RISK PROFILE:
SHIGA-TOXIN PRODUCING *ESCHERICHIA COLI* IN LEAFY VEGETABLES

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by

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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 leafy vegetables. Many such vegetables have the potential to be consumed raw, without cooking, as ready-to-eat products.

STEC infection may result in serious complications often requiring hospitalisation (approximately 32% of cases in New Zealand in 2004). Long term effects can include Haemolytic Uraemic Syndrome (HUS), kidney problems, hypertension, neurological deficits and in a very few cases the disease can be fatal.

Data on the prevalence of serotype O157:H7 in New Zealand leafy vegetables are limited.

The two surveys carried out found that;

- none of 114 samples of mostly lettuce and spinach and 60 herb samples contained O157:H7, and
- Of 474 conventional and organic lettuces tested (95 lots), all samples were negative for O157:H7. Serotype O157:H16 was detected in one organic lot, but this serotype did not possess the toxin genes(s)).

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

In 2004, 91.5% of confirmed STEC infections in New Zealand were caused by serotype O157:H7.

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

Data from overseas surveys have shown no detection of *E. coli* O157:H7 in leafy vegetables, apart from surveys in Mexico. In a review by FAO/WHO (1998), it was found that environmental exposure factors rather than the type of vegetable itself lead to the main differences in microbial loading. The consensus view is that preventing contamination with ruminant faeces in the first instance is a priority.

Decontamination of the vegetables post harvest as a control point is more difficult with limited effectiveness of disinfectants, further complicated by the claim that the pathogen can also be internalised via the plant's roots into the plant tissue itself, thus evading disinfection altogether.

General advice to consumers is that leafy vegetables should be washed thoroughly before consumption, particularly if eaten raw.

E. coli O157:H7 can grow on lettuce at higher temperatures, while at refrigeration temperatures (5^{o}C or less) the organism is stable or declines slowly. Although overseas surveys of leafy vegetables have not found *E. coli* O157:H7, apart from one in Mexico, a number of outbreaks of infection implicating leafy vegetables (mostly lettuce) as a vehicle have occurred, principally in the US. However, there is little evidence to suggest that leafy vegetables represent an important risk for transmission of pathogenic STEC in New Zealand. Limited surveys of leafy vegetables in New Zealand have failed to find *E. coli* O157:H7. Although other STEC serotypes were not analysed, and the risk from these other serotypes needs to be assessed, *E. coli* O157:H7 is the predominant serotype infecting people in New Zealand.

Leafy vegetables are frequently consumed by the adult population and approximately 69% of the consumption is in a raw form. Imported leafy vegetables are apparently a very small component of the New Zealand market. Risk management for STEC contamination of leafy vegetables focuses on prevention of faecal contamination. New Zealand producers, via their industry organisation, Horticulture NZ, limit the use of human or animal waste as fertilizer to material that has been subjected to a controlled composting process designed to eliminate pathogens.

The data gaps identified in this Risk Profile are:

- Information on transmission routes for STEC infection in New Zealand,
- Current prevalence of STEC (not just *E. coli* O157) in leafy vegetables available in New Zealand,
- Data on numbers of STEC in leafy vegetables when contamination does occur; and
- Information on the market size and market structure for leafy vegetables, including consumption patterns in at risk groups.

1 INTRODUCTION

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

Figure 1: Risk Management Framework

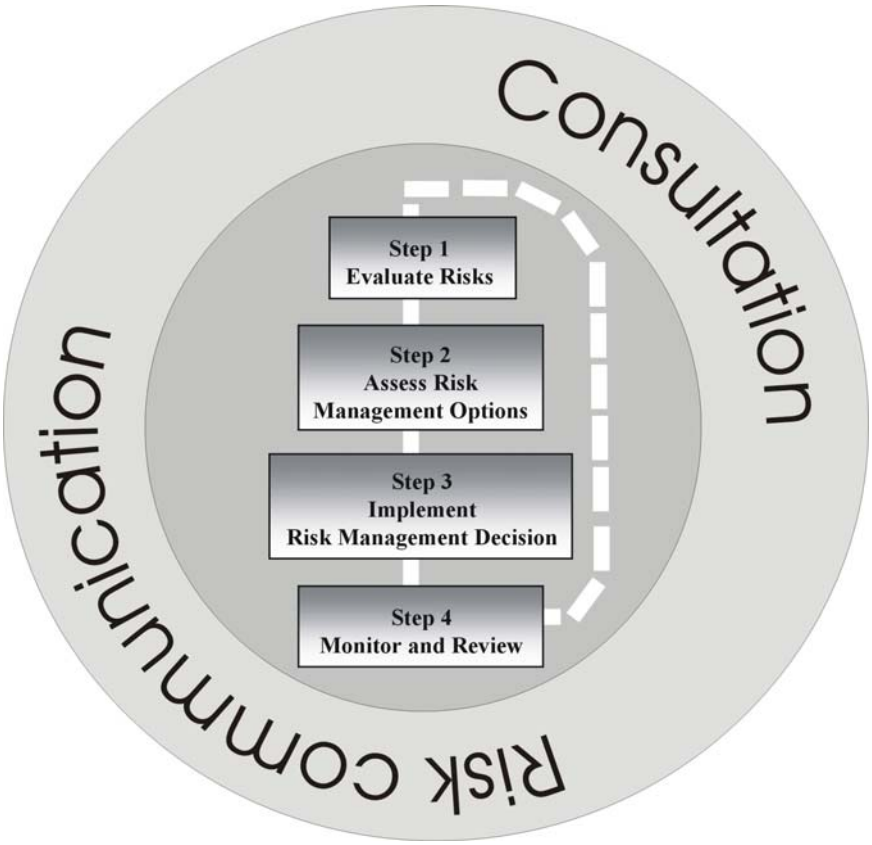


Figure reproduced from "Food Administration in New Zealand. A risk management framework for food safety" (Ministry of Health/Ministry of Agriculture and Forestry, 2000).

In more detail, the four step process is:

1. Risk evaluation

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

2. Risk management option assessment

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

3. Implementation of the risk management decision

4. Monitoring and review.

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

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 leafy vegetables. Shiga-toxins are so named due to their similarity to those produced by some species of *Shigella*. The most well known serotype of STEC is *E. coli* O157:H7 (or H-) but this profile also considers other serotypes. These organisms are important emerging pathogens, recognised for the first time in the United States in 1982. The first recognised human case of illness caused by *E. coli* O157:H7 in New Zealand occurred in 1993 (Baker *et al*., 1999).

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

Hazard identification, including:

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

Hazard characterisation, including:

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

Exposure assessment, including:

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

Risk characterisation:

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

Risk management information

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

Conclusions and recommendations for further action

2. HAZARD IDENTIFICATION: THE ORGANISM

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

The following information is taken from a number of different sources, but unless otherwise referenced, comes from data sheets prepared by ESR under a contract for the Ministry of Health. The information is now located on the NZFSA website and is 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);
http://www.nzfsa.govt.nz/science-technology/data-sheets/escherichia-coli-o157.pdf
http://www.nzfsa.govt.nz/science-technology/data-sheets/non-o157-stec.pdf

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

2.1.1 Nomenclature

E. coli is a member of the family Enterobacteriaceae. It forms part of the normal microflora in the intestinal tracts of humans and other warm blooded animals. The species is Gram-negative, facultatively anaerobic and forms short rods. *E. coli* is generally not pathogenic, but certain strains can be pathogenic to humans. 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 (verocytotoxigenic *Escherichia coli*) and STEC (shiga toxigenic *Escherichia coli*). The acronym VTEC is derived from the fact that the toxin expressed causes a pathological effect on Vero cells 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* 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.

Individual strains 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, *Stx1* and *Stx2* (previously known as Verotoxins). The characteristics of *Stx1* are generally conserved, but there are currently five recognised variants of *Stx2*. *Stx1* is both structurally and immunologically indistinguishable

from Shiga toxin and can be neutralised by anti-Shiga toxin. *Stx2* can not be neutralised in this manner (AIFST, 2003). Although, by definition, all STEC must produce either or both of *Stx1* or *Stx2*, other factors are also required for human pathogenicity and it is the possession of these that seems to determine the virulence of any one serotype. Other factors known to be involved include the ability to adhere to intestinal cells (*eaeA* gene), and the ability to produce haemolysin (*hlyA* gene).

EHEC refers to those STEC that have the capability to cause haemorrhagic colitis (HC) and haemolytic uraemic syndrome (HUS). Through general usage, EHEC includes 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, EHEC is often used incorrectly as a synonym of STEC and VTEC. The correct use of the term EHEC should refer to STEC that have caused haemorrhagic disease in humans or to predict that potential.

STEC will be the acronym used throughout this document, as older information often concerns toxin production rather than human pathogenicity. However, information about strains isolated from human cases is reviewed in this document.

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 strains may survive at lower pH levels. Ingested *E. coli* cells have to survive pH 3 or less 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).

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 serotype of EHEC associated with human disease in New Zealand is *E. coli* O157 (both *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 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^{o}C,

growth was not inhibited under 100% N_2 or 20% CO_2:80% N_2 but was inhibited under 100% CO_2. In a study by Abdul-Raouf *et al*. (1993), numbers of *E. coli* O157:H7 on lettuce and cucumber increased rapidly under an atmosphere of 97% N_2 :3% O_2 (similar to commercial conditions).

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_w = 0.995, minimum a_w permitting growth= 0.950 (about 8% NaCl) (AIFST, 2003).

