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Murine typhus in New Zealand

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Murine typhus, a rickettsial disease, is present in New Zealand. This conclusion is supported by clinical and serological evidence, and confirmed by the recent finding of *Rickettsia typhi* DNA in the white cells of a patient with a compatible illness. Since 1989, when the first case was recognised, fourteen cases have been reported from the greater Auckland region. The mean age \pm SD (range) was 48 ± 17 (23-83) years and there was no gender difference. All cases occurred between May and October. All but one patient lived in a rural setting, with 9 (64%) living north-west of Auckland in the Helensville/Kaukapakapa area. The rat is the usual host and the rat flea, *Xenopsylla cheopis*, is the usual vector for transmitting *R typhi* to humans. Humans are incidental hosts. The illness presents nonspecifically with fever, headache, myalgia and rash. Early diagnosis is based on clinical suspicion and confirmation is by serology. All confirmed cases should be immediately notified to the local medical officer of health. Prevention is directed mainly towards the control of the host, in particular rats, and the flea vector.

Rickettsial diseases are both some of the oldest and also some of the more recently recognised infectious diseases. Despite their worldwide occurrence, the first patient in New Zealand with a rickettsial disease, murine typhus caused by *Rickettsia typhi*, was only reported in 1991.¹ We recently described further eight cases from the Auckland region and although the initial case was diagnosed serendipitously, all of the others had a compatible illness arousing clinical suspicion and at least a four-fold rise in antibody titres to make the diagnosis.²

Rickettsiae are fastidious obligate intracellular bacteria. They are maintained in nature in a cycle which involves a mammalian host and an arthropod vector.³ They are small pleomorphic gram negative coccobacilli that survive only briefly outside the host. Humans are incidental hosts and except for epidemic typhus caused by *Rickettsia prowazekii*, have no role in maintaining the host-vector cycle.

Traditionally the species of the genus *Rickettsia* were divided into three groups: spotted fever, typhus (which includes *R typhi*) and scrub typhus groups. More recently *R tsutsugamushi*, the causative agent of scrub typhus, has been found to be distinct enough to warrant transfer to a new genus, *Orientia*, as *Orientia tsutsugamushi*.⁴ Rickettsiae infect and multiply in almost all organs of the arthropod vectors, which transmit them to mammalian hosts via salivary secretions or faeces. The vectors for the spotted fever and scrub typhus groups are ticks and mites and for the typhus group, lice and fleas.⁵ Humans acquire infection principally by direct contact with infected arthropods. Less commonly, infection with *R typhi* can occur following the inhalation of dust containing infective material or via contamination of the conjunctivae of the host with infected flea faeces.³

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The aim of this article is to summarise the known epidemiology of murine typhus in the greater Auckland region, to alert healthcare practitioners to this disease in order to improve recognition of cases and provide advice about prevention of infection. We have recently reported clinical details on the first nine cases² and will not describe clinical findings in any detail in this article.

Methods

Surveillance: Rickettsial diseases are notifiable. So far, all cases of locally acquired rickettsial disease have been reported from the greater Auckland region. The Virology and Immunology Department at LabPlus, Auckland Hospital, has carried out the serological testing and clinical data has been collected on all patients with positive serology.

Laboratory investigation: Anti-rickettsial antibodies are detected by a commercial indirect immunofluorescence (IFA) assay. MRL Diagnostics (code 1F0100M Cypress, CA) provides

Contents

Murine typhus in New Zealand	73
Surveillance and control notes	76
Surveillance data	78
Public health abstracts	80
Travel health	80

the commercial rickettsia substrate slides. Paired sera are screened for antibodies to *R typhi* and to *R conorii*, representative of the typhus and spotted fever groups respectively. Cross reactivity occurs within the rickettsial groups and with other bacteria.

To confirm the presence of a rickettsial disease in New Zealand, we established an in-house polymerase chain reaction (PCR) method for the detection of rickettsial DNA in white cells, tissue and arthropods. White cells collected early in the illness from case nine and before the administration of doxycycline, had the DNA extracted. This was then used as a template for primers complementary to portions of the coding sequence of the rickettsial citrate synthase gene. The citrate synthase gene of genus *Rickettsia* has been cloned and sequenced. It is found in all spotted-fever and typhus rickettsiae and amplification of a specific sequence, a 1,234-bp fragment, has been shown to be more suitable for rickettsial phylogenetic analysis than 16S ribosomal RNA sequencing.³ DNA amplification was performed using a GeneAmp® PCR System 9700 (PE Applied Biosystems, Foster City, CA, USA). *R prowazekii* DNA provided by the Australian Rickettsia Reference Laboratory was used as a positive control for the PCR analysis.

Investigation of host and vectors: The rat, *Rattus* spp., is the host and the rat flea, *Xenopsylla cheopis*, is the usual vector for transmitting the infection to humans. Seven possums, *Trichosurus vulpecula*, and two rats were trapped on the property of case nine and 16 cat fleas, *Ctenocephalides felis*, were collected from the family cat. DNA was extracted from whole blood from the possums, from liver and spleen tissue from the rats, and from the triturated (pulverised) fleas. The same PCR method as described above was used to amplify rickettsial DNA.

Results

Clinical cases: A patient was considered to have murine typhus if they had a compatible clinical illness and serological evidence of recent rickettsial infection as defined by at least a four-fold rise in antibody titre to *R typhi*. Since 1989 there have been 14 patients with an illness consistent with murine typhus. Demographic data are shown in Table 1. The mean age \pm SD (range) was 48 ± 17 (23-83) years and there was no gender difference. There was a seasonal variation in the timing of the illness; all cases occurred between May and October, winter and spring. All but one patient lived in a rural setting, with nine (64%) living north west of Auckland in the Helensville/Kaukapakapa area (Figure 1). All but one patient were admitted to hospital, and three (21%) were admitted to intensive care.

