INFECTIOUS DISEASES IN NEW ZEALAND:
2002 ANNUAL SURVEILLANCE SUMMARY

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EXECUTIVE SUMMARY

This report aims to provide a concise review of the descriptive epidemiology of the main infectious diseases under public health surveillance in New Zealand, as well as lead absorption, which is also monitored using the notifiable disease surveillance system. The focus is on events and trends that emerged in 2002. The organisation of this report has been extensively updated from previous years with the diseases under surveillance been grouped into categories that reflect modes of transmission and control measures. These categories are similar to those used in the Ministry of Health's Integrated Approach to Infectious Diseases. This report also draws on a wider range of data sources than previously, including New Zealand Health Information Service (NZHIS) hospitalisation data, to provide a more comprehensive description of infectious disease epidemiology in New Zealand.

Quality of surveillance data

It is important to recognise the limitations of the surveillance data contained in this report. The first major limitation of infectious disease surveillance is that not all important infectious diseases (particularly those that are not currently notifiable) are under effective national public health surveillance.

Even where a disease is notifiable for surveillance purposes, the data inevitably have quality limitations. This report has used hospitalisation data to assess some dimensions of the quality of the notifiable diseases surveillance system:

- *Sensitivity* – The sensitivity of meningococcal disease surveillance is probably in excess of 87%. The sensitivity of surveillance for other diseases will often be less, particularly for common enteric diseases where only a small proportion of those infected will present to the health system. The system is inherently less sensitive for surveillance of chronic infections, notably hepatitis B and C, where initial infection is frequently asymptomatic (the same is true for surveillance of lead absorption), and for other conditions resulting from environmental exposure.
- *Positive predictive value* – This is relatively high for most infectious diseases under surveillance. High PPV is necessary for meningococcal disease surveillance, which will be used, among other things, to measure the effectiveness of the planned vaccination programme; and for measles surveillance, as New Zealand approaches the goal of measles elimination.
- *Representativeness* – For some diseases, notification data may be poorly representative of disease epidemiology. For example, hospitalisation data suggest that Maori and Pacific peoples have higher rates of salmonellosis and pertussis than Europeans, which is the opposite to the pattern shown by notification data. Further work would be useful to quantify these biases.
- *Completeness of associated data* – This report has also identified gaps in the completeness of associated data used to describe important demographic, outcome and risk factor, characteristics of cases.

- *Data errors* –There are unquantifiable errors in the data contained in this report. These errors are likely to be greater for rates than for simple counts of cases, since rates depend on both an accurate numerator and an accurate and appropriately chosen denominator.

Vaccine-preventable infections

Influenza: During the 2002 sentinel surveillance season (May through September), the national average weekly consultation rate was 43.2 per 100 000 patient population, compared with 62.8 per 100 000 in 2001. However, the total number of influenza isolates identified rose from 303 in 2001 to 702 in 2002; and the number of hospitalisations due to influenza also increased from 388 in 2001 to 483 in 2002. Influenza A (H3N2) was the predominant strain in 2002, accounting for 78% of the total 702 isolates identified. This strain has previously been associated with more severe influenza seasons, in 1996 and 1999. Reducing the impact of this disease depends on increasing appropriate vaccine coverage.

Pertussis: The national pertussis epidemic, that began in June 1999 and peaked in 2000, persisted into 2001. The incidence rate fell to 28.7 per 100 000 in 2002, but was nevertheless higher than in the preceding inter-epidemic period. In New Zealand pertussis epidemics tend to occur every four to five years as the number of susceptibles in the population increase as a result of incomplete vaccination and waning vaccine immunity. Control of this disease depends on increasing vaccine coverage rates and possibly modification of the immunisation schedule.

Other vaccine preventable diseases: Measles, mumps, and rubella incidence rates remained low in 2002. The measles epidemic which might have been expected in 2002 (based on a five to six year inter-epidemic period) did not eventuate. *Haemophilus influenzae* type b (Hib) disease continued to decline following addition of Hib vaccine to the immunisation schedule in 1994. Hepatitis B incidence has gradually declined following the introduction of universal childhood vaccination in the 1980's and new infections are now largely in adult populations.

Infectious respiratory diseases and diseases from close contact

Acute Rheumatic Fever (ARF): There were 87 new cases of ARF in 2002 giving a rate of 2.3 per 100 000. This incidence is very high for a developed country and is failing to decline appreciably. Most cases were in Maori and Pacific peoples aged 5 to 14 years. These individuals are at risk of developing chronic rheumatic heart disease. Among under-15-year-olds, incidence rates of ARF increased markedly with increasing deprivation (as measured on the NZDep 2001 scale). The number of recurrences of ARF increased from three in 2001 to six in 2002.

Meningococcal disease: The meningococcal epidemic, which began in mid-1991, continued into 2002. A total of 557 cases (including 18 deaths) was identified, giving a rate of 14.9 per 100 000 population. In comparison, 650 cases and 26 deaths were reported during 2001. As in previous years, rates in 2002 were highest in the under-five-years age group, and among Maori and Pacific peoples. For children the risk of disease was strongly associated with increasing deprivation (as measured on the NZDep 2001 scale). Measures to reduce this epidemic include the proposed vaccination programme and efforts to reduce levels of household crowding.

Tuberculosis: The number of cases of tuberculosis increased slightly from 373 cases in 2001 to 384 cases in 2002. This trend follows that of the last 14 years since the low of 295 cases in

1988. Cases arose from both local transmission (29%) and imported disease (71%). Among all age groups, rates of disease increased with increasing deprivation (as measured on the NZDep 2001 scale). Control of this disease relies on a range of strategies described in the Ministry of Health's TB control guidelines.

Cellulitis: Hospitalisations for cellulitis increased markedly during the 1990s, and this group of infections now cause over 6000 hospitalisations a year. This rise has coincided with an increase in methicillin-resistant *Staphylococcus aureus* (MRSA) infection in the community.

Enteric infections

Enteric disease notifications increased further in 2002, totalling 18 800 and comprising 85% of all notifications. In contrast, 17 113 (82% of all notifications) enteric notifications were received in 2001 and 14149 (69%) in 2000.

Campylobacteriosis: This remains the most frequently notified disease in New Zealand. The national incidence in 2002 of 12 489 cases (334.2 per 100 000) was the highest on record and is markedly higher than that reported by other developed countries.

Hepatitis A: The incidence of this disease increased significantly in 2002, partially due to a large foodborne outbreak linked to contaminated blueberries. Such common source outbreaks are unusual in New Zealand. Most cases are sporadic and linked to overseas travel or household contact.

Salmonellosis: The incidence of this disease dropped significantly from a peak of 2417 cases in 2001 to 1870 cases in 2002. However, the trend has been for a continuing increase in incidence over the past two decades, and the rate of 50.0 per 100 000 was still high compared to other developed countries. The dominant serotype in New Zealand during 2002 was *Salmonella* Typhimurium phage type 160, which increased as part of a national outbreak that began in 2000 and peaked in 2001. The range of ecological niches and modes of transmission for specific Salmonella organisms supports the need for ongoing surveillance and investigation to inform control measures.

Other enteric diseases: The incidence of VTEC/STEC in 2002 (73 cases) was similar to the peak of 76 cases in 2001, and is still a matter for concern given the severe spectrum of illness associated with this infection and its potential to cause large outbreaks. Cryptosporidiosis, shigellosis and giardiasis notifications all dropped in 2002; whereas yersiniosis increased slightly.

Infections and chemical poisonings from animal contact and the environment

Brucellosis: The first local transmission of brucellosis in New Zealand for at least a decade occurred in 2002. This was a case of *Brucella suis,* probably transmitted from an infected pig.

Legionellosis: There were 51 cases of legionellosis notified in 2002 compared to 46 in 2001. As in previous years, incidence rates were highest in the elderly. Four deaths were reported. Of note was a decline in the proportion of disease caused by *L. longbeachae.*

Leptospirosis: Leptospirosis remains New Zealand's most important directly transmitted zoonotic disease. Notification, laboratory and hospital data all suggest that the incidence is increasing. Over 90% of cases occurred in occupational groups having contact with farm animals, in particular among meat processing workers (49%), and farmers or farm workers (41%). There is some evidence for the emergence of new reservoirs, notably sheep.

Lead absorption: Notifications of lead absorption decreased from 130 cases in 2001 to 90 in 2002. Most cases (87%) were adults aged 15 years or older, and rates were highest among males aged between 50 and 59 years. The most commonly reported risk factors among adults were occupational or hobby-related exposure to lead and/or exposure to lead paint flakes or dust in the home. The most commonly reported risk factor among children was living in a pre-1970s built house in which old paint was flaking or had recently been stripped.

Rickettsial Disease: Six cases of murine typhus (*Ricketsia typhi*) were notified in 2002, compared to five in 2001. While only demonstrated with certainty in the Auckland region, it is likely that *R typhi* is also present in other regions of New Zealand. Prevention is directed mainly at control of potential flea hosts, such as rats.

Blood, tissue-borne and sexually-transmitted infections

HIV /AIDS: In 2002, a total of 17 notified cases of AIDS, including five deaths, were reported to the AIDS Epidemiology Group. In comparison, 26 cases and 12 deaths were reported in 2001. The number of cases diagnosed with HIV infection in New Zealand has been steadily rising since 1999. In 2002 a total of 136 cases were diagnosed with HIV infection, of whom 107 were diagnosed through Western blot antibody testing and 29 through viral load testing. The predominant risk behaviour category for HIV infection remains homosexual contact, although in 2002, heterosexual contact was responsible for an increasing proportion (65%) of new AIDS notifications.

Sexually transmitted infections: In 2002, the 27 sexual health clinics reported 9298 confirmed STI cases. Genital warts was the most commonly diagnosed STI (3510 cases), followed by chlamydia (3372), NSU in males (1124), genital herpes (713), gonorrhoea (532) and syphilis (47). The incidence of the bacterial STIs, chlamydia, and gonorrhoea remains high in New Zealand, and shows little sign of abating. Syphilis also increased in 2002.

Blood borne and tissue-borne infections: There were 52 cases of Hepatitis C notified in 2002 compared to 59 the previous year. The principal risk factor was a history of injecting drug use, reported by 77% of cases. The data greatly underestimate the true incidence of HCV infection, as most new infections are asymptomatic. Two definite cases and one probable case of Creutzfeldt-Jakob disease (CJD) were notified in 2002. No cases had any features suggestive of new variant CJD.

Antibiotic resistance and hospital acquired infections

Antimicrobial resistance: Most antimicrobial resistance data are only available in a complete analysed form up to the end of 2001. Ongoing laboratory-based surveillance has identified some important trends in the prevalence of resistance among common, important, clinical pathogens: notably, an increasing prevalence of MRSA generally - an increasing proportion of which is multiresistant; a decrease in penicillin non-susceptibility, and a decrease in non-susceptibility to third-generation cephalosporins among *Streptococcus pneumoniae* from cases of invasive disease in 2001; and an increasing prevalence of ciprofloxacin resistance in *Neisseria gonorrhoeae*, with a four-fold increase in resistance in 2001. A more reassuring finding is that vancomycin-resistant enterococci (VRE) have not become established in New Zealand hospitals and multidrug-resistant tuberculosis (MDR-TB) isolates remain uncommon.

*Hospital acquired infections***:** This group of infections are not under national surveillance. Cross sectional studies in New Zealand suggest that these infections may affect at least 5% of people hospitalised in this country.

New, exotic and imported infections

Travel-associated disease: Diseases associated with overseas travel or migration included those that are exotic to New Zealand such as cholera, dengue fever, leprosy, malaria and Ross River virus infection. In 2002 overseas travel was also a major risk factor for paratyphoid (70% of cases had this recorded as a risk factor), typhoid (67%), shigellosis (49%) and hepatitis A (35%).

Vector borne diseases: The incidence of imported dengue fever remains high, although the incidence of 70 cases in 2002 was a significant drop from the peak of 93 notified cases in 2001. The rates of infection were highest among travellers to the Cook Islands with a rate of 158.5 per 100 000 visits. The incidence of malaria increased slightly to 60 cases in 2002. Many cases (15) were military personnel who had been deployed overseas. One case of Ross River Infection was notified, compared to three the year before. The continuing arrival of infected and viraemic people raises the possibility that these arboviral diseases could become established here.

Outbreaks

There were 333 reported outbreaks in 2002, involving 2870 cases. In comparison, 399 outbreaks and 2323 associated cases were reported in 2001. Enteric pathogens were identified or suspected in 317 (95.2%) of the 2002 outbreaks. The most commonly implicated pathogen or toxin was Norovirus (76 outbreaks, 22.8%), followed by *Campylobacter* (51 outbreaks, 15.3%) and *Salmonella* (35, 10.5%). Common source outbreaks, especially those resulting from a common event, were the most frequently reported outbreaks. The number and magnitude of institutional outbreaks increased significantly in 2002: outbreaks in rest homes accounted for nearly one fifth of all outbreak-associated cases.

Impact and incidence of infectious disease

Fatalities: A total of 43 deaths from notifiable diseases was reported in 2002, compared to 52 deaths in 2001. Meningococcal disease accounted for the greatest number of fatalities (18 deaths), followed by tuberculosis (6) and AIDS (5). Case fatality rates were highest among CJD (100%), Hib (33%), listeriosis (16%) and legionellosis (6%) notifications.

Hospitalisations: Hospitalisation status was recorded for 69.5% of the 22 000 cases notified on EpiSurv during 2002. Of these, 12.8% were hospitalised, a slight decrease from the case hospitalisation rate of 13.9% in 2001. In 2002 all cases (for whom hospitalisation status was recorded) of listeriosis, leprosy, Hib and tetanus were hospitalised. Case hospitalisation rates of over 50% were also recorded for meningococcal disease (97%), acute rheumatic fever (94%), typhoid (77%), malaria (65%), legionellosis (63%), tuberculosis disease (55%) and leptospirosis (53%) notifications. Diseases accounting for large numbers of hospitalisations in 2002 included meningococcal disease (531 hospitalisations), campylobacteriosis (515), salmonellosis (206), tuberculosis (193), and pertussis (98). Hospital discharge data frequently indicated more hospitalisations due to a given disease than were notified.

Ethnic disparities: Most of the serious infectious diseases described in this report have higher rates in Maori and Pacific peoples than in Europeans. This is particularly evident for meningococcal disease, tuberculosis, rheumatic fever and hepatitis B. All are associated with household crowding. Enteric diseases and pertussis appear to have higher rates in the European population, based on notification data. However, hospitalisation data suggest the population rates may actually be similar or higher in Maori and Pacific peoples. Pacific peoples also have relatively higher rates of shigellosis, typhoid, hepatitis A and dengue fever, which is probably related to travel to Pacific Islands where these diseases are relatively common. Diseases that showed significantly higher rates among the 'Other' ethnic group compared to all other ethnic groups were tuberculosis and malaria; and diseases that showed significantly higher rates among the European and 'Other' ethnic groups were giardiasis and yersiniosis. Again, these rates may be related to higher levels of migration and travel to or from affected countries.

Age distribution: The diseases most common among young children were the enteric diseases, vaccine-preventable diseases, and meningococcal disease, while rheumatic fever was most common among children and young adults. Hepatitis B and C, tuberculosis, leptospirosis, lead absorption, malaria and STIs were most common among adults, while legionellosis and listeriosis were most common among older adults and the elderly. The age-related distribution of these diseases was similar to the pattern seen in 2001.

Conclusions and implications

These surveillance data highlight many of the same infectious disease problems that were identified in previous surveillance reports, namely:

- New Zealand's ongoing meningococcal disease epidemic
- The increasing burden of enteric disease
- Recurrent pertussis epidemics
- Elevated and increasing rates of the bacterial STIs
- Significant incidence of imported disease, notably dengue fever and tuberculosis

New findings in 2002 were the apparent re-emergence of leptospirosis that had previously declined markedly following successful control programmes in the 1970s, and the potential for hepatitis A to cause large dispersed common source outbreaks.

These data also show that the burden of serious infectious diseases is not spread evenly across the population, with higher rates among Maori and Pacific populations, and those living in more deprived neighbourhoods.

Surveillance data also reinforce some successes in controlling infectious diseases in New Zealand, notably the continuing decline in the incidence of Hib and Hepatitis B following successful immunisation programmes. A notable achievement is that 2002 is the first year without any Hib cases in children.

This report attempts, for the first time, to use hospitalisation data for infectious diseases in a systematic way to supplement surveillance data from other sources. This approach also allows some key quality dimensions of the notifiable diseases surveillance system to be assessed.

Strengths of New Zealand's infectious disease surveillance system include the increasing ability to integrate data from multiple sources for increased sensitivity of surveillance, and also the completeness of data for some diseases. This strength is best illustrated by meningococcal disease surveillance.

Major surveillance gaps are apparent, notably:

- The absence of a national surveillance system for hospital acquired infections (HAIs)
- The reliance on clinic-based reporting for surveillance of STIs
- Low sensitivity of the surveillance system for chronic infections such as hepatitis C
- Poor representativeness of some notification data for some characteristics of the population under surveillance e.g. ethnicity
- The lack of effective surveillance linkage of environmental exposures, hazards and health outcomes.

A number of improvements to surveillance were undertaken in 2002. A major review of the schedule of notifiable diseases is underway and this, together with the change to the Health Act, is likely to have a considerable effect on the future direction of infectious diseases surveillance in New Zealand.

Introduction

The aim of this report is to produce a summary of the distribution of communicable diseases of public health importance in New Zealand. While most of these diseases are currently notifiable under the Health Act 1956 or the Tuberculosis Act 1948, the scope also encompasses some communicable diseases that are under surveillance through other systems. Surveillance data for non-infectious yet notifiable conditions such as lead absorption are also reported.

This report focuses on the distribution of these diseases in 2002 and longer-term incidence. It aims to disseminate information to those who need it to support prevention and control measures.

The organisation of this report has been extensively updated from previous years with the diseases under surveillance been grouped into categories that reflect modes of transmission and control measures. These categories are similar to those used in the Ministry of Health's Integrated Approach to Infectious Diseases.[1] This report also draws on a wider range of data sources than previously, including NZHIS hospitalisation data, to provide a more comprehensive description of infectious disease epidemiology in New Zealand.

Purposes of surveillance

Surveillance is the ongoing systematic collection, analysis and interpretation of health data essential to the planning, implementation and evaluation of public health practice, closely integrated with the timely dissemination of these data to those who need to know. The final link in the surveillance chain is the application of these data to prevention and control.[1]

The main reasons for disease surveillance are as follows:[2]

- to identify cases of disease that require immediate public health control measures
- to monitor disease incidence and distribution, and alert health workers to changes of disease activity in their area
- to identify outbreaks and support their effective management
- to assess disease impact and help set priorities for prevention and control activities
- to identify risk factors for diseases to support their effective management
- to evaluate prevention and control activities
- to identify and predict emerging hazards
- to monitor changes in disease agents through laboratory testing
- to generate and evaluate hypotheses about disease occurrence
- to fulfil statutory and international reporting requirements.

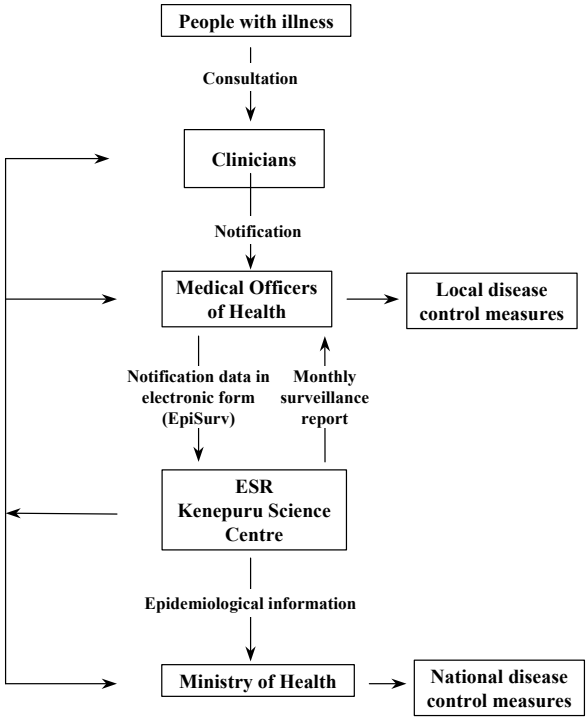
Surveillance Methods

Surveillance systems:

The key sources of data that have been used in compiling this report are as follows:

EpiSurv -the national notifiable disease surveillance system
Under the Health Act 1956 and the Tuberculosis Act 1948, health professionals are required to inform their local Medical Officer of Health of any notifiable disease that they suspect or diagnose. These notifications provide the basis for surveillance and hence control of these diseases in New Zealand. Notification data are recorded on a computerised database (EpiSurv) installed in each of 20 public health services (PHS). Each week, these data are sent to the Institute of Environmental Science and Research (ESR) Ltd where they are collated and analysed on behalf of the Ministry of Health. The data collected on each disease depends on the disease in question but usually includes demographic, outcome, basis of diagnosis, risk factor and some management information. Some of the diseases included only became notifiable with the revised schedule of notifiable diseases which came into effect on 1 June 1996, e.g. measles, yersiniosis. [2] This report includes sections on all of the diseases that are notifiable in New Zealand except those where this country has never had a notified case (yellow fever, viral haemorrhagic fever, hepatitis E, Q fever) or not had any cases for many decades (anthrax, plague).[3]

The communicable disease surveillance system –major components and information flow



Laboratory-based surveillance
Laboratory based surveillance is the collection of laboratory data for public health purposes. Several of the communicable diseases diagnosed by clinical laboratories are either not covered adequately or not covered at all by the notifiable disease surveillance systems. Also, laboratory-based surveillance sometimes takes place to enhance surveillance data gathered by other methods. Examples of organisms covered by laboratory-based surveillance are antimicrobial resistant organisms, legionellae, leptospira, meningococci, respiratory syncytial virus (RSV), enteroviruses, adenoviruses, salmonellae, and streptococci.

Surveillance of HIV & AIDS in New Zealand
Since 1989, the AIDS Epidemiology Group in Dunedin has been contracted to collect information about people diagnosed with AIDS through notification to the Medical Officers of Health. Detailed information has also been collected about people infected with HIV since 1996 through a laboratory-based surveillance system involving the two laboratories that perform confirmatory HIV antibody testing using the Western blot method (ESR and the Virus Laboratory, Auckland Hospital).[4] For each confirmed diagnosis, either the laboratory or the AIDS Epidemiology Group send a letter to the doctor who requested the test seeking information on the likely mode of infection and other demographic data. Coding ensures that the identity of the patient is known only to the reporting doctor, but is sufficiently specific to allow detection of duplicate reports.

Sexually Transmitted Infection (STI) sentinel surveillance system.
Except for AIDS, no STIs are notifiable in New Zealand. Surveillance is primarily based on reporting by sexual health clinics.[5] ESR took over the national operation of the STI sentinel surveillance system in 1995. Sexual health clinics report basic demographic data on cases of chlamydia, gonorrhoea, genital herpes, genital warts, syphilis, and non-specific urethritis (NSU) in males. STI surveillance has progressively been expanded since 1998 to include data from family planning clinics, student and youth health services, and laboratories. Laboratory-based surveillance for chlamydia and gonorrhoea is now operating in the Waikato-Bay of Plenty area, and Auckland.[6]

Influenza sentinel surveillance system
A sentinel surveillance system has been developed which gathers data on the incidence and distribution of influenza.[7] National influenza surveillance is undertaken from May to September each year. This surveillance is based on a network of general practices recruited from all health districts in New Zealand. About 88 practices participate. The number chosen is proportional to the size of the population in each health district covered by the Public Health Service. General practitioners are asked to record the number of consultations for influenza-like illness (defined by a standardised case definition) each week and the age group of each of these suspected cases. Each practice is also requested to collect swabs from up to three patients per week. The swabs are sent to hospital and ESR virus laboratories for viral isolation and strain identification.

New Zealand Paediatric Surveillance Unit (NZPSU)
NZPSU was established in late 1997 to provide active surveillance of acute flaccid paralysis (AFP) to fulfil World Health Organisation requirements for certification of polio eradication. In January 1998, the conditions under surveillance were expanded to include haemolytic uraemic syndrome (HUS), congenital rubella syndrome (CRS), perinatal exposure to HIV, vitamin K deficiency, bleeding, and neonatal herpes simplex infection. Every month, participating

paediatricians and other specialists in paediatric practice send a reply-paid card to the NZPSU on which they indicate whether in the previous month they have seen any cases of the conditions under surveillance. These data are then collated and analysed by the NZPSU. [8] Information from the NZPSU is used in this report to enhance notification data on VTEC/STEC infection (HUS data) and rubella (CRS data).

Outbreak surveillance
ESR introduced an outbreak surveillance system in July 1996 and has been improving this system in a series of planned steps since then. [9] The surveillance system has operated electronically since mid 1997 as an additional module of EpiSurv. In mid 2000, EpiSurv and ESR laboratory reported outbreaks were matched for the first time. Unlike the other surveillance systems described above, this system collects data on disease outbreaks, rather than individual cases.

Hospital-based surveillance
The New Zealand Health Information Service (NZHIS) in the Ministry of Health collates national data on patients discharged from publicly funded Hospitals. These data are stored as part of the National Minimum Dataset (NMDS). Cases are assigned disease codes using the tenth revision of the *International Classification of Diseases* (ICD10) coding system (this replaced the ninth revision, ICD9, during 1999, and data may be supplied using both sets of codes). Up to 99 diagnostic, procedure, and accident codes may be assigned to each admission. The first of these is the principal or primary diagnosis, which is the condition that led to admission. This may be different from the underlying diagnosis that caused the admission. A further change in disease coding that affects the interpretation of historic data was the introduction of the CM version of ICD.9 in 1995. This update included specific codes for emerging 'new' diseases, for example campylobacteriosis and HUS. Hospitalisation data from before this date tend to under-count these diseases so are not presented here.

Hospital discharge data for selected infectious diseases was extracted from NZHIS databases, encrypted and sent to ESR for analysis and comparison with data from other surveillance systems. This data source was largely used to supplement information obtained from the above more specialised surveillance systems. For a few conditions, notably cellulitis, it is the only data source used in this report

Analysis methods:

Key methods used to analyse surveillance data from the above mentioned sources are described below. The majority of data extraction and analysis was performed using SAS version 8.2.

Denominator data

Census: In this report all disease incidence rates for 2002 and 2001 have been calculated using 'usually resident' population data from the 2001 Census, supplied by Statistics New Zealand.

Standard population: The New Zealand population at the 2001 Census has been used as the standard population upon which age-standardised rates (see below) are based.

Ethnicity: Unless otherwise specified, denominators for different ethnic groups are based on a hierarchical (or 'prioritised') classification of ethnicity, with the Maori ethnic group at the top of the hierarchy, followed by Pacific peoples, 'Other' and European ethnic groups. With this approach, the 'Maori' denominator represents the number of individuals identifying with the Maori ethnic group and any additional ethnic group, whereas the 'Pacific peoples' denominator counts the number of individuals identifying with the Pacific peoples ethnic group and any additional ethnic groups with the exception of the Maori ethnic group. In contrast, the 'total response' approach to ethnic denominators simply counts the total number of individuals identifying with any given ethnic group.

Travel: For calculating rates of disease among returning travellers, data on New Zealand residents travelling overseas for less than 12 months in 2002 were used for the denominator. These data were based on the country recorded by travellers as the final destination on their departure cards (provided by Statistics New Zealand). Countries visited by travellers en route to or from the final destination are not included on the departure card. Therefore, the number of travellers to any one country may be an underestimate of the true number of visits and rates of disease in travellers must be interpreted with caution.

Occupation: For calculating rates of disease in certain occupational classes, 2001 Census counts of the employed population aged 15 years and over have been used.

Deprivation: Denominator data for the calculation of rates based on the NZDep2001 index of deprivation was provided by Clare Salmond (Department of Public Health, Wellington School of Medicine and Health Sciences). This index, measuring relative socioeconomic deprivation, is derived from a weighted combination of nine variables (from the 2001 Census), each reflecting a different aspect of material and social deprivation. The deprivation score, which ranges from 1 (least deprived) to 10 (most deprived), is calculated for each meshblock[a] in New Zealand. Roughly equal numbers of people reside in areas associated with each of the ten deprivation levels.

Urban/rural: For the comparison of rates of disease in urban versus rural areas, classification of small area units as either urban or rural was based on a method established by the Public Health Intelligence Group of the Ministry of Health using 2001 Census data.

Numerator data

Ethnicity: Ethnicity data recorded on EpiSurv was cleaned prior to analysis to prevent the overestimate of rates of disease in the 'Other' ethnic group. For example, cases recorded as having 'Other' ethnicity, but who specified their ethnicity as 'Samoan' or 'Tongan' were reclassified as belonging to the 'Pacific peoples' ethnic group. Unless otherwise stated, the hierarchical approach to classification of ethnicity has been used.

Geocodes: The geocoding of the addresses of all notifications in 2002, and the majority of 2001 notifications, was attempted. To optimise the success rate, manual cleaning and geocoding of a given address was undertaken when the batch geocoder failed to resolve the address.

[a] Meshblocks are geographic units defined by Statistics New Zealand, each comprising approximately 90 people.

Health regions: Assignment of cases to District Health Boards (DHBs) was based on the derived geo-coordinates. In situations where geo-coordinates could not be determined, the Territorial Authority Area recorded on EpiSurv was used where possible to make the assignment. Due to the different methods employed, rates for DHBs and corresponding health districts may differ slightly even when the corresponding boundaries coincide.

Hospitalisations: Unless otherwise specified, primary diagnosis codes based on the ICD9 coding system have been used to analyse hospital discharge data. Although inclusion of a greater number of diagnosis codes in the analysis may give a more realistic estimate of hospitalisation rates, primary diagnosis codes are deemed most appropriate in the majority of situations. Categorisation by year is based on date of admission to hospital. Multiple admissions with the same primary diagnosis code in a given year have been excluded from the count. Wherever possible, hospitalisation data has been graphed from 1988 onwards. However, ICD9 codes for some newly emerging diseases were not defined until later, in which case the data has been graphed from the year of introduction of the relevant code(s).

Deprivation: A NZDep2001 index of deprivation was associated with all notified cases living in private dwellings whose addresses geocoded to at least 'Street' level, based on the assignment of geo-coordinates to meshblocks. [10]

Urban/rural: Notified cases whose addresses geocoded to at least 'Suburb/Locality' level were assigned urban or rural status based on their meshblock assignment. In order to eliminate the bias towards rural localities among non-geocoded addresses, cases whose addresses did not geocode at all or to the required level were assigned rural status if their addresses contained rural delivery codes.

Calculation of rates and confidence intervals

Age-standardised rates have been calculated via the 'direct' method, using the entire New Zealand population as the standard. An age-standardised rate calculated this way represents the rate that would occur if the observed age-specific rates were present in a population with an age distribution equal to that of the standard population. Although age-standardisation is critical for the comparison of rates of disease among different ethnic groups, it generally has little impact on the relative magnitude of incidence rates by geographical region. The reader may refer, as necessary, to age-standardised rates by District Health Board in the appendix of this report.

The reader is urged to use caution when interpreting rates printed in this report which are based on fewer than five cases, as these rates are likely to be unstable and imprecise. Care should also be exercised when interpreting and comparing rates based on fewer than 20 cases, as these will usually have very wide confidence intervals. Age and sex data that has been presented graphically has generally been grouped into broader age bands, where necessary, to avoid erroneous conclusions being drawn from unstable rates due to small numerators. Every effort has been made however not to mask statistically significant trends.

Throughout the report, confidence intervals have been used to assess the precision of the point estimates of crude, age-specific and age-standardised rates or proportions. Generally speaking, confidence intervals describe how different a point estimate could have been if the underlying conditions stayed the same, but chance had led to a different set of data. In this

report all confidence intervals have been calculated at the 95% probability level, i.e. there is a 95% chance that the confidence interval covers the true value. It should be emphasised that confidence intervals do not account for several other sources of uncertainty in point estimates, including missing, inaccurate or incomplete data, or bias resulting from different data collection or reporting procedures among regions.

Although the comparison of confidence intervals for two different point estimates is not strictly equivalent to a statistical test of significance, it is used throughout this report as an approximation to one. That is, two rates or proportions are stated to be 'significantly different' if their corresponding confidence intervals do not overlap. The error is conservative, in the sense that occasionally an appropriate statistical test might indicate a statistically significant difference even though the confidence intervals *do* overlap; however, if two confidence intervals *do not* overlap, a comparable statistical test will *always* indicate a statistically significant difference.

Crude and age-specific rates are assumed to follow the Poisson distribution. Confidence intervals have been calculated directly from the Poisson distribution when the number of observed cases is less than 100. When the number of cases is 100 or more, the normal approximation has been used. Confidence intervals for age-adjusted rates have been calculated using a method based on the gamma distribution.[11] Confidence intervals for binomial proportions have been calculated via the score interval method.[12]

Dates

Notification data contained in this report are based on information recorded on EpiSurv as at 4th February 2003. Not all changes made to EpiSurv data by PHS staff after this date will be reflected in this report. Consequently, future analyses of these data may produce revised results. With the exception of meningococcal disease (which is reported according to the earliest date available among onset, hospitalisation, laboratory and notification dates), disease numbers are reported according to the date of notification. On the other hand, laboratory results are reported according to the date the specimen was received, and hospitalisation figures are reported according to the date of admission to hospital.

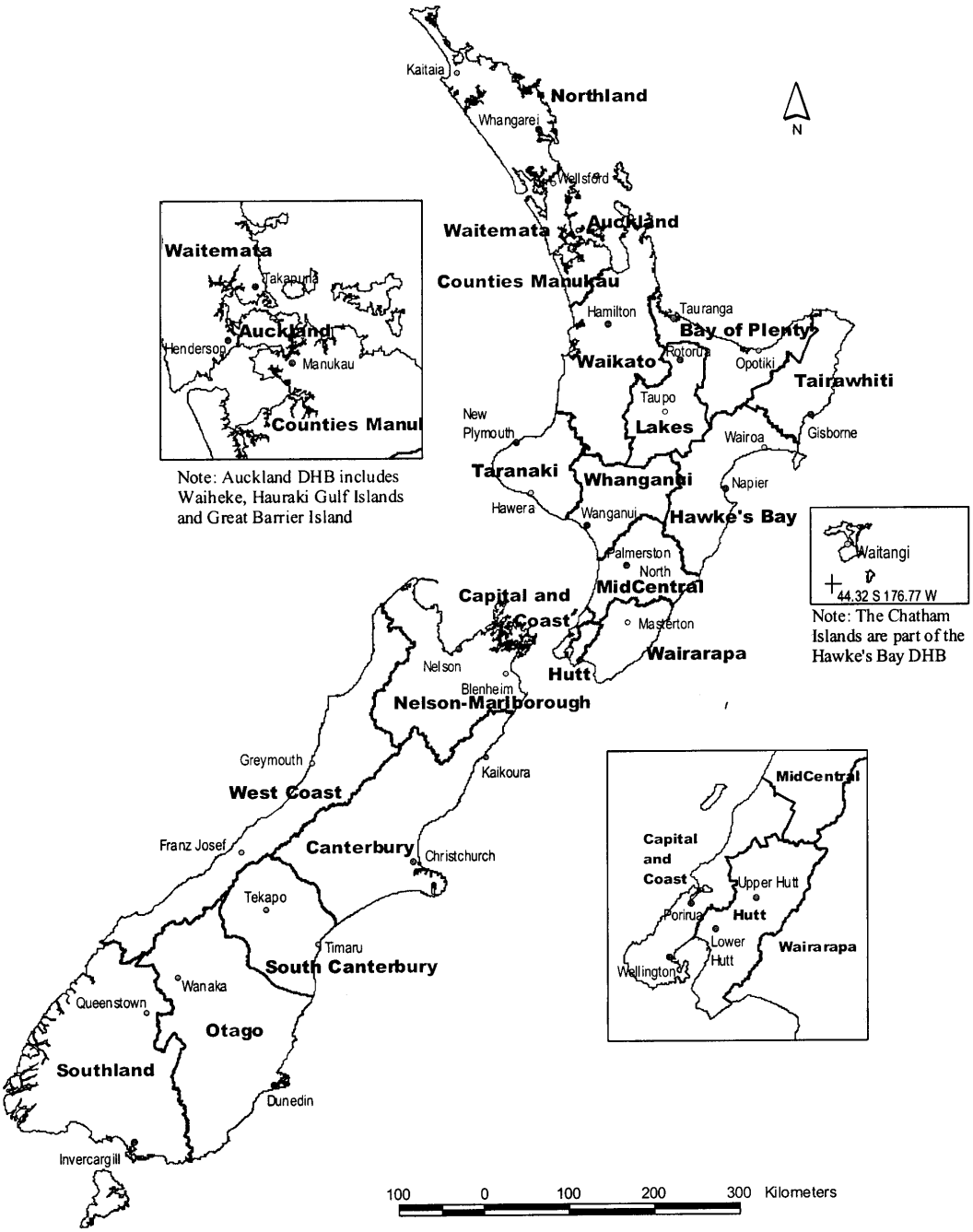
Geographical breakdown

This report provides rates both for Health Districts used in last year's report and for current District Health Boards (DHBs). The 21 DHBs were created through the New Zealand Public Health and Disability Act 2000 on 1 January 2001. In contrast to the former 24 Health Districts, not all DHBs follow Territorial Authority (TLA) boundaries. Six DHBs are split across TLA boundaries. In the following table, Health Districts are listed alongside DHBs to which they most closely correspond. In those instances where DHB and corresponding Health District boundaries do not coincide, a brief description of the difference is provided.

A comparison of District Health Boards and Health Districts

District Health Board (DHB)	Corresponding Health District(s)	Difference in boundaries
Northland	Northland (NL)	None
Waitemata	North West Auckland (NW)	None
Auckland	Central Auckland (CA)	None
Counties Manukau	South Auckland (SA)	None
Waikato	Waikato (WK)	Waikato DHB includes part of Ruapehu District Territorial Authority, whereas Waikato Health District does not.
Bay of Plenty	Tauranga (TG) and Eastern Bay of Plenty (BE) health districts combined	None
Lakes	Rotorua (RO) and Taupo (TP) health districts combined	None
Tairawhiti	Gisborne (GS)	None
Taranaki	Taranaki (TK)	None
Wanganui	Wanganui (WG)	Wanganui DHB includes part of Ruapehu District Territorial Authority, whereas Wanganui Health District does not.
MidCentral	Manawatu (MW)	MidCentral DHB contains part of Kapiti Coast District Territorial Authority, whereas Manawatu Health District does not.
Hawke's Bay	Hawke's Bay (HB)	None
Wairarapa	Wairarapa (WR)	None
Hutt	Hutt (HU)	None
Capital and Coast	Wellington (WN)	Capital and Coast DHB excludes part of Kapiti Coast District Territorial Authority, whereas Wellington Health District includes the entire TA.
Nelson-Marlborough	Nelson-Marlborough (NM)	None
West Coast	West Coast (WC)	None
Canterbury	Canterbury (CB)	Canterbury DHB includes Ashburton District Territorial Authority, whereas Canterbury Health District does not.
South Canterbury	South Canterbury (SC)	South Canterbury DHB excludes Ashburton District Territorial Authority, whereas South Canterbury Health District includes the TA.
Otago	Otago (OT)	Otago DHB includes part of Queenstown-Lakes District Territorial Authority, whereas Otago Health District does not.
Southland	Southland (SO)	Southland DHB excludes part of Queenstown-Lakes District Territorial Authority, whereas Southland Health District includes the entire TA.

New Zealand District Health Boards



Quality of Surveillance Data

In using information contained in this report it is important to consider the quality of the information. Surveillance systems have a number of quality attributes,[13] the most important of which are probably the following:

- *Sensitivity* – The proportion of disease cases that are detected by the surveillance system
- *Positive predictive value* – The proportion of reported cases that actually have the disease under surveillance. This proportion is in turn affected by the specificity of the case definition and incidence or prevalence of the disease under surveillance.
- *Representativeness* – How well the characteristics of cases reported by the surveillance system represent the true characteristics of cases of that disease
- *Accuracy and completeness of associated data* – Whether important demographic, outcome and risk factor information is reported, is accurate, and is analysable (e.g. may be linked to appropriate population denominator data)

Other surveillance system attributes, such as timeliness, are important for effective surveillance, but are less relevant to an annual analysis of the type presented here.

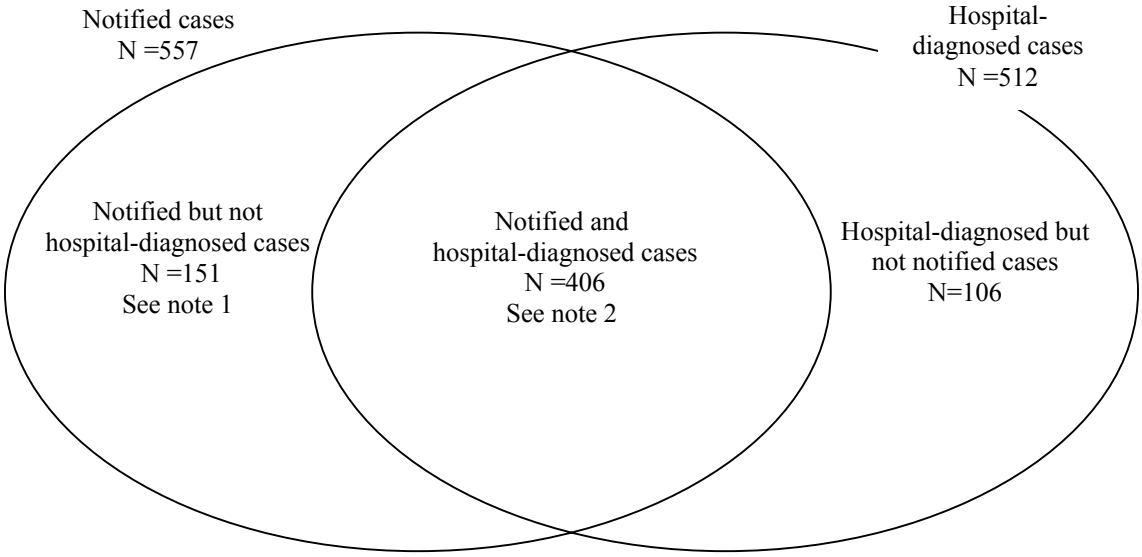
Sensitivity

An analysis of the sensitivity of the notifiable disease surveillance system (EpiSurv) was carried out for meningococcal disease. An attempt was made to match all notified cases with cases recorded in hospitalisation discharge data. The methods and results are briefly described below.

The New Zealand Hospitalisation Information Service (NZHIS) was able to determine NHI numbers for almost all (552/557 or 99.1%) notified cases. NZHIS then matched these notifications with hospitalised cases based on NHI numbers. Of the 557 notifications, 525 (94%) could be matched to hospital admissions during 2002. Only 406[a] notified cases matched with hospital admissions for which "meningococcal disease" (ICD9 code 036) was recorded in any one of the first nine diagnosis fields, and 387[b] notified cases matched with hospital admissions for which meningococcal disease was recorded in the primary diagnosis field. A total of 107 notified cases, recorded as hospitalised on EpiSurv, matched only to hospital admissions which did not have meningococcal disease recorded in any diagnosis field. Of these 107 cases, 59% were laboratory confirmed. In contrast, a total of 512[c] patients admitted to hospital during 2002 had meningococcal disease recorded in at least one of the nine diagnosis fields, and 480 patients had it recorded in the primary diagnosis field. The following diagram illustrates the results of matching notification and hospitalisation data for meningococcal disease cases in 2002.

[a] A further 8 probable matches were obtained, based on matching of sex, date of birth, ethnicity and region.
[b] A further 4 probable matches were obtained, based on matching of sex, date of birth, ethnicity and region.
[c] Repeat admissions have been excluded from the total.

Venn diagram illustrating the results of matching notification and hospitalisation data for meningococcal disease cases in 2002



Note 1: Notified but not hospital-diagnosed (N=151) included:

a) 12 cases who were not hospitalised according to EpiSurv (including 2 cases who died before hospital admission).
b) A further 24 notified cases who could not be matched to hospitalisations (including 5 cases who could not be assigned an NHI number and 19 with no hospital admissions recorded in 2002)
c) 115 cases who could be matched to 2002 hospital admissions, but who did not have meningococcal disease included as a diagnosis. Of these, 63 (59%) cases were laboratory confirmed.

Note 2: Notified and hospitalised cases (N=406) included 311 cases (76.6%) that were recorded as laboratory confirmed.

These data imply the following performance characteristics for the meningococcal disease surveillance system: Sensitivity = (557 – 12 –24) / (557 –12 – 24 + 106) = 83%

This estimate assumes that all additional 106 hospital-diagnosed cases are true cases, and that none of the unmatched notifications (24) were included in the 106 unmatched hospital-diagnosed cases. More realistic assumptions are the following:

a) Approximately 19 (80%) of the 24 unmatched notifications were actually admitted to hospital and should be included in the 106 unmatched hospital-diagnosed cases (lowering our estimate of the number of hospitalised but non-notified cases from 106 to 87).
b) Approximately 70 (80%) of these remaining 87 non-notified hospital-diagnosed cases are true cases of meningococcal disease.

This estimate also assumes that all notified cases are true cases. Again, it would be appropriate to assume that some are not. Given that 74.1% of notified cases (413/557) are

laboratory confirmed, the proportion of true cases is likely to be high. A reasonable estimate would be 90 %, or 501 in 2002.

Using these adjustments, the sensitivity of the meningococcal disease notification system becomes: Sensitivity = (501 – 12 – 5) / (501 –12 – 5 + 70) = 87%

This estimate remains highly qualified. It is very sensitive to assumptions about the hospitalised but non-notified cases of meningococcal disease. It is likely that many of these cases did not have meningococcal disease but had this disease recorded in the differential diagnosis field and subsequently coded as one of their diagnoses. Consequently, this figure may represent a lower bound estimate of system sensitivity.

The sensitivity of the notifiable disease surveillance systems has not been calculated for other diseases. In general, it is likely to be highest for diseases that are more serious and for those that require an active public health response to each case, for example tuberculosis. For less serious infections, such as salmonellosis, notification data will inevitably under-estimate population incidence. ESR laboratory data show that 92.6% (1870/2019) of laboratory confirmed cases of salmonellosis are notified, implying that under-ascertainment is not generally caused by under-notification. The main source of under-ascertainment is likely due to the fact that cases of salmonella infection do not always present to a medical practitioner in the first place. Population based studies carried out overseas suggest the ratio of cases in the community to cases reaching national surveillance may be around 3.1 to 1 for salmonella and 7.6 to 1 for campylobacter.[14]

Positive predictive value

The PPV has not been calculated for any diseases under surveillance. Because the diagnosis and notification of many infectious diseases depends on a positive laboratory test result, the PPV for most is high. This is particularly true for the enteric diseases, where almost all cases are diagnosed and notified after a positive test result. At the other extreme are diseases for which a high proportion of cases is notified based on clinical criteria without subsequent laboratory confirmation. A good example is measles: During periods when measles is not epidemic in New Zealand this disease is rare, whereas other morbilliform rashes in children remain relatively common. Consequently, during inter-epidemic periods, the total number of notified cases may over-estimate the true incidence of disease.

The following table shows the number and proportion of notified cases who have been confirmed via different means. It illustrates the high positive predictive value for the enteric diseases campylobacteriosis and salmonellosis, for which respectively 98% and 95% of notified cases were culture-confirmed. In contrast, the majority (88%) of measles cases was notified on clinical grounds only. Approximately 19% of pertussis notifications were based solely on clinical criteria. Confirmation was most frequently via isolation of *Bordetella* pertussis (45%), although other laboratory methods (26%) and epidemiological methods (10%) also played an important role (10%). Laboratory confirmation was the only method of confirmation for meningococcal disease notifications: 41% were culture-confirmed and a high proportion (33%) was confirmed via ‘other’ laboratory methods.

Method of confirmation recorded for selected notifiable diseases, 2002

Disease	Total cases notified	Laboratory confirmation			Epidemiological confirmation[1] No. (%)	Clinical criteria No. (%)
		Total No. (%)	Culture No. (%)	Other method		
Meningococcal disease	557	413 (74.1%)	229 (41.1%)	184 (33.0%)	-	144 (25.9%)
Campylobacteriosis	12489	12257 (98.1%)	12257 (98.1%)	0 (0%)	12 (0.1%)	220 (1.8%)
Salmonellosis	1870	1780 (95.2%)	1780 (95.2%)	0 (0%)	20 (1.1%)	70 (3.7%)
Measles	25	3 (12%)	0 (0%)	3 (12%)	0 (0%)	22 (88%)
Pertussis	1071	760 (71.0%)	483 (45.1%)	277 (25.9%)	109 (10.2%)	202 (18.9%)

[1] Contact with a confirmed case of the disease or part of an identified common source outbreak. Excludes cases who were laboratory-confirmed.

Representativeness

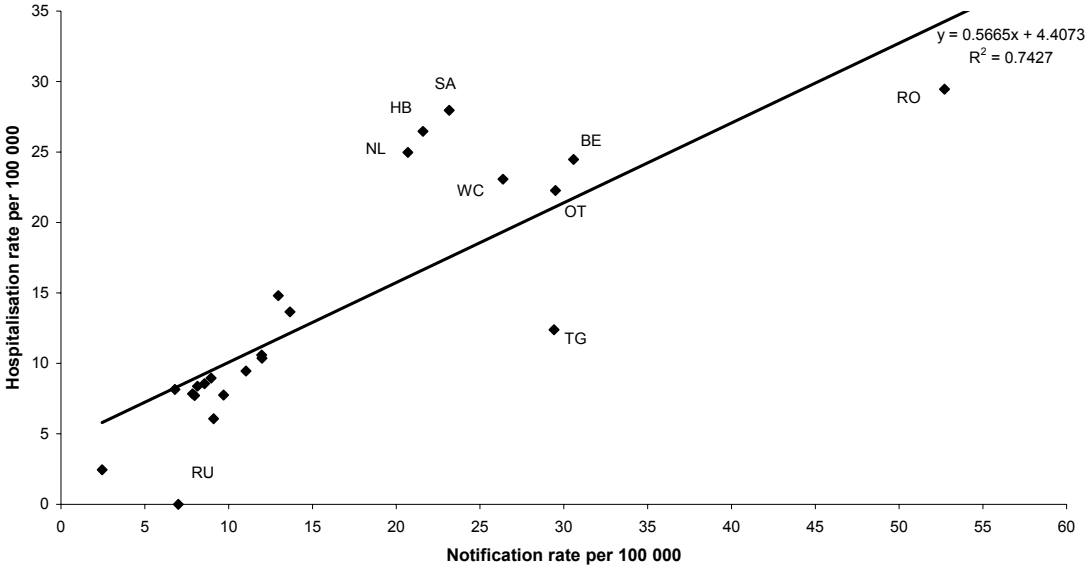
Conclusions about the descriptive epidemiology of the notifiable diseases are based on the assumption that the sensitivity of the system is constant over time, place and person. In other words, that the patterns apparent in the observed data are representative of those actually occurring in the disease under observation. This assumption is clearly not always true. This report includes a brief analysis of the representativeness of two disease characteristics: place (health district) and ethnicity. This analysis is based on a comparison of disease rates suggested by notification rates and hospitalisation rates.

Place (health district): The following graphs illustrate the comparative hospitalisation rates[a] for key diseases, campylobacteriosis, salmonellosis, pertussis and meningococcal disease. A trend line has been added using the method of least-squares[b]. This presentation provides an indication of the extent to which geographic variations in disease incidence among health districts as measured by the notification system are consistent with those detected using hospitalisation data. These graphs show a high correlation for meningococcal disease, a lower correlation for pertussis and salmonellosis, and little correlation for campylobacteriosis.

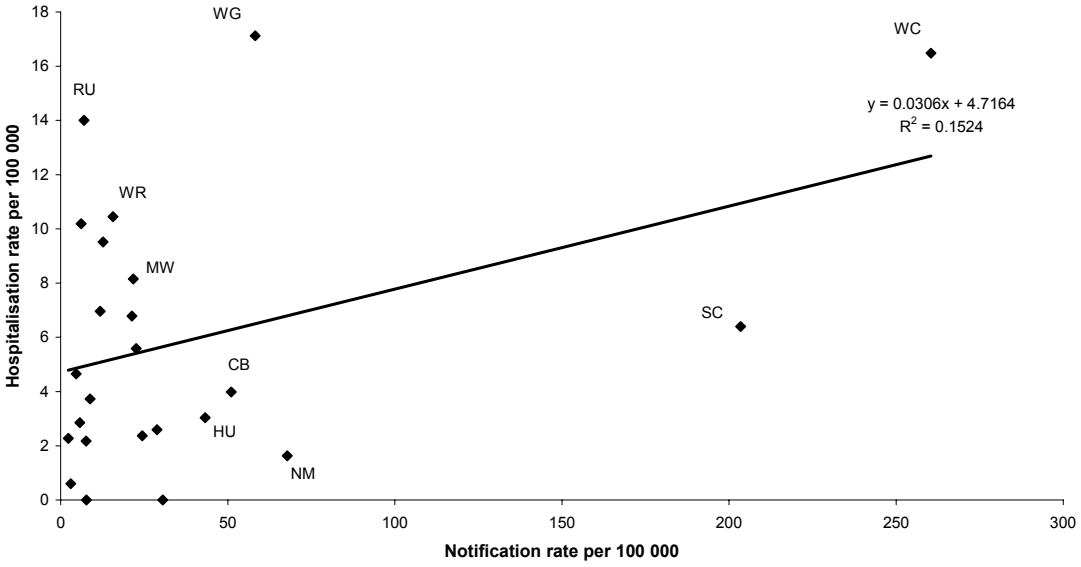
The high sensitivity of meningococcal disease surveillance is largely due to the 100% matching of notified cases and laboratory-reported cases. Salmonellosis is also under laboratory surveillance, and 88% of laboratory-reported cases were also notified in 2002. The high variability in the comparative rates of campylobacteriosis is probably due in part to different reporting practices among Public Health Units (PHUs). South Island PHUs are generally more likely than upper North Island PHUs to individually notify cases which are part of a recognised outbreak - in addition to completing an outbreak report.

[a] Rate of hospitalisation is based on the number of patients with relevant ICD9 code in any diagnosis field
[b] The trend line should be interpreted with caution as it may be unduly influenced by outlying rates with very wide confidence intervals.

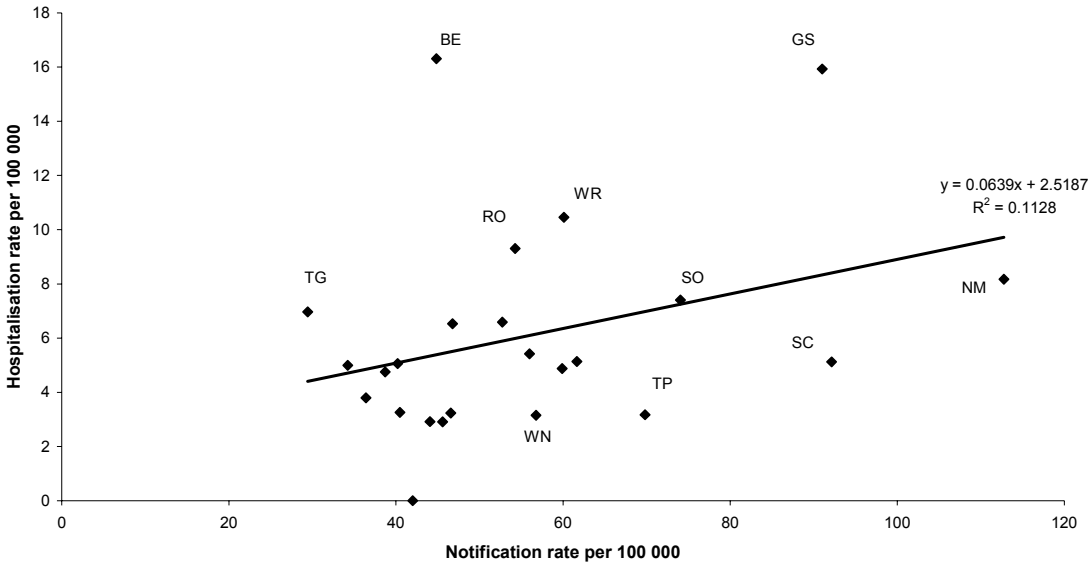
Meningococcal disease notification vs hospitalisation rates for each health district, 2002



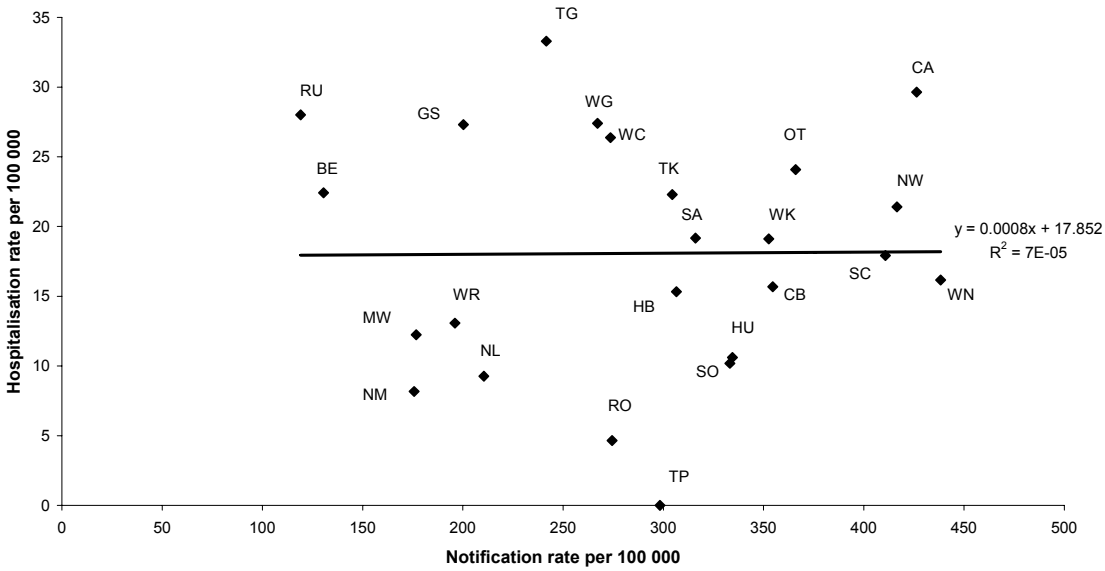
Pertussis notification vs hospitalisation rates for each health district, 2002



Salmonellosis notification vs hospitalisation rates for each health district, 2002



Campylobacteriosis notification vs hospitalisation rates for each health district, 2002



Ethnicity: The following analysis compares ethnic-specific rates and relative risks (relative to the European ethnic group) calculated from notification and hospitalisation data respectively. For all diseases in the table below, the relative risk of hospitalisation for Maori and Pacific peoples was higher than the corresponding relative risk of notification. Notification data appear moderately representative of the ethnic distribution observed in hospitalised cases for meningococcal disease – a serious disease for which a high proportion of cases are hospitalised. However for campylobacteriosis, salmonellosis and pertussis, notification data show a very different pattern to that observed in hospitalisation data. Among the diseases listed below, notification data are least representative of hospitalisation data for pertussis:

Maori and Pacific peoples both had a significantly lower notification rate than Europeans, but nevertheless both experienced a much higher hospitalisation rate than Europeans. Among ethnic groups, notification data was consistently least representative of hospitalisation data for Pacific peoples. For example, Pacific peoples experienced a notification rate one third of the European notification rate for pertussis, yet their hospitalisation rate was over three times the European hospitalisation rate.

