Report to the Food Safety Authority of New Zealand

A Systematic Review of the Aetiology of Human Campylobacteriosis in New Zealand

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Contents

Executive Summary

Aims: To systematically review the available evidence around the aetiology of human campylobacteriosis in the New Zealand setting and to consider the scientific quality of that evidence (particularly concerning foodborne transmission).

Method: Searches for published and unpublished studies and reports were undertaken using: Medline, Google Scholar, Factiva.com, hand searches of selected New Zealand publications, and requests to 24 key informants. Outbreak surveillance data relating to campylobacteriosis for the most recent five-year period (2000 to 2004) was analysed. Additional analyses were conducted on data from the notifiable disease surveillance system (2004 data) and for selected data from various studies. Criterion used by the Institute of Medicine in the United States was used for evaluating causation.

Findings:

Review of case-control studies: This review identified five case-control studies of sporadic campylobacteriosis in New Zealand. One of these, the MAGIC Study, was relatively large and well designed and provides useful information about potential risk factors for campylobacteriosis in this country. Although having a number of methodological limitations, a Christchurch case-control study also provides some potentially valuable information, as does an Auckland study. There was significant overlap in the risk factors identified in these three studies (eg, around undercooked chicken). The other two studies (in Wellington and South Auckland) provide little or no useful information owing to their small size and other methodological problems.

Review and analysis of notification data and related studies: The notification data from the national surveillance system have various limitations (particularly around the completeness of some of the risk factor information). Although the notification data has its uses (eg, indicating the size of the burden to public health), it generally only provides limited quality evidence concerning the aetiology of campylobacteriosis in New Zealand. This evidence is suggestive of the likely heterogeneity of aetiological factors for different age groups, by rurality, by seasonality and the likelihood of changing risk factors or exposure levels (to account for the increasing trend over time). Comparison with data for other notified enteric diseases provides some limited evidence around the likely role of different risk factors (ie, the apparent importance of food relative to: water, animal contact, human contact and overseas travel). Fortunately, there is scope for further improvements in the surveillance system and potential ways to enhance the research agenda into this disease.

Review of outbreak investigation reports: A total of 13 published outbreak investigations were identified along with 16 unpublished ones (that met the quality criteria for inclusion in this review). The quality of the outbreak investigations was mixed, though some used high quality case-control and cohort study techniques, detailed site and food safety assessments, and state-of-the-art laboratory methods. The pattern of foodborne transmission predominating over waterborne was apparent. Foodborne spread was the most commonly described "likely" source of outbreaks in

nine reports. However, there were another six outbreak reports that provided some evidence for possible foodborne transmission. The implicated items included: raw milk, various poultry items, kebabs, and cheerios (often with evidence that these were undercooked). For some of these outbreaks there was high quality laboratory evidence that linked human serotypes with serotypes in the foods. There were also more foodborne outbreaks with statistically significant findings for food/s than there were for waterborne outbreaks (six versus two).

Waterborne spread was the next most commonly described "likely" source of outbreaks in these reports (n = 6). There was evidence for person-to-person transmission in three outbreaks. Zoonotic transmission was considered possible in three outbreaks but the evidence was very weak.

Analysis and review of outbreak surveillance data: A total of 216 outbreaks for campylobacteriosis in the most recent five-year period (2000-2004) were analysed. According to the transmission mode reported by Public Health Unit staff and for which an environmental investigation was undertaken, the following transmission modes were involved: foodborne (84%), waterborne (16%), person-to-person spread (6%), zoonotic (6%), and environmental source (6%) (ie, some outbreaks involved multiple transmission modes). However, the quality of the supporting evidence was mixed. For example, while 65% of outbreaks involved evidence from cases on exposure history, only 23% involved an environmental investigation, only 3% had laboratory evidence on the source, and only 2% involved a proper epidemiological study.

Various other limitations with the data also reduce the scope for using this information to better understand campylobacteriosis aetiology in New Zealand (especially for sporadic cases which comprise most of the disease burden). Nevertheless, the outbreak surveillance data do provide weak evidence to suggest that foodborne disease transmission is more important than waterborne and other transmission mechanisms. Furthermore, there remains substantial scope for improving the outbreak surveillance system so that it can better inform our understanding of the epidemiology of this disease and the research agenda for its control.

Environmental and laboratory studies: A particularly large study identified *Campylobacter* spp. in faecal, food and river water samples in the Ashburton area (a total of 1450 samples). The study identified the significant prevalence of *Campylobacter* for ten of 12 matrices studied (ie, all except rabbit and possum). The serotype patterns for livestock sources were more similar to the human ones than were those from water and the wild ducks. Further statistical analyses (done in this review) support the view that in this rural setting, livestock contact and/or consumption of livestock products may be more relevant to human illness than waterborne transmission or exposure to environmental contamination from wild birds. The data comparing the results for offal and faeces are also somewhat suggestive that foodborne transmission is more relevant for human transmission than is faecal contamination of water or land.

A number of other New Zealand studies have identified similarities between *Campylobacter* serotypes/subtypes from human cases and those found in: poultry, dairy and beef cattle, sheep/sheep products and recreational water. Specific

investigations into elevated notification rates have also identified such similarities between human and poultry isolates. All this work provides additional information but a key underlying concern is the possible instability of the *Campylobacter* genome which may be eroding the value of such comparisons.

Overall evidence – foodborne transmission: When considering all the data in this review, it appears that the Institute of Medicine's criteria for causality are met. That is, there appears to be "sufficient evidence for contaminated food having a causal relationship with campylobacteriosis in the New Zealand setting".

Overall evidence – burden of foodborne transmission: When considering all the findings of this review, it appears that the available evidence indicates that contaminated food is the dominant known cause of campylobacteriosis in the New Zealand setting. This evidence comes from:

- The findings of each of the three largest case-control studies.
- The overall pattern from the 29 outbreak investigation reports reviewed.
- The overall patterns found in five years of outbreak surveillance data.
- The notification data (ie, the case-case comparison analyses, the rural versus urban distribution and to a limited extent the time trend data).
- The similarities between the *Campylobacter* serotypes/subtypes from human and livestock isolates.
- The compatibility with the international epidemiological data indicating that foodborne transmission (particularly involving poultry) is the dominant transmission mode in developed countries.
- The compatibility with the findings from intervention studies and natural experiments in other countries (that show beneficial impacts on disease rates from reducing levels of contaminated poultry).

Although this evidence for the dominant role of foodborne transmission (relative to waterborne and other known forms) is fairly convincing for the New Zealand setting and other developed countries, the precise extent is still hard to determine for this country.

Options for health authorities: The following options could be considered by the relevant government agencies and local health authorities, particularly the Ministry of Health and the NZ Food Safety Authority:

1. Improvements in **disease surveillance** through various changes to the notifiable disease surveillance system.

2. Conducting further studies of the **notification data** such as case-case studies.

3. Improvements in the quality of **outbreak investigations.**

4. Improvements in **outbreak surveillance.**

5. Consideration of **intervention studies** relating to food sources in one region of the country (eg, involving poultry farming, processing of poultry products, mass media campaigns, and various regulatory interventions).

6. Consideration of **supportive national level actions** (eg, obtaining research funding from industry).

Acknowledgements

1 Introduction

Campylobacteriosis is an important foodborne disease in New Zealand and is the one with the highest level of notifications. There were a total of 12,235 notifications in 2004 and 14,790 in 2003 (ESR, 2005c). In the year 2000, there were 373 hospitalised cases attributable to campylobacteriosis (6.3% of the cases notified) (ESR, 2001). Over an eight-year period there were 10 deaths attributable to this disease (ESR, 2001). Furthermore, because of its high incidence, campylobacteriosis is the largest contributor to the economic costs of foodborne diseases in New Zealand (Scott et al, 2000).

The incidence of reported campylobacteriosis has risen fairly steadily in New Zealand since this disease first became notifiable in 1980 (ESR, 2002). The cause of this increase is unknown and the issue of the increase being a surveillance artefact has been considered. However, the authors of the one relevant laboratory survey concluded that changes in laboratory methodologies (at least in the early 1990s) did not appear to account for the national increase in notifications (McNicholas et al, 1995). Comparisons between notification and hospitalisation data also suggest that the increase of campylobacteriosis during the 1990s is a real phenomenon (Sneyd & Baker, 2003). Furthermore, changes in reporting behaviour by doctors are unlikely to have contributed to this observed trend as they have a fairly high level of notification of gastrointestinal diseases that are laboratory-confirmed (Simmons et al, 2002c). Indeed, data on salmonellosis also shows that over the 1995-2001 period about 92% of laboratory identified cases of salmonellosis were also notified (Thornley C. et al, 2002a).

There have been case-control studies, outbreak investigations, analyses of notification data and other studies that have examined risk factors for human campylobacteriosis in the New Zealand setting. There has also been some review work relating to foods posing a potential risk (eg, of *Campylobacter* in poultry (Lake R et al, 2003)) and a three-year investigation in the transmission routes of human campylobacteriosis (Baker et al, 2002). However, a review of the available New Zealand-specific epidemiological evidence concerning the aetiology of human campylobacteriosis in this country has not been undertaken. To address this issue more fully, the New Zealand Food Safety Authority commissioned this particular review.

2 Methodology

Medline searches: Numerous Medline searches were conducted to identify New Zealand specific articles and also to identify international scientific literature from which to put the New Zealand work into context. The search period covered January 1966 to 5 June 2005.

The New Zealand specific searches used the following search terms:
- "Zealand and campy*" (which identified n = 81 items of which some (n = 13) were irrelevant as they referred to *Campylobacter pylori* which is now *Helicobacter pylori* (all in the pre-1990 literature). More general search terms to identify relevant outbreaks and surveillance related studies included: "Zealand and outbreak"; "Zealand and surveillance and enteric"; and "Zealand and surveillance and communicable".
- New Zealand authors with publishing experience relating to *Campylobacter*: "Campy*" and the author names (as per the key informants list below along with others).
- Other New Zealand specific case-control studies relating to enteric pathogens ("Zealand and case-control" and: Salmonell* / Cryptosp* / Giard* / Crypto*").

The Medline searches were successful in identifying literature published in the *New Zealand Medical Journal* and also the *New Zealand Veterinary Journal*. Nevertheless, an additional hand search of the *NZ Veterinary Journal* was done for the period: 1995 (first issue) to June 2005 (most current issue).

Searches for non-Medline indexed literature: The Internet search engine most relevant to the academic literature was used (ie, "Google Scholar" http://scholar.google.com/). Search terms focused on "campylobacter", "food", and "New Zealand". To identify unpublished studies that may have been reported in the New Zealand print media and not elsewhere, the database covering major newspapers (Factiva.com) was searched using the search terms "campylobacter and study" (on 24 June). It identified 61 news items but no new studies.

The following publications were hand searched for articles relating to campylobacteriosis in New Zealand:
- *Communicable Disease New Zealand*: All the A5 format issues for 1991 to 1993.
- *New Zealand Public Health Report*: From the first issue (volume 1 in June 1994) to the last issue (volume 9 in October-December 2002).
- *New Zealand Public Health Surveillance Report*: For all issues up to the most recent available (ie, from the first issue in 2003 to the June issue of 2005). These publications are all available online at the ESR website.
- *New Zealand Journal of Environmental Health* (covering four issues per year for the period January 1990 to March 2005). These publications are not online.
- *Outbreak summary reports*: For all the reports for the period 2001-2003 (as per the ESR website).
- *Surveillance summary reports*: For all the reports for the period 2001-2004 (as per the ESR website).

For all the articles obtained, the bibliographies were searched for additional published and unpublished work of potential relevance to the New Zealand setting.

Requests from key informants: A personalised email request was sent to 24 key informants requesting if they could identify any additional published or unpublished studies or outbreak reports of relevance. The list was based on individuals who had previously done work in the area of campylobacteriosis or enteric disease epidemiology in New Zealand; who were currently engaged as a Medical Officer of Health (involved with communicable disease control); or were a Health Protection Officer known to have investigated a campylobacteriosis outbreak. The list comprised: Dr Derek Bell, Naomi Boxall, Dr Mel Brieseman, Dr Michael Baker, Dr Lester Calder, Dr Donald Campbell, Dr Simon Hales, Dr Andrew Hudson, Dr Jonathan Jarman/Dr Loek Henneveld, Dr Nicholas Jones, Dr Graham MacBride-Stewart, Dr Osman Mansoor, Dr Margot McClean, Dr Caroline McElnay, Dianne Morrison, Dr Annette Nesdale, Carolyn Nicol, Dr Alison Roberts, Dr Phil Shoemack, Dr Greg Simmons, David Speedy, Dr Craig Thornley, Dr Daniel Williams, and Rosemary Whyte. Of these people all but one responded and many provided additional information.

Discussions with key informants: A meeting was held with ESR staff with expertise in the notifiable disease surveillance system and the outbreak surveillance system (Naomi Boxall, Carol Kliem, Dr Graham MacBride-Stewart, and Dr David Phillips).

Examination of outbreak surveillance data reported to ESR: Public Health Services around New Zealand routinely supply ESR with outbreak surveillance data. These were examined for all campylobacteriosis outbreaks (ie, 2 or more cases) for the five-year period encompassing 2000 to 2004.

Considering the quality of the evidence: The categories of evidence used in this review were those used by the Institute of Medicine in the United States (Institute of Medicine, 2000):

> **"Sufficient Evidence of a Causal Relationship**
> Evidence is sufficient to conclude that a causal relationship exists between the action or agent and the outcome. That is, the evidence fulfils the criteria for "Sufficient Evidence of an Association" below and in addition satisfies criteria regarding the strength of association, biologic gradient (dose–response effect), consistency of association, biologic plausibility and coherence, and temporality used to assess causality.

> **Sufficient Evidence of an Association**
> Evidence is sufficient to conclude that there is an association. That is, an association between the action or agent and the outcome has been observed in studies in which chance, bias, and confounding can be ruled out with reasonable confidence. For example, if several small studies that are free from bias and confounding show an association that is consistent in magnitude and direction, there may be sufficient evidence of an association.

Limited or Suggestive Evidence of an Association
Evidence is suggestive of an association between the action or agent and the outcome but is limited because chance, bias, and confounding cannot be ruled out with confidence. For example, at least one high-quality study shows a positive association, but the results of other studies are inconsistent.

Inadequate or Insufficient Evidence to Determine Whether or Not an Association Exists
The available studies are of insufficient quality, consistency, or statistical power to permit a conclusion regarding the presence or absence of an association; or no studies exist that examine the relationship. For example, available studies have failed to adequately control for confounding or have inadequate exposure assessment.

Limited or Suggestive Evidence of No Association
Several adequate studies are mutually consistent in not showing an association between the action or agent and the outcome. A conclusion of "no association" is inevitably limited to the conditions, level of exposure, and length of observation covered by the available studies. In addition, the possibility of a very small elevation in risk at the levels of exposure studied can never be excluded."

Furthermore, the following issues around the scientific quality of the evidence obtained were given particular consideration:
- The strengths and limitations of case-control studies (Rothman & Greenland, 1998; Giesecke, 2002).
- The strengths and limitations of outbreak investigations (Goodman et al, 1990).
- The strengths and limitations of surveillance data (Giesecke, 2002) and particularly New Zealand notification data (Simmons et al, 2002c; ESR, 2003; Sneyd & Baker, 2003).
- The numerous limitations inherent in risk factor epidemiology (Taubes, 1995).

Limitations of this review

The scope of this review was limited in that it did not go into exhaustive detail into studies of peripheral relevance to human campylobacteriosis (eg, studies identifying the presence of *Campylobacter* in species with which humans have little direct contact). The search for studies on other enteric diseases in New Zealand and of overseas studies of potential relevance to New Zealand was also limited. Similarly, there may still be studies of relevance in the grey literature that were not identified in the *Google Scholar* search or by the key informants. Indeed, at least two key informants appeared to have forgotten about unpublished studies that they had authored (which were subsequently identified by other means). Other limitations of note are:

- Various limitations exist around the supplementary statistical analyses undertaken for this review (eg, the case-case comparisons in the analysis of the notification data). These issues are discussed further in the relevant places in the report.

- While this review included some further analysis of notification data – it did not do an in-depth and thorough analysis of national notification data (eg, covering a five-year period). This may ultimately be desirable but it was outside the scope of this project.

- Similarly, various further analyses could have been done on the outbreak surveillance data (though the return rate for new information may not be particularly high).

- Finally, this document has not been externally peer reviewed.

3 Review of Case-Control Studies (Sporadic Cases)

Background

Case-control studies are a useful epidemiological method for assessing risk factors for fairly uncommon conditions. This review identified three published case-control studies relating to campylobacteriosis in New Zealand (McMahon & Mahmood, 1993; Ikram et al, 1994; Eberhart-Phillips et al, 1997). In addition, two unpublished studies were also identified: (Neal G. & Bloomfield, 1997; Bennett et al, 2003).

In considering these studies, the limitations of case-control studies were considered (see the methodology section). Furthermore, the methodologies and results were considered in the context of published case-control studies concerning other enteric diseases in New Zealand:

- Giardiasis (Fraser & Cooke, 1991; Mitchell P. et al, 1993a; Hoque et al, 2001; Hoque et al, 2002; Hoque et al, 2003)
- Salmonellosis (Thornley C. N. et al, 2003)
- Yersiniosis (Satterthwaite et al, 1999)

Findings

1) National case-control study – the MAGIC Study

Background: This study (Eberhart-Phillips et al, 1997) was conducted to identify and assess the contributions of major risk factors for campylobacteriosis in New Zealand. It was funded by the New Zealand Ministry of Health and the Pubic Health Commission.

Methods: Home interviews were conducted over a nine-month period using a standardised questionnaire to assess various exposures. The setting was four major cities (and the surrounding rural areas) with high notification rates (ie, of Auckland, Hamilton, Wellington, and Christchurch). Case patients were 621 people (notified between 1 June 1994 and 28 February 1995) and controls were selected randomly from telephone directories (they were matched 1:1 with case patients in relation to sex, age group, and home telephone prefix).

Main findings (risk/protective factors): The risk of campylobacteriosis was found to be strongly associated with recent consumption of raw or undercooked chicken (matched odds ratio (MOR) = 4.52, 95% confidence interval (95%CI) = 2.88 – 7.10). There was also an increased risk with chicken eaten in restaurants (MOR = 3.85, 95%CI = 2.52 – 5.88) though recent consumption of baked or roasted chicken seemed

to be protective (as did "chicken prepared at own home" or purchased frozen). Other significant risk factors identified included: recent overseas travel, rainwater as a source of water at home, consumption of raw dairy products, contact with puppies and cattle (particularly calves), and occupational contact with cattle carcasses.

Main findings (population attributable risk percentages – PAR%): The results for risk factors where the PAR% was 5% or greater in the past 10 days were having: "no baked/roasted chicken" (27%); having any chicken raw or undercooked (11%); any chicken prepared at a sit-down restaurant (13%); any chicken prepared at someone else's house (6%); other raw or undercooked meat or fish (11%); and any unpasteurised milk (7%). The combined PAR% for the chicken related variables exceeded 50%. Furthermore, the PAR% for a preference for chicken pieces ($\geq$ 1 / wk) was 16%; and for puppy ownership it was 5%.

Strengths of the study:
- The size of the study was very large compared to other case-control studies into communicable diseases in New Zealand and when compared to overseas case-control studies into campylobacteriosis.
- The study participants came from four different sites around New Zealand and included an urban/rural mix (86% versus 14% for cases) that was fairly representative of the nation as a whole.
- The study design appeared to be of high quality and the exposure questionnaire was very detailed. The quality of the statistical analysis appeared to be high (eg, the multivariate analysis and calculating PAR%s).
- The nine-month period for recruiting cases was a relatively long time that only excluded the autumn season.
- There was thoughtful consideration of the limitations of the study in the discussion section (eg, around the likely impacts of selection bias and recall bias).
- The study was published in a high quality peer-reviewed international journal (with a relatively high impact factor).

Specific limitations with the study:
- *Selection bias:* Although attempts were made to reduce the risk of selection bias, the authors note that it was possible that the controls that ended up in the study were simply more likely than case patients to spend time at home. Such a bias could explain some or all of the risk seen with exposures to foods prepared outside the home.
- *Recall bias:* This bias could have still been relevant even though the authors used the technique of using the same recent reference period for both cases and controls (ie, the last 10 days – as used in some studies (Harris et al, 1986)).
- *Social desirability bias:* This bias may have played a role given that some questions could have been embarrassing for some respondents (eg, handling animal faeces, sewerage problems in the home, eating raw foods). However, this would have biased the results towards the null.
- *Questionnaire design:* Food handling issues in the home did not appear to be that thoroughly explored – at least relative to some in other case-control studies eg, (Friedman et al, 2004). Other potentially relevant factors that were

not considered were: antacid use, use of antibiotics during 28 days before illness (Effler et al, 2001), and the use of histamine H(2) receptor antagonists such as omeprazole (Neal K. R. et al, 1996). There was nothing about smoking behaviour (which involves hand-mouth contact); nothing on recreational water use; and nothing on contact with potentially contaminated settings such as beaches. The questionnaire did not include exposure window questions (ie, just the prior 10 days or prior month). (See the discussion around the Wellington case-control study below). However, adding extra questions can make a questionnaire too long and hence lower the validity of responses.

- *Possible overmatching?:* Matching by home telephone prefix (as well as sex and age group) may possibly have resulted in some overmatching (eg, when considering rural water supply issues).
- *Excluding outbreak cases:* There was no laboratory testing to identify particular serotypes of *Campylobacter* in the cases that could have allowed the identification of outbreak cases.

Overall assessment: This appears to be a high quality and relatively large case-control study. Indeed, it is probably the most informative study into human campylobacteriosis conducted to date in the New Zealand setting. The general pattern of the findings is likely to be valid given: (i) biological plausibility; (ii) consistency with other New Zealand evidence concerning campylobacteriosis epidemiology; (iii) consistency with other enteric disease epidemiology in New Zealand; (iv) and consistency with the findings from other studies of campylobacteriosis overseas. Nevertheless, all case-control studies have limitations (especially around selection bias and recall bias) and so the specific quantified findings (eg, ORs and PAR% estimates) should be treated with caution. The findings may also be somewhat out-of-date if the disease epidemiology has changed in the last decade and may be of limited relevance to understanding disease epidemiology in rural New Zealand. One area in which the MAGIC study (or future such studies) could be improved on, is in conducting sensitivity analyses around the issues of recall bias and selection bias. This would better quantify the extent to which such biases may be influencing the findings.

2) Christchurch case-control study

Background: This study (Ikram et al, 1994) was conducted to determine the risk factors for acquiring campylobacteriosis in Christchurch in the summer of 1992/3.

Methods: The study involved 100 cases and controls from urban areas matched for age and sex. Cases and controls were interviewed by telephone using a questionnaire.

Main findings (risk/protective factors): Risk factors identified included: eating poultry at a friend's house (OR = 3.18, 95%CI = 1.0 – 10.73, p = 0.03), at a barbecue (OR = 3.00, 95%CI = 0.99 – 9.34, p = 0.03) or eating undercooked chicken (OR = 4.94, 95%CI = 1.03, 23.62, p = 0.05). In contrast eating poultry at home was protective (OR = 0.36, 95%CI = 0.14 – 0.9, p = 0.02). Other factors associated with non-significantly increased risk, were drinking water from a non-urban supply (OR = 2.7, 95%CI = 0.89 – 8.33, p = 0.09) or consumption of chicken bought fresh (OR = 1.8, 95%CI = 0.85 – 3.82, p = 0.10).

Strengths of the study:
- As the first case-control study into campylobacteriosis in New Zealand of a reasonable size, it probably helped inform the research agenda at the time.
- The study was focused on the summer season that is of particular interest in public health terms due to the relatively high rates of notified campylobacteriosis during this season.
- The study was published in a peer-reviewed journal.

Specific limitations with the study: In addition to some of the general limitations discussed above for the MAGIC study (selection bias, recall bias, social desirability bias, limited range of questions etc) this study had the following specific limitations:
- The focus on just one city may limit the relevance of the findings to the rest of New Zealand.
- The urban focus limited the scope for investigating risk factors associated with rurality (though some participants were exposed to non-urban water supply).
- The size of the study meant that it would have lacked the statistical power to have adequately investigated some relationships.
- The method for control selection was not described in the published article.
- The statistical analysis was limited (no PAR%s calculated and no multivariate analysis). As the analysis was not matched (ie, matched ORs were not calculated) there was a loss of statistical power.
- The study lacked any discussion of possible limitations (eg, on important issues such as potential biases).

Overall assessment: This case-control study has a number of limitations in addition to those normally associated with case-control studies of this size. This suggests that the findings are of limited value and need to be considered in the context of the much larger MAGIC study and other sources of information on the epidemiology of human campylobacteriosis in New Zealand. Nevertheless, the findings are consistent with other New Zealand evidence (particularly the MAGIC study). Of particular note is that the finding "that eating poultry at home was protective" has been found in the MAGIC study and in some overseas studies. One possible explanation is a substitution effect if eating poultry at home displaces eating it from takeaways or at barbecues (where the risks may be higher). Other possible explanations are selection bias or immunity from regular exposure to *Campylobacter* from poultry brought into the home (Friedman et al, 2004).

