ESR

RISK PROFILE:
CAMPYLOBACTER JEJUNI/COLI
IN
POULTRY (WHOLE AND PIECES)

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

by

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PREAMBLE

Risk Profiles are contracted scientific advice provided to the NZFSA by ESR. They do not have status in terms of NZFSA policy. The documents are peer reviewed by NZFSA and other stakeholders to the extent possible and practicable, and their content is taken into account by NZFSA in formulating their response to particular food safety issues.

Risk profiles represent an on-going body of work that is intended to put hazard/food combinations into perspective with respect to ranking and prioritising of food-borne problems in New Zealand.

This Risk Profile concerns *Campylobacter jejuni/coli* in poultry. As noted in the conclusions, the overall picture of transmission of campylobacteriosis in New Zealand remains to be elucidated and it is, therefore, not possible to estimate the relative importance of all transmission pathways. While there is evidence to suggest that poultry meat is an important "foodborne" source of campylobacteriosis, other routes of transmission through water or animal contact are likely to be significant sources of infection. Similarly, rates of campylobacteriosis are so much higher in New Zealand than Australia despite poultry consumption being similar, and data presented in this document do not identify a specific reason for the apparently higher incidence of campylobacteriosis in New Zealand compared to other developed countries.

It is therefore imperative that all routes of possible infection are characterised so that the most appropriate risk management options can be identified and implemented to reduce the burden of campylobacteriosis in New Zealand.

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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.

Notified campylobacteriosis rates in New Zealand are high compared to other developed countries. The disease is also the most commonly reported infectious intestinal disease in New Zealand at 60% of all notifications in 2005. A general increase has been recorded ever since campylobacteriosis became a notifiable disease in 1980.

New Zealand has a relatively high (by world standards) proportion of reported outbreaks in which *Campylobacter* are identified as the causative agent. In 2005, 47 outbreaks of campylobacteriosis involving 252 cases represented 13.6% of the total number of outbreaks. Chicken and chicken-based foods are identified as the transmission vehicle in many of these outbreaks. In sporadic cases evaluated in case-control studies, factors associated with poultry consumption have been linked most strongly with risk of campylobacteriosis. Undercooking or consumption of chicken away from home were the major risk factors.

Over the last 20 years, consumption of poultry meat has increased two-fold, largely at the expense of sheep meat. In New Zealand, surveys indicate that upwards of 50% of fresh raw chicken available for retail sale is positive for the presence of *Campylobacter*. This prevalence is generally similar to findings overseas. *C. jejuni* has also been found on the exterior packaging of 34% raw whole chickens and 14.5% chicken portions. The high prevalence of contamination in raw retail chicken and to a lesser extent on the exterior of the packaging introduces the risk of cross contamination during purchase, transport and handling in the service industries and domestic settings. This can occur either directly to other ready-to-eat foods or indirectly via food contact surfaces, dish-cloths, hands etc.

It seems possible that part of the increase in notified cases of campylobacteriosis over the period 1980 to 2005 is due to increasing consumption of poultry over the same period. However, this does not explain why the reported campylobacteriosis rate in New Zealand is markedly higher than other countries.

As suggested by a study in Ashburton, there may well be differing patterns of transmission of *Campylobacter* between rural and urban populations in New Zealand. Although the overall picture of transmission of *Campylobacter* is not yet clear, the data indicate that poultry is a significant vehicle for the food-borne component.

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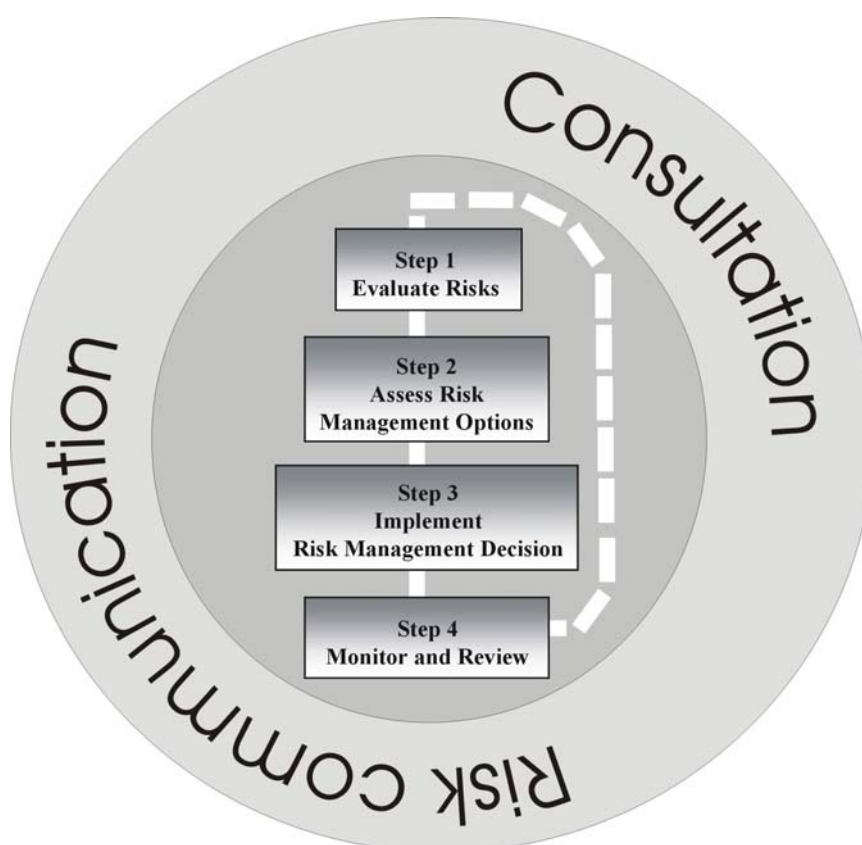
Figure reproduced from "Food Administration in New Zealand. A risk management framework for food safety" (Ministry of Health/Ministry of Agriculture and Forestry, 2000).

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 quantitative risk assessment, or take action, in the form of gathering more data, or immediate risk management activity.

This Risk Profile concerns *Campylobacter jejuni/coli* in poultry. This food/hazard combination was chosen for preparation of a detailed Risk Profile on the basis that the rate of notified cases of campylobacteriosis in New Zealand appears to be high by international standards, epidemiological links between campylobacteriosis and poultry consumption, and the need to establish proportionality among the various potential transmission routes that have been identified. This Risk Profile has been updated from the previous version, produced in 2003.

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 following information is taken from a data sheet (http://www.nzfsa.govt.nz/science-technology/data-sheets/campylobacter.pdf) 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.

2.1 *Campylobacter*

2.1.1 The organism/toxin

Campylobacter spp. are slender, spirally curved rods which are non-sporulating and Gram negative. There are many species 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 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.

The terms thermophilic *Campylobacter* or thermotolerant *Campylobacter* are often encountered in the literature and includes *C. jejuni*, *C. coli*, *C. lari* and *C. upsaliensis*.

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. 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.

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. 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 anaerobic electron transport pathways (Kelly, 2001). The organism can be adapted to aerobic growth (Jones *et al.,* 1993).

Water activity: Optimum growth is at $a_w = 0.997$ ($\equiv 0.5\%$ NaCl), minimum $a_w \geq 0.987$ ($\equiv 2.0\%$ NaCl).

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^oC than at 20^oC. Freezing causes an initial one $\log_{10}$ decrease in numbers of *C. jejuni* followed by a gradual reduction during subsequent storage 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 carbon dioxide-enhanced 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. VNCs may 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^oC-60^oC for several minutes (ICMSF, 1996). D time at 50^oC = 1-6.3 minutes. D time at 55^oC = 0.6-2.3 minutes. D time at 60^oC = 0.2-0.3 minutes. Therefore heat treatments that destroy salmonellae should also destroy *Campylobacter*.

Numbers declined rapidly on sterile meat slices of high and normal pH when incubated at 25^oC (Gill and Harris 1982).

Freezing rates influence survival more than actual frozen storage. Slow freezing rates are more lethal than rapid freezing because of osmotic stress. Significant reductions in *Campylobacter* numbers were observed when inoculated chicken portions were frozen to – 10°C and this effect was attributed to the long freezing time necessary to reach this temperature (19h 40min) (Whyte *et al*., 2005). However legal and practical reasons would currently prevent this time/temperature parameter from being used in industry (see Section 7.1.2). The exception to this are the very high freezing rates (in excess of 10°C/min), which result in mechanical cell damage due to intracellular ice crystals.

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. 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. Poultry primary processing in New Zealand uses immersion chilling, and plant conditions do not permit the same air drying effect afforded to red meat carcasses.

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 application 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 suggested to destroy salmonellae on poultry. 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.

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

Animal: *Campylobacter* can be found in the intestinal tract of a wide variety of wild and domesticated warm-blooded animals which may or may not be symptomatic. The prevalence of the organism within cattle herds and sheep flocks can vary but rarely exceeds 50% (AIFST, 2003). A higher prevalence has been observed in younger animals and in animals from higher stocking densities. *C. coli* is usually the dominant species in pigs. Household pets have been implicated as risk factors of campylobacteriosis in control studies. Flies (Hald *et al*., 2004) 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). Estimates of flock prevalence in New Zealand are approximately 17% (Boxall, 2005) and 89% (Teck Lok Wong, Food Group ESR, personal communication, November 2006). Once a poultry flock is infected, the organism spreads rapidly until within a week most or all the birds are infected.

Food: Since *Campylobacter* are frequently found in livestock intestines, it is not unexpected on meat and poultry at the abattoir and in the retail market.

Raw poultry is frequently contaminated. A prevalence of 89% in retail minced/diced chicken samples in New Zealand has been demonstrated (Wong *et al.,* 2006), while retail cooked chicken is rarely contaminated (0.07% based on a 1995 New Zealand survey).   The prevalence in retail red meat in New Zealand is up to 10% (Wong *et al.*, 2006).   Cross contamination from raw to cooked chicken can occur (see section 5.1.2). *Campylobacter* has also been isolated from watercress, offals from various animals, and raw milk in New Zealand. Pathogenic species were not detected in New Zealand commercially harvested shellfish in one study.

In addition to the foods above, mushrooms, garlic butter, and salads have all yielded *Campylobacter* in overseas studies.

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°C. *Campylobacter* are 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 relative importance of the various potential transmission routes, e.g. foods, recreational water, occupational exposure, is unknown. Determination of the most important pathways is a 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*" describes 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 modification 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 the 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 some evidence for plasticity and instability in the *Campylobacter* genome that has been a problem for the development of a universal typing system (Tam, 2001). However, there are also contradictory data reporting the genetic stability of one strain in a variety of environments over 20 years (Manning *et al*., 2001).

Recent technology has enabled restriction enzyme digestion and pulsed field gel electrophoresis (PFGE) to be used in 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 Centers 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 by classical Penner serotyping and PFGE. Phase two is available to researchers upon request via the internet link;
http://campynet.vetinst.dk/news.htm

In New Zealand, typing has been adopted as a primary tool for epidemiological investigation of pathogen sources from both outbreak and sporadic cases of campylobacteriosis. Efforts have been made by the New Zealand 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 Aotearoa (New Zealand) with an initial focus on *Campylobacter, Salmonella, Listeria* and *Escherichia coli* O157 (Gilpin, 2004). 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 have joined the network. The electronic database helps ensure consistent methods of sub-typing are used, so that the results are comparable both nationally and internationally. The national link up enables New Zealand's laboratories to carry out collaborative studies. This could be especially important for responding to a major food or waterborne disease outbreak – both 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

3.1 Relevant Characteristics of the Food: Poultry, Whole and Pieces

The term 'poultry' principally concerns chickens, but can also include other domestic fowls such as ducks, geese, and turkeys. Game birds, such as pheasant and quail, represent a small proportion of the market in New Zealand.

The water activity (a_w) of poultry meat is about 0.98 to 0.99. The pH of chicken breast muscle is 5.7 to 5.9, while that of leg muscle is 6.4 to 6.7. Both poultry muscle and skin are excellent substrates for supporting the growth of a wide variety of microorganisms (ICMSF, 1998).

Since the minimum growth temperature is unlikely to be exceeded in stored poultry products, growth should not occur. Of more importance is the enhanced ability of *Campylobacter* to survive on refrigerated stored foods.

3.2 The Food Supply in New Zealand

The poultry industry in New Zealand is divided into two main sectors:

- Poultry meat production including livestock breeding; and,
- Table egg production.

These are linked via commercial hatcheries and large feedmills. Differences occur in breeds of hen, i.e. broiler hens are either Ross or Cobb and commercial layers are either Hyline or Shaver. The hatcheries and feedmills are generally owned by the vertically integrated poultry processing and breeding companies (PIANZ, 1999). However, this is not always the case in both the layer and broiler industry.

To the year ending June 2006, New Zealand consumer expenditure on poultry meat totalled $396 million (Vanessa Wintle, PIANZ, personal communication, November 2006). The majority is domestically consumed, with a small, but increasing, trade of live day-old chicks, fertile hatching eggs, poultry meat, edible offal and table eggs exported to the Pacific Islands and a limited number of other countries.

Currently, approximately 40% of poultry is sold as whole carcasses (Vanessa Wintle, PIANZ, personal communication, November 2006).

Competitive poultry retail prices and the popularity of low fat meat has driven the current demand for poultry meat. Production for the year ending June 2006 is 149,000 tonnes dressed weight, down 7,000 tonnes on the previous year. In the year ending June 2006, approximately 98% of poultry consumption was chicken meat, with turkey, duck, and roasting fowl making up the remaining 2% (Vanessa Wintle, PIANZ, personal communication, November 2006). Since 1995, the poultry industry has grown on average by 5.3% per year.

From 1990 to 2006, total meat consumption has been relatively static, but the proportion of poultry meat consumed has increased from 15% to 36%, largely at the expense of sheep meat. Most production (65%) is purchased and consumed by domestic households, the remaining 35% enters the food service industry (including fast food outlets), see website; http://www.pianz.org.nz/IndustStatFacts.htm#PoultryProduction (accessed 29/11/04)

Recent increases in chicken consumption have been mainly in the fresh and further processed areas, with approximately 79% of chicken now sold as fresh product (i.e. chilled) and 21% frozen. This represents a considerable change over the last 10 years. In 1995, 60% of product was sold frozen (Vanessa Wintle, PIANZ, personal communication, November 2006).

