RISK PROFILE: SHIGA TOXIN-PRODUCING *ESCHERICHIA COLI* IN RED MEAT AND MEAT PRODUCTS

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by

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RISK PROFILE: SHIGA TOXIN-PRODUCING *ESCHERICHIA COLI* IN RED MEAT AND MEAT PRODUCTS

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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

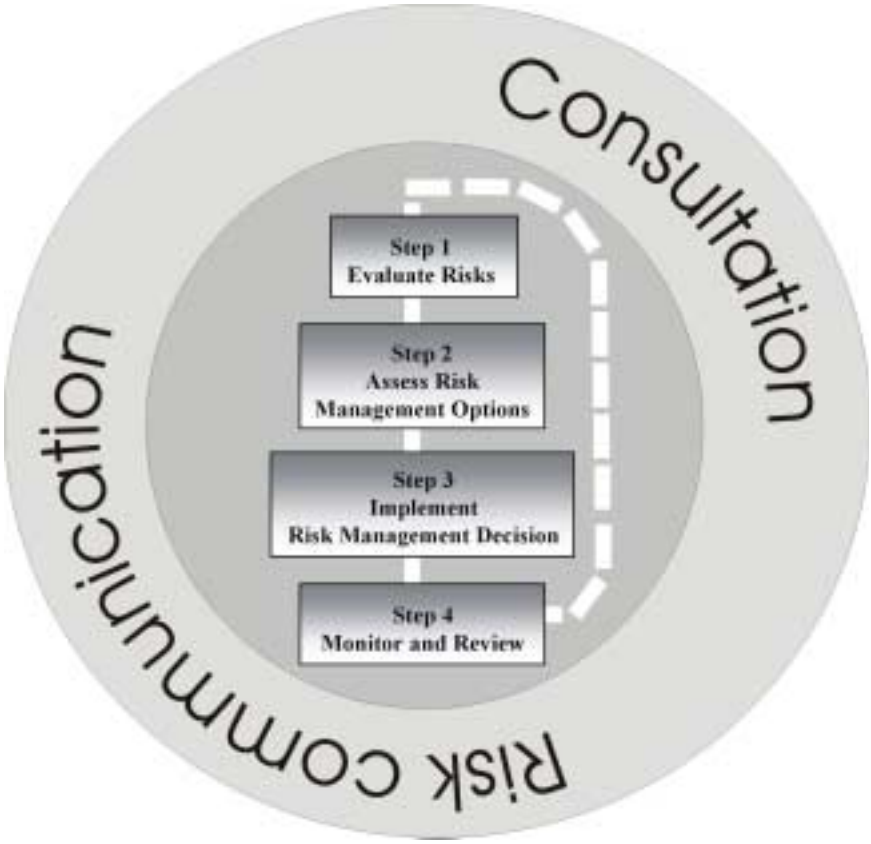

Figure reproduced from "Food Administration in New Zealand. A risk management framework for food safety" (Ministry of Health/Ministry of Agriculture and Forestry, 2000).

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.

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 toxigenic *Escherichia coli* (STEC) (shiga toxins are so named due to their similarity to those produced by some species of *Shigella* bacteria). The most well known of these is *E. coli* O157:H7 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:H7 in New Zealand occurred in 1993 (Baker *et al*., 1999).

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 characterisation:

- 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

Note: Earlier versions of this document were produced as part of a project undertaken by ESR and jointly directed by the Ministry of Health and the Ministry of Agriculture and Forestry. Responsibilities for food safety were combined into the New Zealand Food Safety Authority (NZFSA) in July 2002.

The Australia New Zealand Food Authority (ANZFA) became Food Standards Australia New Zealand (FSANZ), also in July 2002.

Information and reports published by the older organisations have been referenced to those names.

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 in 2000-2001. The data sheets are intended for use by regional public health units. Information for *E. coli* O157:H7 is presented separately from other shiga toxin-producing serotypes. The ability of the serotypes in the latter group to cause disease varies greatly, and a classification of these serotypes according to the evidence for causation of disease is given in Section 4.2.1.

2.1.1 Nomenclature

There are three acronyms that are in common use that pertain to this group of organisms. The two in most common use currently are VTEC (verocytotoxigenic *Escherichia coli*) and STEC (shiga toxigenic *Escherichia coli*). In the former case the organisms produce a toxin that causes a pathological effect on Vero tissue culture cells. In the latter, the organisms produce a shiga-like toxin, which in turn produces pathology on Vero cells. The two acronyms have now become *de facto* synonyms.

The oldest acronym is EHEC which stands for Enterohaemorrhagic *Escherichia coli*. This group of organisms specifically refers to those that cause haemorrhagic colitis (bloody diarrhoea), haemolytic uraemic syndrome or thrombotic thrombocytopaenic purpurea. Strictly it is 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.

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 NM) are considered here to be H-, i.e. without an H antigen.

Note that 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:H7

2.2.1 The organism/toxin

E. coli O157:H7 is a pathogenic variant of an organism that is generally regarded as innocuous.

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).

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 under 100% CO_2. Growth on lettuce was not inhibited by the presence of 30% CO_2, or under 97% N_2 :3% O_2.

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

Survival:

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

pH: Can survive in low pH (down to 3.6) 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 (3h).

Atmosphere: An atmosphere of 100% CO_2 enhanced survival of uninjured cells at both 4 and 10^{o}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.

2.2.3 Inactivation (CCPs and Hurdles)

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

Preservatives: 8.5% NaCl inhibits growth at 37^{o}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:H7 at 12^{o}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 in the USA is generally considered to be low, but estimates of prevalence are rising with improving laboratory techniques. Calves are thought to shed the organism more often than adult cattle. In North America the prevalence in cattle is highest during spring and late summer.

Food: Incriminated foods overseas have usually been contaminated by cattle manure. Foods involved in outbreaks have included hamburgers, other meat products, apple juice, salads, bean sprouts, raw milk, cheese, melons, lettuce and yoghurt.

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 cattle drinking trough sediment.

Transmission Routes: In summary, any food or water source that has been contaminated by the faeces of a ruminant animal. Secondary transmission is also common. Poor personal hygiene can also result in infection; 8 pop festival attendees became infected after the event, which was held in a muddy paddock on which cattle had recently been grazed.

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 toxin(s), as is *E. coli* O157:H7. However, they are of widely differing pathogenic potential, varying from those that can cause disease 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 toxins (denoted Stx1 and Stx2), but other factors are also involved in 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, and the ability to produce a haemolysin.

An isolate possessing the ability to produce either Stx in the absence of other virulence determinants is unlikely to be a major pathogen.

2.3.2 Growth and survival

The behaviour of these organisms is largely the same as for serotype O157:H7. Specific characteristics of individual serotypes are lacking.

2.3.3 Inactivation (CCPs and Hurdles)

The behaviour of these organisms is largely the same as for serotype O157:H7. Specific characteristics of individual serotypes are lacking.

