RISK PROFILE UPDATE: *LISTERIA MONOCYTOGENES* IN CHEESE

Client Report FW13049

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RISK PROFILE:
LISTERIA MONOCYTOGENES
IN CHEESE

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On 1 July 2010, the New Zealand Food Safety Authority (NZFSA) and the Ministry of Agriculture and Forestry (MAF) were amalgamated. On 30 April 2012, MAF was renamed as the Ministry for Primary Industries (MPI).

This Risk Profile uses the names NZFSA and MAF for documents produced during the existence of these organisations.

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GLOSSARY AND ABBREVIATIONS

ANS	The 2009 Adult Nutrition Survey
a_w	Measure of water activity (max = 1.000 = pure distilled water)
ACMSF	Advisory Committee on the Microbiological Safety of Foods
CFU	Colony forming unit
CNS	The 2002 National Children's Nutrition Survey
CrI	Credible Interval
EU	European Union
FSANZ	Food Standards Australia New Zealand
GRAS	Generally recognised as safe
MPI	Ministry for Primary Industries
MPN	Most Probable Number
Neonate	A newborn baby during the first 28 days after birth[1]
NNS	The 1997 National Nutrition Survey
NZFSA	New Zealand Food Safety Authority (now MPI)
PCR	Polymerase Chain Reaction
PFGE	Pulsed Field Gel Electrophoresis
pH	Measure of acidity (min = 0 = most acidic; max = 14)
Perinatal	The period from 20 weeks or more gestation to 7 days after birth[1]
QPS	Qualified presumption of safety
RMP	Risk Management Programme (under the *Animal Products Act* 1999)
RTE	Ready-to-eat
USA	United States of America
USFDA	United States Food and Drug Administration
USDA	United States Department of Agriculture
VBNC	Viable but non-culturable

[1] As defined in Ministry of Health (2010)

SUMMARY

This Risk Profile considers *Listeria monocytogenes* in cheese. This is an update of two Risk Profiles published in 2005, one addressed *L. monocytogenes* in low moisture cheeses and the other *L. monocytogenes* in soft cheeses. The two categories of cheese are now covered in this updated Risk Profile and a revised classification of cheeses has been adopted. Extra-hard cheeses (e.g. parmesan) are excluded from this Risk Profile as these are considered to present a very low risk of causing listeriosis.

The purpose is to critically review new information to answer the following risk management questions:

- What is the public health risk from *L. monocytogenes* in cheese consumed in New Zealand?
- Has the risk of listeriosis from the consumption of cheese in New Zealand changed since the 2005 Risk Profiles were published?

The large number of cheese types and manufacturing processes means that broad statements about risk covering all cheeses are difficult. Instead we consider risk according to contributing factors:

- Ingredients
- Cheese types
- Processes and manufacturing environment
- Consumption

Ingredients

Pasteurisation will eliminate *L. monocytogenes*, but cheeses made from raw milk could be contaminated by milk-borne *L. monocytogenes*. Two surveys of raw cows' milk in New Zealand have found prevalences of *L. monocytogenes* of 0.7% and 4.1% and the concentration of *L. monocytogenes* in positive samples was <1 CFU/mL or <1 MPN/mL. One survey of raw goats' milk found a prevalence of 3.3%. Thus there is a risk that raw milk used for cheese manufacture will be contaminated with *L. monocytogenes*.

Other ingredients may be added to cheese after pasteurisation and potential *L. monocytogenes* contamination of these should be considered as part of any risk management programme.

Cheese types

Overseas outbreaks of listeriosis have been most commonly associated with soft high moisture cheeses, particularly acid or acid/heat coagulated cheeses, i.e. those where curd formation does not use starter cultures, such as the Hispanic soft cheeses. These categories are also considered higher risk by published risk assessments.

Processes and manufacturing environment

Lactic acid concentration/pH and water activity (a_w) appear to be the most important factors affecting whether *L. monocytogenes* will grow during manufacture, ripening and storage. This applies to bacteria within or on the surface of cheese, although pH and moisture can change considerably at the surface during ripening and storage. Achieving a reduction of pH to <5 during fermentation through the use of starter culture is an important control step. Many, but not all, cheeses are inoculated with a starter culture that converts lactose into lactic acid responsible for a reduction in pH.

The rate of any growth or decline of *L. monocytogenes* in cheese once it has been made will be most affected by temperature.

Hygiene in the manufacturing environment is a critical factor in controlling post-pasteurisation contamination. Ripening and storage areas are important potential sources of contamination.

Raw milk cheese manufacturers in New Zealand have to be individually approved by MPI and so their processes will be examined with respect to *L. monocytogenes* growth. This is likely to control risk from locally made raw milk cheeses, and the risk attributed to these processors will also be low at a national level, due to the small number of processors and products.

Consumption

Analysis of nutrition survey data indicates that hard and semi-hard types of cheese (particularly Cheddar) are the most commonly consumed types of cheese in New Zealand. These types will present a lower risk for exposure to *L. monocytogenes* due to low water activity, as will the next most commonly consumed category, cheese with eyes (e.g. Edam, Emmental, Gouda).

After hard/semi-hard cheeses and cheese with eyes, cottage and cream cheese are the next most commonly consumed types. In the absence of low water activity as a control, the acid content of these cheeses is the primary means of preventing growth. If *L. monocytogenes* is present, the numbers will decrease during storage of the final product (Hicks and Lund, 1991; Hudson *et al.*, 2011).

The soft cheeses such as Brie, Camembert and blue are a minor proportion of the total cheese servings, and the consumption of soft cheese does not appear to have changed markedly based on the nutrition survey data. However, the number of specialist cheese manufacturers in New Zealand appears to be increasing, as is the popularity of farmers markets, suggesting that the range and amount of consumption of non-Cheddar cheese is rising.[2] Some of these soft cheeses will allow growth of *L. monocytogenes*.

Changes from the previous Risk Profile

The previous Risk Profiles concluded that the risk of *L. monocytogenes* infection from soft and low moisture cheese in New Zealand was low. Available human health

[2] http://www.newzealandholidaytravel.com/pages/Best-Cheeses-in-New-Zealand accessed 28 May 2014

surveillance data from New Zealand since the previous documents suggests that this situation continues, as there has been only one case and no outbreaks of listeriosis linked to cheese consumption. A high proportion (approximately 80-90%) of cheese consumption in New Zealand is hard, semi-hard, cheese with eyes, and low pH varieties, which present little risk of growth by any contaminating *L. monocytogenes*.

A notable change is the increasing volume of cheese being imported into New Zealand. Some of these cheeses will be soft varieties that present a greater risk of *L. monocytogenes* contamination. Two of three recalls of cheese for *L. monocytogenes* contamination from 2008 – 2014 involved imported cheese.

1 INTRODUCTION

This document updates and combines two Risk Profiles considering *Listeria monocytogenes* in soft and low moisture cheeses completed in 2005 (Lake *et al.*, 2005a; Lake *et al.*, 2005b). Extra hard cheeses (<34% moisture content) are excluded from consideration in this Risk Profile, as they are considered to present a very low risk of exposure to *L. monocytogenes* (Lake *et al.*, 2005b). Cheese spreads are also excluded, as cheese is only one ingredient in these products. This Risk Profile also considers aspects of the milk used to produce cheese and includes cheeses made from pasteurised, thermised and unpasteurised (raw) milk.

This is not a stand-alone document and readers are referred to the 2005 Risk Profiles, plus a 2014 Risk Profile concerning *L. monocytogenes* in raw milk (King *et al.*, 2014). These can be accessed from: http://www.foodsafety.govt.nz/science-risk/risk-assessment/risk-profiles/.

The purpose of this update is to critically review new information to answer the following risk management questions:

- What is the public health risk from *L. monocytogenes* in cheese consumed in New Zealand?
- Has the risk of listeriosis from the consumption of cheese consumed in New Zealand changed since the 2005 Risk Profiles were published?

Risk Profiles provide scientific information relevant to a food/hazard combination for risk managers and describe potential risk management options (NZFSA, 2010a).[3]

[3] Risk Profiles commissioned by MPI and its predecessors can be viewed at: http://www.foodsafety.govt.nz.

2 HAZARD AND FOOD

2.1 The Pathogen: *L. monocytogenes*

KEY FINDINGS

Of the species of *Listeria*, *L. monocytogenes* is the most important risk to human health.

There is more evidence to show that *L. monocytogenes* strains vary in their ability to survive in food and cause disease in humans but more work needs to be done to distinguish between those types most and least associated with disease. There is no change to the view that all *L. monocytogenes* strains need to be considered potentially pathogenic.

Appendix 1 contains additional information on L. monocytogenes.

2.1.1 Disease and Pathogenicity

There are now ten species in the genus *Listeria* but *L. monocytogenes* is considered to be the most important species with respect to human health, and food is considered to be the main transmission route to humans (Cressey and Lake, 2007). The disease resulting from infection, listeriosis, can manifest in two forms 1) an invasive disease which can result in the death of approximately 20% of cases and 2) a milder febrile gastroenteritis (non-invasive listeriosis) not known to cause fatalities. Invasive listeriosis usually occurs in people who are pregnant, old or immunocompromised.

There is growing evidence that *L. monocytogenes* isolates vary in their ability to cause human disease, and isolates of *L. monocytogenes* can be assigned to serotypes and/or lineages. While it is known that certain serotypes of *L. monocytogenes* appear to be associated with human disease and *L. monocytogenes* lineages differ in their contribution to human disease, there is no certainty that any one isolate will (or will not) be pathogenic to humans just because it belongs to a particular group.

A set of genetic markers to determine whether a strain of *L. monocytogenes* will cause human disease has not yet been identified. Until further research provides certainty in this area, and standard methods for testing pathogenicity are validated and implemented, all *L. monocytogenes* need to be considered potentially pathogenic.

2.1.2 Defining Characteristics

L. monocytogenes is notable for being a hardy organism resistant, to an extent, to many physico-chemical methods of control. It is also able to grow at low temperatures, with papers reporting growth at -1.5°C (Hudson *et al.*, 1994). These characteristics confer on the organism the ability to colonise food production facilities since it is able to survive and grow in cool moist conditions. Of relevance to the food under consideration in this Risk Profile is that the organism can contaminate raw milk and subsequently grow in it, even under refrigeration. In addition its ability to colonise processing plants means that there is opportunity for cross contamination to cheese that has been made from pathogen-free milk.

An important observation since the previous Risk Profiles is that *L. monocytogenes* isolates from cheese had higher growth rates under more acidic pH values and higher salt concentrations than two laboratory reference strains, reflecting adaptation to conditions

presented by the cheeses and cheese making environment (see Section 7.1) (Ribeiro *et al.*, 2006).

2.2 The Food: Cheese

KEY FINDINGS

The initial steps in cheese production include coagulation of the milk, often in the presence of a starter culture, to produce curds and whey which are then separated. The curds are pressed to form the solid cheese. There are then many processes to which the curd may be subject including brining, inoculation with bacteria or moulds and ripening. The physico-chemical properties of the final product vary greatly, making its classification difficult. A classification system based on both process and moisture content has been adopted for this document.

Of the estimated volume of cheese available domestically in 2014 (37,500 tonnes), around 18% was imported. The volume of cheese imported into New Zealand is increasing annually.

2.2.1 How cheese is made

The huge variety of cheeses means that there is no standard cheese making method (Fox *et al.*, 2004). The 2005 Risk Profiles provide descriptions of generic methods for the production of low moisture cheeses (<50% moisture), and soft cheeses with and without ripening. Broadly milk is coagulated into curds and whey by a number of methods including addition of rennet, acid or by using heat. Many, but not all, cheeses are inoculated with a starter culture that converts lactose into lactic acid responsible for a reduction in pH. Whey and curd are separated and the curd pressed to form a solid. The pressed curd is often salted by the inclusion of salt or by immersion in brine. The curd can be inoculated with moulds internally to produce blue cheese or externally to produce cheeses like Brie, or with bacteria to produce smear ripened cheeses. The degree of pressing, salt addition and ageing all contribute to the final organoleptic and physico-chemical qualities of the cheese.

2.2.2 Cheese classification

The 2005 Risk Profiles defined cheeses by their moisture content and described a similar cheese classification system used by Food Standards Australia New Zealand (FSANZ) (FSANZ, 2001, 2014), which has not changed. A second classification system was also explained, based on the moisture fat free basis percentage (MFFB%).

For the purposes of this combined and updated Risk Profile, MPI and ESR agreed on a different classification scheme that was not based solely on final moisture content, but also on the method of production. This focuses the risk evaluation on the steps during production and the end-characteristics of the cheeses that impact on *L. monocytogenes* behaviour.

The classification scheme used in this Risk Profile update is presented in Figure 1 (adapted from (McSweeney *et al.*, 2004)). The scheme primarily classifies cheeses based on the method of coagulation. It is not practical to produce a scheme that takes into account all the different approaches to cheese making so there will be cheeses that do not fit neatly into this scheme (e.g. Gruyére is a bacterially surface ripened cheese that can also contain eyes). This is noted in this Risk Profile where necessary. The type of milk used for making cheeses is considered separately from the scheme as a potential source of *L. monocytogenes*.

Acid coagulation involves the addition of an acid-producing starter culture (e.g. *Lactococcus* spp.) or food grade acids (direct acidification, e.g. using lactic or phosphoric acid followed by glucono-δ-lactone) (Farkye, 2004a). Examples of acid coagulated cheeses are Cottage, Quark and Cream cheeses. Acid/heat coagulation involves a high heat treatment (>70°C, with the temperature and time depending on the cheese) followed by acidification using food grade acids (e.g. citric, acetic), fruit juice or acid-whey concentrate (Farkye, 2004b). Examples of acid/heat coagulated cheeses are queso fresco, marscapone and ricotta. Acid and acid/heat coagulated cheeses are more likely to be consumed without ripening, so may be referred to as "fresh cheeses". They are high moisture soft cheeses.

Rennet coagulated cheeses are sometimes called "natural cheeses". Rennet is an enzyme mixture (principally chymosin) traditionally derived from the stomachs of young animals (especially calves). Milk coagulating enzymes can also be extracted from plants (Jacob *et al.*, 2011; Shah *et al.*, 2014).[4] Alternative sources are enzymes from older animals and bacteria that have been modified to express inserted chymosin genes (Fox and McSweeney, 2004). In this Risk Profile, cheeses categorised as "rennet coagulated" include cheeses coagulated using any of these enzyme sources.

After coagulation, the cheeses are further divided based on the ripening agents or manufacturing technology. Three general ripening categories have been selected for the classification scheme:

(i) Surface ripened by yeasts and Gram-positive bacteria (staphylococci, micrococci), which may be acquired from the environment, or purposely smeared onto the surface. Also called smear-ripened, washed-rind or red-smear cheeses. They are usually washed in brine. Examples include Gruyére and Havarti.

(ii) Mould ripened by fungi, principally *Penicillium* spp. Brie and Camembert are examples of surface mould ripened cheeses, while blue, Gorgonzola, Roquefort and Stilton are internally mould ripened cheeses.

(iii) Internally ripened by bacteria that are present in the milk (either raw milk or bacteria that survive pasteurization or thermisation) and/or bacteria that are deliberately inoculated ("starter cultures") or enter the cheese as it is made.[5]

Cheeses that are internally bacterially ripened are further divided by moisture content into extra hard (<34% moisture and not considered in this Risk Profile), hard (34-38% moisture, e.g. Cheddar) and semi-hard/semi-soft (39-50% moisture, e.g. Colby, Monterey).

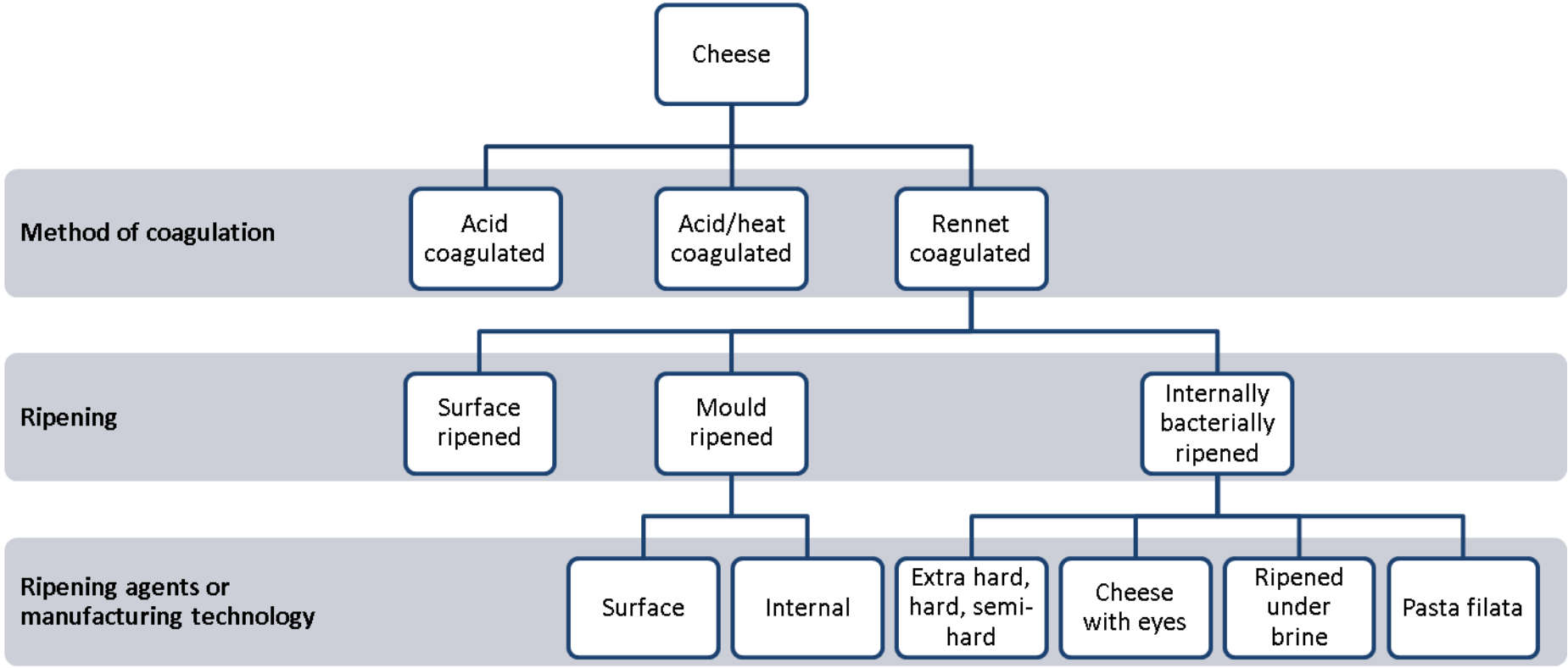
Alternatively, cheeses can be categorised by characteristic manufacturing technology:

- The presence of eyes caused by production of carbon dioxide (Emmental is an example of a hard variety, Edam and Gouda are semi-hard varieties).
- Ripened under brine, e.g. feta.
- Pasta filata or stretched curd cheeses where the curd is stretched in hot water, e.g. mozzarella.

[4] These can be used by cheese makers marketing their cheeses for people following a vegetarian or halal diet.
[5] The indigenous milk enzymes and residual coagulant also aid in the ripening.

Most types of cheese can be further processed, which involves comminuting, melting and emulsifying the cheese into a homogenous molten blend. The process usually includes the addition of emulsifying salts, and is intended to achieve a product with greater shelf life (Guinee *et al*., 2004).

Figure 1: The cheese classification scheme used in this Risk Profile.



Note to Figure 1: This diagram is based on a scheme described by McSweeney *et al.* (2004).

2.2.3 Cheese production in New Zealand

There is no official information on total cheese production in New Zealand (Alex Bartley (MPI), pers. comm., February 2014). Based on New Zealand's cheese exports (see section 2.2.4) and assuming that 90-95% of New Zealand's cheeses (by weight) are exported, an estimated 293,000-309,000 tonnes of cheese was produced during 2014. These figures are slightly lower than the 324,000 tonnes as published by the United States Department of Agriculture (USDA) for New Zealand but are similar to the 2002 and 2003 production values reported in the 2005 Risk Profiles.[6]

The increasing availability of artisanal cheeses in supermarkets and markets, or for sale over the internet, suggests that artisanal cheeses are becoming more widely available. There are no data to show trends in volume or varieties produced, but the number of artisanal cheesemakers appears to have increased. As of February 2015 there were 50 members listed on the website of the New Zealand Specialist Cheesemakers Association Inc. (NZSCA) compared with 34 in 2005.[7] As of February 2015, a search under 'cheese' yielded a total of 43 operators who have either a Risk Management Programme (RMP) or a Food Safety Programme (FSP) registered with MPI and whose scope of activities include the manufacture or processing of cheese.[8] The 2005 Risk Profiles listed 41 registered cheese making premises in New Zealand.

At the end of 2012, the first commercial production of a raw milk cheese was approved in New Zealand by MPI. Following extensive testing, Aroha's raw milk Gouda went on sale to the public.[9] As of February 2015 there were two raw milk cheese producers registered with MPI (T. Soboleva, MPI, pers. comm.). The total weight of raw milk cheese produced is not known.

2.2.4 International trade

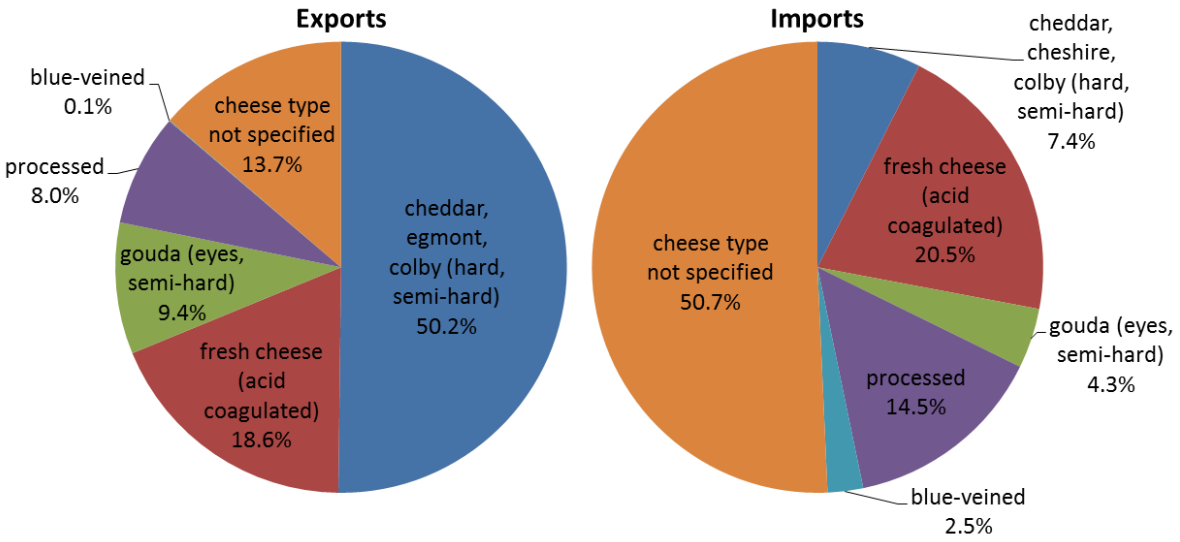
There were 278,143 tonnes of cheese exported from New Zealand in the year ending December 2014.[10] The volume exported is similar to that reported by the 2005 Risk Profiles for the year 2003 (293,000 tonnes). The largest exported volume by weight in 2014 was to Japan (21%), followed by Australia (16%) and China (10%). Hard and semi-hard cheeses, mostly cheddar, made up half of the export volume by weight (Figure 2).

