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RISK PROFILE:
CAMPYLOBACTER JEJUNI/COLI
IN
MAMMALIAN AND POULTRY OFFALS

Prepared as part of a New Zealand Food Safety Authority
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by

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SUMMARY

The purpose of a Risk Profile is to provide contextual and background information relevant to a food/hazard combination so that risk managers can make decisions and, if necessary, take further action. Risk Profiles include elements of a qualitative risk assessment, as well as providing information relevant to risk management. Risk profiling may result in a range of activities e.g. immediate risk management action, a decision to conduct a quantitative risk assessment, or a programme to gather more data. Risk Profiles also provide information for ranking of food safety issues.

This Risk Profile concerns *Campylobacter* in offal (liver and kidney).

In New Zealand, the prevalence of *Campylobacter* in offal in general is high. External contamination of poultry livers in one study was 100%, while internal contamination was 90%. Sheep liver has a contamination prevalence of approximately 38.9% to 66.9%. Bovine and porcine offals appear to be less commonly contaminated (<10%).

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". 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 offal which have been shown to frequently contaminated.

Seventeen small outbreaks of campylobacteriosis associated with poultry liver consumption were reported to the national notifiable diseases surveillance system from 1997 to August 2004. Most of the outbreaks involve chicken livers prepared in restaurants/cafés. Mammalian offal and products such as liver pâté have not been implicated in any outbreaks. The association with offal is based entirely on epidemiology (common exposure by cases); there were no laboratory confirmations where the organism was detected in the food consumed.

An elevated odds ratio for poultry liver consumption was found in the large national campylobacteriosis case control study, but not in smaller more localised case control studies. Liver pâté consumption was found to be a lower risk in these studies. The cooking methods used to prepare pâté have been the subject of a separate study, which found no *Campylobacter* survival, provided the cooking is performed correctly. However, liver pâté cannot be dismissed as a potential source because an outbreak has been associated with the consumption of pâté from undercooked poultry livers in New Zealand.

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

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

The data gaps identified in this Risk Profile are:

- Information on the domestic abattoir handling of offals – particularly cooling methods;
- Data on the sero/genotypes of *Campylobacter* present in offal samples, particularly from poultry livers;
- Data on the contamination of mammalian offal packs; and,
- Information on domestic handling of livers and kidneys, including cooking practices.

1 INTRODUCTION

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

Figure 1:   Risk Management Framework
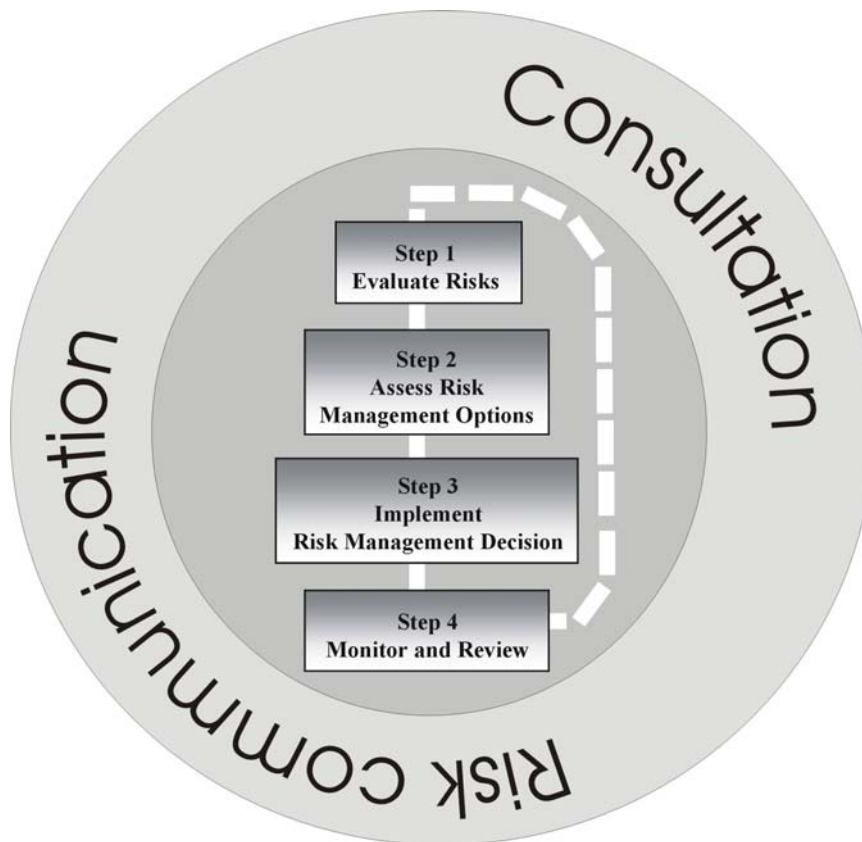
Figure reproduced from "Food Administration in New Zealand. A risk management framework for food safety" (Ministry of Health/Ministry of Agriculture and Forestry, 2000).

In more detail, the four step process is:

1. Risk evaluation

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

2. Risk management option assessment

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

3. Implementation of the risk management decision

4. Monitoring and review.

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

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

This Risk Profile concerns the bacterial species *Campylobacter jejuni and Campylobacter coli* in offal of mammals and poultry. This food/hazard combination was chosen for preparation of a detailed Risk Profile on the basis of food surveys indicating a high prevalence of *Campylobacter* in both mammalian and poultry offals, although almost all data are for livers. The rate of notified cases of campylobacteriosis in New Zealand is high by international standards, and the importance of various potential transmission routes needs to be investigated.

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

Hazard identification, including:

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

Hazard characterisation, including:

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

Exposure assessment, including:

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

Risk characterisation:

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

Risk management information:

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

Conclusions and recommendations for further action

2   HAZARD IDENTIFICATION: THE ORGANISM

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

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

2.1   *Campylobacter*

2.1.1   The organism/toxin

Campylobacter cells are slender, spirally curved rods which are non-sporulating and Gram negative. There are many species of *Campylobacter* but the evidence in New Zealand suggests that only two, *C. jejuni* and *C. coli,* are of significance to public health. Other species, such as *C. upsaliensis, C. fetus, C. hyointestinalis* and *C. lari* have occasionally been reported as causing human illness (AIFST, 2003) but their significance in New Zealand is unknown.

For the sake of simplicity, in this profile, the term *Campylobacter* will refer specifically to the two pathogenic species *C. jejuni* subsp. *jejuni* and *C. coli*. *Campylobacter* spp. will be used to describe other species.

2.1.2   Growth and survival

Growth:

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

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

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

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

Survival:

Campylobacter are sensitive to air, drying and heat.

Temperature: Survival in food is better under refrigeration than at room temperature, up to 15 times as long at 2^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 (AIFST, 2003), although the reduction can vary with the type of food and storage temperature. Freezing therefore does not instantly inactivate the organism in food.

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

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

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

2.1.3   Inactivation (Critical Control Points and Hurdles)

Note that in microbiological terms "D" refers to a 90% (or decimal or 1 log_{10} cycle) reduction in the number of organisms.

Temperature: Rapidly inactivated on the surface of meat by heating at $55 – 60^o$C 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.*

Because offal can be internally contaminated, the NZFSA recommend cooking of liver to an internal temperature of at least 70^oC for 2 to 3 minutes to ensure the elimination of the bacterium, see section 5.1.3.

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

Water activity: Sensitive to even slightly reduced water activity, but under certain refrigeration conditions can remain viable for several weeks (ICMSF, 1996). The drying of surface tissues during air-chilling of red meat carcasses is important in reducing *Campylobacter* prevalence (for example, from 9% before chilling to 0% after chilling on pig carcasses (Oosterom *et al.,* 1985). The prevalence of *Campylobacter* has been found to be significantly lower on air-chilled broilers compared to immersion-chilled broilers (39.3% and

48.7% respectively), although the prevalence at entry to processing was not determined (Sánchez *et al.*, 2002). However, a review of survival by *Campylobacter jejuni* (Murphy *et al.*, 2006) indicated that drying of poultry carcasses would not have the same effect as drying of red meat carcasses, due to a generally shorter cooling period, and the texture of the poultry skin providing cavities which act as niches for survival.

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

Disinfectants: When poultry are cooled in chlorinated water, the numbers of *C. jejuni* on carcasses, livers and gizzards are usually reduced, although it is not clear whether the reduction is due to the chlorine or physical removal.

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

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

2.1.4 Sources

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

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

Environment: Water and soil can be easily contaminated from infected animals' excreta. Environmental survival is considered to be poor, but new information suggests it may be better than currently acknowledged. For example, *Campylobacter* has been detected in dry beach sand. Survival in cold water is good, but reduced at temperatures above 10°C. *Campylobacter* is present in water and sediments more frequently and at higher numbers in the winter months. These data are of interest because environmental survival appears to be

opposite to the trend in the numbers of human cases, i.e. survival is poorer in the warmer months, when the numbers of human infections are highest. From samples taken in New Zealand, 60% of recreational waters (i.e. river waters), 75% of shallow ground waters, 37.5% of roof waters and 29.2% of reticulated drinking waters have been shown to be contaminated by *Campylobacter*. The concentration of *Campylobacter* was low in the drinking waters, up to 0.6 MPN 100ml^{-1}, and most isolates were *C. lari* (Savill *et al.*, 2001). A more recent survey of New Zealand treated drinking water found negligible prevalence of *Campylobacter* (Nokes *et al.*, 2004).

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

2.2   *Campylobacter* Typing

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

Campylobacter genome which has been a problem for the development of a universal typing system (Tam, 2001).

Recent technology has enabled restriction enzyme digestion and pulsed field gel electrophoresis (PFGE) to be used for genotyping (Gibson *et al.,* 1994). However the enzymes used and the conditions under which the gel electrophoresis is undertaken can have a marked influence on the end result. The success of PulseNet USA, and increasing recognition of the international nature of infectious disease, has prompted Canada, European countries, South America and the Asia-Pacific region, including Australia, to attempt to establish similar and compatible networks in each region.

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

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

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

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

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

future studies, outbreak investigations and international comparisons through New Zealand's participation in the development of the regional group 'Pulsenet Asia Pacific' and beyond (Pulsenet Europe, Pulsenet USA etc).

Lastly, in accordance with European initiatives New Zealand is currently investigating the utility of multi-locus sequence typing (MLST), the next generation of typing technologies, as a more robust method for typing genetically unstable *Campylobacter*. MLST is gaining currency as the typing method of choice for *Campylobacter* due to the ease of assignment of sequence types and the direct comparability of data from isolates obtained worldwide. ESR has established a routine procedure for the identification of *Campylobacter* MLST sequence types. A selection of *Campylobacter* isolates currently detailed on the PulseNet Aotearoa database is being analysed. The sequence types identified will be deposited into the database. A central repository of alleles, which 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: Mammalian and Poultry Offals

'Mammalian' refers to the major stock animals: cattle, sheep, pigs, deer and goats. 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. Domestic fowl only will be covered.

Numbers of livestock farmed in New Zealand are discussed in more detail in Section 3.2.

Mammalian offal is defined by Food Standards Australia New Zealand (FSANZ) under Standard 2.2.1 as "those parts of the carcass such as blood, brain, heart, kidney, liver, pancreas, spleen, thymus, tongue and tripe, but excludes meat flesh, bone and bone marrow".

The New Zealand Meat Industry Standard for 'Slaughter and Dressing' of mammals (at the following website), highlights specific offal controls;
Number 5; http://www.nzfsa.govt.nz/animalproducts/meat/meatman/is5/is5.pdf#page25 and
Number 6; http://www.nzfsa.govt.nz/animalproducts/meat/meatman/is6/index.htm.

For the purposes of this Risk Profile, mammalian offal will be considered to be the liver and kidneys as these are the parts most likely to be used for human consumption in New Zealand. Liver and kidney from cattle (often described as "ox"), sheep and pigs can commonly be found on retail sale for human consumption. These offals can also be found processed into products such as liver pâté, liver sausage, steak and kidney pies.

Because of the relatively small size of other organs, poultry offal used for human consumption in New Zealand is generally restricted to the liver from broilers. The other constituents of offal, namely the giblets and the kidneys will not be covered. Poultry livers can be cooked in the form they were purchased or processed as in chicken/duck liver pâté. Liver pastes will not be covered by this Risk Profile.

3.1.1 *Campylobacter* and mammalian liver

Storage trials (Moore and Madden, 2001) of *Campylobacter* on porcine liver found that in general, the offal presented a high nutrient, moist environment which allowed for survival of *Campylobacter* under refrigerated storage conditions but not when incubated at 37°C. For example, *C. coli* survived for 4 days with no reduction in numbers at 4 and 15°C on sterile liver slices. In sterile liver homogenate, the organism reduced in numbers by <1 $\log_{10}$ over 10 days storage at 4°C, but by approximately 3 $\log_{10}$ when stored at 15°C. In contrast, numbers reduced rapidly to non-detectable under the latter conditions when incubated at 37°C. *Campylobacter coli* were sensitive to freezing on porcine liver slices at -18°C. The authors also discovered that the cells survived better on chilled liver slices and in autoclaved liver homogenates than in raw liver homogenates at all temperatures, indicating the presence of a heat-labile antagonistic agent in raw liver homogenates.

Similar survival during storage has been shown in minced beef liver at 4°C with a slight decrease in numbers after 6 days of storage (Hänninen, 1981).

There is currently no information available regarding the survival and growth of *Campylobacter* in ovine or cervine liver.

3.1.2 *Campylobacter* and poultry liver

A literature search did not yield any references relating to the behaviour of *Campylobacter* in poultry liver (at a range of temperatures).

