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Epidemiology of tetanus in New Zealand reinforces value of vaccination

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This paper reports on New Zealand's first infant tetanus case for 35 years. It also reviews long-term trends in tetanus epidemiology using notification data for the period 1921-2000 and hospitalisation and mortality data for the period 1948-2000. Prior to 1960, there was a median of 21 cases per year. Following the introduction of routine infant immunisation against tetanus in 1960, the number of cases fell progressively to a median of two per year. The number of tetanus hospitalisations and deaths also fell markedly over time. Before 1960 most cases were male and age specific rates were highest for children less than 15 years. After 1960 there was a 99% reduction in disease rates among children. In the last twenty years, approximately half (48.3%) of all cases have been over the age of 65 years and nearly two thirds (63.8%) have been female. The case described here is a 15 month unvaccinated female from Northland admitted to hospital in April 2001 with a history of fever and breathing difficulties. She developed trismus and opisthotonos and a diagnosis of tetanus was made. She received human tetanus immune globulin and supportive treatment and required ventilation for 11 days. These findings highlight two important requirements for the prevention of tetanus: the need to achieve high coverage levels in the childhood population, and the importance of adult vaccination, particularly for those over 65 years of age who may have missed primary tetanus vaccination.

Tetanus is an acute disease caused by a neurotoxin produced by the anaerobic spore-forming bacterium *Clostridium tetani*. *C. tetani* spores are ubiquitous in the environment and are a normal inhabitant of soil and animal and human intestines. After entering the body through a wound, the spores germinate and produce toxins. These toxins interfere with the release of neurotransmitters at inhibitory nerve terminals resulting in unopposed muscle contraction and spasm.

The incubation period for tetanus varies from three days to three weeks, usually around eight days. Longer incubation periods are associated with more peripheral injury sites. The onset of symptoms is gradual, over one to seven days, and is characterised by painful muscle contractions mainly in the neck and face but

also in the trunk. In general, the shorter the incubation period the more severe the disease and higher the risk of death.¹

Three different clinical forms of tetanus have been described. Generalised tetanus is the most common and is characterised by increased muscle tone and generalised spasms. These spasms

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are violent and painful and may threaten ventilation. Sustained contraction of the facial muscles results in the classic sign of risus sardonicus. Sustained contraction of the back muscles produces an arched back (opisthotonos). Generalised tetanus occurring in newborns is termed neonatal tetanus (NT). Local tetanus is uncommon and is characterised by persistent contraction in the same area as the injury. Cephalic tetanus is rare and involves cranial nerves, particularly in the facial area.

Effective individual protection against tetanus can only be achieved through active immunisation. Unlike other vaccine preventable diseases, there is no possibility of herd immunity and immunity cannot be naturally acquired. Vaccination with tetanus toxoid stimulates production of antibodies which act against the toxin produced by the organism thus providing protection against the consequences of infection rather than infection itself.

Tetanus occurs world-wide but is more common in hot, damp climates with soil rich in organic matter.¹ Tetanus, particularly NT, remains a significant public health problem in non-industrialised countries, causing an estimated 400 000 deaths each year.² NT is attributable to low maternal immunity in combination with non-sterile delivery and certain traditional umbilical cord care practices.^{3,4} In industrialised countries tetanus has become very uncommon, particularly among infants and children, due to effective childhood immunisation programmes.

Here we report New Zealand's first case of infant tetanus in almost 35 years, review long-term trends in tetanus incidence and distribution, and review the implications for tetanus prevention and control.

Surveillance methods

Tetanus has been notifiable and reported regularly in New Zealand since 1921. Historical notification data for the years 1921-2000 were obtained from ESR records and annual reports of the Department of Health. Hospitalisation and death data from 1948 onwards were obtained from the New Zealand Health Information Service National Minimum Dataset. Case fatality rates were calculated as the number of deaths divided by the number of hospitalised cases. Average annual age-specific rates for the periods 1941-1960, 1961-1980 and 1981-2000 were calculated using midpoint population census data from 1951, 1971 and 1991 respectively. Census data from 1936 were used for calculating rates from the earliest period (1921-1940), as data were not available for 1931.

Results

Case report: On April 10 2001, a 15 month old unvaccinated female Maori infant was admitted to Whangarei hospital with a history of fever, sore mouth and wheeze with difficulty breathing. She had received recent oral antibiotic treatment for respiratory tract infection. There was no history of accidental drug ingestion or recent trauma. She was initially treated for asthma and lower respiratory tract infection. During admission, she was observed to have unusual short cyanotic episodes, each lasting about five seconds, which later developed into "arched back and gasping" episodes lasting about 20 seconds. Intubation was required to protect the airway. Direct inspection of the pharynx and glottis and a computerised tomogram (CT) of the neck revealed nonspecific

soft tissue swelling at the level of the adenoids but no abscess. CT of the head was normal. Chest CT revealed extensive collapse and consolidation of the right upper and left lower lung lobes.