2.3.4 Sources

Human: Some serotypes appear to be restricted to people, e.g. O1, O55:H7 and H:10 and O148:H21.

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 situation will be similar to that for serotype O157:H7. Non-O157 STEC are likely to be much more common than serotype O157:H7 in foods, but only a small proportion of the isolates will be pathogenic to humans.

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: Red Meat and Meat Products

As described in Section 7 below, STEC have been detected in beef, lamb and pork, both raw and in retail products, so the entire range of red meats are relevant to this risk profile. Cooked meats and ready to eat meats can also contain the organism, although it seems likely that this is due to cross contamination (PHLS, 2000).

Many STEC outbreaks have been linked to consumption of undercooked minced (or ground) beef. Risk assessments of STEC conducted in Canada and the United States have confined themselves to that type of meat (Cassin *et al*., 1998; FSIS, 1998). Transmission of the organism in processed meats is aided by its tolerance of acidic conditions, drying and fermentation (Baker *et al*., 1999).

STEC contamination of primal meat cuts will be surface contamination only and any reasonable thermal processing will inactivate the organism. This opinion was supported by the preliminary data for the FSIS risk assessment of *E. coli* O157:H7 in ground beef (FSIS, 1998):

> "Due to a low probability of pathogenic bacteria being present in or migrating from the external surface to the interior of beef muscle, cuts of intact muscle (steaks) should be safe if the external surfaces are exposed to temperatures sufficient to effect a cooked color change. In addition, the cut (exposed) surfaces must receive additional heat to effect a complete sear across the cut surfaces…"

All fresh red meats have water activities (a_w) of >0.99 which provides an excellent environment for microbial growth. Most of the extrinsic factors (salt and sugar addition, drying and smoking) applied to extend shelf life and safeguard against food poisoning act by lowering the a_w (Lawrie, 1998). The flesh of stock animals prior to slaughter has a pH of about 7.1. The pH falls post-slaughter to reach a minimum of 5.4-5.8 within 24 hours of slaughter.

Research carried out by the Meat Industry Research Institute of New Zealand (MIRINZ) on microbial growth at sub-freezing temperatures clearly indicates that meat or meat products stored at product temperatures below -8 °C will not support any microbial growth (Winger, 1984). However, if present, some pathogens will survive freezing temperatures.

E. coli survives well in frozen food. Little change was observed in the number of *E. coli* O157:H7 in beef patties during 9 months storage at -20 °C (Doyle and Schoeni, 1984), and this was confirmed by modest reductions at 2°C and -2°C (1.4 – 1.9 $\log_{10}$ CFU/g) for 4 weeks (Ansay *et al*., 1999). This is significant because most ground beef patties shipped to fast food restaurants are transported frozen. Tempering (pre-incubation of ground beef patties at 15°C for 4 hours) prior to storage at –2°C caused greater reduction in numbers (Ansay *et al*., 1999). Although growth is inhibited by high levels of background microflora in ground beef, STEC cells retained their viability when stored at 5°C (Palumbo *et al*., 1997; Vold *et al*., 2000). At temperatures above 8°C in the presence of a low background flora *E. coli* O157:H7 will grow in ground beef.

In a study that simulated the cooking of ground beef patties in a skillet as would occur in the retail food industry or in the home, it was found that achieving an internal temperature of 155°F (68.3°C) caused a four log reduction in *E. coli* O157:H7 (Juneja *et al*., 1997).

3.2 The Food Supply in New Zealand

There are 17 000 commercial sheep and beef cattle farms in New Zealand, most of which are owned and operated by farming families. Livestock numbers for New Zealand in 2001 are shown in Table 1 (MAF, 2001).

Table 1: Livestock numbers for New Zealand in 2001

Main Classes of Livestock (millions) in 2001	
Total sheep	43.99
Total beef	4.98
Total dairy	4.73
Total pigs	0.37
Total deer	2.66

In 1999 New Zealand produced 570,000 tonnes of beef meat (1% of world production). Over 80% of this production was exported, representing 10% of the world trade in beef.

Approximately 90% of New Zealand's sheep meat production is exported. The majority is frozen, but chilled meat exports now represent 12% of the total. According to the 1999 New Zealand Yearbook total production in 1998 was approximately 416,000 tonnes of lamb and 129,000 tonnes of mutton. From this production, 387,000 tonnes of lamb and 110,000 tonnes of mutton were exported.

New Zealand venison production is expected to reach 32,000 tonnes in 2001-2002. Approximately 80% of production is exported to Europe.

New Zealand has a relatively small pig industry which focuses on the domestic market. Currently about 48,400 breeding sows are farmed, with an estimated 350,700 pigs on farms at any one time (New Zealand Pork Industry Board, 2001). Since 1995 pigmeat production has been relatively static averaging 49,000 tonnes per year (46,500 tonnes in the year to September 2001; MAF, 2001).

3.2.1 Imported food

New Zealand imports relatively small amounts of beef and sheep meat, according to data from Statistics New Zealand. For the year to September 2001 approximately 4454 tonnes of beef carcasses and cuts were imported from Australia, with less than one tonne derived from the United States. For the same period, 3805 tonnes of sheep meat (all types) was imported, all from Australia.

Larger amounts of pigmeat are imported. For the year to September 2001 New Zealand imported 2859 tonnes of pig meat from Australia, 8746 tonnes from Canada, 782 tonnes from Denmark and 284 tonnes from the United States. All were frozen meat carcasses and cuts.

These data, when compared to the production and export figures above, suggest that the approximately 5% of New Zealanders beef and sheep meat for domestic consumption derive from Australia, while approximately 30% of pigmeat for domestic consumption is imported, principally from Australia and Canada.

Small amounts of processed meats are imported, principally from Australia. Meat preparations (with a non-poultry base) comprised 170 tonnes for the year to September 2001. These were not preserved in airtight can or jars. Approximately 3,400 tonnes of processed meats of bovine origin in airtight can or jars are also imported, principally from Australia.

3.2.2 Meat processing

In order to estimate the size of the meat processing industry, the number of geographic units, the number of full time employees involved and the number of retail distribution points in operation the 1999 Australia New Zealand Standard Industrial Classification (ANZSIC) tables were consulted. A summary of the figures considered relevant to the New Zealand meat food industry sector is presented in Table 2.

Table 2: Geographic units and full-time equivalent persons engaged by ANZSIC

		Geographic Units[1]		FTEs engaged[2]	
ANZSIC	**Description**	**1998**	**1999**	**1998**	**1999**
C211100	Meat Processing	221	213	21,590	20,690
C211300	Bacon, Ham and Smallgood Manufacturing	70	78	1,700	1,870
F471100	Meat Wholesaling	168	160	1,120	920
F471200	Poultry and Smallgood Wholesaling	47	54	190	250
G511010	Supermarkets	392	395	24,850	24,700
G511020	Groceries and Dairies	2,279	2,274	7,140	7,230
G512100	Fresh Meat, Fish and Poultry Retailing	765	727	2,790	2,700

1. Generally defined as enterprises with greater than $30,000 annual GST expenses or sales, or enterprises in a GST exempt industry.
2. Full-time Equivalent Persons Engaged (FTE) equal the sum of the full-time employees and working proprietors plus half the part-time employees and working proprietors.

