RISK PROFILE:
CAMPYLOBACTER JEJUNI/COLI
IN
RED MEAT

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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 *Campylobacter* in red meat (beef, pork, and sheep meat).

In New Zealand, there have been few red meat surveys published that give data on the prevalence of *Campylobacter*. Work carried out in the early 1980s reported a prevalence of *C. jejuni* on unweaned veal carcasses of 10%, and boneless veal of 13.3%. A retail survey of ground beef in 1984 did not detect any *Campylobacter* in the 50 samples collected from 27 outlets over a two month period. More recently, a retail survey of 100 raw hamburger patties and 100 fresh raw sausages in Christchurch in 2002 was also unable to detect *Campylobacter*.

From July 2003 to June 2004, ESR carried out a national survey of retail raw minced/diced red meats. This survey found 8/230 (3.5%) samples of beef, 9/90 (10%) samples of bobby veal, 16/231 (6.9%) samples of lamb/mutton and 21/230 (9.1%) samples of pork positive for *Campylobacter*. This prevalence is similar to findings in other countries.

Data on the numbers of *Campylobacter* present on red meats at the retail level are limited, but the available information suggests that numbers are relatively low. The effect of drying during processing is very important as this will reduce the numbers of *Campylobacter.*

Red meat is frequently consumed by New Zealanders, with 78% of the respondents in the 1997 National Nutrition Survey reporting red meat consumption in the previous 24 hours.

Notified campylobacteriosis rates in New Zealand are high compared to other developed countries. A general increase in the number of notified campylobacteriosis cases occurred from 1980 to 2005.

Seventeen outbreaks of campylobacteriosis in New Zealand from January 1999 to August 2004 have been associated with red meat consumption. However, the evidence linking human infection with red meat consumption in these outbreaks is almost always weak.

Red meats have not been identified as important risk factors in the campylobacteriosis case-control studies conducted in New Zealand. The exception is barbecued lamb, which was a significant risk factor, and it is worth noting that barbecues were mentioned in several of the red meat associated outbreaks. This could potentially be linked to the consumption of minced meat products at such events.

The outbreak data and the case-control studies do not provide strong evidence for red meat as a transmission route for campylobacteriosis in New Zealand, apart from exposure through barbecues. Nevertheless there are good data indicating low but consistent prevalence of contamination across pork, beef, and sheep meat, and red meat is a frequently consumed

food. On this basis it seems reasonable to assign red meat consumption as an identified but minor risk factor for exposure to *Campylobacter* in New Zealand.

The data gaps identified in this Risk Profile are:

- Carriage rates for *Campylobacter* in New Zealand red meat livestock;
- Influence of processing and inspection procedures on observed contamination prevalence at retail;
- Prevalence of contamination in deer meat.

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

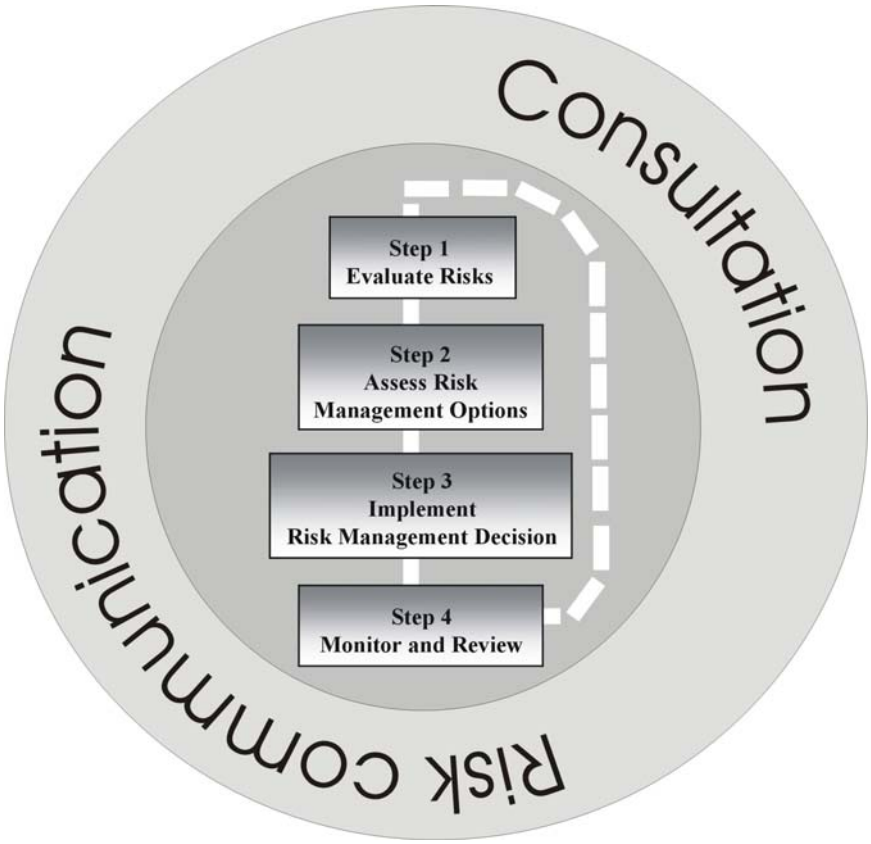


Figure reproduced from "Food Administration in New Zealand. A risk management framework for food safety" (Ministry of Health/Ministry of Agriculture and Forestry, 2000).

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 risk characterisation part of a risk assessment will usually rely on surveillance data.

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

This Risk Profile concerns *Campylobacter* in red meat (primarily ovine, bovine and porcine). This food/hazard combination was chosen for preparation of a detailed Risk Profile because the rate of notified cases of campylobacteriosis in New Zealand is high by international standards, raising the need to establish proportionality among the various potential transmission routes that have been identified.

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

Risk characterisation:

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

Risk management information:

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

Conclusions and recommendations for further action

2 HAZARD IDENTIFICATION: THE ORGANISM

The information contained in this Risk Profile is current to the date of publication. Please be aware that new information on the subject may have arisen since the document was finalised.

The following information is taken from a data sheet prepared by ESR under a contract for the Ministry of Health unless otherwise stated. The data sheet is intended for use by regional public health units.
http://www.nzfsa.govt.nz/science-technology/data-sheets/campylobacter.pdf

2.1 *Campylobacter*

2.1.1 The organism/toxin

Campylobacter cells are slender, spirally curved rods which are non-sporulating and Gram negative. There are many species of *Campylobacter* but the evidence in New Zealand suggests that only two, *C. jejuni* and *C. coli*, are of significance to public health. Other species, such as *C. upsaliensis, C. fetus, C. hyointestinalis* and *C. lari* have occasionally been reported as causing human illness (AIFST, 2003) but their significance in New Zealand is unknown.
For the sake of simplicity, in this profile, the term *Campylobacter* will refer specifically to the two pathogenic species *C. jejuni* subsp. *jejuni* and *C. coli*. *Campylobacter* spp. will be used to describe other species.

2.1.2 Growth and survival

Growth:

Temperature: *Campylobacter* are thermotolerant and grow optimally at 42°C. Neither species grows below 30.5 or above 45°C (AIFST, 2003). The organism is comparatively slow growing (fastest generation time approximately 1 hour) even under optimum conditions and does not grow under refrigeration.

pH: Optimum 6.5 to 7.5, range 4.9 to 9.5 (AIFST, 2003).

Atmosphere: It is generally considered that one of the most important factors for growth of *C. jejuni* is the oxygen and carbon dioxide content of the atmosphere. The bacterium normally requires reduced levels of oxygen – with optimum growth at 5-6% oxygen and 10% carbon dioxide (AIFST, 2003). Conventionally it has been thought that *C. jejuni* and *C. coli* do not grow anaerobically (although some species such as *C. fetus* and *C. lari* can). However evidence is emerging that *C. jejuni* possesses some interesting anaerobic electron transport pathways facilitating growth in the absence of oxygen (Kelly, 2001). The organism can be adapted to aerobic growth (Jones *et al.* 1993), although the significance of this aerotolerance in transmission of the disease is unclear.

Water activity: Optimum growth is at $a_w = 0.997$ (≡0.5% NaCl), (minimum $a_w \geq 0.987$).

Survival:

Campylobacter are sensitive to air, drying and heat.

Temperature: Survival in food is better under refrigeration than at room temperature, up to 15 times as long at 2°C than at 20°C. Freezing causes an initial one $\log_{10}$ decrease in numbers of *C. jejuni* followed by a gradual reduction during subsequent storage (AIFST, 2003) although the reduction can vary with the type of food and storage temperature. Freezing therefore does not instantly inactivate the organism in food.

Atmosphere: Survives well in modified atmosphere and vacuum packaging. Usually survives poorly at atmospheric oxygen concentrations. However, *Campylobacter* can survive and even grow when initially packed under normal atmospheric conditions, as the metabolic activity of the food, such as raw meat, may create a different gaseous environment (ICMSF, 1996).

Water activity: *Campylobacter* are very sensitive to drying, particularly at ambient temperatures. The organism can survive up to an hour on hands that are not dried properly after washing, and on moist surfaces.

Viable but Non-Culturable (VNC) Cells: Under adverse stress conditions, *Campylobacter* are said to undergo a transition to a "VNC" state. The ability of *Campylobacter* to produce VNC cells is becoming more widely, but not universally, accepted. There are claims that VNCs can colonise the intestinal tract of chickens (ICMSF, 1996).

2.1.3 Inactivation (Critical Control Points and Hurdles)

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

Temperature: Rapidly inactivated on the surface of meat by heating at 55 - 60°C for several minutes (ICMSF, 1996). D time at 50°C = 1-6.3 minutes. D time at 55°C = 0.6-2.3 minutes. D time at 60°C = 0.2-0.3 minutes. Therefore heat treatments that destroy salmonellae should also destroy *Campylobacter*.

pH: Growth inhibited in foods at less than pH 4.9 and above pH 9. Rapid death in foods at pH <4.0 especially at non-refrigeration temperatures (ICMSF, 1996). Organic acidulants are more effective than inorganic acidulants at inactivating *Campylobacter*.

Water activity: Sensitive to even slightly reduced water activity, but under certain refrigeration conditions can remain viable for several weeks (ICMSF, 1996). The drying of surface tissues during air-chilling of red meat carcasses is important in reducing *Campylobacter* prevalence (for example, from 9% before chilling to 0% after chilling on pig carcasses (Oosterom *et al.,* 1985). The prevalence of *Campylobacter* has been found to be significantly lower on air-chilled broilers compared to immersion-chilled broilers (39.3% and 48.7% respectively), although the prevalence at entry to processing was not determined (Sánchez *et al*., 2002). However, a review of survival by *Campylobacter jejuni* (Murphy *et al*., 2006) indicated that drying of poultry carcasses would not have the same effect as drying of red meat carcasses, due to a generally shorter cooling period, and the texture of the poultry skin providing cavities which act as niches for survival.

Preservatives: Sensitive to NaCl concentrations above 1%, and death occurs slowly at 2% (D time is 5-10 hours). Ascorbic acid and several spices inhibit growth. The efficacy of a 2% lactic acid spray in controlling *Campylobacter* on pork carcasses has been demonstrated (Epling *et al.,* 1993).

Radiation: Sensitive to γ irradiation. An estimated six-log reduction would result from an exposure to 2 kGy, a dose also suggested to destroy salmonellae on poultry (AIFST, 2003). 10 D would result from 2.5 kGy, therefore a 2 to 3 kGy dose is sufficient to decontaminate meat. D values reported are 0.18 kGy in refrigerated product, 0.24 kGy in frozen product.

A D value for *C. jejuni* in chilled vacuum packed minced pork has been determined to be 0.19 kGy, and a 1.5 kGy dose would give a 5 log10 unit reduction in numbers of this organism (Collins *et al.,* 1996).

Campylobacter is more sensitive to ultraviolet radiation than *E. coli* and commercial UV water treatment units producing 30 mWs/cm^2 are considered adequate to destroy the organism.

2.1.4 Sources

Human: *Campylobacter* is not one of the organisms normally found in the human intestine. Faecal-oral person-to-person transmission is rare.

Animal: *Campylobacter* can be found in the intestinal tract of a wide variety of wild and domesticated warm-blooded animals and birds which may or may not be symptomatic. The prevalence of the organism in cattle herds and sheep flocks can vary although rarely exceeds 50% (AIFST, 2003). A higher prevalence has been observed in younger animals and in animals from higher stocked densities (AIFST, 2003). Carriage rates in dairy cows and calves overseas are reported in the range of 7% to 54% (Baker *et al.,* 2002). *C. coli* is usually the dominant species in pigs. Prevalence of *C. coli* in pig faeces (n = 203) has been reported as 58% (Munroe *et al.,* 1983). Household pets have been implicated as risk factors of campylobacteriosis in control studies. Flies and other insects have been implicated as vectors. Wild or domesticated birds are a primary reservoir. The prevalence in individual poultry flocks overseas can vary from 0 to 100% (AIFST, 2003). Once a poultry flock is infected, the organism spreads rapidly and within a week most, or all, of the birds are infected.

Environment: Water and soil can be easily contaminated from infected animals' excreta. Environmental survival is considered to be poor, but new information suggests it may be better than currently acknowledged. For example, *Campylobacter* has been detected in dry beach sand. Survival in cold water is good, but reduced at temperatures above 10^oC. *Campylobacter* is present in water and sediments more frequently and at higher numbers in the winter months. These data are of interest because environmental survival appears to be opposite to the trend in the numbers of human cases, i.e. survival is poorer in the warmer months when the numbers of human infections are highest. From samples taken in New Zealand, 60% of recreational waters (i.e. river waters), 75% of shallow ground waters, 37.5% of roof waters and 29.2% of reticulated drinking waters have been shown to be contaminated by *Campylobacter*. The concentration of *Campylobacter* was low in the drinking waters, up to 0.6 MPN $100ml^{-1}$, and most isolates were *C. lari* (Savill *et al.,* 2001). A more recent

survey of New Zealand treated drinking water found negligible prevalence of *Campylobacter* (Nokes *et al.*, 2004).

Transmission Routes: Person-to-person transmission is rare, despite large (10^6 – 10^9 cfu/g) microbial loading of faeces from infected individuals. The bacterium does not grow or survive well outside the host, and is unlikely to grow on foods due to unfavourable conditions of temperature, atmosphere or moisture. The importance of undercooked chicken as a source of a proportion of cases of campylobacteriosis is recognised, but the relative importance of other routes, e.g. other foods, recreational water, occupational exposure, is unknown. Determination of the most important pathways is the primary goal of ESR and the Enteric Zoonotic Disease Research Steering Committee (EZDRSC), an interagency initiative of the New Zealand Food Safety Authority (NZFSA), Ministry of Health (MoH), research providers, funders and industry.

2.2 *Campylobacter* Typing

The terms "*subtyping*" or "*typing*" describe a test or assay which is able to distinguish isolates of a microbial species from each other. There are a variety of typing methods, including reaction with antibodies (serotyping), interaction with bacterial viruses called "phage", and analysis of bacterial DNA by a number of different techniques. Subtyping tools can be valuable for:

- Outbreak identification
- Population studies, and,
- Further characterisation of the pathogen.