A wider range of cheeses are now permitted to enter New Zealand compared to 2005 (see Section 5.1.5). The volume of cheese imported (6,624 tonnes in the year to December 2014) is larger than reported in the 2005 Risk Profiles (1,900 tonnes year ending March 2003).[11] Where the type of cheese product was specified, fresh cheeses made up the largest proportion of imported cheeses by weight (Figure 2). The proportion of imported cheeses that are made from raw milk is not known. Australia was the country of origin for 37% of the imported

[6] http://www.indexmundi.com/agriculture/?country=nz&commodity=cheese&graph=production accessed 3 February 2015.
[7] http://www.nzsca.org.nz/members/ accessed 3 February 2015.
[8] http://www.foodsafety.govt.nz/registers-lists/exemption.htm http://www.foodsafety.govt.nz/registers-lists/rmp-dairy/index.htm, scope = "cheese", accessed 3 February 2015. Additional cheese producers will be registered as producers of "milk products".
[9] http://www.organicgoatcheese.co.nz/html/index.htm accessed 9 October 2013
[10] Data from Statistics New Zealand Infoshare, http://www.stats.govt.nz/infoshare/ accessed 2 February 2015. All HS codes 0406 excluding curds, exports + re-exports.
[11] Or 2,189 tonnes for the year ending December 2003 (Statistics New Zealand Infoshare, http://www.stats.govt.nz/infoshare/, accessed 25 February 2014). All HS codes 0406 excluding curds.

cheese by weight; other countries of origin were the USA (19%) and Denmark (14%). In 2003, cheese from the USA only represented 0.2% of total imports. Most (90%) of the cheese imported from the USA in 2014 is categorised as "not elsewhere classified" so the type of imported cheese is not known (54% is "grated or powdered" and possibly used as an ingredient in mixed foods).

Figure 2: Types of cheese exported and imported to New Zealand, by weight, year ending December 2014



Note to Figure 2: Data from Statistics New Zealand (Infoshare, http://www.stats.govt.nz/infoshare/Default.aspx (accessed 2 February 2015).

2.2.5 Amount available to the New Zealand domestic consumer

Based on the higher estimate for New Zealand cheese production (309,000 tonnes), and export and import statistics (278,143 and 6,624 tonnes, respectively), the estimated weight of cheese available to the domestic consumer in 2014 was 37,481 tonnes (18% of this was imported cheeses). This equates to a crude estimate of 23 g/person/day, which is lower than the estimate of 27 g/person/day reported in the 2005 Risk Profiles.[12] Section 2.4.3 contains data on cheese consumption.

[12] The resident population mean for the year ending June 2014 was 4,476,300 people (Statistics New Zealand Infoshare, (http://www.stats.govt.nz/infoshare/) accessed 3 February 2015).

2.3 Behaviour of *L. monocytogenes* in Cheese

KEY FINDINGS

The growth/no growth boundary for *L. monocytogenes* during cheese making is defined by a number of physico-chemical characteristics such as pH, water activity, lactic acid concentration and temperature. In many cheeses growth will only occur during manufacture as conditions are permissive only at that stage. If the pathogen is unable to grow under a given set of conditions then its concentration will decline at a rate largely dictated by the temperature.

It is often not possible to predict how *L. monocytogenes* will behave because there is too much variability in both the results from scientific studies and the cheese making processes. FSANZ have defined the pH and water activity limits for growth of *L. monocytogenes* in ready-to-eat foods: Growth will not occur when the food has a pH<4.4 regardless of water activity, a water activity <0.92 regardless of pH, or a combination of pH<5.0 and water activity <0.94. Recent studies of *L. monocytogenes* behaviour during the ripening and storage of cheeses is consistent with these limits.

2.3.1 Contamination of cheese by *L. monocytogenes*

L. monocytogenes can contaminate cheese from:

- Contaminated ingredients; and/or
- The environment in which the cheeses are made and handled (e.g. equipment, surfaces, brining tanks, food handlers, pests), including any post-process stages such as slicing or grating.

L. monocytogenes could be introduced to cheese by any of the variety of the ingredients (e.g. herbs and spices) added during the cheese making process. While the pathogen has been detected in brines and smear mixes (Barancelli *et al.*, 2011; Wagner *et al.*, 2006), milk is the most important ingredient with regard to *L. monocytogenes* contamination.

Because New Zealand regulations now permit raw milk to be used for manufacturing some cheeses, raw milk must now be considered as an important potential source of *L. monocytogenes* alongside post-pasteurisation contamination or pasteurisation failure. Information on contamination of raw milk by *L. monocytogenes* has been reviewed in the raw milk Risk Profile (King *et al.*, 2014). Briefly, raw milk can become contaminated with *L. monocytogenes* from soiled udders and milking equipment, and animals with listerial mastitis. Listerial mastitis appears rare but can result in a high concentration of *L. monocytogenes* in the milk. There are reports of *L. monocytogenes* in cheeses made from raw milk where the source of contamination was an animal in the milking herd with subclinical listerial mastitis (Delhalle *et al.*, 2012; Pintado *et al.*, 2009; Schoder *et al.*, 2008). The prevalence of *L. monocytogenes* in the farming environment is enhanced by faecal shedding from animals infected by *L. monocytogenes* or fed with poor quality silage or baleage. *L. monocytogenes* can form biofilms on the milking equipment and slough off into the milk (Adetunji and Adegoke, 2008).

The 2005 Risk Profiles contained information that suggested there was potential for *L. monocytogenes* to survive milk pasteurisation. The effectiveness of high-temperature short-time (HTST, ≥72˚C for ≥15 seconds) and batch (≥63˚C for ≥30 minutes) pasteurisation on

survival of *L. monocytogenes* was reviewed by Food Standards Australia New Zealand (FSANZ) in 2007 and it was concluded (based on new, more definitive studies) that *L. monocytogenes* would not survive these pasteurisation treatments. The conservative estimate for the reduction in *L. monocytogenes* after 16 seconds at 72˚C was an 11.4 $\log_{10}$ reduction (Juffs and Deeth, 2007).

New Zealand regulations permit some cheeses to be made with thermised milk (≥64.5˚C for ≥16 seconds) (MAF, 2011b). The review of Juffs and Deeth (2007) did not specifically consider this time/temperature regime, nor did an earlier New Zealand review (Hudson *et al.*, 2003), but the D-times reported in these reviews at temperatures of 63-65˚C suggest that *L. monocytogenes* might survive thermisation (the D time at 63˚C is 42-43 seconds).[13,14] A recent New Zealand study using UHT milk demonstrated variability in heat resistance between 29 *L. monocytogenes* isolates, and then evaluated the effectiveness of different temperatures on the second most heat resistant strain (a clinical isolate, serotype 1/2b) (Pearce *et al.*, 2012). At 62.5˚C for 60 seconds, the 29 strains reduced by between 1.66 and 3.53 $\log_{10}$. When held for 15 seconds in UHT milk, the concentration of the heat resistant strain (approx. $1x10^9$ CFU/mL) decreased by approximately 2 $\log_{10}$ at 63˚C, 4 $\log_{10}$ at 64˚C and 7 $\log_{10}$ (to the limit of detection) at 66˚C. The D-values were calculated as 28±5 seconds at 63˚C and 14±3 seconds at 64˚C, and the z-value as 2.90˚C.[15] However, thermisation is a treatment not intended to control the presence of pathogens, but instead is used to allow the improved development of starter bacteria, or to confer increased shelf life to milk prior to cheese making (Rukke *et al.*, 2011). Therefore, while it does result in some inactivation of *L. monocytogenes* it is intended to be used alongside other hurdles for the production of safe cheese.

Studies of environmental sources of *L. monocytogenes* show that the bacterium could be isolated from the cheese making environment and that the same strains can be found in the environment and on the cheeses (Lake *et al.*, 2005a; Lake *et al.*, 2005b). The ripening process was highlighted as a step when contamination of the cheeses often occurred. Recent surveys of the equipment and surfaces in cheese making facilities have found that:

- Cheese making facilities on dairy farms were more likely to be contaminated with *L. monocytogenes* if the animals were fed silage (Schoder *et al.*, 2011);
- *L. monocytogenes* can be transferred from the farming environment to cheese making facilities, where these are located on the same property (e.g. carried in on work boots) (Fox *et al.*, 2011; Schoder *et al.*, 2011);
- *L. monocytogenes* was often isolated from non-food contact areas such as drains, walls and floors (Cagri-Mehmetoglu *et al.*, 2011; Chambel *et al.*, 2007; D'Amico and Donnelly, 2009; Ho *et al.*, 2007; Schoder *et al.*, 2011), and these areas may offer niches for persistent contamination (Ibba *et al.*, 2013);
- *L. monocytogenes* can contaminate cheese ingredients or equipment that handles such ingredients (De Cesare *et al.*, 2007);

[13] The D-time (or D-value) is the time required to reduce a population of organisms by 90% or 1 $\log_{10}$ unit at a given temperature (Hudson *et al.*, 2003)

[14] The 2005 low moisture cheese Risk Profile also presented information that indicated potential for *L. monocytogenes* to survive thermisation.

[15] The z-value is the temperature change required to alter the D-time by a factor of 10, i.e. the change in temperature required to increase or decrease the length of the heat treatment (in practice, the holding time) by a factor of 10 (Hudson *et al.*, 2003; Juffs and Deeth, 2007).

- Food contact surfaces, such as tables, knives, ladles and buckets, can be contaminated with *L. monocytogenes* (D'Amico and Donnelly, 2008); and
- *L. monocytogenes* strains can persist in the cheese making environment for long periods (>1 year) (Almeida *et al.*, 2013; Ho *et al.*, 2007; Lomonaco *et al.*, 2009; Wagner *et al.*, 2006).

2.3.2 Behaviour of *L. monocytogenes* in cheese

L. monocytogenes will survive and grow in cheeses where conditions are favourable. The factors in cheese that prevent growth of most other pathogenic bacteria (the presence of salt, low water activity, low pH, storage at refrigeration temperatures) will not necessarily prevent growth of *L. monocytogenes*.

The 2005 Risk Profiles noted that it is not possible to predict how *L. monocytogenes* will behave for many categories of cheese because there is too much variability in both the results from scientific studies and the cheese making processes. This remains true, but some general findings can be stated.

2.3.2.1 L. monocytogenes behaviour during manufacture

Growth of *L. monocytogenes* can occur during cheese making, but may be inhibited by the presence of other microflora and sufficient reduction in pH. *L. monocytogenes* inoculated into milk has been shown to grow over the first five hours of manufacture of smear ripened cheese made from pasteurised milk, but not raw milk (Schvartzman *et al.*, 2011b). It was suggested in these studies that the composition of the background flora influences the ability of *L. monocytogenes* to grow, i.e. there is less competition to repress growth in pasteurised milk. However, it should be noted that *L. monocytogenes* grew in cheese made from raw milk during ripening, but not in that made from pasteurised milk and this was linked to the slower production of lactic acid that occurred in the raw milk cheese. Similar results have been reported for experiments using smear ripened semi-soft cheeses from milk inoculated with 500 CFU/mL *L. monocytogenes* (Jordan *et al.*, 2010). No growth was observed in manufacture of cheeses made from raw milk, while in pasteurised milk cheese growth was observed with the maximum growth rate 1.3 $\log_{10}$ CFU/h.

The concentration of *L. monocytogenes* decreased in raw sheep milk cheese over the 24-hour manufacturing period provided the starter culture was at $>10^7$ CFU/mL, which ensured the pH fell from around 6.5 to 4.5. With 10^5 CFU/mL starter culture the pH only fell to 5.6 and *L. monocytogenes* grew (Schoder *et al.*, 2008). Similarly, adding a starter culture to pasteurised sheep and goats' milk reduced the pH from 6.4 to 4.4 during the 24-hour manufacturing period and the concentration of *L. monocytogenes* inoculated into these cheeses remained stable or decreased (Theodoridis *et al.*, 2006).[16]

A challenge trial to determine *L. monocytogenes* behaviour during the initial fermentation step of cheese manufacture used both raw and pasteurised milk, and a mixture of six *L. monocytogenes* isolates, as a high inoculum cocktail (approximately 10^6 cells/mL in milk) (Withers and Couper, 2012). A single strain was also tested separately, as a low inoculum (approximately 10^2 cells/mL in milk). Two different commercial starter cultures were used, to

[16] Theodoridis *et al.* (2006) do not specify the concentration of starter culture, but the information provided in their paper suggests that it is $\geq 10^6$ CFU/mL.

give four parallel experiments at each inoculum level. No significant changes in *L. monocytogenes* numbers were observed, regardless of the milk type, pH change, lactic acid concentration or starter culture used. Thus if *L. monocytogenes* is present in the milk, it will not grow during fermentation, but neither will the numbers be reduced. This result contrasts with that found for the full cheese making process cited above, but perhaps growth only occurred after the initial fermentation step in those experiments (Schvartzman *et al.*, 2011b).

Salt solutions or brines are used in the manufacture of a number of cheeses. These solutions may be reused over long periods, with the amount of salt being topped up from time to time. *L. monocytogenes* is able to survive in brines and so has the potential to contaminate cheeses during manufacture. Contaminated brine has been identified as a source in at least one outbreak (Johnsen *et al.*, 2010). A study of the survival of *L. monocytogenes* in fresh and used brines found that survival was lower in used brines, possibly because the pH was lower (<5) after the development of a lactic acid bacteria population in the used brines (Schirmer *et al.*, 2014). Although numbers declined (up to 4 $\log_{10}$ reduction over 45 days), some strains of *L. monocytogenes* were able to survive in brines for up to 200 days after inoculation.

2.3.2.2 L. monocytogenes behaviour during ripening

Table 1 summarises studies published since 2005 on the behaviour of *L. monocytogenes* in cheeses during ripening. The ripening stage is important as it is much longer than the manufacturing stage, and changes in the characteristics of cheeses during ripening influence *L. monocytogenes* behaviour.

From the observations cited above and those from studies published from 2005 (Table 1), pH and water activity appear to be the most important parameters determining whether growth of *L. monocytogenes* will occur (temperature determines the rate of any growth or decline). FSANZ have defined pH and water activity limits for control of *L. monocytogenes* growth in ready-to-eat foods: Growth will not occur when the food has a pH<4.4 regardless of water activity, a water activity <0.92 regardless of pH, or a combination of pH<5.0 and water activity <0.94 (see Section 5.1.3). The majority of studies report pH values of the cheeses at the start and/or end of ripening, but fewer studies report the water activity values as well. It is therefore difficult to compare and draw definitive conclusions on all studies and cheese types, especially as growth of *L. monocytogenes* in cheese is also dependant on other factors such as salt concentration and temperature during ripening. The available information since 2005 is consistent with the FSANZ limits.

L. monocytogenes concentrations are reduced in cheeses where a heating step is included in their manufacture, e.g. pasta filata varieties exposed to temperatures from 55 to 60°C (McSweeney *et al.*, 2004).

Table 1: The behaviour of *L. monocytogenes* in cheeses during ripening (studies published since 2005)

Cheese category	Behaviour of *L. monocytogenes* during ripening
Acid coagulated, acid/heat coagulated	Not applicable.
Rennet coagulated – bacterially ripened (surface)	Declined in the core and rind of pasteurised milk smear ripened cheeses during the first 9 days of a 28-day ripening period, and increased in the core and rind of raw milk cheeses during the first 4 days followed by survival/decline until the end of ripening (Schvartzman *et al.*, 2011b). The pH decreased to 4.7 and 5.0 in pasteurised and raw milk cheeses, respectively, during the 6-9 day period when there was greatest change in *L. monocytogenes* concentration. In the following 20 days of ripening, pH increased in all cases ranging from pH~5.0 and 5.2 for the core of pasteurised and raw cheeses, respectively to pH ~5.8 and 6.0, for rind of pasteurised and raw milk cheese, respectively. At the end of ripening, the water activity values of the rinds were 0.79 and 0.75 in pasteurised and raw milk cheese, respectively. The water activity was higher in the rind than in the core (pasteurised 0.815; raw 0.892). Lower levels of lactic acid and higher pH values were recorded for the raw milk cheeses compared with pasteurised milk cheeses, probably due to higher background microflora inhibiting the starter culture (Schvartzman *et al.*, 2011b) Grew when sprayed or inoculated onto cheeses (simulating environmental contamination). Growth rates on the surface of smear ripened cheeses during ripening at 15°C in two studies were similar at approximately 0.2-0.3 $\log_{10}$ CFU/day (Izquierdo *et al.*, 2009; Jordan *et al.*, 2010)
Rennet coagulated – mould ripened (surface)	Grew in and on pasteurised milk Camembert during the latter stages of ripening when the pH increased to >5.0 (D'Amico *et al.*, 2008; Liu *et al.*, 2009).[1] The final pH of these cheeses was >7.0. Behaviour was the same on the surface of raw milk Camembert (D'Amico *et al.*, 2008) In Camembert made from raw cows' milk, the concentration of *L. monocytogenes* in the cheese after 14 days of ripening (pH=5.6) was only slightly lower (<1 $\log_{10}$ CFU/g) than that measured in the curd (pH=4.7) (Linton *et al.*, 2008).
Rennet coagulated – mould ripened (internal)	Growth of the moulds raises the pH of the cheeses (lactic acid reduces and ammonia is produced), so *L. monocytogenes* will not grow in young, acidic cheeses (pH~4.9), but will grow in more mature cheeses (pH>6) (Rosshaug *et al.*, 2012). A salt gradient was also apparent in young cheeses (around 2.5 greater concentration of NaCl in the rind compared with the core), but this gradient reduced during ripening.

Table 1 continued

Cheese category	Behaviour of *L. monocytogenes* during ripening
Rennet coagulated – bacterially ripened (internal) – hard, semi-hard	*L. monocytogenes* grew in Chevre Metsovo cheeses during the first five days of ripening, after which the general pattern was one of stability. The pH decreased slowly during the first five days but remained >5.0 (Theodoridis *et al.*, 2006). In Portuguese raw sheep milk cheeses naturally contaminated with *L. monocytogenes* (pH 4.8-4.9 during ripening), the concentration significantly increased between 7 and 42 days of ripening. At the end of ripening (120 days), the average *L. monocytogenes* concentration across 20 cheeses from one maker was 58 CFU/g (range 20-130 CFU/g), and was $4x10^3$ CFU/g (range $5x10^2$-$8x10^3$ CFU/g) across 16 cheeses from another maker (Gameiro *et al.*, 2007). A brining step in Graviera (20% salt) decreased the concentration of a non-pathogenic strain of *L. monocytogenes* and two *L. innocua* strains by 0.8 $\log_{10}$ CFU/g but the remaining cells survived ripening (Samelis *et al.*, 2009).
Rennet coagulated – internally bacterially ripened – cheese with eyes	*L. monocytogenes* did not grow (<0.5 $\log_{10}$ CFU/g) or was inactivated (decreased by >1 $\log_{10}$ CFU/g) in Gouda microcheeses during the first 8 weeks of ripening (Wemmenhove *et al.*, 2013). The *L. monocytogenes* were inoculated into pasteurised bovine milk before addition of the starter culture. The pH of the microcheeses increased from pH 5.3 after 1 day, to pH 5.5 after 7 months and pH 6.1 after 1 year. During further ripening (up to 1 year) significant inactivation was observed, associated with lower water activity (0.98 after 8 weeks, 0.92 after 7 months and 0.84 after 1 year).
Rennet coagulated – internally bacterially ripened – ripened under brine	The concentration of *L. monocytogenes* increased from the drained curd stage through to the end of ripening by 0.8 $\log_{10}$ CFU/g in Iranian white-brined cheese (pH was not reported) (Ehsani and Mahmoudi, 2013).
Rennet coagulated – internally bacterially ripened – pasta filata	Heat treatment of the curd (75ºC, 5 min) reduced *L. monocytogenes* by ~3 $\log_{10}$ in Kashar made from pasteurised cows' milk (Cetinkaya and Soyutemiz, 2007).
Rennet coagulated – internally bacterially ripened – Cheddar	*L. monocytogenes* concentrations in naturally contaminated raw milk Cheddar cheeses (pH 5.5) during a five month ripening period have been reported (Dalmasso and Jordan, 2014). For the first two months the concentration of *L. monocytogenes* did not exceed 20 CFU/g in one batch, and in the other batch the concentration was below enumeration levels and *L. monocytogenes* could only be detected by enrichment. In both batches, *L. monocytogenes* was only detected by enrichment at three months, and was not detected after this.

Table 1 continued

Cheese category	Behaviour of *L. monocytogenes* during ripening
Mixed cheese types or cheese not able to be classified	Rennet coagulated cheese using raw milk, no starter culture: In cheeses where acidification was slow and pH>5 throughout ripening, *L. monocytogenes* grew during the first 8 days then remained stable. In cheeses where acidification was faster and the pH reduced to <5 during ripening, the concentration of *L. monocytogenes* remained fairly stable. Overall, *L. monocytogenes* did not grow in cheeses with pH<5.2. *L. monocytogenes* inoculated onto the surface of the cheeses grew on all cheeses during ripening (rind pH>5.3 throughout the 28 day experiments) (Millet *et al.*, 2006). Growth/no growth boundaries were investigated by monitoring the behaviour of *L. monocytogenes* during the making of a semi-soft cheese with modified pH (lactic acid) and a_w (NaCl) (Schvartzman *et al.*, 2011a). No growth occurred at any pH (5.6-6.5) when a_w=0.944 (8% NaCl), and growth occurred in all experiments when $a_w \geq 0.975$ (0-3% NaCl). The a_w of the cheese had the strongest influence over whether *L. monocytogenes* would grow.

~, approximately; ≈, approximately equal to

[1] Because Camembert has ripening period measured in months, researchers only take samples at set intervals so the actual pH where growth begins is not always determined. Liu *et al*. (2009) first measured growth at 20 days, and the pH at this time was 6.3. D'Amico *et al*. (2008) detected growth at 21 days when the pH values were between 5.16 and 5.91.

2.3.2.3 L. monocytogenes behaviour during storage

Table 2 displays results from studies published since 2005 on the behaviour of *L. monocytogenes* in cheeses during storage. As previously stated above, the majority of studies report pH values of the cheeses at start and/or end of storage but fewer studies report the water activity values as well.

L. monocytogenes growth was observed in acid coagulated cheeses when pH>5.0 (queso blanco, queso fresco and minas frescal) and in most acid/heat coagulated cheeses (ricotta or ricotta salata, pH>5.5), but not in acid coagulated cheeses when pH<5.0. The lowest water activity reported for these cheese types was 0.94. *L. monocytogenes* growth was also reported for most mould-ripened cheeses (washed rind, brie, blue, pH>5.0, $a_w \geq 0.92$). *L. monocytogenes* growth was not observed in any other cheese experiments where storage was at refrigeration temperatures.

These results are in agreement with the FSANZ pH and water activity limits.

Acid curd smear ripened cheeses that were recalled during the 2009/10 outbreak of listeriosis in the European Union (EU), and that were positive for *L. monocytogenes*, were continuously sampled in the laboratory when stored at 4, 15 or 22ºC (Schoder *et al.*, 2012). At 4ºC the concentration of *L. monocytogenes* increased on average by 5.6 $\log_{10}$ CFU/g up until the cheeses were around 50 days old. The increases at 15 and 22ºC were 7.2 and 6.8 $\log_{10}$ CFU/g, respectively.

A study of the ability of 67 cheeses to support the growth of *L. monocytogenes* (inoculated onto cheese slices that were vacuum packed and stored at 25°C for up to 15 days) found that growth occurred on only four cheese types: Gruyére, queso blanco, queso fresco and string (mozzarella or a combination of mozzarella and Cheddar) (Leong *et al.*, 2014). Growth on Gruyére was unexpected since this was a low moisture product (34% moisture) and studies of hard and semi-hard cheeses in Table 2 suggest that *L. monocytogenes* is inactivated faster at higher temperatures. This sample of Gruyére had a lower percentage of salt in the moisture phase (2.9%) when compared with another Gruyére sample in the same study where *L. monocytogenes* did not grow (4.2%), and this may have been enough to allow *L. monocytogenes* to multiply.

Frozen storage at -20°C has been shown to reduce numbers of *L. monocytogenes* in fresh soft cheeses (Ben Slama *et al.*, 2013; Theodoridis *et al.*, 2006).

