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Unintentional house fire deaths in New Zealand 1991-1998

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A review of routinely collected health and fire service data was undertaken to describe individuals who died in house fires in New Zealand in the years 1991-1996 and the characteristics of fatal incidents from 1991 to 1998. Age-specific mortality rates were highest for adults ≥ 65 years and children < 5 years. Age-standardised rates showed a threefold increased mortality for Maori compared with non-Maori (relative risk [RR] 3.4, 95% confidence interval [CI] 2.2-5.2), and an increased risk for males compared with females (RR 1.8, 95% CI 1.3-2.4). Fatal incidents occurred most commonly in winter, and in the early hours of weekend mornings. Findings in New Zealand are consistent with overseas studies and demonstrate the importance of designing effective preventive strategies that reach population groups with the greatest risk. Published studies support the roles of public health workers in advocacy for mandatory smoke detectors and adequate housing standards, injury prevention counselling, smoking cessation and alcohol programmes, and community-based fire safety initiatives.

Residential fire incidents are the principal cause of death from thermal injury for New Zealand children¹ and adults.² Fatal fire incidents also impact on the physical and mental health of survivors.³ Fatal fire incidents are of public health interest because they result in preventable premature deaths, because population-based studies have identified effective intervention strategies, and because such incidents illustrate important issues regarding household and community safety. Because of the over representation of Maori, preventing fatalities in house fires is also likely to contribute to reducing disparities between the health status of Maori and non-Maori.

The aims of this paper are to describe both the personal characteristics of individuals who die as a result of injury from fire and flame in a domestic location in New Zealand, and the characteristics of fatal fire incidents occurring in residential structures in New Zealand.

Methods

Cases were identified from the New Zealand Health Information Service (NZHIS) mortality data in the years 1991-1996 inclusive, using the following ICD-9-CM codes:

- E codes E890-899 (accidents caused by fire and flames); and
- domestic location code.

These codes exclude suicide and deaths from assault by fire. The NZHIS data provided details of the age, gender and ethnicity of the deceased. Rates were calculated using aggregate annual population estimates provided by Statistics New Zealand, and standardised to Segi's world population.

New Zealand Fire Service Fire Incident Reporting System (FIRS) data on fire fatalities were obtained for the period January 1991-June 1998.

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Incidents occurring in residential structures and mobile homes were included in the analysis. Incidents where the presumed cause was unlawful or deliberate, and incidents occurring in commercial premises such as motels, hotels and boarding houses, were excluded.

Results

Individual characteristics of house fire deaths: NZHIS recorded 162 unintentional deaths from injury caused by fire and flame, in the domestic location, from January 1991 to December 1996. There were between 21 and 32 deaths per annum (average 27). Mortality rates varied from 0.6 to 0.9 deaths per 100 000 person years, with no linear trend over time (Chi-squared test for trend 0.16, $p = 0.68$).

Numbers and age-specific rates for males and females are shown in Table 1. Over one-quarter of the deaths occurred in adults ≥ 65 years of age, and over one-fifth in children ≤ 14 years of age. The highest age-specific rates were observed for adults ≥ 65 years (1.8 per 100 000 person years), and children < 5 years of age (1.3 per 100 000 person years). Overall, age-standardised rates for males exceeded those for females by approximately 75% (relative risk [RR] 1.8, 95% confidence interval [CI] 1.3-2.4).

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Table 1: Deaths from injuries caused by fire and flame in New Zealand domestic locations, by age and gender, 1991-1996¹

Age group (years)	Male		Female		Total	
	Number	Rate ²	Number	Rate ²	Number	Rate ²
0-4	16	1.8	7	0.8	23	1.3
5-14	9	0.5	5	0.3	14	0.4
15-24	17	1.0	9	0.5	26	0.8
25-44	21	0.7	12	0.4	33	0.5
45-64	14	0.7	9	0.4	23	0.6
≥65	22	2.1	21	1.5	43	1.8
Total/overall³	99	0.9	63	0.5	162	0.8

Notes: 1 Data from New Zealand Health Information Service and Statistics New Zealand

2 Age-specific rates per 100 000 person years

3 Overall rate age-standardised to Segi's world population

Analysis of ethnicity was restricted to the years 1991-1994 because of changes in numerator and denominator data collection methods in 1995 and 1996. The New Zealand Maori sole ethnic group denominator was used to calculate Maori rates. Between 1991 and 1994 (inclusive), 23 Maori and 80 non-Maori died as a result of injury from fire and flame in a domestic location in New Zealand (Table 2). Age-specific Maori mortality rates exceeded non-Maori rates in all age groups except the 5-14 year group, in which no death was recorded as Maori. The highest age-specific rate for any population group was that for Maori aged ≥65 years. Age-standardised mortality rates for Maori were approximately three times those of non-Maori (RR 3.4, 95% CI 2.2-5.2). The gender disparity was not evident for Maori, with 11 male and 12 female deaths (age-standardised RR 0.8, 95% CI 0.4-1.7).

Table 2: Deaths from injuries caused by fire and flame in New Zealand domestic locations, by age and ethnicity, 1991-1994¹

Age group (years)	Maori		Non-Maori		Relative risk Maori: non-Maori (95% CI)
	Number	Rate ²	Number	Rate ²	
0-4	5	3.2	8	0.8	4.1 (1.4-12.6)
5-14	0	0	20	1.1	– ³
15-24	8	3.0	10	0.5	6 (2.4-15.2)
25-44	4	1.2	9	0.2	5.3 (1.6-17.1)
45-64	2	1.2	9	0.4	1.6 (0.7-3.6)
≥65	4	9.7	24	1.5	6.3 (2.2-18.3)
Total/overall⁴	23	2.1	80	0.6	3.4 (2.2-5.2)

Notes: 1 Data from New Zealand Health Information Service and Statistics New Zealand

2 Age-specific rates per 100 000 person years

3 Not calculated due to zero rate for Maori population

4 Overall rate age-standardised to Segi's world population

Fatal fire incidents: The FIRS database identified 154 fatalities in 133 fire incidents in residential structures attended by the New Zealand Fire Service between January 1991 and June 1998. Most of these incidents were in single dwellings occupied by one or two families (114 incidents, 85.7%), with the remainder in flats or apartments (15 incidents, 11.3%) or temporary dwellings such as caravans or sleepouts (4 incidents, 3%). Data on the room of origin of the fire were available for only 92 (69%) of the fatal fire incidents. Over a third began in a bedroom or sleeping area, over a quarter in a kitchen, and approximately a fifth in a lounge or sitting room (Table 3). The data source did not separately identify living areas being used as sleeping areas. Information about the area of origin of all (fatal and non-fatal) fires in residential structures was obtained from published data.⁴ The most striking observation is the disproportionate number of fatal house fires that began in bedrooms or sleeping areas (Table 3).