3) Auckland case-control study

Background: This unpublished study was conducted in Auckland in October/November 1996 (Neal G. & Bloomfield, 1997). It was designed to investigate an increase in reported campylobacteriosis cases in September (some of which were associated with a specific *C. jejuni* serotype that was previously uncommon in New Zealand). However, as only around 20% of the cases in the study had illness dates that coincided with the apparent "epidemic period", this study basically evolved into one that studied endemic disease.

Methods: The study involved 55 cases and 55 controls aged 15 years and over from the Auckland region. Controls were family and friends and they were *not* matched for

age, sex or ethnicity. They were interviewed by both telephone and face-to-face (using a questionnaire).

Main findings (risk/protective factors): Risk factors identified included: eating barbecued chicken (OR = 10.6, 95%CI = 1.0 – 105.6, p = 0.06); eating undercooked chicken (OR = 9.6, 95%CI = 0.9 – 103.0, p = 0.09); and eating fast food (OR = 2.6, 95%CI 1.1 – 6.1, p = 0.04). There was confounding between these exposures and on further stratified analysis the results were all statistically significant (p = 0.01, p = 0.04 and p = 0.02 respectively). Recent overseas travel was also a significant risk factor (OR = 6.3, 95%CI = 1.0 – 41.4, p = 0.04) even though most cases (5/8) had travelled to developed countries (as opposed to developing countries). A non-statistically significant finding was the increased risk associated with having a rainwater supply (OR = 2.4, p = 0.11).

Strengths of the study:
- The study used stratified analyses to help to address initial design limitations (ie, the non-matching of controls with cases).
- The study focused on the spring season that in this region is when campylobacteriosis notification rates often increase.
- There was thoughtful consideration of the study's limitations.

Specific limitations with the study: In addition to some of the general limitations discussed above for the MAGIC study, this study had the following specific limitations:
- The relatively small size of the study meant that it would have lacked the statistical power to have adequately investigated some relationships.
- The control selection method (nomination by cases) has various limitations (such as overmatching) relative to the selection from the population. However, this approach probably resulted in a higher response rate.
- The lack of matching of the controls (at least by age and sex) and hence the lack of a matched analysis (though analyses adjusted for sex in stratified analyses). There was actually a 10-year difference in the median age of the two groups (older in the control group) that could have reflected selection bias.
- The study questionnaire was designed for the outbreak situation rather than for the study of endemic *Campylobacter* infection. Furthermore, some of the cases (around 20%) may have reflected the tail end of an epidemic event rather than endemic cases. Also the time span involved for collecting cases was brief (six weeks in spring).
- The focus on just one city (and the urban setting) may limit the relevance of the findings to the rest of New Zealand.

Overall assessment: This case-control study has various limitations in addition to those normally associated with case-control studies of this size. In particular, the control selection method was sub-optimal and there was no matching. It was also unusual in that a minority of the cases may have represented an actual outbreak. While the findings should be treated cautiously, they are all consistent with the findings from the MAGIC study.

4) South Auckland case-control study

Background: This study was conducted by the South Auckland Community Health Service to investigate risk factors associated with campylobacteriosis in October to November 1992 (McMahon & Mahmood, 1993).

Methods: The study focused on the spring period as this was a time associated with a peak in notifications in this area. Cases and controls were visited and interviewed face-to-face telephone using a pre-tested questionnaire and by the same person.

Main findings (risk/protective factors): The only statistically significant risk factor was "contact with a sick person" (OR = 10.3 95%CI = 1.8 – 103.0). Raised ORs (>2) were reported for contact with children (< 4 years of age) and for eating: offal, offal at home, offal boiled, beef fried, beef baked, chicken at "other" places, and precooked foods.

Strengths of the study:
- This was the first case-control study into campylobacteriosis published in New Zealand so it provided some information on which to base further studies.
- The focus on adults (20+ years) was probably helpful in simplifying the study, as was the focus on just those who had not travelled recently.

Specific limitations with this study: In addition to some of the general limitations discussed for studies detailed above, this study had the following specific limitations:
- The size of the study was very likely to be too small to have adequately investigated the key associations of interest.
- Similar limitations apply as per the Christchurch and Auckland studies detailed above (eg, the focus on just one urban area, number of risk factors considered, the lack of a matched analysis etc).
- The main control selection method (nomination by cases) has various limitations (eg, overmatching) as does the nomination of some controls on the basis of being "known by one of the authors".
- The study was written up in a brief report format so much of the detail on which to more fully assess the methods and results is not available.
- The authors may have over-interpreted their findings in the discussion section of their report given the lack of statistically significance with most of the results.

Overall assessment: This case-control study is of very limited value – owing mainly to its very small size. Its findings should probably just be ignored, but it could serve as a lesson against conducting underpowered case-control studies.

5) Wellington case-control study

Background: This study (Bennett et al, 2003) was conducted by a group of fifth year medical students as part of a public health project (ie, as part of an educational training experience). The client for the project was Regional Public Health.

Methods: The 50 cases were based on notifications between 25 February and 13 March 2003 (a time after the peak summer notification levels in this area). The 50 controls were matched by age group and Territorial Local Authority area. Cases and controls were interviewed by telephone.

Main findings (risk/protective factors): The strongest association was for consuming "chicken not cooked at home" in the last three days, but this was not at a statistically significant level (adjusted odds ratio = 2.13, 95%CI = 0.91 – 4.92, p = 0.11). Eating some foods was found to be protective when considering either the three or five day window period ie, bacon in the past five days (p < 0.01), eating pork in the last three days or five days (both p = 0.02), and eating yoghurt in the last three days (p = 0.045). Statistically significant non-food exposures that were found were drinking water from a water cooler in the last seven days (p = 0.03) and travel outside the Wellington region in the last seven days (both being protective). The authors noted that some of these associations might have been due to a substitution effect (ie, a food associated with "protection" may merely displace from the diet of the control a food associated with increased risk).

Strengths of the study:
- The focus on cases that had not travelled overseas was probably helpful in simplifying the study.
- The control selection via systematic changes to telephone numbers allowed those with unlisted phone numbers to be potential controls (but did greatly increase the number of phone calls required).
- The questionnaire was fairly comprehensive and the use of exposure windows was a methodologically sophisticated component of the study. It was reported that the interviewees did not appear to have any problem with understanding this method of questioning.
- The analysis was matched.

Specific limitations of this study: In addition to some of the general limitations discussed for studies detailed above, this study had the following specific limitations:
- The size of the study was likely to be too small to have adequately investigated many of the key associations of interest. Having such a broad age range of cases (< 1 to 88) may have lowered the quality of the study.
- The response rate for potential controls agreeing to participate was very low at 25%, and this may have introduced bias. The finding that more than twice as many controls than cases were smokers also suggests differences between the two groups.
- The time window for cases to be eligible for the study was very narrow at three weeks (and covered the end of summer and the start of spring).
- The large number of different interviewers involved (n = 11) as a result of the educational objectives of the project.
- The students conducting the study self-reported that they were not that confident with their interviewing skills. A person familiar with this study also expressed some reservations about the quality of the data collection by the students owing possibly to time pressure constraints and possibly to mixed levels of motivation [Anonymised personal communication].

Overall assessment: This case-control study is of fairly limited value – owing to its small size and various other methodological concerns. Nevertheless, some of the findings are plausible and the study did trial a methodological technique (exposure window assessment) that could be used in future case-control studies in the New Zealand setting.

Discussion

Case-control studies are a useful epidemiological method and they have been widely used internationally for exploring enteric disease epidemiology – including that of campylobacteriosis. Case-control studies have also proven to be useful for studying salmonellosis, yersiniosis and giardiasis in the New Zealand setting.

The New Zealand case-control studies considered in this review all have various specific limitations as detailed above (and summarised in Table 3.1 below). The biggest limitations with such studies probably relate to selection bias. In particular, even if the cases and controls have similar demographic characteristics they are likely to still differ in particular ways – given that the cases represent that minority of people who actually consulted a medical practitioner about their illness (given that most people with symptomatic campylobacteriosis do not (Wheeler et al, 1999)). Even so it seems unlikely that the highly significant key findings of the MAGIC study could be substantially attributable to selection bias.

The issue of recall bias may also be important. In particular, during the 1990s in New Zealand there was some public awareness around undercooked chicken being a risk factor for food poisoning. For example, it was noted by others that there "has been considerable publicity about the relationship between the consumption of undercooked chicken and campylobacteriosis" (Neal G. & Bloomfield, 1997). This type of recall bias could have biased the results away from the null. Nevertheless, it would be surprising if the recall bias accounted for the findings of the MAGIC study. Ideally, however, a sensitivity analysis of such bias (and for selection bias) should be performed with such studies in the future.

Another problem with all case-control studies into campylobacteriosis is the issue of host immunity, which is reasonably well documented for this disease (Allos, 2004; Blaser & Allos, 2005). That is some controls may have been exposed to infection but not become symptomatic due to their immunity. However, this type of misclassification bias would (fortunately), bias any risk factor findings towards the null.

Because of these issues, the precise findings of even large case-control studies need to be treated with some caution. Nevertheless, the largest of the New Zealand case-control studies (the MAGIC study) is still likely to have provided reliable information about human campylobacteriosis – and probably more so than any other New Zealand study to date. The next two largest case-control studies in New Zealand provide information of more limited validity – but the findings do add further support to some of the key findings of the MAGIC study.

Conducting a meta-analysis of the various case-control studies would be difficult due to variations in the questions asked. Also the dominance of the MAGIC study (in terms of size) would suggest that such a meta-analysis would be unlikely to provide new information.

Table 3.1: Brief summary of campylobacteriosis case-control studies conducted in New Zealand

Case-control study	Main findings	Overall assessment
National study – the MAGIC Study (Eberhart-Phillips et al, 1997)	The combined PAR% for the chicken related variables exceeded 50%. Many other specific food related and non-food risk factors were also identified (eg, puppy ownership).	A high quality and relatively large case-control study. Nevertheless, all case-control studies have limitations (especially around selection bias and recall bias) and so the specific quantified findings (eg, ORs and PAR% estimates) should be treated with caution.
Christchurch study (Ikram et al, 1994)	Significant risk factors relating to poultry consumption.	The next largest NZ study after the MAGIC study. Some useful information was obtained but the study had a number of methodological limitations.
Auckland study (Neal G. & Bloomfield, 1997).	Significant risk factors around fast food and travel. Increased risk associated with poultry consumption (but not at a statistically significant level).	This unpublished study provides some information but was limited by its size and other methodological limitations.
South Auckland study (McMahon & Mahmood, 1993).	Contact with a sick person was significant but food related risk factors were non-significant.	This study was probably too small to provide any useful information.
Wellington study (Bennett et al, 2003)	Various findings around certain foods being "protective".	This study is of fairly limited value owing to its small size and various other methodological concerns.

Should further case-control studies of sporadic cases be conducted in New Zealand?

Given the remaining uncertainties about the aetiology of campylobacteriosis in New Zealand (especially the relative importance of identified risk factors) it may be reasonable to undertake further case-control studies into sporadic cases. This may be particularly justifiable if there is evidence of changes in the relative importance of possible risk factors associated with the increase in the notification rate in the 10 years since the MAGIC study. There are various problems with undertaking another large case-control however:

- The high cost of doing a study of an adequate size (ie, ideally hundreds of cases).
- The growing difficulty of obtaining controls who will answer a long list of questions eg, as per the experience with the Wellington case-control study (Bennett et al, 2003). This problem could be partly addressed by providing rewards for participation – but this can be ethically and administratively problematic.
- The possibility of growing social desirability bias (eg, if people are becoming more embarrassed about admitting to poor personal hygiene, poor food hygiene and eating foods that might be considered "unhealthy").

Given these problems it may be more cost-effective and worthwhile for the New Zealand health sector to focus resources on doing more "case-case" studies (where the "controls" are other cases), possibly case cross-over studies, and also actual intervention studies (see Section 8).

Studies in which there is direct observation of food handling are likely to be of higher validity than those involving self-reported practices (Redmond & Griffith, 2003). Nevertheless, such studies are probably particularly expensive to perform.

Summary

This review identified five case-control studies of sporadic campylobacteriosis in New Zealand. One of these, the MAGIC Study, was relatively large and well designed and provides useful information about potential risk factors for campylobacteriosis in this country. Although having a number of methodological limitations, a Christchurch case-control study also provides some potentially valuable information, as does an Auckland study. There was significant overlap in the risk factors identified in these studies (eg, around undercooked chicken).

The other two studies (in Wellington and South Auckland) provide little or no useful information owing to their small size and other methodological problems. Although there are some advantages of undertaking another large case-control study in New Zealand, other options on the research agenda may be more worthwhile.

4 Review and Analysis of Notification Data and Related Studies

Background

Medical practitioners are legally required to notify human campylobacteriosis to the local Medical Officer of Health (Baker & Roberts, 1996). The notification data are collated by all the Public Health Services around New Zealand and routinely provided to ESR (via weekly electronic data submission). This section considers recent notification data on campylobacteriosis and conducts some additional analyses. It also considers published and unpublished studies that have specifically considered notification data on campylobacteriosis.

Findings

Studies identified

In addition to the routine descriptions and analyses of campylobacteriosis notification data in reports by ESR (in the CDNZ, NZPHR, NZPHSR, and Annual Surveillance Reports), the following published reports were identified: (Brieseman, 1985; 1990; Lane & Baker, 1993; McNicholas et al, 1995; Nylen et al, 2002; Simmons et al, 2002c; Hearnden et al, 2003; Kovats et al, 2005).

The unpublished reports identified were: (Williams, 1998; Baker & Sneyd, 2004; Morrison & Smith, 2004). These studies are considered below in order of publication date. Older unpublished reports that were pre-1995 eg, (Mansoor & Durham, 1993) were not reviewed in detail as they add little information. Other studies that included some human data (derived from notifications) but were primarily focused on other aspects of campylobacteriosis (eg, transmission pathways in the environment) are discussed in Section 7.

Study quality and relevance to disease aetiology (studies using notification data)

Christchurch study (1981-83 data): This study (Brieseman, 1985) is limited since it involved a time period so soon after campylobacteriosis became notifiable (mid-1980). Therefore under-reporting may be a particularly major limitation. The level of statistical analysis of the data was also limited. Findings generally mirrored those of the more recent notification data at the national level (see below) but particular points of note were:
- A rural excess when considering local authority areas surrounding Christchurch city ($p < 0.01$ for 1982 and 1983).

- Higher rates in some occupational groups ("housewives", "meat handlers", "farmers" and the "unemployed") – but without statistical testing.
- Exposure information indicating that: 10% of cases had some household contact with a person with diarrhoea in the week preceding symptoms, 60% had eaten meals outside the home, 40% had eaten takeaways, 4% had been on overseas visits, and 73% had frequent contact with animals (mainly domestic). However, no control data was presented to put these results into context.

Second Christchurch study (1981-88 data): This study (Brieseman, 1990) considered eight years of data for the Christchurch Health District. Findings generally mirrored those of the more recent notification data at the national level (see below) but particular points of note were:

- A rural excess was apparent for all but one year (but the statistical significance was not tested).
- The higher rates for some occupational groups described in a previous analysis (Brieseman, 1985) were not confirmed.
- Exposure information for specific foods indicating that: 58% had consumed chicken, 33% seafood, 29% café sandwiches, 26% meat pies and 6% raw milk. However, no control data was presented to put these results into context.
- Exposure information for animal contact indicating that between 52% and 62% of cases handled cats or owned a cat at home (for the years 1985-1988). Similarly for dogs the range was 39% to 44%. Relative to age-specific exposure data from the Christchurch Child Development Study, the figures for cats were similar, but dog exposure was higher among the cases (but this was not analysed statistically).

Review of national data (mainly 1990-1993 data): This study reviewed notification data and conducted additional analyses (Lane & Baker, 1993). The demographic data and seasonal variation were similar to the more recent notification data at the national level (see below). Particular findings of potential relevance to possible aetiology were:

- Comparisons between notification data and laboratory data (Wellington area) indicated that these showed a very similar seasonal pattern, and both showed an increase over the three-year period.
- The increase in notifications was considered unlikely to be a reporting artefact as the proportion of diarrhoeal specimens where *C. jejuni* was isolated also increased.
- The most commonly identified "probable source" was chicken (but 84% of notifications reported the likely source of infection as unknown). The other most frequently reported sources were animal contact, human contact and untreated water (but without the basis for these exposures being reported in the notification data).

Study of laboratory procedures of relevance to notification trends (survey in 1994): This study (McNicholas et al, 1995) surveyed 69 microbiology laboratories concerning their procedures. It identified an increase in laboratory-identified *Campylobacter* positive specimens between 1992 and 1993. It also considered that the changes in laboratory methodologies over a five-year period did not explain regional differences in notification rates or the increase in campylobacteriosis notifications. The study appeared to be well designed with a 100% response rate and coverage of all

but one of the country's 70 laboratories. There appear to be no substantive methodological limitations likely to invalidate the conclusions.

Study of notification data in Canterbury (1996-1998 data): This unpublished study (Williams, 1998) examined notification data for Canterbury (north of the Rakaia). The findings are generally consistent with nationally reported notification data though it reported a rural excess in notification rates (with double the urban rates for ages 0-9 and 15-24, but no excess for those over age 25 years). Monthly notification rates were strongly correlated with average temperature but there was a weaker, negative correlation with rainfall. For urban areas, notification rates were lower in more deprived Census Area Units. The author concluded that this association was likely to reflect differences in general practice utilisation and behaviour.

Study of Auckland notification data quality (year 2000 data): This study (Simmons et al, 2002c) focused on quality aspects of the reporting of foodborne diseases in the area covered by the Auckland Regional Public Health Service (including campylobacteriosis). It matched notification data with laboratory data and concluded that laboratory-based notification was desirable. Specific findings of this study are considered in the context of other quality aspects of the surveillance system (in the next subsection). Overall however, this study found a mix of relative high quality aspects (eg, 76% of laboratory-confirmed cases reported) and poorer aspects (only 7.5% of cases investigated). The study appeared to be a thorough one with no methodological limitations likely to invalidate the conclusions.

International study of seasonality (including NZ data): This study examined the seasonal distribution of campylobacteriosis in nine European countries and New Zealand (Nylen et al, 2002). Data for the period 1993-7 for New Zealand clearly show peaking of notifications in the summer season along with some evidence for smaller peaks in spring. But relative to the comparison countries, the seasonality in New Zealand was "less consistent since the peak was more prolonged". For example, only 7% of New Zealand cases were within the one week of the peak week whereas for the other nine countries the range was 9-14%. It was also noted that the peak week was more variable from year to year in New Zealand than the other countries. This study appears to have assembled some reasonably comparable international data but it does not provide any clear explanations for the different seasonal patterns.

Study of regionality: This study (Hearnden et al, 2003) examined the regionality of campylobacteriosis seasonality in New Zealand. Using notification data for local authority areas it reported significantly higher summer and autumn disease rates for the South Island than the North Island. It also reported lower rates in the Far North and central regions of the North Island. The three major patterns identified were: (i) For the rural North Island – relatively low summer incidence and low inter-seasonal variation; (ii) For the urban North Island – relatively high summer incidence and a greater degree of seasonality than the rural North Island; and (iii) For the urban South Island – the highest summer incidence and in general the greatest seasonal variation. Overall, the modelling suggested that the "space-time visualisations of notified campylobacteriosis incidence do appear to be fairly robust" and that the analysis indicated "that multiple risk factors are at work".

A major concern is the quality of the regional notification rates – given that they do not correlate with hospitalisation rates (Sneyd & Baker, 2003). This issue (which is discussed further below) makes this reviewer very sceptical of the value of the findings of Hearnden et al. Even if these results do reflect the true situation, the implications for understanding disease aetiology also appear to be rather limited (given that both environmental and human behavioural factors change with the seasons).

Additional analysis of notification data in conjunction with hospitalisation data: This unpublished study considered notification data for 2001-2003 (Baker & Sneyd, 2004). It appears to have undertaken the appropriate methodological steps and its relevant findings are considered in the context of other notification data in subsequent parts of this Section.

Analysis of notification data for Christchurch city: This unpublished study considered notification data for 2003 (Morrison & Smith, 2004). The epidemiology for notified campylobacteriosis was found to be fairly similar to that at the national level. A specific finding of note was the higher rates in three out of four of the more rural territorial authorities in Canterbury relative to Christchurch city. Amongst identified types of farm workers there were 17 dairy workers and 6 poultry workers with notified campylobacteriosis – but no denominator data was provided. The authors noted that the review had been prompted by the increase in notifications along with "concerns being expressed about problems at food premises, sewage contamination of bathing beaches and water supply quality".

International study of climate variability including NZ data: This study (Kovats et al, 2005) considered the seasonal pattern of laboratory-confirmed campylobacteriosis in 15 populations from Europe, Canada, Australia and New Zealand (for NZ the data was for 1991-2000). The study found that "all countries in this study showed a distinct seasonality in *Campylobacter* transmission, with many, but not all, populations showing a peak in spring." In fact the New Zealand data showed a summer peak. The seasonality was less pronounced in Australian cities than in New Zealand (and for the Australian sites the peaks were 1-9 weeks earlier). There was also a difference in seasonality between North and South Island (ie, the peak week was six weeks later in the South Island). For all the 15 sites/countries, the timing of the peak of infection was weakly associated with high temperatures three months previously. But no effect of rainfall was found. From this study the authors concluded that "the geographical variation in the timing of the seasonal peak suggests that climate may be a contributing factor to *Campylobacter* transmission."

This study is limited by the likely variations in the quality of reporting between sites/countries. Also some of the time estimates for the delay between onset of symptoms and reporting date was large in some countries (eg, 30 days for one). While providing some results of interest, this study does not provide major new insights into the epidemiology of campylobacteriosis in New Zealand. (See below for a further discussion of issues around seasonality).

Quality of the notification data and the surveillance system

In the Annual Report for 2002 produced by ESR, there was some in-depth consideration of the quality of surveillance data relating specifically to campylobacteriosis. This information, along with more recent data, is considered below and is put into context with other relevant New Zealand and international studies.

Surveillance system sensitivity: This criterion refers to the proportion of disease cases that are detected by the surveillance system. For campylobacteriosis, as for other enteric disease it is likely to be low since only a small proportion of those infected will present to a doctor and have the diagnosis confirmed by laboratory testing. For example, in England the ratio of cases in the community to cases reaching national surveillance 7.6 to 1 for campylobacteriosis (Wheeler et al, 1999).

Positive predictive value (PPV): This criterion refers to the proportion of reported cases that actually have the disease under surveillance. This proportion is in turn affected by the specificity of the case definition and incidence or prevalence of the disease under surveillance. While not specifically calculated in the ESR Report for 2002, the PPV for campylobacteriosis is likely to be very high given that 98.1% of notifications in 2002 were laboratory confirmed (with 0.1% based on epidemiological criteria and 1.8% based on clinical criteria). Early Auckland data for 2000 was not as good as it indicated that only 76% of the laboratory-confirmed cases of campylobacteriosis were notified (95%CI = 75% – 78%) (Simmons et al, 2002c). The proportion notified by hospital practitioners was higher than those in community practice but this difference was not statistically significant.

Representativeness: This criterion refers to how well the characteristics of cases reported by the surveillance system represent the true characteristics of cases of that disease. To access representativeness by place, a comparison between notifications and hospitalisations for campylobacteriosis was undertaken (trend analysis using the method of least-squares) (Sneyd & Baker, 2003). It found that there was little correlation for campylobacteriosis. It concluded that "the high variability in the comparative rates of campylobacteriosis is probably due in part to different reporting practices among Public Health Units (PHUs)". This is a very plausible conclusion and suggests that representativeness by place is poor for this surveillance system regarding notified campylobacteriosis.

However, the comparison between notification and hospitalisation data for 2002 suggests that the ethnicity differential may be real (ie, European rates higher than Maori rates for both data sets). Nevertheless, the scale of the differential was much less for the hospitalisation data. This is suggestive that notifications considerably underestimate the relative burden of campylobacteriosis borne by Maori (and similarly for Pacific peoples).

Accuracy and completeness of associated data: These criteria refer to: (i) whether or not important demographic, outcome and risk factor information is reported; and (ii) if it is accurate and can be appropriately analysed. For recent data, the completeness is high for basic demographic information but much lower for risk factor data (Table 4.1). In 2002, additional completeness data for campylobacteriosis included:

geocoding (96.6%), date of onset (50.4%), and death (87.6%) (Sneyd & Baker, 2003). Other specific completeness data relating to campylobacteriosis has not been reported on, but more general information is available for all notifications (eg, for 2004):

> "Of the notifications with an onset date recorded (62.8% of notifications) in 2004, 40.0% were reported to a public health service within one week of the onset of symptoms and 71.4% were reported within two weeks. In 2004, 95.1% of disease notifications were entered into EpiSurv within one week of being reported to the public health service and 97.0% were entered within two weeks of being reported."

This 2004 report also noted that 52.5 % of all notifications are linked to the National Health Index (ESR, 2005b).

Data from Auckland indicates that the median notification delay for campylobacteriosis was two days (with the 75th centile being three days) (Simmons et al, 2002c). However, there were some very long delays in the notification of campylobacteriosis (eg, up to 224 days) owing to cases having multiple tests and being notified after subsequent positive results. The proportion of all campylobacteriosis notifications actually investigated was only 7.5% (though 56% of "high-risk" cases were investigated).

