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RISK PROFILE:
LISTERIA MONOCYTOGENES
IN LOW MOISTURE CHEESES

Prepared as part of a New Zealand Food Safety Authority
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by

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IN LOW MOISTURE 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 low moisture cheeses, both domestically produced and imported. Low moisture cheeses are defined as having less than 50% moisture content. This includes categories often described (by FSANZ) as:

- Semi-soft (39% to 50% moisture) e.g. Stilton, Roquefort, Gorgonzola, Danish Blue, Limburger, Brick, Trappist, Port Salut, Bel Paesa, Pasta Filata, Provolone, Brick, Gouda, Edam;
- Hard (less than 39% moisture) e.g. Cheddar, Caciocavallo, Emmental, Gruyere; and,
- Very hard (<34% moisture content), e.g. Asiago old, Parmesan, Romano, Grana.

There are currently three methods of milk processing for cheesemaking in legislation for New Zealand. These are;

- Pasteurisation,
- Cheese treatment (thermisation and aging), and
- Methods accepted by FSANZ as being equivalent to the safety levels achieved by pasteurisation controls.

Pasteurisation aims to eliminate pathogenic bacteria such as *L. monocytogenes* from the milk at the beginning of production, whereas the latter two methods use milder heat treatment and/or control during ripening. Currently hard (<39% moisture) and very hard (<34%) 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 only imported thermised milk cheeses permitted into New Zealand are the semi-hard (39% to 50%) Swiss red label Tilsiter, Appenzeller and Vacherin Fribourgeois.

Where properly pasteurised milk is used for manufacture, *L. monocytogenes* would not be expected to occur in low moisture cheeses unless environmental contamination occurs during manufacture or ripening. In such cases, growth of *L. monocytogenes* in low moisture cheeses seems unlikely due to low water activity and low pH. Exceptions may occur when the (surface) pH is raised, such as in some mould or smear ripened cheeses.

For low moisture cheese made with raw milk, or thermised milk (in which *L. monocytogenes* may survive heat treatment), control of the organism is dependent on conditions during manufacture and ripening. The published data indicate that provided the pH is below 5.5 (through use of a starter culture) growth of *L. monocyotgenes* seems unlikely, although the organism may survive. The data are sufficiently variable that a reliable prediction of behaviour, in all low moisture cheeses made with these types of milk, is not possible. For

such cheeses, a careful consideration of the production process, both in generic terms and for a specific manufacturer, would seem to be warranted.

There is considerable discussion as to whether the production and importation of cheeses made from unpasteurised milk should be permitted. This Risk Profile does not address this issue but information contained in this document may be useful to the debate. 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, types and concentration of *L. monocytogenes* in raw milk here.

The majority of low moisture cheeses made in New Zealand or imported into the country are made from pasteurised milk, which means that subsequent contamination from factors such as added ingredients, further processing steps (e.g. grating), and from the environment represent the most likely source of *L. monocytogenes.*

Surveys showing the absence of *L. monocytogenes* in New Zealand retail grated low moisture cheese and semi-soft cheeses suggest that contamination rates are low. Growth is unlikely in most cheese types due to a variety of listeriostatic intrinsic and extrinsic factors (low water activity, competing microflora, acidification from the use of starter culture, time and temperature in storage combinations etc). Consumption of low moisture cheese types represents the majority of cheese consumption in New Zealand.

The rate of reported invasive listeriosis in New Zealand is similar to that found in like 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 consumption of low moisture cheese.

Overall, the available data indicate that *L. monocytogenes* in low moisture cheese in New Zealand currently does not represent a significant risk to human health.

The data gaps identified in this Risk Profile are:

- prevalence of *L. monocytogenes* in particular low moisture cheeses sold in New Zealand, specifically in semi-soft, mould-ripened cheeses, and cheese subject to forms of post production handling other than grating,
- quantitative data on levels of *L. monocytogenes* in low moisture cheeses when contamination does occur,
- prevalence, type and concentration of *L. monocytogenes* in raw and thermised milk in New Zealand. Any survey conducted to determine such data, should be combined with testing for other human pathogens,
- information on environmental *L. monocytogenes* contamination in New Zealand cheese production sites and associated areas, including typing and concentration levels,
- quantitative data on the amount of volumes of raw milk cheeses produced in the FSANZ approved method, both imported and produced domestically in New Zealand, and
- quantitative data on the amount of volumes of cheeses produced by the cheese treatment method, both imported and produced domestically in New Zealand.

1   INTRODUCTION

The purpose of a Risk Profile is to provide contextual and background information relevant to a food/hazard combination so that risk managers can make decisions and, if necessary, take further action. The place of a risk profile in the risk management process is described in "Food Administration in New Zealand: A Risk Management Framework for Food Safety" (Ministry of Health/Ministry of Agriculture and Forestry, 2000). Figure 1 outlines the risk management process.

Figure 1:   Risk Management Framework
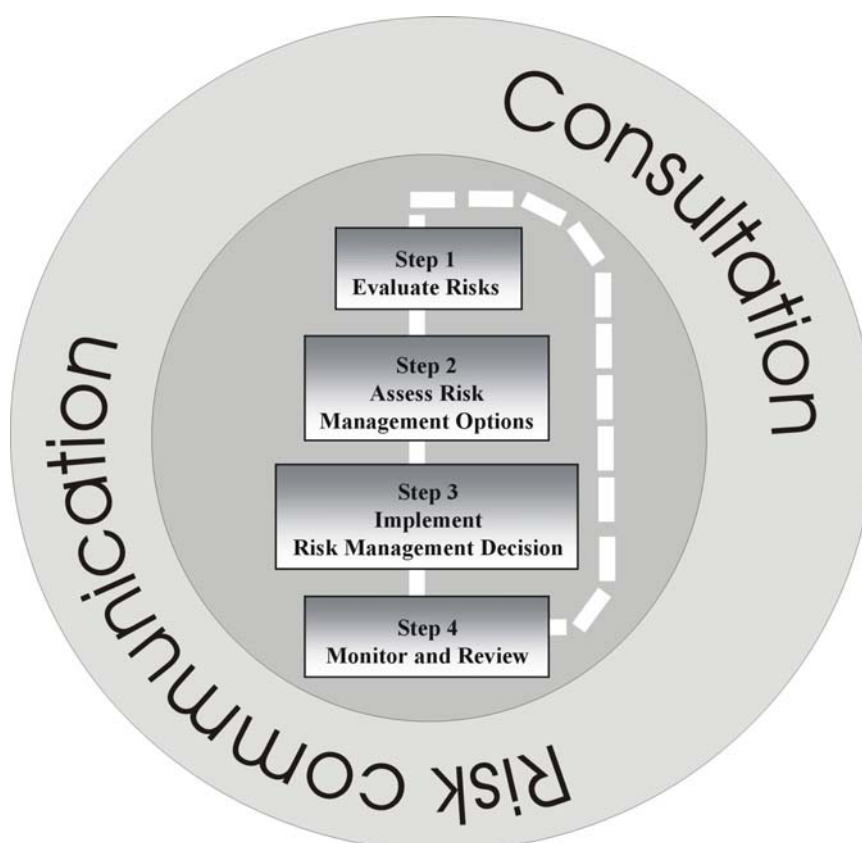
Figure reproduced from "Food Administration in New Zealand. A risk management framework for food safety" (Ministry of Health/Ministry of Agriculture and Forestry, 2000).

In more detail, the four step process is:

1. Risk evaluation

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

2. Risk management option assessment

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

3. Implementation of the risk management decision

4. Monitoring and review.

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

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

This Risk Profile concerns *Listeria monocytogenes* in low (<50%) moisture cheeses and complements other Risk Profiles on *L. monocytogenes* in dairy products (ice cream and soft cheeses).

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* 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.

There are various typing schemes for *L monocytogenes*:

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

While these typing schemes are useful in epidemiological and outbreak investigations, they are of limited use in distinguishing pathogenic from non-pathogenic strains. (ICMSF, 1996).

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.

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 is 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 NaCl and 0.92 in sucrose. The organism can grow in sodium chloride 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 survival appears to depend on the serotype.

Atmosphere: Modified atmospheres containing approximately 75% CO_2 and no oxygen inhibit growth of 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.

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).

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, since the *L. monocytogenes* are most likely located on the surface of these cheeses, 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 modifications (Ennahar *et al.,* 1994).

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

2.1.4   Sources

Human: *L. monocytogenes* is generally regarded as being carried asymptomatically in the faeces of 2-6% of the population. However, a recent study from the USA was able to detect *L. monocytogenes* in only a single stool sample from 827 tested, and the individual concerned was suffering invasive listeriosis (Sauders *et al.*, 2005). 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 and as it is not possible to milk animals aseptically, faecal contamination of raw milk, no matter how slight or infrequent, is inevitable (IFST, 1998). *Listeria* can also be excreted in the milk of healthy cows (Vizcaino and Garcia, 1975) and goats (Løken *et al*., 1982) as well as in mastitis affected animals - the organism can cause listerial mastitis (Back *et al* 1993).

Improperly made silage can be a source of farmed 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 while cheese made during the summer was not.

Food: The organism 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. 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, typically low levels are detected (<100cfu/g), although it has been detected at numbers far in excess of this (Farber and Peterkin, 1991, Sim *et al*., 2002).

Environment: It 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 domestic environments.

Transmission routes: One study estimates that 90% of cases are foodborne (Lake *et al*., 2000). Other reports describe foodborne transmission as the primary source of human infections. Alternative routes include infections acquired in hospital and occupational exposure (e.g. veterinarians).

3   HAZARD IDENTIFICATION: THE FOOD

3.1   Relevant Characteristics of the Food: Low Moisture Cheese

The flowchart in Figure 2 illustrates the production of low moisture cheese from pasteurised or thermised milk. It is reproduced with kind permission from the New Zealand Specialist Cheesemakers Association Inc., Interim Code of Practice (NZSCA, 2002).

Figure 2:   Generic flowchart illustrating the production of low moisture cheese from heat treated milk
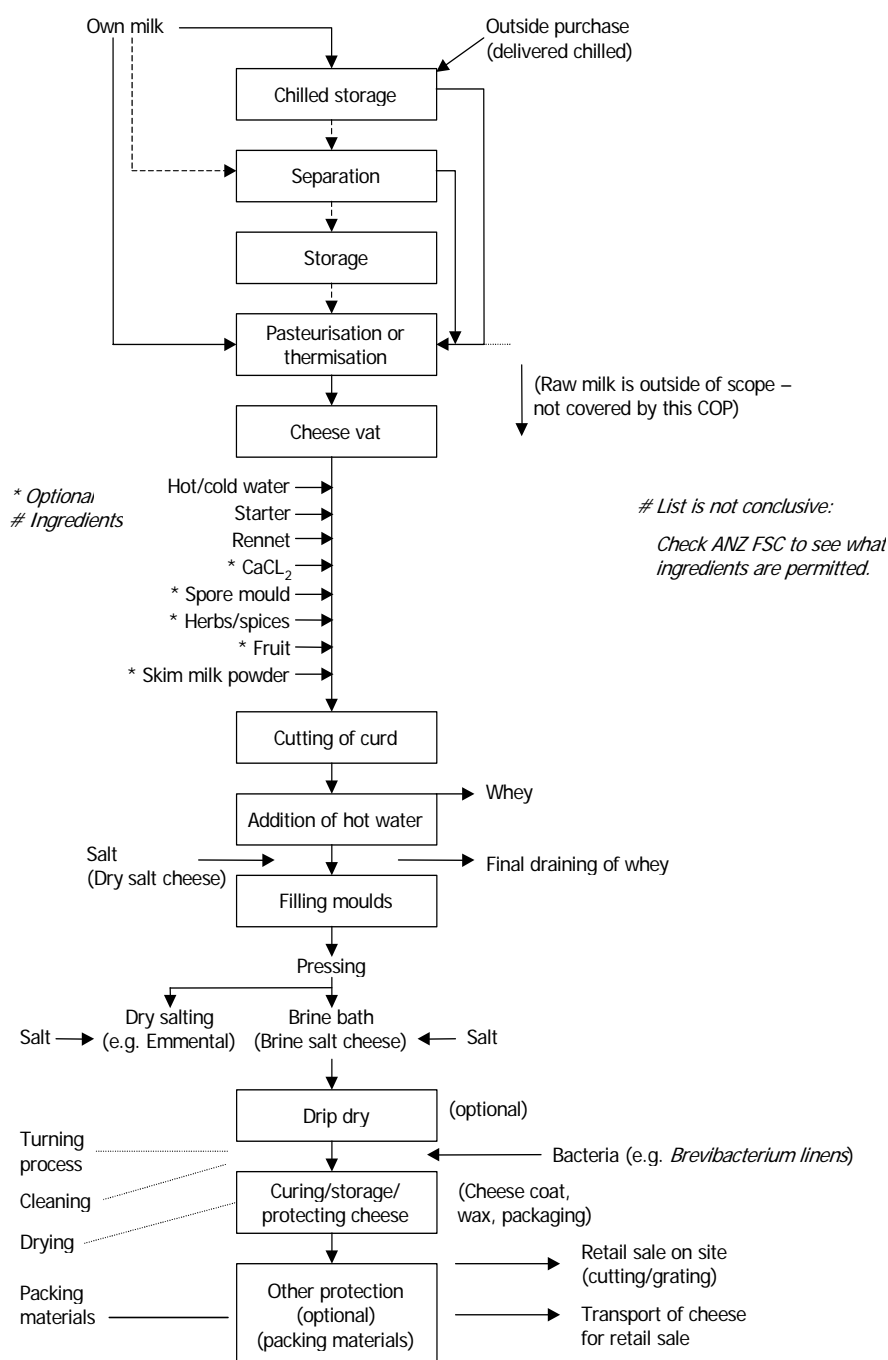
Cheese manufacture from milk is essentially a preservation technique as the dehydration process turns a highly perishable product into a less perishable one. Cheese is defined by Codex (1999b) as "the ripened or unripened soft, semi-hard, hard or extra hard product, which may be coated, and in which 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)".