Survival:

Temperature: Survives well in chilled and frozen foods. For example little change in number 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 those 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 caused by 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 or growth 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 (Pommepuy *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 unproven.

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.

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 O157:H7 at 58°C is 3.4 minutes (AIFST, 2003). When cooking a hamburger, the USFDA recommend 160°F or 71.1°C in the thickest part of patty (no time specified). The UK Food Standards

Agency recommend 70°C for 2 minutes or equivalent (e.g. 75°C for 30 seconds).

Growth may occur in contaminated vegetables, therefore temperature control of processing and storage facilities should not exceed 5°C (AIFST, 2003).

pH: Outside the pH range that allows growth, *E. coli* cells die. 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 has no inhibitory effect when 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 than *Salmonella* spp. For example, *E. coli* is able to tolerate 400µg/mL 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. On lettuce the D value was found to be different depending on the variety of lettuce tested, with D values varying between 0.1 and 0.15 kGy. In lettuce homogenates the D value was much higher (around 0.3 kGy) in three varieties, and slightly less (<0.1 kGy) in Iceberg lettuce homogenate (Niemira *et al*., 2002).

Disinfectants: *E. coli* are generally susceptible to disinfectants used in the food industry, however where the organism has adhered to, or been internalised in plant tissues such as lettuce and sprouted seeds, some protection from disinfectants is afforded the organism. Natural biofilms on sprouts can also protect the bacteria from antimicrobial compounds used to wash/irrigate and, in addition, some of the compounds are toxic to the sprouts (AIFST, 2003).

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 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.

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). *E. coli* O157 has been detected in 0.9-2.0% gull droppings in the UK (Duffy, 2003).

Food: Five ways in which fresh produce can become contaminated have been suggested by FAO/WHO (2002);

(1) Irrigation practices,
(2) Inadequate cleaning,
(3) Cleaning with contaminated water,
(4) Non-hygienic farm workers,
(5) Cross contamination from other products.

Food vehicles identified in overseas outbreaks have usually been 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, watermelons, 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 actual 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.

The relative importance of the various transmission routes is currently not well understood in New Zealand.

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

2.3.1 The organism/toxin

These organisms form a diverse group of *E. coli* serotypes that are capable of producing shiga-toxin(s), as is *E. coli* O157:H7. However, they are of widely differing pathogenic potential, varying from those that can cause illnesses similar to that produced by *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 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.

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.

3 HAZARD IDENTIFICATION: THE FOOD

The Codex Alimentarius Commission system of food classification for commodities includes under the Group 3 category of 'leafy vegetables (except brassica vegetables)':

Chard (silverbeet),	Chicory leaves,
Chinese cabbage (pe-tsai),	Cos lettuce,
Cress, garden,	Endive,
Kale,	Lettuce, head,
Lettuce, leaf,	Mustard greens,
Purslane,	Radish leaves
Spinach,	Sugar beet leaves
Turnip greens,	Watercress.

Cabbage and brussel sprouts, which may be considered to be 'leafy' are included under the Codex 'Group 4 commodity category; brassica vegetables'. For the purposes of this Risk Profile they will be included in the category of leafy vegetables. This group includes;

broccoli,	Brussel sprouts,
cabbage,	Chinese cabbage (pak choi),
red cabbage,	Savoy cabbage,
cauliflower,	collards (smooth leaved kale),
kales,	kohl rabi,
mustard greens.	

The Codex classification places herbs into a separate category, although for many herbs the leaves are the edible component and for the purposes of this Risk Profile these herbs will also be included under the description of leafy vegetables.

Many of these vegetables have the potential to be consumed raw in ready-to-eat products. Consequently we assess the risk of such uncooked products.

3.1 Relevant Characteristics of the Food: Leafy Vegetables

The pH of vegetables is in the range of 5-7 and their composition is such that growth of bacteria is favoured given sufficient moisture and warmth. If contaminated with STEC, this food group may be minimally processed or consumed raw which increases the likelihood of human infection (FAO/WHO, 2002). A Risk Profile written by the European Commission Scientific Committee on Food (2002) on the microbiological contamination of fruits and vegetables eaten raw, states: "Vegetables normally carry a non-pathogenic epiphytic microflora; pathogens may contaminate the plants via a number of routes, e.g. organic fertilisers, sewage sludge, wild and domestic animal droppings and irrigation water. In addition, where the vegetables are further prepared ready for eating; such as cutting, slicing, skinning and shredding, natural protective barriers of the plant are removed." This means that vegetables in good condition support growth less well than those that are damaged or processed by chopping, slicing etc.

Beuchat (1999) found that the release of fluids from vegetable and fruit tissues as a result of cutting or mechanical damage provided sufficient nutrients to support the growth of *E. coli* O157:H7 in the presence of the plant's natural microflora.

E. coli O157:H7 can grow on lettuce at higher temperatures, while at refrigeration temperatures (5°C or less) the organism is stable or declines slowly. Growth of *E. coli* O157:H7 on lettuce has been demonstrated in air and under a variety of modified atmospheres at 13°C, although only 1-2 $\log_{10}$ of growth occurred compared to the approximate 3 $\log_{10}$ which occurred at 22°C (Diaz and Hotchkiss, 1996). At 15°C almost 3 $\log_{10}$ of growth occurred during 2 days of storage, and the population reached a maximum of around 6 $\log_{10}$/g (Li *et al*., 2001). When lettuce was stored at 5°C a gradual decline in numbers occurred; around 1 $\log_{10}$ over 18 days. A small decline in numbers has been shown at 4°C (Beuchat, 1999), but another report showed static numbers at this temperature (Francis and O'Beirne, 2001). Abdul-Raouf *et al*. (1993), while noting a decrease at 5°C, and an increase at 12 and 21°C in numbers on shredded lettuce, noted no influence of the packaging atmosphere (3% O_2: 97% N_2 or air) on these changes.

On dry coleslaw mix a decline in numbers was measured at 4°C, and a rise followed by a fall in numbers noted at 8°C (Francis and O'Beirne, 2001).

The persistence of *E. coli* O157:H7 has been researched in relation to lettuce and parsley grown in fields treated with contaminated manure and polluted irrigation water (Islam *et al*., 2004). Contaminated manure (10^7 cfu/g) was applied the day before lettuce and parsley seedlings were transplanted. Five strips of soil were treated at a rate of 4.5 metric tonnes per hectare. Contaminated irrigation water (10^5 cfu/g) was applied once on the plants as a treatment at a rate of 2 litres per strip, three weeks after the seedlings were planted. Results showed that *E. coli* O157:H7 survived for 154 – 217 days in soil. The soil under the lettuce was devoid of the bacterium after harvest whereas the soil covered with the parsley plants contained the bacterium by > 60 days longer. *E. coli* O157:H7 was detected on the lettuce leaves and parsley up to 77 and 177 days respectively after planting. The authors concluded that the bacterium could persist in soil for >5 months regardless of crop type where the crop had been subjected to contaminated compost or irrigation water.

Studies on the cross-contamination of lettuce with *E. coli* O157:H7 via minced beef have been reported (Wachtel *et al*., 2003). The authors concluded that small numbers of the pathogen can be transferred from contaminated plastic cutting boards used to cut lettuce pieces even after successive leaves have contacted the board, e.g. pathogens were transferred to the 11th leaf (1.75×10^1 CFU on 50cm^2). At an inoculum level of 1.25×10^2 CFU, 46% of leaves including the last 25th leaf were cross-contaminated. Room-temperature stored contaminated boards decreased the number of recoverable pathogens by 1 log CFU. In addition, significant numbers of pathogens remained on these cutting boards after a 15 second warm-water rinse. Human handling of lettuce with contaminated gloved fingers (~15 seconds) resulted in approximately 10% transfer of the pathogen from the contaminated meat to lettuce (inoculum 9.6×10^4 CFU/g). When the inoculum was 9.6×10^3 CFU/g, the transfer rate was approximately 1%.

3.1.1 Effect of leafy vegetable decontamination on STEC

A review (FAO/WHO, 1998) on surface decontamination of fruits and vegetables eaten raw is available from:
http://www.who.int/foodsafety/publications/fs_management/surfac_decon/en/.

The general effect of a number of disinfecting agents (particularly chlorine) on contamination is summarised, although the amount of information specific to STEC is limited, and summarised below:

Trisodium phosphate (TSP):

E. coli O157:H7 was sensitive to 1% TSP: 10^6 cfu/ml or 10^5 cfu/cm^2 of biofilm being killed within 30 seconds at room temperature or 10°C. The pH of TSP solutions is in the 11-12 range, limiting their use on fruits and vegetables commercially.

Quaternary ammonium compounds (Quats):

These chemicals are primarily used for environmental cleaning in processing plants, and are not widely used directly on produce. They are less effective than chlorine against Gram-negative bacteria such as pathogenic *E. coli*.

The review's conclusions are:

- "Heavily contaminated fruits and vegetables should be subjected to a double wash treatment. Success in removing soil or faecal matter, and the contaminants therein, is more likely to be achieved by first washing in potable water and then washing or rinsing in water containing a disinfectant,
- The temperature of wash-water should be higher than that of the fruits or vegetables in order to minimize uptake of microorganisms by tissues [a positive temperature differential discourages infiltration of bacteria via the uptake of water, into stem tissue],
- The lethal effect of chlorine occurs within the first few seconds of treatment. The population of microorganisms decreases as the concentration of chlorine increases to about 300 ppm, above which effectiveness is not proportional to increased concentration,
- Leaving fruits and vegetables wet after disinfecting or washing can negate any beneficial effect of treatment,
- Organic acids (e.g. acetic, lactic, citric and peroxyacetic acids) have good potential as disinfectants for fruits and vegetables, but conditions under which they are most effective have not been defined, and
- Prevention of contamination …at all points from the field to the plate, through application of good agricultural practices (GAP, GMP and HACCP programmes) is preferred to application of chemical disinfectants after contamination has occurred".

