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An outbreak of hepatitis A among an Auckland immigrant community

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There was an outbreak of hepatitis A among a small immigrant community in Auckland during the winter of 1999. A total of 14 cases were identified, and they were all children aged 1-8 years. The outbreak ended one month after the commencement of a free vaccination programme. The likely mode of transmission was person-to-person spread. Outbreak investigation and control were complicated by a number of factors, including the absence of symptoms among young children, incorrectly notified contact details, a general practitioner advising against immunoprophylaxis, reluctance among general practitioners to administer immune globulin, and language and cultural barriers. Control of similar outbreaks of hepatitis A could be improved by enhanced use of immune globulin, comprehensive administration of hepatitis A vaccine, and more effective community engagement.

Auckland Healthcare Public Health Protection identified an outbreak of hepatitis A in mid-September 1999. Although there was no increase in the total number of notifications, a number of new cases of hepatitis A were identified as members of a small Auckland immigrant community. This ethnic group makes up a tiny proportion of the population of Auckland: approximately 185 people or 0.015% of the Auckland population. However, between 1 June and 13 September 1999, this community contributed 50% (7/14) of all the hepatitis A notifications in Auckland.

This article describes the investigation and management of the outbreak, and factors that complicated the investigation and control of the outbreak. In addition, the article provides recommendations for improving the control of similar future outbreaks.

Investigation method

For this outbreak, a case of hepatitis A was defined as an individual of the particular immigrant ethnicity, who had either:

- a positive hepatitis A IgM antibody or
- a raised serum aminotransferase (ALT) and a consistent clinical illness, that is, jaundice, nausea, anorexia, fever.

Prompt interviews with parents of the cases were carried out by telephone using a standardised hepatitis A case questionnaire developed by Auckland Healthcare.

Additional strategies were employed in response to this apparent outbreak. These strategies included enhancing existing surveillance by close scrutiny of the direct reports from the Auckland Virology Laboratory, which performs hepatitis A serology for the greater Auckland

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population, and alerting the general practitioners of cases' families to the need for prompt notification on suspicion.

Results

Fourteen cases of hepatitis A in this ethnic community were identified between 1 July and 22 October 1999.

The parents of 13 (93%) of the 14 cases were interviewed. One family failed to respond to repeated telephone requests for interview.

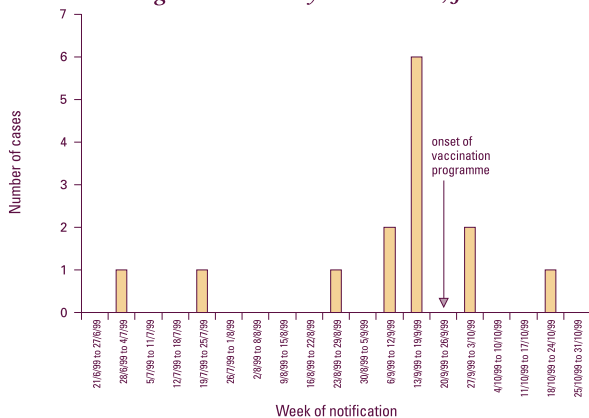
The onset of symptoms in the index case was on 29 June 1999. The first notification was received on 1 July 1999. The majority of the notifications (eight) occurred between 7 and 17 September 1999, with the final notification received on 22 October 1999 (*Figure 1*). All cases were aged 1-8 years. There have been no further notifications of hepatitis A in this ethnic community to date (August 2000).

All cases had been in contact with at least one other case, on at least one occasion, in the 50 days preceding the onset of illness. The majority of cases had multiple contact with other cases. These contacts were usually at meetings of extended family and friends, where food was prepared by the host family and shared with guests. Children mixed freely on these occasions. There were no other common features that linked

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Figure 1: Date of notification of hepatitis A cases among an immigrant community in Auckland, June-October 1999



cases, for example, attendance at the same school or childcare.

There was no increase in hepatitis A notifications among any other ethnic group in Auckland. The cases were not confined to any specific geographical area of Auckland.

Control measures

Cases were visited by public health nurses. Close contacts of cases eligible for immunoprophylaxis were identified. The cases' parents were given advice on preventing spread of the virus by good personal hygiene and careful handwashing, excluding cases from school or childcare until the infectious period had elapsed, and reporting symptoms suggestive of the disease to their medical practitioners.

Immune globulin: Immune globulin (IG) was recommended by Auckland Healthcare for all close contacts of an infectious case whose last contact with the case had been within the previous 14 days.

IG was indicated for 64 individuals in 10 families. However, 13 individuals in one family were contacted too late for IG to be effective, and 24 individuals in five families refused IG. Twenty-seven individuals in four families accepted IG.

The main reason for refusal of IG was the unwillingness on the part of close contacts to receive a blood product. IG was also declined on the advice of a general practitioner, who advised against it on the grounds that they considered hepatitis A in children to be a mild illness which did not need prophylaxis.

Vaccination: In view of the continuing nature of the outbreak and the poor uptake of IG, on 20 September 1999 it was decided to commence a free vaccination programme for close contacts aged <10 years who were not already cases. Free vaccination was only offered to members of the immigrant community.

The vaccination programme was promoted informally with the families of cases and via the general practitioners of cases' families. Vaccination was commenced on 20 September. Since outbreak control was the primary aim of the vaccination programme, a decision was made not to offer free booster doses of vaccine.

Families' general practitioners were willing to be involved in the vaccination programme. However, to expedite the programme, Auckland Healthcare staff visited two families and administered vaccine.

Vaccine was offered for administration in contacts' homes to nine individuals, and accepted by all nine. Vaccine was offered for administration in general practice clinics to 16 individuals, and accepted by three. The main reason for the low uptake of vaccine was the perception among families of low personal risk of infection.

Three cases, from two families, were notified after the onset of the vaccination programme. These three cases were from families of the immigrant community, but had not previously been recognised as contacts, and had not received either IG or vaccine.

Discussion

This report describes an outbreak of hepatitis A among the children of a small immigrant community in Auckland. A number of sources of infection were possible. Person-to-person spread was the most likely source of the infections since the prolonged period over which

cases presented argues against an outbreak due to a point source.

A waterborne source was unlikely, since the cases had only consumed water from the mains city supply and they were geographically spread across the city. If the mains supply had been contaminated, it is likely that there would have been a significant burden of illness among the wider Auckland community.

The investigation of a potential foodborne source was hampered by the long incubation period of the disease. Attempting food recall over a period of seven weeks is highly problematic, particularly when combined with communication difficulties - English was not the first language of the immigrant community. A common foodborne source was unlikely since the onsets of symptoms occurred over several months. In addition, there was no increase in notifications among individuals of the same religion but different ethnicity, who were likely to have consumed food from similar sources as the affected group. However, while the dynamics of the epidemic indicate that a particular food was not likely to be the common source of the infections, food could still have been the vehicle in person-to-person transmission.