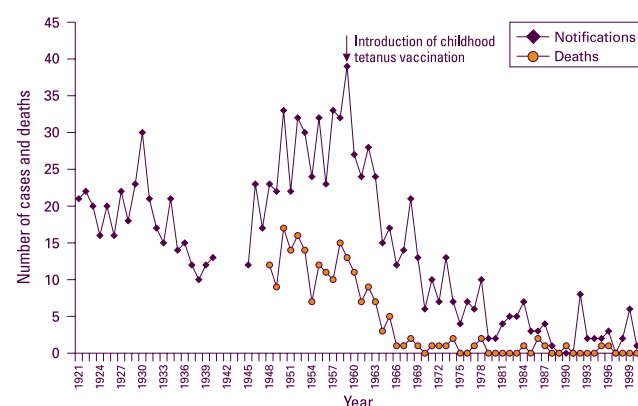
Serum chemistry including calcium and metabolic screen, blood cultures, urine microscopy and toxicology, lumbar puncture, electroencephalogram and magnetic-resonance imaging of the head were all normal. A superficial splinter was found on the medial arch of the left foot and there was superficial cracking of the flexure creases of the toes, which were debrided.

The patient developed trismus and opisthotonos and a diagnosis of tetanus was made on day three following which human tetanus immune globulin (HTIG) 3000 units was administered together with antibiotics. Once alternative diagnoses were excluded by the above investigations, antibiotics were rationalised to metronidazole and benzodiazepines were administered to control the tetanic spasms. Supportive care included airway protection and morphine infusion to counteract autonomic effects. She made a slow recovery and was extubated on day 14, brief spasms were treated with rectal diazepam as needed and she was finally discharged home on day 24. The primary vaccination series was initiated and subsequent catch-up programme was planned.

Epidemiology of tetanus in New Zealand: During the 80-year period from 1921 to 2000 there were 1082 notified cases of tetanus, of which 70% (755/1082) occurred between 1921-1960 (*Figure 1*). In this pre-vaccine period the average annual notification rate was 1.1 per 100 000 with a median of 21 cases per year. The number of cases fell progressively after 1960 following the introduction of routine infant immunisation against tetanus. The median number of tetanus cases per year for the decades 1961-1970, 1971-1980, 1981-1990 and 1991-2000 were nineteen, seven, four and two respectively. The average annual notification rate for the last 10 years (1991-2000) was 0.07 per 100 000, a 94% drop in incidence compared with the pre-vaccine period. The number of hospitalised cases of tetanus exceeded the number of notified cases for each year reviewed. The average annual hospitalisation rate for the period 1961-1980 was 0.64 per 100 000, which was 1.5 times higher than the notified rate for that period. This fell to 0.19 per 100 000 for the period 1981-2000, which was twice the notified rate for that period.

The number of tetanus deaths also fell markedly over time. Between 1948 and 1960, there was a median of 12 deaths per

Figure 1: Notified tetanus cases and deaths, 1921-2000*

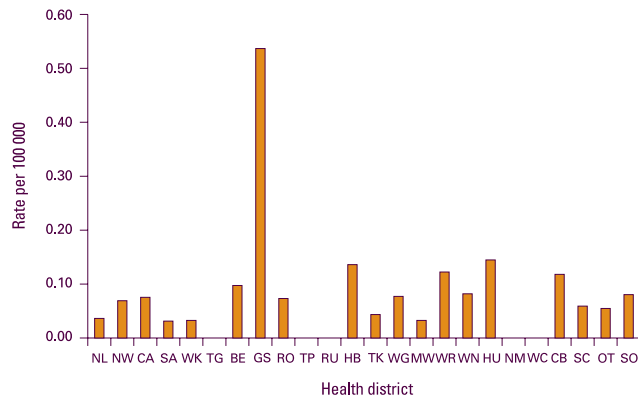


* Notification data unavailable for 1941-44 inclusive, death data available from 1948 onwards

year. There has been a reduction in the average case fatality rate over time from 38% in the period 1948-1960, to averages of 11% and 8% for the periods 1961-1980 and 1981-2000 respectively. There has been no more than one reported tetanus death per year during the last twenty years, with the exception of 1986 when two tetanus deaths were reported.

