table 6.

**Table 6: New Zealand and US serving sizes for soft cheeses**

| Cheese | Percentile serving sizes (g) | | | |
|---|---|---|---|---|
| | 50th | 75th | 95th | 99th |
| **New Zealand (Russell *et al*., 1999)** | | | | |
| Brie | 15 | 22 | 61 | ID |
| Camembert | 20 | 31 | 51 | ID |
| Cottage | 28 | 55 | 78 | 118 |
| Cream | 21 | 37 | 98 | 119 |
| Feta | 15 | 24 | 74 | ID |
| Mozzarella/bocconcini | 30 | 30 | 41 | ID |
| Quark | ID | ID | ID | ID |
| **FDA/FSIS** (2003:page 35**)** | | | | |
| Soft ripened | 28 | 48 | 85 | 168 |
| Soft unripened | 29 | 105 | 226 | 420 |
| Fresh soft cheese | 31 | 85 | 246 | 246 |

ID Insufficient data to determine

5.3.3 Contamination frequency

Available New Zealand data (ESR Laboratory database, recently completed NZFSA soft cheese project) indicate that the frequency of contamination of soft cheese with *L. monocytogenes* is extremely low.

The potential for cross contamination at the retail level has been reported by Farber *et al*. (1987) and Rørvik and Yndestad (1991). However, a similar contamination frequency was found in whole and pre-cut wedges of cheese purchased in Sweden suggesting this did not play an important role there (Loncarevic *et al*., 1995).

5.3.4 Predicted contamination level at retail

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No New Zealand data are available to indicate the numbers of bacteria in soft cheese contaminated with *L. monocytogenes* at retail.

5.3.5 Growth rate during storage and most likely storage time

Growth of *L. monocytogenes* in most soft cheeses during storage is likely where conditions are favourable. *Listeria* can grow at low temperatures, which overcomes one of the key food safety controls, refrigeration. Given that ripening soft cheeses involves time in humid conditions, to encourage mould or microflora growth, potentially high numbers of the pathogen can be reached under favourable conditions. In those cheeses that are ripened using surface moulds, the potential for *L. monocytogenes* growth will be greater because of the pH-raising mould growth at the surface of the cheese. Storage times can vary from immediate consumption after production for fresh unripened cheeses to several weeks for ripened soft cheeses.

The FDA/FSIS quantitative risk assessment (2003;Appendix 5) has modelled exponential growth rates, taking data from levels of contamination at retail and post retail growth. The

most likely storage times and maximum times that US consumers store cheese has been evaluated as follows;

| | Most likely | Maximum |
|---|---|---|
| • fresh soft | 1 to 5 days | 15 to 30 days |
| • soft unripened | 6 to 10 days | 15 to 45 days |
| • soft ripened | 6 to 10 days | 15 to 45 days |

5.3.6 Culinary heat treatment

While approximately 40% of all cheese servings are associated with meals in which the cheese is likely to be heat treated, this appears to be less applicable to soft cheeses with only about 5% of servings likely to be heat-treated. It should be noted that Mozzarella, commonly used in pizza production has mainly two varieties (see section 3.1.4). The type used on pizzas is low moisture (<50%) and therefore this variety does not fit the description of cheese in this Risk Profile.

5.3.7 Exposure summary

Daily consumption of soft cheese in New Zealand appears to be at fairly low levels (approximately 5% of the population) and serving sizes are modest compared to those in the US (see sections 5.2.2 and 5.3.2).

Available New Zealand data (Wilson, 2004) indicates that the frequency of contamination of soft cheese with *L. monocytogenes* is extremely low. However, when post-pasteurisation contamination does occur, the ability of the organism to grow at refrigeration temperatures coupled with possibly long storage times for soft cheeses, means that the final numbers reached may be high.

## 5.4 Overseas Context

Information from the scientific literature on the prevalence of *L. monocytogenes* in soft cheese overseas has been summarised in Table 7. Samples tested are categorised into the following; P (pasteurised), R (raw) and U (unknown) milk types. Quantitative figures are given where known.

Most studies (68%) report prevalences of less than 10%, and in 28% of the surveys *L. monocytogenes* was not detected. Where prevalences were very high (i.e. 40-50%) the countries involved (Brazil and Costa Rica) may have sectors of their food industries that are less technically advanced than in the other countries surveyed. The very high prevalences reported by the UK study of McLauchlin *et al*. (1990) were not included in this analysis as they were associated with an outbreak. Where positive samples do occur the numbers present may be quite high; the highest being $1.5 \times 10^6$/g.

There are few studies that allow a comparison of cheeses from raw or pasteurised milk. The data indicate that both types may be contaminated. Four studies give a higher prevalence of contamination in soft cheeses from raw milk compared to cheeses from pasteurised milk, while the reverse was true in two studies.

**Table 7: Overseas prevalence and quantitative data for *L. monocytogenes* in soft cheeses**

| Country/ Region | Cheese Type | No. samples tested | No. (%) positive for *L. monocytogenes* | Reference |
|---|---|---|---|---|
| Australia | Soft cheeses | 437 - U | 15 (3.4) | Arnold and Coble, 1995 |
| | Soft cheeses (listeriosis investigation) | 28 - U | 1 (3.6) | |
| Brazil | Homemade Minas Frescal (Brazilian soft white cheese) | 17 R | 7 (41.1) | Da Silva *et al*., 1998 |
| | | 33 U | 1 (3.0) | |
| | Manufactured Minas Frescal and Ricotta | 53 U | 3 (5.7) | |
| | Ripened (Gorgonzola, Brie, Roquefort) | | | |
| Canada | Locally produced | 182 – U | 0 (0) | Farber *et al*., 1987 |
| | Imported (Soft and semi-soft – moisture content of 'semi-soft' not given) | 192 - U | 2 (1.0) (both positives from France, phosphatase test positive)[1] | |
| Chile | Soft cheese | 256 - U | 2 (0.8) | Cordano and Rocourt, 2001 |
| Costa Rica | Soft cheese (sold as pasteurised but no strict controls on thermal treatments) | 20 - U | 9 (45.0) no values | Monge *et al*., 1994 |
| England | Soft cheeses | 251 - U | 1 (0.4) | MacGowan *et al*., 1994 |
| England and Wales | UK and imported Cows milk soft ripened cheese | 769 | 63 (8.2) (18/63 R 8/63 P 37/63 U) 13 samples out of the 63 exceeded $10^3$/g, 3 of these >$10^5$/g. Of these 13, 7 were made from raw milk. | Greenwood *et al*., 1991 |
| | Cows milk soft unripened cheese | 366 | 4 (1.1), all 4 samples <500/g | |
| Europe | Red Smear (soft varieties only). | 192 soft<br>Paper states 163 – P & 166 R samples in the survey but semi-soft and hard cheeses are included, with no differentiation | 13(6.8)<br>(5 from raw milk, 8 from pasteurised)<br>Counts ($cm^{-2}$) 7 <10, 1 50, 1 <1,000, 1 2,400, 1 17,000, 1>3,000, 1 > 100,000 | Rudolf and Scherer, 2001 |

| Country/ Region | Cheese Type | No. samples tested | No. (%) positive for *L. monocytogenes* | Reference |
|---|---|---|---|---|
| Germany | Ripened soft European cheeses | 89 (66 German cheeses, 23 other European) - U | 8 (9) | Schönberg *et al.*, 1989 |
| Hungary | Hungarian Soft cheese<br><br>Mould cheese (Hajdu) | 25<br><br>10 | 0 (0)<br><br>2 (20%) (milk had "minimum heat treatment") | Rodler and Korbler, 1989 |
| Ireland | Irish soft farmhouse cheeses | 10 (5 R & 5 P) | 0 (0) | Coveney *et al.*, 1994 |
| Italy | Cheeses with short ripening periods (marscapone, mozzarella, crescenza).<br><br>Cheeses with a few weeks ripening period, thin rind (italico, caciotta) | 54 - P<br><br>67 - U | 0 (0)<br><br>2 (3.0) (from rind) | Massa *et al.*, 1990 |
| Italy | Fresh cheese<br><br>Soft cheese | 239<br><br>1284 | 0 (0)<br><br>65 (15.4)<br><br>all < $10^2$/g<br>"no correlation with the type of milk used" | Comi *et al.* 1990 |
| Netherlands | Imported soft cheese from France | 69 (63 wholesale, 6 retail) | 10 (14.5%)<br>36 P, all negative<br>9/14 R, contaminated<br>1/19 U contaminated<br>Numbers in 7 samples ranged from $10^3$-$10^6$/g | Beckers *et al.*, 1987 |
| Northern Ireland | Soft cheeses | 33 - U | 0 (0) | Harvey and Gilmour, 1992 |
| Norway | Soft cheese (imported), 90% cut in store, 10% unopened, pre-packed portions | 90 - U | 10 (11.0), 7 samples cut from larger cheeses in same store & same serotype at low levels – possibly handling contamination,<br>4 samples > $10^3$/g, 6 < $10^3$/g | Rørvik and Yndestad, 1991 |
| Spain (Navarra) | Soft cheese | 99 - U | 1 (1.0) | Vitas *et al.*, 2004 |
| Spain | Fresh cheeses | 8 - U | 0(0) | Rota *et al.*, 1992 |
| Spain | Fresh cheese | 23 - P | 1 (4.3) | Calpe, 1996 |
| Spain (Tenerife) | Soft goat's cheese (local) | 33 - R | 4 (12.1) | Perez *et al.*, 1998 |

| Country/ Region | Cheese Type | No. samples tested | No. (%) positive for *L. monocytogenes* | Reference |
|---|---|---|---|---|
| Sweden | Locally produced – 27 samples<br><br>Imported (soft and semi-soft cheeses) – 306 samples | Total of 333 samples, of which;<br><br>302 samples pasteurised,<br><br>31 (9%) of the samples from raw milk (30 from France and 1 from Sweden) | Total 20/333 (6)<br><br>(Local 0/27 (0))<br><br>(Imported 20/306 (6.5)) (18 positives from France 5 P and 13 R)), 1 each from Italy (P) and Germany (P))<br><br>Count data: 15 $<10^2$/g, 2 $10^2$-$10^3$/g 2 $10^3$-$10^4$/g, 1 $>10^3$/g. | Loncarevic *et al*., 1995 |
| UK | Various soft and semi-soft | 1437 includes 72 R 405 P 960 U | 16 (1.1)<br><br>1/72 R (1.4)<br>2/405 P (0.5)<br>13/960 (1.3)<br>All positives at <10/g, | Nichols *et al*., 1996 |
| UK | UK produced (45 samples)<br><br>Imported (177 samples) | 222 includes 16 R 41 P 165 U | 23/222 (10.4)<br><br>UK: 2/45 (4.4) Treatment U<br><br>Imported: 21/177 (11.9) Includes 9 U 2 R 10 P Count data: 12 $<10^2$/g, (8 U, 3 P, 1 R) 1 $10^2$/g(P), 7 $10^4$/g(1 R, 6 P), 3 $10^5$/g (all U) | Pini and Gilbert, 1988 |
| UK | Local Anari whey cheese<br><br>Retail<br><br>from factory (from a manufacturer associated with a case) | <br><br>Retail 25<br><br>Factory 24 (Production involves cooking to 85°C) | <br><br>16 (64.0)<br><br>12 (50.0) | McLauchlin *et al*., 1990 |
| USA- California<br><br>Maryland | Fresh soft cheese | 1481 - U<br><br>1450 - U | 1 (0.1) >10-$10^2$/g<br><br>4 (0.3) Counts: 0.04-0.1 2 >10-$10^2$/g 3 | Gombas *et al*., 2003 |
| USA | Locally-produced ricotta | 3 - U | Contained a geometric mean of 1.5 x $10^6$/g | Datta *et al*., 1988 |

[1] The presence of phosphatase in cheese is taken to indicate that it has been made with unpasteurised milk. However the packaging from both positive samples indicated that they were made from pasteurised milk.