3.2 The Food Supply in New Zealand

The Food Balance Sheets for New Zealand (http://apps.fao.org) (data base accessed October 2006) show that in 2004, 199,570 metric tonnes of edible offals were produced domestically. Of these, 64,380 tonnes were exported leaving a balance of 135,190 tonnes. The majority of this amount, 82,930 tonnes, went into feed (predominantly pet food) while 40,450 tonnes went into other uses. The balance of 12,050 metric tonnes was used for human consumption. It should be noted that this figure is for all edible offals, therefore it will be a mixture of poultry and red meat offals. One of the major poultry producers has indicated that approximately 1% of their offal production is destined for human consumption, see Section 3.2.2

3.2.1 Farmed mammalian livestock

In the year to June 2004, there were 39,700,000 sheep, 4,660,000 beef cattle and 355,000 pigs in New Zealand. Carcasses produced in the same year were 114,000 mutton, 434,000 lambs, 592,000 beef, 25,000 veal and 51,300 pork (Situation and Outlook New Zealand Agriculture and Forestry website: http://www.maf.govt.nz/mafnet/rural-nz/statistics-and-forecasts/sonzaf/2004/04-update/httoc.htm). Only a proportion of these carcasses will also be used to obtain offal destined for human consumption, but no detailed information has been located. It is likely that a significant percentage of mammalian offal will be channeled into rendering and pet food.

New Zealand has a relatively small pig industry, which focuses on the domestic market. Currently about 48,400 breeding sows are farmed, with an estimated 350,700 pigs on farms at any one time (New Zealand Pork Industry Board, 2001). Since 1995 pig meat production has been relatively static averaging 49,000 tonnes per year (47,200 tonnes carcass weight in the 2002/2003 season) (see:http://www.pork.co.nz/profile.asp).

3.2.2 Poultry industry

The Poultry Industry Association of New Zealand Incorporated (PIANZ) represents the interests of the majority of 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 98% of poultry production.

Just under 89 million broiler chickens were processed in the year ending December 2005 (Vanessa Wintle, PIANZ, personal communication, November 2006). However, there are few data on the amount of offals from these birds used for human consumption. Information supplied by Inghams indicates that they do not sell giblets (heart, liver, gizzard and neck)

inside carcasses, and 99% of their poultry offal is used as pet food, with only 1% retained for human consumption (Brian Jones, Inghams, personal communication August 2004).

3.2.3 Imported food

There is very little information available on the import of offals covered by this Risk Profile. Table 1 summarises the small amounts of imports of pig kidneys and liver products into New Zealand during the year to March 2003. It is not clear whether, despite being labeled as edible, the products listed are intended for human consumption. The only imported poultry offal is pâté de fois gras which is a cooked product.

Table 1:   Imports of pigs kidneys and various liver products for the year ending March 2003

Food item	Country of Origin	Weight (tonnes)
Pig kidneys	Australia	17.3
Pâté de foie gras	France, United Kingdom	1.8
Liver preparations – animal not specified	Croatia, France, Germany, Italy, Netherlands, Norway, Philippines	5.4

A significant number of other references in the customs data obtained from Statistics New Zealand use the descriptor 'meat or meat offals' and do not allow determination of the amounts of offal involved. The descriptors for the Australian offal/products are 'fresh, chilled' or 'frozen', indicating that they are not cooked.

3.2.4 Preslaughter and processing

Control of *Campylobacter* begins with the livestock at the farm level. The organism can be present in the general environment in which the farmed animal is raised (streams, rivers, wild birds, pastures, other livestock). There are no effective vaccines for *Campylobacter* in the animal species covered in this Risk Profile. There are two commercial sheep vaccines available but they do not offer protection against *C. jejuni* or *C. coli* (see Section 7.1.1.1 and 7.1.1.2).

3.2.4.1 Mammalian livestock preslaughter

Because the main site of *Campylobacter* in the live animal is in the intestines, the overall cleanliness of the animal at time of slaughter can be a factor in contamination of the external liver and kidney organs. Cleanliness and therefore microbial contamination is affected by climate and location, whether the animals originate from pastures or feed lots, the hygiene practices of the farmer, the transport used and finally the lairage conditions and slaughter house procedures.

As live animals can be asymptomatic carriers of the *Campylobacter* organism, they can not be detected at ante-mortem inspections and the organs do not show clinical evidence at the post-mortem inspection in the abattoir. Unlike other meats, where contamination is regarded as being restricted to muscle surfaces, *Campylobacter* can be present in the internal tissue of livers. This may be due to the link between the liver and the gall bladder via the bile duct (Garcia *et al.,* 1985; Bryner *et al.,* 1972, cited in ICMSF, 1998). *Campylobacter fetus* has

been isolated from the bile/gall bladders and bile ducts of 58% of clinically healthy slaughter pigs (Rosef, 1981).

3.2.4.2 Mammalian offal processing

For mammalian offal, the sequence of animal slaughter and processing is generally as follows;

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

The main source of information on processing for mammals and poultry appears in the NZFSA's website; http://www.nzfsa.govt.nz/animalproducts/meat/meatman/. Industry Standard /Industry Agreed Standard 5 Slaughter and Dressing contains specifications for the slaughter and dressing of the main mammalian food animals. Standard 6 covers the processing of edible product. Manual 16 contains the requirements for post-mortem inspection.

In New Zealand, a key difference between the procedures in which different types of offal are handled during processing is that chilling of the larger mammalian offals is often carried out in enclosed cardboard cartons, with an inner plastic liner whereas poultry offals are immersed in water. The cartons are approximately 160mm in depth, typically holding 20kg of product. Once full, the cartons are placed onto shelved racking which allows air circulation around carton surfaces. The cartons are then placed into blast freezers, with high air velocity and temperatures below –20°C (Neil Smith, personal communication, MIA, January 2007).

The temperature of livers initially rise due to metabolic processes, this peaks within half an hour and begins to fall. The New Zealand Standard requires offal to be refrigerated within 60 minutes of leaving the slaughterfloor. Cooling curves must be validated to the NZFSA's satisfaction, primarily to ensure that the rate of cooling controls the growth of *E. coli* (mesophilic indicator). Section 4 of the Industry Standard 6 specifically outlines the criteria for offal cooling (Neil Smith, personal communication, MIA, January 2007).

Surface drying does not appear to occur as the cartons are fully enclosed during the chilling process. Surface drying has been recognized as an effective step in reducing *Campylobacter* numbers, as has been observed on the surfaces of bovine/ovine/porcine carcasses during chilling and storage (ICMSF, 1998). The lack of surface drying of offals has been cited as an important factor in the incidence of *C. jejuni* or *C. coli* on retail offals (ICMSF, 1998).

3.2.4.3 Poultry livestock preslaughter

In a USDA study (Cox *et al*., 2006), broiler breeder hens were acquired at early, middle and late lay cycles (22 – 66 weeks of age). The birds were killed, defeathered and aseptically opened. The thymus (primary lymphoid organ), spleen (secondary lymphoid organ) and liver/gall bladder were aseptically removed prior to removal of ceca, in order to reduce cross contamination. Examination of these organs found *Campylobacter* in 11/43 thymii, 8/43 spleens, 4/43 liver/gallbladders and 30/43 ceca. Of the four positives in the liver/gallbladder, one was *C. jejuni* and three were *C. coli*. Of the 53 isolates, 28 were *C. coli* and 25 were *C. jejuni*. The four positives from the 43 livers/gallbladders tested all originated from birds 66 weeks of age.

Contamination of the liver may also be derived from faecal material during slaughter and dressing, or from the intestinal contents when the bird is alive. Contamination of the poultry liver is not confined to the surface; internal contamination has also been detected (Whyte and Hudson, 2004). Again it is thought that the internal contamination may be due to the link between the liver and gall bladder via the bile duct. In a Czechoslovakian study of slaughtered chickens, *Campylobacter jejuni* was isolated from the bile of 18.6% of birds (Matasovska *et al*., 1992).

3.2.4.4 Poultry offal processing

In the Guidance and Generic Risk Management Programme for the slaughter and dressing of broilers, a process flow diagram for edible offal is depicted; see: http://www.nzfsa.govt.nz/animalproducts/publications/consultation/rmp-draft-broilers.pdf; Product module 2.5, pages 23-30.

In summary, where the offal is destined for human consumption, the process begins with the evisceration step, followed by;

- Separation of liver/heart and gizzard from the other viscera,
- Peeling of the gizzard,
- Washing or immersion chilling in water containing a bactericidal agent (usually chlorine),
- Weighing and packing, (plastic pottle, bag or liner, with cardboard carton outer),
- Labelling,
- Chilling or freezing
- Storage and dispatch.

Once the edible offal (liver, heart and peeled gizzard) have been separated from the rest of the offal, the step of washing or immersion chilling of the edible offal is classed as a critical control point (CCP) because the offal is "likely to be contaminated with unacceptable levels of microorganisms" and "effective chilling and use of permitted bactericidal agent (e.g. chlorine) can reduce overall microbiological counts". Washing without a permitted bactericidal agent is cautioned against as reducing the microbial loading and minimising cross-contamination may not be achieved (p2:27).

In poultry, muscle tissues have a longer shelf-life than giblets, although an early study (Taylor *et al.,* 1968) comparing shelf life of poultry packed with or without giblets found no

difference. The ICMSF states that "giblets placed into the cavities of poultry carcasses can spoil more rapidly and cause product rejection. This can be avoided by controlling the level of contamination, the rate of chilling, and not holding giblets over to a later date" (ICMSF, 1998: p99). In addition, the giblets could be left out of the carcass altogether, a practice which is now becoming popular (see section 3.2.2).

Where the poultry livers are sent for further processing, chicken liver pâté is one of the most common derivatives. Manufactured pâté is normally produced by baking a blended processed mixture of liver with oil/butter, herbs, spices and other required flavours. It undergoes heat treatment which should be sufficient to destroy the *Campylobacter* organism.

4   HAZARD CHARACTERISATION: ADVERSE HEALTH EFFECTS

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

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

4.1   Symptoms

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

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

Condition: Campylobacteriosis.

Toxins: No toxins are produced in foods.

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

Long Term Effects: Campylobacteriosis is a recognised cause of chronic sequelae in the form of Guillain-Barré syndrome (GBS).   The frequency of GBS resulting from campylobacteriosis has been estimated as 0.1% (Altekruse *et al.*, 1999) and this 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 common single identifiable pathogen in the syndrome (Rees *et al.*, 1995). The authors also found that GBS was more likely to develop in men than in women, which suggests either a sex-linked predisposition or more males contracting *C. jejuni* infection in the first instance. The conclusion was that infection with *C. jejuni* precedes Guillain-Barré syndrome and is associated with axonal (peripheral nerve) degeneration, slow recovery, and severe residual disability.

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

et al., 1999).

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

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

4.2   Types Causing Disease

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

Certain serotypes of *C. jejuni,* particularly Penner Serotypes O:19 and O:41, are more frequently associated with GBS (AIFST, 2003). Penner Serotype O:19 has been associated with GBS in Japanese studies, but this was not confirmed in a USA case control study, in which no specific serotypes were associated with GBS (Rees *et al.*, 1995). See Section 6.1.5 for serotypes causing human disease in New Zealand.

4.3   Dose Response

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

Data from experimental studies where volunteers ingested known numbers of *Campylobacter* cells have been investigated for the purpose of modelling the dose-response relationship (Medema *et al.*, 1996; Teunis *et al.*, 1999). Infection, where the microorganism is reproducing in the body, was modelled separately from illness, which is less frequent. The probability of infection increased from approximately 50% at 800 cells to approximately 100% at 1×10^8 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 that there are an optimum number of cells consumed for sickness to occur. This limited study is the only evidence known to suggest this effect and so should be treated with caution.

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

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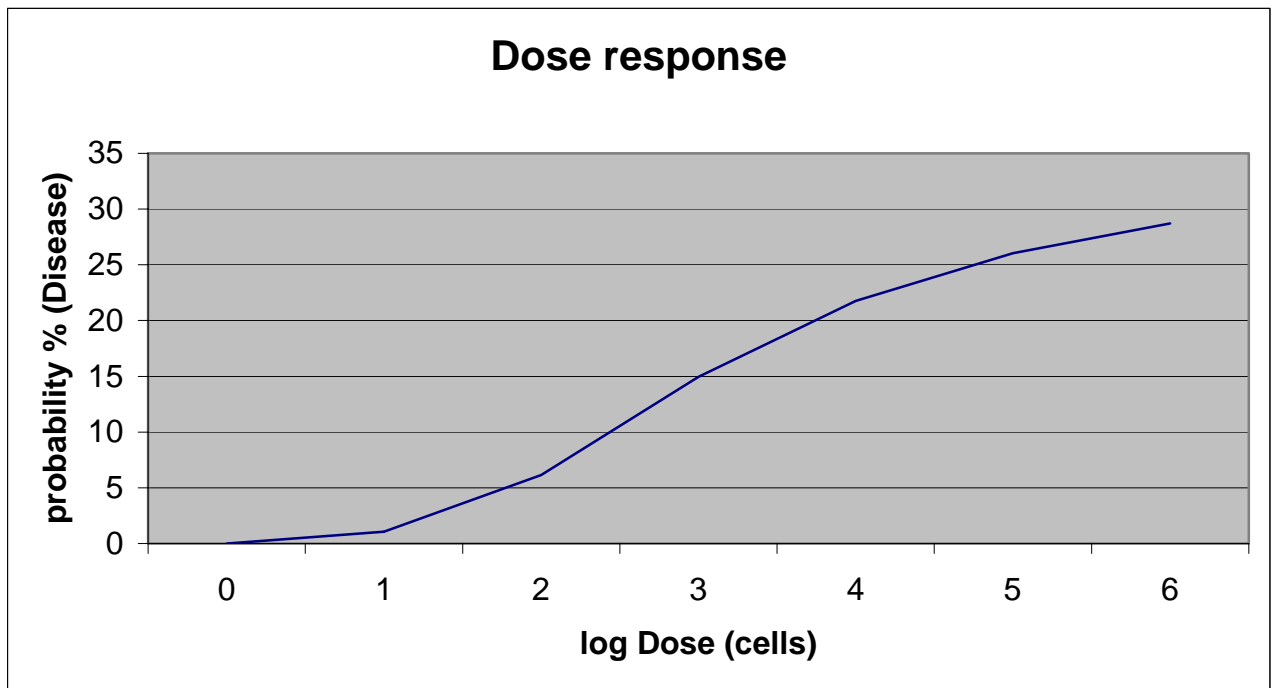
5 EXPOSURE ASSESSMENT

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

Campylobacter contamination is common in and on raw offals in New Zealand, as demonstrated by the information given below. There is less information available on cooked offals.

