RISK PROFILE: SHIGA-TOXIN PRODUCING *ESCHERICHIA COLI* IN UNCOOKED COMMINUTED FERMENTED MEAT PRODUCTS

Prepared as part of a New Zealand Food Safety Authority contract for scientific services

by

Susan Gilbert
Dr Rob Lake
Dr Andrew Hudson
Peter Cressey

August 2007

Institute of Environmental Science & Research Limited
Christchurch Science Centre
Location address: 27 Creyke Road, Ilam, Christchurch
Postal address: P O Box 29 181, Christchurch, New Zealand
Website: www.esr.cri.nz

A CROWN RESEARCH INSTITUTE

Client Report
FW0611

RISK PROFILE: SHIGA-TOXIN PRODUCING *ESCHERICHIA COLI* IN UNCOOKED COMMINUTED FERMENTED MEAT PRODUCTS

Dr Stephen On
Food Safety Programme Manager

Dr Rob Lake
Project Leader

Rosemary Whyte
Peer Reviewer

ACKNOWLEDGEMENTS

The authors would like to thank:

Rob Scott, Goodman Fielder for information and advice.

Shankar Cumaramsy and Frances Clements, New Zealand Pork Industry Board for information and advice.

Dr Tecklok Wong at ESR Christchurch Science Centre for the results of his unpublished surveys of STEC in retail meats and imported and domestically produced pork.

Liza Lopez and Trev Margolin at ESR, Kenepuru Science Centre, Porirua, for surveillance information.

CONTENTS

LIST OF TABLES

LIST OF FIGURES

SUMMARY

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

This Risk Profile concerns Shiga-toxin producing *Escherichia coli* (STEC) in uncooked comminuted fermented meats (UCFM). The production of UCFM is an example of the use of hurdle technology as a means to preserving meat where no anti-microbial factor acting on its own will be sufficient to inhibit the growth of STEC. The hurdles employed are principally reduction in pH, and reduction of water activity by drying and the addition of salt. These measures have been shown to reduce STEC numbers by up to 3 $\log_{10}$ cfu/g during UCFM manufacture. This level of control might be insufficient to assure safety, and depends on the quality of the raw materials and process controls implemented (a 5D reduction has been advocated in the USA). Given the ability of STEC to survive in such products, and the suggestion that pathogenic STEC may be more acid tolerant than other strains, there is a requirement for the quality of ingredient meats to be controlled.

UCFM are infrequently consumed by the New Zealand population, although consumption may be increasing due to the popularity of deli-sandwich outlets. Salami was the only descriptor in the 1997 National Nutrition Survey, and only 1.6% of the population reported consuming salami (in sandwiches, bagels etc) in the previous 24 hour period. (Cooked salami/pepperoni on pizzas etc are not considered for this risk profile due to their thermal treatment).

The amount of UCFM imported into New Zealand each year is unclear, but the majority will originate from Australia, where production will be subject to the FSANZ standard. The relatively low frequency of consumption of UCFM and the smaller serving sizes than for most other meat and meat products may mitigate the relative risk from this meat type.

E. coli O157 has been found in the faeces of cows in New Zealand, as well as a small number of raw meat samples from export processing plants. Non-O157 STEC serotypes have been detected in faecal samples from sheep and cows, as well as samples of meat from retail sources. Some of these serotypes have been involved in human illness. Consequently there is potential for the presence of pathogenic STEC in raw meat in New Zealand, which then may be used as an ingredient in the production of UCFM. There is also the possibility that imported raw meat such as pork from Australia, which has the potential to be contaminated with STEC, ends up in UCFM production in New Zealand.

STEC infections have a relatively high proportion of serious outcomes compared to other bacterial infections, and certain population groups in New Zealand (children up to 4 years) have higher rates of infection.

STEC infection may result in serious complications often requiring hospitalisation (approximately 32% of cases in New Zealand in 2004). Long term effects can include

Haemolytic Uraemic Syndrome (HUS), kidney problems, hypertension, neurological deficits and in a very few cases the disease can be fatal. The population group most at risk are very young children.

The rate of STEC infection in New Zealand has been increasing since the 1990s. From 1998 to 2004, the rate has nearly doubled from 1.3 per 100,000 to 2.4 per 100,000. Most New Zealand cases appear to be sporadic or in family clusters and are predominantly rural. However, information on transmission routes is limited, with little indication of foodborne transmission, and none implicating UCFM.

The 2004 rate of STEC infection in New Zealand is similar to that in England and Scotland (2.1 and 2.9 respectively). The Canadian rate is higher (at 8.8 per 100,000) while the Australian rate at just 0.3 per 100,000 in 2002 is considerably lower.

As was concluded for Australia (FSANZ, 2003), the risk is low but the consequences, particularly for susceptible groups, such as young children, are severe. Given the potential for exposure, on the basis of the observed prevalence of STEC in red meat in New Zealand, the risk needs to be managed by an appropriate measure. A draft New Zealand Standard is currently undergoing public consultation.

The data gaps identified in this Risk Profile are:

- Current information on the CCPs being employed by UCFM producers in New Zealand,
- Current prevalence of STEC (not just *E. coli* O157) in New Zealand UCFM at the retail level or at other points in the production chain, as well as the ingredients – domestic and imported pork, sheep and deer meat,
- Quantitative levels of contamination by STEC, when contamination does occur, of New Zealand UCFM at the retail level (or at other points in the production chain),
- Methods to detect non-O157 STEC with the same sensitivity as *E. coli* O157,
- The prevalence of *E. coli* O157 and *E. coli* in meat from processing plants which deliver only to the domestic market and which can be used in UCFM production,
- Relative importance of transmission pathways for STEC in New Zealand, and
- Information on the market size and market structure for UCFM, including quantification of the Australian product and consumption patterns in at risk groups.

1 INTRODUCTION

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

Figure 1: Risk Management Framework

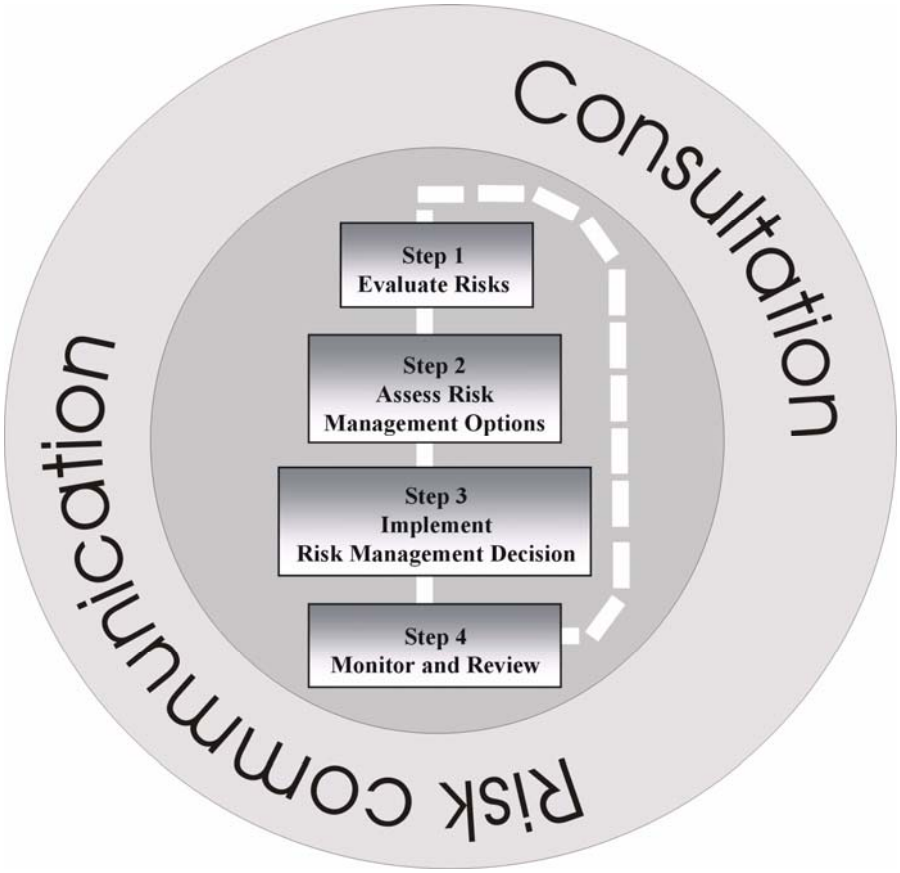

Figure reproduced from "Food Administration in New Zealand. A risk management framework for food safety" (Ministry of Health/Ministry of Agriculture and Forestry, 2000).

In more detail, the four step process is:

1. Risk evaluation

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

- consideration of the results of risk assessment

2. Risk management option assessment

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

3. Implementation of the risk management decision

4. Monitoring and review.

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

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

This Risk Profile concerns shiga-toxin producing *Escherichia coli* (STEC). The most well known of these are *E. coli* O157:H7 and O157 (NM) but this Profile also considers other serotypes. These organisms are important emerging pathogens, recognised for the first time in the United States in 1982. The first human case of illness caused by *E. coli* O157 in New Zealand occurred in 1993 (Baker *et al.*, 1999).

The food group to be addressed in this Risk Profile is uncooked comminuted fermented meat (UCFM) products. This type of food largely relies on pH and water activity reduction to control pathogens, rather than heat treatment. There have been suggestions that some strains of STEC are more acid tolerant than *E. coli* bacteria in general (Tilden *et al.*, 1996) and so control of this bacterium is particularly important for UCFM.

The sections in this Risk Profile are organised as much as possible as they would be for a conventional qualitative risk assessment, as defined by Codex (1999).

Hazard identification, including:

- A description of the organism
- A description of the food group

Hazard characterisation, including:

- A description of the adverse health effects caused by the organism.
- Dose-response information for the organism in humans, where available.

Exposure assessment, including:

- Data on the occurrence of the hazard in the New Zealand food supply.
- Data on the consumption of the food group by New Zealanders.
- Qualitative estimate of exposure to the organism (if possible).
- Overseas data relevant to dietary exposure to the organism.

Risk characterization

- Information on the number of cases of adverse health effects resulting from exposure to the organism with particular reference to the identified food (based on surveillance data)
- Qualitative estimate of risk, including categorisation of the level of risk associated with the organism in the food (categories are described in Appendix 1).

Risk management information

- A description of the food industry sector, and relevant food safety controls.
- Information about risk management options.

Conclusions and recommendations for further action

2 HAZARD IDENTIFICATION: THE ORGANISM

2.1 Shiga-toxin producing *Escherichia coli* (STEC)

The following information is taken from data sheets prepared by ESR under a contract for the Ministry of Health. The data sheets are intended for use by regional public health units. Information for *E. coli* O157 is presented separately from other shiga-toxin producing serotypes. The ability of the serotypes in the latter group to cause disease varies greatly.

2.1.1 Nomenclature

This Risk Profile is concerned with the group of *E. coli* that carry the shiga-toxin genes *Stx1* and *Stx2* (STEC), some of which are classified as enterohaemorrhagic (EHEC). Two acronyms that are in common use that pertain to this group of organisms are VTEC (verocytotoxigenic *Escherichia coli*) and STEC (shiga-toxin producing *Escherichia coli*). The acronym VTEC is derived from the fact that the toxin expressed causes a pathological effect on Vero cells (Konowalchuk *et al*., 1977) in tissue culture (Vero cells are African green monkey kidney cells), while the acronym STEC is derived from the fact that the toxins are shiga-like i.e. similar to those produced by *Shigella dysenteriae* (Chart, 2000). The two acronyms have now become *de facto* synonyms. An alternative meaning to the acronym STEC is "Shiga-like toxin producing *E. coli*"; this is less commonly used although strictly more accurate. The term shiga-toxigenic *E. coli* is used in recent reviews (Baker *et al.*, 1999; Jaeger and Acheson, 2000) and by the International symposia and workshops on shiga toxin (verocytotoxin)-producing *Escherichia coli* infections.

The acronym EHEC, (Enterohaemorrhagic *Escherichia coli*) refers to those *E. coli* that have the same clinical, epidemiological and pathogenic features associated with the prototype EHEC organism *E. coli* O157:H7 (Karmali, 1989). Strictly EHEC are therefore a specific subset of the two groups of organisms described above as some STEC/VTEC have never been associated with human disease. However, EHEC is often used as a synonym of STEC and VTEC.

STEC will be the acronym used throughout this document.

Individual strains of STEC are denoted by their O and H serotypes; O= "ohne hauch" or the somatic antigen, H= "hauch" or the flagellar antigen. Non-motile isolates (normally recorded as NM) are considered here to be H-, i.e. without an H antigen. If the serotype cannot be determined it is described as NT: "non-typable". Some isolates of STEC react (i.e. agglutinate) with all sera; these are described as "rough".

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

2.2 *Escherichia coli* O157

2.2.1 The organism/toxin

E. coli O157 is a pathogenic variant of an organism that is generally regarded as innocuous. The H serotypes associated with this O type in cases of disease are H7 and H-.

2.2.2 Growth and survival

Growth:

Temperature: Optimum 37°C, range 7-8 to 46°C. Doubling time approx. 0.4 hours at 37°C.

pH: Optimum 6-7, range 4.4 to 9.0. The limit at the low pH end depends on the acidulant used. Mineral acids such as HCl are less inhibitory than organic acids (e.g. acetic, lactic – as produced post mortem in meat) at the same pH. Growth was inhibited in the presence of 0.1% acetic acid (pH 5.1). At pH 4.7, growth of the organism occurs at 25°C (Conner and Kotrola, 1995). Growth at pH 4.6 occurred but not at pH 4.5 when incubated at 37°C (Glass *et al.,* 1992).

Atmosphere: Can grow in the presence or absence of oxygen. Growth can occur in vacuum-packed meat at 8-9^{o}C, but not when the meat is packed under 100% CO_2. At 10^{o}C growth was not inhibited under 100% N_2 or 20% CO_2:80% N_2 but was inhibited under 100% CO_2.

Water activity: Optimum growth is at $a_w = 0.995$ minimum $a_w = 0.950$

Preservatives: Growth was observed in up to 6.5% NaCl when incubated at 37°C (Glass *et al*., 1992).

Survival:

Temperature: Survives well in chilled and frozen foods. For example little change was noted in numbers of cells in hamburgers stored at -20°C for 9 months.

pH: Can survive in low pH environments. In fact the organism dies slowly under these conditions and persistence is proportionate to the degree of contamination. For example, numbers reduced by only 100 fold after 2 months storage at 4^{o}C on fermented sausage of pH 4.5. Prior exposure to acidic conditions can increase acid tolerance. Has been shown to survive stomach pH (1.5) for periods longer than that required to clear an average meal (three hours).

Experiments to determine the acid tolerance of strains of enterohaemorrhagic *E. coli* (EHEC) showed that a number of strains could survive (i.e. were able to be recovered at levels up to 100%) at a pH of 2.5 or 3.0 for five hours when incubated at 37°C (Benjamin and Datta, 1995). These data were consistent with outbreaks of EHEC linked with the acidic foods apple cider and mayonnaise. There have been claims that pathogenic *E. coli* are significantly more acid tolerant than non-pathogenic strains, but this has not been clearly established (McClure and Hall, 2000). Significant interstrain variation with respect to acid tolerance is a common feature of both commensal and O157 strains of *E. coli* (Duncan *et al.*, 2000).

Atmosphere: An atmosphere of 100% CO_2 enhanced survival of uninjured cells at both 4 and 10°C. Survival on fermented meat was equivalent when packed under air or under vacuum.

Viable but Non-Culturable (VNC) Cells: Evidence indicates that low temperature is the primary signal for entry into the VNC state in water (Rigsbee *et al*., 1997) although sunlight too has been shown to cause VNC cells to form (Pommepuy *et al*., 1996). Entry into the VNC state is suspected in high salt foods (Makino *et al*., 2000).

2.2.3 Inactivation (CCPs and Hurdles)

UCFM is a classic example of hurdle technology. Literature data shows that no antimicrobial factor acting solely can be expected to inhibit growth of *E. coli* O157 in UCFM. Combinations need to be utilised (Glass *et al*., 1992). Nevertheless, experiments reported in this paper show that it is important to use ingredients with low populations or no *E. coli* O157:H7 in sausage batter, because when initially present at 10^4 cfu/g, the organism can survive fermentation, drying, and storage of fermented sausage regardless of whether an added starter culture is used.

Temperature: Rapidly inactivated by heating at 71°C (recommended temperature for hamburger cooking in the USA, in the UK it is 70°C for 2 minutes). D time at 54.4°C = 40 minutes. D time at 60 = 0.5–0.75 minutes (4.95 minutes in minced beef). D time at 64.3°C = 0.16 minutes.

pH: Inactivation at pH 4.5 in fermented meat created by lactic acid production from glucose by starter cultures.

Water activity: Withstands desiccation well and has caused disease through carriage on venison jerky. Generally accepted that growth is inhibited by a water activity of 0.89 or less when a_w is the sole antimicrobial factor.

Preservatives: 8.5% NaCl inhibits growth at 37°C, growth retarded above 2.5%. The amount of salt required for inhibition reduces as other factors such as temperature and pH become sub-optimal. For example 5% salt inhibited *E. coli* O157 at 12°C.

Radiation: Sensitive to UV and γ irradiation. D (kGy) approx. 0.31 frozen, 0.24 refrigerated in ground beef. A 2-3 kGy dose is sufficient to decontaminate meat.

2.2.4 Sources

Human: Faecal-oral person-to-person transmission is often reported in family members of cases who contracted the disease from food or water.

Animal: Found in the guts of ruminant animals. Cattle are considered primary reservoirs but sheep and deer may also carry the organism. Carriage of the organism by cattle is generally considered to be low, but estimates of prevalence are rising with improved laboratory techniques. Calves are thought to shed the organism more often than adult cattle.

Food: Food vehicles identified overseas have usually been contaminated by cattle manure.

Foods involved in outbreaks have included hamburgers, other meat products, apple juice, salads, bean sprouts, spinach, raw milk, cheese, melons, lettuce and yoghurt. For one case in New Zealand, an indistinguishable isolate was obtained from both the infected child and raw milk present in the home, milk that was originally taken from a dairy farm for the family's new kittens (Anonymous, 2002).

Environment: Water contaminated from faecal sources has been the vehicle involved in a number of large outbreaks overseas. Such waters have included reticulated drinking water and swimming/paddling pool water. Two cases in New Zealand have been attributed to the consumption of contaminated water (neither was reticulated water). The organism has been shown to survive for 150 days in soil and 90 days in cattle faeces. It can also survive for at least 4 months in sediment in cattle drinking troughs.

Transmission Routes: In summary, any food or water source that has been contaminated by the faeces of a ruminant animal. Direct contact with carrier animals is also a recognised transmission route. Secondary transmission is also common. Poor personal hygiene can also result in infection; eight pop festival attendees became infected after the event, which was held in a muddy paddock on which cattle had recently been grazed. The relative importance of the various transmission routes is currently not well understood in New Zealand.

2.3 Non-O157 Shiga-Toxin Producing *Escherichia coli* (STEC)

2.3.1 The organism/toxin

These organisms form a diverse group of *E. coli* that are capable of producing shiga-like toxin(s), as is *E. coli* O157:H7. However, they are of widely differing pathogenic potential, varying from those that can cause illnesses similar to that produced by *E. coli* O157:H7 to those that have never been associated with disease.

By definition all STEC must produce one of two groups of toxins (denoted *Stx*1 and *Stx*2 with subscripts to denote variants), but other factors are required for pathogenicity and it is the possession of these that seems to determine the virulence of any one serotype. Other factors known to be involved include the ability to adhere to intestinal cells (*eaeA* gene), and the ability to produce a haemolysin (*hlyA* gene, also known as the *ehx*A gene).

New Zealand isolates of STEC that have caused disease have, to date, possessed virulence factors in addition to either *Stx* gene.

2.3.2 Growth and survival

The behaviour of these organisms is largely the same as for serotype O157.

2.3.3 Inactivation (CCPs and Hurdles)

The behaviour of these organisms is largely the same as for serotype O157.

2.3.4 Sources

Human: Some serotypes are reported to be restricted to people, e.g. O1, O55:H7 and H:10

and O48:H21 (Bettelheim, 2000)

Animal: Ruminant animals, notably bovines, seem to be a natural reservoir of many of the non-O157 STEC that cause disease in humans.

Food, environment, transmission routes: Little is known about the distribution of these organisms in food and the environment. However, it seems likely that the prevalence will be higher than that for serotype O157 although difficulties with isolating these serovars make true prevalence unknown. Data from the national retail meat survey suggests that the majority of STEC serotypes found in positive samples were non-O157; beef 5.2% (12/233), bobby veal 1.6% (3/183), lamb/mutton 13.4% (31/231), pork 6.1% (14/231) (Dr Tecklok Wong ESR Christchurch, personal communication, August 2007). In a paper by Bosilevac *et al*., (2007), the prevalence of non-O157 STEC in New Zealand samples of lean boneless beef trim imported into the USA was 10%.

Non-O157 STEC have been detected in beef, pork, and lamb mince, and unpasteurised milk.

3 HAZARD IDENTIFICATION: THE FOOD

3.1 Relevant Characteristics of the Food: Uncooked Comminuted Fermented Meat (UCFM) Products

Where fresh meat is not preserved or cured, the rapid growth of indigenous gram-negative bacteria quickly spoils the food. Fermentation by microbial action, which produces acids and lowers pH, along with reduction of water activity by drying and the addition of salt, are preservation techniques that have been used for thousands of years. The lowered pH and water activity are interacting hurdles to microbial growth, in the absence of a heat treatment step. The style of UCFM being produced affects the way these hurdles are employed. Competition for nutrients and production of bacteriocins are also limiting factors.

UCFM has been defined by Food Standards Australia New Zealand (FSANZ) as;

"a comminuted fermented meat which has not had its core temperature maintained at 65°C for at least 10 minutes or an equivalent combination of time and higher temperature during production. To avoid doubt, a UCFM includes comminuted fermented meat which has been heat-treated" (FSANZ 2005).

Some UCFM may receive a final thermal treatment which is sufficient to pasteurise the food, but these products are not included in this Risk Profile. UCFM products can be classified by their water content: dry (20 – 35% water) or semi-dry (35 – 50% water).

Dry sausages are typically heavily seasoned and not smoked. They originate from the Mediterranean region where they are exposed to salt and rapid drying environments from the warm dry climate. Product names are not reliable descriptions of processing methods or characteristics, however the following products and their chemical characteristics are listed in Table 1 as a guide (NS = Not Stated).

