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ANNUAL REPORT
CONCERNING FOODBORNE DISEASE
IN NEW ZEALAND
2006

Prepared as part of a New Zealand Food Safety Authority
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by

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1   INTRODUCTION

The New Zealand Food Safety Authority (NZFSA) has an aim to reduce food-related risks to human health. Its Science Strategy has identified human health surveillance as an essential element of the monitoring and review component of its risk management framework. In addition evidence from notifications, case enquiries, outbreak investigations and other epidemiological studies of human enteric diseases are being increasingly used as sources of data for risk assessments. There is increasing interest in foodborne disease statistics within NZFSA and its stakeholders.

This report for the calendar year 2006 is intended to be the first of a series providing a consistent source of data and method of presentation to allow monitoring of foodborne illness in New Zealand.

1.1   Human Health Surveillance Data and Foodborne Disease

The information in this report concerns reported cases of notifiable disease and reported outbreaks. There are a number of notifiable illnesses which may be caused by transmission of pathogens in foods, but it is important to remember that most of the information concerns the illness, not the mode of transmission. The information needs to be considered with two caveats:

1. Notified cases of illness and reported outbreaks represent a subset of all the cases and outbreaks that occur in New Zealand each year. Many cases do not visit a GP or otherwise come to the attention of the medical system. By using these data as indicators, we are assuming that they are representative of all the cases and outbreaks that occur (see section 3 of this report).
2. Foodborne transmission is only one of the routes by which humans are exposed to pathogens; other routes include water, animal contact and person to person. There are a number of indicators from which we can get information on the proportion of cases caused by foodborne transmission:

   - Reported risk factors: for a proportion of the notified cases, supplemental information is obtained by Public Health Units on risk factors. This information should be interpreted with some caution as it is self reported by cases, no external validation of this information is undertaken, and has limited value as it is not critically evaluated in any way, and often the cases will report several potentially important risk factors. The quality of information from notifiable disease surveillance as an indication for foodborne disease transmission has been reviewed in more detail (Lake *et al.*, 2005).
   - Outbreak reports: the circumstances of an outbreak (multiple cases from a single event) means that investigation is more likely to identify a source of exposure to the pathogen. However, only a small proportion of outbreaks are reported, and experience shows that outbreaks associated with a foodservice premise are more likely to be reported and investigated.
   - Expert opinion: based on their experience in laboratories and epidemiological investigations, as well as knowledge of factors influencing the risk, experts can provide estimates of the proportion of cases caused by foodborne transmission. Estimates for New Zealand have been developed for some foodborne diseases, as presented in relevant report sections. These are not fixed values; changes to the New Zealand food chain may require the values to be amended.

- Overseas analyses and estimates: information for countries with similar food supplies to New Zealand can be helpful, especially for illnesses where a foodborne estimate was not developed. Two sets of published expert opinion estimates are given in Table 1, for the USA (Mead *et al.*, 1999) and Australia (Hall and Kirk, 2005). It is worth noting that although for most of the diseases included in this report foodborne transmission is considered significant, there are several illnesses (shigellosis, giardiasis, cryptosporidiosis, infection with Hepatitis A) it is considered only a small proportion of the total.

Table 1: Overseas estimates of the food attributable proportion of selected microbial diseases

Illness/hazard	USA % Foodborne	Australia % Foodborne
Bacteria		
Bacillus cereus	100	100
Campylobacter spp.	80	75
Clostridium perfringens	100	100
E. coli O157:H7	85	65
Listeria monocytogenes	99	NE
Salmonella non-typhoidal	95	87
Shigella spp.	20	10
Staphylococcus food poisoning	100	100
Yersinia enterocolitica	90	75
Parasitic		
Cryptosporidium parvum	10	10
Giardia lamblia	10	5
Viral		
Hepatitis A virus	5	NE

NE = not estimated

This report considers information for the 2006 calendar year. Information from the scientific literature and other sources concerning food safety for that year have been collated and summarised. However, the time taken to publish scientific information is often lengthy, and it may be that additional information becomes available in the future.

1.2 Conditions Included in Report

The conditions that have been selected for inclusion in the report are those that have:
a) The potential to be caused by foodborne transmission; and,
b) Available historical and current national data sources.

The potentially foodborne conditions that were selected for inclusion in this report are listed in Table 2. Data have been drawn from a number of sources including disease notification, hospitalisation, outbreak report and laboratory surveillance databases

Table 2:   Potentially foodborne conditions included in the report

Disease	Type	Source(s)	ICD*-10 code
Bacillus cereus intoxication	Bacterium	N, O, H	A05.4 Foodborne *Bacillus cereus* intoxication
Campylobacteriosis	Bacterium	N, O, H	A04.5 *Campylobacter* enteritis
Ciguatera poisoning	Toxin	N, O, H	T61.0 Toxic effect: Ciguatera fish poisoning
Clostridium perfringens intoxication	Bacterium	N, O, H	A05.2 Foodborne *Clostridium perfringens* [*Clostridium welchii*] intoxication
Cryptosporidiosis	Protozoan	N, O, H	A07.2 Cryptosporidiosis
Giardiasis	Protozoan	N, O, H	A07.1 Giardiasis [lambliasis]
Hepatitis A virus infection	Virus	N, O, H	B15 Acute hepatitis A
Listeriosis (total and perinatal)	Bacterium	N, O, H	A32 Listeriosis
Norovirus infection	Virus	O, H	A08.1 Acute gastroenteropathy due to Norwalk agent
Salmonellosis	Bacterium	N, O, H, L	A02.0 *Salmonella* enteritis
Scombrotoxicosis	Toxin	N, O	T61.1 Toxic effect: Scombroid fish poisoning
Shigellosis	Bacterium	N, O, H, L	A03 Shigellosis
Staphylococcus aureus intoxication	Bacterium	N, O	A05.0 Foodborne staphylococcal intoxication
STEC/VTEC infection	Bacterium	N, O, L	A04.3 Enterohaemorrhagic *Escherichia coli* infection
Toxic shellfish poisoning	Toxin	N, O	T61.2 Other fish and shellfish poisoning
Yersiniosis	Bacterium	N, O, H	A04.6 Enteritis due to *Yersinia enterocolitica*

Data Sources: EpiSurv notifications (N), EpiSurv outbreaks (O), NZHIS hospitalisations (H), ESR laboratory data (L)
* International Classification of Diseases

The notifiable conditions were selected for inclusion in the report where it was considered that a significant proportion would be expected to be foodborne or the disease organism has been reported as the cause of foodborne outbreaks. *Salmonella* Typhi and *Salmonella* Paratyphi are not included as the majority of cases acquire their infection overseas.

For some diseases (intoxications from *Bacillus*, *Clostridium* and *Staphylococcus* bacteria, and norovirus infection) not every case is notifiable; only those that are part of a common source outbreak.

For some conditions (campylobacteriosis, listeriosis, salmonellosis, VTEC/STEC infection, yersiniosis) the attribution of disease incidence to foodborne transmission was estimated by an expert consultation held on 24 May 2005 (Cressey and Lake, 2005). In the current report the proportions of food-associated cases, derived from expert consultation, have been use to estimate the number of food-associated cases of relevant diseases. In this process it has been assumed that travel-associated cases can be removed from the total cases before application of the food-associated proportion.

This report includes both notifiable diseases in the form of acute gastrointestinal illness, and sequelae which are considered to result from these preceding infections (Table 3). The two sequelae included in the report, haemolytic uraemic syndrome (HUS) and Guillain-Barré Syndrome (GBS) are severe illnesses and often life threatening.

Table 3:   Sequelae to potentially foodborne conditions included in the report

Disease	Source(s)	Comment
Guillain-Barré Syndrome (GBS)	H (G61.0 Guillain-Barré syndrome)	Sequelae following infection with *Campylobacter*
Haemolytic-uraemic syndrome (HUS)	H (D59.3 Haemolytic-uraemic syndrome)	Sequelae to infection with Shiga toxin producing *E. coli*

Data Sources: NZHIS hospitalisations (H)

The data sources above have been selected on the basis of availability of data for the specified reporting period and their availability within the timeframe required for the report.

Some data such as official cause of death are not published until several years after the end of the year in which the event occurred (although deaths may be reported as part of the case notification data recorded in EpiSurv). For this reason these data cannot be included in a report published soon after the end of the calendar year.

2 METHODS

This section includes descriptions of the data sources, and analytical methods used and comments on quality of data (including known limitations).

The report uses the calendar year (1 January to 31 December 2006) for the reporting period(s).

2.1 Data Sources

The key sources of data used in this report are detailed in the following sections.

2.1.1 EpiSurv - the New Zealand notifiable disease surveillance system

Under the Health Act 1956 health professionals are required to inform their local Medical Officer of Health of any notifiable disease that they suspect or diagnose. Notification data are recorded using a web-based application (EpiSurv) available to staff at each of the 20 public health units (PHUs) in New Zealand. These data are transferred to the Institute of Environmental Science and Research (ESR) Ltd., where they are collated, analysed and reported on behalf of the Ministry of Health. Further information about notifiable diseases can be found in the 2006 Annual Surveillance Report (Anonymous, 2007a).

2.1.2 Laboratory-based surveillance

The reference laboratories at ESR maintain databases of laboratory results for notifiable diseases.

The number of laboratory reported salmonellosis cases has until recently always exceeded the number of notifications. The implementation of integration processes in 2004 for notifications and laboratory results at ESR has addressed this problem.

2.1.3 New Zealand Health Information Service (NZHIS)

NZHIS in the Ministry of Health collates national data on patients admitted and discharged from publicly funded hospitals. These data are stored as part of the National Minimum Dataset (NMDS). Cases are assigned disease codes using the tenth revision of the International Classification of Diseases (ICD-10) coding system. Up to 99 diagnostic, procedure, and accident codes may be assigned to each admission. The first of these is the principal or primary diagnosis, which is the condition that actually led to admission. This may differ from the underlying diagnosis.

Hospital admission data include repeated admissions for patients with chronic notifiable diseases e.g. tuberculosis or diseases which have long-term health impacts e.g. meningococcal disease. For some diseases the criteria for notification (clinical and laboratory or epidemiological evidence) do not match those required for diagnostic coding. For these reasons hospitalisation numbers and notifications may differ. In this report hospitalisations, including readmissions, have been reported for all primary disease. For the disease sequelae Guillain-Barré Syndrome (GBS) and Haemolytic-uraemic Syndrome (HUS), for which there is potential for multiple readmissions, hospitalised cases have been reported.

2.1.4 Outbreak surveillance

ESR has operated an outbreak surveillance system in EpiSurv since mid 1997. This enables PHUs to record and report outbreaks for national reporting and analysis. In particular it should be noted that not all cases associated with outbreaks are recorded as individual cases of notifiable disease in EpiSurv. The terms 'setting' and 'suspected vehicle' are used in outbreak reporting to describe likely sources. More information about outbreak reporting system can be found in the 2006 Disease Outbreak Report (Anonymous, 2007b).

2.1.5 Statistics New Zealand

Data from the Statistics New Zealand website www.stats.govt.nz was used to calculate notification and hospitalisation population rates of disease. See analytical methods section for further details.

2.1.6 NZFSA project reports and publications

NZFSA project reports, prepared by ESR or other providers, and publications from the general literature were used to provide specific contextual information on the prevalence of selected pathogens in specific food types.

2.1.7 Risk attribution

Information from a NZFSA project on risk ranking was used to estimate the proportion of disease due to specific pathogens that can be attributed to transmission by food (Cressey and Lake, 2005). Attributable proportions were determined by expert consultation, using a modified double-pass Delphi, with a facilitated discussion between passes. Each expert was asked to provide a minimum ('at least'), a most likely and a maximum ('not more than') estimate of the proportion of a number of microbial diseases that were due to transmission by food. Estimates presented in the current report are mean values from the second pass.

2.2 Analytical Methods

Key analytical methods used include:

2.2.1 Dates

Notification data contained in this report are based on information recorded in EpiSurv as at 1 December 2007. Changes made to EpiSurv data by PHU staff after this date will not be reflected in this report. Consequently, future analyses of these data may produce revised results. Disease numbers are reported according to the date of notification. Laboratory results are reported according to the date the specimen was received.

2.2.2 Data used for calculating rates of disease

All population rates use Statistics New Zealand mid year population estimates as at 30 June 2007 and are crude rates unless otherwise stated.

2.2.3 Geographical breakdown

This report provides rates for current District Health Boards (DHBs). The DHB populations have been derived from the Statistics New Zealand mid year population estimates for Territorial Authorities in New Zealand.

2.2.4 Map classification scheme

The maps classification for the disease rates is quantiles i.e. the data have been divided into three groups containing equal numbers of DHBs. The darkest colour represents the highest rates and the lightest colour the lowest rates. The grey colour shows where there are insufficient data to calculate a rate (less than 5 cases).

2.2.5 Risk factors and source of infection

For many diseases an analysis of exposure to risk factors for the cases is reported. The risk factor questions on the EpiSurv case report forms are those that are currently known for that disease. Often more than one risk factor is reported for each case. The high number of unknown outcomes associated with the risk factors should be noted.

The reporting of exposure to a risk factor does not imply that this was the source of the infection.

2.2.6 Statistical tests

Confidence intervals have been calculated for the disease rates and displayed on the graphs. The historical mean is calculated from the previous three years data.

2.3 Interpreting Data

Data in this report may differ from those published in other reports depending on:
- the date of extraction of data
- the date used to aggregate data (e.g. date reported or date of onset of illness)
- filters used to extract the data

The information in this report shows disease trends by age group, sex, and place of residence (District Health Board).

Because of the low numbers of cases for some conditions and age groups, etc. the rates calculated in this report may be highly variable from year to year and it is necessary to interpret trends with caution.

3   THE ACUTE GASTROINTESTINAL ILLNESS (AGI) STUDY

The Acute Gastrointestinal Illness (AGI) Study is a set of three linked surveys, with the following objectives:

- To determine the magnitude and distribution of self reported AGI in the New Zealand population;
- To estimate the burden of disease associated with AGI;
- To describe and estimate the magnitude of under-ascertainment of AGI at each stage in the national communicable disease surveillance process; and,
- To identify modifiable factors affecting under-ascertainment that, if altered, could reduce case loss throughout the AGI component of the surveillance system.

The three study elements were completed during 2005-2007 and each has been reported separately (available from the NZFSA website: http://www.nzfsa.govt.nz/science/research-projects/index.htm):

- Community study: a twelve month telephone survey conducted from February 2006 – January 2007 and reported as "Acute Gastrointestinal Illness (AGI) Study: Community Survey" (Adlam *et al.*, 2007),
- General practice study: a nationwide incidence study conducted over seven weeks from May – July 2006, using selected practices via a computer network practice management system, supplemented by a postal survey conducted in July 2006. This study has been reported as "Acute Gastrointestinal Illness (AGI) Study: General Practice Study" (Perera and Adlam, 2007), and
- Laboratory study: a postal survey of 45 community and hospital laboratories conducted in June 2006, and reported as "Acute Gastrointestinal Illness (AGI) Study: Laboratory Survey" (King *et al.*, 2007).

The results from the Community survey indicated that the incidence of AGI was 1.12 per person year, representing 4.6 million cases in New Zealand in one year. These illnesses are caused by microbial hazards that may be transmitted by a number of routes, including foods. However, at this stage it is not possible to identify the total fraction of AGI caused by foodborne transmission.

A Final report amalgamated results from the three studies to construct a reporting pyramid for AGI in New Zealand, as shown in Figure 1 (Lake *et al.*, 2007). It is important to recognise that this pyramid applies to AGI in its entirety, and cannot be applied to AGI caused by individual pathogens, which may have quite different ratios.

Figure 1:   **Reporting pyramid (areas to scale) for New Zealand using data from the AGI study***

1	Reported to Notification System
3.5	Positive faecal test results
17.5	Faecal specimens submitted
19	Faecal specimens requested
48	Visits a GP
219	Community Cases
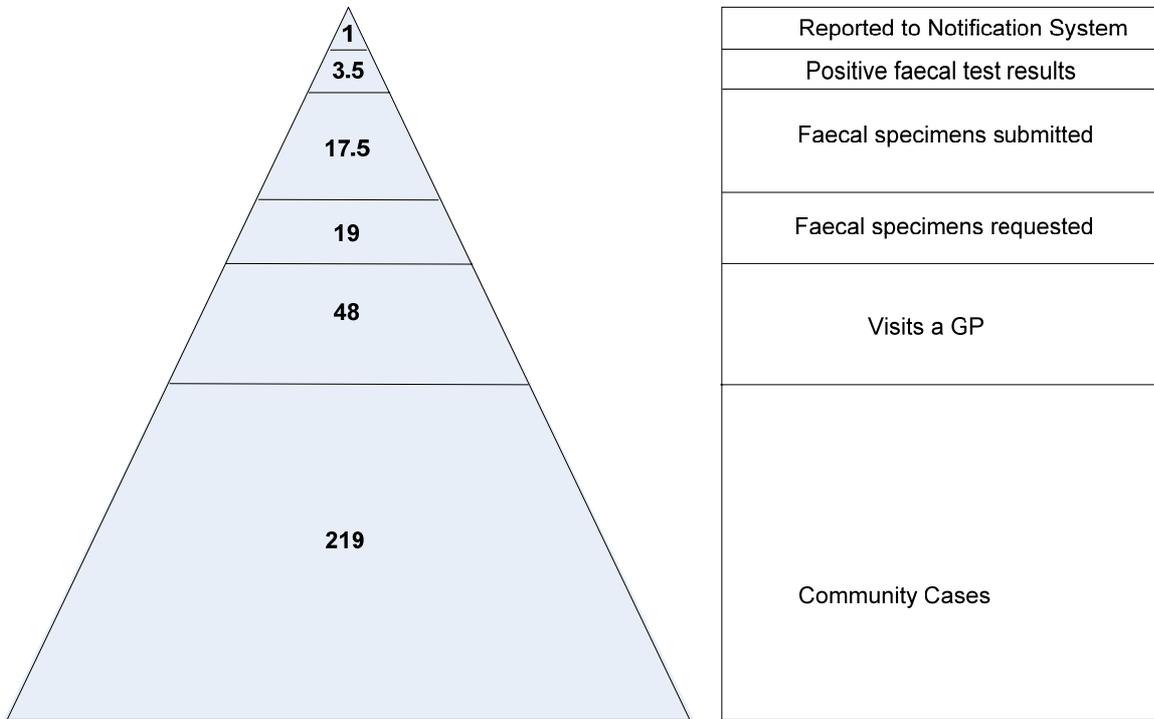
* The reporting pyramid is constructed from data reported from the community survey (Adlam *et al.*, 2007); GP survey (Perera and Adlam, 2007); and laboratory survey (King *et al.*, 2007).

Note that not all positive faecal test results will be for diseases that are notifiable.

4   REPORTING

4.1   Reporting Against Targets

The NZFSA have established three organisational targets, which this and subsequent reports will monitor progress towards:

- 50% reduction in NZ acquired foodborne campylobacteriosis over 5 years
- 30% reduction in NZ acquired foodborne salmonellosis over 5 years
- No increase in NZ acquired foodborne listeriosis over 5 years

The year 2007 will be the baseline. Consideration will also be given to numbers and percentages of cases who had travelled overseas during the incubation period of each disease.

4.2   Incidence and Severity of Selected Foodborne Diseases

This section includes a summary for each potential foodborne condition. For conditions with sufficient numbers (approximately 100 cases or more per year) full analysis, drawn from notification, hospitalisation, mortality, and laboratory data, has been carried out. For diseases with a small number of cases a more limited analysis has been carried out.

These data have been followed by contextual information on the foodborne proportion of the overall incidence of illness. This section will include information on the following topics, where available:

- Statement of estimated foodborne percentage and range provided by an expert elicitation process conducted in 2004-2005. Note that these estimates are only available for some of the illnesses included in this report;
- Statement of estimated foodborne percentage and range for any specific foods provided by the same expert elicitation process;
- Information on pathogen typing (principally from data generated by the Enteric Reference Laboratory), where it is available and informative about foodborne disease;
- Comments on specific food related incidents or outbreaks of the disease that were reported to the notification system during the calendar year;
- Studies on foodborne attribution for the specific disease conducted or published during the calendar year;
- Information on the prevalence of the chemical or microbial hazard in particular foods as a result of surveys conducted during the calendar year; and,
- Regulatory or other risk management actions in New Zealand that might be expected to affect the foodborne disease data.

4.3   *Bacillus cereus* Intoxication

4.3.1   Case definition

Clinical description: Gastroenteritis where either vomiting or profuse watery diarrhoea dominate

Laboratory test for diagnosis: Isolation of ≥10^3/g *B. cereus* from a clinical specimen or ≥10^4 *B. cereus* from leftover food or detection of diarrhoeal toxin in a faecal sample

Case classification:

Probable   A clinically compatible illness that is either a contact of a confirmed case of the same disease, or has had contact with the same common source i.e., is part of an identified common source outbreak

Confirmed   A clinically compatible illness that is laboratory confirmed

4.3.2   *Bacillus cereus* intoxication cases reported in 2006 by data source

In 2006 two notifications of *Bacillus cereus* intoxication and no resulting deaths were reported in EpiSurv.

The ICD-10 code A05.4 was used to extract *Bacillus cereus* intoxication hospitalisation data from the NZHIS NMDS database. There were 6 hospital admissions (0.1 admissions per 100 000 population) recorded in 2006 with *Bacillus cereus* intoxication as another relevant diagnosis.

Expert consultation estimated that 97% (minimum = 90%, maximum = 99%) of *Bacillus cereus* intoxication will be due to foodborne transmission. The expert consultation also estimated that approximately 60% of the foodborne transmission would be due to consumption of rice.

4.3.3   Outbreaks reported as caused by *Bacillus cereus*

All *Bacillus cereus* outbreaks reported in 2006 were foodborne (Table 4)

Table 4:   *Bacillus cereus* outbreaks reported, 2006

Measure (No.)	Foodborne *Bacillus cereus* outbreaks	All *Bacillus cereus* outbreaks
Outbreaks	2	2
Cases	11	11
Hospitalised cases	0	0

From 2004 to 2006, fewer outbreaks and cases of foodborne *Bacillus cereus* intoxication were reported each year in EpiSurv than in any of the four years prior to 2004 (Figure 2).

Figure 2: **Foodborne *Bacillus cereus* outbreaks and associated cases reported by year, 2000–2006**
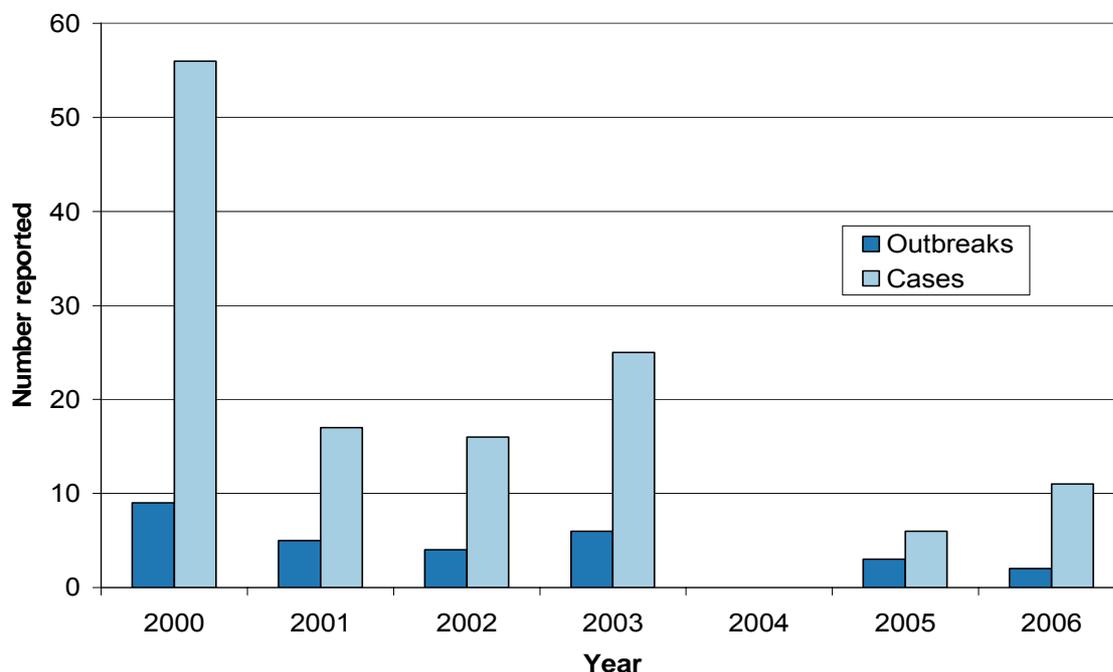
4.3.3.1 Details of food-associated outbreaks

Table 5 contains details of the two food –associated *B. cereus* intoxication outbreaks reported in 2006.

Table 5: **Details of food-associated *Bacillus cereus* outbreaks, 2006**

Public Health Unit (Month)	Suspected vehicle	Setting	Number ill	Confirmation
Rotorua (February)	Rice	Takeaway	3C	2, 5
Rotorua (February)	Rice	Takeaway	8C	2, 5

C = confirmed, P = probable
Confirmation:
1 = Environmental investigation – identified critical control point failures linked to implicated source
2 = Epidemiological – case had history of exposure to implicated source
3 = Epidemiological – case control or cohort study showed elevated risk for cases to implicated source
4 = Laboratory – pathogen suspected to have caused illness identified in food handler
5 = Laboratory – pathogen suspected to have caused illness identified in implicated source (food)
6 = No evidence
7 = Other evidence

The suspected vehicles for both outbreaks are consistent with expert opinion, that rice is the predominant cause of foodborne *Bacillus cereus* intoxication. The two outbreaks were linked.

4.3.3.2 Laboratory investigation of samples from suspected foodborne outbreaks

During investigations of suspected foodborne illness outbreaks by ESR's Public Health Laboratories, *Bacillus cereus* was isolated from rice samples from one of the outbreaks reported in Table 5. High levels of *B. cereus* were also isolated from a faecal specimen, for which the

suspected vehicle was a Chinese meal from a takeaway outlet. Low levels of *B. cereus* were also detected in raw mussels associated with an outbreak, but was not considered to be the causative organism.

4.3.4 Relevant New Zealand studies and publications

A study assessed the frequency and concentration of *B. cereus* in dehydrated potato flakes and hot-held, ready-to-eat mashed potato products (Turner *et al.*, 2006). Of 50 packets of potato flakes tested, eight contained greater than 100 CFU/g *B. cereus* (maximum 370 CFU/g). The temperature of the potato portion of 44 hot-held food products was measured immediately after purchase, and 86% were below the safe hot-holding temperature of 60°C. The potato portions were subsequently tested for *B. cereus.* Only two of the potato portions contained *B. cereus* at greater than 100 CFU/g, a potato-topped pastry (1,000 CFU/g) and a container of potato and gravy (120 CFU/g). To assess multiplication of *B. cereus* in this food, rehydrated potato flakes with naturally occurring *B. cereus* were held at 37, 42, and 50°C and tested over 6 h. By 6 h, the number of *B. cereus* in potato stored at 37°C had exceeded 10^3 CFU/g, was greater than 10^4 CFU/g at 50°C, and was close to 10^6 CFU/g at 42°C.

4.3.5 Relevant regulatory developments

Nil.

4.4 Campylobacteriosis

Summary data for campylobacteriosis in 2006 are given in Table 6.

Table 6:   Summary surveillance data for campylobacteriosis, 2006

Parameter	Value in 2006	Section reference
Number of cases	15 873	4.4.2
Rate (per 100 000)	379.3	4.4.2
Hospitalisations (%)	1 179 (7.4%)	4.4.2
Deaths (%)	1 (0.006%)	4.4.2
Estimated travel-related cases (%)	956 (6.0%)	4.4.3.6
Estimated food-related cases (%)*	8 652 (58%)	4.4.2

* For estimation of food-related cases it was assumed that the proportions derived from expert consultation would exclude travel-related cases

4.4.1 Case definition

Clinical description:	An illness of variable severity with symptoms of abdominal pain, fever and diarrhoea, and often bloody stools
Laboratory test for diagnosis:	Isolation of *Campylobacter* from a clinical specimen
Case classification:	
Probable	A clinically compatible illness that is either a contact of a confirmed case of the same disease, or has had contact with the same common source i.e., is part of an identified common source outbreak
Confirmed	A clinically compatible illness that is laboratory confirmed

4.4.2   Campylobacteriosis cases reported in 2006 by data source

During 2006, 15 873 notifications (379.3 cases per 100 000 population) of campylobacteriosis were reported in EpiSurv.

The ICD-10 code A04.5 was used to extract campylobacteriosis hospitalisation data from the NZHIS NMDS database. Of the 1 179 hospital admissions (28.2 admissions per 100 000 population) recorded in 2006, 967 were reported with campylobacteriosis as the primary diagnosis and 212 with campylobacteriosis as another relevant diagnosis.

One death due to campylobacteriosis was recorded in EpiSurv in 2006.

It has been estimated by expert consultation that 58% (minimum = 37%, maximum = 70%) of campylobacteriosis incidence is due to foodborne transmission. It was further estimated that 53% of foodborne transmission would be due to transmission via poultry.

4.4.3   Notifiable disease data

4.4.3.1   Annual notification trend

The number of campylobacteriosis notifications reported each year has generally increased since 1996, with the 2006 being the highest recorded (Figure 3).

Figure 3:   Campylobacteriosis notifications by year, 1996-2006
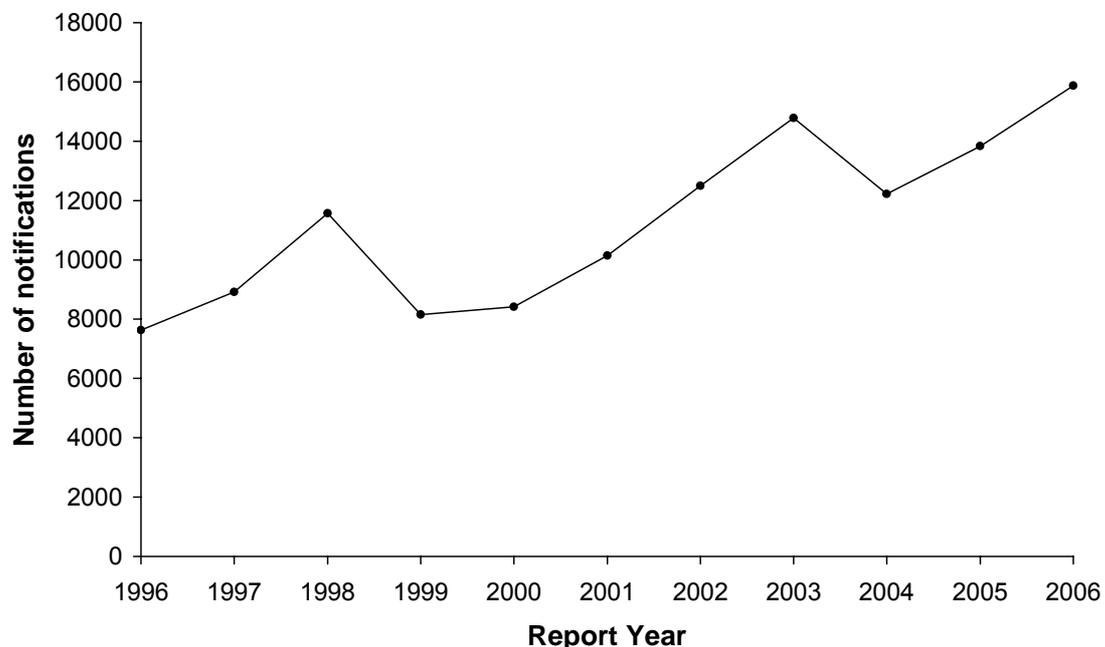
The campylobacteriosis annual rate trend (Figure 4) was very similar to the corresponding annual notification trend; with a general increase in the notification rate observed over the period monitored.

Figure 4: **Campylobacteriosis notification rate by year, 2000-2006**
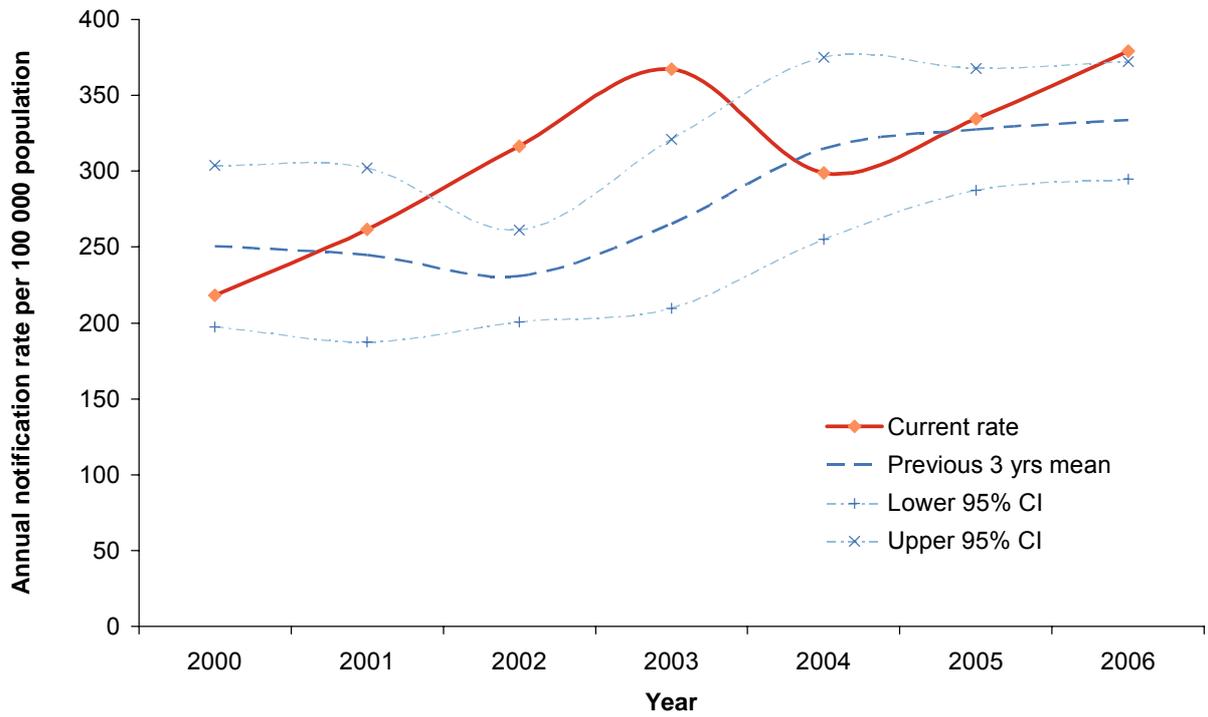
4.4.3.2 Seasonality

The number of notified cases of campylobacteriosis per 100 000 population by month for 2006 is shown in Figure 5. Campylobacteriosis is highly seasonal with a summer peak and winter trough. The pattern in 2006 was different in that there was a second peak in early winter (May/June). The highest monthly campylobacteriosis rate for 2006 was in November.

Figure 5:   Campylobacteriosis monthly rate (annualised) for 2006
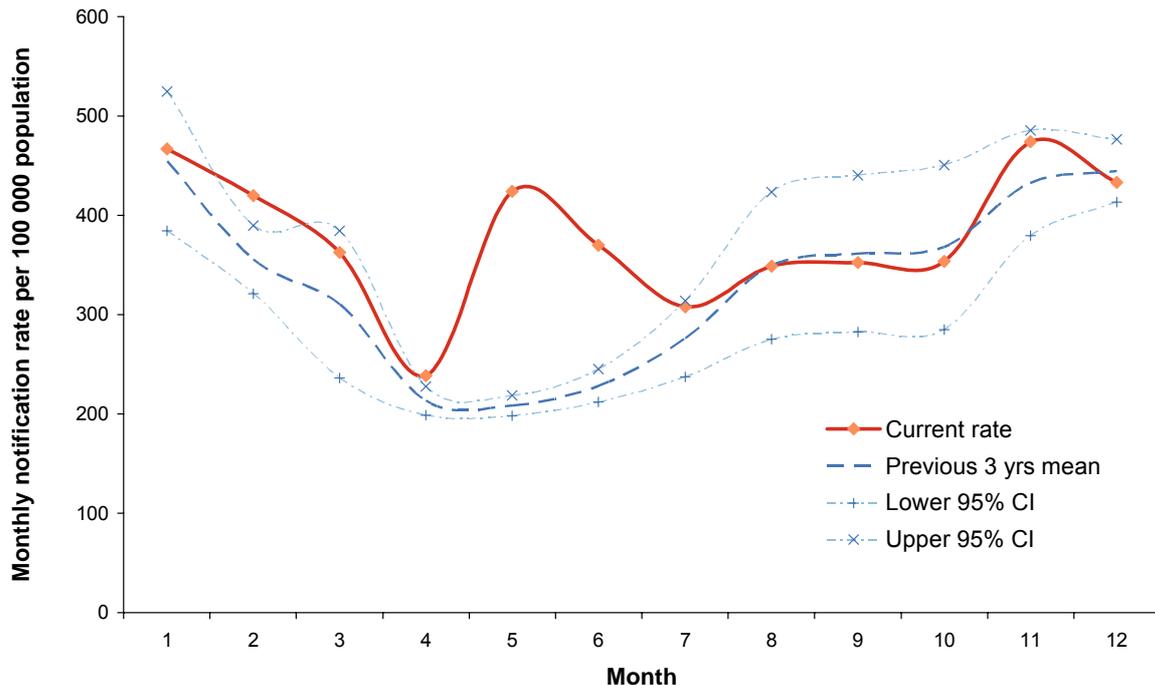
The peak in campylobacteriosis cases at month 5 (May) is inconsistent with historical trends and was the subject of a specific investigation. The results of the investigation were not published during the 2006 year.

4.4.3.3 Geographic distribution of campylobacteriosis notifications

Campylobacteriosis notification rates varied throughout the country in 2006 as shown in Figure 6. The highest rates were recorded in South Canterbury (517.4 per 100 000 population), Capital and Coast (510.4 per 100 000) and Waitemata (459.6 per 100 000) DHBs. South Canterbury has had the highest rates in the three years from 2004 to 2006. Capital and Coast had the second highest rates in 2005 and 2006.