5.1.1 *Campylobacter* in raw poultry livers

Information (derived from a single study) on the prevalence of *Campylobacter* contamination of raw poultry livers in New Zealand is shown in Table 2.

Table 2: **Prevalence of *Campylobacter* in New Zealand raw chicken livers determined by Whyte and Hudson (2004)**

Sample type (Offal)	Prevalence (%)	Reference
Chicken liver (external tissue)	100	Whyte and Hudson, 2004
Chicken liver (internal tissue)	90	Whyte and Hudson, 2004

The survey of retail chicken livers (Whyte and Hudson, 2004) also enumerated *Campylobacter* on the external surface and within the tissue of chicken livers. Of the 30 retail chicken livers tested, all were positive for *Campylobacter* on the external surface, and 27/30 (90%) were contaminated internally. Total counts per liver (internal and external combined) are shown in Figure 3, grouped by order of magnitude. The mode was between 10^2 and 10^3 per liver, and one liver contained $>10^5$ *Campylobacter*. The majority (168 of 171) of the isolates were identified as *C. jejuni* (remaining three were *C. coli*). These results correlate well with the data from overseas. On a per gram basis most (60%) of the livers contained less than 10 MPN *Campylobacter*/g, although the maximum number recorded was $>2.5 \times 10^3$ MPN/g.

Figure 3: **Distribution of counts for *Campylobacter* in 30 retail chicken livers (internal and external counts combined)**
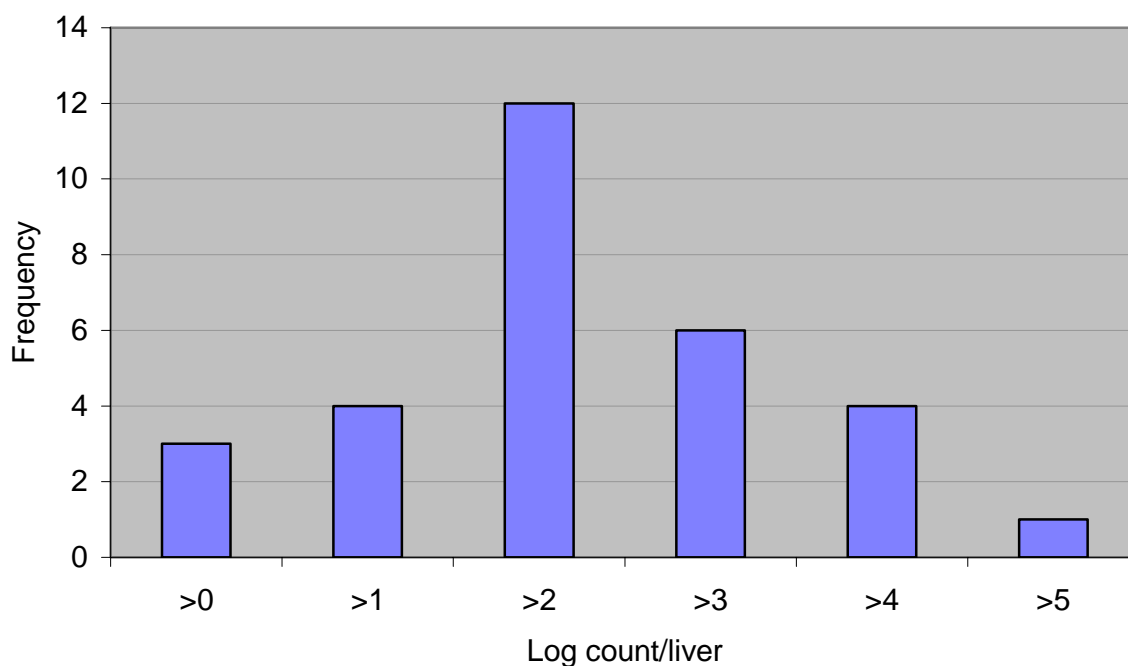
In early 2002, Wong *et al*. (2004) conducted a survey of the exterior of three hundred retail packs of fresh chilled chicken products from fifteen supermarkets in the Christchurch area. Out of the 300 packs, 50 were offals (25 livers and 25 hearts).

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 5.4.1). Out of the 300 packs, 72 (24%) were externally contaminated with *C. jejuni*. Offal samples had the highest rate of external contamination (52%), comprising: liver packs14/25 (56%), heart packs 12/25 (48%). Contamination of other sample types were: whole chickens 17/50 (34%) and chicken portions 29/200 (14.5%).

Of the 14 externally contaminated liver packages:

> ➤ 2 had counts <6 MPN/pack,
> ➤ 7 packages were between 6 and 480 MPN/pack,
> ➤ 3 were between 860 and 1860 MPN/pack,
> ➤ 2 packages had counts >2200 MPN/pack.

These observations suggest that the exterior of chicken liver packs may be a significant source of exposure, although the contribution of this contamination pathway to foodborne illness can only be properly determined by development of a validated risk assessment model.

5.1.2   *Campylobacter* in mammalian offals

Three studies give an overview of the prevalence of *Campylobacter* contamination of mammalian offals in New Zealand. Most reports concern liver samples; there are fewer data on contamination of kidneys. The standard method of taking a 10g sample which would include external and internal tissue means that it is not possible to determine whether the contamination was external or internal. Data from these studies are summarised in Table 3.

Table 3:   A summary of the prevalence of *Campylobacter* in New Zealand retail raw mammalian livers and kidneys

Sample type (Offal)	Prevalence (%)	Reference
Lamb/sheep liver	5/6 (83.3%)	Hudson, 1997
Lamb/sheep kidney	2/5 (40%)	
Ox kidney	0/2 (0%)	
Ox liver	0/2 (0%)	
Ox liver (*C. jejuni*)	15/178(9.0)	Devane *et al.,* 2005
Ox liver (*C. coli*)	1/178 (0.6)	Devane *et al.,* 2005
Sheep liver (*C. jejuni*)	63/162 (38.9)	Devane *et al.,* 2005
Sheep liver (*C. coli*)	6/162 (3.7)	Devane *et al.,* 2005
Pig liver (*C. jejuni*)	9/187 (4.8)	Devane *et al.,* 2005
Pig liver (*C. coli*)	9/187 (4.8)	Devane *et al.,* 2005
Sheep liver	180/272 (66.2)	Cornelius *et al.*, 2005

In the research carried out by Hudson (1997), the number of samples tested were relatively small and the tests were presence/absence only with no enumeration.

The retail ox, sheep, and pig liver results (Devane *et al.*, 2005) were obtained as part of the *Campylobacter* Transmission Routes (CTR) study conducted in the Ashburton region (Baker *et al.*, 2002).

These data suggest that sheep livers are more likely to be contaminated with *C. jejuni* than livers from cattle or pigs.

More recent work enumerating *Campylobacter* in fresh sheep livers has been carried out by ESR (Cornelius *et al.*, 2005) for the NZFSA. Isolates were also obtained concurrently from faeces of human clinical cases of campylobacteriosis from the same geographical area (Christchurch). Of the 272 liver samples collected from butchers and supermarkets during spring and autumn 2002-2003, 180 (66.2%) contained *Campylobacter*. The mean count obtained was 19.3 MPN/g. Twelve of 272 samples (4.4%) exceeded 100 MPN/g. Analysis of the data by retailer, type and season found no significant differences, although there was a difference in the diversity of serotypes when the seasons were compared. *C. jejuni* was the most frequently detected species; where *C. coli* was detected, it was always in the presence of *C. jejuni*.

A total of 212 isolates from both human cases and sheep liver isolates were serotyped and 22 serotypes were identified in total (although 11 or 5.2% were untypable). The six most frequently isolated serotypes from either human or sheep liver samples were;

- 4 complex (20.3%),
- 2 (17.9%),
- 1,44 (12.3%),
- 8 complex (9.9%),
- 23,36 (9.0%), and
- 5 (7.1%).

When the data were seasonally adjusted, 52.2% of sheep liver isolates were also isolated from human cases in the same season. However, this serotyping data must be treated with caution, as the serotypes listed above are common in a variety of matrices.

A search of the PulseNet New Zealand database was carried out for human and offal serotypes (Dr Brent Gilpin, ESR, pers. comm, November 2004). As of November 2004 91 different serogenotypes (291 actual isolates) of *Campylobacter* from human cases had been recorded on the database. A substantial number of serogenotypes from sheep offal have also been identified: 32 serogenotypes from 98 isolates. Of these 21 serogenotypes (82 isolates) also occurred amongst the types from human cases. To date only small numbers (<10) of isolates and serogenotypes from beef and pork offal have been analysed by PulseNet, and none from poultry offal. Therefore at this stage conclusions about commonality between offal sources and human cases cannot be drawn.

5.1.3   *Campylobacter* in cooked poultry and mammalian offals

Foods prepared from undercooked chicken livers (including pâté) have been associated with three outbreaks of campylobacteriosis in Christchurch and one outbreak in Auckland (Whyte *et al.*, 2001). In one of these outbreaks involving 12 cases in Christchurch during December 2000, one of the findings was that chefs had been trained to cook poultry livers (used to prepare pâté) until they were "pink in the middle". This mild heat treatment coupled with the lack of awareness by the chefs of possible contamination of the livers with *Campylobacter* led to a study by ESR (Whyte and Hudson, 2004). The experimental work was carried out in three parts; the first part regarding external and internal liver tissue has already been discussed in section 5.1.1.

The second part looked at the effectiveness of chicken liver pâté cooking methods tested, and the third part examined the effect of heat and time on the destruction of *Campylobacter* in/on chicken liver. A factsheet based on the work has also been developed and can be found at the following website; http://www.nzfsa.govt.nz/consumers/food-safety/safe-cooking-of-chicken-livers/index.htm.

In the second experimental phase, raw chicken livers tested prior to pâté preparation had counts ranging from 23 to 240 MPN/g. The three cooking methods used to prepare chicken liver pâté were: sautéed rare followed by homogenisation, sautéed to > 74ºC and then homogenised, and homogenised and then baked in a bain marie. No *Campylobacter* were detected in any of the pâté samples. However, these experiments demonstrated that the internal tissues of cooked livers could remain pink not only when sautéed rare, but also when cooked to temperatures greater than 74ºC.

In the third experimental phase, time-temperature relationships for cooking to eliminate *Campylobacter* was investigated. The core temperatures of livers did not increase

significantly until after two and a half minutes and the inactivation of *Campylobacter* did not occur until five minutes cooking time. A well-done state was reached after six minutes of cooking. The experiment found that internal liver tissue can remain pink even after four minutes of cooking despite the core temperature reaching the mid 70ºC range. The fact sheet prepared from these experiments recommends sautéing chicken livers for at least 5 minutes or until an internal temperature of >70ºC has been reached and maintaining that temperature for 2 – 3 minutes. This enables destruction of *Campylobacter*, while retaining organoleptic qualities. Figure 4 shows the effect of temperature and time.

Figure 4: **The effect of temperature and time on the survival of *Campylobacter* in chicken livers**
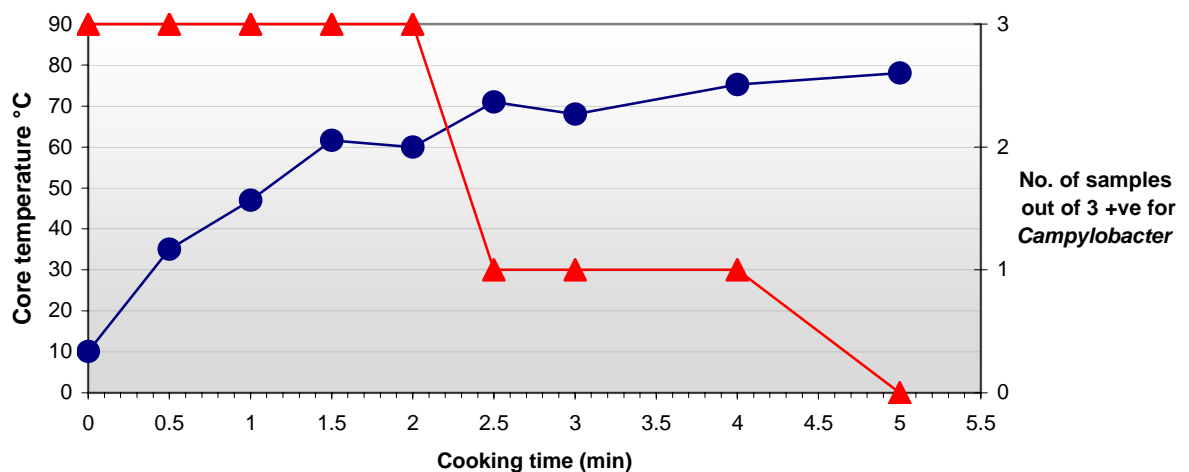
(source: Whyte and Hudson, 2004).

There are no New Zealand data available on the prevalence of *Campylobacter* in cooked mammalian offal or products such as liver pâté. Reported outbreaks of campylobacteriosis linked to offal have all concerned poultry liver products (see Section 6.1.3). However, it should be noted that poultry offal is more likely to be used in the catering industry than mammalian offal. This would increase the chance of an outbreak of campylobacteriosis being detected and reported, compared with mammalian offal, which is more often consumed at home.

