ESTIMATION OF THE BURDEN OF WATER-BORNE DISEASE IN NEW ZEALAND: PRELIMINARY REPORT

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EXECUTIVE SUMMARY

This report has been written to explain the relationship between drinking-water quality and waterborne gastro-intestinal disease (GID) in New Zealand, and the role of the Drinking-water Standards for New Zealand (DWSNZ) in the protection of public health. This interim report provides an overview of the progress that has been made to date on a current research project, summarises the available data, and provides a preliminary estimate for the burden of waterborne GID.

There is ample evidence of waterborne disease outbreaks in New Zealand to indicate a significant risk of contracting GID from drinking-water that is untreated or inadequately treated. An average of 16.8 waterborne outbreaks (range from 6 to 27) occur annually, affecting an average of 145 cases/year (range from 18 to 370). While the largest reported waterborne outbreak affected 3,500 people (Queenstown, 1984), the number of cases involved in most outbreaks is small, averaging nine cases per outbreak in 2001-2005, and is smaller than other countries for which data are available. This probably reflects the larger proportion of water supplies serving small communities in New Zealand compared to most other developed countries. This is consistent with the relatively poor compliance with the DWSNZ of the small community drinking-water supplies compared to that of the larger community supplies.

The occurrence of endemic waterborne gastro-intestinal disease in New Zealand has been demonstrated by a number of epidemiological studies.

Based on currently available data, two separate estimates of the burden of endemic drinking-waterborne gastro-intestinal disease are *ca.* 18,000 and 34,000 cases per annum. Preliminary results from work in progress suggest that these are under-estimates.

There is broad international consensus for the minimum bacteriological drinking-water standard of <1 *E. coli*/100mL or <1 FC/100mL, which is the standard applied in New Zealand.

1 Introduction

Deriving a reliable estimate of the national burden of endemic waterborne infectious disease in New Zealand is the subject of a current research project for the Ministry of Health that is scheduled to be completed in 2008. This interim report provides an overview of the progress that has been made to date, summarises the available data, and provides a preliminary estimate for the burden.

This report has been written to explain the relationship between drinking-water quality and waterborne disease in New Zealand, and the role of the Drinking-water Standards for New Zealand (DWSNZ) in the protection of public health. The disease considered is primarily acute gastro-intestinal disease (GID) caused by pathogens in drinking water. Chemicals in water are excluded.

2 Background

Infectious enteric (gastrointestinal) diseases are caused by the ingestion of pathogens, and these usually originate from the faeces of infected humans and animals. Eating contaminated food, drinking contaminated water or making contact with infected people or animals can cause infection in humans.

In New Zealand, there are three main types of waterborne pathogens that cause gastroenteritis: bacteria (e.g. *Campylobacter*, *Salmonella, Shigella, Yersinia* and toxigenic *Escherichia coli*), protozoa (e.g. *Giardia* and *Cryptosporidium*) and viruses (e.g. enteroviruses and noroviruses, and Hepatitis A virus, although this does not cause gastrointestinal disease). Apart from enteroviruses and noroviruses, humans diagnosed with disease caused by these pathogens are notifiable to the national health surveillance system.

Of the *ca*. 17,000 cases of gastroenteritis that were notified to the national health surveillance system in 2002, only a small proportion of people are likely to have contracted the illness through ingestion of pathogens in drinking water. In deriving an estimate for the prevalence of waterborne disease we distinguish between endemic and epidemic disease. Endemic disease is the background level of disease that occurs sporadically within a region and which has no known focus, whereas epidemic disease is associated with disease outbreaks or clusters with a common focus in time, place or vehicle of infection. Epidemic disease is particularly important for waterborne transmission, caused by the intermittent contamination of drinking water sources that will be almost universally consumed by the people served by that water supply.

The nature of outbreaks dictates that they are more likely to be detected, and a cause established, than endemic disease. Indeed, outbreaks that affect large numbers of people or those with severe health consequences (*e.g.* Milwaukee (MacKenzie *et al.*, 1994), Walkerton (Hrudey *et al.*, 2003)) have tended to drive changes in policy and water treatment practices. However, it is widely recognised that outbreaks comprise only a small fraction of notified disease, with endemic cases generally far outnumbering outbreak cases.

Another issue that needs to be understood before comparing waterborne disease rates is that notified diseases comprise only a fraction of actual disease cases. There are several reasons why this is so:

- Not all people who have an infectious disease visit a medical practitioner (gastroenteritis is a self limiting disease and most people recover without medical intervention);

- Not all cases are diagnosed by the medical practitioner.
- Not all cases present a specimen for laboratory confirmation.
- Not all diagnosed cases are reported to the disease surveillance system.

The proportion of cases that are captured by the notification system is not likely to be consistent for all diseases or population groups. One would expect the proportion of notified cases to be higher for diseases with more serious consequences. Similarly, some sections of the community (*e.g.* infants) are more likely to present to a medical practitioner and therefore are more likely to be represented in notified disease statistics.

A study funded by the New Zealand Food Safety Authority, and currently being undertaken by ESR, will determine the overall prevalence of acute gastrointestinal disease in New Zealand, and shed light on the factors influencing reporting at the GP and laboratory levels. Results are expected in mid-late 2007.

Overseas studies show that acquired immunity through routine exposure to low levels of pathogens probably contributes to the true levels of infection being masked (Neumann *et al*., 2005; Hunter and Quigley, 1998). Communities routinely exposed to low levels of pathogens, such as communities on good quality groundwaters, were reported to have a higher attack rate during outbreaks compared with communities with treated surface waters from sources of poor quality (Craun *et al*., 1998). Serological studies support the conclusion that some communities may be exposed to pathogens to a greater extent than expected. Frost *et al*. (2002), found that a greater portion of individuals in a community receiving drinking water from a river contaminated by animal and human waste was positive for *Cryptosporidium parvum* antigens than was the case in a community receiving uncontaminated groundwater. The development of immunity within a community because of poor water quality may provide the community itself with some resistance to waterborne disease, but offers no protection to visitors and tourists (Neumann *et al*., 2005). However, this has not been included in the estimates of disease burden made in this report because no estimates are available regarding the magnitude of this effect.

While it is the pathogens in water that cause waterborne disease, the safety of drinking-water is assessed by the prevalence and concentration of faecal indicator bacteria. The main reason for this is that there are a large variety of pathogens and it is impractical to test for them all routinely. The World Health Organisation (WHO) has developed drinking-water quality guidelines (WHO, 2004), in which the minimum microbiological quality is set as an absence of faecal indicator bacteria (*i.e. E. coli* or faecal- or thermotolerant coliforms) in a 100mL sample of drinking-water. Bacteriological transgressions occur when these bacteria are detected in any 100mL water sample. The drinking-water standards/guidelines of most developed countries, including New Zealand, use the same limit for bacteriological transgression. There is broad international consensus for the minimum bacteriological drinking-water standard of <1 *E. coli*/100mL or <1 FC/100mL, which is the standard applied in New Zealand.

3 New Zealand Outbreaks

An outbreak is defined as two or more cases linked by a common exposure or source. The Manual for Public Health Surveillance in New Zealand[1] states that the following types of outbreaks should be reported:

- Two or more cases linked to a common source, in particular where the common source is exposure at a common event, food or water dispersed in a community, an environmental source, or a source in an institutional setting.
- A community-wide or person-to-person outbreak (except where the source has become well established as a national epidemic and reporting it as a discrete event no longer serves a useful purpose).
- Any other situation where outbreak investigation or control measures are being used or considered.

There have been a number of outbreaks that have been documented in New Zealand in which drinking-water has been implicated as the source of infection. Four strategies were employed to locate information about these.