A number of outbreaks of hepatitis A have been reported in New Zealand. Most have been transmitted by person-to-person spread. Community-wide outbreaks of hepatitis A due to person-to-person spread characteristically produce sustained outbreaks and are difficult to control. Outbreaks may persist until the pool of susceptible individuals is exhausted. An outbreak in Hawkes Bay in 1994 was thought to have been transmitted via this route.¹ A campaign to raise awareness of the disease and to promote preventive measures was followed by a fall in notified cases.² Person-to-person spread, through high-risk sexual practices, was considered to be responsible for outbreaks of hepatitis A among homosexual men in Wellington and Auckland in 1995.³ Safer sex practices were encouraged via flyers, pamphlets, posters and advertisements in magazines and on gay radio.⁴ The Auckland Public Health Service offered free vaccine to men frequenting sex-on-site venues, and there was an associated rapid fall in notified cases.⁵ An outbreak of unknown source in Northland, probably transmitted by person-to-person spread, led to a campaign in which over 500 people were vaccinated.⁶

Identified common source outbreaks of hepatitis A are rare in New Zealand, but have been reported.⁷ Sources include raw shellfish and inadequately treated drinking water. An outbreak associated with a Wellington delicatessen had an epidemic curve consistent with a common source.⁸ The outbreak persisted for only six weeks, with a clear peak in notifications, and was associated with consumption of foods prepared by an infected foodhandler.

Passive immunisation with pooled normal IG is indicated for close contacts of cases of hepatitis A.⁹ IG is administered both to afford protection to the contact and to reduce the risk of spread into the wider community. Inactivated hepatitis A vaccine is indicated for individuals with an ongoing risk of exposure to hepatitis A. Hepatitis A vaccine has been demonstrated to be effective in controlling community-wide epidemics of hepatitis A in children,^{10,11} including evidence from robust, randomised controlled trials.^{12,13} Outbreaks involving childcare centres have also ended after the use of hepatitis A vaccine,¹⁴⁻¹⁶ although evidence that vaccine was the critical factor is less compelling.

In this outbreak, free vaccine was only offered to children under the age of 10 years. No cases of hepatitis A had been reported in individuals over the age of eight years, presumably because older relatives had been infected while resident in their native country - one with a high incidence of hepatitis A - prior to their arrival in New Zealand. In areas of high endemicity, $\geq 90\%$ adults have evidence of previous infection and most children become infected by 10 years of age.¹⁷ All adults (four out of four), who subsequently had their hepatitis A status checked by their general practitioner, were found to be immune to hepatitis A.

Since no controlled trial was undertaken with the current outbreak, it is difficult to know whether the use of vaccine and IG was the factor that limited the spread of hepatitis A. Uptake of both vaccine and IG were limited, and the outbreak may have been waning anyway.

The use of immune globulin and vaccine to control hepatitis A

Hepatitis A vaccine is widely used to protect travellers and others who expect to be at increased risk of hepatitis A. Vaccination is generally preferred to the use of immune globulin (IG), except where immediate protection is needed. Requirements are different in hepatitis A control situations where a case or an outbreak has already occurred. This brief review is concerned with such situations.

IG offers effective protection against hepatitis A. IG has an exemplary safety profile, with an effective process for the detection/removal of bloodborne viruses, and no recorded transmission of bloodborne viruses in Australasia. However, its usefulness in outbreaks is limited by poor patient compliance, the need for timely recognition of exposure, and its limited duration of protection. Hepatitis A vaccine offers the potential for improved long-term protection against hepatitis A, particularly in propagated epidemics.

A review of the evidence for the most appropriate use of hepatitis A vaccine, using a comprehensive, systematic search and review of the New Zealand and international literature relating to the efficacy of hepatitis A vaccine in outbreaks and as post-exposure prophylaxis, produced 12 articles.^{5,10-16,20-23} These were critically appraised using a systematic, evidence-table format. The articles were used to make evidence-based recommendations for the use of hepatitis A vaccine and IG in outbreaks and as post-exposure prophylaxis.

However, there remained a significant number of children in the immigrant community who were likely to have been susceptible to hepatitis A, given the low prevalence of hepatitis A antibodies in New Zealand children.¹⁸ It is also possible that further children developed unrecognised, asymptomatic infections.¹⁹

The outbreak investigation and control were complicated by a number of factors:

- the absence of symptoms in young children, making the calculation of infectious periods and full case ascertainment impossible
- language and cultural barriers, compromising the accurate completion of case report forms and contact tracing
- a general practitioner advising families against the use of IG
- general practitioners' reluctance to administer IG
- incorrectly notified home addresses and a mobile population, delaying the possibility of early intervention with IG.

The early and widespread use of IG would likely have been effective in controlling this outbreak. IG has proven effectiveness in limiting transmission in defined outbreaks and in post-exposure prophylaxis of contacts. Hepatitis A vaccine is effective in controlling outbreaks of hepatitis A, and may be administered concurrently with IG to provide both immediate and longer-term protection, without reducing the efficacy of vaccine or IG.

Language and cultural barriers limited the effectiveness of outbreak control. These barriers could be reduced by effective engagement with the community at the earliest opportunity.

Acknowledgements: The Food and Communicable Disease Teams of Auckland Healthcare Public Health Protection for their help in outbreak investigation and control, Phyllis Taylor for advice on the review of hepatitis A vaccine, and Lester Calder for peer review.

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Outbreaks of hepatitis A:

- IG is effective in limiting transmission in outbreaks of hepatitis A
- There is moderate evidence that hepatitis A vaccine is effective in controlling outbreaks of hepatitis A.^{5,10-16,20}

Before vaccine is used for outbreak control, consideration needs to be given to the current hepatitis A epidemiology in the wider community, and the population at increased risk needs to be clearly identified. Factors to consider before initiating a vaccine control programme include the feasibility of rapidly vaccinating the target community and the cost.

IG may be administered with vaccine to provide both immediate and longer-term protection, without significantly reducing the efficacy of the vaccine.

Post-exposure prophylaxis:

- IG is effective in limiting transmission to contacts recently exposed to hepatitis A virus, if the last contact with an infectious case occurred within the previous two weeks.

A dose of 0.02 ml/kg should be administered as soon as possible. Screening of contacts for immunity is not recommended, since screening adds to the cost and will delay administration of IG. If the risk of hepatitis A is likely to be ongoing, hepatitis A vaccine may be administered with IG to provide longer-term protection.

