

New Zealand Public Health Report

ISSN 1173-0250

Volume 9 Number 4

October-December 2002

The rising incidence of salmonella infection in New Zealand, 1995-2001

Craig Thornley, Public Health Physician; Michael Baker, Public Health Physician;
Carolyn Nicol, Laboratory Scientist, Institute of Environmental Science and Research Limited

Salmonellosis is caused by human infection with nontyphoidal subtypes of *Salmonella enterica*. Nontyphoidal salmonellosis has been increasing in New Zealand, so it is timely to review the epidemiology of this disease. Data sources were notifications from 1952, matched notification and laboratory data for the 1995-2001 period and disease outbreak data from 1998-2001. The average incidence of notified salmonellosis in New Zealand during 1995 to 2001 was 1706 cases per annum (46.1 per 100 000), including 167 hospitalisations per year. Twelve deaths occurred during the seven-year period (case fatality rate 0.1%). The highest annual incidence recorded in New Zealand was 2417 cases in 2001. Among laboratory-confirmed cases, the most frequently identified serotypes were *S. Typhimurium* (64.3%), *S. Enteritidis* (9.8%) and *S. Brandenburg* (6.1%). *S. Typhimurium* phage types 135 and 160 and *S. Brandenburg* increased over the period. Incidence rates were highest among children under five years (158.4 per 100 000), males (46.5) and Europeans (43.8). Rates were highest in the southern half of the South Island, especially South Canterbury and Southland health districts. Between 1998 and 2001, 137 outbreaks of salmonellosis were reported, with a median of three cases per outbreak. Laboratory-confirmed cases in outbreaks accounted for 5.0% of cases over that period. This review provides good evidence that the incidence of salmonellosis in New Zealand is increasing, particularly in the last five years. Control of this disease requires managing the multiple exposure sources that are important in New Zealand, including contaminated food, drinking water, occupational exposure to farm animals, and secondary transmission. Medical practitioners have a critical role in salmonellosis control, by diagnosing and promptly notifying cases to support outbreak recognition and control, and by advising cases or care-givers on ways of reducing the risk of secondary transmission.

Salmonellae are Gram-negative, non-spore-forming, rod-shaped bacteria, and are widely dispersed in nature, particularly in the gastrointestinal tracts of mammals, reptiles, birds, and insects.¹ Most medically important *Salmonella* organisms are from a single species, *Salmonella enterica*,² subdivided on the basis of capsular and flagella antigens into approximately 2500 serotypes. In practice, the species name tends to be omitted, for example *Salmonella enterica* serotype Typhimurium is abbreviated to *S. Typhimurium*. Organisms are further differentiated into numeric subtypes based on bacteriophage testing. Nontyphoidal salmonellosis is human illness caused by *Salmonella* serotypes other than *S. Typhi* or *S. Paratyphi*. Symptoms typically involve acute enterocolitis, commonly with diarrhoea, abdominal pain, nausea, and vomiting, commencing six to 72 hours after ingestion of contaminated material. Fever, if present, usually resolves in less than two days, and diarrhoea persists less than seven days. Infection may develop into septicaemia or focal infection, causing septic arthritis, cholecystitis,

Correspondence: Dr Craig Thornley, Auckland Regional Public Health Service, Private Bag 92 605, Symonds Street, Auckland. Email: craigt@adhb.govt.nz.

Changes to the New Zealand Public Health Report

From 2003, the New Zealand Public Health Report will be published quarterly as a combined publication with Lablink. We are in the process of reviewing the content and format of the new publication.

meningitis, pericarditis, pneumonia, pyoderma, or pyelonephritis.³

In recent decades, emerging *Salmonella* subtypes have raised the profile of nontyphoidal salmonellosis internationally. Infection with *S. Enteritidis* phage type (PT) 4, transmitted predominantly through eggs or in poultry meat, has become a global pandemic which continues to expand.⁴ The emergence of *S. Typhimurium* definitive type (DT) 104, resistant to a wide spectrum of antibiotics, also presents a major new threat.⁵ Both these pathogens are examples of new forms of zoonotic illness, having animal reservoirs but multiple routes of transmission to humans. Neither appears to have yet established endemic transmission in New Zealand, however, New Zealand has experienced similar zoonotic epidemics due to emerging *Salmonellae*: *S. Brandenburg*⁶ and *S. Typhimurium* DT160.⁷ This report describes the recent epidemiology of salmonellosis in New Zealand.

Contents

The rising incidence of salmonella infection in New Zealand, 1995-2001	25
Surveillance and control notes	29
Surveillance data	30
Public health abstracts	32
Travel health	32

Methods

Nontyphoidal salmonellosis has been notifiable in New Zealand since 1952, and notified cases require a clinical history of gastroenteritis and either laboratory confirmation of *Salmonella* species from a clinical specimen, contact with a confirmed case, or common exposure to the source of illness in a confirmed case. Notification data were obtained from annual reports of the Department of Health and Institute of Environmental Science and Research Ltd (ESR) records for nontyphoidal salmonellosis for the years 1952-2001. Crude incidence rates were calculated using census data as denominators, with linear interpolation used to estimate denominators for intercensal years.

Serotype and subtype information on all clinical *Salmonella* isolates identified by diagnostic laboratories in New Zealand are collected by the Enteric Reference Laboratory (ERL) at ESR, and were obtained for the years 1995-2001. Isolates had been serotyped using somatic (O) and flagellar (H) antigens according to the Kauffman-White scheme⁸. Bacteriophage typing had been used to characterise *S. Typhimurium* and *S. Enteritidis* strains, with definitive and phage types ascribed according to the schemes of Anderson et al.⁹ and Ward¹⁰ respectively. Antimicrobial drug resistance of *Salmonella* isolates in New Zealand is reported separately,¹¹ and is not repeated here. Following removal of duplicate records, these data were matched electronically with notifications using combinations of names, initials, sex, age, health district of residence and year of reporting, to create a dataset of laboratory-confirmed clinical cases. These cases were analysed by serotype and subtype, demographic characteristics, residential health district, risk factors and outcome of illness.

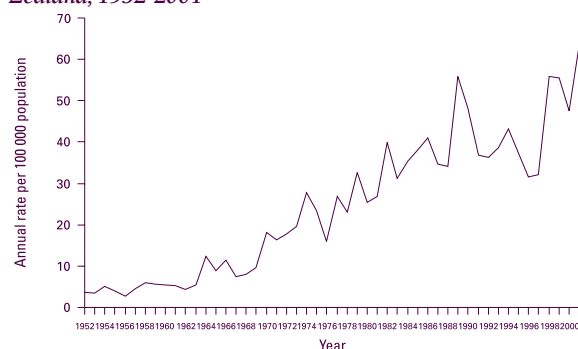
Salmonellosis outbreaks, defined as two or more cases of the same illness linked to a common source exposure, have been under surveillance since 1997. Data on outbreaks of salmonellosis reported between 1998 (the first full year of surveillance) and 2001 were obtained from ESR. Outbreaks were analysed by serotype and subtype, outbreak transmission routes and outbreak sources.