The definition of a number of individual cheeses can be found in the Codex Report of the 6th session on Milk and Milk Products (http://www.codexalimentarius.net/reports.asp).

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 following paragraphs provide an overview of the cheese making process, it may be useful to refer to the flowchart in Figure 2 above for the generic process. Cheese production processes can vary widely, only the main steps from farm to fork are covered here.

3.1.1 Ingredients and processing

The main ingredients of low moisture 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 and its quality begins at the farm dairy. The use of high quality milk in terms of its chemical and microbiological composition is very important in the production of a good quality cheese.

Raw milk may contain *Listeria monocytogenes*, through direct contamination from the lactating mammal (usually cow, sheep and goat) or environmental contamination from sources such as the milking shed 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.

Inappropriate storage of the milk can further add to the microbial loading and the psychrotrophic nature of the organism 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 that 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 x 10^2/ml to 1.9 x 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 x 10^3 to 1.5 x 10^4 MPN/ml (Farber *et al.* 1988b).

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
Turkey	Raw	211	2 (0.9)	Uraz and Yücel, 1999
Netherlands	Raw	137	6 (4.4) <10^2/ml	Beckers *et al.* 1987
Scotland	Raw, bulk tanks	180 180 180	7 (3.8) summer 0 (0) autumn 2 (1.0) winter <1 cell/ml.	Fenlon and Wilson 1989
Switzerland	Raw	340	2 (0.6)	Bachman and Spahr

Country/region	Sample type	No. of samples tested	No.(%) positive for *L. monocytogenes*	Reference
				1994
UK	Raw	610	101 (16.5) <2 log$_{10}$	Food Standards Agency, 2003
	Pasteurised	*1413*	*0*	
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 Raw, dairy	192 64	(1.0) (3.1)	Davidson *et al.*, 1989
USA	Raw Raw Raw	124 121 650	15 (12.0) 15 (12.0) 27 (4.2)	Fleming *et al.*, 1985, Hayes *et al.*, 1986 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, an enhanced testing programme was able to detect *L. monocytogenes* at double this prevalence. 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, some pre-treatments on the milk may take place, such as (ICMSF, 1998);

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

Filtration is a common practice while the latter two treatments tend to be used by larger manufacturers rather than small producers.

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 micro-organisms by a centrifugal filtering process, this is discussed in more detail below.

Bactofugation, a specialised clarifier, reduces bacterial populations particularly spore forming bacteria, such as the *Clostridia* spp. This is particularly important for low moisture cheese production, where certain *Clostridia* may produce an undesirable late blowing or 'gassiness' in the cheese texture.

3.1.1.2 FSANZ approved raw milk methods, cheese treatment and pasteurisation

There are three ways in which milk can be processed to produce low moisture cheese that will comply with legislation in New Zealand (see Section 7.2). These are;

- Methods accepted by FSANZ as being equivalent to the safety levels achieved by pasteurisation controls,
- Cheese treatment, and
- Pasteurisation.

The majority of low moisture cheese imported onto New Zealand or produced domestically will be made from pasteurised milk. However, the amount of domestic or imported cheese produced by the cheese treatment method or FSANZ approved methods is unknown. This represents a data gap.

Raw milk: FSANZ approved methods

These are determined on a case-by-case basis for equivalence of safety in microbiological terms, using a benchmark of a 5 log (100,000 fold) reduction in pathogens. The small number of FSANZ approved methods are discussed in Section 7.2.5.

Thermised milk: Cheese Treatment

Cheese treatment is a heat treatment and aging process which can be influenced by other factors such as water activity, pH and the microbial loading of the raw milk. When the process is controlled, all these factors act to provide unsuitable conditions for *L. monocytogenes* survival.

The heat treatment involved is thermisation that uses lower temperatures than pasteurisation. Thermisation is defined in New Zealand as the rapid heating of milk to at least 64.5°C for at least 16 seconds. Research has shown that thermisation does not eliminate *Listeria*

monocytogenes from raw milk (see below). However, the definition of thermisation differs between countries, as is apparent in scientific papers. Therefore the thermisation conditions must be carefully specified.

Some cheese producers prefer thermised milk to pasteurised milk because enzyme properties in the milk are retained while many spoilage bacteria such as *Pseudomonas* are inactivated. Destroying spoilage bacteria is important because they can create flavour defects and problems with texture in the finished cheese.

Legal aspects of cheese treatment are discussed in more detail in Section 7.2.4. FSANZ has stipulated that cheese treatment may only be used for cheese with <39% moisture content and a pH level <5.6.

Johnson *et al.*, (1990) have carried out a literature review on the microbiological safety of cheese. A New Zealand report on the determination of death rates for pathogens during the thermisation and ripening process (Baldwin, 2001) has also been compiled. The information in these two reviews indicates that thermisation alone does not convey the same bacterial log reduction when compared to pasteurisation. A key study is by Farber *et al.,* (1988b), which found that inoculums (1x 10^5CFU/ml) of 10 *L. monocytogenes* strains into raw bovine milk (and naturally contaminated milk: 10^3-10^4 CFU/ml) did not survive heat treatments greater than 67.5°C (minimum holding time 16.2 seconds), but at 60°C, 61°C, 62°C, 63°C, 64.5°C, 66°C and 67.5°C, *Listeria monocytogenes* survived (holding times 16.2 seconds). At 62°C, there was a 2-log reduction in counts.   Gilmour *et al.*, (1981) also examined thermisation and concluded that the length of time between thermisation and cheese manufacture should be minimised.

Pasteurised milk: Pasteurisation

Pasteurisation is a higher temperature treatment, carried out either through a continuous or batch process. This step is a major critical control point for controlling microbial contamination in most dairy production in New Zealand. Pasteurisation requirements are defined in the relevant New Zealand Standard as;

- Holding method (63-66º for not less than 30 minutes), or
- High-temperature short-time method (>72ºC for not less than 15 seconds), or
- 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 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 with the pasteurisation parameters and any faulty equipment or incorrect operation is quickly identified. For example a high prevalence was reported for Spanish pasteurised milk, where 21.4% of samples from a single processing plant were positive (Fernandez Garayzabal *et al.,* 1986).

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). 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* 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.

It is important to stress the importance of environmental hygiene whichever type of milk treatment is used. If post-pasteurisation hygiene is poor, there are no further heat treatments to eliminate *L. monocytogenes.*

3.1.1.3 Acidification by 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. In a controlled process, pasteurisation kills most of the naturally present milk souring organisms; these are re-introduced into the pasteurised milk by selected cultures, which 'ripen' the milk i.e. transform milk into cheese curd. Where thermised or raw milk is used, most cheesemakers use starter cultures to achieve rapid acidification because the naturally present lactic acid bacteria (mainly lactococci) in raw milk are not sufficient.

Specific starter cultures are used to give characteristic properties to the cheese such as acidity, flavour and aroma. Cultures can be in liquid, frozen and dried forms and are generally added to milk at the rate of 1 to 1.5%. An antibiotic test carried out on the raw milk can identify if there are any inhibitory substances that may affect the starter culture, and therefore this acidification stage.

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. Mesophilic starter cultures result in a curd that can be heat treated at less than 40°C. Thermophilic starter cultures require a higher heat treatment at between 45-54°C to arrest the culture and slow the acidification. Similarly the correct ratio of starter culture to milk is an important point. 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).

3.1.1.5 Curd processing

In curd processing, a number of activities such as cutting, scalding, stirring, pitching, turning and piling (cheddaring) take place in order to expel whey and firm the curd, before it is milled, salted and pressed. The casein (protein) and fat of milk are concentrated approximately tenfold in low moisture cheeses.

Once the cheese has coagulated to the desired consistency, the curd is cut. Smaller cubes for created for low moisture cheese and larger cubes for softer cheeses, as the size of cut determines expulsion of whey.

The water activity of cheeses varies greatly, even within those categorised as low moisture. For example processed cheese has a water activity of 0.90-0.95, aged cheddar cheese 0.80 to 0.90, and Parmesan 0.60 to 0.70. The minimum water activity allowing the growth of *L. monocytogenes* is 0.90-0.92, and so growth in some cheeses is prevented by this parameter alone.

The curd is then 'cooked' or 'scalded' (<40°C for mesophilic starter cultures, up to 54°C for thermophilic starter cultures). The curd cooking stage arrests the starter culture, further removes whey and alters the texture of the curd particles. However, this step does not reach temperatures that will inactivate *L. monocytogenes*.

In several studies, a higher concentration of *L. monocytogenes* has 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). Yousef and Marth (1988) calculated that only 2.4% of the *Listeria* population in their experimental Colby production escaped in the whey.

Various additional ingredients (e.g. fruit, vegetables, moulds, herbs, nuts and seeds) may be added at the renneting or curd cutting stage. The ingredients should be pathogen free in order to avoid post pasteurisation contamination. Stabilisers may also be added during production. One particular optional addition at this stage is skimmed milk powder. Skimmed milk powder appears to have the potential to contaminate the cheese at this point. Doyle *et al.*, (1985) cited in ICMSF (1998) observed a reduction of *L. monocytogenes* during the drying and ambient storage of milk powder however, although the organism declined in numbers during storage, some samples remained positive for *L. monocytogenes* 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

Salting has a general prohibitive effect on bacterial activity, and so stops acidification by starter culture. 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).

However, salt does not have a strong bactericidal effect on *L. monocytogenes* until relatively high concentrations are reached. *L. monocytogenes* behaviour in broths with salt concentrations up to 26% has been studied (Hudson, 1992). At 10°C, *L. monocytogenes* grew at salt concentration of 6%, was static at 16% salt, and declined in 26% salt.

Where cheese is brined after pressing, the solution of the brine should be at least 50% saturated (salt saturation point 36% by weight) and changed or heat treated frequently. Weaker brines require more frequent replacement and maintenance, therefore most cheese makers use saturated brine.

3.1.1.7 Ripening and Storage

The ripening period (also known as curing, maturing, holding period) develops the flavour and texture of the cheese, lowers the pH, and reduces the water activity. Ripening temperatures and humidity depend on the type of cheese being produced. Low-moisture cheeses may ripen for a year or more after processing and are so stable they have a long shelf life after that. For some surface ripened cheeses, such as smear cheeses, ripening is performed at high humidity to encourage the growth of surface micro-organisms for some weeks. Conversely, low humidity is necessary for most low moisture cheeses where internal enzymatic activity is encouraged and surface microbial growth is discouraged (ICMSF, 1998).

Ripening is not viewed as a critical safety factor for pasteurised cheese. However it is critical for those cheeses made from thermised or raw milk.

In the case of cheeses produced by the cheese treatment method (i.e. <39% moisture content and a pH level <5.6) the thermisation process is coupled with a ripening period set down in legislation at a minimum of 90 days at a temperature not less than 7°C from the date of commencement of manufacture.