U=Unknown, P=Pasteurised, R=Raw.

# 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

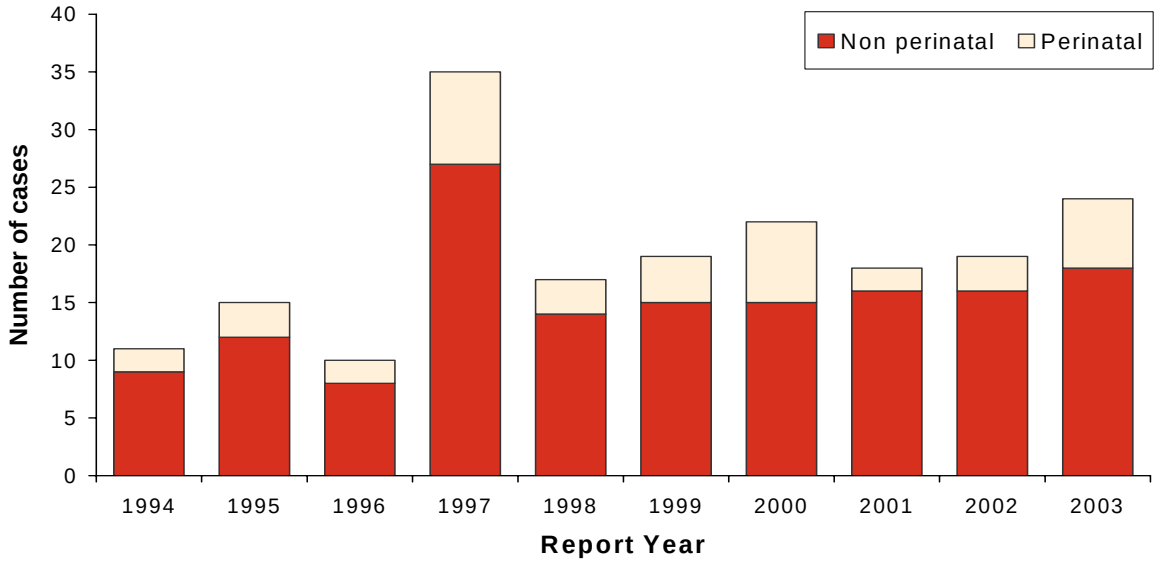
Notification and mortality data from the EpiSurv database for listeriosis for the years 1990 to 2004 are given in Table 8. It is important to note that these cases are not associated with any specific transmission vehicle.

**Table 8: Reported cases of invasive listeriosis and mortality from 1990 to 2004 in New Zealand.**

| Year | Listeriosis cases | Deaths (perinatal) | Deaths (non-perinatal) | Reference (Kieft *et al*., 2000, unless otherwise stated) |
|---|---|---|---|---|
| 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 | Lopez *et al*., 2001 |
| 2001 | 18 | 1 | 1 | Sneyd *et al*. 2002 |
| 2002 | 19 | 3 | 0 | Sneyd and Baker, 2003 |
| 2003 | 24 | 2 | 2 | ESR, 2004 |
| 2004 | 26 | 2 | 3 | ESR, 2005 |

Figure 4 shows a graphical representation of annual case numbers of listeriosis with the proportions of perinatal and non-perinatal cases identified.

**Figure 4: Listeriosis notifications by year 1994 – 2003.**




*Reproduced from ESR (2004)*

### 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 1997-2004 are given in Table 9. 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 9: Outcome data for listeriosis in New Zealand, 1997 to 2004**

| Year | Hospitalised cases | Fatalities | Reference |
|---|---|---|---|
| 1997 | 33/33 (100%) | 8/35 (22.9%) | ESR, 1998 |
| 1998 | 16/16 (100%) | 0/17 (0.0%) | Perks *et al.*, 1999 |
| 1999 | 18/19 (94.7%) | 3/19 (15.8%) | Kieft *et al.*, 2000 |
| 2000 | 22/22 (100%) | 6/22 (27.3%) | Lopez *et al.*, 2001 |
| 2001 | 17/18 (94.4%) | 2/18 (11.1%) | Sneyd *et al.*, 2002 |
| 2002 | 13/13 (100%) | 3/19 (15.8%) | Sneyd and Baker, 2003 |
| 2003 | 22/22 (100%) | 4/24 (16.7%) | ESR, 2004 |
| 2004 | 25/26 (96%)* | 5/26 (19.2%) | ESR, 2005 |

*One case, hospitalisation status not recorded

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 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 a genuine risk factor related to the symptoms presented, 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 cheese was implicated from the financial years; 1997/98 to 2002/03 were reviewed. In this period, consumption of cheese was investigated in approximately 60 episodes of suspected food poisoning. However, cheese was often only one of a number of foods tested, particularly in investigations of cases of listeriosis. In approximately one quarter of these episodes it was possible to identify that the implicated cheese was a soft cheese (cream cheese, cottage cheese, feta cheese or brie). In only one instance was cheese confirmed as the source of the suspect food poisoning and in this episode, the causative organism was found to be *Salmonella*.

#### 6.1.4 Outbreaks

Outbreaks of infection with *L. monocytogenes* in New Zealand are rare. From 1997 to 2003 only three have been reported to the national surveillance system. None of these outbreaks were linked to consumption of cheese. Two of the outbreaks were connected, and associated with consumption of ham and other ready-to-eat meats (Sim *et al.*, 2002; Whyte, 2000) while no food vehicle was identified in the other outbreak (Anonymous, 1998). An earlier small outbreak, in 1992, was linked to the consumption of smoked mussels (Brett *et al.*, 1998).

### 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 10 indicate that New Zealand's rate is similar to that of other developed countries.

**Table 10: Comparison of listeriosis incidence between countries**

| Country | Period | Rate /100,000 | Reference |
|---|---|---|---|
| **New Zealand** | **1999** | **0.5** | **Kieft *et al.*, 2000** |
| **New Zealand** | **2000** | **0.6** | **Lopez *et al.*, 2001** |
| **New Zealand** | **2001** | **0.5** | **Sneyd *et al.*, 2002** |
| **New Zealand** | **2002** | **0.5** | **Sneyd and Baker, 2003** |
| **New Zealand** | **2003** | **0.6** | **ESR, 2004** |
| **New Zealand** | **2004** | **0.7** | **ESR, 2005** |
| Australia | 2000 | 0.3 | Lin *et al*., 2002 |
| Australia | 2002 | 0.3 | OzFoodNet Working Group, 2003 |
| Canada | 1990-1998 | 0.1-0.3 | Health Canada, 2000 |
| Denmark | 2001 | 0.7 | Dansk Zoonosecenter, 2002 |
| Denmark | 2002 | 0.5 | Danish Zoonosis Centre, 2003 |
| France | 1997 | 0.4 | De Valk *et al*., 1998 |
| UK | 1983-2001 | Approx. 0.2 - 0.5 | PHLS, 2002 |
| USA | 2000 | 0.4 | Anonymous, 2001 |
| USA | 2002 | 0.3 | Anonymous, 2003 |

6.2.2 Contributions to outbreaks and incidents

As shown by the data in Table 11, most cases of infection with *L. monocytogenes* are sporadic rather than part of outbreaks, and outbreaks caused by *L. monocytogenes* make up only a very small proportion of the total outbreaks reported.

**Table 11: 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<br>41 (0.0) cases | Todd, 1992 |
| USA | 1989 | 1 (0.2) | 2 (0.0) cases | Bean *et al*., 1996 |
| USA | 1993-1997 | 3 (0.1) | 100 (0.1) cases | Olsen *et al*., 2000 |

NS = Not stated

Table 12 summarises information on well-documented sporadic cases of listeriosis (Codex 2002:30). The case outcomes appear reasonably typical, with death resulting in three of the cases where the outcome was reported. Of the sixteen cases summarised, three (19%) were due to consumption of soft cheese contaminated with *L. monocytogenes*. Some of the sporadic case details are as follows;

A case report from the UK (in Table 12: England, 1988) provides compelling evidence for a single case of listeriosis caused by the consumption of contaminated soft cheese (cross reference Table 5, also Azadian *et al*., 1989; McLauchlin *et al*., 1990). Here a healthy and immunocompetent 40 year old woman contracted listeriosis after the consumption of Anari, a

whey cheese. Production involved a cooking stage to 85°C. Four cheeses from the same batch purchased from the same shop yielded 3.0- 5.0 x $10^7$ *L. monocytogenes*/g, and the phage type of the organism in the cheese was indistinguishable from that isolated from the case. The serotype of the isolates was 4b. Given an estimated consumption of 85g, the estimated dose consumed was 3.5-4.3 x $10^9$ cells.

A similar report, also from the UK, implicated French soft cheese as the vehicle of infection (Bannister, 1987). The cheese manufacturing process included pasteurisation but produced "heavy growth" of *L. monocytogenes* of a phage type indistinguishable from that isolated from the patient. Again the case was immunocompetent and not in any "at risk" group. (Table 11 England 1986).

A single case description concerning a 66 year-old man with predisposing conditions becoming infected by *L. monocytogenes* after consumption of soft cheese (unknown whether raw or pasteurised) has been reported in Canada (Farber *et al*., 1990). Here serotyping and multi locus enzyme electrophoresis were used to demonstrate the link between case and cheese isolates (Table 12 Canada 1989).

Though not listed in Table 12, a similar report originated from Belgium and concerned listeriosis in a 73 year-old man with predisposing conditions who had consumed Camembert cheese whilst on a visit to France (Gilot *et al*., 1997). Isolates from the case and remaining Camembert cheese in the case's refrigerator were indistinguishable by a suite of typing techniques. It is not known from the paper whether the cheese was made from raw or pasteurised milk. *Listeria monocytogenes* could only be isolated from the cheese after enrichment, indicating that it was present in low numbers.

**Table 12: Sporadic cases of foodborne human listeriosis**

| Country | Year | Patient died | Food implicated | Level of *L.m.*/g |
|---|---|---|---|---|
| Belgium | 1989 | No | Ice cream* | $10^3$ - $10^6$ (P) |
| **Canada** | **1989** | **No** | **Soft cheese** | |
| Canada | 1989 | Yes | Alfalfa tablets | |
| Denmark | 1989 | NK | Smoked cod roe | |
| **England** | **1986** | **No** | **Soft cheese** | **'High' (P)** |
| **England** | **1988** | **No** | **Soft cheese** | **$10^7$ (P)** |
| England | 1988 | Yes | Cooked chicken | |
| England | 1988 | Yes | Rennet | |
| Finland | 1989 | No | Salted mushrooms | $10^6$ (P) |
| Italy | 1989 | NK | Sausage | $10^6$ (P) |
| Italy | 1989 | No | Fish | |
| Italy | 1994 | NK | Pickled olives | |
| Sweden | 1993 | No | Mettwurst | |
| USA | 1985 | No | Turkey frankfurters | $10^3$ (P) |
| USA | 1987 | NK | Raw milk | |
| USA | 1989 | No | Sausage | |

NK = Not known

P = Food from patients home, usually opened.