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.

Diarrhoea Symptoms: Diarrhoea is accompanied by severe abdominal cramps. Vomiting may occur but fever is infrequent.

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.. The kidneys are attacked by toxins released by the organism.
>
> *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.

4.2 Serotypes Causing Disease

4.2.1 Non-O157 serotypes

Information from the USA estimates that infection with non-O157 STEC is half as common as infection with the O157:H7 serotype (Mead *et al*., 1999). One of the reasons for this is that not all serotypes have the factors required for pathogenicity. A hospitalisation rate of 29.5% and case fatality rate of 0.8% have been estimated for non-O157 STEC.

Non-O157 STEC serotypes are arranged below in three tiers of significance based on the history of the serotype in causing disease. (N.B. This list is likely to change over time as more STEC are recognised and the virulence of serotypes becomes better established).

Have caused HUS outbreaks or clusters: O26:H11, O111:H-, O113:H21.

Involved in HUS cases but not outbreaks: O2:H6, O5:H-, O6:H1,H4, O9:H-, O18:H7, O22:H8, O26:H-, O46:H31, O48:H21, O55:H6,H7,H10,H-, O75:H5, O86:H40, O91:H10,H21,H-, O98:H-, O103:H2,H7(?), O104:H-,H2, O105ac:H18, O111:H2,H8, O111ac:H-, O112ab:H2, O115:H10, O119:H6, O125ac:H-, O121:H19, 128ab:H2,H25, O118:H12,H16, O145:H25,H28,H-, O146:H8, O153:H25, O163:H19, O165:H-,H19,H25, O168:H-, OX3:H-.

Not implicated in cases of HUS to date: O1:H-,H1, O2:H5,H7,H29, O4:H10,H-, O5:H16, O6:H-, O18:H15,H-, O23:H7,H16, O25:H-, O26:H2,H32, O39:H4, O45:H-,H2, O50:H-,H7, O73:H19,H34, O78:H-, O82:H8, O84:H2,HNT, O91:H14,H40, O100:H32, O101:H-, O104:H21, O105:H18, O107:H27, O111:H5,H35,H49, O113:H7,H32, O114:H4,H48, O117:H4, O118:H-,H2,H12,H30, O119:H-, O121:H-, O125:H8,H-, O126:H-,H8, O128:H2,H8,H12,H-, O128ab:H-,H8, O146:H2,H21,H28,H31, O163:H-, O166:H12, O172:H-, OX3:H2,H21.

Not implicated in human disease: O2:H-, O6:H34, O8:H19, O38:H21, O39:H49, O44:H-, H25,H28,H40, O46:H38, O63:H19, O69:H11, O76:H?, O84:H-, O88:H2,H25, O98:H25, O113:H-, O116:H21, O119:H25, O121:H7, O125:H19, O136:H12, O145:H8, O153:H31, O156:H-,H25, O162:H21, O165:H52, O166:H-,H15, O169:H19.

4.2.2 Overview of international situation

It has long been held that serotype O157:H7 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. One spin off might be that methods for non-O157 STEC become developed by scientific rapid method producers (who are mostly USA based) previously focused on the market resulting from the predominance of *E. coli* O157:H7.

An earlier 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 found 130 patients infected with *E. coli* O157:H7, 29 with non-O157 STEC and seven with both. The higher ratio of non-O157 to O157 infections could be explained by the source of the samples, i.e. it is possible that O157 infections will represent a larger proportion of HUS cases than other forms of the disease.

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). 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:H7, 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 reliable methods for detecting these infections.

The pattern of transmission of 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

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).

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 Red Meat

5.1.1 STEC in meat animals: O157:H7

Only one paper attempting to measure the prevalence of *E. coli* O157:H7 in New Zealand livestock has been published. Buncic and Avery (1997) sampled the faeces of 371 cows originating from 55 farms. Two (0.5%) of these samples were positive for the presence of *E. coli* O157:H7.

5.1.2 STEC in meat animals: other serotypes

There are no reports of isolations of non-O157 STEC from the faeces of New Zealand farmed meat animals.

5.1.3 STEC in meat: O157:H7

The New Zealand Ministry of Agriculture and Forestry has been monitoring meat products for the presence of *E. coli* O157:H7 for approximately 5 years. 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 indicate that to September 2001, from 113,890 samples of bulk meat for export only two (0.002%) were positive for *E. coli* O157:H7 (Dr Roger Cook, NZFSA, personal communication).

5.1.4 STEC in meat: other serotypes

In addition to the isolates from human cases, the ESR Enteric Reference Laboratory has been asked to serotype approximately 90 STEC isolates from meat samples. To date none of these have been serotype O157:H7 or other serotypes associated with high risk of virulent disease (Carolyn Nicol, ESR, personal communication).

A study of the isolation of STEC using a specific agar examined 15 retail meat products (raw minced beef or pork, vacuum packed sliced meat and salami) (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, which has not been involved in HUS cases.

More recently 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 beef, 17% of lamb, and 4% of pork retail raw meat samples (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. Serotype O156:H- is not pathogenic to humans.

Serotypes O5:H- and O91:H-, isolated from lamb have been reported to be involved in HUS cases, but not outbreaks (see section 4.2). The remaining serotypes were either non-typable, not implicated in human disease, or were of unknown pathogenicity. Those serotypes whose

pathogenicity was unknown were analysed for an array of genes which are thought to be indicative of potential pathogenicity. The findings are given in Table 3.

Table 3: Details of Serotypes Described by Brooks *et al.* (2001)

Serotype	*stx1*	*stx2*	*stx1* and *stx2*	*eaeA*	*HlyA*
O144:H2	-	-	+	-	+
O27:H21	-	+	-	-	-
O8:H-	-	+	-	-	-
O15:H27	-	-	+	-	-
O171:H2	-	+	-	-	-
O15:H27	-	-	+	-	-

(Column headings represent pathogenicity determinants. Conventionally serotypes without the *eaeA* gene are considered to be of less concern, although there are exceptions to this generalisation)

The same group had earlier isolated a number of potential STEC from children (Brooks *et al.*, 1997), but only serotypes O26:H11 and O128:H2 were toxigenic and typable.

Work carried out by ESR for the Ministry of Health during 2000-2001 tested 97 beef, 65 chicken, 66 lamb and 73 pork samples of minced or cubed meat for all STEC. Only five lamb samples (7.6%) were positive. The isolates obtained were; ONT:H-, ONT:H8, O123:H51, Orough:H-, O75:H8, and O128, H2 (ONT = non-typeable from O antigen, Orough = agglutinates with all sera). 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 give higher prevalences but need not necessarily have yielded any isolates for serotyping.