5.1.4 Conclusions

The data from the above studies indicate that *Campylobacter* is a frequent contaminant of both raw poultry (up to 100%) and sheep livers (approximately 38.9% - 66.9%), with contamination of pig and ox offals less prevalent (0-10%). The numbers of *Campylobacter* obtained were similar in chicken (majority <10 MPN *Campylobacter*/g: Whyte & Hudson, in 2004) and lamb livers: (mean 19.3MPN/g: Cornelius *et al.,* 2005).

Although some serotyping and PFGE typing data are beginning to be accumulated, comparisons with human cases are not possible at this stage as not all offal types are represented. In addition, the serotypes found in offal are common in other foods.

There are few data available in New Zealand on the microbial quality of cooked offal products. However, experiments on pâté preparation methods suggest that contamination prevalence may be lower than that for uncooked products.

5.2 Food Consumption: Mammalian and Poultry Offals

Food Balance Sheet (for the year 2000) information maintained in the FAO Statistical Databases (http://apps.fao.org/) give a per capita supply of offals for New Zealanders of 4.8 kg/person/year or 13.2 g/person/day. The WHO GEMS/Food European Regional Diet (see http://www.who.int/fsf/GEMS/index.htm) gives a daily consumption for offals, mammalian and poultry, of 12.7 g/person/day. This 'European' estimate is likely to be higher than actually consumed in New Zealand, as some European countries are known to be heavy consumers of offals. Reference to Food Balance Sheet data gives per capita supply figures for offals in some European countries as high as 17.3 kg/person/year.

Estimates of offal consumption in New Zealand are significantly lower than the supply figures. A FSANZ analysis of the data from the 1997 National Nutrition Survey, using a set of standard recipes, gave a mean consumption of offals for all respondents of 2.0 g/person/day, with 95% of this being mammalian offals (ANZFA, 2001). Major contributors are cattle kidneys (0.5 g/person/day) and sheep liver (0.4 g/person/day). It is uncertain why there is such a large disparity between the apparent supply and consumption of offals in New Zealand. One explanation may be the feeding of offal to domestic pets (Bolton *et al.,* 1985).

According to the ANZFA/FSANZ analysis of the New Zealand NNS 4.2% of respondents reported consuming mammalian offals, while 0.5% reported consumption of poultry offals. These figures will include pâté.

The 1995 Australian National Nutrition Survey gave a slightly lower estimate for consumption of 'organ meats and offal, products and dishes' of 1.2 g/person/day for the Australian population aged 19 years and over (Australian Bureau of Statistics, 1999). Estimates for the US population are slightly higher at 2.9 g/person/day for a 70 kg adult (EPA, 1997). About 13% of this consumption is ascribed to poultry offals.

For the UK it has been reported that the average consumer of offals consumes 17.5 g/consumer/day and that 28.6% of the population consume offals on any given day. This equates to a population level average consumption of 5.0 g/person/day. The same report gives a daily consumption for a 97.5[th] percentile consumer of 61g/person/day (http://archive.food.gov.uk/maff/archive/food/infsheet/1998/no160/tables.htm).

5.3   Qualitative Estimate of Exposure

5.3.1   Number servings and serving sizes

The estimation of total number of servings of offals (mammalian and poultry) 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, and
- That the consumption of offals by the population less than 15 years of age will not be significantly different to that for the survey population (The NNS only surveyed people 15 years and older). This assumption is questionable, but information for New Zealanders less than 15 years is currently unavailable.

From the NNS, 216 respondents were identified as consuming mammalian or poultry offals. It will be assumed that this number is likely to be a good approximation to the total number of servings, as it is unlikely that respondents will be consuming more than one serving of offals per day. Using a total survey population of 4636 and a total New Zealand population of 4,054,200 (at 31 March 2004) (http://www.stats.govt.nz/):

Annual number of servings (total population)   = 216 x 4,054,200/4636 x 365
                                                 = 6.9×10^7 servings

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

Percentile	Serving size (g)
50	25
75	39
95	135
99	420

5.3.2   Frequency of contamination

All the available data indicate that *Campylobacter* is frequently present in raw mammalian and poultry offal, particularly sheep/lamb and chicken livers.

5.3.3   Predicted contamination level at retail

Data for chicken and sheep/lamb raw livers indicate *Campylobacter* numbers approximating 10^2-10^3 MPN/liver, or (approximately 20-240 MPN/g) (Section 5.1.1 and 5.1.2).

5.3.4   Growth rate during storage and most likely storage time

Given the biology of the organism (Section 2.1.2), growth is unlikely to occur during refrigerated storage, although survival will be best under refrigerated conditions. Freezing

can reduce but not eliminate the bacterial population. For fresh chilled raw offal, the ICMSF state that their shelf life is shorter than muscle tissue, the most likely storage time will therefore be shorter than for fresh meat.

5.3.5   Heat treatment

Information from a variety of sources, including recipes, indicates that offal meats such as liver are often cooked 'rare'. For example recipes often refer to chicken livers needing to be "seared on the outside", "remain springy when touched" and "pink in the middle". Relying on the internal colour as a measure of cooking has a degree of uncertainty inherent in determining whether the *Campylobacter* organism has been destroyed. The recent ESR study (Whyte and Hudson, 2004) found that internal tissues of poultry liver can remain pink for livers sautéed rare and livers that have been cooked to greater than the recommended safe temperature of 74°C. The authors conclude that observing colour changes is not a good indication of 'doneness'. Heating livers until they reach an internal temperature of over 70°C and holding that temperature for 2 minutes or cooking for at least 5 minutes appears to be acceptable in terms of both pathogen destruction and colour/texture retention.

In one instance known to ESR staff, an organisation was advising carers to grate frozen raw liver onto baby food to increase the iron content. There seems to be ample opportunity for undercooked offal meats to be consumed.

5.3.6   Exposure summary

Contamination of raw chicken and sheep livers is common, while pig and ox liver contamination is less frequent. Consumption of offal meats by New Zealanders is low compared to other types of meat, but dishes such as liver pâté, sautéed chicken livers, lamb's fry, etc. made from these products are often on the menu at food service establishments. Undercooking, particularly of sautéed poultry livers appears to be common, given that outbreaks of campylobacteriosis associated with this type of dish have occurred in Auckland, Wellington and Christchurch (see below).

The high frequency of contamination of raw offal and associated packaging by *Campylobacter* provides an entry point for the pathogen to food preparation areas, and the home in general. Cross contamination from raw to cooked, or to ready-to-eat products may then occur. The proportion of cases that could be attributed to this transmission route is, however, difficult to quantify.

5.4   Overseas Context

5.4.1   *Campylobacter* in offal

Data from the scientific literature concerning the presence and numbers of *Campylobacter* in raw offal overseas are given in Tables 4 and 5 respectively.

Table 4:   Reported prevalence of *Campylobacter* in overseas raw offal products

Country	Product	Samples tested	Positive for *Campylobacter* (%)	Year	Reference
Mammalian offals					
Canada	Cattle offal (gall bladder, large/small intestine, **liver**, lymph nodes)	525	112 (21), most belonged to serogroup 7	NS	Garcia *et al.*, 1985
England	Cattle offal Sheep offal Pig offal	153 232 67	16 (10.5) 71 (30.6) 4 (6.0)	NS	Bolton *et al.*, 1985
England	Offal (liver, kidney and heart)	689	324 (47.0) (for "campylobacters". 90.4% of isolates were *C. jejuni*, 9.3% *C. coli* and 0.3% *C. lari*)	1984-1986	Fricker and Park, 1989
England	Pig liver Ox liver Lamb liver Lamb kidney	11 fresh 5 fresh 2 frozen 2 fresh 2 frozen 1 fresh	7 (64%) 5 (100%, all *C. jejuni)* 0 1 (50%) 1 (50%) 0	NS	Bolton *et al.*, 2002
England	Lamb liver Ox liver Pig liver (From retail sale)	96 96 99	72.9 (75% isolates *C. jejuni*, 13.5% *C. coli*) 54.2 (49.0% of isolates *C. jejuni*, 2.1% *C. coli*) 71.7 (34.3% *C. jejuni*, 42.4% *C. coli*)**	1998	Kramer *et al.*, 2000
Ireland	Offal (red meat)	13	7 (54.0)	NS	http://www.sofht.co.uk/isfht/irish_97_campylobacter.htm
Ireland	Offal	20	12 (60.0)	NS	Cloak *et al.*, 2001
Ireland, Northern	Pig liver	400	6 (67% *C. coli*, 30% *C. jejuni*)	NS	Moore and Madden, 1998
Ireland, Northern	Pig liver	30	5 (16.7) (*Campylobacter* spp.)	NS	Moore 2001
USA	Pig edible offals	405	(1%) *C.jejuni/C. coli*	NS	Zerby *et al.,*1998b cited in Sofos *et al.*, 1999
Poultry Livers					
Canada	Normal livers	50	5 (10%,all *C. jejuni*)	1991	Boukraa *et al.*, 1991
	Macroscopically abnormal livers	223	42 (19%)		
Chile	Frozen chicken livers	126	117 (93%) 78.6% isolates *C. coli*, 21.4% *C. jejuni*	NS	Fernández and Pisón 1996

Country	Product	Samples tested	Positive for *Campylobacter* (%)	Year	Reference
Czechoslovakia	Chicken livers	440	92 (25%) *C. jejuni*	1990-1991	Matasovska *et al*., 1992
Egypt	Chicken livers	50	40%	1990	Khalafalla, 1990
	Duck livers	50	36%		
	Squab (pigeon) livers	50	10%		
	Turkey livers	50	30%		
France	At slaughterhouse: Chicken Liver	7	2 (28.6%)	1999-2000	Denis *et al*., 2001
Switzerland	Fresh poultry liver*	139	43 (31%)	1995	Baumgartner *et al*., 1995
	Frozen poultry liver	144	22 (16%)		
Taiwan	Gizzard/liver	23	79 (52% *C. jejuni*, 48% *C. coli*)	2000	Shih, 2000
UK	Chicken livers	6	4 (67), all *C. jejuni*)		Bolton *et al*., 2002
USA	Chicken livers	60	51 (85%)	1980	Christopher *et al*., 1982
	Turkey livers	86	0		
USA	Chicken livers	117	56 (48%)	1983	Barot *et al*., 1983
NS	Chilled chicken livers	52	67%	NS	Bryan and Doyle, 1995
	Chicken livers	60	85%		

NS = Not stated

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

** More than one species was isolated from some samples.

These data are comparable to those obtained for New Zealand i.e. prevalence of *Campylobacter* is widespread in both poultry livers and mammalian offals for raw chilled product and somewhat less in frozen product. Prevalences are typically in the 50-75% range for raw chilled offal, which makes them equivalent to those reported in chicken meat.

Table 5: **Reported levels of *Campylobacter* on overseas chicken liver products**

Product Type	Count	Reference
Livers	<3 MPN/g 15.0% (-ve) 3-10 MPN/g 0% 11-20 MPN/g 5.0% 21-50 MPN/g 1.7% 51-100 MPN/g 5.0% 101-1100 21.7% >1100 MPN/g 51.7%	Christopher *et al*., 1982
Purge from defrosted frozen chicken livers	< MPN 10/100 ml 10% 10 MPN/100 ml 20% 22 MPN/100 ml 26% 36 MPN/100 ml 10% 51 MPN/100 ml 8% 69 MPN/100 ml 6% 120 MPN/100 ml 2% 160 MPN/100 ml 4% >230 MPN/100 ml 14%	Fernández and Pisón 1996

Product Type	Count		Reference
Fresh liver	<1/g	69.0%	Baumgartner *et al.*, 1995
	1-10/g	17.3%	
	11-100/g	7.2%	
	101-1000/g	4.3%	
	>1,000/g	2.0%	
Frozen liver	<1/g	84.7%	Baumgartner *et al.*, 1995
	1-10/g	11.1%	
	11-100/g	3.5%	
	101-1000/g	0.7%	
	>1,000/g	0%	
Offal	< 0.7 $\log_{10}$/g	16.7%	Cloak *et al.*, 2001
	0.7-1.0 $\log_{10}$/g	0%	
	1.0-1.5 $\log_{10}$/g	25.0%	
	1.5-2.0 $\log_{10}$/g	41.7%	
	2.0-2.75 $\log_{10}$/g	16.7%	

MPN= Most probable number

The quantitative data indicate that contamination is usually at quite low levels. However, given the dose response model for this organism these levels, if consumed, would result in a significant probability of disease.

A comparison of Penner serotypes and phage types from liver has been carried out in the UK. It was concluded that the subtypes isolated from lamb's liver and chicken portions were most like those involved in human cases (Kramer *et al.*, 2000). Pork liver was regarded as "a relatively minor contributor to human campylobacteriosis".

There is little information on the prevalence of *Campylobacter* in cooked offal products overseas. In Ireland, Whyte *et al*. (2004) reported analysis of pork pâté during 2001/2002. The results were from 120 samples tested, one was positive (0.8%).

5.4.2 *Campylobacter* on external packaging

One overseas study has reported external contamination of offal packaging. 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.

Offal accounted for 506 (13.8%) of the samples tested, this was recorded as liver (364; 9.9%), kidney (89; 2.4%) and heart (42; 1.1%). The aim was to identify the extent of external surface contamination. *Campylobacter* was detected from 41 (1.1%) of the external packaging samples. Results specific for offal packaging were:

- 8% (2/24) of the chicken liver packages were contaminated; 1 *C. jejuni* isolate and 1 *C. coli* isolate,
- 3.5% (6/173) of the lambs offal packaging; 6 *C. jejuni* isolates

- 6.3% (2/32) of the lamb heart packaging; 2 *C. jejuni* isolates,
- 0.8% (1/133) of the pork liver packaging; 1 *C. jejuni* isolate.