1. Computer literature searches using the search terms: New Zealand AND outbreak AND water*:

- PubMed (all records to 2006) search revealed 23 articles, of which eight could have been about drinking-water-borne outbreaks of infectious enteric disease.
- Scopus (1960 – 2006) search revealed 20 articles, of which 8 could have been about drinking-water-borne outbreaks of infectious enteric disease.
- Web of Knowledge (1998 – 2006) search revealed 22 articles, of which three could have been about drinking-water-borne outbreaks of infectious enteric disease.
- Index New Zealand to search New Zealand non-journal publications resulted in four articles, of which two were about drinking-water-borne outbreaks of infectious enteric disease.

2. Examination of all issues of Communicable Diseases New Zealand (CDNZ), New Zealand Public Health Report (NZPHR) and New Zealand Public Health Surveillance Report (NZPHSR) for reports of waterborne outbreaks. CDNZ was published monthly from July 1991 – December 1993, NZPHR was published between 1994 and September 2002, and NZPHSR has been published quarterly since late 2004.
3. Examination of the notified disease annual reports produced by ESR from information on notified cases sent by local Public Health Units (PHUs) to the EpiSurv database. In particular the summaries of outbreaks from the dedicated EpiSurv module were consulted. Further details on specific outbreaks were obtained from the EpiSurv outbreaks dataset.
4. Directly requesting Health Protection Officers (HPOs) to supply details of any waterborne outbreaks that they investigated.

The waterborne outbreaks identified by the above means are listed in Table 1.

[1] ESR. (2005). Manual for Public Health Surveillance.

The likelihood of these incidents being of waterborne origin is assessed using the strength of association between clusters of human illness and water as proposed by PHLS (1996) using a scheme based upon epidemiological and microbiological evidence as follows:

Microbiology/water quality:

A. Pathogen identified in clinical cases also found in water.

B. Water quality failure and/or water treatment problem of relevance but outbreak pathogen not detected in water.

Epidemiology:

C. Evidence from an analytical (case-control or cohort) study demonstrates association between water and illness.

D. Descriptive epidemiology suggests that the outbreak is water related and excludes other obvious alternative explanations.

Using the microbiology/water quality and epidemiological evidence categories above, the strength of association for the outbreaks located during the search was summarised according to the following criteria:

strongly associated - if (A+C) or (A+D) or (B+C);

probably associated - if (B+D) or C only or A only;

possibly associated - if B only or D only.

Table 1 Waterborne Outbreaks in New Zealand 1984 - 2006

Incident	Causal agent	Cases	Strength of association	Reference
Queesntown, 1984	unknown	(3,500)	B, D	Thorstensen, 1985
Ashburton, 1986	*Campylobacter*	19	B, D	Brieseman, 1987
Canterbury, 1990	*Campylobacter*	42	B, C	Stehr-Green *et al*., 1991
Havelock North, 1991	*Campylobacter*	12	B	M Hart, Health Care Hawkes Bay, *pers. comm*.
Northland, 1992	HAV	30	B	Calder & Collison, (1992)
Lonsdale Park, Northland, 1992	*Campylobacter*	14	B	Jarman & Hennevald (1993)
Waimate, 1992	*Campylobacter*	?	B	R Parr, Crown Public Health, Timaru, *pers. comm*.
Dunedin	*Giardia*	50*	B, C	Fraser *et al*., 1991
Hawkes Bay, 1992	*Campylobacter*	97	B, C?	CDNZ 92(1):11-12
Auckland, 1993	*Giardia*	34	B	Thornton *et al*., 1993
Raurimu, 1994	*Campylobacter*	16	B	D Vince, Ruapehu District Council, *pers. comm*.
Fairlie, 1994	*Campylobacter*	6	B	R Parr, Crown Public Health, Timaru, *pers. comm*.
holiday camp, 1995	gastroenteritis	*ca* 100	B, D	A Bichan, Hutt Valley Health, *pers. comm*.
Ashburton, 1996	*Campylobacter*	19 (33)	B, D	Holmes, 1996; Lees, 1996; R Parr, Crown Public Health, Timaru, *pers. comm*.
Mt Hutt, 1996	Norovirus	59	B, D	Brieseman *et al*., 2000
Auckland, 1996	*Salmonella typhimurium* 1	2	A, D	Simmons & Smith, 1997
Mt Arthur, 1996	suspected viral gastroenteritis	6	B, C	M Molloy, Nelson-Marlborough Health, *pers. comm*.
Denniston, 1996	*Giardia*	4	B, D	C Bergin, Crown Public Health, *pers. comm*.
Wainui, 1997	*Campylobacter*	6 (67)	A, C	Bohmer, 1997
Waikato district, 1997	*Cryptosporidium*	9 (170)	B, D	D Sinclair, MOH, Health Waikato, pers. comm.
Tauranga district, 1997	*Cryptosporidium*	?	B	TM Fowles, East Bay Health, *pers. comm*.
Te Aute College, 2001	*Campylobacter*	137	A, D	Inkson, 2002.
Banks Peninsula, 2004	*Shigella*	5 (18)	B, D	Morrison & Smith, 2005
camp near Nelson, 2004	*Campylobacter*	3 (13)	B	Todd, 2005
Cardrona skifield, 2006	Norovirus	218	A, D	D Bell, MOH, Public Health South, *pers. comm*.

Further details are given in Appendix 1 for each outbreak that was investigated and for which such information exists. A few of these outbreak investigations have resulted in journal publications; these are reviewed more formally. However, these represent a small fraction of the waterborne outbreaks that were recorded in EpiSurv, the majority of which were not reported in sufficient detail to be more fully described.

In conclusion, the information in Table 1 and Appendix 1 shows that there is ample evidence that waterborne GID outbreaks occur in New Zealand, particularly in areas served by drinking-water supplies that are untreated or inadequately treated.

3.1 Outbreaks as a proportion of total enteric disease

Overseas

Reports from overseas provide estimates of outbreak numbers, and the numbers of associated cases of illness, but very few estimates of the levels of endemic disease are available. Morris and Levin (1995) have estimated that each year in the USA there are approximately 7.1 million cases of mild-to-moderate infections, 560,000 moderate-to-severe cases and 1,200 deaths attributable to waterborne disease, but they emphasise the inadequacy of the available data.

Comparison with statistics from the USA Centres for Disease Control (CDC) Morbidity and Mortality Weekly Report (MMWR) annual summaries shows that reported outbreaks make up only a fraction of the total cases of waterborne disease. The MMWR annual summaries over the years from 1986 to 2002 (the most recent year for which a summary is available) report an average of 12 outbreaks and an average of approximately 28,000 cases of illness per year. This latter figure drops to *ca*. 3,000 cases when the contribution from the massive Milwaukee outbreak is removed from the dataset. Cases from outbreaks, retaining those from Milwaukee, therefore constitute approximately 5% of the moderate-to-severe cases estimated by Morris and Levin.

New Zealand

Details of the number of outbreaks of enteric disease and outbreak cases in New Zealand are reported each year by ESR (Thornley *et al*., 2002; Boxall & Ortega, 2003; ESR, 2004; ESR, 2005; Perera, 2006). The number of notified cases of enteric diseases is also reported annually (Sneyd *et al.*, 2002; Sneyd & Baker, 2003; ESR, 2004; ESR, 2005, 2006). The summary data contained in the following table was sourced from the reports listed above.

Table 2 Total notified and outbreak cases of enteric disease

Year	No. of reported outbreaks	Enteric outbreak cases	Notified enteric disease cases
2001	369	2,095	17,098
2002	317	2,783	18,782
2003	332	2,603	20,337
2004	314	3,974	17,549
2005	338	2,264	18,687

Enteric diseases comprise: campylobacteriosis, cholera, cryptosporidiosis, giardiasis, hepatitis A, paratyphoid, typhoid, salmonellosis, shigellosis, yersiniosis, VTEC/STEC disease and non-specific gastroenteritis.