Table 3: Room of origin of fire incidents in residential structures

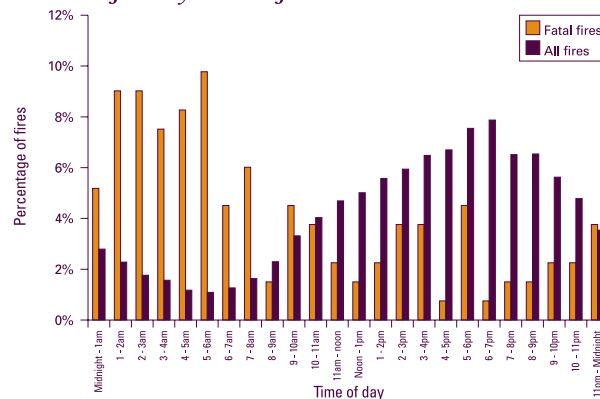
Room	Fatal incidents ¹		All incidents ²	
	Number	Percentage	Number	Percentage
Bedroom, sleeping area	35	26.3	1782	12.5
Kitchen	25	18.8	3587	25.2
Lounge/ sitting room	18	13.5	2274	16.0
Laundry	3	2.3	388	2.7
Other	11	8.3	3522	24.8
Unknown	41	30.8	2664	18.7
Total	133		14 217	

Notes: 1 Data from New Zealand Fire Service Fire Incident Reporting System January 1991-June 1998

2 Data from New Zealand Fire Service for 1995-1998, reference 4

Fatal fire incidents occurred most commonly in winter and spring, with over 70% of the incidents in the calendar years 1991-1997 (inclusive) occurring between June and November. Fatal fire incidents are more likely to occur on weekends: 26% on Sunday, 21% on Saturday and 18% on Friday, compared with an average of 9% each day Monday to Thursday. In contrast with all fires, which occur most commonly in the early evening, fatal fires occur most commonly in the early hours of the morning (Figure 1).

Figure 1: Percentage of fire incidents, and fatal fire incidents, occurring at specified time periods throughout the day, January 1991 to June 1998



Note: 1 Data from New Zealand Fire Service Fire Incident Reporting System

Reliability of data: The datasets used in this study are the most reliable sources available. The NZHIS provides the most comprehensive data available regarding mortality in New Zealand. During the study period, coding was carried out according to internally and externally audited coding standards. FIRS data are recorded manually by fire-fighters at fire scenes and entered into a national database which provides the most comprehensive record of fire incidents in New Zealand. The two datasets have been analysed separately because they do not describe exactly the same fatalities, and further information is required to link them adequately. The Fire Service does not necessarily attend all fatal fires, as some may be extinguished by household members. There may also be differences between the NZHIS definition 'domestic location' and the Fire Service definition 'residential structure'.

Maori rates were calculated using the New Zealand Maori sole ethnic group denominator data from the 1991 census, and inter-censal estimations in subsequent years up to 1994. Numerator data were from death certification. Before changes to the *Births, Deaths and Marriages Act* came into effect in September 1995, Maori ethnicity in death certification was consistently underestimated.^{5,6} Consequently, the rates of house fire deaths would tend to underestimate the excess mortality experienced by Maori. This effect is likely to be greatest for the 5-14 year age group, where undercounting of ethnicity in death certificates was most pronounced.⁷

Discussion

This review identified that the following population groups were at increased risk of fire-related death: Maori, non-Maori males, the elderly and the very young. These findings are consistent with previous injury research in New Zealand,^{1,2,8} and overseas.⁹⁻¹² Although rural residence has been associated with increased risk of fatal fires in the United States,¹³ further information is needed to classify the fires in New Zealand as urban or rural. Further information is also needed to understand factors associated with the winter peak in mortality.

The excess risk of fire-related death among Maori is in the order of three times that of non-Maori. For Maori in 1994, the category 'fire and burns' was the third leading cause of injury death in the 1-14 and over 64 year age groups.¹⁴ Because Maori are over represented in the proportion of the population living in relative social and material deprivation,¹⁵ increased fire-related mortality rates might reflect, at least partly, underlying social and economic determinants of injury.^{9,16-18} However, the relationships between ethnicity, socioeconomic status and mortality are complex and cannot be reduced to socioeconomic factors alone.^{19,20}

Those aged over 65 years are a vulnerable population group which is likely to increase in size with ageing of the population.²¹ Seniors who

die in fire incidents are less likely than younger casualties to be smokers, and more likely to be in a fire caused by faulty or misused electrical items, such as electric blankets or heaters.²² Living alone increases the fire-related mortality risk for older people, particularly individuals with physical or cognitive impairment. The presence of a functioning smoke detector lowers the odds of death for impaired individuals, including those aged over 64 years, by about 60%.²³

The relationship between social and material deprivation and risk of death in fire incidents has been well established for children.^{24,25} Possible explanations for the socioeconomic gradient for childhood mortality from fire include higher prevalence of substandard housing and use of flammable foam furniture,^{26,27} reduced capacity to provide supervision of children,^{18,28} increased involvement of children in activities with fire risk,²⁹ and increased access of children to smoking materials, including matches and lighters.³⁰

Strategies for intervention: Primary causes of house fires, and of house fire mortality, include individual behaviour as well as physical and social environmental factors. A commitment to social justice and political will are needed to reduce and abolish disparities arising from social and material inequalities, including intersectoral review of macroeconomic and social policy, access to safe, high-quality housing, and community development initiatives.³¹

As a secondary prevention measure, the presence of a smoke alarm in a private dwelling has been shown to be associated with lower odds of death when a fire does occur.^{23,32,33} Smoke detector distribution programmes have apparently resulted in reduced rates of domestic fire injury and mortality,^{17,34,35} although randomised controlled trials are needed to adequately evaluate the effectiveness of such programmes.³⁶⁻³⁸ Injury prevention counselling, particularly in the context of well child care, may increase smoke alarm ownership.³⁶ Smoke detector legislation has been enacted in many countries,³⁹ and extension to include detached private dwellings is being considered in New Zealand.⁴⁰ Landlord responsibility is illustrated by Housing New Zealand installing long-life smoke alarms in all their properties.

