New Zealand Food Safety
Haumaru Kai Aotearoa

Food Safety During Pregnancy

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Scientific Interpretative Summary

This Scientific Interpretative Summary is prepared by New Zealand Food Safety (NZFS) risk assessors to provide context to the following report for MPI risk managers and external readers.

FW20017: Food Safety During Pregnancy

Most foods and drinks are safe to consume during pregnancy. But there are some products pregnant women should be careful with or avoid. Public Health and Food Safety authorities in most OECD countries provide risk communication material related to food safety during pregnancy.

Such a resource is available on the New Zealand Food Safety (NZFS) website. However, this advice was published in 2007 and was based on information available then. Since that time a range of new foods has been introduced and become popular in the diet of New Zealanders, while there is improved understanding over which foods might be a risk.

NZFS wants to ensure the food safety advice for pregnancy remains current with the latest science and dietary practices for New Zealand. It is important that food safety advice for pregnant women captures all of the foods that could be a risk, however it needs to balance this with ensuring pregnant women can maintain a varied diet and have access to the widest source of nutrition without being overly restrictive.

To achieve this a research project was commissioned to the Institute of Environmental Science and Research Limited (ESR) to provide a scientifically robust background for updating food safety advice for pregnant women.

Food safety considerations are focused on *Listeria monocytogenes, Toxoplasma gondii*, mercury and caffeine, as these four hazards have known specific impacts on the foetus. Other microbiological and chemical hazards were not targeted as the risks are not pregnancy specific and food safety advice for the general public is also applicable for pregnant women. This report is restricted to food safety issues and does not cover advice on healthy nutrition during pregnancy.

Maternal exposure to the microbiological hazards *Listeria monocytogenes* and *Toxoplasma gondii* is strongly linked to adverse effects on the foetus. For two other microbiological hazards, *Salmonella* spp. and *Campylobacter* spp., associated with adverse outcomes specific to the pregnancy period, the supporting epidemiological evidence is weaker. The report has examined up-to-date information on these two other hazards and provided safety advice where relevant.

The research examined data on complex changes in the maternal immune system that include both down regulation and up regulation of aspects of the immune system. Evidently pregnant women may be more susceptible to some infections than non-pregnant women, but no more susceptible to most types of infections. However, the complications of common infections in pregnant women can be more severe.

The main outcome of the research is an evaluation of evidence for food safety advice during pregnancy. The report included an evaluation of all food groups listed in the current New Zealand Food Safety's guide to food safety in pregnancy. Consideration was also given to a small number of foods that were not previously evaluated in relation to pregnancy. Additional advice was proposed for these foods. NZFS agrees with the suggestion that advice should

be included in the guide for sprouts, recommending that these foods are not eaten unless cooked; for dried herbs recommending thorough cooking and a recommendation to not drink unpasteurised fruit juice and cider (non-alcoholic).

The report confirmed that in most cases, New Zealand Food Safety's advice on foods to eat or not eat during pregnancy are consistent with the available scientific evidence. In a small number of instances, suggestions were made to better align the advice with the current available evidence. NZFS agrees with these suggestions and intends to expand the advice accordingly.

Based on the evidence provided NZFS agrees that the current advice related to low acid soft pasteurised cheeses (e.g. Brie, Camembert, blue, ricotta, mozzarella, feta) should be strengthened to recommend that pregnant women do not eat these cheeses unless cooked.

The report supports NZFS's intention to make its advice on a range of commercial pasteurised dairy products with relatively short shelf-life less restrictive. Currently the advice is to dispose products like pasteurised milk or yoghurt after two days of opening. The reviewed scientific evidence identified that, if that the products are refrigerated in original packaging and care is taken not to contaminate lids when using, it is safe to follow manufacturer's advice on the package. Suggested modifications will allow pregnant women better planning of their daily diets and will also reduce unnecessary food wastage.

The report has suggested the current advice related to soft serve ice cream be reconsidered. However, NZFS's opinion is that, given the likelihood of *Listeria* sloughing into the product through its processing, current advice to avoid this product during pregnancy is adequate.

For some foods the scientific evidence is not currently strong enough to support specific food safety advice on these foods, although the available evidence suggests they may represent potentially emerging risk foods for pregnant women. A brief summary of such foods is provided at the end of the report. NZFS will follow up on any new scientific research related to these products.

NZFS Addendum to the report "Food Safety during Pregnancy"

In the last two years the effect of moderate daily caffeine intake during pregnancy has been the subject of many publications. A longitudinal cohort study, carried by the US National Institute of Child Health and Human Development Fetal Growth Studies (Gleason et al, 2021), reported that caffeine consumption during pregnancy, even in amounts less than 200 mg per day, was associated with smaller neonatal anthropometric measurements.

Similarly, a meta-analysis of cohort studies by Jin and Qiao (2021) concluded that maternal caffeine intake during pregnancy is associated with higher risk of low neonatal body weight and childhood overweight and obesity in their offspring. However, it was noted that current studies do not provide sufficient support for the recommendation of complete avoidance of caffeine intake during pregnancy.

Another systematic review and meta-analysis (Yue *et al*, 2021) of observational studies examined the association between maternal consumption of caffeine or caffeinated products and birth defects. All three of the caffeine consumption categories (low, moderate, and high) were found to be associated with a higher risk of cardiovascular defects and alimentary tract defects, although the association was weak with the odds ratios of 1.17 and 1.35 respectively.

A review (James, 2021) of 37 observational studies concluded that maternal caffeine consumption is reliably associated with negative pregnancy outcomes and assumption about safe levels of maternal caffeine consumption are not supported by current scientific evidence. This conclusion contradicts the advice of major public health and food safety authorities[1], although the latter were issued based on assessing the same studies.

Although some previous studies indicated a possibility of an association between caffeine consumption and negative child neurodevelopment outcomes, the most recent study (Berglundh *et al*, 2021) of a cohort of 64,189 full term pregnancies concluded that low to moderate caffeine consumption during pregnancy was not associated with any persistent adverse effects concerning the child's neurodevelopment.

A study (Lamy *et al*, 2021) of a cohort of French pregnant women observed a significant decrease of neonates' birth length when mothers were consumed at least 100 mg/day of

[1] See relevant advices issued by the ACOG (American College of Obstetricians and Gynecologists. Committee opinion: moderate caffeine consumption during pregnancy, no. 462. 2010, 2016. Available: https://www.acog.org/-/media/Committee Opinions/Committee on Obstetric Practice/co462.pdf8); EFSA (European Food Safety Authority. Panel on dietetic products, nutrition and allergies. Scientific opinion on the safety of caffeine. EFSA J 2015;13:4102) and the NHS (National Health Service. Should I limit caffeine during pregnancy? 2018. Available: https://www.nhs.uk/common-health-questions/pregnancy/should-i-limit-caffeine-during-pregnancy/)

caffeine during the second and third trimesters but this difference was no longer significant after adjustment for potential confounding factors, such as tobacco use.

Although the recently published research still unable to provide conclusive evidence on adverse effects to pregnant women or their offspring due to caffeine intake, NZFS is in opinion that it might be prudent to **limit caffeine to below 200 mg per day during pregnancy**.

This advice is in line with the latest the Ministry of Health's guidelines (MoH, 2020).

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Food Safety during Pregnancy

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CONTENTS

LIST OF TABLES

LIST OF FIGURES

SUMMARY

Food safety authorities in most OECD countries provide risk communication material in relation to food safety during pregnancy**.** Evidence-based advice for pregnant New Zealand women is available on the New Zealand Food Safety website. This advice was first published in 2007 and was based on information available at that time. Since then, in addition to the emergence of new knowledge, a range of new foods have been introduced and have become popular in the diet of New Zealanders. This project evaluates the evidence base for the current New Zealand Food Safety advice, providing updated scientific information and considering the most popular new foods.

Different authorities worldwide offer advice on food safety during pregnancy to address risks associated with various chemical and microbiological hazards. After reviewing the full range of hazards and the basis for concerns, the current exercise focussed on the microbiological hazards *Listeria monocytogenes* and *Toxoplasma gondii,* and the chemical hazards methylmercury and caffeine. Some consideration was also given to the microbial pathogens *Salmonella* spp. and *Campylobacter* spp.

Epidemiological evidence supports associations between maternal exposure to *L. monocytogenes, T. gondii* and methylmercury and adverse effects on the foetus. For *Salmonella* spp., *Campylobacter* spp. and caffeine the evidence is more equivocal. For other microbiological and chemical hazards, food safety concerns during the period of pregnancy are no greater than at any other life stage and pregnant women should consider general food safety advice.

In most cases, New Zealand Food Safety's advice on foods to eat or not eat during pregnancy are consistent with available scientific evidence. In a small number of instances, it was concluded that there was insufficient evidence to support the advice. In a further small number of instances, suggestions were made to better align advice with current available evidence.

1. INTRODUCTION

Food safety authorities in most OECD countries provide risk communication material in relation to food safety during pregnancy**.**

Advice for pregnant New Zealand women is available on the New Zealand Food Safety website (New Zealand Food Safety, 2020). In December 2007, the New Zealand Food Safety Authority (NZFSA) published a resource in conjunction with Dr Cathy Pikholz and Dr Greg Simmons from Auckland Regional Public Health Service. This guidance document has been updated periodically.

Since 2007, in addition to the emergence of new knowledge, a range of new foods have been introduced and have become popular in the New Zealand diet. This project evaluates the evidence base for the current New Zealand Food Safety advice, providing updated scientific information and considering the most popular new foods.

Various physiological changes occur in women during pregnancy, including altered immune mechanisms. These changes are initially discussed in order to place microbial food safety into context. Following this, selected foodborne hazards of interest are introduced along with key information. The final section uses available scientific evidence to evaluate the current advice.

1.1 HAZARDS ON WHICH ADVICE MAY BE PROVIDED

Different authorities worldwide offer advice on food safety during pregnancy to address risks associated with chemical and microbiological hazards. There are some similarities in the microbiological and chemical hazards addressed in food safety advice for pregnant women issued by agencies in other countries (Table 1). Advice about the hazards *Listeria monocytogenes* and mercury are most often included, with *Toxoplasma gondii*, *Salmonella* spp., caffeine and alcohol also commonly included. None of the documents from overseas agencies specifically addressed *Campylobacter* spp. or cadmium.

Table 1. Hazards for which advice for food safety during pregnancy is provided by various authorities

Agency	*L. monocytogenes*	*T. gondii*	*Salmonella* spp.	*Campylobacter* spp.	Mercury	Iodine	Caffeine	Vitamin A	Lead	Cadmium	Dioxins and POPs	Alcohol	Reference
New Zealand and Australia													
NZ Ministry of Health	X	X	X	X	X					X		X	MOH (2017)
FSANZ	X		X		X		X					X	FSANZ (2016)
New South Wales Food Authority	X	X	X		X		X					X	NSW Food Authority (2020)
Tasmanian Department of Health and Human Service	X				X	X							TDHHS (2014)
ACT Health	X				X		X					X	ACTH (2010)
Queensland Health	X	X	X		X								QHSS (2020)

Agency	*L. monocytogenes*	*T. gondii*	*Salmonella* spp.	*Campylobacter* spp.	Mercury	Iodine	Caffeine	Vitamin A	Lead	Cadmium	Dioxins and POPs	Alcohol	Reference
Royal Women's Hospital, Victoria	X				X		X	X				X	RWH (2012)
North America													
Health Canada	X	X											Health Canada (2019)
British Columbia Healthlink	X				X			X					BCHL (2008)
US Food and Drug Administration	X	X	X		X								USFDA (2019)
Other													
UK National Health Service	X	X	X		X		X	X	X		X	X	UKNHS (2020)
Food Safety Authority of Ireland	X				X		X	X				X	(FSAI, 2017)
State of Israel Ministry of Health	X	X	X		X							X	(SIMOH, 2020)
World Health Organization	X	X			X				X		X		(WHO, 2008)

POPs: persistent organic pollutants, FSANZ: Food Standards Australia New Zealand, ACT: Australian Capital Territory

1.2 HAZARDS INCLUDED IN THE CURRENT ASSESSMENT

1.2.1 Microbiological hazards

Pathogens for which there is a well-established relationship between infection and adverse health effects of particular relevance to pregnant women or the foetus are:

***Listeria monocytogenes*.** Pregnant women are an identified at-risk group because of the impact this pathogen can have on the survival or ongoing health of the baby (Lund and O'Brien, 2011). The condition is perinatal listeriosis, i.e. invasive listeriosis affecting pregnant women and their babies. Listeriosis is a notifiable disease in New Zealand. A small number of perinatal listeriosis cases are reported each year (ESR, 2019). See Section 3.1.

***Toxoplasma gondii*.** Pregnant women are an identified at-risk group because of the impact this pathogen can have on the survival or ongoing health of the baby (Lund and O'Brien, 2011). The condition is congenital toxoplasmosis. Toxoplasmosis is not a notifiable disease in New Zealand. Data available from New Zealand hospital discharge records show a small number of reported cases of congenital toxoplasmosis per year (King, 2017). See Section 3.2.

The following pathogens have also been specifically considered in this review, given their importance as causes of foodborne disease in New Zealand:

Salmonella enterica* spp. *enterica

- Typhoidal serotypes (*S*. Typhi, *S*. Paratyphi): Person-to-person spread is an important transmission route in New Zealand but food can be a vehicle of infection. New Zealand

cases might have been exposed while overseas. Transplacental infections have been reported (Crump et al., 2015; Gluck et al., 1994; Guirguis et al., 2017).

- Non-typhoidal serotypes (e.g. *S*. Enteritidis, *S*. Typhimurium): Raw or undercooked eggs are a known vehicle of infection overseas, so the risk from non-typhoidal *Salmonella* spp. are often considered in pregnancy advice issued by other countries. Transplacental infections, with infant mortality, have been reported (Ault et al., 1993; Krishnan et al., 2013; Rai et al., 2014; Zettell et al., 1995).

Salmonella spp. are considered further in Section 3.3.

***Campylobacter* spp. (*C. jejuni* and *C. coli*).** There are some reported cases of foetal death associated with campylobacteriosis, but complications during pregnancy appear uncommon (Farrell and Harris, 1992; McDonald and Gruslin, 2001; Simor and Ferro, 1990). However, a 2015 review suggests that adverse foetal outcomes during the second and third trimester might be occurring more frequently than thought (Kuperman-Shani *et al.*, 2015). Further rationale for consideration of this pathogen is New Zealand's high rate of campylobacteriosis and public awareness of campylobacteriosis (e.g. through reports or involvement in the 2016 Havelock North waterborne outbreak investigation and response). *Campylobacter* spp. are considered further in Section 3.4.

For other foodborne pathogens, pregnant women in New Zealand are not currently considered to be a particularly high risk group (see Appendix A). General food safety advice applies for managing foodborne risks from these hazards.

1.2.2 Chemical hazards

There is evidence for a relationship between exposure and adverse health effects of particular relevance to pregnant women or the foetus for the following chemical hazards:

Mercury. The link between maternal methylmercury exposure (through fish consumption) and neurodevelopment deficits in offspring is well-established through epidemiological studies in the Seychelles and Faroe Islands (Davidson *et al.*, 1998; Debes *et al.*, 2006). However, fish consumption also provides an important source of essential polyunsaturated long chain fatty acids. Dietary advice around fish consumption during pregnancy is increasingly focused on balancing these two factors (Cardoso *et al.*, 2018; Chen *et al.*, 2014).

Caffeine. High maternal caffeine intake (>300 mg/day) has been associated with a range of adverse outcomes, including increased risk of spontaneous abortion, effects on birth size and subsequent growth and effects on neurodevelopment (Temple *et al.*, 2017).

For a number of other chemical hazards, concerns related to exposure during pregnancy may relate to very particular circumstances.

Iodine. There is some evidence that maternal iodine excess, specifically due to consumption of iodine-rich brown seaweeds, may induce hypothyroidism in neonates (Emder and Jack, 2011; Nishiyama *et al.*, 2004). However, given the low iodine status of the New Zealand environment (Hercus *et al.*, 1925), advice with respect to brown seaweed consumption should not compromise messages concerning the importance of adequate iodine nutrition during pregnancy. New Zealand Food Safety's current advice to pregnant woman includes guidance to limit consumption of brown seaweed (New Zealand Food Safety, 2020).

Vitamin A. There is inconsistent evidence that high maternal vitamin A intake during pregnancy, usually from supplement use, is associated with teratogenesis, specifically

malformations (Pike and Zlotkin, 2019). The New Zealand Ministry of Health have provided general guidelines on use of supplements during pregnancy, which does not recommend the use of vitamin A supplements (MOH, 2008).

Lead. The UKNHS advisory on lead relates to the potential for lead shot in game meat and it is unclear whether the concern was specifically pregnancy-related (UKNHS, 2020). While a number of adverse health effects in humans are associated with lead exposure, there is little evidence of increased susceptibility during pregnancy or increased risk to the foetus due to maternal exposure. However, lead exposure in children has been associated with neurodevelopmental deficits and maternal lead status can affect neonatal status through transplacental transfer and through maternal milk (JECFA, 2011; WHO, 2008). Dietary exposure to lead is very low in New Zealand (Pearson *et al.*, 2018). While pregnant women do not appear to be at any excess risk from lead exposure, as with the rest of the population they should be aware of risk factors for elevated lead exposure. A recently-recognised risk factor is consumption of eggs from backyard chickens, particularly where the property is likely to have had lead-based paint applied in the past (Cowie and Gartrell, 2019).

Dioxins and POPs. While there is some evidence of associations between paternal dioxin exposure and sex ratio of progeny, and maternal exposure and thyroid indices in neonates, findings are usually associated with high levels of parental exposure (EFSA, 2019). New Zealand is generally considered to have low levels of dioxin contamination ('t Mannetje *et al.*, 2013; Buckland *et al.*, 1998; Coakley *et al.*, 2018) and dietary adjustments are unlikely to have a significant impact on maternal dioxin exposure.

2. IMMUNE SYSTEM CHANGES DURING PREGNANCY AND INFECTION RISK

2.1 MATERNAL IMMUNE FUNCTION AND TOLERANCE OF THE FOETUS

A number of hormonal and immunological changes take place in a woman's body during pregnancy. While it was previously thought that these changes resulted in a general suppression of the maternal immune system (Smith, 1999), more recent evidence suggests that changes in the maternal immune system are complex and include both down regulation and up regulation of aspects of the immune system (Kourtis *et al.*, 2014). This leads to a situation where pregnant women may be more susceptible to some infections than non-pregnant women, but no more susceptible to other types of infections.

In genetic terms, a foetus is a semi-allogeneic graft; up to one half of the foetus is "foreign" to the mother through the possession/expression of paternal antigens. A number of theories have been proposed to explain why the foetus is not rejected by the mother during pregnancy (Lederman, 1984). It was suggested that suppression of the maternal immune system was probably the most likely mechanism whereby the foetus would be tolerated.

However, it has been demonstrated that foetal-specific CD8+ T lymphocytes were present in half of all pregnancies and often became detectable from the first trimester (Lissauer *et al.*, 2012). The foetal-specific immune response increased during pregnancy and persisted in the postnatal period. It is now thought that foetal tolerance is maintained due to 'fine tuning' of the immune response, rather than its suppression (Solano, 2019).

The interface between the foetus and the uterus occurs where the trophoblast cells, that make up the placenta, meet the decidua, a lining rich in immune cells that coats the uterus during pregnancy (Solano, 2019). Complex production of cell types and expression of regulatory factors appear able to induce foetal tolerance while maintaining a strong immune response to combat any infections to the placenta.

Rowe *et al.* (2013) highlighted the importance of a subset of CD4 T cells called regulatory T cells (Tregs) in maintaining foetal tolerance during pregnancy. The Tregs have potent immune-suppressing properties and undergo a progressive increase in number during normal healthy pregnancies. Blunting of this expansion may result in spontaneous abortion. A side effect of this regulatory suppression of some immune elements is often seen in amelioration or remission of autoimmune disorders, such as rheumatoid arthritis, during pregnancy.

2.2 INCREASED SEVERITY AND SUSCEPTIBILITY TO INFECTION DURING PREGNANCY

There is evidence that, compared with non-pregnant women, pregnant women are more severely affected by infections with some organisms, including influenza virus, hepatitis E virus (HEV), herpes simplex virus (HSV) and malaria parasites (Kourtis *et al.*, 2014). The evidence is more limited for organisms that cause coccidioidomycosis, measles, smallpox and varicella. However, it is likely that the threshold for diagnostic evaluation, as well as hospitalisation and treatment, may be lower for pregnant women than for other patients, and this factor may bias some of the reports of increased disease severity.

While it is not clear why these infections result in higher frequency of severe outcomes in pregnant women, for some infections mechanisms other than immune suppression appear to play a part (Kourtis *et al.*, 2014). For example, cardiopulmonary adaptive changes occurring during pregnancy, such as increased heart rate and stroke volume and reduced pulmonary residual capacity, may increase the risk of hypoxemia and contribute to the increased severity of influenza infections.

Evidence for increased susceptibility to infection during pregnancy is less strong than the evidence for increased severity of outcomes, with credible evidence mainly related to the malaria parasite (*Plasmodium falciparum* or *P. vivax*) and *L. monocytogenes* (Kourtis *et al.*, 2014).

2.3 MECHANISMS FOR BACTERIAL INFECTION AND FOETAL LOSS DURING PREGNANCY

Immunity against most pathogens is maintained through pregnancy. It has been suggested that the ability of some pathogens to infect the foetus may be due to an immunological 'loophole', rather than a general compromise of the immune system. For example, *in vitro* studies have shown that placental trophoblast cells are quite resistant to *L. monocytogenes* infection (Rowe *et al.*, 2013). It has been suggested that a transient suppression of Treg expansion, to allow an immune response to the maternal infection, may compromise the immune tolerance to the presence of the foetus, resulting in adverse outcomes such as abortion and stillbirth. It has also been suggested that this immune response to a bacterial pathogen may result in localised inflammation at the maternal-placental interface, allowing leakage of pathogens past the normally resistant trophoblasts.

Reduction in maternal Tregs have also been noted in association with infection by *S.* Typhimurium) and *T. gondii* (Johanns *et al.*, 2010; Oldenhove *et al.*, 2009).

2.4 SUMMARY

The available evidence suggests that maternal immunity is not suppressed during pregnancy and in most cases pregnant women need only exercise normal care with respect to food safety. However, a limited group of pathogens appear to be able to exploit a loophole in the maternal immune response, potentially resulting in a breakdown in foetal tolerance and infection of the foetus. Potentially foodborne pathogens implicated in this context include *L. monocytogenes, T. gondii* and possibly some strains of *Salmonella enterica*.

3. MICROBIAL HAZARDS AND PREGNANCY

As introduced in Section 1, pregnant women are an identified at-risk group for infections by *L. monocytogenes* or *T. gondii* because of the impact these pathogens can have on the survival or ongoing health of the baby (Lund and O'Brien, 2011). For other foodborne pathogens, pregnant women in New Zealand are not considered to be more susceptible to infection compared to the general population (Appendix A), so general food safety advice applies. Nevertheless, this section also specifically discusses *Campylobacter* spp. and *Salmonella* spp. as microbial hazards in the context of pregnancy. Campylobacteriosis has the highest rate of infection of all notifiable diseases in New Zealand and can be transmitted by food (as well as other transmission routes). Advice to pregnant women to reduce the risk of salmonellosis appears in a number of publications by regulatory authorities in other countries, usually in connection with the risk posed by the *Salmonella* types that can be found inside intact eggs. These types have not been detected in New Zealand (Kingsbury, 2019).

3.1 *LISTERIA MONOCYTOGENES*

Generic information on the survival, growth and inactivation, transmission routes and surveillance data for *L. monocytogenes* is included in Appendix B.

Seventeen species are currently included in the genus *Listeria*, with nine new species added since 2009 (Orsi and Wiedmann, 2016). Six species, including human pathogens *L. monocytogenes* and *L. ivanovii*, form "*Listeria sensu stricto*", a distinct genomic group that share specific phenotypes including motility via flagella and low temperature growth. *L. monocytogenes* is the most important species with respect to human health. *L. monocytogenes* appears to have a particular affinity for placental tissue.

L. monocytogenes causes two forms of disease: a serious invasive disease and a non-invasive gastroenteritis. The invasive form of listeriosis is associated with conditions that compromise the immune system and results in septicaemia, meningitis, or perinatal infection during pregnancy. Invasive listeriosis has been reported to be 13 to 100 fold more frequent in pregnant women than the general population (Desai and Smith, 2017; Kourtis *et al.*, 2014). *L. monocytogenes* is transmitted to the foetus vertically from the mother either through the placenta, or by inhalation of infected amniotic fluid. Infection of the neonate can occur during birth (Becroft *et al.*, 1971).

Depending on the stage of pregnancy, listeriosis can result in abortion or stillbirth, or neonatal septicaemia, meningitis or mortality (Mateus *et al.*, 2013). In contrast, the maternal infections are rarely severe and there is potential that many maternal cases are detected as a consequence of neonatal infections or the increased awareness of listeriosis in pregnancy.

Listeriosis can occur at any time throughout pregnancy although foetal infections mainly occur during the third trimester, with only about 3% of cases occurring in the first trimester (Desai and Smith, 2017; Madjunkov *et al.*, 2017). Foetal mortality is more likely during the first trimester but this observation could be confounded, as perinatal listeriosis is difficult to diagnose during this period unless miscarriage occurs (Desai and Smith, 2017; Madjunkov *et al.*, 2017).

Almost all (99%) of *Listeria* infections are foodborne and outbreaks of listeriosis have been reported worldwide (Mateus *et al.*, 2013). Listeriosis is unusual compared to other foodborne

diseases in that it can have a long incubation period, particularly in pregnancy. The median incubation period for invasive listeriosis in pregnant women has been reported as 27.5 days (range 17–67 days), which is notably longer than for other clinical forms of invasive listeriosis (central nervous system, bacteraemia) (Goulet *et al.*, 2013). The majority (≥95%) of human cases of listeriosis are caused by three (1/2a, 1/2b and 4b) of the thirteen recognised *L. monocytogenes* serotypes (Datta and Burall, 2018).

3.2 *TOXOPLASMA GONDII*

Generic information on the survival, growth and inactivation, transmission routes and surveillance data for *T. gondii* is included in Appendix B.

The recently updated Risk Profile: *Toxoplasma gondii* in red meat and meat products (King, 2017) is a good background source of information with a New Zealand perspective.

T. gondii is a protozoan parasite with a complex life-cycle involving three infectious stages: sporozoites, tachyzoites and bradyzoites (Figure 1). Cats are the only known definitive host. *T. gondii* reproduces sexually in cats and are shed in large numbers with faeces, as environmentally-resistant oocysts. These oocysts become infective after a sporulation period of 1–7 days (Pittman and Knoll, 2015; Shapiro *et al.*, 2019). The mature oocysts are able to infect intermediate hosts including humans and livestock animals. After ingestion, sporozoites in the *T. gondii* oocysts transform into tachyzoites, which spread and replicate in the body (Blader *et al.*, 2015). Under pressure from the host's immune response, the tachyzoites convert to intracellular bradyzoites. Bradyzoites replicate slowly and form tissue cysts (Opsteegh *et al.*, 2016; Watts *et al.*, 2015). These cysts can form in organs, neural tissues and muscle tissues.

Consumption of undercooked or raw meat containing cysts is one of the recognised transmission routes to humans. Viable cysts can also be present in processed meats when their manufacture does not include a cook step or other controls such as salt, irradiation or pressure treatment. Once consumed by humans, the bradyzoites in cysts transform into tachyzoites and the infection commences (Robert-Gangneux and Darde, 2012).

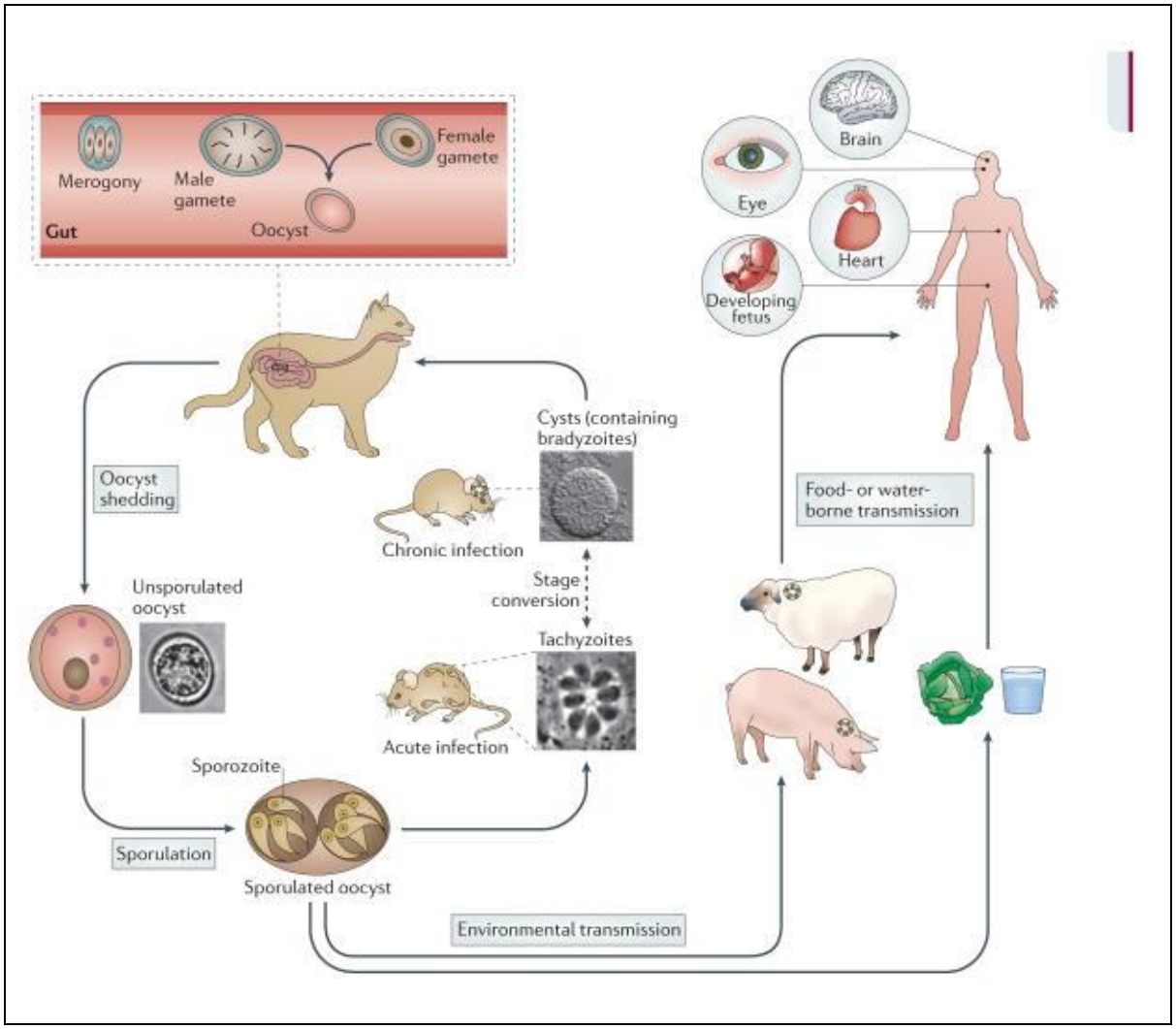

Figure 1. The life cycle of *T. gondii*.

Reproduced from Hunter and Sibley (2012), publicly available from https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3689224/ (accessed 4 March 2020)

Infection (acute toxoplasmosis) in humans is often asymptomatic, but may result in flu-like symptoms or swollen glands, or eye disease. Infections in immunocompromised people commonly manifest as swelling in the brain and can be severe and life-threatening. Immunocompromised people can become symptomatic when cysts already present in their muscles or other parts of the body reactivate (latent toxoplasmosis).

Healthy pregnant women who have already been infected by *T. gondii* and developed an immune response before becoming pregnant have a low risk of being infected again during pregnancy. Rarely, new infections can occur in those with prior immunity (Elbez-Rubinstein *et al.*, 2009). One study suggests that pregnant women are more susceptible to *T. gondii* infection compared to non-pregnant women, finding that the risk of seroconversion for toxoplasmosis was 2.2 times higher in pregnant women (Avelino *et al.*, 2003).

Congenital toxoplasmosis can arise if a woman is infected a few months before conception, since tachyzoites can still be present in her body during the early stages of pregnancy, but this appears to occur rarely (Vogel *et al.*, 1996). When infected during pregnancy, tachyzoites in the mother's body can cross the placental barrier and infect the baby, causing congenital toxoplasmosis. The foetus lacks a mature immune system so tachyzoites can

replicate with little resistance (Blader *et al.*, 2015). The mother may be asymptomatic, but the foetus might be aborted or have birth defects. Eye disease can develop later in the child's life (Gras *et al.*, 2001; Opsteegh *et al.*, 2011). Babies can also be asymptomatic at birth. Clinical problems in newborns are more likely if the mother is infected during the first half of gestation compared to the second half, but the risk of *T. gondii* transmitting from the mother to the unborn child is lowest during the first trimester (Li *et al.*, 2014; Rico-Torres *et al.*, 2016).

Tachyzoites can also be released into the milk of a lactating mammal. Humans can become infected when drinking contaminated raw milk from dairy animals with an active infection, and similarly babies can ingest tachyzoites when breastfed by mothers with an active infection (Oz, 2017). However, there is very little evidence of the latter occurring (Capobiango *et al.*, 2015).

3.3 *SALMONELLA* SPP.

Generic information on the survival, growth and inactivation, transmission routes and surveillance data for *Salmonella* spp. is included in Appendix B.

Salmonella is a very diverse genus, classified into two species, six subspecies and more than 2,600 serotypes (Dekker and Frank, 2015). Typhoidal and non-typhoidal serotypes belong to the same subspecies (*Salmonella enterica* subsp. *enterica*).

The typhoidal serotypes (*Salmonella* Typhi and *Salmonella* Paratyphi) are restricted to the human host. The disease, typhoid fever, can have a wide-ranging clinical presentation and unusual symptoms, such as pancreatitis or pneumonia, can make it challenging to recognize in regions of low prevalence (Yamasato and Burlingame, 2018). A pink macular rash can be seen in up to 30% of patients, but this can be missed due to presenting early in the course of infection (Guirguis *et al.*, 2017). The most common symptoms are generalised malaise, fever, abdominal discomfort, diarrhoea, and headache (Guirguis *et al.*, 2017).

Transplacental infections can occur when a pregnant woman develops typhoid fever and, in the absence of antibiotic treatment, mortality of both foetus and mother can occur (Crump *et al.*, 2015; Gluck *et al.*, 1994; Guirguis *et al.*, 2017). Such severe outcomes are uncommon among adequately treated patients (Touchan *et al.*, 2009). A study in Pakistan found that pregnancy was a risk factor for developing typhoid but no notable effect on birth outcomes was apparent between pregnant women with typhoid fever and pregnant women without typhoid fever (Sulaiman and Sarwari, 2007).

Symptoms for non-typhoid *Salmonella* infections, or salmonellosis, are similar to typhoid fever (Coughlin *et al.*, 2003; Delcourt *et al.*, 2019). Most non-typhoidal serotypes cause mild enteritis but severe systemic disease can develop. The most common serotypes associated with foodborne disease are *Salmonella* Typhimurium and *Salmonella* Enteritidis (Uzzau *et al.*, 2000).

There appears to be no increased susceptibility to salmonellosis when pregnant (Smith, 1999). This is substantiated by a study of 30,471 women who were screened for *Salmonella* faecal carriage at the time of delivery (Roberts and Wilkins, 1987). Rectal swabs of 60 (0.2%) women yielded *Salmonella* spp., 43 (72%) of whom were asymptomatic. Seven of the babies born to the 60 *Salmonella*-positive women (12%) also excreted *Salmonella*, all of which were the same serotype as those isolated from their mothers. No mother or child suffered invasive disease or maternal complications. Another study examined the relationship between occupational contact with animals and adverse pregnancy outcomes in

380 pregnant women in Denmark (Kantsø et al., 2014). Whilst women with occupational exposure to animals were more likely to have serological evidence of a past infection with *Salmonella* than unexposed women, it was concluded that there was no clear evidence of an association with adverse pregnancy outcomes (Kantsø et al., 2014).

A study using placental explants to investigate the extent to which non-typhoidal *Salmonella* can infect the human placenta found that *S.* Typhimurium in low numbers can infect all gestational stages but bacterial replication was nominal (Perry *et al.*, 2019). First trimester placenta were the most susceptible.

Table 2 lists published examples of cases of *Salmonella* infection during pregnancy. Most published reports involve *S*. Typhi but this list provides evidence that, rarely, non-typhoidal *Salmonella* serotypes may also cause sepsis and foetal loss during pregnancy.

Table 2. Examples of *Salmonella* infections during pregnancy[1]

***Salmonella* Serotype**	**Location**	**Gestation (Weeks)**	**Patient Age (Years)**	**Outcome (Foetus)**	**Suspected Transmission/Risk Factors**	**Reference**
Enteritidis	Belgium	14	24	Lost	Travel to Morocco	Delcourt *et al.* (2019)
Heidelberg	USA	28	25	Survived	Consumed undercooked chicken	Ault *et al.* (1993)
Montevideo	Ireland	34	NR[2]	Survived	Partner worked at poultry farm	Rai *et al.* (2014)
Non-typhoid serogroup C1	USA	13-28	NR	Lost	NR	Scialli and Rarick (1992)
Oranienburg	USA	12	22	Lost	Consumed scrambled eggs	Zettell *et al.* (1995)
Paratyphi A	India	26	23	Lost	NR	Sethi *et al.* (2017)
Typhi	USA	26	21	Survived	NR	Gluck *et al.* (1994)
Typhi	USA (Hawaii)	32	17	Survived (emergency caesarean)	Recently arrived from Marshall Islands	Yamasato and Burlingame (2018)
Typhi	USA (Hawaii)	20	21	Survived	Household contacts unwell with respiratory symptoms	Yamasato and Burlingame (2018)
Typhi (ceftriaxone-resistant)	Denmark	15	30's	Survived	Travel to Pakistan	Engsbro *et al.* (2019)
Virchow	UK	16	34	Lost	Consumed undercooked egg omelette (whilst in Turkey)	Coughlin *et al.* (2003)

[1] Examples are from countries non-endemic for *Salmonella* infection, with the exception of the case of *S.* Paratyphi A infection (included to demonstrate this serotype can also cause severe health outcomes for pregnant women).
[2] NR, not reported.

Early diagnosis and treatment of *Salmonella* infection during pregnancy is associated with good outcomes and it is recommended that pregnant women with diarrhoeal illness be tested for salmonellosis by stool culture (Scialli and Rarick, 1992). Antibiotics might be administered. Vaccines are available for *S.* Typhi but these are administered cautiously to pregnant women (Psarris *et al.*, 2019).

3.4 *CAMPYLOBACTER* SPP.