* This is often cited as a case of listeriosis resulting from the consumption of ice cream. However, the original report indicates that crème fraiche mixed in with the ice cream introduced the pathogen.

Table 13 summarises details of outbreaks of listeriosis in which soft cheese was identified as the cause.

**Table 13: Overseas outbreaks of listeriosis where soft cheese was the implicated vehicle**

| Country | Year | Milk type | No. Cases | Cheese Type | Odds ratio | Reference |
|---|---|---|---|---|---|---|
| Canada | 2002 | Sub-pasteurisation temperature treatment. | 17 (3 neonates and 14 adults) | Soft and semi-hard, moistures not given (all 4 types produced were contaminated) | NS | Gaulin *et al.* 2003 |
| France | Between April 2 and May 16 1995 | Raw | 20 ( 2 spontaneous abortions, 4 premature births, 2 stillbirths) | Brie de Meaux soft cheese | 7.0 (CI 1.1-56.2) | Goulet *et al*. (1995) |
| Sweden | 2001 | Raw milk | 48 (febrile gastroenteritis) | On-farm cow, goat and blended milk fresh cheeses from one farm. | Cow's milk cheese 2.23 (CI 1.49-3.34), unknown cheese type 2.23 (CI 1.49-3.34) | Carrique-Mas *et al*. (2003) |
| Switzerland | 1983-1987 | Unknown | 122 cases, 57 in non-pregnant adults and 65cases in newborn infants and pregnant women. | Vacherin Mont D'or, eaten in winter months only. Only observed during the winter months. | 8.0 (CI 2.8-22.6) | Büla *et al*. (1995)<br><br>Codex (2002:28) |
| | May 2005 | Raw | 10 cases at present; 2 elderly fatalities, 2 miscarriages, 6 others hospitalised | Canton Neuenburg's Tomme soft cheese | Not stated | ProMED mail (2005) |

| Country | Year | Milk type | No. Cases | Cheese Type | Odds ratio | Reference |
|---|---|---|---|---|---|---|
| USA | 1985 | Pasteurised milk contaminated with raw milk post processing. | 142 (There were 10 neonatal deaths, 20 stillbirths and 18 non-pregnant adult deaths, total 48 deaths). 85 cases were associated with cheese consumption * | Mexican style (queso fresco, cotija). | 5.5 (CI 1.2-24.8) | Linnan *et al*. (1988); CDC 1985 |
| USA | 2000-2001 | Raw milk cheese unlabelled | 12 | Mexican style (queso fresco) | 17.5 (CI 2.0-152.5) | CDC (2001) |

CI= 95% confidence intervals. NS = Not stated. * Calculated from data in the paper, the other cases were assumed to be "background" sporadic cases.

In the Canadian outbreak, the factory produced cheese from milk of its own cows. Vat milk was negative for the organism. Renovation works to the exterior of the factory led to high levels of mud and construction workers frequently entered the factory. Twelve soil samples from the surrounding factory environment were positive for the organism. Surface samples internally were all negative. The isolates recovered from the cheese were identical to the isolates in the clinical cases. The public health authorities in Quebec observed from 17 cases, the isolates shared the same pulsotype from PGFE tests (strain 85); the 17 cases included three neonates. Thirteen of the 14 adult cases and all three mothers were surveyed. One of the mothers and 8 of the adult cases remembered eating cheese from the factory (9/16: 56%). Eleven of the 17 cases required hospitalisation. From the paper (Gaulin, 2003), the milk was heated treated but the cheese is referred to as raw milk cheese which implies thermisation of the milk. Fifty six cheese packages from 16 different batches were sampled from the maturation room. All cheese samples were positive for *L. monocytogenes*, pulsotype 85. A voluntary recall, suspension of operations and disinfection of equipment and environment in the factory followed. The paper does not enumerate the level of contamination. The authors discuss memory bias in the investigation, around 40% could not remember consuming the suspected vehicle of transmission, in this case, cheese. The authors suggest that cross contamination of foods in the refrigerator should not be excluded and cite research carried out by Pinner *et al*., (1992) see section 8.1.2.

In the Swedish outbreak involving raw milk cheese, *L. monocytogenes* was detected in foods at numbers in some cases exceeding $10^6$/g, this occurred after a period of refrigerated storage. Isolates from stools and cheese samples were indistinguishable by typing. The same cheese samples yielded high levels of *Staphylococcus aureus* and there was evidence of STEC contamination, but the symptoms experienced by the cases were consistent with febrile gastroenteritis. Further work indicated that isolates from the environment of the farm, including the counter across which cheese was sold, were of the same PFGE type (Danielsson-Tham *et al*., 2004).

In the Swiss outbreak, *L. monocytogenes* was detected in retail cheese at $10^4$-$10^6$/g (Codex, 2002:28)

In the 1985 USA outbreak, purportedly pasteurised milk (showing excessive levels of phosphatase) was used to manufacture the Mexican style fresh cheese. The pasteuriser was found to be in good working order. But investigators found documented deliveries and processing of 10% more raw milk than could have been properly heat treated. This means that either the pasteuriser inadequately pasteurised the milk or that raw milk was added to pasteurised milk post processing. The cheese was kept in cold storage for several weeks, but this same style cheese in Mexico is normally eaten fresh within 1 to 2 days. The seven month period over which the cases occurred implies continuous addition of contaminated raw ingredient such as milk from a listeriotic dairy herd or perpetual plant environmental contamination. The incubation period ranged from 1 to 91 days (median 35 days), the serotype involved was 4b. The *L. monocytogenes* contamination rate in cheese from retailers was $10^3$-$10^4$/g (Codex, 2002; Linnan *et al*., 1988).

In the outbreak in the States during October 2000 to January 2001, milk from each cow was tested and was negative for *L. monocytogenes*. Investigators concluded that environmental contamination was the most likely source. This outbreak resulted in North Carolina health authorities banning dairy farms from selling raw milk to non-commercial processors and alerting store owners that selling unregulated dairy products was an offence. Listeriosis was also made a notifiable disease and educational messages were reinforced to pregnant women about eating unpasteurised fresh cheese. The paper did not give the contamination rate of the cheese suspected of causing the outbreak (CDC, 2001).

### 6.2.3 Case-control studies

In a US study of sporadic listeriosis cases, cases were more likely than controls to have eaten soft cheese (OR 2.6, CI 1.4-4.8) (Schuchat *et al*., 1992). It was estimated that 15% of sporadic cases could be attributed to the consumption of Mexican style and Feta cheeses. Although all commercial soft cheeses produced in the USA must be made from pasteurised milk and imports are under similar restrictions, Mexican style soft cheese has been known to be made non-commercially with unpasteurised milk. In certain ethnic groups such as Hispanic populations, it was considered likely that the contribution soft cheese consumption makes to cases of listeriosis would be much greater than for the general population.

A nationwide study of risk factors for sporadic listeriosis in France (de Valk *et al.*, 1998) examined 225 cases during 1997. Cases were more likely to have eaten soft cheese (OR 2.3; 95% CI 1.2-4.6), and 49% of sporadic disease could be attributed to eating this type of cheese. It was not clear from the paper whether the soft cheese consumed by cases was made with raw or pasteurised milk. The authors concluded that soft cheese may account for a substantial proportion of sporadic listeriosis.

### 6.2.4 Risk assessments

A number of risk assessments have now been published concerning *L. monocytogenes.* The United State's joint FDA/FSIS risk assessment was published in September 2003. A further risk assessment by the FAO/WHO (Codex 2002) is in draft form and can be found at; http://www.who.int/foodsafety/micro/jemra/assessment/listeria/en/ under the related documents link.

After the most recent round of revisions, the FAO/WHO (Codex 2002) model has combined aspects of the FDA/FSIS one and almost merged the two. However, since the latest version of the Codex 2002 assessment is still in draft form, only the FDA/FSIS assessment will be discussed here.

The FDA/FSIS Risk Assessment published in September 2003 can be found at;
http://www.foodsafety.gov/~dms/lmr2-toc.html

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 all over the world were used to calculate prevalences in food). We might assume that the hazard characterisation (essentially dose response) would be the same in New Zealand as North America, but the derived risk characterisation will be different because of the different exposure assessments. The large proportion of the North American population of Hispanic origin presumably results in a high level of exposure for the population to *Listeria* in Mexican-style soft cheeses.

The relative risks predicted for the various ready-to-eat food categories in the FDA/FSIS risk assessment are given in Table 14, for various at-risk groups, and also as an overall ranking. One food, frankfurters, may or may not be reheated prior to consumption so is considered as two separate food categories. It is recognised that additional foods or cross-contamination may contribute further cases. Note that the rankings in this table have changed from those given in the draft risk assessment (and quoted in earlier Risk Profiles).

**Table 14: Predicted relative risk rankings for listeriosis based on the North American sub-population using median estimates on a per serving basis.**

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

[a] Food categories are grouped by type of food but are not in any particular order.
[b] A ranking of 1 indicates the food category with the greatest predicted relative risk per serving of causing listeriosis and a ranking of 23 indicates the lowest predicted relative risk of causing listeriosis.
[c] Ranks with the same letter are not significantly different based on the Bonferroni Multiple Comparison Test (alpha = 0.05).

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

The relative risk rankings from the above table along with the corresponding risk estimates are summarised in Table 15. This information is given in terms of predicted number of cases per serving and per annum. The per serving value is the relative risk faced by an individual when a single serving is consumed. This inherent risk is associated with manufacture,

distribution, marketing and use and reflects the degree of control achieved over *L. monocytogenes*. Factors that influence this value are;

- Frequency and extent of contamination,
- Ability of food category to support *L. monocytogenes* growth,
- Duration and temperature of refrigerated storage, and
- Size of serving.

The 'per annum' value is the predicted number of fatal infections per year in the US for each food category. This is influenced by the number of servings of the food category consumed. This second value is derived from the first 'per serving' value, so there is greater uncertainty with these values.