Comparison of ethnic-specific rates and relative risks for infectious disease surveillance data derived from two different sources: notifiable diseases surveillance and hospitalisation data

Disease		Notifications			Hospitalisations[2]		
		European[1]	Maori	Pacific[1]	European[1]	Maori	Pacific[1]
Meningococcal	- Rate	10.7	30.4	44.9	8.8	27.9	45.9
	- Relative risk	1.0	2.8	4.2	1.0	3.2	5.2
Campylobacteriosis	- Rate	307.7	76.9	53.9	20.5	12.2	17.5
	- Relative risk	1.0	0.2	0.2	1.0	0.6	0.9
Salmonellosis	- Rate	48.0	27.0	16.0	4.5	6.8	7.0
	- Relative risk	1.0	0.6	0.3	1.0	1.5	1.6
Pertussis	- Rate	30.2	19.0	10.5	3.1	8.5	10.0
	- Relative risk	1.0	0.6	0.3	1.0	2.7	3.2

[1] Ethnicity classification based on 'hierarchical' approach
[2] Rate of hospitalisation is based on number of patients with relevant ICD9 code in *any* diagnosis field

Accuracy and completeness of associated data

Much of the interpretation of surveillance data depends on the analysis of associated data collected with each event. These data include case location and demographic details, basis for diagnosis, outcome, outbreak, risk and protective factors, and case management data. The quality and usefulness of this analysis depends on the completeness and accuracy of such data. Completeness of key fields for selected diseases is presented in the following table. Among the diseases listed, data was most complete for meningococcal disease, followed by pertussis, salmonellosis and campylobacteriosis – reflecting both the relative seriousness of these diseases, and the extent to which ESR staff chase missing data. For example, ethnicity was recorded for nearly 99% of meningococcal disease cases, but for only 72% of campylobacteriosis cases. In general, demographic data relating to the age and location of a case is well recorded for all diseases.

Completeness of EpiSurv fields for selected notifiable diseases, 2002

Disease	Total cases	Age	Ethnicity	Geocoded[1]	Date of onset	Hospitalised	Died	Overseas travel
Meningococcal disease	557	557 (100%)	549 (98.6%)	537 (96.4%)	537 (96.4%)	545 (97.8%)	544 (97.7%)	471 (84.6%)
Campylobacteriosis	12489	12396 (99.3%)	9041 (72.4%)	12061 (96.6%)	6295 (50.4%)	7735 (61.9%)	10938 (87.6%)	4787 (38.3%)
Salmonellosis	1870	1859 (99.4%)	1516 (81.1%)	1824 (97.5%)	1426 (76.3%)	1473 (78.8%)	1719 (91.9%)	1332 (71.2%)
Pertussis	1071	1070 (99.9%)	927 (86.6%)	1048 (97.9%)	875 (81.7%)	964 (90.0%)	995 (92.9%)	875 (81.7%)

[1] geocoded to at least suburb/locality level

Accuracy of fields is much harder to assess. For the 525 meningococcal disease notifications which matched to hospitalisation admissions in 2002, information on ethnicity recorded on

the two systems was compared. Although there were many instances (71 cases or 13.5%) where the two systems classified a case differently (based on the 'prioritised'[a] approach for both), there was no significant difference in the overall proportion of cases assigned to any given ethnic group.

A further issue that arises whenever rates are calculated is the choice of an appropriate denominator population. The issue of numerator-denominator match (or mismatch) is quite apparent when calculating rates by ethnicity. Again using meningococcal disease, information on ethnicity was combined[b] from both systems to calculate ethnic-specific rates, based on the 'total response'[a] approach. Although results based on the prioritised approach for notification data indicate a statistically significant difference in rates between Maori and Pacific peoples ethnic groups, the same conclusion cannot immediately be drawn from results based on the total response approach for combined notification and hospitalisation data, as confidence intervals overlap. The table below summarises numbers and rates based on three different approaches.

A comparison of rates of meningococcal disease by ethnicity, 2002, using three different approaches

Ethnic group[1]	Prioritised approach for notification data		Total response approach for notification data		Total response approach for combined data	
	Cases	Rate [CI]	Cases	Rate [CI]	Cases	Rate [CI]
Maori	160	30.4 [25.7,35.1]	160	30.4 [25.7,35.1]	184	35.0* [29.9,40.0]
Pacific Peoples	90	44.9 [36.1,55.2]	100	43.1 [34.7, 51.6]	105	45.3 [36.6,54.0]
'Other'	19	7.7	20	7.7	-	-
Asian 'Other'	-	-	-	-	19	8.0
Non Asian'Other'	-	-	-	-	2	8.0
European	280	10.7 [9.5,12.0]	296	10.3 [9.1, 11.5]	330	11.5 [10.3,12.7]

[1] Eight notified cases had no ethnicity recorded.
*The higher rate is due solely to the higher number of cases identifying with the Maori ethnic group in the combined notification and hospitalisation dataset, as the denominators are identical.

Ethnic-specific notification rates based on the 'total-response' approach are slightly lower than ethnic-specific rates based on the 'hierarchical' approach, due perhaps to a lesser tendency for cases to record multiple ethnic groups on EpiSurv Case Report Forms than on census questionnaires. However rates based on numerators obtained via the matching of notification and hospitalisation data produce slightly higher rates. As ethnicity information on EpiSurv is most complete for meningococcal disease cases, this pattern is likely to be repeated for other notifiable diseases.

[a] See introductory section for explanation of this approach.
[b] The ethnicities assigned to a case in the 'combined' dataset, comprise a union of the ethnicities recorded separately in each data source.

Vaccine-preventable infections

This section describes the epidemiology of vaccine-preventable diseases in New Zealand. It includes 10 diseases that are in the National Immunisation Schedule. In addition there are a number of other diseases where vaccine is licenced for use in New Zealand but not routinely funded or widely used. These diseases include pneumococcal disease and varicella, both of which are also described in this section. Vaccine is also available and used to prevent diseases that are more appropriately described in other sections of this report, for example, hepatitis A, tuberculosis and meningococcal disease.

A new national immunisation schedule (see table below) commenced on 1 February 2002. Changes since the previous schedule include the introduction of a fifth dose of the pertussis vaccine (given as DtaP-IPV) to be given at four years of age, and boosters of tetanus to be offered at 45 and 65 years of age. A full description of current vaccination recommendations is contained in the Immunisation Handbook.[15]

The 2002 National Immunisation Schedule

Patient's age	DtaP-IPV	Hib-Hepatitis B	Hepatitis B	IPV	MMR	DtaP-Hib	Td	Influenza
6 weeks	•	•						
3 months	•	•						
5 months	•		•					
15 months					•	•		
4 years	•				•			
11 years				•*			•	
45 years							•	
65 years							•	•

*scheduled at 11 years of age for children who have not previously received four doses

Key: D=Diphtheria, T=Tetanus, aP=acellular Pertussis, IPV=Inactivated Polio Vaccine, Hib=*Haemophilus influenzae* type b, MMR=Measles-Mumps-Rubella, Td=Adult Tetanus-diphtheria

Diphtheria

One case of diphtheria was notified from Auckland District Health Board in June 2002. The case was a four-year old male of Pacific ethnicity, hospitalised with septic arthritis. The case had no toxin-related symptoms, although toxigenic *Corynebacterium diphtheriae* was isolated from a hip aspirate. The child was fully immunised and had a history of travel to Tonga approximately six months prior to diagnosis.

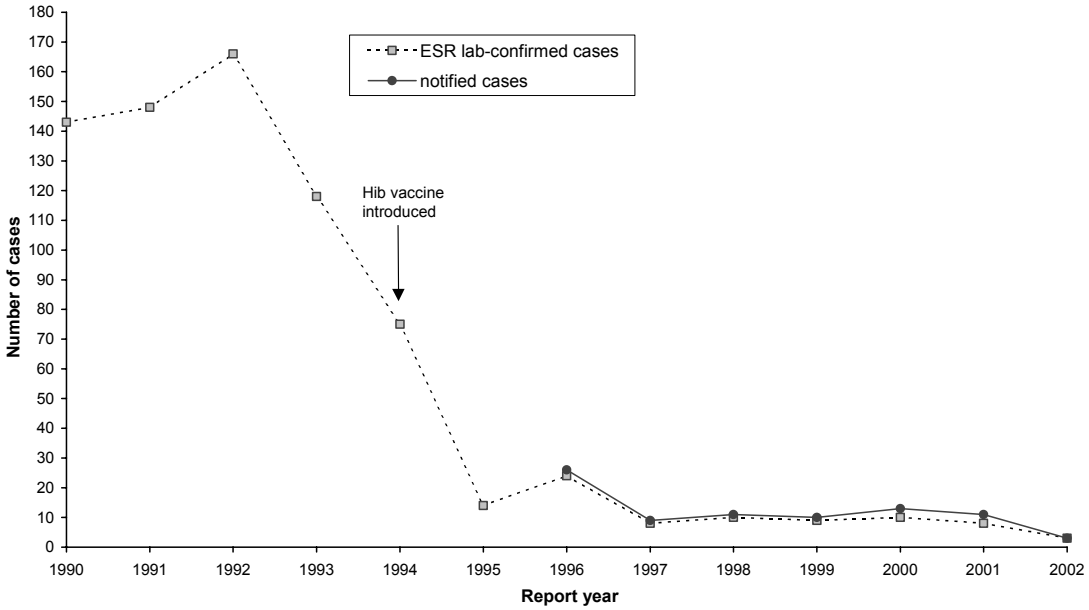
This was only the third time that the toxigenic strain has been isolated from a human infection in New Zealand in the last 16 years. Both of the previous cases had overseas links: a cutaneous infection of a traveller in 1987 and a respiratory case in 1998 [16].

Haemophilus influenzae type b disease

A Hib vaccine was introduced in January 1994. Prior to August 2000, the recommended immunisation schedule consisted of four doses of DTPH vaccine given at six weeks, three months, five months, and fifteen months of age. The new schedule introduced in mid August 2000, and continuing throughout 2002, recommended three doses of Hib vaccine at six weeks, three months and a booster at 15 months.

Three cases of *Haemophilus influenzae* serotype b (Hib) disease, all aged over fifty years, were notified in 2002. All three cases were laboratory confirmed by ESR Invasive Pathogens Laboratory. In comparison, eight laboratory-confirmed cases of Hib were notified during 2001, of whom five were aged less than five years. The following graph shows the number of ESR laboratory-confirmed cases of Hib since 1990, and the number of notified cases of Hib each year since 1996. Since the disease became notifiable in June 1996, the majority of notified cases has been laboratory-confirmed.

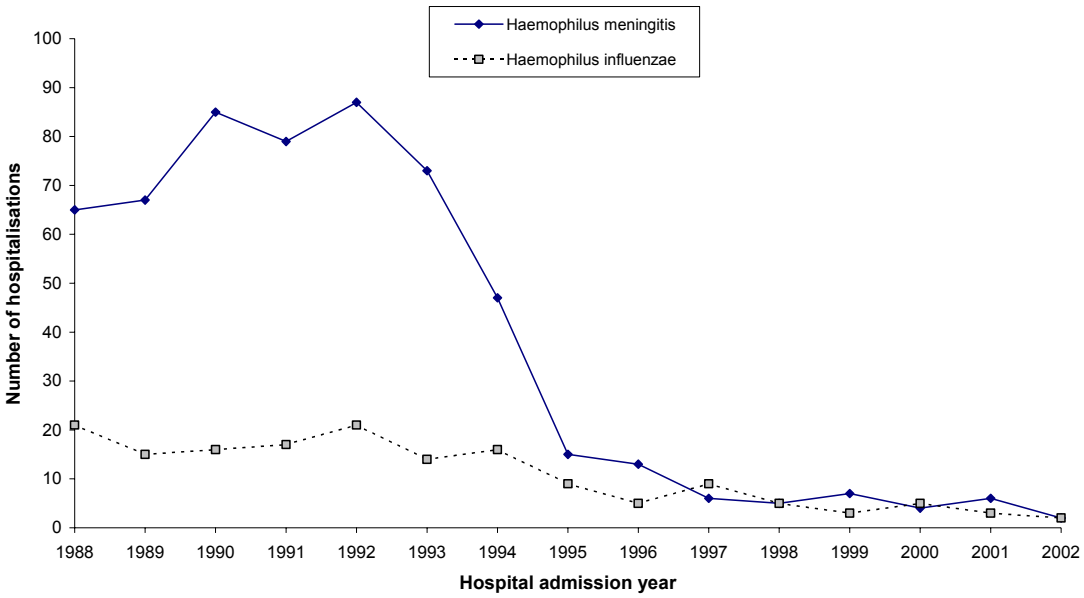
Hib ESR laboratory-confirmed and notified cases by year 1990 - 2002



Two cases in 2002 were males aged 51 and 52 years respectively, and one case was an 86-year-old female. Vaccination status was recorded for two cases, and as expected, given their ages, neither had been vaccinated against the disease. All three cases were hospitalised with septicaemia, and the 86-year-old later died[a]. The following graph shows the number of hospitalisations with primary diagnosis ICD9 code 041.5 (*Haemophilus influenzae*) and the number of hospitalisations with primary diagnosis ICD9 code 320.0 (*Haemophilus* meningitis) each year since 1988, according to hospital discharge data. Each year in the pre-vaccine era (pre 1994), over 90% of cases hospitalised with *Haemophilus* meningitis were aged under five years.

[a] This case died in 2001, although was not notified until 2002.

***Haemophilus influenzae* disease hospitalisations by year, 1988 - 2002**

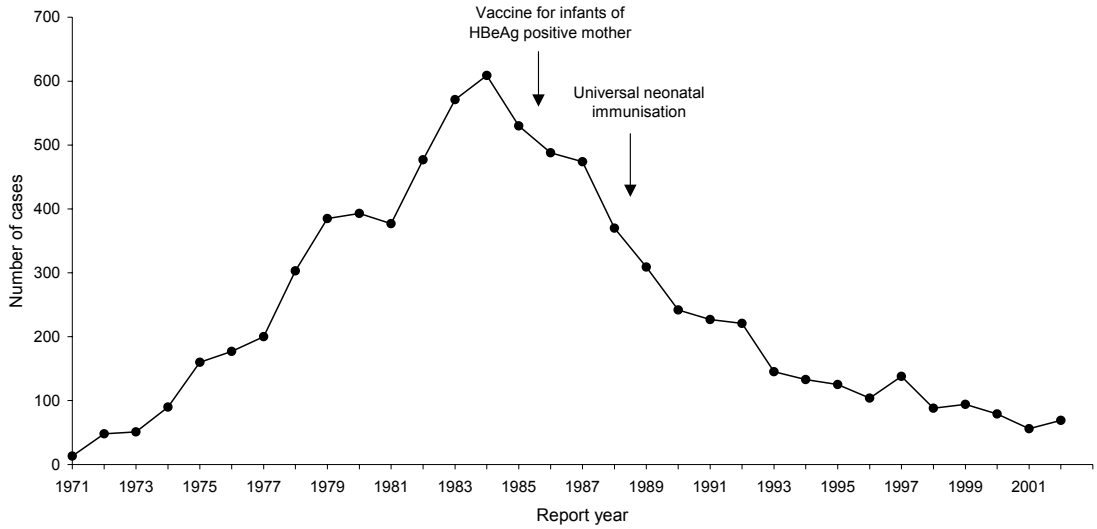


The incidence of Hib disease in 2002 is the lowest reported in New Zealand. This was also the first year without any cases in children. These data provide further evidence for the effectiveness of the national vaccination programme against this disease. [17]

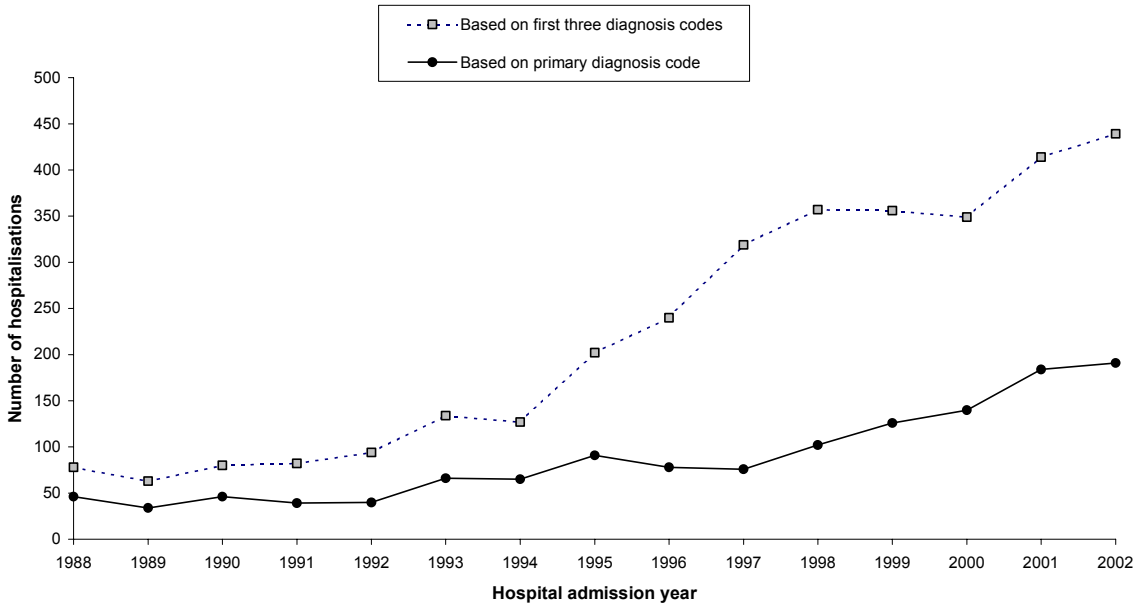
Hepatitis B

A total of 69 cases of acute hepatitis B was notified in 2002. The 2002 rate of 1.8 per 100 000 was similar to the 2001 rate of 1.5. Of the 59 cases for whom hospitalisation status was recorded on EpiSurv, 11 (18.6%) were hospitalised. According to hospital discharge data there were 191 hospitalisations due to hepatitis B (all phases) during 2002. Hepatitis B hospitalisations will often be due to infections that occurred many years previously, so they are not comparable to notifications. The following graphs show (i) the number of notified cases each year since 1971, and (ii) the hospitalisations (ICD9 codes 070.2, 070.3) each year since 1988.

Hepatitis B notifications by year, 1971 - 2002

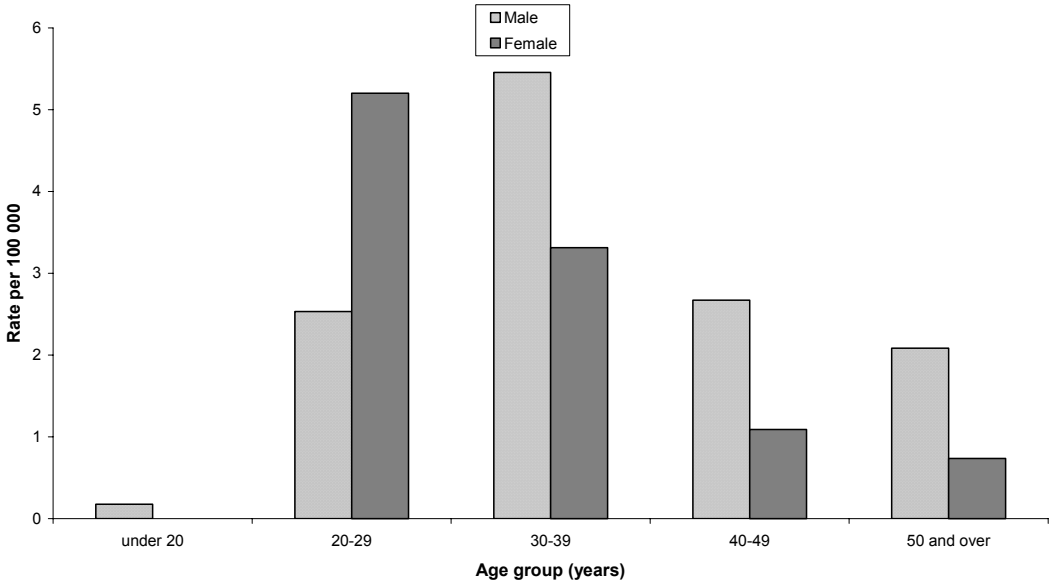


Hepatitis B hospitalisations by year, 1988 - 2002



Tairawhiti DHB recorded the highest hepatitis B incidence rate during 2002, with a rate of 13.6 cases per 100 000 (95% CI: 5.0-29.7), significantly higher than the national rate of 1.8 per 100 000. Notification rates were highest among males in the '30 to 39 years' age group, with a rate of 5.5 per 100 000. Age-specific rates of 3.9 (95% CI: 2.4-6.1) in the '20 to 29 years' age group, and 4.3 (95% CI: 2.8-6.4) in the '30 to 39 years' age group were significantly higher than in the general population. The following chart compares notification rates by age and sex.

Hepatitis B notification rates, 2002
by age and sex



The following tables illustrate the age and ethnic distribution of cases. Incidence rates were highest among Pacific peoples, with a crude rate of 4.5 per 100 000. Rates of disease in both Maori and Pacific peoples were significantly higher than in the European ethnic group.

Hepatitis B notifications and age-specific rates by ethnicity, 2002

Ethnicity	Age group in years											
	<1	**1-4**	**5-9**	**10-14**	**15-19**	**20-29**	**30-39**	**40-49**	**50-59**	**60-69**	**70+**	**Total**
European	0	0	0	0	1	4	14	4	7	1	2	33
	0.0	0.0	0.0	0.0	0.6	1.3	3.5	1.0	2.1	0.4	0.7	1.3
Maori	0	0	0	0	0	7	6	5	2	0	0	20
	0.0	0.0	0.0	0.0	0.0	8.5	7.7	8.6	6.0	0.0	0.0	3.8
Pacific peoples	0	0	0	0	0	5	3	0	0	0	1	9
	0.0	0.0	0.0	0.0	0.0	14.8	9.5	0.0	0.0	0.0	22.6	4.5
Other	0	0	0	0	0	1	1	1	0	0	0	3
	0.0	0.0	0.0	0.0	0.0	2.3	2.2	2.6	0.0	0.0	0.0	1.2
Unknown	0	0	0	0	0	2	1	0	1	0	0	4
Total	0	0	0	0	1	19	25	10	10	1	3	69
	0.0	0.0	0.0	0.0	0.3	3.9	4.3	1.9	2.4	0.4	0.9	1.8

Number of cases
Rate per 100 000

Hepatitis B - crude and age-standardised rates by ethnicity, 2002

Ethnicity	Crude rate [95% CI]	Age-standardised rate* [95% CI]
European	1.3 [0.9, 1.8]	1.2 [0.8, 1.7]
Maori	3.8 [2.3, 5.9]	4.2 [2.5, 8]
Pacific peoples	4.5 [2.1, 8.5]	5.3 [1.8, 14.2]
Other	1.2 [0.3, 3.5]	1.0 [0.2, 6.6]
Total	1.8 [1.4, 2.3]	

* Directly standardised to the NZ population

Three notified cases reported they had been immunised. Two cases were pregnant. The following table provides a summary of risk factor information for hepatitis B in 2002. Several cases had more than one risk factor recorded. Unlike the previous year when only one notified case recorded overseas travel, 10 (20.8%) of the 48 notified cases for whom this information was recorded had travelled overseas during the incubation period for this infection. Sexual or household contact with a confirmed case, and occupational exposure[a] to blood remained commonly reported risk factors in 2002.

Risk factors associated with hepatitis B notifications, 2002

Risk Factor	Yes	No	Unknown	Proportion[1]
Travelled overseas during incubation period	10	38	21	20.8%
Sexual contact with confirmed case or carrier	8	32	29	20.0%
Household contact with case	8	37	24	17.8%
Occupationally exposed to blood	4	41	24	8.9%
Body piercing or tattooing in last 12 months	4	44	16	8.3%
Child of seropositive mother	1	43	25	2.3%
History of injecting drug use	1	47	21	2.1%
Blood product or tissue recipient	0	43	26	0%
Dialysis patient	0	45	24	0%

1 "Proportion" refers to the percentage of cases who answered "yes" out of the total number of cases for whom this information was known.

These surveillance data suggest that the national immunisation programme has been effective in reducing the risk of hepatitis B infection for the cohort of New Zealanders under the age of 20 years. The prevalence of hepatitis B carriage (HbsAg) is estimated to be 5% for Maori, Pacific people and ethnic minorities, and about 0.5% for New Zealanders of European extraction.[18] The risk of infection therefore remains for older age groups, particularly household and sexual contacts and new-born infants of these carriers.

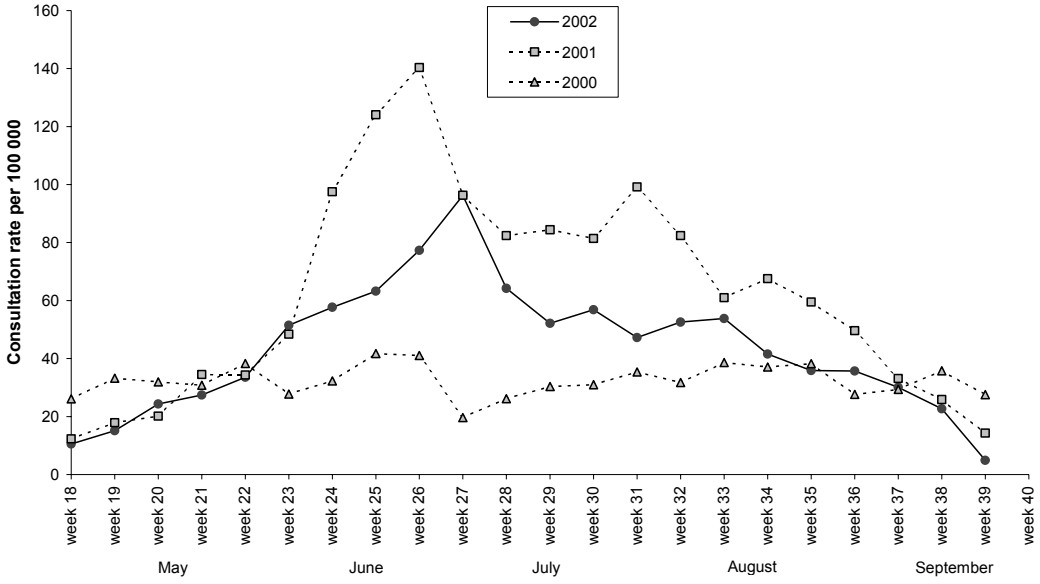
[a] Occupational exposure to blood occurred in three health workers and in one mechanic

Influenza

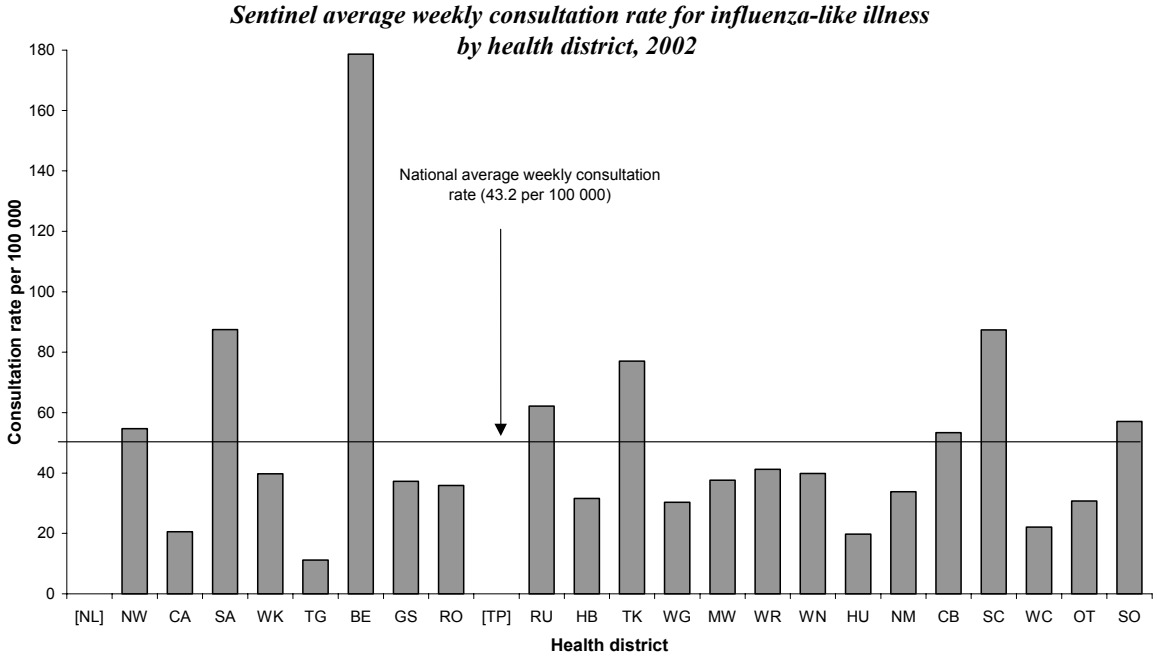
National influenza surveillance in 2002 was undertaken between May and September using a sentinel network of 92 general practices. On average 88 practices, with a total patient roll of 339,954 participated each week.

During the surveillance period, 3159 consultations for influenza-like illness were reported, and the average weekly consultation rate was 43.2 per 100 000 patient population. This rate is the second lowest rate recorded by the sentinel surveillance system, which began in 1991. The 2002 rate was higher than the 2000 rate of 32.5 but lower than the 2001 rate of 62.8. The consultation rate remained relatively low throughout the sentinel surveillance period but peaked in week 27 (at the beginning of July). This pattern was consistent with isolations of influenza virus in the five regional virus laboratories, where peak activity also occurred in week 27, and considerable activity continued almost until the end of the sentinel surveillance period. The following graph compares the weekly consultation rates for influenza-like illness in 2002 with 2001 and 2000.

Weekly consultation rates for influenza-like illness, 2000, 2001 and 2002

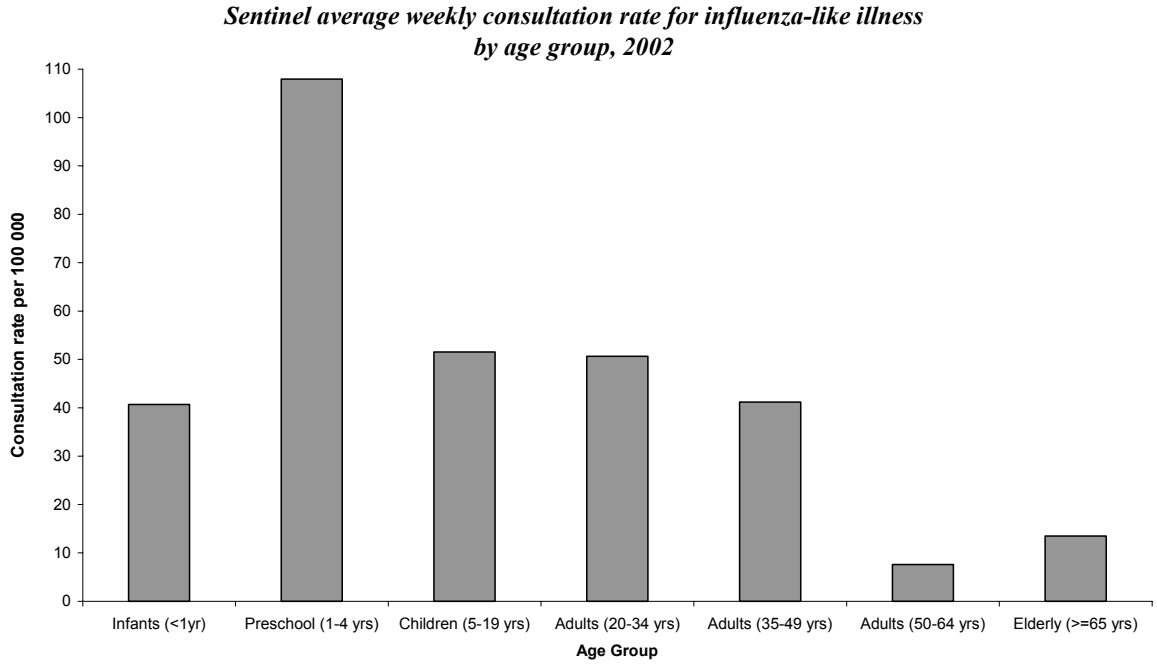


Consultation rates varied between health districts, with rates above the national average in eight of the 22 health districts and rates of more than twice the national average in Eastern Bay of Plenty (178.7 per 100 000) and South Auckland (87.5) health districts. The following bar chart shows the average weekly consultation rates by health district for the influenza season.



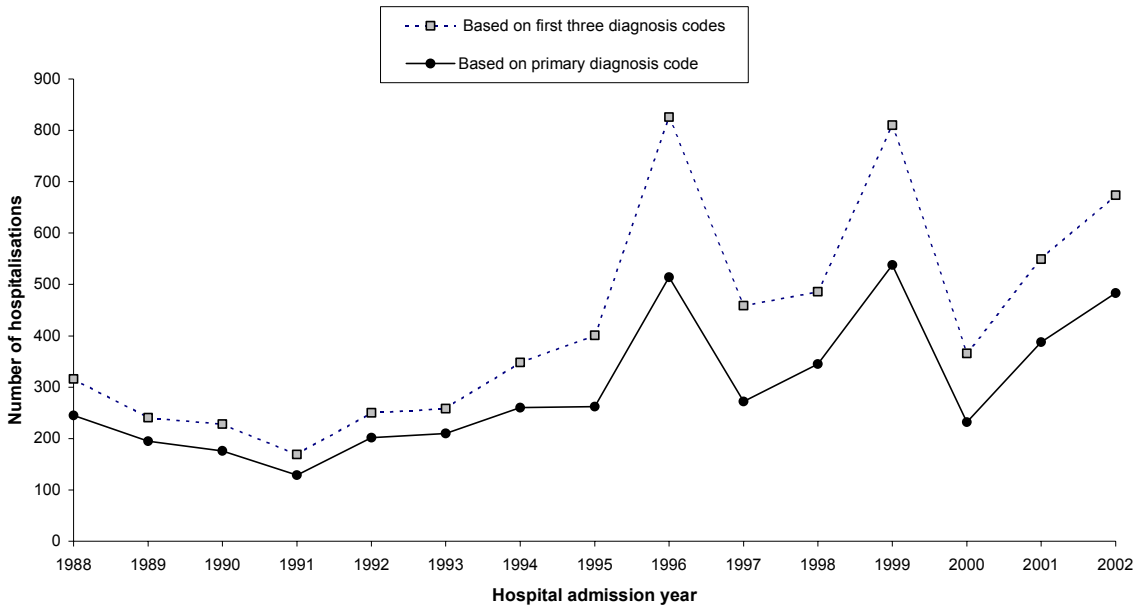
Note [ ] Northland and Taupo health districts did not participate in 2002

Pre-schoolers (aged 1 to 4 years) and children (aged 5 to 19 years) were the most likely to be seen by a general practitioner for an influenza-like illness, with respective age-specific average weekly consultation rates of 107.9 and 51.5. The lower rate of 13.5 in those aged 65 years or over was likely to due, at least in part, to higher levels of vaccination in this age group. The following chart shows the average weekly consultation rate in 2002 by age group.



Although, based on sentinel surveillance, the average weekly consultation rate for influenza dropped from 62.8 per 100 000 patient population in 2001 to 43.2 per 100 000 in 2002, the number of hospitalisations due to influenza has been steadily rising since 2000. The following graph shows the number of hospitalisations (ICD9 code 487) each year since 1988, according to hospital discharge data. There were 483 hospitalisations in 2002 compared to 388 in 2001.

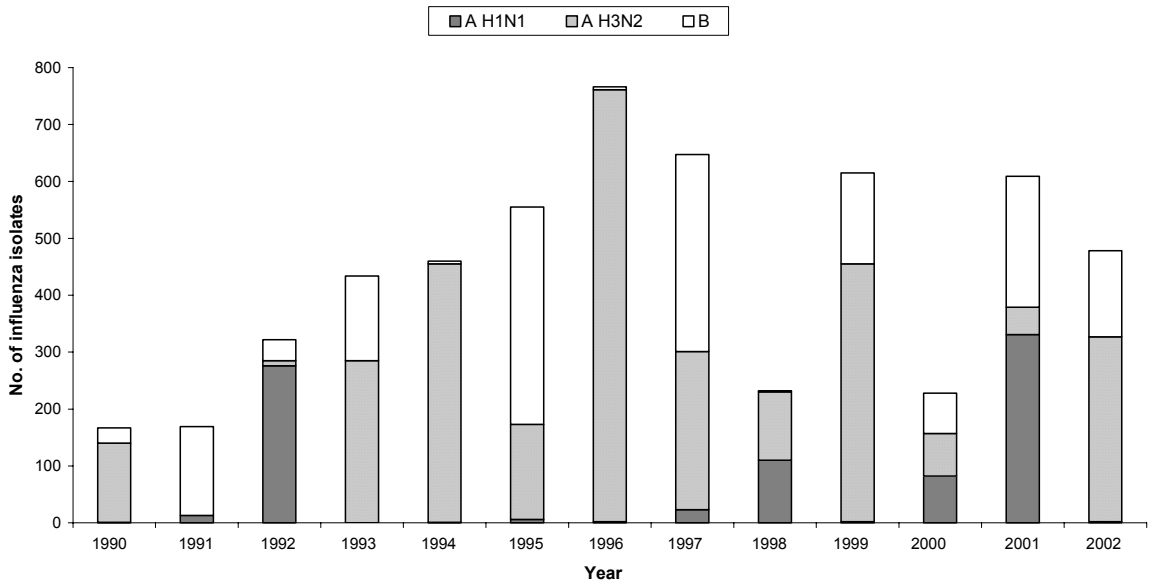
Influenza hospitalisations by year, 1988 - 2002



A total of 702 influenza isolates were identified in 2002 - higher than the 654 isolates in 2001 and more than twice the 303 isolates in 2000. Of the 2002 isolates, 241 came from sentinel practice surveillance during May to September. This is lower than the 313 sentinel isolates identified in 2001, but more than three times higher than the 73 sentinel isolates identified in 2000. There were 461 non-sentinel isolates identified in 2002, compared to 341 in 2001, and 230 in 2000.

During 2002, the majority of influenza isolates (551 or 78% of all isolates) were characterised as influenza A. There were 151 influenza B isolates identified, representing 22% of all influenza isolates. In comparison, influenza B made up 35% of all isolates in 2001, and 24% in 2000. The following graph shows the number and percentage of typed and subtyped (not total) influenza isolates from 1990 to 2002. Three noticeable changes in predominant patterns are described below.

Total influenza isolates by type, 1990-2002



Influenza A(H1N1)

From 1990 to 1999, influenza A(H1N1) predominated or co-dominated only in 1992 (86% of typed/subtyped isolates) and 1998 (47%). However in 2001 and 2000, influenza A(H1N1) *consecutively* predominated. There were 82 A(H1N1) isolates in 2000 (36%) and 331 in 2001 (54%). This is in contrast to 2002, when only two A(H1N1) were isolated.

Influenza A(H3N2)

Influenza A(H3N2) viruses have often been associated with more severe disease and with excess pneumonia and influenza mortality. For example, the peak of 94 deaths in 1996 in New Zealand was recorded during an A(H3N2) epidemic. From 1993 to 2000, A(H3N2) was the predominant or co-dominant strain each year. In 2001, A(H3N2) constituted only 8% of typed/subtyped isolates. However, in 2002 A(H3N2) predominated with 68% of typed/subtyped isolates.

Influenza B

It is well documented that influenza B predominates or co-dominates every second year. This was true during 1990-1999 in New Zealand. Influenza B predominated or co-dominated in 1991, 1993, 1995, 1997, and 1999. When influenza B was not the predominant or co-dominant strain, it comprised a small proportion: 16% in 1990, 11% in 1992, 1% in 1994, 1% in 1996, and 1% in 1998. However, in 2000, although influenza B was not the predominant or co-dominant strain, it comprised 31% of typed/subtyped isolates. In 2001 (38%) and 2002 (32%), influenza B was *consecutively* the co-predominant strain.

Characterisation of the influenza viruses isolated during the 2002 winter indicated a need for a change in the Influenza B components of the vaccine for the 2003 winter. Accordingly, the 2003 Southern Hemisphere winter influenza vaccine has the following composition:

- A/New Caledonia/20/99 (H1N1)-like strain
- A/Moscow/10/99 (H3N2)-like strain
- B/Hong Kong/330/01-like strain

This composition differs from the vaccine used in the 2002 Southern Hemisphere winter, during which the influenza B component was the B/Sichuan/379/99-like strain.

Influenza immunisation is recommended for those at increased risk of complications from influenza due to either age or medical condition (see the *Immunisation handbook* for details). Influenza vaccination has been free for people ≥65 years of age since 1997. Since 1999, it has been extended to younger people with chronic illnesses who are at risk of developing complications from influenza.

These surveillance data suggest a mixed pattern for 2002 with a relatively low incidence of influenza, but with more severe disease as reflected in hospital admissions. More severe disease has been seen in several recent years (1996, 1999) when H3N2 influenza has predominated. The impact of this disease, in terms of hospitalisations and fatalities, is very much higher than is recorded in these surveillance data.[7]. These data provide a further reminder about the importance of increasing influenza vaccination rates.

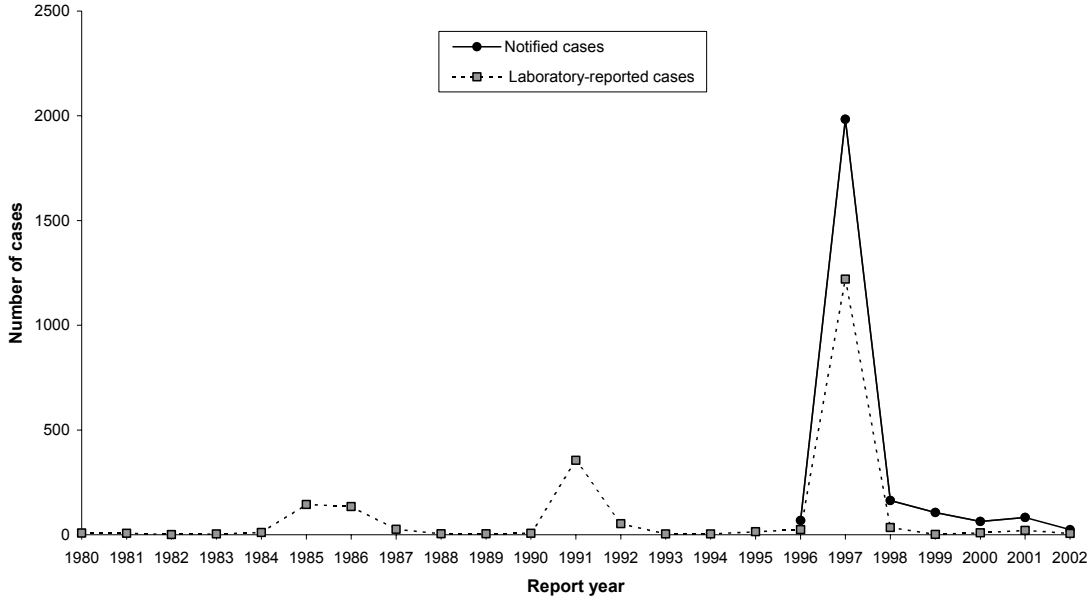
Measles

Measles vaccine has been used in New Zealand since 1969. Despite this, New Zealand has continued to have measles epidemics. Since 1980, major epidemics of measles have occurred in 1985, 1991 and 1997. A mathematical model [19] predicted that measles epidemics would continue in New Zealand if the MMR vaccine continued to be given at 15 months and 11 years of age. Therefore from January 2001 the immunisation schedule was changed to give the second dose of MMR at four years of age prior to school entry, and the first dose continued to be given at 15 months[a]. This same schedule was recommended in 2002.

Measles became a notifiable disease in June 1996. During the 1997 epidemic, a total of 1984 cases was notified. In 2002 there were 25 notifications, and six laboratory-reported cases. Matching of data indicated that three cases were both notified and laboratory-reported. The 2002 notification rate of 0.7 per 100 000 (95% CI: 0.4-1.0) was significantly lower than the 2001 rate of 2.2 (95% CI: 1.8-2.8). The following graph shows notified and/or laboratory-reported cases each year since 1980.

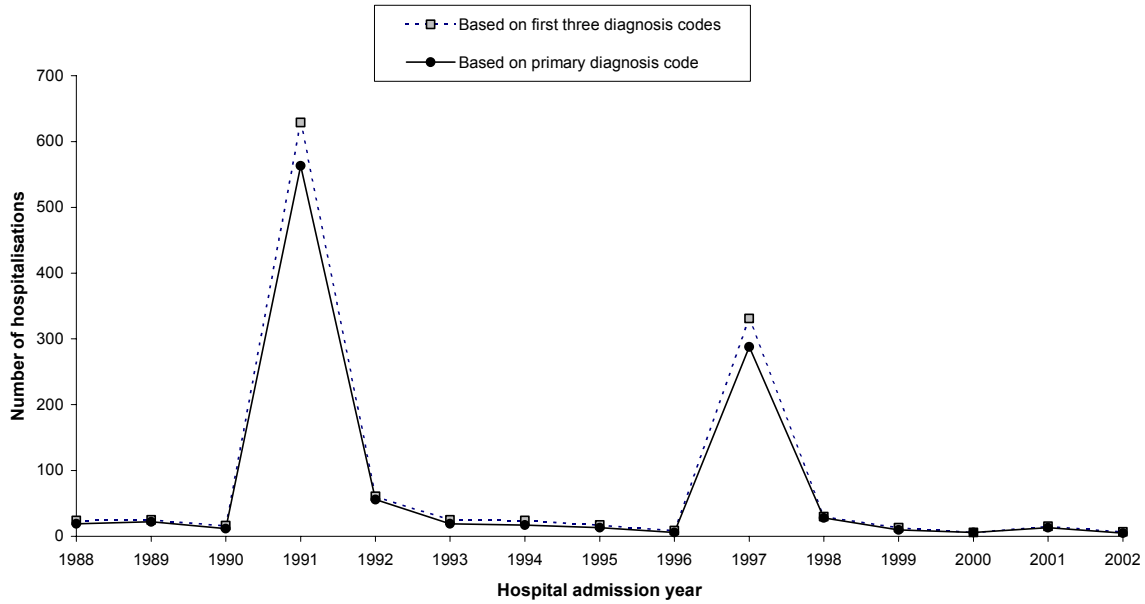
[a] During 2001 there was also an MMR catch-up programme throughout the country for all children aged between 5 and 10 years.

Measles notifications and laboratory-reported cases by year, 1980 - 2002



Of the 20 notified cases in 2002 for whom hospitalisation status was recorded on EpiSurv, none were hospitalised. In contrast, hospital discharge indicated five measles hospitalisations during 2002. The following graph shows the number of hospitalisations (ICD9 code 055) each year since 1988.

Measles hospitalisations by year, 1988 - 2002

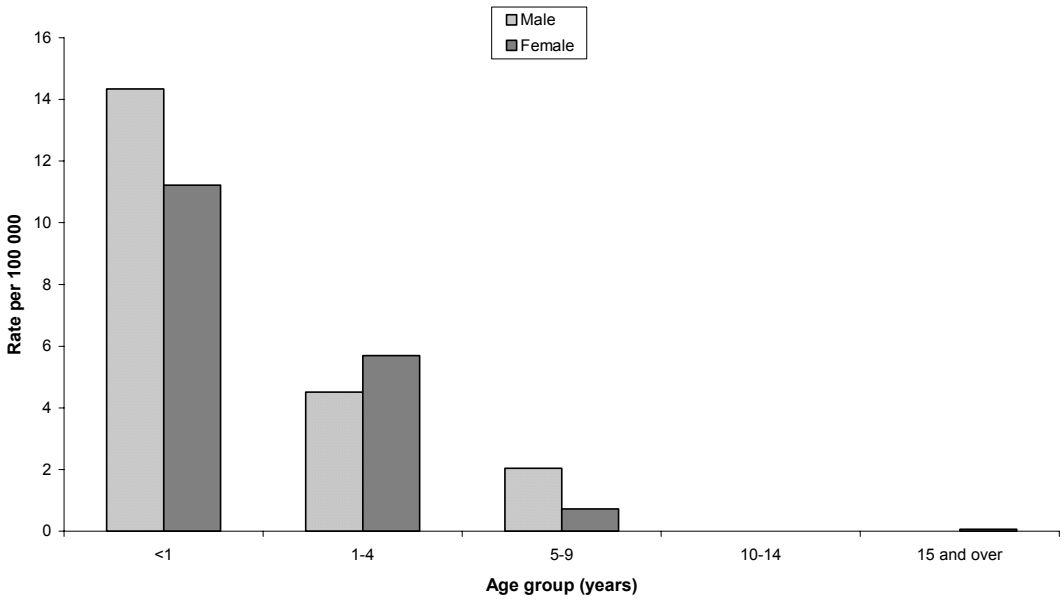


Measles notification rates varied throughout the country in 2002. Rates significantly higher than the national average were recorded in West Coast DHB, with a rate of 16.5 per 100 000 (95% CI: 5.4-38.5), and Nelson-Marlborough DHB (4.9, 95% CI: 1.8-10.7). Age-specific

rates were highest in the 'less than one year' and the '1 to 4 years' age groups, with rates of 12.8 (95% CI: 5.2-26.4) and 5.1 (95% CI: 2.5-9.1) respectively.

Rates were significantly higher in the European ethnic group (3.3, 95% CI: 1.1-7.7) than in the general population. The following bar chart compares notification rates by age and sex.

Measles notification rates, 2002 by age and sex



No measles cases reported overseas travel during the incubation period and no cases reported contact with another case. Of the 15 cases for whom this information was recorded, 5 (33.3%) attended school, pre-school or childcare. Vaccination status was recorded for 20 cases. Of these 5 (25%) had received at least one dose of MMR vaccine. One laboratory-confirmed case had been vaccinated. The table below shows vaccination status by age group.

Age and vaccination status of measles notifications, 2002

Age group[1]	Total cases	Vaccination Status					
		1 dose	2 doses	3 doses	Vaccinated[2] (no dose info)	Not vaccinated	Vaccination status unknown
< 15 mths	9	0	0	0	0	8	1
15 mths - 4yrs	9	1	0	0	0	7	1
5 - 10 yrs	4	0	3	0	1	0	0
11+ yrs	1	0	0	0	0	0	1
Total	**23**[1]	**1**	**3**	**0**	**1**	**15**	**3**

1 Two cases did not have age recorded.
2 Case was immunised but the number of doses was not recorded

These surveillance data all confirm the very low incidence of measles in New Zealand in 2002, which is typical during inter-epidemic periods. It is likely that many of the 'probable' cases notified during 2002 had morbilliform rash and fever caused by diseases other than measles. Indeed, some countries do not accept clinical cases without laboratory

confirmation.[20, 21] The six laboratory-confirmed cases show that the New Zealand population continues to be exposed to this virus and remains vulnerable to future epidemics.

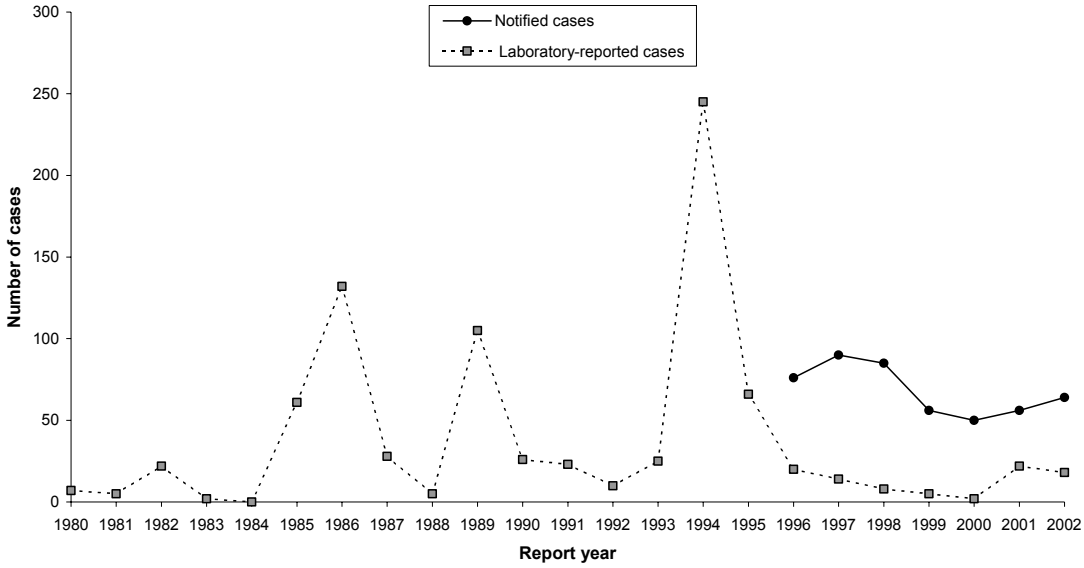
It is difficult to predict if, and when, New Zealand may experience a further measles epidemic. Based on an inter-epidemic period of 5 to 7 years, a further national epidemic could be expected in the 2002-4 period. However, the large vaccination programme associated with the 1997 epidemic, [22] and the change in the immunisation schedule may be sufficient to delay or even prevent this epidemic occurring. An important piece of missing information is the measles vaccine coverage rate in New Zealand.

Mumps

From 1970 until the introduction of the MMR vaccine in 1990, the number of cases of mumps increased every three to four years. The last epidemic was in 1994. Mumps vaccine was introduced for the first time with MMR vaccine in 1990. A second dose was added in 1992, as part of MMR for 11-year-old children. The immunisation schedule was last altered in January 2001, maintaining the first dose of MMR at 15 months and changing the second dose from eleven to four years of age, to prevent future epidemics of measles.

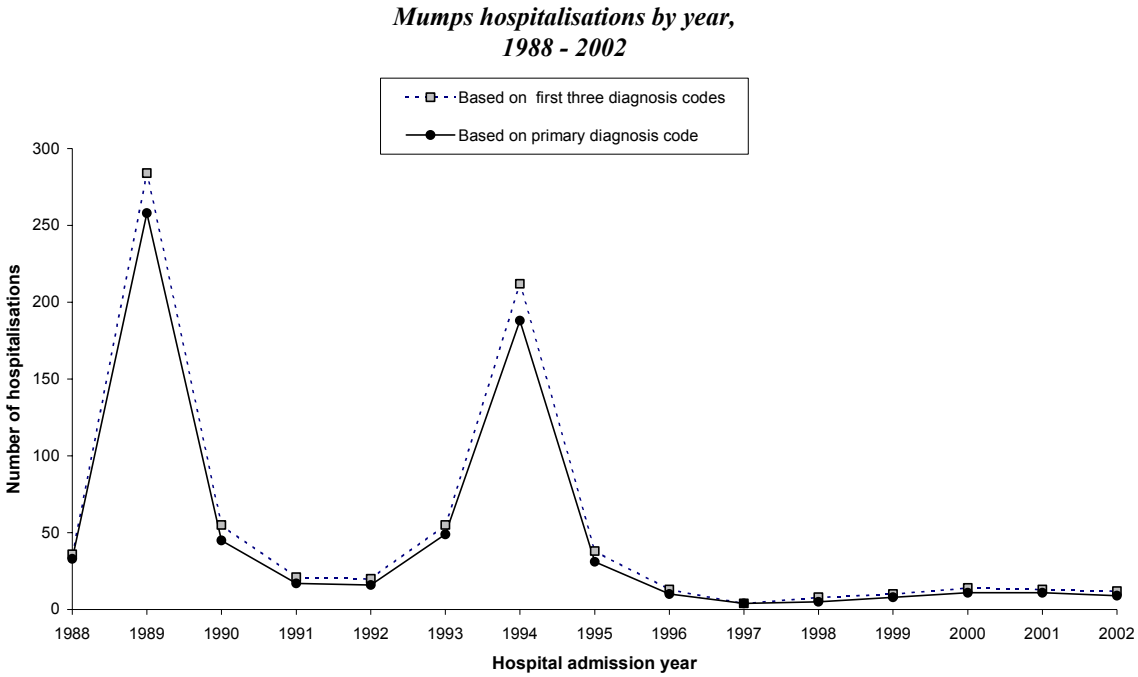
Mumps became a notifiable disease in June 1996. In 2002 there were 64 notifications, and 22 laboratory-reported cases. Matching of data indicated that nine cases were both notified and laboratory-reported. EpiSurv recorded ten laboratory-confirmed cases. The 2002 notification rate of 1.7 per 100 000 (95% CI: 1.3-2.2) was similar to the 2001 rate of 1.5 per 100 000. The following graph shows notified and/or laboratory-reported cases each year since 1980.

Mumps notifications and laboratory-reported cases by year, 1980 - 2002



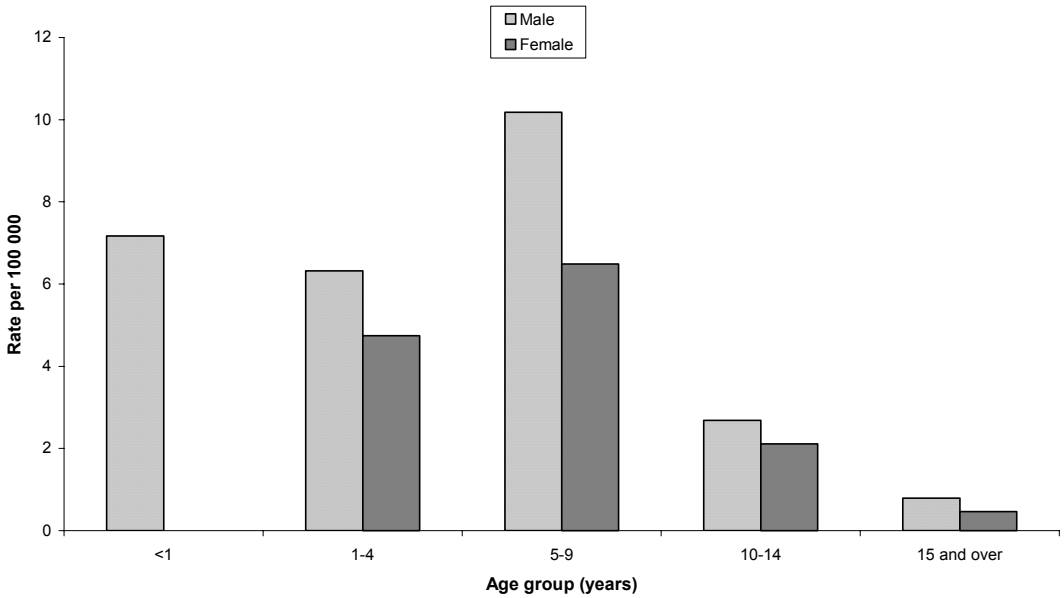
Of the 56 notified cases in 2002 for whom hospitalisation status was recorded on EpiSurv, one (1.8%) was hospitalised. In contrast, hospital discharge indicated nine mumps

hospitalisations during 2002. The following graph shows the number of hospitalisations (ICD9 code 072) each year since 1988.



Mumps notification rates varied throughout the country in 2002. The highest rates were reported from four South Island District Health Boards: Otago (5.3 per 100 000), Nelson-Marlborough (4.9), West Coast (3.3), and Southland (2.9). The rate in Otago (5.3, 95% CI: 2.4-10.0) was significantly higher than the national average. Age-specific rates were highest in the '5 to 9 years' and the '1 to 4 years' age groups, with rates of 8.4 (95% CI: 5.4-12.5) and 5.6 (95% CI: 2.9-9.7) respectively. In all age groups, rates were higher among males than females. The following bar chart compares notification rates by age and sex.

Mumps notification rates, 2002
by age and sex



Crude notification rates were significantly higher in the Pacific peoples ethnic group (5.0, 95% CI: 2.4-9.2) than in the general population. Age-standardised rates were also highest in Pacific peoples. The tables below illustrate the age and ethnic distribution of cases.

Mumps notifications and age-specific rates by ethnicity, 2002

Ethnicity	Age group in years											
	<1	**1-4**	**5-9**	**10-14**	**15-19**	**20-29**	**30-39**	**40-49**	**50-59**	**60-69**	**70+**	**Total**
European	0	2	12	4	2	2	3	1	0	0	1	27
	0.0	1.6	7.1	2.3	1.2	0.7	0.8	0.3	0.0	0.0	0.3	1.0
Maori	1	3	4	0	0	0	0	0	0	0	0	8
	7.1	5.6	6.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	1.5
Pacific peoples	0	5	3	0	0	0	0	1	1	0	0	10
	0.0	26.1	12.7	0.0	0.0	0.0	0.0	4.6	7.5	0.0	0.0	5.0
Other	0	2	1	0	0	1	2	1	0	0	0	7
	0.0	14.3	5.4	0.0	0.0	2.3	4.4	2.6	0.0	0.0	0.0	2.8
Unknown	1	0	4	3	1	3	0	0	0	0	0	12
Total	2	12	24	7	3	6	5	3	1	0	1	64
	3.7	5.6	8.4	2.4	1.1	1.2	0.9	0.6	0.2	0.0	0.3	1.7

Number of cases
Rate per 100 000

Mumps - crude and age-standardised rates by ethnicity, 2002

Ethnicity	Crude rate [95% CI]	Age-standardised rate* [95% CI]
European	1.0 [0.7, 1.5]	1.2 [0.8, 1.7]
Maori	1.5 [0.7, 3]	0.9 [0.4, 4.1]
Pacific peoples	5.0 [2.4, 9.2]	4.0 [1.8, 11.5]
Other	2.8 [1.1, 5.8]	2.6 [1, 8.4]
Total	1.7 [1.3, 2.2]	

* Directly standardised to the NZ population

Just one mumps case reported overseas travel during the incubation period, and three cases (or 8.1% of cases for whom this information was recorded) reported contact with another case. A total of 28 cases (or 58.3% of cases for whom this information was recorded) attended school, pre-school or childcare. Vaccination status was recorded for 46 cases. Of these, 33 (71.7%) had received at least one dose of MMR vaccine. The table below shows vaccination status by age group.