A review of the literature concerning the growth/no growth boundary of *L. monocytogenes* in cheese produced the graph shown in Figure 3 (Horn and Hudson, 2008). This report defined the growth boundaries in terms of pH and salt solution concentration only. The data shown in Figure 3 are consistent with similar data presented by Leong *et al* (2014) for four different foodborne pathogens. The review of Horn and Hudson, (2008) reported that the cheese growth data used did not have clear growth/no growth boundary with cheeses supporting growth or causing inactivation overlapping when defined by pH and salt in solution concentration. This means there will always be some kind of prediction error when trying to use a single boundary for all cheese types with these two variables. The availability of new data and including variables such as water activity, starter culture of lactic acid concentrations, as well as mathematical transformations of variable may assist in reducing the overlap in growth versus no growth data sets.

Table 2: Results from studies on the behaviour of *L. monocytogenes* in cheeses during storage (published from 2005)

Cheese type	Milk treatment (species)	Storage conditions	*L. monocytogenes* inoculation	pH	Water activity	Change in *L. monocytogenes* concentration[1]	Reference
Acid coagulated							
Queso Blanco	Pasteurised (NR)	5°C, ~35 days 10°C, ~27 days 15°C, ~12 days 20°C, ~12 days 25°C, ~6 days	Surface of cheese slices	Start: 6.8 End: NR	0.971	↑ short lag followed by growth then NC at all temperatures. Growth rate ranged from 0.011 log_{10} CFU/h (5°C) to 0.099 log_{10} CFU/h (25°C)	(Uhlich *et al.*, 2006)
Queso Fresco	Pasteurised (NR)	4°C, 28 days	Surface of cheese pieces	Start: 6.1 End: NR	0.977	↑ short lag followed by growth (4 log_{10} CFU/g over 15 days) then NC from 21 days.	(Soni *et al.*, 2010)
Minas Frescal (acidified with lactic acid)	Pasteurised (NR)	5°C, 25 days 10°C, 25 days	Prior to moulding	Start: 6.4 End: 5.4 (5°C), 5.1 (10°C)	0.984	↑ at 5°C by 2.2 log_{10} CFU/g in first 12 days, then NC ↑ at 10°C by 1.8 log_{10} CFU/g in first 6 days, then NC	(Naldini *et al.*, 2009)
Minas Frescal (acidified with starter culture)	Pasteurised (NR)	5°C, 25 days 10°C, 25 days	Prior to moulding	Start: 5.3 End: 4.6 (5°C), 4.7 (10°C)	0.984	NC at both temperatures	(Naldini *et al.*, 2009)
Minas Frescal (acidified with lactic acid, rennet coagulated)	Pasteurised (cow)	8-10°C, 12 days	To salted curd	Start: 5.8 End: 5.2	NR	↑ 5 log_{10} CFU/g	(Vera Pingitore *et al.*, 2012)
Minas Frescal (acidified with lactic acid, rennet coagulated)	Pasteurised (cow)	7°C, 21 days	To salted curd	Start: 6.5 End: 6.5	0.989-0.990	↑ ~5 log_{10} CFU/g	(Malheiros *et al.*, 2012)

Table 2 continued

Cheese type	Milk treatment (species)	Storage conditions	*L. monocytogenes* inoculation	pH	Water activity	Change in *L. monocytogenes* concentration[1]	Reference
Galotyri (factory made and artisan)	Pasteurised (sheep)	4°C, 28 days 12°C, 14 days	Mixed into cheese	Start: 3.8, 4.0 End: 3.8, 4.0	NR	↓ overall (initial decrease within 3 days then NC or slow decrease) for both temperatures and inoculum levels.	(Rogga *et al.*, 2005)
Katiki Domokou	Pasteurised (NR)	5°C, 30 days 10°C, 30 days 15°C, 20 days 20°C, 10 days	Mixed into cheese	Start: 4.5-4.6 End: 4.2-4.3	NR	↓ at all temperatures by ~4 $\log_{10}$ CFU/g	(Kagkli *et al.*, 2009)
Cottage cheese	NR	7.7°C, 1.3 x shelf-life	Mixed into cheese	Start: 4.8, 5.1 End: NR	Start: 0.99 End: NR	↓ in one sample NC in one sample	(Hudson *et al.*, 2011)
Cream cheese	NR	7.7°C, 1.3 x shelf-life	Mixed into cheese	Start: 4.3-4.6 End: NR	Start: 0.99 End: NR	↓	(Hudson *et al.*, 2011)
Acid/heat coagulated							
Ricotta	NR	4°C, 14 days	On cheese pieces	6.2-6.5[3]	NR	↓ overall (increased by 7 days). Acid-adapted cells (lactic acid, pH 5.1) behaved the same	(Cataldo *et al.*, 2007)
Ricotta	Pasteurised (cow)	4°C, 28 days	On cheese pieces	Start: 5.5 End: 6.6	NR	↑ 2 $\log_{10}$ CFU/g	(Martins *et al.*, 2010)
Ricotta	NR	7.7°C, 1.3 x shelf-life	Mixed into cheese	Start: 5.8-6.5 End: NR	Start: 0.99 End: NR	↑	(Hudson *et al.*, 2011)
Ricotta salata	NR (sheep)	4°C, 6 months	On rind	Start: 6.3 End: 5.9	Start:0.95 End:0.94	↑ 5 $\log_{10}$ CFU/g on rind, not detected in paste	(Spanu *et al.*, 2012)
Ricotta salata	NR (sheep)	4°C, 12 months	On rind	Start: 6.3 End: 6.3	Start:0.95 End:0.94	↑ 2 $\log_{10}$ CFU/g on rind, not detected in paste	(Spanu *et al.*, 2013)

Table 2 continued

Cheese type	Milk treatment (species)	Storage conditions	*L. monocytogenes* inoculation	pH	Water activity	Change in *L. monocytogenes* concentration[1]	Reference
Rennet coagulated – surface ripened							
Havarti	NR	7.7°C, 1.3 x shelf-life	Mixed into cheese	Start: 5.1 End: NR	Start: 0.97 End: NR	NC	(Hudson *et al.*, 2011)
Rennet coagulated – mould ripened (surface)							
Washed rind	NR	4°C, 16°C and 22°C for six weeks	On surface of cut segment	Start: Median 6.26 (5.83-6.26) End: Median 7.43 (6.99-7.57)	Start: Median 0.96 (0.95-0.97) End: Median 0.94 (0.92-0.96)	↑ 1.2 $\log_{10}$ CFU/g at 4°C after 42 days, ↑3.28 $\log_{10}$ CFU/g at 16°C after 42 days, ↑1.83 $\log_{10}$ CFU/g at 22°C after 42 days (3.9 $\log_{10}$ CFU/g at 22°C after 21 days)	(Tan *et al.*, 2008)
Rennet coagulated – mould ripened (internal)							
Gorgonzola	NR (cow)	4°C, 14 days	On cheese pieces	6.1-6.7[3]	NR	↓ overall. Acid-adapted cells (lactic acid, pH 5.1) behaved the same	(Cataldo *et al.*, 2007)
Brie	NR	4°C, 16°C and 22°C for six weeks	On surface of cut segment	Start: Median 5.91 (5.67-6.27) End: Median 7.29 (5.63-7.60)	Start: Median 0.97 (0.97-0.97) End: Median 0.96 (0.94-0.97)	↑2.6 $\log_{10}$ CFU/g at 4°C after 42 days, ↑4.2 $\log_{10}$ CFU/g at 16°C after 42 days, (4.3 $\log_{10}$ CFU/g at 22°C after 28 days) ↑4.2 $\log_{10}$ CFU/g at 22°C after 42 days (4.7 $\log_{10}$ CFU/g at 22°C after 28 days)	(Tan *et al.*, 2008)
Brie	Pasteurised (cow)	5°C and dynamic (5°C with spikes up to 14°C) for 21 days	Surface (2 brands)	Start: 7.4, 7.5 End: 7.8, 7.9	Start: 0.97 End: 0.97	↑ by approximately 1.2 $\log_{10}$ CFU/g at both temperatures after 21 days	(Wong *et al.*, 2008)

Table 2 continued

Cheese type	Milk treatment (species)	Storage conditions	*L. monocytogenes* inoculation	pH	Water activity	Change in *L. monocytogenes* concentration[1]	Reference
Blue	NR	4°C, 16°C and 22°C for six weeks	On surface of cut segment	Start: Median 7.10 (6.51-7.27) End: Median 7.14 (6.03-7.58)	Start: Median 0.97 (0.96-0.97) End: Median 0.95 (0.93-0.96)	↑3.6 $\log_{10}$ CFU/g at 4°C after 42 days, ↑4.5 $\log_{10}$ CFU/g at 16°C after 42 days, ↑4.0 $\log_{10}$ CFU/g at 22°C after 42 days	(Tan *et al.*, 2008)
Blue	NR	7.7°C, 1.3 x shelf-life	Mixed into cheese	Start: 5.6-7.3 End: NR	Start: 0.94-0.97 End: NR	↑ pH=7.3, NaCl=3.88% ↑ pH=5.6, NaCl=4.74% NG and ↓ pH=6.7, NaCl=6.26%	(Hudson *et al.*, 2011)
Katiki (high moisture (75%), low salt (1%)	Pasteurised (goat/sheep)	5, 10, 15, 20°C for up to 40 days	Mixed into cheese	4.3-4.5	NR	↓ by approximately 4 $\log_{10}$ CFU/g over 40 days at 5 and 10°C, over 20 days at 15°C, and 10 days at 20	(Mataragas *et al.*, 2008)
Rennet coagulated – internally bacterially ripened – hard, semi-hard							
Chevre Metsovo	Half raw, half pasteurised (goat)	4°C, 60 days	In milk prior to cheese making	Start: 5.2 End: 5.4	NR	↓ slowly (≤1 $\log_{10}$ over 60 days)	(Theodoridis *et al.*, 2006)
Cheddar (0.7 or 1.8% salt, with pH 5.1 or 5.3)	Pasteurised (NR)	4°C, 90 days 10°C, 90 days 21°C, 30 days	In comminuted cheese	Start: 5.1-5.8 End: 5.3-6.9	0.98-0.98	↓ slowly (max 1.5 $\log_{10}$) at all temperatures	(Shrestha *et al.*, 2011)
Greek Graviera	Thermised (NR)	4°C, 60 days 12°C, 60 days 25°C, 60 days	Surface of cheese slices	Start: 5.6 End: NR	Start: 0.948 End: NR	↓ at all temperatures, faster at 25°C. Vacuum packaging prolonged survival	(Giannou *et al.*, 2009)

Table 2 continued

Cheese type	Milk treatment (species)	Storage conditions	*L. monocytogenes* inoculation	pH	Water activity	Change in *L. monocytogenes* concentration[1]	Reference
Rennet coagulated – internally bacterially ripened – cheese with eyes							
Gouda	NR	7.7°C, 1.3 x shelf-life	Mixed into cheese	Start: 5.2, 5.4 End: NR	Start: 0.95, 0.93 End: NR	↓	(Hudson *et al.*, 2011)
Gouda (mild, lower salt)	NR	7.7°C, 1.3 x shelf-life	Mixed into cheese	Start: 5.0 End: NR	Start: 0.97 End: NR	NC	(Hudson *et al.*, 2011)
Rennet coagulated – internally bacterially ripened – ripened under brine							
Feta	Pasteurised[2] (sheep)	3-4°C, 2 months	Prior to adding rennet	Start: <4.6 End: NR	NR	NC	(Konteles *et al.*, 2009)
Feta	Pasteurised[2] (sheep)	3-4°C, 2 months	In brine	Start: <4.6 End: NR	NR	NC	(Konteles *et al.*, 2009)
Feta	Pasteurised (sheep)	4°C, 36 days, MAP[4]	Surface of cheese pieces	Start: 4.6 End: 4.6	NR	↓ 4 $\log_{10}$ CFU/g, not detected at 30 days	(Govaris *et al.*, 2011)
Iranian white-brined cheese	Pasteurised (cow)	4°C, 45 days	In milk	Start: NR End: 4.7	NR	NC	(Ehsani and Mahmoudi, 2013)
Turkish white cheese	NR	4°C, 15 days	Into cheese pieces	4.7-4.8 throughout	NR	↓ ~1 $\log_{10}$ CFU/g in 3.8% salt cheese for both acid and non-acid-adapted strains ↓ 1.3 and 1.9 $\log_{10}$ CFU/g in 6.7% salt cheese for acid and non-acid-adapted strains, respectively.	(Ilhak *et al.*, 2011)

Table 2 continued

Cheese type	Milk treatment (species)	Storage conditions	*L. monocytogenes* inoculation	pH	Water activity	Change in *L. monocytogenes* concentration[1]	Reference
Rennet coagulated – internally bacterially ripened – pasta filata							
Kashar	Pasteurised (cow)	6°C, 7 days, vacuum sealed	In milk prior to cheese making	NR	NR	NC	(Cetinkaya and Soyutemiz, 2007)
Mozzarella	NR (cow)	4°C, 14 days	On cheese pieces	5.4-6.0[3]	NR	↓ overall (increased by 7 days). Acid-adapted cells (lactic acid, pH 5.1) behaved the same	(Cataldo *et al.*, 2007)
Mozzarella	Raw (buffalo)	5°C, 3 weeks 10°C, 3 weeks 20°C, 9 days	Inoculated into conditioning liquid in which the cheese is stored	Start: 4.0 End: 4.8 (5°C), 5.1 (10°C), 4.7 (20°C)	NR	↓ 0.9 $\log_{10}$ CFU/g at 5°C (D-time 4 weeks) ↑ 1.7 $\log_{10}$ CFU/g at 10°C (growth began after 9 days) ↑ 2.2 $\log_{10}$ CFU/g at 20°C (growth began after 3 days)	(Finazzi *et al.*, 2011)
Mixed cheese types or cheese not able to be classified							
Grated processed cheese	NR	4°C, 1 year 12°C, 1 year 22°C, 1 year	Mixed with cheese	Start: 5.0 End: 5.0	Start: 0.93	↓ at all temperatures and inoculum levels.	(Angelidis *et al.*, 2010)
Individually wrapped processed cheese slices	Pasteurised (NR)	5°C, 9 months 22°C, 9 months	Surface of cheese slices	NR	NR	↓ 5 $\log_{10}$ CFU/g by 14 days and not detected up until 9 months for both temperatures.	(Linton and Harper, 2008)
Pichtogalo (rennet coagulated, unripened soft cheese)	Pasteurised (sheep and goat)	4°C, 30 days	In milk prior to cheese making	Start: 4.4 End: 4.3	NR	↓ at both temperatures. 4°C: D-time during rapid inactivation phase = 1.81 days	(Theodoridis *et al.*, 2006)
Crecenza	NR (cow)	4°C, 14 days	On cheese pieces	5.0-5.6[3]	NR	↓ by 7 days then NC. Acid-adapted cells (lactic acid, pH 5.1) grew	(Cataldo *et al.*, 2007)

Table 2 continued

Cheese type	Milk treatment (species)	Storage conditions	*L. monocytogenes* inoculation	pH	Water activity	Change in *L. monocytogenes* concentration[1]	Reference
Goats' milk cheese	NR (goat)	7.7°C, 1.3 x shelf-life	Mixed into cheese	Start: 5.0, 5.1 End: NR	Start: 0.97 End: NR	NC	(Hudson *et al.*, 2011)

NR: not reported

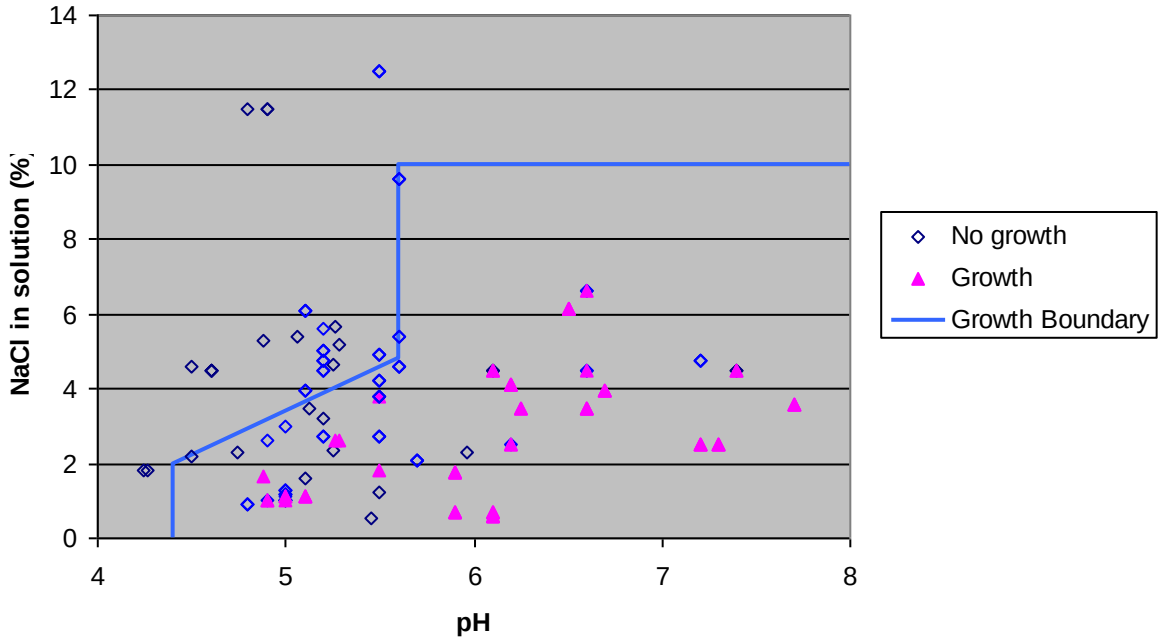
[1] ↓ decrease in concentration, ↑ increase in concentration, NC no change (a change in concentration of <0.5 $\log_{10}$ CFU/g).

[2] Heat-treated at 66-67°C for 20 min.

[3] Cataldo *et al.* (2007) do not report whether this is the pH range at the start or pH range measured over the course of the experiment.

[4] Modified atmosphere packaging, 50% CO_2/50% N_2.

Figure 3: Comparison of observed growth and no growth pH – NaCl (%) for *L. monocytogenes* in a variety of cheeses during ripening or storage



Note to figure: Reproduced from Horn and Hudson (2008).

2.4 Exposure Assessment

KEY FINDINGS

The two surveys of cheese for *L. monocytogenes* in New Zealand described in the previous Risk Profiles did not detect any positive samples. A further survey of locally available cheeses in 2010 also did not detect any *L. monocytogenes*.

Surveys of raw cows' and goats' milk for *L. monocytogenes* have been conducted more recently, which found prevalences of 0.7 – 4.1%.

Cheese consumption in New Zealand appears to have changed little since the previous Risk Profiles, and is dominated by hard and semi-hard varieties, mostly Cheddar.

2.4.1 New Zealand prevalence studies

Two surveys of cheese for *L. monocytogenes* in New Zealand were reported in the previous Risk Profiles. These surveys examined 300 samples of retail packed grated low moisture cheeses and 307 soft (>50% moisture) and semi-soft (39% to 50% moisture level) cheeses. No *L. monocytogenes* was detected, although in one blue semi-soft cheese sample, *L. welshimeri* was found.

A more recent survey, conducted in 2010, of soft, semi-soft and semi-hard imported and domestic cheeses available at retail in New Zealand did not detect *L. monocytogenes* in any of the 303 samples tested (Dr Tanya Soboleva, MPI, personal communication, August 2014). *L innocua* was detected in five samples.

As stated in Section 2.3.1, the major sources of *L. monocytogenes* contamination in cheese are ingredients (milk, particularly raw milk, is most important) and the environment. The prevalence in raw milk in New Zealand has been discussed in the Risk Profile on *L. monocytogenes* in raw milk (King *et al.*, 2014). In summary, *L. monocytogenes* was detected in two surveys of raw cows' milk from farm vats in New Zealand at prevalences of 0.7% and 4.1%. The concentrations of *L. monocytogenes* in the positive samples were low (range 0.047-0.36 CFU/mL). A survey of raw goats' milk (n = 60) found a prevalence of 3.3%.

2.4.2 Product recalls

Between 2008 and March 2014 there were three recalls of cheeses that were potentially contaminated with *L. monocytogenes*:[17]

- May 2011: Raw milk ewe cheese imported from France.
- January 2013: Various soft cheeses (Blue, Camembert, Brie; pasteurised milk) linked to products of the same brand that caused a listeriosis outbreak in Australia.
- March 2014: Blue cheese (pasteurised milk) produced by New Zealand manufacturer.

2.4.3 Food consumption: Cheese

The 2005 Risk Profiles presented data from the 1997 National Nutrition Survey (NNS) of people aged 15+ years (Russell *et al.*, 1999). ESR has since analysed data from two other New Zealand nutrition surveys to estimate consumption of soft and low moisture cheeses (Cressey, 2013; Cressey *et al.*, 2006). The data sets analysed were:

- The 2002 National Children's Nutrition Survey (CNS; 3,275 people aged 5-15 years) (Ministry of Health, 2003); and
- The 2009 Adult Nutrition Survey (ANS; 4,721 people aged 15+ years) (University of Otago and Ministry of Health, 2011).

Table 3 presents data on the consumption of soft cheeses, which includes cream, Brie, Camembert, cottage, mozzarella/bocconani, blue/blue vein, feta and quark/quarg/kwark. The overall pattern of soft cheese consumption by adults has not changed since the 2005 soft cheese Risk Profile (i.e. the 1997 NNS), but consumption of soft cheeses by pregnant women has decreased (Cressey, 2013).

[17] New Zealand food recalls are advertised at http://www.foodsmart.govt.nz/food-safety/recalls/latest-recalls/ (accessed 22 August 2014).

Table 3: Consumption of soft cheeses by New Zealanders (national nutrition surveys)

Statistic	Adult (2009 ANS)	Child (2002 CNS)
Number of respondents	4,721	3,275
Number of servings	274	43
Number of consumers (percentage of total respondents)	246 (5.2%)	40 (1.2%)
Servings/consumer/day (average)	1.1	1.08
Consumer mean (g/person/day)	27.0	31.0
Mean serving size (g)	24.2	28.8
Median serving size (g)	16.3	15.0
95th percentile serving size (g)	76.6	99.8

Table 4Table 4 presents data on the consumption of low moisture cheeses (cheeses with a moisture content of less than 50%). This includes cheeses such as Cheddar, Colby, Edam and Gouda, processed cheese, cheeses as fillings in filled rolls, wraps, croissants and sandwiches, cheese and crackers, and salads (coleslaw, green, pasta) containing cheese. Foods where the cheese had been cooked (e.g. pizza, lasagne, macaroni cheese) or may have received some heat treatment (e.g. omelette, cheeseburger, nachos with cheese) were excluded, as were parmesan and cheese spreads. This approach differs from the 2005 low moisture cheese Risk Profile, which included foods in which the cheese would have received some heat treatment. However, Cressey (2013) compared similar data sets for low moisture cheeses consumed by adults in the 1997 and 2009 nutrition surveys and noted that the overall consumption of low moisture cheeses (as measured by the population mean consumption) had not changed, but the prevalence of consumption had decreased significantly (<0.05) and serving sizes had increased.

Table 4: Consumption of unheated/uncooked low moisture cheese by New Zealanders (national nutrition surveys)

Statistic	Adult (2009 ANS)	Child (2002 CNS)
Number of respondents	4,721	3,275
Number of servings	1,301	640
Number of consumers (percentage of total respondents)	1,093 (23.2%)	539 (16.5%)
Servings/consumer/day (average)	1.2	1.2
Consumer mean (g/person/day)	37.1	39.8
Mean serving size (g)	31.2	33.4
Median serving size (g)	23.5	27.0
95th percentile serving size (g)	80.0	93.0

There are no data on the amount of raw milk cheeses consumed by New Zealanders.