Table 4.1: Completeness of information recorded in campylobacteriosis notifications (2003-2004 data based on the Annual Surveillance Reports on the ESR website)

Information record on:	2003 (n = 14,786)	2004 (n = 12,213)
Sex	98.6%	97.9%
Age	99.5%	99.1%
Ethnicity	78.4%	79.8%
Hospitalisation status	56.1%	53.6%
If case had / had not consumed food from retail premises	27.6%	23.7%
If case had / had not contact with farm animals	23.8%	28.3%
If case had / had not consumed untreated water	27.6%	24.3%
If case had / had not recreational water contact	30.7%	27.0%
If case had / had not contact with faecal matter	NR	26.0%
If case was / was not a food handler	NR	3.3%
If case had / had not been overseas during the incubation period	35.8%	31.7%

Demographic and risk factor information from the notification data of possible relevance to understanding aetiology

Gender: The male excess in campylobacteriosis rates in all age groups in New Zealand (Sneyd & Baker, 2003) has no obvious implications for understanding disease aetiology. This is because it may reflect underlying immunological differences between the genders (as is apparent for many other infectious diseases). Also there are many different and poorly described demographic and behavioural factors that could be involved (eg, different occupations, different food/water consumption levels, different food preferences, different food hygiene and food preparation behaviours, different levels of animal contact, and different levels of contact with young children etc).

Age: The age groups with the highest average annual notification rates are children aged 1-4 years and then adults aged 20-29 years (Sneyd & Baker, 2003). Immunological factors may wholly or partly explain the higher rates for pre-school children (eg, especially for the male excess for infants under 1 year). However, other possibilities include higher rates of contact with animals, environmental exposures from playing outdoors, closer contact with other humans and poorer levels of hygiene than other age groups. Relatively high parental concern about diarrhoea in infants and children could also be relevant.

The likely cause/s of the peak for 20-29 year olds (for both males and females) is also unclear. Possibilities include poorer food hygiene practices than older adults, higher

intakes of food from food premises, and higher exposure to recreational water. The bimodal age distribution seen for New Zealand has also been seen in other developed countries.

Ethnicity: The ethnic differentials in campylobacteriosis may not be real given the differences between notifications and hospitalisation data for the year 2002 (Sneyd & Baker, 2003). Therefore these data appear to provide no useful information relevant to understanding disease aetiology.

Occupation: This variable has not been reported on for national notification data. The data described previously for Christchurch indicate no clear patterns by occupational group (Brieseman, 1990). Data collected on different types of farm workers with notified campylobacteriosis in a Christchurch study (Morrison & Smith, 2004) need denominator data to allow interpretation.

Time trends: The time trend since 1980 shows a fairly steady increase with annual decreases only occurring in two years (1999 and 2003) (Lane & Baker, 1993; Sneyd & Baker, 2003; ESR, 2005b). Hospitalised cases more than tripled from 1995 to 2002 (Sneyd & Baker, 2003) which was even more than the relative increase in notifications over that period (nearly a doubling). Of note is that the incidence of notifications and hospitalisation both dipped in the same year (1999) and both increased in every other year over the 1995-2002 period. As detailed in the work described previously (Lane & Baker, 1993; McNicholas et al, 1995), there is reasonable evidence to suggest that the increase in campylobacteriosis is real and not a surveillance artefact or due to changes in reporting or laboratory practices. The only contradictory piece of evidence is that the increase in reported campylobacteriosis has not been associated with a concurrent increase in known sequelae of this infection (particularly Guillain-Barré syndrome) (Lake R. et al, 2004). However, other quite plausible reasons for this exist, including the possibility that New Zealand may have a lower prevalence of the serological types of *Campylobacter* linked to immune-mediated illnesses.

The increase in reported campylobacteriosis does not however, provide much evidence towards the possible aetiology. This is because the temporal patterns in contamination levels of any animal species or environmental system have not been adequately described (except for a one-year period in one river catchment – (Eyles et al, 2003)). The historical data on raw poultry contamination since 1984 (Lake R et al, 2003) is not extensive enough to determine time trends. Water contamination is unlikely to be a major source for the increase since there has been substantial progress in improving the microbiological quality of reticulated water supplies in New Zealand since the early 1990s (Ministry of Health, 2005).

There have been increased average temperatures over the last two decades in New Zealand (National Institute of Water and Atmospheric Research (NIWA), 2005). This may have also favoured the survival of *Campylobacter* in various settings, given the evidence that the timing of peak infection in various countries is weakly associated with high temperatures three months previously (Kovats et al, 2005). Climatic factors may also contribute to any role that flies play in transmission of this organism (Nichols, 2005).

There is some evidence of an overall increase in poultry consumption which nearly doubled during the 1990s in real terms (Statistics New Zealand, 2000). However, these data are not partitioned by different products eg, frozen versus other forms.

There is limited evidence for changing levels of potential behavioural risk factors over time. For example, spending on "takeaway food" in 2004 has been reported to be almost 50% higher than in 1998 (ie, equivalent to an extra burger per week for every citizen) (Johnson M., 2005). While it is plausible that the frequency of barbequing has also increased over time, this author knows of no relevant data. There may also have been a decline in home cooking and food safety skills in recent decades, but confirmatory data on any such trends also appear to be lacking.

Seasonality: The data in the 2004 Annual Report shows the seasonal pattern in notifications (summer peak and winter trough) – with January the peak month (2004 report). However in 2002 the summer peak was followed by an unusually high incidence rate throughout winter and early spring (Sneyd & Baker, 2003). Analysis of data for the period 2001 to 2003 indicated that 32.0% of notified cases occurred over the summer period (December to February) and only 21.2% occurred in winter (June to August) (Baker & Sneyd, 2004). These findings are consistent with the New Zealand data in the international study that has been discussed above (Kovats et al, 2005).

Nevertheless, the seasonal pattern is not particularly informative of campylobacteriosis aetiology in the New Zealand setting. This is because so many factors are associated with the summer season. For example, sub-optimal food hygiene may be more of a risk, there is more barbequing and eating outdoors, greater exposure to recreational water, and probably higher consumption of untreated water (eg, when people go camping, use baches and go hiking). Also in summer, livestock may spend more time in streams drinking and keeping cool (which may increase water contamination levels), stock density is higher in summer and the data for one river shows slightly higher *Campylobacter* contamination levels in summer (Eyles et al, 2003). Levels of *Campylobacter* in the faeces of dairy cattle vary by season (highest in autumn then summer – (Meanger & Marshall, 1989)), and there may also be fairly different activity levels of birds and other wild animals by season that are relevant.

Rurality: The 2002 Annual Report by ESR reported that there was no statistically significant rural excess for notification rates of campylobacteriosis (as was the case for giardiasis, listeriosis and yersiniosis) (Sneyd & Baker, 2003). Yet such a rural excess was evident for some enteric diseases (cryptosporidiosis, salmonellosis (total), *Salmonella Brandenburg*, and *S. Typhimurium*, VTEC); while significantly higher urban rates occurred for gastroenteritis. However, a more sophisticated (but still unpublished) analysis using both notification and hospitalisation data for the years 2000-2002 collectively reported that:

> "The overall age-adjusted risk of notified campylobacteriosis was significantly higher for rural dwellers than those residing in urban areas" … "Restricting the analysis to children (< 15 years) increased the strength of the rural association for campylobacteriosis for both notifications and hospitalisations. In contrast, for adults there was a significant direct association between urban

residence and both notifications and hospitalisations" (Baker & Sneyd, 2004). (In this analysis settlements of 1000 people or more were classified as "urban" as per a Statistics New Zealand classification system).

More specifically for children under age 15 years there was a rural excess for notifications (ie, relative risk (RR) = 1.12, 95%CI = 1.08 – 1.15) and for hospitalisations (RR = 1.72, 95%CI = 1.31 – 2.26).

The rural excess for campylobacteriosis in children may suggest that in this age group the roles of animal contact or exposure to contaminated environments are of relevance. However, the urban excess for adults may suggest that these factors and also drinking untreated water are not particularly important for adults. But to some extent this could be a surveillance artefact in that adults in rural areas with mild or moderate symptoms from campylobacteriosis may be less likely to consult a doctor.

Regional variations: Rates of notified campylobacteriosis vary markedly by District Health Board with some having over twice the level of others (Sneyd & Baker, 2003). However, given the absence of a correlation with hospitalisation rates (Sneyd & Baker, 2003) these differences may largely reflect a surveillance artefact. Issues around regional (or North versus South Island) variation are also considered in the discussion of studies detailed at the start of this section (Hearnden et al, 2003; Kovats et al, 2005).

Case-case analyses of campylobacteriosis versus other notified enteric diseases

In this section comparisons are made between campylobacteriosis and the other notified enteric diseases. Some statistical analyses have been performed but these need to be treated cautiously given: (i) that there are relatively high levels of incomplete data for risk factors associated with these notifications; (ii) there is no distinction made between sporadic and outbreak cases; and (iii) there may be differential levels of recall bias involved (eg, if people had high awareness of certain associations such as between giardiasis and untreated water; or between campylobacteriosis and uncooked chicken).

1) Food from a food premise: The data for 2002 (Table 4.2) indicate that eating from a food premise during the incubation period was most frequently described for those with acute gastroenteritis (88%). The lowest frequencies were for cryptosporidiosis and giardiasis (33% and 32% respectively). These are plausible results given that the latter diseases are probably more likely to be waterborne than foodborne. Given that for campylobacteriosis the frequency was 54%, this would therefore be consistent with some role for such food being a risk factor. Analysis by this reviewer of the 2004 data (Table 4.3) also suggests that most other enteric diseases have statistically significantly lower rates of being associated with cases' reporting having consumed food from food premise, relative to campylobacteriosis (the exception being yersiniosis in which the difference was not significant).

2) Untreated drinking water: The data for 2002 (Table 4.2) indicate that consumption of untreated drinking water was most frequently described for those with typhoid (67%). The lowest frequencies were for gastroenteritis and yersiniosis (6% and 19%

respectively). These are plausible results given that the latter diseases are probably more likely to be foodborne. Given that for campylobacteriosis the frequency was (20%) – this would therefore be consistent with some possible role (but probably not a large one) for untreated drinking water being a risk factor.

Analysis of the 2004 data (Table 4.3) also indicates for all of the enteric diseases analysed there were higher reported rates of untreated drinking water exposure relative to campylobacteriosis. These results were highly statistically significantly for giardiasis and cryptosporidiosis.

3) Recreational water contact: For exposure to recreational water, the frequency for campylobacteriosis notifications (at 17%) in the 2002 data was between the extremes for gastroenteritis (the lowest at 6%) and cryptosporidiosis (the highest at 35%) (Table 4.2). For the 2004 data it was the lowest for campylobacteriosis, and statistically significantly so relative to yersiniosis, giardiasis and cryptosporidiosis (Table 4.3). These findings are suggestive of a minimal role for recreational water exposure in the aetiology of campylobacteriosis in the New Zealand setting.

4) Farm animal contact: The frequency for "farm animal" contact reported in the 2002 data for campylobacteriosis notifications (at 33%) was between the extremes for gastroenteritis (the lowest at 8%) and cryptosporidiosis (the highest at 59%) (Table 4.2). For the 2004 data it was significantly greater than giardiasis and shigellosis but significantly less than cryptosporidiosis (Table 4.3). It was not significantly different from salmonellosis or yersiniosis. These findings would therefore be consistent with some modest role for this exposure in the aetiology of campylobacteriosis. The pattern for "sick animal" contact was similar (Table 4.2).

5) Human contact: The frequency for different forms of human contact reported for campylobacteriosis notifications was near the bottom of the ranges for contact with faeces/vomit, for contact with cases and for school childcare (Table 4.2). For example, the frequency was 6% for contact with a confirmed case while the figure for gastroenteritis was 67%. For the 2004 data, campylobacteriosis was the lowest in the range for "contact with faeces" and significantly lower than yersiniosis, giardiasis, shigellosis, and cryptosporidiosis (Table 4.3). These findings would suggest a minimal role for this exposure.

6) Overseas travel: The frequency for overseas travel reported for campylobacteriosis notifications in 2002 was also near the bottom of the range at 7% (Table 4.2). Nevertheless, it was still higher than gastroenteritis (at 3%). For the 2004 data, it was non-significantly higher than yersiniosis but was significantly lower than salmonellosis and especially giardiasis (Table 4.3). These findings are consistent with this exposure playing a minimal role in the New Zealand setting.

Table 4.2: Percentage* of enteric disease cases reporting exposure to key selected risk factors (based on 2002 data (Sneyd & Baker, 2003) and ranked by frequency of food premise contact)

| | Risk category | | | | | | | | | |
| Disease | Food and water contact | | | Animal contact | | | Human contact | | | Travel |
	Food premise[1]	Un-treated drinking water	Recreation-al water	Farm animals	Sick animals	Faecal matter, vomit	Other Symp-tomatic case[2]	Con-firmed case[3]	School, Child-care[4]	Over-seas
Gastroenteritis	88%	6%	7%	8%	1%	10%	41%	67%	9%	3%
Campylo-bacteriosis	**54%**	**20%**	**17%**	**33%**	**7%**	**12%**	**12%**	**6%**	**16%**	**7%**
Salmonellosis	50%	24%	18%	29%	6%	16%	17%	12%	28%	14%
Typhoid	50%	67%	33%	18%	11%	30%	22%	40%	23%	67%
Shigellosis	50%	23%	14%	14%	0%	10%	13%	15%	18%	49%
Yersiniosis	45%	19%	20%	30%	3%	14%	8%	3%	21%	6%
Paratyphoid	44%	30%	21%	14%	8%	27%	15%	10%	33%	68%
Giardiasis	33%	36%	33%	26%	5%	46%	37%	31%	35%	17%
Crypto-sporidiosis	32%	39%	35%	59%	23%	31%	27%	15%	46%	7%

* The percentage is total number of cases exposed divided by total number of cases for whom this information was recorded.
[1] Case consumed food at a food premise during the incubation period.
[2] Case had contact with other symptomatic case during incubation period.
[3] Case had contact with a confirmed case during incubation period.
[4] Case attended school, pre-school or childcare.

Table 4.3: Additional analysis of reported risk factor exposure (within the incubation period) for different enteric diseases relative to campylobacteriosis (using 2004 data (ESR, 2005b))

Notified enteric disease	Total notifications	% reporting the risk factor	Numbers reporting this risk factor and the denominator **	Rate ratio (95%CI); p-value*
Food consumption from a food premise				
Campylobacteriosis	12,213	49.8%	1445/2899	1.0 (reference)
Yersiniosis	420	42.6%	55/129	0.86 (0.70 – 1.05)
Salmonellosis	1080	42.4%	212/500	0.85 (0.76 – 0.95); p = 0.002
Giardiasis	1515	33.2%	126/380	0.67 (0.57 – 0.77); p<0.0000001
Shigellosis	140	30.1%	22/73	0.60 (0.43 – 0.86); p = 0.0009
Cryptosporidiosis	612	26.1%	66/253	0.52 (0.42 – 0.65); p<0.0000001
Untreated drinking water				
Campylobacteriosis	12,213	21.1%	626/2970	1.0 (reference)
Salmonellosis	1080	23.1%	134/579	1.10 (0.93 – 1.29)
Yersiniosis	420	25.7%	39/152	1.22 (0.92 – 1.61)
Giardiasis	1515	32.7%	143/437	1.55 (1.33 – 1.81); p<0.0000001
Shigellosis	140	35.9%	28/78	1.70 (1.26 – 2.31); p = 0.002
Cryptosporidiosis	612	47.4%	165/348	2.25 (1.97 – 2.56); p<0.0000001
Exposure to recreational water				
Campylobacteriosis	12,213	14.1%	467/3301	1.0 (reference)
Salmonellosis	1080	14.2%	90/635	1.00 (0.81 – 1.23)
Yersiniosis	420	22.6%	38/168	1.60 (1.19 – 2.14); p = 0.002
Giardiasis	1515	33.2%	170/512	2.35 (2.02 – 2.72); p<0.0000001
Shigellosis	140	15.4%	12/78	1.09 (0.64 – 1.84)
Cryptosporidiosis	612	25.9%	89/343	1.83 (1.51 – 2.23); p<0.0000001
Contact with farm animals				
Campylobacteriosis	12,213	30.9%	1071/3461	1.0 (reference)
Salmonellosis	1080	28.9%	205/710	0.93 (0.82 – 1.06)
Yersiniosis	420	37.0%	74/200	1.20 (0.99 – 1.44)
Giardiasis	1515	26.3%	141/537	0.85 (0.73 – 0.99); p = 0.03
Shigellosis	140	5.4%	4/74	0.17 (0.07 – 0.45); p<0.000002
Cryptosporidiosis	612	71.7%	302/421	2.32 (2.14 – 2.51); p<0.0000001

Notified enteric disease	Total notif-ications	% reporting the risk factor	Numbers reporting this risk factor and the denominator **	Rate ratio (95%CI); p-value*
Contact with faecal matter				
Campylobacteriosis	12,213	12.2%	387/3177	1.0 (reference)
Salmonellosis	1080	14.6%	98/671	1.20 (0.98 – 1.47)
Yersiniosis	420	20.8%	38/183	1.70 (1.27 – 2.30); p = 0.0007
Giardiasis	1515	37.3%	193/518	3.06 (2.64 – 3.54); p<0.0000001
Shigellosis	140	23.6%	17/72	1.94 (1.27 – 2.97); p = 0.004
Cryptosporidiosis	612	24.0%	94/391	1.97 (1.62 – 2.41); p<0.0000001
Overseas travel				
Campylobacteriosis	12,213	7.3%	283/3871	1.0 (reference)
Salmonellosis	1080	21.8%	174/800	2.98 (2.50 – 3.54); p<0.0000001
Yersiniosis	420	5.0%	11/218	0.69 (0.38 – 1.24)
Giardiasis	1515	38.8%	19/49	5.30 (3.67 – 7.67); p<0.0000001
Shigellosis	140	NR	NR	-
Cryptosporidiosis	612	NR	NR	-

* Analysis using Statcalc in EpiInfo by this reviewer.
** The denominator was the number of notifications for which information was recorded.
NR = not reported in ESR's Annual Report.

Comparisons with other countries

ESR's Annual Reports for 2002 and 2003 (but not the 2004 one) had comparisons with notification rates from Australia and Canada. These showed New Zealand rates being several times higher than in these two countries. Although country comparisons have many methodological limitations, the data set for comparisons has been expanded in the table below (Table 4.5). The comparisons still suggest that New Zealand has a particularly high rate of notified campylobacteriosis. Although this might be entirely or partly due to higher levels of diagnosis and reporting, the pattern is compatible with the New Zealand rates being truly higher. However, the implications for aetiology from such international comparisons are unclear. Further analysis could potentially consider comparing food contamination levels and water supply contamination levels between countries. A particular characteristic of New Zealand is the relatively high livestock population and how it generally grazes outside on pasture (relative to living in barns for much of the year). This in turn may influence water contamination levels – given that "microbial contamination of lakes and rivers is widespread" in this country (Parliamentary Commissioner for the Environment, 2004). New Zealanders may also spend relatively more time in outdoor recreational

pursuits than for some other populations (but no relevant data comparisons were identified in this review).

Table 4.5: Country comparisons in crude rates of notified campylobacteriosis

Country	Year	Crude notification rate per 100,000 population	Reference
New Zealand	2004	326.8	(ESR, 2005b)
Australia	2003	116.5	(Miller et al, 2005)
Iceland	2000	116	(Stern et al, 2003)
England and Wales*	1999	103.7	(Gillespie et al, 2002)
Scotland	2003	86.6	(Scottish Centre for Infection and Environmental Health, 2004)
Canada**	2000	40.1	(Public Health Agency of Canada)
Netherlands	2001	37.0	(van Pelt et al, 2003)
18 European countries***	1999	2.9 to 166.8	(Takkinen et al, 2003)
United States (FoodNet sites only)	2003	12.6	(Centers for Disease Control and Prevention, 2004)

* Data since this year (up to 2004) indicate declining numbers of laboratory isolates (Health Protection Agency: http://www.hpa.org.uk/infections/topics_az/campy/data_ew.htm).
** Although no more recent rates are available, the numbers of notified cases were lower in the years 2002 and 2003 (http://www.phac-aspc.gc.ca/publicat/ccdr-rmtc/04pdf/cdr3021.pdf).
*** Includes other countries in this table: Iceland, Netherlands and the UK.

Discussion

Summary of findings: The notification data have various limitations – particularly around the completeness of some of the risk factor information. Also, comparison with the hospitalisation data suggests that there are important limitations with the notification data concerning ethnic-group specific rates and with District Health Board (DHB) rates (the variation in the latter probably having a large surveillance artefact component).

The notification data does not provide any strong evidence concerning the aetiology of campylobacteriosis in New Zealand. However, it provides limited evidence in a number of areas as summarised in Table 4.6.

Table 4.6: Information on campylobacteriosis aetiology provided by notification data and associated analyses

Data	Findings of relevance to informing aetiology
Time trends	There is evidence that the increase over time in national notification rates is real and it is likely to reflect changes in risk factors or exposure levels. However, there is a lack of trend data for most plausible risk factors – except for a near doubling of poultry consumption during the 1990s in real terms and a trend of increasing expenditure on takeaway foods.
Seasonality	The seasonality of notifications with a summer peak is suggestive of either various environmental risk factors or else summer-specific behaviours (but the specific possibilities are large in number). There is weak evidence around temperature factors playing some role (when NZ data are considered along with that from other countries).
Rural / urban distribution	For children (< age 15 years) there appear to be risk factors associated with rurality. This may suggest some role for animal contact (though the possibilities for other specific risk factors are large).
Regional distribution	There may be regional differences in rates that relate to climatic, environmental factors or behavioural factors. However, the lack of correlation between notifications and hospitalisations is suggestive of such differences comprising a large surveillance artefact.
Age distribution	The variation in notification rates by age group is suggestive that aetiological factors are likely to vary accordingly (though for the younger age groups there may be important immunological factors).
Risk factor data – food	The data on the consumption of food from a food premise* is consistent with this being a relevant risk factor for campylobacteriosis. Some data of limited value are suggestive of specific foods such as chicken having a role (data from Christchurch and national data on the "probable source").
Risk factor data – water	The data on untreated water consumption* is consistent with this risk factor having some possible role (but probably not a large one). But the role of exposure to recreational water appears minimal.
Risk factor data – animal contact	The data on "farm animal" contact* is consistent with this risk factor having some modest role. The pattern for "sick animal" contact was similar. Some data of limited value are suggestive of contact with dogs increasing risk for children (Christchurch data).
Risk factor data – human contact	The data on "human contact"* is consistent with this risk factor having a minimal role.
Risk factor data – travel	The data on "overseas travel"* is consistent with this risk factor having a minimal role.
Country comparisons	It is difficult to interpret the higher burden of notified campylobacteriosis relative to other countries – but if it is indeed true it could suggest that certain country-specific risk factors are relevant.

* In the context of the results for other notifiable enteric diseases using a case-case analysis.

Possible implications for the current surveillance system and data analysis: The information provided is suggestive that it is valuable to continue to obtain notification data on all cases of campylobacteriosis. However, the following could be considered:

1. ***Data completeness (sentinel sites):*** Data completeness could be improved eg, perhaps in one or more sentinel DHBs where a serious attempt is made to achieve high (>90%) completeness for all data fields for campylobacteriosis notifications. These DHBs could be provided with additional resources to facilitate achieving this more intensive surveillance eg, funded by the Ministry of Health and New Zealand Food Safety Authority (NZFSA). In such areas, consideration could be given to evaluating remuneration systems to notifiers since medical practitioners used to be reimbursed for notifications. In addition or alternately, in each DHB a random sample (eg, of 5-10% of cases) could have more intensive investigation. Then at a national level, this intensive sample could be analysed separately from the other notified cases.

2. ***A revised (more minimal) standard data set:*** To assist with improving nationwide data quality and completeness a more minimal data set could be used for notified cases of campylobacteriosis. This approach may also free up some public health worker time (eg, to allow more intensive investigation of large outbreaks). The focus of the new standard data set could be on obtaining basis demographic data, the occupation (eg, to identify food handlers), and whether or not the case may be linked to a common function or event. There has been some useful work around standard data sets for enteric disease reporting in Scotland (Cree et al, 2001) and England (Rooney et al, 2000) that could inform further developments in New Zealand.

3. ***Data quality (national):*** To assist with improving nationwide data completeness, ESR could provide an annual feedback report on how each PHU ranks nationally in terms of completing critical data fields for key enteric diseases. Each PHU could also be ranked according to the extent to which notifiable enteric diseases are laboratory reported.

4. ***Laboratory-based surveillance:*** Greater use could be made of laboratory-based surveillance throughout the country. For example, an audit in Auckland has indicated the potential benefits of the greater use of laboratory-based notification (Simmons et al, 2002c). The new Public Health Bill may facilitate the greater use of laboratory-based notification.

5. ***National level analysis:*** Although the current national level analysis is of a high quality, there could be more comparisons made in the Annual Reports between notification data and hospitalisation data (perhaps every few years). Similarly it should be stated in these reports that the variations in notification rates at the DHB level are probably largely a surveillance artefact. Further in-depth analysis of the data could be considered (especially if there was improved completeness of data in an area in which intensive surveillance was conducted). Of particular interest would be analyses of specific foods consumed and occupational data (with comparisons made with other notified enteric diseases).