Table 1: Dry sausage chemical characteristics

Type of UCFM (dry sausages)	Final pH	Lactic acid (%)	Moisture/protein ratio	Moisture loss (%)	Moisture (%)
Dry sausages: -	**5.0 – 5.3 (<5.3)**	**0.5 – 1.0**	**<2.3 : 1**	**25 - 50**	**< 35**
Genoa salami	4.9	0.79	2.3 : 1	28	33 - 39
Cappicola	NS	NS	1.3 : 1	NS	23 – 29
Italian salami, hard or dry	NS	NS	1.9 : 1	30	32 - 38
Cervelat	NS	NS	1.9 : 1	NS	32 - 38
Pepperoni	4.5 – 4.8	0.8 – 1.2	1.6 : 1	35	25 - 32
Thuringer, dry	4.9	1.0	2.3 : 1	28	46 - 50
German "Dauerwurst"	4.7 – 4.8	NS	1.1 : 1	NS	25 - 27

Semi-dry sausages originated in northern Europe. They have a higher water content, are lightly spiced and heavily smoked at cool temperatures. The colder ambient temperatures discouraged spoilage. Examples are given in Table 2.

Table 2: Semi-dry sausage with chemical characteristics

Type of UCFM (semi-dry sausages)	Final pH	Lactic acid (%)	Moisture/protein ratio	Moisture loss (%)	Moisture (%)
Semi-dry sausages: -	4.7 – 5.1 (<5.3)	0.5 – 1.3	2.3 – 3.7 : 1	8 - 15	45 - 50
Lebanon bologna	4.7	1.0 – 1.3	2.5 : 1	10 - 15	56 - 62
Cervelat (soft)	NS	NS	2.6 : 1	10 - 15	NS
Salami (soft)	NS	NS	2.9 – 3.7 : 1	10 - 15	41 - 51
Summer sausage	<5.0	1.0	3.1 : 1	10 - 15	41 - 52
Thuringer, soft	NS	NS	3.7 : 1	NS	46 - 50

NS = not stated (source; Ricke and Keeton, 1997; Ricke *et al*., 2001).

The compositional differences in the two types of fermented sausage are show below in Table 3.

Table 3: Compositional differences between dry and semi-dry UCFM

Parameter[#]	Semi-dry (European sausage)	Dry (summer sausage)
Moisture	50	30
Fat	24	39
Protein	21	21
Salt	3.4	4.2
pH	4.9	4.7
Total acidity	1.0	1.3
Yield	90	64

[#]Values except for pH are expressed as % (wt/wt).
Modified from Ricke *et al*., (2001).

Fermented sausages rely on both a reduced 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. Moisture losses are much higher (25-30%) for dry salami varieties (DeBauch and Savage, 1993). It is the decline in pH coupled with the build-up of lactic acid that results in the low water activity. The product becomes denser and firmer in texture as it dehydrates with age. Lactic acid is the dominant flavour component (Ricke *et al.,* 2001).

UCFM usually contains minced, chopped or ground meat along with salt (2.5-3%), nitrite (<150mg/kg), sodium erythorbate (550-600 mg/kg), glucose (0.4-0.8%), spices, seasonings,

antioxidants (natural or synthetic) and a method of initiating the fermentation process (Ricke *et al*., 2001), as described below. Fermentation takes place in moisture permeable casings.

The meat ingredients are primarily beef and pork, with lamb and mutton meats less commonly used. Venison is a new and upcoming ingredient. Poultry meat is sometimes used but is less desirable as the fat has a higher polyunsaturated fatty acid content, which makes it more susceptible to oxidation and rancidity which produce off-flavours. Where poultry meat is used, it is often supplemented with pork or beef (Ricke *et al*., 2001). Fresh or frozen meats can be used, the use of frozen meat is preferable for several reasons. Often there is a reduction in pathogenic cell numbers present, those that do survive are sub-lethally damaged and growth of pathogens is prevented. The primary requirements are that the raw meat ingredients have a low microbial population (kept refrigerated < 4.5°C), no discolouration or off odours, limited age, no dark, firm, dry tissue (DFD), with blood clots, and glands, sinews, gristle and bruises trimmed off.

Nitrates and nitrites are usually added at a minimum rate of 40 – 50 mg/kg at the start of the production process as no other hurdles are established at this stage. These curing additives develop the colour and taste, retard lipid oxidation and inhibit *Salmonella, Staphylococcus aureus* and *Clostridium botulinum*. However, they are only effective against pathogen growth in the early stages of fermentation as they are rapidly consumed by microbial activity (Ross and Shadbolt, 2001).

During the meat chopping stage, oxygen is incorporated into the mix creating a high redox potential (Eh). The addition of ascorbic acid reduces the Eh which in turn stimulates the action of nitrite, restricts aerobic spoilage bacterial growth and promotes lactobacilli growth (Hasell, 2000). Sodium erythorbate is often added as well to enhance colour and retard the formation of any carcinogenic N-nitrosamines.

Added spices and seasonings include ground pepper, paprika, garlic, mace, pimento, cardamom, red pepper, and mustard. These must be sterile to avoid wild fermentations. Red pepper and mustard have been shown to stimulate lactic acid production (Ricke *et al*., 2001).

If a starter culture is used, the exponential growth of the lactic acid bacteria from the starter inoculum suppresses the indigenous microflora thus inhibiting the growth of pathogens during the initial stages of fermentation. About 0.62g of glucose per kg meat is needed to achieve a pH reduction of 0.1 unit. The native glucose content of post-rigor meats is insufficient to achieve the desired rapid reduction in pH. The chemical acidulant glucono-delta-lactone (GDL) may be added and is rapidly converted to lactic and acetic acid by lactobacilli, either indigenous or added as starter culture (Ricke *et al*., 2001).

Note that the use of a starter culture is mandatory in Australia under the Food Code, and GDL may only be added in small quantities. The sole use of GDL in New Zealand without the use of a starter culture is currently permitted although proposed Regulations set out in the New Zealand Draft Standard could see the use of starter culture become mandatory (draft published for public consultation, February 2007).

There are three methods of initiating fermentation:

- Natural fermentation (the use of indigenous microflora),
- "Back-slopping" from a previous batch, and
- Use of a starter culture.

From a microbiological point of view, and as required under the Food Standards Code and proposed New Zealand Standard, a bacterial inoculum as a starter culture chosen according to the temperatures used in the process and the level of lactic acid desired is preferable (Ricke and Keeton, 1997). Starter cultures offer greater standardisation and their characteristics, classification and metabolism mechanisms have been described (Ricke *et al.,* 2001). The bacteria belong predominantly to the genera *Streptococcus*, *Lactococcus*, *Leuconostoc*, *Pediococcus*, *Bifidobacterium* and *Lactobacillus* and are commercially available as pure cultures or mixed cultures.

During the fermentation process two basic microbial processes occur: (i) the production of nitric oxide, and (ii) a decrease in pH due to anaerobic glycolysis and the production of lactic acid. The processes are synergistic because of the interdependency of pH and nitrite/nitrate reduction.

The traditional practice of initiating fermentation by adding a portion of the previous batch ("back-slopping") is not permitted. Indeed, under the FSANZ Food Standard Code applicable in Australia, and the draft New Zealand Standard, a starter-culture is required to begin the fermentation process.

3.1.1 Production sequence

The following information is taken from Ricke and Keeton (1997), Ricke *et al*. (2001) and Attachment 4 of FSANZ (2003), "Food Technology Report". Production of UCFM involves:

(i) reduction of particle size of the meat, lean meats to 6.35 – 12.7mm in size, fat meats to 12.7 – 25.4mm, then combined to specified fat content at endpoint,

(ii) combination of ingredients,

(iii) mixing and further reduction of particle size to produce a "batter", starter culture is rehydrated from frozen or lypophilised state and kept for one hour at ambient temperature before addition to batch. The fine grind of the mixture results in 3.2 – 4.8 mm particle sizes depending on type of sausage being produced,

(iv) vacuum stuffing into a semi-permeable fibrous or natural casing at 2°C (thereby keeping oxygen exposure low),

(v) incubating (anaerobic fermentation begins, favouring lactic acid bacteria growth) at the temperature optimum of the starter culture, a low-temperature incubation (15 – 26°C) or high-temperature incubation (32.5 – 38.1%) is coupled with different relative humidities between 88%-95%. Incubation temperatures and times and the relative humidity are dependent on type and diameter of the sausage being produced, generally a pH endpoint of <4.7 is the target. Fermentation takes between 1 – 3 days.

(vi) heating (of some products) to inactivate the inoculum and eliminate pathogens, generally not a stage in uncooked product, see definition on page 11 above by FSANZ,

(vii) drying (and sometimes smoking), the drying chamber is between 12.9 and 15.7°C. Drying takes place for ≥12 days up to several weeks until a specified moisture/protein endpoint is reached, depending on the product (Ross and Shadbolt, 2001).

3.2 Hurdles

Hurdle technology "advocates the deliberate combination of existing and novel preservation techniques in order to establish a series of preservative factors (hurdles) that any microorganisms present should not be able to overcome" (Leistner and Gorris, 1995). It is an essential concept for the preservation of foods using "mild" techniques as it allows for the concerted and sometimes synergistic effects of a number of preservative factors that may be required at lower levels than if only one preservative factor, or hurdle, was used. In many cases none of the individual hurdles would be able to provide for a safe product on their own. Salami is an excellent example of a food that is preserved by the application of multiple hurdle technology where the hurdles used are the presence of nitrite, a low redox potential, a preferred competitive flora (inoculum), salt, low pH (from lactic acid), a low water activity and in some cases the presence of antibacterial compounds resulting from smoking.

Each producer of raw fermented sausages will have a recipe and process unique to the establishment, which is regarded as intellectual property and is commercially important to the manufacturer. In Europe considerable differences exist in the styles of the products and this may be reflected in both the recipe formulation and the nature and outcome of each processing step. For example, some products may be quick fermented while others take many days or even weeks.

It was demonstrated in 1992 that *E. coli* O157:H7 can survive but not grow during fermentation, drying and storage (for 2 months) of salami (Glass *et al.*, 1992). Given that the processing of UCFM products is varied and complex, further details relevant to STEC follow.

3.2.1 Batter

The treatment of the batter prior to processing has been shown to have some effect on the survival of *E. coli* O157:H7 (Faith *et al.*, 1998a). Various treatments of the batter (i) refrigerated, (ii) frozen and thawed or tempered, (iii) frozen and thawed were investigated and shown to influence the final numbers of *E. coli* O157:H7. Treatments (ii) and (iii) resulted in higher D reductions (2.1 and 1.6 $\log_{10}$ cfu/g respectively) compared to treatment (i) (1.1 $\log_{10}$ cfu/g) when assessed immediately after drying. The relativity persisted throughout storage at 21°C and 4°C. There seemed to be some advantageous effect of the freezing component of the pre-treatments, possibly by the production of injured cells, which were more susceptible to the treatments that followed.

3.2.2 Production

Table 4 shows data from a number of studies of initial and final concentrations of *E. coli* O157:H7 resulting from salami production.

Table 4: Effect of UCFM product fermentation and drying on numbers of *Escherichia coli* O157:H7

Fermentation conditions	Drying conditions	Concentration of *Escherichia coli* O157:H7 (log_{10} cfu/g)			Overall D reduction	Reference
		Initial	After fermentation	After drying		
24°C, 90% RH to pH ≤ 4.8 (60-64 h)	13°C, 65% RH to M/Pr ratio ≤ 1.9:1 (approx. 21 days)	7.5-7.7	6.0 - 6.8	5.4-6.6	0.9-2.1	Faith *et al.*, 1998a
36°C, 90% RH to pH< 4.8 (14-16h)	13°C, 65% RH to M/Pr ratio ≤ 1.6:1 (12-16 days)	7.5	7.0 (15% fat) 6.9 (20% fat) 6.4 (30% fat)	6.5 6.2 5.8	1.0 1.3 1.7	Faith *et al.*, 1998b
26°C, 85% RH to pH ≤ 5.0 (14-18 h)	13°C, 65% RH to M/Pr ratio ≤ 1.6:1 (15-21 days)	6.9	6.8	5.7 (21 days drying)	1.2	Hinkens *et al.*, 1996
36°C, 90% RH to pH ≤ 4.8 (16 to 20 h)	13°C, 65% RH to M/Pr ratio ≤ 1.6:1 (18 days)	7.8	6.8	5.9	1.9	Faith *et al.*, 1997
22°C, 50% RH for 3 days then 9°C, 40% for 18 hours	38°C 70% RH or 22°C 50% RH for 3 days	7.6 7.6	NR NR	3.0 (+ starter cult.) 6.2 (- starter cult.) 6.7 (+ starter cult.) 7.5 (- starter cult.)		Calicioglu *et al.*, 2002
24°C, 90-95% RH, 3 days	22°C, 80-85% RH until 40% moisture achieved	7.7	NR	+ starter culture 5.7 - starter culture 7.4	2.0 0.3	Calicioglu *et al.*, 2001
29 rising to 41°C, 80% RH to pH 4.6 or 5.0	49 rising to 66°C (sausage internal temp 54°C), 60% RH	8.0 7.8	6.6 (to pH 4.6) 7.5 (to pH 5.0)	<1.0 after heating. 4.6 after heating 2.8 after heating and holding for 30 min <1.0 after heating and holding for 60 min	>7.0 3.2 5.0 >6.8	Calicioglu *et al.*, 1997
21 rising to 38°C, 75% RH to pH 4.8	15°C 64-70% RH until a_w <0.8 (approx. 7 days)	6.7	6.3	5.9	0.8	Riordan *et al.*, 1998
15.6 rising to 35.6°C, to pH 4.8 (13-14 h)	12.8°C, 70% RH until M:Pr ratio <1.9:1 (18-21 days)	4.7	4.4	3.7	1.0	Glass *et al.*, 1992

RH = Relative humidity, M/Pr = moisture to protein ratio, NR = Not Reported

Table 4 shows that fermentation and drying alone do not result in large reductions in the number of *E. coli* O157:H7 with most processes resulting in an approximate 2D reduction in numbers.

In experiments where various parameters in salami manufacture (e.g. NaCl and pH) have been adjusted, a rapid fall in pH to a suitably low level in the presence of a mild heat treatment has been shown to be pivotal in the destruction of *E. coli* O157:H7. Addition of preservatives that inhibit the action of the starter cultures impedes acid production and results in reduced inactivation of *E. coli* O157:H7 (Chikthimmah *et al.*, 2001).

Experiments with pepperoni showed that the conventional recipe and process resulted in a 0.84 D reduction in numbers, while by increasing the NaCl and sodium nitrite concentrations and using a lower final pH (4.4-4.6) this could be increased to a 4.79 D reduction (Riordan *et al.*, 1998).

The concept of cross-protection of *E. coli* O157 from death because of the general stress response was investigated (Semanchek and Golden, 1998). They examined the influence of growth temperature (10°C and 37°C) heat (52, 54 and 56°C), lactic acid (pH 3.2, pH 2.8 and pH 2.5) and freezing-induced inactivation and injury (-20°C up to 7 months) on *E. coli* O157:H7. Three strains were used, originally obtained from outbreaks involving salami, apple cider and ground beef.

As shown in Table 5, the salami outbreak strain was more resistant than beef or cider strains to heat, lactic acid or freezing. The salami environment may select for resistance of bacterial strains when suboptimal conditions ("high stress") are encountered.

Table 5: D values and maximum injury of beef, salami and cider isolates under various stressors

Mean D values (min)								
Growth temp (°C)	**Strain**	**Heated at (°C)**			**Lactic acid soln. (%)**			**Freezing (-20°C)**
		52	**54**	**56**	**0.1**	**0.25**	**0.5**	
10	beef	11.2	4.1	2.5	3.7	1.2	0.35	1.3
	cider	40.7	12.4	5.1	5.3	1.8	0.44	1.7
	salami	37.6	15.9	5.9	9.5	3.6	0.76	1.4
37	beef	17.7	12.9	9.3	6.4	4.2	1.7	1.6
	cider	89.4	29.4	14.1	4.8	2.3	0.47	2.2
	salami	120.5	59.7	26.4	14.8	8.8	1.9	1.7

Source: Semanchek and Golden (1998)

The effect of cross-protection was further investigated in simulated meat and fermented meat systems by Uyttendaele *et al.* (2001). The authors provided data to suggest that survival could be longer under more extreme conditions (e.g. survival at pH 4.5 > pH 5.4 >7.0 at 7 and –18^{o}C). It was suggested that this is because multiple challenges to the organism result in a general stress response, and that the use of hurdle technology would actually result in increased survival. Genes involved in stress response may also make the organism more virulent.

Some investigation into the survival of *E. coli* O111 in Hungarian salami has also been carried out (Pidcock *et al.*, 2002). Salami batter was inoculated with the pathogen and various starter organisms, followed by fermentation at 25°C for 7d (conditions not typical for this product where fermentation is usually for 72 h). With a commercial starter culture, only around a 0.5 D reduction in *E. coli* O111 had occurred after 72 h, while the addition of other cultures could improve on this only by the order of 0.5-1.0 D.

The survival of *E. coli* O157:H7 in Turkish soudjouck (a spicy hot, semi-dry sausage) has been researched (Cosansu and Ayhan, 2000). The batter was inoculated at 10^5 cfu/g and refrigerated overnight at 4°C (population 5.66 log cfu/g) before stuffing the batter into cow intestine casings. Enumeration of the organism was carried out over the eight days of fermenting and drying. Fermentation progressed at 24±2°C and 90-95% relative humidity for 3 days (1st day 5.85 log cfu/g; 2nd day 5.31 log cfu/g; 3rd day 4.54 log cfu/g) followed by drying for 5 days at 22±2°C at a relative humidity of 80-85% (4th day 4.26 log cfu/g, 6th day 3.27 log cfu/g and 8th day 2.01 log cfu/g). Half of the soudjouck was vacuum-packed, the remainder left open to the atmosphere. All samples were then stored at 4°C (55% relative humidity) for 3 months. At this stage no pathogens could be detected. The organism thus decreased by approximately 3-log units during fermentation and drying, though it then survived for longer in vacuum-packed samples (>2 months) than open-air samples (>1 month). For example, after 2 months, in vacuum-packed samples, there were 0.23 log cfu/g but none were found in the open air samples.

Further research into survival of the organism in soudjouck (Calicioglu *et al*., 2002) led to the conclusion that naturally fermented, old-country-type sausage may allow survival of *E. coli* O157:H7 in the absence of a controlled fermentation, post-fermentation cooking and/or an ambient-storage processing step.

3.2.3 Storage

Data for the effect of storage conditions on the survival of *E. coli* O157:H7 in UCFM products are shown in Table 6. Storage at room temperature under air is an effective way to decrease numbers of *E. coli* O157:H7 in correctly manufactured salami. However, such storage may result in undesirable product characteristics and control of microbiological hazards other than *E. coli* O157:H7 also have to be considered. Slightly less effective but technologically acceptable storage conditions may need to be used to increase inactivation. Heat shocking of cells was also found to increase inactivation during storage. It should be noted that both papers by Faith *et al.* (1997, 1998a) and Clavero and Beuchat (1996) focused on sliced (as opposed to whole) salami only.

Table 6: Data for reductions in *Escherichia coli* O157:H7 numbers during storage of UCFM products

Conditions	Temperature (°C)	Storage time (days)	Concentration of *E. coli* O157:H7 (log_{10} cfu/g)		D reduction	Reference
			Initial	Final		
Air	4	90	5.4-6.6	<1-3.0	2.4->6.6	Faith *et al.*, 1998a
	21			<1	>5.4->6.6	
Vacuum	4	90	5.4-6.6	1.4-3.6	1.8-5.2	Faith *et al.*, 1998a
	21			<1	>5.4->6.6	
Air						Faith *et al.*, 1998b
15% fat	4	28	6.5	6.0	0.5	
20% fat	4	28	6.2	5.8	0.4	
35% fat	4	28	5.8	5.5	0.3	
15% fat	21	14	6.5	2.0	4.5	
20% fat	21	14	6.2	1.4	4.8	
35% fat	21	14	5.8	1.3	4.5	
Vacuum						Faith *et al.*, 1998b
15% fat	4	28	6.5	5.8	0.7	
20% fat	4	28	6.2	5.8	0.4	
35% fat	4	28	5.8	5.6	0.2	
15% fat	21	14	6.5	3.0	3.5	
20% fat	21	14	6.2	2.0	4.2	
35% fat	21	14	5.8	1.2	4.6	
Air	-20	90	5.2	3.6	1.6	Faith *et al.*, 1997
	4	90	5.9	3.7	2.2	
	21	28	5.5	<1.0	>4.2	
Vacuum	4	90	6.0	4.3	1.7	Faith *et al.*, 1997
	21	90	5.6	<1	>4.6	
CO_2	-20	90	5.1	3.9	1.2	Faith *et al.*, 1997
	4	90	5.8	4.1	1.7	
Vacuum						Calicioglu *et al.*,
+ starter	21	28	5.7	<1	>4.7	2001
- starter		28	7.4	5.4	2.3	
+ starter	4	28	5.7	3.7	2.0	Calicioglu *et al.*,
- starter		28	7.4	7.2	0.2	2001
pH 5.0, heat	25	7	4.6	4.1	0.6	Calicioglu *et al.*,
to 54°C	4	7	4.6	4.7	0.0	1997
pH 5.0, heat	25	7	2.8	<1.0	>1.8	Calicioglu *et al.*,
to 54°C,	4	7	2.8	2.0	0.8	2001
hold 30 min						
pH 4.8,	4	56	3.7	2.7	1.0	Glass *et al.*, 1992
vacuum						
packed						
pH 4.8, a_w	5	32 (UH)	4.9	2.4	2.5	Clavero and Beuchat,
0.95	5	32 (HS)	4.5	1.1	3.4	1996
	20	16 (UH)	4.9	1.9	3.0	
	20	4 (HS)	4.5	1.9	2.6	
pH 4.63, a_w	5	32 (UH)	4.9	2.2	2.7	Clavero and Beuchat,
0.90	5	32 (HS)	4.5	1.2	3.3	1996
	20	16 (UH)	4.9	1.9	3.0	
	20	4 (HS)	4.5	2.1	2.4	

HS= heat stressed, UH=unheated

It has been suggested that exposing the pathogen to acid or alkali stress may cross protect cells against further stresses, for example heat, salt and irradiation. Alkaline cleaners are widely used in meat processing to remove fats and proteins from equipment so the survival and growth of *E. coli* O157:H7 in salami after the pathogen has been exposed to an alkaline cleaner (pH 11.18) for two minutes was investigated (Sharma *et al.,* 2004). After exposure to the cleaner or control (sterile peptone water, pH 6.9) solution, the pathogen was inoculated into a hard salami (pH 4.9) at a low prevalence (0.003 – 0.582 cfu/g) and high prevalence (0.69 – 31.5 cfu/g). The salamis were then stored at 4, 12 and 20°C. Fortunately, treatment with alkaline cleaner did not cross protect cells against the low pH conditions found in salami, when compared to the control solution.

Irradiation of meat prior to the production of pepperoni to give a >5 $\log_{10}$ reduction in *E. coli* O157:H7 produced a food with acceptable organoleptic qualities, unlike heat-treated salami where texture and colour were adversely affected (Johnson *et al.*, 2000).