Declining prices in real terms, lifestyle changes and consumer perceptions have seen poultry consumption continue to increase, up from 14 kg per capita ten years ago, to 37.1 kg per capita in 2006 (Vanessa Wintle, PIANZ, personal communication, November 2006). This compares to per capita figures overseas of 50.1 kg in the USA, 34.2 kg in Australia, 22.7 kg in Western Europe and 37.1 kg in Canada (MAF, 2003). New Zealand poultry consumption for the year ended June 2006 has decreased by 5.5 %. This follows a trend in production which has decreased in the last quarter of 2005.

The Poultry Industry Association of New Zealand Incorporated (PIANZ) represents the interests of the majority of the poultry processing and breeding companies in this country. Three companies (Tegel Foods Ltd, Inghams Enterprises (NZ) Pty Ltd, and PH van den Brink Ltd.) dominate the industry, accounting for 96% of poultry production.

3.2.1   Imported food

Heat treated (or retorted) poultry meat can be imported under New Zealand's biosecurity rules. Chilled and frozen poultry meat may also be imported, provided that New Zealand's biosecurity requirements are met. The cost of meeting these requirements and the small market size, however, make New Zealand commercially unattractive to overseas poultry exporters (MAF, 2003). Imported poultry therefore is not a consideration for this Risk Profile.

3.2.2   Processing

The New Zealand poultry industry raised 88.766 million broiler chickens in 2005, the majority (99%) barn raised. Around 1,700,000 birds are processed each week, although production in the September-December quarter is generally higher than other quarters. Farms are usually situated within 50 km of processing plants.

The sequence of processing generally involves:
- Electrical stunning;
- Killing by cutting of the carotid artery;
- Bleeding;
- Scalding, defeathering and washing;
- Removal of viscera and other parts;
- Spray washing and chilling; and,
- Grading and packaging (whole or in parts).

The final chilling step can be achieved in a variety of ways:

- Immersion in tanks of cold water, with or without the addition of ice;
- Sprays of cold water;
- Circulation of cold air.

Immersion in chilled water with added chlorine is the method used in New Zealand. The advantage of continuous immersion chilling is that it is efficient and inexpensive. Chlorine is added to the water to control pathogens, although it is rapidly inactivated by organic material. The efficacy of control of pathogens on carcasses by chlorine has historically been considered limited. At 25 ppm chlorine, although total viable bacteria counts on carcasses have been shown to decline, the levels of *E. coli* and *Enterobacteriaceae* did not (Whyte *et al.*, 2001). The primary purpose of the chlorine is to control pathogens in the wash water and reduce the potential for cross contamination. The efficacy of pH control in the spin chiller is currently being assessed by the industry.

Whole or individual parts of birds may be packaged raw for direct sale. Some of the major poultry producers in New Zealand have introduced the use of leak proof packaging. This is intended to prevent chicken juice leakage and potential cross contamination from the exterior of the package. This may contribute to the overall reduction of *Campylobacter* infection in the community.

Where the birds are portioned, they are generally cut into a number of pieces, which are placed on "PLIX" porous food trays (open cell, expanded polystyrene) and covered with a plastic film. "PLIX" is the brand name made by Trays Business Unit, Vertex – Pacific Ltd. The new trays were introduced to the industry in July 2001 and were adopted across the industry over a 12 month period (Ron Starnes, Vertex Pacific Ltd., Auckland, pers. comm., April 2004). Before the absorbent trays were introduced, a "diaper" (absorbent paper with a plastic backing) was often used to catch any liquid released from the meat. However, diapers are becoming less common with the introduction of the absorbent "PLIX" trays, although they may still be in circulation in areas where the older stocks of "diaper" packaging are being used up. Both approaches were intended to prevent excessive drip of meat juice, and not primarily for food safety. The prevention of drip helps to control the spread of pathogens that may be present to the outside of the package and prevents further cross contamination to other foods. This would contribute to the reduction of cross contamination of *Campylobacter*.

Most frozen poultry is packaged in plastic bags clipped at the end and then frozen in high-velocity freezers. Before freezing, poultry may be injected with various salts, flavourings and oils in order to increase the juiciness of the meat.

4 HAZARD CHARACTERISATION: ADVERSE HEALTH EFFECTS

The pathogenic mechanisms of *Campylobacter* infection are poorly understood (AIFST, 2003) and the disease appears to have different manifestations. In developed countries 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). In less developed countries the mechanism for production of watery diarrhoea is proposed to involve attachment of *C. jejuni* to the intestinal cells, and the production of a cytotonic enterotoxin. However, studies indicate that the currently understood 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 maximum 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, with the 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. It should be noted that New Zealand, despite its high rate of campylobacteriosis, has a low rate for Guillain-Barré syndrome.

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).

Treatment: Usually none, but fluids may be given, especially as young and elderly patients may become dehydrated. Some cases warrant treatment with antibiotics. Erythromycin is the drug of choice, although resistant strains are emerging.

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. 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 O19 and O41 have been more frequently associated with GBS than other serotypes (AIFST, 2003). Penner Serotype O19 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).

In a study of 8000 cases of campylobacteriosis associated with three Swedish waterborne outbreaks, none of the cases developed GBS in the six months following infection (McCarthy *et al*., 1999). Given that the cases were associated with only three outbreaks, and so possibly only three subtypes of *C. jejuni*, then GBS caused by subtypes not involved in these outbreaks would have been unrecognised. See section 6.1.5 for serotypes causing human disease in New Zealand.

In New Zealand, the paradox of high campylobacteriosis rates and low GBS may be explained by the low prevalence in New Zealand of the Penner serotypes that have been linked to the syndrome. Serotypes obtained from human cases in New Zealand between 1996 and 2001 found only 0.8% were Penner serotype O19 and 0.5% were serotype O41. Serotype information from poultry also suggests a low prevalence. From a recent national retail survey including minced and diced chicken (Wong *et al*., 2006), out of 200 serotyped isolates, one was serotyped O19 and none serotyped O41. There were 36 serotypes that were untypable in this survey.

4.3   Dose Response

There is a growing consensus that a minimum infectious dose for human pathogens does not exist, and 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, Teunis and Havelaar, 2000), with an overview reported by an expert group assembled by FAO and WHO (FAO/WHO, 2002). Infection, where the microorganism is reproducing in the body, was modelled separately from illness,

which is less frequent. The likelihood of infection increased from approximately 50% at 800 cells to approximately 100% at 1×10^8 cells. In contrast, the likelihood of illness was approximately 20% at 800 cells, rising to approximately 55% at 9×10^4 cells, and declining to 0% at 1×10^8 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 an optimum number of cells are consumed for sickness to occur.

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 them subsequently becoming sick.

To give an idea of the probability of human disease given a variety of doses, Figure 2 illustrates the 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%
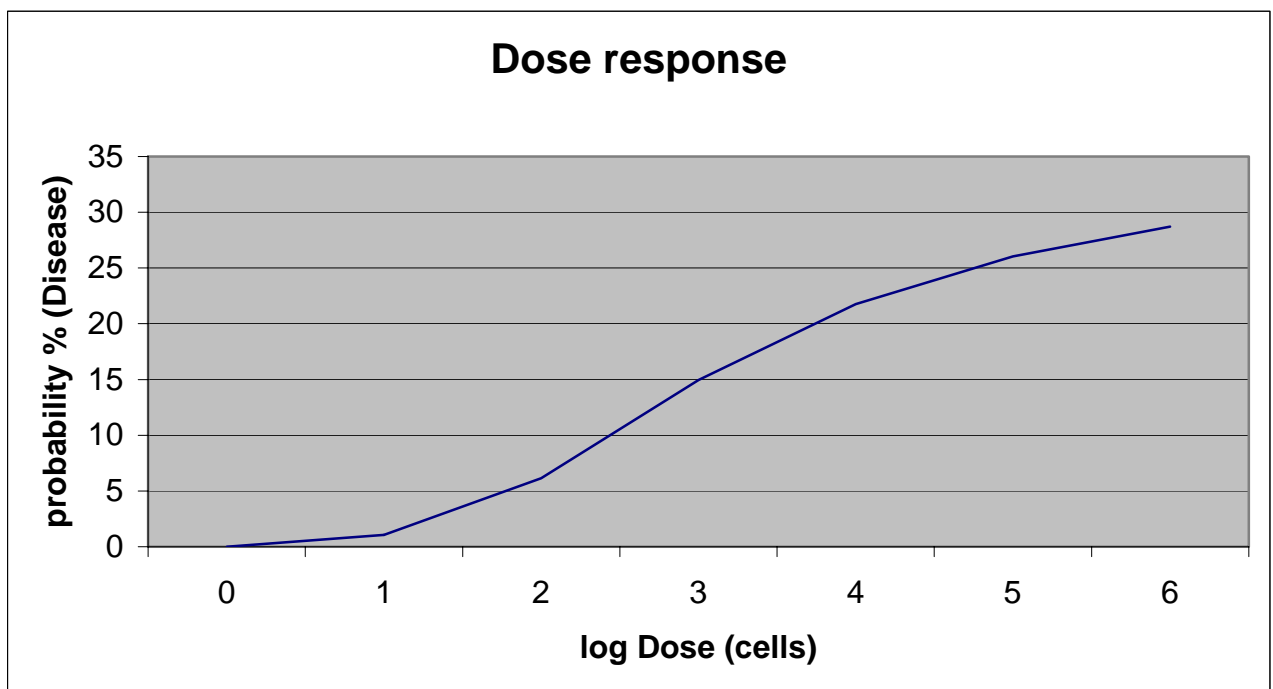
5 EXPOSURE ASSESSMENT

5.1 The Hazard in the New Zealand Food Supply: *Campylobacter* in Poultry

5.1.1 *Campylobacter jejuni* in broiler chickens

Information on the prevalence of *C. jejuni* in New Zealand flocks is limited. One study has indicated a prevalence of contamination on arrival at processing of approximately 17% (Boxall, 2005).

A significant data gap for constructing a model of broiler processing in New Zealand was identified as the prevalence and numbers of *Campylobacter* on freshly slaughtered chickens following bleedout (exsanguination) but prior to scalding. A 2005-2006 study has been completed by ESR involving 200 exsanguinated birds from 39 flocks supplied by 30 farms over 41 consecutive weeks. The birds were derived from three major poultry companies at four processing plants. Whole bird rinsates and caecal swabs were tested for *Campylobacter*. *Campylobacter* spp. isolated were identified as *Campylobacter jejuni and C. coli* by PCR.

Caecal swab cultures from 35 flocks were positive for *Campylobacter*, giving a flock prevalence of 89.7%. All whole bird rinsates were positive for *Campylobacter*, with counts ranging (as shown in Figure 1) from 2.18 to 9.46 $\log_{10}$ cfu bird^{-1} (TeckLok Wong, personal communication, ESR, November, 2006).

Figure 3:   Distribution of *Campylobacter* counts on external surfaces of 200 exsanguinated birds
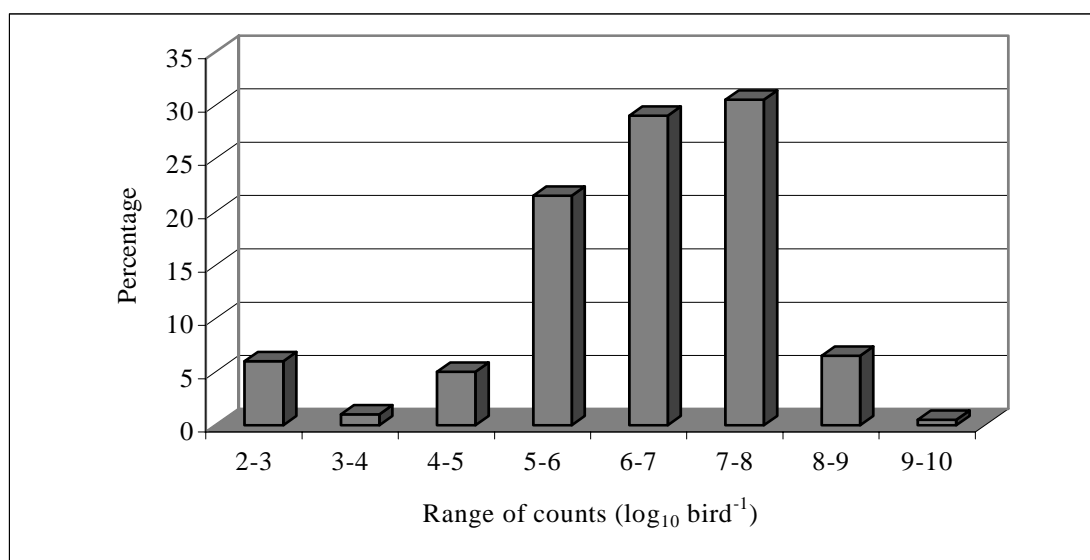
Source: Teck Lok Wong, ESR, personal communication, November 2006

5.1.2 *Campylobacter* in raw and ready-to-eat chicken products

A summary of the surveys of New Zealand raw poultry for *Campylobacter* up to 2003 are given in Table 1.

Table 1:   **Reported prevalence of *Campylobacter* in raw poultry (whole and pieces) in New Zealand**

Products	Samples tested	Positive for *Campylobacter* (%)	Year	Reference
Fresh chilled chicken	22	68.2	1984	Gill and Harris, 1984
Frozen chicken	37	16.2	1984	Gill and Harris, 1984
Whole fresh* chickens	137	56.9	1992/1994	Campbell and Gilbert, 1995
Whole frozen chickens	17	0.0	1992/1994	Campbell and Gilbert, 1995
Fresh chicken portions	113	56.6	1996/1997	Hudson *et al*., 1999
Whole fresh* chickens	50	54.0	1999	Consumers' Institute, 1999
Whole fresh chickens*	40	85	2003	Consumers' Institute, 2003

*Fresh is taken to mean that the samples were chilled and not frozen

A national retail survey of minced and diced meat from supermarkets and butchers during 2003/2004 has been undertaken by ESR for the NZFSA to assess the prevalence of food-borne pathogens in meat (Wong *et al*., 2006). Results for red meat are included here for comparison. The results are shown in Table 2.