The information currently available supports beneficial effects on fire injury rates from smoking cessation and alcohol reduction programmes. Tobacco products have been demonstrated to cause a significant proportion of fatal fires in the United States^{41,42} and the United Kingdom.³⁰ Detailed review of coroners' verdicts, currently being undertaken, will quantify the contribution of tobacco products and smoking to fire fatalities in New Zealand. Development of 'fire-safe' cigarettes is assessed to have a high benefit-cost ratio in preventing injury,⁴³ despite the resistance of tobacco companies.^{44,45} Alcohol is implicated in fatal fires in association with smoking and cooking,^{23,30,46} and may contribute to the male excess in mortality.

Public health workers have an important role in implementing fire prevention strategies through advocacy for mandatory smoke detectors and adequate housing standards, injury prevention counselling, smoking cessation and alcohol programmes, and community-based fire safety initiatives. Smoke alarm installation is particularly important in households with children and elderly occupants, where early warning is essential to allow time to escape.

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Surveillance and control notes

Outbreak of Echovirus 33 infection in Waikato, Auckland and Wellington

The enterovirus Echovirus 33 was isolated from 56 people during the six months between March and August 2000 (Figure 1). The first cases of infection were reported from the Waikato, and were followed by cases in Auckland, Northland and Wellington/Hutt. The cases' ages ranged from 1 week to 43 years, with cases clustered in early infancy (1-6 months) and the 5-14 year age group. There have been more male cases (31) than female cases (25). The majority (29) of cases have been Maori, with 17 cases among Europeans and seven among Pacific Islands people. One case, a neonate, died.

Among the 46 cases who were over 1 year of age, 38 (83%) were diagnosed with aseptic meningitis, one with encephalitis, and one with acute flaccid paralysis. The remainder of the cases over 1 year old presented with less serious illness, such as gastroenteritis, non-specific febrile illness and upper respiratory tract infection. Most (93%) of the cases over 1 year old were hospitalised. Among the 10 cases under 1 year of age, only two were diagnosed with meningitis. All cases under 1 year old were hospitalised.

Detailed clinical information was available for 53 of the 56 cases. The 43 cases over 1 year of age most commonly presented with headache (80%), vomiting (70%), photophobia (63%), sore or stiff neck (61%), and fever (54%). In contrast, the 10 cases under 1 year of age most commonly presented with rash (90%), vomiting (60%), fever (50%), irritability (50%), and anorexia (50%). Several cases had bi-phasic illness, with onset of symptoms such as lethargy and headache, a period of improvement, and then onset of more severe symptoms, usually fever, headache and vomiting. In seven of the nine infants with a rash, the rash was maculopapular and/or petechial.

While the majority of enteroviral infections are asymptomatic, enteroviruses are also the leading cause of aseptic meningitis. Enteroviral infection in the first two weeks of life is often fatal. Outbreaks of meningitis due to Echovirus 33 are relatively uncommon compared with outbreaks due to other enteroviruses. This is the first report of an outbreak of Echovirus 33 infections in the Oceania region. The 56 cases that have been identified probably represent only a small proportion of the illness due to infection with this virus that has actually occurred. Clinicians have reported

cases of meningitis, but not taken specimens when the clinical symptoms are not suggestive of bacterial meningitis.

Enteroviruses are principally transmitted from person to person through the faecal-oral route. Therefore, extra attention to handwashing after using the toilet, before food preparation, and before and after handling babies and their nappies is important in limiting an outbreak. Newborns most commonly acquire the virus in the immediate perinatal period from the mother's blood, cervical and vaginal secretions, and the faecal contamination of the perineum. Therefore, pregnant women should avoid contact with known cases of viral meningitis to minimise the risk of infection and vertical transmission of the virus to their newborn baby. To prevent cross-infection between neonates, neonatal and maternity ward staff, midwives, and obstetricians need to pay particular attention to handwashing, and cleaning and disinfection of surfaces and other potentially contaminated objects.

In order to more precisely define the extent and course of this outbreak, clinicians are encouraged to submit CSF, rectal swabs, faecal specimens, or throat swabs from suspected cases for laboratory investigation.

Norwalk-like viruses leading cause of communicable disease outbreaks in 1999

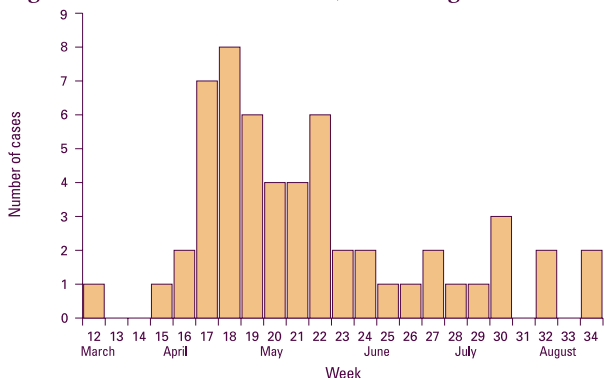
In 1999, 361 communicable disease outbreaks were reported to ESR by public health services; a 15% increase on the 313 outbreaks reported in 1998. Twenty-one of the 24 health districts reported outbreaks during the year. The Auckland area (the three combined Auckland health districts) reported the largest number (191) of outbreaks, followed by Wellington (35), Canterbury (24), and Waikato (19) Health Districts. The largest single outbreak involved 61 cases of cryptosporidiosis, which included 50 primary cases linked to a Christchurch swimming pool.

As in 1998, enteric pathogens, in particular Norwalk-like viruses (NLVs), *Campylobacter*, *Salmonella*, *Giardia*, and *Clostridium perfringens* were the most commonly reported causes of outbreaks (Table 1). Of note is the increase in outbreaks associated with NLV infection: 62 outbreaks compared with 17 in 1998. NLVs are highly infectious and potentially dangerous to immune-compromised and elderly people.

Most outbreaks were either associated with commercial food operations (160, 44.3% of the 361 outbreaks) or occurred within households (134). Among the commercial food operations, restaurants/cafes were most frequently associated with outbreaks (102), followed by takeaway outlets (29). The most common reported modes of transmission of the causative agent were food (202, 56% of the 361 outbreaks) and person-to-person contact (113). Some outbreaks had more than one probable mode of transmission.

