TRANSMISSION ROUTES FOR CAMPYLOBACTERIOSIS IN NEW ZEALAND

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by

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Amendment of report of December 2005

This report contains an amendment to the information provided in the December 2005 report. This concerns the number of New Zealanders on unregistered water supplies. The December 2005 report indicated that this represented 3.7% of the population; in fact, the number is approximately 11%. This number has been changed, but the remainder of the report, and its conclusions, remain the same.

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SUMMARY

Campylobacteriosis is a major public health problem in New Zealand. Based on notification rates, the reported incidence of the illness is high compared to other developed countries, and has generally increased over the last 10-15 years. The objective of this project is to prepare a review document identifying, to the greatest extent possible, the identity and relative importance of different transmission routes for *Campylobacter* infection in New Zealand. The report contains a comprehensive review of information from New Zealand, and selected information from overseas reports.

The high probability of infection at relatively low doses of *Campylobacter* means that the level of contamination of a potential vehicle need not be high to cause illness. This suggests that the route and frequency of exposure, and size of the population who are exposed, are as important as the prevalence of contamination.

The pattern of campylobacteriosis notifications in New Zealand is not markedly different to overseas, apart from the reported rate.

Age and sex distribution, the summer peak, and sporadic nature of the illness are similar to overseas, and this suggests that the overall epidemiology is likely to be similar to that in other developed countries. Hospitalisation and mortality data do not suggest higher pathogenicity of *Campylobacter* strains infecting people in New Zealand. The predominant serotypes amongst isolates from human cases are the same as in the UK.

The high reporting rate in New Zealand may be due to factors within the surveillance system. A corollary of this would be a lower reported:unreported case ratio. Support for this possibility comes from the observation that cases of GBS have not increased in recent years, which suggests that total cases of campylobacteriosis are not sufficient to markedly affect the GBS rate.

Generally there appears to be relatively little difference in notified campylobacteriosis rates between rural and urban regions in New Zealand. Rural cases represent a small proportion (8-12%) of the total reported cases.

The delineation of rural and urban populations on the basis of residence may create an artificial difference that is not reflected in differences in exposures. It seems likely that in New Zealand rural exposures, particularly animal contact, are experienced by many in the "semi-urban" residential population through occupational and social exposures.

Nevertheless, the data showing similar rural and urban relative rates of reported illness suggest that risk management measures most likely to cause a decline in campylobacteriosis rates should be directed towards the risk factors identified from studies that have concentrated on the urban population principally.

Many *Campylobacter* infections are acquired during international travel. In New Zealand, the percentage of cases (of those for whom the information is known) reporting overseas travel prior to illness ranges from 6-12%. Overseas travel was a significant risk factor in the national case-control study, and reported by 5% of cases. It seems reasonable to assign an important but minor proportion of the campylobacteriosis incidence to this risk factor.

Although person-to-person transmission is considered unusual for this illness, contact with a sick person appeared as a statistically significant risk factor in both case-control studies. It may be that this risk factor is under-appreciated, but still was reported by only a small proportion of cases.

Poultry consumption has been identified as a risk factor in case control studies in both New Zealand and developed countries overseas. Supporting evidence for the importance of poultry as a transmission vehicle includes:

- the high prevalence of contamination of retail poultry products in comparison to other meat types, and other foods;
- the apparently higher number of bacteria present on contaminated poultry, compared to other meat types,
- consumption of poultry by a high proportion (approximately 20%) of the population on a daily basis;
- increasing consumption of poultry, and an increasing trend towards fresh product, over the past 10-15 years, during which time campylobacteriosis notification rates have increased (the initiation of availability and increased sales of fresh rather than frozen poultry appears to have been one of the factors behind the sharp increase in campylobacteriosis notifications in Iceland).

Campylobacter spp. are readily destroyed by cooking, and while it is plausible that undercooked poultry should be a risk factor in the two case-control studies, there is a question as to the subjective judgement of undercooking by a high proportion of cases. It is more likely that cross contamination from poultry to hands and kitchen surfaces provides an opportunity for other foods to be contaminated, or else direct ingestion to occur.

Complicating this picture is that consumption of poultry at home was a protective factor in both New Zealand case-control studies, whereas poultry eaten outside the home (at friend's houses or restaurants) was associated with higher risk. Such results have been replicated in other case-control studies in the US and Europe It may be that respondents in case-control studies have a bias to more easily recall poultry meals eaten away from home.

Apart from undercooking, transmission between poultry and humans is likely to be complex, and involve cross contamination. The introduction of a foodstuff with a high likelihood of contamination by *Campylobacter* spp. into the domestic or retail kitchen provides a starting point for such transmission.

Red meat is consumed by the majority (78%) of the New Zealand population on a daily basis. However, meats other than poultry were not found to be risk factors in either of the two New Zealand case-control studies.

The contamination prevalence of beef and sheep meat in the ESR surveys is low in comparison with poultry. Slightly higher contamination has been found for bobby veal and pork. A survey of sausages and hamburgers found no *Campylobacter*.

These data suggest that red meats play a minor role in the transmission of campylobacteriosis in New Zealand.

Offals are consumed by a small (4%) proportion of the population on a daily basis. Although all types of offals tested have shown some contamination, it appears that sheep and chicken livers are contaminated with *Campylobacter* at the highest rate (40% or more). An outbreak investigation in Christchurch in 2000 found that chicken liver pate was the source.

These data suggest that offals are a food vehicle in a small proportion of cases.

Raw milk has been identified as a risk factor in both outbreaks and sporadic cases, either in New Zealand or internationally. Raw milk consumption was a significant risk factor in the large 1992-1993 case control study, but only reported by a small proportion of respondents. The limited analytical data on raw milk does not indicate a high prevalence of *Campylobacter* contamination.

It seems likely that raw milk consumption represents a moderate to high risk for exposure to *Campylobacter* on a per serving basis, but only a small proportion of the population are likely to be exposed. In national terms therefore, this transmission vehicle will be a very minor part of the overall picture.

There are limited data on other foods. The small amount of information concerning the prevalence of contamination of shellfish, and vegetables suggests they will not be important vehicles for transmission of campylobacteriosis.

Data from New Zealand drinking water indicates that *Campylobacter* is not present in the treated water supplies serving the vast majority (89%) of the population. There may be a problem with water supplies serving the remainder; some of these supplies will be rainwater derived and potentially contaminated by birds, but this must represent a minor transmission route. Limited data from *ad hoc* testing by ESR suggest that a small proportion of such supplies are contaminated by *Campylobacter*.

Potable water is consumed in large quantities by the entire population, and it is possible that a very low level of contamination could result in large numbers of cases. However, current information does not indicate even a low level of contamination in treated supplies, and so this transmission route can be considered as a very minor component of the overall incidence.

Data from monitoring of ground and surface waters in New Zealand indicates that *Campylobacter* contamination is widespread, although generally the numbers of bacteria are very low. In terms of a transmission route for human exposure, it is most likely that this would occur during recreational activity, or else during occupational exposure. The analysis by NIWA suggested that recreational freshwater exposure could be responsible for around 4% of campylobacteriosis infections. While this estimate has considerable uncertainty, and does not include recreational exposure to marine waters, it seems reasonable to assign recreational water exposure as a minor transmission route within New Zealand.

The presence of *Campylobacter* in environmental waters (and the environment generally) is likely to play an important role in cycling of the bacterium in animals, causing infection in cows, pigs and sheep, and possibly poultry.

It seems reasonable to expect some transmission from domestic pets to humans. Overseas studies have found that pet ownership, particularly puppies, was a risk factor. Pet ownership in New Zealand is widespread.

From studies of domestic pets overseas it seems that infection with *C. upsaliensis* is significant, along with *C. coli* and *C. jejuni*. *C. upsaliensis* does not figure prominently amongst human isolates in New Zealand, although this species may not be routinely tested for by clinical laboratories. No New Zealand data on carriage of *Campylobacter* by domestic pets has been located, and overseas studies show considerable variability. Dogs/puppies appear to be more frequent carriers than cats, and this is consistent with risk factors identified in several case-control studies.

This limited information suggests that transmission from pets is a minor component of the overall picture, but further investigation is warranted.

Data on the prevalence of *Campylobacter* in the faeces of New Zealand farm animals is sparse, but the available results suggest that contamination may be high (up to 50%), with dairy cows apparently the most commonly contaminated. Contact with animals (particularly bovine) was identified as an important risk factor in the Ashburton study. There have been anecdotal reports of spikes in campylobacteriosis cases occurring in rural areas during calving season. The size of the rural population (14%) will be bolstered by farm visitors, whose exposure will be intermittent.

Animal contact seems likely to be an important part of the exposure of a sector of the New Zealand population. In the national picture however, rural cases only represent 8-12% of the total, and even if the majority of these are caused by animal exposure, from a risk management point of view the importance of animal contact would be less than for poultry.

Based on results from case control studies, surveys of the prevalence and level of contamination, and consumption by a high proportion of the population, poultry products are the most common risk factor and the product most likely to be contaminated. The prevalence of contamination in other foods is at least ten-fold lower, and although consumption of red meats is approximately 4 times greater than for poultry, it seems unlikely that this would result in greater exposure.

The transmission of *Campylobacter* in New Zealand is likely to be complex, with a number of risk factors operating at once. It is possible that no single factor is sufficiently important to provide an opportunity to significantly affect the rate of illness.

However, it is the author's belief that effective management of the risk from *Campylobacter* in poultry will cause an observable reduction in the incidence of campylobacteriosis in New Zealand, for the following reasons:

- The proportion of campylobacteriosis cases in rural areas (8-12%) is similar to the rural population (approximately 14%), and notification rates in rural and urban total populations are similar;

- The majority of cases occur in urban regions, and case-control studies of predominantly urban populations have identified poultry associated risk factors as important (representing over 50% of the population attributable risk in one study);
- Even if there are differing transmission route patterns for urban and rural populations, the majority of the risk management activity should focus on the urban pattern;
- A temporary removal of poultry from the market in Belgium was followed by a 40% drop in campylobacteriosis notifications, and a decline in poultry consumption in 2003 in the Netherlands was associated with a reduction in incidence;
- Successful risk management of the incidence of campylobacteriosis in Iceland by focusing on poultry, alongside consumer education measures.

Of the remaining risk factors for campylobacteriosis, overseas travel and animal contact (for the rural population), appear to be the most important.

Potable water, pets, and environmental water, are likely to be more minor parts of the overall transmission route picture.

This report does not provide an answer for the key question of why the rate of reported campylobacteriosis in New Zealand is high compared to overseas countries. However, the available data from a variety of studies does indicate that poultry, as a source of *Campylobacter* and leading directly or indirectly to infection, is the risk factor whose management is the most likely to lead to a significant drop in illness.

It is acknowledged that reducing the prevalence of contamination of the poultry supply by *Campylobacter* will be difficult. Despite considerable research by the industry and scientific community into on-farm and processing options, a "magic bullet" has yet to be found (some effective options such as irradiation are unacceptable to consumers). In recent years the New Zealand poultry industry has successfully reduced the prevalence of *Salmonella* in retail product to amongst the lowest in the world. However, the measures implemented have not reduced *Campylobacter* contamination. It is the intention of this report to indicate that efforts should continue to manage this risk.

1 INTRODUCTION

A World Health Organisation Consultation in 2000 identified infection with *Campylobacter* as the leading cause of zoonotic enteric infections in developed and developing countries (WHO, 2000). Many risk factors for *Campylobacter* infection have been identified. In developing countries, inadequately treated water and contact with farm animals are assumed to be the most important risk factors, at least in outbreaks (WHO, 2000). In developed countries the pattern of transmission pathways appears to be more complex.

Campylobacteriosis is a major public health problem in New Zealand. Based on notification rates, the reported incidence of the illness is high compared to other developed countries, and has generally increased over the last 10-15 years. Cases of campylobacteriosis have been estimated to cost $62m annually, which comprises the majority (70%) of the total economic cost of infectious intestinal disease in New Zealand (Lake *et al*., 2000; Scott *et al*., 2000).

The objective of this project is to prepare a review document identifying, to the greatest extent possible, the identity and relative importance of different transmission routes for *Campylobacter* infection in New Zealand.

As an important public health problem in New Zealand, campylobacteriosis has been the subject of numerous research projects over the last ten years. This project brings together the results of that research, in order to synthesise a picture of the potential transmission routes, the evidence to support their existence, and their relative importance. This report is unlikely to be the final overview, but is supported by the best evidence currently available.

Information on sources and potential transmission routes of *Campylobacter* is supplemented with data on relevant exposures, such as food consumption data.

This report also presents an overview of the international situation regarding campylobacteriosis and transmission routes. This has been derived from the scientific literature and government reports. It is not intended to be an exhaustive review of the extensive literature on *Campylobacter*; instead the focus has been on surveillance and epidemiological studies, as well as case-control investigations, which generally provide the most information on sources of human infection.

In this document, the term *Campylobacter* will refer specifically to the species *C. jejuni* and *C. coli*.

1.1 Characteristics of *Campylobacter* spp.

Campylobacter jejuni is carried in the intestinal tract of a wide variety of wild and domestic animals, and as a result of faecal contamination during processing, may contaminate foods derived from those animals (Doyle and Jones, 1992). The optimum growth temperature of thermotolerant *Campylobacter* lies between 37 and 42°C. Growth does not occur below 30°C and so actual multiplication during handling or storage at room temperature will not occur in moderate climates (Jacobs-Reitsma, 2000). Refrigeration may promote survival, while freezing, although causing a reduction in numbers, does not eliminate the bacterium. Cooking readily destroys the organism, and it is particularly susceptible to drying (Wallace, 2003).

1.2 Symptoms and Illness

The incubation period for campylobacteriosis ranges from 1 to 10 days (usually between 2 and 5 days). Symptoms typically include muscle pain, headache and fever (known as the "febrile prodrome") followed by watery or bloody diarrhoea, abdominal pain and nausea. Symptoms may last 1 day to 1 week or longer (usually 5 days). Excretion of the organism in stools occurs on average for 2 to 3 weeks, and the illness is mostly self-limiting (Wallace, 2003).

Hospitalisation due to *Campylobacter* infection has been estimated as approximately 8-10% of notified cases, or 0.5 – 0.6% of all community cases. Mortality from campylobacteriosis has been estimated as case-fatality ratio of 1 per 10,000 with a range of 1 per 100,000 to 2 per 10,000 (data reviewed in a study estimating the burden of campylobacteriosis in the Netherlands: Havelaar *et al.*, 2000).

Campylobacteriosis may have chronic sequelae in the form of Guillain-Barré syndrome (GBS) and reactive arthritis. The percentage of cases of campylobacteriosis which result in GBS has been estimated as 0.1% (Altekruse *et al*., 1999). Another lower estimate, from Sweden (McCarthy and Giesecke, 2001), suggests an attributable risk of GBS for laboratory confirmed cases of *Campylobacter jejuni* infection of 30.1 per 100,000 cases (95% confidence interval (CI): 13.9, 57.8). Approximately 20% of patients with GBS are left with some form of disability and approximately 5% die. Penner serotype HS:19 has been associated with GBS in Japanese studies but this was not confirmed in a USA case control study, in which no specific serotypes were associated with GBS (Rees *et al*., 1995).

Campylobacteriosis is also associated with Reiter's syndrome, a reactive arthropathy. The percentage of cases of campylobacteriosis which result in reactive arthritis has been estimated as 1% (Altekruse *et al*., 1999).

Campylobacteriosis affects all ages, but particularly affects young children under 4 years, and adults between 15 and 44 years (Tauxe, 2000). There is some overseas evidence that the higher rates observed in young children are partly due to parents being more likely to seek medical care for these types of illness in their children (Stafford *et al*., 1996). It is also possible that subsidised health care for young children may promote doctor visits. The higher number of cases in the young adult category (20-29 years) may be due to greater travel activity by this age group. An alternative explanation is that people of that age (especially males) have less experience of food preparation, and therefore are less likely to handle food safely.

The seasonal pattern of *Campylobacter* infections in developed countries involves a well-defined summer peak, that is more pronounced with increasing latitude (Tauxe, 2000);

The incidence in males is 1.2 to 1.5 times higher than in females, and the higher rate amongst males is particularly pronounced in the young adult peak (Tauxe, 2000).

Secondary transmission (person to person) is rare in comparison with other enteric infections (Tauxe, 1992).

Infection with *Campylobacter* amongst workers has been most frequently reported in poultry dressers, according to data from the UK between 1996 and 2003 (Wilson, 2004). This report suggested that transmission for the particular case being examined had occurred by aerosolised droplet transmission via the mouth, although hand to mouth transfer is difficult to rule out. Epidemiological evidence from this poultry processing plant indicated that workers were three times more likely to suffer campylobacteriosis than the general population. Occupational campylobacteriosis has also been recorded in others working with animals, meat and food.

There is some evidence for the development of immunity in the general population. Of the three types of antibodies produced during infection, two decline rapidly, whereas IgG antibodies can remain elevated for months or years afterwards. In Denmark the prevalence of *C. jejuni* specific IgG antibodies increased with age, from 20.6% in the 15-34 years age group, to 32.4% in the 50-59 years age group (Linneberg *et al*., 2003). A UK study examined a large dataset of Penner serotypes of *C. jejuni* from cases of human campylobacteriosis (Miller *et al.*, 2005). The most prevalent serotypes were HS:4 complex, HS:2, and HS:1,44 (53.8% of all cases). The ratios of uncommon to common serotypes were higher in age groups of 40 years and older, which along with gradually decreasing incidence in higher age groups, suggests that people develop immunity to the most common types of *C. jejuni* during their lifetimes.

1.3 Dose Response

The conventional view that there is a minimum infectious dose below which infection cannot occur is rapidly declining, and there is increasing acceptance of the notion that ingestion of even a single cell has an associated probability of causing infection even though this probability may be very low (Teunis and Havelaar, 2000). In quantitative risk assessment even low probabilities of infection may result in significant disease predictions if the number of exposure events is high.

Data from experimental studies where volunteers ingested known numbers of *Campylobacter* cells have been investigated for the purpose of modelling the dose-response relationship (Medema *et al.*, 1996; Teunis *et al.*, 1999). Infection, where the micro-organism 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 close to 100% at 1 x 10^8 cells. In contrast, the likelihood of illness was approximately 20% at 800 cells, rising to 46% at 9 x 10^4 cells, and declining to close to 0% at 1 x 10^8 cells.

The FAO/WHO hazard characterization (FAO/WHO, 2002) has explored the idea that there is a conditional probability of disease resulting from infection, as opposed to the model for disease following infection described above (Teunis *et al.*, 1999). This model predicts that in the vast majority of cases where people have become infected, there is >20% and <50% chance of illness.

1.4 Methodology for Isolation and Typing of *Campylobacter*

An extensive review of methodology for the isolation of *Campylobacter* was prepared under the auspices of the Enteric Zoonotic Disease Research in New Zealand Steering Committee (Donnison, 2002) and a survey of phenotypic and genotypic methods used to identify

Campylobacter isolates was completed in 2001 (Klena, 2001). Twelve typing methods were reviewed. Choice of method should be based on the information required.

There is a reasonable amount of information on the serotypes of *Campylobacter* isolates in New Zealand. The majority of this is derived from the "gold standard" reference method heat stable (HS) serotyping of antigens developed by Penner and Hennessy (1980). The antigens are probably capsular i.e. part of the envelope surrounding the cell wall (Chart *et al*., 1996). As the procedures for serotyping are well established information from different studies can be compared. Other methods of typing (e.g. biotyping) have also been employed, but have not been extensively applied to *Campylobacter* spp. in New Zealand (Nicol and Wright, 1998).

More recently information derived from restriction enzyme digestion and pulsed field gel electrophoresis (PFGE) analysis of bacterial DNA has become available (Gibson *et al*., 1994). The enzymes employed, and conditions used for the gel electrophoresis, have a marked influence on the results. Thus it may be difficult to compare PFGE results between studies.

A New Zealand Microbial Typing Database has been established and seeks to harmonise PFGE methodology for *Campylobacter* within New Zealand, and also ensure that it is internationally comparable. The database, which is compatible with the PulseNet USA system (www.cdc.gov/pulsenet), will allow submission of serotyping, PFGE and epidemiological data on *Campylobacter* isolates to a centralised server.

There are additional DNA based methods of typing available for *Campylobacter* e.g. *fla* typing (polymerase chain reaction (PCR) restriction fragment length polymorphism analysis of the flagellin *flaA* and *flaB* genes), and multi-locus sequence typing (MLST) (based on sequencing a number of defined alleles within the bacterial genome). MLST typing is being increasingly applied to *Campylobacter* isolates from New Zealand to provide additional opportunities for investigation and international comparisons.

2 CAMPYLOBACTERIOSIS IN NEW ZEALAND

In New Zealand, campylobacteriosis is an illness caused by infection with *Campylobacter jejuni* and *Campylobacter coli* primarily. There are currently 16 species of *Campylobacter* recognised, but *C. jejuni* and *C. coli* are the ones of significance to public health (*C. fetus* is important in animal health) (Abbott *et al.*, 2004). Other species, such as *C. upsaliensis* and *C. lari* have been reported as causing human illness but their significance in New Zealand is unknown, and not all laboratories will routinely attempt to isolate species other than *C. jejuni* and *C. coli*. Other *Campylobacter* species have occasionally been reported to cause illness overseas e.g. *C. curvus* (Abbott *et al.*, 2004).

2.1 Incidence

The incidence of campylobacteriosis reported to the national surveillance system in New Zealand has shown a general increase since the disease was first made notifiable in 1980. The numbers of reported cases and rates since 1990 are given in Table 1, and the trend in rates of notified cases is shown in Figure 1. In 2004 campylobacteriosis remained the most commonly notified disease in New Zealand, comprising 53.2% of all communicable disease notifications (ESR, 2005).

New Zealand's notified rate of campylobacteriosis declined from 2003 to 2004, and the rate has remained low during the first half of 2005 but does not seem to have declined further (rate for the 12 months to July 2005 was 319.9 per 100,000).

Table 1: Number of reported cases and rates of campylobacteriosis from 1990 to 2004 (Sneyd and Baker, 2003; ESR, 2004, ESR, 2005)

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

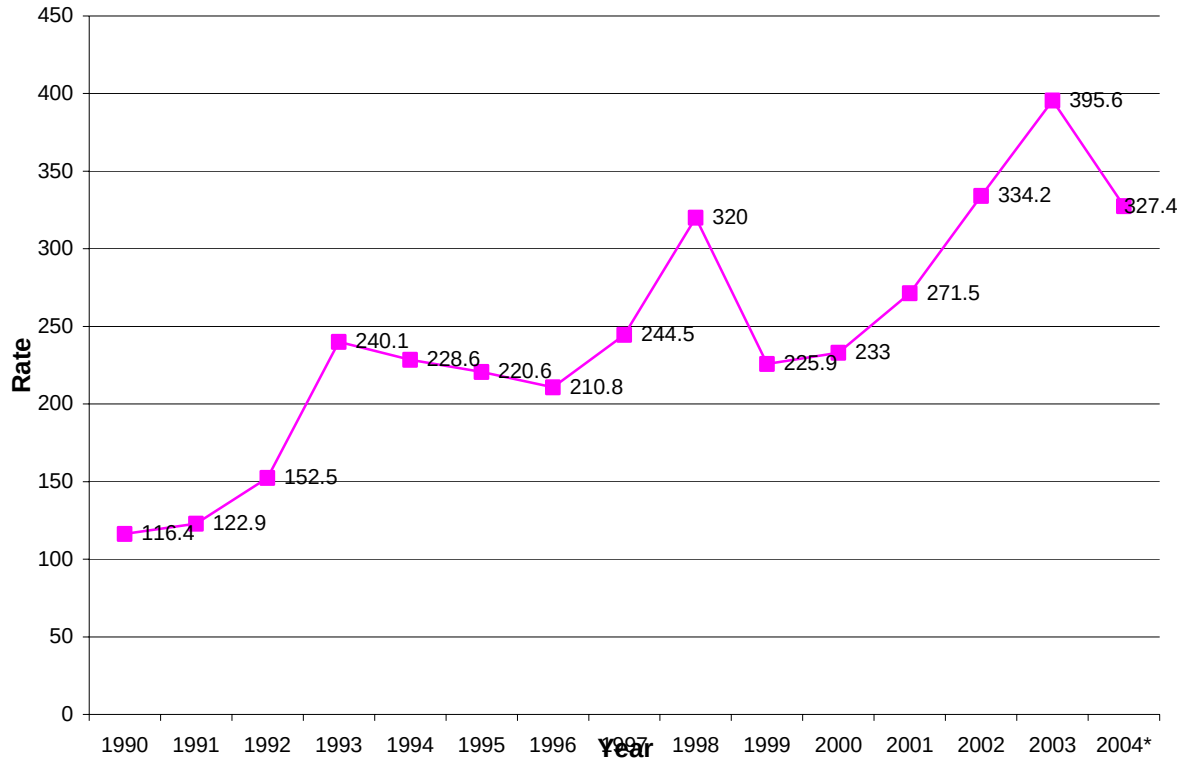

Figure 1: Rates of reported campylobacteriosis 1990 – 2004

New Zealand's rate of reported campylobacteriosis is considerably higher than reported rates from other developed countries, as shown by the data in Table 2. However, such comparisons may be subject to differences in reporting systems; for example, Australia's most populous state New South Wales, does not report campylobacteriosis as a specific illness. Variation within a country can also be significant: of the US FoodNet states, in 2004 California had the highest campylobacteriosis rate (28.6 per 100,000) and Maryland the lowest (5.3 per 100,000).