More recently, considerable research effort has been expended in examining the contamination of produce by *E. coli* O157:H7, because disinfection of leafy vegetables has proved problematic. For example, application of 2,000 mg/litre calcium hypochlorite was not effective in removing *E. coli* O157:H7 from lettuce (Wachtel and Charkowski, 2002)

while 200 ppm (200mg/litre) chlorine only reduced numbers by the same amount as water (Beuchat, 1999). Results from this Beuchat study reveal that even low levels of *E. coli* O157:H7 can survive when applied to lettuce using bovine faeces as a carrier and following storage under commercial and home refrigeration conditions. The bacterium was not easily removed by washing with water or the chlorine wash which led the author to conclude that prevention of contamination with ruminant faeces is essential for minimising risk.

A suggestion has been made that dipping lettuce in water at 45-50^{o}C will prevent browning by enzymes released by cutting the leaves. An analysis of this treatment on the survival of *E. coli* O157:H7 on lettuce has been carried out (Li *et al*., 2001). A reduction of approximately 0.7 to 1.1 log10cfu/g was observed, but there was no significant difference for the presence or absence of 20 ppm chlorine, or whether the temperature of the dip was 20 or 50^{o}C. The numbers of *E. coli* O157:H7 generally declined during subsequent storage at 5^{o}C, while on lettuce stored at 15^{o}C, the *E. coli* O157:H7 population increased; the highest numbers were reached on lettuce which had been treated at 50^{o}C in the presence or absence of chlorine. This may be because more exudates (i.e. potential nutrients) were released by lettuce treated at 50^{o}C, or because competitive flora had been reduced. The authors concluded that heat treatment has promise in reducing the activity of enzymes that cause browning, thereby extending shelf life but this treatment does not inhibit but actually enhances the growth of *E. coli* O157:H7 on cut lettuce. A later study confirmed the finding by noting that treatment with cold water containing 100 ppm chlorine was detrimental to the survival of *E. coli* O157:H7 in lettuce, but when this was carried out in water at 45^{o}C, growth of the organism was favoured in lettuce subsequently stored at 10^{o}C (Delaquis *et al*., 2002).

The US FDA have produced an Industry guide entitled "Guide to Minimize Microbial Food Safety Hazards for Fresh Fruits and Vegetables" See website; http://vm.cfsan.fda.gov/~dms/prodguid.html. On the subject of the temperature of washwater it recommends:
"for some types of produce (apples, celery, tomatoes) the temperature of wash water should be greater than that of the produce or a pressure differential results that can cause water to be pulled into the plant material, causing pathogens that may be present on the produce surface or in the water to be internalized. If pathogens are pulled into the produce, washing is unlikely to reduce these pathogens. Denser products (such as carrots) do not appear to be affected by water temperature differences. For products that may be susceptible to internalization of pathogens, the recommended temperature differential may be achieved either by heating water or by air cooling produce before immersion.
- When it is not practical to expose produce to warmer water temperatures, good manufacturing practices to minimize pathogens in the water or on the surface of produce are especially important. Such practices may include using antimicrobial chemicals in the wash water, using spray-type wash treatments instead of submerging produce, and ensuring that both produce and water are clean before produce is submerged".

The FDA advice on chlorinated washwater is to add chlorine to water at a concentration of at 50 - 200 ppm total chlorine, at a pH of 6.0 - 7.5, with a contact time of 1 - 2 minutes.

The use of chlorinated water in vegetable packing houses has been reviewed by Eckert and Ogawa (1988). Pathogens were markedly reduced with increased concentrations of chlorine to 50 ppm, but increases in concentration to 200 ppm had no substantial additional effect.

Some recent information also suggests that *E. coli* O157:H7 may be internalised within lettuces by being transported from the soil, via the root system to the edible portion of the lettuce plant, thereby evading disinfectants (Solomon *et al*., 2002; Clarke, 2002).

Attachment of *E. coli* O157:H7 to roots and seed coats, as well as within the vasculature has been shown in lettuce seedlings (Wachtel *et al*., 2002b). Attachment has been shown to be similar for intact and cut lettuce leaf surfaces (Takeuchi *et al*., 2000), although statistical analysis showed a preference for adherence to cut surfaces. *E. coli* O157:H7 also adheres to the interior of stomatal pores on the leaf surface, so possibly conferring some degree of resistance to disinfectants. This has been demonstrated in experiments to show the protective effects of leaf structures against inactivation by 200 ppm chlorine. Cells penetrating to the greatest depth (30-40 μm) in cut surfaces were protected the most, those within stomatal pores to a lesser extent, and those on the surface the least (Takeuchi and Frank, 2001a). Similar observations were made when 1% NaCl-$NaHCO_3$ and a proprietary surfactant-based product were used as disinfectants (Takeuchi and Frank, 2001b).

Differential recovery and irradiation sensitivity of *E. coli* O157:H7 from cut lettuce has been shown for four varieties, suggesting that caution is needed when generalising results (Niemira *et al*., 2002).

In a study by Lin *et al*., (2002), a greater reduction in numbers (more than 4 log cfu of *E. coli* O157:H7 per lettuce leaf) while maintaining product quality was achieved by using 2% hydrogen peroxide at 50^{o}C for 60 and 90 seconds followed by a water wash. However it was recognised that the treatment did not have regulatory approval. The control treatment of sterile de-ionised water under all temperature-time combinations achieved approximately 1 log cfu per leaf reduction. There was no statistical difference between the 60 and 90 second parameters, and the authors noted that previous studies had shown that increasing exposure time from 1 to 10 minutes did not significantly further reduce populations of *E.coli* O157:H7. Beuchat *et al*., (1998) concluded that inactivation of most microorganisms on produce occurred within 1 minute after the application of chlorine.

3.1.2 Effect of Dressings on STEC

When leafy vegetables are used to prepare products such as coleslaw the presence of a dressing will influence survival. The acidity of dressing is due to the presence of acetic acid. Mayonnaise usually has a pH of 3.6 to 4.0 which rises when mixed with raw vegetables; coleslaw generally has a pH around 4-4.5.. Acetic acid has been reported as more inhibitory to *E. coli* O157:H7 than lactic, citric or malic acid. However, if the mayonnaise is cross-contaminated by foods such as raw beef, unclean utensils or infected food handlers after the commercial containers are opened, the pathogen may survive at 5^{o}C for several weeks (Hathcox *et al*., 1995).

Wu *et al*., (2002) showed that *E. coli* O157:H7 declined in numbers in coleslaw made with two commercial coleslaw dressings of pH 4.3 and 4.5 stored at 21^{o}C, 11^{o}C and 4^{o}C over 3 days. The greatest reduction in numbers (0.4-0.5 $\log_{10}$ cfu/g) occurred at 21^{o}C, compared to 4 or 11^{o}C (0.1-0.2 $\log_{10}$ cfu/g).

3.2 The Food Supply in New Zealand

3.2.1 Production

A 2003 vegetable industry fact sheet (http://www.vegetables.co.nz/about/4_stat.cfm) indicates that the area in vegetable production is 50,000 ha, with 2,800 commercial growers employing over 25,000 people. The Horticulture New Zealand website gives information on trends in the New Zealand vegetable market (http://www.hortnz.co.nz). Information on the New Zealand Fresh Produce Approved Supplier Programme is given later in section 7.1.1.

A general concentration of buying power has occurred with the development of supermarkets, and this has led to amalgamation of some operations into integrated packhouse and marketing groups. The number of smaller growers has declined to approximately 3,500 in recent years, and their inability to supply large markets like supermarkets has created alternates, such as flea markets, supplying direct to consumers.

Pukekohe remains the largest production area. However production is moving outside the traditional areas with growers increasingly looking at Pukekawa, Waikato and other regions. Increased land costs and pest and disease pressures are making production in other areas more competitive. This has seen increasing production in areas like Gisborne and Hawkes Bay. Other major production areas are Canterbury and Horowhenua. There are a number of areas that specialise in crops, such as Ohakune carrots, Northern Wairoa kumara and Marlborough garlic.

The website http://www.hortresearch.co.nz/files/2004/facts-figs-2003.pdf) provides an overview of the horticultural industry to the year ending June 2003. The following data in Table 1 are extracted from page 14 of the document. Domestic and export values indicate that the vast majority of this produce is destined for the domestic market.

Table 1: New Zealand leafy vegetable data for 2003

Crop	Growers (no.)	Planted area (ha)	Crop volume (tonnes)	Domestic sales value (2002) $million	Exports sales value 2003 (fresh) $million
Broc/Cabbage/Cauli	277	3,746	40,000	80.3	1.3
Lettuce	252	1,287	NA	39.1	1.0
Silverbeet/Spinach	103	396	4,000	13.4	INA

NA= Not available

3.2.2 Imported food

New Zealand import data for the year ending March 2003 identified some imports of fresh or chilled leafy vegetables. These are summarised in Table 2.

The sum of these imported leafy vegetables represents a very modest amount (approximately 0.2-0.3 g/person/day) compared with the New Zealand population overall consumption of approximately 27 g/person/day (see section 5.2).