Laboratory confirmation of *R typhi* in New Zealand: The sequence amplified from the patient's white cells showed 100% homology with *R typhi*. The PCR positive control was sequenced and showed 100% homology with *R prowazekii*. We have thus demonstrated the presence of the genus *Rickettsia* and of a rickettsial disease in a patient in New Zealand.

Host and vectors: *R typhi* DNA was detected in the liver and spleen of one of the rats, demonstrating that the *Rickettsia* genus exists in this species and arguing that rats do transmit this infection in New Zealand as they do in other countries. No rickettsial DNA was found in the possums or the cat fleas nor in a further six rats and four rat fleas collected at a later date from the same property. We continue to try to culture the organism from potential host tissue and arthropod vectors.

Discussion

Until the 1990s, rickettsial diseases had not been considered to be present in New Zealand but we have now demonstrated with certainty that *R typhi*, the causative agent of murine typhus, is

Figure 1: Map showing locations of rickettsia cases, 1989 to 2001.



Table 1: Characteristics of clinical cases of Murine Typhus in New Zealand.

Case	Age	Gender	Area of Residence	Year of Infection	Admitted to Hospital
1	47	F	Makarau	1989	Yes
2	34	M	South Head, Kaipara	1995	Yes
3	66	M	Makarau	1997	Yes
4	61	F	Kaukapakapa	1998	No
5	28	M	Clevedon	1998	Yes
6	52	F	Karaka	1998	Yes
7	23	F	Mangere	2000	Yes
8	52	M	Whitianga	2000	Yes
9	34	F	Kaukapakapa	2000	Yes
10	28	F	Bethells Beach	2001	Yes
11	65	M	Puhoi	2001	Yes
12	83	M	Dairy Flat	2001	Yes
13	52	F	Coromandel	2001	Yes
14	45	M	South Head, Kaipara	2001	Yes

present in the greater Auckland region. It is therefore possible and probably likely that it is present in other regions of New Zealand as well. A number of rickettsial diseases are currently recognised in Australia. These include scrub typhus,⁶ Queensland tick typhus,⁷ Flinders Island spotted fever,⁸ and murine typhus.⁹ With the increasing local awareness of this infection, more cases have been reported in the last two years but, as in other parts of the world, it is highly likely that it will remain an under-recognised and under-reported infection.¹⁰

We found the place of residence an important clue to the diagnosis. A cluster of cases has occurred in Helensville/Kaukapakapa and more recently two cases have been reported from the Coromandel. These areas are predominantly rural where there is an increased risk of exposure to rats and rat fleas. The detection of rickettsial DNA in a rat from the property of case nine supports the idea of conventional rat host and rat flea vector. On the other hand, the transmission of murine typhus in some parts of the United States has been attributed to an opossum-cat flea cycle.^{11,12} The American opossum, *Didelphis marsupialis*, differs from the New Zealand possum (the brushtail possum, an introduced marsupial from Australia) and to date, somewhat surprisingly, only one flea has been found on a possum (personal communication, P. McKenna). Thus it seems unlikely that possums in New Zealand play an important role in the host-vector cycle even though the first two patients we saw told us of their quite extraordinary close environmental association with possums.^{2,13} Presumably where possums are close to homes, so too are rats. The role of either domestic or feral cats and cat fleas in the transmission of murine typhus in New Zealand remains unknown. The majority of cases occurred during the winter months, at a time when rats are more likely to seek shelter in dwellings shared by humans and domestic animals.

Murine typhus is an illness that presents nonspecifically with fever, headache, myalgia and a rash (in 50% of patients) and laboratory tests are unhelpful early in the course of the illness.¹⁴ Presentation is similar to other zoonotic diseases such as leptospirosis and doxycycline, a somewhat effective treatment for leptospirosis, is also the treatment of choice for murine typhus. Thus, murine typhus should be considered as part of the differential diagnosis among patients from rural areas presenting to medical practitioners with undiagnosed febrile illnesses. When you think of leptospirosis, also think of murine typhus.

The average age of the local cases was 48 years, the same average as was seen in a series of 80 patients from South Texas.¹⁴ While there have been no children diagnosed with murine typhus locally, children do get this disease.¹⁴ The clinical course in children is said to be mild.¹⁵ All locally acquired cases occurred during the winter and early spring months in contrast to the series from North America where cases occur predominantly in the spring and summer months.^{14,16}

Early diagnosis is based on clinical suspicion and confirmation is by serology. The traditional Weil-Felix reaction is now obsolete because it is neither sensitive nor specific. The "gold standard" is

the indirect immunofluorescence test. Fifty percent of patients have antibodies within one week and nearly all by 15 days after the onset of the illness.¹⁴ While nucleic acid amplification detection methods such as PCR may provide a faster diagnosis in acute infection, they are not routinely available. Culture is technically very difficult but the isolation of our local strain of *R typhi* will be important in better understanding this organism in the New Zealand environment and its likely origin.

The preferred agent for treatment of *R typhi* infection is a tetracycline, such as doxycycline.¹⁷ The treatment of children and pregnant women is difficult. A single dose of doxycycline is a safe alternative in children.¹⁸ Pregnant women can be treated with a quinolone, doxycycline (late trimester) or chloramphenicol (early trimester).¹⁵ We have treated a pregnant woman with ciprofloxacin without any adverse effect. Recovery from natural infection confers solid, life long immunity to reinfection.