Experiments to determine the survival of *E. coli* O157:H7 inoculated onto salami (pepperoni) slices on top of frozen pizza showed, unsurprisingly, that the amount of inactivation was dependent on the time and temperature of cooking (Faith *et al.*, 1998b). Baking of the pizza at 246^{o}C for 15 minutes or at 191^{o}C for 20 minutes was necessary to reduce the numbers from around 5 $\log_{10}$ cfu/g to non-detectable by enrichment.

An extensive combination of trials in Canada (Naim *et al*., 2003) focused on the survival of *E. coli* O157:H7 in fermented dry sausages, particularly the influence of inoculum preparation, inoculation procedure and selected process parameters. Exposure to acidic conditions found that when the pathogens were grown in a mild acidic broth (pH 4.8) there was no effect on the later survival of the pathogens under pH 2 conditions. However the same strains became sensitive to acidity after 7 days of incubation on the surface of refrigerated beef (the real life scenario). In subsequent sausage production trials, *E. coli* O157:H7 strains inoculated into the batter mix survived. However an approximate 2-log reduction occurred in pathogen numbers when samples were dried to a water activity of 0.91, irrespective of fermentation temperature. When a 5-day pre-drying holding stage was introduced at fermentation temperature of 37°C, inactivation was significantly increased ($P< 0.05$) (but not at 24°C fermentation). The authors concluded that the levels of inactivation following batter inoculation were similar to previous reports i.e. reduction of 2.17 + 0.44 log cfu/g when water activity reached 0.91. The only significant pathogen reduction (4 – 5 log cfu/g) was achievable when the product was extensively dried to a water activity of 0.79.

3.2.4 Conclusion

In conclusion, the production process and storage of UCFM, if properly controlled, may each cause reductions of approximately 2 $\log_{10}$ cfu/g in STEC numbers. More extreme conditions (e.g. longer storage times up to 3 months, lower pH or water activity) or mild post-production heat treatment, are required to achieve more significant reductions in numbers, thereby limiting application to a small range of products.

These results indicate that prevention of contamination of ingredient meats remains an important component of risk management for STEC in UCFM.

3.3 The Food Supply in New Zealand

According to data from Statistics New Zealand, as of February 2001 there were 75 business enterprises engaged in "Bacon, Ham and Smallgood Manufacturing" (New Zealand Standard Industrial Category C211300). These represented 85 geographic locations (i.e. some enterprises operated at more than one location). However, only a proportion of these businesses will be involved in the production of UCFM. The actual number of businesses involved in producing UCFM is probably approximately ten, including six major manufacturers (Francis Clement, Pork Industry Board, personal communication).

The New Zealand Pork Industry Board (NZPIB) has provided an estimate of total UCFM annual production in New Zealand at 343,367 kg (343 tonnes).

3.3.1 Imported food

Data from Statistics New Zealand show that meat preparations (sausages and similar products of meat, meat offal or blood, and food preparations based on these products, with a non-poultry base) (Code 1601.00.00.29) comprised 227 tonnes for the year ending September 2005. The majority came from Australia (208 tonnes; 92%) and Canada (13 tonnes; 6%). This import category includes salami as well as frankfurters, liver sausages, saveloys, bologna, paté, meat pastes etc., so the amount of imported UCFM products is likely to be much less than 227 tonnes. The quantity of UCFM imported from Australia remains unknown.

4 HAZARD CHARACTERISATION: ADVERSE HEALTH EFFECTS

Infection with STEC results in the organism invading the gut and then producing one or more toxins. Toxins are not produced in foods, but only after infection.

This can cause a wide range of outcomes. Some cases will be asymptomatic, others will experience diarrhoea, and a proportion will go on to suffer more serious outcomes including haemorrhagic colitis (HC), haemolytic uraemic syndrome (HUS), thrombotic thrombocytopaenic purpura (TTP) and death (Desmarchelier and Grau, 1997).

4.1 Symptoms

Incubation: 3 to 9 days (mean 4 days) following ingestion of the bacteria.

Symptoms: Diarrhoea is accompanied by severe abdominal cramps. Vomiting may occur (30-60% of cases) but fever is infrequent (less than 30% of cases) (Dundas and Todd, 2000).

Condition: More serious consequences of infection include:

> *Haemorrhagic Colitis (HC):* Bloody diarrhoea, inflammation of the large bowel, severe abdominal pain, vomiting, no fever.
>
> *Haemolytic Uraemic Syndrome (HUS):* HUS follows HC and is normally associated with children. The condition is characterised by renal failure and the consequences of that including seizures, coma, death.
>
> *Thrombotic Thrombocytopaenic purpura (TTP):* A version of HUS most often experienced by the elderly. Involves loss of platelets, skin coloration, fever and nervous system disorder (seizures and strokes) in addition to HUS signs and symptoms. There is no prior episode of diarrhoea. Illness lasts from 2-9 days.

Treatment: Dialysis, maintenance of fluid balance and treatment of hypertension in cases of HUS.

Long Term Effects: HUS: kidney problems, hypertension, neurological deficits.

HUS has been estimated to occur in approximately 4% of cases (Mead *et al.*, 1999). HUS is the most common cause of acute renal failure in children. Mortality is approximately 5% and approximately 10% of survivors are left with severe sequelae (Park *et al.*, 1999).

4.2 Serotypes Causing Disease

E. coli O157 is the most commonly documented serotype causing human illness. However, over 200 non-O157 STEC serotypes have been isolated from humans and are clearly recognized as human pathogens. The World Health Organisation has identified the most important non-O157 STEC serogroups, from an epidemiologic perspective, as O26, O103, O111 and O145 (WHO, 1998). An updated list of STEC, with literature references, is maintained by Dr K. Bettelheim (National *E. coli* Reference Laboratory, Melbourne, Australia) and can be found on the World Wide Web at:

http://www.microbionet.com.au/frames/feature/vtec/brief01.html

The majority of notified STEC infections in New Zealand are caused by *E. coli* O157 (H7 or H-). Other serotypes causing infections have included O113:H21, O26:H-, O91:H21, O145:H-, ONT:H18, ONT:H6, and O128:H-.

Analysis of patient stool samples submitted to the Dunedin Hospital laboratory during 1996 identified two serotypes, O26:H11 and O128:H2, that were toxigenic and typable (Brooks *et al.*, 1997).

Further details of serotypes found in New Zealand are given in Section 6.1.3.

STECS have also been classified on the basis of a genomic pathogencity island of DNA (OI-122) which is physically linked to the locus of DNA containing the pathogenicity factor *eae.* These 5 Karmali groups A through to E are classified based on their frequency in human cases, frequency of involvment in outbreaks, and aossciation with severe disease (HUS). The groups are: Seropathotype A (O157:H7 and O157:NM), Seropathotype B (O26:H11, O103:H2, O111:NM, O121:H19 and O145:NM) and Seropathotype C (O91:H21 and O113:H21). Group D is associated with diarrhoea but not outbreaks or HUS and Group E are not implicated in human disease (Karmali *et al.,* 2003).

4.2.1 Overview of international situation

It has long been held that serotype O157 is the predominant cause of STEC related disease in the USA. However, some recent data indicate that there may be a re-thinking of this position. In a recent review of the impact of foodborne disease in the USA, Mead *et al.* (1999) estimated that illness attributable to non-O157 STEC was approximately 50% of that caused by *E. coli* O157:H7. If these estimates are correct then approximately 33% of STEC-related illness is caused by non-O157 serotypes in the USA, and this represents a major shift in the way this group of organisms is regarded.

A study from Canada (Rowe *et al.*, 1993) reported that of 30 isolates from HUS patients 26 were *E. coli* O157:H7 and four belonged to other serotypes (two of the isolates could not produce verotoxin and so may have not caused the disease, although expression of toxin can be lost on subculture). An earlier study in Alberta (Pai *et al.*, 1988) of faecal samples submitted at hospitals for bacteriological examination found 130 patients infected with *E. coli* O157:H7, 29 with non-O157 STEC and seven with both.

Bitzan *et al.* (1991) demonstrated that 20 of 22 HUS patients in Germany had been infected with type O157, one with O26 and one with O55. This represents an approximate 10% of the cases being caused by non-O157 serotypes.

An Italian study into HUS cases (Luzzi *et al.*, 1995) revealed a somewhat higher proportion of non-O157 cases, with 45 cases having antibodies to O157, 12 to O111, 6 to O26 and 2 to O103 (30.8% non-O157), although the significance of antibodies to STEC remains controversial. In Britain a similar proportion (28.3%) of non-O157 STEC has been recorded in children with HUS (Kleanthous *et al.*, 1990), although an earlier study had shown a smaller proportion, 21% (Scotland *et al.*, 1988).

In Belgium, only 18% of STEC strains were reported to belong to serotype O157:H7 (Pierard, 1992), and a French study reported isolating only O103:H2 from the faeces of six of 69 HUS patients, i.e. no other STEC were isolated (Mariani-Kurkdjian *et al.*, 1993). A more recent French study focused on children with HUS found that 86% of these cases had evidence of STEC infection. Of the HUS cases, 75% showed evidence of infection from *E. coli* O157, but other serotypes identified included O103, O126 and O26 by microbiological testing and, in addition, O9, O103 and O145 by serum antibody testing (Decludt *et al.,* 2000).

Caprioli *et al.* (1997) observed that during 1996 there was a sudden increase in the proportion of non-O157 isolations in Europe. In HUS cases from 1996 up to the time of publication 11% were caused by O103 and 33% by O26 compared to 1.5% and 6.6% respectively in previous years. This trend was describes as "worrisome" because of the lack of implementation of reliable methods for detecting these infections.

The pattern of transmission of sporadic STEC infection in continental Europe may be atypical because of the lack of an epidemiological link between STEC infection and beef products (Pierard *et al.*, 1999).

Tamura *et al.* (1996) reported on investigations of diarrhoeal specimens tested from Asian countries. Only 20.3% of the isolates typed were of serotype O157. The other serotypes identified were similar to those identified in other countries.

Australia has been known to be unusual in respect to STEC types isolated, as type O157 represents a low proportion of the isolates (Goldwater and Bettelheim, 1995), with type O111:H- being more common.

4.3 Dose Response

The survival of *E. coli* (one non-pathogenic strain and one enterohaemorrhagic strain) during passage through the stomachs of young and elderly people has been investigated using mathematical modeling, and a fermentor that mimicked the human gastric pH (Takumi *et al.*, 2000). On average 20-80% of the ingested *E. coli* were estimated to arrive at the small intestine without inactivation by low pH. This was attributed to the temporary increase in gastric pH after consumption of food, as well as acid tolerance of *E. coli*. To illustrate this last point, the *E. coli* O157:H7 isolate tested showed no decline in numbers after incubation for two hours at pH 2.5, and 26% of the cells survived when the pH was 2.0.

This acid tolerance and gastric survival will presumably be reflected in the dose response relationship.

4.3.1 Dose-response for *Escherichia coli* O157:H7

Haas *et al.* (2000) developed a dose-response relationship for *E. coli* O157:H7 based on a prior animal (rabbit) relationship. This model was validated by reference to two well documented human outbreaks; one involving water-borne organisms and the other involving venison jerky. The model gave a dose for infection of 50% of the exposed population of 5.9×10^5 organisms and a risk for consumption of 100 organisms of 2.6×10^{-4}.

Based on a retrospective analysis of foods involved in outbreaks, the capability of person-to-person transmission, and the ability of the pathogen to tolerate acidic conditions, which enables survival in the acidic environment of the stomach, Doyle *et al.* (1997) estimated the infectious dose of *E. coli* O157:H7 to be less than a few hundred cells. A similar estimate of infectious dose has been proposed by CAST (1994). However, the concept of an infectious dose has now been replaced by estimates of the probability of infection from exposure to differing numbers of cells.

Recently an estimate of the dose response for *E. coli* O157:H7 using a beta-Poisson model gives a value of 1.9×10^5 cells as the median dose (50% exposed become symptomatic), with a probability of 0.06 (6×10^{-2}) of infection when exposed to 100 cells (Powell *et al.*, 2001).

Following the outbreak of *E. coli* O157 in the USA in 1995, Tilden *et al*., (1996) calculated that the dose response was less than 50 bacteria.

4.3.2 Dose-response for non-O157:H7 STECs

Haas *et al.* (1999) developed dose-response relationships for *E. coli* O111 and O55 using human volunteers. The relationship gave a dose for infection of 50% of the exposed population of 2.6×10^6 organisms and a risk for consumption of 100 organisms of 3.5×10^{-4}.

5 EXPOSURE ASSESSMENT

5.1 The Hazard in the New Zealand Food Supply: STEC in Meat and UCFM

The faeces of ruminant animals are most likely to be the primary source of meat contamination with STEC. Section 5.1.1 discusses prevalence in cattle and sheep; no data could be located for goat, pig or deer faeces.

5.1.1 Prevalence of STEC in ruminant faeces and pasture

E. coli O157 is a transient contaminant of the gastrointestinal tract of ruminants (Sanchez *et al.*, 2002), with infection usually lasting one to three months (Mechie *et al.*, 1997). Two published surveys have evaluated the prevalence of STEC in New Zealand bovines.

Buncic and Avery, (1997) sampled the faeces of 371 cattle from 55 farms on arrival at a single slaughterhouse in the Waikato area. Two (0.54%) of these samples yielded *E.coli* O157. A further 160 cattle from the farm of one of the positive animals tested negative for *E. coli* O157:H7. More recently, Cookson *et al*., (2003; 2006) surveyed faecal swabs taken from 187 cattle (91 weaned calves, 24 heifers and 72 dairy cattle) and 132 sheep in the lower North Island. The swabs were cultured for *E. coli* and isolated colonies were analysed for the genes involved in virulence. Cattle results were reported as a group, and not differentiated into dairy or beef cattle. STEC were detected in 51 cattle faecal samples (27%) and the *eae*A gene was isolated from 36.5% of the positive samples. In sheep faeces, STEC were detected from 65.9% of samples and the *eae*A gene was isolated from 27.3% of the positives. Overall, 23 isolates were *Stx*1/2 and *eae*A positive and all contained the enterohaemolysin (Ehly) gene (*ehx*A). *E. coli* O157:H7 was not detected in any sample. Several clinically important isolates were detected, including O5:H-. O26:H11, O84:H-/H2, O91:H- and O128:H2.

In the faeces of infected cattle, the concentration of *E. coli* O157 can vary greatly (<100 to $\geq 10^8$ cfu/g). The degree of shedding has been correlated with age, with calves (2 months to 2 years of age) generally excreting higher concentrations than adults, although Ezawa *et al.* (2004) reported heifers were more likely to be infected than calves and other cattle. Increased shedding occurs in dairy cows in the first month of milking (Mechie *et al.*, 1997). In longitudinal studies, all cows in a herd are likely to become shedders at some point with individuals having highly variable *E. coli* O157 concentrations. Some cows can become persistent shedders. However, poor homogeneity of the pathogen in faecal samples can also affect representative sampling and hence the results obtained (Pearce *et al.,* 2004).

Studies on the survival of O26, O111, and O157 in bovine faeces at 5°C, 15°C and 25°C found that all three pathogens survived at 5°C and 25°C for 1 to 4 weeks. At 15°C, survival was longer at 1 to 8 weeks (Fukushima *et al*., 1999).

5.1.2 STEC in meat

The New Zealand Ministry of Agriculture and Forestry has been monitoring meat products for the presence of *E. coli* O157 since 1998 (see Section 7.1.1.2). Baseline surveys of bovine (2400) and ovine (500) carcasses from meat processing plants did not detect any *E. coli* O157:H7. Records from the National Microbiological Database (NMD) indicate that to January 2007, *E. coli* O157 was detected from 0.006% of 284,554 cartons of bulk beef (95% confidence limits, 0.003%-0.035%) and 0.6% (CI, 0.5-3%) for bobby veal (Dr Roger Cook,

NZFSA, personal communication, February 2007). While these samples were all taken from export meat works their ultimate destination may have been either export or domestic consumption. Approximately 61% of total domestic beef supply originates from export meat works (Roger Cook, NZFSA, personal communication). The remaining 39% of domestic beef supply comes from local abattoirs and is not subjected to the *E. coli* O157 testing programme. The prevalence of *E. coli* O157:H7 in meat produced by local abattoirs is therefore not known.

No co-ordinated *E. coli* O157 testing programme is currently in place for ovine or porcine meat produced in New Zealand (Roger Cook, NZFSA, personal communication).

In addition to the isolates from human cases, the ESR Enteric Reference Laboratory has been asked to serotype STEC isolates from meat samples. The isolates derive from meat produced in New Zealand, but the details have not been made available for commercial reasons. Nevertheless, the serotypes have been reported (Bennett and Bettelheim, 2002). Seven isolates from New Zealand beef were typed, and found to belong to four different serotypes. From sheep meat, forty isolates were identified as including 18 different serotypes (although many of these were non-typable or rough). Seven of the meat isolate serotypes (one from beef and six from sheep meat) have also been associated with human disease. Three of the isolates (O5:H-, O91:H-, O104:H-) have been associated with cases of HUS, while the others (O6:H-, O104:H7, O128:H2, O163:H19) have been associated with diarrhoea only.

Up until 2004, there were three limited and localised surveys of STEC in meat; Hudson *et al.,* (2000), Brooks *et* al., (2001), and Hudson (2001). Details from these surveys are given below.

A study of the isolation of STEC using a specific agar examined fifteen retail meat products; five raw minced beef or pork products, six vacuum-packed cooked sliced meat products and four vacuum-packed salami varieties (Hudson *et al.*, 2000). Four of the five minced beef or pork samples yielded presumptive STEC colonies, of which all but one were serotype O163:H19. This serotype is not known to cause HUS. The remaining isolate was non-typable.

Brooks *et al.,* (2001) examined beef (91 samples), mutton and lamb (37 samples), pork (35 samples), chicken (36 samples), mutton/beef mince (10 samples), and sausage mixtures (9 samples) obtained from Dunedin supermarkets and butcheries. STEC were isolated from 12% of the beef samples, 17% of lamb, and 4% of pork; chicken samples tested were negative. Serotypes obtained were, from beef: O128:H2, ONT:H21, O144:H2, O27:H21, ONT:H-, O8:H-, O15:H27, O81:H26, from lamb: O91:H-, O171:H2, ONT:H4, O128:H-, O81:H26, O5:H-, from pork: O156:H-, and from beef and lamb mince O15:H27.

All isolates were tested for the presence of factors associated with virulence i.e. *Stx*1, *Stx*2, Ehly (i.e. *hlyA*) and *eaeA*. All were positive for *Stx*1 and/or *Stx*2, and five (ONT:H21, O128:H2, O144:H2, O81:H26, O5:H-) were positive for Ehly. None were positive for the *eaeA* factor.

Serotypes O5:H-, O128:H-, O128:H2, and O91:H-, have been reported to be involved in diarrhoea and HUS cases, but not outbreaks, overseas. Serotype O128:H- has caused a case of STEC infection in New Zealand.

Work carried out by Hudson (2001) for the Ministry of Health during 2000-2001 tested 300 retail meat samples (minced or cubed) for STEC. Meat types were 97 beef, 64 chicken, 66 lamb and 73 pork. Five samples out of the 300 were positive (1.7%) and all were lamb. The samples provided seven isolates, characterised as follows;

Sample	**Serotype**	***Stx*1**	***Stx*2**	***Stx*1 & *Stx*2**	***eae*A**	***hly*A**
Lamb 1	ONT:HNM	-	-	+	-	+
Lamb 1	ONT:H8	-	-	+	-	+
Lamb 2	Orough:HNM	-	-	+	-	+
Lamb 2	O123:H51	-	-	+	-	+
Lamb 3	O123:H51	-	-	+	-	+
Lamb 4	O75:H8	-	-	+	-	+
Lamb 5	O128:H2	-	-	+	-	+

(ONT = non-typable from O antigen, O rough = agglutinates with all sera).

The first six serotypes do not appear to have caused disease in humans, the last serotype O128:H2, has been associated with illness.

The prevalences found in this study were low, but the method used was focused on obtaining isolates that could be typed. Other methods such as PCR detection might have given higher prevalences but not necessarily have yielded any isolates for serotyping.

STEC in retail meats across New Zealand has been the subject of a national study (Wong *et al*., ESR, Christchurch, unpublished). Five types of raw retail meats (excluding poultry) from August 2003 to May 2005 were sampled and the prevalence of STEC determined from a total of 878 samples. The results are given in Table 7.

Table 7: Prevalence of STEC in retail uncooked meats in New Zealand

Sample numbers	**Beef n=233**	**Unweaned veal n=183**	**Lamb/mutton n=231**	**Pork n=231**
No. samples positive for STEC[#]	12 (5.2%)	4 (2.2%)	34 (14.7%)	15 (6.5%)
No.samples positive for O157:H7	0	1 (0.5%)	3 (1.3%)	1 (0.4%)
No.samples positive for O26:H11	0	2 (1.1%)	0	0

An unpublished pilot survey of pork carcasses originating from Australia, New Zealand, Canada and USA has also been undertaken (Wong *et al*., ESR, Christchurch, unpublished) in order to establish a baseline for a National Microbiological Database for domestic pork. Swabs or excised meat samples were tested for the presence or absence of *E. coli* 0157 and quantitatively for generic *E. coli.* For New Zealand produced carcasses, 1 % (1/100) tested positive for *E. coli* 0157. The prevalence on Australian pig meat samples was 3.1% (2/65). In comparison, *E. coli* O157:H7 was not detected in imported (uncooked) Canadian or US pork. The overall prevalence of *E. coli* O157:H7 in pig meat imported into New Zealand was 1.8%.

Samples of lean boneless beef trim (destined for ground beef) imported into the USA from New Zealand, Australia and Uruguay have been tested for non-O157 STEC and compared to USA domestically produced product, *E. coli* O157 was not isolated (Bosilevac *et al.,* 2007). There were 223 samples of New Zealand origin beef tested. The prevalence of non-O157 STEC was 9.7% in the New Zealand samples (23 samples were positive for either or both of the *stx* genes, detected by PCR from enrichments). An STEC isolate was recovered from 4 of these positive samples (thus confirming the source of the gene), and 2 of these have been associated with HUS.

5.1.3 STEC in UCFM products

There are few data on the prevalence of STEC in UCFM in the New Zealand domestic food supply. The only data found were derived from a study by Hudson *et al.,* (2000) who examined four retail vacuum-packed sliced salami samples from different manufacturers prior to inoculation experiments. STEC was not detected.

5.1.4 Conclusions

The data above indicate that *E. coli* O157 is infrequently found in New Zealand cattle and sheep faeces. A survey to detect non-O157 STEC in cattle and sheep faeces found a much higher prevalence, including several clinically important isolates.