Reported rates of campylobacteriosis appear to be in decline in the US (CDC, 2005), UK (ACMSF, 2005) but not Australia (OzFoodNet Working Group, 2005). A 31% fall in Campylobacter infections in Scotland between 2000 and 2003 has been reported (Strachan *et al.*, 2005).

A review in 1993 (Lane and Baker, 1993) concluded that the increase in notified cases between 1980 and 1992 represented a real increase in the incidence of this disease, rather than being due to changes in reporting procedures. The reasons for this conclusion were that the increase occurred across nearly all regions, across all age and gender groups, and laboratory isolations increased in one area health board for which data were examined. For the period 1995 to 2002, hospitalisations for campylobacteriosis showed a parallel increase to the notification rate, further supporting the conclusion of increased incidence (Sneyd and Baker, 2003).

Table 2: Comparison of reported campylobacteriosis rate between countries

Country	Period	Rate /100,000	Reference
Australia*	2002	112	Yohannes *et al.*, 2004
Australia*	2003	116.5	Miller *et al.*, 2005
Australia*	2004	117	OzFoodNet Working Group, 2005
Canada	2000	40.1	Health Canada, 2003
Denmark	2002	82	Anonymous, 2003a
Iceland	1999 2000	116 33	ACMSF, 2004
Ireland	2001	35.5	NDSC, 2002
UK; *England and Wales*	2001	107.6	NDSC, 2002
Northern Ireland	2001	52.4	NDSC, 2002
Scotland	2003	86.6	SCIEH, 2004
USA	2002	12.9#	CDC, 2005

*Excludes New South Wales which does not report campylobacteriosis except when an outbreak occurs.
Data collected from 9 US States (Foodnet) which represents 13% of total USA population.

An analysis conducted in 1994 examined the question whether the increase in reported campylobacteriosis was due to changes in laboratory procedures (McNicholas *et al.*, 1995). A survey of 69 medical laboratories determined that differences in laboratory methods did not explain regional differences, and were insufficient to account for a marked increase in campylobacter isolations. The difference in trends for salmonellosis and campylobacteriosis over the same period (1992-1993) was further evidence that laboratory procedures were not responsible for the increase in the latter.

It was estimated for the year 2000 that 76% of laboratory confirmed cases of campylobacteriosis in Auckland were notified (Simmons *et al*., 2002). The study compared the numbers of hospital and community laboratory confirmed cases of seven potentially foodborne diseases with those notified to the Auckland Regional Public Health Service. This paper recommended that a change to laboratory based notification could improve public health investigation and control of foodborne disease in New Zealand.

Rates of notification vary throughout New Zealand, with the highest rates for 2004 being reported by South Canterbury (547.5 per 100,000), Southland (452.9), and Auckland (392.9) (ESR, 2005). However, the regional rates vary from year to year, and in 2003 the highest rates were reported by Capital and Coast (618.6 per 100,000), Hutt (502.9), and Waikato (478.7) (ESR, 2004). In addition, high rates occur in both predominantly rural and urban regions. Consequently analyses based on regional rates must be treated with caution.

As is common with enteric diseases, campylobacteriosis is usually seasonal with a summer peak and winter trough. An analysis of the seasonal distribution of *Campylobacter* infections in nine European countries and New Zealand (Nylen *et al*., 2002) found that the European pattern was remarkably consistent across the years 1989-1997. The summer peak occurred at the end of May in Wales, followed about 3 weeks later in Scotland, with Scandinavian

countries peaking about 5-7 weeks after Scotland. Although still exhibiting a summer peak, the pattern was different in New Zealand, where the peak week was much more variable from year to year and the summer increase was much more prolonged.

There does not appear to be a major difference between rates of infection in rural and urban populations in New Zealand, although the differentiation of such populations is not clear-cut (as discussed in Section 3). A preliminary analysis of the rural/urban distribution of enteric disease was published in the Annual Surveillance Summary for 2002 (Sneyd and Baker, 2003). Based on 2001 Census data the proportion of New Zealanders residing in areas classified as "rural" was approximately 12.6%[1]. The proportion of cases residing in rural areas was calculated, along with incidence for both rural and urban populations. The proportion of campylobacteriosis cases residing in rural areas was 12.4% (95% CI 11.9-13.0), while the campylobacteriosis notification rate for rural and urban populations were 339.5 and 330.8 respectively. This similarity in rural and urban rates contrasted with rates for *Salmonella* Brandenburg infection and cryptosporidiosis, where rural rates were markedly higher than urban rates.

An analysis of campylobacteriosis surveillance data from 1993-1997 showed that there was a significant proportion of cases for which there was incomplete address information, and this created uncertainty, particularly in the calculation of rural campylobacteriosis rates (Skelly *et al.*, 2002). Since then geocoding has improved considerably, and analysis of notification data for 2001 and 2002 show that ungeocoded cases represent an insignificant proportion of the total (<0.1%) (C. Skelly, ESR, pers. comm. February 2004, paper in preparation). This analysis shows that in 2001 rural and urban campylobacteriosis notification rates were similar (272 and 282 per 100,000 respectively) while in 2002 rural rates were lower than for urban (237 and 361 per 100,000 respectively). Rural cases represented 12.1% and 8.6% of the total notified cases in 2001 and 2002 respectively.

Campylobacteriosis is more commonly reported for New Zealand males than females; of the cases for which sex was reported in 2004 the male rate was 354.8 per 100,000 and the female rate was 287.0 per 100,000 (ESR, 2005). In 2002 rates of campylobacteriosis for males were higher than for females in each age group (Sneyd and Baker, 2003).

Notification rates and hospitalisation rates are highest amongst New Zealanders of European background: see Table 3 (Sneyd and Baker, 2003; ESR, 2004, ESR, 2005).

Table 3: Ethnicity details for notified cases of campylobacteriosis

Ethnicity	Notifications (per 100,000) 2002	Notifications (per 100,000) 2003	Notifications (per 100,000) 2004
European	307.7	387.3	326.0
Maori	76.9	134.0	110.4
Pacific	53.9	87.9	62.9

[1] Note that the "2001 Census Snapshot 10 (Rural New Zealand)" at the Statistics New Zealand website indicates that 532,740 people were classed as living in rural areas. This represents 14.3% of the total population in 2001 of 3,737,490.

The highest rates of reported campylobacteriosis in New Zealand are seen in the 1-4 and 20-29 age ranges, as shown for 2004 in Table 4 (ESR, 2005). This pattern has been consistent; the same age ranges had the highest rates in 2002 and 2003.

As mentioned in Section 1.2 the cause of a higher rate in very young children may be the greater likelihood of children being taken to the doctor or clinical samples being taken when at the doctor, or else subsidised health care for this age group. Other possibilities include greater exposure to *Campylobacter* through close contact with floors, dirt, pets etc.

Table 4: Age specific rates (per 100,000) for notified cases of campylobacteriosis in 2004 (ESR, 2005)

Age (years)	**<1**	**1-4**	**5-9**	**10-14**	**15-19**	**20-29**	**30-39**	**40-49**	**50-59**	**60-69**	**70+**
Rate (per 100,000) 2004	448.3	515.4	196.4	180.2	314.0	451.8	311.4	292.3	339.4	334.2	279.4

Hospitalisation rates for notified cases of campylobacteriosis in New Zealand from 1997 – 2004 have been between 4 and 8% (ESR, 2005). This is well within the range of up to 10% suggested by a Dutch study (Havelaar *et al.*, 2000). There have been between 0 and 3 deaths due to campylobacteriosis recorded each year (1997 – 2004; average 1.25 per year) amongst the approximately 9,000 – 15,000 notified cases notified (ESR, 2005). Although mortality due to campylobacteriosis may be under-recognised generally (Helms *et al*., 2003), these data suggest that the rate in New Zealand is not higher than estimated overseas (Havelaar *et al.*, 2000).

Internationally, rates of GBS are 1-2 per 100,000 population (Pritchard and Hughes, 2004). In the Netherlands a retrospective study reported a rate of GBS of 1.18 per 100,000 person years (Van Koningsfeld et al., 2000). New Zealand's rates are similar, or perhaps slightly higher than this; recent numbers of cases have been: 2000 71 hospitalised cases (1.9 per 100,000), 2001 103 cases (2.8 per 100,000) and 2002 106 cases (2.8 per 100,000). However, there are a number of other potential antecedent infections associated with the disease; two thirds of cases report symptoms of an infection in the weeks preceding GBS (Pritchard and Hughes, 2004).

An analysis of the incidence of chronic sequelae of campylobacteriosis (GBS and reactive arthritis) has been undertaken for the period 1988 to 2002 (Lake *et al.*, 2004). There was no apparent increase in the number of hospitalised cases of GBS over that period, even though an increase could have been expected, based on the Swedish study (McCarthy and Giesecke, 2001), the estimated number of total campylobacteriosis cases in New Zealand, and the rise in notifications.

No conclusions could be drawn about the incidence of reactive arthritis, due to the lack of specificity in the hospitalisation coding system, and the multiplicity of potential triggering organisms. If the rate of reactive arthritis is indeed 1% of campylobacteriosis cases (Altekruse *et al*., 1999) then we might expect over 1000 cases per year; however the hospitalisation coding system indicates that there are approximately 400 cases per year in New Zealand, with the vast majority being coded as "Unspecified infective arthritis (711.9)" (Lake *et al.*, 2004).

Overseas, campylobacteriosis accounts for only a small proportion of total reported outbreaks (1% of reported foodborne and waterborne outbreaks in the US from 1980 to 1996 (Friedman *et al*., 2000a)) and the disease is regarded as principally sporadic in nature. This has been attributed to the fact that *Campylobacter* do not multiply under aerobic conditions or at room temperature so poor food handling is less likely to result in general spread of the organism. In addition, the relatively long incubation period and low attack rate means that outbreaks are less likely to be recognised and reported (Frost, 2001).

In contrast, the New Zealand data show that *Campylobacter* are identified as the causative agent in approximately 12 - 16% of reported outbreaks, and up to 13% of total outbreak cases. This difference may be due to reporting differences compared to overseas (e.g. differing outbreak definitions), or may be genuine. Nevertheless, the number of reported outbreak cases is still below 3% of the total campylobacteriosis notifications and so the illness in New Zealand is predominantly sporadic.

Penner serotyping based on the heat stable (HS) antigen(s) has been conducted for 1130 *Campylobacter* isolates obtained from human cases in New Zealand between 1996 and 2001. The serotypes identified include: HS:1,44 (16% of serotyped isolates); HS:2 (23%); HS:4 complex (15%); HS:5 (0.6%); HS:10 (0.6%); HS:19 (0.8%); HS:23 (8%); HS:35 (1.3%); HS:37 (4%); HS:41 (0.5%) (Lake *et al*., 2004). While serotype is currently the most common information on strains of *Campylobacter* in New Zealand (see further discussion in Sections 5 and 6), in the future, accumulating PFGE and MLST typing data will offer greater analytical value.

2.2 Comments

The pattern of campylobacteriosis notifications in New Zealand is not markedly different to overseas, apart from the reported rate.

Age and sex distribution, the summer peak, and sporadic nature of the illness are similar to overseas, and this suggests that the overall epidemiology is likely to be similar to that in other developed countries. Hospitalisation and mortality data do not suggest higher pathogenicity of *Campylobacter* strains infecting people in New Zealand. The predominant serotypes amongst isolates from human cases are the same as in the UK.

There has been much discussion about the rural characteristics and livestock density of the New Zealand environment playing a role in transmission; and while this may be important for exposures in the rural population (discussed further below), the available data (above average regional rates in both rural and urban areas, rural/urban similarity in rates nationally) do not suggest that rural exposures play a dominant role in the epidemiology of the illness in this country.

3 NEW ZEALAND EXPOSURE DATA RELEVANT TO POTENTIAL TRANSMISSION ROUTES

The WHO has identified risk factors for *Campylobacter* infection in developed countries as:

- handling and consumption of poultry meat;
- foods of animal origin, including raw milk;
- inadequately treated water;
- contact with farm animals and pets; and,
- foreign travel.

This section provides available information about the size of population groups in New Zealand which are likely to be exposed to *Campylobacter* through these potential risk factors, as well as some trend information. This will assist in assessing the relative importance of these exposure routes, when combined with information on the prevalence and numbers of *Campylobacter* provided in Sections 5 and 6.

3.1 Food

Campylobacter jejuni is found principally in foods of animal origin. Poultry meat has been shown to be frequently contaminated in a number of surveys. At lower frequencies, *Campylobacter* has also been found in beef, pork, other meat products, raw milk and milk products, and in fish and fish products. Shellfish (oysters and mussels) may also contain the organism. Other food items in which *C. jejuni* has been detected are mushrooms, and fresh vegetables (Christensen *et al*., 2001).

Consumption of poultry meat in New Zealand has increased steadily over the last 19 years, from an apparent consumption (poultry available for consumption per capita) of 15 kg/person/year in 1985 to 35.9 kg/person/year for the year ending September 2003 (MAF, 2003)

Over this period total meat consumption has been relatively static, so the proportion of poultry meat consumed has increased from 15% to 25%, largely at the expense of sheep meat. The increase in chicken consumption has been mainly in the fresh and further processed areas, with approximately 74% of chicken now sold fresh (MAF, 2001), and under 40% of chickens sold as whole birds (PIANZ, 1999).

From 1986 to 1999 sales of frozen poultry declined from 54% to 34% of the total market with a commensurate increase in fresh products. Whole bird sales decreased from 59% to 40% over the same period (Cooper-Blanks, 1999).

Based on data from the 1997 National Nutrition Survey (NNS97), the percentage of respondents consuming poultry during the previous 24 hour period is 20.8%.

A comparison with Australian data (Lake *et al.*, 2003) found that consumption of poultry there was similar, both in the amounts and proportion of the population.

Red meat consumption has declined over the last 20 years (MAF, 2003). Most of the decline has been in mutton and lamb consumption, while pork consumption has increased slightly. An analysis of the 1997 NNS data concluded that 77.7% of the New Zealand population consumed red meat (cattle, sheep or pig meat) during any 24 hour period.

According to analysis of the New Zealand 1997 NNS 4.2% of respondents reported consuming mammalian offals, while 0.5% reported consumption of poultry offals. These figures will include pâté.

Food consumption exposures will be augmented by exposure of food preparers handling potentially contaminated materials, where cross contamination to hands and surfaces provide additional routes for ingestion.

3.2 Potable Water

Water supplies that serve more than 25 people for more than 60 days per year are required to be registered in New Zealand. In 2004 registered water supplies in New Zealand covered a population of approximately 3.62 million people (Ministry of Health, 2004). Registered water supplies serving the great majority of New Zealand's population will be monitored for bacteriological quality and risk management measures put in place if contamination (using *E. coli* as an indicator) is detected.

Absent from this total will be single supplies covering small numbers of people, some of which will use rainwater as a source. This suggests that for a total estimated New Zealand population of 4.06 million, approximately 446,000 people (11%) use unregistered water supplies, some of which will be from roofwater (Ministry of Health, 2004). These supplies are the most likely drinking water sources to represent a potential exposure to *Campylobacter*.

Water consumption is generally considered to be in the region of two litres per day, although for most adults some of this will be in the form of hot drinks, which are very unlikely to contain *Campylobacter*.

3.3 Animal Exposure

The "2001 Census Snapshot 10 (Rural New Zealand)" at the Statistics New Zealand website indicates that 532,740 people were classed as living in rural areas. This represents 14.3% of the total population in 2001 of 3,737,490. New Zealand is described as being one of the most highly urbanised countries in the world, along with Australia, the US, UK and Europe.

However, the distinction between rural and urban populations is not rigid. Statistics New Zealand report 71.1% of New Zealanders living in a "main urban area" (population >30,000), 3.0% living in a "satellite urban community", 11.7 living in an "independent urban community", and the remaining 14.2% living in "rural" areas of various levels of urban influence. People in satellite and independent urban communities may have frequent contact with livestock and rural environments that contain *Campylobacter*.

Urban areas comprise less than 3% of New Zealand's land area, and there is a perception that we frequently experience and participate in activities in rural areas. Walking, tramping,

mountaineering, orienteering, river swimming etc. could all expose urban dwellers to rural environments that contain *Campylobacter*. Unfortunately, data on the frequency of such activities are not readily available. An ameliorating factor may be that many such activities take place in National Parks, where livestock are not present.

Statistics New Zealand report 137,500 agriculture and fishery workers (>15 years) in the categorisation of occupations from the 2001 census.

New Zealand is a country with a high livestock population, relative to its land area (250,000 km^2 (Till and McBride, 2004)). Livestock numbers for New Zealand in 2001 are shown in Table 5, derived from information at the Statistics New Zealand and MAF websites.

Table 5: Livestock numbers for New Zealand

Main Classes of Livestock (millions) in 2004	
Total sheep	39.0 (as at June 2004)
Total beef	4.4 (as at June 2004)
Total dairy	5.2 (as at June 2004)
Total pigs	0.36 (as at June 2003)
Total deer	1.7 (as at June 2003)

There are approximately 17,000 dairy farm owners and sharemilkers belonging to the DairyInsight cooperative dairy farm organisation (Annual Report 2002/2003 at website http://www.dairyinsight.co.nz/annualreport.cfm). Fonterra includes 12,600 dairy farmers (Anonymous, 2003b).

The Agricultural Statistics Survey 2002 by Statistics New Zealand reported the following numbers of farm types:

- sheep: 13,000
- beef: 13,000
- mixtures of sheep, beef, grain: 2,250
- dairy: 14,000
- pig: 360
- poultry (meat or eggs): 390
- deer: 2,200
- mixed livestock and not elsewhere classified: 1,830

If each of these approximately 47,000 farms have perhaps an average of 5 inhabitants (family and workers), then an estimate of the population routinely exposed to livestock could be in the region of 235,000.

There are no data on the prevalence of raw milk consumption in New Zealand. If it is assumed that each dairy farm has the potential to supply raw milk to up to 10 family members and friends, then one estimate for the population potentially practising raw milk consumption might be up to 170,000.

An alternative estimate can be derived from the case-control and cohort studies discussed in Section 4. Consumption of unpasteurised milk was reported by 9 of 44 cases (20%) interviewed for the transmission routes study in Ashburton (Baker *et al*., 2002). Consumption of unpasteurised milk was reported by 5.8% of cases and 2.4% of controls in the primarily urban case control study (Eberhart-Philips *et al*., 1997). If 14.3% of New Zealand's population is classed as rural (573430 of estimated 2003 population of 4.01 million), and 20% consume unpasteurised milk then the exposed population could be 115,000. Adding an average of the number of urban cases and controls reporting raw milk consumption (approximately 4%) of the urban population of 3504740 gives an additional 140,000 people, providing an exposed population of perhaps 255,000. The number of people consuming unpasteurised milk may be within the range 170,000-255,000.

There are an estimated 595,000 dogs and 814,000 cats in New Zealand (Forest and Bird fact sheet; http://www.forest-bird.org.nz/dawnchorus/wildlifefriendlyfactsheet.pdf). Obviously contact with cats and dogs is a common exposure, although no information has been located regarding carriage rates of *Campylobacter* by these animals in New Zealand.

3.4 Recreational Water Use

It has been estimated, based on surveys of recreational water use, that approximately 250,000 New Zealanders go for at least one swim at a freshwater site each year (McBride *et al*., 2002). No information has been located for recreational activity in marine waters.

3.5 Comments

Clearly the foods identified as common vehicles for *Campylobacter* transmission, poultry and red meat, are consumed by a high proportion of the population on a daily basis. Risk will therefore depend on the prevalence of contamination and numbers of bacteria present. Offals are infrequently consumed.

On the basis of limited data, other intentionally ingested vehicles, such as unpasteurised milk and untreated water, could potentially expose relatively small proportions of the population. Again risk would depend on prevalence of contamination and numbers of bacteria, and available data are reviewed in Sections 5 and 6.

Livestock numbers are high, but the rural population (and the farming population will be a subset of this) is less than 15% of the total. Urban dwellers may experience rural exposures (i.e. livestock and contaminated environments such as rivers) through recreational activities, although not all these activities will involve farmland. Livestock effluent containing *Campylobacter* may be more important in maintaining environmental contamination and carriage in animals, than direct human exposure.

Ownership by New Zealanders of domestic cats and dogs is common, and these potential exposures will be augmented by other companion animals, petting zoos, and small scale farming operations such as lifestyle blocks. Data associated with these potential exposures have not been located.

4 INFORMATION FROM NEW ZEALAND STUDIES OF HUMAN CASES ON POTENTIAL TRANSMISSION ROUTES

Sections 4-6 inclusive collate information concerning transmission routes that derives from research conducted in New Zealand. Although some studies include information that falls into more than one section, the information has been broadly broken down to:

- Section 4: information derived from human cases;
- Section 5: information on the prevalence of *Campylobacter* in animals, food and water; and,
- Section 6: information on the prevalence of *Campylobacter* in the New Zealand environment.

4.1 Risk Factor Information from Outbreaks and Notifications Reported to the National Surveillance System

A summary of the risk factors reported by cases of campylobacteriosis from 1990-1993 found that 84% of notifications reported the likely source of infection as unknown (Lane and Baker, 1993). The most commonly reported suspected source was chicken but this was reported in only 4.2% of cases. Other suspected sources were animal contact (2.8%), human contact (1.9%) and untreated water (1.5%).

Suspected risk factor information has been reported for a higher proportion of notified campylobacteriosis cases in more recent years (40% in 2001; Andrew Ball, ESR Christchurch Science Centre, pers. comm. May 2004). For example, in 2004, of the campylobacteriosis cases for which information was reported 50% had consumed food from one or more retail premises, 31% had contact with farm animals, 21% had contact with untreated water, 14% had contact with recreational water, 12% had contact with faecal matter, and 8% were food handlers (ESR, 2005). However, these suspected risk factors are unlikely to be reliable, as they will rarely be subject to laboratory or epidemiological investigation, reporting on risk factors may not be comprehensive (for example, the percentages reported above for risk factors in 2004 are based on information supplied for, at most, 30% of all notified cases), and more than one risk factor may be reported for each case.

Despite these problems, the risk factor information is probably more useful in determining the prevalence of overseas travel by cases in the incubation period prior to illness. The number of cases reporting overseas travel, and the percentages of the cases which reported on this risk factor, are given in Table 6. While overseas travel during the incubation period prior to illness is not a guarantee that infection was acquired overseas, these data are a qualitative indicator of the relative importance of this factor.

The 189 outbreaks of campylobacteriosis reported to the national surveillance system between January 2000 and March 2004 were collated and reviewed. Information regarding suspected transmission routes is given in Table 7.

