RISK PROFILE:
SHIGA-TOXIN PRODUCING *ESCHERICHIA COLI*
IN RAW MILK

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

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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 raw milk. Most raw milk consumption concerns cow (bovine) and goat (caprine) milk. The higher fat and protein content of milk from sheep (ovine) means that it is more likely to be consumed as cheese or yoghurt.

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 to approximately 90 per year. The rate of reported infection with STEC in New Zealand is similar to that in other developed countries, although higher than for Australia. The predominant serotype isolated from human cases in New Zealand is *E. coli* O157.

Information on risk factors from human infections indicates that approximately 10% report consumption of raw milk, but this is usually within a farm environment where other risk factors (e.g. animal contact) will occur frequently.

Limited data from testing of dairy and beef cattle in New Zealand suggest that faecal contamination by STEC is common (up to 27%) but *E. coli* O157 prevalence is lower. This indicates the potential for contamination of raw milk by STEC, although data derived from testing raw milk itself are lacking.

Growth of STEC should not occur if it is stored below 8°C, although survival for long periods will occur. Temperature abuse will allow rapid growth but there will be concomitant growth of other organisms which will inhibit STEC growth as well as limiting consumption due to spoilage.

Raw milk is not readily available from retail sources in New Zealand but farmers, their families, visitors and farm workers may consume it frequently.

There is some evidence for the occurrence of STEC in faeces of dairy and beef cattle, but whether this results in contamination of raw milk is uncertain.

Overseas outbreaks of STEC infection involving raw milk often involve small numbers of cases, probably due to the limited distribution of this type of dairy product. The same may be occurring in New Zealand, given the number of small clusters of STEC cases reported.

At present STEC infection via raw milk is a minor risk from the national perspective, given that most consumption will be occurring in the rural population. Such raw milk consumption may be more common than the limited data suggest, and will be augmented by those who
consume raw milk for perceived health and nutritional benefits. However, the risk for those consumers is difficult to assess, given the shortage of raw milk prevalence or animal carriage data. Given the serious nature of the illnesses caused by STEC infection, it would be worthwhile to further investigate this issue. Such investigations should take place within the context of a more general study of all sources of STEC infection in New Zealand.

The data gaps identified in this Risk Profile are:

- Consumption data for raw milk in New Zealand (largely due to current legal restrictions around the sale of raw milk);
- Prevalence of STEC carriage by dairy cattle in New Zealand (research on this topic is underway at ESR);
- Prevalence of STEC in raw milk in New Zealand (research in this topic is being developed by NZFSA); and
- Identification of the principal human infection pathways for STEC in New Zealand.
**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**

![Risk Management Framework Diagram](image)

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 risk characterisation part of a risk assessment 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) in raw milk. Most raw milk consumption in New Zealand will involve cow (bovine) milk as it is the most widely consumed type of milk. This Risk Profile also considers goat (caprine) and sheep (ovine) milk. The higher fat and protein content of ovine milk means that it is more likely to be consumed as cheese or yoghurt.

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 (1999a).

_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 consumption of the food group by New Zealanders
- data on the occurrence of the hazard in the New Zealand food supply
- 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 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

The following information is taken from a number of different sources, but unless otherwise referenced, the sources are data sheets originally prepared by ESR under a contract for the Ministry of Health. These are now located on the New Zealand Food Safety Authority website. 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 (i.e. O111:H-, O26:H11);


The ability of the serotypes in the latter group to cause disease varies greatly.

2.1 Nomenclature

The species *Escherichia coli* is a member of the large Enterobacteriaceae family. *E. coli* forms part of the normal microflora in the intestinal tracts of humans and other warm blooded animals. It is Gram-negative, facultatively anaerobic, forms short rods and is generally not pathogenic. Most of the pathogenic *E. coli* belong to specific groups; enteropathogenic *E. coli* (EPEC), enteroinvasive *E. coli* (EIEC), enterotoxigenic *E. coli* (ETEC), enterohaemorrhagic *E. coli* (EHEC), enteroaggregative *E. coli* (EAEC), diffusely adherent *E. coli* (DAEC), necrotoxigenic *E. coli* (NTEC) and *E. coli* producing cytolethal-distending toxin (CDT) (AIFST, 2003).

This Risk Profile is concerned with the group of *E. coli* which 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 (verocytoxigenic *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 VTEC and STEC 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 has been 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.

EHEC refers to those STEC that have the capability to cause haemorrhagic colitis (HC) and haemolytic uraemic syndrome (HUS). Through general usage, EHEC has been used to include particular serotypes of STEC, for example, *E. coli* O157:H7 or H-, O111:H- and O26:H11 (AIFST, 2003). 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, the term EHEC is often used incorrectly as a synonym of STEC and VTEC.

STEC will be the acronym used throughout this document.

Individual serotypes of STEC can be differentiated from one another serologically on the
basis of three fundamental antigens; somatic (O “ohne hauch”), flagellar (H= “hauch”) and capsular (K) antigens. 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”. Occasionally “rough” variants, lacking O specific polysaccharide chains, occur and are not able to be serotyped.

All STEC produce either or both of the Shiga-toxins, Stxl and Stx2 (previously known as Verotoxins). The characteristics of Stxl are generally conserved, but there are currently five recognised variants of Stx2. Stxl is both structurally and immunologically indistinguishable from Shiga-toxin and can be neutralised by anti-Shiga-toxin. Stx2 cannot be neutralised in this manner (AIFST, 2003). Although, by definition, all STEC must produce either or both of Stxl or Stx2, other factors are also 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 (the eeA gene encoding intimin), and the ability to produce haemolysin (the hly A gene encoding enterohaemolysin).

Phenotypic characteristics of both pathogenic and non-pathogenic E. coli are largely similar, although pathogenic strains have a more limited growth-temperature range and some EHEC may survive at lower pH levels. Ingested E. coli cells have to survive a pH of 3 or below in the stomach before infecting and colonising the intestine. Therefore tolerance of acidic conditions in pathogenic strains may be important in determining virulence (AIFST, 2003). Nine isolates of E. coli O157:H7 have been shown to reduce in numbers by an average of only 50% after 2 h exposure to simulated gastric fluid/saline at pH 1.5 (Tamplin 2005).

2.2 Escherichia coli O157

2.2.1 The organism/toxin

It is difficult to determine precisely which STEC strains have the potential to cause disease with the exception of a few specific serotypes such as E. coli O157, O111 or O26 where the complement of virulence factors is known. The most common serotypes of EHEC associated with human disease in New Zealand are E. coli O157:H7 and O157:H-.

2.2.2 Growth and Survival

Growth:

Temperature: E. coli O157:H7 is slightly more limited in its growth range than other E. coli, its minimum temperature for growth is 8°C, with a maximum of 44-45°C and an optimum of 37°C (ICMSF, 1996).

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 (stomach acid) are less inhibitory than organic acids (e.g. acetic, lactic) at the same pH. Growth was inhibited in the presence of 0.1% acetic acid (pH 5.1).

Atmosphere: As a facultative anaerobe, the bacterium can grow in the presence or absence of oxygen. High levels of carbon dioxide may be inhibitory to growth. For example, at 10°C, growth was not inhibited under 100% N2 or 20% CO2:80% N2 but was inhibited under 100%.
CO₂. In a study using lettuce and cucumber, numbers of *E. coli* O157:H7 increased rapidly under an atmosphere of 97% N₂:3% O₂ (Abdul-Raouf *et al*., 1993).

**Water activity:** Growth is retarded above 2.5% NaCl, but *E. coli* O157:H7 can grow slowly in broth containing up to 6.5% NaCl. Optimum growth is at *a*<sub>w</sub> = 0.995, minimum *a*<sub>w</sub> = 0.950 (AIFST, 2003).

**Survival:**

**Temperature:** Survives well in chilled and frozen foods. For example little change in numbers was noted in hamburgers stored at -20°C for 9 months (ICMSF, 1996).

**pH:** Can survive in low pH (down to 3.6) environments. 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°C on fermented sausage at 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 isolates of EHEC showed that a number of isolates could survive (i.e. were able to be recovered at levels up to 100% of the initial level) at a pH of 2.5 or 3.0 for a number of hours (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 inter-strain variation with respect to acid tolerance is a common feature of both non-pathogenic and O157 *E. coli* (Duncan *et al*., 2000).

**Atmosphere:** Survival of *E. coli* O157:H7 on shredded lettuce was not affected by packaging under modified atmospheres (Abdul-Raouf *et al*., 1993).

**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 (Pompepuy *et al*., 1996). Entry into the VNC state is suspected in high salt foods (Makino *et al*., 2000). However, the concept of the VNC state remains controversial.

### 2.2.3 Inactivation (Critical Control Points and Hurdles)

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. Pasteurisation has traditionally been the food safety hurdle applied to raw milk to achieve significant log reductions of STEC and other pathogens.

**Temperature:** *E. coli* are sensitive to heat, this sensitivity depending on the composition of the food, the pH and water activity. D time at 54.4°C = 40 minutes. D time at 60°C = 0.5–0.75 minute. D time at 64.3°C = 0.16 minute. In minced beef, the D time for *E. coli* O157:H7 at 58°C is 3.4 minutes (AIFST, 2003). The UK Food Standards Agency recommend cooking hamburger patties to 70°C for 2 minutes or equivalent (e.g. 75°C for 30 seconds) to eliminate
**E. coli.**

The most commonly used standards for pasteurising raw milk are the low temperature long time (LTLT) (63.5°C for 30 minutes) method (also known as the “holding method”), and the high temperature short time method (HTST) (71.7°C for 15 seconds). The most commonly used pasteurisation method for milk products in New Zealand is the HTST method. Extended shelf life and ultra heat treated products are pasteurised at 120-124°C and 134-135°C (or higher) respectively, for short periods. The “holding method” is occasionally used for batch pasteurisation of certain products. The efficacy of pasteurisation can be checked by phosphatase enzyme based assays.

A study of the destruction of *E. coli* O157 in raw milk at 63°C found that for a cocktail of isolates, D times were 4.3, 13.8 and 2.8 seconds (D'Aoust *et al.*, 1988).

**pH:** At low pH values the rate of death is dependent on the nature of the acid. For example, inactivation occurs at pH 4.5 in a medium adjusted with lactic acid but there is no inhibitory effect in a medium adjusted with hydrochloric acid (ICMSF, 1996).

**Water activity:** Withstands desiccation well.

**Preservatives:** 8.5% NaCl inhibits growth at 37°C. The amount of salt required for inhibition reduces as other factors such as temperature and pH become sub-optimal. For example 5% salt at 12°C inhibited three isolates of *E. coli* O157:H7, 6% salt at 10°C was inhibitory to 10 enteropathogenic isolates. Pathogenic *E. coli* are more tolerant to sodium chloride and sodium nitrite (this concentration of nitrite is above acceptable limits in food) (ICMSF, 1996).

**Radiation:** Sensitive to UV and γ irradiation. D (kGy) approx. 0.31 frozen, 0.24 refrigerated in ground beef.

**Disinfectants:** *E. coli* are generally susceptible to disinfectants used in the food industry.

### 2.2.4 Sources

**Human:** Faecal-oral person-to-person transmission has been reported in family members of cases.

**Animal:** Found in the guts of ruminant animals. Cattle are considered primary reservoirs but other ruminants, sheep, deer, buffalo and goats 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. Survival for up to four months in cattle manure has been reported (Duffy, 2003). Pigs can become infected where they are inoculated or exposed to ruminants shedding the pathogen. There have been reports of a UK outbreak of *E. coli* O157 involving 10 adults and 2 children who visited a wildlife park. The suggested vehicle for the infection was wild rabbit faeces [http://www.hse.gov.uk/lau/lacs/41-4.htm](http://www.hse.gov.uk/lau/lacs/41-4.htm).

Chickens are not a usual reservoir for *E. coli* O157. Although the organism can colonise birds, STEC has not been detected in poultry faecal studies in the Netherlands and the UK.
(Duffy, 2003). STEC has been isolated from wild birds, flies, horses, ponies, cats and dogs (AIFST, 2003). \textit{E. coli}\,O157 has been detected in 0.9-2.0\% gull droppings in the UK (Duffy, 2003).

Food: Food vehicles identified in overseas outbreaks have usually been those contaminated by cattle manure. Foods involved in outbreaks have included hamburgers, fermented sausages and other meat products, unpasteurised apple juice and cider, salads, bean sprouts, raw milk, cheese, melons, lettuce and flavoured yoghurt. For one case in New Zealand, an indistinguishable isolate was obtained from both the infected person and raw milk present in the home, although the route of infection is uncertain (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 at least 19 weeks in soil, dependent on type, temperature, microflora, moisture content, rainfall etc. (AIFST, 2003). It has also survived for at least 4 months in sediment in cattle drinking troughs.

Transmission Routes: The organism can persist in water, soil and pasture and become a source of infection for animals, birds and crops. Fruits and vegetables can be contaminated directly by faeces/manure fertiliser, dust from livestock areas, water and fruit flies (AIFST, 2003). In summary, any food or water source that has been contaminated by the faeces of a ruminant animal can be a vehicle for the infection.

2.3 Non-O157 Shiga-Toxin Producing \textit{Escherichia coli} (STEC)

2.3.1 The organism/toxin

These organisms form a diverse group of \textit{E. coli} serotypes that are capable of producing shiga-like toxin(s), as is \textit{E. coli}\,O157:H7. However, they are of widely differing pathogenic potential, varying from those that can cause illnesses similar to that produced by \textit{E. coli}\,O157:H7 to those that have never been associated with disease. Cases can be infected simultaneously with non-O157 strains as well as O157.

2.3.2 Growth and survival

The temperature range is slightly broader at optimum 37°C to 40°C, range circa 7-8 to 44-46°C. Doubling time approx. 0.4 hour at 37°C. Otherwise the behaviour of these organisms is largely the same as for serotype O157.

2.3.3 Inactivation (Critical Control Points 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). The laboratory difficulties associated with isolating non-
O157 strains means that the true prevalence, especially in sporadic cases, is unknown (AIFST, 2003).

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

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 although difficulties with isolating these serovars make true prevalence unknown.
3 HAZARD IDENTIFICATION: THE FOOD

3.1 Relevant Characteristics of the Food: Raw Milk

Milk is defined by Codex as “the normal mammary secretion of milking animals obtained from one or more milkings without either addition to it or extraction from it, intended for consumption as liquid milk or for further processing.” (Codex, 1999b). Raw milk has been defined as “milk which has not been heated beyond 40°C or undergone any treatment that has an equivalent effect” (Codex, 1999b; 2004).

Milk is intended to meet the demands of the suckling newborn through nourishment and to provide immunological protection. Whole cow’s milk is made up of water (87.3%), 4.2% fat, 4.6% lactose, 3.25% protein and 0.65% minerals, although these proportions are variable according to breed of animal, feed, age and phase of lactation (ICMSF, 1998).