2.4.3.1 Cheese types consumed in New Zealand

To provide information on consumption of cheese types classified as shown by Figure 1, 24-hour dietary recall records from the 2009 Adult Nutrition Survey and 2002 National Children's Nutrition Survey were analysed. The information on cheese types in these surveys is often incomplete, so these data are indicative. For example, a high proportion of servings involved cheese as a component of recipes. In these cases, cheese was only mentioned in a generic manner or was not mentioned but implicit in the name of the dish. It is likely that a high proportion of the cheese used in recipes will be Cheddar-type cheese, but this cannot be assumed.

For records for which a particular cheese type was specified, numbers of servings are shown in Table 5. These data indicate that cheese consumption in New Zealand is dominated by semi-hard varieties, principally Cheddar. The amount of processed cheese servings was modest (66 adult servings and 82 child servings, predominantly Cheddar-type), as was the amount of very hard cheese (parmesan: 36 adult servings and 10 child servings).

2.4.4 Potential for growth of *L. monocytogenes* along the cheese food chain

It is difficult to predict the potential for growth across the wide range of cheeses considered by this Risk Profile. Growth will depend on how *L. monocytogenes* was introduced (from ingredients (raw milk) and mixed throughout cheese, or surface contamination from the environment), the initial concentration, and cheese characteristics (moisture, salt, pH) (Tienungoon *et al.*, 2000). The rate of growth or decline will depend largely on temperature (Ross *et al.*, 2008).

Table 5: Frequency of reporting of servings of specific cheese types in national nutrition survey 24-hour dietary recall records

Cheese type	Adults (15+ years)[1]		Children (5-14 years)[2]	
	Number of servings	Percent of servings	Number of servings	Percent of servings
Acid Coagulated				
(cottage, quark, cream)	122	9.6%	33	6.3%
Acid/heat coagulated				
(queso fresco, mascarpone, ricotta)	5	0.4%	0	0.0%
Rennet Coagulated				
Surface ripened (Gruyére, Havarti)	5	0.4%	1	0.2%
Mould ripened, surface (Brie, Camembert)	63	5.0%	6	1.1%
Mould ripened, internal (blue, Gorgonzola, Roquefort, Stilton)	34	2.7%	1	0.2%
Rennet coagulated, internally bacterially ripened				
Cheese with eyes (Emmental, Edam, Gouda)	358	28.3%	110	20.8%
Ripened under brine (feta)	33	2.6%	0	0.0%
Pasta filata (mozzarella)	15	1.2%	2	0.4%
Hard and semi-hard (Cheddar, Colby, Monterey)	632	49.9%	375	71.0%

[1] From 2009 Adult Nutrition Survey (University of Otago and Ministry of Health, 2011)
[2] From 2002 National Children's Nutrition Survey (Ministry of Health, 2003)

2.5 Data on *L. monocytogenes* and Cheese from Other Countries

KEY FINDINGS

Surveys of cheeses in the countries that are likely to export cheeses into New Zealand indicate that the prevalence of *L. monocytogenes* is generally low (<2%), as is the concentration (<100 CFU/g).

Values were higher in some surveys for other countries, e.g. 46% prevalence in raw sheep milk cheese from Portugal, 460 CFU/g in a surface ripened cheese from Italy. The data suggest that surface ripened (smear) cheeses may be particularly susceptible to *L. monocytogenes* contamination.

Appendix 1 contains detailed data summarised in this section.

3 EVALUATION OF ADVERSE HEALTH EFFECTS

3.1 Disease Characteristics

KEY FINDINGS

There is no new and relevant information on invasive listeriosis ("listeriosis") or non-invasive listeriosis ("febrile gastroenteritis"), other than one study suggesting that pre-existing gastrointestinal problems such as irritable bowel syndrome and inflammatory bowel disease may be risk factors for febrile gastroenteritis (Schlech *et al.*, 2005).

Appendix 2 contains detail on disease characteristics

3.2 Dose Response

KEY FINDINGS

There has been no change to the view that the presence of *L. monocytogenes* in food at a concentration of <100 CFU/g carries a very low probability of causing disease.

Appendix 2 contains detail on dose response.

3.2.1 Invasive listeriosis

Dose-response models for *L. monocytogenes* were recently reviewed at an inter-agency workshop (Hoelzer *et al.*, 2013). The participants concluded that, while data have been generated to produce new and improved dose-response models, fundamental data are still lacking, e.g. determinants of virulence between different *L. monocytogenes* strains or susceptibilities among humans, the impacts of food matrices and the reliability of extrapolations to lower average doses.

While there has been no change to the view that the only completely safe dose of *L. monocytogenes* is zero, even for healthy people, studies indicate that the probability of invasive disease following exposure to even moderate levels of cells is very low. Most listeriosis cases are due to consumption of ready-to-eat foods able to support growth of *L. monocytogenes* and containing levels markedly above 100 CFU/g (Chen *et al.*, 2003; EFSA, 2007). The first of these papers concludes that 0.22/106 cases of listeriosis could be attributed to the consumption of foods containing up to 100 CFU/g and even when considering all servings containing up to 10^4 CFU/g, only 7.5 of the 106 cases would be attributed to those servings.

3.2.2 Non-invasive listeriosis

Dose response data for febrile gastroenteritis caused by *L. monocytogenes* infection are still limited to point estimates from outbreaks, which are often based on insufficient data. Consumption of more than 10^6 *L. monocytogenes* cells appears to be required to cause febrile gastroenteritis at a high attack rate (the 2005 Risk Profiles hypothesised 10^7 cells, which is not too dissimilar given the lack of data on non-invasive listeriosis). There is no change to the view that it is possible that foods contaminated with lower numbers of *L. monocytogenes* may cause non-invasive listeriosis.

3.3 New Zealand Human Health Surveillance

KEY FINDINGS

Cheese was reported as a risk factor for 15 sporadic listeriosis cases between 2006 and 2013 but was only confirmed as the cause of listeriosis for one case. Consumption of cheese has not been identified as a risk factor in any listeriosis outbreak between 1992 and 2013.

The annual rate of reported listeriosis cases has remained stable since the 2005 Risk Profiles (0.4-0.7 per 100,000) and non-perinatal cases continue to make up the majority. Where the clinical outcome is known, the proportion of cases hospitalised continues to be high (>85%) and a small number of fatalities occur each year.

Febrile gastroenteritis from *L. monocytogenes* infection would only be notified in New Zealand as acute gastroenteritis when there was a suspected common cause, so there are no data on infection rates. No other outbreaks of non-invasive listeriosis have been reported since 2000.

3.3.1 Cheese consumption as a risk factor for *L. monocytogenes* infection

3.3.1.1 Sporadic cases

Consumption of cheese was reported for 15/193 listeriosis cases reported to EpiSurv for the period January 2006 to December 2013, excluding two cases who consumed cheese overseas. However, data on the consumption of cheese is not routinely collected and it is possible that cheese was consumed by a higher proportion of listeriosis cases.

Of the 15 cases, cheese (feta cheese) was confirmed as the cause of listeriosis in one non-perinatal case. For this case, the report states that *L. monocytogenes* was isolated from an open pack of feta cheese (550 CFU/g), and the type was indistinguishable from the isolate from the case. For two other cases, the cheese was tested and found to be negative. For the other 12 cases, no evidence was found implicating a vehicle.

3.3.1.2 Outbreaks

None of the five listeriosis outbreaks reported in New Zealand between 1992 and 2013 were associated with consumption of cheese.

3.3.1.3 Case control studies

No case control studies concerning listeriosis have been conducted in New Zealand.

3.3.2 *L. monocytogenes* infection in New Zealand

Detection of cases with *L. monocytogenes* infection in New Zealand is biased towards detecting cases of invasive listeriosis because laboratories do not normally test faecal samples for *Listeria* spp. as part of a standard faecal screen. Most laboratories only test faecal samples for *Listeria* spp. if requested or if other information indicates that *L. monocytogenes* infection is a risk (e.g. the patient is pregnant) (Nicol *et al.*, 2010).[18] So while it appears that febrile gastroenteritis is

[18] Laboratories usually test specimens from sterile sites (e.g. blood, cerebral spinal fluid, amniotic fluid) for *Listeria* spp.

a rare cause of human disease, this condition might be responsible for a proportion of undiagnosed sporadic gastrointestinal disease reported each year. Moreover, febrile gastroenteritis usually has a short duration (1-3 days) and does not lead to serious complications in healthy people, so normally healthy people are unlikely to seek medical attention and thus remain unreported. Febrile gastroenteritis cases are more likely to be detected if they are part of an outbreak.

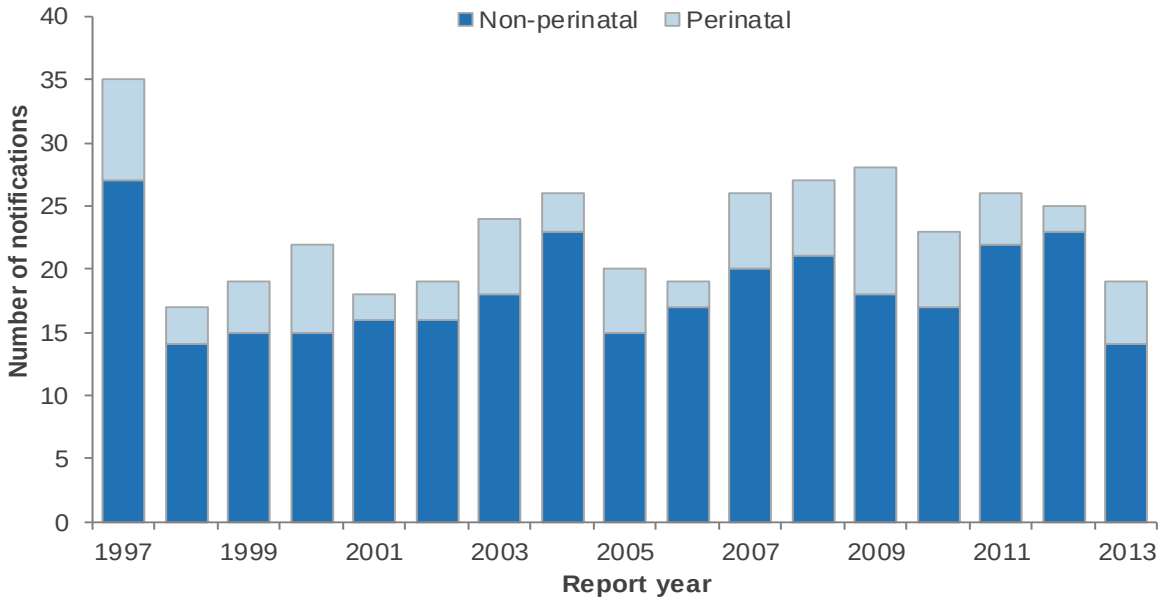
The 2005 Risk Profiles presented listeriosis notification data for the period 1990 to 2004. Table 6 presents data for 2005-2013. Figure 4 displays notified listeriosis cases for the period 1997-2013, identifying the proportion of cases that were perinatal or non-perinatal. Since 1998 the notification rate for listeriosis has been stable, ranging from 0.4 to 0.7 per 100,000.

Table 6: Number of reported cases and rates of invasive listeriosis, 2005-2013

Year	Listeriosis cases	Rate (cases/100,000)
2005	20	0.5
2006	19	0.5
2007	26	0.6
2008	27	0.6
2009	28	0.6
2010	23	0.5
2011	26	0.6
2012	25	0.6
2013	19	0.4

Note to Table 6: Rate data for 2005 to 2008 are from (Gilbert *et al.*, 2009). Case and rate data for 2009-2013 are from (ESR, 2010, 2011, 2012, 2013, 2014)

Figure 4: Reported invasive listeriosis cases by year, 1997-2013



Note to Figure 4: Figure is reproduced from (ESR, 2014)

New Zealand notification data support the assertion that pregnant women and their foetuses, the elderly, and people with an underlying illness are at greater risk. In 2012, 13/23 (57%) non-perinatal cases were aged 70 years and over. In the same year, 16/23 (70%) of non-perinatal cases had an underlying illness such as cancer, autoimmune disease, Crohn's disease, renal failure or other chronic illness (ESR, 2013). Between 2008 and 2012 the risk factor most commonly associated with listeriosis was having an underlying illness. Receiving immunosuppressive drugs and admission to hospital for treatment of another illness were also commonly reported risk factors (Lopez *et al.*, 2013).

While the number of reported listeriosis cases is small compared to other notifiable diseases, the clinical outcomes are often severe. The 2005 Risk Profiles presented hospitalisation and mortality data for the period 1997 to 2004. There has been very little change during subsequent years. Where outcome is known, the proportion of cases hospitalised annually is high (>85%) and a small number of people have died each year (Table 7). Listeriosis is often only diagnosed once patients are admitted to hospital.

Table 7: Listeriosis cases that resulted in hospitalisation and death, 2005-2013

Year	Number of cases	Number hospitalised (%)*	Number of deaths (% infections resulting in death)		Reference
			Non-perinatal	Perinatal	
2005	20	13/15 (87)	1/15 (7)	0/5	(ESR, 2006)
2006	19	16/17 (94)	0/17	1/2 (50)	(ESR, 2007)
2007	26	19/19 (100)	2/20 (10)	2/6 (33)	(ESR, 2008)
2008	27	17/20 (85)	3/21 (14)	2/6 (33)	(ESR, 2009)
2009	28	17/18 (94)	2/18 (11)	2/10 (20)	(ESR, 2010)
2010	23	15/17 (88)	3/17 (18)	4/6 (67)	(ESR, 2011)
2011	26	22/22 (100)	1/20 (5)	0/6	(ESR, 2012)
2012	25	22/23 (96)	4/23 (17)	2/2 (100)	(ESR, 2013)
2013	19	14/14 (100)	2/14 (14)	3/5 (60)	(ESR, 2014)

* Hospitalisation is not always reported. The denominator is the number of cases for which hospitalisation was recorded. Hospitalisation may not be reported for perinatal cases.

3.3.3 Reported outbreaks

The 2005 Risk Profiles reported three listeriosis outbreaks in New Zealand between 1992 and 2003. None of these were associated with cheese. Two additional outbreaks have been reported since: One in 2009 (2 cases) where a suspected food was not identified, and one in 2012 (6 cases) reported to be foodborne (ESR, 2013; Lim *et al.*, 2012; Lim *et al.*, 2011).

3.3.4 Serotypes

ESR's Special Bacteriology Laboratory tests *L. monocytogenes* isolates sent from public health laboratories to see whether they are serotype 4 or 1/2. They do not test for other serotypes. The 2005 Risk Profiles reported that clinical isolates of *L. monocytogenes* for the period 1999-2003 were approximately evenly split between the 1/2 and 4 serotypes. In the period 2004-

2012, each year between 19 and 29 isolates have been tested and serotype 4 has been detected in the largest proportion (58-86%), except in 2012 (48%).

3.4 *L. monocytogenes* Infection Overseas

KEY FINDINGS

Of 15 outbreaks of listeriosis associated with consumption of cheese, all but one were caused by cheeses with a high moisture content, and often implicated cheeses were manufactured without the use of starter cultures. The treatment of the milk used for making the cheeses was known for eight of the outbreaks, and 7/8 were caused by pasteurised milk cheeses which suggests that environmental contamination by *L. monocytogenes* had occurred. This differs from the 2005 Risk Profiles, where 6/9 of the reported outbreaks overseas involved raw milk cheeses.

The incidence of listeriosis in New Zealand is comparable to that reported in other countries, but continues to be at the higher end of the range of reported rates. However, reporting practices can differ between countries.

Appendix 2 contains detailed data summarised in this section.

4 EVALUATION OF RISK

4.1 Existing Risk Assessments

KEY FINDINGS

Available risk assessments for *L. monocytogenes* in cheese have identified that the greatest risk is from soft ripened cheese, particularly those made from raw milk. However, exposure to high numbers of *L. monocytogenes* is required to generate significant risk of illness.

Appendix 2 contains detailed data summarised in this section.

4.1.1 New Zealand risk assessment

No quantitative risk assessments for *L. monocytogenes* in cheese have been conducted in New Zealand.

4.1.2 Risk assessments from other countries

The 2005 Risk Profiles discussed two risk assessments for soft cheeses consumed in France, and another two risk assessments for *L. monocytogenes* in ready-to-eat foods.[19] The FAO/WHO risk assessment was in draft form at the time and has been included in this update alongside more recent risk assessments for *L. monocytogenes* in cheese for Australia, and an additional risk assessment for *L. monocytogenes* in ready-to-eat foods. See Appendix 2, Section 8.3 for details, but in summary:

- Australia (quantitative risk assessment, raw milk cheeses). The assessment concluded that the key determinant for the safety of raw milk cheese was the microbiological quality of the raw milk, but a combination of hurdles during cheese making had the greatest impact on pathogen survival. Raw milk Swiss-type cheeses with a low curd cooking temperature, blue, feta and Camembert cheese pose a high risk to susceptible populations due to the survival and/or growth of *L. monocytogenes* during cheese making. *L. monocytogenes* was found to be a negligible to low risk for the general population for all raw milk cheeses assessed (see section 8.3)
- North America (quantitative risk assessment, soft-ripened cheeses).[20] The predicted number of servings needing to be consumed to result in one case of listeriosis was much fewer for cheeses made from raw milk than cheeses made from pasteurised milk (reflecting greater contamination of the former), and the predicted number of servings was also fewer for immunocompromised populations when compared to the general population.
- FAO/WHO (quantitative risk assessment, ready-to-eat foods). Nearly all listeriosis cases predicted by the model resulted from consuming high numbers of *L. monocytogenes*. The risk assessment did not specifically address cheese, but included risk assessments for pasteurised milk, ice cream, semi-dry fermented meat and cold smoked fish, as example RTE foods.

[19] There were plans for the FDA/FSIS risk assessment to be updated at the time of the preparation of this report, see https://www.federalregister.gov/articles/2011/04/07/2011-8360/update-of-the-2003-interagency-quantitative-assessment-of-the-relative-risk-to-public-health-from (accessed 18 February 2014).

[20] This risk assessment, carried out by USFDA and Health Canada, expressed risk in terms of the number of servings consumed to result in a case of listeriosis. A small number of servings suggests that the risk is high, while a large number of servings suggests the risk is low.

- EU (scientific opinion, ready-to-eat foods). Most listeriosis cases were due to consumption of RTE foods able to support *L. monocytogenes* growth and containing concentrations well above 100 CFU/g.

4.2 Evaluation of Risk for New Zealand

KEY FINDINGS

The previous Risk Profiles concluded that the risk of *L. monocytogenes* infection from soft and low moisture cheese in New Zealand was low. Subsequent human health surveillance data suggest that this situation continues, as there has been only one case and no outbreaks of listeriosis linked to cheese consumption. A high proportion (approximately 80-90%) of cheese consumption in New Zealand is hard, semi-hard, cheese with eyes, and low pH varieties, which are unlikely to support growth of *L. monocytogenes*.

4.2.1 Risk associated with cheese

The 2005 Risk Profiles concluded the following:

- Soft cheeses: Transmission of *L. monocytogenes* by soft cheese has the potential to contribute to a proportion of invasive listeriosis cases, but the current risk of infection via this transmission route in New Zealand for the general population is low (although the risk will be greater for susceptible populations).
- Low moisture cheeses: 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 large number of cheese types and manufacturing processes means that broad statements about risk covering all cheeses are difficult to make. Instead we consider risk according to contributing factors:

- Ingredients
- Cheese types
- Processes and manufacturing environment
- Consumption

Ingredients

Pasteurisation will eliminate *L. monocytogenes*, but cheeses made from raw milk could be contaminated by this organism. Two surveys of raw cows' milk in New Zealand have found prevalences of *L. monocytogenes* of 0.7% and 4.1% and the concentration of *L. monocytogenes* in positive samples was <1 CFU/mL or <1 MPN/mL. One survey of raw goats' milk found a prevalence of 3.3%. Thus there is a risk that raw milk used for cheese manufacture will be contaminated with *L. monocytogenes*.

Other ingredients (e.g. rennet, salt, brine, colour, cultures) are added to cheese after pasteurisation and potential *L. monocytogenes* contamination of these should be considered as part of any risk management programme. If a contaminated ingredient

was added to a cheese in which *L. monocytogenes* may grow then exposure of the population to the pathogen would be increased. This has been shown by the contamination of red smear organisms being applied to freshly produced cheeses in the "old-young" process. There has been a report of an outbreak involving smear ripened cheese (Koch *et al.*, 2010).

Cheese types

Overseas outbreaks of listeriosis have been most commonly associated with soft high moisture cheeses, particularly acid or acid/heat coagulated cheeses, i.e. those where curd formation does not use starter cultures, such as the Hispanic soft cheeses. These categories are also considered higher risk by published risk assessments.

Processes and manufacturing environment

Lactic acid concentration/pH and water activity appear to be the most important factors affecting whether *L. monocytogenes* will grow during manufacture, ripening and storage. This applies to bacteria within or on the surface of cheese, although pH and moisture can change considerably at the surface during ripening and storage. Achieving a reduction of pH to <5 during fermentation through the use of starter culture is an important control step. Many, but not all, cheeses are inoculated with a starter culture that converts lactose into lactic acid responsible for a reduction in pH.

The rate of any growth or decline of *L. monocytogenes* in cheese once it has been made will be most affected by temperature.

Hygiene in the manufacturing environment is a critical factor in controlling post-pasteurisation contamination. Ripening and storage areas are important potential sources of contamination.

Raw milk cheese manufacturers in New Zealand have to be individually approved by MPI and so their processes will be examined with respect to *L. monocytogenes* growth. This is likely to control risk from locally made raw milk cheeses, and the risk attributed to these processors will also be low at a national level, due to the small number of processors and products.

Consumption

Analysis of nutrition survey data indicates that hard and semi-hard types of cheese (particularly Cheddar) are the most commonly consumed types of cheese in New Zealand. These types present a lower risk for exposure to *L. monocytogenes* due to low water activity, as will the next most commonly consumed category, cheese with eyes (e.g. Edam, Emmental, Gouda).

After hard/semi-hard cheeses, and cheese with eyes, cottage and cream cheese are the next most commonly consumed types. In the absence of low water activity as a control, the acid content of these cheeses is the primary means of preventing growth. If *L. monocytogenes* is present, the numbers will decrease during storage of the final product (Hicks and Lund, 1991; Hudson *et al.*, 2011).

The soft cheeses such as Brie, Camembert and blue are a minor proportion of the total cheese servings, and the consumption of soft cheese does not appear to have changed markedly based on the nutrition survey data. However, the number of specialist cheese manufacturers in New Zealand appears to be increasing, as is the popularity of farmers markets, suggesting that the range and amount of consumption of non-Cheddar cheese is rising.[21] Some of these cheeses will allow growth of *L. monocytogenes*.

The previous Risk Profiles concluded that the risk of *L. monocytogenes* infection from soft and low moisture cheese in New Zealand was low. Available human health surveillance data from New Zealand since the previous documents suggests that this situation continues, as there has been only one case and no outbreaks of listeriosis linked to cheese consumption. A high proportion (approximately 80-90%) of cheese consumption in New Zealand is hard, semi-hard, cheese with eyes, and low pH varieties, which present little risk of growth by any contaminating *L. monocytogenes*. A notable change is the increasing volume of cheese being imported into New Zealand. Some of these cheeses will be soft varieties that present a greater risk of *L. monocytogenes* contamination. Two of three recalls of cheese for *L. monocytogenes* contamination from 2008 – 2014 involved imported cheese.

4.2.2 Risks associated with other foods

The following information expands on that presented in the 2005 Risk Profiles.

Foods that pose a high risk for causing listeriosis have the following properties (ILSI Research Foundation/Risk Science Institute expert panel on *Listeria monocytogenes* in foods, 2005):

- Have the potential for contamination with *L. monocytogenes*;
- Support the growth of *L. monocytogenes* to high numbers;
- Are ready-to-eat;
- Require refrigeration; and
- Are stored for an extended period of time.