**Table 15: Relative risk ranking and predicted median cases of listeriosis for the total United States population on a per serving and per annum basis**

| Relative Risk Ranking | Predicted Median Cases of Listeriosis for 23 Food Categories | | | | | |
|---|---|---|---|---|---|---|
| | Per Serving Basis[a] | | | Per Annum Basis[b] | | |
| | Risk level | Food | Cases | Risk level | Food | Cases |
| 1 | **High** | Deli Meats | $7.7x10^{-8}$ | **Very High** | Deli Meats | 1598.7 |
| 2 | **High** | Frankfurters, not reheated | $6.5x10^{-8}$ | **High** | Pasteurized Fluid Milk | 90.8 |
| 3 | **High** | Pâté and Meat Spreads | $3.2x10^{-8}$ | **High** | High Fat and Other Dairy Products | 56.4 |
| 4 | **High** | Unpasteurised Fluid Milk | $7.1x10^{-9}$ | **High** | Frankfurters, not reheated | 30.5 |
| 5 | **High** | Smoked Seafood | $6.2x10^{-9}$ | **Moderate** | Soft Unripened Cheese | 7.7 |
| 6 | **High** | Cooked Ready-to-Eat Crustaceans | $5.1x10^{-9}$ | **Moderate** | Pâté and Meat Spreads | 3.8 |
| 7 | **Moderate** | High Fat and Other Dairy Products | $2.7x10^{-9}$ | **Moderate** | Unpasteurised Fluid Milk | 3.1 |
| 8 | **Moderate** | Soft Unripened Cheese | $1.8x10^{-9}$ | **Moderate** | Cooked Ready-to-Eat Crustaceans | 2.8 |
| 9 | **Moderate** | Pasteurized Fluid Milk | $1.0x10^{-9}$ | **Moderate** | Smoked Seafood | 1.3 |
| 10 | **Low** | Fresh Soft Cheese | $1.7x10^{-10}$ | **Low** | Fruits | 0.9 |
| 11 | **Low** | Frankfurters, reheated | $6.3x10^{-11}$ | **Low** | Frankfurters, reheated | 0.4 |
| 12 | **Low** | Preserved Fish | $2.3x10^{-11}$ | **Low** | Vegetables | 0.2 |
| 13 | **Low** | Raw Seafood | $2.0x10^{-11}$ | **Low** | Dry/Semi-dry Fermented Sausages | <0.1 |
| 14 | **Low** | Fruits | $1.9x10^{-11}$ | **Low** | Fresh Soft Cheese | <0.1 |
| 15 | **Low** | Dry/Semi-dry | $1.7x10^{-11}$ | **Low** | Semi-Soft Cheese | <0.1 |

| Relative Risk Ranking | Predicted Median Cases of Listeriosis for 23 Food Categories | | | | | |
|---|---|---|---|---|---|---|
| | Per Serving Basis[a] | | | Per Annum Basis[b] | | |
| | Risk level | Food | Cases | Risk level | Food | Cases |
| | | Fermented Sausages | | | | |
| 16 | **Low** | Semi-soft Cheese | $6.5 \times 10^{-12}$ | **Low** | Soft Ripened Cheese | <0.1 |
| 17 | **Low** | Soft Ripened Cheese | $5.1 \times 10^{-12}$ | **Low** | Deli-type Salads | <0.1 |
| 18 | **Low** | Vegetables | $2.8 \times 10^{-12}$ | **Low** | Raw Seafood | <0.1 |
| 19 | **Low** | Deli-type Salads | $5.6 \times 10^{-13}$ | **Low** | Preserved Fish | <0.1 |
| 20 | **Low** | Ice Cream and Other | $4.9 \times 10^{-14}$ | **Low** | Ice Cream and Other | <0.1 |
| | **Low** | Frozen Dairy Products | | **Low** | Frozen Dairy Products | |
| 21 | **Low** | Processed Cheese | $4.2 \times 10^{-14}$ | **Low** | Processed Cheese | <0.1 |
| 22 | **Low** | Cultured Milk Products | $3.2 \times 10^{-14}$ | **Low** | Cultured Milk Products | <0.1 |
| 23 | **Low** | Hard Cheese | $4.5 \times 10^{-15}$ | **Low** | Hard Cheese | <0.1 |

[a] Food categories were classified as high risk (>5 cases per billion servings), moderate risk (<5 but >1 case per billion servings), and low risk (<1 case per billion servings).
[b] Food categories were classified as very high risk (>100 cases per annum), high risk (>10 to 100 cases per annum), moderate risk (>1 to 10 cases per annum), and low risk (<1 cases per annum).

Source: FDA/FSIS (September 2003) (http://www.cfsan.fda.gov/~dms/lmr2-ex.html)

The categories relevant to this Risk Profile are: soft unripened cheese and soft ripened cheese. These received moderate and low risk rankings respectively, within the context of the US food supply. Note that the "fresh soft cheese" category in the FDA/FSIS risk assessment refers to queso style cheeses, popular with the Hispanic community, which are unlikely to be widely consumed in New Zealand.

A quantitative risk assessment has been published which is focused entirely on soft cheeses made from raw milk in France (Bemrah *et al*., 1998). The probability of milk contamination with *L. monocytogenes* (due to environmental contamination and/or mastitis) was estimated to be 67% with a concentration ranging from 0 to 33 cfu/ml. The probability of a resident of France consuming contaminated raw milk cheese was estimated at 65.3%, but the probabilities of consuming cheese containing greater than $10^2$, $10^3$ and $5 \times 10^3$ cfu *L. monocytogenes* were 41%, 8.3% and 0.08% respectively. An estimate of risk, based on the consumption of 50 portions of 31g per annum ranged from $1.97 \times 10^{-9}$ to $6.4 \times 10^{-8}$ in the low risk population subgroup, to between $1.04 \times 10^{-6}$ and $7.19 \times 10^{-5}$ in the high risk subpopulation. In a population of 50 million people this equates to 34 to 90 (mean 57) cases and 1 to 23 (mean 21) deaths per annum in the high risk subpopulation, and 0 to 4 cases (0 to 3 deaths) in the low risk subpopulation.

By eliminating the effects of mastitis and the frequency of environmental milk contamination from the model, the exposure to *L. monocytogenes* was much decreased. The average

number of expected cases reduced by a factor of 5 (e.g. 99$^{th}$ percentile was around 100 *L. monocytogenes*/g when mastitis was modelled, and around 20/g when it was not included). The authors discuss at length the assumptions made and the fact that the results need to be treated with care because of these assumptions.

Again in France, a risk assessment of listeriosis and the consumption of raw milk Normandy Camembert and raw milk Brie of Meaux has been carried out (Sanaa *et al*., 2004). Based on data acquired on the two cheeses between 2000 and 2001, the estimated prevalence of *L. monocytogenes* in raw milk was on average 0.8 cells/litre in Normandy and 0.3 cells/litre in Meaux. A Monte Carlo simulation was used to model the time-temperature history of the milk/cheese from farm to table. In the simulation, servings containing no cells were Brie 88%, and Camembert 82%. The 99$^{th}$ percentile of cell numbers in 27g servings were Brie 131 cells and Camembert 77 cells at time of consumption (corresponding to 3 and 5 cells per gramme respectively). With 17 million servings of Brie of Meaux and 480 million servings of Normandy Camembert per year, the expected number of severe listeriosis cases per annum were very low; calculated at $\leq 10^{-3}$ and $\leq 2.5 \times 10^{-3}$ respectively.

## 6.3 Qualitative Estimate of Risk

The information summarised above leads to the conclusion that the transmission of *L. monocytogenes* by soft cheese has the potential to contribute to a proportion of invasive listeriosis cases, but that the current risk of infection via this transmission route in New Zealand for the general population is low (although the risk will be greater for susceptible populations). Evidence for this conclusion comes from:

- food surveys indicating a very low prevalence of *L. monocytogenes* in soft cheeses in New Zealand compared to prevalences found overseas,
- the low level of consumption of these foods in terms of numbers of servings and mean level of consumption,
- the lack of any New Zealand outbreak of infection by *L. monocytogenes* where soft cheese was identified as the vehicle, and,
- the lack of evidence identifying soft cheese as a transmission vehicle in episodes reported from the Investigation of Foodborne Illness Project.

However, if it is present, *L. monocytogenes* is able to survive and grow in (or on) soft cheese, under normal storage conditions (see section 3.2.8). Evidence from overseas confirms that soft cheese is occasionally contaminated with *L. monocytogenes*, and this food has been identified as the cause of both outbreaks and sporadic cases of listeriosis.

## 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 soft cheese compared to other food groups, any 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 |
|---|---|---|---|---|
| *L. monocytogenes* in soft cheese | 1 (>5% serious outcomes) | 4 (<1 per 100,000) | High (control essential) | Incidents attract adverse media attention |

# 7 RISK MANAGEMENT INFORMATION

Cheese production is highly regulated in New Zealand and other developed countries, as is dairy production and processing in general. The legislative and regulatory situation in New Zealand is currently in review, as several long standing documents are brought up to date. This section reviews the current status of controls relevant to pasteurisation and other heat treatments required for milk, and requirements specific for *L. monocytogenes* in cheese, both in New Zealand and overseas.

## 7.1 Relevant Food Controls: International

With no current international agreement on what is an 'acceptable level' of *L. monocytogenes* in foods, together with the different sample methodologies and sampling plans, the relevant food controls in and between countries can become very complex. The draft Codex guidelines for control of *L. monocytogenes* (Codex, 2002, Section 5.2) state that although limits are a responsibility of individual governments, a 99% reduction in the current baseline will be obtained by setting a Food Safety Objective (FSO) at <100 *L. monocytogenes* $g^{-1}$ of food at point of consumption. This figure may be higher or lower in the performance criteria of the food dependent on listericidal treatments, the characteristics of the food, storage temperatures and shelf life. For internationally traded food, at port of entry sampling, those foods which support growth may need lower figures applied so that >100 $g^{-1}$ at consumption does not occur.

The FSO concept places public health risk into a definable goal to provide an appropriate level of protection.

Several countries have adopted a zero-tolerance policy (i.e. absence in 25 g). These countries include New Zealand and Australia, USA, Austria and Italy. However some countries believe this is too overly cautious and using HACCP principles, use risk assessment to establish maximum limits. The result is a range of limits which can vary between 10 cfu $g^{-1}$ to 100 cfu $g^{-1}$ or 1000 cfu $g^{-1}$ depending on the product, risk category and time of consumption. Some countries such as Canada and Denmark, adopt a mixture of zero-tolerance for some foods and tolerance levels for others. The International Commission for Microbiological Specifications for Foods (ICMSF) have stated that microbiological testing of food must be viewed as a tool to verify that HACCP plans are working and are insufficient by themselves to ensure food safety. The ICMSF therefore advocate the following;

- in-pack, heat-treated products – no testing is necessary (documentation for the heat-treatment process),
- raw products and/or products which are to be heat-treated before consumption – no testing is necessary,
- ready-to-eat products, unable to support growth of *L. monocytogenes* – 10 samples should be taken and the lot should be rejected if any sample contains > 100 *L. monocytogenes* $g^{-1,}$ and
- ready-to-eat products, able to support growth of *L. monocytogenes* – 20 samples should be taken and the lot rejected if any sample contains > 100 *L. monocytogenes* $g^{-1}$.

Source: http://www.fao.org/DOCREP/003/X3018E/X3018E06.HTM

The ICMSF explanatory note on the establishment of sampling plans for microbiological safety criteria for foods in international trade can be found on page 34-37 of Codex (2002).

## 7.2 Legislative Environment in New Zealand with Respect to *L. monocytogenes* in Soft Cheese

Codex has produced a Code of Hygienic Practice for Milk and Milk Products (Codex, 2004). The Code covers products in international trade and can serve as a legislative basis in some countries. The overall principles are

- Control measures should achieve appropriate level of public health protection,
- Good hygienic practices should be applied throughout the food chain,
- Hygienic practices implemented via HACCP, and
- Control measures should be validated as effective.