In outbreak identification and investigation, subtyping allows investigators to identify outbreaks out of the general dispersion of sporadic cases, provide tight specific case-definitions for outbreak investigations, link "unrelated" outbreaks, link cases to known outbreaks, provide clues about possible sources of an outbreak, and confirm epidemiological associations with a particular source. Studies of pathogen reservoirs and transmission routes benefit through ability of subtyping to follow strains from suspected sources. Additional levels of subtyping allow determinations of potential virulence, survival, antibiotic resistance etc.

With approximately 35 typing methods or modifications of methods for *C. jejuni*, the benefits of a harmonised system have been investigated in recent years. The majority of information on serotypes in New Zealand has been derived from the "gold standard" reference method by serotyping of heat stable (HS) antigens, a method developed by Penner and Hennessy (1980). Over 60 Penner serotypes have been defined. However, the molecular basis for this typing system has not been determined. DNA based techniques have shown campylobacters to be extremely varied organisms and there is evidence for plasticity and instability in the *Campylobacter* genome which has been a problem for the development of a universal typing system (Tam, 2001).

Recent technology has enabled restriction enzyme digestion and pulsed field gel electrophoresis (PFGE) to be used for genotyping (Gibson *et al.*, 1994). However the enzymes used and the conditions under which the gel electrophoresis is undertaken can have a marked influence on the end result. The success of PulseNet USA, and increasing recognition of the international nature of infectious disease, has prompted Canada, European

countries, South America and the Asia-Pacific region, including Australia, to attempt to establish similar and compatible networks in each region.

The PulseNet USA network was established in 1996 by the Centres for Disease Control and Prevention and now involves the coordinated strain analysis of enteric bacteria by public health laboratories in all 50 states of the USA (www.cdc.gov/pulsenet). Laboratories use PFGE to fingerprint strains of disease causing bacteria. Fingerprint patterns (bar-code like patterns that tend to be the same among strains from a common source) are compared using a centralised database system facilitating the identification, tracing and prevention of food and waterborne disease outbreaks. The databases also assist in the identification of changes in strain distributions and the emergence of new strains.

In 1998, a European Commission funded network to harmonise and standardise molecular typing techniques for *C. jejuni/coli* was established and called "Campynet". The project developed was in two phases: establishment of a reference strain set, and then transfer of the strain set and methodology to participant laboratories (Scientific Committee on Veterinary Measures Relating To Public Health, 2000). Phase one has been completed; 100 strains have been collected and extensively 'characterised' including classical Penner serotyping and PFGE. Phase two is available to researchers upon request via the internet link; http://campynet.vetinst.dk/news.htm.

Efforts have been made by the New Zealand Joint Enteric Zoonotic Disease Research Steering Committee to standardise typing protocols in New Zealand. This was achieved through the commission of a report by Dr. John Klena (then at the University of Canterbury) that surveyed typing methods available (Klena, 2001). This report commented that PFGE is the most commonly used genotypic typing method in New Zealand and is therefore amenable to standardisation.

With support from the Ministry of Health, New Zealand Food Safety Authority and Dairy Insight, ESR is establishing Pulsenet New Zealand with an initial focus on *Campylobacter, Salmonella*, *Listeria*, and *Escherichia coli* O157. The following information has been obtained from Dr. Brent Gilpin, (pers. comm. July 2004). More details of the scheme can be found in the ESR report (Gilpin, 2004).

A central server has been established at ESR with a database that is compatible with the PulseNet USA system. During 2005, additional laboratories from throughout New Zealand joined the network. The electronic database will help to ensure consistent methods of sub-typing are used, so that the results will be comparable both nationally and internationally. The national link up will also enable New Zealand's laboratories to carry out collaborative studies. This could be especially important for responding to a major food or waterborne disease outbreak - either nationally or internationally. The archiving of data will also assist future studies, outbreak investigations and international comparisons through New Zealand's participation in the development of the regional group 'Pulsenet Asia Pacific' and beyond (Pulsenet Europe, Pulsenet USA etc).

Lastly, in accordance with European initiatives New Zealand is currently investigating the utility of multi-locus sequence typing (MLST), the next generation of typing technologies, as a more robust method for typing genetically unstable *Campylobacter*. MLST is gaining currency as the typing method of choice for *Campylobacter* due to the ease of assignment of sequence types and the direct comparability of data from isolates obtained worldwide. ESR

has established a routine procedure for the identification of *Campylobacter* MLST sequence types. A selection of *Campylobacter* isolates currently detailed on the PulseNet Aotearoa database is being analysed. The sequence types identified will be deposited into the database. A central repository of alleles that can be searched, is publicly available (http://pubmlst.org/campylobacter/). New Zealand isolates are being compared to those present in the *Campylobacter* MLST database (Phil Carter, personal communication, ESR, 21.09.05).

3 HAZARD IDENTIFICATION: THE FOOD

Food Standards Australia New Zealand (FSANZ) define meat under Code Standard 2.2.1 as the whole or part of the carcass of any buffalo, camel, cattle, deer, goat, hare, pig, poultry, rabbit or sheep, slaughtered other than in a wild state. Meat flesh is defined as skeletal muscle of any slaughtered animal and any attached animal rind, fat, connective tissue, nerve, blood, and blood vessels.

For the purpose of this Risk Profile 'red meat' is taken to include the skeletal muscular tissue and associated materials from the main mammalian commercial meat species i.e. cattle, sheep, pigs. This includes primal meat cuts and meat that has been further processed i.e. comminuted, diced, minced and made into products such as hamburgers. Cooking as a processing method is included, but meat products that are cured are not covered by this Risk Profile e.g. parma ham, uncooked comminuted fermented meats such as salami, and smoked meats.

3.1 Relevant Characteristics of the Food: Red Meat

Meat contains a high proportion of water and protein. All fresh red meats have water activities (a_w) of >0.99 which provides a suitable environment for microbial growth (ICMSF, 1998). It is important to note that *Campylobacter* is usually on the surfaces of (uncut) meat which will have a lower water activity (unless wrapped in plastic). Typical major components of adult mammalian muscle post rigor mortis as a percentage of the wet weight are water (75%), protein (19%), lipid (2.5%).

Important factors involved in the growth of *C. jejuni* are temperature, medium, microaerophilic atmosphere and pH (Smibert, 1974 cited in Hanninen *et al.*, 1984).

The flesh of stock animals prior to slaughter has a pH of about 7.1. The pH falls post-slaughter to reach a minimum of 5.4-5.8 within 24 hours (at death, when oxygen supply to the muscle is cut off, anaerobic glycolysis of stored glycogen to lactic acid lowers the pH). The ultimate pH value varies between muscles of the same animal and between animals, depending on the glycogen content of the muscle and accessibility of glycogen to glycolysis (ICMSF, 1998). Microbial growth does not lower the pH significantly because muscle tissue is relatively strongly buffered. However on the surface of sheep and beef carcasses, it has been demonstrated that oxygen availability allows aerobic metabolism to continue, meaning that most of the exposed surface tissues have a pH of 6 or above (ICMSF, 1998 citing Carse and Locker, 1974). Although there is an apparent rise in pH at the carcass surface potentially promoting growth, *Campylobacter* are very sensitive to drying, and so the air-drying would be expected to reduce the *Campylobacter* loading. Consequently the pH of meat both internally and at the surface would not be expected to affect *Campylobacter* survival or growth.

3.1.1 Processing

Control of *Campylobacter* begins with the livestock at the farm level. The organism can be present in the general environment in which the farmed animal is raised (streams, rivers, wild birds, pastures, other livestock). Information on carriage rates in New Zealand livestock is sparse. Gill and Harris (1982b) report 50% (25/50) prevalence of *C. jejuni* in calves but no

positive samples from 65 cattle and it was suggested that the organism is part of the normal microflora of immature animals. There was only one positive sample from 42 lambs, but in sheep, the prevalence was 14% (10/71). This result was considered not contradictory since at 4 months the lambs would have the digestive characteristics of adult sheep. However, prevalence in the healthy adult sheep demonstrates that older animals can still carry the bacteria (Gill and Harris, 1982b).

There are no effective vaccines for *Campylobacter* in the animal species covered in this Risk Profile. There are two commercial sheep vaccines available but they do not offer protection against *C. jejuni* or *C. coli* (see Section 7.1.1).

Because the main site of *Campylobacter* in the live animal is in the intestines, the overall cleanliness of the animal at time of slaughter can be a factor in contamination of the external carcass. Research on the amount of mud on the hides of cattle and *Salmonella* contamination established a relationship between greater contamination on dirtier hides of steers and heifers (Sofos *et al.,* 1999). Factors for control include animal fasting and diet prior to slaughter and general farm, transport, lairage and slaughtering hygiene practices. Ante-mortem inspection should remove unacceptably dirty and obviously diseased animals from slaughter. Equipment such as knives, sharpening steels, saws, scabbards and steel mesh gloves together with the clothing, aprons and hands of the personnel in the slaughterhall are all potential sources of cross contamination to the carcass being dressed (ICMSF, 1998).

Since live animals can be asymptomatic carriers of the *Campylobacter* organism, carrying the bacteria in the gastro-intestinal tract, they can not be detected at ante-mortem inspections and the organs do not show clinical evidence at the post-mortem inspection in the abattoir. The main source of contamination to the carcass is from the gastro-intestinal tract and from the hide/fleece. Important operations in the slaughtering process in terms of *Campylobacter* and other microbial contamination are therefore careful evisceration to avoid intestinal content spillage and the hygienic removal of the head, hocks (faeces on the hooves), hide or fleece.

The sequence of processing red meat can be found in New Zealand's Industry Standard; Number 5; http://www.nzfsa.govt.nz/animalproducts/meat/meatman/is5/is5.pdf#page25 and Number 6; http://www.nzfsa.govt.nz/animalproducts/meat/meatman/is6/index.htm.

This generally involves;

- Stunning; (e.g. electrical / captive bolt / carbon dioxide gas),
- Sticking (kill) usually by cutting the carotid arteries,
- Bleeding,
- De-skinning (scalding and dehairing for pigs/goats),
- Viscera removed, [inspection of offal and corresponding carcass and head], edible offals separated from other offal in a separate area of the abattoir,
- Air cooling of carcasses, and
- Grading/cutting and packaging.

It is unlikely that muscle tissue will be contaminated with *Campylobacter* from the stun/sticking procedure. Inadequate bleeding appears to have no effect on microbial growth on meat. Once bleeding is completed, the oesophagus is cleared from the surrounding tissue and tied/clamped close to the rumen. This prevents rumen fluid from contaminating the

neck/pleural area during evisceration. *Campylobacter* numbers are relatively low in the rumen (<100/g) and are probably part of the transient flora (ICMSF, 1998).

Most of the microbial loading on a carcass derives from the skinning procedure (ICMSF, 1998). Hands and equipment of personnel can become heavily contaminated and incisions through the contaminated hides/fleece can carry bacteria onto carcass tissue. Bacteria loading is highest on the carcass where the initial manual removal of skin occurs (ICMSF, 1998).

Work at MIRINZ in the early 1980s detected *C. jejuni* on unweaned veal carcasses (3/30 or 10% positive after chilling) and boneless veal (4/30 or 13.3% positive) (Gill and Harris 1982b). The evidence suggested that the prevalence and count of *Campylobacter* decreased during the carcass dressing process, since the numbers on the carcass reduced from a mean of 15.7 to 1.3/100 cm^2 from swabbing after skinning to swabbing after chilling. On boneless veal the count was 8/100 cm^2. However data in the same paper show a recovery rate of around 1% from meat surfaces.

A further demonstration of the effect of processing hygiene came from a study of feedlot cattle in Ireland (Minihan *et al*., 2004). In this study, a high (58%) prevalence of *Campylobacter* shed was found in the animals faeces (191/327 samples; 57% farm, 55% post-transport and 63% post-lairage). At least once during the study, 82% (90/109) of the animals shed the organism. Yet despite this high prevalence, no *Campylobacter* was isolated from 109 swabs of dressed carcasses. Key findings were that transport and lairage did not increase faecal *Campylobacter* and with appropriate abattoir hygiene, low prevalences on the dressed carcasses can be achieved. There was no visual faecal contamination on the dressed carcasses. Trimming and whole carcass hot water wash (62°C) were the only decontamination procedures used.

A study at US lamb processing plants found a very low rate of contamination (Duffy et al., 2001a). A total of 7 samples from 2226 (0.3%) were positive for *Campylobacter*, three from pre-evisceration, and three from post-evisceration, while all samples from the chiller were negative. All positive samples were from a single plant on a single day.

Control measures for cattle and sheep slaughter and dressing are also relevant for pigs. However, in pig processing skin is normally not removed, the carcass is usually scalded (steam or hot water bath) and scraped to remove hair.

Studies on sources of cross contamination of pig carcasses undergoing processing (Gill and Bryant 1993) found *Campylobacter* among the detritus on dehairing machines (10^3-10^6 /g) and in water used with the same machines ($1x10^1$-$8x10^2$/ml). Carcasses leaving the dehairing machines had *Campylobacter* present at 3-70 /cm^2 at one plant and 1-4 /cm^2 at another. Final polished carcasses at both plants had *Campylobacter* present at 1-6 /cm^2. *C. jejuni* has been isolated from 2.5% of 80 pork slaughterhouse equipment surfaces swabbed prior to slaughtering, and 32.5% during slaughtering (Oosterom *et al.* 1985).

In a study carried out by Pearce *et al.,* (2003) in a US swine slaughter and processing facility, 30 composite pig carcass samples (representing 360 swine carcasses) were taken along with rectal and environmental samples. The slaughtering process involved electrical stunning, exsanguination (bleedout), scalding, dehairing, polishing, singeing, evisceration, washing, and overnight chilling. The results are presented in Table 1.

Table 1: *Campylobacter* spp. detection on pig carcass, rectal, colon and equipment samples

Type of sample	*Campylobacter* spp. detected/no. samples (%)
Carcass processing steps in sequence (composite neck area samples)	
- post bleeding out	10/30 (33%)
- post polishing	0/30 (0%)
- after final wash	2/30 (7%)
- after overnight chilling at 2°C	0/30 (0%)
Rectal samples (composite-after bleedout)	30/30 (100%)
Colon samples from evisceration, non matching individual	48/60 (80%)
Environmental samples	
- slaughtering equipment	2/42 (4.8%)
- processing equipment	1/30 (3.3%)

The study demonstrated that slaughtering techniques can be effective in the reduction or elimination of *Campylobacter*. Of the 202 isolates recovered, *C. coli* was the predominant species (75%), followed by *Campylobacter* spp. (24%) and *C. jejuni* (1%). This predominance of *C. coli* is a common feature of studies on porcine meat.