The greatest inactivation occurs when the holding temperature is high and the pH of the cheese low (Baldwin, 2001). The presumption is that after the 90-day period, taken with the other factors such as low moisture and pH levels, *L. monocytogenes* will have become non-viable (see Sections 3.2.5 and 5.3.4 for further discussion). Care must therefore be taken when further processing takes place, not to re-introduce moisture, for example when slicing or grating takes place.

For FSANZ approved method cheeses (see Section 7.2.5); Emmental, Gruyère and Sbrinz raw milk cheeses are stored for at least 90 and up to 360 days.

For the FSANZ approved raw milk, extra hard grating cheeses (Parmesan style), the criteria is that provided the cheese undergoes a curd heating stage to least 48°C and the cheese has a moisture content <36%, maturing time must be at least 6 months at no less than 10°C.

After ripening, low moisture cheeses are normally kept in the chill chain until retail sale.

Environmental contamination from *Listeria spp*. can occur at any point during production and storage, there is further discussion in Section 7.5.

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 substances inhibitory to starter cultures: to ensure correct acid development,
- milk pasteurisation (if applied): to destroy pathogens,
- correct acidification: to inhibit pathogens, and
- correct salt addition: to inhibit pathogens.

3.1.2   Definition of low moisture 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 low moisture cheeses are defined as a group based on moisture content. There are a variety of definitions for moisture content of cheese and none seem to be universally accepted.

For this Risk Profile, it was agreed with the NZFSA that the definition of low moisture cheese would be less than 50% moisture. Attachment 2 of the User Guide to Standard 1.6.1 Microbiological Limits for Food published in July 2001 by FSANZ gives their classification scheme for cheeses and is presented in Table 2. The definition of "low moisture" used in this Risk Profile therefore coincides with semi-soft, hard and very hard cheese classifications in the FSANZ scheme and corresponds with the FDA/FSIS (2003) risk assessment work carried out in the USA (http://www.cfsan.fda.gov/~dms/lmr2-5.html).

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

Moisture content (%)	Cheese Type
50-85%	*Soft Cheeses* Unripened e.g. Cottage, Quark, Cream, Mozzarella (soft variety). Ripened e.g. Camembert, Brie, Neufchatel, Caciotta. Salt-cured or pickled e.g. Feta, Domiata.
39-50%	*Semi soft* Ripened principally by internal mould growth e.g. Stilton, Roquefort, Gorgonzola, Danish Blue Ripened by bacteria and surface micro-organisms, e.g. Limburger, Brick, Trappist, Port Salut. Ripened primarily by bacteria e.g. Bel Paesa, Pasta Filata, Provolone, Brick, Gouda, Edam.
<39%	*Hard* Without eyes, ripened by bacteria e.g. Cheddar, Caciocavallo. With eyes, ripened by bacteria e.g. Emmental, Gruyere.
<34%	*Very hard* 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 moisture and ripening method. The moisture is determined on a 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 define moisture of cheese into two types; "soft and semi-soft" and "firm and hard cheese", primarily for export reasons. 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.

3.1.2.1 Red smear cheeses

Red smear cheeses are bacterial/yeast surface ripened, often involving the characteristic orange pigmented bacterium *Brevibacterium linens* giving the cheese surface a distinctive red colour. The main yeasts identified are *Geotrichum candidum* and *Debaryomyces hansenii.* Moisture content can vary so these cheeses may be soft, semi-soft or hard (e.g. Port Salut, Limburger, Reblochon, Livarot, Tilsit and Gubbeen cheese). Ripening is carried out under conditions under which a progression of complex microbial consortia occurs on the cheese surface (Rudolf and Scherer 2001). The micro-organisms are added either as a defined culture to the brine, or as a form of "back slopping" where a complex undefined microflora is washed from the surface of ripened cheese and used to inoculate unripe cheeses. The result of the microbial activity at the cheese surface is to raise the pH, however this can allow the growth of *L. monocytogenes* if present (Leuschner *et al.*, 1998).

3.1.2.2 Processed cheeses

Cheeses produced by the methods outlined above are termed natural cheeses. Processed or plastic cheeses are defined by FSANZ in Food Standard Code 2.5.4 as a product manufactured from cheese and products obtained from milk, which is heated and melted, with or without added emulsifying salts, to form a homogeneous mass, although the Code does not define moisture or compositional standards. For the purposes of this risk profile, processed cheese will be included but not processed cheese spreads. The US FDA definition will be used for processed cheese. This requires a moisture content of less than 44%.

Processed cheese spreads are defined by the US FDA as having between 44% to 60% moisture content, and in addition must contain at least 51% cheese as an ingredient. Process cheese spreads do not fall into the scope of this risk profile because cheese is a part ingredient.

One type of processed cheese not heat treated is cold-pack or club cheese manufactured by grinding and mixing natural (mainly cheddar) cheeses (ICMSF, 1998). It can be made from one or more varieties of cheese. The moisture content must not be greater than that which defines the originating cheese(s) and no greater than 44%. Because this cheese is not subsequently heated, spoilage by yeasts and moulds can be problematic. Therefore the constituents must have been produced from pasteurised milk or the product must have been aged for a minimum of 60 days >1.7°C.

Other processed cheeses are prepared by grinding one or more natural cheeses, mixing with melting salts and milk products (depending on type of cheese being made). This blend is then heated to between 85 to 95°C (under pressure to 110°C) for several minutes. The hot, semi-liquid is poured into containers to set or is chilled over rollers as slices. The stability of processed cheeses under refrigeration or ambient temperatures depend upon the heat treatment used, added preservatives and packaging methods. Because of the highly processed manufacture, added preservatives and heat treatments used, it is not an environment conducive to the growth of *L. monocytogenes*.

3.2   Survival and Growth of *L. monocytogenes* in or on Low Moisture Cheeses

L. monocytogenes contamination of cheese may come from two main sources,

- The milk or other ingredients used to make the cheese, or
- From surface contamination / the environment during production and post-production.

L. monocytogenes can be introduced with the starter culture, whether the starter is pH-controlled or not. The pathogen 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, 1999a). Similarly *L. monocytogenes* is rapidly inactivated in colourants and starter distillates used in the manufacture of some kinds of cheeses.

Listeria monocytogenes may grow during the early stages of cheese production prior to the pH reducing to an inhibitory value. No growth should occur at subsequent stages unless conditions change; an example of this being where the pH rises in some cheeses during ripening. Factors influencing the survival of the organism include rate of acidification, effect of lactic acid (which will be influenced by pH), production of other acids in some cheeses, curd cooking temperature, moisture content, and ripening conditions.

The most comprehensive review of *L. monocytogenes* behaviour in low moisture cheeses was collated by the US FDA/FSIS (2003) for the purposes of a Quantitative Risk Assessment (see section 6.2.4). A summary of the studies is presented below.

For "semi-soft" cheese (39-50% moisture), 10 data sets provided growth and survival data. Eight of the data sets show levels declining, with an estimated growth rate of -0.011 to -0.070 $\log_{10}$ cfu/day at 5°C. One set demonstrated survival for 6 weeks in Gouda and one set demonstrated growth (<1 log in 20 weeks) in Tilsiter, Trappist, Havarti, and Limburger cheeses.

The individual studies are listed below.

Type	Temp. (°C)	Growth rate	Reference
Semi-soft Cheese			
Brick (surface ripened)	10	1 to 7-fold decrease in 20 weeks	Ryser and Marth, 1989
*Tilsiter, Trappist Havarti, Limburger	10	<1 log in 20 weeks	Ryser and Marth, 1989
Trappist	10	Initial 1 log during ripening stable 30 days, 1 log decr. for 90 days	Kovincic *et al*., 1991
*Gouda	-	Survival 6 weeks	Northolt *et al.,* 1988
Monterey Jack	4	>2.1 log decr. in 30 days	Genigeorgis *et al*., 1991
Limburger	4	2.26 log decr. in 36 days	
Provolone	4	2.36 log decr. in 36 days	
String cheese	4	2.29 log decr. in 36 days	

| Muenster | 4 | 2.0 log decr. in 36 days |

In hard cheese (<39% moisture), seven studies provided data on growth and survival. Of 11 data points available, 10 indicated declines in *Listeria monocytogenes* populations with an estimated growth rate range of -0.003 to -0.228 $\log_{10}$ cfu/day at 5°C. Growth was only observed in Stilton (0.7 logs in 6 weeks). The storage times for hard cheese in these studies were 30 to 150 days.

The individual studies are listed below.

Type	**Temp. (°C)**	**Growth rate**	**Reference**
Hard Cheese			
*Stilton	4	0.7 log in 6 weeks	Whitley *et al.*, 2000
Colby	4	1.5 log decr. in 100 days	Yousef and Marth, 1988
Cheddar	13	2 log decr. in 75-150 days	Ryser and Marth, 1987
Swiss	7	4 log decr. in 10 days (complete inactivation 66-80 days ripening at 24°C).	Buazzi *et al.*, 1992
Parmesan	-	2 log decr. in 40 days 2 log decr. in 80 days	Yousef and Marth, 1990
Swiss	4	>2.1 log decr. in 36 days	Genigeorgis *et al.*, 1991
Cheddar	4		
Cracker Barrel		1.17 log decr. in 34 days	Genigeorgis *et al.*, 1991
Cheddar, mild		>2.1 log decr. in 30 days	
Cheddar, sharp		>2.1 log decr. in 36 days	
Colby		0.81 log decr. in 36 days	

* Survival or growth observed.

Processed cheese

In processed cheeses, three studies provided six data sets. All six show a decline. Exponential growth rates modelled at 5°C ranged from –0.004 to -0.15 $\log_{10}$ cfu/day.

The individual studies are listed below.

Type	**Temp. (°C)**	**Growth rate**	**Reference**
Processed cheese			
American processed	4	0.18 log **decr**. in 36 days	Genigeorgis *et al.*, 1991

Monterey Jack processed	4	1.84 log **decr**. in 36 days	
Piedmont processed	4	1.62 log **decr**. in 36 days	
Pasteurised process cheese	30	0.6 log **decr**. in 96 hours	Glass *et al*., 1998
Cold pack cheese (non-acid)	3	0.5 log **decr**. in 110 days	Ryser and Marth, 1988
Cold pack (acidified or preservatives)	3	1.0 log **decr.** in 60 days	

3.2.1 Semi-soft cheese

L. monocytogenes added to milk was able to grow during the initial hours of blue cheese manufacture (37.9-40.0% moisture depending on batch) increasing by up to 2 log_{10} units during the first 24 hours. Growth ceased when the pH dropped below 5.0 (Papageorgiou and Marth, 1989). The numbers of *L. monocytogenes* then declined approximately 3 log_{10} units during the first 50 days of ripening. This is probably due to the high salt content (effective salt in water phase concentration of 10.9 to 12.3%) in combination with other hurdles. After 50 days, the pH-raising effect of the growth of *Penicillium roqueforti* allowed survival but no growth. The mould may have contributed to this inactivation through intense proteolysis and lipolysis producing free fatty acids, methyl ketones and corresponding secondary alcohols. The organism was still detected in the cheese 120 days after manufacture where the cheese was ripened at 9-12°C for 84 hours after production and then stored at 4°C.

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 soft 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 otherwise be expected to grow.

L. monocytogenes added to milk during the manufacture of "semi-hard" (moisture level not given) cheese (described as similar to Manchego-type) was able to grow during cheese manufacture when inoculated at approximately 5.3 log_{10} cfu/ml, but not at 3.6 log_{10} cfu/ml. However, a reduction of 1-2 log_{10} cfu/ml occurred after brining. During ripening at 15°C for up to 60 days the numbers of bacteria were stable, or increased slightly (Dominguez *et al.,* 1987). The presence of a starter culture did not seem to influence the survival of the pathogen, and the final pH was beween 5.1 and 5.8.

During the ripening at 12°C of unpasteurised "semi-soft" (moisture level not given) goats' milk cheese the numbers of *L. monocytogenes* declined only slowly in two of six batches of cheese (Tham, 1988). These were the two with the highest inocula of the pathogen (approximately 2 x 10^6/ml). In one batch, with the lowest inoculum (4.5 x10^5/ml), *L. monocytogenes* was not detected, while in the remaining three batches, it was detected by enrichment only up to 16 weeks, 12 weeks and 10 weeks respectively.