Table 6: *Campylobacter* sporadic cases by year (1997-2004) and overseas travel (Data source: EpiSurv 9 April 2004, and ESR, 2005)

Year	Total cases	Cases with overseas travel reporting (% of total)	Overseas travel (yes) (% of overseas travel total)
1997	8924	4588 (51.4)	463 (10.1)
1998	11573	6907 (59.7)	566 (8.2)
1999	8161	4592 (56.3)	528 (11.5)
2000	8417	4586 (54.5)	386 (8.4)
2001	10146	5049 (49.8)	383 (7.6)
2002	12494	4883 (39.1)	359 (7.4)
2003	14788	5437 (36.8)	352 (6.5)
2004	12213	3871 (31.7)	283 (7.3)

Table 7: Campylobacteriosis Outbreaks, January 2000 – March 2004*

Vehicles/Sources suspected	No. of Outbreaks	Laboratory confirmation of implicated source
Poultry meat	50	1**
Poultry livers	13	-
Drinking water	22	5**
Other meat/foods	19	1
Animal contact (incl.faeces)	14	-
Person to person	16	-
Unpasteurised milk	3	-
TOTAL No. of Outbreaks	189	7

* More than one suspected vehicle was reported in some outbreaks

**One outbreak cited undercooked chicken and contaminated water as the implicated source. The actual source was likely to be the roof-collected rainwater which was highly contaminated (Greg Simmons, Auckland District Health Board, pers. comm. 2004).

Of the five confirmed drinking water related outbreaks, two were associated with contaminated roof water supplies, and two with possible contamination from farm runoff after rainfall. For one of the farm runoff associated outbreaks, confirmation was described as the detection of coliforms in the supply rather than *Campylobacter*.

It is apparent that although obvious potential vehicles are suspected in most of these outbreaks, laboratory confirmation is rare. Most of the evidence for a vehicle/source in reported campylobacteriosis outbreaks derives from "history of exposure to implicated source" (141 of 265 outbreaks from 1999-2004).

Consequently the strength of evidence of information on risk factors from notifications and reported outbreaks for indicating transmission routes is low (apart from some indication of the importance of overseas travel).

4.2 Completed Research into Transmission Routes

4.2.1 Case control studies and risk factors

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

The first case-control study (Ikram *et al*., 1994) was conducted in the summer of 1992-1993 in urban Christchurch (patients who did not live in the greater Christchurch area were excluded). One hundred each of cases and controls were included. The questionnaire asked details of contact with cases, occupation, overseas travel, consumption of non-Christchurch water, swimming (no further details supplied), contact and hygiene related to pets, handling of raw meat and chopping blocks, methods of cooking, consumption of fast foods, poultry in various forms, dairy products, shellfish, and barbecued foods. There was no significant risk in the handling of human waste, raw meat, pet ownership or time spent on a farm. Neither was there any risk associated with handling of raw beef, pork, mutton/lamb, chicken or offal, and no risk associated with using the same chopping board for meat and vegetables. Drinking water from a rural water source had an elevated risk (Odds Ratio (OR) 2.7, 95% Confidence Interval (CI) 0.89, 8.33), but this was not statistically significant.

Consumption of undercooked poultry, or poultry eaten at a friend's house, were significantly associated with risk of campylobacteriosis. Poultry consumed at home or bought frozen were associated with reduced risk. There was significant risk associated with consumption of barbecued chicken, but not with consumption of barbecued beef, mutton/lamb or salads.

Risk factors for which data were given are presented in Table 8.

Table 8: Risk factor data from the 1992-1993 Christchurch case-control study (Ikram *et al.*, 1994)

Risk Factor	Cases No (%)	Controls No (%)	Odds ratio (95% CI)
Consumed poultry	81/100	81/100	
Consumed chicken	76/81 (93.8)	78/81 (96.3)	0.89 (0.44-1.82)
Consumed duck	4/81 (4.9)	1/81 (1.2)	4.13 (0.42-96.89)
Consumed turkey	7/81 (8.6)	4/81 (4.9)	1.18 (0.35-4.14)
Where poultry eaten:			
Home	60/81 (74.1)	72/81 (88.9)	0.36 (0.14-0.9)
Friends	14/81 (17.3)	5/81 (6.2)	3.18 (1.0-10.73)
Bought fresh	32/60 (53.3)	28/72 (38.9)	1.8 (0.85-3.82)
Frozen at home	14/32 (43.8)	16/28 (57.1)	0.58 (0.18-1.83)
Bought frozen	30/60 (50.0)	42/72 (58.3)	0.71 (0.34-1.51)
Precooked	5/60 (8.3)	2/72 (62.8)	
Stuffed	2/60 (3.3)	1/72 (1.4)	
Undercooked	**9/81 (11.1)**	**2/81 (2.5)**	**4.94 (1.03-23.617)**
Consumed barbecued food	45/100 (45)	45/100 (45)	
Sausages	32/45 (71.1)	35/45 (77.8)	0.2 (0.24-2.02)
Kebabs	9/45 (20.0)	6/45 (13.3)	1.63 (0.47-5.8)
Pork	2/45 (4.4)	2/45 (4.4)	
Chicken	16/45 (35.6)	7/45 (15.6)	3 (0.99-9.34)

Risk Factor	Cases No (%)	Controls No (%)	Odds ratio (95% CI)
Beef (steak/.hamburger)	21/45 (46.7)	21/45 (46.7)	
Mutton (chop)	12/45 (26.7)	11/45 (24.4)	
Seafood	2/45 (4.4)	3/45 (6.7)	
Other	1/45 (2.2)	2/45 (4.4)	
Salad	28/45 (62.2)	32/45 (71.1)	
Water consumption:			
Sources other than Christchurch	38/100 (38.0)	31/100 (31.10)	1.36 (0.73-2.56)
Town	25/38 (65.8)	24/31 (77.4)	0.56 (0.12-1.85)
Farm/Country/Reserve/National Park	13/38 (34.2)	5/31 (16.2)	2.7 (0.89-8.33)
Other	1/38 (2.6)	4/31 (12.9)	

CI = confidence interval

The more recent (and larger) case control study (Eberhart-Phillips *et al*., 1997; colloquially known as the MAGIC study (Multi-Centre Analysis of Gastroenteritis Induced by Campylobacter)) interviewed primarily urban cases and controls (approximately 85% of subjects were classed as urban) in four centres with high notification rates of campylobacteriosis (Auckland, Hamilton, Wellington and Christchurch) during 1994 and 1995. The number of cases was 621, matched by the same number of controls. A large range of non-food exposures were investigated, including:

- Contact with a person having a similar illness;
- Travel
- Water and sewerage source and/or problem
- Exposure to animal manure
- Pets
- Other animal exposures, occupational or non-occupational.

N. B. Recreational water exposure was not investigated.

Food exposures examined included:

- Poultry foods
- Beef or veal in various forms (e.g. hamburger)
- Pork in various forms (e.g. ham)
- Mutton or lamb
- Fish seafood of various types
- Game meat
- Other meat (e.g. salami)
- Uncooked egg products
- Unpasteurised dairy products
- Raw vegetables/salads
- Barbecued foods
- Takeaways

Some aspects of these food exposures were investigated in more detail, particularly cooking methods for meat, poultry and fish, and home food handling practices.

Odds ratios, confidence intervals, and numbers of cases reported in the paper are shown in Tables 9 and 10. These tables include only those food risk factors which were found to be

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

Amongst the non-food exposures, overseas travel, rainwater as a home water source, and contact with faeces of puppies (in the home) or cattle were associated with campylobacteriosis. Occupational contact with bovine carcasses was also strongly associated with disease.

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

Table 9: Odds ratios for food exposures associated with campylobacteriosis from the 1994-1995 case control study (Eberhart-Phillips *et al*., 1997)

Food consumed in prior 10 days	Cases No (%)	Controls No (%)	Odds ratio (95% CI)
More than one poultry meal	401 (64.6)	364 (58.6)	1.31 (1.03, 1.67)
More than one chicken meal	398 (64.1)	361 (58.1)	1.31 (1.03, 1.66)
Any chicken raw or uncooked	108 (17.4)	27 (4.3)	4.52 (2.88, 7.10)
Cooking method:			
Any barbecued chicken	68 (11.0)	30 (4.8)	2.60 (1.64, 4.12)
Any fried chicken	192 (30.9)	141 (22.7)	1.55 (1.19, 2.01)
Any baked/roasted chicken	265 (42.7)	308 (49.6)	0.75 (0.60, 0.94)
Type chicken serving:			
Any whole chicken	196 (31.6)	271 (43.6)	0.59 (0.46 ,0.74)
Any chicken pieces	418(67.3)	346 (55.7)	1.67 (1.32, 2.13
Site preparation:			
Any chicken prepared at own home	387 (62.3)	441 (71.0)	0.67 (0.52 ,0.85)
Any chicken prepared at someone else's house	82 (13.2)	49 (7.9)	1.75 (1.21, 2.35)
Any chicken prepared at a sit down restaurant	113 (18.2)	36 (5.8)	3.85 (2.52, 5.88)
Any chicken prepared at a takeaway establishment	135 (21.7)	91 (14.7)	1.70 (1.24, 2.32)
Form of purchase:			
Any chicken purchased fresh and uncooked, then frozen at home	118 (19.0)	120 (19.3)	0.98 (0.74, 1.30)
Any chicken purchased fresh and uncooked, and not frozen at home	120 (19.3)	107 (17.2)	1.15 (0.86, 1.54)
Any chicken purchased frozen	187 (30.1)	260 (41.9)	0.61 (0.48, 077)
Any chicken purchased pre-cooked to take home	24(3.9)	16 (2.6)	1.53 (0.80, 2.94)
Dairy products:			
Any unpasteurised milk	36 (5.8)	15 (2.4)	3.10 (1.52, 6.32)
Any unpasteurised cream	12 (1.9)	1 (0.2)	12.00 (1.56, 92.28)

CI = Confidence Interval

Table 10: Odds ratios for non-food exposures associated with campylobacteriosis from the 1994-1995 case control study (Eberhart-Phillips *et al*., 1997)

Risk factor	Cases no (%)	Controls No (%)	Odds ratio (95% CI)
Contact with a person having a similar illness:			
Ill person at home in prior 10 days	60 (9.7)	40 (6.4)	1.57 (1.03,2.38)
Ill person outside of home in prior 10days	86 (13.8)	60 (9.7)	1.51 (1.06, 2.15)
Travel:			
Overseas travel prior 10 d	31 (5.0)	7 (1.1)	4.13 (1.95, 10.06)
Overseas travel to non-OECD country in prior 10 d	16 (2.6)	0 (0.0)	Undefined
Water and sewerage:			
Non-city water supply at home	65 (10.5)	61 (9.8)	1.15 (0.68, 1.95)
Non-city water outside of home in prior 10 d	108 (17.4)	72 (11.6)	1.63 (1.17, 2.27)
Rainwater source for home water supply	23 (3.7)	11 (1.8)	2.20 (1.04, 4.65)
Sewerage problem at home in prior 10 d	20 (3.2)	7 (1.1)	2.86 (1.21, 6.76)
Faeces:			
Any handling of animal faeces in prior 10 d	139 (22.4)	154 (24.8)	0.86 (0.65, 1.14)
Handling of calf faeces in prior 10 d	25 (4.0)	7 (1.1)	5.50 (1.90, 15.96)
Handling of bovine faeces in prior 10 d	43 (6.9)	20 (3.2)	3.09 (1.57, 6.10)
Handling of puppy faeces in prior 10 d	8 (1.3)	1 (0.2)	8.00 (1.00, 63.97)
Pets:			
Any pets at home	423 (68.1)	437 (70.4)	0.89 (0.69, 1.15)
Ownership of puppy (dog < 6 mth old)	26 (4.2)	11 (1.8)	2.67 (1.24, 5.74)
Ownership of two or more puppies	7 (1.1)	0 (0.0)	Undefined
Ownership of caged bird	65 (10.5)	54 (8.7)	1.22 (0.84, 1.79)
Ownership of three or more caged birds	14 (2.3)	4 (0.6)	3.50 (1.15, 10.63)
Any pets in home with diarrhoea in prior 10 d	25 (4.0)	12 (1.9)	2.08 (1.05, 4.15)
Other animals:			
Any contact with animals in prior 10 d or at work	497 (80.0)	534 (86.0)	0.62 (0.44, 0.85)
Any contact with cattle prior 10 d or at work	46 (7.4)	24 (3.9)	2.29 (1.30, 4.05)
Any contact with new/aborted calves in prior 10 d or at work	32 (5.2)	18 (2.9)	2.27 (1.12, 4.62
Occupational contact with any animal carcasses	29 (6.4)	15 (3.4)	1.92 (0.95, 3.85)
Occupational contact with cattle carcasses	20 (4.4)	7 (1.6)	2.83 (1.12, 7.19)
Occupational contact with calf carcasses	10 (2.2)	1 (0.2)	8.00 (1.00, 63.97)

CI = confidence interval

4.2.1.1 Comments

These studies provide important information on risk factors for campylobacteriosis in New Zealand, particularly the larger, nationwide (but still urban) study. Publication of the studies provides peer-reviewed reassurance about methodology, and the number of cases and controls was high compared to many such studies overseas (see Section 7). The results should be treated with some caution, however.

In the MAGIC study there was no association with poultry or chicken consumption in the 10 days prior to illness; it was only "more than one poultry or chicken meal" that was associated. Consumption of raw or uncooked chicken was a strong risk factor; however, it seems remarkable that this type of poultry was consumed by such high numbers of cases (17% in the MAGIC study). Visual assessment of undercooking will be difficult. However, the associations with barbecued or fried chicken (more likely to be undercooked) rather than

baked/roasted chicken, as well as chicken pieces rather than whole chicken (presumably more likely to be baked/roasted) are consistent.

Poultry meals consumed at home were reported by the large majority of cases and controls, and were protective in both studies. Both studies also reported statistically significant, elevated risk for poultry meals consumed at a friend's house. This raises the strange scenario of guests being at higher risk than the hosts. It may be that cases were predisposed to remember poultry meals away from home as a result of knowledge of this risk factor, and a natural inclination to focus on non-home factors. The MAGIC study authors made an effort to reduce such recall bias by asking controls to recall exposures from a shorter time frame but some bias may have persisted.

Nevertheless, the association of illness with undercooked poultry consumption is biologically plausible, given the frequency of contamination of this food type (See Section 5), as is the protective effect of frozen chicken (freezing is known to reduce numbers of *Campylobacter*).

That contact with an ill person was a risk factor is surprising given that campylobacteriosis appears to be infrequently transmitted person to person. However, such contact was reported by only up to 14% of cases. Other risk factors identified in these studies (unpasteurised dairy products, overseas travel, non-city water outside of home, rainwater source of home water, handling calf or puppy faeces, ownership of puppies or caged birds, occupational contact with cattle or cattle/calf carcasses) also appear biologically plausible, but were reported by relatively small numbers of cases and controls.

4.2.2 Outbreaks and other investigations

The earliest report of campylobacteriosis in New Zealand found in the literature was an investigation of a single case in Palmerston North (Brougham and Meech, 1979). The source of infection was suggested to be a chicken dish, and the authors advised: "bear this new pathogen in mind when managing diarrhoeal disease".

The material in this section has been organised according to region. Many of the investigations do not conclusively identify a source, and are not useful in establishing transmission routes. They have been included to provide a complete collation of information located, and to illustrate the complexity of the issue.

4.2.2.1 Northland

In November 1992 an outbreak of *Campylobacter* enteritis occurred amongst a school group staying at a Northland camp (Jarman and Henneveld, 1993). A case-control study was conducted, involving 14 cases (6 culture confirmed). Tap water at the camp site from a reticulated source, as well as a roof water source, had significant faecal coliform contamination (900 cfu per 100 ml and 225 cfu per 100 ml respectively). However, *Campylobacter* was not detected in the water samples. Raw milk from a local farm was also consumed, and people who had eaten cereal with milk had a higher chance of developing campylobacteriosis. However, this increased risk did not apply to people who drank the raw milk by itself. Testing of the raw milk showed a bacterial count within the limits required by the Food Regulations. In the end, a source of infection was not able to be conclusively identified.

4.2.2.2 Auckland

A small case control study in South Auckland was conducted in October-November 1992 (the seasonal peak of notifications) and involved 26 cases and 26 controls (McMahon and Mahmood, 1993). Face-to-face interviews were used to provide information on risk factors. The only risk factor which was statistically significant was contact with a sick person (OR 10.29, CI 1.80-103.0). The actual numbers of cases and controls reporting this risk factor were not given, but it was stated that most contacts occurred in the home, but none of the contacts with cramps and diarrhoea had sought medical attention or had a positive test for *Campylobacter*. Despite these weaknesses, the result was taken to suggest that person-to-person transmission should be considered more important; this transmission route is usually considered minor for campylobacteriosis (Tauxe, 2000) but was identified as a risk factor in the MAGIC study.

An unusually large number of reported cases in late 1996 in Auckland prompted a case-control investigation into risk factors for endemic campylobacteriosis during that period (Bloomfield and Neal, 1997). A total of 55 cases and 55 controls were interviewed. Of the non-food risk factors, only travel outside New Zealand was associated with an increased risk of illness (OR=6.3, 95% CI 1.0-41.4), although this involved very few people (8 cases, one control). The risk from having a rainwater-derived water supply approached statistical significance. There was an increased risk of campylobacteriosis associated with fast foods (OR=2.6, 95% CI 1.1-6.1), and consumption of barbecued chicken (OR=10.6, 95% CI 1.0-105.6), but not chicken cooked by other methods. Eating undercooked chicken increased the risk of illness although this was not statistically significant (OR=9.6, 95%CI 0.9-103.0). No increased risk was associated with a wide range of other foods including meat, seafood and dairy products.

This study supports the surveillance data and the MAGIC study that overseas travel is an important risk factor, but is a minor factor in the overall epidemiology of campylobacteriosis in New Zealand.

An outbreak at a family barbecue (17 cases) in Auckland in October 1998 was investigated by a retrospective cohort study (Bishop, 1998). The most likely source of infection suggested by epidemiological results was chicken kebabs, which had by far the highest odds ratio, but this did not achieve statistical significance and may have been subject to recall bias.

During the power shortage in Auckland in February 1998 a notable increase in notifications of *Campylobacter* spp. isolations by community laboratories led to a case-control study (Calder *et al*., 1998). Of 170 sick people from whom isolates had been obtained, 139 were interviewed, along with 106 controls. The study was unable to determine the source of the increase in cases. An increased risk from consumption of raw eggs was significant, but the relevant question included a prompt for mayonnaise, and the mayonnaise consumed may not have actually included raw eggs. Microbiological testing of mayonnaise from the refrigerators of cases did not indicate the presence of *Campylobacter* (which would be expected considering the low pH of mayonnaise from the vinegar ingredient). Several other risk factors had elevated odds ratios which were not statistically significant. These included using a mains water supply, but this was not considered a likely source due to the wide distribution of cases across several water supplies.

Elevated (but not statistically significant) odds ratios were also associated with eating chicken. However, the relevance of chicken as a risk factor was supported by microbiological typing analysis, focused on the 48 human cases infected with serotype HS:1. Poultry samples (18 meat and 3 liver) were taken from supermarkets habitually patronised by these cases. Ten (7 meat, 3 liver) were positive for *Campylobacter*, and of these, 6 isolates were serotype HS:1 with the same PFGE subtype as the cases.

In November 2001 a marked increase in the number of cases of *Campylobacter* infection prompted Auckland District Health Board to initiate an investigation (Simmons *et al.*, 2002a). Investigation of 49 *Campylobacter* cases resulted in 44 from which a dataset of exposures could also be reviewed. Poultry was also sampled. Where poultry had been consumed by cases within the incubation period of illness, details of the brand, preparation (chilled frozen/pieces) and point of purchase were recorded. Poultry of the same brand and preparation was sampled from point-of-sale and cultured for *Campylobacter*. Isolates were then typed by Penner serotyping and PFGE.

A geographical analysis of *Campylobacter* notification rates by Auckland water zones was also performed, but there appeared to be no relationship between the domicile of cases and drinking water sources or local authority distribution zone.

Of the 38 chicken samples taken, 26 (68%) were positive for *C. jejuni*. From these 29 isolates were typed with the most common serotypes being HS:2 (5/29), HS:4 complex (12/29) and HS:27 (3/29). Three isolates were untypable.

Of the 49 human isolates, the most common were HS:2 (9/49), HS:4 complex (16/49) and HS:27 (6/49). Six isolates were untypable.

Isolates from human cases in Wellington during November 2001 were used for comparison. The most common serotypes were HS:2 (19/92), HS:4 complex (46/92) and HS:1,44 (6/92). Eleven isolates were untypable.

PFGE typing showed that a cluster of 19 isolates (8 human and 11 chicken) of Penner serotype HS:4 complex shared an indistinguishable PFGE profile (Pattern 75). There were four clusters of Penner serotype HS:2 of which two were closely related but only one contained human and chicken isolates. There were two clusters of Penner serotype HS:27, both of which contained human and chicken isolates. These results suggested that a number of types were prominent during the study period but that the most important of these was Penner serogroup HS:4 complex, PFGE Pattern 75. This type also predominated in the Wellington study. A comparison of all the Penner serogroup HS:4 complex, PFGE Pattern 75 isolates showed that 34 were indistinguishable (8 Auckland cases, 11 chicken isolates and 15 in Wellington cases).

The serotypes of human isolates from Wellington and Auckland cases correlated (p=0.0056). The serotypes of Auckland human cases and poultry samples were similar but did not achieve statistical significance. These results, along with the additional comparison between PFGE typing results led to the conclusion that the findings were consistent with the hypothesis that chicken was an important source of infection for the Auckland cases, but in view of the diversity of types among the human cases, poultry was unlikely to be the only source of infection.

The normal seasonal pattern of campylobacteriosis notifications in New Zealand is a spring-summer peak followed by a decline in the winter months. The winter increase in Auckland (and many other parts of New Zealand) in June 2002 was investigated by interviews and typing of isolates from 30 cases (Simmons *et al.*, 2002b). Again poultry of the same brand consumed by cases was sampled from nominated retail stores and tested for *Campylobacter*.

Twenty-seven of the 30 cases were able to be interviewed. No novel sources of infection were identified. Twenty-six of the 27 cases had consumed poultry within the 10 day incubation period for illness. Two of the 27 cases reported consuming chicken products from the same takeaway outlet, although the isolates from the cases differed serotypically (one untypable and one Penner serotype HS:4 complex). An audit of the takeaway outlet was conducted as it had been previously implicated in other sporadic cases, and a number of cross contamination issues were dealt with.

A total of 33 samples of poultry were taken, and of these 23 were positive for *C. jejuni*.

Of the 30 human isolates the predominant serotypes were Penner serotype HS:1,44 (9/30), HS:4 complex (13/30) and HS:2 (3/30). The same three serotypes were the most common amongst the 29 poultry isolates: HS:1,44 (7/29), HS:2 (7/29) and HS:4 complex (10/29). However, when an analysis was undertaken of serotypes of isolates from cases and their matched poultry samples (18 isolates) no correlation was apparent ($p=0.8$). This may have been due to the interval of 3-4 weeks between time of exposure and sampling.

PFGE was used to identify two main clusters of isolates in the investigation. HS:4 complex: PFGE 1 (called pattern 75 in the November 2001 investigation above) was indistinguishable from human and poultry isolates in the November 2001 investigation and from a clonal strain identified in Auckland in 1996 (Bloomfield and Neal, 1997). The other cluster (HS:1,44: PFGE 31b) was indistinguishable from the cluster identified in 1998 Auckland outbreak (Calder *et al.*, 1998).

A comparison of serotypes of human and poultry isolates in the November 2001 and June 2002 investigations showed a significant ($p=0.0005$) increase in HS:1,44 isolates in June 2002. Comparison of poultry isolates also revealed a statistically significant ($p=0.0104$) increase in HS:1,44 in poultry isolates in samples linked to cases.

These four investigations utilising typing data to link isolates from humans and poultry provide some support for poultry as a source of human infection. However, the difficulty with such comparisons is the absence of context; insufficient information is available on types present in other potential sources for similar comparisons. As was noted, poultry is likely to be an important source, but not the only one.

4.2.2.3 Hawkes Bay

During May 2001 an outbreak of campylobacteriosis at a boarding school in the Hawkes Bay affected, at its peak, 30 students (Inkson, 2002). Questionnaires administered to those who may have been exposed failed to identify a significant exposure. However, water samples taken as part of the environmental investigation contained *E. coli* as well as *Campylobacter*, in both the treated and untreated water. The outbreak was attributed to contamination of

source water by unfenced grazing cattle, as well as failure of the ultraviolet light treatment system. The same strain of *C. jejuni* (described as based on DNA/PCR analysis) found in the untreated water was also found in cattle faeces from the inlet area.