Contamination of primal meat cuts will be located on the surface of the meat only. In general, muscle tissue of live healthy animals is sterile. However, surfaces exposed to the environment (hides, fleeces, the mouth and the gastro-intestinal tract) carry contamination, sometimes extensively (Sofos *et al.,* 1999). There is no evidence to indicate that *Campylobacter* can contaminate the interior of the whole muscle. This is in contrast to the liver organ which can be internally contaminated due to its connection with the intestinal tract via the gall bladder and bile duct.

It is considered that *Campylobacter* numbers reduce on carcasses during the chilling process, for example reductions of 100 fold in viable *Campylobacter* counts have been reported in pig carcasses after chilling (AIFST, 2003). In a study by Bracewell *et al.,* (1986), blast chilling reduced numbers slightly more than conventional chilling. The effect of chilling on *Campylobacter* is thought to be mediated through the drying of the carcass surface that occurs during the process.

3.1.2 Post processing behaviour of *Campylobacter* on red meat

The organism survived well on vacuum packed hot boned pork loins, reducing in numbers by less than 1 $\log_{10}$ after 9 days of storage at 1^{o}C (Van Laack *et al.,* 1993). Over the same period and under the same conditions numbers reduced by approximately 2 $\log_{10}$ on pork loin that was refrigerated prior to vacuum packaging, and 3 $\log_{10}$ on pork loin that was chilled unpackaged.

In a study by Hanninen *et al.* (1984), inoculated fresh beef pieces in three different package treatments (under vacuum, 80%N_2:20%CO_2, 85% N_2:10%CO_2:5%O_2 atmosphere – the latter mix was considered "optimal" for growth) at three different temperatures (4°C, 20°C and 37°C) were examined. At 37°C growth occurred, under each of the packaging conditions. At

20°C, a general decline in numbers of approximately 1 log cfu/g was observed. Only slight declines were noted when inoculated onto raw beef and stored for 25 days under vacuum or a 80%N_2:20%CO_2 atmosphere at 4°C.

When inoculated onto beef steaks and stored at –1.5°C (chilled storage, not frozen) under CO_2 or vacuum, no decline in *C. jejuni* numbers was observed over 40 days of storage (Dykes and Moorhead, 2001).

Campylobacter has been shown to grow on high pH (6.4) beef, but not at lower pH (5.8). However, growth only occurred when the temperatures exceeded approximately 30°C (Gill and Harris, 1982a; Hanninen *et al.,* 1984). Survival was good on high pH beef at –1 and –18°C, but was less so on normal pH beef (Gill and Harris, 1982a).

Numbers declined rapidly on sterile meat slices of high and normal pH when incubated at 25°C (Gill and Harris, 1982a).

These results indicate that *Campylobacter* is likely to survive or slowly decline on beef stored at refrigerated temperatures, but growth would only occur in temperature abuse situations.

3.1.3 Minced meat

Minced meat such as hamburgers may be contaminated internally with *Campylobacter,* as the mincing process will internalise any external contamination.

Growth in minced pork and beef meat was not observed at 42°C in a Swedish study; after 2 days the bacterium could not be recovered (Svedhem *et al.,* 1981).

Results from studies at 4°C are mixed. The decline in numbers in hamburgers at 4°C was quite rapid (approximate 5 $\log_{10}$ decline in 15 days) when they were stored in air, and when stored under CO_2 or N_2 a 6 $\log_{10}$ decline occurred over 60 days (Grigoriadis *et al.,* 1997). Conversely, no decline in numbers was observed for *Campylobacter* in minced pork or beef meat (Svedhem *et al.,* 1981) or pork skin when stored at 4°C (Bracewell *et al.,* 1985, Bracewell *et al.,* 1986).

The organism has been shown to survive with no reduction in numbers over 48 hours under aerobic incubation on pork skin at 4°C, whereas there were significant reductions (1 log cfu/g or more) in numbers at –20 and 25°C, and modest reductions at 37 and 42°C (Solow *et al.,* 2003). *C. jejuni* and *C. coli* each showed similar survival properties. Results were similar when incubation was microaerophilic, except at 37 and 42°C where growth occurred.

A slightly different finding on beef trimmings indicated an initial decline of 1-2 $\log_{10}$, followed by a period of stability in counts at –18°C (Moorhead and Dykes, 2002), and similar results were obtained for *Campylobacter* in minced beef at –15°C (Stern and Kotula, 1982).

Cooking readily controls *Campylobacter*. In minced beef, numbers reduced from 10^7 cells/g to <30 in less than 10 minutes after the meat had reached an internal temperature of 70°C (Stern and Kotula, 1982).

3.1.4 *Campylobacter* behaviour on meat

No information has been located about *Campylobacter* behaviour on beef or lamb during processing, but pig meat processing appears to reduce or eliminate contamination.

On meat cuts or minced meat, important factors for the behaviour of *C. jejuni* are temperature, medium, microaerophilic atmosphere, and pH. The available information indicates that *Campylobacter* declines rapidly under ambient or freezing temperatures, but survives well at refrigeration temperatures. The chilling process experienced by carcasses appears to reduce numbers by 100 fold, probably as a result of surface drying rather than the function of temperature control. As a medium for growth, meat is suitable because of the high water and protein content, but high temperatures (at least 30°C) are needed. *Campylobacter* require oxygen for growth but studies show that for optimum conditions, a reduced oxygen level (from normal atmospheric) is needed.

3.2 The Food Supply in New Zealand

There are 17,000 commercial sheep and beef cattle farms in New Zealand, most of which are owned and operated by farming families. Livestock numbers for New Zealand in 2004 from Statistics New Zealand are shown in Table 2

Table 2: Livestock numbers, production and export for New Zealand to June 2004

Livestock type	Number of animals at June 2004*	Meat production in year to June 2004 *(tonnes carcass weight)	Amount exported in tonnes carcass weight and percentage in ()
Sheep	39,700,000	114,000 mutton 434,000 lambs	95,000 (83.3%) mutton 361,000 (83.1%) lamb
Beef cattle	4,660,000	592,000 beef 25,000 veal	Total export 517,000 (83.8%)
Pigs	355,000	51,300	49,200 exports (95.9%) 29,400 imports
Deer	1,690,000	33,000	20,100 (60.9%)

*From MAF (2003) Situation and Outlook New Zealand Agriculture and Forestry website: http://www.maf.govt.nz/mafnet/rural-nz/statistics-and-forecasts/sonzaf/2004/04-update/httoc.htm

As the figures indicate, the vast majority of meat production in New Zealand is exported with the exception of pork. New Zealand accounts for 53% of the world export trade in sheep meat and 9% in beef, making the country the fourth largest in terms of meat trade. The Meat Industry Association of New Zealand (MIA) (www.mia.co.nz) is a voluntary trade association representing companies supplying 99% of sheep meat exports and 100% of the beef exports. The Association provides the interface between the meat industry and the government. More than 150 New Zealand meat companies are licenced to operate with most processing for export. The largest companies are AFFCO (www.affco.co.nz), the Alliance Group (www.alliance.co.nz) and PPCS (www.ppcs.co.nz).

Advances in productivity have meant that since 1990-1991, beef and veal production has increased 22% and lamb production increased 10% despite 32% fewer sheep being farmed (www.nzmeat.co.nz). One major technological advancement has been the extending of shelf life for chilled meat and each year, a greater proportion of meat is being exported as chilled cuts rather than frozen carcasses. In 2003, 20% of all lamb exports were exported chilled (www.mia.co.nz, Annual Report 2003).

New Zealand has a relatively small pig industry which focuses on the domestic market. Currently about 48,400 breeding sows are farmed, with an estimated 350,700 pigs on farms at any one time (New Zealand Pork Industry Board, 2001). Since 1995 pigmeat production has been relatively static averaging 49,000 tonnes per year, although it appears that pigmeat consumption has been slowly increasing.

3.2.1 Imported food

New Zealand imports relatively small amounts of beef and sheep meat, according to data from Statistics New Zealand. For the year to March 2003 approximately 9,900 tonnes of beef carcasses and cuts were imported from Australia, with a smaller amount (11 tonnes) derived from Korea. For the same period, 2,400 tonnes of sheep meat (all types) was imported, all from Australia. These data, when compared to the production and export figures above, suggest that the approximately 6% of New Zealanders beef and sheep meat for domestic consumption derive from Australia.

Imports of pork have increased steadily to now make up approximately 35% of total supply (29,400 tonnes in 2003/2004), mostly sourced from Australia and Canada. All were frozen meat carcasses and cuts; see http://www.pork.co.nz/profile.asp).

Small amounts of processed meats are imported, principally from Australia. Meat preparations (with a non-poultry base) comprised 230 tonnes for the year to March 2003. These were not preserved in airtight can or jars. Approximately 3,700 tonnes of processed meats of bovine origin in airtight can or jars are also imported, principally from Australia.

4 HAZARD CHARACTERISATION: ADVERSE HEALTH EFFECTS

In developed countries, most cases of campylobacteriosis occur in the young adult or young child populations and the disease is characterised by an inflammatory process. The usual symptoms are an acute attack of diarrhoea lasting approximately five days, often accompanied by fever and abdominal pain in the early stages.

The inflammatory process is proposed to occur by invasion and proliferation of the organism within the intestinal epithelium, followed by the production of cytotoxins which cause cell damage and can result in bloody stools and faecal leucocytes. Symptomatic patients shed 10^6 – 10^9 cells of *C. jejuni*/g of faeces (AIFST, 2003). However, studies indicate that the pathogenic determinants of *C. jejuni* strains isolated from patients correlate poorly with clinical symptoms (AIFST, 2003).

4.1 Symptoms

Incubation: One to 10 days (usually between 2 and 5 days).

Symptoms: Typically muscle pain, headache and fever (known as the "febrile prodrome") followed by watery or bloody diarrhoea, abdominal pain and nausea. Symptoms may last 1 day to 1 week or longer (usually 5 days). Excretion of the organism in stools occurs on average for 2 to 3 weeks and is mostly self-limiting. Hospitalisation has been reported in up to 13% of cases. The attack rate is around 45%.

Condition: Campylobacteriosis.

Toxins: No toxins are produced in foods.

At Risk Groups: Can affect any age group but most often isolated from infants (< 1 year) and young (twenties) adults. Incidence higher in males (up to 45 years of age).

Long Term Effects: Campylobacteriosis is a recognised cause of chronic sequelae in the form of Guillain-Barré syndrome (GBS). The frequency of GBS resulting from campylobacteriosis has been estimated as 0.1% (Altekruse *et al.,* 1999) and can occur one to three weeks after enteritis. Approximately 20% of patients with GBS are left with some form of disability and approximately 5% die.

In a case-control study of patients with GBS, evidence for a preceding *C. jejuni* infection was found in 26% of cases, although the true frequency of antecedent *C. jejuni* infection is probably higher, making this *Campylobacter* the most single identifiable pathogen in the syndrome (Rees *et al.,* 1995). The authors also found that GBS was more likely to develop in men than in women, which suggests either a sex-linked predisposition or more males contracting *C. jejuni* infection in the first instance. The conclusion was that infection with *C. jejuni* precedes Guillain-Barré syndrome and is associated with axonal (peripheral nerve) degeneration, slow recovery, and severe residual disability.

Campylobacteriosis is also associated with Reiter's syndrome, a reactive arthropathy. The frequency of this illness has been estimated as 1% of all campylobacteriosis cases (Altekruse *et al.,* 1999).

A number of other less common non-enteric diseases can occur. Invasion of the bloodstream may occur in 1.5 per 100,000 cases, especially in the elderly. A case report has linked "*Vibrio fetus* septicaemia" (an old name for *Campylobacter*) with the consumption of blended raw beef liver (Soonattrakul *et al*., 1971). The organism has also been reported to cause liver abscesses (Brmbolić, 1995). USA data suggest a case-fatality rate of around 3 per 100,000 outbreak associated illnesses.

Treatment: Usually none, but fluids may be given, especially as young and elderly patients may become dehydrated. Some cases warrant treatment with antibiotics. Erythromycin or norfloxacin are usually recommended. Strains resistant to erythromycin and norfloxacin have been isolated from a small number of campylobacteriosis cases in New Zealand, although some of these cases may have acquired their infection overseas (Helen Heffernan, personal communication, ESR January 2007).

4.2 Types Causing Disease

There is, as yet, no definitive evidence to suggest that different types of *Campylobacter* vary in their ability to cause gastrointestinal disease in humans. However, there is speculation that this might be so and some preliminary data support this idea. For example, Lee *et al.* (2000) have shown differential toxin production between isolates. To cause disease, *C. jejuni* must adhere to, invade and damage host cells and therefore must produce adhesion and invasive factors and cytotoxic and/or cytotonic toxins (AIFST, 2003). Despite this, all types need to be regarded as capable of causing disease until further information allows reliable differentiation between types of differing pathogenicity.

Certain serotypes of *C. jejuni,* particularly Penner Serotype O:19 and O:41 have been more frequently associated with GBS than other serotypes (AIFST, 2003). Penner Serotype O:19 has been associated with GBS in Japanese studies. However, this link was not confirmed in a USA case control study, in which no specific serotypes were associated with GBS (Rees *et al*., 1995).

4.3 Dose Response

Teunis and Havelaar (2000) reported that the conventional view of a minimum dose, below which infection can not occur, is being replaced. The growing consensus is that ingestion of even a single cell has an associated probability of causing infection (even though the probability may be very small). If the number of exposure events is high, even low probabilities of infection may be significant.

Data from experimental studies where volunteers ingested known numbers of *Campylobacter* cells have been investigated for the purpose of modelling the dose-response relationship (Medema *et al.,* 1996; Teunis *et al.,* 1999). Infection, where the microorganism is reproducing in the body, was modelled separately from illness, which is less frequent. The probability of infection increased from approximately 50% at 800 cells to approximately 100% at 1 x 10^8 cells. In contrast, the likelihood of illness was approximately 20% at 800 cells, rising to approximately 55% at 9 x 10^4 cells, and declining to 0% at 1 x 10^8 cells.