Listeriosis is considered to be primarily a foodborne disease and ready-to-eat foods are considered high risk because many of these foods support the growth of *L. monocytogenes*, even when stored under refrigeration.

A recent analysis of 503 *L. monocytogenes* isolates from foods, food contact surfaces and clinical cases in New Zealand found that one particular PFGE type was detected in clinical cases from 1999 to 2013 but had not been detected in foods. The transmission route may be an as-yet unidentified food (Hudson and Gilpin, 2013).

Aside from cheeses, RTE meats (including fish) and other dairy products are potential vehicles of infection. In 2008, a listeriosis outbreak in Canada was caused by ready-to-eat meats and resulted in 58 confirmed cases and caused 22 deaths (Weatherill, 2009). The importance of RTE Meats and dairy products is emphasised by a number of studies:

[21] http://www.newzealandholidaytravel.com/pages/Best-Cheeses-in-New-Zealand accessed 28 May 2014

- A USA risk ranking exercise ranked deli meats, unheated frankfurters, pate and meat spreads, unpasteurised fluid milk, smoked seafood and cooked ready-to-eat crustaceans as high risk for listeriosis (per serving basis) (FDA/USDA, 2003).
- An attribution exercise based on *L. monocytogenes* surveillance data from England and Wales concluded that the most important food sources were multicomponent foods (sandwiches and prepacked mixed salad vegetables), finfish and beef (Little *et al.*, 2010). Beef, milk and milk products, and finfish were important sources of infection for pregnancy-associated cases.
- An attribution exercise using USA outbreak data found that 6/14 (43%) listeriosis outbreaks that could be attributed to a single commodity were attributed to the commodity group 'dairy' (which will include milk, cheese and other dairy products) (Gould *et al.*, 2013). Five outbreaks were attributed to 'poultry' and the remaining three to 'beef', 'pork' and 'sprout'. The analysis identified *Listeria* and 'poultry' as the pathogen-commodity pair responsible for the most deaths (16) of all pathogen-commodity pairs.

A variety of other foods have also been implicated in outbreaks of listeriosis. These include raw milk, pasteurised milk, cantaloupe, sandwiches, diced celery and imitation crab meat (CDC, 2012a; Cumming *et al.*, 2008; Farber *et al.*, 2000; Gaul *et al.*, 2013; King *et al.*, 2014; Shetty *et al.*, 2009). Produce outbreaks are often linked to poor growing or storage conditions, or environmental cross-contamination after processing.

4.3 The Burden of *L. monocytogenes* Infection in New Zealand

KEY FINDINGS

On a national scale (and on the basis of existing information), the burden of disease from cheeses contaminated with *L. monocytogenes* is considered to be low relative to other sources of *L. monocytogenes* infection. The burden of disease from all foodborne listeriosis in New Zealand is fourth on a ranked list of six enteric foodborne diseases, a position largely determined by the high mortality rate.

4.3.1 Burden of disease from cheese contaminated with *L. monocytogenes*

On a national scale (and on the basis of existing information), the burden of disease from cheeses contaminated with *L. monocytogenes* is low relative to other sources of *L. monocytogenes* infection because only one case and no outbreaks of listeriosis have been attributed to cheese consumption from 2006 – 2013.

4.3.2 Burden of disease from all *L. monocytogenes* infections

It has been estimated by expert consultation that 85% (minimum 78%, maximum 92%) of listeriosis incidence is due to foodborne transmission (Lake *et al.*, 2010). A recent analysis of the burden of foodborne disease in disability adjusted life years (DALYs) used data from 2011 and multipliers from recent studies to estimate cases not reported to the health system (Cressey, 2012). The total burden of disease from listeriosis and sequelae was calculated as 188 DALYs, with 160 DALYs (5^{th}-95^{th} percentile 31-305) being foodborne. For comparison, the next largest DALYs estimate for foodborne-associated disease was for STEC infection (200, 5^{th}-95^{th} percentile 1.5-783), followed by campylobacteriosis (587, 5^{th}-95^{th} percentile 425-781) and norovirus infection (873, 5^{th}-95^{th} percentile 675-1083). The DALYs estimate for foodborne

listeriosis was higher than that of salmonellosis and yersiniosis. The annual rate of listeriosis (0.4-0.7 per 100,000) is currently lower than all of these diseases (e.g. the 2012 salmonellosis rate was 24.5 per 100,000 (ESR, 2013)) but the DALY value for listeriosis is elevated by the high proportion of fatalities. For most foodborne diseases the burden due to morbidity is the greater part of the burden of disease estimate. For perinatal listeriosis, mortality accounts for more than 99% of the DALY estimate.

An estimate of the total economic cost to New Zealand of six foodborne diseases has been published (Applied Economics, 2010). This estimate converted the individual burden in DALYs to an economic value and was based on data from 2009. Of the estimated total cost ($161.9m), listeriosis accounted for $15.2 million (9%), reflecting the high cost of patient care and the risk of premature death. This estimate was similar to those for salmonellosis ($15.4m) and STEC infection ($14.6m).

These estimates cover all potential food vehicles. There are no separate estimates for transmission of *L. monocytogenes* via cheese.

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

4.4 Data Gaps

KEY FINDINGS

Continuing data gaps identified in this report that impact on the statement of risk are:

- Prevalence and concentration of *L. monocytogenes* in New Zealand cheeses; and,
- Production data for different types of cheeses available in New Zealand.

The data gaps identified in the 2005 Risk Profiles and updated commentary on these are presented in Table 8.

Table 8: Data gaps identified in the 2005 Risk Profiles

Data gap	Commentary
Prevalence and quantitative data on *L. monocytogenes* in soft cheeses sold in New Zealand.	One survey has been conducted since the previous Risk Profiles.
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.	No specific surveys have been conducted since the previous Risk Profiles.
Quantitative data on levels of *L. monocytogenes* in low moisture cheeses when contamination does occur.	No New Zealand data. An Irish study suggests numbers are low (Dalmasso and Jordan, 2014).
Prevalence and concentration of *L. monocytogenes* in raw milk in New Zealand.	Two raw milk surveys have been completed. See Section 2.4.1 and (King *et al*., 2014)
Information on environmental *L. monocytogenes* contamination in New Zealand cheese production sites and associated areas.	No New Zealand data.
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).	Few data are available on cheese production volumes.

Continuing and additional data gaps identified in this report that impact on the statement of risk are:

- Prevalence and concentration of *L. monocytogenes* in New Zealand cheeses; and,
- Production data for different types of cheeses available in New Zealand.

5 AVAILABILITY OF CONTROL MEASURES

5.1 Current Control Measures

KEY FINDINGS

Since the 2005 Risk Profiles, a number of notices and approved criteria have been published that stipulate requirements for farm dairies and dairy product manufacturers. Some cheeses made from raw milk are now permitted to be manufactured in New Zealand or imported.

There are general hygiene requirements for suppliers of milk to cheese makers that will help prevent the contamination or growth of *L. monocytogenes* in the milk, and additional requirements for suppliers of milk for the manufacture of raw milk cheese. Milk suppliers, including those supplying milk for the manufacture of raw milk cheeses, do not have to test the milk for *L. monocytogenes* unless the manufacturer requires them to.

Standards for the heat treatment of thermised milk for cheese making have now been set. Thermised milk is only permitted for the manufacture of cheeses with <39% moisture and pH <5.6, and where the pH of the cheese does not increase on ripening.

A Product Safety Limit (PSL) is in place for *L. monocytogenes* in cheese manufactured in New Zealand: Not detected in 25 g samples throughout the product shelf life. However, a potential change to this PSL to 100/g has been signalled and would align with recent changes to Standard 1.6.1 of the Australia New Zealand Food Standards Code. The PSL applies to all cheeses manufactured under a Risk Management Programme.

There are relevant food safety controls for *L. monocytogenes* in cheese in the areas of:

- Milk supply for cheese making;
- The production and sale of cheeses; and
- Imported cheeses.

The presence of foodborne pathogens in raw milk results from contamination from the environment and/or to direct excretion from the udder of infected dairy animals (Oliver *et al.*, 2005). If contaminated raw milk is made into cheese then the contaminants may grow during production, depending on the cheese type, and increase exposure to the pathogen. Depending on the conditions used, heat-treated milk can effectively be free of pathogens (e.g. pasteurised milk) and for cheese made from treated or raw milk a contaminated product can result if there is a hygiene failure during manufacture, ripening, or distribution of the cheese. Therefore controls need to be implemented to cover each of these segments of the food chain.

The 2005 Risk Profiles described a number of legislative controls and guidelines. The *Animal Products Act 1999* and *Food Act 1981* are still the main legislative tools that provide for microbiological controls for milk and cheese production in New Zealand, but a number of new regulations have been set since the 2005 Risk Profiles.[22] The New Zealand (Milk and Milk Products Processing) Food Standards 2002 have been revoked. A notable change is that some cheeses made from raw milk can now be manufactured in New Zealand and imported.

[22] The requirements for farm dairies and dairy manufacturers are fully described at http://www.foodsafety.govt.nz/industry/sectors/dairy/ (accessed 19 August 2014).

5.1.1 Controls concerning milk supply for cheese making

Farm dairy operators, including those that supply milk used for cheese making, must operate under a MPI-registered Risk Management Programme (RMP) under the *Animal Products Act 1999*.[23] Two Notices have now been issued that describe the specifications that farm dairies must meet when developing a RMP (MAF, 2011b; NZFSA, 2008b). Controls relevant to this Risk Profile include:

- Requirements that milk must come from animals free of diseases caused by pathogens, which may contaminate milk; and
- Milk is to be cooled at prescribed rates to achieve target temperatures.

Approved criteria have also been issued, to elaborate the technical outcomes specified in the Notices (MAF, 2011a; NZFSA, 2008a). While no specific testing requirement for *L. monocytogenes* is specified, raw milk for the manufacture of dairy products must contain an aerobic plate count (30ºC Bactoscan®) of no more than 100,000 CFU/mL (NZFSA, 2008a)

L. monocytogenes is able to infect and cause disease, including mastitis in milk-producing animals (Thompson *et al.*, 2009; Winter *et al.*, 2004). However, subclinical carriage of *L. monocytogenes* has also been reported and implicated as the cause of subsequent contamination of raw milk cheeses (Pintado *et al.*, 2009). Consequently, animal health controls cannot be assumed to prevent excreted *L. monocytogenes* contaminating milk.

Refrigeration of raw milk will slow the growth of *L. monocytogenes* if present in the milk, but not prevent growth.

5.1.2 Farms supplying milk for the manufacture of raw milk cheeses

The Animal Products (Raw Milk Products Specifications) Notice 2009 sets out additional requirements for farm dairies that supply milk for use in manufacturing raw milk products (NZFSA, 2009b). On farm controls include veterinary inspection of animals, ensuring animal feed is not a vector for pathogens, avoiding the use of silage, correct hygiene during milking, ensuring milk is cooled and stored at the correct temperature.

The MPI website also contains supporting material for this Notice, in the form of a Code of Practice (NZFSA, 2010b) and a technical report on on-farm provisions for raw milk production (Compton *et al.*, 2008).

While the requirements in the Notice are more stringent than those outlined in section 5.1.1 above, comments relating to asymptomatic carriage of *L. monocytogenes* and the ability of the organism to grow at refrigeration temperatures are equally applicable.

5.1.3 Controls concerning the production and sale of cheeses

Cheese makers who sell their product on the domestic market (New Zealand and Australia) can opt to operate under a RMP or a Food Safety Programme (FSP) under the *Food Act 1981*. Cheese makers who export their product must operate under a RMP. All dairy businesses must meet legal requirements, which ensure their dairy products are safe and suitable for human

[23] http://www.foodsafety.govt.nz/industry/sectors/dairy/farms-dairies/ accessed 12 November 2014

consumption. Legislation, notices, specifications, and approved criteria that set out the requirements for dairy manufacturers are presented on the MPI website.[24] Some specific technical aspects relevant to *L. monocytogenes* control are outlined below.

The Animal Products (Dairy Processing Specifications) Notice 2011 includes technical requirements for pasteurisation and for thermisation for cheese making (MAF, 2011b). Approved criteria for the operation of dairy heat treatments are included in a separate document (NZFSA, 2010c), while further guidance on the design and operation of dairy heat treatments are also provided (NZFSA, 2003, 2009e).

For dairy products, including cheeses, manufactured under a RMP, microbial Product Safety Limits (PSL) are outlined in an approved criteria document (MAF, 2011a). The PSLs must not be exceeded at any time during the product's shelf life. *L. monocytogenes* must not be detected in composite 25 g samples of cheese throughout the product's shelf-life, irrespective of whether the cheese is produced using thermised or pasteurised milk. The approved criteria document further notes that "Limits of ND/25g and 100/g have now been adopted by the Joint FAO/WHO Food Standards Programme, Codex Committee on Food Hygiene in the "Draft Guidelines for the Control of *Listeria monocytogenes* in Foods". 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. MAF through its *Listeria* Strategy will be developing policy on how the limit of 100/g may be applied to foods and to revise the criteria within the FSC". 'The FSC' is the Australia New Zealand Food Standards Code. The approved criteria document further notes that "All dairy products manufactured in New Zealand, must comply with microbiological limits specified in the Australia New Zealand Food Standards Code (FSC).

Standard 1.6.1 of the Food Standards Code includes Microbiological Specifications for Ready-to-Eat foods (FSANZ, 2014). In May 2013, Proposal P1017 was adopted to amend Standard 1.6.1 with regards to criteria for *L. monocytogenes* limits in ready-to-eat foods. It replaced existing food specific limits for *L. monocytogenes* with limits for all RTE foods based on the Codex Standard discussed above. There are two sets of criteria for *L. monocytogenes* based on whether growth of *L. monocytogenes* will occur or not in the RTE food:

- Ready-to-eat foods in which growth of *L. monocytogenes* will not occur (n=5, c=0, m= <100 CFU/g)
- Ready-to-eat foods in which growth of *L. monocytogenes* can occur (n=5, c=0, m=absence in 25g)

This approach recognises that it is the potential for foods to support growth of *L. monocytogenes* that is a main factor in the risk of acquiring listeriosis. For foods in which the growth of *L. monocytogenes* will not occur, occasional low level detections (<100 CFU/g) do not present a public health risk.

Ready-to-eat foods in which *L. monocytogenes* growth will not occur are characterised by the following:

(a) the food has a pH < 4.4 regardless of water activity; or

[24] http://www.foodsafety.govt.nz/industry/sectors/dairy/manufacturing/ accessed 12 November 2014

(b) the food has a water activity < 0.92 regardless of pH; or

(c) the food has a pH < 5.0 in combination with a water activity of < 0.94; or

(d) the food has a refrigerated shelf life ≤ 5 days; or

(e) the food is frozen (including foods consumed frozen and those intended to be thawed before consumption); or

(f) it can be validated that the level of *Listeria monocytogenes* will not increase by > 0.5 log over the food's stated shelf life; or

(g) the food has not had a listericidal treatment and it can be validated that the level of *Listeria monocytogenes* will not exceed 100 CFU/g throughout the food's stated shelf life.

MPI has also published guidance for the control of *L. monocytogenes* in RTE foods for food operators who produce RTE foods that are not intended to be consumed immediately and that will be stored refrigerated for more than five days before consumption (i.e. cheeses) (MPI, 2012a, 2012b, 2013d).

5.1.4 Manufacturers of raw milk cheeses

The Animal Products (Raw Milk Products Specifications) Notice 2009 sets out additional requirements for manufacturers of raw milk products and is supported by a code of practice (NZFSA, 2009b, 2010b).

There are general controls that will reduce the opportunity for *L. monocytogenes* to contaminate or grow in the raw milk cheese, e.g. only using raw milk from a farm dairies approved to supply milk for the manufacture of raw milk products, controlling the temperature and age of the raw milk, using a starter culture, monitoring the pH and a_w of the ripening cheese, and having a plan to monitor and control pathogens in the manufacturing environment.

The Notice also includes specific food safety criteria, including a requirement that *L. monocytogenes* not be detected in any of five 25 g samples (tested as a composite) taken over the lot while under the control of the manufacturing processor.

5.1.5 Controls for imported cheese

The Food (Imported Milk and Milk Products) Standard 2009 has been introduced and sets out requirements for all milk and milk products imported into New Zealand for sale (NZFSA, 2009d). All cheeses imported into New Zealand must be made from pasteurised or UHT milk, unless the cheeses have been manufactured using an approved alternative processing method that ensures at least an equivalent level of safety protection for consumers as cheese using pasteurised or UHT milk. Some raw milk cheeses have now been approved for importation provided they comply with specified overseas sanitary standards: Emmental, Gruyére and Sbrinz cheeses from Switzerland, and Roquefort cheese (a soft cheese made from raw sheep milk) and extra hard Parmesan style raw milk cheeses (Grana Padano, Pamigiano Reggiano, Romano, Asiago and Montasio) from EU member countries.

Soft and grated cheeses imported and sold in New Zealand must be monitored for the presence of pathogenic bacteria on or in the cheeses or conditions that can sustain or promote their growth (NZFSA, 2007b). Similarly, raw milk cheeses imported and sold in New Zealand are

now considered to be prescribed foods and must be monitored for the presence of pathogenic bacteria (NZFSA, 2007b, 2009a). There are Imported Food Requirements that support each of these cheese types (MPI, 2013b, 2013c). Unless from Australia or supported by a recognised assurance/certification or a multiple release permit, soft cheeses imported into New Zealand will be tested for *L. monocytogenes* according to MPI's sampling and testing protocol (MPI, 2013b). There is a nil tolerance for *L. monocytogenes*. Raw milk cheeses may only be imported to New Zealand from countries that have negotiated a pre-clearance agreement with MPI; these countries (the EU and Switzerland) have controls for *L. monocytogenes* within their laws that have been accepted by MPI as being equivalent to New Zealand public health standards (MPI, 2010, 2013c).

5.2 Additional Options for Risk Management

KEY FINDINGS

There are no on-farm practices that can guarantee that raw milk will be free from pathogens but there are practices that will reduce opportunities for milk contamination.

There are additives and treatments that can be used to control *L. monocytogenes* in cheeses alongside traditional hurdles and some of these are listericidal. Keeping cheeses under refrigeration at retail will limit growth at this stage of the food chain in those cheeses in which *L. monocytogenes* can grow.

Consumer advice on cheese selection and consumption is available to populations who are more at risk from listeriosis, but a 2008 study found that awareness of the risk of listeriosis from raw milk cheeses was low.

The risk management options presented in the 2005 Risk Profiles assumed that environmental contamination was the most likely source of contamination given the requirement at the time for most cheeses to be made from pasteurised milk. Discussion of risk management options was largely confined to options under the existing legislation and consumer awareness.

This section contains information on controls that might be applied on farms producing milk for cheese making, by cheese manufacturers and at retail, plus contains new information on consumer advice. Environmental monitoring and end-product testing are two important control measures which have already been discussed in Section 5.1.

5.2.1 On-farm

On-farm controls are particularly applicable to milk that is intended to be used for cheese making without pasteurisation. On-farm control options for reducing the risk of *L. monocytogenes* contaminating raw milk have been discussed in the raw milk Risk Profile (King *et al.*, 2014).

Additional on-farm controls apply where the cheese making facility is located on the same property as the milking herd. Biosecurity measures to prevent *L. monocytogenes* from entering and colonising the cheese making facility might include:

- Ensuring the building is completely enclosed (walls, windows, roof);

- Establishing a hygiene barrier at the entrance to the cheese making facility that includes disinfection trays and hand washing stations, and requires clothing and footwear to be changed (Schoder *et al.*, 2011);
- Establishing a physical barrier around the cheese making facility to prevent stock from entering the area, and banning the presence of farm machinery, stock feed and other farming supplies from this area.

These measures are well aligned the NZFSA (now MPI) Operational Guideline: Design and Construction of Dairy Premises and Equipment (NZFSA, 2006). Under these guidelines an on-farm cheese making facility would be considered to be a Critical Hygiene Area.

5.2.2 Cheese manufacture

There are many recent reports in the literature describing new methods for controlling *L. monocytogenes* in cheeses. Some are summarised in Table 16 in Appendix 3, along with examples of studies that show their effectiveness. Many of these methods are biological (i.e. bacteria, plant extracts) and these may be more acceptable to consumers. A report giving examples of published cheese challenge studies provides some evidence concerning the factors inhibiting the growth of *L. monocytogenes* during cheese production and maturation/ripening.[25]

5.2.3 Handling at retail

A proportion of cheese available at retail will have been cut and/or repackaged at the retail premises. There is potential for *L. monocytogenes* contamination to occur during such processes, as has been demonstrated for ready-to-eat meats (Lin *et al.*, 2006). Cheeses that are cut to order and displayed or stored at temperatures above 8°C have been shown as more likely to be microbiologically unacceptable according to EU criteria (Little *et al.*, 2008). Appropriate hygienic measures and appropriate temperature control are critical for minimising contamination with and growth of *L. monocytogenes*.

5.2.4 Consumer advice

The 2005 Risk Profiles reported that advice for pregnant women on the risks of consuming soft cheeses was available. New information for consumers has since been published:

- Information on raw milk products, that includes advising high risk groups to avoid consuming these products;[26] and
- Advice specifically targeted to pregnant women and people with low immunity on consuming different types of cheese (MPI, 2013a; NZFSA, 2007a).

Labelling of raw milk cheeses must also include a statement that the milk is raw or unpasteurised in the ingredients declaration (NZFSA, 2009b). A 2008 social study on raw milk products (NZFSA, 2009c) found:

[25] http://foodsafety.govt.nz/elibrary/industry/literature-resources-for-validation-of-raw-milk-products.pdf (accessed 4 November 2014)
[26] http://www.foodsmart.govt.nz/food-safety/high-risk-foods/raw-milk/questions-answers.htm (accessed 26 March 2014)

- The term "raw milk" was not well understood, and for labelling purposes, the term "unpasteurised milk" was favoured over "raw milk" and "non-heat treated milk";
- Some people buy raw milk products without knowing they are made from raw milk;
- Almost a third of the public thought that raw milk cheeses were as safe as pasteurised milk products; and
- There was a low awareness of advisory material on raw milk products and most of the general knowledge about raw milk products appeared to have come from the news media.

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7 APPENDIX 1: HAZARD AND FOOD

7.1 *L. monocytogenes*

There are ten species in the genus *Listeria*: *L. monocytogenes*, *L. ivanovii*, *L. innocua*, *L. seeligeri*, *L. welshimeri*, *L. grayi*, and four recently identified species, *L. marthii*, *L. rocourtiae*, *L. fleischmannii* and *L. weihenstephanensis* (Bertsch *et al.*, 2013; Graves *et al.*, 2010; Lang Halter *et al.*, 2013; Leclercq *et al.*, 2010; Orsi *et al.*, 2011). *L. fleischmannii* was isolated from cheese but did not show any indication of human pathogenicity or virulence (Bertsch *et al.*, 2013). Only *L. monocytogenes* and *L. ivanovii* are considered to be pathogenic. *L. ivanovii* is primarily an animal pathogen. *L. monocytogenes* is also an animal pathogen but is the most important species with respect to human health and most scientific studies focus on this species.