4.2.2.4 Wellington

A small case-control study was conducted in the Wellington region using telephone interviews of 50 cases of campylobacteriosis notified from February – March 2003, and 50 controls (Baker *et al.*, 2005). The investigation was carried out to investigate peak summer notification levels, but delays meant that by the time the study was conducted the summer peak had passed. The strongest association was for consuming "chicken not cooked at home" in the last three days, but this was not statistically significant (OR=2.13, 95% CI 0.91-4.92). Eating some foods (bacon, pork, yoghurt), as well as drinking water from a water cooler and travel outside the Wellington region were found to be statistically significant protective factors. The authors comment that due to a limited sample size and possible biases between cases and controls the study has limited value.

4.2.2.5 Canterbury

An early review of campylobacteriosis in Canterbury (Faoagali, 1984) was prompted by the observation that the Christchurch District Health Office recorded double the number of notifications of the next highest districts, Wellington and Hamilton. An examination of national cases reported in this paper showed that only 5% of cases were secondary to the identified human index case, and in only 4% of cases was source definitely identified (described as animal/farm, food, toilet/sewage, human overseas and occupational – the latter including a chicken factory).

Two outbreaks of campylobacteriosis at school camps in Canterbury were reported in the early 1980s (Briesman, 1984). The majority of cases were culture confirmed (30/44 and 20/30). The water supplies at each camp could not be ruled out as a source of infection, but most suspicion fell on raw milk derived from local farms. Although *Campylobacter* was not isolated from water or raw milk samples collected after the outbreaks (no food or water samples consumed by cases were available), only children were affected, and it was believed that children were more likely to consume the raw milk than adults.

Prior to the case-control study in Christchurch in 1992-1993 (Ikram *et al*., 1994) two studies of the epidemiology of *Campylobacter jejuni* infections in Canterbury were published (Briesman, 1985; Briesman, 1990). Cases between 1981 and 1983 occurred mostly in summer and in the age groups children under 5 years and young adults (15 to 35 years). The rate of illness in rural areas (small towns) was higher than in urban areas. For this reason animal contact was suggested as a risk factor. The more recent analysis covered a wider range of years (1981 – 1988). The paper cites animal data which show a peak prevalence in sheep and dairy cows in late winter/autumn (August – September) which did not correlate with the peak in human cases in spring/summer (November – December – January).

An outbreak of campylobacteriosis in Ashburton in March 1986 was signalled by an unexpectedly high number of notifications: 19 in the two week period from 26 March (Briesman, 1987). Interviews with cases revealed no significant relationships to animals or

consumption of various foods. The only common feature was consumption of water from the borough supply.

Chlorination of the borough water supply was implemented only after heavy rain when river flow rates were high. Heavy rain occurred on the night of 12 March and chlorination commenced the following morning. A water sample taken before the chlorination point on 13 March showed a presumptive faecal coliform count of greater than 180 per 100 ml suggesting that the rain may have washed *Campylobacter* from the surrounding beef and sheep farms into the river. No confirmatory detection of *Campylobacter* in water samples was reported, although a fresh water survey in New Zealand determined that the critical value for *E. coli* as an indicator of increased *Campylobacter* infection was in the range of 200-500 *E. coli* per 100 ml (McBride *et al.*, 2002).

An outbreak of campylobacteriosis at a camp and convention centre near Christchurch was initially detected by the hospitalisation of two persons living at the camp (Stehr-Green *et al.*, 1991). A total of 44 cases was eventually identified, 11 of which were culture-confirmed. Compared with unaffected persons at the camp, cases were more likely to have drunk unboiled water from the water supply, obtained from four springs on the property. After controlling for water consumption, no association between eating food from the camp and illness was seen. Samples from all four springs showed elevated faecal contamination, and three weeks later, although the springs had returned to low faecal contamination levels, holding tank samples still showed elevated levels of faecal contamination.

The similarity in symptoms and time of illness between non-culture confirmed cases strongly suggested that they were also infected with *Campylobacter*. Heavy rain in the middle of the month of the outbreak could have caused pasture run-off and spring contamination, although some cases occurred earlier. Thus epidemiological and microbiological data strongly suggested the water supply as the source of infection. Control measures, including installation of a water treatment system were implemented, and since then no further cases of enteritis had been reported.

A further outbreak in Christchurch has been linked to a water supply (Bohmer, 1997). A total of 67 cases (5 confirmed and 62 suspected) of campylobacteriosis at a school holiday camp occurred in January 1997. A retrospective cohort study identified a contaminated water supply as the most likely source of infection (relative risk 1.51, 95% confidence interval 1.07-2.12). Laboratory testing revealed 95 faecal coliforms per 100 ml in the camp water supply (a borehole) and *Campylobacter* was detected in a water sample from a nearby stream.

An outbreak of campylobacteriosis at a Christchurch boarding school in October 1992 affected 84 students and staff (Mitchell *et al.*, 1993). The majority of cases (75/84) were boarders and five cases were culture-confirmed. There were no statistically significant differences between cases and controls in the meals they ate or the water they drank, but cases were confined to those who had eaten food in the dining hall. Although *Campylobacter* was not detected in food samples, it was noted that sparrows had ready access to the dining hall and kitchen, which may have been a source of contamination. Results from this case-control study were inconclusive.

In December 2000 Crown Public Health investigated an outbreak at a restaurant in Christchurch (Whyte *et al.*, 2001). All 12 cases had eaten chicken liver pâté as an entrée. The investigation revealed that the most likely cause of contamination was the under-cooking of chicken livers use to prepare the dish.

A small outbreak of campylobacteriosis in Christchurch in 2003 involving three cases was attributed to the consumption of pre-cooked cocktail sausages (Graham *et al.*, in preparation 2005). The information collected suggested that cross-contamination was the cause, as the organism was not found in the interior of the sausages. The premise involved was found to have repacked bulk cocktail sausages on a bench also used to repackage raw meats and poultry.

A report by the Canterbury Public Health Service provider, Community and Public Health, discusses "Campylobacteriosis in Christchurch City 2003" (CPH, 2004). The incidence of campylobacteriosis in Canterbury and Christchurch has historically exceeded national rates. The rates were elevated across most age ranges of people over the age of 10, whereas rates in young children were below the national rates. Rates were also elevated across all ethnic groups. Examination of risk factors from notified cases showed that 6.5% of infections were probably acquired overseas. Infection amongst agricultural and animal contact workers was, as expected, more common amongst cases that occurred in rural areas, although the number of cases amongst these occupational classes represented only 72 of the total of 1916 notified cases. Fifteen cases had consumed drinking water from an untreated source, and thirty eight cases reported recreational water contact prior to the onset of infection.

Analysis was undertaken of food premises from which cases reported eating food prior to infection (329 cases reported consuming such food). The categories most common were "café, bar, restaurant, bistro, hotel", and "fast food outlets (franchised)". One hundred and six cases were identified as being workers at Christchurch food premises.

4.2.2.6 Comments

The studies above are useful in illustrating a number of potential transmission routes but do not generally indicate their relative importance. The evidence for transmission, particularly for suspected waterborne outbreaks, is not strong. Some stronger evidence for a link between human cases and poultry is provided by typing data in Auckland, but comparative information from other matrices is lacking. Baseline material data are also lacking.

4.3 Potential Transmission Routes of *Campylobacter* from Environment to Humans: Ashburton Study (Baker *et al.*, 2002).

A major study of transmission routes in the Ashburton area investigating environmental and waterborne sources of *Campylobacter* was completed in 2002. The research was a joint effort by the Ministry of Health, ESR, the University of Canterbury, Crown Public Health, the Ashburton District Council and the Massey University EpiCentre. The focus was on comparing the types of *Campylobacter* present in human cases, river water, animal faeces, meat offal and raw chickens. Typing and epidemiological information was collected.

The Ashburton District was selected for study because the South Canterbury Health District is consistently among those health districts with higher than average rates of campylobacteriosis.

The sample collection period was for the calendar year of 2001, although human clinical samples were collected until the end of January 2002, to account for the incubation period of the organism. Environmental matrices included in the study were: river water, duck faeces, ruminant animal faeces (beef, dairy cattle and sheep) and meat products. The subtypes isolated from these sources were compared with the subtypes isolated from human cases of campylobacteriosis in the study area. When human cases were notified, samples were collected and a questionnaire administered to attempt to identify risk factors that may have been responsible for the infection.

The prevalences from the various samples were:

- human faeces from confirmed cases: *C. jejuni* 57/69 (83%); *C. coli* 6/69 (9%)
- beef offal: *C. jejuni* 15/178 (9%); *C. coli* 1/178 (0.6%)
- sheep offal: *C. jejuni* 63/162 (39%); *C. coli* 6/162 (4%)
- pork offal: *C. jejuni* 9/187 (5%); *C. coli* 9/187 (5%)
- chicken carcasses: *C. jejuni* 56/204 (28%); *C. coli* 2/204 (1%)

The prevalence in fresh chicken was around half that expected from previous studies. This might be because whole chicken carcasses, which were tested in this study, are less frequently contaminated than portions.

The composite sampling regime used for animal faecal samples and water generated a relatively high percentage of positive samples from these matrices. Therefore the data produced by the *Campylobacter* Transmission Routes (CTR) study from ruminant animals and ducks do not represent isolation rates for individual animals (and so are not included in this document).

All the human cases appear to have been sporadic infections. There was no evidence of common source outbreaks in this population. Person-to-person contact with another case was only reported by eight cases (14%). None of the eight cases was identified definitively as a secondary case, due to limited information recorded on the timing and nature of the contact, plus the fact that very few of the related cases had provided a faecal sample for testing.

Analysis of *Campylobacter* isolates revealed a high diversity of subtypes of *C. coli* and *C. jejuni* within each matrix. There were overlaps of subtypes between matrices, which have been informative in demonstrating potential linkages.

- A total of 250 serotype:PFGE subtypes of *C. jejuni* were isolated from matrices in the CTR study. Of these, 44 (19%) were isolated from humans.
- A total of 39 PFGE subtypes of *C. coli* were isolated from matrices sampled in the CTR study. Of these, 5 (13%) were isolated from humans.
- The range of subtypes infecting humans was diverse. There were 44 subtypes of *C. jejuni* found for 56 human isolates (diversity of 78.5%) and 5 subtypes of *C. coli* for 6 human isolates (diversity of 83%).

- Twenty-one subtypes of *C. jejuni* were unique to humans in this study, and these subtypes accounted for 46 % of cases.
- There were 27 human *C. jejuni* cases (48%), infected by subtypes found in other matrices. These 27 cases were used to explore potential relationships with subtyping information obtained from samples collected from other matrices.
- For *C. coli* all of the PFGE subtypes found in humans were also found in other matrices.

The data were too sparse in that there were too many *Campylobacter* subtypes distributed among the small number of human cases for firm conclusions to be made from risk factor analysis. However, indicative results were that contact with bovine animals (dairy, non-dairy, or calves) and live chickens are the more important risk factors for this study population. These judgements were based on whether the human cases reporting risk factors were associated with different subtypes (at the serotype and serotype:PFGE level) than those cases that did not report the risk factor. It is worth noting that 21/56 cases reported bovine exposure, and 12/54 reported live chicken exposure (no live chickens were sampled for *Campylobacter*).

Comparison of *Campylobacter jejuni* subtypes found in human faeces and other matrices was achieved by statistical analysis (the "Czekanowski" index). Analysis of serotype data only, showed that the matrix with the greatest similarity with human isolates was beef faeces, and sheep offal, with dairy faeces and sheep faeces close behind. Analysis using PFGE typing, or serotype and PFGE typing together, provided much lower indices, although the same matrices were prominent (note that chicken carcass and water isolates ranked at a similar level to dairy faeces when PFGE types were compared with human cases).

Analysis on a case-by-case basis by use of bacterial subtyping, temporal and geographical data largely failed to provide compelling evidence to identify transmission routes/linkages definitively. Any analyses of this nature were necessarily complicated by the numerous potential exposures reported by the cases.

The main conclusion that can be drawn from the analyses is that, for the population sampled (a semi-rural population), bovine animal contact, direct or indirect, was the highest risk factor identified in the study.

Limitations of the study were its small size, the lack of dominant subtypes, and the limited overlap between types from human cases and matrices sampled. The study also noted: "Given the previous data for New Zealand which are available, there may be two epidemiologies that predominate, a rural ruminant exposure epidemiology, and an urban one which may involve poultry and possibly other unknown exposures. This last point can be inferred from the large New Zealand case control study (Eberhart-Phillips *et al*., 1997), whose participants were principally located in the four main centres."

4.3.1 Comments

This study is considered to have value in illustrating the prevalence of *Campylobacter* throughout environmental, animal and food sources, and the myriad types present illustrate the difficulty in linking sources and cases. Despite the identification of bovine contact as the dominant risk factor in this analysis, only just over a third of the cases reported this exposure, and discrimination (based on the index statistical method) between matrices was not marked.

For more than half the human cases, the *Campylobacter* subtype present was not identified in any of the matrices sampled. As acknowledged in the report, the risk factors identified are likely to apply mainly to rural populations, but it is difficult to base an epidemiology on 27 cases.

4.4 Regionality (Hearnden *et al.*, 2003)

An examination of regional and temporal variation in campylobacteriosis notification rates provided evidence of differences in seasonality of the illness within New Zealand. Data from 1993 to 2000 were examined on the basis of annualised rates for 75 local authorities. Seasonality appeared to be different between the northern and southern regions of New Zealand, with three seasonal patterns being identified:

1. Low summer incidence and small inter-seasonal variation: The Far North and much of the rural North Island;
2. Higher summer incidence and more seasonality: North Island urban areas (Auckland, Hamilton, Napier and hinterlands) plus a few South Island areas;
3. Highest summer incidence and strong inter-seasonal variation: Christchurch, Dunedin, much of the South Island and lower North Island cities of Wellington and Upper Hutt.

These patterns were taken to suggest that the importance of transmission routes may vary regionally in New Zealand, with a complex ecology unlikely to be explained by a single dominant transmission route across all groupings.

However, as noted in Section 2.1, regions with higher rates are not consistent from year to year, and rates may be affected by a number of reporting factors as well as epidemiology.

5 PREVALENCE AND TYPES OF *CAMPYLOBACTER* FROM HUMAN CASES, ANIMAL SOURCES, AND FOOD SAMPLES IN NEW ZEALAND

Information in this section has been organised according to the study. Each study usually addresses samples for a variety of sources.

5.1 BRENDA Typing (Kakoyiannis *et al.*, 1988)

An early study of types of *Campylobacter* in New Zealand came from Massey University. A restriction endonuclease digestion followed by gel electrophoresis (a technique known as BRENDA) was applied to a total of 731 thermophilic *Campylobacter* isolates from a variety of sources (human: 341; chicken: 128; pig: 147; duck: 9; gull: 88; horse: 1; rat: 18). The isolates comprised 473 *C. jejuni*, 215 *C. coli* and 43 *C. lari*.

A total of 316 human *C. jejuni* isolates produced 60 distinguishable patterns. The poultry isolates of *C. jejuni* (98) produced 23 patterns. Eleven of the poultry isolate patterns were the same as patterns observed amongst the human isolates, and these patterns made up 113 (35%) of the total human isolates tested.

Human isolates of *C. coli* (25) gave 24 unique patterns and two of these were indistinguishable from the three patterns from *C. coli* isolates from chicken (30). Another human isolate gave a pattern indistinguishable to that of a pig isolate.

From the 147 pig isolates of *C. coli* 76 patterns were observed. Only one pig isolate had a pattern indistinguishable from the pattern of a *C. coli* isolate from a human.

The single isolate from cattle examined had a pattern indistinguishable from that of an isolate from a human.

Very high rates of contamination of samples (generally 100% or close) were observed from samples from poultry farms or retail samples.

The authors asserted that the high similarity in patterns between poultry and human isolates supported the view that chickens are an important source of infection for humans. However, the source of the isolates was not clear; it seems that most derived from faecal samples from animals, although some came from carcasses, and some from retail poultry. The lack of commonality between duck or gull and human isolates is noteworthy, given the very limited amount of information on wild birds in New Zealand.

5.2 Typing Information from Human and Non-human Isolates (Nicol and Wright, 1998)

A study of the value of different typing methods for *Campylobacter* was conducted in 1998 by examining human and non-human isolates (isolates from sheep, cattle, dogs and pigs were all faecal isolates, the poultry isolates were obtained from caecae, neckflaps, and carcasses).

In Study 1, 213 isolates, 143 human and 70 non-human, were Penner serotyped. The most common serotype for human isolates was HS:2. Bovine and ovine sources were predominantly HS:2 and HS:4.

In Study 2, 246 isolates from human clinical diagnostic laboratories, showed that the most common types were HS:2 (16%), HS:4 (8%) and HS:8 (8%).

Study 3 was conducted in one region over a two month period. One hundred and eighty isolates (65 human and 116 non-human) were serotyped and PFGE typed. Of the HS:2 isolates (10% of total) 42% were HS:2: PFGE 18. This type was isolated from humans, poultry, cows and sheep. Of the HS:4 isolates, 26% were HS:4:PFGE19 isolated from humans, poultry and untreated water.

These studies provided background data on serotypes for 412 cases of sporadic campylobacteriosis in New Zealand (HS:2, 23%, HS:4, 11%, HS:11, 6%, HS:1, 5%, HS:8, 5%). These data were used for comparison with findings in three separate outbreaks, but the results from only one outbreak were reported.

Outbreak 1: A spike of campylobacteriosis in a large urban area provided 114 isolates, 35 (31%) of which were HS:4. Of the isolates typed after the peak, 11% were HS:4. A subset of 20 serotype HS:4 isolates were further typed using PFGE and 19/20 gave an indistinguishable pattern, and HS:4:PFGE 1 was classed as the outbreak strain but no food vehicle or common source was identified.

The main value of these studies is to identify the most common serotypes amongst human cases in New Zealand. The predominance of HS:4 and HS:2 continues amongst human isolates, as along with HS:1,44 they were the most commonly isolated serotypes from human cases 1994 – 2001 (Lake *et al.*, 2004).

5.3 Human, Animal, Food and Water Sources in Canterbury (Hudson *et al.*, 1999)

A study of isolates from human, animal, water and food sources in the Canterbury region was made during two 4 week periods: August 1996 (winter) and February 1997 (summer). Samples were obtained from:

- A local medical laboratory, faecal samples from Christchurch residents with diarrhoea;
- A local veterinary laboratory, faecal samples from animals with diarrhoea;
- Water sources chosen on the basis of their potential to contain *Campylobacter* (three sites on rivers draining agricultural land (two of which had notices advising against bathing or drinking the water), a stream draining a stockyard site, a pond in a park that was frequented by ducks, and outflow from the city sewage works at the start of an oxidation pond system; four of these sites could be described as recreational);
- Raw chicken portions from 10 retail outlets;
- Raw milk from farms.

Heat stable Penner serotyping and PFGE restriction profiles were determined from isolates taken from positive samples.

5.3.1 Human cases

In August 1996 isolates from 19 cases were obtained, and 46 in February 1997. The predominant serotypes were HS:4 complex (10/19) in August 1996, and HS:2 (12/46) and HS:33 (10/46) in February 1997. Amongst the isolates from August 1996 no single PFGE type was dominant, but in February 1997 the most common types were 18, 18A and 18B (10/46) and 25 (12 of 46).

5.3.2 Animals

Thirteen positive samples in August and five in February were obtained from veterinary sources (dog (1), sheep (11), pig (1), cattle (4), swan (1)). Thirteen isolates from these sources were typed with no single serotype or PFGE type predominating.

5.3.3 Chicken from retail outlets

Of the 113 raw chicken samples 56.6% were positive for *Campylobacter*. A total of 30 isolates were typed from August 1996 and 28 from February 1997. Of the 30 isolates that were serotyped from August 1996 no one type predominated, and the highest proportion were untypable. Isolates from February were predominantly serotype HS:21 (10/28). The PFGE types amongst the chicken isolates were reasonably consistent, with the majority being PFGE Type 25 (11/30 in August 1996 and 15/28 in February 1997). PFGE Types 18, 18A and 18B were infrequent (2/30 in August 1996 and 0/28 in February 1997).

5.3.4 Milk samples

Of the 111 raw milk samples tested only one yielded *Campylobacter*.

5.3.5 Environmental water samples

Most of the August 1996 (winter) water samples (27/36) were positive for *Campylobacter*, while the February 1997 (summer) samples were less likely to be positive (15/48). A large proportion (5/9) of the isolates identified from water samples in February 1997 were *C. coli*.

The serotypes and PFGE types detected were again varied with no single serotype predominant.

5.3.6 Overall typing results

The most common serotypes from all sources present in the winter month (August 1996) were HS:4 complex (18/79) HS:12 (10 of 79) and HS:2 (9/79). There was a marked shift in the summer month (February 1997) with HS:4 complex and HS:12 being less common (6/83 and 2/83 respectively), and HS:6, HS:21 and HS:33 becoming more common (13/83, 12/83 and 16/83 respectively).

Shifts were also observed from winter to summer in the PFGE types observed. Type 25 was the most common in both months (14/79 and 28/83 respectively), but the next most prevalent in winter was PFGE Type 4 (11/79) while in summer it was PFGE Types 18, 18A and 18B (11/83 – almost all isolates from human cases).

5.3.7 Indistinguishable isolates by both typing methods

A number of isolates was found to be indistinguishable by both typing methods. The data from these isolates also suggested a shift between August and February, with the most common type in February (9 human isolates and 5 chicken isolates) being absent in August.

The indistinguishable isolates found in both human cases and chicken samples were considered support for the epidemiological links between campylobacteriosis and the consumption of chicken. Two of these types were also found in water samples in August which prompted the authors to suggest that recreational water exposures should be further investigated.

This study draws a link between human cases and poultry based on PFGE Type 25, although the most common poultry serotype in February (HS:21) does not feature amongst the human isolates. As with the Ashburton study, the plethora of types present in each matrix makes linkages difficult to establish.

5.4 Raw Milk (Stone, 1987)

During 7 months of the 1986/87 dairying season, 71 raw whole milk samples were tested for *Campylobacter jejuni*, *Listeria monocytogenes* and *Yersinia enterocolitica*. None of the milks were positive for *C. jejuni* or *L. monocytogenes*, but three of the early season milks were positive for *Y. enterocolitica*. Sixteen milk samples were positive for other species of *Listeria*.

5.5 Survey of Contamination of Poultry on Farms (Boxall, in preparation)

A PhD study on *Campylobacter* on poultry farms in Auckland and the Waikato has been conducted, with sampling and testing conducted during the year 2000. The part of the study relevant to this report was the prevalence study to observe how many farms, sheds and birds were colonised with *C. jejuni*.

The results were:

(Industry averages prior to first partial depopulation, cloacal swabs collected from birds)

Farm prevalence (95% CI): 14% (1-41%)

Shed prevalence (95% CI): 10% (0-32%)

Bird prevalence (95% CI): 63% (43-86%)

A further part of this PhD study examined the free available chlorine levels in drinking water provided to broiler chickens (Boxall *et al*., 2003). This followed a study of risk factors for *Campylobacter* colonisation in broilers which unexpectedly showed that chlorination of the water supply was a risk factor. The drinking water study revealed that the free available chlorine levels actually present in the drinking water were low, and unlikely to have a sufficient controlling effect on *Campylobacter* levels.

5.6 Poultry and Poultry Products (Campbell and Gilbert, 1995).

A microbiological survey of 159 raw poultry samples collected at random from New Zealand poultry processors in 1992-1993 found that 82 samples (52%) were positive for *Campylobacter*. However, only one sample (0.07%) of ready-to-eat poultry products tested positive for the organism.

5.7 "Consumer" Magazine Surveys of Poultry (Anon., 1999; Anon., 2003c)

In 1999 the magazine "Consumer" published a survey of 50 fresh chickens which were tested for *Campylobacter* and *Salmonella*. Of the 50, 41 were contaminated: 27 (54%) with *Campylobacter*, and 17 (34%) with *Salmonella*.

A further survey in 2003 tested 25 cooked rotisserie chickens and 25 smoked chickens. None tested positive for *Campylobacter*, but out of 40 samples of raw fresh chickens (covering 11 brands) 34 (85%) were positive.