Generic information on the survival, growth and inactivation, transmission routes and surveillance data for *Campylobacter* spp. is included in Appendix B.

Campylobacter spp. usually exist as commensal bacteria in food animals (Silva *et al.*, 2011). In humans, the disease, campylobacteriosis, typically presents as self-limiting gastroenteritis. Severe complications within the general population, such as bacteraemia, are quite rare. Long-term sequelae include Guillain-Barré syndrome (GBS) and reactive arthritis (Smith, 2002).

Campylobacteriosis can be developed in any age group but is most often reported in infants aged <1 year and young adults in their twenties, with the incidence higher in males (up to 45 years of age) (O'Brien, 2017). *Campylobacter jejuni* is the species most often identified in people with campylobacteriosis but other species, such as *Campylobacter coli* and *Campylobacter upsaliensis*, can also cause human disease (Kaakoush *et al.*, 2015). *Campylobacter fetus* has been most often recognised as an infectious agent of animals, often causing septic abortion in farm animals (Duncan *et al.*, 2014). Human *C. fetus* infections are rare and largely appear among immunocompromised patients, with direct animal contact being an important transmission route (Igwaran and Okoh, 2019; Wagenaar *et al.*, 2014). *C. fetus* infection during pregnancy is uncommon in humans (Fujihara *et al.*, 2006).

Like the general population, maternal campylobacteriosis tends to be mild and self-limiting but there are some reported cases of foetal death (Farrell and Harris, 1992; McDonald and Gruslin, 2001; Simor and Ferro, 1990; Skuhala *et al.*, 2016; Smith, 2002). These occasional reports suggest that complications during pregnancy as a result of *Campylobacter* infection, including abortions, still births and neonatal deaths, are uncommon. However, a 2015 review suggested that adverse foetal outcomes as a result of campylobacteriosis during the second and third trimester might be occurring more frequently than thought (Kuperman-Shani *et al.*, 2015). Campylobacteriosis associated with abortion in pregnant women is often reported to occur following an aggressive bowel infection that results in sepsis, with the infection eventually transmitted to the foetus (Simor and Ferro, 1990). *C. jejuni*, *C. coli*, *C. fetus* subsp. *fetus*, and *C. upsaliensis* have all been isolated from such cases but the clinical presentation and outcome of abortion does not differ between these species (Kaakoush *et al.*, 2015). Enteritis that occurs in infants born to women with *Campylobacter*-associated diarrhoea may occur through faecal-oral spread at or near the time of delivery (Gribble *et al.*, 1981).

There is no evidence that healthy pregnant women are at increased risk of *Campylobacter* infection, and the risk of complications such as GBS, reactive arthritis or bacteraemia appears similar to that of the general population (Smith, 2002).

4. CHEMICAL HAZARDS AND PREGNANCY

4.1 MERCURY

Mercury may occur in food in the inorganic form or as methylmercury. From a human health perspective it is the methylmercury form that is of concern since methylmercury is more readily absorbed into the bloodstream than inorganic mercury and is able to readily cross the placental and blood-brain membranes. In finfish or shellfish (fish) mercury is most commonly found in the form of methylmercury and for the purposes of health risk assessment it is assumed that any mercury present is in this form.

4.1.1 Dietary methylmercury exposure

The 2016 New Zealand total diet survey (NZTDS) estimated the mean dietary exposure to methylmercury of an adult New Zealand female to be 0.43 µg/kg body weight/week, based on a simulated typical diet (Pearson *et al.*, 2018). This represents approximately 27% of the provisional tolerable weekly intake (PTWI) for methylmercury. In finfish samples or finfish-derived foods included in the 2016 NZTDS, methylmercury accounted for 95% of the mercury present. In the 2016 NZTDS fresh and battered fish accounted for most of the estimated weekly dietary exposure for an adult female.

A reassessment of dietary methylmercury exposure estimates for the 2009 NZTDS, using dietary modelling, indicated that adult female New Zealanders with high levels of fish consumption could be exposed to levels of methylmercury above the PTWI (Cressey, 2016).

4.1.2 Health risks associated with mercury in pregnancy

Risks for the pregnant woman

Two notable episodes of environmental contamination of mercury that resulted in deaths and illness have provided epidemiological evidence of mercury toxicity to humans. The first occurred in Japan beginning in the 1950s, when industrial discharge contaminated seafood in Minamata Bay. More than 900 people died after eating highly contaminated seafood. An additional 20,000 individuals were thought to have suffered various other forms of neurological damage from this episode (Health Canada, 2007). A second event occurred in Iraq in the winter of 1971–1972 when a food shortage resulted in seed grain coated with a mercury-containing fungicide being used for making bread. Some 6,000 cases were admitted to hospitals and as many as 40,000 individuals may have been poisoned.

A wide range of adverse effects have been observed in humans following methylmercury exposure, the severity largely depending on the dose and duration of exposure. The primary organ affected by methylmercury toxicity is the nervous system, leading to loss of nerve function and muscle control (ataxia), visual disturbances, impaired hearing, paralysis and death (JECFA, 2004).

Chronic exposure to mercury may have adverse effects on the immune system (Maqbool *et al.*, 2017) and there is emerging evidence of potential cardiovascular effects (Genchi *et al.*, 2017a).

There is no reported evidence to suggest that pregnant women are any more susceptible to the toxic effects of mercury than the general population.

Risks for the foetus and infant

Methylmercury readily crosses both the blood-brain barrier and the placenta and the developing foetus is considered to be the most sensitive sub-population. Evidence of neurotoxicity caused by chronic foetal exposure to low doses of methylmercury has come primarily from epidemiological studies of fish-eating populations. While a number of epidemiological studies have examined associations between maternal mercury status and neurotoxicity in the offspring, the following section focuses on studies that specifically considered a food source for maternal mercury burden, particularly from fish consumption.

Two large and carefully performed longitudinal studies were carried out in the Seychelles islands (Davidson *et al.*, 1998; Myers *et al.*, 2003) and the Faroe Islands (Debes *et al.*, 2006; Grandjean *et al.*, 1997).

In the Faroe Islands study, a cohort of 1022 singleton births was recruited, with children followed for up to 14 years to evaluate neurobehavioural development. Prenatal mercury exposure was determined on the basis of mercury in cord blood and maternal hair. It should be noted, that the diet in the Faroe Islands includes episodic consumption of large marine mammals (pilot whales), as well as fish. The major maternal source of methylmercury exposure was estimated to be from consumption of pilot whales (Weihe *et al.*, 2005).

The impact of postnatal exposure to mercury was examined in a sub-cohort of 583 breastfed infants (Grandjean *et al.*, 1995). Contrary to expectations, at 12 months of age, early attainment of developmental milestones (i.e. sitting, creeping and standing) was associated with higher mercury concentrations in infant hair, suggesting that if exposure to methylmercury from human milk had any adverse effect on milestone development in these infants, the effect was compensated for or overruled by the advantages associated with breastfeeding.

At age 7 years, decrements in attention, language, verbal memory and, to a lesser extent, motor speed and visual-spatial function and delays in brainstem auditory-evoked potentials were associated with prenatal methylmercury exposure (previously determined from mercury concentrations in cord blood and maternal hair) (Grandjean *et al.*, 1997). Deficits in motor, attention and verbal tests, and delayed brainstem auditory-evoked potentials persisted at follow-up at 14 years (Debes *et al.*, 2006; Murata *et al.*, 2004). Measurements of child hair mercury levels at 7 and 14 years confirmed no apparent impact of postnatal mercury exposure on neurodevelopmental indices.

The Seychelles Child Development Study was designed to study the developmental effects of prenatal methylmercury exposure in a fish-eating population. Two longitudinal birth cohort studies, referred to as the pilot study and the main study, were performed, including 217 and 804 mother–child pairs, respectively (Myers *et al.*, 1995a; Myers *et al.*, 1995b). Neither study detected significant neurobehavioural deficits in children with high prenatal mercury exposure (maternal hair mercury) at a mean age of 66 months. Rather, the test performance of both cohorts was even enhanced in some instances. Such positive effects have been hypothesised to derive from the intake of beneficial components of fish, such as LCn3 polyunsaturated fatty acids (PUFA) (Davidson *et al.*, 2000). The main cohort from this study was most recently assessed at ages 22 and 24 years (van Wijngaarden *et al.*, 2017). No adverse associations between prenatal mercury exposure and any of the measured endpoints were observed. Some measures of attention, executive function, and delayed recall showed improved performance with increasing prenatal mercury exposure.

In a third, smaller study (*n* = 237) in New Zealand, a group of children whose mothers had eaten at least three fish/seafood meals per week during pregnancy was studied. The study reports for this study are not readily available and the following summary is from a FAO/WHO expert consultation (FAO/WHO, 2011). The fish species consumed were mainly shark, with average methylmercury levels above 2 mg/kg and reaching a maximum of 4 mg/kg. A higher prevalence of abnormal results in the Denver Developmental Screening Test was detected in children with high prenatal mercury exposure (maternal hair mercury concentration >6 mg/kg) at age 4, although significance was dependent on whether or not a single outlier was included. At age 6, poorer scores on full-scale intelligence quotient (IQ), language development, and visual-spatial and gross motor skills were associated with maternal hair mercury concentrations in the range of 13–15 mg/kg (Crump *et al.*, 1998).

More recently, a small group from an ongoing birth cohort study (*n* = 135 mother-child pairs) in Massachusetts, USA, showed a positive association between maternal fish consumption in the second trimester and cognition at 6 months in the infant, but a negative association between cognition and maternal hair mercury (Oken *et al.*, 2005). The same pattern of associations were seen when children were retested at 3 years, with an enlarged cohort (*n* = 341 mother-child pairs) (Oken *et al.*, 2008b). In contrast, examination of an even larger cohort (*n* = 1068 mother-child pairs), with children at a median age of 7.7 years, found no associations between measures of verbal and non-verbal intelligence, visual motor function or visual memory and prenatal maternal fish consumption, or mercury, long-chain n-3 polyunsaturated fatty acids (LCn3PUFA) or selenium status (Oken *et al.*, 2016).

Blood mercury was determined pre-birth in a cohort of British women, with offspring (*n* = 2062) assessed for verbal, performance and total IQ at age 8 years (Golding *et al.*, 2017). Mean full scale IQ was positively associated with maternal blood mercury. When the analysis was segmented for fish consumption, the relationship was stronger for fish-eating mothers, but negative for non-fish-eating mothers.

A cohort study in a high fish-eating region of Spain determined mercury and PUFA concentrations in maternal cord blood and assessed child (*n* = 1362) neuropsychological development at age 4-5 years (Llop *et al.*, 2017). Cord blood mercury was positively associated with the number of servings of fish consumed each week. A doubling of cord blood mercury was associated with a significant increase in score for most assessment tests considered. However, the associations were negative for children of mother consuming fewer than three fish servings per week during the first trimester.

A prospective birth cohort (*n* = 242 mother-child pairs) was recruited in north-eastern Italy (Deroma *et al.*, 2013). Total and methylmercury were determined in maternal hair and breastmilk, and in child hair. Mothers completed a questionnaire concerning the type and frequency of fish consumption during pregnancy. Children underwent neuropsychological testing at 8-9 years to assess verbal, performance and full scale IQ. Fish consumption by the mother during pregnancy was positively, but not significantly, associated with child performance and full scale IQ, but not verbal IQ. Maternal canned fish consumption was negatively associated with all measures of IQ, but again the associations were not statistically significant.

A Japanese birth cohort study, the Tohoku study of child development, included analysis for 498 mother-child pairs (Suzuki *et al.*, 2010). Maternal hair mercury was determined, while maternal seafood consumption was estimated from a semi-quantitative food frequency questionnaire. The neonatal behavioural assessment scale (NBAS) was administered to infants at 3 days after birth. A significant (p = 0.05) negative association was found between

maternal hair mercury and the motor cluster component of NBAS, but not other components. A positive, but non-significant association was found between maternal seafood consumption and the motor cluster score.

The potential for maternal fish consumption to have a positive impact on child neurodevelopment has been further examined. For a cohort of 229 children from the Seychelles study, neurodevelopment was assessed at 9 and 30 months and associations examined with foetal exposure to LCn3PUFA and methylmercury (Strain *et al.*, 2008). At 9 months, psychomotor development index (PDI) was positively associated with exposure to LCn3PUFA, with the association stronger after adjustment for prenatal methylmercury exposure. No significant association between LCn3PUFA and PDI was apparent at 30 months. An adverse negative association between PDI and prenatal methylmercury exposure was apparent at 30 months, but only when LCn3PUFA measures were included in the analysis.

Examination of data from a large cohort (*n* = 25,446) from the Danish National Birth Cohort found positive associations at 6 and 18 months between child development scores and both fish consumption during pregnancy and breastfeeding (Oken *et al.*, 2008a).

The studies summarised above are largely consistent in concluding that prenatal, but not postnatal, exposure to mercury/methylmercury can have an adverse impact on child neurodevelopment and that deficits can persist, at least into adolescence. However, maternal fish consumption, as the source of methylmercury exposure, can also have a positive impact on child neurodevelopment. The positive impact is probably mediated by the presence of LCn3PUFA in fish. Therefore, the type and quantity of fish consumed are likely to dictate the impact of maternal fish consumption on child neurodevelopment. Food safety advice during pregnancy needs to balance the need to minimise methylmercury exposure against the nutritional advantage of fish consumption.

4.2 CAFFEINE

Caffeine (CAS 58-08-2) belongs to a class of compounds called methylxanthines. Caffeine has a range of pharmacological and psychological effects both beneficial (increased energy, alertness, motivation and concentration) and potentially harmful. The most important mechanism of action of caffeine is the antagonistic binding to adenosine receptors. Adenosine receptors have a role in regulating cardiac function and in the release of neurotransmitters, including norepinephrine, dopamine and serotonin, in the brain and the increase of circulating catecholamines (Nawrot *et al.*, 2003). Hence, caffeine has the opposite effect to adenosine, resulting in stimulation and increased heart rate.

4.2.1 Dietary caffeine intake

Dietary intake of caffeine has been estimated for various New Zealand sub-populations (Thomson and Schiess, 2010), using food consumption data from the 1997 National Nutrition Survey (NNS) (Russell *et al.*, 1999). The NNS contains 24-hour dietary recall records for 1388 New Zealand women of childbearing age (16–44 years) and a further 64 women who were pregnant at the time of the survey. The mean and 95th percentile estimated dietary caffeine intakes for the two groups were 226 and 623 mg/day for the general population of women of childbearing age and 125 and 479 mg/day for pregnant women, respectively.

4.2.2 Health risks associated with caffeine in pregnancy

Risks for the pregnant woman

The most significant effect of caffeine is its role as a potent stimulant of the central nervous system (CNS) although its effects are generally milder and of shorter duration than those of amphetamines (Durrant, 2002). Common adverse effects associated with excessive CNS stimulation from caffeine ingestion include dizziness, rapid heartbeat, irritability, anxiety, tremors and insomnia (Durrant, 2002; Nawrot *et al.*, 2003).

Nawrot *et al.* (2003) concluded, from a review of epidemiological studies, that consumption of caffeine at doses >300 mg/day may reduce women's fertility and increase the risk of miscarriage. The UK Committee on Toxicity (COT) also reviewed the available evidence and concluded that there was a positive association of caffeine intake with miscarriage, but that there were uncertainties relating to possible recall bias and confounding factors (COT, 2008). In addition, COT concluded that data on caffeine consumption during pregnancy and associations with other adverse effects such as pre-term birth and congenital malformations were inconclusive (COT, 2008).

An updated systemic review on the adverse effects of caffeine on human health has recently been carried out, considering additional studies published in the period 2001–2015 (Wikoff *et al.*, 2017). The review used the previously proposed intake limit of 300 mg/day as a comparator for considering findings of studies of the effect of maternal caffeine intake on maternal and offspring health. The systematic review identified 58 studies (55 observational; 3 randomised controlled trials) that provided evidence to evaluate potential impacts of the consumption of 300 mg/day of caffeine in pregnant women, or 400 mg/day in healthy adults. It was concluded that maternal caffeine intake of <300 mg/day are generally without significant concern regarding overt adverse effects on fecundability, fertility, spontaneous abortion, recurrent miscarriage and stillbirth.

Risks for the foetus and infant

The review of Nawrot *et al*. (2003) concluded that "based on available evidence, it is suggested that reproductive-aged women should consume less than or equal to 300 mg caffeine per day". The updated systemic review on the adverse effects of caffeine on human health mentioned above (Wikoff *et al.*, 2017) also considered findings of studies of the effect of maternal caffeine intake on offspring health. The systematic review identified 58 studies (55 observational; 3 randomised controlled trials) that provided evidence to evaluate potential impacts of the consumption of 300 mg/day of caffeine in pregnant women, or 400 mg/day in healthy adults, on offspring developmental effects. It was concluded that maternal caffeine intake of <300 mg/day is generally without significant concern regarding overt adverse effects on preterm birth and gestational age, birth defects, and childhood behaviour.

Fourteen studies evaluated foetal growth, of which nine of the included studies reported no effects of maternal caffeine consumption up to the comparator of 300 mg/day for birthweight, small for gestational age (SGA), intrauterine growth rate (IUGR) and placenta weight, placenta diameter, neonatal length, or head circumference. Four studies reported effects on foetal growth at maternal caffeine exposure levels below 300 mg/day. The authors of the systematic review concluded that the overall body of evidence was difficult to consolidate into a refined conclusion, as some studies showed effects on foetal growth at maternal caffeine intakes below 300 mg/day, while others showed no effect at intakes well above 300 mg/day. These differences could not easily be put down to differences in study design.

Eleven studies considered associations between maternal caffeine intake and birth defects. No associations were found for cardiovascular malformations, choanal atresia, cleft lip (with or without cleft palate) or cleft palate only, persistent cryptorchidism, and various other individual birth defects, including anotia/microtia, oesophageal atresia, diaphragmatic hernia, omphalocele, or gastroschisis at any level of maternal caffeine intake. Two studies reported an association between maternal caffeine intakes less than 300 mg/day and birth defects. However, the findings consistently failed to demonstrate a biological gradient (monotonic dose-response curve). For example, one study found marginally significant odds ratios for small intestinal atresia at maternal caffeine intakes of 10-<100 mg/day and 200-<300 mg/day, but not at intakes of 100-<200 mg/day and 300+ mg/day (Browne *et al.*, 2011). The authors of the systematic review concluded a moderate level of confidence in a conclusion that intake of up to 300 mg/day of caffeine by healthy pregnant women was without effect, with respect to birth defects in neonates.

Three studies were reviewed that examined associations between maternal caffeine intake and occurrence of childhood cancers. While some significant associations were found in some studies, results of the three studies were not consistent. Also, as the study was carried out some time after the pregnancy period, the chance of recall bias in the characterisation of the caffeine dose was high. The authors of the systematic review concurred with the findings of the International Agency for Research on Cancer (IARC) that caffeine exposure (drinking coffee in the case of the IARC assessment) was unclassifiable with respect to its carcinogenicity in humans (IARC, 2018).

Several other studies have been published since the publication of this systematic review and are summarised in the following paragraphs.

A French retrospective cohort study recruited 724 women who had given birth during a particular time period, administered a questionnaire concerning intake of caffeine-containing beverages during pregnancy and recorded characteristics of the delivery and the infant (Lamy *et al.*, 2020). Intake of 100 mg/day or more of caffeine during the second and third trimesters was significantly associated with reduced birth length. However, the association was no longer significant after adjustment for potential confounders, such as tobacco use. It was noted that other potential confounders, such as poor dietary habits, had not been accounted for.

The Norwegian Mother and Child Cohort Study considered self-reported caffeine intake and birth outcomes for 67,569 singleton mother-child pairs (Modzelewska *et al.*, 2019). Caffeine intake was assessed by a food frequency questionnaire administered at gestational week 22. A linear regression model, including consideration of confounders such as maternal age and smoking status, found a significant association between maternal caffeine intake and the infant being small for gestational age (SGA). While SGA was significantly associated with neonatal morbidity/mortality and the need for neonatal interventions, caffeine consumption was not significantly associated with these outcomes. The study authors concluded that, although maternal caffeine intake was associated with SGA, intake of <200 mg/day of caffeine did not appear to impair neonatal health.

Data for 941 Irish mother-child pairs enlisted in a cohort study were examined (Chen *et al.*, 2018). Maternal caffeine intake in early pregnancy was assessed by a food frequency questionnaire and associations with a range of birth characteristics. Maternal caffeine intake was significantly associated with lower birth weight, shorter birth length, smaller birth head circumference and shorter gestational age. Higher risk of low birth weight and pre-term birth were also observed. When comparing mother with caffeine intake ≥200 mg/day to those with

caffeine intake <200 mg/day, the high intake group had significantly elevated odds ratios for pre-term birth and low birth weight, while the low intake group did not. The reference group was those with caffeine intake <50 mg/day.

4.2.3 Advice to pregnant women

Advice from the Ministry of Health is that caffeine intake by pregnant and breastfeeding women should be limited to 300 mg/day (MOH, 2008). New Zealand Food Safety does not offer specific guidance on caffeine intake during pregnancy. FSANZ include some very general comments concerning caffeine intake, but does not provide any specific guidance (FSANZ, 2016).

The body of evidence on adverse effects to pregnant women or their offspring due to caffeine intake is less than conclusive. However, a precautionary approach would suggest that pregnant women should be encouraged to keep caffeine intake to a moderate level (<300 mg/day). The Ministry of Health's guidelines include a table of the typical caffeine content of various beverages (MOH, 2008).

5. SCIENTIFIC EVIDENCE FOR ADVICE

The food groups listed in this section are those included in New Zealand Food Safety's pull-out guide to food safety in pregnancy (New Zealand Food Safety, 2019). The focus is on the two microbiological hazards *L. monocytogenes* and *T. gondii*, for which pregnant women are at increased susceptibility for infection and the foetus is at risk of severe health outcomes. Occasionally, in this section, information has been included for *Campylobacter* spp. and *Salmonella* spp. but for these bacteria, and other microbial hazards, general food safety advice applies. Chemical hazards have been addressed in Section 4, but where New Zealand Food Safety provides advice on food chemical issues, these are also discussed in this section.

For all of these foods, application of Good Manufacturing Practice (GMP) and Good Hygienic Practice (GHP) are important controls for reducing or removing any pathogenic microorganisms.

5.1 BREADS AND CEREALS

5.1.1 Breads, all types

CURRENT ADVICE:	OK to eat

Food

Bread is a short shelf life, heated (baked) product made from fermented dough (flour, water, yeast and salt, with other optional ingredients). Baking soda can be used as an alternative raising agent. An internal temperature of 98ºC is required for complete baking but the bread temperature can exceed this during cooking, especially at the crust.[1] Part-baked breads and rolls are available, which require baking by the consumer before consumption.

Hazards

This food has not been linked to *L. monocytogenes* contamination. No growth in the final product is expected.

T. gondii is not expected to be present in bread.

Human illness

There are no reports of listeriosis outbreaks implicating bread. Foodborne illness outbreaks have been associated with uncooked bread ingredients, both in New Zealand and overseas. In New Zealand an outbreak of *S.* Typhimurium PT42 infection, involving 67 cases, was reported in 2008. Epidemiological and laboratory investigations identified flour as the vehicle and eating uncooked baking mixture containing flour as the major risk factor (McCallum *et al.*, 2013).

Controls

Baking kills all vegetative bacterial cells and will kill any *T. gondii* that may be present in raw ingredients. Cooking also lowers the moisture content of the product. Growth of bacterial

[1] New Zealand Baking Industry Research Trust, https://www.bakeinfo.co.nz/Facts/Bread-making/Science-of-bread-making/Baking (accessed 6 May 2020).

spores that have survived cooking is possible inside bread products but fungal spoilage is much more common, especially in humid conditions (ICMSF, 2005). Pathogen growth is unlikely before spoilage becomes evident.

Summary

Present New Zealand Food Safety advice is consistent with current scientific evidence. No issues specific to pregnant women identified.

5.1.2 Cakes, slices, muffins, etc: Plain

CURRENT ADVICE:	OK to eat

Food

Plain bakery items such as cakes, biscuits, slices and muffins are similar to bread products. They are relatively shelf-stable, not requiring cooling or freezing for preservation. Many long-life bakery products, such as fruitcake, are stable because they have low water activity.

Hazards

This food type has not been linked to *L. monocytogenes* contamination. No growth in the final product is expected.

T. gondii is not expected to be present in plain bakery items. The cooking process will kill any *T. gondii* that may be present in raw ingredients.

Human illness

There have been reports of viral outbreaks overseas associated with glazed bakery goods, buttercream icing and raspberry cake filling, all handled by infected food handlers (Todd *et al.*, 2009; Weltman *et al.*, 1996).

In New Zealand in 2016, a small (3 confirmed, 5 probable cases) *S.* Typhimurium PT60 outbreak was linked to domestic preparation of Christmas cookies (Pattis *et al.*, 2017). Both biscuit and biscuit dough tested positive for *Salmonella* spp.

Controls

See Breads (Section 5.1.1).

Other ingredients such as sugar, butter and oils help maintain a safe level of water activity.

Cake and slice icings usually contain high levels of sugar or fat and generally will not support microbial growth due to the low water activity but, as for all foods, their handling by sick food handlers can introduce microbial pathogens to the food.

Summary

Present New Zealand Food Safety advice is consistent with current scientific evidence. No issues specific to pregnant women identified.

5.1.3 Cakes, slices, muffins, etc: With added cream or custard

CURRENT ADVICE:	Don't eat (unless cream is newly opened and custard is home-made and fresh)

Food

Bakery products as described above may be filled with various ingredients such as cream, custard, nuts, fruit and jam. Cream and custard filled varieties present the highest risk in terms of microbial growth.

Cream and custard are excellent microbiological growth media. A New Zealand survey of 250 cream and custard filled bakery products found the majority had high water activity (≥0.98) and a pH of ≥6.0 (NZFSA, 2007).[2] These foods were being displayed at retail at an average temperature of 8ºC (range 1–25ºC).

Hazards

A total of 250 cream and custard filled bakery products were collected from New Zealand bakeries and tested for microbiological quality (*Escherichia coli*, coagulase positive staphylococci, *Bacillus cereus* and *Salmonella* spp.) (NZFSA, 2007). *L. monocytogenes* was not included in the range of analyses. The majority (87%) of the samples were considered to be of good microbiological quality and three samples were considered "potentially hazardous" because of high *B. cereus* concentrations.

As *L. monocytogenes* is a common environmental contaminant and often associated with raw ingredients, it should also be considered a potential hazard in these products. There is potential for growth of *L. monocytogenes* (see "Custard" and "Cream" sections below). A French study of cakes containing butter-cream, whipped dairy cream or custard, found *L. monocytogenes* in 13.7% of samples (Ferron and Michard, 1993).

T. gondii is not expected to be present in these bakery items unless the cream, custard, etc., has been made using unpasteurised milk.

Human illness

In Canada, an outbreak of salmonellosis was linked to cream-filled eclairs and profiteroles.[3] No listeriosis outbreaks associated with cream or custard filled baked goods were located.

Controls

At the time of the 2007 New Zealand bakery product survey, most bakeries throughout the country used good food safety practices (NZFSA, 2007). However, problems with food hygiene, handling, and manufacturing processes were identified in some premises. These included inadequate storage conditions, improper use of equipment (e.g. reuse of piping bags) and inappropriate actions regarding left over product (e.g. stored for use the following day).

Summary

Present New Zealand Food Safety advice is consistent with current scientific evidence. *L. monocytogenes* can be introduced during preparation, and growth of *L. monocytogenes* is

[2] Unpublished data associated with this survey.
[3] https://www.canada.ca/en/public-health/services/public-health-notices/2019/outbreak-salmonella.html (accessed 13 May 2020).

possible in these products. The cool chain may not be maintained thereby allowing microorganisms to grow to unsafe levels.

5.1.4 Cereals: Breakfast cereals, rice, pasta, etc.

CURRENT ADVICE:	OK to eat – refer to dairy products below for advice on milk

Food

Breakfast cereals are manufactured by flaking, puffing or extruding cereals such as wheat, maize, oats and rice. Moisture may be applied during production but it is removed during the cooking process.

After harvest, rice is milled and processed to a variety of end points (e.g. whole grain, white).

Pasta is produced from flour and water and may include other ingredients including egg. Conventional pasta is dried but pathogens may survive the process. Fresh pasta receives little drying and is stored refrigerated, usually in sealed packaging that may contain a modified atmosphere to prevent spoilage.

Hazards

With the exception of fresh pasta, bacterial growth will only occur when moisture is added to these products and they are allowed to remain under favourable conditions for microbial growth (i.e. not eaten within a short time of preparation). Examples include cereal in milk, or cooked porridge, rice or pasta left at room temperature. *B. cereus* is the bacterial species most likely to survive and germinate in cooked cereals if these are not eaten or refrigerated. Refer to sections on “Salads” and “Sushi” for further discussion of hazards associated with cold cooked cereal products, such as pasta and rice.

Survival and growth of *L. monocytogenes* is not generally expected in low moisture foods including dried cereal products, rice and dried pasta. Contamination with *L. monocytogenes* may occur with the use of additives such as dried fruit, nuts, and flavourings but the final product maintains a low water activity (Taylor *et al.*, 2019). Consumption with pasteurised milk is low risk.

T. gondii is not expected to be present in cereals but no surveys were located. Cooking will kill any *T. gondii* that may be present.

Human illness

Refer to sections on “Salads” and “Sushi” for further discussion of human illness associated with cold cooked cereal products, such as pasta and rice.

Controls

There are no additional controls for breakfast cereals since, other than porridge, these are usually consumed without heating.

Rice, fresh pasta and dried pasta are all boiled before consumption. This cooking will destroy any vegetative microbial cells.

Summary

Present New Zealand Food Safety advice is consistent with current scientific evidence. No issues specific to pregnant women were identified. Suggest addition of referral advice on salads and sushi as having cereal ingredients, as well as dairy products. Further data are

needed for fresh pasta, including filled varieties, to determine whether specific advice is needed for this food (Section 5.8.2).

5.2 DAIRY

5.2.1 Unpasteurised (raw) milk

CURRENT ADVICE:	Don't drink or use

Food

Milk is made up of water, protein, fat, lactose, vitamins and minerals, with the types and proportions of each varying with animal breed, feed, age and phase of lactation (King *et al.*, 2014). Milk is almost neutral in pH and has high water and nutrient content, so is an excellent substrate for microbial growth.

In New Zealand, most animal milk comes from cows. Milk is also obtained from dairy sheep, goats or buffalo, but often this milk is used for making dairy products such as yoghurt or cheese (King *et al.*, 2014). Milk from other animals may be imported but this must be pasteurised.

Raw milk is animal milk that has not been processed in a way intended to alter its quality or composition (such as by pasteurisation) or having had anything added to or removed from it (Elias, 2015).

Hazards

Any pathogen that is associated with milk-providing animals could be introduced into the product from faecal contamination, and to a lesser extent, mastitis or a systemic infection. Secondary contamination may result from equipment and handling.

Listeria

The main sources of *Listeria* contamination of raw milk are faecal contamination of udders and milking equipment, and to a lesser extent listerial mastitis. The risk of *L. monocytogenes* in raw milk was recently profiled for New Zealand (King *et al.*, 2014).

Raw cows' milk in New Zealand has a reported prevalence of *L. monocytogenes* in the range of 0.7–4.1 % (King *et al.*, 2014; Paulin *et al.*, 2015). Concentrations of the pathogen are generally low, with positive samples containing <1 MPN/mL or <1 CFU/mL. A survey of goats' milk in New Zealand reported detection of *L. monocytogenes* in 2 of 60 samples (Tanya Soboleva, New Zealand Food Safety, personal communication, January 2014).

Studies have shown that *L. monocytogenes* can survive and grow in raw cows' milk, including at 4°C. Data indicate that there is a lag period of approximately 5 days before growth of >1 log occurs in raw milk inoculated with strains of *L. monocytogenes* (King *et al.*, 2014). Milk held at a higher temperature will result in more growth, e.g. the concentration of *L. monocytogenes* increased by 0.8–2.3 log in raw milk kept at 15°C for 65 hours (Gay and Amgar, 2005). Raw milk naturally contaminated with ~10^4 CFU/mL *L. monocytogenes* exhibited generation times of 25.3±3.5, 10.8±1.2 and 7.4±0.99 hours when incubated at 4, 10, and 15°C respectively (Farber *et al.*, 1990).

The FDA/FSIS (2003) information on *L. monocytogenes* in Appendix C shows growth was observed in all types of raw milk.

Since 2015 there has been one instance reported by MPI of unpasteurised (raw) milk sold to consumers being recalled in New Zealand due to *L. monocytogenes* contamination.[4]

Toxoplasma

T. gondii does not replicate outside the host so will not grow in food. If present in dairy products, *T. gondii* is most likely to be in the tachyzoite stage. It is possible that cysts could be present but confirmation of this requires further study (Dubey *et al.*, 2014).

There are no New Zealand data on the prevalence of *T. gondii* in raw milk. Seroprevalence studies show that goats and sheep in New Zealand can be exposed to this parasite (summarised in King (2017)). Dairy cows are mostly raised in outdoor environments so can also be exposed to *T. gondii* oocysts.

Surveys in other countries have detected *T. gondii* DNA in raw milk from sheep, goats and cows (Cisak *et al.*, 2017; Gazzonis *et al.*, 2019; Iacobucci *et al.*, 2019; Mancianti *et al.*, 2013; Veronesi *et al.*, 2018).

Viable *T. gondii* (mouse bioassay) have been detected in the milk of lactating goats orally inoculated with oocysts, with the positive samples appearing intermittently during the lactation period (Dubey *et al.*, 2014). Another study tested milk from naturally infected lactating ewes three times during a 120-day lactation period (Ossani *et al.*, 2017). Milk from eight sheep with the highest *T. gondii* antibody titres was tested by mouse bioassay, which indicated the presence of tachyzoites in samples collected from one animal at 45 days, and the other seven at 90 days, but none at 120 days, suggesting peak production of tachyzoites mid-lactation. However, the results from the mouse bioassay were based on the presence of *T. gondii* DNA in mouse brains since cysts were not observed in these tissue. In a third study, *T. gondii* DNA and RNA were detected in bulk milk from seropositive sheep herds, with the presence of RNA indicating that viable tachyzoites were in the milk (Ranucci *et al.*, 2020).

A systematic review and meta-analysis of data from 39 case-control studies where raw milk consumption was investigated as a risk factor for toxoplasmosis found an overall significant correlation (Boughattas, 2017). When investigated categorically, consumption of cows' milk was not significantly correlated with toxoplasmosis but consumption of goats' milk was. Raw cows' milk is considered to be "of negligible importance" in the epidemiology of toxoplasmosis (Dubey, 1986; Dubey *et al.*, 2014).

Controls

Raw milk receives no heat treatment or other processing step to reduce the microbial load. It is expected that consumers will keep raw milk refrigerated.

Human illness

Outbreaks of campylobacteriosis (Section 3.4), STEC infection and cryptosporidiosis have been linked to raw milk consumption in New Zealand.[5]

A systematic review of the scientific literature found there was evidence of "moderate support for a causal link between the consumption of raw milk/raw milk products and infection with *Listeria monocytogenes*" (Jaros *et al.*, 2008). Outbreaks of listeriosis

[4] https://www.mpi.govt.nz/food-safety/food-recalls/recalled-food-products/ (accessed 23 March 2020).
[5] https://www.mpi.govt.nz/food-safety/food-safety-for-consumers/is-it-safe-to-eat/raw-milk/ (accessed 11 May 2020).

associated with raw milk appear to be uncommon with few being reported. In 2014 two people in the USA (California and Florida) were infected with the same strain of *L. monocytogenes* and one person died. The source was tracked to contaminated chocolate-flavoured raw milk from a supplier in Pennsylvania.[6] Raw milk has not been implicated in any listeriosis outbreaks reported in New Zealand.

Tachyzoites are sensitive to acid-pepsin digestion so can be inactivated when exposed to gastric juice in the stomach, although survival for up to two hours has been documented (Dubey *et al.*, 1998). Experiments have shown that the addition of milk to gastric fluid raises the pH and protects tachyzoites (Koethe *et al.*, 2017). Outbreaks of toxoplasmosis implicating unpasteurised goat's milk consumption have been reported overseas, showing human illness can occur (Sacks *et al.*, 1982; Skinner *et al.*, 1990). In one familial outbreak, the presence of tachyzoites in milk from one of the family's goats was confirmed by mouse bioassay (Chiari and Neves, 1984).

Summary

Present New Zealand Food Safety advice is consistent with current scientific evidence. Outbreaks of toxoplasmosis or listeriosis linked to raw milk have occurred overseas. In addition, a range of other pathogenic microbes can also be present in raw milk and raw milk-associated outbreaks have been reported in New Zealand. Consumption of raw milk poses a significant health risk.

5.2.2 Pasteurised milk

CURRENT ADVICE:	Drink or use within two days of opening

Food

See unpasteurised (raw) milk.

Pasteurised milk is raw milk from dairy animals that has been heat treated. The most commonly used pasteurisation method for milk products in New Zealand is the high temperature short time (HTST) method (71.7°C for 15 seconds) (Hudson and Wong, 2003). A low temperature long time (LTLT) method (63.5°C for 30 minutes; the "holding method") is occasionally used for batch pasteurisation for certain products. Extended shelf life and ultra-heat treated (UHT) milk is heat treated at 120–124°C and 134–135°C (or higher) respectively, for short periods.

Hazards

Listeria

Pasteurisation is sufficient to inactivate *L. monocytogenes* in raw milk but contamination may sometimes occur during post-pasteurisation handling including during the bottling or packaging stage. Therefore the prevalence of *L. monocytogenes* in pasteurised milk at retail is very low. A comprehensive study completed in the United States found only one sample positive among 5,519 (0.018%) samples of pasteurised fluid milk products tested (Frye and Donnelly, 2005). Samples were collected from products near their sell-by date and included chocolate milk, whole milk and non-fat milk products.

If milk is inadequately heated or contaminated post-pasteurisation it can serve as an excellent growth substrate. In a study of the growth of the *L. monocytogenes* in pasteurised

[6] https://www.cdc.gov/listeria/outbreaks/raw-milk-03-16/index.html (accessed 30 April 2020).

milk the behaviour of six strains was observed in milk held at 15°C for 65 hours. A 2–3.8 log increase in cell numbers occurred, with some variability observed across different strains of *L. monocytogenes* (Gay and Amgar, 2005).

The FDA/FSIS (2003) information on *L. monocytogenes* in Appendix C shows growth was observed in all types of heat-treated milk. For pasteurised milk held at 4°C, a 2 log increase in 7 days was observed.

Toxoplasma

Pasteurisation kills *T. gondii* so this parasite is not expected to be present in pasteurised milk (Dubey *et al.*, 2014).

Human illness

Listeriosis outbreaks implicating pasteurised milk have been reported overseas but these appear to be associated with unusual circumstances and are not common.