It should be noted that most outbreak-related cases are not notified individually in EpiSurv but are estimates made by the DHB operatives who are managing the outbreak investigation. Consequently, while it is not possible to derive a numerical

measure of the proportion of cases that are related to outbreaks from these data, Table 2 does show that outbreak cases comprise a small fraction only.

3.2 Estimate of numbers affected by waterborne outbreaks in New Zealand

Table 3 contains a summary of the number of waterborne outbreaks of enteric disease and the number of cases involved in waterborne outbreaks that are reported each year to ESR (Thornley *et al*., 2002; Boxall & Ortega, 2003; ESR, 2004; ESR, 2005; Perera, 2006).

Table 3 Waterborne enteric disease – cases associated with outbreaks

Year	Total enteric outbreaks	Waterborne outbreaks	Enteric outbreak cases	Waterborne outbreak cases	% outbreak cases
2001	369	22	2,095	370	17.6%
2002	317	6	2,783	18	0.6%
2003	332	7	2,603	36	1.4%
2004	314	22	3,974	116	2.9%
2005	338	27	2,264	184	7.6%

The data in Table 3 show that the proportion of outbreak cases that are listed as being waterborne in the EpiSurv Outbreaks database varies greatly from year to year. In the five-year period 2001-2005 waterborne cases comprised between 0.6% and 17.6% of all reported outbreak cases with a mean of 6%. Table 4 presents reported waterborne outbreak statistics available for a number of countries with New Zealand data included for comparison.

Table 4 Waterborne disease outbreak statistics for a number of countries

Country	Period	No. of outbreaks	No. of cases	Outbreaks /year	Average Cases/year	Cases/ outbreak	Pop. (M)	Annual Cases/10^5
United States [1]	1986-2002	212	459,949	12.5	27,056	2,170	285	9.5
United States (Milwaukee omitted) [1]	*"*	*211*	*56,949*	*12.4*	*3,350*	*270*	*"*	*1.2*
Canada [2]	1974-2001	288	-	10.3	-	-		
Sweden [3]	1975-1984	32	11,847	3.2	1,185	370	8	14.8
Israel [4]	1976-1997	130	23,787	5.9	1,081	183	6	18
England and Wales [5]	1992-1995	19	1,638	4.8	410	86	65	0.6
New Zealand [6]	2001-2005	84	724	16.8	145	9	4	3.6

References: 1 CDC MMWR Annual Summary reports; 2 Schuster *et al.*, 2005; 3 Andersson & Stenstrom, 1986; 4 Tulchinsky *et al.*, 2000; 5 Furtado *et al*., 1998; 6 This report.

The average number of reported waterborne outbreaks in New Zealand per annum is comparable to that for other countries. The average is closer to the two North American entries than the averages from Europe and Israel, which may indicate similar effectiveness of national surveillance systems, or that North American supplies have shortcomings similar to those present in New Zealand systems.

The cases:outbreak ratio provides an indication of the size of communities in which outbreaks are occurring, or if larger communities are involved, the fraction of the population affected. The effect of the single outbreak in Milwaukee can be seen from this ratio. New Zealand's ratio is very low, consistent with small supplies being the primary source of outbreaks, and possibly a greater proportion of small communities in New Zealand. Data from the USA implicate protozoa as important pathogens in waterborne disease outbreaks. In five of the eight MMWR annual summaries covering the years from 1986 to 2002, protozoa have been responsible for 50% or more outbreaks with identifiable, non-chemical causes. Overall, 49% of outbreaks between 1986 and 2002 for which there was a known non-chemical cause were due to protozoa. *Cryptosporidium* and *Giardia* were the two most frequently identified causative agents of outbreaks during this period. The information in Table 1 indicates that while outbreaks due to protozoa do occur in New Zealand, amongst the reported waterborne outbreaks *Campylobacter* is a more frequently identified causative organism.

The important part the protozoa play in waterborne outbreaks in the USA may well be linked to their greater resistance to treatment processes. In New Zealand, chlorine is the most widely used disinfectant, and is capable of inactivating *Giardia* provided the dose is adequate. However, lapses in maintenance of the chlorine level weaken this barrier to the organism. *Cryptosporidium*, on the other hand, is resistant to chlorine and must either be treated with a more powerful disinfectant, or physically removed from the water. To protect consumers against these organisms adequate treatment systems must be in place and they must be performing at a satisfactory level. Achievement of these two requirements in New Zealand's drinking-water supplies is the purpose of Section 5 of the DWSNZ:2005.

4 Endemic waterborne disease

Deriving a reliable estimate of the national burden of endemic waterborne disease in New Zealand has hitherto not been attempted and is the subject of a current research project. However, the burden of endemic waterborne enteric disease is the focus of international attention at present, with the 2006 supplementary issue of the Journal of Water and Health being devoted to the subject. In this issue the various methods that can be used to estimate the risk or burden of waterborne enteric disease were described and the published literature reviewed:

- Observational studies (disease surveillance or ecologic surveys)
- Analytical epidemiological studies (cohort, case-control etc.)
- Risk assessment.

Craun & Calderon (2006) reported on a workshop held in the USA comprising many of the national waterborne disease experts. The workshop evaluated in detail the two main approaches used to attribute sources of infection, and hence estimate the burden of waterborne disease.

Epidemiological ("top down") approach

The epidemiological approach uses information from the human population to derive sources of infection for cases. This approach utilises various types of epidemiologic study, such as case-control and cohort studies, to inform the attribution of sources of infection.

Risk assessment ("bottom up") approach

The risk assessment approach uses information about the prevalence of hazards in potential sources of infection (*e.g.* waterborne microbial hazards) with consumption data to determine exposures to estimate the risk of contracting waterborne GID.

Both of these approaches could be informed by microbial risk assessments and GID surveillance data, particularly regarding the relative importance of the various pathogens that cause waterborne GID. However, risk estimates based on pathogen-specific data will under-estimate the overall risk attributable to drinking-water because the total risk comprises the risk from all of the waterborne pathogens present in the drinking-water. The workshop recommended that microbial risk assessments be carried out for the pathogens most commonly associated with waterborne outbreaks (*i.e. Campylobacter*, *Cryptosporidium*, *E. coli* O157, *Giardia*, norovirus and *Shigella*).

Disease surveillance data can be used to estimate the burden of GID and possibly estimate GID attributable to various exposures such as food and drinking-water. The workshop recommended that waterborne GID be estimated using a method similar to that used by Mead *et al*. (1999) for the US population. Estimates for New Zealand using this approach are described in Section 4.2.

The following important data gaps were identified by the workshop: identification of pathogens causing endemic disease, development of a scheme to categorise water systems in terms of specific risks, and risks associated with sensitive sub-populations, groundwater, distribution system contamination, individual household supplies and secondary transmission of disease.

Following the risk assessment approach, Messner *et al*. (2006) estimated the mean incidence of GID in the USA that was attributable to drinking-water to be 0.06 cases per person-year (95% credible interval of 0.02 – 0.12), which represents 8.5% of all GID cases in the population served by community water supplies.

Following the epidemiological approach, Colford *et al*. (2006) estimated the risk of GID attributable to drinking-water in the USA to be 12% (median AR%) for the immunocompetent population. The risk was lower for supplies using groundwater than surface water.

The annual incidence of GID in the US has been estimated as 6,000 per 100,000 population (Messner *et al*., 2006).

4.1 Evidence of endemic waterborne enteric disease in New Zealand

There have been a number of studies that have investigated the relationship between drinking-water quality and GID in New Zealand.

Duncanson *et al*. (2000) conducted an ecological study to examine the relationship between the incidence of notified cryptosporidiosis and the microbiological quality of

the cases' community drinking-water supply, as estimated by the drinking-water grading. The lowest mean rates of notified cryptosporidiosis were observed in communities served by drinking-water with completely satisfactory public health grading. There was an increased trend in cryptosporidiosis rates as the drinking-water grades declined in quality (refer to Figure 1). While this was an ecological study and therefore unable to attribute causation, the study concluded that better drinking-water quality is likely to be associated with health benefits at the population level.