Age and vaccination status of mumps notifications, 2002

Age group	Total cases	Vaccination Status					
		1 dose	2 doses	3 doses	Vaccinated[1] (no dose info)	Not vaccinated	Vaccination status unknown
< 15 mths	2	0	0	0	0	1	1
15 mths - 4yrs	12	5	3	2	0	2	0
5 - 10 yrs	26	7	9	0	2	3	5
11+ yrs	24	2	2	0	1	7	12
Total	**64**	**14**	**14**	**2**	**3**	**13**	**18**

[1] Case was immunised but the number of doses was not recorded

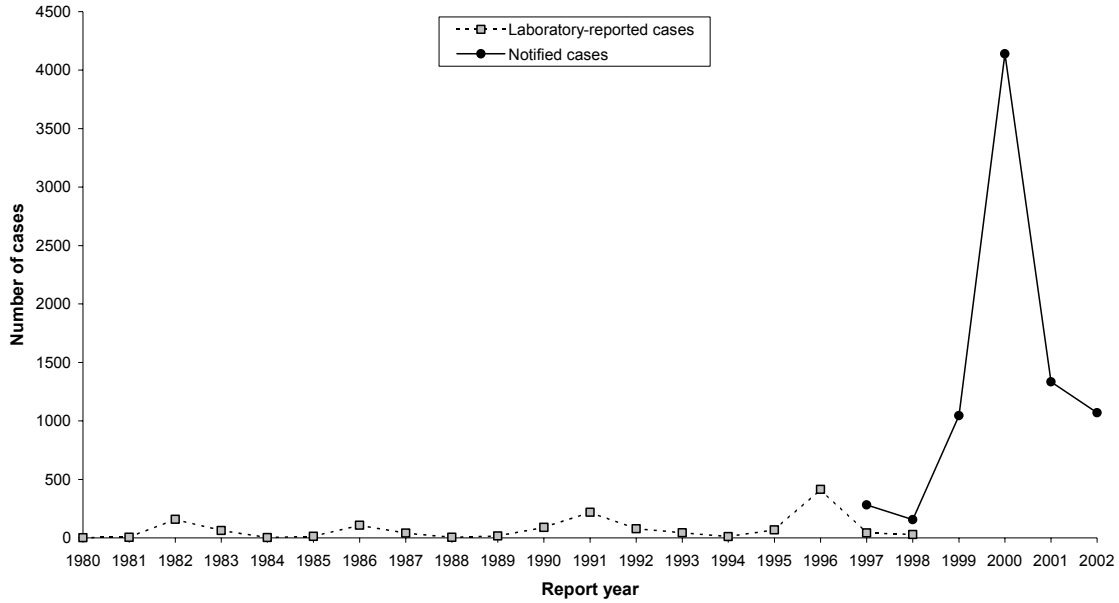
These surveillance data suggest that mumps immunisation has been effective at reducing the incidence of this disease in New Zealand. The periodic national epidemics that resulted in up to 250 hospitalisations a year in the pre-vaccine era have now become less frequent, and may have been eliminated.

Pertussis

Pertussis epidemics show a four to five year periodicity in New Zealand. The recent epidemic started in 1999, was most intense in 2000, and continued into 2001. It appeared to have ceased by 2002, though the incidence of pertussis remained elevated. A total of 1071 cases of pertussis was notified in 2002, representing a rate of 28.7 per 100 000 (95% CI: 26.9-30.4), significantly lower than the rate of 35.5 in 2001. However, the incidence throughout 2002 remained at a level higher than that seen in the last inter-epidemic years of 1997 (284) and 1998 (153). Of the 1071 cases in 2002, 71.0% were laboratory-confirmed, either by serological means, PCR or isolation of *Bordetella pertussis*; a further 10.2% were epidemiologically linked to confirmed cases of the disease. The remainder of the cases was

notified on clinical grounds only. Pertussis became notifiable in June 1996. The following graph shows the number of notified cases and/or laboratory-reported cases each year since 1980.

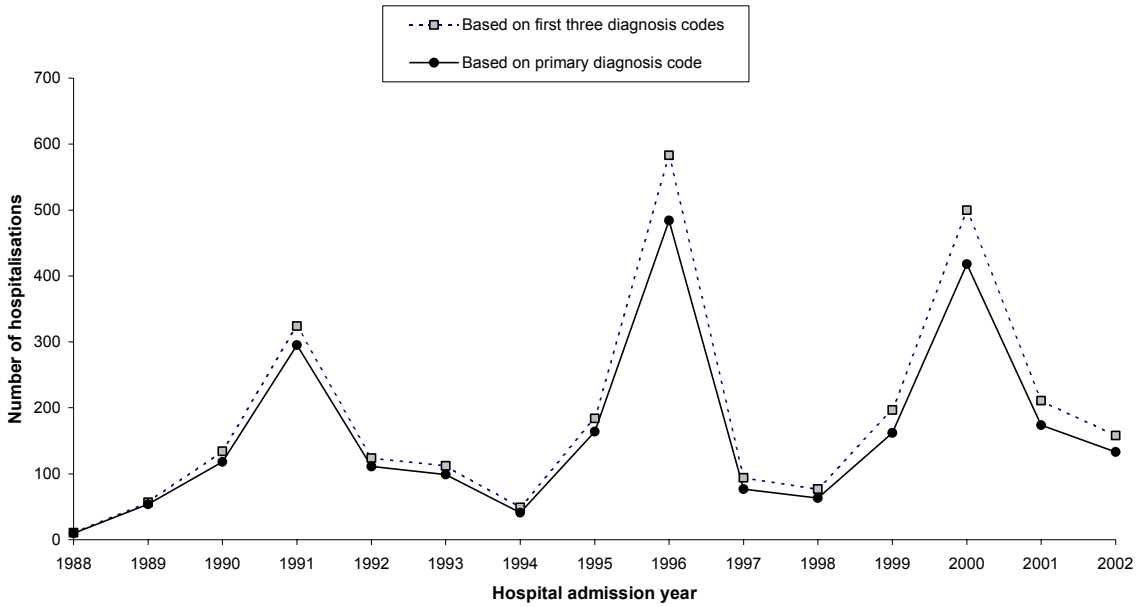
Pertussis laboratory-reported cases and notifications by year, 1980 -2002



One fatal case of pertussis was recorded in 2002, that of a one-month-old female of European ethnicity.

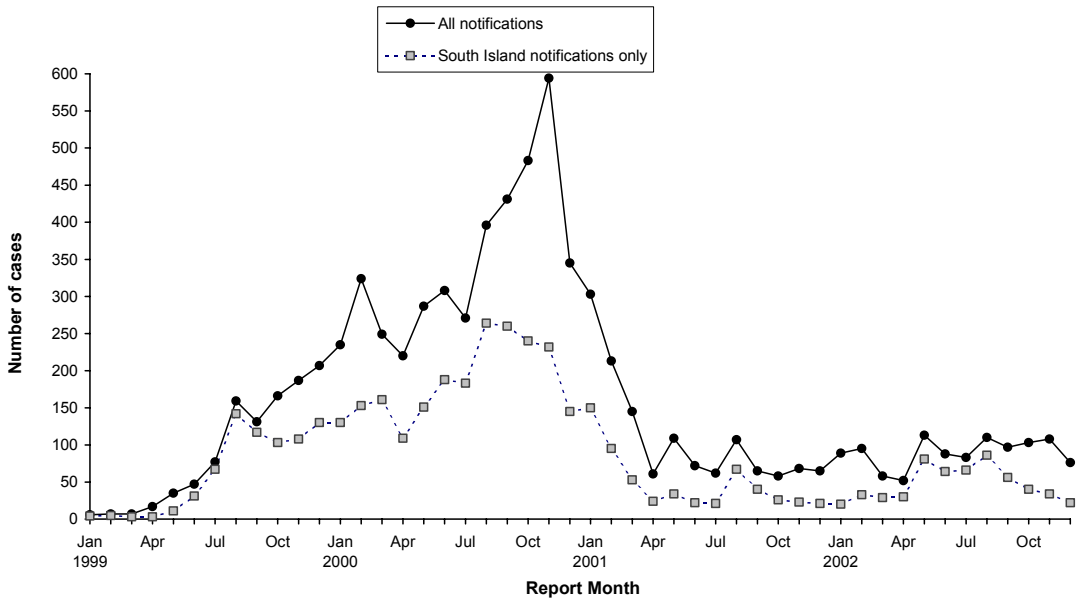
Of the 964 notified cases in 2002 for whom hospitalisation status was recorded on EpiSurv, 98 (10.2%) were hospitalised. According to hospital discharge data (see graph below), hospitalisations (ICD9 code 033) due to pertussis totalled between 133 (based on the primary diagnosis code) and 158 (based on the first three diagnosis codes) in 2002.

Pertussis hospitalisations by year, 1988 - 2002



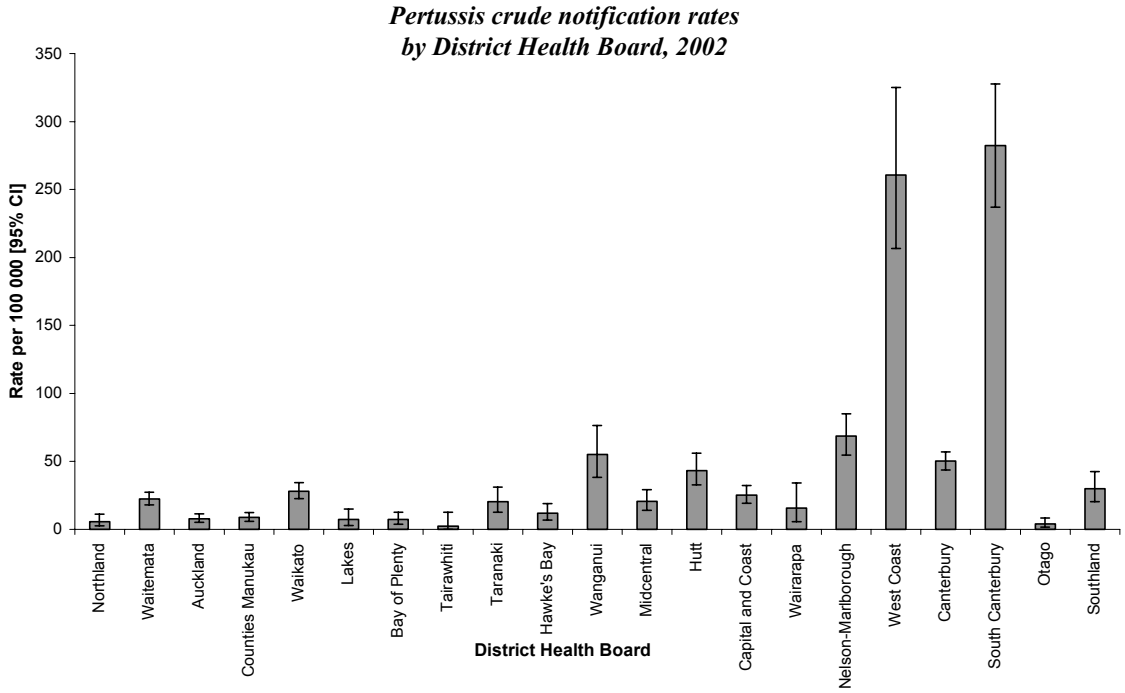
The following graph shows the number of cases of pertussis notified each month from North and South Island District Health Boards since before the last epidemic. Of note is the high proportion of cases notified from the South Island, particularly during the months May through August 2003.

Notified cases of pertussis by month, January 1999 - December 2002



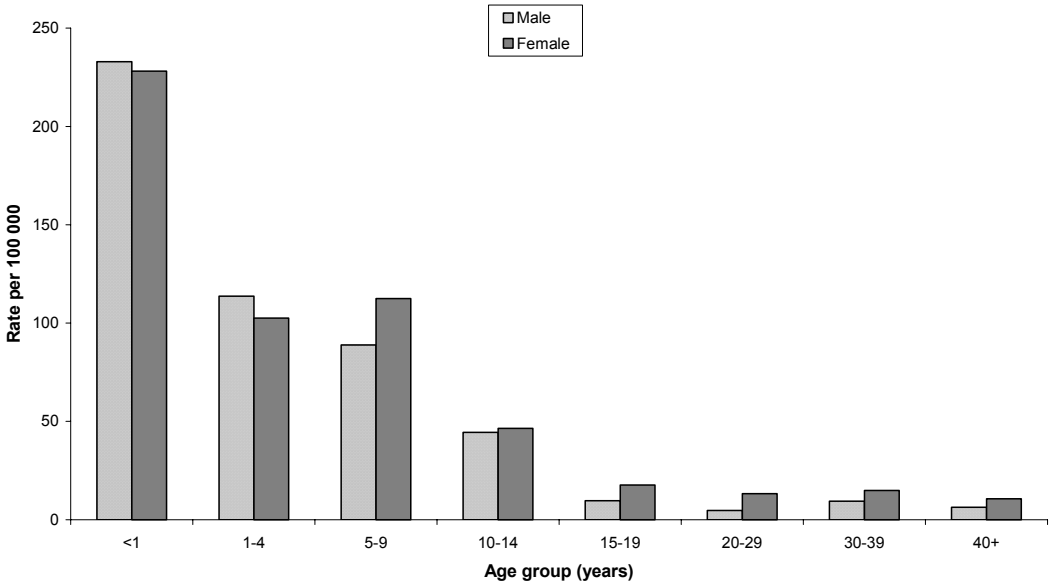
Rates of pertussis varied considerably throughout the country in 2002, as the following bar chart demonstrates. Rates significantly higher than all other District Health Boards were

recorded in South Canterbury (282.3 per 100 000) and West Coast (260.8). Rates significantly higher than the national average of 28.7 cases per 100 000 were also reported by Nelson-Marlborough (68.6), Wanganui (55.0), Canterbury (50.3), and Hutt (43.2) DHBs. Seven outbreaks of pertussis were reported in 2002: five from West Coast DHB and one each from Wanganui and Manawatu health districts.



The following bar chart illustrates how notification rates decreased with increasing age. The age-specific rate of 232.4 (95% CI: 192.0-272.8) in infants less than one year was significantly higher than that for all other age groups. Age-specific rates of 108.7 and 100.7 in the '1 to 4 years' and the '5 to 9 years' age groups respectively, were also significantly higher than in older age groups. There was little or no differentiation in rates between the sexes.

Pertussis notification rates, 2002
by age and sex



The tables below illustrate the age and ethnic distribution of cases. Rates were highest in Maori aged less than one year (with a rate of 235.8), although in the Maori ethnic group rates decreased particularly fast with increasing age. Overall, rates were significantly higher in the European ethnic group than in all other ethnic groups. This difference was even more pronounced when age-standardised rates were compared.

Pertussis notifications and age-specific rates by ethnicity, 2002

Ethnicity	**Age group in years**											
	<1	**1-4**	**5-9**	**10-14**	**15-19**	**20-29**	**30-39**	**40-49**	**50-59**	**60-69**	**70+**	**Total**
European	65	176	214	112	23	33	57	48	32	18	11	790
	218.5	144.7	126.7	63.1	14.3	10.8	14.4	12.1	9.6	7.8	3.8	30.2
Maori	33	23	16	10	5	4	6	3	0	0	0	100
	235.8	43.0	24.2	15.9	10.1	4.9	7.7	5.2	0.0	0.0	0.0	19.0
Pacific peoples	8	4	5	2	0	0	0	2	0	0	0	21
	155.1	20.9	21.2	9.2	0.0	0.0	0.0	9.2	0.0	0.0	0.0	10.5
Other	2	3	5	0	2	0	1	3	0	0	0	16
	53.8	21.4	27.2	0.0	7.1	0.0	2.2	7.9	0.0	0.0	0.0	6.5
Unknown	19	29	48	10	6	7	8	5	3	3	6	144
Total	127	235	288	134	36	44	72	61	35	21	17	1071
	232.4	108.7	100.7	46.1	13.6	9.0	12.5	11.4	8.4	7.4	5.3	28.7

Number of cases
Rate per 100 000

Pertussis - crude and age-standardised rates by ethnicity, 2002

Ethnicity	Crude rate [95% CI]	Age-standardised rate* [95% CI]
European	30.2 [28.1, 32.3]	34.5 [32.1, 37]
Maori	19.0 [15.3, 22.7]	12.3 [9.9, 16.4]
Pacific peoples	10.5 [6.5, 16]	7.1 [4.3, 14.7]
Other	6.5 [3.7, 10.5]	6.1 [3.5, 12.5]
Total	28.7 [26.9, 30.4]	

* Directly standardised to the NZ population

From February 2002 the recommended immunisation schedule for pertussis was a primary course of DtaP-IPV at six weeks, three months and five months of age. A booster was recommended at 15 months with DtaP/Hib, and a further booster at four years of age with DtaP-IPV prior to school entry. During 2002, the EpiSurv Case Report Form captured information on up to four vaccine doses. The following table shows the number of doses of vaccine given to the 992 probable or confirmed notified cases in each relevant age group. Vaccination information was recorded for 789(79.6%) of cases. Of these, 666 cases also had the number of doses recorded. A total of 447 cases were either 'fully vaccinated'[a] or in receipt of three or more doses of pertussis vaccine. Across all age groups, the proportion of cases vaccinated was therefore approximately 447/666 or 67%. In the '5 to 14 months' age group the proportion of cases (among those for whom the number of doses was recorded) in receipt of at least three doses of vaccine was 19/42 or 45.2%; in the '15 months to 4 years' age group it was 115/170 or 67.6% and in the '5+ years' age group it was 291/385 or 75.6%.

To interpret these data fully it would be useful to calculate vaccine efficacy, as has been carried out in analyses of previous pertussis epidemics in New Zealand. [23]

Age and vaccination status of 'probable' or 'confirmed' pertussis notifications, 2002

Age group[3]	Total Cases[1]	Vaccination status[2]						
		One dose	Two doses	Three doses	Four doses	Vaccinated (no dose info)	Not vaccinated	Unknown status
0-6 weeks	14	(0)	(0)	(0)	(0)	0	11	3
6 wks-2 mths	42	13	(0)	(0)	(0)	0	22	7
3-4 months	23	8	8	(1)	(0)	0	6	0
5-14 months	49	5	3	19	(0)	1	15	6
15 mths-4 yrs	196	0	3	18	97	13	52	13
5+ years	667	14	14	134	157	109	66	173
Total	**991**	**40**	**28**	**172**	**254**	**123**	**172**	**202**

1 This calculation was based only on cases assigned either 'probable' or 'confirmed' status in EpiSurv.
2 Bracketed numbers indicate cases ineligible for vaccination
3 One case had unknown age.

[a] Cases are termed 'fully vaccinated' if they received all vaccinations they were eligible for.

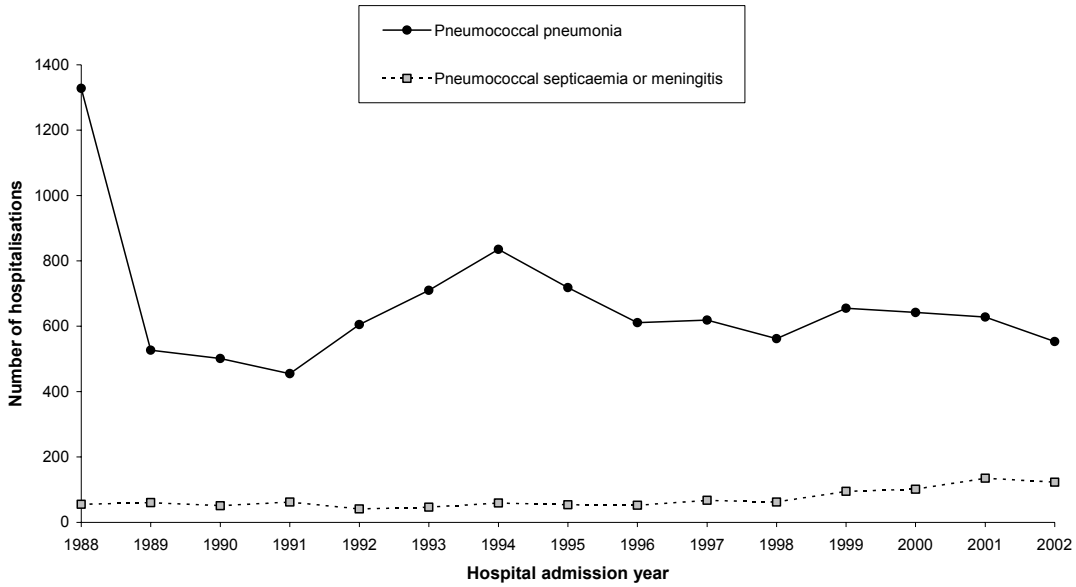
New Zealand's pertussis vaccination programme appears relatively ineffective at controlling this disease. Epidemics are continuing to occur at 4-5 year intervals, and the rate of disease during inter-epidemic periods appears to be increasing, based on notification and hospitalisation data for 2002. A major aim of vaccination is to prevent infection in young children, particularly those less than 6 months of age who are particularly vulnerable. The high rate of disease in this age group, and the death of an infant in 2002, confirm that this goal is not being fully achieved. Control of pertussis depends on increasing appropriate vaccine coverage [24,25] and on-time vaccination. [26]

Pneumococcal disease

Pneumococcal disease is not notifiable, though hospital discharge data provide an indication of disease rates. The ESR Streptococcal Reference Laboratory also collects the causative organism, *Streptococcus pneumonia*, for subtyping and antimicrobial resistance testing. An isolate is not received from every disease case, so laboratory-reported cases underestimate the incidence of this disease in New Zealand

Hospital discharge data for 2002 recorded 553 hospitalisations due to pneumococcal pneumonia (ICD9 code 481), compared to 628 the year before. In 2002 there were also 123 hospitalisations with a primary diagnosis of either pneumococcal septicaemia (ICD9 code 038.2) or pneumococcal meningitis (ICD9 code 320.1). The following graph shows pneumococcal disease hospitalisations each year since 1988.

Pneumococcal disease hospitalisations by year, 1988 - 2002



Serogrouping and serotyping of *Streptococcus pneumoniae* is undertaken to monitor the types causing invasive disease and the likely coverage by licensed or trial vaccines. Isolates were received from 312 cases of invasive pneumococcal disease in 2002 compared with 445 in

2001. The 23-valent vaccine, used only in adults, contains the following capsular antigen types: 1, 2, 3, 4, 5, 6B, 7F, 8, 9N, 9V, 10A, 11A, 12F, 14, 15B, 17F, 18C, 19A, 19F, 20, 22F, 23F and 33. Among the pneumococci derived from cases older than 14 years of age, 91.5% (115/129) belonged to serogroups/serotypes covered by the 23-valent vaccine.

Conjugate vaccines developed for paediatric use deliver seven, nine or eleven capsular antigens. The seven-valent vaccine contains capsular types 4, 6B, 9V, 14, 18C, 19F and 23F; the nine-valent vaccine additionally contains types 1 and 5; and the eleven-valent vaccine additionally contains types 3 and 7F. In 2002, invasive disease isolates were received from 183 children under 15 years of age of which 160 were under 5 years of age. By comparing the serotypes causing disease against the three conjugate vaccines, seven-valent, nine-valent, and eleven-valent, it was predicted that 88.1 % (141/160), 88.7% (142/160), or 88.7% (142/160) respectively of pneumococcal disease in those under 5 years may have been prevented by use of these vaccines.

There are now effective vaccines available against adult and paediatric pneumococcal disease. Improved disease surveillance would help describe the current epidemiology of this disease and also help measure the effectiveness of vaccination as it is introduced. In Australia, for example, invasive pneumococcal disease (IPD) is now notified to the National Notifiable Diseases Surveillance System. [27]

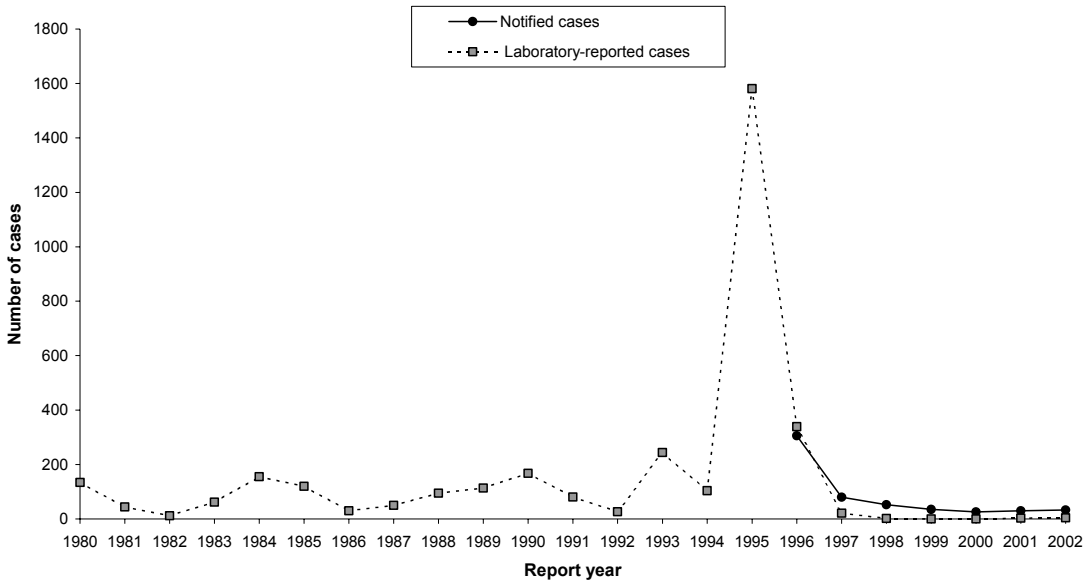
Polio

The last case of wild-type poliovirus infection occurred in New Zealand in 1962 and an imported case was diagnosed in 1976. Cases of vaccine-associated paralytic poliomyelitis (VAPP) occurred in 1970, 1977, 1990 and 1998.[28] New Zealand carried out intensified surveillance of cases of acute flaccid paralysis (AFP) as a component of the certification of New Zealand's polio-free status, which is required as part of the global poliomyelitis eradication programme. In 2002 and 2001 the incidence rate of AFP was 1.3 per 100 000 children under 15 years.

Rubella

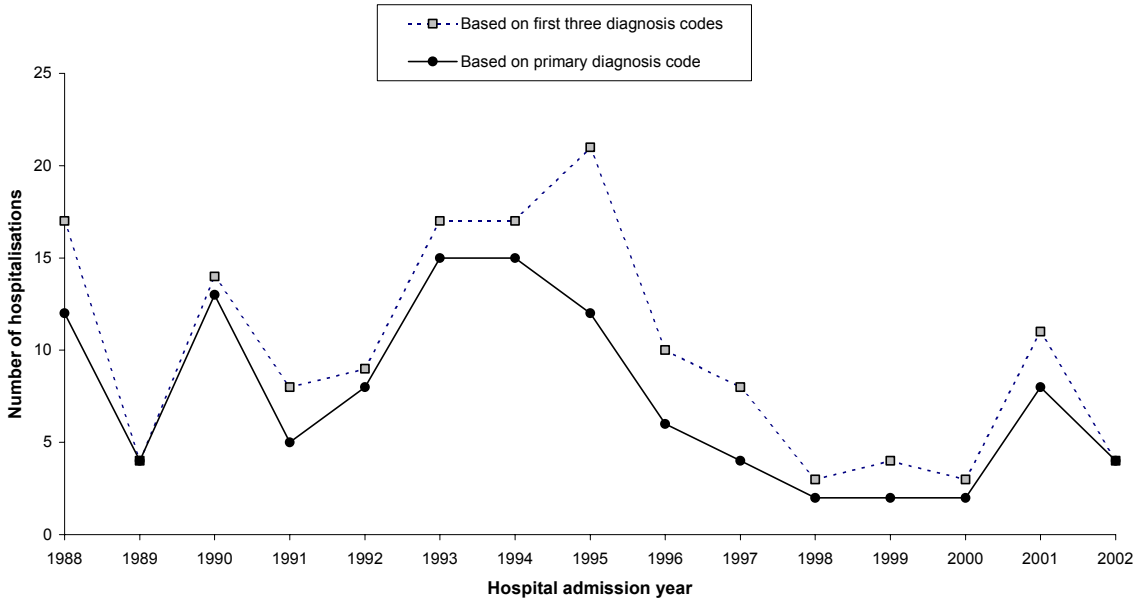
In 2002 there were 33 notifications, and four laboratory-reported cases of rubella. Matching of data indicated that one case was both notified and laboratory-reported. The 2002 notification rate of 0.9 per 100 000 (95% CI: 0.6-1.2) was similar to the 2001 rate of 0.8 per 100 000. Rubella has been notifiable since June 1996. The following graph shows notified and/or laboratory-reported cases each year since 1980.

Rubella notifications and laboratory-reported cases by year, 1980 - 2002



Of the 31 notified cases in 2002 for whom hospitalisation status was recorded on EpiSurv, one (3.2%) was hospitalised. According to hospital discharge data the number of rubella hospitalisations (ICD9 code 056) during 2002 totalled four. The following graph shows the number of hospitalisations each year since 1988.

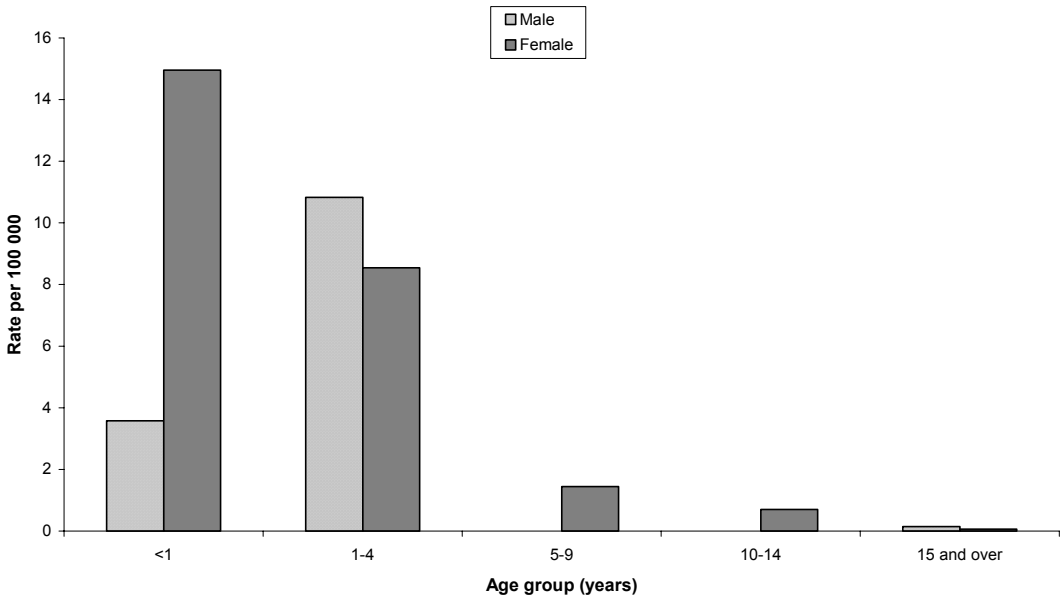
Rubella hospitalisations by year, 1988 - 2002



Hawke's Bay DHB notified the greatest number (11 or 33.3%) of rubella cases in 2002, although none were laboratory-confirmed and none reported contact with a laboratory-confirmed case. Age-specific rates were highest in the '1 to 4 years' and the 'less than one

year' age groups, with rates of 10.2 and 9.2 per 100 000, respectively. The following bar chart compares notification rates by age and sex.

Rubella notification rates, 2002
by age and sex



Of the 27 (New Zealand) notified cases for whom travel information was recorded, none indicated overseas travel. The majority (87.5%) of notified cases was of European ethnicity.

Vaccination status was recorded for 31 cases. Of these, 17 (54.8%) had received at least one dose of MMR vaccine. The table below shows vaccination status by age group.

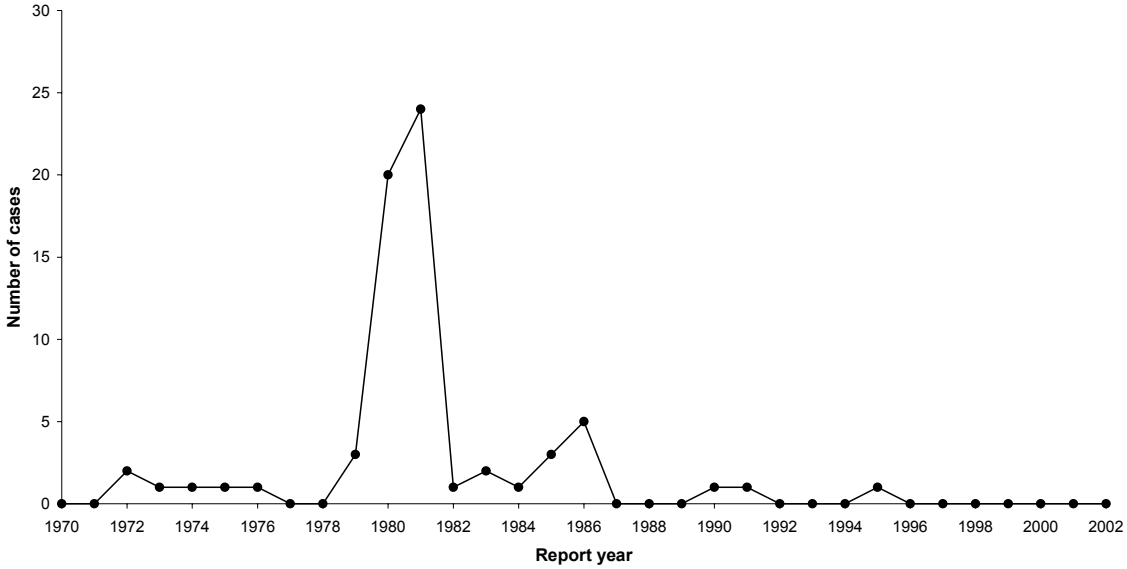
Age and vaccination status of rubella notifications, 2002

Age group	Total cases	Vaccination Status					
		1 dose	2 doses	3 doses[1]	Vaccinated (no dose info)	Not vaccinated	Vaccination status unknown
< 15 mths	7	0	0	1	0	6	0
15 mths - 4yrs	20	11	1	1	0	7	0
5 - 10 yrs	2	1	1	0	0	0	0
11+ yrs	4	1	0	0	0	1	2
Total	**33**	**13**	**2**	**2**	**0**	**14**	**2**

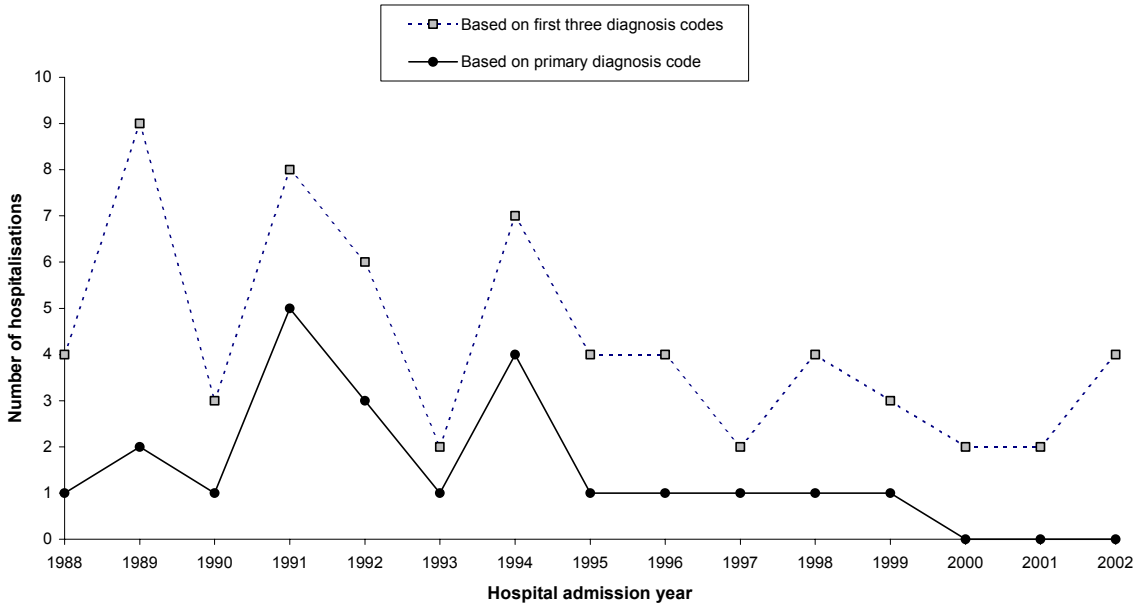
[1] There are assumed to be coding errors among the data, although this has not been verified.

During 2002 there were no cases of congenital rubella notified on EpiSurv or to the New Zealand Paediatric Surveillance Unit. There were also no hospitalisations recorded with a primary diagnosis of congenital rubella. The following graphs illustrate (i) the number of notified cases of congenital rubella since 1970, and (ii) congenital rubella hospitalisations (ICD9 code 771.0) since 1988.

Notified cases of congenital rubella syndrome, 1970 - 2002



Congenital rubella hospitalisations by year, 1988 - 2002

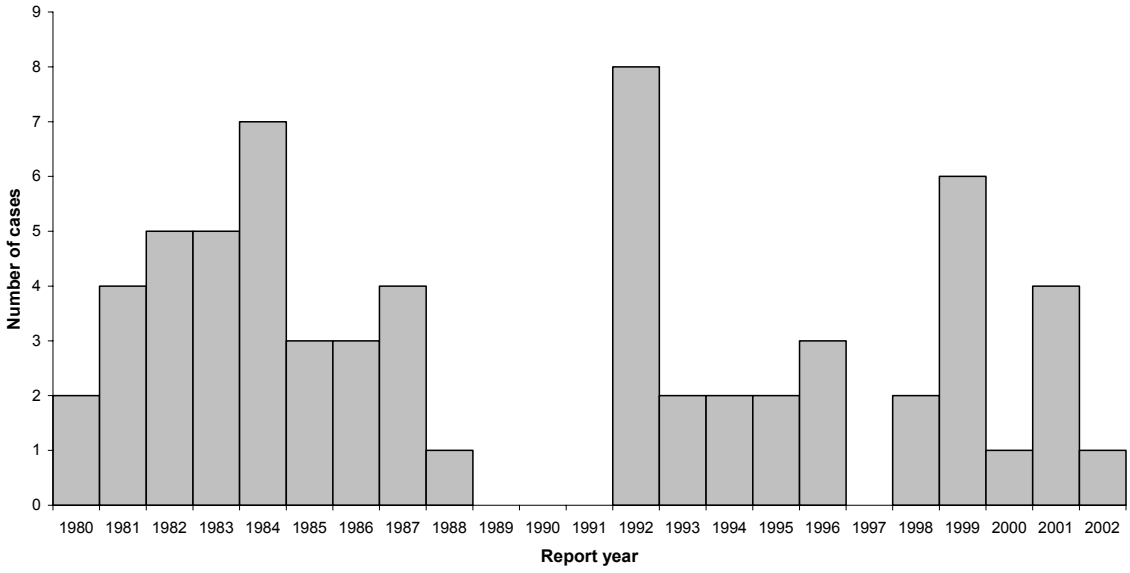


Surveillance data give a relatively incomplete picture of rubella as the disease only became notifiable in 1996 and rarely results in hospital admission. Overall, these data suggest that the incidence of rubella has declined since the last national epidemic in 1995. It is also reassuring that there have not been any cases of congenital rubella syndrome reported to the Paediatric Surveillance Unit since 1998, when a nine-year-old case of Samoan ethnicity was notified.

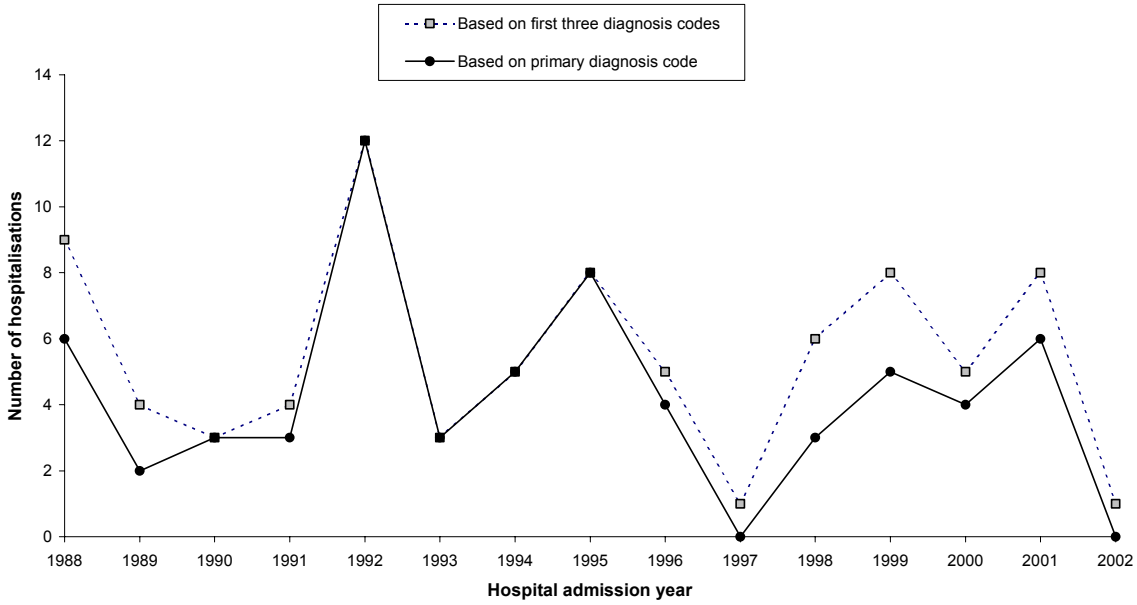
Tetanus

One probable case of tetanus was notified in 2002 from South Canterbury DHB. The case was a European female aged over 70 years who had injured her hand while shovelling horse manure a week prior to developing pain and jaw spasms. The case was hospitalised. The following graphs show (i) the number of cases notified each year since 1980, and (ii) the hospitalisations due to tetanus each year since 1988.

Tetanus notifications by year, 1980 - 2002



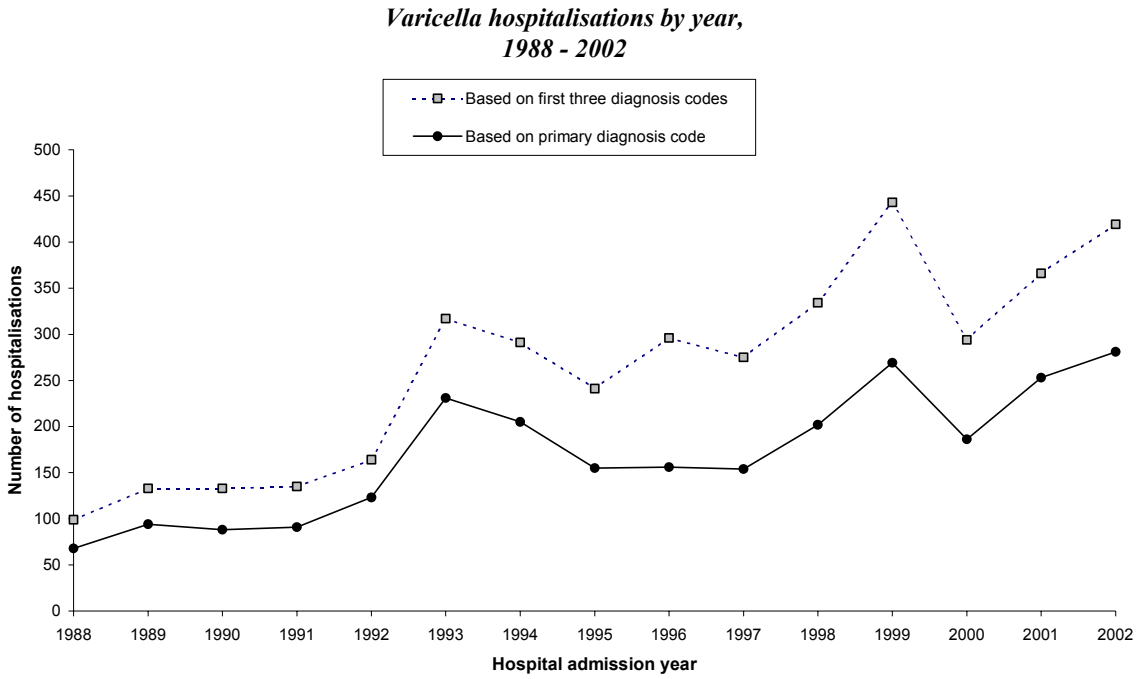
Tetanus hospitalisations by year, 1988 - 2002



These data confirm the sustained downward trend in the incidence of tetanus in New Zealand.[29] This disease is largely confined to older New Zealanders, particularly women, who have relatively less protection from tetanus vaccination.

Varicella

Varicella (chickenpox) is not currently notifiable in New Zealand. Based on the primary diagnosis coding of hospital discharge data, the number of varicella hospitalisations in 2002 totalled 281. This was the highest annual hospitalisation total for the past 15 years. The following graph shows the hospitalisations (ICD9 code 052) each year since 1988.



This analysis of hospitalisation data for varicella extends that previously carried out for the Varicella Workshop in 1996 [30] and immunisation handbook.[15] Two varicella vaccines are currently licensed for use in New Zealand though varicella vaccine has no been added to the childood immunisation schedule.

Infectious respiratory diseases and diseases of close contact

This group of diseases have a number of features in common. They are transmitted by respiratory and physical contact, and they are associated with overcrowding. Disease risk is generally highest among Maori and Pacific populations. They include the bacterial infections of meningococcal disease, tuberculosis, rheumatic fever and cellulitis, as well as respiratory syncytial virus (RSV). They also overlap with the vaccine-preventable diseases (VPDs). Some infectious respiratory diseases are at least partly vaccine preventable, notably meningococcal disease and tuberculosis.

Adenoviruses

Surveillance of adenoviruses is based on isolates confirmed by New Zealand virus laboratories. Infection with these viruses may result in respiratory tract infections, febrile illness, pharyngitis, conjunctivitis, gastroenteritis, or less frequently, meningitis. Some specific types may cause epidemics resulting in severe illness, high hospitalisation rates and occasional fatalities. There were a total of 224 adenovirus isolations in 2002, compared with 216 in 2001.

The most frequently identified serotype in 2002 was adenovirus type 3 (Ad3) with 101 (45.1%) isolations. In comparison, there were just 21 (9.7%) Ad3 isolations in 2001, and 17 in 2000. Ad3 accounts for 13% of all adenovirus isolates typed and reported to WHO. It exhibits an epidemic appearance with a periodicity of 4-5 years. It is most frequently isolated from children below the age of four years. The patients, from whom Ad3 was isolated in 2002, ranged in age from 3 months to 69 years, with a median age of 13.5 years. The male to female ratio was 1.1:1. The patients' illnesses included febrile respiratory illness, pneumonia, conjunctivitis and gastroenteritis. Two encephalitis patients yielded Ad3 from their faeces. The following table shows the number of adenovirus isolations each year since 1993, for the most prevalent serotypes.

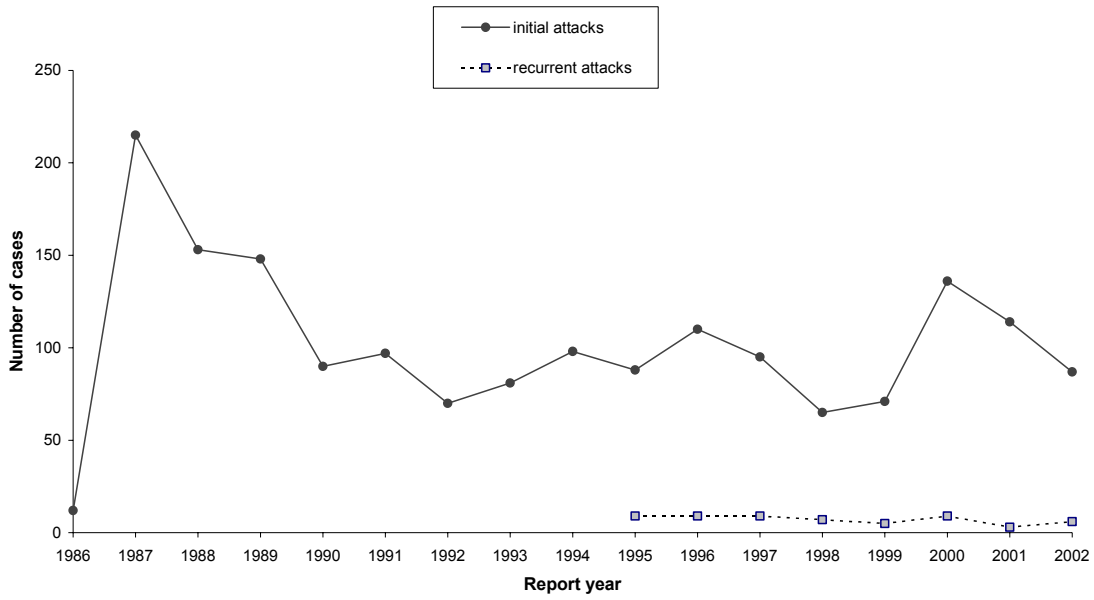
The number of adenovirus serotypes identified each year, 1993-2002

Serotype	Year									
	1993	**1994**	**1995**	**1996**	**1997**	**1998**	**1999**	**2000**	**2001**	**2002**
Adeno type 1	11	24	15	20	18	28	16	19	16	24
Adeno type 2	31	32	18	22	25	22	14	14	13	24
Adeno type 3	39	73	4	28	29	74	38	14	21	101
Adeno type 4	3	3	3	21	276	14	5	2	5	1
Adeno type 7	11	45	39	94	20	11	0	1	10	13
Adeno type 19	1	0	0	0	0	2	0	5	24	2
Adeno type 21	0	0	0	0	0	0	3	7	25	4
Other or untypable	44	28	38	49	47	35	33	50	102	55
Total adenovirus	140	205	117	234	415	186	109	112	216	224

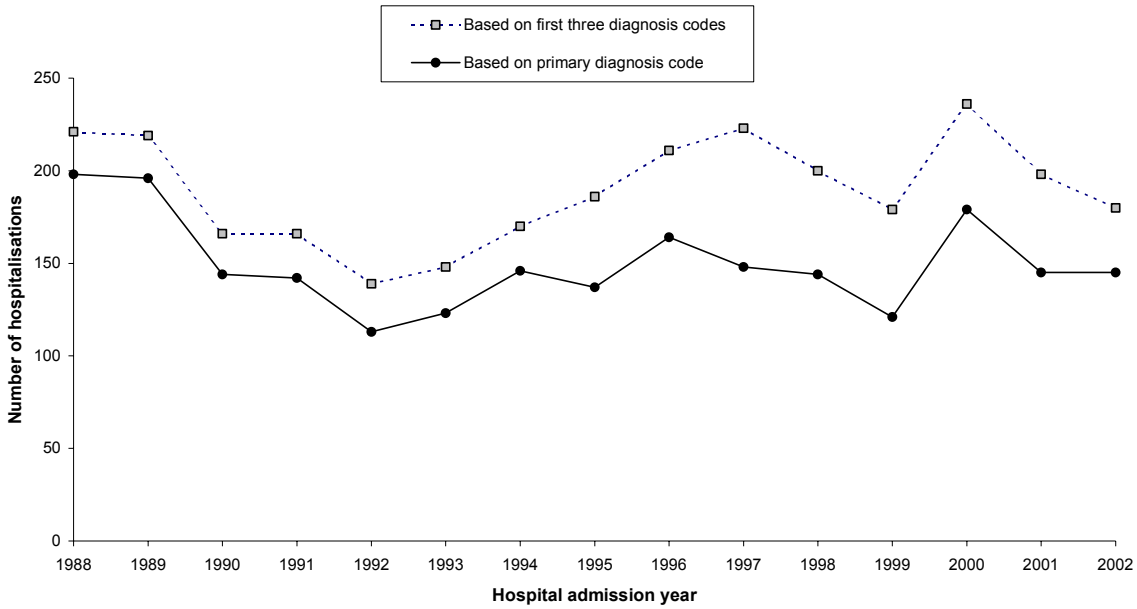
Acute and chronic rheuematic fever

A total of 87 initial attacks of rheumatic fever was notified in 2002, representing a rate of 2.3 cases per 100 000. Notifications have decreased steadily since 2000, when 136 cases were notified. However, due to the fact that many cases in 2000 and 2001 were notified long after the onset of illness, this apparent decline in incidence may be an artificial one. Six recurrent attacks of rheumatic fever were notified in 2002, compared to just three in 2001. Of the 33 acute cases in 2002 for whom hospitalisation status was recorded on EpiSurv, 31 (93.9%) were hospitalised. In contrast, hospital discharge data indicated a total of 145 hospitalisations due to acute rheumatic fever (ICD9 codes 390-392) during 2002. There were also 508 hospitalisations due to chronic rheumatic heart disease (ICD9 codes 393-398) in 2002, compared to 597 the year before. The following graphs show (i) the number of acute rheumatic fever cases and recurrent attacks notified each year since 1986, (ii) acute rheumatic fever hospitalisations each year since 1988, and (iii) chronic rheumatic heart disease hospitalisations each year since 1988.

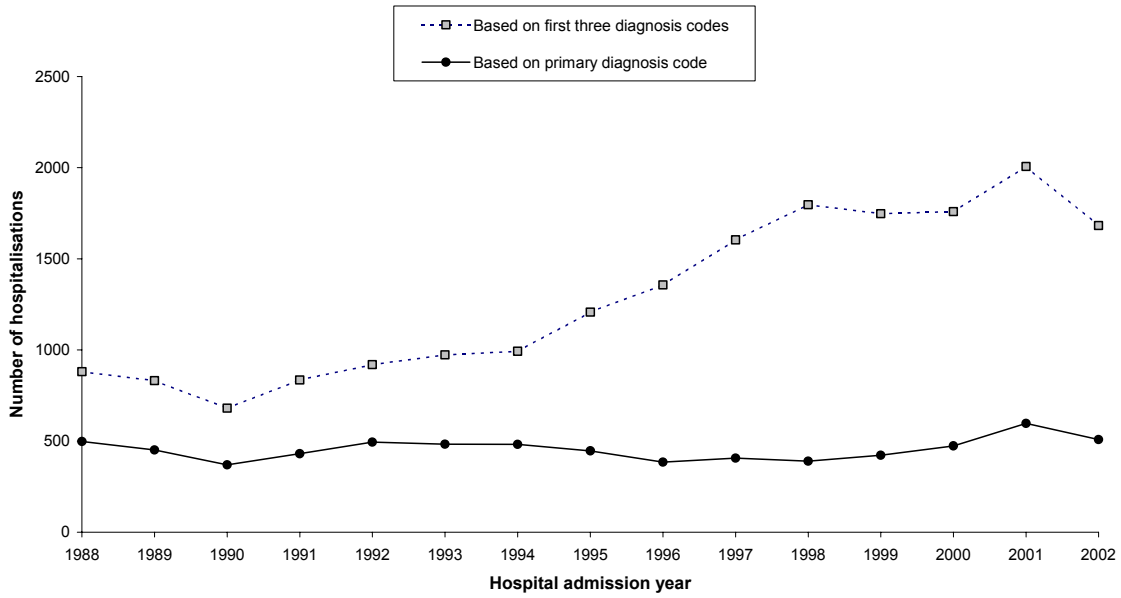
Rheumatic fever notifications by year, 1986 - 2002



Acute rheumatic fever hospitalisations by year, 1988 - 2002

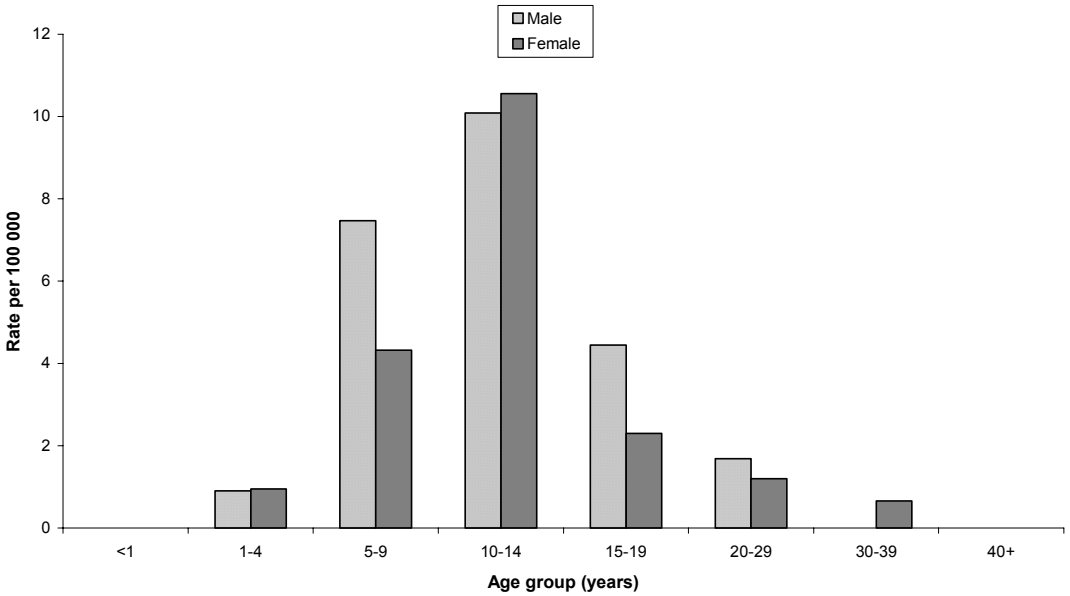


Chronic rheumatic heart disease hospitalisations by year, 1988 - 2002



Rates over three times the national average were reported from Counties Manukau DHB, with a rate of 8.3 cases per 100 000 (95% CI: 5.6-11.7). The majority (86%) of cases was aged 19 years or less. Age-specific rates were highest in the '10 to 14 years' age group, with a rate of 13.1 per 100 000 (95% CI: 9.2-17.9), and next highest in the '5 to 9 years' age group, with a rate of 7.3 (95% CI: 4.5-11.2). The following chart shows notification rates by age and sex, among the 67 cases for whom both age and sex were recorded.

Acute rheumatic fever notification rates, 2002 by age and sex



The following tables illustrate the age and ethnic distribution of cases. Notification rates were significantly higher in Pacific peoples (17.5 per 100 000) than in all other ethnic groups, and overall highest in Pacific peoples aged between 10 and 14 years (64.6 per 100 000). The rate of disease in Maori (6.8 per 100 000) was also significantly higher than the national crude rate of 2.3 per 100 000.