3.2.2 Hard cheese

One of the few studies to demonstrate growth of *L. monocytogenes* on the surface of a low moisture cheese was with inoculated Stilton, (Whitley *et al.*, 2000). The mature cheese was packaged under modified atmospheres (MAP) and samples stored at 2.1-7.2°C. Under a $N_2:CO_2:O_2$ mix of 80:10:10 growth occurred to give a mean 5,000% increase in numbers (or 1.6 log) after six weeks storage. Growth also occurred in a $N_2:CO_2:O_2$ 100:0:0 mix, but a slight reduction occurred with 80:20:0. The pH of the cheese was in excess of 5.9 in all trials at all time points sampled. This study demonstrated that film wrapping is insufficient to control growth of *L. monocytogenes* in Blue Stilton cheese and that MAP, whether using atmospheres containing oxygen or not, does not control growth in such cheese.

Federal US laws stipulate that Colby cheese must not contain more than 40% moisture. During experimental Colby cheese manufacture (cheese moistures ranged from 38 to 43%) some growth of bacteria occurred, followed by a period of stability in numbers and then a gradual decline during ripening at 4°C (Yousef and Marth 1988). The D values for two *L. monocytogenes* isolates ranged from 143 to 51 days. However, in cheeses containing the legal moisture content (<40%) the D times ranged between 51 and 67 days.

Survival of *L. monocytogenes* in Cheddar cheese for over a year has been demonstrated during the manufacture and subsequent storage at 6 and 13°C (Ryser and Marth 1987). Some growth occurred during the first 14-28 days of ripening modest growth (<1.0 $\log_{10}$ units) occurred. An initial inoculum of 500 cells/ml into pasteurised milk was used (therefore results can not predict behaviour of heat-injured cells), and the authors point out that co-mingling of milk from many farms would probably prevent a high level of contamination in industry. Growth of *L. monocytogenes* during manufacture appears to be inhibited by proper acid development resulting from an active starter culture. An extended lag phase could also result from growth competition with the lactic culture. The average pH values obtained from day 0 to end of ripening were all consistently between 5.00 and 5.15. The average moisture content was 37.2%.

No difference was observed between the behaviour of *L. monocytogenes* on reduced fat (36.2% fat) and control (48% fat) cheddar cheeses. Although the reduced fat cheeses in the study had slightly higher moisture contents (36-43%) than control cheese, the water activity was similar and did not appear to affect behaviour. Numbers of *L. monocytogenes* increased by approximately 1.3 $\log_{10}$/g during manufacture and then slowly decreased by 0.6-0.8 $\log_{10}$/g over 20 weeks of ripening (Mehta and Tatini, 1994). Information was provided on initial pH and water activity, but changes during ripening were not monitored.

In Parmesan (moisture levels at 30-31.5% and pH 5 after ripening), inocula of 10^4 to 10^5 cells per ml were added to cheese milk. The pathogen grew to maximum numbers just before cooking of the curd. During the cooking stage, numbers declined and in the ripening stage, there was steady decrease, faster than reported for other hard cheeses (Yousef and Marth 1990). The organism ceased to be detected in most cases at less than 60 days. Federal US standards specify that Parmesan must have at least a 10 month ripening period, contain <32% moisture and a minimum 32% fat in dry matter. The experimental cheeses met these parameters. The authors suggested that this cheese was unfavourable for extended survival of *L. monocytogenes* because;

- Production by the starter culture of bactericidal agents (e.g. bacteriocins). It has been suggested that *Lactobacillus bulgaricus* may be bactericidal to *L. monocytogenes* (Schaack and Marth, 1988),
- Injury of the pathogenic cells during cooking of the cheese curd due to the combined effect of heat and acidity. Conditions in the cheese being unfavourable for the repair of the cells, and
- Drying the cheese blocks to a low water activity (a_w), the relatively high temperature of ripening (12.8°C) and the addition of lipase during the manufacture (with expected release of fatty acids in the cheese).

3.2.3 Swiss cheeses (semi-soft and hard)

In nine batches of Swiss cheese the final pH achieved was between 5.2 and 5.4, and moisture ranged from 35.0 to 39.0% (Buazzi *et al.,* 1992). When *L. monocytogenes* was inoculated prior to the starter culture there was a slight rise in numbers during production followed by the greatest decrease in numbers during brining of the cheese blocks at 7°C for 30 hours. The population continued to decrease during the ripening period (24°C for 80 days). Average D values for the three isolates examined were 29.2, 24 and 22.5 days. After 80 days of ripening the pathogen could not be detected. The typical flavour of Swiss cheese is correlated with its content of acetic acid and propionic acids and the extent of lipolysis. More lactate is fermented to acetate (a typical 60 day old cheese contains 2000 ppm acetic acid) and carbon dioxide than propionate by *Propionibacterium shermanii* used in the production of this cheese, and this may be correlated with the inactivation of the pathogen. It was concluded that Swiss cheese made with milk containing a low initial contamination of *L. monocytogenes* ($\leq 10^2$cfu/ml of cheese milk) should be free of the organism after 60 days of ripening as is required for Swiss cheeses made from raw or heat-treated milk in the US.

In Switzerland, commercially made hard cheeses (assumption <39% moisture) and semi-hard (assumption 39% to 50% moisture) are always made from raw milk in a discontinuous process (in contrast to commercially produced Swiss soft cheeses which are produced only from pasteurised milk). *L. monocytogenes* was not detected in Swiss Emmentaler-type raw milk hard cheese (35% moisture) beyond one day after manufacture (Bachman and Spahr 1995). The disappearance was attributed to the synergistic effect of:

- Active antimicrobial enzyme systems of fresh raw milk,
- Antagonistic starter culture flora,
- Fast acidification,
- Antimicrobial effect of lactic acid,
- High curd cooking temperatures (53°C for 45 minutes), and
- Ripening at relatively high temperatures (10 to 22°C).

However, in Tilsiter-type cheese (39% to 50% moisture), a number of *L. monocytogenes* survived in the interior after 90 days of ripening, although no growth was observed. Rapid acid production was considered the principal factor responsible for most of the reduction of pathogens in Tilsiter-type cheese, and thus the use of an effective starter culture was critical. There was however no bactericidal heating step in the production of this type of cheese. The organism was reported to grow profusely on the surface of this cheese although no data were given because the surface of these cheeses is considered part of the packaging. Therefore with regards to *L. monocytogenes*, the manufacturing parameters of Swiss "semi-hard" (39%

to 50% moisture) cheese are not bactericidal, only bacteriostatic (Bachmann and Spahr, 1995).

Breer and Schopfer (1988) reported that *L. monocytogenes* contamination of a variety of cheeses in Switzerland was restricted to the outer surface of the cheese, suggesting external contamination of the cheese (possibly during ripening), rather than contaminated ingredients. No significant difference was observed in the prevalence of *L. monocytogenes* contamination in cheese produced from pasteurised (12.2% listerial contamination) and non-pasteurised (13.9%) milk reinforcing the view that the contamination was environmental.

3.2.4   Processed cheese

In the study of pasteurised processed cheese slices by Glass *et al.*, (1998), the slices were inoculated with a 3 strain mix to yield approximately 10^3 cfu/g. The slices were then stored at 30°C under aerobic conditions for upto 96 hours. The moisture content ranged between 39.1% and 40.3%, the pH level between 5.61 and 5.84 and the water activity between 0.918 and 0.929. Populations of *L. monocytogenes* decreased by 0.6 $\log_{10}$ CFU/g during the 96 hour period. The authors concluded that the small but consistent decline in numbers was due either to the minimum water activity for listerial growth being lower in media than in processed cheese or an additional inhibitory factor in the cheese.

Although *L. monocytogenes* has not been isolated from commercially available cold-pack cheeses, the ingredients used have the potential to harbour the bacterium. In cold-pack cheese stored at 3°C, Ryser and Marth (1988) inoculated (cheddar) cold-pack cheese lots with approximately 5 x 10^2 cfu/g. The cheese was manufactured with or without preservatives or acidifying agents to determine their effect (8 different formulations). Populations of *L. monocytogenes* decreased in all of the lots, although not unexpectedly the rate of decline was slowest in non-acidified cheese made without preservatives. The results show which preservative is most active against *L. monocytogenes* when used at its maximum allowable concentration (in the USA) in cold-pack cheese. The authors concluded that to decrease the survival chances of *L. monocytogenes*, the addition of preservatives (particularly sorbic acid) and small amounts of lactic and/or acetic acid t(o increase acidity to pH5) would be prudent.

3.2.5   Conclusions

The FDA/FSIS collated studies on the behaviour of *L. monocytogenes* found that for semi-soft cheese, 8 out of the 10 data sets reported a decline in numbers, one set showed survival and one set demonstrated growth. For hard cheese, 10 out of 11 data sets reflected a decline in numbers. A Blue Stilton (Whitley *et al.,* 2000) showed growth, the authors commenting that film wrapping (and Modified Atmospheric Packaging- with or without oxygen) was insufficient to control growth in this cheese.

One of the key studies used in the FDA/FSIS QRA was the comparison of the ability of *L. monocytogenes* to grow on the surface of 24 types of cheeses available in the USA (Genigeorgis *et al*., 1991). The authors found a highly significant correlation between *Listeria* growth, cheese pH values higher than 5.5 and/or an absence of lactic acid starter cultures. The study demonstrated therefore that low moisture cheeses made with the use of starter cultures and at pH values of <5.5, (as well as processed cheeses), would not support

growth of *L. monocytogenes* at 4 to 30ºC if they became surface contaminated at some point after production.

In internally ripened blue cheese, the production of MCFA appeared to act as an inhibitor of *L. monocytogenes* (Kinderlerer *et al*., 1996).

Because of the highly processed manufacture, added preservatives and heat treatments used, processed cheese does not provide an environment conducive to the growth of *L. monocytogenes*.

Where properly pasteurised milk is used for manufacture, *L. monocytogenes* would not be expected to occur in low moisture cheeses unless environmental contamination occurs during manufacture or ripening. In such cases, growth of *L. monocytogenes* in low moisture cheeses seems unlikely due to low water activity and low pH. Exceptions may occur when the (surface) pH is raised, such as in some mould or smear ripened cheeses.

For low moisture cheese made with raw milk, or thermised milk (in which *L. monocytogenes* may survive heat treatment), control of the organism is dependent on conditions during manufacture and ripening. The published data indicate that provided the pH is below 5.5 (through use of a starter culture) growth of *L. monocytogenes* seems unlikely, although the organism may survive. The data are sufficiently variable that a reliable prediction of behaviour, in all low moisture cheeses made with these types of milk, is not possible. For such cheeses, a careful consideration of the production process, both in generic terms and for a specific manufacturer, would seem to be warranted.

3.3   The Food Supply in New Zealand

3.3.1   Production

The total reported production of cheese in New Zealand during 2003 was 275,000 tonnes (MAF, 2003). Exports were actually higher than production (293,000 tonnes; MAF, 2003) as Fonterra exported a large amount of product from its inventory.

New Zealand's level of cheese production is modest compared to countries such as the United States, France and Germany, but 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).