Table 2: Imports of leafy vegetables into New Zealand, year ending March 2003

Food	Country of origin	Weight (tonnes)
Brussel sprouts	Australia	108.1
Cabbages	Australia	0.7
Lettuce	Australia	7.9
Spinach	Australia	1.8
Spinach	Fiji	0.5
Spinach	Belgium	9.9
Spinach	China	60.8
Spinach	Germany	0.2
Spinach	India	0.3
Spinach	Netherlands	98.7
Spinach	United States	87.0

3.2.3 Processing

Pre-cooling and relative humidity control of vegetables prior to packaging is important in prolonging shelf life. Typical commercial packaging notes (from an Australian website; http://www.peakfresh.com/index1.htm) are available and include information on the following vegetables; celery, cauliflower, broccoli, brussel sprouts, cabbages, Chinese cabbages, lettuce, mesclun, parsley, spinach and silverbeet. The information includes advice on post harvest temperatures, post harvest humidity, packaging method, storage temperatures and length of time stored under these conditions.

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 uraemic syndrome (HUS), thrombotic thrombocytopaenic purpura (TTP) and death (AIFST, 2003).

4.1 Symptoms

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

Diarrhoea Symptoms: Diarrhoea is accompanied by severe abdominal cramps. Vomiting may occur (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 Thrombocytopaenic purpura (TTP):* A version of HUS most often experienced by the elderly. Involves loss of platelets, skin coloration, fever and nervous system disorder (seizures and strokes) in addition to HUS signs and symptoms. There is no prior episode of diarrhoea. Illness lasts from 2-9 days.

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

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

4.2 Serotypes Causing Disease

In New Zealand, as in many other countries, *E. coli* O157:H7 is the serotype most commonly isolated from human cases. The importance of non-O157 serotypes as a contribution to human illness is difficult to assess, as information is limited. Overseas data suggest that these serotypes are increasingly recognised as a significant proportion of human infections.

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 means that true prevalence is uncertain. The World Health Organisation has identified the

most important non-O157 STEC serogroups, from an epidemiologic perspective, as O26, O103, O111 and O145 (WHO, 1998). See website http://www.who.int/emc-documents/zoonoses/docs/whocsraph988.html/3surveillanceandfrequency.html for table of serotypes of non-O157 STEC isolated from humans. Serotypes isolated from patients with HUS are highlighted.

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 the bacteriophage 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 suggests 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:H7 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

with type O111:H- being the most prevalent. (Park *et al.*, 1999).

4.3 Dose-Response

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

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

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

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

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.

4.3.2 Dose-response for non-O157:H7 STECs

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

5 EXPOSURE ASSESSMENT

5.1 The Hazard in the New Zealand Food Supply: STEC in Leafy Vegetables

5.1.1 STEC in leafy vegetables: O157:H7

Hydroponically-grown leafy vegetables have been surveyed in New Zealand (Graham and Dawson, 2002). A total of 114 samples of leafy vegetables (comprising lettuce and spinach) and 60 herb samples were tested and none of the samples contained *E. coli* O157:H7. However *E. coli* was detected in 16 (14%) samples of leafy vegetables and 3 (5%) samples of herbs. The authors note the presence of *E. coli* as an indicator of faecal contamination, suggesting the potential for pathogens such as *E. coli* O157:H7 to be present because of the similar characteristics and source.

A MAF policy project (see website; http://www.maf.govt.nz/mafnet/rural-nz/research-and-development/research-results/2002-2003/research-results-01.htm#P404_36490) was carried out between the months of February and May 2003. Samples of conventionally grown lettuce (240) and organically grown lettuce (234) were tested for *E. coli* O157:H7. Conventional lettuces (48 lots of 5 samples) were obtained from 22 growers from around New Zealand. Seven lettuce varieties were included, with Iceberg being the most popular in the market. All samples tested negative for pathogenic *E. coli* O157:H7 per 25 g of sample. The organic lettuces were purchased from 9 growers from around New Zealand. Forty-six lots of 5 samples and one lot of 4 samples were purchased, representing 13 varieties, with Iceberg, Fancy green, Green oak and Cos being the most popular. All samples were negative for *E. coli* O157:H7. However an atypical *E. coli* O157:H16 was isolated from one Fancy green lettuce with a count of 23 MPN/g. This isolate harboured the *eae*A adhesin gene, one of four recognised in pathogenic *E. coli* O157:H7. The *Stx1*, *Stx2* and *hly*A genes were absent. Verocytotoxin was not produced by this isolate. It was concluded that this isolate was of no public health significance as no verocytotoxin was produced.

Neither of these studies tested for non-O157 STEC serotypes.

5.1.2 STEC in leafy vegetables: other serotypes

No information was found regarding non-O157 STEC in leafy vegetables.

5.2 Food Consumption: Leafy Vegetables

The WHO GEMS/Food European regional diets (New Zealand is considered to fit into this group, see http://www.who.int/fsf/GEMS/index.htm) list consumption figures for leafy vegetables, as defined in this risk profile, of approximately 80 g/person/day.

Analysis of dietary records from the 1997 National Nutrition Survey (NNS) (Russell *et al*., 1999) by FSANZ (ANZFA, 2001) gave a considerably lower estimate of 26.6 g/person/day. Approximately 55% of this total is made up of leafy vegetables, as defined by Codex (see section 3 for definitions), and about 45% cabbage and Brussel sprouts. Herbs make up a tiny proportion of the total leafy vegetables consumed. Leafy vegetables are a commonly consumed food, with NNS data indicating that 39% of adults consume leafy vegetables in any given 24-hour period.

Consumption of leafy vegetables is lower for children than adults. Data from the 2002 Children's Nutrition Survey (2002CNS; Ministry of Health, 2003) showed that only 20% of children, aged 5-15 years, consumed leafy vegetables during any 24 hour period, with an average consumption of 10.7 g/person/day.

The Australian Nation Nutrition Survey (Australian Bureau of Statistics, 1999) contains two categories of food which are relevant to the current risk profile; 'cabbage, cauliflower and similar brassica vegetables' and 'leaf and stalk vegetables'. The average daily consumption of these two categories for the population 19 years and over was 38.6 g/person/day. Given that these food categories contain a number of foods that do not fit within the definition of leafy vegetables, the consumption of leafy vegetables in Australia is probably of a similar order of magnitude to consumption in New Zealand. The Australian data show similar age-related patterns to the New Zealand data, with leafy vegetables less likely to be consumed by younger consumer.

The data for New Zealand adults is very similar to published information for the USA population, which gives an average daily consumption for leafy vegetables, as defined in this risk profile, of 30.3 g/person/day for a 70 kg adult (EPA, 1997).

5.3 Qualitative Estimate of Exposure

5.3.1 Number of servings and serving sizes

The estimation of total number of servings of leafy vegetables consumed on a per annum basis involves a number of assumptions:

- That the sample set employed for the NNS is typical of the total population,
- That the results of the 24 hour dietary recalls are typical of the full 365 day period of one year, and
- That the consumption of leafy vegetables by the population less than 15 years of age will not be significantly different to that for the survey population (The NNS only surveyed people 15 years and older).

The FSANZ analysis of the data from the 1997 National Nutrition Survey (ANZFA, 2001) identified 978 respondents consuming a serving of herbs, 800 respondents consuming cabbage or Brussels sprouts, and 1536 respondents consuming leafy vegetables, principally lettuce (as defined by Codex- see section 3 for definitions). The usage pattern of herbs will be very different from other leafy vegetables. For the purposes of analysing serving number and size data, herbs will be considered separately from other leafy vegetables. Assuming a New Zealand total population of 4,054,200 (at 31 March 2004) (http://www.stats.govt.nz/): the total number of servings per annum would be:

Annual number of herb servings (total population) = 4,054,200 x 978/4636 x 365
= 3.1×10^8 servings per annum

The median serving size is 0.1 g, while the 97.5th percentile serving size is 7.4 g.

Annual number of leafy vegetable servings (total population) 4,054,200 x 2336/4636 x 365

$= 7.5 \times 10^8$ servings per annum

The actual annual number of servings of leafy vegetables consumed by the total population will be somewhat lower than this estimate, as children eat leafy vegetables less frequently than adults.

The number of servings derived from the FSANZ study (2336) were almost identical to the number derived from ESR's analysis of the same data (2266). From the ESR analysis 69% of servings would have included raw leafy vegetables, while the remaining 31% were cooked. These proportions are identical for adults and children. The mean serving size for leafy vegetables was 54 g for adults and 45 g for children. The 95th percentile serving sizes were 162 g and 145 g, respectively for adults and children.

5.3.2 Frequency of contamination

The available data suggest that contamination of leafy vegetables by *E. coli* O157:H7 is a rare occurrence (section 5.1.1).

5.3.3 Predicted contamination level at retail

No quantitative surveys of *E. coli* O157:H7 were found.

5.3.4 Growth rate during storage and most likely storage time

Since the minimum temperature for growth of STEC is 7^{o}C, produce which is refrigerated should not support growth. However, when temperatures are within the growth range, the organism has been shown to be able to grow on leafy vegetables. By observation, the conditions under which leafy vegetables are stored by retail outlets are highly variable. The relatively short normal shelf lives of leafy vegetables will tend to limit the amount of growth that occurs prior to consumption. The introduction of modified atmosphere storage and vacuum packing to extend shelf life may affect the potential risk.

5.3.5 Heat treatment

This is not applicable to leafy vegetables, such as lettuce, but may be for others like brassicas. However present consumer trends would suggest that any of these foods might be eaten raw (e.g. cabbage in coleslaw) or lightly cooked (e.g. steam cooking spinach etc).