- There is insufficient evidence to recommend hepatitis A vaccine alone for post-exposure prophylaxis.²¹⁻²³

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Incidence of *Haemophilus influenzae* type b (Hib) disease in 1999

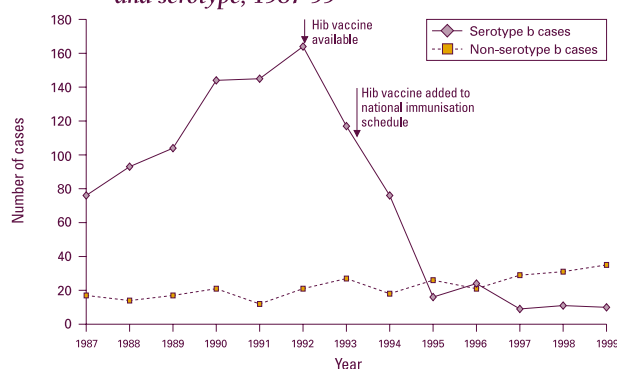
The incidence of Hib disease was again low in 1999 (Figure 1). Ten cases were notified, a rate of 0.3 cases per 100 000. None of the cases died.

Among the 10 cases, two were infants less than one year of age (age-specific rate of 3.7 per 100 000). The other cases were distributed as follows: 1-4 years old (3 cases), 30-49 years (3), and 50-69 years (2). Five of the 10 cases were European (rate of 0.2 per 100 000), two were Pacific Islands people (1.2 per 100 000), and the ethnicity of the other three cases was not known. Six of the cases were female and four were male.

Five of the cases were too old (>10 years) to have been routinely offered Hib vaccination, and one was too young (<5 months) to have completed a protective course of at least three doses of vaccine. Among the four cases who were eligible to have received ≥ 3 doses of vaccine, two were recorded as having a documented history of ≥ 3 doses, one case was incompletely vaccinated with one dose, and the fourth case was unimmunised.

Isolates from 35 cases of non-serotype b *H influenzae* invasive disease were referred to ESR laboratories in 1999 (Figure 1). Five of the isolates were serotype f, two were serotype e, and the remaining 28 were untypable. The annual incidence of non-serotype b (including untypable) invasive disease has remained relatively constant since the introduction of Hib vaccine (Figure 1). Hib vaccine is only effective against serotype b disease.

Figure 1: *Haemophilus influenzae* disease cases by year and serotype, 1987-99



Hospitalisations and fatalities from notifiable communicable diseases in 1999

The total number of deaths from notifiable communicable diseases increased from 54 in 1998 to 65 in 1999 (Table 1). The case-fatality rates presented in Table 1 generally reflect acute mortality as, except for AIDS, only deaths among cases both notified and dying in 1999 are included. The 19 deaths recorded for AIDS are all the deaths from AIDS in 1999, and therefore include deaths among cases notified before 1999. Consequently, a case-fatality rate has not been calculated for AIDS.

Meningococcal disease accounted for the largest number of deaths from a notifiable communicable disease in 1999 (Table 1). The number of meningococcal disease fatalities in 1999 was the same as that in 1998 (23), although the case-fatality rate was not quite as high in 1999: 4.6% compared with 5.2% in 1998.

Deaths from notifiable communicable diseases are relatively rare, and need to be accurately recorded. Under the Health Act 1956, funeral directors are legally required to notify the local medical officer of health of any deaths from infectious diseases. This requirement of funeral directors is in addition to the requirement for medical practitioners to report notifiable diseases. Other communicable

diseases which are not notifiable, for example influenza, also cause significant numbers of fatalities.

Table 1: Fatal cases of notifiable communicable diseases, 1999¹

Disease	Number of fatal cases	Total number of cases	Case-fatality rate (%)
AIDS	19	33	-
Campylobacteriosis	1	8173	0.01
Creutzfeldt-Jakob disease	2	2	100
Legionellosis	1	51	2.0
Listeriosis	3 ²	19	15.8
Meningococcal disease	23	505	4.6
Salmonellosis	1	2079	0.05
Shigellosis	1	147	0.7
Tuberculosis ³	14	456	3.1
Total	65	-	-

Notes: 1 Based on data recorded with the case notification.
2 There were two fatalities from perinatal listeriosis and one from non-perinatal listeriosis.
3 Tuberculosis is a treatable disease, so it is possible that several of these deaths were in people who did not receive treatment in New Zealand. The diagnosis of tuberculosis may have been made at autopsy.

Hospitalisations due to notifiable communicable diseases in 1999 are shown in Table 2. Meningococcal disease accounted for the largest number of hospitalisations, followed by campylobacteriosis, then tuberculosis and salmonellosis. The current pertussis epidemic resulted in 110 hospitalisations for this disease in 1999, compared to 17 in 1998. Several communicable diseases have high rates of hospitalisation, often because of the need for intravenous antimicrobial therapy as well as other intensive care.

Table 2: Hospitalised cases of notifiable communicable diseases, 1999¹

Disease	Number of hospitalised cases	Number of cases for which hospitalisation status reported	Hospitalisation rate (%)
Acute gastroenteritis	22	485	4.5
Campylobacteriosis	304	5701	5.3
Cryptosporidiosis	47	846	5.6
Dengue fever	2	9	22.2
Giardiasis	23	1271	1.8
<i>H influenzae</i> type b disease	10	10	100
Hepatitis A	19	108	17.6
Hepatitis B	19	75	25.3
Hepatitis C	5	62	8.1
Hydatid disease	5	8	62.5
Lead absorption	8	109	7.3
Legionellosis	38	45	84.4
Leprosy	2	4	50.0
Leptospirosis	19	41	46.3
Listeriosis	18	19	94.7
Malaria	25	44	56.8
Measles	2	97	2.1
Meningococcal disease ²	497	505	98.4
Mumps	1	51	2.0
Paratyphoid	2	15	13.3
Pertussis	110	957	11.5
Poliomyelitis ³	1	1	100.0
Rheumatic fever	44	49	89.8
Rubella	2	34	5.9
Salmonellosis	192	1797	10.7
Shigellosis	19	115	16.5
Tetanus	6	6	100.0
Tuberculosis	273	408	66.9
Typhoid	5	9	55.6
VTEC/STEC infection	20	60	33.3
Yersiniosis	26	386	6.7
Total	1766	-	-

Notes: 1 Based on data recorded with the case notification. Hospitalisation data are not available for AIDS or Creutzfeldt-Jakob disease.
2 Five of the eight cases not hospitalised died before this was possible. The other three cases received emergency department assessment and treatment, but were not admitted, even after post-diagnosis follow-up, due to mildness of symptoms.
3 Diagnosed as vaccine-associated, not wild type.

Outbreak of legionnaires disease associated with Melbourne aquarium

An outbreak of legionnaires disease associated with a Melbourne aquarium in April is the largest that has been recorded in Australasia. By mid-May, there had been 101 cases confirmed with *Legionella pneumophila* serogroup 1 infection, two of whom had died and three were in a critical condition. There have been four cases among New Zealanders, three of whom were notified to public health services in New Zealand. All cases appear to have contracted their infection over the two week period between 11 and 25 April, and all cases had either visited or walked past the aquarium. The aquarium's water cooling towers were identified as the probable source of the infections. *L pneumophila* serogroup 1 was identified in samples taken from the towers, before they were disinfected on 27 April.