None of these serotypes have been reported to be involved in HUS cases or outbreaks (see section 4.2).

5.1.5 Conclusions

The main conclusion that can be drawn from these data is that while STEC can be isolated from raw meats in New Zealand, very few of the serotypes isolated correspond to those that have caused serious disease in humans. Internationally the most common serotypes causing outbreaks or clusters of serious infections are; O26:H11, O111:H-, O113:H21 and O157:H7. Many STEC serotypes either infrequently cause disease or have yet to be associated with disease. By far the majority of isolates from New Zealand meat fall into these two categories.

Isolation rates of *E. coli* O157:H7 from raw meats from meat processing plants in New Zealand appear to be particularly low.

5.2 Food Consumption: Red Meat and Meat Products

Red meat consumption has declined since 1985, as shown in Table 4. A major shift in consumption patterns has taken place with major gains by the poultry and pork industries.

Table 4: New Zealand domestic meat consumption per capita 1985, 1995, 1996 & 1999 (kg/person/year)

Year	Sheep and Lamb	Beef and Veal	Pig meat	Total Red meat	Poultry	Total Meat
1985	27.3	36.5	14.2	78.0	15.0	93.0
1995	23.2	34.6	15.7	73.5	26.2	100.1
1996	20.6	37.8	16.1	74.5	25.1	99.8
1999	14.3	31.2	17.1	62.6	26.8	89.5

From New Zealand Meat and Wool Board's Economic Service (MWBES) Annual Review of the Sheep and Beef Industry, 1999-2000.

The meat consumption figures for New Zealand in Table 1 are similar to estimates made for the Australian population (Baghurst, 1999). The Australian consumption levels for 1996-97 were; beef 40.2 kg/person/year, sheep and lamb 17.5 kg/person/year, pig meat 17.9 kg/person/year, and poultry 10.2 kg/person/year.

An international comparison of meat consumption as calculated for 1998 is given in Table 5.

Table 5: International comparison of meat consumption, 1998 (kg/person/year)

Country	Red Meat consumption	White meat consumption	Total meat consumption
Argentina	64.7	24.4	89.1
Australia	57.9	50.0	107.9
Canada	32.8	64.3	97.1
New Zealand	54.1	42.3	96.4
USA	45.2	77.8	123.0
UK	22.2	50.8	73.0

Source: USDA; MWBES

The figures given above represent the meat available for consumption in New Zealand. Information on amounts of meat reported to be actually consumed by individuals can be abstracted from the 1997 National Nutrition Survey (NNS) (Russell *et al.*, 1999). FSANZ have carried out an analysis of this dataset (ANZFA, 2001), including application of a set of standard recipes, to allow composite foods to be reduced to their component parts. Table 6 gives the estimates for New Zealand domestic meat consumption derived by ANZFA and compares those levels of consumption to the estimates based on meat available for consumption (Table 4).

Table 6: Mean estimates of New Zealand domestic meat consumption (total population over 15 years), 1997 and estimates of meat available for consumption, 1996 (g/person/day)

Meat type	Estimated consumption (1997)*	Amount available for consumption (1996)#
Beef and veal	87.9	103.6
Sheep and Lamb	13.7	56.4
Pig meat	32.3	44.1
Deer meat	0.9	
Rabbit meat	0.1	
Total red meat	**134.9**	**204.1**
Poultry	35.4	68.8
Total meat	**170.3**	**272.9**

* from ANZFA analysis of 1997 National Nutrition Survey data (ANZFA, 2001)
\# from Table 1, recalculated from kg/person/year to g/person/day

The difference between these two estimates of consumption will reflect wastage (meat available for consumption, but not consumed), and under-reporting in the NNS. Through use of standard recipes, the FSANZ analysis of the 1997 NNS data will include all meat consumed, including meat which is consumed as a component of a processed food such as meat pies or luncheon meat (ANZFA, 2001).

The analysis of the 1997 NNS data concluded that 77.7% of the population consumed red meat (cattle, sheep or pig meat) during any 24 hour period. The mean daily consumption, for consumers only, was 172.5 g/day. The median daily consumption, for consumers only, was 124.1 g/day. The 97.5th percentile daily consumption, for consumers only, was 616 g/day.

Table 7 represents an analysis of dietary records from the 1997 National Nutrition Survey and shows a breakdown of total red meat and red meat product consumption on the basis of number of servings and on the basis of consumption weight.

Table 7: Types of red meat and meat products consumed, by servings and by weight

Meat type	Percentage of total red meat consumed (by servings)	Percentage of total red meat consumed (by weight)
Beef (including veal)		
Corned beef	6.3	5.0
Beef offals	0.6	0.5
Beef mince and beef mince recipes (pattys, hamburgers, etc)	14.7	24.1
Beef cuts (steak, roast, schnitzel, etc)	20.2	26.2
Sheep meat (Lamb, hoggett and mutton)		
Hoggett/mutton cuts	4.1	3.6
Lamb cuts	6.0	5.2
Lamb mince and lamb mince recipes	0.1	0.1
Lamb offals	0.6	0.6
Pigmeat (including ham and bacon)		
Pigmeat cuts	6.8	8.3
Pigmeat mince	0.1	0.1
Pig offals	0.2	0.1
Bacon	7.3	2.9
Ham	11.5	4.3
Mixed meat products		
Sausages, saveloys, frankfurters and hotdogs	13.6	15.1
Salami	1.6	0.5
Luncheon meat	4.1	1.7
Other meats		
Venison	0.4	0.5

5.3 Qualitative Estimate of Exposure

5.3.1 Number of servings and serving sizes

Red meat and red meat products are commonly consumed products with 77% of respondents in the 1997 National Nutrition Survey reporting consumption of beef, sheep or pigmeat in any 24 hour period. This category of food represents one of the most commonly consumed in New Zealand. Only categories such as dairy products and cereal grains (bread, breakfast cereals, etc.) and water are consumed by a greater percentage of the population on any given day. The greatest contributors to total servings are beef cuts, beef mince and beef mince products, sausages (including saveloys, frankfurters and hotdogs) and ham.

Serving sizes will vary considerably within the red meat and meat products group from hundreds of grams for a meal of meat cuts to a few grams for ready-to-eat meats consumed as a component of a sandwich. According to the FSANZ analysis of the National Nutrition

Survey data the average daily consumption of red meat by consumers (only those reporting consumption of red meat) is similar to average daily consumption for consumers of common fruits and vegetables.

5.3.2 Frequency of contamination

There is little information on the incidence of STEC serotypes in New Zealand meat and meat products. Export monitoring data suggest a very low incidence of *E. coli* O157:H7 of 0.002%, however, this incidence rate is based on only two positive detections and has a 95th percentile confidence interval of 0 – 0.007%. This initially suggests that contamination rates in New Zealand meat may be low by international standards (see section 5.4.1), however, the lack of information on *E. coli* O157:H7 on meat and meat products at retail makes comparison difficult.