Acute rhuematic fever notifications and age-specific rates by ethnicity, 2002

Ethnicity	Age group in years											
	<1	**1-4**	**5-9**	**10-14**	**15-19**	**20-29**	**30-39**	**40-49**	**50-59**	**60-69**	**70+**	**Total**
European	0	0	1	1	1	0	0	0	0	0	0	3
	0.0	0.0	0.6	0.6	0.6	0.0	0.0	0.0	0.0	0.0	0.0	0.1
Maori	0	1	11	17	3	4	0	0	0	0	0	36
	0.0	1.9	16.6	27.0	6.1	4.9	0.0	0.0	0.0	0.0	0.0	6.8
Pacific peoples	0	1	9	14	7	3	0	1	0	0	0	35
	0.0	5.2	38.1	64.6	38.6	8.9	0.0	4.6	0.0	0.0	0.0	17.5
Other	0	0	0	1	0	0	1	0	0	0	0	2
	0.0	0.0	0.0	5.0	0.0	0.0	2.2	0.0	0.0	0.0	0.0	0.8
Unknown	0	1	0	5	2	2	1	0	0	0	0	11
Total	0	3	21	38	13	9	2	1	0	0	0	87
	0.0	1.4	7.3	13.1	4.9	1.8	0.3	0.2	0.0	0.0	0.0	2.3

Number of cases
Rate per 100 000

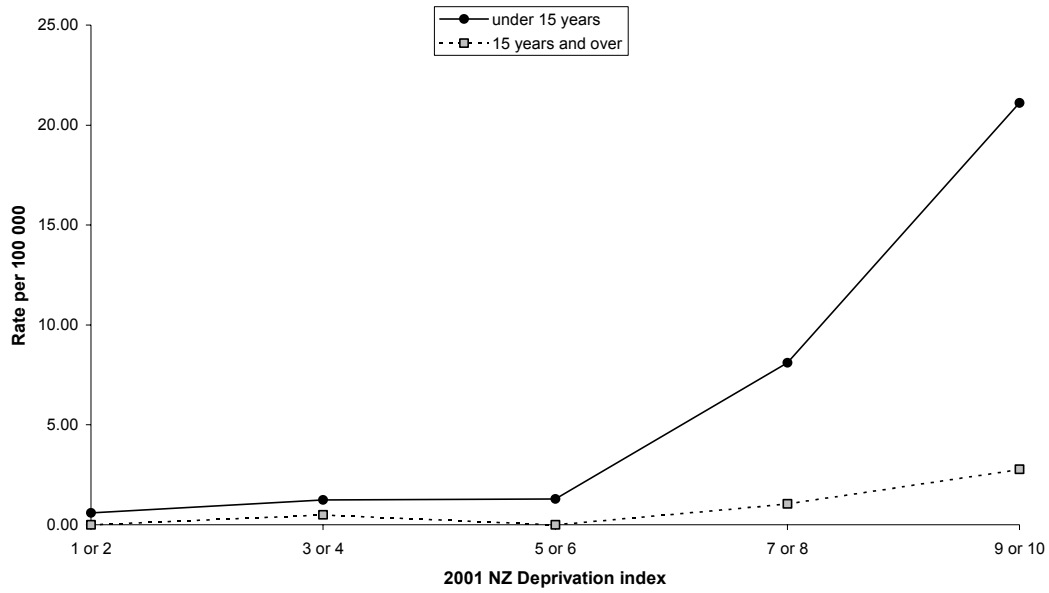
Acute rheumatic fever - crude and age-standardised rates by ethnicity, 2002

Ethnicity	Crude rate [95% CI]	Age-standardised rate* [95% CI]
European	0.1 [0, 0.3]	0.1 [0, 0.4]
Maori	6.8 [4.8, 9.5]	4.5 [3.1, 7.9]
Pacific peoples	17.5 [12.2, 24.3]	12.8 [8.9, 21]
Other	0.8 [0.1, 2.9]	0.7 [0.1, 6.4]
Total	2.3 [1.9, 2.9]	

* Directly standardised to the NZ population

The following graph illustrates the association between rates of acute rheumatic fever and deprivation in New Zealand, as measured on the 1 to 10 scale, with 1 representing the least deprived and 10 representing the most deprived score. Rates in both age groups (under 15 and over 15 years) increase with increasing deprivation, although the association is most marked in the younger age group.

Rates of acute rheumatic fever by age group and associated index of deprivation, 2002



Acute rheumatic fever (ARF) and its sequelae, chronic rheumatic heart disease (CRHD) remain amongst the most important infectious diseases in New Zealand, in terms of its public health impact.[31] Methods for primary prevention of ARF remain controversial.[32] Reductions in household crowding may offer potential to reduce the risk of this disease. Preventing recurrences of ARF with long-term penicillin prophylaxis is well established. Rheumatic fever registers have a role in supporting this intervention. [31]

Cellulitis

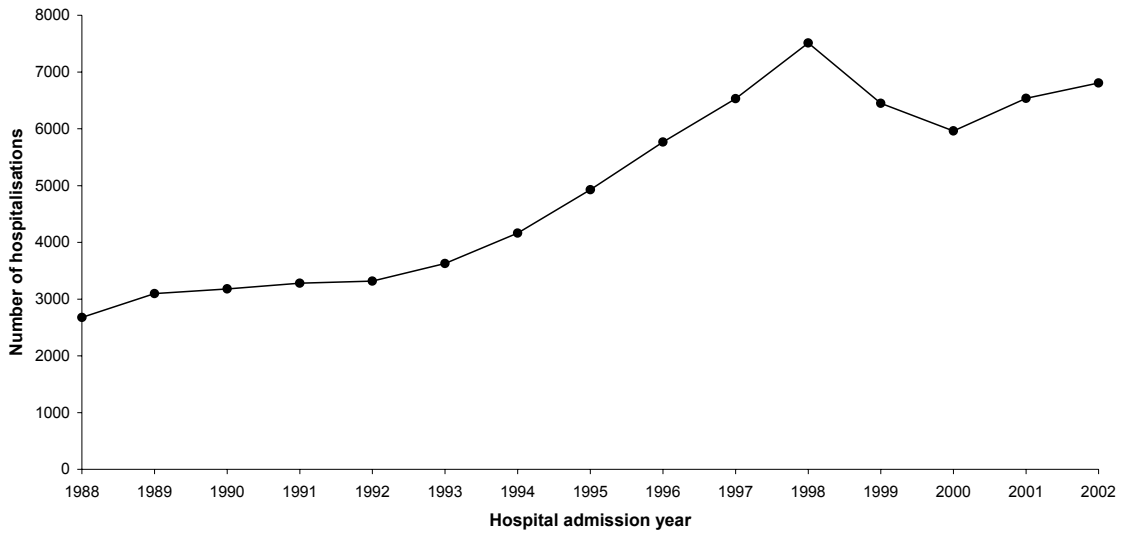
Infection of the skin and subcutaneous tissues are a major potentially preventable burden of human infection in New Zealand. These infections include:

- Streptococcal impetigo or pyoderma
- Streptococcal erysipelas
- Staphylococcal impetigo, folliculitis, furuncles, carbuncles
- Staphylococcal abscesses

None of these conditions are notifiable. Some microbiological surveillance is carried out as part of the MRSA surveillance. An indication of the incidence and distribution of this category of diseases can also be obtained from hospitalisation data.

There were 6808 hospitalisations due to cellulitis (ICD9 codes 681-682, 035, 684)) in 2002, of which 26 were explicitly recorded as due to streptococcal erysipelas (ICD9 code 035) and 119 due to streptococcal impetigo (ICD9 code 684). Hospitalisations due to cellulitis steadily increased from 1988 to 1998, they dropped in 1999 and 2000, but have been increasing again for the past two years.

Cellulitis hospitalisations by year, 1988 - 2002



Enteroviruses

Surveillance of enteroviruses is based on isolates confirmed by New Zealand virus laboratories. Enteroviruses include Coxsackie, Echovirus and Poliovirus groups. Infection with these viruses may result in respiratory tract infections, febrile illness, hand-foot-mouth disease, conjunctivitis, gastroenteritis, stomatitis, meningitis, carditis, paralysis, hepatitis, exanthema, or encephalitis. Some specific types may cause epidemics resulting in severe illness, high hospitalisation rates and occasional fatalities. There were a total of 219 enterovirus isolations in 2002, compared to 381 in 2001.

Echovirus type 13 (E13) was the most frequently identified serotype during both 2001 and 2002. Clinical syndromes associated with E13 include aseptic meningitis and possibly paralysis. During a 14-month period from February 2001 to April 2002, a total of 153 E13 isolates from 129 cases of mainly aseptic meningitis were identified. The first E13 isolation was from faecal and respiratory specimens off a 2-month-old Waikato boy. There was a long lag phase in the winter of 2001 with only two more E13 cases reported. The outbreak increased rapidly in the spring of 2001, peaked in the summer of 2001, and subsided in the autumn of 2002. The last E13 isolation was obtained in April 2002 from a CSF specimen from a 23-day-old infant boy in Wellington. Throughout the outbreak, cases ranged in age from 10 days to 39 years, with a median age of four years. Seventy-six cases were male and 53 were female. E13 is a relatively rare serotype of enterovirus with few reports in the literature. There were no E13 isolations recorded in New Zealand between 1975 to 2000. The 2001/2002 E13 outbreak was the largest recorded echovirus outbreak in New Zealand.

Echovirus type 7 isolations decreased significantly from 14 isolations in 2001 to two in 2002, and echovirus type 30 isolations decreased slightly from 32 isolations in 2001 to 23 in 2002. Eight isolations of Echovirus type 3 and seven isolations of Echovirus type 6 were reported in 2002, compared to just one echovirus type 3 and nil report of Echovirus type 6 in 2001. Aseptic meningitis is a syndrome frequently associated with most echovirus serotypes Paralysis, exanthema, encephalitis, and respiratory diseases have also been associated with echoviruses. The E33 outbreak that occurred in 2000 was confined to that year, and there were no further isolations of this organism in 2001 and 2002.[33]

A total of 14 Coxsackie B isolations were reported in 2002 compared to 26 the previous year. Of these, 7 (50%) were identified as Coxsackie B type 4, compared to 6 (23.1%) isolations in 2001. The clinical features of Coxsackie B cases include pleurodynia, aseptic meningitis, meningoencephalitis, pericarditis, myocarditis, respiratory illness and febrile illness. The following table shows the trend in enterovirus isolations for selected serotypes, over the last decade.

The number of enterovirus serotypes identified each year, 1993-2002

Serotype	Year									
	1993	**1994**	**1995**	**1996**	**1997**	**1998**	**1999**	**2000**	**2001**	**2002**
Echo 3	0	25	1	0	0	0	3	1	1	8
Echo 6	5	65	5	0	0	0	2	0	0	7
Echo 7	13	2	0	13	1	2	1	10	14	2
Echo 9	1	0	26	7	0	0	9	1	0	3
Echo 11	71	11	2	0	0	15	5	5	2	0
Echo 13	0	0	0	0	0	0	0	0	121	32
Echo 17	3	0	1	1	17	1	0	0	0	0
Echo 20	0	0	0	14	0	0	0	0	0	0
Echo 30	5	23	31	10	0	4	2	12	32	23
Echo 33	0	0	0	0	0	0	0	101	0	0
Other Echovirus serotypes	8	5	7	8	2	4	1	4	5	2
Total Echovirus	106	131	73	53	20	26	23	134	175	77
Coxsackie A	46	7	18	9	89	14	15	10	18	5
Coxsackie B	9	57	28	9	25	16	11	13	26	14

Group A streptococcal disease

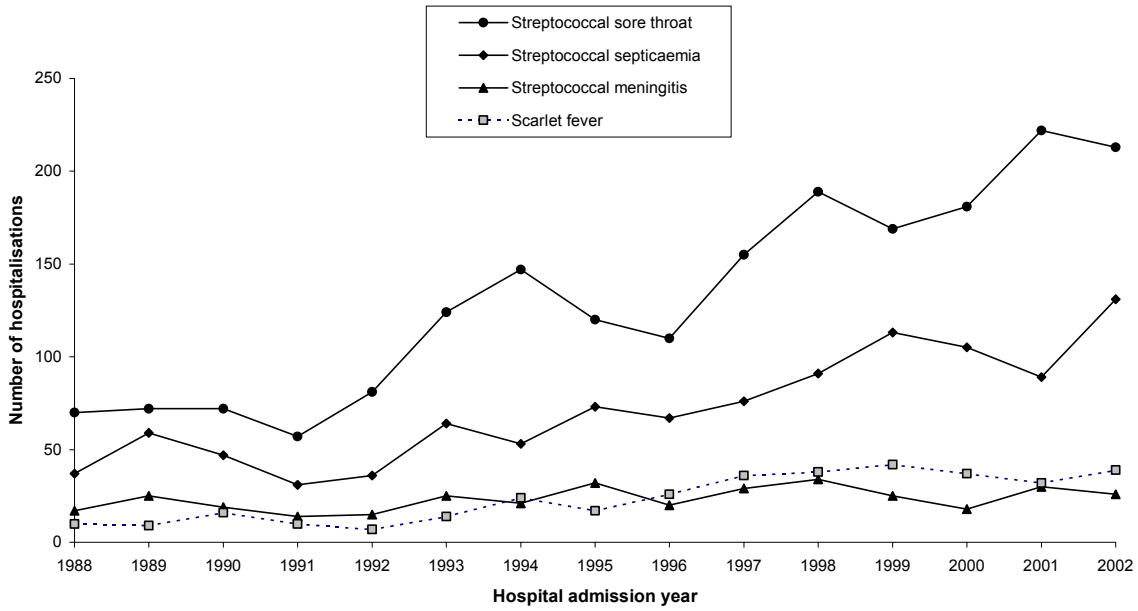
In addition to rheumatic fever, there are several other important diseases caused by infection with *Streptococcus pyogenes* (Group A Streptococcus). These diseases include:

- Invasive streptococcal infections – pneumonia, septicaemia, meningitis
- Streptococcal sore throat
- Scarlet fever
- Post-streptococcal acute glomerulonephritis
- Toxic shock syndrome and necrotising fasciitis

Except for acute rheumatic fever, Group A Streptococcal diseases are not notifiable. Microbiological surveillance depends on the voluntary referral of isolates to the Streptococcal Reference Laboratory at ESR. Hospitalisation data also give an indication of the incidence and distribution of the main diseases caused by this organism.

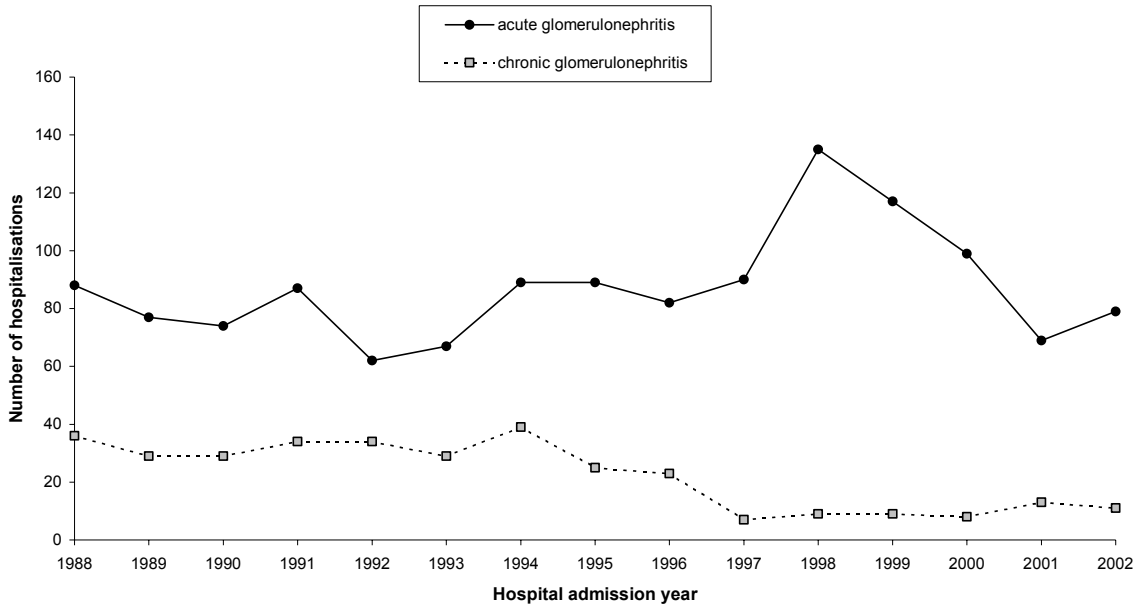
Hospitalisations due to streptococcal sore throat (ICD9 code 034.0) have increased over the last decade. There were 213 hospitalisations due to streptococcal sore throat in 2002, compared to just 57 in 1991. Hospitalisations of streptococcal septicaemia (ICD9 code 038.0) also increased significantly from 89 cases in 2001 to 131 cases in 2002, and hospitalisations due to acute glomerulonephritis[a] (ICD9 code 580) increased slightly from 69 cases in 2001 to 79 in 2002. In 2002 there were 39 hospitalisations due to scarlet fever (ICD9 code 034.1), and 26 due to streptococcal meningitis (ICD9 code 320.2). The following graphs show hospitalisations for selected Group A Streptococcal diseases each year since 1988.

Streptococcal hospitalisations by year, 1988 to 2002



[a] Not all 'acute glomerulonephritis' hospitalisations are necessarily due to post-streptococcal acute glomerulonephritis .

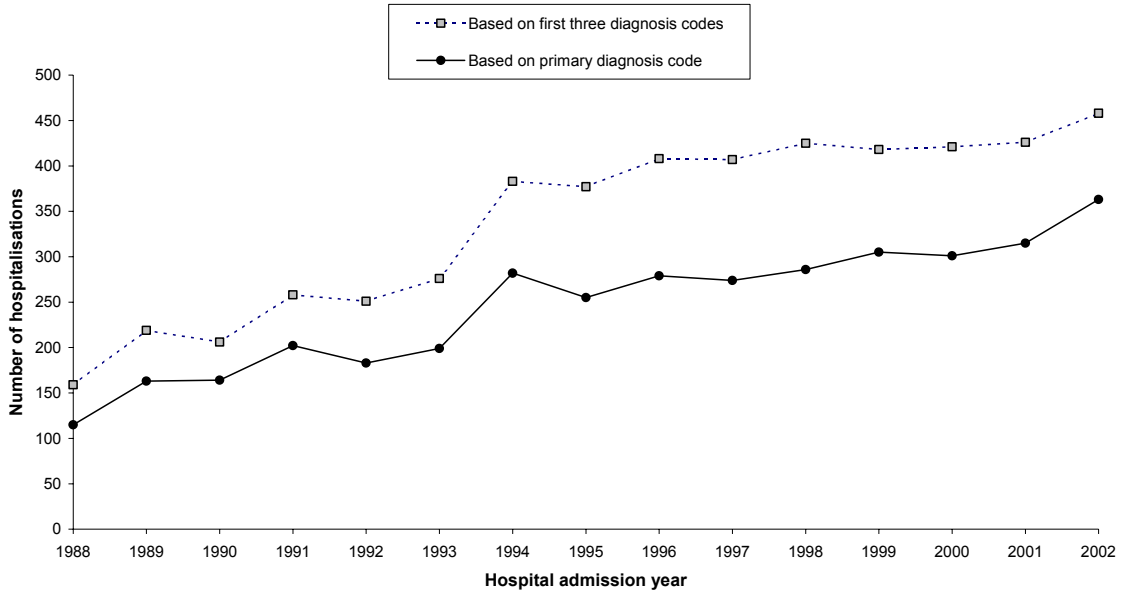
Glomerulonephritis hospitalisations by year, 1988 to 2002



Of the 203 Group A streptococcal isolates received in 2002, 170 were from cases of invasive disease. This represented an increase from 2001 when 120 invasive disease isolates were referred. Forty distinct M/*emm* types occurred among the 108 blood isolates, indicating the wide range of types which can cause significant infection in New Zealand. The most common M/*emm* types were 1, 3, 28, 75, 81, and 89. This was consistent with previous years. Seven isolates were identified as from cases of necrotising fasciitis. No one M/*emm* type predominated among these. Four isolates were from scarlet fever, and all four were M/*emm* types known to cause scarlet fever (types 1,12, 22, and 49). Only one isolate, an M/*emm* 4, was associated with post-streptococcal acute glomerulonephritis. Three isolates were from cases recovered from the throats of cases defined as rheumatic fever.

There were 363 hospitalisations due to major puerperal infection (ICD9 code 670) in 2002. Such hospitalisations have been gradually increasing since 1988, as shown by the graph below. Puerperal sepsis is most commonly caused by group A streptococci. The 10 isolates from cases of puerperal sepsis referred to ESR were of varying M/*emm* type.

Major puerperal infection hospitalisations by year, 1988 - 2002

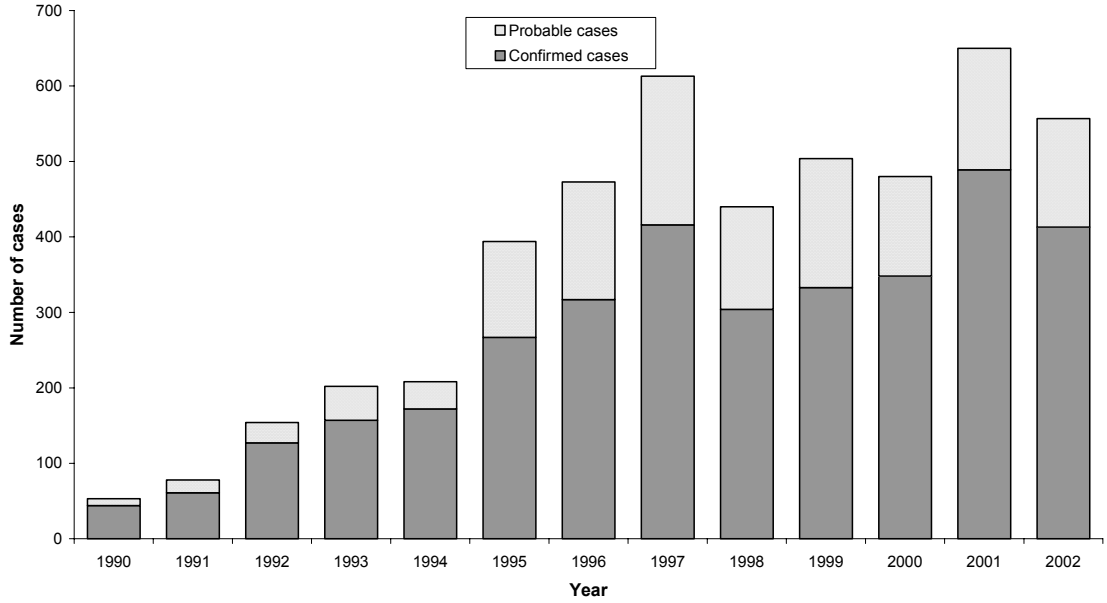


Meningococcal disease

A full description of the epidemiology of meningococcal disease in 2002 is contained in a separate report.[34]

The surveillance of meningococcal disease in New Zealand is based upon the rigorous matching and follow-up of all laboratory and notification data. A total of 557 cases of meningococcal disease was reported during 2002, representing a rate of 14.9 per 100 000 population. This rate is approximately ten times higher than the rate of 1.5 per 100 000 occurring in the immediate pre-epidemic years (1989-1990), and since the start of the current epidemic has been surpassed only by a rate of 17.4 in 2001 and 16.9 in 1997. Of the 557 cases, 413 (74.1%) were laboratory-confirmed. In comparison, 75.2% of cases in 2001 were confirmed - the highest proportion of cases to be confirmed since 1994, when the active promotion of pre-hospital administration of antibiotics reduced the chance of obtaining culture-positive meningococcal disease cases. The following chart shows the proportion of confirmed and probable cases each year since 1990.

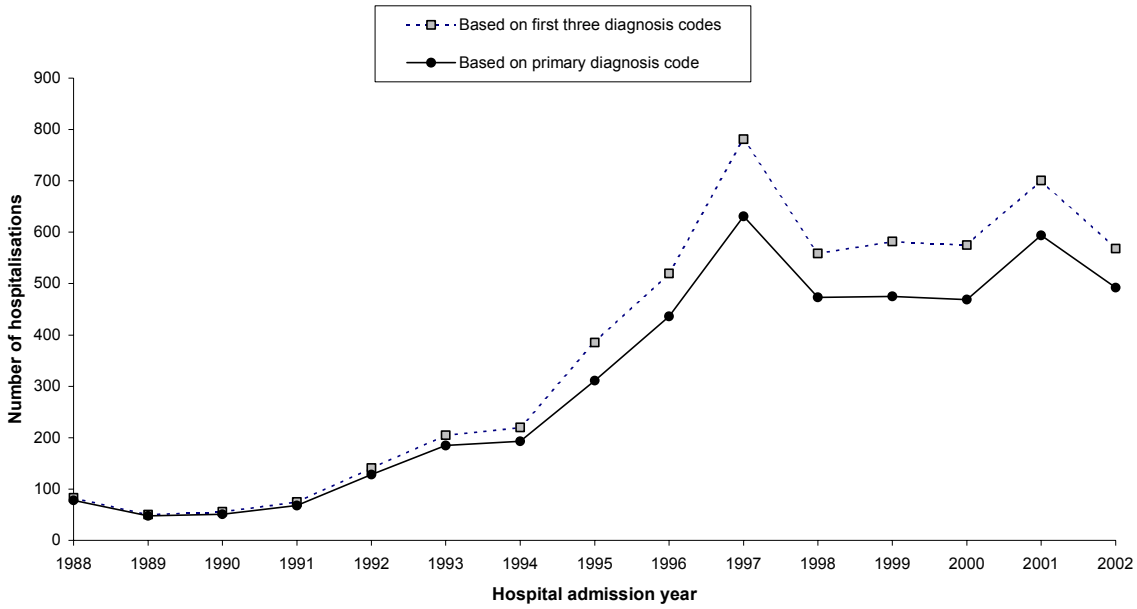
Confirmed and probable meningococcal disease cases, 1990-2002



There were 18 deaths due to meningococcal disease in 2002, equating to a case fatality rate of 3.2%, lower than the average case fatality rate of 4.4% between 1991 and 2001. The '40 years and over' age group experienced the highest case fatality rate of 12.2%, followed by teens aged between 15 and 19 years (6.1%) and infants under the age of one year (4.8%).

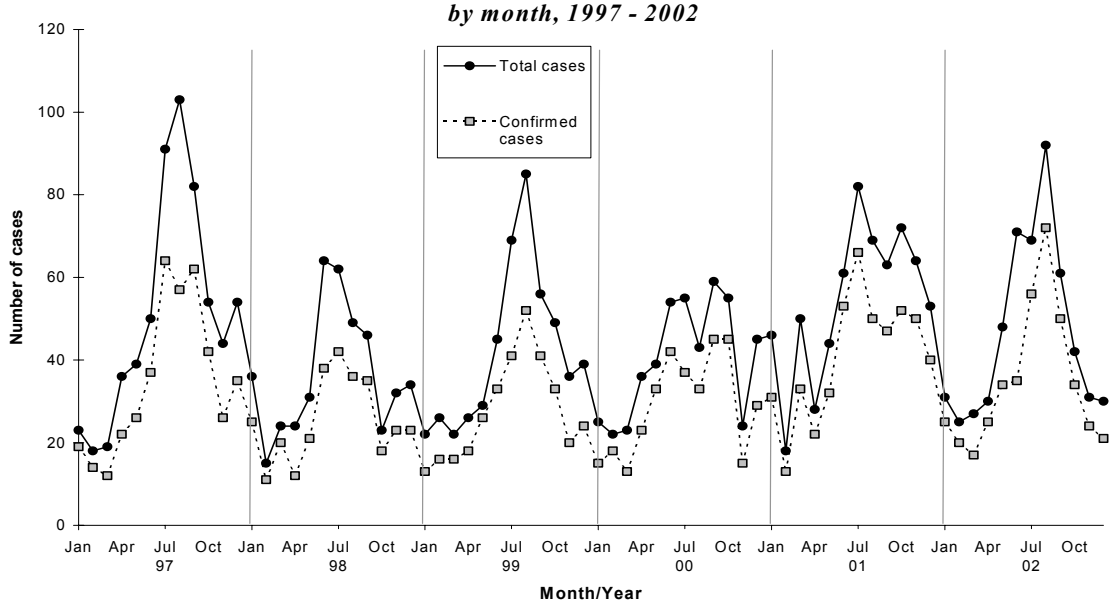
Information on hospitalisation was recorded for 545 (97.8%) meningococcal disease cases in 2002. A total of 531/545 (97.4%) cases were recorded as having been hospitalised. Of the 14 cases who were not hospitalised, two died before being admitted to hospital. In contrast, hospital discharge data indicated just 492 hospitalisations with a primary diagnosis of meningococcal disease (ICD9 code 036) during 2002. (Detailed results of case-to-case matching of notification and hospital discharge data are discussed in the section on surveillance data quality.) The following graph shows the number of hospitalisations recorded each year since 1988. Note that the peaks in 1997 and 2001 mirror the high incidence of meningococcal disease detected by public health surveillance during these years.

Meningococcal disease hospitalisations by year, 1988 - 2002



The following graph shows the number of notified cases and laboratory-confirmed cases each month since January 1997. The marked seasonality in meningococcal disease incidence continued in 2002 with 65.7% notified cases occurring in the winter and spring months (June through November). Although the peak number of cases reported in August 2002 (92 cases), exceeded all previous monthly totals since August 1997, incidence dropped more rapidly than usual during the succeeding months of October, November and December 2002.

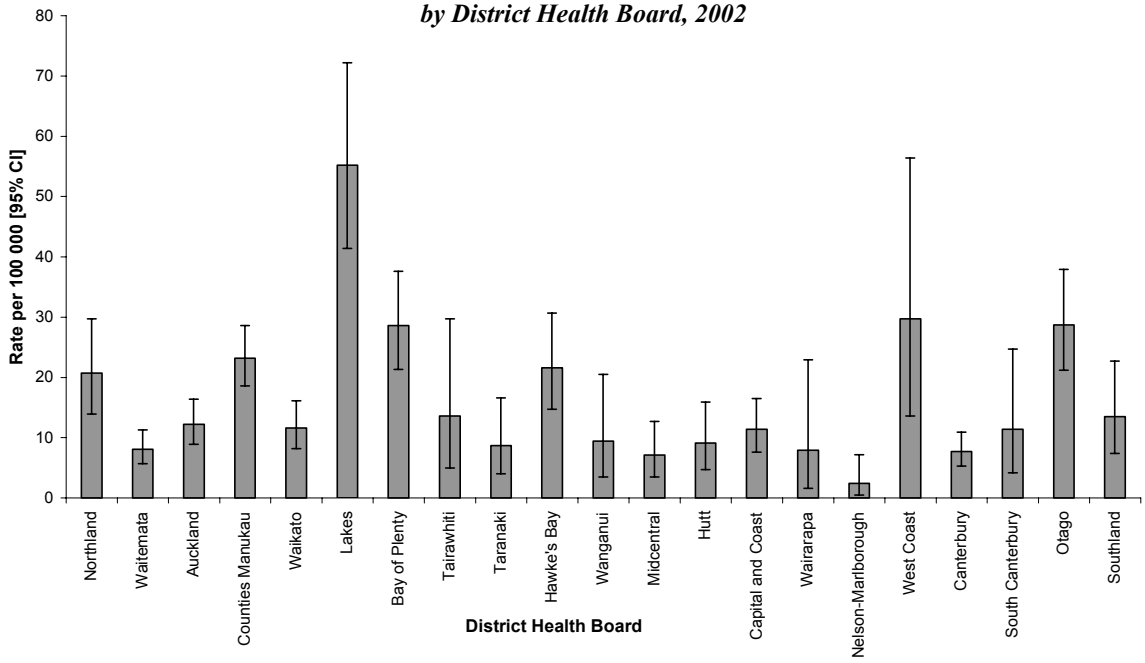
Meningococcal disease notifications and lab-confirmed cases by month, 1997 - 2002



The rate of meningococcal disease varied throughout the country in 2002, with the highest rate of 55.2 per 100 000 (95% CI: 41.4-72.2) being recorded in Lakes DHB. Other District Health Boards reporting rates significantly higher than the national average of 14.9 (95% CI: 13.7-16.1) included West Coast with a rate of 29.7 (95% CI: 13.6-56.4), Otago (28.7, 95% CI: 21.2-37.9), Bay of Plenty (28.6, 95% CI: 21.3-37.6), and Counties Manukau (23.2, 95% CI: 18.6-28.6).

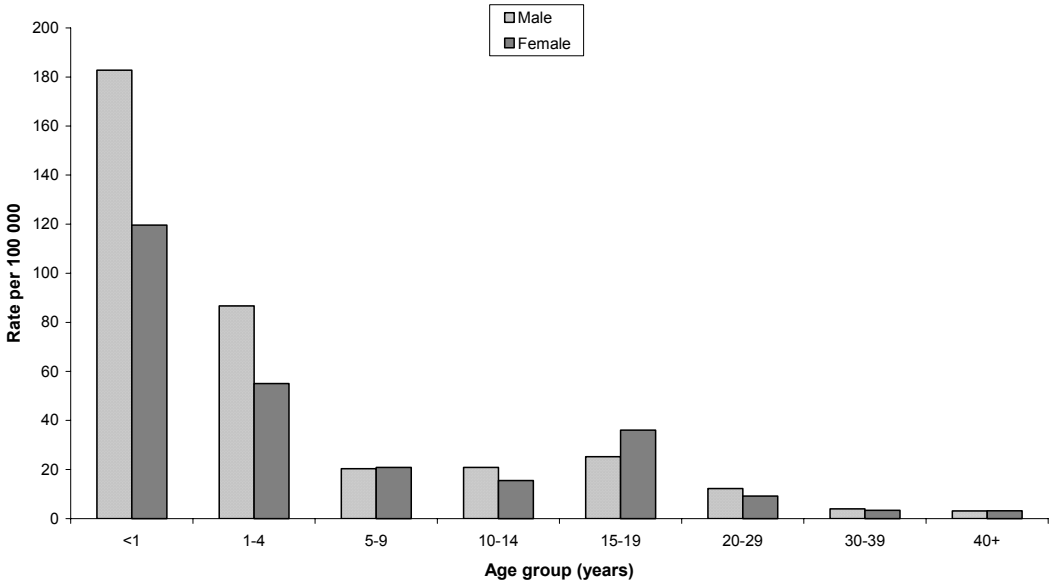
The rate among infants aged less than one year in Lakes DHB was 524.9 (95% CI: 226.6-1034.3). Age-specific rates in West Coast, Bay of Plenty and Counties Manukau DHBs were also highest in the 'less than one year' age group. However, in Otago DHB, rates were highest in the '15 to 19 years' age group. After age-standardisation, Lakes DHB still exhibited the highest incidence rate of meningococcal disease, followed by West Coast, Bay of Plenty, Otago, Northland and Counties Manukau DHBs, in that order. The following graph shows crude notification rates by District Health Board during 2002.

Meningococcal disease crude notification rates by District Health Board, 2002



The age group distribution of meningococcal disease cases has remained fairly constant throughout the epidemic, with the highest rates occurring in the 'less than one year' and the '1 to 4 years' age groups, with rates of 153.7 per 100 000 (95% CI: 122.6-190.3), and 71.2 (95% CI: 60.0-82.5), respectively. During 2002, the '15 to 19 years' age group also experienced a rate (30.9, 95% CI: 24.6-38.4) significantly higher than the national average. The following chart compares notification rates in 2002 by age and sex.

Meningococcal disease notification rates, 2002
by age and sex



The tables below illustrate the age and ethnic distribution of meningococcal disease in 2002. Compared to the rest of the population, rates of disease were significantly higher in Pacific and Maori ethnic groups. Crude notification rates were significantly higher for Pacific peoples than for Maori. The age-standardised rate for Pacific peoples was also higher than for the Maori ethnic group, although it is difficult to tell if this difference is statistically significant.

Meningococcal disease notifications and age-specific rates by ethnicity, 2002

Ethnicity	**Age group in years**											
	<1	**1-4**	**5-9**	**10-14**	**15-19**	**20-29**	**30-39**	**40-49**	**50-59**	**60-69**	**70+**	**Total**
European	23	53	29	22	58	38	17	15	16	4	5	280
	77.3	43.6	17.2	12.4	36.1	12.4	4.3	3.8	4.8	1.7	1.7	10.7
Maori	40	61	15	14	9	9	4	3	4	1	0	160
	285.9	113.9	22.7	22.3	18.2	10.9	5.2	5.2	12.0	5.1	0.0	30.4
Pacific peoples	19	35	11	13	9	3	0	0	0	0	0	90
	368.4	182.5	46.6	60.0	49.6	8.9	0.0	0.0	0.0	0.0	0.0	44.9
Other	1	4	4	3	4	3	0	0	0	0	0	19
	26.9	28.5	21.8	14.9	14.3	6.9	0.0	0.0	0.0	0.0	0.0	7.7
Unknown	1	1	1	2	2	0	0	1	0	0	0	8
Total	84	154	60	54	82	53	21	19	20	5	5	557
	153.7	71.2	21.0	18.6	30.9	10.9	3.6	3.5	4.8	1.8	1.6	14.9

Number of cases
Rate per 100 000

Meningococcal disease - crude and age-standardised rates by ethnicity, 2002

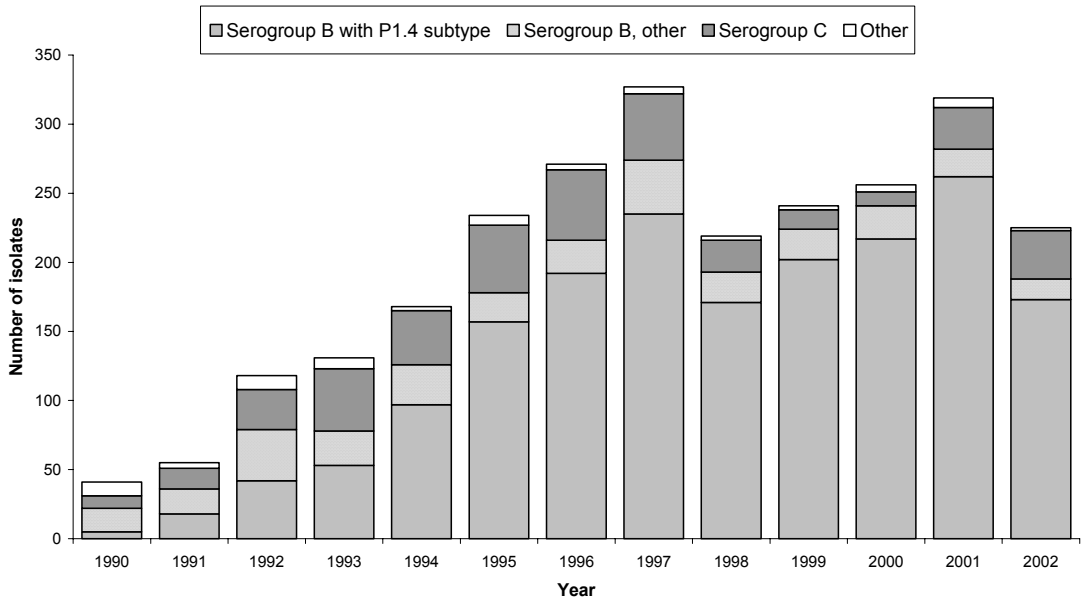
Ethnicity	Crude rate [95% CI]	Age-standardised rate* [95% CI]
European	10.7 [9.5, 12.0]	12.1 [10.7, 13.6]
Maori	30.4 [25.7, 35.1]	20.2 [17.0, 25.0]
Pacific peoples	44.9 [36.1, 55.2]	28.9 [23.2, 38.4]
Other	7.7 [4.6, 12.0]	6.8 [4.1, 13.2]
Total	14.9 [13.7, 16.1]	

* Directly standardised to the NZ population

The increase in disease rates since 1991 has largely been attributable to serogroup B meningococci expressing the PorA P1.7b,4 protein. Serogroup B disease, particularly that caused by the epidemic type, continued to dominate in 2002. Of the isolates obtained from cases 83.6% (188/225) were serogroup B, and of these 173 (92%) were the epidemic type.

In 2002, a total of 35 (15.5%) serogroup C isolates was identified throughout New Zealand, compared to 30 isolates in 2001, and 10 in 2000. Thirteen serogroup C isolates in 2002 were from the Otago District Health Board. One outbreak of serogroup C, accounting for six cases, occurred among school pupils in South Otago. There was also a small outbreak of serogroup C (2 cases) reported from West Coast DHB. Only one isolate each of W135 and Y was identified in 2002. The following graph shows the characteristics of meningococcal isolates typed at ESR during the period 1990 to 2002.

Meningococcal disease isolate serogroup and dominant subtype, 1990-2002



Confirmation of meningococcal disease using PCR was introduced in 1996 but the use of this laboratory test has been slow to be accepted. In 2002, 177 cases (31.8%) of meningococcal disease, not confirmed by isolation of a meningococcus, were confirmed by detection of meningococcal DNA from sterile site specimens. This is the highest percentage of cases confirmed by PCR to date. Of these, DNA was received at ESR for 172. Genotyping using the *siaD* PCR showed that 147 (85.5%) encoded the group B capsular polysaccharide and 10 (5.8%) the C polysaccharide. A further 15 were unable to be defined using the *siaD* PCR. Of those genotyping as group B, 84.4% (124/147) were shown to have DNA encoding the P1.7,4 PorA type. Combining the serotyping results on isolates (n= 225) and genotyping of DNA from PCR positive specimens able to be typed (n= 159), a total of 297 (173 +124) cases out of 384 (77.3%) were caused by meningococci with the P1.7b,4 PorA protein. This is marginally less than the proportion in 2001 when subtype P1.7b,4 was responsible for 80.5% (372/462) cases.

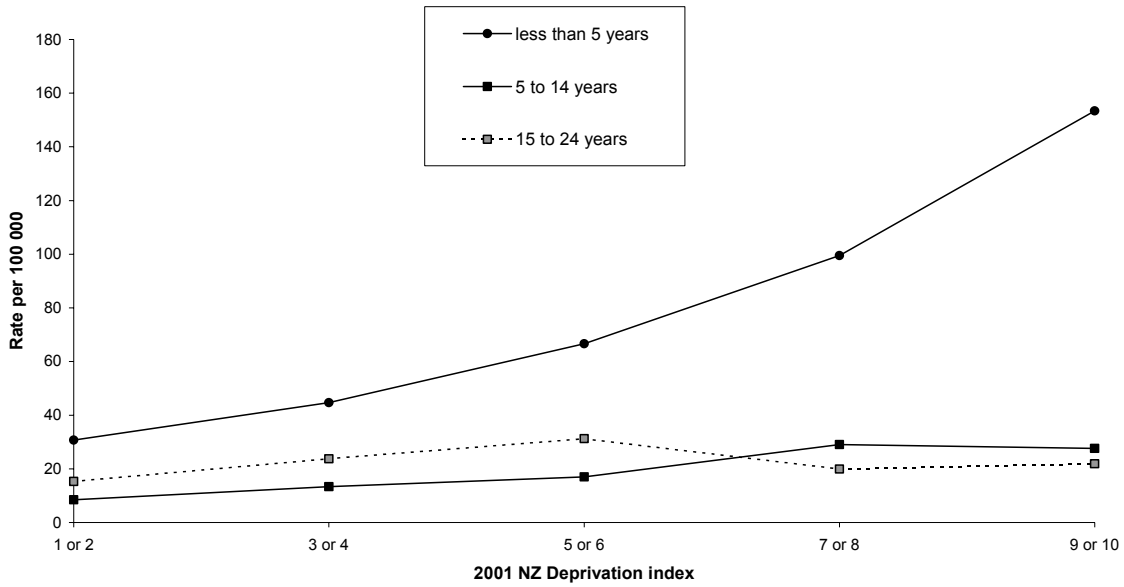
In 2002, an organism was isolated from only 31.3% of cases treated with antibiotics prior to sampling, compared with 45.0% of cases to whom no antibiotics were given. This difference was statistically significant and follows the trend shown between 1996 and 2001. These results illustrate the impact of pre-hospital antibiotic treatment.

The antimicrobial susceptibility of the 223 viable isolates received at ESR from cases of invasive disease in 2002 was tested. All isolates were susceptible to penicillin, ceftriaxone, rifampicin and ciprofloxacin. However, 8.1% (18/223) had reduced penicillin susceptibility, with MICs of 0.12-0.5 mg/L. Up until 2002, all meningococcal isolates with reduced penicillin susceptibility had MICs of 0.12 or 0.25 mg/L. In 2002, one isolate had a penicillin MIC of 0.5 mg/L.

The following graphs illustrate the association between rates of meningococcal disease and deprivation in New Zealand, as measured on the 1 to 10 scale, with 1 representing the least deprived and 10 representing the most deprived score. Note that analysis has only been performed on notified cases whose addresses geocoded to at least 'Street' level[a]. Rates in the 'less than 5 years' age group were found to increase rapidly with increasing deprivation. This association was less apparent in children aged between 5 and 14 years, and not at all apparent in young adults. This finding is consistent with results of the meningococcal disease case-control study which found a strong association between the risk of disease in children and household crowding.[35] Crowding is one of the components of the NZDep index.

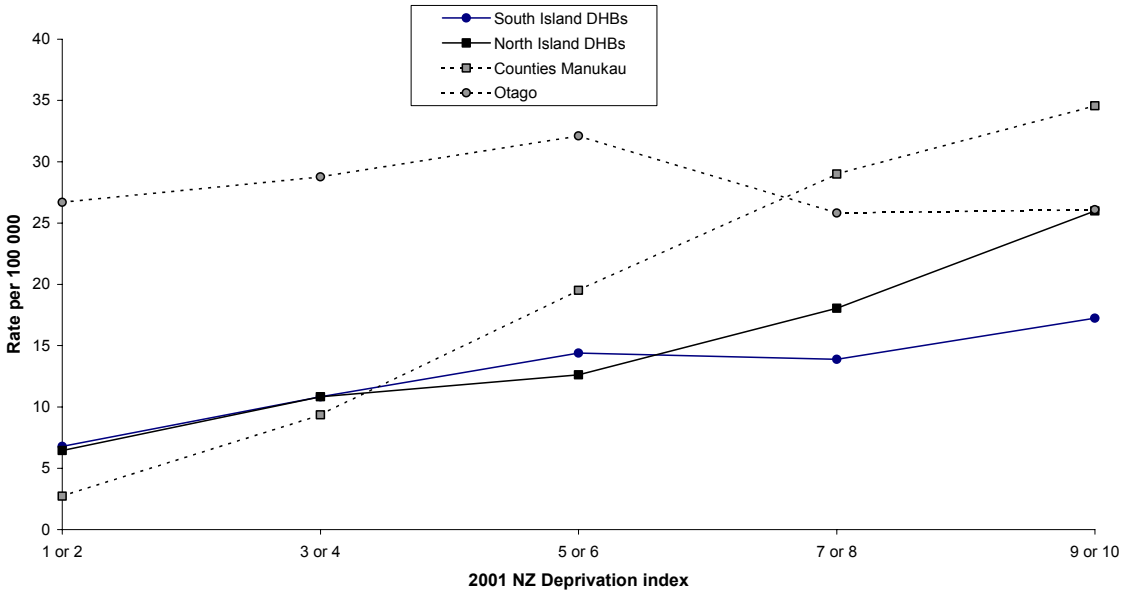
[a]Addresses for eight cases aged 0 to 4 years, three cases aged 5 to 14, and six cases aged 15 to 24, did not geocode to a sufficient level of accuracy.

Rates of meningococcal disease by age group and associated index of deprivation, 2002



The association between rate of disease and deprivation varied considerably throughout the country and was generally much more apparent among North Island than South Island District Health Boards. In Counties Manukau DHB, the rate rose rapidly with increasing deprivation. The following graph illustrates the trend for selected DHBs.

Rates of meningococcal disease in selected District Health Boards by associated index of deprivation, 2002

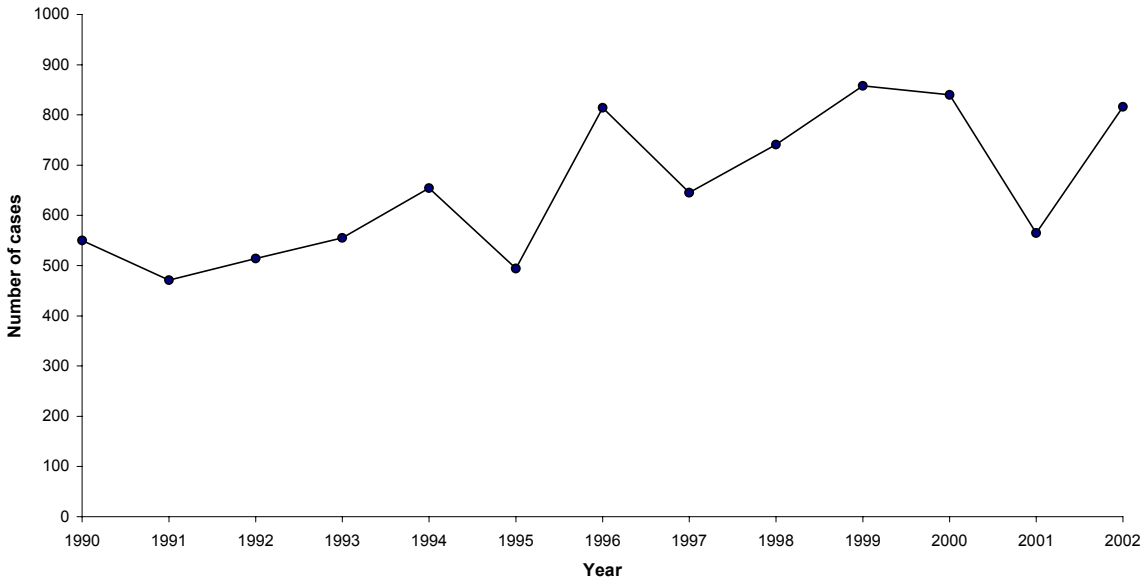


Meningococcal disease remains New Zealand's most important infectious disease problem in terms of its public health impact. The epidemiology of this epidemic has been well described elsewhere. [36,34] Controlling this disease will depend on measures to reduce household crowding and delivering an effective vaccine to the most vulnerable population groups.

Respiratory syncytial virus

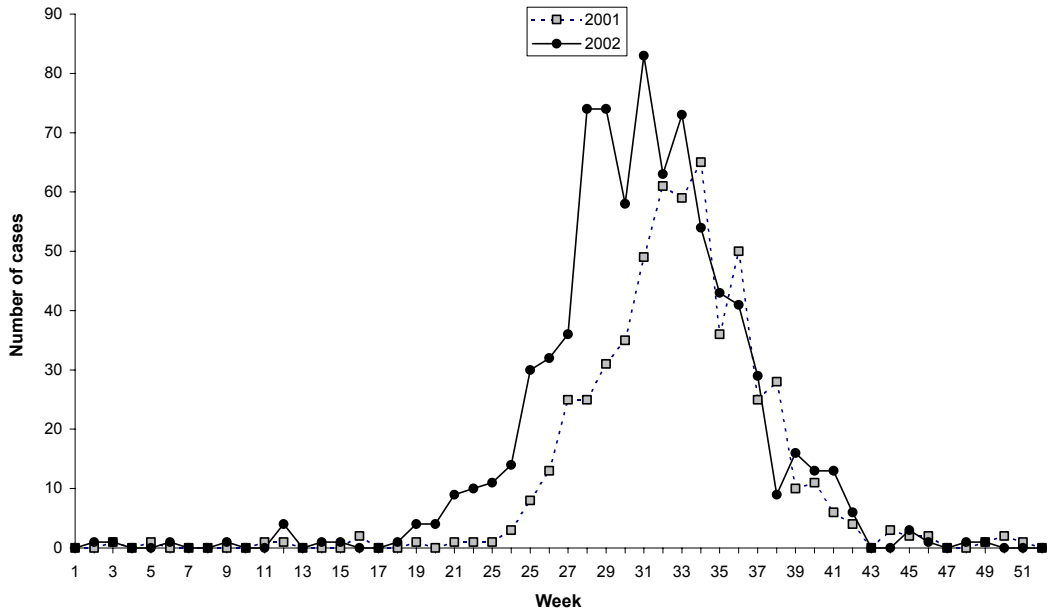
Surveillance of respiratory syncytial virus (RSV) is based on isolates confirmed by New Zealand virus laboratories. During 2002, a total of 816 RSV infections was reported to ESR, compared with 565 during the previous year. The following graph shows the number of laboratory-confirmed RSV cases since 1990. The highest RSV activity occurred in 1999 when 858 cases were reported.

Laboratory-confirmed RSV cases, 1990-2002



The following graph illustrates the seasonal trends in RSV activity over the past two years. In 2002 the RSV activity started to increase in May and peaked in Week 31 (at the beginning of August), three weeks earlier than the peak in 2001. Activity remained at a high level until Week 37 (early September).

Laboratory-confirmed RSV cases by week, 2001 and 2002

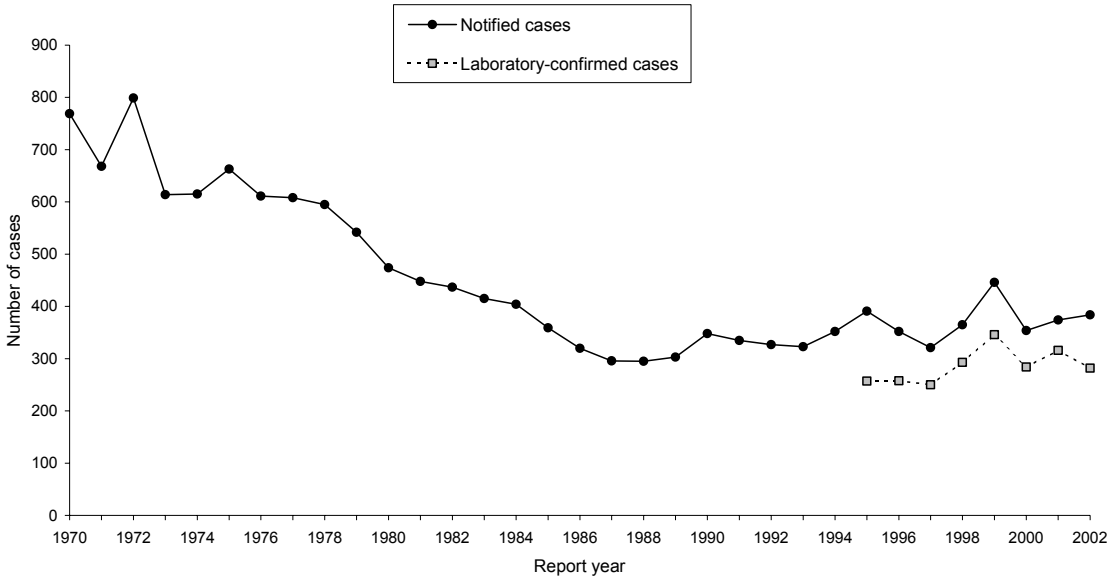


RSV causes bronchiolitis in young children and is one of the most important causes of hospital admission for lower respiratory tract infections in children. There is considerable overseas research evidence that rates are high in more crowded households[37] and for those living in more deprived neighbourhoods. [38]

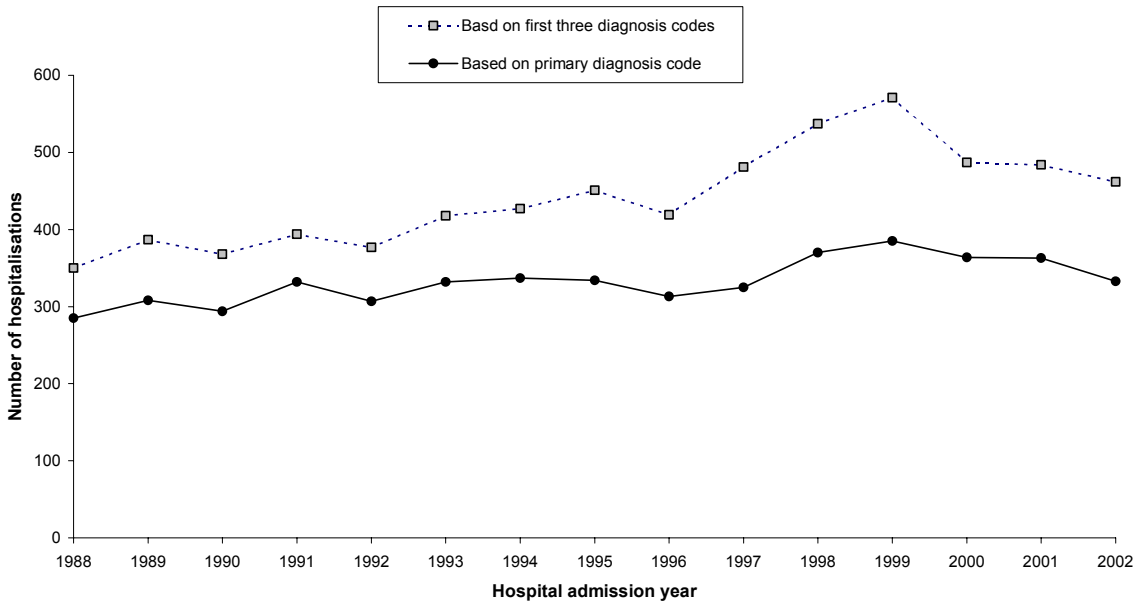
Tuberculosis

There were 384 cases of tuberculosis notified during 2002, of whom 16 (4.2%) were reactivations. A total of 282 (73.4%) cases was laboratory-confirmed. The 2002 rate of 10.3 cases per 100 000 was similar to the rate of 10.0 per 100 000 seen the previous year. Six deaths due to the disease were reported in 2002, compared to two deaths during 2001, and seven in 2000. Of the 348 cases in 2002 for whom hospitalisation status was recorded on EpiSurv, 193 (55.5%) were hospitalised. According to hospital discharge data, the number of hospitalisations due to tuberculosis (ICD9 codes 010-018) totalled 333 during 2002, a slight decrease from the 363 hospitalisations during 2001. The following graphs show (i) the number of notified and laboratory-confirmed cases each year since 1970, and (ii) the hospitalisations each year since 1988.

Tuberculosis notifications and laboratory-confirmed cases by year, 1970 - 2002



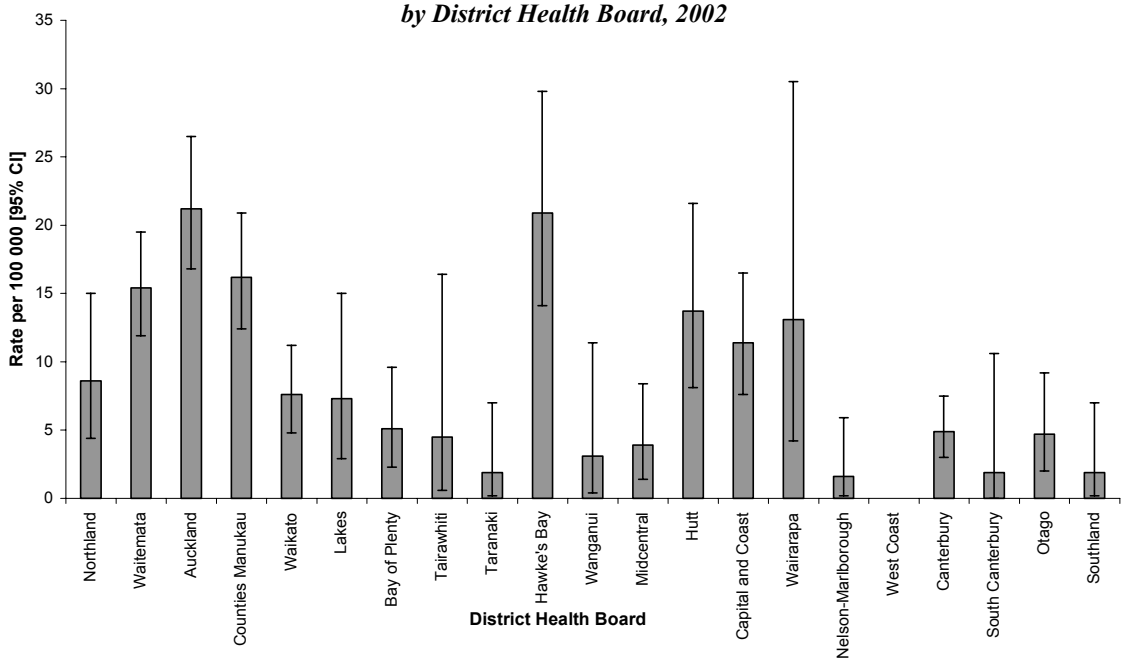
Tuberculosis hospitalisations by year, 1988 - 2002



Rates of tuberculosis varied throughout the country (see chart below). Auckland DHB reported the highest crude notification rate of 21.2 per 100 000 (95% CI: 16.8-26.5). Rates significantly higher than the national average were also recorded in Hawke's Bay (20.9 per 100 000), Counties Manukau (16.2) and Waitemata (15.4) DHBs. Four outbreaks involving a total of 39 cases were reported in 2002. One large outbreak occurred in Hawke's Bay. The index case was diagnosed in June 2002 as a reactivation of tuberculosis disease previously diagnosed and treated in 2000. Contact tracing resulted in the diagnosis of 17 additional cases

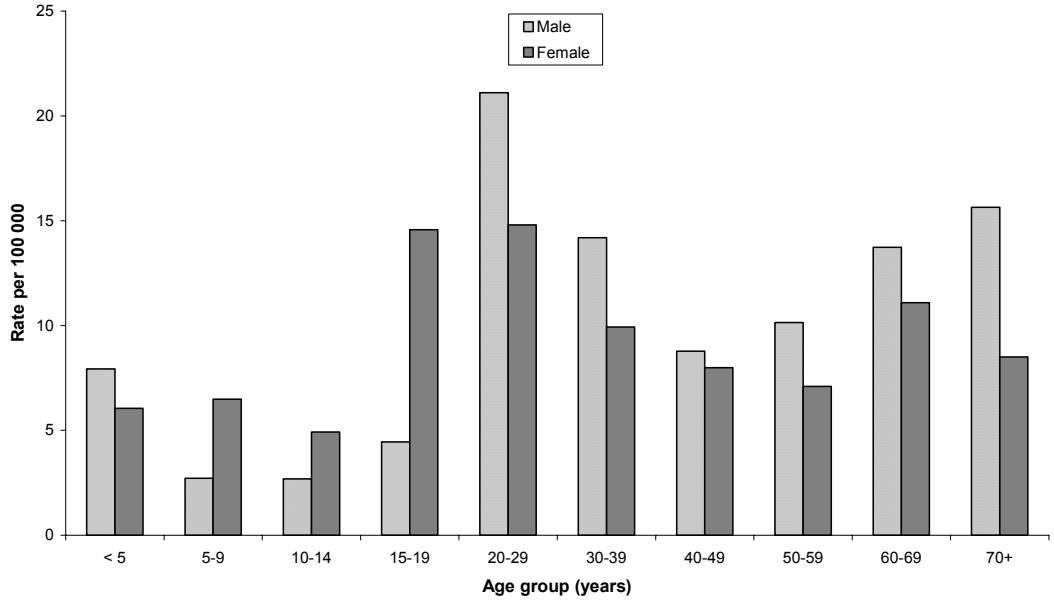
of active tuberculosis disease. Molecular typing (DNA-fingerprinting) of isolates confirmed that the strain was the same as that seen in the Rangipo Prison cluster.