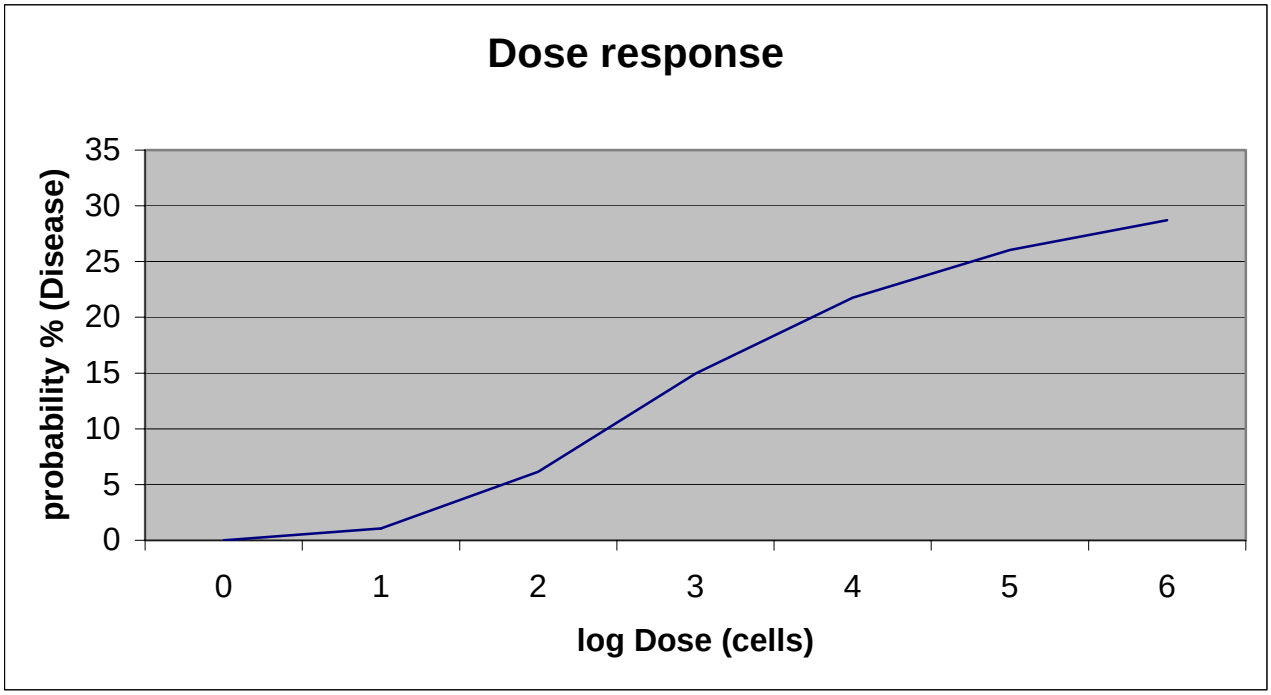
One interpretation of the limited data suggested that the likelihood of illness actually declines with increasing dose once infection is established. Some researchers suggest that exposure to a large dose elicits a stronger host defense response that reduces the probability of illness

(Teunis *et al.*, 1999). Taken in combination with the model for infection, the overall effect is that there is an optimum number of cells consumed for sickness to occur. This limited study is the only evidence known to suggest this effect and so should be treated with caution.

More recently the FAO/WHO hazard characterisation (FAO/WHO, 2002) has explored the idea that there is a conditional probability of disease in humans resulting from infection. This model predicts that in the vast majority of cases where people become infected there is >20% and <50% chance of subsequently becoming sick.

To give an idea of the probability of human disease given a variety of doses, the following information results from application of the FAO/WHO model using a fixed 33% probability of developing disease after infection has occurred.

Figure 2: FAO/WHO dose response model; probability fixed at 33%



5 EXPOSURE ASSESSMENT

5.1 The Hazard in the New Zealand Food Supply: *Campylobacter* in Red Meat at Retail

No campylobacters were detected in a retail study of ground beef (Gill and Harris, 1984). A total of 50 samples were collected from 27 outlets over a two month period. The minimum detection level was 2/g. The same study examined the survival of *C. jejuni* during the hamburger cooking process (see section 5.3.5. Heat treatment).

A small retail survey in Christchurch of 100 raw hamburger patties and 100 raw fresh sausages was unable to isolate *Campylobacter* from any of the samples (Hudson and McGuire, 2002). The method was shown to be able to detect between 6 and 29 *Campylobacter* cells in a 10g spiked sample. The authors discussed the possibility that absence was due to the products were made from frozen meat, or other ingredients (herbs and spices) may have acted as inhibitors, or other competing organisms may have been exerting similar inhibitory effects.

A recent national retail survey of raw minced/diced meat from supermarkets and butchers has been undertaken by ESR for the NZFSA to assess the prevalence of foodborne pathogens. The part of the study which has been concerned with *Campylobacter* has been completed (Wong, 2004). The results are shown in Table 3;

Table 3: National retail survey of Campylobacter in raw minced/diced meat; July 2003 to June 2004

Meat (all minced/diced)	No. samples tested	Total number positive (25g) (%)	*C. jejuni*	*C. jejuni* & *C. coli*	*C. coli*	Counts in positive samples (MPN/g)
Beef	230	8 (3.5)	7	1	0	All <0.3
Bobby veal	90	9 (10)	8	0	1	<0.3 8 samples >10.9 1 sample
Lamb/mutton	231	16 (6.9)	14	1	1	<0.3 14 samples 0.3 2 samples
Pork	230	21 (9.1)	18	0	3	<0.3 20 samples 0.3 1 sample
(Chicken)	230	205 (89.1)	199	5	1	<0.3 to 110

These data indicate a low *Campylobacter* contamination rate in raw red meat in New Zealand. The prevalence ranges from 0% (Hudson and McGuire, 2002, Gill and Harris, 1984) up to 10% (Table 3). In New Zealand as well as overseas, raw red meat has a significantly lower prevalence of *Campylobacter* contamination than raw poultry meat.

5.2 Food Consumption: Red Meat

Red meat consumption has declined over the last 20 years, as shown in Table 4. A major shift in consumption patterns has taken place with major gains by the poultry and smaller gains in the pork industries.

Table 4: New Zealand domestic meat consumption per capita 1985, 1995, 1996, 1999 to 2003 (kg/person/year)

Year	Mutton and Lamb	Beef and Veal	Pig meat	Total Red meat	Poultry	Total Meat
1985	27.3	36.5	14.2	78.0	15.0	93.0
1995	23.2	34.6	15.7	73.5	26.2	100.1
1996	20.6	37.8	16.1	74.5	25.1	99.8
1999	14.3	31.2	17.1	62.6	26.8	89.5
2001[1]	16.6	27.1	16.5	60.2	31.0	91.3
2001/02[2] (Sept. end)	16.1	27.6	17.3	61.0	34.1	95.1
2002/03[3] (March end)	15.3	29.4	17.9 (record high)	62.6	35.9	98.5

From New Zealand Meat and Wool Board's Economic Service (MWBES) Annual Review of the Sheep and Beef Industry, 1999-2000.
[1] Data from website; http://www.beef.org.nz/statistics/slides.asp
[2] Compendium of New Zealand Farm production statistics, April 2003.
[3] PIANZ, December 2003

The meat consumption figures for New Zealand in Table 4 are similar to estimates made for the Australian population (Baghurst, 1999). An international comparison of meat consumption as calculated for 2003 is given in Table 5.

Table 5: International comparison of meat consumption, 2003 (kg/person/year)

Country	Bovine meat	Sheep and goat meat	Pigmeat	Poultry meat
Argentina	54.7	1.5	5.1	19.4
Australia	45.1	14.4	21.1	35.6
Canada	34.3	1.0	27.4	36.3
New Zealand	26.4	24.8	20.7	35.2
UK	20.9	5.9	26.0	30.0
USA	41.9	0.5	30.1	50.2

Source: http://faostat.fao.org/

The figures given above represent the meat available for consumption in New Zealand. Information on amounts of meat reported to be actually consumed by individuals can be abstracted from the 1997 National Nutrition Survey (NNS) (Russell *et al.*, 1999). FSANZ have carried out an analysis of this dataset (ANZFA, 2001), including application of a set of standard recipes, to allow composite foods to be reduced to their component parts. Table 6 gives the estimates for New Zealand domestic meat consumption derived by ANZFA and compares those levels of consumption to the estimates based on meat available for consumption in Table 2.

Table 6: Mean estimates of New Zealand domestic meat consumption in 1997 and estimates of meat available for consumption, 2004

Meat type	Estimated consumption (1997)*	Amount available for consumption (2004)#	
	g/person over 15 yrs/day	kg per person per year	g per person per day
Beef and veal	87.9	29.93	82.0
Sheep and Lamb	13.7	23.54	64.5
Pig meat	32.3	7.86	21.5
Deer meat	0.9	3.28	8.8
Total red meat	**134.9g**	**64.6kg**	**181g**

* from ANZFA analysis of 1997 National Nutrition Survey data (ANZFA, 2001)

\# recalculated from Table 2 and 2003 import data, (population at June 2004; 4,009,200) source: http://www.stats.govt.nz/

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

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

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

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

Meat type	Percentage of total red meat consumed (by servings)	Percentage of total red meat consumed (by weight)
Beef (including veal)		
Corned beef	6.3	5.0
Beef mince and beef mince recipes (pattys, hamburgers, etc)	14.7	24.1
Beef cuts (steak, roast, schnitzel, etc)	20.2	26.2
Sheep meat (Lamb, hoggett and mutton)		
Hoggett/mutton cuts	4.1	3.6
Lamb cuts	6.0	5.2
Lamb mince and lamb mince recipes	0.1	0.1
Pigmeat (including ham and bacon)		
Pigmeat cuts	6.8	8.3
Pigmeat mince	0.1	0.1
Bacon	7.3	2.9
Ham	11.5	4.3
Mixed meat products		
Sausages, saveloys, frankfurters and hotdogs	13.6	15.1
Other meats		
Venison	0.4	0.5
Other	8.9	4.6

5.3 Qualitative Estimate of Exposure

5.3.1 Number servings and serving sizes

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

Serving sizes will vary considerably within the red meat and meat products group from hundreds of grams for a meal of meat cuts to a few grams for ready-to-eat meats consumed as a component of a sandwich. According to the FSANZ analysis of the NNS data the average daily consumption of red meat by consumers (only those reporting consumption of red meat) is similar to average daily consumption for consumers of common fruits and vegetables.

The estimation of total number of servings of red meat consumed on a per annum basis involves a number of assumptions:

- That the sample set employed for the NNS are 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,
- That the consumption of red meat 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). This assumption is questionable, but information for New Zealanders less than 15 years is currently unavailable.

From the NNS, 3569 respondents were identified as consuming red meat. It will be assumed that this number is likely to be a good approximation to the total number of servings, although likely to be on the conservative side as respondents could be consuming more than one serving of red meat per day. Using a total survey population of 4636 and a total New Zealand population of 4,054,200 (at 31 March 2004) (http://www.stats.govt.nz/):

Annual number of servings (total population) = 3569 x 4,054,200/4636 x 365
= 1.1×10^8 servings

5.3.2 Frequency of contamination

From the latest ESR data available (section 5.1.1.), the prevalence of *Campylobacter* on raw minced/diced lamb/mutton, pork and bobby veal is below 10%. Raw beef has the lowest rate of contamination rate at 3.5%. However in a survey of minced meat products such as sausage and hamburgers in Christchurch (Hudson and McGuire, 2002), no isolations were made from 100 samples.

5.3.3 Predicted contamination level at retail

MPN values for *Campylobacter* on red meat are most often in the <0.3 MPN/g category. Given that these counts are for samples which were positive for the presence of *Campylobacter* in a 25g sample, then the most frequent count is actually >0.04 MPN/g and < 0.3 MPN/g, assuming that the presence/absence test is able to detect one cell in the 25 g sample.

5.3.4 Growth rate during storage and most likely storage time

The normal acidity of raw meat together with storage under refrigeration or frozen conditions suggests that levels of contamination are unlikely to increase during storage, provided refrigeration temperatures are maintained. Conversely survival of campylobacters will be best under refrigeration conditions. For whole muscle cuts of fresh meat the period of storage is short and even lower for minced products. A website linked to the American Meat Institute; http://www.meatsafety.org/safehandling/safehandling.htm provides the following recommended periods for safe storage of meats in a refrigerator:

Beef and pork whole muscle cuts and sausages 3 to 5 days; ground mince 1 to 2 days.

In a study by Gill and Harris (1984), freezing of inoculated ground beef reduced numbers of *C. jejuni* by approximately one log.

5.3.5 Heat treatment

The information presented here on exposure to *C. jejuni* through consumption of red meat indicates that the food is commonly eaten, but that the probability of contaminated product after cooking is low. Several circumstances might be expected to have a significant impact on this probability:

- *Campylobacter* are a contaminant to the outside of whole meat muscle cuts and generally have not been found internally in red meat muscle,
- normal cooking temperatures should be adequate to destroy *Campylobacter*,
- however, where contamination may occur internally (in foods such as sausages, burgers and rolled roasts) cooking may be less thorough, especially where barbecued and the internal portions of these foods may be undercooked so that the organism survives.

In the study by Gill and Harris (1984), the cooking of inoculated ground beef hamburgers was assessed in regard to the survival of *C. jejuni*. The hamburgers were cooked on a hot plate at 175°C and turned over half way through any cooking period. The time required to eliminate *C. jejuni* ranged from 2 minutes in 1 cm thick (50g) fresh or frozen/thawed hamburgers, to 8 minutes in 2 cm thick (100g) frozen thawed hamburgers.

In minced beef, numbers reduced from 10^7 cells/g to <30 in less than 10 minutes after the meat had reached an internal temperature of 70^{o}C (Stern and Kotula, 1982).

Survival of the bacteria in cooked minced meat appears to be greater at refrigeration temperatures. For example, when added at around 10^6 cfu/g *C. jejuni* became undetectable (<50 /g) in cooked minced beef after 49 days at 2^{o}C, 23 days at 10^{o}C and 7 days at 20^{o}C (in raw beef mince it became undetectable after 27 days when held at 2°C (Curtis *et al*., 1995).

5.3.6 Exposure summary

Given the high frequency of red meat consumption, even though the prevalence of *Campylobacter* in red meat is low compared to poultry, these bacteria will frequently be introduced into the home on these foods, albeit at low numbers.

Cooking readily destroys *Campylobacter*, but cross contamination may occur during food preparation. Cross contamination to foods occurs with low efficiency, but some of these foods may not be cooked before consumption. The importance of cross contamination as a transmission route can really only be assessed as part of a quantitative risk model.

5.4 Overseas Context

5.4.1 *Campylobacter* in red meat overseas

Table 8 summarises the prevalence of *Campylobacter* in raw red meat overseas.