Table 2:   **National retail survey of *Campylobacter* in chicken and red meat; July 2003 to June 2004**

Meat (all minced/diced)	No. samples tested	Total Number positive (%)	*C. jejuni*	*C. jejuni & C. coli*	*C. coli*	Counts in positive samples (MPN/g)	
Chicken	**230**	**205 (89.1)**	**199**	**5**	**1**	**<0.3 to 110**	
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

Of the 204 chicken samples positive for *C. jejuni*, (Note: One sample was *C. coli* only) the counts (MPN/g) were as follows;

82 samples	<0.3 MPN/g	104 samples	0.3 – 10.9 MPN/g
17 samples	>10.9 – 45.9 MPN/g	1 sample	110 MPN/g

A total of 247 isolates from all sample types were serotyped and Pulse Field Gel Electrophoresis carried out. Of those typable, 17 serotypes were identified. Isolates of all 17 serotypes were present in chicken samples. The results are presented in Table 3.

Table 3: **Serotypes from chicken and other raw meats samples in New Zealand**

Serotypes	Chicken	Pork	Lamb	Beef	Bobby veal
Untypable	36	**-**	2	1	
1,44	35	3	2	-	-
2	23	4	1	2	-
4,13,16,50	14	-	5	1	
6	14	1	-	1	1
8,17	1	-	-	-	1
11	20	4	-	-	-
12	4	1	1	-	1
19	1	-	1	-	-
21	6	-	-	-	-
22	1	-	-	-	-
25	3	-	-	-	-
23,36	3	-	-	-	-
27	13	-	-	-	-
37	1	-	-	-	-
42	13	2	2	-	5
55	7	1	1	-	-
58	5	-	1	-	-

Out of the 230 samples of chicken mince, 37 were supplied pre-packed by primary processors to supermarkets. 36 of these 37 packs were positive for *Campylobacter*. The implication is that it is unlikely that primary contamination with *Campylobacter* occurred at the supermarket, although does not rule out that cross-contamination does not occur.

In early 2002, ESR conducted a survey of three hundred retail packs of fresh chilled poultry products from fifteen supermarkets in the Christchurch area (Wong *et al.,* 2004). The purpose was to determine the prevalence of *Campylobacter* on the exterior of packs, and was prompted by findings in Wales and London (see Section 5.4.3). The results were:

- 72 (24%) packs were externally contaminated with *C. jejuni*.
- Offal samples had the highest rate of external contamination (52%) followed by whole chickens (34%) and chicken portions (14.5%).
- Of the 250 packs of whole or portioned chicken meat sampled, 21 were positive but with low *C. jejuni* counts of <6 MPN/pack, 22 packs recorded counts in the range of 6-190 MPN/pack, and 3 samples recorded 480-2200 MPN/pack.

These observations suggest that packs may be a significant source of cross-contamination, although the contribution of this contamination pathway to food-borne illness can only be properly determined by development of a validated risk assessment model.

Some poor retail practices for stacking traypacks of meat combined with leaky packaging points to a potential for cross-contamination. There are moves being made by the majority of poultry processors and a significant sector of the retail industry to address the problem of the leaky packaging.

The use of tongs during the pan fry cooking of chicken is another recognised vehicle for cross contamination. In a study by Hudson *et al*. (2003) thirty chicken samples were inoculated at 500 cells cm^{-2} with *Campylobacter* and *Salmonella*, then cooked with one turn (using sterile tongs). After cooking, the chicken was transferred using the same tongs to a sterile surface. The 30 tongs were swabbed, 23.2% were positive for *Campylobacter jejuni* by presence/absence testing, 40% were positive for *Salmonella,* and in total 43.3% yielded a pathogen. Out of thirty cooked chicken samples, 36.7% were positive (presence/absence testing) for *Campylobacter* (the same percentage were positive for *Salmonella,* and in total 46.7% of samples yielded a pathogen). The authors concluded that as the use of the same tongs for both raw and cooked meats is a common practice, the number of cross contamination events is likely to be significant.

5.1.3   Ready-to-eat poultry products

Campylobacter are readily killed at normal cooking temperatures. Residual contamination of cooked products could result from undercooking, but is more likely due to cross-contamination from raw products, contaminated contact surfaces (refer the tongs above), or direct contamination by an infected food handler.

Despite the fact that raw poultry has frequently been found to be contaminated with *Campylobacter*, only one sample out of 1320 (approximately, the exact number of samples is not specified) or 0.07% of ready-to-eat chicken products tested positive for the organism in a 1992-1994 New Zealand survey (Campbell and Gilbert, 1995). This implies that those service industries processing the raw poultry in this survey are effectively destroying the organism and cross contamination can be controlled. Although this puts the emphasis for controlling the risk upon the service and domestic sector.

In the most recent survey, by the Consumers' Institute (2003), 25 cooked rotisserie chickens and 25 smoked cooked chickens were tested. None were positive for *Campylobacter.*

5.1.3.1 Subtypes from poultry and human sources in New Zealand

There are three published papers that report on the overlap between poultry and human *Campylobacter* types in New Zealand. Restriction endonuclease analysis of *Campylobacter* isolates found that 49.7% of the human isolates had banding patterns that were indistinguishable from poultry isolates (Kakoyiannis *et al*., 1988). More recently a similar analysis using Penner serotyping and Pulsed Field Gel Electrophoresis (Hudson *et al*., 1999) found that a number of discrete groups, indistinguishable by both typing methods, contained isolates from both humans and raw chicken pieces. The most numerically dominant group was absent in isolates obtained in mid winter, but was the most numerous in the summer, when notified cases of human campylobacteriosis are at their highest. Isolates in this group were only obtained from human faeces and raw chicken pieces.

Some caution must be exercised in interpreting these data, as the isolation of indistinguishable types from humans and chicken does not show a direction of transmission from chicken to human (or the other way round). It does show that those types are capable of causing disease, and so human pathogenic types of *C. jejuni* were isolated from raw poultry in both studies. It seems likely that a proportion, but possibly not all, types isolated from poultry are capable of causing disease in man.

Data in press (Baker *et al*., 2002) failed to show any significant overlap in types isolated from human cases and raw whole chickens in the Ashburton area. 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 predominantly urban areas.

5.1.4   Conclusions

The data that are available show that the organism is quite common in fresh raw poultry at the retail level. The prevalence in frozen poultry is considerably lower, which is to be expected as freezing is known to reduce the numbers of *Campylobacter*, possibly by damaging the outer membrane (Humphrey, 1988). The two studies of frozen chicken samples reported 0% (Campbell and Gilbert, 1995) and 16.2% prevalence (Gill and Harris, 1984). The ESR Public Health Laboratory in Christchurch has isolated *Campylobacter* from frozen chicken with more recently developed methods.

The low prevalence in cooked chicken may also be due to methodological limitations, but given the organism is heat labile, the result is more likely to be a true reflection of cooked chicken products.

5.2   Food Consumption: Poultry

Consumption of poultry meat has increased steadily over the last 19 years, from an apparent consumption (poultry available for consumption per capita) of 15 kg/person/year in 1985 to 37.1 kg/person/year for the year ending June 2006 (Vanessa Wintle, PIANZ, personal communication, November 2006).

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

Summary food consumption statistics can be expressed in terms of 'consumer' (just those people reporting to eat a particular food) or 'persons' (the whole population). Both will be presented here. Information expressed on a 'consumer' basis provides information on likely serving sizes, while information expressed on a 'person' basis provides information on the total amount of poultry being consumed by the population.

The age groups used by the 1995 Australian National Nutrition Survey (Australian Bureau of Statistics, 1999) will initially be used so that an easy point of comparison can be made. These are 16-18 years, 19-24 years, 25-44 years, 45-64 years, 65 years and over. Comparisons with the Australian National Nutrition Survey are complicated as poultry is included in two categories with descriptions 'Poultry and other feathered game' and 'Mixed dishes where poultry or game is the major ingredient'.

Table 4 shows the percentage of respondents in various age-sex groups who reported consuming poultry in the previous 24 hour period. This is assumed to be equivalent to the proportion of the population consuming poultry on any given day.

Table 4:   Total poultry – percentage of respondents consuming

Age (years)	16-18	19-24	25-44	45-64	65+	Total
Male	22.0	28.3	22.7	20.1	13.2	20.7
Female	16.8	21.1	22.8	20.7	17.3	20.9
Total	19.1	24.0	22.8	20.4	15.7	20.8

Food Standards Australia New Zealand (FSANZ) carried out an analysis of the 1997 NNS dataset, including application of a set of standard recipes, to allow composite foods to be reduced to their component parts (ANZFA, 2001). This analysis, as expected, produced a higher estimate of the proportion of the population consuming poultry meat on a daily basis (27.5%). The difference between the FSANZ estimate and the figures in Table 4 will be due to the presence of poultry meat as a component of products such as meat pies and luncheon meat. For comparison, the number of respondents who reported eating pork/beef/lamb, and products or recipes containing these, in the previous 24 hours, according to the FSANZ analysis was 77.6%.

The 24 hour dietary recall records do not routinely identify the source of the food. However 117 of the 1040 (11.3%) respondents who reported eating poultry, identified the source as "KFC".

In the Australian NNS the percentage of respondents eating poultry and other feathered game were; males 17.0%, females 16.5%, all 16.8%. Percentage of respondents eating mixed dishes with poultry or game birds as the major ingredient were; male 10.9%, female 9.8%, all 10.3% (Australian Bureau of Statistics, 1999).

These figures are comparable to those obtained in the Life in New Zealand (LINZ) survey (the previous National Nutrition Survey) which reported 17% of respondents eating poultry and a further 5% eating poultry and vegetables.

Table 5 summarises the median daily consumption of poultry by consumers only. If poultry were only eaten once during the day these figures would represent serving sizes, however, in some cases individuals may consume several servings of poultry during the day.

Table 5: Total poultry – median consumption by consumers (g/day)

Age (years)	16-18	19-24	25-44	45-64	65+	Total
Male	183	193	143	149	135	147
Female	108	119	114	100	84	107
Total	143	140	127	126	99	126

The Australian NNS study reported an overall median (males and females) for respondents aged 19 and over of 72 g/day (poultry and other feathered game) and 162 g/day (mixed dishes with poultry or game as the major ingredient) (Australian Bureau of Statistics, 1999), compared to an overall median of 126 g/day from the New Zealand NNS (Russell *et al.*, 1999).

The LINZ survey reported the mean (not median) intake of poultry as 104 g/day (118 g/day for males and 90 g/day for females). For poultry and vegetables, the mean intake was 179 g/day for consumers (195 g/day for males, 161 g/day for females).

Table 6 summarises the 95th percentile levels of daily consumption of poultry for consumers only. These levels of consumption represent high level consumers of this food product and are often used when considering 'worst-case scenarios' for dietary exposure.

Table 6: Total poultry – 95th percentile consumption by consumers (g/day)

Age (years)	16-18	19-24	25-44	45-64	65+	Total
Male	515	384	503	368	267	466
Female	244	344	296	238	176	276
Total	457	383	357	314	243	347

Table 7 gives the mean level of daily consumption of poultry for the whole population (consumers and non-consumers).

Table 7: Total poultry – mean consumption by persons (g/day)

Age (years)	16-18	19-24	25-44	45-64	65+	Total
Male	50.0	62.7	42.4	34.4	18.0	37.8
Female	18.5	30.8	30.4	22.7	16.5	25.4
Total	32.5	43.9	35.0	28.2	17.1	30.5

The Australian NNS reports an overall mean consumption of 21.9 g/day of poultry and other feathered game and 21.5 g/day of mixed dishes with poultry or game as the major ingredient (Australian Bureau of Statistics, 1999). It is difficult to draw any direct comparisons between the New Zealand and Australian figures. The FSANZ analysis of the 1997 NNS

data, including the use of standard recipes estimated a mean daily intake of poultry meat for all persons of 34.4 g/day (ANZFA, 2001).

The total consumption of poultry in New Zealand is made up almost exclusively of chicken (98%), with very small contributions from turkey and duck. The distribution formed by considering the probability of different levels of daily consumption of poultry approximates to a log normal distribution. Table 8 gives the mean estimates of meat consumption along with the amount available for consumption.

Table 8: **Mean estimates of meat consumption (total population over 15 years), 1997 and estimates of meat available for consumption, year ending Sept. 1999**

Meat type	Estimated consumption (g/person/day) (1997)*	Amount available for consumption (g/person/day) (year ended 30.09.99)#
Beef and veal	87.9	86
Sheep and Lamb	13.7	39
Pigmeat	32.3	46
Total red meat	**134.9**	**171**
Poultry	34.4	73
Total meat	**169.3**	**244**

* FSANZ analysis of 1997 National Nutrition Survey data (ANZFA, 2001)
\# Annual Review of the Sheep and Beef Industry, 1999-2000, http://www.mwi.co.nz/economicservice/publications/samples/AnnualReview.pdf
(Meat and Wool Board Economic Service) recalculated from kg/person/year to g/person/day

The difference between these two estimates of consumption may reflect farming production differences between the two years compared (1997 and year ending September 1999). There will also be wastage (meat available for consumption, but not consumed), and under-reporting in the NNS. Through use of standard recipes, the FSANZ analysis of the 1997 NNS data will include all meat consumed, including meat, which is consumed as a component of a processed food such as meat pies or luncheon meat (ANZFA, 2001). Note that for beef and veal, consumption and availability data are almost the same.

5.3 Qualitative Estimate of Exposure

5.3.1 Number of servings and serving sizes

The estimation of total number of servings of poultry (whole and pieces) 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 poultry 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 however because Australian data suggests that those under 10 years of age may consume only half as much poultry as those over 10 years of age (Australian Bureau of Statistics, 1999). Information for New Zealanders less than 15 years is currently unavailable. US data suggests the assumption may be adequate for those over five years of age (EPA, 1997).

From the NNS, 1344 individual dietary records were deemed to represent consumption of a serving of poultry. 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)   = 1344 x 4,054,200/4636 x 365
                                                 = 4.28 x 10^8 servings

This represents a high number of servings, as would be expected from a commonly consumed food such as poultry.

Based on the data in the NNS database the 50, 75, 95, and 99th percentile serving sizes for poultry in New Zealand were:

Percentile	Serving size (g)
50	84
75	143
95	268
99	506

In other words, half of poultry meals consumed by New Zealanders will result in consumption of 84 g or less of poultry meat, while only 1% of poultry meals will result in consumption of more than 506 g of poultry meat.

A FAO/WHO risk assessment for *Salmonella* in broiler meat reported serving sizes taken from an Irish food consumption survey which gave a mean serving size of 95.5 g, with a standard deviation of 54.4 g/serving.