Poultry, seafood and meat were the most frequently contaminated foods, and were implicated or suspected in 82 outbreaks, up from 27 in 1998. The most common factors probably contributing to the foodborne outbreaks were, inadequate cooling or refrigeration of food (30), cross-contamination (29), inadequate hygiene of foodhandlers (26), undercooking (25),

Figure 1: Echovirus 33 isolations, March-August 2000



Surveillance and control notes

Table 1: Suspected causative agent of outbreaks reported during 1999

Suspected pathogen or toxin	Number (percent) ¹ of outbreaks	Total number (percent) ² of cases
Enteric infections (including food and waterborne diseases)		
Gastroenteritis (unknown agent)	81 (22.4)	375 (15.9)
Norwalk-like virus (NLV)	62 (17.2)	875 (37.1)
<i>Campylobacter</i>	57 (15.8)	189 (8.0)
<i>Salmonella</i>	43 (11.9)	275 (11.7)
<i>Giardia</i>	23 (6.4)	92 (3.9)
<i>Clostridium perfringens</i>	17 (4.7)	167 (7.1)
<i>Bacillus cereus</i>	16 (4.4)	45 (1.9)
Staphylococcal toxin	16 (4.4)	43 (1.8)
<i>Shigella</i>	11 (3.0)	47 (2.0)
<i>Cryptosporidium</i>	10 (2.8)	116 (4.9)
Hepatitis A	5 (1.4)	34 (1.4)
Scombroid poisoning	3 (0.8)	8 (0.3)
Ciguatoxin	2 (0.6)	9 (0.4)
Histamine	2 (0.6)	7 (0.3)
<i>Vibrio parahaemolyticus</i>	2 (0.6)	15 (0.6)
VTEC/STEC infection serotype O157:H7	1 (0.3)	3 (0.1)
Toxic shellfish poisoning	1 (0.3)	2 (0.1)
Other infections (including airborne and person-to-person spread diseases)		
<i>Mycobacterium tuberculosis</i>	6 (1.7)	39 (1.7)
<i>Bordetella pertussis</i>	4 (1.1)	53 (2.2)
<i>Neisseria meningitidis</i>	2 (0.6)	4 (0.2)
Copper poisoning	1 (0.3)	7 (0.3)
Influenza A virus	1 (0.3)	30 (1.3)
Lead absorption	1 (0.3)	2 (0.1)
<i>Leptospirosis hardjo</i>	1 (0.3)	2 (0.1)
Sick building syndrome	1 (0.3)	14 (0.6)
Total	361	2358

Notes: 1 Percentage of the 361 outbreaks. More than one causal agent was reported for eight outbreaks.
2 Percentage of the 2358 cases. Several cases had more than one causal agent.

storage of food at ambient temperature (21), and use of ingredients from an unsafe source (21). The same six factors were the most commonly implicated in 1998.

Outbreaks are an important contributor to the burden of communicable disease in New Zealand. They also provide a good opportunity to identify sources and risk factors for infection, information that can be used to guide control measures. Medical practitioners can support the timely investigation and control of outbreaks by promptly reporting all cases of notifiable diseases to their medical officer of health. Prompt notification is particularly important where there is evidence of a common source of infection, for example, two or more linked cases of acute gastroenteritis.

Typhoid fever epidemic in Samoa

Samoa is currently experiencing an epidemic of typhoid fever, with 122 cases reported between January and July this year. Sixty-one percent (74) of the cases were culture proven. There has been one fatality. The island of Upolu is the most affected, and cases on the island are concentrated around the Apia region. Contaminated food appears to be the most probable source, although contaminated water may be the source of some infections. Eleven *Salmonella* Typhi isolates have been referred from Samoa to ESR for phage typing. Three different types were identified among the isolates: E7 variant (7 cases), E1a (3 cases), and E9 (1 case).

Typhoid is considered endemic in Samoa, with about 30-40 cases per annum over the last three years. The last epidemic occurred between 1992 and 1996, peaking with 387 cases in 1993.

From January to July 2000, 14 cases of typhoid were notified in New Zealand. Information on overseas travel was available for 13 cases; five had visited Samoa during the incubation period,

five had visited India, two had visited Sri Lanka, and one had visited Cambodia. The five cases apparently acquired in Samoa had similar phage types to those identified among the isolates referred from Samoa.

Travellers to countries with endemic typhoid should be given information on safe food and water practices. Typhoid vaccination should only be considered for those spending prolonged periods in basic conditions in typhoid endemic countries.

Shiga toxin-producing *Escherichia coli* (STEC) cases increase again in 1999

There were 64 cases of Shiga toxin-producing *E coli* (STEC), or verotoxigenic *E coli* (VTEC), infection notified in 1999, which is the highest annual total since STEC was first identified in New Zealand in 1993 (Figure 2).

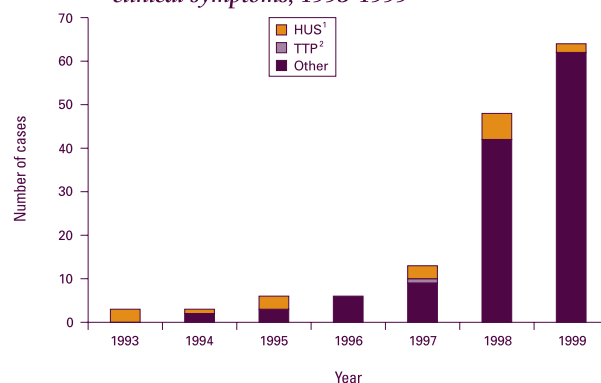
Rates of infection were highest in children aged 1-4 years (14.2 per 100 000) and those aged less than 1 year (9.1). Rates higher than the national average (1.8 per 100 000) were recorded in Waikato (10.6), Tauranga (4.4), Taupo (3.3), Wairarapa (2.6), Gisborne (2.2), Wellington (2.1), Eastern Bay of Plenty (2.0) and Taranaki (1.9) Health Districts. Most of the cases over the last seven years have been in the central North Island.

Among the STEC infections notified in 1999, two were reported with haemolytic uraemic syndrome (HUS) and 19 with haemorrhagic colitis or bloody diarrhoea. There were no cases of thrombotic thrombocytopenic purpura (TTP) (Figure 2). In contrast, seven cases of HUS were reported to the New Zealand Paediatric Surveillance Unit (NZPSU) in 1999. These seven cases included the two STEC infections notified with HUS. The other five cases of HUS reported to the NZPSU were not notified as STEC infections. These HUS cases were most likely also due to STEC infection, but as organism numbers in faeces fall off quite rapidly, isolation of the organism is often not possible by the time a child presents with HUS.