Clinical and serological findings supported the presence of *R typhi*, the causative agent of murine typhus, in New Zealand and more recently we have confirmed this with the detection of rickettsial DNA in the white cells of a patient and a rat. All cases of murine typhus should be reported to local public health authorities. Prevention is directed mainly towards the control of potential flea hosts, in particular rats and the flea vectors. Although generally a mild illness, it can be severe or even fatal. As an indication of its severity, three of the local cases have required intensive care.

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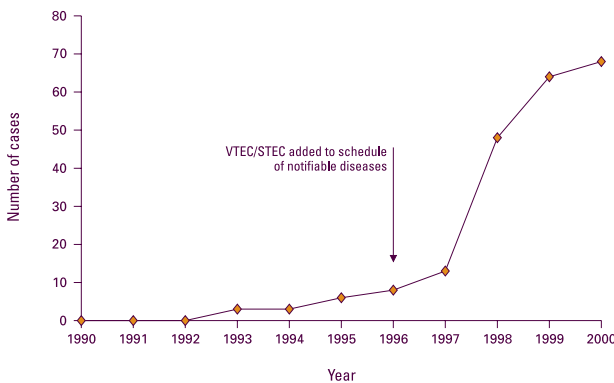
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VTEC/STEC incidence continues steady increase

There were 68 cases of verotoxigenic or shiga toxin producing *Escherichia coli* (VTEC/STEC) infection notified in 2000, a rate of 1.9 per 100 000. This total is the highest number reported in New Zealand in a single year and is a slight increase on the 64 cases (1.8 per 100 000) notified in 1999. Of the 65 cases for whom hospitalisation status was recorded, eleven (16.9%) were hospitalised. Three cases of haemolytic uraemic syndrome associated with VTEC/STEC infection were notified in 2000 to the New Zealand Paediatric Surveillance Unit and are included in this total. In 2000, the Enteric Reference Laboratory at ESR also received 68 VTEC/STEC isolates, all of which could be matched to a notified case on EpiSurv. Sixty-four cases (94%) were identified as serotype O157 and four cases (6%) as other serotypes.

The following graph shows VTEC/STEC infection notifications by year since it was first detected in New Zealand in 1993.

Figure 1: VTEC/STEC infection cases by year, 1990-2000



The rate of VTEC/STEC infection varied throughout the country. Rates higher than the national average were recorded in Taupo (16.3 per 100 000), Waikato (6.6), Taranaki (6.6), South Canterbury (5.0), Otago (4.6), Eastern Bay of Plenty (4.0), Wanganui (3.3), Rotorua (3.1), and Canterbury (2.3) Health Districts.

The highest age-specific rates of notified VTEC/STEC infection in 2000 occurred among young children, with a rate of 11.0 per 100 000 among children aged under one year old, and 14.2 among children aged between one and four years. Overall, 69.1% of VTEC/STEC cases occurred among children aged less than ten years old. The age distribution is similar to that reported in 1999, when 67.1% of cases were aged under ten years. Gender was recorded for 65 (95.6%) of the 68 cases notified in 2000, and of these 32 (49.2%) were male and 33 (50.8%) were female.

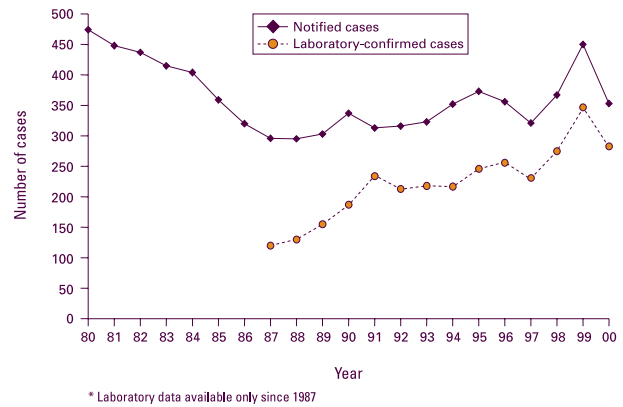
The continuing widespread occurrence of VTEC/STEC infection in New Zealand underlines the need for accurate diagnosis and effective case management, health promotion and health protection strategies. Clinicians should consider the diagnosis of VTEC/STEC in any patient with diarrhoea, particularly bloody diarrhoea, and request specific testing for these organisms. Identified cases should be advised carefully on the need to avoid secondary transmission, and should be rapidly notified to the medical officer of health to help identification and investigation of outbreaks. People should have access to clean drinking water and should be encouraged to thoroughly cook minced meat, consume only pasteurised milk and milk products, reduce cross-contamination from meat and other foods, wash hands thoroughly with soap and water after handling raw meat or touching animals, and to avoid swimming in sewage-contaminated water.

Decline in tuberculosis cases in 2000 mainly driven by drop in local transmission

A total of 353 cases of tuberculosis were notified in 2000 (9.8 cases per 100 000), a 22.2% decrease on the 20-year peak of 456 cases notified in 1999 (12.6 per 100 000). Despite this drop, the trend has been a gradual increase in reported occurrence of tuberculosis since the lowest reported

incidence in New Zealand in 1988 (Figure 2). The number of laboratory-confirmed cases also decreased, from 347 (76.1%) in 1999 to 283 (80.2%) in 2000. Thirty (8.5%) of the cases in 2000 were reactivations.

Figure 2: Tuberculosis cases by year, 1980-2000



Of the 314 cases for whom hospitalisation status was recorded, 199 (63.4%) were hospitalised. Eight deaths were reported in 2000 (a case-fatality rate of 2.3%) compared with fourteen deaths in 1999 (case-fatality rate of 3.1%). One outbreak of tuberculosis was reported in 2000.