An outbreak of 49 cases of listeriosis (7 foetal or infant and 42 immunocompromised adults) in the USA was reported by Fleming *et al.* (1985). Although *L. monocytogenes* was not isolated from the implicated pasteurised product, there was a strong epidemiological link between those with the disease and consumption of whole or low fat milk produced at a single plant. There was no evidence of pasteurisation failure at the plant. It was hypothesised that the milk had come from a *Listeria*-infected herd and it is possible that the milk contained an unusually large inoculum which the pasteurisation process reduced, but not to a concentration which prevented disease.

Pasteurised chocolate milk was the source of an outbreak caused by *L. monocytogenes* involving 45 cases in the USA (Dalton *et al.*, 1997). The milk had been consumed at a picnic and several complaints about the taste and quality were received from the 60 people who had consumed the product. The probable cause of the outbreak was post-pasteurisation contamination due to poor hygiene practices at the production company. Inappropriate storage temperatures on the way to the picnic were also a major contributing factor.

Controls

Pasteurisation treatments normally allow good safety margins to ensure the removal of pathogens and it is generally agreed that the minimum standards specified for milk (72°C for 15 seconds or 63°C for 30 minutes) are sufficient to inactivate *L. monocytogenes* in milk (ICMSF, 2005).

A laboratory study using mouse bioassay demonstrated complete inactivation of *T. gondii* tachyzoites in goats' milk pasteurised at 63ºC for 30 min (LTLT method) (Saridewi *et al.*, 2013).

Summary

Current scientific evidence does not support present New Zealand Food Safety advice. Pasteurisation is the main control for pathogens in milk and the presence of *L. monocytogenes* in pasteurised milk would be unexpected. This is a widely consumed food and no outbreaks have been reported in New Zealand associated with this food. Outbreaks in other countries were associated with unusual circumstances. There is potential for *L. monocytogenes* growth if post-pasteurisation contamination occurs (e.g. once the packaging is opened in the home). It might be more suitable to replace the current two day restriction with additional advice to consume before the manufacturer's best before date and take

actions to prevent contamination, e.g. “keep milk refrigerated in original packaging and take care not to contaminate lids when using.”

5.2.3 Cream: Fresh, unwhipped or whipped, sour cream

(pasteurised)

CURRENT ADVICE:	Buy in sealed packs; eat within two days of opening pack

Food

Cream is the fat-rich part of milk which is usually separated by skimming. It is normally standardised to the desired fat content. The high fat content, which can range from 10 to 55%, may be protective of pathogens, and higher pasteurisation temperatures than those applied to liquid milk are required. Other heat treatments include sterilisation and UHT.

Sour cream is pasteurised cream acidified through fermentation by added lactic acid bacteria or through the use of safe and suitable acidifiers (Aryana and Olson, 2017). The pH of commercial sour cream (n=32) surveyed in the USA was approximately 4.6 (range 3.8–4.8) (Shepard *et al.*, 2013).

Hazards

In terms of microbiological safety, cream may be considered a more sensitive product than milk (ICMSF, 2005). While milk is usually consumed within a short period after opening, cream is used less regularly and packs may remain open for longer periods.

Listeria

As *L. monocytogenes* is unlikely to survive pasteurisation, post-pasteurisation contamination is the most likely route of contamination (ICMSF, 2005).

Only one entry relating to the growth rate of *L. monocytogenes* in cream is recorded in the FDA/FSIS quantitative risk assessment (Appendix C). In this study, *L. monocytogenes* in whipping cream increased by 3.3 log when stored at 4°C for 18 days, and by 4 log when stored at 8°C for 8 days (Rosenow and Marth, 1987). *L. monocytogenes* could potentially grow in sour cream but studies are needed to confirm this.

No recent data on *L. monocytogenes* prevalence in cream or sour cream at retail were located. A study reported in 1991 found no *L. monocytogenes* in pasteurised cream samples (n=40) mostly from retail outlets in Wales and England (Greenwood *et al.*, 1991).

Toxoplasma

T. gondii is not expected to be present in pasteurised cream or similar products. No surveys were located where cream products made from pasteurised milk were tested for the presence of *T. gondii*.

Human illness

No reports of outbreaks were located for New Zealand or other countries (outbreaks where cream was an ingredient in baking products are discussed in Section 5.1.3).

Controls

Pasteurisation is the primary control.

Summary

The current evidence is not sufficient to evaluate New Zealand Food Safety advice. Pasteurisation is the main control for pathogens in cream and the presence of *L. monocytogenes* in pasteurised cream would be unexpected. No outbreaks have been reported associated with this food. It can be concluded from this information that *L. monocytogenes* is unlikely to be present in sealed packs of fresh cream. However, prevalence data for *L. monocytogenes* in the range of fresh cream products consumed in New Zealand are not available and there are no data for sour cream. *L. monocytogenes* could grow in fresh cream if post-pasteurisation contamination occurs (e.g. once the packaging is opened in the home), potentially at a rate that would support the advice to use the cream within two days, although data are limited and also not available for sour cream. In the absence of additional data the current advice is protective. Future modifications of this advice might consider encouraging consumers to keep the cream refrigerated in the original packaging and to take care to prevent contamination when using.

5.2.4 Butter: All types

CURRENT ADVICE:	OK to eat, store in fridge

Food

Butter is a water-in-oil/fat emulsion, containing at least 80% fat, which is made from churned, pasteurised cream. When stored refrigerated the shelf life of butter is in the range of 6–12 weeks (Lewis *et al.*, 2006).

While margarine has similar properties to butter, it is not a dairy product and is considered under the section on spreads (Section 5.7.3).

Hazards

Listeria

Butter has not generally been considered a high-risk product with respect to *L. monocytogenes* (Lewis *et al.*, 2006). *Listeria* can be introduced in the cream used to produce the butter and will be dispersed within the aqueous phase, although this will not occur with adequate cream pasteurisation. Contamination may occur at other points along the production chain. Investigations following a Finnish butter outbreak (below) found the outbreak strain of *L. monocytogenes* in samples from the butter packing machines, a conveyor, and from floor drains suggesting widespread contamination can occur (Lyytikainen *et al.*, 2000).

Various studies have investigated the growth of *L. monocytogenes* in butter at chilled and ambient temperatures. Collectively the studies suggest that *L. monocytogenes* can survive in butter, limited growth may occur, but a decline in cell numbers may also be observed (El-Hajjaji *et al.*, 2020; Michelon *et al.*, 2015; Olsen *et al.*, 1988). Any growth that occurs may be highly variable as was observed in the butter associated with the Finnish outbreak (see below). *L. monocytogenes* did not grow in small packages of butter associated with the outbreak following incubation at 6.5–7.2°C. However, levels increased from a mean of $\log_{10}$ 0.8 to a mean of $\log_{10}$ 3.6 CFU/g in the larger 500 g packages. The concentration of *L. monocytogenes* within different samples of butter was highly variable across the incubation period despite a similar level of contamination at the beginning (Maijala *et al.*, 2001).

Butter is rarely subject to a food safety recall. In the USA, a product recall of butter and butterine (a mixture of 60% butter and 40% margarine) for *L. monocytogenes* contamination occurred in 2004. The recall was the result of routine sampling. As reported in the earlier version of this report, no illnesses were associated with the product (Gilbert *et al.*, 2010).

No data for the prevalence of *L. monocytogenes* in New Zealand butter were found but a survey in the United Kingdom in 2004 does provide some information (Lewis *et al.*, 2006). Samples (n=3,229) were taken from point of production, retail and catering premises over a two month period. Thirteen samples were positive for *L. monocytogenes* (0.4%) and concentrations in all positive samples were <10 CFU/g.

Toxoplasma

T. gondii is not expected to be present in butter made from pasteurised milk. No surveys were located where butter was tested for the presence of *T. gondii*.[7]

Human illness

Overseas outbreaks of listeriosis associated with consumption of butter have been reported. *L. monocytogenes* was isolated from implicated product in two of the outbreaks. In two of the outbreaks the outbreak strain of *L. monocytogenes* was also isolated from environmental samples collected around the plant which produced the butter (Lewis *et al.*, 2006).

As introduced above, a long-running outbreak in Finland during 1989–1999 was associated with butter made from pasteurised cream produced by a single dairy and supplied to a tertiary care hospital (Lyytikainen *et al.*, 2000). Twenty-five patients developed listeriosis and there were six deaths. *Listeria monocytogenes* serotype 3a was isolated from patients, packaged butter and the source dairy. Patients were estimated to have received doses of between 1.4×10^1 and 2.2×10^3 CFU/day based on hospital kitchen data, or between 2.2×10^4 and 3.1×10^5 CFU/day based on the maximum level detected in a wholesale butter sample (Maijala *et al.*, 2001).

Controls

Pasteurisation is the primary control. Other factors controlling pathogen growth are low water activity, salt and regulation of the emulsion droplet size.

Summary

The current evidence is not sufficient to evaluate New Zealand Food Safety advice. The presence of *L. monocytogenes* in butter made from pasteurised cream would be unexpected. There have been no notified listeriosis outbreaks caused by this product in New Zealand, which is consumed widely. This suggests that butter does not pose a significant risk to pregnant women. However, *L. monocytogenes* was detected in a large survey of butter overseas, and outbreaks of listeriosis caused by butter have been reported. A survey of New Zealand butter, including enumeration and supported by growth studies, would provide a better set of evidence to develop advice.

[7] A 2019 paper reports detection of *T. gondii* DNA in “traditional butter” but this paper has since been retracted on the basis of a compromised peer review process Alipour M, Rahimi E, Shakerian A. (2018) Retracted: Prevalence of *Toxoplasma gondii* and *Neospora caninum* in different types of raw milk and traditional dairy product samples. Journal of Food Safety; 38(6): e12575..

5.2.5 Cheese

A large amount and variety of cheese are available in New Zealand, with around 18% being imported, a figure which is increasing annually (Paulin *et al.*, 2015). Where known or specified, the greatest amount of cheese imported is “fresh’ (acid coagulated), followed by ‘processed’ cheese. It is not known what proportion of imported cheese is made from raw milk. The majority of imported cheese comes from Australia (37%), with Denmark and the United States accounting for 14% and 19% of imports respectively.

Pathogens can be introduced into cheese via raw milk, contamination after milk has been pasteurised, via added ingredients, or during processing and handling.

New Zealand risk profiles have been written for *L. monocytogenes* in soft cheeses (Lake *et al.*, 2005b), low-moisture cheeses (Lake *et al.*, 2005a), and updated in a combined document on cheese (Paulin *et al.*, 2015). These documents provide a comprehensive overview of *Listeria* in locally produced and imported cheese available to New Zealand consumers, and should be referred to if information in greater detail is needed.

There is no easy approach to categorising the huge range of cheeses available to New Zealanders. This section has generally followed the approach found in New Zealand Food Safety’s current pull-out guide but with some slight changes to the descriptions (New Zealand Food Safety, 2019). The cheeses have been grouped into those made from raw milk, low acid soft cheeses, low pH soft cheeses (often these are spreadable) and hard cheeses. Further information on cheese types is given in the Risk Profile for *L. monocytogenes* in cheese (Paulin *et al.*, 2015).

5.2.6 Cheese: Unpasteurised (raw milk) cheeses

(e.g. Roquefort, Morbier, Livarot, Reblochon, St Nectaire, Vacherin, raw milk varieties of Brie and Camembert, Sainte-Maure de Touraine, Banon, Valencay)

CURRENT ADVICE:	Don’t eat

Food

Cheeses, usually soft cheeses, made from raw (unpasteurised) milk. These may or may not be ripened and usually do not include a heat treatment during manufacture.

Hazards

Listeria

Raw milk cheeses can be contaminated by *L. monocytogenes* from the milk, during production or post-production (e.g. during ripening). Raw milk from cows and goats in New Zealand can be contain *L. monocytogenes* (Section 5.2.1). A variety of raw milk soft cheeses are also imported into New Zealand and sold at retail.

L. monocytogenes can grow, remain stable or die during cheese-making and ripening but this depends on the particular conditions. Growth can be inhibited by the presence of other microflora and sufficient reduction in pH. For example, one study of raw milk smear-ripened cheese suggested that other microflora inhibited *L. monocytogenes* growth during the first five hours of manufacture, but during ripening growth occurred because slow lactic acid production kept the pH suitable (Schvartzman *et al.*, 2011). Other laboratory experiments demonstrated that with sufficient starter culture, a rapid pH reduction prevented *L. monocytogenes* growth but did not necessarily cause the concentration of this pathogen to decrease (Schoder *et al.*, 2008; Withers and Couper, 2012). Other studies of raw milk

cheeses show the potential for *L. monocytogenes* growth during ripening, or for concentrations to remain reasonably stable (Paulin *et al.*, 2015).

Since 2008, two imported raw sheep milk cheeses have been the subject of food safety recalls for containing *L. monocytogenes* (Paulin *et al.*, 2015).[8]

Toxoplasma

T. gondii does not replicate outside the host so will not grow in food. If present in dairy products, *T. gondii* is most likely to be in the tachyzoite stage. It is possible that cysts could be present but this requires further study (Dubey *et al.*, 2014).

When a mixture of raw goats' milk and tachyzoites was used to make cheese, results from a mouse bioassay showed that *T. gondii* survived the cheese-making process and subsequent storage for 3 days at 4ºC, but was not orally infectious to mice (Dubey *et al.*, 2014). Only subcutaneous injections induced infection in mice, which suggested the tachyzoites were inactivated through gastric digestion. Viable *T. gondii* were not detected in whey.

When the raw milk from experimentally infected goats was used to make cheese, one of four cats fed this cheese began shedding *T. gondii* oocysts (the other three cats remained seronegative) (Dubey *et al.*, 2014).

In another experiment, cheeses were made from raw sheep milk naturally contaminated with viable *T. gondii* tachyzoites (based on the presence of *T. gondii* RNA as an indicator of metabolically active parasites) and ripened for 15 days (Ranucci *et al.*, 2020). While *T. gondii* DNA was detected in all cheeses after 5 and 15 days of ripening, RNA was not. The researchers concluded that the loss of viability was due to the acidification process, as the lactic acid bacteria multiplied, plus the presence of salt (mean concentration 2.48%).

Human illness

Outbreaks of invasive listeriosis, including cases related to pregnancy, have been associated with soft cheeses produced from raw milk. Countries who have reported outbreaks include France (Goulet *et al.*, 1995), Sweden (Carrique-Mas *et al.*, 2003), Switzerland (Bille *et al.*, 2006), and the USA.[9]

No listeriosis outbreaks have been attributed to the consumption of raw milk cheeses in New Zealand.

Tachyzoites are sensitive to acid-pepsin digestion so can be inactivated when exposed to gastric juice in the stomach, although survival for up to two hours has been documented (Dubey *et al.*, 1998). During a 2015-2016 outbreak of toxoplasmosis in Brazil, a case-control study found that consumption of fresh (raw milk) cheese made from cows' milk was the only significant risk factor (OR 6.97, 95% CI 1.65–37.41, $p<0.01$) (da Costa *et al.*, 2019). *T. gondii* DNA was detected in samples of this artisan cheese.

Controls

Two New Zealand producers make and sell raw milk cheese domestically under strict conditions including on farm and manufacturing controls, and a code of practice (Paulin *et al.*, 2015). Raw milk cheese, including soft varieties, may also be imported into New Zealand and

[8] https://www.mpi.govt.nz/food-safety/food-recalls/recalled-food-products/ (accessed 12 March 2020).
[9] USA: https://www.cdc.gov/listeria/outbreaks/soft-cheese-03-17/index.html (accessed 16 March 2020).

sold at retail. Imported products are allowed from countries with *L. monocytogenes* controls and public health standards accepted by MPI as being equivalent to those of New Zealand.

Summary

Present New Zealand Food Safety advice is consistent with current scientific evidence. *L. monocytogenes* can be present in raw milk cheese and these foods have caused listeriosis outbreaks in other countries. There is currently little evidence that *T. gondii* poses a significant hazard.

5.2.7 Cheese: Low acid soft pasteurised cheeses

(e.g. Brie, Camembert, blue, ricotta, mozzarella, feta, Latin-style fresh cheeses)

CURRENT ADVICE:	Generally should not be eaten uncooked while pregnant. However, OK to eat in small quantities if purchased in the manufacturer's original packaging and eaten immediately after opening. Do not reseal to eat later, and do not eat if cheese has been repackaged in a deli or supermarket as may have become contaminated.

Food

Soft (high moisture) cheeses made from pasteurised milk that have low acidity or near neutral pH (final pH generally >5, except for feta, which has a final pH of 4.4-4.6) (Abd El-Salam and Alichanidis, 2004; Cantor *et al.*, 2004; Farkye, 2004; Spinnler and Gripon, 2004). These cheeses might be fresh (e.g. queso fresco, queso blanco), ripened (e.g. Camembert, Brie, blue), salt-pickled (e.g. feta) or made using whey (e.g. ricotta) or heat (e.g. mozzarella).

Hazards

Listeria

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

A study found that *L. monocytogenes* growth on the surface of 24 types of cheese available in the USA was significantly correlated with pH values >5.5 and/or the absence of lactic acid starter cultures during cheese manufacture (Genigeorgis *et al.*, 1991). Information assembled for the FDA/FSIS Quantitative Risk Assessment (Appendix C) also shows the potential for *L. monocytogenes* growth in soft cheeses.

In a New Zealand study, growth of *L. monocytogenes* was observed within the shelf life of three brands of commercially produced ricotta cheese stored at 7.7°C. The pH values of the samples were between 5.8 and 6.5 (Gilbert *et al.*, 2010). In the same survey, two of the three brands of blue cheese tested supported the growth of *L. monocytogenes* within 74 days at 7.7°C. *L. monocytogenes* was inactivated in the third brand within the same period. The pH of the samples was between 5.6 and 7.3.

During 2003/04, ESR carried out a survey of 307 soft and semi-soft cheeses (around 50 samples were semi-soft blue cheese) for *L. monocytogenes* (Paulin *et al.*, 2015). Cheeses tested included Camembert, ricotta, Brie, mozzarella and a range of blue cheeses. Large and small manufacturers were included. Samples were tested after being stored until the

end of their designated shelf life. No soft cheese samples were positive for *L. monocytogenes*. *L. welshimeri* was detected in one semi-soft blue cheese.

A study completed in 2010 did not detect *L. monocytogenes* in 303 samples of a variety of soft, semi-soft and semi-hard domestic and imported cheeses obtained from retail outlets in New Zealand (Paulin *et al.*, 2015).

Since 2008 there have been two food safety recalls in New Zealand for *L. monocytogenes* in soft cheeses made from pasteurised milk. While no cases of listeriosis were recorded in New Zealand, one recall was associated with products (Brie, Blue and Camembert cheeses) from the same brand linked to an outbreak in Australia (Paulin *et al.*, 2015).

Toxoplasma

T. gondii is not expected to be present in cheese made from pasteurised milk unless it has been introduced with another ingredient. No surveys were located where soft cheeses made from pasteurised milk were tested for the presence of *T. gondii*.

Human illness

Fresh, whey and soft varieties of cheese, in particular, have been associated with foodborne illness, including during pregnancy. *L. monocytogenes* remains the pathogen of primary concern. There have been 13 outbreaks of listeriosis in the United States during the period 1998–2014 associated with soft cheese made from pasteurised milk (Jackson *et al.*, 2018). The majority of outbreaks were associated with Latin-style fresh cheeses. The remaining outbreaks were associated with Middle Eastern- or Eastern European-style cheeses, soft-ripened cheese, or sheep-milk Italian-style cheese.

Outbreaks of listeriosis (which included pregnancy-related cases) linked to consumption of soft cheese produced from pasteurised milk have also been reported in Germany (Koch *et al.*, 2010), Canada (Gaulin *et al.*, 2012), Italy (Amato *et al.*, 2017), Portugal (Magalhaes *et al.*, 2016), Norway (Johnsen *et al.*, 2010), Spain (De Castro *et al.*, 2012) and Australia.[10] Multi-country outbreaks have also occurred (Fretz *et al.*, 2010; Schoder *et al.*, 2012; Wagner *et al.*, 2018).

No outbreaks of listeriosis associated with soft cheeses made from pasteurised milk have been reported in New Zealand. Consumption of feta cheese was the confirmed cause of one sporadic non-perinatal case of listeriosis (Paulin *et al.*, 2015).

Controls

In New Zealand, pasteurisation of milk for cheese-making is still considered a critical control point and the majority of domestically-produced cheese is made with pasteurised milk. Thermised milk cannot be used for making cheeses that will have >39% moisture and pH>5.6 (Paulin *et al.*, 2015). Unless the manufacturer requires it, milk suppliers are not required to test milk supplied for cheese-making for *L. monocytogenes*. Cheese processors in New Zealand must operate under a Risk Management Plan (RMP) and comply with a Product Safety Limit (PSL) of no *L. monocytogenes* in 25 g samples tested throughout the shelf life of the product for those cheeses where *Listeria* may grow (Paulin *et al.*, 2015).

[10] Australia: https://www1.health.gov.au/internet/main/publishing.nsf/Content/cda-cdi3704g.htm (accessed 29 April 2020).

Summary

Present New Zealand Food Safety advice is not consistent with current scientific evidence. Surveys of New Zealand soft, low acid pasteurised cheeses have not detected *L. monocytogenes* but recalls indicate contamination can occur. *L. monocytogenes* can potentially grow in these cheeses and listeriosis outbreaks linked to these foods have been reported in other countries. Within this group of cheeses, there appears to be increased risk associated with Latin-style fresh cheeses. Thus the advice to eat in small quantities immediately after opening the manufacturer's original packaging protects consumers from *L. monocytogenes* contamination that may happen in the home, but not from *L. monocytogenes* that might already be present in the packaged cheese. The advice might be revised to encourage consumers to remove the risk of listeriosis by cooking the cheese first, e.g. "Do not eat unless cooked".

5.2.8 Cheese: Low pH soft pasteurised cheeses

e.g. cottage cheese, cream cheese, crème fraiche, quark, halloumi.

CURRENT ADVICE:	Buy in sealed packs; eat cold or cooked within two days of opening pack

Food

Soft cheeses made from pasteurised milk that have a low final pH (pH 4.6–5). These are unripened (fresh) and white curd cheeses such as cottage, cream cheese, quark and halloumi with a high moisture content (>50%). The shelf life when the product is refrigerated is typically in the range of 60 days (FDA/FSIS, 2003).

Hazards

Listeria

Because these cheeses are made from pasteurised milk, the ability of *L. monocytogenes* to grow when introduced post-production is of more relevance than its potential to grow during manufacture.

The FDA/FSIS (2003) summary of growth data for soft unripened cheese (Appendix C) shows the potential for slow growth of *L. monocytogenes.* The majority of data points (20 of 29) from inoculation studies in cottage or cream cheese showed a slow increase in growth at 4°C or 8°C. In the majority of studies growth was not evident until more than 8 days after inoculation. The lower pH of these cheeses, compared to those discussed above, contributes to less optimal conditions for listerial growth. The average growth rate modelled at 5°C was 0.09 ± 0.286 $\log_{10}$ CFU/g per day.

During the period January 2015 to March 2020, there were no recalls issued in New Zealand for cottage cheese, cream cheese, halloumi or quark.[11]

Toxoplasma

T. gondii is not expected to be present in cheese made from pasteurised milk unless it has been introduced with another ingredient. No surveys were located where soft cheeses made from pasteurised milk were tested for the presence of *T. gondii*.

[11] https://www.mpi.govt.nz/food-safety/food-recalls/recalled-food-products/ (accessed 23 March 2020).

Human illness

No domestic or international outbreaks of listeriosis attributed to low pH soft cheeses were identified.

Controls

Pasteurisation of milk and the low pH of the final cheese product are the primary controls for *L. monocytogenes*.

Summary

Present New Zealand Food Safety advice is consistent with current scientific evidence. The lack of documented outbreaks or product recalls suggest that low pH soft pasteurised cheeses have a lower risk profile than other types of cheeses. However, currently the scientific evidence does not support the recommendation to consume these products within two days of opening, although this is protective. If present, *L. monocytogenes* may survive but grow very slowly in these cheeses. Additional surveys of commercial products for contamination by *L. monocytogenes* would provide added consumer confidence, and studies of *L. monocytogenes* growth up until the "use by" or "best before" date on the packaging would underpin advice on the number of days the product should be safe if consumed cold. If the product is to be consumed cooked there is no need to apply a two-day limit on the basis of risk from *L. monocytogenes*.

5.2.9 Cheese: Hard cheeses

(e.g. Cheddar, Parmesan, Colby, Gouda, Edam, processed cheese)

CURRENT ADVICE:	OK to eat, store in fridge

Food

Semi-hard, hard and very hard (low moisture) cheeses made from pasteurised milk. Hard varieties of cheese are the types most consumed in New Zealand (Paulin *et al.*, 2015).

Hazards

Listeria

Because these cheeses are made from pasteurised milk, the ability of *L. monocytogenes* to grow when introduced post-production is of more relevance than its potential to grow during manufacture.

The FDA/FSIS (2003) information on *L. monocytogenes* shows a decline in population in hard cheeses and all processed cheeses (see Appendix C). In the studies, *L. monocytogenes* was either inoculated into the milk prior to making the cheese, or inoculated on finished product. Potential growth of *L. monocytogenes* was then followed during incubation, predominantly at 4°C. In all cases the cheese making process and/or storage resulted in a decline in *L. monocytogenes* levels. For both hard and processed cheese the decrease in numbers expected at 5°C is -0.05 $\log_{10}$ CFU/g per day.

Surveys of hard cheese in other countries suggest that the prevalence of *L. monocytogenes* is low (≤0.3%) (Lake *et al.*, 2005a; Paulin *et al.*, 2015). *L. monocytogenes* was not detected in 300 samples of New Zealand retail packed, grated, low moisture cheeses (Whyte and Wong, 2002).

Toxoplasma

T. gondii is not expected to be present in cheese made from pasteurised milk unless it has been introduced with another ingredient. No surveys were located where hard cheeses made from pasteurised milk were tested for the presence of *T. gondii*.

Human illness

Hard cheese is less often associated with outbreaks of listeriosis compared to other types of cheeses. A 2011 outbreak of invasive listeriosis among hospitalised patients across Belgium was attributed to a hard cheese made from pasteurised milk (Yde *et al.*, 2012). While cheese sampled at the manufacturing plant yielded isolates identical to the outbreak strain, the cause of the contamination was not revealed.

An outbreak of salmonellosis occurred in Northern England during 1997 but was caused by failed pasteurisation during the production of Cheddar cheese (Donnelly, 2004).

Controls

Pasteurisation of milk and the low water activity of hard cheese are the primary controls.

Summary

Present New Zealand Food Safety advice is consistent with current scientific evidence. *L. monocytogenes* is not expected to occur in pasteurised, low moisture cheeses unless environmental contamination occurs during manufacture or ripening, or there is a failure in pasteurisation conditions. Growth is not expected and concentrations will decrease over time.

5.2.10 Custard: Ready-made chilled (packaged)

CURRENT ADVICE:	Eat within two days of opening

Food

Commercially prepared ready-made chilled custard is a pasteurised product containing milk, cornflour, sugar and flavouring.

Hazards

No surveys were located where custard was tested for the presence of *L. monocytogenes* or *T. gondii*. No data for growth of *L. monocytogenes* in ready-made custard was located. *T. gondii* is not expected to be present in pasteurised custard.

Human illness

No domestic or international outbreaks of listeriosis attributed to custard consumption have be identified.

Gilbert *et al.* (2010) listed two Australian recalls for custard potentially contaminated with *Listeria*. No additional recalls were identified.

Controls

Pasteurisation is the primary control. The products are stored under refrigeration.

Summary

The current evidence is not sufficient to evaluate New Zealand Food Safety advice. There are no data for the growth of *L. monocytogenes* in ready-made custard but it should be assumed that growth is possible. As indicated by recalls (noting that these are infrequent and there are no survey data), *L. monocytogenes* can occasionally be present in the final product. *L. monocytogenes* could also be introduced once the package is opened in the home. The current advice manages the potential for contamination once the package is opened but does not address the potential for contamination prior to this. A survey of New Zealand custard, including enumeration and supported by growth studies, would provide a better set of evidence to develop advice.

5.2.11 Custard: Home-made

CURRENT ADVICE:	Eat hot immediately after cooking; reheat leftovers until piping hot (over 70ºC) and eat immediately

Food

Home-made custard can be a sauce prepared from milk and commercially produced custard powder or a starchless baked or steamed egg and milk dessert.

The sauce prepared from the starch-based custard powder requires heating to near boiling in order to thicken. Baked or steamed (stirred) egg custards do not use starch as a thickening agent. These desserts are thickened by gradual heating until the “setting point” of approximately 70°C is reached. Curdling or separation occurs if rapid or excessive heat is applied (>80°C). The cooking process is largely uncontrolled (unless thermometers are used) and physical characteristics are relied on to indicate that the custard has been adequately cooked. Baked or steamed egg custards are unsuitable for reheating as the components will separate during a thorough reheating process.

Hazards

L. monocytogenes and *T. gondii* would be inactivated at cooking temperatures used for home-made custards. There are no studies measuring growth of *L. monocytogenes* in home-made custard, and no survey data. *T. gondii* is not expected to be present in custard made from pasteurised milk.

Human illness

No outbreak reports were located.

Controls

It is likely that pasteurisation conditions are achieved during cooking.

Summary

The current evidence is not sufficient to evaluate New Zealand Food Safety advice. However, adequately cooked home-made custard should be safe to eat immediately after preparation. The advice on leftovers is in keeping with Section 5.7.1, however there are no studies of *L. monocytogenes* growth in custard and should post-cooking contamination occur, growth would be expected. Growth data would provide support for advice over whether custard should be discarded if not eaten immediately.

5.2.12 Ice cream: Packaged

CURRENT ADVICE:	OK to eat

Food

Ice cream has been defined in the Australia New Zealand Food Standards Code as:[12]

> "a sweet frozen food made from cream or milk products or both, and other foods, and is generally aerated….
>
> It must contain no less than: (a) 100 g/kg of milk fat; and (b) 168 g/litre of food solids."

Ice cream is made from milk fat, non-fat milk solids, sugar, and emulsifiers/stabilisers. Stabilisers are polysaccharides used to bind free water and retard ice crystal growth during freezing and storage. Emulsifiers are incorporated to stabilise fat droplets and in some ice creams, eggs are used for this purpose (Lake *et al.*, 2009). Gelato is a variety of ice cream that has a different consistency owing to modifications in the relative quantities of the ingredients and a slower churning process.

Hazards

Listeria

An updated risk profile of *L. monocytogenes* in New Zealand ice cream was published in 2009 (Lake *et al.*, 2009). Prevalence data available for retail ice cream in New Zealand suggests that *Listeria* contamination in both imported and domestic product is rare.

Several studies have investigated the ability of *L. monocytogenes* to survive and grow in ice cream under chilling and freezing conditions (Gougouli *et al.*, 2008). Any *Listeria* present at the time of freezing may survive and be recovered after fourteen weeks of storage at -18°C (Palumbo and Williams, 1991). FDA/FSIS (2003) information for *L. monocytogenes* shows no growth in frozen ice cream (Appendix C).

The addition of flavouring ingredients after pasteurisation might introduce *Listeria* but these bacteria will not grow during frozen storage.

In May 2020, ice cream produced by a USA company was recalled due to the potential for contamination with *L. monocytogenes*, as indicated through routine company product testing. The cause of the contamination had not been established at the time of the recall.[13]

Toxoplasma

T. gondii is not expected to be present in packaged ice cream made from pasteurised milk unless it has been introduced with another ingredient. No surveys were located where ice cream was tested for the presence of *T. gondii*. Tachyzoites are likely to be susceptible to freezing but no studies were located where this was examined.

Human illness

In New Zealand, there have been no listeriosis outbreaks where ice cream was the confirmed vehicle of infection.

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[12] Standard 2.5.6, available at https://www.legislation.gov.au/Series/F2015L00424 (accessed 28 May 2020).

[13] https://www.fda.gov/safety/recalls-market-withdrawals-safety-alerts/ramar-foods-recalls-mint-chocolate-chip-hidden-spinach-ice-cream-because-possible-health-risk (accessed 18 May 2020).

A USA multistate listeriosis outbreak resulted in 10 cases and 3 deaths that were linked over a period of five years and involved products from two ice cream plants serving a single brand.[14] Environmental samples from the plants yielded *L. monocytogenes* strains that were genetically linked to isolates from cases. The ice cream was manufactured under unsanitary conditions.[15] In a separate investigation, whole genome sequencing of environmental samples from an ice cream facility in Florida were linked to 3 sporadic cases of listeriosis that had occurred in the past and not been attributed to a common source (Allard *et al.*, 2019).

An outbreak of toxoplasmosis in Brazil was linked to ice cream but the cause was the use of contaminated water as an ingredient (de Moura *et al.*, 2006).

Controls

Ice cream produced for domestic supply must undergo a heat treatment to allow for effective microbial control in the presence of potentially protective ingredients (fats, sugars, emulsifiers etc.) (NZFSA, 2008; 2009). This heat treatment will be different to typical pasteurisation.

Summary

Present New Zealand Food Safety advice is consistent with current scientific evidence. If *L. monocytogenes* is present it will not grow during frozen storage. This is a widely consumed food and no outbreaks have been reported in New Zealand associated with this food. Outbreaks in other countries were associated with unusual circumstances.

5.2.13 Ice cream: Soft serve

CURRENT ADVICE:	Don't eat

Food

Soft serve ice cream contains essentially the same ingredients as hard ice cream. The manufacturing process is also similar except for the exclusion of a hardening step. Soft serve ice cream mix is usually drawn from the freezer at about -6°C to -7°C and packed immediately after this initial freezing stage (NZICMA, 2012). The product is stored at refrigeration temperature (<5°C) and only frozen at the retail point of sale. Soft serve ice cream produced at the point-of-sale can also be made using a rehydrated powder or a UHT liquid mix.[16]

Hazards

Listeria

Listeria could contaminate soft serve ice cream mix during production. Between production and consumption, there is a greater potential for slow growth of *L. monocytogenes* in soft serve ice cream mix under refrigerated conditions (<5°C) than in hard ice cream (Lake *et al.*, 2009). The pH of soft serve ice cream is close to neutral which can support the growth of *L. monocytogenes* if present (Nichols and de Louvois, 1995). A study in soft serve ice cream (3% fat) also found that *L. monocytogenes* was able to survive at -18°C for three months but

[14] https://www.cdc.gov/listeria/outbreaks/ice-cream-03-15/index.html (accessed 5 May 2020).
[15] https://www.foodsafetymagazine.com/news/19-million-in-fines-for-blue-bell-former-president-also-charged-with-concealing-listeria-contamination/ (accessed 6 May 2020).
[16] http://www.dairyproductsnz.co.nz/our_ingredient/white-gold/ (accessed 13 May 2020).

did not grow (Dean and Zottola, 1996). Machines that dispense soft serve ice cream can also be a source of *L. monocytogenes* contamination (Little and Louvois, 1999).

International surveys of the microbiological quality of soft serve ice cream show that it is more likely to be of poor microbiological quality than hard frozen product (Little and Louvois, 1999). A survey of ice cream, including soft serve products and other edible ices in the UK, detected *L. monocytogenes* in 0.2% of soft serve ice cream samples (n = 964) (Nichols and de Louvois, 1995). Soft serve ice cream was included in a survey of products from ice cream parlours in the Australian Capital Territory (Waters and Krsteski, 2013). Seven soft serve samples were tested and none were found to contain *L. monocytogenes*.

Toxoplasma

T. gondii is not expected to be present in soft serve ice cream made from pasteurised milk unless it has been introduced with another ingredient. No surveys were located where soft serve ice cream was tested for the presence of *T. gondii*. Tachyzoites are likely to be susceptible to freezing but no studies were located where this was examined.

Human illness

There are no New Zealand human illness data indicating problems with soft serve ice cream.

Overseas, a listeriosis outbreak in a hospital was caused by the use of a pasteurised, commercially-produced liquid ice cream mix to make milkshakes (Rietberg *et al.*, 2016). The ice cream mix was the source of contamination and *L. monocytogenes* persisted in the milkshake equipment, even after cleaning. An outbreak of toxoplasmosis in Brazil was linked to ice cream but this was caused by the use of contaminated water as an ingredient (de Moura *et al.*, 2006). The report is not clear whether this ice cream was soft serve.

Controls

See packaged ice cream (Section 5.2.12).

Summary

Present New Zealand Food Safety advice is not consistent with current scientific evidence. While soft serve ice cream is a higher risk compared to packaged ice cream (which is kept frozen once produced) there is little evidence from public health surveillance and overseas surveys that this food poses a significant risk of listeriosis. However, *L. monocytogenes* can become established in point-of-sale dispensing machines and slough off into product passing through. A survey of New Zealand soft serve ice cream, including *L. monocytogenes* enumeration, would provide a better set of evidence to develop advice. In the interim, the current advice is protective.

5.2.14 Yoghurt: Unpasteurised (made from raw milk)

CURRENT ADVICE:	Don't eat

Food

Yoghurt is fermented milk. The Australia New Zealand Food Standards Code defines yoghurt as "fermented milk where the fermentation has been carried out with lactic acid producing microorganisms".[17] The pH must not be higher than 4.5 and the microorganisms

[17] Standard 2.5.3, available at https://www.legislation.gov.au/Series/F2015L00413 (accessed 28 May 2020).

used in the fermentation must remain viable in the product at a concentration greater than 10^6 per gram. Yoghurt may contain other foods (e.g. fruit flavouring). In yoghurt made with raw milk, the milk is not pasteurised before fermentation commences.

Hazards

Listeria

Data on the prevalence of *L. monocytogenes* in yoghurt made from unpasteurised milk are scarce. *L. monocytogenes* was not detected in non-pasteurised yoghurt sampled in Iran and Turkey (Aygun and Pehlivanlar, 2006; Shamloo *et al.*, 2014). *L. monocytogenes* was also not detected in yoghurt made from raw milk sampled in the UK (Kerr *et al.*, 1992).[18] *L. monocytogenes* can be present in raw milk (Section 5.2.1). It is possible that *L. monocytogenes* are inactivated during yoghurt production by the fermentation process and reduced pH, as has been observed in raw milk cheeses (Section 5.2.6), but studies are required to confirm this. In finished product, based on studies of yoghurt made from pasteurised milk, *L. monocytogenes* survival for >1 week could be possible (Section 5.2.15).

Toxoplasma

T. gondii does not replicate outside the host so will not grow in food. If present in dairy products, *T. gondii* is most likely to be in the tachyzoite stage. It is possible that cysts could be present but this requires further study (Dubey *et al.*, 2014).

T. gondii could be present in yoghurt made from raw milk but no surveys were located where yoghurt was tested for the presence of *T. gondii*. A study of cheeses made from raw sheep milk naturally contaminated with viable *T. gondii* tachyzoites suggested that lactic acid bacteria multiplication and subsequent acidification might kill tachyzoites (Ranucci *et al.*, 2020).