The same pattern is found from the meat testing data; isolation of *E. coli* O157 from New Zealand beef is rare (at least in beef for export), while other STEC of varying pathogenic potential are more common. The STEC profile for veal, sheep and pork meat on the domestic market is likely to be similar to that from export premises as processing is similar and source farms are generally not separate to those for export.

The prevalence of STEC in red meat has been the subject of a previous risk profile, see website; http://www.nzfsa.govt.nz/science/risk-profiles/stec-in-red-meat.pdf. This document was published in August 2002.

While quantitative data are lacking, these data clearly show the potential for UCFM ingredient meats to be contaminated with pathogenic STEC.

5.2 Food Consumption: Uncooked Comminuted Fermented Meat Products

Analysis of 24 hour dietary recall records from the 1997 National Nutrition Survey (NNS; Russell *et al.*, 1999) only identified one descriptor (salami) which could be used to describe an UCFM product. It should be noted that not all products described as 'salami' will be UCFM, as some cooked and unfermented products are commonly referred to as salami.

Only 73 NNS respondents (1.6%) reported consuming salami in the previous 24 hour period, mainly as a component of sandwiches, filled rolls or bagels. Salami will be a common component of pizza, a more commonly reported food in the 1997 NNS, however, the thermal processing which salami will receive during pizza manufacture means it is not relevant to the current Risk Profile and has been excluded.

The mean serving size of uncooked salami consumed by the 73 respondents was 33.4 g, while the median serving size was 15.5 g, with a range from 2 to 276 g. The mean daily consumption of uncooked salami by the whole population would be approximately 0.5 g/day.

The New Zealand Pork Industry Board (NZPIB) has provided an estimate of total UCFM annual production in New Zealand at 343,367 kg. Based on a total population of 3,737,490 (2001 Census) this equates to 0.25 g/person/day. The difference between this estimate of consumption and that in the previous paragraph may be due to a range of factors. The higher figure (0.5 g/person/day) may include some imported product and/or product which has been cooked during the production process.

For Australia, the percentage of the population consuming salami of all types without further cooking has been estimated as 1.5% (FSANZ, 2003. Attachment 3).

5.3 Qualitative Estimate of Exposure

5.3.1 Number of servings and serving sizes

UCFM is a minor component of the New Zealand diet. Salami (uncooked) makes up 1.6% of food consumption, on the basis of servings and only 0.5% of food consumption on a weight basis. It should be reiterated that these figures represent upper bound estimates of UCFM consumption, as not all salami will be UCFM.

Median serving size for uncooked salami is 15.5 g, while the 75, 95 and 99th percentile serving sizes are 30, 114 and 275 g respectively.

5.3.2 Frequency of contamination

Given that there is very little data available for New Zealand, overseas data are all that can be used to evaluate this (see section 5.4.1). The percentage of *E. coli* O157:H7 contamination overseas ranges from 0% to 4.8%. No data could be found for non-O157 serotypes in UCFM products. Those data would suggest that contamination of UCFM products by *E. coli* O157 is rare and by non-O157 serotypes is unknown.

5.3.3 Predicted contamination level at retail

Given that the prevalence of *E. coli* O157 is rare, then the level of contamination on these comminuted products that are contaminated is likely to be low. Contamination by non-O157 serotypes is unknown.

5.3.4 Growth rate during storage and most likely storage time

Even if the UCFM products were contaminated during manufacture or distribution, the pH and water activity of UCFM products should be such that growth should not occur. The main factor that is relevant is how long *E. coli* O157 would survive on the products.

5.3.5 Heat treatment

Not relevant to UCFM products considered in this Risk Profile. Some salami-like products may be heat treated, but they are not considered here, as they are essentially a cooked meat product. A proportion of UCFM products will be consumed after cooking (e.g. on pizza) but these have not been included in the exposure estimate.

5.3.6 Exposure summary

The exposure of the New Zealand population to *E. coli* O157 and other STEC serotypes from UCFM consumption is likely to be very low, largely because this type of food is infrequently consumed. However, with the rising popularity of deli-sandwich takeaways, consumption is likely to increase.

5.4 Overseas Context

Overseas studies have tended to concentrate on *E. coli* O157 in UCFM so there are few data on non-O157 prevalence. The study of the prevalence of STEC in imported and domestic lean beef trim in the USA (Bosilevac *et al.,* 2007) found that 30% of samples from Australia, 28% of samples from Uruguay, and 30% of samples from the USA were positive for an *stx* gene by PCR. The prevalence in New Zealand samples was 10%. The rate of STEC recovery from these positive samples was: USA 28/147, Australia 9/67, Uruguay 40/147, and New Zealand 4/23. However, the authors commented that these data should not be used to compare the microbiological quality of the meats, as such comparisons should be on meat tested before export.

5.4.1 STEC in UCFM products: O157

Information summarising data for the prevalence of *E. coli* O157 in UCFM products is given in Table 8.

Table 8: Prevalence of *E. coli* O157 in UCFM products from overseas surveys

Country	Year	Products tested	Number tested	% positive	Reference
Argentina	2000	Dry sausage (dry-cured salami)	30	3.3	Chinen *et al.*, 2001
Australia (western)	1996-1997	Surveillance data of UCFM	>400	0	Cited in FSANZ (2003)
Australia (ACT)	2001	UCFM, including sliced meats	41*	0	Cartwright and Rockliff, 2001
EEC	1996	Dry and semi-dry sausages	3/4491	0.1	Anonymous, 1996
- Belgium			1/21	4.8	
- Germany			1/1040	0.1	
- Spain			1/245	0.4	
England, Wales and Northern Ireland	1998	Dried and fermented meat and meat products	2,981	0	Little *et al.*, 1998
The Netherlands	1996	Cooked or fermented RTE meats	328	0.3	Heuvelink *et al.*, 1999
USA	1995 - 1999	Dry and semi dry fermented sausages	3,445	0	Levine *et al.*, 2001

*Other meat product samples were included in this study and it could not be ascertained from the paper how many samples of UCFM were tested, only that 41 samples were tested for *E. coli.* Unclear whether serotype O157:H7 was specifically tested for.

In general, reported contamination rates for *E. coli* O157 in UCFM are low. Due to the lack of data, no comment can be made on the contamination rates for non-O157 serotypes in UCFM.

6 RISK CHARACTERISATION

The public health significance of infection with STEC derives from the high proportion of cases which have serious consequences following gastrointestinal disease.

6.1 Adverse Health Effects in New Zealand

6.1.1 Incidence and outbreaks

The first New Zealand case of infection with STEC was detected in 1993, and the illness was made a notifiable disease in June 1996. The number of cases of infection with STEC in New Zealand increased steadily throughout the late 1990s. The rates are shown in Table 9. The year 2003 has the highest notification rate in a single year and is more than double the rate of 1998.

Table 9: Rates of infection with STEC in New Zealand 1998 – 2005

Year	Rate per 100,000 (number of notified cases)	Reference
1998	1.3 (48)	Baker *et al.*, 1999
1999	1.8 (64)	Kieft *et al.*, 2000
2000	1.9 (68)	Lopez *et al.*, 2001
2001	2.0 (76)	Sneyd *et al.*, 2002
2002	2.0 (73)	Sneyd and Baker, 2003
2003	2.8 (105)	ESR, 2004a
2004	2.4 (89)	ESR, 2005a
2005	2.5 (92)	ESR, 2006a

In 2005, 36 cases were male (rate 2.0/100,000) and 55 cases female (rate 2.9/100,000). Regional variations were considerable: the highest rates were recorded in Otago (12 cases: 7.0), Bay of Plenty (10 cases: 5.6), Southland (5 cases; 4.8) and the Waikato (15 cases: 4.7 per 100,000) District Health Boards. Notification rates were highest in European (73 cases) followed by Maori (13 cases) ethnic groups (2.8 and 2.5 per 100,000 respectively).

Infection with STEC can affect any age group but most often causes disease in children aged 4 years or less. In 2005, in the <1 age group, there were 10 cases (18.3 per 100,000), in the 1 to 4 age group, 36 cases ; a rate of 16.7 per 100,000. In the elderly populations 60-69, there were 2 cases and in the 70+ age group, 7 cases; 2.2 per 100,000 (ESR, 2006a).

Based on studies in Canada, in New Zealand it has been assumed that 10-12 cases of STEC infection occur for each reported case (Baker *et al.*, 1999). This would equate to 920 to 1104 cases in 2005 in New Zealand.

During 2005 – 2007, ESR has been undertaking a NZFSA funded project to examine acute gastrointestinal illness in New Zealand at the community, general practitioner (GP) and, laboratory levels. These studies will provide information about the overall prevalence of the illness, as well as the factors influencing notification via laboratories and GPs. Although the project will not determine the prevalence of illnesses caused by individual pathogens,

information regarding laboratory testing methods and criteria for stool requesting by GPs (e.g. blood in stool) will be relevant to infection by STEC.

6.1.2 Clinical consequences of STEC infection

The clinical consequences of STEC infection of cases in New Zealand are summarised in Table 10.

Table 10: Summary of clinical consequences of STEC infection in New Zealand

Period	Hospitalised*	HC*	HUS*	TTP*	Fatalities	Reference
Oct '93 - Dec '98	24/58 (41.4%)	21/59 (35.6%)	18/59 (30.5%)	1/59 (1.7%)	2/79 (2.5%)	Baker *et al.*, 1999
1999	20/61 (32.8%)	16/33 (48.5%)	1/27 (3.7)	0/26 (0.0%)	0	Kieft *et al.*, 2000
2000	12/67 (17.9%)	14/51 (27.5%)	4/44 (9.1%)	0/42 (0.0%)	0	Lopez *et al.*, 2001
2001	17/75 (22.7%)	26/52 (50.0%)	3/35 (8.6%)	0/30 (0.0%)	0	Sneyd *et al.*, 2002
2002	16/67 (23.9%)	28/50 (56.0%)	4/36 (11.1%)	1/34 (2.9%)	0	Sneyd and Baker, 2003
2003	24/99 (24.2%)	43/74 (58.1%)	10/59 (16.9%)	1/53 (1.9%)	0	ESR, 2004
2004	27/86 (31.4%)	43/58 (74.1)	5/38 (13.2%)	1/33 (3.0%)	0	ESR, 2005a
2005	25/89 (28.1%)	26/57 (45.6%)	7/50 (14.0)	1/44 (2.3%)	0	**

* Percentages are determined on the basis of cases for which information was available
** Trev Margolin, ESR, personal communication, September 2006.

6.1.3 Serotypes causing disease in New Zealand

The majority of cases of infection with STEC in New Zealand have been with *E. coli* O157. Of the 92 notified STEC cases in 2005, 85 (92.4%) were identified as *E. coli* O157:H7 and 7 as non-O157:H7 (ESR, 2006a). Other serotypes that have caused infections over recent years include;

O26:H-	O26:H11	O75:HNM.	O84:H-	O84:H2	O84:HNM
O91:H21	O107:H51	O113:H21	O117:H-	O117:HNM	O128:H-
O128:H2	O130:H11	O145:H-	O153(rel):HN	ONT:H6	ONT:H18
ONT:H-	ORough:H-	ONT:HNM	ORough:H11	ONT:H8	ONT:H11

ORough:HNM.

(Source: Carolyn Nicol, ESR, personal communication, August 2004);

There have been two deaths in New Zealand attributed to STEC, both in the period 1993 – 1998. One was attributed to serotype O157:H7 (a child) and the other to O113:H21 (elderly

person). The New Zealand isolates of STEC that have caused infection have all possessed the genetic virulence factors in addition to either or both *Stx* genes (Carolyn Nicol, ESR Enteric Reference Laboratory, personal communication, March 2005).

Stool specimens (n=484) from children suffering from diarrhoea submitted to laboratories at Dunedin Public Hospital were examined in a study in 1996 (Brooks *et al.,* 1997). Two serotypes O26:H11, which is capable of causing HUS outbreaks, and O128:H2, were toxigenic and typable (Brooks *et al.*, 2001).

The serotypes O91:H-, O128:H2 and O128:H- have recently been isolated from New Zealand retail meat samples (Brooks *et al.*, 2001). O128:H2 and O128:H- serotypes have been isolated from both meat and a person suffering from diarrhoea, although they were not notified cases of STEC infection.

6.1.4 Outbreaks

The reported number of outbreaks and cases for which STEC was a causative agent between 1998 and 2005 are presented in Table 11.

Table 11: Total number of reported outbreaks and cases for which STEC was identified as the causative agent in New Zealand 1998-2005

Year	No. of outbreaks	Percent	No. of cases	Percent	Reference
1998	8	8/313: 2.6%	20	20/2139: 0.9%	Naing *et al*., 1999
1999	1	1/361: 0.3%	3	3/2358: 0.1%	Galloway and O'Sullivan, 2000
2000	1	1/289: 0.3%	4	4/2296: 0.2%	Lopez *et al*., 2001
2001	4	4/389: 1.0%	10	10/2323: 0.4%	Sneyd *et al*., 2002
2002	1	1/333: 0.3%	3	3/2870: 0.1%	Sneyd and Baker, 2003
2003	2	2/340: 0.6%	4	4/2789:0.1%	ESR, 2004a
2004	3	3/327: 0.9%	6	6/4085: 0.1%	ESR, 2005b
2005	3	3/346:0.9%	8	8/2436: 0.3%	ESR, 2006b
Total	**23**	**Mean 0.9%**	**58**	**Mean 0.3%**	

Small numbers of outbreaks, involving relatively low numbers of cases, have been reported to the national surveillance system each year since 1998, with the highest number being in 1998 (8 outbreaks, 20 cases). These events are probably better described as household clusters.

A review of information available from the Episurv database found that there was no evidence that the STEC outbreaks listed above were associated with the consumption of UCFM. Most outbreaks were associated with farms, there have been no outbreaks associated with regulated food.

6.1.5 Case control studies and risk factors

There have been no New Zealand case control studies to identify risk factors for STEC infection. The overview of 79 New Zealand cases of STEC from 1993 – 1998 reported that

in 1998 there were four household clusters including 9 cases, of which four were classified as caused by secondary transmission. Over the six year period 1993 to 1998 six cases reported living on a farm or visiting a farm regularly. Consumption of unpasteurised milk was reported by eight cases (Baker *et al.*, 1999).

Analysis of risk factors reported from cases in annual surveillance reports indicates that for cases where information is available, contact with pet animals, contact with farm animals, contact with animal manure, consuming non-habitual water supply, recreational contact with water, contact with children in nappies, contact with other animals and contact with sewage were common (ESR, 2005a; Kieft *et al.*, 2000; Sneyd *et al*., 2002). However, these are common factors in New Zealanders' lives and the proportions may simply reflect that fact, and the number of cases is too low to draw meaningful conclusions.

There have been a few episodes where indistinguishable STEC isolates have been isolated from both a human case and a potential transmission route in New Zealand. Contaminated untreated drinking water (one spring and one roof supply) was linked to two episodes of infection, affecting a total of three people in 1999, and one case has been attributed to contact with a calf (Anonymous, 2000). For one case in New Zealand, an indistinguishable isolate was obtained from both the infected child and raw milk present in the home, milk that was originally taken from a dairy farm for the family's new kittens (Anonymous, 2002).

To date, there are no surveillance data to link STEC transmission with UCFM in New Zealand.

6.2 Adverse Health Effects Overseas

6.2.1 Incidence

Incidence data for a selection of countries/states are given in Table 12. Data from New Zealand for 2004 and 2005 included in Table 12 indicate that the reported rate here is similar to other countries. The incidence of infection is considerably higher in the Czech Republic and considerably lower in Australia. The Scottish rate has significantly declined from 8.23 to 2.9 per 100,000 from 1997 to 2003 (PHLS, 2000).

Table 12: Rates of reported infections with STEC by country

Country	Year	Incidence (per 100,000)	No. of lab. confirmed cases	% O157:H7	% Other VTEC	Reference
New Zealand	**2004**	**2.4**	**82**	**91.5**	**8.5**	**ESR, 2005a**
New Zealand	**2005**	**2.5**	**92**	**92.4**	**7.6**	**ESR, 2006a**
Australia[#]	2002	0.3	53	-	-	Yohannes *et al.*, 2004;
	2003	0.2 (SA 2.4[*] Qld 0.2 Vic 0.1 WA 0.2)	49 (-37 -6 -3 -3)	25	15 O111	Miller *et al.*, 2005
Europe Community (17 member states + Norway	2004	1.3	4143	50	25[1]	EFSA, 2005
Austria	2004	0.6	45	29	71	EFSA, 2005
Belgium	2004	0.3	36	56	44	EFSA, 2005
Czech Republic	2004	17.1	1743	18	0	EFSA, 2005
Denmark	2004	3.0	163	27	73	EFSA, 2005
Finland	2004	0.2	10	40	60	EFSA, 2005
Germany	2004	1.1	903	10	42[1]	EFSA, 2005
Ireland	2004	1.4	57	88	12	EFSA, 2005
Netherlands	2004	0.2	30	100	0	EFSA, 2005
Norway[2]	2004	0.3	12	58	42	EFSA, 2005
Poland	2004	0.2	81	99	1	EFSA, 2005
Sweden	2004	1.7	149	-	-	EFSA, 2005
United Kingdom	2004	1.5	898	99	1	EFSA, 2005
(Scotland[3])	2003	2.9				SCIEH, 2004

Country	Year	Incidence (per 100,000)	No. of lab. confirmed cases	% O157:H7	% Other VTEC	Reference
North America						
Canada	1999	4.9				Health Canada (2000b)
Canada	2000	8.8				Health Canada (2000b)
USA (10 States)	2004	0.9[3]				CDC, (2005)
	2005	1.06[3] 0.33[4]				CDC, (2006)
	2006	1.31[3] 0.46[4]				CDC, (2007)

[#] HUS reported in 15 cases, rate 0.1/100,000
[*] 76% of cases are notified in South Australia where bloody stools are routinely tested by PCR for genes coding for shiga toxin.
[1] no information on remaining serotypes
[2] Norwegian data percentages modified from 7 and 5 to 58% and 42% respectively.
[3] rates are for STEC O157
[4] rates are for STEC non-O157

The USA health objective for 2010 for infections with *E. coli* O157 is 1 or less per 100,000. Based on data from 10 US States (Connecticut, Georgia, Maryland, Minnesota, New Mexico, Oregon, Tennessee, California, Colorado and New York), in 2004, this objective was met with an overall total of 0.9. However, since then, the rate has risen to 1.06 in 2005 and 1.31 in 2006. There is no national health objective set for non-O157 infections, the rate per 100,000 in 2005 was 0.33 and in 2006, 0.46. The proportion of STEC infected cases hospitalised in the United States has been estimated as 29.5%, with 0.8% of cases resulting in death (Mead *et al.*, 1999). Although New Zealand's hospitalisation and fatality rates to the end of 1998 were higher than this, there have been no deaths due to STEC since 1999 (see Table 10). In England and Wales, 31% of cases were hospitalised and an overall mortality rate of 3.7% recorded between the years 1992 and 1996 (PHLS, 2000).

6.2.2 Contributions to outbreaks and incidents

The proportion of outbreaks caused by STEC overseas is summarised in Table 13, with New Zealand included for comparison. As in New Zealand, only a small proportion of reported outbreaks are attributed to STEC.

Table 13: Proportions of outbreaks and incidents overseas caused by STEC

Country	Year	Proportion of outbreaks (%)	Reference
New Zealand	2005	0.9	ESR, 2006b
Canada	1982	0.2	Todd, 1992
Canada	1983	0.2	Todd, 1992
Canada	1984	0.1	Todd, 1992
England and Wales	1992-1994	1	Djuretic *et al.*, 1996
England and Wales	1995	1	Evans *et al.*, 1998
England and Wales	1996	1.4	Evans *et al.*, 1998
Sweden	1992-1997	<1	Lindqvist *et al.*, 2000

The Food Safety and Inspection Service (FSIS) of the USDA risk assessment for *E. coli* O157:H7 in ground beef summarised information from 154 *E. coli* O157:H7 outbreaks during the period 1982-1997 (FSIS, 1998). Ground beef was identified as the likely vehicle for infection in 25% of outbreaks, while whole cuts were identified with only 2% of outbreaks and salami with less than one percent.

Meat and meat products are often associated with STEC outbreaks and incidents overseas, however, a much smaller number of incidents have been linked to consumption of UCFM products. These are summarised for *E. coli* O157 and other STEC in Table 14.

Table 14: Specific incidents of disease reported for STEC associated with UCFM products

Location	Serotype	Year	No. affected	No. deaths	Source	Reference
Australia (South)	O111:H-	1995	23 HUS (one death), 30 bloody diarrhoea, 3 adults with TTP, 105 other GI symptoms	1	Mettwurst	CDC, 1995b
Australia (Western)	O157:H7	2001	2 (1 hospitalised)	0	Cacciatore (pork)	FSANZ (2003)
Canada	O157:H7	1998	39	NS	Genoa Salami (naturally fermented)	Williams *et al.*, 2000
Canada	O157:H7	1999	6 HUS; 143 ill (42 hospitalised)	0	Hungarian style sausage	MacDonald *et al.*, 2004
Germany	O157:H- (sorbitol +ve)	1995	28 children with HUS, estimated 300-600 other persons	3	Mortadella and teewurst*	Ammon *et al.*, 1999
USA	O157:H7	1995	20 (including 4 hospitalised and 2 HUS)	0	Presliced deli dry-cured salami	CDC, 1995a

NS=Not Stated. * Mortadella is a cooked product, but teewurst is substantially fermented and dried. The latter product was considered to be the likely vehicle in this outbreak.

6.2.3 Overseas case control studies

A dry-cured salami was the source of infection for 20 laboratory confirmed cases of STEC causing diarrhoea reported in Washington in late 1994 (CDC, 1995a). These cases were subsequently linked with three more in California. A case-control study involving 16 cases and age-matched controls was undertaken. Eleven of the 16 cases (69%) and 1 of the controls (6%) reported eating the implicated brand of dry-cured salami within 7 days before onset of illness ($p>0.01$). No other food item was associated with the illness. All of the salami implicated was purchased from local grocery chain stores. Environmental investigations at three of the stores and food samples collected revealed that there were no handling errors in the stores. However isolates from the three implicated salami samples were found to be indistinguishable by PFGE and Restriction Fragment Length Polymorphism analysis from 15 of 19 clinical isolates. The 15 clinical isolates that matched were from 12 cases who ate the salami, 2 secondary cases and one person who had eaten sliced turkey from a same deli where the implicated salami had been sold, suggesting cross-contamination. The remaining four isolates were from cases who had not eaten the salami. In response, the manufacturing company voluntarily recalled 10,000 pounds of implicated product which had been distributed between California, Oregon and Washington. In the aftermath of the outbreak, 250 dry sausage makers and industry representatives met the USDA's Food Safety Inspection Service to discuss the implications. It was agreed that production methods would be assessed for the survival of *E. coli* O157:H7.