Between 1981 and 2000, the average annual notification rate was highest in Gisborne (0.54 per 100 000) (Figure 2). Hawkes Bay, Wairarapa, Hutt and Canterbury health districts all reported rates between 0.1 and 0.2 per 100 000. All other health districts reported rates of less than 0.1 per 100 000.

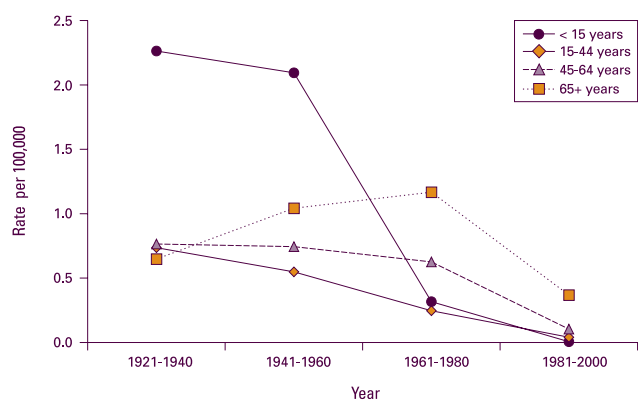
Figure 2: Tetanus notifications by health district, 1981-2000



Prior to 1960, age specific notification rates were highest for children aged less than 15 years (2.2 per 100 000) (Figure 3). This group accounted for 40% of all cases. After 1960 there was a marked reduction in disease rates among children. Between 1981 and 2000, rates for children were the lowest of any age group (0.006 per 100 000) and accounted for less than 2% of all cases, a 99% decline from the pre-vaccination period.

Adult disease rates also declined over the 80-year period but this reduction was much less dramatic. Adults, particularly those over the age of 65 years, accounted for an increasing proportion of cases over time. Between 1921 and 1940, this group accounted for 3.6% of all cases. In the last twenty years nearly half (48.3%) of all cases have occurred in this age group.

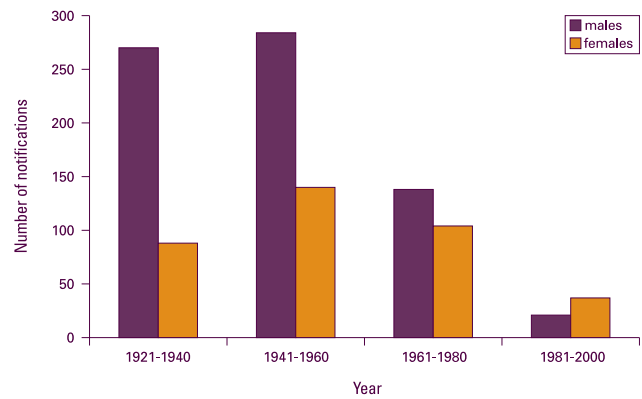
Figure 3. Tetanus age-specific notification rates, 1921-2000



Males accounted for nearly two thirds (713/1082) of all tetanus cases between 1921 and 2000. However, the ratio of male to female cases changed markedly over time (Figure 4). Between 1921 and 1940 the male to female ratio was 3.0:1, falling to 2.0:1 between 1941 and 1960 and 1.3:1 between 1961 and

1980. In the last 20 years, the number of female tetanus cases has outnumbered male cases (male to female ratio 1:1.7).

Figure 4: Tetanus notifications by sex, 1921-2000



Discussion

This review of long-term tetanus epidemiology highlights the significant impact of the tetanus vaccination programme in this country. Prior to 1960, tetanus was primarily a disease of children. Boys were particularly at risk, presumably because they were more likely to engage in activities associated with injury. While improvements in the management of wounds and the use of anti-tetanus serum are likely to have had some impact on disease,⁵⁻⁷ the most dramatic reduction in incidence came after 1960 when routine vaccination was introduced for all infants. The initial schedule recommended doses at three, four and five months of age with a booster dose at five years. In 1964 a booster dose at 18 months was added.⁸ During the subsequent twenty years, childhood tetanus was virtually eliminated.

Because the disease is now very rare, a diagnosis of tetanus may not be initially suspected. This is especially true for children. Laboratory tests are of limited value as the organism may be isolated from a wound in as few as 30% of cases.¹ Furthermore, *C. tetani* can be isolated from patients who do not have the disease. Diagnosis therefore relies on clinical criteria. The current clinical case definition for tetanus is acute onset of hypertonia and/or painful muscle contractions, most commonly of the jaw or neck, which may proceed to generalised muscle spasms.⁹ Clinical signs may be non-specific, particularly in the early stages of the disease and in neonates who may present with apnoea or tonal abnormalities without the classic opisthotonos. Failure by clinicians to consider and/or report the disease is likely to result in undernotification of tetanus cases. The discrepancy between hospitalisations and notifications is likely to be due to both under-reporting of cases and coding errors. Case-fatality rates may be over-estimated as case ascertainment is likely to be higher for fatal disease than for milder disease.