Figure 6: **Geographic distribution of campylobacteriosis notifications, 2003-2006**
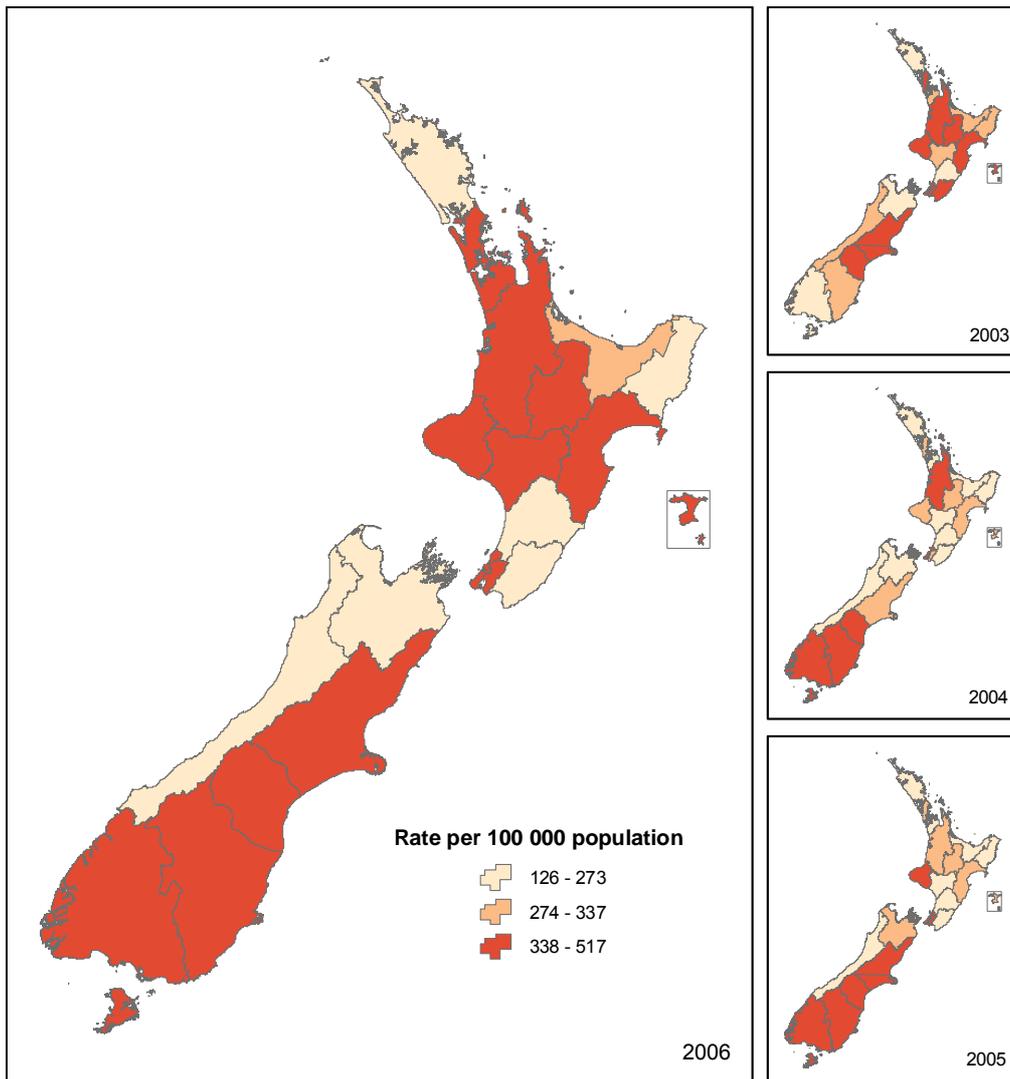
4.4.3.4 Age and sex distribution of campylobacteriosis cases

The number and rate of notifications for campylobacteriosis were higher in males than in females but the hospitalisation rates were similar for both sexes (Table 7).

Table 7: **Campylobacteriosis cases by sex, 2006**

Sex	EpiSurv notifications		Hospitalisations[a]		Deaths recorded in EpiSurv
	No.	Rate	No.	Rate	No.
Male	8 238	402.2	580	28.3	1
Female	7 269	340.3	599	28.0	
Unknown	366				
Total	**15 873**	**379.3**	**1 179**	**28.2**	**1**

[a] NZHIS morbidity data for hospital admissions

The highest age specific campylobacteriosis population rates were reported in children aged 1 to 4 years old (1 227 cases, 540.7 per 100 000), followed by the 20 to 29 years (2 884 cases, 522.9 per 100 000) and the 60 to 69 years age group (1 396 cases, 408.0 per 100 000). The hospitalisation rate for the 70+ years age group were more than double that reported in any other age group (Table 8).

Table 8: Campylobacteriosis cases by age group, 2006

Age groups	EpiSurv notifications		Hospitalisations[a]		Deaths recorded in EpiSurv
	No.	Rate	No.	Rate	No.
<1	237	401.3	20	33.9	
1 to 4	1 227	540.7	41	18.1	
5 to 9	680	233.0	23	7.9	
10 to 14	713	229.7	44	14.2	
15 to 19	1 251	399.0	95	30.3	
20 to 29	2 884	522.9	197	35.7	
30 to 39	2 218	370.5	124	20.7	
40 to 49	2 034	324.1	124	19.8	
50 to 59	1 888	372.6	122	24.1	
60 to 69	1 396	408.0	125	36.5	
70+	1 213	340.6	264	74.1	1
Unknown	132				
Total	**15 873**	**379.3**	**1,179**	**28.2**	**1**

[a] NZHIS morbidity data for hospital admissions

4.4.3.5 Risk factors reported

The risk factor most commonly reported for campylobacteriosis notifications during 2006 was consumption of food from retail premises (52.4%), followed by contact with farm animals (28.5%), and consumption of untreated water (16.4%) (Table 9).

Table 9: Exposure to risk factors associated with campylobacteriosis, 2006

Risk Factor	Notifications			
	Yes	No	Unknown	%[a]
Consumed food from retail premises	2 108	1 918	11 847	52.4%
Contact with farm animals	1 290	3 241	11 342	28.5%
Consumed untreated water	681	3 471	11 721	16.4%
Contact with faecal matter	507	3 873	11 493	11.6%
Recreational water contact	475	3 972	11 426	10.7%
Contact with other symptomatic people	443	3 927	11 503	10.1%
Travelled overseas during the incubation period	305	4 758	10 810	6.0%
Contact with sick animals	177	3 911	11 785	4.3%
Contact with a confirmed case of same disease	184	5 229	10 460	3.4%

[a]Percentage refers to the cases that answered "yes" out of the total number of cases for which this information was supplied. Cases may have more than one risk factor recorded.

Over the five years 2002 to 2006, the consumption of food from retail premises, contact with farm animals, and consumption of untreated water were consistently the most commonly reported risk

factors for campylobacteriosis ad their relative importance has remained reasonably consistent (Figure 7).

Figure 7:   Campylobacteriosis risk factors by percentage of cases and year, 2002 – 2006
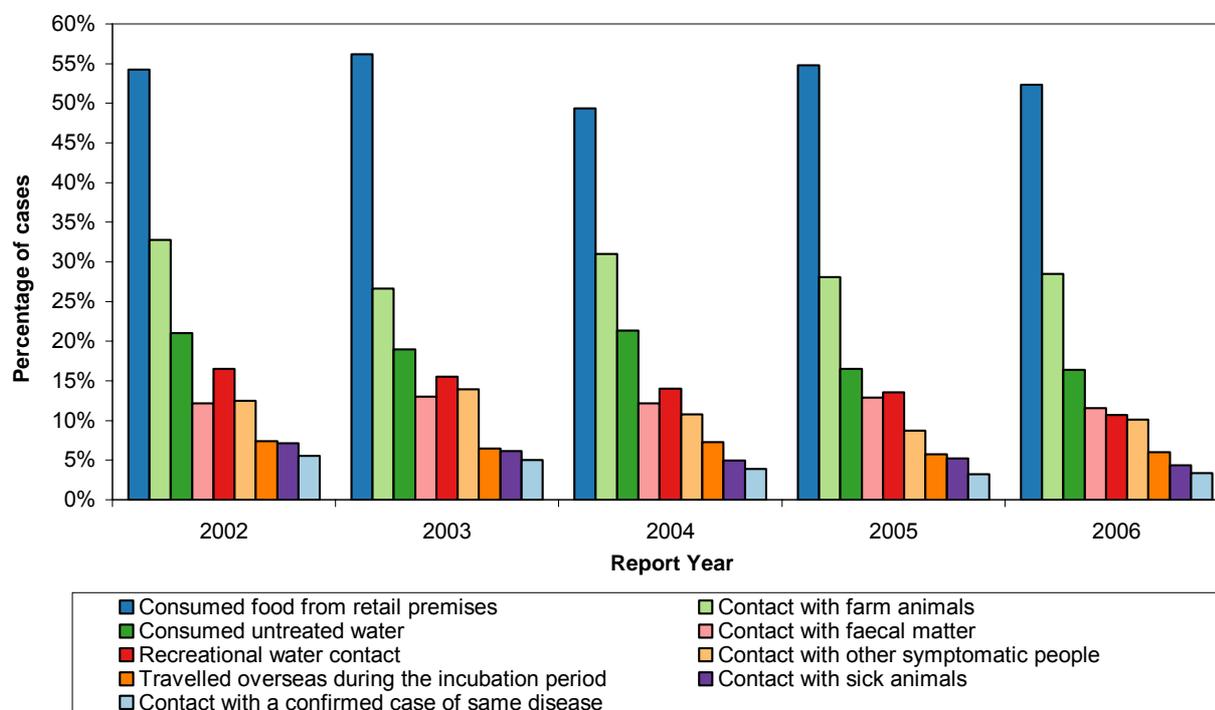
4.4.3.6   Estimate of travel-related cases

For cases where information on travel was provided, 6.0% (95%CI 5.4-6.7%) had travelled overseas during the incubation period. Assuming that the cases for which travel information was provided were representative of all campylobacteriosis cases, a Poisson distribution can be used to estimate the total number of potentially travel related cases of campylobacteriosis in 2006. The resultant distribution has a mean of 956 cases (95% CI 836-1084).

If data from the last four years are considered the estimated proportion of cases travelling overseas within the incubation period of the organism is 6.4% (95% CI 6.0-6.7%).

4.4.4   Outbreaks reported as caused by *Campylobacter* spp.

In this section only *Campylobacter* spp. outbreaks with a suspected or known foodborne source are included unless otherwise stated.

In 2006, 32 (68%) of the *Campylobacter* outbreaks and 135 (61%) of associated cases were reported as food borne related (Table 10). All of the *Campylobacter* cases reported as hospitalised were associated with food borne outbreaks. *Campylobacter* outbreaks accounted for 9.5% (47/495) of all outbreaks and 3.5% (223/6 302) of all associated cases. Only norovirus was identified as the causal agent in more outbreaks (156), with more associated cases (3 945). However, only 14.7% (23/156) of norovirus outbreaks had suspected foodborne transmission.

Table 10: *Campylobacter* spp. outbreaks reported, 2006

Measure (No.)	Foodborne *Campylobacter* spp. outbreaks	All *Campylobacter* spp. outbreaks
Outbreaks	32	47
Cases	135	221
Hospitalised cases	7	7

There has been an increase in the cases associated with foodborne campylobacteriosis outbreaks from 81 cases in 2004 to 135 in 2006 (Figure 8). Over the seven year period from 2000 to 2006 the highest number of outbreaks and cases were reported in 2002 (35 outbreaks, 196 cases) with the second highest reported for 2006 (32 outbreaks, 135 cases).

Figure 8: **Foodborne *Campylobacter* spp. outbreaks and associated cases reported by year, 2000 – 2006**
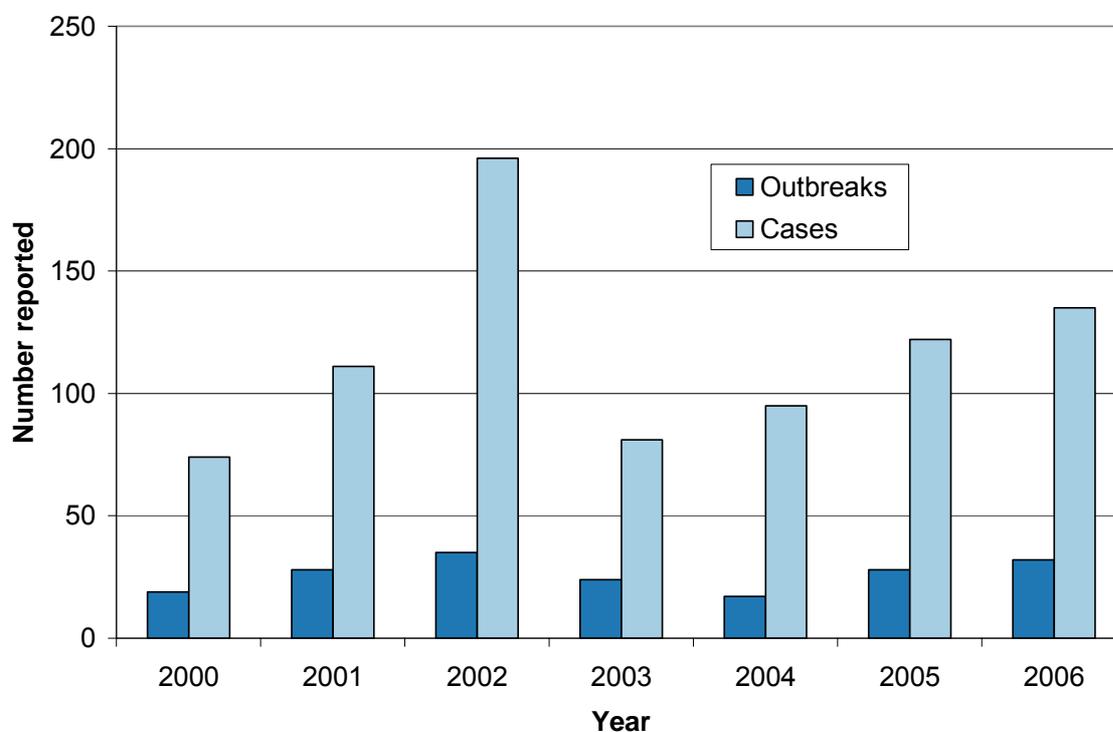
4.4.4.1 *Details of food-associated outbreaks*

Table 11 contains details of the 32 food–associated *Campylobacter* spp. outbreaks reported in 2006.

Table 11: Details of food-associated *Campylobacter* spp. outbreaks, 2006

Public Health Unit (Month)	Suspected vehicle	Setting	Number ill	Confirmation
Auckland (January)	Beef burgers	Restaurant/cafe	1C, 2P	2
Auckland (January)	Chicken liver pate and chicken coq au vin	Restaurant/cafe	2C	1, 2
Auckland (January)	Unknown	Home	2C, 2P	2
Auckland (February)	Home prepared chicken livers or raw eggs	Home	2C	2
Auckland (February)	Takeaway	Home	2C, 1P	None
Auckland (February)	Meals	Hostel/boarding house	4C	2
Auckland (February)	Teriyaki chicken	Restaurant/cafe	2C	2
Auckland (March)	Chicken livers	Restaurant/cafe	2C, 1P	2
Auckland (March)	Chicken livers	Restaurant/cafe	1C, 1P	2
Auckland (May)	Chicken liver pate	Rest home	3C	1, 2
Auckland (May)	Chicken liver pate	Restaurant/cafe	3C	1, 2
Auckland (May)	Chicken livers	Restaurant/cafe	1C, 1P	1, 2, 7
Auckland (June)	Takeaways	Restaurant/café, Home	2C, 1P	1, 2
Auckland (July)	Chicken drumsticks	Takeaway	2C	1, 2
Auckland (July)	Pies	Supermarket	2C	1, 2
Auckland (July)	Chicken livers	Restaurant/cafe	1C, 1P	1, 2
Auckland (August)	Lemon creme brulee	Restaurant/cafe	4C, 2P	2, 3, 4
Auckland (September)	Raw egg	Home	2C	2
Auckland (October)	Unknown	Unknown	2C	2
Auckland (November)	Chicken liver	Restaurant/cafe	2C	1, 2
Auckland (November)	Takeaway	Home	1C, 2P	2
Auckland (November)	Unknown	Unknown	2C	2
Auckland (November)	Unknown	Unknown	2C	2
Auckland (November)	Chicken	Home	1C, 2P	2
Auckland (November)	Unknown	Unknown	1C, 2P	2
Auckland (December)	Chicken roll with stuffing	Caterers	12C, 11P	1, 2
Manawatu (November)	Chicken and leek pie.	Caterers, Home	3C, 5P	1, 2
Nelson (January)	BBQ food	Home	3C	None
Otago (September)	Eggs	Restaurant/cafe	4C, 4P	1, 2
Wellington (April)	Beef (Beef pasta and steak sandwich)	Restaurant/cafe	3C	2
Wellington (June)	Chicken dishes	Restaurant/cafe	3C	6
Wellington (July)	Chicken	Restaurant/cafe	20C	2

C = confirmed, P = probable
Confirmation:
1 = Environmental investigation – identified critical control point failures linked to implicated source
2 = Epidemiological – case had history of exposure to implicated source
3 = Epidemiological – case control or cohort study showed elevated risk for cases to implicated source
4 = Laboratory – pathogen suspected to have caused illness identified in food handler
5 = Laboratory – pathogen suspected to have caused illness identified in implicated source (food)
6 = No evidence
7 = Other evidence

While a high proportion of outbreaks (9/32, 28%) identified chicken liver or chicken liver products as the suspected source of infection, the level of confirmation was not high and most commonly was that the case had had exposure to the food.

4.4.4.2 Laboratory investigation of samples from suspected foodborne outbreaks

During investigations of suspected foodborne illness outbreaks by ESR's Public Health Laboratories, *Campylobacter* was isolated from stool samples in 13 incidents. Implicated foods included ethnic takeaway food, filled rolls, sausage rolls, chicken, chicken salad, chicken roll, mussels, and fish and chips. However, *Campylobacter* was not isolated from any food samples analysed in association with outbreaks during 2006.

4.4.5   Disease sequelae - Guillain-Barré Syndrome (GBS)

Guillain-Barré Syndrome is most commonly preceded by an infection with *Campylobacter jejuni*. Other respiratory or intestinal illnesses and other triggers may also precede an episode of GBS.

The ICD-10 code G61 was used to extract GBS hospitalisation data from the NZHIS NMDS database. Of the 126 hospitalised cases (3.0 cases per 100 000 population) recorded in 2006, 87 were reported with GBS as the primary diagnosis and 39 with this condition as another relevant diagnosis.

Over the period 2002 to 2006 the number of hospitalised cases (any diagnosis code) for GBS have varied in the range 119 to 150 (Figure 9).

Figure 9:   GBS hospitalised cases, 2002 - 2006
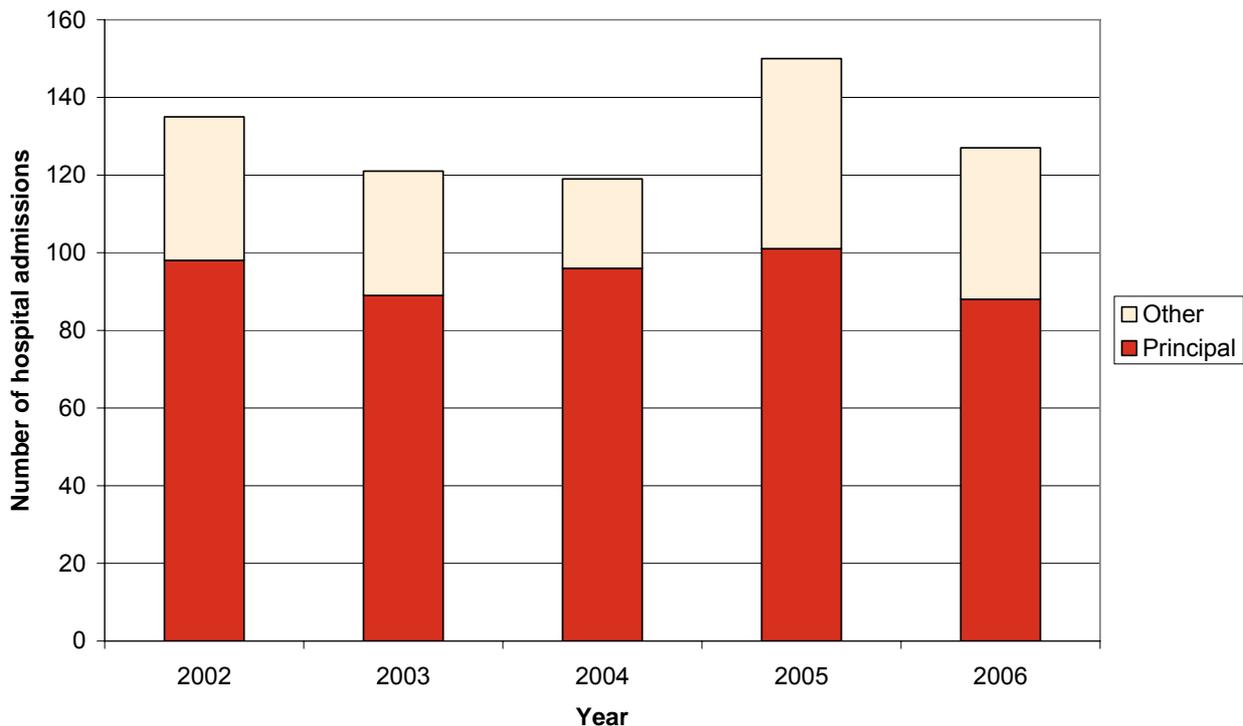
In 2006 the number of GBS hospitalised cases were greater for males than females (Table 12).

Table 12: GBS hospitalised cases by sex, 2006

Sex	Hospitalisations	
	No.	Rate per 100 000
Male	78	3.8
Female	48	2.3
Total	126	3.0

In 2006 the highest hospitalised case rate for GBS occurred in 60-69 year olds (Table 13).

Table 13: GBS hospitalised cases by age group, 2006

Age groups	Hospitalisations	
	No.	Rate per 100 000
<1	0	0.0
1 to 4	6	2.6
5 to 9	2	0.7
10 to 14	6	1.8
15 to 19	8	2.5
20 to 29	10	1.9
30 to 39	11	1.8
40 to 49	14	2.3
50 to 59	24	4.8
60 to 69	24	7.2
70+	21	6.0
Total	**126**	**3.0**

4.4.6   Relevant New Zealand studies and publications

Notification and hospitalisation data on campylobacteriosis in New Zealand were analysed to investigate whether the increase over the last two decades was real, or a surveillance artefact (Baker *et al.*, 2006b). Based largely on comparisons of trends in the two sources of data it was concluded that the increase was real. The highest rates occurred in children aged 1-4 years, Europeans, and those living in urban areas.

A discussion regarding sources of campylobacteriosis was conducted in the New Zealand Medical Journal during 2006. The opening article suggested that although chicken was a significant risk factor, it was not the major source of infection (Nelson and Harris, 2006a). Instead flies were proposed as an important vector, with deposition of *Campylobacter* by flies on fingers and fomites resulting in bacterial ingestion, possibly via food.

The NZFSA responded to this article (Campbell *et al.*, 2006) pointing out that the higher rates of illness in urban dwellers compared to rural population do not support the hypothesis. Nevertheless, the need to implement effective programmes to reduce the known risks of direct foodborne transmission was acknowledged. The NZFSA launched their *Campylobacter* in poultry risk management strategy in November 2006 (see below).

Another response (Wilson *et al.*, 2006) also disputed the flies hypothesis, and stressed the correlation between the rising rates of chicken consumption and campylobacteriosis.

The original authors responded (Nelson and Harris, 2006b) further disputing the primacy of chicken consumption, and offering further hypotheses concerning the importance of dairy cow numbers and foreign tourist visitors.

A final article in 2006 (Baker *et al.*, 2006a) reinforced the importance of chicken consumption as a risk factor and advocated freezing of the entire poultry supply as a short term control measure.

4.4.7   Relevant regulatory developments

During September 2006 NZFSA launch their *Campylobacter in Poultry Risk Management Strategy 2006-2009:*
http://www.nzfsa.govt.nz/publications/media-releases/campylobacter-strategy-nov-2006.htm

The strategy has objectives:
- To reduce the incidence of foodborne human campylobacteriosis;
- To better quantify the proportion of foodborne cases attributable to poultry;
- To understand the relative value of different interventions throughout the food chain in reducing risks to human health;
- To make well-informed risk management decisions on appropriate control measures and their implementation; and
- To design and implement an ongoing monitoring and review programme to assess the effectiveness of risk management decisions

4.5   Ciguatera Fish Poisoning (CFP)

4.5.1   Case definition

Clinical description: Gastroenteritis, possibly followed by neurologic symptoms
Laboratory test for diagnosis: Demonstration of ciguatoxin in implicated fish
Case classification: Not applicable

4.5.2   Ciguatera fish poisoning cases reported in 2006 by data source

No ciguatera fish poisoning cases were reported in EpiSurv in 2006.

The ICD-10 code T61.0 was used to extract ciguatera fish poisoning hospitalisation data from the NZHIS NMDS database. Of the 5 hospital admissions (0.1 admissions per 100 000 population) recorded in 2006, all were reported with ciguatera fish poisoning as the primary diagnosis.

4.5.3 Outbreaks reported as caused by ciguatera fish poisoning

No cases or outbreaks due to ciguatera fish poisoning were reported in 2006 (Figure 10). Very few outbreaks of ciguatera fish poisoning have been reported in recent years. In the three years 2004 to 2006 one outbreak due to ciguatera fish poisoning was reported.

Figure 10: **Outbreaks and associated cases due to ciguatera fish poisoning reported by year, 2000 – 2006**
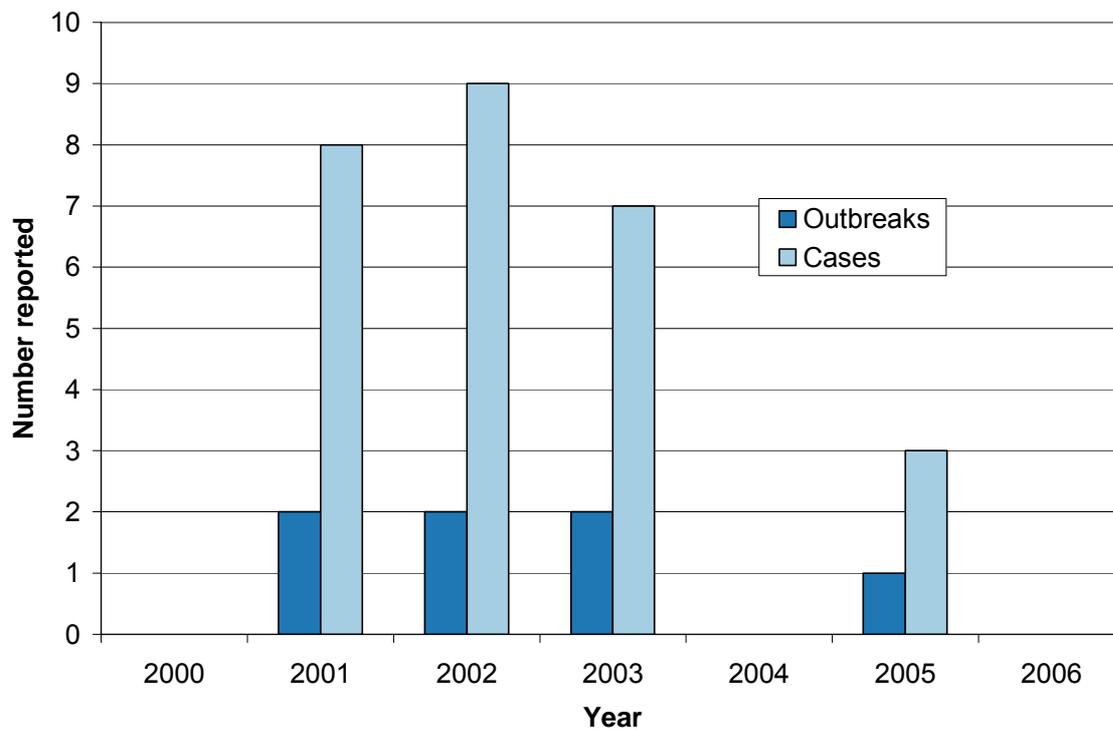
4.5.3.1 Laboratory investigation of samples from suspected foodborne outbreaks

Nil.

4.5.4 Relevant New Zealand studies and publications

Nil.

4.5.5 Relevant regulatory developments

Nil.

4.6   *Clostridium perfringens* Intoxication

4.6.1   Case definition

Clinical description: Gastroenteritis with profuse watery diarrhoea

Laboratory test for diagnosis: Detection of enterotoxin in faecal specimen or faecal spore count of $\geq 10^6$/g or isolation of $\geq 10^5$/g *C. perfringens* in leftover food

Case classification:

Probable   A clinically compatible illness that is either a contact of a confirmed case of the same disease, or has had contact with the same common source i.e., is part of an identified common source outbreak

Confirmed   A clinically compatible illness that is laboratory confirmed

4.6.2   *Clostridium perfringens* intoxication cases reported in 2006 by data source

Six cases of *Clostridium perfringens* intoxication were reported in EpiSurv during 2006 with no resulting deaths recorded.

4.6.3   Outbreaks reported as caused by *Clostridium perfringens*

All *Clostridium perfringens* outbreaks for 2006 were associated with a suspected or known foodborne source (Table 14).

Table 14:   *Clostridium perfringens* outbreaks reported, 2006

Measure (No.)	Foodborne *Clostridium perfringens* outbreaks	All *Clostridium perfringens* outbreaks
Outbreaks	12	12
Cases	62	62
Hospitalised cases	0	0

There has been a steady decrease in the number of foodborne outbreaks associated with *Clostridium perfringens* between 2000 and 2004 with a small increase in the number of outbreaks reported in 2005 and 2006 (Figure 11). The number of cases associated with *Clostridium perfringens* outbreaks has varied over time with a trend towards a fewer number of cases associated with the outbreaks in the last three years (2004 to 2006) compared to the previous four years (2000 to 2003).

Figure 11: Foodborne *Clostridium perfringens* outbreaks and associated cases reported by year, 2000–2006
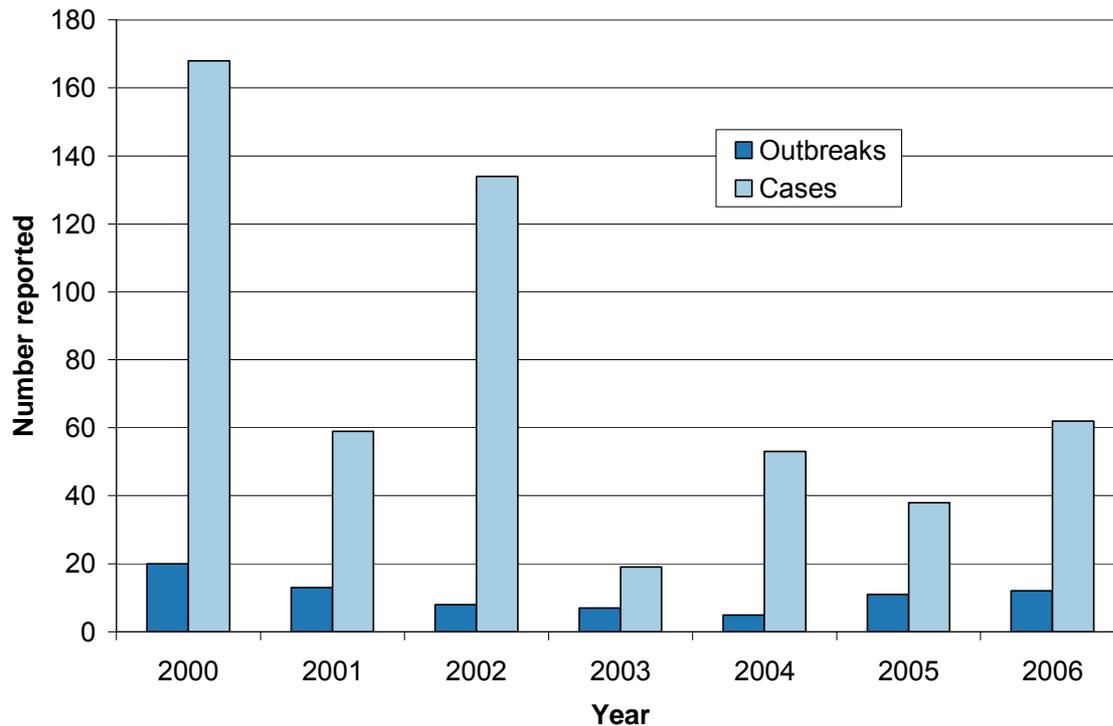
4.6.3.1 Details of food-associated outbreaks

Table 15 contains details of the 12 food–associated *Clostridium perfringens* intoxication outbreaks reported in 2006.

Table 15: Details of food-associated *Clostridium perfringens* outbreaks, 2006

Public Health Unit (Month)	Suspected vehicle	Setting	Number ill	Confirmation
Auckland (April)	Oysters	Seafood market	1C, 1P	2
Auckland (April)	Quiche	Restaurant/café	3P	2
Auckland (May)	Meal	Home, supermarket	1C, 1P	1, 2
Auckland (May)	Roast meals	Takeaway	3C, 7P	1, 2, 5
Auckland (July)	BBQ meat	Restaurant/café, home	2P	2
Auckland (September)	Chinese meal	Takeaway	1C, 2P	1, 2
Auckland (September)	Roast pork	Restaurant/café	2C, 4P	1, 2
Auckland (October)	Unknown	Restaurant/café, home	2P	2
Auckland (November)	Roast Pork	Caterers	2C, 13P	1, 3
Auckland (November)	Unknown	Restaurant/café	1C, 5P	2
Otago (February)	Roast pork	Restaurant/café	2C, 7P	1, 2
Rotorua (November)	Potato Topped Family Mince Pie	Other food retail	2C	1, 2, 5

C = confirmed, P = probable
Confirmation:
1 = Environmental investigation – identified critical control point failures linked to implicated source
2 = Epidemiological – case had history of exposure to implicated source
3 = Epidemiological – case control or cohort study showed elevated risk for cases to implicated source

4 = Laboratory – pathogen suspected to have caused illness identified in food handler
5 = Laboratory – pathogen suspected to have caused illness identified in implicated source (food)
6 = No evidence
7 = Other evidence

Of the 12 food-associated *Clostridium perfringens* intoxication outbreaks, four were associated with roast foods, particularly roast pork.

4.6.3.2 *Laboratory investigation of samples from suspected foodborne outbreaks*

During investigations of suspected foodborne illness outbreaks by ESR's Public Health Laboratories, *C. perfringens* was detected in clinical or food samples from 14 investigations. While the majority of these were outbreaks reported in Table 15, an additional investigation implicated chicken.

A large investigation was carried out at the end of 2006 of a suspected foodborne outbreak involving more than 50 cases in the Tairawhiti DHB region. *C. perfringens* enterotoxin was detected in two faecal samples, with one faecal sample also containing high levels of *C. perfringens.* Moderate levels of *C. perfringens* were detected in turkey meat from a Xmas meal consumed by the cases.

4.6.4 Relevant New Zealand studies and publications

Nil.

4.6.5 Relevant regulatory developments

Nil.

4.7 Cryptosporidiosis

Summary data for cryptosporidiosis in 2006 are given in Table 16.

Table 16:   Summary surveillance data for cryptosporidiosis, 2006

Parameter	Value in 2006	Section reference
Number of cases	737	4.7.2
Rate (per 100 000)	17.6	4.7.2
Hospitalisations (%)	30 (4.1%)	4.7.2
Deaths (%)	Nil	4.7.2
Estimated travel-related cases (%)	61 (8.1%)	4.7.3.6
Estimated food-related cases (%)	NA	

NA = not applicable, no information is available on the food attributable proportion of cryptosporidiosis in New Zealand

4.7.1 Case definition

Clinical description: An illness with diarrhoea and abdominal pain. The infection may be asymptomatic

Laboratory test for diagnosis: Detection of *Cryptosporidium parvum* oocysts in a faecal specimen

Case classification:

Probable   A clinically compatible illness that is either a contact of a confirmed case of the same disease, or has had contact with the same common source i.e., is part of an identified common source outbreak

Confirmed   A clinically compatible illness that is laboratory confirmed

4.7.2   Cryptosporidiosis cases reported in 2006 by data source

During 2006, 737 notifications (17.6 cases per 100 000 population) of cryptosporidiosis were reported in EpiSurv.

The ICD-10 code A07.2 was used to extract cryptosporidiosis hospitalisation data from the NZHIS NMDS database. Of the 30 hospital admissions (0.7 admissions per 100 000 population) recorded in 2006, 20 were reported with cryptosporidiosis as the primary diagnosis and 10 with cryptosporidiosis as another relevant diagnosis.

No deaths were recorded in EpiSurv in 2006.

4.7.3   Notifiable disease data

4.7.3.1   Annual notification trend

Cryptosporidiosis became a notifiable disease in June 1996. The highest number of cases was reported in 2001 (1,208 cases). Since 2001 there has been a general decrease in the number of notifications, although there were a higher number of notifications in 2005 and 2006 than in 2004 (Figure 12).

Figure 12:   Cryptosporidiosis notifications by year, 1996-2006
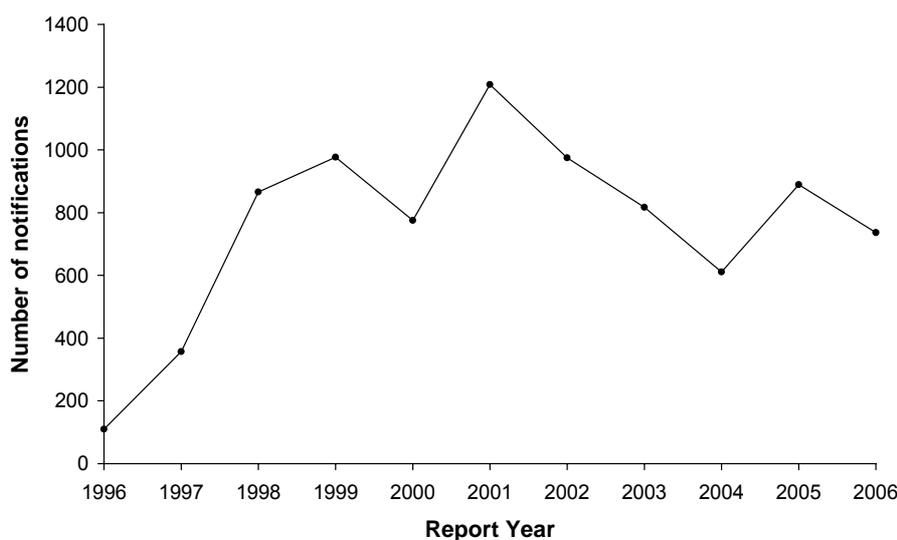
The cryptosporidiosis annual population rate trend is very similar to the corresponding annual notification trend. The highest cryptosporidiosis annual notification rate was reported in 2001 and

has generally decreased since, although both 2005 and 2006 reported slightly higher rates than observed in 2004 (Figure 13).