(http://www.who.int/fsf/mbriskassess/Scientific_documents/mra05.pdf).

5.3.2   Frequency of contamination

The available data indicate that *Campylobacter* are an infrequent contaminant of cooked chicken (0.07% in a 1992-1994 survey and 0% in a 2003 survey). However, raw chickens are frequently contaminated with this organism and so may result in increased exposure to *Campylobacter* if they are not properly handled by cross contamination of a ready-to-eat food.

5.3.3   Predicted contamination level at retail

Data from the most recent ESR survey indicate that counts on poultry were generally low; chicken meat (minced or diced) samples contained less than 110 MPN/g.

5.3.4 Growth rate during storage and most likely storage time

The shelf life of refrigerated raw poultry is quite short in comparison with other meats. A website linked to the American Meat Institute (http://www.meatsafety.org/safehandling/safehandling.htm) provides the following recommended periods for safe storage of poultry:

Chicken and Turkey: Refrigerated: (whole, parts, ground or giblets) 1 to 2 days. Frozen: to assure quality, whole 12 months; parts 9 months; ground 3 to 4 months; giblets 3 to 4 months.

Shelf lives of 7, 5 and 4 days at 4, 7 and 9°C respectively were determined using an end point of approximately 7.2 $\log_{10}$ cfu spoilage bacteria/ml of rinse (Abu-Ruwaida *et al.,* 1994). This end point was accompanied by changes in organoleptic characteristics, which would make the chicken unacceptable to consumers.

Given the biology of the organism, growth is unlikely to occur during refrigerated storage, although conversely survival of *Campylobacter* will be best under refrigeration.

5.3.5 Heat treatment

Normal cooking temperatures should be adequate to destroy *Campylobacter.*

5.3.6 Exposure summary

The information presented indicates that poultry (in particular, chicken) is a commonly consumed food in New Zealand. Survey data indicate that the contamination rate of raw chickens by *Campylobacter* is high at 89%, although the counts are low with most positive samples below 10.9 MPN/g. However, the prevalence of *Campylobacter* in cooked chicken from various categories of caterers and retailers in New Zealand is very low, a survey in 1992-1994 found only 0.07% positive (one positive sample in approximately 1320 samples tested). While a recent survey found no positive samples amongst 50 cooked retail chickens tested. These results are expected given the thermal inactivation characteristics of the organism. Although these data relate to cooked poultry in the retail sector and may not reflect poultry cooked at home, it seems reasonable to expect that despite high consumption, exposure to *Campylobacter* directly from cooked poultry is low.

In a limited telephone survey of meat cooking practices approximately four percent of people who reported eating chicken indicated a preference for the chicken to be cooked to a rare condition (Thomson and Lake, 1995). A similar percentage reported that chicken would be cooked by barbecuing. This may lead to survival of the bacteria and higher levels of exposure. It is acknowledged however that it is an unusual question, to be asked the cooking preference for poultry unlike for example, steak or lamb. The use of same tongs when handling raw and cooked poultry on the barbecue may also lead to cross contamination events (see Section 5.1.2).

The high frequency of contamination of raw poultry by *Campylobacter* provides an entry point for the pathogen to food preparation areas. Cross contamination from raw to cooked, or to ready-to-eat products may then occur. The ability of *Campylobacter* and *Salmonella* to

spread from raw chicken to other sites in kitchens during food preparation has been clearly demonstrated in modelling studies (de Boer and Hahné, 1990) as well as in real-life environments (Cogan *et al.*, 1999; Gorman *et al.*, 2002). Subsequent re-infection of the cooked chicken or other foods can also occur (Humphrey *et al.*, 2001).

The impact of cross-contamination on risk is difficult to quantify, although a risk assessment being conducted by the FAO/WHO is developing models to address this issue (FAO/WHO, 2002).

5.4   Overseas Context

5.4.1   *Campylobacter jejuni* infection in broiler chickens pre-slaughter

The incidence of *C. jejuni* in broilers has been reported to range from 83% to 88% (Grant *et al.*, 1980; USDA FSIS, 1996) in the United States, and 14% to 91% (Simmons and Gibbs, 1977; Ribiero, 1978) in the United Kingdom. More recent data from the United Kingdom indicate that approximately 60% of housed broiler poultry flocks are *Campylobacter* positive at slaughter age (ACMSF, 2004). An Australian study showed that three of four flocks examined carried *C. jejuni* with an isolation rate of 52 to 100% in the positive flocks (Shanker *et al.*, 1982). The variations may be due to differences in sample size, isolation methodology, or variation in flocks from different localities, or all of these factors.

An examination of 160 broiler flocks in Denmark found that at the time of slaughter *Campylobacter* were able to be isolated from 100% of organic flocks, 36.7% of conventional flocks, and 49.2% of extensive indoor flocks (Heuer *et al.*, 2001).

5.4.2   *Campylobacter* in raw and ready-to-eat chicken products

C. jejuni has been isolated from raw poultry products worldwide, often at prevalence rates exceeding 50% (Wempe *et al.*, 1983; Berndston *et al.*, 1992; NACMCF, 1997).

Some data from the scientific literature concerning the presence of *Campylobacter* in raw and ready-to-eat poultry products (fresh unless stated) overseas are given in Tables 9 and 10 respectively.

Table 9:   Reported prevalence of *Campylobacter* in overseas poultry products

Country	Product	Samples tested	Positive for *Campylobacter* (%)	Year	Reference
Belgium	Chicken and turkey parts	60	40	1996	Uyttendaele and Debevere, 1996
Denmark (Danish produced)	Chicken	133	40	1995	Danish Veterinary and Food Administration (1998)
		186	41	1996	
		637	25	1997	
	Turkey	191	25	1995	
		103	24	1996	
		238	29	1997	

Country	Product	Samples tested	Positive for *Campylobacter* (%)	Year	Reference
France	Raw poultry sausages	115	10	1995-1997	Federighi *et al.*, 1999
Germany	Skin, liver and neck samples, slaughtered broilers	111	45.9	1995-1997 (winters only)	Atanassova and Ring, 1999
Germany	Wild pheasants (skin, caecum and liver)	52	25.9	1995-1997 (winters only)	Atanassova and Ring, 1999
Japan	Imported poultry	54	3.7	1993-1998	Ono and Yamomoto, 1999
Japan	Domestic poultry	33	45.8	1993-1998	Ono and Yamomoto, 1999
Mexico	Chicken	92	36	1992	Castillo-Aya, 1992
N. Ireland	Chicken pieces	120	38	1997-1998	Madden *et al.*, 1998
N. Ireland	Chicken wings	153	65	1994	Flynn *et al.*, 1994
Taiwan	Whole chickens	22	68	2000	Shih, 2000
Taiwan	Breast/wing/ drumstick	17	53	2000	Shih, 2000
UK	Duck carcasses	10	80 (mostly *C. coli*)	1998	Ridsdale *et al.*, 1998
UK (South Wales)	Chicken (whole, breast skin on and pieces) Supermarket Butchers	175 125	75 59	Over 7 months, year NS	Harrison *et al.*, 2001
UK (reweighting of data) England Wales Scotland Northern Ireland	Raw retail chicken Fresh and frozen.	4866 2475 800 794 797	50 46.3 42.3 75.4 76.6	April-June 2001	Food Standards Agency, 2003
NS	Frozen chicken parts	165	2	NS	Bryan and Doyle, 1995
NS	Chicken, frozen	23	100	NS	Bryan and Doyle, 1995
NS	Chicken, retail	100	58	NS	Bryan and Doyle, 1995
NS	Chilled chicken parts	143	12	NS	Bryan and Doyle, 1995
NS	Frozen chicken parts	16	0	NS	Bryan and Doyle, 1995
NS	Frozen chickens	82	22	NS	Bryan and Doyle, 1995

Country	Product	Samples tested	Positive for *Campylobacter* (%)	Year	Reference
NS	Chicken wings	94	83	NS	Bryan and Doyle, 1995
NS	Turkey wings	184	64	NS	Bryan and Doyle, 1995
NS	Chicken chilled	22	68	NS	Bryan and Doyle, 1995
NS	Chicken frozen	37	16	NS	Bryan and Doyle, 1995
NS	Turkey wings, frozen	81	56	NS	Bryan and Doyle, 1995
NS	Chicken	360	30	NS	Bryan and Doyle, 1995
NS	Chicken carcasses	862	23	NS	Bryan and Doyle, 1995
NS	Turkey	168	2	NS	Bryan and Doyle, 1995
NS	Chicken, chilled	18	61	NS	Bryan and Doyle, 1995
NS	Chicken, frozen	24	4	NS	Bryan and Doyle, 1995
NS	Chicken	179	61	NS	Bryan and Doyle, 1995
NS	Chicken	98	32	NS	Bryan and Doyle, 1995

NS = Not stated

These data are comparable to those obtained for New Zealand i.e. prevalence is greater than 50% in raw product and somewhat less in frozen poultry, see Table 1.

Over a seven month study period in South Wales, (Harrison *et al*., 2001), raw chicken samples (whole, breast with skin and pieces) were purchased from supermarkets and butchers shops. *Campylobacter* was present in 75% (n = 175) of supermarket (often pre-packaged) chicken and 59% (n = 125) of butcher's shop chicken, which was often loose and packaged at point of sale. Overall, whole chickens were most frequently positive, followed by breast meat and then chicken pieces. In discussion, the authors noted differences in handling practices and on visual assessment of the pre-packaged poultry from supermarkets, 90% contained trapped surface moisture on the inside of the packaging. This micro-environment may be conducive to the survival of the organism.

In ready-to-eat poultry, the levels of contamination are relatively low. One study found a high prevalence, but this was in a food (tacos) that normally contains non-chicken ingredients. It is therefore possible that contamination could have arisen from other sources, for example the use of contaminated water to wash salad vegetables. In countries comparable to New Zealand, contamination of cooked poultry products is rare, as it is in New Zealand.

Table 10: **Reported prevalence of *Campylobacter* in overseas ready-to-eat chicken products**

Country	Product	Samples tested	Positive for *Campylobacter* (%)	Year	Reference
France	Turkey ham	140	0	1995-1996	Federighi *et al.*, 1999
France	Cooked poultry sausages	45	0		Federighi *et al.*, 1999
Mexico	Roasted chicken tacos	100	27.0	2000	Quiñones-Ramirez *et al.*, 2000
UK	Cooked, chilled chicken products	758	0	1996	Joint Food Safety and Standards Group

Reports of quantitative data for *Campylobacter* in chicken products have been summarised in Table 11.

Table 11: **Reported levels of *Campylobacter* on overseas chicken products**

Product Type	Mean count (cfu cm^{-2})	Range (cfu cm^{-2})	Reference
Wings Producer A Producer B	 16.2 5.0	 3.0-81.3 1.0-45.7	Kinde *et al.*, 1983
Broiler carcasses	0.06	0.02-0.08	Izat *et al.*, 1988
Neck Skin	944	501-2,512	Berndston *et al.*, 1992
Oven ready bird	106.7 36.9	38.7-200.0 3.6-97.8	Pearson *et al.*, 1993
Neck skin	-	50,118-158,489	Abu-Ruwaida *et al.*, 1994
Neck skin	-	36.9-200.0	Mead *et al.*, 1995
Broiler carcasses Pericloacal skin	1.7 - 1,000	1.0-6.9 0.4-444 -	Roberts *et al.*, 1998

cfu = colony forming unit

There is a considerable variation in the numbers that have been measured. However, sampling has occurred from many different areas of the bird. It might be expected that pericloacal skin (around the excretory cavity at the end of the intestinal canal) would have a higher count than other parts of the bird, and neck skin may also have a higher count as carcasses are suspended upside down during processing.

The quantity of *Campylobacter* on the surface of a fresh chicken carcass has been estimated to be 10^3 to 10^6 per chicken in one study in the United Kingdom (Hood *et al.*, 1988).

5.4.3   *Campylobacter* on external packaging

A number of studies have reported *Campylobacter* contamination on the external packaging of poultry.

A study by Local Authorities Coordinators Of Regulatory Services (LACORS) and the Health Protection Agency in the UK during September and October 2002 has recently been published (Health Protection Agency, 2004). A total of 3,662 pre-packaged raw meat 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. The aim of the study was to identify the extent of external surface contamination. *Campylobacter* was detected from 41 (1.1%) of the external packaging samples. Results specific for external packaging of raw poultry are presented in Table 12.

Table 12:   ***Campylobacter* detected on external packaging of raw poultry in the UK**

Meat type	Product	No. of samples	*Campylobacter*/swab (%)	*Campylobacter* isolates*	
				C. jejuni	*C. coli*
Chicken	Whole	170	11 (6.5%)	8	1
	Portions	614	14 (2.3%)	4	5
	Offal (liver)	24	2 (8.0%)	1	1
Game Fowl	Whole	9	1 (11.1%)	0	1
Turkey	Portions	68	1 (1.47%)	0	1

* 7 *Campylobacter* isolates not further identified

Heat sealed packaging was less frequently contaminated compared to other types (overwrapped, bag and tie tape). When normal atmosphere, modified atmosphere and vacuum packing were compared, normal atmosphere packs were the type most frequently contaminated. Finally, less contamination was associated with; intact packaging; visually clean packaging and display areas; display temperature below 8°C and where HACCP was in place.

Over a seven month study period in South Wales, (Harrison *et al*., 2001), the retail packaging of raw chicken was sampled (*n*=300). External packaging only and the whole packaging (inside and outside) were sampled for the presence/absence of *Campylobacter*. The results were;

	% positive	
	External	**Whole packaging**
Supermarket (*n*=175)	4	44
Butchers' shop (*n*=125)	2	21
Overall results (*n*=300)	**3**	**34**

In a London study (Bolton *et al*., 1999), 3 – 8% of the external chicken packaging was contaminated with *Campylobacter*. Packaging is often not removed from the food

preparation areas and this may contribute to cross contamination. Although the percentage of contaminated packaging samples was low, the high numbers of packaged chickens sold (700 million per year in the UK), raises the possibility of cross contamination during handling between selection or purchase, and preparation in the kitchen.

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 ten years ago (Lane and Baker, 1993). Notification data for the period 1990 – July 2006 are given in Table 13, and illustrated in Figures 4 and 5. The highest monthly campylobacteriosis total for 2005 was for the month of November when 1666 cases were notified (ESR, 2006a). All references in Table 13 are Lopez *et al*. (2001), except where stated.