A subsequent study investigated several hypotheses for the occurrence of the bacteria in this product by conducting evaluations of the production facility (Tilden *et al.*, 1996). The hypotheses were:

- Organisms present on raw meat ingredients survived a substandard fermentation and drying process;
- Organisms survived a fermentation and drying process that met existing industry and regulatory standards; or,
- Contamination occurred after fermentation and drying, either as a result of the slicing process or as a result of subsequent handling.

The investigation found that the methods used to produce the salami were typical of those used in the plant throughout 1994, and were typical of industry-wide methods used to produce Italian style salami. Record keeping was good, as the plant participated in the USDA FSIS Total Quality Control Program, and these records indicated that the methods complied with existing regulations and recommended good manufacturing practices. The recovery of *E. coli* O157:H7 from intact packages indicated that contamination did not occur at retail level. Extensive environmental and product testing failed to find *E. coli* O157:H7 and it was concluded that the most likely hypothesis was that a limited amount of contaminated meat was introduced into the plant and that the pathogen could survive the accepted processing methods at that time. Calculations estimated that the infectious dose was less than 50 *E. coli* O157:H7 bacteria.

The 1998 Canadian outbreak was investigated by a case-control study that identified Genoa salami from amongst a range of foods (Williams *et al.*, 2000). Although *E. coli* O157:H7 was not detected in any of the original food samples taken from retailers or the homes of cases, it was detected in samples taken from a single plant. The phage typing and pulsed

field gel electrophoresis patterns of these isolates were indistinguishable from those for the isolates obtained from cases. An investigation of the plant by the Canadian Food Inspection Agency revealed significant problems with the manufacturing process: the use of natural fermentation (i.e. instead of a starter culture), poor record keeping, no microbiological tests of incoming raw ingredients or final products, no records of lot specific pH or degree-hours measurements, faulty pH measurement methods and no written procedures for how to manage products with abnormal results.

In this investigation it was considered possible that some cases, who ate deli-sliced products but who did not recall eating Genoa salami, were in fact infected by cross contamination via the slicer.

In British Columbia, an observed five-fold increase above background rates for *E. coli* O157 led to an investigation in November 1999 (MacDonald *et al.,* 2004). A matched case-control study was conducted initially with 12 cases, 7 of whom reported eating salami prior to exhibiting symptoms. No other common exposures were identified and the outbreak was traced to a Hungarian-style salami and a Cervelat salami both produced by the same company. Nineteen cases took part in the case-control study overall, the results are displayed in Table 15.

Table 15: Results from Case-Control study (n = 19) involving UCFM meat in an *E. coli* O157 outbreak in Canada

Risk factor	No. of cases who ate product (%)	No. of controls who ate product (%)	OR	95% CI	*P*
Hungarian salami	11 (58%)	0	10	1.42-434	0.006
Cervelat salami	6 (31.6%)	1 (5.3%)	6	0.73-276	0.06
Both salamis	17 (89.5%)	1 (5.3%)	13	1.95-552	0.001

Source: MacDonald *et al.,* 2004

Over a 12-week period, 143 people were identified with the same PFGE results. Six cases developed HUS and 42 were hospitalised. In the investigation that followed this outbreak it became clear that the interventions recommended by Health Canada to improve the safety of UCFM products were not being followed in some establishments, and regulatory action was instituted (Health Canada, 2000a). During the period 1997 – 1999, the Canadian Food Inspection Agency issued three Health Hazard Alerts for recalls of UCFM with possible STEC contamination.

The outbreak in South Australia in December 1994 – February 1995 was linked to uncooked semi-dry fermented sausage product, mettwurst, from a single manufacturer by a preliminary investigation of cases (CDC, 1995b). It is to date the largest reported community outbreak of HUS associated with *E. coli* O111 infection. During the 8 days preceding onset of illness, 16 of the 23 HUS patients (70%) had consumed the implicated mettwurst. For three cases, the product had recently been in the household although consumption could not be confirmed. Stool specimens from the 23 patients revealed 16 positive samples for *E. coli* O111:NM. Out of the 30 cases with bloody diarrhoea and 3 cases with TTP, 8 stool samples (8/33: 24%)

were Polymerase Chain Reaction (PCR) positive for SLT genes but only 1 sample was positive for *E. coli* O111:NM. Of those 105 reports of gastro-intestinal illness (no bloody diarrhoea), 32 (30%) had a history of consuming the implicated mettwurst.

Of ten sausage samples taken from the homes of nine patients (8 homes in total), eight (all from the same manufacturer) were positive for the Shiga toxin producing genes (detected using PCR) and *E. coli* O111:H- was isolated from four of these samples. During the inquest into the death of the 4 year old child with HUS it was reported that the manufacturing company had been implicated in prior foodborne outbreaks due to *Salmonella* spp. (Desmarchelier, 1997). The product was manufactured from a combination of frozen mutton, pork back fat and fresh beef and mutton, any of which may have been a source of contamination. A starter culture was not used in the fermentation, no monitoring of pH or water activity was evident, and no pasteurisation process was employed.

The findings from a 6-month pilot prospective case-control study in South Australia have been reported (Hundy and Cameron, 2004). The study looked at risk factors for sporadic human infection with STEC in South Australia. It was conducted between February and September 2002 and involved 11 cases and 22 age-matched controls. Exposure to salami/ mettwurst/cabanossi as a risk factor was included in the study. The only food item significantly associated with STEC infection was the consumption of 'berries' (Matched OR 11.00, 95% CI 1.26-96.12). The exposure data for salami were 3/11 cases and 1/22 controls giving a matched odds ratio of 6.00 and a 95% confidence limit between 0.62 and 57.68. Given the small numbers involved in the study, significant results were not anticipated by the authors, who advocate national participation to increase the power of the study.

The outbreak in Germany began in late 1995 and was identified from an increase in the number of children suffering from HUS that were reporting to pediatric haemodialysis centres (Ammon *et al.*, 1999). On the basis that 5-10% of people with EHEC infections develop HUS, it was estimated that 300-600 people may have been infected. Two case-control studies were conducted to determine the cause; the first gathered information on a variety of potential risk factors. Twenty five case families and 76 control families were recruited, and the results showed a dose-response relationship between sausage consumption and illness, particularly among case-children who were hospitalised during December 1995. The second study gathered additional informational on 28 different "sliced" sausages. Of the sliced sausage products eaten by cases, only mortadella and teewurst were more likely to have been eaten by cases than controls (mortadella Odds Ratio 10.65, P = 0.004; teewurst OR 6.2, P = 0.02). Mortadella was considered less likely to be the source of the infection as it is heated during production; however, post heating contamination could not be ruled out. No investigation of the manufacturer was reported.

6.2.4 Risk assessments and other activity overseas

6.2.4.1 Australia

In Attachment 3 of the Final Assessment Report for Proposal P251 concerning processing requirements for UCFM (FSANZ, 2003), a Risk Assessment of EHEC in UCFM is published. The EHEC infectious dose is described as low and ingestion of as little a 1 organism could result in severe adverse health outcomes in susceptible individuals (children under 6 years of age was considered the most susceptible). A very low level of EHEC (0.15

per 100 grams) is likely to be present in approximately 7.2% of UCFM produced in Australia (based on the mean of 2 $\log_{10}$ reduction and 28% weight loss during processing). Although the risk of EHEC infection through the consumption of UCFM products was considered low, the consequence of EHEC infection can be severe such that the risk of EHEC infection though UCFM consumption must be managed through appropriate measures.

Two risk profiles for STEC and UCFM have been published in Australia. The first (Sumner, 2002) concerns the hazard-product pairing of UCFM and pathogenic *E. coli* in South Australia. A reliable (100% or 3 log reduction) and unreliable (99% - 2 log reduction) process was risk-ranked and scored 0 and 54 respectively. The unreliable process equated to 46 predicted illnesses per annum in infants and the elderly.

In the second study, the authors collated information on a range of red meat products and microbiological hazard combinations. This included EHEC in salami in Australia, (Sumner *et al.,* 2005). Risks associated with the pairings were rated low, medium or high according to set criteria which included;

- Severity,
- Occurrence,
- Growth,
- Production/processing/handling,
- Consumer terminal step, and
- Epidemiology

Risk ratings were prepared for hazard-product pairings on a scale of 0-100 (0 =no risk, 100 = everybody eating a meal containing a lethal dose of the hazard every day). A "low" risk equated to <25, "medium" to 26-40 and "high" >40. Because the scale is logarithmic, an increment of 6 in the ranking relates to a 10-fold increase in risk. A scenario of EHEC in salami was worked through based on the inactivation of *E. coli* during fermentation and maturation of salami (Ross and Shadbolt, 2001) and a reduction by 2-log (99%) was assumed. Two concentrations of EHEC were modelled representing "possible" low contamination (0.1/g or 10 cells per serving) or a "Slug" of raw meat with a higher concentration (10/g (or 1000 cells per serving). With a 2-log reduction, EHEC is reduced to 0.0001/g (0.1 cell/serve) and 0.1/g (10 cells/serve) respectively. The effect on susceptible populations (very young and very old) was also estimated. An infectious dose of 1000 cells for healthy individuals was used. Details of the risk rating for EHEC in salami in Australia can be seen in Table 16.

Table 16: Risk Assessment of EHEC in salami in Australia

Factors	General population	Very susceptible population	Very susceptible population
Hazard severity	Moderate	Moderate	Moderate
Population susceptibility	General	Very susceptible	Very susceptible
Frequency of consumption	Weekly	Weekly	Weekly
Proportion consuming (%)	Some (25%)	Some (25%)	Some (25%)
Total population	19.7 million	19.7 million	19.7 million
Proportion (%) of raw product contaminated (concentration)	0.01% (0.1/g, 10 cells /serve)	0.01% (0.1/g, 10 cells /serve)	0.01% (10g/serve, 1000 cells /serve)
Effect of processing on hazard	99% reduction	99% reduction	99% reduction
Post-processing contamination rate (%)	Nil	Nil	Nil
Post-processing control	Not relevant	Not relevant	Not relevant
Increase required to cause infection/intoxication	100	100	None
Effects of preparation before eating on hazard	50% reduction	50% reduction	50% reduction
Predicted cases per annum	1	1	114
Risk Rating	33	33	44

The assessment predicted one illness per annum in the general and very susceptible populations providing that the raw material contamination was low (0.1/g or 10 cells per serving). However, where the raw material was contaminated to the extent of 1000 cells per serving and eaten by susceptible individuals, the prediction rises significantly to 114 annual illnesses.

6.2.4.2 Scotland

A joint Food Standards Agency Scotland and Scottish Executive Task Force on *E. coli* O157 initiative was set up at the end of 2000 to examine the causes of the relatively high rates of STEC being experienced. The group reported their findings and recommendations in June 2001 (Anonymous 2001). The Task Force concluded that more cases of *E. coli* O157 infection in Scotland were associated with environmental contamination, contact with animal faeces, and contamination of water supplies, than with food. The response (Scottish Executive and Food Standards Agency Scotland, 2002) produced an action plan covering research, diagnosis, treatment and care, animals, the environment, water supply, use of rural land, food, education and risk communication.

6.2.5 Secondary transmission

Secondary transmission of STEC infection is a significant cause of cases. In a large beefburger-associated outbreak in the USA, 11% of the identified cases were secondary. A study in Wales between 1994 and 1996 indicated that 11% of cases were secondary, while the household transmission rate was estimated at 7% (summarised in Parry and Palmer, 2000).

6.3 Qualitative Estimate of Risk

Overall, the risk to New Zealanders from STEC in UCFM can be considered low. UCFM is consumed by only a small proportion of the population on a daily basis (1.6%), and there are no data linking notified cases of STEC infection with UCFM consumption.

Nevertheless, the presence of *E. coli* O157 and other STEC, some of which have human pathogenic potential, has been demonstrated in New Zealand livestock and meats. This indicates the potential for STEC contamination of the ingredients of UCFM manufacture. Reduction of STEC numbers during properly conducted manufacture will provide a reduction in risk, but it is likely that contamination will be present as a "slug" i.e. high numbers of bacteria in a small amount of meat. In such circumstances, even correct manufacturing processes will not completely eliminate the risk (a reduction of approximately 2-4 $\log_{10}$ cfu/g seems likely). This is particularly important for young children, who are more at risk of infection.

Only small amounts of beef and sheep meat (approximately 5% of the total supply) are imported, principally from Australia. Approximately 30% of the pigmeat supply in New Zealand is imported from Australia and Canada, although currently Canadian pigmeat is imported uncooked, held in transitional facilities, cooked and then released onto the New Zealand market so would not be used in UCFM production.

In terms of imported UCFM products, data to the year ending September 2005 indicates that 227 tonnes of meat preparations were brought into New Zealand. However the quantity of UCFM included in this figure is unknown. Section 3.3.1 discusses imported food in more detail.

6.4 Risk Categorisation

The rationale for categorisation of food/hazard combinations is presented in Appendix 1.

The proportion of severe outcomes (hospitalisation, long term sequelae, and death) resulting from STEC infection in New Zealand is approximately 10% (Lake *et al.*, 2000) placing this infection in the highest severity category.

For the purposes of estimating the numbers of cases of foodborne disease in New Zealand (Lake *et al.*, 2000) it was assumed that 20% of STEC infections were due to foodborne transmission. However, there is currently no evidence linking red meat consumption in general or UCFM consumption in particular to cases of STEC infection in New Zealand.

Thus the rate of STEC infection due to transmission in UCFM is considered to be less than 1 per 100,000 of population. This places STEC in UCFM in the lowest incidence category.

6.5 Summary

Food/hazard combination	Severity	Incidence	Trade importance	Other considerations
STEC in uncooked comminuted fermented meats	1 (>5% serious outcomes)	4 (<1 per 100,000)	N/A	

7 RISK MANAGEMENT INFORMATION

7.1 Relevant Food Controls: New Zealand

Processing of UCFM is currently regulated under Animal Products Act 1999 or the Food Act 1981. A brief description of both follows. New Zealand does not currently have specific Regulations relating to UCFM manufacture although more specific regulatory requirements have been proposed and are progressing through the public consultation process. Further information about the new proposed Standard and the current public consultation document is contained in section 7.1.2.

A long term review of the domestic food regulatory regime in New Zealand is underway by the NZFSA. Termed the Domestic Food Review, or DFR, one of the proposals is the introduction of Food Control Plans (FCPs). It is envisaged that all 'persons' will have and implement a documented FCP. Alternative arrangements would account for those businesses already with HACCP based systems in place such as RMPs.

7.1.1 Meat processing

The United States (New Zealand's largest beef market) requires that HACCP plans are in place in processing plants, and countries in the European Union also require a partial application of HACCP principles.

Currently New Zealand meat processing plants are registered under the Meat Act 1981. The Meat Regulations 1969, Game Regulations 1975, and subsidiary Industry Standards and Technical Directives apply.

7.1.1.1 Animal Products Act

The Animal Products Act 1999 reforms the New Zealand law that regulates the production and processing of animal material and animal products to:

- manage associated risks; and
- facilitate overseas market access.

The Animal Products Act requires all animal products traded and used to be "fit for intended purpose". This means they must meet New Zealand animal product standards. The New Zealand animal product standards are contained in Part 1 of the Animal Product Regulations 2000.

All animal product primary processing businesses, except those exempt under the Act or under the Animal Products (Exemptions and Inclusions) Order 2000, must have a Risk Management Programme (RMP).

A RMP is a documented programme to identify and manage biological, chemical and physical hazards and is based on the principles of Hazard Analysis and Critical Control Point (HACCP). RMPs are designed by individual businesses for the animal materials used, the processes performed and the product range produced.

Types of businesses that would have a RMP include primary processors of animal material, secondary processors of animal products (intended for human consumption) and retail butchers who are dual operator butchers (DOBs).

7.1.1.2 Food Act 1981

Historically, food premises have been inspected against the Food Hygiene Regulations 1974, however since 1996, there has been an option to develop a Food Safety Programme (FSP) – based on HACCP principles - which exempts the food business from the 1974 Regulations. A FSP is registered under the Food Act 1981. The process is applicable to any size of type of food business in New Zealand.

7.1.1.3 Monitoring compliance with standards

All US listed beef and sheep slaughter premises and packinghouses in New Zealand participate in a mandatory microbiological monitoring programme. The results are collated by the National Microbiological Database (NMD), which is operated by the NZFSA. One reason for the scheme was to demonstrate the microbiological equivalence of New Zealand's food safety controls to those of other countries, in particular the "US Pathogen Reduction; Hazard Analysis and Critical Control Point (HACCP) Systems" rule. Procedures are defined for sampling and analysis for aerobic plate count, *E. coli* and *Salmonella.* In addition to carcasses and primal cuts, samples are collected from boxed manufacturing beef at the end of the production system, immediately prior to freezing. (Source:

http://www.nzfsa.govt.nz/animalproducts/publications/manualsguides/nmd/index.htm

The New Zealand meat hygiene programme along with a national testing regime for *E. coli* O157 (implemented in 1998 under the NMD programme) currently satisfies the US requirement that processors demonstrate that *E. coli* O157 is "reasonably unlikely to occur" on beef exported to the US. While this testing programme and provision of results to the national database is not mandatory, contributions are currently received from 100% of export processing plants (Roger Cook, NZFSA, personal communication, April 2006).

7.1.2 Raw comminuted meat/salami processing

Food Standards Australia New Zealand (FSANZ) has developed a Standard for the production and processing of meat (Standard 4.2.3) which was gazetted on the 24th November 2005. Producers of ready-to-eat meat have two years from the gazettal before they are required to comply. Additional requirements appear in Clause 5 of the Standard regarding UCFM. Until then Clause 9 of Standard 1.6.2 applies. However, the Standard only applies to Australia. At the time of development, New Zealand considered this issue to be outside the scope of the Joint Food Standards System , but is now developing its own requirements.

In February 2007, a proposal to develop a New Zealand Standard was put out for public consultation, see website; http://www.nzfsa.govt.nz/consultation/ucfm/ucfm-discussion-document-final.pdf. It was noted that although there were no notified cases of STEC associated with the consumption of UCFM in New Zealand, this was attributed to the current

low level of STEC in New Zealand's raw meat supply rather than good manufacturing practice. The proposed Standard was drafted from the essential relevant parts of Part 1.6.2 (Australia only) of the FSANZ Food Standards Code, the New Zealand Pork Industry Board, Pork Quality Improvement Process (PQIP 07) Code of Practice and other elements identified by the NZFSA as essential. The closing date for submissions was 30th March 2007. NZFSA recognised that many secondary processors may already follow the PQIP 07 Code of Practice (COP) or the Food Standard Code 1.6.2, and that those presenting the greatest concern for uncontrolled UCFM production would be registered under the Food Hygiene Regulations, and would include businesses such as small boutique-style delicatessen operations.

A draft version of the proposed Standard was also issued with the discussion document and can be found at the following website: http://www.nzfsa.govt.nz/consultation/ucfm/ucfm-standard-draft.pdf. It should be noted that the proposed Standard is a work in progress and is a draft version subject to amendment depending on the submissions received. It is written in a general format but if the proposed Standard is implemented, it will be issued under both the Food Act 1981 and the Animal Products Act 1999, with adaptations to suit the two different regimes.

The proposed Standard for processing UCFM is summarized below, for a full version, refer to the website given above;

- a requirement that UCFM is produced in accordance with an approved Food Safety Programme or registered Risk Management Programme which:
  - i. has been validated to ensure numbers of *E. coli* organisms in the final UCFM comply with the microbiological limits of Standard 1.6.1 (FSANZ Food Standards Code), and
  - ii. demonstrates that the production process can handle variations in *E. coli* numbers present in ingoing raw meat ingredients,

- The *E. coli* count of ingoing raw meat ingredients must be known (to the 98th percentile) and be equivalent to or below the process lethality for the validated process.
  - i. For meat produced in New Zealand, information may be obtained from the New Zealand NMD, (if imported, information from an equivalent overseas data source), or
  - ii.adequate data provided by the company supplying the raw meat ingredients, or
  - iii. raw meat not covered by the NMD, may have adequate data collected by the manufacturer to validate its microbiological status.

- The number of *E. coli* organisms must be measured and recorded for the product after it has finished maturation and is ready for sale (frequency to be determined by the manufacturer),

- Validation may be achieved by predictive modeling or challenge studies,

- Fermentation of UCFM must be initiated through the use of a starter culture,

- Meat and batter mix must, if stored, be kept at 5°C or colder prior to fermentation,

- During production, the following must be monitored and recorded at suitable frequencies;
  i. temperature and time of fermentation, maturation/drying and smoking (if applicable),
  ii. pH and water activity of UCFM.

- Back slopping is not allowed, although re-processing of fully compliant product would be permitted.

The Standard also includes a section of recommended good manufacturing practice that includes;

- Taken a pH reading of the fermenting UCFM every 12 hours over the first 48 hour period (to ensure pH reaches 5.3 or lower within this time frame,
- Weight loss or water activity is taken during the process to ensure required outcome is met,
- To achieve a shelf stability without refrigeration, a UCFM should have:
  i. a pH of less than 5, or
  ii. a water activity of 0.90 or lower, or
  iii. a combination of pH less than 5.0 and a water activity of less than 0.95.

7.1.2.1 History

In the late 1990s the New Zealand Pork Industry Board (NZPIB) led the development of an industry wide standard, the Pork Quality Improvement Process (PQIP), which applied the HACCP approach to the New Zealand pork production process (NZPIB, 1998). This programme included requirements for UCFM of a pH of less than 4.6 and water activity less than 0.9, which depending on specific formulations, gives a weight loss of 40%. It notes that these limits are generally accepted worldwide to inhibit the growth but not destroy *E. coli* O157. In addition to pH and water activity, other hurdle controls are listed such as raw material supply, temperature of the product etc. The programme also stated that "For fermented products, acidification must not allow cross-contamination between batches. Back slopping is forbidden." An appendix to the PQIP programme dealt with *E. coli* O157:H7 in fermented sausages (Buncic, 1995). It described the effects of the various antimicrobial factors in such products, such as;

- Low pH – acid tolerance of *E. coli* O157:H7 very high,
- Sodium chloride, and
- Water activity.

Note: lactic acid bacteria antagonism has no significant effect as the bacteriocins produced are not effective against Gram-negative organisms.

In conclusion, Buncic stated that "In non-pasteurised, fermented sausages, *E. coli* O157:H7 is not expected to grow, but can survive the production process and subsequent storage.

Therefore, *E. coli* O157:H7-free uncooked sausages can be obtained only if raw material is assured to be *E. coli* O157:H7-free."