While a wide range of non-O157:H7 STEC serotypes have been isolated from New Zealand meat samples, the majority of isolates appear to be of serotypes which have not been associated with any adverse human health outcomes.

5.3.3 Predicted contamination level at retail

The paucity of quantitative data for *E. coli* O157:H7 and non-O157 STECs means that it is not possible to predict contamination levels at retail. Limited dose-response information is available for *E. coli* O157:H7, however, information from overseas outbreak investigations (section 5.4.1) suggests that even very low levels of contamination (<0.3 most probable number of cells (MPN)/g) can result in illness.

5.3.4 Growth rate during storage and most likely storage time

The normal acidity of meat and processed meat products and their storage under refrigeration or freezing suggests that levels of contamination are unlikely to increase during storage, provided refrigeration temperatures are maintained.

5.3.5 Heat treatment

STECs are readily inactivated by normal cooking temperature.

5.3.6 Exposure summary

There is little information available on STEC contamination of New Zealand meat and meat products. Export monitoring of *E. coli* O157:H7 contamination of carcass meats suggests that rates of contamination may be low by international standards. However, there is insufficient information at this time to estimate likely exposure to STECs.

5.4 Overseas Context

5.4.1 STEC in Meat: O157:H7

Information summarising data for the prevalence of *E. coli* O157:H7 in meat products is given in Table 8.

Table 8: Prevalence of *Escherichia coli* O157:H7 in meat from overseas surveys

Country	Products tested	Number tested	% positive	Year of publication
Australia	Beef carcasses	893	0.5	Vanderlinde *et al.*, 1998
Australia	Frozen packed beef	685	0	Vanderlinde *et al.*, 1998
Australia	Sheep carcass meat	465	0	Vanderlinde *et al.*, 1999
Australia	Bulk frozen sheep meat	343	0.3	Vanderlinde *et al.*, 1999
Australia	Sheep carcasses	917	0.7	Phillips *et al.*, 2001a
Australia	Frozen boneless sheep meat	467	1.3	Phillips *et al*., 2001a
Australia	Beef carcasses	1275	0.1	Phillips *et al.*, 2001b
Australia	Boxed frozen boneless beef	990	0	Phillips *et al.*, 2001b
Belgium	Beef carcasses	310	1.0*	Korsak *et al*., 1998
Belgium	Pork carcasses	245	2.9*	Korsak *et al*., 1998
Brazil	Hamburgers	886	0	Silviera *et al.*, 1999
Canada	Beef carcasses	125	0	Power *et al.*, 1998
England	Beef products (raw)	3216	1.1	Chapman *et al.*, 2000
England	Lamb products (raw)	1020	2.9	Chapman *et al.*, 2000
England	Mixed meat products (raw)	857	0.8	Chapman *et al.*, 2000
England	Raw prepared meats	2330	0.1	Little and de Louvois,1998
England	Cooked meats	2192	0	Little and de Louvois,1998
England, Wales and Northern Ireland	Dried and fermented meat and meat products	2981	0	Little *et al.*, 1998
Holland	Minced mixed beef and pork	770	0.3	Heuvelink *et al*., 1996
Holland	Raw minced beef	1000	0	Heuvelink *et al*., 1996
Holland	Minced pork	260	0	Heuvelink *et al*., 1996
Holland	Raw minced beef	571	1.1	Heuvelink *et al.*, 1999
Holland	Raw minced mixed beef and pork	402	0.5	Heuvelink *et al.*, 1999
Holland	Raw minced pork	76	1.3	Heuvelink *et al.*, 1999
Holland	Other pork products	393	0.3	Heuvelink *et al.*, 1999
Holland	Cooked or fermented RTE meats	328	0.3	Heuvelink *et al.*, 1999
Holland	Other raw beef products	223	0	Heuvelink *et al.*, 1999
Holland	Sheep or lamb products	46	0	Heuvelink *et al.*, 1999
Spain	Hamburgers	58	5.0	Blanco *et al.*, 1996
USA	Beef products	164	3.7	Doyle and Schoeni, 1987
USA	Pork products	264	1.5	Doyle and Schoeni, 1987

Country	Products tested	Number tested	% positive	Year of publication
USA	Lamb products	205	2.0	Doyle and Schoeni, 1987
USA	Beef carcasses	2089	0.2	McNamara, 1995
USA	Beef carcasses	330	1.8	Elder *et al.*, 2000
USA	Retail ground beef	1400	0	Tarr *et al.*, 1999

[*]Only three isolates from 10 presumptive positives were obtained, and only one of these three (from beef) had the full complement of pathogenicity genes and activities examined. True adjusted prevalences are therefore 0% for pork and 0.3% for beef.

In general contamination of meat is at a low prevalence, mostly being reported in the 0-2% range. The limited New Zealand data available would suggest that contamination rates in New Zealand are at the lower end of this range (see section 5.2.3).

Some quantitative data are available for products involved with outbreaks or cases caused by *E. coli* O157:H7. This information is presented in Table 9.

Table 9: Quantitative data for the prevalence of *Escherichia coli* O157:H7 in meat products associated with outbreaks or cases overseas

Country	Products tested	Prevalence	Reference
England	Minced beef	4 (MPN/g)	Bolton *et al*., 1996
	Beefburgers	0.3 (MPN/g)	Bolton *et al*., 1996
	Beefburgers	2300 (MPN/g)	Bolton *et al*., 1996
	Beefburgers	2.3 (MPN/g)	Bolton *et al*., 1996
	Beefburgers	<0.3 (MPN/g)	Bolton *et al*., 1996
USA	Beef patties	<0.3-15 (median 1.5) (MPN/g)	Tuttle *et al.*, 1999
	Beef carcasses	Maximum <1, mean 0.6 (count/cm^2)	McNamara, 1995

Since these data are for foods that have caused disease it is apparent that the levels of the organism required to cause disease are very low. As with many other pathogens there is no safe level of organisms that can be ingested, and the probability of infection is determined by 1) the individual 2) the food and 3) the dose response characteristics of the organism for the person consuming the food.

6 RISK CHARACTERISATION

The public health significance of infection with STEC derives from the high proportion of cases which have serious consequences, beyond gastrointestinal disease. Infection with STEC can affect any age group but most often causes disease in infants (< 4 years) and the elderly (>65 years).

These consequences, HC, HUS, and TTP, are described in section 4.1. Children under five years are most susceptible to HUS whereas the elderly are more likely to develop TTP (Baker *et al.*, 1999).

6.1 Adverse Health Effects in New Zealand

6.1.1 Incidence

The number of cases of infection with STEC in New Zealand increased steadily throughout the 1990s. The rates are shown in Table 10.