Since milk has an almost neutral pH (6.7), a high water content and a variety of nutrients, it represents an ideal substrate for microbial growth. This is countered to some extent by natural inhibitory factors in raw milk. The main inhibitory factors in raw milk are the lactoperoxidase system (which produces hypot hiocyanate which inactivates enzymes and damages membranes) and lactoferrin (which binds iron) (Frank, 2001). Guidelines have been issued by Codex (1991) on the preservation of raw milk by activating the lactoperoxidase system, although this method should only be used when technical, economical or practical reasons do not allow the use of cooling facilities and where the product is not being exported from the country of origin.

There have been a number of infectious diseases linked to the consumption of unpasteurised milk. These include campylobacteriosis, salmonellosis, yersiniosis, listeriosis, tuberculosis, brucellosis, staphylococcal enterotoxin poisoning, streptococcal infections, and E. coli O157:H7 infection (Headrick et al., 1998).

3.2 STEC in Dairy Livestock

Raw milk can become contaminated by STEC via environmental sources or directly through infection of the udder and into the milk. In terms of environmental sources, as the organism is shed in animal faeces this can in turn contaminate the udder, teats, hide or hair, and subsequently contaminate the milk.

Mastitis is an inflammation of the mammary gland or udder. It may be caused in dairy animals by infection by a number of bacteria, most commonly Streptococci and Staphylococcus aureus species in New Zealand. Mastitis can also be caused by infection with E. coli and other coliforms. STEC may be a causative organism, but there is rarely further genotyping of E. coli identified in cases of coliform mastitis.

Shedding of bacteria directly into milk in coliform mastitis cases occurs mostly within the first 6 – 12 hours of clinical symptoms and it is usually a short lived infection. In Brazil, 182 isolates of E coli (obtained from 2144 milk samples from dairy cattle showing mastitis) were characterised genotypically. Genes characteristic of STEC were found in 22 of these isolates (12%) (Lira et al., 2004).
In the USA, faecal carriage of STEC by dairy animals is common, and cattle are considered to be a reservoir (Hussein and Sakuma 2005). Individual STEC PFGE types have been shown to persist on dairy farms for up to 17 months (Liebana et al., 2005) and the isolation of a single predominant type on Dutch dairy farms suggests horizontal transmission (Heuvelink et al., 1998a). Direct contact with infected animals has resulted in disease (Crump et al., 2002).

*E. coli* O157 is transient in the gastrointestinal tract of ruminants (Sanchez et al., 2002), often lasting only one to three months (Mechie et al., 1997).

In infected cattle faeces, the concentration of *E.coli* O157 can vary greatly (<100 to \( \geq 10^8 \)/g). The degree of shedding has been correlated with age, with calves generally excreting higher concentrations than adults. 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. Several factors affect the intensity of shedding, such as the type of feed, dietary stress and winter/summer months. It is recognised that poor homogeneity of the pathogen in faecal samples can also vary results (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).

*E. coli* from livestock faeces can survive on grass for at least 5-6 months and survive for longer in cattle faeces (Avery *et al.*, 2004). Survival of *E. coli* O157 was found to be greatest in soil cores containing rooted grass. Viable *E. coli* O157 numbers were shown to decline only slightly from approximately \( 10^8 \) per gram of soil to between \( 10^6 \) and \( 10^7 \) per gram of soil after 130 days (Maule, 2000). A similar timeframe was found in research carried out by Islam *et al.* (2004). The authors found that *E. coli* O157 could survive for more than 5 months in manured soil.

In relation to cattle water troughs, culturable *E. coli* O157 were used in simulated microcosms, survival in sediments was noted for at least 245 days. Strains that survived more than 6 months in the contaminated microcosms were infectious to a group of 10-week-old calves with faecal excretion lasting for 87 days after exposure. The authors concluded that water trough sediments contaminated with faeces from infected cattle may serve as a long-term reservoir and source of infection of *E. coli* O157 on farms (LeJeune *et al.*, 2001).

The survival of *E. coli* O157 was also investigated on stainless steel surfaces, (Maule, 2000) a popular material in milking sheds. In air-dried deposits, the pathogen survived more than 60 days. It was most stable at chill temperatures (4°C) and viability was only partially reduced at 18°C.

### 3.3 Survival and Growth of STEC in Raw Milk

Of seven STEC isolates inoculated into raw bovine milk and incubated at 8°C, three did not grow (Massa *et al.*, 1999). The growth of the remaining four was variable, and a maximum
number of around $10^7$ cfu/ml recorded. The fastest growth exhibited was an increase of approximately $2.5 \log_{10}$ units in 6 days following a one day lag period.

Growth of *E. coli* O157 has also been demonstrated in raw and UHT cow’s milk at 7 and 15°C (Heuvelink *et al.*, 1998b). At 7°C growth was modest (less than $1 \log_{10}$ in 150 hours) in raw milk, while survival only was observed in UHT milk. Growth at 15°C was significant, with increases of 3-4 $\log_{10}$ over this time period, in both types of milk. The naturally occurring lactoperoxidase-thiocyanate-hydrogen peroxide system did not apparently affect growth or survival, and activation of this system in raw milk had minimal effect.

At 5°C, the organism declined in numbers by approximately $1 \log_{10}$ after 28 days storage in unpasteurised milk (Wang *et al.*, 1997). At 8°C there was some growth followed by a decline then survival, with similar observations being made at 15 and 22°C. The point at which growth ceased corresponded with visible spoilage, and so the growth of the pathogen was probably inhibited by the presence of the spoilage organisms. The maximum number reached was around $10^6$ cfu/ml in unpasteurised milk and $10^8$/ml in pasteurised milk. The growth rate in unpasteurised milk was always slower than that measured in pasteurised milk, again probably because of the higher number of competing organisms and inhibitory factors in raw milk.

Clearly raw milk represents an excellent growth medium for STEC, should temperature abuse occur. In raw milk at refrigeration temperatures growth will be modest, although the organism survives well at these temperatures and will continue to present a hazard.

### 3.4 The Food Supply in New Zealand

#### 3.4.1 Production

Four dairy companies collect more than 99.5% of the bovine milk production in New Zealand. The four companies are Fonterra Co-operative Group Ltd, Westland Co-operative Dairy Company Limited, Open Country Cheese Ltd and Tatua Co-operative Dairy Company Limited (Diane Schumacher, NZFSA, Personal communication, April 2007). Fonterra is a leading multinational dairy company and is the world’s largest dairy exporter, exporting 95% of its production. The company is owned by 11,600 dairy farmers. The company collects over 13 billion litres of milk a year (Fonterra website, accessed 11th April 2007; http://www.fonterra.com/default.jsp).

The domestic milk market is a fraction of New Zealand’s total milk production, and is estimated at 386 million litres per annum (Diane Schumacher, NZFSA, Personal communication, April 2007). The consumption of raw milk will be a much a smaller fraction of this (discussed in section 5.2).

The historical and projected number of dairy cattle and production of liquid milk for New Zealand is shown in Table 1 (Source: http://www.maf.govt.nz/mafnet/rural-nz/statistics-and-forecasts/sonzaf/2006/04dairy.htm (MAF, 2006)).
Table 1: Number of dairy cattle and production of liquid milk for New Zealand, 2005-2010

<table>
<thead>
<tr>
<th>Year (to May)</th>
<th>2005</th>
<th>2006</th>
<th>2007</th>
<th>2008</th>
<th>2009</th>
<th>2010</th>
</tr>
</thead>
<tbody>
<tr>
<td>Livestock numbers (millions)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cows and heifers in calf or in milk</td>
<td>3.10</td>
<td>4.11</td>
<td>4.12</td>
<td>4.14</td>
<td>4.16</td>
<td>4.19</td>
</tr>
<tr>
<td>Total dairy cattle</td>
<td>5.15</td>
<td>5.07</td>
<td>5.40</td>
<td>5.41</td>
<td>5.43</td>
<td>5.48</td>
</tr>
<tr>
<td>Production (million tonnes)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* Figures for liquid milk production calculated from SONZAF website milk solid figures, using the conversion factor of 8.58%.

The Taranaki province has the highest number of dairy cattle with 507,000 milk or calf animals (MAF New Zealand, website accessed 11th April 2007; http://www.maf.govt.nz/mafnet/rural-nz/statistics-and-forecasts/farm-monitoring/2006/dairy/02overview.htm#Review_of_2005/06)

3.4.2 Imported foods

Raw milk is not imported into New Zealand. An import health standard was issued for the importation of raw milk from New Caledonia, however, this was for evaluation purposes only and the milk was specified for destruction following evaluation (http://www.maf.govt.nz/biosecurity/imports/animals/standards/index.htm).

3.3.3 Processing

The very nature of raw milk means that it undergoes very little processing. This therefore requires a high level of animal health care and hygienic facilities, premises and practices to avoid human illness.

The International Dairy Federation and Fonterra have argued that raw milk cannot be harvested without contamination and is inevitably contaminated with a range of bacteria, some of which may be human or animal pathogens (Pearce, 2001).

Conditions for the hygienic production of milk are set out in a Code of Hygienic Practice (Codex, 2004). This Code reflects the strong European desire to provide a prescriptive list of actions that if followed will provide assurance of raw milk safety. There are additional provisions for the production of raw milk products (the scope of the code does not extend to the production of raw drinking milk). The main areas for attention are those occurring prior to where pasteurisation (if used) would be applied. These include;

- The individual health of the lactating animal;
- Feeding practices;
- Pest control;
- Veterinary drugs;
- Hygienic milking;
• Health and personal hygiene of milking personnel;
• Design and cleansing of milking equipment and storage equipment;
• Collection, transport and delivery procedures, ensuring there is no mixing of milks or cross-contamination; and
• Transport times and temperatures, (the temperature shall not exceed 8°C unless the milk has been collected within 2 hours of milking).
4 HAZARD CHARACTERISATION: ADVERSE HEALTH EFFECTS

Infection with STEC may result in the organism invading the gut and then producing one or more toxins. Toxins are not produced in foods.

Infection may result in 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 uremic syndrome (HUS), thrombotic thrombocytopenic purpura (TTP) and death (AIFST, 2003). In a Dutch survey, 38% of cases evaluated were hospitalised and 15% developed HUS (van Duynhoven et al., 2002).

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 and death.

- **Thrombotic Thrombocytopenic purpura (TTP):** A version of HUS most often experienced by the elderly. Involves loss of platelets, skin colouration, 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 STEC infections (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

This section focuses on non-O157 serotypes because the virulence factors for *E. coli* O157 are well documented.
4.2.1 Non-O157 serotypes

Over 200 non-O157 STEC serotypes have been isolated from humans and are clearly recognised as human pathogens, although the difficulties with isolating non-O157 STEC mean that true prevalence is uncertain. The World Health Organisation has identified the most important non-O157 STEC serovars, from an epidemiologic perspective, as O26, O103, O111 and O145 (WHO, 1998). The website [http://www.who.int/emodocuments/zoonoses/docs/whoecsraph988.html/3surveillanceandfrequency.html](http://www.who.int/emodocuments/zoonoses/docs/whoecsraph988.html/3surveillanceandfrequency.html) includes a table of serotypes of non-O157 STEC isolated from humans. Serotypes isolated from patients with HUS are highlighted.

An updated literature list of STEC, (non-O157) with 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/intro.htm](http://www.microbionet.com.au/frames/feature/vtec/intro.htm). The serotypes O157:H7 and O157:H- references are deliberately not listed because of the numerous references to them in the literature.

4.2.2 Overview of international situation

In the USA, it has long been held that serotype O157 is the predominant cause of STEC related disease. 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 and through the loss of prophage carrying the toxin genes). 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 approximately 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 equivocal. 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 described 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 reported 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). Serotypes more commonly found in Australia are (AIFST, 2003);

- O157:H-,
- O6:H31
- O26:H- and H11
- O91:H10
- O98:H-
- O111:H- and H8
- O113:H21
- O146:H8

Type O111:H- is the most prevalent (Park *et al.*, 1999).

### 4.3 Dose-Response

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

In the past, definitive numbers of cells have been expressed as an infectious dose, this concept has been largely superceded by calculating the probability of infection by exposure to differing numbers of cells.

In terms of probability of infection, Haas *et al.* (2000) have 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 waterborne organisms and the other involving venison jerky. The model gave a dose for infection of 50% of the exposed population of $5.9 \times 10^5$ organisms and a risk for consumption of 100 organisms of $2.6 \times 10^{-4}$. 

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

An analysis of data from an elementary school outbreak of infection with *E. coli* O157:H7 in Japan (Teunis *et al.*, 2004) indicates much higher probabilities of infection at lower doses than previous models.

In terms of the older style infectious dose data, these studies were based on 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 was 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 $2.6 \times 10^6$ organisms and a risk for consumption of 100 organisms of $3.5 \times 10^{-4}$. 

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Risk Profile – STEC in raw milk

July 2007
5 EXPOSURE ASSESSMENT

5.1 The Hazard in the New Zealand Food Supply: STEC in Raw Milk

5.1.1 STEC carriage by livestock in New Zealand

Two published surveys have evaluated the prevalence of STEC in New Zealand ruminants. Buncic and Avery (1997) sampled the faeces of 371 dairy cattle from 55 farms on arrival at a single slaughterhouse in the Waikato area and tested for *E. coli* O157. Two (0.54%) of these samples yielded positive results. A further 160 dairy 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.

Of the 510 cattle and 442 sheep *E. coli* isolates, 69 (13.5%) and 170 (38.5%) respectively were STEC i.e. possessing one or both stx genes.

STEC were detected in 51 of the cattle faecal samples (27%). The eaeA gene was detected in 36.5% of the positive samples. In sheep faeces, STEC were detected from 65.9% of samples. The eaeA gene was isolated from 27.3% of the positives. Overall, 23 isolates were Stx1/2 and eaeA positive and all contained the enterohemolysin (ehxA) gene. Although *E. coli* O157:H7 was not detected in any sample, several clinically relevant serotype isolates were detected, including O5:H-. O26:H11, O84:H-/H2, O91:H- and O128:H2.

Clearly New Zealand dairy cows have the potential to harbour clinically relevant STEC. Although *E. coli* O157 was not specifically sought in the Cookson *et al.* study (2006), the existing data suggest that *E. coli* O157 itself is not widespread.

There is currently a study underway at ESR regarding the prevalence of STEC in faeces of dairy cattle in New Zealand.

5.1.2 Prevalence of STEC in raw milk in New Zealand

There are no published data available for STEC in raw milk in New Zealand. A survey has been carried out on raw whole milk samples for *Campylobacter jejuni*, *Listeria monocytogenes* and *Yersinia enterocolitica* (Stone, 1987) but STEC were not included. This represents a significant data gap.

A quantitative survey of raw milk is currently being conducted by Fonterra. STEC O157:H7 is included as one of the pathogens under surveillance. Most Probable Number (MPN) and Polymerase Chain Reaction (PCR) methods are being used.
5.2 Food Consumption: Milk

5.2.1 Raw milk consumption

Data on raw milk consumption in New Zealand are very limited. No record of raw milk consumption is included in the results of the 1997 National Nutrition Survey (Russell et al., 1999).