Table 13: **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	
1991	4148	122.9	
1992	5144	152.5	
1993	8101	240.1	
1994	7714	228.6	
1995	7442	220.6	
1996	7628	210.8	
1997	8848	244.5	
1998	11578	320.0	
1999	8173	225.9	
2000	8430	233.0	
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 whereby the rate drops in the winter months. In 1998 this did not occur leading to the abnormally high annual figure.

The age distribution of cases is bimodal with peaks in the 1-4 years age group and 20-29 year group. In 2005, 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). 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 4: **Campylobacteriosis notifications by month January 1999 – July 2006**
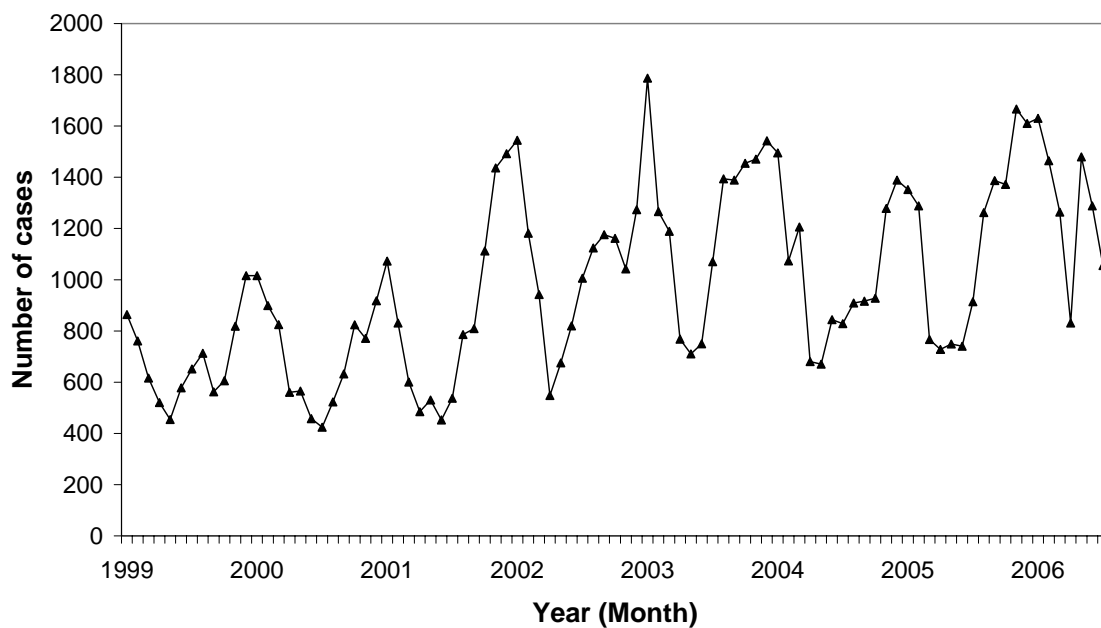
Prepared from ESR data (2006a; ESR 2006b)

Figure 5:   Campylobacteriosis notifications by year 1984-2005
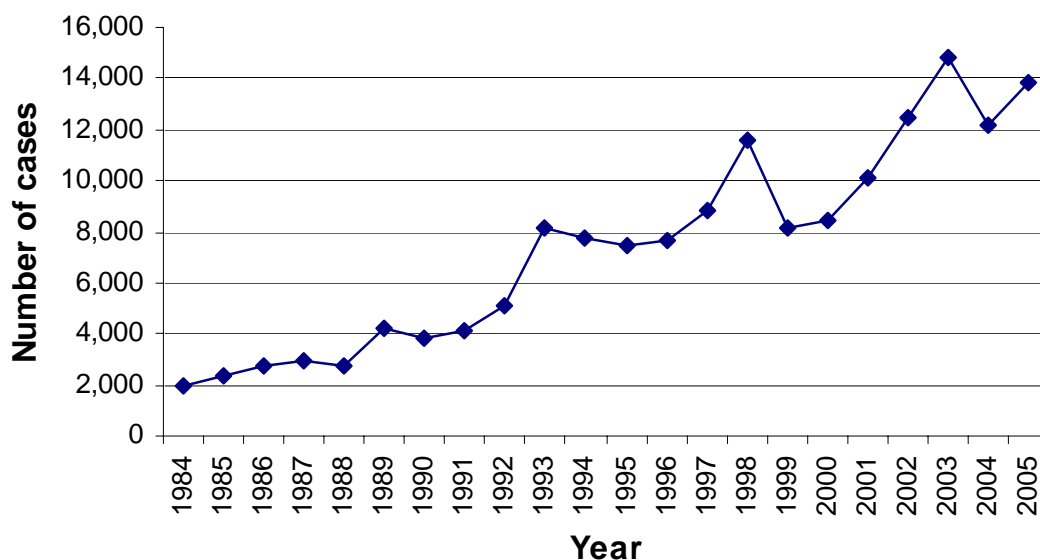
New Zealand's reported rate of campylobacteriosis is high by developed world standards (370.3 per 100,000 in 2005), as shown in Section 6.2.1. However, such comparisons must be made with caution, as reporting practices may differ between countries.

6.1.2   Clinical consequences of *Campylobacter* infection

Hospitalisation and fatality rates for notified cases of campylobacteriosis in New Zealand are given in Table 14. 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 14:   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. It has been claimed that it is 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 overseas outbreaks are less likely to be recognised and reported (Frost, 2001).

In contrast, the New Zealand data summarised in Table 15 show that *Campylobacter* are 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 poultry meat.

Table 15:   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	12.4	140	5.0	ESR, 2004
2004	31	9.5	130	3.2	ESR, 2005b
2005	47	13.6	252	10.3	ESR, 2006b

Outbreaks of campylobacteriosis associated with poultry consumption and reported from 1997 to the end of November 2004 have been summarised in Table 16. Perhaps reflecting the low prevalence of *Campylobacter* in ready-to-eat chicken products, most of the outbreaks appear to have been caused by undercooking of raw chicken or cross contamination. Confirmation of the mode of transmission and the vehicle may be achieved in a variety of ways described by check boxes on the reporting form. The method of confirmation is also given in Table 16.

Table 16: **New Zealand outbreaks of campylobacteriosis with either epidemiological (suspected) links or laboratory confirmation linked with poultry consumption 1997-end November 2004**

Outbreak Number*	Food implicated	Setting	Number ill	Confirmation
AK1997013	Chicken	takeaway	2 C, 3 P	None
BE1998008	Chicken	restaurant	2 C	None
AK1998081	Chicken kebabs-BBQ (u/c)	domestic	4 C, 13 P	None
NL1998006	Microwave cooked chicken (u/c)	domestic	2 C, 1 P	None
NL1999001	Chicken	restaurant	2 C	None
CB1999014	Chicken teriyaki (c/c)	takeaway	2 C	None
WN1999027	Chicken fettucine with pesto	restaurant	2 C	None
WN1999009	Chicken meal	restaurant	3 C	None
WN1999032	Chicken sushi	restaurant	2 C, 1 P	None
AK1999092	Chicken curry (c/c)	domestic	1 C, 1 P	None
AK1999153	Chicken (u/c & c/c)	domestic	3 C	None
WN1999040	Chicken kebab (u/c)	takeaway	2 C	None
AK2000036	Chicken kebabs (lamb kebabs & other high risk foods)	commercial boat trip	5 C, 4 P	None
AK2000103	Chicken kebab (u/c)	restaurant	3 C, 1 P	1, 4
AK2001024	Chicken (u/c), c/c to King Prawns for 3rd case	restaurant	3 C	1, 4
AK2001065	Chicken nuggets	takeaway	2 C	1
TK2002001	Chicken kebabs BBQ	domestic	4 C	2
AK2002025	Chicken kebab (c/c)	takeaway	2 C	1, 4
AK2002074	Chicken salad (c/c)	restaurant	1 C, 1 P	1,4
AK2002092	Chicken, bacon & avocado burger	restaurant	1 C, 1 P	1
AK2002099	Chicken and vegetable pie	restaurant	1 C, 3 P	1
AK2002100	Chicken in combination kebab (c/c)	takeaway	3 C, 1 P	1, 3, 4
AK2002105	Chicken kebab (c/c, poor hot holding)	takeaway	2 C, 1 P	1, 4
AK2002134	Chicken salad (u/c, c/c)	restaurant	1 C, 4 P	1, 4
AK2002151	Chicken & potato top savouries	conference caterers	3 C, 8 P	1, 4
AK2002176	Chicken salad (or acquired overseas)	restaurant	1 C, 1 P	1
CB2002003	Chicken sushi (c/c, inadequate cooling)	takeaway	5 C	None
CB2002004	Chicken kebabs BBQ (u/c)	school	7 C	1
CB2002005	Chicken fettucine, Chicken salad (c/c)	restaurant	4 C	1

Outbreak Number*	Food implicated	Setting	Number ill	Confirmation
CB2002009	Chicken sushi (inadequate cooling/refrigeration)	takeaway	9 C	1
CB2002023	Chicken (c/c)	restaurant	4 C, 2 P	4
CB2002025	Chicken meal (u/c)	restaurant	2 C, 1 P	1
CB2002027	Chicken meal	domestic	3 C, 1 P	1
WC2003006	Chicken	takeaway	1 C, 4 P	1
AK2003033	Chicken salad (u/c), (also raw egg in dressing)	restaurant	1 C, 1 P	1, 4
AK2003034	Chicken burger (u/c, poor hot holding)	takeaway	1 C, 1 P	1, 4
AK2003179	Chicken curry, butter chicken	takeaway	3 C, 2 P	1, 4
WN2003011	Chicken teriyaki and sushi dishes (c/c)	restaurant	1 C, 1 P	1
AK2004135	Chicken nuggets or person to person	restaurant	2 C	1

* Numbers are unique reference numbers assigned by the ESR Notifiable Disease Database (EpiSurv)

u/c = undercooked

c/c= cross contamination

C=confirmed

P=probable

1 epidemiological (suspected) links– cases had history of exposure to implicated source

2 epidemiological (suspected) links– case control or cohort study showed elevated risk for cases exposed to implicated source

3 laboratory – pathogen suspected to have caused illness identified in implicated source

4 environmental investigation (suspected) links – identified critical control point failures linked to implicated source

The category of "environmental investigation" is where identified critical control point failures have linked the outbreaks to the implicated source.

It should be noted that these food implications are based mainly on one category of epidemiological (suspected) links; -case history of exposure to the implicated source. This is probably the weakest confirmatory evidence. Only one outbreak (TK2002001) has epidemiological evidence from a case control or cohort study which is a more reliable assessment.

Only one other outbreak has laboratory confirmation of *Campylobacter* in the implicated food i.e. chicken in a combination kebab (AK2002100). In this particular outbreak, there was also a case history of exposure and critical control point failures linked to the kebab. Out of the 39 reported outbreaks above, 30 were in a restaurant or takeaway setting, compared to 6 in a domestic setting. Where a factor is suggested as a cause of the infection, undercooking appears in 11 of the outbreaks while cross-contamination appears in 13.

Data relating the type of *Campylobacter* in the food and the type causing disease are rare, and greater use of typing would reinforce associations between implicated foods and outbreaks.

6.1.4   Case control studies and risk factors

Two New Zealand case control studies of campylobacteriosis have been published in the scientific literature. An overview of the risk factors related to chicken is presented in Table 17.

Table 17:   New Zealand case control studies containing information on *Campylobacter* in chicken

Risk/Protective factor	Odds ratio (CI)	Reference, year
Eating undercooked poultry (risk)	4.94 (1.03, 23.62)	Ikram *et al*., 1994
Poultry eaten at a friend's house (risk)	3.18 (1.0, 10.73)	Ikram *et al*., 1994
Consuming fresh chicken (as opposed to frozen) (risk)	1.8 (0.82, 3.82)	Ikram *et al*., 1994
Eating poultry at home (protective)	0.36 (0.14, 0.9)	Ikram *et al*., 1994
Freezing fresh chicken before consuming (protective)	0.58 (0.18, 1.83)	Ikram *et al*., 1994
Buying frozen chicken (protective)	0.71 (0.34, 1.31)	Ikram *et al*., 1994
Recent consumption of raw and undercooked chicken (risk)	4.52 (2.88, 7.10)	Eberhart-Phillips *et al*., 1997
Chicken eaten in restaurants (risk)	3.85 (2.52, 5.88)	Eberhart-Phillips *et al*., 1997
Chicken purchased frozen (protective)	0.61 (0.48, 0.77)	Eberhart-Phillips *et al*., 1997
Chicken baked or roasted (protective)	0.75 (0.60, 0.94)	Eberhart-Phillips *et al*., 1997

CI = confidence interval

The first case-control study (Ikram *et al*., 1994) was conducted in the summer of 1992-1993 in urban Christchurch. One hundred each of cases and controls were included and the questionnaire format addressed the major risk factors for campylobacteriosis. The study concluded that poorly cooked or handled chicken was a significant source of human campylobacteriosis. Consumption of undercooked poultry, or poultry eaten at a friend's house were significantly associated with risk of campylobacteriosis. Poultry consumed at home or bought frozen were associated with reduced risk. There was significant risk associated with consumption of barbecued chicken, but not with consumption of barbecued beef, mutton/lamb or salads.

There was no significant risk in the handling of human waste, raw meat, pet ownership or time spent on a farm. The paper also stated that there was no risk associated with handling of chicken or offal, raw beef, pork, mutton/lamb and no risk associated with using the same chopping board for meat and vegetables. This apparently contradicts the conclusion regarding handling of chicken as a risk factor above; however the definition of handling is not given in this section, and neither are data supporting this statement. Drinking water from a rural water source had an elevated odds ratio (OR2.7, CI 0.89, 8.33), but this was not statistically significant.

The more recent (and larger) 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. Some aspects of food exposures were investigated in more detail, particularly cooking methods for meat, poultry and fish, and home food handling practices.

The strongest associations were between campylobacteriosis and undercooked chicken, or consumption of chicken meat in restaurants.   There was no association between meats other than poultry and campylobacteriosis. Salads and vegetables appeared to be protective. There were no links between food preparation practices in the home and campylobacteriosis.

