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[redacted]

Response to your request for official information

Thank you for your request for information under the Official Information Act 1982 (the Act) on 26 February 2019 for:

> *"Any communication (including but not limited to emails, text messages, reports, memos, minutes, or other official correspondence) relating to the Eating and Activity Guidelines Series, between the Ministry of Health and the below:*
>
> *- New Zealand Beef and Lamb/ Beef + Lamb NZ (and any representatives of)*
> *- Fonterra (and any representatives of)*
> *- Unilever (and any representatives of)*
> *- Ministry for Primary Industries*
>
> *To clarify I am asking for any correspondence between any of these entities relating to the development and or ongoing evaluation of the Eating and Activity Guidelines Series."*

On 13 March 2019 you confirmed that the request was intended to capture any communications between the Ministry and these groups relating to the development of the guidelines and any discussions since then regarding the development of subsequent guidelines.

Fifty documents have been identified within scope of your request. These are itemised in Appendix 1 to this letter, and copies of the documents are enclosed.

I trust that this information fulfils your request. Under section 28(3) of the Act you have the right to ask the Ombudsman to review any decisions made under this request.

Yours sincerely

D Woodley

Deborah Woodley
Deputy Director-General
Population Health and Prevention

MINISTRY OF HEALTH
MANATŪ HAUORA

Appendix 1: List of documents for release

#	Date	Title	Decision on release
1	16 April 2014	Email: Draft Eating and Activity Guidelines statements Feedback	Partially released. Some information is withheld pursuant to section 9(2)(a) to protect the privacy of natural persons.
2	16 April 2014	Attachment: Unilever New Zealand FEEDBACK NZ Eating Guideline Statements April 2014	Released in full.
3	22 April 2014	Email: Fonterra Feedback to MoH draft eating guidelines statements	Partially released. Some information is withheld pursuant to section 9(2)(a).
4	22 April 2014	Attachment: MoH Draft Eating and Activity Guidelines Statements Fonterra Brands 170414	Released in full.
5	4 March 2015	Email: Eating and Activity Guidelines	Partially released. Some information is withheld pursuant to section 9(2)(a).
6	4 March 2015	Email: RE: Role of Red Meat report feedback	Partially released. Some information is withheld pursuant to section 9(2)(a).
7	4 March 2015	Attachment: Role of Red Meat Final Draft Feb 15	Released in full.
8	19 March 2014	Email: MPI Opportunity to update you/your team	Released in full.
9	31 March 2014	Email: Limited stakeholder email to MPI	Released in full.
10	31 March 2014	Attachment: Long activity statements 26 Mar 2014 Attachment: Basic guidelines statements 26 March 2014	Released in full.
11	8 December 2014	Email: Food Safety Pages to MPI	Released in full.
12	8 December 2014	Attachment: Draft Eating statement 5	Released in full.
13	9 October 2014	Email: Food Safety during Pregnancy	Partially released. Some information is withheld pursuant to section 9(2)(a).
14	9 October 2014	Attachment: FW09089 Food Safety During Pregnancy CARC_08_02	Released in full.
15	9 February 2015	Email: Food safety pages to MPI v2 - feedback due Tues 24 Feb	Released in full.

#	Date	Title	Decision on release
16	9 February 2015	Attachment: Food Safety section 9 Feb 2015	Released in full.
17	20 October 2015	Email: *Confidential: Eating and Activity Guidelines for New Zealand Adults - Embargoed 28 October 2015	Released in full.
18	20 October 2015	Attachment: Eating Activity Guidelines	Withheld in full. This document is publicly available and can be found by searching "Eating Activity Guidelines" on www.health.govt.nz
19	5 March 2015	Email: RE: Review of the Eating and Activity Guidelines document between 2 and 13 March 2015	Partially released. Some information is withheld pursuant to section 9(2)(a).
20	5 March 2015	Attachment: Food Safety from KK and RLC Attachment: Untitled 1 Attachment: Untitled 2	Released in full.
21	13 March 2015	Email: RE: Review of the Eating and Activity Guidelines document between 2 and 13 March 2015	Released in full.
22	13 March 2015	Attachment: 20150313 MPI submission MOH Eating and Activity Guidelines	Released in full.
23	23 March 2015	Email: RE MPI's Feedback on EAG core document.	Released in full.
24	10 September 2015	Email: RE_ Meeting to discuss changes to EAG document	Partially released. Some information is withheld pursuant to section 9(2)(a).
25	22 June 2015	Email:Re: Feedback on MPI comments re EAGS	Partially released. Some information is withheld pursuant to section 9(2)(a).
26	22 June 2015	Attachment: EAG v14 22 June 2014	Released in full.
27	26 June 2015	Email: Re: MPI comments on the MoH Eating and Activity Guidelines	Released in full.
28	12 August 2015	Email: RE: Catch up re Eating and Activity Guidelines	Partially released. Some information is withheld pursuant to section 9(2)(a).
29	17 September 2015	Email: Embargoed copy of Eating and Actvity Guidelines for New Zealand Adults	Released in full.
30	17 September 2015	Attachment: EAG v15	Released in full.

#	Date	Title	Decision on release
31	22 September 2015	Email: Re EAG and HSR	Released in full.
32	30 September 2015	Email: Re: Bugs	Released in full.
33	30 September 2015	Attachment: Re: Bugs	Released in full.
34	12 June 2015	Email: Re: Feedback on MPI comments reEAGs	Partially released. Some information is withheld pursuant to section 9(2)(a).
35	8 December 2015	Email: Key Messages for MPI	Released in full.
36	8 December 2015	Attachment: Key Messages for MPI	Released in full.
37	24 September 2014	Email: Fw: 3 Dairy a Day and NZ Food and Nutrition Guidelines	Partially released. Some information is withheld pursuant to section 9(2)(a).
38	10 February 2015	Email: RE: Information on the new Eating and Activity Guidelines Series and the update of the adult nutrition guidelines.	Partially released. Some information is withheld pursuant to section 9(2)(a).
39	10 February 2015	Attachment: Updated Red Meat Report first draft with reviewer changes_Nov14	Released in full.
40	17 May 2018	Email: Re: WCRF 3rd expert review.	Partially released. Some information is withheld pursuant to section 9(2)(a).
41	11 July 2018	Email: Agenda for Joint MOH MPI Catch up	Released in full.
42	11 July 2018	Attachment: Agenda for Joint MOH MPI Catch up	Partially released. Information outside of the scope of your request has been withheld.
43	25 July 2018	Email: Agenda for joint meeting	Released in full.
44	25 July 2018	Attachment: Agenda for joint meeting	Partially released. Information outside of the scope of your request has been withheld.
45	31 July 2018	Email: Re: Red meat recommendation in the media	Partially released. Some information is withheld pursuant to section 9(2)(a).
46	3 August 2018	Email: Re: Red meat recommendation in the media	Partially released. Some information is withheld pursuant to section 9(2)(a).

#	Date	Title	Decision on release
47	6 November 2018	Email: Re: Quarterly Catch up MPI and MOH	Partially released. Some information is withheld pursuant to section 9(2)(a).
48	31 January 2019	Email: Policy Guideline for FRSC with regard to dietary Guidelines	Partially released. Some information is withheld pursuant to section 9(2)(a).
49	31 January 2019	Attachment: First draft- Policy Guideline	Released in full.
50	20 February 2019	Email Re_ FW_ List of food safety related topics we are proposing MPI	Released in full.
51	6 June 2014	Email: Re Eating and Activity Guidelines	Partially released. Some information is withheld pursuant to section 9(2)(a).
52	11 March 2015	Email: Fw: Response from BLNZ on EAGs for NZ adults review	Partially released. Some information is withheld pursuant to section 9(2)(a). Information that is out of scope of your request is also withheld.
53	11 March 2015	Email: Re: Response from BLNZ on EAGs for NZ adults review	Partially released. Some information is withheld pursuant to section 9(2)(a).
54	11 March 2015	Attachment: Response to MOH EAGS 100315	Released in full.
55	11 March 2015	Attachment: Response to MOH EAGS March 2015 MPA	Released in full.
56	11 March 2015	Attachment: EAGS v10b F. Greig BLNZ Feedback	Released in full.
57	11 March 2015	Attachment: ANA Abstract Pacific Posters Nov 2012	Released in full.
58	11 March 2015	Attachment: 2013 Beef and Lamb Nutrients Study Published	Released in full.
59	11 March 2015	Attachment: NZMJ paper 251105 Laugesen	Released in full.
60	11 March 2015	Attachment: Proceed Protein Reqs	Released in full.
61	11 March 2015	Attachment: Role of Red Meat Final	This is the same as document 39 included in this response.

Sent by: katherine.tocchini@unilever.com
16/04/2014 12:39 p.m.
To: "Louise_McIntyre@moh.govt.nz" <Louise_McIntyre@moh.govt.nz>,
cc:
bcc:

Subject: Draft Eating and Activity Guidelines statements Feedback

Hi Louise,

Please see attached Unilever New Zealand's feedback on the draft Eating and Activity Guidelines statements.

Unilever recommends that the draft Eating Guidelines Statements be revised and we have provided constructive recommendations for consideration by the Ministry of Health. Our comments are provided in good faith and we trust that they will be fully considered under the due process.

Kind regards, Katherine.

Katherine Tocchini Nutrition & Health Manager - ANZ

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Unilever New Zealand

FEEDBACK NZ Eating Guideline Statements April 2014.pdf

Unilever New Zealand
FEEDBACK
April 2014

To:

Louise McIntyre Advisor

(Nutrition) Nutrition & Physical Activity Policy Public Health Clinical Leadership, Protection & Regulation

New Zealand Ministry of Health

In response to:

Draft New Zealand Eating Guideline Statements 2014

Unilever New Zealand Contact:

Katherine Tocchini

Nutrition & Health Manager

Unilever New Zealand

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Email: katherine.tocchini@unilever.com

CONTENTS

2.0 EXECUTIVE SUMMARY

Unilever New Zealand (Unilever) contends that the science on the important role of polyunsaturated fats in reducing risk of heart disease has strengthened but the draft Eating Guidelines Statements (EGS) advice on polyunsaturated fats to clinicians and consumers does not reflect this.

Since the 1950s, the evidence has clarified and increased to support guidelines that recommend saturated fats need to be replaced with polyunsaturated fats to reduce the risk of coronary heart disease. Since 2003 the evidence has strengthened considerably.

Unilever believes that the guidance to choose foods with minimal fat (Guideline 3) is a continuation of focusing on fat consumption in weight management, rather than energy balance and does not reflect current scientific evidence.

Coronary heart disease (CHD) risk is reduced when saturated fats are replaced with polyunsaturated fats, including vegetable oils, margarines, salad dressings, mayonnaise, nuts and seeds. According to the 2010 Ministry of Health Mortality data, cardiovascular disease remains to be the leading cause of death in New Zealand, accounting for 30% of deaths annually.

Many international public health organisations, including the World Health Organization (WHO), emphasise the major role of fat consumption in the development of obesity and of reducing fat intake in the dietary management of obesity or overweight. More recently, WHO has shifted its emphasis, saying there is convincing evidence that energy balance is critical to maintaining healthy weight and ensuring optimal nutrient intakes, regardless of macronutrient distribution and percentage of total fat. [1]

In addition, the WHO/FAO expert consultation on fats and fatty acids in human nutrition (2010) reported convincing evidence that replacing SFA [Saturated Fatty Acids] with PUFA [Polyunsaturated Fatty Acids] decreases the risk of CHD.

[1] *Food and Agriculture Organization of the United Nations and the World Health Organization, FAO/WHO Expert Consultation on Fats and Fatty Acids in Human Nutrition, 2008, Food and Agriculture Organization of the United Nations and the World Health Organization,.*

The manner in which polyunsaturated fats are currently presented is likely to send consumers the wrong message about polyunsaturated fats and oils and deter them from consuming these foods as part of a healthy, balanced diet. The advice should be strengthened to correctly inform consumers on the important nutritional choices they should be making to reduce risk of heart disease and more broadly ensure healthier lives.

Moreover, the draft EGSs are impractical and do not reflect that the majority of the New Zealand population are not meeting current dietary recommendations for polyunsaturated fats.

In addition the draft EGSs are not in keeping with best international practice. Recently revised dietary guidelines published in Australia, Canada and the United States highlight the need to replace saturated (or bad) fats with polyunsaturated (good) fats and provide clear guidance on servings per day.

Unilever understands that the EGSs are integral to enabling New Zealanders to make the right food choices. All New Zealanders deserve to be provided with the best evidence-based guidance to make better dietary decisions about dietary fats.

Therefore, there needs to be further revision of Guideline 2 & 3 in the draft EGSs .

3.0 INTRODUCTION

Unilever welcomes the opportunity to provide feedback to the Eating Guideline Statements.

Unilever has consistently supported the intent of, and recognised the importance of dietary guidelines. As a major food manufacturer, we are fully committed to assisting New Zealanders to make healthy dietary decisions based on the best available evidence.

However, Unileve is disappointed that despite the current scientific evidence on the important role of polyunsaturated fats in the prevention of heart disease the EGSs continues the outdated emphasis on fat consumption in weight management, rather than energy balance.

Unilever believes that the draft EGSs are not supported by the current scientific evidence for polyunsaturated fats in the diet and are inconsistent with the *Dietary Guidelines for Australians, Americans* and *Canadians*. These recently reviewed Guidelines provide clear advice to replace saturated fats with unsaturated fats and include food-based recommendations and servings per day. A summary of dietary fats and health relationship is included in *Appendix 1.*

There needs to be further revision of Guideline 2 & 3 to ensure that the intended effect on public health occurs, which is to assist in reducing New Zealanders risk of developing coronary heart disease (CHD).

Our submission discusses:

- Contemporary scientific evidence on replacement of saturated fat with polyunsaturated fat and prevention of cardiovascular disease;
- The New Zealand population is not meeting the current recommended dietary intake for polyunsaturated fats;
- The importance of alignment with other international recommendations.

Unilever recommends that the draft EGSs be revised and we have provided constructive recommendations for consideration by the Ministry of Health. Our comments are provided in good faith and we trust that they will be fully considered under the due process.

4.0 KEY ISSUES

4.1 Guideline 3 does not reflect contemporary scientific evidence on replacement of saturated fat with polyunsaturated fat and prevention of cardiovascular disease.

Unilever believes that there is strong contemporary scientific evidence that clearly shows that risk of coronary heart disease (CHD) can be reduced by replacement of saturated fat with polyunsaturated fat.

The WHO/FAO expert consultation on fats and fatty acids in human nutrition (2010) reported, '*convincing evidence that replacing SFA [Saturated Fatty Acids] with PUFA [Polyunsaturated Fatty Acids] decreases the risk of CHD.' The report also found that 'there is probable evidence that replacing SFA with largely refined carbohydrates has no benefit on CHD and may even increase the risk of CHD and favour metabolic syndrome development*.

However, it appears that the advice in Guideline 3 weakly communicates the replacement message and may compromise consumers' ability to understand the protective effect offered by polyunsaturated fats (to reduce cardiovascular risk). This decision will impact current recommendations for unsaturated fat consumption, including the New Zealand Heart Foundation recommendations.

The recommendations included in the *WHO/FAO expert consultation on fats and fatty acids in human nutrition (2010)* are based on consistent findings from epidemiologic, clinical and mechanistic studies.

The following key references demonstrate the importance of the replacement message:

- ***WHO/FAO expert consultation on fats and fatty acids in human nutrition 2010***
  This report outlines convincing evidence that replacing saturated fat (SAFA) with polyunsaturated fat (PUFA) lowers the risk for coronary heart disease.

- ***Mensink RP, Zock PL, Kester AD et al. Effects of dietary fatty acids and carbohydrate on the ratio of serum total to HDL cholesterol and on serum lipids and apolipoproteins: a meta-analysis of 60 controlled trials. AM J Clin Nutr 2003;77:1146-55***

Replacing SAFA with unsaturated fats lowers the serum LDL-cholesterol concentration and the ratio of total/HDL-cholesterol, both of which are associated with lower risk for coronary heart disease.

- ***Mozaffarian D, Micha R, Wallace S.Effects on Coronary Heart Disease of Increasing Polyunsaturated Fat in Place of Saturated Fat: A Systematic Review and Meta-Analysis of Randomized Controlled Trials. PLoS Med 2010;7(3): e1000252.***

  Replacing SAFA with PUFA is also associated with reductions in levels of numerous markers of inflammation. A meta-analysis of randomised controlled trials showed the replacement of SAFA with PUFA to be associated with lower risk for coronary heart disease events.

- **Astrup A et al. The role of reducing intakes of saturated fats in the prevention of cardiovascular disease: where does the evidence stand in 2010? Am J Clin Nutr 2011;93:684-688.**

  The main conclusion of this expert panel is that the current evidence from epidemiologic, clinical, and mechanistic studies is consistent in finding that: 1) the risk of CHD is reduced when SFAs are replaced with polyunsaturated fatty acids (PUFAs). 2) No clear benefit of substituting carbohydrates for SFAs has been shown, although there might be a benefit if the type of carbohydrate replacing SFA is unrefined and has a low glycemic index. 3) Sufficient evidence does not yet exist to judge the effect on CHD risk of replacing SFAs with MUFAs.

- ***Jakobsen MU, O'Reilly EJ, Heitmann BL, Pereira MA, Balter K, Fraser GE, Goldbourt U, Hallmasn G, Knekt P, Liu S, Pietinen P, Spiegelman D, Stevens J, Virtamo J, Willett WC, Ascerio A. Major types of dietary fat and risk of cornonary heart disease: a pooled analysis of 11 cohort studies. Am J Clin Nutr 2009 May;89(5):1425-32.***

  The findings of a pooled analysis of prospective cohort studies were consistent with the randomised controlled trials. However, in this pooled analysis, the replacement of saturated fat by carbohydrate was not associated with lower coronary risk. This is consistent with the similar effects of SAFA and carbohydrate on the serum total/HDL-cholesterol ratio [existing reference 410 (Mensink 2003 AM J CLin Nutr)]. In the absence of randomised controlled trial evidence, it is concluded that no clear benefit of substituting carbohydrates for SAFA has been shown. For coronary heart disease to fall, saturated fats need to be replaced with polyunsaturated fats.

Unilever is disappointed and surprised that key pieces of contemporary research are not reflected, or simply ignored in the draft EGSs. This is of particular concerns given we have been informed that the guidelines have been updated to *"bring them in line with the current published international graded evidence base*", including a review by an external technical advisory group comprising of academics and experts. One can only assume this is due to the fear of dietary fat intake resulting in weight gain, which as previously discussed has no scientific basis.

Unilever recommends that a Scientific Literature Review (SLR), specifically into saturated fat and CHD, be conducted as the significant strengthening of evidence, included in the *The WHO/FAO expert consultation on fats and fatty acids in human nutrition (2010)* has been missed.

Contemporary research demonstrates:

- Replacing saturated fat with carbohydrate, to give a low fat diet, is <u>not</u> associated with a fall in the risk for coronary disease.
- 'Limiting saturated fat' is no longer an evidence-based statement as it implies that substituting saturated fat with carbohydrate provides benefit. It does not.
- Saturated fat needs to be replaced with polyunsaturated fats, for coronary heart disease risk to fall.
- It is no longer good enough to send the message to consumers 'to limit saturated fats.' For coronary heart disease to fall then saturated fats need to be replaced with polyunsaturated fats.

It is therefore questionable as to why guidelines 2 & 3 do not include a recommendation for New Zealanders to reduce saturated fat intake and replace these with polyunsaturated fats.

Recommendation:

The recommendations communicated in the EGSs should be independently reviewed and revised and be based on:

A) A SLR on the relationship between SAFA intake and CHD

Or

B) In the absence of a SLR, the conclusions and recommendations of the following two references should be adopted:

- ***'The role of reducing intakes of saturated fat in the prevention of cardiovascular disease: where does the evidence stand in 2010?'* (Astrup A, et al. AJCN 2011;93:684-8); and**
- ***'Effects on Coronary Heart Disease of Increasing Polyunsaturated Fat in Place of Saturated Fat: A Systematic Review and Meta-Analysis of Randomized Controlled Trials'*. (Mozzaffarian D, et al. PLoS Med 2010;7(3): e1000252.**

The key conclusions and recommendations outlined in these references are:

- **The risk of coronary heart disease is reduced when saturated fatty acids are replaced with polyunsaturated fatty acids.**
- **No clear benefit of substituting carbohydrates for saturated fatty acids has been shown, although there might be a benefit if the type of carbohydrate replacing saturated fatty acids is unrefined and has a low glycemic index.**
- **There is not yet sufficient evidence exists to judge the effect on coronary heart disease risk of replacing saturated fatty acids with monounsaturated fatty acids.**
- **Rather than trying to lower polyunsaturated fat consumption, a shift toward greater population polyunsaturated fat consumption in place of saturated fat would significantly reduce rates of coronary heart disease**

4.2 New Zealanders are not meeting current recommended intakes for polyunsaturated fats.

New Zealander are not consuming enough polyunsaturated fats and advice to eat minimal amounts is not supported by current dietary intake evidence.

Research shows that New Zealand adults are eating more saturated fat and less polyunsaturated fat than current recommendations.

The WHO/FAO expert consultation on fats and fatty acids in human nutrition (2010) minimum recommended value of total polyunsaturated fatty acid consumption for lowering LDL and total cholesterol concentrations, increasing HDL cholesterol concentrations and decreasing the risk of coronary heart disease events is 6% of total energy with an acceptable range for total PUFA (n-6 and n-3 fatty acids) of between 6 and 11% of total energy.[2].

The 2008/09 New Zealand Adult Nutrition Survey[3], funded by the Ministry of Health found that:

- Total fat provided 33.7% and 33.8% of energy for males and females, respectively; this falls within the recommended range of 20–35% energy from total fat,
- Saturated fat provided 13.1% of energy for both males and females; and remains above the recommended 10% contribution of saturated fat to total energy; and
- Polyunsaturated fat provided 4.8% and 4.9% of energy for males and females, respectively.

The proposed Guidelines 2 & 3 do not communicate the benefits of eating healthy polyunsaturated fats every day to replace unhealthy saturated fats and adds to consumer confusion about the need to change. The communication of the benefits of replacing unhealthy fats with healthy fats is vitally important if New Zealanders are to increase their percentage of polyunsaturated fats.

[2] *Food and Agriculture Organization of the United Nations and the World Health Organization, FAO/WHO Expert Consultation on Fats and Fatty Acids in Human Nutrition, 2008, Food and Agriculture Organization of the United Nations and the World Health Organization,.*

[3] University of Otago and Ministry of Health. 2011. *A Focus on Nutrition: Key findings of the 2008/09 New Zealand Adult Nutrition Survey*. Wellington: Ministry of Health.

Recommendation:

To ensure that draft EGSs better reflect contemporary scientific evidence and the New Zealand diet, they should be revised as follows:

Revise the Eating Guidelines statements to include the following:

Adapt Guideline 2 to the following:

- **Include the following new food group as a bullet: *Healthy oil rich foods, including vegetable oils, margarines, dressings, mayonnaise, nuts and seeds .***
- **Remove *nuts and seeds* from the 4th bullet point of *some legumes*, nuts, seeds, fish, eggs, lean poultry or lean red meat.***

Adapt Guideline 2:

- **Reword to the following:**
  Choose and prepare foods and drinks:
  - **low in salt (sodium); if using salt, choose iodised salt**
  - **with little or no added sugar, and**
  - ***replace foods containing saturated fats with foods containing unsaturated fats such as vegetable oils, margarines, dressings, mayonnaise, nuts and seeds.***

4.3 Importance of alignment with international dietary recommendations

Unilever is concerned and must question how on the balance of evidence available that the proposed EGSs differ considerably from other comparable countries' current and recently reviewed dietary guidelines.

The draft EGSs are inconsistent with the Australian, US and Canadian guidelines which provide clear advice to replace saturated fats with unsaturated fats and include food-based recommendations and servings per day.

In order to illustrate our concerns, a comparison of the proposed EGSs with the *Australian Dietary Guidelines (2013)*, *American Dietary Guidelines (2010)* and *Canadian Dietary Guidelines (2007)* is provided at Table 1:

Table 1: International comparison of dietary guidelines

Country	Dietary Fats Guideline
New Zealand EGSs (2013)	Choose and prepare foods and drinks: • with minimal fat, especially saturated fat; if you choose to add fat use plant based oils and spreads
Australian (2013): ADGs	Limit intake of foods high in saturated fat such as many biscuits, cakes, pastries, pies, processed meats, commercial burgers, pizza, fried foods, potato chips, crisps and other savoury snacks. • Replace high fat foods which contain predominately saturated fats such as butter, cream, cooking margarine, coconut and palm oil with foods which contain predominately polyunsaturated and monounsaturated fats such as oils, spreads, nut butters/pastes and avocado.
United States (2010): Dietary Guidelines for Americans[4]	1. Use oils to replace solid fats where possible 2. Consume less than 10 percent of calories from saturated fatty acids by **replacing them with monounsaturated and polyunsaturated fatty**

[4] US Department of Health, "Dietary guidelines for Americans," 2010, http://health.gov/dietaryguidelines/dga2010/DietaryGuidelines2010.pdf, viewed February 2012.

	acids 3. Consume less than 300 mg per day of dietary cholesterol 4. Keep trans fatty acid consumption as low as possible, especially by limiting foods that contain synthetic sources of trans fats, such as partially hydrogenated oils, and by limiting other solid fats
Canada (2007): *Eating Well with* Canada's Food Guide : [5]	• Include a small amount – 30 to 45 mL (2 to 3 Tbsp) – of **unsaturated fat each day. This includes oil used for cooking, salad dressings, margarine and mayonnaise.** • Use vegetable oils such as canola, olive and soybean. • Choose soft margarines that are low in saturated and trans fats. • Limit butter, hard margarine, lard and shortening. • Limit trans fats

Recommendation:

The draft EGSs encourage the consumption of a range of foods that contain unsaturated fats, including vegetable oils, margarines, dressings, mayonnaise, nuts and seeds and provide recommended servings per day.

This can be achieved by revising Guideline 2 and Guideline 3 as stated above.

[5] Canadian Department of Health, "Eating well with Canada's food guide," 2007, http://www.hc-sc.gc.ca/fn-an/alt_formats/hpfb-dgpsa/pdf/food-guide-aliment/view_eatwell_vue_bienmang-eng.pdf, viewed 10 February 2012.

5.0 SUMMARY OF KEY RECOMMENDATIONS

Unilever recommendations to the Ministry in response to the draft Eating Guideline Statements can be summarised as follows:

The research:

The recommendations communicated in the EGSs should be independently reviewed and revised and be based on:

Recommendation:

1. **SLR on the relationship between SAFA and CHD**

   Or

2. **In the absence of a SLR, the recommendations of the following two references should be adopted:**
   - '*The role of reducing intakes of saturated fat in the prevention of cardiovascular disease: where does the evidence stand in 2010?*' (Astrup A, et al. AJCN 2011;93:684-8) [Omitted from ADGs]; and
   - '*Effects on Coronary Heart Disease of Increasing Polyunsaturated Fat in Place of Saturated Fat: A Systematic Review and Meta-Analysis of Randomized Controlled Trials*'. (Mozzaffarian D, et al. PLoS Med 2010;7(3): e1000252[Referenced in ADGs - reference 490].

The advice to clinicians and consumers:

An appropriate solution is for the draft Eating Guideline Statement to encourage the consumption of a range of foods that contain unsaturated fats, including nuts, seeds, vegetable oils, margarines, salad dressings and mayonnaise and provide recommended servings per day.

Practically this can be achieved by making the following amendments to the draft EGSs:

Revise the Eating Guidelines statements to include the following:

Adapt Guideline 2 to the following:

- **Include the following new food group as a bullet: *Healthy oil rich foods, including vegetable oils, margarines, dressings, mayonnaise, nuts and seeds .***
- **Remove *nuts and seeds* from the 4th bullet point of *some legumes*, nuts, seeds, fish, eggs, lean poultry or lean red meat.***

Adapt Guideline 2:

- **Reword to the following:**
  Choose and prepare foods and drinks:
  - **low in salt (sodium); if using salt, choose iodised salt**
  - **with little or no added sugar, and *replace foods containing saturated fats with foods containing unsaturated fats such as vegetable oils, margarines, dressings, mayonnaise, nuts and seeds.***

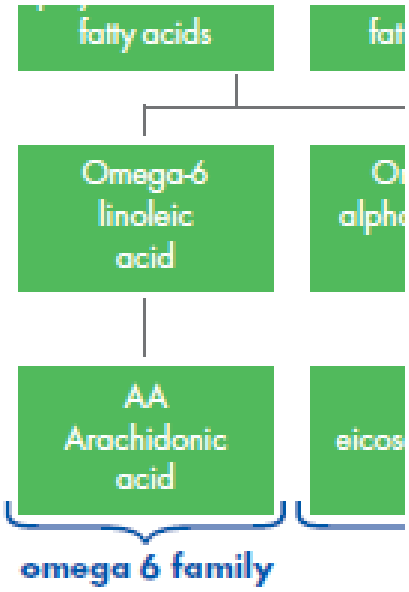
The above recommended changes will ensure the Eating Guideline Statements:

- Reflect contemporary scientific consensus, and live up to Ministry's claim of the guidelines being 'in line with the current published international graded evidence base.'
- Align with the approach of other comparative countries, namely the Australian, US and Canadian Dietary Guidelines.
- Provide practical and meaningful advice to consumers which will enable them to make better diet choices.

6.0 APPENDICES

Appendix 1: Summary of dietary fats and health relationship

Put simply, fats can be divided into two groups – 'good' and 'bad'. 'Good' fats, largely polyunsaturated (PUFA) are beneficial for a healthy heart and 'bad' fats, saturated and trans fats are harmful to heart health. Vegetable omega-3 (alpha-linolenic acid – ALA) and omega-6 PUFA (linoleic acid – LA) are essential fats which muct be provided through the diet because they cannot be made in the body.



Common sources of dietary fat and their effect on cholesterol levels

Fat Type	Health Relationship	Dietary Source
Polyunsaturated fatty acids (PUFA) Omega 6	Intake lowers LDL-C Replacing SFA with omega 6 PUFA reduces risk of CHD.	Sunflower seed oil, margarine, dressings and mayonnaise made from these oils, nuts & seeds,
Polyunsaturated fatty acids (PUFA) Omega 3	Intake reduces triglycerides and LDL-C and increases HDL. Other beneficial health effects.	Plant sources – linseed, soybean & canola oil, nuts & seeds. Marine sources – oily fish such as mackerel, salmon, herrings and fresh tuna.
Monounsaturated fatty acids (MUFA)	There is inconclusive evidence supporting a relationship between dietary cholesterol and CVD outcomes. There is not yet sufficient evidence to judge the effect on CHD risk of replacing SFA's with MUFA.	Olive oil, olive, avocado, nuts
Saturated fatty acids	Increase LDL and triglycerides Intake is associated with CHD	Butter, full fat milk, cheese & cream. Fatty meats and meat products like salami, sausages. Cakes pastries, biscuits, crisps and chocolate.
Trans fatty acids	Increases LDL Is associated with increase CHD incidence and risk for CHD.	Butter and fatty meats, commercial cakes and pastries, biscuits, deep fried fast food and snacks. *Australian intakes of trans fats are low.

Sent by: Vicki.Robinson@huttvalleydhb.org.nz
22/04/2014 08:26 a.m.

To: "Louise_McIntyre@moh.govt.nz" <Louise_McIntyre@moh.govt.nz>,
cc:
bcc:

Subject: FW: Feedback to MoH draft eating guidelines statements

Louise these wee forward to me from Fonterra.

Regards Vicki

From: Mindy Wigzell [mailto:Mindy.Wigzell@fonterra.com]
Sent: Thursday, 17 April 2014 3:30 p.m.
To: Vicki Robinson [HVDHB]
Cc: Dietitians NZ Inc. - Administration
Subject: Feedback to MoH draft eating guidelines statements
Importance: High

Hi Vicky

Please find attached feedback from Fonterra Brands NZ to draft eating guidelines statements from Ministry of Health (specific to milk and milk products only).

I note from the original request from Dietitians NZ that the deadline for submission to you is tomorrow, 18 April. However, I've just noticed a reminder notice suggesting we reply to you by Tuesday 15th. I hope this isn't too late??

Any questions, please let me know.

Many thanks; have a great Easter.

Mindy Wigzell
Innovation Nutritionist
Fonterra Co-operative Group Limited

mindy.wigzell@fonterra.com
tel +649 295 2878 mobile s 9(2)(a)
Private Bag 75806, Manurewa, 2243, Una Place, 2112, Takanini, New Zealand

MoH Draft Eating and Activity Guidelines Statements Fonterra Brands 170414.docx

NZ Ministry of Health Draft Eating and Activity Guidelines Statements 2014 – Feedback from Fonterra Brands NZ

Proposed Eating Guidelines Statements wording, specific to milk and milk products:

"Enjoy a variety of nutritious foods every day including: ...
- some low fat milk products and/or calcium-fortified milk alternatives"

Note previous wording (from existing Food and Nutrition Guidelines for Healthy Adults (2003) and Older People (2013)):

"Have milk and milk products in your diet, preferably reduced or low fat options"

General feedback

- Milk and milk products make a significant contribution of essential nutrients to intakes in New Zealand, and are understood to be the richest dietary source of calcium in particular.
- It is estimated that approximately 65%[1] of children and 59%[2] of adults in New Zealand do not consume the recommended intake of calcium.
- As such, Eating Guidelines Statements should accurately reflect the role of milk and milk products in a healthy eating plan and promote appropriate consumption of this food group.
- Although the specific rationale for the proposed changes to the wording has not been provided, we understand this is based largely on the evidence statements for the Australian Dietary Guidelines[3] and Dietary Guidelines for Americans[4]

Recommended alternative wording:

"Enjoy a variety of nutritious foods every day including:
- milk and milk products and/or calcium-fortified milk alternatives - preferably low or reduced fat"

Issue	Comments	Recommendation
ISSUE ONE: A change from referring to "milk and milk products" to just "milk products"	This could be interpreted as a recommendation to consume products made from milk but not milk itself.	Keep "milk and milk products" for clarity.

ISSUE TWO: A change from promoting consumption of all milk and milk products, with a *preference for low and reduced fat options*, to one that suggests consuming only dairy products which are "low fat"	• We query the evidence base used for a recommendation to completely avoid dairy products which do not meet a "low fat" criteria. Core dairy products (including full fat varieties) eg milk, cheese and yoghurt provide a valuable bundle of nutrients to the diet and are associated with a number of health benefits.[3]
	  • As an example, with regard to the evidence statements for consuming "milk, yoghurt, cheese and/or alternatives, mostly reduced fat" in the 2013 Australian Dietary Guidelines, only one of the ten evidence statement relates specifically to low fat dairy products; the remainder refer to core dairy foods in general.[3]
	We recognise the association between saturated fat and undesirable changes to serum cholesterol levels – a risk factor for cardiovascular disease (CVD). However there is no consistent evidence that consumption of full fat dairy products including milk, cheese or yoghurt (which contain a mixture of fats including saturated fat) cause an increased risk of CVD. In fact, growing evidence suggests dairy product consumption is associated with a reduction in CVD risk, despite its saturated fat content.
	  • FAO (2013) recently concluded that low fat milk and <u>total dairy</u> consumption is generally not associated with cardiovascular disease (CVD) risk and may actually contribute to a reduction of CVD. They also acknowledge that "the effect of particular foods on CHD cannot be predicted solely by their fatty-acid profile and the content of total SFAs, as individual SFAs may have different cardiovascular effects."[5]
	  • Expert bodies such as the Advisory Committee to the 2010 US Dietary Guidelines did not find overall consumption of dairy foods to be linked with increased risk of CVD, in fact they found "moderate evidence also indicates that intake of milk and milk products is associated with a reduced risk of CVD".[6]
	• If the intention is to shift the public away from consuming any full fat milk and milk products, this could result in a substitution of other foods or nutrients. The
		Keep the previous recommendation to consume milk and milk products with a preference for low or reduced fat options.

	replacement of saturated fats with high glycaemic index carbohydrates, for example, can actually worsen CVD risk.[7] • Full fat dairy products are an important contributor to fat soluble vitamin intake including vitamin A. The 2008/09 NZ Adult National Nutrition Survey showed a decrease in NZ adults vitamin A intake between the 1997 and 2008/09 nutrition surveys for both males and females, with a 17.2% prevalence of inadequate vitamin A intake.[2] • "Low fat" by definition in the ANZ Food Standards Code includes only products with 3g of fat or less per 100g for solid foods and 1.5g of fat per 100mL for liquid foods.[8] • This definition of "low fat" excludes virtually all cheese. Milk and milk products <u>including cheese</u> provide valuable nutrients to the diet and are associated with various health benefits[3]	
ISSUE THREE: The proposed wording could be interpreted that milk and milk products should be low fat, but calcium-fortified alternatives do not need to be low fat	• We query the rationale for promotion of low fat dairy but allowing full fat milk alternatives. • If the concern is saturated fat content with regard to cardiovascular health - growing evidence suggests dairy product consumption is associated with a reduction in CVD risk, despite its saturated fat content (see points raised above) • If the concern is total fat content – choosing reduced fat, reduced energy milk <u>and milk alternatives</u> can be helpful for those wishing to reduce their energy intake.	Include reference to low or reduced fat calcium-fortified milk alternatives for clarity
ISSUE FOUR: the use of the term "some" is vague and, by comparison to other food groups such as grains and cereals, suggests milk and milk products should be limited	• Milk and milk products make a significant contribution of a range of essential nutrients to intakes in New Zealand, and are understood to be the richest dietary source of calcium in particular. • It is estimated that approximately 65%[1] of children and 59%[2] of adults in New Zealand do not consume the recommended intake of calcium • As such, Eating Guidelines Statements should accurately reflect the role of milk and milk products in a healthy eating plan and promote appropriate consumption of this food group. • Note the US dietary guidelines which recommend *increasing* the intake of milk and milk products as an example of addressing underconsumption of this food group • We look forward to reviewing suggested serve sizes and frequency of intake for milk and milk products and calcium-fortified alternatives	Remove the word 'some'

See over for references

References

1. Food and Nutrition Guidelines for Healthy Children and Young People: a Background Paper. Ministry of Health. 2012.
2. A Focus on Nutrition: Key findings of the 2008/09 New Zealand Adult Nutrition Survey. University of Otago and Ministry of Health. 2011.
3. Australian Dietary Guidelines. National Health and Medical Research Council. 2013.
4. U.S. Department of Agriculture and U.S. Department of Health and Human Services. Dietary Guidelines for Americans, 2010. 7th Edition, Washington, DC: U.S. Government Printing Office, December 2010.
5. Milk and dairy products in human nutrition. Food and Agriculture Organization of the United Nations (FAO) 2013. pp 121-1.34
6. Dietary Guidelines Advisory Committee. Report of the Dietary Guidelines Advisory Committee on the Dietary Guidelines for Americans, 2010, to the Secretary of Agriculture and the Secretary of Health and Human Services. 2010. US Department of Agriculture, Agricultural Research Service, Washington, DC.
7. Jakobsen MU, Dethlefsen C, Joensen AM, Stegger J, Tjonneland A, Schmidt EB, Overvad K (2010) Intake of carbohydrates compared with intake of saturated fatty acids and risk of myocardial infarction: importance of the glycemic index. *American Journal of Clinical Nutrition*, 91(6):1764-1768.
8. Food Standards Australia New Zealand 2013, Australia New Zealand Food Standards Code - Standard 1.2.7 – Nutrition, Health and Related Claims, accessed 11 April 2014, http://www.comlaw.gov.au/Details/F2013L00054

Sent by: fionag@beeflambnz.co.nz
04/03/2015 09:57 a.m.

To: "elizabeth_aitken@moh.govt.nz" <elizabeth_aitken@moh.govt.nz>,
cc: "Louise_McIntyre@moh.govt.nz" <Louise_McIntyre@moh.govt.nz>,
bcc:

Subject: RE: Eating and Activity Guidelines

Thanks Elizabeth
I will get on to looking at this ASAP

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From: elizabeth_aitken@moh.govt.nz [mailto:elizabeth_aitken@moh.govt.nz]
Sent: Tuesday, 3 March 2015 3:27 p.m.
To: Fiona Greig
Cc: Louise_McIntyre@moh.govt.nz
Subject: Eating and Activity Guidelines

Dear Fiona,
Further to my earlier phone call please accept my apologies for tomorrow's meeting in Auckland. The draft Eating and Activity Guidelines document is attached below which we request you not to circulate more widely. As indicated any questions can be forwarded to Louise and do let us know if it would be helpful for the Ministry to teleconference with you at some stage next week before the feedback is due which could be either with FGC members or separately.
Kind regards
Elizabeth

Re: Review of the Eating and Activity Guidelines document between 4 and 13 March 2015

Please find attached a copy of the Eating and Activity Guidelines Series (EAGs) statements document. The document is written for health practitioners and people who provide health advice on nutrition and physical activity for healthy adults. Specifically the document:

1. brings together the updated eating and physical activity statements for New Zealand adults, outlining each statement and why it is recommended
2. connects the statements to the most robust (systematically reviewed and graded) international evidence currently available
3. provides some information for putting the statements into practice.

Throughout this document, the Ministry has tried to use plain English where possible rather than using technical terminology. Inevitably, some technical terms remain. The statement wording and evidence has been agreed to by a technical advisory group of nutrition and physical activity academics and experts, so feedback is not being sought on these.

Please note, the statements document will be professionally edited and formatted before publication for ease of reading.

What we need from you?

The Ministry is seeking feedback on (but not limited to):

- any essential information you think is missing from a technical or organisational perspective
- any inaccurate deta l
- any other comments, supported by evidence if a change is being proposed.
-

When does the Ministry need your feedback by?

If you would like to provide feedback, please email it to Louise McIntyre (Louise_McIntyre@moh.govt.nz) and me **by 5pm on Friday 13 March 2015**

Elizabeth Aitken
Team Leader & Senior Advisor (Nutrition)
Nutrition and Physical Activity Team
Clinical Leadership, Protection and Regulation Business Unit
Ministry of Health
DDI: 04 816 4335

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mailto:elizabeth_aitken@moh.govt.nz

Sent by: fionag@beeflambnz.co.nz
04/03/2015 03:25 p.m.
To: "Louise_McIntyre@moh.govt.nz" <Louise_McIntyre@moh.govt.nz>,
cc: "elizabeth_aitken@moh.govt.nz" <elizabeth_aitken@moh.govt.nz>, "Maria_Turley@moh.govt.nz" <Maria_Turley@moh.govt.nz>,
bcc:

Subject: RE: Role of Red Meat report feedback

Hi Louise
Thanks for this feedback on our report. I have attached the latest draft which no longer includes processed meat – this information will be used separately for the NZ Meat Processors Association whose membership is comprised of smallgoods/processed meat manufacturers.

See my responses to your queries below -

In addition, when we get a chance to catch up, I would like to talk to you about the upcoming WHO's IARC review on meat and carcinogenity and a campaign we are facilitating in April, called World Iron Awareness Week.

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From: Louise_McIntyre@moh.govt.nz [mailto:Louise_McIntyre@moh.govt.nz]
Sent: Wednesday, 25 February 2015 10:35 a.m.
To: Fiona Greig
Cc: elizabeth_aitken@moh.govt.nz; Maria_Turley@moh.govt.nz
Subject: RE: Information on the new Eating and Activity Guidelines Series and the update of the adult nutrition guidelines.

Hi Fiona

Good to speak with you today and we look forward to meeting up next week. As I mentioned I have a few queries regarding some information in the draft report *The role of Red Meat in a Healthy New Zealand Diet*you sent through a couple of weeks ago.