Wickens et al. (2002) surveyed 293 children in the age range 7-10 for risk factors associated with allergic diseases. In response to questions as to whether they had ever consumed unpasteurised milk during the first two years of life (answers provided by parents), 23% of children currently living on farms responded positively, while only 8% of non-farm children responded positively.

In a review of Campylobacter transmission routes (Lake, 2005), it was calculated that potentially 170,000 – 250,000 New Zealanders could be consuming raw milk. The lower estimate was based on the assumption that each of 17,000 dairy farms had the potential to supply raw milk to up to 10 family members and friends.

The higher estimate was derived from epidemiological studies of campylobacteriosis. Consumption of unpasteurised milk was reported by 9 of 44 cases (20%) interviewed for the transmission routes study amongst a predominantly rural population in Ashburton (Baker et al., 2002). Consumption of unpasteurised milk was reported by 5.8% of cases and 2.4% of controls in the primarily urban case control study (Eberhart-Philips et al., 1997). If 14.3% of New Zealand’s population is classed as rural (573430 of estimated 2003 population of 4.01 million), and 20% consume unpasteurised milk then the exposed population could be 115,000. Adding an average of the number of urban cases and controls reporting raw milk consumption (approximately 4%) of the urban population of 3504740 gives an additional 140,000 people, providing an exposed population of perhaps 255,000.

5.2.2 Total milk consumption

To give some idea of raw milk consumption, total milk consumption is discussed in this section along with serving sizes.

The per capita consumption of milk in New Zealand increased from 1942, when subsidies were first placed on milk, to a peak in 1973. Subsidies were removed in 1985 and per capita consumption has been decreasing steadily since 1976 (Wham and Worsley, 2001). It has been estimated that the New Zealand liquid milk market is approximately 350 million litres per annum (New Zealand Dairy Board, 2000), which equates to 96.7 litres/person per annum for the New Zealand population or 265 g/person/day. This represents the per capita amount of milk available for consumption.

The following information is taken from the New Zealand National Nutrition Survey (NNS) conducted in 1997 (Russell et al., 1999).

Summary food consumption statistics can be expressed in terms of ‘consumers’ (just those people reporting to eat a particular food) or ‘persons’ (the whole population). Both will be presented here. The age groups used by the Australian National Nutrition Survey (1995) will
initially be used so that an easy point of comparison can be made. These are 16-18 years, 19-24 years, 25-44 years, 45-64 years, 65 years and over. Milk was assumed to be analogous to the Australian NNS category of ‘Dairy milk’. Table 2 shows the percentages of respondents in the 1997 National Nutrition Survey who reported consuming milk in the previous 24 hour period. This demonstrates a general trend towards higher frequency of consumption amongst the older population.

Table 2: Milk – percentage of respondents consuming in 24 hour period

<table>
<thead>
<tr>
<th>Age (years)</th>
<th>16-18</th>
<th>19-24</th>
<th>25-44</th>
<th>45-64</th>
<th>65+</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>80.7</td>
<td>76.6</td>
<td>86.6</td>
<td>88.6</td>
<td>91.1</td>
<td>86.9</td>
</tr>
<tr>
<td>Female</td>
<td>74.5</td>
<td>82.3</td>
<td>87.4</td>
<td>84.4</td>
<td>88.4</td>
<td>85.8</td>
</tr>
<tr>
<td>Total</td>
<td>77.2</td>
<td>79.9</td>
<td>87.1</td>
<td>86.4</td>
<td>89.5</td>
<td>86.2</td>
</tr>
</tbody>
</table>

These figures are similar to those observed in the Australian NNS (Australian Bureau of Statistics, 1999) which reported 63.4-90.2% of respondents consuming milk, depending on the age group. The pattern of respondents with respect to age is similar to that seen in Australia, with increasing proportions of people consuming milk with increasing age. For Australians 19 years and over 82.2% of males and 84.3% of females reported consuming dairy milk.

Table 3 gives the median daily consumption level of milk, for consumers only. These figures indicate that, while a greater proportion of the older population report consumption of milk, they generally report consumption of smaller quantities of milk.

Table 3: Milk – median (50\textsuperscript{th} percentile) consumption by NNS respondents who consume milk (g/day)

<table>
<thead>
<tr>
<th>Age (years)</th>
<th>16-18</th>
<th>19-24</th>
<th>25-44</th>
<th>45-64</th>
<th>65+</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>250</td>
<td>250</td>
<td>211</td>
<td>180</td>
<td>180</td>
<td>200</td>
</tr>
<tr>
<td>Female</td>
<td>194</td>
<td>194</td>
<td>180</td>
<td>175</td>
<td>180</td>
<td>180</td>
</tr>
<tr>
<td>Total</td>
<td>228</td>
<td>202</td>
<td>200</td>
<td>180</td>
<td>180</td>
<td>187</td>
</tr>
</tbody>
</table>

The median amounts of milk drunk by consumers are very similar to those reported for the Australian NNS (Australian Bureau of Statistics, 1999). The Australian study reported an overall median (males and females) for respondents aged 19 and over of 187 g/day, the same as that derived from the New Zealand NNS (1997). Overall figures for males and females are also very comparable (males; Australia 206 g/day, New Zealand 200 g/day, females; Australia 165 g/day, New Zealand 180 g/day).

Median daily consumption may represent one or more servings of milk. The USA risk assessment for Listeria monocytogenes (FDA/FSIS, 2003) determined median serving sizes (which may be equal to, or less than median daily intake) for pasteurised or unpasteurised milk as being 244 g/serving, with 75\textsuperscript{th}, 95\textsuperscript{th} and 99\textsuperscript{th} percentile serving sizes of 245, 488 and 732 g/serving respectively. Unpasteurised milk consumption was estimated to be 0.5% of total milk consumption.
In England and Wales, the Dairy Hygiene Inspectorate have estimated that 0.01% of cows’ milk is consumed raw (Food Standards Agency, 2005).

Analysis of the distribution of individual servings of milk reported in the 1997 NNS gives values of 40, 70, 250, 450 g/serving for the 50th, 75th, 95th, 99th percentile serving sizes. The large discrepancy between the USA situation and the New Zealand situation is likely to be that the 1997 NNS included milk added to tea or coffee as a separate serving of milk, whereas it is likely that the USA situation only represents milk consumed as a beverage on its own.

Table 4 shows 95th percentile levels of milk consumption by New Zealand consumers. This level of consumption is considered to be an appropriate indication of a high volume consumer for a particular food.

<table>
<thead>
<tr>
<th>Age (years)</th>
<th>16-18</th>
<th>19-24</th>
<th>25-44</th>
<th>45-64</th>
<th>65+</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>978</td>
<td>942</td>
<td>766</td>
<td>658</td>
<td>540</td>
<td>722</td>
</tr>
<tr>
<td>Female</td>
<td>615</td>
<td>812</td>
<td>679</td>
<td>609</td>
<td>502</td>
<td>618</td>
</tr>
<tr>
<td>Total</td>
<td>857</td>
<td>929</td>
<td>700</td>
<td>650</td>
<td>538</td>
<td>679</td>
</tr>
</tbody>
</table>

Table 5 shows overall mean daily intake values for milk for all respondents who participated in the 1997 National Nutrition Survey.

<table>
<thead>
<tr>
<th>Age (years)</th>
<th>16-18</th>
<th>19-24</th>
<th>25-44</th>
<th>45-64</th>
<th>65+</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>288</td>
<td>253</td>
<td>237</td>
<td>214</td>
<td>192</td>
<td>226</td>
</tr>
<tr>
<td>Female</td>
<td>186</td>
<td>231</td>
<td>212</td>
<td>193</td>
<td>196</td>
<td>205</td>
</tr>
<tr>
<td>Total</td>
<td>231</td>
<td>240</td>
<td>222</td>
<td>203</td>
<td>194</td>
<td>214</td>
</tr>
</tbody>
</table>

The mean amounts of milk drunk by persons in New Zealand (total population, not just consumers) are very similar to those reported for the Australian NNS (Australian Bureau of Statistics, 1999). The Australian study reported an overall mean (males and females) for respondents aged 19 and over of 204 g/day, compared to 214 g/day from the New Zealand NNS (1997). Overall figures for males and females are also very comparable (males; Australia 223g/day, New Zealand 226g/day, females; Australia 184 g/day, New Zealand 205 g/day).

These figures are also consistent with the simulated typical diets formulated for the 1997/98 New Zealand Total Diet Survey (Vannoort et al., 2000), which used an average daily intake of milk for adult males of 235 g/day, and for adult females of 173 g/day. These figures are also consistent with the estimated average amount of milk available for consumption in New Zealand (265 g/person/day).
It is slightly surprising that the average consumption of milk for all New Zealanders is greater than the median consumption for consumers only. This reflects the high percentage of the population who consume milk and the fact that high consumers of milk are consuming very large amounts indeed (refer Table 3).

5.3 Qualitative Estimate of Exposure

5.3.1 Number of servings of raw milk and serving size

As discussed earlier, there are insufficient data to estimate the prevalence of raw milk consumption in New Zealand. Serving size may be extrapolated from the serving sizes of pasteurised milk discussed in section 5.2.2.

5.3.2 Contamination frequency

There are no recent data that would allow the assignment of an overall frequency of contamination to raw milk in New Zealand. Data from overseas would suggest that contamination is a rare event.

5.3.3 Predicted contamination level at retail

No New Zealand data are available on levels of STEC in raw milk which is available for sale.

5.3.4 Growth rate during storage and most likely storage time

If maintained at <5°C there should be no growth in raw milk.

In the USA (FDA/FSIS, 2003) quantitative risk assessment for \( L. \) monocytogenes, shorter consumer storage times were assumed for unpasteurised milk compared to pasteurised milk. This was due to the presence of more extensive spoilage microflora. The times used in the analysis were 0.5 to 10 days, with a most likely time being 2 to 3 days, compared with pasteurised milk; 0.5 to 15 days, most likely 3 to 5 days.

5.3.5 Heat treatment

Not applicable to raw milk. By definition, raw milk has not been heated to more than 40°C.

5.3.6 Exposure summary

Limited data from testing of cattle in New Zealand suggest that faecal contamination by STEC is common (up to 27%) but \( E. \) coli O157 prevalence is lower. This indicates the potential for contamination of raw milk by STEC, although data derived from raw milk itself are lacking.

Growth of STEC should not occur if milk is stored below 8°C, although survival for long periods will occur. Temperature abuse will allow rapid growth but there will be concomitant growth of other organisms which will inhibit STEC growth as well as limiting consumption due to spoilage.
Raw milk is not readily available from retail sources in New Zealand but farmers, their families, visitors and farm workers may consume it frequently.

5.4 Overseas Context

5.4.1 Prevalence of *E. coli* O157:H7 in cattle, goat and sheep faeces overseas

Tables 6 and 7 show the prevalence of STEC in cattle, goat and sheep faeces overseas. The tables have been collated from Blanco *et al.* (2001) and EFSA (2005). Prevalence is generally below 10%.

**Table 6:** Prevalence of *E. coli* O157:H7 in cattle faeces/rectal swabs

<table>
<thead>
<tr>
<th>Country</th>
<th>Sampling year</th>
<th>No. of faecal samples positive/N</th>
<th>% Positive</th>
<th>Type of cattle</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Europe</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Austria</td>
<td>2004</td>
<td>7/287</td>
<td>2.4</td>
<td>STEC*</td>
<td>At slaughter (EFSA, 2005)</td>
</tr>
<tr>
<td>Belgium</td>
<td>2004</td>
<td>2/59</td>
<td>3.4</td>
<td>STEC*</td>
<td>“Animals” (EFSA, 2005)</td>
</tr>
<tr>
<td>Denmark</td>
<td>2004</td>
<td>21/251</td>
<td>8.4</td>
<td>STEC*</td>
<td>Beef herd (EFSA, 2005)</td>
</tr>
<tr>
<td>Finland</td>
<td>2004</td>
<td>20/1603</td>
<td>1.2</td>
<td>STEC*</td>
<td>At slaughter Dairy herd Beef herd (EFSA, 2005)</td>
</tr>
<tr>
<td>Germany</td>
<td>2004</td>
<td>37/273</td>
<td>13.6</td>
<td>24.1</td>
<td>“Animals” Dairy herd Calves</td>
</tr>
<tr>
<td></td>
<td></td>
<td>7/29</td>
<td></td>
<td>STEC*</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>0/97</td>
<td>0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Italy</td>
<td>2004</td>
<td>0/154</td>
<td>0</td>
<td></td>
<td>“Survey animals” Calves</td>
</tr>
<tr>
<td></td>
<td></td>
<td>3/308 (all non-O157)</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Netherlands</td>
<td>1996</td>
<td>75/1152</td>
<td>7</td>
<td></td>
<td>Dairy cattle on farms</td>
</tr>
<tr>
<td></td>
<td>2004</td>
<td>13/153</td>
<td>8.5</td>
<td>13.5*</td>
<td>Dairy cattle Veal calves</td>
</tr>
<tr>
<td></td>
<td></td>
<td>23/171</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Norway</td>
<td>1995</td>
<td>6/1970</td>
<td>0.3</td>
<td></td>
<td>Heifers and milking cows</td>
</tr>
<tr>
<td>Portugal</td>
<td>2004</td>
<td>0/241</td>
<td>0</td>
<td></td>
<td>“Animals” (EFSA, 2005)</td>
</tr>
<tr>
<td>Slovakia</td>
<td>2004</td>
<td>0/100</td>
<td>0</td>
<td></td>
<td>Calves (EFSA, 2005)</td>
</tr>
<tr>
<td>Spain</td>
<td>1993-1994</td>
<td>1/686</td>
<td>0.1</td>
<td></td>
<td>Beef and dairy cattle on</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(Blanco <em>et al.</em>, 1996, 1997)</td>
</tr>
</tbody>
</table>

Risk Profile – STEC in raw milk 26 July 2007
<table>
<thead>
<tr>
<th>Country</th>
<th>Sampling year</th>
<th>No. of faecal samples positive/N</th>
<th>% Positive</th>
<th>Type of cattle</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>North America</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>farms</td>
</tr>
<tr>
<td>Canada</td>
<td>1992/93</td>
<td>12/1478</td>
<td>0.8</td>
<td>Dairy cattle</td>
<td>(Wilson et al., 1996)</td>
</tr>
<tr>
<td>Canada</td>
<td>1993</td>
<td>2/406</td>
<td>0.5</td>
<td>Dairy cattle</td>
<td>(Rahn et al., 1997)</td>
</tr>
<tr>
<td>USA</td>
<td>-</td>
<td>18/1266</td>
<td>1</td>
<td>Dairy cattle</td>
<td>(Wells et al., 1991)</td>
</tr>
<tr>
<td>USA</td>
<td>1991/92</td>
<td>22/5582</td>
<td>0.4</td>
<td>Dairy and beef cattle</td>
<td>(Hancock et al., 1994)</td>
</tr>
<tr>
<td>USA</td>
<td>-</td>
<td>24/351</td>
<td>7</td>
<td>Dairy cattle</td>
<td>(Sanderson et al., 1995)</td>
</tr>
<tr>
<td>USA</td>
<td>1991/92</td>
<td>25/6894</td>
<td>0.4</td>
<td>Dairy heifers</td>
<td>(Garber et al., 1995)</td>
</tr>
<tr>
<td>USA</td>
<td>1993</td>
<td>31/965</td>
<td>3</td>
<td>Dairy cattle</td>
<td>(Zhao et al., 1995)</td>
</tr>
<tr>
<td>USA</td>
<td>1996</td>
<td>52/4361</td>
<td>1</td>
<td>Dairy cows</td>
<td>(Garber et al., 1999)</td>
</tr>
<tr>
<td>USA</td>
<td>May 2000-April 2001</td>
<td>8/415 (seasonal difference noted)</td>
<td>1.9</td>
<td>Dairy cull cows</td>
<td>(Murinda et al., 2002)</td>
</tr>
<tr>
<td>Australasia</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Australia</td>
<td>-</td>
<td>11/588</td>
<td>2</td>
<td>Dairy cattle</td>
<td>(Cobbold and Desmarchelier, 2000)</td>
</tr>
<tr>
<td>Asia</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Thailand</td>
<td>1998</td>
<td>1/55</td>
<td>2</td>
<td>Dairy and beef cattle</td>
<td>(Vuddhakul et al., 2000)</td>
</tr>
</tbody>
</table>

* paper does not give further information on serotype # 2 of the serotypes were non-O157.