Table 10: Rates of infection with STEC in New Zealand 1998 – 2001

Year	Rate per 100,000 (number of 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

The analysis of data up to 1998 showed that rates in children are much higher than for adults. The highest rates of STEC infection in 1998 were in children aged less than one year (14.6 per 100,000) and those aged 1-4 years (10.2 per 100,000). Rates for males and females (1.1 and 1.5 per 100,000 respectively) were similar, as were rates in Europeans and Pacific Islanders (1.4 and 1.2 per 100,000). Rates were lower in Maori (0.4 per 100,000) (Baker *et al*., 1999).

Note that these rates are for all shiga toxin-producing *E. coli* and serotype O157:H7 accounts for around 90% of the notified cases.

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).

6.1.2 Clinical consequences of STEC infection

The clinical consequences of STEC infection of cases in New Zealand are summarised in Table 11.

Table 11: Summary of clinical consequences of STEC infection in New Zealand

Period	Hospitalised*	HC*	HUS*	TTP*	Fatalities
Oct 1993-Dec 1998[1]	24/58 (41.4%)	21/59 (35.6%)	18/59 (30.5%)	1/59 (1.7%)	2/79 (2.5%)
1999[2]	20/60 (33%)	NS	2/64 (3.1%)	NS	0
2000[3]	11/65 (16.9%)	NS	3/68 (4.4%)	NS	0
2001[4]	16/74 (21.6%)	NS	6/76 (7.9%)	NS	0

1 Baker *et al.*, 1999
2 Kieft *et al.*, 2000
3 Lopez *et al.*, 2001
4 Sneyd *et al.*, 2002

*Percentages are determined on the basis of cases for which information was available
NS Not stated

6.1.3 Serotypes causing disease

Approximately 90% of notified STEC infections in New Zealand are caused by *E. coli* O157:H7. Other serotypes causing infections have included O113:H21, O26:H-, O91:H21, O145:H-, ONT:H18, ONT:H6, ONT:H- and O128:H-. There have been two deaths attributed to STEC, one to serotype O157:H7 and the other to O113:H21.

The serotypes ONT:H- and O128:H- have recently been isolated from New Zealand meat samples (Brooks *et al.*, 2001).

6.1.4 Case control studies and risk factors

There have been no New Zealand case control studies to identify risk factors. The overview of 79 cases of STEC in New Zealand 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).

An analysis of risk factors associated with STEC infection for cases from June – December 1999 was given in the Annual Surveillance Summary (Kieft *et al*., 2000). A high (>50%) proportion of cases reported consumption of beef, poultry, processed meats, and raw fruit and vegetables, but consumption of pink/undercooked meat was not. Animal contact was another common factor. However, the analysis cautioned that these are common factors in New Zealanders lives and the proportions may simply reflect that fact, and the number of cases was too low to draw meaningful conclusions.

6.2 Adverse Health Effects Overseas

6.2.1 Incidence

Incidence rates for a selection of countries/states are given in Table 12. New Zealand's incidence has been included for comparison and is on a par with other countries.

Table 12: New Zealand and international rates of reported infections with STEC

Country	Year	Incidence (per 100,000)	Reference
New Zealand	2001	2.0	Sneyd *et al.*, 2002
Australia	2001 (Jan-Mar)	0.4	Communicable Diseases Australia[1]
England and Wales	1990-1998	<2	Pennington, 2000
Finland	1996-1997	0.16	Keskimakii and Siitonen, 1997
Scotland	1990-1998	2-10 (approx.)	Pennington, 2000
USA	1996	2.7	Kightlinger and Schaefer, 1999
USA	1997	2.3	Kightlinger and Schaefer, 1999
USA	1998	2.8	Kightlinger and Schaefer, 1999

[1] http://www.health.gov.au/pubhlth/cdi/cdi2502/pdf/cdi2502o.pdf

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, more recent data show rates closer to overseas values (see Table 11).

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).

For the USA it has been estimated that for each confirmed case of infection with STEC that is reported, 13-27 cases of *E. coli* O157 infection occur in the community (Mead *et al*., 1999). These cases have mild symptoms. The total number of cases of infection with non-O157 STEC has been assumed to be 50% of the rate for O157:H7 (Mead *et al*., 1999).

6.2.2 Contributions to outbreaks and incidents

The proportion of outbreaks caused by *E. coli* O157:H7 overseas is summarised in Table 13. Only a small proportion of outbreaks are attributable to STEC.

Table 13: Proportions of outbreaks and incidents caused by *Escherichia coli* O157:H7

Country	Year	Proportion of outbreaks (%)	Reference
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. These are summarised for *E. coli* O157:H7 in Table 14, and for other STEC in Table 15.

Table 14: Specific Incidents of Disease Reported for *Escherichia coli* O157:H7 Associated with Meat Products

Location	Setting	No. affected	No. deaths	Source	Reference
Canada	Home	25	NS	Salami	Williams *et al*., 2000
Scotland	Church lunch, birthday party and homes	496	20	Retail meats	Pennington, 1998
USA	Community	15 (5 hospitalised, 0 HUS)	0	Frozen beefburgers	CDC, 1997
USA	Community	>700	4	Hamburgers	Tuttle *et al*., 1999
USA	Community	65	NS	Roast beef	Doyle *et al*., 1997
USA	Picnic	2	NS	Ground beef	Doyle *et al*., 1997
USA	Fair	8	NS	Ground beef	Doyle *et al*., 1997
USA	Community	13	NS	Ground beef	Doyle *et al*., 1997
USA	Community	58	NS	Ground beef	Doyle *et al*., 1997
USA	Community	32	NS	Ground beef	Doyle *et al*., 1997
USA	Community	10	NS	Ground beef	Doyle *et al*., 1997
USA	Home	10	NS	Ground beef	Doyle *et al*., 1997
USA	Community	3	NS	Ground beef	Doyle *et al*., 1997
USA	Club barbecue	23	NS	Ground beef	Doyle *et al*., 1997
USA	Community	8	NS	Ground beef	Doyle *et al*., 1997
USA	Home	11	NS	Ground beef	Doyle *et al*., 1997
USA	Home	10	NS	Ground beef	Doyle *et al*., 1997
USA	Unknown	8	NS	Ground beef	Doyle *et al*., 1997
USA	Home, camp	24	NS	Ground beef	Doyle *et al*., 1997
USA	Restaurant	33	NS	Ground beef	Doyle *et al*., 1997
USA	Home	9	NS	Ground beef	Doyle *et al*., 1997
USA	Home	17	NS	Ground beef	Doyle *et al*., 1997
USA	Home	21	NS	Retail meats	Doyle *et al*., 1997
USA	Community	2	NS	Ground beef	Doyle *et al*., 1997
USA	Home	4	NS	Ground beef	Doyle *et al*., 1997
USA	Home	15	NS	Salami	Doyle *et al*., 1997
USA	Home	4	NS	Salami	Doyle *et al*., 1997
USA	Home	6 confirmed, 5 suspected	NS	Home made venison jerky	Keene *et al*., 1997
USA	Community	58	NS	Hamburgers	Cieslak *et al*., 1997
Wales	Community	7 (1 HUS)	NS	Beefburgers	Willshaw *et al*., 1994

NS=Not Stated

Table 15: Specific Incidents of Disease Reported for non-O157 STEC Associated with Meat Products

Serotype	Location	Setting	No. affected	No. deaths	Source	Reference
O111:H-	Australia	Community	23 HUS, 30 bloody diarrhoea, 105 other GI symptoms	1	Fermented sausage	CDC, 1995

HUS = Haemolytic Uraemic Syndrome, GI = Gastrointestinal

In Australia *E. coli* O111:H- is the predominant serotype causing infections (Park *et al.*, 1999).