Rates of tuberculosis above the 2000 national average of 9.8 per 100 000 were recorded in Central Auckland (20.5 per 100 000), South Auckland (20.5), Wellington (19.3), Hutt (12.8), Ruapehu (11.9), and Eastern Bay of Plenty (9.9) health districts. The proportion of female and male cases was similar: 46.2 and 53.8% respectively, with sex not reported for two cases. Table 1 shows the age-specific rates by ethnic group. Rates of tuberculosis declined among all ethnic groups between 1999 and 2000.

Table 1: Tuberculosis notifications by age group and ethnicity, 2000¹

Age group (years)	European		Maori		Pacific people		Other		Unknown	Total	
	Cases	Rate ¹	Cases	Rate ¹	Cases	Rate ¹	Cases	Rate ¹		Cases	Rate ¹
<1	0	0.0	0	0.0	0	0.0	0	0.0	0	0	0.0
1-4	0	0.0	6	10.6	2	10.9	4	35.2	0	12	5.3
5-9	1	0.6	4	5.9	0	0.0	4	28.0	0	9	3.1
10-14	1	0.6	2	3.5	1	5.8	3	18.7	0	7	2.6
15-19	0	0.0	3	5.8	5	29.7	8	40.0	1	17	6.5
20-29	3	0.8	15	16.6	13	41.1	48	158.2	1	80	14.7
30-39	2	0.5	12	15.3	5	18.6	37	103.4	0	56	9.7
40-49	7	1.8	7	13.9	5	27.9	14	54.6	1	34	6.9
50-59	7	2.5	7	23.3	13	128.3	19	185.8	2	48	13.9
60-69	7	3.1	6	34.4	6	105.8	11	203.5	4	34	12.7
70+	15	5.7	10	116.1	13	408.4	13	406.1	5	56	19.3
Unknown	0	-	0	-	0	-	0	-	0	0	-
Total	43	1.7	72	13.8	63	36.4	161	91.8	14	353	9.8

¹ Crude rate per 100 000, based on 1996 census

The decline in tuberculosis incidence in 2000 has been mainly driven by a drop in transmission within New Zealand with fewer of the large outbreaks that increased numbers during 1999. Both the number and proportion of New Zealand-born cases declined between 1999 and 2000. Of the 311 cases in 2000 for whom country of birth had been reported, 37.3% (116) were born in New Zealand, compared with 42.7% (170/398) in 1999 and similar to 36.8% (116/317) in 1998. The trend appears to have continued among cases in the first six months of 2001, with 33.1% (57/172) of those with reported country of birth having been born in New Zealand. Of cases in 2000, a total of 66.5% (103/155) of those born overseas and for whom arrival date was recorded developed tuberculosis within the first five years of arrival in New Zealand, 25.8% (40/155) developing tuberculosis within the first year of arrival.

The causative organism was isolated and identified from 250 cases, and 242 (96.8%) were *Mycobacterium tuberculosis* and eight (3.2%) were *M bovis* isolates. The antimicrobial susceptibility of 249 isolates (all except one *M bovis*) was tested and is shown in Table 2. One

M tuberculosis (0.4%) was multidrug resistant (resistant to at least isoniazid and rifampicin). The isolate was from a patient born in China. Since data collation began in 1995, all multidrug resistant isolates have been from persons born outside New Zealand. The overall prevalence of resistance to isoniazid, rifampicin, ethambutol, pyrazinamide, and streptomycin was 10.0%, 0.4%, 1.2%, 4.0%, and 8.4%, respectively. There was an apparent increase in the prevalence of resistance in 2000, with 16.9% of the isolates being resistant to at least one antimicrobial compared with 9.0% in 1999.

Tuberculosis control in New Zealand relies on prompt diagnosis of cases, thorough contact tracing to detect further cases and infected contacts, and effective treatment of cases and management of infected contacts. Medical practitioners should ensure that tuberculosis cases are notified to medical officers of health.

Table 2: Resistance patterns of *M tuberculosis* and *M bovis*, 2000

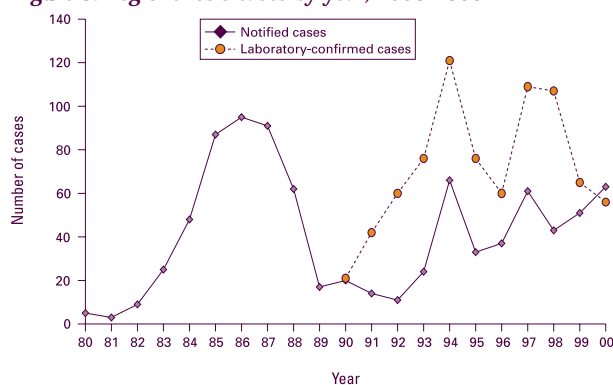
	Agent(s) ¹	Number (%) with each pattern
Resistant to 5 agents	HRZSE	1 (0.4) ²
Resistant to 4 agents	HZSE	1 (0.4)
Resistant to 3 agents	HSE	1 (0.4)
Resistant to 2 agents	HS	7 (2.8)
	HZ	2 (0.8) ³
Resistant to 1 agent	H	13 (5.2)
	S	11 (4.4)
	Z	6 (2.4) ⁴
Fully sensitive	-	207 (83.1)

- ¹ H = isoniazid, R = rifampicin, Z = pyrazinamide, S = streptomycin, E = ethambutol
² multidrug resistant isolate
³ both were *M bovis* isolates which are intrinsically resistant to pyrazinamide
⁴ includes five *M bovis* isolates which are intrinsically resistant to pyrazinamide

Legionellosis in 2000 still dominated by *L longbeachae*

A total of 63 cases of legionellosis were notified in 2000. The 2000 rate of 1.7 per 100 000 is slightly higher than the 1999 rate of 1.4. Of the 55 cases for whom hospitalisation status was recorded, 47 (85.5%) were hospitalised. Five deaths were reported in 2000, giving a case-fatality rate of 7.9%. There were 56 cases of legionellosis reported by laboratories to ESR during 2000. The degree of overlap between these cases and the notified cases during 2000 is unknown. The following graph shows laboratory-reported cases of legionellosis by year since 1990 (recorded by the date that the specimen was received by ESR) and notified cases since 1980 (recorded by the date of notification). 2000 was the first year since laboratory reporting commenced that the number of notified cases has exceeded that of laboratory-reported cases.