Several papers have observed greater faecal shedding of STEC during Spring and Summer months.
Table 7: Prevalence of STEC in goat and sheep faeces/rectal swabs

<table>
<thead>
<tr>
<th>Country</th>
<th>Sampling year</th>
<th>No. of faecal samples positive/N</th>
<th>% Positive</th>
<th>Type of animal</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Austria</td>
<td>2004</td>
<td>1/89</td>
<td>1.1*</td>
<td>Goats and sheep</td>
<td>(EFSA, 2005)</td>
</tr>
<tr>
<td>Greece</td>
<td>2004</td>
<td>0/74</td>
<td>0</td>
<td>Goats and sheep</td>
<td>(EFSA, 2005)</td>
</tr>
<tr>
<td>Italy</td>
<td>2004</td>
<td>3/32</td>
<td>9.4#</td>
<td>Goat</td>
<td>(EFSA, 2005)</td>
</tr>
<tr>
<td></td>
<td>2004</td>
<td>0/585</td>
<td>0</td>
<td>Goats and sheep</td>
<td>(EFSA, 2005)</td>
</tr>
<tr>
<td>Portugal</td>
<td>2004</td>
<td>2/158</td>
<td>1.3</td>
<td>Goats and sheep</td>
<td>(EFSA, 2005)</td>
</tr>
</tbody>
</table>

* no O157 detected.  # all serotypes O157

5.4.2 Prevalence of STEC in raw milk overseas

In order to differentiate between the raw milk intended for direct consumption and the raw milk intended for processing (such as pasteurisation), the information is given where known.

Information from the scientific literature on the prevalence of STEC in raw milk overseas has been summarised in Table 8. Quantitative data are scant. One report concerning goat’s milk has recorded a count of 1.5 \(E. coli\)/ml in one sample, but it is not clear whether the count was for generic \(E. coli\) or the \(E. coli\) O157:H7 detected.
Table 8: Overseas Prevalence Data for STEC in Raw Milk

<table>
<thead>
<tr>
<th>Country/Region</th>
<th>Milk Type</th>
<th>Period of sampling</th>
<th>No. samples tested</th>
<th>No. (%) positive for STEC</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Australia</td>
<td>Raw milk</td>
<td>N/a</td>
<td>147*</td>
<td>0</td>
<td>(Desmarchelier et al., 1998) cited in Duffy et al., 2001</td>
</tr>
<tr>
<td>Canada</td>
<td>Milk filters from farms, filters in milk lines between milking machines and bulk tanks. Milk appears to be destined for processing because of bulk collection.</td>
<td>Dec. 1985 – March 1986</td>
<td>1,012</td>
<td>3 (0.3)¹</td>
<td>(Clarke et al., 1989)</td>
</tr>
<tr>
<td>Canada</td>
<td>Bulk farm tank bovine milk. Raw milk prohibited for sale in Canada, assumption that milk intended for pasteurisation.</td>
<td>October 1995 – May 1996</td>
<td>1,720 samples from pick ups</td>
<td>15 (0.9)²</td>
<td>(Steele et al., 1997)</td>
</tr>
<tr>
<td>Egypt</td>
<td>Unpasteurised bovine milk, unclear from paper whether milk originated from supermarket or farmer’s home</td>
<td>N/a</td>
<td>50¹</td>
<td>3 (6.0)</td>
<td>(Abdul-Raouf et al., 1996)</td>
</tr>
<tr>
<td>Egypt</td>
<td>Raw milk (species not given) collected from markets</td>
<td>N/a</td>
<td>20¹</td>
<td>0 O157</td>
<td>(Aman et al., 1998)</td>
</tr>
<tr>
<td>England</td>
<td>Milk samples from 10 faecal positive cows from a herd implicated in an outbreak</td>
<td>N/a</td>
<td>10*</td>
<td>1 (10.0)</td>
<td>(Chapman et al., 1993)</td>
</tr>
<tr>
<td>England and Wales</td>
<td>Raw milk from 242 retail outlets</td>
<td>May 1996 – July 1997</td>
<td>1,097</td>
<td>3 (0.3) all from different retail outlets, and had satisfactory total bacterial counts. 27 samples contained &gt;100 E. coli organisms /ml.</td>
<td>(de Louvais and Rampling, 1998)</td>
</tr>
<tr>
<td>Country/Region</td>
<td>Milk Type</td>
<td>Period of sampling</td>
<td>No. samples tested</td>
<td>No. (%) positive for STEC</td>
<td>Reference</td>
</tr>
<tr>
<td>-----------------</td>
<td>---------------------------------------------------------------------------</td>
<td>--------------------------</td>
<td>--------------------</td>
<td>--------------------------</td>
<td>------------------------------------------</td>
</tr>
<tr>
<td>England and Wales</td>
<td>Raw caprine milk (liquid and frozen) from unregistered producers; from farm outlets and other retail premises.</td>
<td>Jan – March 1998</td>
<td>100</td>
<td>0 (O157:H7)</td>
<td>(Little and de Louvais, 1999)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>18 <em>E. coli</em></td>
<td></td>
</tr>
<tr>
<td>France</td>
<td>Raw milk (species not given) collected from dairy plant prior to unpasteurised cheese making</td>
<td>N/a</td>
<td>205</td>
<td>44 (21.5)*</td>
<td>(Fach et al., 2001)</td>
</tr>
<tr>
<td>Germany</td>
<td>Raw bovine milk</td>
<td>2004</td>
<td>205</td>
<td>(2.4%)</td>
<td>(EFSA, 2005)</td>
</tr>
<tr>
<td>Germany</td>
<td>Raw milk</td>
<td>N/a</td>
<td>273*</td>
<td>1 (0.4)</td>
<td>(Klie et al., 1997) cited in Duffy et al., 2001</td>
</tr>
<tr>
<td>Germany</td>
<td>Raw milk</td>
<td>N/a</td>
<td>245*</td>
<td>0</td>
<td>(Kuntze et al., 1996) cited in Duffy et al., 2001</td>
</tr>
<tr>
<td>Germany</td>
<td>Raw milk</td>
<td>N/a</td>
<td>180</td>
<td>22 (12.2) Non O157</td>
<td>(Gallien et al., 1998) cited in Duffy et al., 2001</td>
</tr>
<tr>
<td>Italy</td>
<td>Bulk farm storage tank – bovine milk</td>
<td>February – November 1996</td>
<td>100*</td>
<td>0</td>
<td>(Massa et al., 1999)</td>
</tr>
<tr>
<td>Italy</td>
<td>Raw goat milk intended for cheese making</td>
<td>April – September</td>
<td>60*</td>
<td>1 (1.7)</td>
<td>(Foschino et al., 2002)</td>
</tr>
<tr>
<td>Italy</td>
<td>Raw milk</td>
<td>N/a</td>
<td>227</td>
<td>0</td>
<td>(Colombo et al., 1998) cited in Duffy et al., 2001</td>
</tr>
<tr>
<td>Italy</td>
<td>Raw sheep milk</td>
<td>N/a</td>
<td>314*</td>
<td>2 (0.6)</td>
<td>(Rubini et al., 1999)</td>
</tr>
<tr>
<td>Netherlands</td>
<td>Bulk farm storage tanks - bovine milk, not clear from paper what intended use of milk was</td>
<td>May – Nov. 1997</td>
<td>1,011* from 1,011 different herds</td>
<td>0</td>
<td>(Heuvelink et al. 1998b)</td>
</tr>
<tr>
<td>Country/Region</td>
<td>Milk Type</td>
<td>Period of sampling</td>
<td>No. samples tested</td>
<td>No. (%) positive for STEC</td>
<td>Reference</td>
</tr>
<tr>
<td>-----------------</td>
<td>---------------------------------------------------------------------------</td>
<td>--------------------------</td>
<td>--------------------</td>
<td>---------------------------</td>
<td>------------</td>
</tr>
<tr>
<td>Northern Ireland</td>
<td>Raw bovine milk from 2 dairies intended to be pasteurised. Samples collected from milk and environment. The raw milk tanker, silos, balance tank, clarifier and separator desludge unit, drains and floor</td>
<td>July 1999- July 2000</td>
<td>420, not clear from paper how many samples obtained from raw milk itself</td>
<td>9 (2.1)</td>
<td>McKee et al. 2003</td>
</tr>
<tr>
<td>Slovakia</td>
<td>Raw bovine milk,</td>
<td>2004</td>
<td>83</td>
<td>0</td>
<td>EFSA, 2005</td>
</tr>
<tr>
<td>Spain</td>
<td>Bulk tank</td>
<td>March 2003 – June 2004</td>
<td>360 sample 287 ovine 73 caprine</td>
<td>PCR 39 29 (10.1) Micro 4 9 5</td>
<td>Burnens et al., 1995 cited in Duffy et al., 2001</td>
</tr>
<tr>
<td>Switzerland</td>
<td>Raw milk</td>
<td>N/a</td>
<td>93</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>United Kingdom</td>
<td>Raw milk (ex farm)</td>
<td>N/a</td>
<td>151*</td>
<td>0</td>
<td>Neaves et al., 1994</td>
</tr>
<tr>
<td></td>
<td>Raw milk (ex tanker)</td>
<td>N/a</td>
<td>53*</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td></td>
<td>intended use of the milk unclear</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Country/Region</td>
<td>Milk Type</td>
<td>Period of sampling</td>
<td>No. samples tested</td>
<td>No. (%) positive for STEC</td>
<td>Reference</td>
</tr>
<tr>
<td>---------------</td>
<td>-----------</td>
<td>-----------------------------</td>
<td>--------------------</td>
<td>---------------------------</td>
<td>-----------</td>
</tr>
<tr>
<td>United Kingdom</td>
<td>Raw milk at dairy, prior to pasteurisation</td>
<td>March 1999-March 2000</td>
<td>610</td>
<td>1 O157 (0.2%) 316 E. coli (52%)</td>
<td>(Food Standards Agency, 2003)</td>
</tr>
<tr>
<td>Turkey</td>
<td>Raw milk, collected randomly from 10 villages</td>
<td>N/a</td>
<td>100</td>
<td>1 (1.0)</td>
<td>(Öksüz et al., 2004)</td>
</tr>
<tr>
<td>USA</td>
<td>Raw milk</td>
<td>N/a</td>
<td>115*</td>
<td>11 (9.6)</td>
<td>(Padhye and Doyle, 1991)</td>
</tr>
<tr>
<td>USA</td>
<td>Raw milk</td>
<td>N/a</td>
<td>603*</td>
<td>0</td>
<td>(Hancock et al., 1994)</td>
</tr>
<tr>
<td>USA</td>
<td>Raw milk samples from 2 farms (outbreak investigation)</td>
<td>N/a</td>
<td>23*</td>
<td>1 (4.3)</td>
<td>(Wells et al. 1991)</td>
</tr>
<tr>
<td>USA</td>
<td>Raw milk from 15 dairy plants prior to pasteurisation</td>
<td>N/a</td>
<td>42*</td>
<td>0</td>
<td>(Ansay and Kaspar, 1997)</td>
</tr>
<tr>
<td>USA</td>
<td>Bulk tank bovine milk from 30 dairy farms, East Tennessee</td>
<td>May 2000 – April 2001</td>
<td>268*</td>
<td>2 (0.8)</td>
<td>(Murinda et al. 2002)</td>
</tr>
<tr>
<td>USA</td>
<td>Bulk farm tank, raw bovine milk</td>
<td>N/a</td>
<td>131 (different herds)</td>
<td>5 (3.8) 4 encoded for type 2 gene, the remaining isolate encoded for type 1 gene. O157:H7 was not isolated</td>
<td>(Jayarao and Henning, 2001)</td>
</tr>
</tbody>
</table>

* Tested for *Escherichia coli* O157 only.
N/a Not available
6 No typing data provided.
1 Seven isolates were obtained; O26:H11 (group 1), O43:H2 (ungrouped), O153:H25 (group 1), ONT:H8, ONT:H19, ONT:H-, ONT:rough.
3 Isolates were only obtained from 11 samples. The isolates belonged to; O3 (2), O6, O117, O76/O22 (2), O77, O110, O91 and 2 were non-typable.
4 Groups refer to the groups according to relevance to human disease (Gyles et al., 1998). Group 1 are STEC commonly associated with disease in humans, group 2 are STEC less commonly associated with disease in humans and group 3 are not reported to be associated with disease in humans. Groups cannot be assigned with incomplete typing data.
4 Nine isolates in total, from ovine milk, 2 of the isolates belonged to ONT:H7 and one each belonged to ONT:H9 and O45:H38. From caprine milk, one isolate each belonged to O157:H7, O27:H18, O91:H28, O76:H19, ONT:H21.

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**Risk Profile – STEC in raw milk**
These data show a low prevalence of STEC in raw milk with a few exceptions. Some caution must be taken with Fach et al., 2001 as the results were for PCR detection; a lower rate for isolation of STEC from these samples was recorded. Organisms other than STEC may possess genes detected by PCR, depending on the method used. Some of the serotypes isolated pose little public health concern. Where serotype O157 is sought specifically the prevalence is particularly low.

An interesting point made in the Little and Louvois (1999) study was that several of the samples collected as unpasteurised samples, were phosphatase negative indicating that they had been pasteurised. This raises trading standards questions and should also be considered in any sampling programme of raw milk.