6.2.3 Case control studies

Published studies identifying consumption of meat products as risk or protective factors from overseas are summarised in Table 16.

Table 16: Case control studies where meat consumption was identified as a risk factor for infection with STEC

Location	Risk factors identified	Reference
Belgium	No link could be identified between sporadic STEC infection and meat consumption	Pierard *et al*., 1999
Canada	Consuming pink ground beef (sporadic cases)	Le Saux *et al*., 1993
Germany	Mortadella sausage, Teewurst sausage (outbreak)	Ammon *et al*., 1999
UK	Consuming hot dogs (sporadic cases)	Bryant *et al*., 1989
USA	Consuming hamburgers (sporadic cases)	Mead *et al*., 1997
USA	Eating undercooked hamburgers (sporadic cases)	Slutsker *et al*., 1998

There is now emerging a difference in the epidemiology of STEC infections in central Europe compared to the USA. In the USA meat products, and to a lesser extent other foods, are still the main risk factors. In central Europe they appear not to be (note the Italian study cited above).

6.2.4 Risk assessments and other activity overseas

The Food Safety and Inspection Service of the USDA is currently undertaking a risk assessment for *E. coli* O157:H7 in ground beef (see: http://www.fsis.usda.gov/OPHS/ecolrisk/home.htm). The risk assessment is intended to identify the occurrence and concentration of this pathogen at specific points from farm to table, and will contribute to a risk reduction strategy and identify future research needs. Preliminary results from this risk assessment indicated that between 16 and 40% (with a most likely value of 18%) of all cases of infection with *E. coli* O157:H7 are due to ground beef.

A draft of this risk assessment was published in November 2001 (www.fsis.usda.gov/OPDDE/rdad/Notices01.htm). While this contains a significant amount of useful reference material the study is very much oriented to the American meat production system, as would be expected. Since the New Zealand situation is very different with, for example, the virtual absence of feedlot cattle and a different seasonal pattern of human infections, little can be taken from the risk assessment that would assist with the New Zealand situation.

The document does list several data gaps, and carries out sensitivity analysis on factors that could help to mitigate disease. Interestingly the effect of reducing the proportion of contaminated lots of beef seemed to be greater than reducing the level of contamination. Other conclusions, such as the projection that adequate storage of minced beef and correct cooking of hamburgers, meat balls, meat loaf etc. would result in a huge decrease in cases seem obvious. However, given that the transmission routes in New Zealand are unclear, the effect of these interventions cannot be extrapolated to New Zealand.

A quantitative risk assessment for *E. coli* O157:H7 in ground beef hamburgers for Canada was published in 1998 (Cassin *et al*., 1998). A Process Risk Model was constructed to simulate the manufacturing process, and used to develop a mathematical model of exposure assessment and dose-response for *E. coli* O157:H7 in hamburgers. The distribution of risk from a single hamburger meal was used to calculate an average value of the probability of illness from a single meal. For adult members of the population this risk was 5.1×10^{-5}, and for children the probability of illness was estimated to be 3.7×10^{-5} per meal. Using the conditional probabilities of HUS and mortality, the mean probability of HUS and mortality among children was estimated at 3.7×10^{-6} and 1.9×10^{-7}, respectively.

The model was also used to estimate the effect of three mitigation strategies: better retail storage temperature control, pre-slaughter screening to reduce shedding of organisms, and consumer information to improve cooking practices. Of these strategies, the highest predicted reduction in illness derived from improvements in retail storage temperature control (only modest shifts in consumer cooking behavior were anticipated).

The large outbreak of infection with *E. coli* O157:H7 in Scotland in 1996, in which 496 people were affected, with 18 deaths, was intensively investigated by an expert group led by Professor Hugh Pennington. The report recommended a range of control measures to be introduced at all stages of meat supply: the farm, slaughter house, meat production premises/butchers shops, and homes (Parry and Palmer, 2000). At the consumer level the recommendations were for:

- food hygiene training in primary and secondary school curriculum;
- cooking instructions supplied with burgers;
- meat should be capable of reaching an internal temperature of 70°C for 2 minutes or equivalent.

In September 2000 the United Kingdom Food Standards Agency Scotland assembled a Task Force on *E. coli* O157. The Task Force was created in response to evidence that showed that the majority of sporadic cases were associated with contact with animals or from the

environment, as well as contaminated food. Guidance on reducing the risks from these sources was released in February 2001

(http://www.foodstandards.gov.uk/press_releases/scotland/2001/prs010222.htm).

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

Data on the prevalence of non-O157 STEC in meat in New Zealand are limited, and the existing data have not found serotypes which are associated with serious disease (Brooks *et al*., 2001). The prevalence of the dominant disease causing serotype, O157:H7, appears to be extremely low (0.002%) in New Zealand raw meat samples, compared with up to 2% reported for surveys overseas. This may be due to effective control in meat processing facilities, as the data for the presence of the organism in faeces from cows indicate a prevalence of 0.5%.

Approximately 5% of the beef and sheep meat supply in New Zealand appears to be imported from Australia, where higher rates of *E. coli* O157:H7 contamination of raw meat have been found in some surveys (Table 8). Although approximately 30% of the pigmeat supply in New Zealand is imported from Australia and Canada, and *E. coli* O157:H7 has been found in some pork mince samples overseas (Table 8), this type of meat has less frequently been associated with illness (Table 13).

Ground beef is the most common red meat vehicle identified in outbreaks overseas. The limited surveys of retail meat in New Zealand have not isolated pathogenic serotypes of STEC.

Little information on transmission was available from the analysis of cases in New Zealand between 1993 and 1998 (Baker *et al*., 1999). The cases are more common in rural areas suggesting that environmental exposure is important. This would be consistent with experience for sporadic cases in Scotland.

There is currently little information to suggest that transmission of STEC via red meat is occurring in New Zealand.

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. The total rate of STEC infection (including unreported cases) attributable to food contamination in New Zealand was thus estimated to be of the order of 1.4 per 100,000 of population.

However, there is currently no evidence linking red meat consumption to cases of STEC infection in New Zealand. The prevalence of the dominant pathogenic serotype (O157:H7) in red meat in New Zealand is low by international standards.

Thus the rate of STEC infection due to transmission in red meat will be considerably less than 1 per 100,000 of population. This places STEC in red meat and meat products in the lowest incidence category.