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 combined population attributable risk (PAR) percentage for the chicken related variables in the multivariate model exceeded 50%, suggesting that consumption of chicken lay behind more cases of campylobacteriosis in New Zealand than all other risk factors combined. Raw or undercooked meat or fish, as well as unpasteurised milk were the other foods associated with increased risk, with a population attributable risk of 11 and 7% respectively.

While the case control studies reviewed here identified risk factors other than aspects of chicken handling/consumption (e.g. drinking untreated water, unpasteurised milk) it is clear that poultry features significantly in these studies. Some aspects of poultry handling are listed as protective and this is especially so in the New Zealand studies. Some are biologically plausible, for example frozen chickens may be less frequently contaminated and at lower levels than fresh poultry. Others have been explained by heightened awareness of the consumer.

Auckland Healthcare has carried out 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). There was an increased risk of campylobacteriosis associated with fast foods, and consumption of barbecued chicken (but not chicken cooked by other methods). Eating undercooked chicken elevated the risk of illness although this was not statistically significant. No increased risk was associated with a wide range of other foods including meat, seafood and dairy products. Of the non-food risk factors, only travel outside New Zealand was associated with an increased risk of illness, although the risk from having a rainwater-derived water supply approached statistical significance.

An outbreak at a family barbecue (17 cases) in October 1998 was investigated by a retrospective cohort study (Bishop, 1998). The most likely source of infection suggested by epidemiological results was chicken kebabs.

During the power shortage in Auckland in February 1998 a sharp increase in notifications of isolates of *Campylobacter* spp. by community laboratories led to a case-control study (Calder

et al., 1998).   Of 170 sick people from whom isolates had been obtained, 139 were interviewed. The study was unable to determine the source of the epidemic. Elevated (but not statistically significant) odds ratios were associated with eating chicken. Several other risk factors had elevated odds ratios which were not statistically significant. These included using a mains water supply, but this was not considered a feasible source due to the wide distribution of cases across several water supplies.

With the support of the Enteric Zoonotic Disease Research Group (EZDRG), the National Institute of Water and Atmospheric Research Ltd (NIWA) have published a collaborative comparative exposure assessment (QRA) for *Campylobacter* exposure in New Zealand prepared for the Ministry of Health (NIWA, 2004). Collaborative authors were drawn from NIWA, ESR and the NZFSA as part of the EZDRG co-ordinated programme.   The comparative model examined four potential human exposure routes: recreational swimming, drinking water, food (poultry and red meat), and occupational animal contact.

The model for poultry exposure took into account data available on bacterial loading, cross contamination and undercooking. In terms of relative frequency of infection, the National Model tentatively concludes that cross-contamination from poultry was the most important exposure, followed by cross contamination from red meat and occupational contact with infected animals. Eating undercooked poultry was a relatively infrequent cause of infection.

6.1.5   Serotypes causing human disease in New Zealand

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 heat stable HS4 complex, HS2, and HS1,44 (53.8% of all cases).

Certain serotypes, particularly Penner serotype O19 and O41 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).

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 18. New Zealand's rate is high by international standards, although some differences may be due to reporting practices. The dissimilar geographies of the countries listed may also be a factor. Topographical differences within countries such as Canada and the United States (e.g. green pastures mixed with large areas of desert, mountains and tundra) may also dampen down the rate in the overall population.

Table 18:   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	116	ACMSF, 2004
	2000	33	
Ireland	2001	35.5	NDSC, 2002
UK;			
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 which does not report campylobacteriosis except when an outbreak occurs.
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 *et al*., 1993).   In England and Wales increased campylobacteriosis was associated with increased temperature rather than the season *per se*, especially in children under 5 (Louis *et al*., 2005).

In the UK, *Campylobacter* infection is the most prevalent reported foodborne disease. In 2000, 62,867 cases of campylobacteriosis 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* making up 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* infections have 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 decreased 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 here 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 19. The low percentages reinforce the sporadic nature of this illness.

Table 19:   Contribution of *Campylobacter* to reported foodborne disease outbreaks, incidents and cases overseas

Country	Year	No. (%) Outbreaks	No. (%) incidents or cases	Reference
Australia	1995-2000	6 (3)	136 (2) cases	Dalton *et al.*, 2004
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.*, 1998b
UK	1996	8 (1.1)	99 (0.5)	Evans *et al.*, 1998b
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 poultry consumption that have been reported in the scientific literature have been summarised in Table 20.

Table 20:   Overseas campylobacteriosis outbreaks associated with poultry consumption

Country	Food implicated	No. ill	Attack rate	Evidence for food implicated	Reference year
Australia	Chicken casserole	7	58%	Epidemiological. Same type in cases found in raw chicken ingredient.	Rosenfield *et al.*, 1985
England	Chicken pieces	12	38%	Epidemiological. Raw chicken product not cooked.	Murphy *et al.*, 1995
England	None specified	19	NS	Epidemiological. Isolate serotyping.	Pearson *et al.*, 2000
England	Stir fried chicken pieces	12	41%	Epidemiological. Isolate typing.	Evans *et al.*, 1998a
England and Wales	Poultry	4	44%	Descriptive	Pebody *et al.*, 1997
Netherlands	Undercooked/indirect cross-contamination chicken	89	72%	Epidemiological. Live chickens given to 123 cadets to kill/prepare over wood fire.	Brouwer *et al.*, 1979

Country	Food implicated	No. ill	Attack rate	Evidence for food implicated	Reference year
UK	Undercooked chicken	21	15%	Epidemiological, isolate typing, serology	Skirrow *et al.*, 1981
USA	Undercooked chicken (?)	9	60%	NS	Finch *et al.*, 1985
USA	Processed turkey	11	NS	Epidemiological	Shandera *et al.*, 1992
USA	Undercooked barbecued chicken	11	73%	Epidemiological, raised patient antisera, stool +ve	Istre *et al.*, 1984

Again most outbreaks are attributable to the undercooking of chicken products.

Poultry was identified as the vehicle in two of 21 outbreaks of campylobacteriosis in England and Wales between 1992 and 1994. However, unpasteurised milk and water supplies were identified as the source in a greater number of the same set of outbreaks (three and six outbreaks respectively) (Frost, 2001).

In the United States differing epidemiologic characteristics between outbreaks and sporadic cases of campylobacteriosis have been identified (Altekruse *et al.*, 1999). Most outbreaks occur during spring and autumn, and consumption of raw milk (often during school field trips) was implicated in 55% (30/55) of foodborne outbreaks with known identified food sources between 1976 and 1996 (Friedman *et al.*, 2000). Following public health warnings about consumption of raw milk, especially during farm visits by schools, the frequency of milk associated outbreaks has declined in the USA. In fact, there has been a shift in outbreak sources in that country; from 1978 to 1987, water and unpasteurised milk accounted for 56% of all outbreaks, while between 1988 and 1996 other foods accounted for 83% of all outbreaks.

Cross contamination from contaminated raw poultry to other ready-to-eat foods can also be a source of infection. For example, 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).

Handling raw poultry and eating undercooked poultry have been identified as the most important risk factors, with other less important risk factors being drinking untreated water, travelling abroad, eating barbecued pork or sausage, drinking raw milk and contact with pets (Altekruse *et al.*, 1999; Freidman *et al.*, 2000). Person to person or secondary transmission is uncommon. Overlap between the serotypes found in humans, poultry and cattle have been found, suggesting that foods of animal origin play an important role in transmission. A correlation between the seasonal fluctuations in prevalence of *Campylobacter* in broiler flocks and numbers of human cases has been demonstrated in Denmark (Nielsen *et al.*, 1997) although this may only indicate that both are being infected from the same source.

The Belgian dioxin crisis in 1999 inadvertently provided evidence of the importance of poultry as a transmission vehicle for *Campylobacter* (Vellinga and Van Loock, 2002). Following the discovery of dioxin contamination in livestock feed, on 28 May 1999 Belgian authorities ordered the withdrawal from sale of Belgian poultry and eggs. On 4 June 1999, the Belgian government issued a commerce embargo of meat products (pork and beef) with a

minimum of 25% fat content. However, neither meat nor dairy products were withdrawn from sale.

Historical data from the Belgian sentinel surveillance system were used to model the expected number of *Campylobacter* cases for 1999. The actual number of *Campylobacter* infections reported during 1999 fit the model very well (within the 95% confidence interval) except during the four week period when poultry and eggs were removed from the shelves. During that period the number of reported cases of *Campylobacter* infection was 40% below that expected. After four weeks, the ban was lifted and campylobacteriosis notifications returned to the expected level.

6.2.3   Case control studies

Case control studies of campylobacteriosis conducted overseas have been summarised in Table 21.

Table 21:   Case control studies containing information on *Campylobacter* in poultry

Country	Risk/Protective factor	Odds ratio (CI)	Reference
England	Handling any whole chicken in the domestic kitchen that had been bought raw with giblets and eaten at home (protective)	0.41-0.44 (0.24, 0.79)	Adak *et al*., 1995
Southeastern Norway	Poultry bought raw and frozen (risk)	2.42 (1.03, 5.67)	Kapperud *et al*., 1992
Switzerland	Consumption of poultry (risk)	1.8 (1.0, 3.4)	Schorr *et al*., 1994
Sweden	Eating chicken (risk) Hen/chicken breeder (risk) Daily contact with hens/chickens (risk)	2.29 (1.29-4.23) 3.32 (1.56-6.78) 11.83 (3.41-62.03)	Studahl and Andersson, 2000
UK	Eating chicken 1-5 times in previous 2 weeks (risk) Eating chicken 6+ times in previous 2 weeks (risk) Handling raw poultry (risk)	1.5 (1.0, 2.0) 2.9 (1.1, 7.7) 1.4 (1.0, 2.0)	Neal and Slack, 1995
Wales	Eating chicken (risk) Eating out at a fried chicken outlet (risk)	1.61 (1.03, 2.50) 1.82 (1.00, 3.30)	Evans *et al*., 2003

CI = confidence interval

Again the consumption of poultry features as a risk factor in these studies, with one exception (Adak *et al*., 1995) where handling of chicken in the home was found to be protective.

Several case-control studies have been conducted in the USA (Friedman *et al*., 2000). A large study in Seattle in 1981 showed that eating poultry, including chicken, turkey, and Cornish game hen, accounted for over 50% of cases. Other cases were attributed to raw milk (5%), contact with pets (6%), drinking contaminated surface water (8%) and overseas travel (9%). In another study at a Georgia University 70% of cases were attributed to eating

chicken. A study in Colorado identified handling raw chicken, as opposed to eating it, as a risk factor.

Data on cases of *C. jejuni* infection collected during the large study of infectious intestinal disease in England has been reported as a case-control study (Rodrigues *et al.*, 2000). Only two factors were significantly associated with increased risk of campylobacteriosis: travel abroad (considered to explain 9% of cases) and eating chicken in a restaurant or canteen (considered to explain 11% of cases). The odds ratios for other forms of chicken consumption (barbecued, takeaway, fast food, eaten at home etc.) did not approach statistical significance. Neither did a number of other risk factors often thought to be associated with campylobacteriosis, including contact with pets and other animals, and various domestic food handling practices.

One hypothesis for the association between poultry and campylobacteriosis is that cross contamination occurs in the kitchen, so that poultry is the source but not the vehicle of infection. It was acknowledged in this study that domestic food handling practices are notoriously difficult to measure and possibly unhygienic behaviour was not disclosed. An alternative hypothesis suggested by the authors was that otherwise acceptable domestic food handling is insufficient to prevent low level cross contamination, which results in infection (Rodrigues *et al.*, 2000).

A case-control study on *Campylobacter* infections in infants and young children in Australia found that the most important risk factors were ownership of pet puppies and pet chickens. The only food significantly associated with infection was mayonnaise, which the authors were unable to explain (Tenkate and Stafford, 2001).

6.2.4   Risk assessment and other activity overseas

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

In the UK, a detailed *Campylobacter* Sentinel Surveillance Scheme of clinical cases was initiated from May 2000 until April 2003 (Health Protection Agency, 2003). Reference typing focused on cases from 22 District Health Authorities (representing 12.5 million people) representing approximately 15% of all laboratory confirmed cases in England and Wales. In 2001, Scotland and Northern Ireland joined the scheme. The use of case-case analyses for the first year's data revealed significant differences in risk behaviour associated with the two predominant species, *C. jejuni and C. coli*, such as;

- Cases of *C. coli* were more likely to have drunk bottled water or eaten pâté than cases of *C. jejuni*,
- Foreign travel was an important risk factor with a fifth of reported cases acquired abroad.

In a further examination of food exposures in the above study, compared with results from the UK 1999 National Food Survey, campylobacteriosis cases were more likely to have consumed pre-packed sandwiches, pâté, meat pies and offal.

The Advisory Committee on the Microbiological Safety of Food (ACMSF, 2004) Second Report on *Campylobacter* written for the UK Food Standards Agency, made priority recommendations for dealing with the organism. One such priority recommendation is the expansion of these population studies and the gathering of more extensive data. This has been implemented (November 2004) so that the Sentinel Scheme now applies across the UK. The Co-ordinated Local Authority Sentinel Surveillance of Pathogens (CLASSP) coordinates the long-term scheme between voluntary local authorities taking part and the Food Standards Agency. The Authorities are requested to commit to both;

- Low-level submission of poultry samples (whole raw chicken carcass), and
- Foodborne disease investigations for sporadic laboratory confirmed human cases of *Campylobacter* and *Salmonella* infection reported in their area.

The set protocols include guidance on sampling and disease investigation. For example, the overall total of frozen items should form approximately 25% of the samples taken and about 5% of the samples should be production types other than intensely reared (e.g. free range).

The FAO/WHO (2002) have recently published the first part of a quantitative risk assessment that deals with the hazard identification, hazard characterization and exposure assessment for *Campylobacter* in broilers. Since the risk assessment is incomplete, no conclusions can yet be drawn about the contribution that this risk/hazard combination makes towards disease.

Much of the information presented is based on the poultry industry in the UK, but there is significant commonality between the processes described and what occurs in New Zealand. The document presents a detailed description of the process and explains the modelling used at each step. A novel aspect of this document is the attempt to model cross contamination in the home. It is clear that while models may be constructed, there are very few data to be used in the models. Also detailed are approaches to modelling the cooking of chicken products, none of which seems to be entirely satisfactory.

The model has not yet progressed to producing a full exposure assessment, but when it does, it is likely to use data from the USA or the UK. Some aspects of this model have been published (Hartnett *et al.*, 2001).