Figure 1. Cryptosporidiosis rate in relation to drinking-water grade.

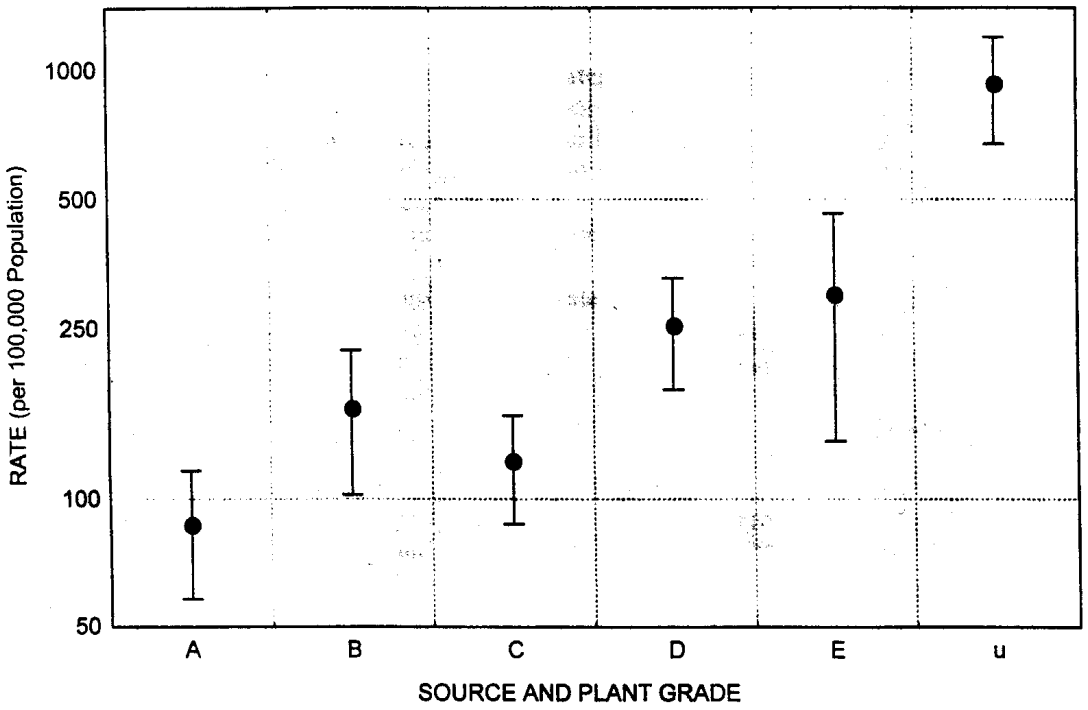

Fig. 1. Rates of notified cryptosporidiosis per 100,000 population in New Zealand June 1996–August 1998 by source and plant component of the public health grading of community drinking water supply where A = very satisfactory, B = satisfactory, C = marginal, D = unsatisfactory, E = very unsatisfactory and u = ungraded. Log scale on Y axis. Error bars indicate standard error. Data source NZ Ministry of Health.

Sloan (1990) conducted a correlational study in Whangarei to determine whether the source of *Giardia* in the city was waterborne (Sloan, 1990). The incidence of giardiasis in the population served by the untreated water supply was higher than in the remainder of the city that was served by filtered and chlorinated water (relative risk 2.77, $CI_{95\%}$ 1.38 - 5.56). *Giardia* cysts were subsequently detected at concentrations of between 13 and 18 cysts/100L in the water from the water in the untreated distribution zone.

Fraser & Cooke (1991) conducted a cohort study to determine the incidence of laboratory-confirmed giardiasis in Dunedin. The incidence of giardiasis was higher (RR = 3.3; $CI_{95\%}$ = 1.1 – 10.1) in the area of Dunedin where the drinking-water contained no effective protozoal treatment (*i.e.* 23 μm filtration and chlorination only) compared with the remainder of the city, whose water was fully treated (*i.e.* coagulation, flocculation, dual media filtration and chlorination).

Ikram *et al*. (1994) conducted a case-control study on 100 laboratory-diagnosed campylobacteriosis cases notified in Christchurch during the 1992-3 summer and controls of the same age and gender selected by the same medical practitioner. Drinking water from a non-urban supply (OR = 2.70; $CI_{95\%}$ = 0.89 – 8.33) was among the risk factors although was not significant at the 95% confidence level. However, drinking water from another town supply was not a risk factor (OR = 0.56; $CI_{95\%}$ = 0.12 – 1.85).

A case-control study conducted on 621 campylobacteriosis cases notified in four urban centres between June 1994 and February 1995 and controls matched for age group, gender and area (Eberhart-Phillips *et al*., 1997). Two drinking-water exposures were identified as risk factors: having a home roof water supply (OR = 2.20; $CI_{95\%}$ = 1.04 – 4.62) and drinking non-city water outside the home within 10 days of the onset of symptoms (OR = 1.63; $CI_{95\%}$ = 1.17 – 2.27).

A case-control study was conducted on under-five year-olds in the Auckland region using 69 giardiasis cases and 98 controls (Hoque *et al*., 2003). Use of drinking-water other than from the Auckland metropolitan supply (adjusted OR = 8.6; $CI_{95\%}$ = 3.5 – 21.2) and consumption of water away from home (adjusted OR = 4.7; $CI_{95\%}$ = 2.2 – 10.1) were associated with increased giardiasis. A population attributable risk (PAR) of 57.8% was reported for the consumption of drinking-water other than from the Auckland metropolitan supply.

Ball *et al*., (2003) conducted a multi-centre cohort study to investigate the relationship between drinking-water quality and GID in primary school children. Children attending schools served by a community drinking-water supply of poor microbiological quality (Dd – Ee grades) were found to have a higher risk of GID (RR = 1.26; $CI_{95\%}$ = 0.97 – 1.63) than those served by microbiologically compliant (Aa – Cb grades) supplies.

Close *et al*., (submitted) compared the number of cases of GID from the region of Canterbury encompassing dairying within major irrigation schemes with two comparison groups: those resident in dairying areas without irrigation (CG1) and those resident in the rest of Canterbury (CG2). An increase in the age-standardised rates of notified campylobacteriosis (RR = 1.51; $CI_{95\%}$ = 1.31-1.75) and cryptosporidiosis (RR = 2.08; $CI_{95\%}$ = 1.55-2.79) was shown relative to CG1. An increase in the age-standardised rates of notified campylobacteriosis (RR = 1.51; $CI_{95\%}$ = 1.33-1.72), cryptosporidiosis (RR = 5.33; $CI_{95\%}$ = 4.12-6.90) and salmonellosis (RR = 2.05; $CI_{95\%}$ = 1.55-2.71) was shown relative to CG2.

In conclusion, drinking water of poor microbiological quality, low public health grading or that receives inadequate treatment has been demonstrated to contribute to the burden of GID in New Zealand. While no epidemiological studies have been carried out to derive the national burden of GID from contaminated drinking-water, population attributable risks have been derived for some communities.

4.2 Estimated burden of endemic waterborne enteric disease in New Zealand

Two estimations of the burden of endemic waterborne GID in New Zealand are available. These are described below, with preliminary estimates from additional work in progress.

"Top down" approach.