Research by Mechie et al. (1997) pointed out a distinction between foremilk and mid stream. They isolated O157 from fore milk samples but not from mid stream samples. They concluded that thorough teat sanitisation and removal of foremilk may help to prevent contamination.
6 RISK CHARACTERISATION

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

6.1 Adverse Health Effects in New Zealand

6.1.1 Incidence

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 trend over the period 1993-2004 is also shown in Figure 2. The number of notified cases of STEC infection where raw milk consumption was reported (as one of several potential risk factors) are also included in Table 9. Generally, the percentage of cases exposed to raw milk out of the overall number of STEC cases ranges between 1 to 10%. Reporting of raw milk consumption as a behaviour in the period prior to illness does not conclusively indicate that the infection was acquired from that source; many of these cases live on farms where there will be other potential exposures to STEC e.g. direct animal contact.

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

<table>
<thead>
<tr>
<th>Year</th>
<th>Rate per 100,000 (number of notified cases)</th>
<th>Number of cases who reported raw milk consumption¹</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>1998</td>
<td>1.3 (48)</td>
<td>3</td>
<td>Baker et al., 1999</td>
</tr>
<tr>
<td>1999</td>
<td>1.8 (64)</td>
<td>1</td>
<td>Kieft et al., 2000</td>
</tr>
<tr>
<td>2000</td>
<td>1.9 (68)</td>
<td>11</td>
<td>Lopez et al., 2001</td>
</tr>
<tr>
<td>2001</td>
<td>2.0 (76)</td>
<td>4</td>
<td>Sneyd et al., 2002</td>
</tr>
<tr>
<td>2002</td>
<td>2.0 (73)</td>
<td>6</td>
<td>Sneyd &amp; Baker, 2003</td>
</tr>
<tr>
<td>2003</td>
<td>2.8 (105)</td>
<td>8</td>
<td>ESR, 2004</td>
</tr>
<tr>
<td>2004</td>
<td>2.4 (89)</td>
<td>5</td>
<td>ESR, 2005a</td>
</tr>
<tr>
<td>2005</td>
<td>2.5 (92)</td>
<td>Data not analysed</td>
<td>ESR, 2006a</td>
</tr>
</tbody>
</table>

¹ Based on information given in Table 10
From the ESR (2005a) report, of the 89 cases notified in 2004, 82 were confirmed by the ESR Enteric Reference Laboratory from faecal isolates, (75 were O157 (91.5%) and the other 7 were non-O157 serotypes). The remaining 7 cases were notified based on clinical symptoms. In terms of gender, 36 cases were male (rate 2.0/100,000) and 53 cases female (rate 2.8/100,000). Regional variations were found. The highest rates were recorded in the Waikato (30 cases: 9.4 per 100,000), Bay of Plenty (15 cases: 8.4) and Tairawhiti (2 cases: 4.6) District Health Boards. The high incidence of STEC in the Waikato may be associated with the high number of cattle in the district (section 5.1.1).

Notification rates in 2004 were highest in European (69 cases) and Pacific Peoples (3 cases) ethnic groups (2.6 and 1.5 per 100,000 respectively). There were 6 cases reported from Maori groups, a rate of 1.1.

Infection with STEC can affect any age group but most often causes disease in children aged 4 years or less. In 2004, in the <1 age group, there were 11 cases (20.1 per 100,000), in the 1 to 4 age group, 35 cases; 16.2 per 100,000. In the elderly populations 60-69, there were 5 cases; 1.8 per 100,000 and in the 70+ age group, 4 cases; 1.2 per 100,000 (ESR, 2005a). Children under five years are most susceptible to HUS whereas the elderly are more likely to develop TTP (Baker et al., 1999).

Based on Canadian studies, in New Zealand it has been assumed that a further 10-12 cases of STEC infection occur for each reported case (Baker et al., 1999). This would equate to 890 to 1068 cases in 2004 in New Zealand.

In the USA, this figure has been estimated to be much higher, i.e. for each confirmed case of STEC infection reported, 13-27 cases of E. coli O157 infection occur in the community.
The total number of cases with non-O157 STEC has been assumed to be 50% of the rate for O157:H7 (Mead et al., 1999).

A search of the Episurv database was carried out in December 2005 to identify cases of STEC infection where raw milk consumption was reported as a risk factor. These cases had consumed raw milk in the week prior to illness. In the majority of these cases, other risk factors were also present. The results are presented in Table 10.

Table 10: Cases of STEC infection who reported raw milk consumption; 1997 – November 2005

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Gender/age</th>
<th>Serotype</th>
<th>HUS</th>
<th>HC</th>
<th>TTP</th>
<th>Type of milk</th>
<th>Hospital</th>
<th>Living on farm</th>
</tr>
</thead>
<tbody>
<tr>
<td>1997CB02650</td>
<td>M/ 6 years</td>
<td>O113: H21</td>
<td>U</td>
<td>U</td>
<td>U</td>
<td>N/a</td>
<td>No</td>
<td>Visits farms regularly</td>
</tr>
<tr>
<td>1998RO00060</td>
<td>M/ 11 months</td>
<td>O157:H7</td>
<td>U</td>
<td>U</td>
<td>U</td>
<td>N/a</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>1998RO00252</td>
<td>M/ 2 years</td>
<td>N/a</td>
<td>U</td>
<td>U</td>
<td>U</td>
<td>N/a</td>
<td>Yes</td>
<td>No – but animal contact</td>
</tr>
<tr>
<td>1998WK01547</td>
<td>M/ 1 year</td>
<td>O157</td>
<td>U</td>
<td>U</td>
<td>U</td>
<td>Pet milk^</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>1999WK00576</td>
<td>F/ 1 year</td>
<td>O157:H7</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>From dairy</td>
<td>No</td>
<td>Yes -dairy</td>
</tr>
<tr>
<td>2000BE00040</td>
<td>F/ 2 years</td>
<td>N/a</td>
<td>U</td>
<td>U</td>
<td>U</td>
<td>N/a</td>
<td>No</td>
<td>No but animal contact</td>
</tr>
<tr>
<td>2000CB00303</td>
<td>F/ 6 years</td>
<td>O157:H NM</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>Farm milk</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>±2000RO0092</td>
<td>F/ 8 years</td>
<td>O157:H NM</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>Farm milk</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>±2000RO0093</td>
<td>F/ 1 year</td>
<td>O157</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>Farm milk</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>±2000RO0096</td>
<td>F/ 8 years</td>
<td>O157</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>Farm milk</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>2000WK00263</td>
<td>M/ 1 year</td>
<td>N/a</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>Farm milk</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>2000WK00376</td>
<td>M/ 2 years</td>
<td>O157</td>
<td>U</td>
<td>U</td>
<td>U</td>
<td>N/a</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>2000WK00435</td>
<td>F/ 35 years</td>
<td>O157</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>N/a</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>2000WK00637</td>
<td>M/ 1 year</td>
<td>O157</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>N/a</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>2000WK01399</td>
<td>F/ 1 year</td>
<td>N/a</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
<td>N/a</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>2000BE00074</td>
<td>M/ 1 year</td>
<td>N/a</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>N/a</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>2001CB01476</td>
<td>M/ 16 years</td>
<td>O157</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
<td>N/a</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>2001RO00052</td>
<td>M/ 32 years</td>
<td>O157:H7</td>
<td>No</td>
<td>Yes</td>
<td>U</td>
<td>Raw milk from farm vat</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>2001WK01060</td>
<td>M/ 1 year</td>
<td>O157:H7</td>
<td>U</td>
<td>U</td>
<td>U</td>
<td>Farm milk</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>2001WK01273</td>
<td>M/ 11 months</td>
<td>O157</td>
<td>No</td>
<td>U</td>
<td>U</td>
<td>bought– raw goat’s milk</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>2002AK03748</td>
<td>M/ 2 years</td>
<td>N/a</td>
<td>U</td>
<td>Yes</td>
<td>U</td>
<td>N/a</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>2002AK03975</td>
<td>F/ 7 years</td>
<td>N/a</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>Raw cream at petting farm</td>
<td>No</td>
<td>Visitor to petting farm</td>
</tr>
<tr>
<td>2002CB01740</td>
<td>F/ 67 years</td>
<td>O rough :H7</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
<td>Farm milk</td>
<td>Yes</td>
<td>Visitor to relative’s farm</td>
</tr>
<tr>
<td>2002WK00426</td>
<td>F/ 1 year</td>
<td>O157</td>
<td>U</td>
<td>U</td>
<td>U</td>
<td>N/a</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>2002WK01405</td>
<td>M/ 1 year</td>
<td>N/a</td>
<td>U</td>
<td>U</td>
<td>U</td>
<td>Neighbour’s farm milk</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>2002WK01420</td>
<td>M/ 1 year</td>
<td>O157</td>
<td>U</td>
<td>Yes</td>
<td>U</td>
<td>Farm milk</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>2003NL00081</td>
<td>M/ 4 years</td>
<td>O157</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td>Farm milk</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Case No.</td>
<td>Gender/age</td>
<td>Serotype</td>
<td>HUS</td>
<td>HC</td>
<td>TTP</td>
<td>Type of milk</td>
<td>Hospital</td>
<td>Living on farm</td>
</tr>
<tr>
<td>---------------</td>
<td>------------</td>
<td>----------</td>
<td>-----</td>
<td>----</td>
<td>-----</td>
<td>--------------</td>
<td>----------</td>
<td>-------------------------</td>
</tr>
<tr>
<td>2003WK00671</td>
<td>M/1 year</td>
<td>O157</td>
<td>U</td>
<td>U</td>
<td>U</td>
<td>N/a</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>2003WK00701</td>
<td>M/1 year</td>
<td>N/a</td>
<td>U</td>
<td>Yes</td>
<td>U</td>
<td>N/a</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>2003WK00706</td>
<td>F/1 year</td>
<td>N/a</td>
<td>U</td>
<td>U</td>
<td>U</td>
<td>Farm milk</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>2003MW00303</td>
<td>F/1 year</td>
<td>O157</td>
<td>U</td>
<td>U</td>
<td>U</td>
<td>Farm milk</td>
<td>No</td>
<td>Visitor to relative’s farm</td>
</tr>
<tr>
<td>2003TK00348</td>
<td>F/23 years</td>
<td>O84:HN</td>
<td>U</td>
<td>U</td>
<td>U</td>
<td>N/a</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>2003SC00410</td>
<td>M/2 years</td>
<td>O157:H7</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>Farm milk</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>2003OT00684</td>
<td>F/4 years</td>
<td>N/a</td>
<td>U</td>
<td>No</td>
<td>U</td>
<td>Cowshed milk</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>2004TG00041</td>
<td>M/2 years</td>
<td>O157:H7</td>
<td>U</td>
<td>No</td>
<td>U</td>
<td>Farm milk</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>2004TK00319</td>
<td>F/1 year</td>
<td>O157</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>Farm bulk tank</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>2004RO00363</td>
<td>F/2 years</td>
<td>O157:H7</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
<td>N/a</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>2004TG00479</td>
<td>M/35 years</td>
<td>N/a</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>N/a</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>2004SO00857</td>
<td>M/11 years</td>
<td>O84:H2</td>
<td>No</td>
<td>U</td>
<td>No</td>
<td>Consumed lots of farm milk</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>2005TG00264</td>
<td>F/1 year</td>
<td>O157:H7</td>
<td>U</td>
<td>U</td>
<td>U</td>
<td>Farm milk</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>2005RO00289</td>
<td>M/2 years</td>
<td>O157</td>
<td>U</td>
<td>U</td>
<td>U</td>
<td>Pasteuriser on farm, assumed to have consumed pasteurised milk</td>
<td>No</td>
<td>Yes</td>
</tr>
</tbody>
</table>

N/a: not available from Episurv information  
U: unknown  
#: case fed pet lambs with high colostrum raw milk  
±: family outbreak

Out of the 41 cases summarised above, 29 lived in a farm environment and 4 were visitors. It must be noted that consumption of raw milk was often not the only risk factor present because living in a farm environment, there would often be exposure to farm animals, manure, private water supplies etc. Serotype O157 was responsible for 26 of the infections, information was not available for 12 cases and in the remaining 3 cases, the serotypes were O84:H2, O84:HNM and O113:H21.

Case number 2001CB01476 involving a 16 year old male in Canterbury was detailed in Lablink Journal (ESR, 2001a) and involved an E. coli O157 isolate that was indistinguishable from the isolate from a one year old male case in Canterbury, although the cases did not know each other. The one year old case does not appear under the raw milk consumption linked cases in Episurv. However in Monthly Surveillance Report, September 2001 (ESR, 2001b) a report indicates that the one-year old had HUS. The case lived on a farm where raw milk was fed to seven cats and the child had possibly licked milk out of the cat feeding bowls. Molecular subtyping established an indistinguishable serotype isolated from both the child and the raw milk. This was the first isolate of E. coli O157 from a food source in New Zealand.
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

<table>
<thead>
<tr>
<th>Period</th>
<th>Hospitalised*</th>
<th>HC*</th>
<th>HUS*</th>
<th>TTP*</th>
<th>Fatalities</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oct ‘93-</td>
<td>24/58 (41.4%)</td>
<td>21/59</td>
<td>18/59</td>
<td>1/59</td>
<td>2/79</td>
<td>Baker et al., 1999</td>
</tr>
<tr>
<td>Dec 98</td>
<td></td>
<td>(35.6%)</td>
<td>(30.5%)</td>
<td>(1.7%)</td>
<td>(2.5%)</td>
<td></td>
</tr>
<tr>
<td>1999</td>
<td>20/61 (32.8%)</td>
<td>16/33</td>
<td>4/44</td>
<td>0/26</td>
<td>0</td>
<td>Kieft et al., 2000</td>
</tr>
<tr>
<td>2000</td>
<td>12/67 (17.9%)</td>
<td>14/51</td>
<td>1/35</td>
<td>0/30</td>
<td>0</td>
<td>Lopez et al., 2001</td>
</tr>
<tr>
<td>2001</td>
<td>17/75 (22.7%)</td>
<td>26/52</td>
<td>3/35</td>
<td>0/30</td>
<td>0</td>
<td>Sneyd et al., 2002</td>
</tr>
<tr>
<td>2002</td>
<td>16/67 (23.9%)</td>
<td>28/50</td>
<td>4/36</td>
<td>1/34</td>
<td>0</td>
<td>Sneyd and Baker, 2003</td>
</tr>
<tr>
<td>2003</td>
<td>24/99 (24.2%)</td>
<td>43/74</td>
<td>10/59</td>
<td>1/53</td>
<td>0</td>
<td>ESR, 2004</td>
</tr>
<tr>
<td>2004</td>
<td>27/86 (31.4%)</td>
<td>43/58</td>
<td>5/38</td>
<td>1/33</td>
<td>0</td>
<td>ESR, 2005a</td>
</tr>
<tr>
<td>(74.1%)</td>
<td></td>
<td>(71.2%)</td>
<td>(13.2%)</td>
<td>(3.0%)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* Percentages are determined on the basis of cases for which information was available
Data updated from EpiSurv 07.04.06.