Fifty-seven (89%) of the cases in 1999 were identified as serotype O157. One case provided the first documented link between a human and animal isolate of serotype O157 in New Zealand. *E coli* O157:H7 isolates from a case and a calf from the same property were indistinguishable by molecular typing.

Medical practitioners should consider STEC infection when patients present with haemorrhagic colitis, HUS or TTP. A faecal specimen should be sent for microbiological testing and cases should be notified to the local medical officer of health. In 1999 the surveillance of STEC infections was enhanced to include the collection of risk factor information specific for STEC, as there is little known about the reservoirs and sources of this organism in New Zealand.

Figure 2: Cases of Shiga toxin-producing *E coli* infection by clinical symptoms, 1993-1999



Notes: 1 HUS, haemolytic uraemic syndrome
2 TTP, thrombotic thrombocytopenic purpura

Surveillance data

National surveillance data - May 2000

Disease ¹	Current year - 2000 ²			Previous year - 1999			Trends - May 2000
	May 2000 cases	Cumulative total year-to-date	Current rate ³	May 1999 cases	Cumulative total year-to-date	Previous rate ³	
AIDS	5	15	1.0	1	12	0.7	
Acute gastroenteritis ⁴	21	259	17.9	28	217	15.5	*
Campylobacteriosis	564	3864	243.5	454	3218	273.5	***
Cholera	0	0	0	0	0	0	
Creutzfeldt-Jakob disease	0	1	0.1	0	0	0	
Cryptosporidiosis	26	103	17.1	72	463	27.4	***
Dengue fever	1	2	0.1	0	8	0.4	**
Giardiasis	178	747	44.9	165	916	56.5	***
<i>H influenzae</i> type b disease	1	5	0.2	3	6	0.4	
Hepatitis A	2	24	2.0	19	69	3.9	***
Hepatitis B (acute) ⁵	11	38	2.4	8	44	2.8	
Hepatitis C (acute) ⁵	9	34	2.4	7	44	2.7	
Hydatid disease	0	0	0.1	2	5	0.2	
Influenza ⁶	5	11	19.3	90	109	15.0	***
Lead absorption	8	51	3.8	11	67	3.3	
Legionellosis ⁶	4	25	2.1	2	19	1.4	*
Leprosy	0	0	0.1	1	6	0.2	
Leptospirosis	6	46	2.4	6	19	1.7	
Listeriosis	1	12	0.7	2	7	0.4	
Malaria	5	23	1.3	5	23	1.6	
Measles	6	35	2.9	8	36	3.0	
Meningococcal disease	42	145	14.6	29	126	11.9	**
Mumps	7	24	1.6	4	22	1.8	
Paratyphoid	2	5	0.4	0	7	0.6	
Pertussis	291	1319	63.3	35	72	4.6	*** 1264
Rheumatic fever	3	24	1.4	5	37	2.4	**
Rubella	2	5	0.7	8	16	1.2	*
Salmonellosis	210	852	46.9	142	1232	60.6	***
Shigellosis	11	51	3.4	8	75	3.9	
Tetanus	1	1	0.1	0	3	0.1	
Tuberculosis	33	151	11.7	38	179	11.4	
Typhoid	1	10	0.4	0	4	0.6	
VTEC/STEC infection	5	37	1.8	7	35	1.7	
Yersiniosis	24	192	12.5	26	244	13.5	

Notes: 1 No cases of the following notifiable diseases were reported in May: anthrax, arboviral diseases, brucellosis, cysticercosis, diphtheria, meningococcal disease - primary amoebic, plague, poliomyelitis, rabies, rickettsial diseases, trichinosis, viral haemorrhagic fever, or yellow fever
2 These data are provisional
3 Rate is based on the cumulative total for the current year (12 months to May 2000) or the previous year (12 months to May 1999), expressed as cases per 100 000
4 Cases with suspected common source, person in a high risk category (eg foodhandler, childcare worker, healthcare worker)
5 Only acute cases of this disease are currently notifiable
6 Surveillance data based on laboratory-reported cases only
7 Percentage change is the difference between the number of cases in the current year (12 months to May 2000) and the previous year (12 months to May 1999). This difference is expressed as a percentage of the number of cases seen in the previous year

Surveillance data

Surveillance data by health district - May 2000

Cases this month Current rate¹

Disease	Cases for May 2000, ² and current rate ^{1,2} by health district ^{3,4}																									
	Northern				Midland						Central						Southern									
	Northland	NW Auck	Central Auck	South Auck	Waikato	Tauranga	Eastem BOP	Gisborne	Rotorua	Taupo	Taranaki	Ruapehu	Hawkes Bay	Wanganui	Manawatu	Wairarapa	Wellington	Hutt	Nelson-Marl	West Coast	Canterbury	South Cant	Otago	Southland		
AIDS ³	4				0						0						1									
Acute gastroenteritis	0	6	3	3	0	0	0	0	0	1	0	0	0	0	0	0	0	1	0	0	0	7	0	0	0	0
Campylobacteriosis	8	44	52	44	44	11	9	4	8	2	8	0	25	2	12	8	67	20	10	2	100	23	36	25	129.8	229.1
Cholera	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Creutzfeldt-Jakob disease	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Cryptosporidiosis	1	0	5	2	3	1	0	1	3	0	0	0	1	0	0	1	3	1	0	1	2	0	0	1	11.7	5.8
Dengue fever	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Giardiasis	1	29	30	11	16	6	0	1	8	0	2	0	9	2	4	2	11	9	2	4	16	4	9	2	23.3	60.1
H influenzae type b disease	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Hepatitis A	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	1	0	0	0	0	0
Hepatitis B	0	1	0	2	2	0	0	1	0	0	0	0	1	0	0	1	0	1	0	0	2	0	0	0	1.5	0.5
Hepatitis C	0	0	0	0	0	3	1	0	1	0	0	0	0	0	2	0	0	0	0	0	2	0	0	0	3.6	0.5
Hydatids	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Influenza ⁵	1	0	3	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	1.5	15.2
Lead absorption	1	0	0	2	0	0	0	1	0	0	0	0	2	0	0	0	0	0	0	0	1	1	0	0	0.7	0.5
Legionellosis ⁵	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	1	0	1	0	1	1	0	0	0.7	0
Leprosy	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Leptospirosis	0	0	0	0	1	0	0	0	0	0	1	0	0	0	0	0	0	0	2	1	1	0	0	0	8.8	0.5
Listeriosis	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0.7	1.3
Malaria	1	0	1	0	1	0	0	0	0	0	0	0	0	0	1	0	1	0	0	0	0	0	0	0	2.2	0.8
Measles	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0	1	1	0	3	2.2	2.5
Meningococcal disease	2	2	5	11	7	1	1	1	2	0	1	0	2	0	1	0	1	1	0	0	3	0	1	0	27.7	10.4
Mumps	0	2	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	2	0	3	0	0	0	1.5	0.5
Paratyphoid	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0
Pertussis	13	18	16	7	26	7	1	4	4	2	0	1	1	0	10	3	25	30	7	91	5	19	1	32.8	52.3	
Rheumatic fever	2	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	13.9	0
Rubella	0	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1.5	1.3
Salmonellosis	4	16	10	7	15	8	1	1	4	0	1	12	4	35	8	15	7	10	0	18	2	26	5	23.3	39.3	
Shigellosis	1	3	1	1	2	0	0	0	0	0	0	0	0	0	0	2	0	0	1	0	0	0	0	0	5.8	4.8
Tetanus	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0
Tuberculosis	1	6	7	5	3	0	1	0	0	0	1	0	1	0	0	1	3	2	0	1	1	1	0	0	16.8	11.9
Typhoid	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0.7	0.8
VTEC/STEC infection	0	0	0	0	4	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0
Yersiniosis	1	5	5	2	2	4	0	0	0	1	0	0	0	0	0	1	0	0	0	1	1	1	1	0	8.8	15.5