Legionella exist in both aquatic (often man-made) and terrestrial environments. *L pneumophila* is generally isolated from potable water including hot water cylinders, shower rosettes, spa pools and cooling towers. In addition, potting mix appears to be a source of *Legionella* in New Zealand. *L longbeachae* is the commonest species isolated from potting mix. An increasing proportion of legionellosis cases in New Zealand are due to *L longbeachae*, with 52.3% of cases due to this species in 1999. Groups at increased risk of legionellosis include those over 50 years, heavy smokers, heavy drinkers, diabetics, people with chronic lung disease, and those who are immune compromised.

New Zealand has regulations covering domestic hot water systems and water cooling systems, as well as maintenance and testing standards for water cooling systems. The regulations and standards are intended to prevent the growth of *Legionella* in these water systems. People working with potting mix should decrease the risk to their health by taking measures to prevent inhalation of the mix. These measures include having adequate ventilation and moistening the potting mix before use.

Patterns of measles, mumps and rubella

The total number and age distribution of cases of measles, mumps and rubella notified in 1999 is shown in Table 3. Rates of measles and rubella were highest in children under one year of age, whereas rates of mumps were highest in the 1-4 year age group. Cases of rubella were fairly evenly distributed between females and males, but males predominated among cases of measles and mumps: 57.5% and 69.1%, respectively. The rates of hospitalisation for these diseases ranged from 2-5.9% (Table 2), and no deaths were recorded. There were no cases of congenital rubella syndrome notified in 1999. Only a small proportion of the notified cases of these three diseases were laboratory confirmed: 21 (19.8%) of the measles cases, 5 (8.9%) of the mumps cases and 2 (5.4%) of the rubella cases.

Measles, mumps and rubella have generally shown cyclical incidence patterns in New Zealand, with epidemics of each of the diseases occurring periodically (Figures 2, 3 and 4). The interepidemic period of these three diseases depends on the rate at which the number of susceptible people in

Table 3: Measles, mumps and rubella notifications by age group, 1999

Age group (years)	Measles		Mumps		Rubella	
	Cases	Rate ¹	Cases	Rate ¹	Cases	Rate ¹
<1	31	56.7	1	1.8	12	21.9
1-4	44	19.6	24	10.7	15	6.7
5-9	10	3.5	13	4.5	3	1.0
10-14	8	3.0	7	2.6	3	1.1
15-19	3	1.1	0	0	2	0.8
20-29	6	1.1	4	0.7	2	0.4
30-39	3	0.5	3	0.5	0	0
40-49	1	0.2	2	0.4	0	0
50-59	0	0	1	0.3	0	0
60-69	0	0	0	0	0	0
≥70	0	0	0	0	0	0
Unknown	0	-	1	-	0	-
Total	106	2.9	56	1.5	37	1.0

Note: 1 Crude rate per 100 000, based on 1996 Census

the population accumulate. Over the last 10 years, the interepidemic periods and the size of epidemics have been affected by changes in vaccination policies and by various MMR vaccination campaigns. In late 1990, mumps immunisation was introduced into the national immunisation schedule, when the infant measles vaccination was replaced with MMR vaccination. In 1992, the rubella vaccination of 11 year old girls was changed to MMR vaccination and extended to include boys. In 1991, and again on a larger scale in 1997, there were MMR vaccination campaigns to control measles epidemics. In 1994, there was a relatively small MMR vaccination campaign to control a mumps epidemic.

Eradication of the measles virus is possible. Measles has already been eliminated from some parts of the world, such as the Americas and Finland, through mass vaccination campaigns or high coverage with two routinely scheduled doses of MMR. While New Zealand now has a 2-dose MMR schedule, coverage for the first dose is low (estimated to be only around 80% in 1999). It has been recommended that the timing of the second dose be changed from 11 years to around 5 years of age to reduce the number of children that remain susceptible to measles (through failure to receive or respond to the first dose), and thereby enhance the possibility of measles being eliminated in due course from this country. However, in the meantime, medical practitioners should ensure children receive two doses of MMR as scheduled at 15 months and 11 years. Where a person's vaccination history is uncertain, they should be given a dose of MMR. There are no adverse effects from receiving an extra dose of this vaccine.

Figure 2: Measles cases by year, 1980-99

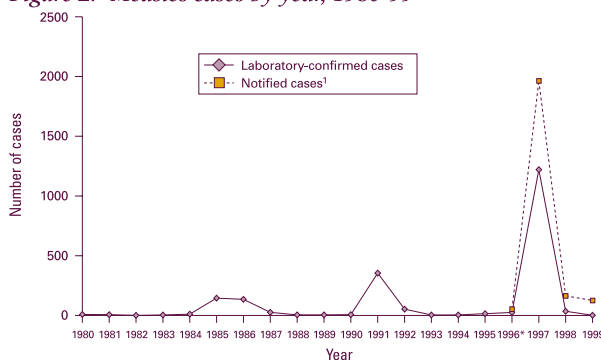


Figure 3: Mumps cases by year, 1980-99

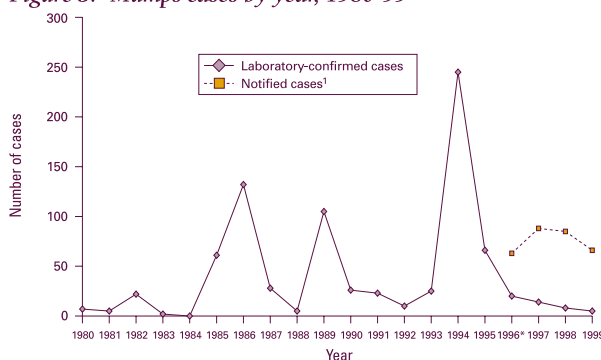
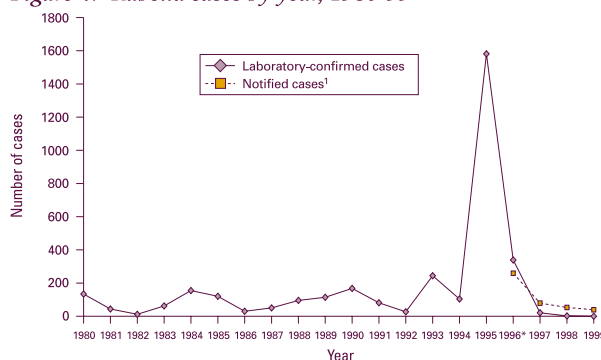