6.1.3 Serotypes causing disease in New Zealand.

In New Zealand, the predominant STEC isolated from human cases is *E. coli* O157. For example, of the 89 notified STEC cases in 2004, 82 were confirmed isolates and 75 (91.5%) were caused by *E. coli* O157:H7 (ESR, 2005a). Other serotypes that have caused infections over recent years include O113:H21, O26:H-, O84:HNM, O84:H2, O91:H21, O145:H-, ONT:H18, ONT:H6, and O128:H-. Some isolates causing infection are ONT:H- i.e. have not been typable. There have been two deaths attributed to STEC (both prior to 1999), one to serotype O157 and the other to O113:H21 (Carolyn Nicol, ESR Enteric Reference Laboratory, personal communication, March 2005).

Stool specimens (n=484) from children suffering from diarrhoea submitted to the Dunedin Public Hospital laboratory were examined in a study in 1996 (Brooks et al., 1997). Sixteen cultures were identified as *E. coli* cytotoxic to Vero cells, but only serotypes O26:H11, capable of causing HUS, and O128:H2, were toxigenic and typable. Retrospective analysis of five of these STEC showed that the O26:H11 isolate was positive for the Stx1, hlyA and eaeA genes, while the others (O128:H2, OR:H2, OR:H-) were positive for Stx1 and Stx2 but not the other genes (Brooks et al., 2001).

The serotypes O91:H-, O128:H2 and O128:H- have been isolated from New Zealand retail meat samples (Brooks et al., 2001). The latter serotype has also been isolated from a notified human case (see above). Serotype O128:H2 is another that has been isolated from both meat
and a person suffering from diarrhoea, although this was not a notified case of STEC infection.

6.1.4 Outbreaks

The reported number of outbreaks and cases for which STEC was a causative agent between 1998 and 2004 are presented in Table 12.

Table 12: Total number of reported outbreaks and cases for which STEC was identified as the causative agent in New Zealand 1998-2004

<table>
<thead>
<tr>
<th>Year</th>
<th>No. of outbreaks</th>
<th>Percent of reported outbreaks</th>
<th>No. of cases</th>
<th>Percent of cases in reported outbreaks</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>1998</td>
<td>8</td>
<td>8/313: 2.6%</td>
<td>20</td>
<td>20/2139: 0.9%</td>
<td>Naing et al., 1999</td>
</tr>
<tr>
<td>1999</td>
<td>1</td>
<td>1/361: 0.3%</td>
<td>3</td>
<td>3/2358: 0.1%</td>
<td>Galloway and O'Sullivan, 2000</td>
</tr>
<tr>
<td>2000</td>
<td>1</td>
<td>1/289: 0.3%</td>
<td>4</td>
<td>4/2296: 0.2%</td>
<td>Lopez et al., 2001</td>
</tr>
<tr>
<td>2001</td>
<td>4</td>
<td>4/389: 1.0%</td>
<td>10</td>
<td>10/2323: 0.4%</td>
<td>Sneyd et al., 2002</td>
</tr>
<tr>
<td>2002</td>
<td>1</td>
<td>1/333: 0.3%</td>
<td>3</td>
<td>3/2870: 0.1%</td>
<td>Sneyd and Baker, 2003</td>
</tr>
<tr>
<td>2003</td>
<td>2</td>
<td>2/340: 0.6%</td>
<td>4</td>
<td>4/2789: 0.1%</td>
<td>ESR, 2004</td>
</tr>
<tr>
<td>2004</td>
<td>3</td>
<td>3/327: 0.9%</td>
<td>6</td>
<td>6/4085: 0.1%</td>
<td>ESR, 2005b</td>
</tr>
<tr>
<td>Total</td>
<td>20</td>
<td>Mean 0.9%</td>
<td>50</td>
<td>Mean 0.3%</td>
<td></td>
</tr>
</tbody>
</table>

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.

In terms of overall outbreaks in 2004, STEC outbreaks accounted for 0.9% of the total, the number of cases involved was very low at 0.1% (6/4085) (ESR, 2005b).

A search of the Episurv database found 22 STEC outbreaks listed for the period 1998 to November 2005. In terms of source and vehicles involved in transmission, two of the outbreaks listed raw milk amongst other risk factors. There is no further information on these outbreaks. Other risk factors listed for these outbreaks were environmental sources, water supply, farm kill, home vegetable plot and person-to-person spread.

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. An overview of 79 cases of STEC in New Zealand between 1993 and 1998 found that 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. Animal contact was another common factor.

A further collation of risk factor information was reported for 2001 (Sneyd et al., 2002). Again there were high (>50%) proportions of cases who reported consumption of dairy products, beef, poultry, processed meats, raw fruit/vegetables, contact with animals and contact with raw meat/offal.

From the 2004 ESR Report (2005a), for cases where risk information was recorded for the week before becoming ill, 83.9% (52/62) reported contact with animals, (of these, 89.8% were pets). 64.3% (27/42) reported contact with farm animals, 52.4% (22/42) reported contact with animal manure, 35.6% (16/45) had consumed water from a non-habitual supply, 35.5% (22/62) recreational contact with water and 26.4% (14/53) reported contact with children in nappies. 25.7% (9/35) had contact with other animals and 5.4% (3/56) reported contact with sewage.

However, these analyses 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.

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, 2000a). For one New Zealand case, an indistinguishable isolate was obtained from both the infected person and raw milk present in the home, although the route of infection is uncertain (Anonymous, 2002).

6.2 Adverse Health Effects Overseas

6.2.1 Incidence

Incidence data for a selection of countries are given in Table 13. New Zealand’s incidence has been included for comparative reasons and is similar to other countries. The incidence of infection is however considerably higher in the Czech Republic and considerably lower in Australia. The reported infection rate in Scotland has declined significantly from 8.23 per 100,000 in 1997 (PHLS, 2000) to 2.9 per 100,000 in 2003. A Task Force on E. coli O157 was set up in Scotland to report on the disease, its final report was published in 2001 (see section 6.2.4.3).
Table 13: Rates of reported infections with STEC by country

<table>
<thead>
<tr>
<th>Country</th>
<th>Year</th>
<th>Incidence (per 100,000)</th>
<th>No. of lab. confirmed cases</th>
<th>% O157:H7</th>
<th>% Other VTEC</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>New Zealand</td>
<td>2004</td>
<td>2.4</td>
<td>82</td>
<td>91.5</td>
<td>8.5</td>
<td>ESR, 2005a</td>
</tr>
<tr>
<td>New Zealand</td>
<td>2005</td>
<td>2.5</td>
<td>92</td>
<td>92.4</td>
<td>7.6</td>
<td>ESR, 2006a</td>
</tr>
<tr>
<td>Australia</td>
<td>2002</td>
<td>0.3</td>
<td>53</td>
<td>-</td>
<td>-</td>
<td>Yohannes et al., 2004;</td>
</tr>
<tr>
<td></td>
<td>2003</td>
<td>0.2</td>
<td>49 (-37 -6 -3)</td>
<td>25</td>
<td>15 O111</td>
<td>Miller et al., 2005</td>
</tr>
<tr>
<td>Europe</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Community (17 member states + Norway)</td>
<td>2004</td>
<td>1.3</td>
<td>4143</td>
<td>50</td>
<td>25¹</td>
<td>EFSA, 2005</td>
</tr>
<tr>
<td>Austria</td>
<td>2004</td>
<td>0.6</td>
<td>45</td>
<td>29</td>
<td>71</td>
<td>EFSA, 2005</td>
</tr>
<tr>
<td>Belgium</td>
<td>2004</td>
<td>0.3</td>
<td>36</td>
<td>56</td>
<td>44</td>
<td>EFSA, 2005</td>
</tr>
<tr>
<td>Czech Republic</td>
<td>2004</td>
<td>17.1</td>
<td>1743</td>
<td>18</td>
<td>0</td>
<td>EFSA, 2005</td>
</tr>
<tr>
<td>Denmark</td>
<td>2004</td>
<td>3.0</td>
<td>163</td>
<td>27</td>
<td>73</td>
<td>EFSA, 2005</td>
</tr>
<tr>
<td>Finland</td>
<td>2004</td>
<td>0.2</td>
<td>10</td>
<td>40</td>
<td>60</td>
<td>EFSA, 2005</td>
</tr>
<tr>
<td>Germany</td>
<td>2004</td>
<td>1.1</td>
<td>903</td>
<td>10</td>
<td>42¹</td>
<td>EFSA, 2005</td>
</tr>
<tr>
<td>Ireland</td>
<td>2004</td>
<td>1.4</td>
<td>57</td>
<td>88</td>
<td>12</td>
<td>EFSA, 2005</td>
</tr>
<tr>
<td>Netherlands</td>
<td>2004</td>
<td>0.2</td>
<td>30</td>
<td>100</td>
<td>0</td>
<td>EFSA, 2005</td>
</tr>
<tr>
<td>Norway²</td>
<td>2004</td>
<td>0.3</td>
<td>12</td>
<td>58</td>
<td>42</td>
<td>EFSA, 2005</td>
</tr>
<tr>
<td>Poland</td>
<td>2004</td>
<td>0.2</td>
<td>81</td>
<td>99</td>
<td>1</td>
<td>EFSA, 2005</td>
</tr>
<tr>
<td>Sweden</td>
<td>2004</td>
<td>1.7</td>
<td>149</td>
<td>-</td>
<td>-</td>
<td>EFSA, 2005</td>
</tr>
<tr>
<td>United</td>
<td>2004</td>
<td>1.5</td>
<td>898</td>
<td>99</td>
<td>1</td>
<td>EFSA,</td>
</tr>
</tbody>
</table>

¹ Includes 15 O111
² Includes another 99 O111
<table>
<thead>
<tr>
<th>Country</th>
<th>Year</th>
<th>Incidence (per 100,000)</th>
<th>No. of lab. confirmed cases</th>
<th>% O157:H7</th>
<th>% Other VTEC</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kingdom</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>2005</td>
</tr>
<tr>
<td>(Scotland')</td>
<td>2003</td>
<td>2.9</td>
<td>148</td>
<td>-</td>
<td>-</td>
<td>SCIEH, 2004</td>
</tr>
<tr>
<td>North America</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Canada</td>
<td>2000</td>
<td>8.8</td>
<td></td>
<td></td>
<td></td>
<td>Health Canada (2000)</td>
</tr>
<tr>
<td>USA³</td>
<td>2004</td>
<td>0.9</td>
<td></td>
<td></td>
<td></td>
<td>Centers for Disease Control &amp; Prevention, 2005</td>
</tr>
</tbody>
</table>

* 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.
³ 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

The USA health objective for 2010 for infection with *E. coli* O157 is 1 or less per 100,000. From Table 12 above, it appears that they have currently achieved this objective. 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. In England and Wales, 31% of cases were hospitalised and an overall mortality rate of 3.7% was recorded between the years 1992 and 1996 (PHLS, 2000).

6.2.2 Contributions to outbreaks and incidents overseas

Table 14 shows the contribution of STEC to foodborne disease outbreaks and incidents overseas with New Zealand included for comparison.
Table 14: Contribution of STEC to foodborne disease outbreaks and incidents overseas

<table>
<thead>
<tr>
<th>Country</th>
<th>Year</th>
<th>Proportion of outbreaks (%)</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>New Zealand</td>
<td>2004</td>
<td>0.9</td>
<td>ESR, 2005b</td>
</tr>
<tr>
<td>Canada</td>
<td>1982</td>
<td>0.2</td>
<td>Todd, 1992</td>
</tr>
<tr>
<td>Canada</td>
<td>1983</td>
<td>0.2</td>
<td>Todd, 1992</td>
</tr>
<tr>
<td>Canada</td>
<td>1984</td>
<td>0.1</td>
<td>Todd, 1992</td>
</tr>
<tr>
<td>England and Wales</td>
<td>1995</td>
<td>1</td>
<td>Evans et al., 1998</td>
</tr>
<tr>
<td>England and Wales</td>
<td>1996</td>
<td>1.4</td>
<td>Evans et al., 1998</td>
</tr>
</tbody>
</table>

It can be concluded that only a small proportion of reported outbreaks are attributed to STEC infections in both New Zealand and overseas.

Table 15 summarises outbreaks of STEC infections from overseas, where unpasteurised milk was implicated. These outbreaks are mostly linked with consumption of milk from local farms. The milk type was cow’s milk unless otherwise stated.

Table 15: Overseas outbreaks of STEC infections where unpasteurised milk was the implicated vehicle

<table>
<thead>
<tr>
<th>Country</th>
<th>Year</th>
<th>No. Cases</th>
<th>Product</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Austria</td>
<td>2001</td>
<td>2 (both serotype O26)</td>
<td>Unpasteurised milk</td>
<td>Allerberger et al., 2003</td>
</tr>
<tr>
<td>Canada (Ontario)</td>
<td>2005</td>
<td>3</td>
<td>Unpasteurised milk</td>
<td>ProMED-mail, 2005a</td>
</tr>
<tr>
<td>Canada</td>
<td>2001</td>
<td>5 children, two with HUS</td>
<td>Unpasteurised goat’s milk</td>
<td>McIntyre et al., 2002</td>
</tr>
<tr>
<td>Canada</td>
<td>1986</td>
<td>43</td>
<td>Unpasteurised milk</td>
<td>Borczyk et al., 1987</td>
</tr>
<tr>
<td>Czech Republic</td>
<td>1995</td>
<td>5</td>
<td>Unpasteurised goat’s milk</td>
<td>Bielaszewska et al., 1997</td>
</tr>
<tr>
<td>England (Sheffield)</td>
<td>1993</td>
<td>6</td>
<td>Unpasteurised milk</td>
<td>Anonymous, 1993</td>
</tr>
<tr>
<td>England (West Sussex)</td>
<td>1998</td>
<td>7 (3 consumed cream, 4 were relations of cases)</td>
<td>Unpasteurised cream</td>
<td>Anonymous, 1998: ProMED-mail, 1998</td>
</tr>
<tr>
<td>England (North west)</td>
<td>2000</td>
<td>4</td>
<td>Unpasteurised milk</td>
<td>Anonymous, 2000b</td>
</tr>
<tr>
<td>England (South west)</td>
<td>2000</td>
<td>2 (children)</td>
<td>Unpasteurised milk</td>
<td>Anonymous, 2000b</td>
</tr>
</tbody>
</table>
Table 16 summarises outbreaks of STEC infections from overseas, where pasteurised milk was implicated due to a fault in production.