An estimate of overall infectious intestinal disease burden, and the foodborne component, was published in 2000 (Lake *et al*., 2000; Scott *et al.*, 2000), largely based on notified diseases data from 1998 and hospitalisation data from 1997. This study estimated the ratio between these reported cases, and the unreported community cases by using ratios derived from a large prospective UK study (Wheeler *et al*., 1999). This study investigated a large cohort to determine the overall numbers of gastrointestinal diseases cases, obtained faecal samples from all of them to try to identify the pathogen, and identified the cases which did visit their GP as a result of the illness. This was considered to be the most rigorous study available, and the most applicable to the New Zealand situation. The UK study estimated that salmonellosis and campylobacteriosis were under-reported by factors of 3.2 and 7.6 respectively, and an intermediate factor of five was used for yersiniosis and shigellosis. For more severe illnesses, such as those caused by infection with *Listeria monocytogenes*, Lake *et al.*, (2000) assumed that no unreported cases occurred. The incidence of non-notifiable enteric diseases, especially those caused by viruses, were estimated from either the UK study or outbreak data.

Overall, the annual prevalence of ten infectious intestinal diseases (chosen because foodborne transmission makes up a substantial part of their aetiology) was estimated as 199,000 cases. By using estimates of the percentage attributable to foodborne transmission, the number of foodborne cases was estimated as 119,000.

To estimate the overall prevalence of infectious intestinal disease, additional estimates for illnesses caused by pathogens not usually transmitted by food, such as *Cryptosporidium, Giardia*, and several other types of virus (usually transmitted person to person), were added. This gave an additional estimated approximately 140,000 cases. Finally, the UK study found that for over half the ill people in the cohort, no pathogen was identified in the faecal sample. If the same is true in New Zealand, then there was estimated to be an additional approximately 487,000 people in this category. Overall, there could be up to 823,000 cases of infectious intestinal disease each year in New Zealand. Although this number was based on many substantial assumptions, it represented the best estimate at the time.

This approach was adapted by Rosevears (2004) to estimate the number of cases and burden of illnesses associated with drinking-water. Using notification data from 2004 for the illnesses: campylobacteriosis, infection with *E. coli* O157, cryptosporidiosis, giardiasis, salmonellosis, yersiniosis, toxins (algal) and viruses (including Hepatitis A), this study used the same multipliers and further information to estimate the number of cases of these illnesses per annum, and then assigned a percentage to waterborne transmission (the highest was cryptosporidiosis which was assigned as being 30% waterborne). This report estimated the burden of waterborne illness to be approximately 18,000 cases, the largest component of which was *ca.* 12,000 cases of campylobacteriosis.

The notified enteric disease risk factor information reported for cases and held in EpiSurv has been examined by Ball (2006) in an attempt to derive the burden of GID from drinking-water. The proportion attributed to drinking-water was estimated to be between 4.1% and 37%, although the reliability of this method is questioned by the author because of the erratic nature of risk factor reporting in EpiSurv. However, the lower estimate of 4.1%, which was based on notified cases for which drinking-water

was the likely or sole risk factor, may serve as a conservative estimate of the national burden of drinking-waterborne GID.

Using the estimate of 823,000 for the annual number of all GID cases derived by Lake *et al.* (2000) and the lowest estimate for the risk attributed to drinking-water derived by Ball (2006) of 4.1%, the annual number of GID from drinking-water can be estimated at 33,743. Using the 2000 census population estimate of 3,842,800 the annual rate of GID from drinking-water can be estimated at 878/100,000.

For comparison, using the rate of notified enteric disease for 2000 of 378 cases/100,000 (Sneyd *et al*., 2002), 4.1% - 37% waterborne represents 15.5 – 140 cases/100,000. The main point of note is that the actual disease rates are considerably higher than is indicated by the disease notification system.

"Bottom up" approach.

Close *et al*., (2006) used a Monte Carlo simulation-based risk assessment to derive the risk of waterborne *Campylobacter* infection from drinking untreated drinking-water from shallow unconfined groundwater underlying dairying with border-strip irrigation in Canterbury. The probability of infection on any given day during the irrigation season was estimated to be between 0.0042 and 0.0050, which translates to an average risk of infection of 54% - 60% for the irrigation season. This estimate was backed up with epidemiological evidence from notified GID in the same district (Close *et al*., submitted).

A preliminary risk assessment has been carried out using the pathogen concentration data from the five recreational water sites in the survey conducted for the Freshwater Microbiology Research Programme that also served as source waters for community drinking-water supplies (McBride *et al*., 2002). Concentrations of *Salmonella*, *Campylobacter*, *Giardia* cysts and *Cryptosporidium* oocysts were obtained for samples from each of the five sites at fortnightly intervals for 15 months. The @Risk software package (Pallisade Corp., NY) was used to obtain best-fit probability distributions for the pathogen concentrations. However, only *Campylobacter* was present in a sufficiently high proportion of samples for a robust distribution to be fitted, this being a logistic distribution with parameters of $\alpha = 78.053$ and $\beta = 155.13$.

Using a fixed value for daily (unboiled) drinking-water intake of 1 litre per person per day, and probability of infection (P_d) by *Campylobacter* (β-Poisson infectious dose model: $\alpha = 0.145$; $ID_{50} = 986$), the mean risk of infection was estimated to be 0.2289, per person day for untreated water. This is converted to an annual risk of infection by application of the following formula: $P = 1-(1-P_d)^{365}$.

However, the risk of waterborne GID will decrease as an increasing level of water treatment is applied. Estimates of the risk of waterborne GID corresponding to treatment that removes 1 to 5 logs (*i.e.* 90% to 99,999%) of the pathogens present in the source water are shown in Table 5.

Table 5 Estimated risk of waterborne infection following various water treatment efficacies (expressed as log removals)

Water Treatment	Estimated risk of *Campylobacter* infection	
	Mean daily individual risk	Annual risk (per 100,000)
None	0.2289	100,000
1 log	0.098	100,000
2 log	0.0221	99,440
3 log	0.0027	41,836
4 log	0.0003	5,299
5 log	0.0001	543

While the risk of *Campylobacter* infection clearly diminishes as the degree of drinking-water treatment increases, it is not known how many log reductions of *Campylobacter* are achieved by conventional treatment. This is because bacteriological assessments are normally measured using indicator bacteria such as coliforms and *E. coli* rather than pathogens. Results from Nokes (2006), indicates that full conventional water treatment achieves at least a 5-log reduction of coliforms. Given that *Campylobacter* is more sensitive to chlorination than *E. coli* and other coliforms, one would expect the same treatment to achieve more than a 5-log reduction of *Campylobacter*. Without such information, it is not feasible to estimate the risk of *Campylobacter* infection from treated drinking-water.

However, an assessment can be made of the burden of campylobacteriosis from untreated registered drinking-water supplies upon the following assumptions:

- All people on such supplies will become infected at least once during the course of a year (derived from Table 5).
- 30% of *Campylobacter* infections give rise to campylobacteriosis (WHO, 2004).
- 60,930 people were served by registered community drinking-water supplies derived from surface waters that received no recognised bacteriological treatment during 2005.

From these assumptions it can be deduced that *ca.* 18,000 campylobacteriosis cases would have arisen from registered water supplies during 2005. Further campylobacteriosis cases could be expected to arise from ingestion of unregistered or individual water supplies. The estimate of a further 23,000 cases is based on the following assumptions:

- A further 460,000 people were served by unregistered or individual water supplies, which are split evenly between surface water, roof water and groundwater supplies, half of which are estimated to be untreated.

These estimates represent a fraction of waterborne GID cases because account also needs to be taken of:

- campylobacteriosis contracted via ingestion of untreated roof water and groundwater;
- campylobacteriosis contracted via ingestion of water following various drinking-water treatments and;
- other waterborne diseases.

Consequently, should the assumptions behind these estimates be accurate then the national burden of GID would be higher than estimated using the "top down" approach. However, we are not yet in a position to make a more complete estimate of the national burden of waterborne GID.

5 The Protective Effect of Drinking-water Standards

The protective effect of applying drinking-water standards has been demonstrated in Israel. Disease data from Israel over the period from 1976 to 1997 shows the impact more stringent drinking water standards can have on the percentage of total cases gastrointestinal disease attributable to drinking water. The figures are shown in Table 6.