6. ***Risk factor comparisons (case-case studies):*** Regular comparison between risk factors for the different enteric diseases should be routinely undertaken (as per Tables 4.2 and 4.3). However, improved levels of data collection would improve the validity of such analyses.

Possible research implications: Undertaking more intensive campylobacteriosis surveillance in one or more DHBs (as detailed above) could be combined with various research objectives. This work could better calibrate the quality of the surveillance system for campylobacteriosis – especially if the DHB had all laboratories participating in notifying campylobacteriosis and doctors were particularly encouraged to have a low threshold for conducting laboratory tests on cases with diarrhoea.

Intensive surveillance in one area would also be helpful if any intervention studies were planned (eg, intensive actions to lower *Campylobacter* contamination levels in poultry).

The work to date is suggestive that the analysis of campylobacteriosis hospitalisation data (especially when analysed in conjunction with notification data) is of value. This area could be explored further, especially to develop more accurate information on the spatial distribution of campylobacteriosis in New Zealand. However, the limitations of the quality of hospitalisation data for campylobacteriosis should also be explored in an additional study. This is because the diagnosis might not always be properly recorded as it may be made after the person has left hospital (eg, after being admitted with dehydration secondary to severe diarrhoea).

Summary

The notification data from the national surveillance system have various limitations (particularly around the completeness of some of the risk factor information). Although the notification data has a number of uses (eg, indicating the size of the burden to public health), it generally only provides limited quality evidence concerning the aetiology of campylobacteriosis in New Zealand. This evidence is suggestive of the likely heterogeneity of aetiological factors for different age groups, by rurality, by seasonality and the likelihood of changing risk factors or exposure levels (to account for the increasing trend over time). Comparison with data for other notified enteric diseases provides some limited evidence around the likely role of different risk factors (ie, the apparent importance of food relative to: water, animal contact, human contact and overseas travel). Fortunately, there is scope for further improvements in the surveillance system and potential ways to enhance the research agenda into this disease.

5 Review of Outbreak Investigation Reports

Background

Outbreaks of campylobacteriosis in New Zealand have been described in the literature since the 1980s. A review of published and unpublished outbreak investigations relating to human campylobacteriosis (based on the literature search detailed in the methods section) is conducted in this section. The search strategy for identifying these reports is detailed in the methods section.

These outbreaks were considered in the context of other published outbreak reports for enteric diseases in New Zealand: (Stefanogiannis et al, 2001; Hill et al, 2002; Thornley C. N. et al, 2003).

Findings – Published Reports

A total of 14 publications were identified in the literature: (Brieseman, 1984; 1987; Stehr-Green et al, 1991; Health Protection Programme - Hawke's Bay Area Health Board, 1992; Jarman & Henneveld, 1993; Mitchell R. et al, 1993b; Bohmer, 1997; McElnay, 1997; Calder et al, 1998; Whyte et al, 2001; Health Protection - Choice Health Wairarapa, 2002; McElnay & Inkson, 2002; Simmons et al, 2003). In some cases these publications described outbreaks at multiple sites (Brieseman, 1984) or multiple outbreaks affecting different groups visiting the same site eg, (Health Protection Programme - Hawke's Bay Area Health Board, 1992; Bohmer, 1997).

One outbreak was described in two publications ie, the outbreak described by Stehr-Green et al was also described in: (Centers for Disease Control, 1991). These 13 outbreak investigations are reviewed below in chronological order of publication date.

Outbreaks at two camp sites – Christchurch (Brieseman, 1984)

Implicated risk factor: The investigation concluded that "circumstantial evidence implicates raw milk as the cause of the illness". However, the hazard posed by inadequately treated or untreated stream water used by the camps was also noted.

Key findings: The investigation collected data from 15 separate camp events at two separate sites.
* *Campylobacter* was cultured from the faeces of 30 cases (attending Camp 1) and 29 cases (attending Camp 2).

- The time courses of the outbreaks were not described in detail but appeared to be suggestive of point source outbreaks. However, subsequent intra-family transmission of campylobacteriosis was also described (Camp 1).
- For Camp 1 the water supply was from a stream and was "irregularly and unsatisfactorily chlorinated". For Camp 2 the water supply was untreated stream water. Faecal coliforms were detected in the water supply for both camp sites.
- For Camp 2 food there was no refrigeration and food was stored in a chilly bin or sometimes in large bowls in the stream.
- The only reported animal contact was a visit to a dog-dosing strip (only for one camp event).
- Raw milk was supplied to some of the camp events at both camp sites. The data reported indicate that only children and no adults reported illness (but the denominator data presented were incomplete). Laboratory testing found no *Campylobacter* in the raw milk supplied to both camp sites.

Assessment (re aetiology): The epidemiological aspects of this investigation were limited in that it collected group-level rather than individual-level information. There were also no statistical analyses. Indeed, analysis of the group-level data by this reviewer does suggest that raw milk supply to a camp event was associated with the occurrence of any reported illness among children at a camp event when considering data from all 15 camp events (RR = 5.71, Fisher exact test p-value = 0.041). However, for diagnosed campylobacteriosis this association was not statistically significant (p = 0.08). There were also no cases of campylobacteriosis reported for camp events in which no raw milk was used.

Although questionnaire data on food, water and milk consumption was lacking, the absence of any cases of reported illness among adults (who probably were less likely to drink milk except in tea) also favours raw milk as the source of these outbreaks (relative to water). However, adults may also have consumed less untreated water (if they tended to consume hot beverages). While the evidence is incomplete, the conclusion of the author that raw milk was the likely cause of these outbreaks appears reasonable. Nevertheless, untreated water is a fairly plausible alternative, given the results of the microbiological testing.

Outbreak in a rural town (Brieseman, 1987)

Implicated risk factor: The investigation concluded that: "epidemiological evidence indicates contamination of the town water supply as the likely source".

Key findings:
- The epidemic curve was fairly consistent with a point source outbreak (after the two-week period there was a return to the background level of notifications).
- The onset of the outbreak was concurrent with reports of the discolouration of the borough water supply, consequent upon heavy rains (which caused flooding in nearby townships).
- The cases were distributed throughout the borough and included four children and 15 adults.

- Questionnaire data was reported to show no relationships with animal contact or food consumption (but there were no data obtained on controls and no statistical information was provided).
- All the cases reported consuming water though the precise details were not all provided (eg, untreated versus boiled and the daily amounts).
- Investigations determined that the river water is not routinely chlorinated and that there was a period (half a day) after the heavy rain before the chlorination was commenced (in the days just preceding the outbreak). There was also a new infiltration gallery in the water supply system that was considered to lack adequate filtering mechanisms (ie, lack of an overlying grass sward). Tests on the water after chlorination commenced indicated a high coliform count, indicating heavy faecal contamination. However, no *Campylobacter* was tested for.

Assessment (re aetiology): This investigation included a site investigation, laboratory data and some epidemiological information (but this lacked control group data and did not consider the results statistically). Nevertheless, when considering all the available information obtained (including the microbiological evidence for water contamination), it appears that the author's conclusions about the town water supply being the most likely cause, are reasonable.

Outbreak at a camp near Christchurch (Centers for Disease Control, 1991; Stehr-Green et al, 1991)

Implicated risk factor: The investigation "strongly suggested the water supply as the source of infection". There was no evidence suggestive of other causes.

Key findings:
- The epidemic curve was strongly suggestive of a point source outbreak that began a few days after "heavy rains".
- *Campylobacter jejuni* was cultured from the faeces of 25% of the cases (n = 11).
- The presence of high coliform counts of the water from all four springs that supplied the camp was highly suggestive of faecal contamination of the water supply. The water was not chlorinated or filtered before use.
- Cases drank more unboiled water than unaffected persons (p = 0.03), and were more likely to have drunk ≥ 2 cups of unboiled water a day, p < 0.01). No associations between eating food from the camp or for farm animal and pet animal contact were detected.
- There was evidence of a potential contamination source in that "a large number of farm animals grazing around the area was the probable source".
- No further cases associated with the camp were reported after control measures to improve the water supply were enacted.

Assessment (re aetiology): This was a very thorough investigation using both epidemiological and microbiological methods. Although *Campylobacter* was not actually identified in the untreated water supply, the authors' conclusions as to the most likely cause of the outbreak being the water supply appear to be fairly well justified.

Children's camp in Hawke's Bay (Health Protection Programme - Hawke's Bay, 1992)

Implicated risk factor: No definite source could be identified. However one identified hazard was untreated bore water.

Key findings:
- *Campylobacter* was cultured from 21% of the stool specimens (from 56 people).
- The time distribution of cases was suggestive of two separate groups being both exposed to a point source.
- The bore water supply to the camp was not treated at the time of the outbreak.
- Cases, on average, consumed significantly *fewer* cups of water than those who did not become ill (p = 0.01). But there were no other statistically significant associations found.
- Most of the water supply specimens indicated faecal coliforms (but *Campylobacter* was not isolated).
- No further cases were reported after discontinuation of the bore water supply and flushing and super-chlorination of the reticulation system.

Assessment (re aetiology): This investigation used both epidemiological and microbiological methods. While the findings of the two methods were contradictory – the overall evidence is somewhat compatible with the untreated water supply being involved.

Camp in Northland (Jarman & Henneveld, 1993)

Implicated risk factor: No definite source could be identified. Nevertheless, plausible risk factors included the consumption of unsafe water and raw milk consumption.

Key findings:
- *Campylobacter jejuni* was cultured from the faeces of six cases.
- The time distribution was suggestive of a point source outbreak.
- The water supply to the kitchen was untreated roof water and all water samples contained significant amounts of faecal coliform contamination (though *Campylobacter* organisms were not isolated).
- Water supply to the kitchen was untreated roof water and all water samples contained significant amounts of faecal coliform contamination (though *Campylobacter* organisms were not isolated). The odds ratio (OR) for drinking river water on the overnight camp was raised – but it was not statistically significant (OR = 4.0, 95%CI = 0.78 – 22.6).
- Raw milk was supplied to the camp and the only significant odds ratio for food consumption was for cereal and milk for breakfast (OR = 10.4, 1.17 – 278.1). However, the OR for consuming a glass of milk was not significantly raised. *Campylobacter* was not identified in any of the milk samples. The

extent to which some of the milk was heat-treated on the camp could not be ascertained.

Assessment (re aetiology): This investigation used both epidemiological (actually a cohort study) and microbiological methods. The quality of the questionnaire information would however, have been limited by the four-week delay and the poor response to some of questions on the questionnaire. Both the consumption of unsafe water and raw milk are plausible causes of this outbreak – but the former may be more likely given the microbiological evidence for faecal contamination of the water supply.

Boarding school – Christchurch (Mitchell R. et al, 1993b)

Implicated risk factor: This investigation did not identify a likely cause.

Key findings:
- *Campylobacter jejuni* was cultured from the faeces of five of the cases.
- The time distribution was strongly suggestive of a point source outbreak – with some evidence suggestive of a lunch event on a particular day. Cases were confined to those who had eaten food in the dining hall.
- There were no statistically significant differences between cases and non-cases in the food or water they drank.
- The only food hygiene problem identified was that sparrows had ready access to the dining hall and kitchen. The inspection of the premises of the meat wholesaler that supplied the school did not identify any problems.
- The school had its own well for water supply but a water sample from the kitchen tap indicated no bacteriological contamination. Well water was considered an unlikely source of infection since it also supplied the day-student population who were largely unaffected.

Assessment (re aetiology): This investigation appears to have been fairly thorough and it made use of both epidemiological (cohort study) and microbiological methods. The contamination of food by birds is plausible given the data from the UK on transmission to humans from birds consuming milk from the tops of bottles (Riordan et al, 1993). However, other causes are quite possible such as the contamination of a food item (eg, the mince tacos consumed at the suspected lunch event) and inadequate cooking of this food. This outbreak investigation also provided no evidence for or against there being an infected food handler involved in the kitchen (though this is considered to be a relatively rare cause of campylobacteriosis outbreaks).

School holiday camp near Christchurch (Bohmer, 1997)

Implicated risk factor: The investigation "identified a contaminated water supply as the most likely source of infection". There were some deficiencies with the hygiene of the camp kitchen but no evidence of food safety deficiencies or of specific foods being implicated.

Key findings:
- The two epidemic curves for the two groups were strongly suggestive of a point source outbreak.
- *Campylobacter jejuni* was cultured from the faeces of five out of the six cases providing specimens.
- A positive association was found for drinking camp water as a risk factor (RR = 1.51, 95%CI = 1.07 – 2.12).
- The presence of high coliform counts was detected in the camp water and *Campylobacter* was detected in the stream water.
- There was evidence of water supply deficiencies including back-flow of stream water into the camp supply at times of high demand.
- The presence of high coliform counts was detected in the camp water and *Campylobacter* in the stream water.
- There were no further cases associated with the camp after the drinking water was routinely boiled.

Assessment (re aetiology): This investigation was thorough and used epidemiological (cohort study), site inspection and microbiological methods. Although the risk ratio was not particularly strong, the author's conclusions as to the most likely cause of the outbreak being the water supply appear to be reasonable.

Community outbreak in Havelock North (McElnay, 1997)

Implicated risk factor: The investigation did not identify a specific source. However, a single food outlet was considered the most likely source given the predominance of a single *Campylobacter* serotype.

Key findings:
- The epidemic curve for cases from which *Campylobacter* serotype 23,26 was isolated, was strongly suggestive of a point source outbreak.
- The cases ranged in age from 4 to78 years and the majority lived in Havelock North.
- The public water supply was "assessed" and considered to be an unlikely source.
- *Campylobacter* was cultured from the faeces of 15 cases (with 14/15 being serotype 23,26).
- Food outlets considered to "present the highest risk" were assessed and food safety standards were discussed with staff (but details of the findings were not covered in this brief report).

Assessment (re aetiology): This investigation used epidemiological, site inspection and fairly sophisticated microbiological methods. Although a specific source was not identified, the author's conclusions as to the most likely cause of the outbreak being a food outlet seem reasonable.

Community outbreak in Auckland (Calder et al, 1998)

Implicated risk factor: The investigation "failed to find an unequivocal source of the epidemic". However, there was some reasonable microbiological and (limited) epidemiological evidence suggestive that poultry consumption was involved.

Key findings:
- The epidemic curve is fairly suggestive of a point source outbreak.
- *Campylobacter* was cultured from the faeces of 66 cases and in 73% (n = 48) of these *C. jejuni* serotype 1 was identified. Preliminary DNA macrorestriction analysis by PFGE (pulsed field gel electrophoresis) indicated that all serotype 1 isolates were closely related. As there was evidence that this strain is usually unknown at the national level and in Auckland – it was surmised to be the outbreak strain.
- A case-control study using friends or workmates as age, ethnicity and gender matched controls was conducted (48 cases and 106 controls).
- There were no statistically significant findings. There was weak evidence of a link with chicken consumption (OR = 1.79, 95%CI = 0.53 – 7.83).
- Microbiological testing of chicken meat and liver samples from retailers usually patronised by the cases indicated *Campylobacter* in around half (10/21) of samples. Also, six of the isolates were serotype 1 with DNA macrorestriction analysis profiles closely related to those of the serotype 1 isolates from the cases (with such serotype 1 isolates not previously having been isolated from poultry sources).
- There was no evidence that the electrical power shortages, that were occurring in Auckland at the time, were involved (only two cases reported eating food purchased in the area affected by the power shortages).

Assessment (re aetiology): This investigation used epidemiological methods (with a very detailed questionnaire and use of a case-control methodology). The microbiological methods were also relatively advanced. The control selection process may have contributed to the lack of findings (given that the approach taken poses risks of over-matching). Nevertheless, the overall evidence (and particularly the laboratory evidence) is fairly suggestive that poultry consumption was implicated in this outbreak.

Christchurch restaurant (Whyte et al, 2001)

Implicated risk factor: The investigation "revealed that the most likely cause of contamination was the under cooking of chicken livers used to prepare the paté."

Key findings: The investigation covered two groups who had visited the restaurant on consecutive evenings (with the same set meal provided).
- *Campylobacter jejuni* was cultured in eight out of the nine faecal specimens provided.
- The time distribution of illness was not adequately described but was somewhat suggestive of a point source outbreak.
- All but one of the people consuming the chicken liver paté reported illness.

- The investigation revealed that the same batch of chicken liver paté was used on both nights and was prepared on site by the sous chef. Discussions with this chef were suggestive that the livers were undercooked and that no temperature measurement had been used to determine this.
- A sample of paté obtained tested negative for *Campylobacter*. However, as the specimen smelt "off", there could have been out-competition by spoilage organisms.

Assessment (re aetiology): This investigation inadequately documented the epidemiological findings and did not analyse the results statistically. Analysis of the data for paté consumption by this reviewer, indicated that it was strongly associated with reported illness ($p < 0.00001$ using Statcalc in EpiInfo). However, data regarding the other six menu items (including chicken breasts) was not analysed (ie, the detailed data were not presented in the report).

Although this investigation was sub-optimal in some ways, the overall suggestion that the chicken liver paté was involved seems reasonable when considering all the evidence collectively. Furthermore, it is consistent with the evidence that the authors presented concerning other outbreaks associated with undercooked chicken livers in both Christchurch and Auckland (see Section 6). The MAGIC study also reported a statistically significant associated with chicken liver consumption (Eberhart-Phillips et al, 1997). Various microbiological studies have also report the presence of *Campylobacter* in chicken livers at high levels (as reviewed by Whyte et al).

Boarding school in Hawke's Bay (McElnay & Inkson, 2002) (Public Health Unit: Hawkes Bay, 2001)

Implicated risk factor: The investigation implicated the school's water supply.

Key findings:
- *Campylobacter* was cultured from the faeces of two students.
- The questionnaire data collected on all pupils and staff revealed no particular associations with exposures (data on water consumption were not provided in this abstracted report or in the unpublished report obtained). The outbreak was not restricted to any particular class or hostel.
- The time course of the outbreak involved an abrupt increase in cases (but was not precisely detailed).
- The school was found to have its own water supply, "a spring which drained into a swamp where cattle frequently graze the surrounding area." The ultraviolet treatment of the water supply was on site, but the system had malfunctioned and was not repaired for 3-4 days. The failure date on or about 18 May was just prior to 21 May when a substantial increase in diarrhoeal illness was reported. Furthermore, the bulb in the ultraviolet treatment system was past its replacement date and the "plant sleeves" needed cleaning.
- *Campylobacter* organisms were isolated from the water both pre- and post-ultraviolet light treatment, from cattle faeces around the water source, and from sewage effluent from the school. DNA/PCR assay confirmed that these organisms were the same strain of *C. jejuni* (though human faecal specimens were not assayed). Subsequent testing of the water system over the next four

months also revealed *E. coli* and total coliforms on several occasions (possibly due to a mistake being made with the opening of a valve).

Assessment (re aetiology): This investigation used both epidemiological (cohort study) and high quality laboratory investigative methods. Given all the information available, it is indeed highly probable that this outbreak was related to consumption of untreated water.

Christmas function – Wairarapa (Health Protection - Choice Health Wairarapa, 2002)

Implicated risk factor: The investigation reported that the "most likely sources of the outbreak was thought to be either the sliced roast pork with gravy dish or untreated roof water supply". A number of hazardous food handling practices were identified that could have contributed to the outbreak.

Key findings:
- *Campylobacter* spp. were cultured from four out of the five specimens.
- The questionnaire data revealed a significant association with consumption of the pork dish, but not for any of the other food items in the buffet (including the chicken) or for water consumption.
- A HACCP analysis was performed on the site and it identified several deficiencies in food handling (including possible time/temperature abuse of the pork roast, inappropriate use of untreated water for food preparation etc).
- None of the pork meal was available for sampling.
- Analysis of the roof water sample showed faecal coliform contamination.

Assessment (re aetiology): This investigation used both epidemiological (cohort study), site investigation and food safety assessment, and microbiological methods. The statistical analysis of the questionnaire data was slightly sub-optimal and recalculation of the results by this reviewer gave a stronger association with the pork dish (Fisher exact, p-value = 0.002, using Statcalc in EpiInfo). Nevertheless, the available evidence does not strongly favour this food item over the possibility of contaminated water use from a roof supply (given the available New Zealand data that 38% of roof water supplies tested found *Campylobacter* (Savill M. G. et al, 2001)).

School camp in the Waikato (Simmons et al, 2003)

Implicated risk factor: Untreated roof water linked to the roosting of feral turkeys on the roof used for water catchment was considered to be the most likely cause of the outbreak. Multiple other plausible risk factors were identified but considered to be less likely causes (ie, animal contact, drinking raw milk, swimming in a local stream, access of domestic pets to the food preparation area, time/temperature abuse of perishable food on the survival adventure at the camp, potential exposure to a sewage effluent pond, the generally poor hand hygiene of the campers, and the lack of a protocol for hand hygiene on the "survival adventure").

Key findings:
- The epidemic curve was strongly suggestive of a point source outbreak with *Campylobacter jejuni* in the faeces of many of the cases (n = 18).
- The presence of *Campylobacter* spp. in water from the kitchen tap.
- The lack of any monitoring of the water supply by those operating the camp.
- Virtually all those using the camp were exposed to the roof-collected rainwater supply in some way (and there was a raised RR for consumption of roof-collected rainwater in plastic containers (RR = 4.04, 95%CI = 0.62 – 26.33)).
- There was evidence of a contamination source for the roof water (ie, both feral turkeys and also doves).
- Analysis of the questionnaire data did suggest significant associations with certain foods, exposure to horses and recreational exposure to stream water. However, the authors provided arguments to suggest that confounding by age may explain some of the statistically significant results (ie, the proportion of adults who were cases was lower than for the children).

Assessment (re aetiology): This was a very thorough investigation using epidemiological (cohort study), site inspection and laboratory methods. The authors' conclusions as to the most likely cause of the outbreak appear to be fairly well justified. The conclusion that contaminated roof water was involved is consistent with other New Zealand data which indicates that *Campylobacter* spp. have been found in 38% of roof water supplies tested (Savill M. G. et al, 2001). It is also consistent with the international literature on *Campylobacter* infection in turkeys (Wallace et al, 1998).

Findings – Unpublished Reports

The request sent to key informants resulted in the provision of a number of unpublished outbreak investigation reports. Out of these, the ones that involved relatively high quality investigative methods were examined (ie, a case-control study, or a cohort study, or laboratory testing of the suspected source for *Campylobacter*). The findings and comments on study quality are summarised below in chronological order of outbreak year.

Family barbecue – Auckland (Bishop, 1998)

Implicated risk factor: The epidemiological study indicated that chicken was the likely source, and there was information from cases to suggest that the chicken was undercooked.

Key findings:
- The epidemic curve was suggestive of a point source. The cases included 4 laboratory confirmed cases and 13 probable cases.
- The cohort study (17 cases and 29 non-cases) identified consumption of chicken as the only statistically significant risk factor (p < 0.05).
- Some cases reported that the chicken was undercooked in the second batch and they thought that this may have been due to the diminishing light or because the cook did not want to delay between the first and second helpings.
- No leftover food samples were available for testing.

Assessment (re aetiology): This investigation involved only epidemiological methods (a cohort study). There were some minor statistical errors in the analysis but recalculation by this reviewer for all the food items confirms that the chicken was the only statistically significant risk factor (p = 0.008, Fisher exact test). The report included discussion of potential recall bias given that "people are more likely to associate illness with chicken foods than any other foods". Nevertheless, recall bias is unlikely to explain chicken consumption being such a statistically significant finding and in the context of a barbeque being a more unusual event (relative to other meals during the week).

Auckland yachting team (Manning, 1999)

Implicated risk factor: The investigation concluded that the most likely source of infection was a lunch event involving kebabs.

Key findings:
- The epidemic curve was suggestive of a point source outbreak. There were 16 cases.
- Many cases did not eat breakfast at their hotel and evening meals were varied with few people sharing common meals.

- The cohort study of 40 respondents produced no statistically significant results. However, the highest risk ratios were for the tomato and cream pasta (RR = 2.81, 95%CI = 0.94 – 8.45, p = 0.09) and kebabs (RR = 2.50, 95%CI = 0.68 – 9.25, p = 0.14). It was noted however, that food recall was poor.
- It was reported that one batch of the kebabs were made early in the morning and eaten at lunch (and not refrigerated or chilled since this time). Also the circumstances in which they were made were outside of the usual routine for the staff of the food premises. A later batch of kebabs was prepared later in the morning.
- The investigators consider that the kebabs were a more likely source of infection than the vegetarian pasta meal (due to the former being described as "high risk").
- The relevant food premises were not inspected at the time of the investigation (due to a confidentiality agreement).

Assessment (re aetiology): This investigation used epidemiological methods (a cohort study) but noted poor food recall by respondents. The assessment that kebabs were the most likely source seems reasonable (and appears to this reviewer to be more compatible with the epidemic curve data than the pasta meal since most infections occur 2-4 days after exposure (Blaser & Allos, 2005)). Water is probably a very unlikely cause of this outbreak given that Auckland City has a treated water supply.

Boat cruise event – Auckland (Ma et al, 2000)

Implicated risk factor: No risk factors were specifically identified. However, potentially hazardous food handling appeared to have occurred and some of the chicken kebabs may have been undercooked.