In Annex 1 of the Code, additional provisions are given for the production of milk used for raw milk products.

Australia and New Zealand are members of the World Trade Organisation (WTO) and both countries are signatories to the SPS (The Agreement on the Application of Sanitary and Phytosanitary measures). FSANZ is the organisation that ensures food standards are consistent with the obligations of both countries as members. While there are no consistent international standards on the use of raw milk for cheese-making, international trade cannot be restricted if it can be demonstrated that products have an equivalent and acceptable level of safety. Therefore each application made from international trading partners is considered on a case-by-case assessment by FSANZ.

New Zealand legislation relating to the safety of foods, including soft cheeses either specifies the production requirements, or the finished food requirements.

The production requirements are legislated for dairy material and dairy product under the Animal Products Act (superseding the Dairy Industry Act 1952 on 1st June 2005). Associated NZFSA Dairy Specifications and Approved Criteria can be found at the following website http://www.nzfsa.govt.nz/dairy/publications/specifications/index.htm.

Regulations made under the Animal Products Act together with the Specifications and Approved Criteria represent the bulk of the requirements for dairy producers, and provide detailed information for operators, such as the hygiene outcomes they must achieve.

The finished food requirements for sale of product within Australia and New Zealand are legislated for under the Food Act 1981, New Zealand (Milk and Milk Products Processing) Food Standards 2002, the Australia New Zealand Food Standards Code and associated guidelines. These standards focus largely on conditions for pasteurisation, and microbiological limits to be achieved in products for the domestic market.

### 7.2.1 NZFSA Dairy Standards

The NZFSA requirements for Dairy Product Safety specify minimum product safety outcomes for all dairy products. Criteria are given by which a dairy Risk Management Programme (RMP) holder may be judged to satisfactorily achieve the outcomes described in the Dairy Processing Specification (particularly "All dairy products must be safe and wholesome"). One of the criteria is a Product Safety Limit (PSL) for *L. monocytogenes* of ND (not detected)/25g. The following comment is made with respect to this organism: "*Listeria monocytogenes*: A figure of 100/g has been proposed by the Joint FAO/WHO Food Standards Programme, Codex (2002) Committee on Food Hygiene in the "Draft Guidelines for the Control of *Listeria monocytogenes* in Foods" and is obtaining increasingly wide acceptance. In the future, it may be appropriate to adopt a PSL of 100/g in circumstances where it can be shown that growth is extremely unlikely to occur during the life of the product. However, before this occurs, NZFSA and the dairy industry will need to be convinced that the 100/g figure has become accepted by reputable food safety authorities worldwide."

Previously Dairy Standard D109 Dairy Product Conformance was used to specify sampling and testing requirements for dairy products. This Standard has been superseded by the requirements for Dairy HACCP Plans that specify how HACCP principles and guidelines are used to develop HACCP plans that are components of RMPs. This recognises that a RMP holder can meet the required outcomes of a RMP in a variety of ways, including the outcome of ensuring product compliance with the requirements for Dairy Product Safety. This provides the potential for products to be exempt from sampling and testing for pathogens on the basis of product type or production process. The requirements state: "Routine testing of product safety attributes may not be required where a HACCP plan can demonstrate an equivalent level of confidence in meeting these product safety outcomes".

The requirements for Dairy Product Safety apply to all dairy products that are delivered to the retail distribution chain within New Zealand or are exported. Exporters will also have to comply with the requirements of the country to which the product is exported.

Currently, pasteurisation conditions, checking and validation are in a transitional period with the MRD Standard 3 and 4 being fully superseded on 1 June 2005 with the new requirements for Dairy Heat Treatments. The new requirements for Dairy Heat treatments were introduced on 14 April 2003 to allow milk processors a transitional period. Most milk-produce processing in dairy plants would have switched or will be switching to this new standard pending an equipment upgrade. The new pasteurisation conditions in the new requirements will continue to control *L. monocytogenes* effectively.

Dairy requirements administered by the NZFSA and associated Codes of Practice include traceback and disposal requirements in the event of *L. monocytogenes* detection or other non-conformances in product or in the processing environment. These requirements include:

- Isolation of all positive and suspect product,
- Testing of product at increased frequency for the relevant pathogen,
- Traceback exercise including swabbing,
- Clean-up of the plant and associated areas,
- Reporting by the manufacturer to their Recognised Agency auditor who reports the information on to NZFSA,

- Following source identification and corrective action, the manufacturer submits a product disposal request to the NZFSA. Depending on the non conformance, disposal options may include; sale as a dairy product, change of purpose, sub-lotting (separation of conforming and non-conforming product), relabelling, use as dairy raw materials (reprocessing), use as animal feed, sale for non-food and non-feed uses, or destruction.

The above requirements apply to manufacturers operating a Risk Management Programme. Other manufacturers operating under Food Safety Programmes would be required to inform and liaise with their local Public Health Unit in the event of a positive result and recall.

### 7.2.2 Animal Products Act

Risk Management Programmes (RMPs) are part of the emerging food assurance system in New Zealand. They form part of the Animal Products Act (APA) 1999. These have been integrated with Product Safety Programmes (PSPs) required by the Dairy Industry Act 1952. http://www.nzfsa.govt.nz/dairy/subject/animal-products-act/index.htm.

More information on the Animal Products Act can be found at the NZFSA website:

http://www.nzfsa.govt.nz/animalproducts/legislation/aparmp.htm

The Animal Products Act 1999 has been amended with specific dairy regulations.

Cheese manufacturers producing for export will be required to comply with the APA and operate under a registered RMP. Manufacturers producing for the "domestic" market (New Zealand and Australia) will have another option; complying with an approved Food Safety Programme, developed from an approved Code of Practice. The option for domestic dairy manufacturers to operate under the Food Hygiene Regulations 1974 will be removed one year after commencement of the Dairy Animal Products legislation.

### 7.2.3 The Approved Code of Practice and the NZSCA

A Code of Practice for Cheese Production has been written by the New Zealand Specialist Cheesemakers Association Inc. (NZSCA, 2002). The full title of the Code is the "Interim Code of Practice for the development of a Food Safety Programme (Food Act 1981) or Product Safety Programme (Dairy Industry Act 1952) for Specialist Cheeses". The NZFSA approved the Code under Regulation 59 of the Dairy Industry Regulations 1990 in Circular no. 80 dated 6th April 2004. Copies of the Interim code are available to members of the New Zealand Specialist Cheesemakers Association Inc.

This Circular revokes a previous Circular (no. 8) and withdraws approval of the 'Generic Product Safety Programme for Small-scale Cheese Manufacturers 1992'.

Specialist cheesemakers had until 1 August 2004 to implement a Food Safety Programme under the Food Act 1981 or a Product Safety Programme under the Dairy Industry Act 1952.

Businesses opting to develop a Food Safety Programme (FSP) ('for domestic markets; New Zealand and Australia) must base their FSP on the Interim Code of Practice.

Businesses exporting product outside of the New Zealand/Australia market must implement a Risk Management Programme (RMP) based on the Interim Code of Practice and some additional requirements, see bullet points below.

A Risk Management Programme will be registered if it conforms to the code, and in addition;

- The NZFSA requirements for Risk Management Programme Reporting Requirements, Dairy Heat Treatments and Independent Verification Programme,
- Environmental Pathogen Surveillance Programmes, and
- Any Importing Country Requirements (ICRs).

At the beginning of 2005, there were 34 members of the New Zealand Specialist Cheesemakers Association Inc. (NZSCA). It is estimated that members of the Association represent over 90% of the total commercial cheese making operations in New Zealand (Dianne Kenderdine, Secretary NZSCA, personal communication, February 2005).

### 7.2.4 Food Act 1981 and New Zealand (Milk and Milk Products Processing) Food Standards 2002

All food for sale in New Zealand must comply with the Food Act 1981. The Act allows for the restricted sale of raw milk (section 11A; at the 'farm gate' and not exceeding 5 litres at a time). On 20th December 2002, under section 11C of the Food Act 1981, the New Zealand (Milk and Milk Products Processing) Food Standards 2002 were introduced. This Standard updated and consolidated previous dairy regulations and sets the minimum legal requirement for the quality and safety of milk and milk products. There are currently three methods legislated in New Zealand which cover milk processing in relation to cheese-making. These are listed under Clause 4 of the Standard and are as follows;

- Pasteurisation,
- Cheese treatment, and
- Methods accepted by FSANZ as being equivalent to the safety levels achieved by pasteurisation controls e.g. Ordinance on Quality Assurance in the Dairy Industry, Swiss Federal Council 18th October 1995.

The term "pasteurisation" is defined in the Standard under Clause 3 (c) and stipulates three methods;

- Holding method (63-66º for not less than 30 minutes),
- High-temperature short-time method (>72ºC for not less than 15 seconds), and
- Any other heat treatment method that is as effective in terms of bacterial reduction as the methods above.

[Raw milk is defined (Codex 1999c) as milk that has not been heated beyond 40°C)].

Clause 5 of the New Zealand (Milk and Milk Products Processing) Food Standard 2002 gives a table listing permitted methods of dairy product processing. This states that cheese must be pasteurised, unless the cheese has a moisture content <39% and a pH level <5.6 with no increase in pH upon ripening. For such cheeses, permitted processing methods are pasteurisation or cheese treatment (defined elsewhere in the Standard).

Therefore soft cheese is not permitted to be made from unpasteurised milk in New Zealand because it would contain more than 39% moisture.

### 7.2.5 FSANZ

Pasteurisation requirements for cheese in Australia are set by the Australia New Zealand Food Standards Code Standard 1.6.2, which does not apply in New Zealand. Part 2 of this Standard provides heat treatments for cheese products such that any soft cheese (>50% moisture) must be made from milk products that have been pasteurised (no less than 72ºC for a period of no less than 15 seconds).

There is no FSANZ approval permitting soft unpasteurised milk cheeses. This follows the FSANZ rejection of the Australian Specialist Cheesemakers Association application A270 "Cheeses made from fresh milk that has not been pasteurised or subjected to another heat treatment". This was with a view to produce hard dry and soft moist cheeses from raw milk.

Permission for unpasteurised milk cheeses may be granted by FSANZ following a case by case assessment, guided by a general process for determining the equivalence of food safety measures (see:
http://www.foodstandards.gov.au/mediareleasespublications/publications/draftproposedguideli1507.cfm).

To be approved, the unpasteurised milk cheese must undergo a production process that has been demonstrated to provide an equivalent safety level to that achieved by heat treatments based on microbiological parameters. The general consensus is that a process is considered equivalent where it achieves at least a 5-log reduction of pathogens, a 5-log reduction figure is therefore used as a benchmark in considering equivalency. Currently hard and very hard Swiss cheeses with a very long storage period (at least 90 days up to 360 days), specifically Emmental, Gruyère and Sbrinz (ANZFA, 1998), and extra hard grating cheese (Parmesan style) (FSANZ 2002) are the only raw milk cheeses permitted for import into New Zealand.

The French government has submitted an application to FSANZ to import unpasteurised Roquefort (a semi-soft cheese made from raw sheep's milk). A draft assessment report (Application A499) was issued on 23rd March 2005 http://www.foodstandards.gov.au/standardsdevelopment/applications/applicationa499toper2374.cfm that recommended permitting the sale of Roquefort cheese. FSANZ is currently seeking public comment (until 4th May 2005).