Table 6 Total and waterborne GID in Israel

	Year				
	1976-80	1981-85	1986-90	1991-95	1996-97
Waterborne GID cases	10,084	11,457	1,986	260	-
Total GID Cases	25,576	25,523	8,983	5,770	2,632
Percentage of total cases that were waterborne	39%	45%	22%	5%	0%

More stringent water regulations, which amongst other things introduced compulsory chlorination, came into effect in early 1989.

It is not possible to conduct a New Zealand comparison analogous to that carried out using Israeli data because until this year there was no distinction made between food and water in the source of infection field in EpiSurv. However, Duncanson *et al*. (2000) demonstrated an increasing trend in cryptosporidiosis rates as the drinking-water grades declined in quality (refer to Figure 1), which suggests that the overall waterborne disease rate would decrease as the level of drinking-water treatment improved.

Experience has also shown that interventions in improving access to safe water favour the lower socio-economic population in particular, whether in rural or urban areas, and can be an effective part of poverty alleviation strategies (WHO, 2004).

6 Conclusions

There is ample evidence of waterborne disease outbreaks in New Zealand to indicate a significant risk of contracting GID from drinking-water that is untreated or inadequately treated. An average of 16.8 waterborne outbreaks (range from 6 to 27) occur annually, affecting an average of 145 cases/year (range from 18 to 370). While the largest reported waterborne outbreak affected 3,500 people (Queenstown, 1984), the size of most outbreaks is small, averaging nine cases per outbreak in 2000-2004, and is smaller than any other countries for which data are available. This probably reflects the larger contribution of water supplies serving small communities in New Zealand compared to most other developed countries. This is in keeping with the relatively poor compliance with the DWSNZ of the small community drinking-water supplies compared to that of the larger community supplies.

The occurrence of endemic waterborne gastro-intestinal disease in New Zealand has been demonstrated by a number of epidemiological studies.

Based on currently available data, estimates of the burden of endemic drinking-waterborne gastro-intestinal disease are of the order of *ca.* 18,000 and 34,000 cases per annum. Preliminary results from work in progress suggest that this is an under-estimate.

There is broad international consensus for the minimum bacteriological drinking-water standard of <1 *E. coli*/100mL or <1 FC/100mL, which is the standard applied in New Zealand.

The DWSNZ uses the WHO drinking-water guideline minimum bacteriological value of <1 *E. coli*/100mL, as do most developed countries.

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APPENDIX 1

Ashburton, 1986. During early 1986, an outbreak of campylobacteriosis was reported in Ashburton, peaking in late March with 19 cases notified within two weeks (Brieseman, 1987). Cases were distributed throughout the borough. Routine investigations were carried out on the notified cases, from which the consumption of drinking-water from the town water supply was revealed as the only common link. The Ashburton water supply was fed from several bores and a recently installed infiltration gallery on the Ashburton River. Although the water was drawn from the infiltration gallery and was served by a chlorination plant, it was normal practice in the borough to only chlorinate the water "as required", namely when the river level was high after heavy rain. The bores drew water from 3-60 m depths and were not chlorinated. Heavy rain fell on the night of 12 March, yet chlorination did not occur until 09:00 the following morning, at which time the inlet water contained a high concentration of coliforms. It is likely that the delay in the commencement of chlorination may have been contributed to the outbreak. No samples were taken of the chlorinated water, nor were water samples tested for the presence of *Campylobacter*. However, the reticulated water was observed to be quite turbid and therefore a reduction in the effect of chlorination was likely and given the large numbers of livestock present in the catchment area, the likelihood of *Campylobacter* being present in the water is high.

Canterbury, 1990. Following the occurrence of two cases of *Campylobacter* gastroenteritis at a camp in Canterbury during August 1990, 42 cases of campylobacteriosis were identified (Stehr-Green, 1991). All people present at the camp between 27and 31 August 1990 were interviewed in person or via telephone using a standard questionnaire. Cases were defined as those displaying any of the following within a week of being at camp: *C. jejuni* detected in stool, diarrhoea lasting two or more days, or four or more of the following symptoms (diarrhoea for one day, nausea, vomiting, stomach pain, fever, headache, myalgia, malaise). Symptoms did not differ much between cases who were culture-confirmed or not. Of the 116 persons present at the camp over the critical period, 99 were interviewed (a response rate of 85%), comprising 42 cases and 57 controls. Details were only reported for a three exposures and quantitatevely for only two: contact with animals and the consumption of unboiled water. Contact with farm animals and pets was reported to be similar for cases and controls (77% *cf*. 69% respectively). Consumption of ≥2 cups of unboiled water was greater for cases (100%) than controls (58%) ($p<0.01$). The camp was situated on native bush and farmland containing a range of livestock and drinking water was supplied from four springs on the property with neither filtration nor disinfection. At the time of the outbreak, the drinking water contained 1-92 faecal coliforms / 100 mL, whereas only low concentrations of coliforms were present three weeks later, which may indicate that the water quality was worse than normal at the time of the outbreak. Examination of the springs revealed two of the four had lids may have allowed surface run-off to enter the supply after recent heavy rain. The case-control study implicated contaminated drinking-water as the source of infection, a conclusion that was backed up by microbiological evidence of faecal contamination that could be explained by contamination of two of the springs by surface runoff and lack of water treatment. However, the statistical assessment was based on univariate analysis and the possible influence of confouners was not reported. Nor was an assessment of potential effect of bias. Despite an

inability to assess bias and confounding, the relative risk (derived from the paper to be 1.72, $p<0.01$), microbiological evidence of faecal contamination of the untreated drinking-water, a heavy rain event a few days prior to the outbreak and resolution following chlorination of the water supply are suggestive of the outbreak being waterborne.

Wainui, 1997. An outbreak of gastroenteritis was reported following two successive camps at Wainui on the Akaroa peninsula during January 1997 (Bohmer, 1997). The epidemic curve was consistent with a point-source outbreak. A retrospective cohort study was conducted to attempt to identify the source of infection. Information was sought about symptoms and exposures including food and water consumed at camp and outdoor activities while at camp using a telephone questionnaire. A suspect case was defined as having diarrhoea and/or vomiting and/or abdominal cramps, or nausea with associated headache and/or fever within 1-10 days of first attending the camp. Of the 149 people who had attended the camp over two consecutive weeks, 109 (73.2%) were contacted, 67 (the suspected cases) showed symptoms of campylobacteriosis and *Campylobacter* was detected in five (the confirmed cases) of the six of the stool specimens submitted. Noroviruses were also detected in the stool specimens of one confirmed and one suspected campylobacteriosis case. The attack rate was 61.5% over the two weeks. Consumption of camp drinking water was identified as the likely vehicle of infection (relative risk 1.51, $CI_{95\%}$ 1.07-2.12). The drinking-water supply was drawn from a bore without treatment and contained 95 faecal coliforms / 100 mL but was not tested for pathogens. However, *Campylobacter* was isolated from the stream nearby and back-flow of stream water into the camp drinking-water supply was observed during periods of high demand.

Retrospective studies always have the potential to introduce recall bias. However, the effect normally tends to broaden the confidence interval rather than change the relative risk unless the participants have some preconception that one of the exposures being investigated caused the disease, in which case the Hawthorne effect may cause the relative risk of that exposure to be over-estimated. The report did not address the possible influence of confounding and no results were given regarding the other exposures. However, it is not unusual for articles describing outbreaks to focus on the cause and public health outcomes rather than the technical details in this type of publication.