Tuberculosis crude notification rates by District Health Board, 2002



The chart below compares notification rates of tuberculosis by age and sex. In the adult population, rates were generally higher in males than females. Males in the '20 to 29 years' age group experienced the highest rate of 21.1 per 100 000, followed by males in the '70+ years' age group (15.6 per 100 000). The age-specific rate of 17.9 (95% CI: 14.3-22.1) for the '20 to 29 years' age group was significantly higher than the national average rate.

Tuberculosis notification rates, 2002 by age and sex



The tables below illustrate the age and ethnic distribution of cases. Rates of tuberculosis were significantly higher in the 'Other' ethnic group (74.4 per 100 000) than in all other ethnic groups. The rate of disease of 34.9 per 100 000 in Pacific peoples was significantly higher than in the Maori ethnic group with a rate of 12.3. Rates were low in the European population.

Tuberculosis notifications and age-specific rates by ethnicity, 2002

Ethnicity	Age group in years											
	<1	**1-4**	**5-9**	**10-14**	**15-19**	**20-29**	**30-39**	**40-49**	**50-59**	**60-69**	**70+**	**Total**
European	0	0	0	0	1	4	5	5	6	4	14	39
	0.0	0.0	0.0	0.0	0.6	1.3	1.3	1.3	1.8	1.7	4.9	1.5
Maori	0	3	1	2	6	11	10	5	3	20	3	65
	0.0	5.6	1.5	3.2	12.1	13.4	12.9	8.6	9.0	102.5	31.0	12.3
Pacific peoples	1	11	7	2	8	12	7	5	10	3	4	70
	19.4	57.4	29.6	9.2	44.1	35.5	22.1	22.9	75.0	40.1	90.5	34.9
Other	0	3	1	4	10	58	48	28	14	8	10	184
	0.0	21.4	5.4	19.8	35.7	133.4	105.0	74.1	71.7	72.5	178.3	74.4
Unknown	0	1	4	3	1	2	2	3	3	1	6	26
Total	1	18	13	11	26	87	72	46	36	36	37	384
	1.8	8.3	4.5	3.8	9.8	17.9	12.5	8.6	8.6	12.7	11.5	10.3

Number of cases
Rate per 100 000

Tuberculosis - crude and age-standardised rates by ethnicity, 2002

Ethnicity	Crude rate [95% CI]	Age-standardised rate* [95% CI]
European	1.5 [1.1, 2]	1.3 [0.9, 1.8]
Maori	12.3 [9.5, 15.7]	17.9 [13.1, 24.6]
Pacific peoples	34.9 [27.2, 44.2]	40.3 [29.6, 55.2]
Other	74.4 [63.6, 85.1]	78.8 [65.7, 94.9]
Total	10.3 [9.2, 11.3]	

* Directly standardised to the NZ population

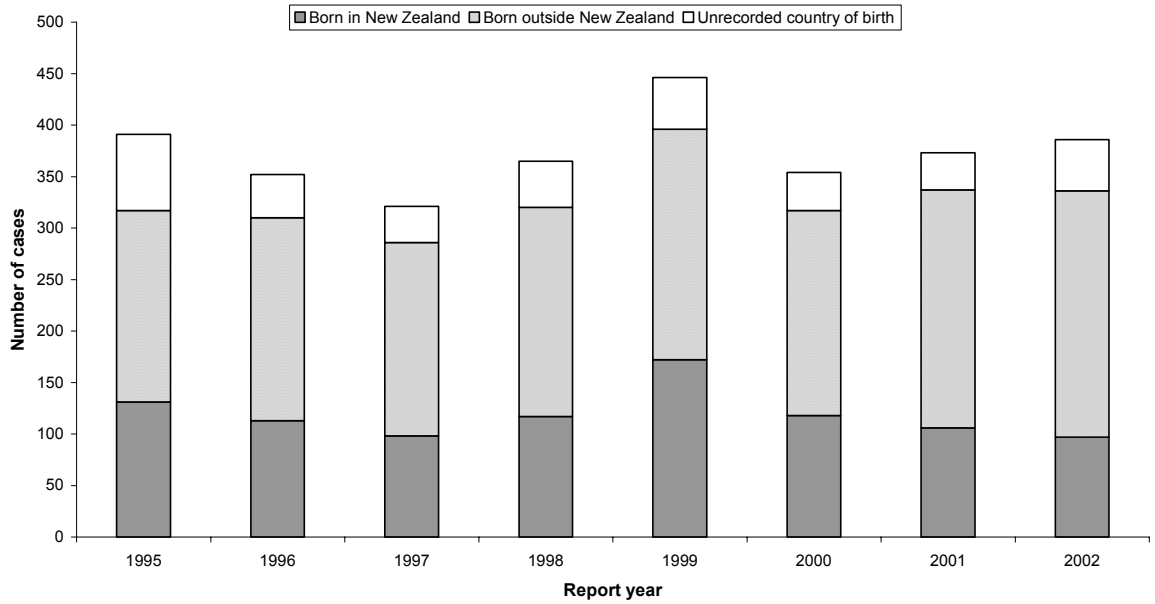
Of the 384 cases of tuberculosis notified during 2002, two cases from Waikato DHB were also notified with AIDS, and a further four cases (two from Auckland, one from Wellington and one from Hawke's Bay) were co-infected with HIV. All six cases were recorded as having been born overseas - four in Africa and two in Asia.

Information on how cases were discovered was recorded for 330 cases in 2002. Of these, 217 (65.8%) saw a practitioner with symptoms, 57 (17.3%) were detected through contact follow-up, and 23 (7.0%) through refugee or immigrant screening.

Information on country of birth was recorded for 336 cases. Of these, 239 (71.1%) were born overseas. In comparison, 68.7% of cases notified in 2001 and 62.3% of those in 2000 were overseas-born. All cases of 'Other' ethnicity were born overseas, 71% of cases of Pacific ethnicity (for whom country of birth was recorded) were born overseas, and no cases of Maori

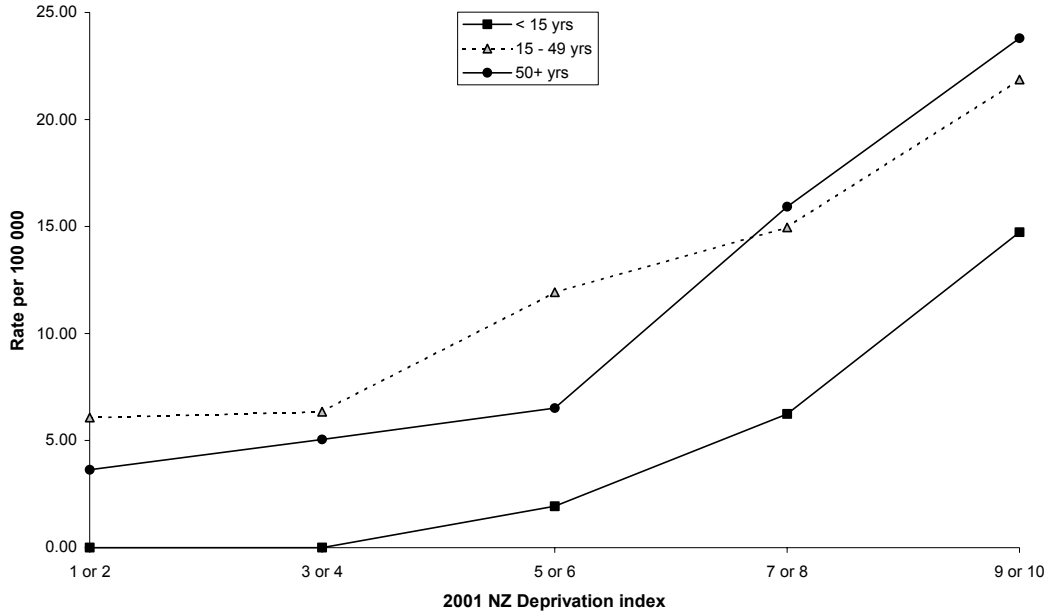
ethnicity were born overseas. The majority (71.2%) of cases of 'Other' ethnicity (for whom arrival date in New Zealand was recorded) developed tuberculosis within five years of arriving in New Zealand, whereas only 50% of cases of Pacific ethnicity were notified with the disease within five years. The following graph illustrates how the proportion of notified cases of tuberculosis disease that was born in New Zealand has decreased each year since 1999.

Notified cases of tuberculosis disease by country of birth, 1995 - 2002



The following graph illustrates the association between rates of tuberculosis and deprivation in New Zealand, as measured on the 1 to 10 scale, with 1 representing the least deprived and 10 representing the most deprived score. Rates in all three age groups (<15, 15-49 and 50+ years) increase markedly with increasing deprivation. This relationship is likely to be affected by both patterns of local disease transmission and by migration and settlement of people from countries with a high incidence of TB. Both patterns would tend to increase the incidence of TB in more deprived suburbs that also contain a higher proportion of crowded households.

Rates of tuberculosis by age group and associated index of deprivation, 2002



Antituberculosis drug resistance

The data contained in this section is based on the antimicrobial susceptibility of the 268 isolates referred to and isolated in the Mycobacteriology Reference Laboratories at Auckland, Wellington and Waikato Hospitals during 2002.

During the year, 264 *Mycobacterium tuberculosis* and four *M. bovis* isolates were identified. All isolates were tested for susceptibility to isoniazid, rifampicin, ethambutol, pyrazinamide and streptomycin. The proportion of isolates resistant to each antimicrobial is shown below.

Resistance to each antimicrobial, 2002

Antimicrobial	Number tested	Number resistant[1]	Percent resistance[1]
Isoniazid	268	26	9.7
Rifampicin	268	4	1.5
Ethambutol	268	5	1.9
Pyrazinamide	268	11[2]	4.1
Streptomycin	268	15	5.6

Notes: 1 includes resistance alone or in combination with other antimicrobials
2 includes the four *M. bovis* isolates

The majority (83.2%) of isolates were susceptible to all five antimicrobials tested (see table below). Three isolates were multidrug resistant (MDR-TB), that is, resistant to at least isoniazid and rifampicin. Two of these isolates were from cases who had arrived in New Zealand less than two years prior to diagnosis. The third MDR isolate was also from an immigrant, although multidrug resistance appears to have developed during treatment in New

Zealand. All 13 previously recorded cases of MDR-TB in New Zealand have occurred in people born overseas who were assumed to have acquired MDR-TB overseas.

Distribution of resistance patterns, 2002

Susceptibility	Number (%)	Resistance pattern[1]	Number (%) of isolates with each pattern
Fully susceptible	223 (83.2)	-	-
Resistant to 1 agent	32 (11.9)	H R Z S E	13 (4.9) 1 (0.4) 9 (3.4)[2] 5 (1.9) 4 (1.5)
Resistant to 2 agents	11 (4.1)	HR[3] HZ HS	1 (0.4) 1 (0.4) 9 (3.4)
Resistant to 3 agents	1 (0.4)	HRZ[3]	1 (0.4)
Resistant to 4 agents	1 (0.4)	HRSE[3]	1 (0.4)

Notes: 1 H, isoniazid; R, rifampicin; Z, pyrazinamide; S, streptomycin; E, ethambutol
2 includes the four *M. bovis* isolates
3 MDR-TB, multidrug-resistant tuberculosis, that is, resistant to at least isoniazid and rifampicin

Nine (3.4%) of the total 268 isolates were from cases categorised as tuberculosis reactivations. There were no significant differences in resistance among reactivations compared with new cases of tuberculosis. Isolates from overseas-born cases were significantly more resistant to isoniazid and streptomycin, and less resistant to pyrazinamide. Isolates from New Zealand-born cases are more resistant to pyrazinamide as *M. bovis* infections are more common in New Zealand-born cases and this species is intrinsically resistant to pyrazinamide.

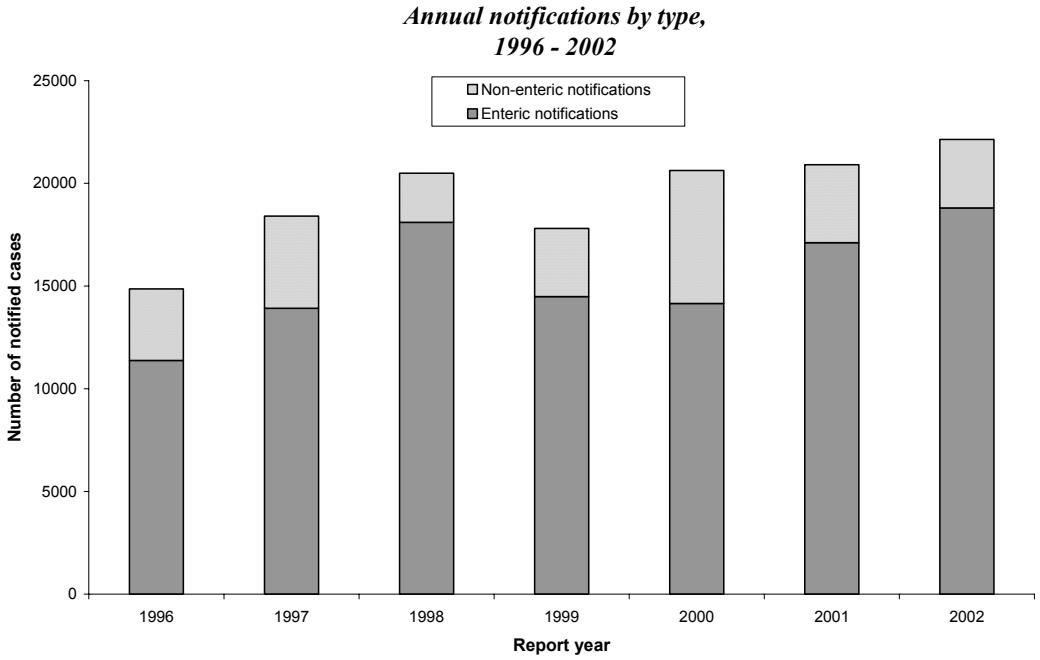
Notification, laboratory, and hospitalisation data all show similar trends in tuberculosis incidence. Disease declined for several decades before reaching a plateau in the mid 1980s. Since then the incidence has risen gradually. A key factor is migration of people from high incidence countries to New Zealand. Control of this disease relies on a range of strategies described in the Ministry of Health's TB control guidelines.[39]

Enteric infections

Enteric diseases, or infectious intestinal diseases, may be caused by bacteria, protozoa or viruses. They are grouped together because they share common transmission pathways from contaminated, food, water and environments, and from direct contact with other infected people or animals.

Incidence of enteric diseases

There were 18800 enteric notifications in 2002, representing 85% of total notifications. The following graph illustrates the relative proportion of notifications each year that have been due to enteric diseases. The high number and proportion of enteric notifications in 1998 and 2002 was largely driven by elevated incidence of campylobacteriosis during these years.



Risk factors for enteric diseases

Most enteric diseases are reported on a single Enteric Case Report Form in EpiSurv. For these diseases it is possible to compare the proportion of cases reporting exposure to a given set of risk factors. The following table shows the percentage of cases for whom exposure to certain risk factors was recorded, for each notifiable enteric disease reported on the EpiSurv Enteric Case Report Form. This information needs to be interpreted with caution for a number of reasons: The percentage of cases for whom risk factor information is recorded on EpiSurv varies between diseases and risk categories, and is generally quite low. Percentages calculated for low incidence diseases will in general have wide confidence intervals. Interpretation is also limited by the absence of a comparison control population. For relatively uncommon exposures, such as overseas travel in the days preceding onset of illness, these exposures are highly suggestive of an aetiological link. Whereas, for other more common exposures, such

as consuming food from a food premises during the incubation period, it is difficult to evaluate the importance of the exposure.

Percentage of enteric disease cases reporting exposure to key risk factors, 2002*

Risk category	**Food and water contact**			**Animal contact**		**Human contact**				**Travel**
Disease	Food premise[1]	Untreated drinking water	Recreational water	Farm animals	Sick animals	Faecal matter, vomit	Other symptomatic case[2]	Confirmed case[3]	School, childcare[4]	Overseas
Campylobacter	54%	20%	17%	33%	7%	12%	12%	6%	16%	7%
Cryptosporidiosis	32%	39%	35%	59%	23%	31%	27%	15%	46%	7%
Gastroenteritis	88%	6%	7%	8%	1%	10%	41%	67%	9%	3%
Giardiasis	33%	36%	33%	26%	5%	46%	37%	31%	35%	17%
Paratyphoid	44%	30%	21%	14%	8%	27%	15%	10%	33%	68%
Salmonellosis	50%	24%	18%	29%	6%	16%	17%	12%	28%	14%
Shigellosis	50%	23%	14%	14%	0%	10%	13%	15%	18%	49%
Typhoid	50%	67%	33%	18%	11%	30%	22%	40%	23%	67%
Yersiniosis	45%	19%	20%	30%	3%	14%	8%	3%	21%	6%

* percentage is total number of cases exposed divided by total number of cases for whom this information was recorded
[1] consumed food at a food premise during the incubation period
[2] had contact with other symptomatic case during incubation period
[3] had contact with a confirmed case during incubation period
[4] case attends school, pre-school or childcare

Food and water contact: 'Eating out' during the incubation period was most frequently reported by gastroenteritis cases (88%), followed by campylobacteriosis (54%) and salmonellosis (50%) cases, implying common food-borne transmission of these diseases. Over a third of all typhoid, cryptosporidiosis and giardiasis cases reported drinking untreated water during the incubation period. Recreational water contact, including use of swimming pools, was also frequently reported by cryptosporidiosis cases (35%), followed by giardiasis (33%) and typhoid (33%) implying that contaminated water may in some circumstances be a source of infection for this disease.

Animal contact[a]*:* Contact with farm animals was most commonly reported by cryptosporidiosis cases (57%), followed by campylobacteriosis cases (33%). Contact with sick animals was also most frequently reported by cryptosporidiosis cases (23%), implying direct zoonotic transmission of this disease.

Human contact: Contact with other symptomatic cases was most frequently reported by gastroenteritis cases (41%), followed by giardiasis cases (37%). Over a third of cryptosporidiosis (46%) and giardiasis (35%) cases attended school, pre-school or childcare. Contact with faecal matter or vomit was also most frequently reported by giardiasis cases (46%), implying common person-to-person transmission (via contaminated faeces) of this disease.

Overseas travel: Typhoid and paratyphoid were both frequently travel-associated, with around two thirds of cases reporting overseas travel during the incubation period. Nearly half (49%) of all shigellosis cases were also linked to overseas travel.

[a] Contact with farm animals was also frequently reported by notified cases of VTEC/STEC infection (58.5% of cases), but because a different Case Report Form is used for the reporting this disease, the results are not directly comparable.

Rural/urban distribution of enteric disease

Based on Census 2001 data, the proportion of New Zealanders residing in areas classified as 'rural' was approximately 12.6%. The following table shows the proportion of cases which reside in rural parts of New Zealand, along with incidence rates for both the rural and urban population. The notification rates of *Salmonella* Brandenburg were almost six times higher in rural areas than in urban areas, and the rates for cryptosporidiosis were almost three times higher in rural areas. Only gastroenteritis notification rates were significantly higher in urban areas than in urban areas.

The proportion of enteric disease cases residing in rural areas, and notification rates for both the rural and the urban population

Enteric disease	Proportion of cases[1] residing in rural areas	95% CI for rural proportion	Notification rate[2] in rural population	Notification rate[2] in urban population
Campylobacter	12.4%	[11.9, 13.0]	339.5	330.8
Cryptosporidiosis	27.8%	[25.1, 30.7]	59.0	21.2
Gastroenteritis	5.8%	[4.5,7.3]	13.6	30.8
Giardiasis	12.2%	[10.6, 13.9]	41.1	41.2
Listeria	16.7%	[5.8, 39.2]	0.7	0.5
Salmonella Brandenburg	44.4%	[33.9, 55.3]	7.7	1.3
S. Typhimurium 160	17.0%	[13.9, 20.5]	18.7	12.7
Salmonellosis (Total)	16.0%	[14.4, 17.7]	65.8	47.8
VTEC	26.8%	[17.9, 38.1]	4.2	1.6
Yersiniosis	10.5%	[8.1, 13.6]	11.0	12.9

[1] Proportion of notified cases with addresses recorded on EpiSurv
[2] Rate per 100 000 population

Botulism

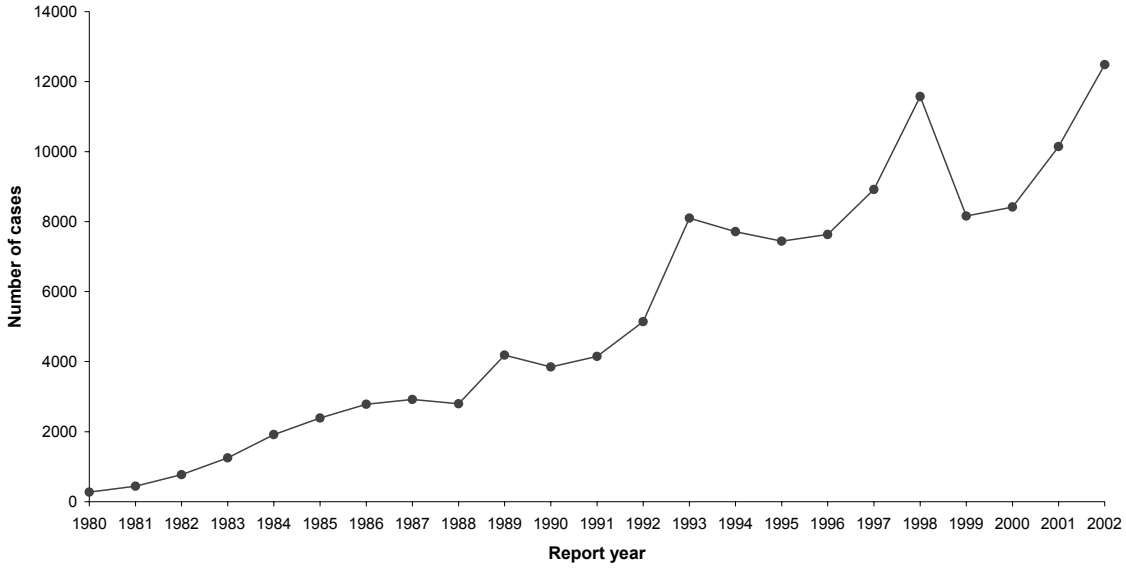
There have been no notifications of botulism in New Zealand since at least 1987. Hospitalisation data record one hospitalisation due to botulism in 1989, two hospitalisations in 1994 and one in 1995. Two cases that occurred in 1985 were reported as the first in New Zealand. [40]

Campylobacteriosis

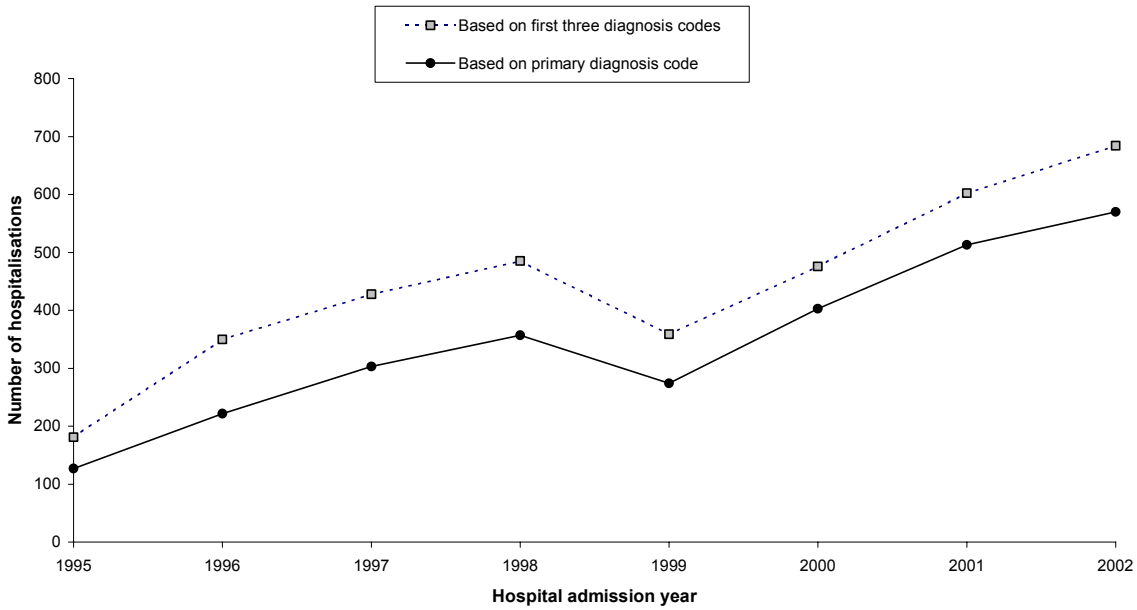
There were 12 489 cases of campylobacteriosis notified in 2002, of whom 99.3% were reported as confirmed. Incidence has increased steadily since 1999 when 8161 cases were notified. The 2002 rate of 334.2 cases per 100 000 was significantly higher than the 2001 rate of 271.5. The death of an elderly woman due to campylobacteriosis was reported in 2002. Of the 7735 cases for whom hospitalisation status was recorded on EpiSurv, 515 (6.7%) were hospitalised. According to hospital discharge data, hospitalisations in 2002 totalled 570. A total of 51 outbreaks was reported in 2002, of which 30 (59%) occurred in the Auckland region. The following graphs show (i) the number of cases notified each year since 1980, and

(ii) the hospitalisations (ICD9 code 008.43) each year since 1995. Both surveillance data sources show a similar pattern of increasing disease incidence over time.

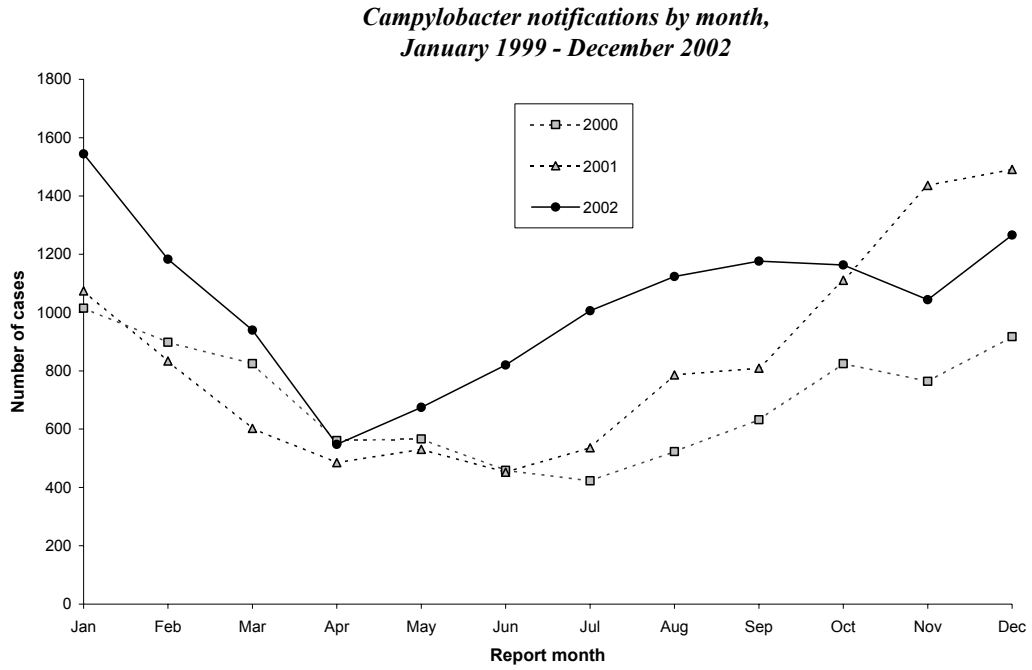
Campylobacteriosis notifications by year, 1980 - 2002



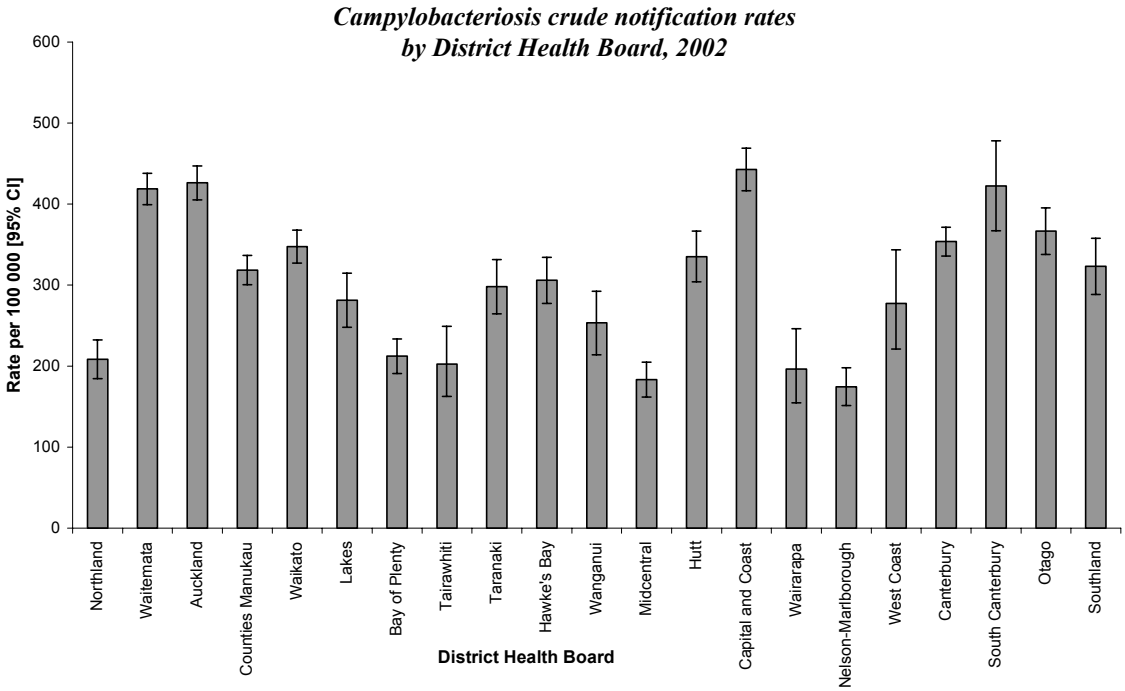
Campylobacteriosis hospitalisations by year, 1995 - 2002



Like most enteric disease, campylobacteriosis is highly seasonal with a summer peak and winter trough. The pattern in 2002 was atypical in that an unusually high incidence was sustained throughout winter and early spring. The following graph illustrates these points.

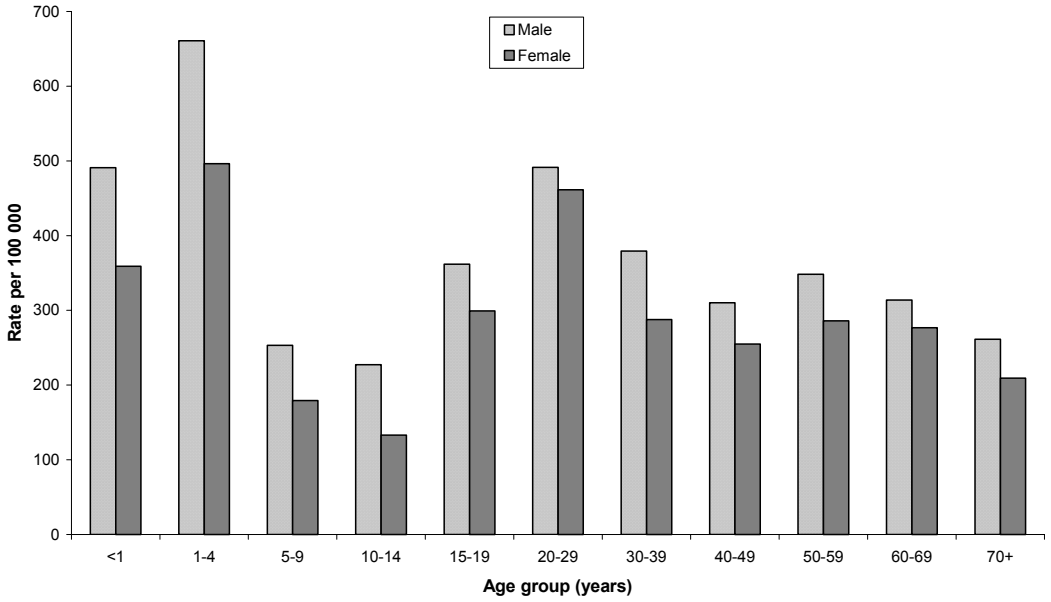


Rates varied throughout the country, with rates significantly higher than the national average being reported by Capital and Coast (442.5 per 100 000), Auckland (426.1), South Canterbury (422.5), Waitemata (418.6), Otago (366.6) and Canterbury (353.6) District Health Boards. The following graph shows the crude notification rates by District Health Board in 2002.



In every age group rates of campylobacteriosis were higher in males than in females. The peak age-specific rate of 599.1 (95% CI: 566.5-631.7) occurred in children aged between one and four years. Rates significantly higher than the national average of 334.2 (95% CI: 328.3-340.0) were also reported in the '20 to 29 years' (484.6, 95% CI: 465.2-504.3) and the 'less than one year' (435.6, 95% CI: 380.2-490.9) age groups. The following chart compares notification rates by age and sex.

Campylobacteriosis notification rates, 2002 by age and sex



Ethnicity was recorded for 72.4% of all campylobacteriosis notifications during 2002. As in previous years, the highest rate occurred among those of European ethnicity (307.7 per 100 000), followed by those of 'Other' ethnicity (197.2). Rates of disease were particularly low in the Pacific peoples ethnic group (53.9), which experienced a rate significantly lower than all other ethnic groups. Age-standardised rates were also highest in the European ethnic group. The tables below illustrate the age and ethnic distribution of cases.

Campylobacteriosis notifications and age-specific rates by ethnicity, 2002

Ethnicity	**Age group in years**											
	<1	**1-4**	**5-9**	**10-14**	**15-19**	**20-29**	**30-39**	**40-49**	**50-59**	**60-69**	**70+**	**Total**
European	143	803	371	320	545	1440	1241	1063	915	597	572	8040
	480.7	660.1	219.6	180.4	339.5	471.3	312.5	267.3	273.5	257.3	199.0	307.7
Maori	12	62	15	16	45	110	62	32	26	15	7	405
	85.8	115.8	22.7	25.4	90.8	133.7	79.9	55.2	78.2	76.9	72.3	76.9
Pacific peoples	2	19	8	5	11	18	18	12	8	4	2	108
	38.8	99.1	33.9	23.1	60.7	53.2	56.7	55.0	60.0	53.5	45.3	53.9
Other	11	70	62	45	53	105	48	38	25	16	10	488
	295.9	499.0	337.2	223.2	189.3	241.5	105.0	100.6	127.9	145.0	178.3	197.2
Unknown	70	341	174	148	251	686	577	387	373	215	172	3448
Total	238	1295	630	534	905	2359	1946	1532	1347	847	763	12489
	435.6	599.1	220.2	183.6	341.2	484.7	337.4	285.1	322.1	299.8	236.6	334.2

Number of cases
Rate per 100 000

Campylobacteriosis - crude and age-standardised rates by ethnicity, 2002

Ethnicity	Crude rate [95% CI]	Age-standardised rate* [95% CI]
European	307.7 [301, 314.5]	315.4 [308.5, 322.5]
Maori	76.9 [69.4, 84.4]	76.6 [68.1, 86.7]
Pacific peoples	53.9 [43.7, 64.1]	53.3 [42.4, 68.0]
Other	197.2 [179.7, 214.7]	192.6 [174, 213.9]
Total	334.2 [328.3, 340.0]	

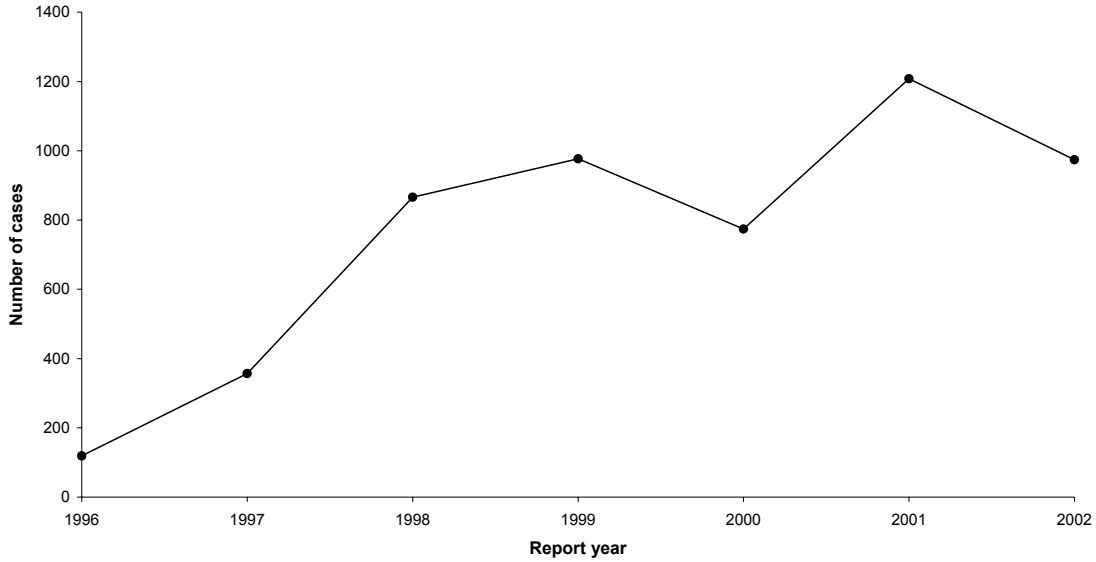
* Directly standardised to the NZ population

Notification and hospitalisation data both show a continuing rise in campylobacteriosis incidence over more than decade suggesting a real increase in the incidence of this disease. The 2002 notification total was the highest reported to date. The only national case-control study carried out in New Zealand has identified a number of risk factors for sporadic disease, notably consumption of undercooked chicken. [41] The limited risk factor data provided by routine surveillance do not point to animal, human, or water sources as being particularly important. Controlling this disease will largely depend on reducing human exposure to the infection from contaminated food.

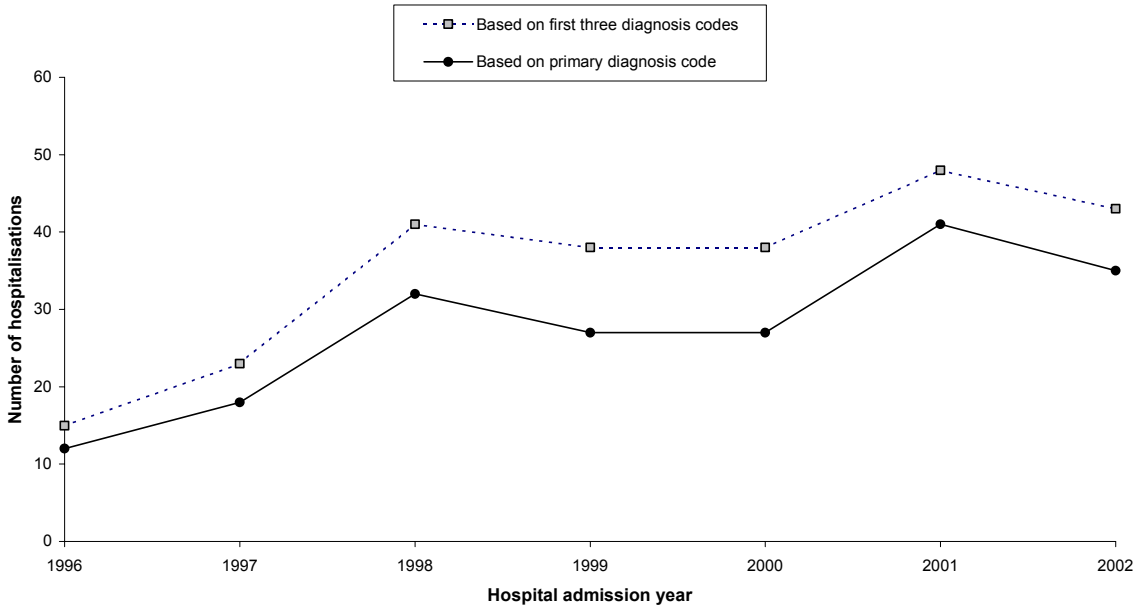
Cryptosporidiosis

There were 974 cases of cryptosporidiosis notified in 2002, of whom 952 (97.7%) were confirmed. The 2002 rate of 26.1 (95% CI: 24.4-27.7) was significantly lower than the 2001 rate of 32.3 (95% CI: 30.5-34.1). Of the 859 cases in 2002 for whom hospitalisation status was recorded on EpiSurv, 40 (4.7%) were hospitalised. According to hospital discharge data, hospitalisations in 2002 totalled 35. The following graphs showing (i) the annual number of notifications, and (ii) the hospitalisations (ICD9 code 007.4) each year since 1996, exhibit similar trends in incidence.

Cryptosporidiosis notifications by year, 1996 - 2002

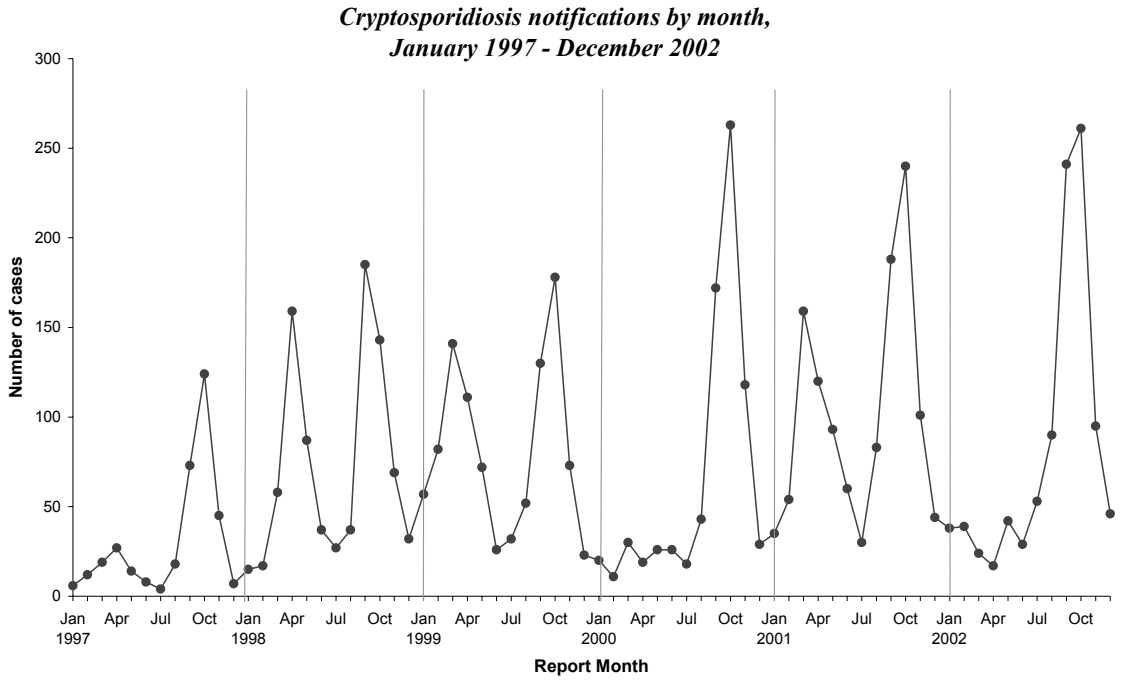


Cryptosporidiosis hospitalisations by year, 1996 - 2002



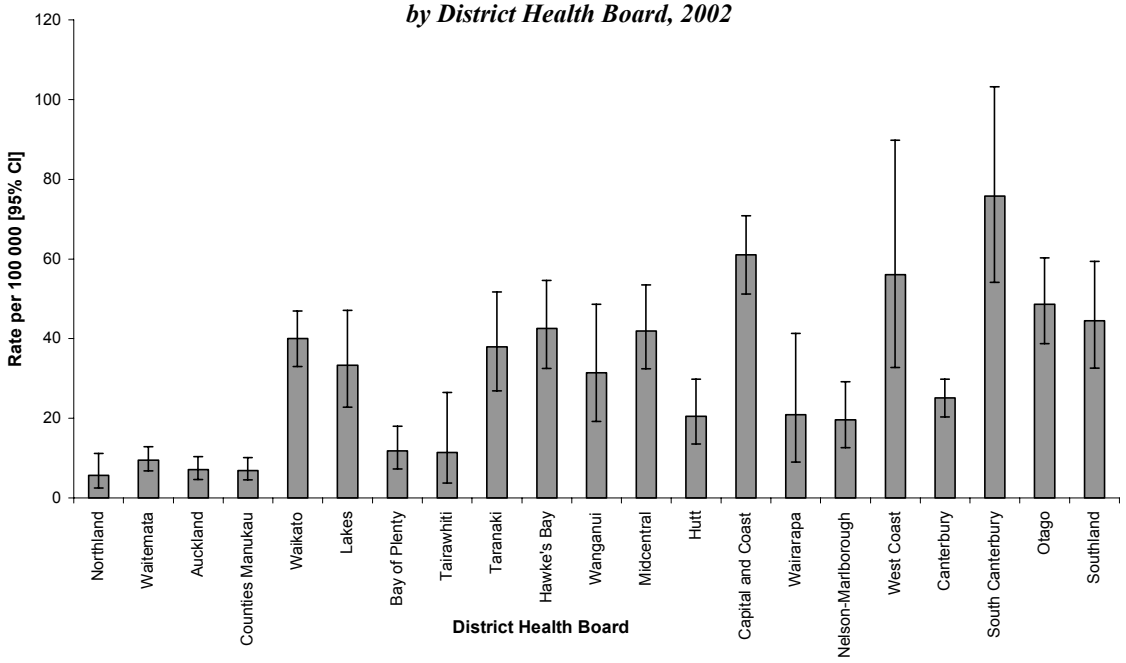
A total of 15 outbreaks of cryptosporidiosis, involving 120 cases, was reported in 2002. Two large outbreaks in winter were linked to contaminated swimming pool water at two separate locations in Wellington. During the winter months (June to August) 60.7% of notified cases reported recreational contact with water and 14.5% reported contact with farm animals. In contrast, during the spring (September to November), the proportion reporting recreational contact with water dropped to 23.6%, and the proportion reporting contact with farm animals rose to 32.8%.

The following graph illustrates the seasonal pattern of cryptosporidiosis notifications since January 1997. There has been a spring peak in all years. An autumn peak has occurred in all years except 2002 and 2000.



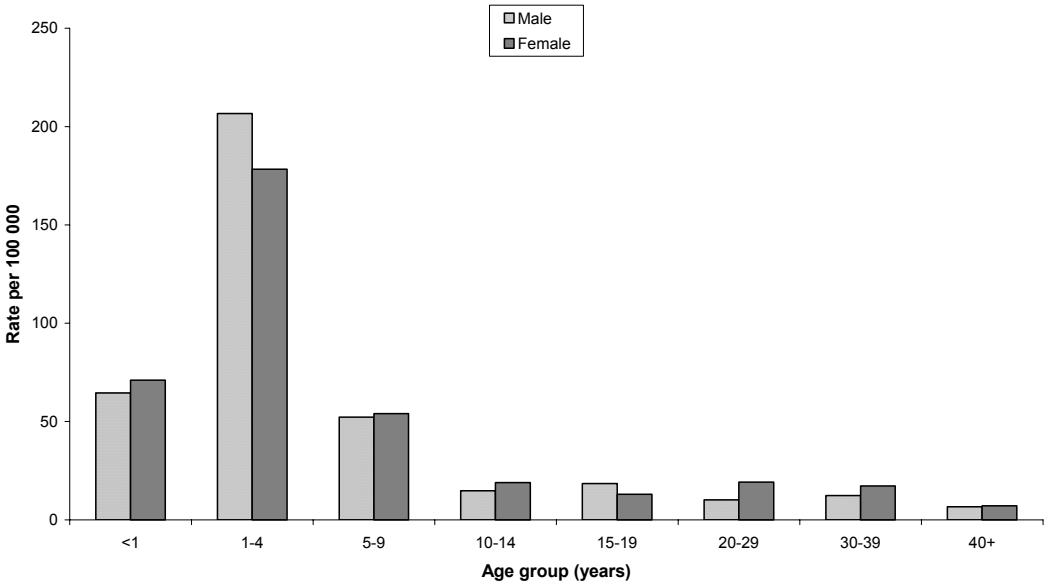
The rate of cryptosporidiosis varied throughout the country in 2002, as the following bar chart demonstrates. South Canterbury DHB recorded the highest rate of 75.8 per 100 000. Rates significantly higher than the national average were also reported by Capital and Coast (61.0), West Coast (56.1), Otago (38.7), Waikato (33.0), Southland (32.6), Hawke's Bay (32.5) and Midcentral (32.4) District Health Boards. As discussed earlier, the rate of cryptosporidiosis in rural areas was calculated to be nearly three times the rate in urban areas.

Cryptosporidiosis crude notification rates by District Health Board, 2002



Age-specific notification rates were significantly higher in the '1 to 4 years' age group than in all other age groups. Rates significantly higher than the national average of 26.1 per 100 000 were also seen in the 'less than one year' (71.4, 95% CI: 50.8-97.6) and '5 to 9 years' (53.1, 95% CI: 44.7-61.6) age groups. The following chart compares notification rates by age and sex.

Cryptosporidiosis notification rates, 2002 by age and sex



The tables below illustrate the age and ethnic distribution of cases. Children aged between one and four years of European ethnicity experienced the highest rate of 247.4 per 100 000. As in previous years, crude and age-standardised rates were significantly higher among those of European ethnicity than in all other ethnic groups. Rates were particularly low in the Pacific peoples ethnic group.

Cryptosporidiosis notifications and age-specific rates by ethnicity, 2002

Ethnicity	Age group in years											
	<1	**1-4**	**5-9**	**10-14**	**15-19**	**20-29**	**30-39**	**40-49**	**50-59**	**60-69**	**70+**	**Total**
European	31	301	113	39	37	65	66	56	23	8	8	749
	104.2	247.4	66.9	22.0	23.1	21.3	16.6	14.1	6.9	3.4	2.8	28.7
Maori	4	26	6	0	1	2	4	1	3	0	0	47
	28.6	48.6	9.1	0.0	2.0	2.4	5.2	1.7	9.0	0.0	0.0	8.9
Pacific peoples	0	6	0	0	0	1	0	0	0	0	0	7
	0.0	31.3	0.0	0.0	0.0	3.0	0.0	0.0	0.0	0.0	0.0	3.5
Other	0	8	4	2	0	1	5	1	1	0	0	22
	0.0	57.0	21.8	9.9	0.0	2.3	10.9	2.6	5.1	0.0	0.0	8.9
Unknown	4	80	29	8	4	5	11	4	2	0	0	149
Total	39	421	152	49	42	74	86	62	29	8	8	974
	71.4	194.8	53.1	16.8	15.8	15.2	14.9	11.5	6.9	2.8	2.5	26.1

Number of cases
Rate per 100 000

Cryptosporidiosis - crude and age-standardised rates by ethnicity, 2002

Ethnicity	Crude rate [95% CI]	Age-standardised rate* [95% CI]
European	28.7 [26.6, 30.7]	32.9 [30.6, 35.4]
Maori	8.9 [6.6, 11.9]	6.4 [4.5, 10.2]
Pacific peoples	3.5 [1.4, 7.2]	2.2 [0.9, 9.2]
Other	8.9 [5.6, 13.5]	8.7 [5.4, 15.5]
Total	26.1 [24.4, 27.7]	

* Directly standardised to the NZ population

Notification and hospitalisation data suggest that the incidence of cryptosporidiosis has reached a plateau in New Zealand. The striking seasonality and wide geographic variations in incidence are highly suggestive of environmental sources of infection. The consistent marked spring peak in incidence tends to occur in rural areas with most cases reporting direct contract with farm animals. The late summer-autumn peak has occurred in three of the last five years and is associated with swimming pool outbreaks of this disease.[42]

Gastroenteritis

Gastroenteritis is a syndrome rather than a specific diagnosis. These surveillance data are therefore highly affected by the choice of case definition used and different reporting practices. Unlike the other notifiable diseases described here, the majority (64.0%) of gastroenteritis notifications in 2002 was 'self-notifications'. Outbreak investigations led to the notification of a further 175 (16.1%) cases. The data reported in this section therefore should be regarded as indicative of some aspects of gastroenteritis epidemiology rather than providing a measure of disease burden.

Until mid 2000, cases of gastroenteritis had been reported according to the case definition for acute gastroenteritis.[a] Since July 2000, public health services have also been encouraged to record all cases of gastroenteritis caused by non-notifiable or unknown foodborne intoxicants, in addition to all cases of viral gastroenteritis that may be part of an outbreak.

A total of 1084 cases of gastroenteritis[b] was notified in 2002. This was the highest annual number of notifications on record. The 2002 rate of 29.0 per 100 000 (95% CI: 27.3-30.7) was significantly higher than the 2001 rate of 25.2 (95% CI: 23.5-26.8).

A pathogen was identified in 280 (25.8%) gastroenteritis notifications. Of these, the most commonly identified pathogens were Norovirus (198/280 or 70.7%), *Clostridium perfringens* (31/280 or 11.1%), rotavirus (20/280 or 7.1%), *Bacillus cereus* (7/280 or 2.5%) and Staphylococcus aureus (7/280 or 2.5%). In comparison, enteric disease outbreaks reported during 2002 included 73 Norovirus[c] outbreaks involving 1419 cases, seven *Clostridium perfringens* outbreaks (127 cases), two rotavirus outbreaks (37 cases), four Bacillus cereus outbreaks (16 cases) and four Staphylococcus aureus outbreaks (9 cases). There were also 87 outbreaks (317 cases) specified as outbreaks of gastroenteritis without any microbiological agent identified. The following table shows the major causes of gastroenteritis recorded on EpiSurv during the past three years.

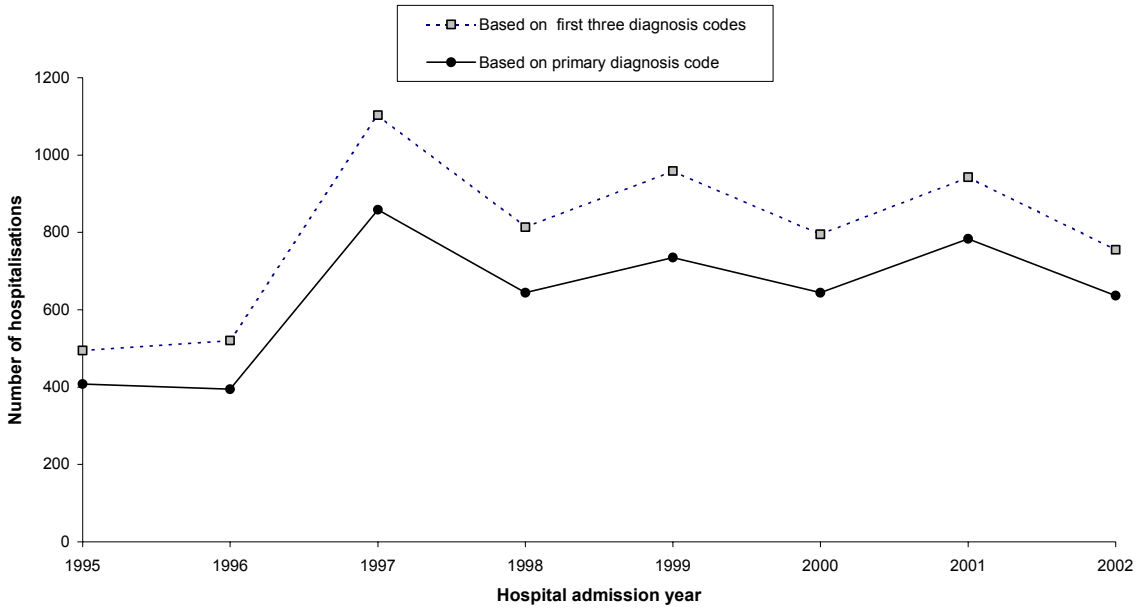
Causal agents recorded on EpiSurv for notified cases of gastroenteritis

Causal agent	2000 No. (%)	2001 No. (%)	2002 No. (%)
Unknown causal agent	561 (77.4%)	754 (80.2%)	804 (74.2%)
Norovirus infection	32 (4.4%)	107 (11.4%)	198 (18.3%)
Clostridium perfringens intoxication	24 (3.3%)	16 (1.7%)	31 (2.9%)
Rotavirus infection	38 (5.2%)	49 (5.2%)	20 (1.8%)
Bacillus cereus intoxication	27 (3.7%)	1 (0.1%)	7 (0.6%)
Staphylococcal intoxication	1 (0.1%)	4 (0.4%)	7 (0.6%)
Other causal agents	42 (5.8%)	9 (1.0%)	17 (1.6%)
Total	725 (100%)	940 (100%)	1084 (100%)

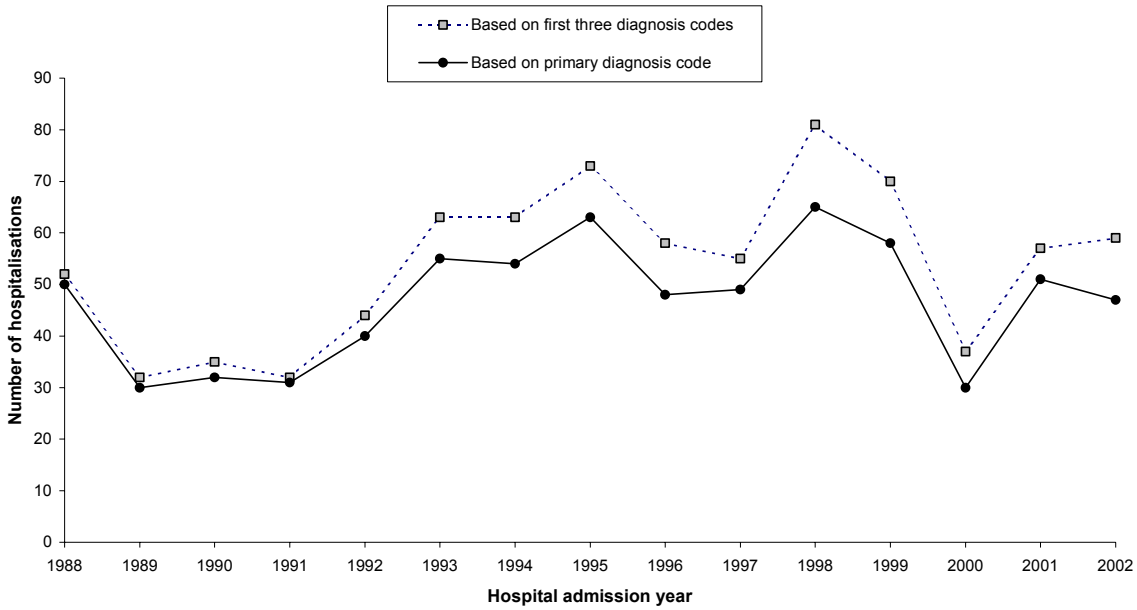
[a] Defined as a case of acute gastroenteritis where either (i) a common source was suspected; or (ii) the case was in a high risk category, or (iii) the case suffered from chemical, bacterial, or toxic food poisoning.
[b] The information reported here as 'acute gastroenteritis/gastroenteritis' excludes cases of enteric disease that are notifiable in their own right (e.g. salmonellosis or campylobacteriosis) and also excludes cases of toxic shellfish poisoning (TSP) which are reported separately in this report.
[c] A further three outbreaks with multiple pathogens/toxins (including norovirus) were reported.