The domestic cheese market in New Zealand is dominated by two large companies; New Zealand Dairy Foods (NZDF) and Mainland. 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, along with overseas markets information; cheese type produced is classified as firm and hard (moisture in solids not fat (MSNF)=60%). The only other category in this table is processed

cheese which is indicated by the registration number. N.B. cheesemakers who make only soft or semi-soft cheese (MSNF >60%), cottage or cream cheese are not included in this table. 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	Markets
802	Alpine Cheese Ltd.	
9682	Aroha Organic Goat	
137	Art of Cheese Ltd.	
94	Barry's Bay Cheese	
595	Blue River Dairy Products Ltd.	BR
905	Canaan Cheeses	
795	Canary Enterprises Ltd.	
1630	Delago Limited	
2015	Dairymaid foods Ltd.	
30	Evansdale Cheese Factory	
1203	Hautapu Cheese Dev. Fonterra Ltd.	SL
1273	Hautapu Cheese Dev. Fonterra Ltd.	BR, EU, SL
1538	Clandeboye Grated |Parmesan, Fonterra Ltd.	BR,EU,NC,PA,SL
1571	Fonterra Ltd.	EU, PA, SL
1573	Clandeboye Cheese, Fonterra Ltd.	BR,EU,NC,PA,SL
2065	Auckland Samples, Fonterra Ltd.	
2573	Lichfield Cheese 1 & 2, Fonterra Ltd.	BR, EU, SL
3673	Edendale cheese, Fonterra Ltd.	BR, EU, SL, PA
4773	Whareroa Cheese 1 & 2, Fonterra Ltd.	BR,EU,NC,PA,SL
7373	Waitoa Cheese, Fonterra Ltd.	BR, EU, SL
7473	Stirling Cheese, Fonterra Ltd.	BR,EU,PA,SL
85	Kemp Roberts Creamery Ltd.Glenbrook Foods Ltd.	
115	Grated cheese Co. Ltd.	
168	Kapiti Fine Foods Ltd.	
40	Karikaas Natural Dairy Products Ltd.	
65	Mahoe Cheese	
162 (P)	Mainland Products Ltd.	BR, CR, SL
31 (H & P)	Natural Pak, Mainland Products Ltd.	BR
38 (H & P)	Mainland Products Ltd. – Grated Cheese Division	BR
21	Mainland Products Ltd.	BR
1450	Matatoki Farm Cheese	
90	Mercer Cheese Ltd.	
95	Meyer Gouda Cheeses	
705 (H & P)	Milligans Food Group	
530	South Island Beverages Plant (NZDF)	
4 (H & P)	Puhoi Valley Cheese Co. Ltd. (Div. of NZDF)	
785	Talbot Forest Cheese Ltd.	
34 (P)	Tatua Co-operative Dairy Co. Ltd.	BR, EU
51	Waimata Cheese	
60	White Stone Cheese Ltd.	
750	Zany Zeus	

BR=Brazil EU=European Union SL=Sri Lanka CR=Costa Rica PA=Panama NC=Nicaragua
(H & P) = hard cheese and processed cheese manufactured)
(P) = processed cheese

AC Neilson retail purchase statistics from supermarkets in 1996 list "natural cheese" (mainly cheddar and edam) as the highest category by weight at 845,606 kg sold, followed by processed cheese at 18,701 kg.

3.3.2   Imported foods

Imported cheese is reported to make up 15% of the cheese consumed in New Zealand (MAF, 2003).

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 came from Australia (82%), followed by Denmark (7%), France (3%) and Italy (2.2%). It is uncertain what proportion of this is low moisture cheese, as the bulk of the cheese imports are classified as 'cheese, (other than in tins, not grated, powdered or processed), not elsewhere specified'. 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 low moisture cheese are comparable in terms of microbial safety.

About 10% of cheese imports list New Zealand as the country of origin and this probably relates to cheese being re-imported after processing.

Border surveillance exists in New Zealand for high risk foods. For cheese this means all soft cheese and grated/powdered cheese including low moisture cheese such as Cheddar, Colby, Cheshire, Egmont and Gouda, principally because of the possibility of *L. monocytogenes* contamination. The cheeses are monitored by sampling and testing for the organism and sampling regimes are outlined on the following website: http://www.nzfsa.govt.nz/imported-food/high-risk/01softcheesenf.htm. A nil tolerance for *L. monocytogenes* per 25g is the criterion 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 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 cheddar cheese, the fat micelles can protect the pathogens against human gastric acids (D'Aoust, 1985).

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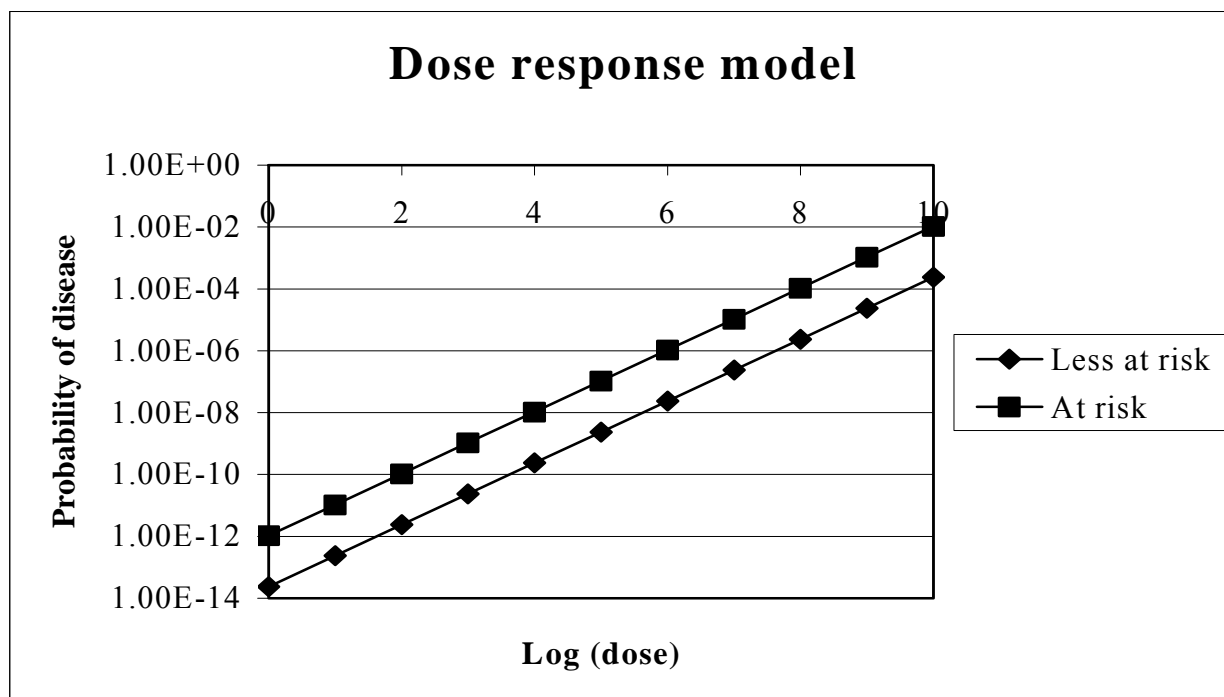
It is becoming increasingly realised that the only completely safe dose of *L. monocytogenes* is zero, even in healthy people. However the probability of invasive disease following exposure to even moderate levels of cells is very low.

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 which means that there is a probability of illness associated with each cell consumed. It is therefore total consumption of cells that dictates risk; there is no "infectious dose", and there is no difference to risk if a small number of cells are eaten frequently or many cells eaten at the same time as long as the total eaten is the same. Figure 3 shows dose response curves for at risk and not at risk groups.

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

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

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 subtype. 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 make to human disease. However, these lineages are not based on serotyping. The conservative approach is to treat all isolates as potentially capable of causing disease.

4.3.2   Febrile gastroenteritis

Dose response data for febrile gastroenteritis are limited. In a New Zealand outbreak involving ham, 21 of 24 (87.5%) people consuming the food contaminated with 1.8×10^7 *L. monocytogenes* cells/g became ill with symptoms of febrile gastroenteritis (Sim *et al*., 2002). Assuming approximately 100g of ham were 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 2.9×10^{11} cfu. In other outbreaks it is difficult to estimate dose responses as portion sizes are not detailed or the number of cells present not accurately known. However, of all of the other outbreaks, the lowest number in food that has been shown to cause febrile non-invasive listeriosis is 1.9×10^5 cfu g^{-1} (Miettinen *et al*., 1999), although the serving sizes were not detailed. In this incident all five people eating the contaminated fish became ill with gastroenteritis, nausea, abdominal cramps and diarrhoea. Therefore consumption of more than, perhaps, 10^7 cells appears to be sufficient to cause *L. monocytogenes* febrile gastroenteritis at a high infection rate in some circumstances. It is possible that foods contaminated with lower numbers of *L. monocytogenes* may also cause febrile non-invasive gastrointestinal disease, and because this organism is not routinely screened for in clinical laboratories many cases of non-invasive listeriosis may evade detection.

4.4   High Risk Groups in the New Zealand Population

Although all healthy individuals may become infected by *L. monocytogenes*, there are some higher risk groups in the population (Sutherland and Porritt, 1997, Schuchat *et al.,* 1991). 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.

In the Schuchat study, 69% of listeriosis cases in the USA population occurred in men and non-pregnant women who were; cancer patients, had AIDS, were organ transplant recipients or who were receiving corticosteroid therapy.

The following sections provide information on the New Zealand population of these high risk 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 found to be 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 Low Moisture Cheese and Human Cases

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}.

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" (moisture content not given, assumption 39% to 50% moisture) cheese, while serotype 1/2a was more common in soft cheeses.

Malak *et al*. (2001) carried out typing of *L. monocytogenes* from cheese and patients with listeriosis in Belgium by randomly amplified polymorphic DNA (RAPD) analysis. This method allowed subdivision of serovar clusters and indicated that the population of strains of *L. monocytogenes* found in cheese differed from the strains isolated from patients with listeriosis during the same period.

In New Zealand the clinical isolates of *L. monocytogenes* for the period 1999 to 2003 are approximately evenly split between serotypes the 1/2 and 4 (Pat Short, ESR Enteric Reference Laboratory, Kenepuru Science Centre, pers. comm., December 2003).

While there may be differences in the distribution of isolate types from low moisture cheeses and from human cases, data are limited. Since the relationship between type and pathogenicity is complex, the conservative position that all *L. monocytogenes* isolates are equally likely to cause disease in humans is generally adopted.

5   EXPOSURE ASSESSMENT

5.1   The Hazard in the New Zealand Food Supply: *Listeria* in Low Moisture Cheeses

Whyte and Wong (2002) analysed 300 samples of retail packed grated low moisture cheeses for *L. monocytogenes*. All samples were negative. It was concluded that these results reflect the correct application of critical control points in cheese manufacture and the advent of critical hygiene areas dedicated to cheese grating activity and packaging. It is assumed all cheese analysed was pasteurised.

During 2003/2004 ESR carried out a survey of 307 soft (>50% moisture) and semi-soft (39% to 50% moisture level) cheeses from a wide range of small and large manufacturers for *L. monocytogenes*. Approximately 50 samples were of semi-soft blue cheese, and would fall into the category covered by this Risk Profile. Results from this survey found that no soft or semi-soft cheese samples tested positive for *L. monocytogenes*. In one blue semi-soft cheese sample, *L welshimeri* was detected (Wilson, 2004).

5.2   Food Consumption: Low Moisture Cheeses

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 Low moisture 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
Colby/Mild/Tasty	9	26	57	8
Specialty	62	33	5	0

The last three categories (Edam/Gouda, Colby/Mild/Tasty, and Specialty) cover the most common low moisture cheese types available in New Zealand. Women are more likely to consume Edam/Gouda or specialty cheeses than men. Men are as likely to consume Colby/Mild/Tasty (cheddar) cheeses as women. Maori and Pacific Islanders are less likely to consume low moisture cheeses than European and other New Zealanders.

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 low moisture 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)
Cheddar (Mild/Tasty)	37.9	38.4	6.35
Colby	17.0	19.1	3.16
Edam	15.2	16.5	2.72
Gouda	0.3	0.5	0.08
Parmesan	2.6	1.0	0.16
Processed	7.2	6.7	1.10
Other low moisture cheeses	1.3	0.9	0.15
Total (low moisture cheese)	**81.5**	**83.1**	**13.72**

Cheese purchasing data from the United Kingdom suggests a similar proportion of total cheese consumption (70-80%) would be of low moisture varieties, with cheddar making up almost 60% of all cheese purchased (http://www.milk.co.uk). It is likely that Colby cheese will be included in the UK estimates of cheddar cheese consumption. Approximately 40% of all cheese servings are associated with meals in which the cheese is likely to be heat-treated.

5.3   Qualitative Estimate of Exposure

5.3.1   Number of servings of low moisture cheese and serving size

5.3.1.1 Total population

From the National Nutrition Survey (NSS), 2120 individual dietary records were deemed to represent consumption of a serving of low moisture 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) $= 2120 \times 4,054,200/4636 \times 365$
                                                   $= 6.76 \times 10^8$ servings

This can be compared with the calculated number of servings of low moisture cheese for the US population of 2.28×10^{10} servings (processed cheese 1.2×10^{10}, hard cheese 9.0×10^9 and semi-soft cheese 1.8×10^9). Based on a total population of 293,494,282 at 14 June 2004 (http://www.census.gov/cgi-bin/popclock). These figures produce quite different results for the number of servings per person per annum of 78 (US) and 167 (NZ).