Key findings:
- The epidemic curve was not described in detail, but appeared to be constrained in time. The cases included 5 laboratory confirmed cases and 4 probable cases.
- The cohort study (9 cases and 49 non-cases) identified juice as the only statistically significant risk factor (RR = 4.8, 95%CI = 1.6 – 14.5, p = 0.017). However, the juice was only consumed by 50% of the cases and a variety of different brands of juice were consumed. Other food items with elevated RRs were cheese, water, and chicken kebabs (eg, for the latter: RR = 1.6, 95%CI = 0.23 – 11.28, p = 1.0).
- Some respondents commented that some of the chicken kebabs appeared to be undercooked.
- No leftover food samples were available for testing.
- An onsite food safety inspection was not carried out, as this was a private function on a chartered boat. However, the crewman who cooked the meats was not a trained food handler and reported using the same tongs for both raw and cooked meats.

Assessment (re aetiology): This investigation involved epidemiological methods (cohort study) and limited information about the food handling processes. While it failed to identify a source, the investigation did identify inappropriate food handling and the potential undercooking of chicken kebabs.

School camp in Hawke's Bay in 2000

Implicated risk factor: No specific source was identified but environmental contamination associated with animals and contaminated lake water were considered to be possibilities.

Key findings:
- There were no significant findings from the epidemiological methods (detailed food questionnaire) or from the site inspection (food handling processes). However, it was noted that some animals at the camp had diarrhoea (though animal contact was not a statistically significant risk factor).
- The laboratory testing of the tap water was negative for *Campylobacter*.

Assessment (re aetiology): The documentation on this investigation was rather limited ie, in the form of a letter to a school principal from the Medical Officer of Health (McElnay, 2000). The investigators concluded that the likely source was not identified but that environmental contamination associated with animals and contaminated lake water were considered to be possibilities. Given the available evidence this appears to be a reasonable conclusion.

Institution in Canterbury (Smith, 2001)

Implicated risk factor: No risk factor was identified specifically but contaminated water considered the "most likely source". There was some evidence of secondary person-to-person spread.

Key findings:
- The epidemic curve showed two peaks, suggestive of a repeated point source.
- The cohort study identified higher risk for the residents of a particular building (p = 0.0002) and for particular volunteers (p = 0.0004) but not for any foods or water consumption (but specific details for the latter were not provided in the report). However, it was considered by the investigator that the questionnaire had design deficits (ie, not identifying a soft drink dispensing machine).
- The water supply to the institution had failed and water supply testing identified *E. coli* on two occasions. Testing a filter cartridge of a soft drink dispensing machine was negative for *E. coli* and *Campylobacter* (but this was after "shock chlorination" had been undertaken).
- In relation to this outbreak an additional case of campylobacteriosis in a preschooler was notified and this was assumed to be from person-to-person spread.

Assessment (re aetiology): This investigation involved the following methods: epidemiological (cohort study with 27 cases and 89 non-cases), site inspection, and laboratory testing. Although some of the detailed results were not available in the report, the conclusion that contaminated water was the most likely source appears reasonable given all the available evidence.

Organised multi-day farm walk (Choice Health, 2001)

Background: This outbreak was registered in one DHB and investigated by another.

Implicated risk factor: None specifically identified but there was microbiological evidence for faecal contamination of multiple water supplies.

Key findings:
- There were three confirmed cases and five possible cases (amongst two separate parties of walkers).
- It was consider impractical to conduct an investigation of the 114 walkers over a period of 17 days. There was no evidence of staff illness.
- It was identified that each "hosting owner" was involved in limited food preparation for the walkers in their own household kitchens. There was only one specific report of sub-optimal food handling (carrying fish on an extended car trip with no chilly bin).
- No formal hygiene training had been undertaken by any of the food preparers.
- Streams that people may take water from are along the walk. However, stock apparently had "access to most areas".
- Water testing at one lodge with a rainwater tank supply found that it had a "moderate to high level of contamination" at the kitchen tap (total coliforms and *E. coli* but *Campylobacter* could not be isolated). Various sites at another lodge showed high levels of total coliforms and *E. coli* (though testing for *Campylobacter* was not done at these other sites).

Assessment (re aetiology): The investigation used only limited epidemiological methods, a limited inspection of sites and microbiological testing of water supplies. Given the presence of contaminated water at some sites (even though *Campylobacter* was not detected), this is may be the most likely source of the outbreak.

Restaurant in a town near Auckland (Zhu & Callaghan, 2002)

Implicated risk factor: None specifically identified but there was some limited supportive evidence for both: (i) a role for undercooked chicken; and (ii) the use of contaminated water during food preparation.

Key findings:
- The epidemic curve was highly suggestive of a point source on the day of a particular buffet dinner (involving six laboratory confirmed cases and 18 probable cases).
- The cohort study (22 cases and 40 non-cases) did not find any statistically significant risk factors. However, some foods had elevated risks of over 1.5 ie, the roasted vegetable medley, the chicken seasonal vegetables, and the carvery roast chicken with the highest relative risk (RR = 1.88, 95%CI = 0.97 – 3.62). But for water consumption the RR was only 1.10 (95%CI = 0.56 – 2.18).
- Laboratory analysis of a raw chicken sample isolated *C. jejuni*.

- Three water samples from the building's water exceeded the Maximum Acceptable Values for both total coliforms and *E. coli* and *C. jejuni* was also isolated in two samples (water supply was roof catchment and bore water and was filtered through two 5 micron filters). The bore water also contained coliforms. Swab samples from a cutting board and carvery knife were negative for *Campylobacter*.
- A HACCP assessment was carried out on the foods prepared for the buffet and it indicated that temperature monitoring did not occur (so the premises were unable to verify if the chicken was sufficiently heated internally). Wooden cutting boards were also not disinfected between uses and were not designated (and similarly for the knives). It was reported that ice water was used for cooling down the seasonal vegetables after cooking, and that untreated water was used to wash the vegetables, fruits and the defrosted chicken (in the suspect meal).

Assessment (re aetiology): This investigation involved epidemiological methods (cohort study with a high response rate); site inspection and HACCP assessment, and laboratory testing. While no specific source was definitively identified, there appears to be some supportive evidence for both: (i) a role for undercooked chicken; (ii) contaminated water used during food preparation.

Investigation into a mid-winter increase in campylobacteriosis in Auckland (Simmons et al, 2002b)

Background: This investigation was undertaken in response to an unusual winter peak of campylobacteriosis notifications in May 2002 in Auckland (over twice the average rate for the previous two years). This mid-winter increase was also reported in "most health districts of the North Island".

Implicated risk factor: The statistically significant increase over time in the same Penner serotype (HS 1,44) amongst both human cases and poultry isolates is suggestive that poultry exposure was partly involved in this mid-winter increase in notified cases.

Key findings:
- The epidemic curve for the month of June was suggestive of a mix of point source and continuous source.
- The investigation focused on 30 consecutive cases of campylobacteriosis that were identified by an Auckland laboratory.
- Isolates were Penner serotyped and where appropriate underwent PFGE at the Enteric Reference Laboratory. Where cases could recall the source of the poultry they consumed, samples were obtained of the same brand, preparation (eg, chilled/frozen/pieces) and point of sale.
- Questionnaires were administered to 26 cases. 96% of cases had consumed poultry within a 10-day period prior to their illness. Only 11% had been swimming, 7% were exposed to non-reticulated water, 7% to international travel, 7% to animal faeces, and 4% to contact with an ill human. No "novel sources of infection were identified" though two cases were linked to a common source (a takeaway outlet). Food safety failures at the outlet were

identified in a detailed HACCP-based food safety audit (ie, cross-contamination issues).

- The Penner serotype HS 1,44 was significantly higher in prevalence among cases when comparing data for November 2001 with June 2002 (from 2% to 30%, p = 0.0005). Similarly among poultry samples, this serotype also increased from nil to 24% of isolates over this same period (p = 0.01). However, there was no direct correlation between the serotypes of human isolates and the serotypes in their matched poultry samples (p = 0.8).

Assessment (re aetiology): This investigation used limited epidemiological methods (information on cases only) but detailed laboratory methods. There were limitations in the sampling methodology for the poultry isolates (non-random sampling). The study did not show a correlation between the serotypes of case isolates and the linked poultry samples. However, the investigators gave plausible reasons for this: (i) poultry often carry multiple serotypes; (ii) there was a 3-4 week delay in sampling relative to the time of infection.

The significant increase over time in the same Penner serotype (HS 1,44) amongst both human cases and poultry isolates is suggestive that poultry exposure was partly involved in this mid-winter increase in notified cases. Furthermore, this strain was noted by the authors to be the epidemic strain in a previous case-control study in Auckland (Neal G. & Bloomfield, 1997) and was found in another case-control study linked to poultry in 1998 (Calder et al, 1998). Other possible explanations for these patterns (eg, contaminated water supplied to both poultry farms and humans or wild birds infecting both poultry and humans) seem much less plausible possibilities.

Investigation into an increase in campylobacteriosis in Auckland (Simmons et al, 2002a)

Background: This investigation was undertaken in response to an increase in the number of cases of campylobacteriosis notifications in the winter of 2001 and the following spring (with November rates being double those of the preceding year).

Implicated risk factor: The predominance of the same Penner serotype 4 amongst both human cases and poultry isolates is suggestive that poultry exposure was at least partly involved in the increase in notified cases in the Auckland and the North Island in 2001. Spatial analyses by type of water supply did not suggest a significant role for contaminated water exposure.

Key findings:
- One part of the investigation was into a geographic cluster of 15 cases. However, this failed to identify a source (though this investigation involved no comparison group).
- An in-depth investigation into 20 cases (a 20% sample for a week in November) found that all had consumed chicken within the incubation period for illness.
- Questionnaire data was also collected on 49 consecutive laboratory-proven cases. Of these, 89% had consumed chicken.

- Isolates from the 49 cases were Penner serotyped. It was found that Penner serotype 4 complex was the predominant type in these cases ie, in 33% (and also for a concurrent investigation in Wellington – at 50%). The distribution pattern of the serotypes between the two cities was highly correlated (p = 0.006).
- Penner serotype 4 was also shown to be the predominant type in the Auckland chicken samples at 41% (the poultry sampling was linked to cases – as described for the outbreak investigation directly above). However, the distribution patterns were not similar enough to achieve statistical significance.
- The same PFGE pattern (pattern 75) was found in a cluster of 19 isolates of the serotype 4 complex (8 human and 11 chicken). This same indistinguishable pattern was seen in 15 isolates from Wellington cases. There were also two main PFGE clusters involving serotype 27 in both humans and poultry.
- A GIS analysis of *Campylobacter* cases by seven water zones was performed for cases during a two-week period. There were no statistically significant differences – though the rate was slightly higher for populations on non-reticulated supply (33 versus 27 per 100,000).

Assessment (re aetiology): This investigation used limited epidemiological methods (information on cases only) but very detailed laboratory methods. It concluded that an increase in serotype 4 was probably responsible for the increase in human cases in the North Island in the latter part of 2001. It was also considered that the findings "are consistent with the hypothesis that chicken was an important source of infection for the Auckland cases". However, "in view of the diversity of types among human cases, poultry was unlikely to be the only source of infection". These conclusions seem reasonable given the evidence available.

School camp in Marlborough (Speedy, 2003)

Implicated risk factor: The investigation implicated contaminated well water (the same serotype of *C. jejuni* was found in the camp drinking water and from the faeces of a case). Other hazards that may have contributed were exposure to animals at a pet farm and immersion in a river.

Key findings:
- There were nine laboratory confirmed cases and 33 symptomatic cases. There were cases amongst two school groups that had used the camp. The timing of symptom onset was not precisely detailed but was suggestive of a point source outbreak (with symptoms beginning within three days of each camp beginning).
- Questionnaire data from 67 people apparently did not implicate any food items or other risk behaviours (but no detailed data or any analyses were presented in the report). Camp activities included swims in a river and waterfall pool. Some children visited a pet farm with various animals (but 11 of the cases had not attended this pet farm).
- Inspection of the camp kitchens did not identify any problems.

- The drinking water for the camp came from shallow wells with an unprotected well head. The area surrounding the wells had animal faeces (suggestive of inadequate fencing). The outbreak appeared to follow a "very high rainfall" event (75 mm) that resulted in run-off from a deer paddock entering the camp wells.
- The same serotype of *C. jejuni* was isolated from: drinking water (2 samples), the faeces of a child attending Camp 2 and from the faeces of a farmyard sheep. A different species of *Campylobacter* was isolated from river water. Differing serotypes of *C. jejuni* (from those formerly mentioned) were isolated from another child (Camp 1), a farmyard ostrich, a farmyard duck, and the water supply of the camp manager's house. High levels of *E. coli* were also found in the drinking water reticulation of the camp.

Assessment (re aetiology): This investigation used limited epidemiological methods (case history data), a site inspection and detailed environmental sampling and laboratory analyses. Given the laboratory and other evidence, it seems reasonable to conclude that contaminated drinking water was the likely cause, but that exposure to farm animals and recreational water may also have played some role.

Sports club attendees in a Canterbury town (Morrison, 2003a)

Implicated risk factor: The lunch was considered "the most probable source of infection" although the specific reasons for this could not be determined.

Key findings:
- There were five laboratory confirmed cases (and another 11 meet the case definition).
- Cases were from seven different teams (n = 15 teams involved).
- The epidemic curve was highly suggestive of a point source.
- The cohort study identified that consumption of the lunch, corned beef, ham, and boiled potatoes were all significantly associated with illness (all p < 0.05). There were no statistically significant findings for water consumption or for other foods (eg, sandwiches). There were no cases amongst those who brought their own lunch (n = 10). However, the way the statistical results were presented in the report was suggestive of a sub-optimal analysis.
- The water supply used was reticulated town supply (treated by chlorination).
- The site inspections revealed some deficiencies in the kitchen (concerning towels and refrigeration space) and none with the local butchery.

Assessment (re aetiology): This investigation involved the following methods: epidemiological (cohort study with 16 cases and non-cases – with the latter not detailed in the report but estimated by this reviewer to number 50); site inspections; and laboratory testing. The statistical analysis may have been suboptimal but reanalysis by this reviewer for consumption of the lunch (based on the data presented) indicates that it was a highly significant risk factor (p < 0.0001, Fisher exact test). The evidence for foodborne transmission associated with consumption of the lunch appears to be strong based on the epidemiological evidence.

Household outbreak in Christchurch (Morrison, 2003b)

Implicated risk factor: There was very strong laboratory evidence (identical PFGE findings) that consumption of cheerios was the source of infection for two of the cases. Subsequent person-to-person spread was also strongly suggested (laboratory and epidemiological evidence). Site inspection indicated the potential for cross-contamination between raw and cooked meat at the relevant butchery.

Key findings:
- Two household members were notified with campylobacteriosis and it was ascertained that they had consumed cheerios (without these being cooked after purchase).
- The cheerios were purchased with other meat items (steak, saveloys, mince and frozen chicken). The items were purchased in plastic bags (not vacuum packed) and the adult purchaser broke the packs down into smaller lots for freezing (in used supermarket bags).
- Frozen cheerios from the same batch were available for laboratory analysis. This indicated that the *Campylobacter* was a surface contaminant and not present internally. The PFGE typing indicated that the *C. jejuni* isolates from the cheerios and the three faecal samples were indistinguishable (serotype 3,14).
- The third case (laboratory confirmed) was in an infant aged five months who did not eat the suspected food but was bathed with an infected sibling (aged 18 months) while he was still symptomatic with campylobacteriosis.
- The inspection of the butchery involved revealed that the cheerios obtained from the chain's main factory in another city had been broken down into smaller retail lots. The bench used for repacking was also used to prepare and pack raw meat products including chicken (albeit with cleaning and sanitising between packing raw and cooked products).

Assessment (re aetiology): This investigation involved the following methods: epidemiological, site inspection, and very detailed laboratory testing. The identical PFGE results in the suspected food product and the faecal specimens of the three cases strongly indicate that this was the source. The view of the investigating Health Protection Officer that there was a significant risk of cross-contamination occurring at the butchery seems to be a reasonable assessment. Further information on this outbreak may be available when a report is published in the scientific literature (ie, CF Graham et al "Outbreak of campylobacteriosis following pre-cooked sausage consumption", submitted for publication).

Restaurant in Christchurch (Smith, 2004)

Implicated risk factor: None identified but the relevant venue did use untreated well water of unknown microbiological quality.

Key findings:
- The epidemic curve was highly suggestive of a point source (on the day of a particular barbecue event).

- Questionnaires were distributed by email (since email addresses were available for 54 out of the 67 people who had attended the barbecue function). However, the response rate was low (28%). Data analysis did not indicate significant results for any specific food item (but the numbers involved were small).
- One problem was the 18-day delay between the event and the outbreak being discovered.
- Site inspection revealed no obvious risks but the premises use their own well to supply water and this had not been tested (despite it qualifying as a "community drinking water supply" under the *Drinking Water Standards for New Zealand 2000*).
- Some of the attendees took leftovers from the barbecue home with them.

Assessment (re aetiology): This investigation involved epidemiological methods (cohort study) and site inspection. It is unfortunate that the well water test results were not included in the report (as it was recommended to the venue that they arrange for such testing). Although an innovative form of questionnaire dispersal was used (ie, via email), more could possibly have been done to ensure a higher response rate (eg, repeat requests).

Auckland restaurant (birthday party) outbreak (Skelton, 2004)

Implicated risk factor: The investigation concluded that the likely source of infection was "undercooked duck livers or possibly by cross-contamination from the duck livers to other foods". But the investigation also considered that "*Norovirus* is also a possible cause of illness due to the incubation periods and symptoms of at least two cases" (given other evidence for outbreaks relating to oyster consumption at this time).

Key findings:
- The epidemic curve was suggestive of a point source infection – for the 12 cases (4 laboratory confirmed) who met the case definition.
- 47 people completed questionnaires (a 72% response rate). The analysis found statistically significant positive associations for consumption of duck liver parfait (RR = 4.0, 95%CI = 1.22 – 13.09, p = 0.02), Clevedon oysters (RR = 4.27, 95%CI = 1.52 – 11.97, p = 0.009), smoked salmon (RR = 3.94, 95%CI = 0.96 – 16.22, p = 0.03), and cured venison en croute (RR = 5.45, 95%CI = 1.34 – 22.20, p = 0.005).
- A food safety assessment identified that the duck liver parfait was prepared with medium to medium-rare duck livers. Cross-contamination of foods through serving utensils and bowls was considered to be possible (ie, the chopping boards used for poultry were also used for red meats and were not cleaned between uses).
- There were no leftover foods for sampling.
- Although no other pathogens were isolated from the faecal specimens, the investigation noted that the involvement of other infections was possible since three outbreaks involving oysters had been reported to this Public Health Service in early July 2004 (involving a batch of oysters distributed on 2 July). This restaurant used this particular batch at this particular birthday party on 3

July. Furthermore, one of the cases reported that the only food he had eaten all night at the party was "one oyster".

Assessment (re aetiology): This investigation used epidemiological methods (cohort study) and a food safety assessment. It concluded that the likely source of infection was "undercooked duck livers or possibly by cross-contamination from the duck livers to other foods". But they also considered that "*Norovirus* is also a possible cause of illness due to the incubation periods and symptoms of at least two cases." These conclusions seem reasonable given the evidence available from this investigation and other New Zealand evidence relating to poultry livers and campylobacteriosis outbreaks eg, (Whyte et al, 2001).

School camp – Canterbury (Pink, 2004)

Implicated risk factor: The investigation did not identify a specific source, but potentially hazardous consumption of untreated stream water was reported along with potential hazards (eg, spreading effluent from a septic tank).

Key findings:
- There were eight confirmed and 11 probable cases. The epidemic curve was highly suggestive of a point source outbreak – but affecting two groups of camp attendees.
- The camp site had an ultraviolet light treatment water system.
- There were some reports of students drinking directly from a stream during a tramp. Also kayaking and rafting on a river often involved total immersion.
- A cohort of 60 students and teachers were given a questionnaire. There were no statistically significant results for food consumed, recreational water activities, animal contact, or for spreading effluent from the septic tank. However, consumption of untreated water was not documented in the report and the list of questions was not that comprehensive.
- Site inspection suggested adequate kitchen facilities and the foods consumed at the camp were considered to be "low risk".
- After the students had spread effluent from the septic tank, a staff member whose caravan was adjacent to this area became ill with diarrhoea (but did not seek medical attention).

Assessment (re aetiology): This investigation used epidemiological methods (cohort study) and a site inspection. It seems reasonable to conclude that a number of potentially hazardous behaviours may have occurred (ie, drinking untreated stream water, immersion in river water, and spreading septic tank effluent).

Church group camp in the Nelson area (Todd, 2005)

Implicated risk factor: The investigation did not identify a specific source, but it did identify faecal contamination of the camp water supply and potentially hazardous recreational water use.

Key findings:
- There were 58 camp attendees and 13 had symptoms suggestive of campylobacteriosis (of whom three had laboratory confirmed infection).
- The epidemic curve was highly suggestive of a point source outbreak (though there were two outlying cases which may have had different illnesses).
- Questionnaires were distributed by email and post but the response rate was low (n = 19). The data did not implicate water consumption or recreational water activities at a statistically significant level (though cases were actually more likely to have consumed "chilled and tap water" and to have participated in "swimming", or using a "waterslide, kayak, and wakeboard").
- It was presumed that the water consumed was untreated given that the camp had limited facilities for boiling and chilling large quantities of water. The camp drinking water was sourced from a spring and went through a coarse filter. Water samples identified *E. coli* in water from the kitchen tap, from the concrete water storage tank, and from the pond where attendees swam. But no *Campylobacter* was detected in three samples.
- There was conflicting information about the switching of water tanks at around the time of the outbreak – but if this did occur it could have "mobilised sludge from the bottom of an empty water tank".
- It was noted that heavy rainfall prior to the outbreak might have caused some water contamination.

Assessment (re aetiology): This investigation used epidemiological methods (limited cohort study data), a site inspection and laboratory methods. While no specific source was identified, the evidence for faecal contamination of the water supply probably makes this source the most plausible one.

Discussion

The findings of published outbreak reports as to the suspected source are summarised in the table below. The quality of the outbreak investigations was mixed. Some used sub-optimal epidemiological methods and suffered from delays in questionnaire administration (eg, up to four weeks). However, some used high quality case-control and cohort study techniques with appropriate statistical analyses. Some also used detailed site and food safety assessments. Several used laboratory methods that included identifying the specific serotype and/or the use of DNA/PCR analysis – though such techniques were not available for the earlier outbreaks. Despite the various limitations of these investigations, the conclusions of the authors of these reports about the likely cause of the outbreak appeared reasonable to this reviewer and appropriately cautious.

There may have been some publication bias towards outbreak investigations in which contaminated water supplies were implicated (since this type of outbreak was the most common when just considering published reports). It is plausible that some waterborne outbreaks produce higher number of cases, are of greater potential public health concern (especially if a reticulated supply is involved), and may have greater novelty value than foodborne outbreaks (which appear to be more common when the surveillance data are considered). However, when relatively high quality unpublished

reports are considered in conjunction with published reports – the pattern of foodborne transmission predominating over waterborne is the same as that seen for the outbreak surveillance data (see Section 6).

These reports came from outbreaks in just seven districts: Northland (1), Auckland (8), Waikato (1), Hawke's Bay (4), Wairarapa (2), Nelson/Marlborough (2), and Canterbury (11). This pattern appears to be very non-representative of New Zealand as a whole and may reflect the following:

- Main centres having more staff and resources to commit to outbreak investigations.
- Main centres having more staff with expertise in epidemiological methods and closer proximity to laboratories capable of in-depth analyses.
- The location of part of ESR in Christchurch (covering food and water) – since some ESR staff have assisted with local investigations in Canterbury.

The settings for the outbreaks were clearly rural in 11 reports, semi-rural in three (at least in terms of having a non-reticulated water supply), small town in two, and city in 13. On a population basis this pattern is suggestive of a higher occurrence of outbreaks in rural settings. This may reflect the number of outbreaks associated with school camps in rural settings and also those involving untreated water supplies.

Foodborne spread: Foodborne spread was the most commonly described "likely" source of outbreaks in these 29 outbreak reports (n = 9) (Table 5.1). However, there were another six outbreak reports that provided some evidence for possible foodborne transmission (with three overlapping with possible waterborne transmission). In terms of statistically significant findings for a particular food/s there were six such outbreaks (compared to two for waterborne spread). Similarly when comparing *Campylobacter* strains in humans and a suspected source there was evidence of four foodborne outbreaks (Calder et al, 1998; Simmons et al, 2002a; Simmons et al, 2002b; Morrison, 2003a) compared to two waterborne ones (McElnay & Inkson, 2002);(Speedy, 2003).

The implicated items included: raw milk, chicken liver paté, duck liver parfait, chicken, chicken kebabs, kebabs, and cheerios (often with evidence that these were undercooked). There are other reports of poultry products and especially chicken liver paté being involved in outbreaks in New Zealand (see the surveillance data in the next section). The three investigations in Auckland that involved detailed laboratory analysis of human and poultry isolates also suggest that poultry consumption is a significant risk factor.

In the investigation involving contaminated cheerios, there was very detailed epidemiological evidence and laboratory evidence involving an identical strain based on PFGE analysis – in both the food product and the human cases (Morrison, 2003b). However, in this outbreak the source of the contaminated cheerios may have been another meat product (eg, raw chicken). Similarly, in one outbreak the use of contaminated water to prepare food may have been relevant (Zhu & Callaghan, 2002).

Waterborne spread: Waterborne spread was the next most commonly described "likely" source of outbreaks in these reports (n = 6) (Table 5.1). However, there were

another eight outbreak reports that provided some evidence for possible waterborne transmission (with three overlapping with possible foodborne transmission).