The current tetanus notification rate in this country (0.07 per 100 000) is higher than that reported from Australia (0.04 per 100 000),¹⁰ and the US (0.02 per 100 000).¹¹ In England and Wales there has been an average of nine tetanus notifications each year between 1980 and 2001¹² which represents a rate of 0.01 per 100 000. In all developed countries, tetanus has become a disease of older adults.¹⁰⁻¹⁴ In New Zealand, individuals over the age of 65 years currently have the highest incidence of any age group with the majority of these cases being women. Similarly, in Australia between 1976 and 1995, 80% of cases

were individuals over 55 years of age and 60% were women.¹⁵ In both countries this most likely reflects the fact that these individuals were born before the introduction of childhood vaccination, that the population aged over 55 contains more women than men, and also that women missed out on vaccinations preferentially administered to troops during the Second World War.¹⁶

Routine adult tetanus vaccination was introduced in New Zealand in 1971⁸ and since 1994 diphtheria-tetanus boosters have been recommended every 10 years.¹⁷ However, adult population coverage is thought to be poor as vaccination is not actively promoted in this age group and is often triggered only by an injury event. In a recent review of 15 tetanus cases, none gave a history of having received a full course of tetanus immunisation prior to the illness; four had not received any prior tetanus and seven had received only one dose of tetanus toxoid.¹⁸ Adults who have not received a primary course of tetanus vaccine should have three doses of combined tetanus-diphtheria vaccine at least one month apart. From 2002, adult boosters will be recommended at 45 and at 65 years of age. The hope is that age-specific recommendations may enable primary care providers to link vaccination with the delivery of other preventive health measures, thereby encouraging greater uptake.¹⁷ This recommendation will also ensure that unnecessary boosters causing considerable local adverse reactions are avoided.

Ensuring adequate immunity to tetanus is important, as precipitating injuries may not always come to the attention of clinicians. Certain types of wounds associated with the growth of tetanus may be clearly apparent (eg, compound fractures, burns and other wounds with extensive damage) and are likely to receive appropriate wound management and followup vaccination. However, a significant proportion of precipitating injuries are trivial and remain unnoticed as illustrated by this case. Recent reports also highlight the need to be aware of the new "rusty nails". In the US, a recent increase in the proportion of young adult cases has been attributed in part to an increased number of cases among injecting drug users in California.^{11,12} The recent report of a case of cephalic tetanus in a young woman who had her tongue pierced²⁰ emphasises the importance of emerging risk factors, including non-sterile body piercing. Details of the current recommendations for vaccination and HTIG regimens following injury are contained in the Immunisation Handbook.¹⁷

This case report and the current distribution of tetanus cases in New Zealand reinforces two key immunisation measures for medical practitioners: the importance of adult vaccination, particularly for those over 65 years who may have missed primary vaccination, and the need to raise coverage levels in the childhood population. In 1996, an immunisation coverage survey in Northland and Auckland indicated that only 63% of children were fully immunised by the age of 2 years.²¹ Continuing low levels of vaccination coverage among children in this country mean that further cases of tetanus similar to the one reported here are inevitable. It is important that clinicians notify all cases of suspected tetanus to improve surveillance of this disease.

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AIDS and HIV infection in 2000

The number of AIDS cases notified in 2000 (27) was similar to that in 1999 (30) (Figure 1). Eight-eight people were newly diagnosed with HIV infection in 2000, a 23.9% increase from the 71 cases diagnosed in 1999. By the end of 2000, 729 cases of AIDS had been notified in New Zealand since surveillance began in 1984, and 1478 people had been diagnosed with HIV infection since testing became possible in 1985. There were 13 deaths from AIDS in 2000.