- p4/5 summary of the report states that the "Current average intakes of red meat in New Zealand are below the amount recommended by the World Cancer Research Fund, of up to 500g cooked red meat per week" - Im not aware that national level weekly red meat intake data exists so I would be interested to know what this statement is based on. Im aware of the Adult Nutrition Data as described in pg 23, and of the extra analysis done by Otago University which provides the average daily intake data as mentioned on pg 31, but not that there is an accurate weekly intake amount.

  We have used population data from the secondary analysis of the adult nutrition survey which shows approximately 50g/day consumed, which we have equated to about 350g/week which does fall under the 500g (71g/day) WCRF recommendation.

		2008/09		1997
Total beef + lamb	41.1 + 9.3 =	**50.4**	45.5 + 11.0 =	**56.5**
Men beef + lamb	53.0 + 10.8 =	**63 8**	60.0 + 14.0 =	**74.0**
Women beef + lamb	30.3 + 7.9 =	**38.2**	31.0 + 8.0 =	**39.0**

- Pg 31, the 50g per ?day intake quoted from Parnell et al 2012, I suspect this is the amount over the total population, whereas the average amount per red meat consumers is around 180g (beef and veal) which is 3 x as much. The 500g recommendation from World Cancer Research Fund (WCRF) is a personal health recommendation so I dont think it works to compare it to the population wide intake. Any rationale for this?

  We can amend this wording to reflect this. Suggest:

  As research in this area continues, it may be prudent to avoid very high intakes, particularly of processed meats, and to limit very-high-temperature cooking methods. The WCRF (2007) have recommended that intakes of cooked red meat can be up to 500g per week ~~this is higher than the current average intake of red meat in New Zealand of around 50g per day (Parnell et al; 2012) equating to 350g/week, so a reduction in average intakes is unnecessary.~~. Twenty-four hour recall data from the New Zealand adult national nutrition shows a daily serving of 179.6g beef and veal, and 137.2g lamb and mutton, which reflects an average portion size. At a population level, New Zealanders are eating an average of 41.1g/day of beef and veal, and 9.3g/day of lamb and mutton (Parnell et al, 2012).

- The WCRF guidance, including the 2007 report and the Continuous Update Project report (2011) on colorectal and rectum cancer both say "Red meat is a convincing cause of colorectal cancer". The level of evidence provided is deemed 'convincing' and the specific personal intake recommendation being to 'limit intake to less than 500gm week' This contrasts with pg 36 of the draft report which says "Some scientific studies have suggested an association between red meat consumption and colorectal cancer. However, associations are weak and overall evidence is mixed".

  Given there is a lot of conflict around the topic of meat and cancer, the RORM report has been written as an evidence-based report navigating through the available evidence the best way possible, recognising the WCRF report, which has been put together by a large group of experts, but has been heavily criticised since it was written by leading researchers including epidemiolgists, and hence also presenting the counter evidence. We are also satisfied with what we have said given Jim Mann reviewed this section and did provide input to what we should and shouldn't include.

- The WCRF report also says "Processed meat is a convincing cause of colorectal cancer", and recommends it is avoided. The draft report doesnt seem to reflect the strength of this information and it would be good to talk to you about that.
  We have since removed the processed meat information out of the RORM as it will report on fresh red meat only. The processed meat information will used independently for issues management. Again, the report will be considering both sides of the processed meat and cancer evidence. Emphasis on low consumption in NZ (g/day at population level).
- I take the point on pg 24 of the draft report which states "the recommended number of servings from the meat and alternative group, along with serving sizes, may need to be reconsidered " Last year the Ministry began a review of its current serving size advice. We can fill you in alittle more on this when we meet next week.

Will be interested to hear more on this.

Cheers

Louise McIntyre
Advisor - Nutrition and Physical ActivityTeam
Public Health Group
Clinical Leadership, Protection and Regulation
Ministry of Health
DDI: 04 816 3382
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From: Fiona Greig <fionag@beeflambnz.co.nz>
To: "Louise_McIntyre@moh.govt.nz" <Louise_McIntyre@moh.govt.nz>,
Date: 10/02/2015 09:48 a.m.
Subject: RE: Information on the new Eating and Activity Guidelines Series and the update of the adult nutrition guidelines.

Hi Louise
My mistake, draft attached

Thanks

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From: Louise_McIntyre@moh.govt.nz [mailto:Louise_McIntyre@moh.govt.nz]
Sent: Tuesday, 10 February 2015 9:46 a.m.
To: Fiona Greig
Subject: RE: Information on the new Eating and Activity Guidelines Series and the update of the adult nutrition guidelines.

Hi Fiona

Thanks for your email. I cant seem to find a copy of the latest repo t attached to the email - only a link to the 2009 report. Have I missed it or you may have just forgotten to attach? -

Cheers

Louise McIntyre
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Public Health Group
Clinical Leadership, Protection and Regulation
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DDI: 04 816 3382
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From: Fiona Greig <fionag@beeflambnz.co.nz>
To: "Louise_McIntyre@moh.govt.nz" <Louise_McIntyre@moh.govt.nz>,
Date: 09/02/2015 04:05 p.m.
Subject: RE: Information on the new Eating and Activity Guidelines Series and the update of the adult nutrition guidelines.

H Louise
Please find attach a draft of our report: Role of Red Meat in a healthy New Zealand Diet as requested.

This will be formatted and distributed in due course, but please use at your discretion in the meantime

Feedback on referencing the Food and Nutrition guidelines is appreciated

Regards

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From: Louise_McIntyre@moh.govt.nz [mailto:Louise_McIntyre@moh.govt.nz]
Sent: Wednesday, 4 February 2015 3:02 p.m.
To: Fiona Greig
Subject: RE: Information on the new Eating and Activity Guidelines Series and the update of the adult nutrition guidelines.

Hi Fiona

Firstly apologies for not replying to this email until now.

I would be interested in reading your report on the role of red meat in a NZ diet when it is available if possible? The plan at this point is to publish our guidelines document - draft title is "Eating and Activity Guideline Statements for New Zealander Adults" around the middle of the year.

Cheers

Louise McIntyre
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From: Fiona Greig <fionag@beeflambnz.co.nz>
To: "Louise_McIntyre@moh.govt.nz" <Louise_McIntyre@moh.govt.nz>,
Date: 22/01/2015 12:08 p.m.
Subject: RE: Information on the new Eating and Activity Guidelines Series and the update of the adult nutrition guidelines.

Hi Louise
Thanks for this information from last year which aided in my updating our international meat nutritionist group last October.

I am close to getting our own Role of Red Meat in a Healthy New Zealand Diet report update from 2009 finalised – this is the 2009 version FYI http://beeflambnz.co.nz/resources/factsheets-theroleofredmeatinahealthynewzealanddiet.pdf which provides the base of the 2015 review with additional (80+) references.

Where we have referenced the adult nutrition guidelines, how far away is MOH from releasing the EAGs so we can have the most up-to-date information and reference available to review please?

I look forward to hearing from you.

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From: Louise_McIntyre@moh.govt.nz [mailto:Louise_McIntyre@moh.govt.nz]
Sent: Friday, 26 September 2014 2:15 p.m.
To: Fiona Greig
Cc: Harriette_Carr@moh.govt.nz
Subject: Information on the new Eating and Activity Guidelines Series and the update of the adult nutrition guidelines.

Hi Fiona

The update of the adult guideline statements is part of the Mnistry's new Eating and Activity Guidelines Series as explained below. This is a brief overview of the EAGS and the update of the guidelines statements for adults.

Background
In early 2015, the Ministry intends to publish a core document for health practitioners on key healthy eating and physical activity messages for adult New Zealanders. This document is the first of the new integrated Eating and Activity Guidelines Series (EAGS) that will gradually replace the existing Food and Nutrition Guidelines series and physical activity guidelines.

The EAGS is being developed following an independent evaluation of the current Food and Nutrition Guidelines in 2011. The evaluation showed the Guidelines are valued by many health practitioners, but some changes to their development process and format would strengthen them and make them accessible to a wider audience. The EAGS will aim to meet the future needs of the health sector and the Ministry by:

- being more accessible to a wider range of health practitioners and others who provide information on nutrition and activity
- having a more comprehensive web presence,
- improving the timeliness for updates to nutrition and physical activity priority issues, and
- by having a more transparent and robust (graded systematic) evidence base underpinning the key guidelines statements.

The EAGS will consist of the core document described above and a range of other supporting documents produced over time. These will include a series of detailed issue-based papers on specific food, nutrition and physical activity topics and updated health education resources for the public.

Process for updating the nutrition and physical activity guidelines for adult New Zealanders includes:

- A technical advisory group (TAG) made up of nutrition and physical activity academic specialists is advising the Ministry on its review of the current healthy eating and physical activity messages for adult NZers, including advising on the most appropriate evidence bases to underpin the statements. The use of international graded evidence to underpin the statements will ensure greater transparency and confidence in the guideline statements.
- Limited stakeholder consultation on the draft updated guidelines involving the health, nutrition and physical activity sectors including Māori, Pacific and Asian health organisations and the Food and Grocery Council.
- Focus group testing of the draft updated guidelines with the public, including representatives from the Māori, Pacific, South Asian and European communities.
- Internal and external (to the Ministry) peer review of the final draft core document.
-

Evidence base
Graded systematic evidence bases that underpin the national nutrition guidelines of a number of developed countries, including Australia, United States of America and the Nordic countries. Evidence developed by international organisations such as the World Health Organization and the World Cancer

Research Fund.

Happy to discuss if useful.

Cheers

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Role of Red Meat_Final_Feb15.doc

The Role of Red Meat in a Healthy New Zealand Diet

Written by Amanda Johnson BSc(hons), MSc, PG Dip Diet

February 2015

Contents

Acknowledgement to the following for reviewing sections of The Role of Red Meat in a Healthy New Zealand Diet report:

Dr Tatjana Buklijas, Liggins Institute Centre for Human Evolution, Adaptation & Disease
Dr Laurence Eyres, FoodInc
Delvina Gorton, New Zealand Heart Foundation
Dr Anne-Louise Heath, University of Otago
Professor Jim Mann CNZM, University of Otago
Associate Professor Winsome Parnell, University of Otago
Professor Elaine Rush MNZM, Auckland University of Technology
Professor Christine Thompson, University of Otago
Dr Pam von Hurst, Massey University

Summary

Historically, red meat has played a central role in the human diet.

Around 4 to 5 million years ago, it is believed the ancestral hominid line emerged from the receding forests to become bipedal open grassland dwellers, evolving to require higher-quality foods based around meat protein and fat.

This was accompanied by subsequent physiological and metabolic adaptations involving the development of a larger brain and a smaller gastro-intestinal tract.

Evidence from fossil stable isotope analysis demonstrates a growing reliance on consumption of meat as humans evolved.

Red meat continues to play an important role in the human diet today; it is an excellent source of protein and, trimmed of visible fat, is low in total fat and saturated fatty acids. It also makes a significant contribution to the monounsaturated and omega 3 fatty acids in our diet.

In addition, meat from ruminant animals, such as beef and lamb, provides conjugated linoleic acid (CLA), which has been found to have cancer preventive and immunomodulatory properties in animal models.

In terms of micronutrients, red meat (particularly beef and lamb) is an excellent source of bioavailable iron and zinc, and also provides selenium, vitamin D, and B vitamins, with red meat being one of our major sources of vitamin B_{12}. Red meat also contains bioactive compounds such as taurine, carnitine, creatine and some endogenous antioxidants.

Lean meat has an important role to play in the diets of all age groups in New Zealand, providing nutrients that enable optimal growth and development in childhood as well as maintenance of health and wellbeing throughout adulthood and well into old age.

For those who exclude meat, careful consideration needs to be given to the nutritional adequacy of the diet as more restrictive diets are associated with a greater risk of deficiencies. In particular, vegans (who exclude all animal products) need to take extra care to ensure their nutritional needs are met.

The low fat and saturated fatty acid content of lean red meat makes it an ideal food to include as part of a heart-healthy, balanced diet.

The protein content may play a useful role in weight management due to its effect on appetite control, satiety and food craving. There is also emerging evidence that as dietary protein intake falls, energy intake increases. Due to the impact protein has on the glyacemic index of a meal, there is an important role in diabetic blood sugar control.

With respect to cancer, epidemiological and mechanistic data on associations between red meat and cancer are weak in magnitude and are inconsistent. Current average

intakes of red meat in New Zealand are below the amount recommended by the World Cancer Research Fund (WCRF, 2007) of up to 500g cooked red meat per week.

Lean red meat had a central role in the diet of early man and continues to do so in modern times (Ruxton et al., 2013). It is consumed by nine out of ten New Zealanders once or more a week. Red meat provides a unique package of nutrients that make an important contribution to optimal health as part of a balanced diet and active lifestyle.

1. Introduction

In New Zealand, 94.5% of the adult population consumes red meat (University of Otago & Ministry of Health, 2011). Among children, 95% consume an omnivorous diet (Parnell et al., 2003).

As the majority of New Zealanders consume meat, consideration of its contribution to nutritional intakes and its role in health and disease is important.

This report provides background information on human evolution and the increasing importance of meat consumption as humans evolved. It reviews current scientific knowledge in terms of the nutritional content of red meat, its contribution to the diets of New Zealanders and its role in health and disease.

Some of the myths and misconceptions about meat are discussed in Appendix 1 and information about current farming practices in New Zealand, sustainability, and risk management at processor level are covered in Appendix 2.

The term 'red meat' in this report refers to beef, veal, lamb and mutton. For the purpose of this report, where meat is mentioned it refers to red meat flesh, which is defined as the skeletal muscle of beef, lamb, veal and mutton which includes any attached fat, connective tissue, rind, nerve, blood and blood vessels (FSANZ, 2012).

2. Human evolution and meat consumption

Meat has played a central role in our diets throughout evolution and there is good evidence that over the last 2 million years the human ancestral line has been consuming increasing quantities of meat (Mann, 2000). The earliest evidence for scavenged meat goes back to the late Australopithecines after early hominins were herbivores. In fact, it has been suggested that even mild and intermittent shortages of meat can have adverse consequences for energy and micronutrient-sensitive tissues such as the brain (Williams and Dunbar, 2013).

Not only have there been changes in cranio-dental features to enhance our ability to bite and tear animal flesh, but comparative gut morphology shows humans are truly omnivorous (Mann, 2007). In addition, fossil isotope ratios indicate consumption of a high-meat diet in early hominids, as early as 1.8 million years ago (Mann, 2000).

More recent human history, from archaeological records of around 40,000 years ago, shows the use of bone and antler tools such as spear tips and harpoons.

There is also evidence to suggest animal traps and bows and arrows were used subsequent to this time (Ulijaszek, 2002). Around 9,000 years ago the settling and growth of populations and the domestication of both plants and animals began (Biegert, 1975).

Primates in general and humans in particular, have larger brain sizes than would be expected for their body size, a phenomenon described as encephalisation. In humans, there has been a dramatic increase in brain size over the last 2-3 million years (Aiello & Wheeler, 1995).

The consumption of meat rich in fats (particularly the unsaturated fats) is one theory to be the factor responsible for the threefold increase in brain size over the last 4.5 million years (Chamberlain, 1996; Mann, 1998). It has been estimated that whenever it was ecologically possible, hunter-gatherers consumed 45-65% of their energy from animal foods, with protein providing 19-35% of energy at the expense of carbohydrates, which provided 22-40% of energy (Cordain et al., 2000). Another theory, the social brain hypothesis suggests brain size in primates is linked to the size of groups of which they live (Dunbar, 2009).

It has been suggested diets high in meat can be associated with high cholesterol levels and elevated risk of heart disease (Snowdon et al., 1984; Huijbregts et al., 1995; Menotti et al., 1999). However, a diet high in animal foods does not necessarily elicit unfavourable blood lipid profiles. An analysis of a type of hunter-gatherer diet by Cordain et al. in 2002 found that although 65% of energy was provided by animal foods, many hunter-gatherer societies are relatively free of the signs and symptoms of cardiovascular disease (CVD). More intense exercise and work patterns are likely to have provided pre-agricultural people with protection against CVD. In addition, qualitative differences in fat intake, including a higher intake of monounsaturates and polyunsaturates and a lower *n*-6:*n*-3 ratio, would have served to inhibit the development of cardiovascular disease among these populations.

Other dietary factors, such as a high intake of antioxidants, fibre, vitamins and phytochemicals and a low intake of salt and sugar, along with low levels of stress and no smoking, would further deter the development of cardiovascular disease.

Reverting to the diet and lifestyle of the hunter-gatherer has been shown to result in health benefits. A study of middle-aged, overweight, diabetic Aborigines in Australia, who reverted to their traditional hunter-gatherer diet for seven weeks, found improvements in all aspects of carbohydrate and lipid metabolism linked with insulin resistance (O'Dea, 1984). Despite the high contribution of animal foods to energy intake in this study (64%), the diet was low in total fat (13%) due to the very low fat content of the wild animals.

While there is no consensus on what the Palaeolithic diet is exactly, it is generally characterised by higher protein, less total fat, more essential fatty acids, lower sodium and higher fibre (Turner & Thompson, 2013).

It has been proposed that the relatively recent deviation from the Palaeolithic diet and lifestyle may be the basis of many, if not all, current diseases of civilisation (Kuipers et al., 2012), yet in some cases positive selection was so strong that in the 10,000

years since the beginning of Neolithic age, populations in Europe and East Africa evolved to use dairy beyond infancy (Tishkoff et al., 2006). A recent randomised controlled trial of one Palaeolithic diet among 70 post-menopausal women (mean age 60 years) found strong effects on fat mass, body weight and abdominal obesity after 6 months, although there were no significant differences at 24 months (Mellberg et al, 2014). In this study, the Palaeolithic diet provided 30% of energy from protein, 40% from fat and 30% from carbohydrate; the diet was based on lean meat, fish, eggs, vegetables, fruits, berries, nuts, avocado, rapeseed oil and olive oil; the diet was high in monounsaturated and polyunsaturated fats.

In summary, meat has been a significant part of our diet for millions of years and still makes an important contribution today. A diet high in lean red meat has been shown to lower plasma cholesterol, contribute significantly to tissue *n*-3 fatty acids, and provides a good source of iron, zinc and vitamin B_{12} (Mann, 2000).

A Palaeolithic dietary pattern may be beneficial in terms of aiding weight loss, but further research is needed into the longer-term effects of this type of diet before firm recommendations can be made (Mellberg et al, 2014).

3. Key nutrients in beef and lamb

The primary components of meat are water, fat and protein. The proportions of these constituents can be highly variable, depending on the species and breed of animal, the age of the animal at slaughter, the season, and the types of grass and feed used. The amount of trimming of fat both before and after purchase, and the cooking method used will also influence the nutritional composition of the meat as eaten (BNF, 1999).

There are a number of valuable vitamins, minerals and trace elements in lean meat. In particular, red meat is an excellent source of iron and zinc, which are present in a highly bioavailable form. Red meat also provides a number of B vitamins, along with vitamin D, and offal is a good source of vitamin A.

A summary of the nutrients in selected cuts of lean beef and lamb can be found in Table 1.

Table 1: Nutritional composition of selected cuts of lean, meat (per 100g)

Nutrient	Beef* (composite cuts)	Lamb* (composite cuts)	Adult NZ RDI**
Energy (kJ)	841	792	5,600-18,600
Protein (g)	30.7	27.4	46-81
Fat (g)	8.6	8.9g	-
Thiamine (mg)	trace	trace	1.1-1.4
Riboflavin (mg)	trace	trace	1.1-1.6
Niacin (mg)	9.0	11.0	14-18
Vitamin B_6 (mg)	0.22	0.18	1.3-2.0
Vitamin B_{12} (µg)	1.6	1.8	2.4-2.8
Total folate (µg)	6.8	0	400-600
Sodium (mg)	40	68	460-920^{+}

Potassium (mg)	280	320	2800-3800[+]
Calcium (mg)	6.1	15	1000-1300
Iron (mg)	2.6	1.8	8-27
Zinc (mg)	5.1	4.0	8-14
Selenium (µg)	3.3	6.3	60-75

Sources: *Sivakumaran et al., 2014; **NHMRC, 2006

RDI is the Recommended Dietary Intake (the average daily dietary intake level sufficient to meet the nutrient requirements of nearly all (97-98%) healthy individuals in a particular life stage and gender group).

[+] Adequate Intake (AI), used when an RDI cannot be determined.

Detailed background information on different nutrients and their role in the prevention of deficiency can be found in the National Health and Medical Research Council report *Nutrient Reference Values for Australia and New Zealand including Recommended Dietary Intakes* (NHMRC, 2006). This report also provides information on optimising diets to reduce chronic disease risk.

3.1. Fat

A small amount of fat can contribute to the palatability and flavour of meat. However, it is advisable to remove the visible fat from meat before eating to reduce overall fat content. Red meat cuts as sold have undoubtedly become leaner in recent years (Laugesen, 2005).

Since 1997, the red meat industry's Quality Mark has required the trimming of beef and lamb cuts to no more than 5mm external fat (see Appendix 2 for further information). This has ensured leaner cuts have become the norm for those buying meat as steaks or chops.

A study into the impact of this initiative found the trimming of fat from red meat before sale (supported by virtually all butchers) resulted in 30% less fat and 65% less saturated fat than 20 years ago (Laugesen, 2005).

Data from the 2008/09 New Zealand Adult Nutrition Survey revealed that intake of total fat was 33.7% and 33.8% for men and women respectively (University of Otago & Ministry of Health, 2011), down from 40% in 1977 and 35% in the 1997 survey (Russell et al., 1999).

The same survey showed beef and veal contributed 4.8% to total fat intake and lamb and mutton contributed 2%, down from the previous survey of 8% of total fat intake from beef and lamb. Sausages and processed meats contributed 4%,and pies and pasties contributed 3.5% to fat intake.

The total fat and fatty acid content of selected meats is shown in Table 2, where it is to be noted the saturated fat content is about 33% of total fat of lean cuts compared to over 50% in the fat trimmed from the meat.

3.1.1. Saturated fatty acids (SFA)

Saturated fatty acids (SFA) are fully saturated with hydrogen and contain no double bond. They are the main types of fatty acids found in foods such as milk, cream, cheese, meat from most land animals, palm oil and coconut oil as well as in pies, biscuits, cakes and pastries (NHMRC, 2006).

Only around half the fat in meat is saturated (see Table 2). The rest is mainly monounsaturated fats, with small amounts of polyunsaturated fats, including some *n*-3 fatty acids. The main saturated fatty acids in meat are palmitic and stearic acid (Higgs, 1999); and stearic acid has almost no effect on blood cholesterol (FAO, 2010).

The density of saturated fatty acids in a 100g portion of lean meat is quite low (see Table 2). For example, one tablespoon of olive oil contains more saturated fat (2.3g) than two slices of lean roast silverside of beef (1.7g) (Sivakumaran et al., 2014). In the 2008/09 New Zealand Adult Nutrition Survey, the contribution to intake of saturated fat from beef and veal was 5% and 2.3% from lamb and mutton. (University of Otago & Ministry of Health, 2011). For more information on saturated fats and coronary heart disease, see section 8.1.1.

3.1.2. Monounsaturated fatty acids (MUFA)

Monounsaturated fatty acids (MUFA) have one double bond; the main MUFA is oleic acid (NHMRC, 2006).

Monounsaturates have been found to help lower the amount of LDL cholesterol in the blood, while maintaining HDL blood cholesterol levels. This is likely to be a factor in the ability of Mediterranean diets, which are rich in monounsaturates, to protect against cardiovascular disease (BNF, 2005).

A significant proportion of the fatty acids in meat are monounsaturates (see Table 2), principally oleic acid (Higgs, 1999). In New Zealand, the contribution to intake of monounsaturated fat from beef and veal is 5.8% and 2.1% from lamb and mutton (University of Otago & Ministry of Health, 2011).

3.1.3. Polyunsaturated fatty acids (PUFA)

Polyunsaturated fatty acids (PUFA) contain two or more double bonds. There are two main types of PUFA: omega-3 and omega-6 (abbreviated as *n*-3 and *n*-6). The balance of *n*-3 and *n*-6 in the diet is thought to be important for health. High intakes of *n*-6 PUFA have been linked with a lower risk of coronary heart disease (CHD) and lower LDL-cholesterol levels (NHMRC, 2006; BNF, 2005). The *n*-3 PUFA have little effect on blood cholesterol, but reduce triglyceride levels and have a beneficial effect on blood clotting. In addition, experimental studies have shown *n*-3 PUFA modify inflammatory and immune reactions (Simopoulus, 2002).

Fish and seafood are the richest dietary sources of *n*-3 PUFA, with concentrations 5-15 times higher than meat (Howe et al., 2006); however meat is also likely to make a significant contribution to intakes of *n*-3 PUFA when the relative amounts eaten are considered. Australian data show meat, poultry and game contribute 43% to overall intakes of *n*-3 PUFA, with beef contributing 22.3% and lamb contributing 5.9%

(Howe et al., 2006). Whilst this is high in percentage terms, it is still relatively low in absolute long chain *n*-3 amounts.
Meat from animals raised on grass, as in New Zealand, contains higher levels of *n*-3 PUFA than meat from animals raised on grain. One study found, for example, there was 2-4 times the amount of *n*-3 PUFA in beef from grass-fed animals (including 18:3) than in meat from concentrate-fed animals, except for 20:4 *n*-3 where there was 10 times the amount in the grass group (Enser et al., 1998). The same study found similar results for lamb from animals grazed on grass. Enser (1995) also showed lean grass-fed beef has a much higher amount of phospholipids, which are rich in *n*-3, particularly docosapentaenoic acid (22:5) and a source of choline.

A more recent study compared the effects of consuming red meat from either grass-fed or concentrate-fed animals, and found that dietary intakes, as well as plasma and platelet concentrations of long chain *n-3* PUFA were significantly higher in those subjects who consumed the grass-fed animals (McAfee et al., 2011). The difference in intake of long chain *n-3* PUFA between the groups that was attributable to the red meat consumed was estimated at 18mg/day.

A significant amount of the *n*-3 PUFA in meat are from docosapentaenoic acid (DPA), which is an intermediate in the production of docosahexaenoic acid (DHA) from eicosapentaenoic acid (EPA). DPA has been shown to be a more potent inhibitor of platelet aggregation than EPA or DHA (Akiba et al., 2000), and in the Kuopio Ischaemic Heart Disease Risk Factor Study, reduction in risk of acute coronary events correlated significantly with serum concentrations of DPA and DHA in individuals whose mercury status was low (Rissanen et al., 2000). Epidemiological data on DPA are, however, limited and more information is needed on the nutritional and health benefits of consumption of DPA (Howe et al , 2006).

Given the evidence linking EPA, DHA and DPA to health, it would seem prudent to encourage increased consumption of these fatty acids in the diet. An intake in the region of 0.4g/day for women and 0.6g/day for men is recommended (NHMRC, 2006). Overall, red meat in New Zealand could make an important contribution to intakes of *n*-3 PUFA, particularly in those who don't eat much fish (Knowles et al., 2004).

Table 2: Fat and fatty acid content of lean cooked meat (per average 100g serving)

Meat Cut	Total fat (g)	SFA (g)	MUFA (g)	PUFA (g)
Lean beef, cooked, composite cuts	8.6	2.9	3.1	0.4
Beef mince, premium, simmered	3.3	1.2	0.9	0.2
Beef silverside, lean, roasted	5.0	1.7	1.8	0.3
Beef topside, lean, braised	7.3	2.5	2.6	0.4
Lean lamb, cooked, composite cuts	8.9	3.2	2.3	0.5
Lamb leg, lean, roasted,	6.4	2.1	1.8	0.4
Lamb, rump, lean, roasted	5.3	1.8	1.3	0.3

Source: Sivakumaran et al., 2014

3.1.4. *Trans* fatty acids

Trans fatty acids (TFAs) are unsaturated fatty acids that have at least one double bond in the *trans* configuration. There is good evidence TFAs have a more adverse effect on cardiovascular disease risk than saturated fatty acids (FAO, 2010), although, quantitatively TFAs constitute a much smaller proportion of the diet than saturated fatty acids (NHMRC, 2006). Most of the *trans* fat in the diet is found in margarines and products such as cakes, biscuits and pastrie. Some *trans* fats can also occur naturally at low levels in ruminant animal foods, formed as a result of biohydrogenation by rumen bacteria. However, the predominant ruminant TFA is vaccenic acid (Turpeinen et al., 2002), which has not been associated with coronary heart disease (Willett et al., 1993). There is an average of 0.2g *trans* fat in lean cuts of beef and lamb (New Zealand FOODfiles, 2013).

The World Health Organisation has recommended TFAs contribute no more than 1% of total dietary energy (WHO, 2003). In New Zealand, current intakes are around 0.6% of total dietary energy (FSANZ, 2014) which is due to the removal of partially hydrogenated fat from margarines by manufacturers a decade ago. It is recommended saturated fatty acids and TFAs together contribute no more than 8-10% of total energy (NHMRC, 2006).

3.1.5. Conjugated linoleic acid

The term conjugated linoleic acid (CLA) generally refers to mixtures of positional and geometric conjugated isomers of linoleic acid. The principle dietary form of CLA is the cis-9, *trans*-11 isomer (Pariza et al., 2000), which provides over 90% of our intake (Nakamura et al., 2008). CLA has been shown in animal studies to inhibit carcinogenesis and atherosclerosis, enhance immunologic function, affect body composition change (reducing fat gain and enhancing lean body mass gain), and stimulate growth (Pariza et al., 2000). More recently, CLA has been found to modulate immune function in humans (O'Shea et al., 2004). However, studies in humans into the effects of CLA are generally less conclusive than animal studies, with conflicting and inconsistent findings (Plourde et al., 2008; Nakamura et al., 2008) as dosages of around 3g/day were required.

The highest levels of dietary CLA are found in the meat and milk from ruminant animals (Nakamura et al., 2008). The method of feeding may affect the levels of CLA present in the meat. For example, beef from pasture-fed cattle may have a higher CLA content than beef from silage- or grain-fed cattle (Mir et al., 2004). CLA in meat is located in the interstitial, non-visible fat, evenly distributed along the muscle fibres, as well as in the subcutaneous deposits (Eynard & Lopez, 2003), whereas visible fats are often, and easily discarded, interstitial fats will be eaten. Thus, lean meat could potentially make an important contribution to the human intake of CLA.

The current dietary intake of CLA in Western populations is too low to provide the beneficial effects seen in animal studies (Turpeinen et al., 2002) and further research is needed into the potential benefits of dietary sources of CLA. In particular,

investigations are needed to develop an understanding of the molecular action of CLA isomers and their potential use in chronic disease therapy (Nakamura et al., 2008).

In addition, a significant portion of the lipids in lean meat are in the form of phosopholipids which are a source of choline, which contributes to normal homocysteine and fat metabolism.

3.2 Protein

Red meat is an excellent source of high biological value protein; the protein is highly digestible and provides all essential amino acids (lysine, threonine, methionine, phenylalanine, tryptophan, leucine, isoleucine and valine) with no limiting amino acids (Williams, 2007). A 100g portion of cooked lean beef or lamb provides around 25-30g of protein (see Table 1).

On average, beef and veal contribute 7.8% to protein intakes in New Zealand, lamb and mutton contribute 2% (University of Otago & Ministry of Health, 2011).

Diets with as little as 10% energy from protein are adequate to meet basic protein requirements, but intakes above 15% energy from protein appear to be required for ensuring adequate intakes of micronutrients.

Evidence is accumulating that increasing intake of high quality protein to a level above the recommended intake may be beneficial during weight loss (see section 8.3). However, an upper limit of 25% energy from protein has been suggested until more is known about the long-term effects of a high-protein diet (NHMRC, 2006).

3.3. Micronutrients

3.3.1. Iron

Iron is needed for the production of a number of proteins in the body, including haemoglobin, myoglobin, cytochromes and enzymes involved in redox reactions. Iron is also important for early brain development and for supporting a healthy immune system.

Iron is present in food in two forms – haem and non-haem. Haem iron (found in meat and fish) is more bioavailable than non-haem iron, with conservative estimates that 25% is absorbed (Hallberg & Rossander-Hulthen, 1991). Non-haem iron (found in meat, legumes, nuts, cereals, some fruits and dark green vegetables such as spinach) is less bioavailable and absorption is influenced by other dietary components. For example, foods containing vitamin C can increase absorption of non-haem iron. In contrast, foods containing phytates (found in legumes and wholegrain cereals) can inhibit non-haem iron absorption. Absorption of iron from vegetarian diets has been estimated to be around 10% (Institute of Medicine Panel on Micronutrients, 2001) and it has been suggested there can be a 10-fold difference in the absorption of iron from different meals with a similar iron content (Hallberg & Hulthen, 2000). Absorption of iron is about 18% from a mixed diet, so iron requirements for vegetarians, who rely on non-haem sources, will be about 80% higher than for those who eat meat (NHMRC, 2006).

Beef and lamb are among the richest sources of bioavailable iron in the diet and, in addition, meat enhances the absorption of non-haem iron from foods eaten at the same time. The nature of the enhancing effect is thought to be related primarily to the muscle proteins (Hurrell et al., 2006). In New Zealand, beef and veal have been found to contribute 7% to our total iron intake and lamb and mutton provide a further 1.5% (University of Otago & Ministry of Health, 2011). The actual contribution of meat to iron intake is much greater, however, owing to the higher proportion of iron absorbed.

Inadequate intakes of iron can lead to varying degrees of deficiency; from low iron stores (indicated by low serum ferritin and reduced iron-binding capacity) to iron-deficiency anaemia (low haemoglobin and haematocrit as well as reduced mean corpuscular haemoglobin and volume) (NHMRC, 2006). The recommended intake of iron in different population groups is shown in Table 3.

Iron deficiency is the most common and widespread nutritional disorder in the world. As well as affecting a large number of children and women in developing countries, it is one of only a few nutrient deficiencies which are also significantly prevalent in developed countries such as New Zealand. The numbers are staggering: 2 billion people – over 30% of the world's population – are anaemic (WHO, 2012), with a substantial proportion of these anaemias resulting from iron deficiency.

The adverse effects of iron deficiency anaemia include poor cognitive development, fatigue, reduced tolerance to work, and decreased aerobic capacity. Iron deficiency anaemia can also have an impact on behaviour. In infants, iron deficiency anaemia has been associated with maintaining closer contact with caregivers, showing less pleasure and delight, being more wary, hesitant and easily tired, being less attentive to instructions and being less playful (Lozoff et al. 1998). Severe, chronic iron deficiency anaemia in infancy has also been associated with reduced mental and motor functioning, and continued developmental and behavioural risk more than 10 years after iron treatment (Lozoff et al., 2000).

Approximately 4% (Soh et al., 2004) to 6% (Grant et al., 2007b) of infants and toddlers in New Zealand have iron deficiency anaemia. However, non-anaemic iron deficiency is considerably more common than iron deficiency anaemia in New Zealand infants and young children (Soh et al., 2004), and may be associated with subtle negative effects on cognitive function and fatigue, as well as an increased risk of developing iron deficiency anaemia if the infant is exposed to a physiological challenge such as rapid growth, infection, or injury.

A study in Auckland children aged 6-24 months found 14% were iron deficient, with the occurrence among Māori and Pacific Island children even higher at 20% and 17% respectively (Grant et al., 2007b). Iron intake was less than the estimated average requirement (EAR) for 25% of the infants. Not meeting the EAR increased the risk of iron deficiency for children aged 6-11 months (relative risk (RR) = 18.45, 95% confidence interval [CI]: 3.24-100.00) and 12-23 months (RR = 4.95, 95% CI: 1.59-15.41). In comparison with New Zealand Europeans, Pacific children had a greater daily iron intake ($p = 0.04$) and obtained a larger proportion of iron from meat and meat dishes ($p = 0.02$) (Wall et al., 2008).

Iron requirements in the first year of life are greater than at any other time due to rapid growth and blood volume expansion (Grant et al., 2007a). The depletion of iron stores accrued in utero, and increased demands for growth, mean that after six months of age infants depend on complementary foods to provide iron (Ministry of Health, 2008a). Meat has been found to play an important role as a complementary food. For example, the addition of powdered red meat to a weaning gruel has been shown to markedly increase total iron absorption (Hallberg et al., 2003). Puréed meat can be introduced once an infant is 6 months of age. Given the risk of iron deficiency in infants and young children, it has been suggested public health campaigns should encourage adequate meat intake to help reduce the problem (Mira et al., 1996).

The importance of both meat and fortified milk for providing iron in the diets of toddlers was demonstrated in a New Zealand trial. The trial assessed the effect of increased red meat consumption, or the use of iron-fortified milk, for improving iron status in healthy non-anaemic toddlers aged 12-20 months (Szymlek-Gay et al., 2009). In this 20 week randomised placebo-controlled trial, toddlers were assigned to either a red meat group (encouraged to consume approximately 2.6mg iron from red meat dishes daily), a fortified milk group (toddlers' regular milk was replaced with iron-fortified milk containing 1.5mg iron per 100mls) or a control group (toddlers' regular milk was replaced with a non-iron-fortified milk containing 0.01mg iron per 100mls). Whereas serum ferritin tended to decrease in the control group, it increased by 44% in the fortified milk group, and did not change in the red meat group. The authors concluded that iron-fortified milk can increase iron stores in healthy non-anaemic toddlers and red meat can prevent their decline (Szymlek-Gay, 2009).

Iron deficiency is also prevalent in Auckland high school students (Schaaf et al., 2000), particularly in girls, where iron deficiency and anaemia were each ten times more common (9.6% and 8.7% respectively) than in boys (0.8% and 0.7%). In females, iron deficiency was two to three times more common and anaemia was three to four times more common in Māori, Pacific Island and Asian adolescents compared with Europeans. Iron deficiency in this study was defined as any two or more of the following: serum ferritin less than 12 µg/L, iron saturation less than 14%, or red cell distribution width greater than 14.5%. Anaemia was defined as haemoglobin less than 120g/L for females and less than 130g/L for males. The level of iron deficiency and anaemia in this study was higher than that reported in an earlier Dunedin longitudinal survey (Fawcett et al., 1998), which found the prevalence of iron deficiency (ferritin less than 12 µg/L) at age 21 was 0.24% in men and 6.7% in women. The higher prevalence in the Auckland study is likely to be due to the different age group studied; adolescent girls have higher requirements for iron due to growth super-imposed on menstrual losses.

Concern has also been expressed in relation to the sub-optimal iron status of women of childbearing age in New Zealand. One study (Ferguson et al., 2001) estimated that the prevalence of sub-optimal iron status among 15-49 year old women was between 7% (serum ferritin less than 12 µg/L) and 13% (serum ferritin less than 16 µg/L). The authors stated that this situation is unacceptable given the negative consequences of even mild iron deficiency. The latest New Zealand Adult Nutrition Survey found that from 1997 to 2008/09 the prevalence of iron deficiency in females had increased from 2.9% to 7.2%. After adjusting for age and ethnicity, there was also an increase in the prevalence of low iron stores in females (University of Otago & Ministry of Health,

2011). For certain high-risk sub-groups (for example vegetarians, athletes, pregnant women, Pacific people and Māori), the prevalence of iron deficiency and iron deficiency anaemia is often much higher (Gibson et al., 2002).

A further study in premenopausal women in Auckland showed that, for women who had children, following a dietary pattern that was higher in meat and vegetables was associated with a 25% lower risk of sub-optimal iron status (Beck et al., 2014).

Pregnant women in particular are vulnerable to iron deficiency, as requirements are significantly increased to meet the needs of the growing foetus as well as increased maternal blood volume. An iron-rich diet, which includes the regular consumption of red meat, chicken and fish, has been recommended (Grant et al., 2007a). Non-haem sources of iron such as grains, cereals, legumes and eggs should also be encouraged along with foods containing vitamin C to enhance absorption.

Table 3: Recommended daily intakes for iron in New Zealand

Population Group	RDI* (mg/day)
Infants (0-6 months)[+]	0.2 (AI**)
Infants (7-12 months)	11
Children (1-3 years)	9
Children (4-8)	10
Children (9-13)	8
Boys (14-18 years)	11
Girls (14-18 years)	15
Women (19-50 years)	18
Pregnant women	27
Breastfeeding women[++]	9-10
Women over 50 years	8
Men over 19 years	8

*RDI is the Recommended Dietary Intake (the average daily dietary intake level sufficient to meet the nutrient requirements of nearly all (97-98%) healthy individuals in a particular life stage and gender group).
**AI is the Adequate Intake, used when an RDI cannot be determined.
[+] Amount normally received from breast milk.
[++] Assumes menstruation does not resume until after 6 months of breastfeeding

Source: NHMRC, 2006

In cases of iron deficiency anaemia, iron supplementation is accepted as the most appropriate method of treatment. However, a New Zealand study investigated whether dietary treatment of non-anaemic iron deficiency could improve iron status in pre menopausal Dunedin women. The study found that dietary intervention involving increased intakes of both haem iron (from flesh food) and enhancers of iron absorption (such as vitamin C), along with a decrease in intake of inhibitors of iron absorption (such as phytic acid), may improve the iron status of pre-menopausal women with low iron stores (Heath et al., 2001). Although the changes in iron status were less with dietary intervention than with supplements, in motivated women with low iron stores, dietary intervention may be an appropriate first-line treatment as long as they are monitored to ensure the treatment has been effective.

Prevention and treatment of iron deficiency among vulnerable groups within New Zealand is an important public health issue. In particular, we need to ensure optimal

intakes of iron among groups such as infants, children, adolescents and pregnant women.

3.3.2. Zinc

Zinc is a component of various enzymes that help maintain the structural integrity of proteins and regulate gene expression (NHMRC, 2006). It is also known to play a central role in the immune system, with zinc deprivation leading to an increased susceptibility to pathogens because of impaired immune response (Shankar & Prasad, 1998). Zinc deficiency can also lead to impaired growth and adverse pregnancy outcomes (NHMRC, 2006).

Those at increased risk of zinc deficiency include older people, vegetarians and people with renal insufficiency (Ibs & Rink, 2003). Zinc deficiency has also been found among New Zealand school children; the 2002 Children's Nutrition Survey (Parnell et al, 2003) found 16% of children had low serum zinc concentrations (21% of males and 10% of females).

Further analysis of data from the 2002 Children's Nutrition Survey by Gibson et al. (2011) found that among Pacific children aged 5-15 years, the prevalence of low serum zinc concentrations was 21%, compared with 16% of Māori children and 15% of European children. In this study, children derived 23% of their zinc intakes from meat, poultry and fish.

It has been suggested that pre-school children may be at even higher risk of zinc deficiency. A recent intervention study found that at baseline 38% of toddlers (12-20 months of age) had low serum zinc concentrations despite seemingly adequate zinc intakes (Morgan et al., 2010). However, researchers in this study found that providing either red meat or fortified milk did not improve zinc status despite increasing zinc intakes.

New Zealand women have also been found to be at risk of zinc deficiency. A study into the zinc status of pre-menopausal Dunedin women found zinc status was lower than had been found in earlier studies (Gibson et al., 2001). It is suggested that changes over time in food selection patterns may account for this change. An example would be the decline in consumption of flesh foods – specifically beef and lamb, which are rich sources of bioavailable zinc. Certainly, in this study, the women who included red meat in their diet had a superior biochemical zinc status to that of those who avoided eating red meat.

The recent New Zealand Adult Nutrition Survey 2008/09 estimated that the prevalence of inadequate zinc intake was 24.7% (males 39.1%, females 11.2%). The highest prevalence was among older males aged 71+ years (89.7%) although these data should be interpreted with caution as the EAR may be set too high, and biochemical zinc status was not determined (University of Otago & Ministry of Health, 2011). An earlier study into the zinc status of Dunedin women aged 70 to 80 years old found 12% had low serum zinc levels (de Jong et al., 2001) and the authors concluded that promotion of nutrient-dense foods or trace element supplements for New Zealand seniors should be considered.