Table 8: Prevalence of *Campylobacter* in raw red meat overseas

Country	Product	Number Samples tested	Positive for *Campylobacter* (%)	Reference
Australia	Beef carcasses: Domestic Export Weekend chill	 124 533 96	 1 (0.8) 1 (0.2) 0	Vanderlinde *et al.,* 1998
Australia	Sheep carcasses, domestic Sheep carcasses, export	140 330	NS (2.1) NS (0.9)	Vanderlinde *et al.,* 1999
Belgium	Pork carcasses Beef carcasses (pooled samples of 5 carcasses)	49 62	1 (2.0) 6 (10.0)	Korsak *et al.,* 1998
Canada	Pork carcass Beef carcass Veal carcass	463 598 267	78 (12.1) 135 (14.7) 115 (34.5)	Lammerding *et al.,* 1988
Canada	Pork carcass diaphragms	200	47 (23.5)	Mafu *et al.,* 1989
England	Minced meats Sausage meats	135 143	3 (2.2) 1 (0.7)	Bolton *et al.,* 1985
England	Beef Pork Lamb	127 158 103	30 (23.6) 29 (18.4) 16 (15.5)	Fricker and Park, 1989
England	Raw sausages Raw burgers Other raw meat products	1197 1015 118	4 (0.3) 10 (1.0) 1 (0.8)	Little and de Louvais, 1998
England and Wales	Minced beef Minced pork Sausage/sausage meat Other (beef, beefburger, pork, lamb, rabbit kidney)	2015 342 1448 2278	21 (1.0) 1 (0.3) 2 (0.1) 74 (3.2)	Turnbull and Rose, 1982
Ireland	Minced beef Pork	20 20	4 (20.0) 0	Cloak *et al.,* 2001
Ireland	Raw beef Raw pork Raw lamb	221 197 262	7 (3.2) 10 (5.1) 31 (11.8)	Whyte *et al*., 2004
Italy	Pork Sausage	27 41	1 (3.7) 1 (2.4)	Zanetti *et al.,* 1996
Italy	Beef Pork	151 175	2 (1.3) 18 (10.3)	Pezzotti *et al.,* 2003
Japan	Beef Pork	94 52	2 (2.1) 0	Tokumaru *et al.,* 1990
Japan	Beef Pork	112 126	0 0	Ono and Yamamoto, 1999
Japan	 Deer meat	 30	 0	Kanai *et al.,* 1997
Netherlands	Pig carcasses after evisceration Pig carcasses after cooling Minced pork	210 210 248	19 (9.1) 0 0	Oosterom *et al.,* 1985

Country	Product	Number Samples tested	Positive for *Campylobacter* (%)	Reference
Northern Ireland	Lamb carcasses	100	0	Madden *et al.,* 1998
	Beef carcasses	100	0	
	Pork retail packs	50	0	
	Beef retail packs	50	0	
Northern Ireland	Beef carcasses	100	0	Madden *et al.,* 2001
Poland	Porcine carcasses (prechill)	105	3 (2.9)	Kwiatek *et al.,* 1990
	Bovine carcasses (prechill)	114	1 (0.9)	
Spain	Lamb carcasses	30	0	Sierra *et al.,* 1995
Sweden	Pork and beef mince	9	9 (100.0)	Svedhem *et al.,* 1981
USA	Pork carcasses post kill*	30	10 (33.0)	Pearce *et al.,* 2003
	Pork carcasses post polish*	30	0	
	Pork carcasses prechill*	30	2 (6.7)	
	Pork carcasses post chill*	30	0	
USA	Pork carcass after slaughter, conventional chill	75	8 (10.7)	Epling *et al.,* 1993
	Pork carcass 24h post mortem, conventional chill	75	3 (4.0)	
	Pork carcass after slaughter, intermittent spray chill	75	8 (10.7)	
	Pork carcass 24h post mortem, intermittent spray chill	75	9 (12.0)	
USA	Beef	230	1 (0.4)	Harris *et al.,* 1986a
	Pork	149	1 (0.7)	
	Lamb	37	0	
USA	Frozen beef cheek meat	35	0	Stern *et al.,* 1984
	Frozen ground beef	45	0	
	Frozen pork sausage	45	0	
	Frozen pork cheek meat	40	0	
	Frozen lamb flank meat	50	1 (2.0)	
	Fresh beef cheek meat	39	0	
	Fresh ground beef	50	0	
	Fresh pork sausage	40	1 (2.5)	
	Fresh pork cheek meat	41	0	
	Fresh lamb flank meat	36	2 (5.5)	
USA	Beef carcasses	2064	NS (4.0)	McNamara, 1995
USA	Beef	13	0	Christopher *et al.,* 1982
USA	Pork chop	360	18 (5.0)	Stern *et al.,* 1985
	Pork sausage	360	15 (4.2)	
	Ground beef	360	13 (3.6)	
	Beef flank	360	17 (4.7)	
	Lamb stew	360	27 (8.1)	
USA	Pork	181	3 (1.7)	Zhao *et al.,* 2001
	Beef	182	1 (0.5)	
USA	Pork carcasses	932	0	Miller *et al.,* 1997

Country	Product	Number Samples tested	Positive for *Campylobacter* (%)	Reference
USA	Lamb carcasses pre-evisceration	636	3 (0.5)	Duffy *et al.*, 2001a
	Lamb carcasses post-evisceration	636	4 (0.6)	
	Lamb carcasses from chiller after processing	954	0 (0)	
USA	Pork products from processing plants	120	8 (6.7)	Duffy *et al.*, 2001b
	Pork products from retail	384	5 (1.3)	

NS Not stated
* Composite samples

There is a considerable variability in the prevalence which could be due to various methods of sampling, unit size and analysis methodology. Values range from 0 to around 25%, with one result 100% in Sweden. The Canadian survey found a significantly higher prevalence of thermotolerant *Campylobacter* in veal carcasses compared to beef carcasses. The authors suggest that this result may reflect the differences in intestinal flora due to age or rearing practices. There is perhaps a trend for more recent studies to give lower prevalences which might reflect steadily increasing standards in dressing hygiene. It should be noted that pork products are more usually contaminated with *C. coli* rather than *C. jejuni*. As an example, a ratio of 10 *C. coli* for every one *C. jejuni* was reported for isolates from pork carcass diaphragms (Mafu *et al.,* 1989). Otherwise almost all other isolates are *C. jejuni*, with other species being rarely identified. For example, in examining 724 isolates from beef, pork, veal, and poultry carcasses 3 (0.4%) were *C. lari* (Lammerding *et al.,* 1988).

The second report on *Campylobacter* issued by the UK Advisory Committee on the Microbiological Safety of Food (ACMSF, 2004) includes some testing results provided by a leading UK multiple food retailer. The results of testing for *Campylobacter* in raw meats on retail sale in February 2002 were that none were detected in fresh retail cuts of beef (53 samples), lamb (69) and pork (25), minced/reformed beef (41), lamb (3) and pork (12). Further testing in September 2002 also failed to detect *Campylobacter* in retail meats. The report's authors commented it was not surprising that the high carriage rates in red meat animals did not carry through to the final product as, compared to poultry, there are significant differences in the way that animals such as cattle, pigs and sheep are reared, transported and slaughtered. Control measures are in place to minimise faecal contamination of hides and fleeces, reducing the potential for contamination during dehiding and evisceration.

Few quantitative data are available. The geometric mean of *Campylobacter* on positive carcasses in the USA was 0.1 MPN cm^{-2} (McNamara, 1995). Given the low level of recovery of *Campylobacter* from meat surfaces, this number is consistent with that described for New Zealand veal above (Gill and Harris, 1982b). The maximum number on the beef carcasses was <1.0 MPN cm^{-2}. Of 19 carcasses positive for *C. jejuni* when tested immediately after

evisceration all but one contained <100 organisms per carcass, and the one exception contained 460 (Oosterom *et al.,* 1985).

Of four *Campylobacter* positive mince samples, only one had a count above the level of detection, and that was in the range of 0.7-1.0 $\log_{10}$ /g. The other three were therefore <0.7 $\log_{10}$/g (Cloak *et al.,* 2001).

5.4.2 *Campylobacter* on raw meat external packaging

In an overseas study, external contamination of raw meat packaging was reported. The study by Local Authorities Coordinators Of Regulatory Services (LACORS) and the Health Protection Agency in the UK during September and October 2002 was recently published (Health Protection Agency, 2004). A total of 3,662 pre-packaged raw meat, poultry and offal samples were collected from 2,304 retail premises across the UK, frozen and canned product was deliberately excluded. Details of the study are available to subscribers of the LACORS website; www.lacors.gov.uk. Red meat accounted for 526 (14.4%) of the samples tested. Positive samples were detected from beef steak 1/330:0.3% (*C. coli*), lamb chops 1/150;2.4% (*C. jejuni*) and pork diced/cubed 1/46:2.2% (*C. jejuni*). *Campylobacter* was thus detected from 3 (0.6%) of the external red meat packaging samples.

In this study, the external surface of heat sealed packaging was less frequently contaminated with *Campylobacter* and *E. coli* compared to other types (overwrapping, bag and tie tape). External packaging was intact for the majority of samples, proportionately *Campylobacter* and *E. coli* prevalence was greater when the packaging was not intact and when the display areas were not visually clean.

5.4.3 Serotypes overseas

An analysis of the serotypes isolated from human cases, environmental and food samples was performed in the United Kingdom in 1984-1986 (Fricker and Park 1989). The results relevant to red meats are reproduced in Table 9.

Table 9: The occurrence and rank order of the 10 serogroups of campylobacters most frequently isolated from human faeces and in meat samples

Penner serotype	Sample type			
	Human faeces: 212 samples	Beef: 30 samples	Lamb: 16 samples	Pork: 15 samples
2	18.9%	12 (40%)	3 (18.8%)	NI
4	14.2%	3 (10%)	2 (12.5%)	NI
1	8.3%	2 (6.7%)	1 (6.3%)	NI
3	6.9%	NI	NI	NI
13/16	6.3%	3 (10%)	NI	NI
8	5.1%	2 (6.7%)	NI	NI
23	3.6%	NI	NI	NI
24	2.2%	NI	NI	6.9
9	2.1%	1 (3.3%)	2 (12.5%)	NI
10	1.5%	NI	NI	NI

NI=Not isolated. Source: (Fricker and Park, 1989).

Serotyping is not in itself highly discriminatory, but these data suggest that the serotypes in human campylobacteriosis cases are similar to those isolated from beef and lamb, and least like those isolated from pork. This comparison must be treated with caution however; for example Serotypes 2, 4, 1, 3, 13/16 and 8 were also frequently isolated from poultry, offal and river water sources.

6 RISK CHARACTERISATION

6.1 Adverse Health Effects in New Zealand

6.1.1 Incidence

Campylobacteriosis has consistently been the most commonly reported infectious intestinal disease in New Zealand at 63.3% of all total notifications (23,349) in 2003, 53.2% of all notifications (22,944) in 2004 (ESR, 2005a) and 60.0% (23,083) in 2005 (ESR, 2006a). The disease was discussed as a potential epidemic over 10 years ago (Lane *et al*., 1993). Notification data for the period 1990 – 2005 are given in Table 10, and illustrated in Figures 3 and 4. The highest monthly campylobacteriosis total for 2005 was for the month of November when 1666 cases were notified (ESR, 2006a).

Table 10: Number of reported cases and rates of campylobacteriosis from 1990 to 2005

Year	Number of cases of campylobacteriosis	Rate per 100,000*	Reference
1990	3850	116.4	Lopez *et al*., 2001
1991	4148	122.9	Lopez *et al*., 2001
1992	5144	152.5	Lopez *et al*., 2001
1993	8101	240.1	Lopez *et al*., 2001
1994	7714	228.6	Lopez *et al*., 2001
1995	7442	220.6	Lopez *et al*., 2001
1996	7628	210.8	Lopez *et al*., 2001
1997	8848	244.5	Lopez *et al*., 2001
1998	11578	320.0	Lopez *et al*., 2001
1999	8173	225.9	Lopez *et al*., 2001
2000	8430	233.0	Lopez *et al*., 2001
2001	10148	271.5	Sneyd *et al*., 2002
2002	12489	334.2	Sneyd and Baker, 2003
2003	14786	395.6	ESR, 2004
2004	12213	326.8	ESR, 2005a
2005	13839	370.3	ESR, 2006a

* The New Zealand population increases by up to an estimated 2% per annum (http://www.stats.govt.nz/analytical-reports/dem-trends-05/default.htm). The campylobacteriosis rates are calculated using the most recent census data (e.g. 2001 census for rates from 2001 to 2005). An annual rate increase of more than 2% therefore represents an increase in reported notification rate.

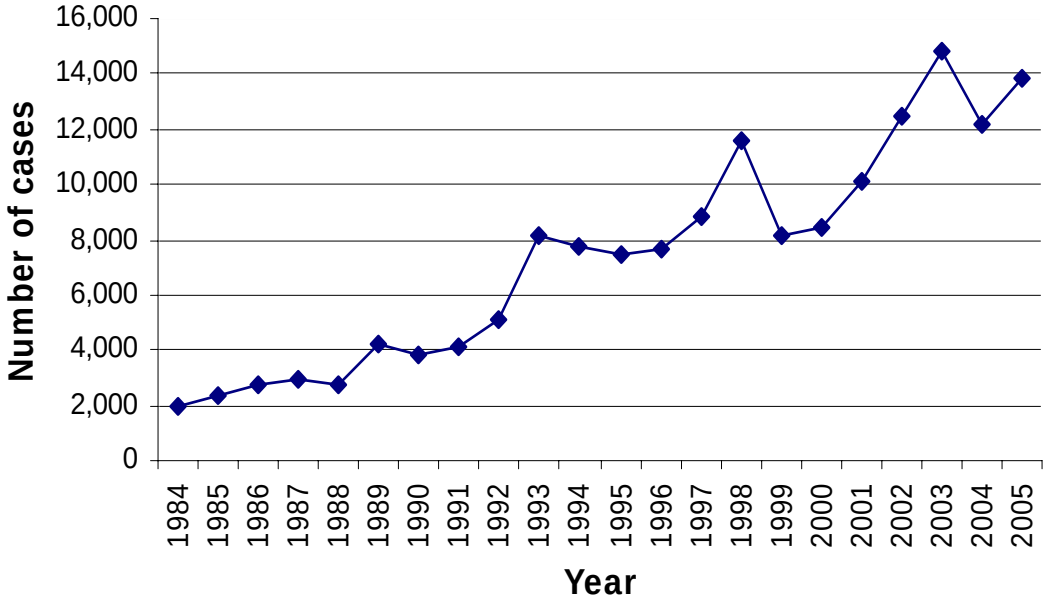
The study of the number of cases of infectious intestinal disease in New Zealand (Lake *et al.,* 2000) used a reported:unreported ratio for campylobacteriosis of 1:7.6 derived from a prospective UK study (Wheeler *et al.* 1999). This suggests that the total rate of campylobacteriosis in New Zealand using the most recent data is approximately 3,000 per 100,000.

The peak in notifications seen in 1998 seems to have been the result of a deviation from the normal seasonal trends observed for this disease. Normally the rate drops in the winter months, but in 1998 this did not occur leading to the abnormally high annual figure. The figures from 2002 to July 2006 have now exceeded the 1998 rate.

The age distribution of cases is bimodal with peaks in the 1-4 years age group and 20-29 year group. In 2006, the highest age-specific rate occurred among children aged 1 – 4 years (511.2 per 100,000; 1105 cases). The rate for 20 to 29 year olds was 501.8 per 100,000; 2442 cases. The lowest rate was in the 10 to 14 age group at 198.1 per 100,000; 576 cases (ESR 2006a).