Notes: 1 Current rate is based on the cumulative total for the last 12 months expressed as cases per 100 000

2 These data are provisional

3 AIDS data given by divisions of the Health Funding Authority

4 Further data are available from the local medical officer of health

5 Surveillance data based on laboratory-reported cases only

Surveillance data

National surveillance data - June 2000

Disease ¹	Current year - 2000 ²			Previous year - 1999			Trends - June 2000
	Jun 2000 cases	Cumulative total year-to-date	Current rate ³	Jun 1999 cases	Cumulative total year-to-date	Previous rate ³	
AIDS	2	17	1.1	0	12	0.7	
Acute gastroenteritis ⁴	45	318	18.8	24	241	14.8	***
Campylobacteriosis	458	4324	240.2	578	3796	268.1	***
Cholera	0	0	0	0	0	0	
Creutzfeldt-Jakob disease	1	2	0.1	1	1	0	
Cryptosporidiosis	26	129	17.1	26	489	27.1	***
Dengue fever	0	2	0.1	0	8	0.4	**
Giardiasis	161	908	45.9	126	1042	54.7	***
<i>H influenzae</i> type b disease	1	6	0.3	0	6	0.3	
Hepatitis A	13	37	2.2	6	75	3.7	***
Hepatitis B (acute) ⁵	4	42	2.2	11	55	2.8	
Hepatitis C (acute) ⁵	8	39	2.3	8	52	2.7	
Hydatid disease	1	1	0.1	0	5	0.2	
Influenza ⁶	6	17	13.7	211	320	20.0	***
Lead absorption	8	58	3.6	15	82	3.5	
Legionellosis ⁶	1	26	2.0	3	22	1.3	*
Leprosy	0	0	0.1	1	7	0.3	
Leptospirosis	3	49	2.4	3	22	1.7	*
Listeriosis	1	13	0.7	1	8	0.4	
Malaria	6	28	1.4	2	25	1.5	
Measles	4	38	2.6	14	50	2.9	
Meningococcal disease	55	195	14.7	46	172	11.5	***
Mumps	5	28	1.5	7	29	1.8	
Paratyphoid	0	5	0.3	4	11	0.6	*
Pertussis	304	1619	70.3	47	119	5.7	*** 1142
Rheumatic fever	7	31	1.4	8	45	2.4	**
Rubella	1	6	0.7	2	18	1.1	
Salmonellosis	80	934	46.5	98	1330	60.1	***
Shigellosis	12	63	3.3	16	91	4.0	
Tetanus	0	1	0.1	1	4	0.1	
Tuberculosis	27	178	11.2	43	222	11.5	
Typhoid	1	12	0.4	1	5	0.6	
VTEC/STEC infection	2	39	1.8	3	38	1.6	
Yersiniosis	30	222	12.7	23	267	13.4	

- Notes: 1 No cases of the following notifiable diseases were reported in June: anthrax, brucellosis, cysticercosis, diphtheria, meningococcal disease - primary amoebic, plague, poliomyelitis, rabies, trichinosis, viral haemorrhagic fever, or yellow fever
2 These data are provisional
3 Rate is based on the cumulative total for the current year (12 months to June 2000) or the previous year (12 months to June 1999), expressed as cases per 100 000
4 Cases with suspected common source, person in a high risk category (eg foodhandler, childcare worker, healthcare worker)
5 Only acute cases of this disease are currently notifiable
6 Surveillance data based on laboratory-reported cases only
7 Percentage change is the difference between the number of cases in the current year (12 months to June 2000) and the previous year (12 months to June 1999). This difference is expressed as a percentage of the number of cases seen in the previous year