General information on the growth, survival and inactivation of *L. monocytogenes* is presented in the microbiological datasheet available from:
http://www.foodsafety.govt.nz/science-risk/hazard-data-sheets/pathogen-data-sheets.htm

The following new information is relevant to cheese:

- A study of the growth of 23 *L. monocytogenes* isolates from cheeses at 30°C in laboratory broth acidified with HCl confirmed that the minimum pH for growth under these conditions was pH 4.4 (Pintado *et al.*, 2005). N.B. the minimum pH will be higher than 4.4 for most cheeses as the acid present will be an organic acid.
- When *L. monocytogenes* (approximately 4 $\log_{10}$ CFU/mL) was inoculated into brines with different salt concentrations and stored at 4°C, the number of cells declined slowly. The reductions were to approximately 3 $\log_{10}$ CFU/mL over 90, 30 and 7 days in 13%,15% and 19% NaCl respectively (Durmaz *et al.*, 2009).
- The growth rates of *L. monocytogenes* isolates from cheeses and a cheese making environment varied when exposed to combinations of NaCl (5-10%) and pH (lactic acid or NaOH, pH 4-8.5) in laboratory broth (Ribeiro *et al.*, 2006). The pH was more important for growth than salt concentration for some isolates, but both were important for others. Overall, higher growth rates in the cheese isolates were observed under more acidic pH values and higher salt concentrations than in two laboratory reference strains, reflecting adaptation to conditions presented by the cheeses and cheese making environment.
- The recovery of *L. monocytogenes* from cheese is inhibited when cheeses are stored at -80°C (Rossmanith *et al.*, 2010). It is not known whether this is due to the freezing causing a longer lag phase (so the cells survive, but are not necessarily recovered using standard methods) or due to the cells dying in the cheese.

7.1.1 Disease and Pathogenicity

The disease resulting from infection, listeriosis, can manifest in two forms 1) an invasive disease which can result in the death of approximately 20% of cases and 2) a milder febrile gastroenteritis not known to cause fatalities.

In invasive listeriosis the clinical outcomes are principally intra-uterine infection, meningitis and septicaemia (McLauchlin *et al.*, 2004). In perinatal cases there can be a severe systemic infection in the unborn child or neonate, and a mild flu like illness in the mother. In adults

listeriosis causes central nervous system infection or septicaemia. Cases mostly occur in "at risk" groups; the immunosuppressed, those with malignant neoplasms, AIDS patients, diabetics, people with new heart valves or replacement joints, as well as people suffering from alcoholism/liver disease. A number of rarer presentations occur. The case fatality rate is between 20 and 40%.

The other form of the disease is an acute, self-limiting febrile gastroenteritis that occurs in otherwise healthy people (Ooi and Lorber, 2005). The predominant symptoms are fever (60-100% of cases), diarrhoea (33-88%), aching joints (20-100%) and headache (15-88%). The incubation period is usually around 24 h but has been reported to be as long as 10 days. Invasive disease can rarely follow these infections.

The serotype most often associated with cheese and cheese-related outbreaks is 1/2a (Acciari *et al.*, 2011; de Castro *et al.*, 2012; Fretz *et al.*, 2010; Jackson *et al.*, 2011; Lomonaco *et al.*, 2009). However this association is not exclusive as serotypes 1/2b (Gianfranceschi *et al.*, 2006; Pintado *et al.*, 2005) and 4b (Koch *et al.*, 2010; Little *et al.*, 2008; Pintado *et al.*, 2005) have also frequently been associated with cheese-related incidents. The 2005 Risk Profiles also reported these three serotypes as being frequently isolated from cheeses and listeriosis cases related to cheese consumption, so this does not appear to have changed.

L. monocytogenes isolates can be grouped according to phylogenetic characteristics. These are called lineages, and there are currently four (I-IV) recognised lineages (Orsi *et al.*, 2011). Molecular analyses are used to determine the lineage of a *L. monocytogenes* isolate. Lineages are closely correlated with serotypes (Table 9) (Nadon *et al.*, 2001).

Table 9: Characteristics of the four *L. monocytogenes* lineages

Lineage	Serotypes	Distribution of isolates
I	1/2b, 3b, 3c, 4b	Various sources; overrepresented among human isolates
II	1/2a, 1/2c, 3a	Various sources; overrepresented among food and food environments as well as natural environments
III and IV	4a, 4b, 4c	Most isolates from ruminants

Note to Table 9: Table is adapted from Orsi *et al.* (2011).

Orsi *et al*. (2011) provides a recent, critical review of the history and characteristics of these lineages. There is epidemiological, phenotypic and molecular evidence to support the hypothesis that lineage I isolates are more capable of causing disease in humans and that lineage II isolates are better adapted to survive in foods and food environments, but both lineages are important for human infection. The majority of human listeriosis outbreaks worldwide have been linked to lineage I serotype 4b isolates. Some outbreaks have been caused by lineage I serotype 1/2b isolates and lineage II serotype 1/2a isolates. Analysis of sporadic cases has revealed some regional variation. For example, lineage II serotype 1/2a strains appear to be more common among human listeriosis cases in Northern Europe, while lineage I strains dominate human listeriosis cases in the United States of America (USA). Lineage III strains have occasionally been isolated from human clinical cases, so these cannot be considered non-pathogenic to humans (Orsi *et al.*, 2011). No reports of Lineage IV strains isolated from human clinical cases were located.

Groups of genetically-related strains that have been implicated in geographically and temporally unrelated outbreaks have been assigned to "epidemic clones" (ECs) (Rocha *et al.*, 2013). These are distinguished by ribotyping or multi-virulence-locus sequence typing (MVLST). Seven ECs have been proposed to date (Lomonaco *et al.*, 2013).

A set of genetic markers to determine whether a strain of *L. monocytogenes* will cause human disease has not yet been identified. One gene that has received particular attention is *inlA*, coding for internalin A, which has a role in enabling *L. monocytogenes* to cross the intestinal barrier. A recent analysis of 1,009 *L. monocytogenes* isolates from human listeriosis cases and ready-to-eat (RTE) foods showed that a greater proportion of isolates from RTE foods carried a truncated form of this gene that makes them less invasive (Van Stelten *et al.*, 2010). This suggests that strains of *L. monocytogenes* carrying this mutation are less likely to cause human disease. However, expression of virulence genes is also influenced by environmental factors, e.g. increased transcription of some virulence genes was observed in four *L. monocytogenes* strains after cold (4°C) and freezing (-20°C) stress (Miladi *et al.*, 2013).

7.1.2 Viable but non-culturable (VBNC)

There is conflicting evidence about whether *L. monocytogenes* can be resuscitated following starvation and entry into the VBNC state (Cappelier *et al.*, 2005; Cappelier *et al.*, 2007; Lindback *et al.*, 2010; Rudi *et al.*, 2005). A real-time PCR-based approach to detect low numbers of VBNC *L. monocytogenes* on Gouda-like cheeses has been published (Rudi *et al.*, 2005). However, the ability of resuscitated *L. monocytogenes* to be infectious is still unclear.

7.2 *L. monocytogenes* in Cheeses Overseas

7.2.1 Detection of *L. monocytogenes* in cheeses overseas

Prevalence and concentration data for cheeses are difficult to summarise because of the variation in cheese types and characteristics. The 2005 low moisture cheese Risk Profile found that, overall, 0.3% of hard cheese samples were positive for *L. monocytogenes*, 5.5% of semi-soft/semi-hard cheeses were positive, and 9.0% of the smear (surface ripened) cheeses were positive. The 2005 soft cheese Risk Profile found the prevalence to be <10% in most of the studies. Some studies reported higher prevalences (>40%), most are data from South America and Spain. No cheeses from South America were imported into New Zealand between 2005 and 2013, but Spain exported fresh and processed cheese to New Zealand during this period (imports of fresh, unfermented cheese from Spain began in 2011 and 2.7 tonnes were imported in 2013).[27]

Table 10 summarises data published since 2005 on the prevalence and concentration of *L. monocytogenes* in cheeses. The table only includes cheese types likely to be imported into New Zealand and focuses on countries from which the majority of New Zealand's imported cheese originates (Australia, the USA and EU member states, see Section 2.2.4).[28] The classification of the cheeses in this table was done using available information.

[27] Statistics New Zealand Infoshare (http://www.stats.govt.nz/infoshare/Default.aspx, accessed 5 March 2014).
[28] There have been multiple surveys of cheeses from Turkey and South American countries published since 2005 but these have not been included in Table 10 since New Zealand does not receive cheese imports from these countries.

L. monocytogenes was not detected or was detected at a low prevalence (<2%) in most of the surveys in Table 10. While a particularly high prevalence (46%) was reported for surface ripened cheeses made from raw sheep milk in Portugal, it should be noted that many of the publications did not report the milk treatment (raw, thermised, pasteurised) so it is not possible to make any comment on whether the use of raw milk is associated with higher *L. monocytogenes* prevalences. When *L. monocytogenes* was enumerated in positive samples, most of these samples contained ≤100 CFU/g. Higher concentrations were not associated with any particular cheese type, and the highest concentration reported was 460 CFU/g in a sample of Taleggio (rennet coagulated, surface ripened) from Italy. Serotypes 1/2a, 1/2b, 1/2c and 4b were all detected.

Data for contamination of cheeses in Europe are available and shown in Table 10 (EFSA, 2013). These data were obtained for single samples, but the data have been re-analysed to assess compliance with an n=5 for the presence of *L. monocytogenes* at >100 CFU/g in at least one of the five samples. If it assumed that the results for individual samples within the batch are uncorrelated then the probability of compliance was estimated to be 0.997, while if the samples were perfectly correlated then the probability increased to 0.999. This reflects the low level of contamination of cheeses detected in the original survey.

Additional data are available through the EU food surveillance systems where cheeses can be tested singly or in batches at the farm, at the processing plant or at retail, and data for 2012 support the view the *L. monocytogenes* prevalence and concentration in cheeses is generally low (EFSA/ECDC, 2014). During the 2012 year, *L. monocytogenes* was detected in approximately:

- 1.7% of samples of soft and semi-soft cheeses made with raw or low heat treated milk;
- 0.1% of samples of soft and semi-soft cheeses made with pasteurised milk;
- 0.7% of samples of hard cheeses made with raw or low heat treated milk; and,
- 0.1% of samples of hard cheeses made with pasteurised milk.

In separate samples tested by enumeration, and categorised as ≤100 CFU/g and >100 CFU/g respectively (approximate-read from graph):

- 0.5% and 0.2% of samples of soft and semi-soft cheeses made with raw or low heat treated milk;
- 0.2% and 0.2% of samples of soft and semi-soft cheeses made with pasteurised milk;
- 0.5% and 0.8% of samples of hard cheeses made with raw or low heat treated milk; and,
- 0.2% and 0.1% of samples of hard cheeses made with pasteurised milk.

The prevalence and concentration data presented in the 2005 Risk Profiles included data from countries that do not export cheese to New Zealand. Even when omitting results from these countries, it appears that both the prevalence and concentration of *L. monocytogenes* in cheeses has reduced, perhaps due to efforts in these countries towards controlling *L. monocytogenes* in RTE foods. One consistent finding is that higher prevalence values are associated with surface ripened cheeses.

Table 10: Prevalence and concentration of *L. monocytogenes* in cheeses sampled in other countries (data published since 2005)

Cheese type	Milk used	Study location	Study period	Sample source	No. samples	Prevalence: No. positive (% positive)	Conc. (CFU/g)	Sero-type	Reference
Acid coagulated									
Unripened soft (fresh)	Raw or thermised	UK	2004	Retail outlets	62	1 (1.6)	ND 61 samples <100 1 sample	NR	(Little *et al.*, 2008)
Unripened soft (fresh)	Pasteurised	UK	2005	Retail outlets	412	1 (0.2)	ND 411 samples <100 1 sample	NR	(Little *et al.*, 2008)
Anthotyros Myzithra Cottage Katiki	NR for all cheeses	Greece	2010	Retail outlets	28 12 6 2	ND	NR	NR	(Angelidis *et al.*, 2012)
Acid/heat coagulated									
Manouri Ricotta	NR for all cheeses	Greece	2010	Retail outlets	23 2	ND ND	NR	NR	(Angelidis *et al.*, 2012)
Rennet coagulated – surface ripened									
Castelo Branco	Raw (sheep)	Portugal	1995/96	Cheese makers	63	29 (46)	NR	1/2a, 1/2b, 4b	(Pintado *et al.*, 2005)
Taleggio	NR	Italy	2005/06	Retail outlets	324	21 (6)	<0.36-460	1/2a, 1/2c	(Acciari *et al.*, 2011; Prencipe *et al.*, 2010)
Rennet coagulated – mould ripened (surface)									
Brie Camembert	NR	Italy	2005/06	Retail outlets	300 178	3 (1.0) ND	<0.36-110 NR	1/2a, 1/2b NR	(Acciari *et al.*, 2011; Prencipe *et al.*, 2010)
Brie Camembert	NR Pasteurised (cow)	Greece	2010	Retail outlets	10 6	ND ND	NR	NR	(Angelidis *et al.*, 2012)

Table 10 continued

Cheese type	Milk used	Study location	Study period	Sample source	No. samples	Prevalence: No. positive (% positive)	Conc. (CFU/g)	Sero-type	Reference
Rennet coagulated – mould ripened (internal)									
Gorgonzola	NR	Italy	2003/04	One manufacturer	1,489 at end of packaging 167 at end of shelf-life	31 (2) – end of packaging 8 (5) – end of shelf-life	NR	NR	(Manfreda *et al.*, 2005)
Gorgonzola	NR	Italy	2005/06	Retail outlets	444	21 (5)	0.36-9.3	1/2a, 1/2c	(Acciari *et al.*, 2011; Prencipe *et al.*, 2010)
Blue Gorgonzola Kopanisti Roquefort	NR for all cheeses NR for all cheeses NR Raw (sheep)	Italy	2010	Retail outlets	24 2 1 11	ND ND ND ND	NR	NR	(Angelidis *et al.*, 2012)
Blue vein	Pasteurised milk	Italy	Oct 2009 – April 2010	Single cheese factory	100 internal pastes 100 external rinds	0 (0) 55 (55)	NR	NR	(Bernini *et al.*, 2013)
Rennet coagulated – internally bacterially ripened – hard, semi-hard									
Semi-hard	Raw or thermised	UK	2004	Retail outlets	951	8 (0.8)	ND 943 samples <100 7 samples 220 1 sample	1/2a, 4b	(Little *et al.*, 2008)

Table 10 continued

Cheese type	Milk used	Study location	Study period	Sample source	No. samples	Prevalence: No. positive (% positive)	Conc. (CFU/g)	Sero-type	Reference
Semi-hard	Pasteurised	UK	2005	Retail outlets	584	2 (0.3)	ND 582 samples <100 2 samples	1/2a	(Little *et al.*, 2008)
Idiazabal (semi-hard)	Raw (sheep)	Spain	NR	Retail outlets	51	ND	NR	NR	(Arresse and Arroyo-Izaga, 2012)
Rennet coagulated – internally bacterially ripened – ripened under brine									
No studies found.									
Rennet coagulated – internally bacterially ripened – pasta filata									
Mozzarella	NR for all cheeses	Greece	2010	Retail outlets	10	ND	NR	NR	(Angelidis *et al.*, 2012)
Mixed cheese types or cheese not able to be classified									
Tulum (semi-hard cheese)	Raw milk	Turkey	March 2004-March 2005	Markets in Istanbul	250	12 (4.8%)	NR	NR	(Colak *et al.*, 2006)
Soft cheese, 1% salt	Pasteurised	Italy	2007 2008	At manufacturer	80 20	2 (3) 0[1]	NR NR[1]	NR	(Alessandria *et al.*, 2010)
Soft cheese Fresh cheese Sheep milk cheese	NR	Austria	2003/04	Retail outlets	200 25 27	11 (6) 1 (4) ND	<100 <100 NR	NR	(Wagner *et al.*, 2007)
Soft cheese Fresh cheese Sheep milk cheese	NR	Austria	2003/04	Households	33 27 9	ND ND ND	NR	NR	(Wagner *et al.*, 2007)
Fresh cheese	NR	Croatia	NR	NR	60	2 (3)	NR	NR	(Frece *et al.*, 2010)

Table 10 continued

Cheese type	Milk used	Study location	Study period	Sample source	No. samples	Prevalence: No. positive (% positive)	Conc. (CFU/g)	Sero-type	Reference
Soft cheese	NR	Wales	2008/09	Retail outlets	473	ND	NR	NR	(Meldrum *et al.*, 2010)
Ripened soft	Raw or thermised	UK	2004	Retail outlets	806	8 (1.0)	ND 798 samples <100 8 samples	1/2a, 4b	(Little *et al.*, 2008)
Ripened soft	Pasteurised	UK	2005	Retail outlets	1622	1 (0.1)	ND 1621 samples <100 1 sample	1/2a, 4b	(Little *et al.*, 2008)
Cheeses	NR	Italy	2001/02	NR	13,858	148 (1.1)	NR	NR	(Busani *et al.*, 2005)
Hard and soft	Raw (cow, sheep and goat)	Portugal	2005/06	Mostly retail outlets	70 (21 hard, 49 soft)	2 (3)[2]	ND (<10) 68 samples 30 1 sample 200 1 sample	NR	(Almeida *et al.*, 2007)
Crescenza (soft)	NR	Italy	2005/06	Retail outlets	437	1 (0.2)	<0.36	1/2c	(Acciari *et al.*, 2011; Prencipe *et al.*, 2010)
NR	Raw (goat) Raw (cow)	Norway	NR	Cheese makers	49 73	ND 1 (1.4)	NR	NR	(Jakobsen *et al.*, 2011)
Soft and semisoft	NR	29 EU Member States and Norway	2010-2011	Retail	3393	16 (0.47)	12: <10 CFU/g 2: 10-100 CFU/g 2: >1000 CFU/g	NR	(EFSA, 2013)

NR, not reported (includes tests that were not carried out); ND, not detected

[1] Using qPCR, 8/20 (40%) samples were positive and 4/8 positive samples could be enumerated without enrichment (counts ranged from $1.0x10^3$ to $1.9x10^4$ CFU/g).

[2] The text in this paper reports 8/70 positive, but the numerical results in the tables do not support this statement. The prevalence for the hard and soft cheeses was not reported separately. The two positive samples were both soft cheeses made from raw sheep milk.

7.2.2 Recalls

Food recall data provide an indication of how often *L. monocytogenes* has been detected in cheeses released onto the market. Recalls are not necessarily linked to human illness. Table 11 provides a summary of food recalls from Australia, Canada, the USA, the EU and the UK for cheeses contaminated with *Listeria* spp. or *L. monocytogenes*, for the period January 2009-January 2014. Recall data were not included in the 2005 Risk Profiles. Recalls where cheese was one of several possible contaminated ingredients, such as feta salad, have been excluded, and recall reports clearly involving the same product have been combined. Hard, semi-hard and soft cheeses were all the subject of recalls. Contamination may have occurred post-processing (e.g. during cutting and packaging – see recalls of shredded cheeses). In the USA many recalls are of Mexican-style soft-cheeses (e.g. queso fresco), while in the EU many recalls are of raw milk cheeses.

Table 12 provides data on concentrations of *L. monocytogenes* in cheese recalls in the EU. Gorgonzola and raw milk cheeses are frequently recalled. The blue mould in Gorgonzola raises the internal pH of the cheese allowing *L. monocytogenes* growth.

Table 11: Recalls of cheeses for contamination with *Listeria* spp. or *L. monocytogenes*: Australia, Canada, the EU and the USA (January 2009-January 2014)

Country/countries where recalled	Date of recall notice (month, year)	Product	Product country of origin
Australia[a]	January 2014	Various hard cheeses	Australia
	January 2014	Tulum (feta-type)	Australia
	February 2013	Blue cheese	Italy
	January 2013	Duetto cheese (mascarpone and gorgonzola)	Australia
	December 2012 – January 2013	Various soft cheeses[1]	Australia
	March 2012	Soft cheese[2]	Australia
	October 2010	Various cheeses	Australia
North America			
Canada[b]	September 2013	Surface-ripened firm raw milk cheese	Canada
	July – August 2013	Gorgonzola	Italy
	March 2013	Gorgonzola	Italy
	October 2012	Semi-hard sheep milk cheese (kashkaval)	Bulgaria
	June 2011	Semi-hard ripened	NR
	May – June 2011	Blue stilton	England
	February 2011	Aged washed rind cheese	Canada
	November – December 2010	Processed cheese slices	Canada
	August 2010	Cheese	Canada
	April 2010	Mozzarella	Canada
	June 2009	Fresh cheese	Canada

Table 11 continued

Country/countries where recalled	Date of recall notice (month, year)	Product	Product country of origin
USA[c]	July 2013	Raw milk cheeses	France
	July 2013	Various cheeses	USA
	June 2013	Gouda	USA
	November 2012	Cheddar, Colby	USA
	October 2012	Various soft cheeses[3]	NR
	September – October 2012	Ricotta salata	Italy
	August 2012	Various cheeses	NR
	June – July 2012	Queso fresco	NR
	March 2012	Queso fresco, string cheese	USA
	January 2012	Firm, unripened cheeses	NR
	January 2012	Various shredded cheeses	NR
	December 2011	Various shredded hard and soft cheeses	NR
	December 2011	Various soft cheeses[4]	NR
	December 2011	Various cheeses	NR
	October 2011	Blue cheese	USA
	September 2011	Queso fresco	NR
	June 2011	Blue cheese	NR
	May – June 2011	Blue Stilton	England
	May 2011	Various soft cheeses	Belgium
	January 2011	String cheese	NR
	November 2010	Cheddar cheeses[5]	USA
	November 2010	Various cheeses	NR
	August – September 2010	Raw milk hard cheeses[6]	USA
	August 2010	String Cheese, queso fresco	NR
	July 2010	String Cheese, queso fresco	NR
	May 2010	Sheep and goats' milk cheeses	NR
	April 2010	Queso fresco	NR
	February – March 2010	Various cheeses	USA
	February 2010	Queso fresco, panela, requeson	NR
	January 2010	Various Cheddar cheeses	USA
	August 2009	Various soft cheeses	NR
	August 2009	Various soft cheeses	USA
	June 2009	Various soft cheeses	NR
	March – April 2009	Mexican-style cheese	NR

Table 11 continued

Country/countries where recalled	Date of recall notice (month, year)	Product	Product country of origin
USA	February – March 2009	Queso fresco	NR
	January 2009	Blue cheese	England
USA and Canada[c]	November – December 2011	Various cheeses[7]	Canada
EU (excluding UK)[d]			
Switzerland	January 2014	Gorgonzola	Italy
France	January 2014	Goat cheese	France
Germany	December 2013	Soft cheese	France
Germany	December 2013	Raw milk sheep cheese	France
Luxembourg	December 2013	Sheep cheese	France
France	November 2013	Raw cows' milk cheese	France
Denmark	November 2013	Cream cheese	Denmark
France	October 2013	Raw milk cheese	France
France	August 2013	Cows' milk cheese	France
France	July 2013	Mascarpone with gorgonzola	Italy
France	June 2013	Raw milk cheese	France
France	June 2013	Gorgonzola	Italy
Italy	May 2013	Diced mozzarella	Italy
Germany	April 2013	Sheep cheese	France
France	March 2013	Frozen soft raw milk sheep cheeses	France
Switzerland	February 2013	Gorgonzola	Italy
France	December 2012	Different cheeses (goat, sheep and cows' milk)	France
France	November 2012	Raw milk cheese	France
Italy	November 2012	Raw milk cheese	Italy
Belgium	October 2012	Cheese	Belgium
France	October 2012	Mozzarella	Spain, Lithuania
Italy	October 2012	Ricotta	Italy
Spain	September 2012	Fresh cheese	Portugal
Italy	July 2012	Cheese	Italy
France	June 2012	Raw milk cheese	France
Denmark	June 2012	Camembert	Denmark
France	June 2012	Raw milk cheese	France
Belgium	June 2012	Goat cheese	Belgium
Germany	May 2012	Manouri sheep cheese	Greece, Cyprus
France	April 2012	Raw milk sheep cheese	France
Ireland	January 2012	Blue	Ireland
Austria	December 2012	Cheese	Austria
Italy	November 2011	Gorgonzola	Italy

Table 11 continued

Country/countries where recalled	Date of recall notice (month, year)	Product	Product country of origin
France	October 2011	Munster	France
France	October 2011	Cheese	France
France	October 2011	Gorgonzola	Italy
France	October 2011	Cows' milk cheese	France
France	October 2011	Gorgonzola	Italy
France	October 2011	Gorgonzola	France, Italy
France	August 2011	Raw milk cheese	France
Belgium	May 2011	Cheeses	Belgium
France	May 2011	Raw milk sheep cheese	France
Germany	April 2011	Gorgonzola	Italy
France	April 2011	Cheese	Belgium
Belgium	March 2011	Cheese	Belgium
Finland	March 2011	Cheese	Spain
France	February 2011	Shredded Emmentaler cheese	Germany, Denmark, Netherlands
Denmark	February 2011	Sheep milk cheese	Italy
France	December 2010	Raw milk raclette	France
Belgium	November 2010	Goats' cheese	Belgium
Ireland	July 2010	Lavistown	Ireland
Poland	June 2010	Gouda	Poland
Austria	January 2010	Syrečky (Quargel Käse)	Austria
France	January 2010	Raw milk Brie	France
France	January 2010	Cheese tray	France
Poland	December 2009	Cheese products	Poland
France	August 2009	Epoisse	France
France	August 2009	Fourme d'Ambert	France
France	July 2009	Gorgonzola	Italy
France	April 2009	Raw milk cheeses	France
Belgium	April 2009	Raw milk soft cheese	Belgium
Italy	April 2009	Gorgonzola	Italy
Germany	February 2009	Gorgonzola	Germany, Italy
France	February 2009	Raw milk Camembert	France
France	January 2009	Raw milk Camembert	France
France	January 2009	Gorgonzola	Italy

Table 11 continued

Country/countries where recalled	Date of recall notice (month, year)	Product	Product country of origin
UK[e]			
UK	February 2013	Unpasteurised cheese, cream cheese, ricotta, various farmhouse cheeses	Scotland
	May 2011	Cheese slices	NR

References:

a. http://www.foodstandards.gov.au/industry/foodrecalls/recalls/Pages/default.aspx (accessed 4 February 2014). Data from 2009 were kindly provided by FSANZ.

b. http://www.inspection.gc.ca/about-the-cfia/newsroom/food-recall-warnings/eng/1299076382077/1299076493846 (accessed 5 February 2014).

c. http://www.fda.gov/Safety/Recalls/ArchiveRecalls/default.htm (accessed 5 February 2014). Recalls from July to December 2011 are not available.

d. Rapid Alert System for Food and Feed (RASFF) portal, https://webgate.ec.europa.eu/rasff-window/portal/ (accessed 5 February 2014). Search function parameters: Notified between 01/01/2009 and 31/01/2014 Type = Food; Classification = alert; Product category = milk and milk products; Hazard category = pathogenic microorganisms. 66 relevant records retrieved.

e http://www.food.gov.uk/enforcement/alerts/ (accessed 5 February 2014). Search keyword = cheese. Recalls only available back to 2010.

