Ministry for Primary Industries
Manatū Ahu Matua

Risk profile: *Campylobacter jejuni/coli* in Poultry (whole and pieces)

MPI Technical Paper No: 2015/02

Prepared for the Ministry for Primary Industries
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ISBN No: 978-0-477-10546-0 (online)
ISSN No: 2253-3923 (online)

August 2013

New Zealand Government

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

Client report FW11065

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CAMPYLOBACTER JEJUNI/COLI
IN
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Prepared for the Ministry for Primary Industries
under project MRP/10/01 - Microbiological Risk Profiles,
as part of an overall contract for scientific services

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ACKNOWLEDGMENTS

We thank the Poultry Industry Association of New Zealand (PIANZ) and Michael Brooks (Chief Executive) in particular, for supplying information and data during the updating of this Risk Profile.

We also thank Dr Lucy Rivas of ESR for helpful comments on the draft.

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SUMMARY

The purpose of a Risk Profile is to provide 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.

The food/hazard combination addressed by this Risk Profile is *Campylobacter jejuni/coli* in poultry (whole and portions). This is an update of a Risk Profile published in 2007.

This Risk Profile has been commissioned in order to address the following specific risk management questions:

- What is the public health risk from *Campylobacter* in poultry (whole and portions) consumed in New Zealand?
- Has the risk of campylobacteriosis from the consumption of poultry (whole and portions) changed since the 2007 Risk Profile?

The literature on *Campylobacter* and poultry is extensive. The focus in this Risk Profile has been on studies that have been performed in New Zealand, and overseas studies that are informative about attribution and risk management interventions. In general, earlier data reported in the previous Risk Profile have not been repeated.

Poultry is a food frequently consumed by New Zealanders. Although the prevalence and concentration of *Campylobacter* on poultry in New Zealand have declined since interventions were introduced in 2006 – 2007, there is still a high probability (>50%) that poultry purchased by consumers will contain *Campylobacter*.

There is evidence that the prevalence and mean concentration of *Campylobacter* on poultry in New Zealand is significantly lower than in 2006. This reduction is linked to the interventions applied to the poultry supply and which are associated with an approximately 50% decline in the incidence of reported cases of campylobacteriosis. Nevertheless, the public health burden of this disease in 2011 is still a substantial component of the overall burden of foodborne enteric disease in New Zealand, based on disability adjusted life year (DALY) estimates. Campylobacteriosis is the most important foodborne bacterial enteric disease.

Two different approaches to attribution of *Campylobacter* infections in New Zealand have indicated that poultry as a source, and chicken consumption as a pathway, still represents the most important component of the epidemiology of the disease.

A summary of overseas studies and risk management interventions for *Campylobacter* in poultry is provided in Appendix 3. These studies highlight the difficulty of preventing the introduction of *Campylobacter* infection on broiler farms and the need to identify farm specific risk factors for control.

Increased chemical decontamination of carcasses during processing is an option, but a 2008 survey by the NZFSA found that there is consumer resistance to this approach.

In summary, the risk of campylobacteriosis from the consumption of poultry in New Zealand has declined considerably following risk management interventions by the Ministry for

Primary Industries and the poultry industry. However, poultry remains an important vehicle for infection, and further risk management is warranted.

The data gap identified in this Risk Profile is:

- Risk factors for infection of broilers with *Campylobacter* that are specific to particular regions and/or farms in New Zealand.

1 STATEMENT OF PURPOSE

The purpose of a Risk Profile is to provide information relevant to a food/hazard combination so that risk managers can make better informed decisions and, if necessary, take further action. Risk Profiles are part of the Risk Management Framework (RMF)[1] approach taken by the Ministry for Primary Industries (MPI) Food Safety. The Framework consists of a four step process, as shown in Figure 1.

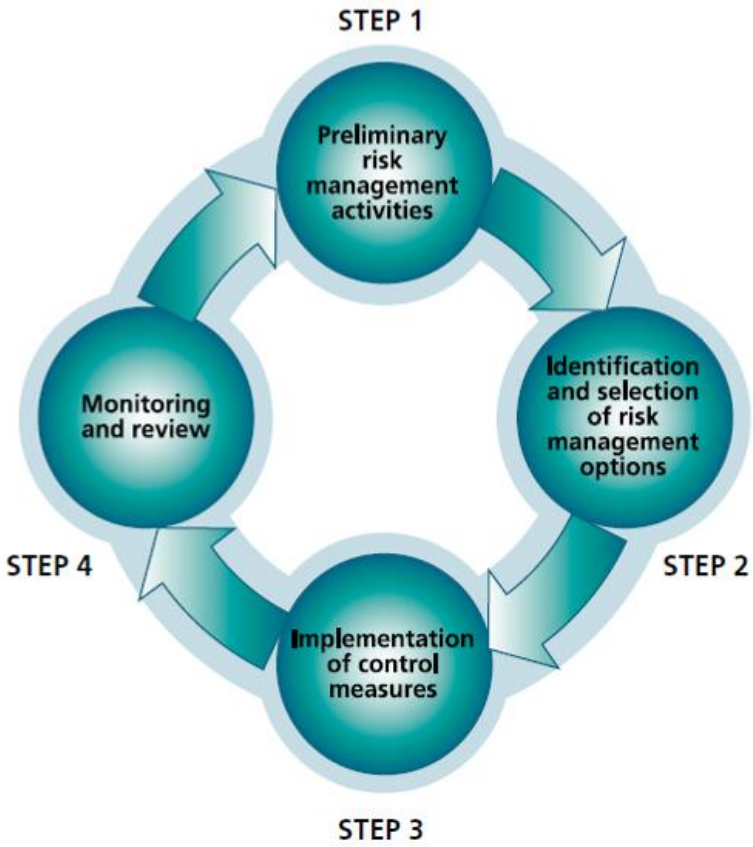


Figure 1: The four steps of the Risk Management Framework

This initial step in the RMF, Preliminary Risk Management Activities, includes a number of tasks:

- Identification of food safety issues
- Risk profiling
- Establishing broad risk management goals
- Deciding on the need for a risk assessment
- If needed, setting risk assessment policy and commissioning of the risk assessment
- Considering the results of the risk assessment

[1] http://www.foodsafety.govt.nz/elibrary/industry/RMF_full_document_-_11604_NZFSA_Risk_Management_Framework_3.1.pdf accessed 2 November 2011

- Ranking and prioritisation of the food safety issue for risk management action.

Risk profiling may be used directly by risk managers to guide identification and selection of risk management options, for example where:

- Rapid action is needed;
- There is sufficient scientific information for action;
- Embarking on a risk assessment is impractical.

1.1 Food/hazard Combination and Risk Management Questions

The food/hazard combination addressed by this Risk Profile is *Campylobacter jejuni/coli* in poultry (whole and portions). This is an update of a Risk Profile published in 2007 (Lake *et al.*, 2007c).

This Risk Profile has been commissioned in order to address the following specific risk management questions:

- What is the public health risk from *Campylobacter* in poultry (whole and portions) consumed in New Zealand?
- Has the risk of campylobacteriosis from the consumption of poultry (whole and portions) changed since the 2007 Risk Profile?

The literature on *Campylobacter* and poultry is extensive. The focus in this Risk Profile has been on studies that have been performed in New Zealand, and overseas studies that are informative about attribution and risk management interventions. In general, earlier data reported in the previous Risk Profile have not been repeated.

2 HAZARD AND FOOD

2.1 The Pathogen

The information in this section represents a summary of a microbiological data sheet relevant to this Risk Profile. These data sheets are prepared by ESR for a number of different foodborne pathogens as requested by MPI.[2] Additional information on the hazard and food are included in Appendix 1.

2.2 *Campylobacter*

Campylobacter spp. are non-sporulating, Gram-negative bacteria that appear as slender, spirally curved rods under the microscope. There are many species of *Campylobacter* but the evidence in New Zealand suggests that two species, *C. jejuni* and *C. coli*, are of major significance to public health. Other species, such as *C. upsaliensis*, *C. fetus*, *C. hyointestinalis* and *C. lari* have occasionally been reported as causing human illness but their significance in New Zealand is unknown as different isolation methods are required for these organisms.

The terms thermophilic or thermotolerant *Campylobacter* are often encountered in the literature, and include the species *C. jejuni*, *C. coli*, *C. lari* and *C. upsaliensis*. In this Profile, the term *Campylobacter* will only refer specifically to the two human pathogenic species *C. jejuni* subsp. *jejuni* and *C. coli*.

2.3 Sources of *Campylobacter*

Human: *Campylobacter* are not one of the organisms normally found in the human intestine. Faecal-oral person-to-person transmission is reportedly rare. A New Zealand regional study estimated that, based on reported contact of notified cases with gastroenteritis cases, person-to-person transmission may account for 1.7% (95th percentile confidence interval 0.5-5.0%) of notified cases (Lake *et al.*, 2011).

Animal: *Campylobacter* can be found in the intestinal tract of a wide variety of wild and domesticated warm-blooded animals which may or may not be symptomatic. *C. jejuni* is the dominant species in poultry while *C. coli* is usually the dominant species in pigs. Household pets have been implicated as risk factors for campylobacteriosis in case-control studies, although a New Zealand source attribution study concluded that less than 2% of human cases could be attributed cats and dogs (French *et al.*, 2011). Flies and other insects have been implicated as vectors for infection of poultry flocks (Hald *et al.*, 2004).

Wild or domesticated birds are a primary reservoir for *Campylobacter*. Poultry flock prevalence data for New Zealand from 2006-2009 indicate that up to 60% of flocks may be infected with *Campylobacter*[3]. Once the microorganism is introduced into a poultry flock it spreads rapidly whereby most birds in a flock are infected within a week. Microbiological

[2] A full set of the data sheets can be found at: http://www.foodsafety.govt.nz/science-risk/hazard-data-sheets/pathogen-data-sheets.htm accessed 2 November 2011

[3] http://www.foodsafety.govt.nz/elibrary/industry/caecal-testing-discussion-document/caecal-testing-review-and-options-assessment.pdf accessed 2 November 2011

surveys of lamb, sheep and dairy cattle faeces in New Zealand have found *Campylobacter* prevalence of up to 80% (Moriarty *et al.*, 2011; Moriarty *et al.*, 2008).

Food: Since *Campylobacter* are frequently found in livestock intestines, it is often found on meat and poultry at the abattoir and in the retail market.

Raw poultry is frequently contaminated by *Campylobacter*. A 2003/2004 microbiological survey found a prevalence of 89% in retail minced/diced chicken samples in New Zealand (Wong *et al.*, 2007b), and more recent data for 2005–2008 indicate a prevalence in retail poultry of 75% (French and Molecular Epidemiology and Veterinary Public Health Group, 2008). Following risk management initiatives, a survey in 2009 found the prevalence of *Campylobacter* in minced/diced retail chicken meat to be 70% (Wong and Hudson, 2011). Retail cooked chicken is rarely contaminated (0.07% based on a 1995 New Zealand survey). The prevalence of *Campylobacter* in minced/diced retail red meat in New Zealand was up to 10% (ranging from 3.5% in beef to 10% in meat from very young calves) in the 2003/2004 survey (Wong *et al.*, 2007c). *Campylobacter* has also been isolated from offals from various animals (Cornelius *et al.*, 2005; French and Molecular Epidemiology and Veterinary Public Health Group, 2008; Whyte *et al.*, 2006) and raw milk (Hudson, 1997) in New Zealand.

Environment: Water and soil can be easily contaminated from infected animals' excreta. *Campylobacter* can survive in cold water but numbers are reduced at temperatures above 10^{o}C and by ultraviolet radiation from sunlight. *Campylobacter* are present in water and sediments more frequently and at higher numbers in the winter months. These data are of interest because environmental survival appears to be opposite to the trend in the numbers of human cases, i.e. survival is poorer in the warmer months when the numbers of human infections are highest. Although surface waters are often contaminated with *Campylobacter* (McBride *et al.*, 2002), the types found are principally associated with wild birds and do not occur frequently amongst isolates from human cases (French *et al.*, 2011). A survey of New Zealand treated drinking water found a negligible prevalence of *Campylobacter* (Nokes *et al.*, 2004).

Transmission Routes: Reports of confirmed person-to-person transmission are rare, despite large (6-9 $\log_{10}$ CFU per g) microbial loading of faeces from infected individuals. Poultry, ruminant animals, pets (dogs/cats) and wild birds have all been evaluated as sources for *Campylobacter* types found in human cases in New Zealand, with poultry still the most important source despite a decline in attribution estimates from 2006 to 2010 (French *et al.*, 2010). Pathway modelling also attributes poultry food consumption as the most important pathway, with occupation, overseas travel, living in a rural environment, recreational water contact, and consumption of other foods also contributing to human infection and illness (Lake *et al.*, 2011).

2.4 Typing Methods

The terms "*subtyping*" or "*typing*" describes a test or assay which is able to distinguish isolates of a microbial species from each other. Subtyping tools can be valuable for:

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

The most commonly applied methods of typing of *Campylobacter* in New Zealand have been pulsed field gel electrophoresis (PFGE) and multi-locus sequence typing (MLST). Alternative methods are amplified fragment length polymorphism (AFLP) or short variable region (SVR) sequencing, based on the flagellin (*fla*) genes. Further details on these and other typing techniques are provided in Appendix 1.

2.5 The Food

2.5.1 Definitions

The specific foods considered by this Risk Profile are poultry and poultry products. Poultry includes chickens (*Gallus gallus*), turkeys and ducks that are commercially produced (i.e. not from flocks kept for non-commercial purposes or harvested from wild populations).

Poultry products include:

- Whole poultry and poultry pieces/portions (e.g. wings, drumsticks, breasts);
- Raw value-added poultry products, e.g. marinated or crumbed portions, stuffed whole birds, rolled breasts, frozen nuggets, sausages;
- Packaged ready-to-eat poultry products, e.g. cooked slices, smoked products;
- Ready-to-eat poultry products served by the food service industry.

The term "broiler" is often used for a chicken that is bred specifically for meat production. A "layer" bird is one that has been bred specifically for egg production. At the end of their lifetime, "end of lay" (EOL) birds may be processed for meat production, although currently there is little demand for poultry meat from this source and EOL birds are more likely to be gassed and rendered (PIANZ, personal communication, August 2013). Breeder birds (parent and grandparent stock for the chicks raised on broiler farms) may also be processed for meat production. Meat may also be recovered from EOL carcasses by some secondary processors and used as ingredients in a variety of processed products, including chicken sausages and chicken soup. As breeder birds reach the end of their productivity, they are culled and processed in the poultry companies' processing plants. Meat and carcass products are recovered from the carcasses and further processed into chicken products, for example chicken rolls, luncheon, sausages, chicken bacon and chicken nuggets. Once the meat is removed, the body frames are used for the recovery of mechanically separated meat (MSM). Wings from breeder birds also provide a source of large wings and nibbles for the retail market.

This Risk Profile excludes other avian species harvested for meat, such as goose, pigeon and ostrich. It also excludes chicken livers, which are covered as offal in a separate Risk Profile (Lake *et al.*, 2007a).

The water activity (a_w) of poultry meat is about 0.98 to 0.99. The pH of chicken breast muscle is 5.7 to 5.9, while that of leg muscle is 6.4 to 6.7. Both poultry muscle and skin are excellent substrates for the growth of a wide variety of microorganisms (ICMSF, 2005). The shelf life of raw poultry is quite short in comparison with other meats. Shelf lives of 7, 5 and 4 days at 4, 7 and 9^{o}C, respectively have been reported in a Middle Eastern study using an end point of approximately 7.2 $\log_{10}$ CFU spoilage bacteria per ml of half-carcass rinse (Abu Ruwaida *et al.*, 1996). This end point was accompanied by changes in organoleptic

characteristics, which were considered to make the chicken unacceptable to consumers. However, longer shelf lives for storage at 3.5-4°C have also been reported in an Australian (Sexton *et al.*, 2006). These were 12-13 days for untreated carcasses, and 14 days for carcasses treated with acidified sodium chlorite, and these times are considered more realistic by the New Zealand poultry industry (PIANZ, personal communication, August 2013).

2.5.2 The food supply in New Zealand: Poultry and poultry products

The Poultry Industry Association of New Zealand Incorporated (PIANZ) represents the interests of poultry processing and breeding companies in New Zealand and has a role in commenting on poultry standards. Membership is voluntary, but the following 11 producers of almost all of New Zealand's poultry meat choose to be represented by PIANZ:[4]

- Tegel Foods Ltd.;
- Inghams Enterprises (NZ) Pty Ltd.;
- PH van den Brink Ltd.;
- Turk's Poultry;
- A & J Heron Holdings Ltd.;
- Aviagen;
- Bromley Park Hatcheries;
- Canter Valley Processors;
- Crozier's Turkeys Ltd.;
- Eastherbrook Farm Ltd.; and
- Quack a duck.

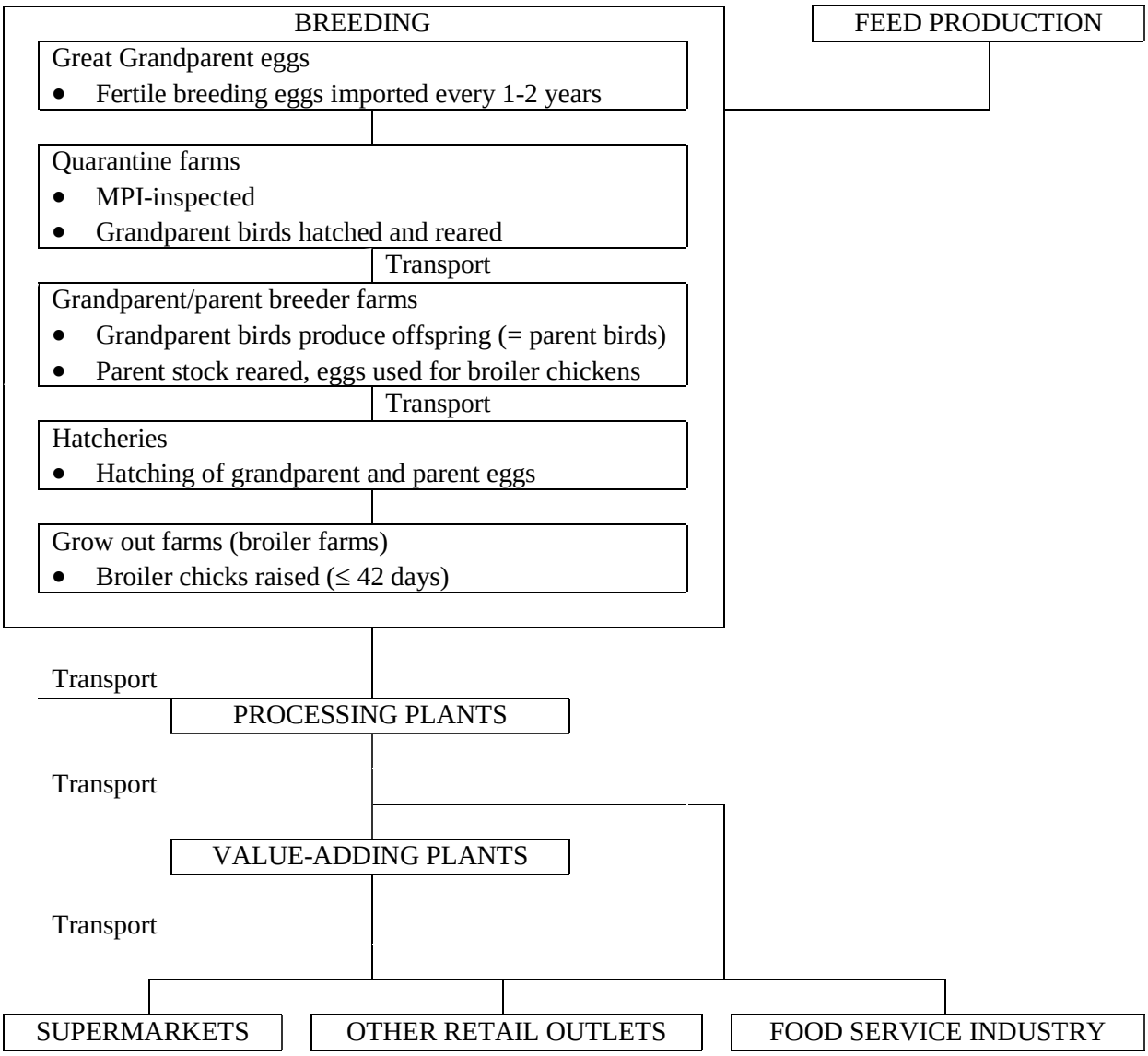
Larger companies (e.g. Tegel Foods and Inghams Enterprises (NZ) Pty Ltd.) are vertically integrated, i.e. manage all aspects of poultry meat production within their separate companies from feed production to breeding, processing and value-adding.

Tegel Foods Ltd., Inghams Enterprises (NZ) Pty Ltd., and PH van den Brink Ltd., together have the greatest market share in New Zealand. There are also a number of small or niche poultry producers who are not members of PIANZ (e.g. Heuvels, Henry's Poultry and Mahurangi Ducklings).

Figure 2 outlines the product flow within the poultry industry in New Zealand.

[4] As listed at http://www.pianz.org.nz accessed 2 November 2011.

Figure 2: Generic flow of product within the poultry industry in New Zealand



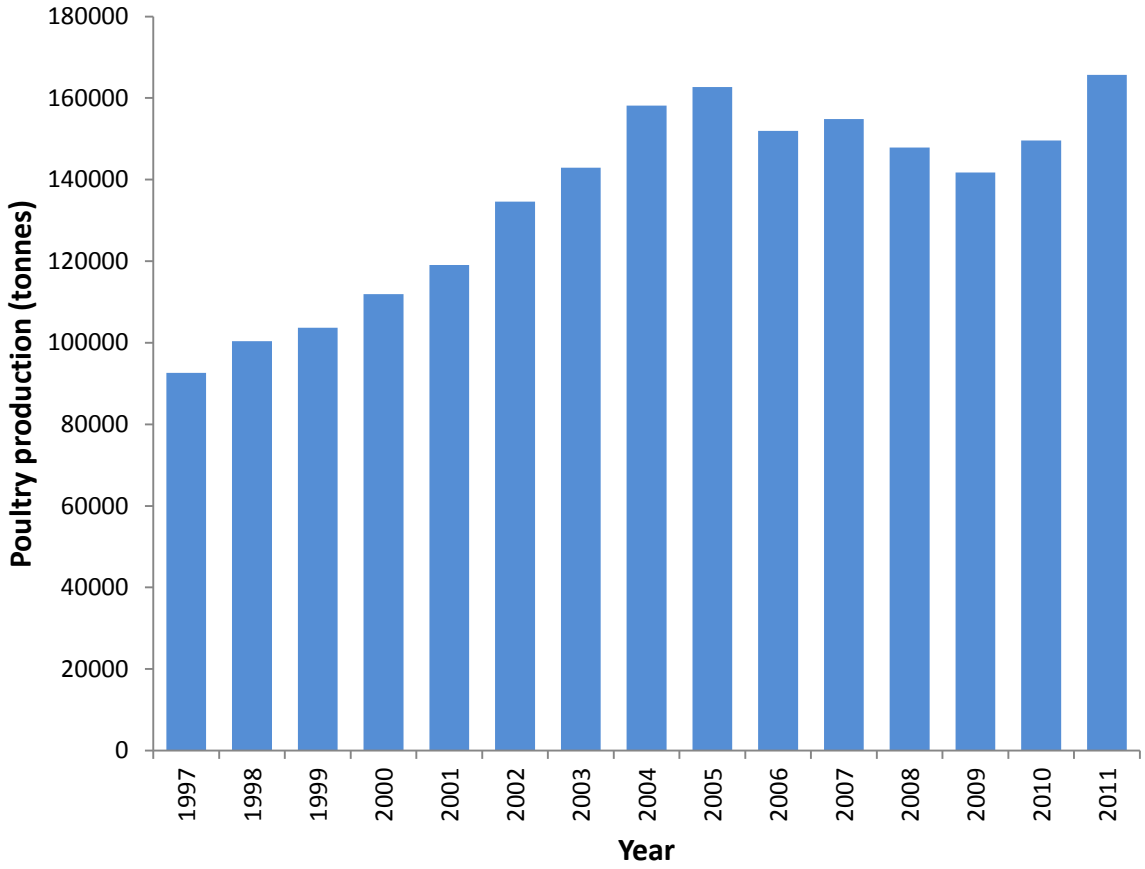
Reproduced from (Lake et al., 2005)

2.5.2.1 Production

Data from PIANZ indicates that there were approximately 166,000 tonnes of poultry meat produced in 2011.[5] Broiler chicken meat production has been steady at between 140,000 and 160,000 tonnes per year since 2003. Production figures for the period 1997-2011 are shown in Figure 3.

[5] See http://www.pianz.org.nz/industry-information/industry-statistics accessed 15 March 2013

Figure 3: New Zealand poultry meat production, 1997-2011



Data in this figure come from the PIANZ website[6]

The majority of broilers produced in New Zealand are barn-raised. However, free-range raised broilers now account for approximately 5% of broiler production, with this percentage likely to increase (PIANZ, personal communication, August 2013). An overview of broiler farming in New Zealand assembled in 2006 using information from the four largest companies found that there were 130 farms with a total of approximately 500 sheds (Hudson *et al.*, 2008). Approximately half of the farms held between 50,000-100,000 birds per growing cycle, with a further third holding between 100,000 and 200,000 birds. A few farms held more than 200,000 birds per growing cycle. A survey of 60 New Zealand poultry farms reported an average shed capacity of approximately 25,000 birds (Lake *et al.*, 2008b).

In 2006, approximately 40% of poultry was sold as whole carcasses (PIANZ, personal communication, November 2006, quoted in the previous Risk Profile). In the year ending June 2006, approximately 98% of poultry consumption was chicken meat, with turkey, duck, and roasting fowl making up the remaining 2% (PIANZ, personal communication, November 2006). A more recent report puts the proportion of turkey, duck and roasting fowl as 5% (French and Molecular Epidemiology and Veterinary Public Health Group, 2009).

It is estimated that approximately half of chicken production in New Zealand is purchased and consumed by domestic households, while the other half enters the food service industry

[6] http://www.pianz.org.nz/pianz/wp-content/uploads/2012/10/NZ-Poultry-Meat-Production-Statistics-1997-to-2011.pdf Accessed 15 March 2013

(including fast food outlets) (PIANZ, personal communication, August 2013). In 2006 approximately 79% of chicken was sold as fresh product (i.e. chilled) and 21% frozen. This represents a considerable change over the previous ten years; 60% of product was sold frozen in 1995 (PIANZ, personal communication, November 2006).

2.5.2.2 New Zealand exports

New Zealand exports only a small proportion of poultry production. The approximately 4,000 tonnes of chicken meat exported in the year ending March 2011 represented 2.7% of the approximately 150,000 tonnes total production. However, the amount of poultry production exported is increasing, with the total amount of poultry (mainly chicken and some turkey) exported increasing from approximately 4,000 tonnes in the 2009 year to approximately 8,000 tonnes in the 2012 year.[7] Poultry is exported mainly to Australia and the Pacific Islands.

2.5.2.3 New Zealand imports

Raw chicken is currently not permitted for import into New Zealand and there are import health standards in place for the following:

- Importing specified cooked poultry meat products for human consumption from Australia (meapouic.aus); and
- Importing turkey meat and meat products from approved countries (pouturic.gen).[8]

These standards require the poultry products to be cooked, although raw turkey products may be imported if the importer can demonstrate disease-free status.