6.5 Summary

Food/hazard combination	Severity	Incidence	Trade importance	Other considerations
STEC in red meat and meat products	1 (>5% serious outcomes)	4 (<1 per 100,000)	High (control essential)	

7 RISK MANAGEMENT INFORMATION

7.1 Relevant Food Controls

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.

The application of HACCP based food safety plans is being promoted by MAF. 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. In addition to the National Microbiological Database that has been established by MAF, a separate voluntary testing regime is in place for STEC, principally for exports to the United States.

This legal situation is changing with the introduction of the Animal Products Act.

7.1.1 The 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.

The Animal Products Act (except for Part 2) and the transitional Act commenced on 1 November 1999. Part 2 of the Animal Products Act commenced on 20 November 2000. Part 2 provides the requirements for risk management programmes.

The risk management system potentially applies anywhere in the value chain from production, through processing to the market. The risk management system comprises the following main types of controls:

- risk management programmes;
- regulated control schemes; and
- controls relating to the export of animal material and animal products.

By 1 November 2002, 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.

A risk management programme is a documented programme to identify and manage biological, chemical and physical hazards. The programme is to be based on the principles of Hazard Analysis and Critical Control Point (HACCP): identifying the hazards, the systems of

control, and demonstrating that the controls are effective. Risk management programmes are to be designed by individual businesses for the animal materials used, the processes performed and the product range produced.

7.1.2 Monitoring compliance with standards

All USA listed beef and sheep slaughter premises and packing houses in New Zealand participate in a mandatory microbiological monitoring programme. The results are collated by the National Microbiological Database (NMD) which is operated by MAF. The rationale for the scheme was to demonstrate the 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.* Samples are collected from boxed manufacturing beef at the end of the production system, immediately prior to freezing. (Source: http://www.maf.govt.nz/animalproducts/publications/manualsguides/nmd/index.htm).

In 1998 MAF assessed the needs of New Zealand beef exporters with respect to the food safety and due diligence requirements of the USDA Food Safety and Inspection Service with respect to *E. coli* O157:H7. It was concluded that the New Zealand meat hygiene programme along with a national testing regime for *E. coli* O157:H7 as part of the NMD satisfied the USA requirements for a "validated pathogen reduction intervention on beef carcasses". Such a testing programme was then developed by the New Zealand Meat Industry Standards Council, and industry results are provided to the NMD. Provision of results to the NMD is not mandatory but is voluntary, and highly recommended.

7.1.3 Raw comminuted meat/salami processing

In 1999 a survey of the manufacturing practices of some of the larger salami processors in New Zealand was conducted on behalf of the Ministry of Health with the support of the Pork Industry Board (Hasell, 2000). It was found that while HACCP based food safety programmes were not in evidence in the major companies producing raw comminuted meat products in New Zealand, all the companies surveyed had much of their systems documented and were working towards the adoption of HACCP.

It was recommended that the industry should be supported in their initiative to develop food safety programmes. Once these are available, it was recommended that the Ministry of Health consider making HACCP based food safety programmes compulsory for the manufacturers of uncooked meat products. This would now be a decision for the NZFSA.

7.1.4 Consumers

The current advice in the United States on cooking of hamburger meat to prevent STEC infection is as follows:

"What can you do to prevent *E. coli* O157:H7 infection?

Cook all ground beef and hamburger thoroughly. Because ground beef can turn brown before disease-causing bacteria are killed, use a digital instant-read meat thermometer to ensure

thorough cooking. Ground beef should be cooked until a thermometer inserted into several parts of the patty, including the thickest part, reads at least 160º F. Persons who cook ground beef without using a thermometer can decrease their risk of illness by not eating ground beef patties that are still pink in the middle."

(from Centres for Disease Control website
http://www.cdc.gov/ncidod/dbmd/diseaseinfo/escherichiacoli_g.htm)

This advice to use a thermometer replaces previous advice to cook hamburger until it turned brown. However, a study by the USDA found that 25% of hamburger patties had turned brown by the time they had reached a temperature of 150°F, while nearly 50% of patties retained some pink colour when cooked to 160°F (study reported at: http://www.fsis.usda.gov/OPHS/prebrown.htm).

7.2 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.

In the United States the estimated annual foodborne illness cost of *E. coli* O157:H7 and *E. coli* non-O157 STEC has been estimated as US$1 billion (figures for 1998 updated for 2000). These costs derive from estimated annual cases of approximately 94,000, 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, nontyphoidal *Salmonella* and *Listeria monocytogenes* (Crutchfield and Roberts, 2000). These figures are high in comparison with New Zealand 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*., 1999). The percentage of cases caused by foodborne transmission in the United States has more recently been estimated as 85% (Mead *et al*., 1999).

8 CONCLUSIONS

8.1 Description of Risks to New Zealand Consumers

8.1.1 Risks associated with red meat and meat products

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; no outbreaks have yet been detected. Information on transmission routes is meager.

As stated in Section 6.3 there is little evidence to suggest that red meat represents a risk for transmission of pathogenic STEC in New Zealand. Nevertheless *E. coli* O157:H7 has been found in the faeces of cows in New Zealand as well as raw meat samples. A small proportion (approximately 5%) of beef and sheep meat in New Zealand also derives from Australia where contamination of carcasses and frozen meat may occur (Table 8). Consequently there is potential for the presence of STEC in retail meats in New Zealand. Currently available information on the prevalence of STEC in retail meat products suggests that only serotypes of low pathogenic potential are present.

8.1.2 Risks associated with other foods

In the United States ground beef/hamburger is the food vehicle most likely to be implicated in outbreaks of *E. coli* O157:H7, while the limited information from Europe suggests that meat consumption is not associated with sporadic cases. Other food vehicles implicated in outbreaks suggest contaminated foods not cooked prior to consumption (lettuce, salads, coleslaw) or consumption of 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. There is no current information to indicate the relative risk of red meat compared with other foods as a vehicle in New Zealand.

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 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:H7 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 would be premature, and could not be conducted 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 the likelihood of foodborne transmission in red meat. 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 domestic meat supply. This approach matches efforts in the United States to control STEC in red meat.

The adoption of similar programmes by New Zealand cooked meat product manufacturers is in progress. A follow-up examination of the status of HACCP programmes within this industry group, alongside assistance with implementation, could be valuable.

Control efforts directed towards consumers would aim to achieve adequate cooking of meat products. Thorough cooking is one of the key messages already being promoted by the New Zealand Foodsafe Partnership. Given that there is little evidence to suggest that red meat represents a risk for transmission of STEC in New Zealand additional efforts seem unnecessary at this stage.

Compared to the United States, New Zealand appears to be fortunate in being able to take a preventative approach to the transmission of STEC in red meat. However, the current rate of STEC infection in New Zealand is similar to overseas. The absence of information on local transmission routes presents the main target for further investigation.

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APPENDIX 1: CATEGORIES FOR RISK PROFILES

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

1. Incidence

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

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

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