Results

The incidence of notified nontyphoidal salmonellosis has progressively increased during the years 1952-2001, reaching a peak of 2417 cases (64.7 per 100 000) in 2001 (Figure 1). During the 50 year period 1952 to 2001 there were 40 167 notified cases of salmonellosis. The median number of cases per year for the ten-year periods 1952-1961, 1962-1971, 1972-1981, 1982-1991 and 1992-2001 was 102, 249, 761, 1239, and 1431 respectively. There were 11 942 notifications during 1995-2001, an average annual incidence of 46.1 per 100 000. Of the 10 763 cases with information recorded on hospitalisation, 1170 (10.9%) had been hospitalised during their illness, with highest rates among those aged less than one year (15.6%) or older than sixty years (23.8%). Twenty cases were known to have died, although eight of these had died of a cause other than salmonellosis. The case-fatality rate was 0.1% (12/11 942).

During 1995-2001, 12 972 individual cases with nontyphoidal *Salmonella* infection were identified by laboratories. Of the notified cases for the same period, 11 245 (94.0%) could be matched with a corresponding laboratory record, and further analysis was restricted to these laboratory-confirmed cases. Sufficient typing information was available to report the infecting serotype for 11 083 cases (98.6%) (Table 1). Of these, 7125 (64.3%) were infected with *S. Typhimurium*, 1087 (9.8%) with *S. Enteritidis*, and 671 (6.1%) with *S. Brandenburg*.

Figure 1: Annual incidence of nontyphoidal salmonellosis in New Zealand, 1952-2001



Other serotypes responsible for >1% of cases were *S. Infantis* (286 cases), *S. Saintpaul* (190), *S. Heidelberg* (174), *S. Agona* (140) and *S. Hadar* (115).

The *S. Typhimurium* subtype was known for 6926 (97.2%) of cases with this serotype. Of these, the largest proportion were infected with definitive type (DT) 135 (1620, 23.4%) followed by DT1 (1076, 15.5%), DT160 (892, 12.9%), DT156 (828, 12.0%) and DT101 (705, 10.2%). The *S. Enteritidis* subtype was known for 1039 (95.6%) of cases with this serotype. Of these, the largest proportion were infected with PT9a (500 cases, 48.1%), followed by PT4 (363, 34.9%), PT1 (62, 6.0%) and PT26 (34, 3.3%).

The annual rate of *S. Typhimurium* infection remained stable between 1995 (20.2 cases per 100 000) and 1997 (18.0), then increased to 35.7 in 1998 and to 40.3 in 2001. The annual rate of *S. Enteritidis* remained stable during the seven-year period (4.8 in 1995 and 4.1 in 2001). Rates of infection with *S. Brandenburg* increased from 0.6 in 1995 to a peak of 4.4 in 2000. Rates of infection with *S. Heidelberg* remained stable for much of the period, at between 0.1 and 0.6, but increased to 3.3 in 2001.

Table 1: Selected *Salmonella* serotypes and subtypes of notified salmonellosis cases, 1995-2001

Subtype	1995	1996	1997	1998	1999	2000	2001	Total	%
<i>S. Typhimurium</i>									
DT135	67	74	114	296	459	370	240	1620	14.4
DT1	194	132	95	201	174	123	157	1076	9.6
DT160				1	4	169	718	892	7.9
DT156	135	105	99	149	151	91	98	828	7.4
DT101	38	39	62	258	131	112	65	705	6.3
DT9	40	32	52	149	43	84	38	438	3.9
DT42	55	37	31	55	68	49	32	327	2.9
DT23	38	9	38	37	29	20	53	224	2.0
DT12a	15	25	28	7	17	22	24	138	1.2
DT8	28	26	11	16	12	9	6	108	1.0
Other or unknown	120	133	122	123	118	76	77	769	6.8
Total	730	612	652	1292	1206	1125	1508	7125	63.4
<i>S. Enteritidis</i>									
PT9a	62	52	66	83	106	65	66	500	4.4
PT4	83	62	51	49	54	42	22	363	3.2
Other or unknown	29	25	24	32	27	21	66	224	2.0
Total	174	139	141	164	187	128	154	1087	9.7
<i>S. Brandenburg</i>	23	23	35	156	149	165	120	671	6.0
<i>S. Infantis</i>	25	43	29	21	71	32	65	286	2.5
<i>S. Saintpaul</i>	22	37	33	32	31	21	14	190	1.7
<i>S. Heidelberg</i>	14	22	2	6	5	3	122	174	1.5
<i>S. Agona</i>	17	19	16	25	35	12	16	140	1.2
<i>S. Virchow</i>	13	9	15	34	33	19	9	132	1.2
<i>S. Hadar</i>	27	13	12	17	20	14	12	115	1.0
<i>S. Weltevreden</i>	5	9	5	19	38	9	19	104	0.9
Other serotypes	166	129	134	164	139	141	186	1059	9.4
Unknown serotype	20	17	19	18	18	29	41	162	1.4
TOTAL	1236	1072	1093	1948	1932	1698	2266	11245	

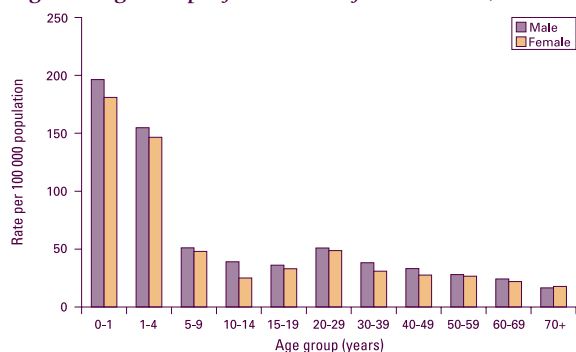
Demographic characteristics: Patient age was reported for all cases. The highest age-specific incidence rate was 191.5 per 100 000 for children aged under one year. 27.4% of cases were aged less than five years. The age-specific rates declined with increasing age and remained stable among adults, with a slight increase among adults aged 20-29 years. Information on sex was recorded for 99.1% of cases. The sex-specific rate was higher among males (46.5) than among females (40.4) overall and in each age group except persons aged 70 years or older (Figure 2). Ethnicity was recorded for 85.8% of cases. Of those with recorded ethnicity, the highest rate was for Europeans, at 43.8. The ethnic-specific rates were 21.9 among Maori, 26.3 among Pacific peoples, and 36.2 among other ethnic groups combined.