5.8 Packaging on Retail Poultry Samples (Wong *et al.*, 2004)

In early 2002 ESR conducted a survey of three hundred retail packs of fresh chilled poultry from 15 supermarkets in the Christchurch area. The purpose was to determine the prevalence of *Campylobacter* on the exterior of packs, and was prompted by findings in Wales and London. The results were (Wong *et al.*, 2004):

- 24% of chicken packs were externally contaminated with *Campylobacter jejuni*.
- Offal samples had the highest rate of external contamination (52%) followed by whole chickens (34%) and chicken portions (14.5%).
- Of the 300 packs sampled, 32 were positive but with low *Campylobacter jejuni* counts of <6 MPN/pack, 26 packs recorded counts in the range of 6-200 MPN/pack, and 9 samples recorded 480-2200 MPN/pack. Two packs of chicken livers had outer packaging counts of >2200 MPN/pack.

The external contamination of whole chicken packs should have been reduced over the last year or so, as processing companies and supermarkets have introduced leak-proof packaging.

5.9 Shellfish (Hudson, 1999)

A survey was conducted of fresh shellfish obtained from wholesalers, packhouses and farms from the major commercial shellfish growing areas of New Zealand. A total of 197 samples of oysters, scallops and mussels were tested for *Campylobacter* spp. No sample tested positive for pathogenic *Campylobacter* species, although four samples were positive for *C. lari*, which is an infrequent cause of infections in humans. These samples were all from one area.

The low level of pathogenic *Campylobacters* in shellfish in New Zealand is in contrast to levels found overseas. This suggests that commercially harvested shellfish are not an important source of human *Campylobacter* infections in New Zealand. This tentative conclusion is consistent with the result from the MAGIC study that raw or undercooked

shellfish were not a statistically significant risk factor. However, the report noted that the significance, if any, of recreationally harvested shellfish, remains unknown.

5.10 Meat and Cooked Foods (Gill and Harris, 1982a)

Although not a prevalence study, this report describes an investigation into the growth of *C. jejuni* in various meat based materials. Of importance to this report are the results of storage on meat. Frozen storage (-18°C) resulted in a decrease in *Campylobacter* numbers during the first two weeks but there was little subsequent change. The number of cells decreased by about one order of magnitude on high pH (6.4) meat, but by two orders of magnitude on normal pH (5.8) meat. Bacterial numbers on chilled (-1°C) high pH meat declined slowly throughout the storage period but declined rapidly on chilled normal pH meat until bacteria could not be detected (after 8 days). Storage of meat at 25°C resulted in equally rapid declines on both high and normal pH meat.

A long lag phase (1-2 days) when grown in cooked meat medium at 37°C, as well as death in meat pies stored at 37°C and 43°C suggested that there would be few circumstances under which bacterial numbers would increase in a warm food. The authors concluded that large doses of the organisms from meat sources were unlikely, although the consumption of small doses was possible, and better dose-response information was needed.

5.11 Red Meat Carcasses (Gill and Harris, 1982b)

Faecal samples (from viscera shortly after removal from carcasses), meat and work surfaces were examined in this study based at a single abattoir. Faecal samples from two of four groups of sheep included several animals carrying the organism, but only one positive sample was obtained from 42 lambs and none from 65 cattle. *Campylobacter* was consistently present only in unweaned calves.

Small numbers of *C. jejuni* could be isolated on some occasions from a variety of work surface sites, but the only surfaces with a high density of cells and rate of contamination were full viscera trays. Routine cleaning appeared to be largely effective in eliminating *C. jejuni* from these trays.

Consideration of the efficiency of sampling and recovery led to an estimation that initial *Campylobacter* contamination of both carcass and equipment surfaces would generally be of the order of 1-10 organisms/cm^2. It was also concluded that the organism does not persist in the work environment, and would probably decline during chilling.

5.12 Red Meats: Sausage and Hamburger (Hudson and McGuire, 2002).

One theory proposed to explain the summer peak in campylobacteriosis in New Zealand is that the warmer months result in more barbecued foods being consumed with consequent lapses in good cooking and hygiene practices. To investigate this issue a survey of 100 samples each of sausage and hamburger was conducted to determine the presence and number of *Campylobacter*. None of the foods tested were found to contain *Campylobacter*. This may have been due to the use of frozen meat in production, or possibly the effect of other ingredients.

5.13 Hamburgers and Broiler Chickens (Gill and Harris, 1984)

This investigation was primarily designed to assess the degree of contamination of ground meat and chicken and the effects of freezing and cooking.

Campylobacter spp, were not isolated from 50 samples of ground beef obtained over a period of 2 months from 27 outlets (detection limit 2 cfu/g). Chilled and frozen chicken carcasses were obtained from 21 retail outlets over a 3 month period. All isolates were identified as *C. jejuni*. About 70% (15/22) of the chilled carcasses were contaminated by *C. jejuni* with levels ranging from 10^2 – 10^5 per half carcass (mean 6 x 10^4 per half carcass). Frozen chickens were less contaminated, and at lower levels; approximately 16% (6/37) were contaminated, with a range of 10^1-10^3 per half carcass (mean 8 x 10^2 per half carcass).

Freezing (-18°C for 7 days) of inoculated ground beef reduced numbers of *C. jejuni* by approximately 1 log cycle. Cooking of hamburgers for 2-8 minutes (depending on thickness) on a hotplate at 175°C was sufficient to eliminate *C. jejuni* even though the centre of the hamburgers still appeared raw.

Chickens roasted in a conventional oven for up to 25 minutes at 190°C or in a microwave at 600W for up to 20 minutes eliminated *C. jejuni*. It was concluded that minimal cooking of all muscle tissue would eliminate the organism.

Several half carcasses with moderate *C. jejuni* contamination (10^3-10^4 per half carcass) were cooked and then carved on the same surface with the same implements, left unwashed after preparation of the raw chicken. No *C. jejuni* was subsequently isolated from any work or instrument surfaces, or from 4/6 carved meat samples. Single *C. jejuni* colonies were observed with 10 ml samples of meat homogenate from two carcasses, indicating recontamination at the level of 10 per half carcass.

The authors of this study concluded that it seemed unlikely that there was a link between ground beef and human campylobacter infection in New Zealand. Cooking easily eliminates *C. jejuni* from chicken carcasses and it was also concluded that heavy contamination of chicken carcasses would be necessary to achieve detectable cross contamination.

5.14 Surveys of Retail Samples for the NZFSA

During 2003-2004 a national retail survey of minced and diced meat from supermarkets and butchers was undertaken by ESR on behalf of the NZFSA to assess the prevalence of food-borne pathogens in meat. The part of the study focusing on *Campylobacter* has been completed and a draft report written (ESR FW 0478; Wong *et al.*, paper in preparation). Results for red meat are included here for comparison. The results are shown in Table 11.

Table 11: National retail survey of *Campylobacter* in chicken and red meat; July 2003 to June 2004

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

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

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

The ESR surveys of retail chicken mince indicate a range of contamination of <0.3 – 110 MPN/g. If it is assumed that a chicken carcass of approximately 2.5 kg yields 1.5kg of meat, then carcass contamination could be of the order of <500 - 1.6 x 10^5 cfu/carcass. This very approximate calculation suggests that carcass contamination is of a similar order to that seen in the United States (Stern and Robach, 2003). These numbers are also somewhat higher than those found in retail chicken in the Netherlands (DuFrenne *et al.*, 2001).

The counts from red meat samples appeared to be lower than for poultry:

Beef:	8/8 samples	<0.3 MPN/g	
Bobby veal:	7/8 samples	<0.3 MPN/g	1/8 samples >10.9 MPN/g
Lamb or mutton:	13/15 samples	<0.3 MPN/g	2/15 samples 0.3 MPN/g
Pork:	17/18 samples	<0.3 MPN/g	1/18 samples 0.3 MPN/g

Although the absence of higher counts may only be a reflection of the low numbers of positive samples, it seems a reasonable conclusion that overall counts from red meat are lower.

A total of 247 isolates were subtyped by serotyping and PFGE. Eighty five subtypes were obtained (Wong *et al.*, 2005). When the subtypes were compared to historical data from human cases (approximately 330 isolates), 6/7 beef isolates, 7/15 mutton/lamb isolates, 8/18 pork isolates, 84/204 poultry isolates, and 1/8 veal isolates were common to both sources

5.15 Sheep Liver (Cornelius *et al.*, 2005)

In this study fresh sheep liver samples were obtained from butchers and supermarkets in Christchurch. Of the 272 samples collected, 180 (66.2%) contained *Campylobacter*. The positive samples comprised 100 at <3 MPN/g, 36 at 3-9 MPN/g, 32 at 10-99 MPN/g, and 12 samples (4.4%) at >100 MPN/g.

An analysis of the isolates from the liver samples, as well as 200 isolates from human cases (also from Christchurch over the same time period) was conducted using both serotyping and PFGE. More than half (61.1%) of the *C. jejuni* isolates from liver were of subtypes that were also isolated from human cases.

5.16 Chicken Livers

Since it has been recognised in recent times that poultry offal may be a transmission route for campylobacteriosis (Whyte *et al*., 2001) ESR CSC has performed some analyses of chicken livers to enumerate *Campylobacter* on the external surface, as well as within the tissue (Rosemary Whyte, ESR Christchurch Science Centre, pers. comm.). Of the 30 livers tested 29 (96.7%) contained *Campylobacter* on external surfaces and 27/30 (90%) were contaminated internally. The modal number was between 100 and 1000 per liver, while at the higher extreme one liver contained $>10^5$ *Campylobacter*. Most of the isolates were identified as *C. jejuni*. The count per gram in the modal range, given a mean liver weight of approximately 50g is 2-20 *Campylobacter*/g.

5.17 Offal (liver) (Hudson, 1997)

A small, unpublished ESR survey (Hudson, 1997) of offal samples detected *Campylobacter* in 9 of 22 (40.9%) ruminant (2 bovine and 7 ovine) offals tested.

5.18 Watercress (Edmonds and Hawke, 2004)

Watercress is harvested from waterways both for personal use and for commercial sale in New Zealand. This study examined microbiological contamination in water and watercress samples from a variety of sites in the lower North Island. The stream sites selected covered a range of urban, semi-urban and rural catchments and a range of water quality and sediment characteristics. The sites selected were representative of catchment types found elsewhere in New Zealand.

All of the sites showed variable but significant levels of *E. coli* in both the watercress and water samples and therefore the potential for enteric waterborne pathogens to be present. *Campylobacter* was detected in 80% of the samples from the growing waters (across all sites) and in 11% of the watercress samples.

5.19 Hydroponically-Grown Vegetables (Graham and Dawson, 2002)

A survey in 1998-1999 of 291 samples of hydroponically-grown vegetables in New Zealand (117 sprout samples, 114 leafy vegetables, 60 herb samples) conducted analyses for a range of pathogens, including *Campylobacter*. No *Salmonella*, *Campylobacter*, *E. coli* O157 or *L.*

monocytogenes were detected, although *Campylobacter* would not be expected to be present in such growing systems..

5.20 Dairy Farms – Studies at Massey University

A study by Massey University (Adhikari *et al*., 2002) examined the prevalence of *Campylobacter* spp. in dairy cows (52 faecal samples), sparrows (53 droppings), rodents (rectal contents from 65 trapped rodents) and flies (56 trapped on farm) and compared the isolates using PFGE typing. The study was conducted at No. 4 Dairy Unit at Massey University. Additional samples taken included: sparrow droppings (56) from an urban area in Palmerston North, and environmental samples from the farm (silage, aprons, boots, water from grazing troughs).

Only *Campylobacter jejuni* was isolated from all samples. The prevalences were:

Dairy cows	53.8% (28/52)
Sparrows (urban)	39.6% (21/53)
Sparrows (farm)	37.7% (20/53)
Rodents	10.8% (7/65)
Flies	8.9% (5/56)
Silage	40% (2/5)
Boots	100% (2/2)
Aprons	50% (1/2)
Water	100% (2/2)

A presentation to MAF in 2003 by researchers at Massey University indicated that the prevalence of *Campylobacter* in faecal samples of cows from No. 4 Dairy Unit was highly variable (12-81%) depending on the time of year and type of cow. The researchers postulated that mechanisms of infection and reinfection were occurring, and shedding by cows may be periodic and this should be considered when evaluating transmission routes.

5.21 Dairy Cows (Wu, 2001)

Research at Massey University No. 4 dairy farm showed a prevalence of 52% in dry cows, 15% in cows after calving, 81% in heifers, 37% in yearlings and 22% in calves (Wu, 2001, referred to in Adhikari *et al*., 2002). An earlier study (Ahmed, 1999 referred to in Adhikari *et al*., 2002) isolated *Campylobacter* from 53.7% of dairy cattle on the same farm, and from 56% of environmental samples collected from beef and sheep abattoirs.

5.22 Possums and Rabbits (Savill *et al.*, 2001a)

To investigate the hypothesis that feral rabbits and possums were acting as environmental reservoirs for *Campylobacter*, faecal samples were collected from shot animals around Ashburton. Samples from a total of 72 rabbits and 197 possums were tested. The isolation rate of *C. jejuni* was zero, while *C. coli* was detected in one rabbit sample. This suggested that these animals were not acting as a reservoir for *Campylobacter*, at least in the Ashburton region of New Zealand.

5.23 Dairy Cattle and Sheep (Meanger and Marshall, 1989)

The seasonal prevalence of *Campylobacter* in dairy cows as determined from rectal swabs of cows on Massey University farms has been reported. The prevalence was 24%, 31% and 12% during summer, autumn, and winter respectively. *C. jejuni* and *C. coli* were isolated in approximately equal proportions.

Rectal swabs from sheep were also examined, and 27 isolates were typed although the prevalence was not reported. A comparison of the cow and sheep isolates was undertaken using the BRENDA typing system. Seventeen different BRENDA patterns were produced from the isolates from dairy cows, and six from the 27 sheep isolates. Of these 21 patterns, only two were common to both sheep and cattle.

5.24 Summary

The studies cited above provide data which can be used to give a limited overview of the prevalence of *Campylobacter* contamination in some foods and animals in New Zealand.

Retail poultry has been found in several surveys (including national surveys) to be contaminated with *Campylobacter* at up to 90%. Cooked ready-to-eat poultry products are infrequently contaminated.

Red meat is less frequently contaminated than raw poultry, with prevalences ranging from 0% on hamburger and sausage, to between 3 and 13% on beef, lamb, pork and bobby veal. Again the sampling for these results was national.

Offal is frequently contaminated, particularly chicken livers (90%).

Counts of bacteria appear to be lower on red meat than poultry, although the number of positive results was fewer.

Data for other foods are very limited, but suggest infrequent contamination. Two small (and old) surveys of raw milk did not find frequent contamination by *Campylobacter*.

The data on carriage rates in animals is insufficient to generalise, particularly as the high carriage rates determined in the Ashburton study cannot be used, due to compositing of the samples. The information available suggests that dairy cows and beef cattle are frequently contaminated, perhaps up to 50%. Most of the results however, derive from studies at Massey University and may not reflect the national situation.

Information on wild animals indicates that possums and rabbits around Ashburton rarely carry *Campylobacter*, while sparrows are frequent carriers (40%), at least around Massey University.

6 ENVIRONMENTAL CONTAMINATION INFORMATION FROM NEW ZEALAND

6.1 Rivers (Donnison and Ross, 1999)

A study of faecal pollution in New Zealand rivers used freshwater mussels as a sentinel organism, as well as direct analysis of waste waters that had undergone minimal treatment.

Campylobacter jejuni (and occasionally *Campylobacter coli*) were detected in the majority of sentinel mussel samples located at waste water sites (9/14 samples from 4 sites), but not the non-polluted forest control.

The levels of *Campylobacter* were highest in waste water samples from beef and sheep processing plants (40 to 10^5 per 100 ml and 10^4 to 10^5 per 100 ml respectively). Levels were lower in sewage (8 to 10^2 per 100 ml).

Most animal-sourced *C. jejuni* were identified as Penner serotypes HS:1 and HS:4.

6.2 Risk Assessment for the Freshwater Microbiology Research Programme (McBride *et al.*, 2002)

As part of the development of guidelines for the management of the microbiological quality of freshwater, a research programme was funded by the Ministry of Research Science and Technology in 1997. Ten indicators and pathogens were examined at 25 freshwater sites throughout New Zealand. These represented five different categories of predominant environmental impact: birds, dairy farming, forestry/undeveloped, municipal and sheep/pastoral. In addition five of the sites were source waters for treated drinking water supplies.

The main study measured the ten variables fortnightly at the 25 sites for 15 months, from December 1998 to February 2000. The results were utilised for a risk assessment, the main outcomes of which were:

- Of the pathogens assessed in this study, *Campylobacter* and human adenoviruses are the pathogens most likely to cause human waterborne illness to recreational freshwater users (e.g. swimmers, water skiers, wind surfers);
- Using data from all sites, an estimated 4% of notified *Campylobacter* infection in New Zealand could be attributable to water contact recreation;
- The critical value for *E. coli* as an indicator of increased *Campylobacter* infection is in the range of 200-500 *E. coli* per 100 mL;
- Infection risks of other pathogens examined have not been able to be related to *E. coli* concentrations in fresh waters.

Other findings were that the *Campylobacter* detection rate was 60%, and 5% of all samples were above the test's detection limit (i.e. >110 cfu per 100 ml). The levels of *Campylobacter* were highest in late summer-early autumn (April). *C. jejuni* was the most frequent thermotolerant species identified, being present in at least 48% of the positive samples. The overall pattern of distribution of *Campylobacter* species was similar between catchment

types, except that the sheep catchments contained elevated levels of *C. lari* (33% of positive samples, versus 14% for all other catchments).

In the study, a choice was made to assess the risk of infection rather than illness. This meant that data from many epidemiological and outbreak studies could not be used, but allowed the use of dose response data which assess both infection and illness. This choice was made on the basis that infected people without symptoms may still contribute infected faecal wastes into the system. However, using some assumptions about infection versus illness rates, comparisons were able to be made between computed infection rates and illness rates, and hence to published figures on notifiable campylobacteriosis data.

Routes of infection considered were ingestion and inhalation. Input data were obtained for:

- The duration of a swimming event (regarded as a betaPERT distribution with a minimum and maximum duration of 0.25 and 2 hours, with a mode of 0.5 hours)
- The volume of water ingested or inhaled per hour (regarded as a betaPERT distribution with minimum and maximum volumes of 10 and 100 ml, with a mode of 50 ml)
- The microorganism concentration (derived from the sampling and testing data).

The end result of these analyses was a set of risks for two target populations:

- Individuals using a particular recreational site;
- The population at large using a multitude of sites.

Three infections were modelled (campylobacteriosis, adenovirus, enterovirus). From surveys of recreational water use it was estimated that about 250,000 people go for at least one swim at a freshwater site each year, and most immerse their head while swimming. The risk analysis determined that the median or mean campylobacteriosis infection rate spread over all recreational sites was approximately 0.04 (i.e. mean 40/1000 people, standard deviation 102, based on contamination levels at sites, volumes ingested, and dose-response). Therefore the typical number of infections per annum equates to 0.04 x 250,000 = 10,000.

For a New Zealand population of 4 million, this water recreation use infection rate is 250 per 100,000 persons per annum. It was assumed that the notified illness rate reflected 13% of the actual illness rate, giving a summertime illness rate of approximately 3,000 cases per 100,000 population (based on a notification rate of approximately 400 per 100,000 during summer months i.e. a "multiplier" of 7.6 from a UK study). The infection rate was also assumed to be double the actual rate (or illness rate). Thus it was estimated that the campylobacteriosis infection rate in New Zealand was 6,000 per 100,000 persons per annum. Therefore the median proportion of campylobacteriosis that is attributable to freshwater contact recreation is 250/6,000, i.e. approximately 4%.

This analysis relies on a number of assumptions and has a large standard deviation, so the actual value should be treated as indicative only.

6.3 Pathogen Transmission Routes: Cross-Departmental Research

In a follow-up to the research above, a three year cross departmental research project was initiated in July 2002 to further examine transmission routes of pathogens from domestic farmed animals (dairy cattle, beef cattle, sheep) into water bodies (streams, lakes, dams) and to identify and assess any mitigation strategies. The project involves a number of agencies working on different parts of the process:

- AgResearch and NIWA: the effects of riparian barriers around waterways on the movement of animal faeces and pathogens (*Campylobacter*);
- ESR: the movement of *Campylobacter* from pastures into groundwater;
- Massey University: movement of microorganisms (*Campylobacter*, *Giardia*, *Cryptosporidium*) through tile drains;
- AgResearch: animal behaviour with respect to drinking water sources;
- Landcare: geographic analysis of New Zealand farming systems to locate the risk factors identified in other work.

A report from ESR to MAF Policy for the first year of the groundwater section (Close and Savill, 2003) summarised results from groundwater wells in a dairying catchment in South Canterbury characterised by border strip irrigation. Positive *Campylobacter* results were obtained from 6 of 42 samples (14%). The detection of *Campylobacter*, albeit generally at low levels (<0.6 MPN/L), and the selection of the wells to avoid septic tank contamination meant that the contamination was most likely derived from dairying combined with border strip irrigation.

6.4 Recreational and Drinking Waters (Savill *et al.*, 2001b).

A combined approach using polymerase chain reaction detection with most probable number (MPN) enumeration was used to study *Campylobacter* in water samples from rivers, drinking water, roof water and shallow ground water. Water samples from five rivers, reticulated drinking water in four towns and ground water from three bores were collected monthly for six months. In addition 24 different roof water samples were collected over the same period at a rate of four samples per month from a site in the North Island with little reticulated water.

Results were:

- Surface water, 60% positive, median 0.18 MPN/100ml (range <0.12->11 per 100 ml)
- Shallow groundwater 75% positive, median 0.12 MPN/100 ml (range <0.06-0.72 per 100 ml)
- Roof water 37.5% positive, median <0.06 MPN/100 ml (range <0.06-0.56 per 100 ml)
- Reticulated drinking water 29.2% positive, median <0.07 MPN/100 ml (range <0.06-<0.07 per 100 ml).

These data referred to the total prevalence of thermotolerant *Campylobacter* species. The prevalence of *C. jejuni* and *C. coli* in the samples were approximately:

- Surface water, *C jejuni* 35% positive, *C. coli* 35% positive

- Shallow groundwater, *C jejuni* 12% positive, *C. coli* 30% positive
- Roof water, *C jejuni* 0% positive, *C. coli* 12% positive
- Reticulated drinking water, *C jejuni* 4% positive, *C. coli* 8% positive

The degree of risk represented by these sources of *Campylobacter* will depend on the amount consumed, frequency of consumption, and the population exposed.

6.5 Drinking Waters

Following the surveys which found *Campylobacter* in river waters (McBride *et al.*, 2002) and some treated drinking water supplies (Savill *et al.*, 2001b), a larger survey of treated drinking waters was commenced (Nokes *et al.*, 2004). A total of 31 treated drinking water supplies drawing directly or indirectly from surface waters (23 chlorinated and 8 treated by ultraviolet light) were sampled for approximately 12 months from January 2003. About half the drinking water supplies were chosen because of the likelihood that they would contain *Campylobacter*, while the remainder were randomly chosen. Twelve samples were collected after the treatment plants over the survey period from most supplies leading to a total of 363 samples from these locations, and six samples were taken from the distribution zones providing a total of 186 samples from the reticulation networks. Samples were also taken from source waters (pre-treatment) for 27 of the 31 supplies, for the final six months of the survey.

The water samples were analysed using a two phase process employing filtration, culture enrichment, and polymerase chain reaction detection of viable cells. *Camplylobacter* were detected in two samples taken after the treatment plant, and none from the distribution zone. Both of the positive samples were from the same supply: a small camping ground supply using UV disinfection (possibly ineffective). In contrast, *Campylobacter* were detected in 23 (16%) of the 146 source water samples, representing 11 of the 27 source waters sampled.

ESR also conducts analyses of drinking water supplies on an *ad hoc* basis, most commonly as part of an investigation of cases of illness. Some of these are from supplies derived from roof water. From 2001-March 2004 ESR tested 32 roof water supplies for *Campylobacter*, and one (3%) was positive (Andrew Ball, ESR, pers. comm).

A study in four rural Auckland districts from 1996 to 1998 examined 125 domestic roof-collected rainwater supplies (Simmons *et al.*, 2001). *Campylobacter* spp. were not detected, which was considered a surprising result, in view of the potential for wild bird contamination.

The differing results observed in the studies in this section may be partly due to differences in methods applied, their sensitivity and reproducibility.