According to data released by Statistics New Zealand, in the year ending March 2011, the three largest imported poultry products by weight were:

- Chicken preparations preserved in airtight containers or jars (not meat pastes or combined with vegetables or other substances): 367 tonnes;
- Poultry preparations preserved in airtight cans or jars (not turkey, livers or homogenised preparations and prepared without other food substances): 186 tonnes; and
- Chicken preparations preserved in airtight containers or jars (in combination with vegetables or other food substances) or meat pastes: 168 tonnes.

Other imported poultry products included cooked, shelf stable sausages and liver products.

Thailand is the major source of imported poultry products, followed by the United States of America (US) and Australia.

[7] http://www.stats.govt.nz/infoshare/TradeVariables.aspx?DataType=TEX accessed 16 August 2013
[8] Both these standards are available from http://www.biosecurity.govt.nz/regs/imports/ihs accessed 2 November 2011

2.5.3 Behaviour of *Campylobacter* in poultry: on the farm

The internal temperature of chickens is approximately 40°C, which is an ideal temperature for the growth of *Campylobacter*. The number of cells required to infect chickens is low; an estimated dose of approximately 500 CFU is required to provide a 50% probability of infection (Line *et al.*, 2008). *Campylobacter* spp. populations can increase rapidly in the gut of colonized birds, and therefore faecal droppings can often contain large numbers of *Campylobacter* cells. The numbers of *Campylobacter* in caecal contents have been found to vary between 1.7 to 8.6 $\log_{10}$ CFU per g (Hansson *et al.*, 2010b). *Campylobacter* numbers in the caeca and external contamination in the feathers have been shown to be correlated (Cason *et al.*, 2007). This contaminated faecal material is therefore available to serve as a high-level inoculum source for other birds in the flock. A study of the transmission of *Campylobacter* in commercial broiler flocks has estimated that a colonised flock of 20,000 broilers would have an increase in within-flock prevalence to 95% within 4.4 - 7.2 days after colonisation of the first broiler (Van Gerwe *et al.*, 2009).

The types of *Campylobacter* present in an infected flock appear to be dynamic. A German study found that while multiple genotypes could occur in an infected flock, a single type was predominant at any one time. A succession of dominant AFLP genotypes in individual flocks was identified through weekly testing through the one year period of the study (Alter *et al.*, 2011). Shifts in the dominant strain of *Campylobacter* in broiler flocks during the grow-out period have been observed in other studies (see Appendix 1).

Reviews of the scientific literature concerning on-farm factors affecting the introduction of *Campylobacter* into broiler flocks indicate that multiple sources and risk factors are involved (Hudson *et al.*, 2008; Newell *et al.*, 2008; Newell and Fearnley, 2003). These include:

- Workers clothing and equipment (pallets, crates, vehicle beds and wheels, boots);
- Level of cleaning and disinfection between flocks;
- Feed and water;
- Flies and other insects;
- Vertebrate pests;
- Birds, other livestock;
- Partial depopulation (thinning); and,
- Transport and crate contamination.

A summary of more recent on-farm studies is provided in Appendix 1. A survey by poultry veterinarians of 60 of the approximately 160 broiler farms in New Zealand found that in general, many aspects of biosecurity were good (Lake *et al.*, 2008b) (see Section 5.2.2 for further details). The survey did identify some areas where improvements could be made but it was conducted prior to the release of the Poultry Industry Biosecurity Manual (August 2007) so improvements since then were not captured.

2.5.4 Behaviour of *Campylobacter* in poultry: primary and secondary processing

The primary processing of poultry in New Zealand follows a general sequence (Lake *et al.*, 2007b):

- Receiving
- Killing (electrical stunning followed by cutting of the carotid artery)

- Bleeding
- Scalding
- Defeathering
- Washing
- Eviscerating (viscera removal and washing)
- Chilling
- Weighing, grading and packaging
- Shipping

A more detailed and generalised summary of the primary production process can be found in the Codex *Guidelines for the Control of Campylobacter and Salmonella in Chicken Meat*, the development of which was jointly led by New Zealand and Sweden (Codex, 2011).

A systematic review of the changes in *Campylobacter* prevalence on carcasses during poultry processing has been published (Guerin *et al.*, 2010). Studies that sampled carcasses before and after scalding or chilling, or both, showed that the prevalence of *Campylobacter* generally decreased immediately after these process stages (scalding: 20.0 to 40.0% decrease; chilling: 100.0% decrease to 26.6% increase). The prevalence of *Campylobacter* increased on carcasses following defeathering (10.0 to 72.0%) and evisceration (15.0%). The change in prevalence of *Campylobacter* after washing was inconsistent among studies (23.0% decrease to 13.3% increase). Eleven studies reported the concentration of *Campylobacter*, as well as, or instead of, the prevalence. Studies that sampled carcasses before and after specific stages of processing showed that the concentration of *Campylobacter* decreased after scalding, evisceration, washing and chilling, and increased after defeathering.

Defeathering is a major source of contamination with *Campylobacter* due to faecal leakage from the cloaca, while evisceration may also contaminate the carcass if the intestines are cut or broken during the process (ICMSF, 2005). The diversity of strain types found on carcasses has been shown to be greater following defeathering compared to other processing steps, supporting the importance of this step in contributing to carcass contamination with *Campylobacter* (Nielsen *et al.*, 2006).

Similar effects of individual processing steps have been found in a study of a turkey processing line (Alter *et al.*, 2005).

New Zealand processors use a scalding temperature of between 56 and 58°C for approximately 2 minutes (i.e. "hard scald") and immersion chilling (Lake *et al.*, 2007b). Spray washes and chilling water may be chlorinated to control microbiological contamination onto the carcasses.

A summary of more recent studies of processing is given in Appendix 1.

2.5.4.1 Secondary processing

Secondary processing refers to additional processing, including portioning and cooking, of poultry after the whole carcass stage. Products include:

- Portions for fast food outlets
- Portions for retail sale

- Portions with added ingredients for home heating
- Smoked carcasses
- Chicken nuggets (deep fried)
- Chicken sausages
- Chicken luncheon rolls
- Shredded chicken or formed patties (often for foodservice sector)
- Chicken bacon

Whole or individual parts of birds may be packaged raw for direct sale. Poultry producers in New Zealand and some supermarkets have introduced the use of leak proof packaging, intended to eliminate the risk of leaking drip fluid from these products contaminating the retail environment. Provided the products are properly handled by consumers, this provides the potential for preventing cross contamination in the home. The older procedure of packing chicken portions for retail sale on trays wrapped in stretch wrap or cling film continues, but at a decreasing frequency (PIANZ, personal communication, August 2013).

Most frozen poultry is produced by packing the product into plastic bags clipped at the end and which are then frozen in high-velocity freezers. Before freezing, poultry may be injected with various salts, flavourings and oils in order to increase the juiciness and tenderness of the meat (PIANZ, personal communication).

A study of secondary processing by the New Zealand poultry industry reported that at the consumer level, the percentages of fresh, frozen, and precooked poultry consumption via domestic or foodservice channels was approximately 63%, 23% and 14% respectively (Lake *et al.*, 2008a).

The inclusion of chicken skin in processed products such as sausages or burgers has been found to increase the probability (2.2 times) that the raw product will contain *Campylobacter* (Sampers *et al.*, 2008). This finding is consistent with experiments that quantified the survival of *Campylobacter* on poultry skin compared to meat under differing storage conditions (Davis and Conner, 2007). Meat and skin samples were packaged in polystyrene trays, covered with film, and then subjected to one of several refrigerated (4ºC) and frozen (-3ºC) storage condition regimes. Populations of surviving *Campylobacter* were not affected by storage conditions, but surviving *Campylobacter* populations were affected by sample type (skin vs. meat). *Campylobacter*, in the absence of competing microorganisms, survived well on poultry skin and meat at the temperatures tested, but in all experiments, higher *Campylobacter* populations were established on the inoculated skin compared to the inoculated meat. These populations remained consistently 0.4 to 0.9 $\log_{10}$ CFU per g higher on skin compared to those recovered from the meat samples. It was suggested that poultry skin topography may have contributed to the variation in the *Campylobacter* numbers observed between the two sample types (although higher fat content in skin may also play a role).

However, it should be noted that a study of *Campylobacter* survival under frozen conditions (-20ºC) found better survival on cut muscle and skinned muscle, than on skin (Ritz *et al.*, 2007). It is uncertain whether the contradictory results found in these two studies are due to different freezing temperatures used or some other experimental variable.

C. jejuni was the most commonly found species in a long term (1997 to 2003) survey of a variety of chicken products from slaughterhouses, production plants and retail in Belgium (Ghafir *et al.*, 2007). Unusually this survey also tested for a range of other *Campylobacter* species and related bacteria. The prevalences found were: *C. jejuni* (86.8%), *C. coli* (10.5%), *C. lari* (0.8%), *C. fetus* (0.4%) and *C. upsaliensis* (0.0%). This species distribution is consistent with the predominance of *C. jejuni* in human cases, although testing methods for analysis of human faecal samples may not be optimal for detecting species other than *C. jejuni/coli*.

A three-year study in the Manawatu region of New Zealand found similar results, with 91.4% of poultry *Campylobacter* culture positive samples being confirmed as *C. jejuni* (French and Molecular Epidemiology and Veterinary Public Health Group, 2008). In the same study, 88.5% of human *Campylobacter* positive cultures were found to be *C. jejuni.*

2.5.5 Behaviour of *Campylobacter* in poultry: preparation and cooking

In a New Zealand consumer survey, the majority of poultry (62.9%) was purchased fresh (rather than frozen), and most consumers (94.4%) claimed that the time taken from food selection to reaching their home was one hour or less (Gilbert *et al.*, 2007). Approximately 64% of poultry purchased would be frozen once the consumer got it to their home.

New Zealand studies on the impact of freezing on survival of *Campylobacter* have consistently shown that freezing decreases *Campylobacter* concentrations of chicken under a range of circumstances (McIntyre, 2009). Skin-on chicken breast portions inoculated with two strains of *Campylobacter* were frozen to a defined internal temperature of -12°C followed by frozen storage at -12°C for up to 73 days. Mean *C. jejuni* populations declined by approximately 1.2 to 2.2 $\log_{10}$ CFU over a 1 week storage period, while reductions of 3.3 and 4.1 $\log_{10}$ CFU were achieved after 34 and 73 days of frozen storage, respectively. These mean reductions were of similar magnitude to those determined previously under both simulated domestic and commercial conditions, and suggest that the longer the frozen storage period, the better the pathogen reduction achieved.

Commercial freezing (-30°C), commercial frozen storage (-21°C) for two weeks and domestic storage (-18°C) for a further eight weeks produced significant but variable reductions in *C. jejuni* numbers for both strains, with reductions most rapid during the 14 days of commercial frozen storage (McIntyre, 2008). Overall, mean *C. jejuni* populations declined by approximately 1.8 to 3.5 $\log_{10}$ CFU over a 1 to 6 week storage period following commercial freezing, but were not completely eliminated. Under the specific conditions investigated, commercial freezing and short term storage (up to 28 days) produced significantly greater mean *C. jejuni* reductions versus domestic freezing. Similar but larger reductions were achieved by both processes over a longer storage period (up to 70 days), suggesting that frozen chicken should ideally be stored for at least 4 weeks to achieve maximum pathogen reductions.

A New Zealand study of so-called "crust freezing" inoculated a cocktail of three *C. jejuni* isolates onto the skin of chicken portions, which were chilled to –2 or –10ºC under two different cooling profiles (Whyte *et al.*, 2005). The final count on chicken portions chilled to –2°C did not differ from the pre-cooling count. When chilled to –10°C an approximate 1 $\log_{10}$ difference in counts could be measured, with the most likely reason being the time for which the samples were frozen (around 19 hours compared to 4 hours at –2°C).

The times and temperatures during transportation to home by consumers of purchased poultry products have been examined in a New Zealand study (Gilbert *et al.*, 2006). The increases in temperature for fresh poultry observed during the average transportation times were not sufficient to reach the minimum growth temperature of *Campylobacter* spp.

The Codex *Guidelines for the control of Campylobacter and Salmonella in chicken meat* recommends that "washing of raw chicken in the kitchen should be discouraged so as to minimise the possibility of contamination of other foods and surfaces that come in contact with food and humans. Where deemed necessary washing of raw chicken carcasses and/or chicken meat, should be carried out in a manner which minimises the possibility of contamination of other foods and surfaces that come in contact with other foods and humans" (Codex, 2011).

The D values[9] of *Campylobacter* (e.g. D value at 60°C = 0.2-0.3 minutes) indicate that normal cooking times and temperatures should rapidly eliminate the organism. Cells of *Campylobacter* are usually located on the surface of the poultry meat which contributes to greater heat inactivation of the microorganism during cooking (Luber and Bartelt, 2007; Scherer *et al.*, 2006). Therefore exposure of the microorganism through undercooking is less probable than cross contamination from poultry to surfaces such as hands, or other foods which are not subsequently cooked before consumption (Humphrey *et al.*, 2001). A review of published risk assessments supported this conclusion (Luber, 2009). Cross contamination may be particularly important in the context of barbecues, as shown by a reported outbreak in Germany where the host of the barbecue who handled the chicken became ill, despite not eating the chicken (Allerberger *et al.*, 2003).

Further information on studies of cross contamination and survival in domestic kitchens is provided in Appendix 1.

2.6 Exposure Assessment

2.6.1 *Campylobacter* on poultry: broiler flocks

Testing for *Campylobacter* on poultry in New Zealand was introduced in 2007 under the National Microbiological Database (NMD) specifications.[10] One part of the testing programme involved caecal sampling of birds at the primary processing plant. From 30 March 2007, all broiler primary processing premises were required to test a pooled sample of 10 caecal samples from each cut (i.e. batch of birds) of each shed being processed. Summary results from this testing programme were included in a 2009 document reviewing the need for continued caecal testing.[11]

These results indicated that over the 21 month period of testing, the prevalence of *Campylobacter* across all cuts was approximately 60-80% with no temporal trend apparent.

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

[10] http://www.foodsafety.govt.nz/elibrary/industry/animal-products-national-nmd/schedule-2011.pdf accessed 2 November 2011

[11] http://www.foodsafety.govt.nz/elibrary/industry/caecal-testing-discussion-document/caecal-testing-review-and-options-assessment.pdf accessed 2 November 2011

The prevalence in first cuts was lower (30-60%), which is to be expected given that the risk of flock infection is increased by the taking of cuts from sheds. The caecal sampling programme was discontinued in July 2009 following a review by MPI (then the Ministry of Agriculture and Forestry – MAF).

A study to produce quantitative and qualitative data for *Campylobacter* on exsanguinated broiler chickens sampled prior to scalding in four New Zealand poultry processing plants has been reported (Wong, 2006). A total of 200 birds (50 from each plant) from 39 flocks supplied by 30 farms were sampled over 41 consecutive weeks (April 2005 to February 2006). Whole bird rinsates were tested for presence and numbers of *Campylobacter*, while caecal swabs were tested only for the presence of this pathogen. *Campylobacter* spp. were isolated from 100% of birds with counts ranging from 1.53×10^2 to 2.90×10^9 CFU per bird. Caecal swab cultures from 35 flocks of birds were positive for *Campylobacter*, giving a flock prevalence of 89.7%.

A survey of *Campylobacter* in ducks and turkeys obtained samples from the three major producers in New Zealand (Wong, 2010). Caecal samples from 10 turkeys from each cut were pooled into single samples. Of the turkey cuts tested, 39/40 were positive for *Campylobacter* spp. (19 *C. jejuni* only, 16 *C. jejuni* and *C. coli*, and 4 *C. coli* only). The ducks were sampled as pooled samples from the caeca of 5 birds from each cut. The prevalence of *Campylobacter* was 28/28 (100%), and all samples contained *C. jejuni* only.

Caecal samples were tested from 16 flocks of breeder birds, with 13 flocks (81.3%) positive for *Campylobacter* spp. and three flocks negative for *Campylobacter* spp. (Wong and Chung, 2010). *Campylobacter* spp. were isolated from the caecal contents of EOL birds in 11 out of 13 (84.6%) flocks screened in the same study.

This Risk Profile does not include non-commercially produced poultry. However for context purposes, a New Zealand report has described the prevalence of *Campylobacter* in domestic "backyard" poultry in Canterbury (Anderson *et al.*, 2012). Faecal samples from 35 flocks were found to have an overall prevalence of 86% for *Campylobacter* (*C. jejuni* alone 57%, *C. coli* alone 6%, both *C. jejuni* and *C. coli* 23%).

2.6.2 *Campylobacter* on poultry: carcasses

Another part of the NMD testing programme for *Campylobacter* on poultry involves carcass rinse sampling at the end of primary processing. Carcass sampling is conducted by standard throughput premises on each processing day (enumeration of three carcass samples) while very low throughput (VLT) premises sample at least three carcasses each week or part week of processing.[12] VLT poultry premises are defined as those that slaughter product from one million (1,000,000) birds or fewer per annum. The testing method involves spread plating of a volume of rinsate and has a detection limit of 200 CFU per carcass.

Summary results from this programme have been included in the MPI *Campylobacter* Risk Management Strategy documents[13] and are presented in Table 1 along with subsequent

[12] http://www.foodsafety.govt.nz/elibrary/industry/animal-products-national-nmd/amdt-notice-2011.pdf accessed 2 November 2011

[13] http://www.foodsafety.govt.nz/industry/general/foodborne-illness/campylobacter/strategy.htm accessed 2 November 2011

results (Gail Duncan, MPI, personal communication). These results are for thermophilic *Campylobacter* (i.e. *C. jejuni* and *C. coli*). The range of results for numbers of *Campylobacter* on the NMD carcasses is not available in the MPI *Campylobacter* Risk Management Strategy documents. A Swedish study has found that the within-flock variation in *Campylobacter* numbers was up to 3.2 $\log_{10}$ CFU per ml of carcass rinse sample (Hansson *et al.*, 2010b).

Table 1: NMD carcass sampling and enumeration results for *Campylobacter* 2007 - 2012

Quarter and year	Number of carcasses tested	Prevalence (%)	Mean log count, all samples
Q2 2007	890	57	3.07
Q3 2007	936	53.8	3.06
Q4 2007	916	45.1	2.75
Q1 2008	1309	45	2.70
Q2 2008	1528	30.6	2.41
Q3 2008	1587	32.8	2.45
Q4 2008	1609	44	2.70
Q1 2009	1582	46.5	2.58
Q2 2009	1489	31.1	2.41
Q3 2009	1489	24.5	2.31
Q4 2009	1421	29.8	2.35
Q1 2010	1440	42.2	2.47
Q2 2010	1456	36.1	2.42
Q3 2010	1467	35.1	2.43
Q4 2010	1443	42.1	2.49
Q1 2011	1459	37.0	2.44
Q2 2011	1590	36.9	2.45
Q3 2011	1635	37.4	2.45
Q4 2011	1542	40.3	2.48
Q1 2012	1555	37.1	2.46
Q2 2012	1563	29.2	2.34
Q3 2012	1631	27.2	2.32
Q4 2012	1659	37.7	2.47

*Samples where *Campylobacter* was not detected were given a value of 2.00 $\log_{10}$ CFU/carcass.

A study has been conducted to evaluate the prevalence of *Campylobacter* in rinsates reported as 'Not Detected (ND)' (Lake, 2009). Overall, 23 rinsate samples reported as ND (<200 CFU) were tested; of these 8 (34.8%) were found to be positive following enrichment. Although 34.8% of the ND rinsate samples in this study were positive, it was acknowledged that such samples (with up to 200 CFU per carcass) would contribute a very small part of the overall risk to human health from *Campylobacter* on poultry.

Mechanically separated meat (MSM) is produced from carcass frames following the removal of portions and breast meat from the carcasses. These products are used to manufacture chicken sausages and, to a lesser extent, chicken nuggets (PIANZ, personal communication, August 2013). A study of the *Campylobacter* prevalence in MSM from February – mid-August, 2010 found that of a total of 145 MSM samples from three different processing plants combined, *Campylobacter* was countable in 87%, 66% and 33% of samples from the three processors (Wong *et al.*, 2011). The distribution of bacteria varied with each processor. The median counts (5th to 95th percentile) for *Campylobacter* in MSM at the three processors were 1.74 (ND - 3.17) $\log_{10}$ CFU per g, 1.18 (ND - 2.55) $\log_{10}$ CFU per g, and ND (ND - 2.08) $\log_{10}$ CFU per g.

2.6.3 *Campylobacter* on poultry: retail

A national retail survey was performed to determine the prevalence of *Campylobacter* in minced and diced meat purchased from supermarkets and butchers during 2003-2004 (Wong *et al.*, 2007c). A second and similar microbiological survey was conducted in 2009 for retail poultry (Wong and Hudson, 2011) with an objective to obtain comparative data for the 2003-2004 survey. The 2003-2004 quantitative data were reworked to provide comparative numbers. One hundred and seventy-five uncooked, chilled retail chicken samples (minced, diced, or cut into strips) were purchased fortnightly between April and July 2009 from various retail outlets in the five main cities in New Zealand (Auckland, Hamilton, Wellington, Christchurch and Dunedin).

The results of both surveys are shown in Table 2. It was concluded that *Campylobacter* spp. prevalence in uncooked retail chicken meats in 2009 was significantly lower than in 2003-2004 ($P<0.001$). When the sampling period and duration of sampling were taken into account, the reduction in prevalence (16%) of *Campylobacter* spp. between the two surveys remained significant ($P = 0.002$). In addition, a decrease in the distribution of concentration data in *Campylobacter*-positive samples was measured in the 2009 survey. The percentage of counts in the higher ranges in the 2009 survey (>1.0 $\log_{10}$ CFU per g) is lower than the percentage of counts obtained in 2003-2004 survey.

Table 2: National retail survey of *Campylobacter* in minced poultry July 2003 to June 2004

Period	No. samples tested	Total Number positive (%, 95% Confidence Interval)	*C. jejuni*	*C. jejuni* & *C. coli*	*C. coli*	% of samples with counts in positive samples
July 2003 – June 2004	230	205 (89.1, 75.9 – 93.1)	199	5	1	81%: -1.4 – 1.0 $\log_{10}$ CFU/g 9%: >1.0 $\log_{10}$ CFU/g
April – July 2009	175	122 (69.7, 62.3 - 76.4)	122	0	0	67%: -1.4 – 1.0 log10 CFU/g 2%: >1.0 $\log_{10}$ CFU/g

Data from whole carcass testing have been provided by a sampling programme conducted as part of a sentinel site study in the Manawatu (French *et al.*, 2008). Fresh whole poultry carcasses from retail outlets in Palmerston North were sampled from 2005 to February 2008 and had a prevalence of 80.8% presumptive *Campylobacter*, with 75.3% confirmed *C. jejuni*. These data however were affected by the inclusion of samples taken prior to poultry industry

interventions beginning in 2006. Results for all of 2008 were 50% confirmed *C. jejuni* (Professor Nigel French, Massey University, personal communication, 2010).

The numbers of *Campylobacter* on the carcasses tested in this study were also enumerated from rinsates and reported to range between 2 – 4 $\log_{10}$ CFU per carcass, apart from samples from a single manufacturer that provided rinsates containing greater than 4 $\log_{10}$ CFU per carcass during the first quarter of the project period.

A survey of retail whole carcasses from all major processers sampled in October – November 2007 found a prevalence of *Campylobacter* spp. in rinsates from 73/163 carcasses (44.8%, 95% CI 37.0% - 52.8%) (Chrystal *et al.*, 2008). This prevalence is lower than found in other surveys, probably because the testing involved spread plating of rinsates and the detection limit was 400 (2.6 $\log_{10}$) CFU per carcass. The mean count of *Campylobacter* from positive carcasses was 3.60 $\log_{10}$ CFU per carcass (standard deviation 0.885 $\log_{10}$ CFU per carcass).

Another study of poultry meat samples sourced from suppliers (in a ready for sale form) or supermarkets in the Manawatu between December 2008 and May 2009 determined the prevalence of presumptive *Campylobacter* spp. in end of lay chickens, turkeys and ducks (French and Molecular Epidemiology and Veterinary Public Health Group, 2009). The method involved enrichment. The prevalences reported were:

- End of lay carcasses (48/48, 100%)
- Duck (73/75, 97%)
- Turkey (52/63, 83%)

Another survey of ducks and turkeys from major manufacturers in the first half of 2009 used an enumeration method for *Campylobacter* in rinsates from processed carcasses (Wong, 2010). These carcasses were taken at the end of primary processing. Two hundred samples of turkey rinsates were enumerated for *Campylobacter* spp. Thirty-four percent of these samples contained < 2.48 $\log_{10}$ CFU *Campylobacter* per carcass. This result, which is below the limit of detection of the method, would normally be reported as "Not Detected" under the broiler NMD reporting, and so the putative prevalence was 66%. It should be noted that due to the larger carcass size, 600 ml rinsates were used for turkeys, compared to the usual 400 ml used in the broiler NMD programme. Fifty percent of counts were between 2.48–4.0 $\log_{10}$ CFU per carcass. *Campylobacter* spp. were enumerated at levels between 4.1–6.0 $\log_{10}$ CFU per carcass in 14.5% of rinsates. Only 1.5% or three of the samples were found to contain *Campylobacter* spp. at > 6.0 $\log_{10}$ CFU per carcass.

A total of 135 duck samples were enumerated in the study. Twenty-seven (20%) of the samples had *Campylobacter* spp. counts of < 2.30 $\log_{10}$ CFU per carcass, and so the putative prevalence was 80%. Of the 16% of samples that had *Campylobacter* spp. counts exceeding 4.0 $\log_{10}$ CFU per carcass, only one duck rinsate exceeded 5.0 $\log_{10}$ CFU per carcass.

The lower prevalences for turkey and duck carcasses found in the second survey probably reflects the absence of a separate enrichment procedure to provide prevalence data.

2.6.4 *Campylobacter* on poultry: packaging and ready-to-eat products

In early 2002, ESR conducted a survey of three hundred retail packs of fresh, chilled poultry products from fifteen supermarkets in the Christchurch area (Wong *et al.*, 2004). The

objective of the study was to determine the prevalence of *Campylobacter* on the exterior of packs. The results for the New Zealand study were:

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

Following publication of this study, poultry companies and some supermarkets in New Zealand have introduced leakproof packaging for retail product. A survey to examine the numbers of *Campylobacter* in the liquid within such packaging was commissioned by NZFSA (now MPI) (Wong, 2008). Retail packs of leak-proof packaged poultry products were sampled in Auckland and Christchurch over a four week period in October 2007. The products included thirty whole birds from three brands, twenty five trays of chicken portions packed in leak-proof packaging (five samples each of skinless breasts, skin-on breasts, thighs, drums and nibbles) and five pottles of chicken livers. The volume of drip recovered from the leak-proof packaged whole chickens ranged from 1.6 ml to 106.4 ml. In the testing 1 ml of drip was spread over three plates (dilutions of the drip were also made to facilitate enumeration). Twenty five of the drip samples obtained from whole birds were positive for *Campylobacter* (25/30, 83%). *C. coli* was identified from one bird, while all other isolates were identified as *C. jejuni*.

Campylobacter counts in positive drip samples from whole birds ranged from <0.30 – 4.26 $\log_{10}$ CFU per total drip volume. While the containment of this liquid prevents cross contamination at the retail, purchase, and transport stages, it is important that careful handling in the home is used when the package is opened.

In contrast, another survey of drip from whole carcasses in leakproof packaging conducted in October – November 2007 found a prevalence of 20/164 (12.2%) (Chrystal *et al.*, 2008). In these tests, a 10 microlitre loop of the drip water sample was spread onto a plate. No *Campylobacter* were detected in swab samples from external packaging of the 165 whole bird samples in this survey (swab area 25 cm^{-2}, detection limit 20 CFU per 25 cm^{-2} area).