Chicken liver packaging (8%) was the second highest contaminated sample following whole game fowl at 11.1%.

Most raw offal samples were packed in a polystyrene tray with an over wrap (40%), followed by a plastic tray with heat sealed lid (23%), and polystyrene tray and heat sealed lid (16%). Most *Campylobacter* were detected on the overwrap rather than the heat sealed packaging. When normal atmosphere, modified atmosphere packing and vacuum packing were compared, more samples with *Campylobacter* were detected which were packed under normal atmosphere.

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. It was 63.3% of total notifications (23,349) in 2003, 53.2% of all notifications (22 944) in 2004 (ESR, 2005) and 60.0% (23,083) in 2005 (ESR, 2006a). The disease was discussed as a potential epidemic over 10 years ago (Lane *et al.*, 1993). Notification data for the period 1990 – 2005 are given in Table 6, and illustrated in Figures 5 (1980 – 2005) and 6 (1999 – July 2006). All references in Table 6 are Lopez *et al.* (2001) unless otherwise stated.

Table 6:   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, 2005
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 estimated 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 may be approximately 3,000 per 100,000.

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

The age distribution of cases is bimodal with peaks in the 0-4 years age group and 20-29 year group. In 2006, the highest age-specific rate occurred among children aged 1 – 4 years (511.2 per 100,000; 1105 cases). The rate for 20 to 29 year olds was 501.8 per 100,000; 2442 cases. The lowest rate was in the 10 to 14 year olds at 198.1 per 100,000; 576 cases (ESR, 2006a).

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

Figure 5:   Campylobacteriosis notifications by year 1984-2005
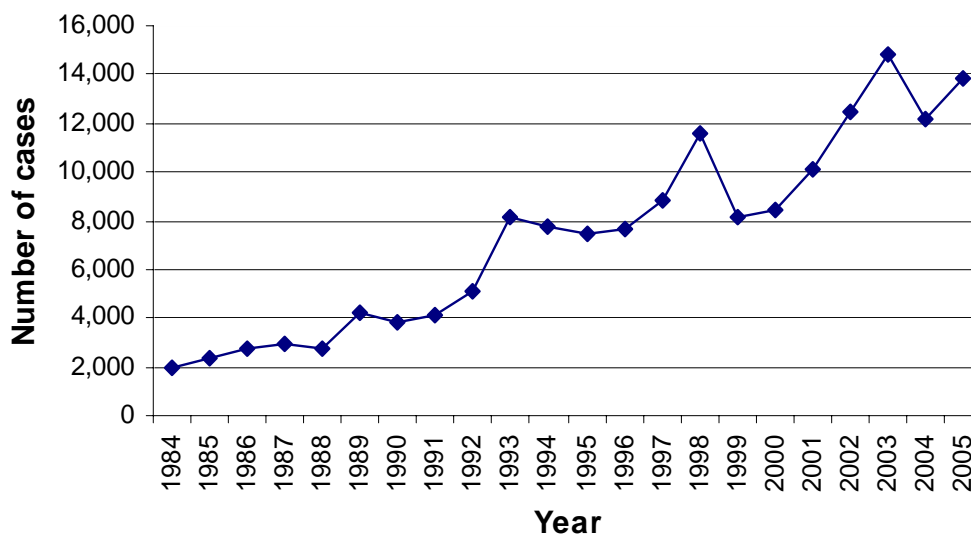
Figure 6:   Campylobacteriosis notifications by month January 1999 – July 2006
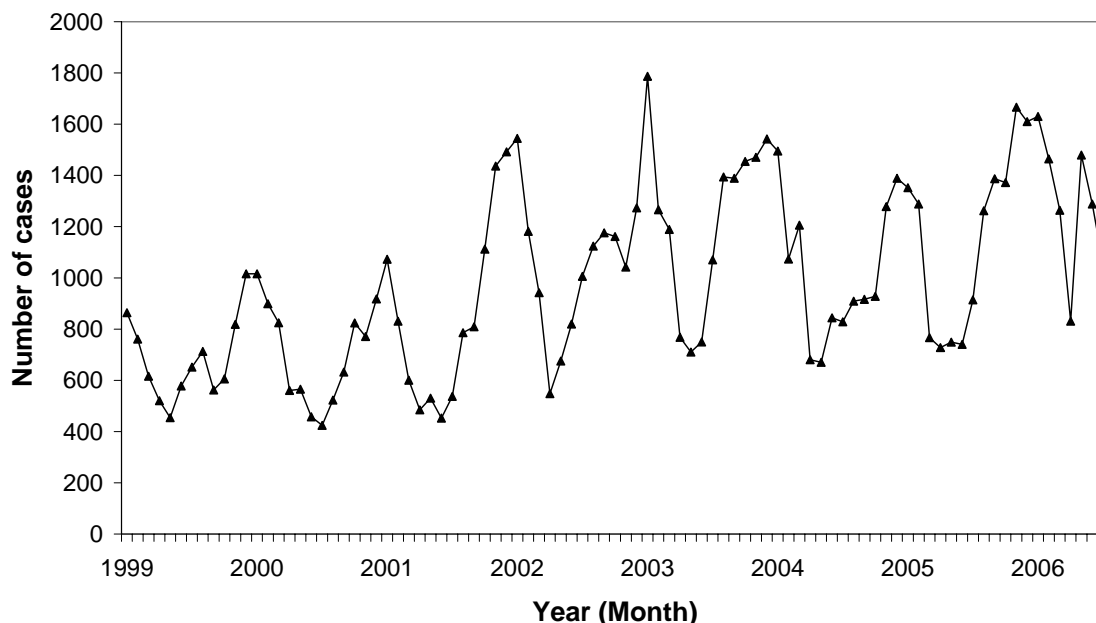
Prepared from ESR data (2006a; ESR 2006b)

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

6.1.2   Clinical consequences of *Campylobacter* infection

Hospitalisation and fatality rates for notified cases of campylobacteriosis in New Zealand are given in Table 7. 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 7:   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, 2005
2005	635/7887 (8.1%)	1/13839 (0.01%)	ESR, 2006a

6.1.3 Outbreaks

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

In contrast, the New Zealand data summarised in Table 8 show that *Campylobacter* is identified as the causative agent in around 10 - 15% of reported outbreaks. There are several possible explanations for this; 1) the result is genuine, 2) New Zealand is better at detecting outbreaks caused by campylobacteriosis or 3) the differences in rates are actually attributable to different surveillance philosophies. The average number of cases per outbreak was 3.3. It should be noted that these figures represent all outbreaks of campylobacteriosis and not just those attributed to mammalian/poultry offal.

Table 8: **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, 2005
2005	47	13.6	252	10.3	ESR, 2006c

Outbreaks of campylobacteriosis associated with poultry liver consumption reported from 1999 to August 2004 have been summarised in Table 9. Most of the outbreaks involve chicken livers prepared in restaurants/cafés. Not listed are any incidents where cross contamination is thought to have occurred.

Table 9: New Zealand outbreaks of campylobacteriosis with epidemiological (suspected) links with poultry liver consumption 1999-August 2004

Outbreak Number*	Food implicated	Number of cases (total)
AK1999030	Chicken liver	2
AK1999094	Chicken liver, undercooked	2
WN1999006	Chicken liver; undercooked	3
WN1999007	Chicken liver; undercooked	3
WN1999041	Duck liver	2
AK2000038	Chicken liver; undercooked	2
AK2000070	Chicken liver meal; undercooked	2
CB2000016	Chicken liver pâté; undercooked	12
AK2001060	Chicken liver; undercooked	3
AK2001091	Chicken liver; undercooked	2
AK2001129	Chicken liver; undercooked	2
AK2001132	Chicken liver pâté	2
AK2001142	Chicken liver; undercooked	2
AK2001136	Chicken liver; undercooked	4
AK2002142	Home made chicken liver pâté	2
AK2003154	Chicken liver salad; undercooked	2
AK2004033	Chicken liver; undercooked	2

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

The association with offal is based entirely on epidemiology (common exposure by cases); there were no laboratory confirmations where the organism was detected in the food consumed. 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.

An outbreak of 12 cases of campylobacteriosis (CB2000016) occurred in Christchurch in December 2000 and was investigated by Crown Public Health (Whyte *et al.*, 2001). The most likely cause was undercooking of chicken livers prior to pâté preparation, see Section 5.1.3.

6.1.4   Case control studies and risk factors

Two New Zealand case control studies of campylobacteriosis have been published in the scientific literature.

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 *Campylobacter* infection. Chicken pâté was eaten too infrequently to assess the risk. Consumption of mammalian offal was not mentioned. There was no significant risk in the handling of human waste, raw meat, pet ownership or time spent on a farm. Neither was there any 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. 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 meat, or consumption of chicken meat in restaurants. A statistically significant elevated odds ratio was found for consumption of chicken liver (OR 2.37, 95% CI 1.31-4.28), but not consumption of chicken pâté or beef or veal offal. 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.

Auckland Healthcare has carried out three investigations into *Campylobacter* in recent years. An outbreak in late 1996 prompted a case-control investigation into risk factors for endemic campylobacteriosis during that period (Bloomfield and Neal, 1997). Neither offal or pâté gave statistically significant elevated odds ratios for the risk of campylobacteriosis. An outbreak at a family barbecue (17 cases) in October 1998 was investigated by a retrospective cohort study (Bishop, 1998). The third case-control study (Calder *et al*., 1998) took place following a power shortage in Auckland in February 1998 and a sharp increase in *Campylobacter* spp notifications. Consumption of pâté did not give a statistically significant elevated odds ratio.

6.1.5 Serotypes causing human disease in New Zealand

Penner serotyping based on the heat stable antigen has been conducted for 1130 *Campylobacter* isolates obtained from human cases in New Zealand between 1996 and 2001. The serotypes identified include: 1,44 (16% of serotypes isolates), 2 (23%), 4 complex (15%), 5 (0.6%), 10 (0.6%), 19 (0.8%), 23 (8%), 35 (1.3%), 37 (4%), 41 (0.5%) (Lake *et al.*, 2004).

Although the source of these serotypes is unknown, the most prevalent (1,44, 2 and 4 complex) are also the most common in UK cases. A UK study examined a large dataset of Penner serotypes of *C. jejuni* from cases of human campylobacteriosis (Miller *et al.*, 2005a). The most prevalent serotypes were HS:4 complex, HS:2, and HS:1,44 (53.8% of all cases).

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

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 10. New Zealand's reported rate is high by international standards, although some differences may be due to reporting practices.

Table 10:   Comparison of reported campylobacteriosis incidence between countries

Country	Period	Rate /100,000	Reference
New Zealand	**2005**	**370.3**	**ESR, 2006a**
Australia*	2003	116.5	Miller *et al*., 2005b
Canada	2000	40.1	Health Canada, 2003
Denmark	2002	82	Anonymous, 2003
Iceland	1999 2000	116 33	ACMSF, 2004
Ireland	2001	35.5	NDSC, 2002
England & 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.
[#]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 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 11. The low percentages reinforce the sporadic nature of this illness.

Table 11: **Contribution of *Campylobacter* to reported foodborne disease outbreaks, incidents and cases overseas**

Country	Year	No. (%) Outbreaks	No. (%) incidents or cases	Reference
Canada	1984	NR	19 (1.6) incidents	Todd, 1992
England and Wales	1992-1994	19 (1)	NR	Djuretic *et al*., 1996
Germany	1993-1998	21 (2.3)	NR	www.who.it/docs/fdsaf/fs_su rvprog.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

The one overseas outbreak of campylobacteriosis associated with offal consumption that has been reported in the scientific literature is summarised in Table 12.

Table 12: **Overseas campylobacteriosis outbreaks associated with offal consumption**

Country	Food implicated	No. ill	Attack rate	Reason for food implicated	Reference
USA	Chopped liver salad contaminated by raw chicken liver juices	119	20.5	Mixed infection with *Salmonella. Campylobacter* isolated from 39 of 93 (42%) faecal specimens. Epidemiological. Foods not available for testing.	Layton *et al*., 1997

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.

In contrast, sporadic cases peak during summer months (Altekruse *et al*., 1999). 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 has 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.

6.2.3   Case control studies

Only a single case control study of campylobacteriosis conducted overseas which contained information on mammalian and poultry offal has been located. A study in Switzerland (Schorr *et al.*, 1994) found a statistically significant odds ratio for consumption of poultry liver (OR 5.7, 95% CI 1.4, 22.8), but consumption of other types of mammalian offal was not included in the study.

6.2.4   Risk assessment and other activity overseas

Disease caused by infection with *Campylobacter* is recognised as an increasing problem in many countries, and national and international efforts are being made to assess and control the problem. There are a number of risk assessments that have been conducted overseas; many of these deal with poultry meat, but mammalian and poultry offal is not often considered specifically.

A consultation paper was issued in 2003 by the Food Standards Agency, outlining a strategy for the control of *Campylobacter* in UK produced chickens reared for meat (broilers).

A detailed *Campylobacter* Sentinel Surveillance Scheme of clinical cases was initiated in the UK 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) 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 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.

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

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

6.3 Estimate of Risk for New Zealand

In New Zealand, the prevalence of *Campylobacter* in offal in general is high. External contamination of poultry livers is up to 100%, while sheep liver has a contamination prevalence of approximately 38.9% to 66.9%. Bovine and porcine offal appears to be less commonly contaminated. These results are similar to those overseas.

Offal is not as frequently consumed as poultry or red meat, but several small campylobacteriosis outbreaks, involving between 2 and 12 cases, associated with undercooked offal have been reported. There is evidence that undercooking of offal in restaurants may be routine. Limited evidence from New Zealand and overseas suggests that pâté is less frequently contaminated; nevertheless an outbreak associated with undercooked chicken liver pâté has occurred in New Zealand.