Figure 3: Legionellosis cases by year, 1980-2000



The rate of legionellosis varied throughout the country. Rates higher than the national average were recorded in Wairarapa (13.0 per 100 000), Ruapehu (6.0), Waikato (4.3), Northland (3.6), Tauranga (3.5), West Coast (3.1), Hutt (2.3), Canterbury (2.1), Wellington (2.1), and Southland (1.8) Health Districts.

The highest age-specific rate of notified legionellosis in 2000 occurred among older adults, with a rate of 5.6 per 100 000 occurring among adults aged between 60 and 69 years. Overall, 76.2% of legionellosis cases occurred among adults aged fifty years old or greater. The age distribution is similar to that reported in 1999, when 75.0% were aged fifty years or older. Gender was recorded for 62 (98.4%) of the 63 cases. Thirty-six cases (58.1%) were male and 26 (41.9%) were female.

Table 3: Laboratory-reported legionellosis, by species, 1995-2000

Legionella species	Year					
	1995	1996	1997	1998	1999	2000
<i>L pneumophila</i>	12	12	45	67	13	15
<i>L anisa</i>	1	0	2	1	0	1
<i>L bozemanii</i>	1	0	1	2	2	4
<i>L dumoffii</i>	1	0	1	2	2	0
<i>L longbeachae</i>	6	0	10	13	34	28
<i>L micdadei</i>	6	2	13	3	6	2
<i>L jordanis</i>	3	3	3	2	2	0
<i>L feelei</i>	1	3	1	0	0	0
<i>L gormanii</i>	0	4	0	0	2	2
Unidentifiable <i>Legionella</i> sp.	28	21	33	15	4	4
Total	59	45	109	105	65	56

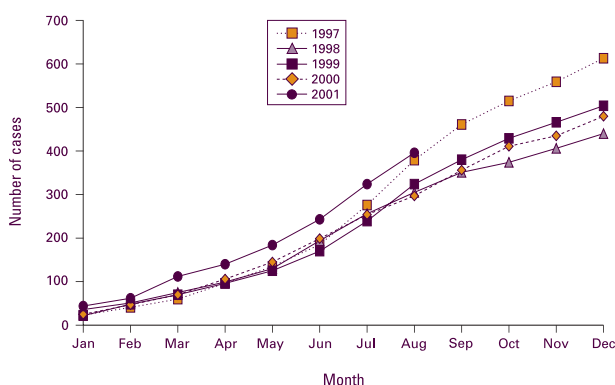
Table 3 compares the species/serogroup distribution of laboratory-reported *Legionella* over the last six years. An increasing proportion of cases over time have been attributed to *L longbeachae* infection, from 9.2% in 1997 to 50.0% in 2000.

Infection with *L longbeachae* can be associated with inhalation of bacteria released from gardening products such as potting mix, mulch and compost. Older adults, smokers and those with chronic illnesses should be advised to take precautions when using gardening products. These measures include working in well-ventilated environments, opening bags of soil products slowly and away from the face, dampening soil products before use and avoiding excessive dust, washing hands thoroughly after working in the garden, and seeking medical attention for any flu-like illness that appears to worsen. Gardening is however a generally healthy activity and should not be discouraged.

Meningococcal disease update

A total of 72 cases of meningococcal disease were notified during August, bringing the year to 31 August total to 396. This is the highest number of cases notified for the January to August period for any year since the epidemic began. The epidemic remains dominated by serogroup B organisms and the P1.7b,4 subtype. A total of 19 deaths due to meningococcal disease have been reported for the year to 31 August, compared with eleven deaths for the same period during 2000. Figure 4 shows the cumulative total of meningococcal disease cases by month between January 1997 and August 2001. These data are combined from case reports and laboratory surveillance, and use the earliest available date of illness (ie, either date of illness onset, hospitalisation or notification).

Figure 4: Meningococcal disease cases, cumulative total by month, January 1997 – August 2001