Pre-cooked poultry is rarely contaminated by *Campylobacter*. Only one sample out of 1320 (approximately, the exact number of samples is not specified) or 0.07% of ready-to-eat chicken products tested positive for *Campylobacter* in a 1992-1994 New Zealand survey (Campbell and Gilbert, 1995). For a survey in 2003 by the Consumers' Institute (2003), 25 cooked rotisserie chickens and 25 smoked cooked chickens were tested. None were positive for *Campylobacter.*

2.6.5 Poultry consumption

Consumption of poultry meat increased steadily over a 20 year period, from an apparent consumption (poultry available for consumption per capita) of 14 kg/person/year in 1986 to 34.1 kg/person/year in 2006. This figure decreased to 30.4 kg/person/year in 2009, as part of a general 6.6% decrease in meat consumption compared to the previous year. In 2009, New

Zealanders consumed 136,728 tonnes of poultry meat, which constituted 35.8% of total meat consumption.[14]

Food Standards Australia New Zealand (FSANZ) carried out an analysis of the 1997 National Nutrition Survey dataset (Russell *et al.*, 1999), including application of a set of standard recipes, to allow composite foods to be reduced to their component parts (ANZFA, 2001). This analysis gave an estimate of the proportion of the population consuming poultry meat on any given day of 27.5%.

The information in the following sections is taken from the New Zealand National Nutrition Survey (NNS) conducted in 1997 (Russell *et al.*, 1999), the 2002 Children's National Nutrition Survey (CNS) (Ministry of Health, 2003), and the 2008-2009 Adult Nutrition Survey. [15]

This analysis refers only to chicken consumed as chicken meat or chicken portions and not to chicken consumed as a minor component of a recipe. Therefore, figures will differ from those of the FSANZ analysis described above.

2.6.5.1 Proportion of population consuming poultry

For the adult New Zealand population (over 15 years of age), 19.4% reported consuming chicken in the previous 24-hour period. This is lower than the FSANZ figure given above (27.5%) and presumably reflects different assumptions concerning the chicken content of complex foods. Using data from the qualitative food frequency questionnaire (QFFQ), administered as part of the NNS, estimates of 12.2% of adults consuming chicken (roasted, fried, steamed or barbecued) and 9.8% consuming chicken mixed dishes were obtained.

For children aged 5-15 years, 24.4% of respondents to the CNS reported consuming chicken in the previous 24-hour period. The QFFQ, administered as part of the CNS suggests a much higher frequency of chicken consumption of approximately 34%.

Consumption of other poultry types, such as duck and turkey, was negligible.

A more recent survey of foods consumed by 12-24 month New Zealand children found that 22% of respondents reported consumption of chicken or turkey by the children at least once on three randomly-selected non-consecutive days (Szymlek-Gay *et al.*, 2010).

The 2008-2009 New Zealand Adult Nutrition Survey confirms that poultry remains a frequently consumed food. Only 6.6% of respondents reported not having consumed poultry in the preceding 4 weeks, while 56.4% of respondents consumed poultry 1-2 times per week.

2.6.6 Mean daily consumption of poultry

Consumers are defined as those who report consumption of a particular food within the survey timeframe. Analysis of poultry serving data from the 1997 NNS gave a mean daily

[14] http://www.pianz.org.nz/industry-information/industry-statistics/meat-consumption/meat-consumption-percentages accessed 2 November 2011

[15] http://www.moh.govt.nz/moh.nsf/indexmh/focus-on-nutrition-survey-2008-09 accessed 2 November 2011

intake for consumers of poultry of 136 g/person/day. The corresponding data for the child population (5-15 years) gave a mean daily consumption for consumers only of 114 g/person/day. It should be noted that these figures represent the amount of poultry consumed by a consumer on a day when poultry was part of the diet and does not represent long-term, habitual daily consumption of poultry, which will include days when poultry was consumed and days when it wasn't consumed.

For 12-24 month old New Zealand consumers of chicken and turkey, the median daily intake was 22 g/person/day (Szymlek-Gay *et al.*, 2010).

2.6.7 Types of poultry consumed and cooking method used

The following section summarises information on portion types and cooking methods for chicken servings reported in the NNS and CNS.

For adult New Zealanders, the most commonly consumed poultry portion type was breast (28% of servings), followed by drumstick (11.4%), light meat[16] (11.4%), leg (9.8%), thigh (9.1%) and wing (8.2%). Overall, 10.2% of servings were described as 'Chicken, KFC'. The most common cooking method was baking/roasting (39.2% of servings), followed by frying (12.5%), stewing/braising (12.3%), and grilling/barbecuing (8.9%). The cooking method was not specified for 16.7% of servings.

For New Zealand children, the most commonly consumed portion type was drumstick (25.9%), followed by breast (19.9% of servings), wing (10.7%), light meat (8.8%), thigh (7.1%) and leg (6.7%). Only 4.2% reported consuming 'Chicken, KFC'. The most common cooking method was baking/roasting (44.4% of servings), followed by frying (15.7%), stewing/braising (10.1%) and grilling/barbecuing (10.1%).

These data on cooking methods are in broad agreement with the results of a postal survey of meat handling practices (Gilbert *et al.*, 2005). In this survey, 50% of respondents reported that they would always or very frequently roast or bake chicken, while 31% reported that they would always or very frequently pan fry chicken.

A risk model for *Campylobacter* in the New Zealand poultry food chain suggested that barbecuing and microwave cooking were cooking methods with a greater potential to result in undercooked product, with a higher risk of *Campylobacter* survival (Lake *et al.*, 2007b).

2.6.8 Evaluation of exposure

2.6.8.1 Frequency of consumption and serving sizes

Estimates of the proportion of the population consuming poultry meat on any given day ranged from 19.4% (adults) to 34% (children). The amount of poultry consumed is similar for adults and children (mean approximately 100 g).

[16] Chicken meat may be classified into light (or white) and dark. These designations reflect different locations and uses of the muscles. Dark meats occur in the legs where muscles contain a large amount of myoglobin. Light or white meat, generally comes from the breasts of the birds and contains little of the meat-darkening myoglobin

2.6.8.2 Frequency of contamination

The information in Section 2.6.2 indicates that the prevalence of *Campylobacter* in raw chicken products at the end of primary processing or at retail has declined since 2003-2007. Data for 2008 – 2009 indicate the prevalence in whole carcasses is 40-50%, while in minced/chopped poultry products it is 70%. The prevalence of *Campylobacter* on other types of poultry (end of lays, turkeys and ducks), as determined by presence/absence testing of pooled caecal contents, is higher (>80%), although these products represent only a small proportion (5%) of the total poultry food supply.

Results from the NMD and other microbiological surveys (two surveys of minced/chopped products and samples taken in the Manawatu) suggest that the concentration of *Campylobacter* on chicken products also has declined over the same time period.

2.6.8.3 Growth rate during storage and most likely storage time

The shelf life of refrigerated raw poultry is quite short in comparison with other meats. Given the biology of the organism, growth will not occur during refrigerated storage, although conversely survival of *Campylobacter* will be best under refrigeration.

2.6.8.4 Heat treatment

Normal cooking temperatures should be adequate to destroy *Campylobacter*. Cooking to an internal temperature of 74ºC will give at least a 7 $\log_{10}$ decrease in *Campylobacter* concentrations on poultry (Codex, 2011).

2.6.9 Exposure summary

Poultry is a food frequently consumed by New Zealanders. Although the prevalence and concentration of *Campylobacter* on poultry in New Zealand have declined since interventions were introduced in 2006 – 2007, there is still a high likelihood (>50%) that poultry purchased by consumers will contain *Campylobacter*.

2.7 Overseas Context

Overseas surveys reporting prevalence of *Campylobacter* in broiler flocks have been summarised in Appendix 1.

2.7.1 Post processing and retail

As part of its commitment to reducing foodborne disease, the Food Standards Agency (FSA) in the United Kingdom (UK) chose a strategic target in 2005 to deliver a 50% reduction in the prevalence of *Campylobacter* in UK-produced chicken by 2010. The baseline was 70% based on surveillance data available at the time. To measure progress towards this target a survey was conducted between May 2007 and September 2008 of fresh chicken at retail (FSA, 2009). Both presence/absence and enumeration methods were used for the detection of *Campylobacter*, and the measured prevalence was found to vary according to the method used. Surprisingly the prevalence found using the presence/absence method (with enrichment) was lower than found by the enumeration (spread plate) method. This result was attributed to the enrichment step in the presence/absence method, which may allow slower

growing *Campylobacter* to be out-competed by background microflora, and may also preferentially select certain strains of *Campylobacter*. In New Zealand the presence/absence method used by the NMD is considered more sensitive than the direct plating method.

The overall prevalence of *Campylobacter* in chicken at retail in the UK (weighted for market share), based on both methods combined, was 65.2% (95% CI 62.1% – 68.2%). Prevalence in chicken of UK origin was 76.1% compared to 26.5% in chicken of non-UK origin. FSA reported that these data will be used as a more robust evidence base for setting future targets, as the 70% baseline in 2005 was no longer considered valid (see Appendix 3, Section 10.4).

A microbiological survey in the Czech Republic examined fresh and frozen poultry carcasses sampled at retail across the nation (Bardoň *et al.*, 2011). The prevalence of *Campylobacter* in fresh poultry was 90/120 (75%, 64/90 *C. jejuni*, 15/90 *C. coli*, 11/90 *C. jejuni* and *C. coli*). Equivalent data for frozen carcasses was 44/120 (37%, 30/44 *C. jejuni*, 9/44 *C. coli*, 5/44 *C. jejuni* and *C. coli*). Counts of *Campylobacter* on fresh poultry ranged from <10 to 4 $\log_{10}$ CFU per g of neck skin, while counts on frozen carcasses were an order of magnitude lower.

A large scale microbiological survey of poultry carcasses from 20 United States processing plants during 2005 has been published (Berrang *et al.*, 2007). Forty carcasses were tested for *Campylobacter* per plant, sampled at the rehang point (before evisceration) and post chill. The mean prevalence of *Campylobacter* across all plants at rehang was 596/800 (75%) while at post chill the mean prevalence was 279/800 (35%). Enumeration of a 100 ml rinsate from these carcasses showed that the mean numbers of *Campylobacter* at rehang for different plants ranged from 0.78 – 4.49 $\log_{10}$ CFU per ml (approximately 2.8 – 6.49 $\log_{10}$ CFU per carcass) with a mean of 2.66 $\log_{10}$ CFU per ml (4.7 $\log_{10}$ CFU per carcass). Post chill the numbers of *Campylobacter* were considerably lower, with a mean of 0.43 $\log_{10}$ CFU per ml (approximately 2.4 $\log_{10}$ CFU per carcass). Mean values were calculated for *Campylobacter*-positive samples only. The reduction in numbers between the rehang point and post chill varied considerably between plants and depended on the presence or absence of a reprocessing step and the specific chemical used for this. Acidified sodium chlorite, used as a post chill treatment was particularly effective.

Poultry carcasses sampled at the end of primary processing in abattoirs in Alberta, Canada from 2004-2005 had a *Campylobacter* prevalence (based on rinsate testing) of 75% (Bohaychuk *et al.*, 2009).

Carcass rinsate data from 13 broiler chicken processing plants in the United States from 2003 – 2004 found that approximately 74% of the samples did not yield *Campylobacter* by the direct plating method (Stern and Pretanik, 2006). The data were aggregated to derive a distribution of counts across all plants as follows:

$\log_{10}$ CFU per carcass	%
<3	74.4
3-3.9	12.5
4-4.9	9.5
5-5.9	2.9
6-6.9	0.5
7-7.9	0.2

A survey of retail poultry in Belgium in 2007, found that the prevalence determined by spread plating of rinsates was higher than that found by enrichment (Habib *et al.*, 2008) which is consistent with the UK study above. The prevalence of *Campylobacter* contamination in minced forms of chicken meat (burgers, minced meat and sausages) was 128/316 (40.5%) compared to 187/340 (55.0%) of portioned forms (breasts, legs, wings).

A 2005 – 2006 survey of raw poultry products from retail in New South Wales and South Australia found *Campylobacter* on 482/549 (87.8%) and 289/310 (93.2%) of samples respectively (Pointon *et al.*, 2008). Contamination occurred across all product types (skin on, skin off and whole bird) and retail sources (butchers, supermarkets and speciality stores). The mean *Campylobacter* counts were 0.87 ± 0.45 $\log_{10}$ CFU per cm^2 and 0.78 ± 0.44 $\log_{10}$ CFU per cm^2 for New South Wales and South Australia respectively. Although enumeration was determined using a rinsate, results were reported on the basis of surface area. The surface area of the whole birds and portions were calculated using formulae included in Australian Standard AS 1766.3.2 – 1979.

This survey also tested the exterior of packaging, soaker pads within the packaging, and drip juice. *Campylobacter* was isolated from the exterior of 9/219 (4%) of the samples. The majority of the soaker pads were positive for *Campylobacter* (120/167, 72%), with mean counts of 2.38 ± 0.26 and 1.12 ± 0.32 $\log_{10}$ CFU per pad in New South Wales and South Australia, respectively. Only a few drip samples were tested, where 3 of 4 samples (75%) were positive for *Campylobacter*, at a mean concentration of 1.15 ± 0.21 $\log_{10}$ CFU per ml.

The prevalence of *Campylobacter* on carcasses taken from 10 slaughterhouses in Sweden over a one year period from 2002 – 2003 was 104/636 (16%) (Lindblad *et al.*, 2006b). This total was based on positive results from either enrichment or direct plating, and was slightly lower than the prevalence rates from previous *Campylobacter* surveys of broiler cuts which were 18-20% positive. Prevalence of *Campylobacter* was low (almost zero) during winter and early spring. The majority (97%) of isolates were *C. jejuni*, while the remaining isolates were *C. coli*. Quantitative results were obtained from 88 samples, with up to 7 $\log_{10}$ CFU per carcass recorded, although almost all results were less than 5.5 $\log_{10}$ CFU per carcass.

The prevalence of *Campylobacter* in ready to eat products overseas is negligible. Two recent surveys in Canada (101 samples of chicken wieners taken in 2001) and the UK (1073 samples of unsliced ready to eat poultry taken between 1995 and 2003) found no positive samples (Bohaychuk *et al.*, 2006; Meldrum *et al.*, 2005).

Additional data from surveys of post processing samples or retail products (published since 2004) are summarised in Table 3.

Table 3: Reported prevalence of *Campylobacter* in overseas poultry products from survey published after 2004

Country	Product	Samples tested	Positive for *Campylobacter* (%)	Year	Reference
Canada	Raw chicken legs from retail	100	62.0	2001	(Bohaychuk *et al.*, 2006)

Country	Product	Samples tested	Positive for *Campylobacter* (%)	Year	Reference
Denmark	Fresh, chilled broiler meat	Approximately 250-1,000 samples per year	17.8 12.3 7.9 8.1	2004 2005 2006 2007	(Rosenquist *et al.*, 2009)
France	Carcasses at slaughterhouses	425	87.5	2008	(Hue *et al.*, 2011)
Germany	Turkey meat at retail	100	34	2007	(Atanassova *et al.*, 2007)
Japan	Retail poultry products	NS	60	NS	(Suzuki and Yamamoto, 2009)
N. Ireland	Raw poultry (chicken, turkey, duck) at retail	336	91	2007 - 2008	(Moran *et al.*, 2009)
South Africa	Whole carcasses (fresh and frozen)	99	32.3	2005	(van Nierop *et al.*, 2005)
South Africa	Whole carcasses (fresh from supermarkets)	45	48.9	2005	(van Nierop *et al.*, 2005)
Thailand	Chicken thighs	72	47.2	2000 - 2003	(Padungtod and Kaneene, 2005)
US	Chickens retail – organic conventional	 198 61	 76 74	2005	(Cui *et al.*, 2005)
UK	Poultry meat retail	2104	57.3 (chicken 60.9%, duck 50.7%, turkey 33.7%, other 34.2%)	2003 - 2005	(Little *et al.*, 2008)
UK (Wales)	Fresh chicken carcasses at retail	2001 and 2002:553 2003:544 2004:578	 70.2 73.5 71.8	Nov 2001 – Dec 2004	(Meldrum *et al.*, 2006)
UK (Wales)	Frozen chicken carcasses at retail	2001 and 2002:186 2003:192 2004: 173	 72.6 71.9 58.4	Nov 2001 – Dec 2004	(Meldrum *et al.*, 2006)
UK (Wales and Northern Ireland)	Raw whole chickens Fresh Frozen	 727 150	 72.4 60.0	March and December 2005	(Meldrum and Wilson, 2007)

NS = Not stated

The prevalence of *Campylobacter* on chicken carcasses and on retail poultry products in many of these overseas studies is similar to the prevalence in New Zealand. An exception is the Scandinavian countries (Denmark, Sweden) where prevalence of *Campylobacter* of less than 20% is achieved. Similarly, *Campylobacter* counts on poultry carcasses are similar to those determined in New Zealand, with maximum counts of approximately 5-6 $\log_{10}$ CFU per carcass being reported across a number of studies.

3 EVALUATION OF ADVERSE HEALTH EFFECTS

Supplemental information on adverse health effects is given in Appendix 2.

3.1 Disease Characteristics

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 a day to one week or longer (usually 5 days). Excretion of the microorganism in stools occurs on average for 2 to 3 weeks and is mostly self-limiting. Hospitalisation has been reported in up to 13% of cases. The maximum attack rate is around 45%.

Condition: Campylobacteriosis.

Toxins: No toxins are produced in foods.

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

Long Term Effects: Campylobacteriosis is a recognised cause of chronic sequelae in the form of Guillain-Barré syndrome (GBS) and reactive arthritis. Campylobacteriosis has also been reported to increase the risk of developing inflammatory bowel disease (IBD) (Mangen *et al.*, 2004).

The frequency of GBS resulting from campylobacteriosis has been estimated as 0.02-0.03% (McCarthy and Giesecke, 2001; Tam *et al.*, 2006) and can occur up to two months after enteritis. Approximately 15-20% of patients with GBS are left with some form of disability (Mangen *et al.*, 2004) and approximately 3-5% die (Kemmeren *et al.*, 2006).

Campylobacteriosis is also associated with reactive arthritis. The frequency of this illness has been estimated as 1% of all campylobacteriosis cases (Altekruse *et al.*, 1999) or 3-16% of more serious (GP attending) campylobacteriosis cases (Hannu *et al.*, 2002; Johnsen *et al.*, 1983; Locht and Krogfelt, 2002; Rees *et al.*, 2004).

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

3.2 Dose Response

There is a growing consensus that a minimum infectious dose for human pathogens does not exist, and ingestion of even a single cell has an associated probability of causing infection (even though the probability may be very small). If the number of exposure events is high, even low probabilities of infection may be significant.

Data from an experimental study where volunteers ingested known numbers of *Campylobacter* cells (Black *et al.*, 1988) have been investigated for the purpose of modelling the dose-response relationship (Medema *et al.*, 1996; Teunis and Havelaar, 2000; Teunis *et*

al., 1999), with an overview reported by an expert group assembled by the Food and Agriculture Organisation (FAO) and the World Health Organisation (WHO) (FAO/WHO, 2002). Infection, where the microorganism is reproducing in the body, was modelled separately from illness, which is less frequent. The likelihood of infection increased from approximately 50% at 800 cells to approximately 100% at 1 x 10^8 cells. In contrast, the likelihood of illness was approximately 20% at 800 cells, increasing to approximately 55% at 9 x 10^4 cells, and declining to 0% at 1 x 10^8 cells.

One interpretation of the limited data suggested that the likelihood of illness actually declines with increasing dose once infection is established. Some researchers suggest that exposure to a large dose elicits a stronger host defence response that reduces the probability of illness (Teunis *et al.*, 1999). Taken in combination with the model for infection, the overall effect is an optimum number of cells are consumed for sickness to occur.

More recently the FAO/WHO hazard characterisation (FAO/WHO, 2002) has explored the idea that there is a conditional probability of disease in humans resulting from infection. This model predicts that in the vast majority of cases where people become infected there is >20% and <50% chance of the person subsequently becoming sick. Data from two outbreaks, related to consumption of unpasteurised milk (Evans *et al.*, 1996; van den Brandhof *et al.*, 2003) have recently been analysed, in conjunction with the data from the volunteer study to further develop these dose-response models (Teunis *et al.*, 2005). The updated dose response relationship showed increased infectivity at low doses and a steeper increase with dose than previously reported.

3.3 New Zealand Outbreak Information and Human Health Surveillance

Campylobacteriosis has consistently been the most commonly reported infectious intestinal disease in New Zealand. Notifications for the period 1997-2012 are shown in Figure 4, while notification rates for the period 2003-2012 are shown Figure 5. The campylobacteriosis annual notification rate trend was very similar to the corresponding trend in the annual number of notifications; with a general increase in the notification rate observed over the period 2000-2006 followed by a sudden reduction in 2007. The notification rate has been fairly stable in the period 2008-2012.

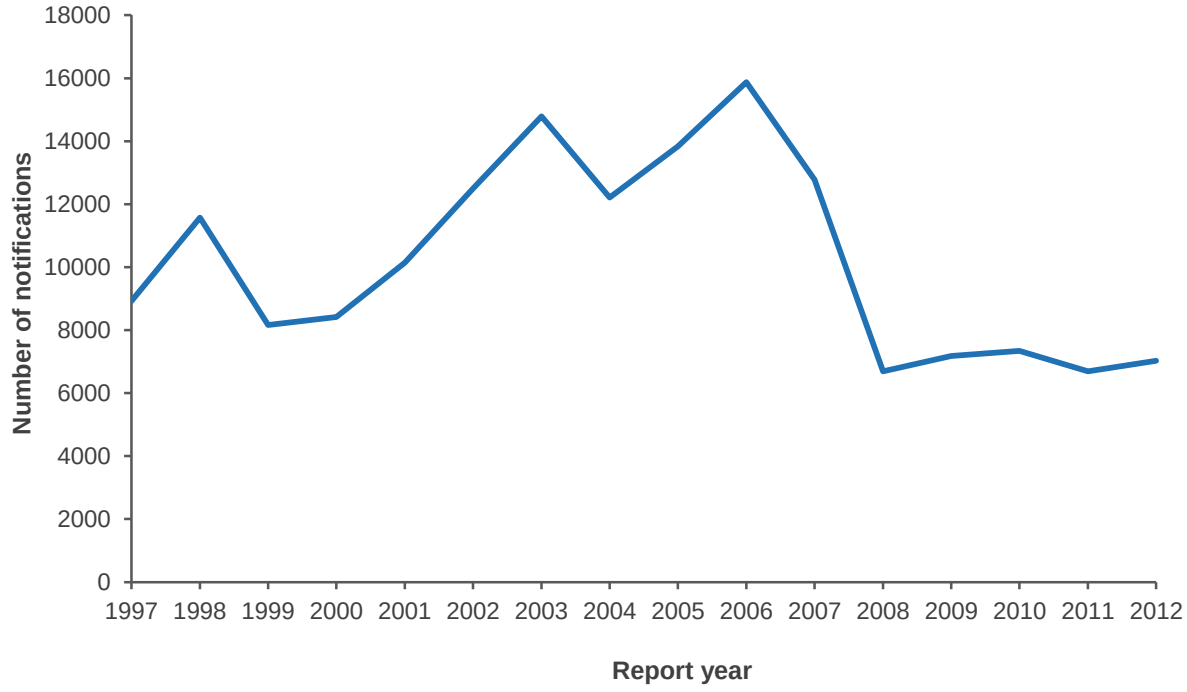
A recent study has estimated the total number of campylobacteriosis cases for New Zealand, accounting for cases that do not come to the attention of the medical reporting systems (Cressey and Lake, 2011). Mean estimates for the period 2000-2009 were 119,579 (90^{th} percentile confidence interval 56,170-198,405) or 363,490 (90^{th} percentile confidence interval 184,508-551,434) depending on the approach used.

The age distribution of notified cases in New Zealand is bimodal with peaks in the 1-4 year age group and 20-29 year group. In 2012, the highest age-specific rate occurred among children aged 1 – 4 years (300.1 per 100,000; 754 cases). The rate for 20 to 29 year olds was 179.0 per 100,000 (1,124 cases). The lowest rate was in the 10 to 14 age group at 94.8 per 100,000 (274 cases) (Lopez *et al.*, 2013).

The reported rates of campylobacteriosis in Maori and Pacific Peoples populations in 2012 were less than half of the rate for Europeans (Lopez *et al.*, 2013). For cases where ethnicity was recorded (93.2% in 2012), the rate amongst New Zealanders with European ethnicity was 182.9 per 100,000. This is higher than for other groups (Maori: 75.7 per 100,000;

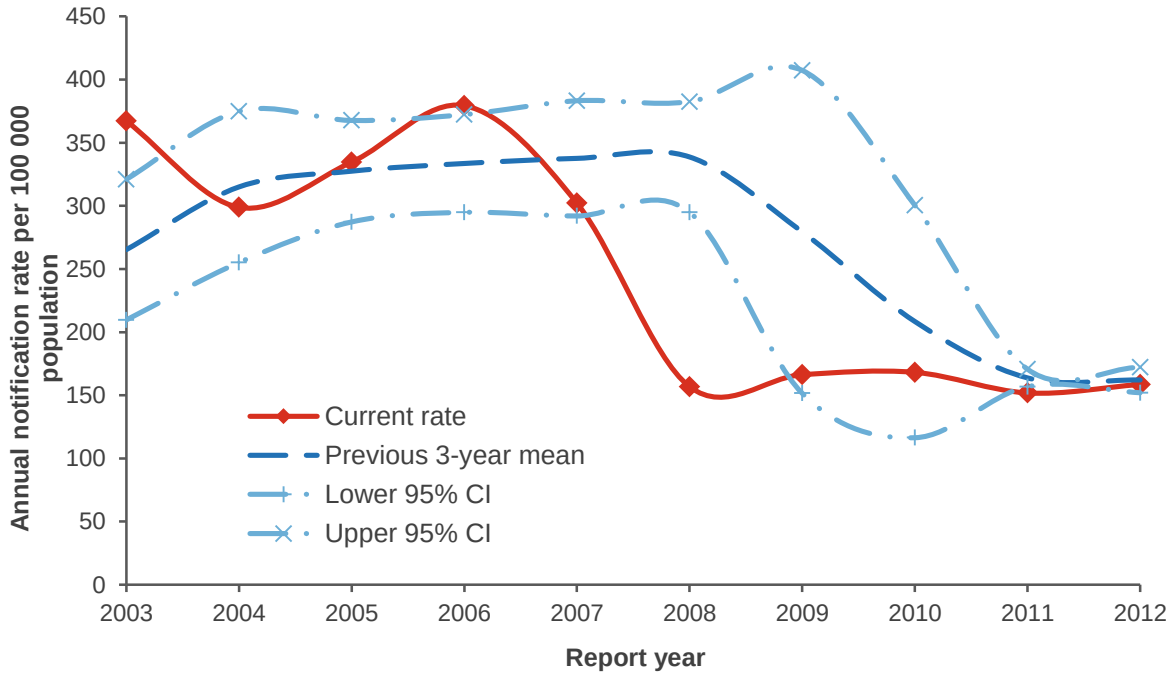
Pacific Peoples: 41.6 per 100,000, Asian: 75.0 per 100,000, and Middle Eastern/Latin American/African (MELAA): 74.2 per 100,000).