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. Figures from 1993-1997 indicate that numbers of cases more than double in late summer and autumn, with 10-19 year olds most commonly affected.

The Risk Profile also described a case-control study (227 cases/250 controls) carried out in Denmark from May 1996 to September 1997. Significant risk factors were:

- Insufficiently heat treated poultry (especially chicken) (OR 5.5, p=0.003),
- Meat prepared by grill or fire (OR 2.3, p=0.002)
- Poor quality drinking water from a private well (OR 3.0, p=0.008).
- Travel abroad (figures not given).

The above risk factors were considered to explain approximately 50% of the human cases, (5-8% insufficiently heat treated poultry, 15-20% red meat prepared by grill, 5-8% to drinking water, and 15-20% to journeys abroad).

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, which has control of *Salmonella* as the 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 thermophilic *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. It was concluded that 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   Qualitative Estimate of Risk

In New Zealand, surveys from 1984 to 2004 suggest that upwards of 50% of fresh raw chicken available for retail sale contains *Campylobacter*. This prevalence is generally similar to findings overseas. In the most recent national retail survey, 89.1% of the minced/diced chicken samples were positive for *Campylobacter*. *C. jejuni* has also been found on the exterior packaging of raw chicken in New Zealand; 34% of whole chicken and 14.5% of chicken portions (Wong *et al.,* 2004). This raises the potential for cross contamination during purchase, transport, and handling in the home.

New Zealand has a relatively high proportion of outbreaks in which *Campylobacter* are identified as the causative agent. In 2005, 47 outbreaks of campylobacteriosis involving 252 cases represented 13.6% of the total number of outbreaks(ESR, 2006b).

Chicken and chicken-based foods are associated with transmission in many of these outbreaks. Similarly in sporadic cases evaluated in case-control studies, factors associated with poultry meat consumption have been linked most strongly with risk of campylobacteriosis. Undercooking or consumption of chicken meat away from home were the major risk factors.

It seems reasonable to conclude that poultry meat is an important vehicle for transmission of campylobacteriosis 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 the foods included in the study totaled 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 2005 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. From the population attributable risks assigned in the case control study (Eberhart-Phillips *et al*., 1997), consumption of poultry represented the large majority of the risk. This suggests that *Campylobacter* in poultry should be assigned the highest incidence category (>100 per 100,000).

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 poultry	3 (<0.5% serious outcomes)	1 (>100 per 100,000)	Low currently, but likely to rise	Poor overseas image, NZ is "*Campylobacter* capital of the world"

7   RISK MANAGEMENT INFORMATION

7.1   Relevant Food Controls

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

- Reduction of the prevalence of the hazard in poultry flocks,
- Control of the hazard during or following processing, and
- Elimination of the hazard by the end users i.e. consumers and the food service industry.

7.1.1   On farm control

Many on-farm studies of the epidemiology of infection of broiler flocks by *Campylobacter* have been published and they cannot all be summarised here. Presented below are some of the factors that have been considered in these studies, but no clear consensus on the most important transmission routes of flock infection by *Campylobacter* has yet emerged.

Campylobacter are not usually found in broilers until the third week of life. There is no agreement as to why. Some suggestions include; maternal anti-*Campylobacter* antibodies in egg yolks and young chicks, or the presence of bacterial flora that are antagonistic to *Campylobacter*.

Bacteria may enter the flock environment from a variety of sources: contaminated water, feed, domestic/wild animals including pests such as flies, transport crates, vehicles, personnel etc. Although a number of authors have investigated the potential for vaccination, an effective vaccine strategy directed against *Campylobacter* in broiler chickens has yet to be developed (Newell and Wagenaar, 2000).

Incentives for the farmer are limited, however, because colonisation of most animal species with *Campylobacter* does not represent an animal health/welfare issue; nor is it a problem for farmers in terms of animal production. In addition, prevention of infection in broiler flocks appears to be extremely difficult.

Control measures introduced to control *Salmonella* in broilers in the United Kingdom and New Zealand have included treatment of feed, biosecurity in the hatchery, in the feedmill and on the farm, *Salmonella*-free parent and grandparent flocks, vaccination of breeders and competitive exclusion.   While these measures appear to be effective in controlling *Salmonella*, similar measures appear to be ineffective against *Campylobacter* (Corry and Atabay, 2001). The use of dedicated boots for each poultry house and the regular use of foot dips have been found to be important factors in preventing the introduction of *Campylobacter* in broiler flocks, but even with the most stringent biosecurity measures, infection appears to be impossible to prevent completely. Once infection has entered the chicken house, most or all birds become *Campylobacter* carriers very quickly (Pattison, 2001).

Feed withdrawal is another on-farm control aimed at minimising cross contamination of bacteria through the spillage of gut contents and faeces during processing. Fasting periods of 8 hours are the standard in New Zealand, while overseas they can be between 7 and 20 hours once catching, transportation and lairage are taken into account. This does not necessarily mean that longer fasting periods are beneficial. There are pros and cons to fasting and

currently there is no consensus on whether this is a beneficial step (ACMSF, 2004). On the plus side, there is a reduction of gut contents which reduces pressure and leakage should the intestinal tract become cut or torn. On the other hand, most reduction occurs in the crop and least in the caeca and cloaca. The contents, particularly of the crop and cloaca become wetter with longer deprivation while the caecal contents become slightly drier. Birds also tend to consume more litter as the fasting period progresses. Fasting therefore progressively increases the number of *Campylobacter* in the gastrointestinal tract, especially in the caeca and cloaca. It also reduces the amount of faeces deposited on transport crates that may affect intra- and inter-flock spread prior to slaughter (Rigby and Pettit, 1981).

Stress may also predispose the fasting birds to *Campylobacter* infection (ACMSF, 2004). One study has shown that the longer the fasting period (up to 24 hours), the higher prevalence of *C. jejuni* in crop samples before slaughter (Byrd *et al*., 1998).

The establishment of strict hygienic barriers at each poultry house has apparently resulted in reduced flock prevalences in Scandinavia (Scientific Committee on Veterinary Measures Relating To Public Health, 2000). These barriers include:

- Hygienic routines when farm workers enter the rearing room;
- Avoiding partial slaughter of flocks;
- Active pest control;
- Avoiding contact with other animals and non-authorised personnel;
- Disinfection of drinking water.

The Committee's report claimed that use of such methods (particularly the "all in and all out" approach) had enabled 60% of Swedish farms to consistently produce batches of broilers without *Campylobacter*. The overall flock prevalence of *Campylobacter* was stated to have dropped from 50% to 10%.

7.1.1.1 Poultry industry controls to prevent flock contamination by Campylobacter in New Zealand

The poultry industry in New Zealand undertakes specific measures to control and monitor *Salmonella* contamination in broilers, feed, and the environment (buildings) (PIANZ, 1995). There are also generalised hygiene and biosecurity controls for broiler houses (PIANZ, 1995), which will assist in the control of *Campylobacter* infection in flocks. Specific additional control measures targeted at *Campylobacter* have not been identified.

Basic biosecurity measures in New Zealand currently include;

- Controls on visitors
- Use of footbaths and designated shed boots
- Disinfection of drinking water supplies with 2ppm free available chlorine
- Non use of surface water
- Full shed cleanout each run and sanitation with approved chemicals
- Use of approved sources of litter material (must be clean, dry and untreated)
- Shed design must minimize rodent access
- Control programs for rodents and wild birds

- Heat treated feed, and
- A standard 8 hour feed withdrawal program

(Brian Jones, Inghams, personal communication, 20 Dec 2004).

7.1.2 Control during or after processing

Control of cross contamination at slaughter is considered difficult to implement. The primary steps at which cross contamination could occur are:

- In contaminated cages during transit to the plant,
- At the beginning of processing plant prior to scalding,
- Scalding,
- Defeathering,
- Evisceration, and
- Chilling.

It has been claimed that the poultry processing system makes cross-contamination from *Campylobacter*-infected to *Campylobacter*-free carcasses unavoidable (Corry and Atabay, 2001). Improvements in processing procedures that have been suggested are (Jacobs-Reitsma, 2000):

- Counterflow water systems during scalding and chilling
- Rinsing and washing of equipment to minimise or reduce cross contamination
- Washing and rinsing carcasses to reduce overall bacterial load
- "Logistical" slaughter of uninfected flocks before infected ones.

Campylobacter declines in numbers during frozen storage on chicken skin (Lee *et al*., 1998) and so the likelihood of detecting a positive sample also reduces with time. If the numbers of *Campylobacter* cells on frozen poultry decrease then the probability of exposure to *Campylobacter* resulting from the consumption of the product must also decline. Freezing of poultry products may therefore present a risk management option. However, since the observed effect of freezing may, at least in part, be a reflection of the limits of analytical methodology, then this option would need further verification or evidence to suggest efficacy before being considered for adoption.

A project on the effect of freezing and chilling temperatures on *Campylobacter* on poultry meat has recently been completed by ESR (Whyte *et al*., 2005). The project was comprised of a literature review, a survey of industry "crust freezing" techniques and experiments to determine the effect of different freezing rates and temperatures on the reduction of *Campylobacter* numbers.

The literature review concluded that freezing rate will influence *Campylobacter* survival more-so than frozen storage. Slow freezing was more lethal than rapid freezing because of osmotic stress. Very high rates of freezing (in excess of 10°C/min) can reduce bacterial survival by creating intracellular ice crystals and subsequent mechanical cell damage, though these rates are difficult to achieve in industry. Overall, the literature suggested that

Campylobacter was reasonably tolerant of chilling but reductions could be made if an optimum freezing rate and temperature was used.

The second part of the project was the assessment of "crust freezing" on the survival of *Campylobacter*. The industry crust freezing process involves lowering the temperature of chicken products from 0 to -2°C over 110 minutes, holding for 150 minutes, then allowing the temperature to rise to 2°C over the following 24 hours. Crust freezing was developed to extend shelf life rather than reduce the number of pathogenic bacteria that might be present on the product. Naturally occurring *Campylobacter* was measured on chicken portions obtained prior to and following crust freezing in two factories. The data indicated that crust freezing did not cause a significant change in *Campylobacter* numbers. No evidence of cellular injury was found. The conclusion was that crust freezing, was not reducing the *Campylobacter* contamination on fresh poultry.

The third part of the project was conducted in a laboratory setting. Two sets of experiments were carried out, the first assessing *Campylobacter* survival when frozen to temperatures of between -2 and -10°C in a chicken juice medium, the second investigating *Campylobacter* survival when inoculated onto chicken portions and frozen at two different rates to -2 or -10°C. Significant reductions in *Campylobacter* numbers were only observed when inoculated chicken portions were frozen to -10°C. This effect is possibly due to the longer cooling time necessary to reach -10°C (19h 40min), compared to a target temperature of -2°C (4h, 20min), when maintaining a set rate of cooling. Legal and practical reasons would currently prevent these longer time/temperature parameters from being used in industry.

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.
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.

By 1 November 2002, all animal product primary processing businesses, except those exempt under the Act or under the Animal Products (Exemptions and Inclusions) Order 2000 , must have a risk management programme. A risk management programme is a documented programme to identify and manage biological, chemical and physical hazards. The programme is to be based on the principles of Hazard Analysis and Critical Control Point (HACCP): identifying the hazards, the systems of control, and demonstrating that the controls are effective. Risk management programmes are to be designed by individual businesses for the animal materials used, the processes performed and the product range produced.

7.1.3 Consumers

General consumer advice for control of pathogens in poultry 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 website also advocates measures to avoid cross contamination from raw poultry.

In New Zealand, the Foodsafe Partnership (see website: http://www.foodsafe.org.nz) has included cross contamination and the cooking of poultry amongst the general advice it has promoted in various campaigns.

Information is also available with food safety tips regarding *Campylobacter* from the poultry industry website; www.pianz.co.nz and associated company websites.

7.1.4 Risk management studies overseas

Risk management may target factors contributing to contamination, or else introduce new treatments to reduce or prevent contamination. Management may take place on farm, during slaughter/processing, or else during handling in domestic or foodservice environment.

A number of decontamination methods during processing have been investigated, but only irradiation appears to be completely effective (Corry and Atabay, 2001). Irradiation of packaged fresh or frozen poultry products at 1.5 to 3.0 kG has been approved by the FDA in the USA and several other countries (Jacobs-Reitsma, 2000) but is not permitted in New Zealand.

Processing controls that are either in use, or in development, include:

- Gamma radiation,
- Ultra-violet radiation,
- Electron beam radiation,
- Antimicrobials;
    - Chlorinated water sprays/ spin-washes (only potable water can be used in EU processing plants),
    - Acidified sodium chlorite dips (Oyarzabal, 2004),
    - Cetylpyrinium chloride,
    - Sodium hypochlorite,
    - Chlorine dioxide,
    - Ozone,
    - Peroxyacetic acid,
    - Trisodium phosphate (TSP),
- Removal of skin,
- Air chilling to reduce carcass temperature (drying effect),
- Use of high temperatures (scalding treatments), and
- Low temperatures (crust freezing, super-chilling in liquid nitrogen).

In the US, chlorine in the form of sodium hypochlorite, calcium hypochlorite tablets or chlorine gas is the most commonly used disinfectant in the poultry industry (Russell and Axtell, 2005). However, the effectiveness of chlorine as an antimicrobial is quickly counteracted by organic matter in chill water. Chemical alternatives to chlorine include organic acids (e.g. lactic acid), although these may cause skin discolouration, and alkaline solutions (trisodium phosphate at pH 11.5) (ICMSF, 1998). The effectiveness of 10% trisodium phosphate in controlling pathogenic microorganisms has been shown (Whyte *et al.*, 2001).

A review from a US perspective has been carried out on commercial antimicrobials by Oyarzabal (2005). He found acidified sodium chlorite (ASC) dips were especially effective. The ASC SANOVA® produced by Ecolab, (Alcide Corporation, Redmond, Wash.) has FDA approval and is used in industry in the USA. ASC combines with organic matter producing several broad spectrum oxychlorous antimicrobial compounds. These oxidize sulphide and di-sulphide bonds on cell membrane surfaces. ASC is sprayed or used in a dip solution before the prechill or chill tank stage. Concentrations are between 500 and 1200 ppm, acids used are generally recognised as safe (GRAS) such as citric acid (final solution pH 2.5 to 2.9). The author cites several studies that found the combination of bird washers with ASC sprays removed faecal contamination (a primary source of contamination). Reductions up to 99.2% in *Campylobacter* numbers were achieved. It is suggested that the application of ASC exerts indirect stress on the *Campylobacter* cells during the subsequent chilling process.