NR, not reported.

[1] Multiple recalls due to contaminated soft cheeses produced by Lactalis Jindi Pty Ltd. that caused a listeriosis outbreak (see Section 8.2.2).

[2] Also contaminated with *E. coli*.

[3] A multi-state listeriosis outbreak was caused by imported Frescolina Marte Brand Ricotta Salata cheese (see Section 8.2.2).

[4] Two cases of listeriosis may have had exposure to these products.

[5] Also contaminated with *E. coli* O157:H7.

[6] Also contaminated with *Staphylococcus aureus*.

[7] One reported case of listeriosis was linked to consumption of one of these cheese products (http://www.inspection.gc.ca/about-the-cfia/newsroom/food-recall-warnings/complete-listing/2011-11-24b/eng/1357653786939/1357653786970, accessed 5 February 2014).

Table 12: Concentration of *L. monocytogenes* in samples of cheeses recalled in the EU for contamination with *L. monocytogenes*, January 2009-January 2014

Cheese description	*L. monocytogenes* concentration (CFU/g)
Blue	4,300
Brie (raw milk)	<10
Camembert (raw milk)	460, 5500
Cream cheese	<10, 230
Epoisse	11000
Fourme d'Ambert	<100
Gorgonzola	<10, 40, 150, 300, 800, <1500, 2100, 4100, 4400, 5500, 9700, 9900
Lavistown	290
Manouri sheep cheese	1900
Mascarpone with gorgonzola	<10
Mozzarella	1800
Munster	6500
Raclette (raw milk)	<100
Emmentaler (shredded)	<10
Syrečky (Quargel Käse)	<10
Cheese	<100, 350, 460, 1100, >330000
Cheese products	NR
Cheese tray	3600
Cheese (fresh)	1400, 3100
Cheese (cows' milk)	<10
Cheese (goats' milk)	120
Cheese (sheep milk)	130
Cheese (cows', goats' and sheep milk)	190 - 4500
Cheese (raw milk)	<10, <100, 170, 220, 220, 460, 850, 4000, 5500?, 7200, 200000
Cheese (raw cows' milk)	300
Cheese (raw sheep milk)	110, 210, 230
Cheese (raw sheep milk, soft, frozen)	40

Source: Rapid Alert System for Food and Feed (RASFF) portal, https://webgate.ec.europa.eu/rasff-window/portal/ (accessed 5 February 2014). Entries where *L. monocytogenes* was recorded as "present" were ignored.

7.2.3 Consumption of cheeses

7.2.3.1 Australia

Data from the 1995-1996 Australian National Nutrition Survey indicates that Cheddar (25.6%) was the most frequently consumed cheese with an average of 35 g consumed per serving. Consumption of extra hard, Swiss-type, blue, Feta and Camembert cheeses is significantly lower with only 2.3%, 0.3%, 0.5%, 0.6% and 0.6% of the population surveyed consuming these cheese types, respectively (FSANZ, 2009).

Dairy Australia reports that since 2009/2010 cheese consumption has stabilised at around 13.5 kg per person per annum. The split between Cheddar to non-Cheddar varieties has also been stable, with nearly 55% being Cheddar types and the remaining 45% spread across the wide range of non-Cheddar cheese varieties available in Australia.[29]

[29] http://www.dairyaustralia.com.au/Markets-and-statistics/Production-and-sales/Consumption-Summary.aspx accessed 27 May 2014

8 APPENDIX 2: EVALUATION OF ADVERSE HEALTH EFFECTS

8.1 Dose Response

Dose response information is presented as an estimated number of cells that have caused infection (point estimate) or the probability of infection by exposure to differing numbers of cells. Point estimates can be calculated from outbreaks. Dose response studies for *L. monocytogenes* have not been conducted on humans due to ethical concerns. Animal feeding trials and data from outbreaks provide data to model dose response and calculate probability of infection.

8.1.1 Invasive listeriosis

8.1.1.1 Point estimates from outbreaks

No outbreaks were located where all critical information necessary to estimate dose was known (i.e. the concentration of *L. monocytogenes* in the food consumed and the amount of food consumed). Three listeriosis outbreaks provide indicators:

- USA, meat frankfurters: The outbreak strain (serotype 4b) was only detected at low concentrations (<0.3 CFU/g) in the recalled product, but product testing also recovered a *L. monocytogenes* strain of serotype 1/2a at concentrations up to 3,000 CFU/g (Mead *et al.*, 2006).
- Switzerland, ham: Testing of two ham samples found concentrations of 4,800 and 470 CFU/g *L. monocytogenes*, respectively. A point estimate can be calculated as 4.7×10^4 CFU (470 CFU/g x 100 g) (Hächler *et al.*, 2013).
- Finland, butter, immunocompromised people: Exposure estimated as being between 3-4 $\log_{10}$ CFU, or up to 6 $\log_{10}$ CFU for a highly contaminated sample (Lyytikainen *et al.*, 2000).

Several other outbreaks provide information on the concentration of *L. monocytogenes* in cheeses associated with outbreaks:

- Europe, quargel: Cheese from the refrigerator of a listeriosis case yielded 2.1×10^6 CFU/g *L. monocytogenes* five days after the patient was hospitalised and 18 days after the cheese was purchased (Fretz *et al.*, 2010). An additional 39 recalled cheeses contained between ≤100 and 6.6×10^7 CFU/g (mean 1.2×10^7 CFU/g) (Rossmanith *et al.*, 2010).
- Switzerland, tomme cheese: Cheese from manufacturer contained up to 3.2×10^4 CFU/g *L. monocytogenes* (Bille *et al.*, 2006).
- Germany, acid-curd cheese: Unopened cheese samples from a patient's refrigerator contained 5×10^2-1×10^5 CFU/g *L. monocytogenes* (Koch *et al.*, 2010).
- Italy, gorgonzola: Unopened cheese from a listeriosis patient's refrigerator contained 1,200 CFU/g *L. monocytogenes* (the patient had consumed large quantities of the cheese and was being treated for cancer) (Gianfranceschi *et al.*, 2006).
- Norway (hospital), Camembert: Unopened cheese contained up to 6×10^6 CFU/g *L. monocytogenes*, the serving size for patients was approximately 60 g of cheese, thus the

maximum dose was estimated to be $3.6x10^8$ CFU (Johnsen *et al.*, 2010). The estimated attack rate was 1.2-2.4%.

8.1.1.2 Probability of infection

The 2005 Risk Profiles presented the FAO/WHO single hit models for the probability of listeriosis for the population with increased susceptibility (assumed to represent 15% to 20% of the total population but 90 to 98% of the cases) and the rest of the population (FAO/WHO, 2004b). These models have since been used by the European Food Safety Authority as part of their risk assessment for *L. monocytogenes* in RTE foods (EFSA, 2007). The different population susceptibilities are illustrated by the following example: For a dose of $1x10^{10}$ CFU/serving (e.g. 100 g of a food contaminated at $1x10^8$ CFU/g), the median listeriosis rate in the susceptible population was estimated at 1 in 100 servings, with lower (5%) and upper (95%) uncertainty bounds of 1 in 400 and 1 in 10. For the same dose, in the healthy population, the median listeriosis rate was estimated at 1 in $4x10^3$ servings (lower bound 1 in 30,000, upper bound 1 in 400) (EFSA, 2007).

Modelling based on USA data has generated R-values[30] for a susceptible population based on *L. monocytogenes* virulence as determined by a strain belonging to lineage I or lineage II (see Section 7.1.1) (Chen *et al.*, 2006). The R-values calculated for lineage I and lineage II strains were $1.3x10^{-8}$ and $5.0x10^{-11}$, respectively. For comparison, the probability of illness independent of lineage as calculated by this model was $1.1x10^{-10}$. Such R-values are only of use where *L. monocytogenes* strains isolated from human clinical cases or foods have been assigned to a lineage based on molecular analysis or by assuming the lineage classifications based on serotype, where possible.

There has been considerable discussion about the potential for a relaxation of the zero tolerance approach for *L. monocytogenes* contamination of food adopted by some countries, to a tolerance of up to 100 CFU/g in foods in which it cannot grow. While the only completely safe dose of *L. monocytogenes* is zero, even for healthy people, the model indicates that the probability of invasive disease following exposure to even moderate levels of cells is very low. Most listeriosis cases are due to consumption of RTE foods able to support growth of *L. monocytogenes* and containing levels markedly above 100 CFU/g (Chen *et al.*, 2003; EFSA, 2007).

Experiments using Savak Tulum cheese, a traditional Turkish variety (pH 4.6), found that during ripening over 90 days a decline in numbers of *L. monocytogenes* of 4.1 $\log_{10}$ CFU/g occurred (Dikici and Calicioglu, 2013). This study also examined the survival of the remaining bacteria when exposed to simulated gastric fluid (SGF). Although the numbers of bacteria were reduced, a significant proportion survived. For example, *L. monocytogenes* isolated after 90 days ripening at 3.1 $\log_{10}$ CFU/g, declined to 1.3 $\log_{10}$ CFU/g after 90 minutes exposure to SGF. It was suggested that during processing and ripening, acid-tolerance mechanisms of the pathogen had been activated.

8.1.2 Non-invasive listeriosis (febrile gastroenteritis)

The raw milk Risk Profile (King *et al.*, 2014) summarises the concentration of *L. monocytogenes* in foods consumed in seven outbreaks of non-invasive listeriosis, some of

[30] R is a parameter in the equation describing the dose-response relationship

which are mentioned in the 2005 cheese Risk Profiles. The results indicated that high numbers of *L. monocytogenes* need to be ingested to cause disease at a high attack rate (attack rates ranged 52-100% across the six outbreaks), but estimating the dose ingested by cases for most outbreaks was difficult as portion sizes were unknown and the concentration of *L. monocytogenes* in the food at the time of consumption was often not known.

8.2 *L. monocytogenes* Infection Overseas

8.2.1 Incidence

The 2005 Risk Profiles reported that the incidence of listeriosis in New Zealand was similar to Australia, North America and some European countries for periods during the late 1990s/early 2000s, although the New Zealand rate was near the higher end of the reported rates. Table 13 shows the reported incidence of listeriosis for several countries for the years 2011 or 2012. New Zealand's 2013 listeriosis rate of 0.4 per 100,000 is similar to the rates in most developed countries. Comparisons of listeriosis rates between countries must be made cautiously, as reporting practices may differ.

8.2.1.1 Community level estimates

The number of notified *L. monocytogenes* infections only represents a proportion of total cases, since not all cases will come into contact with public health agencies. Estimates for the annual number of community *L. monocytogenes* infections and annual rates of infection have been published recently (such estimates were not reported in the 2005 Risk Profiles):

- USA: 1,607 (90% CrI: 563-3,193) cases of domestically-acquired *L. monocytogenes* infection, of which 99% were estimated as being foodborne (1,591 cases, 90% CrI: 557-3,161) (Scallan *et al.*, 2011). This was based on surveillance data from 2000 to 2008. Using the 2006 USA population of 299 million, both case numbers correspond to a rate of 0.5 per 100,000.
- Canada: 0.6 cases of domestically-acquired foodborne *L. monocytogenes* infection per 100,000 people per year (Thomas *et al.*, 2013). This estimate was based on surveillance data from 2000 to 2010 plus relevant international literature, and was produced through a modelling approach that accounted for underreporting and underdiagnosis.

These estimates are similar to the reported incidence of listeriosis in those countries because the serious health effects caused by listeriosis means that most cases will be notified, and the underreporting and underdiagnosis multipliers are therefore small.

Table 13: Reported incidence data for notified cases of listeriosis overseas

Country	Year	Incidence (cases/100,000)	No. of notified cases	Ref.[1]
Australia	2010	0.3	71	a
	2011	0.3	70	a
	2012	0.4	93	a
North America				
USA[2]	2013	0.3	145	b
Canada	2011	0.4	NR	c
EU countries				
EU notifications	2012	0.4	1,642	d
Austria	2012	0.4	36	d
Belgium	2012	0.8	83	d
Czech Republic	2012	0.3	32	d
Denmark	2012	0.9	50	d
Finland	2012	1.1	61	d
France	2012	0.5	348	d
Germany	2012	0.5	412	d
Ireland	2012	0.2	11	d
Netherlands	2012	0.4	73	d
Poland	2012	0.1	54	d
Spain	2012	0.9	107	d
Sweden	2012	0.8	72	d
United Kingdom	2012	0.3	183	d
Non-EU countries				
Iceland	2012	1.3	4	d
Norway	2012	0.6	30	d
Switzerland	2012	0.5	39	d

[1] References:
a. (Australian Government, 2013)
b. (Crim *et al.*, 2014)
c. (NESP, 2013)
d. (EFSA, 2014)

[2] Data are for the 10 sentinel states monitored by FoodNet, not the whole of the USA.

8.2.2 Outbreaks

The 2005 Risk Profiles together reported nine outbreaks of listeriosis linked to consumption of cheese (six of these outbreaks were linked to cheeses made from raw or thermised milk). Table 14 summarises 15 outbreaks of invasive and non-invasive listeriosis linked to consumption of cheese reported from 2005 onwards where details are published in the scientific literature. The classification of the cheeses in this table was made using available information (e.g. some Hispanic-style cheeses are rennet-coagulated but this type of detail is rarely available in outbreak reports).

In all but one of the outbreaks the cheeses implicated were those with high moisture content. The treatment of the milk used for making the cheeses was known for eight of the outbreaks, and all but one used pasteurised milk which suggests that environmental contamination by *L. monocytogenes* had occurred.

It is important to note that peer-reviewed outbreak reports in the scientific literature represent a proportion of the reported listeriosis outbreaks linked to cheese. Numerous press releases from government authorities and media reports are also available on the internet and these show that listeriosis outbreaks linked to cheese consumption are occurring each year.[31] The US Centres for Disease Control (CDC) operate a searchable database of foodborne outbreaks and eight outbreaks of foodborne listeriosis attributed to the consumption of cheese (3 unpasteurised, 4 pasteurised) were reported from 2005 to 2011.[32] Together, these outbreaks caused 68 cases of which 32 (47%) were hospitalised and 3 (4%) died. Recent reviews of surveillance data from the USA also provides evidence for cheese as a potential vehicle for *L. monocytogenes*:

- USA, foodborne outbreaks, 1998-2008: Of 24 confirmed outbreaks, cheese was the food vehicle in five (21%) (Cartwright *et al.*, 2013). Four of these outbreaks were caused by Mexican-style cheeses (three made from raw milk) and the fifth by a sheep milk cheese made from pasteurised milk.
- USA, *Listeria* illnesses, deaths and outbreaks, 2009-2011: Of 12 reported outbreaks of invasive listeriosis affecting 224 patients, five outbreaks affecting 34 patients implicated soft cheeses made from pasteurised milk that were likely contaminated during cheese making (CDC, 2013a). Four of these outbreaks implicated Mexican style cheese, and one implicated chive cheese and ackawi cheese (a white brine cheese). A further outbreak affecting 15 people (14 cases of febrile gastroenteritis and one of invasive listeriosis) was associated with aged blue vein cheese made from unpasteurised milk and aged for 60 days.

8.2.3 Review of information linking listeriosis with cheese consumption

A review of information on raw milk cheeses and human disease published in 2008 was updated to cover information from 2000-2010 (Hall and French, 2011; Jaros *et al.*, 2008). In the 2008 report, it was concluded that the available evidence was of high relevance to New Zealand and provided moderate support for a causal relationship between consumption of raw milk and raw milk products and infection of listeriosis. It was noted that the prolonged

[31] See, for example, http://outbreakdatabase.com/ and use the search parameters: Vehicle = milk; Organism = *Listeria monocytogenes* (accessed 19 February 2014).
[32] http://wwwn.cdc.gov/foodborneoutbreaks/Default.aspx accessed 7 October 2013

incubation period of listeriosis made it very difficult to prove temporality when using a case-control study design.

In the 2011 report, 41 reports were located which covered more than 50 outbreaks related to raw milk cheese consumption. Of these, *L. monocytogenes* was the pathogen identified in 8 outbreaks in multiple countries. In three of these outbreaks, Mexican style or queso fresco cheeses were implicated. These outbreaks are included in Table 14, apart from a single case mentioned in a recall notice for queso fresco cheese.[33]

[33] http://www.fda.gov/Safety/Recalls/ArchiveRecalls/2010/ucm207627.htm accessed 27 may 2014

Table 14: Overseas outbreaks of listeriosis where cheese was an implicated vehicle (2005 onwards, reported in the scientific literature)

Cheese type	Milk used	Country	Year	Total cases	Hospitalis-ations	Deaths[1]	Serotype	Evidence linking cheese to cases	Reference
Acid coagulated cheeses									
Quargel (surface ripened)[2]	NR	Multiple European countries	2009/10	34	NR	8 (NP)	1/2a (2 strains)	Outbreak strains isolated from cheeses from factory	(Fretz *et al.*, 2010; Schoder *et al.*, 2012)
Acid-curd (harzer käse; ripened)	Pasteurised	Germany	2006/07	189[3]	81% of cases	14% of cases	4b[2]	Outbreak strain isolated from cheese from a patient's home and from manufacturer	(Koch *et al.*, 2010)
Acid/heat coagulated cheeses									
Mexican-style cheese	Raw	USA	2000	13	13	5 (P)	4b	Outbreak strain isolated from cheese samples from stores and one patient, and from raw milk from a supplier to cheese manufacturers	(MacDonald *et al.*, 2005)
Tomme	NR	Switzerland	2005	12 (2 non-invasive listeriosis)	10	3 (NP) 2 (NP)	1/2a (2 strains)	Outbreak strains isolated from cheeses from manufacturer	(Bille *et al.*, 2006)
Latin-style fresh cheese	Pasteurised	Spain	2012	2	2	0	1/2a	Outbreak strain isolated from cheese from retail outlet	(de Castro *et al.*, 2012)
Asadero	Pasteurised	USA	2008/09	8	8	2 (P)	1/2a	Outbreak strain isolated from cheese from manufacturer	(Jackson *et al.*, 2011)
Ricotta salata	NR	USA	2012	22	20	4 (NP) 1 (P)	NR	Outbreak strain isolated from unopened cheese	(CDC, 2012b)

Table 14 continued

Cheese type	Milk used	Country	Year	Total cases	Hospitalis-ations	Deaths[1]	Serotype	Evidence linking cheese to cases	Reference
Semi-soft Hispanic-style cheeses	NR	USA	2014	3 (NP) 5 (P)	7	1		Outbreak strain isolated from unopened cheese	(ProMED-mail, 2014)
Rennet coagulated – surface ripened									
Semi-soft brine-washed rind cheese	NR	USA	2013	6	6	1 (NP) 1 (P)	NR	Outbreak strain isolated from cheese samples from retail and manufacturer	(CDC, 2013b)
Soft washed rind cheese	Pasteurised	Canada	2008	22 (NP) 16 (P)	21 (NP) 16 (P)	2 (NP) 3 (foetal deaths)	1/2a	Outbreak strain (PFGE type) isolated from cheese and environmental samples from retailers, processing plants, but not lactating cows	(Gaulin *et al*., 2012)
Rennet coagulated – mould ripened									
Camembert	Pasteurised	Norway	2007	16 (NP)[4] 1 (P)[4]	16[4]	3 (NP) 2 (P)[4]	1	Outbreak strain isolated from unopened cheese samples from hospital kitchen	(Johnsen *et al*., 2010)
Brie, Camembert, Blue	NR	Australia	2012/13	34	NR	6 (NP) 1 (P)	4	Epidemiological investigation, laboratory testing, food sampling. Outbreak strain isolated from product from retail and manufacturer.	(OzFoodNet Working Group, 2013)
Brie	NR	Chile	2008	53 (NP) 38 (P)	NR	4 (NP) 1 (P)	NR	*L. monocytogenes* detected in sample from a patient's refrigerator	(ProMED-mail, 2008)

Table 14 continued

Cheese type	Milk used	Country	Year	Total cases	Hospitalis-ations	Deaths[1]	Serotype	Evidence linking cheese to cases	Reference
Soft ripened Frere cheese[5]	Pasteurised	USA	2013	6	6	1 (and one miscarriage)	NR	Outbreak strain (PFGE) found in unopened cheese, deficiencies found at manufacturing plant, risk of post-pasteurisation contamination	(Choi *et al.*, 2014)
Multiple cheese types or cheese category not known									
Multiple cheeses from a single manufacturer (soft and semi-hard)	Pasteurised [6]	Japan	2001	38 (all non-invasive listeriosis)	15	0	1/2b	Outbreak strain isolated from cheese samples from manufacturer	(Makino *et al.*, 2005)

NR, not reported.

[1] P = perinatal, NP = non-perinatal.

[2] The concentration of *L. monocytogenes* measured in 15 recalled sample lots of red smear acid curd cheeses was $1x10^2 – 3x10^7$ CFU/g (counts are mean values for replicates) (Schoder *et al.*, 2012). There was no association between the concentration of *L. monocytogenes* and the age of the cheese (ages ranged 30-71 days) when they were initially sampled. Contamination is likely to have occurred in the smearing process.

[3] 189 cases fitted the case definition, but limited information meant that only 34/47 cases reported eating the implicated cheese and 14/16 of the clinical isolates available for molecular typing were matched to the isolates from the cheeses. Of 37 cases where serotyping information was available, 30 were 4b, 5 were 1/2a and two were 1/2b.

[4] 16/17 cases had predisposing underlying conditions and were already in hospital. The one perinatal case was pregnant with triplets, and 2/3 of the babies died.