Campylobacter has also been found on the exterior of packaging of chicken liver and heart packs in New Zealand with a prevalence of approximately 56% for liver and 48% for heart (Wong *et al*., 2004). This raises the potential for cross contamination from packaging during purchase, transport and handling in the home.

It seems likely that exposure to *Campylobacter* from offal consumption in New Zealand is important, and the risk is ameliorated only by the low frequency of consumption of this food.

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 totalled 48% (Eberhart-Phillips *et al*., 1997), and USA estimates of the proportion of cases due to foodborne transmission of 55-70% (Buzby *et al*., 1996) and 80% (Mead *et al*., 1999).

The reported rate of campylobacteriosis in 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. The "*Campylobacter* in poultry" Risk Profile assigned that food/hazard combination to Category 1 (>100 per 100,000). As the contamination rates for offal are similar, but consumption is less than 20% of that for poultry, it seems reasonable to assign the *Campylobacter* in offal food/hazard combination to Category 2 (10-100 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 mammalian & poultry offals	3 (<0.5% serious outcomes)	2 (10-100 per 100,000)		

7 RISK MANAGEMENT INFORMATION

7.1 Relevant Food Controls

Options for managing the risk from *Campylobacter* in mammalian and poultry offal include:

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

No risk management measures applicable specifically to offal (other than educational material) have been found.

7.1.1 On farm control

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

7.1.1.1 Poultry biosecurity

Reduction of the prevalence of contamination of broiler chickens by *Campylobacter* as a general measure, should also reduce the prevalence of contamination of poultry offal. However, prevention of infection in broiler flocks appears to be extremely difficult, and the reported results are mixed.

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, all birds become *Campylobacter* carriers very quickly (Pattison, 2001). Non-chlorinated contaminated water derived from dams, rivers and shallow wells has been identified as a possible source for infection in flocks (Anonymous, 2006).

In contrast, the establishment of strict hygienic barriers at each poultry house has apparently worked in Scandinavia (Scientific Committee on Veterinary Measures Relating To Public Health, 2000). 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%. The Swedish production system however has some marked differences to the New Zealand system.

Although a number of authors have investigated the potential for vaccination, an effective vaccination strategy directed against *Campylobacter* in broiler chickens has yet to be developed (Newell and Wagenaar, 2000).

7.1.1.2 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.

7.1.1.3 Mammalian farm animals

While *C. jejuni* and *C. coli* are commonly found in ruminants' intestinal tracts, and *C. coli* in the intestinal tract of pigs, these organisms are not associated with any specific animal diseases. This means that farmers do not take any specific action outside of general hygiene precautions. There is a *Campylobacter* spp. (*C.fetus* subsp. *fetus*) which has been found to cause sporadic abortion in sheep and infertility, early embryonic death and abortion in cattle. Two commercial sheep vaccines are available; Campyvax3-Agvax and Campylovexin Shering Plough, however they do not offer cross protection for *C. jejuni* or *C. coli* (Graeme Jarvis, Meat and Wool New Zealand Ltd, personal communication 27/09/04). *C. jejuni* has been implicated as a cause of sporadic abortion in sheep although there are no reliable data on how important it is, but in comparison with *C. fetus* subsp. *fetus* it is minor. These reports originate in Australia, particularly Tasmania where it is common practice to feed grain to sheep on the ground, attracting birds to the feed and bird droppings into the vicinity. Sheep in New Zealand are seldom, if ever, fed grain (David West, Massey University, personal communication 28/09/04) , although wild birds may be attracted to nearby water sources.

7.1.2 Control during or after processing: general slaughterhouse hygiene

7.1.2.1 Mammalian offal

Good hygiene practices in the slaughterhouse would include the rejection of excessively dirty animals at antemortem, and careful dressing of the carcass so that no ruptures and therefore spillages of gastrointestinal contents occur over the offals. Good forced air movements around offals to ensure surface drying when chilling should also be encouraged.

The restriction that where delayed evisceration takes place, the period from stun to completion of evisceration must not exceed 45 minutes has been removed from the New Zealand Standard. However where delayed evisceration does occur much beyond 1 hour, it

is common for affected offals to be downgraded to petfood (Neil Smith, MIA, personal communication, January 2007). Where the viscera are removed, care must be taken not to puncture the gastro-intestinal tract to avoid contamination with pathogenic bacteria (including *Campylobacter*) onto the carcass and edible offal. Livers must be presented for inspection with at least one lymph node attached. Gall bladders are not removed from condemned livers.

7.1.2.2 Poultry offals

Control of cross contamination for broilers at slaughter is considered harder to implement. 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 of carcasses to reduce bacterial load
- "Logistical" slaughter of uninfected flocks before infected ones.

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). Irradiation has not been approved in New Zealand.

7.1.2.3 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.

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

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

7.1.3 Consumers and foodservice operators

General consumer advice for control of pathogens in mammalian and poultry offal 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 NZFSA website also contains advice on the safe cooking of chicken livers, see website http://www.nzfsa.govt.nz/consumers/food-safety-topics/foodborne-illnesses/safe-cooking-of-chicken-livers/index.htm. This factsheet is directed at consumers as well as foodservice operators, and the intention is that the NZFSA will disseminate paper copies to operators via Health Protection Officers.

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

A useful catalogue of measures to prevent *Campylobacter* contamination based on the UK experience and Scandinavian countries has been compiled by the ACMSF (2004) in its second report to the Food Standards Agency in the UK. The document can be found at the following website: http://www.food.gov.uk/multimedia/pdfs/acmsfcampyloreport.pdf.

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, as well as a high incidence of campylobacteriosis caused by strains of which 90% are genetically indistinguishable from those that occur in poultry. The risk assessment model being created from the study is expected to benefit other countries (Stern *et al.*, 2003).

In Denmark, a quantitative risk model to investigate campylobacteriosis associated with poultry has been developed (Rosenquist *et al.,* 2003) although it concentrates on chicken carcasses rather than offal.

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

In 2000, Ireland reported 44% contamination of offal samples (all meat types) with *Campylobacter.* 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.

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 mammalian and poultry offal alone.

The cost estimate of $40,136,000 assumed that the ratio of notified (visit a GP) to unreported (community) cases of campylobacteriosis was 1:7.6, based on data from a prospective English study (Wheeler *et al*., 1999). The notification figure for this estimate was taken from the most up to date reported cases rate at the time, i.e. 1998 at 320 per 100,000. In the last two years, the reported cases rate have decreased from 395.6 in 2003 to 326.8 in 2004. A rate similar to the 1998 figure. Campylobacteriosis still represents the majority of infectious intestinal disease costs.

7.3   Other Transmission Routes

7.3.1   Other transmission routes: food

Undercooked poultry has been the transmission vehicle most commonly identified in case control studies of campylobacteriosis. Unpasteurised milk has been associated with several outbreaks in the United Kingdom (Frost, 2001). In New Zealand *Campylobacter* has also been isolated from watercress and was the subject of a Director-General of Health statement in 2000.

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

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

7.3.2   Other transmission routes: environment

Campylobacter is widespread in the environment although clear routes for transfer from the environment to the consumer have yet to be identified (Jones, 2001). The seasonal incidence of intestinal disease caused by *Campylobacter* has characteristics suggesting waterborne transmission, and internationally several outbreaks have been associated with drinking water, albeit usually from private, non-reticulated water supplies (Jones, 2001). In the UK from 1992 to 1994, the number of outbreaks associated with water outnumbered those associated with poultry (Frost, 2001).

In a study in New Zealand *Campylobacter* appears to be widespread (60-75% positive) but at low numbers in river water and shallow ground water, while roof 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 year long survey of treated drinking water supplies, commissioned by the Ministry of Health, has shown almost no contamination prevalence, except for a small supply whose UV treatment process had failed (Nokes *et al.*, 2004).

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

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

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

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

8   CONCLUSIONS

8.1   Description of Risks to New Zealand Consumers

8.1.1   Risks associated with mammalian and poultry offal products

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

In New Zealand, the prevalence of *Campylobacter* in offal in general is high. External contamination of poultry livers is up to 100% while internal contamination is up to 90%. Sheep liver has a contamination prevalence of approximately 38.9% to 66.9%. Beef and pig offals appear to be less commonly contaminated (<10%).

Evidence from reported and investigated campylobacteriosis outbreaks indicates that offals represent a transmission route for this illness in New Zealand. This is supported by an elevated odds ratio for poultry liver consumption in the large national case control study, but not smaller more localised case control studies. Pâté consumption may be a lower risk, based on the largest case control study in New Zealand, and the demonstration that properly prepared pâté did not contain *Campylobacter* in the experimental studies described in Section 5.1.3.

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". 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 offal which have been shown to frequently contaminated.

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

8.1.2   Risks associated with other foods

There is evidence from case control studies and other sources that consumption of poultry meat plays an important role in the transmission of this infection in New Zealand.

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 meat is a significant vehicle for the foodborne component. It seems possible that part of the increase in notified cases of campylobacteriosis over the period 1990 to 2003 is due to increasing consumption of poultry meat over the same period. However, this does not explain why the campylobacteriosis rate in New Zealand is markedly higher than other countries. The prevalence of *Campylobacter* in New Zealand uncooked poultry products appears similar to the levels in other countries. While the comparison of consumption of

poultry meat with Australia is not clear-cut, the amounts do not seem to differ sufficiently to explain the difference in disease incidence.

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

8.1.3   Risk assessment options

A quantitative risk assessment (QRA) for exposure to *Campylobacter* via offal is likely to be feasible. Reasonable data are available for the prevalence of the organism in raw chicken and sheep livers although local numerical data are few. Good consumption data are available although there is a large disparity between the supply on the market and consumption figures which has not been adequately explained. Occupational exposure through handling of offal packs and preparation for pet food may need to be included.

Information on cooking practices is scarce and may well tend towards undercooking. Dose response relationships are available and could be used to produce a risk characterisation. Targeted projects to provide information on data gaps would greatly assist a QRA, and cooperation with industry would be essential.

However application of QRA to cross contamination in the domestic and retail environments, which are likely to be significant, would be difficult, and not achievable given current data. There are two aspects to this:

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

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

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

Understanding this would allow modellers to focus on what is important to produce useful, simple cross contamination modules.

Given the high level of campylobacteriosis in New Zealand, a QRA would be useful to assess the significance of mammalian and poultry offal as a source of infection so that risk based interventions/standards could be justified and then implemented.

8.2 Commentary on Risk Management Options

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

- Attempt to obtain better control on farms, thereby reducing the prevalence of contamination in animals,
- Reduced sale of fresh offal and increased sales of frozen products, and
- Further consumer food safety education campaigns, including chef training (e.g. addressing the undercooking of livers).

Investigation of potential sources of infection and "on farm" control measures specifically for *Campylobacter* could reduce contamination levels of mammalian and poultry offal products at retail, although cross contamination during processing would remain a problem. Efforts to reduce contamination in animals in general will apply to both meat and offal, and so specific risk management efforts for offal at this level do not appear to be required, except perhaps for some aspects of processing.

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 are currently being supplemented by educational efforts directed at chefs (as described in Section 7.1.3). Given the potential for internal contamination of offal (unlike meat), specific educational efforts to address offal cooking are warranted. These should be supported by further investigation into the factors that affect the handling of mammalian and poultry offal in domestic kitchens, particularly in regard to cross contamination.

8.3 Data Gaps

The data gaps identified in this Risk Profile are:

- Information on the domestic abattoir handling of offals – particularly cooling methods;
- Data on the sero/genotypes of *Campylobacter* present in offal samples, particularly from poultry livers;
- Data on the contamination of mammalian offal packs; and,
- Information on domestic handling of livers and kidneys, including cooking practices.

9   REFERENCES

ACMSF (2004) Second report on *Campylobacter*: memorandum on research. United Kingdom Advisory Committee on the Microbiological Safety of Food. Available from: http://www.food.gov.uk/foodindustry/Consultations/completed_consultations/completeduk/acmsfcampylobacter

Adak GK, Cowden JM, Nicholas S, Evans HS. (1995) The public health laboratory service national case-control study of primary indigenous sporadic cases of *Campylobacter* infection. Epidemiology and Infection; 115: 15-22.

AIFST. (2003) *Campylobacter*, Foodborne Microorganisms of public health significance, 6th edition, NSW Branch, Chapter 10.

Altekruse SF, Stern NJ, Fields PI, Swerdlow DL. (1999) *Campylobacter jejuni*-An emerging foodborne pathogen. Emerging Infectious Diseases; 5: 28-35.

Anonymous. (1999) Chicken tonight? Consumer; 381: 6-9.

Anonymous. (2003) Annual Report on Zoonoses in Denmark, 2002, Ministry of Food, Agriculture and Fisheries. Denmark. www.vetinst.dk

Anonymous (2006) Avian *Campylobacter* Infection : Introduction. The Merck Veterinary Manual http://www.merckvetmanual.com/mvm/index.jsp?cfile=htm/bc/201600.htm

Australian Bureau of Statistics. (1999) National Nutrition Survey. Foods Eaten. Canberra: Australian Bureau of Statistics.

Baker M, Ball A, Devane M, Garrett N, Gilpin B, Hudson JA, Klena J, Nicol C, Savill M, Scholes P, Williams D. (2002) Potential transmission routes of *Campylobacter* from environment to humans. Report prepared for the Ministry of Health and New Zealand Food Safety Authority. Christchurch: ESR.

Barot MS, Mosenthal AC, Bokkenhauser VD. (1983) Location of *Campylobacter jejuni* in infected chicken livers. Journal of Clinical Microbiology; 17: 921-922.

Baumgartner A, Grand M, Liniger M, Simmen A. (1995) *Campylobacter* infections of poultry liver-consequences for food handlers and consumers. Archiv für Lebensmittelhygiene; 46: 11-12.

Bishop J. (1998) Family BBQ O/B AK1998081. Auckland: Auckland Healthcare.