5.3.6 Exposure summary

From the results of surveys reported in section 5.1.1, pathogenic STEC have not been detected in leafy vegetables in New Zealand. Therefore the exposure of the population is likely to be very low.

5.4 Overseas Context

5.4.1 STEC in Leafy Vegetables: O157:H7

Information summarising data for the prevalence of *E. coli* O157:H7 in leafy vegetable products is given in Table 3. All studies except one failed to detect *E. coli* O157:H7 in the products tested, which comprise mostly lettuces. The one study where detections were made was in Mexico where lapses in hygiene have led to numerous instances of foodborne disease in destination markets of Mexican produce. It can be concluded that *E. coli* O157:H7 is a rare contaminant of leafy vegetables produced in countries similar to New Zealand.

Table 3: Prevalence of *E. coli* O157:H7 in vegetables from overseas surveys

Country	Products tested	Number tested	No. (%) positive	Year of publication
Greece	Lettuce-containing sandwiches	61	0	Dontorou *et al*., 2003
Mexico	Cabbage Cilantro Coriander	4 41 10	1 (25.0) 8 (19.5) 2 (20.0)	Beuchat, 1996
Norway	Lettuce Herbs Parsley/dill	200 130 100	0 0 0	Johannessen *et al*., 2002
Spain	Raw lettuce Ready-to-eat lettuce	40 40	0 0	Soriano *et al*., 2001
United Kingdom	Imported whole lettuces	151	0	Little *et al.*, 1999
United Kingdom	Organic ready-to-eat vegetables: Cabbage Cress Lettuce Watercress Other (includes spinach, leeks, shallots, chard)	 159 12 415 65 208	 0 0 0 0 0	Sagoo *et al*., 2001
United Kingdom	Unwrapped salad vegetables	2950	0	Sagoo *et al*., 2003a
United Kingdom	Bagged ready-to-eat salad vegetables	3820	0	Sagoo *et al*., 2003b
USA (imported)	Cilantro Lettuce Parsley	177 116 84	0 0 0	http://www.cfsan.fda.gov/~dms/prodsur6.html
USA (domestic)	Cilantro Lettuce Parsley	85 142 90	0 0 0	http://www.cfsan.fda.gov/~dms/prodsur10.html

6 RISK CHARACTERISATION

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

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 has increased steadily since 1994. The rates are shown in Table 4. The trend over the period 1995-2004 is also shown in Figure 2.

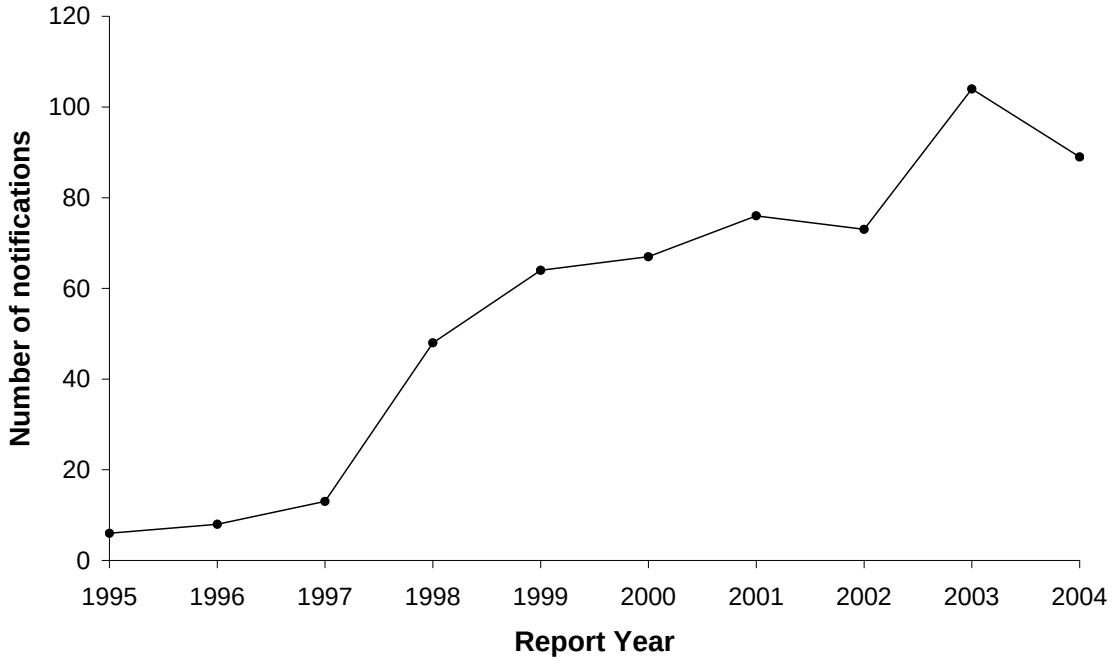
The year 2003 has the highest notification rate in a single year and is more than double the rate of 1998.

Table 4: Rates of infection with STEC in New Zealand 1998 – 2004

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

Note that these rates are for all STEC. Serotype O157:H7 accounts for around 97% of the notified cases in 2003 and 91.5% in 2004.

Figure 2: STEC notifications by year, 1995 –2004



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.

Notification rates 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 per 100,000.

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. In the elderly populations 60-69, there were 5 cases; 1.8 and in the 70+ age group, 4 cases; 1.2 (ESR, 2005a).

Based on studies in Canada, in New Zealand it has been assumed that 10-12 cases of STEC infection occur for each reported case (Baker *et al*., 1999). This would equate to 890 to 1068 cases in 2004 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 5.

Table 5: Summary of clinical consequences of STEC infection in New Zealand

Period	Hospitalised*	HC*	HUS*	TTP*	Fatalities	Reference
Oct 93-Dec 98	24/58 (41.4%)	21/59 (35.6%)	18/59 (30.5%)	1/59 (1.7%)	2/79 (2.5%)	Baker *et al.*, 1999
1999	20/60 (33%)	NS	2/64 (3.1%)	NS	0	Kieft *et al.*, 2000
2000	11/65 (16.9%)	NS	3/68 (4.4%)	NS	0	Lopez *et al.*, 2001
2001	16/74 (21.6%)	NS	6/76 (7.9%)	NS	0	Sneyd *et al.*, 2002
2002	16/64 (25.0%)	NS	5/73 (6.8%)	NS	0	Sneyd and Baker, 2003
2003	24/99 (24.2%)	NS	4/105 (3.8%)	NS	0	ESR 2004a
2004	27/84 (32.1%)	NS	3/89 (3.4%)**	NS	0	ESR, 2005a

* Percentages are determined on the basis of cases for which information was available
** A further one case with HUS was reported but was not notified or laboratory confirmed
NS Not stated

6.1.3 Serotypes causing disease in New Zealand

Of the 89 notified cases in 2004, 82 were confirmed STEC isolates, 75 (91.5%) were caused by *E. coli* O157 ((ESR, 2005a). Other serotypes that have caused infections over recent years include;

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

ORough:HNM.

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

Some isolates causing infection have not been typable e.g. ONT:H-. The H- isolate is motile but no H factor could be found. HNM is an isolate which is non-motile so no H factor can be proven.

There have been two deaths attributed to STEC; these were in the period 1993 – 1998. One was attributed to serotype O157:H7 and the other to O113:H21. The New Zealand isolates of STEC that have caused infection have all possessed the genetic virulence factors in addition to either or both *Stx* genes (Carolyn Nicol, ESR Enteric Reference Laboratory, personal communication, March 2005).

The most prevalent serotype in Australia (O111:H-) is absent from the most common serotypes in New Zealand (see section 4.2.2 above).

Stool specimens (n=484) from children suffering from diarrhoea submitted to the Dunedin 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 *Stx*1, *hly*A and *eae*A genes, while the others (O128:H2, OR:H2, OR:H-) were positive for the *Stx*1 and *Stx*2 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). O128:H2 and O128:H- serotypes have been isolated from both meat and a person suffering from diarrhoea, although they were not notified cases of STEC infection.

6.1.4 Outbreaks

The reported number of outbreaks and cases which STEC was a causative agent between 1998 and 2004 are presented in Table 6.

Table 6: Total number of reported outbreaks and cases for which STEC was identified as the causative agent in New Zealand 1998-2004

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

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

In terms of overall pathogens involved in outbreaks in 2004, STEC outbreaks were 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 that none of the STEC outbreaks listed above were associated with leafy vegetables.

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

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

There have been a few episodes where indistinguishable STEC isolates have been isolated from both a human case and a potential transmission route in New Zealand. Contaminated untreated drinking water (one spring and one roof supply) was linked to two episodes of infection, affecting a total of three people in 1999, and one case has been attributed to contact with a calf (Anonymous, 2000). For one case in New Zealand, an indistinguishable isolate was obtained from both the infected person and raw milk present in the home, although the actual route of infection is uncertain (Anonymous, 2002).

6.2 Adverse Health Effects Overseas

6.2.1 Incidence

Incidence data for a selection of countries/states are given in Table 7. New Zealand's incidence has been included for comparison and is similar to other countries. The incidence of infection is however considerably higher in the Czech Republic and considerably lower in Australia. The Scottish rate has significantly declined from 8.23 to 2.9 per 100,000 from 1997 to 2003 (PHLS, 2000).