Although zinc is widely distributed in foods, meat, fish and poultry are major contributors, with cereals and dairy foods also providing substantial amounts (NHMRC, 2006). Beef and lamb in particular are among the richest dietary sources of zinc, with a 100g portion providing at least a quarter of adult requirements (see Table 1).

3.3.3. Selenium

Selenium is an integral part of glutathione peroxidase, an enzyme that protects against oxidative damage (NHMRC, 2006). Selenium is also important for the production of other key selenoproteins such as iodothyronine deiodinase (Arthur et al., 1999). Dietary selenium is essential for the efficient operation of many aspects of the immune system (Arthur et al., 2003; Broome et al., 2004) and for optimal thyroid hormone metabolism (Arthur et al., 1999). It may also be anti-carcinogenic (Combs, 2005). Intakes of selenium higher than the recommended intake may be required for protection against cancer and may have other health benefits. However, there is an urgent need for more large scale trials to assess any such beneficial effects and to estimate the level of selenium intake that is protective (Thomson, 2004a).

Overt deficiency of selenium in humans is rare but is seen as Keshan disease, an endemic cardiomyopathy in adolescent or pre-adolescent years in low selenium areas of China (Yang et al., 1988). More marginal deficiency may contribute to reduced immune function, some cancers and viral diseases (Broome et al., 2004).

The New Zealand Adult Nutrition Survey 2008/09 (University of Otago & Ministry of Health, 2011) found that among adults, selenium is provided by fish and seafood (11.6%), bread (15.1%), poultry (9.6%) and meat and meat products (15.1%). Analysis of data from the 2002 Children's Nutrition Survey showed that among children aged 5-14 years, selenium was provided by bread and grains (33%), meat (14.8%), poultry (11.2%), and fish and seafood (8.6%) (Thomson et al., 2007).

Regional differences in selenium intake in New Zealand were observed in the Children's National Nutrition Survey (Thomson et al., 2007). Analysis of the selenium status of children aged 5-14 years showed children in the upper North Island had mean serum selenium concentrations higher than those in the lower North Island and South Island. Younger children had lower selenium intakes than older children (Thomson et al., 2007). These differences have been partly attributed to the different levels of selenium found in bread, since the selenium content of bread is lower in the South Island than the North Island where higher selenium wheat from Australia is used. Another reason for the differences is the high fish and poultry intakes of Pacific children, of whom there was a higher proportion in the north of the North Island (60%) compared with the lower North Island (18%) and the South Island (11%). As a whole, our children fall in the middle of the range of international serum selenium concentrations. However, the selenium status of South Island children is among the lowest values reported internationally (Thomson et al., 2007).

Pregnant and breastfeeding women may be at risk of low selenium concentrations due to the increased selenium demands of the growing foetus and the increased demands of lactation. In addition, infancy is a vulnerable time, with rapid growth and development also leading to increased selenium requirements. A study of South Island

children, aged 6-24 months, and their mothers, found dietary selenium intakes were below recommended levels (McLachlan et al., 2004) with intakes of 7.9 ± 6.2 µg/d in infants; 13.7 ± 8.4 µg/d in toddlers and 38 ± 25 µg/d in mothers. The low intakes were reflected in blood selenium concentrations, which were at the lower end of international levels. The authors recommend dietary strategies to improve selenium intakes are implemented, for example, the inclusion of selenium-rich foods such as fish, meat and unrefined cereals.

The last New Zealand Adult Nutrition Survey 2008/09 estimated that the prevalence of inadequate selenium intake was 45% (males 32%, females 58%). Females aged 15-18 years had a consistently high prevalence of inadequate intakes (over 70%) across all ethnic groups (University of Otago & Ministry of Health, 2011).

Overall the selenium status of the New Zealand population has been increasing and continues to do so. This is due in part to some extent to the increase in selenium concentration of meat, but also to the increase in contribution of imported foods to our diet (Thomson, 2004).

3.3.4. Vitamin A

Vitamin A is a fat-soluble vitamin, which helps maintain normal reproduction, vision and immune function (NHMRC, 2006). The term vitamin A includes retinol from animal sources, and pro-vitamin A carotenoids, such as beta-carotene, which are precursors of vitamin A.

In New Zealand, 17.2% of the population have been found to have inadequate intakes of Vitamin A, with a higher prevalence among younger people aged 15-18 years; 37.5% of males and 27.4% of females (University of Otago & Ministry of Health, 2011). Lower intakes of beta-carotene among younger people contributed to their inadequate intakes.

Carcass meat contains little vitamin A, but liver is a particularly good source of this vitamin in the form of retinol. Chronic intake of large amounts of retinol over time can be toxic and pregnant women should limit their intake of liver as vitamin A can be teratogenic (ie can cause defects in the growing foetus). In some countries, pregnant women are advised to avoid liver altogether; however, in New Zealand, animal feeding practices are different and levels of vitamin A in liver are likely to be lower. The Ministry of Health in New Zealand advises up to 100g of liver may be consumed per week during pregnancy, although liver pâté is not recommended as there is a risk of food-borne illness such as listeriosis (Ministry of Health, 2006a). No more than 10g of liver or pâté per week should be offered to infants and toddlers (Ministry of Health, 2008).

3.3.5. B Vitamins

Red meat is an excellent source of vitamin B_{12}, which is only found naturally in foods of animal and microbial origin. Throughout life, the dietary supply of vitamin B_{12} and other methyl donors are essential for normal growth, development and function and is an essential nutrient for one carbon metabolic pathways. It is key for protein, fat and carbohydrate metabolism including the synthesis of fatty acids in myelin in the

nervous system, and the synthesis and stability of deoxyribose nucleic acid conjunction with folate, for DNA synthesis (Stabler et al., 2013; Rush et al., 2014). Ensuring an adequate intake of vitamin B_{12} particularly in pregnancy and lactation is essential for optimising health of the offspring. A 100g portion of cooked beef or lamb provides almost the entire daily requirement for vitamin B_{12} (see Table 1). For vegans, who avoid all animal products, fortified foods or supplements will be necessary to provide adequate B_{12} (see section 4).

A 100g serving of beef or lamb also provides around half the daily requirement for niacin, along with some thiamine, riboflavin and vitamin B_6, as shown in Table 1. These B vitamins are important for numerous metabolic functions in the body, particularly, as their respective co-enzyme forms, in energy metabolism.

3.3.6. Vitamin D

The main function of vitamin D is to help maintain plasma calcium concentrations by enhancing the absorption of calcium in the small intestine and controlling urinary losses. Over the past decade, deficiency of this vitamin has been associated with higher risk of multiple sclerosis and poorer immune function (Harandi et al, 2014) and prevention of diabetes (Harinarayan, 2014) and some cancers (Ananthakrishnan et al, 2014).

Vitamin D status is generally maintained by the exposure of skin to sunlight. Where exposure to sunlight is inadequate, dietary sources of vitamin D become important. Sub-optimal vitamin D status is associated with low bone mineral density and the risk of osteoporosis later in life (Holick & Chen, 2008).

A high prevalence of vitamin D insufficiency was found in an analysis of the 2002 National Children's Nutrition Survey; with 4% of New Zealand children aged 5-14 years vitamin D deficient (<17.5nmol/L) and 31% vitamin D insufficient (<37.5nmol/L) (Rockell et al., 2005). The children studied had a mean serum 25-hydroxyvitamin D concentration of 50nmol/L, with mean concentrations in sub-groups ranging from 32nmol/L in Pacific girls aged 11-14 years, to 62nmol/L in New Zealand European and other boys aged 5-6 years. Children of Māori and Pacific ethnicity may be at particular risk of low vitamin D status because of low vitamin D intakes, New Zealand's high latitude (35-47^{o}S) and skin colour (Rockell et al., 2005).

New Zealand adolescents and adults have also been found to be at risk of vitamin D insufficiency (Rockell et al., 2006). Analysis of serum 25-hydroxyvitamin D levels using data from the 2008/09 New Zealand Adult Nutrition Survey found 4.9% were vitamin D deficient (<25nmol/L) including 0.2% with severe deficiency (<12.5nmol/L), and one in four adults (27.1%) were below the recommended level of vitamin D, but did not have vitamin D deficiency. The prevalence of vitamin D insufficiency was higher among Pacific adults who were 2.3 times as likely to have vitamin D deficiency as non-Pacific adults.

A study of elderly Dunedin women also found vitamin D deficiency was common, particularly in women over 70 years of age, who had a high bone fracture risk (McAuley et al., 1997). Deficiency was most marked in winter months.

Red meat provides vitamin D. A study into the vitamin D content of beef and lamb found them to be a source of both vitamin D_3 and its active metabolite 25-hydroxyvitamin D_3 (Purchas et al., 2007). 25-hydroxyvitamin D_3 is suggested to have 1.5 to 5 times the activity of vitamin D_3, and the authors of this study estimate (assuming 1µg of 25-hydroxyvitamin D_3 is equivalent to 3µg of vitamin D_3) that, on average, 100g of beef strip loin would contain 1.2µg of total vitamin D_3 and 100g of cooked lamb leg steak would contain 2.6µg.

Current FoodFILES data shows an average of 0.14ug/100g vitamin D3 for lean, raw beef cuts and 0.04ug/100g vitamin D3 for lean, raw lamb cuts (Sivakumaran et al; 2014).

Although this is a small amount compared to the amount needed (adequate intake of 5-10ug/day in adults under 70 years) to improve the vitamin D status of New Zealanders to optimal levels, there is some interest in determining whether meat has a role to play in providing vitamin D. A study by Crowe et al (2011) for example, found that plasma 25 (OH) D_3 concentrations were lower in vegetarians and vegans than in meat and fish eaters. These results do need confirmation through further research (Glossmann, 2011), particularly as the vitamin D content of meat may be variable and dependant on the animal feed and exposure of the animal to sunlight.

3.3 Bioactive substances

In addition to the essential nutrients, meat also provides a number of bioactive substances (Williams, 2007). Meat is a rich source of taurine, an amino acid that may be important during lactation and times of immune challenge, and may offer protection against oxidative stress. Meat also provides carnitine, which transports long chain fatty acids across the inner mitochondrial membranes to produce energy during exercise; requirements for carnitine may be increased in pregnancy and after strenuous exercise. Red meat is the principle human dietary source of creatine, which plays a role in energy metabolism. Meat is also a source of a number of endogenous antioxidants, for example ubiquinone, glutathione, lipoic acid, spermine, carnosine and anserine.

4. Nutritional implications of a meatless diet

A diet that excludes meat can be nutritionally adequate, but if an increasing number of foods are excluded it becomes important to plan the diet carefully to ensure nutrient needs are met. Intakes of iron, zinc and vitamin B_{12} need careful consideration – especially for vegans.

Vitamin B_{12} is of notable concern as it is only found naturally in foods of animal origin (see section 3.3.5). South Asian vegetarian women living in New Zealand have been found to have low serum B_{12} status (Gammon et al., 2012) and research into pre-adolescent Indian migrant girls in New Zealand has shown asymptomatic B_{12} deficiency (Rush et al., 2009).

Vegans are at particular risk of deficiency (Mann et al., 1999) as all animal foods are excluded from the diet. Among adults, a diet devoid of vitamin B_{12} may not lead to symptoms of deficiency for many years as most of us have significant body stores. In contrast, newborn infants have only small body stores and breastfed infants of unsupplemented vegan mothers may be at particular risk. One case study, for example, found a 14-month old boy who was exclusively breastfeed until 9 months of age had severe vitamin B_{12} deficiency caused by his mother's, presumably unsupplemented, vegan diet. Supplemental B_{12} rapidly improved haematological and neurological symptoms, although cognitive and language development remained seriously delayed at the age of two years (von Schenck, 1997).

Vegan mothers who are breastfeeding need to ensure an adequate intake of vitamin B_{12} and it is advised they supplement their diet to the recommended level during pregnancy and lactation (NHMRC, 2006). For vegan infants who are not breastfed, an appropriate soy-based infant formula should be used. Once a vegan infant has started to consume complementary foods, it is important to ensure a daily intake of vitamin B_{12}, with fortified foods or a supplement (Ministry of Health, 2008a).

Diets that exclude animal foods also have the potential to have low iron and zinc bioavailability. Eliminating meat, along with increasing intake of phytate-containing legumes and whole grains, reduces the absorption of both iron and zinc (Hunt, 2003) and a higher intake of these nutrients will be required in order to meet nutritional requirements. Vegetarians need iron intakes about 80% higher than non-vegetarians (NHMRC, 2006), and zinc intakes about 50% higher – particularly vegans (Hunt, 2003; NHMRC, 2006).

5. Food and Nutrition Guidelines in New Zealand

The majority of New Zealanders consume meat, and as meat is such a nutrient-dense food it can be particularly useful in the diets of population groups with high nutrient needs. It is recommended we include 1-2 servings a day of iron-containing foods in our diet. For recommended serving sizes, see Table 4.

5.1. Infants and toddlers

Lean meat can make an important contribution to the diets of infants and toddlers, providing protein, vitamins and minerals, in particular iron and zinc, which are present in a highly bioavailable form (see sections 3.3.1 and 3.3.2). Once an infant is around six months of age, puréed meat can be added to the diet with finely chopped tender meat being introduced as swallowing develops (Ministry of Health, 2008a). Iron-fortified infant cereals can be introduced from 6 months and foods containing vitamin C (eg fruits and vegetables) should be offered with meals and snacks, to assist in non-haem iron absorption. If an infant is not breast-fed, it is important to use an iron-fortified infant formula until 12 months of age. Foods containing iron inhibitors such as tea and coffee should be avoided by young children. Overall, it is important to offer a wide variety of foods from the different food groups to ensure nutritional needs are met during this period of rapid growth and development.

5.2 Children and young people (2-18 years old)

Nutritional needs are highest during rapid growth, for example during early childhood and during the adolescent growth spurt; iron needs are particularly high in menstruating girls (Ministry of Health, 2012).

Ensuring optimal iron and zinc intake remains important among children and young people. New Zealand adolescent girls, especially those of Māori or Pacific ethnicity are at greatest risk of iron deficiency anaemia; young children may also be at risk.

To ensure adequate iron intakes, it is recommended animal foods should be included in the diet, for example meat, poultry, fish and seafood along with plant foods such as breads, cereals vegetables, legumes, nuts and fruit which provide non-haem iron. Eating foods rich in vitamin C will help to enhance absorption of non-haem iron; children and young people should also avoid drinking tea with meals. At least 1-2 servings a day of iron-containing foods should be provided in the diets of children and young people. Lean meat, poultry, fish and shellfish are also good bioavailable sources of zinc (Ministry of Health, 2012).

5.3. Healthy adults

The Ministry of Health's *Food and Nutrition Guidelines for Healthy Adults* recommends maintaining a healthy weight, eating well, and being physically active every day. A variety of foods from the four main food groups should also be included daily (vegetables and fruits; breads and cereals; milk and milk products; and lean meat, poultry, seafood, eggs or alternatives). In addition, we are advised to choose foods with minimal fat, sugar and salt, to drink plenty of liquids (especially water) and to limit alcohol intake (Ministry of Health, 2003b). At least one serving a day is recommended from the meat and alternatives group to provide protein, B vitamins, iron, zinc, magnesium, copper, potassium, phosphorus and selenium.

5.4. Pregnant and breastfeeding women

Iron requirements increase significantly during pregnancy (see Table 3). However, routine iron supplements are not recommended in New Zealand as the proportion of iron absorbed from food increases in response to the increased need. They should only be given after diagnosis of iron deficiency anaemia. Iron requirements during breastfeeding are substantially lower than in pregnancy while women are not menstruating.

To ensure adequate iron intake among pregnant and breastfeeding women, dietary strategies should include consumption of at least two servings of iron-containing foods a day (Ministry of Health, 2006a). Beef and lamb can make a particularly useful contribution to intakes of iron as they are rich sources of bioavailable iron. Other sources of iron are poultry, seafood, eggs, nuts and seeds, and legumes. Monitoring of iron status throughout pregnancy is important to identify current or potential iron deficiency and all women should receive advice on dietary sources of iron and factors affecting iron absorption, in order to avoid iron deficiency. Pregnant vegetarian and vegan women may find it difficult to meet iron requirements and should be encouraged to consume plenty of iron-containing plant-based foods along with foods rich in vitamin C and to have blood levels of iron checked regularly.

Pregnant and breastfeeding women should be advised to consume a variety of nutritious foods from the main food groups to ensure adequate nutritional status.

5.5. Older people

Among older people in New Zealand (aged 65 years and older) nutrition deserves special attention as good nutrition is essential for good health and can prevent malnutrition, support physical function, reduce the risk of chronic disease, support mental health and prevent disability.

Older people should include a variety of foods in the diet from the main food groups and should drink plenty of fluids each day; in addition, at least 30 minutes of physical activity is recommended on most days of the week (Ministry of Health, 2013a). It is recommended older people have at least one serving a day of iron-containing foods, such as lean meat, skinless chicken, seafood, eggs and legumes.

There is a growing area of New Zealand research to address the issue of the difficulty of chewing meat in older adults, and subsequent reduced energy intake.

Table 4: Recommended serving sizes for meat and alternatives

Serving size for lean meat, chicken, seafood, eggs, legumes
2 slices (100g) cooked meat
¾ cup (195g) mince or casserole
1 egg (50g)
1 medium fillet of cooked fish (100g)
1 medium steak (120g)
¾ cup (135g) cooked dried beans
2 drumsticks or 1 chicken leg (110g)

Source: Ministry of Health, 2003b

6. Eating patterns of New Zealanders

Two significant national nutrition surveys carried out in New Zealand provide a comprehensive picture of New Zealanders' eating patterns. The 2008/09 New Zealand Adult Nutrition Survey, *A Focus on Nutrition* (University of Otago & Ministry of Health, 2011), studied New Zealanders aged 15 years and older. This survey updates the 1997 survey *NZ Food: NZ People* (Russell et al., 1999). The 2002 survey, *NZ Food NZ Children* (Parnell et al., 2003) looked at New Zealand children aged 5 to 14 years.

In the Adult Nutrition Survey (2008/09), most of the population (94.5%) reported eating red meat in the previous four weeks, with red meat eaten 1-2 times per week by 30.1% and 3-4 times per week by 45.4%. More than half the population trimmed the excess fat from meat regularly or always (University of Otago & Ministry of Health, 2011).

Among children, 95% consume an omnivorous diet, with just 3.6% avoiding red meat and 0.7% avoiding all meat (Parnell et al., 2003). Meat makes a valuable contribution to the intake of a range of nutrients for many New Zealanders (see section 3).

7. Are current recommendations for meat intake adequate?

Overall, the Ministry of Health's food and nutrition guidelines suggest 1-2 portions per day should be eaten from the meat and alternatives food group. However, in order to optimise health and prevent chronic disease, recommendations on the number of servings consumed from the meat and alternatives food group may need to be reviewed. Dietary modelling in Australia (NHMRC 2011) demonstrated the most limiting nutrient of the 10 nutrients modelled in the low energy Omnivore Foundation Diets was iron. The dietary models developed were unable to provide sufficient iron to fulfil the estimated requirements of pregnant females as a group.

Legumes, nuts and seeds certainly provide valuable nutrients and should be included in a balanced diet, but these foods are not direct substitutes for foods of animal origin in terms of the nutrients they provide. The recommended number of servings from the meat and alternatives group, along with the serving sizes, may need to be reconsidered and recommendations in relation to legumes, nuts and seeds as alternatives to meat may need to be revised.

Specific and separate advice and recommendations may be needed for lacto-vegetarians and vegans to ensure their nutritional requirements are met. However, the total combination of foods consumed over time is a more important consideration than the intake of individual foods, so further research and analysis of this issue is warranted before firm recommendations can be made.

8. The role of red meat in health and disease

Meat consumption has been linked to a number of diseases, most notably cancer and heart disease. Research on heart disease and cancer is reviewed in sections 8.1.1 and 8.1.2.

Research evaluating the role of high-protein diets in promoting satiety and aiding weight loss is reviewed in section 8.3. Red meat is an excellent source of protein (see section 3.2) and could make a valuable contribution to protein intakes (along with other protein foods). This may be helpful for those managing their weight. The effects of a diet high in protein have also been evaluated in those with Type 2 diabetes and insulin resistance, with initial results showing such diets may be helpful (see section 8.4).

Emerging research suggests that red meat may also play a role supporting optimal mental health. Specific analysis of red meat in relation to mood and anxiety disorders among women has shown that those consuming less than the recommended intake of red meat are at increased risk of depression, although more research is needed in this area to confirm which dietary strategies support optimal mental health (see section 8.5).

The role of meat in health and disease has been evaluated in a number of studies, which are outlined below.

8.1. Coronary heart disease (CHD)

8.1.1 Dietary fats and coronary heart disease

A Cochrane review on modifying dietary fat intakes for preventing cardiovascular disease concluded that replacing saturated fats with plant oils and unsaturated spreads may reduce risk of heart and vascular disease, although it was not clear whether monounsaturated or polyunsaturated fats are more beneficial (Hooper et al., 2012).

More recent research has questioned whether a higher consumption of polyunsaturated fats and a lower consumption of saturated fats should be encouraged, and suggests this approach is not supported by recent evidence (Chowdhury et al., 2014; Schwingshackl & Hoffmann, 2014; Calder, 2013; Ramsden et al., 2013; Ravnskov et al., 2014; Thornley et al., 2014). However, the meta-analysis of dietary fatty acids and risk of coronary heart disease by Chowdhury et al. (2014), which reports no significant association betweem CHD outcomes and intakes of SFA, MUFA and both *n-3* PUFA and *n-6* PUFA has been highly criticised. Experts claim it contains multiple errors and omissions, and the conclusions are seriously misleading (Willett et al., 2014).

There have been a number of systematic reviews that have supported a reduction in intake of saturated fats; for example a pooled analysis of 11 prospective cohort studies by Jakobsen et al in 2009 found that substituting 5% of energy from saturated fats with 5% of energy from polyunsaturated fats was associated with a significant reduction in CHD events. MUFA was not associated with CHD in this review. A further review by Mozaffarian et al in 2010, which was a meta-analysis of 8 randomised controlled trials, also found that replacing saturated fats with polyunsaturated fats reduced CHD events. Further, a recent New Zealand review of the highest quality systematic reviews suggests that replacing 5% of daily energy consumed as saturated fat with polyunsaturated fats would reduce ischaemic heart disease events by about 10% and that such a dietary change would be desirable and feasible for the New Zealand population (Foster & Wilson, 2013). There are no benefits in replacing saturated fats with refined starches, especially sugar (Hooper et al., 2012; Lawrence, 2013; Te Morenga et al., 2014a, Te Morenga et al., 2014b).

Experts in New Zealand have recently reviewed the available literature and conclude that numerous high quality experimental trials have provided unequivocal evidence that dietary saturated fat raises serum cholesterol levels when compared with polyunsaturated and monounsaturated fats (Te Morenga et al., 2014a).

The New Zealand Heart Foundation recommends replacing foods high in saturated fat with unsaturated fats (Gorton, 2014). The Heart Foundation advises that a diet of mostly minimally processed foods (including plenty of vegetables and fruit; plus legumes, nuts, whole grains, plant oils, and fish; as well as choosing lean meats and reduced-fat dairy) is the best way of eating for a healthy heart. Dietitians New Zealand considers there not to be any substantive evidence that saturated fat is good for you in the long term (Dietitians New Zealand, 2014). It has been suggested that

saturated fats be replaced with more *n-3* polyunsaturated fatty acids. (Te Morenga et al., 2014a).

It is appropriate to regularly review nutrition recommendations in the light of new evidence; however, the best quality evidence at the present time supports current advice to reduce intake of SFA (Te Morenga et al., 2014b).

8.1.2 Meat and coronary heart disease

Health messages specifically in relation to meat can be confusing and misleading (Li et al., 2005) and advice to reduce red meat as part of a heart-healthy diet is inappropriate. A number of studies have shown lean red meat can be included in a heart-healthy diet as it is low in total fat and saturated fatty acids.

8.1.2.1. The role of lean meat in cholesterol-lowering diets

A study aiming to differentiate between lean beef and beef fat as risk factors for elevated plasma cholesterol, found total cholesterol concentrations fell significantly within one week of commencing a low-fat diet that included lean beef, and rose as beef dripping was added in a stepwise manner (O'Dea et al., 1990). This demonstrates clearly it is the beef fat and not the lean beef that is associated with elevations in cholesterol levels and shows lean beef can be part of a cholesterol-lowering diet. A further study, which looked at dietary determinants of ischaemic heart disease (IHD) in health conscious individuals, concluded that dietary saturated animal fat and cholesterol are important in the aetiology of IHD (Mann et al., 1997a). These factors, rather than simply meat, appeared to explain the higher IHD rates reported in meat eaters compared with vegetarians.

Substituting poultry for lean red meat is unlikely to have any effect on total or LDL cholesterol levels. A randomised controlled trial among hypercholesterolaemic free-living men and women comparing lean red meat with lean white meat found both produced similar reductions in LDL cholesterol and elevations in HDL cholesterol (Davidson et al., 1999). A further randomised cross-over study, with two 36-week phases separated by a 4-week washout period, compared the effects of lean red meat and poultry in reducing cholesterol in people with hypercholesterolaemia (Hunninghake et al., 2000). Results showed both had an identical effect, with a 1% reduction in total cholesterol and a 2% reduction in LDL cholesterol. In this study, the lean meat was part of a diet providing less than 30% energy from fat and 8-10% energy from saturated fatty acids.

A further study on hypercholesterolaemic men (Beauchesne-Rondeau et al., 2003) found diets containing lean beef or poultry reduced plasma total and LDL cholesterol concentrations by 8% each, with a 5% reduction in the lean fish-containing diet.

Analysis of the diets of adolescent girls also suggests that lean red meat may be included in a healthy diet without unfavourable effects on lipid profiles (Bradlee et al., 2013). Another meta-analysis found that changes in the fasting lipid profile were not significantly different with beef consumption compared with poultry and/or fish consumption (Maki et al., 2012). Including lean beef in the diet increases the variety of food choice and may improve the long-term adherence with dietary recommendations for lipid management, say the authors of this study.

It has been suggested iron can contribute to oxidative stress and inflammation, which are possible risk factors for heart disease and diabetes. One study investigated the effects of lean red meat on markers of oxidative stress and inflammation in humans (Hodgson et al., 2007). Sixty subjects were randomised to either maintain their usual diet, or to partly replace energy from carbohydrate with 200g of lean red meat daily. No elevation of oxidative stress or inflammation was found among the meat-eating group.

A review of 54 studies that looked at meat consumption and CHD risk factors, found substantial evidence that lean meat trimmed of visible fat does not raise blood cholesterol and LDL cholesterol levels (Li et al., 2005), as long as the overall diet is low in total and saturated fat. In fact, the overall effect was diets low in saturated fatty acids, which included lean red meat, were associated with a reduction of LDL cholesterol levels in both hypercholesterolaemic and healthy subjects. Thrombotic risk factors such as thromboxane and prostacyclin production, platelet function and haemostatic factors also remain unchanged with the inclusion of lean red meat (Mann et al., 1997b).

Where lean meat is eaten, there appears to be little difference between meat-eaters and vegetarians in terms of blood lipid levels, as long as the overall diet is low in fat and saturated fatty acids. The Heart Foundation states small or moderate servings of lean meat can be included as part of a normal, varied diet (National Heart Foundation, 1999).

8.1.2.2. Advice on meat in relation to CHD

The New Zealand Guidelines Group (2012) for cardiovascular risk factor management recommends including fish or dried peas, beans and soy products, or a small serving of lean meat or skinned poultry, at one or two meals each day. A serving of meat is 2 slices (100-120g) or half a cup of minced meat (125g). Intake of fatty meat and meat products (eg meat pies, sausage rolls, tinned corned beef and salamis) should be low, and all visible fat should be trimmed from meat before consumption (National Heart Foundation, 1999).

8.2. Cancer

8.2.1. Incidence of colorectal cancer in New Zealand

In 2008, 2,801 people were diagnosed with bowel cancer and 1,280 people died from the disease. Colorectal cancer was the second most common cause of death from cancer in New Zealand, accounting for 15% of all deaths from cancer (Ministry of Health, 2011).

8.2.2. Red meat and colorectal cancer

Some scientific studies have suggested a link between red meat consumption and colorectal cancer and the World Cancer Research Fund report (WCRF, 2007) and the 2011 Continuous Update Project (CUP) Report on Colorectal Cancer concluded red and processed meats are a convincing cause of colorectal cancer based on a substantial amount of data from cohort studies showing a dose-response relationship. However, this remains controversial and the subject of scientific debate. Diet is

remarkably difficult to measure, and the separation of the effects of individual food components is extremely complicated, given the multiple correlations that exist between the different elements (Boyle et al., 2008). The recommendation from the World Cancer Research Fund is to consume up to 500g cooked red meat per week with the average beef and lamb intakes in New Zealand currently sit below this level at around 400g/week. The 2011 CUP report on colorectal cancer confirms the evidence for a protective effect from foods containing dietary fibre has strengthened since the 2007 WCRF report.

Some meta-analyses have found that a high intake of red and processed meat is associated with an increased risk of colorectal cancer. (Sandhu et al., 2001; Norat et al., 2002; Larsson et al., 2006; Chan et al., 2011).

The meta-analysis by Sandhu et al. (2001), found a daily increase of 100g of all meat or red meat was associated with a significant 12-17% increased risk of colorectal cancer. A significant 49% increased risk was found for a daily increase of 25g of processed meat. However, as only a few of the studies reviewed attempted to examine the independent effect of meat intake on colorectal cancer risk, the overall association may have been confounded by other factors.

A further meta-analysis by Norat et al. in 2002 also found a high intake of red meat, and particularly processed meat, was associated with a moderate but significant increase in colorectal cancer risk. Average relative risks and 95% confidence intervals (CI) for the highest quantile of red meat consumption were 1.35 (CI: 1.21-1.51) and for processed meat were 1.31 (CI: 1.13-1.51). No significant association was found for total meat consumption and colorectal cancer risk. The relative risks for total and red meat were higher in studies including processed meat in the definition of these two meat groups, than in studies that evaluated fresh meat and fresh red meat.

Similar results were found in a meta-analysis in 2006 by Larsson et al., which found consumption of red meat and processed meat was positively associated with risk of both colon and rectal cancer. The summary relative risks of colorectal cancer for the highest versus the lowest intake categories were 1.28 (95% CI: 1.15-1.42) for red meat and 1.20 (95% CI: 1.11-1.31) for processed meat.

A more recent meta-analysis (Chan et al., 2011) found the summary relative risk of colorectal cancer for the highest versus the lowest intakes of meat was 1.22 (95% CI = 1.11-1.34). Relative risk for every 100g/day increase was 1.14 (95% CI = 1.04-1.24). The mean values of the highest category of red and processed meat intake in the studies ranged from 46g to 211g per day. The authors conclude that overall evidence supports limiting red and processed meat consumption as one of the dietary recommendations for the prevention of colorectal cancer.

One of the largest studies of diet and health ever undertaken is the European Prospective Investigation into Cancer (EPIC). Results from this study, based on 478,040 men and women, support the hypothesis that colorectal cancer risk is positively associated with intake of red and processed meat (highest intake was over 160g per day, versus the lowest intake which was less than 20g per day) (Norat et al., 2005). The association with colorectal cancer was stronger for processed than for unprocessed red meat.

An expert workshop held in New Zealand in 1999, concluded there is no convincing evidence from published epidemiological studies that moderate intakes of lean red meat increase the risk of colorectal cancer when eaten as part of a mixed diet including carbohydrates, vegetables and fruits, and dairy products (Tasman-Jones et al., 2000).

More recent meta-analyses have shown that associations between red meat consumption and colorectal cancer are generally weak in magnitude, with most relative risks being below 1.5 and not statistically significant, there is also a lack of a clear dose response trend (Alexander & Cushing, 2010; Alexander et al., 2011).

8.2.3. Possible mechanisms linking meat consumption with colorectal cancer

There are a number of possible mechanisms for a link between meat consumption and colorectal cancer; including the promotion of carcinogenesis by high-fat diets, the production of carcinogenic heterocyclic amines (HCAs) and polycyclic aromatic hydrocarbons (PAHs), the promotion of carcinogenesis by haem iron, and the formation of carcinogenic N-nitroso compounds (NOCs) both within meat and endogenously (WCRF, 2007; Baghurst, 2007; Santarelli et al., 2008).

Although fat intake from meat has been suggested to explain a link between colorectal cancer and meat intake, experimental studies show inconsistent results and epidemiological studies have failed to confirm a link (Santarelli et al., 2008). There is now little support for the notion that fat in meat promotes carcinogenesis (Baghurst, 2007). The 2011 WCRF Continuous Update Project (CUP) reports the evidence suggesting that consumption of foods containing animals fats is a cause of colorectal cancer is limited.

HCAs are produced during high-temperature cooking of meat, such as frying and when using a barbecue. Such high cooking temperatures cause amino acids and creatine to react together to form HCAs (WCRF, 2007). PAHs are produced from the incomplete combustion of organic compounds; the main sources are cooked and smoked meat and fish (notably barbecued meat) and tobacco smoke (Santarelli et al., 2008). Around one third of meat consumed on a daily basis in New Zealand is cooked by methods likely to result in the formation of HCAs (Thomson, 1999). However, a recent review of HCAs concluded there is not sufficient scientific evidence to support the hypothesis that human cancer risk is specifically due to the intake of HCAs in the diet (Alaejos et al., 2008). Data on PAHs in overcooked meat suggest these may be a risk factor, but there is insufficient evidence to draw firm conclusions (Santarelli et al., 2008). A more recent Australian population-based case-control study asked subjects to complete questionnaires on lifestyle and meat consumption (Tabatebaei et al., 2011). Baked red meat had a statistically significant inverse trend of association with colorectal cancer. Overall, results did not support an association between meat consumption or meat cooking practices, and the risk of colorectal cancer.

Haem iron may catalyse the formation of NOCs from natural precursors in the gut. Red meats are a richer source of haem iron than white meats, so such an effect may theoretically explain a stronger association between red meat and colorectal cancer, than between white meat and colorectal cancer. It would not explain, however, why white meat and fish (which also contain haem iron) appear to be protective against

colorectal cancer (Baghurst, 2007). The 2011 CUP panel agreed the evidence suggesting foods containing iron to be a cause of colorectal cancer to be limited.

NOCs are alkylating agents that can react with DNA and are produced by the reaction of nitrite and nitrogen oxides with secondary amines and N-alkylamides (Santarelli et al., 2008). NOCs are present in certain processed meats (eg grilled bacon), smoked fish, cheeses and beer, and can be formed endogenously after red and processed meat consumption (Santarelli et al., 2008). Although some research has linked NOCs to cancer, it is not yet clear whether red and processed meat-induced NOCs are colon carcinogens (Santarelli et al., 2008). Further research is needed in this area.

8.2.5. Reducing risk of colorectal cancer (CRC)

Epidemiological and mechanistic data on associations between red meat and colorectal cancer are inconsistent, and underlying mechanisms are unclear; there is a need for further research into the differences between white meat, red meat and processed meat and there is a need for further investigation of biomarkers of meat intake and cancer occurrence (Oostindjer et al., 2014).

There are many dietary and lifestyle factors that have an influence on the development of cancer. In many studies looking at the effects of diet on cancer, it is difficult to disentangle the dietary and lifestyle factors that may be involved (such as Western lifestyles, high intake of refined sugars, alcohol, low intakes of fruits, vegetables and fibre) and behavioural factors (such as smoking, lack of physical activity and high body mass index). This limits the ability to analytically isolate the independent effects of red meat consumption; currently available epidemiological evidence is not sufficient to support an independent positive association between red meat consumption and colorectal cancer (Alexander & Cushing, 2010).

The key focus in terms of cancer prevention should be to: avoid smoking, limit sun exposure, maintain a healthy body weight and be physically active as part of everyday life. In terms of diet, it is recommended we eat at least five portions of a variety of fruits and vegetables each day, along with relatively unprocessed cereals and pulses, and limit intake of alcohol to no more than 1 unit a day for women and 2 units a day for men (WCRF, 2007). The 2011 CUP colorectal report confirms the evidence for ethanol from alcoholic drinks being a cause of colorectal cancer in men is convincing and probable in women. In addition the CUP panel agree the evidence showing physical activity to protect against colon cancer is convincing, and greater body and abdominal fatness as a CRC cause to be convincing.

Lean beef and lamb can make an important nutritional contribution to a balanced diet and complete avoidance is unnecessary in terms of cancer prevention. Also of note is that some of the nutrients in red meat, such as selenium, and vitamins B6, B12 and D, may have anti-cancer properties. Adjusting the dietary balance between meat and other dietary components may be critical in protecting against potential cancer (Ferguson, 2009). The 2011 CUP CRC report confirms evidence for foods containing vitamin D and/or selenium is limited.

As research in this area continues, it may be prudent to avoid very high intakes, particularly of processed meats, and to limit very-high-temperature cooking methods. The WCRF (2007) have recommended that intakes of cooked red meat can be up to

500g per week – this is higher than the current average intake of red meat in New Zealand of around 50g per day (Parnell et al; 2012) equating to 350g/week, so a reduction in average intakes is unnecessary.

8.3. Obesity

Conservative estimates suggest over 1 billion people worldwide are overweight or obese (Simpson & Raubenheimer, 2005). Obesity is certainly a significant problem in New Zealand, three out of ten adults (31%) are now obese, and one in nine (11%) of children aged 2-14 years are obese. This means that 1.2 million New Zealanders are obese (Ministry of Health 2013b).

8.3.1 Protein and weight management

Evidence is accumulating that increasing intakes of high-quality protein to a level above the recommended intake may be beneficial during weight loss (Layman, 2004), as well as for diabetes and cardiovascular disease (Rodriguez & Garlik, 2008). This may be related to the positive effects of protein on appetite control, satiety and food craving and reduction of intake of fat and carbohydrate (Santesso et al, 2013).

Astrup et al. 2013 looked at the effect of protein-induced satiety on appetite hormones which concluded protein dose-dependency increased satiety and postprandial changes in circulating GLP-1, PYY and glycagon were in part, responsible for the appetite suppressant effect of protein.

Energy intake has been shown to increase as dietary protein falls from 20% to 10%; a recent review on protein leverage and energy intake concludes that dilution of protein in the diet stimulates excess energy intakes, and that there is strong support for a role of protein leverage in lean, overweight and obese humans Gosby et al., 2014).

For most of our existence, the human diet has consisted of a high proportion of animal foods, with meat consumed from wild animals typically having a low fat content (see section 2). As a result, we have limited evolutionary experience of excess carbohydrates or fats and it has been suggested natural selection against over-consumption of these nutrients would not have been strong; this may account for their high level of palatability and may predispose us to their over-consumption today (Simpson & Raubenheimer, 2005).

In terms of systematic reviews and meta-analyses, 15 randomised controlled trials looked at the long term effects of higher vs lower protein diets on health outcomes including adiposity (Schwingshackl & Hoffman, 2013). Data analysis revealed no significant changes for weight, waist circumference, fat mass, total cholesterol and blood pressure. Significant decreases for fasting insulin were observed in high protein diets compared to low protein diets in the primary analysis, but not so in the secondary analysis. It summarised high protein diets exerted neither specific beneficial or detrimental effects on outcome markets of obesity, cardiovascular disease or glycaemic control.

In a large European study (Larson et al., 2010), 773 participants completed a low calorie phase, and were then randomly assigned to one of five weight maintenance diets; 548 completed the intervention. The mean initial weight loss on the low-calorie

diet was 11.0kg; among those who completed the study, only those following the low-protein, high-glycaemic index diet significantly re-gained the weight. The authors conclude that a modest increase in protein (to 25% of energy), and a modest reduction in glycaemic index, led to an improvement in study completion and maintenance of weight loss.

More recently, the DIOGENES 12 month randomised clinical trial (Aller et al., 2014) looked at both the GI and protein content effect on overweight subjects who were put on an initial low calorie diet for 8 weeks. After the intervention, the trial found no consistent effect of GI on weight gain, but did show a diet higher in protein (23-28%) improved weight loss maintenance in overweight and obese adults.

More evidence for the effect of protein is provided by a meta-analysis of 87 studies comprising 165 intervention groups, which found low carbohydrate, high-protein diets affected body mass and composition favourably, independent of energy intake (Kreiger et al , 2006). This supports the proposed metabolic advantage of such diets.

A more recent systematic review and meta-analysis compared an energy restricted standard protein diet with an isocalorically prescribed high protein diet and found a beneficial effect on weight loss, body composition and triglycerides with higher protein intakes (Wycherley et al., 2012).

Concern has been expressed about the effects of high-protein diets on renal function; however, there is little evidence high-protein diets pose a serious risk to kidney function in healthy people (Halton & Hu, 2004). Similarly, the impact of high-protein diets on markers of bone turnover has not been found to be deleterious (Noakes et al., 2005; Farnsworth et al., 2003).

Many of the studies into protein and weight loss have been relatively small. Optimal amounts and sources of protein cannot be determined, but the weight of evidence suggests it may be beneficial to partly replace refined carbohydrate intake with low-fat protein sources (Halton & Hu, 2004).

8.3.2 Fat and carbohydrates and weight management

It is important to consider not only the protein content of the diet but also the fat and carbohydrate content. Research into low-carbohydrate versus low-fat diets is mixed. One study on 96 normoglycaemic insulin-resistant women randomised to one of three dietary interventions (high carbohydrate, high fibre; high fat; or high protein) for 8 weeks of supervised weight loss, and 8 weeks of supervised weight maintenance, found significantly greater reductions in weight on the high-fat and high protein diets, when compared with the high-carbohydrate diets (McAuley et al., 2005). However, the authors suggest that to achieve similar benefits on a high-carbohydrate diet, it may be necessary to increase fibre-rich whole-grains, legumes, vegetables and fruits, and to reduce saturated fatty acids to a greater extent. Although the high-fat diet was successful for weight loss in the short term, the authors expressed concern that lipid levels should be monitored owing to the possible deleterious effects of this diet in the long-term.

A recent meta-analysis compared the effects of low-carbohydrate diets (≤ 45% energy from carbohydrates) versus low fat diets (≤ 30% energy from fats) on metabolic risk

factors and weight loss (Hu et al., 2012). Reductions in body weight, waist circumference and other metabolic risk factors were not statistically significant between the two diets.

When looking at high carbohydrate diets it is important to consider carbohydrate quality, which is not addressed in many studies. Recent research has compared a high protein diet (28% of energy from protein, of which 75% was derived from animal sources), with a low-fat high-carbohydrate diet (22% energy from protein) that was rich in dietary fibre from minimally processed grains, cereals and legumes (Te Morenga & Mann, 2012). Each diet provided 24g/d and 39g/d of dietary fibre respectively. After 8 weeks both groups lost weight, but the high protein participants lost 1.3kg more weight and achieved a greater reduction in diastolic blood pressure. Although improvements in risk factors were most marked on the high protein diet, results did not achieve statistical significance.

A recent position statement by the New Zealand Dietetic Association considers there not to be any evidence that a diet high in fat and low in carbohydrates is more beneficial for sustained weight loss than any other dietary regimen that results in a lower intake of energy (Dietitians New Zealand, 2014).