Figure 1: AIDS notifications and diagnosed HIV infections by year, 1984-2000

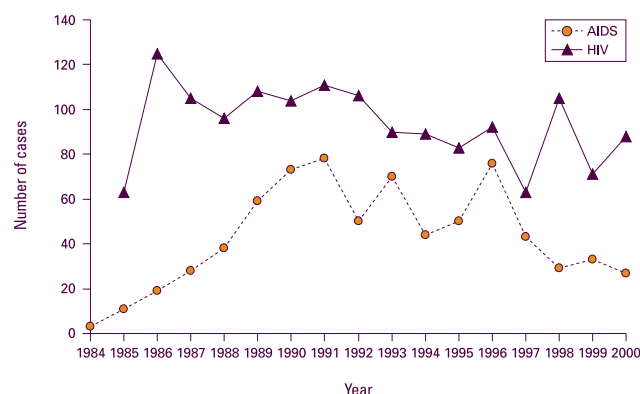


Table 1 shows the most likely means of infection for people notified with AIDS and diagnosed with HIV infection in 2000 and in total up until the end of 2000. Homosexual contact remains the predominant risk behaviour category for new AIDS diagnoses (79.4%), but is relatively less important for new cases of HIV infection (34.0%). Heterosexual contact was responsible for 42.0% of new cases of HIV infection in 2000, an increase from 35.2% in 1999. Most of these new cases of HIV infection (89.2%, 33/37) occurred overseas. Of all the HIV infections newly identified in 2000, 56.8% (50/88) were reported to have occurred overseas.

While combination therapy is reducing the number of people with HIV progressing to AIDS, resistance to these drugs is developing and there is concern that the perception that HIV is now treatable is leading to less safe sex being practised. Health education on sexually transmitted infections must be

Table 1: Exposure category of people notified with AIDS and people with diagnosed HIV infection in 2000

Risk behaviour category	Sex	AIDS				HIV antibody positive ¹			
		12 months to 31.12.00		Total to 31.12.00		12 months to 31.12.00		Total to 31.12.00	
		Cases	%	Cases	%	Cases	%	Cases	%
Homosexual contact	Male	17	63.0	579	79.4	30	34.0	778	52.6
Homosexual & IDU	Male	0	0.0	10	1.4	2	2.3	15	1.0
Heterosexual contact	Male	4	14.8	40	5.5	18	20.5	118	8.0
	Female	1	3.7	28	3.8	19	21.6	135	9.1
Injecting drug user (IDU)	Male	1	3.7	13	1.8	1	1.1	32	2.2
	Female	0	0.0	5	0.7	0	0.0	8	0.5
Blood product recipient	Male	1	3.7	16	2.2	0	0.0	29	2.0
	Female	0	0.0	1 ²	0.1	1	1.1	6	0.4
Transfusion related	Male	0	0.0	1 ²	0.1	0	0.0	5	0.3
	Female	0	0.0	1 ²	0.1	0	0.0	6	0.4
Perinatal	Unknown	0	0.0	0	0.0	0	0.0	5	0.3
	Male	0	0.0	1	0.1	0	0.0	6	0.4
	Female	1	3.7	3	0.4	2	2.3	6	0.4
Awaiting information/undetermined	Male	0	0.0	29	4.0	8	9.1	284	19.2
	Female	1	3.7	2	0.3	4	4.5	27	1.8
	Unknown	0	0.0	0	0.0	0	0.0	14	0.9
Other	Male	0	0.0	0	0.0	0	0.0	2	0.1
	Female	1	3.7	1	0.1	3	3.4	7	0.5
TOTAL		27	100	729	100	88	100	1478	100

Notes: ¹ Includes people who have developed AIDS

² Occurred overseas

integrated into broader messages on sexual health, and messages must be culturally appropriate to their audience (reported by AIDS Epidemiology Unit, University of Otago).

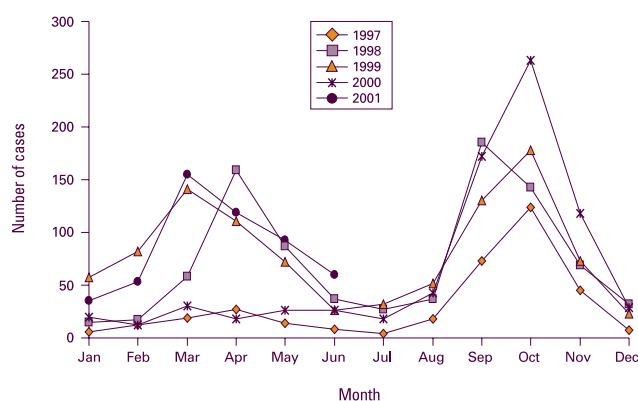
Cryptosporidiosis autumn peak in 2001 higher than in previous years

A total of 60 cases of cryptosporidiosis were notified in June, bringing the total for the first six months of 2001 to 520 cases. This is the highest total for the first six months of the year since cryptosporidiosis became notifiable in 1996. The total number of notified cryptosporidiosis cases for the first six months of the year was 132 in 2000, 489 in 1999, 373 in 1998 and 86 in 1997.

During the twelve months to 31 June 2001, rates of notified cryptosporidiosis cases exceeded those of the national rate (32.1 per 100 000) in Hawkes Bay (111.5), South Canterbury (78.0), Rotorua (69.7), Taupo (48.9), Waikato (47.3), Gisborne (37.2), Wellington (37.1), Southland (36.8), Wanganui (35.8), and Hutt (35.4) health districts.