Queenstown, 1984. The largest recorded waterborne outbreak in New Zealand occurred in Queenstown during the spring of 1984, when an estimated 3,500 people were affected (Thorstensen, 1985). There is little clinical information available, except that patients suffered a range of gastrointestinal symptoms that were in most cases fairly mild and persisted 2-3 days, although a number of people were hospitalised. The extent of the outbreak was best followed by school attendance, where almost half the pupils were absent at the height of the outbreak. The cause of the outbreak was thought to be a sewer overflow that discharged sewage into a creek that entered Lake Whakatipu within 200 m of the intake to the public water supply, which was not adequately treated. The outbreak abated when the sewage overflow was noticed and remedied and the water supply was heavily chlorinated. Faecal coliforms were detected in all water samples taken at the time and, given this scenario, it is likely that a range of waterborne pathogens were involved.

Auckland district, 1997. A familial outbreak of waterborne salmonellosis over a ten-month period was described by Simmons & Smith (1997). All four family members

suffered from gastroenteritis, with *Salmonella typhimurium* phage type 1 isolated from the two persons tested. There was no obvious foodborne source but analysis of the household drinking-water supply revealed faecal coliforms (13/100 mL) and *S. typhimurium* phage type 1. The drinking-water supply was untreated roof water. The roof was a perch for gulls and frequented by rats, possums and the family cat, which often defecated on it.

Mt. Hutt, 1996. A protracted outbreak of gastroenteritis occurred at the Mt. Hutt ski field in the 1996 winter in which several dozen cases were investigated and which probably affected many more than the 58 cases in 11 parties reported (Brieseman *et al*., 2000). Based upon the illness and exposure data reported, an odds ratio of 109 and a $CI_{95\%}$ of 5 – 76 can be calculated. Five faecal specimens from two parties were tested and all contained noroviruses. Epidemiological investigation implicated the consumption of drinking water as the source of infection. All water samples tested were free of faecal coliforms but F-RNA bacteriophage was isolated from one of the water filters and enterovirus was detected in another, indicating faecal contamination. The water supply was taken from a river downstream of the sewage discharge and stored for several weeks in a frozen lake before being used to supplement the reticulation system. A site visit revealed that he cartridge filters had been removed and the UV was inoperative. It was postulated that the faecal indicator bacteria in the source water were removed by the protracted storage and UV treatment, whereas the long-lived pathogens such as viruses survived. The detection of more than one strain of norovirus suggests that the outbreak was not caused by a single point source but is in keeping with a contaminated drinking-water supply.

Hawke's Bay, 1992. An outbreak of campylobacteriosis was reported in a youth camp in Hawkes Bay in January 1992 affecting 97 people (CDNZ 92(1):11-12) Anon. (1992). No risk factors were identified except that the people who developed symptoms consumed more water each day than those not affected (p=0.01). The source of drinking water at the camp was untreated bore water and was found to contain between 2 and 11 faecal coliforms /100mL, although *Campylobacter* was not detected in the two bore water samples tested subsequently.

A number of incidents have been reported to me during the course of this investigation.

Waimate, 1992. A cluster of campylobacteriosis cases were reported in Waimate in South Canterbury in March 1992 (R Parr, Crown public Health, Timaru, *pers. comm*.). Anecdotal evidence suggested the outbreak was more widespread, with chemists reporting a high demand for anti-diarrhoea medication over the same period, and 40% of children absent from kindergarten with gastro-intestinal symptoms. While pets and food were not ruled out as sources of infection, the water supply was suspected as the chlorinator was inoperative between at least from 24-27 February. Subsequent analysis of the source water before chlorination resulted in faecal coliform concentrations of 92 and >240 /100mL. However, the water was not tested for *Campylobacter*.

Fairlie, 1994. Six cases of diagnosed campylobacteriosis and several cases of stomach upsets occurred at Fairlie between 28 March and 15 April 1994 (R Parr, Crown public Health, Timaru, *pers. comm*.). The onset of illness was 22-30 March for most patients, and all had either consumed or cleaned their teeth in tap water. Drinking water was suspected because there was heavy rain on 19 March and the

water, which is obtained from a spring adjacent to the Opihi River, develops increased turbidity during and after heavy rain that can reduce the efficacy of chlorination.

Ashburton, 1996. There was another peak in campylobacteriosis notifications in Ashburton in February 1996, with 33 cases (only 19 of them notified) occurring between 10th and 18th February (Holmes, 1996; Lees, 1996; R Parr, Crown Public Health, Timaru, *pers. comm*.). There were no common foods or events. The only common feature was that they received drinking-water from the Ashburton town supply. On 6-8 February, during three days of heavy rain, there was chlorinator failure at the infiltration gallery that resulted in the reticulated water supply not being chlorinated for at least 24 hours during a time when the river water was turbid and probably contained a high concentration of faecal material from the surrounding agricultural areas. The onset period of most patients was consistent with an initial infection during this period. *Campylobacter* and high faecal coliform counts were detected in water sampled from the gallery intake on 4 March, following another period of heavy rain on the previous two days, which further substantiated the hypothesis of a waterborne source of this outbreak.

Lake Hawea, 1989. A single case of salmonellosis was successfully investigated by the Central Otago District Council (Dunlop, *pers. comm*.). The patient was the only one of three to drink water while on holiday at Lake Hawea camping ground on 4-5 February 1989, and was the only person to develop gastroenteritis 1-2 days later. Tap water from the campsite was tested and contained >180 faecal coliforms/100mL and *Salmonella saintpaul*, which was the same serovar isolated from the patient's faecal specimen.

Havelock North, 1991. In April 1991 a cluster of 12 campylobacteriosis cases were notified in Havelock North (M Hart, Health Care Hawkes Bay, *pers. comm*.). Investigation revealed no common source but drinking water was suspected, although faecal coliforms were not detected in routine surveillance samples. Subsequently, a potential for back siphoning was discovered where water may have entered the reticulation system via a roadside drain contamination with a high level of faecal coliforms.

Mt. Arthur, 1996. Six people in two groups became ill with gastroenteritis after drinking untreated rainwater at the Mt. Arthur hut in September 1996 (M Molloy, Nelson-Marlborough Health, *pers. comm*.). The other people who stayed at the hut and did not drink the water were not affected. Symptoms of stomach cramps, nausea, vomiting, mild diarrhoea and fever within 30 hours of consumption, with resolution the following day are consistent with viral gastroenteritis. No microbiological analysis was performed on water or clinical specimens.

Raurimu, 1994. Sixteen campylobacteriosis cases occurred in 1994 that were linked to a private non-chlorinated water supply at Raurimu (D Vince, Ruapehu District Council, *pers. comm*.). Visitors were affected more than locals. A high concentration of faecal coliforms was detected in the rainwater tank but the water was not tested for *Campylobacter*.

Giardiasis cases are generally quite sporadic and evidence is generally confined to families or small communities. In many of the cases where drinking-water was implicated as the vehicle if infection, the quality of the drinking-water was poor.

Denniston, 1996. Four cases of giardiasis from the Buller district on the west coast occurred in late February 1996 (C. Bergin, Crown Public Health, Westport, *pers.*

comm.). The common link was that all consumed water from the Denniston water supply, which is unregistered, untreated and unprotected. While the water not tested for *Giardia*, the water had a turbidity of 8 NTU.

Peketa A further three cases of giardiasis were reported at Peketa, where drinking water was also the common link (BW Ingram, Kaikoura District Council, *pers. comm*.). The groundwater water supply was contaminated with faecal coliforms and was discoloured.

Hutt Valley camp, 1995. An outbreak of gastroenteritis was reported at a holiday camp in October 1995 affecting approximately one hundred people (A Bichan, Hutt Valley Health, *pers. comm.*). From investigation it was determined that it was probably a common source outbreak but that a foodborne source was unlikely. The causal agent was not identified but 30 faecal coliforms/100mL were detected in the drinking water. No recurrence of the problem has been observed since the water treatment was upgraded.