Table 7: New Zealand and international rates of reported infections with STEC

Country	Year	Incidence (per 100,000)	No. of lab. confirmed cases	% O157	% Other VTEC	Reference
New Zealand	**2003**	**2.8**	**94**	**97**	**3**	**ESR, 2004a**
New Zealand	**2004**	**2.4**	**82**	**92**	**8**	**ESR, 2005a**
Australia[#]	2002	0.3	53	-	-	Yohannes *et al.*, 2004;
Australia (cont.)	2003	0.2 (SA 2.4[*] Qld 0.2	49 (-37 -6	25	15 O111	Miller *et al.*, 2005

Country	Year	Incidence (per 100,000)	No. of lab. confirmed cases	% O157	% Other VTEC	Reference
		Vic 0.1 WA 0.2)	-3 -3)			
Europe Community (17 member states + Norway	2004	1.3	4143	50	25[1]	EFSA, 2005
Austria	2004	0.6	45	29	71	EFSA, 2005
Belgium	2004	0.3	36	56	44	EFSA, 2005
Czech Republic	2004	17.1	1743	18	0	EFSA, 2005
Denmark	2004	3.0	163	27	73	EFSA, 2005
Finland	2004	0.2	10	40	60	EFSA, 2005
Germany	2004	1.1	903	10	42[1]	EFSA, 2005
Ireland	2004	1.4	57	88	12	EFSA, 2005
Netherlands	2004	0.2	30	100	0	EFSA, 2005
Norway[2]	2004	0.3	12	58	42	EFSA, 2005
Poland	2004	0.2	81	99	1	EFSA, 2005
Sweden	2004	1.7	149	-	-	EFSA, 2005
United Kingdom	2004	1.5	898	99	1	EFSA, 2005
(Scotland[3])	2003	2.9				SCIEH, 2004
North America						
Canada	1999	4.9				Health Canada (2000)
Canada	2000	8.8				Health Canada (2000)
USA[3]	2004	0.9				Centers for Disease Control &

Country	Year	Incidence (per 100,000)	No. of lab. confirmed cases	% O157	% Other VTEC	Reference
						Prevention 2005

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

The USA health objective for 2010 for infection with *E. coli* O157 is 1 per 100,000. The proportion of STEC infected cases hospitalised in the United States has been estimated as 29.5%, with 0.8% of cases resulting in death (Mead *et al*., 1999). Although New Zealand's hospitalisation and fatality rates to the end of 1998 were higher than this, there have been no deaths due to STEC since 1999 (see Table 5). 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).

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).

6.2.2 Contributions to outbreaks and incidents overseas

The proportion of outbreaks caused by *E. coli* O157:H7 overseas is summarised in Table 8. This illustrates that only a small proportion of outbreaks are attributable to STEC.

Table 8: Proportions of outbreaks and incidents overseas caused by *E. coli* O157:H7

Country	Year	Proportion of outbreaks (%)	Reference
New Zealand	**2004**	**0.9**	**ESR, 2005b**
Canada	1982	0.2	Todd, 1992
Canada	1983	0.2	Todd, 1992
Canada	1984	0.1	Todd, 1992
England and Wales	1992-1994	1	Djuretic *et al*., 1996
England and Wales	1995	1	Evans *et al*., 1998
England and Wales	1996	1.4	Evans *et al*., 1998
Sweden	1992-1997	<1	Lindqvist *et al*., 2000

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

The Food Safety and Inspection Service (FSIS) of the USDA risk assessment for *E. coli* O157:H7 in ground beef summarised information from 154 *E. coli* O157:H7 outbreaks during the period 1982-1997 (FSIS, 1998). Ground beef was identified as the likely vehicle

for infection in 25% of outbreaks, while whole cuts were identified with only 2% of outbreaks and salami with less than one percent.

An analysis of outbreaks in England and Wales attributed to the consumption of salad vegetables and fruit found that two (2.4%) involved VTEC O157 as the aetiological agent. No details are given as to the exact nature of the food involved (Long *et al*., 2002).

Data from the USA show that the category "vegetables, salad bars" was the vehicle in 5.9% of *E. coli* O157:H7 outbreaks between 1982 and 1994 (Doyle *et al*., 1997).

Table 9 lists specific incidents linked to leafy vegetables.

Table 9: Specific Incidents of Disease Reported for *E. coli* O157:H7 Associated with Leafy Vegetable Products

Location	Setting	No. affected	No. deaths	Source	Reference
Canada	Hospital	23	0	Iceberg lettuce	Sewell and Farber, 2001
Sweden	Community	120 (7 HUS)	0	Iceberg lettuce, OR 13. Implicated crop irrigated by stream	Söderström *et al.,* 2005
USA, California	Restaurant (mainly)	20	0	Salad mix	Promed, 9/10/2003
USA, Connecticut and Illinois	Community	>61	0	Mesclun lettuce	Hillborn *et al*., 1999
USA, Maine	Boy scout camp	30	NS	Lettuce	Tauxe *et al*., 1997
USA, Maine	Community	4	1	Manured vegetables*	Cieslak *et al*., 1993
USA, Montana	Community	92 possible, 40 confirmed	0	Lettuce	Ackers *et al*., 1998
USA, Nebraska	Restaurant	72	NS	Iceberg lettuce	Wachtel and Chaskowski, 2002
USA, Ohio	Restaurant chain	46	NS	Coleslaw	Wu *et al*., 2002; Wachtel and Charkowski, 2002
USA, Washington	Cheerleading camp	50 (29 confirmed)	NS	Romaine lettuce in caesar salad	Promed: *E. coli* O157, lettuce-USA: Alert 200207304893

NS=Not Stated. * Nature of vegetables not defined.

In 1996, the largest known outbreak of *E. coli* O157 infection occurred in Japan, spread over several districts. Over 2-3 months, more than 9000 people (mainly school children) were reported as affected, the number of confirmed cases is uncertain and the episode may be a

series of outbreaks. The source of the infection was not identified although radish sprouts were implicated in some of the cases (WHO, 1996).

No specific incidents of disease were found for non-O157 STEC associated with leafy vegetable products.

6.2.3 Case control studies overseas

Published studies identifying consumption of leafy vegetable products as risk or protective factors from overseas are few. A study by Wachtel *et al*., (2002a) on cabbage plants found that an accidental release of tertiary-treated sewage (no chlorine treatment) on the plants lead to *E. coli* strains (not containing *Stx1*, *Stx2* or *eae* genes) being associated with the plant roots. Control fields of cabbages did not have the bacteria.

6.2.4 Risk assessments and other activity overseas

6.2.4.1 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, 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.

There were five recommendations made in relation to salads/vegetables;

- Clear labelling/instructions for ready to cook/eat salad vegetables were required, supported by regulations,
- Growers of salads/vegetables to be eaten raw should be advised on the correct handling of organic wastes,
- Adequate training to be made available and undertaken by all of those involved in the preparation, handling and distribution of salad and vegetable crops,
- All salad/vegetables to be consumed in their raw state, even though pre-washed, should be washed prior to consumption,
- An education programme be targeted at smaller businesses, caterers and consumers on the need for a high standard of personal hygiene and for effective washing of all "raw" salad and vegetable products prior to sale and consumption, (to include procedures for sprouted seeds).

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 salads/vegetables recommendations by including these key messages in the Agency's ongoing "Food Hygiene" Campaign for commercial and domestic audiences and updating guidance notes on existing food labelling requirements.

The *E. coli* taskforce report is available at website;
http://www.foodstandards.gov.uk/scotland/fsascotwork/ecolitask, and the FSA/Scottish Exec *E. coli* action plan is available at website;
http://www.foodstandards.gov.uk/multimedia/pdfs/rrec.pdf.

6.2.4.2 Codex Risk Profile on EHEC

A Risk Profile (FAO/WHO, 2002) on EHEC (including identification of commodities of concern, mainly sprouts, ground beef and pork) is a priority item of work for the joint FAO/WHO Food Standards Programme. A discussion paper (and revised Risk Profile in Appendix A) has been produced (FAO/WHO, 2004) which can be found at the following website; ftp://ftp.fao.org/codex/ccfh36/fh0410be.pdf.

Page 10 (Table 3) of the FAO/WHO, (2002) Risk Profile, lists Risk Assessments for *E. coli* O157:H7. There are eleven in total, of which nine are associated with animal/meat products. Two risk assessments, both Canadian, cover;

- seeds/beans and sprouted seeds/beans, and
- unpasteurised fruit juice/cider.

Under the heading of Data Gaps, fresh leafy vegetables are noted as being of concern due to potential contamination by bovine faeces as opposed to feral animal or human faeces, and the Commission recommended a farm-to-table risk assessment for ground beef and leafy green vegetables. The recommendation includes an on-farm module in order to assess the impact of various manure control strategies on cases of human *E. coli* O157:H7 illness. Information from this study could then be used to amend existing guidance documents or annexes developed. It is suggested that one document that could make use of this information is the "Draft Code of Hygienic Practice for Fresh Fruit and Vegetables" (Appendix II).

6.2.5 Secondary transmission

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

6.3 Qualitative Estimate of Risk

There are few data on the prevalence of the predominant serotype; O157:H7 in New Zealand in relation to leafy vegetables. The two surveys carried out (details in section 5.1.1) did not detect *E. coli* O157 in hydroponic vegetables or lettuce.

Little information on transmission is available from the analysis of cases in New Zealand between 1993 and 1998 (Baker *et al*., 1999) or risk factor information (ESR, 2004a). The cases are more common in rural areas suggesting that environmental or animal exposure may be important.

There is currently no information to indicate that transmission of STEC via leafy vegetables is occurring in New Zealand. Available information indicates that the risk is low.

6.4 Risk Categorisation

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

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

For the purposes of estimating the numbers of cases of foodborne disease in New Zealand (Lake *et al*., 2000) it was assumed that 20% of STEC infections were due to foodborne transmission. The total rate of STEC infection (including unreported cases) attributable to food contamination in New Zealand was thus estimated to be of the order of 1.4 per 100,000 of population.