5.3.1.2 Elderly population

From the NNS, 450 individual dietary records were deemed to represent consumption of a serving of low moisture 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) $= 450 \times 615,580 /1087 \times 365$
                                                    $= 9.30 \times 10^7$ 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)   = 5.75 x 10^8 x 0.0277 x 0.25
                                                    = 3.98 x 10^6 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)   = 5.75 x 10^8 servings

Based on the data in the NNS database the 50, 75, 95, and 99th percentile serving sizes for low moisture cheese in New Zealand compared to the US serving sizes for the same percentiles were;

Percentile	NZ Serving size (g)	US Serving size (g)*
50	21	21 to 28
75	38	38 to 57
95	84	84 to 142
99	150	122 to 227

* The US data for low moisture cheese were produced from the categories; processed cheese, hard cheese and semi-soft cheese.

These figures suggest that a median New Zealand serving size is comparable to the US equivalent.

5.3.2   Contamination frequency

Listeria monocytogenes has not been detected in any of the approximately 350 samples of low moisture cheese tested in New Zealand by ESR. However, there have been recalls of low moisture cheeses. In November 2001 and May 2002 Mainland recalled several varieties of cheeses, including processed cheese, for *L. monocytogenes* contamination.

Overseas data (Table 5) suggest that, in general, the prevalence of *L. monocytogenes* in low moisture cheese is low. The studies from which the data are derived often do not specify whether the cheese is made from pasteurised or raw milk, and the small number of studies that do makes it impossible to draw conclusions concerning prevalence and the type of milk used.

5.3.3   Predicted contamination level at retail

In New Zealand, no data are available concerning *L. monocytogenes* contamination levels of low moisture cheeses at retail as there have been no isolations of *L. monocytogenes* from this food group at the retail level.

5.3.4   Growth rate during storage and most likely storage time

In general, once ripened, low moisture cheeses have a long shelf life (weeks to months). In the FDA/FSIS (2003:Appendix 5) risk assessment, exponential growth rates have been

modelled, taking data from levels of contamination at retail and post retail growth. The most likely storage times and maximum times that consumers store cheese has been evaluated as follows;

The most likely storage time for hard cheese was 6 to 10 days and the maximum was 90 to 180 days. For semi-soft cheese (moisture 39-50%), the most likely storage time was 6 to 10 days and maximum storage time was 15 to 45 days. Processed cheese was 6 to 10 days and 45 to 90 days respectively.

Growth of *L. monocytogenes* in low moisture cheeses during storage is unlikely. Out of 21 data sets described above; 18 data indicate a decline, one survival and two indicate growth. Where growth occurred it was slow: <1 $\log_{10}$ in 20 weeks in Tilsiter, Trappist, Havarti and Limburger and 1.6 $\log_{10}$ in 6 weeks in Stilton.

In general, *L. monocytogenes* can grow during the manufacturing process, but the subsequent ripening process reduced the numbers present. Exceptions are those cheeses which are mould ripened, such as blue cheeses, where the rising pH occurring during ripening can provide conditions in which the organism can survive or grow.

5.3.5   Culinary heat treatment

Many of the low moisture cheese servings (approximately 40% according to the NNS data) are subjected to heat treatment i.e. cooking, before consumption. The low moisture variety of Mozzarella used extensively in the pizza industry would fall into this category.

5.3.6   Exposure summary

Consumption of low moisture cheese types represents the majority of cheese consumption in New Zealand.

Contamination is infrequent, based on limited existing data. Growth is unlikely in most cheese types due to a variety of listeriostatic factors (low water activity, competing microflora, acidification from the use of starter culture).

5.4   Overseas Context

Information from the scientific literature on the prevalence of *L. monocytogenes* in low moisture cheese overseas has been summarised in Table 6.

Table 6:   Overseas prevalence and quantitative data for *L. monocytogenes* in low moisture cheese

Country	Type of milk	Food	No. samples tested	No. (%) positive for *L. monocytogenes*	Reference
Chile	Unknown	Hard cheese (domestically produced)	155	0	(Cordano and Rocourt 2001)

Country	Type of milk	Food	No. samples tested	No. (%) positive for *L. monocytogenes*	Reference
England and Wales	Contaminated sample prepared from raw milk, other 65 samples unknown milk status	Cows milk hard cheese (UK and imported)	66	1 (1.5) (<500/g)	(Greenwood *et al.,* 1991)
Europe (cheeses from 6 countries)	NB Soft, semi-soft and hard cheese information collated so can not differentiate Cow, goat and ewe milk	Semi-soft red smear cheese Austria Denmark France Germany Italy Switzerland Hard red smear cheese Austria Denmark France Germany Italy Switzerland German cheese:	92 4 1 25 42 14 6 45 2 - 1 26 4 12	7 (7.6) 4 R, 3 P 0- 0- 0- 4 (9.5) 3 (21.4) 0- 5 <10cfu/cm^2 1 2x10^{2}cfu/cm^2 1 8x10^{2}cfu/cm^2 2 (4.4) 0- - 0- 1 (3.8) (P) 1 0 German cheese: <10cfu/cm^2	(Rudolf and Scherer 2001)
France	Unknown	"other" (i.e. not soft cheese), moisture content unknown French cheese sampled during 1986/87	135	0	(Ryser, 1999 b)
Germany	Unknown	Imported and domestic sampled 1987/1988: NB. Semi-soft and semi-hard not defined, so. < 50% moisture assumed. Semi-soft /cows' milk Semi-soft/ ewes' milk "Semi-hard" /cows' milk Hard /cows' milk	 144 6 22 4	 19 (13.2) 0 0 0	(Ryser, 1999 b)
Hungary	Cheeses "produced by different technologies"	Domestically produced Semi-soft cheese (assumed 39 to 50%) Hard cheese Processed cheese	25 10 20	0 0 0	(Rodler and Korbler, 1989)

Country	Type of milk	Food	No. samples tested	No. (%) positive for *L. monocytogenes*	Reference
Ireland		Semi soft cheeses (moisture content not given)	17	0	(Coveney *et al.*, 1994)
Italy	"manufactured with raw or pasteurised milk"	Sampled between 1986/1990; "Semi-hard" (assumption 39% to 50%) Hard	118 99	0 (4.2% +ve for *L. innocua)* 0	(Comi *et al.*, 1990)
Korea	Pasteurised	Imported Processed Cheese (unknown moisture content)	45	0	(Baek *et al.* 2000)
Spain	Pasteurised	Processed cheese	3	0	(Rota *et al.* 1992)
Spain	Raw milk (goat "occasionally mixed with cow milk")	Valdeón Blue- a local Spanish cheese	11 (interior samples)	0	(Lopez-Diaz *et al.,* 1995)
Spain (Asturias)	All pasteurised milk.	Regional cheeses sampled from 1992/1993: Moisture Afuega 'l Pitu 49% Beyos 41% Penamellara 40% Oscos 36% Vidiago 45%	 17 17 34 17 16	 7 (41.1) 0 1 (2.9) 0 1 (6.3)	(Margolles *et al.,* 1996)
Spain (Tenerife)	Raw goat's milk	Locally produced cheese 40-47.3% moisture, most consumed fresh, some ripened 30 to 60 days	3	2 (66.7)	(Perez *et al.,* 1998)

Country	Type of milk	Food	No. samples tested	No. (%) positive for *L. monocytogenes*	Reference
Sweden	9% made with raw milk 31/333. Sampling between 1989 and 1993	Locally produced and imported. White moulds (most likely soft cheeses) Green/blue moulds White-green/blue moulds Smear surface Without moulds/smear	333 154 95 27 26 31 total domestic. 27 import. 306	20 (6%) 15 (9.7) 0 1(3.7) 4 (15.4) 0 0 20 (6.5) (18 positives from France, 15 were white mould therefore possibly soft cheeses, leaving 3 smear cheeses)	(Loncarevic *et al.,* 1995)
Switzerland	Unknown	Swiss cheeses Hard cheeses Semi soft cheeses (moisture content not given, assumed 39 to 50%) Surface ripened (possibly smear but not clear in text)	88 205 343	0 4 (2.0) 33 (9.6)	(Breer and Schopfer 1988)
USA	Raw milk	"domestic" manufactured but footnote states that this includes imported cheese Blue, Brick, Cheddar, Edam, Colby, Goat, Gouda, Monterey Jack, Swiss, other	181	1 (0.6)	(Ryser, 1999 b)
USA	Unknown	Retail Blue-veined cheese Unspecified whether domestically produced or imported	1,623	23 (1.42) 18 0.04-0.1/g, 3 >0.1-1/g, 1 >1-10/g, 1>10-100/g	(Gombas *et al.* 2003)

The study of Loncarevic *et al*. (1995) classes cheese according to ripening category rather than moisture content. White mould-ripened cheeses are most likely to be soft cheeses and therefore not within the scope of this profile but results are reported because the other categories may include low moisture cheese. The study concludes that cheeses made from raw milk were more frequently contaminated than those made from heat treated milk and the prevalence of the organism in whole cheeses and pre-cut wedges was similar. Isolates from two of the French smear cheeses were serogroup 4 while the remaining isolates were all serogroup 1. In contrast, Breer and Schopfer (1988) found no differences in contamination between pasteurised and raw milk cheeses and also that colonisation by *Listeria* spp. was restricted to external surfaces only. Again mould type categories are given in this study that are difficult to categorise in relation to their moisture content.

The data presented in the table above are difficult to summarise because of the heterogeneity of low moisture cheeses, as well as the diversity of other factors discussed above. In addition, for some studies, data include soft cheeses that are difficult to differentiate. However, contamination is, generally, low at around 1-2%. A brief analysis of the data in Table 5 for data where the cheese type is unequivocal indicates that 1/313 (0.3%) of hard cheese samples were positive for *L. monocytogenes*, 23/415 (5.5%) of semi-soft/semi-hard cheeses were positive, and 46/506 (9.0%) of smear cheeses were positive. This is in accordance with the data from survival/growth studies

The numbers of pathogens present are also, generally, low although there are few data available concerning this.

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 report. 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 2003 are given in Table 7. It is important to note that these cases are not associated with any specific transmission vehicle.

Table 7:   Reported cases of invasive listeriosis and mortality from 1990 to 2004

Year	Listeriosis cases	Deaths (perinatal)	Deaths (non-perinatal)	Reference
1990	16	2	NA	Kieft *et al.*, 2000
1991	26	1	NA	Kieft *et al.*, 2000
1992	16	0	NA	Kieft *et al.*, 2000
1993	11	2	NA	Kieft *et al.*, 2000
1994	8	0	NA	Kieft *et al.*, 2000
1995	13	1	0	Kieft *et al.*, 2000
1996	10	1	0	Kieft *et al.*, 2000
1997	35	6	2	Kieft *et al.*, 2000
1998	17	0	0	Kieft *et al.*, 2000
1999	19	2	1	Kieft *et al.*, 2000
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

NA = Not Available

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
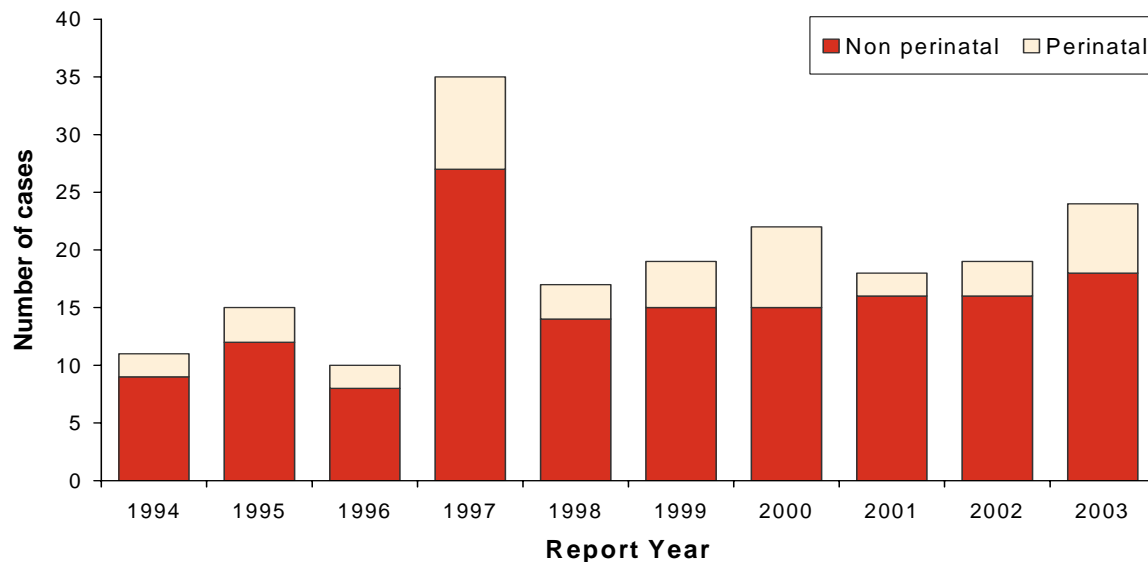
Reproduced from ESR (2004)

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 8. 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 8:   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. . If the laboratory investigation identifies a known foodborne pathogen in the suspect food at levels sufficient to cause illness, and the symptoms 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 1997/98 financial year to 2002/03 were reviewed. In this period, consumption of cheese was investigated in relation to 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 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*. There was insufficient information to determine whether the implicated cheese was low moisture or not.