Human illness

No reports of illness associated with yoghurt made using raw milk were located.

Controls

The low pH will make conditions less favourable for *L. monocytogenes*.

Summary

The current evidence is not sufficient to evaluate New Zealand Food Safety advice. Studies of *L. monocytogenes* prevalence and behaviour in yoghurt made from raw milk are needed. However, given the potential for *L. monocytogenes* to be present in raw milk and for this pathogen to survive (but not grow) in finished yoghurt, the current advice is protective until additional data are available.

[18] 100 samples were tested, including those made from pasteurised milk. The number of samples made from raw milk was not reported separately.

5.2.15 Yoghurt: Pasteurised

CURRENT ADVICE:	Eat within two days of opening

Food

See unpasteurised yoghurt. In yoghurt made with pasteurised milk, the milk is pasteurised before cultures are added, and fermentation commences.

Hazards

Listeria

Given this food is made from pasteurised milk, the concern relates to post-production recontamination and the behaviour of *L. monocytogenes* in the finished product. The combination of low pH, presence of lactic acid and live lactic acid bacteria generate an unfavourable environment for pathogenic organisms, including *L. monocytogenes*.

The FDA/FSIS (2003) growth and survival information shown in Appendix C records a decline in population for all three papers cited. One of these studies showed survival of *L. monocytogenes* up to 24 days but the greatest decrease in numbers in all three studies occurred within 12 days. A later review of challenge studies also showed that the concentration of *L. monocytogenes* decreases in yoghurt during storage, but some bacteria were still detected weeks after initial inoculation (Aryana and Olson, 2017). Survival was enhanced at cooler temperatures. For example, the concentration of *L. monocytogenes* decreased during 16 days of storage after inoculation into a commercially produced yoghurt, with a D-value of 244 h for *L. monocytogenes* in yoghurt stored at 6ºC, and 87 h when stored at 15ºC (Szczawinski *et al.*, 2016).

Data on the prevalence of *L. monocytogenes* in yoghurt made from pasteurised milk are scarce.[19] *L. monocytogenes* was not detected in yoghurt made from pasteurised milk sampled in the UK (Kerr *et al.*, 1992).[20] The pH of these yoghurts was in the range 3.6–4.6.

Toxoplasma

T. gondii is not expected to be present in yoghurt made from pasteurised milk unless it has been introduced with another ingredient. No surveys were located where yoghurt was tested for the presence of *T. gondii*.

Human illness

Overseas, yoghurt has been the cause of occasional outbreaks, including STEC infection and salmonellosis, but the available information shows that improper processing and post-production contamination were causes (Aryana and Olson, 2017).

Two outbreaks of staphylococcal food poisoning associated with yoghurt have been reported in New Zealand (ESR, 2001). The product was prepared in institutional kitchens using domestic yoghurt making machines. Contamination by food handlers and slow growth of the starter culture due to incorrect temperature contributed to the outbreaks.

No records of listeriosis outbreaks were located.

[19] Some available reports do not specify the pasteurisation status of the milk used for making the yoghurt so have not been considered further.
[20] 100 samples were tested, including those made from raw milk. The number of samples made from pasteurised milk was not reported separately.

Controls

The use of pasteurised milk, plus the low pH and presence of live, acid-producing bacteria in the yoghurt, combine to control pathogenic bacteria.

Summary

Present New Zealand Food Safety advice is not consistent with current scientific evidence. There is no evidence from public health data that *L. monocytogenes* is a problem in this widely-consumed food. If present (e.g. if introduced after the package is opened), *L. monocytogenes* will not grow, although it can survive. Unless some factor occurs which causes the pH to rise after opening (e.g. mould contamination and growth), the product should remain stable during storage.

Given that *L. monocytogenes*, if present, is unlikely to grow in yoghurt made from pasteurised milk, the present New Zealand Food Safety advice "eat within two days of opening" is not supported. This advice might be altered to refer consumers to the manufacturer's instructions, which usually advise keeping the product refrigerated and eating it within seven days of opening or before the best before date.

Yoghurt made in the home from pasteurised milk or commercial premixes may not have the same low level of risk as commercially prepared product. These products require additional study to determine whether separate advice is needed.

5.3 EGGS

Salmonella spp. is the predominant pathogen associated with eggs. A Risk Profile is available that provides a New Zealand perspective on non-typhoidal salmonellae in eggs (Rivas and King, 2016).

5.3.1 Raw eggs

In egg flips, eggnog, smoothies, home-made mayonnaise and dressings, home-made ice cream, mousse and tiramisu, etc.

CURRENT ADVICE:	Don't eat

Food

Most eggs for human consumption are derived from chickens, but eggs from other birds such as ostriches, ducks and quail are also consumed. The majority of eggs are marketed and consumed as shell eggs, and this form is the focus of this section. Raw egg contents are also available, either mixed or separated into whites and yolks, which are largely used by food manufacturers and in food service operations. These might be processed by pasteurisation or drying, and could be mixed with other ingredients.

Hazards

Egg contents are moist (approximately 64% water) and contain the nutrients required for bacterial growth.

Salmonella

Internationally, the major cause of egg-related outbreaks is internal contamination of eggs with specific egg shell-invasive *Salmonella* strains (predominantly *S.* Enteritidis phage type 4, plus a few other *S*. Enteritidis phage types, and *Salmonella* Heidelberg). These shell-

invasive strains have not been detected in New Zealand (Kingsbury, 2018; Rivas and King, 2016).

Surveys in New Zealand have not found any evidence of internal contamination of locally produced eggs (Kingsbury, 2018). Other serotypes have been detected on the external surface of shells; *S*. Thompson or *S*. Infantis were isolated from 13/93 samples in 2001 and *S*. Infantis was isolated from 9/514 samples in 2005. The 2005 survey showed concentrations to be low, with eight samples harbouring <5 MPN/egg and the other positive sample carrying 44 MPN/egg (Wilson, 2007).

There is a data gap regarding the prevalence of *Salmonella* spp. on eggs from other bird species such as ducks, quail and geese.

Salmonella spp. will not grow when on a dry, clean egg shell, and the concentration will usually slowly decrease over time (Kingsbury, 2019). The conditions may be suitable for growth if salmonellae are transferred from the egg shell into the raw egg contents or food containing raw egg. Faecal contamination of the egg shell improves survival of *Salmonella* spp. (Kingsbury, 2019).

Listeria

L. monocytogenes has not been isolated from the contents of intact eggs but has been found on egg shells and in the environment of laying hens (Rivoal *et al.*, 2010). It has also been found as an environmental contaminant in raw and pasteurised liquid egg (FDA/FSIS, 2003).

Toxoplasma

Eggs are not considered to be a risk for toxoplasmosis. A review of laboratory studies, where layers were experimentally infected with varied doses of *T. gondii* tachyzoites or cysts, found the raw hen eggs were unlikely to be a source of infection for humans (Dubey, 2010).

However, one survey has detected *T. gondii* DNA in egg contents from domestic and commercially produced eggs (Khademi *et al.*, 2018). Further studies are needed to confirm this finding, and to determine whether infectious *T. gondii* are present.

When tested by bioassay, viable *T. gondii* were detected in the yolk of raw chicken eggs inoculated with tachyzoites and kept at room temperature for three weeks (Kunert and Werner, 1963). Survival extended to four weeks when the yolks were kept at 4ºC. The survival times were shorter in raw egg whites (1 week at room temperature and 2 weeks at 4ºC).

Human illness

There is a very low reported incidence of egg-associated salmonellosis in New Zealand (Kingsbury, 2019). In 2015, a risk assessment summarised surveillance data for the period 2000–2009 and found eggs were implicated in 3 of 204 salmonellosis outbreaks (MPI, 2015). More recently, data were reviewed for the period 2010–2016 and four outbreaks were associated with eggs as a potential vehicle (Kingsbury, 2019). However, in three of these outbreaks the evidence was weak, and in the fourth, eggs were mixed with other ingredients that could also have been the source of *Salmonella* contamination.

Internationally, a significant proportion of foodborne nontyphoidal salmonellosis infections have been attributed to the consumption of contaminated eggs (Kingsbury, 2019). For example, in Australia there were 351 salmonellosis outbreaks reported during the period 2009–2014, of which eggs were confirmed or suspected in 45% (Kingsbury, 2019). *S.*

Enteritidis had not been detected in Australian poultry or eggs during that period. In the European Union there were 296 foodborne salmonellosis outbreaks reported during 2018 where there was strong evidence linking cases to foods (EFSA/ECDC, 2019). Of these, 135 (46%) were linked to eggs and egg products and 84/135 egg-associated outbreaks were caused by *S.* Enteritidis.

The consumption of duck eggs from a single supplier in the United Kingdom has been linked to a widely-dispersed outbreak of infection by *S.* Typhimurium DT8, and *S.* Enteritidis cases have also been reported associated with this food (ACMSF, 2016). Sporadic cases of *S.* Enteritidis infection linked to imported eggs from quail have also been reported in Europe (ACMSF, 2016).

Apart from one small, two-case, episode, there have been no reported outbreaks of foodborne listeriosis due to the consumption of raw eggs (FDA/FSIS, 2003).

Controls

The production of raw shell eggs might involve activities intended to reduce the risk of *Salmonella* spp. being present on the final product but there are no sterilisation steps (Rivas and King, 2016).

Summary

Present New Zealand Food Safety advice is consistent with current scientific evidence. *Salmonella* spp. (and potentially *L. monocytogenes*) can be present on the external surface of raw shell eggs. It is possible that consumers could become ill through handling raw eggs (through hand-mouth transfer of microbes), but it is more likely that illness will arise through consuming foods made with raw egg, especially if these foods support microbial growth. Overseas, outbreaks have occurred linked to consumption of raw eggs or foods containing raw eggs.

5.3.2 Cooked eggs

Fried, scrambled, baked, poached, etc.

CURRENT ADVICE:	Cook well (firm yolks, firm scrambled eggs)

Food

See raw eggs (Section 5.3.1).

Hazards

Salmonella

See raw eggs (Section 5.3.1). *Salmonella* spp. can be present on the shell of raw eggs but there is currently no evidence of these bacteria being present in the contents of intact eggs in New Zealand.

Experiments have shown that hard boiling or soft boiling eggs, or poaching eggs until the yolk thickened were effective for inactivation of a *Salmonella* inoculum (Davis *et al.*, 2008). Scrambling eggs until no visible liquid remained, frying eggs “over easy” (cooking on both sides) or lightly poaching eggs was less effective. Frying eggs “sunny side up” was not effective. Microwave cooking was also effective, but only if the eggs were cooked until the yolks were firm (Bates and Spencer, 1995). An evaluation of 226 egg recipes (e.g. quiche, custard, omelette) found that the recipes often used time or visual cues as a measure of

doneness rather than a recommended cooking temperature (71ºC) (Godwin *et al.*, 2016). Upon testing three recipes, a lemon pie and quiche were both found to exceed 71ºC when instructions were followed, but a "breakfast casserole" (meat, dairy, bread and egg dish) did not. However, this latter dish did not appear cooked ("set") and the researchers concluded that experienced consumers were likely to cook it longer.

Listeria

L. monocytogenes will be killed during egg cooking, but will grow in cooked egg products if re-introduced after cooking. This has been demonstrated that when *L. monocytogenes* was inoculated into separated white and yolk, and mashed white and yolk combined, from hard-boiled eggs and stored at 4°C, 8°C or 12°C (Claire *et al.*, 2004). Counts increased from 10^2 to >10^6 CFU/g in 3–20 days, and the levels of were dependent on the storage temperature and the atmosphere of the packaging atmosphere.

Toxoplasma

When tested by bioassay, *T. gondii* tachyzoites injected into the yolk or white (albumin) of intact eggs remained viable after 5 minutes of boiling, but not after 6 or 7 minutes (Kunert and Werner, 1963). *T. gondii* were also able to survive in the yolk when the egg was fried for 3 minutes.

Human illness

In 2019, it was reported that 47 people contracted salmonellosis from consumption of temperature-abused soft-scrambled eggs linked to dishes in a Hong Kong restaurant.[21] In a similar incident in 2017, 22 people were infected with *Salmonella* spp. after eating scrambled egg in fusilli or noodle dishes at a restaurant. In both these incidents, semi-cooked egg was prepared and held for a prolonged period before being added to dishes without reheating.

A USA multistate outbreak of listeriosis starting in 2019 was caused by environmental contamination of bulk, peeled hard boiled eggs from a single commercial supplier.[22] The outbreak involved eight cases, five of whom required hospitalisation and there was one death. One was a perinatal case, infected while the mother was pregnant.

Controls

Cooking all parts of the egg thoroughly, and all foods containing raw eggs thoroughly, will kill pathogenic microorganisms. This means the white and yolk should be firm, not runny, or the egg dish should have a firm texture.

Summary

Present New Zealand Food Safety advice is consistent with current scientific evidence. There are no issues identified for thoroughly cooked eggs eaten immediately after cooking. However, additional information might be added to advise consumers to cook egg dishes (e.g. quiche, omelette) until all parts are firm.

[21] https://www.foodsafetynews.com/2019/08/storage-and-cooking-temperature-at-fault-for-salmonella-outbreaks/ (accessed 21 April 2020).
[22] https://www.cdc.gov/listeria/outbreaks/eggs-12-19/index.html (accessed 12 May 2020).

5.4 MEAT AND POULTRY

The New Zealand Food Safety advice relates to "any raw meat, raw chicken or other poultry, beef, pork and similar" (New Zealand Food Safety, 2020). It is unclear whether this description is intending to include edible offals. For the current assessment, the food descriptors have been extended to include edible offal.

5.4.1 Raw meat and edible offal

Any raw meat (raw chicken or other poultry, beef, pork, etc.) or raw offal (e.g. heart, liver, kidneys, etc.)

CURRENT ADVICE:	Don't eat or taste; don't touch face, mouth or eyes while preparing; wash and dry hands well after touching raw meat

Food

Meat is skeletal muscle tissue and any attached animal rind, fat, connective tissue, nerve, blood and blood vessels (Australia New Zealand Food Standard Code, Standard 2.2.1).[23] Edible offals are other edible parts of the animal, usually organs (e.g. liver, kidney). The main commercial meat species in New Zealand are cattle, sheep, pigs and chickens. Meat and edible offals from other types of poultry (e.g. ducks) and mammals (e.g. deer) are also available.

Animal tissue can become contaminated with pathogens and spoilage organisms during primary processing operations including skinning and evisceration (ICMSF, 2005). Animal faecal material is a particularly rich source of pathogenic bacteria, viruses and protozoa (Moriarty *et al.*, 2008). In addition, animal tissues might already contain microbes in the form of encysted parasites, or bacteria or viruses as a result of a systemic infection (e.g. hepatitis E virus). Raw meat is a suitable environment for microbial growth.

Hazards

Salmonella spp., *Campylobacter* spp. and STEC have all been detected on raw meat sampled in New Zealand (Wong *et al.*, 2005b; Wong *et al.*, 2007a; Wong *et al.*, 2007b). The concentrations were generally low (<10 MPN/g).

Listeria

There have been surveys for *Listeria* spp. in New Zealand raw meat and meat processing environments, but these did not measure *L. monocytogenes* concentrations (Gilbert *et al.*, 2009). A survey in two North Island abattoirs of 100 bovine and 100 ovine carcass swabs taken immediately after dressing found *Listeria* species (*L. innocua* and *L. ivanovii*) on two ovine samples (Hudson and Mott, 1994). A South Island mutton slaughterhouse survey found *L. monocytogenes* in 7/218 (3.2%) samples collected from ovine carcasses, processing plant, and the environment (Pociecha *et al.*, 1991). *Listeria* were not found on freshly dressed carcasses (n=111 samples) or from meat contact surfaces in the processing area (n=45), however four samples (4/31, 13%) from the chilling rooms were positive. The remaining three positive samples came from environmental samples (n=31) collected from external areas of the plant (drain effluent, soil, fodder). An earlier survey, published in 1998, examined 142 samples from New Zealand cattle and sheep slaughter plants, including whole carcasses, cuts of boned meat, offal, pelts, hides, viscera, equipment, work surfaces

[23] https://www.legislation.gov.au/Series/F2015L00427 (accessed 29 April 2020).

and effluent (Lowry and Tiong, 1988). Seventy-five retail display samples of beef mince, pork cuts and poultry portions were also tested. *L. monocytogenes* were not detected in any viscera or offal samples but was present in meat samples (20–60%), equipment and work surfaces (27–73%) and hide/pelt samples (17–43%). This pathogen was also detected in 48, 68 and 92% of retail samples of poultry portions, cuts of pork and beef mince respectively.

Toxoplasma

T. gondii does not replicate outside the host so will not grow in food. If present in meat products, *T. gondii* is most likely to be in the cyst (bradyzoite) stage. *T. gondii* cysts can be present in raw meat and edible offal from pigs, sheep, goats, poultry, cattle and horses (Opsteegh *et al.*, 2019; Stelzer *et al.*, 2019). Meat from free range animals has a higher risk of containing *T. gondii* cysts because these animals are exposed to pasture and soil where oocysts can be present (Jones and Dubey, 2012).

Seroprevalence studies show that deer, goats and sheep in New Zealand can be exposed to this parasite (summarised in King *et al.* 2017). When a variety of tissue samples from individual sheep in a New Zealand herd were tested by mouse bioassay, viable *T. gondii* were detected in at least one sample from 23/34 (68%) animals (Jacobs and Moyle, 1963). Viable *T. gondii* were detected in the diaphragms from 18 animals and the psoas from 15 animals, demonstrating the presence of cysts in muscle tissue. In addition, diaphragms from 80 cattle were tested by bioassay but viable *T. gondii* were not detected. There is no information on the prevalence of *T. gondii* cysts in meat and meat products at retail in New Zealand but a survey of retail New Zealand lamb imported into Canada found *T. gondii* DNA in 26% (n=53) of samples (Lafrance-Girard *et al.*, 2018). The lamb had been frozen so the cysts were probably not viable in these samples.

The presence of *T. gondii* cysts in raw meat has been demonstrated in studies in other countries (summarised in King *et al.* 2017). For example:

- A survey of fresh lamb mince sampled at retail in Adelaide, Australia, over a six month period detected *T. gondii* DNA in 34/79 (43%) samples (Dawson *et al.*, 2020). Each sample was tested in quadruplicate and for these 34 samples, 3 or 4 of the quadruplicate samples were positive. In a further 20 samples, *T. gondii* DNA was detected in 2/4 tests. This imparts and overall prevalence of 54/79 (68%, 95% CI 57%-78%). Lamb is often served rare in Australia.
- Testing by mouse bioassay detected a low prevalence of viable *T. gondii* in samples of retail lamb (2/750) and pork (1/750) in the USA (Dubey *et al.*, 2020).
- A survey of retail fresh meat in Canada detected a low prevalence of *T. gondii* DNA in ground beef (2% positive, 2/93), ground pork (3% positive, 3/94) and 9/234 (4%) of individual chicken breasts (Iqbal *et al.*, 2018). Another survey, using a different approach, also found similarly low DNA prevalence for beef (0.6% positive, 2/329) and pork (1.1% positive, 1/87) (Lafrance-Girard *et al.*, 2018). The prevalence for lamb was higher (25/59, 42%). Ground meats were more likely to be positive.
- Mouse bioassay tests confirmed that viable *T. gondii* can be found in heart, brain and muscle tissue from free-range chickens (Dubey, 2010). Overall, the prevalence of viable *T. gondii* in leg and breast muscle was 44% (15/34) and 19% (16/86), respectively.
- Viable *T. gondii* was not detected using cat and mouse bioassays on 2,094 beef or 2,094 chicken samples from retail stores in the USA (Dubey *et al.*, 2005). The prevalence for

pork samples was low, with 7/349 pooled samples containing viable *T. gondii*, where one pooled sample contained meat from six samples. Individual testing was only possible for 12 meat samples from two pools, and from these, three were positive. The overall prevalence in pork was calculated to be 0.38%.

Viable *T. gondii* have also been detected by bioassay in pig and sheep hearts (Djokic *et al.*, 2016; Yang *et al.*, 2017).

Human illness

Several outbreaks of toxoplasmosis have been reported overseas where consumption of raw or rare red meat products was implicated through epidemiological evidence (Smith, 1993). For example, three cases of toxoplasmosis in France, two being perinatal cases, were most likely caused by eating raw horse meat (Pomares *et al.*, 2011). See also cooked meats (Section 5.4.2).

Other pathogens have also caused outbreaks of foodborne infection linked to consumption of raw (or rare) meat or offal, e.g. pathogenic *E. coli*, *C. jejuni*, *Salmonella* Dublin and hepatitis A virus (the latter was linked to an infected food handler in a meat distribution plant) (Braeye *et al.*, 2014; Robesyn *et al.*, 2009; Tompkins *et al.*, 2013).[24]

Controls

Following basic hygiene rules when handling raw meat or edible offal is the best approach to avoid cross-contamination or accidental ingestion of pathogens. This includes using separate cutting boards and knives when handling raw meat, ensuring any surfaces, containers or utensils that have been in contact with the raw meat are washed thoroughly with hot, soapy water, and ensuring good hand hygiene (avoid touching the face, mouth and eyes, wash hands after handling raw meat). Poultry should not be washed prior to cooking because this can contaminate the sink and food preparation areas, which may result in transfer of pathogens to other foods (Cates *et al.*, 2019).

Advice regarding not eating or tasting raw meat is entirely appropriate (for any consumer) given the potential microbial hazards.

The viability of *T. gondii* cysts also reduces when raw meat is frozen. Studies of this effect have been summarised and these show a large variation in time/temperature regimes, although generally, cooler temperatures shortened the time to inactivation (De Berardinis *et al.*, 2017; Guo *et al.*, 2015). A recent review of freezing as an inactivation method for *T. gondii* concluded "*Toxoplasma gondii* in pork, mutton, and other meat is completely inactivated by freezing at between −7 and −13°C for 2–4 days" (Franssen *et al.*, 2019). However, freezing should not be relied upon as a sterilisation process, particularly since adequate freezing temperatures might not be achievable in domestic freezers. See also Section 3.2.1.

Summary

Present New Zealand Food Safety advice is consistent with current scientific evidence. Additional advice might be added regarding cleaning work surfaces and utensils, and not washing raw poultry.

[24] https://www.cdc.gov/salmonella/dublin-11-19/index.html (accessed 20 May 2020).

5.4.2 Cooked meat and edible offal

Cooked beef, pork, chicken, mince, sausages, liver, etc.

CURRENT ADVICE:	Cook until piping hot throughout, and until juices run clear (use a meat thermometer to check temperatures); eat while hot; never eat rare or undercooked meats; store leftovers covered in fridge and eat within two days; reheat leftovers until piping hot (over 70ºC)

Food

See raw meat and edible offal (Section 5.4.1). Cooking is where the meat or offal is heated in some way (fried, boiled, roasted, etc.).

Hazards

Listeria

See raw meat and edible offal (Section 5.4.1). *L. monocytogenes* can potentially grow in refrigerated cooked meats (Appendix C). Generation times range from an average of 8.8 to 20.6 hours following incubation at 10°C and 5°C respectively. The kinetics of *L. monocytogenes* growth were similar for cooked meat under aerobic and vacuum packaging conditions (Hudson and Mott, 1993a).

Toxoplasma

T. gondii cysts form inside the muscle and organs of meat producing animals. Humans can be infected when they consume meat or edible offal raw or undercooked. *T. gondii* does not replicate outside the host so will not grow in food. Data on the presence of *T. gondii* in cooked meat arises through investigations of outbreaks or experiments looking at the effect of thermal treatment on this parasite (see below). No surveys were located.

Human illness

There are no reports of listeriosis outbreaks or toxoplasmosis outbreaks in New Zealand linked to cooked meat or offal. Listeriosis has occurred from consumption of processed ready-to-eat meats (Section 5.4.3).

Toxoplasmosis can arise when meat is undercooked or served rare. Consumption of undercooked game meat (e.g. venison and wild boar) has caused outbreaks overseas (Pinto-Ferreira *et al.*, 2019a). For example, a group of seven hunters developed toxoplasmosis after consuming wild deer steak cooked rare (Gaulin *et al.*, 2020). In France, three people became ill after consuming undercooked leg of lamb and *T. gondii* was isolated from the lamb meat (Ginsbourger *et al.*, 2012).

Controls

Following basic food hygiene rules when cooking and cooling meats reduces the risk. However, temperature abuse and/or undercooking may contribute to situations where foodborne illness may occur. Meat should not be tasted during cooking.

L. monocytogenes is rapidly inactivated at 70ºC or above, with a the *D* time at 70°C of 10–15 seconds (Farber and Peterkin, 1991). Longer times are needed for inactivation when cooking at lower temperatures (Section 3.1.1).

Heat kills *T. gondii* cysts, with the rate of inactivation increasing with temperature. Cysts are quickly inactivated above 60ºC (De Berardinis *et al.*, 2017). On the basis of experimental findings it has been recommended that "it is best to cook whole cuts of pork, lamb, veal, or beef to a least 150°F (65.6°C) with a 3-minute rest. Ground meat and wild game meat should be cooked to 160° F (71.1°C) or higher, and poultry should be cooked to 165°F (73.9°C) or higher as measured with a food thermometer. Ground meats, wild game meats, and poultry do not require a rest time. Microwave cooking is unreliable for killing *T. gondii*" (Jones and Dubey, 2012). Another review recommends that whole poultry should be cooked to 82ºC as measured in the thigh to ensure doneness (Hussain *et al.*, 2017). See also Section 3.2.1.

In New Zealand, poultry preparation and consumption have been associated with campylobacteriosis (Lake *et al.*, 2019). *Campylobacter* spp. are susceptible to heat and will be killed when poultry is thoroughly cooked. The temperatures recommended for killing *Listeria* spp. and *T. gondii* will also kill *Campylobacter* spp.

Temperature-related advice is only useful when temperature probes are routinely used (as in the food industry), which is not typically the case in the domestic situation. Alternative statements such as "cooking to piping hot" or "until juices run clear" are commonly used, which, although potentially subjective, are likely to result in adequately cooked (or even potentially overcooked) meats. Another common statement using colour (or "pinkness") as an indication of meat doneness can, however, be potentially misleading and should be avoided (King and Whyte, 2006). The statement "cook thoroughly until piping hot throughout" is of particular relevance to comminuted meat products such as burgers and sausages where microorganisms are distributed through the entire meat matrix and may survive cooking if the centre of the product is not cooked to a sufficiently high temperature.

Summary

Present New Zealand Food Safety advice is consistent with current scientific evidence. Additional advice might be added regarding recommended cooking temperatures to support the advice to use a food thermometer. The advice on leftovers is in keeping with Section 5.7.1, since the heating step will inactivate any *L. monocytogenes* that might have contaminated the meat after cooking. However, the advice might be extended to encompass scenarios where consumers have purchased cooked meats retail (e.g. at delicatessens), e.g. "reheat leftovers and cold cooked meats until piping hot (over 70ºC)". These cold, ready-to-eat meats are not considered under processed meats (Section 5.4.3).

5.4.3 Processed meats

Ham, salami, luncheon, pâté, pastrami, biltong, or jerky (dried meat), etc.

CURRENT ADVICE:	Don't eat unless heated until piping hot (over 70ºC)

Food

Processed meats are difficult to classify due to the wide range of types available, including uncooked cured shelf stable meats (e.g. fermented salami), dried meats (e.g. jerky), cooked perishable uncured meats (e.g. pâté) and cooked perishable cured meats (e.g. ham). These are available at retail, pre-packed by the manufacturer, packaged in-store and as loose product in delicatessens and butchers. While some processed meats, such as ham and bacon, can be purchased raw and cooked at home, this section primarily concerns ready-to-eat products.

Hazards

International food safety advice makes no distinction between manufacturer-packaged and re-packaged meats, and advises pregnant women to avoid all cold processed meats, and to eat them only if hot. Equipment used to slice product in retail stores for presentation of unpackaged ready-to-eat meats can be an important source of pathogens and cross-contamination between products.

Listeria

While there is considerable variability in the processes used for making processed meats, *L. monocytogenes* concentrations will generally reduce through drying, fermentation, salting and cooking steps. However, if post-processing contamination occurs (or *L. monocytogenes* survives processing), the conditions may be suitable for growth during subsequent storage. The amount of growth depends on the product, but products with higher pH (>5) are more favourable (Gilbert *et al.*, 2009; King *et al.*, 2012).

There have been three New Zealand surveys of ready-to-eat processed meats. These have revealed a low prevalence of *L. monocytogenes* in both packaged and unpackaged ham, pâté, and a range of other ready-to-eat meats.

During 2003/04, in a survey of pre-packaged ham samples, 104 samples were tested consisting of 16 brands (Wong *et al.*, 2005a). All samples were held at 4°C and tested at the end of their shelf life. One sample contained *L. monocytogenes* (50 CFU/g), and six samples contained other *Listeria spp*. including two samples with *L. welshimeri* (<50 and 450 CFU/g), and four with *L. innocua* (<50, 50, 200 and 400 CFU/g). In the same study, 60 batches from nine brands of pâté were also surveyed. *L. monocytogenes* (1,700 CFU/g) was isolated from one of the 300 pâté samples tested. The concentration in the sample may have been higher but the aerobic plate count of 10^8 CFU/g inhibited laboratory testing.

During the period 2004–2006, samples of unpackaged ham were tested based on market share, i.e. 80% of samples were from supermarkets, 20% from other delicatessens (Cornelius *et al.*, 2008). From the 301 samples tested, 10 (3.3%) were contaminated with only *L. monocytogenes*. Eight of the samples (2.7%) contained *L. innocua* only and 3 samples (1%) contained both *L. monocytogenes* and *L. innocua.* Overall, *L. monocytogenes* was present at a prevalence of 4.3%, at concentrations of <50 CFU/g except for two samples containing >100 CFU/g.

A survey of retail packaged ready-to-eat meat products in New Zealand was completed during 2011/12 and included a variety of salted, cured, dried, and cooked meats (Rivas *et al.*, 2017). Products from 32 producers were surveyed and 1,485 samples from 297 lots were analysed. *L. monocytogenes* was isolated from 19 lots of product, resulting in a prevalence of 6.4%. The majority of lots (15 of 19) contained <50 CFU/g *L. monocytogenes*. Sliced ham was the product most often associated with *L. monocytogenes* contamination (8/19 lots), with lots coming from 5 different producers. The other ready-to-eat meat types positive for *L. monocytogenes* were silverside, beef, bloedworst, luncheon, andoullie, salami and pork. Fourteen of the 19 lots contaminated with *L. monocytogenes* also contained another *Listeria spp*., either *L. welshimeri*, *L. innocua,* or *L. seeligeri*.

Ready-to-eat (processed) meats including ham, terrine, and sausage products have been the subject of four New Zealand recalls since 2015 for having *L. monocytogenes* levels above regulatory limits.[25]

Toxoplasma

T. gondii does not replicate outside the host so will not grow in food. If present in meat products, *T. gondii* is most likely to be in the cyst (bradyzoite) stage. There are no surveys of *T. gondii* in processed meats in New Zealand. Surveys in other countries show that viable *T. gondii* have been detected in ham (Gomez-Samblas *et al.*, 2015), and *T. gondii* DNA in cured salami, smoked meat products, sausages (including dry sausages) and ham (Costa *et al.*, 2018; Sroka *et al.*, 2019). Other surveys of ham and pastrami did not detect *T. gondii* (Bayarri *et al.*, 2012; Yildirim *et al.*, 2017).

Human illness

Processed meat has been the source of two New Zealand outbreaks of listeriosis. During 2000 there was an outbreak involving 30 cases of non-invasive febrile gastroenteritis caused by *L. monocytogenes*, which was associated with ham and corned silverside (Sim *et al.*, 2002). Another outbreak in 2012 involved ready-to-eat meat from a single producer which was served at a two hospitals and resulted in six cases of listeriosis (Rivas *et al.*, 2019).

A quantitative risk assessment undertaken in Australia concluded that ready-to-eat meats were responsible for up to 40% of cases of listeriosis, which was consistent with epidemiological data available at the time (Ross *et al.*, 2009).

During 2017/18 the world's largest outbreak of listeriosis occurred in South Africa and was caused by a ready-to-eat processed meat product (polony) produced by a single manufacturer (Tchatchouang *et al.*, 2020). There were approximately 1,060 confirmed cases and 216 deaths.

In New Zealand, outbreaks of campylobacteriosis have also been linked to chicken liver pâté (Section 3.3.3), and during 1999 an outbreak of salmonellosis was linked to cold cocktail sausages from a New Zealand butchery (MacLeod, 2000).

Controls

Salami and other high acid fermented meats rely on low pH (4.6–5.3) and a reduced water activity of <0.95 for microbial stability (ICMSF, 1996). If the moisture reduction during drying is less than 15%, smoking and mild heat treatment may be additional steps to restrict microbial growth. Dried meats such as biltong and jerky rely on low water activity for microbial stability at ambient temperature, and must be dried to a water activity ≤0.85 (Australia New Zealand Food Standards Code, Standard 2.2.1).[26] Nitrite and sodium ascorbate added to processed meat products may also inhibit bacterial growth (Gilbert *et al.*, 2009; King *et al.*, 2012).

The preparation of some processed meats includes a cook step. However, contamination of any processed meat can occur during packaging, slicing or handling.

The salting, curing and drying processes undertaken by processed meat manufacturers can inactivate *T. gondii* cysts (Deng *et al.*, 2020). However, there is a large variety of processes

[25] https://www.mpi.govt.nz/food-safety/food-recalls/recalled-food-products/ (accessed 23 March 2020).
[26] Available at https://www.legislation.gov.au/Series/F2015L00427 (accessed 28 May 2020).

so it is not easy to predict whether *T. gondii* cysts will survive through to the final product. For example, studies have shown that the curing process used for ham reduced the risk of viable *T. gondii* being present in the final product but complete inactivation may not always be achieved (Genchi *et al.*, 2017b; Gomez-Samblas *et al.*, 2016; Herrero *et al.*, 2017).

Summary

Present New Zealand Food Safety advice is consistent with current scientific evidence. Although *L. monocytogenes* prevalence and concentration appears to be low in New Zealand processed meats, the survey results described above do indicate the occasional presence of this pathogen in pre-packaged processed meats. Processed meats have been associated with two outbreaks in New Zealand, and large outbreaks in other countries.

It is recognised that the risks differ between different processed meats, ham vs. jerky for example, but providing separate advice for individual products within this wide food group is likely to create confusion, particularly since production processes are so varied. It might be useful to add additional advice for raw ham and bacon that aligns with the advice for cooking meat (Section 5.4.2).

5.4.4 Cold cooked poultry

Any cold pre-cooked poultry (e.g. chicken, turkey)

CURRENT ADVICE:	Don't eat unless heated until piping hot (over 70ºC)

Food

See raw meat and poultry (Section 5.4.1). Cold cooked poultry has been cooked thoroughly then cooled and refrigerated.

Hazards

The same considerations as for cooked meats apply. Cross-contamination and undercooking are potential mechanisms for the contamination of cooked poultry. Research from the USDA has identified raw poultry as the primary source of *L. monocytogenes* in commercial chicken further processing plants (Berrang *et al.*, 2010). *L. monocytogenes* is able to grow on cold cooked poultry (and other cold cooked meats such as beef and pork) (Gilbert *et al.*, 2009).

Human illness

No listeriosis or toxoplasmosis outbreaks have been reported in New Zealand associated with consumption of cold cooked poultry. Listeriosis outbreaks associated with cooked poultry have occurred in other countries.[27] An outbreak in Australia during 2009 involved 40 cases (including eight perinatal infections with three foetal deaths) and was caused by cooked chicken meat served in wraps and sandwiches (OzFoodNet, 2009). Other cold cooked meats have also caused listeriosis outbreaks. For example, during 2019 an outbreak in Spain was caused by *L. monocytogenes* on chilled roasted pork meat from a single manufacturer. There were 222 confirmed cases, including 38 pregnant women of whom six were reported to have had miscarriages as a result of listeriosis.[28]

[27] https://www.canada.ca/en/public-health/services/public-health-notices/2019/outbreak-listeria-infections-cooked-diced-chicken.html (accessed 12 May 2020).
[28] https://www.who.int/csr/don/16-september-2019-listeriosis-spain/en/ (accessed 12 May 2020).

Controls

The same considerations as for cooked meats apply.

Summary

Present New Zealand Food Safety advice is consistent with current scientific evidence. There is also evidence that other cold cooked meat types pose a risk for listeriosis (above, and Section 5.4.3). An option is to alter this food description from “cold cooked poultry” to “cold cooked meats, e.g. chicken, turkey, beef and pork.”

5.5 SEAFOOD

5.5.1 Raw fish

Any raw fish (including marinated raw fish)

CURRENT ADVICE:	Don’t eat

Food

Raw fish is the uncooked flesh of cold-blooded aquatic vertebrates of the *Pisces* class. Sashimi is a Japanese delicacy of very fresh, raw fish, often used as an ingredient in sushi. Gravad fish is raw fish cured in salt and sugar without any thermal treatment (Jami *et al.*, 2014). Other marinades may also be used, e.g. curing in salt and lemon juice followed by addition of coconut milk/cream and other ingredients.

Hazards

Fish can present chemical and microbial hazards. Some fish species may be high in mercury (Section 4.1), and others may expose consumers to histamine (produced by bacteria on fish in the Scombroidae and Scomberesocidae families) or ciguatoxin (sequestered by herbivorous fish consuming toxin-producing epiphytic dinoflagellates or carnivorous fish consuming contaminated herbivorous fish) (Feng *et al.*, 2016; Rhodes *et al.*, 2020). The latter two toxins are the most common causes of foodborne disease associated with fish consumption (Barrett *et al.*, 2017).

Listeria

Listeria can occur naturally in freshwater and marine environments and therefore it is possible for *L. monocytogenes* to be associated with wild and farmed fish (Jami *et al.*, 2014). The prevalence of *Listeria* spp. in surface water may be influenced by agricultural run-off and be elevated after rain events. Contamination occurs on the surface of fish which can be spread during processing and more widely to plant equipment leading to cross-contamination of processed flesh or ready-to-eat products. Cross-contamination can also occur from food contact surfaces or equipment in premises managing multiple foods. *L. monocytogenes* have been isolated from non-food contact areas in New Zealand seafood processing environments (Cruz and Fletcher, 2011).

A review of a large number of studies suggests that the prevalence of *L. monocytogenes* in wild fish ranges from 0% to 30% depending on freshwater or saltwater habitats and geographic locations (Jami *et al.*, 2014). These figures also depend on the sampling methodology, with gill sampling more likely to return positive results. The prevalence in marinated fish can be higher (almost 40%).

No growth of *L. monocytogenes* is expected under frozen storage but slow growth is possible at refrigeration temperatures (Holck *et al.*, 2018; Liu *et al.*, 2016).