Bloomfield A, Neal G. (1997) A case control study of *Campylobacter* infections notified to Auckland Healthcare during a 25 day period in October/November 1996. Auckland: Auckland Healthcare.

Bolton FJ, Hawkins HC, Hutchinson DN. (1985) Biotypes and serotypes of thermophilic campylobacters isolated from cattle, sheep and pig offal and other red meats. Journal of Hygiene, Cambridge; 95:1-6.

Bolton FJ, Williamson JK, Allen G, Wareing DR, Frost JA. (1999) Prevalence of *C. jejuni* and *C. coli* in meat products and packaging sold at retail: a potential public health problem. 10th International Workshop on *Campylobacter*, *Helicobactor* and Related Organisms. Baltimore, Maryland 12-16 September 1999.

Bolton FJ, Sails AD, Fox AJ, Wareing DRA, Greenway DLA. (2002) Detection of *Campylobacter jejuni* and *Campylobacter coli* in foods by enrichment culture and polymerase chain reaction enzyme-linked immunosorbent assay. Journal of Food Protection; 65(5): 760-767.

Boukraa L, Messier S, Robinson Y. (1991) Isolation of *Campylobacter* from livers of broiler chickens with and without necrotic enteritis. Avian Diseases; 35: 714-717.

Boxall N, Ortega J. (2003) Annual summary of outbreaks in New Zealand 2002. ESR Client Report FW0326. Porirua: ESR.

Boxall N. (2005) The epidemiology of *Campylobacter jejuni* in commercial broiler flocks in New Zealand. PhD thesis, Massey University, Palmerston North, New Zealand.

Brmbolić B. (1995) Multiple abscesses of the liver caused by *Campylobacter jejuni*. Journal of Clinical Gastroenterology; 20: 307-309.

Bryan FL, Doyle MP. (1995) Health risks and consequences of *Salmonella* and *Campylobacter jejuni* in raw poultry. Journal of Food Protection; 58: 326-344.

Burch D. (2005) Avian vibrionic hepatitis in laying hens. The Veterinary Record; 157: 528.

Buzby JC, Roberts T, Lin, C-TJ, MacDonald JM. (1996) Bacterial foodborne disease: Medical costs and productivity losses. Agricultural Economic Report Number 741. Washington DC: United States Department of Agriculture Economic Research Service,.

Calder L, Manning K, Nicol C. (1998) Case-control study of Campylobacteriosi*s* epidemic in Auckland. Auckland: Auckland Healthcare.

Campbell KW, Gilbert SA. (1995) National Food Project: Poultry Quality Assessment. Wellington, New Zealand: Ministry of Health.

CDC (2003) Preliminary FoodNet data on the incidence of foodborne illness-Selected sites, United States, 2002. Morbidity and Mortality Weekly Reports, April 18 2003; 52: 340-343 http://www.cdc.gov/mmwr/preview/mmwrhtml/mm5215a4.htm

Christopher FM, Smith GC, Vanderzant C. (1982) Examination of poultry giblets, raw milk and meat for *Campylobacter fetus* subsp. *jejuni*. Journal of Food Protection; 45: 260-262.

Cloak OM, Duffy G, Sheridan JJ, Blair IS, McDowell DA. (2001) A survey on the incidence of *Campylobacter* spp. and the development of a surface adhesion polymerase chain reaction (SA-PCR) assay for the detection of *Campylobacter jejuni* in retail meat products. Food Microbiology; 18: 287-298.

Codex. (1976) Recommended Code of Hygienic Practice for Poultry Processing. CAC/RCP 14) Rome: Codex Alimentarius Commission.

Codex. (1999) Draft principles and guidelines for the conduct of microbiological risk assessment. Report of the thirty first session of the Codex committee on food hygiene. ALINORM 99/13A. Rome: Codex Alimentarius Commission.

Cogan TA, Bloomfield SF, Humphrey TJ. (1999) The effectiveness of hygiene procedure for prevention of cross-contamination from chicken carcasses in the domestic kitchen. Letters in Applied Microbiology; 29: 354-358.

Cornelius AJ, Nicol C, Hudson JA. (2005) *Campylobacter* spp. In New Zealand raw sheep liver and human campylobacteriosis cases. International Journal of Food Microbiology;99:99-105.

Corry JEL, Atabay HI. (2001) Poultry as a source of *Campylobacter* and related organisms. Journal of Applied Microbiology; 90: 96S-114S.

Cox NA, Richardson LJ, Buhr RJ, Fedorka-Cray PJ, Bailey JS, Wilson JL, Hiett KL. (2006) Natural presence of *Campylobacter* spp. in various internal organs of commercial broiler breeder hens. Avain Diseases; 50: 450-453.

Danish Veterinary and Food Administration. (1998) Risk profile for pathogenic species of *Campylobacter* in Denmark. Available from:
http://www.lst.min.dk/publikationer/publikationer/publikationer/campuk/cameng_ref.doc

De Boer E, Hahné M. (1990) Cross contamination with *Campylobacter jejuni* and *Salmonella* spp. from raw chicken products during food preparation. Journal of Food Protection; 53: 1067-1068.

Denis M, Refrégier-Petton J, Laisney M-J, Ermel G and Salvat G. (2001) *Campylobacter* contamination in French chicken production from farm to consumers. Use of a PCR assay for detection and identification of *Campylobacter jejuni* and *Camp. coli*. Journal of Applied Microbiology; 91: 255-267.

Devane M, Scholes P, Hudson JA, Klena JD Savill M. (2001) Validation and application of a multiplex PCR system to determine the significance of feral rabbits and possums as environmental reservoirs of *Campylobacter*. Poster presented at NZMS annual meeting, Wellington, New Zealand.

Devane M, Nicol C, Ball A, Klena J, Scholes P, Hudson JA, Baker M, Gilpin B, Garret N, Savill M. (2005) The occurrence of *Campylobacter* subtypes in environmental reservoirs and potential transmission routes. Journal of Applied Microbiology; 98: 980-990.

Djuretic T, Wall PG, Ryan MJ, Evans HS, Adak GK, Cowden JM. (1996) General outbreaks of infectious intestinal disease in England and Wales 1992 to 1994. CDR Review; 6: R57-R63.

Eberhart-Phillips J, Walker N, Garrett N, Bell D, Sinclair D, Rainger W, Bates M. (1997) Campylobacteriosis in New Zealand: results of a case control study. Journal of Epidemiology and Community Health; 51: 686-691.

EPA (US Environmental Protection Agency). (1997) Exposure Factors Handbook. Volumes I, II and III. http://cfpub.epa.gov/ncea/cfm/exposfac.cfm?ActType=default

ESR. (2002) Annual summary of outbreaks in New Zealand 2001. Porirua: ESR.

ESR (2003) Risk Profile: *Campylobacter jejuni/coli* in Poultry (whole and pieces). Client Report 0109, Christchurch, ESR.

ESR (2004) Notifiable and other diseases in New Zealand. Annual Report 2003. ESR Client Report FW0426. Porirua: ESR.

ESR. (2005) Notifiable and other diseases in New Zealand. Annual Report 2004 ESR Client Report FW 0532, Porirua, April 2005.

ESR. (2006a) Notifiable and other diseases in New Zealand. Annual Report 2005 ESR Client Report FW 0621, Porirua, April 2006.

ESR (2006b) Monthly Surveillance Report – July 2006. Public Health Surveillance; Porirua http://www.surv.esr.cri.nz/surveillance/monthly_surveillance.php?we_objectID=1119 (website accessed 22.09.06)

ESR (2006c) Annual Summary of Outbreaks in New Zealand, 2005. ESR Client Report FW00623, Porirua, April 2006

Evans HS, Madden P, Douglas C, Adak GK, O'Brien SJ, Djuretic T, Wall PG, Stanwell-Smith R. (1998b) General outbreaks of infectious intestinal disease in England and Wales: 1995 and 1996. Communicable Disease and Public Health; 1: 165-171.

FAO/WHO (2002) Risk Assessment of *Campylobacter* spp. in broiler chickens and *Vibrio* spp. in seafood, a joint FAO/WHO consultation, Bangkok, Thailand. 5-9 August 2002 http://www.who.int/foodsafety/publications/micro/august2002/en/

Fernández, H., Pisón V. (1996) Isolation of thermotolerant *Campylobacter* from commercial chicken livers. International Journal of Food Microbiology; 29: 75-80.

Fricker CR, Park RWA. (1989) A two-year study of the distribution of "thermophilic" campylobacters in human, environmental and food samples from the Reading area with particular reference to toxin production and heat-stable serotype. Journal of Applied Bacteriology; 66: 477-490.

Friedman CR, Neimann J, Wegener HC, Tauxe RV. (2000) Epidemiology of *Campylobacter jejuni* infections in the United States and other industrialised nations. In: *Campylobacter* (I Nachamkin and MJ Blaser (eds.)). Washington DC: American Society for Microbiology: 121-138.

Frost JA. (2001) Current epidemiological issues in human campylobacteriosis. Journal of Applied Microbiology; 90: 85S-95S.

Garcia MM, Lior H, Stewart RB, Ruckerbauer GM, Trudel JRR, Skljarevski A. (1985) Isolation, characterization, and serotyping of *Campylobacter jejuni* and *Campylobacter coli* from slaughter cattle. Applied and Environmental Microbiology; 49: 667-672.

Gibson JR, Sutherland K, Owen JR. (1994). Inhibition of DNAase activity in PFGE analysis of DNA from *Campylobacter*. Letters in Applied Microbiology; 19: 357-358.

Gill CO, Harris LM. (1982) Contamination of red-meat carcasses by *Campylobacter fetus* subsp. *fetus*. Applied and Environmental Microbiology; 43: 977-980.

Gilpin B. (2004) New Zealand microbial typing database development 2003 – 2004. FW0457. Christchurch: ESR.

Graves TK, Bradley KK, Crutcher JM. (1998) Outbreak of *Campylobacter* enteritis associated with cross-contamination of food-Oklahoma, 1996. Mortality and Morbidity Weekly Reports; 47: 129-131.

Hänninen, M-L. (1981) Survival of *Campylobacter jejuni/coli in* ground refrigerated and in ground frozen beef liver and in frozen broiler carcasses. Acta Veterinaria Scandinavica; 22: 566-577.

Harrison WA, Griffith CJ, Tennant D, Peters AC. (2001) Incidence of *Campylobacter* and *Salmonella* isolated from retail chicken and associated packaging in South Wales. Letters in Applied Microbiology; 33: 450-454.

Hartnett E, Kelly L, Newell D, Woolridge M, Gettinby G. (2001) A quantitative risk assessment for the occurrence of *Campylobacter* in chickens at the point of slaughter. Epidemiology and Infection; 127:195-206.

Health Protection Agency. (2003) Annual Report 2002. Division of Gastrointestinal Infections. Central Public Health Laboratory. United Kingdom. http://www.hpa.org.uk/srmd/div_dgi/DGI%20Annual%20Report%202002.pdf

Health Protection Agency (2004) Bacteria on raw meat packaging: Press release. http://www.hpa.org.uk/hpa/news/articles/issues/2004/040415_meat_packaging.htm. 15 April 2004.

Health Canada. (2003). Notifiable Diseases On-line. Laboratory Centre for Disease Control of Health Canada. http://dsol-smed.hc-sc.gc.ca/dsol-smed/cgi-bin/ndischart2?DATA_TYPE=R&YEAR_FROM=88&YEAR_TO=00&CAUSE=016&AREA=00&AGE=0&SEX=3&CTIME1=View+Chart

Hudson, JA. (1997) Typing of *Campylobacter* isolates from human cases, raw poultry, milk and water, and a comparison of two methods for the detection of *Campylobacter* in foods. A report for the Ministry of Health Client Report FW9725. Christchurch: ESR.

Hudson JA, Nicol C, Wright J, Whyte R, Hasell SK. (1999) Seasonal variation of *Campylobacter* types from human cases, veterinary cases, raw chicken, milk and water. Journal of Applied Microbiology; 87: 115-124.

Humphrey TJ, Martin KW, Slader J, Durham K. (2001) *Campylobacter* spp. in the kitchen: spread and persistence. Journal of Applied Microbiology; 90: 115S-120S.

ICMSF. (1996). Microorganisms in foods 5. Microbiological specifications of food pathogens. International Commission on Microbiological Specifications for Foods (ICMSF). London: Blackie Academic and Professional.

ICMSF. (1998). Micro-organisms in foods 6. Microbial ecology of food commodities. International Commission on Microbiological Specifications for Foods (ICMSF). London: Blackie Academic and Professional.

Ikram R, Chambers S, Mitchell P, Brieseman M, Ikram OH. (1994) A case control study to determine risk factors for *Campylobacter* infection in Christchurch in the summer of 1992-3. New Zealand Medical Journal; 107: 430-432.

Jacobs-Reitsma W. (2000) *Campylobacter* in the food supply. In: *Campylobacter* (I Nachamkin and MJ Blaser (eds.)). Washington DC: American Society for Microbiology: 467-481.

Jones DM, Sutcliffe EM, Rios R, Fox AJ, Curry A. (1993) *Campylobacter jejuni* adapts to aerobic metabolism in the environment. Journal of Medical Microbiology; 38: 145-150.

Jones K. (2001) *Campylobacter* in water, sewage and the environment. Journal of Applied Microbiology; 90: 68S-79S.

Kelly DJ (2001) The physiology and metabolism of *Campylobacter jejuni* and *Helicobacter pylori.* Journal of Applied Microbiology; 90:16S-24S.

Khalafalla FA. (1990) *Campylobacter jejuni* in poultry giblets. Journal of Veterinary Medicine; 37: 31-34.

Kieft C, Perks M, Baker M, Galloway Y, Sutherland H. (2000) Annual Surveillance Summary 1999. Institute of Environmental Science and Research Client Report FW0059. Porirua: ESR.