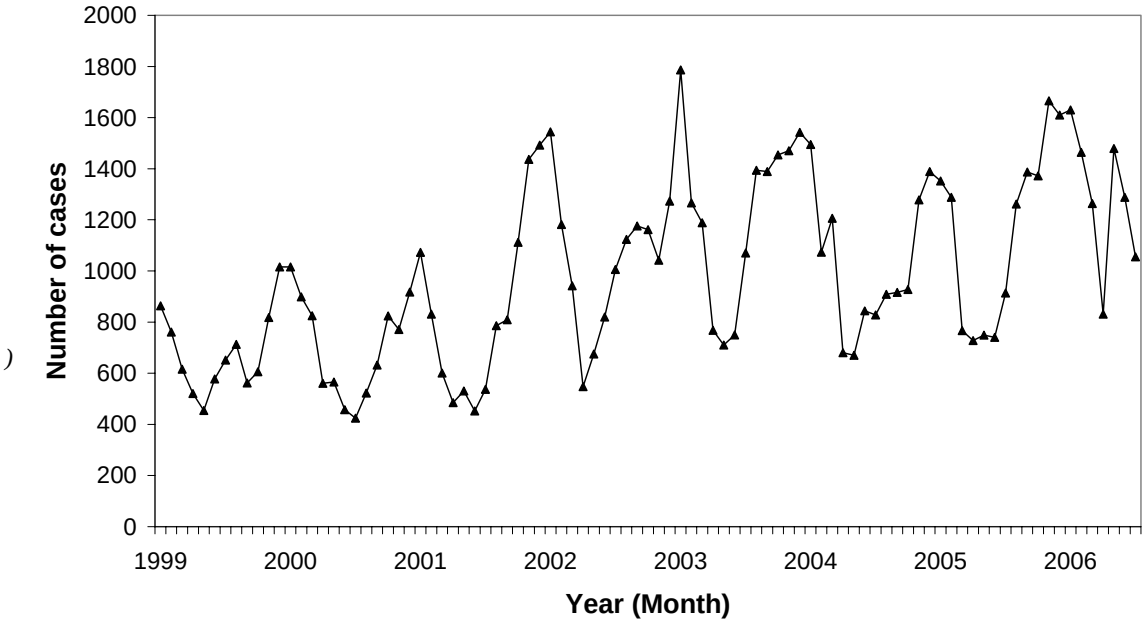
The reported rates of campylobacteriosis in Maori and Pacific Peoples populations in 1993 were approximately one fifth of the rate for Europeans (Lane and Baker, 1993). For cases where ethnicity is recorded (78.4% in 2005), the rate amongst New Zealanders with European ethnicity is highest (363.4 per 100,000 in 2004). This is higher than for other groups (Maori: 124.1 per 100,000; Pacific Peoples: 65.9 per 100,000, Other ethnic groups: 234.2 per 100,000). The reasons for these differences are unknown, reporting factors may well play a role (ESR, 2006a).

Figure 3: Campylobacteriosis notifications by year 1984 – 2005



Reproduced from ESR (2006a)

Figure 4: Campylobacteriosis notifications by month January 1999 – July 2006



Prepared from ESR data (2006a; ESR 2006b)

New Zealand's reported rate of campylobacteriosis is high by developed world standards (370.3 per 100,000 in 2005), as shown in section 6.2.1. However, such comparisons must be made with caution, as reporting practices may differ between countries.

6.1.2 Clinical consequences of *Campylobacter* infection

Hospitalisation and fatality rates for notified cases of campylobacteriosis in New Zealand are given in Table 11. These outcomes are not always reported for each case, so percentages are expressed in terms of the number of cases for which outcomes are known. For 2005, 57% of cases had hospitalisation data recorded.

Table 11: Outcome data for campylobacteriosis in New Zealand

Year	Hospitalised cases	Fatalities	Reference
1997	319/6440 (5.0%)	2/8848 (0.02%)	ESR, 1998
1998	369/8805 (4.2%)	2/11578 (0.02%)	Perks *et al.*, 1999
1999	304/5701 (5.3%)	1/8173 (0.01%)	Kieft *et al.*, 2000
2000	373/5887 (6.3%)	3/8430 (0.04%)	Lopez *et al.*, 2001
2001	393/6356 (6.2%)	1/10148 (0.01%)	Sneyd *et al.*, 2002
2002	515/7735 (6.7%)	1/12489 (0.01%)	Sneyd and Baker, 2003
2003	633/8302 (7.6%)	0/14786	ESR, 2004
2004	499/6542 (7.6%)	0/12212	ESR, 2005a
2005	635/7887 (8.1%)	1/13839 (0.01%)	ESR, 2006a

6.1.3 Outbreaks

Overseas, campylobacteriosis accounts for only a small proportion of total reported outbreaks (0.5 to 6%). Indeed, the disease is regarded as occurring mostly in sporadic cases and not in outbreaks. Pebody *et al.* (1997) comment that although *Campylobacter* in England and Wales has been the commonest enteric pathogen isolated from humans since 1981, only 21 general outbreaks of campylobacteriosis were reported between the years 1992 to 1994. This is generally considered to be due to the fact that *Campylobacter* do not multiply in air or at room temperature, so poor food handling is less likely to result in multiplication and consequent spread of the organism. In addition, the relatively long incubation period means that outbreaks are less likely to be recognised and reported (Frost, 2001).

In contrast, the New Zealand data summarised in Table 12 show that *Campylobacter* is identified as the causative agent in around 10 - 15% of reported outbreaks. There are several possible explanations for this; 1) the result is genuine 2) New Zealand is better at detecting outbreaks caused by campylobacteriosis or 3) the differences in rates are actually attributable to different surveillance philosophies. The average number of cases per outbreak was 3.3. It should be noted that these figures represent all outbreaks of campylobacteriosis and not just those attributed to red meat.

Table 12: Total number of reported outbreaks and cases for which *Campylobacter* was identified as the causative agent in New Zealand 1998-2005

Year	No. of outbreaks	Percent	No. of cases	Percent	Reference
1998	47	15.0	241	11.3	Naing *et al.*, 1999
1999	57	15.8	189	8.0	Perks *et al*., 2000
2000	37	12.8	144	6.3	Lopez *et al*., 2001
2001	56	14.4	301	13.0	ESR, 2002
2002	50	14.8	237	8.2	Boxall and Ortega, 2003
2003	42	12.35	140	5.0	ESR, 2004
2004	31	9.5	130	3.2	ESR, 2005a
2005	47	13.6	252	10.3	ESR, 2006b

Outbreaks of campylobacteriosis associated with red meat consumption and reported from 1997 to August 2004 have been summarised in Table 13.

Table 13: New Zealand outbreaks of campylobacteriosis with either epidemiological (suspected) or laboratory (confirmation) linkage with red meat consumption January 1999-August 2004

Outbreak Number*	Food implicated	Number ill	Link to red meat consumption
AK1999128	Kebabs -lamb & chicken	16	Epidemiological
AK2001082	Meat patties BBQ undercooked	2	Epidemiological
AK2001187	Steak BBQ raw/medium, chicken	2	Epidemiological
AK2001202	Sausages & chicken BBQ meal	2	Epidemiological
AK2002044	Ham/ pizza	3	Epidemiological
CB2000001	Sausages BBQ undercooked	6	Not stated
RO1999015	Bacon BBQ undercooked	3	Not stated
MW2003002	Ham, salami, steak, sausages	2	Epidemiological
AK2003018	Cold meats	2	Epidemiological
AK2003144	Lamb enchilada/ lamb stroganoff	2	Epidemiological
#CB2003013	Cocktail sausages	3	Laboratory
AK2003128	Chilli con carne	2	Epidemiological
AK2003206	Beef teriyaki & another beef dish	4	Epidemiological
CB2004003	Cold roast beef	8	Epidemiological
AK2004063	Mixed kebab –lamb & chicken	2	Epidemiological
AK2004090	Meatballs, hamburger	2	Epidemiological
AK2004115	Mince meal home cooked	3	Epidemiological

See outbreak comments below; cross contamination likely cause

Of the 17 reported outbreaks from January 1999 to August 2004, five highlighted barbecuing as a factor.

It should be noted that the evidence linking the food with human infection in most of these outbreaks is relatively weak; based mainly on epidemiological evidence, specifically a common exposure to an implicated source rather than a case-control or other study. In seven of these outbreaks a critical control point failure was also identified. Laboratory confirmation was obtained in only one outbreak, as described below. Greater use of typing would help to strengthen linkages between implicated foods and outbreaks.

One outbreak of campylobacteriosis in Christchurch (#CB2003013) has been attributed to cross contamination of a red meat product (Graham *et al*., 2005). In this incident, two cases were attributed to the consumption of pre-cooked cocktail sausages, and there was a secondary case. The pre-cooked sausages were purchased from a butcher's shop along with other items. The contamination probably came from raw chickens being repackaged in the same area of the butcher's shop, cross contaminating the sausages with *C. jejuni.* The sausages were repackaged in the home into smaller lots and frozen. The following day, a bag of sausages was removed from the freezer and defrosted in the refrigerator. Several of the sausages were then consumed without reheating. A sample of the sausages from the freezer of the cases was examined by ESR along with three case faecal specimens. *C. jejuni* Penner complex 4 were isolated from the three faecal and one sausage sample, PFGE analyses showed all four isolates were indistinguishable. The organism was only isolated from sausage rinse samples, suggesting external contamination only. Two other cases occurring a month before were, by chance, linked to the outbreak by DNA typing information. These

isolates were obtained from a study underway in Christchurch at the same time that the outbreak occurred. The authors noted that the sausage samples which tested positive for *C. jejuni* had been stored frozen in the home of the cases for approximately 10 days prior to testing. Detection of the organism indicates that the numbers present were initially potentially very high. The authors concluded that testing of frozen foods suspected in outbreaks of campylobacteriosis is of value given contemporary culture methods.

6.1.4 Case control studies and risk factors

Two case control studies of risk factors for campylobacteriosis have been carried out in New Zealand.

A case-control study of one hundred each of cases and controls (Ikram *et al*., 1994) was conducted in the summer of 1992-1993 in urban Christchurch. The results of the study found no risk associated with handling of raw beef, pork, mutton or lamb and no risk associated with the consumption of barbecued beef, pork, mutton or lamb chops. The study concluded that poorly cooked or handled chicken was a significant source of human *Campylobacter* infection, (eating barbecued chicken had an odds ratio of 3 and a confidence interval of 0.99 – 9.34). Drinking water from a rural water source constituted some risk (OR-2.7, CI 0.89, 8.33), but this was not statistically significant. Amongst the non-food exposures, overseas travel, rainwater as a home water source, and contact with faeces of puppies (in the home) or cattle were associated with campylobacteriosis. Occupational contact with bovine carcasses was also strongly associated with disease.

The other case control study (Eberhart-Phillips *et al.,* 1997) is also known as the MAGIC study. Data were collected over a 9 month period from 621 cases notified with *Campylobacter* infection and the same number of matched controls. Interviews of cases and controls were carried out (approximately 85% of subjects were classed as urban) in four centres with high notification rates of campylobacteriosis (Auckland, Hamilton, Wellington and Christchurch) during 1994 and 1995. The results for red meat were reported as generally not significant and in the case of 'other minced beef', 'boiled minced meat' and 'cooked ham/roast pork', the results suggested a protective effect. Only barbecued lamb was significant in terms of a red meat risk factor (OR 1.40, 95% confidence level 1.03-1.91). The main outcome of the study was that the risk of campylobacteriosis was strongly associated with recent consumption of raw or undercooked chicken (adjusted odds ratio = 4.69, 95% confidence interval = 3.02, 7.28)

Auckland Healthcare has also undertaken three investigations into *Campylobacter* in recent years. An outbreak in late 1996 prompted a case-control investigation into risk factors for endemic campylobacteriosis during that period (Bloomfield and Neal, 1997). An outbreak at a family barbecue (17 cases) in October 1998 was investigated by a retrospective cohort study (Bishop, 1998). The third case-control study (Calder *et al*., 1998) took place following a power shortage in Auckland in February 1998 and a sharp increase in human campylobacteriosis notifications. None of the studies found any significant increased risk with the consumption of red meat

6.1.5 Serotypes and genotypes causing human disease in New Zealand

Penner serotyping based on the heat stable antigen has been conducted for 1130 *Campylobacter* isolates obtained from human cases in New Zealand between 1996 and 2001. The serotypes identified include: 1,44 (16% of serotypes isolates), 2 (23%), 4 complex (15%), 5 (0.6%), 10 (0.6%), 19 (0.8%), 23 (8%), 35 (1.3%), 37 (4%), 41 (0.5%) (Lake *et al.*, 2004). Although the source of these serotypes is unknown, the most prevalent (1,44, 2 and 4 complex) are also the most common in UK cases. A UK study examined a large dataset of Penner serotypes of *C. jejuni* from cases of human campylobacteriosis (Miller *et al.*, 2005a). The most prevalent serotypes were HS:4 complex, HS:2, and HS:1,44 (53.8% of all cases).

Certain serotypes, particularly Penner serotype O:19 and O:41 have been associated with GBS (AIFST, 2003) but this was not confirmed in a USA case control study, in which no specific serotypes were associated with GBS (Rees *et al*., 1995).

A search of the PulseNet New Zealand database was carried out for human and red meat serotypes (Angela Hough, ESR, pers. comm, June 2005). As of June 2005, 302 isolates of *Campylobacter* from human cases have been serogenotyped (*SmaI* enzyme) and recorded on the database. Only small numbers of isolates from red meat sources (<20 each from sheep, beef and pork meat) have so far been analysed by the PulseNet system so no conclusions regarding commonality can be drawn.

6.2 Adverse Health Effects Overseas

6.2.1 Incidence

Data on the incidence of reported cases of campylobacteriosis overseas have been summarised in Table 14. New Zealand's reported rate is high by international standards, although some differences may be due to reporting practices.

Table 14: Comparison of reported campylobacteriosis incidence between countries

Country	Period	Rate /100,000	Reference
New Zealand	**2005**	**370.3**	**ESR, 2006a**
Australia*	2003	116.5	Miller *et al*., 2005b
Canada	2000	40.1	Health Canada, 2003
Denmark	2002	82	Anonymous, 2003
Iceland	1999 2000	116 33	ACMSF, 2004
Ireland	2001	35.5	NDSC, 2002
England and Wales	2001	107.6	NDSC, 2002
Northern Ireland	2001	52.4	NDSC, 2002
Scotland	2003	86.6	SCIEH, 2004
USA	2002	13.4[#]	CDC, 2003

*Excludes New South Wales that does not report campylobacteriosis.

[#]Data collected from 9 US States (Foodnet) which represents 13% of total USA population

Notifications are generally highest in spring and summer months, both in New Zealand and overseas (Frost, 2001; Lane and Baker, 1993).

In the UK, *Campylobacter* infection is the most prevalent reported foodborne disease. In 2000, 62,867 cases of *Campylobacter* were reported, with 50,773 acquired within the United Kingdom (see website: http://www.food.gov.uk/science/sciencetopics/microbiology/58736). *C. jejuni* is the predominant species with *C. coli* the majority of the remainder. To achieve the Food Standard Agency target of reducing UK acquired foodborne illness by 20% by 2006, reducing *Campylobacter* infection is a priority.

In the USA, human *Campylobacter* infection has been steadily declining in incidence to the extent that the USA 2010 health objective to reduce campylobacteriosis to 12.3 per 100,000, looks to be achievable.