### Table 16: Overseas outbreaks of STEC infections where improperly pasteurised milk was the implicated vehicle

<table>
<thead>
<tr>
<th>Country</th>
<th>Year</th>
<th>No. Cases</th>
<th>Product</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Slovakia</td>
<td>Not stated</td>
<td>5 children, all hospitalised three with HUS. 4 adults were infected but asymptotically</td>
<td>Cream made from unpasteurised cow’s milk</td>
<td>Liptakova et al., 2004</td>
</tr>
<tr>
<td>USA</td>
<td>1973-1992</td>
<td>1 outbreak, 6 cases</td>
<td>Unpasteurised milk</td>
<td>Headrick et al., 1998</td>
</tr>
<tr>
<td>USA (prolonged outbreak)</td>
<td>1992-1994</td>
<td>16</td>
<td>Unpasteurised milk</td>
<td>Keene et al., 1997</td>
</tr>
<tr>
<td>USA (Wash.St.)</td>
<td>2005</td>
<td>16</td>
<td>Unpasteurised milk</td>
<td>ProMED-mail, 2005b</td>
</tr>
</tbody>
</table>

In a review of milkborne outbreaks of infectious intestinal disease (Gillespie et al., 2003) covering England and Wales over the period of 1992-2000, 9 were attributed to STEC. Five outbreaks involved unpasteurised milk, 3 outbreaks involved milk sold as pasteurised and one outbreak was mixed. The serotype O157 was the second most commonly detected pathogen (33%) following salmonellae (37%). Most outbreaks were linked to farms (67%). Unpasteurised milk (52%) was the most commonly reported vehicle of infection in milkborne outbreaks, followed by pasteurised milk (37%).

Among cases in Wisconsin, 7% reported exposure to unpasteurised milk/dairy products (Proctor and Davis, 2000).
A single case, with a consequent secondary case, was attributed to the consumption of unpasteurised goats’ milk in Austria (Allerberger et al., 2001). Two unrelated cases of haemolytic uraemic syndrome in the USA have also been attributed to raw milk consumption (Martin et al., 1986).

In 1998, during an outbreak of O157 linked to the consumption of unpasteurised cheese in Scotland, a 3 year old grandson of the cheesemaker developed diarrhoea and abdominal pain. He had not consumed the cheese although he had been drinking unpasteurised milk on the same farm. He was hospitalised and later discharged with no ill effects (Reid, 2001). Unfortunately the author does not mention whether stool samples from the case were positive for O157; however the 7 year old sister was later shown to be stool positive although asymptomatic.

Of cases in the Netherlands where a known risk factor was recorded, one of 43 cases reported the consumption of unpasteurised milk prior to illness (van Duynhoven et al., 2002).

6.2.3 Case-control studies overseas

There have been very few case-control studies of STEC infection that have considered raw milk consumption.

Case control studies have been carried out on two of the outbreaks summarised in Table 14. In the Czech Republic, Bielaszewská et al. (1997) describes a case-control study of an O157:H7 outbreak associated with the consumption of unpasteurised goat’s milk. Fifteen regular drinkers of the milk were identified and their antibodies compared to 45 age-matched controls (who had never drunk goat’s milk). Five of the fifteen drinkers had significantly higher anti-O157 lipopolysaccharide antibodies than the 45 controls, P = 0.0005. One observation from this study was the lack of symptoms and lack of E. coli O157 infection in farm residents, all of whom were extensively exposed to the pathogen. One suggestion was a possible protective role of anti-VT2 antibodies in the farm residents as a result of longstanding consumption of raw goat’s milk. This observation concurs with Karmali et al. (1994) and Reymond et al. (1996) in studies where a high percentage of farm family members had neutralizing antibodies to STEC infection.

A case-control study was carried out on a large outbreak involving 114 cases in Cumbria, North England in 1999 (Goh et al., 2002). Twenty cases and 52 controls were recruited. Only one variable was independently significantly associated with illness in the case, that was drinking milk from a local farm (OR 33.15, 95% CI (5.77;250.5) (P = <0.0001)). Part of milk production was pasteurised on-farm and sold to local residents. Subsequent enquiries at the farm revealed a fault in the heat exchanger plates, a failure of automatic recording of flow diversion activity and the absence of adequate temperature monitoring. The remainder of the milk production was collected by a national milk processor and pasteurised elsewhere.

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 unpasteurised dairy products as a risk factor was included in the study. The only food item
significantly associated with STEC infection were the consumption of ‘berries’ (Matched OR 11.00, 95% CI 1.26-96.12). The exposure data for unpasteurised dairy products did not find a significant association with STEC infection. 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.

A case control study was carried out by Crump et al. (2002) among visitors to a dairy and petting farm in the USA. The study focused on direct transmission from animals and their environment to humans, and concluded that contact with calves (petting) and their environment (touching railings or contact with manure) was associated with an increased risk of infection whereas hand washing was protective (3/20 cases, 18/40 controls washed hands OR 0.5, 95% CI 0.2-1.1). Raw milk was not served at the farm.

6.2.4 Risk assessments

Risk assessments for STEC infection have most often focused on meat as a risk factor; studies on raw milk are scarce.

6.2.4.1 Ireland

A study in Ireland by Buckley et al. (1998) evaluated the number of on-farm families and their visitors who were consuming raw milk, the extent of exposure to vulnerable groups and the microbiological status of the milk they were consuming. Registered liquid milk producers in eight counties were included. The results for consumption on farms are presented in Table 17.

<table>
<thead>
<tr>
<th>County</th>
<th>No. of farms surveyed</th>
<th>No. of farms where raw milk is consumed</th>
<th>Percentage of farms where raw milk is consumed</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cork</td>
<td>100</td>
<td>83</td>
<td>83%</td>
</tr>
<tr>
<td>Carlow</td>
<td>8</td>
<td>7</td>
<td>88%</td>
</tr>
<tr>
<td>Donegal</td>
<td>15</td>
<td>13</td>
<td>87%</td>
</tr>
<tr>
<td>Kildare</td>
<td>38</td>
<td>37</td>
<td>97%</td>
</tr>
<tr>
<td>Waterford</td>
<td>29</td>
<td>18</td>
<td>62%</td>
</tr>
<tr>
<td>Westmeath</td>
<td>10</td>
<td>9</td>
<td>90%</td>
</tr>
<tr>
<td>Wexford</td>
<td>10</td>
<td>9</td>
<td>90%</td>
</tr>
<tr>
<td>Wicklow</td>
<td>20</td>
<td>18</td>
<td>90%</td>
</tr>
<tr>
<td>TOTAL</td>
<td>230</td>
<td>194</td>
<td>84%</td>
</tr>
</tbody>
</table>

(Source: Buckley et al., 1998).

Further data were collected from the 100 farms surveyed in Cork; 75 families were regular consumers, 8 families occasionally and 17 families never drank raw milk. The age profiles of the family members on the 100 farms surveyed are summarized in Table 18.
Table 18: Age profile of raw milk consumers from farming communities, Cork, Ireland

<table>
<thead>
<tr>
<th>Age (years)</th>
<th>Number of respondents consuming raw milk</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Regular</td>
</tr>
<tr>
<td>0-10</td>
<td>56</td>
</tr>
<tr>
<td>10-20</td>
<td>64</td>
</tr>
<tr>
<td>20-65</td>
<td>220</td>
</tr>
<tr>
<td>&gt;65</td>
<td>60</td>
</tr>
<tr>
<td>Total</td>
<td>400</td>
</tr>
<tr>
<td>Percentage</td>
<td>79%</td>
</tr>
</tbody>
</table>

The authors concluded that the majority of people on farms in the survey were consuming raw milk. Out of the total population of 504 surveyed, there were 116 (23%) in the higher risk groups (young and elderly) who were consuming raw milk on a regular basis. The authors advocate that farming families cease drinking raw milk. They suggest the use of home pasteurisers or that families purchase pasteurised milk from the normal retail channels.

Since the publication of this report, the Food Safety Authority of Ireland have produced a leaflet aimed at farming families drinking raw milk and promoting the use of home pasteurisers; [http://www.fsai.ie/publications/leaflets/farm_industry/farmPasteurisation3.pdf](http://www.fsai.ie/publications/leaflets/farm_industry/farm Pasteurisation3.pdf).

### 6.2.4.2 South Australia

In South Australia, a Risk Profile for pathogenic *E. coli* in unpasteurised milk and pasteurised milk was compiled as part of a document on primary industries (Sumner, 2002). A risk ranking of HUS from the consumption of unpasteurised and pasteurised milk was undertaken and a summary is reproduced below in Table 19.

Table 19: Risk ranking of HUS from consumption of unpasteurised and pasteurised milk (South Australia)

<table>
<thead>
<tr>
<th>Risk criterion</th>
<th>Unpasteurised milk</th>
<th>Pasteurised milk</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dose and severity</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hazard severity</td>
<td>severe</td>
<td>severe</td>
</tr>
<tr>
<td>Susceptibility</td>
<td>general – all population</td>
<td>general – all population</td>
</tr>
<tr>
<td>Probability of exposure</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Frequency of consumption</td>
<td>daily</td>
<td>daily</td>
</tr>
<tr>
<td>Proportion consuming</td>
<td>all (100%)</td>
<td>all (100%)</td>
</tr>
<tr>
<td>Size of population</td>
<td>1,000 consumers</td>
<td>South Australia (1.5 million)</td>
</tr>
<tr>
<td>Probability of contamination</td>
<td>Probability of raw product contaminated</td>
<td>0.01%</td>
</tr>
</tbody>
</table>
### Risk Profile – STEC in raw milk

**Table 6.1: Risk of STEC Infection in Raw and Pasteurised Milk**

<table>
<thead>
<tr>
<th>Risk criterion</th>
<th>Unpasteurised milk</th>
<th>Pasteurised milk</th>
</tr>
</thead>
<tbody>
<tr>
<td>Effect of processing</td>
<td>no effect</td>
<td>reliably eliminates</td>
</tr>
<tr>
<td>Possibility of recontamination</td>
<td>none</td>
<td>none</td>
</tr>
<tr>
<td>Post-process control</td>
<td>well controlled</td>
<td>well controlled</td>
</tr>
<tr>
<td>Increase to infective dose</td>
<td>none</td>
<td>10x</td>
</tr>
<tr>
<td>Further cooking before eating</td>
<td>not effective in reducing hazard</td>
<td>not effective in reducing hazard</td>
</tr>
<tr>
<td>Total predicted illnesses per annum in selected population</td>
<td>36</td>
<td>0.05 (one every 20 years)</td>
</tr>
<tr>
<td>Risk ranking (0-100)*</td>
<td>77</td>
<td>43</td>
</tr>
</tbody>
</table>

(Source; Sumner, 2002)

*Risk rankings are 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.

Assumptions used in this risk ranking are that

- Raw milk was consumed daily by 1,000 consumers, each consuming a 250ml serving;
- In 0.01% of servings, there will be sufficient pathogens to cause infection; and,
- Pasteurised milk would need a 10-fold increase in pathogens to achieve an infective dose because the initial count/ml will be lower than in unpasteurised milk.

Further assumptions are that pasteurised milk is consumed daily by all of South Australia’s 1.5 million consumers. In addition, the hazard in pasteurised milk is completely eliminated during the pasteurising process although the potential for recontamination during cooling is included.

Based on these assumptions, with a ranking of 77 for unpasteurised milk, this equates to 36 predicted illnesses per annum among 1,000 consumers. For pasteurised milk, a ranking of 43 equates to one predicted illness every 20 years.

The government response (South Australian Government, 2003) to the Risk Profile was to ban the sale of unpasteurised cow’s milk under the provisions of the Food Act 2001. Raw goat’s milk was considered a lower risk than raw cow’s milk however, all goat milk producers in South Australia must have a HACCP based Food Safety Program that is audited by the Dairy Authority.

### 6.2.4.3 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 infection being reported. 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 sale of raw bovine milk and cream in Scotland is already prohibited, although the Task Force was to focus on the situation in Scotland, it was asked to consider situations outside of the country. Consequently the Task Force recommended that all raw drinking milk and raw cream for sale and intended to be drunk raw should be heat-treated in England and Wales.

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. The action plan addressed the heat-treatment recommendation by launching a consultation process in Wales. The action plan was “noted” in England, but the prevailing policy remained (see section 7.2.4).

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 Estimate of Risk for New Zealand

There is a shortage of data on which to base a risk assessment for STEC in raw milk in New Zealand. There is information that suggests consumption of raw milk is widespread amongst the rural population, but the size of the exposed population, and the frequency of consumption are uncertain.

There are no data indicating the prevalence of STEC in raw milk. Data from examination of animal faeces indicate that serotypes with the potential to cause human illness do occur in cattle, although some of the samples will not be from dairy cattle. The most commonly isolated serotype from human cases in New Zealand, E. coli O157, does not occur frequently amongst isolates from animal faeces.

Consumption of raw milk is a frequently reported risk factor from notified cases of STEC infection, but other agricultural risk factors will also apply to most of these cases.

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 greater than 10% (Table 11) placing this infection in the highest severity category.

The reported rate of STEC infection in 2004 was 2.4 per 100,000. Given that raw milk consumption is only one of many potential transmission routes, it seems reasonable to assign the actual rate of infection due to contamination with raw milk as less than 1 per 100,000.
### 6.5 Summary

<table>
<thead>
<tr>
<th>Food/hazard combination</th>
<th>Severity</th>
<th>Incidence</th>
<th>Trade importance</th>
<th>Other considerations</th>
</tr>
</thead>
<tbody>
<tr>
<td>STEC in raw milk</td>
<td>1 (≥5% serious outcomes)</td>
<td>4 (&lt;1 per 100,000)</td>
<td>High (control essential)</td>
<td>Incidents attract adverse media attention</td>
</tr>
</tbody>
</table>
RISK MANAGEMENT INFORMATION

The New Zealand (Milk and Milk Products Processing) Food Standard 2002 contains a number of risk management options for unpasteurised milk and associated products. The primary risk management measure for STEC in raw milk in New Zealand is the requirement that the milk is harvested under an approved Risk Management Programme (RMP) appropriate for milk that is intended for direct human consumption.

Milk intended to be sold to the person defined in Regulation (2) (b) below is required under the Animal Products Act 1999 to be harvested under an approved Risk Management Programme, the RMP must be specific enough to reflect the direct nature of consumption. To date no RMP has been approved for the harvesting of raw milk intended to be sold in this manner.

In terms of unpasteurised milk products, these are either cheeses that are made from thermised milk and undergo a “cheese treatment” or raw milk cheeses that are manufactured under the methods set down by the Swiss Federal Council (dated 18th October 1995) for Emmental, Gruyere or Sbrinz. To date no RMP or FSP has been approved for the manufacture of an unpasteurised cheese in New Zealand using cheese treatment or the Swiss cheese methods.

This section principally deals with risk management associated with raw milk intended for direct human consumption.

7.1 Relevant Food Controls: New Zealand

All food for sale in New Zealand must comply with the Food Act 1981. The sale of raw milk is restricted to farm gate sales and to milk processors under Section 11A of the Act. In effect, no raw milk can be sold or resold in New Zealand except to end users in small quantities, or to milk processors. The Act states that;

“(2) A milk producer may sell raw milk to any person if –

a) It is sold –
   i) at the producer’s dairy premises; and
   ii) in a quantity not exceeding five litres at any one time; and
b) The person intends the milk for consumption by the person or the person’s family; - and the person may buy it accordingly”.