Seasonal distribution: The largest proportion of cases were reported in late Summer and early Autumn, peaking in March (13.1%), and the smallest proportion were reported in Winter, with the lowest point reached in July (4.9%). Most serovars and subtypes followed this general pattern, with the exception of *S. Brandenburg*, *S. Enteritidis* PT4 and *S. Typhimurium* DT160, which peaked in Spring, and *S. Enteritidis* PT9a which peaked in mid-Autumn.

Regional distribution: Rates of salmonellosis were highest in the southern half of the South Island during 1995-2001, with highest rates reported from South Canterbury (76.2 per 100 000) and Southland (72.7) health districts. The national average of 43.8 was exceeded in Wairarapa (66.3), Otago (59.4), Wellington (52.8), Canterbury (50.8), West Coast (48.5), Manawatu (44.2), Waikato (44.1), and Nelson-Marlborough (43.8) health districts. All health districts reported increasing numbers of cases over the seven-year period, and *S. Typhimurium* was universally the dominant serotype.

Risk factors: Information about recent travel was recorded for 9316 cases, 82.9% of the total. Of these cases, 1458 (15.7%) had a history of

Figure 2: Age-sex specific incidence of salmonellosis, 1995-2001



travel outside New Zealand during the incubation period of their illness. The most common region last visited by these cases was Southeast Asia (632, 43.3%), followed by the Pacific islands (297, 20.4%). The most commonly identified organism among cases with a history of overseas travel was *S. Enteritidis* PT4. Of cases between 1995 and 2001 infected with this subtype, 237 (83.7%) had a history of recent overseas travel, predominantly to Southeast Asia. Information about other risk factors was infrequently recorded for cases. Of those with information recorded, 23.0% had a history of recent consumption of food from a food premises, 13.6% had a history of recent consumption of untreated water, 8.7% had a history of recreational water use, 11.4% had a history of contact with another person with gastrointestinal symptoms, 9.5% had a history of contact with faecal matter or vomitus, and 23.3% had a history of contact with farm animals during the incubation period of their illness.

Outbreaks: Between 1998 and 2001, 137 salmonellosis outbreaks were reported. Case numbers were available for 134 outbreaks, and involved a total of 955 cases (median of three cases per outbreak). Of these cases, 390 were confirmed by laboratory testing, and accounted for 5.0% (390/7844) of confirmed cases notified between 1998 and 2001. Of the 112 (81.8%) outbreaks with information on infecting serotype or subtype, the most common infecting organism was *S. Typhimurium* DT135 (28 outbreaks), followed by *S. Typhimurium* DT160 (14) and *S. Typhimurium* DT1 (13). The most common form of transmission was foodborne (60.5%), followed by person-to-person spread (35.0%), animal contact (14.0%), environmental (13.8%) and waterborne transmission (10.5%). Of 29 foodborne outbreaks with single foods identified as the outbreak source, chicken was the most commonly reported source vehicle, accounting for 21 (72.4%) outbreaks.

Discussion

This analysis of routinely collected data suggests that the incidence of notified nontyphoidal salmonellosis has been increasing in New Zealand, particularly in the last five years, and in 2001 the incidence reached the highest ever recorded level. In contrast, nontyphoidal salmonellosis has recently declined in the United States,¹² Canada,¹³ Ireland,¹⁴ Scotland,¹⁵ and England and Wales.¹⁶ Over the course of the twentieth century, the aetiology of salmonella infections in New Zealand has shifted from *S. Typhi*, a solely human pathogen, to nontyphoidal salmonellae, which are widespread in the environment and colonise many domesticated and wild animal species. This transition has been observed in other developed countries.¹⁷

S. Typhimurium remains the dominant serotype among humans in New Zealand, as reported previously.¹⁸ Annual numbers of *S. Brandenburg*, *S. Typhimurium* DT160, *S. Typhimurium* DT135 and *S. Typhimurium* DT101 cases were at low or non-existent levels in 1995 but have substantially increased during the subsequent seven years. Both *S. Brandenburg* and *S. Typhimurium* DT160 have been associated with zoonotic transmission from specific species; from sheep⁶ and wild birds¹⁹ respectively. These serotypes may gradually decline in importance, as has been the case with *S. Hadar* which was the third most common serotype in 1992¹⁸ but accounted for only 1% of cases during 1995-2001. The decline of *S. Hadar* was largely due to eradication of the pathogen from poultry flocks. Incidence of infection with *S. Enteritidis* PT4 has declined during the last seven years, and the bulk of cases have a history of recent travel outside New Zealand. This suggests that endemic transmission of *S. Enteritidis* PT4 is currently rare and is not related to egg consumption in New Zealand.

The burden of salmonellosis in New Zealand falls most severely on the young, as over a quarter of notified cases aged less than five years. Children are susceptible to a lower infectious dose of *Salmonella* than are adults,²⁰ have wider levels of exposure to sources of infection,²¹ and are particularly vulnerable to intrafamilial transmission.²² Sex-specific incidence of infection was higher in males than in females, as has been noted previously.²³ This difference was observed in almost all age groups, so cannot be attributed to differential occupational exposures. Salmonellosis is more common during late summer and early autumn than at other times of the year. This is also characteristic of salmonellosis epidemiology in the Northern Hemisphere and has been attributed to variation in infection trends in animal hosts and to unsafe foodhandling during warmer months.¹² *S. Brandenburg* and *S. Typhimurium* DT160 infection rates peaked in spring due to seasonality in infection rates within their respective zoonotic reservoirs.^{6,7}

Incidence of salmonellosis during 1995-2001 has been highest in health districts at the southern tip of the North Island and in the South Island south of Canterbury. The emergence of *S. Brandenburg* has contributed to elevated rates in Southland, Otago and South Canterbury. These and other districts with elevated rates, with the exception of Wellington and Canterbury, characteristically have a high proportion of the population living in rural areas or having exposure to farming and agriculture.

The analysis suggests that infection acquired overseas, consumption of contaminated food and water, and contact with other persons with gastroenteritis are important salmonellosis risk factors. This information has limited value in understanding the sources of salmonellosis in New Zealand as it is largely collected from case histories and subject to selective recall, although overseas travel history is less likely to be subject to this bias. Travel can contribute to the introduction of new *Salmonella* serotypes,²⁴ with consequent threat to New Zealand's biosecurity (as would be the case with importation of *S. Enteritidis* PT4).