Figure 4: Campylobacteriosis notifications by year, 1997–2012



Reproduced from (Lopez et al., 2013)

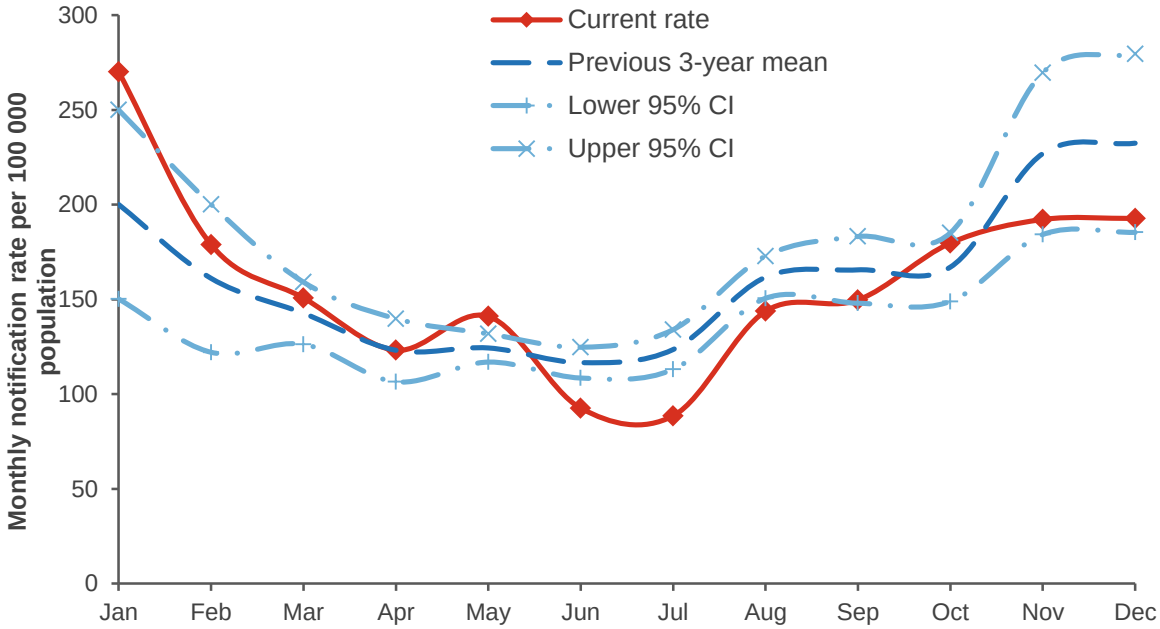
Figure 5: Campylobacteriosis notification rate by year, 2003–2012



Reproduced from (Lopez et al., 2013)

The number of notified cases of campylobacteriosis per 100,000 population by month for 2012 is shown in Figure 6 . The pattern in 2012 is similar to previous years, with a summer peak and winter trough. The monthly number of notifications in 2012 ranged from 327 notifications (July) to 998 notifications (January).

Figure 6: Campylobacteriosis monthly rate (annualised), 2012



Reproduced from (Lopez et al., 2013)

3.3.1 Clinical outcomes: Campylobacteriosis in New Zealand

Hospitalisation and fatality rates for notified cases of campylobacteriosis in New Zealand are given in Table 4. These outcomes are not always reported for each case, therefore percentages are expressed in terms of the number of cases for which outcomes are known. For 2012, 62.2% of cases had hospitalisation data recorded.

Table 4: Outcome data for campylobacteriosis in New Zealand, 2003-2012

Year	Hospitalised cases	Fatalities	Reference
2003	633/8302 (7.6%)	0/14786	(ESR, 2004b)
2004	499/6542 (7.6%)	0/12212	(ESR, 2005b)
2005	635/7887 (8.1%)	1/13839 (0.01%)	(ESR, 2006b)
2006	677/9231 (7.3%)	1/15873 (0.01%)	(ESR, 2007b)
2007	581/6916 (8.4%)	1/12776 (0.01%)	(ESR, 2008b)
2008	312/3213 (9.7%)	0/6693	(ESR, 2009b)
2009	376/3324 (11.3%)	0/7176	(ESR, 2010b)
2010	438/4000 (11.0%)	0/7346	(ESR, 2011b)
2011	397/3759 (10.6%)	0/6692	(ESR, 2012b)
2012	459/4370 (10.5%)	0/7031	(ESR, 2013b)

3.3.2 Outbreaks

The New Zealand data summarised in Table 5 show that *Campylobacter* was identified as the causative agent in around 10-15% of reported outbreaks prior to 2007.

Table 5: Total number of reported outbreaks and cases for which *Campylobacter* was identified as the causative agent in New Zealand 2003-2012

Year	No. of outbreaks	Percent of reported outbreaks	No. of cases	Percent of reported cases	Reference
2003	42	12.4	140	5.0	(ESR, 2004a)
2004	31	9.5	130	3.2	(ESR, 2005a)
2005	47	13.6	252	10.3	(ESR, 2006a)
2006	47	9.5	223	3.5	(ESR, 2007a)
2007	20	4.1	54	0.7	(ESR, 2008a)
2008	16	3.6	109	1.7	(ESR, 2009a)
2009	12	1.9	65	0.6	(ESR, 2010a)
2010	29	4.8	113	1.8	(ESR, 2011a)
2011	29	5.0	123	1.6	(ESR, 2012a)
2012	32	4.5	282	2.7	(ESR, 2013a)

The full records for all reported campylobacteriosis outbreaks from 2005 to September 2011 were retrieved from the EpiSurv database and reviewed for information linking the outbreaks with food, and poultry meat in particular. The number of outbreaks is given in Table 6. In a proportion of outbreaks, chicken meat was suspected, but in no outbreaks was this reported as confirmed by a formal epidemiological analysis or finding *Campylobacter* in the suspected food. Outbreaks where chicken liver/pâté was a suspected vehicle are listed, but are not formally part of this Risk Profile.

Table 6: Number of reported campylobacteriosis outbreaks where chicken meat or liver were suspected vehicles in New Zealand 2005 - 2010

Year	Number of outbreaks*	Chicken meat suspected	Chicken liver suspected
2005	47	10	3
2006	47	5	9
2007	20	5	0
2008	18	1	1**
2009	14	2	2
2010	30	3	2

* The number of outbreaks differs slightly from the Annual Summaries above due to finalisation of outbreaks after annual reporting

**The only outbreak that reported finding *Campylobacter* in the suspected food

3.3.3 Case control studies and risk factors

Two major case control studies of campylobacteriosis were conducted in New Zealand in the 1990s (Eberhart-Phillips *et al.*, 1997; Ikram *et al.*, 1994). Both of these studies found a strong association between poultry consumption and infection. Further details are provided in Appendix 2.

A review of campylobacteriosis surveillance data from 1995 to 2003 concluded that the observed increase in notifications was indicative of a real increase in the incidence of the disease in New Zealand (Baker *et al.*, 2007). This was largely based on the similarity in temporal patterns in notifications and hospitalisations recorded. A follow-up analysis of data up to 2008 concluded that the observed reduction in incidence of notified campylobacteriosis by approximately 50% from 2006 to 2008 was also real and attributable to interventions in the poultry food chain (Sears *et al.*, 2011). The evidence for this was again due to the temporal pattern of notifications and hospitalisations, consistency of the decline across all population subgroups, and the absence of change in the notification rates of other enteric diseases. The link with interventions in the poultry supply was based on the timing of the interventions and changes found in source attribution analyses across the key period of 2006 – 2008. A decline in the number of cases of GBS across the same period has also been linked to the decline in campylobacteriosis rates (Baker *et al.*, 2012).

3.3.4 Attribution studies

Two reviews of epidemiological data from New Zealand and overseas literature to assess transmission routes in New Zealand have been conducted. One reported that "contaminated food is the dominant known cause of campylobacteriosis in the New Zealand setting" and the data were compatible with international evidence showing that poultry was the dominant vehicle (Wilson, 2005). Another review identified that "effective management of the risk from *Campylobacter* in poultry will cause an observable reduction in the incidence of campylobacteriosis", and that overseas travel and animal contact were also important risk factors, while potable water, pets, and environmental water were likely to be minor parts of the overall transmission route picture (Lake, 2006).

A sentinel site study in the Manawatu has provided a detailed picture of *Campylobacter* sources for human infections in that region (French *et al.*, 2010; Müllner *et al.*, 2009a; Müllner *et al.*, 2010b). In a series of reports for MPI covering the period 2005 – 2010, the source attribution for campylobacteriosis has been examined using data from MLST typing of the strains of *Campylobacter* found in a range of food and environmental sources, and cases notified to the Horizons District Health Board. These analyses are based on associations between animal hosts and sequence types (STs) which have been found to be consistent across a number of countries (Sheppard *et al.*, 2010)

The proportional similarity index has been used to demonstrate the correlation between MLST types found in poultry from three suppliers in the Manawatu, and human cases in the same region. This correlation supported the need for control measures and also identified two specific sequence types (ST3069 and ST474) that have been rarely found overseas, but were common in New Zealand poultry (Müllner *et al.*, 2010a). A study investigating the epidemiology of campylobacteriosis at the genotype-level between 2005 and 2008 combined with epidemiological surveillance and population genetics provided evidence that poultry was the leading cause of human campylobacteriosis in New Zealand, causing an estimated

58-76% of cases with widely varying contributions by individual poultry suppliers (Müllner *et al.*, 2009b).

From 2007 – 2010 a project funded by the Cross Departmental Research Pool (CDRP) was undertaken by MPI, the Ministry for the Environment, Massey University, the National Institute for Water and Atmospheric Sciences (NIWA) and ESR. The project was called "*Campylobacter* in food and the environment: examining the link with public health": The reports from this project are on the MPI website.[17] Parts of this project examined source attribution for human infections with *Campylobacter* and represented an extension of the Manawatu sentinel site studies described above. This allowed consideration of additional sources: pet cats and dogs, urban and other wild birds, as well as the updating of source attribution estimates (French *et al.*, 2011).

The most recent report from this project presents a series of attribution estimates (as shown below) that illustrate the decline in attribution to poultry sources, although this remains the greatest single source (French *et al.*, 2010).

- 2005/6: poultry 75%, ruminants 18%, other 7%
- 2006/7: poultry 80%, ruminants 11%, other 9%
- 2007/8: poultry 58%, ruminants 29%, other 13%
- 2008/9: poultry 47%, ruminants 43%, other 10%
- 2009/10: poultry 50%, ruminants 33%, other 17%

The continued importance of poultry as a source of infection is consistent with the pathway attribution modelling conducted for the CDRP project (Lake *et al.*, 2011). In this analysis a series of risk factors (overseas travel, living in a rural environment, occupation) and exposure pathways (consumption of various foods and water, recreational water) were considered for the years 2006 and 2008. Although there was a marked decline in the relative importance of poultry consumption as an exposure pathway between 2006 and 2008 (80% and 53% respectively), poultry as a food remained the most important pathway.

Attribution of the decline in campylobacteriosis notifications to interventions in the poultry food chain has been confirmed by two studies. One study already mentioned applied a national perspective and examined the timing of interventions in particular (Sears *et al.*, 2011). Another study used data from the Manawatu to conduct spatial, temporal and molecular strain typing analyses (Müllner *et al.*, 2011). This study demonstrated a reduction in disease risk attributable to a reduction in the number of poultry-associated campylobacteriosis cases. Before the implementation of interventions, poultry-associated cases were more prevalent in urban areas than rural areas in the Manawatu, whereas the reverse was evident for ruminant-associated cases. In addition to the over-all reduction in the incidence of cases, this study also showed a stronger intervention effect in urban areas where poultry sources were more dominant, compared to rural areas.

[17] http://www.foodsafety.govt.nz/elibrary/industry/examining-link-with-public-health/index.htm accessed 2 November 2011

3.4 Adverse Health Effects Overseas

3.4.1 Incidence

Data on the incidence of reported cases of campylobacteriosis overseas have been summarised in Table 7. Despite significant reductions in recent years, New Zealand's rate of campylobacteriosis remains high by international standards. However, some differences may be due to reporting practices by each country's authority.

Table 7: Comparison of reported campylobacteriosis incidence between countries

Country	Period	Rate /100,000	Reference
New Zealand	**2012**	**158.6**	**(ESR, 2013b)**
Australia*	2010	112.7	NNDSS[1]
Canada	2009	22.8	NESP[2]
Czech Republic	2011	177.9	EFSA/ECDC[3]
EU (Total)	2011	50.3	EFSA/ECDC[3]
UK	2011	115.4	EFSA/ECDC[3]
US#	2012	14.3	FoodNet[4]

*Excludes New South Wales which does not report campylobacteriosis except when an outbreak occurs.
Data collected from 9 US States (FoodNet) which represents 13% of total US population.
[1] National Notifiable Diseases Surveillance System (NNDSS) http://www9.health.gov.au/cda/source/CDA-index.cfm
[2] National Enteric Surveillance Program (NESP) http://www.nml-lnm.gc.ca/NESP-PNSME/index-eng.htm
[3] European Food Safety Authority and European Centre for Disease Prevention and Control (ECDC). The European Union Summary Report on Trends and Sources of Zoonoses, Zoonotic Agents and Food-borne Outbreaks in 2011 http://www.efsa.europa.eu/en/efsajournal/pub/3129.htm
[4] FoodNet – Foodborne Diseases Active Surveillance Network http://www.cdc.gov/foodnet/

An investigation of the nine fold difference in incidence of campylobacteriosis notifications between Australia and the US in 2001 examined the frequency of healthcare seeking behaviour and stool culture frequency (Vally *et al.*, 2009). The analysis concluded that culture confirmed infections underestimated the incidence of community cases by similar ratios in both countries, and therefore these factors did not explain the difference in reported rates.

3.4.2 Attribution studies overseas

An examination of *Campylobacter* isolates from human cases and potential sources using several typing systems in Quebec, Canada, concluded that MLST was the most useful typing method (Levesque *et al.*, 2008). MLST analysis found that poultry, surface water and raw milk were the most likely sources of *Campylobacter* infection as MLST sequence types obtained from these sources were most similar to sequence types found from human cases.

Although the Czech Republic has a high rate of reported campylobacteriosis, a 2005 study of isolates using PFGE and *fla* typing found only a 6% overlap between 110 human and 92 poultry isolates (Nebola and Steinhauserova, 2006).

A study in Scotland identified the MLST sequence types (STs) of a large number of *C. jejuni* and *C. coli* isolates from human cases (5674 isolates) and potential sources (3419 isolates from chickens, cattle, sheep, the environment, wild birds, pigs, turkeys) (Sheppard *et al.*,

2009). In this analysis, chicken associated genotypes were over-represented among isolates from clinical infection with 9 out of 19 of the major disease causing lineages being associated with chicken. In fact, with the exception of the ST-206, ST-61, ST-42, and ST-403 complexes (ruminant genotypes), all of the lineages that were responsible for more than 1% of human disease were common among STs from chicken isolates.

A study in Grampian, Scotland of *Campylobacter* isolates using MLST typing, focused on isolates from young children (<5 years of age) as it was observed that this age group had a high differential of reported case rates between rural and urban dwelling children (Strachan *et al*., 2009). Proportions of *Campylobacter* isolates recovered from young children in rural areas were attributed to cattle (42%), non-chicken avian sources (24%), chicken (19%) and sheep (12%). The same analysis revealed a different order for the attribution of *Campylobacter* isolates recovered from young children in urban areas: chicken (43%), cattle (35%), sheep (15%) and non-chicken avian sources (6%). Very few *Campylobacter* isolates recovered from young children (<1.4%) were attributed to types from pigs in both rural and urban areas.

C. coli, as a species, represents approximately 10% of the isolates found in human campylobacteriosis cases. A study in Denmark, using AFLP typing of 42 *C. coli* isolates from human cases and 174 isolates from pigs and various poultry (principally chicken and turkey) showed that there was a difference in the distribution of *C. coli* isolates from pig and poultry (chicken, duck, turkey, and ostrich) species and that the various poultry species (and not pigs) were likely to be sources of human *C. coli* infection (Siemer *et al*., 2005). A later study on *C. coli* infections in the northwest of England, using MLST typing and a case-case analysis comparison with *C. jejuni* cases, found that the epidemiologies of infection with *C. jejuni* and *C. coli* were remarkably similar (Sopwith *et al.*, 2010).

The European Food Safety Authority (EFSA) published a Scientific Opinion in 2010 concerning the quantification of the risk posed by broiler meat to human campylobacteriosis in the European Union (EU) (EFSA, 2010). Handling, preparation and consumption of broiler meat was considered to account for 20% to 30% of human cases of campylobacteriosis, while 50% to 80% was attributed to the chicken reservoir as a whole. The higher figure was obtained using data from the microbial subtyping studies, while the lower figure was obtained using data from the case-control studies and outbreaks. The differences between the figures may be due to a number of factors:

- The difference between poultry as a source and broiler meat as a pathway, with indirect routes such as the environment, direct contact, and colonisation of other hosts increasing the contribution of poultry associated strains;
- Underestimation of non-poultry sources by strain typing analyses due to incomplete information;
- Underestimation of risk by case-control studies due to inaccurate exposure assessment and confounding by acquired immunity; and,
- Limited value of outbreak analyses since *Campylobacter* is an infrequent cause of reported outbreaks.

The EFSA report also contains a detailed review of available attribution methods that have been applied to the burden of campylobacteriosis.

3.4.2.1 Outbreak analysis

An analysis of 894 campylobacteriosis outbreaks reported in 2005 and 2006 in seventeen European countries has been published (Pires *et al*., 2010). Outbreaks were assigned to food sources which were categorised according to a modification of the system originally developed by the Centres for Disease Control in the US. Analysis performed according to the total number of people with campylobacteriosis suggested that drinking water was the most important source of *Campylobacter* outbreak-associated disease (18%, 95% CI 0%–46%), followed by the consumption of chicken (10%, 95% CI 5%–20%) and other poultry products (2%, 95% CI 0.3%–6%). The attribution analysis performed by the number of outbreaks was considerably different. In particular, the proportion of campylobacteriosis attributed to drinking water was considerably lower (0.3%, 95% CI 0.0 – 1.0%). On an outbreak basis the proportion attributed to chicken consumption was similar to the by case basis, at 10% (95% CI 5-21%), and to other poultry products at 1.4% (95% CI 0.3 – 4%).

An examination of 33 *Campylobacter* outbreaks between 2001 and 2006 in Australia found that a vehicle or suspected vehicle was identified in 16 of the 27 foodborne outbreaks, and that poultry (chicken or duck) was associated with 11 of these outbreaks. However, it was observed that outbreak cases presented a very small fraction (approximately 0.1%) of the total notified cases (Unicomb *et al.*, 2009).

3.5 Health Burden of Campylobacteriosis

An estimate of the burden of foodborne disease for New Zealand (Lake *et al.*, 2010) included an estimate for foodborne campylobacteriosis of 880 (90th percentile credible interval 586-1174) disability adjusted life years (DALYs). This represented 57.5% of the total 1554 DALYs for campylobacteriosis, with the percentage foodborne being derived from an expert consultation process (Lake *et al.*, 2010). Of the total burden of disease, 97% was due to morbidity, with only 3% due to *Campylobacter*-related fatalities. Sequelae to the initial gastrointestinal disease (GBS, reactive arthritis, inflammatory bowel disease) account for two-thirds of the estimated burden (Cressey and Lake, 2007).

This burden of disease study was largely based on 2005 notification data, prior to the significant decrease in notifications that were reported in 2007-2008. An updated estimate was prepared using surveillance data from 2011 (Cressey, 2011). From this analysis, foodborne campylobacteriosis remained the enteric illness with the greatest burden provided a relevance criterion was applied. This criterion is a parameter that reflects the perceived trivial nature of very mild cases of gastroenteritis, and so these cases are not included in the burden estimate. If this criterion is not applied, then the total burden of foodborne norovirus infection (which causes a very high proportion of mild cases) was higher than that for foodborne campylobacteriosis.

An economic estimate of the cost of foodborne disease in New Zealand was carried out based on 2009 notification data (Applied Economics, 2010). This assessment included additional costs including:

- Costs of regulation and surveillance incurred by the Government;
- Costs borne by businesses, including costs of compliance and the consequential costs of food incidents and disease outbreaks; and

- Personal and lifestyle costs incurred by households and individuals in connection with private disbursements and pain, suffering and disruption, including the possibility of premature death.

The estimated cost of foodborne campylobacteriosis was $36 million, excluding government and industry costs (this was second to the cost from norovirus infections of $51 million). Government and industry costs were a further $28.7 million, but were not apportioned to particular foodborne diseases.

A recent US study arrived at an estimate of the cost of illness (both cost of illness and lost quality of life in monetary terms) due to *Campylobacter* of $US 1.75 billion, with a cost per case of approximately $US 2,070 (Batz *et al.*, 2011).

A Dutch study estimated the burden of total campylobacteriosis (foodborne and other transmission routes) as 1,300 DALYs (Kemmeren *et al.*, 2006). The estimated cost of campylobacteriosis was approximately €20 million. The population of the Netherlands is approximately four times the population of New Zealand. The mean cost per case in the Netherlands would therefore be approximately €330. The methodology used in the New Zealand studies outlined above (Lake *et al.*, 2010) was based on this Dutch study.

The costs and benefits of the recent interventions to control *Campylobacter* by the New Zealand industry have been examined (Duncan, 2011). Using updated estimates of the cost of illness, industry data on the cost of implementing a variety of interventions, and government costs for the *Campylobacter* Risk Management Strategy, it was found that there was a high benefit cost ratio. This positive ratio was reduced, but still greater than 1, when alternative economic methods for valuing indirect costs (i.e. lost production) were used.

3.5.1 Adverse health effects summary

The incidence of reported campylobacteriosis in New Zealand, despite the decline since 2006, is still high compared to other developed countries. Overall, the epidemiology of the reported disease (summer peak, higher rates in young children and young adults, low proportion of cases reported as outbreaks) is similar in New Zealand and overseas. Attribution analyses for New Zealand support the continued important role of poultry as a source and transmission pathway.

4 EVALUATION OF RISK

4.1 Existing Risk Assessments

The existing models and risk assessments for *Campylobacter* in poultry in New Zealand have been described in Section 3.3.4.

4.2 Estimate of Risk for New Zealand

4.2.1 Risk associated with poultry

There is evidence that the prevalence and mean concentration of *Campylobacter* on poultry in New Zealand is significantly lower than in 2006. This reduction is linked to interventions applied to the poultry supply and which are associated with an approximately 50% decline in the incidence of reported cases of campylobacteriosis (Sears *et al.*, 2011). Nevertheless, the public health burden of this disease in 2011 is still a substantial component of the overall burden of foodborne enteric disease in New Zealand, based on DALY estimates. Campylobacteriosis is the most important foodborne bacterial enteric disease.

Two different approaches to attribution of *Campylobacter* infections in New Zealand have indicated that poultry as a source, and chicken consumption as a pathway, still represents the most important component of the epidemiology of the disease (see Section 3.3.4).

In summary, the risk of campylobacteriosis from the consumption of poultry in New Zealand has declined considerably following risk management interventions by the Ministry for Primary Industries and the poultry industry. However, poultry remains an important vehicle for infection, and further risk management is warranted.

4.2.2 Risks associated with other foods

With the decline in the incidence of campylobacteriosis, which is apparently a reduction in the number of poultry associated cases, the relative importance of ruminant sources has increased. However, consumption of red meats (including offal) as a pathway made a very small contribution to exposure in the CDRP analysis (Lake *et al.*, 2011). It has been commented that many of the cases attributed to cattle and sheep could have been infected via non-food exposures such as untreated drinking water, and this could explain the prominence of ruminant associated *Campylobacter* strains in rural areas of the Manawatu, and the relatively high notification rate in rural pre-school children (French *et al.*, 2011).

Consequently the risk of campylobacteriosis from foods other than poultry is small, when measured as a contribution to the total disease burden. However, the risk from individual servings of highly contaminated but infrequently consumed foods such as chicken livers, is likely to be high.

4.3 Data Gaps

The data gap identified in this Risk Profile is:

- Risk factors for infection of broilers with *Campylobacter* that are specific to particular regions and/or farms in New Zealand.

5 AVAILABILITY OF CONTROL MEASURES

Control measures summarised in the previous version of this Risk Profile have not been repeated here and the reader is encouraged to refer to that document (Lake *et al.*, 2007c) and Appendix 3 of this report. This section focuses on developments in New Zealand since the last version of this Risk Profile was released. Overseas developments during the same period are included in Appendix 3.

A range of regulatory and industry interventions and activities were introduced from 2006 through 2008, with the aim of reducing poultry-associated foodborne campylobacteriosis in New Zealand (Sears *et al.*, 2011).

5.1 Risk Management Strategy

In 2007 MPI (then the New Zealand Food Safety Authority) published the *Campylobacter* in Poultry Risk Management Strategy for 2007 – 2010, with an initial focus on broiler production. [18] The report declared that as it had been scientifically established that poultry meat was a primary exposure pathway in New Zealand, a comprehensive risk management strategy had been developed. This strategy aimed to achieve a sustainable reduction in *Campylobacter* levels in chicken meat through scientifically robust interventions at appropriate points in the food chain, and the adoption of a multi-pronged approach to *Campylobacter* risk reduction.

The objectives of the strategy were to:

- estimate the proportion of foodborne cases attributable to poultry and other sources;
- determine the relative contributions of different interventions throughout the food chain in reducing risks to human health;
- continue to make well-informed risk management decisions on appropriate control measures and their implementation;
- assess the effectiveness of risk management decisions by utilising a monitoring and review programme; and,
- coordinate and prioritise research activities.

The strategy was updated for 2008 to 2011, and again for 2010 to 2013, with its focus widened to consider sources of *Campylobacter* other than poultry.

A key element of the strategy was the establishment of a performance target for the monitoring programmes for *Campylobacter* in poultry already established (in April 2007) under the NMD programme. These monitoring programmes were:

- Presence/absence testing of 10 pooled caeca from each cut of birds arriving for primary processing and,
- Enumeration of rinsates of carcasses sampled at the end of primary processing, i.e. post-chill.

[18] http://www.foodsafety.govt.nz/industry/general/foodborne-illness/campylobacter/strategy.htm accessed 2 November 2011

Results from the monitoring programmes have been discussed in Section 2.6.2.

The Poultry *Campylobacter* Performance Target (CPT) is specified in the "Animal Products (National Microbiological Database Specifications) Notice 2012"[19] and was implemented in April 2008. The CPT uses a limit to determine compliance of 6000 CFU/carcass, 3.78 $\log_{10}$ CFU/carcass. There are two classes of CPT failure, specified for either standard or very low throughput (VLT) premises.

(1) Enumeration Failure (EF): EF CPT non-compliance will be generated upon detection of a value greater than 6000 CFU per carcass (3.78 $\log_{10}$ CFU/carcass) in:

Standard: seven (7) or more individual carcass samples in a 45 sample, 3 successive processing periods moving window; OR

VLT: two (2) or more individual carcass samples in a 9 sample, 3 successive processing periods moving window.

(2) Detection Failure (DF): DF CPT non-compliance will be generated upon detection of 2.30 $\log_{10}$ CFU/carcass or greater in:

Standard: 30 or more individual carcass samples in a 45 sample, 3 successive processing period moving window; OR

VLT: Six (6) or more individual carcass samples in a 9 sample, 3 successive processing period moving window.

The number of classes of CPT failure were reduced from four to two following a review.[20] The high count failure and quarterly failure were discontinued, as only one premises had incurred an alert based on each CPT component in the period 2008-2011. In both cases the premises also had a 'moving window' (EF) alert. A CPT based on a combination of EF and DF was considered to provide "the greatest opportunity to "fine tune" expectations of improved microbiological process control against practical issues associated with responding to the number of alerts triggered". The *Campylobacter* Management Plan Failure (MPF) was also discontinued on the condition that equivalent responses were included in the proposed failure responses

Required responses by operators to the discovery of any of these compliance failures are specified, along with follow-up actions by MPI.

A review of the caecal testing programme resulted in this testing being discontinued in July 2009. [21] In July 2011, the sampling requirements for VLT premises were amended to reduce the numbers tested from five carcasses per week to three.