For those outbreaks where waterborne spread was "likely", the water treatment was either non-existent or sub-optimal at a rural camp (n = 4) or a semi-rural school (n = 1). In only one outbreak was waterborne spread association with the reticulated water supply of a town (Brieseman, 1987) and in this situation there was evidence of special circumstances (a delay in initiating chlorination and recent work on the infiltration gallery). In some of these outbreaks there was plausible evidence that contamination of the water was from livestock, wild birds and/or had occurred after heavy rainfall. The link with heavy rainfall is consistent with work showing major increases in *Campylobacter* levels in river water during a flood event in New Zealand (Eyles, 2003).

Person-to-person transmission: Three outbreaks described secondary intra-family person-to-person spread (Brieseman, 1984; Smith, 2001; Morrison, 2003b). In one of these there was very detailed epidemiological evidence and laboratory evidence involving an identical strain based on PFGE results (Morrison, 2003b).

Zoonotic transmission: In one outbreak the questionnaire data suggested an association with animal contact – but overall this transmission route was considered unlikely (Simmons et al, 2003). In another one with an unclear source it was noted that some animals at the camp had diarrhoea (McElnay, 2000). One outbreak identified *Campylobacter* in animals from a pet farm (ostrich and duck) but these were of different serotypes to those found in the two human cases for whom there was data (Speedy, 2003).

Table 5.1: Summary of published and unpublished campylobacteriosis outbreak investigations in New Zealand based on reports identified in this review

Likely cause of outbreak	Published outbreak investigations		Unpublished outbreak investigations**	
	No. of out-break reports*	References	No. of outbreak reports	References
Likely to have been "**foodborne**" transmission	2	(Brieseman, 1984; Whyte et al, 2001)	7	(Bishop, 1998; Manning, 1999; Simmons et al, 2002a; Simmons et al, 2002b; Morrison, 2003a; b; Skelton, 2004)
Unclear but limited evidence for "**foodborne**"	2	(McElnay, 1997; Calder et al, 1998)	1	(Ma et al, 2000)
Likely to have been "**waterborne**" transmission	5	(Brieseman, 1987; Stehr-Green et al, 1991; Bohmer, 1997; McElnay & Inkson, 2002; Simmons et al, 2003)	1	(Speedy, 2003)
Unclear but limited evidence for "**waterborne**" transmission	1	(Health Protection Programme - Hawke's Bay Area Health Board, 1992)	4	(Choice Health, 2001; Smith, 2001; 2004; Todd, 2005)
Some evidence for a possible role from either untreated **water or** contaminated **food** exposures	2	(Jarman & Henneveld, 1993; Health Protection - Choice Health Wairarapa, 2002)	1	(Zhu & Callaghan, 2002)
Unclear	1	(Mitchell R. et al, 1993b)	2	(McElnay, 2000; Pink, 2004)
Total	13		16	

* One of these was actually a series of multiple small outbreaks in two separate camp sites (Brieseman, 1984).
** For reports in which there was a relatively high quality epidemiological investigation (case-control or cohort study) or in which laboratory investigations were conducted on a potential source.

Implications for future outbreak investigations

Outbreak investigations are mainly undertaken to address immediate public health threats and to help maintain awareness in the importance of maintaining appropriate food handling behaviours and water supply quality. But they also contribute to improving the understanding of the epidemiology of campylobacteriosis in New Zealand. Conducting high quality investigations is very time and resource consuming and so it is quite understandable that they are not always a priority for Public Health

Unit staff. A number of potential ways to optimise such investigations are listed below as recommendations to the relevant central government agencies (Ministry of Health and the NZ Food Safety Authority):

1. That more resourcing is targeted at building **expertise** amongst Health Protection Officers and other Public Health Unit staff around appropriate investigative methods and the potential advantages of obtaining in-depth laboratory analyses (on serotypes/subtypes etc).

2. That consideration is given to targeting more resourcing on establishing a stronger **repository of central expert advice** to DHBs on how to conduct outbreak investigations (eg, a specific department in a university, or part of an agency such as ESR). Of note is that Auckland University staff have published a number of high quality case-control studies into another enteric disease – giardiasis.

3. That consideration is given to providing more detailed **national guidelines** on how to conduct investigations into campylobacteriosis outbreaks. This could include a standardised design for outbreak investigation reports. Summaries of examples of high quality investigations could be appended to these guidelines and perhaps using some of the details in this review.

4. That central agencies (and DHB management) do more to encourage their staff to **publish the findings** of outbreak investigations as journal articles or letters (with relevant New Zealand publications being: the NZ Medical Journal, the NZ Journal of Environmental Health, and various ESR publications – eg, the NZ Public Health Surveillance Report. Such publications can be used to highlight to food handlers and the public the importance of taking appropriate care.

5. That central agencies (and PHU management) do more to encourage **collaborative information sharing** between PHUs. There are some good examples of such collaboration (Simmons et al, 2002a; Simmons et al, 2003).

6. That a **national review** of outbreak investigations is commissioned on a regular basis (eg, five yearly) with all DHBs being required to supply all relevant reports.

More specific recommendations for PHU management and staff include the following:

1. That where possible, outbreak investigations should make greater use of state-of-the-art **laboratory techniques** to better identify the source of an outbreak. This may involve budgetary planning to ensure that such testing can be paid for. (Alternately central agencies could provide supplementary funding for this purpose eg, for outbreaks over a specific size).

2. That consideration is given to exploring alternatives to case-control studies (given that it can be hard to recruit population controls). One alternative is conducting **case-case studies** (where collective case data are used from other campylobacteriosis outbreaks or outbreaks of other enteric diseases eg, salmonellosis). These have been used to study infectious diseases (McCarthy & Giesecke, 1999) and campylobacteriosis specifically (Gillespie et al, 2002; Gillespie et al, 2003). Another option is to make more use of the **case-crossover study** design.

3. That consideration is given to expanding the use of **email questionnaires** as part of outbreak investigations. However, to ensure adequate response rates these may have to be backed up with reminders (by email or phone).

Summary

A total of 13 published outbreak investigations were identified along with 16 unpublished ones (that met the quality criteria for inclusion in this review). The quality of the outbreak investigations was mixed, though some used high quality case-control and cohort study techniques, detailed site and food safety assessments, and state-of-the-art laboratory methods.

The pattern of foodborne transmission predominating over waterborne was apparent as for the outbreak surveillance data (see Section 6). Foodborne spread was the most commonly described "likely" source of outbreaks in nine reports. However, there were another six outbreak reports that provided some evidence for possible foodborne transmission. The implicated items included: raw milk, various poultry items, kebabs, and cheerios (often with evidence that these were undercooked). For some of these outbreaks there was high quality laboratory evidence that linked human serotypes with serotypes in the foods. There were also more foodborne outbreaks with statistically significant findings for food/s that there were for waterborne outbreaks (six versus two).

Waterborne spread was the next most commonly described "likely" source of outbreaks in these reports (n = 5). There was evidence for person-to-person transmission in three outbreaks. Zoonotic transmission was considered possible in three outbreaks but the evidence was very weak.

6 Analysis and Review of Outbreak Surveillance Data

Background

Public Health Services around New Zealand routinely provide ESR with data as part of a national outbreak surveillance system. This system was introduced by ESR in July 1996 and has had various on-going refinements (ESR, 2005b). A standardised outbreak reporting form is used and the data are provided electronically to ESR as an additional module of the EpiSurv software (used for national notifiable disease surveillance). As part of this review, data for the most recent five-year period (2000 to 2004) was obtained from ESR and analysed. This period was selected since in the first few years of the outbreak surveillance system data quality may have been poorer. Information about the outbreak surveillance system was also obtained from key informants, ESR's annual outbreak reports and other available documentation.

Findings

There were 218 outbreaks involving *Campylobacter* in the five-year period studied. But for this analysis two outbreaks in which other enteric pathogens were also identified were excluded. The resulting 216 outbreaks represented a small proportion of total outbreaks of notified conditions reported in EpiSurv (10.7% for the year 2004).

Out of these outbreaks, there were 13 outbreaks with reports that remained "interim" (6.0%). These "interim" reports were spread out over the five-year period (with no statistically significant trend in the proportion). A written report on the outbreak was reportedly prepared in 49.1% (n = 106) of outbreaks.

The species of *Campylobacter* was infrequently identified at a species level (eg, *jejuni* in 27 (12.5%) and *Thorburn* (n = 1)).

Cases in the outbreaks: The 216 outbreaks of campylobacteriosis were associated with a total of 992 cases – an average of 198 outbreak cases per year (Table 6.1). Of these cases, 480 were laboratory confirmed (48.4%), another 212 (21.4%) were also classified as confirmed, and 300 (30.2%) were classified as "probable cases".

A majority (52.3%) of the outbreaks involved just two cases, and another 19.9% involved 3 cases. Only 6.9% of outbreaks involved 10 or more cases. The largest outbreak involved 116 cases.

Outbreak spatial distribution: The Auckland grouping of DHBs reported half of all the outbreaks. Canterbury reported the next highest number (at 10.6% of outbreaks). In contrast one DHB reported nil outbreaks in the five-year period and three only

reported one (Table 6.1). This variation in reporting is probably a surveillance artefact associated with differing levels of enthusiasm for campylobacteriosis outbreak investigation by DHB. For this reason, the rates of outbreaks per 100,000 population have not been calculated in this analysis.

Table 6.1: Campylobacteriosis outbreaks for the five-year period (2000 – 2004) by reporting DHB / DHB Grouping

DHB / DHB grouping	Outbreaks		Cases				
	No.	Percent	Range of cases (No.)	Mean No. of cases per outbreak	Total cases	Total cases laboratory confirmed	Percent laboratory confirmed
Northland	0	0.0	-	-	-	-	-
Auckland	108	50.0%	2-49	3.6	385	202	52.5%
Waikato	2	0.9%	3-8	5.5	11	9	81.8%
Tauranga	2	0.9%	2-3	2.5	5	2	40.0%
Rotorua	13	6.0%	2-11	3.5	46	29	63.0%
Gisborne	2	0.9%	2	2.0	2	1	50.0%
Taranaki	3	1.4%	2-4	3.0	9	8	88.9%
Hawke's Bay	4	1.9%	4-116	32.5	130	8	6.2%
Wanganui	3	1.4%	2	2.0	6	4	66.7%
Manawatu	20	9.3%	2-9	2.6	51	42	82.4%
Wairarapa	1	0.5%	9	9.0	9	2	22.2%
Wellington	8	3.7%	2-5	3.5	28	22	78.6%
Nelson	3	1.4%	2-7	3.7	11	6	54.5%
Marlborough	1	0.5%	32	32.0	32	9	28.1%
West Coast	8	3.7%	2-5	3.1	25	12	48.0%
Canterbury	23	10.6%	2-28	8.7	199	97	48.7%
South Canterbury	1	0.5%	3	3.0	3	2	66.7%
Otago	7	3.2%	2-4	2.4	17	15	88.2%
Southland	7	3.2%	2-7	3.0	21	7	33.3%
Total	**216**	**100.0%**	**2-116**	**4.6**	**992**	**480**	**48.4%**

Distribution by season and month: The seasonal distribution of outbreaks indicates a summer peak and winter low (Table 6.2) with the difference between these two seasons being significant (rate ratio = 1.69, 95%CI = 1.19 – 2.40, p = 0.002). The summer peak was more pronounced for outbreaks classified as "waterborne" compared to "foodborne" ones. When excluding outbreaks that were classified as involving both forms of transmission (n = 11), the seasonal difference between foodborne and waterborne outbreaks was significant (p = 0.027, Fisher exact test). Indeed, for some winter months there were no waterborne outbreaks at all (Table 6.3).

When considering the seasonal distribution of outbreaks by region, there were some statistically significant differences in the seasonal rates (Table 6.4). The peak season was spring in the northern North Island (with September being the peak month) (Table 6.4 and Table 6.5). In the rest of the North Island the peak was later in summer (with December being the peak month). In both of the South Island regions the peak season was autumn. Within regions the summer rate of outbreaks was statistically significantly greater than the winter rate (and also the autumn rate) in just one region – the "Rest of the North Island", p < 0.01 for both).

Table 6.2: Seasonal distribution of campylobacteriosis outbreaks (2000-2004)

Season	All outbreaks		Foodborne outbreaks		Waterborne outbreaks	
	No.	Percent	No.	Percent	No.	Percent
Summer	66	30.6%	43	35.5%	14	48.3%
Autumn	54	25.0%	25	20.7%	7	24.1%
Winter	39	18.1%	27	22.3%	1	3.4%
Spring	57	26.4%	26	21.5%	7	24.1%
Total	**216**	**100.0%**	**121**	**100.0%**	**29**	**100.0%**

Table 6.3: Distribution of campylobacteriosis outbreaks by month (2000-2004)

Month	All outbreaks		Foodborne outbreaks		Waterborne outbreaks	
	No.	Percent	No.	Percent	No.	Percent
January	22	10.2%	14	11.6%	4	13.8%
February	20	9.3%	13	10.7%	5	**17.2%**
March	18	8.3%	9	7.4%	2	6.9%
April	18	8.3%	9	7.4%	2	6.9%
May	18	8.3%	7	5.8%	3	10.3%
June	11	5.1%	7	5.8%	0	0.0%
July	14	6.5%	10	8.3%	0	0.0%
August	14	6.5%	10	8.3%	1	3.4%
September	23	10.6%	10	8.3%	2	6.9%
October	15	6.9%	5	4.1%	1	3.4%
November	19	8.8%	11	9.1%	4	13.8%
December	24	**11.1%**	16	**13.2%**	5	**17.2%**
Total	216	100.0%	121	100.0%	29	100.0%

Table 6.4: Distribution of campylobacteriosis outbreaks by season and region (2000-2004)

Season	Northern North Island*		Rest of North Island*		North of South Island*		Rest of South Island*	
	No.	Percent	No.	Percent	No.	Percent	No.	Percent
Summer	28	22.4%	21	**51.2%** ** #	12	34.3%	5	33.3%
Autumn	28	22.4%	7	17.1%	13	**37.1%** #	6	**40.0%**
Winter	29	23.2%**	3	7.3%	5	14.3%	2	13.3%
Spring	40	**32.0%** **	10	24.4%	5	14.3%	2	13.3%
Total	**125**	**100.0%**	**41**	**100.0%**	**35**	**100.0%**	**15**	**100.0%**

* Northern region of North Island: Northland, Auckland, Waikato, Tauranga, and Rotorua;
Rest of North Island: Gisborne, Taranaki, Wanganui, Manawatu, Hawke's Bay, Wairarapa, Wellington.
Northern region of South Island: Nelson, Marlborough, Canterbury, and West Coast.
Rest of South Island: South Canterbury, Otago, and Southland.

** Significantly higher rate for this region than the other regions combined in this season (p < 0.05).
# Significantly higher rate for this region than the region directly to the north (p < 0.05).

Table 6.5: Distribution of campylobacteriosis outbreaks by month and region (2000-2004)

Month	Northern North Island*		Rest of North Island*		North of South Island*		Rest of South Island*	
	No.	Percent	No.	Percent	No.	Percent	No.	Percent
January	10	8.0%	7	17.1%	5	**14.3%**	0	0.0%
February	8	6.4%	5	12.2%	4	11.4%	3	**20.0%**
March	9	7.2%	1	2.4%	5	**14.3%**	3	**20.0%**
April	9	7.2%	3	7.3%	4	11.4%	2	13.3%
May	10	8.0%	3	7.3%	4	11.4%	1	6.7%
June	9	7.2%	1	2.4%	1	2.9%	0	0.0%
July	8	6.4%	2	4.9%	2	5.7%	2	13.3%
August	12	9.6%	0	0.0%	2	5.7%	0	0.0%
September	16	**12.8%**	3	7.3%	3	8.6%	1	6.7%
October	10	8.0%	4	9.8%	1	2.9%	0	0.0%
November	14	11.2%	3	7.3%	1	2.9%	1	6.7%
December	10	8.0%	9	**22.0%**	3	8.6%	2	13.3%
Total	**125**	**100.0%**	**41**	**100.0%**	**35**	**100.0%**	**15**	**100.0%**

* See the preceding table for a list of DHBs included in each region.

Outbreak type and setting: The most common type of outbreak was that classified as a "common event" outbreak (46.8%) (Table 6.6). Following this was "transmission in a single household" (23.6%). The home was the most common type of setting involved in outbreaks (37.0%) (Table 6.7). Following this were "restaurants/cafés" (24.5%) and then "takeaways" (6.5%).

Table 6.6. Type of campylobacteriosis outbreak (2000 – 2004)

Type of Outbreak (mutually exclusive categories)	No.	Percent
Common event	101	46.8%
Household (transmission in a single household)	51	23.6%
Common source in specific place (eg, environmental site, farm animals)	29	13.4%
Common source dispersed in community (eg, food/water/environmental site)	9	4.2%
Institutional (transmission in a defined setting)	5	2.3%
Community-wide, person to person transmission	1	0.5%
Other	3	1.4%
Unknown	17	7.9%
Total	**216**	**100.0%**

Table 6.7: Setting where exposure/transmission occurred for all campylobacteriosis outbreaks 2000-2004 (not mutually exclusive categories)

Setting	No.	Percent
Home	80	37.0%
Farm	10	4.6%
Camp	8	3.7%
Workplace	5	2.3%
Hotel/motel	4	1.9%
Childcare centre	3	1.4%
School	3	1.4%
Rest home	2	0.9%
Prison	1	0.5%
Hospital (Acute care)	1	0.5%
Hospital (Continuing care)	0	0.0%
Hostel/boarding house	0	0.0%
Tangi/hui	0	0.0%
Community/church gathering	0	0.0%
Swimming/spa pool	0	0.0%
Food-related service		
Restaurant/Café	53	24.5%
Takeaway	14	6.5%
Supermarket/delicatessen	5	2.3%
Caterers	4	1.9%
Abattoir/meat processing plant	2	0.9%
Other food outlet	4	1.9%
Other setting	12	5.6%
Total	**211**	97.7%

Reported mode of transmission: The most common mode of transmission reported was "foodborne" (56% of outbreaks) followed by "person-to-person spread" (23.1%) (Table 6.8). When considering outbreaks with varying levels of more detailed evidence, the proportion of outbreaks that were categorised as involving "foodborne" or "waterborne" transmission increased.

The most common form of evidence for determining mode of transmission was epidemiological information concerning the exposure history of the cases (64.8%) (Table 6.9). However, only a tiny proportion of outbreaks (1.9%) were investigated using ideal epidemiological techniques (eg, a case-control or cohort study). Higher proportions of investigations were based on laboratory evidence (3.2%) and an environmental investigation (23.1%). Also, only in 2.3% of outbreaks (n = 5) was the "source" considered in be "definite", with most being categorised as the source being "suspect" (ie, in 63.9%, n = 138).

Table 6.8: Reported mode of transmission for all campylobacteriosis outbreaks 2000-2004 (not mutually exclusive categories)

	All outbreaks (n = 216)		Outbreak with more detailed evidence* (n = 11)		Evidence from environmental investigation** (n = 50)	
	No.	%	No.	%	No.	%
Foodborne, from consumption of contaminated food or drink (excluding water)	121	56.0%	8	72.7%	42	84.0%
Person to person spread, from (non-sexual) contact with an infected person	50	23.1%	2	18.2%	3	6.0%
Waterborne, from consumption of contaminated drinking water	29	13.4%	5	45.5%	8	16.0%
Zoonotic, from contact with an infected animal	19	8.8%	1	9.1%	3	6.0%
Environmental, from contact with an environmental source (eg, swimming)	5	2.3%	1	9.1%	3	6.0%
Sexual contact / parenteral / vector borne	0	0.0%	0	0.0%	0	0.0%
Other mode of transmission	1	0.5%	0	0.0%	0	0.0%
Unknown mode of transmission	32	14.8%	0	0.0%	0	0.0%

* Either higher quality epidemiological evidence (case control or cohort study) or else laboratory evidence identifying the pathogen in the implicated source (eg, leftover food, water or environmental source).
** Evidence from environmental investigation that identified critical control point failures linked to the implicated source.

Table 6.9: Evidence for mode of transmission and vehicle / source for campylobacteriosis outbreaks (all outbreaks 2000-2004, not mutually exclusive categories)

Type of evidence	No.	Percent
Epidemiological – cases had history of exposure to implicated source	140	64.8%
Epidemiological – case control or cohort study showed elevated risk for cases exposed to implicated source	4	1.9%
Laboratory – pathogen/toxin/chemical suspected to have caused illness identified in implicated source eg, leftover food, water, animal or environmental source	7	3.2%
Laboratory – pathogen suspected to have caused illness identified in food handler	0	0.0%
Environmental investigation – identified critical control point failures linked to implicated source	50	23.1%
Other evidence*	9	4.2%
No evidence obtained	27	12.5%

* This category included epidemiological information obtained after discussions with the cases and various information that should possibly have been classified in other categories listed in this table.

Foodborne outbreaks: When considering those factors which were identified as contributing to outbreaks classified as "foodborne", then "undercooking" was the most common (34.7%) (Table 6.10). This was followed by "cross-contamination" (33.1%). However the reported "unknown factors" was relatively high at 31.4%. Of these foodborne outbreaks, environmental investigation identified critical control point failures linked to the implicated source in 34.7% of outbreaks (n = 42).

In the reported foodborne outbreaks, various types of specific foods or meals were listed in 97 outbreaks. Where a sole type of food was mentioned, then chicken and chicken liver were the most frequently described items (ie, 61.1% for poultry overall). The overall ratio of specific poultry items to specific red meat items being described was 8.3 to 1. Similarly, when considering mixed lists of foods, poultry foods still dominated over red meats, seafood and dairy products. The level of evidence for implicating specific foods (ie, poultry and specific red meats) was mixed (see the footnotes to Table 6.11) but in at least two cases it was of relatively high quality. One included a detailed investigation that has also been described in a published outbreak investigation report (Whyte et al, 2001). Another included laboratory identification of the same strain of campylobacteriosis in the food eaten (a raw meat item) as in the faeces of the cases and stated that this was written up in an article sent for publication (see the section on unpublished outbreak reports in Section 5).

One published outbreak annual report (covering the year 2001) also notes six outbreaks with "confirmatory evidence" that involved the specific foods of chicken and chicken liver (Thornley C. et al, 2002b).

Table 6.10: Factors described as contributing to outbreaks classified as "foodborne" (n = 121 outbreaks, not mutually exclusive categories)

Factors	No.	Percent
Time/temperature abuse		
Undercooking	42	34.7%
Inadequate cooling or refrigeration	11	9.1%
Improper storage prior to preparation	9	7.4%
Improper hot holding	6	5.0%
Inadequate thawing	4	3.3%
Inadequate reheating of previously cooked food	3	2.5%
Preparation too far in advance	2	1.7%
Contamination of food		
Cross-contamination	40	33.1%
Contamination from an infected food handler	2	1.7%
Chemical contamination	0	0.0%
Unsafe sources		
Consumption of raw food	4	3.3%
Use of untreated water in food preparation	3	2.5%
Consumption of unpasteurised milk	1	0.8%
Use of ingredients from unsafe sources	0	0.0%
Other factors		
Unknown factors	38	31.4%
Other factors*	9	7.4%

* This category included information that should possibly have been classified in the other categories listed in this table.

Table 6.11: Foods described* in regard to outbreaks classified as "foodborne" (n = 97 outbreaks with food described, mutually exclusive categories)

Food item/s described	No.	Percent
Poultry#		
Chicken (sole specific item)	37	38.1%
Chicken "undercooked" (sole specific item)	5	5.2%
Chicken liver (sole specific item)	11	11.3%
Chicken liver "undercooked" (sole specific item)	3	3.1%
Duck liver (sole specific item)	1	1.0%
Poultry not otherwise specified (sole specific item)	1	1.1%
Other		
Red meat (as the sole specific item eg, ham)#	7	7.2%
Kebab (sole specific item, not otherwise specified)	2	2.1%
Raw milk (sole specific item)	1	1.0%
Seafood (in a pancake)	1	1.0%
Mix of foods**	21	21.6%
Mix of food/s** and contaminated / untreated water	7	7.2%
Total	**97**	**100.0%**

* Based on data in the "suspected vehicles or source" field and the "comments" field of the data set.
** Out of these two categories: 14 of the outbreaks had meals that included chicken, 10 included at least one type of red meat, 3 a type of seafood, 2 raw milk, and 1 other dairy product (cheese).
\# The evidence for these specific items (n = 65) was epidemiological history (83.1%); environmental investigation (43.1%); epidemiological studies (3.1%) – one of which has been published (Whyte et al, 2001); and a laboratory test of the source (3.1%). For one of the meats it was reported that laboratory investigation had found an "identical strain of *Campylobacter* identified in the food eaten and the faeces of the cases."

Waterborne outbreaks: When considering those factors which were identified as contributing to outbreaks classified as "waterborne", the use of an untreated water supply was the most common (75.9%) (Table 6.12). This was followed by "contamination of source water" (44.8%). Of these waterborne outbreaks, environmental investigation identified critical control point failures linked to the implicated source in 27.6% (n = 8).