Data collected during disease outbreak investigations can provide better information about sources of salmonellosis. Transmission of bacteria in food (particularly poultry), by person-to-person spread and by animal contact have been implicated as causes of salmonellosis outbreaks reported between 1998 and 2001, although often without evidence from analytic epidemiological, microbiological or environmental investigation. Internationally, disease outbreak investigations have helped identify novel salmonellosis transmission mechanisms (for example, unpasteurised orange juice,²⁵ cheese,²⁶ ice cream,²⁷ chocolate,²⁸ marijuana,²⁹ and drinking water³⁰).

Most salmonella infections in New Zealand occur as sporadic cases and not in outbreaks. Unfortunately, case-control studies of sporadic salmonellosis have generally been unable to explain the majority of cases,³¹ although consumption of undercooked poultry³²⁻³⁴ and of undercooked eggs (in countries with endemic *S. Enteritidis* PT4)^{35,36} have consistently emerged as risk factors. Internationally, the proportion of salmonellosis linked to food is not known, but based on outbreak reports is considered to be high.³⁷ Characteristics of the New Zealand population suggest that zoonotic and environmental transmission may be more important than overseas, however it is likely that foodborne transmission has an important role.

Regardless of the final route of transmission of infection to humans, domesticated farm animals are likely to be the principal reservoirs of *Salmonella* within New Zealand, as elsewhere. Internationally, *Salmonella* carriage among domesticated animals has been increasing over time,³⁸ and the distribution of *Salmonella* serotypes in animals is similar to that causing salmonellosis in humans.³⁹ Information on *Salmonella* carriage among animals in New Zealand is, however limited. A nationwide survey of poultry products in 1992 and 1993 found that 15.2% of 164 poultry samples collected from poultry processors contained *Salmonella*, with a wide range of serotypes identified.⁴⁰

Globally, factors behind the increasing importance of nontyphoidal salmonellosis include human demographic change, alteration in consumer food preferences toward greater consumption of pre-prepared food or minimally cooked food, greater industrialisation of food production, globalisation of food supply, increased travel, altered pathogen characteristics and deterioration in public health infrastructure²⁴. Changes in animal husbandry practices may also have increased exposure of humans to pathogenic nontyphoidal *Salmonella*. In one example, successful programmes to eliminate poultry infection with *S. Gallinarum* (nonpathogenic to humans⁴¹) may have allowed poultry

colonisation with the previously competitively-excluded *S. Enteritidis*, which is nonpathogenic to poultry but causes illness among humans.⁴² Tauxe calls the current trend in salmonellosis epidemiology a 'postmodern paradox', because human salmonellosis is predominantly due to consumption of food produced from animals that are colonised by *Salmonella* but remain healthy.¹⁷

These data underestimate the true salmonellosis burden in New Zealand because many infected individuals will not visit a doctor or have confirmatory tests performed. In the US and the UK, studies have shown that the number of persons who receive a microbiological diagnosis of *Salmonella* infection is a small minority of those in the community with diagnosable illness.^{43, 44} New Zealand general practitioners refer diarrhoeal specimens from less than 25% of those with acute gastroenteritis.⁴⁵ The apparent increase in notified salmonellosis may therefore be partly due to changes in this under-ascertainment due to increased healthcare-seeking behaviour and utilisation of diagnostic testing. However, New Zealand Health Information Service (NZHIS) hospitalisation data shows that admissions for nontyphoidal salmonellosis have also increased, from 98 in 1995 to 135 in 2000 (personal communication, Chris Lewis, NZHIS), demonstrating that the same trend has been observed in independently-collected data.

Control of human salmonellosis requires a multi-level approach throughout food production, processing and preparation. Points of intervention to reduce salmonellosis include selection of salmonella-free stock; protection of animal feed and water from contamination; hygienic disposal of farm and abattoir waste; cleaning to reduce contamination of intensive animal-rearing areas; use of hygienic practices in abattoirs, such as cleaning animals before slaughter, use of chillers to prevent pathogen growth on food, and improvements to evisceration processes to minimise rupture of intestinal contents; irradiation of packed product; and high standards of food safety during food handling, whether in commercial or domestic settings, including policies to manage sick food handlers.⁴¹

Medical practitioners have a crucial role in salmonellosis control. Stool cultures should be collected from persons presenting with persistent diarrhoea. Notification of persons with confirmed salmonellosis enables public health workers to detect outbreaks and implement control measures to limit ongoing outbreaks.²⁷ Other benefits of salmonellosis surveillance include detection of emerging organisms or hazards, characterisation of the burden of illness, and evaluating the impact of control strategies. Antibiotic treatment of enteric salmonellosis prolongs *Salmonella* excretion in faeces, and is therefore contraindicated.⁴⁶ It is also important to advise cases or their caregivers to take care to reduce secondary transmission, by promoting meticulous hand hygiene (emphasising the importance both of washing and drying hands) and exclusion from food preparation and early childhood centres until well. Food workers require two negative stool specimens, taken at least 48 hours apart, before returning to food preparation.

Acknowledgements: Thanks to Michael Eglinton and Kylie Gilmore for the data-matching, to ERL staff for laboratory testing, and to public health units and medical practitioners throughout New Zealand for continuing support of salmonellosis surveillance.

References

- Miller S, Pegues D. *Salmonella* species, including *Salmonella* typhi. In: Mandell G, Bennett J, Dolin R, eds. Principles and practice of infectious disease. Vol. 5. Philadelphia: Churchill Livingstone; 2000.
- Threlfall J, Ward L, Old D. Changing the nomenclature of *Salmonella*. Commun Dis Public Health 1999; 2:156-7.
- Chin J. Control of communicable diseases manual. Washington: American Public Health Association; 2000.
- Herikstad H, Motarjemi Y, Tauxe RV. *Salmonella* surveillance: a global survey of public health serotyping. Epidemiol Infect 2002; 129:1-8.
- Wall PG, Morgan D, Lamden K, et al. A case control study of infection with an epidemic strain of multiresistant *Salmonella typhimurium* DT104 in England and Wales. Commun Dis Rep CDR Rev 1994; 4:R130-5.
- Peacock D, Baker M, Fraser G, Holmes J. An investigation of a recent *Salmonella* Brandenburg outbreak in southern New Zealand. Dunedin: HealthCare Otago; 1999.
- Alley MR, Connolly JH, Fenwick SG, et al. An epidemic of salmonellosis caused by *Salmonella* Typhimurium DT160 in wild birds and humans in New Zealand. N Z Vet J 2002; 50:170-6.
- Popoff MY. Antigenic formulas of the *Salmonella* serovars. Paris: WHO Collaborating Centre for Reference and Research on Salmonella; 2001.
- Anderson ES, Ward LR, Saxe MJ, de Sa JD. Bacteriophage-typing designations of *Salmonella typhimurium*. J Hyg (Lond) 1977; 78:297-300.