Figure 13: Cryptosporidiosis notification rate by year, 2000-2006
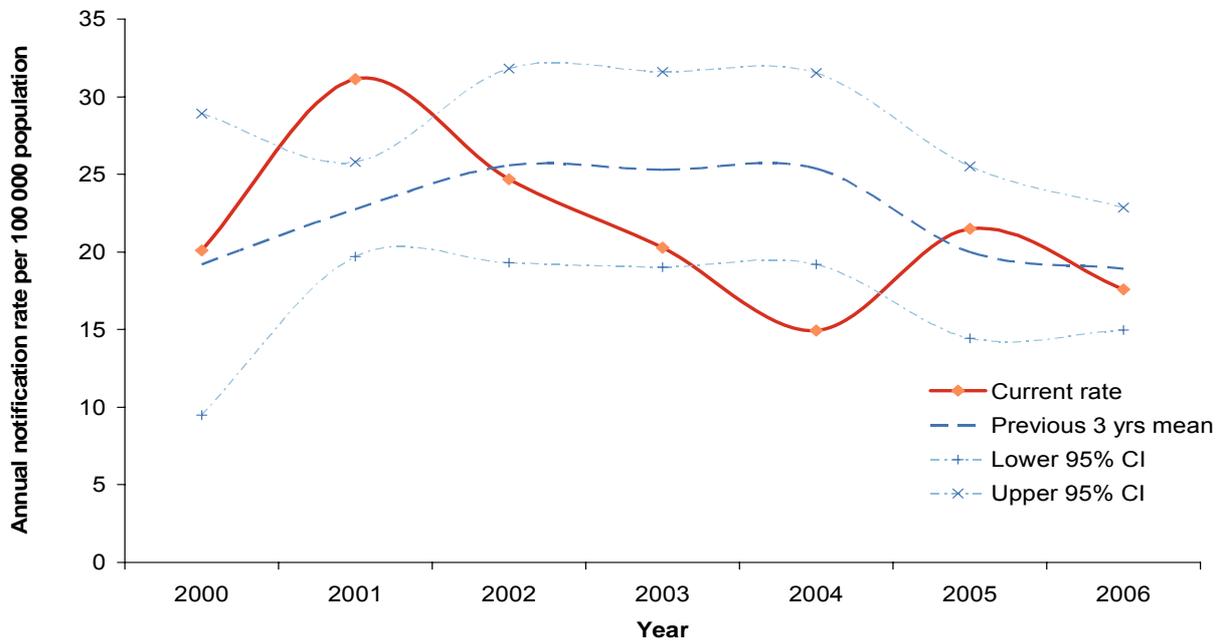
4.7.3.2 Seasonality

The number of notified cases of cryptosporidiosis reported per 100 000 population by month for 2006 was similar to previous years. Cryptosporidiosis has a consistent spring peak (September/October) (Figure 14).

Figure 14:   Cryptosporidiosis monthly rate (annualised) for 2006
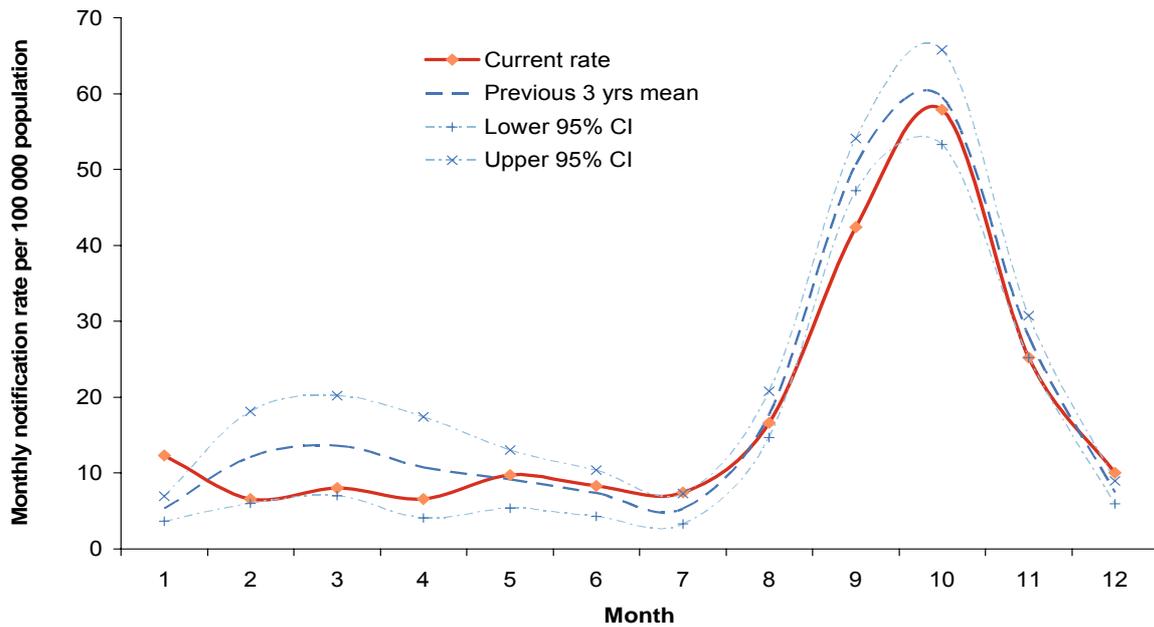
4.7.3.3   *Geographic distribution of cryptosporidiosis notifications*

There were consistently higher population rates of reporting of cryptosporidiosis notifications in the predominantly rural DHBs compared to the more urban DHBs (Figure 15). In 2006, the highest rates were reported in South Canterbury (74.4 per 100 000 population), Wairarapa (63.2 per 100 000) and West Coast (49.8 per 100 000) DHBs. South Canterbury has reported the highest cryptosporidiosis rates for the past four years.

Figure 15:   Geographic distribution of cryptosporidiosis notifications, 2003-2006

Rate per 100 000 population

3 - 15
16 - 34
35 - 90

2006

2003

2004

2005
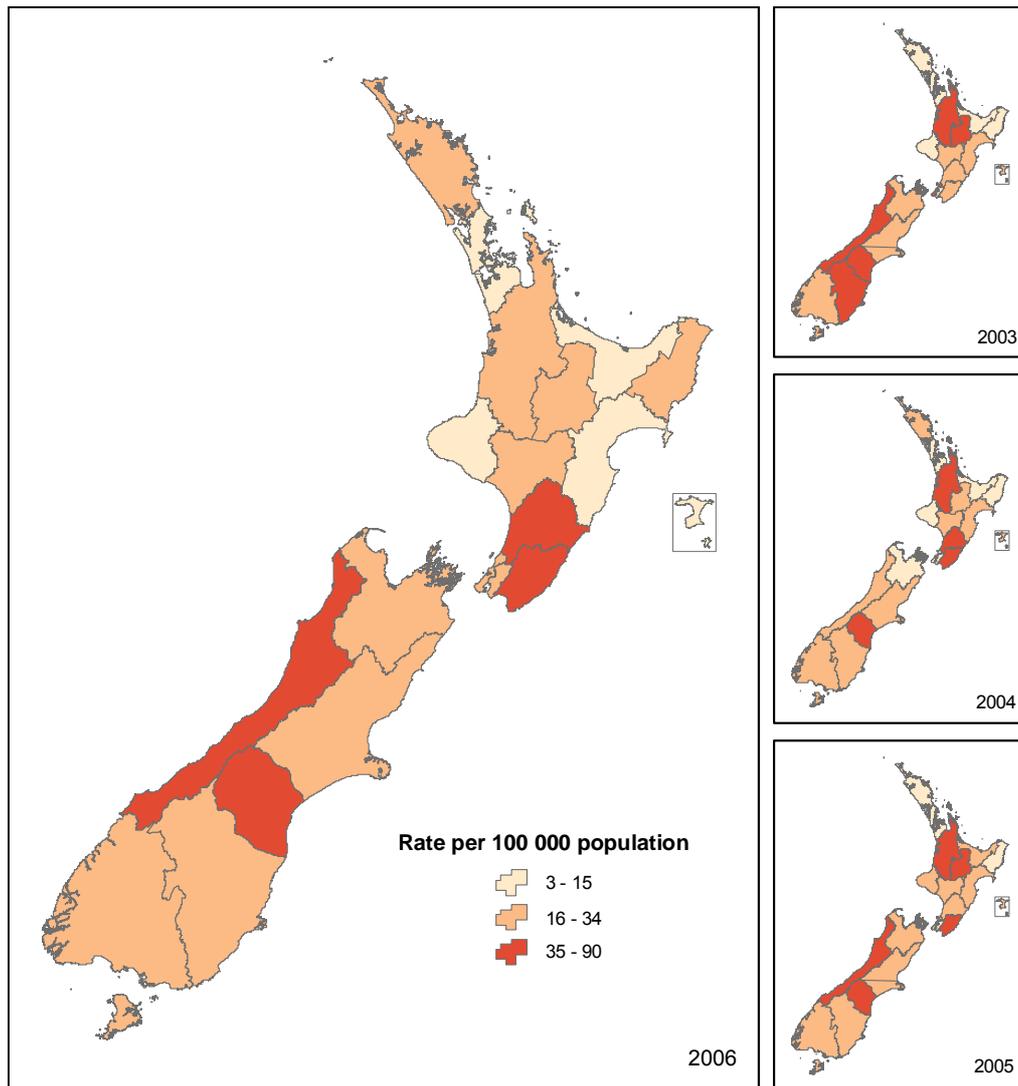
4.7.3.4   Age and sex distribution of cryptosporidiosis cases

The number and notification rates for cryptosporidiosis were similar for males and females but more males were hospitalised than females (Table 17).

Table 17:   Cryptosporidiosis cases by sex, 2006

Sex	EpiSurv notifications		Hospitalisations[a]		Deaths recorded in EpiSurv
	No.	Rate	No.	Rate	No
Male	364	17.8	18	0.9	
Female	362	16.9	12	0.6	
Unknown	11				
Total	**737**	**17.6**	**30**	**0.7**	**-**

[a] NZHIS morbidity data for hospital admissions

In 2006 the highest cryptosporidiosis age specific notification rates were in the 1 to 4 years age group (252 cases, 111.0 per 100 000 population), followed by the less than one years (19 cases, 32.2 per 100 000) and the 5 to 9 years (88 cases, 30.1 per 100 000) (Table 18).

Table 18:   Cryptosporidiosis cases by age group, 2006

Age groups	EpiSurv notifications		Hospitalisations[a]		Deaths recorded in EpiSurv
	No.	Rate	No.	Rate	No.
<1	19	32.2	2	3.4	
1 to 4	252	111.0	3	1.3	
5 to 9	88	30.1	6	2.1	
10 to 14	59	19.0	3	1.0	
15 to 19	39	12.4	1	0.3	
20 to 29	88	16.0	4	0.7	
30 to 39	90	15.0	4	0.7	
40 to 49	54	8.6	3	0.5	
50 to 59	32	6.3	1	0.2	
60 to 60	9	2.6	0	0.0	
70+	6	1.7	3	0.8	
Unknown	1				
Total	**737**	**17.6**	**30**	**0.7**	**-**

[a] NZHIS morbidity data for hospital admissions

4.7.3.5   Risk Factors Reported

In 2006 the most commonly reported risk factor for cryptosporidiosis notification cases was contact with farm animals (52.5%), followed by consumption of untreated water (35.6%), contact with faecal matter (30.3%), and recreational water contact (29.7%) (Table 19).

Table 19:   Exposure to risk factors associated with cryptosporidiosis, 2006

	Notifications			
Risk Factor	**Yes**	**No**	**Unknown**	**%[a]**
Contact with farm animals	286	259	192	52.5%
Consumed untreated water	160	290	287	35.6%
Contact with faecal matter	153	352	232	30.3%
Recreational water contact	146	345	246	29.7%
Consumed food from retail premises	80	227	430	26.1%
Contact with other symptomatic people	121	373	243	24.5%
Contact with sick animals	107	343	287	23.8%
Travelled overseas during the incubation period	43	490	204	8.1%
Contact with a confirmed case of same disease	29	424	284	6.4%

[a]Percentage refers to the cases that answered "yes" out of the total number of cases for which this information was supplied. Cases may have more than one risk factor recorded.

Over the five year period 2002 to 2006 the most consistently reported risk factors for cryptosporidiosis were contact with farm animals and consumption of untreated water (Figure 16).

Figure 16: Cryptosporidiosis risk factors by percentage of cases and year, 2002 – 2006
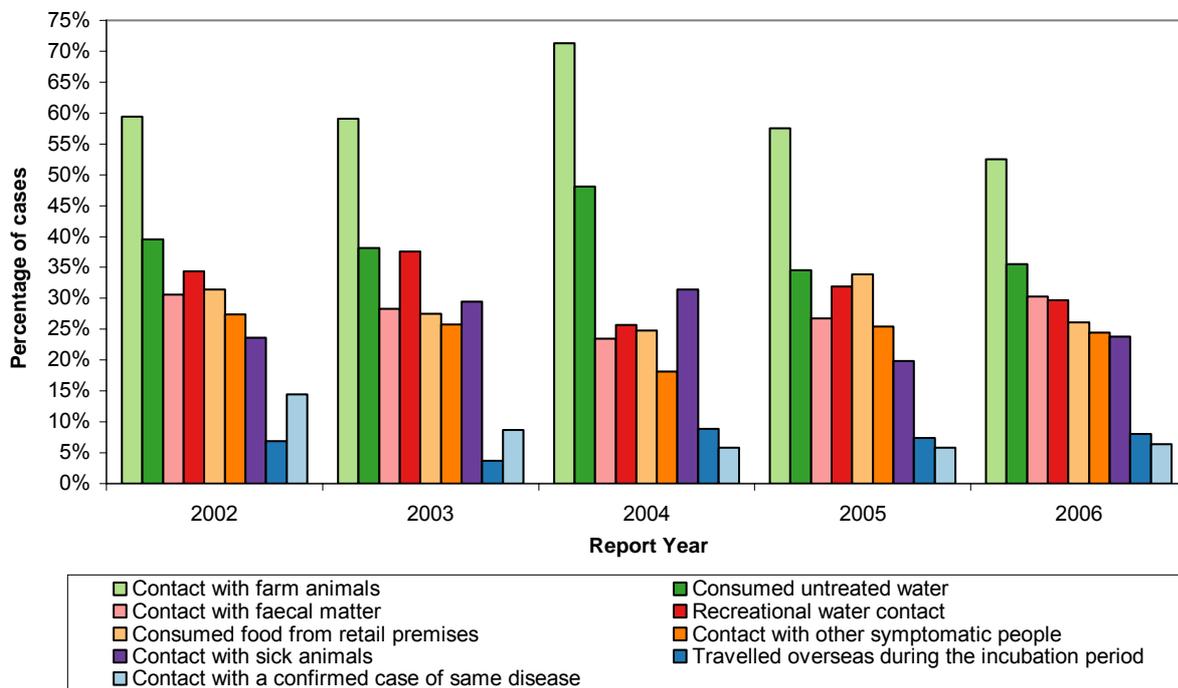
4.7.3.6 Estimate of travel-related cases

For cases where information on travel was provided, 8.1% (95%CI 6.0-10.9%) had travelled overseas during the incubation period. Assuming that the cases for which travel information was provided were representative of all cryptosporidiosis cases, a Poisson distribution can be used to estimate the total number of potentially travel related cases of cryptosporidiosis in 2006. The resultant distribution has a mean of 61 cases (95% CI 39-87).

4.7.4 Outbreaks reported as caused by *Cryptosporidium* spp.

No foodborne *Cryptosporidium* outbreaks were reported in 2006 (Table 20).

Table 20: *Cryptosporidium* spp. outbreaks reported, 2006

Measure (No.)	Foodborne *Cryptosporidium* spp. outbreaks	All *Cryptosporidium* spp. outbreaks
Outbreaks	0	25
Cases	0	116
Hospitalised cases	0	0

Foodborne *Cryptosporidium* outbreaks are rare with not more than one outbreak reported each year in the seven year period, 2000 to 2007 (Figure 17). The largest outbreak with 8 associated cases was reported in 2004.

Figure 17: **Foodborne *Cryptosporidium* spp. outbreaks and associated cases reported by year, 2000 – 2006**
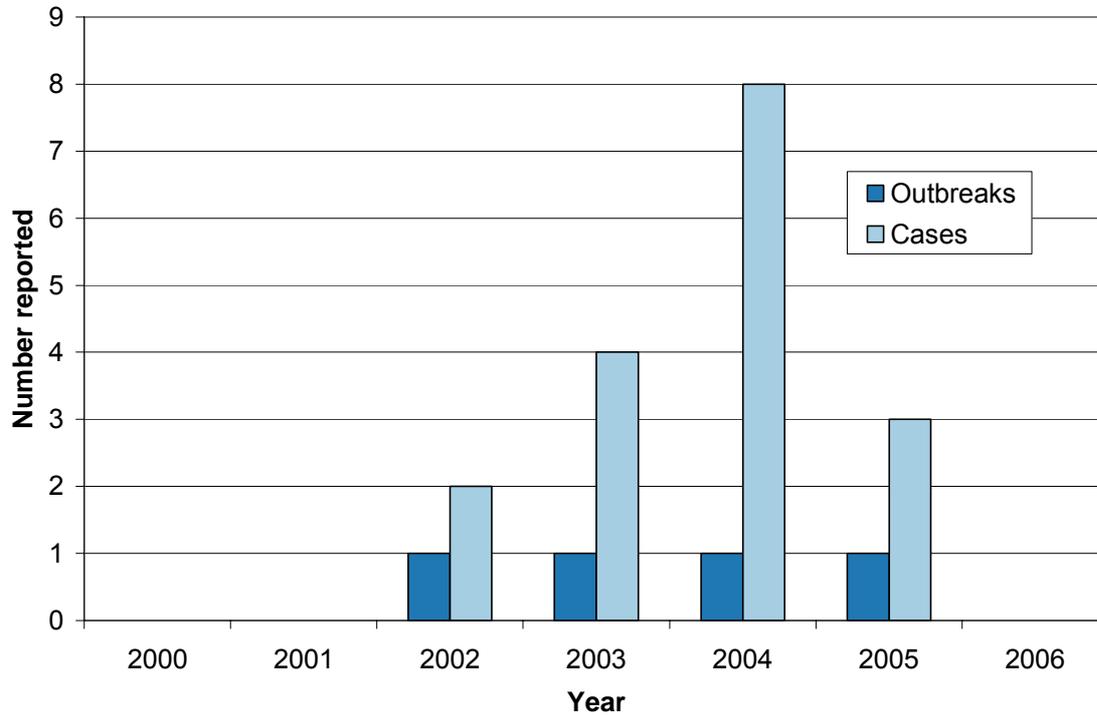
4.7.5 Relevant New Zealand studies and publications

Nil.

4.7.6 Relevant regulatory developments

Nil.

4.8 Giardiasis

Summary data for giardiasis in 2006 are given in Table 21.

Table 21: **Summary surveillance data for giardiasis, 2006**

Parameter	Value in 2006	Section reference
Number of cases	1 214	4.8.2
Rate (per 100 000)	29.0	4.8.2
Hospitalisations (%)	71 (5.8%)	4.8.2
Deaths (%)	Nil	4.8.2
Estimated travel-related cases (%)	323 (26.6%)	4.8.3.6
Estimated food-related cases (%)	NA	

NA = not applicable, no information is available on the food attributable proportion of giardiasis in New Zealand

4.8.1 Case definition

Clinical description: An illness characterised by diarrhoea, abdominal cramps, bloating, weight loss or malabsorption. The infection may be asymptomatic

Laboratory test for diagnosis: Detection of *Giardia* cysts or trophozoites in a specimen from the human intestinal tract OR detection of *Giardia* antigen in faeces

Case classification:

Probable A clinically compatible illness that is either a contact of a confirmed case of the same disease, or has had contact with the same common source i.e., is part of an identified common source outbreak

Confirmed A clinically compatible illness that is laboratory confirmed

4.8.2 Giardiasis cases reported in 2006 by data source

During 2006, 1 214 notifications (29.0 cases per 100 000 population) of giardiasis were reported in EpiSurv.

The ICD-10 code A07.1 was used to extract giardiasis hospitalisation data from the NZHIS NMDS database. Of the 71 hospital admissions (1.7 admissions per 100 000 population) recorded in 2006, 43 were reported with giardiasis as the primary diagnosis and 28 with giardiasis as another relevant diagnosis.

No deaths were recorded in EpiSurv in 2006.

4.8.3   Notifiable disease data

4.8.3.1   Annual notification trend

Giardiasis became a notifiable disease in 1996. Since 1998 there has been a steady decrease in the number of cases reported each year (Figure 18).

Figure 18:   **Giardiasis notifications by year, 1996-2006**
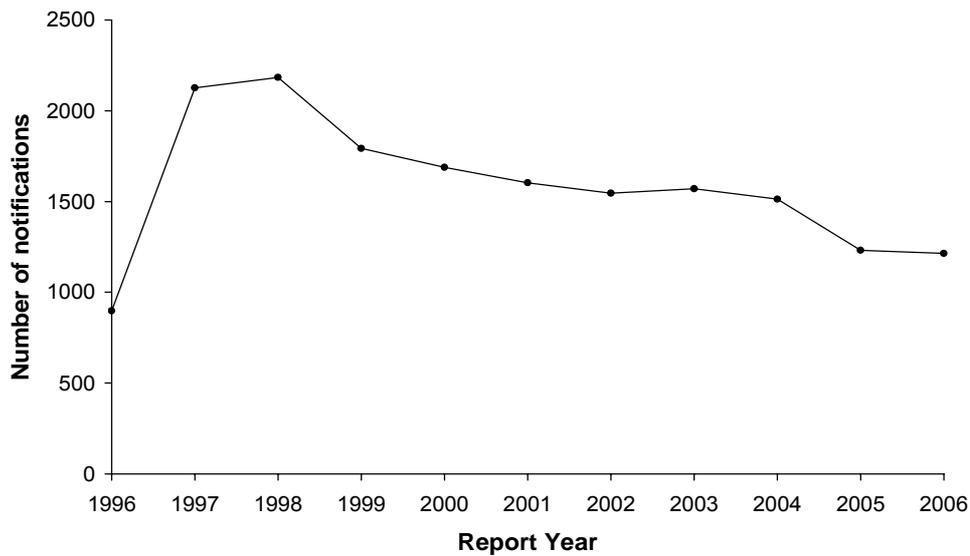
* Notification of giardiasis began midway through 1996.

Between 2000 and 2006 the giardiasis notification rate has steadily declined from 43.8 per 100 000 population in 2000 to 29.0 per 100 000 in 2006 (Figure 19).

Figure 19:   **Giardiasis notification rate by year, 2000-2006**
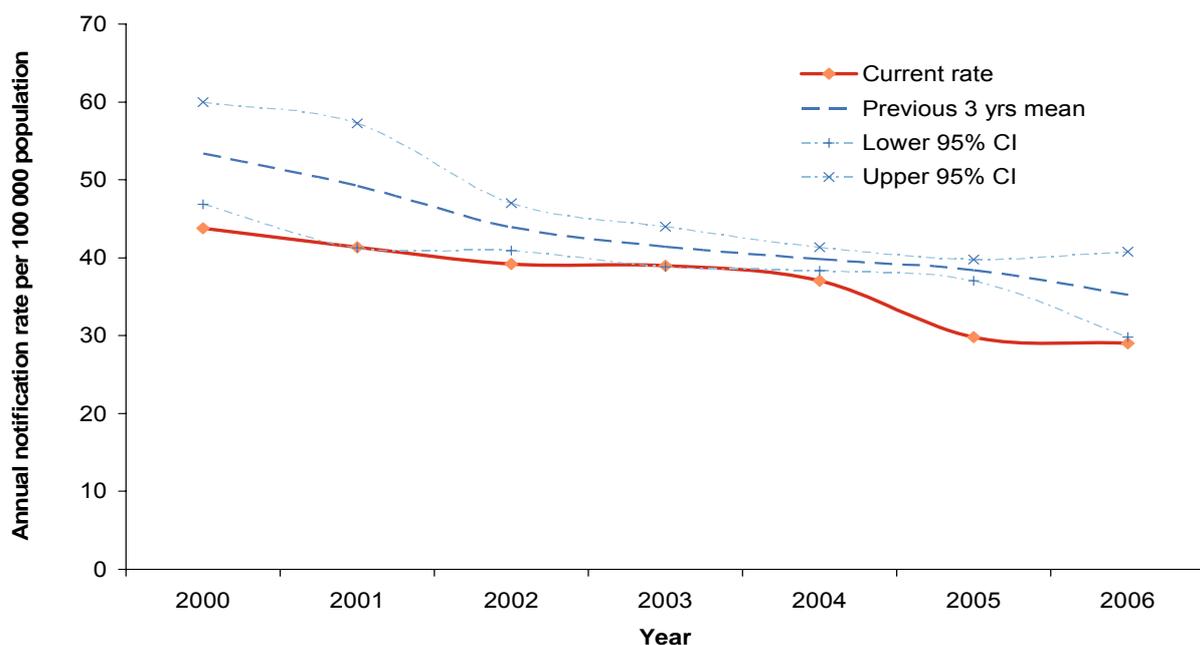
4.8.3.2 Seasonality

There was no strong seasonal pattern in the population rate of giardiasis notifications reported by month either historically or in 2006 (Figure 20).

Figure 20:   Giardiasis monthly rate (annualised) for 2006
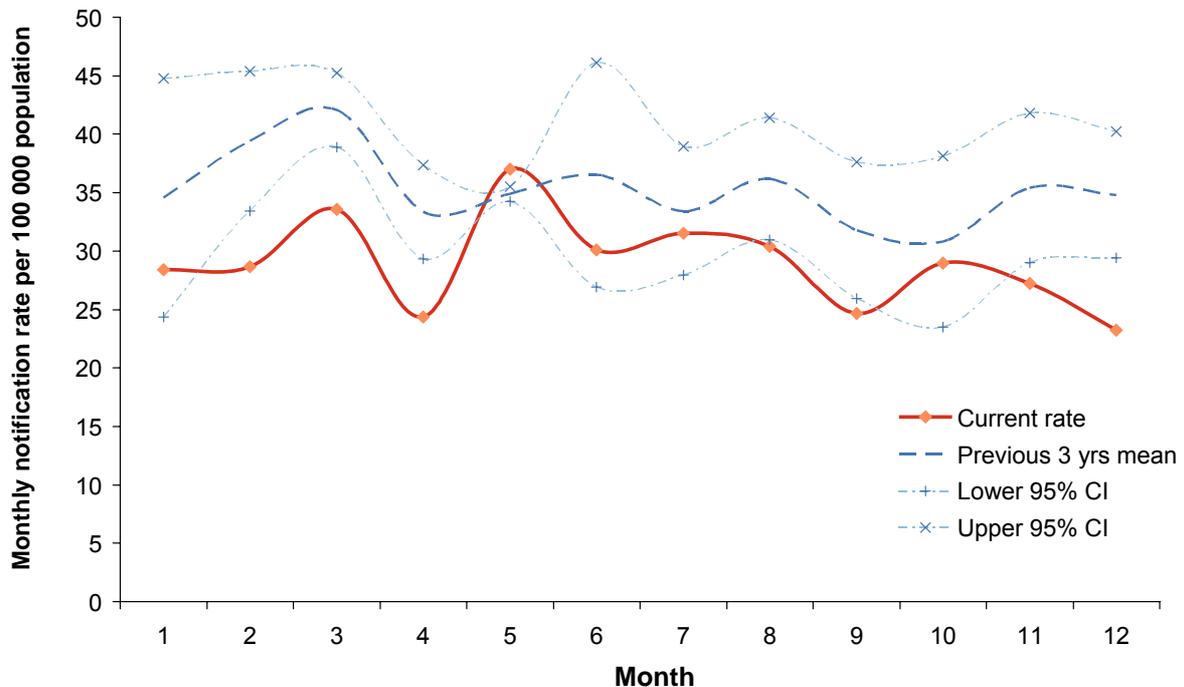
4.8.3.3 Geographic distribution of giardiasis notifications

Notification rates of giardiasis varied throughout the country (Figure 21). Since 2003 there has been a steady decrease in the rate of giardiasis in most district health boards (DHB), especially in the West Coast DHB. In 2006 the highest giardiasis notification rates were reported in Northland, Waikato and Whanganui DHBs.

Figure 21:   Geographic distribution of giardiasis notifications, 2003-2006
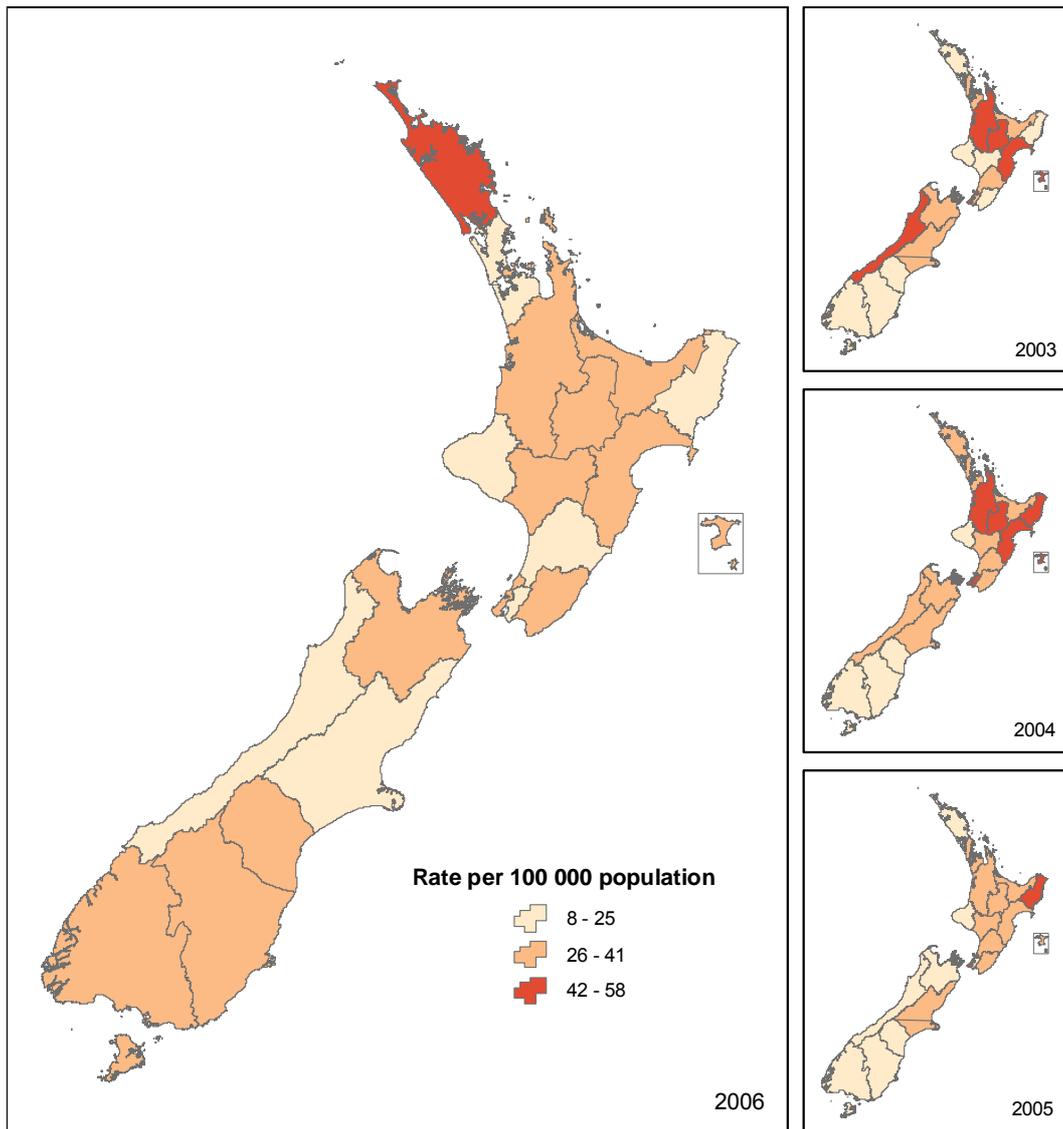
4.8.3.4   Age and sex distribution of giardiasis cases

The giardiasis notification and hospitalisation rates are slightly higher for males than females (Table 22).

Table 22:   Giardiasis cases by sex, 2006

Sex	EpiSurv notifications		Hospitalisations[a]		Deaths recorded in EpiSurv
	No.	Rate	No.	Rate	No.
Male	620	30.3	39	1.9	
Female	566	26.5	32	1.5	
Unknown	28				
Total	**1 214**	**29.0**	**71**	**1.7**	**-**

[a] NZHIS morbidity data for hospital admissions

In 2006 the highest age-specific giardiasis notification rates were in those aged one to four years (111.9 per 100 000) followed by the 30-39 year age group (48.3 per 100 000) and cases aged less than one year (40.6 per 100 000) (Table 23). The highest hospitalisation rates were in those aged less than one year.

Table 23:   Giardiasis cases by age group, 2006

Age groups	EpiSurv notifications		Hospitalisations[a]		Deaths recorded in EpiSurv
	No.	Rate	No.	Rate	No.
<1	24	40.6	6	10.2	
1 to 4	254	111.9	13	5.7	
5 to 9	76	26.0	7	2.4	
10 to 14	24	7.7	4	1.3	
15 to 19	17	5.4	2	0.6	
20 to 29	122	22.1	8	1.5	
30 to 39	289	48.3	7	1.2	
40 to 49	160	25.5	3	0.5	
50 to 59	117	23.1	8	1.6	
60 to 60	83	24.3	4	1.2	
70+	41	11.5	9	2.5	
Unknown	7	-			
Total	**1 214**	**29.0**	**71**	**1.7**	**-**

[a] NZHIS Morbidity data for hospital admissions

4.8.3.5   Risk Factors Reported

The most commonly reported risk factor for giardiasis notification cases was consumption of untreated water (36.3%). Other frequently reported risk factors included contact with faecal matter (31.5%) and contact with other symptomatic people (30.8%) (Table 24).

Table 24:   Exposure to risk factors associated with giardiasis, 2006

	Notifications			
Risk Factor	Yes	No	Unknown	%[a]
Consumed untreated water	169	296	749	36.3%
Contact with faecal matter	151	328	735	31.5%
Contact with other symptomatic people	147	330	737	30.8%
Recreational water contact	139	362	713	27.7%
Contact with farm animals	140	368	706	27.6%
Consumed food from retail premises	101	272	841	27.1%
Travelled overseas during the incubation period	158	436	620	26.6%
Contact with a confirmed case of same disease	108	417	689	20.6%
Contact with sick animals	24	416	774	5.5%

[1]Percentage refers to the cases that answered "yes" out of the total number of cases for which this information was supplied. Cases may have more than one risk factor recorded.

The risk factors associated with giardiasis cases have remained consistent from 2002 until 2006 (Figure 22). From 2002 onwards the trend suggests a growing importance of overseas travel during the incubation period and the decreasing importance of contact with faecal matter.

Figure 22:   Giardiasis risk factors by percentage of cases and year, 2002 – 2006
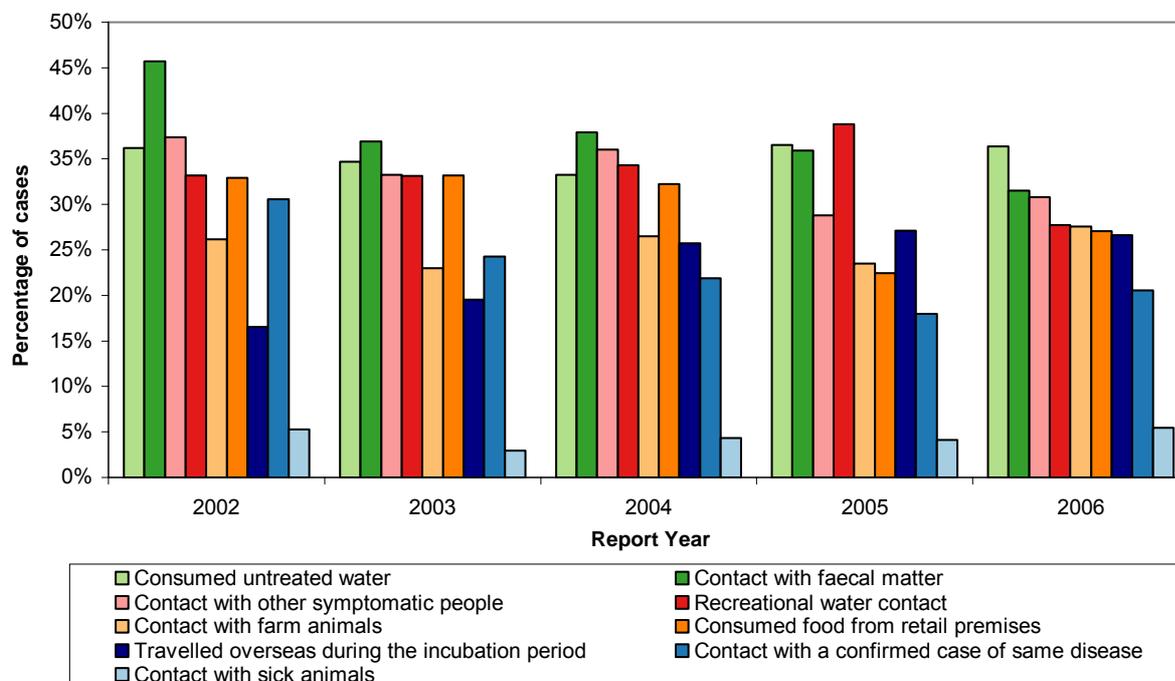
4.8.3.6   Estimate of travel-related cases

For cases where information on travel was provided, 26.6% (95%CI 22.6-30.9%) had travelled overseas during the incubation period. Assuming that the cases for which travel information was provided were representative of all giardiasis cases, a Poisson distribution can be used to estimate the total number of potentially travel related cases of giardiasis in 2006. The resultant distribution has a mean of 323 cases (95% CI 264-387).