Further research in needed into the optimal dietary balance of fats and carbohydrates for long-term weight-loss. However, it seems reasonable to suggest that relatively higher protein intakes are an appropriate option for the treatment and avoidance of excess body fat (Te Morenga & Mann, 2012). For weight loss, the diet should be reduced in energy, and carbohydrate sources should be low in glycaemic index with minimally processed grains, cereals and legumes. The inclusion of lean red meat as part of a balanced diet would contribute to increased protein intakes.

8.4. Type 2 Diabetes

High-protein, low-carbohydrate diets have also been examined for treatment of Type 2 diabetes mellitus. Positive effects have been found on glycaemic regulation, including: reductions in fasting blood glucose; reductions in post-prandial glucose; reductions in insulin responses; and a reduced percentage of glycated haemoglobin (Layman et al., 2008). More recently, it has been claimed that although low carbohydrate diets are still controversial, they have continued to demonstrate effectiveness with little risk and good compliance for those with diabetes (Feinman et al., 2015).

A study comparing a high protein diet (28% of energy) with a low protein diet (16% of energy) in 54 obese men and women with Type 2 diabetes, during 8 weeks of energy restriction and 4 weeks of energy balance, found both diets improved the cardiovascular disease risk profile as a consequence of weight loss (Parker et al., 2002). However, there were greater reductions in total and abdominal fat mass in women, and greater LDL cholesterol reduction in both sexes, with the high protein diet. This suggests high protein diets are a valid choice for reduced risk of cardiovascular disease in people with Type 2 diabetes. Subjects in this study derived their protein from foods such as beef, chicken and dairy products.

A further study on overweight and obese hyperinsulinaemic men and women found no difference in weight loss or fat mass loss between subjects fed a high protein diet (27% of energy) compared with a lower protein diet (16% of energy) during 12 weeks of energy restriction and 4 weeks of energy balance (Farnsworth et al., 2003). However, in women total lean mass was significantly better preserved with the high protein diet (-0.1 ± 0.3 kg) than with the standard protein diet (-1.5 ± 0.3 kg). Further, those on the high protein diet had significantly less glycaemic response and a greater reduction in triacylglycerol concentrations than those on the standard protein diet.

Although there have been relatively few studies comparing the effects of high protein diets varying in macronutrient composition on insulin sensitivity, limited data do suggest that moderately high protein, weight loss diets (25-30% energy) improve glucose metabolism and insulin sensitivity when compared with low fat, high carbohydrate diets (Te Morenga & Mann, 2012).

Further studies are warranted into the effect of high protein diets on delaying the progression to Type 2 diabetes in obese adults with insulin resistance. Initial indications are however, for some individuals a diet providing an increased level of protein and a reduced level of carbohydrate may be effective for weight management, may improve lipid profiles, and may improve glycaemic regulation (Layman et al., 2008). However, it is important to note again the nature of the carbohydrates consumed in many studies was not considered (Te Morenga & Mann, 2012). More research is needed to determine the types and amounts of macronutrients to include in an optimal diet for people with diabetes

8.5 Mental Health

Mental health is an integral part of health and well being, as reflected in the definition of health in the Constitution of the World Health Organization: “Health is a state of complete physical, mental and social well-being and not merely the absence of disease or infirmity.” (WHO, 2013).

Depression alone accounts for 4.3% of the global burden of disease and is among the largest single causes of disability worldwide (WHO, 2013). In New Zealand, rates of diagnosed mental health conditions are on the rise with rates of psychological distress high among Maori and Pacific adults, and adults living in the most deprived areas (Ministry of Health, 2014).

Recent research suggests nutrition is a key factor that underpins depression, with healthy dietary practices associated with a reduced likelihood of both clinically diagnosed depressive and anxiety disorders, and unhealthy dietary habits associated with an increased likelihood of major depressive disorder, dysthymia and anxiety disorders (Jacka et al., 2010).

Healthy dietary patterns consisting of vegetables, salads, fruits, rice, pasta, cereals, wine and non-processed meats have been compared with ‘Western’ dietary patterns consisting of processed meats, pizza, salty snacks, chocolates, sugar, sweets, soft drinks, margarine, mayonnaise, French fries, beer, coffee, cake and ice-cream. Those on better quality diets have been shown to be loss likely to be depressed whereas a higher intake of unhealthy and processed foods has been associated with an increased

level of anxiety (Jacka et al., 2011a). The hypothesis of reverse causality is not supported by the available data, in other words, the reported associations do not reflect poorer eating habits as a consequence of mental health problems (Jacka et al., 2011b).

Specific analysis among women eating red meat in relation to mood and anxiety disorders, has shown for those women consuming less than the recommended intake of red meat per week (3-4 serves of 65-100g a week of red meat such as beef and lamb), the odds for major depressive disorder/dysthymia were more than doubled compared to those consuming the recommended intakes (Jacka et al., 2012). The authors conclude that red meat consumption may play a role in mental health independently of overall dietary quality, but further studies are needed before recommendations can be made.

It is likely it is overall dietary patterns that are protective, rather than a single food component. I has been suggested, for example, the synergistic combination of *n-3* fatty acids together with other unsaturated fatty acids and antioxidants from olive oil and nuts, flavanoids and other phytochemicals from fruit and other plant foods, and large amounts of natural folates and other B vitamins exert a fair degree of protection against depression (Sánchez-Villegas et al., 2009).

A randomised controlled trial is currently underway to investigate the efficacy of dietary improvements in the treatment of depression (O'Neil et al., 2013). If the results of this study are positive, dietary intervention could provide an alternative or adjunctive treatment strategy for the management of mental disorders. In the meantime, it makes sense to follow a healthy diet for optimal physical and mental wellbeing.

9. Conclusions

Meat has been an important part of the human diet throughout our evolutionary history and today most New Zealanders include meat in their diet. Lean New Zealand beef and lamb are nutrient-dense foods that play a pivotal role throughout the life cycle – from young infants and children, through to adults and older people.

In particular, red meat is a rich source of bioavailable iron, which is important for vulnerable groups such as infants and toddlers, adolescents and women of childbearing age. Meat also provides zinc, selenium, B vitamins (particularly vitamin B_{12}), vitamin D and *n*-3 fatty acids, and liver is an excellent source of vitamin A. Red meat is also an excellent source of protein, and when fully trimmed, is low in total and saturated fatty acids.

The combination of nutrients found in meat can play an important role in the health issues facing many New Zealanders today. For example, lean meat can be a helpful part of a heart-healthy diet for those at risk of cardiovascular disease; it can form a part of a weight-reducing diet for obese and overweight people; and may have beneficial effects in preventing and managing Type 2 diabetes. In terms of cancer prevention, the key focus should be to avoid smoking, limit sun exposure, maintain a healthy weight, and be physically active. In relation to diet, the emphasis should be on fruits, vegetables and unprocessed cereals and pulses, as well as limiting alcohol

intake. A reduction in red meat intake in New Zealand is unnecessary based on current scientific evidence. However, it may be prudent to avoid very high intakes, particularly of processed meats, and to limit very-high-temperature cooking methods.

Appendix 1: Common myths and misconceptions about meat

Red meat is high in fat

When trimmed of all visible fat, lean red meat is low in fat. For example, a 100g portion of cooked beef silverside contains 5g fat and within that, only around half the fat in meat is saturated; the rest is mainly the beneficial monounsaturated and polyunsaturated fats. Since 1997, the red meat industry's Beef and New Zealand Quality Mark has required the trimming of beef and lamb cuts to be no more than 5mm of external fat and resulted in 30% less total fat and 65% less saturated fat in beef and lamb cuts.

People with heart disease should avoid red meat

A number of studies have shown lean red meat can be included in a cholesterol-lowering diet. Intake of fatty meat and meat products should be low for people with heart disease and all visible fat should be trimmed from meat before consumption. The New Zealand Guidelines Group (2012) for cardiovascular risk factor management recommends including fish or dried peas, beans and soy products, or a small serving of lean meat or skinned poultry, at one or two meals each day.

Weight-loss diets should exclude red meat

Lean red meat is low in fat and calories, and moderate amounts can be included in a weight-reducing diet. Evidence is accumulating that increasing the intake of high-quality protein to a level above the recommended daily amount (RDA), may be beneficial during weight loss; protein has been found to suppress food intake as it contributes to satiety, promoting a feeling of fullness. The inclusion of lean red meat as part of a balanced diet may therefore help weight loss as part of a reduced energy diet.

Red meat causes cancer

Some scientific studies have suggested an association between red meat consumption and colorectal cancer. However, associations are weak and overall evidence is mixed. An expert workshop in New Zealand concluded a moderate intake of lean meat as part of a balanced diet, which also provides adequate cereals and grain foods, vegetables and fruit, is not associated with an increased risk of bowel cancer. There are many dietary and lifestyle factors that influence the development of cancer and the key focus in terms of cancer prevention should be to avoid smoking, limit sun exposure, maintain a healthy body weight, be physically active, eat at least five portions of a variety of fruits and vegetables each day, along with unprocessed cereals and pulses, and limit intake of alcohol. The World Cancer Research Fund recommendation is that people who eat red meat (defined as beef, lamb and pork) should consume less than 500g cooked red meat per week. Current average red meat intakes in New Zealand are below this amount.

Meat-eaters should become vegetarian if they want to be healthy

A diet excluding meat can be nutritionally adequate, but as more foods are excluded it becomes important to plan the diet carefully to ensure nutrient needs are met. In particular, intakes of iron, zinc and vitamin B_{12} need careful consideration – especially for vegans. In terms of chronic disease, vegetarians have a lower mortality rate than omnivores, although it is likely much of this effect can be achieved by not smoking, by exercising more and by consuming a diet higher in fruits, vegetables and fibre. It is difficult to disentangle which features of a vegetarian diet may be protective, and there is currently no evidence to suggest meat eaters should change to an entirely vegetarian diet for health reasons.

Spinach is the best source of dietary iron

Spinach is a good source of iron, but the iron is present in the non-haem form, which is poorly absorbed therefore should be eaten with vitamin C-rich foods such as tomato or capsicum. Also, spinach contains substances that inhibit the absorption of iron, such as polyphenols and oxalic acid. As a result, spinach is a relatively poor source of iron, especially when compared with red meat, which contains the more readily-absorbed haem iron.

Eating too much meat can lead to an excess iron intake

Absorption of iron from dietary sources is well controlled by the body and although red meat is an excellent source of iron, including it regularly in the diet will not lead to an excess iron intake for healthy people. In fact, iron deficiency is much more likely to be a problem. The most common iron overload condition in New Zealand is hereditary haemochromatosis, a genetic condition that causes poor control of iron absorption. This condition is managed by therapeutic phlebotomy - in other words, the removal of blood on a regular basis, not by the avoidance of meat.

Meat takes a long time to digest

From an evolutionary perspective, humans are naturally omnivores and our digestive system is well adapted to digesting meat. Around 94% of the protein in meat is digested; this compares with 86% in whole wheat and 78% in beans (Williams, 2007). Meat is therefore an easily digested food. In addition, the nutrients in meat are well absorbed and utilised by the body.

Appendix 2: Production of red meat in New Zealand

Farming practices

The unique climate and landscape in New Zealand has set the global benchmark for pastoral farm production. Most meat is produced using naturally available resources – grass, rain and sunshine. In New Zealand, there is year-round access to grass, including hay, silage and feed crops.

Sustainability

Researchers in the USA have suggested a meat-based diet requires more energy, land and water resources than a lacto-ovo-vegetarian diet (Pimentel & Pimentel, 2003) implying the lacto-ovo-vegetarian diet is more sustainable. However, these suggestions often assume land used for grazing animals can be diverted to other uses, such as crop production (Thomason, 2007). Furthermore, these studies are likely to have made the comparison with feedlot beef, rather than the extensive pastoral systems. In New Zealand, most livestock production takes place on land unsuitable for producing crops, and if this land were not used for grazing, it would essentially be agriculturally unproductive

Water foot-printing of New Zealand beef and lamb production shows that the majority of water used is from natural rainfall, rather than from other sources of water, and often not reflected in sustainability comparisons.

As such, beef and lamb production in New Zealand is highly sustainable.

Greenhouse emissions

A recent report has shown that greenhouse gases, including carbon dioxide (CO_2), methane (CH_4) and nitrous oxide, have continued to climb during 2013 once again reaching historic high values. Atmospheric CO_2 concentrations increased by 2.8 ppm in 2013, reaching a global average of 395.3 ppm for the year (Blunden & Arndt, 2014). Globally, livestock contributes a significant share towards emissions, but can also deliver a significant share of the necessary mitigation efforts (Gerber et al., 2013).

New Zealand is a signatory to the Kyoto Treaty on climate change and has made a commitment to reduce greenhouse emissions to 1990 levels. To help achieve this aim, the Pastoral Greenhouse Gas Research Consortium (PGgRc) was set up in 2002. A key goal of the PGgRc is to develop strategies to reduce and mitigate the two greenhouse gases associated with livestock: methane and nitrous oxide.

The contribution of agriculture to New Zealand's emissions profile is currently 46.1%, down from 50% in 1990. Emission levels from the beef and sheep sector have been decreasing and are now 17% lower than in 1990. Over the same period, emissions from agriculture have been increasing, therefore the sheep and beef sectors' contribution as a proportion is lower than it was 15 years ago. Breeding programmes along with the production of fewer but larger animals are largely responsible for the increase in efficiency in this sector to date.

Looking to the future, a recent report by PGgRc outlines 5 objectives which will build on knowledge and research tools developed by PGgRc over the last 10 years. These objectives are to: breed low-CH_4 emitting ruminants; identify low-greenhouse-gas feeds, develop a vaccine to reduce ruminant CH_4 emissions, identify inhibitors that reduce ruminant CH_4 emissions and to extend and enable technologies that can be readily adopted by farmers (Aspin et al., 2014).

Other environmental issues

The foundation of the New Zealand farming industry is fertile land, clean water and fresh air and a number of programmes are in place in New Zealand aimed at supporting environmental sustainability.

A significant environmental challenge is maintaining soil fertility while limiting nutrient loss to waterways. A recent high-profile project, funded by industry, farmers and government was 'The Wise Use of Nitrogen Fertiliser' project. This was a 4 year project aimed at promoting the sound use of nitrogen fertilisers in a range of hill farming situations, in order to encourage practices that enhance long-term profitability while minimising any detrimental effects to the environment.

The National Policy Statement for Freshwater Management (2011) requires communities to establish objectives that maintain or improve the quality of all freshwater in a region, with a planning framework to manage resources to achieve to the objectives. National bottom lines have been set for key water quality objectives.

The New Zealand economy is dependent on the environment to support activities such as agriculture. To sustain the environment, a range of policy initiatives are currently being implemented in the beef and sheep sector to ensure the industry remains economically profitable and environmentally sustainable in future years.

Antibiotics

In New Zealand, antibiotics are used sparingly in animals for therapeutic reasons only. Any treatment with antibiotics is recorded and statutory declarations are made. Animals treated with antibiotics are required to be withheld from the market for a specified period of time.

Hormonal growth promotants

Hormonal growth promotants are only used in a very small number of livestock (less than 1%) and are only provided under veterinary supervision. Their use in New Zealand is very tightly controlled and any animals which have received such hormones must be tagged and included in a central government database.

There is no evidence of any adverse effect on human health through consumption of meat produced from animals given hormonal growth promotants. Any beef or lamb

products displaying the Quality Mark have come from animals not treated with hormonal growth promotants.

Risk management

The Animal Products Act 1999 (APA) is legislation that requires all animal products traded and used in New Zealand be fit for intended purpose and this is achieved through risk management programmes, which involve identifying and managing hazards and other risks (NZFSA, 2010). Individual plants must operate a risk management programme that is independently audited by the Ministry for Primary Industries (MPI).

Risk management programmes must comply with the required industry standards. Plants may also operate ISO (International Organisation for Standardisation) standards that incorporate HACCP (Hazard Analysis and Critical Control Points).

If a plant is to supply the overseas market, then the appropriate standards for the destination country must be met. For example, meat exported to the USA must meet United States Department of Agriculture market access standards, and meat being exported to the European Union (EU) must meet EU standards.

New Zealand Beef and Lamb Quality Mark

The New Zealand Beef and Lamb Quality Mark was introduced in 1997 to ensure consistent quality of New Zealand beef and lamb. The quality mark is a black, red and gold rosette and it provides assurance that the highest standards have been met for leanness, tenderness and food safety.

Meat must be trimmed to a maximum of 5mm external fat along with the removal of internal fat where practical. Often cuts are trimmed completely and have no visible fat at all. To be eligible for the Quality Mark, mince must contain less than 10% fat.

A significant amount of New Zealand beef and lamb (ie cuts containing less than 4% saturated fat with a maximum 5mm fat trim) also qualifies for the Heart Foundation's Two Ticks, being recognised as core foods as part of a healthy diet.

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Sent by: Louise McIntyre/MOH
19/03/2014 01:24 p.m.

To: "Jenny Reid (Jenny)" <Jenny.Reid@mpi.govt.nz>,
cc:
bcc:

Subject: Re: Opportunity to update you/your team members of Eating and Activity Guidelines (replacing F&N G/lines)

Will do - see you at 1pm

Louise McIntyre
Advisor - (Nutrition)
Nutrition & Physical Activity Policy
Public Health
Clinical Leadership
Protection & Regulation
Ministry of Health
DDI: 04 816 3382
Fax: 04 816 2191

http://www.health.govt.nz
mailto:Louise_McIntyre@moh.govt.nz

From: "Jenny Reid (Jenny)" <Jenny.Reid@mpi.govt.nz>
To: "Louise_McIntyre@moh.govt.nz" <Louise_McIntyre@moh.govt.nz>,
Cc: "Elizabeth Aitken(MOH)" <Elizabeth_Aitken@moh.govt.nz>, "Martin_Dutton@moh.govt.nz" <Martin_Dutton@moh.govt.nz>, Sally Johnston <Sally.Johnston@mpi.govt.nz>, Julia Edmonds <Julia.Edmonds@mpi.govt.nz>, Michelle Gibbs <Michelle.Gibbs@mpi.govt.nz>, Jenny Miller <Jenny.Miller@mpi.govt.nz>, Francesca Crowe <francesca.crowe@gmail.com>, Clare Chandler <Clare.Chandler@mpi.govt.nz>
Date: 19/03/2014 12:42 p.m.
Subject: Re: Opportunity to update you/your team members of Eating and Activity Guidelines (replacing F&N G/lines)

Sounds great. Come to us. Cheers Jenny

Sent from my iPhone

On 19/03/2014, at 10:41 am, "Louise_McIntyre@moh.govt.nz" <Louise_McIntyre@moh.govt.nz> wrote:

> Hi Jenny
>
> Lets go for 1pm this Friday and and if Elizabeth is needed elsewhere our colleague Martin can attend in her place. Shall we come to you or would you prefer to come here?
>
> Cheers
>
> Louise McIntyre
> Advisor - (Nutrition)
> Nutrition & Physical Activity Policy

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From: "Jenny Reid (Jenny)" <Jenny.Reid@mpi.govt.nz>
To: "Louise_McIntyre@moh.govt.nz" <Louise_McIntyre@moh.govt.nz>,
Cc: "Elizabeth Aitken(MOH)" <Elizabeth_Aitken@moh.govt.nz>
Date: 18/03 2014 10:12 p.m.
Subject: RE: Opportunity to update you/your team members of Eating and Activity Guidelines (replacing F&N G/lines)

Thanks Louise Thursday and Friday mornings are looking crazy - I could do Friday at 1pm but realise that may not work for Elisabeth?

From: Louise_McIntyre@moh.govt.nz [mailto:Louise_McIntyre@moh.govt.nz]
Sent: Tuesday, 18 March 2014 2:04 p m.
To: Jenny Reid (Jenny)
Cc: Elizabeth Aitken(MOH)
Subject: RE: Opportunity to update you/your team members of Eating and Activity Guidelines (replacing F&N G/lines)

Hi Jenny

Elizabeth has asked me to liaise with you re finding a good time to meet this week in relation to the Eating and Activity Guidelines.

Elizabeth and I are happy to come to you or conversely you are more than welcome to come to us - whichever you prefer. To get the ball rolling re times, two options that work for us are: Thurs (20th) 11-12 or Fri (21st) 10-11am - do either of these work for you? If not plse suggest some alternative dates. Elizabeth has requested we avoid Friday afternoon though.

As already identified, the focus of the meeting is to update you, and interested others, on the plans we have for the current Food and Nutrition Guidelines. These plans are based on the comprehensive independent evaluation of the Guidelines done during 2011. We are also interested in talking to you about the place of food safety in the Guidelines and perhaps ways we can work together to update the food safety specific info we have.

Happy to discuss further by phone if useful.
Look forward to hearing from you.

Cheers

Louise McIntyre
Advisor - (Nutrition)
Nutrition & Physical Activity Policy

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http://www.health.govt.nz
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From: "Jenny Reid (Jenny)" <Jenny.Reid@mpi.govt.nz>
To: "Elizabeth Aitken(MOH)" <Elizabeth_Aitken@moh.govt.nz>,
Cc: "Louise_McIntyre@moh.govt.nz" <Louise_McIntyre@moh.govt.nz>, "Martin_Dutton@moh.govt.nz" <Martin_Dutton@moh.govt.nz>, Sally Johnston <Sally.Johnston@mpi.govt.nz>, "Craig Thornley (Craig)" <Craig.Thornley@mpi.govt.nz>
Date: 17/03/2014 09:40 p.m.
Subject: RE: Opportunity to update you/your team members of Eating and Activity Guidelines (replacing F&N G/lines)

Hi elizabeth I think a meeting with a general update on work would be good – I know we wont be able to get everyone there so maybe we should pick a couple of dates. Im away all next week so we could try and meet with a smaller group this week if you want to talk guidelines sooner??

Cheers Jenny

From: elizabeth_aitken@moh.govt.nz [mailto:elizabeth_aitken@moh.govt.nz]
Sent: Thursday, 13 March 2014 4:39 p.m.
To: Jenny Reid (Jenny)
Cc: Louise_McIntyre@moh.govt.nz; Martin_Dutton@moh.govt.nz
Subject: Opportunity to update you/your team members of Eating and Activity Guidelines (replacing F&N G/lines)

Hi Jenny,
We would like the opportunity to provide you and members of your team with where we are at with revising the adult G/lines statements for eating and activity/the process to date and going forward, including discussing how we can engage with MPI on the food safety statement. We would like to do this sooner rather than later so I am wondering if you can let us know who should be involved from MPI and when could this happen ? We could either do it as a one off or possibly a teams get together to provide an update on each others work esp that of mutual interest, if that is preferred. Good to hear what you think.
Regards
Elizabeth
Elizabeth Aitken
Team Leader & Senior Advisor (Nutrition)
Nutrition and Physical Activity Team
Clinical Leadership, Protection and Regulation Business Unit
Ministry of Health
DDI: 04 816 4335

Sent by: Louise McIntyre/MOH
31/03/2014 03:48 p.m.
To: "Jenny Reid (Jenny)" <Jenny.Reid@mpi.govt.nz>, "Sally Johnston" <Sally.Johnston@mpi.govt.nz>, Craig Thornley (Craig) <Craig.Thornley@mpi.govt.nz>,
cc: Elizabeth Aitken/MOH@MOH, Martin Dutton/MOH@MOH,
bcc:

Subject: RE: Eating and Activity Guidelines

Hi Jenny, Sally and Craig

Plse find below the email we sent out last Friday as part of our limited stakeholder consultation. Im sending it through as promised to give you with a little more context around the project and our request for feedback from you. As mentioned in a previous email, your feedback regarding the food safety statement is likely to be more substantial expecially in light of our discussion regarding an appropriate evidence base and any other info to include as content.

Im happy to meet again to discuss the food safety statement in more detail or provide you electronically or verbally with anything more specific if thats of any help to you.

Regards

Louise McIntyre

Dear Colleagues

Feedback on the Eating and Activity Guidelines statements

Later this year, the Ministry intends to publish a concise document based on revised key healthy eating and physical activity messages (guidelines statements) for adult New Zealanders. **As part of the next stage of revision, the Ministry invites your organisation to provide feedback on the draft guideline statements.** These statements will also be included in the Ministry's health education resources written for the public.

Background

The concise document will be for health practitioners and others who provide advice on nutrition and physical activity to the general public. The revised statements which the document is based on have been updated to i) bring them in line with the current published international graded evidence base and ii) consider health literacy and communication perspectives. The first part of the revision involved seeking feedback on the current statements from an external technical advisory group comprising of academics and experts from other non-government agencies, and the second part involved revision following feedback from communications specialists. The resulting statements (attached) are currently undergoing focus group testing with members of the public, with emphasis on their meaning and relevance to key population groups including Māori, Pacific, South Asian and European/other.

We are aware that considering different factors ie evidence base, health literacy and communication will require careful balancing of word choice and phrasing. To retain integrity the statements ultimately need to accurately reflect the evidence base. Further work can be done by the Ministry to develop consumer friendly key messages and by the sector to make the statements understandable.

As well as the key messages (guidelines statements) the concise document (and related webpages) will include examples of foods and activities, as well as advice and links to other information sources for the practical application of the statements will be included.

What we need from your organisation

We are interested in your feedback on both draft guidelines statements attached below. The first document below provides the basic eating and activity messages. The second document contains an extended version of the activity statements which includes the 'why' and some ideas on 'how'. If positively received we may develop a similar, extended version for the eating statements.

As mentioned, these statements are written for the public and will also be used by health practitioners advising the public. We are interested in any feedback you have on content, word choice, order of statements, and the messages, for these statements as health education/promotion tools. Any other feedback you think may be useful would also be appreciated.

Please note these two documents are draft and not for further dissemination.

Basic guidelines statements 26 March 2014.doc

Long activity statements 26 Mar 2014.doc

When do we need your feedback by?

The Ministry would be grateful if you could provide feedback including any alternate wording or examples of any points you make to maximise our understanding of them by midday Tues 22 April.

How do we want feedback?

Please email to. If you wish to discuss this consultation further you can contact me by email or phone on the details below.

Yours sincerely

----- Document: RE: Eating and Activity Guidelines, forwarded by Louise McIntyre on 31/03/2014 03:38 pm -----

Sent By: Louise McIntyre/MOH on 21/03/2014 3:34:58 p.m.
To: "Jenny Reid (Jenny)" <Jenny.Reid@mpi.govt.nz>
Copy To: "Craig Thornley (Craig)" <Craig.Thornley@mpi.govt.nz>, "Elizabeth Aitken(MOH)" <Elizabeth_Aitken@moh.govt.nz>, "Martin_Dutton@moh.govt.nz" <Martin_Dutton@moh.govt nz>, "Sally Johnston" <Sally.Johnston@mpi.govt.nz>
Subject: RE: Eating and Activity Guidelines

Great - Will do.

Thanks

Louise McIntyre
Advisor - (Nutrition)
Nutrition & Physical Activity Policy
Public Health
Clinical Leadership

Protection & Regulation
Ministry of Health
DDI: 04 816 3382
Fax: 04 816 2191

http://www.health.govt.nz
mailto:Louise_McIntyre@moh.govt.nz

From: "Jenny Reid (Jenny)" <Jenny.Reid@mpi.govt.nz>
To: "Louise_McIntyre@moh.govt.nz" <Louise_McIntyre@moh.govt.nz>, "Sally Johnston" <Sally.Johnston@mpi.govt.nz>, "Craig Thornley (Craig)" <Craig.Thornley@mpi.govt.nz>,
Cc: "Elizabeth Aitken(MOH)" <Elizabeth_Aitken@moh.govt.nz>, "Martin_Dutton@moh.govt.nz" <Martin_Dutton@moh.govt.nz>
Date: 21/03/2014 02:56 p.m.
Subject: RE: Eat ng and Activity Guidelines

That's fine Louise – but do feel free to contact Sally and Craig as well, as between them they will be critical in the overall input and management of our feedback. Thanks for meeting with us and would appreciate receiving the context information next week – send to both sally and Craig –

Cheers and thanks Jenny

From: Louise_McIntyre@moh.govt.nz [mailto:Louise_McIntyre@moh.govt.nz]
Sent: Friday, 21 March 2014 2:52 p.m.
To: Jenny Reid (Jenny)
Cc: Elizabeth Aitken(MOH); Martin_Dutton@moh.govt.nz
Subject: Eating and Activity Guidelines

Hi

Thanks again for meeting with me to discuss the new Guidelines. Im conscious I need to provide some more context around the statements I have asked you and your team to consider, along with a timeframe for the feedback. We are
currently finalising the information that will accompany the draft statements to be emailed to key stakeholders next Friday. Once finalised, hopefully early-mid next week, I will send it to you. . We are asking for the key stakeholder feedback to the draft statements by 18 April.

Obviously your input on the food safety statement will be more significant than feedback on the other statements. We can talk more about what, how and when that particular feedback/input is provided in the next week or two (I realise you aren't around next week).

Happy for further conversations and meetings with you and your team as would be useful.

Thanks again

Louise McIntyre
Advisor - (Nutrition)
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Draft Eating and Activity Guidelines Statements 2014

Draft Eating and Activity Guidelines Statements	
Draft eating guideline statements	**Draft activity guideline statements**
1. To be a healthy weight, balance your intake of food and drinks with your activity levels. 2. Enjoy a variety of nutritious foods every day including: • plenty of different coloured vegetables and fruit • a range of grains and cereals that are naturally high in fibre • some low fat milk products and/or calcium-fortified milk alternatives • some legumes*, nuts, seeds, fish, eggs, lean poultry or lean red meat. *Legumes include cooked dried beans (eg baked beans), split peas, lentils and chickpeas. 3. Choose and prepare foods and drinks: • with minimal fat, especially saturated fat; if you choose to add fat use plant based oils and spreads • low in salt (sodium); if using salt, choose iodised salt • with little or no added sugar 4. Make water your first choice for drinks. 5. Buy, prepare, cook and store food to ensure food safety. 6. If you drink alcohol, keep your intake low. Don't drink if you are pregnant or planning to become pregnant.	1. Sit less, move more! Reduce sedentary behaviour and break up long periods of sitting. 2. Do at least 150 minutes (2 ½ hours) of moderate-intensity or 75 minutes (1¼ hours) of vigorous-intensity physical activity spread throughout the week. 3. For weight management and extra health benefits, aim to do at least 300 minutes (5 hours) of moderate- or 150 minutes (2 ½ hours) of vigorous-intensity physical activity spread throughout the week. 4. Include some muscle- and bone-strengthening activities on at least two days per week. 5. If you currently do no physical activity, start by doing some activity, and then build up to the recommended amount.

Draft Activity Guidelines Statements 2014 – including 'why' and 'how'

1. **Reduce sedentary behaviour and break up long periods of sitting (sit less, move more).**
   - Sitting less can help you live healthier and longer.
     - *Stand up and move regularly throughout the day, at least every hour.*
     - *If you are watching television, get up during the ad breaks.*
     - *If you sit a lot at work, get into the habit of getting up and moving at least every hour.*
     - *See standing and moving as an opportunity, not an inconvenience.*

2. **Do at least 150 minutes (2 ½ hours) of moderate-intensity or 75 minutes (1 ¼ hour) of vigorous-intensity physical activity spread throughout the week.**
   - Moderate- and vigorous-intensity activities are great for the heart, lungs, and overall fitness and wel being. Examples of th se activities can be found in Table X
     - *Moderate-intensity activities cause a slight but noticeable increase in breathing and heart rate.*
     - *Vigorous-intensity activities significantly increase breathing and heart rate.*
     - *You can achieve this by doing 30 minutes of moderate-intensity, or 15 minutes of vigorous-intensity physical activity on five days per week.*
     - *If you have been physically inactive for some time, are just starting out, or have certain health conditions you may wish to consult a health practitioner or physical activity specialist to ensure your safety before you start being physically active.*

3. **Aim to do at least 300 minutes (5 hours) of moderate-intensity or 150 minutes (2 ½ hours) of vigorous-intensity of physical activity for extra health benefits and to manage your weight.**
   - o If you already meet the guidelines, increase the amount of physical activity you do for extra health benefits.
     - *Double the recommended amount of time being active to reduce weight.*
     - *Increase the intensity of your activity for other health benefits including*

Comment [M1]: To be added in.

4. **Include some muscle- and bone-strengthening activities on at least two days per week.**
   - Muscle and bone strengthening activities are important for keeping your body strong lifting and carrying, and reducing the risk of falling or injury.
     - *Strengthen your muscles and bones with resistance activities such as walking up hills or stairs, yoga, Pilates, swimming, aerobics, heavy gardening or weight lifting.*

5. **If you currently do no physical activity, start by doing some activity, and then build up to the recommended amount.**
   - Doing something is better than doing nothing.
     - *Walk or cycle to work, the marae or church, play actively with the children, meet friends for a walk, do active jobs around the house.*
     - *Build the activities into your daily routine that you are likely to stick to!*
     - *Consider joining a gym or sports club.*
     - *Set yourself goals to achieve.*
   - Being physically active with others is good for your overall wellbeing and can motivate you to stay active.
     - *Being physically active with whānau is good for the hinengāro (mental and emotional wellbeing) of tangata.*
     - *Do a variety of activities with whānau and friends that you enjoy and want to keep doing.*

Sent by: Louise McIntyre/MOH
08/12/2014 05:29 p.m.

To: "Roger Cook (Roger Cook)" <Roger.Cook@mpi.govt.nz>,
cc: Julia.Edmonds@mpi.govt.nz,
bcc:

Subject: RE: Food safety statement and the Eating and Activity Guidelines

Hi Roger

How did you get on with the food safety section? I think you had passed it up the line for approval last time we were emailing.

Regards

Louise McIntyre
Advisor - Nutrition and Physical ActivityTeam
Public Health Group
Clinical Leadership, Protection and Regulation
Ministry of Health
DDI: 04 816 3382
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----- Document: RE: Food safety statement and the Eating and Activity Guidelines, forwarded by Louise McIntyre on 08/12/2014 05:27 pm -----

Sent By: Louise McIntyre/MOH on 9/10/2014 2:38:29 p.m.
To: "Roger Cook (Roger Cook)" <Roger.Cook@mpi.govt.nz>
Copy To: Julia Edmonds <Julia.Edmonds@mpi.govt.nz>
Subject: RE: Food safety statement and the Eating and Activity Guidelines

Hi Roger

Thank you so much. Look forward to reading it.

Regards

Louise McIntyre
Advisor - Nutrition and Physical ActivityTeam
Public Health Group
Clinical Leadership, Protection and Regulation
Ministry of Health
DDI: 04 816 3382
Fax: 04 816 2191

http://www.moh.govt.nz
mailto:Louise_McIntyre@moh.govt.nz

From: "Roger Cook (Roger Cook)" <Roger.Cook@mpi.govt.nz>
To: "Louise_McIntyre@moh.govt.nz" <Louise_McIntyre@moh.govt.nz>,
Cc: Julia Edmonds <Julia.Edmonds@mpi.govt.nz>
Date: 08/10/2014 09:04 a.m.
Subject: RE: Food safety statement and the Eating and Activity Guidelines

Louise

Have drafted comments and sent upwards for approval.

Roger

From: Louise_McIntyre@moh.govt.nz [mailto:Louise_McIntyre@moh.govt.nz]
Sent: Thursday, 2 October 2014 12:26 p.m.
To: Roger Cook (Roger Cook)
Cc: Julia Edmonds
Subject: Re: Food safety statement and the Eating and Activity Guidelines

Hi Roger

Wondering how you are getting on reviewing the food safety pages for our new Guidelines document.

Cheers
Louise McIntyre
Advisor - Nutrition and Physical ActivityTeam
Public Health Group
Clinical Leadership, Protection and Regulation
Ministry of Health
DDI: 04 816 3382
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----- Document: Re: Food safety statement and the Eating and Activity Guidelines, forwarded by Louise McIntyre on 02/10/2014 12:25 pm -----

Sent By: Louise McIntyre/MOH on 20/09/2014 11:57:10 a.m.
To: "Roger Cook (Roger Cook)" <Roger.Cook@mpi.govt.nz>
Copy To: Julia Edmonds <Julia.Edmonds@mpi.govt.nz>
Subject: Re: Food safety statement and the Eating and Activity Guidelines

Hi Roger

Thx for the email. Yes I thought you were probably snowed under and I have only just heard about Craig leaving so that will impact for sure. Im more than happy for a pragmatic approach to the evidence base issue as I understand there is little capacity at present for anything else - hence my suggestions of the Aussies evidence base but keen to get your view on whether it does work. The core paper and statements will be reviewed overtime so we can change the evidence base used to underpin the food safety statement in the future.

Thanks for looking at the 2 page section and look forward to your feedback. Happy to meet with you for that feedback if that is easier.

Regards

Louise McIntyre

Advisor - Nutrition and Physical ActivityTeam
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http://www.moh.govt.nz
mailto:Louise_McIntyre@moh.govt.nz

From: "Roger Cook (Roger Cook)" <Roger.Cook@mpi.govt.nz>
To: "Louise_McIntyre@moh.govt.nz" <Louise_McIntyre@moh.govt.nz>,
Cc: Julia Edmonds <Julia.Edmonds@mpi.govt.nz>
Date: 19/09/2014 06:18 p.m.
Subject: Re: Food safety statement and the Eating and Activity Guidelines

Sorry Louise. Totally buried in priority work, and now Craig had departed. I am running yacht race regattas over the next three weekends so will attempt to fit in ASAP during the week. Roger

Sent from Roger's iPhone

Sent by: Louise McIntyre/MOH
19/09/2014 06:11 p.m.
To: "Roger Cook (Roger Cook)" <Roger.Cook@mpi.govt.nz>,
cc: Julia.Edmonds@mpi.govt.nz,
bcc:

Subject: Food safety statement and the Eating and Activity Guidelines

Hi Roger

I havent heard from you re the previous emails so Im just powering ahead hoping all is well?! Please find attached the draft food safety info that will go into our Eating and Activity Guidelines core guidelines paper. We intend the core paper to be around 30-50 pages long and written in plain English using less technical wording. We need the document to be accessible to a wide range of practitioners including those with little or no nutrition or science background.

Draft Eating statement 5.docx

The purpose of core paper is to:

1 To bring together the key eating and activity guideline statements (key messages) for all New Zealanders into one document. *The first core paper will include statements relevant to all. It is planned that over time, ie in subsequent editions, statements relevant to specific population groups will also be included. For example, infants, toddlers, children, young people, pregnant and breastfeeding women (and young people).*

2 Provide a snapshot of the important and relevant background information related to statements (key messages).

3 Show the evidence that underpins each specific statement.

Target audience for paper:

- Health practitioners and those involved in promoting healthy eating and/or physical activity to New Zealanders - including health promoters and community health workers

Other interested groups including:

- Educators; local, regional and national policy advisors;regulators; the food industry; the fitness industry; researchers; and professional bodies

Re the evidence base to underpin the food safety statement - do you have a view on the one used for the Australian Dietary Guidelines?

http://www.eatforhealth.gov.au/sites/default/files/files/the_guidelines/n55d_dietary_guidelines_evidence_report.pdf

The food safety info can be found on page 1062 onwards - as our document is at present only for adults we wont be including the info related to infants and pregnant women.

Happy to take any other more appropriate solutions. Im aware you mentioned the ideal would be for MPI to pull together the evidence base for its own advice, but from a pragmatic stance would this Australia info work in the short term?

Regards

Louise McIntyre
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----- Document: Food safety statement and the Eating and Activity Guidelines, forwarded by Louise McIntyre on 19/09/2014 05:35 pm -----

Sent By: Louise McIntyre/MOH on 16/09/2014 1:33:42 p.m.
To: "Roger Cook (Roger Cook)" <Roger.Cook@mpi.govt.nz>
Copy To: Julia.Edmonds@mpi.govt.nz
Subject: Food safety statement and the Eating and Activity Guidelines

Hi Roger

As discussed previously Im currently writing our core document containing the updated eating and activity statements for adult New Zealanders (See previous email send to you and Julia in July). This core document includes a specific food safety statement which currently reads:

"Buy, prepare, cook and store food to ensure it is safe to eat."

The food safety section to be 2 x A4 pages which will include the following three sections: 'Background', 'Evidence base' and 'Practical ideas. Im planning to use information from MPI website and the 3 C's work as previously linked me by Sally Johnson.

At our meeting in June we discussed whether there was an evidence base available that underpins the basic and general food safety information MPI provides to the public. You thought ESR's YOPI booklet could provide some of this info. Could you send me a copy of this document?

My other query is are you aware of any recent general info on food safety in New Zealand? Specifically Im looking for anything with incidence rates of food poisoning, anything really to give me info on why food safety is an important consideration for New Zealanders. I see this going into the 'Background' section.

As documented below we also discussed MPI reviewing the 2 page food safety section to make sure you were happy with its info and messages. Is this still a possibility? Im hoping to send you a draft of the 2 pages by the end of this week and could give you until the 30 September to review. The document is for health practitioners but we are aiming to keep it as simple and straightforward as possible so hopefully the reviewing the 2 pages would not be too onerous. Having said that Im aware of the very busy workload you all have.

Happy to discuss further if useful.

Regards

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----- Document: Food safety statement and the Eating and Activity Guidelines, forwarded by Louise McIntyre on 16/09/2014 01:03 pm -----

Sent By: Louise McIntyre/MOH on 7/07/2014 5:23:41 p.m.
To: Julia.Edmonds@mpi.govt.nz, "Roger Cook (Roger Cook)" <Roger.Cook@mpi.govt.nz>
Copy To:
Subject: Food safety statement and the Eating and Activity Guidelines

Hi there

Firstly apologies for not getting back to you sooner following our meeting on Friday 20 June. I had promised to summarise our discussion and the plan we discussed.

Key points from the discussion:

- The Ministry is currently reviewing its food and nutrition guidelines statements for adults as part of the transition from the current Food and Nutrition Guidelines Series (FNGS) to the new Eating and Activity Guidelines Series (EAGS). One of the first steps in this transition is to produce a core paper for the sector which will be based on the eating (nutrition) and activity statements. A key feature of the transition from FNGS to EAGS is to have a stronger (systematic and graded if possible) and more transparent evidence base to underpin the statements. The core paper will include a summary/description or link to the evidence base.
- We are keen for MPI's input on reviewing the food safety related statement. This statement is currently: "Buy, prepare, cook and store food in ways to ensure food safety." Feedback from Sally Johnson in May 2014 suggested incorporating the 3 'C' into the statement as these already are known and promoted. Sally also linked us to appropriate consumer focused MPI webpages that could be used for how-to information.
- MOH is also interested to have the evidence base used by MPI that underpins the general food safety advice for the public. A brief summary will be put into the core paper with any links or key documents placed on the EAGS webpage. As per our discussion, the evidence is currently not all together in one document and would need to be collated. This was thought possible but would take some time (ie would not be ready by planned date for finalising the core paper content - 31 July 2014).
- There is a documented evidence base for the YOPI group which is contained in an ESR report. This could be used to underpin the food safety statements.
-

Plan

1 MPI to link me to ESR YOPI booklet evidence base.

2 MPI to consider what could be used as a temporary, quick and easy evidence base for food safety statement. ?

3 MPI to speak with Team re possiblity of collating the evidence base that would underpin the food safety statement in the longer term.

4 MOH will review current draft food safety statement and consider the 3C's idea and the supporting information from MPI website. Once re-work will send to MPI for review.

Let me know if this is how you remember things and if you are still happy with the plan. Not surprisingly we are looking at pushing out the 31 July date so will let you know what new date we are aiming for to finalise content.

Regards

-

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Eating statement 5

Buy, prepare, cook and store food to ensure food safety.

Evidence base for statement:

? Use Australian Dietary Guidelines evidence base

Background:

Micro-organisms or bugs that cause illness are called pathogens. These pathogens can be bacteria, fungi, parasites or viruses. Foodborne illness, also sometimes known as 'food poisoning' is any illness that results from eating foods contaminated with pathogens. It is estimated that around 200,000 New Zealanders suffer a food borne illness every year. The most common health effects are gastrointestinal such as abdominal pain, vomiting and/or diarrhoea with varying degrees of severity. In a small number of cases longer term illness or even death can result.