- Ward LR, de Sa JD, Rowe B. A phage-typing scheme for *Salmonella enteritidis*. Epidemiol Infect 1987; 99:291-4.
- Antibiotic susceptibilities of *Salmonella*. Lablink 2002; 9:11.
- Olsen SJ, Bishop R, Brenner FW, et al. The changing epidemiology of salmonella: trends in serotypes isolated from humans in the United States, 1987-1997. J Infect Dis 2001; 183:753-61.
- Centre for Infectious Disease Prevention and Control. Notifiable diseases annual summary 1999. Canada Comm Dis Rep 2001; 27S6:1-147.
- National Disease Surveillance Centre. Annual report of the National Disease Surveillance Centre, 2000. Ireland: National Disease Surveillance Centre (NDSC); 2001.
- Scottish Centre for Infection and Environmental Health. Review of communicable diseases in Scotland 1999. Glasgow: Scottish Centre for Infection and Environmental Health; 2000.
- Trends in selected gastrointestinal infections - 2001. CDR Wkly (Online) 2002; 12.
- Tauxe RV. *Salmonella*: a postmodern pathogen. J Food Protect 1991; 54:563-8.
- Fraser D. *Salmonella* infection. N Z Med J 1993; 106:510.
- Thornley C, Simmons G, Nicol C, et al. Investigation of an outbreak of *Salmonella* Typhimurium DT160. Porirua: Institute of Environmental Science & Research Limited; 2002.
- Blaser MJ, Newman LS. A review of human salmonellosis: I. Infective dose. Rev Infect Dis 1982; 4:1096-106.
- Schutze GE, Sikes JD, Stefanova R, Cave MD. The home environment and salmonellosis in children. Pediatrics 1999; 103:E1.
- Wilson R, Feldman RA, Davis J, LaVenture M. Salmonellosis in infants: the importance of intrafamilial transmission. Pediatrics 1982; 69:436-8.
- Fraser D. The epidemiology and control of *Salmonella* infection in New Zealand. Commun Dis N Z 1993; 93:73-7.
- Altekruse SF, Cohen ML, Swerdlow DL. Emerging foodborne diseases. Emerg Infect Dis 1997; 3:285-93.
- Cook KA, Dobbs TE, Hlady WG, et al. Outbreak of *Salmonella* serotype Hartford infections associated with unpasteurized orange juice. JAMA 1998; 280:1504-9.
- Hedberg CW, Korlath JA, D'Aoust JY, et al. A multistate outbreak of *Salmonella javiana* and *Salmonella oranienburg* infections due to consumption of contaminated cheese. JAMA 1992; 268:3203-7.
- Hennessy TW, Hedberg CW, Slutsker L, et al. A national outbreak of *Salmonella enteritidis* infections from ice cream. N Engl J Med 1996; 334:1281-1325.
- Kapperud G, Gustavsen S, Hellesnes I, et al. Outbreak of *Salmonella typhimurium* infection traced to contaminated chocolate and caused by a strain lacking the 60-megadalton virulence plasmid. J Clin Microbiol 1990; 28:2597-601.
- Taylor D, Wachsmuth I, Shangkuang Y, et al. Salmonellosis associated with marijuana: a multistate outbreak traced by plasmid fingerprinting. New England Journal of Medicine 1982; 306:1249-53.
- Angulo FJ, Tippen S, Sharp DJ, et al. A community waterborne outbreak of salmonellosis and the effectiveness of a boil water order. Am J Public Health 1997; 87:580-4.
- Kapperud G, Lassen J, Hasseltvedt V. *Salmonella* infections in Norway: descriptive epidemiology and a case-control study. Epidemiol Infect 1998; 121:569-77.
- Kass PH, Farver TB, Beaumont JJ, Genigeorgis C, Stevens F. Disease determinants of sporadic salmonellosis in four northern California counties. A case-control study of older children and adults. Ann Epidemiol 1992; 2:683-96.
- Cowden JM, Lynch D, Joseph CA, et al. Case-control study of infections with *Salmonella enteritidis* phage type 4 in England. BMJ 1989; 299:771-3.
- Humphrey TJ, Mead GC, Rowe B. Poultry meat as a source of human salmonellosis in England and Wales. Epidemiological overview. Epidemiol Infect 1988; 100:175-84.
- Hayes S, Nylen G, Smith R, et al. Undercooked hens eggs remain a risk factor for sporadic *Salmonella enteritidis* infection. Commun Dis Public Health 1999; 2:66-7.
- Delarocque-Astagneau E, Desenclos JC, Bouvet P, Grimont PA. Risk factors for the occurrence of sporadic *Salmonella enterica* serotype *enteritidis* infections in children in France: a national case-control study. Epidemiol Infect 1998; 121:561-7.
- Tauxe RV, Blake PA. Salmonellosis. In: Last JM, Wallace RB, Barrett-Conner E, eds. Public health and preventative medicine. Norwalk, Connecticut: Appleton & Lange; 1992.
- Cohen ML, Tauxe RV. Drug-resistant *Salmonella* in the United States: an epidemiologic perspective. Science 1986; 234:964-9.
- Khakhria R, Woodward D, Johnson WM, Poppe C. *Salmonella* isolated from humans, animals and other sources in Canada, 1983-92. Epidemiol Infect 1997; 119:15-23.
- Gilbert S. Campylobacter and Salmonella contamination of poultry. Commun Dis N Z 1993; 93:63-4.
- Baird-Parker AC. Foodborne salmonellosis. Lancet 1990; 336:1231-5.
- Rabsch W, Hargis BM, Tsois RM, et al. Competitive exclusion of *Salmonella enteritidis* by *Salmonella gallinarum* in poultry. Emerg Infect Dis 2000; 6:443-8.
- Chalker RB, Blaser MJ. A review of human salmonellosis: III. Magnitude of *Salmonella* infection in the United States. Rev Infect Dis 1988; 10:111-24.
- Wheeler JG, Sethi D, Cowden JM, et al. Study of infectious intestinal disease in England: rates presenting to general practice, and reported to national surveillance. BMJ 1999; 318:1046-50.
- Sarfati D, Bates MN, Garrett N, Baker MG. Acute gastroenteritis diagnostic practices of New Zealand general practitioners. N Z Med J 1997; 110:354-6.
- Aserkoff B, Bennett JV. Effect of antibiotic therapy in acute salmonellosis on the fecal excretion of salmonellae. N Engl J Med 1969; 281:636-40.