Under experimental conditions, *L. monocytogenes* grew on marinated or gravid fish at 8ºC (Mejlholm and Dalgaard, 2007). A small survey in Italy detected *L. monocytogenes* in marinated seafood products but the pathogen was no longer detected at the end of shelf life (Gambarin *et al.*, 2012).

A survey of sashimi collected from sushi restaurants in Portugal during summer or winter months found *L. monocytogenes* in 23/114 (20.2%) samples (Migueis *et al.*, 2016). The concentrations were above regulatory limits in four samples collected during the summer.

Toxoplasma

T. gondii does not replicate outside the host so will not grow in food. Fish are not considered to be natural biological hosts for *T. gondii* but can carry the parasite on and in their body if they live in water receiving freshwater inputs from land (Marino *et al.*, 2019). If present in fish, *T. gondii* is most likely to be in the oocyst (sporozoite) stage. Sporulated oocysts can remain viable in seawater for months, particularly at cooler temperatures (Lindsay and Dubey, 2009). Laboratory studies have demonstrated that viable *T. gondii* oocysts can be retained in the alimentary canals of sardines and anchovies for several hours (Massie *et al.*, 2010).

There are no surveys for New Zealand fish but overseas studies indicate *T. gondii* can be found on and in fish. For example, *T. gondii* DNA was detected in pooled samples of gill, intestine and skin/muscle tissues from a range of finfish species sampled from markets in Italy (Marino *et al.*, 2019). A study from China also detected *T. gondii* DNA on finfish, but only one sample was positive (1/456 carp) (Zhang *et al.*, 2014).

Human illness

No reports of listeriosis associated with the consumption of raw fish in New Zealand were located. Overseas, there are very few reports (smoked fish have been implicated more frequently and with stronger evidence) (Jami *et al.*, 2014). There are no reports of toxoplasmosis from the consumption of raw (or undercooked) fish (Marino *et al.*, 2019).

See sushi (Section 5.7.4).

Controls

Temperature control (keeping fish cool) is the main control over the microbial quality of raw fish. Salt and acid in marinades will make conditions less favourable for *L. monocytogenes* but a marinade should not be relied upon as a control.

Summary

Present New Zealand Food Safety advice is consistent with current scientific evidence, although there is little evidence from outbreak data to support this food as being an important cause of listeriosis. Contamination of raw fish with *L. monocytogenes* from the processing environment appears to be of more concern than natural contamination from the live animal's habitat. The risk from viable *T. gondii* oocysts has not been demonstrated. Overseas surveys have demonstrated that *L. monocytogenes* can be detected on raw fish, where it can grow.

5.5.2 Raw shellfish

Any raw shellfish (including marinated raw mussels)

CURRENT ADVICE:	Don't eat

Food

This food group includes both saltwater and freshwater invertebrates, including molluscs (e.g. clams, mussels, oysters and scallops), crustaceans (e.g. shrimps, prawns and lobsters) and echinoderms (e.g. sea urchin).

Hazards

The filter-feeding habit of molluscan shellfish means these animals accumulate microbial pathogens if these are present in their growing waters.

Listeria

In New Zealand, *L. monocytogenes* has been isolated from commercially grown green-lipped mussels (Cruz and Fletcher, 2011). The prevalence of *L. monocytogenes* in raw (n=831) and processed (n=2,313) mussels from three processing facilities was 0.24% and 0.17% respectively. There was also evidence of persistent strains (pulsotypes) of *L. monocytogenes* within processing facilities, including non-food contact areas, and there is potential for cross-contamination of product from internal and external environments.

Overseas studies report variable *L. monocytogenes* prevalence in bivalve shellfish, ranging from 0% to 24% (Jami *et al.*, 2014). *L. monocytogenes* have also been detected in shrimp and prawns. No studies were located that investigated whether or not *L. monocytogenes* will grow in shellfish held on ice or refrigerated post-harvest.

Salt, sugar and acids in marinades will make conditions less suitable for *L. monocytogenes* growth. Under experimental conditions, *L. monocytogenes* were inactivated on mussels in various marinades but not predictably (Bremer and Osborne, 1995). Marinades will be less effective in naturally contaminated mussels where the *L. monocytogenes* will be inside the mussel.

Toxoplasma

T. gondii does not replicate outside the host so will not grow in food. Shellfish are not considered to be natural biological hosts for *T. gondii* but can bioaccumulate oocysts as they filter seawater (Coupe *et al.*, 2018). If present in shellfish, *T. gondii* is most likely to be in the oocyst (sporozoite) stage. Sporulated oocysts can remain viable in seawater for months, particularly at cooler temperatures (Lindsay and Dubey, 2009).

In a New Zealand survey, *T. gondii* DNA was detected in 13/104 (12.5%, 95% CI 8-20%) individual green-lipped mussels (*Perna canaliculus*) sampled from retail outlets (Coupe *et al.*, 2018). Only the haemolymph was tested. The odds of a positive sample was 15 times higher in samples collected during summer compared with winter. Sporozoite-specific mRNA was detected in 4/7 haemolymph samples, confirming the presence of sporulated oocysts.

There are multiple reports of *T. gondii* being detected in raw shellfish tested from other countries; a recent review summarises several reports of *T. gondii* being detected in commercial and non-commercial shellfish including clams, mussels and oysters (Shapiro *et al.*, 2019). Detection is usually by PCR. For example, *T. gondii* DNA was detected in 31% (n=1440) of individual oysters collected over two years from six sites in the USA's Gulf of

Maine (Marquis *et al.*, 2019). Small numbers of shrimp and crawfish were positive for *T. gondii* DNA in a Chinese study (Zhang *et al.*, 2014). A study in California found an association between the presence of *T. gondii* DNA in mussels and proximity to freshwater run-off and collection during the wet season, although the overall prevalence was low (13/959) (Shapiro *et al.*, 2015).The long-term survival of viable oocysts in mussels and oysters has been confirmed by bioassay under experimental conditions (Arkush *et al.*, 2003; Lindsay *et al.*, 2004).

Human illness

No reports of listeriosis associated with consumption of raw or undercooked shellfish were located.

There are no reports of toxoplasmosis from the consumption of raw (or undercooked) shellfish (Marino *et al.*, 2019). However, shellfish are less likely to be recognised as a potential vehicle of transmission compared to contaminated water, contact with cat faeces or consumption of undercooked meat (Robertson, 2007).

In contrast to the above, there are multiple reports of outbreaks caused by consumption of raw (or undercooked) shellfish contaminated with other pathogens, including norovirus, hepatitis A virus, *Shigella* spp. and *Vibrio* spp., both overseas and in New Zealand (Bellou *et al.*, 2013; King *et al.*, 2013; Simmons *et al.*, 2007).[29]

Controls

Controls for filter feeding bivalve molluscs in New Zealand focus mainly on water quality in growing and processing areas. Salt and acid in marinades will make conditions less favourable for *L. monocytogenes* but a marinade should not be relied upon as a control.

Summary

Present New Zealand Food Safety advice is consistent with current scientific evidence based on the risk of illness from a range of pathogens, not just *L. monocytogenes* and *T. gondii*. The current food description “shellfish” might be altered to encompass other marine invertebrates such as shrimps and prawns.

5.5.3 Smoked fish, shellfish and crustacean

Chilled, pre-cooked fish, mussels, oysters*, scallops*, salmon, crayfish, prawns, etc.

CURRENT ADVICE:	Don’t eat unless heated until piping hot (over 70ºC)

* Bluff and Pacific oysters and queen scallops contain more cadmium than other foods. We recommend you eat these shellfish only once per month during pregnancy.

Food

See raw fish (Section 5.5.1) and raw shellfish (Section 5.5.2).

Smoked products are produced by placing salted fish/shellfish in a smoke chamber and heating at >60°C for 6 to 10 hours (hot smoking) or 20–30°C for 2 to 4 days (cold smoking) (Moorhead, 2011). Following smoking, fish are often trimmed, sliced and vacuum packaged for commercial sale.

[29] https://www.mpi.govt.nz/news-and-resources/media-releases/food-poisoning-associated-with-consumption-of-raw-mussels/ (accessed 12 May 2020).

Hazards

See raw fish (Section 5.5.1) and raw shellfish (Section 5.5.2). Mercury concentration varies with fish species (Section 4.1). Cadmium levels in Bluff oysters and Queen scallops can be high. However, cadmium exposure is a chronic health concern and no excess risks associated with pregnancy have been identified.

The concentration of *L. monocytogenes* is reduced through a combination of heat, salt and phenol compounds in the smoke treatment (Hwang *et al.*, 2009; Porsby *et al.*, 2008). *Listeria* are known to persist in seafood processing environments and is the pathogen of most concern in cooked seafood products, where there is potential for cross-contamination through post-process handling, such as slicing. In addition, if the smoking process is not adequately controlled, it is not a lethal process for listeriae. The prevalence of *L. monocytogenes* in smoked seafood worldwide is well established (Jami *et al.*, 2014).

A survey in 1991 examined ready-to-eat shellfish and finfish from retail establishments (Hudson *et al.*, 1992). Fourteen of the 25 shellfish products were smoked mussels, and of these, 5 (35.7%) were positive for *L. monocytogenes*. In addition, 8/12 (75%) samples of smoked salmon in the survey were positive.

Another New Zealand survey focused on end of shelf life testing of hot or cold smoked, vacuum packed salmon (Nortje *et al.*, 2001). Products were sourced directly from four New Zealand manufacturers and the same brands from supermarkets. There was also one imported cold smoked salmon sample from a supermarket. All samples were stored at 4°C until ±2 days of their 'best before or use by' dates. Of the 136 cold smoked samples tested, 15 manufacturer and 15 supermarket samples were positive for *L. monocytogenes.* Concentrations above 100 CFU/g were recorded for two of the positive supermarket samples and 11 of the manufacturer samples. *L. monocytogenes* was isolated from 8/80 hot smoked salmon samples (four each from manufacturers and supermarkets). Only one sample (from a supermarket) had a concentration above 100 CFU/g.

A more recent survey of ready-to-eat cold and hot smoked salmon in New Zealand was completed in 2010. Samples (n=1,212) of smoked salmon products from 19 producers were purchased from retail outlets or directly from processors over a period of 12 months. Eight samples, all from cold-smoked products, were positive for *L. monocytogenes* resulting in a prevalence of 0.7% (95% CI 0.3–1.3) (Moorhead, 2011).

Five smoked fish or shellfish products have been the subject of a MPI food safety recall since 2015 for *L. monocytogenes* levels above the regulatory limit.[30]

There are no data on *T. gondii* in smoked fish. Smoking appears to inactivate tissues cysts (Lunden and Uggla, 1992) but oocysts are the more resistant form of the parasite.

Human illness

A well-documented outbreak of perinatal listeriosis in New Zealand was associated with the consumption of smoked mussels (Baker *et al.*, 1993; Brett *et al.*, 1998). There were four clinical cases, including newborn twins who died. The investigation led to the testing of an unopened packet of smoked mussels from the refrigerator of the twins' mother. PFGE analysis showed the *L. monocytogenes* serogroup 1/2a isolates from each twin's blood, from the other two cases, and from the mussels were indistinguishable. However one of these

[30] https://www.mpi.govt.nz/food-safety/food-recalls/recalled-food-products/ (accessed 23 March 2020).

cases had not consumed smoked mussels. The mother of the twins had consumed a significant quantity of the mussels throughout her pregnancy in order to raise her iron levels.

Overseas, listeriosis outbreaks have also been caused by smoked fish (Gillesberg Lassen *et al.*, 2016; Lopez-Valladares *et al.*, 2018).

There are no reports of toxoplasmosis from the consumption of smoked seafood.

Controls

Temperature control and the smoking process are the primary controls.

Summary

Surveys have demonstrated the presence of *L. monocytogenes* on smoked fish and growth of *L. monocytogenes* is possible in these products. There are no reported listeriosis outbreaks associated with smoked fish in New Zealand but outbreaks have been reported in other countries. There has been an outbreak of listeriosis associated with smoked mussels in New Zealand.

Present New Zealand Food Safety advice is consistent with current scientific evidence. The recommended heating step will destroy any *L. monocytogenes* that may be on the product.

5.5.4 Cooked fish, shellfish and crustacean

Freshly cooked fish, mussels, oysters, crayfish, scallops, etc.

CURRENT ADVICE:	(not specifically included)

Advice considered in Gilbert *et al.* (2010) was “Cook thoroughly until piping hot throughout; eat while hot.”

Food

See raw fish (Section 5.5.1) and raw shellfish (Section 5.5.2). It is common for seafood to be lightly cooked so the inside is still raw or undercooked.

Hazards

See raw fish (Section 5.5.1) and raw shellfish (Section 5.5.2). Mercury concentration varies with fish species (Section 4.1). Cadmium levels in Bluff oysters and Queen scallops can be high. However, cadmium exposure is a chronic health concern and no excess risks associated with pregnancy have been identified.

It is unlikely that *L. monocytogenes* or *T. gondii* oocysts are present inside muscle tissues but the possibility of cross-contamination during filleting cannot be excluded.

For pre-cooked seafood available at retail, similar considerations as for smoked seafood apply. *L. monocytogenes* can grow on cold, cooked seafood, with crustacea (e.g. shrimp, prawns) appearing to provide the very suitable conditions (Appendix C). A small survey of ready-to-eat finfish and shellfish from New Zealand retail did not detect *L. monocytogenes* in any cooked product (Hudson *et al.*, 1992).

Human illness

There are no reports of outbreaks of foodborne illness associated with cooked seafood in New Zealand.

Consumption of shrimp at a party was the likely cause of ten cases of listeriosis, including two pregnant women, in the USA (Riedo *et al.*, 1994). There are no reports of toxoplasmosis from the consumption of cooked seafood.

Controls

Similar considerations as for cooked meats apply. Adequate cooking will inactivate microorganisms.

Cooking to at least 60ºC appears necessary for inactivation of *T. gondii* oocysts, based on experiments with oocysts in water and considering the short cooking times usually used for seafood (Dubey, 1998). A more recent experiment expands on this finding: An oocyst suspension heat-treated at 60ºC for 1 minute was not infectious when tested by mouse bioassay (Travaillé *et al.*, 2016). However, a heat treatment of 80ºC for 2 minutes was needed to reduce the metabolic activity of the oocysts, based on detection of mRNA. It is unclear whether metabolically active oocysts will recover and become infectious.

Summary

The current scientific evidence would be consistent with the advice “Cook thoroughly until piping hot throughout; eat while hot.” Pre-cooked seafood available at retail is susceptible to *L. monocytogenes* contamination in the same way that smoked seafood is, and *L. monocytogenes* can grow on cooked seafood. If advice will not be specifically included for cooked fish, then it is suggested that the food group “Smoked fish, shellfish and crustacean” be extended to include pre-cooked seafood, e.g. “Smoked or pre-cooked fish, shellfish and crustacean.”

5.5.5 Recommended servings for fish species to minimise mercury intakes

CURRENT ADVICE:

No restriction necessary	Anchovy • Arrow squid • Barracouta • Blue cod • Brill/Turbot • Brown trout (except from Lake Ellesmere) • Cockles • Eel, long or short finned • Elephant fish • Flounders • Gurnard • Hoki • John Dory • Monkfish or stargazer • Mussels (green and blue) • Orange perch • Oysters (except Bluff and Pacific) • Parore • Scallops (except Queen) • Rainbow trout (only from nongeothermal regions) • Skipjack tuna (No data for yellowfin tuna) • Sole (except Lemon sole) • Southern blue whiting • Surf clams (e.g. tuatua) • Tarakihi • Toothfish, Antarctic • Warehou (common, silver and white) • Whitebait (Inanga)
3 – 4 servings per week acceptable	Albacore tuna • Alfonsino • Bass • Bluenose • Gemfish • Ghost sharks • Hake • Hapuka (Groper) • Javelin Fish • Kahawai • Kingfish • Lake Taupo trout • Leatherjacket • Lemon sole • Ling • Mackerel (blue and jack) • Orange Roughy • Oreo dories • Red cod • Ribaldo • Rig (Lemonfish, Spotted dogfish) • Rock Lobster • Salmon (farmed) • Sea perch • Silverside • Skate • Smooth oreo • Snapper • Sprats • Trevally

1 serving per 1 – 2 weeks acceptable	Cardinal fish • Dogfish (excluding rig) • Lake Rotomahana trout • Lake trout from geothermal regions • School shark (Greyboy, Tope) • Marlin (striped) • Southern bluefin tuna • Swordfish

Advice to pregnant women on fish consumption has generally been directed to minimising exposure to methylmercury. For example, advice to pregnant women in New Zealand with respect to fish consumption, is species specific (MOH, 2008; New Zealand Food Safety, 2020), including.

- For those New Zealand fish species with low mercury levels, no restriction of consumption is recommended.
- For New Zealand fish species with moderate mercury levels, 3-4 servings per week are recommended.
- For New Zealand fish species with elevated mercury levels, 1 serving per 1-2 weeks is acceptable.

Not all fish are equivalent in terms of mercury concentration. Mercury, and particularly methylmercury, accumulates along the food chain, with bigger, older, fish further up the food chain containing higher concentrations of mercury than younger or smaller fish lower in the food chain. Thus exposure to mercury is influenced by the choice of fish consumed.

In 2010, FAO and WHO convened an expert consultation to consider the risks and benefits associated with fish consumption (FAO/WHO, 2011). The consultation concluded that “When comparing the benefits of LCn3PUFAs with the risks of methylmercury among women of childbearing age, maternal fish consumption lowers the risk of suboptimal neurodevelopment in their offspring compared with the offspring of women not eating fish in most circumstances evaluated”. The consultation developed dose-response relationships for the impact of methylmercury (negative) and LCn3PUFA (positive) on child neurodevelopment. Along with information on the methylmercury and LCn3PUFA content of various fish species, these dose-response relationships were used to estimate the overall impact on child neurodevelopment of maternal fish consumption. The summary tables from this analysis are reproduced in Tables 3 and 4 below.

Table 3. Classification of 96 fin fish and shellfish species by content of LCn3PUFA (EPA plus DHA) and total mercury

		LCn3PUFA (EPA + DHA)			
		x ≤ 3 g/kg	3 < x ≤ 8 g/kg	8 < x ≤ 15 g/kg	x > 15 g/kg
Mercury	x ≤0.1 mg/kg	**Fish:** butterfish; catfish; cod, Atlantic; cod, Pacific; croaker, Atlantic; haddock; pike; plaice, European; pollock; saithe; sole; tilapia **Shellfish:** clams; cockle; crawfish; cuttlefish; oysters; periwinkle; scallops; scampi; sea urchin; whelk	**Fish:** flatfish; John Dory; perch, ocean and mullet; sweetfish; wolf fish **Shellfish:** mussels; squid	**Fish:** redfish; salmon, Atlantic (wild); salmon, Pacific (wild); smelt **Shellfish:** crab, spider; swimcrab	**Fish:** anchovy; herring; mackerel; rainbow trout; salmon, Atlantic (farmed); sardines; sprat **Fish liver:** cod, Atlantic (liver); saithe (liver) **Shellfish:** crab (brown meat)
	0.1 < x ≤ 0.5 mg/kg	**Fish:** anglerfish; catshark; dab; grenadier; grouper; gurnard; hake; ling; lingcod and scorpionfish; Nile perch; pout; skate/ray; snapper, porgy and sheepshead; tuna, yellowfin; tusk; whiting **Shellfish:** lobster; lobster, American	**Fish:** bass, freshwater; carp; perch, freshwater; scorpion fish; tuna; tuna, albacore **Shellfish:** crab; lobster, Norway; lobsters, spiny	**Fish:** bass, saltwater; bluefish; goatfish; halibut, Atlantic (farmed); halibut, Greenland; mackerel, horse; mackerel, Spanish; seabass; seabream; tilefish, Atlantic; tuna, skipjack	**Fish:** eel; mackerel, Pacific; sablefish
	0.5 < x ≤ 1.0 mg/kg	**Fish:** marlin; orange roughy; tuna, bigeye	**Fish:** mackerel, king; shark	**Fish:** alfonsino	**Fish:** tuna, Pacific bluefin
	>1.0 mg/kg		**Fish:** swordfish		

Reproduced from FAO/WHO (2011)

LCn3PUFA: long-chain n-3 polyunsaturated fatty acids, EPA: eicosapentaenoic acid, DHA: docosahexaenoic acid

Table 4. Estimated changes in child IQ resulting from the child's mother having consumed fish with different methylmercury and EPA plus DHA contents at one, two, four and seven servings per week

			LCn3PUFA (EPA + DHA)			
			x ≤ 3 g/kg	3 < x ≤ 8 g/kg	8 < x ≤ 15 g/kg	x > 15 g/kg
		Median	2	5.5	11.5	20
One serving per week						
Methylmercury	x ≤0.1 mg/kg	0.05	-0.02 -0.08 +0.77	-0.02 -0.08 +2.1	-0.02 -0.08 +4.4	-0.02 -0.08 +5.8
	0.1 < x ≤ 0.5 mg/kg	0.30	-0.12 -0.47 +0.77	-0.12 -0.47 +2.1	-0.12 -0.47 +4.4	-0.12 -0.47 +5.8
	0.5 < x ≤ 1.0 mg/kg	0.75	-0.30 -1.2 +0.77	-0.30 -1.2 +2.1	-0.30 -1.2 +4.4	-0.30 -1.2 +5.8
	>1.0 mg/kg	1.5	-0.60 -2.3 +0.77	-0.60 -2.3 +2.1	-0.60 -2.3 +4.4	-0.60 -2.3 +5.8
Two servings per week						
Methylmercury	x ≤0.1 mg/kg	0.05	-0.04 -0.2 +1.5	-0.04 -0.2 +4.2	-0.04 -0.2 +5.8	-0.04 -0.2 +5.8
	0.1 < x ≤ 0.5 mg/kg	0.30	-0.2 -0.9 +1.5	-0.2 -0.9 +4.2	-0.2 -0.9 +5.8	-0.2 -0.9 +5.8
	0.5 < x ≤ 1.0 mg/kg	0.75	-0.6 -2.3 +1.5	-0.6 -2.3 +4.2	-0.6 -2.3 +5.8	-0.6 -2.3 +5.8
	>1.0 mg/kg	1.5	-1.2 -4.7 +1.5	-1.2 -4.7 +4.2	-1.2 -4.7 +5.8	-1.2 -4.7 +5.8
Four servings per week						
Methylmercury	x ≤0.1 mg/kg	0.05	-0.08 -0.31 +3.1	-0.08 -0.31 +5.8	-0.08 -0.31 +5.8	-0.08 -0.31 +5.8
	0.1 < x ≤ 0.5 mg/kg	0.30	-0.48 -1.9 +3.1	-0.48 -1.9 +5.8	-0.48 -1.9 +5.8	-0.48 -1.9 +5.8
	0.5 < x ≤ 1.0 mg/kg	0.75	-1.2 -4.7 +3.1	-1.2 -4.7 +5.8	-1.2 -4.7 +5.8	-1.2 -4.7 +5.8
	>1.0 mg/kg	1.5	-2.4 -9.3 +3.1	-2.4 -9.3 +5.8	-2.4 -9.3 +5.8	-2.4 -9.3 +5.8
Seven servings per week						
Methylmercury	x ≤0.1 mg/kg	0.05	-0.14 -0.5 +5.4	-0.14 -0.5 +5.8	-0.14 -0.5 +5.8	-0.14 -0.5 +5.8
	0.1 < x ≤ 0.5 mg/kg	0.30	-0.84 -3.3 +5.4	-0.84 -3.3 +5.8	-0.84 -3.3 +5.8	-0.84 -3.3 +5.8
	0.5 < x ≤ 1.0 mg/kg	0.75	-2.1 -8.2 +5.4	-2.1 -8.2 +5.8	-2.1 -8.2 +5.8	-2.1 -8.2 +5.8
	>1.0 mg/kg	1.5	-4.2 -16.3 +5.4	-4.2 -16.3 +5.8	-4.2 -16.3 +5.8	-4.2 -16.3 +5.8

Reproduced from FAO/WHO (2011)
LCn3PUFA: long-chain n-3 polyunsaturated fatty acids, EPA: eicosapentaenoic acid, DHA: docosahexaenoic acid

Fish serving size was estimated to be 100 g. Ratio of DHA to EPA + DHA was assumed to be 0.67. Maternal body weight was assumed to be 60 kg. The numbers in the upper row in each cell are estimates of IQ points lost from methylmercury exposure, with the lower value of the two values calculated using a central estimate of −0.18 and the higher value calculated using an upper-bound estimate of −0.7. The number in the lower row in each cell is the estimate of IQ points gained from DHA exposure using the coefficient of 4 IQ points for 100 mg of DHA intake. The maximum positive effect from DHA was estimated at 5.8 points. Shaded cells represent the estimates where the net effect on child IQ, using the upper-bound estimate for methylmercury, is negative.

Summary

Advice based on the analysis in Tables 3 and 4 would be functionally similar to current advice provided by New Zealand Food Safety, assuming that an acceptable outcome from consumption of seven fish servings per week is equated to no restriction necessary. A New Zealand project, currently nearing completion, analysed methylmercury and LCn3PUFA in samples of six fish species caught in New Zealand waters (Andrew Pearson, New Zealand Food Safety, personal communication). Analysis the same as that shown in Table 4 was

carried out for these six species and the results confirmed that no consumption restrictions were necessary for barracouta. Additionally, the analysis indicated that no consumption restrictions were necessary for gemfish, ling, orange roughy, oreo dories (black oreo) and smooth oreo.

While it is uncertain how relevant the data used in the FAO/WHO analysis are to fish caught in New Zealand waters. It is also uncertain whether a particular fish common name refers to the same species. Given these uncertainties, the FAO/WHO analysis suggests albacore tuna, bass, hake, hapuka, skate, snapper and sprats, which are currently classified as '3-4 servings per week acceptable' could be classified as 'no restriction necessary'. No species were identified for which the current New Zealand guidelines were too permissive.

Further New Zealand-specific information on the methylmercury and LCn3PUFA content of fish species would allow for further refinement of the New Zealand guidelines.

5.6 VEGETABLES, SALADS AND FRUITS

5.6.1 Fruit: All fresh fruits

CURRENT ADVICE:	Wash and dry well just before eating

Food

Fruits are the fleshy seed-associated structures of certain plants that are (usually) sweet and edible in the raw state.

Hazards

Listeria

Listeria spp. are commonly found in the soil and can contaminate fresh fruit in the environment, particularly types of fruit that grow or fall on the ground prior to harvest (Alegbeleye *et al.*, 2018). Soil can therefore be a source of *L. monocytogenes* on the surface of fresh fruit from home gardens and commercial growers. Packing facilities, equipment, water, and cold storage can also be sources of *L. monocytogenes* that sporadically contaminate fresh produce (Li *et al.*, 2018; Zhu *et al.*, 2017). Fruit such as canteloupes (rockmelon) with rough surfaces can be a particular problem as not all microorganisms may be removed during washing.

Overseas surveys have detected *L. monocytogenes* on fruit sampled at retail, including berries, avocados and tomatoes (Cabrera-Diaz *et al.*, 2018; FSANZ, 2010; Ponniah *et al.*, 2010). There are no published data for New Zealand.

Toxoplasma

T. gondii does not replicate outside the host so will not grow in food. If present in fresh produce, *T. gondii* is most likely to be in the oocyst (sporozoite) stage. No data on *T. gondii* prevalence on fresh fruit were located. *T. gondii* could be introduced through contact with soil (e.g. strawberries, melons) or contaminated irrigation water. Under experimental conditions, *T. gondii* oocysts inoculated onto raspberries and blueberries were still infectious (mouse bioassay) after eight weeks at 4ºC (Kniel *et al.*, 2002).

Human illness

In a recent study fruit was estimated to be responsible for 13.5% of produce-related outbreaks reported globally (Li *et al.*, 2018).

Fresh fruit has been implicated in several large listeriosis outbreaks. Canteloupes (rockmelon) from a single producer caused illness in 146 people across 28 states in the USA during 2011.[31] There were 33 deaths and a miscarriage recorded, with five subtypes of *L. monocytogenes* linked to the outbreak. Rockmelons were also the source of an Australian outbreak of listeriosis in early 2018 which included 22 cases and resulted in seven deaths and one miscarriage (Das, 2019).

In 2014, commercially prepared and packaged caramel apples were the source of a listeriosis outbreak which lead to 35 cases and 7 deaths across 12 states in the USA.[32] Eleven of the illnesses were pregnancy-related and there was one foetal loss. Three children (aged 5–15 years) developed meningitis. *L. monocytogenes* strains closely related to the outbreak strain were recovered from environmental sampling of the apple-packing facility where the Gala and Granny Smith apples had been sourced.

No outbreaks of listeriosis have been attributed to fresh fruit in New Zealand. There has been an outbreak of salmonellosis involving 19 cases, epidemiologically linked to consumption of watermelon purchased from a roadside stall (McCallum *et al.*, 2010). There has also been an outbreak of hepatitis A infection linked to fresh blueberries (Calder *et al.*, 2003), and clusters of hepatitis A cases linked to imported frozen berries.[33] Frozen berries have been the subject of numerous New Zealand and international recalls for viral contamination.[34]

There are no reported outbreaks of toxoplasmosis linked to whole or cut fresh fruit.

Controls

Raw fruit should be stored correctly in the fridge (i.e. covered, and not directly underneath raw meat, etc.) or in a separate place such as a clean bowl on the bench.

Fruit intended for eating raw should be washed with clean water (Shapiro *et al.*, 2019). Peeling will also reduce risk but cross-contamination from the skin to the edible parts is possible. Common household products such as detergents, antimicrobial soaps and bleach are not recommended for use on fresh produce (these are not effective at killing *T. gondii* oocysts) (Shapiro *et al.*, 2019).

When growing fruit at home that is close or in contact with the soil (e.g. strawberries), netting or other deterrents should be used to prevent cats from entering gardens. Gloves should also be worn when working with soil, and hands washed before picking fruit (Shapiro *et al.*, 2019).

Microorganisms will be killed when fruit are cooked (e.g. baking, stewing).

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[31] https://www.cdc.gov/listeria/outbreaks/cantaloupes-jensen-farms/index.html (accessed 4 May 2020).
[32] https://www.cdc.gov/listeria/outbreaks/caramel-apples-12-14/index.html (accessed 4 May 2020).
[33] Public Health AIDE 05 October 2016, Institute of Environmental Science and Research.
[34] For example, https://www.mpi.govt.nz/food-safety/food-recalls/recalled-food-products/fruzio-frozen-berries/ (accessed 14 May 2020).

Summary

Present New Zealand Food Safety advice is consistent with current scientific evidence. This is the only practical option for reducing any risk from pathogenic microorganisms when consuming fresh fruit, which are an important part of the diet. Advice might be added for (i) whole melons, advising that these are thoroughly washed and dried before cutting and (ii) frozen berries, advising these are cooked.

5.6.2 Vegetables: All fresh vegetables

CURRENT ADVICE:	Wash and dry well just before eating raw, or wash before cooking

Food

In culinary terms vegetables are usually defined as an edible plant, or part of a plant, other than a sweet fruit or seed.

Hazards

With the exception of *B. cereus*, *Clostridium botulinum* and *L. monocytogenes*, which are commonly found in soil, the presence of other pathogens is usually a result of faecal contamination originating from either humans or animals.

Listeria

Listeria can survive in soil under the right conditions and therefore can contaminate vegetables during cultivation (Alegbeleye *et al.*, 2018). Vegetables can also be contaminated by irrigation water, manure applied as fertiliser, and by animal faeces deposited in home and commercial gardens. As for fresh fruit, vegetables may also become contaminated in pack houses, during washing, and by equipment (Zhu *et al.*, 2017).

A survey of *L. monocytogenes* was undertaken over six weeks in vegetables, soil and water from three farms (98 samples in total), and from vegetables at retail in Canterbury, New Zealand (Zhu, 2015). No *L. monocytogenes* were detected in any of the samples, however many of the samples, including retail vegetables, contained non-pathogenic *Listeria* spp. showing that conditions supported survival of related species.

Surveys of vegetables in other countries have revealed that *L. monocytogenes* can be isolated from carrots, cabbages, lettuces, cucumbers, spinach and collard greens with prevalence ranging from 2% to 24.2% (Zhu *et al.*, 2017).

L. monocytogenes can multiply on some whole, fresh vegetables, on others the concentration remains stable, or it can decrease (King *et al.*, 2015).

Toxoplasma

T. gondii does not replicate outside the host so will not grow in food. If present on fresh produce, *T. gondii* is most likely to be in the oocyst (sporozoite) stage. Cats are known to deposit faeces in kitchen gardens, which is a potential source of contamination for home-grown vegetables (Bastien *et al.*, 2019). Contaminated irrigation water can also introduce this parasite to growing areas (Hussain *et al.*, 2017).

There are no data on *T. gondii* prevalence on fresh produce but surveys of vegetables in other countries have detected this parasite, most commonly on leafy vegetables (Shapiro *et al.*, 2019). For example:

- A survey of pre-packaged or bulk leafy green vegetables sampled from retail outlets in Canada found a prevalence of 0.26% among 1,171 samples tested by PCR (Lalonde and Gajadhar, 2016). The three positive samples were all baby spinach.
- *T. gondii* DNA was detected on 10/279 (4%) samples of fresh vegetables obtained from markets in China (Lass *et al.*, 2019). The positive samples were leafy vegetables: lettuce, spinach, pak choi, red cabbage and rape.
- A Polish survey of carrots, radishes and lettuce found molecular evidence of *T. gondii* in all three vegetable types, with an overall prevalence of 10% (21/216) (Lass *et al.*, 2019).
- *T. gondii* DNA was also detected in 8% (7/93) of carrots and (12%) (13/109) of cucumbers sampled from the fields and storage facilities in the Czech Republic (Slany *et al.*, 2019).

The above examples do not demonstrate the presence of viable *T. gondii* but it is likely that the molecular methods were detecting environmentally resilient oocysts.

Human illness

Outbreak data summarised for New Zealand showed only two outbreaks where the epidemiological evidence implicated fresh vegetables as the vehicle of disease (King *et al.*, 2015). These were an outbreak of salmonellosis linked to carrots, and an outbreak of yersiniosis linked to lettuces and/or carrots. Fresh vegetables have not been identified as the source of any outbreaks of listeriosis in New Zealand.

Overseas, fresh vegetables have been the cause of numerous foodborne outbreaks (King *et al.*, 2015). For example, in 2015 a large outbreak (907 cases) of *Salmonella* Poona in the USA was linked to cucumbers imported from Mexico (Kozyreva et al., 2016).

Listeriosis outbreaks associated with fresh vegetables are not commonly reported. In 2010, ten patients in a hospital in Texas were infected with *L. monocytogenes* from diced celery, and five patients died. The outbreak strain was sourced back to environmental samples and additional celery samples collected at the processing plant (Gaul *et al.*, 2013). Enoki mushrooms from South Korea have been the source of a recent food safety alert due to *L. monocytogenes*. In the USA, mushrooms have been linked to 36 cases of listeriosis across 17 States and have resulted in 4 deaths.[35] Australia has also had six cases linked to the same outbreak and the products have been recalled.[36]

There are no reported outbreaks of toxoplasmosis in New Zealand where fresh produce was the implicated vehicle of infection. Outbreaks caused by fresh vegetables have been reported overseas.

During 2009, a cluster of acute toxoplasmosis cases was identified in a factory in Brazil (Vielmo *et al.*, 2019). A case control study enrolled 8 acute and 3 asymptomatic cases (all with serological evidence of a current or recent infection), plus 20 controls (seronegative). There were two significant risk factors for infection, being consumption of escarole ($P<0.05$, but only two cases and no controls consumed this food) and consumption of “green vegetables” ($P<0.005$). A second outbreak has been reported in Brazil, also implicating vegetables (Pinto-Ferreira *et al.*, 2019b). The outbreak occurred at a research institute

[35] https://www.cdc.gov/listeria/outbreaks/enoki-mushrooms-03-20/index.html (accessed 6 May 2020).
[36] https://www.foodstandards.gov.au/industry/foodrecalls/recalls/Pages/Green-Co.-Enoki-Mushrooms-200g%20and%20300g.aspx (accessed 6 May 2020).

during 2015/16. A case control study, involving 20 cases and 45 controls, found that consumption of vegetables was the only significant risk factor (OR 14.7, 95%CI 2.4–333.7). Environmental and food sampling did not detect *T. gondii* DNA.

Controls

See fresh fruit; similar controls apply.

Raw vegetables should be stored correctly in the fridge (i.e. covered, and not directly underneath raw meat, etc.), particularly salad vegetables as they are likely to be eaten raw.

Summary

Present New Zealand Food Safety advice is consistent with current scientific evidence. Washing vegetables to be eaten raw is the only practical option for reducing any risk from pathogenic microorganisms when consuming fresh vegetables, which are an important part of the diet. The advice to wash vegetables before cooking provides a level of protection for fresh vegetables that are only lightly cooked (e.g. blanched), when the cooking temperature/time may not be sufficient to inactivate any pathogenic microorganisms.

5.6.3 Vegetables: Frozen vegetables

CURRENT ADVICE:	Cook; don't eat uncooked frozen vegetables

Food

Prior to packaging and freezing, most vegetables are washed, chopped if required, and blanched (times and temperatures vary) (EFSA BIOHAZ Panel *et al.*, 2020). Blanching is a technological heat treatment applied for enzymatic inactivation.

Hazards

Growth of *L. monocytogenes* in frozen vegetables is not expected but it may survive if the product is contaminated prior to freezing. Pathways to contamination are the same as described for fresh vegetables above (Section 5.6.2) prior to processing. The time/temperature combinations for blanching and cooling are particularly important if the frozen product is eaten without further cooking (EFSA BIOHAZ Panel *et al.*, 2020).

Frozen vegetables imported from Belgium and the UK into New Zealand were part of an international food safety recall in 2018.[37]

There are no data for *T. gondii* on frozen vegetables. There is potential for viable oocysts to be present but the combination of washing, blanching and freezing make this unlikely.

Human illness

Traditionally considered to be a lower risk food, frozen vegetables have been the source of outbreaks of listeriosis. Between 2013 and 2016 nine cases, including three deaths, were associated with frozen vegetables from a single producer in the USA, but were sold under different brand names.[38] The cases occurred across four States and were linked through molecular typing and sequencing techniques.

[37] https://www.mpi.govt.nz/food-safety/food-recalls/recalled-food-products/various-frozen-vegetable-products/ (accessed 6 May 2020).
[38] https://www.cdc.gov/listeria/outbreaks/frozen-vegetables-05-16/index.html (accessed 6 May 2020).

Blanched frozen vegetables were responsible for an outbreak of *L. monocytogenes* during the period 2015–2018 that affected multiple countries, beginning in the EU (EFSA BIOHAZ Panel *et al.*, 2020). There were 53 cases linked to the outbreak, with 10 deaths. The cause was environmental contamination at the freezing plant by a persistent strain of *L. monocytogenes*.

Controls

The blanching process is variable, depending on the product. Blanching at temperatures ≥75ºC inactivate *L. monocytogenes* on various vegetables, given sufficient time (EFSA BIOHAZ Panel *et al.*, 2020). Storage at -18°C prevents bacterial growth. In consumer homes, the most important control is cooking.