Table 6.12: Factors contributing to waterborne outbreaks (n = 29 outbreaks, not mutually exclusive categories)

Factor	No.	Percent
Untreated water supply	22	75.9%
Contamination of source water	13	44.8%
Treatment process failure	1	3.4%
Post treatment contamination	0	0.0%
Contamination of reservoir(s)/holding tank(s)	0	0.0%
Other factor*	3	10.3%
Unknown factors	7	24.1%

* That is: "no treatment process on supply"; "river water not private supply"; "slug of dirty water".

Discussion

Quality of the data

Incompleteness of outbreak reporting: There are many limitations with the routine outbreak surveillance data. Firstly it is very unlikely to under-represent the true burden of campylobacteriosis outbreaks given the highly variable pattern of reporting around the country. The variation in reporting of outbreaks by DHBs is not just apparent for campylobacteriosis – but for other outbreaks as well (as suggested by the data in ESR's annual outbreak summary reports). Possible factors include:

- Differing levels of laboratory notification within DHBs – making it easier to identify outbreaks in areas where such systems are in place.
- Differing levels of laboratory capacity (eg, to identify the similar serotypes that may suggest an outbreak) and the capacity of DHBs to afford this level of investigation.
- Differing levels of responsiveness by members of the public or by clinicians to reporting suspected outbreaks to health authorities. Indeed, relatively few PHUs actively promote to the public that they have a service for investigating food poisoning (Whyte, 2003).
- Differing levels of human and other resources for outbreak investigation by DHBs, and different quality of the reporting systems.

Survey data indicates that PHUs generally have a heavy reliance on informal and unstructured systems for outbreak detection (Whyte, 2003). For example, 72% of the 18 PHUs surveyed had no system in place for detecting trends of reported foodborne illness. Also, only 33% used standardised forms for recording the details of investigations into premises implicated in suspected food poisoning cases (Whyte, 2003). Some PHUs did not have a policy for requesting faecal specimens from cases of suspected foodborne illness (11% of those surveyed).

Furthermore, some detected outbreaks that do meet the formal definition of an outbreak may not be formerly reported by PHUs. This is thought to be because such reporting would lead to many reports of minor outbreaks that created a lot of additional work for perceived limited benefit (MacBride-Stewart & Boxall, 2005). One anecdotal report suggests that a particular PHU only investigates campylobacteriosis outbreaks where the number of cases is eight or more (in contrast to the official national definition of an outbreak involving two or more cases linked to a common source).

Further preliminary evidence for incomplete identification of clusters through the surveillance system comes from New Zealand work on subtyping human isolates. This has found that 65% of human isolates in and around Christchurch city could be grouped into clusters of between 2 and 26 cases (Gilpin et al, 2005).

Incompleteness of data: As detailed in the findings section above, there is incomplete information relating to a range of aspects of outbreaks. This is likely to reflect the level of resourcing of outbreak investigation, although in some cases all of the available information may not be entered on the form. Also, some reports appear to have "interim" status for some years after the outbreak has occurred.

Other limitations: Other limitations with the outbreak surveillance system that have been noted include: sub-optimal timeliness (ie, only weekly data downloads); inconsistency around sending in reports on suspicion versus a confirmed outbreak; and the lack of a formal system for cross-border communication concerning outbreaks (ie, between DHBs) (MacBride-Stewart & Boxall, 2005). ESR staff also note that while there is the capacity to link information between the Outbreak Module of EpiSurv with the rest of the notification data, this is incompletely used by PHUs.

Relevance for understanding disease aetiology

The aetiological factors attributed to campylobacteriosis outbreaks may differ from those causing sporadic cases. For example, "waterborne transmission" may feature relatively highly in outbreak reports because it is so often a readily "plausible" mechanism given that many New Zealand water supplies are still sub-optimally treated. It is also easier to test a water supply (and find faecal coliforms) than it is to obtain and test leftover food items from a suspected meal. Nevertheless, it is probably reasonable to assume that the transmission mechanisms for outbreaks and for sporadic cases (some of whom may be part of undetected outbreaks) are of broadly similar importance. If such a broad similarity is assumed, then the outbreak data do provide weak supportive evidence for the following:

- That foodborne transmission is probably relatively important in the New Zealand setting (ie, relative to waterborne transmission).
- That exposure to poultry products is probably a relatively important part of foodborne transmission (ie, relative to other specific food sources). Some poultry products such as chicken liver have been frequently associated with outbreaks.
- That sub-optimal food handling behaviours (particularly undercooking and cross-contamination) appear to play a role in foodborne transmission.
- That waterborne transmission probably accounts for a significant proportion of the New Zealand disease burden (while appearing to be less important than foodborne transmission).
- The greater seasonality of waterborne outbreaks (relative to foodborne outbreaks) is suggestive that waterborne transmission may play a role in the national seasonal pattern of the notification rates.
- That zoonotic transmission and transmission from other environmental sources play relatively minor roles in disease aetiology (relative to foodborne and waterborne transmission).

A previous review relating to poultry and campylobacteriosis risk in New Zealand (Lake R et al, 2003) reported that between 1997-1999 there were 14 outbreaks in which poultry were "implicated". However, the confirmation of these was only "epidemiological" except for one case where there was laboratory confirmation. It

was also noted that there was a lack of data relating the type of *Campylobacter* in the food and the type causing human illness.

Improving outbreak surveillance for campylobacteriosis

There is much scope for improving outbreak surveillance relating to campylobacteriosis in New Zealand. Furthermore, if such surveillance was working in an optimal manner then it would probably substantively inform our understanding of disease epidemiology and help advance the research agenda into possible interventions.

Various improvements to the reporting outbreak surveillance system have recently been suggested (MacBride-Stewart & Boxall, 2005). These should be implemented with the appropriate software changes to EpiSurv, to the forms and the relevant supporting documentation. In addition to these, the following options for improving outbreak surveillance could be considered by the relevant parties (the Ministry of Health, the NZ Food Safety Authority, ESR and DHBs):

1. **An in-depth audit** of outbreak investigations into campylobacteriosis could be done for one DHB (eg, a large one with relatively good systems). This could ascertain more precisely the scope for improvement and the resource implications for such improvements (eg, to assist with planning a sentinel system).

2. The outbreak investigation system could have a more explicit **prioritising system** built in to it to ensue that there is greater focus given to particular outbreak investigations. These could be the outbreaks that pose the greatest potential threat to public health (eg, outbreaks in which the following may be implicated: a reticulated water supply, a food processor, a food handler, or a food outlet). Of note however, is that these associations may only become evident after routine investigations have proceeded to some depth.

3. **Sentinel sites** for intensive outbreak surveillance could be developed (ie, select DHBs). These could then be provided with additional national level funding to raise the quality of the investigations and outbreak reporting. Investigations in these sentinel sites could make greater use of serotyping and DNA technologies for more fully investigating outbreaks. If greater use of such techniques proves to be useful and cost-effective, then national guidelines for their appropriate use could be developed. Ideally it would be desirable to have a sentinel site covering a relatively urban DHB and another covering a relatively rural DHB.

4. Greater use could be made of routine **laboratory reporting** of campylobacteriosis as this would assist in improving the level of outbreak detection.

In addition to these changes to the surveillance system, the routine analysis of outbreak surveillance data could be expanded upon. That is, some of the analyses undertaken in this review could be repeated on an annual basis, and in regular in-

depth reviews every few years. However, such analyses would benefit from quality improvements in the data.

Summary

A total of 216 outbreaks for campylobacteriosis in the most recent five-year period (2000-2004) were analysed. According to the transmission mode reported by Public Health Unit staff and for which there was an environmental investigation undertaken, the following transmission modes were involved: foodborne (84%), waterborne (16%), person-to-person spread (6%), zoonotic (6%), and environmental source (6%) (ie, some outbreaks involved multiple transmission modes). However, the quality of the supporting evidence was mixed. For example, while 65% of outbreaks involved evidence from cases on exposure history, only 23% involved an environmental investigation, only 3% had laboratory evidence on the source, and only 2% involved a proper epidemiological study.

Various other limitations with the data also reduce the scope for using this information to better understand campylobacteriosis aetiology in New Zealand (especially for sporadic cases which comprise most of the disease burden). Nevertheless, the outbreak surveillance data do provide weak evidence to suggest that foodborne disease transmission is more important than waterborne and other transmission mechanisms. Furthermore, there remains substantial scope for improving the outbreak surveillance system so that it can better inform our understanding of the epidemiology of this disease and the research agenda for its control.

7 Review of Relevant Environmental and Laboratory Studies

This section considers additional published studies identified in the literature review that provide information of relevance to campylobacteriosis in the New Zealand setting.

The Ashburton study

The largest New Zealand study on potential environmental reservoirs for human campylobacteriosis has only recently been published (Devane et al, 2005). This very detailed study used laboratory methods to identify *Campylobacter* spp. in faecal, food and river water samples. A total of 1450 samples of 12 matrix types were obtained from a defined geographical area (Ashburton). It was noted that this town has one primary reticulated water source and that most of its inhabitants live and work in the area. A total of 61 human faecal specimens from notified cases were obtained along with specimens from ducks (wild); dairy cattle (faeces); beef cattle (faeces and offal), sheep (faeces and offal), pork (offal), chicken carcasses, rabbit (faeces) and possum (faeces).

The study identified the significant prevalence of *Campylobacter* for ten of these 12 matrices (ie, all except rabbit and possum). The serotype patterns for livestock sources were more similar to the human ones than were those from water and the wild ducks. The Penner serotyping and *Sma*I-PFGE analysis also indicated similar patterns across the matrices. In particular, it was tentatively suggested that there could be further investigation into the possibility of cattle acting as a significant reservoir for human campylobacteriosis.

In a more detailed report on the same study (Baker et al, 2002), the Czekanowski index of similarity analyses was presented for the matrices. These results indicated that the highest similarity was between human cases and beef cattle faeces, followed in descending order by sheep offal, dairy cow faeces, sheep faeces, beef offal and chicken carcasses (for Penner serotypes). For PFGE subtypes the equivalent order was: sheep faeces, beef faeces/sheep offal (second equal), and dairy faeces/chicken carcasses/water (all third equal).

The Devane et al study also reported the prevalence of *Campylobacter* infection found in the chicken samples was relatively low (at 27.5% for *C. jejuni*) relative to other estimates in New Zealand. They considered that this could be because all the chicken was supplied by one company, which may have a lower rate than other companies. Also the difference between testing whole chickens versus chicken portions may have been relevant.

Overall, this study appears to be a very detailed one that has provided a lot of valuable information of potential transmission mechanisms in the New Zealand rural setting. It

appropriately notes that the findings do not imply causal pathways (eg, humans and livestock could be infected from the same source rather than one being a cause of infection for the other). It also notes that not all reservoirs and transmission routes were considered in the study.

Nevertheless, a possible limitation with this study was the lack of statistical analysis of the data (including of the Czekanowski index results). To address this, some of the data from the study were collated and further statistical analysis undertaken by this reviewer (see Tables 7.1 to 7.3). Of note however is that a more sophisticated statistical analysis could probably be done involving additional biostatistical expertise.

Further analysis of data obtained by: (Devane et al, 2005): The largest difference from the human pattern was that for ducks (in all serotype and subtype categories where there were over 2 samples) with all being statistically significant (Table 7.1). The next largest difference was between the pattern for humans versus that for water (statistically significant in all serotype and subtype categories). In all of the three serotype and subtype categories considered, the differences between the human pattern and the water pattern were greater than for human versus the "all livestock" pattern. The trend comparisons in Table 7.2 and 7.3 also show statistically significant patterns (ie, between those for humans, livestock and water / ducks). These findings are suggestive that in this rural setting, livestock contact and/or consumption of livestock products may be more relevant to human illness than: (i) water exposure or consumption; or (ii) exposure to environmental contamination from wild birds (at least duck faeces in public parks).

When considering the differences between the human pattern and that for various livestock species in all three serotype and subtype categories, the most similar pattern to the human one was for dairy cattle faeces (1 category), beef offal (1), and sheep offal (1). The second most similar pattern in each category was for sheep offal (1 + 1 equal), dairy faeces (1 equal), and beef offal (1). The most *different* pattern from the human one was chicken (1 category), beef faeces (1), and pork offal (1). The second ranked most different patterns were sheep faeces (1 + 1 equal), chicken (1), beef offal (1 equal), and pork offal (1 equal). These findings are therefore in agreement with those stated Devane et al that the similarity between the human pattern and that of dairy cattle is of note and may possibly reflect a relevant transmission pathway.

The data are also somewhat suggestive that the pattern in offal is more similar to the human one than for faeces, when species-specific data are compared (ie, for 3/3 categories for sheep, 3/3 categories for beef). Furthermore, beef and sheep faeces were statistically significantly different from the human pattern in 2 and 3 categories respectively, while beef and sheep offal were only statistically significantly different in zero and 1 category respectively. These differences are suggestive that offal is more relevant for human transmission than is faecal contamination of water or land (at least for these two species).

Table 7.1: Laboratory analysis of isolates from humans, water and various animals by *Campylobacter* species, serotype and subtype (derived from data in: (Devane et al, 2005) and excluding those that were untypable)

	Human faeces	Water	Duck faeces	Dairy faeces	Beef faeces	Sheep faeces	Beef offal	Sheep offal	Pork offal	Chicken carcass	All livestock (faeces, offal, carcass)
Species (from Table 1)											
C. jejuni (No.)	57	162	60	89	73	52	15	63	9	56	357
C. coli (No.)	6	12	1	9	14	41	1	6	9	2	82
% *C. jejuni*	90%	93%	98%	91%	84%	56%***	94%	91%	50%**	97%	81%
Heat stable serotype (from Table 2)											
Same as human ones (No.)	52	68	17	77	60	32	11	49	8	24	261
Different from human ones (No.)	0	30	19	5	5	9	0	3	1	15	38
% The same#	100%	69%***	47%***	94%	92%	78%***	100%	94%	89%	62%***	87%**
Same as top 5 human ones (No.)	37	40	6	71	44	20	7	39	5	18	204
Other ones (No.)	15	73	39	11	21	27	5	18	4	23	109
% The same	71%	35%***	13%***	87%*	68%	43%**	58%	68%	56%	44%**	65%

	Human faeces	Water	Duck faeces	Dairy faeces	Beef faeces	Sheep faeces	Beef offal	Sheep offal	Pork offal	Chicken carcass	All livestock (faeces, offal, carcass)
C. jejuni Sma-I-PFGE types (from Table 4)											
Same as human ones (No.)	17	20	4	40	22	12	2	17	2	15	110
Different from human ones (No.)	0	50	13	12	13	6	1	7	1	7	47
% The same#	100%	29%***	24%***	77%*	63%**	67%*	67%	71%*	67%	68%*	70%**
Subtypes of C. jejuni (from Table 6)											
Same as human ones (No.)	32	11	3	40	22	11	6	23	1	11	114
Different from human ones (No.)	0	10	6	10	7	3	1	3	1	7	32
% The same#	100%	52%***	33%***	80%**	76%**	79%*	86%	88%	50%	61%***	78%**
Same as most frequent human ones (≥ 2 isolates) (No.)	18	5	1	29	8	3	1	9	0	1	51
Other ones (No.)	14	16	8	21	21	11	6	17	2	17	95
% The same	56%	24%*	11%*	58%	28%	21%*	14%	35%	0%	6%***	35%*

Mantel-Haenszel chi-square tests or Fisher exact (2-tailed) where cell values were less than 5.
* $p < 0.05$ (relative to the pattern for human isolates)
** $p < 0.01$ (relative to the pattern for human isolates)
*** $p < 0.001$ (relative to the pattern for human isolates)
# The data in these rows are the results that are discussed in the text.
The Table numbers refer to those in the paper: (Devane et al, 2005)

Table 7.2: Comparison between human, livestock and water isolates of *Campylobacter* (derived from data in: (Devane et al, 2005) and excluding those that were untypable)

	Human faeces	All livestock (faeces, offal, carcass)	Water	Chi square for trend	p-value
Species					
% C. jejuni	90%	81%	93%	3.9	0.047
Heat stable serotype					
% Same as human ones	100%	87%	69%	25.6	< 0.00001
% Same as top 5 human ones	71%	65%	35%	28.3	< 0.00001
C. jejuni Sma-**I-PFGE types**					
% Same as human ones	100%	70%	29%	42.9	< 0.00001
Subtypes of C. jejuni					
% Same as human ones	100%	78%	52%	14.8	0.001
% Same as most frequent human ones (≥ 2 isolates)	56%	35%	24%	6.5	0.01

For actual numbers see Table 7.1

Table 7.3: Comparison between human, livestock and wild animal (duck) isolates of *Campylobacter* (derived from data in: (Devane et al, 2005) and excluding those that were untypable)

	Human faeces	All livestock (faeces, offal, carcass)	Duck faeces	Chi square for trend	p-value
Species					
% *C. jejuni*	90%	81%	98%	1.33	0.25
Heat stable serotype					
% Same as human ones	100%	87%	47%	37.5	< 0.00001
% Same as top 5 human ones	71%	65%	13%	30.8	< 0.00001
***C. jejuni Sma*-I-PFGE types**					
% Same as human ones	100%	70%	24%	19.9	0.00001
Subtypes of *C. jejuni*					
% Same as human ones	100%	78%	33%	15.4	0.00008
% Same as most frequent human ones (≥ 2 isolates)	56%	35%	11%	7.8	0.005

For actual numbers see Table 7.1

Studies of a river catchment

One of these studies examined the spatial and temporal patterns of *Campylobacter* contamination in the lower Taieri River in Otago (Eyles et al, 2003). It found seasonal variation in *Campylobacter* levels in river water with higher median levels in summer. This season is one when human exposure through recreational water use is considered to be maximal. Changes in river levels of *Campylobacter* appeared to reflect inputs from farms and instream losses (eg, from settling and death).

The study used local notification data (for the local catchment area and Dunedin City) and reported that a "decrease in notified cases of campylobacteriosis in the human population was observed when levels of *Campylobacter* at the main recreational bathing site on the river were low".

This study provides useful information on the *Campylobacter* contamination of river water over a one-year period. However, the temporal relationship between *Campylobacter* contamination levels in water (at the one site out of 10 sites selected for comparison) and notified cases, appears to be weak to this reviewer. Furthermore, there was no statistical modelling undertaken to access this relationship and there were no data presented on the extent to which the local population actually had recreational contact with the river water. Indeed, the proportion could be fairly low for most of the denominator population who would live at least 20 km from the Taieri River in Dunedin City. Also any relationship could partly relate to the use of the Taieri River for drinking water. Even though some of the drinking water supply for

Dunedin City comes from upper parts of the Taieri Catchment and is chlorinated (Eyles et al, 2003) – this may not apply to those living in the catchment itself and the City water supply quality control may not always be effective. Finally, there are many other environmental and behavioural risk factors that may differential impact on the seasonal risk of campylobacteriosis (as discussed in Section 4).

As part of a PhD thesis, Eyles compared typing results from local human isolates with those from the water in the Taieri Catchment (Eyles, 2003). The extent of overlap depended on the typing method: 62% for serotyping, 19% for PFGE with *Kpn*I, 10% for PFGE with *Sma*I, and 1.5% for combined Penner: *Kpn*I subtypes. The author's interpretation was that the mid-range results were likely to be the most informative and therefore exposure to freshwaters may explain 10-20% of campylobacteriosis in this population. The possibility of human contamination of the water was considered an unlikely source (as opposed to animal sources) though it was noted that there are three sewage outflows in an upper part of the catchment. Also recreational human use of the river "could also result in low levels of microbial contamination". But other limitations with drawing conclusions from these results comes from literature cited by Eyles suggesting that there is genetic instability of the *Campylobacter* genome (eg, uptake of extracellular DNA and DNA recombination) that limits the capacity to interpret such laboratory results. Furthermore, these results were not analysed to determine statistical significance.

Other studies of environmental reservoirs and potential transmission routes

A New Zealand study using genotyping found that nearly half (49.7%) of human isolates typed were indistinguishable from poultry isolates (Kakoyiannis et al, 1988). The study also found that rats were infected with strains of *C. jejuni* with patterns "indistinguishable from those infecting humans, poultry and a horse". However, pigs were found to be only a minor source of *C. coli* infection for humans and none of the isolates of *Campylobacter* spp. from wild birds gave patterns similar to those of isolates from humans.

Another New Zealand study published in 1989 measured *Campylobacter* isolation from dairy herds over three seasons (Meanger & Marshall, 1989). It found high isolation rates in summer (24%), autumn (31%), but low in winter (12%). It also found that the same genotypes of *C. jejuni* and *C. coli* were found in sheep and dairy cows on the same farm. Such a finding is suggestive of cross infection between these two livestock species. While this study found no correlation between farm animal and human genotypes of *C. jejuni* (from a collection of 60 human specimens) the authors suggested that this was due to the limited scale of the study.

In 2003, a New Zealand study of environmental reservoirs of *Campylobacter* identified that *C. jejuni* was commonly found in faeces from dairy cows, beef cattle, sheep and ducks, chicken carcasses, sheep offal and surface waters (Savill M. et al, 2003). Of note was that the "preliminary analysis of Penner types was suggestive of transmission to humans from dairy and beef cattle and possibly from sheep".

A study conducted in the Manawatu region found *Campylobacter* spp. in dairy cow faeces, sparrow faeces (urban and rural), rodent faeces and in whole flies (Adhikari et

al, 2004). It considered that the "identical clones of *C. jejuni* carried by cattle, sparrows, flies and rodents probably indicate a common source of infection" – with asymptomatic carriage by healthy dairy cows "being sufficient to maintain infections within the dairy farm surroundings". From a human risk perspective the authors' noted that "the high level of asymptomatic carriage of *C. jejuni* by dairy cows is a potential source of contamination of the human food chain". The data collection from the boots and aprons of dairy workers was very limited, but was positive for *C. jejuni* in three samples.

Another study examined isolates of raw sheep liver and from human campylobacteriosis cases (Cornelius et al, 2005). It found that more than half (61%) of the *C. jejuni* isolates (n = 106) from liver were of subtypes that were also isolated from human cases. This finding suggested the possibility of this food being a reservoir for human infection.

The Devane et al study also noted previous New Zealand work that has shown some similarities in serotypes between human and bovine and ovine (sheep) sources (ie, (Hudson J. A. et al, 1999) and work by Nicol and Wright). This work also found that "some isolates from human cases were indistinguishable from others isolated from water and raw chicken" (Hudson J. A. et al, 1999). Devane et al also cited work by Nicol indicating similarities between human isolates for Wellington/Hutt Valley in 1997 and a serotype frequently isolated from chicken.

Summary

The largest study on environmental reservoirs in New Zealand identified *Campylobacter* spp. in faecal, food and river water samples in the Ashburton area (a total of 1450 samples). It found *Campylobacter* in ten of 12 matrices studied (ie, all except rabbit and possum). The serotype patterns for livestock sources were more similar to the human ones than were those from water and the wild ducks. Further statistical analyses (done in this review) support the view that in this rural setting, livestock contact and/or consumption of livestock products may be more relevant to human illness than waterborne transmission or exposure to environmental contamination from wild birds. The data comparing the results for offal and faeces are also somewhat suggestive that foodborne transmission is more relevant for human transmission than is faecal contamination of water or land. However, more statistical analysis of these findings is desirable.

A river catchment study conducted over a one-year period found seasonal variation in *Campylobacter* levels in river water with higher median levels in summer. It reported that a "decrease in notified cases of campylobacteriosis in the human population was observed when levels of *Campylobacter* at the main recreational bathing site on the river were low". However, this suggested evidence for possible waterborne transmission to the human population must be regarded as very weak for a number of methodological reasons. Other work on the same catchment was suggestive of some similarities between isolates from the water and for local human cases – but there are various limitations with interpreting this finding.

A number of studies have identified similarities between *Campylobacter* serotypes/subtypes from human isolates and those found in: poultry (Kakoyiannis et al, 1988; Hudson J. A. et al, 1999); in dairy and beef cattle (and possibly sheep) (Savill M. et al, 2003); for sheep liver (Cornelius et al, 2005), and for bovine and sheep (Hudson J. A. et al, 1999) (and from other unpublished work cited by (Devane et al, 2005)). Specific investigations into elevated notification rates have also identified such similarities between human and poultry isolates (see Section 5).

All this work provides additional information, but a key underlying concern is the possible instability of the *Campylobacter* genome which may be eroding the value of such comparisons.

8 Overall Assessment

This final section considers the collective evidence in the preceding sections along with other non-human evidence and international evidence. When considering issues of possible causation, it uses the evidence categories described in the methodology section (Institute of Medicine, 2000).

Is contaminated food a cause of campylobacteriosis in the New Zealand setting?

The relevant evidence for this can be summarised as follows:

Evidence of association (case-control studies): The highly statistically significant findings from the largest and relatively high quality case-control study into sporadic disease are relevant (Eberhart-Phillips et al, 1997). It provides evidence for an association for various forms of poultry, other raw or undercooked meat; and for unpasteurised milk. The consistent and statistically significant findings from the next two largest case-control studies also provide such evidence relating to certain forms of poultry consumption (and one for fast food consumption) (Ikram et al, 1994; Neal G. & Bloomfield, 1997). When considering these studies collectively, the possibility that these results are entirely due to chance, bias, or confounding can be ruled out with reasonable confidence.