Rare anaphylactic reaction following a Mantoux test

During a TB contact tracing clinic held by Regional Public Health in Wellington in November 2001, a 40 year old woman was given a 5TU mantoux test. She had a BCG as an adolescent so this was thought to be her second exposure to tuberculin. She was asked to remain in the clinic for 20 minutes following the Mantoux administration in accordance with the service protocol.

Approximately 10 minutes after the Mantoux test the woman complained of feeling unwell. She was pale, sweaty and cool to touch. Her pulse was 80 but of weak volume. Her conscious state was unaltered but she said that she felt she was "slipping off the edge". She was placed in the recovery position and given oxygen, adrenaline was drawn up and an ambulance called. Approximately 12 minutes after the Mantoux she developed an audible wheeze, complained of chest pain and was agitated. Adrenaline 1:1000 0.5ml was given intramuscularly. Paramedics arrived within ten minutes of symptoms and she was transported to Wellington Hospital. On arrival in the emergency department, she was noted to be wheezy and was still experiencing severe chest pain. She was given intravenous morphine and nebulised ventolin. She was observed in hospital for 12 hours and then discharged under cardiology review.

Reports of serious adverse reactions to tuberculin are extremely rare despite the millions of doses which have been administered world-wide. In New Zealand the Centre for Adverse Reactions Monitoring (CARM) have recorded eight reports of adverse reactions since 1965. There was only one previous report of anaphylaxis. This occurred in 1997 in a 47 year old woman after a second screening Mantoux test. The woman required emergency intervention of adrenaline and, later, hydrocortisone.

Of note both cases of anaphylaxis in New Zealand occurred in highly sensitised individuals. The case in 1997 was allergic to penicillin. The 2001 Wellington case was allergic to penicillin, tetracyclines and wasp stings, and was an asthmatic.

This event highlights the need for routine anaphylaxis procedures to be in place when performing Mantoux testing. Following injection of tuberculin people should remain for 20 minutes in the clinic. Staff administering tuberculin should be trained in CPR, carry adrenaline and have access to a phone. (reported by Margot McLean, Hutt Valley Health).

Reptile-associated Salmonellosis in an Auckland child

A case of salmonellosis due to *Salmonella* Paratyphi B was recently investigated by the Auckland Regional Public Health Service. The case involved a three year-old female who developed symptoms of lethargy, stomach cramp and abdominal pain on 8 April 2002. These symptoms progressed to fever (40°C), diarrhoea and vomiting over the next 12 hours. The case was hospitalised on 11 April 2002, discharged 2 days later but not notified until 23 April 2002. Stool cultures identified *Salmonella* spp. later confirmed as *Salmonella* Paratyphi B. Symptoms persisted for 14 days and no antibiotics were administered.

The case had attended an early childhood education centre (ECC) on 8 April 2002. Nine other children from the centre had been reported absent with gastrointestinal symptoms (short duration diarrhoea and vomiting) in the preceding week. Children with gastrointestinal symptoms were requested to produce faecal samples but the response was poor with only one submitting a negative sample.

The case investigation did not identify any of the risk factors or sources commonly found for paratyphoid cases in New Zealand. The food history for the 10 days prior to the onset of illness was unremarkable. All meals except one were prepared at home by the case's mother and no shellfish, untreated water, raw fruit or vegetables were consumed during the period.

There was no history of overseas travel or contact with visitors from overseas. There had been no exposure to recreational water or farm animals although there had been contact with a household cat. On one occasion food was consumed from a takeaway outlet.

On the day prior to the onset of illness the case had visited her grandmother who kept a pet turtle in an aquarium in the home. There had been contact between the case and the snake-necked turtle (*Chelodina longicollis*). After feeding the turtle and putting her hands in the aquarium water the case was observed on one occasion to lick her fingers.

The environmental investigation consisted of culturing the turtle food, aquarium water, and the faeces of the turtle after removing it from the aquarium.

The turtle food was culture negative but *Salmonella* Paratyphi B was isolated from the aquarium water and the turtle's faeces. *Salmonella* Paratyphi B isolates from the case, aquarium water and turtle faeces were indistinguishable on pulsed field gel electrophoresis.

Reptiles in general and turtles in particular may be colonised with *Salmonella* spp. and shed the organism in their faeces¹⁻³. Reptile-associated salmonellosis has been well documented overseas⁴, and particularly the relationship between exposure to pet turtles and salmonellosis in early childhood⁵.

Pet shop proprietors are aware of the salmonellosis hazard that turtles represent and generally counsel buyers about the risks. Public health recommendations have been made in the United States to prevent the transmission of *Salmonella* from reptiles to humans⁶. These recommendations are just as relevant to New Zealand and include:

- The avoidance of contact between reptiles and those at highest risk of infection (children < 5 years old and the immune compromised) and the exclusion of pet reptiles from homes where there are children under one year old, immune compromised people and from early child care centres.
- Restriction of pet reptiles to cages or aquarium environments and exclusion from the kitchen and food preparation areas.
- Thorough hand hygiene (washing hands with soap and water and drying hands on a clean, dry towel) after touching reptiles, or having touch contact with their enclosures.

This case highlights the morbidity associated with paratyphoid salmonellosis and its risk of transmission by turtles. Questioning about contact with reptiles should be routine when investigating cases of salmonellosis and in particular paratyphoid.

Reported by Dr Greg Simmons and Public Health Nurse Jill Miller

References

1. Burnham B, Atchley D, DeFusco R, et al. Prevalence of faecal shedding or *Salmonella* organisms among captive green iguanas and potential public health implications. J Am Vet Med Assoc 1998; 213:48-50
2. Kaufmann A, Fox M, Morris G, et al. An epidemiologic study of salmonellosis in turtles. AM J Epidemiol 1966; 84:364-70
3. Shane S, Gilbert R, Harrington K. *Salmonella* colonization in commercial pet turtles (*Pseudemys scripta elegans*). Epidemiol Infect 1990; 105:307-16
4. Ackman D, Drabkin P, Birkhead G, et al. Reptile-associated salmonellosis in New York State. Pediatr Infect Dis J 1995; 14:955-9
5. Altman R, Gorman J, Bernhardt L, et al. Turtle-associated salmonellosis: II. The relationship of pet turtles to salmonellosis in children in New Jersey. AM J Epidemiol 1972; 95:518-20
6. Anonymous. Reptile-associated salmonellosis - selected states, 1996-98. MMWR 1999; 48:1009-1012