4.8.4   Outbreaks reported as caused by *Giardia* spp

In 2006 there were 32 giardiasis outbreaks reported with two of these associated with a suspected or known foodborne source (Table 25).

Table 25:   *Giardia* spp. outbreaks reported, 2006

Measure (No.)	Foodborne *Giardia* spp. outbreaks	All *Giardia* spp. outbreaks
Outbreaks	2	32
Cases	4	98
Hospitalised cases	0	0

Since 2003 one or two foodborne giardiasis outbreaks have been reported in EpiSurv each year (Figure 23). These outbreaks involved a small number of cases.

Figure 23: **Foodborne *Giardia* spp. outbreaks and associated cases of reported by year, 2000 – 2006**
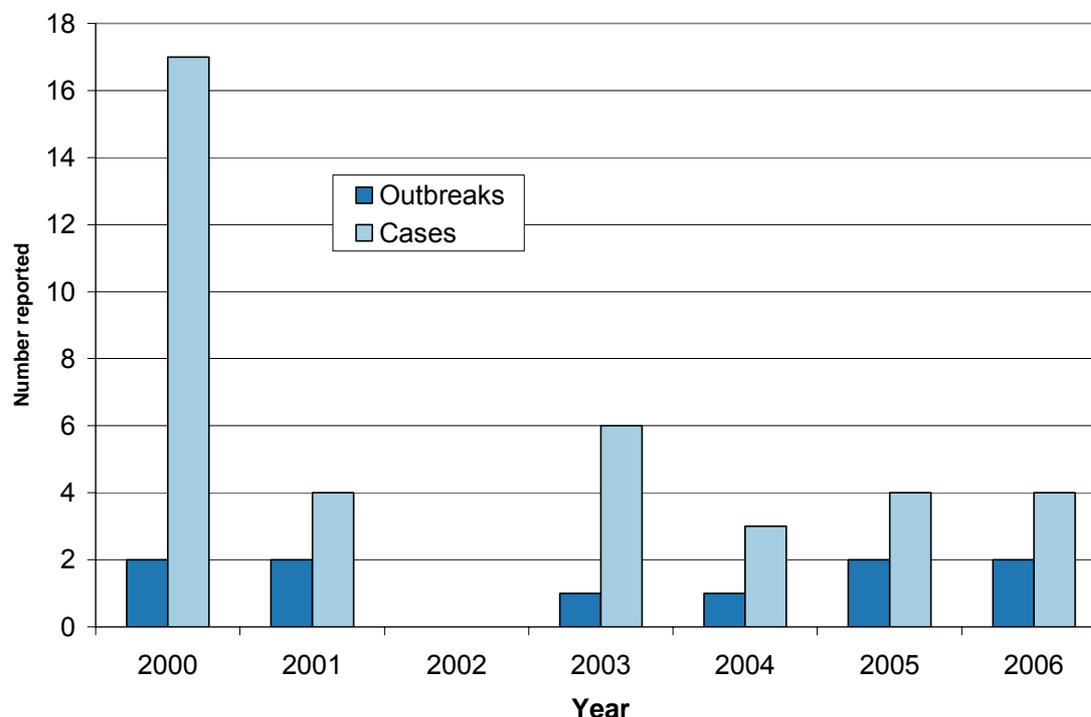
4.8.4.1 Details of food-associated outbreaks

Table 26 contains details of the two food–associated *Giardia* spp. outbreaks reported in 2006.

Table 26:   **Details of food-associated *Giardia* spp. outbreaks, 2006**

Public Health Unit (Month)	Suspected vehicle	Setting	Number ill	Confirmation
Tauranga (June)	Unknown	Restaurant/café	2C	6
South Canterbury (February)	Meats, Kava	Other (Fijian village)	2C	2

C = confirmed, P = probable
Confirmation:
1 = Environmental investigation – identified critical control point failures linked to implicated source
2 = Epidemiological – case had history of exposure to implicated source
3 = Epidemiological – case control or cohort study showed elevated risk for cases to implicated source
4 = Laboratory – pathogen suspected to have caused illness identified in food handler
5 = Laboratory – pathogen suspected to have caused illness identified in implicated source (food)
6 = No evidence
7 = Other evidence

4.8.4.2 Laboratory investigation of samples from suspected foodborne outbreaks

During investigations of suspected foodborne illness outbreaks by ESR's Public Health Laboratories, *Giardia* was not detected in any samples analysed.

4.8.5   Relevant New Zealand studies and publications

Nil.

4.8.6   Relevant regulatory developments

Nil.

4.9   Hepatitis A

Summary data for hepatitis A in 2006 are given in Table 27.

Table 27:   Summary surveillance data for hepatitis A, 2006

Parameter	Value in 2006	Section reference
Number of cases	123	4.9.2
Rate (per 100 000)	2.9	4.9.2
Hospitalisations (%)	47 (38.2%)	4.9.2
Deaths (%)	Nil	4.9.2
Estimated travel-related cases (%)	56 (45.9%)	4.9.3.6
Estimated food-related cases (%)	NA	

NA = not applicable, no information is available on the food attributable proportion of hepatitis A in New Zealand

4.9.1   Case definition

Clinical description:	An illness with a discrete onset of symptoms (fever, malaise, anorexia, nausea, or abdominal discomfort) with jaundice and/or elevated serum aminotransferase levels
Laboratory test for diagnosis:	Positive anti HAV IgM in serum
Case classification:	
Probable	A clinically compatible illness that is either a contact of a confirmed case of the same disease, or has had contact with the same common source i.e., is part of an identified common source outbreak
Confirmed	A clinically compatible illness that is laboratory confirmed

4.9.2   Hepatitis A cases reported in 2006 by data source

During 2006, 123 notifications (2.9 cases per 100 000 population) of hepatitis A were reported in EpiSurv.

The ICD-10 code B15 was used to extract hepatitis A hospitalisation data from the NZHIS NMDS database. Of the 47 hospital admissions (1.1 admissions per 100 000 population) recorded in 2006, 33 were reported with hepatitis A as the primary diagnosis and 14 with hepatitis A as another relevant diagnosis.

No deaths resulting from hepatitis A were recorded in EpiSurv.

4.9.3 Notifiable disease data

4.9.3.1 Annual notification trend

There has been a general decrease in the number of hepatitis A notifications since 1997 (347 cases) with the exception of a small increases in 2002 and 2006 (Figure 24). The 123 cases reported in 2006 was the largest number recorded since 1998 (145 cases) and was more double the number reported in 2005 (51 cases).

Figure 24:   Hepatitis A notifications by year, 1996-2006
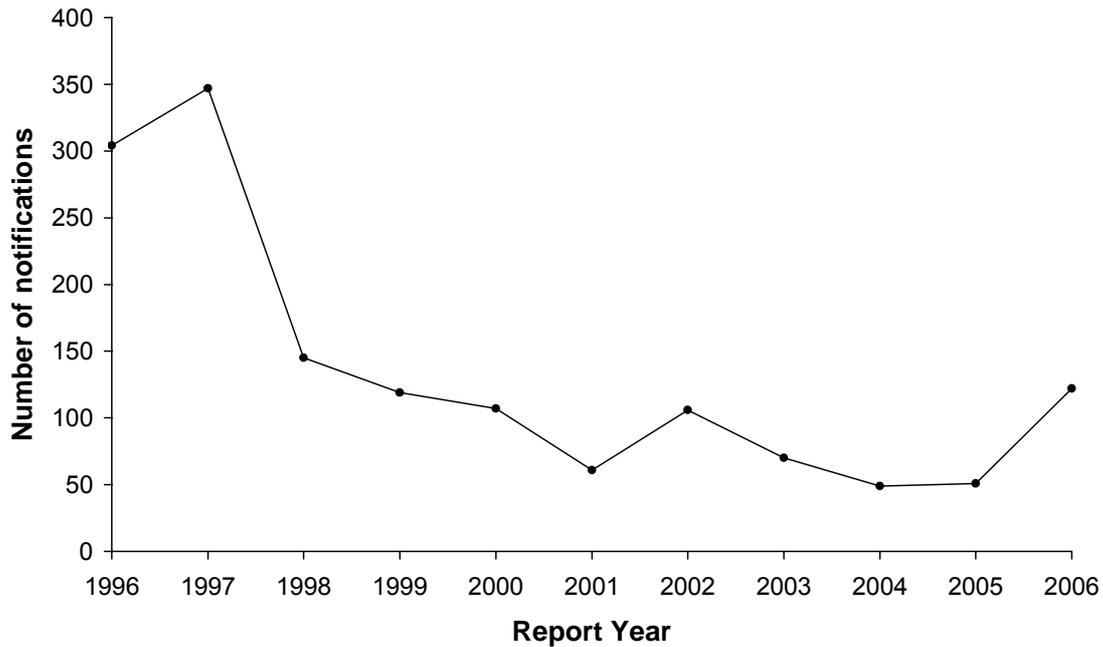
Hepatitis A notification rates varied throughout the six year period, 2000 to 2006 (Figure 25). The highest hepatitis A notification rate for the period was in 2006 with the previous two years experiencing the lowest notification rate (1.2 per 100 000 population).

Figure 25:   Hepatitis A notification rate by year, 2000-2006
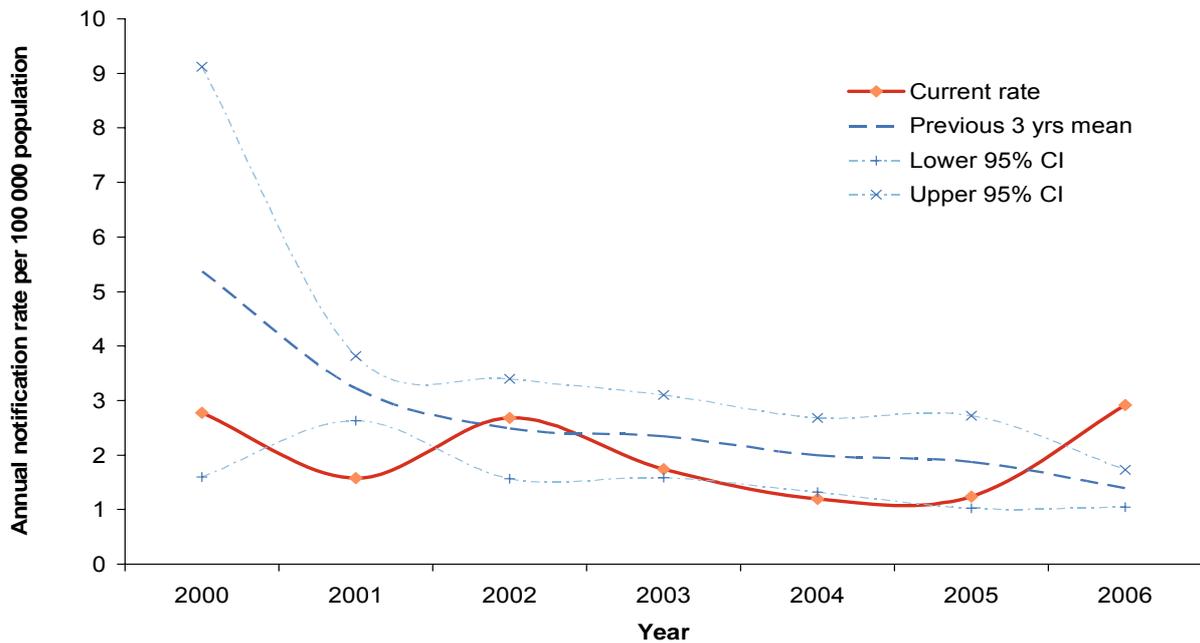
4.9.3.2   Seasonality

In 2006 there was an unusually large number of hepatitis A notifications reported at the beginning of the year (Figure 26). Many of these cases were linked to a hepatitis A outbreak in Christchurch associated with multiple modes of transmission including foodborne.

Figure 26:   Hepatitis A monthly rate (annualised) for 2006
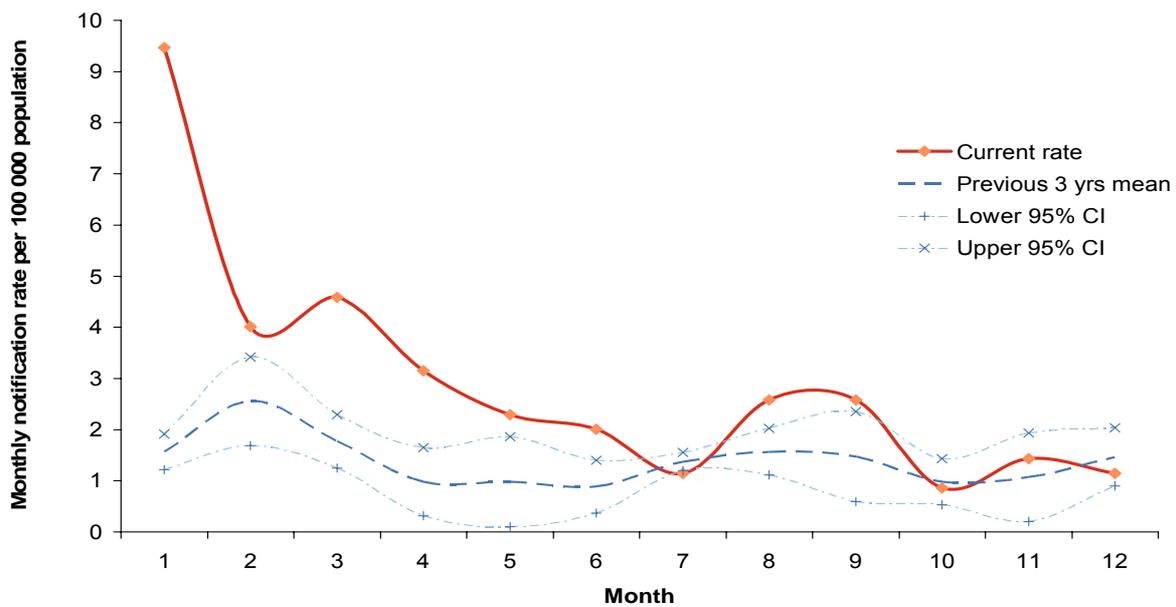
4.9.3.3 Age and sex distribution of Hepatitis A cases

In 2006 the hepatitis A notification and hospitalisation rate was slightly higher for males than females (Table 28).

Table 28:   Hepatitis A cases by sex, 2006

Sex	EpiSurv notifications		Hospitalisations[a]		Deaths recorded in EpiSurv
	No.	Rate	No.	Rate	No
Male	65	3.2	28	1.4	
Female	56	2.6	19	0.9	
Unknown	2	-	0	-	
Total	**123**	**2.9**	**47**	**1.1**	

[a] NZHIS morbidity data for hospital admissions

The age-specific hepatitis A notification rate in 2006 was highest for those aged one to four years (7.1 per 100 000), followed by five to nine year olds (6.5) and 15 to 19 year olds (4.5) (Table 29).

Table 29:   Hepatitis A cases by age group, 2006

Age groups	EpiSurv notifications		Hospitalisations[a]		Deaths recorded in EpiSurv
	No.	Rate	No.	Rate	No.
<1	0	0.0	0	0.0	
1 to 4	16	7.1	1	0.4	
5 to 9	19	6.5	5	1.7	
10 to 14	11	3.5	2	0.6	
15 to 19	14	4.5	6	1.9	
20 to 29	11	2.0	9	1.6	
30 to 39	17	2.8	5	0.8	
40 to 49	11	1.8	4	0.6	
50 to 59	10	2.0	8	1.6	
60 to 60	6	1.8	6	1.8	
70+	8	2.2	1	0.3	
Unknown	0	-	0	-	
Total	**123**	**2.9**	**47**	**1.1**	**-**

[a] NZHIS morbidity data for hospital admissions

4.9.3.4 Risk Factors Reported

The most commonly reported risk factor for hepatitis A in 2006 was contact with contaminated food or drink and was reported by all cases (Table 30). Other frequently reported risk factors included overseas travel during the incubation period (45.9%) and household contact with a confirmed case (34.3%).

Table 30:   Exposure to risk factors associated with hepatitis A, 2006

	Notifications			
Risk Factor	Yes	No	Unknown	%ᵃ
Contact with contaminated food or drink	122	0	1	100.0%
Travelled overseas during the incubation period	51	60	12	45.9%
Household contact with confirmed case	35	67	21	34.3%
Contact with confirmed case in previous 3 months	21	71	31	22.8%
Occupational exposure to human sewage	4	93	26	4.1%
Sexual contact involving possible faecal-oral transmission	3	87	33	3.3%

[a]Percentage refers to the cases that answered "yes" out of the total number of cases for which this information was supplied. Cases may have more than one risk factor recorded.

Between 2002 and 2006 the risk factors associated with hepatitis A cases have generally occurred in the same order of importance and with the same magnitude each year (Figure 27). Every year contact with contaminated food or drink has been reported by all hepatitis A cases.

From 2002 to 2005 an increasing number of cases reported overseas travel during the incubation period as a risk factor. The percentage of cases with a risk factor of household contact with a confirmed case and contact with a confirmed case in the previous three months was considerably higher during 2006 than in previous years due to the hepatitis A outbreak mentioned above. The initial cases were overseas during the incubation period and subsequent cases reported risk factors of person to person contact and foodborne contamination.

Figure 27:   Hepatitis A risk factors by percentage of cases and year, 2002 – 2006
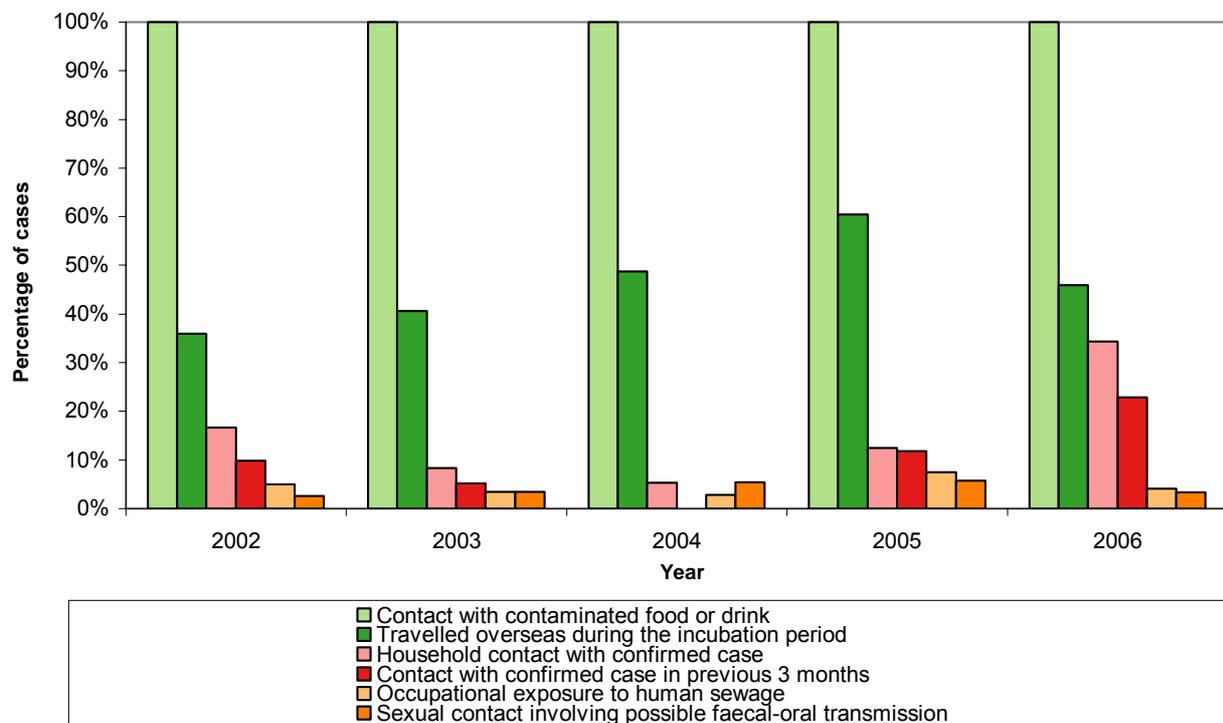
4.9.3.5 Estimate of travel-related cases

For cases where information on travel was provided, 45.9% (95%CI 33.6-58.9%) had travelled overseas during the incubation period. Assuming that the cases for which travel information was provided were representative of all hepatitis A cases, a Poisson distribution can be used to estimate the total number of potentially travel related cases of hepatitis A in 2006. The resultant distribution has a mean of 56 cases (95% CI 36-79).

If data from the last four years are considered the estimated proportion of cases travelling overseas within the incubation period of the organism is 46.0% (95% CI 38.1-54.6%).

4.9.4   Outbreaks reported as caused by hepatitis A virus

During 2006 one of the eight hepatitis A outbreaks reported in EpiSurv was associated with a suspected or known foodborne source (Table 31). Thirty four cases were associated with the outbreak, with three people being hospitalised. This outbreak was associated with multiple transmission modes.

Table 31:   Hepatitis A virus outbreaks reported, 2006

Measure (No.)	Foodborne Hepatitis A virus outbreaks	All Hepatitis A virus outbreaks
Outbreaks	1	8
Cases	34	81
Hospitalised cases	3	6

Foodborne hepatitis A virus outbreaks are rare with only two reported in the period 2002 to 2007 (in 2002 and 2006) (Figure 28). Although occurring infrequently, each foodborne outbreak has been associated with many cases.

Figure 28: **Foodborne hepatitis A virus foodborne outbreaks and associated cases reported by year, 2000–2006**
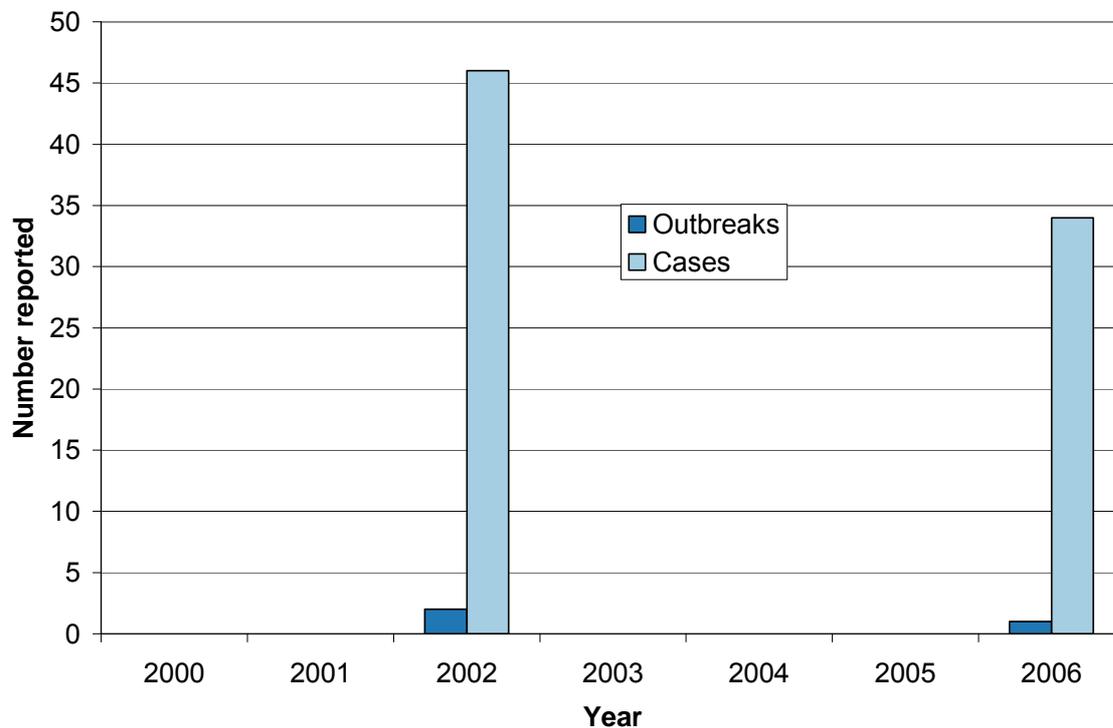
4.9.4.1 Details of food-associated outbreaks

Table 32 contains details of the food –associated hepatitis A outbreak reported in 2006.

Table 32: **Details of food-associated hepatitis A virus outbreak, 2006**

Public Health Unit (Month)	Suspected vehicle	Setting	Number ill	Confirmation
Canterbury (January)	Person-to-person	Restaurant/café, childcare, home	34C	2

C = confirmed, P = probable
Confirmation:
1 = Environmental investigation – identified critical control point failures linked to implicated source
2 = Epidemiological – case had history of exposure to implicated source
3 = Epidemiological – case control or cohort study showed elevated risk for cases to implicated source
4 = Laboratory – pathogen suspected to have caused illness identified in food handler
5 = Laboratory – pathogen suspected to have caused illness identified in implicated source (food)
6 = No evidence
7 = Other evidence

While foodborne transmission was identified in relation to this outbreak, no specific foods were identified.

4.9.4.2 Laboratory investigation of samples from suspected foodborne outbreaks

During investigations of suspected foodborne illness outbreaks by ESR's Public Health Laboratories, no samples were found to contain hepatitis A virus.

4.9.5   Relevant New Zealand studies and publications

An alert was issued in the media following discovery that a food handler at an Auckland McDonald's restaurant had hepatitis A.
http://www.nzherald.co.nz/topic/story.cfm?c_id=304&objectid=10417259

A press release issued by NZFSA and the New Zealand Foodsafe Partnership confirmed the importance of hand hygiene in preventing spread of hepatitis A and other pathogenic micro-organisms.
http://www.nzfsa.govt.nz/publications/media-releases/2006-01-17.htm

4.9.6   Relevant regulatory developments

Nil.

4.10   Histamine (Scombroid) Fish Poisoning

4.10.1 Case definition

Clinical description:	Tingling and burning sensation around mouth, facial flushing, sweating, nausea and vomiting, headache, palpitations, dizziness and rash
Laboratory test for diagnosis:	Detection of histamine levels ≥ 50mg/100g fish muscle
Case classification:	Not applicable

4.10.2 Histamine (scombroid) fish poisoning cases reported in 2006 by data source

No cases of histamine (scombroid) fish poisoning were reported in EpiSurv during 2006.

The ICD-10 code T61.1 was used to extract scombroid fish poisoning hospitalisation data from the NZHIS NMDS database. Of the 5 hospital admissions (0.1 admissions per 100 000 population) recorded in 2006, all were reported with scombroid fish poisoning as the primary diagnosis.

4.10.3 Outbreaks reported as caused by histamine (scombroid) fish poisoning

Four histamine (scombroid) fish poisoning outbreaks were reported in 2006 involving a total of 14 associated cases, with one case hospitalised (Table 33). All outbreaks reported foodborne transmission.

Table 33:   Histamine (scombroid) fish poisoning outbreaks reported, 2006

Measure (No.)	Foodborne histamine fish poisoning outbreaks	All histamine fish poisoning outbreaks
Outbreaks	4	4
Cases	14	14
Hospitalised cases	1	1

Between 2000 and 2006 the number of foodborne histamine (scombroid) fish poisoning outbreaks reported each year has ranged from one to six (Figure 29). The highest number of outbreaks was reported in 2004 (6 outbreaks, 21 cases) but the highest total number of associated cases was reported in 2002 (5 outbreaks, 32 cases). Since 2002, the total number of cases associated with the outbreaks has generally decreased.

Figure 29:   Histamine (scombroid) fish poisoning outbreaks and associated cases reported by year, 2000 – 2006
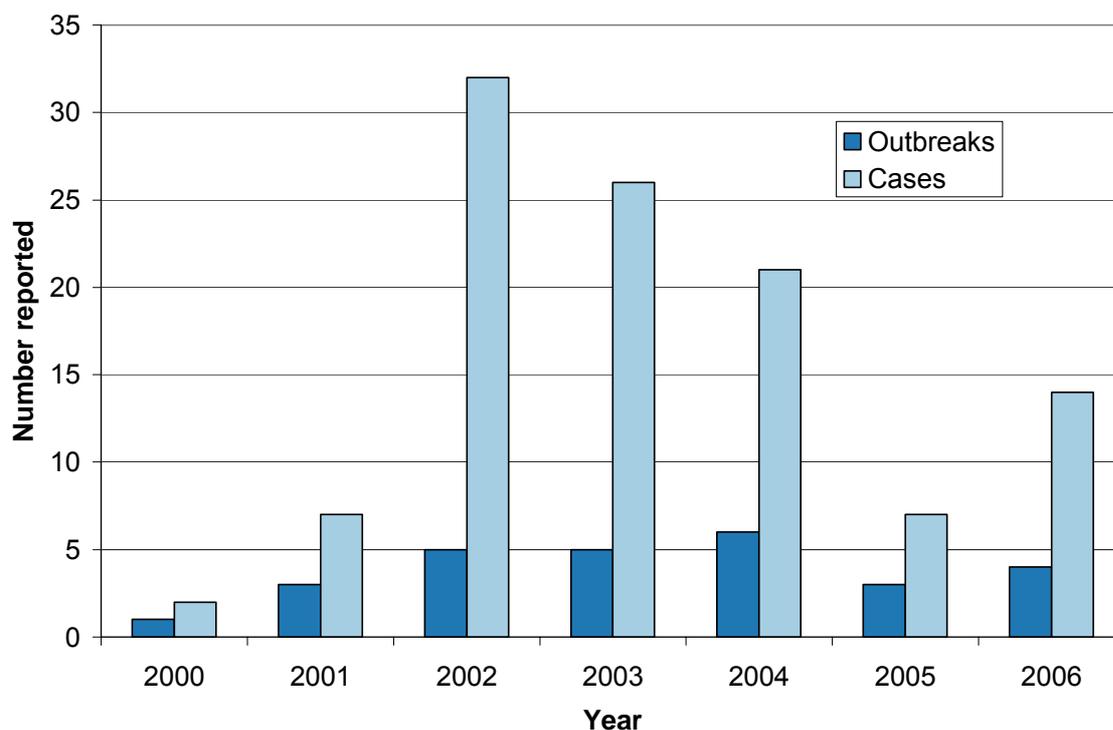
4.10.3.1 Details of food-associated outbreaks

Table 32 contains details of the four food–associated histamine poisoning outbreaks reported in 2006.

Table 34:   Details of food-associated histamine poisoning outbreaks, 2006

Public Health Unit (Month)	Suspected vehicle	Setting	Number ill	Confirmation
Auckland (January)	Smoked tuna	Home, Takeaway	2P	2, 5
Auckland (February)	Fish	Supermarket	4C, 2P	2, 5
Auckland (November)	Smoked kawahai	Fisk smoking plant, supermarket	2C	2, 5
Rotorua (February)	Kingfish	Restaurant/café	4P	5

C = confirmed, P = probable
Confirmation:
1 = Environmental investigation – identified critical control point failures linked to implicated source
2 = Epidemiological – case had history of exposure to implicated source
3 = Epidemiological – case control or cohort study showed elevated risk for cases to implicated source
4 = Laboratory – pathogen suspected to have caused illness identified in food handler
5 = Laboratory – pathogen suspected to have caused illness identified in implicated source (food)
6 = No evidence
7 = Other evidence

4.10.3.2 Laboratory investigation of samples from suspected foodborne outbreaks

During investigations of suspected foodborne illness outbreaks by ESR's Public Health Laboratories, analyses were carried out on fish samples from the four outbreaks listed in Table 32. Additional samples were analysed from a two person outbreak in the Waikato. The histamine concentrations in fish samples analysed in relation to outbreaks were in the range 470-4100 mg/kg.

4.10.4 Relevant New Zealand studies and publications

Nil.

4.10.5 Relevant regulatory developments

Nil.

4.11   Listeriosis

Summary data for listeriosis in 2006 are given in Table 35.

Table 35:   Summary surveillance data for listeriosis, 2006

Parameter	Value in 2006	Section reference
Number of cases	19	4.11.2
Rate (per 100 000)	0.5	4.11.2
Hospitalisations (%)	23 (121%)	4.11.2
Deaths (%)	1 (5.2%)	4.11.2
Estimated travel-related cases (%)	0 (0%)	4.11.3.4
Estimated food-related cases (%)*	16 (85%)	4.11.2

* For estimation of food-related cases it was assumed that the proportions derived from expert consultation would exclude travel-related cases

4.11.1 Case definition

Clinical description: An infection which produces several clinical syndromes including stillbirths, listeriosis of the newborn, meningitis, bacteraemia, or localised infections. Pregnant women, the immunosuppressed and the frail elderly are at greatest risk

Laboratory test for diagnosis: Isolation of *Listeria monocytogenes* from a site that is normally sterile, including the foetal gastrointestinal tract

Case classification:
Probable   Not applicable
Confirmed   A clinically compatible illness that is laboratory confirmed

4.11.2 Listeriosis cases reported in 2006 by data source

During 2006, 19 notifications (0.5 cases per 100 000 population) of listeriosis were reported in EpiSurv, of which two were perinatal. Twenty cultures were received by the ESR Special Bacteriology Laboratory.

The ICD-10 code A32 was used to extract listeriosis hospitalisation data from the NZHIS NMDS database. Of the 23 hospital admissions (0.5 admissions per 100 000 population) recorded in 2006, 13 were reported with listeriosis as the primary diagnosis and 10 with listeriosis as another relevant diagnosis.

One perinatal death was recorded in EpiSurv in 2006.

It has been estimated by expert consultation that 85% (minimum = 78%, maximum = 92%) of listeriosis incidence is due to foodborne transmission. It was further estimated that approximately 50% of foodborne transmission was due to consumption of ready-to-eat meats, while approximately 7% was due to ice cream consumption.

4.11.3 Notifiable disease data

4.11.3.1 Annual notification trend

The number of listeriosis notifications has remained consistent between years (Figure 30). The highest number of notifications was reported in 1997 followed by 2004. Two (10.5%) of the 2006 cases were recorded as perinatal, a decrease from 2005 (5 cases) and similar to 2004 (3 cases).

Figure 30:   **Listeriosis non-perinatal and perinatal notifications by year, 1996-2006**
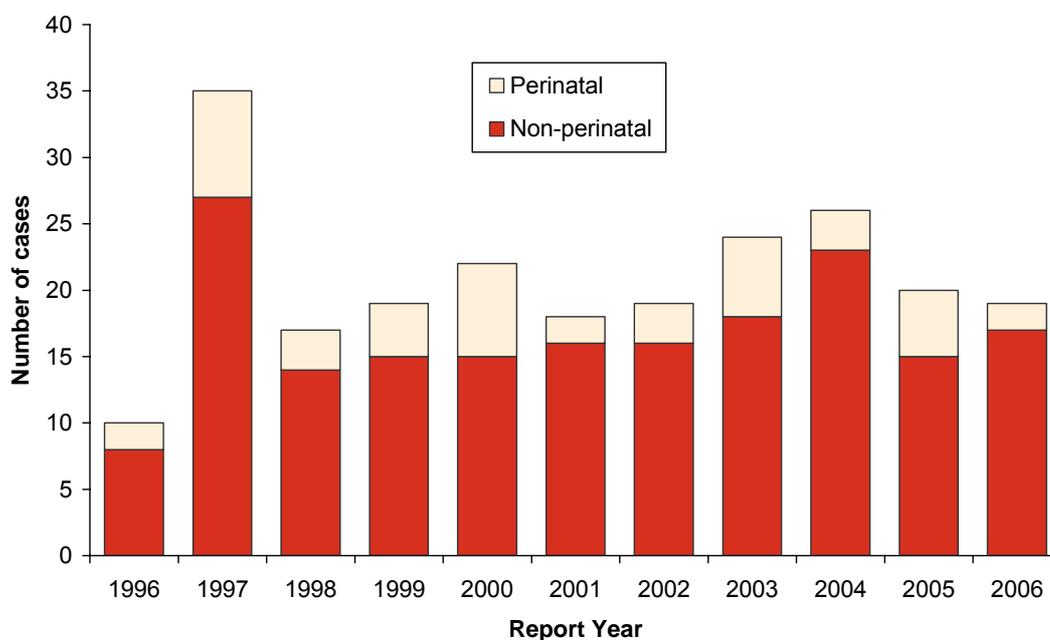
4.11.3.2 Age and sex distribution of listeriosis cases

In 2006 the number and rate of notifications for listeriosis was similar for males and females but more males than females were reported as hospitalised (Table 36).

Table 36: Listeriosis cases by sex, 2006

Sex	EpiSurv notifications		Hospitalisations[a]		Deaths recorded in EpiSurv[b]
	No.	Rate	No.	Rate	No.
Male	10	0.5	15	0.7	
Female	7	0.3	8	0.4	
Unknown	2				
Total	**19**	**0.5**	**23**	**0.5**	

[a] NZHIS morbidity data for hospital admissions

[b] Perinatal cases are recorded in terms of the mother's demography and perinatal deaths are not recorded in this table

In 2006 the age specific listeriosis notification rates were highest in the 70+ years age group (6 cases, 1.7 per 100 000 population), followed by the 60 to 69 years age group (4 cases, 1.2 per 100 000) (Table 37). The highest hospitalisation rates were in the 60 to 69 years age group.

Table 37: Listeriosis cases by age group, 2006

Age groups	EpiSurv notifications		Hospitalisations[a]		Deaths recorded in EpiSurv[b]
	No.	Rate	No.	Rate	No.
<1	1	1.7	1	1.7	
1 to 4	1	0.4	1	0.4	
5 to 9	0	0.0	0	0.0	
10 to 14	0	0.0	0	0.0	
15 to 19	0	0.0	0	0.0	
20 to 29	2	0.4	1	0.2	
30 to 39	1	0.2	2	0.3	
40 to 49	2	0.3	3	0.5	
50 to 59	2	0.4	5	1.0	
60 to 69	4	1.2	6	1.8	
70+	6	1.7	4	1.1	
Total	**19**	**0.5**	**23**	**0.5**	

[a] NZHIS morbidity data for hospital admissions

[b] Perinatal cases are recorded in terms of the mother's demography and perinatal deaths are not recorded in this table

4.11.3.3 Risk Factors Reported

In 2006 the most common risk factor reported for listeriosis was an underlying illness (69.2%), hospital admission for another illness (57.1%), and receiving immunosuppressive drugs (25.0%) (Table 38).