In 1999 a survey of the manufacturing practices of some of the larger salami processors in New Zealand was conducted by ESR on behalf of the Ministry of Health with the support of the Pork Industry Board (Hasell, 2000). Five processors, representing a large proportion of UCFM production in New Zealand were interviewed or visited and several areas of concern identified. None of the companies were testing raw meat, or batter, for *E. coli* as identified in the FSANZ Code. The time between preparing meat for a batch and starting the fermentation process was controlled by the processing environment rather than from a CCP perspective. The monitoring of pH in the early stages of fermentation, a critical monitoring point, was not being universally carried out. Not all companies had permanent printed records of process readings for specific batches and testing for final water activity was not common. None had verified their process in terms of *E. coli* reduction. Due to company confidentiality, the survey did not investigate whether the correct fermentation temperature was being used to suit the meat starter culture, nor whether the recipe formulation was appropriate for the culture. Overall, in 1999, the move towards HACCP-based food safety programmes amongst UCFM manufacturers in New Zealand was described as "only just beginning".

The report also considered the requirements and advisory guidelines developed by FSANZ for Australia as to their appropriateness for New Zealand. At that time FSANZ (then ANZFA) were proposing that UCFM production processes should achieve a 3D (1000 fold) reduction in *E. coli*. The comment was made that if the FSANZ code was followed correctly then the product must pose minimal risk. However, it was crucial to know which CCPs were required to obtain the 3D reduction, and the wide variety of products means that each manufacturing plant would need its own discrete HACCP programme to achieve the 3D reduction. An alternative to exhaustive testing of raw meat for the presence of *E. coli* was suggested: that companies producing salami source their raw meat from suppliers with HACCP-based food safety programmes in place. This was considered likely to result in a more enduring control on the quality of the raw material than testing alone, and mitigated against the adoption of the FSANZ code (Hasell, 2000).

The report concluded that the UCFM industry in New Zealand should be supported in its initiative to develop generic food safety programmes (in collaboration with MAF), and that once these were available the Ministry of Health should consider making such programmes compulsory. It was also recommended that the Ministry of Health provide industry with guidance as to the outcomes and performance outcomes that need to be achieved by a food safety programme. At present, a generic HACCP plan exists for the manufacture of beef jerky (MAF, 1999) but this generic plan is restricted to intact muscle cuts and does not cover UCFM.

7.1.2.2 Summary

The consultation document and proposed Standard as detailed in section 7.1.2 above are currently a work in progress. Of the three risk management options, development of a regulatory Standard was recognized as the most feasible, practicable and effective. The other two options were take no further action or to prohibit the production of UCFM. It is believed that many of the major UCFM manufacturers in New Zealand are already using either the Australian Standard or the PQIP COP. The implementation of this proposed Standard will

therefore most likely affect smaller producers who may not have a HACCP based system in place. Some of these businesses may face increased compliance costs and it is possible that some producers may choose to cease production rather than bear the cost.

7.1.3 Current status of HACCP based food safety programmes in the New Zealand UCFM industry

The PQIP programme contains a number of modules for different sectors of the pork industry. The module for Pork Processors (PQIP07) has been approved as a Code of Practice by the Ministry of Health (now New Zealand Food Safety Authority) for all processed pork products. PQIP was initially written without reference to UCFM. However, an additional chapter covering UCFM issues was added in Revision 1, on 1 July 2004. and is pending approval by the NZFSA as a COP, although the requirements for water activity and other controls have been approved already.

Issues under discussion in this chapter are:

- Appropriate indicator organisms for challenge testing,
- Separation of raw, UCFM, and cooked meats,
- How validation of processes will be performed,
- The value of computer modeling of processes to assess pathogen control, and,
- The standard which will have to be achieved by the process.

The HACCP plan described in the UCFM section has been implemented and validated by two major New Zealand UCFM manufacturers. A challenge protocol developed for Verkerks by Dr. Tecklok Wong (formerly with Envirolink, now with ESR) has been used to validate processes. The earlier generic *E. coli* protocol used by Verkerks was improved by the use of non-pathogenic acid tolerant *E. coli*. Challenge trials using this protocol have been completed at major manufacturer and demonstrate that water activity reduction was an important hurdle (Rob Scott, Goodman Fielder, personal communication, April 2006).

In terms of computer modeling, a predictive modeling tool "*E. coli* inactivation in fermented meats" has been developed for Meat and Livestock Australia by Food Science Australia (version 2.2, July 2004, model available from MLA, Australia). Hundreds of experiments on fermenting batters were carried out over a range of temperatures, salt concentrations and pH value combinations. Several types of UCFM were tested. The rate of *E. coli* death was determined and interpreted into a series of mathematical equations. To make the model user-friendly, calculators have been used which require time and temperature information on fermentation and maturation. The model estimates the average log-kill of *E. coli* with the specified parameters chosen by the enquirer. The output is displayed as the total predicted *E. coli* inactivation in log CFUs.

7.2 Relevant Food Controls: Overseas

The outbreaks that have occurred in the US, Canada, and Australia have prompted a variety of regulatory and industry responses for controls on UCFM production.

7.2.1 United States

In 1993 beef producers (National Cattlemen's Beef Association) in the United States established a Blue Ribbon Task Force to address the *E. coli* O157:H7 problem. One of their reports addressed UCFM products (Nickelson *et al.*, 1996). Using preliminary information from this report and other data, and coupled with an implicated outbreak to a dry fermented sausage product, in 1995 the USDA issued a Directive (Reed, 1995) to fermented meat processors. The initial intent was that all fermented sausage processes achieve a 5-log reduction or take additional equivalency steps such as a validated 2-log reduction using HACCP. The options for the control of *E. coli* O157:H7 were (Bacus, 1997; MacDonald *et al*., 2004; Getty *et al*., 2000);

- Achieve 5-log kill using a heat processing step (e.g. 63^{o}C for 4 minutes),
- Develop and validate individual 5-log inactivation treatment plans,
- Conduct a "hold and test" programme for finished product. Depending on type of product, 15 – 30 individual chubs must be sub-sampled per lot,
- Propose other process combinations to assure at least a 5-D inactivation, or
- Initiate a HACCP system that includes raw batter testing and a validated 2-D inactivation in fermentation and drying steps.

The FSIS guideline to conduct a validation study remained in all the options, a five-strain mixture (human and meat isolates including salami outbreak isolate (380-94)) of *E. coli* O157:H7 must be used to inoculate meat batter at 7.3 log cfu/g and after fermenting and drying, obtain a detection limit <1.0 log cfu/g. Assessing the range of acid tolerance responses and mechanisms among isolates was important which explains the use of the specific salami outbreak strain, growing cultures in glucose and harvesting in the stationary phase to allow optimum survival (the worst case scenario).

Various validation studies (reviewed by Getty *et al*., 2000) on *E. coli* O157:H7 found that the organism was able to survive fermentation and drying, and that additional thermal processing would be required to achieve the required 5-log reduction. Trials on thermotolerance have been conducted on pepperoni (Riordan *et al*., 2000), which have fed data into a thermal-death time curve and associated equation for non-acid adapted cells (post-fermentation to pH 4.8).

The "hold and test" requirement has been brought into question by an investigation into the outbreak in British Columbia (MacDonald *et al.*, 2004). Forty-three cases had left-over salami, yet *E. coli* O157 was isolated from 34 of these 43 samples (79%). Two earlier batches of salami which had passed the "hold and test" had been exported to the USA but on re-test, *E. coli* O157 was found, necessitating a full recall.

In February 2001 a proposed rule for the federal meat and poultry inspection regulations which covered ready-to-eat meats and partially heat-treated meat and poultry products was published in the federal register (see: http://www.fsis.usda.gov/oppde/rdad/frpubs/97-

013p.htm). Draft compliance guidelines were issued at the same time, and discussed the following options, in relation to fermented products:

See: http://www.fsis.usda.gov/oppde/rdad/frpubs/97-013p/rteguide.pdf

1. Heating processes to achieve a 6.5-$\log_{10}$ reduction of *Salmonella* and 5-$\log_{10}$ reduction of *E. coli* O157:H7 (formerly Option 1)

2. Processes to achieve a 5-$\log_{10}$ reduction or equivalent reduction of *E. coli* O157:H7 only (does not apply to *Salmonella*)

   a) Validated processes for a 5-$\log_{10}$ reduction of *E. coli* O157:H7 (formerly Option 2) – adapted from Table 6 of the Blue Ribbon Task Force report
   b) No *E. coli* O157:H7 detected in raw product and a validated 2D, 2-log reduction, process (formerly Option 5)

3. Fermentation processes that achieve at least a 5-$\log_{10}$ reduction of *E. coli* O157:H7 in specific products

The basis for the 5 $\log_{10}$ reduction of *E. coli* is derived from a hypothetical, worst case raw product of 10^4 CFU/g *E. coli* O157:H7 on red meat. The worst case raw product was deduced by the US National Advisory Committee for Microbiological Criteria for Foods (NACMCF) using data from the risk assessment of *E. coli* O157:H7 in ground beef performed by the USDA Food Safety and Inspection Service Office of Public Health and Science.

The option of a 2 $\log_{10}$ reduction is presumably predicated on sampling of raw ingredients to demonstrate the absence of *E. coli* O157:H7 and provides a safety margin over this.

7.2.2 Canada

In December 1996 and again in September 1998 the Canadian Food Inspection Agency notified UCFM manufacturers of the five interventions developed by the United States agencies and recommended that establishments adopt one of these. However, following the second outbreak of *E. coli* O157:H7 linked with UCFM, Health Canada decided to institute regulatory action. Interim guidelines using the five options were issued in February 2000, along with a challenge protocol to evaluate the process in terms of its ability to control *E. coli* O157:H7 (Health Canada, 2000a). Following appropriate consultation with industry and consumer groups the guidelines were intended to be developed into a regulation.

It is also worth noting that a survey in Toronto in 1994-1995 found that non-heat treated salami and sausage products made on-site at small deli-type establishments were more likely to be left constantly unrefrigerated, and have a higher pH and water activity than products made at larger commercial plants (Lee and Styliadis, 1996). This suggests that smaller processors of UCFM may need special attention for the introduction of controls.

7.2.3 Australia

In 1996, as an emergency response to the Australian outbreak from mettwurst (CDC, 1995b), FSANZ (then ANZFA) amended Standard C1 60A of the National Food Standards Code to incorporate additional requirements under Clause 9, Standard 1.6.2. for UCFM products. This Standard required that the production process reduce *E. coli* organisms by 99.9% (3-$\log_{10}$ reduction). In practice this was difficult for enforcement agencies because;

- Until recently there was a lack of objective means to determine industry compliance,
- The 3-log reduction did not necessarily achieve *E. coli* end-product specifications due to the variation in the initial microbial load, and
- The performance criterion was found to be unnecessarily prescriptive.

In addition to the emergency response, two complementary Advisory Guides were issued by ANZFA. The first, a Users' guide to Clause 60A of Standard C1 of the Food Standards Code (ANZFA, 1996a) and secondly "Advisory guidelines for making uncooked fermented comminuted meat products" (ANZFA, 1996b). The Australian Smallgoods Industry have also issued advisory guidelines to members which outlined model HACCP plans, offers assistance with plans, and reviews plans already in place (Meat and Allied Trades Federation of Australia, 1992).

The additional requirements in the ANZFA Advisory Guide were (Bacus, 1997):

- That the finished product must be free of *E. coli* in 0.1g of the food (specified method);
- That the number of *E. coli* in raw meat ingredients and finished product must be "monitored and recorded"; and,
- That processing must allow for a reduction in the numbers of *E. coli* that may have been present by "99.9% or 1000 fold", i.e. effectively a 3-$\log_{10}$ reduction, or
- Alternatively the product must be cooked at 65˚C for at least 10 minutes.

The use of starter cultures was also mandatory, no "back-slopping", and transport and storage of raw meat < 5°C. Additional requirements were stipulated for the control of coagulase positive staphylococci and *Salmonella*.

The basis for the choice of a 3D or (1000 fold) reduction appears to be the limit specified in the Advisory Guide that raw ingredients should be less than 100 CFU/g generic *E. coli.* Thus, a 1000-fold reduction, together with the relatively low prevalence and concentration of EHEC on meats used in UCFM manufacture, was deemed adequate to protect public health (Ross and Shadbolt, 2001).

In March 2002, FSANZ (then ANZFA) initiated a proposal to review the Food Standards Code with regard to UCFM production (Proposal P251 13 March 2002). This applied to Australia only, as New Zealand is not bound to apply the Food Standards Code food safety provisions under the Treaty. . The review took note of a report commissioned by Meat and Livestock Australia (Ross and Shadbolt, 2001) which concluded that:

- Many UCFM processes currently used, either in Australia or overseas, can not comply with the 3D kill requirement (although improvements were considered to have been

achieved in 2002, by September only 39% of manufacturers were considered to be achieving a 3-$\log_{10}$ reduction); and,
- Given the inability of most processes currently used in Australia to satisfy the requirement of Clause 60A, that the utility of this prescriptive regulation be reconsidered.

Following the draft assessment, and public consultation, a final assessment was published, (FSANZ, 2003). Processing requirements for UCFM are summarised below.

A verified food safety management system is required and a production process that can handle variations in *E. coli* contamination in the ingoing raw meat. The following steps must also be monitored and recorded, records must be kept for a minimum of 12 months after the use by or best before date.

- Number of *E. coli* in raw meat ingredients and in product after fermentation,
- pH of a fermenting UCFM,
- Temperature and time of fermentation of UCFM,
- Temperature and time of maturation/drying of UCFM,
- Temperature and time of smoking of UCFM, and
- Weight loss or water activity.
- Starter cultures must be used in order to initiate fermentation, 'back-slopping' is prohibited. In addition, the raw meat and batter mixes must be stored at 5°C or below prior to fermentation.

Microbiological limits in the final product are specified in Standard 1.6.1;

Food	**Micro-organism**	**n**	**c**	**m**	**M**
UCFM	Coagulase-positive staphylococci/g	5	1	10^3	10^4
	Escherichia coli/g	5	1	3.6	9.2
	Salmonella/25 g	5	0	0	-

There is provision for alternative technology or method specified elsewhere in the Food Standards Code, provided that the equivalent food safety outcome is achieved.

Two tools were developed by the National Expert Advisory Panel on UCFM Safety to assist with the implementation of the amended clause 9 of Standard 1.6.2 Firstly a "Protocol for Assessing HACCP-based Food Safety Programs in the UCFM Sector" and secondly a "Competency Criteria for UCFM Manufacturers – a guideline to facilitate the compliance with the skills and knowledge requirement of the food safety standards".

Following the review of processing requirements in Proposal P251 and amendments to the Food Standards Code, a review of the Food Safety Programs was initiated with a draft assessment report P289 published in May 2005, and the Final Assessment report published October 2005 (FSANZ 2005).

The final outcome of Proposal P289 has been an amendment to the Food Standards Code with Standard 4.2.3, Primary Production and Processing Standard for Meat and Standard 4.2.2, Primary Production and Processing Standard for Poultry Meat. Both Standards were gazetted on the 24th November 2005, however producers of ready-to-eat meat will have two years from the gazettal before they are required to comply.

The new Standard 4.2.3 incorporates many of the recommendations from the National Risk Validation Project – Final Report (Food Science Australia and Minter Ellison Consulting, 2002) and the conclusion of the cost/benefit analysis (Allen Consulting Group, 2002). Mandatory FSPs for producers of manufactured and fermented meats will be nationally required from 24 November 2007.

7.3 Risk Management Options

At the present time, the PQIP programme and the FSANZ Standard 1.6.2 are the risk management controls most commonly used by UCFM manufacturers in New Zealand. These Standards are not mandatory. There are proposals to introduce Regulations in New Zealand to formalise the situation.

The USA requirement of achieving a 5D reduction in STEC in UCFM presents significant practical difficulties. Incze (1998) concluded that the only practical way to achieve a 5D kill is to apply some form of heating process as commonly used methods only achieved a 1-2D kill. Altering other components, such as the salt concentration, was considered to result in unacceptable organoleptic qualities of the product (as does thermal processing).

Given the difficulty of achieving a high (5D) reduction in STEC numbers or even a 3D reduction during UCFM manufacture, the two risk management options appear to be:

1. Use of meat produced under a HACCP system.

To limit the amount of contamination of raw materials with the pathogen, this includes on-farm controls and hygienic conditions during processing in the abattoir and use of HACCP in the UCFM production facility. Good on-farm and processing controls could keep STEC numbers in meat ingredients sufficiently low so that a 5D (or even 3D) reduction would be unnecessary.

2. Heat treatment.

The heating of UCFM products does not necessarily have to be such that it cooks the product, and a number of low temperature (*circa* 50^{o}C) protocols have been described (Bacus 1997; Riordan *et al*., 2000). Typically holding times at these temperatures are of the order of 60 minutes.

As already mentioned the report prepared for Meat and Livestock Australia (Ross and Shadbolt, 2001) concluded that most UCFM processes in current use in Australia do not reliably achieve a 3D kill of *E. coli*. A range of experiments were proposed in the report to examine the extent of inactivation of *E. coli* caused by several parameters. The effects of temperature were regarded as well understood, but the effects of other factors (pH, organic acid levels, water activity, redox potential) were regarded as data gaps.

The report suggested that the maturation temperature could be raised by a few degrees to increase control of *E. coli*. This was considered unlikely to greatly affect the aesthetic qualities of the final product, but would accelerate inactivation rates. The faster drying from

ripening at higher temperatures, and the attendant risk of case hardening, could be offset by higher relative humidity in the ripening rooms.

The data summarised in Tables 4 and 6 in Section 3.2 suggest that pathogen reductions during fermentation and drying are relatively modest. However, a storage period at approximately 21˚C following fermentation and maturation could achieve D kills of 3 $\log_{10}$ or greater.

The risk management potential of advice to consumers is limited to refrigerated storage once packaging is opened, due to the ready-to-eat nature of UCFM at time of retail purchase.

7.4 Economic Costs

An analysis of the incidence and costs of foodborne disease in New Zealand estimated that STEC cost $507,000 in direct and indirect costs (Lake *et al.*, 2000; Scott *et al.*, 2000). This was based on an estimated total of 248 reported and unreported cases, of which 20% were assumed to be caused by foodborne transmission. This amount represented 0.9% of the total foodborne illness cost. This economic estimate covers all potential food vehicles. No data are available on the proportion of transmission by individual foods.

In the United States the estimated annual cost of O157 STEC infections was $405 million (based on 2003 dollar). This included $370 million for premature deaths, $30 million for medical care and $5 million in lost productivity. These figures were based on 73,000 infections annually, resulting in 2000 hospitalisations and 60 deaths. The average cost per case varying between $26 for no medical care required to $6.2 million for a case who died from HUS (Frenzen *et al*., 2005).

An earlier report considered all O157:H7 and non-O157 STEC infections. The estimated cost was US$1 billion (figures for 1998 updated for 2000). These costs were based on 94,000 annual cases, with approximately 2,800 hospitalisations and 78 deaths. This is from a total foodborne illness cost of US$6.9 billion which also includes diseases caused by *Campylobacter* species, non-typhoidal *Salmonella* and *Listeria monocytogenes* (Crutchfield and Roberts, 2000).

These figures are high in comparison with New Zealand figures as they include productivity losses due to chronic illness caused by STEC infection, which were not included in the New Zealand estimate. The estimate also assumed that 80% of cases were caused by foodborne transmission which is unlikely to be appropriate for New Zealand (Buzby *et al.*, 1996). The percentage of cases caused by foodborne transmission in the United States has more recently been estimated as 85% (Mead *et al.*, 1999). In England and Wales, 31 of 55 (56%) general outbreaks of O157:H7 reported to the PHLS between 1992 and 1997 were found to have a foodborne transmission route (Hansard, 1998).

8 CONCLUSIONS

8.1 Description of Risks to New Zealand Consumers

8.1.1 Risks associated with UCFM

The current rate of STEC infection in New Zealand is similar to overseas countries at approximately 2 notified cases per 100,000 population. All cases have been sporadic or in clusters involving small numbers of cases; widespread outbreaks affecting large numbers of people have not occurred in New Zealand. Information on transmission routes is meager, but as was also concluded in the Risk Profile on STEC in red meat, there is little evidence to suggest that UCFM currently represents a vehicle for transmission of pathogenic STEC in New Zealand.

Nevertheless *E. coli* O157 has been found in the faeces of cows in New Zealand, as well as a small number of raw meat samples from export processing plants and retail outlets. Non-O157 STEC serotypes have been detected in faecal samples from sheep and cows, as well as samples of meat from retail sources. Some of these serotypes have been associated with human illness. Consequently there is potential for the presence of pathogenic STEC in raw meat in New Zealand, which then may be used as an ingredient in the production of UCFM. There is also the possibility that imported raw meat such as pork from Australia which has the potential to be contaminated with STEC ends up in UCFM production in New Zealand.

The amount of UCFM imported into New Zealand each year is unclear, but the majority will originate from Australia, where production will be subject to the standard created by FSANZ. Imported UCFM represents an unquantified risk in New Zealand.

The consumption of UCFM is low in comparison with other red meat types, both in terms of servings (1.6% of all red meat servings) and weight (0.5% of all red meat consumed). The relatively low frequency of consumption of UCFM and the smaller serving sizes than for most other meat and meat products may mitigate the relative risk from this meat type although consumption may be increasing due to the popularity of deli-sandwich outlets.

STEC infections have a relatively high proportion of serious outcomes compared to other bacterial infections, and certain population groups in New Zealand (children up to 4 years) have higher rates of infection.

As was concluded for Australia (FSANZ, 2003), the risk is low but the consequences, particularly for susceptible groups, such as young children, are severe. Given the potential for exposure, on the basis of the observed prevalence of STEC in red meat in New Zealand, the risk needs to be managed by an appropriate measure.

8.1.2 Risks associated with other foods

In the United States ground beef/hamburger is the food vehicle most frequently implicated in outbreaks of *E. coli* O157:H7 infection, while the limited information from Europe suggests that meat consumption is not associated with sporadic cases. Other food vehicles implicated in outbreaks have been contaminated foods not cooked prior to consumption (lettuce, spinach, salads, coleslaw) or unpasteurised foods (milk, apple juice). Contact with animals,

and consumption of contaminated drinking water or other water sources have also been identified as transmission pathways.

In New Zealand, there is no evidence that regulated foods are a transmission route for *E. coli* O157.

8.1.3 Quantitative risk assessment

The main barrier to a comprehensive risk assessment is the limited data on the prevalence of contamination by STEC of New Zealand meats at the retail level (or at other points in the production chain), and the absence of quantitative levels of contamination. Further, there is no information from robust human surveillance studies to link meat or meat products with cases so far detected in New Zealand, and therefore no means to validate a QRA model. Current methodology will need to be improved to provide the same sensitivity for broad screen STEC detection techniques as are available for specific *E. coli* O157 methods.

The relative importance of foodborne transmission of STEC in New Zealand is unclear from the information gathered on cases to date. Consequently a quantitative risk assessment for STEC in red meat and meat products is unlikely to be significant and would be premature, with currently available data.