With no evidence linking leafy vegetable consumption to cases of STEC infection in New Zealand, the rate of STEC infection due to transmission in leafy vegetables will be considerably less than 1 per 100,000 of population. This places STEC in leafy vegetables in the lowest incidence category.

6.5 Summary

Food/hazard combination	Severity	Incidence	Trade importance	Other considerations
STEC in leafy vegetables	1 (>5% serious outcomes)	4 (<1 per 100,000)	High (control essential)	Incidents attract adverse media attention

7 RISK MANAGEMENT INFORMATION

7.1 Relevant Food Controls: New Zealand

All food for sale in New Zealand must comply with the Food Act 1981.

7.1.1 The Fresh Produce Industry's Approved Supplier Programme

The NZ Vegetable and Potato Growers' Federation (Vegfed) developed the Approved Supplier Programme and launched it in 1999. This programme is designed to provide retailer and consumer knowledge about food safety and quality. The NZ Fruitgrowers' Federation (NZFF) also recognised the benefits of a single industry programme and responded to the same consumer concerns, joining the Programme in 2000, to create the NZ Fresh Produce Approved Supplier Programme (www.approvedsupplier.co.nz). Vegfed and NZFF have now joined to form Horticulture New Zealand (www.hortnz.co.nz).

The implementation of the NZ Fresh Produce Approved Supplier Programme was viewed by the industry as a proactive move by New Zealand growers to address consumer concerns relating to food safety and quality. It was also a pre-emptive move to address a 1996 amendment to the Food Act, which allowed for food retailers to operate food safety programmes in place of inspections by Public Health Officers.

Following an initial training workshop, suppliers are assessed by AgriQuality on an annual basis. This Approved Supplier Programme is intended to cover vegetable and fruit growers and the supply chain participants; transport, inputs (e.g. agrichemicals and fertiliser) and wholesalers. AgriQuality NZ is the training and auditing body in the Approved Supplier programme.

The programme, www.approvedsupplier.co.nz, defines twelve assessment criteria,. These are:

- Product and staff safety. Systems and documentation are in place to ensure safety of both product and staff (e.g. OSH, GROWSAFE).
- Quality control. There is monitoring of product, and recording against specification to maintain the integrity of the system.
- Product identification and traceability. The packaging of the finished product is clearly identified. Quality Assured Product must be traceable so investigations can be readily facilitated if required and records of product identification and product destination will be maintained by the Approved Supplier.
- Product management. Crops are grown and harvested in accordance with sustainable production practices. All stages of the production process are controlled to meet the customer's needs.
- Complaints/corrective action. Approved Suppliers have systems in place to address complaints and take appropriate action to reduce the likelihood of the problem occurring again.
- Independent assessments. Independent assessments are carried out on Approved Suppliers on an annual basis.
- Internal assessments. Approved Suppliers complete an internal assessment of their operation at least once a year to ensure their systems are effective, and product safety and quality is maintained.

- Handling, packaging, storage and delivery. Produce is handled, packaged, stored and delivered, in such a way so that damage, mix-ups or improper use is minimise.
- Training. Those carrying out the Approved Supplier Programme are properly trained to protect the integrity of the programme. The Programme ensures people who carry out tasks during processes critical to food safety and quality are trained to do the task properly.
- Records and documentation. Records and documentation are maintained to verify the integrity of the operation.
- Management Commitment. All Approved Suppliers have shown their commitment to quality assurance by displaying a Quality Statement. This statement will include quality goals and aims for the operation.
- Purchase of goods and services. Systems and documentation ensure purchased items such as seeds and fertilisers meet agreed specifications.

7.2 Relevant Food Controls: Overseas

This section collates information on the regulatory regimes overseas.

The Codex Committee on Food Hygiene (CCFH) has prepared a Code of hygienic practice for fresh fruits and vegetables (at Step 8 of the procedure, see report from the October 2001 meeting, reported by Codex in 2003 at: ftp://ftp.fao.org/codex/alinorm03/al03_13e.pdf). This Code includes annexes for "Ready-to-eat Fresh Pre-cut Fruits and Vegetables" and "Sprout Production". The Code was developed in response to growing concerns that fruits and vegetables were sources for foodborne pathogens. The Code addresses Good Agricultural Practices (GAP) and Good Manufacturing Practices (GMP). These practices should help to control microbial hazards from primary production to packing. The following areas of importance for microbial control are acknowledged;

- Environmental hygiene,
- Hygienic production;
  - Water,
  - Manure,
  - Soil,
  - Agricultural chemicals,
  - Biological control,
  - Indoor facilities, and
  - Personal hygiene,
- Handling,
- Storage,
- Transport,
- Cleaning,
- Maintenance, and
- Sanitation.

No microbiological specifications are given in the Code; instead the Code refers to the Codex Recommended International Code of Practice – General Principles of Food Hygiene (http://www.fao.org/DOCREP/005/Y1579E/y1579e02.htm#bm2).

7.2.1 Europe

The EU Regulation 80/778/EEC requires potable water to be used in food production except for water which does not come into contact with food such as firefighting water.

The EU and USA have recognised the potential for organic production systems to pose risks through the use of animal manure. The EU governs organic food production through a strict EC Council Regulation 2092/91, see website; (http://europa.eu.int/eur-lex/en/consleg/pdf/1991/en_1991R2092_do_001.pdf). The Regulation came into force in 1993 and sets out the inputs and practices which are permitted in organic farming together with the inspection regime to ensure this. All foods sold as organic must originate from growers, processors and importers registered with an approved certification body and they are subject to regular inspections.

The UK implemented Regulation 2092/91 initially in the form of UK Register of Organic Food Standards (UKROFS) which are due to be replaced by a Compendium of UK Organic Standards at a date yet to be announced. The Advisory Committee on Organic Standards (ACOS) set up in 2003 has now superceded UKROFS. More information on ACOS can be found at the following website; http://www.defra.gov.uk/farm/organic/acos/

The application of manure to ready-to-eat crops during the growing season is prohibited. Draft guidelines have been produced in the UK regarding the use of farm manures and food safety; http://www.foodsafetynetwork.ca/food/managingfarmmanures.pdf. With respect to wash water, a ruling by UKROFS has prohibited the use of enhanced levels of chlorine (i.e. above normal "town's water") for washing ready-to-eat fresh produce as is common practice in non-organic ready to eat salad and vegetable processing. The organic industry now uses replacement products based on organic acids (IFST, 2003). This issue was identified by the Institute of Food Science and Technology (IFST) see website: http://www.ifst.org/hottop24.htm

In addition the IFST recommend;

- "Whole fruit and salad vegetables (whether organic or non-organic) for consumption without cooking should be thoroughly washed before consumption;
- all retailers should provide in-store advice to that effect and it should be printed on the packaging of consumer pre-packs of whole fruit and salad vegetables".

Following the investigation by Professor Hugh Pennington (1998) into the circumstances surrounding the 1996 *E. coli* O157:H7 outbreak in Central Scotland and subsequent recommendations, a number of actions falling into four main categories followed; namely

- Stricter hygiene and enforcement measures,
- Enhanced Surveillance,
- Research, and
- Improved handling in control of outbreaks.

7.2.2 North America

The USA has introduced a national programme aimed at developing standards for organic foods (http://www.nal.usda.gov/afsic/ofp/). Essentially farmers are not permitted to apply raw manure within 120 days of harvest, or must use manure which has been composted to kill pathogens.

The US government has also been active in issuing "guidance" to the produce industry. This was the result of (then) President Clinton's "Initiative to Ensure the Safety of Imported and Domestic Fruits and Vegetables". In 1998 the FDA produced "Guidance for Industry-Guide to minimise microbial food safety hazards for fresh fruits and vegetables" (http://vm.cfsan.fda.gov/~dms/prodguid.html). This document covers: manure and municipal biosolids, worker health and hygiene, sanitary facilities, field sanitation, packing facility sanitation, transport and traceback.

Similar documents have been produced by the Canadian Food Inspection Agency (http://www.cfia-acia.ca/english/plant/fresh/read-eat.html).

These documents all deal, in terms of microbiological hazards, with the prevention of contamination of the food. This includes both prevention of contamination by animal faeces, and human faeces through, for example, the provision of adequate toilet and handwashing facilities for workers at all stages of the supply chain.

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.

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 varied between $26 for no medical care required, to $6.2 million for a case who died from HUS (Frenzen *et al*., 2005).

These figures are high in comparison with New Zealand as they include productivity losses due to chronic illness caused by STEC infection, which were not included in the New Zealand estimate. The US 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

In addition to the Approved Supplier Programme (discussed in Section 7.1.1) there is one Standard and several guidelines for the use of fertiliser materials on farms growing leafy vegetables. These will all contribute to the prevention of STEC contamination:

- New Zealand Standard for compost, soil conditioners and mulches,
- Guidelines for the application of biosolids to land, and
- Guidelines for utilisation of sewage effluent on land.

7.4.1.1 Composts, soil conditioners and mulches

Composts, soil conditioners and mulches are regulated by Standard NZS 4454: 2005. This focuses on the composition, compliance, sampling and testing methods. *E. coli* or faecal indicators must be less than 100 MPN/g to ensure microbiological quality.

7.4.1.2 Biosolids

Biosolids are sewage sludges or sewage sludges mixed with other materials. This does not include untreated raw sewage sludges, sludges from industrial processes, animal manures, food processing or abattoir wastes. Septic tank sludges may become biosolids depending on their level of treatment. The treatment that biosolids receive must enable safe, beneficial application to land.