One earlier investigation was identified in which *L. monocytogenes* (serotype 1/2) was isolated from Edam cheese taken from the refrigerator of a listeriosis case. It is uncertain from the case documentation whether the cheese was the source of the infection.

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 (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 9 indicate that New Zealand's rate is similar to that of other developed countries.

Table 9: **Comparison of listeriosis incidence rates between New Zealand and overseas**

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	2002	0.3	OzFoodNet Working Group, 2003
Canada	1990-1998	0.1-0.3	Health Canada, 2000
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	2002	0.3	Anonymous, 2003
USA	2003	0.33	Centers for Disease Control, 2004

* provisional data

6.2.2   Contributions to outbreaks and incidents

As shown by the data in Table 10 outbreaks of *L. monocytogenes* infections comprise only a small proportion of total outbreaks. In a review of foodborne disease outbreaks in England and Wales in 1992-1993, Cowden *et al.* (1995) did not identify any outbreaks due to *L. monocytogenes* infection.

Table 10: **Contribution of *L. monocytogenes* to foodborne disease outbreaks and incidents overseas**

Country	Year	No. (%) Outbreaks	No. (%) incidents or cases	Reference
Australia	1995-200	5 (2.0)	41 (<1.0) cases	Dalton *et al.*, 2004
Canada	1981	NS	1 (0.2) incidents 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

A very limited number of outbreaks of listeriosis associated with low moisture cheeses have been reported in the literature (Table 11). In both reported outbreaks the cheese implicated included types that might be expected to support growth of *L. monocytogenes*; semi-soft cheese (Gaulin *et al.,* 2003) and blue cheese (Jensen *et al.,* 1994).

Table 11:   Overseas outbreaks of listeriosis where low moisture cheese was the implicated vehicle

Country	Year	No. Cases	Cheese Type	Odds ratio	Reference
Canada	2003	17	Four types of cheese, both soft and "semi-hard" (moisture content not given, assumption 39% to 50% for "semi-hard") all 4 types contaminated. Heat-treated (but not pasteurised)	NS	Gaulin *et al.,* 2003
Denmark	1989-90	26 (6 deaths)	Brand X Blue Cheese. Milk status unknown	4.6	Jensen *et al.,* 1994

De Buyser *et al*., (2001) reviewed four sporadic cases of listeriosis implicating milk products. Low moisture cheeses were not implicated in any of these cases.

6.2.3   Case-control studies

Only a single case-control study was identified that implicated low moisture cheese as a cause of listeriosis (Table 12). The implicated cheese was a blue mould cheese. As discussed in section 3.2, the process of mould formation is often associated with an increase in the pH of the cheese to a level that will support microbial survival or growth.

Table 12:   Case control studies containing information on *Listeria* in low moisture cheese

Country	Risk factor	Odds ratio (CI)	Reference
Denmark[*]	Five different Danish Blue-mould cheeses	2.9	Jensen *et al.,* 1994
	One of above brands (X) blue mould cheese	4.6 (1.0-2.1) p<0.1	

CI = confidence interval
[*]Associated with the outbreak in Table 10 above.

6.2.4   Risk assessments

A number of risk assessments have now been published concerning *L. monocytogenes.*

Two draft risk assessments for *L. monocytogenes* in ready-to-eat foods have been produced, one by the FDA/FSIS in January 2001, updated September 2003 (FDA/FSIS, 2003) (http://www.foodsafety.gov/~dms/lmr2-toc.html) and the other by FAO/WHO (http://www.who.int/fsf/mbriskassess/Scientific_documents/mra001.pdf).

After the most recent round of revisions the FAO/WHO model has combined aspects of the FDA/FSIS one and almost merged the two. While the FAO/WHO report has recently been

released (FAO/WHO 2004) it does not specifically deal with cheese as a food. Therefore it is not relevant to this Risk Profile.

It should be noted that the FDA/FSIS model is very much a North American risk assessment and so used an exposure assessment which is particular to that part of the world (even though data from anywhere in the world were used to calculate prevalences in food). We might assume that the hazard 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 relative risks predicted for the various ready-to-eat food categories in the FDA/FSIS risk assessment are given in Table 13 (from http://vm.cfsan.fda.gov/~dms/lmrisk1.html). These risk rankings are quite consistent with results from case control studies.

Low moisture cheeses were general ranked quite low with respect to their risk of causing listeriosis, with hard cheese <39% moisture, the lowest ranked of the 23 foods considered, while processed cheese was ranked 20 and semi-soft cheese 39-50% moisture ranked 16. This was attributed to;
- The small amount consumed,
- Low contamination level at retail,
- Little, if any, growth during storage, despite long storage times,
- Moderate annual number of servings, and
- Hard cheeses typically have a high salt content.

Table 13:   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 to 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

Food Categories[a]	Sub-Population			
	Intermediate Age[b]	Elderly[b]	Perinatal[b]	Total [b,c]
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)

6.3   Qualitative Estimate of Risk

The information summarised above leads to the conclusion that the risk of transmission of *L. monocytogenes* by low moisture cheeses in New Zealand is low for the general population (although the risk will be higher for susceptible populations). Evidence for this conclusion comes from:

- New Zealand surveys indicating a very low prevalence of contamination in semi-soft and grated low moisture cheeses;
- low prevalence of *L. monocytogenes* in low moisture cheeses overseas, and when contamination does occur, cell numbers are low;
- rarity of linkages between the presence of *L. monocytogenes* in low moisture cheeses and clinical cases, in outbreak investigations and case-control studies overseas;
- the properties of many types of low moisture cheese which result in the inactivation of *L. monocytogenes* during ripening, and
- the very low values for R in the dose response model, even for "at risk" groups, which means that at low cell numbers, the probability of infection is very low.

The heterogeneous nature of this food group requires some qualification of this conclusion. There is some evidence for increased risk of *L. monocytogenes* growth in semi-soft cheeses which have been mould-ripened or bacterial smear ripened, causing the pH to rise. However, hard and very hard non-mould cheese makes up the majority of low moisture cheese consumed in New Zealand.

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 low moisture 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 low moisture 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 number of illnesses will be obtained by setting a food safety objective 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.

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 *Listeria monocytogenes* in low moisture 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 low moisture cheese 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 (superceding 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 plan 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, pers. comm., 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).

The term "Cheese treatment" is defined under Clause 3 (d) of the Standard as

(i)   The rapid heating of milk or a milk product to be used in the manufacture of cheese to a temperature of not less than 64.5°C retaining it at that temperature for not less than 16 seconds (thermisation); and

(ii)   Storing the cheese prior to sale at a temperature of not less than 7°C for not less than 90 days from the date of commencement of manufacture.

In addition, the cheese shall be stored prior to sale at a temperature of not less than 2°C for a period of not less than 90 days from the date of commencement of manufacture.

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/draftproposedguide li1507.cfm).

There have been two such assessments to date:

- A357 Swiss raw milk cheeses (ANZFA, 1998); and,
- P263 Raw milk very hard cooked-curd cheeses (FSANZ, 2002; FSANZ, 2003).

A proposal from the Swiss Federal Veterinary Office in 1998 requested permission to use raw milk for the manufacture of several hard and "semi-hard" (39-50% moisture content) cheeses.

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 and domestic production within New Zealand. In effect this means that Emmental, Gruyère, Sbrinz and Parmesan style cheese can be imported or produced in New Zealand by pasteurisation, cheese treatment or FSANZ approved methods from raw milk.

The evaluation regarding *Listeria* in the processing steps of Swiss Hard Raw Milk Cheeses considered the following steps and their effect on *L. monocytogenes*;

- Raw milk/storage: The organism is psychrotrophic and may grow if present,
- Acidification: Inhibits growth,
- Curd treatment: Results in a 2 to 7 $\log_{10}$ reduction,
- Brining: Inhibits growth and decreased populations,
- Ripening*: "the environment in the product is not conducive to the survival or proliferation of pathogenic micro-organisms", and
- Overall reduction: >5 $\log_{10}$.

*The cheeses are stored for very long periods (for at least 90 days up to 360 days).

ANZFA's conclusions regarding Swiss "semi hard" (39% to 50%) cheeses under the assessment were that they could only be supplied to the market when made from thermised milk (or pasteurised milk). This would apply to the import of Tilsiter, Appenzeller and Vacherin Fribourgeois. One "semi-hard" cheese, Tete de Moine, was not accepted, as it is always made from raw milk and the process of manufacture does not provide an equivalent level of safety. This decision follows the FSANZ rejection of the Australian Specialist Cheese Maker's 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.

A second assessment, applicable to Australia only, concerned Proposal P263 – Safety assessment of raw milk very hard (Parmesan style) cooked-curd cheeses. This was to determine whether an amendment of the Code (Standard 1.6.2 –applicable in Australia only) should be made to permit continued import and sales of these cheeses (eg. Parmigiano, Reggiano, Grana Padano).

Scientific evaluation found that very hard cheese manufacturing processes in general could achieve a 5 log reduction of bacterial pathogens. The Final Assessment Report recommended an amendment to Standard 1.6.2 of the Code to exempt very hard cheeses from heat treatment requirements provided they undergo curd heating to at least 48°C. This applies to cheeses of <36% moisture content that have been matured/stored for at least 6 months at no less than 10°C. (It was agreed that Cheddar cheese did not fit the definition outlined in the variation to Standard 1.6.2 because cheddar has a low curd cooking temperature of around 39°C). Attachment 2 of the full report contains the scientific evaluation of the safety of very hard cheeses produced from raw milk. In summary, the evaluation (determined from assessments) considered the following steps and their effect on *L. monocytogenes*;

- Raw milk/storage: The organism is psychrotrophic and may grow if present,
- Acidification: No growth occurs,
- Curd treatment: 4.6 $\log_{10}$ reduction,
- Ripening: 5-$\log_{10}$ reduction, and
- Overall reduction: >5-$\log_{10}$.

The 48°C curd cook step was evaluated as having a "low impact" on *Listeria monocytogenes*, the low moisture of cheese to have "some impact", maturation time (6 months) to have "high impact" and the final product risk to be "very low" (FSANZ 2003).

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 14. Failure to comply 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 sample units).

Table 14:   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**	**5**	**0**	**0**	
	Salmonella/25 g	5	0	0	
All raw milk cheese (cheese made from milk not pasteurised or thermised)	*Listeria monocytogenes*/**25 g**	**5**	**0**	**0**	
	Salmonella/25 g	5	0	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 for *L. monocytogenes* in ready-to-eat (RTE) foods, which includes low moisture 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. Cheeses and cheese processing plants, other than cottage cheese, do not appear to have been examined under this initiative. There was a clear correlation between *Listeria* in the plant environment and in the final product.

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 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. 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 a paper (Chen *et al.,* 2003). Since the organism cannot be eliminated from the environment or from all food products despite extensive control measures, the authors 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* (i.e. <100/g) pose very little risk, and that eliminating foods containing higher concentrations would 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 grow to high levels rather than all RTE foods is suggested. Comparisons with countries that operate such a strategy (e.g. Canada and several European countries) show that rates of listeriosis are not noticeably different from those countries with a zero tolerance policy. This approach appears to be in line with that put forward by the ICMSF for the standard in internationally traded foods (section 7.1).

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 25 g (consisting of 5 samples of 5 g taken from different parts of the same product). For hard cheese, *L. monocytogenes* should be absent from 1 g samples. These parameters are based on time of removal from the processing establishment and do not reflect the quality expected at point of sale or consumption.