6.6 Taieri River (Eyles *et al.*, 2003)

A study of the spatial and temporal distribution of Campylobacter in the lower Taieri River was conducted at 10 sites from June 2000- June 2001 at fortnightly intervals. MPN enumeration of *Campylobacter* was used for detection (positive scoring was by plating out and microscopic confirmation). The percentage of samples positive for presumptive

Campylobacter was generally high although only a small proportion contained >11 MPN/100 ml:

Winter: 91% positive, 8% >11MPN/100ml
Spring: 85% positive, 0% >11MPN/100ml
Summer: 84% positive, 13% >11MPN/100ml
Autumn: 55% positive, 7% >11MPN/100ml

Slightly higher numbers of *Campylobacter* were observed in summer than in winter, which was unexpected given the general opinion that lower water temperature and UV levels in winter permit greater *Campylobacter* survival. It was suggested that such factors as higher stock levels, greater likelihood of stock entering the water, and minor flood events may have been responsible.

Comparison of *Campylobacter* levels at the sampling site most likely to be used for recreational bathing, and notified cases of campylobacteriosis from the region showed some correspondence although firm conclusions could not be drawn. On the majority (93%) of occasions positive water samples showed *Campylobacter* concentrations of <11 MPN/100 ml which were considered unlikely to present a public health risk. The remaining samples were >11 MPN/100ml.

6.7 Rural Streams (Donnison and Ross, 2003)

A study of two rural streams in the Waikato region of New Zealand took fortnightly samples from February 2001 to January 2002. Each stream was surrounded by dairy farms. *Campylobacter* was recovered from almost all samples (19 of 21 from one stream, 21 of 22 from the other) at median concentrations of 2.3 MPN/100 ml and 4.3 MPN/100 ml. The range of *Campylobacter* levels for each stream was 0-210 MPN/100ml and 0-110 MPN/100ml. The high levels on one occasion meant that an infectious dose would be contained in no more than 500 ml of water. Although not explicitly stated in the reference given, a preliminary report on these results (Hudson *et al*., 2001) indicated that the high levels of *Campylobacter* occurred after a heavy rainfall event and flooding.

Although *Campylobacter* numbers were generally low, it was suggested that the constant presence of the bacterium in rural streams may lead to cycling in farmed animals drinking from streams, and indirectly contribute to human infection (it is suggested that *Campylobacter* contamination in streams may end up in shellfish, although the one survey of commercial shellfish did not find *Campylobacter* contamination).

6.8 Wastewater Treatment

A study of the Christchurch wastewater treatment plant examined the behaviour of a range of microorganisms, including *Campylobacter* between July and September (winter-spring) 2002 (Leonard *et al*., 2003). The numbers of *C. jejuni* in screened raw sewage influent had a median of >6980 MPN/100 ml (range >6980 to >69800, N=9), which declined ten fold by the stage of the secondary treatment (trickling filters, aerobic contact and clarification) and had declined further to a median of 0.45 MPN/100 ml (range <0.45 to 1.8, N=9) by the tertiary stage of oxidation ponds. *C. coli* numbers were much lower at all stages.

The high numbers of *C. jejuni* in the influent material was unexpected, but the overall >4 $\log_{10}$ reduction revealed the effectiveness of the treatment.

More recent data was provided in evidence for a hearing under the Resource Management Act for the Christchurch City Council (Dr M Leonard, pers. comm, September 2005).

Influent:

> Winter 2003: *C. jejuni* median 46,000 MPN/100ml (range: 9,000-110,000), thermotolerant *Campylobacter* 46,000 MPN/100 ml (range: 9300-110,000)

Oxidation pond discharge:

> Winter 2003: *C. jejuni* median 3 MPN/100ml (range 1.5-150), thermotolerant *Campylobacter* 6.5 MPN/100 ml (range 1.5-23)

Campylobacter spp. in Christchurch oxidation pond discharges in 2004 and summer 2005 were below the detection limit (3 MPN/100ml).

In contrast, analysis in Auckland of sewage at various stages of the treatment process has shown a very low level of *Campylobacter* contamination (Dr Andrew Ball, ESR Christchurch Science Centre, pers. comm.). A survey for Project Manukau found that thermophilic *Campylobacter* spp. were either absent or <10 per 1000 ml. The reason for the difference between Christchurch and Auckland is currently unknown.

A Cross-Departmental Research Project for the Ministry for the Environment (Waste Solutions Ltd, 2005) examined pathogens in sewage effluent from 9 wastewater treatment plants throughout New Zealand. Sampling took place from December 2003-May 2004. Three samples from each plant were taken over that period. Results for *Campylobacter* in both influent and effluent were uniformly low; mostly <2 MPN/100ml. The highest result was 41 MPN/100ml.

The reason for the difference between results from this project, and the influent results for Christchurch are currently unknown, but nevertheless it appears that sewage treatment from oxidation ponds in New Zealand are not discharging large numbers of *Campylobacter* into the environment.

6.9 Work in Progress Relevant to Transmission Routes: Exposure Model

A joint interagency programme on "Enhanced Co-ordination and Development of Enteric Zoonotic Disease Research in New Zealand" was initiated by the Ministry of Health in May 2000. The Programme's mission is to reduce the burden of enteric zoonoses in New Zealand. The programme is led by the Enteric Zoonotic Disease Research Steering Committee, who are responsible for:

- coordination of funding from private and public sector sources
- identification of research goals and prioritisation of research projects.

The Steering Committee is assisted and advised by two expert technical sub-committees: a methodology group and a risk management group.

The committees work to ensure that research on enteric zoonoses is both focused and well co-ordinated. The first research project undertaken was the construction of a conceptual *Campylobacter* risk model (by Graham McBride and colleagues at NIWA) to determine knowledge gaps.

In 2004 the Steering Committee established a Modelling Group to apply these techniques to zoonotic issues and human disease. Representatives from NIWA, ESR, the NZFSA and Massey University are included. The first output from this group has been a comparative exposure model (McBride *et al.*, 2005). This model collated data to provide comparative estimates of the risk of infection from four sources:

- food (poultry, red meat) either from consumption or cross contamination;
- potable water;
- recreational water (freshwater swimming); and,
- occupational animal contact.

The tentative conclusions from this study are that cross-contamination from preparation or storage of poultry, and to a lesser extent red meat, were the most important exposures. The least important was drinking water that had been properly treated. Occupational contact with livestock was also identified as important, though very little information exists to verify this result. Sensitivity studies showed that the input parameters to which the model was most sensitive were the probabilities that poultry or red meat were contaminated.

7 INFORMATION FROM OVERSEAS ON TRANSMISSION ROUTES

This section provides contextual information from overseas which is intended to help assess the data from New Zealand. The focus has largely been on case-control studies, and contamination rates in food and water. Most information is drawn from developed countries more likely to be relevant to New Zealand.

The section has been organised according to country of origin, rather than transmission route. As much of the information derives from case control studies it was thought more appropriate to keep such information together by country.

7.1 Country by Country Overview

7.1.1 Iceland

Icelanders experience campylobacteriosis at a rate similar to other developed countries, while having a limited size (population approximately 280,000), a self contained and well characterised poultry industry and an intensive human disease surveillance system. Consequently it has been the subject of a joint Icelandic-Canadian-US investigation to create a risk assessment model, which will have spin-offs for other countries (Stern *et al*., 2003).

The study examined:

- Prevalence of *Campylobacter* contamination in flocks from 5 major and 4 minor farms, which varied from 0 (1 major farm and the group of 4 minor farms), 42% (one major farm) to 100% (3 major farms);
- Frequency of *Campylobacter* contamination on processed carcasses from farms 1 (100% flock prevalence) and 2 (42% flock prevalence), the frequency being generally near 100% for farm 1, and variable (0-100%) for farm 2;
- Human case rates, first calculated at 13.8 per 100,000 population in 1997, increased to 52 per 100,000 in 1998 and 116 per 100,000 in 1999 and then declined to 33 per 100,000 in 2000. Actual case numbers were relatively small: 38, 143, 326, and 92 for the years 1997 – 2000 inclusive;
- Foreign travel as a risk factor: in 1999 the ratio was 4.0 domestically acquired cases to 1.0 foreign case, while in 2000 the ratio dropped to 0.7 domestic to 1.0 foreign case;
- Qualitative and quantitative data related to human exposures including levels of *Campylobacter* on carcasses in 1999 and 2000 that varied between 3.3 and 5 $\log_{10}$ cfu/carcass.

A fundamental change in food consumption was contemporaneous with the observed epidemic of campylobacteriosis between 1997 and 2000. Prior to 1996 only frozen poultry had been marketed in Iceland. After 1996 there was an increasing demand for chilled poultry, which would increase human exposure to higher numbers of *Campylobacter* cells as refrigeration is less effective in reducing numbers (Bhaduri and Cottrell, 2004)..

The paper also reports that poultry consumption in Iceland, although increasing, is only approximately one third of US consumption. The basis for this comparison is not given.

Various control measures were also introduced in 2000 including public education, increased on-farm biological security measures, and the freezing of process lots of broiler carcasses which came from flocks testing positive for *Campylobacter* (flocks testing positive 1 week prior to slaughter and from the subsequent two flocks raised in the same broiler house). The lower prices for frozen poultry reinforced the emphasis on on-farm measures. It was not considered possible to assign credit to which of the interventions were most influential, but there was a dramatic decrease in the public health burden of campylobacteriosis.

7.1.2 Denmark

In Denmark reported rates of campylobacteriosis remained relatively constant from 1980 to 1992 at approximately 20-30 cases per 100,000, but have increased nearly threefold from 1992 to 2002 (Anonymous, 2003a).

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

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

A quantitative risk model to investigate campylobacteriosis associated with poultry has been developed (Rosenquist *et al*., 2003). Simulations designed to predict the effect of different mitigation strategies showed that the incidence of campylobacteriosis associated with the consumption of chicken meals could be reduced 30 times by introducing a 2 log reduction of the number of *Campylobacter* on chicken carcasses. To achieve a similar reduction in incidence, the flock prevalence should be reduced approximately 30 times or the kitchen hygiene improved approximately 30 times. Cross contamination from positive to negative flocks during slaughter had almost no effect, which suggested that logistic slaughter (i.e. slaughtering negative flocks before positive flocks) would have only a minor influence on risk.

A case control study was conducted in Denmark during 1996-1997 involving 282 cases and 319 controls (Neiman *et al*., 2003). Two estimates of the odds ratios were determined for each risk factor, with or without "protective factors" fitted into the final model. Consumption of undercooked poultry, consumption of red meat at a barbeque, consumption of grapes, and drinking unpasteurised milk were identified as risk factors in both models. Frequent consumption of pork chops and daily contact with domestic animals and pets were identified as risk factors in one of two models only. Foreign travel was also a significant risk factor.

Poultry exposures were investigated in detail however, and poultry in general was not a significant risk factor. The report also stated that the level of contamination of poultry products at retail in Denmark was 10-30%.

Another case control study, in 2000-2001, involved 107 acute sporadic cases and 178 controls (Wingstrand *et al.*, 2005). Travel to Southern Europe (OR 15.81, 95% CI 2.63, 94.93) and outside Europe (16/1207 cases exposed versus 1/178 controls) were identified as strong risk factors. Analysis of a subset of 74 domestic cases and 114 matched controls identified consumption of fresh, as opposed to frozen chicken to be the only significant risk factor for human campylobacteriosis (OR 5.80, 95% CI 2.11, 15.93).

A study of the serotypes of *C. jejuni* found in Danish human cases, chicken and cattle was conducted in 1995-1996 (Nielsen *et al*., 1997). The predominant types found from all three sources were HS:1,44, HS:2 and HS:4 complex.

A study of faecal shedding of *Campylobacter* spp. by healthy puppies (n=72) and kittens (n=42) in Denmark found that 29% of puppies and 5% of cats were positive (Hald and Madsen, 1997). The puppies carried *C. jejuni* predominantly (76%), with the remainder comprising *C. coli* (5%) and *C. upsaliensis* (19%). Each of the two *Campylobacter* positive cats carried *C. upsaliensis*.

7.1.3 Sweden

A case-control study in Western Sweden, involving 101 cases (Swedish residents, and so more likely to be domestically-acquired infections) and 198 controls found the usual collection of risk factors (Studhal and Andersson, 2000):

- Drinking unpasteurised milk (OR 3.56, 95% CI 1.46-8.94)
- Eating chicken (OR 2.29, 95% CI 1.29-4.23)
- Eating pork with bones (chops OR 2.02, 95% CI 1.17-3.64, loin of pork OR 1.83, 95% CI 1.07-3.120
- Barbecuing (OR 1.98, 95% CI 1.10-4.34)
- Living or working on a farm (farm OR 3.06, 95% CI 1.58-6.62, hen/chicken-breeder OR 3.32, 95% CI 1.56-6.78)
- Daily contact with chickens or hens (OR 11.83, 95% CI 3.41-62.03).

The study reported that in 1995, 5580 human cases of campylobacteriosis were reported, of which 2551 were caused by indigenous sources. The other cases were infected abroad (mostly tourists).

Sweden has instituted various programmes on farms, and succeeded in reducing the proportion of *Campylobacter*-infected chickens to a level of 10-15%. Despite this, the number of campylobacteriosis cases has actually increased. The study did not find any reason why this might be the case, although the high proportion of cases infected abroad might make it difficult for domestic poultry risk management to affect the overall rate.

An analysis of geographical environmental risk factors and *Campylobacter* infections across Sweden found a positive association between (domestically-acquired) campylobacteriosis incidence and average water pipe length per person and ruminant density, and a negative

association with the percentage of the population receiving water from a public water supply (Nygard *et al.*, 2004). The results suggested an association between living in an area with a high ruminant density and campylobacter infection, although having a public (rather than private) water supply conferred protection.

This study also reported that of the 23,481 campylobacteriosis notifications from 1998-2000, 13715 (58%) were reported as acquired abroad. This gave an overall annual rate of domestic infection of 26.3 per 100,000 population.

7.1.4 Norway

An epidemiological investigation of risk factors for *Campylobacter* colonisation in Norwegian broiler flocks found that 18% of flocks were infected, and the preventive measure most likely to reduce the prevalence was disinfection of the broiler drinking water (Kapperud *et al*., 1993).

A case-control study in 1989-1990 (Kapperud *et al*., 1992) of 52 cases and 103 controls found the following risk factors for campylobacteriosis in Norway:

- Consumption of sausages at a barbeque (OR 7.64)
- Daily contact with a dog (OR 4.26)
- Eating poultry that was brought into the house raw (frozen or refrigerated) (OR 3.20).

These risk factors were believed to be relevant to domestically acquired cases of infection, as cases who had traveled abroad in the 2 weeks prior to illness were excluded.

When poultry consumption was examined by country of origin, eating of poultry produced in Denmark or Sweden was strongly associated with illness (OR 13.66) whereas consumption of poultry produced in Norway was less so (OR 1.33). The study reported that there was a comparatively low prevalence of *Campylobacter* in Norwegian broiler chicken flocks.

The majority of retail poultry in Norway is sold frozen. Normally frozen poultry is regarded as being of lower risk, or even protective, and the study authors suggested that the result may be due to the survival of low numbers of *Campylobacter*, even under frozen conditions.

An action plan against *Campylobacter* spp. in Norwegian broilers was implemented in May 2001 (Hofshagen and Kruse, 2005). This included surveillance of flocks at slaughter, advisory services for farms delivering positive flocks, and surveys at the retail level. Between 2002 and 2004 there was a reduction in flock prevalence from 6.3% to 3.3%, and a reduction in retail product prevalence from 8% to 5%. Prevalence of *Campylobacter* in both flocks and retail products peaked in summer. It was difficult to evaluate the public health effect of these reductions, due to the multi-factorial nature of campylobacteriosis. Nevertheless, the authors considered that the action plan would have contributed to the approximately 25% reduction in campylobacteriosis cases between 2001 and 2003, as consumption of poultry meat purchased raw had been identified as an important risk factor in Norway.

7.1.5 Finland

A recent study in Finland identified *Campylobacter* isolates from human clinical faecal samples submitted to clinical microbiology laboratories (Vierikko *et al.*, 2004). Isolates were selected from cases who had not been abroad for two weeks before becoming ill; the intention of the study was to generate information on domestically-acquired campylobacteriosis. The number of domestic cases from the isolate collection was estimated as approximately 40% of the total number of cases notified to the National Infectious Diseases Register. This was equivalent to a rate of 41.2 per 100,000 population (presumably the overall rate was approximately 100 per 100,000).

The predominant serotypes (61% of the total isolates) were: HS: 1,44 (5%), HS: 2 (9%), HS: 4 cluster (11%), HS: 6,7 (16%), HS: 12 (13%), and HS: 27 (7%).

From May to September 1999, 1, 132 chicken flocks, representing most of the chicken meat produced and consumed in Finland, were monitored for campylobacters. Thirty one (2.7%) positive flocks were identified and the most common serotypes were HS6,7 and HS:12.

The authors considered that these results showed the possibility that humans and chickens may share a common source for *C. jejuni*, or humans may acquire the infection from contaminated chicken.

A large outbreak of campylobacteriosis occurred in Finland in 1998 (Kuusi et al., 2005). The estimated number of ill persons was 2700, and *C. jejuni* was isolated from stool samples of 45/74 (61%) of patients tested. Of the survey participants, 4% consulted a physician, 0.6% were hospitalised, and 8.4% were absent from work because of illness. Epidemiological and environmental evidence suggested water mains repair as the source of contamination (possibly from a nearby ditch from a cattle farm). The risk of illness was associated with drinking the non-chlorinated water supply, and the risk increased with increasing consumption. Although *Campylobacter* was not detected in the water supply, sampling only commenced several days after large numbers of patients began presenting to health centres.

7.1.6 Netherlands

Attempts to estimate the importance of water exposure in causing campylobacteriosis in the Netherlands have been reported (in Koenraad *et al*., 1997). A risk analysis carried out in the early 1990s suggested that 0.2-0.5% of all registered (i.e. notified) *Campylobacter* infections may be caused by contaminated unboiled drinking water. Another estimate gave the range of campylobacteriosis due to exposure to contaminated recreational waters as between 1.2 and 170 per 100,000. The comment was made that the attribution of water-borne campylobacteriosis cases is low, but may be higher than had been assumed up to that time.

In 2002 various science and regulatory agencies in the Netherlands initiated the Campylobacter Risk Management and Assessment (CARMA) project (see: http://www.rivm.nl/carma). The aim of the project was to advise the Dutch government on the effectiveness and efficacy of measures aimed at reducing campylobacteriosis in the Dutch population. The project includes risk assessment and economic analysis and research.

In the Netherlands the rate of campylobacteriosis is approximately 110 per 100,000 (based on 18,000 GP visits in a population of 16 million, although only 6,000 cases are notified). A decline (approximately 25%) in this rate was observed in 2003 and was attributed to a decline in poultry consumption due to concern about avian influenza (A. Havelaar, RIVM, pers. comm,, 2005).

A preliminary estimation of the relative importance of *Campylobacter* transmission routes from the CARMA project has been presented (http://www.rivm.nl/carma/resultaten/CHRO%202003/CHRO%202003%20Evers.pdf). This considered three categories of transmission routes:

- Food: chicken, turkey, pig, cattle, milk, sheep, fish, shellfish, crustaceans, vegetables and fruit;
- Direct contact with animals: pets, farm animals, and city farm animals;
- Water: swimming in indoor pools, swimming in recreational water, consumption of drinking water

Exposure was assessed by calculating the mean number of *Campylobacter* cells ingested per person per day. The mean number of campylobacters ingested per day was calculated as:

- Water: 0.0015 (only swimming in recreational water was significant)
- Food 0.048 (principally from raw milk and raw chicken)
- Direct contact: 0.08 (derived mostly from exposure from city farm animals and farm animals)

The highest individual exposure was from city farm animals, followed by raw chicken and raw milk. Foreign travel was not evaluated in the study.

Information about city farms was found here: http://www.farmgarden.org.uk/About/what-are/content.html

"City farms and community gardens are community-managed projects working with people, animals and plants…… They exist mainly in urban areas and are created in response to a lack of access to green space, combined with a desire to encourage strong community relationships and an awareness of gardening and farming. …….Most projects provide food-growing activities, training courses, school visits, community allotments and community businesses. In addition, some provide play facilities and sports facilities, and after school and holiday schemes."

In 1999 broiler flocks in the Netherlands were 29.8% positive for *Campylobacter*. A survey of retail poultry found that contamination levels varied from <10 (18%) to more than 5,500 (18%) per fresh carcass. In contrast, most frozen samples (57%) contained <10 MPN *Campylobacter* per carcass (Dufrenne *et al.*, 2001).

Based on a case control study in 2003, 23% of domestically-acquired cases of campylobacteriosis were attributed to chicken meat. This was assigned as the lower bound of the estimated attribution range, with the upper bound of 40% derived from the Belgian study (see Section 7.2.12) (A. Havelaar, RIVM, pers. comm., 2005).

A risk assessment of *Campylobacter* in shellfish in the Netherlands found that the majority of isolates from this source was *C. lari*, presumably from contamination by gulls (Teunis *et al.*, 1997).

7.1.7 France

A survey of *Campylobacter* in French chicken production used both PCR and culture methods (Denis *et al*., 2001). Of the supermarket samples (all portions), 75% (53/70) were *Campylobacter* positive.

7.1.8 USA

7.1.8.1 Rates

An overview of campylobacteriosis in the USA during the 1980s was published in 1992 (Tauxe, 1992). In this report, various small scale studies were used to estimate the actual rate of infection. It was estimated that if all clinical laboratories tested for *Campylobacter* spp. in faecal specimens at the same rate as they tested for *Salmonella* and *Shigella*, then the reported rate would be approximately 40 per100,000 population. If all physicians requested stool sample testing for patients presenting with symptoms, then the rate was estimated to be 54-60/100,000. The ratio of reported to unreported cases was estimated as 18:1.

In 1995 active surveillance of enteric infections began in the US, in the form of FoodNet, which initially covered five states, and then seven states from 1998 (Friedman *et al*., 2000b). Rates of reported campylobacteriosis from this network were 20-25/100,000 from 1996-1998. That there had been a drop in rates from the 1980s was supported by the repeat of one of the studies used above to determine the frequency of isolation of *Campylobacter*, *Salmonella* and *Shigella* from clinical stool samples. The ratio of *Campylobacter* isolations to those of the other two bacteria had declined between the study in 1980 and the repeat in 1992, suggesting that the decline in the incidence of reported illness was real.

More recently in the United States, the rate of campylobacteriosis, as determined by the FoodNet surveillance network declined, from a peak of 25.2 per 100,000 in 1997 to 17.3 per 100,000 in 1999. This decline is not explained by changes in culturing or reporting practices, and may represent a true reduction in risk (Tauxe, 2000). The author stated that the decline has occurred at the same time as substantial improvements in the disinfection of water used in chill tanks in the poultry industry, and other slaughter sanitation improvements.

Preliminary data for 2004 indicate that the campylobacteriosis rate in areas covered by FoodNet sites has fallen further, to 12.9 per 100,000 (see: http://www.cdc.gov/mmwr/preview/mmwrhtml/mm5414a2.htm). There is a strong regional pattern to the incidence of campylobacteriosis in the US, with westernmost states having higher rates (28.6 per 100,000 in California).

7.1.8.2 Epidemiology and case control studies

The epidemiology of outbreaks and sporadic cases in the USA during the 1980s has been described as quite different (Tauxe, 1992). In outbreaks between 1978 and 1987 where a vehicle was identified, the predominant vehicles were water supplies and raw milk (56% of

outbreaks). In contrast, for sporadic illnesses, which represent the bulk of actual cases, the predominant vehicle was poultry. This conclusion was based on various case control studies of sporadic cases, particularly a large study in Seattle. This study found that eating poultry (including chicken, turkey, and game hens) accounted for 50% of cases, while 5% were attributed to raw milk, 6% to contact with pets, 9% to overseas travel, and 8% to contaminated surface water. This may be assumed to represent urban risk factors.

Seasonality (summer peak), age distribution (peaks in those up to 4 years of age, and from 20-29 years of age), and gender distribution (predominance of males) were the distinguishing features of reported cases in the USA (Tauxe, 1992), as for New Zealand.

An update of the outbreak data, to 1996 (Friedman *et al*., 2000a) showed a change in the pattern of identified vehicles. Between 1988 and 1996 "other foods" (poultry, meats, eggs, fruit etc) comprised the vehicle in 83% of the identified outbreaks. The change was attributed to better control of groundwater, and raised awareness about the risk of raw milk consumption (particularly during school trips). It was suggested that the range of foods identified in outbreaks during the 1990s were those with the potential for cross contamination, particularly in the home.