Surveillance data

Surveillance data by health district - June 2000

Cases this month Current rate¹

Disease	Cases for June 2000, ² and current rate ^{1,2} by health district ^{3,4}																							
	Northern				Midland						Central						Southern							
	Northland	NW Auck	Central Auck	South Auck	Waikato	Tauranga	Eastem BOP	Gisborne	Rotorua	Taupo	Taranaki	Ruapehu	Hawkes Bay	Wanganui	Manawatu	Wairarapa	Wellington	Hutt	Nelson-Marl	West Coast	Canterbury	South Cant	Otago	Southland
AIDS ³	1				0						1						0							
	1.8				0.3						1.2						0.5							
Acute gastroenteritis	0	6	7	3	0	0	0	0	0	1	0	0	0	0	0	2	0	0	0	23	1	0	2	
	13.1	19.5	28.3	15.5	15.9	2.7	0	87.4	1.5	123.8	10.3	0	3.5	0	0.7	0	14.8	3.8	2.6	0	53.5	5.0	15.1	6.3
Campylobacteriosis	9	40	38	43	41	16	4	1	3	3	11	4	22	5	11	6	60	23	7	1	46	11	29	24
	132.0	218.9	242.7	191.4	269.0	149.8	111.4	159.6	150.3	175.9	178.8	149.2	257.9	153.0	133.6	223.6	350.3	242.1	174.1	148.0	336.3	416.2	302.3	312.6
Cholera	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	0	0.3	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Creutzfeldt-Jakob disease	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	0	0	0.3	0	0	0.9	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Cryptosporidiosis	0	3	9	2	1	0	0	1	1	0	0	0	2	0	1	0	3	1	0	0	1	0	1	0
	11.7	6.6	8.4	7.3	38.0	13.3	8.0	13.1	6.2	3.3	13.1	0	30.0	6.5	26.6	28.6	16.5	11.3	6.0	6.2	23.5	71.7	15.6	22.5
Dengue fever	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	0	0	0.3	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0.3	1.3	0	0
Giardiasis	7	21	25	5	13	6	0	2	5	1	2	0	12	1	1	1	25	3	1	2	16	3	6	3
	27.7	60.9	72.9	35.1	63.1	55.9	17.9	35.0	41.8	58.6	17.8	35.8	70.4	22.8	23.9	23.4	56.4	41.5	20.6	67.8	39.3	31.4	34.2	23.4
H influenzae type b disease	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	1.5	0.3	0.9	0.3	0	1.8	0	0	0	0	0.9	0	0	0	0	0	0	0	0	0	0	0	0	0
Hepatitis A	0	1	2	1	0	0	0	0	2	0	0	0	0	0	0	0	0	0	0	0	7	0	0	0
	0	1.5	5.8	3.8	0.7	3.5	2.0	6.6	3.1	0	1.9	0	1.4	1.6	1.3	0	2.9	1.5	0.9	0	2.6	2.5	0.6	0
Hepatitis B	0	0	0	0	2	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	1	0	0
	1.5	0.5	2.6	0.9	5.3	3.5	0	4.4	1.5	3.3	0	6.0	3.5	1.6	0.7	2.6	1.6	4.5	0.9	0	2.8	6.3	1.7	0.9
Hepatitis C	0	1	0	0	0	2	0	0	2	0	1	0	0	0	0	0	0	0	0	0	1	0	1	0
	2.9	0.8	0.3	0.3	0	16.0	6.0	0	18.6	0	0.9	0	4.9	0	1.3	0	0.8	1.5	0.9	0	4.7	2.5	3.5	0
Hydatids	0	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	0	0	0	0	0.3	0	0	2.2	0	0	0	0	0	0	0	0	0.4	0	0	0	0	0	0.6	0
Influenza ⁵	0	0	4	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0	1	0
	1.5	6.8	14.5	18.7	10.9	0	15.9	6.6	1.5	0	2.8	0	1.4	9.8	0.7	10.4	8.6	0	18.0	9.3	53.5	17.6	9.8	7.2
Lead absorption	0	0	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	3	1	1	0
	0	0.3	1.4	0.9	5.9	0	2.0	4.4	3.1	0	3.7	0	4.2	1.6	6.6	7.8	3.7	0.8	2.6	3.1	9.3	18.9	2.9	2.7
Legionellosis ⁵	0	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	0.7	0	1.2	0	7.6	0.9	2.0	0	0	3.3	0	6.0	1.4	0	0.7	7.8	2.5	3.8	0	3.1	4.9	0	1.7	0.9
Leprosy	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	0	0	0	0.6	0	0	0	0	0	0	0	0.7	0	0	0	0	0	0	0	0	0	0	0	0
Leptospirosis	0	1	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0
	8.0	0.8	0	0.3	3.3	2.7	0	4.4	0	3.3	6.6	6.0	4.9	3.3	3.3	0	0	0	4.3	12.3	2.6	8.8	2.3	2.7
Listeriosis	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0
	0.7	1.3	0.6	0.3	0.3	0.9	0	0	1.5	0	0	0	0	0	0.7	0	0	1.5	0.9	0	0.8	3.8	0	1.8
Malaria	0	0	0	0	0	0	0	0	0	0	0	0	0	0	2	0	0	1	0	0	3	0	0	0
	1.5	0.8	2.0	1.2	1.0	0	4.0	0	0	0	0	0.7	1.6	3.3	0	1.2	1.5	3.4	0	2.1	0	2.3	0	
Measles	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	1	0	0
	2.9	2.0	1.4	1.5	0	0.9	0	0	0	6.5	0	0	9.1	0	4.7	7.8	0.8	3.8	4.3	12.3	3.1	3.8	5.2	5.4
Meningococcal disease	4	5	4	17	2	2	1	2	3	0	1	0	1	2	0	1	2	2	0	0	3	1	2	0
	23.3	11.4	24.6	38.6	13.6	8.0	23.9	15.3	21.7	16.3	6.6	0	12.5	11.4	1.3	13.0	4.9	12.1	1.7	0	10.1	2.5	14.5	12.6
Mumps	1	1	1	0	0	0	0	0	0	0	0	1	1	0	0	0	0	0	0	0	0	0	0	0
	1.5	0.8	1.7	2.3	0	1.8	0	0	3.1	6.5	2.8	0	3.5	1.6	1.3	0	0.4	0.8	4.3	3.1	2.1	0	1.7	0
Paratyphoid	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	0	0.3	0	0.6	0.7	0	0	0	0	0	0	0	0	0	0	0	0.8	0.8	0	0	0.5	0	0.6	0
Pertussis	7	15	15	5	24	3	0	1	1	0	0	4	0	0	10	13	21	30	48	71	2	32	2	
	35.7	56.1	28.9	34.5	56.5	33.7	6.0	21.9	10.8	6.5	19.7	11.9	15.3	3.3	6.6	124.8	30.9	69.4	236.7	215.9	168.9	137.1	81.1	274.9
Rheumatic fever	1	0	0	0	1	1	2	0	0	0	0	0	0	0	0	0	1	1	0	0	0	0	0	0
	12.4	0	0.6	0.3	2.0	3.5	6.0	8.7	0	3.3	0.9	6.0	2.1	0	2.6	1.6	2.3	0	0	0	0	0	0	0
Rubella	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0
	1.5	1.3	0.3	0.3	0	0.9	0	0	0	0.9	0	1.4	0	0	0	0.8	0	0	0	6.2	0.3	0	3.5	0.9
Salmonellosis	3	3	5	3	6	0	1	2	1	0	3	0	8	0	5	6	2	4	7	0	9	4	4	4
	26.3	36.5	35.3	28.1	44.0	37.2	29.8	41.5	34.1	32.6	24.3	35.8	49.5	21.2	81.1	169.0	60.5	44.5	52.3	12.3	45.5	91.8	72.4	84.4
Shigellosis	1	5	0	2	2	0	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	1	0
	6.6	5.8	4.9	6.1	2.0	0	6.0	6.6	7.7	19.5	0.9	0	0	0	1.3	2.6	2.5	5.3	0	1.0	1.3	1.7	0.9	
Tetanus	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	0	0	0	0	0	0	0	0	0	0	0	0.7	1.6	0	2.6	0	0	0	0	0	0	0	0	0
Tuberculosis	0	3	12	2	1	1	0	0	0	1	1	0	1	0	1	0	2	2	0	0	0	1	0	0
	16.0	10.9	24.0	19.9	9.6	9.8	11.9	0	4.6	0	1.9	6.0	10.5	14.7	6.6	2.6	17.7	21.1	2.6	0	5.4	5.0	2.3	0.9
Typhoid	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0	0
	0	0.8	1.4	1.5	0	0.9	0	0	0	0	0	0	0	0	0	0.8	0	0	0	0	0	0	0	0
VTEC/STEC infection	0	0	0	0	0	0	1	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0
	0	0.3	0.6	0.9	7.9	1.8	6.0	2.2	0	19.5	2.8	0	0	0	0	2.1	0.8	0	0	2.6	0	2.3	0	
Yersiniosis	0	3	0	6	3	3	1	0	1	0	0	0	1	0	0	3	1	0	1	2	1	0	4	
	8.8	16.0	17.4	13.5	12.6	14.2	9.9	2.2	10.8	16.3	1.9	0	18.8	3.3	3.3	2.6	15.2	16.6	7.7	33.9	15.3	22.6	2.3	7.2