Monthly numbers of cryptosporidiosis notifications typically show two seasonal peaks during the year, as shown in Figure 2. Most cases (52.9% in 1997-2000) occur during a peak in spring (September to November), and a smaller peak (25.6% in 1997-2000) occurs in autumn (March to April). Autumn peaks were absent in 1997 and 2000.

Figure 2: Cryptosporidiosis notifications by month, 1997-2001



An analysis of cryptosporidiosis cases notified between July 1999 and June 2001 found that reported risk factors varied between the two seasonal peaks. The proportion of cases reporting recreational contact with fresh water was significantly higher ($p < 0.01$) among cases notified during autumn (46.5%) than among cases notified during spring (16.4%). The proportion reporting a history of swimming in a public pool was also higher among autumn cases (40.9%) than among spring cases (12.5%). Conversely, a history of contact with infected animals was more frequently reported among spring cases (28.3%) than among those notified in autumn (5.6%).

Prevention of cryptosporidiosis associated with swimming pool use should focus on reducing the risk of *Cryptosporidium* contamination of pool water. People who have had diarrhoea in the previous two weeks should not use swimming pools, and pool users should be asked to shower properly, with hot water and soap, before going into the water. Preventing transmission from infected animals depends on adequate hand washing and other personal hygiene precautions, particularly following contact with calves and lambs.

Surveillance data

National surveillance data - June 2001

Disease ¹	Current year - 2001 ²			Previous year - 2000			Trends - June 2001
	Jun 2001 cases	Cumulative total year-to-date	Current rate ³	Jun 2000 cases	Cumulative total year-to-date	Previous rate ³	
AIDS	0	14	0.7	2	17	1.1	
Campylobacteriosis	454	3964	222.6	459	4338	240.6	***
Cholera	0	0	0	0	0	0	
Creutzfeldt-Jakob disease	0	1	0.1	1	1	0.1	
Cryptosporidiosis	60	520	32.1	26	133	17.2	***
Dengue fever	3	5	0.3	0	2	0.1	233
Gastroenteritis ⁴	53	365	20.7	76	350	19.7	
Giardiasis	143	816	44.1	160	907	45.8	
<i>H influenzae</i> type b disease	3	7	0.4	1	6	0.3	
Hepatitis A	4	30	2.8	13	37	2.2	
Hepatitis B (acute) ⁵	4	34	2.0	3	42	2.2	
Hepatitis C (acute) ⁵	3	26	1.9	8	39	2.3	
Hydatid disease	1	2	0.1	1	1	0.1	
Influenza ⁶	10	40	7.6	6	14	13.7	***
Lead absorption	10	70	3.8	8	59	3.6	
Legionellosis ⁶	4	40	2.3	1	26	2.0	
Leprosy	0	1	0.1	0	1	0.1	
Leptospirosis	10	54	2.9	3	49	2.4	
Listeriosis	0	7	0.4	1	13	0.7	
Malaria	5	34	3.1	6	32	1.5	*** 113
Measles	9	30	1.6	4	37	2.6	**
Meningococcal disease	54	242	14.6	53	193	14.6	
Mumps	6	26	1.3	5	28	1.5	
Paratyphoid	3	12	0.8	1	6	0.3	** 150
Pertussis	72	910	94.8	307	1619	70.4	***
Rheumatic fever ⁷	10	71	5.0	12	35	1.7	*** 195
Rubella	2	14	0.9	1	6	0.7	
Salmonellosis	150	1104	54.4	79	934	46.5	***
Shigellosis	13	93	4.0	12	63	3.3	
Tetanus	1	3	0.1	0	1	0.1	
Tuberculosis	33	196	10.4	27	173	11.0	
Typhoid	1	14	0.6	1	12	0.4	
VTEC/STEC infection	3	42	1.9	2	40	1.8	
Yersiniosis	22	208	10.6	30	223	12.7	**

Notes: 1 Other notifiable infectious diseases reported in June: Nil

2 These data are provisional

3 Rate is based on the cumulative total for the current year (12 months up to and including June 2001) or the previous year (12 months up to and including June 2000), expressed as cases per 100 000

4 Cases of gastroenteritis from a common source or foodborne intoxication eg, staphylococcal intoxication or toxic shellfish poisoning.

5 Only acute cases of this disease are currently notifiable

6 Surveillance data based on laboratory-reported cases only

7 Eighty-six rheumatic fever cases were first entered in June: 10 had June 2001 report dates, 41 had report dates from Jan-May 2001, and the remaining 35 were from the year 2000.