Summary

Present New Zealand Food Safety advice is consistent with current scientific evidence. Cooking frozen vegetables prior to consumption will inactivate any *L. monocytogenes* present.

5.6.4 Salads: Ready-made salads and coleslaws from delis, salad bars, etc.

Vegetable-based salads, pasta salads, rice salads, fruit salads, etc.

CURRENT ADVICE:	Don't eat

Food

This sub-group contains a wide variety of salad types, including pasta, rice and vegetable salads (including coleslaw), which may also contain dressing, meat or seafood, and also fruit salads. The fresh produce is usually cut in some way and these cut surfaces can provide a suitable place for microorganisms to survive or grow. The presence of proteins (e.g. meat, nuts, cheese and eggs) and dressings can enhance or suppress microbial growth.

Hazards

Listeria

Historically, *L. monocytogenes* has been of primary concern in salads due to its ability to grow at refrigeration temperatures and the variety of ingredients that are used. One of the first recognised outbreaks of listeriosis was associated with vegetable mix for coleslaw in Canada in 1981 (Zhu *et al.*, 2017). *L. monocytogenes* and other *Listeria* spp. have been isolated from mixed salads and pre-packed salads overseas (Little *et al.*, 2007; Woodward, 2018).

In New Zealand, a survey of retail ready-to-eat salads with dressings was undertaken to determine the prevalence of *L. monocytogenes* (Gilbert *et al.*, 2010). Ready-to-eat salad samples (n=302) were selectively purchased from four main cities in New Zealand (Auckland, Wellington, Christchurch and Dunedin). The salads tested were bean, pasta, potato, pulse/seed, rice, seafood-based, coleslaw and miscellaneous (i.e. varieties outside of these descriptions). Salads under the various designations may have contained small amounts of cooked meats, cooked eggs, spices and fresh herbs. *L. monocytogenes* was found in 14 samples (prevalence 4.6%, CI 2.6–7.7). One sample of coleslaw contained 100 CFU/g of *L. monocytogenes* while another contained 30 CFU/g. The remaining 12 positive samples contained <10 CFU/g of *L. monocytogenes*.

A survey of 307 pre-packaged fresh leafy salads in New Zealand was performed in 2012 (Hewitt and Rivas, 2015). Included in the survey were products containing lettuce (green, fancy, cos, iceberg), baby spinach, watercress, mesclun, rocket, brassica, mixed, and unspecified lettuce salad. Products were purchased from retailers over a one year period in three major cities. No *L. monocytogenes* was detected in any of the products tested, however, *Listeria* spp. were identified in 6.2% of the products (19/307, 95% CI 3.8–9.5%).

L. monocytogenes can also grow in fresh-cut fruit salads depending on the overall acidity (Ziegler *et al.*, 2018). Fruit salads with less acidic fruits such as melons, fresh coconut and mangos are more likely to support growth. Retail fresh-cut fruit salads (ready-to-eat, non-retorted) were surveyed in New Zealand during 2013/14 (D'Sa and Hudson, 2014). The products were purchased at supermarkets or from online retail stores and 75 composite samples were tested. *L. monocytogenes* was detected in four samples (5.3%) at concentrations <100 CFU/g. Three of the positive samples were melon and the fourth sample was mixed fruit salad also with melon.

Packaged fresh spinach and salad greens have been the subject of food safety recalls in New Zealand due to potential contamination with *L. monocytogenes* above regulatory limits.[39]

Toxoplasma

See fresh vegetables (Section 5.6.2) and fresh fruit (Section 5.6.1). *T. gondii* is unlikely to be present in cereal-based salads (Section 5.1.4) but could be introduced into any salad with cooked or processed meat (Sections 5.4.2 and 5.4.3) or fresh produce grown close to the ground.

There are no New Zealand data on the prevalence of *T. gondii* in salads. In Italy, *T. gondii* DNA was detected on 5/648 (0.8%) packages of ready-to-eat salad at concentrations in the range 62–554 oocysts/g (as estimated by quantitative PCR) (Caradonna *et al.*, 2017).

Human illness

There have been no reported listeriosis outbreaks in New Zealand linked to ready-made salads. An outbreak of norovirus infection in New Zealand during 2011, involving 31 cases, was epidemiologically linked to fresh fruit salad served in a restaurant that was most likely contaminated by an infected food handler (summarised in King *et al*. 2015).

A listeriosis outbreak involving 33 cases in the USA and Canada was associated with pre-packaged leafy green salads (Self *et al.*, 2019). Also in the USA, a salmonellosis outbreak occurred in 2019, which involved 165 cases and was associated with consumption of fresh-cut fruit salad (honeydew melon, cantaloupe, pineapple, and grapes) (USCDC, 2020).

Controls

Sanitisers are used by the produce industry to prewash pre-packaged salads, and for post-harvest processing of some whole fruits and vegetables (King *et al.*, 2015). Ready-made salads, by their nature, do not require the consumer to take any further steps before consumption.

[39] https://www.mpi.govt.nz/food-safety/food-recalls/recalled-food-products/ (accessed 23 March 2020).

Summary

Present New Zealand Food Safety advice is consistent with current scientific evidence in reference to ready-made salads from delicatessens, salad bars, etc. *L. monocytogenes* can be present and grow in these products. However, the scientific evidence also shows the potential for pre-packaged leafy green salads to cause listeriosis. A protective approach could be to expand the food description to make it clearer that pre-packaged salads are included.

5.6.5 Salads: Home-made

Vegetable-based salads, pasta salads, rice salads, fruit salads, etc.

CURRENT ADVICE:	Wash salad ingredients well before using

Food

See salads (Section 5.6.4).

Hazards

See salads (Section 5.6.4). *L. monocytogenes* and *T. gondii* could both be present on salad vegetables or fruits, or protein-based foods added to salads.

Human illness

No reports of listeriosis outbreaks linked to home-made salads were located for New Zealand. A home-made rice salad was the likely vehicle of infection in a listeriosis outbreak in Italy, with the *L. monocytogenes* possibly being introduced through cross-contamination (Salamina *et al.*, 1996). *B. cereus* intoxication from a temperature abused home-made pasta salad has caused an outbreak, with one fatality (Dierick *et al.*, 2005).

Controls

Sanitisers are used by the produce industry to prewash pre-packaged salads, and for post-harvest processing of some whole fruits and vegetables (King *et al.*, 2015). In the consumer's home, washing fresh produce with water before cutting, not using damaged fruit or vegetables, ensuring good food safety practices in preparing cooked ingredients, and keeping salads cold after preparation will mitigate risk. See also fresh fruit (Section 5.6.1) and fresh vegetables (Section 5.6.2).

Summary

Present New Zealand Food Safety advice is consistent with current scientific evidence although this only applies to salads made from fresh produce. Additional advice might be added to promote hygiene and temperature controls around preparation of proteinaceous or farinaceous ingredients.

5.6.6 Herbs: Fresh home-grown and store-bought

CURRENT ADVICE:	Wash well before using

Food

Herbs are plants, valued for their flavour, aromatic properties and other qualities. Herbs are mostly used in cooking and medicines. Fresh herbs have not been dried.

Hazards

As with fresh fruits and vegetables above, herbs have the potential to become contaminated via growing practices or handling.

Listeria

Herbs can become contaminated by *Listeria* in the environment, and during processing and packaging, as described for vegetables above. However, the prevalence of *L. monocytogenes* naturally found associated with herbs appears to be low. *L. monocytogenes* was not found in a survey of 60 samples of hydroponically-grown herbs from New Zealand producers (Graham and Dawson, 2002). A meta-analysis of studies in Europe reported a pooled prevalence of *L. monocytogenes* in 1.063% (95% CI 0.429─2.608) of spices and herbs, either packaged or unpackaged, at retail (Nunes Silva *et al.*, 2017).

Inoculation studies suggest that *Listeria* can be transferred from soil to the leaf surface of herbs such as parsley during rain or irrigation (Girardin *et al.*, 2005). *L. monocytogenes* inoculated onto the surface of parsley through irrigation water did not survive very long, with levels declining by several logs within two days of application (Dreux *et al.*, 2007). Under conditions of high (100%) relative humidity the levels of *L. monocytogenes* on parsley leaves remained higher for longer.

Fresh Italian parsley grown in New Zealand was recalled in 2015 for containing *L. monocytogenes* above regulatory limits[40]

Toxoplasma

T. gondii does not replicate outside the host so will not grow in food. If present on fresh herbs, *T. gondii* is most likely to be in the oocyst (sporozoite) stage. Cats are known to deposit faeces in kitchen gardens, which is a potential source of contamination for home-grown herbs (Bastien *et al.*, 2019). Contaminated irrigation water can also introduce this parasite to growing areas (Hussain *et al.*, 2017).

There are no New Zealand data on *T. gondii* prevalence on fresh herbs. *T. gondii* has been detected on fresh parsley during a survey of vegetables in Brazil (Marchioro *et al.*, 2016).

Human illness

There have been no reported outbreaks of listeriosis associated with fresh herbs in New Zealand.

There are no reported outbreaks of toxoplasmosis in New Zealand where fresh herbs were implicated as a vehicle of infection.

Controls

See vegetables (Section 5.6.2). Washing, correct storage and cooking are the only practical control options for consumers.

Summary

Present New Zealand Food Safety advice is consistent with current scientific evidence. As for fresh vegetables, washing fresh herbs to be eaten raw is the only practical option for reducing any risk from pathogenic microorganisms.

[40] https://www.mpi.govt.nz/food-safety/food-recalls/recalled-food-products/ (accessed 23 March 2020)

5.6.7 Herbs: Dried

CURRENT ADVICE:	(not specifically included)

Advice considered in Gilbert *et al.* (2010) was “Cook thoroughly.”

Food

See Herbs (Section 5.6.6). Spices are the dried (often ground) parts of plants that are also used to add flavour and aromatics too food, e.g. pepper, cumin, cloves, turmeric. These are also discussed in this section. Dried herbs and spices can be added to food before or after cooking, or foods that are not cooked.

Hazards

The microorganisms associated with dried herbs are mainly those that become associated with the plant during growth (e.g. soil microbes) and subsequently survive the drying process (ICMSF, 2005). These are most likely to be spore forming bacteria and moulds, but pathogens such as *Salmonella* spp., *B. cereus* and *C. perfringens* may also be present. Surveys and outbreak data show that these are the most important pathogens associated with dried herbs and spices (FAO/WHO, 2014). For example, a survey of 2,680 dried herb samples in Canada only detected *B. cereus* in two herb samples (0.07%, 2/2680), *S. aureus* at elevated levels in one herb sample (0.06% 1/1773) and *Salmonella* spp. in 1 herb sample (0.04%, 1/2680) (CFIA, 2019). A 2014 review found that *L. monocytogenes* had not been detected in dried herbs and spices, although there were relatively fewer studies testing for this bacterial species relative to other pathogenic species (FAO/WHO, 2014). No additional data were located.

If present, growth of *L. monocytogenes* is not expected in dried herbs and spices. There are no data to show whether *T. gondii* oocysts could be found on dried herbs and spices. Oocysts are naturally resistant to dry environments to ensure their survival in the environment, but will not multiply in food.

Human illness

There are no reported outbreaks of listeriosis or toxoplasmosis where dried herbs or spices were implicated as a vehicle of infection.

Outbreaks of salmonellosis, and intoxication from *B. cereus* or *C. perfringens* have been reported (FAO/WHO, 2014). For example, a *Salmonella* Montevideo outbreak (272 cases) occurred in the USA in 2010 and was attributed to black and red pepper used in the manufacture of Italian-style meat products (USCDC, 2010).

Controls

Herbs and spices may be treated to reduce microbial concentrations e.g. using irradiation, steam pasteurisation or chemical treatments (FAO/WHO, 2014).

Cooking is the most effective method of pathogen control at consumer level.

Summary

The advice considered in Gilbert *et al*. (2010) was “Cook thoroughly.” Current scientific evidence continues to support this advice. Current data suggests that *L. monocytogenes* is not a common contaminant of dried herbs and spices, although a New Zealand survey would provide a more robust evidence base. Such data would support the development of advice

for food servings where the herbs or spices will not be subjected to cooking, e.g. turmeric- or cinnamon-based hot and cold drinks, dukkah, cracked pepper.

5.7 MISCELLANEOUS

5.7.1 Leftovers: Cooked foods

CURRENT ADVICE:	Store leftovers covered in fridge and eat within two days; reheat leftovers until piping hot (over 70ºC); never eat cold leftovers

Food

Relevant to all foodstuffs. Considerations as for cooked meat, poultry and seafood apply.

Hazards and Controls

The growth rate of *L. monocytogenes* under refrigeration depends on the food type. Adequate reheating will destroy any *L. monocytogenes* that may be present in the food. *L. monocytogenes* is inactivated at temperatures above 70°C, depending on the properties of the food.

Summary

Present New Zealand Food Safety advice is consistent with current scientific evidence. It is difficult to determine a suitable time limit for keeping leftovers because of the range of foods and their potential for *L. monocytogenes* contamination and growth under consumer refrigeration conditions. The current two-day advice provides a practical compromise to minimise bacterial growth and the reheating step provides a final control step.

5.7.2 Canned foods

Canned fruit, vegetables, fish, seafood, meat, sauces, etc.

CURRENT ADVICE:	Remove from can for storage; store uneaten leftovers covered in fridge and eat within two days

Food

Canned (or tinned) foods are commercially sterile, shelf-stable products that have undergone a heat treatment to ensure the destruction of all viable organisms. In New Zealand, a canned product is one that is packed in hermetically sealed packaging and is heat treated before or after sealing in the packaging (MPI, 2018). These retorted foods can be packaged in cans, glass jars, trays and pouches. The heat treatment depends on the food, with the acidity being particularly important.

Hazards

No survival or growth of *L. monocytogenes* (or other pathogens) is expected in unopened canned product. Growth in leftovers depends on the type of food.

Surveys/Outbreaks

Canned foods have occasionally caused outbreaks of botulism (i.e. due to the presence and outgrowth of surviving *C. botulinum* spores when the canning process fails). No reports of outbreaks of listeriosis linked to canned foods were located involving food contaminated prior to opening.

Controls

Refrigerated storage of canned leftovers reduces possibility of pathogen growth. Once opened, canned foods are not usually able to be sealed again for storage and are best shifted to clean containers with good sealing lids for refrigerated storage. This helps prevent cross-contamination.

Summary

Present New Zealand Food Safety advice is consistent with current scientific evidence. It is difficult to determine a suitable time limit for keeping leftovers because of the range of foods and their potential for *L. monocytogenes* contamination and growth under consumer refrigeration conditions. The current two-day advice provides a practical compromise to minimise bacterial growth in canned foods which may not be heated, e.g. canned fruit. However, the current advice does not specifically provide for canned foods that might be heated (not cooked) and not eaten immediately, e.g. leftover soup, baked beans. The advice on leftovers (Section 5.7.1) could be expanded to include these canned foods, e.g. by modifying the food description to “Leftovers: Cooked or heated foods”.

5.7.3 Sauces, dressings and spreads

Salad dressings (oil and vinegar), bought mayonnaise, tomato sauce, margarine-type spreads, etc.

CURRENT ADVICE:	Store in the fridge once opened; check maximum storage time

Food

This is a wide food group that encompasses products that are shelf-stable (until opened) and refrigerated.

Mayonnaise is an emulsion of oil and an acidifier, such as lemon juice or vinegar, stabilised by egg yolk or whole egg. Other ingredients can be added, such as herbs, salt, sugar, spices, vegetable or dairy products. Other salad dressings are also usually based on an oil/acid emulsion and can have a range of ingredients, although these foods generally have less fat/oil compared to mayonnaise. The presence of acidifying ingredients lowers the pH of mayonnaise and other salad dressings.

Shelf-stable sauces (e.g. tomato sauce, mint sauce) and refrigerated sauces (e.g. fresh pasta sauce) are preserved through heat treatment and other hurdles such as low water activity, low pH and salt.

Margarine and other edible oil spreads are water and oil emulsions that may have other added ingredients including proteins, salt, milk products, and microorganisms used for producing lactic acid or flavour. Margarine is defined as an edible oil spread containing no less than 800 g/kg of edible oils.[41] Margarine is produced through a process of pasteurisation, crystallisation and setting (Vatankhah *et al.*, 2020).

Hazards

Inoculation studies in seven different yellow fat spreads, including margarine, dairy/non-dairy spreads, and butter-margarine spreads found that *L. monocytogenes* did not grow in any

[41] Australia New Zealand Food Standards Code (Standard 2.4.2), https://www.legislation.gov.au/Series/F2015L00461 (accessed 19 May 2020).

product incubated at 4.4°C, 10°C and 21°C for up to 94 days, except the butter-margarine blend (Holliday and Beuchat, 2003). However, if contaminated, the products may allow the survival of *L. monocytogenes* at refrigeration temperatures for at least a month, and in some products longer. In a related challenge study it was shown that the pH and level of preservative within yellow fat spreads contributed to the inhibition of *L. monocytogenes* growth, particularly when incubated at 21°C (Holliday *et al.*, 2003).

The concentration of *L. monocytogenes* decreased in commercially produced, shelf stable, ranch and blue cheese salad dressings (mayonnaise-based dairy products) held at 25ºC (Beuchat *et al.*, 2006). Initial pH values of the dressings were between 2.8 and 3.8. Samples were inoculated with low (2.5 $\log_{10}$ CFU/g) and high (5.3 $\log_{10}$ CFU/g) concentrations of *L. monocytogenes*. The *L. monocytogenes* concentration fell to undetectable levels within 8 days. Reductions of >1.4 $\log_{10}$ CFU/ml and >4.3 $\log_{10}$ CFU/ml occurred within 3 and 6 days respectively. Both low and high fat versions of the dressings were included in the survey and, regardless of inoculum concentration, inactivation of *L. monocytogenes* appeared to be largely unaffected by composition.

No surveys of *L. monocytogenes* in sauces, mayonnaise, dressings or spreads were located.

Human illness

Listeriosis outbreaks have been associated with mayonnaise-based salads, where the complex food has been contaminated (Section 5.6.4). In contrast, outbreaks of salmonellosis have occurred where the mayonnaise was the contaminated ingredient. Almost all of these outbreaks have been associated with the use of contaminated eggs in product prepared in the home, restaurants or institutions. No recent outbreaks implicating shelf-stable mayonnaise were located.

No listeriosis outbreaks were located associated with edible oil spreads, sauces or dressings.

Controls

Pasteurisation provides a control step during production (Vatankhah *et al.*, 2020). In general the high fat, low pH or acidic nature of many of these products is inhibitory to the growth and survival of *L. monocytogenes*. However, low fat versions may support the survival or growth of *L. monocytogenes* if they are not acidified sufficiently (Hwang *et al.*, 2019). The addition of salt and preservatives may also inhibit the survival and growth of *L. monocytogenes*.

The high fat content of edible oil spreads provides a barrier to microbial growth (Delamarre and Batt, 1999).

Summary

Present New Zealand Food Safety advice is consistent with current scientific evidence. *L. monocytogenes* is not expected in properly formulated commercial product, although survey data are lacking. The product formulations also make it unlikely that *L. monocytogenes* will grow if this bacterium contaminates these foods after opening, although this depends on the product pH and other inhibitory ingredients (e.g. preservatives).

Refrigerated heat-and-eat pasta sauces are now widely available to consumers as an alternative to shelf-stable canned pasta sauces. Some of these could support *L. monocytogenes* growth but any vegetative cells will be inactivated with the expected heat

treatment in the consumer's home. Data on *L. monocytogenes* prevalence and growth are required to develop specific advice related to these products. In the interim, the current advice might be altered to encourage consumers to handle any heat-and-eat sauce products according to the manufacturer's instructions.

5.7.4 Sushi: Store-bought (all types – even without raw seafood)

CURRENT ADVICE:	Don't eat

Food

Sushi is made with vinegared rice, nori (seaweed) with fillings including vegetables, cooked meat, fish or tofu, raw fish, raw shellfish, and/or sauces such as mayonnaise.

Hazards

See raw fish (Section 5.5.1), raw shellfish (Section 5.5.2), smoked fish (Section 5.5.3), cooked meat (Section 5.4.2), cold cooked poultry (Section 5.4.4), sauces (Section 5.7.3), and vegetables (Section 5.6.2).

The manual nature of sushi making may result in food handler-related cross-contamination. This could occur directly via an infected food handler, or via inadequate hand washing after handling raw materials such as seafood.

Listeria

There are no New Zealand data for *L. monocytogenes* in sushi. Overseas, *L. monocytogenes* has been isolated during surveys of sushi or sashimi from restaurants and other retail outlets. A survey of freshly prepared sushi from sushi bars and frozen sushi from supermarkets was undertaken in northern Germany (Atanassova *et al.*, 2008). Samples (n=250, 125 fresh and 125 frozen) represented typical products on menus and at retail. *Listeria monocytogenes* was isolated from three samples (2.4%) of fresh sushi, but was not isolated from any frozen sushi samples.

The New South Wales (NSW) Food Authority undertook a survey of 89 retail sushi outlets across NSW during 2006/2007 (NSW Food Authority, 2008). *L. monocytogenes* was detected in 2.9% (13/446) of samples collected during winter months and in 3.2% (13/404) summer samples. The mean pH of 73 rice samples was 4.4 (range 3.8–6.8).

Samples (n=300) of sushi were tested from five restaurants and one supermarket in Turkey (Cadun *et al.*, 2016). Popular types of sushi were analysed including take roll, sesame roll, California roll and sake roll. The pH of the rice in all samples was below 4.6 and no *L. monocytogenes* was detected in any of the samples.

Human illness

No outbreaks of listeriosis have been attributed to consumption of sushi in New Zealand.

In 2015, an outbreak (64 cases) of *Salmonella* Paratyphi B was recorded in the USA associated with consuming sushi containing raw tuna imported from a typhoid endemic country (Indonesia) (USCDC, 2015).

Controls

Reducing the pH of the rice to below pH 4.6 may be used to slow or prevent the growth of bacteria. Refrigeration is recommended.

Summary

Present New Zealand Food Safety advice is consistent with current scientific evidence. While there are no New Zealand data on the prevalence of *L. monocytogenes* in sushi, surveys overseas have demonstrated this bacterial species can be found. There is potential for *L. monocytogenes* to grow, depending on the ingredients, pH and storage temperature.

5.7.5 Sushi: Home-made

CURRENT ADVICE:	Use freshly cooked rice, and don't use raw or cold cooked meat or seafood; eat immediately; don't eat leftovers

Food

See sushi (Section 5.7.4).

Hazards

There is no specific data available for home-made sushi. The hazards depend on the ingredients selected by the consumer and how these are prepared.

Controls

The consumer is responsible for undertaking proper cooking/cooling and hygiene controls for the rice and sushi ingredients.

Human illness

No surveys or outbreaks associated with home-made sushi were located.

Summary

The current evidence is not sufficient to evaluate New Zealand Food Safety advice. However, the advice is protective. The advice might be amended to advise against using smoked seafood.

5.7.6 Stuffing

Stuffing from chicken or turkey

CURRENT ADVICE:	Don't eat unless stuffing is cooked separately (in a dish); eat hot; store leftovers in fridge and eat within two days; reheat leftovers until piping hot (over 70ºC)

Food

Stuffing is a seasoned mixture of ingredients used to fill cavities in meat and vegetables. It is usually a breadcrumb-based mix stuffed into poultry (e.g. chickens or turkeys). Bread (Section 5.1.1) stuffing may include ingredients such as vegetables (Section 5.6.2), herbs (Section 5.6.6) or fruit (Section 5.6.1). Less traditional recipes may include meat, sausage (Section 5.4.1) or shellfish (Section 5.5.2).

Hazards

Stuffing may become contaminated with pathogens from ingredients or through cross-contamination from the raw poultry. Stuffing placed into the cavity of a roasting bird prior to cooking must reach adequate cooking temperatures to achieve sufficient pathogen

reductions. Stuffing cooked inside the bird may also be associated with long cooling times which, combined with the low oxygen environment, is favourable for the survival of some pathogens, e.g. *C. perfringens* (Woodburn and Kim, 1966).

A UK survey of ready-to-eat stuffing (n=147), prepared as a separate food product, did not detect *Campylobacter* spp. or *Salmonella* spp. but some samples contained elevated levels of microbes associated with poor food practices, e.g. *E. coli* and *S. aureus* (Richardson and Stevens, 2003). The survey did not test for *L. monocytogenes*.

No data were located on the prevalence and potential for growth of *L. monocytogenes* in stuffing.

Human illness

No reported listeriosis outbreaks were located related to stuffing. Occasional reports of other foodborne diseases occur associated with consumption of stuffed meats. For example, an outbreak of salmonellosis (*S.* Enteritidis) in the USA, involving seven cases including one death, was attributed to undercooked home-roasted turkey with stuffing containing eggs (Ravenholt *et al.*, 1996). In France, 17 hepatitis E cases (5 symptomatic) were linked to consumption of roasted piglet prepared with pork liver stuffing (Guillois *et al.*, 2015).

Controls

It is generally considered good practice to cook stuffing separately, allowing the bird and the stuffing to each achieve adequate cooking temperatures.

If stuffing is cooked within a roasting bird or rolled meat, adequate internal temperature for sufficient time must be reach to inactivate any pathogens. After monitoring the time-temperature histories and cooking times for turkeys oven roasted at 162.8°C, the slowest heat point was found to be the stuffing geometric centre and the wing joint (Chang *et al.*, 1998). The temperature profile of the meat started at 4.44°C and the endpoint was 82.2°C in the thigh joint and breast. Turkeys were divided into five weight classes and divided into fresh, frozen, stuffed, unstuffed and cooked shielded or unshielded (single piece of aluminium foil loosely over breast). Shielding with aluminium foil prolonged cooking times. A meat thermometer inserted into the stuffing and monitored throughout cooking ensures an adequate temperature is reached.

Summary

Present New Zealand Food Safety advice is consistent with current scientific evidence. Cooking the stuffing separately ensures that all vegetative microbes are destroyed. The advice might be altered to include stuffing from red meat roasts. Stuffed, hot-held, ready-to-eat whole poultry are readily available in New Zealand supermarkets. A survey of the microbial quality of these products would support development of advice to pregnant women considering purchasing these foods.

5.7.7 Hummus and tahini

Store-bought or home-made

CURRENT ADVICE:	Don't eat

Food

Hummus, a traditional and popular Middle Eastern food, is made from cooked chickpeas with tahini (sesame paste), garlic, lemon juice or citric acid, and seasonings. Other varieties are available with additional ingredients. It is eaten as a spread or dip without further cooking. Hummus has a high water activity and pH in the range 5–7 (Al-Holy *et al.*, 2006). Lower pH values have been reported; unpublished data in Gilbert *et al.* (2010) showed that the pH of hummus sampled from New Zealand all fell within the range 3.9–4.5.

Tahini is produced from roasted, ground sesame seeds (Lake *et al.*, 2010). As well as a key ingredient in hummus, tahini can be used alone as a spread or an ingredient in other condiments (e.g. salad dressings, sauces) or confectionary (e.g. halva).

Hazards

Based on outbreak data (below), the main microbiological hazard associated with hummus and tahini is *Salmonella* spp. However, hummus can be a suitable environment for a range of microbial pathogens to survive and grow.

Salmonella

Salmonella spp. were not detected in samples of imported tahini (n=169), hummus (powdered, n=10) or halva (n=77) in a survey of products sold in New Zealand during 2001 (Wong *et al.*, 2003).

At 4 or 10ºC, *Salmonella* spp. survived but did not grow in experimentally inoculated hummus (pH 4.50–4.52) (Alali *et al.*, 2012).

Listeria

A 1999 survey in New Zealand did not detect *L. monocytogenes* in 50 samples (from 10 batches) of commercially prepared hummus (Wilson, 2000). Measurement of pH was not included as part of the survey but random samples tested showed pH values in the range 4.7–5.2.

The 2001 survey of tahini, hummus and halva (above) also tested for *L. monocytogenes*, but did not detect this pathogen (Wong *et al.*, 2003).

A large study in Canada did not detect *L. monocytogenes* in 2,400 tahini samples.[42]

At 4 or 10ºC *L. monocytogenes* can survive, but not grow in experimentally inoculated hummus (Alali *et al.*, 2012). The products tested had pH values in the range 4.50–4.52 and this acidity was the main control. Experiments with *L. innocua* indicate that *Listeria* spp. could grow in tahini if the moisture level is high enough (Anas *et al.*, 2013).

[42] https://www.inspection.gc.ca/food-safety-for-industry/chemical-residues-microbiology/microbiology/salmonella-listeria-monocytogenes-and-generic-e-co/eng/1470417503143/1470417529128 (accessed 1 May 2020).

Human illness

A USA case-control study, enrolling sporadic cases of confirmed listeriosis, identified two food-related risk factors for illness, these being eating hummus or eating melons prepared at a commercial establishment (Varma *et al.*, 2007). Cases were 5.7 times more likely to have eaten hummus prepared at a commercial establishment than controls ($p<0.01$) (the odds ratio for melons was 2.6).

No peer reviewed reports of listeriosis outbreaks linked to hummus or tahini were located, but other reports show that outbreaks have occurred.[43]

An outbreak of salmonellosis in New Zealand during 2003, involving 10 cases, was caused by imported tahini (Unicomb *et al.*, 2005). Imported tahini was again implicated in a salmonellosis outbreak (27 cases) in New Zealand during 2012 (Paine *et al.*, 2014). Three *Salmonella* serotypes (Montevideo, Mbandaka, Maastricht) were identified in the paste and the product was recalled by the importer.

There have been several New Zealand food recalls issued for potential contamination of hummus or tahini with *Salmonella* spp. A 2018 recall was connected to products linked to outbreaks in other countries.[44,45]

Controls

If low enough, the pH of hummus will inhibit growth of *L. monocytogenes* and *Salmonella* spp.

Tahini and crushed sesame seeds (and any products containing them) are 'foods of regulatory interest' and their importation is subject to New Zealand Food Safety clearance requirements, which includes evidence that the product is not contaminated with *Salmonella* spp.

Summary

Present New Zealand Food Safety advice is consistent with current scientific evidence, on the basis of this being a high risk product for salmonellosis. Growth of *L. monocytogenes* is not expected in properly formulated commercial product but may occur in home-made hummus where the acidity is less controlled. More recent prevalence data on *Salmonella* spp. and *L. monocytogenes* in tahini and hummus, including *L. monocytogenes* enumeration, would provide a better set of evidence to develop advice for commercial products. In the interim, the current advice is protective.

[43] https://www.foodsafetynews.com/2019/11/long-term-listeria-outbreak-solved-in-denmark/ (accessed 1 May 2020).
[44] https://www.mpi.govt.nz/food-safety/food-recalls/recalled-food-products/achva-brand-and-la-mamma-brand-tahini/ (accessed 14 May 2020).
[45] https://www.foodsafetynews.com/2018/12/israel-reports-cases-in-salmonella-tahini-outbreak/ (accessed 14 May 2020).

5.7.8 Seaweed: Brown

Brown seaweed** (i.e. kelp, kombu, wakame, arame, quandai-cai, hiziki/hijiki, or *Sargassum fusiforme*)

CURRENT ADVICE:	Limit to 1 serve per week

**Brown seaweeds contain naturally very high iodine concentrations. Brown seaweeds are typically sold dry and are used in soups, stewed dishes, kelp salt and seaweed salads.

Food

Brown algae (Phaeophyceae) include a number of species of seaweed (macroalgae) (Banach *et al.*, 2020). There are a range of edible species of brown seaweeds with common names such as hijiki and wakame. China and Indonesia are the countries where the majority of global supply is produced for human consumption and other uses. Products can be fresh, fresh frozen and dried.

Hazards

Seaweeds accumulate elements from their environment and these can present chemical hazards to humans. These hazards include iodine (although essential for humans and animals excessive iodine can cause adverse health effects) and metals such as arsenic, cadmium, lead and mercury (Banach *et al.*, 2020).

A recent report has collated available information on seaweed hazards, using literature review, data base analysis and expert opinion (Banach *et al.*, 2020). Seaweeds can become contaminated by microbial hazards during cultivation, processing or handling in food service. *Salmonella* spp., *Bacillus* spp., pathogenic *E. coli*, *Listeria* spp., *Staphylococcus aureus*, *Vibrio* spp., norovirus and hepatitis E virus are all potential microbial hazards. *B. cereus*, as a spore-former, is able to survive the heating and drying steps used to make rolled seaweed or other dried products.

Prevalence data for *L. monocytogenes* on seaweed are scarce, and absent for *T. gondii*. *L. monocytogenes* was not detected in raw, or heat-treated and frozen, seaweed sampled from a commercial growing area in Norway (Blikra *et al.*, 2019).[46] *T. gondii* can adhere to marine plants (Mazzillo *et al.*, 2013).

Human illness

There is some evidence that maternal iodine excess, specifically due to consumption of iodine-rich brown seaweeds, may induce hypothyroidism in neonates (Emder and Jack, 2011; Nishiyama et al., 2004).

No listeriosis outbreaks implicating seaweed were located.

Controls

Maintaining hygienic growing and processing facilities are the main controls. The process of producing dried seaweed can kill vegetative pathogenic bacteria, particularly if this involves a roasting step.

[46] The number of samples tested is not reported but may be as low as two, i.e. one sample from each of two seaweed species.

Summary

New Zealand Food Safety's current advice to pregnant women includes guidance to limit consumption of brown seaweed (New Zealand Food Safety, 2020). However, given the low iodine status of the New Zealand environment (Hercus *et al.*, 1925), advice with respect to brown seaweed consumption should not compromise messages concerning the importance of adequate iodine nutrition during pregnancy.

The current evidence is not sufficient to determine whether there should be additional advice relating to microbial hazards. The heating and drying used to produce seaweed products is likely to be listericidal but prevalence data are needed.

5.7.9 Seaweed: Red or green

Red or green seaweed (including nori and karengo) used in sushi and dulse

CURRENT ADVICE:	Ok to eat, see advice on sushi

Food

See brown seaweed (Section 5.7.8). Red algae (Rhodophyta) and green algae (Chlorophyta) are types of seaweed (macroalgae) (Banach *et al.*, 2020). Within each group there are a range of edible species with common names such as laver, nori, karengo or parengo. These are usually dried and roasted for commercial sale.

Hazards

See brown seaweed (Section 5.7.8).

A study of the microbial quality of laver (called nori in Japan) during the roasting process by six Korean manufacturers did not detect *L. monocytogenes* (Choi *et al.*, 2014).

Human illness

No listeriosis outbreaks implicating seaweed were located.

There have been outbreaks of salmonellosis (due to unsanitary growing conditions) and norovirus infection (Nichols *et al.*, 2017; Sakon *et al.*, 2018).[47]

Controls

See brown seaweed (Section 5.7.8).

Summary

The current evidence is not sufficient to evaluate New Zealand Food Safety advice. The heating and drying used to produce dried seaweed products is likely to be listericidal but prevalence data are needed. Other microbial hazards have caused outbreaks, which demonstrates the potential for contamination during or after processing.

[47] https://www.foodsafetynews.com/2019/09/norway-norovirus-outbreaks-linked-to-seaweed-salad-from-china/ (accessed 14 May 2020).

5.8 ADDITIONAL FOODS

The scientific evidence shows that sprouts are a higher risk food for vulnerable people and can be contaminated with *L. monocytogenes*. We propose that separate advice be included for sprouts, as detailed below. In addition, recall and outbreak data concerning *L. monocytogenes* were scanned to identify any foods that may be emerging or unidentified risks for listeriosis, see below.

5.8.1 Sprouts

Food

Sprouts are the emergent plantlets germinated from a variety of seeds, including alfalfa, mung bean, chickpea, radish, broccoli, soybean and sunflower (King *et al.*, 2015). The seeds used to produce sprouts are soaked in water, and then put in warm, humid conditions to sprout. They are usually kept refrigerated for sale and eaten raw, having a shelf life typically in the range 5-7 days.

Hazards

The germination conditions for sprouts are highly favourable for bacterial growth. Pathogenic bacteria can be introduced with the seeds (the most common source of contamination), irrigation water or from the growing environment. Recall data indicate that *Salmonella* spp., *Listeria* spp. and STEC are important microbial hazards (King *et al.*, 2015).

A New Zealand survey detected *Salmonella* spp. in 1/50 lots of pre-packed sprouted seeds and shoots purchased from retail outlets and tested within +/- two days of the expiry or best before date (D'Sa and Paulin, 2015). The concentrations were ≤0.04 MPN/g.

A New Zealand survey detected *L. monocytogenes* in 1/50 lots of pre-packed sprouted seeds and shoots purchased from retail outlets and tested within +/- two days of the expiry or best before date (D'Sa and Paulin, 2015). Six samples also tested positive for other *Listeria* spp. All *Listeria* spp. counts were <100 CFU/g, including L. monocytogenes.

T. gondii is unlikely to be present on sprouts unless it is introduced with irrigation water, however there are no data.

Human illness

In New Zealand, an outbreak of *S.* Typhimurium, involving 70 cases, was linked to alfalfa sprouts and occurred during 2018/19.[48]

Overseas, outbreaks of listeriosis have been reported where sprouts were the vehicle of infection (Buchanan *et al.*, 2017b; Dechet *et al.*, 2014).

A large international outbreak of STEC infection during 2011 caused 4,321 cases, including 852 cases of HUS and 50 deaths (Buchholz *et al.*, 2011). The cause was contaminated sprout seeds produced by a single supplier.

Controls

There are mandatory microbiological limits for *L. monocytogenes* and *Salmonella* spp. in sprouts sold in New Zealand. There are no additional controls.

[48] Public Health AIDE 17 April 2019 and 22 May 2019. Institute of Environmental Science and Research.

Summary

Sprouts are considered a high risk food for vulnerable people because they can be contaminated with microbial pathogens and have caused outbreaks. To minimise risk to pregnant women, it is suggested that new advice be included for sprouts, advising that these foods are not eaten raw, e.g. “Don’t eat, unless cooked.”

5.8.2 Emerging or currently unrecognised foods

Food products and preferences evolve over time and have been influenced by increased travel and the globalisation of food. To assess whether there are any emerging food trends or products that may be of risk during pregnancy, food safety advice from other countries, food recalls, outbreak notices and scientific literature were reviewed. Table 5 includes foods that the available evidence suggests may be potentially emerging risk foods for pregnant women, however scientific evidence is not currently strong enough to support specific food safety advice for New Zealand.