Table 38:   Exposure to risk factors associated with listeriosis, 2006

	Notifications			
Risk Factor	**Yes**	**No**	**Unknown**	**%[a]**
Underlying illness	9	4	4	69.2%
Admitted to hospital for treatment of another illness	8	6	3	57.1%
Received immunosuppressive drugs	3	9	5	25.0%
Travelled overseas during the incubation period	0	7	10	0.0%

[a]Percentage refers to the cases that answered "yes" out of the total number of cases for which this information was supplied. Cases may have more than one risk factor recorded. Perinatal cases are excluded from this analysis.

Between 2002 and 2006 the risk factors associated with listeriosis cases have generally occurred in a similar order of importance each year (Figure 31). Every year an underlying illness was the risk factor most commonly reported for listeriosis. Overseas travel is not reported to be an important risk factor for listeriosis.

Figure 31:   Listeriosis risk factors by percentage of cases and year, 2002 – 2006
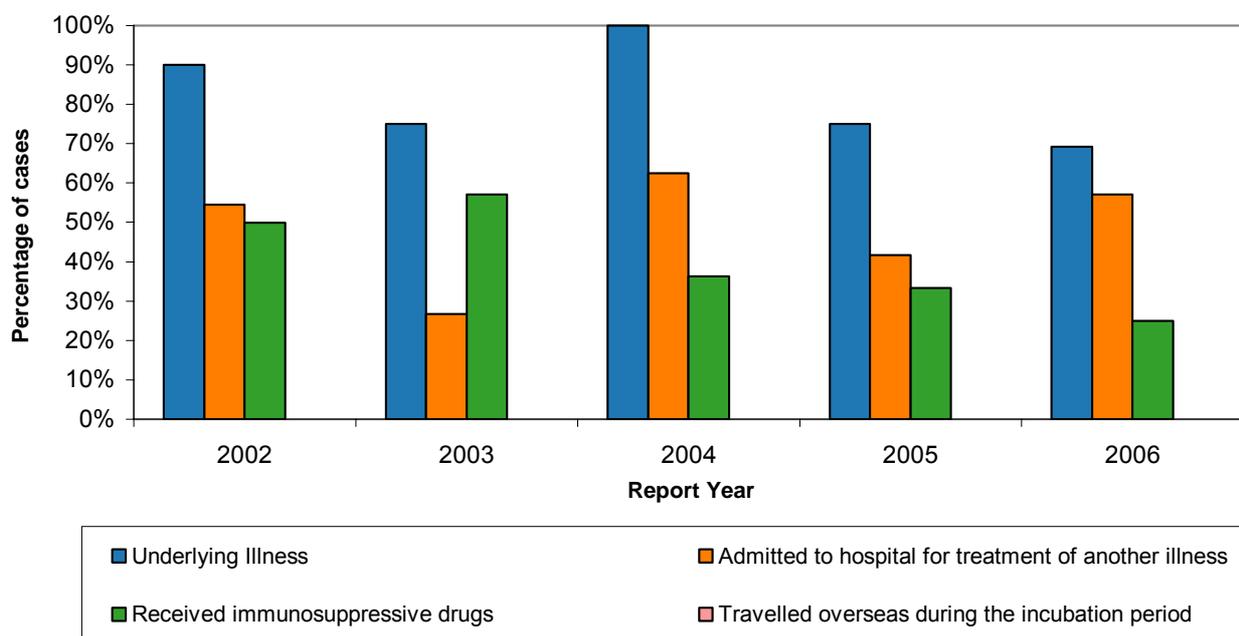
4.11.3.4 Estimate of travel-related cases

No cases reported overseas travel within the incubation period for the disease during 2006.

4.11.4 Outbreaks reported as caused by *Listeria* spp.

No listeriosis outbreaks were reported in EpiSurv in 2006.

4.11.4.1 Laboratory investigation of samples from suspected foodborne outbreaks

During investigations of suspected foodborne illness outbreaks by ESR's Public Health Laboratories, no samples were found to contain *Listeria monocytogenes.*

4.11.5 Recent Surveys

Exposure Assessment of *Listeria monocytogenes* via Unpackaged Ready-to-eat Meats (Hudson, 2006)

The aim of the project was to produce data on the prevalence and numbers of *L. monocytogenes* in unpackaged ready-to-eat ham to enable this transmission route to be assessed in terms of relative risk to the population, and to inform risk mitigation prioritisation.

A total of 301 unpackaged ham samples purchased from retail outlets in Auckland, Wellington and Christchurch were examined for the presence and number of *L. monocytogenes* after storage in a laboratory refrigerator for seven days at 5°C prior to analysis (to simulate domestic storage conditions). Of the samples tested 13 (4.3%) contained the pathogen. Eight contained the organism at <50 CFU/g three contained 50 CFU/g, one 1.5×10^2 CFU/g and one 1.6×10^3 CFU/g. In addition 13 samples contained other *Listeria* spp., 11 containing *L. innocua* and two *L. welshimeri* at <50 CFU/g. *L. monocytogenes* and *L. innocua* were isolated from the same sample on three occasions.

In other experiments attempts were made to identify *Listeria*-contaminated batches of retail ham and to incubate them at 5°C over approximately three weeks to assess the rate of growth of natural contaminants in this food. No batch of ham in which growth occurred, or where *Listeria* spp. could be isolated on repeated sampling, contained *L. monocytogenes*. However growth curves were obtained for one sample containing *L. welshimeri* and four samples containing *L. innocua*. Growth was slow in all but one sample; with most in the range of approximately 0.002 to 0.004 log h^{-1}. However, in one sample *L. innocua* grew at 0.02 log h^{-1}, although the maximum number reached was only $4.0\text{-}5.0 \times 10^3$ CFU/g. In five other batches of ham *Listeria* spp. could be detected intermittently during incubation, indicating that little growth, if any, occurred.

In conclusion, *L. monocytogenes* was isolated infrequently from the ham samples tested. When present the pathogen was usually at low numbers, but the two samples exceeding 100 CFU/g indicate that improvements in handling of this food in retail outlets is desirable. Growth of naturally occurring *Listeria* spp. in ham at refrigeration temperatures was generally slow or did not occur, and moderate levels were reached only after incubation periods which would be most unlikely to occur in foods deemed fit for consumption.

Exposure Assessment to *Listeria monocytogenes* via Deli Ready-to-eat Salads (with Dressings) (Wong, 2007)

A national quantitative survey of *L. monocytogenes* and other *Listeria* species in ready-to-eat (RTE) salads (with dressings) from retail outlets was undertaken in New Zealand from February 2006 to February 2007. The aim was to determine:
- The prevalence, numbers and genotypes of this pathogen in RTE salads;
- Whether pH and temperature hurdles are adequate in controlling listerial growth in the salads; and

- The management of shelf life of salads by suppliers and retailers.

Three hundred and two RTE salad samples were purchased from four main cities in New Zealand (Auckland, Wellington, Christchurch and Dunedin). RTE salads included bean, pasta, potato, pulse/seed, rice, seafood-based, coleslaw and miscellaneous (other salad varieties containing dressing from the delicatessen retail outlets that did not fit with the described designations). Salads under the various designations may also contain small amounts of cooked meats, cooked eggs, spices and fresh herbs.

All salads were enriched in buffered *Listeria* selective broth and screened for presence of *Listeria* spp. on PALCAM and ALOA solid media. *Listeria* spp were enumerated by spread plating sample homogenate onto PALCAM agar. The prevalence of *Listeria* spp. in RTE salads was 7.0% (95% confidence interval, 4.6 – 10.8) of which 4.6% (2.6 - 7.7) were contaminated with *Listeria monocytogenes*. One sample of coleslaw contained 100 CFU/g of *L. monocytogenes* while another contained 30 CFU/g. The remaining twelve samples produced a <10 CFU g^{-1} count of *L. monocytogenes*. Samples positive for other *Listeria* spp. also had a <10 CFU g^{-1} count.

Genotyping of the *L. monocytogenes* isolates by PFGE using two restriction enzymes, *Asc*I and *Apa*I, showed three patterns from the RTE salads were indistinguishable from human non-perinatal *L. monocytogenes* isolates archived in PulseNet Aotearoa. The PFGE patterns also showed that one salad producer could have one genotype that has colonised its plant. Cross-contamination by a delicatessen was also demonstrated.

Temperature and pH hurdles recorded from the salads showed that these parameters were only partially adequate (using a temperature of 5ºC and pH 4.6 as hurdle references) in controlling *Listeria* spp. from growing in RTE salads over the shelf life at retail. Better control of these hurdles by the suppliers and the retailers could assist in preventing the potential re-growth of *Listeria* spp. in RTE salads.

Information gathered indicated that the shelf life of most salads on display was for 1-2 days, but expiry dates on bulk salad packages suggested that the shelf-life recommended by suppliers could stretch up to 24 days for certain salad varieties. This is acceptable provided the pH is optimally controlled, i.e. ≤ pH 4.6. Therefore any failure to control the hurdles adequately at retail and in the home of the consumer would be compounded by the extended shelf life of most salads from the date of manufacture.

The data gathered in this study will be useful for any future revision of the *L. monocytogenes* in RTE salads risk profile. Feed back of information to the RTE salad supplier and retailer sectors could also improve the hurdle control needed to prevent potential *Listeria* regrowth in salads.

4.11.6 Relevant New Zealand studies and publications

Nil.

4.11.7 Relevant regulatory developments

Nil.

4.12   Norovirus Infection

4.12.1 Case definition

Clinical description: Gastroenteritis usually lasting 12-60 hours

Laboratory test for diagnosis: Detection of NLV in faecal or vomit specimen or leftover food

Case classification:

Probable   A clinically compatible illness that is either a contact of a confirmed case of the same disease, or has had contact with the same common source i.e., is part of an identified common source outbreak

Confirmed   A clinically compatible illness that is laboratory confirmed

4.12.2 Norovirus infection cases reported in 2006 by data source

The ICD-10 code A08.1 was used to extract norovirus infection hospitalisation data from the NZHIS NMDS database. Of the 58 hospital admissions (1.4 admissions per 100 000 population) recorded in 2006, 15 were reported with norovirus infection as the primary diagnosis and 43 with norovirus infection as another relevant diagnosis.

An expert consultation estimated that 40% of norovirus infections were due to foodborne transmission and of these 40% were due to consumption of molluscan shellfish.

4.12.3 Outbreaks reported as caused by norovirus

During 2006 there were 156 norovirus outbreaks reported in EpiSurv and of these 23 were associated with a suspected or known foodborne source (Table 39). A total of three hundred and forty-six cases were associated with these foodborne outbreaks.

Table 39:   Norovirus outbreaks reported, 2006

Measure (No.)	Foodborne norovirus outbreaks	All norovirus outbreaks
Outbreaks	23	156
Cases	346	3 945
Hospitalised cases	0	96

Since 2000 the number of foodborne associated norovirus outbreaks reported each year has ranged from 14 (in 2003) to 25 (in 2004). The total number of cases associated with these outbreaks has ranged from 131 (in 2005) to 346 (in 2006). In 2006 the highest number of outbreak associated norovirus cases was reported since 1999 when there were 35 outbreaks and 361 associated cases.

Figure 32:   Foodborne norovirus outbreaks and associated cases reported by year, 2000 – 2006
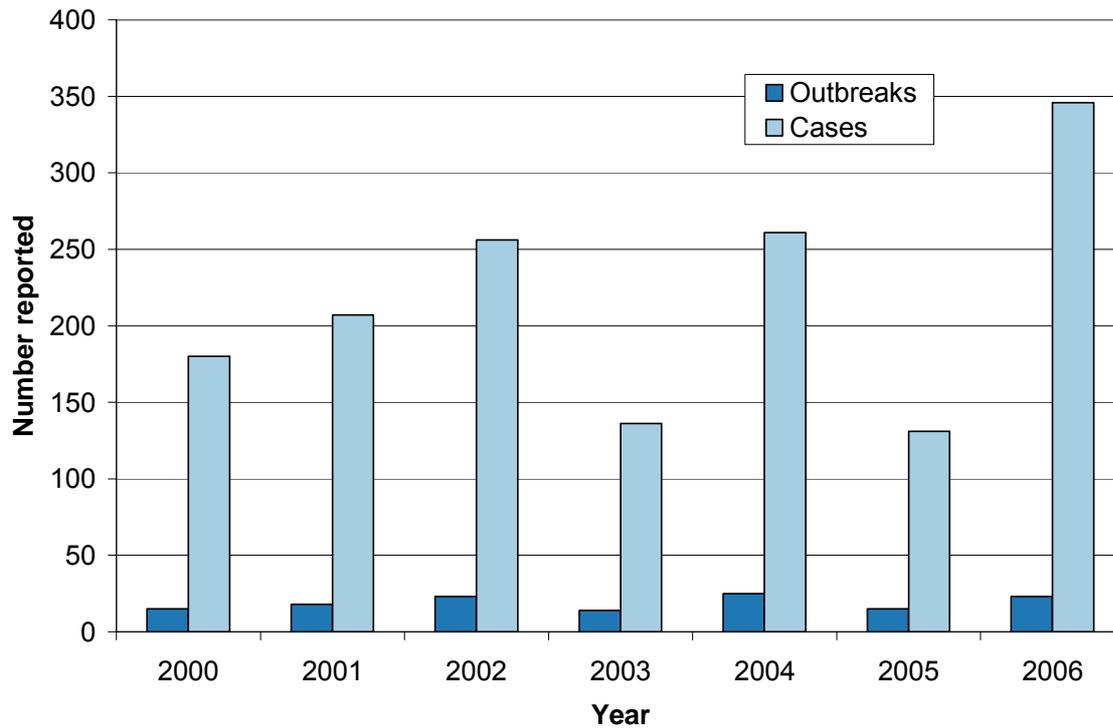
4.12.3.1 Details of food-associated outbreaks

Table 40 contains details of the 23 food–associated norovirus infection outbreaks reported in 2006

Table 40:   Details of food-associated norovirus outbreaks, 2006

Public Health Unit (Month)	Suspected vehicle	Setting	Number ill	Confirmation
Auckland (February)	Fish	Home	1C, 1P	6
Auckland (March)	Chinese meal	Takeaway	3C, 4P	1, 2
Auckland (March)	Meal	Restaurant/café, home	3C	2
Auckland (June)	Oysters	Sports arena	4C, 112P	1, 2, 3, 5
Auckland (July)	Ham	Home, unknown	1C, 1P	2
Auckland (August)	Food handler	Restaurant/café	1C, 5P	2, 4
Auckland (October)	Unknown	Takeaway	2C, 3P	1, 2
Auckland (October)	Precooked chicken	Home, unknown	2C, 7P	2
Canterbury (January)	Unknown	Restaurant/café	13P	2
Canterbury (June)	Unknown	Rest home	21C, 1P	2
Hawke's Bay (March)	Unknown	Restaurant/café	2P	None
Hawke's Bay (March)	Oysters	Caterers, home	2C, 7P	5
Manawatu (October)	Food handler	Restaurant/café, hotel/motel	5C, 5P	1, 2
Nelson (March)	Food handler	School	4C, 85P	2, 4
Otago (February)	Oysters	Home	3P	2, 5
Otago (July)	Oysters	Workplace	3C, 1P	7
Tauranga (May)	Oysters	Restaurant/café	1C, 2P	2, 5
Tauranga (October)	Chicken	Takeaway	1C, 1P	6

Public Health Unit (Month)	Suspected vehicle	Setting	Number ill	Confirmation
Taranaki (August)	Oysters	Restaurant/café	2C	5
Taranaki (December)	Unknown	Restaurant/café	10C	2
Wanganui (July)	Chicken meal	Takeaway	2C, 3P	6
Wellington (April)	Antipasto (and other items on menu)	Restaurant/café	19C	2, 4
Wellington (June)	Oysters	Restaurant/café	3C	2, 3

C = confirmed, P = probable
Confirmation:
1 = Environmental investigation – identified critical control point failures linked to implicated source
2 = Epidemiological – case had history of exposure to implicated source
3 = Epidemiological – case control or cohort study showed elevated risk for cases to implicated source
4 = Laboratory – pathogen suspected to have caused illness identified in food handler
5 = Laboratory – pathogen suspected to have caused illness identified in implicated source (food)
6 = No evidence
7 = Other evidence

Oysters were implicated in approximately one-third of norovirus-associated outbreaks (7/23; 30%). There was often stronger evidence implicating oysters (e.g. organism detected in suspect food) than for other food vehicles, due to the availability of methods to detect norovirus in oysters. Such methods are not generally available for foods.

4.12.3.2 Laboratory investigation of samples from suspected foodborne outbreaks

During investigations of suspected foodborne illness outbreaks by ESR's Public Health Laboratories, norovirus was detected in faecal samples from 90 investigations and oyster samples from six investigations. However, a large number of the positive faecal samples were from investigations into outbreaks in rest homes and were probably not foodborne.

4.12.4 <u>Relevant New Zealand studies and publications</u>

Nil.

4.12.5 <u>Relevant regulatory developments</u>

Nil.

4.13   Salmonellosis

Summary data for salmonellosis in 2006 are given in Table 41.

Table 41:   Summary surveillance data for salmonellosis, 2006

Parameter	Value in 2006	Section reference
Number of cases	1 335	4.13.2
Rate (per 100 000)	31.9	4.13.2
Hospitalisations (%)	161 (12.1%)	4.13.2
Deaths (%)	1 (0.07%)	4.13.2
Estimated travel-related cases (%)	257 (19.4)	4.13.3.6
Estimated food-related cases (%)*	658 (61%)	4.13.2

* For estimation of food-related cases it was assumed that the proportions derived from expert consultation would exclude travel-related cases

4.13.1 Case definition

Clinical description: Salmonellosis presents as gastroenteritis. Asymptomatic
                                  infections may occur

Laboratory test for diagnosis: Isolation of *Salmonella* species (excluding *S. typhi*) from any
                                  clinical specimen

Case classification:
Probable   A clinically compatible illness that is either a contact of a
                                  confirmed case of the same disease, or has had contact with
                                  the same common source i.e., is part of an identified
                                  common source outbreak
Confirmed   A clinically compatible illness that is laboratory confirmed

4.13.2 Salmonellosis cases reported in 2006 by data source

The salmonellosis cases presented here exclude disease caused by *S.* Paratyphi and *S.* Typhi.

During 2006, 1 335 notifications (31.9 cases per 100 000 population) of salmonellosis were
reported in EpiSurv. The Enteric Reference Laboratory at ESR confirmed 1 343 *Salmonella*
isolates (32.1 cases per 100 000).

The ICD-10 code A02 was used to extract salmonellosis hospitalisation data from the NZHIS
NMDS database. Of the 161 hospital admissions (3.8 admissions per 100 000 population) recorded
in 2006, 122 were reported with salmonellosis as the primary diagnosis and 39 with salmonellosis
as another relevant diagnosis.

One death resulting from salmonellosis was recorded in EpiSurv in 2006.

It has been estimated by expert consultation that 61% (minimum = 45%, maximum = 69%) of
salmonellosis incidence is due to foodborne transmission. It was further estimated that 36% of
foodborne transmission was due to transmission via poultry.

4.13.3 Notifiable disease data

4.13.3.1 Annual notification trend

Since 1996 there has been a general annual increase in the number of salmonellosis notifications
with the highest number reported in 2001 (2 417 cases) (Figure 33). After 2001 the number of
notifications decreased to a low in 2004 (1 081 cases) before increasing slightly in more recent
years.

Figure 33: **Salmonellosis notifications and laboratory reported cases by year, 1996-2006**
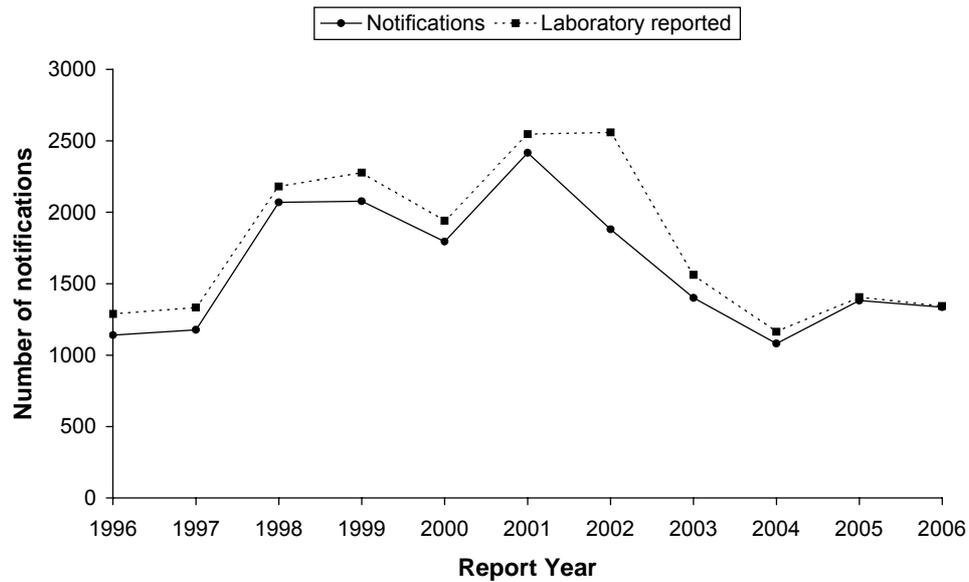
The 2006 salmonellosis notification rate was 31.9 per 100 000 population. Over the seven year period from 2000 to 2006 the salmonellosis annual notification rate was highest in 2001 before decreasing from 2002 to 2004 and levelling off after that.

Figure 34: **Salmonellosis notification rate by year, 2000-2006**
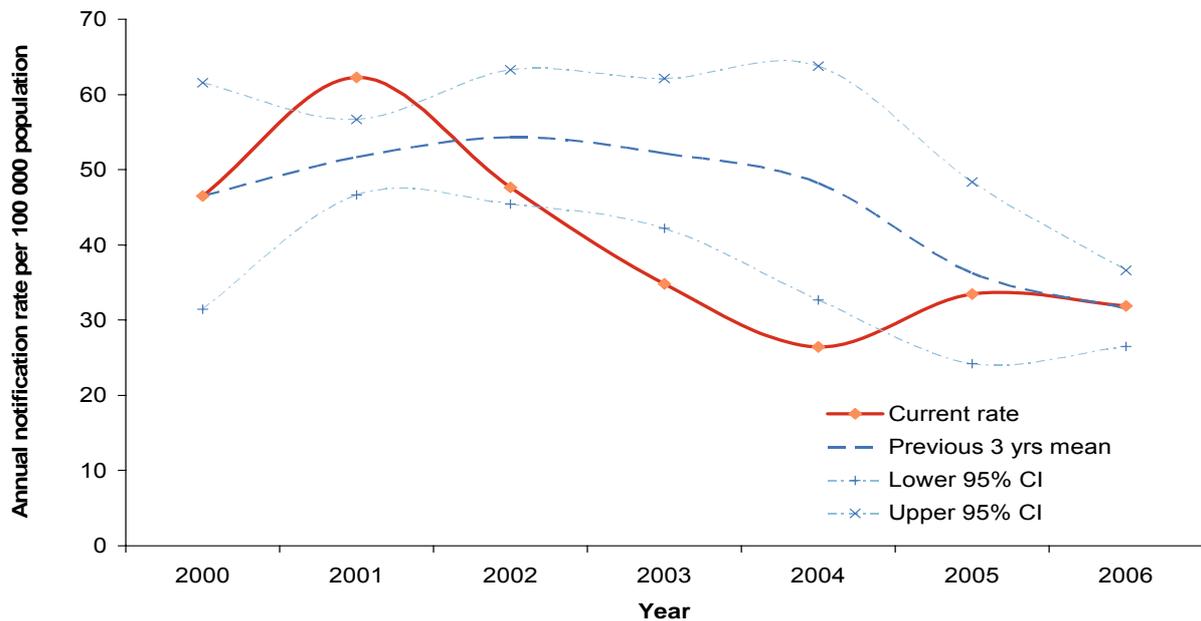
4.13.3.2 Seasonality

Salmonellosis notifications reported per 100 000 population by month for 2006 show a clear seasonal pattern with notification being highest during summer and autumn and lowest in mid winter (Figure 35). A similar trend is seen in the historic mean rate.

Figure 35:   Salmonellosis notification monthly rate (annualised) for 2006
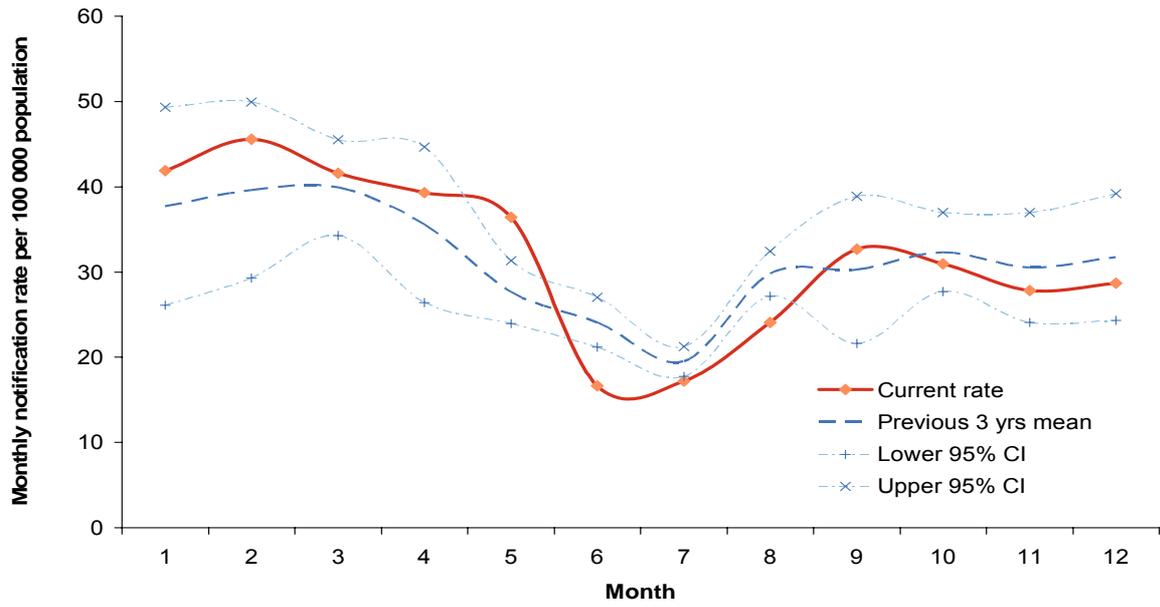
4.13.3.3 Geographic distribution of salmonellosis notifications

Rates vary throughout the country as illustrated in Figure 36. The highest salmonellosis notification rate during the four year period 2003 to 2006 was reported in South Canterbury during 2006 (67 per 100 000 population, 37 cases). South Canterbury and Southland DHB consistently feature in the quantile with the highest notification rates. Population rates are consistently low for the West Coast and MidCentral DHBs.

Figure 36:   Geographic distribution of salmonellosis notifications, 2003-2006
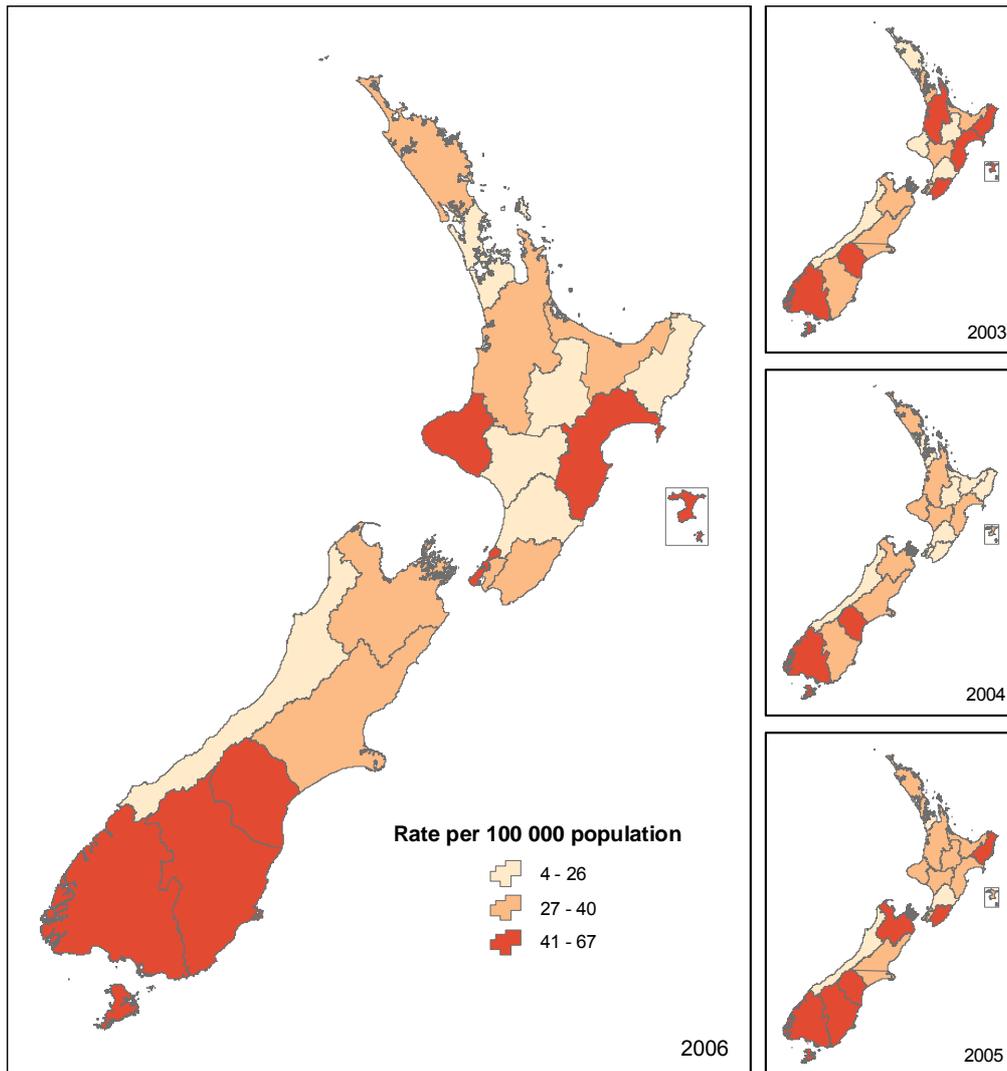
4.13.3.4 Age and sex distribution of salmonellosis cases

In 2006 the numbers and rates of notification and hospitalisation for salmonellosis was generally similar for males and females with slightly more males than females being reported in EpiSurv, and more females than males being hospitalised (Table 42).

Table 42:   **Salmonellosis cases by sex, 2006**

Sex	EpiSurv notifications		Hospitalisations[a]		Deaths recorded in EpiSurv
	No.	Rate	No.	Rate	No
Male	673	32.9	73	3.6	1
Female	639	29.9	88	4.1	0
Unknown	23	-	0	-	0
Total	**1 335**	**31.9**	**161**	**3.8**	**1**

[a] NZHIS morbidity data for hospital admissions

In 2006 age-specific salmonellosis rates are highest for those aged less than one for both the EpiSurv notifications (140.5 per 100 000) and NZHIS hospitalisations (27.1 per 100 000 population) (Table 43). One to four year olds also have a high salmonellosis rate compared to other age groups (123.4 per 100 000).

Table 43:   **Salmonellosis cases by age group, 2006**

Age groups	EpiSurv notifications		Hospitalisations[a]		Deaths recorded in EpiSurv
	No.	Rate	No.	Rate	No.
<1	83	140.5	16	27.1	
1 to 4	280	123.4	17	7.5	
5 to 9	93	31.9	12	4.1	
10 to 14	55	17.7	3	1.0	
15 to 19	67	21.3	8	2.6	
20 to 29	185	33.5	19	3.4	
30 to 39	132	22.1	7	1.2	
40 to 49	119	19.0	13	2.1	
50 to 59	143	28.2	14	2.8	
60 to 60	101	29.5	19	5.6	
70+	73	20.5	33	9.3	1
Unknown	4	-	0	-	
Total	**1 335**	**31.9**	**161**	**3.8**	**1**

[a] NZHIS Morbidity data for hospital admissions

4.13.3.5 Risk factors reported

The most commonly reported risk factor for salmonellosis notification cases during 2006 was consumption of food from retail premises (46.0%) followed by contact with farm animals (25.3%) and consumption of untreated water (20.2%) (Table 44).

Table 44:   Exposure to risk factors associated with salmonellosis, 2006

Risk Factor	Notifications			
	Yes	**No**	**Unknown**	**%**[a]
Consumed food from retail premises	312	366	657	46.0%
Contact with farm animals	218	645	472	25.3%
Consumed untreated water	149	589	597	20.2%
Travelled overseas during the incubation period	186	772	377	19.4%
Recreational water contact	134	670	531	16.7%
Contact with faecal matter	117	704	514	14.3%
Contact with other symptomatic people	107	709	519	13.1%
Contact with sick animals	37	750	548	4.7%
Contact with a confirmed case of same disease	35	742	558	4.5%

[1]Percentage refers to the cases that answered "yes" out of the total number of cases for which this information was supplied. Cases may have more than one risk factor recorded.

Between 2002 and 2006 the risk factors associated with salmonellosis cases have generally occurred in the same order of importance and to the same magnitude on a yearly basis (Figure 37). The consumption of food from retail premises has been the most commonly reported risk factor for salmonellosis cases every year and was considerably higher than contact with farm animals, the next most common risk factor.

Figure 37:   Salmonellosis risk factors by percentage of cases and year, 2002 – 2006
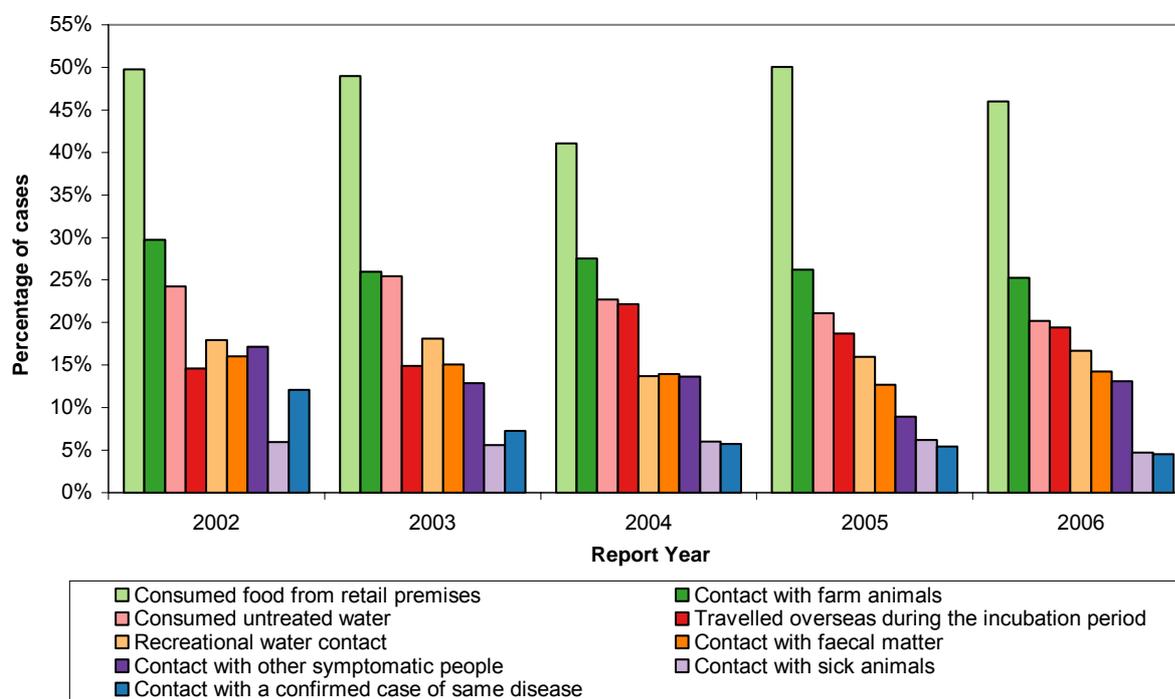
4.13.3.6 Estimate of travel-related cases

For cases where information on travel was provided, 19.4% (95%CI 16.6-22.2%) had travelled overseas during the incubation period. Assuming that the cases for which travel information was

provided were representative of all salmonellosis cases, a Poisson distribution can be used to estimate the total number of potentially travel related cases of salmonellosis in 2006. The resultant distribution has a mean of 259 cases (95% CI 211-309).

If data from the last four years are considered the estimated proportion of cases travelling overseas within the incubation period of the organism is 18.7% (95% CI 17.3-20.1%).

4.13.4 Outbreaks reported as caused by *Salmonella* spp

In 2006 there were 33 *Salmonella* spp. outbreaks reported and 9 of these were foodborne related (Table 45). All of the *Salmonella* spp. hospitalisations were associated with foodborne outbreaks.

Table 45:   ***Salmonella* spp. foodborne outbreaks reported, 2006**

Measure (No.)	Foodborne *Salmonella* spp. outbreaks	All *Salmonella* spp. outbreaks
Outbreaks	9	17
Cases	33	60
Hospitalised cases	6	6

The number of foodborne outbreaks reported between 2000 and 2006 ranged from nine to twenty, generally decreasing in number over time (Figure 38). The total numbers of cases associated with the outbreaks have also decreased over the period.