8 Percentage change is the difference between the number of cases in the current year (12 months up to and including June 2001) and the previous year (12 months up to and including June 2000). This difference is expressed as a percentage of the number of cases in the previous year.

Surveillance data

Surveillance data by health district - June 2001

Cases this month Current rate¹

Disease	Cases for June 2001, ² and current rate ^{1,2} by health district ^{3,4}																							
	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	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Campylobacteriosis	9	61	49	43	42	14	5	5	10	4	11	6	15	9	12	4	56	27	8	3	24	10	15	12
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
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
Cryptosporidiosis	0	5	4	1	3	1	0	0	1	3	2	0	6	3	1	0	20	3	1	0	4	5	0	2
Dengue fever	0	0	1	1	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0
Gastroenteritis	2	11	7	0	1	0	0	0	0	1	3	0	0	2	6	0	1	0	2	0	9	1	7	0
Giardiasis	2	17	18	13	13	5	4	1	2	0	3	0	6	3	5	0	11	5	3	1	22	1	4	4
H influenzae type b disease	1	0	1	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0
Hepatitis A	0	0	1	2	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0
Hepatitis B	0	2.0	3.8	6.7	0.7	0.9	0	15.3	0	0	0	0	0	0	0	0	1.6	0	1.7	0	8.8	2.5	1.2	1.8
Hepatitis C	0	0	0	0	0	2	0	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0
Hydatids	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0
Influenza ⁵	0	0	2	0	2	0	0	0	0	0	0	0	0	0	0	0	2	0	0	0	2	0	2	0
Lead absorption	0	0	1	1	1	0	0	0	0	0	0	0	0	0	2	0	0	0	0	0	2	1	1	1
Legionellosis ⁵	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	2	0	0	0	1	0	0	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
Leptospirosis	1	0	0	0	3	1	1	0	0	1	0	2	0	0	0	0	0	0	0	0	0	1	0	0
Listeriosis	0	0.8	0.6	1.5	0.3	0	0	0	0	0	0	0	0	0	0	0	0.4	0.8	0	0	0.5	1.3	0	0
Malaria	0	0	1	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	1	1	1	1	0	0
Measles	0	1	0	0	0	1	0	0	0	0	0	0	1	0	0	0	0	0	0	2	2	0	1	1
Meningococcal disease	2	0	11	12	4	1	3	4	4	1	0	0	3	1	0	0	2	0	2	2	1	0	1	0
Mumps	2	0	1	0	0	0	0	0	0	0	0	0	1	0	1	0	0	0	0	0	1	0	0	0
Paratyphoid	0	0	3	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Pertussis	4	2	2	2	11	1	0	0	0	0	0	2	0	0	0	19	6	1	3	7	1	1	10	
Rheumatic fever	1	0	2	5	0	0	0	0	0	0	0	1	0	0	0	0	1	0	0	0	0	0	0	0
Rubella	0	0.5	0.6	0.3	0	0	0	0	0	0	0	0	4.9	0	0	0	2.1	2.3	1.7	0	2.6	0	0.6	0.9
Salmonellosis	4	17	21	20	8	7	0	1	1	1	4	1	4	1	3	3	19	8	4	1	8	3	7	4
Shigellosis	0	1	3	3	3	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	1	1	0
Tetanus	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0
Tuberculosis	1	5	5	3	1	1	0	0	1	0	0	6	0	3	0	2	0	1	0	3	1	0	0	0
Typhoid	0	1.0	1.4	2.0	0.3	0	0	1.5	0	1.9	0	0	0	0	0	0.4	0	0	0	0.5	0	0	0	0
VTEC/STEC infection	0	0	0	0	3	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Yersiniosis	1	2	2	6	3	1	0	0	0	1	0	0	1	1	1	0	1	0	1	1	1	1	0	0

Notes: 1 Current rate is based on the cumulative total for the 12 months up to and including June 2001, expressed as cases per 100 000

2 These data are provisional

3 AIDS data are reported for the greater Auckland and Wellington areas, rather than by health district

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

5 Surveillance data based on laboratory-reported cases only

Detection of biological and chemical terrorism requires swift reporting by clinicians