A number of foodborne illnesses are covered by New Zealand's notifiable disease regulations. As a result information on rates of these illnesses is available from the Institute of Environment Science and Research Limited (ESR) Public Health Surveillance website: https://surv.esr.cri.nz/ Pathogens that most commonly cause foodborne outbreaks (more than two cases linked to a common source) include norovirus, *Camplyobactacter spp*, *Giardia spp*, *Clostridium perfringes* and *Salmonella spp*.

Foodborne illness is mostly preventable. The suspected key contributing factors from 2013's data were 'time and temperature abuses' such as incorrect storage prior to preparation, not enough reheating of previously cooked food and undercooking. Food became contaminated with bugs by coming in contact with infected or raw food or via an infected food handler. Consumption of 'unsafe' food such as raw food is also thought to have contributed to outbreaks and cases.

The term 'food safety' generally describes handling, preparation and storage of food that prevents foodborne illness. Using these techniques as part of everyday food handling is the best way to keep food safe and avoid foodborne illness.

Practical considerations:

Buy, prepare, cook and store food to ensure food safety.

- Thinking about food safety starts before the food and drinks are bought:
    - *Don't buy food past its use-by date (check the label).*
    - *Avoid any packaged foods where the packaging is damaged eg dented tins, ripped packaging, etc.*
    - *Go for undamaged and unripe or just-ripe fresh vegetables and fruit*

- The Ministry for Primary Industries (MPI) is responsible for food safety in New Zealand. Their key tips for consumers on food safety include the 3Cs (Clean, Cook and Chill):

Clean

- wash your hands thoroughly with soap and warm water and dry them with a clean dry towel or paper towel
- wash and dry your hands before and after handling food
- wash and dry your hands every time after you touch raw meat or chicken
- before you start handling food, make sure all tools and all surfaces which you put food on are clean

Cook

- defrost frozen foods thoroughly before cooking
- pre-cook chicken, meat patties and sausages before barbecuing. Minced meat and sausages should be cooked right through, and pork and poultry juices should run clear. Use a meat thermometer to check temperatures
- use one set of utensils for raw meat and chicken and another set for cooked food
- when cooking or eating outdoors ensure that all food remains covered and cool until ready to cook or eat
- reheat leftovers until steaming hot throughout and do not reheat more than once

Chill

- ensure your fridge is operating at a temperature of between 2 and 4 degrees celsius
- keep all perishable foods cold until you are ready to use them
- use an icepack or chilly-bin to keep food cold outdoors
- ensure raw meat and chicken is properly wrapped to stop drips; also keep raw meat and chicken away from other foods and below ready-to-eat foods in the refrigerator
- cover and refrigerate food as soon as possible after cooking. Throw out perishable foods that you have left at room temperature for more than two hours.

- MPI has a user friendly website with lots of useful information on ways to ensure food is 'safe': http://www.foodsmart.govt.nz/food-safety/ The information available looks at food safety in many different situations, for example:
  - barbeque food safety;
  - hunting, collecting, fishing and homekill;
  - food for fundraising and promotions;
  - food safety tips for event organisers.

- MPI also has specific information for specific groups such as pregnant women, babies, older people and others at higher risk of infection http://www.foodsmart.govt.nz/
- The MPI website has useful information and resources that may be of interest to Māori, Pacific and other cultures: http://www.foodsmart.govt.nz/information-for/maori-pacific-other-cultures/ Topics such as *Food safety practices in preparing and cooking a hangi*; *Umu Pasifika – Food Safety for Pacific Peoples*; *Safe sushi*, among others.
- The information can either be downloaded directly from the MPI website or hard copies of resources ordered via the site.

Sent by: Louise McIntyre/MOH
09/10/2014 02:45 p.m.

To: Julia Edmonds <Julia.Edmonds@mpi.govt.nz>,
cc: "Roger Cook (Roger Cook)" <Roger.Cook@mpi.govt.nz>,
bcc:

Subject: Re: Food Safety During Pregnancy – Evidence Based Source Document

Thanks very much Julia. Was this from our joint discussion a few months back re the evidence base for the basic food safety recommendations? I see this one's focus is during pregnancy. Do you or Roger think any of the evidence used could be used for non-pregnant adult?

regards

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From: Julia Edmonds <Julia.Edmonds@mpi.govt.nz>
To: "Louise_McIntyre@moh.govt.nz" <Louise_McIntyre@moh.govt.nz>,
Cc: "Roger Cook (Roger Cook)" <Roger.Cook@mpi.govt.nz>
Date: 08/10/2014 08:14 a.m.
Subject: Food Safety During Pregnancy – Evidence Based Source Document

Hi Louise,

We have managed to locate the ESR document we mentioned sometime ago – 'Food Safety During Pregnancy – Evidence Based Source Document'.

Thanks
Julia

Julia Edmonds | Acting Manager, Food Science
Biosecurity Science, Food Science & Risk Assessment Directorate | Regulation & Assurance Branch
Ministry for Primary Industries - Manatū Ahu Matua | Pastoral House 25 The Terrace | PO Box 2526 | Wellington | New Zealand
Telephone: 64-4-8940286 | s 9(2)(a) | Web: www.mpi.govt.nz

[attachment "FW09089 Food Safety During Pregnancy CARC_08_02.pdf" deleted by Louise McIntyre/MOH]

Sent by: Julia.Edmonds@mpi.govt.nz
08/10/2014 08:14 a.m.
To: "Louise_McIntyre@moh.govt.nz" <Louise_McIntyre@moh.govt.nz>,
cc: "Roger Cook (Roger Cook)" <Roger.Cook@mpi.govt.nz>,
bcc:

Subject: Food Safety During Pregnancy – Evidence Based Source Document

Hi Louise,

We have managed to locate the ESR document we mentioned sometime ago – 'Food Safety During Pregnancy – Evidence Based Source Document'.

Thanks
Julia

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Telephone: 64-4-8940286 s 9(2)(a) | Web: www.mpi.govt.nz

FW09089 Food Safety During Pregnancy CARC_08_02.pdf

FOOD SAFETY DURING PREGNANCY

EVIDENCE-BASED SOURCE DOCUMENT

Prepared for New Zealand Food Safety Authority
under project CARC/08/02 – Risk Communication
as part of overall contract for scientific services

by

Susan Gilbert
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April 2010

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Client Report
FW09089

FOOD SAFETY DURING PREGNANCY

EVIDENCE-BASED SOURCE DOCUMENT

April 2010

Stephen On
Food Safety Programme Leader

Maurice Wilson
Project Leader

Dr Andrew Hudson
Peer Reviewer

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CONTENTS

LIST OF TABLES

1 INTRODUCTION

Most countries in the developed world provide risk communication material in relation to food safety and pregnancy. A number of leaflets on this topic have been published in New Zealand over the years, provided by agencies such as the Ministry of Health. 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 leaflet "Food Safety in Pregnancy", updated August 2009, forms the central part of this document.

Following various queries regarding information contained in the leaflet, the NZFSA requested an evidence-based document to underpin future statements regarding food safety during pregnancy.

Lowered immunity mechanisms in pregnant women are discussed in order to place microbial food safety into context and each foodborne hazard of interest is introduced along with key information. The food groups listed in the NZFSA document are discussed in relation to the hazards of interest.

Extensive growth data for *Listeria monocytogenes* in foods from the 2003 USDA (FDA/FSIS) Quantitative Risk Assessment Document have been included in Appendix 1 as supplementary information.

2 IMMUNE SYSTEM OF MOTHER AND BABY DURING PREGNANCY

2.1 Down Regulation of Cellular Immune Function

A number of hormonal and immunological changes must take place in a woman's body in order to carry a baby successfully to full term. This means that a pregnant woman has a very different immunity status to that of a healthy non-pregnant woman of the same age. The most important change is the down-regulation of the cellular immune function and a substantial increase in progesterone production (Smith, 1999).

In genetic terms, a foetus is a semi-allogenic graft, i.e. up to one half of the foetus is "foreign" to the mother through the possession of paternal antigens. A number of theories have been proposed to explain why the foetus is not rejected by the mother during pregnancy.

2.1.1 Tolerance of the foetus

Three possibilities are suggested by Lederman, (1984):

- the foetus and its placental tissues are within an immunologic protected environment;
- cells of foetal origin are somehow resistant to immunologic effector mechanisms; or
- suppression of the maternal immune response that fails to reject the foetal allograft. Suppression is either through maternal mechanisms or foetal or placenta factors.

The first and second hypothesis have been largely discounted because the placenta is not an immunologically privileged site as foetal blood cells gain access to maternal circulation and maternal sera contain antibody to foetal major histocompatibility complex (MHC) antigens. Rather, evidence points to the third hypothesis of a diminished maternal immune response (Lederman, 1984).

2.1.2 Progesterone production

A pregnant female produces a number of steroid hormones at levels several times higher than in her non-pregnant counterparts. These hormones include estradiol, estriol and progesterone. Production of progesterone is absolutely required for the maintenance of pregnancy (Smith, 1999).

There is evidence that progesterone, progesterone-induced blocking factor, prostaglandin E_2 and early pregnancy factor are immunosuppressants preventing immunological rejection of the embryo, allowing it to develop and survive (Smith, 1999; Nahum *et al.*, 2003).

The consensus is that the foetus is tolerated in part by suppression of the maternal cell-mediated immune response. This is of course beneficial to the foetus but results in susceptibility of the mother to a variety of pathogens including foodborne ones.

2.2 Humoral immunity and cell-mediated immunity

Our bodies have two major immunity mechanisms:

- Humoral immune response (HIR), and
- Cell-mediated immunity (CMI)

HIR involves substances found in the humours or body fluids. The immune response is mediated by secreted antibodies and produced in the cells of the B lymphocyte lineage (B cell). Antigens on the surfaces of invading micro-organisms bind to secreted antibodies. The antibodies flag the pathogen for destruction (e.g. phagocytosis).

The CMI response is directed primarily at those micro-organisms that survive in phagocytes and viruses, fungi, protozoans, intracellular bacteria, and cancers which infect non-phagocytic cells. The principle functions of the response are the activation of T lymphocytes, activation of macrophages and natural killer cells and stimulation of cells to secrete cytokines. When pathogenic micro-organisms invade host cells, their subsequent intracellular location protects those micro-organisms from the HIR but not from the CMI response. However, when the CMI function is lowered, as in pregnancy, susceptibility to infection is raised (Smith, 1999).

A review of animal studies by Smith (1999) found that both mother and foetus were at risk of serious disease where the mother became infected. Pathogens were not cleared from the spleen or liver as effectively as in infected virgin mice. Pregnant mice also had a higher mortality rate. Pregnant mice were also susceptible to *Listeria* induced encephalitis. Effects on the pups included foetal resorption, abortion, stillbirth, abnormalities and heavy infections if the pups survived. Greater adverse effects were observed when infection took place mid-gestation as opposed to earlier or later infections during the pregnancy. Conversely in humans, listeriosis infections are most common in the third trimester, rare in the second trimester and very rare in the first trimester. Early gestation infection is associated with septic abortion while late listeriosis can result in premature delivery of a stillborn or septic baby. Where diagnosis is prompt and antibiotic treatment started, the outcome can be favourable for both.

Research on pregnant and non-pregnant rats and mice has led researchers to conclude that estrogen-induced decreases in immunity account for the suppression of cellular immunity in pregnant experimental animals.

2.3 Suppressed immunity in multiple gestations

Since the increase in the availability of assisted reproductive technologies (ART), multiple pregnancies have increased resulting in a steady increase in the number of live births from ART cycles in Australia and New Zealand since 1995 (Wang *et al.*, 2006). In Australia and New Zealand, approximately 16 percent of ART cycles are twin deliveries (Wang *et al.*, 2006). The percentage of multiple births peaked during 2002 which may reflect the then current practice of transferring one or two embryos per treatment cycle compared to three of four some years ago.

The question of whether multiple gestations increase the risk of perinatal listeriosis has been studied in California by Mascola *et al.,* (1994). Active surveillance and medical record reviews of 301 perinatal listeriosis cases were carried out over the period of January 1985 to December 1992. Twelve of the cases occurred in women with multiple gestations. The researchers found that the rate of listeriosis in pregnant women per 100,000 live births and foetal deaths was 19.8 for a single baby and 74.9 for multiple gestations. The risk of listeriosis was greatest in triplet gestation pregnancies (risk ratio 38.4, 95% CI 9.6 - 153.3) followed by twin gestations (risk ratio 3.2, CI 1.7 – 6.0). The age group ≥ 35 years old with multiple gestations was at highest risk of contracting listeriosis (risk ratio 13.6, CI 5.2 - 35.5). The authors concluded that pregnant women should routinely receive advice on avoiding listeriosis from their obstetricians but particular counselling should be given to women with multiple gestations because of the greater risk.

In New Zealand, specific information on multiple gestation is not collected in terms of listeriosis surveillance and numbers of cases are very small. The only information available from EpiSurv regarding perinatal listeriosis and multiple gestations is for neonatal twins diagnosed in November and December 1992, twins born in May 1999, twins born November 2003 and twins born in August 2008 (Esther Lim, ESR, personal communication, November 2008).

In New Zealand, the number of live births recorded for the 2007 calendar year was 64,040 (http://www.stats.govt.nz/) from a population of 4,228,300. The annual rate of multiple births is approximately 3% of total births.

3 MICROBIAL HAZARDS AND PREGNANCY

Pregnant women are no more susceptible to most foodborne hazards than the general healthy population (Smith, 1999). However, there are some pathogens for which the pregnant woman and her foetus are more susceptible or that produce a more severe illness.

Smith (1999), in a review of foodborne infections during pregnancy, identified Hepatitis E virus, *Coxiella burnetii*, *Listeria monocytogenes* and *Toxoplasma gondii* as the intracellular pathogens with a particular tendency to infect pregnant women and their foetus. *L. monocytogenes* and *T. gondii* were regarded as the most important of these pathogens during pregnancy. A summary of the effects of these and other foodborne pathogens on mother and foetus during pregnancy can be found in Appendix 2

Pikholz and Simmons (2004) reviewed New Zealand data and concluded that foodborne illness is likely to be an important cause of morbidity during pregnancy. Notification and hospitalisation data from 1996 to 1999 for the incidence of the main six potentially foodborne diseases amongst women aged 15 to 49 were obtained. These data were adjusted for under-reporting, the proportion of the total incidence attributable to food, and the proportion of New Zealand women who were pregnant. The results are presented in Table 1.

Table 1: Estimated morbidity cases per year due to foodborne illness in pregnant women in New Zealand

Cause of morbidity	Estimated mean no. of cases in a year (illness in pregnant women)	Estimated range (no. of cases)
Campylobacteriosis	715	602 - 938
Toxoplasmosis	82	65 - 102
Salmonellosis	58	41 - 75
Yersiniosis	18	15 - 20
Listeriosis	5	2 - 8
Shigellosis	3	2 - 4

(source Pikholz and Simmons, 2004)

There is no estimation of mortality in this study.

Of the main six potential foodborne illnesses reported above, listeriosis and toxoplasmosis are the most likely to result in foetal death or stillbirth in New Zealand These two diseases are therefore, the main focus of this document and are discussed in relation to each of the food groups.

Other foodborne pathogens which may affect pregnant women and their foetus or newborn are summarised and discussed in Appendix 2. *Salmonella* is cited by some authors as a pathogen of interest during pregnancy. Contamination of eggs by specific strains of this pathogen has also been shown to make a significant contribution to illness in the general population in several overseas countries (Lake *et al.*, 2004). For this reason, *Salmonella* contamination and foodborne illness are

considered in the section relating to eggs. Similarly, other pathogens with a strong relationship to a particular food group are also discussed in the relevant sections

Hepatitis E and *C. burnetii*, pathogens which Smith (1999) also regarded as of particular concern during pregnancy, will not be discussed further in this document. *C. burnetii* (the cause of Q fever in humans) is not present in New Zealand animals and while occasional human cases have been reported, investigations have indicated that they are likely to have originated overseas (MAF, 2009; ESR, 2004). Hepatitis E is also rarely reported in New Zealand. Although the virus is principally transmitted by the faecal-oral route and appears to have the potential for foodborne transmission, corroborating information for this route of infection is lacking (Fitzmaurice, 2004).

3.1 *Listeria monocytogenes*

Six species of *Listeria* bacteria have been recognised (ICMSF, 1996). *L. grayi* and *L. innocua* are considered non-pathogenic, while *L. seeligeri*, *L. ivanovii*, and *L. welshimeri* rarely cause human infection. *L. monocytogenes* is the most important species with respect to human health.

Two forms of disease caused by this organism are now recognised; a serious invasive disease and a non-invasive gastroenteritis. The invasive form of listeriosis disease has major or fatal consequences for the foetus and neonate (general septicaemia, meningitis and intra-uterine death).

3.1.1 Parameters for survival, growth and inactivation

3.1.1.2 Survival and Growth

The pathogen **s**urvives freezing very well. The optimum temperature for growth is 30 - 37°C with a range of -1.5 to 45°C. The two key food safety controls of refrigeration and salt are less relevant to *Listeria* spp. than to other foodborne pathogens. The pH optimum for growth is 7.0 with a range 4.4-9.4 (ICMSF, 1996). The pathogen grows optimally under microaerophilic conditions and can grow in food packaged under vacuum or nitrogen gas (AIFST, 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 lower a_w values. The organism can grow in NaCl concentrations up to 10%, with some laboratories reporting growth up to 12% NaCl (if the pH is sufficiently high) (AIFST, 2003)

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 of gastric acids by the use of antacids may also have a similar effect. Antacids were found to be a factor in an outbreak of *L. monocytogenes* 4b infections involving eight Boston Hospitals. More case patients took antacids or cimetidine compared with matched controls (Ho *et al.,* 1986).

3.1.1.2 Inactivation

Listeria monocytogenes is rapidly inactivated at temperatures above 70°C, depending on the properties of the food. The D value at 50°C can be in the order of hours, 5-10 minutes at 60°C and approximately 10 seconds at 70°C. 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. *Listeria* can remain viable for long periods in dry environments.

3.1.2 Transmission routes

According to a New Zealand expert elicitation process, 84.9% of invasive listeriosis cases are foodborne (Cressey and Lake, 2007). Most cases are sporadic. Alternative routes include infections acquired in hospital (nosocomial) and, very rarely, occupational exposure via for example, skin infections (Cain and McCann, 1986).

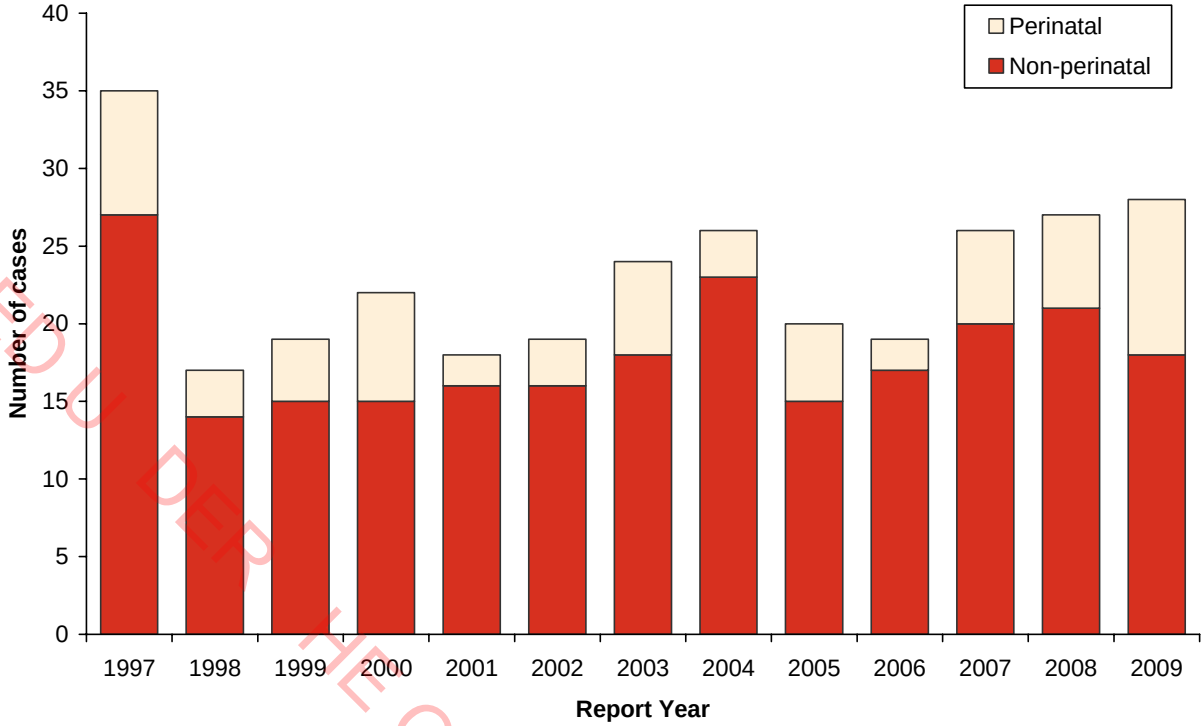
L. monocytogenes is carried asymptomatically in the gut of 2-6% of the population. The faeces from an infected person can contain high numbers of the organism ($\geq 10^4$/g). However, reports of person-to-person transmission are uncommon (other than mother to foetus).

The organism should be considered as potentially present in all raw foods and ingredients. It may also be present in cooked or RTE foods as a result of post-processing contamination. The risk posed is likely to be greatest in ready-to-eat cooked foods with long shelf lives on which *L. monocytogenes* can grow. It has been isolated from a wide variety of ready-to-eat and raw foods in NZ studies, although it is mostly present in low numbers (<10/g).

3.1.3 *Listeriosis* in New Zealand

Numbers of notified perinatal and non-perinatal cases occurring in New Zealand in the period 1997 to 2009 are shown in Figure 1 (Lim *et al.*, 2010). In 2009, 28 cases of listeriosis were notified, a rate of 0.6 per 100,000. Ten of the 28 cases were perinatal, the highest number of perinatal cases in a year since listeriosis became notifiable. The gestation period for all cases ranged from 20 to 40 weeks. Two cases of 20 and 31 weeks gestation died. One mother was of Māori ethnicity in the 15–19 years age group and the other was European and in the 20–29 years age group.

Figure 1 **Number of notified cases (perinatal and non-perinatal) of invasive listeriosis between 1997 and 2009**



3.1.3.1 New Zealand Outbreaks

Recognition of outbreaks is often difficult because of the prolonged incubation period of the disease (anywhere between 1 and 90 days). However, in pregnancy-cases exposure and onset of illness is often less than 90 days. Recent scientific advancements in molecular typing/fingerprinting e.g. Pulsed Field Electrophoresis (PFGE) 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 1997 to 2009 only four were reported, included one of non-invasive lsiteriosis. One outbreak of invasive listeriosis involved smoked mussels as the vehicle (see below), but the source of the other two was never identified. The third outbreak of non-invasive listeriosis occurred in early 2000 and was associated with ham and corned silverside.

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. PFGE analysis 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 a food history of consuming mussels but did not specify the brand in question. Patient 4 had a history of unpasteurised milk consumption 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 in order to raise her iron levels.

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

3.2 *Toxoplasma gondii*

The recently updated Risk Profile: *Toxoplasma gondii* in red meat and meat products by ESR (Lake *et al* , 2008) is a good background source of information with a New Zealand perspective.

T. gondii is a protozoan parasite. It requires both intermediate and final (definitive) hosts to complete its life cycle. Felines are the only known animals to act as definitive hosts. The organism reproduces sexually to large numbers in the host and oocysts (unsporulated) are shed with the faeces. The oocysts require an external maturation period of 1 to 5 days to sporulate and become infective. The mature oocysts are able to infect intermediate hosts including humans and livestock animals. Infections in animals typically occur as cysts, particularly in muscular tissue (Goldsmid *et al*., 2003). Consumption of undercooked or raw meat infected with cysts is one of the recognised transmission routes to humans.

Infection (toxoplasmosis) in humans is often asymptomatic but may result in flu-like symptoms or swollen glands. Pregnant women can pass the infection to the foetus, resulting in birth defects. Disease in the immunocompromised can be severe, widely disseminated, and result in brain lesions.

3.2.1 Parameters for survival, growth and inactivation

3.2.1.1 Survival and Growth

The oocysts can survive outside of susceptible hosts. In faeces or water suspensions, infectivity can last up to 400 days (temperature range 4 to 37°C). Freshly excreted unsporulated oocysts are killed within 1 to 7 days when frozen at -21°C, but sporulated oocysts survive freezing (Frenkel and Dubey, 1973). Cysts (such as in those occurring in meat) may survive for 4 days in 8% salt solution. The parasite does not grow in foods or in other environments outside of a suitable host.

3.2.1.2 Inactivation

Cysts in meat are inactivated by cooking, freezing and irradiation. The first two will be discussed here.

Heating meat to reach a temperature in excess of 66°C throughout is sufficient to kill cysts in meat (Goldsmid *et al.,* 2003). A linear regression model compiled by Dubey *et al.,* (1990) gives the following D times 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.

Lundén and Uggla (1992) carried out microwave cooking of mutton steaks to 65°C. Residual infective cysts were detected and this was attributed to uneven heating of the steak. Spatial heating in microwaves may have improved since. However, a recent paper (El-Nawawi *et al.,* 2008) reported the microwaving of infected sheep meat (medium power for 5 minutes, in accordance with the recipe provided by the manufacturer). The authors found that microwave cooking did not kill the tissue cysts. Unfortunately temperatures were not stated in the paper.

Freezing causes cysts in meat tissue to lose infectivity. In a study of the effects of commercial freezing conditions on *T. gondii* cysts in pork tissue, Kotula *et al*., (1991) determined that -12.4°C was the theoretical temperature required to render cysts instantly non-infective.

3.2.2 Transmission routes

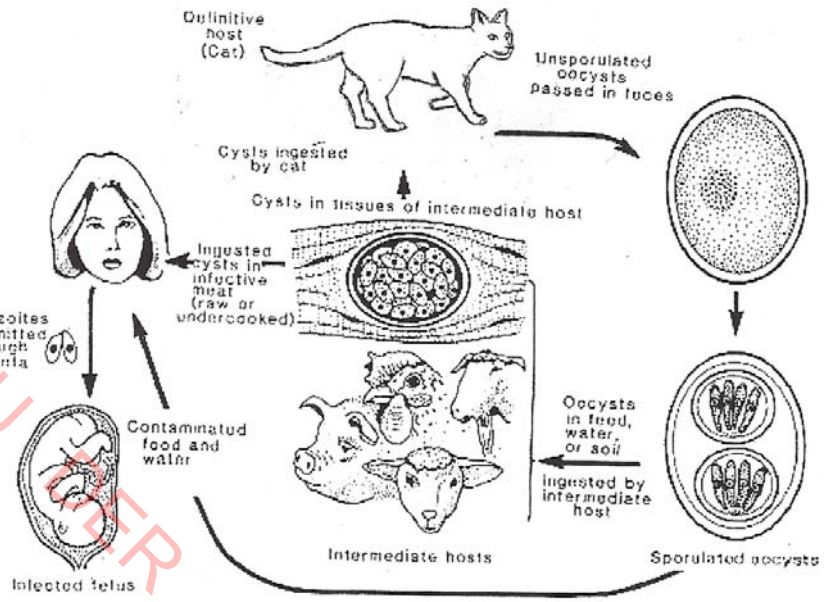
There are three transmission routes for *T. gondii* of relevance to pregnant women (Leroy and Hadjichristodoulou, 2005);

- Ingestion of raw or inadequately cooked infected meat (containing bradyzoites or cysts), or ingestion of uncooked foods that have been in contact with such meat;
- Ingestion of oocysts through direct contact with faeces passed by an infected cat or indirect contact e.g. contaminated soil on unwashed vegetables, or drinking water that has been in contact with contaminated cat faeces;
- Transplacental infection during a primary infection of the mother.

The first two transmission routes both have a food-borne aspect and are acquired, while the latter transmission route is congenital.

The life cycle is complicated and is illustrated in Figure 2. *T. gondii* oocysts are shed from the cat as definitive host and transmitted to humans via contaminated food and water.

Figure 2: Diagram of the life cycle of *T. gondii*



Source: Dubey and Beattie, (1988).

In the congenital transmission route, if a previously unexposed pregnant woman becomes infected with *T. gondii*, the parasite can cross the placenta to infect the developing foetus. The mother may remain asymptomatic (Goldsmid *et al.,* 2003). Generally speaking, the older the mother, the more likely she is to have been exposed to the parasite and seroconverted prior to pregnancy. As long as the mother remains healthy and is not immunocompromised, she will not transmit the parasite to her foetus (Lake *et al*., 2008). Conversely, younger mothers are more likely to be serum antibody negative prior to pregnancy and may experience initial infection with the parasite whilst pregnant.

To determine seroconversion, sera from pregnant women are tested for the presence the immunoglobulin G (IgG) or IgM. The presence of IgG (which can persist in the body for long periods) indicates a previous infection. IgM levels rise early in the infection and indicate that a *T. gondii* infection has recently been contracted. Seroconversion in pregnancy refers to cases where IgG and IgM are absent prior to conception but appear in blood samples following delivery, thus demonstrating infection during pregnancy (Lake *et al.,* 2008).

Luft and Remington (1982) reported that pregnant mice were more susceptible to infection with *T. gondii* than virgin controls. Earlier infection and an increased rate of mortality were also observed. In humans, Avelino *et al.,* (2003) reported that pregnant women were at 2.2 times higher risk of seroconversion for toxoplasmosis than non-pregnant women.

3.2.3 Toxoplasmosis in New Zealand

Toxoplasmosis is not a notifiable disease in New Zealand and information on fatalities due to infection is only available from New Zealand Health Information Service records. The risk profile by Lake *et. al*., (2008) includes a survey of the seropositivity of blood samples from pregnant women (n=500) in Auckland by Morris and Croxson (2004). The authors estimated that up to 2.2% of women could have initial antenatal serology consistent with recent infection. Infections during the first trimester have a 10% chance of being transmitted to the foetus and it is therefore possible that two cases of first trimester congenital toxoplasmosis occur per 1,000 pregnancies in Auckland. However, this level of clinical outcome is not currently being detected.

The available data on seropositivity for the whole New Zealand population indicates that infection with *Toxoplasma* is as prevalent here as it is in other developed countries (Lake *et al.*, 2008). Predictions of illness, however, do not appear to be borne out by observations. The number of cases of diagnosed acquired toxoplasmosis in New Zealand tota led 84 for the years 2000 to 2006.

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). Pikholz and Simmons (2004) estimated 50% of toxoplasmosis infections in New Zealand were foodborne.

3.2.4 Case-control studies

A table of case-control studies in relation to *T gondii* infection in pregnant women is attached in Appendix 3. Only one study was found to have collected information "transversally" i.e. prospective information on both risk and disease infection collected simultaneously. This study was conducted in Serbia-Montenegro between 1988 and 1991 (Bobic *et al*., 1998) and involved 1157 reproductive age females (15 years-49 years) in Belgrade. Undercooked meat consumption and exposure to soil were important risk factors. Ownership of cats was not significant. Case-control studies are not the best evidence on which to investigate risk factors, especially those using personal retrospection because of recall bias. Risk factors were also found to vary according to local food customs, food hygiene and lifestyles, so that results are difficult to interpret.

3.3 *Salmonella*

Salmonella is a facultative intracellular pathogen, and according to Smith (1999) there is no increased susceptibility when pregnant. However, in the absence of antibiotic treatment, human specific serovars such as *S*. Typhi may cause mortality of both foetus and mother.

While most published reports of *Salmonella* infection in pregnancy involve *S*. Typhi there is some evidence that, rarely, non-typhoidal salmonellosis may also result in sepsis and foetal loss. Zettell *et al.* (1995) reported a first trimester septic abortion caused by *S*. Oranienberg and a case of *Salmonella* sepsis and second-trimester pregnancy loss associated with a group C1 *Salmonella* sepsis was reported by Scialli and Rarick (1992). Early diagnosis and treatment of *Salmonella* infection during pregnancy are associated with good outcomes and the authors recommended that pregnant women with diarrheal illness be tested for salmonellosis by stool culture.

Pregnancy specific incidence data for salmonellosis in New Zealand are not available.

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. In finfish or shellfish (fish) mercury is most commonly found in the form of methylmercury and for the purposes of health risk it is assumed that any mercury present is in this form (Health Canada, 2007).

4.1.1 Contribution of fish to mercury exposure

The 2003-2004 New Zealand total diet survey (NZTDS) estimated the average dietary exposure to total mercury of an adult New Zealand female to be 0.6 µg/kg body weight/week, based on a simulated typical diet (Vannoort and Thomson, 2005a). This represents approximately 38% of the provisional tolerable weekly intake for methyl mercury. Most mercury exposure is from fish consumption. In the 2003-2004 NZTDS fresh, battered and canned fish accounted for 70% of the estimated weekly intake for an adult female.

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.

New Zealand seafood species with low mercury concentrations include anchovy, arrow squid, barracouta, blue cod, brill/turbot, some brown trout depending on where it is grown (e.g. from lake Ellesmere), cockles, eel, elephant fish, flounders, gurnard, hoki, John dory, monkfish, mussels, orange perch, oysters, parore, scallops, rainbow trout, salmon (farmed), skipjack tuna, sole, southern blue whiting, surf clams, tarakihi, toothfish, warehou and whitebait (John Reeve, 2009, NZFSA, personal communication).

New Zealand seafood species with moderate mercury concentrations include albacore tuna, alfonsino, bass, bluenose, gemfish, ghost sharks, hake, hapuka, javelin fish, kahawai, kingfish, lake Taupo trout, leatherjacket, lemon sole, ling, mackerel, orange roughy, oreo dories, red cod, ribaldo, rig, rock lobster, sea perch, silverside, skate, smooth oreo, snapper, sprats and trevally (John Reeve, 2009, NZFSA, personal communication).

New Zealand seafood species with elevated mercury concentrations include cardinal fish, dogfish, Lake Rotomahana trout, other North island trout, school shark, marlin southern blue tuna and swordfish (John Reeve, 2009, NZFSA, personal communication).

4.1.2 Health risks associated with mercury in pregnancy

4.1.2.1 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 (Clarkson, 2002).

A wide range of adverse effects has 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 (WHO, 2004).

Chronic exposure to mercury may have adverse effects on the immune system (Moszcysnki, 1997) and there is emerging preliminary evidence of potential cardiovascular effects (Stern, 2005, Virtanen *et al*., 2005).

4.1.2.2 Risks for the foetus and infant

Methylmercury readily crosses both the blood-brain barrier and the placenta and the developing foetus is considered 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, particularly the Seychelle Islands in the Indian Ocean and the Faroe Islands in the North Atlantic.

4.1.3 Advice to pregnant women

Because of the variability of mercury concentration with fish species, and the benefits of fish consumption (Ginsberg and Toal, 2008, Health Canada, 2007, Mozaffarian and Rimm, 2006,), advice to pregnant women in New Zealand with respect to fish consumption, is species specific (NZFSA, 2009a).

- 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.

4.2 Cadmium

4.2.1 Contribution of oysters and scallops to cadmium exposure

The 2003-2004 New Zealand total diet survey (NZTDS) estimated the dietary cadmium exposure of an adult New Zealand female to be 1.5 µg/kg body weight/week, based on a simulated typical diet (Vannoort and Thomson, 2005a). This equates to 97.5 µg/week for a 65 kg adult female. The simulated diet includes an average consumption of 2 g/day of oysters (based on a single 30 g serving of oysters in a 14 day period, equivalent to two oysters) and no consumption of scallops. Exclusion of oysters from the simulated diet reduces the estimated weekly exposure to 0.9 µg/kg body weight/week. The Provisional Tolerable Weekly Intake (PTWI; a permissible human weekly exposure to contaminants unavoidably associated with consumption of otherwise wholesome and nutritious foods) for cadmium is 7 µg/kg body weight/week (WHO, 2004).

A Department of Health circular from 1983 stated that a cadmium concentration of 7 mg/kg in dredge oysters was 'acceptable'. An earlier survey reported a mean cadmium concentration for dredge oysters of 3.9 mg/kg, with a range of 0.12-7.9 mg/kg (Nielsen and Nathan, 1975). Analysis of oysters in the 1997-1998 NZTDS gave concentrations of cadmium in the range 0.27-8.2 mg/kg (Vannoort *et al.*, 2000), while the 2003-2004 NZTDS gave a range of 0.63-5.3 mg/kg (Vannoort and Thomson, 2005b).

The New Zealand Food Composition Database gives a standard weight for a single oyster of 15 g. On this basis a single oyster with a cadmium concentration of 8 mg/kg would contribute 120 µg of cadmium. The consumption of oysters containing high cadmium concentrations has the potential to significantly impact on dietary exposure.

4.2.2 Health risks associated with cadmium in pregnancy

4.2.2.1 Risks for the pregnant woman

Divalent metals, including cadmium, iron and zinc, compete for common absorption pathways from the gastrointestinal tract into the portal circulation (Fox, 1988). Several studies have noted a negative correlation between cadmium indices (blood cadmium, urinary cadmium) and indices of iron status such as serum ferritin (Akesson *et al.*, 2002; Berglund *et al.*, 1994; Järup *et al.*, 1998) and iron intake (Nishijo *et al.*, 2004). This appears to be due to depletion of iron stores leading to increased intestinal absorption of iron and, consequently, cadmium.

This effect is seen most consistently for blood cadmium, rather than urinary cadmium and Hernandez *et al*. observed no significant change in urinary cadmium during pregnancy and postpartum in a cohort of non-occupationally exposed Spanish women (Hernandez *et al.*, 1996).

The reverse hypothesis, that increased cadmium absorption negatively impacts iron indices, does not appear to have been tested. However, a study on rats demonstrated a dose related decrease in the iron concentration in foetal liver and foetal membrane when mothers received 1.0 or 10 mg/kg body weight/day of cadmium (Kuriwaki *et al.*, 2005). Significantly decreased zinc and copper concentrations in foetal liver, and

copper concentrations in foetal membrane and placenta were observed in the high cadmium dose group. No impact was seen on the concentrations of these elements in foetal kidney, cerebrum, cerebellum or hypothalamus.
Cadmium has been reported to be teratogenic and embryotoxic in animal studies (Thompson and Bannigan, 2008).

4.2.2.2 Risks for the foetus and infant

Decreased birth weight/height

Several studies have investigated the relationship between maternal cadmium status and infant birth weight or birth height.

In one study, a weak correlation was found between maternal blood cadmium and amount smoked, but no correlation between maternal blood cadmium concentration and infant birth weight for 50 consecutive mother-infant pairs (Odland *et al.*, 1999). Smokers generally have higher cadmium indices than non-smokers, due to greater absorption of cadmium from the lungs than from the gastrointestinal tract (Schoeters *et al.*, 2006). Kuhnert *et al*. differentiated different predictors of infant birth weight for smoking and non-smoking mothers (Kuhnert *et al.*, 1987). In smokers (n = 77), maternal blood cadmium, placental cadmium and placental zinc concentrations were found to be negatively correlated with birth weight, while for non-smokers (n = 125) birth weight was positively correlated with cord vein red blood cell zinc levels, but not with maternal cadmium status. In an attempt to differentiate the effect of cadmium from the more general effects of smoking a cohort of women living near a lead smelter (n = 106) were compared to a control group of non-exposed women (n = 55) (Loiacono *et al.*, 1992). While placental cadmium concentrations in the exposed group were similar to those previously reported for smoking women, no association was found between placental cadmium and birth weight.

Nishijo *et al.* found that the birth weight and height of infants born to mothers with elevated urinary cadmium (n = 12) was significantly lower than for those born to mothers with lower urinary cadmium (n = 45) (Nishijo *et al.*, 2002). However, the study also noted a higher rate of pre-term babies in the high cadmium maternal group and the lower birth weights and heights were ascribed to early delivery rather than a direct impact of cadmium on birth weight. A further study by the same group of 55 mothers from Toyama, Japan found a significant inverse relationship between maternal blood cadmium and birth height (Nishijo *et al.*, 2004). The relationship was still significant after adjustment for gestational age. A small Italian study of 45 non-smoking mothers found that birth weight was negatively correlated with maternal blood cadmium and cord blood cadmium (Salpietro *et al*., 2002).

A Mexican study found significant correlations between maternal blood cadmium and cord blood cadmium and between cord blood cadmium and newborn blood cadmium, but not between maternal blood cadmium and newborn blood cadmium (n = 49) (Galicia-Garcia *et al*., 1997). Birth weight was inversely correlated with cord blood cadmium only. A Chinese study of 44 mother-infant pairs found no significant associations between indices of cadmium exposure (maternal blood cadmium, cord blood cadmium, placenta cadmium) and birth outcomes (premature labour, neonatal asphyxia) (Zhang *et al*., 2005). Cord blood cadmium, but not maternal blood cadmium or placenta cadmium, was significantly associated with decreased birth height, but not birth weight.

Impaired cognitive/psychomotor development

A small body of evidence is available linking cadmium exposure to developmental aspects of the central nervous system.

A study in the US (Maryland) looked at the lead and cadmium content of hair from 149 children aged 5-16 years (Thatcher *et al.*, 1982). Significant negative correlations were found with intelligence and school achievement scores, but not motor impairment scores. Cadmium had a stronger effect on verbal IQ, while lead had a stronger effect on performance IQ. Marlowe *et al.* (1983) found higher concentrations of hair lead and cadmium in a group of children suffering mental retardation and those with borderline intelligence than in a control group. Neither of these studies established whether the elevated hair metal concentrations were related to maternal exposure

A French study took samples of maternal and newborn hair at birth for lead and cadmium analysis (Bonithon-Kopp *et al.*, 1986). Children (n = 26) were tested for psychometric performance at six years of age. Maternal hair cadmium was significantly negatively correlated with general cognitive performance. Of five sub-scales, maternal hair cadmium was significantly correlated with perceptual, quantitative, and motor scores, but not verbal or memory. Newborn hair cadmium was significantly negatively correlated with perceptual and motor scores.

The plausibility of this hypothesis is supported by animal studies that show an impact of maternal cadmium exposure on brain development in the newborn (Gupta *et al.*, 1991). However, there is inadequate evidence to support a causative role for maternal cadmium exposure on child psychomotor development.

4.2.3 Advice to pregnant women

Since Bluff oysters and Queen scallops have high cadmium concentrations, New Zealand pregnant women are advised to minimise intake of these foods (regardless of how they are prepared) during pregnancy (NZFSA, 2009a).

4.3 Conclusion

Mercury

Wigle *et al.* (2008) conducted an extensive review of epidemiologic evidence for relationships between environmental chemical exposures, including mercury, and a wide range of child health outcomes. Table 2 summarises the conclusions of this investigation concerning the impact of methylmercury on a range of outcomes. The authors classified the available evidence as:

- Sufficient – at least one expert group has reviewed the available evidence and published a peer-reviewed report indicating a consensus view that there is a causal relationship;
- Limited – evidence is suggestive of an association between the agent and the outcome but is limited because chance, bias and confounding cannot be ruled out with confidence;

- Inadequate – available studies are of insufficient quality, consistency or statistical power to permit a conclusion regarding the presence of absence of an association or no studies exist that examine the relationship.