Surveillance data

National surveillance data - August 2001

Disease ¹	Current year - 2001 ²			Previous year - 2000			Trends - August 2001	
	Aug 2001 cases	Cumulative total year-to-date	Current rate ³	Aug 2000 cases	Cumulative total year-to-date	Previous rate ³	Percentage change ⁷	
AIDS	3	20	0.8	0	19	0.9	-100% -75% -50% -25% 0% 25% 50% 75% 100%	
Campylobacteriosis	771	5266	232.5	524	5286	229.1	Significance of difference: * p<0.05 ** p<0.01 *** p<0.001	
Cholera	2	2	0.1	0	0	0		
Creutzfeldt-Jakob disease	0	1	0.1	0	1	0.1		
Cryptosporidiosis	80	631	33.5	43	194	16.5	*** 103	
Dengue fever	34	60	1.8	0	3	0.1	*** 1500	
Gastroenteritis ⁴	89	592	24.4	52	440	18.2	***	
Giardiasis	137	1076	43.6	152	1186	46.7	*	
<i>H influenzae</i> type b disease	4	12	0.4	1	9	0.3		
Hepatitis A	10	43	2.2	13	70	2.8		
Hepatitis B (acute) ⁵	6	43	1.8	11	57	2.3		
Hepatitis C (acute) ⁵	6	40	1.7	12	61	2.6	**	
Hydatid disease	0	3	0.1	0	1	0.1		
Influenza ⁶	172	596	21.6	36	64	3.9	*** 450	
Lead absorption	10	96	4.0	9	75	3.3		
Legionellosis ⁶	2	46	2.2	4	33	1.9		
Leprosy	0	2	0.1	0	3	0.2		
Leptospirosis	13	76	2.8	6	73	2.7		
Listeriosis	2	10	0.4	4	19	0.7		
Malaria	3	40	2.6	2	56	2.0		
Measles	10	43	1.5	7	53	2.3	*	
Meningococcal disease	68	395	16.1	42	292	13.3	**	
Mumps	9	43	1.6	6	36	1.5		
Paratyphoid	5	18	1.0	0	6	0.3	*** 260	
Pertussis	109	1079	81.0	397	2287	82.3		
Rheumatic fever ⁷	13	102	4.2	4	94	3.0	**	
Rubella	4	24	0.9	9	18	0.9		
Salmonellosis	194	1446	58.6	128	1125	46.8	***	
Shigellosis	9	120	4.4	7	74	3.0	**	
Tetanus	0	3	0.1	0	1	0.1		
Tuberculosis	25	252	10.1	29	239	11.1		
Typhoid	0	17	0.7	0	14	0.4		
VTEC/STEC infection	15	58	2.1	10	51	2.0		
Yersiniosis	34	268	10.5	28	286	12.7	**	

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

2 These data are provisional

3 Rate is based on the cumulative total for the current year (12 months up to and including August 2001) or the previous year (12 months up to and including August 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 Percentage change is the difference between the number of cases in the current year (12 months up to and including August 2001) and the previous year (12 months up to and including August 2000). This difference is expressed as a percentage of the number of cases in the previous year.