The incidence of the disease has also been declining in Scotland (SCIEH, 2004) and Ireland (NDSC, 2002). The rates in Ireland have declined from 57.5 per 100,000 in 1999 and 44.5 in 2000 to 35.5 in 2001. Despite the decline, campylobacteriosis is still the main cause of gastrointestinal infection in Ireland. The disease follows a similar pattern in Ireland as in other temperate climates, i.e. more frequently occurring in very young children, male cases and in the summer months.

6.2.2 Contribution to outbreaks and incidents

Estimates of the proportion of outbreaks due to *Campylobacter* overseas (0.5 to 6%) are given in Table 15. The low percentages reinforce the sporadic nature of this illness.

Table 15: Contribution of *Campylobacter* to reported foodborne disease outbreaks, incidents and cases overseas

Country	Year	No. (%) Outbreaks	No. (%) incidents or cases	Reference
Canada	1984	NR	19 (1.6) incidents	Todd, 1992
England and Wales	1992-1994	19 (1)	NR	Djuretic *et al*., 1996
Germany	1993-1998	21 (2.3)	NR	www.who.it/docs/fdsaf/fs_survprog.htm
Sweden*	1992-1997	29 (6)	31 (6) incidents 335 (3) cases	Lindqvist *et al*., 2000
UK	1995	4 (0.5)	140 (0.7) cases	Evans *et al*., 1998
UK	1996	8 (1.1)	99 (0.5)	Evans *et al*., 1998
USA	1993-1997	25 (0.9)	539 (0.6) cases	Olsen *et al*., 2000

* Of 13 outbreaks where a food was implicated, 11 were attributed to chicken
NR = Not reported

Overseas outbreaks of campylobacteriosis associated with red meat consumption that have been reported in the scientific literature have been summarised in Table 16.

Table 16: Overseas campylobacteriosis outbreaks associated with red meat consumption

Country	Food implicated	No. ill	Reason for food implicated	Reference
England	Mixed meat fajitas and salad	8	Statistical analysis, possibility of cross contamination	Pebody *et al.,* 1997
Germany	Cabbage stew with beef as part of a school meal	556	Epidemiological	Steffen *et al.,* 1986
Japan	Vinegared pork as part of a school meal	800	Considered "most likely" source by author	Yanagisawa, 1980
USA	Raw beef and/or raw egg	19	Not stated	Finch and Blake, 1985

While no details were given, three outbreaks of campylobacteriosis were attributed to contaminated red meat/meat products in England and Wales during 1995-1999 (Frost *et al.,* 2002), one of these outbreaks is summarised above (Pebody *et al*., 1997). An outbreak among soldiers has also been attributed to the consumption of raw hamburger in the Netherlands (Oosterom *et al.,* 1980).

6.2.3 Case control studies

There are several case control studies of campylobacteriosis conducted overseas that contain information on red meats. They are summarised below in Table 17.

Table 17: Case control studies overseas containing information on *Campylobacter* in red meat

Country	Risk factor	Odds ratio (CI)	Reference
England and Wales	Occupational exposure to raw meat*	9.37 (2.03-43.3)	Adak *et al.,* 1995
Netherlands	Pork consumption	P=0.048	Oosterom *et al.,* 1984
Norway	Eating at a barbecue Eating undercooked pork	3.2 (1.7-6.1) 4.1 (1.7-9.9) 9.0 (0.9-91.7) 37.0 (1.6-830.9) Two models used.	Kapperud *et al.,* 2003
Sweden	Consumption of red meat at a barbecue[1]	2.3 (1.3-3.9) 4.1 (1.5-10.9) Two models used.	Neiman *et al.,* 2003
Sweden	Eating pork chops Eating pork loin	2.0 (1.2-3.6) 1.8 (1.1-3.1)	Studahl and Andersson, 2000
Southeastern Norway	Consumption of sausages at a barbecue	7.6 (1.8-31.9)	Kapperud *et al.,* 1992

CI = confidence interval

* Does not exclude chicken as a meat

[1] Population attributable risk for this risk factor 23.8% (15.9-33.9% CI)

The association between the consumption of pork products and campylobacteriosis is interesting because, as stated above, *C. coli* is the *Campylobacter* species most often associated with pork, yet human cases are caused by *C. jejuni* on around 90% of occasions.

A focused study on the consumption of meats in the week prior to *C. jejuni* infection determined that the meats with associated risk were poultry and seafood (Harris *et al.,* 1986b). Red meats included in the study were pork, beef and lamb/mutton, processed or unprocessed.

In the UK, detailed surveillance of clinical campylobacteriosis cases took place from May 2000 until April 2003 (Health Protection Agency, 2003). Reference typing has focused on a Sentinel Surveillance Scheme in 22 District Health Authorities (12.5 million people) which represent approximately 15% of all laboratory confirmed cases in England and Wales. In 2001, Scotland and Northern Ireland joined the scheme.

One of the ways in which the data were analysed was a case-case study (The *Campylobacter* sentinel surveillance scheme collaborators, 2003). Risk factors for "cases" (those that may have been involved in an outbreak) were compared against "controls" (apparently sporadic cases if campylobacteriosis). In analysing data for exposures in the fortnight prior to illness in the household, the consumption of organic meats in the winter (December, January, February) was a significant risk factor for cases [i.e. in outbreaks] (OR 6.86, 95% CI 1.49-31.69). Other risk factors included contact with a pet suffering from diarrhoea or a farm visit. The same risk factor was not identified in the examination of illness in the community. However, the definition of organic meat was open, and may have included the consumption of organic chicken or turkey, bearing in mind the Christmas season.

In another case-case study, this time between *C. coli* and *C. jejuni* infections, data from 7,360 campylobacteriosis cases in England and Wales were analysed (Gillespie *et al.,* 2002). The first year's data were analysed to compare cases of *C. coli* infections against cases of *C. jejuni* infections. The results revealed significant differences in risk behaviour associated with the two predominant species and produced a number of hypotheses which merit further investigation. Those which concerned meat products were;

1) those with *C. coli* infections were more likely to have consumed pâté than those with *C. jejuni* infections, and
2) retired people with *C. coli* infections were more likely than those with *C. jejuni* infections to have consumed meat pies.

6.2.4 Risk assessment and other activity overseas

Disease caused by infection with *Campylobacter* is recognised as an increasing problem in many countries, and national and international efforts are being made to assess and control the problem.

The Danish Veterinary and Food Administration have published a "Risk Profile for Pathogenic Species of *Campylobacter* in Denmark" (Danish Veterinary and Food Administration, 1998). The report was initiated following concern about the more than two-fold increase in human cases of campylobacteriosis during the 1990s. Cases occur most frequently in late summer and autumn, with 10-29 year olds most commonly affected.

The Risk Profile also described a case-control study carried out in Denmark from 1996 to 1997. Significant risk factors were: travel abroad, insufficiently heat treated poultry (OR 5.5, p=0.003), meat prepared by grill or fire (OR 2.3, p=0.002) and poor quality drinking water from a private well (OR 3.0, P=0.008). These risk factors were considered to explain approximately 50% of the human cases (5-8% insufficiently heat treated poultry, 15-20% meat prepared by grill, 5-8% to drinking water, and 15-20% to journeys abroad). The Risk Profile indicated that 20-30% of samples of table fresh poultry were positive for *Campylobacter*, whereas only 1% of samples of beef and pork were positive.

The conclusion of this Risk Profile was that a risk assessment concerning *C. jejuni* in foods and water should be conducted, with the caveat that significant data gaps would have to be filled during the assessment. These data gaps included other possible sources of infection, other risk factors, and typing methods.

The European Union launched a programme to control foodborne zoonoses in 2001; it has control of *Salmonella* as its priority. As a lead up to the development of these control efforts, a review of information on foodborne zoonoses in Europe was carried out (Scientific Committee on Veterinary Measures Relating to Public Health, 2000). Their report included a risk assessment for thermotolerant *Campylobacter*, which in size and content resembles a risk profile. Although the reported incidence of campylobacteriosis in Member States varies widely from 9.5 in Spain to 108 per 100,000 in Scotland in 1997 (probably due to differences in surveillance systems), a general increase in cases was noted. This, along with the increasing fluoroquinoline resistance amongst *Campylobacter* isolates means that the risk to humans will increase in the future.

A number of risk factors were identified, which were the same as mentioned in other studies, but no attempt was made to assign the proportion of cases caused by these risk factors. However, to reduce the risk in the future, more work is required to elucidate the causes of infection. A reduction in the prevalence of *Campylobacter* in food was also recommended.

6.3 Estimate of Risk for New Zealand

Survey results described in Section 5.1.1 reveal a low to moderate (0-10%) prevalence of contamination of red meat by *Campylobacter*. The numbers of bacteria on red meat at retail appear to be low. It seems likely that the generally drier processing procedures for red meat (compared to poultry processing) reduce the numbers of bacteria present. Normal refrigerated storage temperatures will prevent growth, although *Campylobacter* survive well at these temperatures.

The risk of exposure to *Campylobacter* will be increased by the frequent consumption of red meat by New Zealanders. A number of small outbreaks of campylobacteriosis have been associated with red meat dishes in New Zealand although the evidence is relatively weak. Barbecuing was identified as a factor in several of these outbreaks.

Red meat consumption is not strongly associated with increased risk in case control studies in New Zealand, although the MAGIC case control study found that barbecued lamb was a significant risk factor, and several overseas case control studies have found significantly elevated risk factors for consumption of barbecued red meat. Cooking will readily destroy the organism, but barbecuing may have a greater potential for undercooking. In addition, cross contamination remains a potential exposure route.

The outbreak data and the case-control studies do not provide strong evidence for red meat as a transmission route for campylobacteriosis in New Zealand, apart from exposure through barbecues. Nevertheless there are good data indicating low but consistent prevalence of contamination across pork, beef, and sheep meat, and red meat is a frequently consumed food. On this basis it seems reasonable to assign red meat consumption as an identified but minor risk factor for exposure to *Campylobacter* in New Zealand.

6.4 Risk Categorisation

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

In the study of the incidence of foodborne infectious intestinal disease in New Zealand (Lake *et al*., 2000) it was assumed that 65% of campylobacteriosis was foodborne. This was supported by a New Zealand case control study in which the population attributable risk percentages associated with consumption of foods included in the study totalled 48% (Eberhart-Phillips *et al*., 1997), and USA estimates of the proportion of cases due to foodborne transmission of 55-70% (Buzby *et al*., 1996) and 80% (Mead *et al*., 1999).

The reported rate of campylobacteriosis in 2006 in New Zealand was 370.3 per 100,000 population, while the total rate is estimated as approximately 3,000 per 100,000 (see Section 6.1.1). If 65% of this is considered to be foodborne, the foodborne rate is approximately 1,950 per 100,000. In the Risk Profile for *Campylobacter* in offal this food/hazard combination with high prevalence, low consumption attributes was assigned to incidence Category 2 (10-100 case per 100,000). It seems reasonable to assign *Campylobacter* in red meat, with low prevalence high consumption attributes, to the same category.

The proportion of severe outcomes (hospitalisation, long term sequelae, and death) resulting from campylobacteriosis is approximately 0.3% (Lake *et al*., 2000) placing this infection in the lowest severity category.

6.5 Summary

Food/hazard combination	Severity	Incidence	Trade importance	Other considerations
Campylobacter in red meat	3 (<0.5% serious outcomes)	2 (10-100 per 100,000)		

7 RISK MANAGEMENT INFORMATION

7.1 Relevant Food Controls

Options for managing the risk from *Campylobacter* in red meat include:

- Attempt to reduce the prevalence of the hazard in animals,
- Control of the hazard during or following processing, and
- Elimination of the hazard by the end user i.e. consumers and the food service industry.

7.1.1 On farm control.

While *C. jejuni* and *C. coli* are commonly found in ruminants' intestinal tracts (*C. coli* in pigs), these organisms are not associated with any specific animal diseases with the exception of sporadic abortion in sheep. This means that farmers do not take any specific control measures outside of general hygiene precautions.

The *Campylobacter* spp. that has been found to cause sporadic abortion in sheep is *C.fetus* subsp. *fetus.* Two commercial sheep vaccines are available; Campyvax3-Agvax and Campylovexin Shering Plough, however they do not offer cross protection for *C. jejuni* or *C. coli* (Graeme Jarvis, Meat and Wool New Zealand Ltd, personal communication 27/09/04). *C. jejuni* has been implicated as a cause of sporadic abortion in sheep although there are no reliable data on how important it is, but in comparison with *C. fetus* subsp. *fetus* it is minor. These reports originate in Australia, particularly Tasmania where it is common practice to feed grain to sheep on the ground, attracting birds to the feed and bird droppings into the vicinity. Sheep in New Zealand are seldom, if ever, fed grain (David West, Massey University, personal communication 28/09/04).

7.1.2 Control during or after processing

Since campylobacters are frequently found in the intestines of farm animals, the spread of the bacteria from intestine to carcass depends largely on the hygiene precautions taken during slaughtering and dressing (AIFST, 2003). Examples include ensuring clean animal hides and fleeces, reducing the potential for cross contamination during dehiding, and care taken during evisceration.

During evisceration of sheep, leakage from the rumen is controlled by clipping of the sheep oesophagus close to the rumen. The alimentary tract is dropped into the gut cavity, with control of spillage being provided by sphincter contraction. Some operations do insert plugs down the anus for scoury mobs to control contamination, with others they may plugs in a more wide spread fashion. The speed of New Zealand sheep processing facilities would not readily allow colon tying (Neil Smith, personal communication, MIA, January 2007).

In cattle, bagging of the rectum (with a plastic bag) is standard practice in New Zealand to control contamination (Neil Smith, personal communication, MIA, January 2007).

There are already a number of measures in place during the slaughtering process to minimise faecal material being transferred from the gut to the carcass. These hygiene measures are aimed at preventing contamination of the carcass with pathogens and other bacteria such as salmonellae and STEC. The UK Advisory Committee on the Microbiological Safety of Food

(ACMSF, 2004) considered that the control measures required to achieve minimisation of *Campylobacter* contamination will be essentially the same as for organisms such as *Salmonella* and STEC. Consequently they did not consider that there was a need for *Campylobacter* specific measures.

In an Irish study (Minihan *et al*., 2004), appropriate abattoir hygiene couple with trimming and a whole carcass hot water wash (62°C) resulted in none of the 109 swabs of dressed carcasses proving positive for the organism, even though there was a high prevalence in the faeces of the cattle.

Traditional inspection procedures during meat processing involves palpation and incision to observe certain body parts. In Canada, the USA and Australia, meat inspection procedures have already changed in favour of less palpation and incisions. One of the primary drivers for the change has been the production of microbiologically cleaner carcasses.