8.2 Commentary on Risk Management Options

Given the serious consequences of STEC infection it is essential that efforts continue to prevent foodborne transmission in red meat products generally. The high proportion of meat production that is exported means that mandatory HACCP based programmes will exist in most New Zealand meat processing plants and this will act to protect the portion of the domestic meat supply derived from the same source. This approach matches efforts in the United States to control STEC in red meat.

The prevalence of *E. coli* O157, and indeed *E. coli*, in meat from plants that deliver only to the domestic market is unknown and represents a significant data gap. It is estimated that meat from these plants make up approximately 40% of the total supply in New Zealand. Information of this type would be of value as a baseline, as these suppliers develop Risk Management Programmes as required under the Animal Products Act.

Options for control of STEC specifically in UCFM include the following:

1. A large reduction in bacterial levels during manufacture (5D) to cover all scenarios of bacterial contamination (US requirement);
2. Lower reductions in bacterial levels (2D-3D) providing a safety margin to back up low levels of generic *E. coli* contamination (or the absence of STEC), based on sampling and testing of raw ingredients (earlier FSANZ proposal);
3. As above with raw ingredients obtained from suppliers with a HACCP programme allied with microbiological testing programmes; and,
4. HACCP based food safety programmes that are validated to achieve a defined low level of *E. coli* in the finished product, as well as defined limits for *E. coli* in ingoing ingredients (later FSANZ proposal option (a));

5. An alternative measure that achieves an equivalent food safety outcome (later FSANZ proposal option (b)); and,
6. Microbiological testing of all end product before release (later FSANZ proposal option (c)).

Option 1 appears to be very difficult to achieve without some form of heat treatment, which may adversely affect the characteristics of the product.

Due to the wide variety of UCFM products and processes used to make them, generic Critical Control Points are unlikely to be achievable. Therefore Options 2 - 5 would require process verification in the form of challenge testing using an agreed protocol and indicator bacterium, as well as accepted and validated sampling and testing procedures.

While a 1-2D reduction is all that might be expected during fermentation and drying/maturation, there is scope for further reductions during storage. A greater reduction (perhaps up to 3D) should be able to be achieved by introducing only minor process changes.

Data from the New Zealand National Microbiological Database Programme for 1997 to 2000 indicates that 8.0% of boneless beef samples intended for export (i.e. from plants with a HACCP based Risk Management Programme) were positive for generic *E. coli*. The mean $\log_{10}$ CFU/g was 1.11, with the 99th percentile being $\log_{10}$ 3.0, and the maximum was $\log_{10}$ 4.31 (Roger Cook, New Zealand Food Safety Authority, personal communication). The very low levels of *E. coli* O157 in such meat products have been reported in Section 5.1.2.

At least two overseas outbreaks of STEC infection involving UCFM involved processes that did not use starter cultures. Their use would seem to reduce the risks associated with natural fermentation and "back-slopping" in UCFM production.

Any risk management options chosen are likely to be effectively implemented via the New Zealand Pork Processors Association and the PQIP programme, which will cover the majority of UCFM production in New Zealand. There may be a few "traditional" or "boutique" manufacturers outside this coverage, and a targeted intervention may be needed to achieve complete industry implementation of food safety measures.

There are proposals to introduce specific Regulations under the Food Act 1981 and the Animal Products Act 1999 regarding the manufacture of UCFM in New Zealand. The proposed New Zealand Standard which is based upon the FSANZ Food Standards Code, Standard 1.6.2 (Australia only) and PQIP COP No. 7 has been issued for public consultation along with a consultation document dated February 2007. The closing date for submissions was 30 March 2007. Submissions are currently being considered.

8.3 Data gaps

The data gaps identified in this report are:

- Current information on the CCPs being employed by UCFM producers in New Zealand,
- Current prevalence of STEC (not just *E. coli* O157) in New Zealand UCFM at the retail level or at other points in the production chain, as well as the ingredients – domestic and imported pork, sheep and deer meat,

- Quantitative levels of contamination by STEC, when contamination does occur, of New Zealand UCFM at the retail level (or at other points in the production chain),
- Methods to detect non-O157 STEC with the same sensitivity as *E. coli* O157,
- The prevalence of *E. coli* O157 and *E. coli* in meat from processing plants which deliver only to the domestic market and which can be used in UCFM production,
- Relative importance of transmission pathways for STEC in New Zealand, and
- Information on the market size and market structure for UCFM, including quantification of the Australian product and consumption patterns in at risk groups.

9 REFERENCES

Adesiyun AA, Webb LA, Romain H, Kaminjolo JS. (1997) Prevalence and characteristics of strains of *Escherichia coli* isolated from milk and feces of dairy farms in Trinidad. Journal of Food Protection; 60, 1174-1181.

Allen Consulting Group (2002) Food Safety Management Systems – Costs, Benefits and Alternatives. Department of Health and Ageing, Canberra.

Ammon A, Petersen LR, Karch H. (1999) A large outbreak of hemolytic uremic syndrome caused by unusual sorbitol-fermenting strain of *Escherichia coli* O157. Journal of Infectious Diseases; 179: 1274-1277.

Anonymous (1996) EU report – Co-ordinated programme for the official control of foodstuffs for 1996. http://europa.eu.int/comm/food/fs/sfp/oc01-pc96.pdf

Anonymous. (2000) Verocytotoxin-producing *Escherichia coli* (VTEC/STEC). Lablink; 7(1): 8.

Anonymous (2001) Task Force on *E. coli* O157. Final Report. Commissioned by Food Standards Agency (Scotland) and Scottish Executive. June 2001. http://www.food.gov.uk/multimedia/pdfs/ecolitaskfinreport.pdf

Anonymous. (2002) Verocytotoxin-producing *Escherichia coli* (VTEC/STEC). Lablink; 9(1): 8.

ANZFA (1996a) Standard C1 Meat, game and related products. Users' Guide. August 1996. Australia New Zealand Food Authority, Canberra.

ANZFA (1996b) Advisory guidelines for making uncooked fermented comminuted meat products. October 1996. Australia New Zealand Food Authority, Canberra.

Avery SM, Moore A, Hutchison ML. (2004). Fate of *Escherichia coli* originating from livestock faeces deposited directly onto pasture. Letters in Applied Microbiology; 38**:** 355-359.

Bacus J. (1997) Processing procedures to control *Salmonella* and *E. coli* in fermented sausage products. Food Australia; 49: 543-545.

Baker M, Eyles R, Bennett J, Nicol C. (1999) Emergence of verotoxigenic *Escherichia coli* (VTEC) in New Zealand. New Zealand Public Health Report; 6: 9-12.

Benjamin MM, Datta AR. (1995) Acid tolerance of enterohemorrhagic *Escherichia coli*. Applied and Environmental Microbiology; 61: 1669-1672.

Bennett J, Bettelheim KA. (2002) Serotypes of non-O157 verocytotoxigenic *Escherichia coli* isolated from meat in New Zealand. Comparative Immunology, Microbiology and Infectious Disease; 25: 77-84.

Bettelheim KA. (2000) Role of non-O157 VTEC. Journal of Applied Microbiology Symposium Supplement; 88: 38S-50S.

Bitzan M, Moebius E, Ludwig K, Müller-Wiefel DE, Karch H. (1991) High incidence of serum antibodies to *Escherichia coli* O157 lipopolysaccharide in children with hemolytic-uremic syndrome. Journal of Pediatrics; 199: 380-385.

Bosilevac JM, Guerini MN, Brichta-Harhay DM, Arthur TM, Koohmaraie M. (2007) Microbiological characterization of imported and domestic boneless beef trim used for ground beef. Journal of Food Protection; 70: 440-449.

Brooks HJL, Bettelheim KA, Todd B, Holdaway MD. (1997) Non-0157 Verocytotoxin producing *Escherichia coli*: aetiological agents of diarrhoea in children in Dunedin, New Zealand. Comparative Immunology, Microbiology and Infectious Diseases; 20(2):163-70.

Brooks HJL, Mollison BD, Bettelheim KA, Matejka K, Paterson KA, Ward VK. (2001) Occurrence and virulence factors of non-O157 shiga toxin-producing *Escherichia coli* in retail meat in Dunedin, New Zealand. Letters in Applied Microbiology; 32: 118-122.

Buncic S. (1995) Assessment of fate of *Escherichia coli* O157:H7 in fermented sausages produced by processes described by New Zealand Pork Industry Board. Report prepared by the Meat Industry Research Institute of New Zealand. Appendix D to Standard for Food Safety New Zealand Bacon Curing Industry Part 1. September 1998.

Buncic S, Avery SM. (1997) *Escherichia coli* O157:H7 in healthy dairy cows. New Zealand Veterinary Journal; 45: 45-48.

Buzby JC, Roberts T, Lin C-TJ, MacDonald JM. (1996) Bacterial foodborne disease: Medical costs and productivity losses. Agricultural Economic Report Number 741. Washington DC: United States Department of Agriculture Economic Research Service.

Cartwright M, Rockliff S. (2001) Microbiological quality of smallgoods. July 2001 – October 2001. ACT Health Services, Food Survey Reports 2000-2001.
http://www.health.act.gov.au/c/health?a=da&did=10052864&pid=1063343007

Calicioglu M, Faith NG, Buege DR, Luchansky JB. (1997) Viability of *Escherichia coli* O157:H7 in fermented semidry low-temperature-cooked beef summer sausage. Journal of Food Protection; 60: 1158-1162.

Calicioglu M, Faith NG, Buege DR, Luchansky JB. (2001) Validation of a manufacturing process for fermented semidry Turkish Soudjouk to control *Escherichia coli* O157:H7. Journal of Food Protection; 64: 1156-1161.

Calicioglu M, Faith NG, Buege Dr, Luchansky JB. (2002) Viability of *Escherichia coli* O157:H7 during manufacturing and storage of a fermented, semi-dry soudjouk-style sausage. Journal of Food Protection; 65: 1541 – 1544.

Caprioli A, Tozzi AE, Rizzoni G, Karch H. (1997) Non-O157 shiga toxin-producing *Escherichia coli* infections in Europe. Emerging Infectious Diseases; 3: 578-579.

CAST (Council for Agricultural Science and Technology). (1994) Foodborne pathogens: risks and consequences. Task Force Report No. 122.

CDC. (1995a) *Escherichia coli* O157:H7 outbreak linked to commercially distributed dry-cured salami- Washington and California, 1994. Morbidity and Mortality Weekly Reports; 44: 157-160.

CDC. (1995b) Community outbreak of hemolytic uremic syndrome attributable to *Escherichia coli* O111:NM-South Australia, 1995. Morbidity and Mortality Weekly Reports; 44: 550-551, 557-558.

CDC. (2005) Preliminary FoodNet data on the incidence of infection with pathogens transmitted commonly through food-10 sites, United States, 2004. Morbidity and Mortality Weekly Reports; 54:352-356.

CDC. (2006) Preliminary FoodNet data on the incidence of infection with pathogens transmitted commonly through food-10 States, United States, 2005. Morbidity and Mortality Weekly Reports; 55:392-395.

CDC. (2007) Preliminary FoodNet data on the incidence of infection with pathogens transmitted commonly through food-10 States, United States, 2006. Morbidity and Mortality Weekly Reports; 56:336-339.
http://www.cdc.gov/mmwr/preview/mmwrhtml/mm5614a4.htm

Chart H. (2000) VTEC enteropathogenicity. Journal of Applied Microbiology Symposium Supplement; 88: 12S-23S.

Chikthimmah N, Anantheswaran RC, Roberts RF, Mills EW, Knabel SJ. (2001) Influence of sodium chloride on growth of lactic acid bacteria and subsequent destruction of *Escherichia coli* O157:H7 during processing of Lebanon Bologna. Journal of Food Protection; 64: 1145-1150.

Chinen I, Tanaro JD, Miliwebsky E, Lound LH, Chimelli G, Ledri S, Baschkier A, Scarpin M, Manfredi E, Rivas M. (2001) Isolation and characterization of *Escherichia coli* O157:H7 from retail meats in Argentina. Journal of Food Protection; 64: 1346-1351.

Clavero MRS, Beuchat LR. (1996) Survival of *Escherichia coli* O157:H7 in broth and processed salami as influenced by pH, water activity, and temperature and suitability of media for its recovery. Applied and Environmental Microbiology; 62: 2735-2740.

Codex. (1999) Draft principles and guidelines for the conduct of microbiological risk assessment. Report of the thirty first session of the Codex Committee on Food Hygiene. ALINORM 99/13A. Rome: Codex Alimentarius Commission.

Cookson A, Taylor S, Attwood G. (2003) Detection of Shiga toxin-producing *Escherichia coli* from cattle and sheep in the lower North Island, New Zealand. Rumen Microbial Functional Genomics, Nutrition and Behaviour Group, AgResearch, Grasslands Research

Centre, Palmerston North, New Zealand. PP15.2 Proffered paper abstract MicroNZ, Annual Scientific Meeting and Exhibition.

Cookson A, Taylor S, Attwood G. (2006) The prevalence of Shiga toxin-producing *Escherichia coli* in cattle and sheep in the lower North Island, New Zealand. New Zealand Veterinary Journal; 54(1): 28-33.

Conner DE, Kotrola JS. (1995) Growth and survival of *Escherichia coli* O157:H7 under acidic conditions. Applied and Environmental Microbiology; 61: 382-385.

Cosansu S, Ayhan K. (2000) Survival of enterohaemorrhagic *Escherichia coli* O157:H7 strain in Turkish soudjouck during fermentation, drying and storage periods. Meat Science; 54 : 407 – 411.

Crutchfield SR, Roberts T. (2000) Food safety efforts accelerate in the 1990s. Food Review; 23 (3): 44-49.

CSIRO (1995) A bulletin for the Australian Food Industry. Special issue on Mettwurst and *Escherichia coli* O111. May 1995. Food Science Australia. http://www.foodscience.afisc.csiro.au/fshbull/mettwurt.htm

DeBauch ED, Savage GP. (1993) Salami: brewing a sausage. The Food Technologist; Autumn 1993: 31-39.

Decludt B, Bouvet P, Mariani-Kurkdjian P, Grimont F, Grimont PAD, Hubert B, Loirat C and the Société de Néphrologie Pédiatrique. (2000) Haemolytic uraemic syndrome and shiga toxin-producing *Escherichia coli* infection in children in France. Epidemiology and Infection; 124: 215-220.

Desmarchelier PM. (1997) Enterohaemorrhagic *Escherichia coli* – the Australian perspective. Journal of Food Protection; 60: 1447-1450.

Desmarchelier PM, Grau FH. (1997) *Escherichia coli*. In: Foodborne microorganisms of public health significance. Fifth Edition. Sydney: Australian Institute of Food Science and Technology Inc.

Djuretic T, Wall PG, Ryan MJ, Evans HS, Adak GK, Cowden JM. (1996) General outbreaks of infectious intestinal disease in England and Wales 1992 to 1994. CDR Review; 4: R57-R63.

Doyle MP, Zhao T, Meng J, Zhao S. (1997) *Escherichia coli* O157:H7. In: Doyle MP, Beuchat LR, Montville TJ. (eds). Food Microbiology Fundamentals and Frontiers. Washington, DC: American Society for Microbiology Press. pp 171-191.

Duncan SH, Booth IR, Flint HJ, Stewart CS. (2000) The potential for the control of *Escherichia coli* O157 in farm animals. Journal of Applied Microbiology Symposium Supplement; 88: 157S-165S.

Dundas S, Todd WTA. (2000) Clinical presentation, complications and treatment of infection with verocytotoxin-producing *Escherichia coli*. Challenges for the clinician. Journal of Applied Microbiology Symposium Supplement; 88: 24S-30S.

EFSA. (2005) The Community Summary Report on Trends and Sources of Zoonoses, Zoonotic Agents and Antimicrobial Resistance in the European Union in 2004. European Food Safety Authority, 21 December 2005. (The version published on 21 December will be replaced by edited version for printing February 2006). http://www.efsa.eu.int

Erkkilä S. (2001) Bioprotective and probiotic meat starter cultures for the fermentation of dry sausages. PhD Dissertation. Department of Food Technology. University of Helsinki, Finland. http://ethesis.helsinki.fi/julkaisut/maa/elint/vk/erkkila/bioprote.pdf

ESR. (2001) Enteric pathogens. Lablink. December 2001; 8: 37.

ESR. (2004a) Notifiable and other diseases in New Zealand. Annual Report 2003. ESR Client Report FW0426. Porirua: ESR.

ESR (2004b) Month Surveillance Report December 2004. Porirua: ESR. http://www.surv.esr.cri.nz/PDF_surveillance/MthSurvRpt/200412DecRpt.pdf

ESR (2005a) Notifiable and other diseases in New Zealand. Annual Report 2004. ESR Client Report FW 0532. Porirua: ESR.

ESR (2005b) Annual summary of outbreaks in New Zealand 2004. ESR Client Report FW 0543. Porirua: ESR.

ESR (2006a) Notifiable and other diseases in New Zealand. Annual Report 2005, ESR Client Report FW 0621, Porirua, April 2006.

ESR (2006b) Annual summary of outbreaks in New Zealand 2005. ESR Client Report FW 0623. Porirua: April 2006.

Evans HS, Madden P, Douglas C, Adak GK, O'Brien SJ, Djuretic T, Wall PG, Stanwell-Smith R. (1998) General outbreaks of infectious intestinal disease in England and Wales: 1995 to 1996. Communicable Disease and Public Health; 1: 165-171.

Ezawa A, Gocho F, Kawata K, Takahashi T, Kikuchi N. (2004). High prevalence of enterohemorrhagic *Escherichia coli* (EHEC) O157 from cattle in selected regions of Japan. Journal of Veterinary Medical Science; 66(5): 585-587.

Faith NG, Parniere N, Larson T, Lorang T, Luchansky JB. (1997) Viability of *Escherichia coli* O157:H7 in pepperoni during the manufacture of sticks and the subsequent storage of slices at 21, 4 and –20°C under air, vacuum and CO_2. International Journal of Food Microbiology; 37: 47-54.

Faith NG, Parniere N, Larson T, Lorang T, Kaspar CW, Luchansky JB. (1998a) Viability of *Escherichia coli* O157:H7 in salami following conditioning of batter, fermentation and drying of sticks, and storage of slices. Journal of Food Protection; 61: 377-382.

Faith NG, Wierzba RK, Ihnot AM, Roering AM, Lorang TD, Kaspar CW, Luchansky JB. (1998b) Survival of *Escherichia coli* O157:H7 in full- and reduced-fat pepperoni after manufacture of sticks, storage of slices at 4°C or 21°C under air and vacuum, and baking of slices of frozen pizza at 135, 191 and 246°C. Journal of Food Protection; 61: 383-389.

Food Science Australia and Minter Ellison Consulting (2002). National Risk Validation Project Final Report. Department of Health and Ageing. Australia.

Frenzen PD, Drake A, Angulo FJ, The Emerging Infections Program Foodnet Working Group (2005) Economic cost of illness due to *Escherichia coli* O157 infections in the United States. Journal of Food Protection; 68: 2623-2630.

FSANZ (2003) Review of processing requirements for uncooked comminuted fermented eat (UCFM) products. Final Assessment Report, Proposal P251. 10/03. 16 July 2003. The Terrace, Wellington.

FSANZ (2005) Food Safety Programs for the producers of manufactured & fermented meats. Final Assessment Report. Proposal P289. 7-05. 5 October 2005. The Terrace, Wellington. http://www.foodstandards.gov.au/standardsdevelopment/proposals/proposalp289foodsafe2450.cfm

FSIS (Food Safety and Inspection Service, US Department of Agriculture). (1998) Preliminary Pathways and Data for a Risk Assessment of *E. coli* O157:H7 in Beef. http://www.fsis.usda.gov/ophs/ecolrisk/prelim.htm

Fukushima H, Hoshina K, Gomyoda M. (1999). Long-term survival of shiga toxin-producing *Escherichia coli* O26, O111, and O157 in bovine feces. Applied Environmental Microbiology; **65**: 5177-81.

Getty KJK, Phebus RK, Marsden JL, Fung DYC, Kastner CL. (2000) *Escherichia coli* O157:H7 and fermented sausages: A review. Journal of Rapid Methods and Automation in Microbiology; 8 : 141-170.

Glass KA, Loeffelholz JM, Ford JP, Doyle MP. (1992) Fate of *Escherichia coli* O157:H7 as affected by pH or sodium chloride and in fermented, dry sausage. Applied and Environmental Microbiology; 58: 2513-2516.

Goldwater PN, Bettelheim KA. (1995) The role of enterohaemorrhagic *Escherichia coli* serotypes other than O157:H7 as causes of disease in Australia. Communicable Diseases Intelligence; 19: 2-4.

Haas CN, Rose JB, Gerba CP. (1999) Quantitative microbial risk assessment. New York: John Wiley.

Haas CN, Thayyar-Madabusi A, Rose JB, Gerba CP. (2000) Development of a dose-response relationship for *Escherichia coli* O157:H7. International Journal of Food Microbiology; 56: 153-159.

Hansard (1998) *E. coli* O157. In response to Countess of Mar Question. Parliamentary Secretary, MAFF (Lord Donoughue) reply. 19th November 1998. House of Lords. http://www.publications.parliament.uk/cgi-bin/ukparl_hl?DB=ukparl&STEMMER=en&WORDS=J0donoughue+&COLOUR=Red&STYLE=s&URL=/pa/ld199798/ldhansrd/vo981119/text/81119w01.htm#81119w01_wqn7

Hasell S. (2000) Hazard management guidelines for fermented meat products. ESR Client Report FW0092. Christchurch: ESR. Revised July 2000.

Health Canada. (2000a) Interim guidelines for the control of Verotoxinogenic *Escherichia coli* including *E. coli* O157:H7 in ready to eat fermented sausages containing beef or a beef product as an ingredient. Available from; http://www.hc-sc.gc.ca/fn-an/legislation/guide-ld/guideline_fermented_sausages-directives_saussisson_fermente_e.html

Health Canada. (2000b). Notifiable Diseases On-line. Laboratory Centre for Disease Control of Health Canada. http://cythera.ic.gc.ca/dsol/ndis/c_ind_e.html#top_list

Heuvelink AE, Zwartkruis-Nahuis JTM, Beumer RR, De Boer E. (1999) Occurrence and survival of verocytotoxigen-producing *Escherichia coli* O157:H7 in meats obtained from retail outlets in the Netherlands. Journal of Food Protection; 62: 1115-1122.

Hinkens JC, Faith NG, Lorang TD, Bailey P, Buege D, Kaspar CW, Luchansky JB. (1996) Validation of pepperoni processes for control of *Escherichia coli* O157:H7. Journal of Food Protection; 59: 1260-1266.

Hudson JA, Nicol C, Capill J, Bennett J. (2000) Isolation of shiga toxin-producing *Escherichia coli* (STEC) from foods using EHEC agar. Letters in Applied Microbiology; 30: 109-113.

Hudson JA. (2001) The significance of non-O157 STEC in foods. Draft prepared for the Ministry of Health. Client No. FW0132. April 2001, ESR, Christchurch.