[19] http://www.foodsafety.govt.nz/elibrary/industry/animal-products-national-nmd/nmd-notice-amended-includes-schedule-2012.pdf accessed 15 March 2013
[20] http://www.foodsafety.govt.nz/elibrary/industry/draft-poultry-nmd/discussion-paper-e-coli-campylobacter.pdf accessed 18 July 2013
[21] http://www.foodsafety.govt.nz/elibrary/industry/animal-products-national-nmd/amendments/nmd-09-amendment-cover-sheet-draft.pdf accessed 15 March 2013

5.2 Relevant Food Controls

5.2.1 Industry controls

In parallel with the development of MPI's *Campylobacter* Risk Management Strategy, the poultry industry in New Zealand has been applying measures to reduce the prevalence and counts of *Campylobacter* on poultry. These measures concerned biosecurity on broiler farms as well as adjustments to primary processing conditions. These interventions were described in a paper discussing the fall in notifications (Sears *et al.*, 2011). They included:

- Broiler Growing Biosecurity Manual (see Section 10.1.4.2);
- Code of Practice for Primary Processors (see Section 10.1.4.1)
- Improvements in procedures for catching and transporting birds, in particular single use of catching crates each day, to allow proper cleaning and drying between uses to reduce cross contamination;
- Adjustment of primary processing conditions, including immersion chiller temperature and pH, chlorine use and water flow; and,
- Leak proof packaging of retail poultry.

Technical interventions that have been identified as important by the poultry industry include (PIANZ, personal communication, August 2013):

- Maintenance, adjustment and, if required, replacement of vent opening and evisceration equipment; and
- Implementation of sprays to ensure that carcasses are washed after processing steps with the potential for contamination.

5.2.2 Research in New Zealand commissioned by MPI/MAF/NZFSA

A systematic review of on-farm factors that affect *Campylobacter* contamination of broilers was conducted (Hudson *et al.*, 2008). This document also provided an overview of the New Zealand broiler farming industry sector. This review was followed by a survey of 60 of the approximately 160 broiler farms in New Zealand (Lake *et al.*, 2008b). All farms were visited by veterinarians to complete an extensive questionnaire and view aspects of the farms related to biosecurity. In general, many aspects of biosecurity were found to be good:

- The majority of farm grounds were well maintained;
- Surface waters were rarely used as a drinking water source;
- Most farms chlorinated their broiler drinking water and monitored the treatment;
- Dead bird collection and disposal was generally frequent and controlled;
- Both sheds and annexes were usually cleaned, sanitised and dried between flocks;
- Regular biosecurity audits were conducted;
- Staff biosecurity facilities (boots, shed entry barriers, hand washing facilities) were provided in most sheds;
- Visitor cleanliness and vehicle decontamination facilities were standard on most farms; and,
- Pest control and exclusion (birds, rodents) was standard and apparently effective.

The survey did identify some areas where improvements could be made:

- More rigorous monitoring of chlorination of drinking water (also identified as a problem in some biosecurity audits);
- More frequent or rigorous cleaning of drinker lines;
- More stringent exclusion of pets from shed surroundings;
- Universal provision of hand washing or hand hygiene facilities for staff and visitors;
- Repairs or replacement of shed and annex structural features to improve cleanability;
- Upgrading or replacement of end pads and universal cleaning and sanitising between flocks;
- More universal availability of facilities for vehicle decontamination; and,
- Provision of dedicated clothing for each shed, in addition to the dedicated boots already available.

However, it was acknowledged that this survey was conducted prior to the release of the Poultry Industry Biosecurity Manual (August 2007). Anecdotal reports from the industry indicated that aspects of on-farm biosecurity had been addressed since the manual release. Biosecurity associated with catching gang operations (thinning or partial depopulation) was also identified as an area where improvements could be made.

A study of the effect of 0.7% added caprylic acid in poultry feed on *Campylobacter* concentrations in poultry caeca has been conducted for MPI (Ravindran and French, 2011). The birds were inoculated with two commonly found *C. jejuni* MLST types, followed by the administration of feed with caprylic acid. Addition of caprylic acid was found to have no effect on broiler performance in terms of weight gain, feed intake, and feed conversion ratio. Caecal counts declined in both treatment and control groups after day 33, but there was no significant effect of treatment. Although the birds were inoculated with an equal mixture of ST 474 and ST 45, only ST 474 was recovered from samples later in the trial. This indicates that ST 474 rapidly out-competed ST 45 in both treatment and control groups. These results are consistent with US studies which found that caprylic acid reduced the probability of colonisation but did not alter caecal populations once the infection was established (De Los Santos *et al*., 2010).

A quantitative study of the changes in *Campylobacter* spp. carcass loading, as defined by rinsate counts, during different stages of primary poultry processing has been conducted (Paulin, 2011). Rinsate samples were taken from the cavity, neck, vent, wings, legs and skin of carcasses at three separate stages of processing: following the de-feathering, evisceration and spin-chill stages. The *Campylobacter* numbers at the de-feathering and evisceration stages differed between the two processors, although the reduction in numbers after the spin chill stage was considerable at both processors. The pattern of the location of the *Campylobacter* cells on the carcass also differed between the processors. This study demonstrated the benefits of such analyses at individual plants, and one processor subsequently installed new dressing equipment, which improved performance.

A New Zealand study of the potential dissemination of *Campylobacter* by farmers overalls on broilers farms showed that loose debris shaken from two of ten overalls tested positive for *Campylobacter*, while one of these also tested positive from a subsequent rinsate (Wong, 2009).

Consumer knowledge and attitudes towards *Campylobacter* in poultry and campylobacteriosis were assessed in a telephone survey of 1,000 consumers in 2008 (Gilbert and Cressey, 2008). The key findings of the survey were:

- From a selected list of foods, 89% of respondents thought chicken was likely to cause food poisoning, followed by other meats (21-58% of respondents, depending on the meat type), milk and dairy products (25% of respondents) and fresh fruit and vegetables (4% of respondents).
- Consumers receive information on chicken-related food safety issues from a range of media sources, including television>newspaper>journals/magazines>radio
- Consumers appear to be eating chicken more frequently than 10 years ago, with the main reasons being taste, convenience, healthiness and value for money.
- Boneless portions are the most commonly purchased form of chicken, with fresh/raw being the most commonly purchased state.
- There appears to have been an increase in the practice of thawing chicken in the refrigerator since 2005.
- Most current chicken purchasers (84%) claimed that they would buy chicken if only frozen chicken was available. Loss of convenience was seen as the major disadvantage of freezing.
- Stricter farm management practices were seen as the most acceptable means of controlling bacteria on chicken, with chemical treatment the least acceptable. Out of the 988 respondents who consume chicken, approximately one-quarter stated that they would be prepared to pay a 10-20% premium for safer chicken achieved through stricter farm management practices.

5.3 Options for Risk Management

A summary of overseas studies and risk management interventions for *Campylobacter* in poultry is provided in Appendix 3, and has prompted the suggestions below.

5.3.1 On farm

Studies highlight the difficulty of preventing the introduction of *Campylobacter* infection on broiler farms, and the need to better identify farm-specific risk factors. Despite evidence that thinning (partial depopulation, taking more than one 'cut' from a flock) represents a biosecurity risk for *Campylobacter* introduction, thinning is a currently accepted practice in poultry production in New Zealand. The need for thinning in New Zealand is accentuated by small processing plants, the large range of poultry products (requiring bird size variability), and the cost of more sheds.

Studies in Denmark, Norway and Sweden have shown that positive flocks are produced by only a segment of the producers and that it may be possible to identify regions or farms where enhanced biosecurity could provide reduced prevalence, through the examination of National Microbiological Database (NMD) caecal testing results. Although the New Zealand poultry industry has introduced a Biosecurity Manual, it is likely that given the geographical spread of broiler farms in this country further control measures may be farm or region specific and thus require targeted study.

5.3.2 Primary/secondary processing

Increased chemical decontamination of carcasses during processing is an option. A consumer survey indicated that consumers do not favour chemical decontamination (Gilbert and Cressey, 2008). However, this may be the best currently available risk management option to control *Campylobacter* to acceptable levels on poultry carcasses. It does however highlight the need to continue to research alternative risk management options which may be more acceptable to consumers. Also there may be a need to educate consumers about the safety of such risk management options.

5.3.3 Retail/food service

The universal use of sealed packaging may reduce the risk of cross contamination in retail and distribution environments. Contamination from leakage or external contamination of poultry meat packs might be better controlled if bags were available at the poultry chiller, allowing the immediate separation of poultry from other groceries in trolleys or when being packed.

5.3.4 In the home

Although poultry is the vehicle on which *Campylobacter* enters the home, it appears to be unhygienic handling and cross contamination which create most human exposures, as proper cooking will destroy the organism. Nevertheless, there are cooking events, such as barbecuing, which pose a greater risk of undercooking.

MPI provides advice on preventing raw meat cross contamination on its website.[22] Consumer education campaigns concerning general kitchen hygiene have been conducted in the past and could be repeated (Simmons *et al.*, 2001). However, it is acknowledged that influencing consumer hygiene behaviour is difficult.

[22] http://www.foodsmart.govt.nz/food-safety/high-risk-foods/raw-meat-cross-contamination/ accessed 10 May 2013.

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7 APPENDIX 1: HAZARD AND FOOD

The following information is taken from a number of different sources but, unless otherwise referenced, is primarily derived from a data sheet prepared by ESR under a contract for the Ministry of Health in 2000-2001 (ESR, 2001). The data sheets are located on the MPI website[23] and are intended for use by regional public health units and will be updated from time to time. Please be aware that new information on the subject may have arisen since this document was finalised.

7.1 *Campylobacter*

Growth:

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

pH: Optimum 6.5 to 7.5, range 4.9 to 9.5.

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

Water activity: Optimum growth is at a_w = 0.997 (≡0.5% NaCl), minimum a_w ≥0.987 (≡2.0% NaCl).

Survival:

Campylobacter are sensitive to air, drying and heat.

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

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

[23] See http://www.foodsafety.govt.nz/science-risk/hazard-data-sheets/pathogen-data-sheets.htm accessed 2 November 2011

Water activity: *Campylobacter* are very sensitive to drying, particularly at ambient temperatures. The microorganism 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 (Talibart *et al.*, 2000). Transition to a VNC state was demonstrated for two New Zealand *Campylobacter* strains, although attempts to resuscitate the organisms were unsuccessful (Brandt and Podivinsky, 2008). VNCs may colonise the intestinal tract of chickens (ICMSF, 1996).

Inactivation (Critical Control Points and Hurdles)

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

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

Numbers declined rapidly on sterile meat slices of high and normal pH when incubated at 25^{o}C (Gill and Harris, 1984).

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

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

Water activity: Sensitive to even slightly reduced water activity but under certain refrigeration conditions can remain viable for several weeks (ICMSF, 1996). The drying of surface tissues during air chilling of red meat carcasses is important in reducing *Campylobacter* prevalence (for example, from 9% before chilling to 0% after chilling on pig carcasses (Oosterom *et al.*, 1985)). A review of survival by *Campylobacter jejuni* 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 (Murphy *et al.*, 2006). Poultry primary processing in New Zealand uses immersion chilling, and plant conditions do not permit the same air drying effect afforded to red meat carcasses.

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

(Epling *et al.*, 1993).

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

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

7.1.1 Typing methods

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 from such methods because subtyping can link strains to suspected sources. Additional levels of subtyping allow determinations of potential virulence, survival, antibiotic resistance etc.

Serotyping using agglutination reactions according to the Penner system, once used as the principal international reference typing scheme, is now rarely applied (Penner and Hennessy, 1980).

C. jejuni and *C. coli* have two flagellin genes, *fla*A and *fla*B. The ends of these genes are highly conserved, while there is considerable sequence variation in the region in-between. A number of primers have been designed that amplify specific regions of this gene cluster. Variability within each amplicon may be identified by digestion followed by restriction fragment length polymorphism detection on an electrophoresis gel (this technique is known as amplified fragment length polymorphism (AFLP) or restriction fragment length polymorphism (RFLP). Alternatively the fragments may be sequenced. The *fla* Short Variable Region (SVR) method is based on the nucleotide sequence found in a single locus of the two flagellin genes. Due to the small size of the SVR (321 bp), sequence determination is quick and reliable. Sequence comparison of the *fla*A SVR is nearly as discriminating as the complete *fla*A sequencing (Meinersmann *et al.*, 2005).

New typing systems continue to be developed, including a method based on binary typing derived from the presence or absence of a set of putative virulence genes (Cornelius *et al.*, 2010).

The most commonly applied methods of typing of *Campylobacter* in New Zealand have been pulsed field gel electrophoresis (PFGE) and multi-locus sequence typing (MLST).

7.1.1.1 PFGE

Restriction enzyme digestion and pulsed field gel electrophoresis (PFGE) has been extensively used in the genotyping of *Campylobacter* (Gibson *et al.*, 1994). As the enzymes used and the conditions under which the gel electrophoresis is undertaken can have a marked influence on the end result, standardised protocols are essential. 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.

The PulseNet USA network was established in 1996 by the Centers for Disease Control and Prevention and now involves several international networks.[24] New Zealand is part of the PulseNet Asia Pacific branch through the participation of ESR.[25]

7.1.1.2 MLST

Isolates of *Campylobacter* may also be strain typed using multilocus sequence typing (MLST). This technique involves amplification and sequencing of seven "housekeeping" genes i.e. genes which are conserved in all strains of *Campylobacter* but which exhibit sufficient variation to enable differentiation between strains.

Data from MLST can be compared with types found in other laboratories using an International database, hosted by Oxford University.[26]

7.1.2 Behaviour of *Campylobacter* in poultry: on the farm

Vertical transmission (i.e. via eggs from breeders to broilers), appears unlikely to be important in the transmission of *Campylobacter* to broilers (Callicott *et al*., 2006).

An intensive study of housed broiler flocks in the UK using MLST typing has demonstrated the importance of environmental contamination as a source of *Campylobacter* (Bull *et al.*, 2006). *Campylobacter* spp. were detected in the environment surrounding the broiler house, prior to as well as during flock colonization, for six of the ten flocks studied. On two occasions, *Campylobacter* isolates detected in a puddle just prior to the birds being placed (i.e. chicks placed in the house) were indistinguishable from those colonizing the birds. Once flocks were colonized, indistinguishable strains of *Campylobacter* were found in the feed and

[24] http://www.pulsenetinternational.org/Pages/default.aspx
[25] http://www.pulsenetinternational.org/networks/Pages/asiapacific.aspx
[26] http://pubmlst.org/campylobacter/

water as well as in the air of the broiler house. *Campylobacter* spp. were also detected in the air up to 30m downwind of the broiler house, which suggest that airborne transmission is possible. At any time during rearing, broiler flocks were colonized by only one or two strain types (determined by MLST) but these changed, with some strains superseding others.

A study around broiler farms in Sweden found that *Campylobacter* spp. were widespread in the environment, but there was no difference in environmental prevalence between farms which often delivered *Campylobacter* positive batches compared to those that did not (Hansson *et al*., 2007b). It was concluded that physical barriers between the inside and outside of the houses were important in preventing flock infection.

A study of the risk factors for broiler flock infection in Iceland from 2001 – 2003 found a prevalence of *Campylobacter* of approximately 15%, with most (95%) of the infected flocks being raised during the summer months of April–September (Barrios *et al*., 2006). The odds of a flock being positive for *Campylobacter* spp. increased with age and flock size. Vertical ventilation systems were also strongly associated with positive flocks (OR = 5.3). There was no evidence of an effect on the risk of a *Campylobacter* positive flock from: year; company; *Campylobacter* being carried over from one flock to the next; time interval between flocks; using (at the hatcheries) eggs laid on the floor; density of bird housing, or the number of catch lots a flock was divided into for slaughtering purposes. With respect to thinning as a risk factor, it was noted that in Iceland, special hygienic measures are taken by the catching crews to prevent introduction of *Campylobacter*, these crews are usually made up of workers from the source farm, and do not move from farm to farm. The importance of flock size was reinforced in a subsequent study in Iceland analysing risk factors for *Campylobacter* infection in flocks between 2001 and 2004 (Guerin *et al.*, 2007a). Factors associated with an increased risk of *Campylobacter* were increasing median flock size on the farm ($p \leq 0.001$), spreading manure on the farm ($p = 0.004$ to 0.035), and increasing the number of broiler houses on the farm ($p = 0.008$ to 0.038). Protective factors included the use of official (municipal) ($p = 0.004$ to 0.051) or official treated ($p = 0.006$ to 0.032) water compared to the use of non-official untreated water, storing manure on the farm ($p = 0.025$ to 0.029) and the presence of other domestic livestock on the farm ($p = 0.004$ to 0.028).

A longitudinal study of flocks in the United Kingdom found that the key predictors of infection were mean temperature and mean rainfall in the month of slaughter and also the presence of natural ventilation (Rushton *et al*., 2009). *Campylobacter* survives better in wet conditions. The effect of natural ventilation was possibly due to magnified effect of external weather conditions (forced ventilation gives greater temperature control) or else an indirect factor such as increased access by *Campylobacter* vectors such as flies.

Another UK study of risk factors for *Campylobacter* infection found that cattle on or adjacent to the farm increased the risk (OR = 1.7, 95% CI:1.1-2.7), whereas chlorinated drinking water reduced risk (OR = 0.5, 95% CI 0.2-0.9) (Ellis-Iversen *et al.*, 2009). If the first removed batch from the previous flock in the house had been *Campylobacter* positive, the first batch of the following flock was also more likely to be colonised (OR = 3.2, 95% CI 2.1-4.9). This association was more likely due to a persistent risk practice or source of *Campylobacter* on the farm than a direct carry-over from previous flock.

A German study used farm-and flock-specific information obtained from questionnaires, to identify three risk factors for *Campylobacter* colonization (Näther *et al.*, 2009). *Campylobacter* prevalence was significantly higher in flocks from free-range and organic

farms, in flocks with a size up to 15,000 birds and with more than 25,000 birds, and in flocks using nipple drinkers with trays. There was no evidence of an effect of slaughter age, time interval between successive flocks, hygiene measures, number of broiler houses on a farm, partial slaughter, source of water supply, and number of farm employees on the *Campylobacter* infection rate

The genetic diversity of the types of *Campylobacter* found on poultry carcasses seems to depend on the farm type. In a Swedish study, genotypes were characterised by macrorestriction profiling (Lindmark *et al*., 2006). Isolates from post chill carcasses, neck skin and cloacal swabs from housed flocks in Sweden were found to be limited to one or two different *Campylobacter* genotypes from whole carcass groups. In contrast, a study of free range broilers in the UK which characterised isolates using MLST and *fla*A SVR sequencing, found much higher diversity of *Campylobacter* strains (Colles *et al*., 2010). Up to five types of *Campylobacter* per bird were identified from live birds, and up to eight types per carcass from carcasses.

Examination of the types of *Campylobacter* found on broilers and other livestock (cattle, pigs, bantams, laying hens) on the same Swiss farms have shown a high number of matching types, suggesting cross contamination between different livestock types (Zweifel *et al*., 2008).

Risk factors for the presence of *Campylobacter* in conventional broiler flocks in Northern Ireland have been studied (McDowell *et al*., 2008). Statistically significant risk factors included the observation of rodents or rodent droppings, age of birds at sampling, season (summer risk was double that of other seasons), farms with three or more broiler houses, frequency of footbath disinfectant changes and a variable reflecting general tidiness and cleanliness of the broiler house anteroom. There was no significant evidence of carryover of infection from one production cycle to the next, and (in contrast to the Swiss study above) no evidence of other animal species acting as a source of infection.

A study in Denmark has shown that the summer peak of prevalence of infection in flocks correlates with a summer peak in prevalence in retail samples (Boysen *et al*., 2011).

Approximately 40% of Swedish broiler producers deliver *Campylobacter*-negative broilers in 90–100% of their flocks, showing that it is possible to produce *Campylobacter*-free broilers in Sweden (Hansson *et al*., 2010a). A study using interviews with farmers and farm visits found that factors significantly associated with increased proportion of *Campylobacter*-positive flocks were the presence of other livestock on the farm, or the presence of cattle, swine, poultry, or fur animals within 1 km of the farm. Poor or average general tidiness were associated with increased proportion of *Campylobacter*-positive flocks, but the proportion decreased if split slaughter (i.e. thinning) was seldom or never applied, or if farm workers changed footwear twice or three times instead of once before entering the broiler house. The age of birds at slaughter was not considered as a model variable.

Norway has a low prevalence of contamination by *Campylobacter* in broiler flocks compared to other developed countries (Hofshagen and Kruse, 2005). A case-control study was conducted in 2005 to identify risk factors for the presence of *Campylobacter* spp. in Norwegian broiler flocks (Lyngstad *et al.*, 2008). A total of 131 broiler farms (44 cases and 87 controls) were included in the study, and one flock from each farm was included in the statistical analyses. Factors associated with an increased risk of testing positive for

Campylobacter spp. included water from a private water source, swine holdings closer than two km, a specific slaughterhouse, a hired animal caretaker, transport personnel passing through the hygiene barrier when delivering day-old chickens, less than nine days between depopulation and restocking, and multiple broiler houses on the farm.

7.1.2.1 Thinning (partial depopulation)

The importance of partial depopulation (thinning) has been a matter of debate. A Dutch study suggested that the observed increased in the prevalence of *Campylobacter* in flocks after thinning was attributable to the older age of the flock, regardless of thinning practices (Russa *et al*., 2005). However, a study in the UK in 2005-2006 found identical PFGE types of *Campylobacter* on thinning personnel and equipment as those in flocks that became *Campylobacter* positive after thinning (Allen *et al*., 2008). Nevertheless, 41% of the flocks in this study became *Campylobacter* positive before thinning, highlighting the general importance of biosecurity.

Evidence for the ability of *Campylobacter* on transport crates to contaminate birds at slaughter has been found in a study in Sweden (Hansson *et al.*, 2005). However, the contamination was found in external neck skin samples, rather than cloacal samples. Residual *Campylobacter* contamination was found on 57% of transport crates, after cleaning. Similar results have been found in a study in Belgium (Rasschaert *et al.*, 2007). In three of four flocks that were *Campylobacter* negative before transport, *Campylobacter* were isolated from swabs taken from the head and breast of chickens after arrival at the slaughterhouse, but the birds remained negative when the caeca was sampled. The *Campylobacter* strains found were characterised using PFGE typing, and half the types found on the birds were also isolated from crates. It was suggested that the remaining types had originated from the workers handling the chickens.

7.1.2.2 Insects

Studies of insects, particularly flies, found on Danish broiler farms have supported the theory that insects can act as vectors for infection and that excluding insects from broiler houses by the use of fly screens can reduce infection rates from 51% to 15% (Hald *et al.*, 2008; Hald *et al.*, 2007).

The ability of darkling beetles inoculated with *Campylobacter*, as well as naturally contaminated beetles, to colonise poultry when eaten by birds has been demonstrated in a study in the Netherlands, indicating the importance of controlling these insects in sheds (Hazeleger *et al.*, 2008).

7.1.3 Behaviour of *Campylobacter* in poultry: primary and secondary processing

A study has shown that the numbers of *Campylobacter* on broilers are highest after the scalding/defeathering stages in a German processing plant (Reich *et al.*, 2008). The numbers of *Campylobacter* were lower after evisceration and chilling, with a mean reduction in numbers of 0.7 $\log_{10}$ CFU per carcass after chilling. The prevalence of carcass contamination in flocks whose caeca were positive for *Campylobacter* was generally close to 100%, but flocks which had negative caecal samples showed *Campylobacter* contamination of up to 50%, and that the prevalence was dependent on the *Campylobacter* status of the previous flock.

A study at two Danish commercial slaughter plants examined six broiler flocks determined to be *Campylobacter* positive prior to slaughter (Rosenquist *et al.*, 2006). Neck skin samples were taken at four locations within each slaughter plant. Evisceration at one of the plants led to a significant increase in the *Campylobacter* concentration of 0.5 $\log_{10}$ CFU per g in average, whereas no significant changes were observed during this operation at the other plant. Air chilling and water chilling, both including a carcass wash prior to the chilling operation, caused similar, but significant reductions of 0.83 and 0.97 $\log_{10}$ CFU per g, respectively. In packed frozen chickens, an additional average reduction of 1.38 $\log_{10}$ CFU per g was due to the freezing operation, whereas in packed chilled chickens the number of thermotolerant *Campylobacter* per gram remained at the same level as after air chilling.

The effects of immersion chilling and air chilling on *Campylobacter* numbers have been shown to be comparable, with up to a 90% reduction in *Campylobacter* concentration achievable (Huezo *et al.*, 2007). Another study reached the same conclusion, however, the numbers of *Campylobacter* were lower on immersion chilled carcasses compared to air chilling but the difference was not large (<1 $\log_{10}$ CFU per ml of rinsate) and may have been due to simple dilution (Berrang *et al.*, 2008).

7.1.3.1 Environmental contamination

The processing environment for poultry may provide *Campylobacter* that contaminates carcasses from uninfected or partially infected flocks. Cross contamination may occur from infected flocks to those that follow in the processing line via contamination of equipment, or else general contamination of the processing environment may affect prevalence. Extensive contamination of the processing environment by the same genotypes of *Campylobacter* as found in an infected flock has been demonstrated in a Norwegian study, and residual contamination remained after an overnight disinfection process (Johnsen *et al.*, 2007). As only a low proportion (<5%) of Norwegian flocks are infected, this environmental contamination presented risks for the introduction of *Campylobacter* contamination into uninfected flocks. These results are consistent with a French study, where survival of *Campylobacter* on food processing equipment surfaces after cleaning and disinfection, and the ability of these strains to contaminate carcasses was demonstrated (Peyrat *et al.*, 2008).

A study in the UK reported that the types of *Campylobacter* found on carcasses from uninfected flocks were not the same as those found on infected flocks processed before the uninfected flocks, suggesting that the source of contamination on carcasses from the uninfected flock was the environment (Elvers *et al.*, 2011). This was in contrast to an earlier UK study, where the types of *Campylobacter* found on low prevalence flocks were similar to those on high prevalence flocks slaughtered immediately before (Allen *et al.*, 2007). This study also found that *Campylobacter* strains were isolated in considerable numbers from aerosols, particles and droplets in the hanging-on, plucking and evisceration areas of the processing plants but not in the chillers. This occurred even when the microorganism was not isolated from either the target flock or the one preceding it.

A study of the number of *Campylobacter* on carcasses passing through the primary processing chain in a Thai poultry plant found the expected pattern of contamination, whereby relatively little change in *Campylobacter* numbers was found following the. scalding, plucking and evisceration stages, but *Campylobacter* numbers decreased by approximately 2 $\log_{10}$ CFU per carcass after immersion chilling (Osiriphun *et al.*, 2011).

This study was conducted for the purpose of developing a quantitative risk assessment model, and noted that the frequency of punctured intestines was approximately 5% by manual evisceration and 20% by machine evisceration. An analysis of the process suggested improvements, including maintaining and monitoring the chill water at 6.0 – 6.5ºC and increasing the residual chlorine in the chill water.

Samples taken from 39 flocks in three Belgian poultry slaughterhouses included crop swabs before slaughter and intestines and neck skins during slaughter (Rasschaert *et al.*, 2006). A total of 309 *Campylobacter* isolates were identified at species level and further characterized by *fla*A and PFGE typing. Isolates were identified as *C. jejuni* (90%), *C. coli* (8.7%), and *C. lari* (2.2%). Seventy-two percent of the flocks arriving at the abattoir were colonized with *Campylobacter*. After slaughter, 79% of the flocks had contaminated neck skins. In six flocks, *Campylobacter* genotypes isolated from the neck skins were also found in the alimentary tract from previously slaughtered flocks. Four of these flocks were initially free of *Campylobacter*. The researchers commented that these four flocks might have had no contaminated carcasses if logistic slaughtering (i.e. processing uninfected flocks before infected ones) had been practised.