Surveillance data

Surveillance data by health district - August 2001

Cases this month Current rate¹

Disease	Cases for August 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	1	0	1	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0
	0	1.6	0	1.8	0	0	0	0	0	0	0	0	0	0	0	0.5	0.9	0	0	1.6	0	0	0	0
Campylobacteriosis	29	127	96	72	91	214	7	3	13	11	31	0	26	13	15	7	66	31	13	6	51	15	20	4
	160.5	254.7	235.7	191.7	273.0	215.4	117.3	148.6	164.3	241.0	216.2	113.4	239.7	174.2	105.1	150.8	335.5	240.5	116.6	209.7	296.7	313.1	231.7	260.5
Cholera	0	1	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	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	0.7	0	0	0	0	0	0	0	0.5	0	0	0
Cryptosporidiosis	1	4	0	6	36	4	0	4	0	4	0	0	3	3	6	0	1	0	0	0	6	4	2	0
	17.5	17.8	28.3	22.8	55.9	24.8	6.0	37.2	75.9	48.9	16.8	0	113.6	42.3	27.9	28.6	39.1	34.7	2.6	30.8	22.5	78.0	33.6	36.8
Dengue fever	2	9	7	4	2	2	0	0	1	0	0	0	0	0	0	0	3	1	1	0	0	1	1	0
	1.5	3.3	5.2	2.9	1.0	2.7	0	0	1.5	0	0	0	0	0	1.3	0	2.1	1.5	0.9	0	0.3	1.3	0.6	0.9
Gastroenteritis	2	11	15	4	1	0	0	21	1	0	1	0	0	1	0	0	4	0	4	0	20	0	3	1
	21.2	24.6	28.6	14.0	3.6	8.0	0	52.5	23.2	29.3	32.8	0	1.4	11.4	35.9	31.2	4.5	10.6	30.0	27.8	69.1	60.4	24.3	4.5
Giardiasis	1	22	17	10	10	4	0	3	2	4	2	1	14	2	4	1	10	4	14	0	4	2	6	0
	29.2	48.2	61.3	42.1	51.9	65.6	37.8	32.8	54.2	45.6	22.5	23.9	69.7	26.1	31.9	20.8	62.2	29.4	31.7	77.1	34.9	32.7	24.9	18.0
H influenzae type b disease	0	0	2	0	0	0	0	0	0	0	0	0	0	0	0	0	1	1	0	0	0	0	0	0
	0.7	0.5	0.9	0.3	0	0	2.0	2.2	0	0	0.9	0	0	0	0	0	0.4	2.3	0	0	0.5	0	0	0
Hepatitis A	0	0	1	5	1	0	0	1	0	0	0	0	0	0	0	0	1	1	0	0	0	0	0	0
	0	1.8	4.3	7.3	0.7	0.9	0	19.7	0	0	0	0	0	0	0	0	2.1	0.8	0.9	0	2.6	1.3	1.2	0.9
Hepatitis B	0	0	0	0	1	2	0	0	0	1	0	0	0	0	0	0	1	0	1	0	0	0	0	0
	2.2	1.8	1.7	1.5	3.3	2.7	0	1.5	6.5	0	0	3.5	0	1.3	7.8	1.6	0.8	2.6	6.2	1.6	0	0.6	0.9	
Hepatitis C	0	0	0	1	0	3	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0
	0.7	0.3	0.6	0.6	0.3	16.8	4.0	4.4	6.2	3.3	0	0	2.1	0	0	0	2.5	0.8	2.6	3.1	1.8	0	0.6	2.7
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	0.3	0	0	0	0.9	0	2.2	0	0	0	0	0	0	0	0	0.4	0	0	0	0.3	0	0	0
Influenza ⁵	0	0	44	0	11	0	0	0	0	0	0	0	0	0	0	0	20	0	0	0	89	0	8	0
	0	0	71.7	0.3	57.8	0	0	0	0	0	0	0	0	0	0	0	28.0	0	0	0	70.6	0	9.3	0
Lead absorption	1	0	1	0	2	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0	2	1	2	0
	1.5	1.0	2.3	1.2	5.9	1.8	0	2.2	3.1	0	4.7	23.9	3.5	8.1	7.3	2.6	3.3	0	6.0	3.1	7.2	15.1	8.7	2.7
Legionellosis ⁵	0	1	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	3.6	1.3	0.9	0.9	7.3	0.9	0	0	0	0	0.9	0	0	3.3	0.7	5.2	2.9	5.3	0	0	4.1	1.3	2.3	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	0.3	0.6	0	0	0	0	0	0	0	0	0	0	0	0	0.4	0	0	0	0	0	0	0
Leptospirosis	5	1	0	1	1	1	0	1	1	0	0	0	1	0	0	0	0	0	0	0	0	1	0	0
	9.5	0.8	0.6	0.9	7.6	4.4	2.0	15.3	3.1	3.3	2.8	0	7.7	1.6	4.0	2.6	0.4	0	1.7	3.1	1.6	8.8	0.6	1.8
Listeriosis	1	0	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	0.7	0.8	0.6	0.6	0.3	0	0	2.2	0	0	0.9	0	0	0	0	0	0	0	0	0	0.3	1.3	0	0
Malaria	1	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	1	0	0	0	0	0	0
	0.7	0.5	1.7	1.8	2.6	0	0	2.2	0	3.3	0.9	11.9	0	0	27.3	0	2.9	2.3	3.4	3.1	1.6	2.5	0.6	1.8
Measles	0	1	1	0	0	2	0	0	0	1	0	2	0	0	0	0	0	1	0	1	0	1	0	0
	0.7	1.3	2.3	0.3	0	4.4	0	2.2	0	0.9	0	4.2	1.6	1.3	0	0.4	0.8	1.7	9.3	2.8	0	1.7	1.8	
Meningococcal disease	5	5	5	10	9	2	1	0	1	1	2	1	2	3	3	0	3	5	0	0	6	0	3	1
	26.3	9.9	24.3	32.8	19.5	13.3	35.8	21.9	23.2	35.8	8.4	17.9	20.9	13.0	12.0	13.0	10.7	9.8	9.4	9.3	5.2	1.3	15.6	7.2
Mumps	1	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0	3	1	0	0	3	0	0	0
	5.1	0.3	1.7	1.5	0	0	4.0	0	3.1	0	0	6.0	3.5	0	1.3	0	3.7	2.3	0.9	0	2.3	2.5	1.2	0
Paratyphoid	0	0	1	2	0	1	0	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0
	0	0.5	2.9	1.2	1.0	0.9	0	0	0	0	0.9	0	3.5	1.6	2.0	0	0	1.5	0	0	1.0	0	0	0
Pertussis	1	6	4	4	7	2	0	0	1	1	0	2	0	0	0	0	2	10	48	3	10	2	1	5
	62.7	30.7	38.5	34.8	149.1	63.8	83.5	35.0	75.9	52.1	7.5	83.6	45.3	16.3	7.3	145.6	65.5	120.7	299.3	293.0	151.3	71.7	104.2	70.1
Rheumatic fever	1	1	1	5	4	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0
	6.6	2.5	14.5	12.6	4.3	0	19.9	4.4	3.1	0	0	0	3.5	0	0	2.6	2.1	1.5	0	0	0	0	0	0
Rubella	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0	2	0	0	0	1	0	0	0
	0	0.8	0.3	0.3	0	0	0	0	0	0	0	0	5.6	0	0	0	2.5	1.5	1.7	0	1.6	0	0.6	1.8
Salmonellosis	8	21	14	13	15	8	2	0	3	4	5	0	4	5	5	2	19	7	7	6	17	1	19	9
	46.7	47.7	49.2	47.1	53.5	50.5	39.8	32.8	43.4	55.4	59.0	65.7	46.0	57.0	55.9	59.8	68.8	54.3	72.0	61.7	62.1	101.8	95.6	113.2
Shigellosis	0	1	1	4	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	2	0	0	0
	0.7	5.6	12.7	12.3	1.7	0	0	2.2	3.1	0	0	0	1.4	1.6	0	0	2.9	1.5	1.7	0	5.7	5.0	2.3	0
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
	0.7	0	0	0	0	0	0	0	0	0	0.9	0	0	0	0	0	0	0.9	0	0	0	0	0	0
Tuberculosis	0	5	3	3	1	1	1	0	0	2	0	0	2	0	1	1	4	0	1	0	0	0	0	0
	6.6	8.4	21.4	19.9	5.9	5.3	11.9	8.7	7.7	13.0	0	6.0	14.6	1.6	10.0	10.4	21.4	9.8	2.6	3.1	3.6	2.5	4.6	4.5
Typhoid	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	1.3	1.4	2.0	0	0	0	1.5	0	1.9	0	0	0	0	0	0	0	0	0	0	0.8	0	0.6	0
VTEC/STEC infection	0	0	0	0	6	1	0	0	0	0	1	0	0	1	0	0	0	0	0	0	4	0	2	0
	0	1.0	0.3	0.6	7.3	5.3	2.0	0	4.6	0	3.7	6.0	2.1	4.9	0	0	0.4	0	0.9	0	2.1	5.0	4.6	2.7
Yersiniosis	0	6	5	5	5	2	0	1	1	1	0	0	0	2	1	0	1	1	0	0	2	1	0	0
	2.9	11.2																						