Guidelines for the application of biosolids to land were recently published by the New Zealand Water and Wastes Association and approved by the Ministries of Environment, Health, and Agriculture and Forestry (NZWWA, 2003). These new guidelines partly supercede the Department of Health's Public Health 1992 Guidelines relating to sewage sludge application to land (Department of Health, 1992).

There are two grades of biosolids, Grade A and Grade B. Grade A relies on accredited quality assurance plus one of the following pathogen reduction processes from the following options, (refer to the NZWWA, 2003 for details);

1. Time/temperature process
   - ≥ 7% dried solid,
   - < 7% dried solid,
   - Composting
     - In-vessel
     - Windrow, minimum of 5 turnings

2. High pH - high temperature process

3. Other processes - agreed comprehensive process demonstrates that Grade A pathogen levels can be consistently met.

Grade B relies on verified quality assurance and storage. For salad crops that may be eaten unpeeled or uncooked, further recommended controls are soil incorporation and a further waiting period of at least 1 year before crops are sown (in the meantime, the land may be utilised for other purposes).

Standards have been set for faecal coliforms in biosolids but not specifically STEC. For *E. coli*, there must be less than 100 MPN/g and 100% compliance is required.

7.4.1.3 Sewage effluent

New Zealand guidelines for the utilisation of sewage effluent on land (New Zealand Land Treatment Collective, 2000) refer to the Public Health Guidelines (Department of Health, 1992). The recommended microbiological guidelines for the irrigation of sewage effluent on Category I salad crops is <10 faecal coliforms per 100ml. The typical treatment requirements are "conventional" biological oxidation or equivalent with tertiary disinfection. The guidelines also state no harvesting of crops when wet with irrigated water.

Outside of New Zealand, the recent suggestion by Scottish researchers that slugs are a novel vector of *E. coli* O157 (Sproston *et al*., 2006) may be an area for further research in New Zealand.

7.4.2 Ruminant faecal contamination

Preventing ruminant faecal contamination has been recognised as a priority. 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) have highlighted 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.

How manure can be treated or incorporated into the ground has also been researched. 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).

7.4.3 Consumer advice

There is a general consensus that leafy vegetables (organic or non-organic) should be washed thoroughly by the consumer before consumption, particularly if eaten raw. The NZFSA website gives advice to consumers on fresh produce preparation;

> **"Washing fresh produce before use** is the most effective way of minimising the risk of foodborne illness. This will reduce the presence of surface pathogens and any residues left on the food surface.
> Always use high quality drinking water for preparing food and washing up."

http://www.nzfsa.govt.nz/consumers/food-safety-topics/recalls-and-product-advice/fresh-produce/index.htm.

7.5 Other transmission routes

Other transmission routes include;

- other foods
- non-reticulated water supplies,
- recreational contact with water,
- animal contact,
- contact with children in nappies,
- contact with sewage,
- secondary infection from another case.

There are limited data concerning the risk of STEC infection from other foods in New Zealand, all of which is related to meat. Data from the National Microbiological Database indicate that to September 2001, from 113,890 samples of bulk meat for export, only two samples (0.002%) were positive for O157:H7 (Dr Roger Cook, personal communication). The figure is extremely low when compared to overseas surveys (Lake *et al.*, 2002).

Retail raw meat samples (91 beef, 37 mutton or lamb, 35 pork, 36 chicken, 10 mutton/beef mince, and 9 sausage mixture) were tested for STEC in Dunedin (Brooks *et al.*, 2001). Serotype O157:H7/H- was not isolated from any of the meat samples. A number of non-O157 STEC were isolated from beef, lamb/mutton and pork samples, but not chicken. One isolate, *E. coli* O128:H2 from beef mince, was the same serotype isolated from faeces of two children with diarrhoea in Dunedin.

Another study in New Zealand (Bennett and Bettelheim, 2002), collated isolates of STEC (none of which were O157) from bovine and ovine meat from the South Island and compared the isolates with those from humans and meats in other parts of the world. At least seven of the STEC serotypes isolated from New Zealand meats were types which have been associated with human disease in different parts of the world, including New Zealand.

Two published surveys have evaluated the prevalence of STEC in New Zealand bovines. Buncic and Avery, (1997) sampled the faeces of 371 cattle from 55 farms on arrival at a single slaughterhouse in the Waikato area. Two (0.54%) of these samples yielded *E.coli* O157 which is significantly lower than overseas data. A further 160 cattle from the farm of one of the positive animals tested negative for *E. coli* O157:H7. In a more recent report (Cookson *et al*., 2003), no *E. coli* O157:H7 was detected in faecal samples in healthy cattle and sheep in the lower North Island of New Zealand. However of the 189 cattle samples, non-O157 STEC was found in 51 (27%). Of the 132 sheep samples, 65% contained STEC. Among the isolates detected were several clinically important serotypes such as O5:H-, O26:H11, O84:H-/H2, O91:H- and O128:H2.

8 CONCLUSIONS

8.1 Description of Risks to New Zealand Consumers

8.1.1 Risks associated with leafy vegetables

The current rate of STEC infection in New Zealand is similar to that of overseas countries at 2.4 notified cases per 100,000 population (89 cases). It is higher than the USA, UK or Australia, but lower than Canada.

All New Zealand cases appear to be sporadic; no common source outbreaks have yet been detected, although household clusters have been identified. Information on transmission routes is very limited, with little indication of foodborne transmission, and none implicating leafy vegetables.

E. coli O157:H7 can grow on lettuce at higher temperatures, while at refrigeration temperatures (5^{o}C or less) the organism is stable or declines slowly. Although overseas surveys of leafy vegetables have not found *E. coli* O157:H7, apart from one in Mexico, a number of outbreaks of infection implicating leafy vegetables (mostly lettuce) as a vehicle have occurred, principally in the US. However, there is little evidence to suggest that leafy vegetables represent an important risk for transmission of pathogenic STEC in New Zealand. Limited surveys of leafy vegetables in New Zealand have failed to find *E. coli* O157:H7. Although other STEC serotypes were not analysed. and the risk from these other serotypes needs to be assessed, *E. coli* O157:H7 is the predominant serotype infecting people in New Zealand.

Leafy vegetables are frequently consumed by the adult population and approximately 69% of the consumption is in a raw form. Imported leafy vegetables are apparently a very small component of the New Zealand market. Risk management for STEC contamination of leafy vegetables focuses on prevention of faecal contamination. New Zealand producers, via their industry organisation, Horticulture NZ, have an Approved Supplier Programme that limits the use of human (biosolids) or animal waste as fertilizer to material that has been subjected to a controlled composting process designed to eliminate pathogens. In addition, New Zealand Water and Wastes Association (NZWWA, 2003) have guidelines on the safe application of biosolids to land in New Zealand.

8.1.2 Risks associated with other foods

The main vehicle implicated in foodborne outbreaks of STEC infection overseas 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 (milk, apple juice, cider – particularly where 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 leafy vegetables compared with other foods as a vehicle in New Zealand.

8.1.3 Quantitative risk assessment

The main barrier to a comprehensive risk assessment is the limited data on the prevalence of contamination by STEC of New Zealand leafy vegetables at the retail level (or at other points in the production chain), and the absence of data concerning the numbers of STEC present. Further, there is no information from human surveillance studies to link leafy vegetables with cases so far detected in New Zealand, and therefore no means to validate a QRA model. Current methodology will need to be improved to provide the same sensitivity for broad screen STEC detection techniques as are available for specific *E. coli* O157:H7 methods.

The relative importance of foodborne transmission of STEC in New Zealand is unclear from the information gathered on cases to date. Data from overseas indicates that STEC has not been detected in leafy vegetables (apart from Mexico) and consequently a quantitative risk assessment may not be warranted.

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 prevent the likelihood of foodborne transmission.

The FAO/WHO (1998) review found that environmental exposure factors lead to the main differences in microbial loadings and has led to a consensus that preventing contamination with ruminant faeces is a priority in the first instance. Where organic methods of farming are used and ruminant faeces are applied to farmland, both the EU and the USA have put into place various safeguards such that the application of fresh faeces is prohibited and that manure has been properly composted (specific timespans, internal temperatures, etc.) before being applied.

The control measure of decontaminating the leafy vegetables appears to be less effective with the results of various disinfection treatments and temperatures often in contradiction of one another. There is however consensus that exposure of greater than 60 seconds to a disinfectant wash with chlorine has no further significant effect.

A further complication in the decontamination issue is the suggestion that the pathogen can be internalised via the roots into the plant tissue itself thus evading decontamination altogether.

8.3 Data gaps

The data gaps identified in this Risk Profile are:

- Information on transmission routes for STEC infection in New Zealand,
- Current prevalence of STEC (not just *E. coli* O157) in leafy vegetables available in New Zealand,
- Data on numbers of STEC in leafy vegetables when contamination does occur, and
- Information on the market size and market structure for leafy vegetables, including consumption patterns in at risk groups.

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APPENDIX 1: CATEGORIES FOR RISK PROFILES

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

1. Incidence

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

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

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

* not recalculated.

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

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

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

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

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

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

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

2. Severity

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

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

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

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

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

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

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

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

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

Severity category 1:

Bacteria

Clostridium botulinum

Protozoa

Toxoplasma

Severity category 3:

Bacteria

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

Viruses

Others (e.g. rotavirus)

Protozoa

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

Proposed Category Matrix

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

Alternatives:

No evidence for foodborne disease in New Zealand

No information to determine level of foodborne disease in New Zealand