Figure 4: Rubella cases by year, 1980-99



Note: 1 Measles, mumps and rubella become notifiable diseases in June 1996

Surveillance data

National surveillance data - March 2000

Disease ¹	Current year - 2000 ²			Previous year - 1999			Trends - March 2000
	Mar 2000 cases	Cumulative total year-to-date	Current rate ³	Mar 1999 cases	Cumulative total year-to-date	Previous rate ³	
AIDS	2	5	0.8	7	9	0.8	
Acute gastroenteritis ⁴	68	156	16.6	75	158	15.4	
Campylobacteriosis	820	2716	238.6	616	2257	299.1	***
Cholera	0	0	0	0	0	0	
Creutzfeldt-Jakob disease	0	0	0	0	0	0	
Cryptosporidiosis	30	60	20.9	141	280	29.2	
Dengue fever	1	1	0.1	1	7	0.7	***
Giardiasis	169	455	46.5	221	568	58.3	***
<i>H influenzae</i> type b disease	2	3	0.3	0	1	0.3	
Hepatitis A	8	15	2.5	10	45	3.9	***
Hepatitis B (acute) ⁵	4	24	2.5	14	26	2.7	
Hepatitis C (acute) ⁵	5	21	2.5	14	25	2.6	
Hydatid disease	0	0	0.1	0	3	0.1	
Influenza ⁶	0	6	22.0	2	8	12.3	***
Lead absorption	12	32	4.0	11	38	2.8	**
Legionellosis ⁶	4	16	2.0	3	13	2.0	
Leprosy	0	0	0.1	3	5	0.2	
Leptospirosis	14	31	2.0	6	12	1.9	
Listeriosis	3	10	0.7	2	4	0.4	
Malaria	3	14	1.2	3	15	1.7	
Measles	7	24	3.0	8	20	3.2	
Meningococcal disease	25	73	14.1	24	72	12.0	*
Mumps	4	14	1.5	8	14	2.0	
Paratyphoid	1	2	0.4	1	5	0.5	
Pertussis	252	808	50.7	7	21	3.9	*** 1209
Rheumatic fever	7	18	1.5	17	29	2.5	**
Rubella	2	4	1.0	4	6	1.1	
Salmonellosis	212	470	47.3	311	837	59.6	***
Shigellosis	11	37	3.6	20	55	4.0	
Tetanus	0	0	0.1	1	3	0.1	
Tuberculosis	35	96	11.9	40	116	11.3	
Typhoid	2	8	0.4	0	4	0.7	
VTEC/STEC infection	16	26	2.0	7	17	1.6	
Yersiniosis	43	153	12.9	46	188	14.5	

Notes: 1 No cases of the following notifiable diseases were reported in March: anthrax, 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 March 2000) or the previous year (12 months to March 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 March 2000) and the previous year (12 months to March 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 - March 2000