Table 2: Strength of epidemiologic evidence for a relationship between methylmercury exposure and developmental and child health outcomes (Wigle *et al.*, 2008)

Outcome	Strength of evidence
Spontaneous abortions	Maternal exposure, inadequate evidence
Stillbirths	Maternal exposure, inadequate evidence
Neural tube birth defects	Maternal exposure, inadequate evidence
Cardiac birth defects	Maternal exposure, inadequate evidence
Developmental milestones (mental retardation,speech, autism, dyslexia,ADHD)	High maternal exposure, sufficient evidence Low maternal exposure, inadequate evidence High childhood exposure, limited evidence
Cognitive function (0-2 yrs) Cognitive function (>3 yrs)	High maternal exposure, sufficient evidence Low maternal exposure, inadequate evidence High maternal exposure, sufficient evidence Low maternal exposure, limited evidence Childhood exposure, inadequate evidence
Problem behaviours	Maternal exposure, inadequate evidence Childhood exposure, inadequate evidence
Motor function (0-2 yrs) Motor function (≥3 yrs)	High maternal exposure, sufficient evidence Low maternal exposure, limited evidence Childhood exposure, limited evidence Maternal exposure, limited evidence Childhood exposure, inadequate evidence
Auditory function	High maternal exposure, sufficient evidence Low maternal exposure, limited evidence Childhood exposure, inadequate evidence
Visual function	High maternal exposure, sufficient evidence Low maternal exposure, inadequate evidence High childhood exposure, sufficient evidence Low childhood exposure, inadequate evidence
Postnatal height growth	Maternal exposure, inadequate evidence Lactational exposure, inadequate evidence

Very broadly, high exposure is indicated by mercury concentration in hair greater than 20µg/g of mercury and low is less than 10µg/g.

Current evidence supports a causal association between high maternal exposure to methylmercury and impaired developmental milestones, cognitive function from birth to beyond 3 years, motor function in 0-2 year olds, auditory and visual functions. High childhood exposure also leads to impaired visual function.

There is limited evidence of a causal association between low, or unspecified, maternal exposure to methylmercury and cognitive function of children over 3 years

old, motor function from birth to over 3 years and auditory function. There is also limited evidence of an association between childhood exposure to methylmercury and developmental milestones and motor function of 0-2 year olds.

Cadmium

Nutritional deficiencies that may occur during pregnancy (e.g. iron deficiency, zinc deficiency) have the potential to increase cadmium absorption and add to the maternal body burden. It is also plausible that elevated dietary cadmium may inhibit absorption of divalent nutrient ions. However, no human studies were found that specifically examined this hypothesis for pregnant women.

An extensive review of epidemiologic evidence for relationships between reproductive and child health outcomes and environmental chemical exposures, including cadmium, has been conducted (Wigle *et al.*, 2008). Table 3 summarises the conclusions of this investigation concerning the impact of cadmium on a range of outcomes. The authors classified the available evidence as:

- Sufficient – at least one expert group has reviewed the available evidence and published a peer-reviewed report indicating a consensus view that there is a causal relationship;
- Limited – evidence is suggestive of an association between the agent and the outcome but is limited because chance, bias and confounding cannot be ruled out with confidence;
- Inadequate – available studies are of insufficient quality, consistency or statistical power to permit a conclusion regarding the presence of absence of an association or no studies exist that examine the relationship.

Table 3: Strength of epidemiologic evidence for a relationship between cadmium exposure and reproductive and child health outcomes (Wigle *et al.*, 2008)

Outcome	Strength of evidence
Stillbirths	Maternal exposure, inadequate evidence
Pre-term birth	Maternal exposure, inadequate evidence
Foetal growth deficit	Maternal exposure, inadequate evidence
Neural tube birth defects	Maternal exposure, inadequate evidence
Cardiac birth defects	Maternal exposure, inadequate evidence
Musculoskeletal birth defects	Maternal exposure, inadequate evidence
Childhood leukaemia	Parental or childhood exposure, inadequate evidence

This review concluded that there was inadequate evidence for a causative role for cadmium in any of the reproductive or child health outcomes considered. However, nutritional outcomes were not considered. For most health outcomes information was scant. For the impact of maternal cadmium exposure on foetal growth deficit (birth weight, birth height) there is rather more information.

As discussed by Wigle *et al.* (2008), the information linking maternal cadmium exposure to decreased birth weight is inconsistent and lacking in statistical power. Furthermore, some studies do not adequately control for the confounding influence of smoking.

5 NZFSA PREGNANCY BOOKLET – SCIENTIFIC EVIDENCE FOR ADVICE

The food groups listed in this section are those included in the NZFSA pregnancy advice booklet. The information in the shaded boxes is taken directly from that document.

There are a number of publications issued by the New Zealand Ministry of Health and the New Zealand Food Safety Authority (NZFSA) and other authorities that address food safety during pregnancy issues. A list of these and material available overseas can be found in Appendix 4.

5.1 Breads and Cereal

Breads	All types	OK to eat

Food
Bread is a short shelf life, heated (baked) product made from fermented dough (flour, water, yeast and salt). The pH values range from 5.3 to 5.8 (USFDA, 2009) and the water activity is around 0.95. The water activity of the crust is lower.

Hazards
This food has not been linked to *L. monocytogenes* contamination (FDA/FSIS, 2003), and no outbreaks reported following its consumption. No growth is expected.

Controls
During baking, the internal temperature of the product reaches 98°C (ICMSF, 2005) and all vegetative bacterial cells are destroyed.

In uncut loaves, the lower water activity of the crust affords some protection from contamination after baking. Growth of spores that have survived cooking is possible inside bread products but fungal spoilage is much more common, especially in humid conditions (ICMSF, 2005). In less humid situations the surface of the bread may dry before mould growth becomes obvious but in either case, pathogen growth is unlikely before spoilage becomes evident.

Surveys/Outbreaks
Foodborne illness outbreaks have been associated with uncooked bread ingredients. A dispersed outbreak of 67 *Salmonella* Typhimurium PT42 cases was recently reported in New Zealand. Epidemiological and laboratory investigations identified flour as the vehicle and eating raw batter as the major risk factor. http://www.nzfsa.govt.nz/publications/food-focus/2009-11/food-focus-november-2009-website.pdf

Summary
No issues specific to pregnant women identified.

Present NZFSA advice is consistent with current scientific evidence.

Cakes, slices, muffins etc	Plain	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 of very low water activity values.

Hazards
In common with many other foods, contamination by infected food handlers is a possible source of pathogens in these products.

This food has not been linked to *L. monocytogenes* contamination (FDA/FSIS, 2003), and no outbreaks reported following its consumption. No growth is expected.

Controls
Cooking destroys vegetative bacterial cells and lowers the moisture content of the product. Other ingredients such as sugar, butter and oils also 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.

Surveys/Outbreaks
There have been some overseas reports of Hepatitis A and *Norovirus* outbreaks associated with iced buns and glazed doughnuts handled by infected food handlers (Whyte *et al*., 2002). One large outbreak of foodborne illness due to contaminated bakery products resulted in 3,000 people becoming ill. In this incident a baker making butter cream icing for buns remained at work while suffering gastroenteritis, and subsequently contaminated the food with viruses during handling.

Summary
No issues specific to pregnant women identified.

Present NZFSA advice is consistent with current scientific evidence.

Cakes, slices, muffins etc	With cream or custard	Don't eat (unless cream is newly open 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 pathogen growth.

Cream and custard are excellent microbiological growth media. Both are near neutral pH: cream pH 6.5 and custard between pH 6.2 – 7.5 (USFDA 2009; Bassen *et al*., 1989) In an Australian survey, water activities for cream and custard filled bakery products ranged from 0.89 a_w (imitation cream) to 0.95 a_w (vanilla slice) at ambient temperature. Most products were in the 0.92-0.94 range (Dunn *et al*., 2001). A New Zealand survey of 250 filled bakery products found 99.2% with water activity $\geq$0.98.

Hazards

The degree of handling associated with production of cream and custard filled bakery products increases the potential for contamination (ICMSF, 2005).

Staphylococcus aureus and *Salmonella* appear to be the most common pathogens identified in outbreaks implicating cream or custard filled products reported from overseas (ICMSF, 2005). These outbreaks are often attributed to inadequate refrigeration during manufacture and storage. 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 a potential for growth of *L. monocytogenes* (see "Custard" and "Cream" sections below).

While *Staphyloccus aureus* is a potential contaminant of these products there does not appear to be any significant issues associated with staphylococcal illness acquired during pregnancy (see Appendix 2).

Controls

A questionnaire administered in conjunction with a New Zealand survey (described below) showed most bakeries throughout the country used good food safety practice. However some problems with food hygiene, handling, and manufacturing processes were identified. 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) (NZFSA, 2009).

Surveys/Outbreaks

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).

In a recent New Zealand survey, a total of 250 cream and custard filled bakery products were tested for microbiological quality (*Escherichia coli*, coagulase positive staphylococci, *Bacillus cereus* and *Salmonella*), pH and water activity (Cornelius, 2007). *L. monocytogenes* was not included in the range of analyses. Eighty-seven percent (217) of the samples were considered to be of good microbiological quality when compared against the criteria given in the FSANZ "Guidelines for the microbiological examination of ready-to-eat foods (Food Standards Australia New

Zealand, 2002). Of the remaining samples, 24 were of "marginal quality", 6 unsatisfactory and 3 were considered "potentially hazardous" because of high *B. cereus* concentrations. The pH of the majority (88%) of samples was ≥ 6.0 and the majority had water activity values above 0.98. No specific issues arising from foodborne *B. cereus* intoxications during pregnancy were found in the scientific literature (see Appendix 2).

A small challenge test study of 23 types of cream and custard filled products was conducted in Australia in 2001. Growth rates of *E. coli*, *B. cereus* and coagulase positive staphylococci inoculated into the products were recorded at ambient temperature (19°C – 21°C) over an 8 hour period. One of five mock cream filled products showed significant growth (> 1 $\log_{10}$ increase) of *B. cereus* within 2 to 8 hours and 6 of 18 custard filled products showed a significant increase in numbers of one of more of the inoculated bacterial types within a 4 to 8 hour period (http://www.health.vic.gov.au/foodsafety/downloads/custardcream.pdf).

Summary
Growth of *L. monocytogenes* is possible in these products.

Present NZFSA advice is consistent with current scientific evidence.

Cereals	Breakfast cereals, rice, pasta etc	OK to eat – refer to milk and milk products below

Food
Cereals are high in carbohydrate and protein and have a near neutral pH.

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.

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. Both fresh and dried pasta are boiled before consumption.

Hazards
Bacteria are only able to grow in cooked or processed product and cooked rice is the cereal product most often associated with food poisoning (ICMSF, 2005).

The bacterial species which cause the most concern in cereal products are spore formers which survive cooking (e.g. *Bacillus cereus*) and other pathogens such as *Staphylococcus aureus* and *Salmonella* which may contaminate grains and flour and grow during subsequent processing steps (ICMSF, 2005).

Growth of *L. monocytogenes* is not expected in dried cereal products. Refer to sections on "Salads" and "Sushi" for further discussion of hazards associated with cold cooked cereal products.

Contamination may occur with the use of additives such as dried fruit and flavourings but the final product maintains a low water activity. Consumption with pasteurised milk is low risk.

In pasta production, there are no thermal treatment steps and bacterial growth during manufacture is possible.

Controls
Microbial growth is normally controlled by drying to a water activity of less than 0.70.
There have been some concerns around the formation of staphylococcal enterotoxin in some pasta products. The toxin is not destroyed by cooking.

Surveys/Outbreaks
Outbreaks associated with pasta are uncommon. One widespread outbreak of staphylococcal food poisoning associated with lasagne has been reported (Woolaway *et al.*, 1986). The outbreak was attributed to the use of unpasteurised egg during manufacture and drying at inadequate temperature. As substantial temperature abuse is required before hazardous levels of *S. aureus* and enterotoxin are reached, product produced under good manufacturing practices should be low risk (ICMSF, 2005).

Summary
Current NZFSA advice is appropriate for dry cereal products.

Cooked cereal products eaten cold, such as rice and pasta salads, are not specifically mentioned in this section of the NZFSA document and no advice regarding leftovers is given.

5.2 Milk and Milk Products

Cheese	Soft unpasteurised (raw milk) cheese (e.g. Roquefort)	Don't eat

Food
Soft cheese is defined by FSANZ as containing 50% - 85% moisture. Soft cheese types include unripened (e.g. cottage, cream, Mozzarella), ripened (e.g. Camembert and Brie), salt-pickled (e.g. feta) and whey (ricotta). Soft cheese has relatively high water activity and near neutral pH values in comparison to low moisture cheeses.

Hazards
The water activity and pH values can provide conditions favourable for pathogen growth. Soft cheese is a high risk product, particularly when produced from raw milk.

Heat may be used in the production of some types of soft cheese (e.g. Mozzarella – described later under soft Italian types), but for most raw milk cheeses with less than 60 day ripening periods there are no pathogen control steps in the production process. Safety depends on raw milk quality and prevention of contamination during manufacture, handling and storage. Acid production by lactic acid bacteria may slow the growth of pathogens but most soft cheeses do not develop acidity below pH 5 during fermentation. A rise in pH occurs during ripening (ICMSF, 2005).

There is a potential for growth of *L. monocytogenes*. Based on averaged FDA/FSIS data for fresh soft Mexican style cheese, the growth expected in 2 days at 5°C is 0.2 $\log_{10}$ CFU/g. Data for other cheese types is given in the itemised sections below.

Controls

In New Zealand, pasteurisation of milk for cheese making is a major critical control point. There are currently no unpasteurised soft cheeses permitted for import into New Zealand. Those raw milk cheeses that have approval for import are extra-hard grating cheeses (such as Parmesan), Gruyere, Sbrinz and Emmental (NZFSA, 2009b). Imported semi-hard raw milk Roquefort cheese has also recently become available in New Zealand but the NZFSA has advised vulnerable groups including pregnant women to avoid this product http://www.nzfsa.govt.nz/consumers/higher-risk-specific-foods/raw-milk/NZFSA_RoquefortCheeseDL.pdf

The advice given in the NZFSA pregnancy document is consistent with international recommendations regarding these products.

Surveys/Outbreaks

Table 4 summarises details of overseas outbreaks of listeriosis in which cheese has been identified as the cause. The majority of these episodes were transmitted by soft cheeses made from raw milk.

Table 4: Overseas outbreaks of listeriosis where cheese was the implicated vehicle

Country	Milk type	No. Cases	Cheese Type	Reference
Canada, 2002	Sub-pasteurisation temperature treatment	17 (3 neonates and 14 adults)	Soft and semi-hard, moistures not given (all 4 types produced were contaminated)	Gaulin *et al.* (2003)
France, 1995	Raw	20 (2 spontaneous abortions, 4 premature births, 2 stillbirths)	Brie de Meaux soft cheese	Goulet *et al.* (1995)
Sweden, 2001	Raw	48 (febrile gastroenteritis, 3 pregnant, 1 had symptoms of headache, vomiting and fatigue. All 3 treated with amoxycillin and gave birth normally)	On-farm cow, goat and blended milk fresh cheeses from one farm	Carrique-Mas *et al.* (2003)
Switzerland, 1983-87	Unknown	122 cases, 57 in non-pregnant adults and 65 cases in newborn infants and pregnant women.	Vacherin Mont D'or, eaten in winter months only. Only observed during the winter months	Büla *et al.* (1995) Codex (2002:28)

Country	Milk type	No. Cases	Cheese Type	Reference
Switzerland, 2005	Raw	10 cases; 2 elderly fatalities, 2 septic abortions, 6 others hospitalised	Canton Neuenburg's Tomme soft cheese	Bille *et al.*, 2006
USA, 1985	Pasteurised milk contaminated with raw milk post processing.	142 (10 neonatal deaths, 20 stillbirths and 18 non-pregnant adult deaths, total 48 deaths). 85 cases were associated with cheese consumption*	Mexican style (queso fresco, cotija).	Linnan *et al.* (1988); CDC (1985)
USA, 2000-01	Raw milk cheese unlabelled	12 (10 were pregnant, resulting in 5 stillbirths, 3 premature deliveries and 2 infected newborns)	Mexican style (queso fresco)	CDC (2001)
Denmark, 1989-90	Unknown	69 (10 pregnant, no information on outcomes)	Blue Cheese and hard cheese	Jensen *et al.*, (1994)

* Calculated from data in the paper, the other cases were assumed to be "background" sporadic cases.

Summary

There is a potential for growth of *L. monocytogenes* in these products. However, there are currently no examples of domestically produced soft cheeses made from raw milk in New Zealand.

While Roquefort is used as an example of a "soft unpasteurised cheese" in the NZFSA document this cheese is usually categorised as a "semi-hard" variety.

Cheese	Soft pasteurised cheese (brie, camembert, blue, ricotta, mozzarella, feta etc)	Generally do not eat unless heated until piping hot*

* If these products are purchased in the primary producer's original packaging, small quantities can be eaten immediately after opening. Do not reseal and eat later, and do not eat if they have been repackaged in a deli or supermarket as they may become contaminated with pathogens during this process.

Food

See definition for soft unpasteurised cheese above. Further information is given in the Risk Profile for *L. monocytogenes* in soft cheese, available on the NZFSA website: http://www.nzfsa.govt.nz/science/risk-profiles/FW0382_L_Mono_in_soft_cheese_November_2005.pdf (Lake *et al.*, 2005a)

Soft Italian type cheese

Soft unripened Italian style cheeses (e.g. mozzarella, provolone, carioccvallo, bocconcini, scamorze) are identified individually in the FSANZ (2005) "Questions and answers" document.

Blue vein

Internally ripened blue-vein cheese (e.g. blue gorgonzola, stilton, roquefort) undergo ripening after being inoculated with *Penicillium* spores. They are usually classified as semi-soft cheeses.

Hazards

As soft cheese manufactured in New Zealand is 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.

The ability of *L. monocytogenes* to grow on the surface of 24 types of cheese available in the USA was studied by Genigeorgis *et al*. (1991). Overall, a highly significant correlation of *Listeria* growth with cheese pH values >5.5 and/or the absence of lactic acid starter cultures during cheese manufacture was observed. Information provided in the FDA/FSIS Quantitative Risk Assessment tables (Appendix 1) also shows growth in fresh soft cheese, most soft unripened cheeses and half of the soft ripened cheeses tested.

Potential for growth of *L. monocytogenes*:
Brie and Camembert - growth expected in 2 days at 5°C is 0.1 $\log_{10}$ CFU/g
Ricotta – growth expected in 2 days at 5°C is 0.3 $\log_{10}$ CFU/g.
Mozzarella - growth expected in 2 days at 5°C is 0.4 $\log_{10}$ CFU/g

Feta cheese – decrease in numbers expected at 5°C is - 0.2 $\log_{10}$ CFU/g per day.
For semi-soft cheese – the decrease in numbers expected at 5°C is - 0.04 $\log_{10}$ CFU/g per day (averaged data from FDA/FSIS).

Blue vein – FDA/FSIS (2003) data shows the expected decrease in numbers at 5°C is -0.05 $\log_{10}$ CFU/g per day (FDA/FSIS, 2003). Recent New Zealand data is presented in the "survey" section below".

Surface mould ripened cheese

The relatively high moisture content of surface ripened cheese (e.g. Brie and Camembert), along with a nearly neutral pH at the surface of fully ripened cheeses allow rapid growth of the pathogen (FSANZ, 2005). With a possible exception noted for Italico cheese (Comi *et al.* 1990).

Many studies indicate that the surface of mould ripened cheeses have conditions more favourable for the growth of *L. monocytogenes* than at the centre.

Salt-cured or pickled

Feta cheese appears to be particularly difficult to categorise in terms of *L. monocytogenes*. The collated data from the FDA/ FSIS shows that the pathogen generally decreases over time. Differing advice between overseas agencies may be due to data obtained from different isolates.

Controls

Soft Italian type cheese

These cheeses are typically subjected to 71-88°C during processing, sufficient to inactivate any *L. monocytogenes* present, although post-processing contamination is a possibility. Mozzarella for example, is produced by the "pasta filata" process which involves heating curd of a suitable pH in a water bath, where the curd is kneaded and stretched, ensuring it is smooth and free from lumps. In a study by Buazzi *et al.,* (1992), the "stretching" process in mozzarella production was carried out for 3-4 minutes in 77°C water. The curd reached 58-65°C and this process was found to eliminate *L. monocytogenes* inoculated at 6.2 x 10^4 CFU/g (Lake *et al.,* 2005a; Abdalla *et al*., 1993). It should be noted, however, that different manufacturers may use different, and possibly less stringent, processes.

The advice from FSANZ is that soft Italian style cheeses are safe to consume provided they are pre-packaged. Because of the risk of post-processing contamination these cheeses should not be consumed if purchased from the delicatessen counter or similar situation.

Surface mould ripened cheese

Growth generally does not occur in the core of the cheese as pH values are inhibitory. Liu *et al.,* (2007) analysed the effect of spatial distribution of survival of *L. monocytogenes* in five sampling locations during manufacture and ripening of Camembert cheese. The core of the cheese had the lowest *L. monocytogenes* population due to low pH values. Growth rate at other locations was similar but the top surface supported the highest population. Over time, the population decreased up to 20 days of ripening because of the low pH values (pH slightly below 5.5). Thereafter, the population increased as the pH increased to >5.5.

Whey cheese

Whey cheeses such as ricotta and ricotone are usually cooked to between 82 and 88°C, therefore *L. monocytogenes* will be completely destroyed during processing. However there is the potential for post-processing contamination (FSANZ, 2005).

Ricotta is produced by direct acidification and has a low pH and high moisture content. It was observed to be the best growth substrate compared to the other cheese types since it supported growth from 4°C to 30°C despite the presence of acetic acid and the preservative potassium sorbate (FDA/FSIS, 2003).

Blue vein

Comparisons have been made between interior and surface methods of ripening. Kinderlerer *et al*., (1996) carried out a study comparing internally ripened blue-vein cheese (Bleu d'Auvergne and Fourme d'Ambert) with surface mould ripened cheese (Brie), both made from unpasteurised milk. *L. monocytogenes* was isolated only from the surface mould ripened cheese. Higher concentrations of inhibitory free medium chain fatty acids (MCFA) were found in the veins of blue mould ripened cheese as opposed to the white regions of the same cheese. The acids were not detected in Brie. The study concluded that the higher concentrations of MCFA (hexanoic, octanoic, decanoic, dodecanoic and tetradecanoic acids) present in the blue veins of internally mould-ripened cheeses could act as a natural preservative and inhibit the growth of *Listeria* in conditions where they might be expected to grow. However, as

highlighted by FSANZ (2005), this does not preclude the surface from being highly contaminated during ripening.

Salt-cured or pickled

The FSANZ (2005) view is that feta can support the growth of *Listeria* during the initial stage of ripening and that the brine solution is a potential source of contamination. Although growth will stop when the pH falls to 4.6 during ripening, the bacteria can survive for more than 90 days (although numbers will gradually decline during this period).

Surveys/Outbreaks

During 2003/2004, ESR carried out a survey of 307 soft and semi-soft cheeses (around 50 samples were semi-soft blue cheese) for *L. monocytogenes*. 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* but *L. welshimeri* was detected in one semi-soft blue cheese (Wilson, 2004).

The ESR laboratory database contains the results for testing approximately 1,335 soft cheeses for *L. monocytogenes* (from 2001 to October 2008). The majority of these cheeses were imports being tested before release onto the New Zealand market. *L. monocytogenes* was detected in a Mozzarella sample (rejected in Taiwan and re-imported into New Zealand) and both *L. monocytogenes* and *L. innocua* from a Gorgonzola sample (a blue veined cheese) (personal communication, Craig Houston, ESR October 2008).

In a recent 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 (JA Hudson, *pers. comm.*).

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 (JA Hudson, *pers. comm.*).

Summary

There is currently no evidence to link soft pasteurised cheese consumption with listeriosis in New Zealand and food surveys indicate a very low prevalence of *L. monocytogenes* in locally produced soft cheeses (Lake *et al.,* 2005a).

There is, however, the potential for growth of *L. monocytogenes* in most of these products if post process contamination occurs.

Cheese	Hard yellow cheese (cheddar, parmesan etc)	Store in fridge

Food
Hard cheese has a moisture content defined as less than 50% (based on FSANZ definitions). A Risk Profile for *L. monocytogenes* in low moisture cheese can be found on the NZFSA website: http://www.nzfsa.govt.nz/science/risk-profiles/FW0440_L_mono_in_low_moisture_cheese_Final_Mar_2007.pdf (Lake *et al.*, 2005b)

Hazards
The FDA/FSIS (2003) information on *L. monocytogenes* shows a decline in population in most semi-soft cheeses, hard cheeses and all processed cheeses (see Appendix 1). For both hard and processed cheese the decrease in numbers expected at 5°C is -0.05 $\log_{10}$ CFU/g per day.

The published data indicate that provided the pH is below 5.5 (through use of a starter culture) growth of *L. monocytogenes* seems unlikely, although the organism may survive. These data are sufficiently variable that a reliable prediction of behaviour, in all low moisture cheeses made with these types of milk, is not possible. For such cheeses, a careful consideration of the production process, both in generic terms and for a specific manufacturer is warranted. However, all showed a decrease in *L. monocytogenes* concentration over time during ripening (Genigeorgis *et al*., 1991; Lake *et al*., 2005b).

Controls
Where properly pasteurised milk is used for manufacture, *L. monocytogenes* would not be expected to occur in low moisture cheeses unless environmental contamination occurs during manufacture or ripening. In such cases growth is unlikely due to low water activity and low pH (<5.5). Exceptions may occur when the (surface) pH is raised, such as in some mould or smear ripened cheeses.

For low moisture cheese made with raw milk, or thermised milk (in which *L. monocytogenes* may survive heat treatment), control of the organism is dependent on conditions during manufacture and ripening.

Processed cheese is not specifically mentioned in the above categories but is regarded as safe to eat. The product undergoes heat treatment during manufacture and also contains preservatives.

Surveys/Outbreaks
Whyte and Wong (2002) analysed 300 samples of retail packed grated low moisture cheeses for *L. monocytogenes*. All samples were negative.

Results of a 2003/2004 ESR survey of 307 soft (>50% moisture) and semi-soft (39% to 50% moisture level) cheeses are given in the soft cheese section above.

Very few outbreaks have been attributed to low moisture cheeses. In Canada during 2003, 17 cases of listeriosis were notified, implicating 4 types of "soft and semi-hard" cheese (Gaulin *et al*., 2003). In 1997, an outbreak of *Salmonella* Gold-coast was

notified in Northern England (CDR, 1997). Analysis showed a strong association with consumption of a Somerset produced coloured cheddar cheese.

Summary
Where pasteurised milk is used for manufacture, *L. monocytogenes* is not expected to occur in low moisture cheeses unless environmental contamination occurs during manufacture or ripening. Growth is not expected and concentrations will decrease over time

Present NZFSA advice is consistent with current scientific evidence.

Cheese	Cottage cheese, cream cheese etc.	Buy in sealed packs; eat cold or cooked within two days of opening pack

Food
Soft unripened and white curd cheeses such as cottage and cream cheeses have high moisture content and low pH (4.6 – 5). Shelf life is usually short.

Hazards
The FDA/FSIS (2003) growth data for soft unripened cheese (Appendix 1) shows the potential for slow growth of *L. monocytogenes*. Nine of 29 data sets indicated a decline while the other 20 sets show an increase. Growth or decline appears largely dependent on pH values. Exponential growth rates modelled at 5°C ranged from –0.333 to 1.423 $\log_{10}$ CFU/day. The average growth rate modelled at 5°C was 0.09 $\log_{10}$ CFU/day.

For cottage cheese - the expected growth in 2 days at 5°C is 0.07 $\log_{10}$ CFU/g (FDA/FSIS, 2003). For cream cheese - the FDA/FSIS information includes data from two studies. One indicated an expected decrease in numbers at 5°C of -0.06 $\log_{10}$ CFU/g per day while the other showed growth in 2 days at 5°C of 2.8 $\log_{10}$ CFU/g.

Controls
The low pH values are the primary controlling factor for *L. monocytogenes*.

Surveys/Outbreaks
In a recent survey of three brands of New Zealand cream cheese, no growth of *L. monocytogenes* was observed when incubated for up to 280 days at 7.7°C (JA Hudson, *pers. comm.*). The pH of the samples was between 4.3 and 4.6.

Summary
Providing the cheese has a sufficiently low pH, the potential for growth of *L. monocytogenes* is small.

Butter	All types	Store in fridge

Food
Butter is a water-in-oil emulsion, containing at least 80% fat.

Hazards

There is no survival or growth data specific for butter in the FDA/FSIS (2003) review. Properly formulated butter with well dispersed moisture droplets is considered an unlikely growth environment for *L. monocytogenes* (ICMSF, 2005; Lewis *et. al.*, 2006). However, while various studies have shown no or poor growth in inoculated butter, others have reported growth at chill and ambient temperatures (Lewis *et. al.*, 2006; Olsen *et al*., 1988).

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. No illnesses were associated with the product http://www.fda.gov/oc/po/firmrecalls/zanders04_04.html

Controls

In addition to the low water activity other factors controlling pathogen growth include pH, salt and regulation of the emulsion droplet size.

Margarine and other yellow fat spreads are not specifically mentioned in the NZFSA document but consumers will probably assume that these products are in the same category. Margarine, also a water-in-oil emulsion containing at least 80% fat, has largely been superseded by other yellow fat spreads, usually with lower fat content (between 40 and 70%). These products are often more vulnerable to microbiological problems than margarine (ICMSF, 2005). Control measures include in-line pasteurisation, pH, use of preservatives and attention to process hygiene and equipment design.

The viability of *L. monocytogenes* in yellow fat spreads at a range of storage temperatures has been investigated by Holliday and Beuchat (2003). A six strain cocktail of *L. monocytogenes* was inoculated into seven yellow fat spreads at approximately 6 $\log_{10}$ CFU/g. The spreads were margarine (1), butter-margarine blend (1), and dairy/ non-dairy spreads (5). Storage was at 4 4°C, 10°C and 21°C for up to 94 days. *L. monocytogenes* did not grow in six of the products. In the butter/margarine blend however, the population increased between 42 and 63 days when stored at 10°C. The population also increased between days 3 and 7 when the blend was stored at 21°C. The authors concluded that traditional margarine and spreads not containing butter are "not potentially hazardous foods" because they do not support the growth of *L. monocytogenes*.

In a related study, Holliday *et al*. (2003) examined the potential effects of cross-contamination and temperature abuse by consumers of multi-use containers. Survival and growth characteristics of a range of pathogens including *L. monocytogenes* were determined in three butter products (pH 4.58 - 6.40, fat 43 - 78%), two yellow fat spreads (pH 4.5 - 5.37, fat 49 - 61%) and one light margarine (pH 5.34, fat 31%). All products, except two high fat butter samples, contained preservative. Water condensation was induced onto the surface of inoculated products before storage at 4.4°C or 21°C for up to 21 days. At 21°C *L. monocytogenes* grew on sweet cream whipped salted butter (pH 6.40) between 1 and 2 days and between 7 and 14 days at 4.4°C. Growth was not supported on any of the other products up to 21 days. Inactivation was more rapid in products stored at 21°C, compared with 4.4°C, and in products containing preservatives and acidulants.

Surveys/Outbreaks
Three overseas outbreaks of listeriosis associated with consumption of butter have been reported. Two of the episodes involved pregnancy related cases and the third outbreak was in a cancer ward. *L. monocytogenes* was isolated from implicated product in two of the outbreaks (11,000 CFU/g and 180 CFU/g).

There are no reported cases of foodborne illness associated with the consumption of margarine or reduced-fat spreads (ICMSF, 2005).

No data for the prevalence of *Listeria monocytogenes* or other *Listeria* species 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. There were 99.4% satisfactory samples (not detected in 25g, <20 CFU/g), 0.5% were acceptable ($<10^2$) and 0.1% unacceptable ($\geq 10^2$).

Summary
The behaviour of *L. monocytogenes* in butter is not well understood. However, there is generally little evidence identifying butter as a transmission vehicle and there have been no notified *L. monocytogenes* outbreaks caused by this product in New Zealand. The risk from butter appears to be low, but given the long shelf life of this product, advice to buy in small quantities should be considered.

No growth is expected in margarine products and properly formulated yellow fat spreads.

Cream	Fresh, unwhipped or whipped, sour cream etc	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.

Hazards
As *L. monocytogenes* is unlikely to survive the heat process, post-pasteurisation contamination is the most likely potential cause of illness associated with this product (ICMSF, 2005).

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

Only one entry relating to the growth rate of *L. monocytogenes* in cream is recorded in the FDA/FSIS QRA. Rosenow and Marth (1987) found an increase of 3.3 $\log_{10}$ units in 18 days when whipping cream was stored at 4°C and 4 $\log_{10}$ units in 8 days when stored at 8°C.

Growth expected in 2 days at 5°C is 0.5 $\log_{10}$ CFU/g.

Controls

The high fat content, which can range from 10 to 55%, may have a protective effect on pathogens, and requires higher pasteurisation temperatures than those applied to liquid milk. Other heat treatments include sterilisation and UHT.

Surveys/Outbreaks

No surveys or outbreaks were located for New Zealand. Most overseas outbreaks are associated with salmonellosis in desserts or dishes prepared with cream (ICMSF, 1998). Other pathogens associated with cream are *Staphylococcus aureus* and STEC. Seven cases of *E. coli* O157 were reported in England in 1998. Three primary cases were known to have consumed unpasteurised cream, infection spread person-to-person (CDSC, 1998).

Summary

Pasteurisation is the major control factor for pathogens in cream. There is a potential for growth of *L. monocytogenes* where post-pasteurisation contamination occurs.

Custard	Ready-made chilled (packaged)	Eat within two days of opening

Food

Commercially prepared ready-made chilled custard is a pasteurised product containing milk, cornflour, sugar and flavouring. As with other pre-packaged heat treated foods they are generally regarded as safe to consume cold.

Hazards

Bacillus spores which may be present in the powder may survive heating processes, but will not become a food safety issue unless the custard is allowed to cool slowly and then stored at ambient temperature. This is unlikely under commercial preparation protocols.

No specific data for growth of *L. monocytogenes* in ready-made custard was located.

Controls

Pasteurisation and good manufacturing practice.

Surveys/Outbreaks

No surveys were located for New Zealand. No domestic or international outbreaks of listeriosis attributed to custard consumption have be identified but there have been four recalls of ready-made chilled custard product reported on the Food Standards Australia New Zealand web site (http://www.foodstandards.gov.au) over the last ten years. The recalls (all in Australia) were for *Listeria* contamination (2), spoilage before expiry date (1) and contamination (1).

Summary

Pasteurisation and good manufacturing practice are the main controls used in the manufacture of this product. While no data for the growth of *L. monocytogenes* in ready-made custard was located it should be assumed that growth is possible. The "eat within two days of opening" advice acknowledges the potential for contamination and growth after opening.

Custard	Home-made	Eat while hot immediately after cooking; don't eat leftovers

Food
Home made custard may 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 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
Bacillus spores which may be present in the powder can survive cooking but will not become a food safety issue unless the custard is allowed to cool slowly and then stored at ambient temperature.

No specific data located for growth of *L. monocytogenes* in home-made custard was located.

Controls
It is likely that pasteurisation conditions are achieved during cooking.

Surveys/Outbreaks
No surveys on home-made product or outbreak reports were located.

Summary
Adequately cooked home-made custard is safe to eat immediately after preparation. The "don't eat leftovers" advice may be conservative. Although the custard is a good growth medium for microbial contaminants, leftovers which have been promptly chilled should be safe to eat if the advice given for other leftover cooked foods is followed (see section 5.7).

Milk	Pasteurised	Ideally drink or use within two days of opening

Food
Milk is almost neutral in pH (6.7) and has high water and nutrient content.

Hazards
Milk is an excellent growth substrate for a wide range of microorganisms including *L. monocytogenes* and other foodborne pathogens. Faecal contamination and to a lesser extent listerial mastitis are generally the main sources of *Listeria* contamination of raw milk.

Growth of the pathogen has been observed in pasteurised milk (Gay and Amgar, 2005). The behaviour of six strains was observed in pasteurised milk kept at 15°C for 65 hours. A 2 to 3.8 log increase (strain dependent) occurred. The FDA/FSIS (2003) information on *L. monocytogenes* in Appendix 1 shows growth observed in all types of heat-treated milk. For pasteurised milk held at 4°C, a 2 log increase in 7 days was observed. Growth expected in 2 days at 5°C is 0.7 $\log_{10}$ CFU/g.

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 *Listeria monocytogenes* in milk (ICMFS, 2005).

Surveys/Outbreaks
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 (7 foetal or infant and 42 immunocompromised adults) in the USA was investigated by Fleming *et al*. (1985). Although *Listeria* 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. There was no evidence of pasteurisation failure at the plant and although post-pasteurisation contamination could not be completely excluded, it was considered unlikely (Rebagliati *et al*., 2009). It was established, however, 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.

A 45 case outbreak of *Listeriosis* caused by pasteurised chocolate milk in the USA was reported by 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 (Rebagliati *et al*., 2009)

Summary
Pasteurisation is the major control factor for pathogens in milk. There is a potential for growth of *L. monocytogenes* where post-pasteurisation contamination occurs.

Milk	Unpasteurised (raw)	Don't drink or use

Food
Refer to milk above.

Hazards
Any pathogen that is associated with milk providing animals may be an accidental direct contaminant of the product. Secondary contamination may result from equipment and handling.

Gay and Amgar (2005) report an increase of *L. monocytogenes* in raw milk kept at 15°C for 65 hours at 0.8 to 2.3 log units. The FDA/FSIS (2003) information on *L. monocytogenes* in Appendix 1 shows growth observed in all types of raw milk.

Growth expected in 2 days at 5°C is 0.4 $\log_{10}$ CFU/g.

Controls
Raw milk receives no heat treatment or other processing step to reduce the microbial load.

Surveys/Outbreaks
A survey of raw whole milk in New Zealand during 1986/87 found none of 71 samples contained *L. monocytogenes* although 14.1% of samples contained *L. innocua* (Stone, 1987).

Data on the consumption of raw milk in New Zealand are very limited, with no raw milk consumption recorded in the 1997 National Nutrition Survey. In a review of *Campylobacter* transmission routes (Lake, 2005), an estimated 170,000 to 250,000 New Zealanders could be consuming raw milk. The lower assumption was based on each of 17,000 dairy farms potentially supplying raw milk to up to 10 family members and friends. The higher estimate was based on epidemiological studies of campylobacteriosis.

Outbreaks of *T. gondii* implicating unpasteurised goat's milk consumption have been reported from overseas (Skinner *et al.,* 1990; Sacks *et al*., 1982).

Summary
Consumption of raw milk poses a significant health risk not just for pregnant women but also to the general population (ICMSF, 1998).

The current NZFSA advice is consistent with scientific evidence.

Ice cream	Packaged	Choose single serve pots, tubs or slices

Food

Ice cream has been defined in the Food Standards Code (Standard 2.5.6) as

> "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."

The principal components of ice cream are milk fat, non fat milk solids, sugar, and emulsifiers/stabilisers. The latter include polysaccharides to bind free water and retard ice crystal growth during freezing and storage, while emulsifiers are incorporated to stabilise fat droplets (Belitz and Grosch, 1987). In some ice creams, eggs are used as a source of emulsifying agents.

Hazards

Potential pathogens in packaged ice cream include *T. gondii* and *L. monocytogenes.* FDA/FSIS (2003) information for *L. monocytogenes* shows no growth in ice cream at temperatures lower than -18°C (Appendix 1).

Some flavouring ingredients may be added after pasteurisation and these ingredients may result in contamination of the product (ICMSF, 1998). However, it is unlikely that any bacterial growth will occur during storage.

Controls

The New Zealand Food (Milk and Milk Products Processing) Standard 2007 requires ice cream mix to undergo an "ice cream treatment" prior to freezing. The times and temperature used for this heat treatment differ from those used for milk as the thermal resistance of *Listeria* may be increased due to the protective effect of some ice cream components (fats, sugars, emulsifiers etc.).

Surveys/Outbreaks

Prevalence data available for retail ice cream in New Zealand suggests that *Listeria* contamination in both imported and domestic product is rare (Lake *et al*. 2003). However, if present, *Listeria* will survive freezing. One study has shown essentially 100% recovery of *L. monocytogenes* from ice cream after 14 weeks of storage at -18°C (Palumbo and Williams, 1991).

There have been no *L. monocytogenes* or *T. gondii* outbreaks implicating ice cream confirmed in New Zealand

Summary

Despite high consumption in New Zealand, evidence suggests that ice cream is a low risk product.

Ice cream	Soft serve	Don't eat

Food

Soft-serve ice cream is produced from pre-made mix. It 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 minus 6°C to minus 7°C and packed immediately after this initial freezing stage. The product is stored at refrigeration temperature (<5°C) and only frozen at the retail point of sale (NZICMA, 2004).

Hazards

There are no New Zealand human illness data indicating problems with soft serve ice cream. However, between production and consumption, there is a greater potential for slow growth of *L. monocytogenes* under refrigerated conditions (<5°C) than in hard ice cream (Lake *et al.,* 2003).

The pH of ice cream is close to neutral and does not provide any hurdle to the growth of the *L. monocytogenes* pathogen (Nichols and de Louvois, 1995).

FDA/FSIS (2003) data shows no growth of *L. monocytogenes* in soft-serve ice cream at temperatures below -18°C (Appendix 1). However, no data could be found for growth rates in the temperature range at which this product is normally stored before dispensing.

Controls

The New Zealand Food (Milk and Milk Products Processing) Standard 2007 requires ice cream mix to undergo an "ice cream treatment" prior to freezing. The times and temperature used for this heat treatment differ from those used for milk as the thermal resistance of *Listeria* may be increased due to the protective effect of some ice cream components (fats, sugars, emulsifiers etc.).

Surveys/Outbreaks

International surveys of the microbiological quality of soft serve ice-cream show that it is more likely to be of poorer microbiological quality than hard frozen product (Little *et al.*, 1999). Freezing and dispensing machines are also found to be major contributors to contamination.

Summary

No specific growth data was located but it is probable that *L. monocytogenes* could grow in this product.

Yoghurt	All types	Check use-by date; ideally eat within two days of opening

Food

The Australia New Zealand Food Standards Code defines yoghurt as "fermented milk where the fermentation has been carried out with lactic acid producing microorganisms" (Standard 2.5.3). The pH must not be higher than 4.5 and the microorganisms 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).

Hazards

Yoghurt and other fermented milk products have been associated with very few food poisoning outbreaks (ICMSF, 1998). The combination of pasteurisation, 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 1 records a decline in population for all three papers cited. One of these studies study showed survival of *L. monocytogenes* up to 24 days but the greatest drop in numbers in all three studies occurred within 12 days. The decrease in numbers expected at 5°C is -0.2 $\log_{10}$ CFU/g per day.

Controls

The Food (Milk and Milk Products Processing) Standards 2007 require that the manufacture of yoghurt includes a pasteurisation process. The standards also require that any substance added after the product has been pasteurised must meet appropriate food safety requirements.

Surveys/Outbreaks

Two outbreaks of staphylococcal food poisoning associated with yoghurt have been reported in New Zealand (Anon 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.

Summary

Commercially produced pasteurised yogurt is a low risk product for pregnant women and evidence suggests that there is no potential for growth of *L. monocytogenes*. 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. The "eat within two days of opening" advice is, therefore, conservative.

Yoghurt made in the home (or in premises exempt from the usual processing requirements) may not have the same low level of risk as commercially prepared product.