Figure 38:   **Foodborne *Salmonella* spp. outbreaks and associated cases reported by year, 2000–2006**
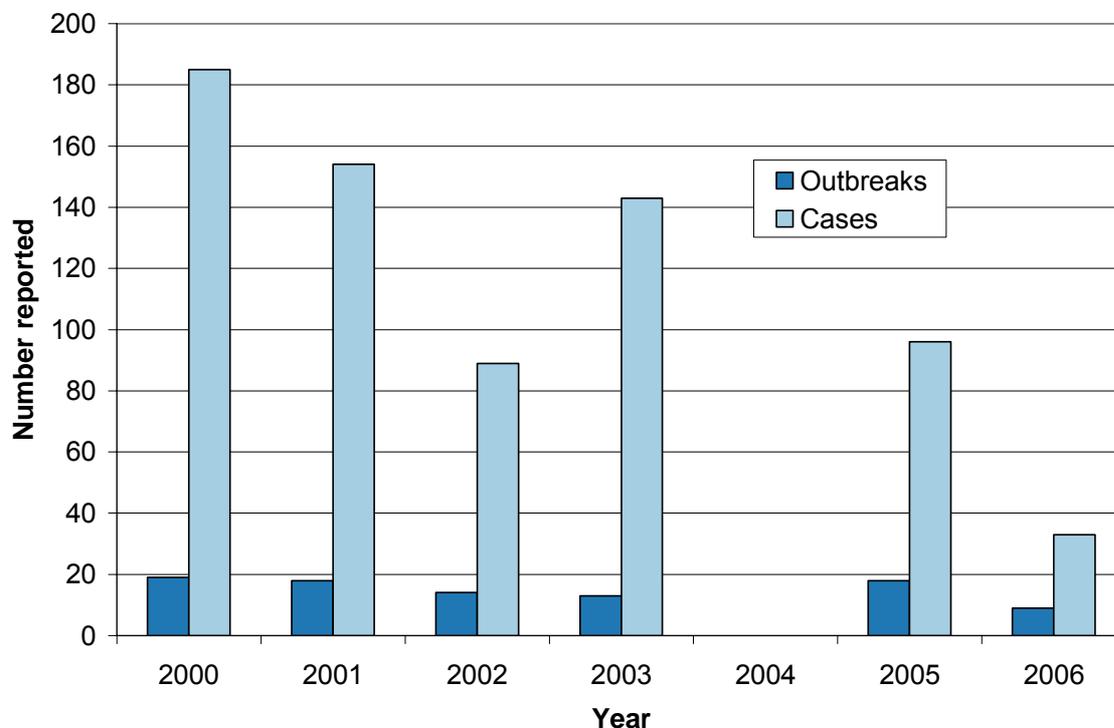
4.13.4.1 Details of food-associated outbreaks

Table 46 contains details of the nine food–associated *Salmonella* spp. outbreaks reported in 2006.

Table 46:   Details of food-associated *Salmonella* spp. outbreaks, 2006

Public Health Unit (Month)	Suspected vehicle	Setting	Number ill	Confirmation
Auckland (January)	Ham	Home, unknown	1C, 1P	None
Auckland (February)	Fish	Home	1C, 3P	2
Auckland (March)	Pizza	Takeaway	1C, 1P	2
Auckland (April)	Egg sandwiches	Restaurant/café	1C, 1P	1, 2
Auckland (October)	Unknown	Unknown	1C, 1P	None
Auckland (December)	Unknown	Home, unknown	1C, 1P	2
Nelson (January)	Beef lasagne	Restaurant/café	2C	1, 2, 7
Wellington (April)	Taro in coconut cream, BBQ lamb flaps, chop suey in coconut cream, taro and vermicelli, pork buns	Other food retail (markets)	15C	2
West Coast (February)	Unknown	Home	2C	None

C = confirmed, P = probable
Confirmation:
1 = Environmental investigation – identified critical control point failures linked to implicated source
2 = Epidemiological – case had history of exposure to implicated source
3 = Epidemiological – case control or cohort study showed elevated risk for cases to implicated source
4 = Laboratory – pathogen suspected to have caused illness identified in food handler
5 = Laboratory – pathogen suspected to have caused illness identified in implicated source (food)
6 = No evidence
7 = Other evidence

Evidence linking salmonellosis outbreaks to particular food vehicles was generally weak.

4.13.4.2 Laboratory investigation of samples from suspected foodborne outbreaks

During investigations of suspected foodborne illness outbreaks by ESR's Public Health Laboratories, no samples were found to be positive for *Salmonella* spp.

4.13.5 *Salmonella* types commonly reported

4.13.5.1 Human isolates

A total of 1 343 non-Typhi human isolates were typed by ESR's Enteric Reference Laboratory during 2006. Of these isolates, 733 (55%) were *Salmonella* Typhimurium.

Table 47 shows the number of cases of selected *Salmonella* types reported by the Enteric Reference Laboratory at ESR. The incidence of all *S.* Typhimurium definitive types (DT) was similar to 2005 numbers but not to the levels seen in 2003. DT160 remained the most common single type.

Table 47: Selected *Salmonella* serotypes and subtypes of laboratory-confirmed salmonellosis, 2003 – 2006

Subtype	2003	2004	2005	2006
S. Typhimurium	953	580	757	733
DT160	334	221	248	260
DT1	110	65	114	72
DT135	68	30	54	16
DT156	95	56	75	87
DT101	66	31	67	71
Other or unknown	280	177	199	227
S. Enteritidis	137	142	151	107
PT9a	65	50	73	53
PT4	22	24	9	9
Other or unknown	50	68	69	45
S. Infantis	89	63	67	58
S. Brandenburg	55	86	68	55
S. Saintpaul	27	33	65	35
S. Thompson	10	22	16	30
S. Montevideo	37	8	6	8
S. Heidelberg	11	3	7	14
Other or unknown serotypes	282	599	269	303
Total	**1 601**	**1 164**	**1 406**	**1 343**

4.13.5.2 Non-human isolates

A total of 1 407 non-human *Salmonella* isolates were typed by the Enteric Reference Laboratory during 2006 (Table 48).

Table 48: Selected *Salmonella* serotypes and subtypes from non-human sources, 2006

Subtype	2006	Major Sources
S. Typhimurium	543	
DT101	189	Miscellaneous poultry (102), Poultry environmental (56)
DT160	75	Equine (14), Avian (12), Feline (11)
DT1	40	Bovine (24), Poultry environmental (6)
DT42	36	Bovine (11), Poultry environmental (9)
Other or unknown	203	
S. Brandenburg	319	Ovine (230), Bovine (43)
S. Hindmarsh	162	Ovine (150)
S. Infantis	68	Meat/bone meal (19), Poultry environmental (13)
S. Agona	34	Poultry environmental (24)
S. Derby	30	Poultry feed (19)
Other or unknown serotypes	261	
Total	**1 417**	

4.13.5.3 Outbreak types

Table 49 shows the number of hospitalised cases and total cases by subtype for foodborne *Salmonella* outbreaks reported during 2006. *Salmonella* Typhimurium phage type 160 was associated with three outbreaks; each of the remaining six outbreaks was associated with a different subtype. The *Salmonella* Thompson outbreak was associated with the most cases (15) and was the only *Salmonella* outbreak where cases were hospitalised (6).

Table 49:   *Salmonella* subtypes reported in foodborne outbreaks, 2006

Pathogen and Subtype	Outbreaks	Hospitalised cases	Total cases
Salmonella Infantis	1	0	2
Salmonella Thompson	1	6	15
Salmonella Typhimurium phage type 101	1	0	2
Salmonella Typhimurium phage type 156	1	0	2
Salmonella Typhimurium phage type 160	3	0	6
Salmonella Weltevreden	1	0	4
Salmonella Weltevreden 15+	1	0	2

4.13.6 Recent surveys

Survey of *Salmonella* Contamination of Retail Eggs (Wilson, 2007)

This survey assessed the presence of *Salmonella* in and on eggs available through retail outlets in Auckland and Christchurch.

A total of 514 sample units of eggs were tested over a twelve-month period. Samples were retail packs of at least six eggs and were representative of the three production systems (cage, free range and barn). All samples were purchased and analysed within their stated shelf life.

Fifty different brands or sub-brands were identified. Twenty-eight of the sample units were in unlabelled cartons.

One egg from each sample unit was tested quantitatively for surface contamination and the remaining eggs from each retail pack were tested qualitatively for *Salmonella* species (3 710 eggs).

Salmonella was isolated from nine shell surface samples (overall prevalence 1.8%). All isolates were identified as *Salmonella* Infantis and all were from cage laid eggs (3.6% of cage laid eggs). Levels of *Salmonella* on eight of the samples were <5 MPN/egg and the other sample had a count of 44 MPN/egg. *Salmonella* positive samples were from four different brands and identified brands originated from three different farms.

No egg contents were positive for *Salmonella*.

Although the difference in prevalence between cage and free-range production was considered statistically significant, the number of barn egg samples was insufficient (and positive rate in cage eggs too low) to demonstrate a statistically significant difference between cage and barn production.

The results of this survey are consistent with two previous studies in indicating an absence of internal contamination of New Zealand eggs and enumeration tests have shown that the number of *Salmonella* present on the surface of contaminated eggs is low.

The pilot study suggests that, in New Zealand, the risk to consumers from *Salmonella* in eggs is low. Food handling practices which minimise the possibility of cross contamination from shells will further reduce the risk.

Salmonella in Uncooked Retail Meats in New Zealand (Wong *et al.*, 2007a)

A national quantitative survey of *Salmonella* in five types of uncooked retail meats was undertaken from August 2003 to May 2005 to establish baseline proportionality data. The overall prevalence of *Salmonella* in 1108 meat samples was 1.1% (95% Confidence Interval 0.6-1.9). Low prevalence of *Salmonella* in each meat type was observed, with 0.4% (0–2.4) in beef, 0.5% (0–3.0) in unweaned veal, 3% (1.2–6.1) in chicken, 1.3% (0.3–3.8) in lamb/mutton and 0% (0–1.6) in pork.

Salmonella serotypes isolated were *S*. Infantis from beef, *S*. Typhimuriun DT 1 from unweaned veal and chicken, *Salmonella* sp. 6,7:k:-, *S*. Enteritidis PT9a, *Salmonella* sp. 4,5,12:-:-, *Salmonella* sp. 4,12:-:- and *S*. Typhimurium DT160 from chicken, and *Salmonella* sp. 4:-:2 and *S*. Brandenburg from lamb. The three *Salmonella* sp. 4,5,12:-:- and *Salmonella* sp. 4,12:-:- isolates from chicken were very similar phenotypically and serologically to the attenuated *Salmonella* vaccine strain used in Megan[™]Vac1 for poultry.

One lamb sample yielded a count of 4.24 MPN/g of *S*. Brandenburg while all other positive samples were <1.0 MPN/g.

The results provide baseline proportionality data for *Salmonella* in retail uncooked meats that will contribute invaluably towards future risk assessment in combination with other information, such as consumption data.

Microbiological Survey of Imported and Domestic Pet Chews: *Salmonella* (Wong et al., 2007?-b)

The aims of this project were to survey the prevalence of *Salmonella* in imported and domestic pet chews to assess their:
  (1) Potential to introduce novel pathogenic and antimicrobial resistant strains into New Zealand, and
  (2) Role as vehicles of salmonellosis in the domestic home environment.

Three hundred samples each of imported and domestic pet chews were analysed qualitatively for *Salmonella*. Immuno-magnetic separation using Dynabeads[®] anti-*Salmonella* was used to concentrate *Salmonella* from pre-enriched culture followed by selective enrichment in Rappaport-Vassiliadis Soya Peptone broth. Selective plating on Xylose Lysine Desoxycholate and Hektoen Enteric agars resulted in 16 (5.3%) isolations of *Salmonella* from imported pet chews, and 20

(6.7%) isolations from domestic pet chews. The prevalence of *Salmonella* in imported and domestic products was not significantly different (P>0.05, Chi square test).

Salmonella Borreze isolated from Australian rawhide has never been recorded before in New Zealand. Three isolates of *S.* London from Australian pet chews were resistant to ampicillin and gentamicin, and two isolates of *S.* Infantis from Chinese pet chews were resistant to nalidixic acid, one of which was also resistant to streptomycin.

It was concluded that novel pathogenic and antimicrobial-resistant *Salmonella* are being introduced into New Zealand through the importation of pet chews, but the significance of this exposure pathway has yet to be determined. Pet chews are a potential source of exposure to *Salmonella* in the domestic home environment and humans are at risk of exposure either directly, through handling, or inadvertently by cross-contamination of food contact surfaces and food in the home environment.

4.13.7 Relevant New Zealand studies and publications

A study into the ongoing *Salmonella* Brandenburg epidemic was reported (Baker *et al.*, 2007). A case-control study found that human infection was significantly associated with occupational contact with sheep by the case or a close family member. There was no association between eating sheep meat in the three days prior to infection and the risk of infection. A number of food exposures were associated with a decreased risk of infection.

4.13.8 Relevant regulatory developments

Nil.

4.14 Shigellosis

Summary data for shigellosis in 2006 are given in Table 50.

Table 50:   Summary surveillance data for shigellosis, 2006

Parameter	Value in 2006	Section reference
Number of cases	102	4.14.2
Rate (per 100 000)	2.4	4.14.2
Hospitalisations (%)	15 (14.7%)	4.14.2
Deaths (%)	Nil	4.14.2
Estimated travel-related cases (%)	62 (60.5%)	4.14.3.6
Estimated food-related cases (%)	NA	

NA = not applicable, no information is available on the food attributable proportion of shigellosis in New Zealand

4.14.1 <u>Case definition</u>

Clinical description: Shigellosis presents as gastroenteritis

Laboratory test for diagnosis: Isolation of Shigella spp. from a clinical specimen

Case classification:
Probable   A clinically compatible illness that is either a contact of a confirmed case of the same disease, or has had contact with the same common source i.e., is part of an identified common source outbreak
Confirmed   A clinically compatible illness that is laboratory confirmed

4.14.2 <u>Shigellosis cases reported in 2006 by data source</u>

During 2006 102 notifications (2.4 cases per 100 000 population) of shigellosis were reported in EpiSurv. The Enteric Reference Laboratory at ESR confirmed 96 *Shigella* isolates (2.3 per 100 000 population).

The ICD-10 code A03 was used to extract shigellosis hospitalisation data from the NZHIS NMDS database. Of the 15 hospital admissions (0.4 admissions per 100 000 population) recorded in 2006, 13 were reported with shigellosis as the primary diagnosis and two with shigellosis as another relevant diagnosis.

No deaths resulting from shigellosis were recorded in EpiSurv in 2006

4.14.3 <u>Notifiable disease data</u>

4.14.3.1 Annual notification trend

The number of notifications and laboratory reported cases of shigellosis fluctuates each year (Figure 39).

Figure 39:   Shigellosis notifications and laboratory reported cases by year, 1996-2006
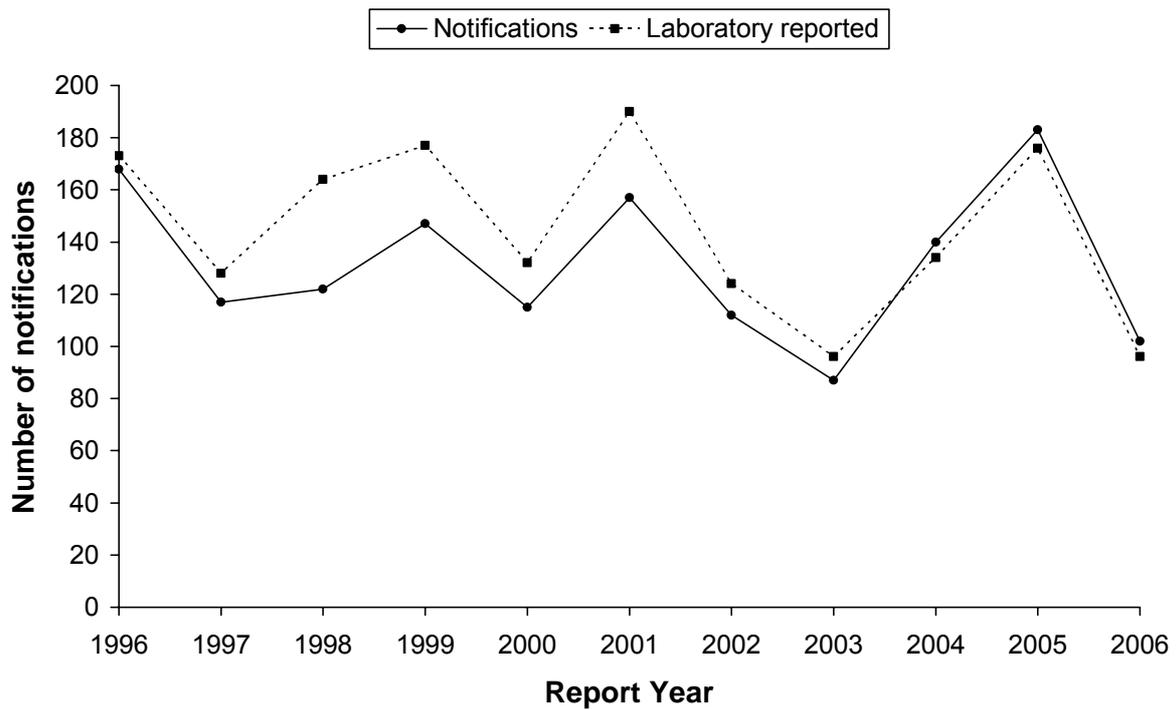
The 2006 shigellosis notification rate was 2.4 per 100 000 population. This is a decrease from the previous year and was one of the lowest rates since 2000 (Figure 40).

Figure 40:   Shigellosis notification rate by year, 2000-2006
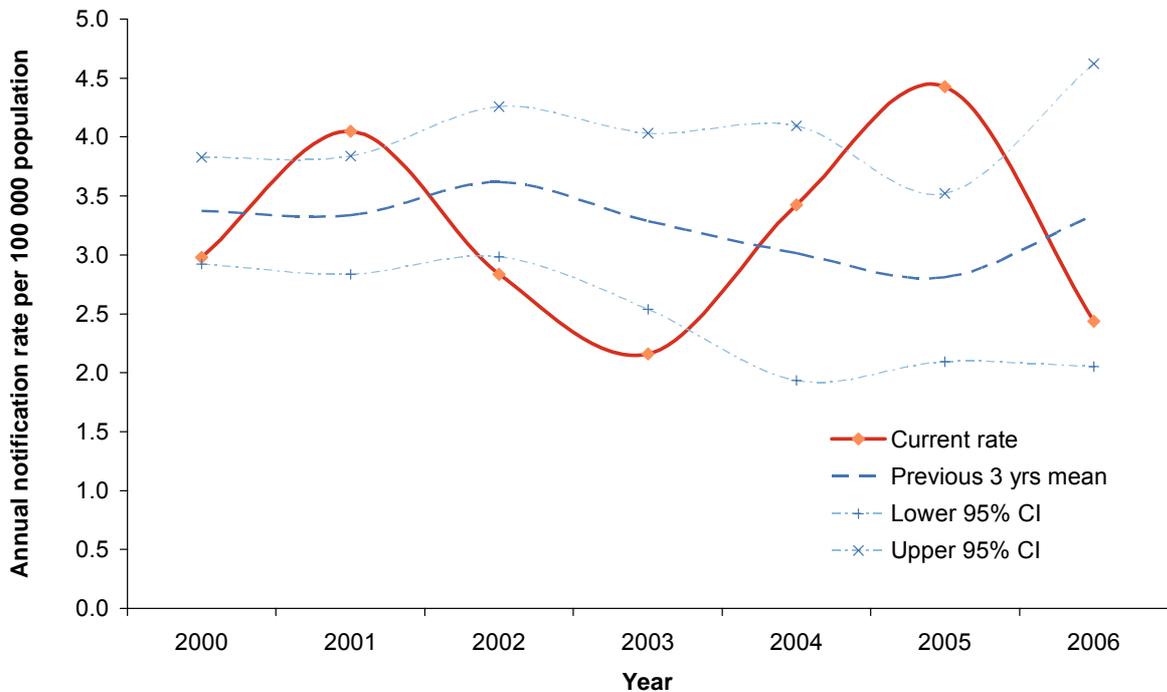
4.14.3.2 Seasonality

The number of notified cases of shigellosis per 100 000 population by month for 2006 is shown in Figure 41. In 2006 shigellosis notifications were highest in January and February. There is a peak in the historical mean in November due to a large shigellosis outbreak in Northland and Auckland in 2005.

Figure 41:   Shigellosis monthly rate (annualised) for 2006
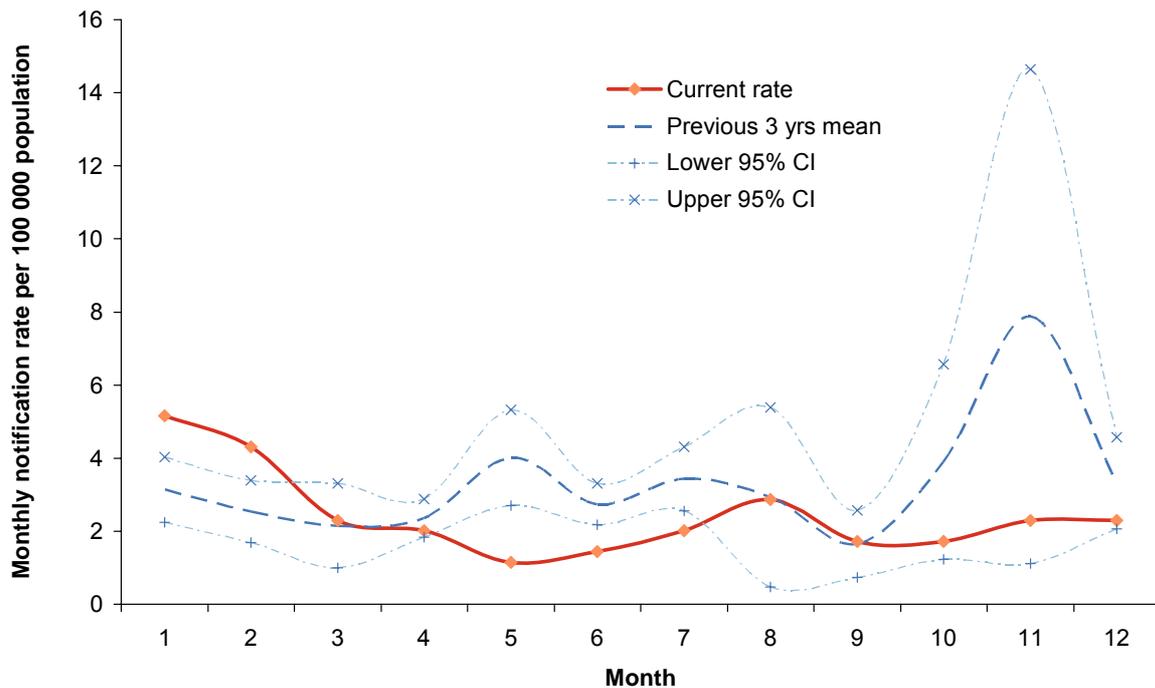
4.14.3.3 Age and sex distribution of shigellosis cases

The number and rates of notifications and hospitalisations for shigellosis were generally similar for males and females (Table 51).

Table 51:   Shigellosis cases by sex, 2006

Sex	EpiSurv notifications		Hospitalisations[a]		Deaths recorded in EpiSurv
	No.	Rate	No.	Rate	No
Male	46	2.2	8	0.4	
Female	52	2.4	7	0.3	
Unknown	4				
Total	**102**	**2.4**	**15**	**0.4**	

[a] NZHIS Morbidity data for hospital admissions

Age-specific shigellosis notification rates were highest for those aged between 1 and 4 years. This was consistent for the EpiSurv notifications (145.3 per 100 000) and NZHIS hospitalisations (27.1

per 100 000 population) (Table 52). One to four year olds also had a high shigellosis rate compared to all other age groups. Notification and hospitalisation rates were lowest for those aged 10 to 14 years and 30 to 49 years.

Table 52:   Shigellosis cases by age group, 2006

Age groups	EpiSurv notifications		Hospitalisations[a]		Deaths recorded in EpiSurv
	No.	Rate	No.	Rate	No.
<1	0	-	0	0.0	
1 to 4	15	6.6	4	1.8	
5 to 9	10	3.4	2	0.7	
10 to 14	2	0.6	1	0.3	
15 to 19	3	1.0	0	0.0	
20 to 29	18	3.3	1	0.2	
30 to 39	20	3.3	2	0.3	
40 to 49	13	2.1	0	0.0	
50 to 59	10	2.0	4	0.8	
60 to 60	7	2.0	0	0.0	
70+	3	0.8	1	0.3	
Unknown	1	-			
Total	**102**	**2.4**	**15**	**0.4**	**-**

[a] NZHIS Morbidity data for hospital admissions

4.14.3.4 Risk factors reported

The most commonly reported risk factor for shigellosis in 2006 was overseas travel during the incubation period (reported by 60.5% of cases) followed by consumption of food from retail premises (48.9%) and recreational water contact (22.2%) (Table 53).

Table 53:   Exposure to risk factors associated with shigellosis, 2006

	Notifications			
Risk Factor	**Yes**	**No**	**Unknown**	**%[a]**
Travelled overseas during the incubation period	49	32	21	60.5%
Consumed food from retail premises	23	24	55	48.9%
Recreational water contact	10	35	57	22.2%
Contact with other symptomatic people	11	46	45	19.3%
Contact with a confirmed case of same disease	8	49	45	14.%
Consumed untreated water	6	39	57	13.3%
Contact with faecal matter	3	49	50	5.8%
Contact with farm animals	2	43	57	4.4%
Contact with sick animals	0	45	57	0.%

[a]Percentage refers to the cases that answered "yes" out of the total number of cases for which this information was supplied. Cases may have more than one risk factor recorded.

With the exception of 2004, overseas travel during the incubation period and consumption of food from retail premises were the two most commonly reported risk factors for shigellosis during the five year period 2002 to 2006 (Figure 42).

Figure 42: **Shigellosis risk factors by percentage of cases and year, 2002 – 2006**
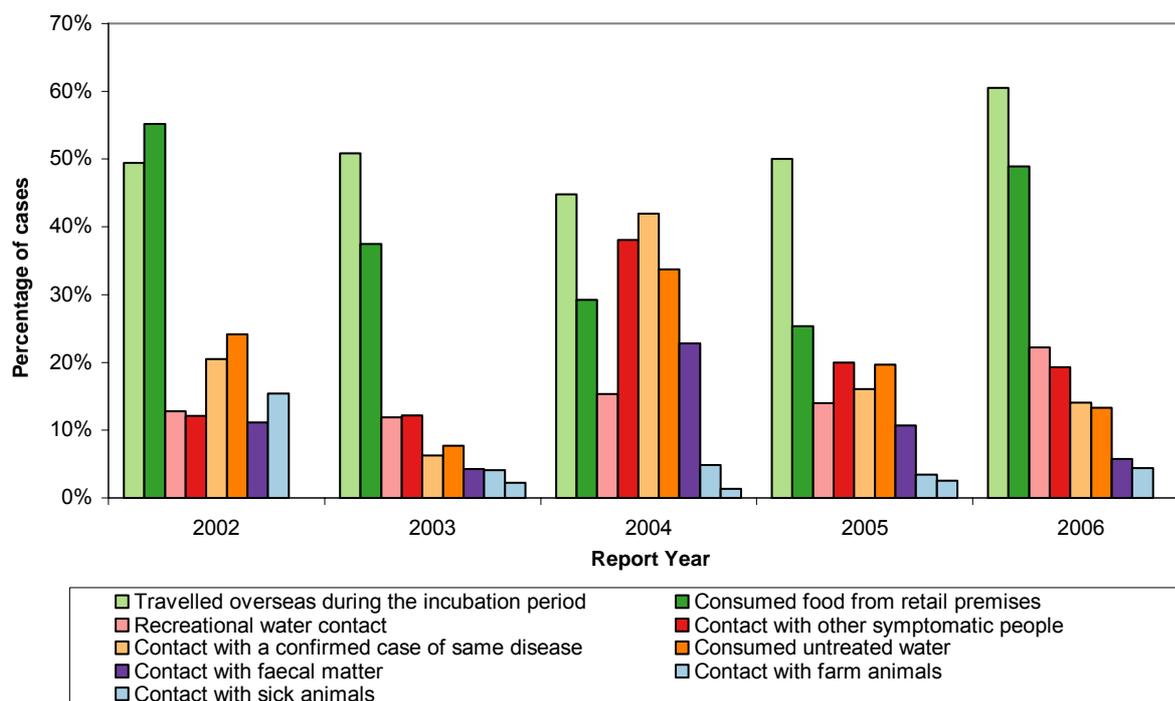
4.14.3.5 Estimate of travel-related cases

For cases where information on travel was provided, 60.5% (95%CI 44.8-78.6%) had travelled overseas during the incubation period. Assuming that the cases for which travel information was provided were representative of all shigellosis cases, a Poisson distribution can be used to estimate the total number of potentially travel related cases of shigellosis in 2006. The resultant distribution has a mean of 62 cases (95% CI 41-87).

If data from the last four years are considered the estimated proportion of cases travelling overseas within the incubation period of the organism is 51.4% (95% CI 44.3-59.0%).

4.14.4 Outbreaks reported as caused by *Shigella* spp

Two of the eight *Shigella* outbreaks reported in EpiSurv in 2006 were foodborne (Table 54).

Table 54: *Shigella* **spp. outbreaks reported, 2006**

Measure (No.)	Foodborne *Shigella* spp. outbreaks	All *Shigella* spp. outbreaks
Outbreaks	2	8
Cases	10	27
Hospitalised cases	0	3

Foodborne shigellosis outbreaks are rare with not more than two outbreaks being reported each year from 2000 to 2006 (Figure 43).

Figure 43: Foodborne *Shigella* spp. outbreaks and associated cases reported by year, 2000 – 2006
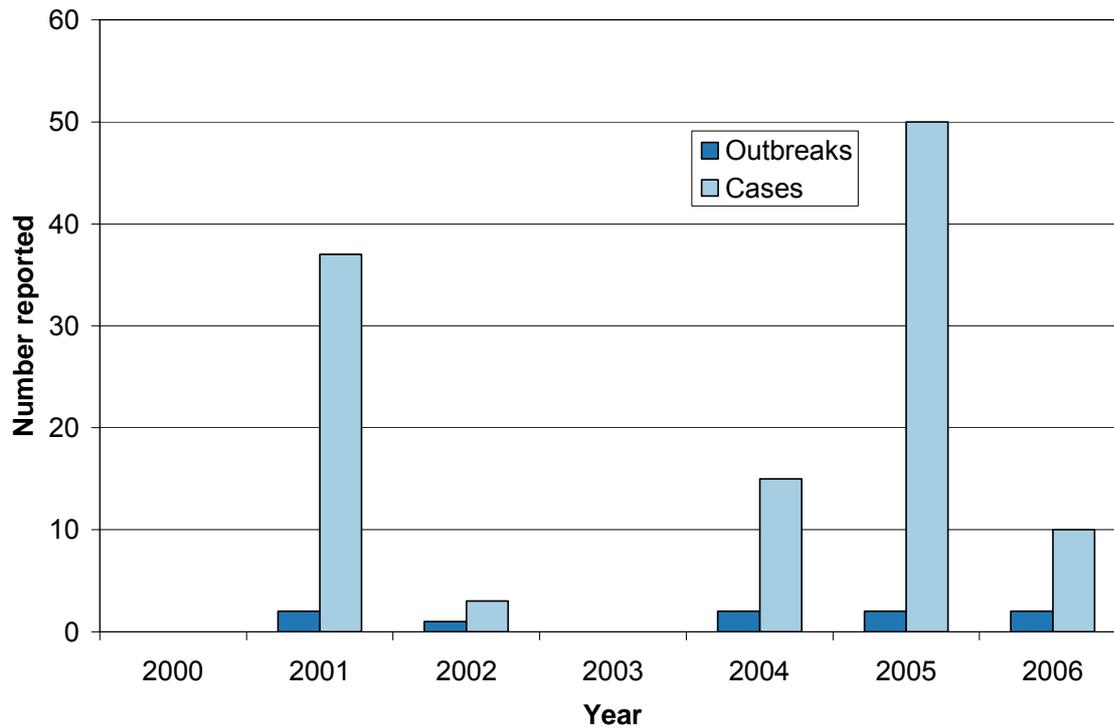
4.14.4.1 Details of food-associated outbreaks

Table 55 contains details of the two food–associated *Shigella* spp. outbreaks reported in 2006

Table 55: Details of food-associated *Shigella* spp. outbreaks, 2006

Public Health Unit (Month)	Suspected vehicle	Setting	Number ill	Confirmation
Auckland (February)	Raw fish	Home	3C	2
Nelson (September)	Ham or chicken filled rolls	Other food retail (bakery)	4C, 3P	2

C = confirmed, P = probable
Confirmation:
1 = Environmental investigation – identified critical control point failures linked to implicated source
2 = Epidemiological – case had history of exposure to implicated source
3 = Epidemiological – case control or cohort study showed elevated risk for cases to implicated source
4 = Laboratory – pathogen suspected to have caused illness identified in food handler
5 = Laboratory – pathogen suspected to have caused illness identified in implicated source (food)
6 = No evidence
7 = Other evidence

Evidence confirming the suspect food as the source of the outbreak was generally weak.

4.14.4.2 Laboratory investigation of samples from suspected foodborne outbreaks

During investigations of suspected foodborne illness outbreaks by ESR's Public Health Laboratories, no samples were found to contain *Shigella* spp.

4.14.5 *Shigella* types commonly reported

There were 96 isolates of *Shigella* confirmed in 2006, compared with 176 in 2005. *S. sonnei* biotypes accounted for 49 of the isolates, while *S. flexneri* accounted for a further 40 isolates.

Table 56 summarises typing information for outbreaks that occurred during 2006.

Table 56:   Pathogen subtypes reported in foodborne *Shigella* spp. outbreaks, 2006

Pathogen and Subtype	Outbreaks	Hospitalised cases	Total cases
Shigella flexneri 2b	1	0	7
Shigella sonnei Biotype a	1	0	3

4.14.6 Relevant New Zealand studies and publications

Nil.

4.14.7 Relevant regulatory developments

Nil.

4.15   *Staphylococcus aureus* Intoxication

4.15.1 Case definition

Clinical description: Gastroenteritis with sudden severe nausea and vomiting

Laboratory test for diagnosis: Detection of enterotoxin in faecal or vomit specimen or in leftover food or isolation of $\geq 10^3$/gram coagulase-positive *S. aureus* from faecal or vomit specimen or $\geq 10^5$ from leftover food

Case classification:
Probable   A clinically compatible illness that is either a contact of a confirmed case of the same disease, or has had contact with the same common source i.e., is part of an identified common source outbreak
Confirmed   A clinically compatible illness that is laboratory confirmed

4.15.2 *Staphylococcus aureus* intoxication cases reported in 2006 by data source

In 2006 five notifications of *Staphylococcus aureus* were reported in EpiSurv with no resulting deaths.

The ICD-10 code A05.0 was used to extract foodborne staphylococcal intoxication hospitalisation data from the NZHIS NMDS database. Of the two hospital admissions recorded in 2006, one was reported with foodborne staphylococcal intoxication as the primary diagnosis and one with this condition as another relevant diagnosis.

4.15.3 Outbreaks reported as caused by *Staphylococcus aureus*

Between 2000 and 2003 there was a steady decrease in the number of *Staphylococcus aureus* outbreaks reported (Figure 44) followed by a small increase in 2004 and 2005. In 2006 no *Staphylococcus aureus* outbreaks were reported in EpiSurv.

Figure 44: **Foodborne *Staphylococcus aureus* outbreaks and associated cases reported by year, 2000 – 2006**
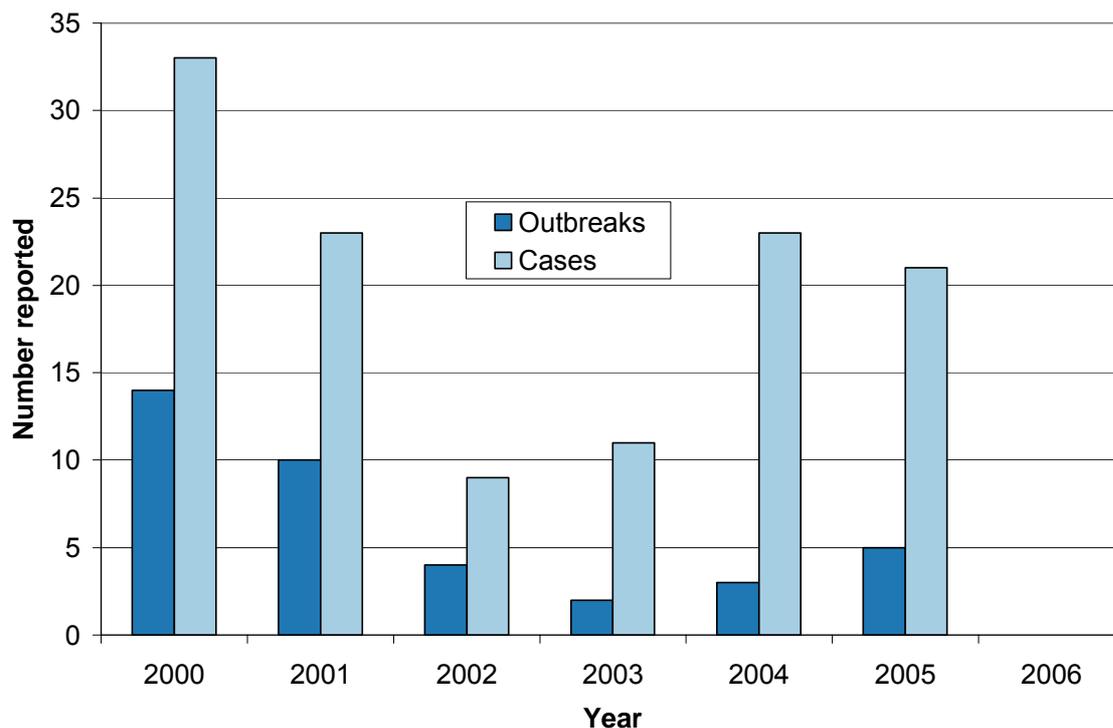
4.15.3.1 Details of food-associated outbreaks

In 2006, no *Staphylococcus aureus* outbreaks were reported in EpiSurv.

4.15.3.2 Laboratory investigation of samples from suspected foodborne outbreaks

During investigations of suspected foodborne illness outbreaks by ESR's Public Health Laboratories, eight investigations revealed evidence of *S. aureus* intoxication. These included six investigations where elevated levels of *S. aureus* and/or the associated enterotoxin were found in faecal specimens and two investigations where the toxin was detected in an implicated food (battered fish, Chinese meal). Other implicated foods were seafood salad, salad roll, and hot dog.