Notes: 1 Current rate is based on the

Pre-hospital thrombolysis increases survival after acute myocardial infarction

A meta-analysis of six randomised controlled trials confirms pre-hospital administration of thrombolysis for acute myocardial infarction (AMI) improves survival. All-cause hospital mortality was significantly reduced (odds ratio 0.83, 95% confidence interval 0.70-0.98) among patients who received thrombolysis before they were hospitalised, compared with patients who were not treated until after hospitalisation. The absolute risk reduction equates to one life saved for every 62 patients with overt AMI given pre-hospital thrombolysis. The average time between symptom onset and thrombolysis treatment was 104 minutes for patients in the pre-hospital treatment group versus 162 minutes in the in-hospital treatment group. Results were similar regardless of the training and experience of the provider (Morrison LJ, Verbeek PR, McDonald AC, et al. Mortality and prehospital thrombolysis for acute myocardial infarction. A meta-analysis.

JAMA 2000; 283: 2686-92).

Editorial note: The American Heart Association's (AHA) *Guidelines for the management of patients with acute myocardial infarction* recommend the administration of pre-hospital thrombolysis when there is a significant delay in transferring the patient to hospital. The availability of well-tolerated bolus thrombolytics, which can be stored at room temperature, now make this recommendation a practical proposition. Some regions in New Zealand (Coromandel, Queenstown, and parts of Hawkes Bay and Nelson) have already introduced general practitioner (GP)-administered pre-hospital thrombolysis for rural patients who experience delays in transport to a coronary care unit. New Zealand guidelines on pre-hospital thrombolysis are being finalised, and emphasise the importance of GP training and GP liaison with the hospital their patient is admitted to.

Antibiotic treatment of children with *Escherichia coli* O157:H7 infection increases the risk of haemolytic uraemic syndrome

A study in four states in the United States found that antibiotic treatment of children with *Escherichia coli* O157:H7 infection increases the risk of haemolytic uraemic syndrome (HUS). The prospective cohort study included 71 children aged <10 years, with *E coli* O157:H7 identified in their stool samples. Medication history was based on drugs taken on or before the seventh day of illness, with prescription drug use verified by the medical provider or by consulting the child's medical record. HUS developed in 10 of the 71 children (14%), including five of nine children given antibiotics (56%) and five of 62 children not given antibiotics (8%). After adjustment for potential confounding factors (initial white-cell count and the day of illness on which the stool sample was obtained for culture), antibiotic administration was significantly associated with the development of HUS (relative

risk 17.3, 95% confidence interval, 2.2-137). The characteristics of the children who were given antibiotics were similar to those of the other children (Wong CS, Jelacic S, Habeeb RL, et al. The risk of the hemolytic-uremic syndrome after antibiotic treatment of *Escherichia coli* O157:H7 infections. N Engl J Med 2000; 342: 1930-6).

Editorial note: The Infectious Disease Advisory Committee to the Ministry of Health reviewed this paper and made the following recommendations: (1) faecal specimens from cases of acute diarrhoeal illness should be collected for laboratory investigation of the causative agent, (2) antibiotics should only be used if a specific pathogen has been identified, and the illness is neither benign nor self-limited, and (3) if *E coli* O157 is detected, antibiotics should not be used.

Travel health

Cholera epidemic in Federated States of Micronesia

A cholera outbreak in the Federated States of Micronesia has resulted in 954 cases and 9 deaths up to 26 June 2000. This country includes four main island groups (Pohnpei, Truk, Yap and Kosrae) and is located in the northern Pacific Ocean. The outbreak was first reported on 17 April. The causative organism has been confirmed as *Vibrio cholerae* serogroup O1 subtype Ogawa. A range of control measures have been implemented, including health education on water disinfection and safe disposal of faeces, controls on some forms of social gatherings, active case-finding and selective chemoprophylaxis of households, and restrictions on travel to outer islands (Outbreak news. Cholera, Federated States of Micronesia. Weekly Epidemiol Rec 2000; 28: 225, supplemented by information on Promed).

Editorial note: Cholera poses a very small risk to New Zealand travellers. Only two imported cases have been notified since 1996. Both were linked to travel to a Fiji resort island. Usual precautions against enteric disease provide the best form of protection. These precautions include only drinking water that has been boiled or disinfected with chlorine or iodine; avoiding ice, unless it is known to have been made from safe water; only eating food that has been thoroughly cooked and is still hot when served; avoiding raw seafood and other raw food, except fruit and vegetables which can be peeled just before eating; and boiling unpasteurised milk.

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