Evidence of association (outbreak studies): Foodborne spread has been implicated as the likely cause in nine studies of reasonable quality in the New Zealand setting (Table 5.1, Section 5). There were statistically significant findings for a particular food/s in six such outbreaks. Laboratory comparisons between *Campylobacter* strains in humans and a suspected source food for the outbreak indicated similar patterns in all outbreaks where this was studied. Where this relationship was statistically evaluated in one study, it was highly statistically significant (Simmons et al, 2002b).

Other New Zealand workers have reported that unsafe domestic food handling has been identified in outbreaks involving *Campylobacter* (Lake R. & Simmons, 2001).

Evidence of association (outbreak surveillance data): The outbreak surveillance data for the most recent five-year period indicates that foodborne transmission is described as the most common form of transmission (see Section 6). It was described for 121 outbreaks overall (56%). When considering outbreaks with more detailed evidence there were eight outbreaks with more detailed epidemiological or laboratory evidence for foodborne transmission (8/11 – with some of these overlapping with the outbreak investigation studies detailed above). When considering outbreaks with evidence from environmental investigations, there were 42 that reported foodborne transmission. However, there are various limitations with the quality of this outbreak surveillance data (see Section 6).

Evidence of association (notification data): The case-case comparison analysis involving different enteric diseases (Section 4) is compatible with foodborne transmission having an important role (ie, reporting of "food from a food premise" was significantly higher than for cases with giardiasis, salmonellosis or cryptosporidiosis). In particular, the significantly higher rate for campylobacteriosis relative to salmonellosis, is of note given the evidence for foodborne transmission of salmonellosis in New Zealand (Thornley C. et al, 2002a; Thornley C. N. et al, 2003). However, these data may have a number of biases and so only provide limited evidence.

The notification data include some information that is suggestive of specific foods such as chicken having a role (data from Christchurch and national data on the "probable source"). However, this information is of very limited value.

The strength of association: The association appears to be strong eg, for recent consumption of raw or undercooked chicken the matched odds ratio was 4.52 (95%CI = 2.88 – 7.10) in the largest case-control study. Similarly high odds ratios and rate ratios are apparent in the other studies of sporadic cases and for outbreak investigations involving contaminated food.

The biologic gradient: Some New Zealand evidence for a biologic gradient comes from the largest case-control study (Eberhart-Phillips et al, 1997). It found the following to be significant risk factors: "more than one poultry meal in the last 10 days", "more than one chicken meal in the last 10 days", "preference for chicken liver ≥ 1 / mth", and "preference for chicken pieces ≥ 1 / wk". To some extent the finding around the different levels of risk for well cooked chicken (ie, baked or roasted) versus "barbecued" or "raw or undercooked" chicken also represents a gradient (given that the *Campylobacter* levels of the latter forms of food can be assumed to be higher). Similarly, the low risk associated with frozen chicken found in the MAGIC study also suggests a gradient as there is New Zealand evidence for lower levels of contamination in frozen poultry products compared to raw ones (reviewed in: (Lake R et al, 2003)). International work has found that the attack rate is dose dependent for consumption of *Campylobacter* in milk (Robinson, 1981) and in an experimental setting (Black et al, 1988).

Consistency of association: The association is particularly consistent with regard to undercooked animal products (ie, chicken, chicken livers, and unpasteurised milk). There is some evidence that consuming baked or roasted chicken is protective along with eating chicken in the home. This may be a substitution effect whereby these people are less likely to consume other forms of chicken that are more likely to be inadequately cooked. But it may also reflect selection bias in studies or the impact of "immunity from repeated previous exposures to *Campylobacter* via contaminated poultry eaten at their home" (Friedman et al, 2004).

Some outbreak investigations have occasionally implicated non-animal product foods. However, this could generally be explained by cross-contamination associated with food preparation. Contamination from environmental sources (eg, bird droppings on plant foods) is another possibility in some circumstances.

Biological plausibility: That food consumption can cause campylobacteriosis in New Zealanders is highly biologically plausible since there is ample evidence for various livestock in this country being infected ie, dairy cows, beef cattle, sheep, pigs and poultry (Kakoyiannis et al, 1988; Meanger & Marshall, 1989; Savill M. et al, 2003; Adhikari et al, 2004; Cornelius et al, 2005; Devane et al, 2005). Some of this work also indicates serotype and subtype similarities between *Campylobacter* in various livestock and in humans (Kakoyiannis et al, 1988; Savill M. et al, 2003; Cornelius et al, 2005).

In particular, the in-depth study in Ashburton (Devane et al, 2005) and the further analysis presented in Section 7, provides some evidence that the serotype and subtype patterns for human infection have similarities with those in livestock (relative to water and wild birds). However, this could reflect direct contact with these animals, environmental contamination from their faeces, or consumption of uncooked livestock food products. The evidence favouring a possible role for food consumption comes from the data indicating that the pattern for dairy cattle was more similar pattern than for beef, sheep, pork and chicken. This could reflect the role of unpasteurised milk consumption being more important than undercooked meat consumption in this setting. Also, the data are suggestive that the serotype and subtype patterns in offal are more similar to the human one than for faeces where species-specific data are compared (for beef and sheep).

There is also abundant evidence for *Campylobacter* being detected on certain food products on retail sale in New Zealand. This is especially so for poultry (Gilbert, 1993; Consumers' Institute, 1999; Hudson J. A. et al, 1999; Consumers' Institute, 2003) and reviewed by Lake et al (Lake R et al, 2003). New Zealand evidence also includes evidence for *Campylobacter* on the outside of packets of raw chicken (Wong et al, 2004). Experimental evidence from New Zealand also indicates *Campylobacter* cross-contamination via tongs during the cooking of chicken (Hudson JA. et al, 2003).

The biological plausibility of foodborne campylobacteriosis in New Zealand is also somewhat supported by the evidence that other enteric disease transmission occurs by this route. For example, a review of salmonellosis in New Zealand reported 29 foodborne outbreaks with single foods identified as the outbreak source (Thornley C. et al, 2002a). Chicken was the most commonly reported source vehicle, accounting for 21 (72.4%) outbreaks. A case-control study of salmonellosis in New Zealand also

found that consumption of fast food was a significant risk factor (Thornley C. N. et al, 2003). Some chicken samples in New Zealand have also been reported to be contaminated with *Salmonella* (Gilbert, 1993) and with *E. coli* and vancomycin resistant *Enterococcus faecalis* (Robson, 2002).

Coherence: The New Zealand evidence concerning foodborne transmission appears to be fairly coherent. In particular, the evidence revolves around those specific foods from animal products in which contamination is known to occur. Outbreak investigation and other data also show that foodborne transmission is often associated with sub-optimal food handling practices in food premises and that some people report consuming undercooked or raw animal products (see Sections 5 & 6). For example, in 35% of outbreaks classified as foodborne, "undercooking" of food was reported (Table 6.10). Also, environmental investigation identified critical control point failures linked to the implicated food source in 42 outbreaks.

Others have noted that many opportunities exist for cross-contamination in food catering settings in the New Zealand setting (Johnston et al, 1992). Not all district councils in New Zealand require food handlers to be trained (Kalpokas, 2002). Other reports also indicate that some food outlets have low hygiene gradings eg, for Wellington (Johnson A-M., 2000). This situation has also been described for household settings in New Zealand (Hodges, 1993). A recent pilot survey indicated that around a third of domestic refrigerators are set at temperatures that are higher than ideal (ESR, 2005a).

The New Zealand evidence is also fully compatible with very extensive international evidence that campylobacteriosis can be a foodborne disease (Allos, 2004; Blaser & Allos, 2005). In particular, there is also evidence from overseas case-control studies and outbreaks that have implicated poultry and raw milk consumption (see Section 8).

The reduction in campylobacteriosis in the population as a result of interventions relating to poultry production in Iceland (Stern et al, 2003) provide further evidence. So does the impact of removing poultry from sale in Belgium (Vellinga & Van Loock, 2002) on reducing reported disease in the community along with the resurgence of those rates when poultry sales were re-established.

Temporality: There is evidence from New Zealand outbreak investigations that consumption of contaminated food has preceded infection (within the known incubation period for this disease).

Overall evidence: When considering all the above issues it appears that the Institute of Medicine's criteria for causality are met. That is, there appears to be "sufficient evidence for contaminated food having a causal relationship with campylobacteriosis in the New Zealand setting". While building on this evidence base is desirable, it is probably not a priority for the research agenda to further address the issue of causality (ie, as opposed to clarifying the relative roles of different transmission mechanisms).

Is food the dominant known cause of campylobacteriosis in the New Zealand setting?

The plausible important alternatives to foodborne transmission of campylobacteriosis include: waterborne transmission, person-to-person transmission, zoonotic transmission, and transmission from contaminated environmental sources. There is evidence that "overseas travel" is a risk factor for campylobacteriosis in New Zealanders but the available New Zealand evidence usually indicates that less than 10% of cases have travelled overseas during the incubation period (it was 1.1% in the MAGIC study).

Airborne transmission is very unlikely to be important for transmission to humans, even though it may be relevant for spread between chickens in the farm setting (Berndtson et al, 1996). Similarly, other forms of transmission of campylobacteriosis (ie, sexual, perinatal and via transfusion) appear to be very rare (Blaser & Allos, 2005). Disease outbreaks associated with intentional poisoning have been described in the literature (eg, for salmonellosis) – but these are extremely rare. The evidence for foodborne transmission versus other forms (particularly waterborne) in the New Zealand setting is summarised below:

Evidence from case-control studies: The finding from the largest and relatively high quality case-control study into sporadic disease was that foodborne transmission dominated (Eberhart-Phillips et al, 1997). The combined PAR% for the chicken related variables exceeded 50%. For other raw or undercooked meat or fish the PAR% was 11%, and for any unpasteurised milk it was 7%. Compared to these, the only PAR% for a different transmission mechanism that was 5% or greater was zoonotic transmission (ie, for puppy ownership it was 5%). That is the PAR% for "rainwater as a source for home water supply" was less than 5%, as was "sewerage problems at home".

The Christchurch case-control study found statistically significant associations for various foods but not for "drinking water from a non-urban supply" (ie, p = 0.09) (Ikram et al, 1994). The same applied to the Auckland case-control study ie, statistically significant findings for various foods, but non-significant (p = 0.11) for "having a rainwater supply" (Neal G. & Bloomfield, 1997).

Evidence from outbreak studies: Foodborne spread was the most commonly described "likely" source of outbreaks in the 29 published and unpublished outbreak reports examined (9 versus 6 outbreaks) (Table 5.1, Section 5). In terms of statistically significant findings for particular sources, there were six for foodborne outbreaks versus two for waterborne ones. Similarly when comparing *Campylobacter* strains/serotypes in humans and a suspected source, there was evidence for four foodborne outbreaks compared to only two waterborne ones.

Outbreak reports only rarely described person-to-person transmission (3/27 outbreaks). Zoonotic transmission was mentioned as a possibility in three outbreaks but there was no good evidence for this in any of these outbreaks.

Evidence from outbreak surveillance data: The outbreak surveillance data for the most recent five-year period indicates that foodborne transmission was more frequently described than other forms of transmission (see Section 6). It was described for 121 outbreaks (56%) versus 29 (13%) for waterborne outbreaks. When considering outbreaks with evidence from environmental investigations the respective balance was 42 to 8 outbreaks. When considering outbreaks with more detailed evidence, the respective balance was eight to five outbreaks (though in some both transmission mechanisms were reported). Other forms of transmission reported in the outbreak surveillance data and where an environmental investigation was undertaken included: person-to-person spread (6%), zoonotic (6%), and contact with a contaminated environmental source (6%). However, as detailed in Section 6, there are various limitations with the quality of this outbreak surveillance data.

Evidence from notification data: The case-case comparison analysis involving different enteric diseases (Section 4) is suggestive that for cases with campylobacteriosis the risk factor pattern is significantly closer to enteric diseases that are more generally regarded as foodborne (eg, salmonellosis and yersiniosis) than are those that are generally regarded to be waterborne (eg, giardiasis and cryptosporidiosis). However, these data may have a number of biases and so only provide only limited evidence.

The rural excess in notifications appears to be confined to children (< 15 years) (Baker & Sneyd, 2004). This finding is suggestive of zoonotic transmission or environmental contamination being more relevant for this age group in the rural setting. Also the finding that there is an urban excess for adults (for both notifications and hospitalisations) suggests water contamination is not a particularly important transmission pathway. Since rural water supplies are generally of poorer quality than urban supplies, a rural excess would be expected for adults if water supply was an important transmission route.

There is reasonable evidence that the increase in notified campylobacteriosis in the last decade or so is real (Section 4). During this time there has been substantial progress in improving the microbiological quality of reticulated water supplies in New Zealand (Ministry of Health, 2005). This would tend to count against water quality playing a major role in the increase in notified campylobacteriosis. In contrast, there is evidence for an overall increase in poultry consumption, which nearly doubled during the 1990s in real terms (Statistics New Zealand, 2000). Also the extent to which New Zealanders eat takeaways appears to have increased (Johnson M., 2005). However, other potentially relevant trend data are lacking and so this is all very weak evidence for comparing foodborne and waterborne transmission.

Evidence from environmental reservoir studies: The in-depth study in Ashburton (Devane et al, 2005) and the further analysis presented in Section 7, provide some relevant data, at least for a rural setting. The data on serotypes and subtypes are suggestive that livestock are a more likely source of human infection than water exposure or environmental contamination (at least from the faeces of wild birds such as ducks). Although the similarity between the human and livestock patterns may reflect zoonotic transmission from direct contact or environmental contamination from animal faeces, there is some evidence that consumption of livestock derived

foods is more relevant (eg, including that the patterns in offal were somewhat more similar than those in faeces).

A number of other New Zealand studies have identified similarities between *Campylobacter* serotypes/subtypes from human isolates and those found in: poultry; dairy and beef cattle; and sheep/sheep organs. Specific investigations into elevated notification rates have also identified such similarities between human and poultry isolates (see Section 5).

Consistency with international epidemiological evidence: Recent reviews in infectious disease textbooks and journal articles have highlighted the role of foodborne transmission of campylobacteriosis. For example:

- "Consumption of undercooked poultry is estimated to be responsible for 50% to 70% of sporadic *Campylobacter* infections" (Blaser & Allos, 2005).
- "Increases in the isolation of *Campylobacter* spp. reflect both improved recognition and increased consumption of poultry in recent years" (Blaser & Allos, 2005).
- "The principal route of infection with *C. jejuni* in developed countries is through preparation and consumption of chicken" (Allos, 2004).
- "In industrialised countries, most infections are acquired through the handling and consumption of poultry meat" (Butzler, 2004). "Chickens constitute by far the largest potential source of human infection."
- "Foods of animal origin, in particular poultry, have been identified as significant sources of this enteropathogen as a result of infection and contamination at the pre-harvest and harvest levels" (Moore et al, 2005).
- "Undercooked meats, especially poultry, have been associated with infection. Other vehicles include raw clams, raw or undercooked beef, and unpasteurised cheeses and goat's milk." (Blaser & Allos, 2005).

Furthermore, a recent analysis for England and Wales also concluded that "the most important cause of indigenous foodborne disease was contaminated chicken" (Adak et al, 2005). In this study *Campylobacter* caused more cases of foodborne disease than any other identified pathogen. The largest case-control identified in this review (1316 cases) covering FoodNet sites in seven US states found that the largest population attributable fraction was consumption of chicken prepared at a restaurant (24%), followed by non-poultry meat prepared at a restaurant (21%) (Friedman et al, 2004). Other food-related risk factors were: "ate turkey prepared at a restaurant" (4%), "ate undercooked or pink chicken" (3%), "ate raw seafood" (3%) and "drank raw milk" (1.5%).

Other specific case-control studies of sporadic cases indicate that poultry consumption plays a dominant role eg, in the USA (Deming et al, 1987); in Sweden (Studahl & Andersson, 2000); and in the UK (Neal K. R. & Slack, 1995). A review of 14 case-control studies (after excluding those from New Zealand) reported food as being a risk factor in 13 of them compared to two for water (Neimann et al, 2003). Of the studies identifying foods as risk factors, nine reported poultry, seven reported raw milk, and three reported other foods (sausages, pork, and mushrooms).

In contrast to foodborne transmission is the evidence in review texts and articles on other transmission mechanisms:

- "Infected school-age children rarely may transmit *Campylobacter* infection" (Blaser & Allos, 2005).
- "Transmission from infected food handlers who are asymptomatic is at best uncommon" (Blaser & Allos, 2005).
- "Person-to-person transmission of *C. jejuni* has been reported but is rare. Likewise, transmission via infected food handlers or health care workers has been described but is rare. Unlike *Shigella*, *Giardia*, or hepatitis A infections, there have been no outbreaks of *C. jejuni* infection reported in day care centres or institutions for the mentally retarded" (Allos, 2004).

Quantifying the role of non-food transmission mechanisms appears to be uncommon in the international literature. The large FoodNet case-control study in seven US states found that the non-food population attributable fractions were: "had contact with animal stool" (6%), "had pet puppy" (5%), "drank untreated water from a lake, river, or stream" (3%), "had contact with farm animals (for persons aged 2 to < 12 years)" (2%) (Friedman et al, 2004). In total this study found that the population attributable fractions for food-related risk factors totalled 53.5% and for non-food ones the total was 20%. However, there may also be a lot of country variation for non-food risk factors. For example, areas that are reliant on well water may be particularly vulnerable to waterborne outbreaks eg, after spring run-off and heavy rains. A case-control study in Sweden amongst young children (< 6y) found that the waterborne PAR% totalled 39% (well water and lake/river exposure), food-related totalled 34%, and dog exposure was 30% (Carrique-Mas et al, 2005).

Consistency with international evidence concerning outbreaks: Raw milk has been frequently implicated in campylobacteriosis outbreaks overseas: "Investigations of more than 50 outbreaks indicate that unpasturized (raw) milk is such a vehicle [for transmission]" (Blaser & Allos, 2005). Waterborne outbreaks of *C. jejuni* have also been reported in the United States, Europe, and Israel (Allos, 2004) and contamination of drinking water with faeces from birds or animals is usually the cause. However, data from 11 European countries indicates that foodborne outbreaks dominate over waterborne ones (Takkinen et al, 2003). This survey reported that in 48% of outbreaks food was the likely vehicle of transmission, 15% were attributed to unpasteurised milk, 15% were waterborne and 21% were of unknown cause (or not reported).

Consistency with international laboratory evidence: Danish work has suggested a significant overlap between *Campylobacter* serotypes between human cases and broiler flocks (Petersen et al, 2001). This study also noted that "the relatively low number of wildlife strains with an inferred clonal relationship to human and chicken strains suggests that the importance of wildlife as a reservoir of infection is limited". Another study in the Netherlands obtained *Campylobacter* isolates from random clinical human faecal samples and poultry products (Duim et al, 1999). It found the same genotypes present in both matrices. Various other overseas studies show similarities for human and poultry isolates (Wu et al, 2002) and for human poultry and cattle isolates (On et al, 1998; Nielsen et al, 2000).

Consistency with intervention studies and natural experiments: Evidence for the importance of foodborne transmission of *Campylobacter* also comes from intervention studies and natural experiments. For example, interventions relating to poultry production and processing appear to have reduced disease rates in Iceland

(Stern et al, 2003). There is also the evidence from the impact from a "natural experiment" relating to the removal of poultry products from sale in Belgium that led to a 40% reduction in reported cases (Vellinga & Van Loock, 2002). In the US "decreased rates have been attributed to prevention efforts implemented in food service establishments, meat and poultry processing plants" (Allos, 2004).

Overall evidence: When considering all the above issues it appears that the available evidence indicates that contaminated food is the dominant known cause of campylobacteriosis in the New Zealand setting. This evidence comes from:

- The findings of each of the three largest case-control studies.
- The overall pattern from a review of 29 published and unpublished outbreak investigation reports of relatively high quality.
- The overall patterns found in a review of five years of outbreak surveillance data.
- The notification data (ie, the case-case comparison analyses, the rural versus urban distribution and to a limited extent the time trend data).
- The environmental reservoir study in Ashburton (ie, the comparisons between serotype and subtype patterns in humans, livestock, wild birds and water).
- The compatibility with the international epidemiological data indicating that foodborne transmission is the dominant transmission mode in developed countries.
- The compatibility with the findings from intervention studies and natural experiments in other countries (that show beneficial impacts on disease rates from reducing levels of contaminated food).

Although this evidence for the dominant role of foodborne transmission (relative to waterborne and other known forms) is fairly convincing for the New Zealand setting and other developed countries, the precise extent is hard to determine for this country. A crude working assumption might be that the PAR% estimate from the MAGIC study may not be unreasonable (ie, exceeding 50%). However, it is clear that much further work is required and that the research agenda needs to focus on clarifying the role of foodborne transmission more precisely.

Wider health and economic benefits of reducing *Campylobacter* contamination of food

Health authorities and other government agencies need to consider a number of wider potential benefits of reducing the risk of foodborne campylobacteriosis – in addition to the morbidity prevention benefits. These benefits may increase the justification for making further improvements to surveillance, research and conducting intervention studies. For example, additional health benefits include:

- Improving food handling by professional food handlers and by the general public will reduce the risk for other enteric diseases in New Zealand (eg, salmonellosis).
- Improving the safety profile of poultry foods may facilitate reductions of saturated fat intake in New Zealand, if it further encourages consumers to shift from fatty meats to poultry. This could have benefits in reducing rates of

cardiovascular disease given the highly atherogenic and thrombogenic nature of the New Zealand diet (Laugesen & Swinburn, 2000).

- Improving the safety profile of poultry foods may facilitate reductions of red meat consumption. This may favour a reduction in colon cancer risk given that red meat appears to be associated with this disease and poultry is not – according to a high quality recent cohort study of 478,000 participants (Norat et al, 2005).

Additional economic benefits of reducing foodborne campylobacteriosis include:
- Reductions in lost productivity from sick workers or when parents stay on home to care for sick dependents.
- Reductions in health care costs from doctor visits and hospitalisations.
- Protection of New Zealand's food export markets and the tourism sector (which could both be damaged by adverse media publicity around high rates of campylobacteriosis or if large outbreaks occurred).

Possible actions for health authorities to consider

The evidence for a high and recently increasing burden of campylobacteriosis in New Zealand suggests that further improvements in surveillance of sporadic cases, outbreak surveillance and investigations, and other research into the epidemiology of this disease should be a public health priority. The following options could be considered by the relevant government agencies and local health authorities, particularly the Ministry of Health and the NZ Food Safety Authority:

1. Improvements in **disease surveillance** through various changes to the notifiable disease surveillance system (see the discussion part of Section 4).

2. Conducting further studies of the **notification data** such as case-case studies (see the discussion part of Section 4).

3. Improvements in the quality of **outbreak investigations** (see the discussion part of Section 5).

4. Improvements in **outbreak surveillance** (see the discussion part of Section 6).

5. Consideration of **intervention studies** directed at food sources in one region (eg, covering 10-20% of the population). These could be justified on the basis of the success of interventions and natural experiments in Iceland, Belgium, and the USA (as detailed above). Furthermore, it has been reported that 60% of Swedish farms consistently produce batches of broilers without *Campylobacter* (report cited in the review by (Lake R et al, 2003)). Careful monitoring could be used to determine if interventions in New Zealand had a significant impact in the targeted region, relative to the rest of the country (eg, in terms of food contamination levels and incidence of campylobacteriosis and perhaps salmonellosis as well). Some possible options for interventions in the target region include:

- Intensifying specific measures to reduce the infection levels in broiler poultry. Some of the possibilities have been reviewed (Lake R et al, 2003) and are detailed in the international literature eg, (Evans, 1992).

- Changing food processing techniques – particularly concerning refrigeration and freezing of poultry products eg, the NZFSA is exploring this approach already (Collins, 2005).

- Promoting the use of alternatives to the sale of *fresh* poultry (ie, promoting just cooked or frozen poultry) to consumers via a mass media campaign in the target region. This potential benefit of a shift towards the use of frozen poultry has been suggested previously (Lake R et al, 2003).

- Requiring that publicly funded institutions in the target region restrict use of poultry products to only frozen items (eg, all public hospitals, rest homes, prisons etc).

- Tightening food premise licensing requirements in the target region so that the sale of poultry products in those premises that are in breech of food safety requirements are banned (eg, for a one-year period).

- Tightening labelling requirements in the target region so that food handling information and warnings are more prominent on all forms of poultry.

- Requiring higher quality packaging of poultry products (ie, to reduce the number of holes in plastic packaging of for-sale poultry products).

6. Consideration of **supportive national level actions** such as:

- Adopting a national health objective of a much lower incidence of campylobacteriosis eg, the US objective is 12.3 cases per 100,000 population by 2010 (Friedman et al, 2004).

- Imposing a research levy on the poultry industry to fund improvements in campylobacteriosis surveillance, research and intervention studies. This approach could be similar to the New Zealand agricultural sector funding research on how to reduce greenhouse gas emissions from livestock. Some of this research funding could go towards developing *Campylobacter* vaccines for animals and/or humans – given that there is some promising data from animal models (Butzler, 2004).

- Continuing to upgrade the quality of community water supplies and to promote greater public awareness of the importance of protecting water supplies from contamination (eg, domestic roof water and well water supplies).

Others have raised some of these options before. For example, it has been suggested previously that there be an intervention study to control campylobacteriosis in a rural area (Baker et al, 2002).

The probable cost of some of approaches has not been estimated here. Nevertheless, many of them may be justifiable from a public health perspective in the context of the

high morbidity burden that currently exists from campylobacteriosis in the New Zealand setting.

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