4.15.4 Relevant New Zealand studies and publications

Nil.

4.15.5 Relevant regulatory developments

Nil.

4.16   Toxic Shellfish Poisoning

4.16.1 Case definition

Due to the diverse nature of toxins that may cause toxic shellfish poisoning, no consistent clinical description is provided for this condition. Depending on the toxin involved toxic shellfish poisoning may results in various combinations of gastrointestinal, neurosensory, neurocerebellar/neuromotor, general neurological and other symptoms. Case definitions for suspected cases of toxic shellfish poisoning are:

Amnesic Shellfish Poisoning (ASP): Vomiting or diarrhoea or abdominal cramps occurring within 24 hours of consuming shellfish AND no other probable cause identified by microbiological examination of faecal specimen from the case or microbiological testing of leftover food AND/OR one or more of the neurological symptoms from group C occurring within 48 hours of consuming shellfish.

Diarrhoeic Shellfish Poisoning (DSP): Vomiting or diarrhoea occurring within 24 hours of consuming shellfish AND no other probable cause identified by microbiological examination of faecal specimen from the case or microbiological testing of leftover food.

Neurotoxic Shellfish Poisoning (NSP): Two or more of the neurological symptoms from groups A and B occurring within 24 hours of consuming shellfish.

Paralytic Shellfish Poisoning (PSP): Paraesthesia occurring within 12 hours of consuming shellfish AND one of the neurological symptoms from group B.

Toxic Shellfish Poisoning (TSP) type unspecified: Vomiting or diarrhoea occurring within 24 hours of consuming shellfish AND no other probable cause identified by microbiological examination of faecal specimen from the case or microbiological testing of leftover food OR any of the neurological symptoms from groups A and B occurring within 24 hours of consuming shellfish OR one or more of the neurological signs/symptoms from group C occurring within 48 hours of consuming shellfish.

Case definitions for probable cases of toxic shellfish poisoning are:

Meets case definition for suspect case AND detection of relevant biotoxin at or above the regulatory limit in shellfish obtained from near or same site (not leftovers) within seven days of collection of shellfish consumed by case.
Current level:
ASP: 20 ppm domoic acid/100 g shellfish
DSP: 20 µg/100 g or 5 MU/100 g shellfish (MU = mouse units)
NSP: 20 MU/100 g shellfish
PSP: 80 µg/100 g shellfish

Case definitions for confirmed cases of toxic shellfish poisoning are:

Meets case definition for suspect case AND detection of TSP biotoxin in leftover shellfish at a level resulting in the case consuming a dose likely to cause illness.

Current dose level:
ASP: 0.05 mg/kg body weight
DSP: ingestion of 48 µg or 12 MU
NSP: 0.3 MU/kg body weight

PSP: 10 MU/kg body weight ($\cong$ 2µg/kg body weight)

Clinical symptoms for assigning status:
Group A:
• paraesthesia - i.e. numbness or tingling around the mouth, face or extremities
• alteration of temperature sensation

Group B:
• weakness such as trouble rising from seat or bed
• difficulty swallowing
• difficulty breathing
• paralysis
• clumsiness
• unsteady walking
• dizziness/vertigo
• slurred/unclear speech
• double vision

Group C:
• confusion
• memory loss
• disorientation
• seizure
• coma

4.16.2 Toxic shellfish poisoning cases reported in 2006

There was one case of toxic shellfish poisoning reported in EpiSurv in 2006. This continues the low number of toxic shellfish poisoning notifications in recent years. The poisoning occurred after the consumption of steamed mussels. Diarrhoeic shellfish poisoning toxins were detected.

The ICD-10 code T612 was used to extract hospitalisation data for 'other fish and shellfish poisoning' from the NZHIS NMDS database. Of the 21 hospital admissions recorded in 2006, 17 were reported with 'other fish and shellfish poisoning' as the primary diagnosis and four with this condition as another relevant diagnosis. Note that this ICD-10 code includes shellfish and other fish.

4.16.3 Outbreaks reported as caused by TSP

In 2006 there were no outbreaks due to toxic shellfish poisoning reported in EpiSurv.

4.17 VTEC/STEC Infection

Summary data for VTEC/STEC infection in 2006 are given in Table 57.

Table 57:   Summary surveillance data for VTEC/STEC infection, 2006

Parameter	Value in 2006	Section reference
Number of cases	87	4.17.2
Rate (per 100 000)	2.1	4.17.2
Hospitalisations (%)	7 (8.0%)	4.17.2
Deaths (%)	Nil	4.17.2
Estimated travel-related cases (%)	2 (2.7%)	4.17.3.5
Estimated food-related cases (%)*	34 (40%)	4.17.2

* For estimation of food-related cases it was assumed that the proportions derived from expert consultation would exclude travel-related cases

4.17.1 Case definition

Clinical description:

An illness of variable severity characterised by diarrhoea (often bloody) and abdominal cramps. Illness may be complicated by haemolytic uraemic syndrome (HUS), or thrombotic thrombocytopaenic purpura (TTP)

Laboratory test for diagnosis:

Isolation of Shiga toxin (verotoxin) producing *Escherichia coli* OR detection of the genes associated with the production of Shiga toxin in *E. coli*

Case classification:

Probable

A clinically compatible illness that is either a contact of a confirmed case of the same disease, or has had contact with the same common source i.e., is part of an identified common source outbreak

Confirmed

A clinically compatible illness that is laboratory confirmed

4.17.2 VTEC/STEC infection cases reported in 2006 by data source

During 2006, 87 notifications (2.1 cases per 100 000 population) of VTEC/STEC infection were reported in EpiSurv. The Enteric Reference Laboratory received 86 isolates (2.1 per 100 000).

The ICD-10 code A043 was used to extract enterohaemorrhagic *Escherichia coli* infection hospitalisation data from the NZHIS NMDS database. Of the 7 hospital admissions recorded in 2006, five were reported with enterohaemorrhagic *Escherichia coli* infection as the primary diagnosis and two with this condition as another relevant diagnosis.

No deaths due to VTEC/STEC infection were recorded in EpiSurv in 2006.

It has been estimated by expert consultation that 40% (minimum = 27%, maximum = 51%) of VTEC/STEC incidence is due to foodborne transmission. The expert consultation also estimated that approximately 30% of foodborne VTEC/STEC transmission was due to red meat of which two-thirds was considered to be due to consumption of uncooked, fermented, comminuted meat.

4.17.3 Notifiable disease data

4.17.3.1 Annual notification trend

The 2006 VTEC/STEC infection notification rate was 2.1 per 100 000 population. As shown in Figure 45, there has been a general increase in the notifications with the highest number of notifications reported in 2003 (104 cases).

Figure 45:   VTEC/STEC infection notifications by year, 1996-2006
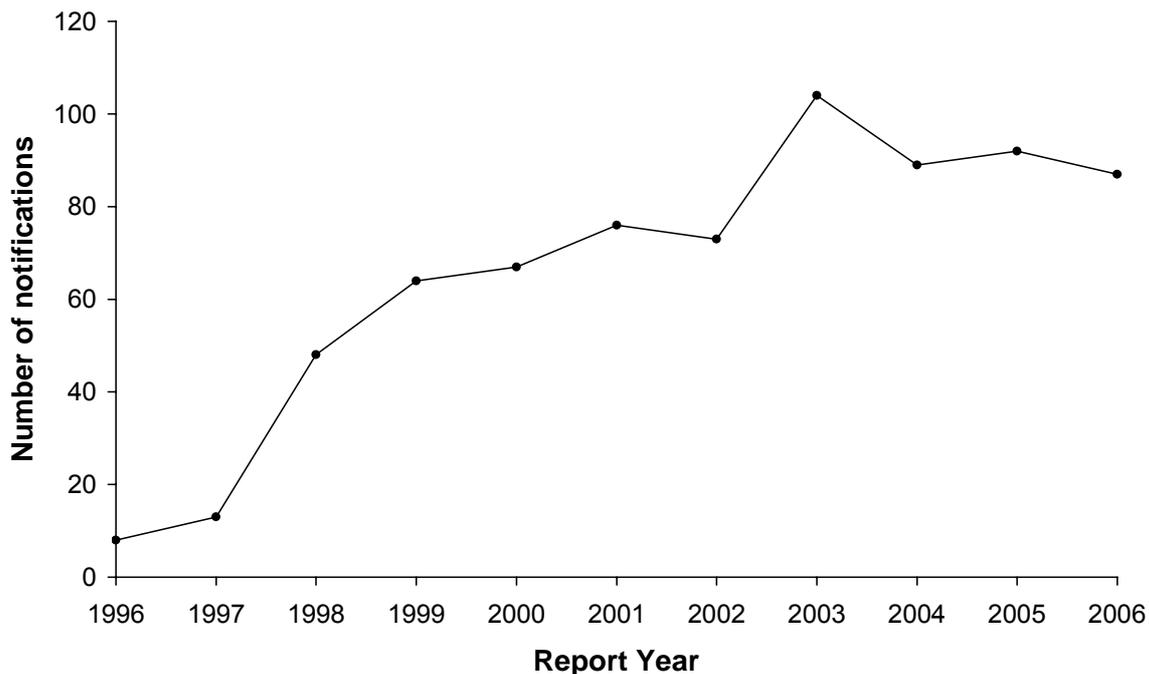
Over the period 2000 to 2006 The VTEC/STEC infection notification rates has varied little with the highest population rate being reported in 2003 (2.6 cases per 100 000 population) (Figure 46).

Figure 46:   VTEC/STEC infection notification rate by year, 2000-2006
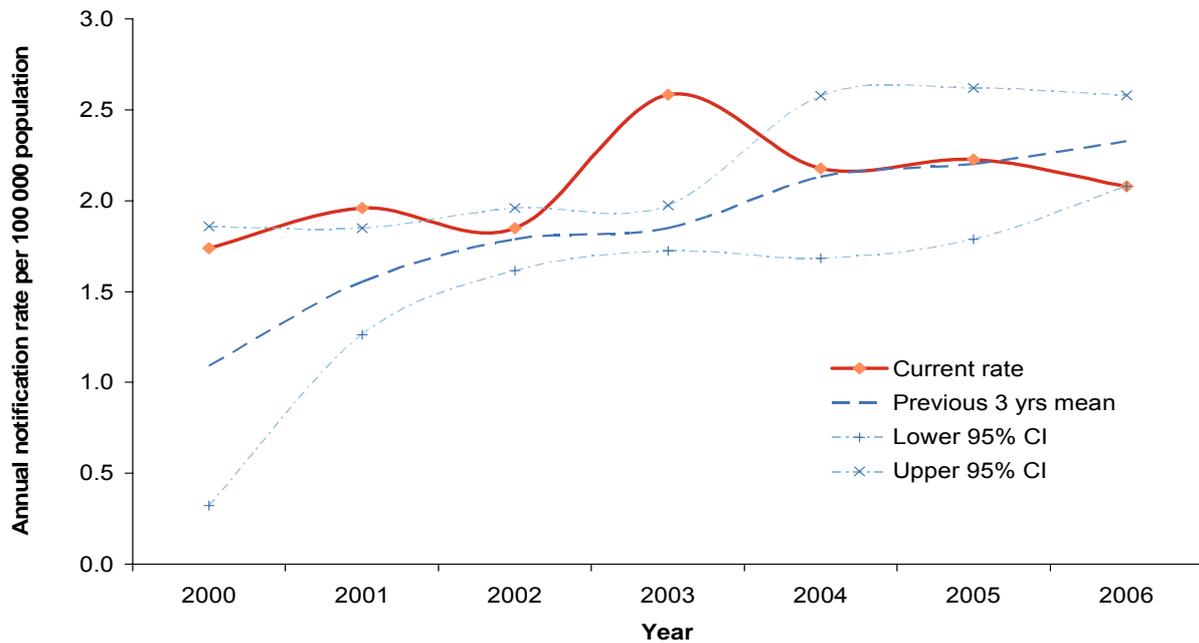
4.17.3.2 Seasonality

The number of notified cases of VTEC/STEC infection per 100 000 population by month for 2006 is shown in Figure 47. The notification rate varied each month with a peak in March while the mean historic rate peaked a month later in April.

Figure 47:   VTEC/STEC infection notification monthly rate (annualised) for 2006
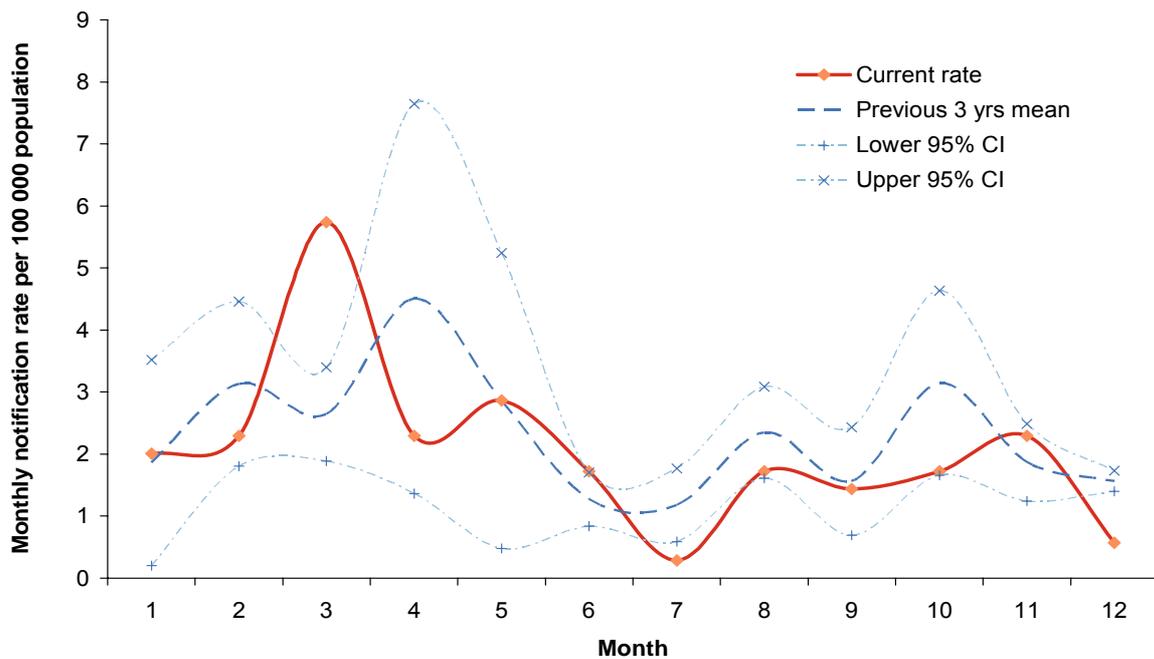
4.17.3.3 Age and sex distribution of VTEC/STEC infection

In 2006 the number and notification rate for VTEC/STEC infection was similar between males and females but hospitalisation was higher in males than females (Table 58).

Table 58:   VTEC/STEC infection by sex, 2006

Sex	EpiSurv notifications		Hospitalisations[a]		Deaths recorded in EpiSurv
	No.	Rate	No.	Rate	No
Male	44	2.1	6	0.3	
Female	42	2.0	1	0.05	
Unknown	1				
Total	**87**	**2.1**	**7**	**0.2**	**-**

[a] NZHIS morbidity data for hospital admissions

In 2006 the age specific notification VTEC/STEC infection rates were highest in the 1 to 4 years age (38 cases, 16.7 per 100 000 population), followed by the less than one year age group (6 cases, 10.2 per 100 000). The 1 to 4 years age group also had the highest hospitalisation rates (Table 59).

Table 59:   VTEC/STEC infection by age group, 2006

Age groups	EpiSurv notifications		Hospitalisations[a]		Deaths recorded in EpiSurv
	No.	Rate	No.	Rate	No.
<1	6	10.2	0	0.0	
1 to 4	38	16.7	2	0.9	
5 to 9	5	1.7	1	0.3	
10 to 14	3	1.0	0	0.0	
15 to 19	1	0.3	0	0.0	
20 to 29	7	1.3	1	0.2	
30 to 39	4	0.7	1	0.2	
40 to 49	5	0.8	1	0.2	
50 to 59	3	0.6	0	0.0	
60 to 60	8	2.3	0	0.0	
70+	5	1.4	1	0.3	
Unknown	**2**				
Total	**87**	**2.1**	**7**	**0.2**	**-**

[a] NZHIS morbidity data for hospital admissions

4.17.3.4 Risk factors reported

It should be noted that each disease has its own investigation module, and the identification of a large number of risk factors for VTEC/STEC infection is a reflection of the content of the investigation module, rather than a characteristic of the disease in New Zealand.

In 2006 the most commonly reported risk factor for VTEC/STEC infection was consumption of raw fruit/vegetables (92.3%), followed by contact with household pets (87.5%), consumption of poultry products (83.3%), and consumption of dairy products (81.8%) (Table 60).

Table 60:   Exposure to risk factors associated with VTEC/STEC infection, 2006

Risk Factor	Notifications			
	Yes	**No**	**Unknown**	**%**[a]
Consumed raw fruit/vegetables	60	5	22	92.3%
Contact with household pets	56	8	23	87.5%
Consumed poultry products	55	11	21	83.3%
Consumed dairy products	54	12	21	81.8%
Consumed beef products	45	19	23	70.3%
Consumed processed meats	44	21	22	67.7%
Consumed fruit/vegetables juice	35	23	29	60.3%
Contact with farm animals	29	30	28	49.2%
Contact with animal manure	27	28	32	49.1%
Contact with children in nappies	28	37	22	43.1%
Contact with persons with similar symptoms	23	35	29	39.7%
Recreational water contact	19	48	20	28.4%
Consumed lamb products	14	49	24	22.2%
Contact with other animals	12	44	31	21.4%
Consumed home killed meats	9	57	21	13.6%
Consumed raw milk or products from raw milk	5	55	27	8.3%
Consumed pink or undercooked meats	3	59	25	4.8%
Travelled overseas during the incubation period	2	71	14	2.7%

[a]Percentage refers to the cases that answered "yes" out of the total number of cases for which this information was supplied. Cases may have more than one risk factor recorded.

Figure 48:   **VTEC/STEC infection foodborne risk factors by percentage of cases and year, 2002 – 2006**
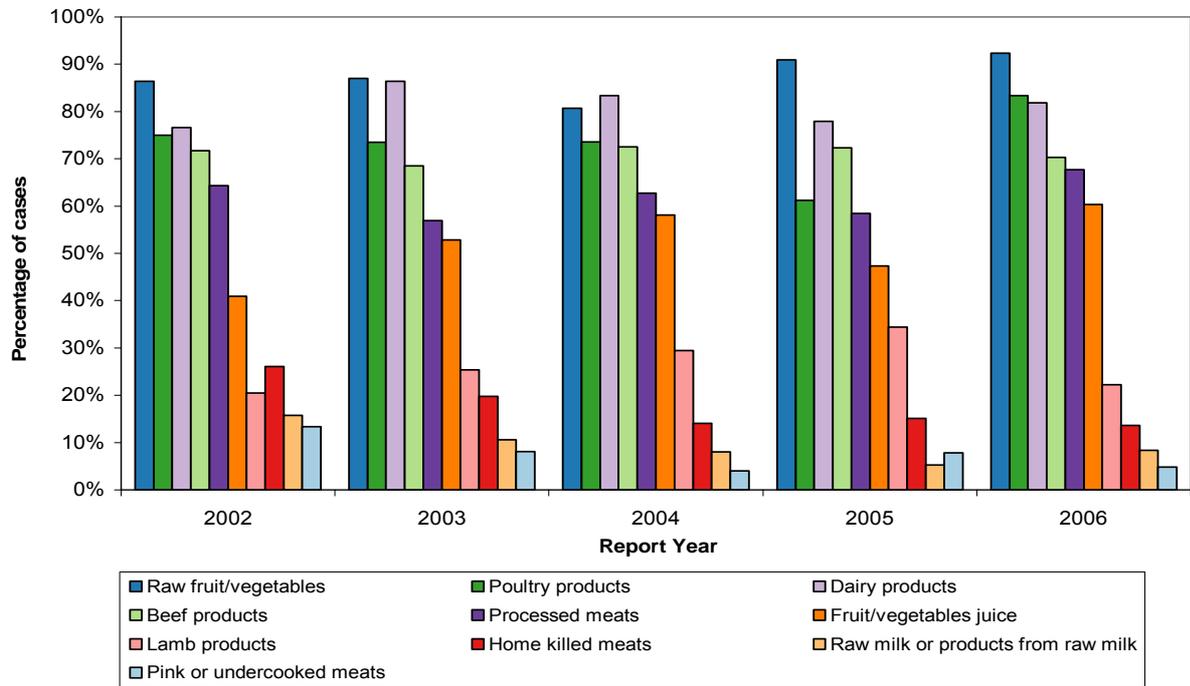
The two most consistently reported risk factors for VTEC/STEC infection over the five year period 2002 to 2006 were the consumption of raw fruit/vegetables (Figure 48) and contact with household pets (Figure 49). The reporting of contact with animal manure, consumption of home killed meats, raw milk or milk products, and undercooked meats as risk factors have generally decreased since 2002. Contact with other symptomatic people was more frequently reported in 2006 than in any other recent year.

Figure 49:   VTEC/STEC infection risk factors excluding food consumption by percentage of cases and year, 2002 - 2006
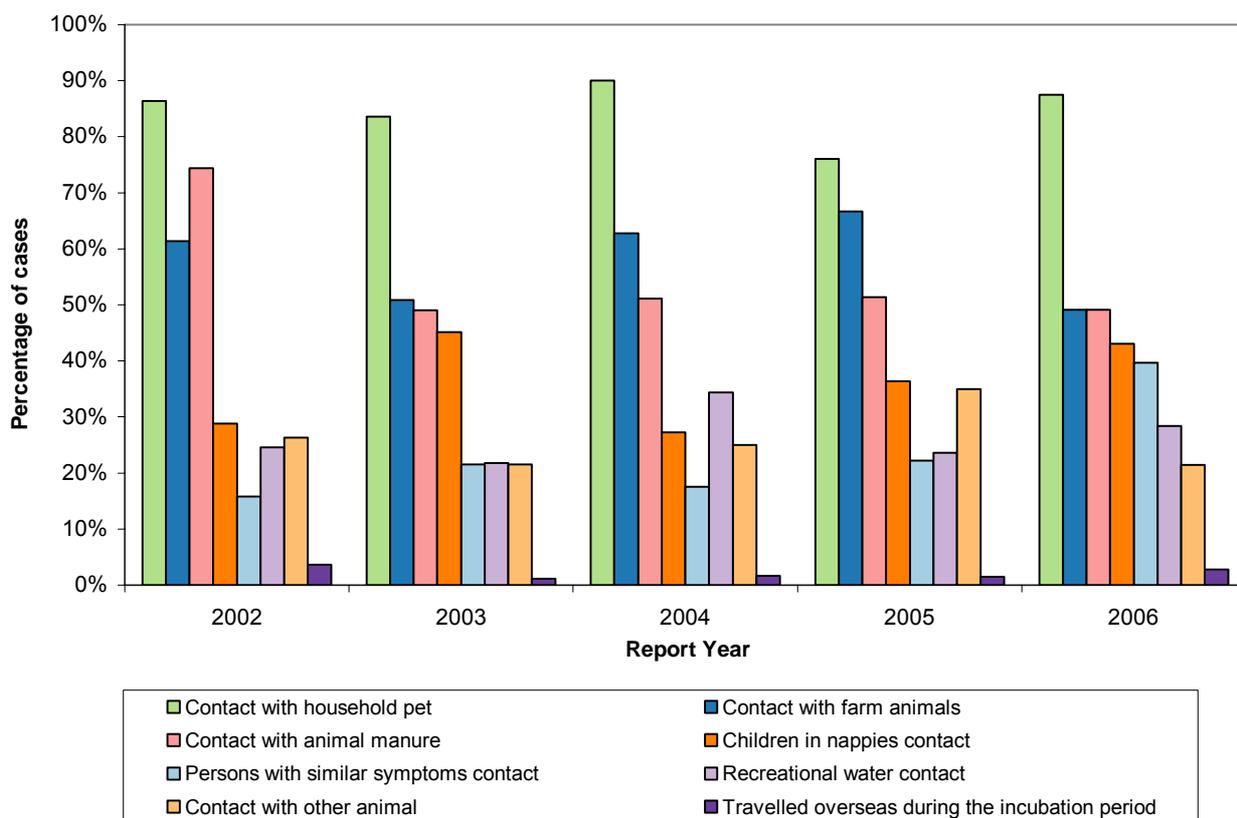
4.17.3.5 Estimate of travel-related cases

For cases where information on travel was provided, 2.7% (95%CI 0.3-7.7%) had travelled overseas during the incubation period. Assuming that the cases for which travel information was provided were representative of all VTEC/STEC infection cases, a Poisson distribution can be used to estimate the total number of potentially travel related cases of VTEC/STEC infection in 2006. The resultant distribution has a mean of 2 cases (95% CI 0-8).

4.17.4 Outbreaks reported as caused by VTEC/STEC

No foodborne VTEC/STEC outbreaks were reported in 2006 (Table 61).

Table 61:   VTEC/STEC outbreaks reported, 2006

Measure (No.)	Foodborne VTEC/STEC outbreaks	All VTEC/STEC outbreaks
Outbreaks	0	5
Cases	0	16
Hospitalised cases	0	0

Over the seven year period 2000 to 2006 there have been no more than two foodborne outbreaks of VTEC/STEC reported each year (Figure 50). The most recent foodborne outbreak was reported in 2005 involving four cases.

Figure 50:   Foodborne VTEC/STEC outbreaks and associated cases reported by year, 2000 – 2006
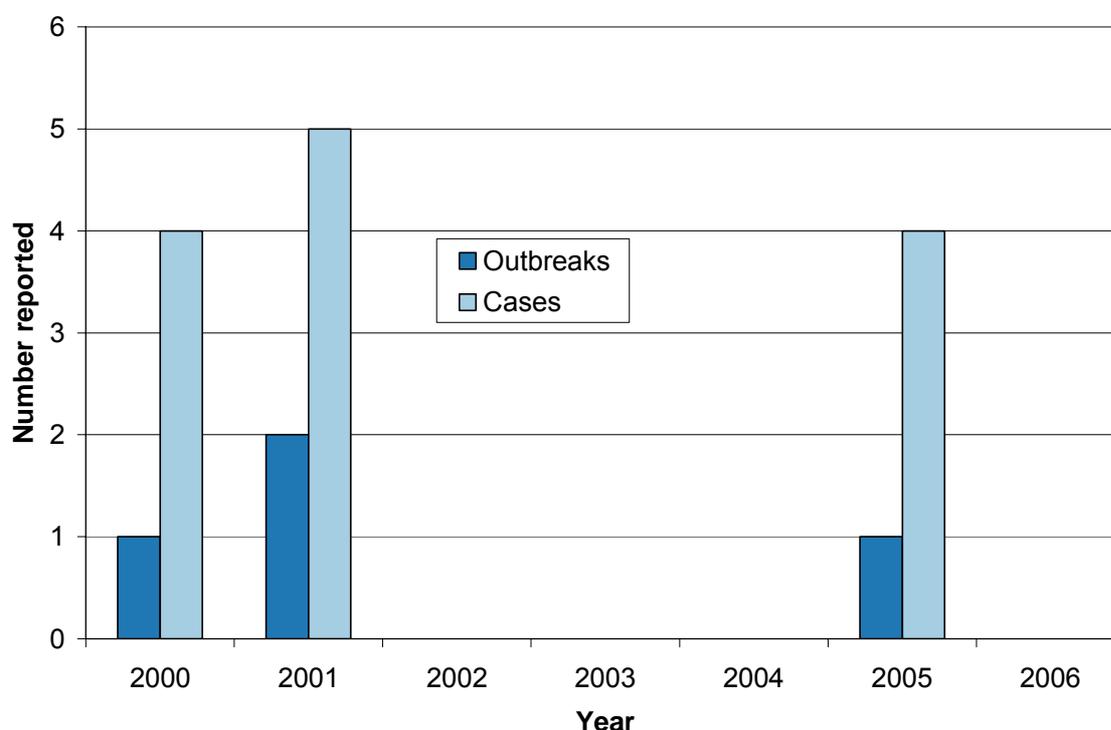
4.17.4.1 Details of food-associated outbreaks

No foodborne VTEC/STEC outbreaks were reported in 2006.

4.17.4.2 Laboratory investigation of samples from suspected foodborne outbreaks

During investigations of suspected foodborne illness outbreaks by ESR's Public Health Laboratories, a non-O157 VTEC/STEC was detected in a faecal sample from one investigation. While food was discussed in the outbreak report, this was not identified as a suspected foodborne outbreak.

4.17.5 VTEC/STEC types commonly reported

A total of 86 VTEC/STEC isolates were typed in 2006, of which 80 were *E. coli* O157:H7. The remaining six isolates were of types O111:H21 (associated with a HUS case), O91:H21, O128:H2 (two cases), O176:HNM and O103:H25.

4.17.6 Recent surveys

Shiga toxin-producing *Escherichia coli* and *E. coli* biotype 1 in uncooked retail meat products in New Zealand(Wong *et al.*, 2007?-a)

A national quantitative survey of shiga toxin-producing *Escherichia coli* (STEC) and generic *E. coli* in 878 samples of uncooked retail meats in New Zealand was undertaken from August 2003 to May 2005 to establish baseline proportionality data. The prevalence of STEC was 5.2% (95% Confidence Interval 2.7-8.8) in beef, 2.2% (0.6-5.5) in unweaned veal, 14.7% (10.4-20.0) in lamb/mutton and 6.5% (3.7-10.5) in pork (Note that poultry was not tested in this survey).

The counts of STEC obtained from positive meat samples were very low; one sample of lamb produced a count of 3.3 MPN/g, five samples (1 beef, 3 lamb and 1 pork) with counts of 1.0 MPN/g, four samples (2 lamb, 1 mutton and 1 pork) with 0.33 MPN/g and 49 samples with <0.33 MPN/g. Counts in three lamb samples were estimated at >1 MPN/g and one sample each of pork and lamb was estimated at <3.3 MPN/g.

Sixty-five isolates of STEC were identified, of which five were *E. coli* O157:H7 and two were *E. coli* O26:H11 isolates possessing *stx*1 and/or *stx*2 genes in conjunction with the intimin (*eae*A) and enterohaemolysin (*hly*A) genes. Less than 13% of 877 samples had *E. coli* biotype 1 counts above 100 CFU/g, 2.5% were above 1000 CFU/g and only 0.8% exceeded 5,000 CFU/g.

4.17.7 Disease sequelae - Haemolytic-uraemic syndrome (HUS)

Haemolytic-uremic syndrome is a serious sequela of an STEC enteric infection.

The ICD-10 code D59.3 was used to extract HUS hospitalisation data from the NZHIS NMDS database. Of the 34 hospitalised cases (0.8 cases per 100 000 population) recorded in 2006, 22 were reported with HUS as the primary diagnosis and 12 with this condition as another relevant diagnosis.

Over the five year period 2002 to 2006 between 20 and 34 hospitalised cases of HUS have been reported each year (Figure 51).

Figure 51:   HUS hospitalised cases, 2002 - 2006
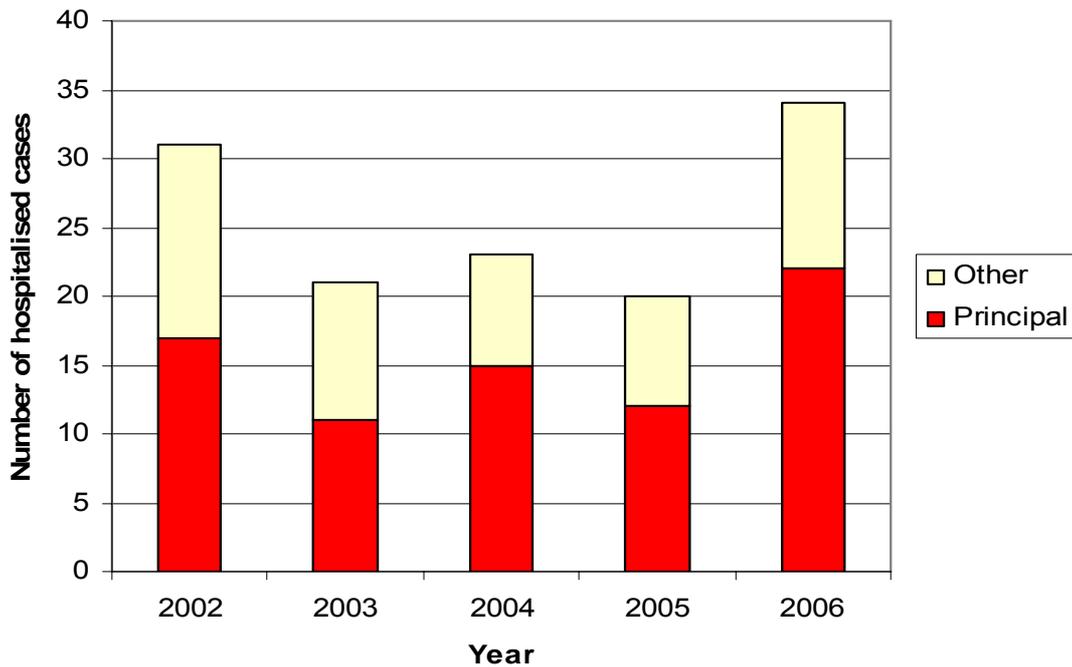
In 2006 the number of HUS hospitalised cases for males and females were very similar (Table 62).

Table 62:   HUS hospital admissions by sex, 2006

Sex	Hospitalised cases	
	No.	Rate per 100 000
Male	18	0.9
Female	16	0.7
Total	34	0.8

In 2006 the highest hospital admission rate for HUS occurred in 1 to 4 year olds (Table 63).

Table 63: HUS hospitalised cases by age group, 2006

Age groups	Hospitalisations	
	No.	Rate per 100 000
<1	1	1.7
1 to 4	14	6.2
5 to 9	4	1.4
10 to 14	0	0
15 to 19	3	1.0
20 to 29	2	0.4
30 to 39	2	0.3
40 to 49	2	0.3
50 to 59	3	0.6
60 to 69	2	0.6
70+	1	0.3
Total	**34**	**0.8**

4.17.7.1 Haemolytic-uraemic syndrome cases reported to the New Zealand Paediatric Surveillance Unit (NZPSU)

During 2006, 12 cases of HUS were reported to the NZPSU, with a mean age of 3.5 years (range 1.1 − 8.3 years). Ten of these cases had a diarrhoeal prodrome, with half of these (five cases) having *E. coli* O157:H7 isolated from their stools (Source: http://dnmeds.otago.ac.nz/departments/womens/paediatrics/research/nzpsu/pdf/2006_report.pdf).

4.17.8 Relevant New Zealand studies and publications

Nil.

4.17.9 Relevant regulatory developments

Nil.

4.18 Yersiniosis

Summary data for yersiniosis in 2006 are given in Table 64.

Table 64: Summary surveillance data for yersiniosis, 2006

Parameter	Value in 2006	Section reference
Number of cases	487	4.18.2
Rate (per 100 000)	11.6	4.18.2
Hospitalisations (%)	55 (11.3%)	4.18.2
Deaths (%)	Nil	4.18.2
Estimated travel-related cases (%)	32 (6.7%)	4.18.3.6
Estimated food-related cases (%)*	255 (56%)	4.18.2

* For estimation of food-related cases it was assumed that the proportions derived from expert consultation would exclude travel-related cases

4.18.1 <u>Case definition</u>

Clinical description: An acute illness with diarrhoea, fever and abdominal pain. Mesenteric adenitis may occur and complications include arthritis and systemic infection

Laboratory test for diagnosis: Isolation of *Yersinia enterocolitica* or *Y. pseudotuberculosis* from blood or faeces OR detection of circulating antigen by ELISA or agglutination test

Case classification:

Probable A clinically compatible illness that is either a contact of a confirmed case of the same disease, or has had contact with the same common source i.e., is part of an identified common source outbreak

Confirmed A clinically compatible illness that is laboratory confirmed

4.18.2 <u>Yersiniosis cases reported in 2006 by data source</u>

During 2006, 487 notifications (11.6 cases per 100 000) of yersiniosis were reported in EpiSurv.

The ICD-10 code A04.6 was used to extract yersiniosis hospitalisation data from the NZHIS NMDS database. Of the 55 hospital admissions (1.3 admissions per 100 000 population) recorded in 2006, 29 were reported with yersiniosis as the primary diagnosis and 26 with yersiniosis as another relevant diagnosis.

No deaths resulting from yersiniosis were recorded in EpiSurv in 2006.

It has been estimated by expert consultation that 56% (minimum = 42%, maximum = 71%) of yersiniosis incidence is due to foodborne transmission. Approximately 50% of foodborne transmission was estimated to be due to consumption of pork.

4.18.3 <u>Notifiable disease data</u>

4.18.3.1 Annual notification trend

In 2006, 487 yersiniosis notifications were reported in EpiSurv, the highest number of notifications since 1999 (503 notifications) (Figure 52). Yersiniosis became notifiable in 1996, with the highest number of notifications reported in 1998 (546 notifications). Over the six years period 2000 to 2005 the number of cases reported increased between 2000 and 2002 before gradually declining to 407 cases in 2005.