The 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 the member state's judicial system and in cases of intra-Community trade. An example of this is Germany, Netherlands and France, who have a tolerable level (<100 cfu/g) at the point of consumption. Italy, like the USA, has a zero tolerance (absence of *L. monocytogenes* in 25 g of food). Denmark, like Canada, has a tolerance of below 100 cfu/g for some foods and a zero tolerance for others (especially foods that support growth and have extended shelf lives) (sections 7.3.6 and 7.3.7).

The European Commission set up a Scientific Committee on Veterinary Measures related to Public Health on *Listeria monocytogenes* 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 100 cfu/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.

- (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, immuno-compromised) which represent a considerable and growing section of the total population."(Anonymous 1999:27).

The Commission report also recommends the grouping of ready-to-eat foods on the basis of the control potential for *L. monocytogenes*. The suggested groupings and the proposed limits for *L. monocytogenes* are given in Table 15.

Table 15: Grouping of ready-to-eat foods relative to the control potential for *L. monocytogenes* and suggested limits

Group	Description	Limit for *L. monocytogenes*
A	Foods heat-treated to listericidal level in the final package	Not detected in 25 g at the time of production
B	Heat-treated products that are handled after heat treatment. The products support growth of *L. monocytogenes* during the shelf life at the stipulated storage temperature	Not detected in 25 g at the time of production
C	Lightly preserved products, not heat-treated. The products support growth of *L. monocytogenes* during the shelf life at the stipulated storage temperature	Not detected in 25 g at the time of production
D	Heat-treated products that are handled after heat treatment. The products are stabilized against the growth of *L. monocytogenes* during the shelf life at the stipulated storage temperature	<100 cfu/g at the time of consumption
E	Lightly preserved products, not heat-treated. The products are stabilized against the growth of *L. monocytogenes* during the shelf life at the stipulated storage temperature	<100 cfu/g at the time of consumption
F	Raw ready-to-eat foods	<100 cfu/g at the time of consumption

Low moisture cheese is most likely to be considered in group D.

7.3.3 Engand and Wales

In the United Kingdom, the statute law; the Food Safety Act 1990 Sections 7, 8, and 14, provides the legal framework for dealing with the microbial quality of food. No cases have been taken under Section 7, which is 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 for cases of unfit food. 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 Environmental Health Officers (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 and 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 i.e. 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 *L. monocytogenes* are summarised in Table 16.

Table 16: **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/ 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 10^2CFU/g at point of sale/consumption would represent a potential risk to health.

It is also noted that *Listeria* spp. (total) are listed under 'Indicator organisms' where on occasions "some strains may be pathogenic".

7.3.4   The Specialist Cheesemakers Association in the United Kingdom 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 milk cheese in the United Kingdom. A political lobby group, with H.R.H. The Prince of Wales as patron, promotes the interests of members to government and the media. The association defines itself as supplying to a market demanding flavour and character from cheese. These characteristics are, according to the lobby group, usually derived from cheeses 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).

7.3.5   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

However ANZFA made a decision to reject an application (A270) from ASCA requesting a variation to the Food Standards Code to permit the use of non-heated milk for hard dry and soft moist specialty cheeses.

7.3.6   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 tolerances as summarised (Table 17).

Table 17:   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*
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 100 cfu/g of *Listeria monocytogenes* is regarded as posing a health risk to consumers (Food Act s.12), control activities include prohibition of sale and recalls.

7.3.7   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 18.

Table 18:   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, 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.

Category	Foods	Microbiological criteria For *L. monocytogenes*	Action level
3 (two types of foods)	• supports growth with refrigerated shelf-life of <10 days	≤100 cfu/g with adequate GMP	Immediate action- allow sale. Follow up action- follow up at plant level.
	• all other ready-to-eat foods not supporting growth ➢ pH 5.0 – 5.5 and a_w < 0.95 ➢ pH <5.0 regardless of a_w ➢ a_w ≤0.92 regardless of pH ➢ frozen foods.	≤100 cfu/g with inadequate or no GMP	Immediate action- consider class II recall or stop sale. Follow up action-follow up at plant level.
		>100 cfu/g	Class II recall or stop sale. Follow up action- follow up at plant level.

Low moisture cheeses would fall into category 3.

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

Possible sources of *L. monocytogenes* contamination are;

- Use of contaminated thermised or raw milk (for cheeses produced by the cheese treatment/FSANZ approved methods),
- Ingredients added after pasteurisation of milk, and
- Environmental contamination.

Environmental contamination is relevant to all low moisture cheese production and is examined in more detail in this section.

Breer and Schopfer (1988) reported that *L. monocytogenes* contamination of Swiss cheeses was restricted to the outer surfaces, suggesting external contamination of the cheese (possibly during ripening), rather than contaminated ingredients, (see section 5.4, table 5). In addition to sampling cheeses, the authors also sampled production items during different types of cheese production. *Listeria monocytogenes* was detected in 9.0% of smear brine samples, 19% of shelves, 25% of brushes and in none of starting/intermediary materials (water, rennet, starter cultures, raw milk, pasteurised milk, curd whey, cheese before salting and brine). 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*. Product testing showed that when *L. monocytogenes* was detected in product it had been concurrently detected in the environment, or was detected 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 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 authors interpreted these results to mean that cheese contamination was largely occurring during the cheese-ripening process. Environmental samples analysed in the study were 5.4 times likely to yield *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.

Post-production cheese handling leading to *Listeria* contamination has been reported via display surfaces (Kerr *et al*., 1996), wooden shelves used during ripening and brine brushes (Ryser 1999a). Smear cheeses may be more prone to post-production contamination because of the extra handling they receive.

A year long survey of two Northern Ireland milk processing plants for *L. monocytogenes* found extensive environmental contamination (Kells and Gilmour, 2004). The overall incidence of *Listeria* on equipment was 18.8% (6.3% *L. monocytogenes*), in the environment was 54.7% (40.6% *L. monocytogenes*) and in raw milk 44.4% (22.2% *L. monocytogenes*). On one occasion, *L. welshimeri* was isolated from pasteurised milk, probably demonstrating post-pasteurisation contamination of product. The main environmental sources of L. monocytogenes were considered to be a floor drain and stainless steel steps.

7.6 Risk Management Options

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, competing microflora and whether pasteurised or raw milk was used. Intrinsic factors pH 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.

The mandatory nature of pasteurisation or cheese treatment (thermisation and aging) for low moisture cheese manufactured or imported into New Zealand means that subsequent contamination from factors such as added ingredients, further processing steps (e.g. grating) and from the environment represent the most likely source of *L. monocytogenes* in heat treated low moisture cheese. This conclusion is based on the assumption that the pasteurisation or cheese treatment steps have been effectively applied.

In New Zealand monitoring for *L. monocytogenes* in diary products 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*. Targeting 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.

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 cheese making 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 3 methods legislated for in New Zealand in relation to milk processing for cheesemaking. These are;

- Pasteurisation,
- 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 10^5cfu/ml). Extrapolation to overseas data could be misleading because of the differences in herd management, year round grazing, use of silage etc.

The ripening or aging period of some low moisture cheeses can reduce the risk due to the potential for reduction over time of the *Listeria* pathogen. This principle was important in allowing the import of raw milk Swiss Emmental, Gruyère and Sbrinz cheese because of the long maturation period of at least 90 days and up to 360 days.

There is evidence to suggest that *L. monocytogenes* contamination of low moisture cheeses is usually greatest on the surface and likely to be due to contamination during ripening, indicating that good hygiene control in ripening facilities is an important control measure. While most studies point to a slow inactivation of *L. monocytogenes* during ripening, the broad definition of this food and, hence, the relatively wide range of moisture levels and ripening protocols means that there may be some circumstances where growth may occur on cheeses with a moisture content less than 50%. Pasteurisation and good environmental hygiene are more important in those low moisture cheeses with 39% to 50% moisture ("semi-soft"), especially mould ripened cheese and smear cheeses where the pH level can rise, permitting growth or survival. This indicates that risk assessment and management needs to be determined on a case-by-case basis.

Advice regarding consumption of "semi-soft" mould ripened and bacterial ripened smear cheeses is a risk management option, which should be linked to the well-categorised risk groups for listeriosis. The NZFSA and the Ministry of Health in New Zealand already use direct educational campaigns targeted towards pregnant women.

8   CONCLUSIONS

8.1   Description of Risks to New Zealand Consumers

8.1.1   Risks associated with low moisture cheese

The rate of reported invasive listeriosis in New Zealand is similar to that found in like countries (Table 8) 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 consumption of low moisture cheese.

The incidence of non-invasive disease from *L. monocytogenes* infection in New Zealand is unknown. It is not normal practice for clinical laboratories to examine faecal specimens from cases of gastrointestinal disease for the presence of *L. monocytogenes* and it might be that more outbreaks will be reported as this form of the disease gains recognition.

L. monocytogenes is able to grow during manufacture of low moisture cheese (see section 3.2), but is generally inactivated slowly during subsequent ripening. However, some exceptions exist, usually related to mould-induced pH increases during ripening (e.g. blue cheese).

The mandatory nature of pasteurisation or equivalent cheese treatment (thermisation and aging) for cheese production in New Zealand means that subsequent contamination from factors such as added ingredients, further processing steps (e.g. grating) and from the environment represent the most likely source of *L. monocytogenes* in low moisture cheese.

Surveys showing the absence of *L. monocytogenes* in New Zealand retail grated low moisture cheese and semi-soft cheeses suggest that contamination rates are low. However, the data available are limited, and grating as just one aspect of commercial post processing handling.

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. However 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.

Overall, the available data indicate that *L. monocytogenes* in low moisture cheese in New Zealand currently does not represent a significant risk to human health.

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 FDA/FSIS (2003) risk assessment listed as high (5 or above) relative risks of listeriosis for the following food groups (Table 12):

1. Delicatessen 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 linked outbreaks have been reported (from the same incident) involving cooked RTE meat products.

Other food vehicles (see "*Listeria* in ready-to-eat meats" and "*Listeria* in soft cheese" Risk Profiles) represent a more likely route of exposure to this organism.

8.1.3 Quantitative risk assessment

A quantitative risk assessment would be feasible for *L. monocytogenes* in low moisture cheese, provided sufficient data on the prevalence of the organism in the product at a retail level could be obtained. However, it is difficult to see how the conclusions would be markedly different to those derived from the assessment conducted by the US FDA/FSIS.

8.2 Commentary on Risk Management Options

The low risk association with *L. monocytogenes* in low moisture cheeses in New Zealand indicates that additional risk management measures are unnecessary.

The majority of low moisture cheeses can be accepted as low risk products due to the very low risk of bacterial growth, even if post pasteurisation contamination has occurred. Post pasteurisation contamination is most likely to introduce only low numbers of cells and the pH and water activity of most low moisture cheeses appears to result in inactivation, rather than growth, of *L. monocytogenes*.

Pasteurisation and environmental hygiene measures are likely to be important for semi-soft cheeses and cheeses where mould-ripening increases the pH to levels that permit growth or survival of *L. monocytogenes*. Consequently the case-by-case assessment process adopted by FSANZ is appropriate.

8.3 Data Gaps

The data gaps identified in this Risk Profile are:

*   Prevalence of *L. monocytogenes* in particular low moisture cheeses sold in New Zealand, specifically in semi-soft, mould-ripened cheeses, and cheese subject to forms of post production handling other than grating,
*   Quantitative data on levels of *L. monocytogenes* in low moisture cheeses when contamination does occur,
*   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,

- Information on environmental *L. monocytogenes* contamination in New Zealand cheese production sites and associated areas,

- Quantitative data on amounts of imported and domestic cheeses produced by either the cheese treatment method or FSANZ approved methods (equivalency to safety levels achieved by pasteurisation controls), and

- Quantitative data on amounts of imported and domestic cheeses produced by either the cheese treatment method or FSANZ approved methods (equivalency to safety levels achieved by pasteurisation controls).

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APPENDIX 1: CATEGORIES FOR RISK PROFILES

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

1. Incidence

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

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

Disease/organism	Food rate (/100,000 population) Calculated for 12 months to June 2001	Food rate (/100,000 population) Calculated for 12 months to December 1998
Campylobacteriosis	1320	2047
Listeriosis	0.4	0.4
VTEC/STEC	1.9	1.4
Salmonellosis	176	230
Yersiniosis	38	62
Shigellosis	7	7
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 Major contributor to foodborne NV
2	10-100	Major contributor to foodborne salmonellosis 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