Contact with animal faeces represents a significant risk factor for *Escherichia Coli* O157 infection

A prospective matched case control study in Scotland was conducted to determine environmental risk factors for *Escherichia coli* O157 infection. The study ran from 1996 to 1999 and involved 183 cases and 543 matched controls. A standard structured questionnaire was administered covering demographic and clinical details, food, water, animal and environmental exposures. Twenty-one percent of the cases had contact with animal faeces (Odds ratio [OR] 3.7, 95% CI 1.8-7.3) and a further 28.4% had likely contact with animal faeces (OR 4.8, 95% CI 2.4-9.5). Thus, these two factors emerged as strong risk factors for infection. Drinking bottled water was found to have a protective effect (OR 0.3, 95% CI 0.2-0.5) as 20.6% of cases were exposed in contrast to 33.6% of controls. There was no association between *E coli* O157 infection and food exposure. This study highlights that transmission of *E coli* O157 does not occur only through contaminated food. The authors advise that members of the public should be aware

of the potential danger posed by exposure to animal faeces (Locking ME, O'Brien SJO, Reilly WJ, et al. Risk factors for sporadic cases of *Escherichia coli* O157 infection: the importance of contact with animal excreta. *Epidemiol Infect* 2001; 127: 215-20).

Editorial Note: In New Zealand most cases of verotoxigenic *Escherichia coli* (VTEC) infection are caused by *E coli* O157. The incidence of VTEC infection has risen steadily since the first confirmed cases in 1993 (see Surveillance and Control note in this issue on page 76). In 2001, from January to August, *E coli* O157 caused all 59 cases of VTEC infection. Of these cases, 26% reported contact with animal faeces prior to infection. No case-control study has been carried out in New Zealand to investigate sources for this disease. Advice on avoiding exposure to animal faeces is also important here. Medical practitioners should consider VTEC among individuals who present with acute diarrhoea and who have a history of contact with animal faeces.

Exposure to environmental tobacco smoke increases respiratory symptoms in adults with asthma

This study investigated environmental tobacco smoke (ETS) exposure in adults with asthma to elucidate the health effects. Fifty non smoking subjects from an ongoing asthma cohort study in North California wore a passive nicotine badge monitor for seven days and completed an ETS exposure survey at the end of this period. The survey assessed exposure during the previous seven days in six microenvironments (own home, another person's home, work, vehicle, bars and nightclubs and other locations) and recorded health outcomes such as sensory irritation, respiratory symptoms and extra bronchodilator use. The sites with the greatest maximal self reported ETS exposure were bars and nightclubs (55hr) and the home (50hr). Higher levels of self reported ETS exposure and monitored nicotine levels ($>0.05 \text{ mg/m}^3$) were associated with increased risk of respiratory symptoms (Odds ratio [OR] 6.8, 95% CI 1.4-32.3) and extra bronchodilator use (OR 8.1, 1.3-50) (Eisner MD, Katz PP, Yelin EH, et al. Measurement of environmental tobacco smoke exposure among adults with asthma. *Environ Health Perspect* 2001; 109: 809-14).

Editorial note: New Zealand research also finds higher levels of ETS and respiratory symptoms in bars and restaurants. One study (Bates M, Fawcett J, Dickson S, et al. Assessment of exposures of New Zealand hospitality workers to environmental tobacco smoke. ESR Client Report for the Ministry of Health. Porirua: Institute of Environmental Science and Research; 2001) found that there was a higher prevalence of respiratory and irritation symptoms among non-smoking hospitality workers than workers in smokefree workplaces. Hair nicotine levels have also been found to vary strongly according to the smokefree policy at the workplace among non smoking bar and restaurant staff (Al-Delaimy W, Fraser T, Woodward A. Nicotine in hair of bar and restaurant workers. *N Z Med J* 2001; 114 : 80-2). These findings support measures to reduce ETS in all environments, particularly for people with asthma and other groups who are more vulnerable to such exposures.

Travel health

Meningococcal disease in African meningitis belt, 2001

Meningococcal disease is one of the world's leading causes of epidemic emergencies. There are about 1.2 million cases a year, including 135 000 deaths. The most frequent and largest epidemics occur in the 18 sub-Saharan countries that make up the African meningitis belt. Most of these outbreaks are caused by *N meningitidis* serogroup A with a smaller contribution from serogroup C and W135. In 2001, six countries in the meningitis belt had large epidemics: Benin, Burkina Faso, Central African Republic, Chad, Ethiopia, and Niger. In these countries the epidemics tended to rise during the first 3 months of the year, reaching peaks from February to April, and then decline. Emergency mass vaccination with meningococcal A&C vaccine was used in all of these countries (Epidemics of meningococcal disease, African meningitis belt, 2001. *Wkly Epidemiol Rec* 2001; 37: 282-8.)

Editorial note: In sub-Saharan Africa, epidemics of serogroup A or C meningococcal disease occur frequently during the dry season (December through June). Meningococcal disease vaccination should be considered for people who plan to live or work in endemic countries and also for those visiting countries during epidemic periods. A tetravalent vaccine is available that provides protection against serogroup A, C, Y and W135 disease. A single injection induced antibodies after 10-14 days in 90% of recipients over the age of two years. Protective immunity lasts for at least three years.

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