Cases this month Current rate¹

Disease	Cases for March 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	Eastern BOP	Gisborne	Rotorua	Taupo	Taranaki	Ruapehu	Hawkes Bay	Wanganui	Manawatu	Wairarapa	Wellington	Hutt	Nelson-Marl	West Coast	Canterbury	South Cant	Otago	Southland
AIDS ³	0				0						1						1							
	1.2				0.3						1.1						0.4							
Acute gastroenteritis	0	4	5	22	0	0	0	0	7	0	0	0	0	0	0	4	0	0	0	21	0	0	0	0
	14.6	19.0	27.2	15.8	16.2	3.5	0	87.4	1.5	22.8	9.4	0	3.5	0	0	11.1	3.8	5.1	6.2	49.4	8.8	1.2	2.7	0
Campylobacteriosis	12	57	64	53	61	25	4	4	6	12	20	0	22	8	19	3	80	41	20	2	157	36	68	46
	127.7	239.7	268.7	192.0	268.4	141.9	95.5	155.2	170.5	162.9	175.0	125.4	280.2	171.0	134.3	205.4	334.3	251.1	171.5	148.0	311.4	401.1	273.3	262.3
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	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	0	0	0	0	0	0	0	0	0	0
Cryptosporidiosis	0	0	1	2	1	0	0	0	0	0	0	0	0	0	2	1	8	6	0	1	2	0	6	0
	11.7	10.4	9.0	8.5	42.0	17.7	8.0	15.3	0	3.3	13.1	0	34.8	8.1	32.6	28.6	16.5	18.1	6.0	6.2	41.6	78.0	17.4	23.4
Dengue fever	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	0.3	0.3	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0.3	0	0	0
Giardiasis	2	23	16	19	15	9	1	3	3	0	0	0	10	1	3	1	22	12	0	6	14	2	5	2
	25.5	60.6	70.9	35.4	63.1	61.2	15.9	32.8	31.0	68.4	18.7	35.8	72.5	24.4	33.9	26.0	55.6	41.5	31.7	61.7	44.5	27.7	29.0	18.9
H influenzae type b disease	1	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	1.5	0.5	0.6	0	1.8	0	0	0	0	0.9	0	0	0	0	0	0	0	0	0	0	0.8	0	0	0
Hepatitis A	0	0	1	3	1	1	0	0	0	0	1	0	1	0	0	0	0	0	0	0	0	0	0	0
	0.7	2.3	5.5	6.7	0.7	1.8	2.0	6.6	1.5	0	1.9	0	2.1	1.6	1.3	0	3.7	1.5	1.7	0	0.8	1.3	1.2	0.9
Hepatitis B	0	0	1	0	0	0	0	0	0	0	1	0	0	0	0	1	0	0	0	0	0	0	1	0
	2.2	0.3	2.6	2.3	5.0	2.7	0	4.4	3.1	3.3	0	11.9	5.6	1.6	0.7	0	1.6	4.5	1.7	0	3.4	6.3	1.7	1.8
Hepatitis C	0	1	0	0	0	1	0	0	2	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0
	3.6	0.5	0.9	0.6	0	15.1	4.0	0	13.9	0	0	0	4.9	0	1.3	0	1.2	1.5	0.9	0	7.2	5.0	2.9	0
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	0	0	0	0	0	2.2	0	0	0	0	0	0	0	5.2	0.4	0	0	0	0	0	0.6	0
Influenza ⁵	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	1.5	18.3	29.2	35.4	31.1	0.9	33.8	10.9	4.6	3.3	10.3	0	6.3	13.0	3.3	10.4	10.7	0	18.0	9.3	62.1	22.6	14.5	8.1
Lead absorption	0	0	1	0	0	0	0	0	0	1	0	0	1	4	0	0	0	0	0	2	1	0	2	2
	2.2	0.5	1.7	0.6	7.3	0.9	2.0	4.4	1.5	0	4.7	0	7.0	1.6	8.6	7.8	3.3	0.8	0.9	12.3	9.8	18.9	2.3	2.7
Legionellosis ⁵	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	2	0	1	0	
	0	0	0.9	0	7.6	0.9	2.0	0	3.3	1.9	6.0	1.4	0	0.7	10.4	1.6	3.8	0	0	5.2	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	0.7	0	0	0.8	0	0	0	0	0	0	0
Leptospirosis	2	0	0	1	0	0	0	0	0	1	1	1	0	0	0	0	0	2	1	2	1	0	2	
	9.5	0.5	0	3.6	0.9	0	2.2	0	3.3	5.6	6.0	6.3	3.3	3.3	0	0.4	0	3.4	9.3	1.6	5.0	0.6	2.7	
Listeriosis	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	3	0	0
	0.7	1.3	1.2	0.3	0.3	0.9	0	1.5	0	0	0	0	0	0.7	0	0	0.8	0.9	0	0.8	5.0	0	0.9	
Malaria	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	2	0	0	0	1	0
	2.2	0.5	1.7	0.9	1.3	0	4.0	2.2	0	0	0	0.7	1.6	2.0	0	0.4	0.8	4.3	0	2.1	0	2.3	0	
Measles	1	2	0	0	0	0	0	0	0	0	0	1	0	0	1	0	0	0	0	2	0	0	0	
	2.2	2.3	2.0	2.6	0.3	0.9	0	4.4	0	6.5	0	9.8	0	5.3	7.8	2.5	4.5	4.3	12.3	2.8	2.5	8.1	2.7	
Meningococcal disease	2	0	5	7	4	2	1	0	0	0	0	1	0	0	0	0	0	0	0	1	0	2	0	
	26.3	10.1	22.0	33.7	13.2	13.3	27.8	13.1	21.7	22.8	4.7	6.0	12.5	9.8	1.3	10.4	4.9	10.6	1.7	0	10.1	2.5	13.9	15.3
Mumps	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0	0	2	0	0	1	0	0	0	
	1.5	0	1.4	2.6	0	2.7	0	0	3.1	9.8	3.7	0	3.5	0	2.0	0	0.8	3.0	1.7	3.1	1.3	0	2.9	0.9
Paratyphoid	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	0.6	0.7	0	0	0	1.5	0	0	0	0.7	0	1.3	0	0.4	0	0	0	0.5	0	0.6	0	
Pertussis	4	18	20	12	7	5	1	0	2	0	1	0	1	0	3	4	6	5	73	1	70	3	9	7
	23.3	44.6	18.5	29.9	30.4	17.7	4.0	4.4	3.1	0	19.7	6.0	14.6	3.3	5.3	13.0	35.0	33.9	167.2	18.5	112.8	132.0	53.3	286.6
Rheumatic fever	2	0	0	2	0	0	0	0	0	0	0	2	0	0	0	1	0	0	0	0	0	0	0	0
	13.1	0	0.6	0.6	2.6	3.5	0	8.7	3.1	3.3	0.9	6.0	2.1	0	2.6	2.1	1.5	0	0	0	0	0	0	
Rubella	1	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	3.6	1.0	0.6	0.6	0	0.9	0	0	0	0.9	0	1.4	0	0	0	0.8	0	2.6	6.2	0.3	0	5.2	0.9	
Salmonellosis	6	19	14	15	11	6	1	2	1	1	6	0	8	2	5	6	46	13	2	0	22	10	9	7
	29.2	43.4	44.5	29.3	46.3	36.3	25.9	39.3	44.9	22.8	29.9	35.8	45.3	17.9	65.8	88.4	73.3	44.5	42.9	21.6	53.3	93.0	54.4	75.5
Shigellosis	0	2	2	2	0	0	0	0	1	0	0	0	0	0	0	2	1	0	0	0	0	1	0	
	5.8	4.1	9.3	7.0	2.3	0	6.0	4.4	7.7	19.5	0.9	0	0	0	1.3	2.6	3.3	4.5	0	0	0.8	2.5	1.2	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	2.2	0	0	0	0	0.7	0	0	2.6	0	0	0	0	0	0	0	0	0
Tuberculosis	0	2	5	9	4	1	2	0	2	0	0	0	2	0	1	0	6	0	0	1	0	1	0	0
	16.8	10.9	27.8	22.8	9.9	11.5	9.9	2.2	6.2	0	0	0	11.2	8.1	7.3	0	18.5	18.9	3.4	3.1	5.7	2.5	2.9	1.8
Typhoid	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0	0
	0.7	0.8	1.2	0.9	0	0.9	0	0	0	0	0	0	0	0	0	0.4	0	0	0	0	0	0	0	0
VTEC/STEC infection	0	0	0	5	0	1	0	0	4	1	0	0	0	0	0	0	0	0	0	3	0	2	0	
	0	0	0.9	0.3	9.9	3.5	4.0	2.2	1.5	16.3	1.9	0	0	0	0.7	0	2.1	1.5	0	0	3.1	0	2.3	0
Yersiniosis	2	11	5	2	1	0	1	0	0	1	0	0	3	0	1	0	2	3	1	2	5	2	1	0
	9.5	14.5	19.4	12.9	12.2	8.0	9.9	6.6	10.8	13.0	6.6	0	18.1	3.3	5.3	2.6	16.1	19.6	7.7	37.0	16.3	27.7	1.7	3.6

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

Surveillance data

National surveillance data - April 2000

Disease ¹	Current year - 2000 ²			Previous year - 1999			Trends - April 2000
	Apr 2000 cases	Cumulative total year-to-date	Current rate ³	Apr 1999 cases	Cumulative total year-to-date	Previous rate ³	
AIDS	5	10	0.9	2	11	0.8	
Acute gastroenteritis ⁴	44	233	17.9	31	189	15.5	*
Campylobacteriosis	567	3300	240.4	521	2770	290.1	***
Cholera	0	0	0.0	0	0	0.0	
Creutzfeldt-Jakob disease	1	1	0.1	0	0	0.0	
Cryptosporidiosis	17	77	18.3	111	391	27.9	***
Dengue fever	0	1	0.1	1	8	0.7	***
Giardiasis	114	572	44.6	186	751	57.7	***
<i>H influenzae</i> type b disease	1	4	0.3	2	3	0.3	
Hepatitis A	7	22	2.5	5	50	3.7	**
Hepatitis B (acute) ⁵	2	26	2.3	10	36	2.8	
Hepatitis C (acute) ⁵	5	26	2.3	12	37	2.7	
Hydatid disease	0	0	0.1	0	3	0.1	
Influenza ⁶	0	6	21.7	11	19	12.6	***
Lead absorption	12	44	3.9	17	56	3.2	
Legionellosis ⁶	5	21	2.0	4	17	1.9	
Leprosy	0	0	0.1	0	5	0.2	
Leptospirosis	7	39	2.2	1	13	1.6	
Listeriosis	2	12	0.7	1	5	0.4	
Malaria	3	17	1.2	3	18	1.6	
Measles	5	30	3.0	8	28	3.2	
Meningococcal disease	36	104	14.2	25	97	12.0	**
Mumps	5	18	1.5	4	18	1.9	
Paratyphoid	1	3	0.4	2	7	0.6	
Pertussis	224	1031	56.4	17	37	4.0	*** 1300
Rheumatic fever	2	20	1.5	3	32	2.4	**
Rubella	1	4	0.9	2	8	1.1	
Salmonellosis	169	639	44.9	254	1091	61.0	***
Shigellosis	4	41	3.3	12	67	4.0	
Tetanus	0	0	0.1	0	3	0.1	
Tuberculosis	22	120	11.9	24	140	11.1	
Typhoid	1	9	0.4	0	4	0.6	
VTEC/STEC infection	6	32	1.9	11	28	1.6	
Yersiniosis	15	168	12.5	30	218	14.0	