Surveillance data

National surveillance data - October to December 2002

Disease ^{1,2}	Current year - 2002 ³			Previous year - 2001			Disease trends - Year ending December 2002
	Oct-Dec 2002 cases	Cumulative total since 1 January	Current rate ⁴	Oct-Dec 2001 cases	Cumulative total since 1 January	Previous rate ⁴	
AIDS	2	18	0.5	7	26	0.7	
Campylobacteriosis	3473	12488	334.1	4039	10145	271.4	**
Cholera	0	1	0	1	3	0.1	
Cryptosporidiosis	400	974	26.1	385	1208	32.3	**
Dengue fever	10	70	1.9	24	93	2.5	
Gastroenteritis ⁵	361	1084	29.0	243	942	25.2	**
Giardiasis	337	1548	41.4	394	1603	42.9	
<i>H influenzae</i> type b disease	0	3	0.1	1	11	0.3	
Hepatitis A	15	108	2.9	17	61	1.6	**
Hepatitis B (acute) ⁶	17	69	1.8	13	56	1.5	
Hepatitis C (acute) ⁶	8	52	1.4	13	59	1.6	
Hydatid disease	1	2	0.1	3	7	0.2	
Influenza ⁷	23	698	18.7	21	666	17.8	
Lead absorption	18	90	2.4	22	130	3.5	**
Legionellosis ⁷	10	48	1.3	11	57	1.5	
Leprosy	1	4	0.1	0	3	0.1	
Leptospirosis	32	142	3.8	27	105	2.8	*
Listeriosis	5	18	0.5	5	18	0.5	
Malaria	7	60	1.6	11	54	1.4	**
Measles	4	21	0.6	35	83	2.2	**
Meningococcal disease ⁸	103	557	14.9	188	649	17.4	
Mumps	19	64	1.7	6	56	1.5	**
Paratyphoid	2	16	0.4	8	32	0.9	***
Pertussis	287	1068	28.6	191	1334	35.7	
Rheumatic fever	23	92	2.5	8	117	3.1	
Rickettsial disease	0	6	0.2	2	5	0.1	
Rubella	4	33	0.9	4	30	0.8	
Salmonellosis	394	1879	50.3	745	2417	64.7	**
Shigellosis	21	112	3.0	21	157	4.2	**
Tetanus	0	1	0	1	4	0.1	
Tuberculosis	114	380	10.2	107	373	10.0	
Typhoid	4	23	0.6	7	27	0.7	
VTEC/STEC infection	13	73	2.0	12	76	2.0	
Yersiniosis	125	476	12.7	131	429	11.5	

Notes: 1 Data on surveillance of Creutzfeld-Jacob disease have been removed from the quarterly surveillance data tables, and replaced by rickettsial disease surveillance data. Annual summary statistics for Creutzfeld-Jacob disease will be available in the Annual Surveillance Summary.

2 Other notifiable infectious diseases reported in October to December 2002: Nil

3 These data are provisional

4 Rate is based on the cumulative total for the current year (12 months up to and including December 2002) or the previous year (12 months up to and including December 2001), expressed as cases per 100 000

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

6 Only acute cases of this disease are currently notifiable

7 Surveillance data based on laboratory-reported cases only

8 Totals and rates are based on the EpiSurv report date as opposed to the earliest available date used in the meningococcal disease section

9 Percentage rate change is the difference between the number of cases in the current year (12 months up to and including September 2002) and the previous year (12 months up to and including September 2001). This difference is expressed as a percentage of the number of cases in the previous year

Surveillance data

Surveillance data by health district - October to December 2002 quarter

Cases this quarter Current rate²

Disease	Cases for October to December 2002, ² and current rate ^{1,2} by health district ^{3,4,5}																							
	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 ⁴	0	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Campylobacteriosis	64	480	412	368	361	68	18	27	58	26	91	7	103	45	77	24	333	123	75	26	365	68	152	102
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
Cryptosporidiosis	1	15	8	7	47	8	2	9	8	15	4	29	12	25	4	23	6	11	12	38	35	51	30	
Dengue fever	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0	1	2	0	0	0	6	0	0	0
Gastroenteritis	1	29	27	20	9	2	0	14	1	4	4	0	0	3	1	3	25	12	6	2	105	64	26	3
Giardiasis	11	38	55	26	40	9	2	2	8	3	4	0	19	4	12	1	36	10	4	2	23	8	13	7
H influenzae type b 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
Hepatitis A	1	2	2	5	1	0	1	0	0	0	0	0	1	0	0	0	0	0	0	0	1	0	1	0
Hepatitis B	2	1	1	1	2	1	0	1	0	1	0	0	0	1	0	1	0	1	2	0	2	0	0	0
Hepatitis C	0	1	1	0	0	1	1	0	1	0	0	0	0	0	0	0	2	0	0	1	0	0	0	0
Hydatids	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Lead absorption	1	1	0	0	2	0	0	0	0	2	0	2	0	1	1	1	0	1	0	1	1	1	3	1
Legionellosis ⁶	0	1	0	0	0	0	0	0	1	0	0	2	0	0	1	1	1	1	0	0	2	0	1	0
Leprosy	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Leptospirosis	2	1	0	1	2	0	0	3	0	0	3	1	4	1	3	1	0	0	5	0	1	1	3	0
Listeriosis	0	1	2	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	1	0	0	0
Malaria	0	2	0	0	1	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0	2	0	0	1
Measles	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	1	2	0	0	0
Meningococcal disease ⁷	4	5	9	10	5	7	2	0	9	4	2	0	5	2	1	0	8	3	0	1	10	3	10	3
Mumps	1	2	2	3	0	1	0	0	1	0	0	0	0	0	0	0	0	1	2	0	1	0	5	0
Paratyphoid	0	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Pertussis	3	39	3	7	30	6	1	0	1	2	13	0	5	28	18	2	8	25	32	14	39	8	0	3
Rheumatic fever	1	2	2	10	2	1	0	0	0	0	0	0	1	0	0	1	2	0	1	0	0	0	0	0
Rickettsial 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
Rubella	0	0	0	0	0	0	0	0	0	0	0	0	3	0	0	0	0	0	0	1	0	0	0	0
Salmonellosis	9	44	39	30	28	14	5	13	9	1	4	3	18	7	9	5	37	15	9	3	38	12	19	23
Shigellosis	0	1	6	2	3	0	0	0	0	0	0	0	2	1	0	0	0	0	1	0	3	5	2	0
Tetanus	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1.3	0	0
Tuberculosis	3	33	19	13	6	3	0	0	0	0	1	0	13	0	1	0	7	4	0	0	9	1	1	0
Typhoid	0	1	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	1	0	0	0
VTEC/STEC infection	0	0	0	0	3	0	0	0	0	0	0	0	1	1	0	0	2	0	0	0	5	0	0	1
Yersiniosis	2	20	21	11	12	3	1	0	1	1	0	0	6	1	2	1	7	1	0	9	14	6	3	3

Notes: 1 Data on surveillance of Creutzfeldt-Jacob disease have been removed from the quarterly surveillance data tables, and replaced by rickettsial disease surveillance data. Annual summary statistics for Creutzfeldt-Jacob disease will be available in the Annual Surveillance Summary. Influenza surveillance data have also been removed from the health district data table, but are retained in the national surveillance data table.