Of the 969 gastroenteritis cases for whom hospitalisation status was recorded on Episurv, 21 (2.2%) were hospitalised. According to hospital discharge data in 2002, there were 637 hospitalisations due to rotavirus (ICD9 code 008.61); 47 hospitalisations due to bacterial food poisoning (ICD9 code 005) including staphylococcal, *Clostridium perfringens, Bacillus cereus* but excluding notifiable diseases; four admissions for norovirus (ICD9 code 008.63); and four for adenovirus (ICD9 code 008.62). In addition, there were a further 1350 hospitalisations due to viral gastroenteritis, not otherwise specified (ICD9 code 008.8). The following graphs show the number of hospitalisations due to rotavirus, bacterial food poisoning and gastroenteritis (unspecified) each year. Hospitalisations for unspecified gastroenteritis have been steadily increasing since 1996 and reached their highest level in 2002.

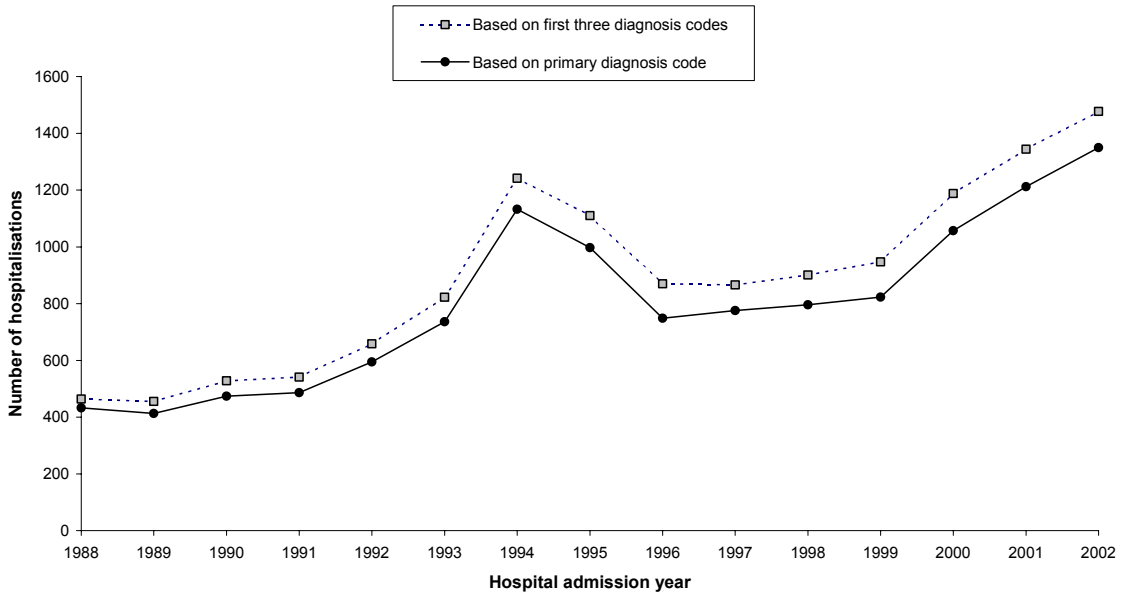
Rotavirus hospitalisations by year, 1995 - 2002



Hospitalisations due to bacterial food poisonings by year, 1988 - 2002

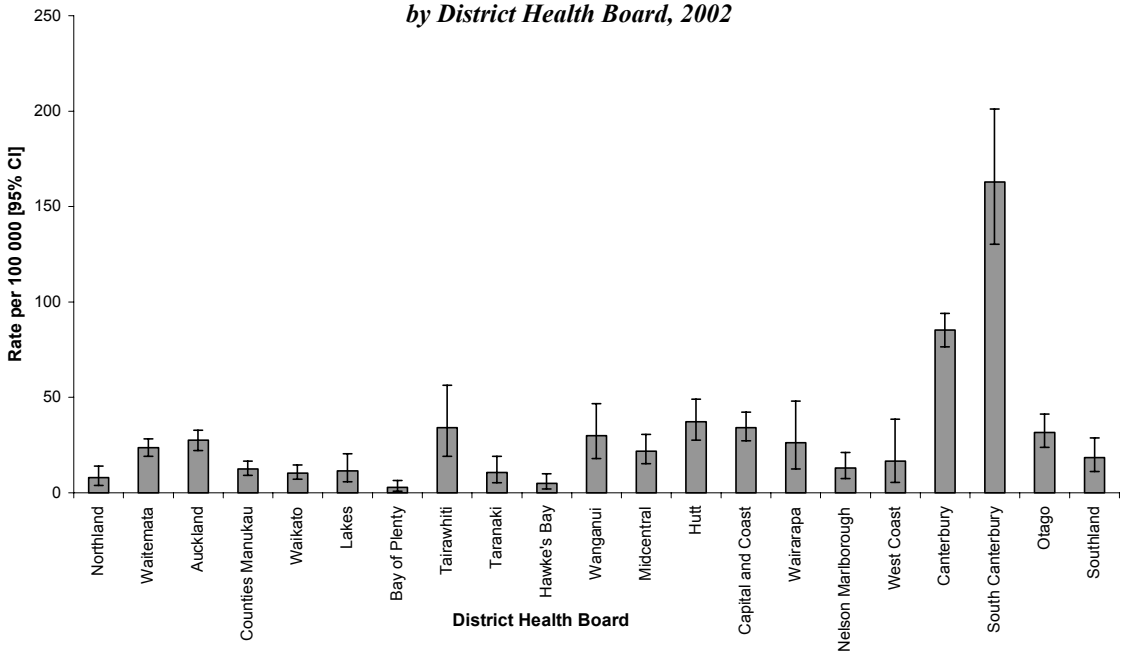


Viral gastroenteritis (unspecified) hospitalisations by year, 1988 - 2002



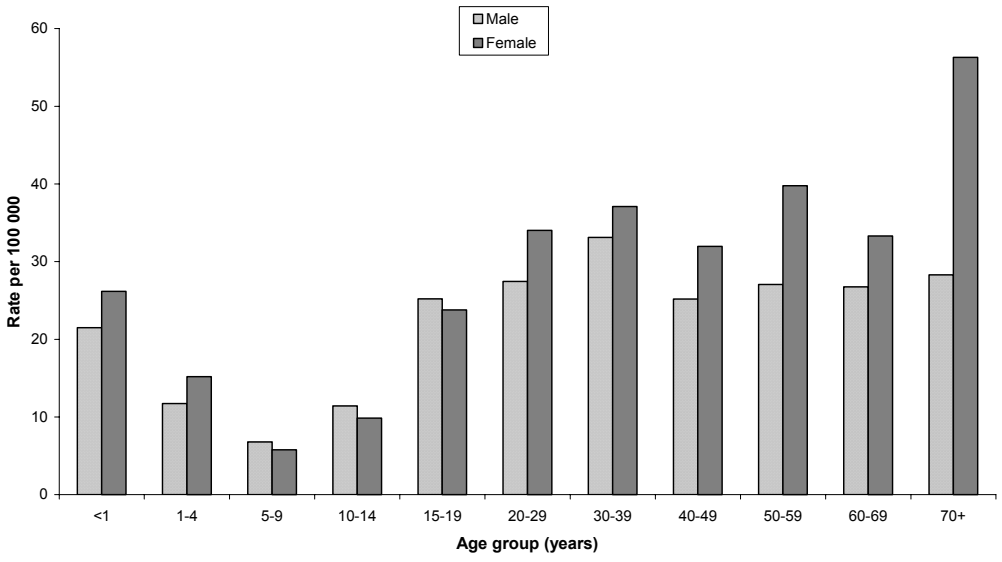
The definite or suspected source of infection was recorded as person-to-person contact for 180 (16.6%) cases, and through contact with contaminated food or drink for 500 (46.1%) cases. Gastroenteritis notification rates were significantly higher in South Canterbury DHB than all other District Health Boards, as the following chart demonstrates.

Gastroenteritis crude notification rates by District Health Board, 2002



The following graph compares notification rates by age and sex. In most age groups rates were higher among females than males. The peak age-specific rate was in the '70+ years' age group with a rate of 44.6 per 100 000 (95% CI: 37.4-51.9).

Gastroenteritis notification rates, 2002 by age and sex



The tables below illustrate the age and ethnic distribution of cases. As in previous years, rates were significantly higher in the European ethnic group (33.9 per 100 000) than in all other ethnic groups.

Gastroenteritis notifications and age-specific rates by ethnicity, 2002

Ethnicity	Age group in years											
	<1	**1-4**	**5-9**	**10-14**	**15-19**	**20-29**	**30-39**	**40-49**	**50-59**	**60-69**	**70+**	**Total**
European	8	22	11	28	57	117	168	127	119	72	132	887
	26.9	18.1	6.5	15.8	35.5	38.3	42.3	31.9	35.6	31.0	45.9	33.9
Maori	1	1	1	2	2	6	3	5	1	1	2	28
	7.1	1.9	1.5	3.2	4.0	7.3	3.9	8.6	3.0	5.1	20.7	5.3
Pacific peoples	1	0	0	0	0	1	9	3	0	0	1	15
	19.4	0.0	0.0	0.0	0.0	3.0	28.4	13.8	0.0	0.0	22.6	7.5
Other	0	1	1	0	3	8	4	4	3	3	0	27
	0.0	7.1	5.4	0.0	10.7	18.4	8.7	10.6	15.4	27.2	0.0	10.9
Unknown	3	5	5	1	3	22	19	16	17	9	9	127
Total	13	29	18	31	65	154	203	155	140	85	144	1084
	23.8	13.4	6.3	10.7	24.5	31.6	35.2	28.8	33.5	30.1	44.6	29.0

Number of cases
Rate per 100 000

Gastroenteritis - crude and age-standardised rates by ethnicity, 2002

Ethnicity	Crude rate [95% CI]	Age-standardised rate* [95% CI]
European	33.9 [31.7, 36.2]	32.1 [30, 34.3]
Maori	5.3 [3.5, 7.7]	6.2 [3.5, 11.1]
Pacific peoples	7.5 [4.2, 12.4]	9.0 [4.5, 18.5]
Other	10.9 [7.2, 15.9]	10.6 [6.8, 17.9]
Total	29.0 [27.3, 30.7]	

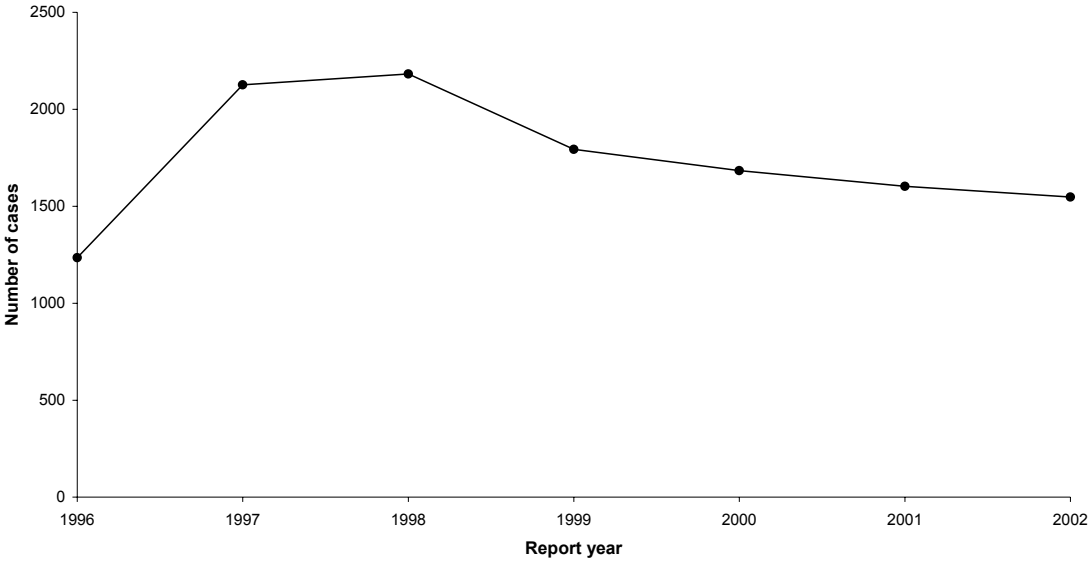
* Directly standardised to the NZ population

More than for any other notifiable disease, the geographical, age and ethnic distribution of gastroenteritis notifications is affected by local surveillance practices. A better indication of the burden of gastroenteritis and its causal agents is provided by outbreak surveillance data presented later in this report.

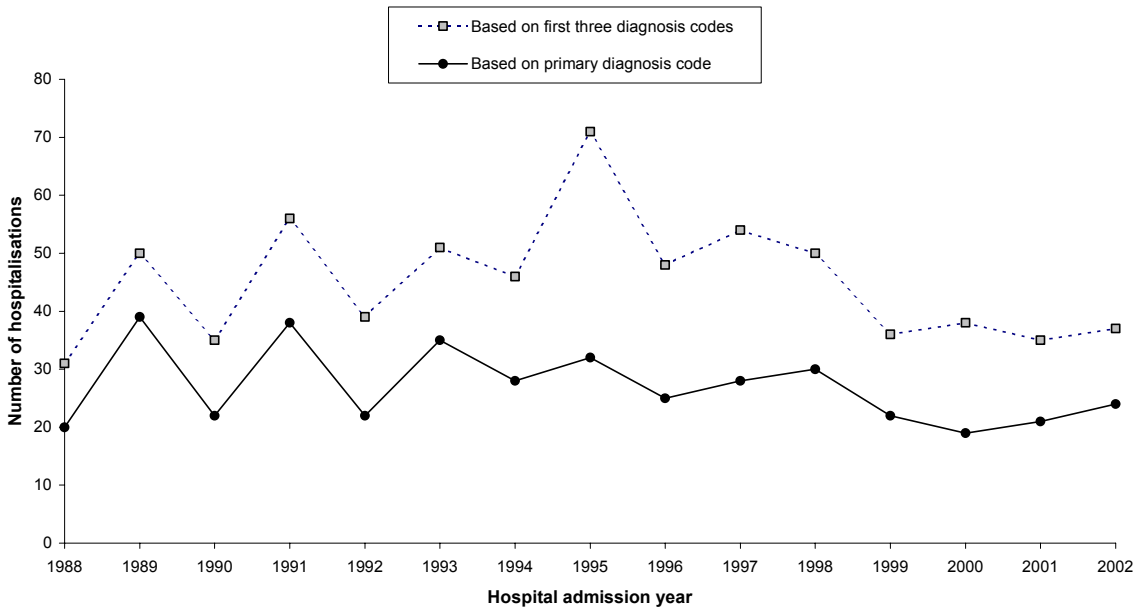
Giardiasis

There were 1548 cases of giardiasis notified in 2002, of whom 98.6% were confirmed. This is the lowest annual notification total since the disease became notifiable. The 2002 rate of 41.4 cases per 100 000 (95% CI: 39.4-43.5) was slightly lower than the 2001 rate of 42.9 (95% CI: 40.8-45.0). Of the 1088 cases for whom hospitalisation status was recorded on EpiSurv, 28 (2.6%) were hospitalised. According to hospital discharge data, hospitalisations due to giardiasis (ICD9 code 007.1) totalled 24 in 2002, and have fluctuated between 20 and 40 cases annually over the past 15 years. The following graphs show (i) the number of cases of giardiasis notified each year since 1996, and (ii) the hospitalisations each year since 1988.

Giardiasis notifications by year, 1996 - 2002

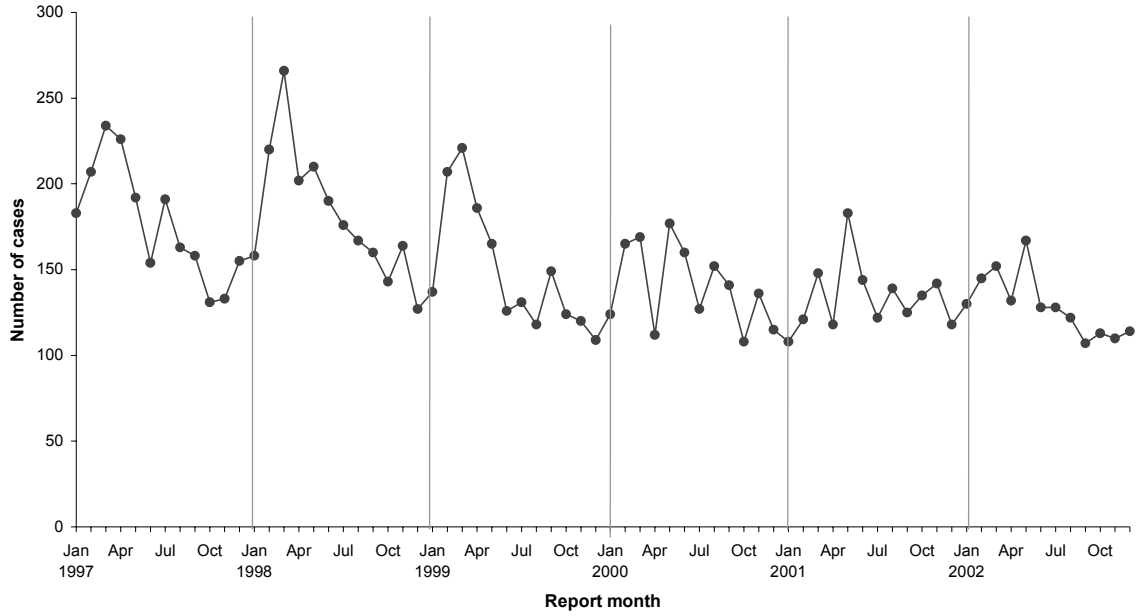


Gidardiasis hospitalisations by year, 1988 - 2002



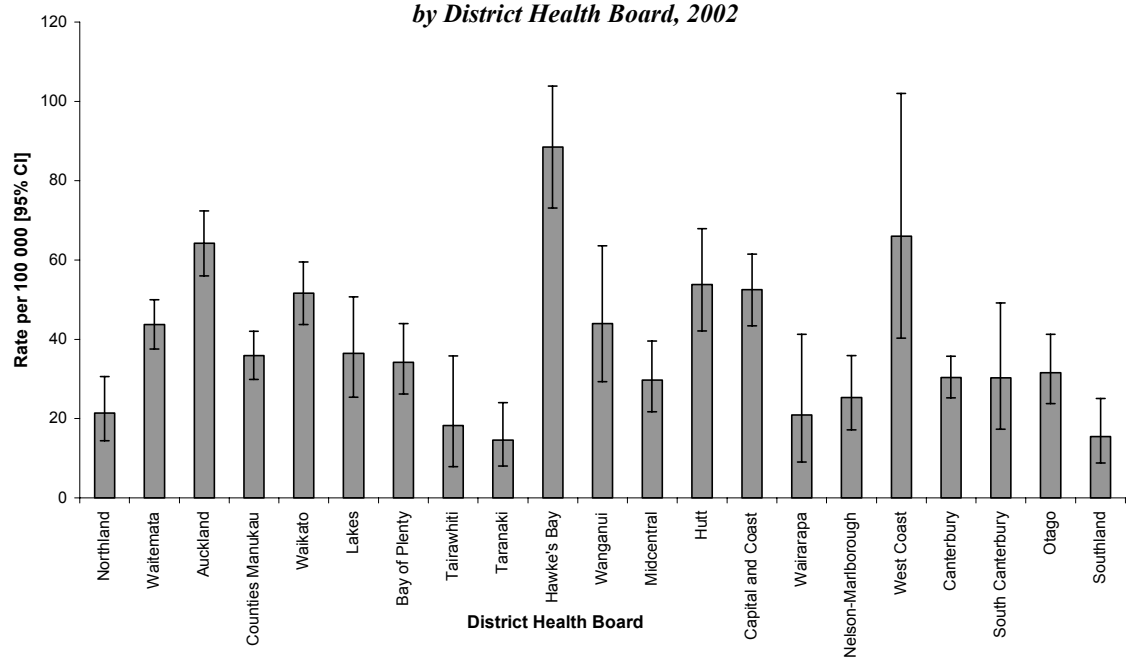
A total of 11 giardiasis outbreaks involving 68 cases was reported in 2002. The following graph shows the number of notified cases each month since January 1997. As in 2000 and 2001, and in contrast to 1998 and 1999, there was little seasonality apparent in 2002 notifications.

Giardiasis notifications by month, 1997 - 2002



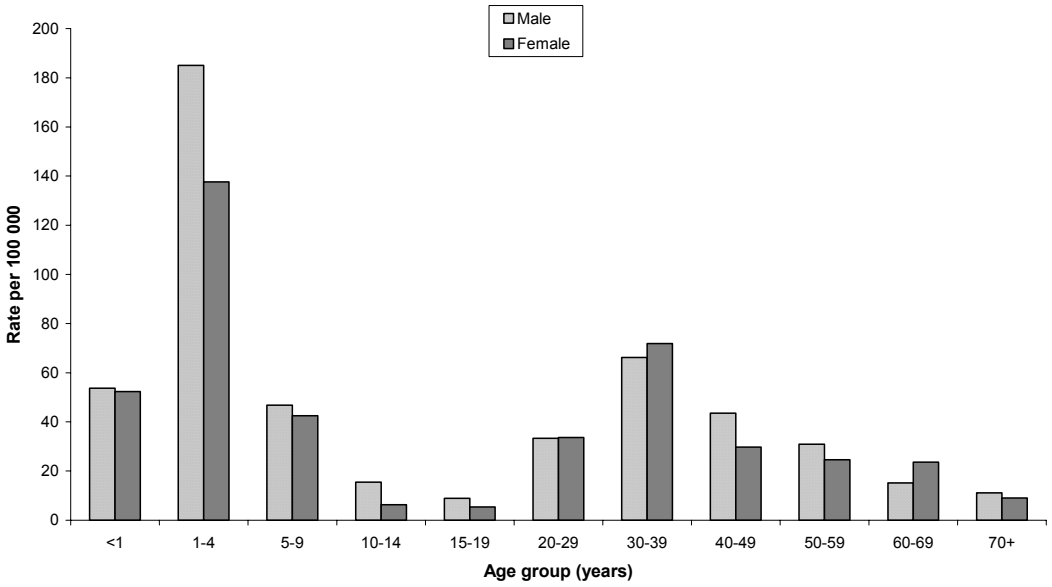
The rate of giardiasis varied throughout the country in 2002. Hawke's Bay DHB recorded the highest rate of 88.5 per 100 000 (95% CI: 73.1-103.9). Rates higher than the national average were also reported by West Coast (66.0 95% CI: 40.3-102.0), Auckland (64.2, 95% CI: 56.0-72.4), Capital and Coast (52.5, 95% CI: 43.4-61.5) and Waikato (51.6, 95% CI: 43.7-59.5) District Health Boards. The following graph shows crude notification rates by District Health Board.

Giardiasis crude notification rates by District Health Board, 2002



Among all enteric notifiable diseases, contact with faecal matter or vomit was most frequently reported by giardiasis cases (45.9% of cases for whom the information was recorded, 95% CI: 42.0-49.8%). After gastroenteritis, contact with other symptomatic cases was also most frequently reported by giardiasis cases (37.2%, 95% CI: 33.6-41.0%). Of the 722 cases with this information recorded, 251(34.8%) attended school, pre-school or childcare. There were two peaks in the age-specific rates of giardiasis notifications: the largest one in the '1 to 4 years' age group (165.2, 95% CI: 148.0-182.3) and a smaller yet significant peak in the '30 to 39 years' age group (70.0, 95% CI: 63.2-76.9).

Giardiasis notification rates, 2002 by age and sex



Rates of giardiasis were highest in the 'Other' ethnic group, with a rate of 38.8 per 100 000, and next highest in the European ethnic group (37.7). Rates were low among Maori and Pacific peoples. Of note is the fact that 48 (50%) of the 96 notified cases of 'Other' ethnicity were either of Indian ethnicity or had travelled to India during the incubation period. The tables below illustrate the age and ethnic distribution of cases.

Giardiasis notifications and age-specific rates by ethnicity, 2002

Ethnicity	Age group in years											
	<1	**1-4**	**5-9**	**10-14**	**15-19**	**20-29**	**30-39**	**40-49**	**50-59**	**60-69**	**70+**	**Total**
European	20	236	82	18	10	89	260	132	74	34	28	986
	67.2	194.0	48.5	10.1	6.2	29.1	65.5	33.2	22.1	14.7	9.7	37.7
Maori	2	32	3	1	0	7	6	5	3	1	0	60
	14.3	59.8	4.5	1.6	0.0	8.5	7.7	8.6	9.0	5.1	0.0	11.4
Pacific peoples	0	3	0	0	1	0	0	1	1	0	0	6
	0.0	15.6	0.0	0.0	5.5	0.0	0.0	4.6	7.5	0.0	0.0	3.0
Other	1	15	8	6	6	29	16	9	3	3	0	96
	26.9	106.9	43.5	29.8	21.4	66.7	35.0	23.8	15.4	27.2	0.0	38.8
Unknown	7	71	35	8	2	44	122	49	37	17	4	400
Total	30	357	128	33	19	169	404	196	118	55	32	1548
	54.9	165.2	44.7	11.3	7.2	34.7	70.0	36.5	28.2	19.5	9.9	41.4

Number of cases
Rate per 100 000

Giardiasis - crude and age-standardised rates by ethnicity, 2002

Ethnicity	Crude rate [95% CI]	Age-standardised rate* [95% CI]
European	37.7 [35.4, 40.1]	40.2 [37.7, 42.8]
Maori	11.4 [8.7, 14.7]	9.1 [6.8, 13.3]
Pacific peoples	3.0 [1.1, 6.5]	2.8 [0.9, 10.3]
Other	38.8 [31.4, 47.4]	35.0 [28.2, 44.7]
Total	41.4 [39.4, 43.5]	

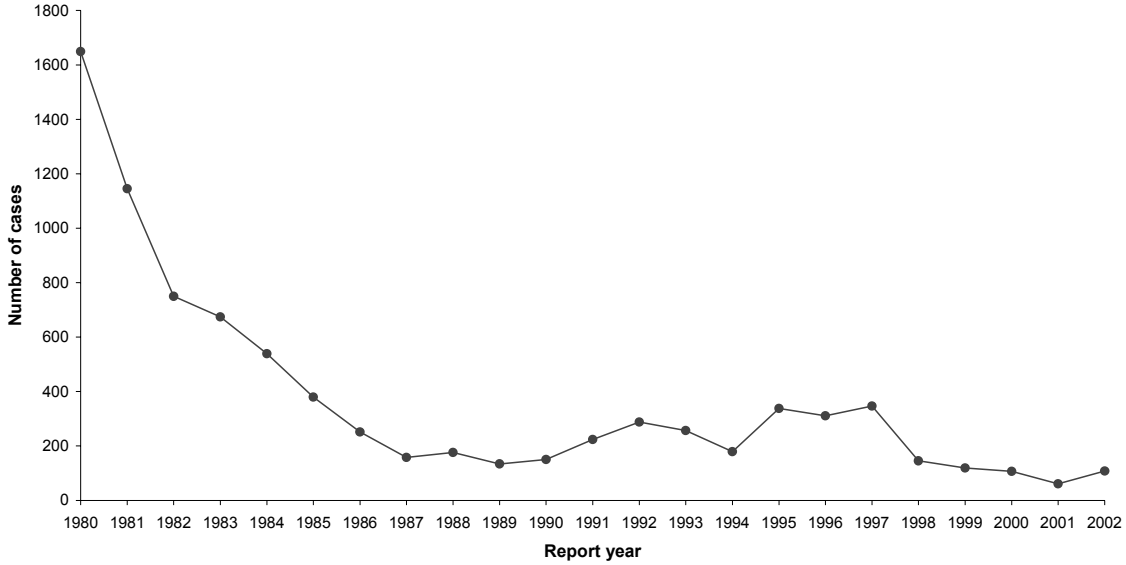
* Directly standardised to the NZ population

These surveillance data suggest a relatively stable incidence of giardiasis in New Zealand. The high rates in young children are consistent with the hypothesis of person-to-person transmission between children at early childhood centres and with further transmission to parents during nappy changing and other forms of contact. [43] Consistent with this, a relatively high proportion of cases (46%) report contact with children in nappies, sewage or other types of faecal matter or vomit during the incubation period.

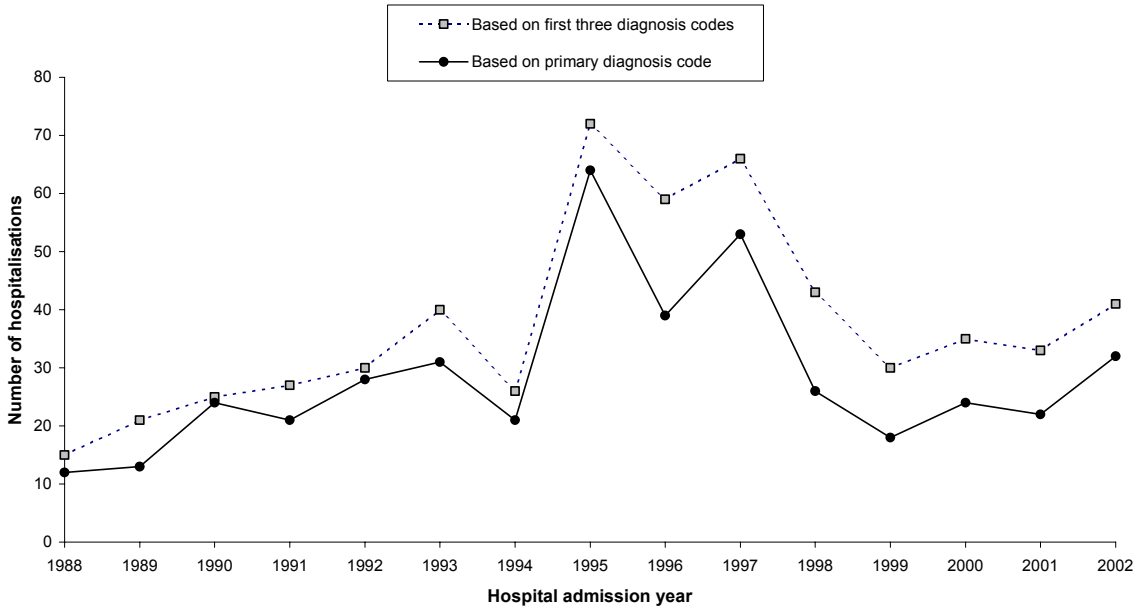
Hepatitis A

A total of 108 cases of hepatitis A was notified in 2002. The 2002 rate of 2.9 per 100 000 (95% CI: 2.3-3.4) 100 000 was significantly higher than the 2001 rate of 1.6 (95% CI: 1.2-2.1). Of the 105 cases for whom hospitalisation status was recorded, 24 (22.9%) were hospitalised. According to hospital discharge data, hospitalisations for hepatitis A (ICD9 codes 070.0, 070.1) in 2002 totalled 32, compared to 22 the previous year. The following graphs show (i) the number of notified cases of hepatitis A each year since 1980, and (ii) the hospitalisations each year since 1988.

Hepatitis A notifications by year, 1980 - 2002



Hepatitis A hospitalisations by year, 1988 - 2002

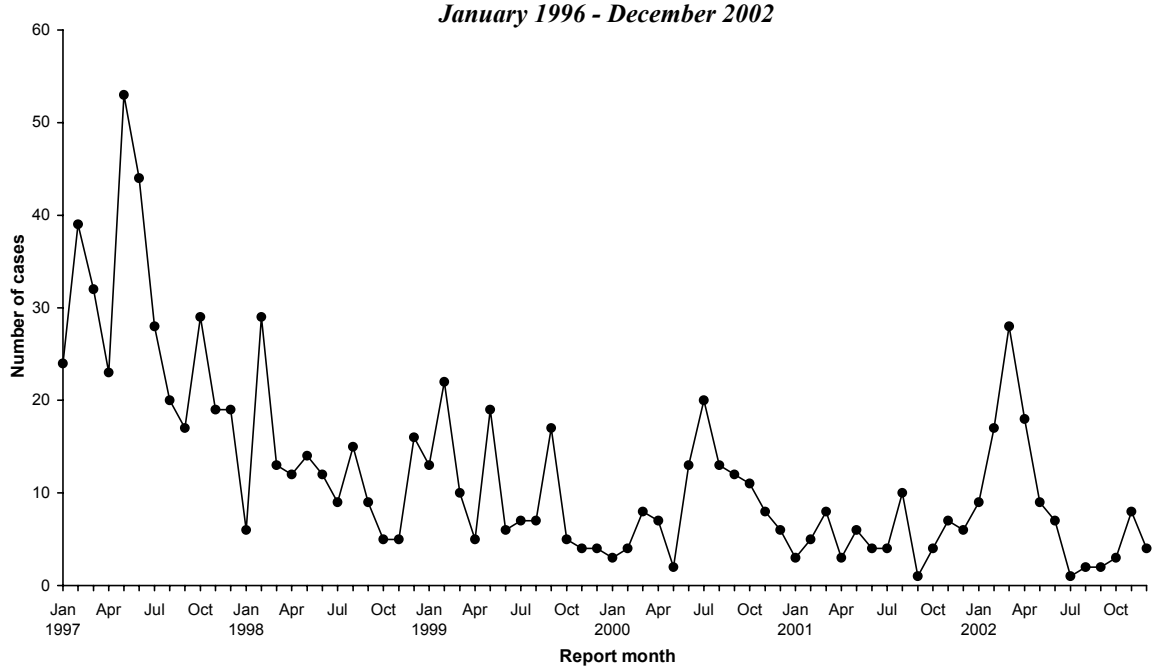


Four outbreaks of hepatitis A involving a total of 34 cases were reported in 2002, compared to three outbreaks and 11 outbreak-associated cases the previous year. It is unclear exactly how many of the outbreak-associated cases in 2002 were also individually notified. The largest outbreak in 2002, linked to the consumption or raw blueberries, occurred across multiple health districts during February, March and April. Half (50%) the 40 cases individually notified during this time had consumed blueberries during the incubation period. Throughout the year a total of 31 (28.7%) notified cases reported either consuming blueberries or visiting a blueberry farm implicated in the outbreak. [44]

A national investigation into the increased number of hepatitis A cases commenced in March 2002, following recognition of a possible dispersed common source of infection among Auckland cases. Cases were eligible for inclusion in the epidemiologic investigation if notified with hepatitis A between 1 January and 10 April 2002 and aged over 15 years. The study included 39 cases and 79 controls. Analysis of the data showed that illness was significantly associated with consumption of raw blueberries (odds ratio = 7.60; 95% CI: 2.64-22.41), and was not confounded by age or other factors. Illness was not significantly associated with any other exposures. An environmental investigation of the outbreak was also started in March. Blueberry brands consumed by hepatitis A cases were traced back to their sources, and subsequent hazard analysis critical control point (HACCP) investigations were performed to identify potential points of blueberry contamination.

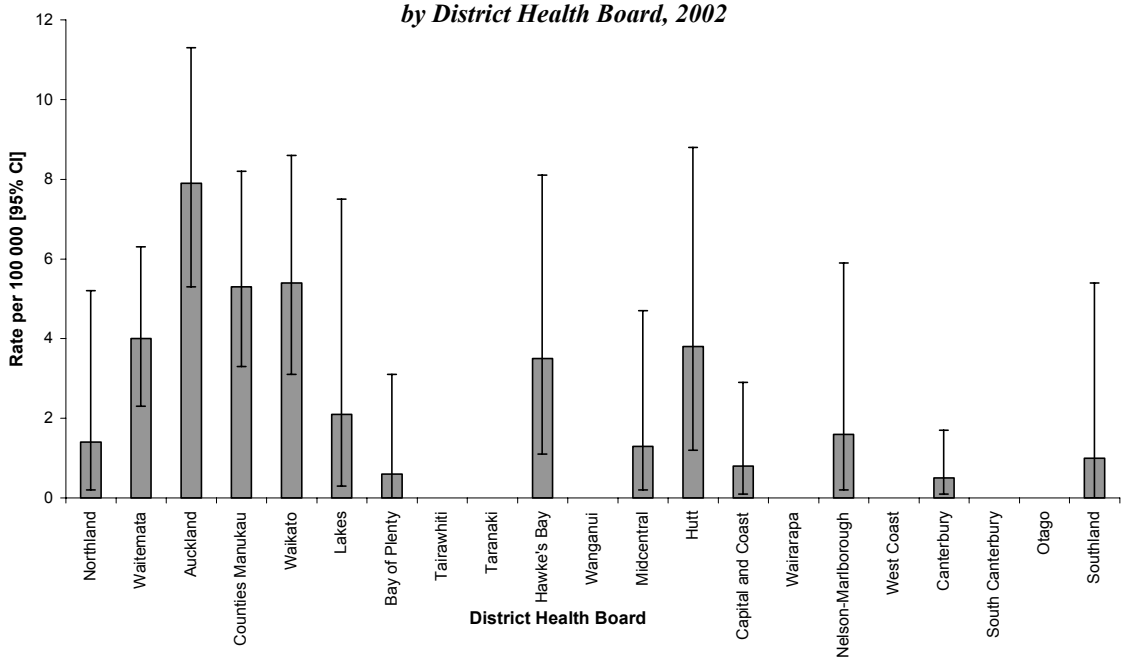
The following graph shows the number of cases of hepatitis A notified each month since January 1996. A sharp peak in incidence in March 2002 was due to the dispersed outbreak described above.

Hepatitis A notifications by month, January 1996 - December 2002



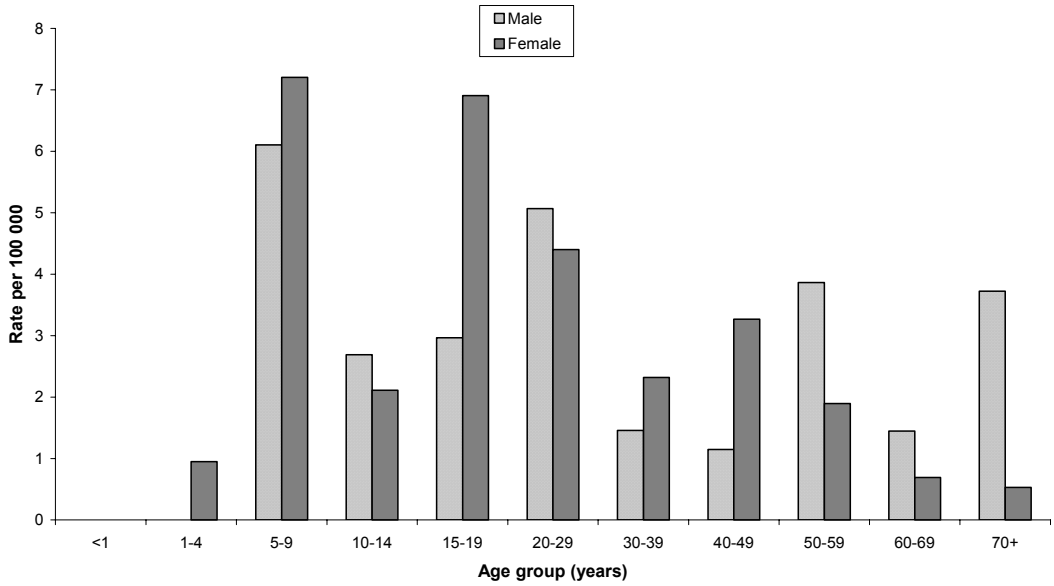
Rates of hepatitis A varied throughout the country, as shown by the following bar chart. The rate reported by Auckland DHB (7.9 per 100 000, 95% CI: 5.3-11.3) was significantly higher than the national average of 2.9 per 100 000. High rates were also reported in Waikato (5.4), Counties Manukau (5.3), Hutt (3.8) and Hawke's Bay (3.5) District Health Boards.

Hepatitis A crude notification rates by District Health Board, 2002



Rates significantly higher than the overall rate of 2.9 per 100 00 were seen in children aged between five and nine years, with a rate of 6.6 per 100 000 (95% CI: 4.0-10.4). Age-specific rates were also high in the '15 to 19 years' (4.9, 95% CI: 2.6-8.4) and the '20 to 29 years' (4.7, 95% CI: 3.0-7.1) age groups, although due to the wide confidence intervals it is difficult to tell if this is significant. The following bar chart shows notification rates of hepatitis A by age and sex. Note that rates based on small numerators may be unstable and imprecise.

Hepatitis A notification rates, 2002 by age and sex



The crude notification rate among Pacific peoples (13.5 per 100 000, 95% CI: 8.9-19.6) was significantly higher than in all other ethnic groups. The second highest rate (4.8) was experienced by people of 'Other' ethnicity. Age-standardised rates were also highest in Pacific peoples. Just one case of Pacific ethnicity and one case of 'Other' ethnicity were part of the outbreak linked to contaminated blueberries. The tables below illustrate the age and ethnic distribution of cases.

Hepatitis A notifications and age-specific rates by ethnicity, 2002

Ethnicity	Age group in years											
	<1	**1-4**	**5-9**	**10-14**	**15-19**	**20-29**	**30-39**	**40-49**	**50-59**	**60-69**	**70+**	**Total**
European	0	0	7	1	2	6	7	10	11	3	6	54
	0.0	0.0	4.1	0.6	1.2	2.0	1.8	2.5	3.3	1.3	2.1	2.1
Maori	0	0	2	0	0	1	0	1	1	0	0	5
	0.0	0.0	3.0	0.0	0.0	1.2	0.0	1.7	3.0	0.0	0.0	0.9
Pacific peoples	0	1	9	4	5	7	1	0	0	0	0	27
	0.0	5.2	38.1	18.5	27.6	20.7	3.2	0.0	0.0	0.0	0.0	13.5
Other	0	0	1	1	3	5	2	0	0	0	0	12
	0.0	0.0	5.4	5.0	10.7	11.5	4.4	0.0	0.0	0.0	0.0	4.8
Unknown	0	0	0	1	3	4	1	1	0	0	0	10
Total	0	1	19	7	13	23	11	12	12	3	6	108
	0.0	0.5	6.6	2.4	4.9	4.7	1.9	2.2	2.9	1.1	1.9	2.9

Number of cases
Rate per 100 000

Hepatitis A - crude and age-standardised rates by ethnicity, 2002

Ethnicity	Crude rate [95% CI]	Age-standardised rate* [95% CI]
European	2.1 [1.6, 2.7]	2.0 [1.5, 2.6]
Maori	0.9 [0.3, 2.2]	1.0 [0.3, 4.3]
Pacific peoples	13.5 [8.9, 19.6]	9.8 [6.4, 17.7]
Other	4.8 [2.5, 8.5]	3.7 [1.9, 9.5]
Total	2.9 [2.3, 3.4]	

* Directly standardised to the NZ population

The following table provides a summary of risk factor information for hepatitis A in 2002. Note that some cases had more than one risk factor recorded. In 2002, the suspected or definite source of infection for over half (67.3%) the cases (for whom this information was recorded), was contaminated food or water. This proportion is significantly higher than during 2001, when only 27.8% of cases were thought to be linked to contaminated food or water. It should also be noted that during 2002, although 12 (17%) of the 70 cases for whom the information was recorded, reported either household or sexual contact with a confirmed case, a total of 18 cases had some form of contact with a confirmed case of the disease.

Risk factors associated with hepatitis A, 2002

Risk Factor	Yes	No	Unknown	Proportion[1]
Consumed suspected/definite contaminated food/water	37	18	53	67.3%
Overseas travel during incubation period	28	51	29	35.4%
Household contact with confirmed case	11	56	41	16.4%
Occupational exposure to human sewage	4	57	47	6.6%
Sexual contact involving possible faecal-oral transmission or contact	2	59	47	3.3%

[1] "Proportion" refers to the percentage of cases who answered "yes" out of the total number of cases where this information was known

Among the 28 cases who reported overseas travel during the incubation period, the most commonly implicated countries were Fiji (5 cases), Samoa (4) and Australia (4). One case each had been in Tonga, Vanuatu and the Solomon Islands, seven cases had been in a variety of Asian countries and four cases in Europe or America. Excluding recent visitors, migrants, and refugees to New Zealand, the highest rates of infection occurred among travellers to Oceania (1.8 cases per 100 000 visits), in particular Samoa (21.0 per 100 000 visits) and Fiji (7.5 per 100 000 visits).

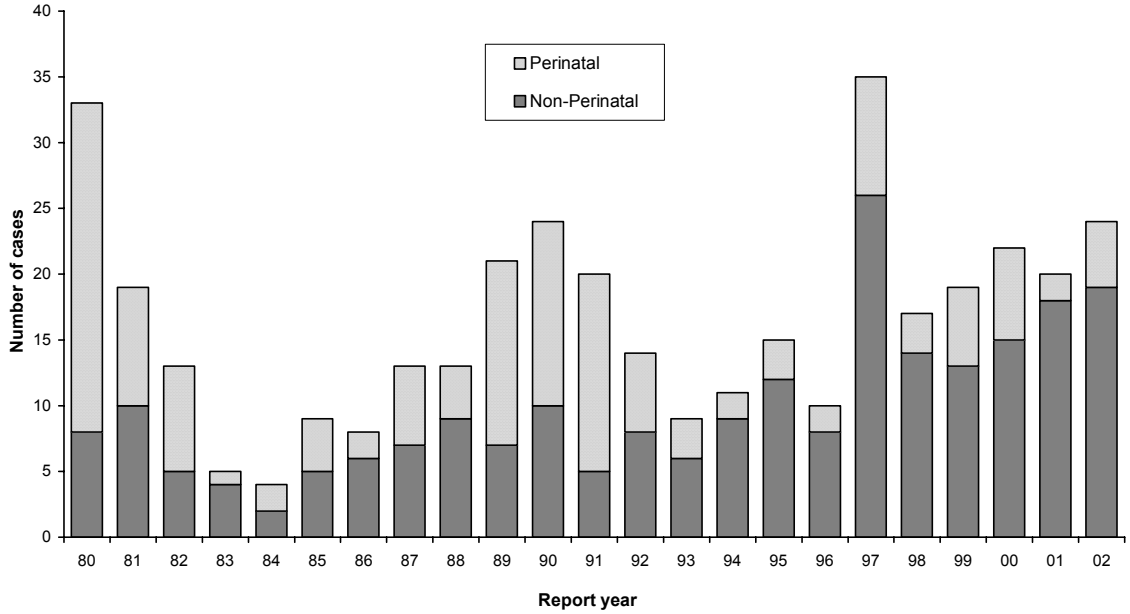
Since the mid-1980s, New Zealand has become a low incidence country for hepatitis A infection. The relatively large common-source food borne outbreak described here was unusual, with the last similar outbreak being in 1996.[45] Excluding such events, the most common sources of infection are now overseas travel and secondary household transmission. These modes of transmission can be controlled by, respectively, pre-travel hepatitis A vaccination and post-exposure prophylaxis of contacts.

Listeriosis

A total of 19 listeriosis cases was notified in 2002, five of whom were perinatal cases and 14 non-perinatal cases. The 2002 rate of 0.5 per 100 000 was similar to the rate in 2001 when 18 cases were notified. During 2002, the ESR Enteric Reference Laboratory received 19 listeriosis isolates, all of which could be matched to EpiSurv notifications. Eight cases were identified as serotype 04 and eleven as serotype 1/2.

All 13 cases in 2002 for whom hospitalisation information was recorded on EpiSurv were hospitalised. Three deaths occurred in perinatal cases. Eleven (78.6%) of the 14 non-perinatal cases were aged 50 or over, nine (64.3%) had an underlying illness, and four (28.6%) were receiving immunosuppressive drugs. Six cases were admitted to hospital for treatment of an illness other than listeriosis. No outbreaks of listeriosis were reported in 2002. The following chart shows the number of notified cases of listeriosis each year since 1980.

Listeriosis notifications by year, 1980 -2002



Age-specific rates of non-perinatal listeriosis were highest in the '70+ years' age group with a rate of 1.6 per 100 000, followed by the '60 to 69 years' age group, with a rate of 1.4. The majority (75%) of cases for whom ethnicity was recorded was of European ethnicity. Canterbury and Waitemata District Health Boards recorded the greatest number of cases with four cases each. Other notifications were also received from Auckland (3), Counties Manukau (2), Bay of Plenty (2), Waikato (1), Midcentral (1), Hutt (1) and Southland (1) DHBs.

The overall incidence of listeriosis has remained relatively constant in New Zealand over the past two decades. One change has been an increasing proportion of non-perinatal cases.

Norovirus

In August 2002, the International Committee on Viral Taxonomy reclassified the Norwalk-like virus (NLV) group into a new Genus *Norovirus* in the Caliciviridae Family. In New Zealand, Noroviruses are the most widely recognised cause of outbreaks of foodborne gastroenteritis and are monitored by combined laboratory and epidemiological surveillance. Genotyping of Norovirus strains commenced in New Zealand in 1996, and the majority of strains identified since are similar to those occurring overseas.

There were 93 outbreaks[a] or clusters of laboratory-confirmed Norovirus-associated gastroenteritis during 2002. Norovirus was also confirmed in a number of individual cases not known to be linked to any outbreaks. Outbreaks were reported every month during 2002, with 35.5% of outbreaks occurring in the final quarter. In 2002, extensive spread of Noroviruses through institutionalised settings was observed both in New Zealand and

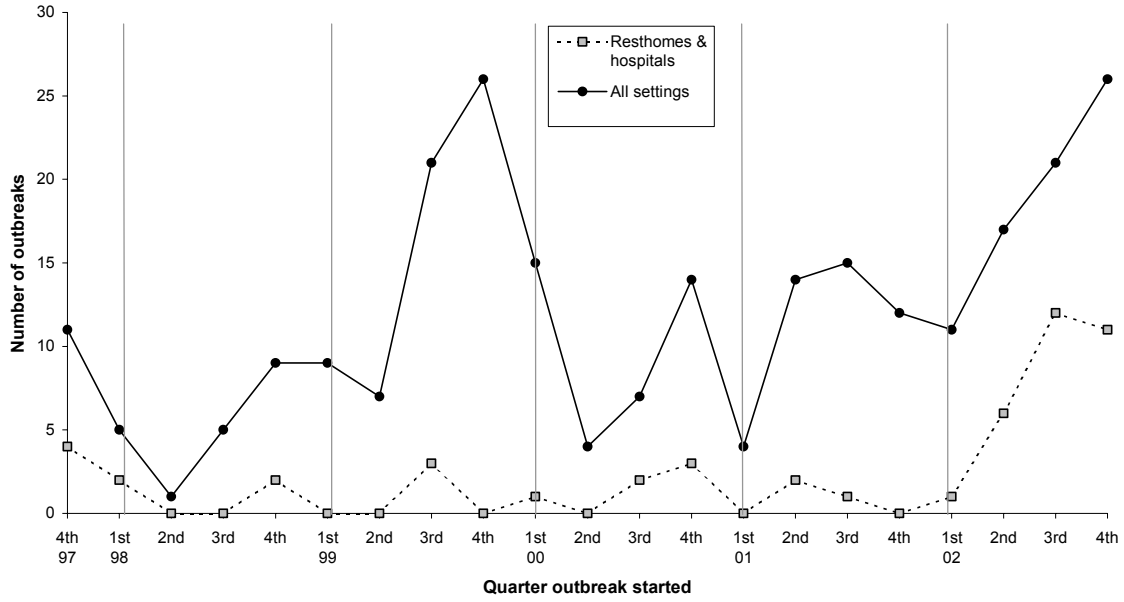
[a] These outbreaks were identified using combined surveillance methods. Only 76 norovirus-related outbreaks were recorded in the Outbreak Module of EpiSurv during 2002.

overseas. In New Zealand, 34 institutional rest home and hospital outbreaks were reported.[a] The majority of these (29, 85.3%) were caused by the GII/1,4,8 'global strain cluster' and occurred in the winter months. Eight outbreaks occurred in child-related settings. Of these, four occurred in childcare centres or commercial children's play centres, two in school camps and two in school hostels. Other settings included restaurants, cafes, takeaway bars and catered functions and several family groups around the country. The extent of Norovirus infection originating in the home is unknown.

A wide range of genotypes circulated during 2002. The predominant genotype again was the common 'Lordsdale virus Global strain cluster', GII/1,4,8 (58, 62.4%). The other common genotypes were GII/2 (Melksham virus) and GII/6,7,9 (Napier, Florida and Gwynedd viruses). Uncommon genogroup I strains GI/1 (Norwalk virus) GI/3 (Desert Shield virus) and GI/4 (Chiba virus) are still circulating in New Zealand. A previously rare genotype, GII/5 (White River virus) was identified from five outbreaks. Only one strain of Mexico virus (GII/3) was identifed in 2002 and it was associated with the consumption of imported oysters. This genotype has been linked with several oyster-related outbreaks in previous years but has not been identified in New Zealand since December 2000. Imported oysters were implicated in a total of four outbreaks. Noroviruses were identified in one imported oyster sample tested. For the majority of outbreaks, person to person transmission was the likely transmission route, with either food or foodhandling implicated in at least 30 outbreaks.

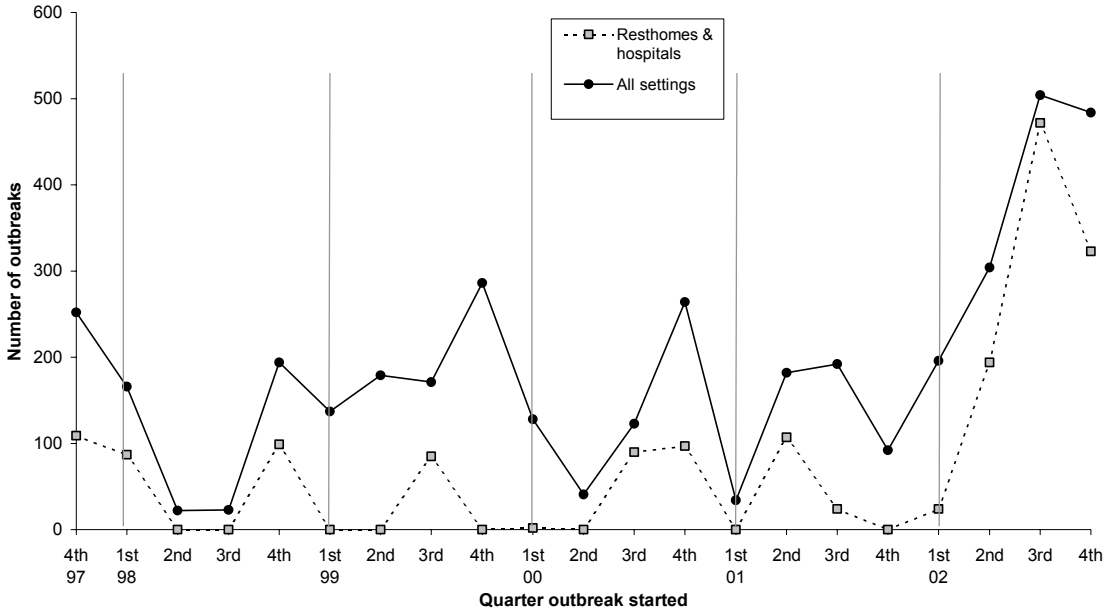
The following graphs illustrate the number of Norovirus outbreaks recorded in the Outbreak Module of EpiSurv, each quarter since 1997. They should be interpreted with caution due to the possible incompleteness of EpiSurv outbreak data. These data suggest some seasonality in norovirus infections, with the highest incidence in the final quarter in most years. The most important settings for outbreaks caused by this organism remain rest-homes and restaurants.[46]

Number of Norovirus outbreaks reported each quarter by setting, 1997-2002



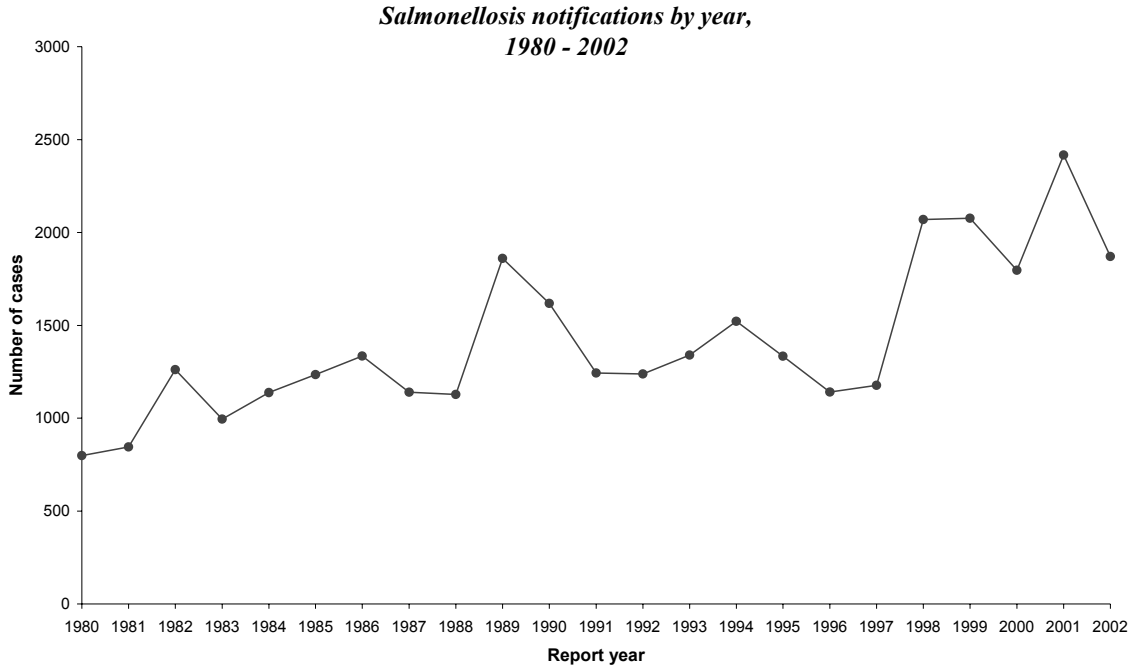
[a] The total of 34 was obtained from combined surveillance methods. Slightly fewer outbreaks were recorded in the Outbreak Module of EpiSurv

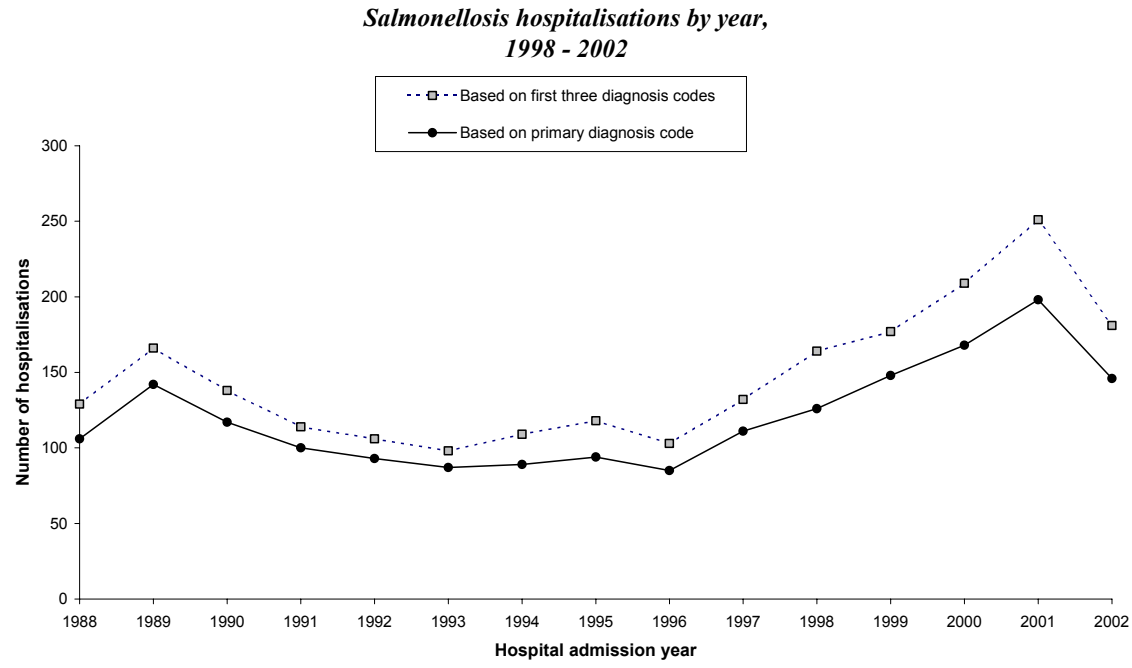
Total number of cases attributed to Norovirus outbreaks each quarter by setting, 1997-2002



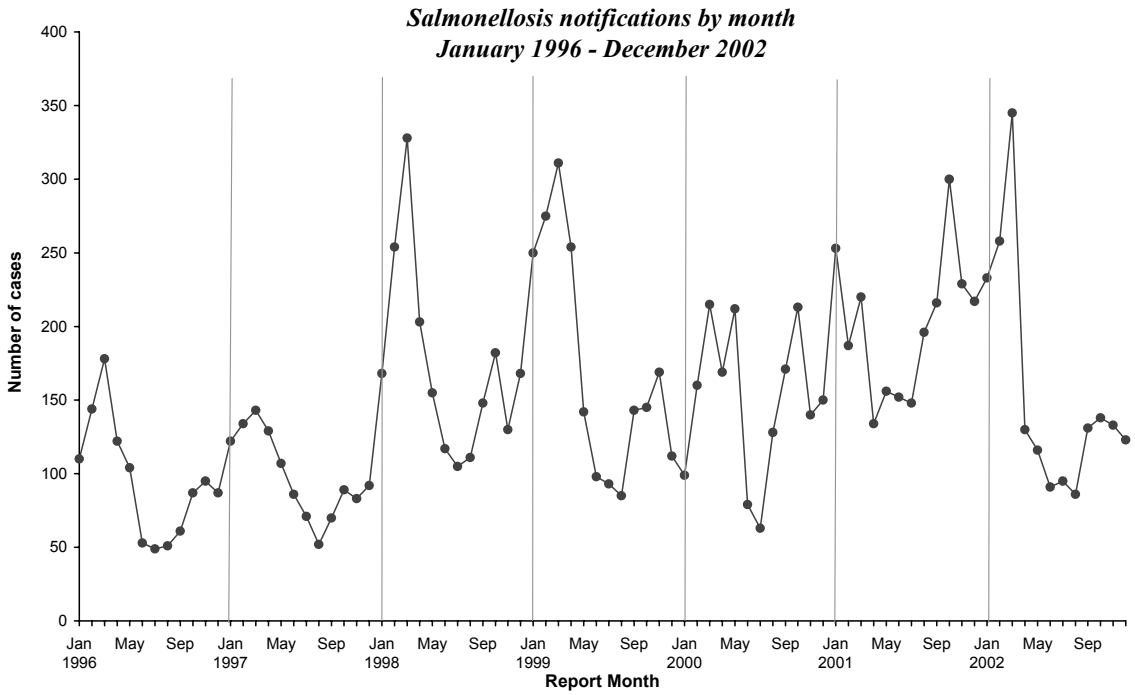
Salmonellosis

There were 1870 cases of salmonellosis notified in 2002, representing a notification rate of 50.0 per 100 000 (95% CI: 47.8-52.4), significantly lower than the 2001 rate of 64.7(95% CI: 62.1-67.3). Based on the matching of 2002 notifications and isolates received by the Enteric Reference Laboratory (ERL) at ESR, 1780 (95.2%) notified cases were laboratory-confirmed. A total of 2019 (non-typhoidal) isolates were received by ERL in 2002, implying that the sensitivity of the notification system in 2002 was around 88%. Of the 1473 cases for whom hospitalisation status was recorded on EpiSurv, 206 (14.0%) were hospitalised. According to hospital discharge data, hospitalisations due to salmonellosis (ICD9 code 003) dropped to146 in 2002, from a peak of 198 the previous year. The following graphs show (i) the number of notified cases each year, and (ii) hospitalisations each year since 1988.