Notes: 1 No cases of the following notifiable diseases were reported in April: anthrax, brucellosis, cysticercosis, diphtheria, 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 April 2000) or the previous year (12 months to April 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 April 2000) and the previous year (12 months to April 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 - April 2000

Cases this month Current rate¹

Disease	Cases for April 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	Eastern 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						1						0							
	1.4				0.3						1.2						0.3							
Acute gastroenteritis	0	1	3	1	0	0	0	0	0	0	0	0	0	0	0	2	0	0	0	8	0	24	2	
	13.9	17.8	27.5	15.5	16.5	2.7	0	87.4	1.5	120.5	9.4	0	3.5	0	0.7	0	11.5	3.8	2.6	0	49.7	6.3	15.1	4.5
Campylobacteriosis	10	35	56	46	32	10	2	4	3	6	8	1	10	1	10	7	55	7	11	6	146	19	44	38
	129.1	233.6	261.7	196.7	265.7	140.1	95.5	163.9	155.0	182.4	174.1	125.4	270.4	164.4	133.6	210.6	337.6	239.8	175.8	163.4	328.3	409.9	289.0	283.0
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	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	0	0	0.3	0	0	0.9	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Cryptosporidiosis	0	5	3	3	1	0	0	0	0	0	0	0	0	0	0	3	0	0	0	0	2	0	0	0
	10.9	9.4	9.0	8.2	38.3	16.0	8.0	10.9	0	3.3	13.1	0	30.7	6.5	27.3	28.6	15.2	16.6	6.0	6.2	28.7	76.7	16.8	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	0	0	0
Giardiasis	2	17	17	12	12	4	1	2	1	1	2	0	3	0	2	1	11	3	1	3	13	0	4	2
	25.5	56.8	70.6	35.4	62.1	60.3	17.9	35.0	24.8	65.1	17.8	35.8	66.9	22.8	27.9	15.6	54.8	39.2	28.3	52.4	41.4	23.9	30.7	20.7
H influenzae type b disease	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	1.5	0.3	0.6	0	0	2.7	0	0	0	0	0.9	0	0	0	0	0	0	0	0	0	0.5	1	0	0
Hepatitis A	0	0	2	1	0	2	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	1	0
	0.7	2.0	5.8	6.7	0.7	3.5	2.0	6.6	1.5	0	1.9	0	2.1	1.6	1.3	0	3.7	0.8	1.7	0	0.8	2.5	1.2	0.9
Hepatitis B	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1
	2.2	0.3	2.6	1.8	4.0	3.5	0	4.4	3.1	3.3	0	11.9	4.2	1.6	0.7	0	1.6	4.5	0.9	0	3.1	6.3	1.7	1.8
Hepatitis C	0	0	0	0	0	2	0	0	0	0	0	0	1	0	0	0	0	0	0	0	2	0	0	0
	3.6	0.5	0.6	0.3	0	15.1	4.0	0	13.9	0	0	0	5.6	0	1.3	0	1.2	1.5	0.9	0	5.9	3.8	2.9	0
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	0	0	0	0	0	2.2	0	0	0	0	0	0	0	5.2	0.4	0	0	0	0	0	0.6	0
Influenza ⁵	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	1.5	17.8	28.6	35.1	29.7	0.9	33.8	10.9	4.6	3.3	10.3	0	6.3	13.0	2.0	10.4	10.7	0	18.0	9.3	62.1	22.6	14.5	8.1
Lead absorption	0	0	1	0	1	0	0	1	0	0	0	0	2	0	0	2	0	1	0	0	2	1	1	0
	1.5	0.5	1.7	0.6	5.6	0	2.0	6.6	1.5	0	4.7	0	7.0	1.6	8.0	7.8	3.7	0.8	1.7	12.3	9.8	18.9	2.3	2.7
Legionellosis ⁵	1	0	1	0	1	0	0	0	0	0	0	0	0	0	0	2	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	10.4	2.5	3.8	0	0	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	0.7	0	0.7	0	0	0.8	0	0	0	0	0	0
Leptospirosis	0	0	0	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	2	2	0
	9.5	0.5	0	0.3	4.0	1.8	0	2.2	0	3.3	5.6	6.0	6.3	3.3	3.3	0	0.4	0	3.4	9.3	1.6	7.5	1.7	2.7
Listeriosis	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1
	0.7	1.5	0.9	0.3	0.3	0.9	0	0	1.5	0	0	0	0	0	0.7	0	0	0.8	0.9	0	0.8	5.0	0	1.8
Malaria	0	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	2.2	0.8	2.0	1.2	1.0	0	4.0	0	0	0	0	0	0.7	1.6	1.3	0	0.4	0.8	4.3	0	2.1	0	2.3	0
Measles	0	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	1	1	0	0	0	0	1	0
	2.2	2.5	1.7	2.6	0	0.9	0	2.2	0	6.5	0	8.4	0	5.3	7.8	2.1	6.0	5.1	12.3	2.6	2.5	8.7	2.7	
Meningococcal disease	3	2	7	13	2	0	1	0	0	0	0	0	2	0	0	0	1	1	1	0	0	0	3	0
	27.0	10.1	22.8	35.7	12.2	10.6	23.9	13.1	20.1	22.8	4.7	6.0	13.9	8.1	1.3	10.4	5.4	11.3	2.6	0	9.6	2.5	15.1	15.3
Mumps	0	0	1	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	1	0	1	0	0
	1.5	0	1.7	2.6	0	2.7	0	0	3.1	9.8	3.7	0	2.8	0	2.0	0	0.8	1.5	2.6	3.1	1.6	0	2.9	0.9
Paratyphoid	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	0.6	0.7	0	0	0	0	0	0	0	0.7	0	1.3	0	0.4	0.8	0	0	0.5	0	0.6	0
Pertussis	3	11	5	6	35	8	1	2	0	2	0	2	0	2	23	2	11	27	8	67	1	7	1	
	24.8	47.7	20.0	31.3	41.0	24.8	6.0	8.7	3.1	0	21.5	6.0	15.3	3.3	6.6	72.8	32.9	40.7	188.7	43.2	129.9	130.8	57.3	288.4
Rheumatic fever	0	0	0	0	1	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0
	12.4	0	0.6	0.6	2.6	3.5	0	8.7	1.5	3.3	0.9	6.0	2.1	0	5.2	2.1	1.5	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
	2.9	1.0	0.6	0.6	0	0.9	0	0	0	0.9	0	1.4	0	0	0	0.8	0	2.6	6.2	0.3	0	4.6	0.9	
Salmonellosis	6	22	4	8	11	4	3	0	1	0	2	0	3	3	5	25	7	6	2	1	20	5	21	10
	24.8	40.3	35.0	27.2	42.6	34.6	29.8	39.3	41.8	19.5	25.3	29.8	43.9	19.5	64.5	148.2	65.0	40.7	39.4	24.7	49.4	93.0	61.4	78.2
Shigellosis	0	2	0	1	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	5.1	4.3	7.8	6.7	2.0	0	6.0	6.6	7.7	19.5	0.9	0	0	0	1.3	2.6	2.9	3.8	0	0	0.8	2.5	1.2	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	2.2	0	0	0	0	0	0.7	0	2.6	0	0	0	0	0	0	0	0	0
Tuberculosis	0	2	6	1	0	0	0	0	0	0	0	0	3	0	1	0	4	4	0	0	1	0	0	0
	16.8	10.7	26.3	21.9	9.3	11.5	9.9	2.2	6.2	0	0	0	13.2	13.0	8.0	0	19.3	20.4	2.6	3.1	5.9	2.5	2.9	0.9
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	1.2	1.2	0	0.9	0	0	0	0	0	0	0	0	0	0.4	0	0	0	0	0	0	0	0
VTEC/STEC infection	0	1	0	2	1	0	0	0	1	0	0	0	0	0	0	1	0	0	0	0	0	0	0	0
	0	0.3	0.6	0.9	8.3	1.8	4.0	2.2	1.5	19.5	1.9	0	0	0	0	2.5	0.8	0	0	3.1	0	2.3	0	
Yersiniosis	0	3	3	2	2	1	0	0	0	0	0	0	0	0	0	2	0	0	0	1	0	0	1	
	8.0	14.2	18.8	13.2	12.2	8.9	9.9	4.4	10.8	13.0	4.7	0	18.1	3.3	4.7	2.6	15.2	18.1	7.7	30.8	16.0	25.1	1.7	4.5