Figure 52:   Yersiniosis notifications by year, 1996-2006
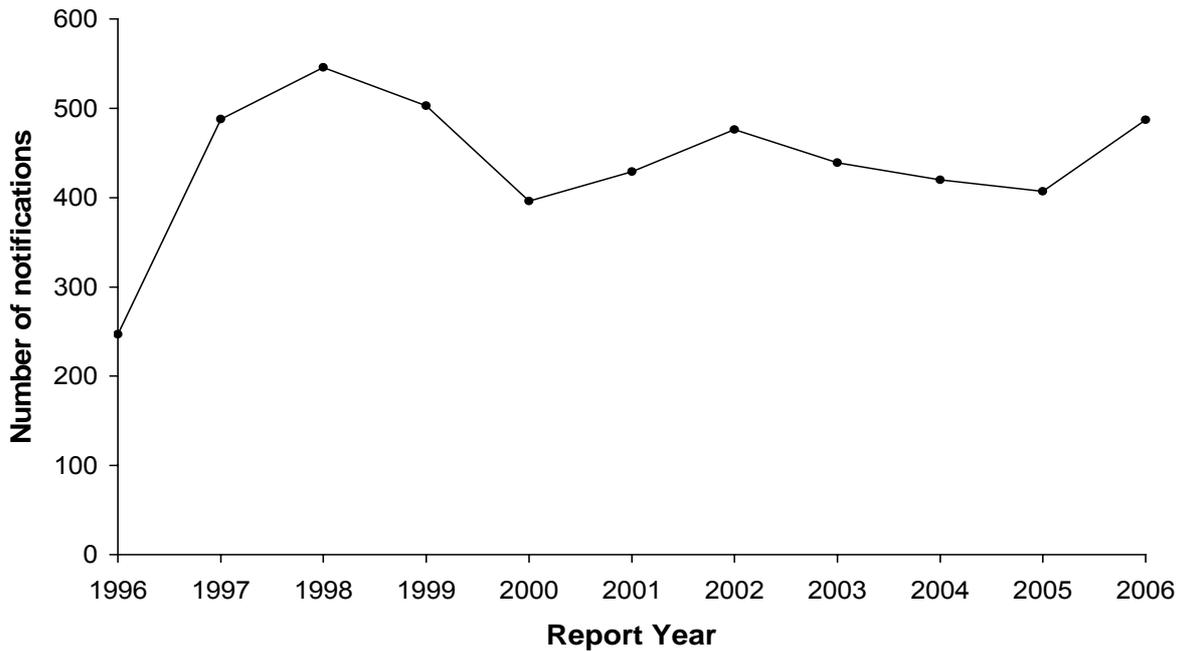
In 2006 the yersiniosis notification rate was 11.6 cases per 100 000 population. The yersiniosis notification rate has varied little (ranging from 9.9 to 12.1 per 100 000) between 2000 and 2006 (Figure 53).

Figure 53:   Yersiniosis notification rate by year, 2000-2006
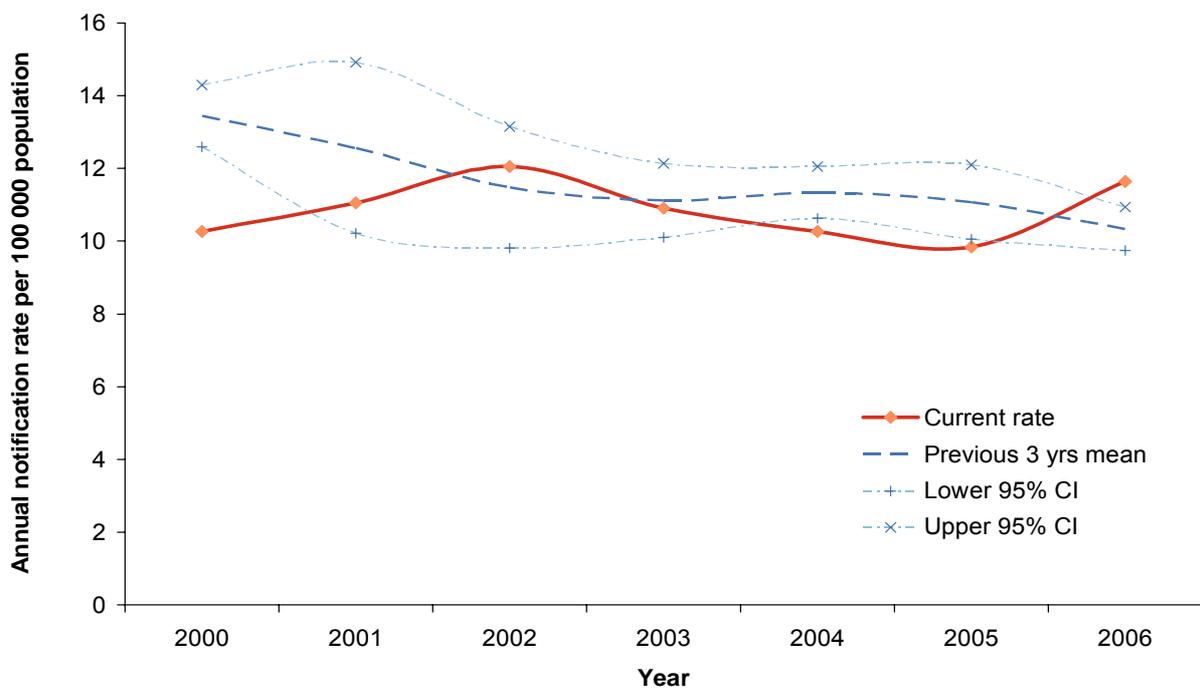
4.18.3.2 Seasonality

The number of notified cases of yersiniosis per 100 000 population by month for 2006 is shown in Figure 54. The historic mean rate shows a general downward trend in yersiniosis rates between January (highest notification rate) and June, and a general increase in rates for the remainder of the year. The 2006 notification rate follows a similar pattern but with peaks observed in May, August and October.

Figure 54: Yersiniosis monthly rate (annualised) for 2006
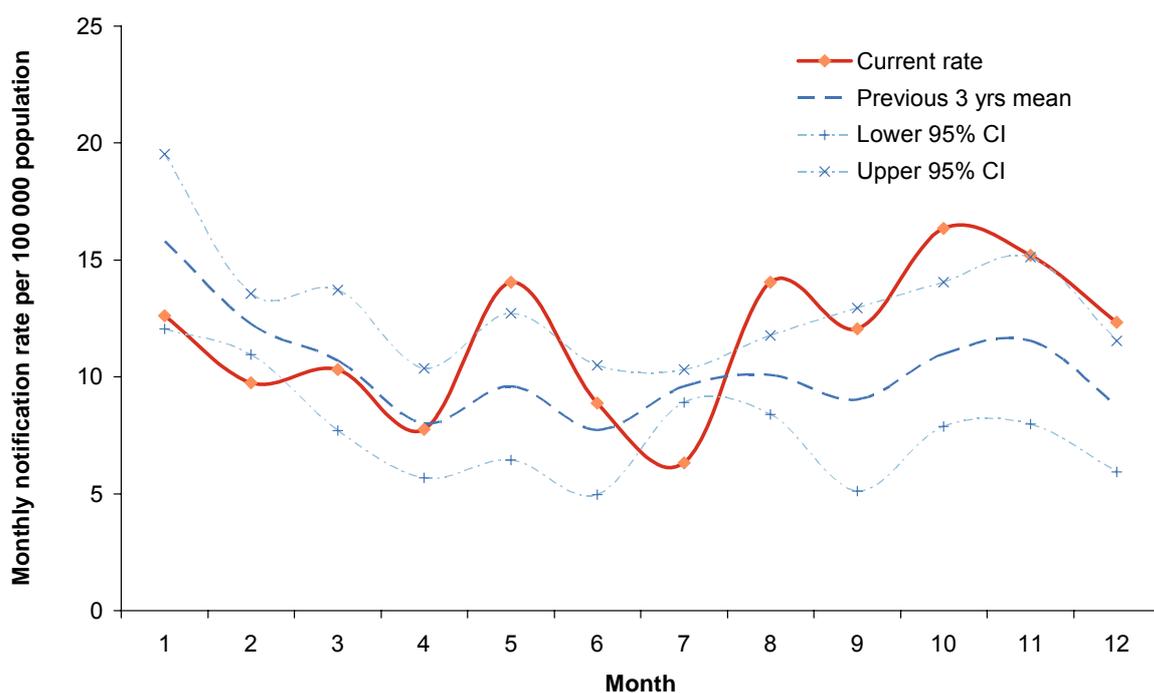
4.18.3.3 Geographic distribution of yersiniosis notifications

Yersiniosis notification rates vary throughout New Zealand as illustrated in Figure 55. The past two years have seen high notification rates for the majority of the South Island, with the exception of Southland DHB. Consistent with previous years, West Coast, South Canterbury and Capital and Coast DHBs recorded the highest rates for 2006. Similarly, MidCentral, Taranaki and Northland consistently had low yersiniosis notification rates.

Figure 55: **Geographic distribution of yersiniosis notifications, 2003-2006**

Rate per 100 000 population

2 - 7

8 - 11

12 - 50

2006

2003

2004

2005
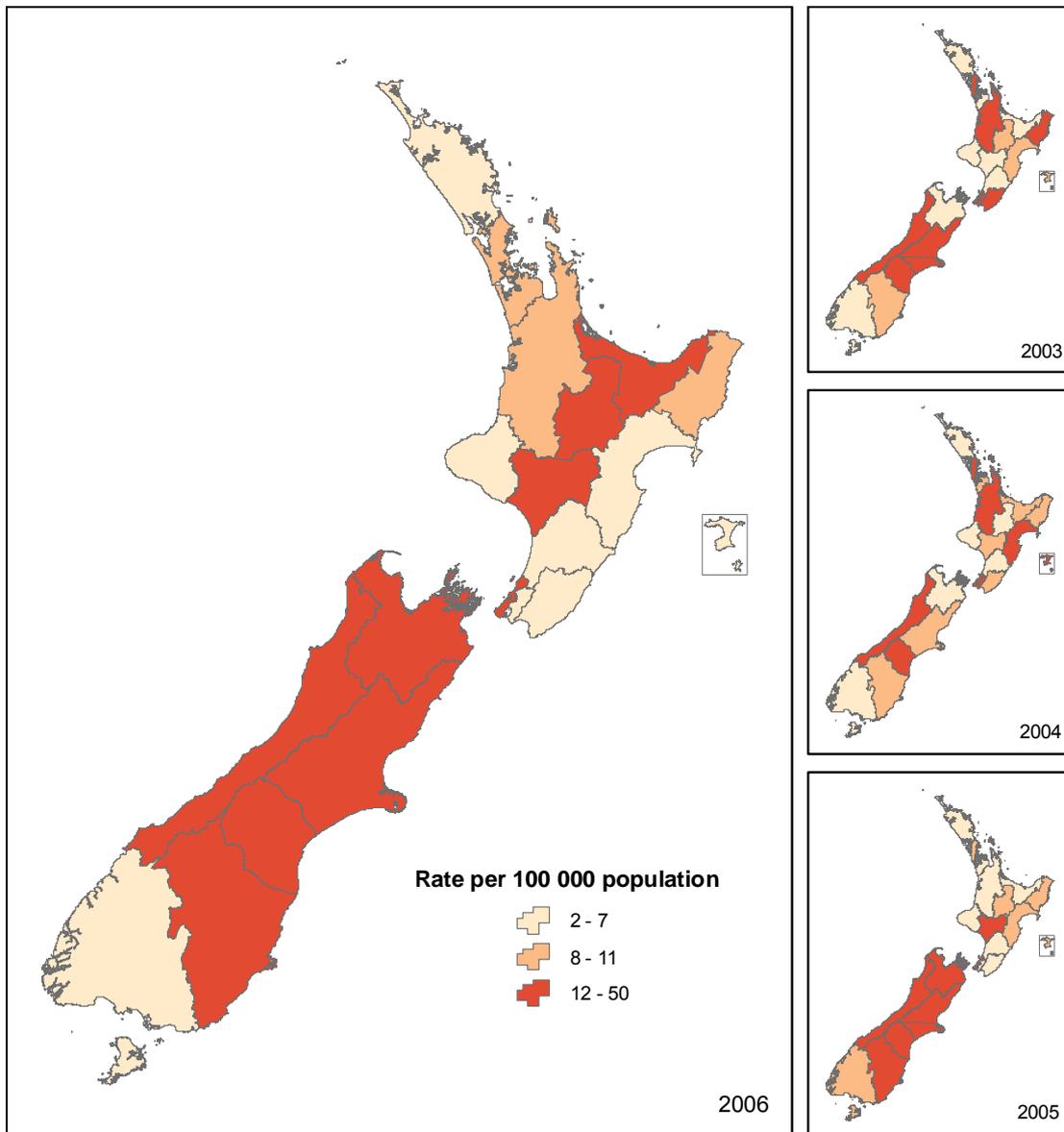
4.18.3.4 Age and sex distribution of yersiniosis cases

The yersiniosis notification rate was similar for males and females, with notification rates being slightly higher for males; conversely, the hospitalisation rate was slightly higher for females (Table 65).

Table 65: **Yersiniosis cases by sex, 2006**

Sex	EpiSurv notifications		Hospitalisations[a]		Deaths recorded in EpiSurv
	No.	Rate	No.	Rate	No
Male	247	12.1	25	1.2	
Female	222	10.4	30	1.4	
Unknown	18	-	0	-	
Total	**487**	**11.6**	**55**	**1.3**	

[a] NZHIS morbidity data for hospital admissions

In 2006 the highest age-specific yersiniosis notification rate was for those aged less than one year for notifications (47.4 per 100 000 population) and hospitalisations (8.5 per 100 000 population) (Table 66). The notification rate for those aged one to four years (37.5 per 100 000 population) was more than three times higher than for any other age group.

Table 66:   Yersiniosis cases by age group, 2006

Age groups	EpiSurv notifications		Hospitalisations[a]		Deaths recorded in EpiSurv
	No.	Rate	No.	Rate	No.
<1	28	47.4	5	8.5	
1 to 4	85	37.5	1	0.4	
5 to 9	10	3.4	0	0.0	
10 to 14	12	3.9	2	0.6	
15 to 19	14	4.5	1	0.3	
20 to 29	60	10.9	12	2.2	
30 to 39	69	11.5	5	0.8	
40 to 49	60	9.6	2	0.3	
50 to 59	63	12.4	8	1.6	
60 to 60	37	10.8	7	2.0	
70+	46	12.9	12	3.4	
Unknown	3	-	0	-	
Total	**487**	**11.6**	**55**	**1.3**	**-**

[a] NZHIS Morbidity data for hospital admissions

4.18.3.5 Risk factors reported

The most commonly reported risk factor for yersiniosis notification cases during 2006 was consumption of food from retail premises (34.6%) followed by contact with farm animals (21.6%) (Table 67).

Table 67:   Exposure to risk factors associated with yersiniosis, 2006

Risk Factor	Notifications			
	Yes	No	Unknown	%[a]
Consumed food from retail premises	72	136	279	34.6%
Contact with farm animals	58	210	219	21.6%
Consumed untreated water	47	187	253	20.1%
Contact with faecal matter	49	202	236	19.5%
Recreational water contact	29	222	236	11.6%
Contact with other symptomatic people	24	222	241	9.8%
Travelled overseas during the incubation period	19	266	202	6.7%
Contact with sick animals	11	230	246	4.6%
Contact with a confirmed case of same disease	1	201	285	0.5%

[a]Percentage refers to the cases that answered "yes" out of the total number of cases for which this information was supplied. Cases may have more than one risk factor recorded.

Between 2002 and 2006 the risk factors associated with yersiniosis cases have generally occurred in the same order of importance and to the same magnitude each year (Figure 56). Over the past five years the consumption of food from retail premises has been the most commonly reported risk factor associated with yersiniosis cases followed by contact with farm animals. The percentage of cases with the risk factors recreational water contact and contact with faecal matter varies from year to year.

Figure 56:   Yersiniosis risk factors by percentage of cases and year, 2002 – 2006
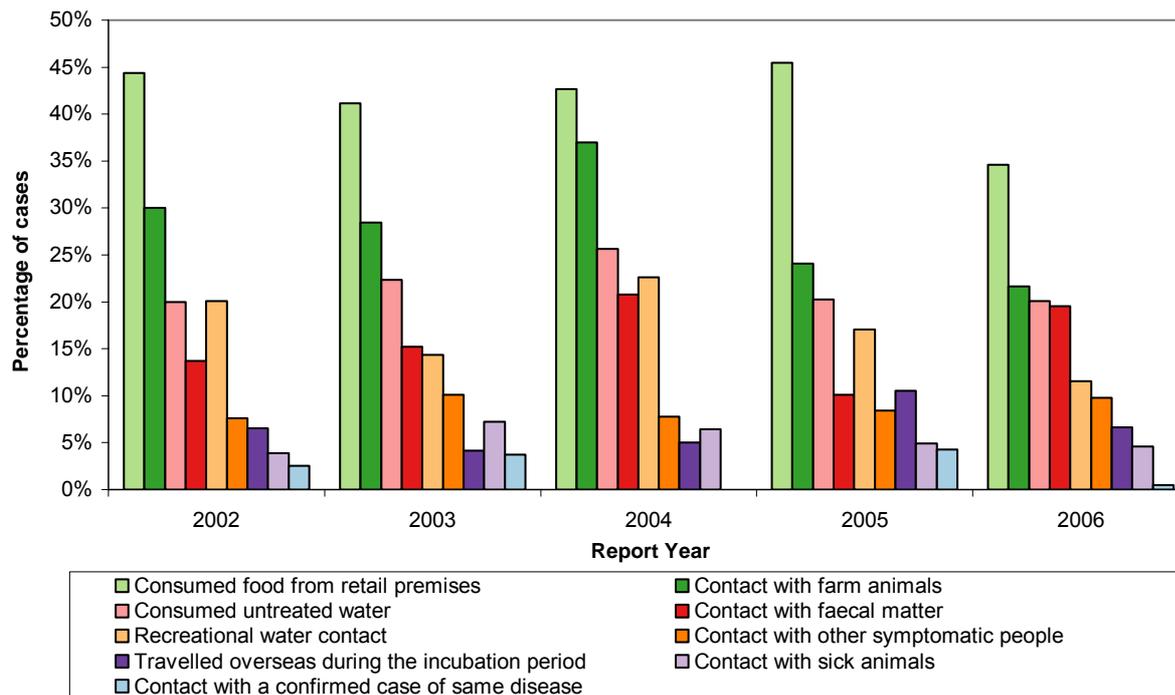
4.18.3.6 Estimate of travel-related cases

For cases where information on travel was provided, 6.7% (95%CI 4.0-10.0%) had travelled overseas during the incubation period. Assuming that the cases for which travel information was provided were representative of all yersiniosis cases, a Poisson distribution can be used to estimate the total number of potentially travel related cases of yersiniosis in 2006. The resultant distribution has a mean of 32 cases (95% CI 16-53).

If data from the last four years are considered the estimated proportion of cases travelling overseas within the incubation period of the organism is 6.7% (95% CI 5.1-8.4%).

4.18.4 Outbreaks reported as caused by *Yersinia* spp.

No *Yersinia* spp. outbreaks were reported in EpiSurv in 2006.

Between 2001 and 2005 very few foodborne *Yersinia* spp. outbreaks were reported in EpiSurv (two or less each year), with a small total number of associated cases (ranging from two to eight) (Figure 57).

Figure 57: **Foodborne *Yersinia* spp. outbreaks and associated cases reported by year, 2000 – 2006**
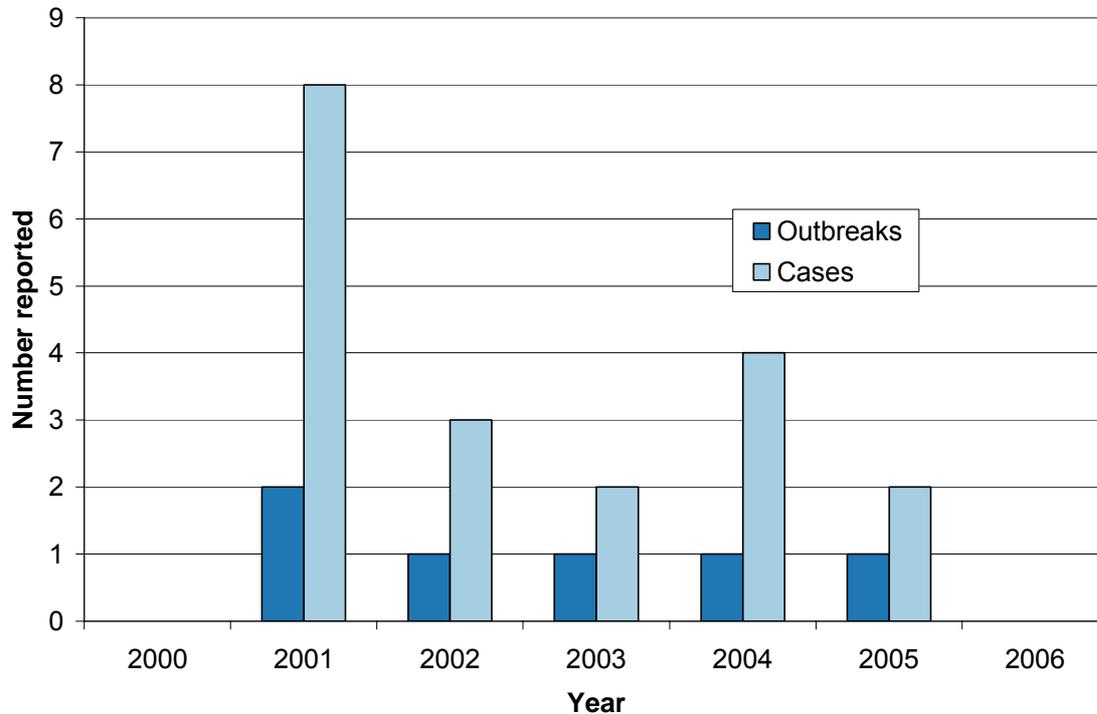
4.18.4.1 Details of food-associated outbreaks

No *Yersinia* spp. outbreaks were reported in EpiSurv in 2006.

4.18.4.2 Laboratory investigation of samples from suspected foodborne outbreaks

During investigations of suspected foodborne illness outbreaks by ESR's Public Health Laboratories, no samples were found to contain *Yersinia enterocolitica.*

4.18.5 Recent surveys

***Yersinia* in meat: Analytical Development and Survey (King and Hudson, 2006)**

The detection and isolation of pathogenic *Y. enterocolitica* from foods is confounded by the likelihood that the bacterium is present in small numbers, and by the possible presence of faster-growing microflora and other *Yersinia* species. An improved method for the detection (presence/absence) and enumeration from meat of *Y. enterocolitica* containing the pYV virulence plasmid (*Ye*P+) is reported. The detection method combines a multiplex PCR targeting the *ail* and *virF* genes with a number of selective media, which were evaluated for their capacity to detect, isolate and identify *Ye*P+ from the surface of meat portions and from comminuted meats.

Enumeration is achieved using the most-probable number (MPN) method. A presumptive result is available within 24 hours of sample receipt and any *Ye*P+ isolates confirmed within four days. The

conservative detection limit for meat surfaces was 10 CFU/cm^2, and was 100 CFU/g for comminuted meats.

The presence/absence and MPN methods were evaluated in a pilot survey of 41 raw pork meats purchased from retail outlets in Christchurch, New Zealand. *Ye*P+ was detected by PCR on 32% of whole meat samples tested (steak, chop, schnitzel) and in 86% of the comminuted meat samples. *Ye*P+ isolates were obtained from 18% and 43% of the whole and comminuted meat samples, respectively. The count of *Ye*P+ on whole meat samples ranged from 0.30 to 5.42 MPN/cm^2, and from 0.31 to >42.90 MPN/g in comminuted meats. This improved method for the detection and enumeration of *Ye*P+ from meat samples will be used to provide data for exposure assessment and is amenable to outbreak investigations.

4.18.6 <u>Relevant New Zealand studies and publications</u>

Nil.

4.18.7 <u>Relevant regulatory developments</u>

Nil.

5   SUMMARY TABLES

This appendix brings together data from different sources as summary tables to facilitate comparisons between conditions.

Table 68:   **Cases and rates per 100 000 population of notifiable diseases in New Zealand during 2005 and 2006**

Disease	2005		2006		Change [b,c]
	Cases	**Rates**	**Cases**	**Rates**	
Campylobacteriosis	13 836	337.6	15 873	379.3	→
Cryptosporidiosis	889	21.7	737	17.6	←
Gastroenteritis[a]	557	13.6	931	22.5	→
Giardiasis	1 231	30.0	1 214	29.0	←
Hepatitis A	51	1.2	123	2.9	→
Listeriosis	20	0.5	19	0.5	←
Salmonellosis	1 382	33.7	1 335	31.9	←
Shigellosis	183	4.5	102	2.4	←
Toxic shellfish poisoning	3	0.1	3	0.1	
VTEC/STEC infection	92	2.2	87	2.1	←
Yersiniosis	407	9.9	487	11.6	→

[a] Cases of gastroenteritis from a common source or foodborne intoxication e.g. staphylococcal intoxication
[b] Only acute cases of this disease are currently notifiable
[c] ←= Significant decrease, → = Significant increase, -- = No change, ← = Not significant decrease, → = not significant increase
[e] The Mantel-Haenszel chi-square test was used to determine statistical significance. P-values less than or equal to 0.05 are considered to be significant at the 95% level of confidence.

Table 69:   Deaths due to notifiable diseases recorded in EpiSurv from 1997 to 2006

Disease	1997	1998	1999	2000	2001	2002	2003	2004	2005	2006
Campylobacteriosis	2	2	1	3	1	1	0	0	1	1
Gastroenteritis	0	0	0	0	0	1	0	0	0	0
Giardiasis	1	0	0	0	0	0	0	0	0	0
Listeriosis - non perinatal	2	0	1	2	1	0	2	3	1	0
Listeriosis - perinatal	6	0	2	4	1	3	2	2	0	1
Salmonellosis	2	2	1	7	2	1	0	0	1	1
Shigellosis	0	0	1	0	0	0	0	0	0	0
VTEC/STEC infection	1	1	0	0	0	0	0	0	0	0
Yersiniosis	0	2	0	0	0	0	0	1	0	0

Note: The numbers in this table are those recorded in EpiSurv where the notifiable disease was the primary cause of death. Information on deaths is most likely to be reported by Public Health Services when it occurs close to the time of notification and investigation.

Table 70:   NZHIS death data for selected potential foodborne diseases, 2003

Disease	ICD 10 Codes	2001		2002		2003 [a]	
		Underlying[b]	Contributory[c]	Underlying[b]	Contributory[c]	Underlying[b]	Contributory[c]
Campylobacteriosis	A04.5	2	0	0	0	1	0
Cryptosporidiosis	A072					1	0
Giardiasis	A07.1	1	0	0	0		0
Hepatitis A	B15	0	1	1	0		0
Listeriosis	A32	1	0	1	0	2	
Salmonellosis	A02	2	0	0	0	1	0
Shigellosis	A03	0	0	0	0	0	0
Yersiniosis	A04.6	0	0	0	0	0	0

[a]Underlying – main cause of death

[b]Contributory – selected contributory cause of death (not main cause of death)

Note : Mortality data has not yet been published by NZHIS for years after 2003

Table 71:   Hospital admissions for selected notifiable diseases, 2004 - 2006

Disease	ICD 10 Codes	2004		2005		2006	
		Principal diagnosis	Other relevant diagnosis	Principal diagnosis	Other relevant diagnosis	Principal diagnosis	Other relevant diagnosis
Bacillus cereus intoxication	A05.4					0	6
Campylobacteriosis	A04.5	747	173	871	199	967	212
Ciguatera fish poisoning	T61.0					5	0
Cryptosporidiosis	A07.2	16	8	34	8	20	10
Giardiasis	A07.1	30	25	27	25	43	28
Hepatitis A	B15	12	16	21	15	33	14
Histamine fish poisoning	T61.1					5	0
Listeriosis	A32	13	18	8	11	13	10
Norovirus infection	A08.1					15	43
Salmonellosis	A02	105	42	130	36	122	39
Shigellosis	A03	26	5	20	2	13	2
Staphylococcus aureus intoxication	A05.0					1	1
Toxic shellfish poisoning	T61.2					17	4
VTEC/STEC infection	A04.3					5	2
Yersiniosis	A04.6	17	13	12	15	29	26

Note: Hospital admission data may include multiple admissions (to the same or different hospitals) for the same case and admissions may relate to cases first diagnosed in previous years.

Table 72:   Cases reported in 2006 by ethnic group

Ethnic Group	European	Maori	Pacific People	Other	Unknown	Total
Campylobacteriosis	10 787	818	200	702	3 366	15 873
Cryptosporidiosis	597	49	8	28	55	737
Gastroenteritis	737	40	16	33	107	933
Giardiasis	866	69	10	65	204	1 214
Hepatitis A	53	7	42	13	8	123
Listeriosis	12		4	1	2	19
Salmonellosis	958	109	45	61	162	1 335
Shigellosis	45	6	11	18	22	102
VTEC/STEC infection	75	4	2	2	4	87
Yersiniosis	316	31	8	48	84	487

Table 73: Cases and rates per 100 000 population in 2006 by sex

Disease	Male Cases	Male Rate	Female Cases	Female Rate	Unknown Cases	Unknown Rate	Total Cases	Total Rate
Campylobacteriosis	8 238	402.2	7 269	340.3	366		15 873	379.3
Cryptosporidiosis	364	17.8	362	16.9	11		737	17.6
Gastroenteritis	361	17.2	553	27.1	19		933	22.5
Giardiasis	620	30.3	566	26.5	28		1 214	29.0
Hepatitis A	65	3.2	56	2.6	2		123	2.9
Listeriosis	10	0.5	7	0.3	2		19	0.5
Salmonellosis	673	32.9	639	29.9	23		1 335	31.9
Shigellosis	46	2.2	52	2.4	4		102	2.4
VTEC/STEC infection	44	2.1	42	2.0	1		87	2.1
Yersiniosis	247	12.1	222	10.4	18		487	11.6

Table 74: Cases and rates per 100 000 population in 2006 by age group

Disease	<1 Cases	<1 Rate	1 to 4 Cases	1 to 4 Rate	5 to 9 Cases	5 to 9 Rate	10 to 14 Cases	10 to 14 Rate	15 to 19 Cases	15 to 19 Rate	20 to 29 Cases	20 to 29 Rate	30 to 39 Cases	30 to 39 Rate	40 to 49 Cases	40 to 49 Rate	50 to 59 Cases	50 to 59 Rate	60 to 69 Cases	60 to 69 Rate	70+ Cases	70+ Rate	Unknown Cases	Unknown Rate	Total Cases	Total Rate
Campylobacteriosis	237	401.3	1227	540.7	680	233.0	713	229.7	1251	399.0	2884	522.9	2218	370.5	2034	324.1	1888	372.6	1396	408.0	1213	340.6	132		15873	379.3
Cryptosporidiosis	19	32.2	252	111.0	88	30.1	59	19.0	39	12.4	88	16.0	90	15.0	54	8.6	32	6.3	9	2.6	6	1.7	1		737	17.6
Gastroenteritis	4		22	9.8	10	3.5	19	6.3	36	11.5	82	14.9	139	23.7	132	21.4	131	26.2	79	23.4	219	61.0	60		933	22.5
Giardiasis	24	40.6	254	111.9	76	26.0	24	7.7	17	5.4	122	22.1	289	48.3	160	25.5	117	23.1	83	24.3	41	11.5	7		1214	29.0
Hepatitis A			16	7.1	19	6.5	11	3.5	14	4.5	11	2.0	17	2.8	11	1.8	10	2.0	6	1.8	8	2.2			123	2.9
Listeriosis	1	1.7	1	0.4							2	0.4	1	0.2	2	0.3	2	0.4	4	1.2	6	1.7			19	0.5
Salmonellosis	83	140.5	280	123.4	93	31.9	55	17.7	67	21.3	185	33.5	132	22.1	119	19.0	143	28.2	101	29.5	73	20.5	4		1335	31.9
Shigellosis			15	6.6	10	3.4	2	0.6	3	1.0	18	3.3	20	3.3	13	2.1	10	2.0	7	2.0	3	0.8	1		102	2.4
VTEC/STEC infection	6	10.2	38	16.7	5	1.7	3	1.0	1	0.3	7	1.3	4	0.7	5	0.8	3	0.6	8	2.3	5	1.4	2		87	2.1
Yersiniosis	28	47.4	85	37.5	10	3.4	12	3.9	14	4.5	60	10.9	69	11.5	60	9.6	63	12.4	37	10.8	46	12.9	3		487	11.6

Table 75: **Disease notifications and incidence rates per 100 000 population by District Health Board, 2006**

Disease	Campylobacteriosis		Cryptosporidiosis		Gastroenteritis		Giardiasis		Hepatitis		Listeriosis		Salmonellosis		Shigellosis		VTEC/STEC		Yersiniosis	
District Health Board	Cases	Rate	Cases	Rate	Cases	Rate	Cases	Rate	Cases	Rate	Cases	Rate	Cases	Rate	Cases	Rate	Cases	Rate	Cases	Rate
Northland	374	249.9	24	16.0	3		69	46.1	4		1		45	30.1	3		5	3.3	9	6.0
Waitemata	2319	459.6	50	10.0	79	15.8	118	23.5	15	3.0	4		131	26.0	19	3.8	12	2.4	41	8.2
Auckland	1813	421.1	34	7.9	83	19.3	158	36.7	10	2.3	1		109	25.3	21	4.9	3		45	10.5
Counties Manukau	1548	350.3	25	5.7	53	12.0	103	23.3	38	8.6	3		96	21.7	14	3.2	4		34	7.7
Waikato	1220	356.3	96	28.0	59	17.2	134	39.1	3		1		132	38.9	4		16	4.7	33	9.6
Lakes	410	403.5	16	15.7	14	13.8	37	36.4	3				17	16.7	2		3		20	19.7
Bay of Plenty	650	327.1	25	12.6	17	8.6	66	33.2	3		1		53	26.7	2		2		30	15.1
Tairawhiti	58	130.3	8	18.0			10	22.5	1				10	22.5			1		4	
Taranaki	472	448.8	16	15.2	6	5.7	12	11.4	1				47	44.7			2		7	6.7
Hawke's Bay	518	344.0	16	10.6	20	13.3	52	34.5			2		69	45.8	3		3		10	6.6
Whanganui	233	375.2	19	30.6	45	72.5	23	37.0	3				15	24.2	5				9	14.5
MidCentral	359	219.9	58	35.5	234	143.3	33	20.2	2		1		35	21.4			1		10	6.1
Hutt	606	437.9	28	20.2	35	25.3	33	23.8	2		1		40	28.9	1				8	5.8
Capital and Coast	1425	510.4	52	18.6	72	25.8	98	35.1	4		1		118	42.3	9	3.2			69	24.7
Wairarapa	89	226.9	25	63.2	1		14	35.7	1				16	40.8					1	
Nelson	359	262.6	24	17.6	23	16.8	38	27.8	1		1		48	35.1	7	5.1	6	4.4	18	13.2
West Coast	74	242.5	16	49.8	5	16.4	5	16.4					5	16.4	1				9	29.5
Canterbury	1904	398.4	88	18.4	136	28.5	110	23.0	29	6.1			153	32.0	9	1.9	15	3.1	89	18.6
South Canterbury	285	517.4	41	74.4	4		16	29.9			1		37	67.2			4		10	18.7
Otago	742	406.2	44	24.1	29	15.9	52	28.5	1				86	47.1	2		6	3.3	27	14.8
Southland	415	379.6	31	28.4	15	13.7	33	30.2	1		1		73	66.8			4		4	
Total	15873	379.3	737	17.6	933	22.5	1214	29.0	123	2.9	19	0.5	1335	31.9	102	2.4	87	2.1	487	11.6

Note : Where less than 5 cases have been notified a rate has not been calculated and the cell has been left blank.

Table 76:   Notifiable disease cases by year and source, 1987-2006

Note: cell is blank where data are unavailable

Disease	1987	1988	1989	1990	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000	2001	2002	2003	2004	2005	2006
Campylobacteriosis	2921	2796	4187	3850	4148	5144	8101	7714	7442	7635	8924	11572	8161	8418	10146	12494	14787	12215	13836	15873
Cryptosporidiosis										119	357	866	977	775	1208	975	817	611	889	737
Gastroenteritis										555	310	492	601	726	940	1087	1025	1363	557	933
Giardiasis										1235	2127	2183	1793	1688	1604	1547	1570	1514	1231	1214
Hepatitis A	158	176	134	150	224	288	257	179	338	311	347	145	119	107	61	106	70	49	51	123
Listeriosis	12	7	10	16	26	16	11	8	13	10	35	17	19	22	18	19	24	26	20	19
Salmonellosis	1140	1128	1860	1619	1244	1239	1340	1522	1334	1141	1177	2069	2077	1795	2417	1880	1401	1081	1382	1335
Shigellosis	143	145	137	197	152	124	128	185	191	167	117	122	147	115	157	112	87	140	183	102
VTEC/STEC infection							3	3	6	7	13	48	64	67	76	73	104	89	92	87
Yersiniosis										330	488	546	503	396	429	476	439	420	407	487

Table 77:   Foodborne outbreaks and associated cases by agent type, 2006

Agent type	No. of outbreaks	No. of cases
Campylobacter spp.	32	135
Norovirus	23	346
Clostridium perfringens	12	62
Salmonella spp.	9	33
Histamine	4	14
Bacillus cereus	2	11
Giardia	2	4
Shigella spp.	2	10
Hepatitis A virus	1	34
Unidentified pathogen[a]	57	253
Total	**146**	**907**

[a] All outbreaks with no pathogen identified were classified as gastroenteritis

Table 78:   Outbreaks associated with commercial food operators, 2006

Outbreak setting	No. of outbreaks[1]	% of total outbreaks (n=495)	No. of cases[1]	% of total cases (n=6300)
Restaurant/Café	73	14.7	479	7.6
Takeaway	23	4.6	86	1.4
Caterer	5	1.0	61	1.0
Other food outlet	5	1.0	37	0.6
Supermarket/deli	4	0.8	12	0.2

Table 79: Foodborne outbreaks and associated cases by implicated food source, 2006

Implicated food source	No. of outbreaks[a]	% of outbreaks (n=146)	No. of cases	% of cases (n=907)
Poultry	22	15.1	148	16.3
Meat (lamb, beef, pork)	17	11.6	90	9.9
Rice/noodles/pasta	14	9.6	74	8.1
Fish	9	6.2	28	3.1
Shellfish	8	5.5	142	15.6
Eggs	6	4.1	22	2.4
Fruit and vegetables	4	2.7	29	3.2
Sandwich/burger	4	2.7	11	1.2
Pies	2	1.4	10	1.1
Processed meat	1	0.7	7	0.8
Deli foods	1	0.7	19	2.1
Dairy	1	0.7	6	0.7
Cereal	1	0.7	2	0.2
Unclassifiable	14	9.6	91	10.0
Unknown vehicles	4	2.7	139	15.3

a   More than one food source was implicated in some outbreaks

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