2 Current rate is based on the cumulative total for the 12 months up to and including December 2002, expressed as cases per 100 000

3 These data are provisional

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

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

6 Surveillance data based on laboratory-reported cases only

7 These totals and rates are derived from the EpiSurv report date as opposed to the earliest available date used in the meningococcal disease section

Public health abstracts

Legionellae and hot water tanks

An environmental case control study in Spain found an association between the presence of cisterns and hot water tanks in residential water systems and the colonisation of the water by legionellae. The aim of this two year matched case control study was to determine the factors associated with the occurrence of community acquired legionnaire's disease unrelated to large buildings. Several factors related to residential water distribution systems and public drinking water systems were studied for 124 case homes and 354 controls. Levels of legionellae in domestic water samples were measured from 113 case homes and 32 control homes. Legionellae was isolated in 8% of the case homes and in 19% of the control homes. Among home water systems from which samples positive for legionellae were obtained, the percentage of homes with a cistern or hot water tank were 73% and 47% respectively. However, the presence of these did not seem to be a risk factor for the acquisition of legionellosis. Problems occurring in the public water systems during the 2 weeks prior to the patients symptoms were not associated with community acquired legionellosis. None of the studied variables seem to increase the incidence

of community acquired legionnaires disease (Codony F, Alvarez J, Oliva JM, et al. Factors promoting colonization by Legionellae in residential water distribution systems: an environmental case control survey. Eur J Clin Microbiol Infect Dis, 2002; 21: 717-21).

Editorial Note: Fifty one Legionella cases were notified from January to December 2002. None of these cases were part of an outbreak. A New Zealand study surveying one hundred households with electrically heated water systems in the Wellington area found Legionella present in six to twelve percent of the houses surveyed. The only environmental risk factor found to be associated with the presence of legionella was recent plumbing work on the hot water systems which may be related to the disturbance of biofilms- the linings of the associated pipework. (Bates MN, Maas E, Martin T, et al. Investigation of the prevalence of Legionella species in domestic hot water systems. N Z Med J 2000; 113: 218-20) The New Zealand building code requires that hot water be stored at temperatures high enough (60C) to prevent the growth of Legionella species in hot water storage tanks.

Travel health

Schistosomiasis is an important hazard of swimming in African waterways

Schistosomiasis (bilharziasis) is a parasitic infection caused by blood flukes belonging to five species of *Schistosoma*. Infection is acquired by exposure to fresh water that contains cercariae released by infected snails. The increase in ecotourism and adventure tourism has resulted in an increasing number of cases in travellers. Some tourist destinations in Africa, notably Lake Malawi, are common sources of infections. Clinical presentation of the infection in non-immune tourists typically differs from that in the local population in endemic areas. The majority of initial infections cause mild transient symptoms, so go unnoticed, while the infection progresses to the chronic phase. The diagnosis is usually made on the basis of clinical features and travel history, often years after the trip. Urinary infection may present with haematuria, dysuria and nocturia. Gastrointestinal infection may cause bloody diarrhoea or chronic colitis. *Schistosoma* infection is definitively diagnosed by demonstrating the worm eggs in urine or faeces. Should these tests be negative, and the disease is still suspected, then serological testing is worthwhile. Antibodies may not develop for some time, so serological testing should not be done until at least three months after exposure. Praziquantel is a safe and highly effective drug for the treatment of schistosomiasis. (Corachan M. Schistosomiasis and international travel. Clin Infect Dis 2002; 35: 446-50.)

Editorial note: The incidence of schistosomiasis in returning travellers to New Zealand is unknown. An Auckland survey from the Worldwide travellers health centres over the 1997-2001 period showed an incidence of the disease of 4% (8/205) in returning travellers. The majority of these cases acquired their infection in African lakes and rivers, including Lake Malawi. Few cases are hospitalised, with an averaged of 5 cases a year from 1995-2002. All travellers to Africa, and relevant areas of South America, the Caribbean, the middle East and Asia, should be warned about the risks of swimming in shallow, still or slow moving waterways. Clinicians assessing patients with a history of travel to infected regions should have a high level of suspicion for this disease.

Acknowledgements

The Editors and Editorial Committee thank the following people for their contributions to the New Zealand Public Health Report in 2002:

- The authors of the lead articles
- Alison Williams, Helen Heffernan, Nikki Turner, Mel Brieseman, Trevor Taylor, Fabrizio Bruschi, Tim Blackmore, Greg Appleyard, Ashley Bloomfield, John Kaldor, Richard Meech, Fiona Thomson-Carter, Greg Simmons, John Threlfall, Ministry of Health staff, and ESR staff for reviewing articles and expert opinion.
- Liz Sneyd, Liza Lopez, Trev Margolin, Rebecca McDowell, Michael Eglington, and Carol Kliem for preparation of data and copy of the surveillance and control notes, and surveillance data.
- Medical practitioners, clinical laboratory staff, and public health unit staff who provide surveillance data.

New Zealand Public Health Report is produced quarterly by ESR for the Ministry of Health.

Internet website: <http://www.moh.govt.nz/nzphr.html>

Scientific Editor: Michael Baker, Public Health Physician, ESR
Managing Editor: Rabia Khan, Public Health Researcher, ESR
Editorial Committee: Sally Gilbert, Senior Advisor, Ministry of Health
Douglas Lush, Senior Advisor, Ministry of Health

Reprinting: Articles in the *New Zealand Public Health Report* may be reprinted provided proper acknowledgement is made to the author and to the *New Zealand Public Health Report* as source.

Contributions to this publication are invited, in the form of concise reports on surveillance, outbreak investigations, research activities, policy and practice updates, or brief review articles. Please send contributions to:

Scientific Editor, New Zealand Public Health Report, ESR, PO Box 50-348, Porirua, Wellington, New Zealand. Phone: (04) 914 0700; Fax: (04) 914 0770; Email: michael.baker@esr.cri.nz

The content of this publication does not necessarily reflect the views and policies of ESR or the Ministry of Health.



Phone: (04) 914 0700
Fax: (04) 914 0770



MANATU HAUORA
Phone: (04) 496 2000
Fax: (04) 496 2340