(3) A milk producer may sell raw milk to a dairy processor (as defined in section 4(1) of the Animal Products Act 1999) who –

a) purchases the milk for processing for sale or export; and
b) is a person who –
   i) carries out the processing under a risk management programme registered under the Animal Products Act 1999 or under a Food Safety Programme”…the person may also be exempt or excused from the requirement to operate under a Risk Management Programme.
7.2 Relevant Food Controls: Overseas

This section collates information on the regulatory regimes overseas.

7.2.1 Australia

In Australia, milk must be processed according to Clause 1 of Standard 1.6.2 of the Food Code;

“(1) Milk must be pasteurised by –
(a) heating to a temperature of no less than 72°C and retaining at such
    temperature for no less than 15 seconds and immediately shock cooling to a
    temperature of 4.5°C; or
(b) heating using any other time and temperature combination of equal or
    greater lethal effect on bacteria;
    unless an applicable law of a State or Territory otherwise expressly provides.

(2) Liquid milk products must be heated using a combination of time and
    temperature of equal or greater lethal effect on the bacteria in liquid milk that would be
    achieved by pasteurisation or otherwise produced and processed in accordance with any
    applicable law of a State or Territory” FSANZ

No Australian State permits the general sale of unpasteurised cows milk for drinking purposes. In Queensland, unpasteurised goats’ milk may be sold where it is produced by an accredited supplier and is appropriately labelled. Queensland also has limited exemptions for personal consumption from own animals (www.safefood.qld.govt.au). There have been sales of raw cows milk in Queensland where the product is sold as a “cosmetic” such as in bath milk. The legality of this is unknown.

7.2.2 United States of America

An overview of the raw milk statutes and administrative codes in the USA at 1 December 2004 can be found at the following website; http://www.realmilk.com/milk-laws-1.html.

In 1987, the USDA regulated that all milk sold interstate must be pasteurised. At 1 December 2004, 28 States allowed the intrastate sale of raw milk. The estimated volume of raw milk sold as a percentage of total milk was less than 1% (Headrick et al., 1998). In addition, five States permit sale for animal consumption, but not for human consumption.

Research by Headrick et al. (1998) examined the epidemiology of raw milk-associated foodborne outbreaks (including STEC outbreaks) in the US between 1973-1992. The study compared the source of outbreaks and the legality of raw milk sales within each State. Forty (87%) of the 46 reported raw milk associated outbreaks occurred in States where the sale of raw milk was legal. The outbreak rates were recorded as 40 outbreaks/544 state-years where raw milk sales were legal (7.35 outbreaks/100 state-years), compared to 6 outbreaks/476 state-years where raw milk sales were prohibited (1.26 outbreaks per 100 state-years). The majority of the outbreaks were caused by Campylobacter followed by Salmonella, Staphylococcus and E. coli O157:H7. The authors concluded that those States allowing the sale of raw milk had significantly higher rates of raw milk associated illness.
Prohibition in some States is being overcome by “cow share” programmes whereby farmers keep and milk cows owned by individuals in the scheme. Such a programme was involved in the recent outbreak of STEC infection from raw milk in Washington State and Oregon (ProMED-mail, 2005b).

7.2.3 Canada

The Food and Drug Regulations 1991 require all milk for retail sale to be pasteurised. A statement issued by Health Canada (http://www.hc-sc.gc.ca/fn-an/securit/facts-faits/rawmilk-laitcru_e.html) reinforces the message that the sale of raw milk in Canada is prohibited.

7.2.4 United Kingdom

A paper has been produced by the Food Standards Agency (2005) that differentiates between the requirements regarding raw milk in each country of the United Kingdom.

In England and Wales, the primary legislation that governs raw cows’ milk is Regulation 32, Schedule 6 of The Food Hygiene (England) Regulations 2005 which comes into effect 1 January 2006. Only a registered production holding where the cows are kept can sell the milk. The sale of the milk must be to the ultimate consumer, to a temporary guest or visitor to the farm for refreshment or part of a meal or to a distributor who can only sell on to the final consumer. Sales through other outlets are banned. The raw milk plate count at 30°C (cfu/ml) must be less than or equal to 20,000 and coliforms (cfu/ml) must be less than 100. This is much more stringent than for milk intended for pasteurisation. Milk that has not been pasteurised must bear a label or a notice must be displayed. The wording must read as follows;

“This milk has not been heat treated and may therefore contain organisms harmful to health”

There are no accurate figures available on the actual quantity of raw milk being sold however over recent years, registered holdings have declined from 570 in 1997 to 149 in 2005 (Food Standards Agency, 2005). The dairy herd must be officially tuberculosis and brucellosis free, the dairy must comply with hygiene rules and monitoring inspections are carried out twice a year. The Dairy Hygiene Inspectorate test quarterly to monitor for total bacterial count and coliforms. Sampling and analysis costs are borne by the occupier of the production holding, with a current fee of £63.

Raw drinking milk from goats is not subject to the same restrictions as cows’ milk. However, the goats must be brucellosis-free, the dairy must comply with hygiene rules and the milk must carry the same health warning.

Raw cream is not subject to sale outlet restrictions and is not required to carry the health warning.

In Northern Ireland, the controls are similar to England and Wales although there are no known sales of raw milk or cream.
In Scotland, the sale of raw cows’ milk and cream has been prohibited since The Milk (Special Designations) (Scotland) Order 1980 was implemented on 1 August 1983. The prohibition followed several milk-borne infections and 12 potentially associated deaths. There has been a decline in associated illness since. The sale of raw drinking milk and raw cream from sheep, goats and buffaloes are permitted from the farm gate only. There is no health warning requirements on these species’ of milk.

The Government of England, Wales and Northern Ireland has assessed a ban on raw milk three times since 1984. The most recent assessment was in 1997. Producers and consumers have consistently opposed a ban, however restrictions have been imposed subsequent to each assessment.

From 1 January 2006, new European Commission hygiene rules will allow member states to specifically introduce rules prohibiting or restricting raw milk or raw cream. In general, there will be little change in the UK. In Scotland, Ministers wish to extend the ban on raw cows’ milk and cream to the other species. In Wales, enhanced labeling requirements will be introduced and in England, existing controls will be maintained.

7.2.5 Ireland

Since July 1997 the sale of raw milk directly for human consumption has been banned (Buckley et al., 1998).

7.3 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 and non-O157 STEC infections. The estimated cost was $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).

### 7.4 Risk Management Options

#### 7.4.1 On-farm controls

There is considerable interest in finding ways to reduce *E. coli* faecal shedding, particularly in cattle. The nature and composition of foodstuffs, diet additives and immunisation have all been reported in the scientific literature as ways to suppress the organism (McDowell and Sheridan, 2001). Recent research (Brashears et al., 2003; Tkalcic et al., 2003) has investigated the use of probiotic formulas such lactobacillus-based direct-fed microbials (DFMs) to reduce faecal shedding of *E. coli* O157:H7 and *E. coli* O111.

Treatment of manure has also been studied. For example, cattle manure can be treated with carbonate to eliminate *E. coli* (Jarvis et al., 2001) and subsurface injection of manure 25 cm below soil surface can reduce the pathogen’s survival (Avery et al., 2004).

In terms of safe harvesting of milk, ensuring that only healthy animals are used and that the teats are clean prior to milking is a key factor.

Dairy Processing Criteria (DPC) 2: Animal Products (Dairy), the Approved Criteria for Farm Dairies was published in June 2006 by the NZFSA. This document sets out additional criteria (building on DPC 1 – General Dairy Processing) for raw milk and states requirements against which Risk Management Programmes on farm dairies can be assessed. These include:

Part 3, 10 (1) “Milking Animal Health”, states that raw milk must be supplied from healthy animals, with further qualifying requirements in paragraphs (2) to (15).

And,

Part 2, 8 (11) states that there must be procedures for milking only animals with clean udders and teats. There are no further guidelines in this respect. Part 2, 9 (3) states that after filtering, milk must be cooled to 7°C or below within 3 hours of the completion of milking, and kept at or below 7°C until it is collected or until the next milking.

There is considerable debate internationally on the issue of washing and disinfection of the teats. Washing procedures are advocated by some while others argue that good husbandry should keep the teats clear of manure and other soiling. Some hold the view that by introducing a washing step, further complications in udder health can arise. For example, milking wet teats increases the likelihood of mastitis.

The following steps are recommended by the Global Organisation for Mastitis Control and Milk Quality (website accessed April, 2007 - [www.nmconline.org/milkprd.htm](http://www.nmconline.org/milkprd.htm));

- Clipping of hair from udders to reduce dirt and manure adherence;
• Washing hands and drying thoroughly before milking begins;
• Checking foremilk for “clotty, stringy or watery” milk, on each quarter of every cow;
• Use of a strip cup when checking foremilk, milk should never be stripped into the hand;
• Washing teats only, not the entire udder;
• Individual paper towels to finish drying the teats;
• Pre-dipping entire length of teat into a sanitizer works best when the teats are relatively clean;
• Predip contact time is at least 30 seconds, then thoroughly wiped-off prior to milking unit attachment;
• After milking, dipping at least the lower third of each teat in a commercial teat antiseptic product destroys organisms on teats and prevents canal colonization. Teat dip cups should be kept in a clean, sanitary manner; and
• Logistic milking procedures that allows clean mastitis-free cows to be milked first and finishes with cows with clinical mastitis.

Because goat faeces are drier than those from cattle, soiling of the udders is generally less of an issue in these animals. A factsheet prepared by the Ministry of Agriculture, Food and Rural Affairs in Canada provides advice on udder preparation for goats:
http://www.omafra.gov.ca/english/livestock/goat/facts/03-061.htm

7.4.2 Consumer advice

Advice to consumers on the drinking of raw milk is available at the NZFSA website; http://www.nzfsa.govt.nz/consumers/food-safety-topics/foodborne-illnesses/raw-milk/rawmilk.htm

This advocates the avoidance of unpasteurised milk, especially for vulnerable groups. To minimise risks, the advice is to refrigerate (below 4°C) and to discard the milk if it has been out of refrigeration for more than two hours.

The Canterbury District Health Board (2004) advocate the home treatment of raw milk. Time/temperature combinations given are 72°C to 75°C for 15 seconds. The one page leaflet can be accessed at the following website; http://www.cpublichealth.co.nz/files/MED0176.pdf

Further research into the safety of raw milk and raw milk products is being coordinated by the NZFSA. The most recent media release was in October 2005; http://www.nzfsa.govt.nz/publications/media-releases/2005-10-21-milk.htm

7.5 Other Transmission Routes

Non-foodborne transmission routes for infection with STEC include;
• Non-reticulated water supplies;
• Recreational contact with water;
• Animal contact;
• Contact with children in nappies;
• Contact with sewage; and
• Secondary infection from another case.
8 CONCLUSIONS

8.1 Description of Risks to New Zealand Consumers

8.1.1 Risks associated with raw milk

As discussed in Section 6.3 there is a shortage of data on which to base a risk assessment for STEC in raw milk in New Zealand. Major data gaps exist, both for the consumption of raw milk and prevalence of STEC in raw milk.

There is some evidence for the occurrence of STEC in faeces of dairy and beef cattle, but whether this results in contamination of raw milk is uncertain. Laboratory studies indicate that STEC survive well in raw milk, and will grow rapidly if temperature abuse occurs. Information on risk factors from human infections indicate that approximately 10% report consumption of raw milk, but this is usually within a farm environment where other risk factors will occur frequently.

Overseas outbreaks of STEC infection involving raw milk often involve small numbers of cases, probably due to the limited distribution of this type of dairy product. The same may be occurring in New Zealand, given the number of small clusters of STEC cases reported.

At present STEC infection via raw milk is a minor risk from the national perspective. However, if legislation regarding the sale of raw milk were to change to permit wider sales and consumption were to increase, it would be reasonable to expect an increase in notified STEC cases. Raw milk consumption in the rural community may be more common than the limited data suggest, and will be augmented by those who consume raw milk for perceived health and nutritional benefits. However, the risk for those consumers is difficult to assess, given the shortage of raw milk prevalence or animal carriage data. Given the serious nature of the illnesses caused by STEC infection, it would be worthwhile to further investigate this issue. Such investigations should take place within the context of a more general study of all sources of STEC infection in New Zealand.

8.1.2 Risks associated with other foods

The main vehicle implicated in foodborne outbreaks of STEC infection is red meat. In the United States ground beef/hamburger is the food vehicle most likely to be implicated in outbreaks of E. coli O157:H7. Other food vehicles implicated in outbreaks overseas are contaminated foods not cooked prior to consumption such as salads or consumption of unpasteurised foods. For example fruit juices, particularly apple juice and cider have been implicated, especially where the apples have been in contact with animal faeces or manure. Contact with animals, and consumption of contaminated drinking water or contact with recreational waters have also been identified as transmission pathways. There is no current information to indicate the relative risk of raw milk compared with other foods as a vehicle in New Zealand.

8.1.3 Risk assessment options

A quantitative risk assessment would be feasible, provided sufficient data on the prevalence of the organism in the product could be obtained. Consumption data will be difficult to
quantify because raw milk is not sold through the usual retail outlets. Information on farm gate sales and consumption by farmers, their immediate family, visitors and farm workers would be required.

8.2 Commentary on Risk Management Options

Given the serious consequences of STEC infection and growing rates of STEC infection in New Zealand, it is essential that efforts continue to minimise the likelihood of foodborne transmission.

8.3 Data Gaps

The data gaps identified in this Risk Profile are:

- Consumption data for raw milk in New Zealand;
- Prevalence of STEC carriage by dairy cattle in New Zealand (research on this topic is underway at ESR);
- Prevalence of STEC in raw milk in New Zealand (research in this topic is being developed by NZFSA); and,
- Identification of the principal human infection pathways for STEC in New Zealand.
9 REFERENCES


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.

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

* 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:

<table>
<thead>
<tr>
<th>Category</th>
<th>Rate range</th>
<th>Comments/examples</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>&gt;100</td>
<td>Significant contributor to foodborne campylobacteriosis Major contributor to foodborne NLV</td>
</tr>
<tr>
<td>2</td>
<td>10-100</td>
<td>Major contributor to foodborne salmonellosis Significant contributor to foodborne NLV</td>
</tr>
<tr>
<td>3</td>
<td>1-10</td>
<td>Major contributor to foodborne yersiniosis, shigellosis</td>
</tr>
<tr>
<td>4</td>
<td>&lt;1</td>
<td>Major contributor to foodborne listeriosis</td>
</tr>
</tbody>
</table>

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).

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

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

<table>
<thead>
<tr>
<th>Incidence</th>
<th>&gt;100</th>
<th>10-100</th>
<th>1-10</th>
<th>&lt;1</th>
</tr>
</thead>
<tbody>
<tr>
<td>Severity 1</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Severity 2</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Severity 3</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Alternatives:

No evidence for foodborne disease in New Zealand

No information to determine level of foodborne disease in New Zealand