7.1.4 Behaviour of *Campylobacter* in poultry: preparation and cooking

There have been a number of studies examining the parameters of transfer of *Campylobacter* from poultry to surfaces, such as hands and cutting boards and subsequently to other foods. The transfer rates of naturally occurring *Campylobacter* on chicken breast fillets to hands, cutting boards and knives were found to be low (<5%) (Luber *et al.*, 2006). Subsequent transfer from boards and knives to a second food (cucumber) was found to be higher (10%). The higher transfer rate for the second step suggested that the transfer rates from lower numbers of initial cells were higher. This was consistent with another study which evaluated transfer of *Campylobacter* from poultry skin and meat to high density polyethylene cutting board surfaces (Fravalo *et al.*, 2009). In these experiments the percent transfer rate was found to be inversely related to initial load. The highest transfer rate for *Campylobacter* observed was approximately 3% for ~ 1.4 $\log_{10}$ CFU per g of skin, while at 4 $\log_{10}$ CFU per g the transfer rate was approximately 0.003%.

Studies carried out in New Zealand also produced similar results, with higher transfer rates at lower initial numbers of cells (Wong *et al.*, 2007a). Naturally-occurring *Campylobacter* on chicken breasts was transferred to chopping boards (mean 5.8-6.6%), knives (mean 0.03-0.4%) and hands (mean 0.4-1.5%). *Campylobacter* were shown to be subsequently transferred from contaminated chopping boards to sliced tomato (mean 3.6%) and shredded lettuce (mean 20.9%) and from contaminated knives to sliced tomato (mean 32.0%) and shredded lettuce (mean 63.9%).

The use of calculations incorporating contact time, portion weight and initial concentration was advocated for quantitative risk assessment models. However, the conclusion regarding the relationship between transfer rate and initial load has been disputed as an artefact of the data analysis (Nauta, 2010).

The use of tongs during the pan fry cooking of chicken is another recognised vehicle for cross contamination. In a study by Hudson *et al*. (2003), thirty chicken samples were inoculated at 500 cells cm^{-2} with *Campylobacter* and *Salmonella*, then cooked with one turn (using sterile tongs). After cooking, the chicken was transferred using the same tongs to a

sterile surface. The 30 tongs were swabbed and 23.2% were positive for *C. jejuni* by presence/absence testing, 40% were positive for *Salmonella,* and in total 43.3% yielded a pathogen. Out of thirty cooked chicken samples, 36.7% were positive (presence/absence testing) for *Campylobacter* (the same percentage were positive for *Salmonella,* and in total 46.7% of samples yielded a pathogen). The authors concluded that as the use of the same tongs for both raw and cooked meats is a common practice in food preparation and the number of cross contamination events during cooking and preparation is likely to be significant.

In a study simulating the barbecue cooking process, cooked barbecued chicken was handled using tongs that had been used to handle raw chicken (Wong *et al.*, 2007a). The cooked chicken was also returned to a plate which had held raw chicken inoculated with *Campylobacter*. Approximately 10% of the *Campylobacter* on the plate and tongs was transferred to the cooked chicken.

A survey of Dutch consumers in 2005 found that breast fillet was the most popular form of poultry, followed by drumsticks (Bergsma *et al.*, 2007). Stir frying and frying were the most common means of cooking. Consumer cooking methods were replicated in the laboratory, with visual assessment of "doneness". D values derived from these experiments were combined with data on the concentration of *Campylobacter* on retail poultry and recommended cooking times. This assessment concluded that the recommended cooking times were only marginally safe, in terms of the log reduction in *Campylobacter* numbers.

Marinades, particularly low pH (<3) solutions such as wine vinegar, have been shown to be effective in reducing *Campylobacter* counts on breast fillets by approximately 1 $\log_{10}$, but required a time of 3 days (Birk *et al.*, 2010).

Campylobacter survives well on poultry meat at refrigeration temperatures, and there is some evidence that the presence of chicken meat juice and the expression of particular *Campylobacter* genes as a response to the juice can enhance the survival of the microorganisms (Ligowska *et al.*, 2011).

Freezing has been shown to reduce (but not eliminate) the numbers of *Campylobacter* on poultry in several studies, most of which have used inoculated samples. An initial reduction of approximately 1 $\log_{10}$ CFU per g was found for *Campylobacter* numbers on naturally contaminated poultry meat after one day at -22°C, but further reductions over time were not significant, and *Campylobacter* could still be recovered from samples after 84 days (Sampers *et al.*, 2010). Addition of salt (1.5%) to a minced meat preparation had no additional effect on survival of *Campylobacter* during freezing. Cooking experiments conducted as part of this study using pan frying of chicken burgers containing up to 4.5 $\log_{10}$ CFU per g of *Campylobacter* found that numbers declined after 2 minutes (internal temperature approximately 38°C) and had dropped below detectable levels (<10 CFU/g) after 4 minutes (internal temperature approximately 57.5°C).

The effect of freezing on *Campylobacter* numbers has been found to differ depending on the substrate (Ritz *et al.*, 2007). Survival was least on skin, better on skinned muscle, and best on cut muscle. In these experiments, an initial 1–2 $\log_{10}$ CFU per sample drop in *Campylobacter* counts occurred in the first 24 hours, a modest decline after that time, and no further effect was observed after two weeks storage. These data are similar to those found in a study in Iceland (Georgsson *et al.*, 2006). Among five lots of broilers, levels of

Campylobacter on carcasses were reduced by log mean values ranging from 0.65 to 2.87 after freezing to -20°C and 31 days of storage. The level of *Campylobacter* was reduced by approximately one log immediately after freezing and remained relatively constant during the 31-220 days of frozen storage. The levels were constant during 7 days of refrigerated storage.

The use of rapid cooling (at -20°C/min) enhanced the survival of all the *Campylobacter* strains chilled to 4°C compared to standard refrigeration in a domestic refrigerator (El-Shibiny *et al.*, 2009). In this study freezing of poultry meat to -20°C reduced viable counts by 2.2-2.6 $\log_{10}$ CFU per cm^2 in 24 hour.

It has been demonstrated that some strains of *Campylobacter* have a high heat resistance (De Jong *et al.*, 2012). A study examining the heat resistance of selected *Campylobacter* strains belonging to the MLST ST-474, ST-190, ST- 48 and ST-4 (known to be the types most commonly associated with illness in New Zealand) was undertaken. This study investigated the behaviour of these strains in broths and on foods and found that they did not have unusual heat resistance or oxygen tolerance compared to other literature values published for *Campylobacter* (Al Sakkaf *et al.*, 2010). Some differences in the behaviour of *Campylobacter* isolates of clonal complexes 21 and 45 have been found in studies in Belgium (CC21 survived better than CC45 under heat and chill stress, while CC45 survived better under oxidative and freezing stress) (Habib *et al.*, 2010).

7.2 Exposure Assessment: Overseas Context

7.2.1 Broiler flocks

The reported rate of human infection with *Campylobacter* in the Czech Republic is similar to that of New Zealand (see Section 3.4.1). Nationwide monitoring of the prevalence of thermotolerant *Campylobacter* spp. in broilers was initiated in the Czech Republic in September 2005. Cloacal swabs from 10 birds from each flock were pooled and tested using enrichment (Bardoň *et al.*, 2009). In 2006, *C. jejuni* and *C. coli* were detected in 46% and 3% of the tested samples, respectively. In 2007, *C. jejuni* and *C. coli* were found in 43% and 2% of the samples, respectively. As *Campylobacter* are better detected by direct examination of the intestine, from 2008 the Czech monitoring programme changed to taking caecal samples.

A study of risk factors from broiler flock infection in the United Kingdom from 2003 to 2006 (n = 797) found a prevalence of 36% *Campylobacter* in flocks at first full or partial depopulation (Jorgensen *et al.*, 2011). Prevalence was highest in summer months between June and November. Of the *Campylobacter* strains identified, MLST ST45 had the strongest seasonal pattern, peaking in June.

Swedish studies have examined the relationship between *Campylobacter* prevalence before scalding and post-chilling and the importance of choosing an appropriate detection method (Lindblad *et al.*, 2006a). From flocks whose cloacal samples before scalding were negative for *Campylobacter*, the carcasses at the post chill stage that had a *Campylobacter* prevalence of 2% by enrichment, and 10% by direct plating. This difference was partly attributed to inhibition of species other than *C. jejuni* in the enrichment medium, as only 2% of the 10% direct plating results were found to be *C. jejuni*. Positive carcasses from flocks with negative cloacal results were found to carry only about 3 $\log_{10}$ CFU *Campylobacter* per carcass,

compared to carcasses from positive flocks, which carried about 5 $\log_{10}$ CFU *Campylobacter* per carcass.

A survey comparing organic and conventional production of broilers and turkeys in the US collected intestinal tracts from the start of processing (Luangtongkum *et al*., 2006). The prevalence of *C. jejuni* and *C. coli* plus other *Campylobacter* species in conventionally raised broilers was 227/345 (66%), while the prevalence of these microorganisms in conventionally raised turkeys was 299/360 (83%). The prevalences of *Campylobacter* spp. in organically raised broilers and turkeys were 317/355 (89%) and 201/230 (87%), respectively.

A study in Germany from May 2004 to April 2005 (northern hemisphere spring), using caecal samples, found that of the 146 flocks tested, 44% were *Campylobacter*-positive and most were infected with *C. jejuni* (Näther *et al.*, 2009). A higher *Campylobacter* prevalence was found during the months of May to October (52%; northern hemisphere summer-autumn). Caecal testing of New Zealand flocks (now discontinued) showed no temporal trends.

8 APPENDIX 2: EVALUATION OF ADVERSE HEALTH EFFECTS

While much remains to be understood, an alternative view of the pathogenic mechanisms of *Campylobacter* infection has been proposed (Wassenaar, 2011). The search for virulence genes and pathogenicity factors (such as toxins) that are conserved amongst pathogenic strains has been described as largely unsuccessful. Instead, symptoms of enteritis may result from a local over-reaction of the intestinal innate immune system. When invasive and motile *Campylobacter* cells penetrate the intestinal mucus layer, they are engulfed by the intestinal cells, a process which ultimately leads to the release of cytokines, part of the immune response. The cytokines are mostly responsible for the symptoms of diarrhoea (van Putten *et al.*, 2009). This suggests that most, if not all *Campylobacter* strains are able to cause disease.

It has also been suggested that the differences observed in the prevalence of *Campylobacter* strains amongst human disease may be due to the superior ability to some strains to survive in the environment and have an opportunity to cause infection, rather than the presence of specific virulence factors in those strains (On *et al.*, 2006).

In developed countries campylobacteriosis affects all age groups, with slightly elevated rates for young children and young adults. The predominant symptoms are diarrhoea, abdominal cramps, fever, and bloody stools. In developing countries, the predominant symptom is watery diarrhoea, and campylobacteriosis occurs predominantly in infants. This has been considered to provide evidence for protection from clinical disease, perhaps from protective immunity acquired over time with repeated exposure (Havelaar *et al.*, 2009). Alternatively, the reduced rate of symptoms in adults in developing countries may be due to dampening of the innate immune response (Wassenaar, 2011).

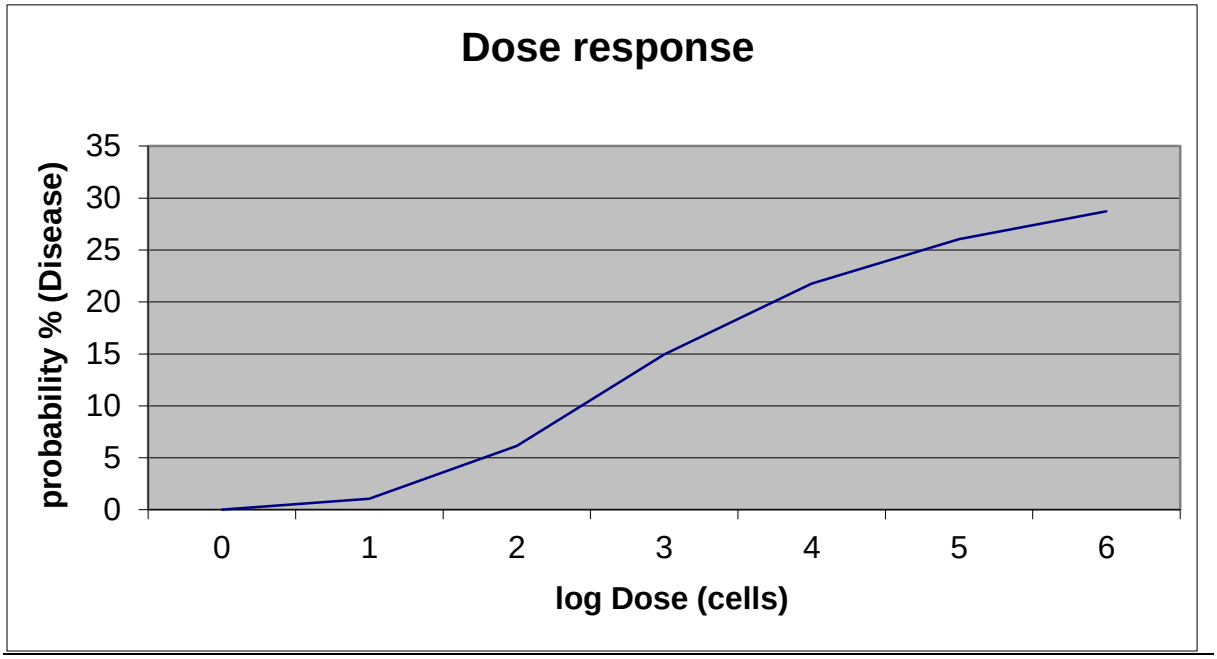
A study of *Campylobacter* isolates from human cases in Scotland used Penner serotyping data to examine the age distribution of types (Miller *et al*., 2005). The reduced occurrence of infection from the common serotypes with age supported the hypothesis of increased immunoprotection in the older population. However, it is possible that changing exposure to *Campylobacter* with age would confound the results.

Reported campylobacteriosis is associated with a summer peak. Investigations in England and Wales for reported cases from 1990 – 1999 showed that increased campylobacteriosis rates were found to be correlated with temperature (Louis *et al*., 2005). The most marked seasonal effect was observed for children under the age of 5 years. The seasonal pattern of *Campylobacter* infections indicated a linkage with environmental factors rather than food sources.

8.1 Dose Response

To give an idea of the probability of human disease given a variety of doses, Figure 7 illustrates the results from application of the FAO/WHO model using a fixed 33% probability of developing disease after infection has occurred.

Figure 7: FAO/WHO dose response model; probability fixed at 33%



One of the two strains of *C. jejuni* used in the original dose-response challenge trial was 81-176 (Black *et al.*, 1988). The problem with using this strain is that it produces a molecule (a ganglioside) which is thought to be a cause of the severe sequel of infection, GBS. Consequently another strain, CG8421 has been characterised, which does not produce this molecule and so is considered a safer strain for use in challenge trials (Tribble *et al.*, 2009). A trial of 23 subjects who received 1 x 10^6 or 1 x 10^5 CFU of *C. jejuni* found that 100% and 93% of subjects became ill (i.e. attack rate) respectively. The model constructed from these data was used to estimate that an attack rate of 75% required some 4 $\log_{10}$ fewer CFU than estimated from the 81-176 trial. Further studies with this strain found that subjects given a repeat exposure after 28-49 days had complete protection while attenuated illness was found for subjects challenged again after one year. This demonstrates that without repeated exposure immunity wanes over time (Tribble *et al.*, 2010).

8.2 Types Causing Neurological Disease

Certain serotypes of *C. jejuni*, particularly Penner Serotype O19 and O41 have been more frequently associated with GBS than other serotypes (Wallace, 2003). Penner Serotype O19 has been associated with GBS in Japanese studies. However, this link was not confirmed in a US case control study, in which no specific serotypes were associated with GBS (Rees *et al.*, 1995). Similar results were found in a Dutch/Belgian molecular epidemiological study, that found a wide range of *Campylobacter* types associated with GBS cases (Endtz *et al.*, 2000).

Certain types of lipopolysaccharides on the exterior of *Campylobacter* cells have been linked to GBS and Miller Fischer syndrome (a clinical variant of GBS). A study in the United States has found that commercial poultry products carry a relatively high prevalence of *C. jejuni* strains that express these molecules and have been associated with neuropathic sequelae (Hardy *et al.*, 2011).

8.3 Adverse Health Effects in New Zealand

8.3.1 Anti-microbial resistance

A study of the anti-microbial resistance of 193 New Zealand isolates of *C. jejuni* (n=193) originating from retail poultry carcasses in 2005-2006 showed that the majority of isolates (99%) were fully susceptible to the six drugs that were tested (Pleydell *et al.*, 2010). One of 193 *C. jejuni* isolates was resistant to erythromycin, and microbroth dilution assays confirmed that this panel of *C. jejuni* was generally susceptible to antibacterial drugs.

8.3.2 Case control studies and risk factors

Two major New Zealand case control studies of campylobacteriosis have been published in the scientific literature in the 1990s. Details of these studies were given in the previous Risk Profile.

8.4 Adverse Health Effects Overseas

8.4.1 Outbreaks

In May/June 2005 an outbreak of diarrhoeal illness occurred among company employees in Copenhagen (Mazick *et al*., 2006). Cases were reported from seven of eight companies that received food from the same catering kitchen. Stool specimens from three patients working for two companies were positive for *C. jejuni*. A retrospective cohort study found that of 247 employees who ate canteen food, 79 were cases, and the attack rate (AR) was 32%. Consuming canteen food on 25 May was associated with illness (AR 75/204, RR=3.2, 95%CI 1.3-8.2). Consumption of chicken salad on this day, but not other types of food, was associated with illness (AR=43/97, RR=2.3, 95%CI 1.3-4.1). Interviews with kitchen staff indicated the likelihood of cross-contamination from raw chicken to the chicken salad during storage. The low number of positive specimens identified in this outbreak suggests a general under-ascertainment of adult cases in the laboratory reporting system by a factor of 20.

8.4.2 Case control studies

Case control studies of campylobacteriosis conducted overseas since 2005 have been summarised in Table 8.

Table 8: Case control studies containing information on *Campylobacter* in poultry since 2005

Country	Risk/Protective factor	Odds ratio (CI)	Reference
Australia	Chicken, cooked Chicken, undercooked	1.4 (1.0 – 1.9) 4.7 (2.6 – 8.4)	(Stafford *et al.*, 2008)
Australia	Restaurant prepared chicken	2.3 (1.5 – 3.5)	(Unicomb *et al.*, 2008)
Denmark	Eating fresh unfrozen chicken	5.8 (2.1 – 15.9)	(Wingstrand *et al.*, 2006)
Ireland	Consuming chicken	6.8 (2.1 -21.9)	(Danis *et al.*, 2009)

Country	Risk/Protective factor	Odds ratio (CI)	Reference
Netherlands	Chicken consumption April – December January - March	 1.4 (1.2 – 1.8) 3.0 (1.8 – 5.1)	(Doorduyn *et al.*, 2010)
UK	Regularly ate chicken (at least once a week) Do not regularly eat chicken, but ate chicken in previous 5 days Regularly ate chicken, and eating commercially prepared chicken in the previous 5 days Regularly ate chicken and eating home prepared chicken in previous 5 days	1.6 (1.2 – 2.0) 5.0 (2.1–11.9) 4.0 (2.8 -5.8) 1.5 (1.1 – 2.1)	(Tam *et al*., 2009)

CI = confidence interval

The large case control study in the UK during 2005 – 2006 (2,381 cases and 5,256 controls) found that chicken consumption was strongly associated with infection, as was the use of acid suppressing medication (see Table above) (Tam *et al.*, 2009). The population attributable risk (PAR) of chicken consumption was 41%.

PAR estimates have been calculated from an Australia case-control study conducted in 2001 – 2002 amongst people ≥5 years of age (Stafford *et al.*, 2008). Overall, the PAR for chicken consumption (both cooked (21.2%) and undercooked (8.1%)) was 29.3%. However, the size of this estimate has been challenged, on the basis of the marginal statistical significance of consumption of cooked chicken as a risk factor (OR 1.4, 95% CI 1.0 – 1.9) (Gillespie, 2009).

The case–control study in the Hunter region of New South Wales, Australia investigated 354 cases and 593 controls for potential risk factors for domestically acquired *Campylobacter* illness (Unicomb *et al*., 2008). Risk factors for 0-4 year olds, and older people were examined separately, but for the younger age group there were no risk factors examined that were associated with increased risk of disease. As shown in Table 8, restaurant prepared chicken was associated with risk of infection in older people, and the PAR was estimated as 9.9%.

The case-control study in the Netherlands found differing risk factors for different age groups (Doorduyn *et al*., 2010). With a PAR of 28%, consumption of chicken was the most important risk factor, followed by consumption of meat prepared at a barbecue, grill or microwave oven (12%), eating in a restaurant (10%) and consumption of undercooked meat (9%). Less important risk factors were consumption of steak tartare (3%) and undercooked seafood (4%). Of the non-food factors, strong associations were found for use of proton pump inhibitors, occupational exposure to raw meat and having one of the following chronic intestinal illnesses: inflammatory bowel disease (IBD), irritable bowel syndrome (IBS) or coeliac disease. However, because only limited numbers of cases were exposed to these risk factors, the corresponding PARs were relatively low. Ownership of dogs, especially several young dogs, and ownership of cats were identified as risk factors with relatively low PARs.

For young children (0–4 years) and the elderly (>60 years) consumption of undercooked meat and meat prepared at a barbecue, grill or microwave oven remained risk factors in age specific models. Visiting farm animals, contact with persons with gastroenteritis symptoms and ownership of farm animals were predominant risk factors for *C. jejuni* enteritis in young

children: an estimated 19%, 12% and 9% of the cases in this age group were attributable to these factors, respectively. Consumption of products containing raw egg was a unique risk factor for young children and was not associated with illness in any other age group. Predominant risk factors for *C. jejuni* enteritis in the elderly were eating in a restaurant (PAR 19%), use of proton pump inhibitors (PAR 14%) and having a chronic intestinal illness (PAR 14%). Consumption of ready-to-eat sandwiches was a unique risk factor for the elderly.

A case control study in Denmark found that risk factors for young children differed from those for adults (Ethelberg *et al.*, 2005). Living in types of housing found in rural areas and living in areas with a low population density were both associated with an increased risk of infection. This relation concerned children in particular and explained one third of cases among children in the countryside. Furthermore, in some counties there was an association between infection and the drinking-water company serving the home. This study indicated that contact with animals or the environment is the source of a substantial proportion of sporadic *Campylobacter* infections in the Danish countryside, particularly among children. These results are consistent with differing urban-rural epidemiologies of campylobacteriosis, some evidence for which has been found in New Zealand (Garrett *et al.*, 2007).

Different risk factors for young children have also been found in a US study using the FoodNet surveillance network (Fullerton *et al.*, 2007). Infants 0-6 months of age with *Campylobacter* infection were less likely to be breast-fed than controls [odds ratio (OR); 0.2; 95% confidence interval (CI), 0.1-0.6]. Risk factors for infants 0-6 months of age included drinking well water (OR 4.4; CI, 1.4-14) and riding in a shopping cart next to meat or poultry (OR 4.0; CI, 1.2-13.0). Risk factors for infants 7-11 months of age included visiting or living on a farm (OR 6.2; CI, 2.2-17), having a pet with diarrhoea in the home (OR 7.6; CI, 2.1-28) and eating fruits and vegetables prepared in the home (OR 2.5, CI 1.2-4.9). *Campylobacter* infection was associated with travel outside the United States at all ages (OR 19.3; CI, 4.5-82.1).

A telephone survey of people in FoodNet sites in the US from 2006 – 2007 established that 13.6% of children younger than 3 years were exposed to raw meat or poultry while riding in shopping carts during shopping (Patrick *et al.*, 2010). This could include exposure to *Campylobacter*.

In contrast to other case-control studies, a study in France did not find that cases were more likely to report poultry consumption than controls (Gallay *et al.*, 2008). Instead, eating undercooked beef, eating at a restaurant, and poor utensil hygiene in the kitchen were the main independent risk factors.

8.4.3 Risk assessment and other activity

The FAO/WHO (2002) have published a quantitative risk assessment that deals with the hazard identification, hazard characterization and exposure assessment for *Campylobacter* in broilers. Much of the information presented is based on the poultry industry in the UK, but there is significant commonality between the processes described and what occurs in New Zealand. The document presents a detailed description of the process and explains the modelling used at each step. Some aspects of this model have been published (Hartnett *et al.*, 2001).

A study of the incidence of *Campylobacter* in broilers and humans, and the seasonal variation and long-term trends in longitudinal surveillance data in six Northern European countries (Denmark, Finland, Iceland, Norway, Sweden and the Netherlands) has been reported (Jore *et al.*, 2010). The incidence of *Campylobacter* colonization in broiler flocks and incidence of campylobacteriosis in humans showed a concordant seasonality for all the countries. There was a strong association between the incidence in both broilers and humans in a given month and the mean temperature of the northern hemisphere in the same month, as well as the preceding month.

A model of *Campylobacter* transmission between and within broiler flocks on farms has evaluated control options (Katsma *et al.*, 2007). The efficacy of three control scenarios was evaluated; (1) a ban on other livestock on broiler farms, (2) a ban on thinning, and (3) a reduction of the between-flock transmission. The third option was shown to be most effective, and theoretically, this is accomplished by improved biosecurity. However, the impact of improved biosecurity could not be specified into specific control measures.

A quantitative risk model of broiler chicken processing has been published for the Netherlands (Nauta *et al.*, 2005; Nauta *et al.*, 2007). Other models examine the concentration of *Campylobacter* as they pass through each processing stage, and model changes based on microbiological data. The Dutch model has a mechanistic basis and includes transfer coefficients of bacteria from the poultry skin and intestines to the processing environment and from the environment back to the skin. This approach is considered better able to predict the effects of risk management interventions as it includes non-linear effects (Nauta *et al.*, 2009a). The difficulty of this approach is the shortage of data to parameterise the model. Expert judgement was used to fill data gaps (Van Der Fels-Klerx *et al.*, 2005).

An update to the Dutch model has been published which takes advantage of more recent microbiological data and Bayesian analysis to generate better parameter estimates (Kurowicka *et al.*, 2010). Extension of the mechanistic modelling approach to food preparation involving chicken has been reported (Mylius *et al.*, 2007). This model indicated that cross contamination contributed significantly to the risk from *Campylobacter*, and cleaning frequency of kitchen utensils and thoroughness of rinsing of raw food items (e.g. salads) after preparation were important model components contributing to risk of infection. To improve the parameter estimates for this model, transfer rates for various single food preparation steps have been examined in another Dutch study (Van Asselt *et al.*, 2008).

Another model reported by Dutch researchers has estimated that the mean transfer rate of *Campylobacter* from inoculated chicken breast fillets to chicken salads prepared according to a method allowing cross contamination is 0.12% (Verhoeff-Bakkenes *et al.*, 2008). The chicken salad dish recipe involved boiling chicken and mixing it with chopped fruit. Replacing the cutting board and cutlery after handling raw chicken and the prevention of hand contact considerably reduced final contamination levels. Transfer rates for the individual steps were reported.

A quantitative risk model to investigate campylobacteriosis associated with poultry in Denmark has been developed (Rosenquist *et al.*, 2003). The model suggests that logistic slaughter (i.e. slaughtering negative flocks before positive flocks) would have only a minor effect. A Danish model of the consumer phase of the poultry food chain has also been published (Christensen *et al.*, 2005). Monte Carlo simulations showed that the probability of ingesting a risk meal was highest for young males (aged 18-29 years) and lowest for the

elderly above 60 years of age. This was principally due to differences in hygiene levels and behaviour.