A case-control study was conducted from 1998-1999 in one of the FoodNet sites, Maryland, to investigate the unusually low rate of campylobacteriosis (6.72 per 100,000 in 2002). The study found that cases were more likely than controls to be white, to have recently eaten in a restaurant, traveled internationally, eaten chicken luncheon meat or ham, had contact with a puppy, dog or cat or visited a petting zoo. Cases were less likely than controls to have purchased, stored or cooked raw chicken (see: http://www.cdc.gov/foodnet/pub/iceid/2002/Klatka_l.htm). The study authors suggested that exposure to contaminated chicken may be lower in the state.

A case-control study was conducted in the US from 1998-1999 using patients with culture confirmed *Campylobacter* infections from FoodNet sites (Friedman *et al*., 2000b). A total of 1463 patients and 1317 controls was involved. Foreign travel was strongly associated with illness (OR 10.4, 95% CI 6.2-17.4). Among persons with no foreign travel the following exposures were significant risk factors for infection:

Eating undercooked poultry (OR 1.9, 95% CI 1.3-2.9)
Eating chicken or turkey that was cooked outside the home (OR 2.4 95% CI 2.0-2.9)
Eating non-poultry meat that was cooked outside the home (OR 2.2, 95% CI 1.1-2.7)
Eating raw seafood (OR 1.8, 95% CI 1.1-2.7)
Drinking raw milk (OR 3.5, 95% CI 1.4-8.7)
Living on or visiting a farm (OR 2.1, 95% CI 1.6-2.8)
Having contact with farm animals (OR 2.2, 95% CI 1.5-3.1)
Having contact with puppies (OR 2.0, 95% CI 1.5-2.80

Eating chicken or turkey cooked in the home was a protective factor (OR 0.5, 95% CI 0.4-0.6).

The identification of raw seafood as a risk factor in this investigation was new and recommended for further investigation.

Hawaii has the highest rate of reported campylobacteriosis infections in the US: 69/100,000 in 1997. A case-control study in 1998 involving 211 cases and the same number of controls found that that the strongest predictors of infection were consuming chicken from a restaurant in the previous 7 days (OR 1.8, 95% CI 1.1-2.9) and consumption of antibiotics in the previous 28 days (OR 3.3, 95% CI 1.1-9.6) (Effler *et al*., 2001). Other exposures explored were limited but did include other types of meat, unpasteurised milk, untreated water, and contact with animals (including puppies). The association with antibiotic use was unusual, although such use had been identified previously as a risk factor for *Salmonella* infection.

The association between consumption of poultry outside the home and campylobacteriosis was supported by a re-analysis of the two case-control studies cited above (Cox, 2002). This paper asserted that the available data supported the hypothesis that poor hygiene in some restaurants may be a predominant cause of campylobacteriosis in humans. The paper attributed the risk of sporadic campylobacteriosis primarily to commercial cooking of hamburgers, chicken and other meats. The paper recommends focusing risk management efforts on restaurants rather than chicken.

Food handlers in domestic kitchens will be exposed to *Campylobacter* from contaminated poultry during meal preparation. A strong association between preparing or handling chicken, but not chicken consumption, has been demonstrated in a small case-control study among ten cases and fourteen controls from domestic consumers in Colorado (Hopkins and Scott, 1983). Of the cases, 9 had prepared or handled raw chicken, whereas only one of the controls had done the same.

7.1.8.3 Pets/animal exposure

Campylobacteriosis cases (218) were compared to uninfected controls (526) with respect to animal contacts in a study in the state of Washington in 1982-1983 (Saeed *et al*., 1993). There was no increase in risk of enteritis associated with contact with various animals (dogs, cats, birds, reptiles, cattle, sheep, pigs, goats, fish, rodents). However, direct contact with a diarrheoic animal during the week prior to onset was identified as a risk factor (OR 4.3, 95% CI 1.9-9.7), and the highest risk was associated with dogs and puppies rather than cats. The proportion of cases in the population attributable to exposure to diarrhoeic animals was 6.3%.

Other risk factors identified by the study were: chicken consumption (OR 2.4, 95% CI 1.6-3.6), non-household member with enteritis (OR 2.5, 955 CI 1.6-4.0), travel to under-developed countries (OR 32.9, 95% CI 10.2-133.6), household member with enteritis (OR 1.9, 95% CI 1.2-3.0), non-home well or surface water (OR 1.8, 95% CI 1.1-2.9), any animal with diarrhoea (OR 4.3, 955 CI 1.9-9.7), and raw milk consumption (OR 4.6, 95% CI 2.1-10.4).

A study in Tennessee examined the potential exposure to zoonotic enteric pathogens from cats and dogs (Ahmed *et al*., 2004). *Campylobacter* was found in the faeces of only 1% of dogs with acute or chronic diarrhoea and no cats.

A further study, of carriage rates in animals and companion animals in Arizona (Lee et al., 2004), isolated *C. jejuni* from the faeces of a variety of animals, and beef carcasses. The prevalence results were:

- 13.8% of dogs;
- 5% of goats;
- 28.3% of dairy cattle;
- 0% of range cattle;
- 73.5% of feedlot cattle; and,
- 94.7% of beef carcasses (presumably from feedlot cattle).

This supports the higher risk of faecal carriage of *C. jejuni* in feedlot cattle, as has also been found in Australia.

7.1.8.4 Risk factors amongst students

A case control study among students at the University of Georgia interviewed 45 case-control pairs in 1983-1984 (Deming *et al*., 1987). Three risk factors were identified: eating fully cooked chicken (OR 4.7 95% CI 1.3-16.2), eating chicken reported to be raw or undercooked (OR 9.0, 95% CI 1.1-71.0), and contact with a cat or kitten (OR 9.0. 95% CI 1.1-71.0).

7.1.8.5 Campylobacter levels in poultry

Per capita consumption of poultry by US citizens is apparently the highest in the world, apart from Hong Kong, according to data assembled by the USDA (http://www.fas.usda.gov/dlp2/circular/2000/00-10lp/poultpcc.pdf). However, consumption patterns may differ to New Zealand; it has been reported that in the US over 40% of chicken is eaten away from home in restaurants and fast-food outlets (Rogers, 2002) compared to 35% in New Zealand.

Data on the prevalence of *Campylobacter* on retail poultry and other meats in the US has been summarised (Jacobs-Reitsma, 2000). Contamination rates were highly variable; *Campylobacter* was found on 28-98% of retail chicken samples in surveys conducted between 1990 and 1997. Contamination levels for other meats were lower: beef carcasses 0.9%, and pork carcasses 2.9%.

A petition to support the development of a microbiological standard for *Campylobacter* in poultry has been prepared by the US Centre for Science in the Public Interest (see: http://www.cspinet.org/new/fs_petition2-14-02.pdf). That document reviews the prevalence of *Campylobacter* contamination in US poultry carcasses from a number of sources published in the scientific literature and US government reports. Data from studies conducted from 1998-2000 show contamination levels of 70-90% in retail chicken products. The studies included a USDA FSIS monitoring programme.

It has been stated that for the United States the number of *Campylobacter* per processed carcass has been dramatically reduced in the years up to 2003 (Stern *et al*., 2003).

Commercial flocks of broiler chickens were tested for *Campylobacter* in both 1995 and 2001, in an investigation to determine whether the reduction in the frequency of campylobacteriosis in the US might have been due to a reduction in exposure from poultry (Stern and Robach, 2003). Carcass samples were obtained after the chilling step. A significant reduction from an average of $10^{4.11}$ cfu per carcass in 1995 to an average of $10^{3.05}$ cfu per carcass in 2001

was observed. Although the broiler flocks were all from northern Georgia, it was hypothesised that this decrease may have been responsible for the drop in incidence of campylobacteriosis (this assumes that local production is supplied to local retail outlets).

Corresponding FoodNet data on campylobacteriosis is:

- USA FoodNet 1997 24.7 per 100,000
- Georgia FoodNet 1997 13.7 per 100,000

- USA FoodNet 2001 13.8 per 100,000
- Georgia Food Net 2001 7.4 per 100,000

Georgia is the top broiler producing state in the United States (http://www.ers.usda.gov/Briefing/Poultry/background.htm).

More recent data comes from a project called "Campy-Check" being undertaken by the USDA (results from a presentation by Dr Norman Stern, at a meeting organised by the Poultry Industry Association of New Zealand, Auckland, September 2005). This project is nationwide and includes post-chiller samples from 9 of the 10 major poultry producers in the US.

Overall *Campylobacter* counts Sept 2003 – Sept 2004 (n = 4,200)

Log 10 cfu/carcass	%
Not detected	74
<3	0.5
3.0 – 3.9	12.5
4.0 – 4.9	9.5
5.0 – 5.9	2.5
6.0 – 6.9	0.5
7.0 – 7.9	0.2

These results were described as a dramatic improvement from 1995 when 95% of carcass samples were positive with a mean count of $\log_{10}$ cfu/carcass of 4.2-4.3.

7.1.9 Australia

The national rate of notifications of campylobacteriosis in 2002 for Australia was 112/100,00, representing a 10% decline compared with 125/100,000 in 2001 (note that the most populous state, New South Wales, does not report campylobacteriosis so these data represent the remainder of the Australian population). The highest notification rate is among children aged 0-4 years, with a smaller peak in young adults 20-29 years old. Cases of infection of males were generally higher than in females across all age ranges, with the overall ratio being 1.2:1 (Yohannes *et al.*, 2004; OzFoodNet Working Group, 2005). Monthly notifications were consistent with previous years (1998-2002), with a peak in the third quarter of the year (spring-summer), although the peak was less pronounced in 1999 and 2000.

The decline did not continue, with the notification rate rising to 116.9 per 100,000 in 2003 (Miller et al., 2005) and 117 per 100,000 in 2004 (OzFoodNet Working Group, 2005).

A major prospective multi-centre case-control study of risk factors for sporadic *Campylobacter* infection across five states in Australia was conducted by the OzFoodNet Working Group over a twelve month period during 2001-2002 using a telephone-administered questionnaire. Results from this study were presented at the CHRO conference in Brisbane in September 2005 (Stafford et al., 2005). Risk of campylobacteriosis in cases 5 years and over was associated with cooked chicken (OR 1.4, 95%CI 1.0, 1.9) and strongly associated with recent consumption of undercooked chicken (OR 4.7, 95% CI 2.6, 8.4). There was also an increased risk with recent consumption of offal (OR 2.0, 95% CI 1.0, 4.0). The population attributable risk for eating undercooked chicken was 8.1% which equates to 14,500 cases, but it was estimated that a further 49,000 cases associated with cooked chicken could be prevented each year if the risks associated with the handling, preparation and cooking of raw chicken could be eliminated.

Overall, 237,000 (95% CI 77,000-396,000) cases of campylobacteriosis are estimated to occur in the Australian population over 5 years of age each year. A further 43,000 cases are estimated to occur in children below 5 years of age.

No foodborne risk factors were identified among cases aged 0-4 years.

Non-foodborne factors significantly associated with infection were having a household dog aged <6 months (OR 2.1), having backyard chickens aged <6 months (OR 12.4), overseas travel (OR 7.2), and persons having a chronic gastrointestinal condition (OR 2.3).

These risk factors are consistent with those found in New Zealand and most case-control studies from developed countries.

7.1.9.1 Children

A case-control study to investigate risk factors for children aged 0-35 months was conducted in Queensland in 1996-1997 (Tenkate and Stafford, 2001). Eighty-one culture-confirmed cases were compared with 144 controls. Risk factors independently associated with illness were: ownership of pet puppies (OR 16.58, 95% CI 3.73-73.65) and pet chickens (OR 11.80, CI 1.37-101.75), and consumption of mayonnaise (OR 4.13, 95% CI 1.61-10.59). A mechanism for the association with raw mayonnaise was difficult to identify.

7.1.9.2 Queensland

A review of the epidemiology of cases of campylobacteriosis in Queensland over the period 1991-1995 found that the highest notification rate was in children aged 12-23 months (Stafford *et al*., 1996). The age-adjusted annual rate for the five year period was 82.5 per 100,000, and the trend was declining, whereas for the whole of Australia over the same period, the rate increased. In Queensland, there was a markedly higher rate amongst urban dwellers, and those from higher socioeconomic status (possibly from economic and health access factors).

The seasonality of reported infections was variable and showed only a slightly higher incidence in summer months. However, the climatic conditions in Queensland will be different to New Zealand.

7.1.9.3 Pets

A study in South Australia (Adelaide) in 1997 looked at the *Campylobacter* species present in faecal samples from cats (n=195) and dogs (n=289), including stray and owned cats and dogs, and feral cats (Baker *et al*., 1999). Overall, *Campylobacter upsaliensis* and *C. jejuni* were isolated from 11% and 4% of cats respectively, whereas dogs carried *C. upsaliensis* (34%), *C. jejuni* (7%) and *C. coli* (2%). Intensive housing of the animals e.g. animal shelters, and access to open drains were found to be significant risk factors for animal infection. It was concluded that the bacteria did not cause enteritis in the animals. Although cats and dogs represent a potential reservoir of the bacteria, it was unclear whether the animals acquire infection from the same sources as people, or act as an infection source.

The following data come from an Australian study conducted in 2002 (Food Safety and Hygiene Bulletin September 2003). Nineteen commercial cattle and sheep properties in New South Wales and Queensland were selected: six dairy cattle properties, four feedlot beef cattle properties, four pasture beef cattle properties, two prime lamb properties and three mutton sheep properties. *Campylobacter* were found in all production systems and 14 of the 19 herds or flocks tested. The median prevalences were:

- Dairy cattle: 6%
- Feedlot beef cattle: 58%
- Pasture beef cattle: 2%
- Mutton sheep 0%
- Prime lambs 8%

7.1.9.4 Microbiological status of raw chilled chicken

A survey in 1999-2000 conducted by the Australian Capital Territory Government health portfolio provides some data on the prevalence of *Campylobacter* in raw chicken (see Food Survey Reports 1999-2000 under Publications at http://www.health.act.gov.au).

The overall isolation rate of thermophilic *Campylobacter* from raw chicken samples collected from ACT retail establishments was 20.6% (55/266). The prevalence in a similar survey in 1995-1996 was 12.3% (15/112).

A number of chicken isolates and isolates from human campylobacteriosis cases in the ACT was also tested for the presence of the *Cla1* gene, on the basis of a theory that this gene is highly conserved amongst chicken isolates (the report states that Professor Coloe and Dr Ben Fry of the Royal Melbourne Institute of Technology (RMIT), hypothesise that the *Cla1* gene is highly conserved in chicken *Campylobacter* strains and can be used as a stable genetic marker for epidemiological purposes). Approximately 90% of chicken-derived isolates (n=20) possessed this gene, as did 62% of isolates from human cases (n=81). These data were used to estimate that around 55% of human cases of campylobacteriosis could be attributed to bacteria derived from chicken.

In the absence of supporting evidence, this method of linking poultry with human cases must be considered as a hypothesis only.

7.1.10 United Kingdom

The number of faecal isolates of *Campylobacter* in England and Wales increased steadily from 1986 to 1998, following which there was a decline to a steady level of 54,000-56,000 from 1999 to 2001 (Health Protection Agency website). In 2003 49,050 reports of campylobacteriosis were recorded by the Public Health Laboratory Service, compared with a peak of 65,209 in 1998 (DEFRA, 2004). The reported rate in 1999 was 103.7 per 100,000 (Gillespie *et al*., 2002) while the reported rate for 1998 was 111 per 100,000 (Tam, 2001). The reporting peak was in late spring, and the reported rate was highest in males under 1 year of age with a secondary peak in adults aged 25-34.

As in the United States, the most commonly identified vehicles in campylobacteriosis outbreaks in England and Wales are water supplies and unpasteurised milk (Frost, 2001). However, the vast majority of *Campylobacter* infections are sporadic.

A study of the trends in indigenous (i.e. non-travel associated) foodborne disease in England and Wales (Adak *et al*., 2002) used data from the Infectious Intestinal Disease study to estimate that in 2000 approximately 550,000 cases of campylobacteriosis occurred (including 86 deaths). This represented a 45% increase from 1992 estimates, although overall infectious intestinal disease was considered to have declined by over half. Nearly 80% of these cases were considered to be foodborne.

A multi-centre case-control study of sporadic *Campylobacter* infections in 1990-1991 interviewed 598 cases and the same number of controls (Adak, 1995). Risk factors with statistically significant odds ratios were:

- Occupational exposure to raw meat: OR 9.37, 95% CI 2.03-43.3
- Having a household pet with diarrhoea: OR 2.39, 95% CI 1.09-5.25
- Ingesting untreated water from lakes, rivers and streams: OR 4.16, 95% CI 1.45-11.9

Consumption or cooking of chicken at home was generally not a statistically significant risk factor; in fact consumption/handling of chicken (fresh or frozen) cooked and eaten at home (with giblets) was protective. Consumption of other barbecued meats was not a risk factor. In addition, occupational contact with livestock or their faeces was associated with a significant decrease in the risk of infection.

That handling raw chicken at home was protective is surprising (although consistent with New Zealand and other case-control studies), and it has been suggested that people who handle raw chicken regularly may develop immunity, while newcomers are more likely to be infected (Corry and Atabay, 2001). Such a hypothesis has support from studies of young male college students whose high prevalence of *Campylobacter* infection has been attributed to their preparing chicken in the kitchen.

A case-control study in 1993-1994 was prompted by the excess of campylobacteriosis cases in Nottingham compared to the UK national average (Neal and Slack, 1995). A total of 282 laboratory-confirmed cases and 318 culture-negative controls were enrolled from the adult

population. Foreign travel was associated with 25% of cases. Eating chicken and handling raw poultry were the main risk factors for UK-acquired infections. Half the infections could be attributed to these risk factors. Contact with pets or other animals were not significant risk factors.

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

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

A sentinel surveillance scheme for *Campylobacter* was launched in England and Wales in May 2000. Twenty-two health authorities with a population of approximately 12 million people took part in the scheme which ended in April 2003. Publications from the scheme reported information on transmission routes.

A comparison of cases of *C. coli* infection with cases of *C. jejuni* infection was used to generate hypotheses for infection (Gillespie *et al*., 2002). Bottled water, pate and meat pie consumption, as well as foreign travel were more common (statistically significant) among cases of *C. coli* infection than *C. jejuni* cases. Conversely contact with animals was more common amongst cases of *C. jejuni* infection.

Another hypothesis generating study was conducted by postal interview of 7,360 cases of *Campylobacter* infection, whose food exposures were compared to data from the 1999 UK National Food Survey (Frost, 2002). Cases were more likely to report eating pre-packed sandwiches, pate, meat pies and offal than the national average, whereas poultry consumption amongst cases was the same as the national average.

An analysis of cases of infection with ciproflaxin-resistant *C. jejuni*, compared with infections of non-antibiotic resistant *C. jejuni*, showed that over half the *Campylobacter* infections acquired abroad were resistant. For the travel associated cases, infection was associated with travel to Spain (OR 6.87, 95% CI 3.52-13.38), Portugal (OR 22.40, 95% CI 4.36-114.99) or Cyprus (OR 11, 95% CI 1.28-1-8.02), the consumption of chicken (OR 4.95, 95% CI 2.12 – 11.56) or bottled water (OR 3.70, 95% CI 1.69-8.10) (Campylobacter Sentinel Surveillance Scheme Collaborators, 2002).

Outbreaks of campylobacteriosis are generally regarded as rare; however the sentinel surveillance scheme examined a large number of cases (3070) and found that many (509,

17%) reported another individual or individuals within the household with similar symptoms at the same time (Campylobacter Sentinel Surveillance Scheme Collaborators, 2003a). These cases were compared with cases of *C. jejuni* infection who did not report other illness in the home or community. An examination of exposures in the fortnight prior to illness found that cases were more likely to be schoolchildren or pre-school children and were more likely to be Asian. Illness in the home was associated with consuming organic meats in the winter, having contact with a pet suffering from diarrhoea or visiting a farm in the 2 weeks before the onset of symptoms. Illness in the community was associated with the consumption of foods in restaurants or drinking unpasteurised milk.

The sentinel surveillance team have also published a study specifically examining foreign and domestic travel as a risk factor (Campylobacter Sentinel Surveillance Scheme Collaborators, 2003b). Epidemiologic information was gained from 7360 cases of *Campylobacter* infection for the 2 weeks prior to illness, and recent foreign travel was reported by 20% of those cases. Domestic travel was reported by 13% of cases. The highest relative risk was associated with travel to: Africa (10.7), Bangladesh (20.0), Cyprus (5.9), India (18.8), Mexico (8.1), Middle East (5.4), Other Asia (Thailand, Nepal, Sri Lanka, Malaysia etc) (26.4), Other Pacific (Indonesia/Bali, Singapore, The Philippines) (591.8), Pakistan (39.2), Portugal (7.60, South and Central America (15.9), Spain (7.2).

The relative risk of travel to New Zealand (3.3) was similar to that for travel to Australia, the Caribbean, Central and Eastern Europe, Egypt, Greece, and other European countries. Relative risk for travel to the USA was 0.6.

In June 2003 the UK Food Standards Agency released a proposed strategy for the control of *Campylobacter* in chickens (see: http://www.foodstandards.gov.uk/news/newsarchive/campchickenstrategy).

The strategy notes that "there is strong evidence to suggest the most significant food source is chicken, and a reduction of the levels of *Campylobacter* on chicken would be expected to lead to a reduction in the number of human *Campylobacter* cases". The main focus of the strategy was action on farms, improving biosecurity, with measures in slaughterhouses, based on HACCP principles, to follow. Surveys by the UK FSA have shown that more than 50% of chickens on retail sale contain *Campylobacter*. The objective of the strategy is to reduce the prevalence of *Campylobacter* present in UK-produced chicken on retail sale significantly. The UK industry produces 800 million chickens per year.

7.1.10.1 Pets

A specific case-control investigation looked at the likelihood of puppy ownership being a risk factor for campylobacteriosis in children aged 0-5 years in Darlington UK, in 1984-1985 (Salfield and Pugh, 1987). A significant association (Chi squared 9.49, 0.01>p>0.001) was found.

Examination of the faeces of dogs and cats in several geographically distinct populations by a group at the University of Birmingham found that they harboured *C. upsaliensis* and *C. lari* far more frequently than *C. jejuni* (Jennings *et al.*, 2005). Overall the carriage rate was low.

7.1.10.2 Bottled water and salads

A retrospective cohort study in 2001 of patients submitting faecal samples in Cardiff compared 213 persons with *Campylobacter* infection and 1144 person with negative results (Evans *et al*., 2003). The study identified five risk factors for *Campylobacter* infection that, if taken together, could account for most sporadic cases. These were:

- Eating chicken (OR 1.79 95% CI 1.19-2.69)
- Eating salad vegetables other than lettuce (e.g. tomatoes, cucumber) (OR 1.53 95% CI 1.09-2.21)
- Drinking bottled water (OR 1.41 95% CI 1.02-3.42)
- Eating out at a fried chicken outlet (OR 1.94 95% CI 1.10-3.42)
- Contact with cows or calves (OR 5.07 95% CI 1.30-19.74)

The attributable proportions of infections associated with these risk factors were: chicken 31%, salad vegetables 21%, bottled water 12%, fried chicken outlet 4%, contact with cows or calves 1%.

It was suggested that salad vegetables were more likely to be a risk factor following cross-contamination in domestic or commercial kitchens (a large survey of organic fruit and vegetables and pre-prepared salads in the UK found no pathogens).

Drinking water as a source of exposure is difficult to assess; testing data for *Campylobacter* in bottled water sources are rare.

7.1.10.3 Retail foods contamination

A Public Health Laboratory Service (PHLS) survey in 1998 provided an indication of contamination in some retail meats in the UK. Chicken portions had the highest rate of contamination (83%), but were closely followed by lambs liver (73%), ox liver (54%) and pigs liver (72%) (Kramer *et al*., 2000). This study made the point that almost 30% of the meat samples yielded more than one strain of *Campylobacter*. Unpublished PHLS data were cited stating that co-infection with multiple strains of *Campylobacter* occurs in 5 to 10% of cases.

An extensive survey of 1400 butchery products and premises in the UK isolated *Campylobacter* spp. from 15 of 2330 raw meat products (Little and De Louvois, 1998).