[5] Cheese not clearly described.

[6] As reported by Hall and French (2011) who requested this information from Makino (Hall and French, 2011).

8.2.4 Case control studies investigating cheese as a risk factor

From three case control studies reported in the 2005 Risk Profiles, consumption of soft cheese was a significant risk factor for listeriosis in two studies, and the third study identified a particular brand of low moisture, blue mould cheese as a significant risk factor.

Table 15 summarises two case control studies which included cheese consumption as one of the risk factors considered for non-perinatal cases. Camembert, blue cheese and hard cheese other than Cheddar had statistically significant elevated odds ratios.

No case control studies were identified that focussed on perinatal cases.

The ability of case-control studies to determine the risk of cheese is sometimes compromised by the low numbers of cases and controls amongst the study population consuming the product, e.g. a study of perinatal cases in Australia had only 2/12 controls and 0/19 cases who reported consuming Camembert so the odds ratio was not able to be calculated (Dalton *et al.*, 2011).

Table 15: Case control studies containing information on *L. monocytogenes* in cheeses since 2005: Non-perinatal cases

Time period	Country	Risk factor	Number of participants		Reporting of risk factor		Odds ratio (95% confidence interval) by		Reference
			Cases	Controls	Cases (%)	Controls (%)	Univariate analysis	Multivariate analysis	
2001-2004	Australia	Consumption of: Camembert Blue-veined cheese Feta	112	85	 14 (13) 11 (10) 16 (14)	 7 (8) 5 (6) 7 (8)	 2.5 (0.9-7.4) 1.9 (0.5-7.2) 1.9 (0.6-5.7)	 **4.7 (1.1-20.6)** NS NS	(Dalton *et al.*, 2011)
2005-2008	England (people ≥60 years old)	Consumption of: Cheddar Other hard cheese Blue cheese Camembert Brie Other cheese	159	18,115	 112 (71) 54 (35) 26 (17) 8 (5) 10 (7) 57 (36)	 13,513 (75) 3,394 (19) 1,482 (8) 206 (1) 874 (5) 4,659 (26)	 0.9 (0.6-1.2) **2.4 (1.7-3.3)** **2.2 (1.5-3.4)** **4.8 (2.3-9.9)** 1.4 (0.7-2.6) **1.7 (1.2-2.3)**	NR	(Gillespie *et al.*, 2010)

NR = not reported, NS = not significant and values not reported.

8.3 Risk Assessment and Other Activities Overseas

Two risk topics are applicable to *L. monocytogenes* in cheeses; 1) assessments that consider cheeses and 2) assessments that consider *L. monocytogenes* in RTE foods.

8.3.1 Risk assessments considering cheeses

8.3.1.1 Australia

In a Risk Profile of dairy products in Australia that considered cheese, soft cheeses were rated "higher risk" for the consumer relative to other dairy products because they are prone to contamination after final heat treatment and provide a favourable environment for pathogens (FSANZ, 2006).

Subsequently, a microbiological risk assessment for raw milk cheeses has focussed on representative types of ripened and unripened cheeses from different moisture categories, made from cows', goats' or sheep milk (FSANZ, 2009). The assessment concluded that the key determinant for the safety of raw milk cheese is the microbiological quality of the raw milk. It was noted that it was difficult to provide information on the risks associated with broad classes or categories of cheese because data were lacking or highly variable.

The risk assessment found that raw milk Swiss-type cheeses with a low curd cooking temperature, blue, feta and Camembert cheese pose a high risk to susceptible populations due to the survival and/or growth of *L. monocytogenes* during cheese making. *L. monocytogenes* was found to present negligible or very low risk for the general population for extra hard, Swiss, and Cheddar cheeses, although *L. monocytogenes* represented a high risk to susceptible populations in Swiss Appenzeller, Tilsiter, Tête de Moine and Vacherin Fribougeois cheeses. *L. monocytogenes* presents a greater risk in Cheddar produced from raw sheep milk, due to its reported higher prevalence in raw sheep milk, compared to cow and goat milk.

Quantitative models were used to estimate the change in concentration of *L. monocytogenes* during the production of raw milk Cheddar, blue, Camembert, and feta cheeses. The greatest amount of growth was predicted for *L. monocytogenes* in Camembert, e.g. from a starting concentration of 0.001 *L. monocytogenes* cells/mL of milk, the final mean concentration of *L. monocytogenes* in a wedge was predicted to be 2.1×10^3 cells/g (5^{th}-95^{th} percentiles of 1.0×10^3-3.8×10^3).

The assessment also included an evaluation of the impact of cheese making steps on the microbiological safety of raw milk cheeses. The factors that have the greatest impact were the microbiological quality of the raw milk, the acidification step, the temperature and duration of curd cooking, and the temperature and duration of maturation. They concluded, however, that it was the combination of hurdles rather than any individual processing step or physicochemical property that had the greatest impact on pathogen survival.

8.3.1.2 North America

The USFDA and Health Canada have published a draft quantitative risk assessment of the risk of listeriosis from soft-ripened cheese consumption in the USA and Canada (FDA/Health Canada, 2012a, 2012b). Modelling was used to predict the risk of listeriosis from *L.*

monocytogenes in soft-ripened cheeses (Camembert-like cheeses) and to evaluate risk-reduction measures for soft-ripened cheese made from raw milk.

The risk of listeriosis was expressed as the predicted number of servings resulting in one case of listeriosis for the general population and for susceptible subpopulations (elderly, pregnant, immunocompromised). Overall, the predicted number of servings resulting in one case of listeriosis was much lower for cheeses made from raw milk than cheeses made from pasteurised milk, and the predicted number was lower for immunocompromised populations when compared with the general population.

Based on modelling, the most effective intervention for reducing the risk of listeriosis from consumption of raw milk cheeses was testing all cheese lots for *L. monocytogenes* and removing any positive lots from the market. Almost as effective was testing raw milk from the farm bulk milk tank and removing any milk lots that were positive for *L. monocytogenes*.

Important data gaps were the prevalence and concentration of in-plant contamination, and growth of *L. monocytogenes* in naturally contaminated cheeses.

8.3.1.3 United Kingdom

A qualitative risk assessment, for the main categories of UK retailed cheeses, has been published by the UK FSA (Banks, 2006).

The report considers each processing step and ingredients for potential *L. monocytogenes* contamination and factors affecting survival and growth. These are discussed in terms of "representative types" of cheese. A qualitative risk ranking approach was applied to generate scores for factors: frequency of contamination, contamination concentration, effect of process step, recontamination, recontaminant concentration, and growth during storage. The highest scores were for three raw milk varieties.

The report states that, in the categories of cheese that account for the majority of UK consumption by volume, the vulnerability from *L. monocytogenes* appears to be very low, and this is supported by the paucity of data on foodborne listeriosis associated with cheese. Most cheese types offer a dynamic and changing physico-chemical environment to the microorganisms present therein, making prediction of the fate of *L. monocytogenes* complex and uncertain.

8.3.1.4 Model for a risk based approach: L. monocytogenes in soft cheese

A compartment based mathematical model of the process for making soft cheese with respect to *L. monocytogenes* contamination and possible growth has been developed (Tenenhaus-Aziza *et al.*, 2014). The model considers steps after pasteurisation, and opportunities for cross-contamination.

8.3.2 Risk assessments considering *L. monocytogenes* in RTE foods

8.3.2.1 FAO/WHO

In 2004, the Food and Agricultural Organisation of the United Nations (FAO) and the World Health Organisation (WHO) published a risk assessment of *L. monocytogenes* in RTE foods

(FAO/WHO, 2004a, 2004b).[34] One of the objectives was to estimate the risk of serious illness from *L. monocytogenes* in foods that support its growth, and foods that do not, under specific storage and shelf-life conditions.

Some of the key findings from this risk assessment were:

- Nearly all the listeriosis cases predicted by the model were the result of eating high numbers of *L. monocytogenes* (i.e. consumption of foods that do not meet current standards, whether that is zero-tolerance or 100 CFU/g); and
- Control measures that reduce frequencies of contamination have proportional reductions in rate of illness. Control measures that prevent high levels of contamination at point of consumption would be expected to bring about the greatest reduction in rate of illness.

8.3.2.2 European Union

In 2007 EFSA published a scientific opinion on *L. monocytogenes* in RTE foods, which updated and expanded a previous scientific opinion published by the European Commission, and took the form of a risk assessment (EFSA, 2007). One objective of the updated opinion was to provide scientific advice on different levels of *L. monocytogenes* in RTE foods (absence in 25 g, 100 CFU/g and higher levels) and the related risk for human illness. They found that most listeriosis cases were due to consumption of RTE foods able to support *L. monocytogenes* growth and containing concentrations well above 100 CFU/g, and these foods should be the target of risk management measures. The Panel suggested that, for RTE foods in which *L. monocytogenes* can grow, applying a zero tolerance for *L. monocytogenes* (absence in 25 g) throughout the shelf life might result in foods being classified as unsatisfactory, although they are of low risk. Alternatively, tolerating 100 CFU/g throughout shelf life means accepting the probability that foods with more than 100 CFU/g will be consumed, since it is impossible to predict with certainty that this level will not be exceeded. The impact on public health would depend on whether concentrations markedly above 100 CFU/g are reached.

[34] This risk assessment was mentioned, but not discussed, in the 2005 Risk Profiles.

9 APPENDIX 3: CONTROL MEASURES

9.1 Relevant Physico-chemical and Biological control measures in cheese making.

Table 16: Examples of options for controlling *L. monocytogenes* in cheeses during manufacturing

Control method	Examples	Assessment
Milk cooling	Quantitative risk modelling of *L. monocytogenes* in cheese made from raw goats' milk found that cooling the milk quickly was more effective at reducing the final concentration of *L. monocytogenes* in the cheese than reducing the pH of the milk by 0.5 by adding acid prior to fermentation (i.e. before adding the starter culture and rennet) (Delhalle *et al.*, 2012). Combining these two controls was more effective than either one alone.	Cooling of milk or rapid use without cooling is required for any raw milk cheese.
Bacteriocin-producing bacteria (selected as starter cultures or added in addition to starter cultures)	• The concentration of *L. monocytogenes* inoculated into Minas Frescal cheese increased by 5 $\log_{10}$ CFU/g over 12 days at 8-10°C, but did not change in the presence of a bacteriocin-producing strain of *Enterococcus mundtii* (Vera Pingitore *et al.*, 2012). • There was no change in the concentration of *L. monocytogenes* sprayed onto a smear-ripened cheese (Munster) seven days into ripening (simulating environmental contamination) when the cheeses were sprayed with a bacteriocin-producing strain of *Enterococcus faecium* near the start of ripening (Izquierdo *et al.*, 2009). *L. monocytogenes* grew 5 $\log_{10}$ CFU/g in control cheeses. • The addition of a bacteriocin purified from a strain of *Lactobacillus casei* to finished unripened soft cheeses reduced the concentration of *L. monocytogenes* by around 5 $\log_{10}$ CFU/mL when stored at 4°C for 60 days (Mojgani *et al.*, 2010). The reduction was enhanced when a culture of *L. casei* was also added to the finished cheese.	This is a valid and potentially effective approach. The safety of the bacteriocin-producing organism needs to be verified unless it is QPS or GRAS. The addition of a new organism to the process could result in technological changes to the product.
Bacteriophage	• A bacteriophage was demonstrated to be effective in reducing counts of *L. monocytogenes* when applied to the surface of an artificially contaminated surface ripened red-smear soft cheese (type "Munster") (Carlton *et al.*, 2005). A dose-dependent reduction in numbers of *L. monocytogenes* in the surface of the cheese was observed, with the highest dose reducing numbers to below the detection limit, compared to controls of >7 $\log_{10}$ CFU/g..	A valid approach, and there are commercial products available. Success depends on the phage, the concentration delivered and the step in the process of delivery.

Table 16 continued

Control method	Examples	Assessment
Antimicrobials/ preservatives	• Nisin (12.5 mg/kg) added to pasteurised milk before making Minas Fresco cheese did not prevent growth of *L. monocytogenes* in the cheese and only reduced the final concentration by around 1 $\log_{10}$ CFU/g (Vera Pingitore *et al.*, 2012). • Nisin and/or caprylic acid (>0.4g/kg) reduced the numbers of *L. monocytogenes* inoculated into queso fresco at 4 $\log_{10}$ CFU/g by at least 3 $\log_{10}$ CFU/g after 20 days storage (Gadotti *et al.*, 2014) • When lauric arginate (200 ppm), bacteriophage P100 (10^8 PFU/g) and potassium lactate-sodium diacetate (PL-SD, 2.8%-0.2%) were applied singly and in combinations to the surface of Queso Fresco cheeses inoculated with *L. monocytogenes*, only PL-SD in combination with either lauric arginate or P100 reduced the concentration of *L. monocytogenes* and prevented re-growth (Soni *et al.*, 2012). • Sodium lactate (2% w/v) and sodium propionate (2% w/v), in combination with sodium acetate (0.25% w/v) immediately reduced the concentration of *L. monocytogenes* on minas frescal and Coalho cheeses but did not prevent regrowth at 10°C (Silva *et al.*, 2012). • An anionic peptides-enriched extract (20 mg/g), produced by nanofiltration of a tryptic hydrosylate from whey proteins inhibited growth of *L. monocytogenes* in reconstituted Cheddar cheese by 1-1.5 $\log_{10}$ CFU/g (Demers-Mathieu *et al.*, 2013).	Nisin is a bacteriocin approved for use in some foods and so could be more widely applied. Other preservatives will have pros and cons with respect to issues such as organoleptic changes to the cheese, and consumer acceptability.
Plant essential oils	• The essential oil of *Mentha longifolia L.* inhibited growth of *L. monocytogenes* in Iranian white-brined cheese during ripening and storage, but the taste of cheeses with the essential oil at an effective concentration was not acceptable (Ehsani and Mahmoudi, 2013). • The essential oils of pennyroyal, spearmint and tarragon did not prevent growth of *L. monocytogenes* in fresh white cheese and nor did monolaurin (a food-grade antimicrobial), but the combination of tarragon essential oil and monolaurin was listericidal (Hamedi *et al.*, 2014). • The concentration of *L. monocytogenes* inoculated onto feta cheese and packaged under modified atmosphere decreased faster in the presence of essential oils from thyme or oregano, and cheeses containing these essential oils were still acceptable to consumers (Govaris *et al.*, 2011).	Possibly the main drawback is that a sufficiently high concentration of essential oils may impair the flavour of the cheese. Therefore may be of more use in flavoured cheeses.

Table 16 continued

Control method	Examples	Assessment
Ripening conditions	• *L. monocytogenes* grew on an uncooked pressed cheese during ripening, but the concentration was lower when the cheeses were ripened under 93% relative humidity compared with 97% relative humidity (Callon *et al.*, 2011). • Wooden shelves are favoured for cheese ripening but can be contaminated with *L. monocytogenes*. It has been found that the wood is not in itself listericidal and can protect *L. monocytogenes* from being removed or inactivated by soaking and scrubbing. However, *L. monocytogenes* was not detected after the scrubbed shelves were heat treated at 80°C/5 min and 65°C/15 min (Zangerl *et al.*, 2010).	Ripening conditions are usually part of the process of producing a cheese of a particular characteristic and so less amenable to manipulation. Maintenance of hygienic conditions is of critical importance to prevent contamination of manufactured cheeses.
Anti-listeria biofilms	• The presence of *Lactobacillus* spp. biofilms in the cheese making vessel did not prevent growth of *L. monocytogenes* in a soft cheese, and only reduced the final concentration by around 1 $\log_{10}$ CFU/g (Speranza *et al.*, 2009). • In the presence of an active resident biofilm formed on wooden shelves during cheese ripening, *L. monocytogenes* survived or decreased in concentration (Mariani *et al.*, 2011). When the resident biofilm was thermally inactivated, *L. monocytogenes* grew on the shelves.	Currently it would not be practical for small/medium cheesemaker to curate an effective biofilm. *L. monocytogenes* may also colonise the biofilm.
Cheese wrappings	• The concentration of *L. monocytogenes* inoculated onto ricotta cheese which was then covered with an edible coating containing nisin initially reduced under storage at 4°C, but the reduction was not sustained with storage >7 days (Martins *et al.*, 2010). The coating without nisin also inhibited growth, but did not prevent it. • The concentration of *L. monocytogenes* inoculated onto Cheddar cheese which was then wrapped in a polyvinylidene chloride film reduced under storage at 4°C when the film contained 1.5% or 3% sorbic acid (Limjaroen *et al.*, 2005).	There is potential to introduce antimicrobials into cheese wrappings/coatings but these are, by definition, only active at the cheese surface.
Heat treatment	*L. monocytogenes* sprayed onto the rind of ricotta salata cheeses at a concentration of $1x10^6$ CFU/g was not detected after cheeses were vacuum-packed and heated in a water bath at 85°C for 90 minutes, even when tested after 12 months storage at 4°C (Spanu *et al.*, 2013).	Of obvious use, but in a limited range of cheeses.
Irradiation	The concentration of *L. monocytogenes* decreased in feta irradiated with 1.0, 2.5 or 4.7 kGy (Konteles *et al.*, 2009).	Problems with consumer acceptance

Table 16 continued

Control method	Examples	Assessment
Hydrostatic pressure	• Pressure treatment (500 Mpa, 10 min, 20°C) of raw milk containing L. monocytogenes at a concentration of up to 4 log10 CFU/mL eliminated the pathogen and it was not detected in ripened Camembert cheeses subsequently made using the milk (Linton *et al.*, 2008). • Two strains of *L. monocytogenes* added to model cheese (dry matter 55%, 1.5% salt in moisture) at approximately 7.5 $\log_{10}$ CFU/g were reduced by high hydrostatic pressure treatment (López-Pedemonte *et al.*, 2007). • Reductions of approximately 1, 2 and 5 $\log_{10}$ CFU/g were observed after 10 minutes at 5°C and 300, 400 and 500 milliPascals; at 20°C reductions were slightly greater. Reductions in *L. monocytogenes* numbers have also been shown in experiments using queso fresco, although a second preservation technique would be required during storage (Tomasula *et al.*, 2014).	Of potential use for larger manufacturers as long as the properties of the cheese are not altered. Not practical for small/medium sized cheese makers.
Plasma	The concentration of *L. monocytogenes* inoculated onto sliced cheese decreased when non-thermal atmospheric pressure plasma was applied, with the reduction depending on both input power and plasma exposure time (Song *et al.*, 2009).	Of limited use as this is a surface disinfection technique (example is with sliced cheese) and unlikely to be used by small/medium cheesemakers.

9.2 International Control Measures

9.2.1 Codex Alimentarius Commission

Codex has produced six standards specifically for cheese types included in this Risk Profile update (CAC, 2010a, 2010b, 2010c, 2010d, 2013a, 2013b). However, these standards cover aspects such as composition and labelling and do not directly address hygiene controls. Readers are directed to general hygiene standards that do not specifically consider *L. monocytogenes*, but will assist in its control in the manufacturing environment (CAC, 2003, 2009b).

In 2009, Codex published *Guidelines on the Application of General Principles of Food Hygiene to the Control of Listeria monocytogenes in Foods* (CAC/GL 61-2007) (CAC, 2009a). The document provides general advice on controls for *L. monocytogenes* in ready-to-eat foods, plus suggests the following microbiological criteria that are applicable to cheeses:

- A ready-to-eat food in which growth of *L. monocytogenes* can occur (i.e. the concentration of *L. monocytogenes* increases by more than 0.5 $\log_{10}$ CFU/g for at least the expected shelf life under reasonably foreseeable conditions of distribution, storage and use): Of five samples tested, *L. monocytogenes* should not be detected (<0.04 CFU/g) in a 25 g portion of each sample (i.e. n=5, c=0, m=absence in 25 g).
- A ready-to-eat food in which growth of *L. monocytogenes* will not occur: Of five samples tested, *L. monocytogenes* should not be detected at >100 CFU/g in a 25 g portion of each sample (i.e. n=5, c=0, m=100 CFU/g).

9.2.2 European Union

The 2005 Risk Profiles discussed the lack of standardisation across European countries in terms of microbiological limits for *L. monocytogenes* in cheese. A suite of food hygiene regulations came into effect in January 2006, known as the EU hygiene package (European Commission, 2005). The hygiene package includes three hygiene regulations that contain general principles relevant to the manufacture of cheeses (EC 852/2004, EC 853/2004 and EC 854/2004). Regulation (EC) No 852/2004 lays down general rules for food business operators on the hygiene of foodstuffs based on HACCP principles (European Commission, 2009). Regulation (EC) 853/2004 enables EU member states to establish or maintain national rules regarding the use of raw milk for cheese making (European Commission, 2012). Regulation (EC) 854/2004 describes official controls on primary production and animal health in relation to animal products (European Commission, 2011).

The European Commission Regulation 2073/2005 sets out microbiological criteria for foodstuffs that has applied to EU member states since January 2006 (European Commission, 2013). There are three microbiological specifications that apply to cheese for general consumption:

- Ready-to-eat foods in which *L. monocytogenes* growth can occur, where the manufacturer can demonstrate that *L. monocytogenes* will not exceed 100 CFU/g throughout the shelf life: Of five samples tested, none shall exceed 100 CFU/g *L. monocytogenes* during their shelf life (i.e. n=5, c=0, m=100 CFU/g).

- Ready-to-eat foods in which *L. monocytogenes* growth can occur, where the manufacturer cannot demonstrate that *L. monocytogenes* will not exceed 100 CFU/g throughout the shelf life: Of five samples tested before the food leaves the control of the manufacturer, *L. monocytogenes* should not be detected in a 25 g portion of each sample (i.e. n=5, c=0, m=absence in 25 g).
- Ready-to-eat foods unable to support the growth of *L. monocytogenes*: Of five samples tested, none shall exceed 100 CFU/g *L. monocytogenes* during their shelf life (i.e. n=5, c=0, m=100 CFU/g).

More stringent criteria are set for *L. monocytogenes* in ready-to-eat foods intended for infants or for special medical purposes (n=10, c=0, m=absence in 25 g).

At the retail level, the EU provides microbiological criteria classifications in relation to several pathogens for cheeses based on Recommendations 2004/24/EC and 2005/175/EC (Little *et al.*, 2008). The criteria with respect to *L. monocytogenes* are: Satisfactory (not detected), borderline (detected, $<10^2$), unsatisfactory ($>10^2$).

Cheeses imported into New Zealand (including raw milk cheeses) must meet the standards for *L. monocytogenes* set by Regulation 2073/2005.[35]

9.3 Control Measures in Specific Countries

9.3.1 Australia

On 10 July 2014, FSANZ published a call for submissions on Proposal P1022 "Primary Production and Processing Requirements for Raw Milk Products".[36] This proposal was prepared to assess "additional requirements for milk production, transport and processing for the safe production of raw milk products where it can be demonstrated:

- That the intrinsic physico-chemical characteristics of the raw milk product do not support the growth of pathogens, and
- There is no net increase in pathogen levels during processing."

Additional provisions for these raw milk products will be included in Standard 4.2.4, and these include:

- "The requirement for a food safety program
- Specific control measures for primary production, transport and processing businesses that must be included in the food safety program
- Specified processing measures/outcomes at manufacture."

[35] http://www.biosecurity.govt.nz/imports/animals/standards/daiediic.eec.htm (accessed 2 April 2014).
[36] http://www.foodstandards.govt.nz/code/proposals/pages/proposalp1022primary5627.aspx (accessed 18th August 2014)

There are also changes to Standard 1.6.1 which apply to New Zealand. Existing limits for butter, raw milk cheese and raw milk unripened cheese will be amalgamated into "raw milk products". The new applicable microbiological criteria are:

Raw milk products	*Salmonella*	Not detected in 25 g (n=5)
	Staphylococcal enterotoxins	Not detected in 25 g (n=5)

Limits for *L. monocytogenes* will also apply to raw milk products. In the context of raw milk products which do not allow the growth of pathogens the criterion would be <100 CFU/g.