Table 5. Potentially emerging risk foods for pregnant women

Food	Hazard	Evidence
Unpasteurised fruit juice and cider (non-alcoholic)	*L. monocytogenes, E. coli*	Food safety during pregnancy advice from US FDA and Health Canada recommend avoiding this food (Health Canada, 2019; USFDA, 2019).
	Toxoplasma	An investigation of a cluster of acute toxoplasmosis cases in Brazil during 2013 identified 73 cases, all of whom had consumed fresh açaí juice (Morais *et al.*, 2016). It is not known how the fruit (or juice) became contaminated.
Nuts, nut butters	*L. monocytogenes*	Eleven US FDA recalls, market withdrawals or safety alerts related to nuts (macadamia, cashews, almonds) or nut butters between January 2017 and March 2020.[49] *L. monocytogenes* can survive on a variety of low moisture products including nuts, chickpeas, pine nuts, etc. (Brar and Danyluk, 2018; Salazar *et al.*, 2019).
Filled pasta e.g. ravioli, tortellini, tortelloni (fresh or frozen)	*L. monocytogenes*	Food safety or market recalls in US and Europe. No data were located for *Listeria* spp. in fresh pasta. *Salmonella* spp. were not detected during a packaging trial of a cheese-filled, pasteurised product (Sanguinetti *et al.*, 2011).

[49] https://www.fda.gov/safety/recalls-market-withdrawals-safety-alerts (accessed 24 March 2020).

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APPENDIX A: PREGNANCY AND OTHER FOODBORNE PATHOGENS

Table 6 lists several pathogenic microorganisms that can be transmitted via food, although person-to-person or waterborne transmission are more common for most of these. Other than hepatitis E virus, of the types not endemic in New Zealand, there is no evidence for pregnant women being more susceptible to infection to these pathogens compared to the general population. Effects on the foetus or neonate have been reported for some of these pathogens, sometimes resulting in death or other severe outcomes.

Table 6. The susceptibility of pregnant women to foodborne (or potentially foodborne) pathogens and effects on the baby

Foodborne pathogen	Susceptibility of pregnant women to pathogen during pregnancy compared to general population	Effect on foetus or newborn	Reference
Viruses			
Hepatitis A virus	No evidence of increased susceptibility. Can cause gestational complications.	Not usually associated with serious outcomes (no reports of foetal death).	Chaudhry and Koren (2015)
Hepatitis E virus (HEV)	Pregnant women are at increased risk of HEV infection, as evidenced from countries where HEV is endemic (maternal death can occur). The genotypes associated with poor maternal outcomes (HEV-1 and HEV-2) are not endemic in New Zealand.	Vertical transmission from an infected, pregnant mother to her foetus is well-documented in HEV-endemic countries. This is an important cause of foetal and perinatal mortality in these regions. This transmission route is associated with HEV-1 and HEV-2 genotypes, and but not with HEV-3 and HEV-4 genotypes. The HEV-3 and HEV-4 genotypes are more common in non-endemic countries such as New Zealand.	King *et al.* (2017)
Norovirus	No evidence of increased susceptibility.	No reports of any effect on foetus or newborn.	No references located.
Bacteria			
Bacillus cereus	No evidence of increased susceptibility.	No risk found from foodborne *B. cereus* intoxications during pregnancy. Environmental sources can be a problem for premature neonates with low immunity.	Hilliard *et al.* (2003)
Clostridium botulinum	No evidence of increased susceptibility. Foodborne botulism during pregnancy has been reported. Can cause severe maternal	While medical interventions have been required to save foetuses, this was usually as a result of the mother being gravely ill. There are no reports of congenital	Badell *et al.* (2017)

Foodborne pathogen	Susceptibility of pregnant women to pathogen during pregnancy compared to general population	Effect on foetus or newborn	Reference
	health outcomes including death.	botulism or of neonates having neurologic symptoms consistent with exposure to botulinum toxin. The botulinum toxin does not cross the placenta.	
Clostridium perfringens	No evidence of increased susceptibility. Cases have been reported, some associated with invasive procedures.	Foetal death has been reported.	(Kirkpatrick *et al.*, 1982; Lantelme *et al.*, 1995; Plachouras *et al.*, 2004)
Shiga toxin-producing *Escherichia coli* (including *E. coli* O157)	No evidence of increased susceptibility. There are no reports pregnancy complications associated with STEC infection but maternal cases of haemolytic uremic syndrome (HUS) have been reported.	Neonatal HUS caused by STEC transmission from mother to the newborn during delivery has been reported.	Sacerdoti *et al.* (2018)
Shigella spp.	No evidence of increased susceptibility but cases have been reported. Gestational complications can arise leading to premature birth.	Neonatal infections have been reported and the health outcomes can be severe.	Parisot *et al.* (2016)
Staphylococcus aureus	No evidence of increased susceptibility. *S. aureus* can naturally colonise the rectovaginal area. It appears that pregnant women with rectovaginal colonisation are more susceptible to infection compared to pregnant women without *S. aureus* rectovaginal colonisation.	Foetal death has been reported but *S. aureus* is more likely to be transferred from mother to baby during birth. Infants born to *S. aureus*–colonised mothers were not at higher risk of *S. aureus* infections compared to infants born to *S. aureus*-negative mothers.	(Jimenez-Truque *et al.*, 2012; Negishi *et al.*, 1998; Top *et al.*, 2012)
Vibrio parahaemolyticus, Vibrio vulnificus	No evidence of increased susceptibility.	No information located.	No references located.
Yersinia enterocolitica	No evidence of increased susceptibility. Infection significantly affecting the	Rare in newborns.	(Asarkua *et al.*, 2015; Grzelczyk-Wielgórska

Foodborne pathogen	Susceptibility of pregnant women to pathogen during pregnancy compared to general population	Effect on foetus or newborn	Reference
	course of pregnancy is very rare.		*et al.*, 2011)
Parasites			
Cryptosporidium parvum	No evidence of increased susceptibility.	No evidence of transplacental transmission or foetal infection. The newborn might be infected during or after birth.	(Pedersen *et al.*, 2014; Smith, 1999)
Giardia lamblia	No evidence of increased susceptibility.	No evidence of transplacental transmission or foetal infection. The newborn might be infected during or after birth.	(Rodriguez-Garcia *et al.*, 2002; Smith, 1999)

APPENDIX B: GENERIC INFORMATION ON MICROBIAL PATHOGENS OF RELEVANCE TO FOOD SAFETY DURING PREGNANCY

B.1 *LISTERIA MONOCYTOGENES*

B.1.1 Parameters for survival, growth and inactivation

Survival and Growth

Listeria monocytogenes is found commonly in the environment, including food processing and agricultural areas, particularly associated with damp or moist soil (Buchanan *et al.*, 2017a). The optimum temperature for growth of *L. monocytogenes* is 30–37°C with a range of -1.5–45°C. Significantly, *L. monocytogenes* can survive and grow at refrigeration temperatures, from -0.5 to 9.3°C, and survive at freezing temperatures (0 to -18°C). The pH optimum for growth is 7.0 with a range 4.4-9.4 (ICMSF, 1996). *L. monocytogenes* can survive at pH<4.3 but will not grow.

Food processing strategies to suppress or inactivate microorganisms may be ineffective against *Listeria*. Strains of *L. monocytogenes* can be resistant to high salt or acidic conditions and also survive in low oxygen or low humidity environments (Buchanan *et al.*, 2017a). *L. monocytogenes* grows optimally under microaerophilic conditions and can grow in food packaged under vacuum or nitrogen gas (Sutherland *et al.*, 2003). The organism has a low water activity (a_w) limit for growth; 0.92 in both sodium chloride (NaCl) and sucrose. It can survive for extended periods at a_w values as low as 0.83. Recent evidence suggests that *L. monocytogenes* can survive for months in a variety of low moisture foods (a_w ranging from 0.28 to 0.65) including raw nuts, nut butters, nonfat milk powder, wheat flour and infant formula (Taylor *et al.*, 2019).

The organism can grow in environments with NaCl concentrations up to 10%, with some laboratories reporting growth up to 12% NaCl (if the pH is sufficiently high) (Sutherland *et al.*, 2003).

Conditions recognised to not support the growth of *L. monocytogenes* are pH<4.4, a_w<0.92, NaCl >16%, or pH<5.0 combined with a_w <0.94 (CAC, 2009).

Foods which form some protection against gastric acids may increase the ability of *L. monocytogenes* to survive passage through the stomach into the intestine. Neutralisation or suppression of gastric acids by the use of antacids or proton pump inhibitors may also have a similar effect (Bavishi and DuPont, 2011). *L. monocytogenes* is able to adapt to acidic or low pH conditions in food or the gastrointestinal tract through the arginine deaminase pathway and the glutamate decarboxylase system (Gahan and Hill, 2014).

Biofilms are readily formed by *L. monocytogenes* and may provide an environment in which bacterial cells can survive for an extended period of time, for example in food processing environments. Within a biofilm, *L. monocytogenes* cells are resistant to anionic acids, chlorine and iodine (Colagiorgi *et al.*, 2017).

Inactivation

The temperature and time conditions (*D*-values) needed to inactivate *L. monocytogenes* are dependent on the intrinsic properties of the food. For example, *L. monocytogenes* in “all raw

meats" will be inactivated by heating at 55, 60 or 70°C for 95.6, 15.2 or 0.4 minutes respectively (Horn, 2015). Generally, *L. monocytogenes* is rapidly inactivated at temperatures above 70°C, depending on the properties of the food.

The organism is inactivated at pH values below 4.4 - the rate depending on the acidulant and temperature. Organic acids, such as acetic, are more effective than mineral acids (e.g. hydrochloric). In a study by Glass *et al.* (1995), acetic acid reduced *L. monocytogenes* more effectively than malic or citric acids in a fresh soft cheese.

Nonthermal treatment technologies including ultraviolet light (UV), pulsed electric fields (PEFs), high hydrostatic pressure (HHP) and manosonication (MS) have been assessed for their ability to inactivate *Listeria* (Cebrian *et al.*, 2016). The effectiveness of these technologies is influenced not only by the a_w and pH of the matrix containing *L. monocytogenes* (for PEF or HHP treatment), but also the growth phase of the bacterium, and the optical properties of the matrix (for UV treatment). *L. monocytogenes* appears to be more resistant to inactivation by UV when compared to other foodborne pathogens (Cebrian *et al.*, 2016). The lethality of all technologies is further enhanced by combining with an increase in treatment temperature.

B.1.2 Transmission routes

Most cases of listeriosis are sporadic and not linked to a common source outbreak. It has been estimated by expert consultation that 86.3% (95th percentile credible interval: 52.8–98.5%) of *L. monocytogenes* infection incidence is due to foodborne transmission (Cressey and Lake, 2013). It was further estimated that approximately 54% of foodborne transmission was due to transmission via ready-to-eat meats. An analysis of ProMED reports over a 22-year period found that the foods most commonly identified as vehicles of *L. monocytogenes* transmission were unpasteurized milk and dairy products, soft cheeses (e.g. queso fresco, feta and Camembert), cooked ready-to-eat sausages and sliced meats, refrigerated smoked seafood, and refrigerated pâtés or meat spreads (Desai *et al.*, 2019).[50]

Rare cases of nosocomial (hospital acquired) transmission of *L. monocytogenes* have been reported. Of particular note was the transmission of *L. monocytogenes* from a pregnancy-associated infected neonate to another neonate in the same birthing unit (Hof *et al.*, 2000; Lazarus *et al.*, 2013). Other hospital-acquired clusters have occurred through contaminated food, including the New Zealand outbreak in 2012 (Rivas *et al.*, 2019).

Under rare circumstances *L. monocytogenes* can also cause cutaneous listeriosis characterised by the development of skin papules or pustules (Godshall *et al.*, 2013). This form is seen almost exclusively in farmers or veterinarians after direct contact with aborted calves during delivery. For healthy individuals, the infection is usually restricted to the skin, but can be invasive if the exposed person is immunocompromised.

L. monocytogenes can be carried asymptomatically in the gut of healthy people, including pregnant women (Hernandez-Milian and Payeras-Cifre, 2014; Lamont and Postlethwaite, 1986). The prevalence of carriage can vary depending on the population assessed (Sauders *et al.*, 2005). Faeces from an infected person can contain high numbers of the organism (up to 10^4/g) (Sim *et al.*, 2002). However, reports of person-to-person transmission are uncommon (other than mother to foetus).

The widespread distribution of *L. monocytogenes* in food processing and agricultural environments suggests the organism should be considered as potentially present in all raw

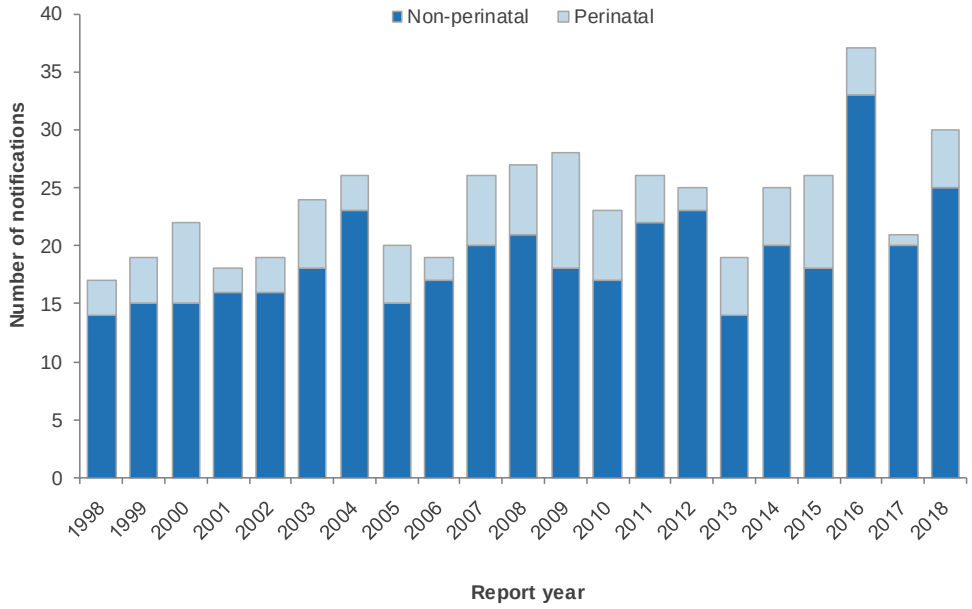
[50] ProMED is a global reporting system for infectious disease outbreaks, https://promedmail.org/.

foods and ingredients. It may also be present in cooked or ready-to-eat foods as a result of post-processing contamination. The risk posed is likely to be greatest in ready-to-eat cooked foods on which *L. monocytogenes* can grow.

B.1.3 Listeriosis in New Zealand

Sporadic cases

Listeriosis has been a notifiable disease since 1983 (Jeffs *et al.*, 2020). Cases are classified as perinatal (pregnancy-associated) if illness occurs in a pregnant woman, foetus, or infant aged ≤28 days old (Pattis *et al.*, 2019). During the period 1998–2018, between 5 and 36% of notified listeriosis cases each year were perinatal, resulting in a total of 41 deaths (Figure 2 and Table 7).



Reproduced from Pattis *et al.* (2019)

Figure 2. Number of notified listeriosis cases (perinatal and non-perinatal), 1998–2018

Table 7. Notified listeriosis cases (perinatal and non-perinatal) and perinatal deaths, 1998–2018*

Year	All Cases	Perinatal Cases	% Cases Perinatal	Perinatal Deaths
1998	17	3	18	0
1999	19	4	21	2
2000	22	7	32	4
2001	18	2	11	1
2002	19	5	26	3
2003	24	6	25	2
2004	26	3	12	2
2005	20	5	25	4

Year	All Cases	Perinatal Cases	% Cases Perinatal	Perinatal Deaths
2006	19	2	11	1
2007	26	5	19	2
2008	27	6	22	2
2009	28	10	36	2
2010	23	6	26	4
2011	26	4	15	0
2012	25	2	8	2
2013	19	5	26	3
2014	25	5	20	2
2015	26	8	31	3
2016	36	4	11	2
2017	21	1	5	0
2018	30	5	17	0

* Data collated from annual surveillance summaries, https://surv.esr.cri.nz/surveillance/annual_surveillance.php (accessed 10 March 2020), and Pattis *et al.* (2019).

Hospitalisation data combined from the ESR Notifiable Disease Database (EpiSurv) and the National Minimum Dataset (NMDS) identified 102 cases of listeriosis associated with pregnancy from 1997 to 2016, with 88 infections occurring in pregnant women and the remainder occurring in neonates (Jeffs *et al.*, 2020). The pregnancy-associated incidence of listeriosis during the study period was 12.3 per 100,000 births and there were no significant trends across time. Approximately one third (34%) of the infections during pregnancy resulted in foetal loss.

Outbreaks

Recognition of outbreaks caused by *L. monocytogenes* is often difficult due to the prolonged incubation period of the organism before disease occurs (anywhere between 1 and 90 days). However, in pregnancy-associated cases, the period between exposure and onset of illness is often less than 90 days. Scientific advancements in molecular methods, including typing/fingerprinting and whole genome sequencing, means better identification of clusters of cases and linkages to possible sources.

Reported outbreaks of infection with *L. monocytogenes* in New Zealand are rare. From 1992 to 2017 only four outbreaks have been reported. An outbreak in 1992 involved smoked mussels as the vehicle (see below). The second outbreak was non-invasive listeriosis, occurring in early 2000, and was associated with ham and corned silverside. A food vehicle was not identified in the invasive third outbreak (2009) and the last outbreak in 2012 was invasive and was associated with sliced, cold, ready-to-eat meats.

A well-documented outbreak of perinatal listeriosis in New Zealand was associated with the consumption of smoked mussels (Baker *et al*., 1993; Brett *et al*., 1998). There were four clinical cases, two neonate twins in Auckland that died (Patient 1 and 2), one in Nelson (patient 3) and one in Invercargill (patient 4). Patients 1 and 2 were diagnosed a month apart, when *L. monocytogenes* serogroup 1/2a was isolated from their blood. A food history of one brand of smoked mussels led to the sampling of an unopened packet of mussels from

the refrigerator of the mother. Pulsed-filed gel electrophoresis (PFGE) typing found isolates from patients 1 and 2 were indistinguishable from isolates found in the mussels.

Two further cases, patients 3 and 4, also yielded *L. monocytogenes* isolates with an indistinguishable PFGE pattern. Patient 3 had consumed mussels, but did not specify the brand in question. Patient 4 had consumed unpasteurised milk, but not mussels. In conclusion, a microbiological link was made between three clinical cases, a food source and the manufacturing environment where the mussels were processed. The strain was uncommon in New Zealand. The mother of the twins had consumed a significant quantity of the mussels throughout her pregnancy with the intention of raising her iron levels.

B.2 *TOXOPLASMA GONDII*

B.2.1 Parameters for survival, growth and inactivation

Survival and growth

T. gondii oocysts do not multiply in the environment (only the sporozoites within the oocyst multiply during the first few days after excretion from a cat). Oocysts survive for long periods (months, years) in the environment (Hill and Dubey, 2016).

Cysts can remain viable in fresh meat and offal but do not multiply. *T. gondii* cysts can also remain viable in processed meats, depending on the ingredients, processes and characteristics (e.g. pH, salt and preservatives).

Inactivation

T. gondii in the form of oocysts were inactivated in water when heated to 60°C for 1 minute (Dubey, 1998). Under freezing, freshly excreted, unsporulated oocysts were killed within 1–7 days when frozen at -21°C but sporulated oocysts survived at least 14 days at this temperature, and 28 days or more at -6ºC (Frenkel and Dubey, 1973). Oocysts can also be inactivated by UV irradiation, but show resistance to disinfectants (Wainwright *et al.*, 2007a; Wainwright *et al.*, 2007b).

Cysts in meat are inactivated by cooking and freezing but time, temperature and the type of meat all influence inactivation rates (Guo *et al.*, 2015). Other controls, such as irradiation and hydrostatic pressure, can also inactivate cysts in meat products but these are not relevant to the domestic environment and are not discussed here.

Heat will quickly inactivate cysts, specifically heating above 60°C, although inactivation will occur at lower temperatures with extended heating times (De Berardinis *et al.*, 2017). For example, the following D times have been calculated for destroying cysts in pork: 336 seconds at 49°C, 44 seconds at 55°C, 6 seconds at 61°C and 1 second at 67°C (the 99% upper confidence limit at 67ºC was 3 seconds) (Dubey *et al.*, 1990). A review of studies evaluating methods for inactivating cysts and oocysts found that both forms were inactivated at temperatures of 50ºC or above, although heating times as long as one hour were required at lower temperatures (Mirza Alizadeh *et al.*, 2018).

Microwave cooking appears to be unreliable for inactivation of *T. gondii*, which may be due to uneven heating but this is not certain. After mutton steaks were microwaved to 65°C residual infective cysts were detected (Lunden and Uggla, 1992). In another study, microwaved sheep meat still contained viable tissue cysts but the final meat temperature was not reported (El-Nawawi *et al.*, 2008).

T. gondii cysts are sensitive to freezing. Freezing of at least two days below -12°C inactivates cysts, and freezing at -20ºC for three days has been recommended for *T. gondii* control (De Berardinis *et al.*, 2017; Kotula *et al.*, 1991; Mirza Alizadeh *et al.*, 2018). However, longer periods at higher freezing temperatures also inactivate cysts (Mirza Alizadeh *et al.*, 2018).

Processes such as curing, smoking, drying and fermentation can also inactivate *T. gondii* cysts. Available data on the impact of various meat processes on the viability of *T. gondii* were reviewed as part of a qualitative risk assessment for the USA (Guo *et al.*, 2015). The outcome of this review was a series of tables describing the critical parameters required for cyst inactivation through moisture enhancement,[51] curing, smoking, drying, fermentation, thermal processing, etc. For example, the process of smoking had no effect unless heat was included (hot smoking). The effectiveness of salt varied, with temperature and treatment duration both being important. The presence of other curing ingredients (e.g. nitrite) enhanced the effect of salt in some studies. Researchers conducting a study examining bradyzoite inactivation during pepperoni production concluded that "If pH is <4.6 and ≤5.2, and % NaCl ≥1.3%, 4 h of fermentation is required to achieve complete inactivation of *T. gondii* bradyzoites (in tissue cysts). This proposed rule is not valid for pH outside of the range (4.6, 5.2), nor for NaCl <1.3%." (Fredericks *et al.*, 2019). It was noted that this lower limit of salt concentration (1.3%) was lower than usually used during pepperoni production (Fredericks *et al.*, 2019; Hill *et al.*, 2018). Salt is a more effective control than pH (Franssen *et al.*, 2019).

T. gondii in cysts can be killed when raw meat is injected with salts and water (Hill *et al.*, 2004; Jones and Dubey, 2012).

B.2.2 Transmission routes

There are four main *T. gondii* transmission routes that are relevant to pregnant women (Leroy and Hadjichristodoulou, 2005; Pinto-Ferreira *et al.*, 2019a; Shapiro *et al.*, 2019):

- Ingestion of cysts (bradyzoites) with raw or undercooked meat, or ingestion of uncooked foods that have been in contact with such meat;
- Ingestion of oocysts (sporozoites) through direct contact with cat faeces or indirect contact where oocysts have spread in the environment with soil, water or invertebrates, e.g. contaminated vegetables, shellfish or drinking water;
- Ingestion of tachyzoites with raw milk from an animal with an active infection;
- Transplacental infection during a primary infection of the mother.

The first three transmission routes have a foodborne aspect and the resulting disease is termed acquired, while the latter transmission route results in congenital toxoplasmosis.

Serological tests able to detect the presence of sporozoites have been developed and these provide an indication of whether a person has been infected through consuming oocysts. This approach showed that 78% of acutely infected mothers (n=76) in North America, with congenitally infected infants, were probably infected by sporozoites from ingesting oocysts (Boyer *et al.*, 2011). The other 22% were probably infected by ingesting tissue cysts. A study in Chile indicated that 43% of pregnant women with serological evidence of a recent *T. gondii* infection (n=72) were probably infected through ingesting oocysts (Munoz-Zanzi *et al.*,

[51] Moisture enhancement refers to the process of injecting meat with solutions containing salt, phosphates, antioxidants and/or flavourings

2010). These studies show that ingestion of oocysts and tissue cysts are both important in congenital infections.

B.2.3 Toxoplasmosis in New Zealand

Sporadic cases

Toxoplasmosis is not a notifiable disease in New Zealand. Data available from New Zealand hospital discharge records show a small number of reported cases of congenital toxoplasmosis per year, totalling 15 during the period 2007-2016 (King, 2017). All but two of these 15 cases were less than one year of age (the others were aged 3 and 21 years).

There is no information currently available to link cases of toxoplasmosis with foodborne transmission or to assess its importance relative to other transmission routes (Lake *et al.*, 2008). It has been estimated by expert consultation that 25.3% (95th percentile credible interval: 3.6–55.6%) of *T. gondii* incidence is due to foodborne transmission (Cressey and Lake, 2013). However, it should be noted that, for this expert consultation, *T. gondii* was the organism for which the experts self-reported the lowest level of expertise.

Outbreaks

There have been no reported outbreaks of toxoplasmosis in New Zealand. In other countries, outbreaks have been associated with consumption of rare/undercooked meat, drinking water, sand, soil or dust, vegetables and raw milk (Jones and Dubey, 2012; Jones *et al.*, 2016; Meireles *et al.*, 2015; Nunes do Rego e Silva *et al.*, 2019; Pinto-Ferreira *et al.*, 2019a).

Case-control studies

Case-control studies offer information on risk factors when such information is not routinely collected through public health surveillance, as is the case for toxoplasmosis in New Zealand. However, the results from overseas studies can be difficult to interpret in the New Zealand context because of the varied ways questions are asked, and because of the variety of local food customs, food hygiene and lifestyles across the populations involved.

Case control studies involving a range of population cohorts have shown that “the risk factors commonly associated with being seropositive were being older, contact with soil, contact with cats, contact with domestic animals, living in a rural area, eating unwashed vegetables, eating or being in contact with raw/undercooked meat and being male. Drinking untreated water, consuming unpasteurised milk and having multiple pregnancies were also significant in several studies” (King, 2017). Raw shellfish consumption has also been identified as a risk factor (Chiang *et al.*, 2014).

A systematic review and meta-analysis of toxoplasmosis case control studies concluded that *T. gondii* infections were significantly associated with consumption of raw/undercooked meat (OR 3.44, 95% CI 1.29-9.16), and separately, raw/undercooked beef (OR 2.22, 95% CI 1.57-3.12) and raw/undercooked sheep meat (OR 3.85, 95% CI 1.85-8.00) (Belluco *et al.*, 2018). These results were based on nine studies, of which five had investigated risk factors for pregnant women.

The results of case control studies focussing on *T. gondii* infection among pregnant women are summarised in Appendix D. Eating raw, cured or undercooked meat was a consistent risk factor identified through these studies. Other significant risk factors were eating raw vegetables (with one study describing these as being unwashed), contact with soil or gardens, infrequently washing knives between cutting raw meat and preparing other foods,

and having a pet cat or activities associated with having a cat. Interestingly, one study found that cases who “never or rarely” froze meat were twice as likely to seroconvert during pregnancy as those who froze meat more frequently, but this was not a strong risk factor relative to others identified in the study (95% CI 1.0-4.2, p-value not reported) (Baril *et al.*, 1999). Similarly, cases of acute toxoplasmosis were approximately twice as likely to consume unpasteurised milk or untreated water in another study, but eating raw/undercooked meat or tasting meat during cooking were relatively more important (Cook *et al.*, 2000).

B.3 *SALMONELLA SPP.*

B.3.1 Parameters for survival, growth and inactivation

Data in this section are derived from *Salmonella* datasheets prepared for the Ministry for Primary Industries (ESR, 2018b; c), unless otherwise referenced.

Survival and growth

Non-typhoidal salmonellae

Non-typhoidal salmonellae survive well in the environment, on foods, human skin and other materials. Notably, they can survive for extended periods (months or years) in dry environments, including foods with low water activity (a_w) such as black pepper, chocolate and peanut butter (minimum a_w 0.94). They can also tolerate acidic conditions (minimum pH 3.8), enhancing survivability.

Survival is longer at chilled temperatures, compared with ambient, and is dependent on factors such as pH and a_w. These bacteria can survive for long periods in frozen foods, and some foods protect bacteria during freezing and frozen storage (e.g. raw meat, fish and liquid egg).

Persistence of non-typhoidal salmonellae is enhanced by formation of disinfectant and antibiotic-resistant biofilms in host, non-host and food processing environments. These bacteria can also transition to the Viable but Non-Culturable (VBNC) state after exposure to low temperatures (5°C) in nutrient-limited conditions.

The optimum growth temperature for non-typhoidal salmonellae is 35–37°C, with a minimum of 5.2°C and maximum of 49.5°C. They can grow in the absence or presence of oxygen, and on foods stored in modified nitrogen or carbon dioxide (20–50%) atmospheres.

Typhoidal salmonellae

The growth and survival characteristics are similar to those of non-typhoidal salmonellae. They can survive for long periods in dry (minimum a_w 0.94) and low pH environments (minimum pH 3.8). *S.* Typhi can survive in ice for more than 90 days. The minimum growth temperature is slightly higher (7°C) than that of non-typhoidal salmonellae, and the maximum growth temperature slightly lower (45-47°C). Protective biofilms can be formed and VBNC states can enable persistence.

Inactivation

Most *Salmonella* spp. are sensitive to heat and are killed by normal cooking conditions (core temperature of 75°C instantaneously, or an equivalent time/temperature combination, e.g. 70°C for 2 min). Some strains of *Salmonella* Senftenberg are reported to be more resistant to heat when in culture medium. High fat and low moisture foods require severe heat

treatments to kill *Salmonella* spp. For example, the D_{80^oC} for *S*. Typhimurium in milk chocolate with <10% moisture is 222 minutes.

Salmonellae die slowly under frozen conditions and can survive for long periods, particularly when in proteinaceous foods (e.g. raw meat).

Salmonellae are sensitive to preservatives commonly used in foods (e.g. benzoic, sorbic and propionic acid). Inhibition is enhanced by combining interventions (e.g. nisin and ρ-cymene had a synergistic antimicrobial effect on *S.* Typhi when added to ready-to-eat pork sausages).

Salmonellae are also sensitive to disinfectants, high pressure processing and other treatments such as ozone and UV light, but these are not discussed here.

B.3.2 Transmission routes

Data in this section are derived from *Salmonella* datasheets prepared for the Ministry for Primary Industries (ESR, 2018b; c), unless otherwise referenced.

Salmonellae are ubiquitous in domestic and wild animals, and are widespread in food animals, pets and reptiles (WHO, 2018). They enter the environment (soil, water) with faeces from infected humans or animals. Infected animals are often carriers and do not develop disease.

Non-typhoidal salmonellae

In humans, salmonellosis is usually associated with the consumption of contaminated food of animal origin, although other foods, such as green vegetables, have been implicated in transmission (WHO, 2018).

Transmission routes include:

- Contact with infected persons.
- Contact with domesticated food animals (e.g. pigs, poultry, cattle), pets (e.g. birds, cats, dogs, turtles), or wild animals (e.g. birds, fish, amphibians) (WHO, 2018).
- Consumption of foods of animal and non-animal origin which may have faecal contamination, e.g. eggs, meat, fruit and vegetables.
- Contact with the environment (including pasture and soil) and water contaminated by faeces from animals. This is also a transmission route for animals to be infected.

It has been estimated by expert consultation that 62.1% (95th percentile credible interval: 35.2–86.4%) of salmonellosis incidence is due to foodborne transmission (Cressey and Lake, 2013). It was further estimated that approximately 19% of foodborne transmission was due to transmission via poultry.

Typhoidal salmonellae

Salmonella serotypes Typhi and Paratyphi do not infect other animals, thus transmission routes are associated with humans:

- Person-to person contact via faecal-oral pathway.
- Consumption of any foods or beverages contaminated by contact with body fluids from infected humans.

- Consumption of raw vegetables or shellfish fertilised by soil or water contaminated by faecal shedding from infected humans.
- Contact with the environment (including pasture and soil) and water contaminated by faecal shedding from infected humans.

Travel to regions endemic for typhoidal *Salmonella* is a significant risk factor. Approximately 75% of *Salmonella* Typhi cases in the US are linked with international travel (Guirguis *et al.*, 2017). Contaminated water is likely the predominant vector in endemic regions (Kim *et al.*, 2019).

B.3.3 Infections from *Salmonella* spp. in New Zealand

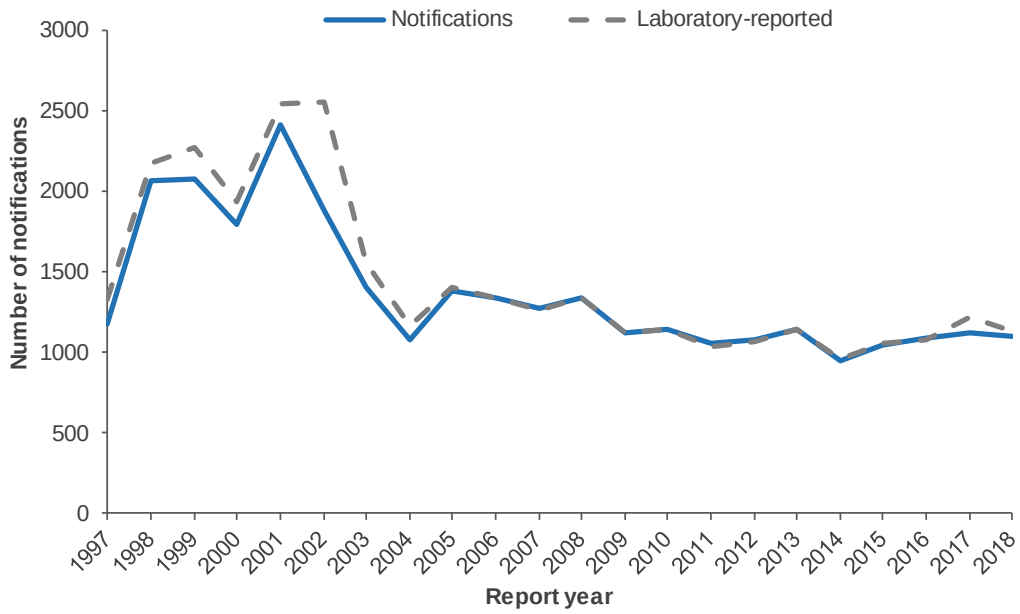
Sporadic cases

Salmonellosis, typhoid fever and paratyphoid fever are notifiable diseases in New Zealand, with salmonellosis occurring relatively more frequently (Table 8). There was a decrease in the annual number of reported cases between 2001 and 2004 and since 2015, between 1,053 and 1,216 cases have been notified each year (Figure 3) (Pattis *et al.*, 2019). Notifications are characterised by a late summer peak and winter trough.

Table 8. Number of notified cases of *Salmonella* spp. infection, 2008–2018[1]

Year	Salmonellosis	Typhoid Fever	Paratyphoid Fever
2008	1339	29	25
2009	1128	34	25
2010	1146	31	19
2011	1055	45	13
2012	1081	44	22
2013	1143	50	25
2014	955	43	19
2015	1051	43	34
2016	1091	38	32
2017	1119	60	47
2018	1100	-[2]	-[2]

[1] Data collated from Pattis *et al.* (2019) and ESR (2019).
[2] Data not available.



Reproduced from Pattis et al. (2019)

Figure 3. Salmonellosis notifications and laboratory-reported cases by year, 1997–2018

The most common *Salmonella* serotypes identified in New Zealand (2017) were *S.* Typhimurium and *S.* Enteritidis, the phage types of which appear consistent over the last 5 years. The number of cases of *Salmonella* Bovismorbificans has noticeably increased, from four cases in 2014 to 52 cases in 2017 (ESR, 2019).

Outbreaks

Typhoidal salmonellae

From 2016, the annual reporting of foodborne outbreak data specific to typhoid and paratyphoid fever ceased in New Zealand. During the period 2010-2015, there were 1–5 outbreaks per year involving 2–17 cases. Person-to-person transmission was the primary mode of infection, followed by cases associated with overseas travel to endemic areas (ESR, 2018b).

Notable international food-associated outbreaks of typhoidal *Salmonella* infection are shown in

Table 9.

Table 9. Large food-associated outbreaks of typhoidal *Salmonella* infection reported overseas

Year	Serotype	Food	Location	Cases (Deaths)	Suspected Transmission/Risk Factors	Reference
2015	Paratyphi B	Raw tuna (frozen)	USA	64 (0)	Sushi consumption (94% cases). Imported fish from endemic region (Indonesia)	USCDC (2015)
2015	Paratyphi B	Nut butter (sprouted)	USA	13 (0)	Single supplier to multiple retail outlets.	USCDC (2016)
2014	Typhi	Salad	Japan	7 (0)	Poor kitchen hygiene. Contamination by asymptomatic food handler.	Kobayashi *et al.* (2016)
2010	Typhi	Mamey sapote fruit pulp (frozen)	USA	12 (0)	Poor kitchen hygiene and manufacturing practices. Imported fruit from endemic region (Guatemala).	Loharikar *et al.* (2012)
1997	Typhi	Pork	France	13 (0)	Poor kitchen hygiene. Contamination by chronically infected food handler.	Pradier *et al.* (2000)

Non-typhoidal salmonellae

A large proportion of foodborne outbreaks of salmonellosis have no identifiable source (ESR, 2018c). A review of 204 salmonellosis outbreaks reported during the period 2000–2009 found that while non-typhoid salmonellosis was primarily a foodborne disease in New Zealand, there was insufficient information to identify important food vehicles (King *et al.*, 2011).

Table 10 lists the annual number of salmonellosis outbreaks and the number each year with foodborne transmission recorded as one of the likely modes of transmission. The foods implicated through epidemiological and/or laboratory evidence were varied.

Table 10. Number of salmonellosis outbreaks and those recorded as foodborne transmission, and some of the implicated foods, 2008–2018[1]

Year	Number of Outbreaks	Number of Foodborne Outbreaks[2]	Implicated Foods[3]
2008	15	4	Raw flour
2009	12	6	Watermelon, raw milk
2010	23	10	(none)
2011	15	8	Food remains from umu
2012	27	11	Tabouli, tahini
2013	18	9	Boiled egg and ham sandwich
2014	23	7	(none)
2015	18	3	(none)
2016	24	12	Biscuit, biscuit dough
2017	13	4	(none)
2018	14	5	(none)

[1] Data collated from the annual foodborne disease reports, available from New Zealand Food Safety and at https://www.mpi.govt.nz/food-safety/food-safety-and-suitability-research/human-health-surveillance/foodborne-disease-annual-reports/ (accessed 6 April 2020).
[2] An outbreak has been classed as foodborne if a food was recorded as one of the likely modes of transmission.
[3] Implicated by epidemiological evidence (case control or cohort study showed an elevated risk for cases consuming the food) and/or laboratory evidence (pathogen identified in a food handler or the food). A range of other foods are implicated for the foodborne outbreaks each year but the aforementioned evidence is not available for these foods.

A range of other foods have been implicated in salmonellosis outbreaks in New Zealand but there was insufficient evidence to conclude these were the likely vehicles of infection. These foods include eggs, meats (e.g. spit roast (undercooked), pork, beef, poultry, fish), rice and fresh produce.

Notable large (100+ cases) food-associated salmonellosis outbreaks also show that a range of foods can cause illness (Table 11).