An examination of the *Campylobacter* serotypes, ribotypes and PFGE types in Denmark found considerable overlap between the types found in human cases and those found in food (66% overlap), broiler chickens (59%), and cattle (83%) (Nielsen *et al.*, 2006). Travel associated cases had a higher diversity of types, and *C. coli* was more common (10%) in these cases than in infection acquired domestically (3%).

A quantitative risk model of the German poultry food chain between retail and domestic consumption has been created (Brynestad *et al.*, 2008). Reducing the load of *Campylobacter* on chicken was found to be more important in reducing risk than lowering prevalence of contamination.

Bayesian analysis of data from Finland comparing MLST types of isolates from human cases, bovine and poultry samples showed that 44.3% of the human isolate types were found in bovine-associated clusters and 45.4% of the human isolate types were found in the poultry-associated cluster (De Haan *et al*., 2010a). This contrasted with other countries where poultry was the dominant source, and the difference was attributed to the low prevalence of *C. jejuni* in poultry flocks in Finland (6.5% in 2003). A longitudinal analysis of isolates from human cases and poultry sources found that the annual overlap between STs from human and chicken isolates decreased from 76% at the beginning of the study period (1996) to 58% at the end (2006) (De Haan *et al*., 2010b). A study of PFGE *Campylobacter* types found in poultry and cattle in Finland, observed that there were a number of cattle associated types found in human infections, but these types were not found on beef carcasses and so transmission pathways other than food consumption were considered likely (Hakkinen *et al.*, 2009).

A Swedish model of the consumer end of the poultry food chain has estimated the effectiveness of intervention options (Lindqvist and Lindblad, 2008). For *Campylobacter* spp. prevalence but not concentration, there was a one-to-one relation with risk. The effect of a 100-fold reduction in the numbers of *Campylobacter* spp. on raw chicken reduced the risk by a factor of 12 (fresh chicken) to 30 (frozen chicken). Highly-contaminated carcasses contributed most to risk and it was estimated that by limiting the contamination to less than 4 $\log_{10}$ CFU per carcass, the risk would be reduced to less than 17% of the baseline scenario. Diverting all positive flocks to freezing was estimated to result in 43% as many cases as the baseline. The second best diversion option (54% of baseline cases) was to direct all chickens from the two worst groups of producers, in terms of percentages of positive flocks delivered, to freezing. Improvements in consumer handling had considerable potential to reduce risk but were acknowledged as challenging to achieve.

A review and comparison of six models for *Campylobacter* in broiler meat was published in 2009 (Nauta *et al.*, 2009a). The models were the FAO/WHO model, and country specific models from the United Kingdom, Denmark, Germany, the Netherlands (see above) and New Zealand. The paper considered that all models predicted a negligible effect of logistic slaughter and that the most effective intervention measures aim at reducing the *Campylobacter* concentration, rather than reducing the prevalence. Cross-contamination was generally considered to be more relevant than undercooking. An important finding was that the tails of the distributions describing the variability in *Campylobacter* concentrations

between meat products and meals determine the risks, not the mean values of those distributions.

The relative importance of risk factors (poultry consumption, travelling abroad, pet contact, and "other") has been evaluated for age groupings (0-4 years, 5-19 years, 20-34 years, 35-59 years, >60 years) in a study in Switzerland (Buettner *et al*., 2010). The analysis was conducted using exposure assessment and the results of a case-control study. Overall the relative importance of poultry consumption was greatest for the 5-19 and >60 years age groups, and was 27% (95% CI 17–39) for the total population.

The Joint FAO/WHO Expert Meetings on Microbiological Risk Assessment (JEMRA) has developed a decision support tool for the management of *Campylobacter* and *Salmonella* in chicken meat has been developed to support a risk based approach to the management of these pathogens and can be used in conjunction with the Codex *Guidelines for the Control of Campylobacter and Salmonella in Chicken Meat* (Codex, 2011).[27] The on-line tool is designed to compute the residual risk between a baseline process flow and a process flow applying selected interventions as outlined in the guidelines. The residual risk measure may be used to evaluate the overall effectiveness of the applied interventions. The process flow can be customised by the user to represent their particular situation.

[27] http://www.mramodels.org/poultryRMTool/ Accessed 28 March 2013

9 APPENDIX 3: CONTROL MEASURES

9.1 Current Risk Management Measures

9.1.1 Legislation

9.1.1.1 The Animal Products Act

The *Animal Products Act 1999* regulates the processing of animal material into products for use, trade, and export through managing associated risks and facilitating overseas market access.[28]

The Act requires all animal products traded and used to be "fit for intended purpose". The main means for ensuring that animal products are fit for their intended purpose is by requiring that the processing of animal materials and products occurs under a registered risk management programme. Poultry processors must operate under a risk management programme, and Part 2 of the Act provides for the registration and verification of these risk management programmes (Section 9.1.2.1).

Part 3 of the Act provides for the setting of regulated control schemes where risk factors cannot be managed under risk management programmes, or where special provision is required for overseas market access. The Animal Products (Regulated Control Scheme – Contaminant Monitoring and Surveillance) Regulations 2004 provides for the monitoring of agricultural compounds, veterinary medicines and environmental contaminants in poultry.

Part 4 of the Act provides for the setting of standards that must be met before an animal product can be considered fit for intended purpose, and for the setting of any specifications necessary to ensure the standards are met. The New Zealand animal product standards are contained in the Animal Products Regulations 2000 (Section 9.1.1.2) and the Australia New Zealand Food Standards Code (Section 9.1.1.3).

9.1.1.2 Animal Products Regulations

The Animal Products Regulations 2000 set out animal product standards and provide for the setting of specifications.[29] Section 6(1) requires that, taking into consideration its intended use, animal products must be free from biological, chemical, and physical hazards in amounts that may be directly or indirectly harmful to humans or animals. However, specifications can be set that specify what hazards are unacceptable in relation to any type of animal product (e.g. raw or ready-to-eat poultry), and the acceptable or unacceptable levels of these hazards (Section 6(2) of the regulations).

9.1.1.3 Australia New Zealand Food Standards Code

Chapters 1 and 2 of the Australia New Zealand Food Standards Code contain many requirements that are applicable to the poultry industry (e.g. requirements for labelling (Part 1.2 and Standard 2.2.1) and substances added to food (Part 1.3), limits for fluid loss

[28] The Act may be viewed at http://www.legislation.govt.nz (accessed 9 March 2011).
[29] The Regulations may be viewed at http://www.legislation.govt.nz (accessed 9 March 2011).

(Standard 2.2.1)).[30] Standard 1.6.1 sets out the microbiological limits for specific food products. No limits have been set for *Campylobacter* in poultry or poultry products.

9.1.1.4 Animal Products Notices

The *Animal Products Act 1999* provides for the issuing of notices.[31] The Animal Products (Specifications for Products intended for Human Consumption) Notice 2004 applies to risk management programme operators who are processing animal material or animal product intended for human consumption, i.e. poultry primary processors. The Notice (and subsequent amendments) sets out requirements for how these facilities should be designed and maintained, and how they should operate, including detail such as the maximum chilling (7°C) or freezing (-12°C) temperatures, water quality monitoring and transportation.[32] Other Notices relevant to poultry production include:

- Animal Products (Contaminant Monitoring and Surveillance) Notice 2012, covering residue monitoring requirements;
- Animal Products (Contaminant Specifications) Notice 2008, specifying residue limits for animal products, including poultry;
- Animal Products (National Microbiological Database Specifications) Notice 2012, specifying sampling and testing requirement for particular microbiological contaminants;
- Animal Products (Specifications for the Ante-mortem And Postmortem Examination of Poultry Intended for Human or Animal Consumption) Notice 2005, specifying inspection requirements for poultry processors;
- Animal Products (Requirements for Risk Management Programme Outlines) Notice 2008, covering the required content of risk management programmes; and
- Animal Products (Risk Management Programme Specifications) Notice 2008, covering specifications, recordkeeping and amendment with respect to risk management programmes.

9.1.1.5 Code of Welfare for fully housed broilers

The Animal Welfare (Broiler Chickens: Fully Housed) Code of Welfare 2003 was issued under the *Animal Welfare Act 1999* by the National Animal Welfare Advisory Committee (NAWAC). Under this Act, codes of welfare set by the NZWAC are deemed to be regulations and can contain minimum standards that have legal effect. Codes of welfare may also contain recommended practice and recommended best practice that are not legally binding.

The Animal Welfare (Broiler Chickens: Fully Housed) Code of Welfare 2003 (NAWAC, 2003) applies to all persons responsible for the welfare of broiler chickens in controlled environment broiler production systems, i.e. the chickens are kept in enclosed housing and are reliant on human management for all their daily requirements. There are no specific standards for *Campylobacter* spp., but many of the standards will help control this

[30] The Australia New Zealand Food Standards Code is available at http://www.foodstandards.gov.au/foodstandards/foodstandardscode.cfm (accessed 9 March 2011).
[31] All Notices can be viewed at http://www.foodsafety.govt.nz/industry/general/animal-products/documents/specs.htm
[32] The 2004 Notice and amendments can be viewed at http://www.foodsafety.govt.nz/elibrary/industry/animal-products-specifications-asd/index.htm

microorganism (e.g. all hatcheries must have a documented cleaning, sanitising and hygiene programme, housing systems must be vermin-proof, and other than in some exceptional circumstances litter must be replaced after every growing cycle).

9.1.2 Mandatory requirements

9.1.2.1 Risk management programmes

The *Animal Products Act 1999* defines a risk management programme (RMP) as a programme designed to identify and control, manage, and eliminate or minimise hazards and other risk factors in relation to the production and processing of animal material and animal products in order to ensure that the resulting animal product is fit for intended purpose. RMPs must manage risks from hazards to human health, animal health, false or misleading labelling and risks to the wholesomeness of animal material or product (NZFSA, 2009).

A RMP is 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. The Act requires that RMPs are tailored for each animal product business according to the animal materials used, the processes performed and the product range produced. Operators must build any relevant regulatory limits (e.g. microbiological limits) into their RMP, but can also set their own measurable limits to ensure the food is safe and fit for purpose.

Primary processors of poultry must have a RMP in place, and so must secondary processors of poultry unless they are covered by a food safety plan under the *Food Act 1981* and its subsequent amendments or operate under the *Food Hygiene Regulations 1974* and are registered with their local council. Poultry producers (i.e. broiler farms) and transporters of poultry to primary processing facilities do not need to have a RMP (NZFSA, 2009).

The operator of the primary or secondary processing facility is responsible for developing and registering their RMP but the programmes are subject to independent verification. A generic RMP for the slaughter and dressing of broilers was issued in 2002 to support operators to develop their own RMPs (NZFSA/PIANZ, 2002).[33] In this document, *Salmonella* is frequently used as an example of an identified hazard that requires control.

9.1.2.2 National microbiological database (NMD) programme

The National Microbiological Database (NMD) Programme is a Government programme applied to industry that monitors animal carcass hygiene after processing. For poultry this includes tests for *Salmonella* spp. and *Campylobacter* spp. Premises operating to process broiler chickens must have the NMD programme in place. Further details and recent results are presented in Section 2.6.2.

[33] The generic RMP is available at http://www.foodsafety.govt.nz/industry/general/rmp/documents/rmp-generic/ (accessed 9 March 2011).

9.1.3 Non-mandatory guidelines and codes of practice

9.1.3.1 Ministry of Health criteria (1995)

The New Zealand Ministry of Health has published microbiological criteria for foods intended as a guide for food producers where no mandatory standard exists (Ministry of Health, 1995). There are microbiological criteria for *Campylobacter* in poultry products and for generic categories of foods that will include poultry products. The criteria for *Campylobacter* are listed in Table 9.

Table 9: Ministry of Health microbiological reference criteria applicable to *Campylobacter* in poultry products

Product		***Campylobacter* per:**	**Criteria***		
			n	**c**	**m**
Poultry	Raw	10 g or whole bird carcass rinse	5	2	0
	Nuggets, patties, etc. requiring further cooking (> 70°C)	10 g	5	1	0
	Cooked	10 g	5	0	0
	Cured and/or smoked	10 g	5	0	0
Meat and meat products	Chopped, minced or manufactured meat – uncooked	10 g	5	1	0
	Manufactured, cured or fermented meat - ready-to-eat	10 g	5	0	0
	Meat paste or spread - including pâté	10 g	5	0	0
	Hot smoked	10 g	5	0	0
	Vacuum packed - semi-preserved but perishable products	10 g	5	0	0
Foods – cooked, ready-to-eat (or with subsequent minimal heating < 70°C)	All components cooked in manufacturing process	10 g	5	0	0
	Some components not cooked in manufacturing process (e.g. sandwiches)	10 g	5	0	0

* n = the minimum number of sample units that must be examined from a lot of food; c = the maximum allowable number of defective sample units; m = the acceptable microbiological level in a sample unit (values above it are marginally acceptable or unacceptable).

9.1.3.2 FSANZ guidelines (2001)

FSANZ has produced generic guidelines for the microbiological examination of ready-to-eat foods that apply to foods sampled at the point of sale or distribution to consumers (FSANZ, 2001). Under these guidelines, *Campylobacter* spp. should not be detected in a 25 g sample of a ready-to-eat food.

9.1.4 New Zealand poultry industry interventions

9.1.4.1 Code of practice for poultry processors

The NZFSA (and now MPI), in consultation with the Poultry Industry Association of New Zealand (PIANZ), have been developing a code of practice for poultry primary processors. The purpose of the code of practice is to help poultry processors meet the requirements of the Animal Products Act 1999 (Section 9.1.1.1) and risk management programmes (Section 9.1.2.1), and to produce poultry products for human and animal consumption that are safe and suitable for their purpose (NZFSA, 2007).

Development of the code of practice was begun in 2007 and several chapters have been released.[34] Once complete, the code of practice will cover good manufacturing practice and process control, HACCP application, and the identification and control of risk factors related to wholesomeness and labelling. Once complete, the code of practice will replace the 1998 Poultry Industry Processing Standard 5 (PIPS 5), published by the Poultry Industry Standards Committee.[35] PIPS 5 sets the minimum standards for producers of poultry products for human consumption with the aim of minimising the potential food safety hazards associated with poultry, based on HACCP principals.

The code of practice does not specifically address *Campylobacter* in poultry products. However, many of the good manufacturing practices will help to reduce any *Campylobacter* contamination on poultry carcasses, and prevent cross-contamination (e.g. wash steps, equipment cleaning and maintenance).

9.1.4.2 Broiler growing biosecurity manual

The Broiler Growing Biosecurity Manual describes the recommended minimum standards to be used in New Zealand's broiler production systems (PIANZ, 2007). One of the Manual's biosecurity objectives is to minimise the incidence and spread of organisms of public health concern (*Salmonella*, *Campylobacter*, haemolytic *E. coli*). The Manual covers the set-up and operation of production facilities, management of personnel, and controls over inputs such as water and feed or potential routes of contamination such as vehicles and wildlife. These practices will help to control *Campylobacter* spp. contamination during poultry production. Each poultry company has its own biosecurity manual and these incorporate aspects of the Broiler Growing Biosecurity Manual (Michael Brooks (Executive Director, PIANZ), pers. comm., 23 May 2011).

9.1.4.3 Poultry industry agreed standards and codes of practice

The 1995 Poultry Industry Agreed Standards and Codes of Practice (PIANZ, 1995) have been superseded by the Broiler Growing Biosecurity Manual, the NZFSA code of practice and the risk management programmes (Michael Brooks (Executive Director, PIANZ), pers. comm., 23 May 2011).

[34] The code of practice is available at http://www.foodsafety.govt.nz/elibrary/industry/processing-code-practice-poultry/index.htm accessed 2 November 2011

[35] PIPS5 is available from http://www.foodsafety.govt.nz/industry/sectors/meat-ostrich-emu-game/meatman/pips5/ accessed 2 November 2011.

9.1.5 Review of *Campylobacter* in poultry risk management interventions

Risk management to control human exposure to *Campylobacter* from poultry may take place on farm, during slaughter/processing, or else during handling in domestic or foodservice environment. Interventions may target factors contributing to broiler infection, or else treatments to reduce contamination once it has occurred.

A recent review of the poultry food chain in terms of potential control measures has been published, along with the status of research into their implementation (Wassenaar, 2011). Briefly the options are:

On the farm:

- Biosecurity (control of human activity, training of personnel, decontamination of rooms, boots, equipment and vehicles)
- Insect exclusion (flies)
- Vaccination
- Competitive exclusion
- Phage therapy

Transport and primary processing

- Decontamination of transport crates, vehicles, boots, workers before progression to another farm
- Optimisation of individual processing steps for reduced prevalence and concentration
- Logistic slaughter (processing uninfected flocks before infected flocks)
- Product channeling (i.e. uninfected flocks used for fresh product, infected flocks used for frozen product and secondary processing)

Secondary processing and retail

- Decontamination after processing (forced air chilling, crust freezing, steam, ultrasound, etc.)
- Packaging (leak proof packaging, packing under gas mixtures containing oxygen to inhibit *Campylobacter*)

Kitchens

- Consumer hygiene and prevention of cross contamination
- Adequate cooking (to achieve at least a 7 $\log_{10}$ reduction in *Campylobacter* counts)

9.1.6 On farm control

Three general strategies have been proposed to control *Campylobacter* in poultry at the farm level: (1) reduction of environmental exposure (biosecurity measures), (2) an increase in poultry's host resistance to reduce *Campylobacter* carriage in the gut (e.g., competitive exclusion, vaccination, and host genetics selection), and (3) the use of antimicrobial alternatives to reduce and even eliminate *Campylobacter* from colonised chickens (e.g., bacteriophage therapy and bacteriocin treatment). Except for biosecurity measures, the other intervention approaches are currently not commercially available and are still under development (Lin, 2009).

Bacteria may enter the flock environment from a variety of sources: contaminated water, feed, domestic/wild animals including pests such as flies, transport crates, vehicles, personnel etc. Although a number of authors have investigated the potential for vaccination, an effective vaccine strategy directed against *Campylobacter* in broiler chickens has yet to be developed. The challenges have been identified as: (1) the identification of novel cross-protection-inducing antigens, (2) the induction of a rapid, potent immune response, and (3) the development of novel adjuvants to further stimulate immunity against *Campylobacter* (de Zoete *et al*., 2007).

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

Control measures introduced to control *Salmonella* in broilers in the United Kingdom and New Zealand have included treatment of feed, biosecurity in the hatchery, in the feedmill and on the farm, *Salmonella*-free parent and grandparent flocks, vaccination of breeders and competitive exclusion. While these measures appear to be effective in controlling *Salmonella*, similar measures appear to be ineffective against *Campylobacter* (Corry and Atabay, 2001). The use of dedicated boots for each poultry house and the regular use of foot dips have been found to be important factors in preventing the introduction of *Campylobacter* in broiler flocks. Flies have been identified as a potential vector for introduction of *Campylobacter* into broiler houses (Hald *et al.*, 2004). Installation of fly screens in 20 Danish broiler houses in 2006 resulted in a lower prevalence of positive flocks (15.4%) than in control houses with no screens fitted (51.4%) (Hald *et al.*, 2007). However, even with the most stringent biosecurity measures, infection appears to be impossible to prevent completely. Once infection has entered the chicken house, most or all birds become *Campylobacter* carriers very quickly (Pattison, 2001).

Feed withdrawal is another on-farm control aimed at minimising cross contamination of bacteria through the spillage of gut contents and faeces during processing. Fasting periods of 8 hours are the standard in New Zealand, while overseas they can be between 7 and 20 hours once catching, transportation and lairage are taken into account. This does not necessarily mean that longer fasting periods are beneficial. Stress may also predispose the fasting birds to *Campylobacter* infection. One study has shown that the longer the fasting period (up to 24 hours), the higher the prevalence of *C. jejuni* in crop samples before slaughter (Byrd *et al.*, 1998).

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

- Hygienic routines when farm workers enter the rearing room;
- Avoiding partial slaughter of flocks (i.e. partial depopulation or thinning);
- 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 reduced from 50% to 10%.

The effect of enhanced biosecurity related to thinning has been studied on 21 farms in the UK (Ridley *et al.*, 2011a). The enhanced biosecurity measures included: (1) cleaning and disinfection of all vehicles entering the farm site (2) provision of a mobile mess/changing room for the catching crew, including handwashing and sanitation facilities, and (3) a requirement for catchers to bring fresh clothing, dedicated footwear, facemasks and gloves. A high proportion (up to 60%) of samples from vehicles, crates, and personnel tested before enhanced biosecurity were positive for *Campylobacter*. There was a significant reduction in positive samples after enhanced biosecurity was implemented, particularly for samples from transport lorries. However, samples from catcher vehicles (e.g. forklifts) did not show a decrease in prevalence. The effect on flocks was also negligible. Of the flocks that were negative before thinning, all were positive at final clearance, despite the enhanced biosecurity. PFGE typing of isolates showed that 38% of strains that appeared in flocks were also isolated from crates and modules. The need for improved practices associated with footwear (better cleaning regimes and boots designed for easier cleaning) was identified. Thinning of flocks is apparently essential for the UK poultry industry, and this study has shown the difficulty of managing this risk factor for flock infection.

The addition of silver ions to the polymer used to make transport crates has been shown to reduce the concentration of *Campylobacter* (and other microbes) on the crates during use, thus contributing to reduced cross contamination (Hastings *et al.*, 2011).

The difficulty of containing *Campylobacter* within a broiler house has been highlighted by another UK study of a single farm across 15 grow-out cycles (Ridley *et al*., 2011b). There was no environmental location that was consistently positive while the flock was negative, indicative the absence of a potential persistent source. However, overall the longitudinal observations suggested that cattle housed in the yard adjoining the broiler chicken farm may have constituted a reservoir (i.e., a site of amplification) for certain *Campylobacter* strains. The prevalence of contamination in environmental samples increased markedly, once a flock became contaminated, including approximately half of the aerosol samples which were all negative prior to colonisation. Molecular typing showed that strains from house surroundings and an adjacent dairy farm were similar to those subsequently detected in the flock and that several strains intermittently persisted through multiple crop cycles.

Competitive exclusion studies have used adult chickens found to be *C. jejuni*-free (Zhang *et al.*, 2007). The best chickens were identified by challenging with 6 $\log_{10}$ CFU *C. jejuni* per chicken and determining undetectable caecal shedding of *Campylobacter* at 4 weeks. Screening of bacterial colonies obtained from nine donor chickens yielded isolates inhibitory to six *C. jejuni* strains *in vitro*. Of these, the most strongly inhibitory were *Lactobacillus salivarius*. Another strain of this species of bacteria, and the bacteriocin it produces, had been identified as inhibitory to *C. jejuni* in a Russian study (Stern *et al.*, 2006; Svetoch *et al.*, 2011).

A study of three broiler farms in France found that sanitary barriers kept the chickens *Campylobacter* spp. free until they had access to an open outside area (Rivoal *et al.*, 2005). They were then rapidly colonized by the *Campylobacter* strains isolated from the soil.

A number of studies have investigated phage control for *Campylobacter* in broilers (e.g. (Wagenaar *et al.*, 2005), both the inhibit colonisation and to reduce numbers. However, no commercially available product is yet available.

9.1.7 Control during or after processing

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

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

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

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

The first three of these measures are widely used in New Zealand.

In a US study, the ability of carcass washes to reduce *Campylobacter* contamination was shown to be very dependent on additives in the water (Bashor *et al.*, 2004). Wash water containing 25-35 ppm chlorine resulted in an average decrease in *Campylobacter* concentrations of 0.5 $\log_{10}$/ml of carcass rinse (100 ml phosphate-buffered peptone water). Washer systems with trisodium phosphate (TSP) or acidified sodium chlorite (ASC) reduced *Campylobacter* populations on average by an additional log 1.03 to log 1.26, respectively.

The effect of freezing on *Campylobacter* numbers on carcasses post-chill has been studied in the context of the Norwegian Action Plan against *Campylobacter*, where carcasses from flocks identified as positive before slaughter are either heat treated or frozen for 5 weeks before consumption (Sandberg *et al.*, 2005). Overall a 2 $\log_{10}$ CFU reduction in *Campylobacter* numbers was found after freezing at -20°C for 3 weeks, and only a marginal extra effect was achieved by extending the freezing to 5 weeks. In 80% of the samples, *Campylobacter* could still be detected after 120 days (17 weeks).

New Zealand studies on freezing

A project on the effect of freezing and chilling temperatures on *Campylobacter* on poultry meat included a literature review, a survey of industry "crust freezing" techniques and experiments to determine the effect of different freezing rates and temperatures on the reduction of *Campylobacter* numbers (Whyte *et al.*, 2005). The literature review concluded that the freezing rate influenced *Campylobacter* survival more strongly than the period of frozen storage. Slow freezing was more lethal than rapid freezing because of osmotic stress. Very high rates of freezing (in excess of 10°C/min) can reduce bacterial survival by creating intracellular ice crystals and subsequent mechanical cell damage, though these rates are difficult to achieve in industry. Overall, the literature suggested that *Campylobacter* was reasonably tolerant of chilling but reductions could be made if an optimum freezing rate and temperature was used.

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

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

Further New Zealand experiments on the effect of freezing have used locally isolated strains common amongst human cases (McIntyre, 2008). The reduction of two *Campylobacter jejuni* strains, ST3609 and ST 474, was determined on skin-on chicken breast portions frozen to a defined internal temperature of -12°C followed by frozen storage at -12°C for up to 73 days. Significant but variable reductions in *C. jejuni* numbers were observed over time for both strains, although *C. jejuni* was still detectable on samples after 73 days of storage. Overall, mean *C. jejuni* populations declined by approximately 1.2 to 2.2 $\log_{10}$ CFU over a 1 week storage period, while reductions of 3.3 and 4.1 $\log_{10}$ CFU were achieved after 34 and 73

days of frozen storage, respectively. These mean reductions were of similar magnitude to those determined previously under both simulated domestic and commercial conditions, and suggest that the longer the frozen storage period, the better the pathogen reduction achieved. While freezing has a significant impact on the reduction of *C. jejuni* on poultry, the reductions obtained in this work are likely to be the maximum possible given that *C. jejuni* survives better on muscle than on chicken skin (Ritz *et al.*, 2007).

It should be noted that another study reported better survival of *Campylobacter* on skin than meat during storage under a mixture of chilled (4ºC) and frozen (-3ºC) conditions (Davis and Conner, 2007).

Logistic slaughtering

Logistic slaughtering, where *Campylobacter* positive flocks are processed at the end of the day, has often been suggested as a risk management option. Up to 2005, this was practised in Norway. It was stopped partly because modelling studies suggested that the effect on reduced prevalence would be low. To examine this conclusion, a study was made of the extent to which *Campylobacter* from a positive flock was transmitted to the next flock through cross contamination during the slaughtering process (Johannessen *et al.*, 2007). Cross contamination was demonstrated to occur, based on the same amplified length fragment polymorphism (AFLP) type from infected preceding flocks being isolated from uninfected following flocks. However, the extent of this cross contamination was limited. Of the three carcasses sampled from each flock, only the carcass from the start of the uninfected flock was positive for the type of *Campylobacter* from the preceding flock, and the numbers were low. Carcasses taken from the middle and end of these flocks were negative. Although the number of carcasses tested was very low, these results support the limited effectiveness of logistic slaughter.

A similar study has been conducted using PFGE typing to examine links between *Campylobacter* types found in flocks (Potturi-Venkata *et al.*, 2007). Where a flock that had negative results for *Campylobacter* (based on faecal testing) was processed after a flock that was faecal positive, the same *Campylobacter* PFGE type was found on the carcasses from the faecal negative flock. These results were based on rinsates from each of ten carcasses from each flock; however, it was not clear whether the carcasses were sampled randomly, or whether all of the carcasses from the negative flocks were positive from cross contamination. The mean counts on the cross contaminated carcasses were lower than the mean of counts from faecally positive flocks, but the difference was small (0.1 $\log_{10}$ CFU per carcass) in some trials, and in a single trial both the first and second flocks after the positive flock were apparently cross contaminated. The data from this trial suggested that logistic slaughter might have more value than determined by Norwegian study.