Retail samples of 241 raw whole chickens were examined for *Campylobacter* and *Salmonella* spp. during the winters of 1998/1999 and 1999/2000 (Jørgensen *et al*., 2002). *Campylobacter* spp. were present on 83% of the chickens. On 56% of samples *Campylobacter* were present both on the meat and on the exterior of the packaging. The $\log_{10}$ cfu of *Campylobacter* spp. were 2.70-4.99 in 18% of samples and 5.00-6.99 in 20%. *Campylobacter* isolates (425) comprised *C. jejuni* (98%) and *C. coli* (2%) and 98 different sero/phage types.

In September and October 2002 the UK Health Protection Agency sampled 3662 raw meats for microbiological analysis. The external packaging of game fowl exhibited the highest

contamination from *Campylobacter* (3.6%), followed by chicken (3.0%), lamb (1.6%), turkey (0.8%), pork (0.2%) and beef (0.1%) (Little *et al.*, 2004).

A 2003 study by the UK Food Standards Authority of chicken available to the public in Wales found that 73% (536/736) of raw whole chickens were positive for *Campylobacter* (DEFRA, 2004). Another study in Wales in 2001-2002 found almost the same prevalence: 71% of retail whole raw chicken samples, with no difference between frozen and fresh samples (although numbers of bacteria may have been different (Meldrum *et al.*, 2004). A follow-up survey in 2003 found almost identical results: 73.1% positive for *Campylobacter*, and no difference between fresh and frozen samples (Meldrum *et al.*, 2005).

To examine the seasonality of human *Campylobacter* infection and isolates from poultry, a Welsh study compared data on human cases with isolation rates from fresh retail chicken samples between January and December 2002 (Meldrum *et al.*, 2005). Human isolates peaked in early June (weeks 22-25) whereas poultry isolates peaked in late June (between weeks 24 and 26). The authors postulated that the seasonal rise in humans is not caused by a rise in isolation rates in poultry, but that both are associated with an as yet unidentified source.

7.1.10.4 Ready-to-eat foods

A survey of 4,469 samples of ready-to-eat foods from retailers and restaurants in Wales during 2000-2001 found no *Campylobacter* (Meldrum and Ribiero, 2003). Foods sampled included cooked meats, cooked poultry, sandwiches, sauces, desserts and ice creams, pate, rice, pasta-based salads, fresh fruit, fresh herbs, and pizzas.

A low prevalence of contamination was also found in a UK survey of ready-to-eat cold sliced meats and pate from catering and retail premises in the UK (Elson *et al.*, 2004). A total of 4078 cold meat and pate samples. *Campylobacter* spp. were detected in only one sample, a cold meat.

7.1.10.5 Animals

A 2003-2004 survey of faeces/caecal contents from cattle, sheep and pigs intended for human consumption in Great Britain collected samples from 93 participating abattoirs. The presence of thermophilic *Campylobacter* species was 54.6% for cattle, 43.8% for sheep, and 69.3% for pigs (DEFRA, 2004).

7.1.11 Ireland

The following information is taken from the "Report on Zoonoses in Ireland 2000 and 2001" published by the Food Safety Authority of Ireland in March 2004 (http://www.fsai.ie/). Notification of campylobacteriosis was not required by law in Ireland in 2000 and 2001, but laboratory data on isolates from human cases of "Food poisoning (bacterial other than salmonella)" gave an estimated rate of 41 and 33 per 100,000 respectively, with the highest rates being in children under 5 years of age.

The percentage of poultry carcasses found to be harbouring *Campylobacter* at processing plants was 54% in both 2000 (1849/3422) and 2001 (1745/3214). The prevalence was lower at the retail level: 39% of raw poultry samples (152/391) were contaminated in 2000, along with 44% of offal (all meat types) samples. In 2001 the level of raw retail poultry contamination was 13% (19/151). The prevalence of contamination in other meat and food products was very low, with only poultry products (presumably further processed) showing contamination at 1% and 0.1% in 2000 and 2001 respectively.

A survey of broilers at the slaughterhouse in 2000 found that 55 out of the 65 flocks tested (85%) were positive for *Campylobacter*.

A survey undertaken to investigate antibiotic resistance amongst *Campylobacter* isolates from retail poultry in Ireland (Wilson, 2003) found that campylobacters could be isolated from 50% of local chickens and 28% of imported chickens.

7.1.12 Belgium

The Belgian dioxin crisis in 1999 inadvertently provided evidence of the importance of poultry as a transmission vehicle for *Campylobacter* (Vellinga and Van Loock, 2002). Following the discovery of dioxin contamination in livestock feed, on 28 May 1999 Belgian authorities ordered the withdrawal from sale of Belgian poultry and eggs. On 4 June 1999, the Belgian government issued a commerce embargo of meat products (pork and beef) with a minimum of 25% fat content. However, neither meat nor dairy products were withdrawn from sale.

Historical data from the Belgian sentinel surveillance system were used to model the expected number of *Campylobacter* cases for 1999. The actual number of *Campylobacter* infections reported during 1999 fit the model very well (within the 95% confidence interval) except during the four week period when poultry and eggs were removed from the shelves. During that period the number of reported cases of *Campylobacter* infection was 40% below that expected. After four weeks, the ban was lifted and campylobacteriosis notifications returned to the expected level. The effect was attributed to poultry, as eggs are not associated with *Campylobacter* contamination; *Campylobacter* is sensitive to drying on the surface of eggs, and egg albumin in the interior is toxic to *C. jejuni* so survival is unlikely. This has been confirmed by surveys (Wallace, 2003).

7.1.13 Canada

7.1.13.1 Walkerton

A large outbreak of *C. jejuni* and *E. coli* O157 infection occurred in the Canadian town of Walkerton in 2000 (Bruce-Grey-Owen Sound Health Unit, 2000; Clark *et al*., 2003). A total of 1,346 cases were reported, of which 174 stool samples had presumptive laboratory evidence of *E. coli* O157, and 116 samples contained *Campylobacter* spp. Overall 65 patients, were admitted to hospital, 27 developed HUS, and 6 died.

Investigations indicated that bacteria entered the town water supply from neighbouring farms, through contamination of well water by surface water carrying livestock waste immediately after heavy rains and flooding. Environmental testing of 13 surrounding

livestock farms discovered *Campylobacter* species in animal manure on 9, and both *Campylobacter* and *E. coli* O157:H7 on two. Two particular *Campylobacter* strains were identified, and the study reported the value of typing and subtyping methods in investigating the outbreak.

7.1.13.2 Case control study in Estrie, Quebec

A prospective case control study involving 142 cases and 281 controls was conducted in 2000-2001 in Estrie, where the rate of campylobacteriosis (55.5 per 100,000) was higher than the average for the Quebec province (36.2 per 100,000) (Michaud *et al*., 2004). This part of Canada appears to be largely rural, with small towns and villages. Eating undercooked poultry had the strongest OR (5.38) for infection but explained only 10% of the cases. Other contributors to infection were identified as: contaminated water (9%), animal contact (9%), raw milk (6%), beef (4%), other food (8%), travel (4%), infectious contact (1%), other risk factors (1%). The source of infection in 44% of the sporadic cases was not identified.

Chickens from 58 food stores in the region were also sampled and tested for *Campylobacter*. The prevalence of contamination was varied, remaining low (0-25%) from November to July 2001, but rising markedly in August-October 2001 (up to 69%). This change came after the peak in human infections (July 2001). The study commented that although chicken consumption is an important risk factor for campylobacteriosis, it does not explain either the seasonal or regional variations in the incidence of sporadic cases.

8 CAMPYLOBACTERIOSIS IN NEW ZEALAND: OVERVIEW OF TRANSMISSION ROUTES AND THEIR RELATIVE IMPORTANCE

Campylobacter are widespread throughout certain animals, some foods, water environments, and the possibility of cross-contamination within both the commercial and domestic kitchen makes the tracing of sources of *Campylobacter* difficult (Frost, 2001). As in other countries, the epidemiology of campylobacteriosis in New Zealand is poorly understood.

This report has examined the information regarding the transmission of *Campylobacter* infection in New Zealand from two directions: information from human cases, working back to risk factors and sources, and information about potential sources, working forward to human exposures. Inevitably, as the information is extrapolated, uncertainty increases.

The high probability of infection at relatively low doses of *Campylobacter* means that the level of contamination of a potential vehicle need not be high to cause illness. This suggests that the route and frequency of exposure, and size of the population who are exposed, are as important as the prevalence of contamination.

A study of 53 *Campylobacter*-positive faecal samples in the UK in 1999 found that 49 patients were infected with a single strain, while two strains of *Campylobacter jejuni* were detected in the faeces of 4 patients (Richardson *et al*., 2001). While this indicates that typing information from single isolates derived from individual cases will be useful, the wide variety of *Campylobacter* types that occur in both sources and human cases makes the detection of correlations difficult. This is evident in the Ashburton study. Correlations between types present in human isolates and a particular source must be treated with caution, in the absence of contextual information for a variety of sources.

For this reason no conclusions have been drawn in this document from the available typing data, apart from noting that the predominant serotypes from human cases in New Zealand are the same as those found in the UK and Denmark.

8.1 Is the Epidemiology of Campylobacteriosis in New Zealand Different to Overseas?

It was noted in Section 2.2 that the pattern of campylobacteriosis notifications in New Zealand, in terms of a number of criteria, is similar to that overseas, apart from the reported rate.

The comparison of rates of reported illness between countries is complicated by differences in reporting systems. However, it seems reasonable to compare broadly, rates between countries with laboratory-based systems, and a widespread or nationally-based surveillance system. These countries include the USA, Western Europe, and Australia.

Table 2 suggests the following distinctions in rates of reported disease: North America (<50/100,000) < Europe and Australia (approximately 80-120/100,000) < New Zealand (approximately 320 per 100,000).

The high reporting rate in New Zealand may be due to factors within the surveillance system. A corollary of this would be a lower reported:unreported case ratio. Support for this

possibility comes from the observation that cases of GBS have not increased in recent years, which suggests that total cases of campylobacteriosis are not sufficient to markedly affect the GBS rate.

The marked difference in reported rates of campylobacteriosis between the USA and New Zealand is difficult to explain. Poultry related risk factors are identified in most US case-control studies, and the prevalence of *Campylobacter* contamination in retail products was high, at least up until the year 2000. The most recent data suggest that both the prevalence of contamination and numbers of bacteria per carcass have declined markedly in the last 5-10 years. This may explain the decline in reported campylobacteriosis rates over the same period, and also contribute to the lower overall rate c.f. New Zealand. However, the prevalence of *Campylobacter* contamination of retail poultry in several other countries (particularly the UK) is comparable to New Zealand while reported rates of illness are lower.

It should also be noted that while the overall FoodNet reported rate of campylobacteriosis is low in the US, there are considerable regional differences, with much higher rates reported in California and Hawaii.

Generally there appears to be relatively little difference in notified campylobacteriosis rates between rural and urban regions in New Zealand. Rural cases represent a small proportion (8-12%) of the total reported cases.

The delineation of rural and urban populations on the basis of residence may create an artificial difference that is not reflected in differences in exposures. It seems likely that in New Zealand rural exposures, particularly animal contact, are experienced by many in the "semi-urban" residential population through occupational and social exposures.

Nevertheless, the data showing similar rural and urban relative rates of reported illness suggest that risk management measures most likely to cause a decline in campylobacteriosis rates should be directed towards the risk factors identified from studies that have concentrated on principally the urban population. Approximately 70% of New Zealand's population live in urban areas (centres with populations >30,000).

Little research into risk factors for young children (<5 years) in New Zealand has been conducted. The recent Australian case-control study (Stafford *et al.*, 2005) indicated that foodborne risk factors were not significant for this group. It would be helpful to better characterise this group in terms of reasons for the higher rate of illness, and risk factors.

8.2 Indicators of Transmission Routes and their Relative Importance

8.2.1 Overseas travel and person-to-person transmission

Many *Campylobacter* infections are acquired during international travel. It has been estimated that travel related exposures may account for 5-10% of cases in the United States, 10-15% of cases in Great Britain and Denmark, and 50-65% of cases in Sweden and Norway (Tauxe, 2000).

In New Zealand, the percentage of cases, for whom the information is known, reporting overseas travel prior to illness ranges from 6-12%. Overseas travel was a significant risk

factor in the national case-control study, and reported by 5% of cases. It seems reasonable to assign an important but minor proportion of the campylobacteriosis incidence to this risk factor.

Although person-to-person transmission is considered unusual for this illness, contact with a sick person appeared as a statistically-significant risk factor in both case-control studies. It may be that this risk factor is under-recognised, but was reported by only a small proportion of cases.

8.2.2 Food

8.2.2.1 Poultry

Poultry consumption has been identified as a risk factor in case-control studies in both New Zealand and other developed countries.

Comparing the case-control studies with the Ashburton study suggests that in New Zealand this transmission route may be more important for urban populations (although the unusually low prevalence of retail poultry contamination in the Ashburton study may not be replicated in other urban areas). Supporting evidence for the importance of poultry as a transmission vehicle includes:

- the high prevalence of contamination of retail poultry products in comparison to other meat types, and other foods;
- the apparently higher number of bacteria present on contaminated poultry, compared to other meat types,
- consumption of poultry by a high proportion (approximately 20%) of the population on a daily basis;
- increasing consumption of poultry, and an increasing trend towards fresh product, over the past 10-15 years, during which time campylobacteriosis notification rates have increased (the initiation of availability and increased sales of fresh rather than frozen poultry appears to have been one of the factors behind the marked increase in campylobacteriosis notifications in Iceland).

Campylobacter spp. are readily destroyed by cooking, and while it is plausible that undercooked poultry should be a risk factor in both case-control studies, the subjective judgement of undercooking by a high proportion of cases needs more definition.

Surveys from New Zealand and overseas indicate that the prevalence of *Campylobacter* in ready-to-eat foods is low.

Complicating this picture is that consumption of poultry at home was a protective factor in both New Zealand case-control studies, whereas poultry eaten outside the home (at friends or restaurants) was associated with higher risk. Such results have been replicated in other case-control studies in the US and Europe It may be that respondents in case-control studies have a bias to more easily recall poultry meals eaten away from home.

The contamination of the exterior of poultry packaging at retail premises creates the potential for exposure to *Campylobacter* during shopping. However, recent changes to leak-proof packaging by the major processor in New Zealand will help to reduce this risk.

Apart from under-cooking, transmission between poultry and humans is likely to be complex, and involve cross contamination from poultry to hands and kitchen surfaces. The introduction of a foodstuff with a high likelihood of contamination by *Campylobacter* spp. into the domestic or retail kitchen provides a starting point for such transmission.

8.2.2.2 Red meat

Red meat is consumed by the majority (78%) of the New Zealand population on a daily basis. However, meats other than poultry were not found to be risk factors in either of the two major New Zealand case-control studies.

The contamination prevalence of beef and sheep meat in the ESR surveys is low in comparison with poultry. Slightly higher contamination has been found for bobby veal and pork. A survey of sausages and hamburgers found no *Campylobacter*.

These data suggest that red meats play a minor role in the transmission of campylobacteriosis in New Zealand.

8.2.2.3 Offals

Offals are consumed by a small proportion of the population (4%) on a daily basis. Although all types of offals tested have shown some contamination, it appears that sheep and chicken livers are contaminated with *Campylobacter* at the highest rate (40% or more). An outbreak investigation in Christchurch in 2000 found that chicken liver pate was the source.

These data suggest that offals are a food vehicle in a small proportion of cases.

8.2.2.4 Raw milk

Raw milk has been identified as a risk factor in both outbreaks and sporadic cases, either in New Zealand or internationally. Pasteurisation of dairy products is mandatory in New Zealand, expect in limited circumstances associated with direct farm availability.

Raw milk consumption was a significant risk factor in the large 1992-1993 case control study, but only reported by a small proportion of respondents. Raw milk consumption was more common in the Ashburton study (reported by 20% of respondents).

The limited analytical data on raw milk does not indicate a high prevalence of *Campylobacter* contamination.

It seems likely that raw milk consumption represents a moderate to high risk for exposure to *Campylobacter* on a per serving basis, but only a small proportion of the population are likely to be exposed. In national terms therefore, this transmission vehicle will be a very minor part of the overall picture.

8.2.2.5 Other foods

There are limited data on other foods. The small amount of information concerning the prevalence of contamination of shellfish, and vegetables suggests they will not be important vehicles for transmission of campylobacteriosis.

8.2.3 Water

8.2.3.1 Potable water

There is no doubt that *Campylobacter* in drinking water can be the source of large numbers of cases of infection, and very large outbreaks have occurred overseas and more limited ones in New Zealand (although usually the evidence linking water contamination and outbreaks is weak). Contamination of drinking water by *Campylobacter* is likely to be associated with an unusual event (e.g. heavy rainfall/flooding or breakdown of water treatment), and likely to affect people as an outbreak situation.

Data from New Zealand drinking water indicates that *Campylobacter* is not present in the treated water supplies serving the vast majority (89%) of the population. There may be a problem with water supplies serving the remainder; some of these supplies will be rainwater derived and potentially contaminated by birds, but this must represent a minor transmission route. Limited data from *ad hoc* testing by ESR suggest that a small proportion of such supplies are contaminated by *Campylobacter*. A survey of a variety of water supplies found *Campylobacter* spp. in 37% of roof-water supplies, but none of the positive samples contained the species *C. jejuni* (Savill *et al.*, 2001b). Contaminated roof-water has been confirmed as a source in two outbreaks between 2000 and 2003.

A similar conclusion was reached in an analysis of data on campylobacters in water, sewage and the environment in the UK (Jones, 2001). Apart from the consumption of contaminated food, the author could not identify a clear route of transfer for *Campylobacter* from the environment to the consumer. In the United Kingdom, analysis of campylobacteriosis outbreaks derived from drinking water show that they are confined to private water supplies, predominantly found in small rural systems and more likely to be contaminated with animal waste.

Potable water is consumed in large quantities by the entire population, and it is possible that a very low level of contamination could result in large numbers of cases. However, current information does not indicate even a low level of contamination in treated supplies, and so this transmission route can be considered as a very minor component of the overall incidence.

8.2.3.2 Environmental (including recreational) water

Data from monitoring of ground and surface waters in New Zealand indicates that *Campylobacter* contamination is widespread, although generally the numbers of bacteria are very low. In terms of a transmission route for human exposure, it is most likely that this would occur during recreational activity, or else during occupational exposure. The analysis by NIWA suggested that recreational freshwater exposure could be responsible for around 4% of campylobacteriosis infections. While this estimate has considerable uncertainty, and

does not include recreational exposure to marine waters, it seems reasonable to assign recreational water exposure as a minor transmission route within New Zealand.

Occupational water exposure will principally affect the rural community, and will be linked to animal exposure. It seems reasonable to consider this an important exposure for the rural population. However the rural population comprises approximately 14% of the total New Zealand population and so as a contributor to overall campylobacteriosis infections occupational exposure to environmental water seems likely to be a minor transmission route.

The presence of *Campylobacter* in environmental waters (and the environment generally) is likely to play an important role in cycling of the bacterium in animals, causing infection in cows, pigs and sheep, and possibly poultry.

8.2.4 Animal contact (including occupational exposure)

8.2.4.1 Pets/companion animals

It seems reasonable to expect some transmission from domestic pets to humans. Overseas studies have found that pet ownership, particularly of puppies, was a risk factor. Pet ownership in New Zealand is widespread.

Ownership of a puppy or three or more caged birds were identified as risk factors in the 1994-1995 case-control study (Eberhart-Phillips *et al*., 1997), but the number of cases or controls reporting such contact was small (<5%). In general, having a pet at home was not a significant risk factor in this study.

From studies of domestic pets overseas it seems that infection with *C. upsaliensis* is significant, along with *C. coli* and *C. jejuni*. *C. upsaliensis* does not figure prominently amongst human isolates in New Zealand, although this species may not be tested for routinely by clinical laboratories. No New Zealand data on carriage of *Campylobacter* by domestic pets has been located, and overseas studies show considerable variability. Dogs/puppies appear to be more frequent carriers than cats, and this is consistent with risk factors identified in several case-control studies.

This limited information suggests that transmission from pets is a minor component of the overall picture, but further investigation is warranted.

8.2.4.2 Farm animals

Data on the prevalence of *Campylobacter* in the faeces of New Zealand farm animals is sparse, but the available results suggest that contamination may be high (up to 50%), with dairy cows apparently the most commonly contaminated. These levels are higher than results from Australia, where the prevalence of infection in farm animals was lower (<10%), except for feedlot cattle.

Occupational contact with calves or cattle was a significant risk factor in the 1994-1995 case-control study of a predominantly urban population (Eberhart-Phillips *et al*., 1997), but only small numbers of cases or controls were involved (<8%). Overall, contact with animals in the prior 10 days or at work was protective in that study.

Contact with animals, particularly bovines, was identified as an important risk factor in the Ashburton study. There have been anecdotal reports of spikes in campylobacteriosis cases occurring in rural areas during calving season. The size of the rural population (14%) will be bolstered by farm visitors, whose exposure will be intermittent.

Animal contact seems likely to be an important part of the exposure of a sector of the New Zealand population. In the national picture however, rural cases only represent 8-12% of the total, and, even if the majority of these are caused by animal exposure, from a risk management point of view, the importance of animal contact would be less than for poultry.

8.3 Risk Management

The key questions are:

- Is foodborne transmission of campylobacteriosis significant enough to warrant risk management?
- If so, which foods represent the greatest risk?

The second question is easier to answer. Based on results from case control studies, surveys of the prevalence and level of contamination, and consumption by a high proportion of the population, poultry products are the most common risk factor and the product most likely to be contaminated. The prevalence of contamination in other foods is at least ten-fold lower, and although consumption of red meats is approximately 4 times greater than for poultry, it seems unlikely that this would result in greater exposure.

The transmission of *Campylobacter* in New Zealand is likely to be complex, with a number of risk factors operating at once. It is possible that no single factor is sufficiently important to provide an opportunity to significantly affect the rate of illness.

However, it is the author's belief that effective management of the risk from *Campylobacter* in poultry will cause an observable reduction in the incidence of campylobacteriosis in New Zealand, for the following reasons:

- The proportion of campylobacteriosis cases in rural areas (8-12%) is similar to the rural population (approximately 14%), and notification rates in rural and urban total populations are similar;
- The majority of cases occur in urban regions, and case-control studies of predominantly urban populations have identified poultry associated risk factors as important (representing over 50% of the population attributable risk in one study);
- Even if there are differing transmission route patterns for urban and rural populations, the majority of the risk management activity should focus on the urban pattern;
- A temporary removal of poultry from the market in Belgium was followed by a 40% drop in campylobacteriosis notifications, and a decline in poultry consumption in 2003 in the Netherlands was associated with a reduction in incidence;
- Successful risk management of the incidence of campylobacteriosis in Iceland by focusing on poultry, alongside consumer education measures.

Of the remaining risk factors for campylobacteriosis, overseas travel and animal contact (for the rural population), appear to be the most important.

Potable water, pets, and environmental water, are likely to be more minor parts of the overall transmission route picture.

This report does not provide an answer for the key question of why the rate of reported campylobacteriosis in New Zealand is high compared to overseas countries. However, the available data from a variety of studies does indicate that poultry, as a source of *Campylobacter* and leading directly or indirectly to infection, is the risk factor whose management is the most likely to lead to a significant drop in illness.

The available New Zealand data are drawn from generally small scale and/or limited studies, and results should be treated with caution. Nevertheless, the main tools of epidemiological and microbiological investigation have been applied to the problem of campylobacteriosis in New Zealand and further research is unlikely to provide complete illumination of the transmission route matrix. The value of typing data (particularly newer techniques such as PFGE and MLST) will increase as numbers of typed isolates from various sources increase.

The notified rate of campylobacteriosis has declined markedly in the last year. It may be that leak-proof packaging for poultry introduced by processors and supermarkets has contributed. Such variations make the observation of the effect of risk management interventions more difficult to detect.

It is acknowledged that reducing the prevalence of contamination of the poultry supply by *Campylobacter* will be difficult. Despite considerable research by the industry and scientific community into on-farm and processing options, a "magic bullet" has yet to be found (some effective options such as irradiation are unacceptable to consumers). In recent years the New Zealand poultry industry has successfully reduced the prevalence of *Salmonella* in retail product to amongst the lowest in the world. However, the measures implemented have not reduced *Campylobacter* contamination. It is the intention of this report to indicate that efforts should continue to manage this risk.

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