In Australia, a small trial using ASC dip (SANOVA®) on poultry carcasses was recently carried out to determine its effectiveness (Sexton *et al.*, 2005). *Campylobacter* was one of the pathogens being evaluated. A known positive flock was selected for the treatment. The *Campylobacter* prevalence reduced from 30/30 (100% - untreated controls) to 7/30 (23%) and was statistically significant (p= <0.0001). The mean log of the positives reduced by 3.8 log (from 39/cm^2 to 0.006/cm^2), this equates to a count of 75,660 cfu reducing to 12 cfu on a 1.5 kg carcass. In brief, the methodology used focused on the carcasses as they exited the

screw chiller. 30 carcasses were selected for treatment, 6 at a time were placed into clean plastic crates and completely immersed in a 600 litre solution of SANOVA® (concentration 900-1000ppm sodium chlorite and pH 2.5 – 2.6) for 20 seconds. A control of 30 birds were also collected and bagged. The concentration of sodium chlorite remained at 960 ppm before and during the trial, reducing to 949 ppm after the trial. Organoleptic assessments were favourable, despite a bleached appearance immediately after treatment, pink colouration returned within a day and taste testers unable to detect any taste or visual differences. Shelf life on the controls was 12 – 13 days and treated carcasses 14 days. The only visual difference recorded was a darkening on the wingtip extremity of one of the treated carcasses.

The next stage proposed is a commercial-scale trial to validate the treatment effect and examine shelf-life benefits.

The poultry industry in New Zealand is currently evaluating acidified sodium chlorite treatments and is carrying out pilot evaluation trials.

Tackling *Campylobacter* at the primary (and secondary) production stage should reduce the prevalence of the bacteria bought into the home and catering establishments. Further hygiene measures recommended by the ACMSF (2004) in the UK are;

- Use of meat thermometers in the catering environment,
- Industry guidance to produce consistent on-pack cooking instructions, to feature prominently,
- Unpackaged meat to be accompanied by similar instructions in some form,
- Active discouragement of washing poultry and meat, (wipe with paper towel if necessary),
- Advice on cooking, cross contamination and handwashing provided by producers/retailers of raw poultry,
- Targetted advice on cooking/hygiene practices when cooking with a barbecue,
- Packaged meats/poultry intended for barbecuing, contains the above safety advice,
- Attention drawn to carriage of *Campylobacter* in domestic pets and precautions to take,
- Further targeted education of primary/secondary school children in food safety, and
- UK FSA to consider targeted campaign on cross-contamination from raw poultry.

A useful catalogue of measures which cover all three areas has been compiled by the ACMSF (2004) in its second report to the Food Standards Agency in the UK. The report is based on the UK and selected Scandinavian countries (Denmark, Norway, Sweden). The document can be found at the following website:
http://www.food.gov.uk/multimedia/pdfs/acmsfcampyloreport.pdf. The following is a brief summary of risk management interventions in Iceland, Denmark, Ireland, Norway and Sweden.

Iceland: A major risk management study of the entire production chain for poultry in Iceland has been carried out by Icelandic scientists, the USDA Agricultural Research Service and the Canadian Food Inspection Agency. Iceland has a closed system for poultry production and consumption. Prior to 1996, only frozen poultry was available. The introduction of chilled poultry in 1996 and steadily increasing consumption was paralleled by increases in reported rates of campylobacteriosis: 1997: 13.7 per 100,000 population, 1998: 52 per 100,000, 1999

116 per 100,000. A high proportion (90%) of *Campylobacter* isolates from humans were genetically indistinguishable from those occurring in the poultry.

A dramatic reduction in cases of campylobacteriosis occurred in 2000, with notified cases reduced to 33 per 100,000. On-farm biosecurity measures and a public education campaign introduced in 2000 were partly acknowledged for the reduction together with a targetted freezing regime. This involved testing 4 week old flocks for the bacterium, and where positive flocks were identified, the carcasses from the processing lot were frozen prior to distribution. As farmers received lower prices for the frozen commodity, there was an incentive to improve on-farm biosecurity measures. The risk assessment model being created from the study is expected to benefit other countries (Stern *et al*., 2003).

Denmark; A quantitative risk model to investigate campylobacteriosis associated with poultry has been developed (Rosenquist *et al*., 2003). The model suggests that logistic slaughter (i.e. slaughtering negative flocks before positive flocks) would have only a minor effect.

Hot summers in Denmark present animal welfare issues, leading to the opening of the broiler houses for improved ventilation which undermines biosecurity. The Danish belief is that on-farm controls are difficult so the emphasis is on reduction rather than elimination of the bacterium in the flocks and interventions during/after processing. Interventions currently being investigated are heat treatment (75°C for 15 seconds) and freezing at –18°C for 10 days (ACMSF, 2004).

Danish consumers are prepared to pay a premium price for *Campylobacter*-free chicken products. Legislation in Denmark restricts this status to those flocks which are controlled, giving a 95% guarantee that less than 1% of the birds are infected. A minimum of 300 samples per flock are required to be tested, and only where all the results are negative can the products be marketed as *Campylobacter*-free.

Ireland; In September 2002, the Food Safety Authority of Ireland published a report on "Control of *Campylobacter* species in the food chain" which outlined 38 recommendations for industry and government agencies;
http://www.fsai.ie/publications/reports/campylobacter_report.pdf

Consumption and handling of poultry meats was acknowledged as a major risk factor for campylobacteriosis, but the relative importance as a cause of human disease was unclear. Risk management involved efforts throughout the production and consumption process. Controls on poultry farms and during processing were recommended, as well as for the food service industry and consumers. On the farm the primary measures were the avoidance of "thinning" (i.e. complete destocking was preferred), control of visitors, management of animal waste to prevent environmental contamination and minimisation of pre-slaughter stress.

Norway; There has been a marked increase in the number of human campylobacteriosis cases in Norway since 1997 (annual incidence around 100 per 100,000). Approximately half the infections are thought to be acquired abroad. Leading risk factors for human infection were consumption of non-disinfected water, consumption of poultry purchased raw, attending outdoor barbeques, and professional contact with animals.

The figures given for *Campylobacter* positive flocks in Norway were: 1991: 18%, 1998: 4%, 2001-2002: 7.6%.

A national plan to control *Campylobacter* in broilers has been launched with three elements;
- Surveillance of live animals/animals at slaughter/poultry meat,
- Follow up of positive farms, standardized consultations and flock biosecurity measures such as disinfection of drinking water and physical hygiene barriers in the broiler houses,
- Farm-based research to identify risk factors

Contamination of fresh poultry products ranged from 4-10% over the period 1995-1998. More recent data showed contamination on 2002 of 2%.

Sweden; In addition to some large waterborne outbreaks, chicken meat is recognised as a common source of campylobacteriosis. In 2002, 2,453 cases were reported as being acquired in Sweden. For those cases where a suspected source was identified, the most common risk factor mentioned was eating chicken meat (351 cases) and poultry contact at work or at home (46 cases).

A key factor in the Swedish broiler system is the reduction in positive flocks with a correlation with litter dryness. Data referred to in the ACMSF (2004) report suggest that *Campylobacter* spp. may be less infectious in dry as opposed to wet litter. Improvements were made when Swedish farmers began scoring the condition of their chicken's feet (i.e. for hock and pad burn). Physical damage indicated contact with poor litter quality. The scoring system was then used as a parameter for adjusting the bird densities in the sheds. Other risk management controls include changing clothing and footwear at the entrance to each house, "all in, all-out" system and the worst affected farms receiving veterinary advisor visits. In 2000, almost half of all farms had no *Campylobacter*-positive flocks.

A recent independent study undertaken by AgriQuality looking at welfare indicators such as breast blisters and hock burn, which are recognised indicators of litter quality, showed New Zealand farms to be superior to their overseas counterparts

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 poultry meat alone.

This cost estimate 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 two years, the reported rate has increased to 326.8 in 2004 and 370.3 in 2005. Consequently the estimated cost will increase. Campylobacteriosis still represents the majority of infectious intestinal disease costs.

7.3 Other Transmission Routes

7.3.1 Other transmission routes: food

There is some evidence for red meats and offal as vehicles for *Campylobacter* infection in New Zealand, although *Campylobacter* contamination occurs less frequently, and apparently at generally lower levels than for poultry. Data has been reviewed in forthcoming Risk Profiles for *Campylobacter* in red meats and in mammalian and poultry offals.. In the red meat Risk Profile, data from a national retail meat survey from July 2003 to June 2004 (Wong *et al.,* 2006) shows that *Campylobacter* contamination occurs in red meats; beef 3.5%, bobby veal 10%, lamb/mutton 6.9% and pork 9.1%. In the one laboratory confirmed outbreak, where the implicated food was a red meat (cocktail sausages), cross contamination from raw poultry to the sausages was the likely cause of contamination.

The prevalence of *Campylobacter* in poultry offal in New Zealand is high. In a study undertaken by ESR (Whyte and Hudson, 2004), 90% of retail chicken livers were contaminated internally, and 100% were contaminated externally. All of the outbreaks associated with offal consumption since 1999 have been from poultry offal. Most have involved chicken livers prepared in restaurants or cafés.

Campylobacter have also been detected however in mammalian offals intended for human consumption. Cornelius *et al.,* (2005) have reported a rate of 66.2% contamination in sheep liver in New Zealand. Hudson (1997) reported 40.9% contamination in lamb and ox liver.

The consumption of poultry and mammalian offal is low in comparison to other meat types. However the high prevalence of *Campylobacter* in raw sheep and chicken livers is of concern, especially when some advice to consumers is to cook chicken livers "until they're pink in the middle" or "lightly sautéed" (Whyte and Hudson, 2004). In addition, there may be a risk of infection through exposure due to the handling of offal for pet food and/or cross contamination from the exterior of packs of chicken meat and offal which have been shown to be frequently contaminated (Wong *et al.*, 2004).

It seems reasonable to consider offal as a minor but definite transmission route for campylobacteriosis in New Zealand.

In New Zealand *Campylobacter* has also been isolated from 11% of watercress samples (Edmonds and Hawke, 2004) and was the subject of a Director-General of Health statement in 2000.

The organism has been isolated from raw milk at a low prevalence (Hudson *et al*., 1999) and so may pose a risk to consumers of unpasteurised milk.

7.3.2   Other transmission routes: environment

Campylobacter are 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 that suggest 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 water sources were less commonly contaminated (37% positive) (Savill *et al*., 2001). The numbers of cells in roof water were very low, but the maximum numbers in river water were not established. A more recent survey of drinking water supplies, commissioned by the Ministry of Health, has shown extremely low contamination prevalence in treated drinking waters in New Zealand (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 for *Campylobacter* , 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 subtypes 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.

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 poultry products

Notified campylobacteriosis rates in New Zealand are high by world standards. A general increase in the number and rate of notified campylobacteriosis cases has occurred from 1980 to 2005. January 2003 saw the highest reported case numbers (1787) since records began in 1980.

There is evidence from case control studies and other sources to suggest that consumption of poultry plays an important role in the transmission of this infection in New Zealand. The prevalence of *Campylobacter* in uncooked poultry products at the retail level is upwards of 50%. The most recent survey suggests that 89.1% of minced/diced chicken is contaminated (See Table 1 and 2). Consumption of poultry has increased by approximately two-fold from 1985 to 2000. The prevalence of *Campylobacter* in chicken carcasses and livers at the factory level is also high indicating that cross contamination could be widespread from faecal matter to the carcass and offal during processing.

It seems possible that part of the increase in notified cases of campylobacteriosis over the period 1980 to 2005 is due to increasing consumption of poultry over the same period. However, this does not explain why the reported campylobacteriosis rate in New Zealand is markedly higher than other countries. The prevalence of *Campylobacter* in New Zealand uncooked poultry products appears similar to that in other countries. While the comparison of consumption of poultry with Australia is not clear-cut, the levels of consumption appear similar and therefore do not explain the observed difference in disease incidence.

As suggested by the study in Ashburton, there may well be differing patterns of transmission of *Campylobacter* between rural and urban populations in New Zealand. Although the overall picture of transmission of *Campylobacter* is not yet clear, the data indicate that poultry is a significant vehicle for the foodborne component.

8.1.2 Risks associated with other foods

There are data to indicate that offal foods in New Zealand have a high prevalence of *Campylobacter*, but red meats are infrequently contaminated. 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.

Potable water is consumed in large quantities by the entire population, and it is possible that a very low level of contamination could result in large numbers of cases. However, current information does not indicate even a very low level of contamination in treated supplies, which serve the majority of New Zealanders, and so drinking water can be considered a minor transmission route (Lake, 2006).

8.1.3   Quantitative risk assessment

A quantitative risk model developed at ESR is currently being reviewed by NZFSA, the model describes the poultry food chain from the entrance to processing to consumption, including both domestic and foodservice channels.

8.2   Commentary on Risk Management Options

Options for improved control of *Campylobacter* transmission in poultry include:

- Better control on farms (biosecurity, improving natural resistance), to reduce the prevalence of contamination in broiler flocks,
- Improved slaughter/processing controls,
- Further consumer/caterer food safety education campaigns.

Investigation of potential sources of infection and "on farm" control measures specifically for *Campylobacter* could reduce contamination levels of poultry products at retail, although cross contamination during processing would remain a problem. The forthcoming results of the studies in Iceland, together with the interventions put into place by the other Scandinavian countries should provide valuable indicators of effective intervention strategies. However, risk management options for the New Zealand industry will require further research to determine which measures would be most effective for *Campylobacter* control.

As stated in the section on risk management studies overseas (section 7.1.4), acidified sodium chlorite dips have been found to be effective in the USA (Oyarzabal, 2005).   Work in the New Zealand industry on this potential intervention is ongoing.

Even with improvements in *Campylobacter* control during production, 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 poultry in domestic kitchens, particularly cross contamination.

8.3   Data gaps

The data gaps identified in this Risk Profile are:

- Prevalence and numbers of *Campylobacter* on frozen chicken in New Zealand;
- Chemical intervention options; and,
- Data on farm biosecurity and flock prevalence, and broiler prevalence and levels at end of processing (national baseline studies on these issues are currently underway).

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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