### 7.2.6 Controls on *L. monocytogenes* in cheese in New Zealand

On 20 December 2002, the New Zealand Food Regulations 1984 were revoked, replaced or retained, principally to make way for the joint Food Standards Australia New Zealand (FSANZ) Food Standards Code. Any regulations falling outside of the joint system (not covered by 'the code') are contained in the Food (Safety) Regulations 2002, (applicable only in New Zealand).

Under Chapter 1 of the Food Standards Code, Standard 1.6.1 (see website:http://www.foodstandards.gov.au/_srcfiles/Standard_1_6_1_Micro_v70.doc), "Microbiological Limits for Food" lists the maximum permissible levels for foodborne micro-organisms which pose a risk to human health. It is unlawful to exceed these limits.

Sample lots or consignments of food that do not fall within these limits are seen as posing a risk to public health and should be withdrawn. An extract relating only to cheese is presented in Table 16. Failure to comply would be where the number of defective sample units is greater than c or where any of the sample units exceeds M. Effectively there is a 'zero tolerance' for *Listeria monocytogenes* as absence is required in a 25 g sample (made up of 5 units).

**Table 16: Microbiological limits in cheese, FSANZ Code, Standard 1.6.1**

| Food | Micro-organism | n | c | m | M |
|---|---|---|---|---|---|
| All cheese | *Escherichia coli*/g | 5 | 1 | 10 | $10^2$ |
| **Soft and semi-soft cheese (moisture content > 39%) with pH >5.0** | ***Listeria monocytogenes*/25 g**<br>*Salmonella*/25 g | **5**<br>5 | **0**<br>0 | **0**<br>0 | |
| **All raw milk cheese** (cheese made from milk not pasteurised or thermised) | ***Listeria monocytogenes*/25 g**<br>*Salmonella*/25 g | **5**<br>5 | **0**<br>0 | **0**<br>0 | |
| Raw milk unripened cheeses (moisture content > 50% with pH > 5.0) | *Campylobacter*/25 g | 5 | 0 | 0 | |

Source: FSANZ Food Standards Code 1.6.1 (2002) http://www.foodstandards.gov.au/foodstandardscode/

Under Chapter 2 – Food Product Standards, Part 2.5 of the Code itemises the Dairy Products, under which Standard 2.5.4 is Cheese. This defines cheese and processed cheese and sets its compositional requirements. Clause 4 of this Standard (Processing of milk and milk products in New Zealand) relates to Clause 7 (d) of the Milk Processing Standards 2002 mentioned above, whereby compliance with one means compliance with the other.

## 7.3 Relevant Food Controls: Overseas

### 7.3.1 USA: FDA Dairy Safety Initiatives and current legislation

The United States of America has a 'zero tolerance' policy for *L. monocytogenes* in ready-to-eat (RTE) foods, which includes soft cheeses. This means that RTE foods contaminated at a detectable level with the organism are deemed to be adulterated.

Following a number of outbreaks of listeriosis in the USA in the mid 1980s the FDA implemented the Dairy Safety Initiatives from 1st April 1986 to 30th September 1988 (Kozak *et al*., 1996). This involved the collection of both finished product and environmental samples for *Listeria* testing, as well as plant inspections. Because of funding limitations, environmental samples were collected only when a finished product tested positive. A total of 1370 inspections were carried out and 2.7% of the plants were manufacturing products positive for *Listeria* spp.

The result of this was the production of "Recommended Guidelines for Controlling Environmental Contamination in Dairy Plants". The focus of this document was preventing post-pasteurisation contamination by *L. monocytogenes*.

Further to the Joint Risk Assessment carried out by FDA/FSIS (2003), an update to the *Listeria* action plan in the USA was formulated in November 2003. The interim goal is to reduce *L. monocytogenes* caused illness by 50 percent by 2005. The new action plan can be found at the following FDA website ( http://www.cfsan.fda.gov/~dms/lmr2plan.html).

The six areas for action are;

1. Develop and revise guidance for processors that manufacture or prepare ready-to-eat foods and develop or revise guidance for retail and food service and institutional establishments.

2. Develop and deliver training and technical assistance for industry and food safety regulatory employees.

3. Enhance consumer and health care provider information and education efforts.

4. Review, redirect, and revise enforcement and regulatory strategies, including microbial product sampling.

5. Enhance disease surveillance and outbreak response.

6. Coordinate research activities to refine the Risk Assessment, enhance preventive controls, and support regulatory, enforcement, and educational activities.

The zero tolerance policy adopted in the 1980s makes no distinction between foods contaminated at high or low levels, contamination at a detectable level is enough to deem the food as unfit. This current regulatory approach has been challenged because it concentrates on further reducing prevalence of the organism in RTE foods and continues zero-tolerance for all RTE foods. Recently the Food and Drug Administration (FDA) announced (May 24 2004) that a petition had been filed by fifteen US food industry trade associations that requests that the agency establish a regulatory limit of 100 cfu per gram for *Listeria monocytogenes* in foods that do not support the growth of the micro-organism. The agency is requesting comment on the petition.

This microbial risk assessment approach is supported by Chen *et al* (2003). Since the organism can not be eliminated from the environment or from all food products despite extensive control measures, Chen *et al* argue that non-zero tolerance as an alternative strategy may have a greater impact in the level of risk reduction. The report concludes that foods containing low levels of *L. monocytogenes* (e.g., <100/g) pose very little risk; eliminating the higher concentrations can reduce the number of predicted cases by >99%. Therefore, directing limited resources to those foods in which *L. monocytogenes* is likely to be present and likely to grow to high levels rather than all RTE foods is put forward. Comparisons with countries that operate such a strategy (e.g. Canada and several European countries) show that rates of listeriosis are not noticeably different. This approach appears to be in line with that proposed by the ICMSF and Codex (2002: Food Safety Objective 5.2) for the standard in internationally traded foods, see section 7.1.

It has been estimated that a Food Safety Objective of <100 cfu/g of *L. monocytogenes* at point of consumption would provide a similar level of consumer protection to a standard which requires absence of *L. monocytogenes* in 25 or 50g (Szabo *et al*., 2003).

### 7.3.2 European Union

The EU Council Directive 92/46/EC (1992) provides microbiological standards for cheese in Chapter II of the Directive. For cheese, other than hard cheese, under compulsory criteria, *L. monocytogenes* must be absent in 25g (to consist of 5 specimens of 5g taken from different parts of the same product). These parameters are based on time of removal from the processing establishment and does not reflect the quality expected at point of sale or consumption. This lack of microbiological reference values has led to food being declared unfit for human consumption because of non-quantified contamination with *L. monocytogenes*, leading to controversy in member state's judicial system (see Lanarkshire Blue case 7.3.4) and in cases of intra-Community trade. An example of this is Germany, Netherlands and France, who have a tolerable level less than 100 cfu $g^{-1}$ at the point of consumption. Italy, like the USA, has a zero tolerance (absence of *L. monocytogenes* in 25g of food). Denmark, like Canada, has a tolerance of below 100 cfu $g^{-1}$ for some foods and a zero tolerance for others (especially foods that support growth and have extended shelf lives), refer to sections 7.3.7 and 7.3.8 respectively.

The European Commission set up a Scientific Committee on Veterinary measures relating to public health on *Listeria monocytogenes* (Anonymous 1999) to assess the risk to health from this organism in ready to eat foods. Its report, from 1999, can be found at the following website; http://europa.eu.int/comm/food/fs/sc/scv/out25_en.pdf

Seven recommendations came from the Scientific Committee report, the second and fifth recommendations were;
"(2) An objective must be to keep the concentration of *L. monocytogenes* in food below 100cfu/g and to reduce the fraction of foods with a concentration above 100 *L. monocytogenes* per gram significantly. This objective should be expressed as a Food Safety Objective. The effect of initiatives to this end must be evaluated through surveillance investigations of food, especially including quantitative investigations, as well as efficient monitoring of human listeriosis. *Note this is in line with the draft FAO/WHO Codex guidelines (Codex 2002:5.2) refer to section 7.1*
(5) Strategies for risk communication must be implemented. Apart from advice to the general public, special attention should be addressed to consumer groups at increased risk (i.e. young, old, pregnant, immunocompromised) which represent a considerable and growing section of the total population"(Anonymous 1999:27).

### 7.3.3 England and Wales

In the United Kingdom, the statute law; the Food Safety Act 1990 Sections 7, 8, and 14 provide the legal framework for dealing with the microbial quality of food. No cases have been taken under Section 7 where a person renders the food injurious to health. Section 8 (2)(b) *'unfit for human consumption'* or Section 8(2)(c) *'so contaminated that it would not be reasonable to expect it to be used for human consumption in that state'* are the two sections most commonly used. Section 14 '*any food which is not of the nature or substance or quality demanded by the purchaser*' is used where there is an issue of trading quality and is rarely used for bacterial contamination.

Regulations made under the Food Safety Act, namely the Dairy Products (Hygiene) Regulations 1995 interpret the EU Council Directive 92/46/EC into national law. See the following website for details;
http://www.hmso.gov.uk/si/si1995/Uksi_19951086_en_1.htm.

Schedule 3 relates to the requirements for raw milk and Schedule 5 sets out the requirements for raw milk, thermised milk, pasteurised milk and UHT milk. Schedule 6 contains the requirements for milk-based products, Part I of this Schedule contains the microbiological criteria upon removal from the processing establishment. In relation to *Listeria,* the Regulations stipulate;

| | ***Product*** | ***Type of Micro-organism*** | ***Standard (ml, g)*** |
|---|---|---|---|
| (i) | Cheese, other than hard cheese | *Listeria monocytogenes* | Absence in 25g where n = 5, c = 0 |

Guidelines have been issued by Public Health Laboratory Service (PHLS) for the microbiological quality of some ready-to-eat foods sampled at the point of sale (Gilbert *et al*. 2000). The guidelines have no legal standing in their own right. The purpose of the guidelines is to assist food examiners and EHOs to determine the bacteriological quality and indicate the level of contamination that is considered to represent a significant potential risk to health. This information can then be used to assist the enforcement officer in deciding which Section of the Food Safety Act 1990 should be used to initiate a prosecution.

The criteria for *Listeria* spp. has been modified since the 1992 & 1996 revised guidelines. The term *Listeria* spp. (total) is used so that it is fully inclusive of all *Listeria* species. The guidelines state that although *Listeria* spp. other than *Listeria monocytogenes* are rarely implicated in illness, they are indicators for the likely presence of *L. monocytogenes*.

The quantitative levels given under the 'unacceptable/potentially hazardous' column represent a potential hazard to those who eat such food. This means on the basis of current information, "it is unacceptable that ready-to-eat foods contain any serogroup of *L. monocytogenes* at levels at or above 100 cfu per gram. Some serotypes/phage types of *L. monocytogenes* may rarely be associated with human infection, but their presence represents an inadequate level of hygiene" (Gilbert *et al* 2000). The guidelines add that certain foods such as soft ripened cheese have a long shelf life under refrigeration and the presence of *L. monocytogenes* at any level may be of significance here due to its potential for growth during storage, this explains the 'Not detected in 25g for certain long shelf-life products under refrigeration' criteria for *L. monocytogenes.*

The guidelines for *Listeria* spp. (total) and *Listeria monocytogenes* are summarised in Table 17.