5.3 Eggs

Raw eggs	In egg flips, eggnog, smoothies, home-made mayonnaise and dressings, home-made ice cream, mousse and tiramisu etc	Don't eat

Food

Most eggs for human consumption are derived from hens, but eggs from other birds such as ostriches, ducks, and quail are also consumed. Eggs are generally marketed and consumed as shell eggs. For commercial use, and in food service operations, eggs are broken from their shells, and may then be mixed whole or separated into whites and yolks. Further processing includes pasteurisation, drying, and possibly mixing with other ingredients.

Hazards

Egg contents are moist (approximately 64% water) and contain the nutrients required for bacterial growth. *Salmonella* is the predominant pathogen associated with eggs. *L. monocytogenes* has not been isolated from the contents of intact eggs but has been found on eggshells and in the environment of laying hens. It has also been found as an environmental contaminant in raw and pasteurised egg pulp. Apart from one small, two-case, episode, there have been no outbreaks of foodborne *L. monocytogenes* illness outbreaks due to the consumption of eggs or egg products documented (FDA/FSIS, 2003; Rivoal *et al* ., 2010).

Salmonella contamination in or on eggs has been shown to make a significant contribution to human illness in several overseas countries (Lake *et al*. 2004). In New Zealand, however, the relationship between the consumption of eggs and human salmonellosis is not clear.

Internationally, the major public health issue in egg related outbreaks is associated with internal contamination of eggs with specific *Salmonella* strains (predominantly *S*. Enteritidis phage type 4, plus a few other *S*. Enteritidis phage types, and *S*. Heidelberg).

Controls

Biosecurity and animal husbandry practices are used to minimise contamination in laying hens and the production environment. Physical barriers preventing bacterial contamination of the contents of unbroken eggs include the cuticle, shell and associated membranes, and antimicrobial components of the albumen (ICMSF, 1998; Lake *et al*., 2004).

Surveys/Outbreaks

Surveys in New Zealand have not found any evidence of internal contamination of locally produced eggs. Other serotypes have, however, been found as contaminants on the external surface of shells (*S*. Thompson or *S*. Infantis isolated from 13 of 93 samples in 2001 and *S*. Infantis isolated from 9 of 514 samples in 2005). The 2005 survey showed concentration to be low – eight samples were less than 5 MPN/shell and the other positive sample was 44 MPN/egg (Wilson, 2007).

In New Zealand egg and egg dishes are often recorded as a suspect vehicle in reported foodborne illness episodes (23 outbreaks in the 10 year period to 2008). However, there have been no confirmed outbreaks of salmonellosis due to the consumption of eggs. In a two person outbreak in 2001, the same strain of *Salmonella* was isolated from both the cases and the implicated food (raw egg mayonnaise), but contamination of the food after illness could not be discounted (Lake *et al.* 2004).

In recent years in Australia, eggs and dishes containing eggs (especially those lightly cooked or containing raw egg) have become the most common food vehicles identified in *Salmonella* outbreaks (Stephens, 2008). During 2006 and 2007 egg related foodborne illness episodes due to various strains of *S.* Typhimurium were reported from all but one jurisdiction. Foods implicated in the outbreaks included chocolate mousse, salad dressing, egg-nog milkshake and undercooked or lightly cooked eggs. The reasons for the recent increase in outbreaks linked to eggs in Australia are unclear but inadequate food handling and/or storage procedures were identified as contributing factors in the many of the outbreaks (Stephens, 2007).

Summary
The external surface of the shell is the most likely source of *Salmonella* contamination of New Zealand eggs. The probability of contracting food poisoning directly from the egg appears to be low. However, cross contamination of the egg contents or other foods from the shell is possible if good food handling practices are not followed.

No data were found for *L. monocytogenes* and raw egg but it is a possible vehicle for listeriosis.

Cooked eggs	Well cooked (fried, scrambled, baked, poached, etc)	Cook well (firm yolks, firm scrambled eggs)

Food
See raw egg section above.

Hazards
See raw egg "Hazards" section above.

L. monocytogenes will grow in cooked egg products. Claire *et. al*, (2004) demonstrated the growth in hard-boiled eggs stored at 4°C, 8°C and 12°C. Counts increased from 10^2 to $>10^6$ CFU/g in 3–20 days, depending on the packaging atmosphere and storage temperature. Growth in mashed egg stored aerobically for 2 days at 4°C was approximately 1.2 $\log_{10}$ CFU/g.

Controls
International recommendations for the safe cooking of eggs are based on the destruction of *Salmonella* and are generally consistent; cook eggs thoroughly and cook foods containing eggs thoroughly. The white and yolk should be firm, not runny. Scrambled eggs should have a firm texture (Smith 1999).

Several studies have shown that this advice is valid irrespective of cooking method - hard boiled, poached, scrambled or microwaved (Chantarapanont *et al*., 2000; Bates and Spencer, 1995; Davis *et al*., 2008). Special care is required with scrambled eggs to ensure that all the egg mass comes into contact with the heat source long enough to be evenly heated (Davis *et al.,* 2008).

Surveys/Outbreaks
No surveys or outbreaks related to cooked eggs were located.

Summary
There are no issues identified for thoroughly cooked eggs eaten immediately after cooking.

Cooked egg foods, including intact hard boiled eggs, are however, perishable products and the generic recommendations regarding uneaten leftovers should apply. The current document gives no advice regarding leftover egg

5.4 Meat

Raw meat	Any raw meat, raw chicken or other poultry, beef, pork etc.	Don't eat; don't taste, or touch face, mouth or eyes while preparing; wash and dry hands well after touching raw meats

Food
Meat is generally skeletal muscle tissue and any attached animal rind, fat, connective tissue, nerve, blood and blood vessels (FSANZ Food Standard Code 2.2.1). The main commercial meat species include cattle, sheep, pigs and chickens. All fresh red meats have water activities (a_w) of >0.99 that provide a suitable environment for microbial growth (ICMSF, 1998). Animal tissue is essentially sterile prior to slaughter, but can become contaminated with pathogens and spoilage organisms as a result of primary processing operations including skinning and evisceration (ICMSF, 2005). Animal faecal material is a particularly rich source of pathogens which may include *E. coli* O157:H7, *Salmonella*, *Campylobacter* spp., *Listeria monocytogenes*, *Yersinia enterocolitica*, *Staphylococcus aureus*, the spore forming bacteria, viruses and protozoa (Moriarty *et al*., 2008).

Hazards
Although pathogens may be present on raw meat, their numbers are generally low.

In terms of *T. gondii*, no information is available on the frequency of contamination of red meat and red meat products with the parasite in New Zealand. Direct surveys in foods are difficult, as the methodology requires the concentration of the organism from muscle tissue prior to detection. This latter step has until recently required the use of live animals. The organism does not grow in foods or in other environments outside of a suitable host (Lake *et. al.,* 2008).

Controls
Following basic hygiene rules, when handling raw meat, avoid touching the face, mouth and eyes. Wash hands after handling raw meat to prevent cross-contamination.

Surveys/Outbreaks

There have only been a few studies of *L. monocytogenes* prevalence in New Zealand raw meat. None of the survey reports have included quantitative data (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*) in only two ovine samples (Hudson and Mott, 1994). A South Island mutton slaughterhouse survey found *L. monocytogenes* in seven of 218 samples from ovine carcasses and the immediate environment (Pociecha *et. al.*, 1991). *Listeria* was not found in freshly dressed carcasses (111 samples) or from meat contact surfaces (45) but was present in environmental samples from other parts of the plant (cold-rooms, soil and fodder). An earlier survey by Lowry and Tiong (1988) examined 142 samples from New Zealand cattle and sheep slaughter plants. Whole carcasses, boned meat, cuts, offal, hides, pelts, viscera, equipment, work surfaces and effluent were included in the survey. Seventy-five retail display meats of beef mince, pork cuts and poultry portions were also tested. *L. monocytogenes* was 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%). It was found in 48 - 92% of retail samples.

Several microbiological surveys have been conducted in New Zealand to determine the prevalence and concentration of other pathogens on raw chicken, beef, pork, lamb and mutton, and veal available for retail sale. The overall prevalence of *Salmonella* was 1.1% (Wong *et al*., 2007b), with the highest prevalence (3%) observed for chicken. Very low counts (<1 MPN/g) were observed for all but one positive samples. A similar retail survey for *Campylobacter jejuni* and *coli* (Wong *et al*., 2007a) revealed a far higher prevalence on chicken (89.1%), and a prevalence of 3.5 – 10% on other meats. The concentration of *Campylobacter* was <10 MPN g^{-1} for >90% of samples, with 0.5% >100 MPN/g. A comparative survey for shiga-toxin producing *E. coli* (STEC) and biotype 1 *E. coli* has recently reported an STEC prevalence of 14.7% in lamb and mutton, 6.5% in pork, 5.2% for beef and 2.2% for veal (Wong *et al*., 2009). Counts were again low, with only 2.5% of samples exceeding 1,000 CFU/g.

Summary

Advice regarding not eating or tasting raw meat is entirely appropriate (for any consumer) given the potential microbial hazards. Avoiding the face, mouth and eyes and washing hands after handling raw meat is appropriate to prevent cross-contamination. Given that the transfer of bacteria from wet hands is greater than from dry hands, hand drying is always good advice to include in conjunction with hand washing.

Cooked meats	Beef, pork, chicken, mince, sausages etc.	Cook thoroughly until piping hot throughout, and until juices run clear; eat while hot; never eat rare or undercooked meats; don't eat cold leftovers

Food
See raw meat definition in Food section above.

Hazards
See raw meat hazards defined in section above. There is a potential for *L. monocytogenes* to grow in refrigerated cooked meats (Appendix 1). Generation times range from 8.7 hours to 21.8 days depending on the meat type and the packaging conditions used (Hudson and Mott, 1993a; Nyati, 2000).

Based on the mean growth rate data for non-vacuum-packed meats, the growth of *L. monocytogenes* expected in 2 days at 5°C is 0.6 $\log_{10}$ CFU/g.

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.

"Cook thoroughly until piping hot throughout" is defined elsewhere in the NZFSA advice document as above 70°C. At these temperatures *L. monocytogenes* is rapidly inactivated (the D time at 70°C is about 10 seconds). Cooking meat to a temperature of at least 66°C (Goldsmid *et al*., 2003) or freezing it prior to cooking (Kotula *et al*., 1991) will inactivate *T. gondii* cysts which may otherwise be of concern, particularly for rare or undercooked meat (Smith, 1993).

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 minced 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.

Post-cooking contamination is of concern, especially for pathogens such as *L. monocytogenes*. The advice not to eat cold leftovers assumes post-process pathogen contamination is likely and sufficient time for growth to occur. This may be a more relevant to store-bought cooked meats where greater opportunities for cross-contamination exist, as opposed to home-cooked meat.

Surveys/Outbreaks
MacLeod (2000) reported an eight case *S.* Typhimurium PT135 outbreak linked to cold cocktail sausages from a New Zealand butchery. Outbreak investigations found several areas of concern including the cooking process. The sausages were cooked in boiling water but, because they floated, some parts of the sausage may not have reached sufficient temperatures to destroy the pathogen. There were also opportunities for cross-contamination by staff members in the chiller stores and on the display counter. With no physical barrier between raw chicken and the cooked sausages on display, the sausages were treated as a ready-to-eat product and handed out as cold snacks to customer's children.

No surveys were located for New Zealand.

Summary
There is a potential for *L. monocytogenes* to grow in refrigerated cooked meats and post-cooking contamination is of concern. The advice not to eat cold leftovers assumes post-process pathogen contamination is likely and sufficient time for growth to occur. This may be a more relevant to store-bought cooked meats where greater opportunities for cross-contamination exist, as opposed to home-cooked meat.

Cold cooked poultry	Any cold pre-cooked poultry (e.g. chicken, turkey)	Don't eat unless heated until piping hot

Food
See raw meat definition above.

Hazards
The same considerations as for cooked meats apply. Cross-contamination and undercooking are potential mechanisms for the contamination of cooked poultry. Recent USDA research has identified raw poultry as the primary source of L. monocytogenes in commercial chicken cooking plants (http://www.ars.usda.gov/is/pr/2010/100419.htm).

Controls
The same considerations as for cooked meats apply. The growth of *L. monocytogenes* on chilled cooked poultry expected in 2 days at 5°C is 0.5 $\log_{10}$ CFU/g (FDA/FSIS, 2003).

Surveys/Outbreaks
No issues located for New Zealand.

Summary
The same considerations as for cooked meats apply.

Processed meats	Ham, salami, luncheon, pâté, pastrami, biltong, or jerky (dried meat) etc.	Generally do not eat unless heated until piping hot *

* If these products are purchased in the manufacturer's original packaging small quantities can be eaten immediately after opening. Do not reseal and eat later, and do not eat if they have been repackaged in a deli or supermarket as they may become contaminated with pathogens during this process.

Food

Smallgoods are difficult to define due to the wide range of types available. This section of the NZFSA document includes raw cured shelf stable meats (e.g. fermented salami), dried meats (e.g. jerky), cooked perishable uncured meats (pâté) and cooked perishable cured meats (e.g. ham). Hazards and controls are different for each.

Hazards

Processed meats such as ham, luncheon and pâté do have a high water activity and near neutral pH, and will support the growth of *L. monocytogenes* and other pathogens (Appendix 1). Ham and pâté have proven to be particularly problematic items due to their long shelf life under refrigeration. The growth expected in pâté in 2 days at 5°C is 0.5 $\log_{10}$ CFU/g (FDA/FSIS, 2003). Growth expected in other processed meats in 2 days at 5°C is 0.6 $\log_{10}$ CFU/g.

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.

Controls

Salami and other high acid fermented meats rely on low pH (4.6-5.3) and a reduced water activity (a_w) of <0.95 for microbial stability (ICMSF, 1998). Alternatively either a pH of less than 4.5 or a water activity of <0.91 may achieve the same result (Ross and Shadbolt, 2001). 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. Standard 1.6.2 of the Food Standards Code (mandatory only for Australia) requires a water activity of ≤ 0.85 for dried meats. At this water activity, the growth of all bacterial pathogens of concern is controlled (NZFSA 2009c). Most commercially produced jerky products in New Zealand have a water activity of 0.75 to 0.80

Commercial processes used in the manufacture of cooked perishable cured and uncured meats will normally destroy pathogens (ICMSF, 1998). However, contamination can occur during packaging, slicing or handling. Products sold in the container or packing in which they are cooked generally have a longer shelf life than those that are repackaged but are still regarded as semi-perishable (i.e. require refrigerated storage).

Surveys/Outbreaks

An outbreak of non-invasive listeriosis, associated with ham and corned silverside, was reporting in New Zealand in early 2000 (Sim *et al*., 2002).

It is difficult to determine evidence for transmission in New Zealand because of the small number of cases and difficulty tracing food histories. A quantitative risk assessment was recently undertaken in Australia, where contamination and consumption prevalence is similar to New Zealand (Ross *et al.*, 2008). The authors concluded that ready-to-eat meats were responsible for up to 40% of cases of listeriosis. It is possible that the same situation applies in New Zealand.

Recent microbiological surveys conducted in New Zealand have revealed a low prevalence of *L. monocytogenes* in both packaged and unpackaged ham and pâté. During 2003/2004, in a survey of pre-packaged ham samples (Wong *et al.*, 2005), 104 samples were tested consisting of 16 brands. 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 seven samples contained other *Listeria spp*.

During 2004/2006, Cornelius *et al.,* (2008) conducted an unpackaged ham survey where the sampling was based on market share, i.e. 80% of samples were from supermarkets, 20% from other delicatessens. Samples were collected in Auckland, Wellington and Christchurch at a rate of 150 per year. 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%, with most concentrations < 50 CFU/g and two samples > 100 CFU/g.

A survey of nine brands of pâté on retail sale in Auckland and Christchurch was conducted in 2002 (Wong *et al*., 2005). *L. monocytogenes* (1,700 CFU/g) was isolated from one of the 300 samples tested. The concentration in the sample may have been higher but the aerobic plate count of 10^8 CFU/g inhibited further growth. Six samples of pâté contained *Listeria* species other than *L. monocytogenes,* two *L. welshimeri* (<50 and 450 CFU/g), and four *L. innocua* (<50, 50, 200 and 400 CFU/g).

Summary

The current NZFSA pregnancy advice document does not discriminate between dried meats such as biltong and jerky, which are microbiologically stable at ambient temperature, and processed meats with high water activity which will support growth of *L. monocytogenes*.

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 the pathogen in pre-packaged processed meats. The additional advice in the current NZFSA document regarding consumption of "small quantities" of these meats "if purchased in the manufacturer's original packaging" suggests greater assurance around the safety of these products. While this may be true for some products sold in the packaging in which they are cooked, *Listeria* contamination can occur in products which are sliced or handled prior to packaging.

5.5 Seafood

Raw fish	Any raw fish (including marinated raw fish)	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.

Hazards
Microbial contamination of raw fish occurs from environmental contamination which is determined predominantly by water temperature and pollution levels. The most common causes of foodborne disease associated with fish consumption include ciguatera intoxication via marine algae and the production of biogenic amines (scombroid) by spoilage bacteria (ICMSF, 2005). It is possible for bacterial pathogens such as *L. monocytogenes* to be associated with fish, but generally only where agricultural run-off is an issue. Growth of *L. monocytogenes* in 2 days at 5°C is 0.3 $\log_{10}$ CFU/g (based on averaged FDA/FSIS data for raw fish and raw crustaceans).

Overseas, the presence of *Clostridium botulinum* is of concern in marine sediments, but this is not the case in New Zealand (Fletcher *et al*., 2008).

Some fish species may be high in mercury, see Chapter 4.

Controls
Temperature control is key to the microbial quality of raw fish. No growth of *L. monocytogenes* is expected under frozen storage but slow growth is possible at refrigeration temperatures.

Surveys/Outbreaks
See section on sushi.

Summary
Growth of *L. monocytogenes* is possible at refrigeration temperatures.

Mercury concentration varies with fish species and advice given is species specific. See Chapter 4.

Raw shellfish	Any raw shellfish (including marinated raw mussels)	Don't eat

Food
Both saltwater and freshwater invertebrates are considered shellfish. Categories of shellfish include molluscs (e.g. clams, mussels, oysters, and scallops), crustaceans (e.g. shrimps, prawns, lobsters) and echinoderms (e.g. sea urchin).

Hazards
Viral diseases and algal shellfish toxins are typical hazards associated with the consumption of shellfish (ICMSF, 2005). Molluscan shellfish concentrate viruses such as Hepatitis A and *Norovirus* via feeding in contaminated waters. The concentrations of these contaminants will not be influenced by temperature abuse.

Cadmium levels in Bluff oysters and Queen scallops may be high.

No growth of *L. monocytogenes* was observed in oysters in 21 days at 4°C (Leung *et. al*, 1992)

Controls
Controls for filter feeding bivalve molluscs in New Zealand focus mainly on water quality in growing and processing areas. Factors relating to bacteriological and marine biotoxin hazards are regularly monitored

Surveys/Outbreaks
See results for sushi. There have been several outbreaks of *Norovirus* related to consumption of raw Korean oysters in New Zealand (manufacturer advised cooking before consumption). See http://www.nzfsa.govt.nz/publications/media-releases/2006-07-06.htm - accessed 26.04.10. Outbreaks related to locally produced product have also been reported.

Summary
As with all raw animal products it is not advisable for pregnant women to consume raw shellfish. In relation to cadmium, pregnant women are advised to minimise their consumption of Bluff oysters and Queen scallops.

Smoked fish, shellfish and crustacean	Chilled, pre-cooked fish, mussels, oysters, salmon, crayfish, prawns etc.	Don't eat unless heated until piping hot

Food
See fish and shellfish definitions.

Hazards
The main hazards associated with smoked fish product in New Zealand are *L. monocytogenes* and scombroid poisoning. Inadequate thermal processing, post-process contamination and temperature abuse may all contribute to the presence of pathogens in these foods (ICMSF, 2005). *Listeria* in particular is known to persist in seafood processing environments and is the pathogen of most concern in cooked

products where post-process handling such as slicing occurs. In addition, if not adequately controlled, smoking, particularly cold-smoking of fish products is not a lethal process for listeriae and subsequent growth of *L. monocytogenes* in smoked seafood is well established (Appendix 1). The potential for growth of *L. monocytogenes* in smoked seafood in 2 days at 5°C is 0.3 $\log_{10}$ CFU/g (FDA/FSIS, 2003).

Cadmium levels in Bluff oysters and Queen scallops may be high. Some fish species may be high in mercury, see Chapter 4.

Controls

Pre-cooking and chilling of fish, shellfish and crustaceans will destroy vegetative bacteria and extend the product's shelf life. Well controlled commercial hot-smoking regimes can eliminate both *L. monocytogenes* and the more heat-sensitive bacterial species responsible for histamine formation (Fletcher *et al.*, 2003).

Surveys/Outbreaks

Listeria spp. are present in finfish and shellfish. A survey in 1991 examined ready-to-eat fin fish and shellfish (Hudson *et al*., 1992). Eight of 25 (32%) of fin fish were positive for *L. monocytogenes.* For the shellfish results, 5/25 (20%) were positive. Fourteen of the shellfish were mussels, and of these, 5 (35.7%) were positive for *L. monocytogenes*. In another survey published in 1991, *L. monocytogenes* was isolated from retail packages of cold-smoked salmon. Overall, 77% of samples examined were positive for the pathogen (Fletcher and Rogers, 1991).

A survey of the microbiological quality of vacuum packed smoked salmon (Nortje *et al*., 2001) focused on end of shelf-life testing of hot- and cold- smoked salmon. 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 151 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 of the 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 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 new born 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 in November and December 1992 respectively when *L. monocytogenes* serogroup 1/2a was isolated from their blood. A food history of one brand of smoked mussels led to the testing of an unopened packet of mussels from the refrigerator of the mother. PFGE analysis found isolates from patients 1 and 2 were indistinguishable from isolates found in the mussels.

Two further cases, patients 3 and 4 also had the same PFGE pattern. Patient 3 had a food history of consuming mussels but did not specify the brand in question. Patient 4 had a history of unpasteurised milk consumption but not mussels. A microbiological link was therefore made between three clinical cases, a food source and the environment where the mussels were processed. The mother of the twins had

consumed a significant quantity of the mussels throughout her pregnancy in order to raise her iron levels.

Summary
Growth of *L. monocytogenes* is possible in these products.

In relation to cadmium, pregnant women are advised to minimise their consumption of Bluff oysters and Queen scallops.

Mercury concentration varies with fish species and advice to pregnant women is species specific (see Chapter 4).

Cooked fish, shellfish and crustacean	Freshly cooked fish, mussels, oysters, crayfish, scallops etc.	Cook thoroughly until piping hot throughout; eat while hot

Food
See earlier definitions of fish and seafood.

Hazards
Similar considerations as for cooked meats apply. Cadmium levels in Bluff oysters and Queen scallops may be high. Some fish species may be high in mercury, see Chapter 4.

Pre-cooked seafood available on retail sale may experience excessive handling and temperature abuse. FSANZ also recommend avoiding cooked peeled prawns in prawn cocktails, sandwich fillings and pre-made salads.

Growth of *L. monocytogenes* in cooked crustaceans in 2 days at 5°C is 0.8 $\log_{10}$ CFU/g (FDA/FSIS, 2003).

Controls
Adequate cooking will inactivate microorganisms, although it should be noted that heat will not inactivate algal toxins or histamine present as a result of environmental contamination and/or temperature abuse.

Surveys/Outbreaks
None located

Summary
There is potential for *L. monocytogenes* to grow in these products.

Pregnant women are advised to minimise their consumption of Bluff oysters and Queen scallops in relation to cadmium.

Mercury concentration varies with fish species. Advice to pregnant women with respect to fish consumption is species specific (see Chapter 4).

5.6 Vegetables, Salads and Fruits

Fruit	All fresh fruits	Wash and dry well just before eating
Vegetables	All fresh vegetables	Wash and dry well just before eating raw, or wash before cooking

Food
Fruits are the fleshy seed-associated structures of certain plants that are (usually) sweet and edible in the raw state. In culinary terms vegetables are usually defined as an edible or part of a plant other than a sweet fruit or seed.

Hazards
The initial microflora of vegetables and fruits is acquired from their interaction with soil, air, water, wildlife and insects (ICMSF, 2005; Beuchat, 2006a). However, a wide variety of pathogenic bacteria, protozoa and viruses may gain access to a particular produce item at different points in the food chain. With the exception of *B. cereus*, *C. 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. *T. gondii* has been implicated by several authors (Kapperud *et al*., 1996; Cliver, 2001; Goldsmid *et al.*, 2003) as a potential risk associated with vegetables, mainly through the potential for contamination of soil from the faeces of the definitive host, the cat family.

L. monocytogenes growth expected in fruit and vegetables in 2 days at 5°C is 0.1 $\log_{10}$ CFU/g (averaged data from FDA/FSIS, 2003).

Controls
Beyond washing, there is no decontamination step available at consumer level to reduce microbial loads on fresh produce (McIntyre *et al*., 2008).

Raw fruits and 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.

Surveys/Outbreaks
Whole undamaged fruits and vegetables are less likely to support the growth of bacterial pathogens than foods of animal origin (Appendix 1), but certain fruits such as tomatoes and melon varieties with higher pH values have been implicated in a number of outbreaks internationally (Harris *et al*., 2003). Vegetables implicated in overseas outbreaks include lettuce, spinach and spring onions. Internalisation of pathogens or cross-contamination of fruit flesh during cutting may both contribute to this problem. The FDA/FSIS (2003) information on *L. monocytogenes* in Appendix 1 shows that growth occurs in lettuce; however, some salad components such as carrots appear to be listericidal. Growth rates vary.

Surveys in New Zealand have failed to detect *E. coli* O157 and *Salmonella* spp. in 765 samples of hydroponically grown leafy vegetables, sprouted seeds and herbs (Graham and Dawson, 2002) and conventional and organically-grown lettuces

(Wong, 2003). *Salmonella* Typhimurium DT12a was however detected in one of 230 batches of organic apples (Wong, 2003) and more recently *Salmonella* Typhimurium phage type RDNC-May 06 was confirmed in two samples of domestic organic lettuce (McIntyre & Cornelius, 2009).

Only one confirmed produce-related outbreak – Hepatitis A in raw blueberries – has been documented in New Zealand (Calder *et al*., 2003). Other unconfirmed outbreaks of salmonellosis in New Zealand related to the consumption of raw carrots (Neuwelt *et al*., 2006) and water melon have been reported.

While underreporting may be an issue, the low number of reported outbreaks is in agreement with national and international survey data which indicate very low pathogen prevalence in fresh produce. Where pathogens have been detected, *Salmonella* has predominated (McIntyre and Cornelius, 2009).

Summary
Beyond washing and/or cooking, there is no decontamination step available at consumer level to reduce microbial loads on fresh produce.

Vegetables	Frozen vegetables	Cook; don't eat uncooked frozen vegetables

Food
Prior to packaging and freezing, most vegetables are blanched at 95°C to 99°C for 1 to 5 minutes (ICMSF, 2005). This is done primarily to inactivate degradative enzymes, but also serves a number of additional functions including fixing the colour of certain vegetables, inducing wilt and displacing entrapped air (Jay, 1992).

Hazards
Growth of *L. monocytogenes* in frozen vegetables is not expected. Frozen vegetables are considered to be a low risk food but survival of some microorganisms is inevitable

Controls
The blanching process is sufficient to reduce bacteria and viruses, although spore-forming bacteria will survive. Storage at -18°C will also inactivate protozoa and prevent the growth of bacteria either surviving blanching or recontaminating the product post-process. Time-temperature control and good sanitation practices are therefore the most important means to minimise microbial loads in frozen vegetables. As many vegetables are not cooked prior to blanching, additional cooking is required to render them organoleptically acceptable. This also serves to further inactivate any microorganisms present.

Surveys/Outbreaks
No issues identified with frozen vegetables

Summary
Growth of *L. monocytogenes* in frozen vegetables is not expected. Cooking frozen vegetables prior to consumption will inactivate any pathogens present.

Salads	Ready-made salads and coleslaws from delis, salad bars etc	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 meat or seafood.

Hazards
Historically, *L. monocytogenes* has been of primary concern in salads due to its ability to grow at refrigeration temperatures. The first link between listeriosis and food consumption was associated with coleslaw in Canada in 1981.

Based on the growth data in Appendix 1, deli-prepared salads are predominantly inhibitory to growth of the organism, depending on salad formulation and shelf life. According to FDA/FSIS (2003) data the estimated decrease over 2 days at 5°C is -0.3 $\log_{10}$ CFU/g. Only crab and shrimp salads (prepared in-store) allowed growth of the organism. Growth expected in 2 days at 5°C, growth is 0.2 $\log_{10}$ CFU/g.

Controls
A risk profile conducted by Lake *et al*. (2005) to assess the risks associated with *L. monocytogenes* in lettuce and cabbage-based salads without dressings concluded that these salads would be unlikely vehicles for infection in New Zealand, and that good agricultural and manufacturing practices, in conjunction with microbiological testing already being done by the industry, are the best means of managing this risk. Results from national surveys of produce to date support this finding.

Surveys/Outbreaks
A recent survey of retail ready-to-eat salads with dressings determined the prevalence, concentrations and genotypes of *Listeria* spp. and *L. monocytogenes*, and whether pH and temperature hurdles were adequate in controlling listerial growth. The following information has been provided by Dr TeckLok Wong (personal communication, November 2008).

RTE 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 (varieties outside of these descriptions). Salads under the various designations may have contained small amounts of cooked meats, cooked eggs, spices and fresh herbs. The prevalence of *Listeria* spp. in RTE salads was 21/302 (7%) (95% CI, 4.6 – 10.8) of which 14 (4.6%) (CI 2.6 - 7.7) were contaminated with *L. monocytogenes*. One sample of coleslaw contained 100 CFU/g of *L. monocytogenes* while another contained 30 CFU/g. The remaining 12 *L. monocytogenes*-contaminated samples contained <10 CFU/g of *L. monocytogenes*. Samples positive for other *Listeria* spp. also had <10 CFU/g.

Temperature and pH values recorded from the salads showed that these parameters were only partially adequate (using a temperature of 5ºC and pH 4.6 as hurdle references) in controlling growth of *Listeria* spp. in RTE salads over the shelf life at retail. It was concluded that better control of these hurdles by the suppliers and the

retailers could assist in preventing the potential re-growth of *Listeria* spp. in RTE salads.

These data are very similar to *L. monocytogenes* prevalence data of 4.8% and 3.8% respectively reported in a British survey of mixed raw vegetable salads containing cooked meat (76/1268) or cooked seafood (54/1418) (Little *et al*., 2005). Two salads containing chicken were found to have concentrations of ≥100 CFU/g *L. monocytogenes*, while all of the positive seafood salads were at concentrations <100 CFU/g. One salad in each category contained between 10 and 99 CFU/g. As with the New Zealand RTE salads survey, a variety of other food ingredients (pasta, rice, mayonnaise, eggs, etc.) were included in these salads.

Summary
L. monocytogenes has the potential to grow in these products due to their long shelf life. It is advisable for pregnant women to avoid eating open salads available from salad bars, delicatessen counters etc.

Current NZFSA advice is consistent with scientific evidence.

Salads	Home-made	Wash salad ingredients well before using

Food
See definition of salads above.

Hazards
L. monocytogenes and *T. gondii* are two of the major hazards associated with home salad vegetables. No data was available for growth of *L. monocytogenes* in home prepared salads.

Controls
As with fresh vegetables above, the only practical means of controlling the microbiological quality and safety of salad vegetables at the consumer level is to store them appropriately under refrigeration, avoid using damaged items and wash them well before use. Chlorine washing is used by the produce industry during processing of bagged lettuce, pre-cut stir-fry vegetables and sprouted seeds (McIntyre *et al*., 2008) but it is limited in its effectiveness and requires control of pH and temperature which would be unlikely to be achieved at a domestic level.

Surveys/Outbreaks
Salamina *et. al*., (1996) reported an outbreak of listeriosis attributed to the consumption of home prepared rice salad. The 39 cases were previously healthy, non-pregnant adults. Most had predominantly gastrointestinal symptoms but 4 were hospitalised with acute febrile illness. The epidemiologically implicated rice salad was not tested for *L. monocytogenes* but strains indistinguishable from the outbreak strain were found in other foods served at the function.

Summary
Pregnant women should be particularly cautious of garden soiled fresh salad vegetables, especially those consumed raw. Products must be well washed before consumption.

Herbs	Fresh home-grown or store bought	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.

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

Girardin *et al.,* (2005) used *L. innocua* as a surrogate (for *L. monocytogenes*) to assess quantitatively the survival and transfer of the pathogen on parsley under field conditions. *L. innocua* in the soil declined by 7 $\log_{10}$ cycles in 90 days and was detected on leaves in low numbers (> 0.04 MPN/g) during the first 30 days. The major cause of transfer of *L. innocua* from soil to parsley leaves was splashing due to rain and irrigation. As few as 1 CFU/g *Listeria* in soil led to contamination of parsley leaves. Internalisation of parsley through roots was not observed. The authors concluded that under their conditions of soils and climate, (in France), a delay of 90 days between application of potentially contaminated fertiliser and harvest should be sufficient to eliminate *L. monocytogenes*.

More recently, Dreux *et al.* (2007) investigated the population dynamics of *L. monocytogenes* and *L. innocua* in relation to the aerial surfaces of parsley. The authors concluded that even with a high inoculum and under protected conditions such as plastic tunnels, populations of *L. monocytogenes* on the surface of parsley in the field would decrease by several $\log_{10}$ cycles within two days. Direct contamination of aerial surfaces of parsley with *L. monocytogenes* (i.e. via contaminated irrigation water) will not lead to contaminated produce unless it occurs very shortly before harvest.

Controls
Washing and cooking are the only practical decontamination opt ons for consumers.

Surveys/Outbreaks
Surveys in New Zealand have failed to detect *E. coli* O157 and *Salmonella* spp. in fresh herbs (Graham and Dawson, 2002).

Summary
The same considerations as for raw vegetables and salad ingredients apply.

Store bought herbs may be chlorine rinsed before sale but, other than cooking, washing before use is the only practical decontamination option available to consumers.

Herbs	Dried	Cook thoroughly

Food
See definition of herbs above. Spices have also been included in the discussion below.

Hazards
Herbs are not major contributors to foodborne illness, but some may contain pathogens (ICMSF, 2005).

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 (ICMFS, 2005). These are most likely to be spore forming bacteria and moulds, but pathogens may also be present.

Some spices, such as pepper, may be used on food that is eaten raw or added to food after cooking. Spore forming bacteria such as *B. cereus* and *C. perfringens* are commonly found in spices, but usually in small numbers (ICMSF, 2005). To cause illness they have to multiply to $10^5 – 10^6$ CFU/g of food to which the spice has been added. As the spores of these organisms may survive cooking, generic considerations concerning the opportunity for pathogen growth to occur during storage apply.

Growth of *L. monocytogenes* is not expected in dried herbs and spices.

Controls
Herbs and spices may be treated to reduce microbial concentrations using irradiation, steam pasteurisation, etc. While pathogens would normally be expected to be present at low to negligible concentration, cooking is probably the most effective method of control at consumer level.

Imported pepper, paprika and cinnamon are prescribed foods and, except for product imported from Australia, subject to NZFSA import clearance requirements with respect to *Salmonella*.

Surveys/Outbreaks
Salmonella have been found in a number of spices. A nationwide outbreak of salmonellosis (1,000 cases) in Germany in 1993 was traced to contaminated paprika used to powder potato chips. Several different serotypes of *Salmonella* were detected in both the food and in patients (ICMSF, 2005). Concentration of the pathogen in the implicated food appeared to be very low (as few as 0.04 per gram). Another *Salmonella* outbreak implicating spices is currently under investigation in the USA (http://www.cdc.gov/salmonella/montevideo/index.html). A total of 252 individuals across 44 states have been infected with the outbreak strain (*S.* Montevideo). This strain has also been identified in samples of black and red pepper used in the manufacture of Italian-style meat products. *S.* Senftenberg has also been found in retail foods and nine patients during the outbreak investigation.

A survey of 2965 dried herb and spice samples from retail and production premises was undertaken in the United Kingdom in 2004 (Sagoo *et al*. 2009). *Salmonella* spp. were detected in 1.5% and 1.1% of dried spices and herbs sampled at production and retail, respectively. Overall, 3.0% of herbs and spices contained high counts of *B.*

cereus (1%, $\geq 10^5$ CFU/g), *C. perfringens* (0.4%, $\geq 10^3$ CFU/g) and/or *E. coli* (2.1%, $\geq 10^2$ CFU/g). The authors identified a potential public health risk from dried herbs and spices used in products without further processing and emphasised the importance of correct food handling practices by end users

A 2007 survey in Australia examined a range of spices, including whole, ground and mixed products, *Salmonella*, *B. cereus* and *C. perfringens*. Samples were collected from supermarkets, small retailers, health food shops, market stalls and other retail outlets or upon import into Australia. Of the 217 samples analysed, no *Salmonella* spp. were detected but many of the samples (34%) contained low levels of *C. perfringens* (<100 CFU/g). *B. cereus* was detected in 69% of the samples. While concentrations were generally low, higher levels were found in a small number of samples (fennel and caraway seeds ($<10^3$ CFU/g, ground ginger, cumin, fennel and nutmeg ($<10^4$ CFU/g), and Tandoori spice mix ($10^2 - 10^5$ CFU/g). The survey report concluded that the risk to public health associated with consumption of spices in Australia was low. (http://www.health.vic.gov.au/foodsafety/downloads/survey_spices.pdf)

Summary
Growth of *L. monocytogenes* is not expected in dried herbs and spices but other pathogens may be present.

No issues are identified for adequately cooked product.

The current NZFSA document refers only to herbs. The same considerations apply to spices.

5.7 Miscellaneous

Leftovers	Cooked food	Store uneaten leftovers covered in fridge; eat within two days; never eat cold leftovers – always rehea until piping hot (over 70°C)

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

Hazards and Controls
Growth rate of *L. monocytogenes* is dependent on food type but is irrelevant if the food is adequately reheated immediately prior to consumption. *L. monocytogenes* is rapidly inactivated at temperatures above 70°C, depending on the properties of the food. The D value at 70°C is approximately 10 seconds.

Tinned foods	Tinned fruit, vegetables, fish, seafood, meat, sauces etc	Eat immediately after opening tin (hot or cold); store uneaten leftovers covered in fridge and eat within two days. Remove from can for storage.

Food
Tinned (or canned) foods are commercially sterile shelf-stable products which have undergone a heat treatment to ensure the destruction of all viable organisms.

Hazards
The extent of heat treatment is dependent on the pH of the food and its ability to support the growth of the anaerobic spore-former *C. botulinum*.

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

Controls
High acid (low pH) foods undergo relatively mild heat treatments to prevent spoilage while low acid foods (pH >4.6) such as fish are processed using a more severe 12D "botulinum cook" to ensure their safety (Jay, 1992). Spoilage of canned foods can sometimes occur due to under processing, inadequate cooling, seam leakage and pre-process spoilage (Jay, 1992).

Microbial stability of non-retorted canned products such as condensed milk, anchovies and olives is achieved primarily by low water activity. Mild heat treatment and low pH may also be used as additional hurdles for some products.

Refrigerated storage of canned leftovers reduces possibility of pathogen growth. Removing food from open cans prior to refrigerated storage may serve two separate purposes. It may help to avoid cross-contamination due to inadequate sealing of the can, but perhaps more importantly will avoid leaching of tin from the can into the food in the presence of oxygen (http://www.inchem.org/documents/jecfa/jecmono/v46je12.htm#_46122210). This generally occurs with acidic foods which can corrode the tinplate, and consumption can result in nausea, vomiting, abdominal pain, diarrhoea and headaches. However, there is little evidence that this is an issue of greater concern to pregnant women in particular (http://www.inchem.org/documents/jecfa/jecmono/v46je12.htm#_46122210).

Surveys/Outbreaks
No surveys or outbreaks located, other than reports of botulism.

Summary
No issues identified for product in undamaged cans. Growth of *L. monocytogenes* in leftovers is dependent on food type.

Sauces and dressings	Salad dressings (oil and vinegar), bought mayonnaise, tomato sauce etc	Store in fridge once opened, check maximum storage time.

Food

Mayonnaise is defined by Codex as an emulsion of vegetable oils in an aqueous phase of vinegar. The oil-in-water emulsion is stabilised by egg yolk (ICMSF, 1998). Other ingredients may include sugar, salt, herbs, spices, dairy products and other acidifying agents. The vegetable oil content is greater than 65% and pH between 3.6 and 4. Total fat content (oil plus egg yolk) is between 70% and 80%. Egg yolk may be replaced with modified starch in low fat varieties. The aqueous phase may contain up to 12% salt and 10% sugar (except in low-calorie formulations).

Salad dressings are similar to mayonnaise but typically have a smaller fat phase (30% oil). They contain cooked starch which contributes to their consistency. The pH is typically between 3.0 and 4.2 and either vinegar or lemon juice can be used for acidification. Sugar and salt contribute little to microbial stability due to the dilution effect of the larger aqueous phase water in dressings.

Hazards

Salmonella has been the pathogen of concern in many of the outbreaks reported overseas. While some early episodes implicated commercial product, almost all recent outbreaks have been associated with the use of contaminated eggs in product prepared in the home, restaurants or institutions.

The absence of reports of foodborne illness implicating commercially prepared acidic dressings and sauces is evidence of their safety in recent years (ICMSF, 2005; Smittle, 2000).

Beuchat *et. al.* (2006b) studied the death rates of *Salmonella*, *E. coli* O157 and *L. monocytogenes* in commercially produced, shelf stable, ranch and blue cheese salad dressings (mayonnaise-based dairy products). 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) numbers of *L. monocytogenes* and stored at 25°C. While *L. monocytogenes* exhibited the highest tolerance among the three pathogens to the acidic conditions of the dressings, numbers fell to undetectable concentrations 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 growth of *L. monocytogenes* is expected in properly formulated commercial product.

Controls

The microbial stability of commercially prepared acidic dressings and sauces is primarily due to toxic effect of acetic and to a lesser extent lactic and citric acids. According to Smittle (2000) the highest manufacturing target pH for dressings and sauces is 4.4, which is below the reported inhibitory pH of 4.5 for foodborne

pathogens in the presence of acetic acid. Citric acid, at the same pH, is a less effective inhibitor of both *Salmonella* and *Listeria* (ICMSF, 2005).

Surveys/Outbreaks

No surveys were located for New Zealand. In the United Kingdom, 1208 sauce samples were examined for *E. coli*, *S. aureus, Salmonella* spp, and *Bacillus* spp. during a survey of kebab takeaway restaurants (Meldrum *et al*., 2007). Fifty five (4.7%) of the sauce samples were of unsatisfactory microbiological quality. A further seven were of unacceptable quality (due to *Bacillus* spp. ($>10^5$ CFU/g) or the presence of *Salmonella* Agbeni (1 sample)). More samples of chilli sauce (8.7%) were of unsatisfactory or unacceptable microbial quality than any other sauce types.

No outbreaks associated with acidic sauces were located, other than an outbreak in the USA associated with canned chilli sauce and botulism. Eight cases were reported, and all eight had eaten hot dog chilli sauce the day before onset (CDC, 2007).

Summary

No growth of *L. monocytogenes* is expected in properly formulated commercial product. It is also likely that pathogens that may contaminate dressings after opening would die over time unless spoilage organisms (such as mould) grow and alter the pH.

Home made product is of greater concern should the pH reached be inadequate.

Sushi	Store bought (all types – even without raw seafood)	Don't eat

Food

The use of raw seafood and fish has been discussed above. Sushi is made with vinegared rice, seaweed, vegetables, and often raw fish.

Hazards

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. Inadequate temperature control of cooked rice may also permit the production of *B. cereus* toxin.