In Europe, new European Union legislation proposes meat inspection techniques that favour observation and greater ante-mortem inspection;

"Reports indicate that the post-mortem inspection of pre-slaughter of apparently healthy animals detects only 20% of all the macroscopic lesions that are actually present in 1% or less of animals. On the other hand, food animals also carry pathogenic micro-organisms in their gastrointestinal tract and/or on coat without any signs of disease ante-mortem, or visible lesions post-mortem. During slaughter and dressing procedures, these pathogens, including *E. coli* O157 and other VTEC, *Salmonella* spp., *Campylobacter jejuni* and *Listeria monocytogenes*, can be directly or indirectly transferred onto the meat surface, but will not be visible to the meat inspection staff during conventional meat inspection of sheep/goats." (EFSA, 2004)

Previous meat inspection techniques have been based upon the requirements of European Directive 64/433/EEC, which involved a high physical degree of palpation, certain muscular cuts and incisions of specific lymph nodes. These techniques have been recognised as increasing the risk of cross contaminating the carcass. The new techniques are aimed particularly at younger animals such as veal, lambs and kid goats and are much less physical. Further details of the Scientific Panel Opinions can be found at the following websites;

Revision of meat inspection of veal calves:
http://europa.eu.int/comm/food/fs/sc/scv/out65_en.pdf

Biological hazards and meat inspection procedures for lambs and kid goats:
http://www.efsa.eu.int/science/biohaz/biohaz_opinions/452_en.html

In New Zealand, a project is currently underway to examine the meat inspection procedure for each livestock species and how the procedure can be modified in order to produce a microbiologically cleaner carcass (Bob Jackman, NZFSA, personal communication, 14/10/04). The project has concentrated initially on lamb inspection. Currently the inspection procedure for lambs involves a high degree of physical palpation, in the abdominal and thoracic cavities and of the external carcass. Two issues in particular for lambs are the inspection of the ischiatic lymph node situated in the pelvic cavity, where faecal material is often present and inspection of the deeply situated popliteal lymph node for the presence of lymphadenitis caseosa (*Corynebacterium pseudotuberculosis*).

Campylobacters are highly sensitive to drying, an effect seen in a study on the chilling effect on *Campylobacter* on pork skin, (Bracewell *et al*., 1986, see section 3.1) where blast chilling reduced numbers only slightly more than conventional chilling. Table 18 summarises the effect of chilling on carcasses at abattoirs from a number of studies.

Table 18: Effects of chilling (drying) on campylobacters on carcasses and intestines at abattoirs

Type	Country	Intestine		Carcasses Before chilling		Carcasses After chilling	
		No. samples	% positive	No. samples	% positive	No. samples	% positive
Calf	Australia	24	54	65	97		
Calf	NZ	50	52	30	20	30	10
Cattle	Australia	96	12.5	114	12.3	34	2.9
Sheep	Australia	580	2.8	377	19		
Pork	UK			100	59	100	2
Pork	USA			112	12.5	100	0

(Source: Wallace, 2003)

7.1.2.1 The Animal Products Act

Risk Management Programmes (RMPs) are part of the emerging food assurance system in New Zealand. They form part of the Animal Products Act (APA) 1999. These will eventually be integrated with the Food Safety Programmes (FSPs) and Product Safety Programmes (PSPs) required by the Food Act 1981.
See website: http://www.nzfsa.govt.nz/dairy/subject/animal-products-act/index.htm

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

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

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

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

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

- risk management programmes;

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

The Animal Products (Ancillary and Transitional Provisions) legislation has enabled a staggered implementation of RMPs under the Act. This schedule was developed by NZFSA. All animal product primary processing businesses are required to have a RMP except those exempt under the Act or exempt under the Animal Products (Exemptions and Inclusions) Order 2000.

A risk management programme is a documented programme to identify and manage biological, chemical and physical hazards. The programme is to be based on the principles of Hazard Analysis and Critical Control Point (HACCP): identifying the hazards, the systems of control, and demonstrating that the controls are effective. Risk management programmes are to be designed by individual businesses for the animal materials used, the processes performed and the product range produced.

7.1.3 Consumers

In New Zealand, general consumer advice for control of pathogens in red meat is based upon the clean, cook, cover, chill campaign. The website; http://www.nzfsa.govt.nz/consumers/food-safety-topics/foodborne-illnesses/advice/background.htm, contains this advice for cooking;

"Chicken, meat patties and sausages need to be cooked thoroughly. Raw meat is a prime source of *Salmonella* and *Campylobacter*. One way of ensuring this is to cut the food and check that there are no traces of pink in the meat and that the juices are not pink either. It is wise to pre-cook these items before barbecuing".

The use of colour perception to judge adequate cooking is controversial. The latest advice from the USA government is that the USDA; "found that frozen meat turns brown at lower temperatures than hamburgers prepared from fresh meat. Some of these hamburgers may have been incorrectly perceived as cooked to a safe temperature based on their color. FSIS now recommends using a thermometer to cook hamburgers to 160 degrees [Fahrenheit]". Source: http://ers.usda.gov/Briefing/consumerfoodsafety/consumption/index.htm

Images of "pink but cooked" and "brown but undercooked" burgers have been used by the USDA in a consumer education campaign to get the message across about internal temperature rather than colour perception; http://www.hi-tm.com/Documents2000/Pinkburger.html

A summary of the research carried out on perception of colour to determine meat 'doneness' can be found at the following website; http://www.hi-tm.com/Documents/Meat-color.html.

7.2 Economic Costs

Cases of campylobacteriosis caused by foodborne transmission have been estimated to cost $40,136,000 annually, which comprises 73% of the total economic cost of foodborne infectious intestinal disease in New Zealand (Scott *et al*., 2000). This is by far the majority of the cost of foodborne illness; all the other nine foodborne enteric diseases included in the study each represented costs of less than 10% of the total. The number of cases and

outcomes used for this estimate were based on an average of notification and hospitalisation data from 1991 to 1998 (Lake *et al*., 2000). This estimate was based on several assumptions, the most important of which was that 65% of all cases of campylobacteriosis were caused through foodborne transmission (see Section 6.4 for supporting references). The estimated dollar value includes direct and indirect medical costs, the value of productive days lost, and the statistical value of mortality, but not the value of lost quality of life.

This estimate covers all potential food vehicles. No data are available on the proportion of transmission due to red meat alone.

The cost estimate of $40,136,000 assumed that the ratio of notified (visit a GP) to unreported (community) cases of campylobacteriosis was 1:7.6, based on data from a prospective English study (Wheeler *et al*., 1999). The notification figure for this estimate was taken from the most up to date reported cases rate at the time, i.e. 1998 at 320 per 100,000. In the last few years, the reported cases rate has increased, and consequently the cost estimate will be higher. Campylobacteriosis still represents the majority of infectious intestinal disease costs.

7.3 Other Transmission Routes

7.3.1 Other transmission routes: food

Undercooked poultry has been the transmission vehicle most commonly identified in case control studies of campylobacteriosis. The prevalence of *Campylobacter* in retail samples of lamb, ox and pig liver in New Zealand is also high (Hudson, 1997), and unpasteurised milk has been associated with several outbreaks in the United Kingdom (Frost, 2001). In New Zealand *Campylobacter* has also been isolated from watercress and was the subject of a Director-General of Health statement in 2000.

The high prevalence of *Campylobacter* in raw chicken may cause direct infection of food handlers, as well as indirect infection via food contact surfaces (Humphrey *et al*., 2001). It has been generally assumed that *Campylobacter* do not persist outside of the animal reservoirs, but more sensitive detection methods have recovered the bacteria at low levels from surfaces 24 hours after contamination. In general though, conditions common in kitchens such as high or low temperatures and exposure to drying on kitchen surfaces will induce sublethal injury (Humphrey *et al*., 2001). Cross contamination from chicken to domestic kitchen surfaces has been demonstrated (De Boer and Hahné, 1990; Cogan *et al*., 1999) and an outbreak of campylobacteriosis in the United States involving 14 people was attributed to cross contamination between raw chicken and lettuce via a contaminated surface (Graves *et al*., 1998).

Against this theory are the results from case control studies that handling raw chicken and eating chicken at home can actually represent protective factors (Adak *et al*., 1995; Ikram *et al*., 1994).

7.3.2 Other transmission routes: environment

Campylobacter is widespread in the environment although clear routes for transfer from the environment to the consumer have yet to be identified (Jones, 2001). The seasonal incidence of intestinal disease caused by *Campylobacter* has characteristics suggesting waterborne transmission, and internationally several outbreaks have been associated with drinking water,

albeit usually from private, non-reticulated water supplies (Jones, 2001). In the UK from 1992 to 1994, the number of outbreaks associated with water outnumbered those associated with poultry (Frost, 2001).

In a study in New Zealand *Campylobacter* appears to be widespread (60-75% positive) but at low numbers in river water and shallow ground water, while roof waters were less commonly contaminated (29-37% positive) (Savill *et al*., 2001). A more recent year long survey of treated drinking waters, conducted by ESR for the Ministry of Health, has shown almost no contamination by *Campylobacter*, except for a small supply whose UV treatment process had failed (Nokes *et al*., 2004).

Recent studies carried out by ESR examining environmental reservoirs have shown that possums and rabbits are not significant carriers of the organism, at least in the areas studied (Devane *et al.,* 2005). None of the 260 possum faecal samples analysed were positive, while only one from 99 rabbit faecal samples was positive for *C. coli*.

A study of transmission routes in the Ashburton area investigating environmental and waterborne sources of *Campylobacter* has recently been completed (Baker *et al*., 2002; Devane *et al.,* 2005). The research was a joint effort by the Ministry of Health, ESR, the University of Canterbury, Crown Public Health, the Ashburton District Council and the EpiCentre. The focus was on comparing the genetic types of *Campylobacter* present in human cases, river water, animal faeces, meat animal offal and raw chickens. Results showed that exposure to ruminant faeces, either directly or indirectly, was probably responsible for most of the cases where isolates were obtained. However, this study was carried out in a largely rural area, as evidenced by the high degree of "rural exposure" reported by cases. The report concludes that the results from Ashburton may be like other rural areas of New Zealand, but may not represent those areas which are predominantly urban, i.e. where the greatest proportion of the population resides.

A New Zealand study (Meanger and Marshall, 1989) examined seasonal prevalence of *C. jejuni/coli* in the faeces of dairy cows, the results were 17/72 (24%), 33/106 (31%) and 11/95 (12%) during summer, autumn and winter respectively. Approximately half of the isolates were *C. jejuni* and the other half *C. coli*.

Given the previous data for New Zealand which are available, there may be two epidemiologies that predominate, a rural ruminant exposure epidemiology, and an urban one which may involve poultry and possibly other unknown exposures. This last point can be inferred from the large New Zealand case control study (Eberhart-Phillips *et al*., 1997), whose participants were principally located in the four main centres.

8 CONCLUSIONS

8.1 Description of Risks to New Zealand Consumers

8.1.1 Risks associated with red meat

Notified campylobacteriosis rates in New Zealand are high by world standards. A general increase in the number of notified campylobacteriosis cases has occurred from 1980 to 2005, although it should also be noted that the resident population of New Zealand has also increased significantly during this time period.

The prevalence of *Campylobacter* in red meat at the retail level has been the subject of a recent national survey. The results show that contamination in red meats is low (up to 10% in bobby veal) but consistent across beef, pork and sheep meat. These levels are consistent with results from overseas, and reflect the observation that although the prevalence of contamination in farm animals may be high, controls during processing effectively reduce contamination on carcasses.

Data on the numbers of *Campylobacter* present on red meats at the retail level are limited, but the available information suggests that numbers are relatively low. The effect of drying during processing is very important as this will reduce the numbers of *Campylobacter.*

Consumption of red meat has been declining for 20 years, with only pig meat consumption showing a small increase. This is compared with an approximate two-fold increase in poultry consumption over the same period. Nevertheless, a high proportion of the New Zealand population (78%) consumes red meat on a daily basis. Cooking will readily destroy the organism, although cross contamination remains a potential exposure route. Foods, such as hamburgers, where contamination may be spread throughout the food may pose more of a campylobacteriosis risk than other meats, such as steak, where contamination is only external.

No New Zealand data, and very little overseas data, have been found concerning *Campylobacter* carriage in deer, and contamination in venison.

Although a number of small outbreaks of campylobacteriosis have been linked to consumption of red meat dishes, red meats have not been identified as important risk factors in the case-control studies conducted in New Zealand. The exception is barbecued lamb, which was a significant risk factor, and it is worth noting that barbecues were mentioned in several of the red meat associated outbreaks. This could potentially be linked to the consumption of minced meat products at such events.

It seems reasonable to assign red meat consumption as a minor risk factor for exposure to *Campylobacter* in New Zealand.

8.1.2 Risks associated with other foods

Data cited in Risk Profiles for *Campylobacter* in poultry and *Campylobacter* in offal indicate that these meat types in New Zealand have a high prevalence of contamination by *Campylobacter*, but data for other foods are lacking. Raw or undercooked meat or fish, and

unpasteurised milk were identified as risk factors in the most recent New Zealand case-control study, but were less important than risk factors involving chicken consumption.

8.1.3 Risk assessment options

A quantitative risk assessment (QRA) would be feasible, particularly given data from the current New Zealand meat survey, combined with some data from overseas. Good consumption data are available, but information on cooking practices is scarce. Dose response relationships are available and could be used to produce a risk characterisation. Targeted projects to provide information on data gaps would greatly assist a QRA, and cooperation with industry would be essential.

However application of QRA to cross contamination in the domestic and retail environments, which are likely to be significant, is difficult. There are two aspects to this:

- Modelling to simulate the effects of various handling practices, and
- Behavioural information on how people prepare and cook red meat in the domestic/food service kitchen.

Recent efforts by the FAO/WHO have gone into producing the mathematical model, but the data required to run it are not yet available. A recent presentation at the 1st International Conference on Microbial Risk Assessment (Schaffner, 2002) indicated that there is still some way to go before cross-contamination modules can be included in quantitative risk assessments. The author identified three areas that need work to determine;

- What factors are important in controlling transfer rate?
- What routes are important?
- What behaviours are important?

Understanding this would allow modellers to focus on what is important to produce useful, simple cross contamination modules. Some information on these topics will be provided by the current NZFSA project investigating domestic meat handling practices.

Given the high level of reported campylobacteriosis in New Zealand, a QRA would be useful to assess the significance of red meat as a source of infection so that risk based interventions/standards could be justified and then implemented. A limited QRA for *Campylobacter* in red meat from retail to consumption has been commissioned by the NZFSA for ESR during 2005-2006.

8.2 Commentary on Risk Management Options

As noted by the UK Advisory Committee on the Microbiological Safety of Food, general hygiene improvements for microbial quality of carcasses during processing will address *Campylobacter* contamination, along with other bacterial pathogens. Procedures specific for *Campylobacter* do not appear to be necessary.

Even if improvements in *Campylobacter* control during production are achieved, consumer food safety education campaigns such as those conducted by the New Zealand Foodsafe Partnership will continue to be essential (Simmons *et al*., 2001). These should be supported by further investigation into the factors that affect the handling of red meat in domestic

kitchens, particularly cross contamination of ready to eat food and undercooking, especially minced meat products.

8.3 Data Gaps

The data gaps identified in this Risk Profile are:

- carriage rates for *Campylobacter* in New Zealand red meat livestock,
- influence of processing and inspection procedures on observed contamination prevalence at retail, and
- prevalence of contamination in deer meat.

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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