Table 11. Large foodborne salmonellosis outbreaks reported overseas

Year(s)	Serotype(s)	Food	Location	Cases (Deaths)	Suspected Transmission/Risk Factors	Reference
2017-2020	Enteritidis	Eggs	Europe (15 countries)	858[1]	Inconclusive. Some cases linked to Polish and German producers.	ECDC (2020)
2019	Javiana	Cut fruit (honeydew melon, cantaloupe, pineapple, and grapes)	USA	165	Single supplier to long-term care facilities, hospitals, hotels, schools and supermarkets.	USCDC (2020)
2018	Infantis	Raw chicken (ground, pieces, whole, ground pet food)	USA	129 (1)	Multiple suppliers. Serotype also identified in poultry flocks.	USCDC (2019b)
2018	Newport	Raw beef	USA	403	Single supplier to multiple retail outlets.	USCDC (2019a)
2018	Sandiego, *S. enterica* IIIb	Pasta salad	USA	101	Single retailer, multiple outlets.	USCDC (2018)
2015	Poona	Cucumbers	USA	907 (6)	Imported from Mexico. Unsanitary storage.	Kozyreva *et al.* (2016)
2014	Heidelberg	Raw chicken	USA	634	Incorrect handling / cooking of raw product.	Gieraltowski *et al.* (2016)
2014	Enteritidis	Eggs	UK/Europe	287 (6)	Imported from Germany. Product contaminated.	Inns *et al.* (2015)

[1] 656 confirmed and 202 probable cases.

B.4 *CAMPYLOBACTER SPP.*

B.4.1 Parameters for survival, growth and inactivation

Data in this section are derived from the *Campylobacter* datasheet prepared for the Ministry for Primary Industries (ESR, 2018a), unless otherwise referenced.

Survival and growth

Compared to other food-borne pathogens, C*ampylobacter* spp. are more fastidious in their growth requirements and are very susceptible to various environmental stressors. *Campylobacter* spp. are sensitive to air, drying and heat. These bacteria survive better on food under refrigeration compared to room temperature. Because they are sensitive to atmospheric conditions, preferring reduced oxygen, they also survive better on foods under modified atmosphere or vacuum packaging. However, *Campylobacter* spp. can survive and even grow when initially packed under normal atmospheric conditions, as the metabolic activity of the food, such as raw meat, may create a carbon dioxide-enhanced gaseous environment (ICMSF, 1996). *Campylobacter* spp. are very sensitive to drying but can survive up to an hour on hands that are not dried properly after washing, and on moist surfaces.

Campylobacter jejuni/coli are thermotolerant and grow optimally at 42°C. Neither species grows below 30.5 or above 45°C. These species are comparatively slow growing (fastest generation time approximately 1 hour) even under optimum conditions and does not grow under refrigeration.

Under stressful conditions, *Campylobacter* spp. are said to undergo a transition to a VBNC state (Kassem *et al.*, 2013; Ramamurthy *et al.*, 2014). The ability of *Campylobacter* spp. to produce VBNC cells is becoming more widely, but not universally, accepted.

C. jejuni also appear capable of forming single or multi-species biofilms (Tram *et al.*, 2020). However, the ability of these bacteria to form biofilms under the conditions of a food processing environment appears limited (Teh *et al.*, 2014). Attaching to existing biofilms formed by other species could contribute to the survival of *C. jejuni* in the food-related environment.

Campylobacter spp. produce several different cytotoxins but none are produced in foods (Bolton, 2015).

Inactivation

Campylobacter spp. are sensitive to heat and are rapidly killed by normal cooking conditions (core temperature of 60°C instantaneously).

Freezing causes an initial decrease in numbers of *C. jejuni* followed by a gradual reduction during subsequent storage. Freezing rates influence survival more than actual frozen storage temperature. Slow freezing rates are more lethal than rapid freezing because of osmotic stress, as are very high freezing rates (in excess of 10°C/min), which result in mechanical cell damage due to intracellular ice crystals. During storage, the rate of reduction depends on the type of food and storage temperature.

Campylobacter spp. are also inactivated under low pH (<4.0), NaCl concentrations above 2% and air-drying conditions.

B.4.2 Transmission routes

Reports of person-to-person transmission are rare, despite large (6-9 $\log_{10}$ CFU/g) microbial loading of faeces from infected individuals. Source attribution analysis of campylobacteriosis

in New Zealand has been ongoing since 2004. The classical seven-gene multi-locus sequence typing (MLST) has been used to genotype isolates from human clinical cases, domestic and wild animals and samples from food and environmental water. These analyses are based on associations between animal hosts and particular sequence types (STs) which have been found to be consistent across a number of countries (Sheppard *et al.*, 2010). All source attribution reports for New Zealand to date have provided strong evidence that poultry is a major reservoir for human infections but also that ruminants are a reservoir (French and Marshall, 2014; Marshall *et al.*, 2015; Marshall *et al.*, 2017; Nohra *et al.*, 2016).

It has been estimated by expert consultation that 62.6% (95th percentile credible interval: 43.4–82.5%) of campylobacteriosis incidence is due to foodborne transmission (Cressey and Lake, 2013). It was further estimated that approximately 74% of foodborne transmission was due to transmission via poultry.

A source-assigned case-control study of notified human cases of campylobacteriosis in New Zealand during 2018/19 was recently published (Lake *et al.*, 2019). The source assignment models provided estimates of the number of cases that could be assigned to a source as follows:

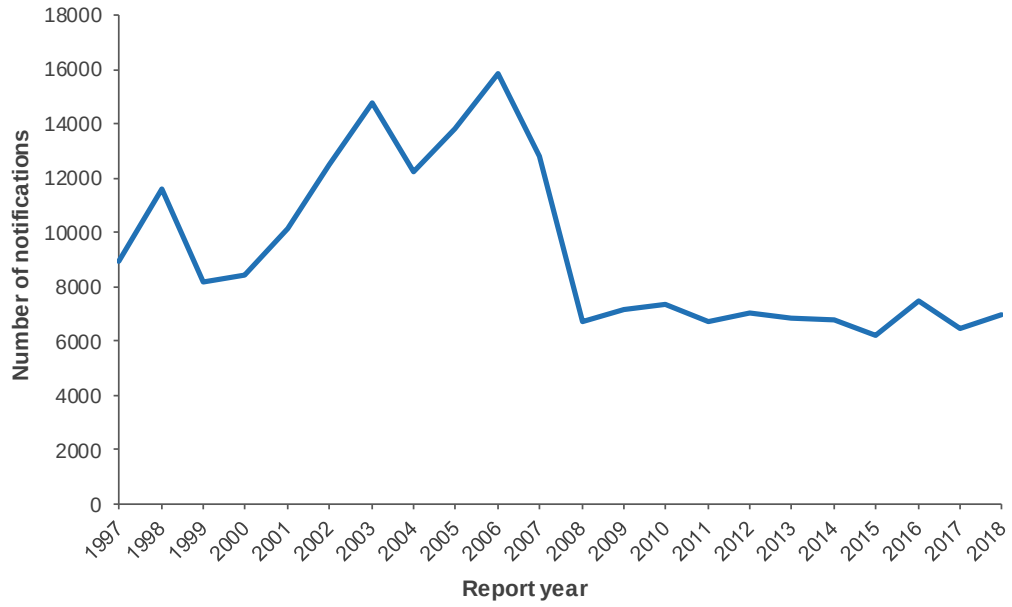
- Poultry 84%
- Sheep 0%
- Cattle 14%
- Unassigned 2%

The overall findings of the study suggest that poultry meat remains the dominant pathway for exposure and poultry-specific risk factors were statistically associated with campylobacteriosis, e.g. preparing poultry in the home, consumption of undercooked chicken, consumption of chicken outside the home. There was a range of other significant risk factors that involved foodborne transmission (e.g. consuming raw milk), zoonotic transmission (e.g. direct animal contact) and waterborne transmission (e.g. rainwater/roof tank as a water source). Risk factors associated with preparing and eating red meat were less important compared to contact with meat animals.

B.4.3 Campylobacteriosis in New Zealand

Sporadic cases

Campylobacteriosis is a notifiable disease in New Zealand and the case definition encompasses disease caused by any *Campylobacter* species (MOH, 2019). The number of cases decreased during 2007/08 and has remained stable since, with the exception of 2016 during which an outbreak linked to contaminated drinking water occurred (Figure 4) (Pattis *et al.*, 2019). The 50% reduction in disease incidence in 2008 compared to the 2002–2006 incidence was attributed to the poultry industry implementing regulatory and voluntary *Campylobacter* control strategies (Muellner *et al.*, 2011; Sears *et al.*, 2011). Since 2008, between 6,218 and 7,456 cases have been reported each year (Table 12). Cases who are pregnant at the time of infection are not reported separately.



Reproduced from Pattis et al. (2019)

Figure 4. Campylobacteriosis notifications and laboratory-reported cases by year 1997-2018

Table 12. Number of notified cases of campylobacteriosis, 2008–2018[1]

Year	Campylobacteriosis cases
2008	6694
2009	7177
2010	7346
2011	6686
2012	7016
2013	6837
2014	6782
2015	6218
2016	7456
2017	6482
2018	6957

[1] Data collated from Pattis *et al.* (2019) and (ESR, 2019).

Outbreaks

Table 13 lists the annual number of campylobacteriosis outbreaks and the number each year with foodborne transmission recorded as one of the likely modes of transmission. Chicken liver and raw (unpasteurised) milk were implicated through epidemiological and/or laboratory evidence.

Table 13. Number of campylobacteriosis outbreaks and those recorded as foodborne transmission, and some of the implicated foods, 2008–2018[1]

Year	Number of Outbreaks	Number of Foodborne Outbreaks[2]	Implicated Foods[3]
2008	16	8	chicken liver paté
2009	12	7	Raw milk
2010	29	14	(none)
2011	29	11	Chicken liver mousse
2012	32	11	(none)
2013	40	16	Raw chicken liver smoothies
2014	35	18	chicken liver pâté, chicken liver parfait
2015	19	11	(none)
2016	15	8	(none)
2017	7	4	(none)
2018	16	7	Raw milk

[1] Data collated from the annual foodborne disease reports, available from New Zealand Food Safety and at https://www.mpi.govt.nz/food-safety/food-safety-and-suitability-research/human-health-surveillance/foodborne-disease-annual-reports/ (accessed 6 April 2020).

[2] An outbreak has been classed as foodborne if a food was recorded as one of the likely modes of transmission.

[3] Implicated by epidemiological evidence (case control or cohort study showed an elevated risk for cases consuming the food) and/or laboratory evidence (pathogen identified in a food handler or the food). A range of other foods are implicated for the foodborne outbreaks each year but the aforementioned evidence is not available for these foods.

A very large waterborne outbreak of campylobacteriosis occurred in 2016 in New Zealand, caused by drinking water contaminated with sheep faeces (Government Inquiry into Havelock North Drinking Water, 2017).[52] Although 964 cases were notified during the outbreak, centred in Havelock North, it is estimated that the actual number of cases was over 7,000 in a town with a population of 14,000. The Hawke's Bay District Health Board was notified of 45 hospitalisations linked to the outbreak. It is possible that the outbreak contributed to three deaths, and an unknown number of residents suffering health complications (such as reactive arthritis and GBS) were reported.

The number of cases who were pregnant at the time of the outbreak is not reported in the available literature. This outbreak represents an opportunity to investigate whether pregnant women are at increased risk of campylobacteriosis or complications. The notified case information collected as a part of the outbreak investigation and surveillance may include this level of detail.

[52] ESR, unpublished data.

APPENDIX C: INACTIVATION, SURVIVAL OR GROWTH OF *L. MONOCYTOGENES* IN FOOD

The following data are compiled from Appendix 8 of the US FDA/FSIS (2003) quantitative risk assessment of *Listeria monocytogenes* in selected ready-to-eat-foods. The information is presented as the growth rate of *L. monocytogenes* in different food categories. The exponential growth rate at 5°C was determined from modelling data points extracted from the listed references. A detailed explanation on how the exponential growth rates were modelled and derived can be found in the risk assessment (FDA/FSIS, 2003).

Abbreviations:

EGR = Exponential Growth Rate
GT = Generation Time
logs = Log_{10} cfu/g

MILK

TYPE	TEMP. (°C)	GROWTH RATE	EGR AT 5°C (LOG_{10} CFU/DAY)	REFERENCE
Unpasteurised milk	5 7	GT in 3.5 days GT in 1 day	0.085 0.173	Northolt *et al.* (1988)
Pasteurised milk	4	2 log units in 7 days	0.407	Northolt *et al.* (1988)
Unpasteurised milk	4 10 15	GT in 25.3 hours GT in 10.8 hours GT in 7.4 hours	0.404 0.204 0.142	Farber *et al.* (1990)
UHT milk	12	GT in 4.7 hours	0.337	Rajkowski *et al.* (1994)

CHEESE
Low acid soft cheese

TYPE	TEMP. (°C)	GROWTH RATE	EGR AT 5°C (LOG_{10} CFU/DAY)	REFERENCE
Feta	4	Survival > 90 days Scott A 1.28 log **decr**. 3.07 log in 90 days	0 -0.034	Papageorgiou and Marth (1989a)
Mozzarella	5	4 log in 21 days	0.190	Stecchini *et al.* (1995)
Brie	4	0.6 log in 30 days 0.6 log in 14 days	0.020 0.043	Genigeorgis *et al.* (1991)
Feta	4	>2.0 log **decr**. in 8 days >2.0 log **decr**. in 8 days >2.0 log **decr**. in 8 days	-0.250 -0.250 -0.250	
Camembert	6 ripening	4 log in 45 days	0.066	Ryser and Marth (1987b)
Camembert	4	2 to 3 log **decr.** in 365 days	-0.007	Farber *et al.* (1987)
Camembert	3 6 10	0.9 log in 10 days 1.5 log in 15 days 2.4 log in 15 days	0.197 0.074 0.049	Back *et al.* (1993)
Blue cheese	5	Decr. during storage 3 log in 56 days	-0.054	Papageorgiou and Marth (1989b)
Camembert	14	4.5 log in 34 days	0.022	Sulzer and Busse (1993)
Blue cheese	4	>2.0 log **decr.** in 36 days	-0.056	Genigeorgis *et al.* (1991)
Camembert	4	0.64 log in 36 days	0.018	

TYPE	TEMP. (°C)	GROWTH RATE	EGR AT 5°C (LOG_{10} CFU/DAY)	REFERENCE
Queso blanco	4	1.4 log in 14 days	0.142	Glass *et al.* (1995)
Queso fresco	3 7	0.13 log in 1 day 0.5 log in 1 day	0.284 0.285	Mendoza-Yepes *et al.* (1999)
Queso fresco, Queso Ranchero Queso Panella	4	2.0 log **decr**. in 30 days 0.8 log **decr** in 10 days 0.3 log in 30 days 0.3 log in 18 days 2.13 log in 10 days 0.21 log in 30 days 0.44 log in 36 days	-0.067 -0.080 0.010 0.017 0.212 0.007 0.012	Genigeorgis *et al.* (1991)

Soft unripened cheese

TYPE	TEMP. (°C)	GROWTH RATE	EGR AT 5°C (LOG_{10} CFU/DAY)	REFERENCE
Cottage cheese (multiple brands)	8 4	0.59 log in 18 days 1.87 log **decr**. in 36 days 0.42 log in 24 days 1.13 log in 8 days 1.87 log **decr.** in 8 days 0.39 log in 24 days 0.34 log in 24 days 0.41 log in 16 days 0.94 log in 36 days 1.87 log **decr**. in 8 days	0.015 -0.024 0.007 0.064 -0.106 0.023 0.020 0.036 0.037 -0.333	Genigeorgis *et al.* (1991)
Teleme	8 4	2.2 log in 36 days 0.42 log **decr**. in 36 days	0.028 -0.017	Genigeorgis *et al.* (1991)
Ricotta (3 brands)	8 4	2.11 log in 8 days 1.75 log in 6 days 1.88 log in 8 days 1.53 log in 30 days 3.58 log in 36 days 1.97 log in 22 days	0.120 0.132 0.106 0.072 0.141 0.127	Genigeorgis *et al.* (1991)
Cream cheese	8 4	2.0 log **decr**. in 30 days 2.0 log **decr**. in 36 days >2.0 log **decr**. in 36 days	-0.030 -0.079 -0.056	Genigeorgis *et al.* (1991)
Cream cheese	4	2 log in 2 days	1.423	Cottin *et al.* (1990)
Ricotta(whey)	5 12	16.2 – 20.2 hours GT 5.1 – 5.8 hours GT	0.397 0.292	Papageorgiou *et al.* (1996)
Cottage cheese	4 7	2.0 log in 40 days 2.4 log in 10 days	0.071 0.137	Chen and Hotchkiss (1993)
Cottage cheese	5	2 log in 22 days	0.091	Fedio *et al.* (1994)
Cottage cheese	'refrigerated' 6	0.5 – 1.5 log **decr**. in 1 to 5 weeks 1 log **decr**. in 21 days	-0.048 -0.035	El-Shenawy and Marth (1990)

Semi-soft Cheese

TYPE	TEMP. (°C)	GROWTH RATE	EGR AT 5°C (LOG_{10} CFU/DAY)	REFERENCE
Brick (surface ripened)	10	1 to 7-fold decrease in 20 weeks	-0.043	Ryser and Marth (1989)
Tilsiter, Trappist Havarti, Limburger	10	<1 log in 20 weeks	0.015	Ryser and Marth (1989)
Trappist	10	Initial 1 log during ripening stable 30 days, 1 log decr. for 90 days	-0.011	Kovincic *et al.* (1991)
Gouda	NS	Survival 6 weeks	0.000	Northolt *et al.* (1988)
Monterey Jack	4	>2.1 log decr. in 30 days	-0.070	Genigeorgis *et al.* (1991)
Limburger	4	2.26 log decr. in 36 days	-0.064	
Provolone	4	2.36 log decr. in 36 days	-0.066	
String cheese	4	2.29 log decr. in 36 days	-0.064	
Muenster	4	2.0 log decr. in 36 days	-0.056	

Hard cheese

TYPE	TEMP. (°C)	GROWTH RATE	EGR AT 5°C (LOG_{10} CFU/DAY)	REFERENCE
*****Stilton (packed under modified atmosphere)	4	0.7 log in 6 weeks	0.0285	Whitley *et al.* (2000)
Colby	4	1.5 log decr. in 100 days	-0.053	Yousef and Marth (1988)
Cheddar	13	2 log decr. in 75-150 days	-0.003	Ryser and Marth (1987a)
Swiss	7	4 log decr. in 10 days	-0.228	Buazzi *et al.* (1992)**
Parmesan	NS	2 log decr. in 40 days 2 log decr. in 80 days	-0.048 -0.025	Yousef and Marth (1990)
Swiss Cheddar (Cracker Barrel) Cheddar, mild Cheddar, sharp Colby	4 4 4 4 4	>2.1 log decr. in 36 days 1.17 log decr. in 34 days >2.1 log decr. in 30 days >2.1 log decr. in 36 days 0.81 log decr. in 36 days	-0.058 -0.049 -0.070 -0.058 -0.022	Genigeorgis *et al.* (1991)

***** Survival or growth observed, **complete inactivation 66-80 days ripening at 24°C).

Processed cheese

TYPE	TEMP. (°C)	GROWTH RATE	EGR AT 5°C (LOG_{10} CFU/DAY)	REFERENCE
American processed	4	0.18 log **decr**. in 36 days	-0.005	Genigeorgis *et al.* (1991)
Monterey Jack processed	4	1.84 log **decr**. in 36 days	-0.051	Genigeorgis *et al.* (1991)
Piedmont processed	4	1.62 log **decr**. in 36 days	-0.045	Genigeorgis *et al.* (1991)
Pasteurised process cheese	NS	0.6 log **decr**. in 96 hours	-0.15	Glass *et al.* (1998)
Cold pack cheese (non-acid)	3	0.5 log **decr**. in 110 days	-0.004	Ryser and Marth (1988)
Cold pack (acidified or preservatives)	3	1.0 log **decr.** in 60 days	-0.017	Ryser and Marth (1988)

YOGHURT

TYPE	TEMP. (°C)	GROWTH RATE	EGR AT 5°C (LOG_{10} CFU/DAY)	REFERENCE
Yoghurt	4	decr. ~1 log (surv. 4-12 days)	-0.18	Schaack and Marth (1988)
Yoghurt	4	decr ~ 2 log (surv. 21-24 days)	-0.12	Choi *et al.* (1988)
Yoghurt	Not stated	decr. ~ 2 logs in 3-6 days	-0.40	Siragusa and Johnson (1988)

ICE CREAM

TYPE	TEMP. (°C)	GROWTH RATE	EGR AT 5°C (LOG_{10} CFU/DAY)	REFERENCE
Ice cream	-18 to -25	0 logs in 2 months	0.000	Berrang *et al.* (1988)
Soft serve	-18	0 logs in 3 months	0.000	Dean and Zottola (1996)
Ice cream	-18	0 logs in 14 weeks	0.000	Palumbo and Williams (1991)

PROCESSED MEATS

TYPE	TEMP. (°C)	GROWTH RATE	EGR AT 5°C (LOG_{10} CFU/DAY)	REFERENCE
Pâté	4	4 log in 680 hours	0.143	Hudson and Mott (1993a)
Pâté	5	0.361 log in 1 day	0.361	Farber *et al.* (1995)
Bologna	4.4	1 to 2 log in 14 days	0.131	Glass and Doyle (1989)
Corned beef	4.8	0.13 log in 1 day	0.130	Grau and Vanderlinde (1992)
Vacuum-packed ham	5	0.30 log in 1 day	0.300	Grau and Vanderlinde (1992)
Cooked ham	4.4	2 to 3 log in 28 days	0.131	Glass and Doyle (1989)
Cooked ham	7	6 log in 35 days	0.098	Beumer *et al.* (1996)
Vac packed ham Cooked ham	8 8	0.16 log per day 0.2 log per day	0.0725 0.091	Bredholt *et al.* (1999)

COOKED MEATS

TYPE	TEMP. (°C)	GROWTH RATE	EGR AT 5°C (LOG_{10} CFU/DAY)	REFERENCE
Cooked chicken	4.5	19.5 days GT	0.438	Nyati (2000)
Beef sirloin	4.5	21.8 GT	0.392	Nyati (2000)
Roast beef	5 10	5 log in 15 days 5 log in 6 days	0.333 0.254	Grant *et al.* (1993)
Sliced chicken	4.4	4.15 log in 14 days	0.364	Glass and Doyle (1989)
Vac. Packed chicken	4.4	5.90 log in 14 days	0.517	Glass and Doyle (1989)
Chicken breaded fillets	5	0.9 log in 6 days	0.150	Siragusa *et al.* (1988)
Turkey sliced	4.4 4.4	2 log in 14 days 3.11 log in 28 days	0.175 0.136	Glass and Doyle (1989)
Turkey sliced	4.4 4.4	3.08 log in 14 days 3.83 log in 14 days	0.270 0.336	Glass and Doyle (1989)
Vac. packed turkey	4.4	5.09 log in 14 days	0.446	Glass and Doyle (1989)

TYPE	TEMP. (°C)	GROWTH RATE	EGR AT 5°C (LOG_{10} CFU/DAY)	REFERENCE
Cooked beef (aero)	5	11.9 hours GT	0.607	Hudson and Mott (1993a)
Cooked beef (vac)	5	8.7 hours GT	0.83	Hudson and Mott (1993a)

RAW SEAFOOD

TYPE	TEMP. (°C)	GROWTH RATE	EGR AT 5°C (LOG_{10} CFU/DAY)	REFERENCE
Fresh trout	4	1 log in 15 days	0.100	Fernandes *et al.* (1998)
Catfish	4	2 log in 15 days	0.185	Fernandes *et al.* (1998)
Raw shrimp, crab surimi and whitefish	7	GT in 12 hours	0.342	Lovett *et al.* (1990)
Raw oysters	4	No growth in 21 days	0.000	Kaysner *et al.* (1990)
Catfish	4	1-1.5 log in 12 days	0.133	Leung *et al.* (1992)
Raw shrimp & fin fish	Ice-chest	No growth	0.000	Shineman and Harrison (1994)
Raw shrimp and fin fish	Ice-chest	No growth	0.000	Harrison *et al.* (1991)

SMOKED SEAFOOD

TYPE	TEMP. (°C)	GROWTH RATE	EGR AT 5°C (LOG_{10} CFU/DAY)	REFERENCE
Cold-smoked salmon	4 8 4 8	2.1 log in 28 days 5.4 log in 21 days 2.0 log in 21 days 4.6 log in 14 days	0.107 0.116 0.136 0.149	Duffes *et al.* (1999)
Hot-smoked trout	4 8-10	0.5 log in 20 days 6.5 log in 20 days	0.035 0.120	Jemmi and Keusch (1992)
Cold-smoked salmon	5 10	4 log in 650 hours 4-4.5 log in 125 hrs	0.148 0.249	Hudson and Mott (1993b)
Smoked salmon	4 10	3.9 log in 28 days 2.7-4.3 log in 9 days	0.198 0.119	Szabo and Cahill (1999)
Cold-smoked cod	4	2.8 log in 21 days	0.190	Dillon and Patel (1993)
Smoked salmon (26-30°C)	4 10	1.0-1.5 log10 days 3-3.5 log in 10 days	0.177 0.099	Guyer and Jemmi (1991)
Cold-smoked salmon	5 5 10 10	2.5-5 log 40 days 2 log in 40 days 4.5-7 log 10 days 5 log in 11 days	0.092 0.050 0.249 0.139	Pelroy *et al.* (1994a)
Cold-smoked salmon	5 10	4 log in 50 days 4.5 log in 15 days	0.080 0.092	Pelroy *et al.* (1994b)
Cold-smoked salmon	5 5 10 10 10	3 log in 20 days 2.5 log in 20 days 4 log in 7 days 3.7 log in 7 days 6 log in 20 days	0.150 0.125 0.175 0.162 0.092	Peterson *et al.* (1993)
Smoked salmon	4 8	1 log in 10 days 3 log in 14 days	0.142 0.097	Rosso *et al.* (1996)
Cold-smoked salmon	5	5 log in 9 days	0.556	Nilsson *et al.* (1997)

COOKED READY-TO-EAT CRUSTACEA

TYPE	TEMP. (°C)	GROWTH RATE	EGR AT 5°C (LOG_{10} CFU/DAY)	REFERENCE
Pasteurised crabmeat	5	GT in 21.8 hours	0.343	Rawles *et al.* (1995)
Cooked lobster, shrimp, crab and smoked fish	4	2-3 log in 7 days	0.508	Farber (1991)
Pasteurised crabmeat	5	3 log in 10 days	0.300	Buchanan and Klawitter (1992)

VEGETABLES AND FRUIT

TYPE	TEMP. (°C)	GROWTH RATE	EGR AT 5°C (LOG_{10} CFU/DAY)	REFERENCE
Lettuce, whole RTE	5 12	0.0-0.3 log in 7 days 0.0-2.03 log in 7 days	0.043 0.004	Steinbruegge *et al.* (1988)
Lettuce whole RTE, sealed	25	0.0-0.31 log in 7 days	0.002	
Lettuce whole RTE, open	25	0.0-0.35 log in 7 days	0.002	
Lettuce, shredded	5	0.0-0.1 log in 15 days	0.007	Beuchat and Brackett (1990b)
Lettuce, shredded	10	1.5-2.0 log in 3 days	0.204	
Lettuce, whole	10	1.0 log in 15 days	0.067	
Lettuce, butterhead	10	1.5 log in 7 days	0.065	Nguyen-The and Carlin (1994)
Lettuce, lamb's	10	1.0 log **decr.** in 7 days	-0.044	
Lettuce	8	1.5 log in 7 days	0.097	Francis and O'Beirne (2001)
Endive, broad leaved	10	1.0 log in 7 days	0.044	Carlin *et al.* (1996)
Endive, broad leaved	10	1.5 log in 7 days	0.065	Nguyen-The and Carlin (1994)
Endive, curly-leaved	10	0.5 log in 7 days	0.022	
Tomatoes	10 21	No growth (death in chopped tomatoes) Growth	0.0 -	Beuchat and Brackett (1991)
Carrots, whole and shredded	5 15	No growth up to 7 days No growth up to 7 days	0.00 0.00	Beuchat and Brackett (1990a)
Cabbage, raw, shreds	5	4 log in 10 days	0.400	Beuchat *et al.* (1986)
Broccoli	4 15	0.25 -0.5 log in 14 to 21 days 3.0 log in 4 days	0.059 0.109	Berrang *et al.* (1989)
Cauliflower	4 15	$\leq$ 0.25 log in 14 to 21 days 3.0 log in 4 days	0.020 0.109	Berrang *et al.* (1989)
Swede	8	1.75 log in 12 days	0.066	Francis and O'Beirne (2001)
Bean sprout	8	1.75 log in 8 days	0.099	Francis and O'Beirne (2001)
Salads, mixed, prepacked, including fruit and nuts	4	0.30 log in 4 days	0.106	Sizmur and Walker (1988)
Fruit				
Orange juice	4	1.0 log in 35 days (pH 5.0)	0.041	Parish and Higgins (1989)
Apple slices (fresh cut)	5 10	0 log in 6 days 2 log in 6 days	0 0.102 (air)	Conway *et al.* (2000)

TYPE	TEMP. (°C)	GROWTH RATE	EGR AT 5°C (LOG_{10} CFU/DAY)	REFERENCE
Apple slices (fresh cut)	5 10	0 log in 4 days 2.8 log in 10 days	0 0.086 (microaero)	Conway *et al.* (2000)

DELI-SALADS

TYPE	TEMP. (°C)	GROWTH RATE	EGR AT 5°C (LOG_{10} CFU/DAY)	REFERENCE
Crab salad (store prep.)	5	1 log in 10 days	0.100	Eblen (2002)
Shrimp salad (store prep.)	5	2 log in 14 days	0.143	
Shrimp salad (plant prep.)	5	No change	0.0	
Chicken salad: (store prep); (plant prep.)	5	 No change 3 log **decr.** in 18 days	 0.0 -0.167	
Potato salad: (store prep); (plant prep)	5	 2 log **decr.** in 13 days 3 log **decr.** in 10 days	 -0.154 -0.333	
Coleslaw: (store prep); (plant prep)	5	 3 log **decr.** in 13 days 3 log **decr**. in 6 days	 -0.231 -0.500	Eblen (2002)
Egg salad (store prep.)	5	No change	0.0	
Tuna salad (store)	5	No change	0.0	
Ham salad (store)	5	3 log **decr**. in 13 days	-0.231	
Imitation crab salad (store)	5	3 log **decr**. in 19 days	-0.158	
Chicken salad: (high pH); (low pH)	4	 1 log **decr.** in 20 days 1 log **decr**. in 7 days	 -0.050 -0.143	Johnson (1993)
Potato salad: (high pH); (low pH)	4	 1 log **decr**. in 20 days 1 log **decr**. in 4 days	 -0.050 -0.250	
Pasta salad: (high pH); (low pH)	4	 1 log **decr.** in 9 days 1 log **decr.** in 6 days	 -0.111 -0.250	
Seafood salad (high pH); (low pH)	4	 1 log **decr**. in 23 days 1 log **decr**. in 23 days	 -0.043 -0.043	

APPENDIX D: *T. GONDII* CASE CONTROL STUDIES

Table 14 summarises the results of several case control studies investigating the risk factors for *T. gondii* infection among pregnant women. This table only lists significant risk factors related to foodborne, waterborne, zoonotic and/or environmental transmission. Non-significant risk factors, and risk factors associated with demographics, level of education, employment, travel, etc., are not included.

Table 14. Case control studies investigating the risk factors for *T. gondii* infection among pregnant women

COUNTRY	COHORT	RISK FACTORS: UNIVARIATE[1]	RISK FACTORS: MULTIVARIATE[1]	REFERENCE
Japan	459 seropositive pregnant women 4007 seronegative pregnant women[2]	History of raw meat intake (OR not reported, 95% CI 0.54-0.94; *P*=0.02)[3]	History of raw meat intake (by people residing in one region) (OR not reported, 95% CI 0.36-0.79; *P*=0.0015)	Sakikawa *et al.* (2012)
France	80 seroconverted pregnant women 80 seronegative pregnant women	Consumption of rare or medium beef (OR 5.8, 95% CI 2.4-17.0) Consumption of rare or medium lamb (OR 5.2, 95% CI 2.0-17.3) Consumption of undercooked meat outside the home (OR 8.3, 95%CI 2.5-43.1) Never or rarely freezing meat (OR 2.0, 95% CI 1.0-4.2) Raw vegetables frequently eating outside the home (OR 2.8, 95% CI 1.4-5.9)	Consumption of undercooked beef (OR 5.5, 95% CI 1.1-27) Raw vegetables eaten outside home (OR 3.1, 95% CI 1.2-7.7) Having a pet cat (OR 4.5, 95% CI 1.0-19.9)	Baril *et al.* (1999)
Italy	42 IgM-positive pregnant women (indicating recent *T. gondii* infection) 2906 seronegative pregnant women	Eating cured pork more than once a week or once a month (OR 2.9, 95% CI 1.6-5.5) Eating raw meat more than once a week or once a month (OR 2.6, 95% CI 1.4-4.7) Gardening once a week or once a month (OR 2.0, 95% CI 1.1-3.7)	Different models, with different denominators, were used. Eating cured pork or raw meat were consistent risk factors across the models.	Buffolano *et al.* (1996)

COUNTRY	COHORT	RISK FACTORS: UNIVARIATE[1]	RISK FACTORS: MULTIVARIATE[1]	REFERENCE
Norway	63 seroconverted or IgM-positive pregnant women 128 seronegative pregnant women	Living with a cat: – Aged <1 year (OR 3.6, 95% CI 1.1-12.2, *P*=0.04) – Fed raw meat scraps (OR 9.3, 95% CI 1.1-82.0, *P*=0.04) – That used a cat litter box (OR 6.7, 95% CI 1.9-24.3, *P*=0.003) Cleaning cat litter box (OR 5.9, 95% CI 1.6-21.3, *P*=0.007) Preferring beef raw or rare (OR 3.5, 95% CI 1.1-10.7, *P*=0.03) Eating raw or undercooked: – Mutton (OR 7.1, 95% CI 2.0-25.7, *P*=0.003) – Pork (OR 4.7, 95%CI 1.8-12.3, *P*=0.001) – Minced meat (OR 3.2, 95% CI 1.5-6.6, *P*=0.002) – Poultry (OR 8.9, 95% CI 1.9-41.5, *P*=0.005) Tasting raw meat while preparing food (OR 5.6, 95% CI 2.4-13.1, *P*<0.001) Eating tartar (OR 4.6, 95% CI 1.4-15.1, *P*=0.01) Washing knives infrequently (OR 4.6, 95% CI 1.2-17.6, *P*=0.03) Washing cutting boards/chopping blocks infrequently (OR 4.0, 95% CI 1.0-15.6, *P*=0.05) Eating unwashed, raw vegetables (OR 5.7, 95% CI 2.0-15.7, *P*<0.001) Eating unwashed, unpeeled fruits (OR 2.4, 95% CI 1.2-4.8, *P*=0.01)	Eating: – Raw or undercooked minced meat (OR 4.1, 95% CI 1.5-11.2, *P*=0.007) – Unwashed raw vegetables or fruits (OR 2.4, 95% CI 1.1-5.6, *P*=0.03) – Raw or undercooked mutton (OR 11.4, 95% CI 2.1-63.1, *P*=0.005) – Raw or undercooked pork (OR 3.4, 95% CI 1.1-10.4, *P*=0.03) Cleaning the cat litter box (OR 5.5, 95% CI 1.3-22.7, P=0.02) Washing the kitchen knives infrequently after preparation of raw meat, prior to handling another food item (OR 7.3, 95% CI 1.1-50.2, *P*=0.04)	Kapperud *et al.* (1996)

COUNTRY	COHORT	RISK FACTORS: UNIVARIATE[1]	RISK FACTORS: MULTIVARIATE[1]	REFERENCE
Europe (multi-centre)	252 seroconverted or IgM-positive pregnant women 858 seronegative pregnant women	Eating raw sausage ≥1/week (OR 3.2, 95% CI 1.2-9.0, *P*=0.02) Eating dry to cured meat ≥1/week (OR 1.8, 95% CI 1.2-2.8, *P*=0.01) Eating salami ≥1/week (OR 1.6, 95% CI 1.1-2.4, *P*=0.02) Eating raw/undercooked beef (OR 2.4, 95% CI 1.6-3.4, *P*<0.001) Eating raw/undercooked lamb (OR 3.2, 95% CI 1.5-6.9, *P*=0.002) Eating other raw/undercooked meat (OR 3.9, 95% CI 1.6-9.5, *P*=0.003)[4] Taste meat cooking <1/week (OR 2.3, 95% CI 1.5-3.4, *P*<0.001) Taste meat cooking ≥1/week (OR 4.7, 95% CI 2.1-10.9, *P*<0.001) Consuming unpasteurised milk (OR 1.8, 95% CI 1.1-2.9, *P*=0.01) Consuming untreated water (OR 1.7, 95% CI 1.1-2.6, *P*=0.02) Contact with soil (OR 1.9, 95% CI 1.1-2.8, *P*<0.001) Working with animals (OR 1.9, 95% CI 1.1-3.0, P=0.01)	Eating raw/undercooked beef (OR 1.73, 95% CI 1.1-7.2, *P*=0.01) Eating raw/undercooked lamb (OR 3.13, 95% CI 1.4-7.2, *P*=0.007) Eating "other" meat (OR 4.12, 95% CI 1.6-10.9, *P*=0.004)[4] Contact with soil (OR 1.81, 95% CI 1.2-2.7, *P*=0.005)	Cook *et al.* (2000)
UK	452 seropositive (IgG-positive) pregnant women 2158 seronegative (IgG-negative) pregnant women	Eat undercooked or rare meat (OR 1.34, 95% CI 1.14-1.71, P=0.001) Drink unpasteurised milk (OR 1.51, 95% CI 1.12-2.03, P=0.006)[5]	Eat undercooked or rare meat (OR 1.64, 95% CI 1.29-2.08, p<0.0001) Drink unpasteurised milk (OR 1.38, 95% CI 1.01-1.88, p=0.05)	Flatt and Shetty (2013)

[1] OR, odds ratio; 95% CI, 95% confidence interval; p-values listed when reported (usually the level of significance is treated as $P \leq 0.05$).

[2] 4,035 women completed the interview on risk factors but it is not clear how these were distributed in the two groups.

[3] Cat ownership was almost significant (OR not reported, 95% CI 0.68-1.00; *P*=0.05).

[4] Other meat includes venison, horse, rabbit, whale and game birds.

[5] Being vegetarian was protective, OR 0.67 (95% CI 0.45-0.99, p=0.04). Eating unpasteurised or soft cheeses was not a significant risk factor in the univariate or multivariate analysis.

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