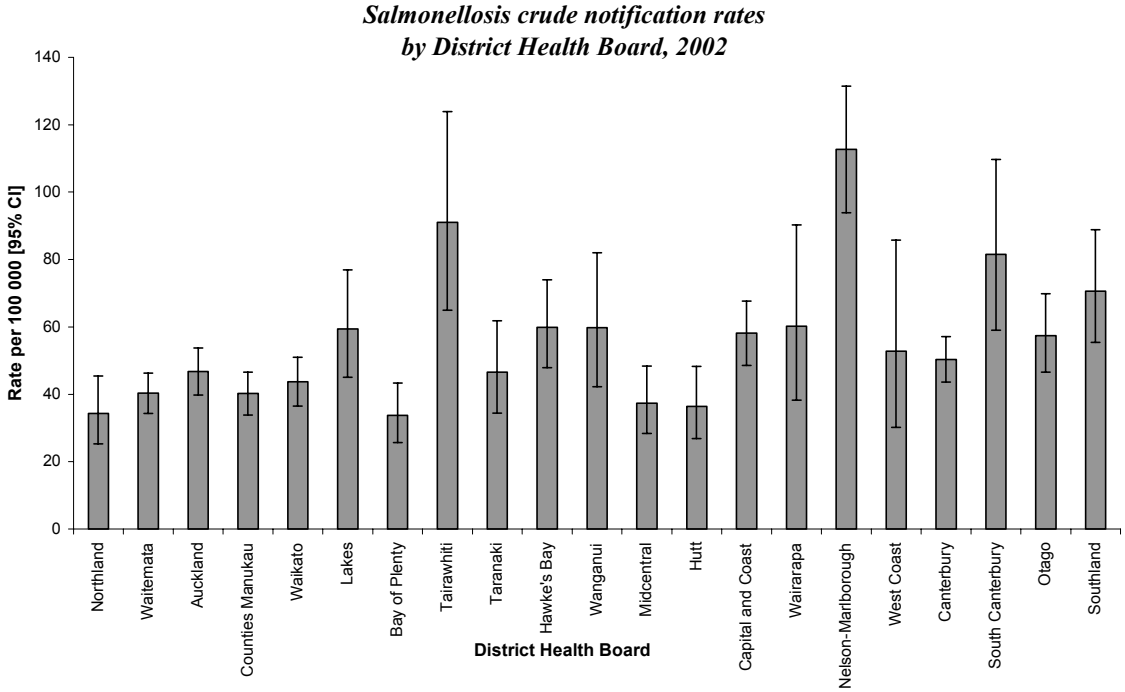




The following graph shows the number of cases notified each month since January 1996. During 2002, salmonellosis notifications peaked in March, partially as a result of several large South Island outbreaks of *Salmonella* Typhimurium phage type 1. There was also a smaller peak in the spring (September through November) led by a rise of Salmonella Brandenburg cases in Otago and Southland during these months. As in previous years, incidence was low during the 2002 winter.

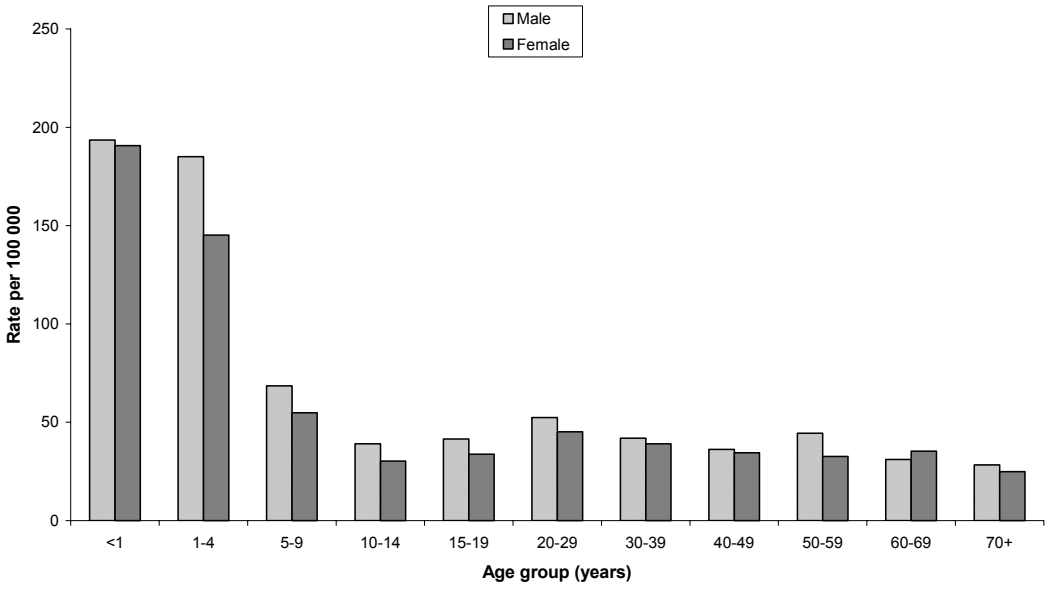


A total of 35 salmonellosis outbreaks, involving 249 cases, was reported in 2002. Of these, 20 (57%) were reported from the Auckland region. It is possible that incidence rates are understated in the Auckland region due to the policy of not individually notifying outbreak-associated cases. The following bar chart shows the notification rates by District Health Board in 2002. The highest rate of 112.7 per 100 000 was recorded in Nelson-Marlborough DHB. Rates significantly higher than the national average of 50.0 were seen in Tairawhiti (91.0), South Canterbury (81.5) and Southland (70.6) District Health Boards.



Age-specific rates of 197.7 and 166.1 per 100 000 for the 'less than one year' and the '1 to 4 years' age groups respectively, were significantly higher than for all other age groups. The rate of 62.2 (95% CI: 53.1-71.3) in the '5 to 9 years' age group was also significantly higher than the overall rate of 50.0 (95% CI: 47.8-52.4). The following bar chart compares notification rates by age and sex.

Salmonellosis notification rates, 2002 by age and sex



The following tables illustrate the age and ethnic distribution of cases. Both crude (48.0 per 100 000) and age-standardised (51.1) rates were significantly higher in the European ethnic group than in all other ethnic groups. Rates were next highest in those of 'Other' ethnicity.

Salmonellosis notifications and age-specific rates by ethnicity, 2002

Ethnicity	Age group in years											
	<1	**1-4**	**5-9**	**10-14**	**15-19**	**20-29**	**30-39**	**40-49**	**50-59**	**60-69**	**70+**	**Total**
European	54	227	119	71	67	165	158	135	115	68	70	1255
	181.5	186.6	70.4	40.0	41.7	54.0	39.8	33.9	34.4	29.3	24.4	48.0
Maori	14	35	11	4	8	21	16	11	11	6	5	142
	100.1	65.4	16.6	6.4	16.1	25.5	20.6	19.0	33.1	30.7	51.6	27.0
Pacific peoples	2	6	4	1	1	1	7	3	3	2	2	32
	38.8	31.3	16.9	4.6	5.5	3.0	22.1	13.8	22.5	26.8	45.3	16.0
Other	10	23	9	2	2	10	14	7	9	0	0	87
	269.0	164.0	48.9	9.9	7.1	23.0	30.6	18.5	46.1	0.0	0.0	35.2
Unknown	28	68	35	23	24	44	39	36	25	18	10	354
Total	108	359	178	101	102	241	234	192	163	94	87	1870
	197.7	166.1	62.2	34.7	38.5	49.5	40.6	35.7	39.0	33.3	27.0	50.0

Number of cases
Rate per 100 000

Salmonellosis - crude and age-standardised rates by ethnicity, 2002

Ethnicity	Crude rate [95% CI]	Age-standardised rate* [95% CI]
European	48.0 [45.4, 50.7]	51.1 [48.3, 54.1]
Maori	27.0 [22.5, 31.4]	27.9 [22.4, 35.2]
Pacific peoples	16.0 [10.9, 22.6]	18.6 [11.5, 30.2]
Other	35.2 [28.2, 43.4]	34.0 [27.1, 43.9]
Total	50.0 [47.8, 52.3]	

* Directly standardised to the NZ population

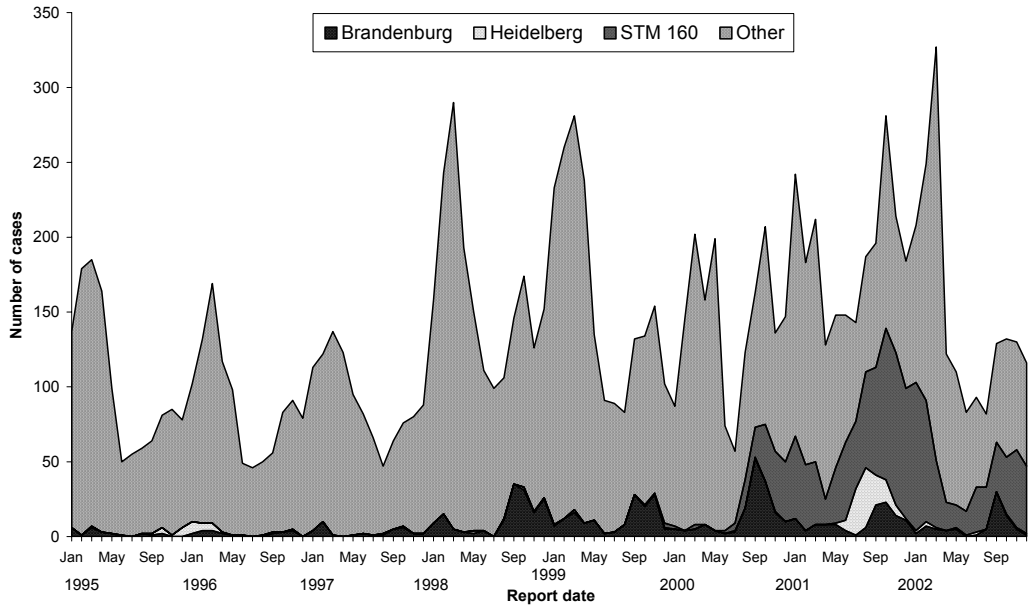
The table below shows the number of cases of selected Salmonella types, among notified cases who were matched to the ESR laboratory records. *Salmonella* Heidelberg, which was prominent in 2001, declined dramatically in 2002. The incidence of most Typhimurium definitive types also declined, with the exception of *S.*Typhimuium DT1. Notifications of *S.* Infantis, *S.* species 6,7:k and *S.* Montevideo reached a peak in 2002. DT160 remained the most common single type, despite a decline in incidence.

Selected Salmonella serotypes and subtypes of laboratory-confirmed salmonellosis notifications, from 1995 to 2002

Subtype	1995	1996	1997	1998	1999	2000	2001	2002
S. Typhimurium								
DT160	0	0	0	0	4	169	718	501
DT1	194	132	95	201	174	123	157	214
DT135	67	74	114	296	459	370	240	141
DT156	135	105	99	149	151	91	98	80
DT101	38	39	62	258	131	112	65	41
Other or unknown	296	262	282	388	287	260	230	176
Total	730	612	652	1292	1206	1125	1508	1153
S. Enteritidis								
PT9a	62	52	66	83	106	65	66	70
PT4	83	62	51	49	54	42	22	30
Other or unknown	29	25	24	32	27	20	66	39
Total	174	139	141	164	187	127	154	139
S. Infantis	25	43	29	21	71	32	65	86
S. Brandenburg	23	23	35	156	149	165	120	79
*S.*species 6,7:k	2	3	1	1	4	7	9	34
S. Saintpaul	22	37	33	32	31	21	14	33
S. Thompson	4	4	3	2	5	8	16	20
S. Montevideo	5	4	5	13	6	14	3	19
S. Heidelberg	14	22	2	6	5	3	122	13
Other or unknown serotypes	237	185	192	261	268	196	255	204
TOTAL	**1236**	**1072**	**1093**	**1948**	**1932**	**1698**	**2266**	**1780**

The following chart illustrates examples of Salmonella types that have emerged in recent years and their changing contribution to the overall *Salmonella* burden in New Zealand. The contribution of S. Typhimurium DT160 remained significant throughout 2002, whereas the contribution of *Salmonella* Heidelberg declined rapidly. There was a prominent spring peak in the incidence of *Salmonella* Brandenburg in 2002, as in the past five years.

Laboratory-confirmed notified cases of Salmonella, January 1995 - December 2002



The matching of notification with laboratory data enabled the following analysis of laboratory-confirmed salmonellosis notifications in 2002.

Laboratory-confirmed salmonellosis notifications and cases part of reported outbreaks, 2002

Year 2002	Individual notifications				Reported outbreaks[3]	
Subtype	**# notified cases**	**%* overseas**	**%* rural**	**% of total[1]**	**# outbreak cases**	**% of total[2]**
S. Typhimurium						
DT160	501	2%	17%	28%	49	20%
DT1	214	0%	16%	12%	119	48%
DT135	141	3%	10%	8%	2	1%
Other or unknown	297	8%	21%	17%	12	5%
Total	1153	3%	17%	65%	182	73%
S. Enteritidis						
PT9a	70	0%	17%	4%	0	0%
PT4	30	92%	3%	2%	0	0%
Other or unknown	39	81%	8%	2%	0	0%
Total	139	42%	11%	8%	0	0%
S. Infantis	86	7%	8%	5%	0	0%
S. Brandenburg	79	0%	44%	4%	0	0%
*S.*species 6,7:k	34	0%	3%	2%	25	10%
S. Saintpaul	33	0%	24%	2%	0	0%
S. Thompson	20	8%	15%	1%	2	1%
S. Montevideo	19	54%	0%	1%	0	0%
Other or unknown serotypes	217	61%	9%	12%	40[4]	16%
Total	1870	14%	16%	100%	249	100%

* % of those cases for whom the information was recorded
[1] % of total notifications
[2] % of total outbreak cases
[3] includes outbreaks reported on the Outbreak Module of EpiSurv
[4] includes 13 cases from a *S.* Weltevreden outbreak

Outbreaks: S. Typhimurium outbreaks accounted for the majority (73%) of outbreak-associated cases. *S.* Typhimurium DT1 was isolated from only 12% of notified cases, but was identified with 48% of outbreak-related cases. *S.* species 6,7:k accounted for 10% of outbreak-associated cases, although only 2% of notified cases.

Overseas travel: 42% of all *S.*Enteritidis cases and 92% of cases of *S.* Enteritidis PT4 were linked to overseas travel during the incubation period, compared to an overall proportion of 14% for salmonellosis notifications.

Rural/urban split: 44% of *Salmonella* Brandenburg cases, 24% of *Salmonella* Saintpaul cases, 17% of S. Typhimurium cases and 17% of S. Enteritidis PT9a cases resided in areas classified as rural. In comparison, just 12.6% of New Zealand was classified as rural at the last census.

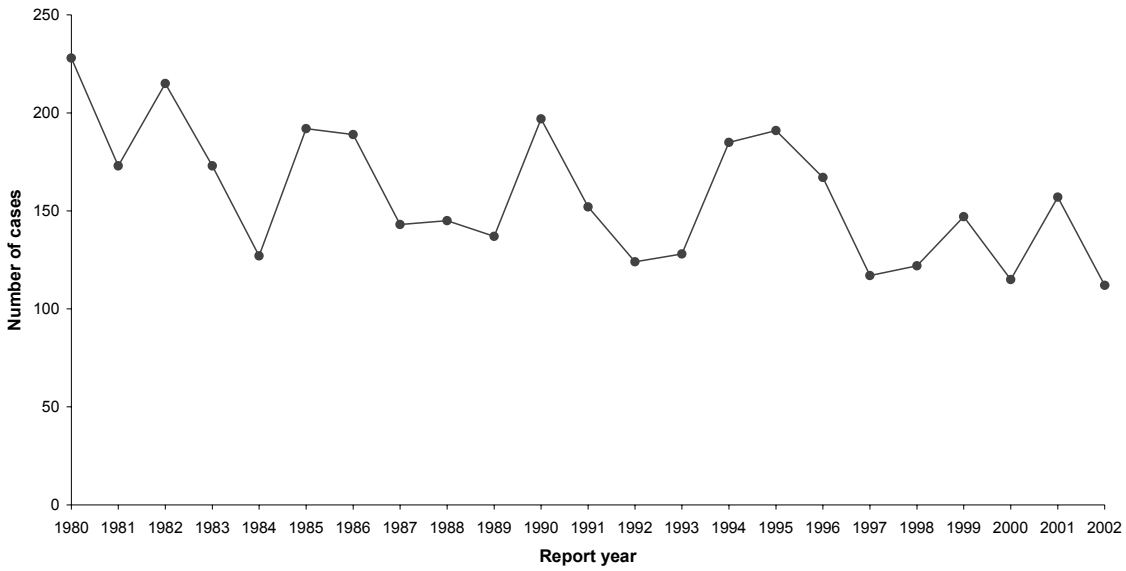
The above data are useful in suggesting sources for salmonella infection. In the case of Salmonella types that appear entirely exotic to New Zealand, such risk factor information can explain most, if not all cases. More often, such information can only suggest hypotheses that require more definitive investigation, using for example a case-control study, as was undertaken to identify sources of STM160 in 2001. [47]

These surveillance data suggest a generally increasing incidence of salmonellosis over the past two decades or more. The national incidence of disease is composed of the sum of many individual epidemics, as illustrated by the emergence of STM160 and S. Brandenburg over the past few years. Control measures will need to reflect this diversity of reservoirs and modes of transmission.

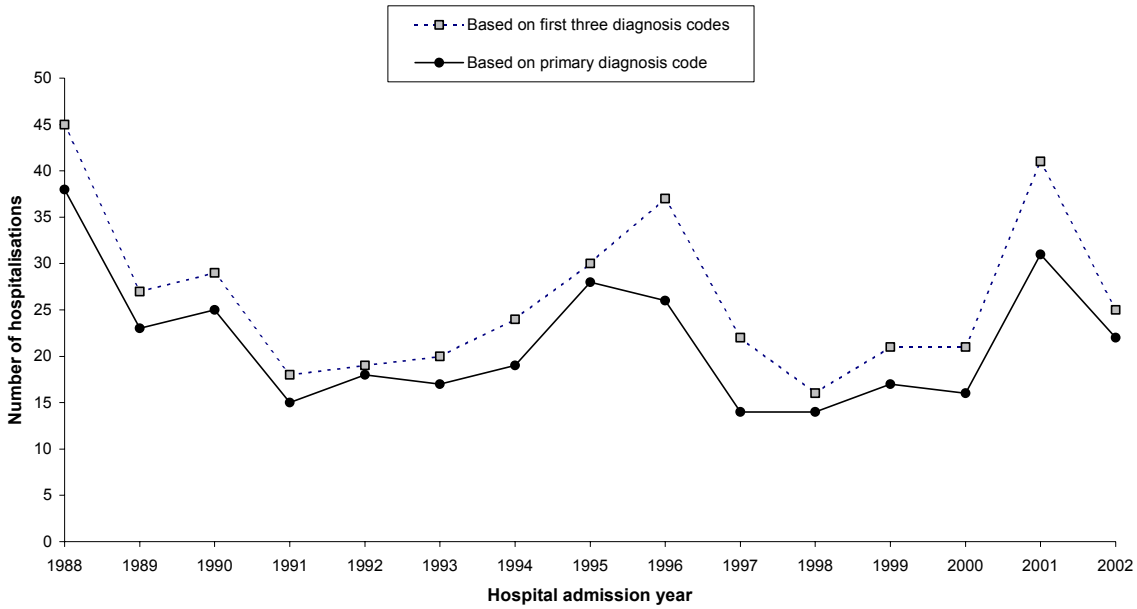
Shigellosis

There were 112 cases of shigellosis notified in 2002, representing a rate of 3.0 per 100 000 (95% CI: 2.4-3.6). This was slightly lower than the notification rate in 2001 of 4.2 (95% CI: 3.5-4.9). In comparison, a total of 123 isolates was received by the ESR Enteric Reference Laboratory in 2002 – significantly fewer than the 190 isolates received in 2001. Of the 89 cases for whom hospitalisation status was recorded on EpiSurv, 24 (27.0%) were hospitalised. According to hospital discharge data, the number of shigellosis hospitalisations (ICD9 code 004) in 2002 totalled 22, compared to 31 the previous year. The following graphs show (i) the number of notified cases each year since 1980, and (ii) annual hospitalisations since 1988.

Shigellosis notifications by year, 1980 - 2002

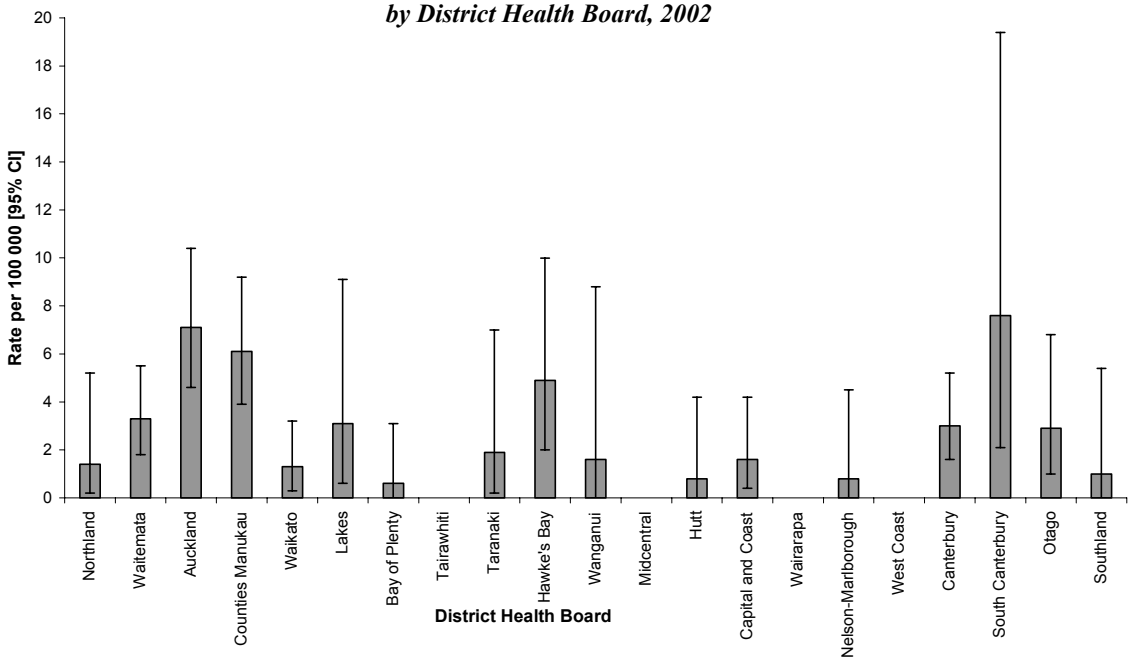


Shigellosis hospitalisations by year, 1988 - 2002



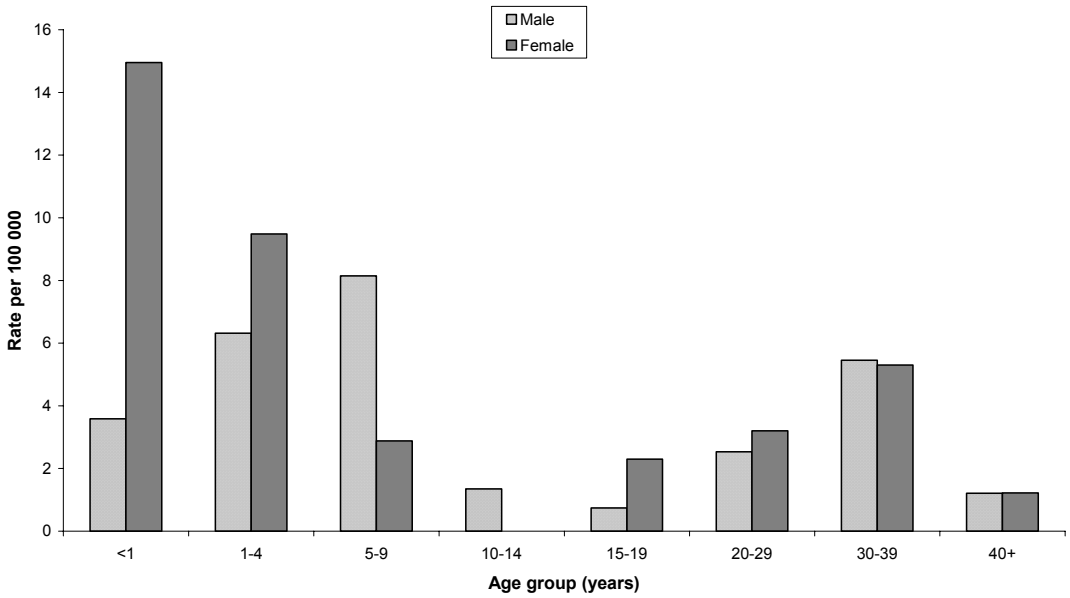
Seven shigellosis outbreaks, involving a total of 27 cases, were reported in 2002. Five outbreaks were reported from the Auckland region, and one each from Canterbury and South Canterbury. The notification rate was highest in South Canterbury DHB (7.6 per 100 000), although this should be interpreted with caution, due to the wide confidence intervals illustrated on the chart below. Rates significantly higher than the national average of 3.0 per 100 000 were seen in Auckland (7.1) and Counties Manukau (6.1) District Health Boards.

Shigellosis disease crude notification rates by District Health Board, 2002



Age-specific rates were highest in children aged less than ten years, with a rate of 6.8 per 100 000. The '30 to 39 years' age group also experienced a rate (5.4, 95% CI: 3.7-7.6) significantly higher than the general population. The following bar chart compares notification rates by age and sex.

Shigellosis disease notification rates, 2002
by age and sex



Ethnicity was recorded for 85 (75.9%) notified cases. Crude and age-standardised rates of disease were highest in Pacific peoples (crude rate: 13.5 per 100 000), and next highest among those of 'Other' ethnicity (6.5). Among the 16 cases of Pacific ethnicity who provided further details of their ethnic group, 14 (87.5%) were Samoan. Rates were low in both the Maori (1.1) and the European (1.4) ethnic groups. The tables below illustrate the age and ethnic distribution of shigellosis notifications.

Shigellosis notifications and age-specific rates by ethnicity, 2002

Ethnicity	**Age group in years**											
	<1	**1-4**	**5-9**	**10-14**	**15-19**	**20-29**	**30-39**	**40-49**	**50-59**	**60-69**	**70+**	**Total**
European	3	2	4	0	2	5	10	4	4	0	2	36
	10.1	1.6	2.4	0.0	1.2	1.6	2.5	1.0	1.2	0.0	0.7	1.4
Maori	0	0	2	0	1	0	2	0	1	0	0	6
	0.0	0.0	3.0	0.0	2.0	0.0	2.6	0.0	3.0	0.0	0.0	1.1
Pacific peoples	1	9	8	1	0	1	4	2	1	0	0	27
	19.4	46.9	33.9	4.6	0.0	3.0	12.6	9.2	7.5	0.0	0.0	13.5
Other	0	3	1	1	0	5	3	2	0	1	0	16
	0.0	21.4	5.4	5.0	0.0	11.5	6.6	5.3	0.0	9.1	0.0	6.5
Unknown	1	3	2	0	1	4	12	1	3	0	0	27
Total	5	17	17	2	4	15	31	9	9	1	2	112
	9.2	7.9	5.9	0.7	1.5	3.1	5.4	1.7	2.2	0.4	0.6	3.0

Number of cases
Rate per 100 000

Shigellosis - crude and age-standardised rates by ethnicity, 2002

Ethnicity	Crude rate [95% CI]	Age-standardised rate* [95% CI]
European	1.4 [1, 1.9]	1.5 [1.1, 2.1]
Maori	1.1 [0.4, 2.5]	1.1 [0.4, 4.4]
Pacific peoples	13.5 [8.9, 19.6]	10.4 [6.7, 18.6]
Other	6.5 [3.7, 10.5]	6.0 [3.4, 12.4]
Total	3.0 [2.4, 3.6]	

* Directly standardised to the NZ population

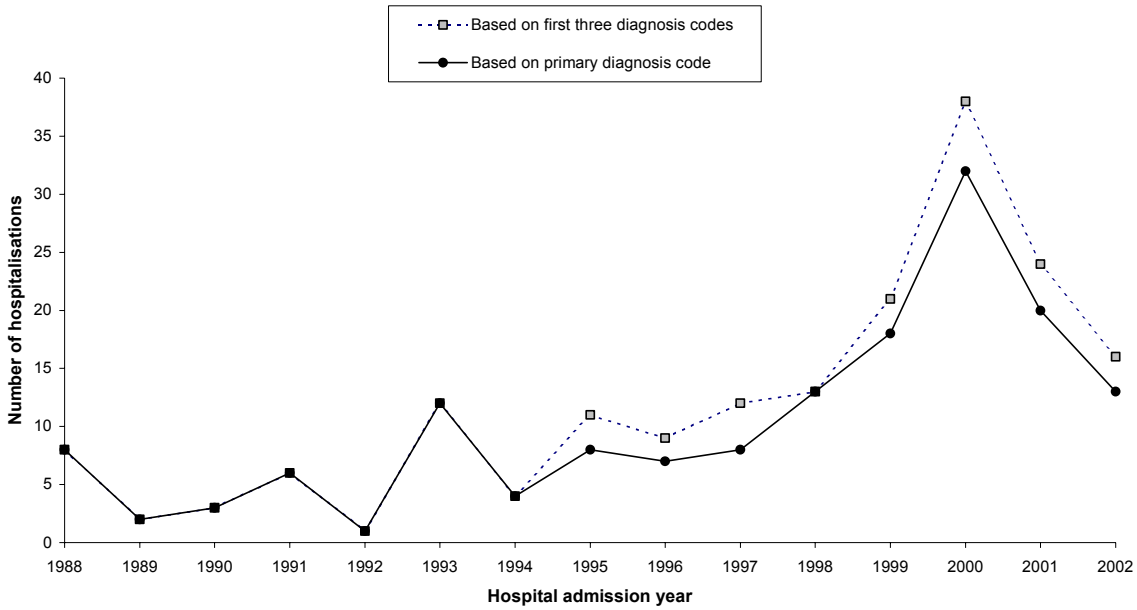
Information on overseas travel was recorded for 83 cases, and of these, 41 (49.4%) cases reported travelling overseas during the incubation period. The most commonly implicated countries were India (7 cases), Indonesia (6) and Thailand (4). In total, 25 cases had been in Asia, ten in Oceania, four in America, and one in Europe. Assuming notified cases included few migrants, visitors or refugees to New Zealand, the highest rates of infection occurred among travellers to Asia (14.7 cases per 100 000 visits), in particular India (80.0 per 100 000 visits).

Overall, these data suggest a gradual decline in the incidence of shigellosis in New Zealand and relatively low levels of transmission in this country. About half of the cases are linked to overseas travel, including travel to the Pacific where the disease remains endemic. [48]

Toxic shellfish poisoning

One suspect case of toxic shellfish poisoning was notified from West Coast DHB in 2002. The case, a 36-year-old European male, had become ill after consuming mussels. The case was not hospitalised. According to hospital discharge data, there were 13 hospitalisations due to fish or shellfish poisonings (ICD9 code 988.0) in 2002, compared to 20 the year before, and a peak of 32 hospitalisations in 2000. The following graph shows the number of hospitalisations each year since 1988.

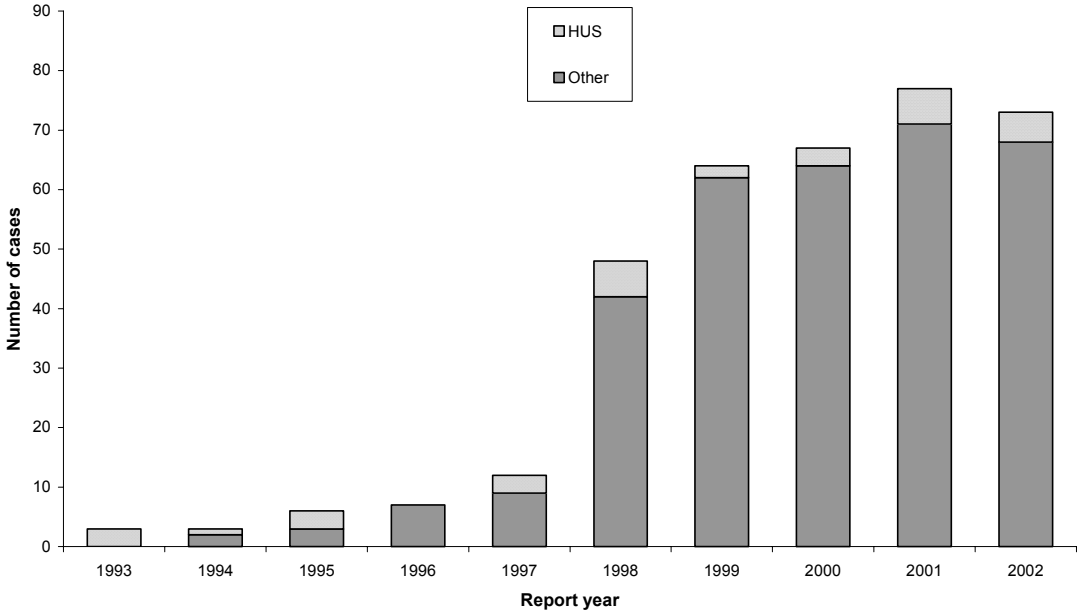
Hospitalisations due to fish and shellfish poisonings by year, 1988 - 2002



VTEC/STEC infection

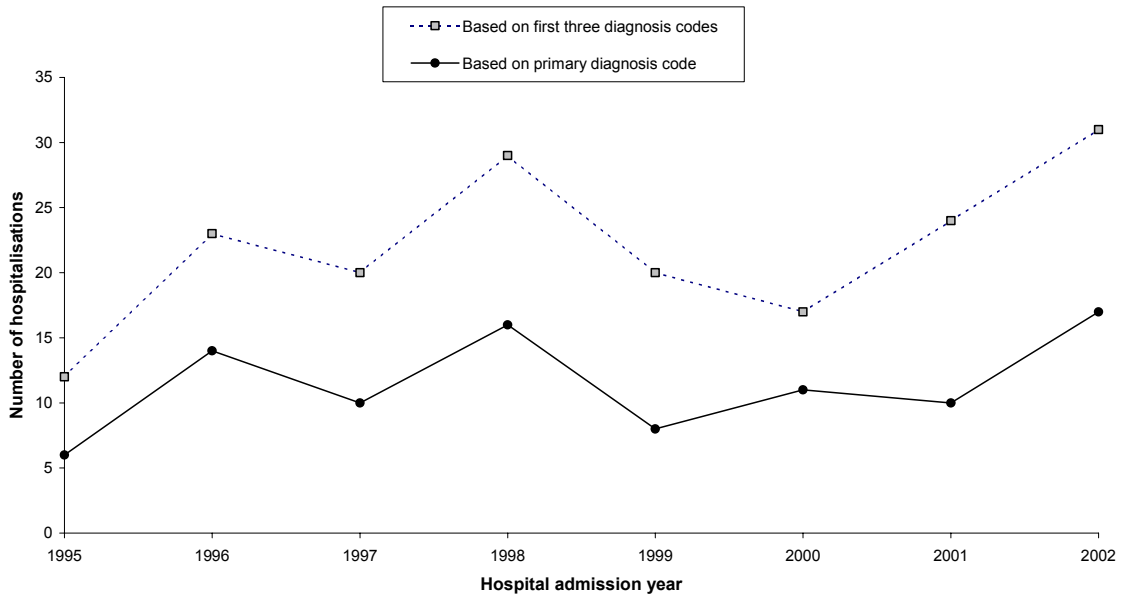
There were 73 cases of Verocytotoxigenic *Escherichia coli* infection (VTEC), also known as Shigatoxigenic *Escherichia coli* infection (STEC), notified in 2002. The 2002 notification rate of 2.0 cases per 100 000 (95% CI: 1.5-2.5) was similar to that in 2001 when 76 cases were notified. In 2002, the ESR Enteric Reference Laboratory received a total of 67 VTEC/STEC isolates: 63 (94%) were identified as serotype O157, and four as other serotypes. In comparison, 75 isolates were received in 2001, of which two were identified as non-O157 serotypes. Matching of notification and laboratory records indicated that all laboratory-reported cases in 2002 were notified. Five cases of VTEC/STEC-associated haemolytic uraemic syndrome (HUS) were reported in 2002 to the New Zealand Paediatric Surveillance Unit (NZPSU). All five cases were notified. The following chart shows the number of notified cases of VTEC/STEC infection each year since 1993.

Verotoxigenic E. coli notified cases by clinical symptoms, 1993 - 2002



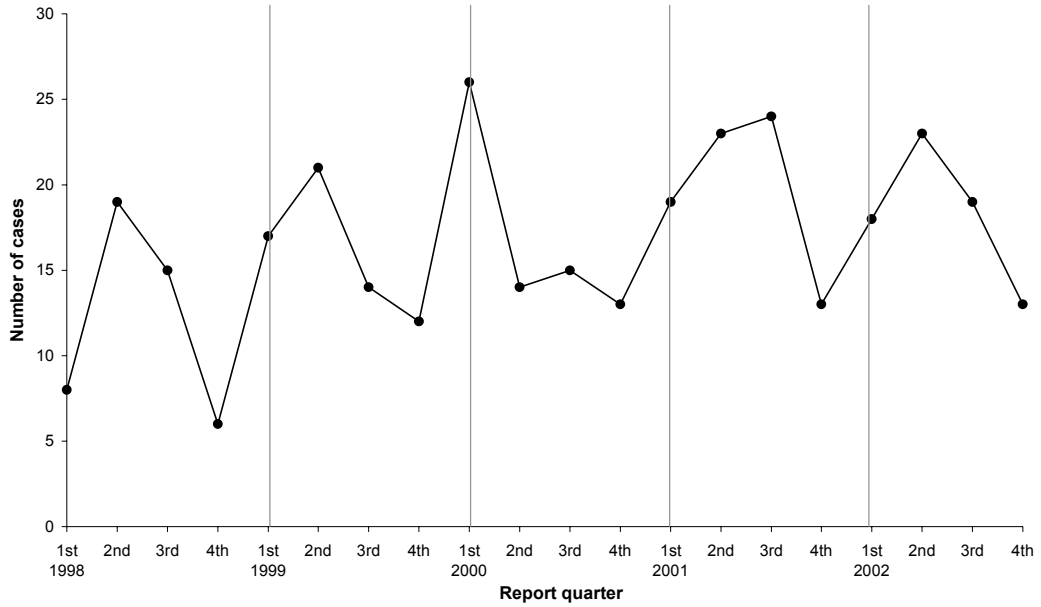
Of the 64 notified cases of VTEC/STEC for whom hospitalisation status was recorded on EpiSurv, 16 (25%) were hospitalised. According to hospital discharge data, there were 17 hospitalisations due to HUS (not necessarily all VTEC/STEC-associated) in 2002, compared to 10 the previous year. The following graph shows HUS hospitalisations (ICD9 code 283.11) each year since 1995.

Haemolytic uraemic syndrome hospitalisations by year, 1988 - 2002



VTEC/STEC infections have tended to be seasonal, as the following graph demonstrates. In most years there has been an Autumn peak and a Spring trough in incidence.

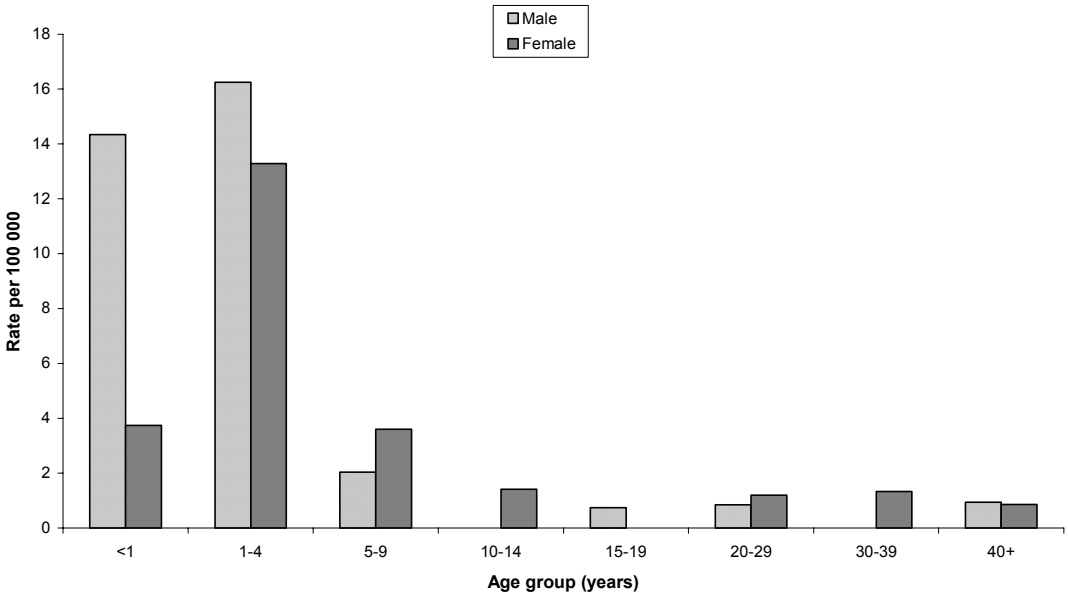
VTEC/STEC notifications by quarter, 1998 - 2002



Rates of VTEC/STEC infection varied throughout the country in 2002, with the highest rates reported by Taranaki (4.9, 95% CI: 1.6-11.3), Waikato (4.7, 95% CI: 2.6-7.8), Canterbury (4.2, 95% CI: 2.5-6.7) and Lakes (4.2, 95% CI: 1.1-10.7) District Health Boards. One Canterbury outbreak involving three cases was reported. The proportion of cases in 2002 which geocoded to rural areas was 26.8% (95% CI: 17.9-38.1%). In comparison, just 12.6% of the New Zealand population is classified as rural. Among cases for whom this information was recorded, 87.3% reported contact with animals (59% with farm animals) the week before becoming ill; 76.3% reported contact with animal manure; 29.1% reported contact with children in nappies; 26.5% reported recreational contact with water; and 13.2% reported contact with a person with similar symptoms.

Rates significantly higher than the overall rate of 2.0 per 100 000 occurred among children aged four years or less, with an age-specific rate of 15.7 (95% CI: 10.9-22.0) in the '1 to 4 years' age group, and a rate of 9.2 (95% CI: 3.0-21.4) in the 'less than one year' age group. The following bar chart compares notification rates by age and sex.

VTEC/STEC notification rates, 2002
by age and sex



Notification rates were highest in the 'Other' and the European ethnic groups, with rates of 3.2 and 2.0 per 100 000, respectively. There were no cases in Pacific peoples. The tables below illustrate the age and ethnic distribution of cases.

VTEC/STEC notifications and age-specific rates by ethnicity, 2002

Ethnicity	Age group in years											
	<1	**1-4**	**5-9**	**10-14**	**15-19**	**20-29**	**30-39**	**40-49**	**50-59**	**60-69**	**70+**	**Total**
European	4	23	6	2	0	3	3	0	4	3	4	52
	13.4	18.9	3.6	1.1	0.0	1.0	0.8	0.0	1.2	1.3	1.4	2.0
Maori	1	2	0	0	0	0	0	0	0	0	0	3
	7.1	3.7	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.6
Pacific peoples	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
Other	0	2	1	0	1	2	1	1	0	0	0	8
	0.0	14.3	5.4	0.0	3.6	4.6	2.2	2.6	0.0	0.0	0.0	3.2
Unknown	0	7	1	0	0	0	0	0	0	2	0	10
Total	5	34	8	2	1	5	4	1	4	5	4	73
	9.2	15.7	2.8	0.7	0.4	1.0	0.7	0.2	1.0	1.8	1.2	2.0

Number of cases
Rate per 100 000

VTEC/STEC - crude and age-standardised rates by ethnicity, 2002

Ethnicity	Crude rate [95% CI]	Age-standardised rate* [95% CI]
European	2.0 [1.5, 2.6]	2.2 [1.6, 2.9]
Maori	0.6 [0.1, 1.7]	0.3 [0.1, 3.5]
Pacific peoples	0.0 [0, 1.5]	-
Other	3.2 [1.4, 6.4]	2.8 [1.2, 8.6]
Total	2.0 [1.5, 2.5]	

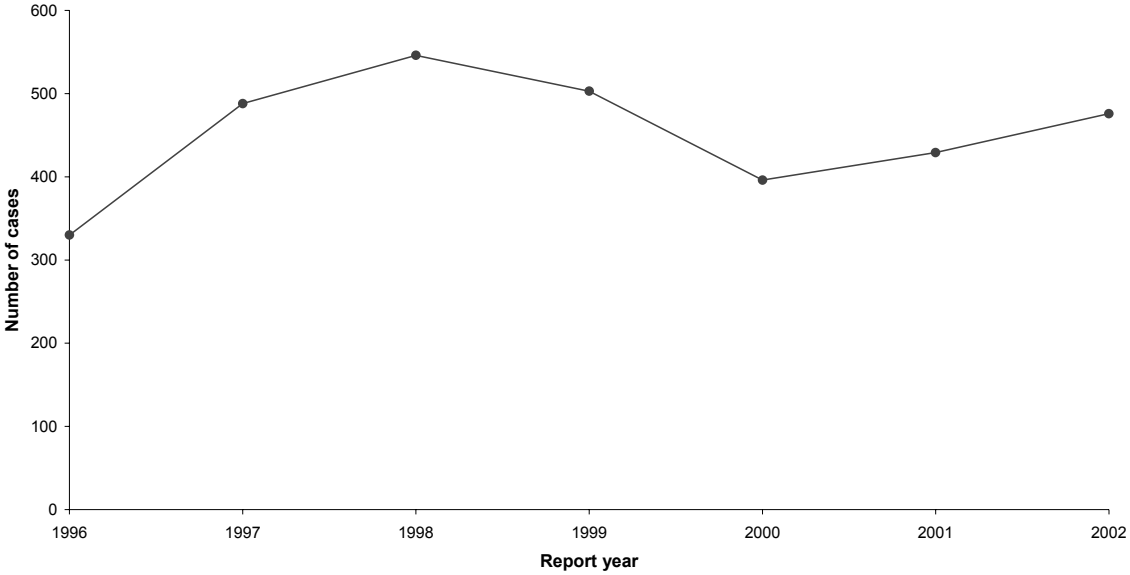
* Directly standardised to the NZ population

These data suggest that the rate of infection with VTEC in New Zealand has reached a plateau of about 70 cases a year. The emergence of this enteric disease is of concern because of its relatively severe spectrum of associated disease, including HUS, as well as its outbreak potential.[49] These surveillance data continue to suggest that infection is occurring as a result of contact with farm animals and contaminated environments, with no common source food or waterborne outbreaks. Growing international experience with outbreaks of this infection reinforce the need for prevention measures.[50]

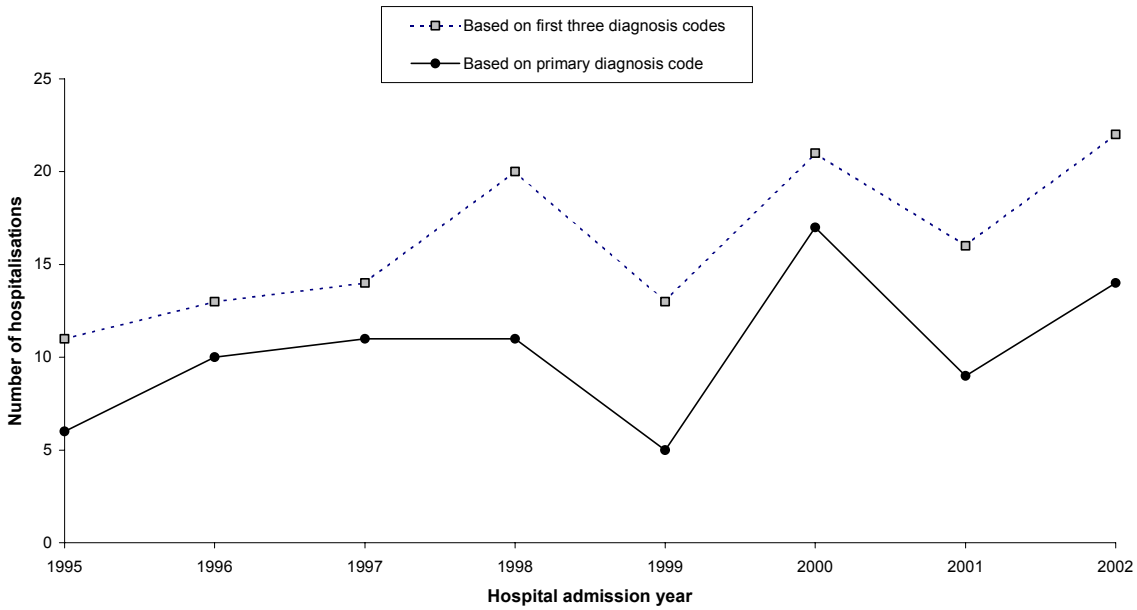
Yersiniosis

There were 476 cases of yersiniosis notified in 2002, representing a rate of 12.7 per 100 000 (95% CI: 11.6-13.0), slightly higher than the rate of 11.5 (95% CI: 10.4-12.6) in 2001. Of the 328 cases for whom hospitalisation status was recorded on EpiSurv, 31 (9.5%) were hospitalised. According to hospital discharge data, the number of hospitalisations (ICD9 code 008.44) in 2002 totalled 14. The following graphs show (i) the number of cases of yersiniosis notified each year since 1996, and (ii) the hospitalisations each year since 1995.

Yersiniosis notifications by year, 1996 - 2002

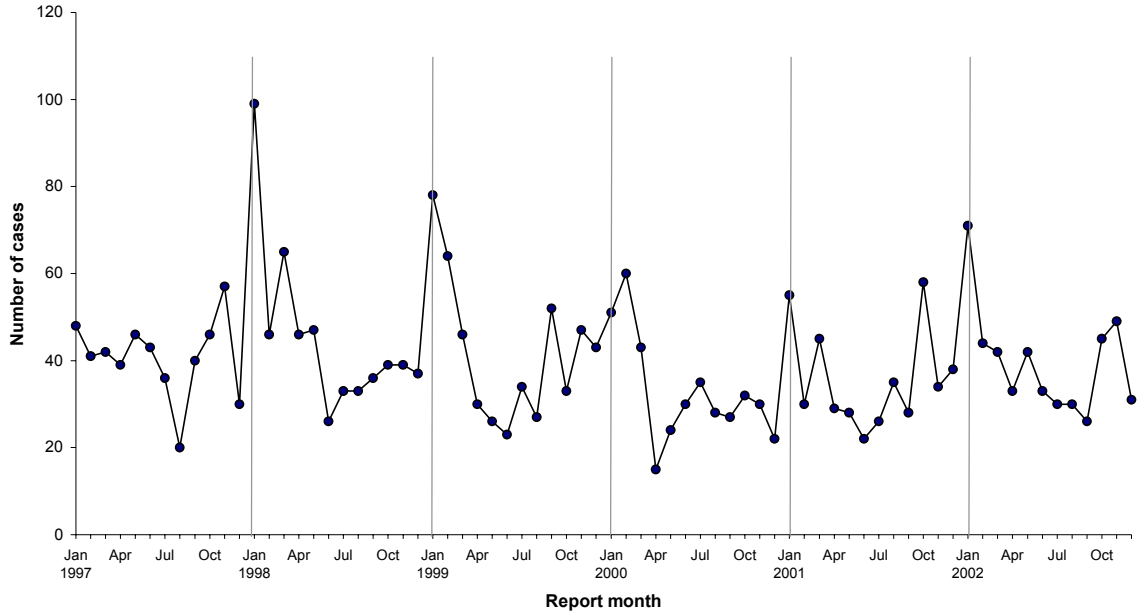


Yersiniosis hospitalisations by year, 1995 - 2002



The following graph shows the notified cases each month since January 1997. The incidence shows some seasonality, usually with a summer peak and a winter trough, but this pattern is less pronounced than for other enteric diseases.

Yersiniosis notifications by month, 1997 - 2002

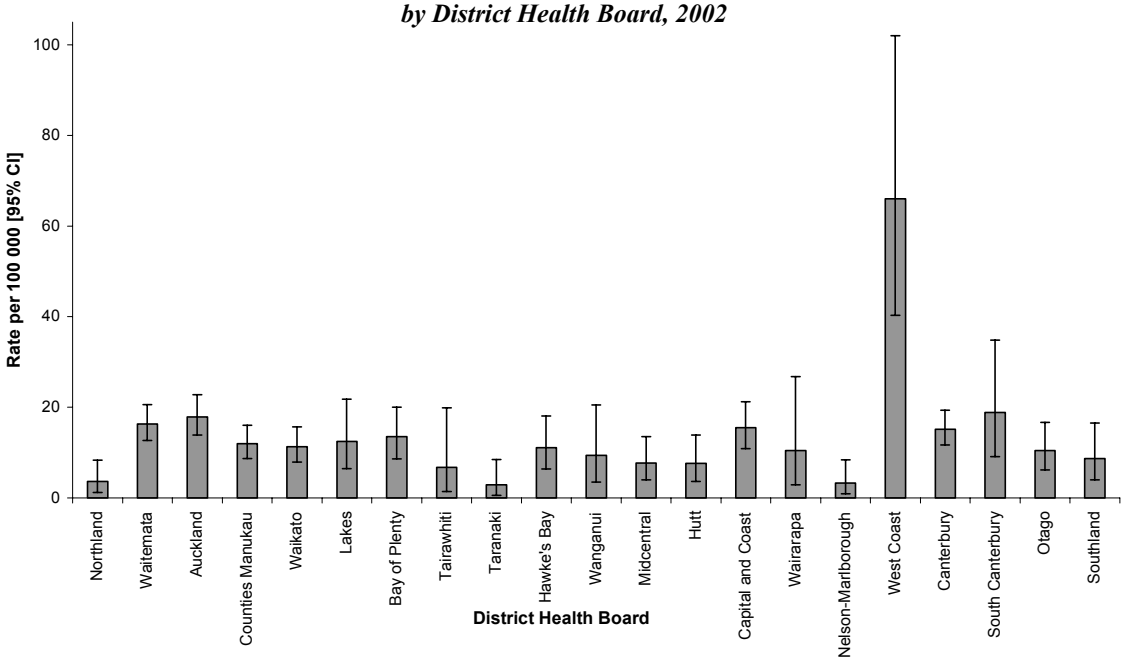


Information on contact with farm animals was recorded for 200 cases, of whom 59 (29.5%) reported contact. Analysis of free text fields identified a total of 91 cases who had contact with live farm animals, wild animals or pets. In 2002, occupations[a] were recorded for 119 notified cases aged 15 or over. Of these, 7 (5.9%) worked in the meat processing industry. In comparison, just 1.2% of the employed population aged 15 or over were meat processing workers at the last census.

Three outbreaks were reported in 2002. One Manawatu outbreak occurred in a daycare centre, one outbreak in a West Coast home was linked to duck breeding, and one Auckland outbreak was attributed to contaminated ham. The incidence rate of 66.0 per 100 000 (95% CI: 40.3-102.0) in West Coast District Health Board was significantly higher than in all other District Health Boards, as the following chart demonstrates. The next highest rates were seen in South Canterbury (18.9) and Auckland (17.9) DHBs.