Waikato, 1997. During September-October 1997, following investigation of a notified case of cryptosporidiosis, it was revealed that over 170 people in a district in Waikato had reported symptoms consistent with cryptosporidiosis over the same period (David Sinclair, MOH, Health Waikato, *pers. comm.*). *Cryptosporidium* was detected in 9/25 of faecal samples tested. From the questionnaire used to investigate the outbreak, the likely source of infection was considered unlikely to be either farm contact, common foods or person-to-person contact. However, the incident was considered to be waterborne as most cases followed two turbidity spikes in the drinking-water supply. The first of these peaked at 0.4 NTU and was possibly associated with a filter backwash at the treatment plant. The second of these spikes occurred two weeks after the first and occurred over a three-day period during which time the turbidity was approximately 0.6-0.8 NTU. This corresponded to reports of taste problems and discoloration of the water supply and was not related to filter backwash but backflow from farms in the area was suspected. Analysis of the chlorinated supply revealed no faecal coliforms and no *Cryptosporidium* oocysts were detected.

Tauranga district, 1995. Late in 1995, a possible cryptosporidiosis outbreak was notified in the Tauranga district following a notification and other suspected cases in a school in the area (TM Fowles, Toi Te Ora Health, *pers. comm.*). Drinking water was supplied from a bore and was untreated except for an aged filter that was totally clogged at the time of sampling. Faecal coliforms were not detected in the bore water but counts up to 54/100 mL were observed in the storage tank. The storage tank was open and was frequented by birds.

Hawke's Bay school, 2001. A campylobacteriosis outbreak occurred at a boarding school in Hawke's Bay during May 2001 (Inkson, 2002.). Outbreak questionnaires were completed by 182 (62%) of the 295 people at the school of whom 137 (75%) reported gastrointestinal symptoms. *Campylobacter* was detected in the stools of two students. No significant exposures were identified. However, *Campylobacter* were isolated in both pre- and post-treated drinking-water and from the faeces of cattle which had access to the source water. The UV treatment system malfunctioned at about the time of the outbreak.

Banks Peninsula, 2004. An outbreak comprising five confirmed and 18 probable cases of shigellosis at a retreat on Banks Peninsula in 2004 was reported by Morrison

& Smith (2005). Following concerns raised by residents regarding suspected contamination of the drinking-water supply the water supply system was investigated. A break in the septic tank effluent pipe was found and was discharging effluent directly above the intake of the spring-fed drinking-water supply. The drinking-water was tested and found to contain *E. coli* but was not tested for the presence of *Shigella*. *Shigella* is likely to have been introduced to the septic tank in September by two overnight visitors to the retreat who had returned from India with diarrhoeal illness a month previously, one of whom subsequently tested positive for *Shigella*. Confirmed and suspected cases occurred from late September until mid November when the first shigellosis case associated with this outbreak was notified. While the initial cases were thought most likely to have been waterborne, person-to-person spread became more likely as the outbreak progressed.

Nelson camp, 2004. A campylobacteriosis outbreak at a self-catered camp near Nelson occurred during late December of 2004 and was reported by Todd (2005). A questionnaire was posted or emailed to the 40 other attendees of the four-day long camp seeking information about symptoms and exposures of which nine (22.5%) were returned. No statistical analyses were reported but "no obvious risk factors relating to the activities undertaken and food consumed during the camp were identified from analysis of the questionnaire data". However, the drinking-water supply was suspected. Drinking-water for the camp was obtained from a spring and was untreated other than being passed through a coarse filter before entry to three storage tanks. Spring, tap and tank water samples were tested for *E. coli* resulting in counts of <1, 2 and 11/100 mL respectively. The former two were also tested for the presence of *Campylobacter* but with negative results. It is difficult to assess whether the likelihood of this being a waterborne outbreak because of the poor response rate and the commensurate inability to assess the roles of the possible risk factors.

Lonsdale Park, 1992. A campylobacteriosis outbreak affecting 14 (29%) of children attending a camp at Lonsdale Park, Northland, was notified on 25 November 1992 (Jarman & Henneveld, 1993). A case-control study was conducted on the attendees at the camp held between 9 and 13 November. All attendees of the camp completed a questionnaire about symptoms, exposures and demographic characteristics a month after the incident. Two risk factors were identified: consumption of cereal and milk breakfast (OR 10.4, $CI_{95\%}$ = 1.17-278.1) and river water on the overnight camp (OR 4.0, $CI_{95\%}$ = 0.78-22.6). Suspicion initially fell upon the milk, was obtained from a local farmer and some of which was consumed unpasteurised. However, the odds ratio for a glass of milk was 0.9 ($CI_{95\%}$ = 0.17-4.57) and a sample of raw milk obtained later from the same supply complied microbiologically with 1984 Food Regulations. The water supplies were all highly contaminated. Faecal coliform counts in the untreated roof water camp drinking-water supply and the farm supply used at the overnight camp were 225 and 900/100 mL respectively. *Campylobacter* was not detected in any of the water or milk samples. The investigation was inconclusive but implicated raw milk and drinking-water as vehicles of infection. This outbreak illustrates the various problems that can be encountered during outbreak investigations. Recall bias may have influenced this result given the month-long delay before the questionnaire was administered; this normally widens the 95% confidence interval so may be pertinent in the interpretation of the drinking-water result. The odds ratio obtained for drinking river water was reasonably high but inconclusive according to the convention applied to the 95% confidence interval. The response rate to the questionnaire was 100% but many participants left some

questions unanswered so it may be more appropriate to apply the response rate to individual questions in this instance. The Hawthorne effect could have influenced the results by inflating the odds ratios for exposures perceived by the participants to be likely causes of the outbreak. Participant perceptions were not investigated but diseases from raw milk and untreated drinking-water have been widely publicised. However, the low odds ratio for a glass of milk suggests that the result for the cereal and milk breakfast were not much influenced by the Hawthorne effect. There were multiple water and milk, the two most likely causes of this outbreak. This adds to the complexity of the investigation and may have increased the likelihood of misclassification errors. Another common problem is that there are few microbiologically confirmed cases. In this outbreak there were six, with the remaining eight cases being attributed from symptoms alone. The proportion of microbiologically confirmed cases is higher for this outbreak compared to many outbreaks. However, the lack of laboratory confirmation means that it is quite conceivable that there was more than one pathogen involved with two or more separate sources of infection. This can only be determined by testing specimens from all cases and sub-typing the isolates, which is seldom, if ever, done. It is difficult to investigate effectively outbreaks with multiple causes in investigations such as this for which the case-control study is the most commonly used quantitative epidemiological method.

Cardrona skifield, 2006. The most recent waterborne disease outbreak occurred at the Cardrona skifield during July 2006 that has been linked to 218 staff and visitors but is likely to have affected many more people (Derek Bell, MOH, pers. comm.). The first cases were reported to have occurred on 24 July. Investigations by the public health staff revealed that the usual drinking-water supply was being supplemented with water from a stream with the intake being downstream of the septic tanks and the effluent holding pond. Furthermore, an effluent overflow was reported to have occurred 1-2 days before the first reported cases became ill. At the time of the outbreak the drinking-water supply was not registered. While water treatment comprised cartridge filtration and UV treatment, the cartridge did not comply with the requirements of the DWSNZ and the efficacy of the UV treatment was unknown. Furthermore, the water supplied to some of the staff quarters was able to bypass the treatment process. Water testing revealed excessive *E. coli* (range 7.4 – 220/100mL) in the drinking-water supply. Norovirus was detected in a number of faecal specimens tested from cases and also in a sample of drinking-water collected on 27 July, the day that the outbreak was notified. While norovirus was the primary pathogen associated with this outbreak a number of cases caused by other waterborne pathogens were also attributed to the same incident. This is consistent with drinking-water that is contaminated with sewage in which a range of human enteric pathogens can be expected.