Scheduling

A risk management approach based on the diversion of flocks with high numbers of *Campylobacter* away from fresh meat production to alternate channels such as frozen or pre-cooked product, has been reviewed (Nauta *et al.*, 2009b). The implementation of such a measure would require a rapid on-site test to detect high numbers of *Campylobacter*. In this trial as lateral flow immunoassay was developed and used to test faecal samples. In a separate report, the performance of this test was evaluated, and the detection probability was shown to rise from 0% at approximately 5 $\log_{10}$ CFU per g of faeces, and rise steeply to

100% at approximately 7.5 $\log_{10}$ CFU per g (Evers *et al.*, 2009). However, in the scheduling trial, poor correlation between the rapid test and traditional microbiological methods, and limited correlation between *Campylobacter* contamination of caecal samples and breast meat samples, meant that the effectiveness of the planned scheduling approach was compromised.

9.2 Decontamination During Processing

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 kGy has been approved by the FDA in the US and several other countries (Jacobs-Reitsma, 2000), but is not permitted in New Zealand.

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

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

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

A review from a US perspective has been carried out on commercial antimicrobials by Oyarzabal (2005). Acidified sodium chlorite (ASC) dips were especially effective when applied post-chill, reducing both prevalence and concentration markedly (Oyarzabal *et al.*, 2004). The ASC SANOVA® produced by Ecolab, (Alcide Corporation, Redmond, Wash.) has FDA approval and is used in industry in the US. ASC combines with organic matter producing several broad spectrum oxychlorous antimicrobial compounds. These oxidize

sulphide and di-sulphide bonds on cell membrane surfaces. ASC is sprayed or used in a dip solution before the prechill or chill tank stage. Concentrations are between 500 and 1200 ppm, acids used are generally recognised as safe (GRAS) such as citric acid (final solution pH 2.5 to 2.9). The author cites several studies that found the combination of bird washers with ASC sprays removed faecal contamination (a primary source of contamination). Carcasses processed on-line in this manner had a reduction of *Campylobacter* of 1.75 $\log_{10}$ CFU/ml of carcass rinse compared to off-line reprocessing (Kere-Kemp *et al.*, 2001). In another study, reductions up to 99.2% in *Campylobacter* numbers were achieved by the combined effect of bird washers and ASC sprayed prechill (Kere-Kemp and Schneider, 2002). Post-chill dipping with ACS was also more effective in decreasing *Campylobacter* prevalence and counts on broiler carcasses than inside-outside bird washes or chilling (Oyarzabal *et al.*, 2004) It is suggested that the application of ASC exerts indirect stress on the *Campylobacter* cells during the subsequent chilling process.

In Australia, a small trial using ASC dip (SANOVA®) on poultry carcasses has been carried out to determine its effectiveness (Sexton *et al.*, 2007). *Campylobacter* was one of the pathogens evaluated. Thirty carcasses from a known positive flock were selected for treatment. Six at a time were placed into clean plastic crates and completely immersed in a 600 litre solution of SANOVA® (concentration 900-1000 ppm sodium chlorite and pH 2.5 – 2.6) for 20 seconds. A control of 30 birds were also collected and bagged. The concentration of sodium chlorite remained at 960 ppm before and during the trial, reducing to 949 ppm after the trial. The *Campylobacter* prevalence reduced from 30/30 (100% - untreated controls) to 7/30 (23%) and was statistically significant ($p < 0.0001$). The mean log of *Campylobacter* numbers on the positive carcasses reduced by 3.8 log (from 39 per cm^2 to 0.006 per cm^2), this equates to a count of 75,660 CFU reducing to 12 CFU on a 1.5 kg carcass. Organoleptic assessments were favourable, despite a bleached appearance immediately after treatment, pink colouration returned within a day and taste testers unable to detect any taste or visual differences. Shelf life on the controls was 12 – 13 days and treated carcasses 14 days. The only visual difference recorded was a darkening on the wingtip extremity of one of the treated carcasses (Sexton *et al.*, 2006).

A FAO/WHO Expert Meeting on the risks and benefits of chlorine-containing antimicrobials concluded that there was evidence for a reduction of pathogens on poultry carcasses by application of acidified sodium chlorite and chlorine dioxide (FAO/WHO, 2009). There was also evidence that no pathogen reduction is achieved by application of sodium hypochlorite on poultry carcasses. Limited data provided evidence for reduction of cross-contamination by the application of disinfectants (in particular, sodium hypochlorite) in wash and flume waters. The identified residues of chlorine-containing disinfectants and disinfection by-products did not raise health concerns based on estimated dietary exposures. Potentially dietary exposure and toxicological evaluation of ACS has also been carried out by the Joint FAO/WHO Expert Committee on Food Additives (JECFA) (FAO/WHO, 2008).

A comparison of the effect of forced air chilling, crust freezing, and a steam-ultrasound commercial process on *Campylobacter* numbers post-processing has been published (Boysen and Rosenquist, 2009). Although each technique resulted in significant reduction in numbers, none were as effective as freezing. The same study also examined the effect of visceral rupture during evisceration. This was found to result in an increase of 0.9 $\log_{10}$ CFU per carcass.

Inactivation of *Campylobacter* by heat and high hydrostatic pressure has been investigated (Lori *et al.*, 2007). Steam treatment has been found to be effective, but application of steam for longer than 12 seconds was found to cause the skin to shrink and change colour (James *et al.*, 2007).

Ultraviolet light has been shown to be effective in reducing concentrations of *Campylobacter* on poultry meat (by 1-2 $\log_{10}$ CFU per g) and packaging (by approximately 4 $\log_{10}$ CFU per cm^2) (Haughton *et al.*, 2011).

Campylobacter bacteriophages may also be used to control the bacterium on carcasses. The first reported applications of phages to poultry skin for the control of *Campylobacter* were published in 2003. In one study the effectiveness of phages to reduce populations of *S.* Enteritidis and *C. jejuni* applied separately to chicken skin was evaluated (Goode *et al.*, 2003). Skin was inoculated at levels of ~10^3 and 10^4 CFU per cm^2 and phages applied at 10^3 and 10^6 plaque forming units (pfu) per cm^2, respectively, and the samples stored at 4°C for up to 48 h. In the presence of phages, host counts were reduced by over 2 $\log_{10}$ CFU per cm^2 (99.5%) after 24 h incubation.

A similar approach was adopted when investigating the reduction of *C. jejuni* on chicken skin using a different phage. Skin sections (2 cm^2) were inoculated with host cells and phages applied at a number of concentrations to create a range of phage:pathogen ratios. Treated and untreated skin samples were incubated at either 4 or -20°C for up to 5 days, and counts made on the entire skin sample. Reductions in the number of *C. jejuni* in the presence of phage were measurable when phages were present at a concentration of 10^7 pfu and the host was present at 10^6 and 10^4 CFU, with reductions of 1.1-1.2 $\log_{10}$ CFU occurring after 1 day (Atterbury *et al.*, 2003).

Survival under chilled conditions (4-5ºC) of different *C. jejuni* strains exposed to different gas mixtures usually used for gas packaging (70/30% O_2/CO_2, and 70/30% N_2/CO_2) of food was examined (Boysen *et al.*, 2007). When inoculated onto chicken fillets, the *C. jejuni* strains died significantly faster in the oxygen-containing gas mixture, reaching reductions of 2.0–2.6 log10 CFU/g after 8 days. In the gas mixture without oxygen (70/30% N_2/CO_2), no reductions were observed.

9.3 Kitchens

Kitchen hygiene measures recommended by the UK Advisory Committee on the Microbiological Safety of Food (ACMSF, 2004) includes active discouragement of washing poultry and meat (wipe with paper towel if necessary). Splashing of rinse water is considered to distribute *Campylobacter* in the kitchen.

Studies that mimic food preparation and cleaning behaviours in domestic kitchens have found that the method used to clean cutting boards is a key part of preventing cross contamination (De Jong *et al.*, 2008). In trials using rinsing with hot or cold water, and washing with soap, the most important factor in reducing the number of cells on the cutting boards appeared to be the exposure time to hot water (68°C). Shorter rinsing times, even with washing with soap, were less effective, and rinsing with cold water did not affect contamination levels of prepared salads. The use of different cutting boards for raw meat and other ingredients was recommended, as was thorough washing of hands.

The Codex *Guidelines for the control of Campylobacter and Salmonella in chicken meat* includes consumer level Good Hygienic Practice (GHP)-based and hazard based control measures (Codex, 2011). These include discouraging washing of raw chicken in the kitchen, washing and disinfection of kitchen surfaces after raw chicken preparation and cooking by a process that achieves at least a 7 Log_{10} reduction in both *Campylobacter* and *Salmonella*.

9.4 Codex Guidelines

In 2011, Code release *Guidelines for the control of Campylobacter and Salmonella in chicken meat* (Codex, 2011). The Guidelines were developed to provide information to governments and industry on the control of these microbial pathogens in chicken meat, to reduce foodborne disease from this source while ensuring fair practices in international food trade. The Guidelines contain both Good Hygienic Practice (GHP)-based and hazard-based approaches to controlling *Campylobacter* and *Salmonella* in chicken meat, and covers the food chain from grandparent flock management to consumer handling and cooking. The identified steps in the food chain and control measures specific to *Campylobacter*, or suggested for both *Campylobacter* and *Salmonella* are summarised in Table 10.

Table 10: Food chain stages and control measures for *Campylobacter* (Codex, 2011)

Food chain step	GHP-based controls	Hazard-based control
Manage grandparent flocks	Application of combination of biosecurity and personal hygiene measures	
Transport eggs to hatchery		
Parent hatchery		
Transport day-old chicks to parent farm	Personnel involved with chick transport should not enter any livestock buildings and should prevent cross-contamination during loading and unloading	
Manage parent flocks	Application of combination of biosecurity and personal hygiene measures	
Transport eggs to hatchery		
Hatchery		
Transport day-old chicks to grower sheds	Personnel involved with chick transport should not enter any livestock buildings and should prevent cross-contamination during loading and unloading. Live bird transport crates and modules cleaned, disinfected and dried to greatest extent possible	
Manage chickens	Application of combination of biosecurity and personal hygiene measures. Pest control programmes should be designed for local conditions	Use of fly screens. Has been shown to decrease *Campylobacter*-positive flocks from 51.4 to 15.4%
Depopulation (partial or full)	Full depopulation if possible. If not, attention to strict biosecurity and hygiene of catchers and their equipment. Scheduling of sheds for partially depopulation ahead of those for full depopulation on a particular day. If feed withdrawal used, use water additives	

Food chain step	GHP-based controls	Hazard-based control
	(e.g. lactic acid) to lower post-harvest crop contamination	
Transport to slaughterhouse	Live bird transport crates and modules cleaned, disinfected and dried to greatest extent possible	
Receive at slaughterhouse	Where possible, information about the *Campylobacter* status of flock provided in a timely manner to allow logistic slaughter and/or channelling of poultry meat to treatment Where practical, flocks to be slaughtered after 8-12 hours of feed withdrawal. Minimise chicken stress (dim lighting, minimal handling, avoid delays)	
Ante-mortem inspection	Moribund, unhealthy or otherwise unsuitable chickens not processed. Where numbers dead, moribund, unhealthy or unsuitable at receipt exceed expectations, processor to notify relevant responsible person	
Slaughter	Positive flocks diverted for specific processing or treatment Minimise bird stress at live hanging Bleeding to be substantially complete before scalding	
Dress - general	Minimise contamination of carcasses by: • Washing with abundant potable water • Trimming • Disposal or reprocessing of carcasses with extensive faecal contamination • Use of approved chemical	

Food chain step	GHP-based controls	Hazard-based control
	decontaminants • Use of approved other physical methods Control measures may be applied alone or in combination Any carcass re-hang done mechanically All chickens that drop to the floor to be condemned or reprocessed	
Dress - scalding	Minimise contamination of carcasses by: • Use of counter-current flow • High water flow rates with adequate agitation • Optimum scalding temperature • Use of approved chemical, such as pH regulators Other design factors include: • Degree of agitation • Multi-staged tanks • Pre-scald wash systems • Raising temperature during processing breaks to kill pathogens in scalders • Scald tanks empties and cleaned at end of processing period • Scald tanks cleaned and disinfected at least daily • Hygiene measures applied to reused/recycled water	
Dress - defeathering	Cross-contamination can be minimised by: • Fasting of chickens pre-slaughter • Prevention of feather build-up on	

Food chain step	GHP-based controls	Hazard-based control
	equipment • Continuous rinsing of equipment and carcasses • Regular adjustment and maintenance of equipment, with particular attention to cleaning moving parts, and regular inspection and replacement of plucker fingers	
Dress – head pulling	Should be carried out in a manner that prevents leakage from the crop	
Dress – evisceration	Minimise viscera rupture and faecal spread by: • Limiting size variation in birds in batches • Careful adjustment and regular maintenance of equipment	
Dress – crop removal	Where possible, crops should be extracted in a manner that limits carcass contamination	
Inside/outside wash	Inside and outside of carcasses should be thoroughly washed, with sufficient pressure to remove visible contamination. Equipment should ensure direct water contact with the carcass. Inclusion of a brushing apparatus may aid the process	Carcass washing systems with 1-3 washers, using water with 25-35 ppm chlorine, shown to reduce *Campylobacter* by 0.5 $\log_{10}$ CFU/ml carcass rinse. Post wash sprays with ACS or TSP may further reduce *Campylobacter* by an average of 1.3 $\log_{10}$ CFU/ml or 1.0 $\log_{10}$ CFU/ml carcass rinse, respectively
On-line reprocessing		Spray systems with ASC has been shown to reduce *Campylobacter* by 2.1 log10 CFU/ml carcass rinse. Dipping in 10% TSP reduced *Campylobacter* on neck skin by 1.7 $\log_{10}$ CFU/g
Post-mortem inspection	Line speed and lighting should be appropriate	

Food chain step	**GHP-based controls**	**Hazard-based control**
	for effective inspection	
Chill carcass (air or immersion)	Chicken meat should be chilled as quickly as possible, with system design to ensure target temperature is achieve before carcasses exit the chiller	
Chill carcass – air	If water sprays are used to prevent desiccation they should be arranged to minimise cross-contamination	Forced air chilling may decrease *Campylobacter* by 0.4 $\log_{10}$ CFU/carcass
Chill carcass - immersion	Where necessary, processing aids may be added to chiller water. These may include: • Free chlorine • Organic acids • Other oxidants Chlorine may not directly decontaminate carcasses, but may inactivate *Campylobacter* washed off the carcass Water should be potable, chilled counter-current and may be agitated Excess water should be allowed to drain from the carcass following chilling to minimise subsequent cross-contamination	Immersion chilling has been shown to reduce *Campylobacter* by 1.1-1.3 $\log_{10}$ CFU/ml carcass rinse
Post-chill applications		Immersing carcasses in ASC (600-800 ppm, pH 2.5-2.7, 15 seconds) has been shown to reduce *Campylobacter* by 0.9-1.2 $\log_{10}$ CFU/ml carcass rinse
Portion		
Pack whole carcass or portions	Care should be taken to minimise external pack contamination (absorbent pads, leakproof packaging Pre-packed chicken for cooking by consumers	Various doses of gamma rays or electron beams applied to warm, chilled or frozen carcasses have been shown to eliminate *Campylobacter*

Food chain step	GHP-based controls	Hazard-based control
	should be labelled with safe handling, cooking and storage instructions	
Chill/freeze		Freezing followed by 31 days of frozen storage (-20ºC) has been shown to reduce *Campylobacter* by 0.7-2.9 $\log_{10}$ CFU/g Crust freezing of skinless breast fillets has been shown to reduce *Campylobacter* by 0.4 $\log_{10}$ CFU/fillet
Storage		
Transport		
Wholesale premises		
Transport		
Retail/Food service – retail	Hygiene measures should be in place to prevent cross-contamination between raw chicken meat and other foods Retailers should separate raw and cooked products Hands should be washed and sanitised after handling raw chicken meat packs Packs for consumer selection should be leak-proof. Extra packaging should be provided to allow customers to separate chicken from other purchases	Chicken meat should be cooked by a process capable of achieving at least a 7 $\log_{10}$ reduction in *Campylobacter*
Retail/Food service – food service	Thawing of frozen chicken should be carried out in a manner that minimises microbial growth potential and prevents cross-contamination. Washing of raw chicken should be avoided, as this is likely to spread contamination Operators should be trained in separation of raw and cooked chicken products	Chicken meat should be cooked by a process capable of achieving at least a 7 $\log_{10}$ reduction in *Campylobacter*

Food chain step	GHP-based controls	Hazard-based control
	Operators should have in place hygiene measures that minimise cross-contamination from raw chicken to hands, surfaces, utensils and other foods	
Transport		
Consumer	Consumer education should focus on handling, hand washing, cooking, storage, thawing, prevention of cross-contamination and prevention of temperature abuse Special attention should be paid to educating food preparers, particularly those preparing for YOPIs Information to consumers should be provide through multiple channels Washing of chicken in the kitchen should be discouraged to minimise potential for cross-contamination. If necessary, washing should be carried out in a manner that minimises cross-contamination Consumers should wash and disinfect food contact surfaces after raw chicken preparation	Chicken meat should be cooked by a process capable of achieving at least a 7 $\log_{10}$ reduction in *Campylobacter*

ASC = acidified sodium chlorite
TSP = trisodium phosphate
YOPI = young, old, pregnant or immunocompromised

9.5 Interventions in Specific Countries

United Kingdom

A joint government and industry target to reduce *Campylobacter* in the United Kingdom was announced in January 2011.[36] The target is to reduce *Campylobacter* contamination on whole chickens in UK slaughterhouses and will be based on *Campylobacter* counts in individual chickens rather than overall prevalence in the birds because higher counts are associated with increased public health risk. The target will be set at the end of primary processing.

The aim of the target is to reduce the levels of the most highly contaminated chickens at the end of the slaughter process (post chill). The target will be monitored using a banding approach, where samples are grouped into 3 bands according to whether the *Campylobacter* counts in chicken are above or below a set level (i.e. <100 CFU per g, 100-1,000 CFU per g, and >1,000 CFU per g).

The UK target for reduction of *Campylobacter* is a reduction in the percentage of chickens produced in UK poultry slaughterhouses that have the highest level of contamination, i.e. those with more than 1,000 CFU per gram, from a baseline of 27% in 2008 to 10% by 2015, measured post-chill.

The target will be achieved through the implementation of interventions along the chicken production chain. A phased approach has been agreed, with initial interventions focusing on primary production whilst interventions at the slaughterhouse and retail points are further developed and trialled.

A decline in the incidence of reported campylobacteriosis in Scotland (10% nationally) between 2001 and 2006 has been investigated to determine the role of poultry (Gormley *et al.*, 2008). There was no statistically significant change in the prevalence or counts of *Campylobacter* on poultry samples taken in each year (prevalence in 2001 was 80.9%, while in 2006 it was 90.4%). There were changes in the distribution of MLST types found in chicken, although the association between MLST type and pathogenicity is uncertain. This study did not find evidence for the role of chicken in the decline in notifications. However, the size of the decline was smaller than in New Zealand, and not apparently accompanied by interventions in the poultry supply.

Norway

An action plan against *Campylobacter* in broilers was launched in 2001 (Hofshagen and Kruse, 2005). This had three parts:

1. Surveillance of all Norwegian broiler flocks slaughtered before 50 days of age;
2. A follow-up advisory service for farms delivering *Campylobacter* positive flocks; and,
3. Surveys of broiler meat products at the retail level.

[36] http://www.food.gov.uk/multimedia/pdfs/campytarget.pdf accessed 16 September 2011

Between 2002 and 2004, 4.8% of flocks were positive, and the flock prevalence declined over this period from 6.3% in 2002 to 3.3% in 2004. A decline in the proportion of farms delivering *Campylobacter* positive flocks was also noted (28.4% to 17.8%). The temporal association between these changes and the initiation of the programme was taken as evidence for its effectiveness. The prevalence of *Campylobacter* contaminated meat products at retail did not decline as consistently (2002 8.1%, 2003 5.0%, 2004 5.1%). This was attributed partly to non-random sampling.

Iceland

A major risk management study of the entire production chain for poultry in Iceland has been carried out by Icelandic scientists, the USDA Agricultural Research Service and the Canadian Food Inspection Agency. Iceland has a closed system for poultry production and consumption. Prior to 1996, only frozen poultry was available. The introduction of chilled poultry in 1996 and steadily increasing consumption was paralleled by increases in reported rates of campylobacteriosis: 1997: 13.7 per 100,000 population, 1998: 52 per 100,000, 1999 116 per 100,000. A high proportion (90%) of *Campylobacter* isolates from humans were genetically indistinguishable from those occurring in the poultry.

A dramatic reduction in cases of campylobacteriosis occurred in 2000, with notified cases reduced to 33 per 100,000. On-farm biosecurity measures and a public education campaign introduced in 2000 were partly acknowledged for the reduction together with a targetted freezing regime. This involved testing four week old flocks for the bacterium, and where positive flocks were identified, the carcasses from the processing lot were frozen prior to distribution. As farmers received lower prices for the frozen commodity, there was an incentive to improve on-farm biosecurity measures (Stern *et al.*, 2003). Accompanying studies of risk factors for broiler infection have been published (Guerin *et al.*, 2007b).

The types of *Campylobacter* in poultry batches (caecal and retail samples) and human cases in Iceland have been compared using *fla*A short variable region (SVR) typing (Callicott *et al*., 2008). The study was conducted from 2001–2004. A total of 1,617 isolates from 327 individual product lots were genetically matched to 289 isolates from cases of human campylobacteriosis whose onset was within approximately 2 weeks from the date of processing. In all, 84% of human isolates had genotypes seen in broiler-associated isolates collected during processing and post processing. The matching process also showed that there were apparent temporal clusters of cases with identical types that were associated with the types found in product lots.

US

The United States Department of Agriculture (USDA) has published a compliance guideline for controlling *Salmonella* and *Campylobacter* in broilers, aimed principally at small and very small poultry processing plants (USDA, 2010). This describes optimal physical and chemical parameters to control the pathogens at each step of primary processing, with references to studies indicating their effectiveness.

Also announced by the USDA in 2010 were new performance standards for *Salmonella* and *Campylobacter* in broilers[37,38]. The standard for *Campylobacter* in broilers will employ the 400 ml rinsate sample from carcasses at both rehang and post-chill, as used in the existing programme monitoring *Salmonella* in broilers. A 1 mL portion will be plated for both qualitative (presence/absence) and quantitative (enumeration) results. A 30 mL portion will be first enriched and then plated for qualitative (presence/absence) results only. The performance standard itself is set at the prevalence found in a baseline survey, the Young Chicken Baseline Survey (YCBS). The standard is for sample-specific positive results, which is the YCBS estimated sample-specific prevalence for 1 mL and 30 mL results combined, at 46.7 percent with no more than 27 of 51 samples positive.

Comments on the new standards were due by July 13 2010. A summary of comments and responses was published in March 2011[39]. The new standards have been implemented from July 2011.

Netherlands

Hypothetical risk based standards for *Campylobacter* in broilers have been explored in a study based on information from the Netherlands (Nauta and Havelaar, 2008). Microbiological criteria are proposed which are assessed using a quantitative risk model and linked to acceptable levels of protection.

A cost-utility analysis of interventions to control *Campylobacter* on broilers in the Netherlands investigated direct intervention costs and disability adjusted life years (DALYs) avoided (Mangen *et al.*, 2007). Three interventions showed favourable cost-utility ratios: limiting faecal leakage during processing, carcass decontamination by dipping in a chemical solution, and phage therapy. Further analysis of intervention options confirmed these options, and added flock scheduling (separation of meat from contaminated and uncontaminated flocks) as a promising intervention (Havelaar *et al.*, 2007).

Sweden

A monitoring programme for *Campylobacter* in broiler chickens was initiated in 2001, and a review of data up to 2005 has been published (Hansson *et al.*, 2007a). Sampling was of 40 cloacal swabs per batch taken after stunning and bleeding. The annual incidence of positive slaughter batches decreased from 20% on 2002 to 13% in 2005. A summer peak in prevalence was observed. The decline in incidence was attributed to increased farmer knowledge of the importance and use of hygiene barriers. Approximately 40% of producers seldom delivered *Campylobacter* positive flocks, indicating that infection free broilers can be consistently produced.

Belgium

An economic analysis of potential interventions to control *Campylobacter* in the Belgian poultry meat chain used a published model to estimate the health benefits in reduced costs of

[37] http://www.usda.gov/wps/portal/usda/usdahome?contentidonly=true&contentid=2010/05/0246.xml accessed 4 September 2011
[38] http://www.fsis.usda.gov/OPPDE/rdad/FRPubs/2009-0034.pdf accessed 4 September 2011
[39] http://www.fsis.usda.gov/oppde/rdad/frpubs/2010-0029.pdf accessed 4 September 2011

illness and industry data to estimate the cost of implementing the intervention (Gellynck *et al.*, 2008; Hartnett *et al.*, 2001). The benefit:cost ratio was greatest for decontaminating carcasses with electrolysed oxidising water applied in the processing plant, followed by the use of lactic acid, and phage therapy. However these technologies appeared to need greater development (Kim *et al.*, 2005). Although irradiation was effective, the cost was high due to the need to send product to a specialised treatment plant. Interventions at the consumer level were considered ineffective due to the difficulty in achieving change through mass media campaigns. The paper includes a comment that in Denmark, marketing of *Campylobacter* free product has not been successful due to consumer unwillingness to pay a premium for such product.

Denmark

In 2003, the Danish voluntary strategy to control *Campylobacter* was intensified (Rosenquist *et al*., 2009). The focus was on biosecurity, allocation of meat from *Campylobacter*-negative broilers to the production of chilled products, and consumer information campaigns. From 2002 to 2007, the percentage of *Campylobacter*-positive broiler flocks at slaughter decreased from 43% to 27%. After processing, *Campylobacter*-positive samples of chilled broiler meat fell from 18% in 2004 to 8% in 2007. Furthermore, the number of registered human *Campylobacter* cases decreased by 12%.

The process for identifying and implementing interventions was guided by extensive risk assessment and modelling. Initial interventions were introduced on farms (intensified education of broiler producers in hygiene barriers), at retail (monitoring and production and launch of a *Campylobacter*-free frozen broiler), and at the consumer level (consumer information on bacteria present in broiler meat and guidance on kitchen hygiene via consumers' magazines and leaflets in supermarkets). Then in 2003 additional interventions were introduced. These included:

On-farm:

- Reinforcement of the compliance with the industry code of practice (hygiene and biosecurity)
- Increased bonus for *Campylobacter*-negative broiler flocks and/or for compliance with the industrial code of practice
- Limitation of partial slaughter and/or research in methods to improve biosecurity during this procedure

Processing:

- Channelling of *Campylobacter*-negative flocks to fresh, chilled meat production and *Campylobacter*-positive flocks to frozen production (as much as possible)
- Logistic slaughter

Retail:

- Promotion of the production and sale of *Campylobacter*-free chilled broilers

Consumer:

- Information campaign aimed specifically at young people focusing on how to handle poultry products in domestic kitchens

Although a drop in the number of reported cases was observed, this was modest. This was attributed partly to the volume of imported poultry meat (60-80% of the total market), which is a key difference between Denmark and countries where greater reductions in cases have been achieved, such as New Zealand and Iceland which do not permit poultry imports. In addition, limitations in testing methodology reduced the effectiveness of channelling in Denmark.