Refer to data for raw fish above in relation to *L. monocytogenes* and mercury.

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, although not always used by sushi bars, etc. as it is perceived to have a negative impact on the sensory properties of the sushi (http://www.nzfsa.govt.nz/consumers/food-safety-topics/foodborne-illnesses/sushi/factsheet-sushi.htm).

Surveys/Outbreaks

A survey of 79 sushi samples collected from 30 premises in Wellington and Christchurch was conducted over the summer of 1998/1999 (Table 5). A questionnaire was completed on the practices of the premises at the same time (Hough, 2000). Sushi flavours were limited to salmon (raw and smoked), tuna and vegetarian. Of the 79 samples collected, 15 did not comply with microbiological reference criteria. Control point failures were identified in over a third of the

premises. These included: utensils not sanitised regularly, preparation surface not sanitised regularly, potential for environmental contamination and cross contamination from raw ingredients possible.

Table 5: Microbial survey of sushi (Wellington and Christchurch 1998/99)

Flavour	No. of samples collected	No of alert samples (%)
Smoked salmon	12	1 (8%)
Raw salmon	14	4 (29%)
Salmon (not identified further)	8	2 (25%)
Vegetarian	26	5 (19%)
Tuna	18	2 (11%)
Prawn	1	1 (100%)
TOTAL	79	15 (19%)

Nine of the samples had high aerobic plate counts (35°C), 9 had detectable *E. coli* and 5 had high faecal coliform results. *Salmonella* and *Vibrio* spp were not in any of the samples. High concentrations of coagulase positive staphylococci were found in follow up samples of tuna sushi taken from one of the premises. Samples were not examined for *Listeria*

Summary
Mercury concentration varies with fish species and advice to pregnant women with respect to fish consumption is species specific (see Chapter 4). There is the potential for *L. monocytogenes* growth in this product.

Sushi	Home-made	Use freshly cooked rice, and don't use raw or cold cooked meat or seafood; eat immediately; don't eat leftovers.

Food
Refer above.

Hazards
There is no specific data available for the growth of *L. monocytogenes* in home prepared sushi.

Controls
In the absence of raw or smoked seafood, there is no reason why home-made sushi would be a problem from a food safety perspective assuming that the rice is freshly cooked. Generic considerations concerning the opportunity for pathogen growth or contamination to occur during storage apply.

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

Summary
Pregnant women are advised not to consume raw or smoked seafood. There are no issues identified for home-made sushi if made with cooked ingredients and consumed immediately.

Reheating is not an option for this product. Generic considerations regarding leftovers apply. Current NZFSA advice is appropriate.

Stuffing	Stuffing from chicken or turkey	Don't eat unless stuffing is cooked separately (in a dish); eat hot; store uneaten leftovers in fridge and eat hot within two days

Food
Stuffing is a seasoned mixture of ingredients used to fill cavities in meat and vegetables. It is usually associated with breadcrumb based ingredients stuffed into poultry (e.g. chickens or turkeys).

Hazards
Stuffing may become contaminated with pathogens (e.g. *Listeria*, *Campylobacter* and *Salmonella* spp) through cross contamination from the raw poultry. The dry mixture may also contain spore forming bacteria such as *B. cereus*. 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 and growth of *C. perfringens*.

No data is available for the potential for growth of *L. monocytogenes*.

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

Chang *et al*. (1998) determined time-temperature histories and cooking times for turkeys oven roasted at 162.8°C. 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). The slowest heat point was found to be the stuffing geometric centre and the wing joint. The difference in cooking time for unshielded turkeys was 155 min + 11min/kg unstuffed and 200 min + 8.8 min/kg stuffed. Shielding prolonged cooking times.

Survival of *C. perfringens* during baking and holding of turkey stuffing was demonstrated in a study by Woodburn and Kim (1966). *C. perfringens* spores and vegetative cells were inoculated into bread and onion stuffing used to season eight lightweight and eight heavyweight turkeys. Turkeys were cooked to "doneness" at three different oven temperatures. Endpoint temperatures in the thigh-body junction were 73°C in a 94°C oven and 82°C in 163°C and 232°C ovens. The temperature of the stuffing at the endpoint was 73°C (after 987 minutes cooking at 94°C), 63°C (238

minutes at 82°C) and 55°C (161 minutes at 232°C). Vegetative cells reduced rapidly during cooking but spore concentrations remained constant until the temperature of the stuffing rose above that permitting growth. Cells of a strain of *C. perfringens* considered to be not especially heat resistant survived all cooking regimes. Storage of the stuffing resulted in marked reduction in numbers after 6 days at 5°C and increases after 24 hours at 23°C.

Surveys/Outbreaks
No surveys were located specifically related to stuffing.

Summary
There are no issues identified for adequately heated product that has been cooked separately. Pathogens may survive and subsequently multiply if cooking and cooling procedures are not managed appropriately.

No data is available for potential growth of *L. monocytogenes*.

Hummus	Store-bought or home-made	Don't eat

Food
Hummus, a traditional and popular Middle Eastern food, is made from cooked chickpeas with tahini, garlic, lemon juice or citric acid, and seasonings. It is eaten as a spread or dip without further cooking.

Hazards
Hummus is considered a suitable growth medium for a wide range of microorganisms and contamination can be introduced from raw ingredients, manufacturing utensils, food handlers, and the environment.

Based on international recall data it is clear that hummus can become contaminated during processing, and can allow the growth of *L. monocytogenes* (and potentially other pathogens if temperature abused).

Tahini (crushed sesame seed paste) has been implicated in outbreaks of foodborne disease in New Zealand and product has been recalled due to contamination with *Salmonella* (http://www.nzfsa.govt.nz/processed-food-retail-sale/recalls/products/2008/recalled-food-productscooksfg.htm).

Raw garlic may also contribute microorganisms but it also possesses antimicrobial activity against *L. monocytogenes* and *C. botulinum* due to allyl sulfonyl and allyl sulphide (ICMSF, 2005). However, it is unclear as to how much of the antimicrobial is present and active in typical product formulations.

Controls
According to Yamani and Al-Dababseh (1994), the pH of traditionally produced hummus is affected by the buffering capacity of the chickpea proteins, lemon juice or citric acid ingredients, and microbiological activity, and is expected to be approximately pH 5.

Imported tahini and crushed sesame products are prescribed foods and, except for product imported from Australia, subject to NZFSA import clearance requirements.

Surveys/Outbreaks

A 1999 survey in New Zealand examined 10 batches (50 samples) of commercially prepared hummus (Wilson, 2000). Samples from four batches had high aerobic plate counts (up to 10^7 CFU/g) but *L. monocytogenes* was not detected in any sample. 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.

A more recent survey of three brands of commercially produced hummus in New Zealand reflected greater control. All three contained citric acid (lemon juice and sorbic acid was also present in two of the brands. The pH was between 3.9 and 4.5. No growth of *L. monocytogenes* was observed up to 55 days at 7.7°C (JA Hudson, *pers. comm*.)

Summary

Growth of *L. monocytogenes* is not expected in properly formulated commercial product. However there is a potential for growth in home made hummus unless adequately acidified

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APPENDIX 1: USDA FDA/FSIS QUANTITATIVE RISK ASSESSMENT (2003)

COMPILATION OF DATA: INACTIVATION, SURVIVAL OR GROWTH OF *L. MONOCYTOGENES* IN FOOD

MILK

Growth observed in all types of treated and untreated 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

EGR = Exponential Growth Rate, GT = Generation Time, Log = Log_{10} cfu/g

CHEESE

Fresh soft cheese

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

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	4	2.0 log **decr**. in 30 days 0.8 log **decr** in 10 days 0.3 log in 30 days	-0.067 -0.080 0.010	Genigeorgis *et al.*, 1991
Queso Ranchero	4	0.3 log in 18 days	0.017	
Queso Panella	4	2.13 log in 10 days 0.21 log in 30 days 0.44 log in 36 days	0.212 0.007 0.012	

EGR = Exponential Growth Rate, GT = Generation Time, Log = Log_{10} cfu/g

Soft unripened cheese

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

Type	Temp. (°C)	Growth rate	EGR at 5°C ($\log_{10}$ cfu/day)	Reference
Cottage cheese (multiple brands)	8	0.59 log in 18 days	0.015	Genigeorgis *et al.*, 1991
		1.87 log **decr**. in 36 days	-0.024	
		0.42 log in 24 days	0.007	
		1.13 log in 8 days		
		1.87 log **decr.** in 8 days	0.064	
			-0.106	
		0.39 log in 24 days		
	4	0.34 log in 24 days	0.023	
		0.41 log in 16 days	0.020	
		0.94 log in 36 days	0.036	
		1.87 log **decr**. in 8 days	0.037	
			-0.333	
Teleme	8	2.2 log in 36 days	0.028	Genigeorgis *et al.*, 1991
	4	0.42 log **decr**. in 36 days	-0.017	
Ricotta (3 brands)	8	2.11 log in 8 days	0.120	Genigeorgis *et al.*, 1991
		1.75 log in 6 days	0.132	
		1.88 log in 8 days	0.106	
	4	1.53 log in 30 days	0.072	
		3.58 log in 36 days	0.141	
		1.97 log in 22 days	0.127	
Cream cheese	8	2.0 log **decr**. in 30 days	-0 030	Genigeorgis *et al.*, 1991
	4	2.0 log **decr**. in 36 days	-0.079	
		>2.0 log **decr**. in 36 days	-0.056	
Cream cheese	4	2 log in 2 days	1.423	Cottin *et al.*, 1990
Ricotta(whey)	5	16.2 – 20.2 hours GT	0.397	Papageorgiou *et al.*, 1996
	12	5.1 – 5.8 hours GT (generation time)	0.292	
Cottage cheese	4	2.0 log in 40 days	0.071	Chen and Hotchkiss, 1993
	7	2.4 log in 10 days	0.137	
Cottage cheese	5	2 log in 22 days	0.091	Fedio *et al.*, 1994
Cottage cheese	'refrigerated'	0.5 – 1.5 log **decr**. in 1 to 5 weeks	-0.048	El-Shenawy and Marth, 1990
	6	1 log **decr**. in 21 days	-0.035	

Soft ripened cheese

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

Type	Temp. (°C)	Growth rate	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, 1989
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, 1987a
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,1989
Camembert	14 7 4	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	

EGR = Exponential Growth Rate, GT = Generation Time, Log = $\log_{10}$ cfu/g

Semi-soft Cheese

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

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	

EGR = Exponential Growth Rate, GT = Generation Time, Log = Log_{10} cfu/g

Hard Cheese

Seven studies provided data on growth and survival. Of 11 data points available, 10 indicated declines in *L. monocytogenes* populations with an estimated growth rate range of -0.003 to -0.228 $\log_{10}$ cfu/day at 5°C. Growth was only observed in Stilton- categorised as semi-soft by FSANZ (Whitley *et al.,* 2000), the authors commenting that film wrapping (and modified atmospheric packaging- with or without oxygen) was insufficient to control growth in this cheese. The storage times for hard cheese in these studies were 30 to 150 days.

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, 1987b
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	>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

EGR = Exponential Growth Rate, GT = Generation Time, Log = $\log_{10}$ cfu/g

* Survival or growth observed, **complete inactivation 66-80 days ripening at 24°C)

Processed cheese
All six data sets show a decline.

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 & Marth, 1988
Cold pack (acidified or preservatives)	3	1.0 log **decr.** in 60 days	-0.017	Ryser & Marth, 1988

EGR = Exponential Growth Rate, GT = Generation Time, Log = Log_{10} cfu/g

YOGHURT
Declines in population observed for all papers.

Temp. (°C)	Growth rate	EGR at 5°C (log_{10} cfu/day)	Reference
4°C	decr. ~1 log (surv. 4-12 days)	-0.18	Schaack& Marth *al.*, 1988
4°C	decr ~ 2 log (surv. 21-24 days)	-0.12	Choi *et al.*, 1988
Not stated	decr. ~ 2 logs in 3-6 days	-0.40	Siragusa & Johnson, 1988

EGR = Exponential Growth Rate, GT = Generation Time, Log = Log_{10} cfu/g

ICE CREAM
No growth observed of *L. monocytogenes* in ice cream.

Type	Temp. (°C)	Growth rate	EGR at 5°C (log_{10} cfu/day)	Reference
Ice cream	-18 to -25°C	0 logs in 2 months	0.000	Berrang *et al.*, 1988
Soft serve	-18°C	0 logs in 3 months	0.000	Dean & Zottola, 1996
Ice cream	-18°C	0 logs in 14 weeks	0.000	Palumbo & Williams, 1991

EGR = Exponential Growth Rate, GT = Generation Time, Log = Log_{10} cfu/g

PROCESSED MEATS

Growth observed in two papers for pâté and in the deli meats listed below.

Type	Temp. (°C)	Growth rate	EGR at 5°C (log_{10} cfu/day)	Reference
Pâté	4°C	4 log in 680 hours	0.143	Hudson & Mott, 1993a
Pâté	5°C	0.361 log in 1 day	0.361	Farber *et al.*, 1995
Bologna	4.4°C	1 to 2 log in 14 days	0.131	Glass and Doyle, 1989
Corned beef	4.8°C	0.13 log in 1 day	0.130	Grau and Vanderline, 1992
Vac.packed ham	5°C	0.30 log in 1 day	0.300	Grau and Vanderline, 1992
Cooked ham	4.4°C	2 to 3 log in 28 days	0.131	Glass and Doyle, 1989
Cooked ham	7°C	6 log in 35 days	0.098	Beumer *et al.*, 1996
Vac packed ham Cooked ham	8 C 8°C	0.16 log per day 0.2 log per day	0.0725 0.091	Bredholt *et al.*, 1999

EGR = Exponential Growth Rate, GT = Generation Time, Log = Log_{10} cfu/g

COOKED MEATS

Several papers show growth of *L. monocytogenes* in cooked meats.

Type	Temp. (°C)	Growth rate	EGR at 5°C (log_{10} cfu/day)	Reference
Cooked chicken	4.5°C	19.5 days GT	0.438	Nyati, 2000
Beef sirloin	4.5°C	21.8 GT	0.392	Nyati, 2000
Roast beef	5°C 10°C	5 log in 15 days 5 log in 6 days	0.333 0.254	Grant *et al.*, 1993
Sliced chicken	4.4°C	4.15 log in 14 days	0.364	Glass and Doyle, 1989
Vac. Packed chicken	4.4°C	5.90 log in 14 days	0.517	Glass and Doyle, 1989
Chicken breaded fillets	5°C	0.9 log in 6 days	0.150	Siragusa & Johnson, 1988
Turkey sliced	4.4°C 4.4°C	2 log in 14 days 3.11 log in 28 days	0.175 0.136	Glass and Doyle, 1989
Turkey sliced	4.4°C 4.4°C	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°C	5.09 log in 14 days	0.446	Glass and Doyle, 1989
Cooked beef (aero)	5°C	11.9 hours GT	0.607	Hudson and Mott, 1993a
Cooked beef (vac)	5°C	8.7 hours GT	0.83	Hudson and Mott, 1993a

EGR = Exponential Growth Rate, GT = Generation Time, Log = Log_{10} cfu/g

RAW SEAFOOD

Little or no growth observed in raw seafoods.

Type	Temp. (°C)	Growth rate	EGR at 5°C (log_{10} cfu/day)	Reference
Fresh trout	4°C	1 log in 15 days	0.100	Fernandes *et al*., 1998
Catfish	4°C	2 log in 15 days	0.185	Fernandes *et al*., 1998
Raw shrimp, crab surimi and whitefish	7°C	GT in 12 hours	0.342	Lovett *et al*., 1990
Raw oysters	4°C	No growth in 21 days	0.000	Kaysner *et al*., 1990
Catfish	4°C	1-1.5 log in 12 days	0.133	Leung *et al*., 1992
Raw shrimp & fin fish	Ice-chest	No growth	0.000	Shineman & Harrison, 1994
Raw shrimp and fin fish	Ice-chest	No growth	0.000	Harrison *et al*., 1991

EGR = Exponential Growth Rate, GT = Generation Time, Log = Log_{10} cfu/g

SMOKED SEAFOOD

Growth observed in all types of smoked seafoods.

Type	Temp. (°C)	Growth rate	EGR at 5°C (log_{10} cfu/day)	Reference
Cold-smoked salmon	4°C 8°C 4°C 8°C	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°C 8-10°C	0.5 log in 20 days 6.5 log in 20 days	0.035 0 120	Jemmi and Keusch, 1992
Cold-smoked salmon	5°C 10°C	4 log in 650 hours 4-4.5 log in 125 hrs	0 148 0.249	Hudson and Mott, 1993b
Smoked salmon	4°C 10°C	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°C	2.8 log in 21 days	0.190	Dillon and Patel, 1993
Smoked salmon (26-30°C)	4°C 10°C	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°C 5°C 10°C 10°C	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°C 10°C	4 log in 50 days 4.5 log in 15 days	0.080 0.092	Pelroy *et al*., 1994b
Cold-smoked salmon	5°C 5°C 10°C 10°C 10°C	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°C 8°C	1 log in 10 days 3 log in 14 days	0.142 0.097	Rosso *et al*., 1996
Cold-smoked salmon	5°C	5 log in 9 days	0.556	Nilsson *et al*., 1997

COOKED READY-TO-EAT CRUSTACEA

Growth observed in all three papers.

Type	Temp. (°C)	Growth rate	EGR at 5°C (log_{10} cfu/day)	Reference
Pasteurised crabmeat	5°C	GT in 21.8 hours	0.343	Rawles *et al.*, 1995
Cooked lobster, shrimp, crab and smoked fish	4°C	2-3 log in 7 days	0.508	Farber, 1991
Pasteurised crabmeat	5°C	3 log in 10 days	0.300	Buchanan and Klawitter, 1992

EGR = Exponential Growth Rate, GT = Generation Time, Log = Log_{10} cfu/g

VEGETABLES AND FRUIT

Growth generally for *L. monocytogenes* in lettuce. Some salad components appear to be listericidal, such as lamb's lettuce, mung beans, carrots and chopped tomatoes. Various growth rates were observed in other selected vegetables.

Type	Temp. (°C)	Growth rate	EGR at 5°C (log_{10} cfu/day)	Reference
Lettuce, whole RTE	5°C	0.0-0.3 log in 7 days	0.043	Steinbrugge *et al.*, 1988
	12°C	0.0-2.03 log in 7 days	0.004	
Lettuce whole RTE, sealed	25°C	0.0-0.31 log in 7 days	0.002	
Lettuce whole RTE, open	25°C	0.0-0.35 log in 7 days	0.002	
Lettuce, shredded	5°C	0.0-0.1 log in 15 days	0.007	Beuchat and Brackett, 1990a
Lettuce, shredded	10°C	1.5-2.0 log in 3 days	0.204	
Lettuce, whole	10°C	1.0 log in 15 days	0.067	
Lettuce, butterhead	10°C	1.5 log in 7 days	0.065	Nguyen and Carlin, 1994
Lettuce, lamb's	10°C	1.0 log **decr.** in 7 days	-0.044	
Lettuce	8°C	1.5 log in 7 days	0.097	Francis and O'Beirne, 2001
Endive, broad leaved	10°C	1.0 log in 7 days	0.044	Carlin *et al.*, 1996
Endive, broad leaved	10°C	1.5 log in 7 days	0.065	Nguyen and Carlin, 1994
Endive, curly-leaved	10°C	0.5 log in 7 days	0.022	

Type	Temp. (°C)	Growth rate	EGR at 5°C (log_{10} cfu/day)	Reference
Tomatoes	10°C 21°C	No growth (death in chopped tomatoes) Growth	0.0 -	Beuchat and Brackett, 1991
Carrots, whole and shredded	5°C 15°C	No growth up to 7 days No growth up to 7 days	0.00 0.00	Beuchat and Brackett, 1990b
Cabbage, raw, shreds	5°C	4 log in 10 days	0.400	Beuchat *et al.*, 1986
Broccoli	4°C 15°C	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°C 15°C	$\leq$ 0.25 log in 14 to 21 days 3.0 log in 4 days	0.020 0.109	Berrang *et al.*, 1989
Swede	8°C	1.75 log in 12 days	0.066	Francis and O'Beirne, 2001
Bean sprout	8°C	1.75 log in 8 days	0.099	Francis and O'Beirne, 2001
Salads, mixed, prepacked incl fruit and nuts	4°C	0.30 log in 4 days	0.106	Sizmur and Walker, 1988
Fruit				
Orange juice	4°C	1.0 log in 35 days (pH 5.0)	0.041	Parish and Higgins, 1989
Apple slices (fresh cut)	5°C 10°C	0 log in 6 days 2 log in 6 days	0 0.102 (air)	Conway *et al.*, 2000
Apple slices (fresh cut)	5°C 10°C	0 log in 4 days 2.8 log in 10 days	0 0.086 (microaero)	Conway *et al.*, 2000

EGR = Exponential Growth Rate, GT = Generation Time, Log = Log_{10} cfu/g

DELI-SALADS

A decrease in populations of *L. monocytogenes* recorded in most deli-type salads except for a crab and shrimp salad.

Type	Temp. (°C)	Growth rate	EGR at 5°C (log_{10} cfu/day)	Reference
Crab salad (store prep.)	5°C	1 log in 10 days	0.100	Eblen, 2002
Shrimp salad (store prep.)	5°C	2 log in 14 days	0.143	
Shrimp salad (plant prep)	5°C	No change	0.0	
Chicken salad: (store prep); (plant prep.)	5°C	 No change 3 log **decr.** in 18 days	 0.0 -0.167	
Pototo salad: (store prep); (plant prep)	5°C	 2 log **decr.** in 13 days 3 log **decr.** in 10 days	 -0.154 -0.333	
Coleslaw: (store prep); (plant prep)	5°C	 3 log **decr.** in 13 days 3 log **decr**. in 6 days	 -0.231 -0.500	Eblen, 2002
Egg salad (store prep.)	5°C	No change	0.0	
Tuna salad (store)	5°C	No change	0.0	
Ham salad (store)	5°C	3 log **decr**. in 13 days	-0.231	
Imitation crab salad (store)	5°C	3 log **decr**. in 19 days	-0.158	
Chicken salad: (high pH); (low pH)	4°C	 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°C	 1 log **decr**. in 20 days 1 log **decr**. in 4 days	 -0.050 -0.250	
Pasta salad: (high pH); (low pH)	4°C	 1 log **decr.** in 9 days 1 log **decr.** in 6 days	 -0.111 -0.250	
Seafood salad (high pH); (low pH)	4°C	 1 log **decr**. in 23 days 1 log **decr**. in 23 days	 -0.043 -0.043	

EGR = Exponential Growth Rate, GT = Generation Time, Log = Log_{10} cfu/g

APPENDIX 2: EFFECTS OF FOODBORNE PATHOGENS ON MOTHER AND FOETUS DURING PREGNANCY

Foodborne pathogen	Susceptibility to pathogen during pregnancy	Effect on foetus or newborn	Reference
Hepatitis A virus	No increased susceptibility	Transplacental transmission not reported; no increased risk for spontaneous abortion, stillbirth or congenital malformation; infection of woman during third trimester may increase preterm delivery; baby may contract disease from mother during neonatal period	Gold *et al*., 1991 Pastorek 1993 Watson *et al*., 1993 Zeldis and Crumpacker, 1995
Hepatitis E virus (HEV)	HEV appears to have a high predilection for pregnant women with a death rate of 15 to 25% (HEV generally limited to underdeveloped nations).	Transplacental transmission may occur; increased abortion and intrauterine death; increased preterm delivery, baby can be infected during birth	Gold *et al*., 1991 Khuroo *et al.,* 1995 Mast and Krawczynski, 1996 Pastorek 1993 Rab *et al.,* 1997 Tsega *et al.,* 1992
Brucella species (generally *B. melitensis*)	No increased susceptibility in humans (tropism occurs in ungulate placenta due to presence of erythritol); brucellosis is a rare disease in the United States (and New Zealand).	Transplacental transmission, abortion and the presence of the organism in mother's milk have all been reported	Al-Eissa and Al-Mofada 1992 Al-Mofada *et al.,* 1993 Chheda *et al*., 1997 Lubani *et al*., 1988 Poole *et al*., 1972 Smith and Ficht, 1982; Young, 1983
Campylobacter jejuni	No increased susceptibility	Transplacental transmission has been reported; abortion may occur if woman is infected early in pregnancy; later infection may result in stillbirth or preterm delivery; newborn may be infected during delivery	Goh and Flynn, 1992 Skirrow and Blaser, 1995 Sweet, 1991
Coxiella burnetii (Q fever)	*C. burnetii* appears to have a tropism for placental tissure	Transplacental infection may occur; abortion, preterm birth, intrauterine growth retardation intrauterine death may occur; baby may be uninfected even when organisms are present in placental tissue.	Fournier *et al.,* 1998 Friedland *et al.,* 1994 Ludham *et al*., 1997 Raoult and Stein, 1994 R echman *et al.,* 1988 Stein and Raoult, 1998 Téllez *et al.* 1998

Foodborne pathogen	Susceptibility to pathogen during pregnancy	Effect on foetus or newborn	Reference
Escherichia coli O157:H7 (causing haemolytic uraemic syndrome (HUS)	Infections in pregnant women have not been reported. However, HUS in pregnancy is associated with high mortality or long term morbidity unless the woman is treated	HUS in pregnancy may lead to preterm delivery or intra-uterine death of foetus	Dashe *et al.*, 1998 Egerman *et al.*, 1996 Martínez-Román *et al.*, 1996 Weiner, 1987
Listeria monocytogenes	Organism has predilection for foetal-placental unit	Transplacental infection of foetus occurs with the possibility of a live congenitally infected baby; infection early in pregnancy poses greatest risk to foetus and newborn; abortion, intrauterine death, stillbirth, premature labour may occur, babies may be infected during delivery.	Bortolussi and Schlech 1995 Klink and Rudnicka, 1995 Luft and Remington, 1982 Schlech 1993 Schuchat *et al.*, 1991 Sweet, 1991
Salmonella Typhi (typhoid)	No increased susceptibility; in absence of antibiotic treatment, there is significant foetal and maternal mortality	Transplacental infection of foetus may occur; abortion, stillbirth, premature labour may occur; baby may be infected during delivery	Dildy *et al.*, 1990 Pickering *et al.*, 1995 Sweet, 1991
Salmonella species (non-typhoid)	No increased susceptibility	No transplacental transmission; bacteraemia in mother may lead to stillbirth; infection of baby during delivery is uncommon	Pickering *et al.*, 1995 Scialli and Rarick, 1992 Sweet, 1991
Shigella species	No increased susceptibility	No transplacental transmission; shigellosis is not identified as a cause of abortion, stillbirth or premature labour; infection of baby during delivery is uncommon	Pickering *et al.*, 1995 Sweet, 1991
Vibrio cholerae	No increased susceptibility but cholera is more severe in pregnant women	No transplacental transmission; abortion, premature labour and intrauterine foetal death death may occur	Bennish, 1994 Sweet, 1991
Yersinia enterocolitica	No increased susceptibility	No transplacental transmission; little evidence of abortion in humans; infection of baby during delivery does not appear to occur.	Paisley and Lauer, 1992 Sweet, 1991
Cryptosporidium parvum	No increased susceptibility	No transplacental transmission; foetal distress may occur if mother's diarrhoea is severe; baby may be infected during delivery	Chan and Sweet, 1991 Dale *et al.*, 1987 Lähdevirta *et al.*, 1987

Foodborne pathogen	Susceptibility to pathogen during pregnancy	Effect on foetus or newborn	Reference
Entamoeba histolytica	No increased susceptibility but pregnant women are at greater risk of rapid disease development	No transplacental transmission; infection of woman early in pregnancy may lead to abortion; dehydration and malnutrition induced by disease in mother may lead to intrauterine growth retardation; baby may be infected during delivery	Chan and Sweet, 1991 Pastorek, 1990
Giardia lamblia	No increased susceptibility; disease may be more severe in pregnancy	No transplacental transmission; severe maternal infection that compromises nutrition may affect foetal growth; baby may be infected during birth by faecal contamination	Arvin and Maldonado, 1995 Kreutner *et al*., 1981
Toxoplasma gondii	Parasite has predilection for foeto-placental unit	Transplacental transmission occurs with possibility of live congenitally infected baby; still-birth and early perinatal death may occur; it is not certain that parasite induces abortion in humans	Chan and Sweet, 1991 Luft and Remington, 1982 Remington *et al.*, 1995 Walker *et al*., 1997
Trichinella spiralis	Increased susceptibility?	Infection of placenta has been observed; transplacental transmission? (there are no reports of congenital trichinosis in live newborns); abortion, stillbirth, premature labour may occur; baby not infected during birth (the parasite has been found in infected mother's milk, which can result in infected baby.	Chan and Sweet, 1991

Source: Smith (1999)

The Table below itemises those pathogens not mentioned by Smith (1999).

Foodborne pathogen	Susceptibility to pathogen during pregnancy	Effect on foetus or newborn	Reference
Clostridium botulinum	There does not appear to be an increased susceptibility.	Four reported cases in the scientific literature regarding botulism during pregnancy. Conclusions from these cases: Botulism toxin is too heavy on a molecular scale to passively cross the placental membrane. In four reported cases, the foetus had no toxicological effects, three were born healthy. One born with oxygen shortage side effects. Appears that symptoms experienced by the mother (difficulty in breathing, eating, opportunistic infections e.g. pneumonia etc) can result in lowered oxygen, poor nutrition and other risks to the foetus, but no risk from the botulinal toxin itself. Animal experiments confirm that the toxin does not cross the placenta.	Robin *et al*., 1996 St Clair *et al.,* 1975 Polo *et al.,* 1996 Magri *et al.,* 2006 Cherington 1998 (review)
Staphylococcus aureus	There does not appear to be an increased susceptibility.	No reports located that *S. aureus* is responsible for birth defects or miscarriage. Illness likely to be acute for mother but no long term effects for the foetus or newborn reported.	No scientific references located
Norovirus	There does not appear to be an increased susceptibility.	Dehydration of the mother appears main issue. No reports of any effect on foetus or newborn.	No scientific references located
Bacillus cereus	There does not appear to be an increased susceptibility.	No risk found from foodborne *B. cereus* intoxications during pregnancy. Environmental sources can be a problem for premature neonates with low immunity.	No scientific references located

APPENDIX 3: *T. GONDII* – CASE CONTROL STUDIES

Country	Cohort	Risk factors	Reference
France	80 seroconverted pregnant women v 80 seronegative pregnant women	• Consumption of undercooked (centre raw) beef, Odds Ratio (OR) = 5.5 (95% Confidence Interval (CI) = 1.1 - 27) • Having a pet cat OR=4.5 (95% CI 1.0–19.9) • Frequent consumption of raw vegetables outside the home OR 3.1 (95% CI 1.2 – 7.7).	Baril *et al.*, 1999
Naples	3518 women (42 recently infected, 1380 previously infected and 2096 susceptible)	• 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)	Buffolano *et al.*, 1996
Norway (1992-1994)	37,000 pregnant women, a total of 63 women with recent primary infection and 128 seronegative controls were matched.	• Eating raw or undercooked minced meat products OR 4.1 (95% CI 1.5-11.2) • Eating unwashed raw vegetables or fruits OR 2.4 (95% CI 1.1-5.6) • Eating raw or undercooked mutton OR 11.4 (95% CI 2.1-63.1) • Eating raw or undercooked pork OR 3.4 (95% CI 1.1-10.4) • Cleaning cat litter box OR 5.5 (95% CI 1.3-22.7) • 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)	Kapperud *et al.*, 1996
Europe	252 seroconverted pregnant	• Eating raw/undercooked	Cook *et al.*, 2000

Country	Cohort	Risk factors	Reference
(multi-centre)	women compared to 852 controls	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 • Contact with soil OR 1.81 (95% CI 1.2-2.7) P=0.005 • Travel outside of Europe/USA or Canada OR 2.33 (95% CI 1.3-4.1) P=0.003	

In the Cook (2000) study, weaker associations (p>0.05) were observed for tasting meat during meal preparation, eating salami, drinking unpasteurised milk and working with animals.

* specifically venison, horse, rabbit, whale and game birds

APPENDIX 4: NEW ZEALAND RISK COMMUNICATION MATERIAL

- "Food Safety in Pregnancy", NZFSA, updated August 2009 (also available in Maori and Pacific Island languages);

- "Food safety: avoiding *Listeria*" Ministry of Health, revised September (2004a);

- "Your pregnancy Tō Hapūtanga. A guide to pregnancy and childbirth in New Zealand", Ministry of Health, revised August (2004b);

- "Eating for Healthy Pregnant Women", Ministry of Health, revised October (2007);

- "Food and Nutrition guidelines for Health Pregnant and Breastfeeding women: A background paper" Ministry of Health, 2006. This paper informs the "Eating for Health Pregnant Women" pamphlet listed above.

Other general food safety advice based on microbiological hazards the Foodsafe Freddie initiative which covers the Cook, Clean, Cover, Chill and handwashing advice (www.foodsafe.org.nz)

Gardening

The NZFSA gives advice under the 'Gardening and food safety' section of the leaflet regarding *Toxoplasma* cysts [*sic* – technically oocysts]. Advice includes the wearing of gloves and not touching the face, mouth or eyes while gardening. This is to prevent direct infection with oocysts. In addition, one of the recommendations is to avoid the stirring up or breathing in of dust from soil. However, the only reference found in the literature relating to the inhalation of *T. gondii* is a German paper by Kunert and Schmidtke (1954) entitled 'Inhalation experiments with *Toxoplasma gondii'*. There are other pathogens associated with the breathing in of soil, dust etc such as *Legionella* that are not relevant in the context of pregnancy.

Overseas risk communication material

Overseas, comparable advice for food safety during pregnancy has been published by counterparts of the NZFSA. They are referred to in this document in relation to each food group discussed.. The material includes:

- Australia and New Zealand: Food Standards Australia New Zealand (FSANZ) launched a review *Listeria* pamphlet in 2004 "*Listeria* and food"; Advice for people at risk. Some commentary surrounding the development of the pamphlet is contained in a powerpoint presentation available at the following web address: http://www.foodstandards.gov.au/_srcfiles/Listeria_presentation-AIFST20051.pdf#search=%22listeria%22; Further background information has been posted on the FSANZ website http://www.foodstandards.gov.au/_srcfiles/Listeria_Q_%20A_Version_FINAL.pdf;

- Australia: New South Wales Food Authority, "Food Safety during Pregnancy"

http://www.foodauthority.nsw.gov.au/_Documents/consumer_pdf/pregnancy_table.pdf;

- United Kingdom: "Pregnancy Book" Health Protection Agency (2007)

- USA: "Food safety for mums to be": "*Listeria*, frequently asked questions", and "*Toxoplasma*, frequently asked questions". U.S. Food and Drug Administration (USFDA) (2004);

- Position of the American Dietetic Association: Nutrition and lifestyle for a healthy pregnancy outcome (2008), USA.

In the last of these, advice is based upon research by Kendall *et al*., (2003). Forty-one food safety experts were asked to rate food handling behaviours (related to 13 pathogens) in regard to pregnant women, infants and young children, elderly people and people with compromised immune systems because of disease or pharmacologic therapy. Those behaviours rated as being of special importance to pregnancy (80% or more of the expert panel) are shown below:

Behaviours of special importance in relation to pregnancy (by panel of US food safety experts)

Behaviour	Pathogen
Avoid soft cheeses, cold smoked fish and cold deli salads	*L. monocytogenes*
Avoid hot dogs and lunch meats that have not been reheated to steaming hot or 165°F (73.88°C)	*L. monocytogenes*
Use cheese and yoghurt made from pasteurised milk	*Salmonella* species
Avoid eating foods containing raw eggs/ cook eggs until both the yolk and white are firm	*Salmonella* Enteritidis
Do not clean cat litter boxes if pregnant Use plastic gloves when cleaning cat litter boxes Do not handle pets when preparing foods Keep pets out of food preparation areas	*Toxoplasma gondii*

Sent by: Louise McIntyre/MOH
09/02/2015 04:10 p.m.

To: "Roger Cook (Roger Cook)" <Roger.Cook@mpi.govt.nz>,
cc: Julia.Edmonds@mpi.govt.nz, Martin Dutton/MOH@MOH,
bcc:

Subject: Food safety pages to MPI v2 - feedback due Tues 24 Feb

Hi Roger

I spoke with Julia last Friday who suggested I re-send you the food safety pages for your review. As below this chapter is to be included in the new *'Eating and Activity Guideline Statements for New Zealand Adults*' document.

We have changed the content of the food safety section a little to try and improve its readability so here is the updated version:  Food Safety section 9 Feb 2015.docx

We have also reworked the purpose of the document which is now:

1. brings together the updated eating and physical activity statements (*statements)* for New Zealand adults, outlining each statement and why it is recommended
2. connects the statements to the most robust (systematically reviewed and graded) international evidence currently available
3. provides some information for putting the statements into practice.

More detailed information on how to incorporate the *statements* into people's daily lives can be found in the accompanying health education resources. See www.healthed.govt.nz.

The rest of the info below is still correct, including the fact the document is for health practitioners.

The document goes for final review to the Technical Advisory Group, internal Ministry Groups and key external Govt agencies, including MPI on 2 - 13 March. It would be good to get your feedback by Tues 24 Feb. If this is not possible let me know. Im happy to meet with you to discuss this further if useful.

We are still looking for an evidence base to underpin this food safety statement and Ive included below directions to the Australian Guidelines evidence base document to see if that is appropriate.

Regards

Louise McIntyre
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----- Document: Food safety pages to MPI, forwarded by Louise McIntyre on 09/02/2015 03:58 pm -----

Sent By: Louise McIntyre/MOH on 19/09/2014 6:11:09 p.m.
To: "Roger Cook (Roger Cook)" <Roger.Cook@mpi.govt.nz>
Copy To: Julia.Edmonds@mpi.govt.nz
Subject: Food safety pages to MPI

Hi Roger

I havent heard from you re the previous emails so Im just powering ahead hoping all is well?! Please find attached the draft food safety info that will go into our Eating and Activity Guidelines core guidelines paper. We intend the core paper to be around 30-50 pages long and written in plain English using less technical wording. We need the document to be accessible to a wide range of practitioners including those with little or no nutrition or science background.

Draft Eating statement 5.docx

The purpose of core paper is to:

1 To bring together the key eating and activity guideline statements (key messages) for all New Zealanders into one document. *The first core paper will include statements relevant to all. It is planned that over time, ie in subsequent editions, statements relevant to specific population groups will also be included. For example, infants, toddlers, children, young people, pregnant and breastfeeding women (and young people).*

2 Provide a snapshot of the important and relevant background information related to statements (key messages).

3 Show the evidence that underpins each specific statement.

Target audience for paper:

- Health practitioners and those involved in promoting healthy eating and/or physical activity to New Zealanders - including health promoters and community health workers
- Other interested groups including:
    - Educators; local, regional and national policy advisors;regulators; the food industry; the fitness industry; researchers; and professional bodies

Re the evidence base to underpin the food safety statement - do you have a view on the one used for the Australian Dietary Guidelines?

http://www.eatforhealth.gov.au/sites/default/files/files/the_guidelines/n55d_dietary_guidelines_evidence_report.pdf

The food safety info can be found on page 1062 onwards - as our document is at present only for adults we wont be including the info related to infants and pregnant women.

Happy to take any other more appropriate solutions. Im aware you mentioned the ideal would be for MPI to pull together the evidence base for its own advice, but from a pragmatic stance would this Australia info work in the short term?

Regards

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----- Document: Food safety statement and the Eating and Activity Guidelines, forwarded by Louise McIntyre on 19/09/2014 05:35 pm -----

Sent By: Louise McIntyre/MOH on 16/09/2014 1:33:42 p.m.
To: "Roger Cook (Roger Cook)" <Roger.Cook@mpi.govt.nz>
Copy To: Julia.Edmonds@mpi.govt.nz
Subject: Food safety statement and the Eating and Activity Guidelines

Hi Roger

As discussed previously Im currently writing our core document containing the updated eating and activity statements for adult New Zealanders (See previous email send to you and Julia in July). This core document includes a specific food safety statement which currently reads:

"Buy, prepare, cook and store food to ensure it is safe to eat."

The food safety section to be 2 x A4 pages which will include the following three sections: 'Background', 'Evidence base' and 'Practical ideas. Im planning to use information from MPI website and the 3 C's work as previously linked me by Sally Johnson.

At our meeting in June we discussed whether there was an evidence base available that underpins the basic and general food safety information MPI provides to the public. You thought ESR's YOPI booklet could provide some of this info. Could you send me a copy of this document?

My other query is are you aware of any recent general info on food safety in New Zealand? Specifically Im looking for anything with incidence rates of food poisoning, anything really to give me info on why food safety is an important consideration for New Zealanders. I see this going into the 'Background' section.

As documented below we also discussed MPI reviewing the 2 page food safety section to make sure you were happy with its info and messages. Is this still a possibility? Im hoping to send you a draft of the 2 pages by the end of this week and could give you until the 30 September to review. The document is for health practitioners but we are aiming to keep it as simple and straightforward as possible so hopefully the reviewing the 2 pages would not be too onerous. Having said that Im aware of the very busy workload you all have.

Happy to discuss further if useful.

Regards

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----- Document: Food safety statement and the Eating and Activity Guidelines, forwarded by Louise McIntyre on 16/09/2014 01:03 pm -----

Sent By: Louise McIntyre/MOH on 7/07/2014 5:23:41 p.m.
To: Julia.Edmonds@mpi.govt.nz, "Roger Cook (Roger Cook)" <Roger.Cook@mpi.govt.nz>
Copy To:
Subject: Food safety statement and the Eating and Activity Guidelines

Hi there

Firstly apologies for not getting back to you sooner following our meeting on Friday 20 June. I had promised to summarise our discussion and the plan we discussed.

Key points from the discussion:

- The Ministry is currently reviewing its food and nutrition guidelines statements for adults as part of the transition from the current Food and Nutrition Guidelines Series (FNGS) to the new Eating and Activity Guidelines Series (EAGS). One of the first steps in this transition is to produce a core paper for the sector which will be based on the eating (nutrition) and activity statements. A key feature of the transition from FNGS to EAGS is to have a stronger (systematic and graded if possible) and more transparent evidence base to underpin the statements. The core paper will include a summary/description or link to the evidence base.
- We are keen for MPI's input on reviewing the food safety related statement. This statement is currently: "Buy, prepare, cook and store food in ways to ensure food safety." Feedback from Sally Johnson in May 2014 suggested incorporating the 3 'C' into the statement as these already are known and promoted. Sally also linked us to appropriate consumer focused MPI webpages that could be used for how-to information.
- MOH is also interested to have the evidence base used by MPI that underpins the general food safety advice for the public. A brief summary will be put into the core paper with any links or key documents placed on the EAGS webpage. As per our discussion, the evidence is currently not all together in one document and would need to be collated. This was thought possible but would take some time (ie would not be ready by planned date for finalising the core paper content - 31 July 2014).
- There is a documented evidence base for the YOPI group which is contained in an ESR report. This could be used to underpin the food safety statements.
-

Plan

1 MPI to link me to ESR YOPI booklet evidence base.
2 MPI to consider what could be used as a temporary, quick and easy evidence base for food safety statement. ?
3 MPI to speak with Team re possiblity of collating the evidence base that would underpin the food safety statement in the longer term.
4 MOH will review current draft food safety statement and consider the 3C's idea and the supporting information from MPI website. Once re-work will send to MPI for review.

Let me know if this is how you remember things and if you are still happy with the plan. Not surprisingly we are looking at pushing out the 31 July date so will let you know what new date we are aiming for to finalise content.

Regards

•

Louise McIntyre
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Public Health Group
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