Hundy RL, Cameron S. (2004) Risk factors for sporadic human infection with shiga toxin-producing *Escherichia coli* in South Australia. Communicable Diseases Intelligence; 28: 74-79

ICMSF. (1998) Micro-organisms in food. 6 Microbial ecology of food commodities. 1 Meat and meat products. VI Raw and comminuted meats. International Commission on Microbiological Specifications for Foods (ICMSF). London: Blackie Academic and Professional.pp39-42.

Incze K. (1998) Dry fermented sausages. Meat Science; 49: S169-S177.

Islam M, Doyle MP, Phatak SC, Millner P, Jiang XP. (2004). Persistence of enterohemorrhagic *Escherichia coli* O157: H7 in soil and on leaf lettuce and parsley grown in fields treated with contaminated manure composts or irrigation water. Journal of Food Protection; 67:1365-1370.

Jaeger JL, Acheson DW. (2000) Shiga toxin-producing *Escherichia coli*. Current Infectious Disease Reports; 2: 61-67.

Johnson SC, Sebranaek JG, Olson DG, Wiegland BR. (2000) Irradiation in contrast to thermal processing of pepperoni for control of pathogens: Effects on quality indicators. Journal of Food Science; 65: 1260-1265.

Kang DH, Fung DY. (1999) Effect of diacetyl on controlling *Escherichia coli* O157:H7 and *Salmonella Typhimurium* in the presence of starter culture in a laboratory medium and during meat fermentation. Journal of Food Protection; 62: 975 – 979.

Kang DH, Fung DY. (2000) Stimulation of starter culture for further reduction of foodborne pathogens during salami fermentation. Journal of Food Protection; 63: 1492 – 1495.

Karmali MA. (1989) Infection by verocytotoxin-producing *Escherichia coli*. Clinical Microbiological Reviews; 2: 15-38.

Karmali MA, Mascarenhas M, Shen S, Ziebell K, Johnson S, Reid-Smith R, Isaac-Renton J, Clark C, Rahn K, Kaper JB. (2003) Association of Genomic O Island 122 of *Escherichia coli* EDL 933 with Verocytotoxin-Producing *Escherichia coli* Seropathotypes that are linked to epidemic and/or serious disease. Journal of Clinical Microbiology; 41: 4930-4940.

Keskimaskii M, Siitonen A. (1997) From Finland: Surveillance of STEC infections. www.iss.it/scientifica/pubblica/notiziario/v10n10s1/v10n10s1.htm.

Kieft C, Perks M, Baker M, Galloway Y, Sutherland H. (2000) Annual surveillance summary 1999. Client Report FW0059. Kenepuru: ESR.

Kleanthous H, Smith HR, Scotland SM, Gross RJ, Rowe B, Taylor CM, Milford DV. (1990) Haemolytic uraemic syndrome in the British Isles, 1985-8: association with verocytotoxin producing *Escherichia coli*. Part 2: microbiological aspects. Archives of Diseases in Childhood; 65: 722-727.

Konowalchuk J, Speirs JI, Stavric S. (1977) Vero Response to a Cytotoxin of *Escherichia coli.* Infection and Immunity; 18: 775 – 779.

Lake RJ, Baker MG, Garrett N, Scott WG, Scott HM. (2000) Estimated number of cases of foodborne infectious disease in New Zealand. New Zealand Medical Journal; 113: 278-281.

Lake R, Hudson J, Cressey P. (2002) Risk Profile; Shiga toxin-producing *Escherichia coli* in red meat and meat products. FW 0154. August 2002. Christchurch. http://www.nzfsa.govt.nz/science/risk-profiles/stec-in-red-meat.pdf

Lee MB, Styliadis S. (1996) A survey of pH and water activity levels in processed salamis and sausages in metro Toronto. Journal of Food Protection; 59: 1007-1011.

Lejeune JT, Kauffman MD. (2005). Effect of sand and sawdust bedding materials on the fecal prevalence of *Escherichia coli* O157:H7 in dairy cows. Applied Environmental Microbiology; 71: 326-30.

LeJeune JT, Besser TE. Hancock DD. (2001). Cattle water troughs as reservoirs of *Escherichia coli* O157. Applied Environmental Microbiology; 67: 3053-7.

Leistner F, Gorris LGM. (1995) Food preservation by hurdle technology. Trends in Food Science and Technology; 6: 41-46.

Levine P, Rose B, Green, S, Ransom, G, Hill W. (2001) Pathogen testing of ready-to-eat meat and poultry products collected at federally inspected establishments in the United States, 1990 to 1999. Journal of Food Protection; 64: 1188-1193.

Lindqvist R, Andersson Y, de Jong B, Norberg P. (2000) A summary of reported foodborne disease incidents in Sweden, 1992 to 1997. Journal of Food Protection; 63: 1315-1320.

Little CL, Monsey HA, Nichols GL, DeLouvois J. (1998) The microbiology of ready-to-eat dried and fermented meat and meat products. International Journal of Environmental Health Research; 8: 277-284.

Lopez L, Baker M, Perks M. (2001) Annual surveillance summary 2000. Client Report FW0156. Kenepuru: ESR.

Luzzi I, Tozzi AE, Rizzoni G, Niccolini A, Benedetti I, Minelli, F, Caprioli A. (1995) Detection of serum antibodies to the lipopolysaccharide of *Escherichia coli* O103 in patients with hemolytic uremic syndrome. Journal of Infectious Diseases; 171: 524-515.

MacDonald DM, Fyfe M, Paccagnella A, Trinidad A, Louie K, Patrick D. (2004) *Escherichia coli* O157:H7 outbreak linked to salami, British Columbia, Canada, 1999. Epidemiology and Infection; 132: 283-289.

MAF. (1999) A Guide to HACCP systems in the meat industry. Appendix X.6. Generic HACCP plan for the manufacture of beef jerky. Volume 2. MAF Food Assurance Group (Animal Products Group). Amendment 6, June 1999. http://www.nzfsa.govt.nz/animalproducts/meat/meatman/haccp/meat/haccp_v2_appx-6.pdf

Makino S-I, Kii T, Asakura H, Shirahata T, Ikeda T, Takeshi K, Itoh, K. (2000) Does enterohaemorrhagic *Escherichia coli* O157:H7 enter the viable but nonculturable state in salted salmon roe? Applied and Environmental Microbiology; 66: 5536-5539.

Mariani-Kurkdjian P, Denamur E, Milon A, Picard B, Cave H, Lambert-Zechovsky N, Loirat C, Goullet P, Sansonetti PJ, Elion J. (1993) Identification of a clone of *Escherichia coli* O103:H2 as a potential agent of hemolytic-uremic syndrome in France. Journal of Clinical Microbiology; 31: 296-301.

Maule A. (2000). Survival of verocytotoxigenic *Escherichia coli* O157 in soil, water and on surfaces. Journal of Applied Microbiology; 88: 71S-78S.

McClure PJ, Hall S. (2000) Survival of *Escherichia coli* in foods. Journal of Applied Microbiology Symposium Supplement; 88: 61S-70S.

Mead PS, Slutsker L, Dietz V, McCaig LF, Breese JS, Shapiro C, Griffin PM, Tauxe RV. (1999) Food-related illness and death in the United States. Emerging Infectious Diseases; 5: 607-625.

Meat and Allied Trades Federation of Australia (1992). Australian Smallgoods Food Safety Guidelines; Model HACCP plan no. 7 – uncooked fermented comminuted products.

Mechie, SC; Chapman, PA; Siddons, CA. (1997). A fifteen-month study of *Escherichia coli* O157:H7 in a dairy herd. Epidemiology and Infection; 118:17-25.

Midgley J, Fegan N, Desmarchelier P. (1999) Dynamics of shiga toxin-producing *Escherichia coli* (STEC) in feedlot cattle. Letters in Applied Microbiology; 29, 85-89.

Miller M, Roche P, Yohannes K, Spencer J, Bartlett, Brotherton J, Hutchinson J, Kirk M, McDonald A, Vadjic C. (2005) Australia's notifiable diseases status, 2003. Annual Report of the National Notifiable Diseases Surveillance System. Communicable Disease Intelligence; March 2005, 29: 1-61
http://www.health.gov.au/internet/wcms/publishing.nsf/Content/cda-cdi2901atoc.htm

Ministry of Health/Ministry of Agriculture and Forestry. (2000) Food Administration in New Zealand: A Risk Management Framework for Food Safety. Wellington: Joint Ministry of Health and Ministry of Agriculture and Forestry Food Harmonisation Project.

Naim F, Messier S, Saucier L, Piette G. (2003) A model study of *Escherichia coli* O157:H7 survival in fermented dry sausages – influence of inoculum preparation, inoculation procedure, and selected process parameters. Journal of Food Protection; 66: 2267-2275.

Naing T, Baker M, Galloway Y, Margolin T. (1999) Annual summary of outbreaks in New Zealand in 1998. Porirua, ESR

Nickelson R II, Luchansky J, Kaspar C, Johnson E. (1996) Dry fermented sausage and *E. coli* O157:H7. Blue Ribbon Task Force, National Cattlemen's Beef Association, Chicago, Research Report No. 11-316.

NZPIB (1998) Standard for Food Safety New Zealand Bacon Curing Industry – Pork Quality Improvement Process. 02/Part I/A1. September 1998, New Zealand Pork Industry Board.

Pai CH, Ahmed N, Lior H, Johnson WM, Sims HV, Woods DE. (1988) Epidemiology of sporadic diarrhoea due to verocytotoxin-producing *Escherichia coli*: a two year prospective study. Journal of Infectious Diseases; 157: 1054-1057.

Park S, Worobo RW, Durst RA. (1999) *Escherichia coli* O157:H7 as an emerging foodborne pathogen: A literature review. Critical Reviews in Food Science and Nutrition; 39: 481-502.

Parry SM, Palmer SR. (2000) The public health significance of VTEC O157. Journal of Applied Microbiology Symposium Supplement; 88: 1S-9S.

Pearce MC, Fenlon D, Low JC, Smith AW, Knight HI, Evans J, Foster G, Synge BA, Gunn GJ. (2004) Distribution of *Escherichia coli* O157 in bovine fecal pats and its impact on

estimates of the prevalence of fecal shedding. Applied and Environmental Microbiology; October: 5737-5743.

Pennington TH. (2000) VTEC: lessons learned from British outbreaks. Journal of Applied Microbiology Symposium Supplement; 88: 90S-98S.

PHLS. (2000) Subcommittee of the Public Health Laboratory Service (PHLS) advisory committee on gastrointestinal infections. Guidelines for the control of infection with Vero cytotoxin producing *Escherichia coli* (VTEC). Communicable Disease and Public Health; 3: 14-23. http://www.hpa.org.uk/cdph/issues/CDPHvol3/No1/vtec.pdf

Pidcock K, Heard GM, Henriksson A. (2002) Application of nontraditional meat starter cultures in production of Hungarian salami. International Journal of Food Microbiology; 76:75-81.

Pierard D. (1992) Infections with verotoxin-producing *Escherichia coli*. Acta Clinica Belgica; 47: 387-396.

Pierard D, Crowcroft N, De Brock S, Potters D, Crabbe G, Van Loock F, Lauwers S. (1999) A case-control study of sporadic infection with O157 and non-O157 verocytotoxin-producing *Escherichia coli*. Epidemiology and Infection; 122: 359-365.

Pond TJ, Wood DS, Mumin IM, Barbut S, Griffiths MW. (2001) Modeling the survival of *Escherichia coli* O157:H7 in uncooked, semidry, fermented sausage. Journal of Food Protection; 64: 759-766.

Powell MR, Ebel E, Schlosser W, Walderhaug M, Kause J. (2001) Dose-response envelope for *Escherichia coli* O157:H7. Quantitative Microbiology; 2: 141-163.

Pommepuy M, Butin M, Derrien A, Gourmelon RR, Cormier M. (1996) Retention of enteropathogenicity by viable but nonculturable *Escherichia coli* exposed to seawater and sunlight. Applied and Environmental Microbiology; 62: 4621-4626.

Reed CA. (1995) Challenge study – *Escherichia coli* O157:H7 in fermented sausage. Letter to Plant managers; April 28, USDA, FSIS, Washington DC.

Ricke SC, Keeton JT. (1997) Fermented meat, poultry and fish products. In: Food Microbiology: Fundamentals and Frontiers (eds. MP Doyle, LR Beuchat, Montville TJ). ASM Press: Washington DC: 610-628.

Ricke SC, Díaz IZ, Keeton JT. (2001) Fermented meat, poultry and fish products. In: Food Microbiology: Fundamentals and Frontiers (eds. MP Doyle, LR Beuchat, Montville TJ). 2nd edition, ASM Press: Washington DC: 681-700.

Rigsbee W, Simpson LM, Oliver JD. (1997) Detection of the viable but nonculturable state in *Escherichia coli* O157:H7 . Journal of Food Safety; 16: 255-262.

Riordan DCR, Duffy G, Sheridan JJ, Eblen BS Whiting RC, Blair IS, McDowell DA. (1998) Survival of *Escherichia coli* O157:H7 during the manufacture of pepperoni. Journal of Food Protection; 61: 146-151.

Riordan DC, Duffy G, Sheridan JJ, Whiting RC, Blair IS, McDowell DA. (2000) Effects of acid adaptation, product pH and heating on survival of *Escherichia coli* O157:H7 in pepperoni. Applied Environmental Microbiology; 66: 1726-1729.

Rogerie F, Marecat A, Gambade S, Dupond F, Beaubois P, Lange M. (2001) Characterization of shiga toxin producing *E. coli* and O157 serotype *E. coli* isolated in France from healthy domestic cattle. International Journal of Food Microbiology; 63: 217-223.

Ross T, Shadbolt CT. (2001) Predicting *E. coli* inactivation in uncooked comminuted fermented meat products. Prepared for Meat and Livestock Australia by the Centre for Food Safety and Quality, School of Agricultural Science, University of Tasmania. North Sydney: Meat and Livestock Australia.

Rowe PC, Orbine E, Lior H, Wells GA, McLaine PN. (1993) A prospective study of exposure to verotoxin-producing *Escherichia coli* among Canadian children with hemolytic uremic syndrome. Epidemiology and Infection; 110: 1-7.

Russell DG, Parnell WR, Wilson NC *et al*. (1999) NZ Food: NZ People. Key results of the 1997 national Nutrition Survey. Wellington: Ministry of Health.

Sanchez S, Lee MD, Harmon BG, Maurer JJ, Doyle MP. (2002). Animal issues associated with *Escherichia coli* O157:H7. Journal of the American Veterinary Medical Association; 221: 1122-1126.

SCIEH. (2004) Scottish Centre for Infection and Environmental Health Weekly Report, 6th January 2004; NHS Scotland, Volume 38: 2004/01
http://www.show.scot.nhs.uk/scieh/PDF/pdf2004/0401.pdf

Scotland SM, Rowe B, Smith HR, Willshaw GA, Gross RJ. (1988) Verocytotoxin-producing strains of *Escherichia coli* from children with haemolytic uraemic syndrome and their detection by specific DNA probes. Journal of Medical Microbiology; 25: 237-243.

Scott WG, Scott HM, Lake RJ, Baker MG. (2000) Economic cost to New Zealand of foodborne infectious disease. New Zealand Medical Journal; 113: 281-284.

Scottish Executive and Food Standards Agency Scotland. (2002) Response to the Report of the Task Force on *E. coli* O157. Scottish Executive. http://www.scotland.gov.uk/Resource/Doc/46922/0014164.pdf

Semanchek JJ, Golden DA. (1998) Influence of growth temperature on inactivation and injury of *Escherichia coli* O157:H7 by heat, acid and freezing. Journal of Food Protection; 61: 395-401.

Sharma M, Richards GM, Beuchat LR. (2004) Survival and growth of *Escherichia coli* O157:H7 in roast beef and salami after exposure to an alkaline cleaner. Journal of Food Protection; 67 : 2107-2116.

Sneyd E, Lopez L, Eglinton M, McDowell R, Margolin T. (2002) Annual surveillance summary 2001. Client Report FW0156. Kenepuru: ESR.

Sneyd E, Baker M. (2003) Infectious Diseases in New Zealand: 2002 Annual Surveillance Summary. ESR Client Report FW0332. Porirua: ESR.

Sumner J, Ross T, Jenson I, Pointon A. (2005) A risk microbiological profile of the Australian red meat industry: Risk ratings of hazard-product pairings. International Journal of Food Microbiology; 105: 221-232.

Takumi K, de Jonge R, Havelaar A. (2000) Modelling inactivation of *Escherichia coli* by low stomach pH: application to passage through the stomach of young and elderly people. Journal of Applied Microbiology; 89: 935-943.

Tamura K, Sakazaki R, Murase M, Kosako Y. (1996) Serotyping and categorisation of *Escherichia coli* strains isolated between 1958 and 1992 from diarrhoeal diseases in Asia. Journal of Medical Microbiology; 45: 353-358.

Tilden J Jr, Young W, McNamara A-M, Custer C, Boesel B, Lambert-Fair MA, Majkowski J, Vugia D, Werner SB, Hollingsworth J, Morris JG Jr. (1996) A new route of transmission for *Escherichia coli*: infection from dry fermented salami. American Journal of Public Health; 86: 1142-1145.

Todd EC. (1992) Foodborne disease in Canada- a 10 year summary from 1975 to 1984. Journal of Food Protection; 55: 123-132.

Uyttendaele M, Taverniers I, Debevere J. (2001) Effect of stress induced by suboptimal growth factors on survival of *Escherichia coli* O157:H7. International Journal of Food Microbiology; 66: 31-37.

Varney, K. (2004). Quarterly review of diagnostic cases – April to June 2004. Surveillance. 31(3):21-25.

WHO. (1998) Zoonotic Non-O157 Shiga Toxin-Producing *Escherichia Coli* (STEC). Report of a WHO Scientific Working Group Meeting. Berlin, Germany, 23-26 June 1998. Available from: http://www.who.int/emc-documents/zoonoses/whocsraph988c.html

Williams RC, Isaacs S, Decou ML, Richardson EA, Buffett MC, Slinger RW, Brodsky MH, Ciebin BW, Hockin J. (2000) Illness associated with *Escherichia coli* O157:H7 in Genoa salami. Canadian Medical Association Journal; 162: 1409-1413.

Willshaw GA, Smith HR, Roberts D, Thirlwell T, Cheasty T, Rowe B. (1993) Examination of raw beef products for the presence of verocytotoxin producing *Escherichia coli*, particularly those of serogroup O157. Journal of Applied Microbiology; 75: 420-426.

Yohannes K, Roche P, Blumer C, Spencer J, Milton A, Bunn C, Gidding H, Kirk M, Della-Porta T. (2004) Australia's notifiable diseases status, 2002. Communicable Diseases Intelligence; 28: 6-68.

APPENDIX 1: CATEGORIES FOR RISK PROFILES

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

1. Incidence

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

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

Disease/organism	Food rate (/100,000 population) Calculated for 12 months to June 2001	Food rate (/100,000 population) Calculated for 12 months to December 1998
Campylobacteriosis	1320	2047
Listeriosis	0.4	0.4
VTEC/STEC	1.9	1.4
Salmonellosis	176	230
Yersiniosis	38	62
Shigellosis	7	7
NLV*	478	478
Toxins*	414	414
Typhoid*	0.3	0.3
Hepatitis A*	0.4	0.4

* not recalculated.

These are **total** foodborne rates, so it is probably safe to assume that in most cases the rates associated with a particular food are likely to be an order of magnitude lower. For instance, a category of ">1000" would only be assigned if it was decided that all campylobacteriosis was due to a single food/food type.

The following categories are proposed for the rates attributable to a single hazard/food (or food group) combination:

Category	Rate range	Comments/examples
1	>100	Significant contributor to foodborne campylobacteriosis Major contributor to foodborne NLV
2	10-100	Major contributor to foodborne salmonellosis Significant contributor to foodborne NLV
3	1-10	Major contributor to foodborne yersiniosis, shigellosis
4	<1	Major contributor to foodborne listeriosis

A further category, of "no evidence for foodborne disease in New Zealand" is desirable, but it was considered more appropriate to make this separate from the others. Also separate is another category, of "no information to determine level of foodborne disease in New Zealand".

The estimation of the proportion of the total foodborne disease rate contributed by a single food or food group will require information from a variety of sources including:

- exposure estimates
- results from epidemiological studies (case control risk factors)
- overseas estimates

For illnesses where the rate is <1 per 100,000 the ability to assign a proportion is unlikely to be sensible. For such illnesses it may be more useful to consider a Risk Profile across the range of all high risk foods, rather than individual foods or food groups.

2. Severity

Severity is related to the probability of severe outcomes from infection with the hazard.

The outcomes of infectious intestinal disease are defined in the estimate of the incidence (Lake *et al.*, 2000) as:

- death
- hospitalised and long term illness (GBS, reactive arthritis, HUS)
- hospitalised and recover
- visit a GP but not hospitalised
- do not visit a GP

The first three categories of cases were classed as severe outcomes. Some hospitalisations will result from dehydration etc. caused by gastrointestinal disease. However, for infections with *Listeria* and STEC hospitalisation will result from more severe illness, even if recovery is achieved.

The proportion of severe outcomes resulting from infection with the hazards can be estimated from the proportion of cases hospitalised and recover, hospitalised and long term illness, and deaths (Lake *et al*., 2000).

Disease/organism	Percentage of outcomes involving death or long term illness from foodborne cases
Campylobacteriosis	0.3
Listeriosis	60.0
VTEC/STEC	10.4
Salmonellosis	1.0
Yersiniosis	0.4
Shigellosis	2.7
NLV	Assumed to be <0.5%
Hepatitis A	15.4
Typhoid	83.3
Toxins	Assumed to be <0.5%

Categories for the probability of severe outcomes are suggested as follows:

Severity Category	Percentage of cases that experience severe outcomes	Examples
1	>5%	listeriosis, STEC, hepatitis A, typhoid
2	0.5 – 5%	salmonellosis, shigellosis
3	<0.5%	campylobacteriosis, yersiniosis, NLV, toxins

There are a number of hazards for which the incidence of foodborne disease is uncertain. These have been assigned to the above severity categories as follows:

Severity category 1:

Bacteria

Clostridium botulinum

Protozoa

Toxoplasma

Severity category 3:

Bacteria

Aeromonas/Plesiomonas
Arcobacter
E. coli (pathogenic, other than STEC)
Pseudomonas
Streptococcus
Vibrio parahaemolyticus

Viruses

Others (e.g. rotavirus)

Protozoa

Giardia
Cryptosporidium
Cyclospora
Others (e.g. *Entamoeba*)

Proposed Category Matrix

Incidence	>100	10-100	1-10	<1
Severity 1				
Severity 2				
Severity 3				

Alternatives:

No evidence for foodborne disease in New Zealand

No information to determine level of foodborne disease in New Zealand