Highlighting the magnitude and nature of injuries due to medical error

This paper reviews the prevalence, consequences, and common types of medical error; which clinicians make errors; and the risk factors that increase the likelihood of injury from error. A benchmark 1984 Harvard study, and a 1995 Australian study, provide the only population level data on the rates of injuries to hospitalised patients. In the Harvard study, 3.7% of hospital admissions suffered an adverse event, compared with 16.6% in the Australian study. Both studies found that the majority of adverse events were caused by errors or were preventable. In the United States, medical error results in 44 000-98 000 unnecessary deaths each year and 1 000 000 excess injuries. About half of the adverse events occurring among inpatients result from surgery – perhaps because such mishaps are hard to disguise. Complications from drug treatment, therapeutic mishaps, and diagnostic errors

are the most common non-operative adverse events. Mistakes may be more common when the clinician is inexperienced and when new techniques are introduced. Older age, complex care, urgent care, and a prolonged hospital stay are associated with more errors (Weingart SN, Wilson RM, Gibberd RW, et al. *Epidemiology of medical error*. *BMJ* 2000; 320: 774-7).

Editorial note: This issue of the *BMJ* was devoted entirely to the topic of reducing error and improving safety in healthcare. Another review included in the journal illustrates that age itself is not an independent predictor of preventable adverse events, and suggests that the association is probably due to the clinical complexity of the care of the elderly. Research into adverse events in New Zealand hospitals, along the lines of the Harvard and Australian studies, is currently underway.

Risk factors for yersiniosis

An Auckland study has identified that the major risk factors for *Yersinia enterocolitica* infection (yersiniosis) include not being on a town water supply, not having a reticulated sewerage connection, and consumption of pork. The prospective case-control study included 186 cases, from whom *Y enterocolitica* was isolated from a faecal specimen between April 1995 and June 1996, and 379 randomly selected age-matched controls. Population attributable risk analysis suggested that 89% of *Y enterocolitica* infections could be prevented if the risk exposures identified in the study were removed. To reduce the incidence of yersiniosis, the authors recommend audit of the processes involved in the slaughter and subsequent handling of pork to ensure adequate controls to minimise the risk of contamination with *Yersinia*, public education on the need to cook pork thoroughly, registration of small community water

supplies to ensure adequate treatment, and provision of advice on appropriate treatment of private water supplies (Satterthwaite P, Pritchard K, Floyd D. A case-control study of *Yersinia enterocolitica* infections in Auckland. *Aust NZ J Public Health* 1999; 23: 482-5).

Editorial note: Yersiniosis became notifiable in New Zealand in June 1996. In 1999 there were 503 notifications of yersiniosis, and it was the 6th most common notifiable enteric disease. Analysis of risk factors recorded for the 1537 cases notified between 1997 and 1999 showed that 145 (9.4%) cases had definite or suspected contact with contaminated food or drink. Of these, 41 cases had consumed pork. Consumption of private supply water and/or untreated water was recorded for 28 cases, consumption of ham for 18 cases and consumption of luncheon or sausage meat for 16 cases.

Travel health

Unusual outbreak of Q fever following a safari trip

This paper reports an outbreak of four cases of Q fever among a group of 50 safari travellers to Kenya. Two cases had overt infection, while the other two had asymptomatic illness which was detected by serological testing. All four cases had entered or looked inside a tribal shack, which was made of cattle hides and housed goats. This shack may have been the source of their infection (Sandler RH, Bolte ER, Chez MG, et al. *Clin Infect Dis* 2000; 30: 214-5).

Editorial note: Q fever is an acute rickettsial disease caused by infection with *Coxiella burnetii*. It is a febrile illness of variable severity with non-specific symptoms, including chills, sweats, headache, weakness and malaise. A wide range of domestic and wild animals and birds can act as reservoirs. Airborne dust, from areas contaminated with animal tissues and excreta, is the common mode of transmission of *C burnetii*. The organism may also be transmitted by direct contact with infected animals and contaminated areas. New Zealand is one of the few countries where Q fever is not endemic. Therefore, any confirmed cases need to be carefully investigated. Travellers should be advised to avoid contact with farm and other animals to reduce their risk of zoonotic illnesses.

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