The Centers for Disease Control and Prevention has published recommendations to reduce the vulnerability of the United States to biological and chemical terrorism. The focus areas identified were preparedness and prevention, detection and surveillance, diagnosis and characterisation of biological and chemical agents, emergency response and communication systems. Biological agents on the high priority list are those that can be easily disseminated, cause high mortality, have a major public health impact, cause public panic and social disruption, and require special action for public health preparedness. These agents include variola major (smallpox), *Bacillus anthracis* (anthrax), *Yersinia pestis* (plague) and *Clostridium botulinum* toxin (botulism). The chemical agents identified range from warfare agents such as sarin and tabun to industrial toxic chemicals such as benzene, mercury and chloroform. The terrorism preparedness plan includes developing public health communication infrastructure, a multilevel network of diagnostic labs and an integrated disease surveillance system as well as training, research and support. Since the initial detection and response to a bioterrorist attack would probably occur at the local level, one of the recommendations was

that state and local health authorities should have the expertise and resources to detect and respond to clusters of rare and unusual illnesses. These initiatives would also be able to detect naturally occurring outbreaks and industrial injuries. The success of this plan depends on developing partnerships not only between medical and public health professionals but also with emergency management, military and law enforcement professionals (Biological and chemical terrorism: strategic plan for preparedness and response, MMWR 2000; 49 (RR-4):1-14).

Editorial note: Many of the principles outlined in this plan are also applicable in New Zealand. Medical practitioners should immediately notify their local Medical Officer of Health if they see any illness that could potentially be caused by biological or chemical terrorism. Infections caused by the major biological agents, as well as chemical poisoning from the environment, are notifiable conditions. Clinicians should also report suspicious syndromes including any cluster of people with similar symptoms suggestive of a common exposure. Responses should be planned within the framework of existing emergency procedures.

One in ten hospital patients experience an adverse event

A study of two London hospitals demonstrated that 10.8% of patients experienced an adverse event, with an overall rate of 11.7% when multiple adverse events were included. An adverse event was defined as an unintended injury caused by medical management rather than by the disease process and which is sufficiently serious to lead to prolongation of hospitalisation or to temporary or permanent impairment or disability to the patient at the time of discharge. A third of these adverse events led to moderate disability, permanent disability or death, and half were considered preventable with ordinary standards of care. The methodology was modelled on the Harvard Medical Practice Study (HMPS) and involved a retrospective review of 1014 medical and nursing case records. The authors estimate that preventable adverse events in hospitals in England and Wales result in 3 million additional

bed days and a cost of £1 billion annually to NHS (Vincent C, Neale G, Woloshynowych M. Adverse events in British hospitals: preliminary retrospective record review. *BMJ* 2001; 322: 517-9).

Editorial note: Similar studies have been conducted using the HMPS method in New Zealand and Australia. In the New Zealand study, 10.7% of 1326 hospital records included evidence of an adverse event. A third of these adverse events were found to be preventable and a third resulted in moderate or permanent disability or death. The Quality in Australian Health Care Study had slightly higher rates where 16.6% of admissions were associated with an adverse event, of which half were considered preventable and half led to disability or death. The original HMPS study found that 3.7% of hospital admissions in the United States led to an adverse event, a third were preventable and a third led to disability or death.

Travel health

West Nile Virus outbreaks emphasise importance of mosquito precautions in temperate countries

West Nile (WN) virus, a mosquito-transmitted flavivirus, was first isolated in 1937 in Uganda and has recently caused large human outbreaks in the US (1999), Russia (1999) and Romania (1996). The outbreak caused by WN virus in New York in the summer of 1999 was the first time the virus was isolated in the Americas. The means of introduction into the United States is still unknown. There were sixty-two cases with seven fatalities in 1999 and twenty-one cases with two deaths in 2000 in New York. Investigations showed that most infections caused by WN virus were mild with only influenza-like symptoms. Severe infections were usually in the elderly and in some cases caused meningoencephalitis, profound muscle weakness, paralysis, and death. High numbers of avian and equine deaths were also reported in association with the outbreak. Despite the substantial geographic expansion of WN virus activity in both birds and mosquitoes in the US eastern seaboard in 2000, numbers and distribution of human infections were limited by the implementation of integrated vector management programmes. This article is a part of an entire journal issue on the biology, ecology and epidemiology of WN virus (Peterson L, Roehrig J. West Nile Virus: A reemerging global pathogen. *Emerg Infect Dis* 2001; 7: 611-4. Available online at <http://www.cdc.gov/ncidod/EID/vol7no4/petersen.htm>).

Editorial note: The different types of arboviral encephalitis are important emerging infectious diseases and are endemic worldwide, although not in New Zealand. Some of these arboviruses, such as WN virus and dengue, are expanding their range. The emergence of WN virus in continental US emphasises the importance of advising travellers to temperate and tropical regions of the need to take precautions against mosquito and tick bites, especially if travelling during summer. Advice to wear long sleeves and to use mosquito repellent is appropriate. Full recommendations on prevention of bites are given on the website www.cdc.gov/ncidod/dvbid/westnile/q&a.htm.

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