**Table 17: Guidelines for the microbiological quality of *Listeria* spp (total) and *Listeria monocytogenes* in foods at point of sale in England and Wales.**

| | Microbiological quality (cfu per gram) | | | |
|---|---|---|---|---|
| **Criterion** | **Satisfactory** | **Acceptable** | **Unsatisfactory** | **Unacceptable/ potentially hazardous** |
| *Listeria* spp. (total) | <20 | 20-<100 | ≥100 | N/a* |
| *Listeria monocytogenes* | <20** | 20-<100 | N/a[#] | ≥100 |

* It is noted that a prosecution based solely on high colony counts and/or indicator organisms (such as *Listeria* spp. (total) in the absence of other criteria of unacceptability is unlikely to be successful therefore quantitative levels in the 'unacceptable/potentially hazardous' column have been made non-applicable.
**Not detected in 25g for certain long shelf-life products under refrigeration.
[#] Not applicable as some quality standards require a zero level at the production stage of a food and $10^2$CFU/g at point of sale/consumption would represent a potential risk to health.
Source: (Gilbert *et al* 2000).

The guidelines note that for the aerobic colony count 30°C/48h, this may not apply to soft cheese as acceptability is based on appearance, smell, texture and the levels or absence of indicator organisms or pathogens. It is also noted that *Listeria* spp. (total) is listed under 'Indicator organisms' where on occasions "some strains may be pathogenic".

### 7.3.4 Scotland and the Lanark Blue cheese prosecution

An infamous case was taken under Section 8 of the Food Safety Act 1990 in Scotland regarding *Listeria* in Lanark Blue Cheese. Details about the case can be obtained from the following website; http://www.foodlaw.rdg.ac.uk/uk/cases.htm.

Following a survey of soft cheeses for microbial quality, concern was raised over the results from an unpasteurised ewes milk cheese manufactured by H J Errington & Co. in the district of Clydesdale. A condemnation order was sought in the courts to declare 44 batches of the cheese as unfit for human consumption based on the presence of *Listeria monocytogenes.* Following lengthy resampling and judicial processes, two main issues arose; (1) were the results upon which the Local Authority based its application correct? (2) were the actual levels of *Listeria monocytogenes* in the cheese such as to make the food unfit for human consumption?

The case was taken to the Sheriff of South Strathclyde, Dumfries and Galloway. The Sheriff concluded that there was sufficient doubt over the accuracy of the Local Authority's results and the question of whether the cheese was injurious to health, led to the question whether all strains of *Listeria monocytogenes* must be regarded as pathogenic. The Sheriff did not accept that there was sufficient evidence to prove that consuming the strains of *L. monocytogenes* present in the Lanark Blue cheese would cause harm. Epidemiological evidence also showed that although 63,000 portions of cheese must have been consumed, no illness had been reported. The Sheriff concluded that "the evidence does not support the claim that all strains of *Listeria monocytogenes* should be regarded as potentially dangerous – and hence likely to be injurious to health." The cheese producer was compensated for his economic losses as the

time taken for a judicial decision on the case meant that the 44 batches of cheese could not be sold.

### 7.3.5 The Specialist Cheesemakers Association in the U.K. and Ireland

The Specialist Cheesemakers Association was founded in February 1989 following an announcement by the Minister of Agriculture of his intention to ban the sale of unpasteurised cheese in the United Kingdom. A political lobby group with H.R.H The Prince of Wales as patron, it represents the interests of members to government and the media. The association defines itself in a market which demands flavour and character from cheese. This is derived usually from being handmade on a farm, on a small scale using traditional methods and often from unpasteurised milk. The Specialist Cheesemakers Code of Best Practice was produced by the association with assistance from various Government agencies. A practical document, it is intended to raise the quality of the cheeses produced by its members and is also referred to by enforcement officers during inspections. More information and the Code of Best Practice can be found on the association website (http://specialistcheesemakers.co.uk).

### 7.3.6 The Specialist Cheesemakers Association in Australia

The Australian Specialist Cheesemakers Association was established in 1994 and there are now over 70 specialist cheese manufacturers around Australia. In the year 1999/2000 Australia produced around 27,000 tonnes of specialty cheese – about 8% of total Australian cheese production. Total production is growing at about 3% annually, see website; http://www.food.vic.gov.au/CA256D3A001F9796/all/77AAF84743546639CA256DD5007AF20C?open

### 7.3.7 Denmark

Nørrung *et al.,* (1999) describe the control of *Listeria monocytogenes* in Denmark. The regulatory policy is based on HACCP and a health risk assessment approach. Ready-to-eat foods are categorised into six subsets with the following tolerances (Table 18).

**Table 18: Food groups and tolerances for *L. monocytogenes* in Denmark**

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

| Category | Food groups | No. of samples (*n*) | Absence in 25g (*c*) | *m* | *M* |
|---|---|---|---|---|---|
| V | Lightly preserved, not heat treated, stabilised against growth during shelf life | 5 | 1 | 10* | 100* |
| VI | Raw, ready to eat foods | 5 | 2 | 10* | 100* |

* denotes *L. monocytogenes* per g.

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

7.3.8 Canada

Canada has implemented a three-category system for *L. monocytogenes* in ready-to-eat foods based upon the health risk (Farber *et al*., 1996). This categorisation system is summarised in Table 19.

**Table 19: The microbiological criteria for *L. monocytogenes* for different categories of food and corresponding action levels in Canada**

| Category | Foods | Microbiological criteria for *L. monocytogenes* | Action level |
|---|---|---|---|
| 1 | Foods causally linked to listeriosis, (includes soft cheese) with a shelf-life >10 days. | absence in 50g | >0 cfu/50g Immediate action-Class I recall to retail level. |
| 2 | All other ready-to-eat foods capable of supporting growth, refrigerated shelf-life of >10 days. | absence in 25g | >0 cfu/25g Immediate action-Class II recall to retail level. |
| 3 (two types of foods) | • supports growth with refrigerated shelf-life of <10 days<br><br>• all other RTE foods not supporting growth;<br>➢ pH 5.0 – 5.5 and $a_w$ < 0.95<br>➢ pH <5.0 regardless of $a_w$<br>➢ $a_w$ ≤0.92 regardless of pH<br>➢ frozen foods. | ≤100 cfu/g with adequate GMP<br><br>≤100 cfu/g with inadequate or no GMP<br><br>>100 cfu/g | Immediate action-allow sale.<br>-follow up at plant level.<br><br>Immediate action-consider class II recall or stop sale.-follow up at plant level.<br><br>Class II recall or stop sale.-follow up at plant level. |

## 7.4 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 studies 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 by individual foods.

## 7.5 Environmental Contamination

The organism *L. monocytogenes* is ubiquitous in the environment. In soft cheese production, pasteurisation is the risk management tool in New Zealand. Assuming that pasteurisation is effective, there are two important possible sources of *L. monocytogenes* contamination;

- Ingredients added after pasteurisation, and
- Environmental contamination.

Breer and Schopfer (1988) reported that *L. monocytogenes* contamination of Swiss cheeses was restricted to the outer surface of the cheese, suggesting external contamination of the cheese (possibly during ripening), rather than contaminated ingredients.

A survey conducted in Australia detected *Listeria* in 19% of dairy factory environmental samples (Venables, 1989). Of the isolates, 93% were subsequently identified as *L. monocytogenes*. Testing showed that when *L. monocytogenes* was detected in product it had also been detected in the environment, or the bacterium was found soon after the positive product result was known. It was concluded that "Control of *Listeria* in the factory environment is a critical point in prevention of *Listeria* contamination of products"

Jacquet *et al*. (1993) examined product and environmental samples from within a dairy plant for *Listeria* contamination. *L. monocytogenes* strains were recovered during the ripening and rind washing stages, but not before. Isolates with the same serotype and phage type were isolated from cheese samples and ripening shelves, indicating that cheese contamination occurred during ripening.

Pak *et al*. (2002) carried out an extensive analysis of risk factors for *L. monocytogenes* contamination of dairy products in Switzerland. The strongest predictor of a positive culture for *L. monocytogenes* in the finished product was samples from cheese-ripening plants (OR 1.54; 95% CI: 1.14, 2.08). In-processing sampling produced a higher odds ratio (OR = 1.28) than end-product sampling (OR = 1.00). The study authors interpreted these results to mean

that cheese contamination was largely occurring during the cheese-ripening process. Environmental samples analysed in the study had 5.4 times higher odds of culturing positive for *L. monocytogenes* than the edible part of the cheese. The study also reported higher probability of surface contamination on hard and semi-hard cheeses than soft cheese.

Environmental contamination sources can be broadly categorised into personnel, equipment and pests. Control programmes to minimise contamination from these sources would include;

- Personnel hygiene,
- Personnel training,
- Equipment, fixtures and fittings, cleaning and maintenance,
- Hygiene of food contact materials,
- Transport and retail management, and
- Pest management plans.

### 7.6 Risk Management Options

Risk management falls into two categories, control of the microorganism in the foodstuff and education of the at-risk sub-populations.

In the Code of Hygienic Practice for Milk and Milk Products (Codex 2004), Annex 2 discusses the selection of individual control measures. The control measures are grouped according to their primary function;

- Microbiocidal, that reduce microbial load (e.g. pasteurisation, aging),
- Microbiostatic, that prevent, limit or retard growth of organisms by chemical/physical means (e.g. Extrinsic factors include time/temperature control and competing microflora, pasteurised or raw milk. Intrinsic factors include preservatives, water activity and pH), and
- Microbiostatic controls that prevent direction contamination (e.g. appropriate packaging).

Combinations of control measures have two main objectives; that during processing, pathogens are kept or reduced to acceptable levels and after processing, that the pathogens are kept under control through the product's shelf life.

Microbiocidal control measures (i.e. pasteurisation) take a predominant role in the risk management of soft cheeses in New Zealand. Microbiostatic controls then aim to prevent post pasteurisation contamination.

In New Zealand monitoring for *L. monocytogenes* after pasteurisation in processing plants is based primarily on environmental monitoring required under PSPs, with some additional end product testing. The rationale is that environmental contamination is the most likely source of contamination. Positive results from this monitoring are reported to the NZFSA and may result in risk management measures such as recalls.

The greatest risk for foodborne transmission of listeriosis is from foods with high numbers of *L. monocytogenes*. Targetting those foods for application of zero tolerance, or least to ensure a count of <100 cfu/gram at point of consumption 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 (Chen *et al*., 2003).

Conditions likely to result in large numbers of organisms becoming present in a food will include the following and risk management steps could be targeted at any of these points;

- 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 (either a long period of refrigerated storage or lesser periods of temperature abuse), and
- The absence of a listericidal step prior to consumption.

Milk being collected for cheesemaking purposes may be contaminated. Information on the status of raw milk in New Zealand in the scientific literature is limited (Stone, 1987), but the presence of *Listeria* spp. has been demonstrated.