Klena J. (2001) A survey of phenotypic and genetic methods used to identify and differentiate thermotolerant *Campylobacter* spp. strains. Report to the Ministry of Health.

Kramer JM, Frost JA, Bolton FJ, Wareing DRA. (2000) *Campylobacter* contamination of raw meat and poultry at retail sale: Identification of multiple types and comparison with isolates from human infection. Journal of Food Protection; 63: 1654-1659.

Lake RJ, Baker MG, Garrett N, Scott WG, Scott HM. (2000) Estimated number of cases of foodborne infectious disease in New Zealand. New Zealand Medical Journal; 113: 278-281.

Lake R, Baker M, Nicol C, Garrett N. (2004) Lack of association between long-term illness and infectious intestinal disease in New Zealand. New Zealand Medical Journal; 117. http://www.nzma.org.nz/journal/117-1194/893/.

Lane L, Baker M. (1993) Are we experiencing an epidemic of *Campylobacter* infection? Communicable Disease New Zealand; 93: 59-62,68.

Layton MC, Calliste SG, Gomez TM, Patton C. (1997) A mixed foodborne outbreak with *Salmonella heidelberg* and *Campylobacter jejuni* in a nursing home. Infection Control and Hospital Epidemiology; 18: 115-121.

Lee A, Smith SC, Coloe PJ. (2000) Detection of a novel *Campylobacter* enterotoxin. Journal of Applied Microbiology; 89: 719-725.

Lindqvist R, Andersson Y, De Jong B, Norberg P. (2000) A summary of foodborne disease incidents in Sweden, 1992 to 1997. Journal of Food Protection; 63: 1315-1320.

Lopez L, Baker M, Perks M. (2001) Annual surveillance summary 2000. Client Report FW0156. Porirua: ESR.

MAF. (2003) Situation and Outlook for New Zealand Agriculture and Forestry 2003. http://www.maf.govt.nz/mafnet/rural-nz/statistics-and-forecasts/sonzaf/2003/2003-sonzaf-23.htm#P2045_200399

MAF. (2005) Situation and Outlook for New Zealand Agriculture and Forestry 2003. http://www.maf.govt.nz/mafnet/rural-nz/statistics-and-forecasts/sonzaf/2005/2005-update/2005-sonzaf-10.htm

Matasovska N, Sladka, O, Mraz, O, Matyas, Z, Tomancova, I. (1992) *Campylobacter jejuni* in slaughtered chickens from the viewpoint of food hygiene. ACTA Vet. BRNO; 61: 61-67.

McCarthy N, Andersson Y, Jormanainen V, Gustavsson O, Giesecke J. (1999) The risk of Guilain-Barré syndrome following infection with *Campylobacter jejuni*. Epidemiology and Infection; 122: 15-17.

Mead PS, Slutsker L, Dietz V, McCaig LF, Bresee JS, Shapiro C, Griffin PM, Tauxe RV. (1999) Food-related illness and death in the United States. Emerging Infectious Disease; 5: 607-625.

Meanger JD, Marshall RB. (1989) Seasonal prevalence of thermophilic *Campylobacter* infections in dairy cattle and a study of infection of sheep. New Zealand Veterinary Journal; 37:18-20.

Medema GJ, Teunis PFM, Havelaar AH, Haas CN. (1996) Assessment of the dose-response relationship of *Campylobacter jejuni*. International Journal of Food Microbiology; 30: 101-111.

Miller G, Dunn GM, Reid TMS, Ogden ID, Strachan NJC. (2005a) Does age acquired immunity confer selective protection to common serotypes of *Campylobacter jejuni*? BMC Infectious Diseases; 5: 66.

Miller M, Roche P, Yohannes K, Spencer J, Bartlett M, Bortherton J, Hutchinson J, Kirk M, McDonald A, Vadjic C. (2005b) Australia's notifiable diseases status, 2003. Communicable Diseases Intelligence; 29: 1-61.

Ministry of Health/Ministry of Agriculture and Forestry. (2000) Food Administration in New Zealand: A Risk Management Framework for Food Safety. Wellington: Joint Ministry of Health and Ministry of Agriculture and Forestry Food Harmonisation Project.

Moore JE. (2001) An optimised recovery method for thermophilic *Campylobacter* from liver. BMC Microbiology; 1:32.

Moore JE, Madden RH. (1998) Occurrence of thermophilic *Campylobacter* spp. In porcine liver in Northern Ireland. Journal of Food Protection; 61(4): 409-413.

Moore JE, Madden RH (2001) Survival of *Campylobacter coli* in porcine liver. Food Microbiology; 18: 1-10.

Munroe DL, Prescott JF, Penner JL. (1983) *Campylobacter jejuni* and *Campylobacter coli* serotypes isolated from chickens, cattle and pigs. Journal of Clinical Microbiology; 18: 877-881.

Murphy C, Carroll C, Jordan KN. (2006) Environmental survival mechanisms of the foodborne pathogen *Campylobacter jejuni*. Journal of Applied Microbiology; 100: 623-632.

Naing T, Baker M, Galloway Y, Margolin T. (1999) Annual summary of outbreaks in New Zealand 1998. Porirua: ESR.

NDSC. (2002) Report on Campylobacteriosis in Ireland, 2001.
http://www.ndsc.ie/Publications/CampylobacterAnnualReports/d927.PDF

Newell DG, Wagenaar JA. (2000) Poultry infections and their control at the farm level. In: *Campylobacter* (I Nachamkin and MJ Blaser (eds.)). Washington DC: American Society for Microbiology: 497-509.

Nokes C, Devane M, Scholes P, Nourozi F, Ritchie J, Gilpin B, Ball A, Savill M, McBride G, (2004) Survey of *Campylobacter* in New Zealand's treated drinking waters. Proceedings of the New Zealand Water and Wastes Association annual conference, 2004, Christchurch, New Zealand.

Olsen SJ, MacKinon LC, Goulding JS, Bean NH, Slutsker L. (2000) Surveillance for foodborne disease outbreaks-United States, 1993-1997. Morbidity and Mortality Weekly Reports; 49(SS01): 1-51.

Oosterom J, De Wilde GJA, De Blaauw LH, Karman H. (1983) Survival of *Campylobacter jeuni* during poultry processing and pig slaughter. Journal of Food Protection; 46: 702-706.

Oosterom J, Dekker R, De Wilde GJA, van Kempen-de Troye F. (1985). Prevalence of *Campylobacter jejuni* and *Salmonella* during pig slaughtering. Veterinary Quarterly; 7: 31-34.

Pattison M. (2001) Practical intervention strategies for *Campylobacter*. Journal of Applied Microbiology; 90: 121S-125S.

Pebody RG, Ryan MJ, Wall PG. (1997) Outbreaks of *Campylobacter* infection: rare events for a common pathogen. CDR Review; 7: R33-R37.

Penner JL, Hennessy JN. (1980) Passive haemagglutination technique for serotyping *Campylobacter fetus* subsp.*jejuni* on the basis of heat-stable antigens. Journal of Clinical Microbiology; 12: 732-7.

Perelman B, Greiff M, Kuttin ES, Rogol M. (1992) Campylobacteriosis in ostriches. Israel Journal of Veterinary Medicine; 47: 116-119.

Perks M, Galloway Y, Baker M. (1999) Annual Surveillance Summary 1998. Porirua: ESR.

Perks M, Baker M, Galloway Y. (2000) Annual summary of outbreaks in New Zealand 1999. Porirua: ESR.

PIANZ (Poultry Industry Association of New Zealand). (1995) Poultry Industry Agreed Standards and Code of Practice. Poultry Industry Association of New Zealand Inc.

PIANZ (Poultry Industry Association of New Zealand). (1999) An outline of the New Zealand poultry industry. The poultry meat industry. http://www.pianz.org.nz/about.htm

Rees JH, Soudain SE, Gregson NA, Hughes RAC. (1995) *Campylobacter jejuni* infection and Guillain-Barré syndrome. New England Journal of Medicine; 333: 1374-1379.

Rosef O. (1981) Isolation of *Campylobacter fetus* subsp. *jejuni* from the gallbladder of normal slaughter pigs using an enrichment procedure. Acta Veterinaria Scandinavica; 22: 149-151.

Rosenquist H, Nielsen NL, Sommer HM, Nørrung B, Christensen BB. (2003) Quantitative risk assessment of human campylobacteriosis associated with thermophilic *Campylobacter* species in chickens. International Journal of Food Microbiology;83:87-103.

Savill MG, Hudson JA, Ball A, Klena JD, Scholes P, Whyte RJ, McCormick RE, Jankovic D. (2001) Enumeration of *Campylobacter* in New Zealand recreational and drinking waters. Journal of Applied Microbiology; 91: 38-46.

Sánchez MX, Fluckey WM, Brashears MM, McKee SR. (2002) Microbial profile and antibiotic susceptibility of *Campylobacter* spp. and *Salmonella* spp. in broilers processed in air-chilled and immersion-chilled environments. Journal of Food Protection; 65: 948-956.

Schaffner D. (2002) Modelling cross-contamination in quantitative microbial risk assessment. 1st International Conference on Microbial Risk Assessment: Foodborne Hazards, College Park, MD, USA.

Schorr D, Schmid H, Rieder HL, Baumgartner A, Vorkauf H, Burnens A. (1994) Risk factors for *Campylobacter* enteritis in Switzerland. Zentralblatt fur Hygiene und Umweltmedizin; 196: 327-337.

SCIEH (Scottish Centre for Infection and Environmental Health) (2004) Vol. 38;2004/01 http://www.show.scot.nhs.uk/scieh/PDF/pdf2004/0401.pdf

Scott WG, Scott HM, Lake RJ, Baker MG. (2000) Economic cost to New Zealand of foodborne infectious disease. New Zealand Medical Journal; 113: 281-284.

Shih DY-C. (2000) Isolation and identification of enteropathogenic *Campylobacter* spp. from chicken samples in Taipei. Journal of Food Protection; 63: 304-308.

Simmons G, Lindsay A, Harrison S. (2001) Evaluation of the inaugural New Zealand Foodsafe Partnership Food Safety campaign. Journal of the New Zealand Dietetic Association; 55: 8-13.

Sneyd E, Lopez L, Eglinton M, McDowell R, Margolin T. (2002) Annual surveillance summary 2001. Client Report FW0156. Porirua: ESR.

Sneyd E, Baker M. (2003) Infectious diseases in New Zealand: 2002 Annual Surveillance Summary. ESR Client Report FW0332. Porirua: ESR.

Soonattrakul W, Andersen BR, Bryner JH. (1971) Raw liver as a possible source of Vibrio fetus septicemia in man. American Journal of the Medical Sciences; 261: 245-248.

Stern NJ, Hiett KL, Alfredsson GA, Kristinsson KG, Reiersen J, Hardardottir H, Briem H, Gunnarsson E, Georgsson F, Lowman R, Berndtson E, Lammerding AM, Paoli GM,

Stephens CP, On SLW, Gibson JA. (1998) An outbreak of infectious hepatitis in commercially reared ostriches associated with *Campylobacter coli* and *Campylobacter jejuni*. Veterinary Microbiology; 61: 183-190.

Tam CC. (2001) *Campylobacter* reporting at its peak year of 1998; don't count your chickens yet. Communicable Disease and Public Health; 4 (3): 194-199.

Taylor MH, Helbacka NVL, Bigbee DE. (1968) The effect of packaging giblets separately on quality of fresh broilers. Poultry Science; 47: 1963-1966.

Taylor J, Boland C. (2001) Antimicrobial resistance review – a New Zealand perspective. Agricultural Compounds and Veterinary Medicines Group, MAF. Available from: http://www.maf.govt.nz/acvm/publications/information-papers/nzva-abpaper.pdf

Teunis PFM, Havelaar AH. (2000) The beta poisson dose-response model is not a single hit model. Risk Analysis; 20:513-520.

Teunis PFM, Nagelkerke NJD, Haas CN. (1999) Dose response models for infectious gastroenteritis. Risk Analysis; 19: 1251-1260.

Thornley C, Sneyd E. (2002) ESR Monthly Surveillance Report, July 2002. Porirua: ESR.

Todd EC. (1992) Foodborne disease in Canada - a 10 year summary from 1975 to 1984. Journal of Food Protection; 55: 123-132.

USDA FSIS. (1996) Nationwide Broiler Chicken Microbiological Baseline Data Collection Program. Washington, DC: U.S. Department of Agriculture, Food Safety and Inspection Service.

Wheeler JG, Sethi D, Cowden JM, Wall PG, Rodrigues LC, Tompkins DS, Hudson MJ, Roderick PJ. (1999) Study of infectious intestinal disease in England: rates in the community, presenting to general practice, and reported to national surveillance. British Medical Journal; 318: 1046-1050.

Whyte P, McGill K, Cowley D, Madden RH, Moran L, Scates P, Carroll C, O'Leary A, Fanning S, Collins JD, McNamara E, Moore JE, Cormican M. (2004) Occurrence of *Campylobacter* in retail foods in Ireland. International Journal of food Microbiology;95:111-118.

Whyte R, Hudson A, Morrison D, Burt P. (2001) Outbreak of campylobacteriosis from chicken liver pâté. New Zealand Journal of Environmental Health; 24: 9-10.

Whyte RJ and Hudson JA. (2004). Undercooked Chicken Livers as a Vehicle for Campylobacteriosis. FW 0411, ESR, Christchurch,

Wong TL, Whyte RJ, Hough AJ, Hudson JA (2004). Enumeration of *Campylobacter* and *Salmonella* on chicken packs. British Food Journal; 106: 651-662.

Wong TL, Hollis L, Cornelius A, Nicol C, Cook R, Hudson JA. (2006) Prevalence and numbers of *Campylobacter jejuni* (with sub-typing) and *C. coli* in retail uncooked meat samples. Journal of Food Protection – in press.

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