As far as a listericidal step is concerned, there are currently three methods legislated for in New Zealand in relation to milk processing for cheesemaking. These are:

- Pasteurisation for all soft cheeses,

The other two methods are applicable to low-moisture cheeses only:

- Cheese treatment (thermisation and aging), and
- Methods accepted by FSANZ as being equivalent to the safety levels achieved by pasteurisation controls (can involve a cook curd step and/or long aging period).

Effective pasteurisation relies on the correct processing as well as the microbial quality of the raw milk (Dairy Industry Standard D115.1 requires that raw milk collected at the farm should not have an aerobic plate count at 30°C of more than $10^5$cfu/ml). Extrapolation to overseas data could be misleading because of the differences in herd management, year round grazing, use of silage etc.

Advice regarding consumption of soft cheeses is a risk management option, which should be linked to the well categorised risk groups for listeriosis. These include pregnant women, the elderly, adults with a compromised immune system e.g. renal transplant patients, patients on corticosteroid treatment, and HIV/AIDS patients.

Education of consumer groups most at risk, especially for pregnant women is an important intervention. Direct education campaigns targeted towards pregnant women are already used by the NZFSA and the Ministry of Health in New Zealand.

# 8 CONCLUSIONS

## 8.1 Description of Risks to New Zealand Consumers

### 8.1.1 Risks associated with soft cheese

Data on the prevalence of *L. monocytogenes* in domestically produced soft cheeses indicate that contamination rates are very low. The mandatory pasteurisation of milk for making soft cheeses in New Zealand means that contamination from the environment after this step is the most likely source of *L. monocytogenes* in this food. There are risk management measures in place under dairy industry Product Safety Programmes to detect and control environmental contamination during cheese manufacture. However, the ubiquitous nature of *L. monocytogenes* in the environment means that contamination of soft cheeses may occur at any point post-pasteurisation through to retail sale and domestic handling. Validation and verification of the pasteurisation process and avoidance of post-pasteurisation contamination are key areas to control.

Contamination of soft cheese may occur during handling in ripening rooms, wrapping and packaging stages or at retail/domestic cutting stages. Surface ripened cheeses are especially at risk because consequent rises in pH and other factors at the external crust may allow *L. monocytogenes* to grow, in contrast to the core of the same cheese which will remain at a low pH.

As an organism widespread in the environment, the general population and susceptible populations will be frequently exposed to *Listeria* spp. The available dose-response data indicate that for the general population the probability of invasive disease following exposure to even moderate levels of cells is very low.

New Zealand effectively has a zero tolerance for *L. monocytogenes* in dairy products (i.e. absence is required in a 25g sample). Certain countries overseas advocate a tolerance level of 100cfu per gram at point of consumption. The draft Codex guidelines for control of *L. monocytogenes* (Codex, 2002, Section 5.2) state that although limits are a responsibility of individual governments, a 99% reduction in the number of illnesses will be obtained by setting a Food Safety Objective at <100 *L. monocytogenes* per gram of food at point of consumption.

Consumption of soft cheese in New Zealand is modest, particularly when compared to European countries. When this is considered alongside the mandatory nature of pasteurisation, and data indicating a very low prevalence of contamination, the current risk from this food/hazard combination to the general New Zealand population must be considered low, although the risk to susceptible populations (with reduced immunity) will be greater.

The potential for growth of *L. monocytogenes* in soft cheeses depends on a number of factors. Intrinsic factors include the pH level, water activity and use of preservatives and starter cultures. Extrinsic factors include the time and temperature combinations in ripening/storage, humidity levels under which the cheese is ripened and whether pasteurised or raw milk was used. Because *L. monocytogenes* is a psychrotroph, refrigeration cannot be relied upon to inhibit growth.

There is considerable discussion as to whether the production and importation of cheeses made from unpasteurised milk should be permitted in New Zealand. This Risk Profile does not address the potential risk of such types of cheese for New Zealand. Overseas information indicates that contamination of raw milk by *L. monocytogenes* does occur, and at a high prevalence in some countries. Any risk assessment of cheese production from raw milk for New Zealand would require additional data, in particular the prevalence and concentration of *L. monocytogenes* in raw milk here.

The rate of reported invasive listeriosis in New Zealand is similar to that found in like countries (Table 10) at approximately 0.5 per 100,000 population. As in other countries, most cases are sporadic, with outbreaks being rare. There is currently no evidence to link cases of *L. monocytogenes* infection in New Zealand with soft cheese consumption.

Relative to other foodborne diseases, the number of invasive listeriosis cases reported each year is very small (26 in the year 2004). The low incidence described for the general population would be higher if calculations were done specifically for "at risk" groups. It is the high proportion of serious outcomes i.e. hospitalisation (100% of cases in the years 2002 and 2003) and death (approximately 15% of cases) which increases the importance of this disease.

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.

### 8.1.2 Risks associated with other foods

Foods appear to be a major vehicle of human infection with *L. monocytogenes* (ICMSF, 1996). It is likely that ready-to-eat foods contribute to foodborne listeriosis but foods on which it cannot grow, or which have a short shelf life are less likely to contribute to the disease burden significantly as the organism should not reach high numbers.

The USDA risk assessment listed as high (5 or above) relative risks of listeriosis for the following food groups (Table 14):

1. Deli meats;
2. Non-reheated frankfurters;
3. Pâté and meat spread;
4. Fluid unpasteurised milk; and
5. Smoked seafood.

In New Zealand, an outbreak of invasive listeriosis linked to smoked mussels has been identified. With regard to non-invasive listeriosis, two outbreaks have been reported (from the same incident) involving cooked RTE meat products.

The issue of cross contamination from other foods was considered in a paper on the role of foods in sporadic listeriosis (Pinner *et al*., 1992). Foods were collected from cases' refrigerators and isolates from cases and food were subtyped to find if there was any association. Samples of the same foods were also obtained from retail sources. From the 123 listeriosis cases, 79 (64%) had *L. monocytogenes* cultured from at least one food from their refrigerator. Twenty six of these 79 (33%) shared the same isolate in the food and in the patient. Those foods most likely to match the patient strain were ready to eat, grew by direct plating method (measure of rate of contamination) and contained serotype 4b.

In addition because of the long incubation period of invasive listeriosis (mean 30 days: range 1 – 90 days), it can be difficult to obtain accurate food histories and to avoid memory bias.

#### 8.1.3 Quantitative risk assessment

A quantitative risk assessment would be feasible for *L. monocytogenes* in soft cheese, provided sufficient data on the prevalence of the organism in the product at a retail level could be obtained. The NZFSA/ESR exposure assessment survey (Wilson, 2004) completed in 2004 did not detect *L. monocytogenes* in soft cheese, but the absence of positive results precludes the necessary quantitative data. However, it is difficult to see how the conclusions of such a risk assessment would be markedly different to those derived from the assessment conducted by the US FDA.

### 8.2 Commentary on Risk Management Options

The low level of risk for the general population from *L. monocytogenes* in soft cheese, as described in this Risk Profile, indicates that additional risk management measures are unnecessary. Nevertheless, information from France (de Valk *et al.*, 1998) indicates that soft cheeses have the potential to be a major vehicle for *Listeria* infection.

There is considerable discussion regarding whether the production and importation of cheese made from unpasteurised milk should be permitted in New Zealand. This Risk Profile does not address the potential risk of such types of cheese for New Zealand. Overseas information (Section 3.1.1.1) indicates that contamination of raw milk by *L. monocytogenes* does occur, and at a high prevalence in some countries. Any risk assessment of cheese production from raw milk for New Zealand would require additional data, in particular the prevalence and concentration of *L. monocytogenes* in raw milk here. There is little published information on this topic (only Stone, 1987), although industry sources may have unpublished data.

The use of unpasteurised milk to manufacture low-moisture cheese for the New Zealand market currently requires a combination of other controls to be used in order to produce an inactivation equivalent to pasteurisation, as described in the exemptions from pasteurisation granted by FSANZ for Swiss raw milk cheeses. Wherever contamination occurs, the exposure to the population will be greatly influenced by the ability of particular cheeses to permit growth.

The information in Section 3 indicates that is it not possible to make definite predictions about the behaviour of *L. monocytogenes* in types of soft cheese, and all soft cheese types must be regarded as potentially allowing growth. Consequently each cheese manufacturer would need to determine the risk from *L. monocytogenes* in their own process on a case by case basis.

Those well documented cases linking soft cheese consumption with listeriosis have generally occurred where high numbers of the organism have been reached. However, a cheese with a low level of contamination which is eaten frequently will also result in exposure to the population which may be significant.

In those cheeses where the organism is not inactivated, correct storage of cheese at refrigeration temperatures will not stop the growth of the organism, it will only slow growth down. Consequently a high level of hygiene during storage, as in ripening facilities, is essential.

There is currently no international agreement on what is an 'acceptable level' of *L. monocytogenes* contamination in foods. In addition there is no agreement on sample methodologies or sampling plans. For internationally traded foods, harmonisation in microbiological criteria based on risk assessment has been called for by FAO/WHO. It has been estimated by Codex that a 99% reduction in number of illnesses will be obtained by setting a food safety objective at <100 *L. monocytogenes* $g^{-1}$ of food at point of consumption (Codex, 2002).

Currently New Zealand, like Australia and the USA, has adopted a zero tolerance policy for *Listeria monocytogenes* in food. Some countries believe this approach is overly cautious and have adopted a non-zero tolerance policy.

The ICMSF have stated that microbiological testing of food must be viewed as a tool to verify that HACCP plans are working and are insufficient by themselves to ensure food safety. They advocate a series of actions depending on the product and its ability to support growth of *L. monocytogenes*. Soft cheese would fall into the category "ready to eat products; able to support growth of *L. monocytogenes,* whereby 20 samples should be taken and the lot rejected if any sample contains > 100 *L. monocytogenes* $g^{-1}$".

A regulatory regime that required some degree of end product testing would offer the option of the USA's approach where environmental and ingredient testing is conducted only after *L. monocytogenes* was found in product (although extensive environmental monitoring is likely to locate at least some *L. monocytogenes* in each manufacturing plant). Any such finding should be followed by full identification of the isolate (e.g. serotyping, PGFE) to improve the ability to locate and eliminate the source of contamination.

### 8.3 Data Gaps

The data gaps identified in this Risk Profile are:

- Prevalence and quantitative data on *L. monocytogenes* in soft cheeses sold in New Zealand. The 2004 NZFSA/ESR soft and semi-soft cheese *L. monocytogenes* prevalence survey did not detect any contamination, and provided few opportunities for

determination of quantitative data, other than to infer that a negative result corresponds to <0.04 cfu/g (absence in 25g) (Wilson, 2004);

- Prevalence and concentration of *L. monocytogenes* in raw milk in New Zealand. Any survey conducted to determine such data should be combined with testing for other human pathogens; and,
- Information on environmental *L. monocytogenes* contamination in New Zealand cheese production sites and associated areas.

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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, which 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 |
| NV[+]* | 478 | 478 |
| Toxins* | 414 | 414 |
| Typhoid* | 0.3 | 0.3 |
| Hepatitis A* | 0.4 | 0.4 |

*not recalculated.
[+] Norovirus

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<br>Major contributor to foodborne NV |
| 2 | 10-100 | Major contributor to foodborne salmonellosis<br>Significant contributor to foodborne NV |
| 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 |
| NV | 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, NV, 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