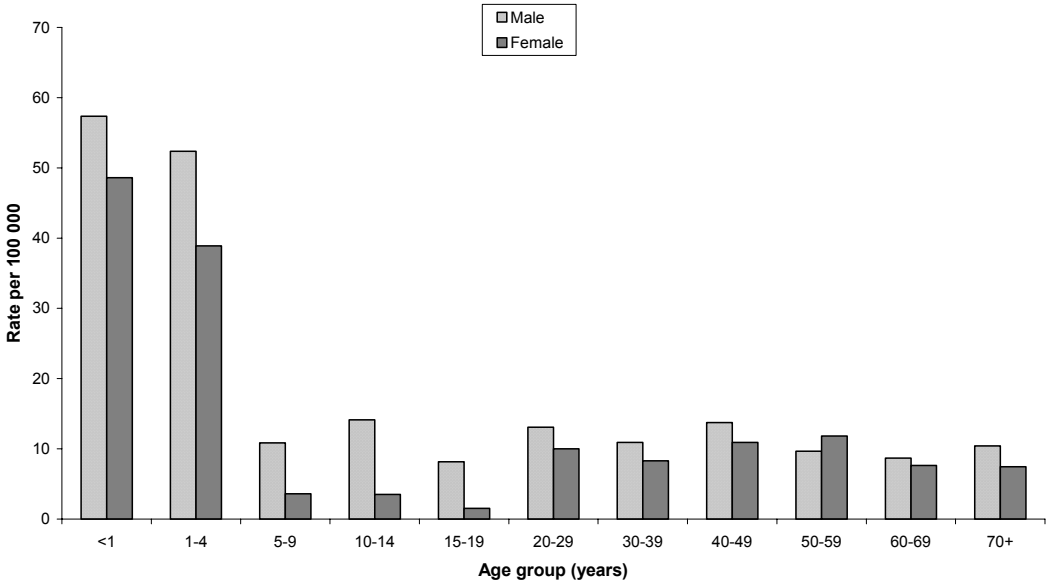
[a] Excludes homemakers, sickness beneficiaries, retired people, and students

Yersiniosis crude notification rates by District Health Board, 2002



Age-specific rates were significantly higher in the 'less than one year' (54.9 per 100 000) and the '1 to 4 years' age groups (48.1) than in all other age groups. In almost all age groups the rate among males exceeded the rate among females. The following chart compares notification rates by age and sex.

Yersiniosis notification rates, 2002 by age and sex



Ethnicity was recorded for 334 (70.2%) notified cases. Rates were significantly higher in the 'Other' and European ethnic groups (with rates of 15.8 and 10.1 respectively) than in all other ethnic groups. The tables below illustrate the age and ethnic distribution of cases.

Yersiniosis notifications and age-specific rates by ethnicity, 2002

Ethnicity	Age group in years											
	<1	**1-4**	**5-9**	**10-14**	**15-19**	**20-29**	**30-39**	**40-49**	**50-59**	**60-69**	**70+**	**Total**
European	11	40	15	17	7	30	31	42	32	17	21	263
	37.0	32.9	8.9	9.6	4.4	9.8	7.8	10.6	9.6	7.3	7.3	10.1
Maori	1	11	0	1	0	4	4	3	1	1	0	26
	7.1	20.5	0.0	1.6	0.0	4.9	5.2	5.2	3.0	5.1	0.0	4.9
Pacific peoples	0	1	0	1	0	0	2	1	0	1	0	6
	0.0	5.2	0.0	4.6	0.0	0.0	6.3	4.6	0.0	13.4	0.0	3.0
Other	6	14	1	2	1	5	4	3	1	0	0	39
	161.4	99.8	5.4	9.9	3.6	11.5	8.7	7.9	5.1	0.0	0.0	15.8
Unknown	12	38	5	6	6	18	14	18	13	4	7	142
Total	30	104	21	27	14	57	55	67	47	23	28	476
	54.9	48.1	7.3	9.3	5.3	11.7	9.5	12.5	11.2	8.1	8.7	12.7

Number of cases
Rate per 100 000

Yersiniosis - crude and age-standardised rates by ethnicity, 2002

Ethnicity	Crude rate [95% CI]	Age-standardised rate* [95% CI]
European	10.1 [8.8, 11.3]	10.4 [9.2, 11.8]
Maori	4.9 [3.2, 7.2]	4.3 [2.7, 8]
Pacific peoples	3.0 [1.1, 6.5]	3.3 [1.1, 11]
Other	15.8 [11.2, 21.5]	14.1 [9.9, 21.6]
Total	12.7 [11.6, 13.9]	

* Directly standardised to the NZ population

The incidence of yersiniosis remains fairly constant, though high by world standards. Rates are particularly high in children under five years of age. The sources of infection are still poorly understood in New Zealand, though these surveillance data suggest direct contact with farm animals or their carcasses is likely to be a source for at least some cases. The elevated rate among people of 'Other' ethnicity could have a relationship to travel or dietary patterns. The only investigation of sporadic yersiniosis carried out in New Zealand identified contact with untreated water, unreticulated sewerage and consumption of pork as risk factors. [51]

Diseases from the environment and animal contact

This section includes zoonoses, diseases from contaminated environments and infectious diseases acquired occupationally. This category overlaps with many of the enteric diseases where farm animals, domestic animals, and contaminated environments are often important sources of human infection. This appears to be particularly the case for cryptosporidiosis, VTEC infection, and some Salmonella serogroups (notably Brandenburg).

Brucellosis

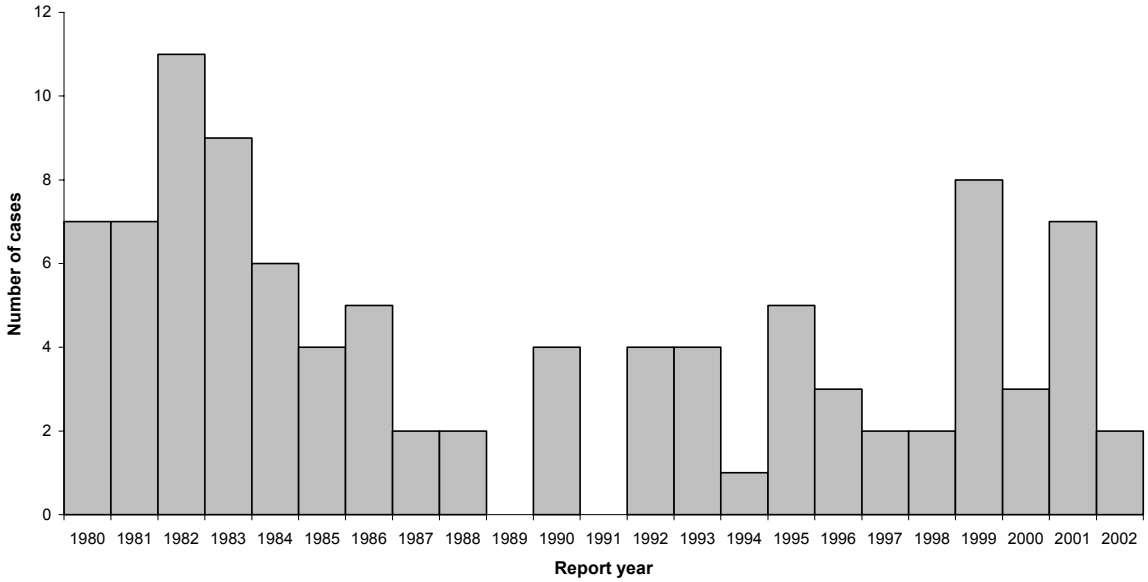
Two cases of brucellosis were notified in 2002. One case, a five-year-old male from Wellington, was believed to have acquired the infection in India, where he had consumed unpasteurised milk during the incubation period. The second case was a 43-year-old male of Pacific ethnicity from South Auckland. Epidemiological evidence suggested that the infection was locally acquired. The case had slaughtered two pigs a fortnight before the onset of illness on 1 January 2002, and was hospitalised a month later. Culture and serology results identified the infecting organism as *Brucella suis*. The Ministry of Agriculture and Forestry screened pigs on 42 farms but no infected animals were found. [52]

New Zealand has for some time claimed *Brucella*-free status in its cattle and pigs. Only *Brucella abortus*, which is maintained in cattle, has ever been endemic in New Zealand, and no new cases have been diagnosed in cattle since 1989. In humans, there has been no confirmed transmission of Brucella abortus in New Zealand in the past decade.

Hydatid disease

Two cases of hydatid disease were notified in 2002, compared to seven cases the previous year. In 2002, one confirmed case was a 76-year-old male, and one probable case was a 47-year-old male who had confirmed hydatids as a child. Both cases were from the Auckland region. The latter case was hospitalised. The following chart shows the number of hydatids cases notified each year since 1980.

Hydatid disease notifications by year, 1980 - 2002

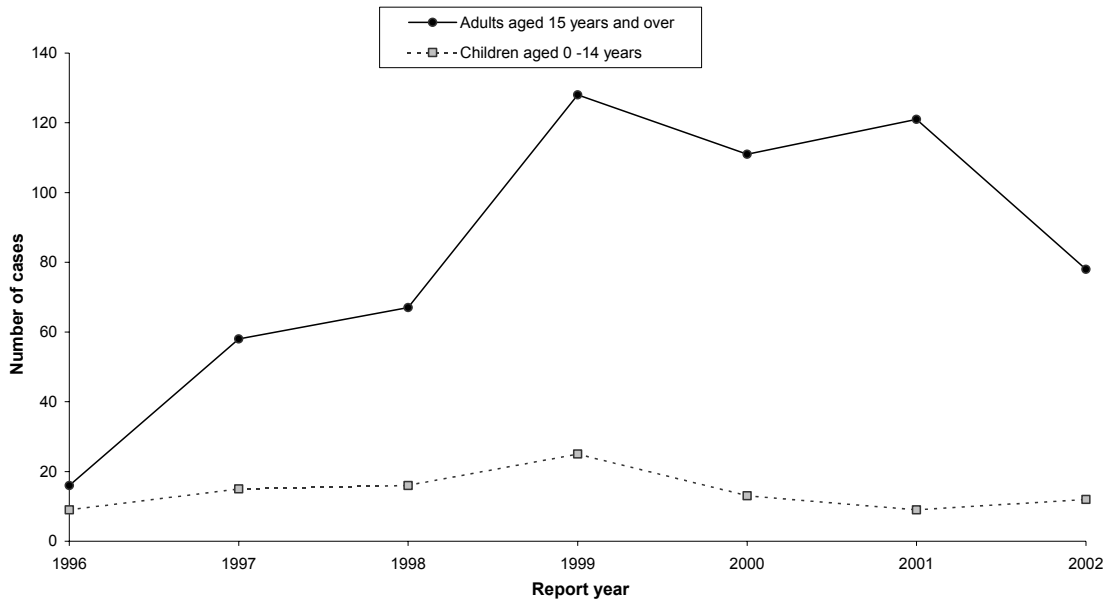


Lead absorption and chemical poisoning

No cases of chemical poisoning were notified in 2002. In comparison, four cases of chemical poisoning, including one confirmed case of arsenic poisoning was notified in 2001.

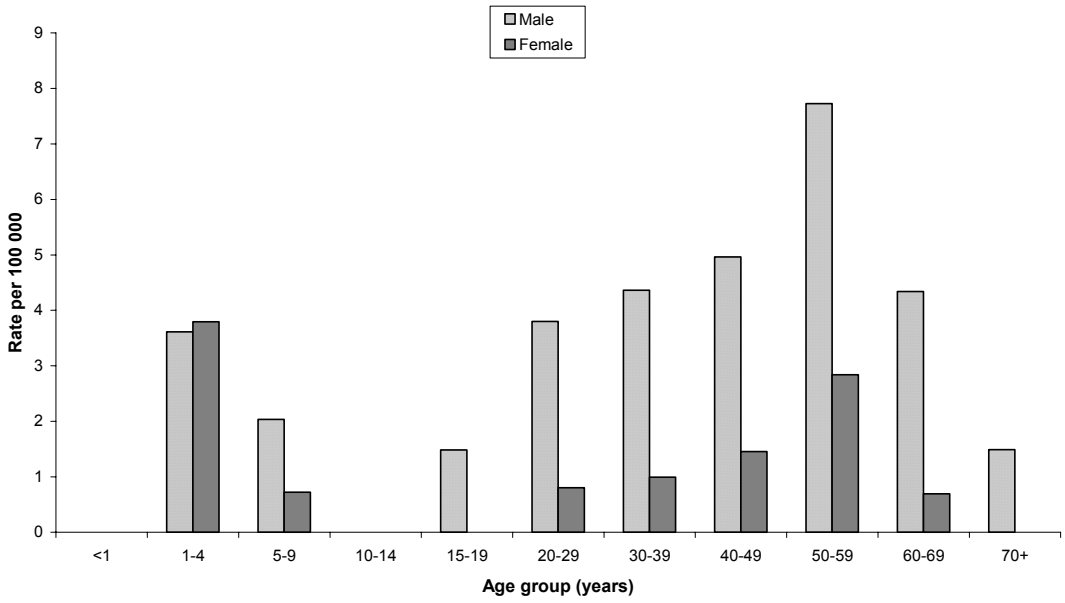
Lead absorption became a notifiable disease in June 1996 at the level of 0.72 μmol/l. A total of 90 cases of lead absorption was notified in 2002, representing a rate of 2.4 per 100 000. Of these 90 cases, 12 (13.3%) were children aged 14 years or younger. In comparison, 130 cases were notified during 2001, nine (6.9%) of whom were children. Of the 66 cases in 2002 for whom hospitalisation status was recorded, 3 (4.6 %) were hospitalised. The following graph illustrates the number of lead absorption notifications in both children and adults, each year since 1996.

Notified cases of lead absorption in children and adults 1996 - 2002



Rates of lead absorption during 2002 were fairly uniform around the country. Tairawhiti DHB recorded the highest rate of 9.1 per 100 000 (95% CI: 2.5-23.3). Rates were higher among males than females in every age group except the '1 to 4 years' age group. Males aged between 50 and 59 years experienced the highest rate of 5.5 per 100 000 (95% CI: 3.5-8.3), significantly higher than the general population. The following chart compares the notification rates by age and sex.

Lead absorption notification rates, 2002 by age and sex



The tables below illustrate the age and ethnic distribution of cases. Notification rates were significantly higher among people of European ethnicity than in other ethnic groups. Indeed, there were no notified cases among Pacific peoples or those of 'Other' ethnicity.

Lead absorption notifications and age-specific rates by ethnicity, 2002

Ethnicity	Age group in years											
	<1	**1-4**	**5-9**	**10-14**	**15-19**	**20-29**	**30-39**	**40-49**	**50-59**	**60-69**	**70+**	**Total**
European	0	5	4	0	2	8	12	12	19	5	2	69
	0.0	4.1	2.4	0.0	1.2	2.6	3.0	3.0	5.7	2.2	0.7	2.6
Maori	0	1	0	0	0	1	0	1	1	0	0	4
	0.0	1.9	0.0	0.0	0.0	1.2	0.0	1.7	3.0	0.0	0.0	0.8
Pacific peoples	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
Other	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
Unknown	0	2	0	0	0	2	3	4	3	2	0	17
Total	0	8	4	0	2	11	15	17	23	7	2	90
	0.0	3.7	1.4	0.0	0.8	2.3	2.6	3.2	5.5	2.5	0.6	2.4

Number of cases
Rate per 100 000

Lead absorption - crude and age-standardised rates by ethnicity, 2002

Ethnicity	Crude rate [95% CI]	Age-standardised rate* [95% CI]
European	2.6 [2.1, 3.3]	2.6 [2, 3.3]
Maori	0.8 [0.2, 1.9]	0.9 [0.2, 4.2]
Pacific peoples	0.0 [0, 1.5]	-
Other	0.0 [0, 1.2]	-
Total	2.4 [1.9, 3]	

* Directly standardised to the NZ population

Of the 78 adult notifications in 2002, a total of 28 (35.9%) cases was exposed to lead through their work: 17 builders, painters or sanders; two radiator repairers; one engineer; one electrician; one leadlighter; one welder; one panelbeater; one foundry worker; one fitter and turner; one lead sinker maker; and one trophy maker. Two of these 28 cases were also recreational shooters. Of the remaining 50 cases, for whom no occupational exposure to lead was indicated, 13 (16.7% of all adult notifications) were exposed to lead paint flakes or dust in their home[a], 10 (12.8%) were recreational shooters, one case was a leadlighter hobbyist, and one made link sinkers as a hobby. No risk factor information was recorded for the remaining 25 (33.3%) adult cases.

[a] Several occupationally exposed cases were also recorded as living in pre-70's buildings in which the paint was chalking, flaking or had been recently stripped.

Of the twelve notifications in children, six lived in pre-70s built homes in which old paint was flaking or had recently been stripped. Three cases were also potentially exposed to lead through parents' occupations, and one case had swallowed a lead product. Blood lead concentrations were recorded for nine of the twelve children, and ranged from 0.72 to 2.04 μmol/l with a median of 0.9 μmol/l.

Passive surveillance of lead absorption is highly influenced by the amount of testing carried out, and almost certainly under-estimates the prevalence of lead absorption in the population. These surveillance findings suggest two population groups continue to be vulnerable to lead absorption: young children living in older houses, and adult males with occupational and recreational contact with lead.

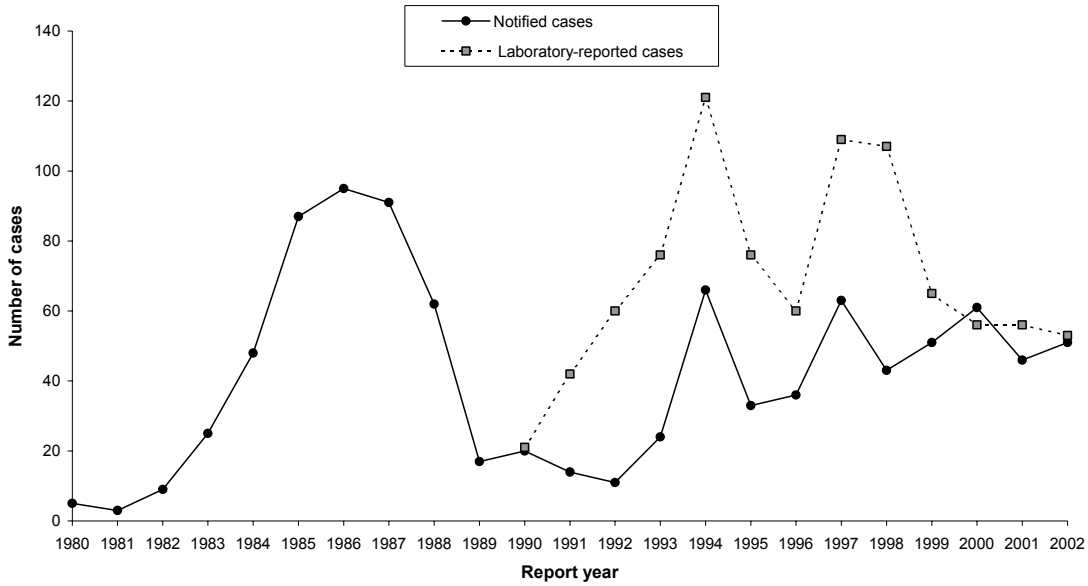
Legionellosis

A total of 51 cases of legionellosis was notified in 2002, representing a rate of 1.4 per 100 000. In comparison, there were 53 laboratory-reported cases during 2002. Of the laboratory-reported cases, 44 (83%) were confirmed and 9 (17%) were regarded as probable cases. Based on the laboratory testing of the 51 notifications, 40 (78.4%) were confirmed, five were deemed probable cases, and four were previously exposed. Hospitalisation status was recorded on EpiSurv for 43 notified cases. Of these, 27 (62.8%) cases were hospitalised. According to hospital discharge data, hospitalisations due to legionellosis (ICD9 code 482.83) in 2002 totalled 84. There were four deaths[a] caused by legionellosis in 2002. The death of one case infected with *L. longbeachae* followed exposure to commercially prepared composted material.

The following graph shows laboratory-reported cases of legionellosis by year since 1990 and notified cases since 1980. The notification rate increased slightly in 2002, although the number of laboratory-reported cases fell slightly.

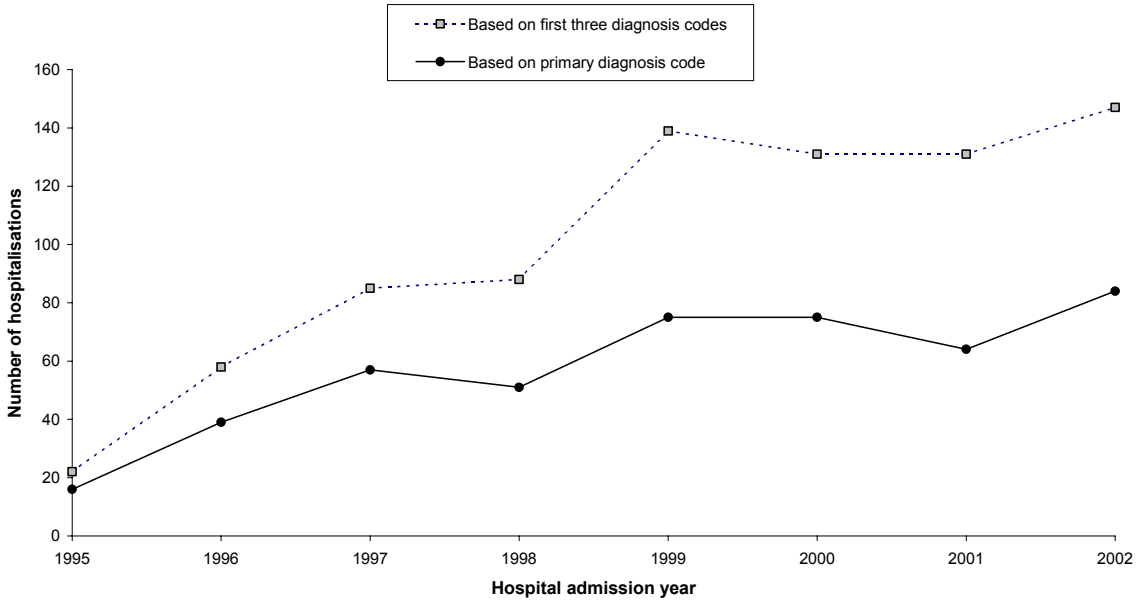
[a] One death occurred in a 'probable case' not yet notified.

Legionellosis notifications and laboratory-reported cases by year, 1980 - 2002



The graph below shows hospitalisations since 1994, as recorded by hospital discharge data. Hospitalisations rose 31% from 64 cases in 2001 to 84 in 2002.

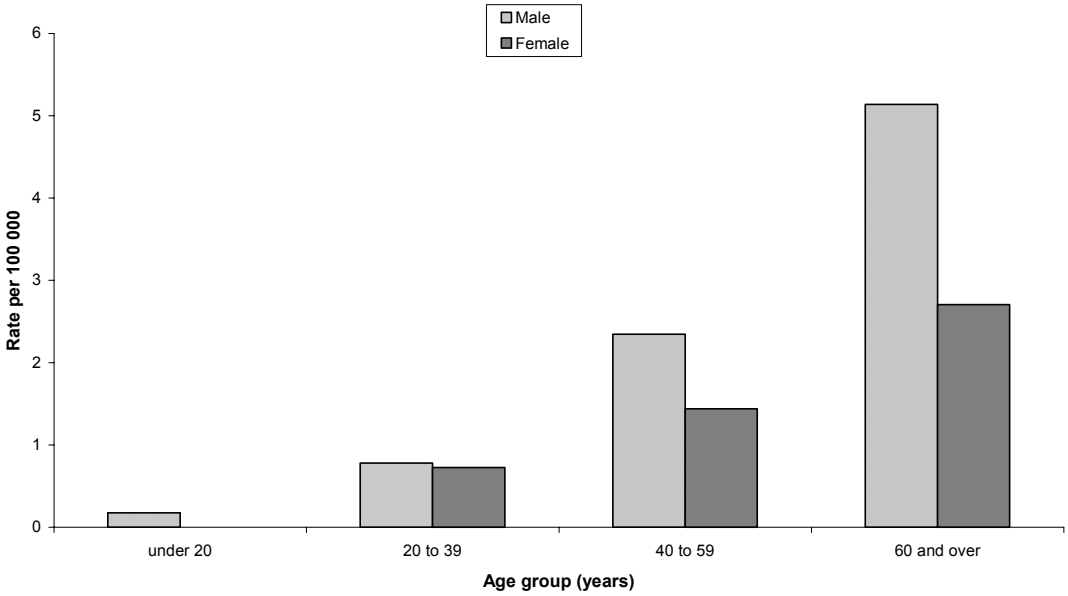
Legionellosis hospitalisations by year, 1995 - 2002



Notification rates varied throughout the country. The highest age-standardised rates were recorded in Wairarapa DHB, with a rate of 4.6 (95% CI: 0.6-20.3) and Hutt DHB, with a rate of 3.0 (95% CI: 0.8-7.9). Of the 40 cases for whom ethnicity was recorded, the majority (39/40 or 97.5%) were of European ethnicity. One case was of Maori ethnicity. Rates of disease were highest among males aged 60 years or over. The age-specific rate of 4.3 per

100 000 (95% CI: 2.4-7.3) in persons aged 70 or over, was significantly higher than the overall rate of 1.4 (95% CI: 1.0-1.8). The following bar chart compares notification rates by age and sex.

Legionellosis notification rates, 2002 by age and sex



The table below provides a summary of risk factor information for notified cases of legionellosis in 2002. Note that some cases had more than one risk factor recorded.

Risk factors associated with legionellosis, 2002

Risk Factor	Yes	No	Unknown	Proportion[1]
Contact with definite or suspected environmental source of infection	24[2]	6	21	80%
Overseas travel during incubation period	0	38	13	0%
Smokers or ex-smokers	9	30	12	23%
Pre-existing immunosuppressive or debilitating condition	14	26	11	35%

[1] "Proportion" refers to the percentage of cases who answered "yes" out of the total number of cases for whom this information was recorded
[2] Of these 24 cases, 20 reported exposure to potting mix, compost or mulch during the incubation period

The following table compares the species/serogroup distribution of laboratory-reported *Legionella* over the last eight years. Of note is the proportion of *L. longbeachae* cases, which decreased from 55% of all laboratory-reported cases in 2001, to 23% in 2002. The greatest proportion (34%) of laboratory-reported cases in 2002 was *L. pneumophila*, more than double the proportion during 2001 (16%).

Species/serogroup distribution of laboratory-reported Legionella, 1995-2002

Legionella species/ Serogroup	Year							
	1995	**1996**	**1997**	**1998**	**1999**	**2000**	**2001**	**2002**
L. pneumophila	12	12	45	67	13	15	9	18
L. anisa	1	0	2	1	0	1	0	0
L. bozemanii	1	0	1	2	2	4	0	4
L. dumoffii	1	0	1	2	2	0	6	3
L. longbeachae	6	0	10	13	34	28	31	12
L. micdadei	6	2	13	3	6	2	2	1
L. jordanis	3	3	3	2	2	0	0	1
L. feelei	1	3	1	0	0	0	0	1
L. gormanii	0	4	0	0	2	2	3	4
*L. hackelei**	-	-	-	-	-	-	-	3
Non- L. *pneumophila* strains	0	0	0	0	0	0	2	0
Unidentifiable *Legionella* sp.	28	21	33	15	4	4	2	6
Total	**59**	**45**	**109**	**105**	**65**	**56**	**56**	**53**

* Testing for this species commenced 2002.

During 2002, there were no clear seasonal trends in laboratory-reported cases[a]. This is in contrast to previous years, when confirmed cases peaked in spring and early summer (September-December) and very few cases were reported in winter.

Three sources of surveillance data: notification, laboratory and hospitalisation data all suggest a relatively stable incidence of legionellosis in New Zealand. The main change has been a decline in *L. longbeachae* infection. This may indicate a decrease in exposure to environmental sources of this agent. There have not been any well-documented common-source outbreaks of legionellosis in New Zealand since the first outbreak in 1990. [53] However, this organism clearly has the potential to cause large common-source outbreaks as demonstrated by the 2000 outbreak linked to the newly opened Melbourne Aquarium.[54]

Leptospirosis

A total of 142 cases of leptospirosis was notified in 2002. In comparison, 181 cases were laboratory-reported in 2002[b]. This latter figure is known not to include all cases confirmed by Auckland A+ Laboratories. Matching of laboratory-reported and notified cases indicated that 125 cases were both notified and laboratory-reported during 2002, suggesting that 17 cases were notified but not lab-reported, and conversely, at least 56 cases were laboratory confirmed but not notified. Of the 103 notified cases in 2002 for whom hospitalisation status was recorded, 55 (53.4%) were hospitalised. According to hospital discharge data, hospitalisations (ICD9 code 100) in 2002 totalled 63. Two outbreaks, involving a total of five cases, were reported in 2002 - one from the Manawatu and one from the Nelson region.

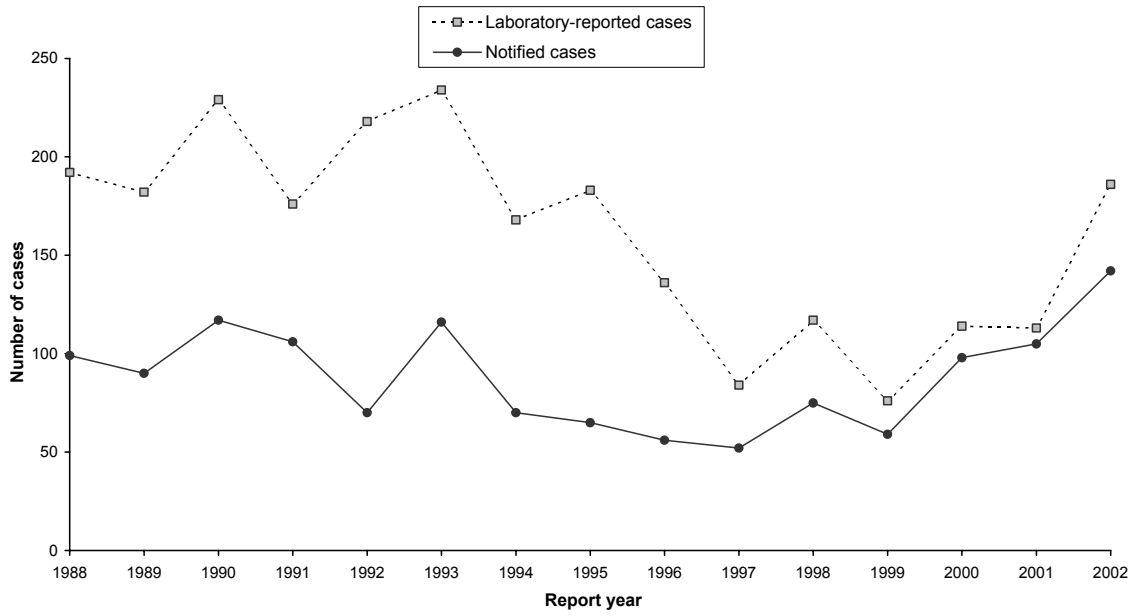
The 2002 notification rate of 3.8 per 100 000 (95% CI=3.2-4.4) was slightly higher than the 2001 rate of 2.8 (95% CI=2.3,3.3). Although 95% confidence intervals for notification rates in 2002 and 2001 overlap, data from laboratory and hospital sources suggest the incidence of

[a] 32%, 23%, 27% and 18% of confirmed laboratory-reported cases occurred in summer, autumn, winter and spring, respectively.
[b] Laboratory data is based on the date the specimen was received at ESR or other laboratory.

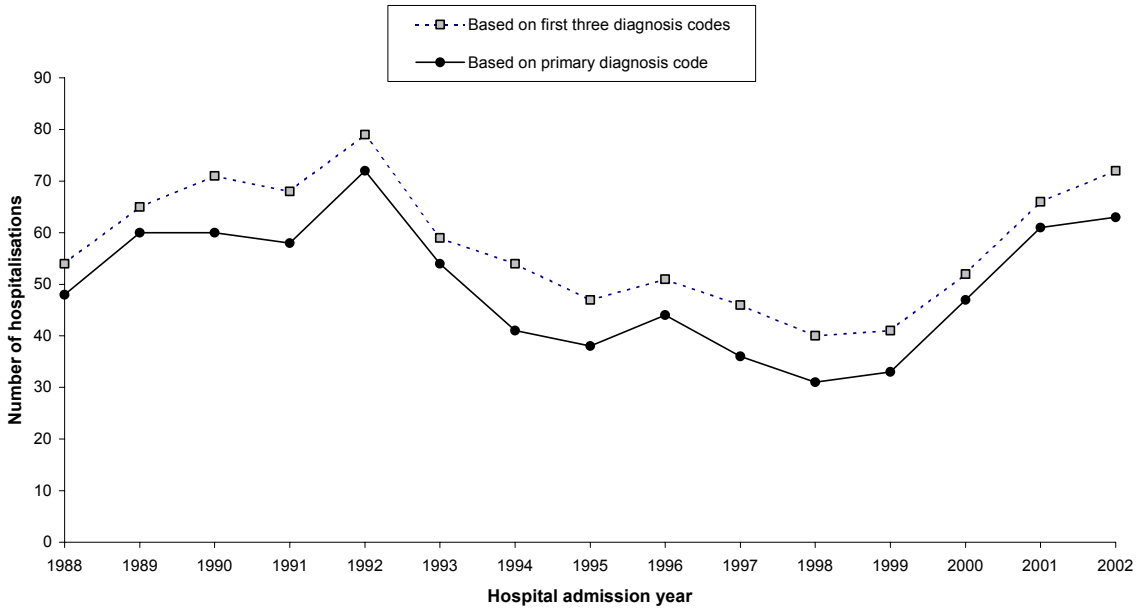
leptospirosis has been steadily increasing since 1999. There would also appear to be a high level of under-notification of the disease.

The following graphs show (i) the number of notified and laboratory-reported cases of leptospirosis each year since 1988, and (ii) the hospitalisations each year since 1988.

Leptospirosis laboratory-reported and notified cases by year, 1988 - 2002

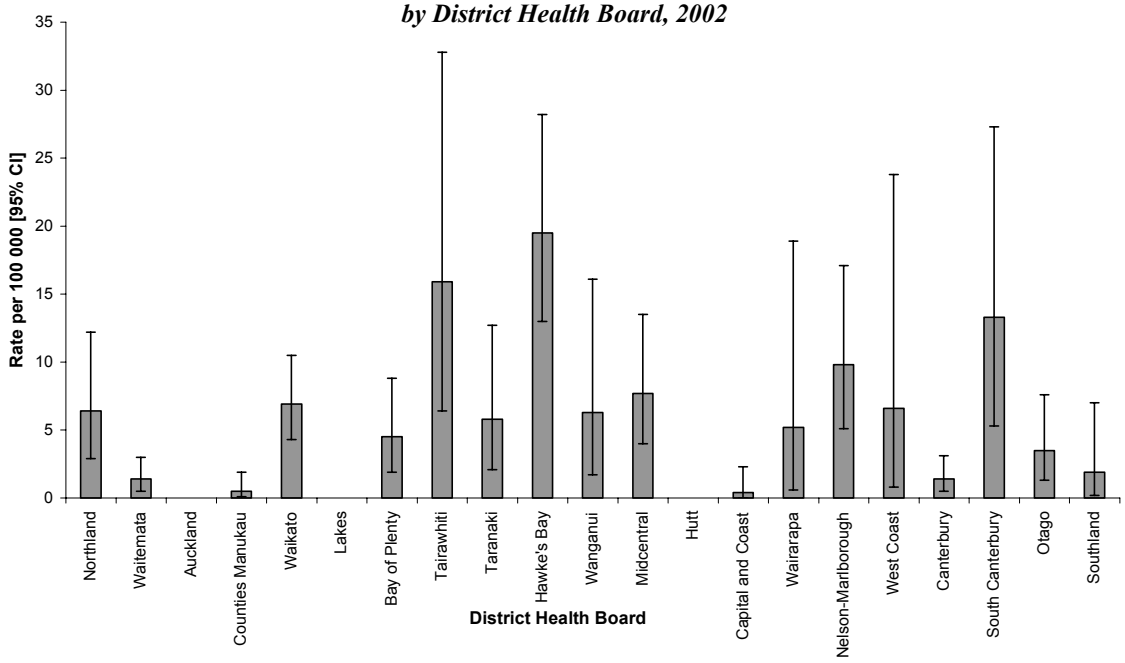


Leptosporidiosis hospitalisations by year, 1988 - 2002



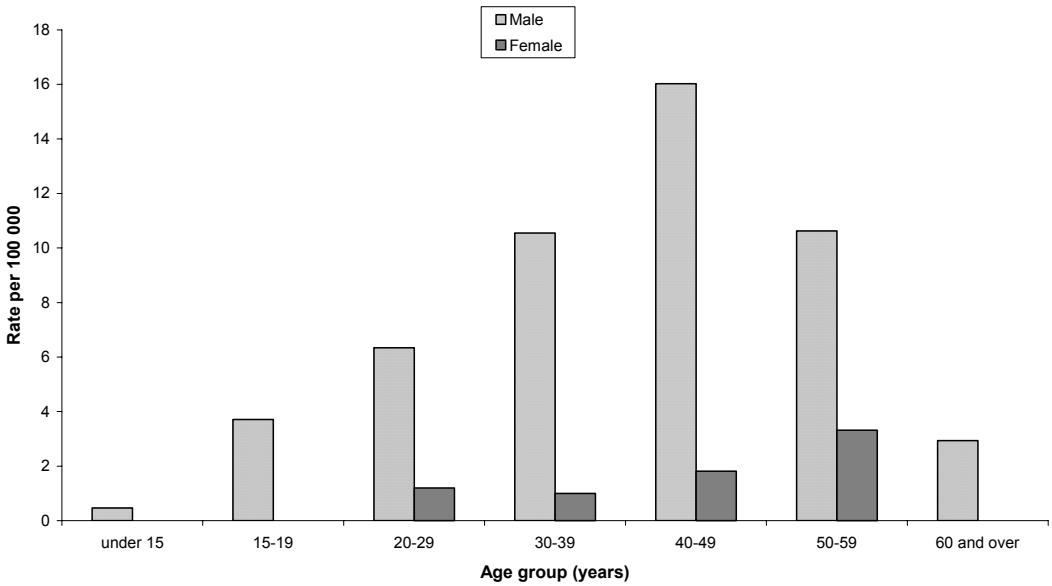
The rate of leptospirosis varied throughout the country (see chart below), with the highest crude rate of 19.5 per 100 000 (95% CI: 13.0-28.2) recorded in Hawke's Bay DHB. Rates significantly higher than the national average were also recorded in Tairawhiti (6.4 per 100 000), South Canterbury (5.3) and Nelson-Marlborough DHBs.

Leptospirosis crude notification rates by District Health Board, 2002



The following bar chart compares rates of leptospirosis by age and sex. Across all age groups, rates of disease were significantly higher among males than females. Age-specific rates were highest in the '40 to 49 years' age group, with a rate of 8.9 per 100 000 (95% CI: 6.6-11.8), followed by the '50 to 59 years' age group with a rate of 6.9 per 100 000 (95% CI: 4.6-10.0).

Leptospirosis notification rates, 2002
by age and sex



The following two tables illustrate the age and ethnic distribution of cases. Note that there were no notified cases of Pacific or 'Other' ethnicity during 2002. The age-standardised rate among Maori (6.2) was significantly higher than that in the European population (3.2). Overall, the highest rate of 19.0 per 100 000 (five times the national rate) occurred in persons of Maori ethnicity, aged between 40 and 49 years.

Leptospirosis notifications and age-specific rates by ethnicity, 2002

Ethnicity	Age group in years											
	<1	**1-4**	**5-9**	**10-14**	**15-19**	**20-29**	**30-39**	**40-49**	**50-59**	**60-69**	**70+**	**Total**
European	1	0	0	1	3	8	22	27	20	4	0	86
	3.4	0.0	0.0	0.6	1.9	2.6	5.5	6.8	6.0	1.7	0.0	3.3
Maori	0	0	0	0	1	4	5	11	4	1	0	26
	0.0	0.0	0.0	0.0	2.0	4.9	6.4	19.0	12.0	5.1	0.0	4.9
Pacific peoples	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
Other	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
Unknown	0	0	0	0	1	6	5	10	5	3	0	30
Total	1	0	0	1	5	18	32	48	29	8	0	142
	1.8	0.0	0.0	0.3	1.9	3.7	5.5	8.9	6.9	2.8	0.0	3.8

Number of cases
Rate per 100 000

Leptospirosis - crude and age-standardised rates by ethnicity, 2002

Ethnicity	Crude rate [95% CI]	Age-standardised rate* [95% CI]
European	3.3 [2.6, 4.1]	3.2 [2.6, 4]
Maori	4.9 [3.2, 7.2]	6.2 [4, 10.4]
Pacific peoples	0.0 [0, 1.5]	-
Other	0.0 [0, 1.2]	-
Total	3.8 [3.2, 4.4]	

* Directly standardised to the NZ population

The following table compares the number of different species of laboratory-reported *Leptospira* over the last seven years. As some results have been obtained via serological techniques, rather than by isolation, they must be interpreted with caution, due to the potential for cross-reacting among antibodies. The most commonly identified serovar among laboratory-reported cases was *L. hardjo* (43%), followed by *L. pomona* (35%).

Species/serovar distribution of laboratory-reported leptospira, 1995-2002

Leptospira species / Serovar	Year							
	1995	1996	1997	1998	1999	2000	2001	2002
L. interrogans sv *australis*	0	0	0	2	1	2	1	0
L. borgpetersenii sv *ballum*	25	28	12	27	17	22	15	15
L. interrogans sv *bratislava*	17	13	0	5	0	0	0	0
L. interrogans sv *canicola*	1	0	1	0	0	1	1	0
L. interrogans sv *copenhageni*	6	3	4	8	7	2	1	5
L. interrogans sv *pomona*	29	21	22	20	11	27	46	64
L. borgpetersenii sv *tarassovi*	10	4	7	12	9	8	11	15
L. borgpetersenii sv *hardjo*[2]	87	57	32	32	21	50	35	77
Unidentified *Leptospira* species	17	10	6	11	10	2	4	0
Total[1]	**183**	**136**	**84**	**117**	**76**	**114**	**113**	**181**

[1] More than one serovar was recorded for some cases
[2] Previously denoted *as L. interrogans* serovar *hardjo*

Occupation was recorded for 131 (92.3%) of the 142 notifications. Of these, 118 cases (90.1%) were recorded as engaged in occupations previously identified to present high risk for exposure to *Leptospira spp.* in New Zealand[18]. The proportion of 2002 leptospirosis cases in high-risk occupations has not changed appreciably over the last two years (cf. 88.4% in 2001 and 88.1% in 2000).

Exactly half the cases notified in 2002 had addresses classified as rural[a]. Of the 131 cases with recorded occupation, 54 (41.2%) were farmers or farm workers, and 64 (48.9%) worked in the meat processing industry (as either freezing workers, butchers, or meat inspectors). Twenty-two of the 54 farmers indicated they were dairy farmers. Leptospirosis cases also included one wool drier, a livestock consultant, a stock truck driver, and a crop field worker.

[a] 12.6% of the New Zealand population is classified as 'rural'

The majority (89% or 23/26) of Maori cases were meat workers, whereas meat workers accounted for less than half (48%) of the European cases. At the last Census, the number of employed meat processing workers aged 15 or over totalled 20 157, or 1.17% of the employed population aged 15 or over. The notification rate of leptospirosis among meat workers in 2002 was therefore around 3 per 1000 workers.

The most commonly identified serovars among laboratory-confirmed notifications were *L. hardjo* (56 cases), *L. pomona* (43 cases), *L. ballum* (12), *L. tarassovi* (7) and *L. copenhageni* (3). The table below shows the percentage of these cases recording contact with pigs, cattle, sheep, rodents and deer, respectively. Note that cases frequently recorded contact with more than one animal species. The proceeding table compares the proportion of notified cases in key occupations, by serovar. Together these two tables imply the following: *L. hardjo* is commonly associated with exposure to cattle and sheep in meat workers and farmers; *L. pomona* is commonly identified in meat workers; *L. ballum* is most frequently associated with exposure to cattle and rodents among dairy farmers.

The percentage of leptospirosis notified cases recording contact with different animals, by serovar

Serovar	Animal contact				
	Pigs	Cattle	Sheep	Rodents	Deer
L. hardjo (n=56)	5%	39%	23%	4%	4%
L. pomona (n=43)	7%	9%	21%	0%	5%
L. ballum (n=12)	0%	67%	8%	50%	0%
L. tarassovi (n=7)	14%	57%	14%	0%	0%
L. copenhageni (n=3)	0%	33%	0%	33%	0%

The number and percentage of leptospirosis notified cases in key occupations, by serovar

Serovar	Occupation				
	Dairy farmer	Other farmer	Meat worker	Other	Unknown
L. hardjo (n=56)	8 (14%)	17 (30%)	21 (38%)	3 (5%)	7 (13%)
L. pomona (n=43)	1 (2%)	9 (21%)	31 (72%)	1 (2%)	1 (2%)
L. ballum (n=12)	5 (42%)	3 (25%)	1 (8%)	2 (17%)	1 (8%)
L. tarassovi (n=7)	3 (43%)	1 (14%)	1 (14%)	1 (14%)	1 (14%)
L. copenhageni (n=3)	1 (33%)	1 (33%)	0 (0%)	1 (33%)	0 (0%)

Leptospirosis remains an important zoonotic disease in New Zealand, and arguably, our most important occupational infection.[55] There is consistent evidence from notification, laboratory and hospitalisation data that the incidence of disease is rising. This rise is being driven by an increase in *L. pomona* and to a slightly lesser extent, *L. hardjo.* These serovars are associated with work in the meat processing industry and with sheep and cattle contact. The data suggest that sheep may be emerging as a more important reservoir than previously thought.

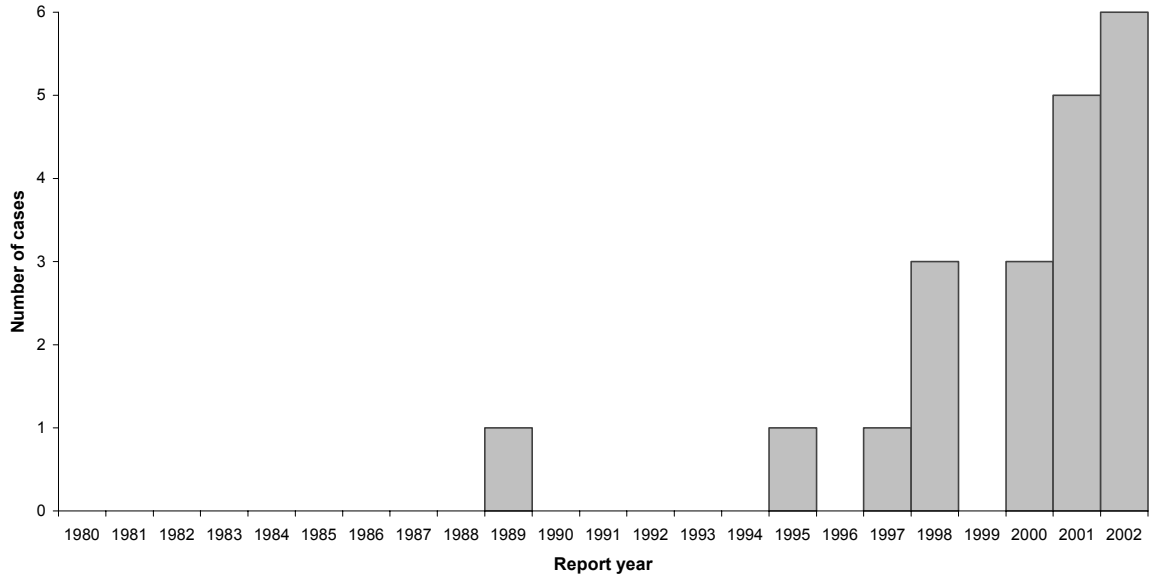
Primary amoebic meningoencephalitis

The last notification of primary amoebic meningoencephalitis was in 2000, when one case was notified. [56] The only previously reported cases were four in 1968 and one in 1972.

Rickettsial disease

Six cases of rickettsial disease were notified in 2002, all of whom were laboratory-confirmed. Five were recorded as murine typhus (*Ricketsia typhi*). In comparison, five confirmed cases of rickettsial disease were notified during 2001. Hospitalisation status was recorded for all cases in 2002, of whom two (33.3%) were hospitalised. The following graph shows the number of notified cases of rickettsial disease each year since 1980.

Rickettsial disease notifications by year, 1980 - 2002



All six notifications in 2002 were reported from Waitemata DHB. With the exception of one case, all past notified cases of rickettsial disease resided in the Auckland area. Two cases in 2002 were children, aged five and ten years respectively, and the remaining four cases were aged between 40 and 49 years. No cases recorded overseas travel during the incubation period. One female case was a cat and horse breeder. Her son was also a case. One male case was a waste management worker. The presence of rats was confirmed in the house and environs of another male case. No risk factors were recorded for the remaining two cases.

The presence of rickettsial disease in New Zealand was not confirmed until 2000, when testing of a DNA fragment from white cells collected from an Auckland case was found to have 100% homology with *R typhi*.[57] While only demonstrated with certainty in the Auckland region, it is likely that *R typhi*, the causative agent for murine typhus, is also present in other regions of New Zealand. Prevention of murine typhus is directed mainly at control of potential flea hosts, such as rats.

Taeniasis and cysticercosis

One case of taeniasis was notified in 2002. The case was a 27-year-old European male who had recently returned to New Zealand after spending several years in China. His hospitalisation status was not recorded. This is the first taeniasis notification since 1999 (when two cases were notified) and brings the total number of notifications since 1960 to 31. Just three notifications of cysticercosis have been received since 1969: one each in 1973, 1974 and 1992. According to hospital discharge data, there were two hospitalisations due to 'other' cestode infection, including taenaisis and cysticercoisis (ICD9 code 123) in 2002, compared to three in 2001, and a total of 19 hospitalisations in the last 15 years.

Trichinosis

There were no notifications of trichinosis in 2002, in contrast to the three cases notified during 2001.[58] The only other notified case of trichinosis was reported in 1992. Since 1988, hospital discharge data indicate a total of eight hospitalisations due to trichinosis (ICD9 code 124): one in 1988, three in 1992, one in 1996 and three in 2001.

Blood, tissue-borne and sexually-transmitted infections

This group of blood and tissue-borne diseases includes those spread by contaminated human blood and blood produces, injecting drug use which may expose people to contaminated blood, and human tissues used for transfusions purposes. This group of diseases overlaps with the sexually transmitted infections (STIs).

Creutzfeldt-Jakob disease[a]

The New Zealand Creutzfeldt-Jakob Disease (CJD) Registry was established in 1996 to monitor the various forms of CJD (sporadic, familial, iatrogenic and variant) in New Zealand. During 2002, five possible cases of CJD were referred to the New Zealand Registry. One subject proved on post mortem examination to have vascular dementia, and another is now considered on follow-up examination to have progressive supranuclear palsy. Two cases had CJD confirmed by post mortem examination and brain biopsy respectively. One further deceased subject has a current diagnosis of probable CJD with post mortem results awaited.

Creutzfeldt-Jacob disease cases, 2002

Age (years)	Sex	Site	Diagnosis
72	M	Christchurch	Definite
57	F	Dunedin	Definite
73	F	Hamilton	Probable

In comparison, one confirmed case of CJD was notified in 2001, and three confirmed cases were notified in 2000.

No cases of potential variant CJD (vCJD) have been detected in New Zealand. Ongoing surveillance of vCJD in the United Kingdom suggests that the increasing trend is slowing down. The death rate peaked in 2000. These findings are encouraging, although mortality might increase again in the future. [59]

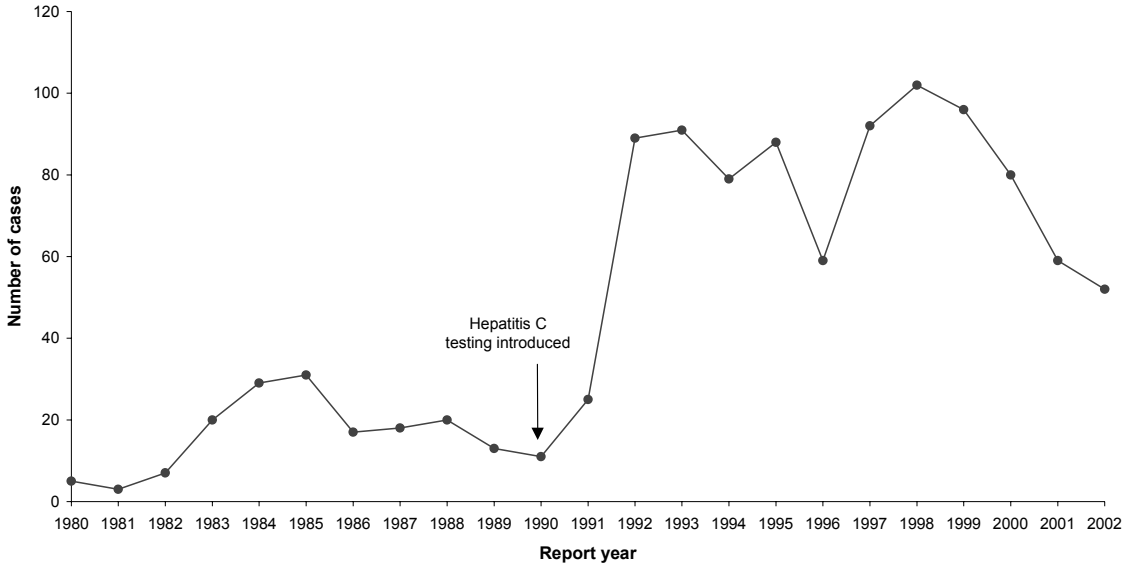
Hepatitis C

The surveillance of hepatitis C (HCV) in New Zealand focuses on acute rather than prevalent infections. Prior to June 1996, acute cases of hepatitis C were notifiable as non-A, non-B hepatitis (NANB). The majority of NANB hepatitis notifications over this time are likely to have been due to HCV. From 1 June 1996, acute HCV was made a notifiable condition in its own right. Surveillance of new or acute HCV infections is difficult because acute infection is often asymptomatic. Notifications of acute HCV are known to significantly underestimate the true number of new infections each year.

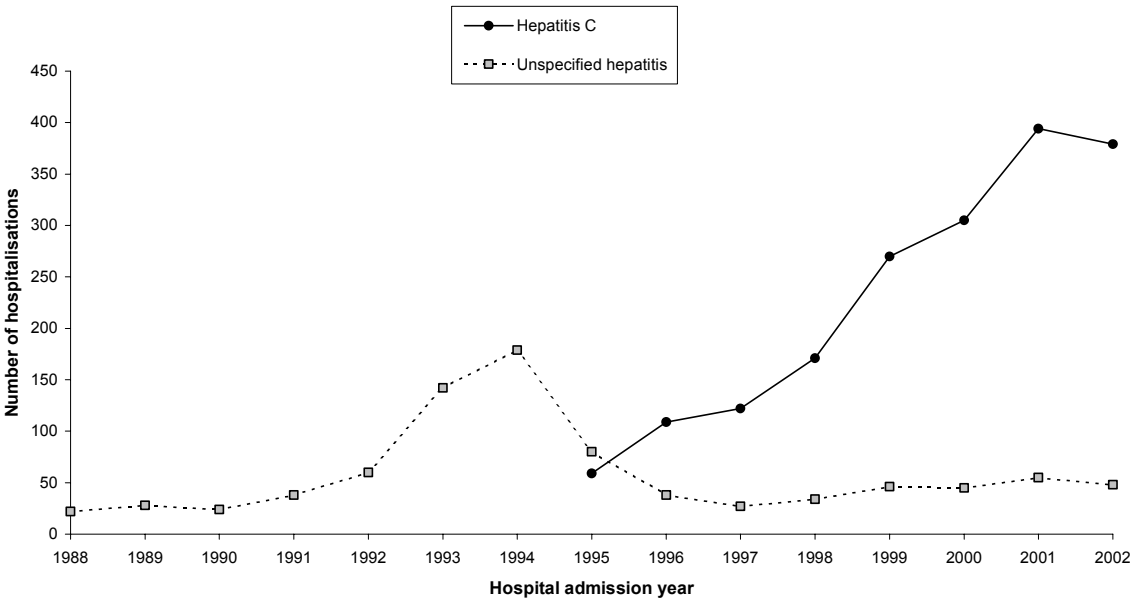
[a] Information supplied by Martin Pollock, Department of Preventive and Social Medicine, University of Otago

A total of 52 cases of acute hepatitis C was notified in 2002, representing an incidence rate of 1.4 per 100 000. This continues the downward trend in hepatitis C notifications since 1998. Of the 35 cases for whom hospitalisation status was recorded, 3 (8.6%) were hospitalised. In contrast, 379 hospitalisations due to hepatitis C (ICD9 codes 070.41, 070.44, 070.54) were recorded on hospital discharge data during 2002. This was a slight decrease from the 394 hospitalisations recorded the previous year. The following graphs show (i) notifications of NANB or hepatitis C each year since 1980, and (ii) hospitalisations due to 'unspecified' hepatitis (ICD9 codes 070.49,070.59,070.6,070.9) or hepatitis C each year since 1988.

Notifications of NANB hepatitis 1980 - 1995, notifications of hepatitis C 1996 - 2002

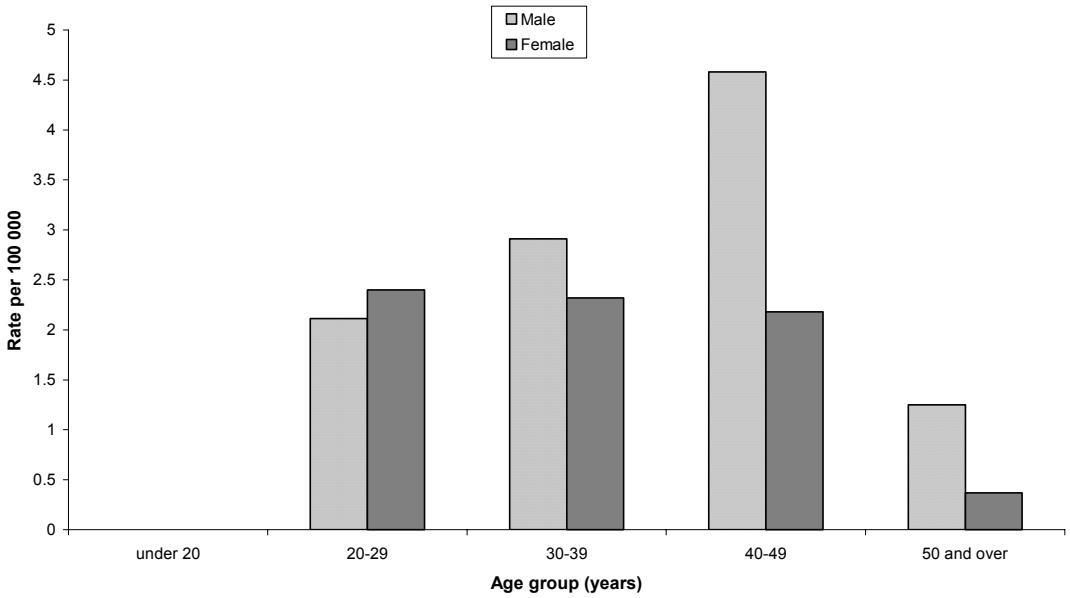


Hepatitis C and and unspecified hepatitis hospitalisations by year, 1988 - 2002



One Auckland outbreak of hepatitis C involving three cases was reported during 2002. Bay of Plenty DHB recorded a rate of 9.0 cases per 100 000 (95% CI: 5.1-14.6), significantly higher than the national average of 1.4 per 100 000. Notification rates were highest among males in the '40 to 49 years' age group, with a rate of 4.6 per 100 000. The age-specific rate of 3.3 (95% CI: 2.0-5.3) in the '40 to 49 years' age group was significantly higher than in the general population. The following chart compares notification rates by age and sex.

Hepatitis C notification rates, 2002
by age and sex



Ethnicity was recorded for 39 (75%) of the 52 notified cases. Of these, 30 were of European ethnicity, seven of Maori ethnicity and two of 'Other' ethnicity. It is difficult to estimate incidence rates among different ethnic groups, based on this incomplete data.

The following table shows a summary of risk factor information for hepatitis C in 2002. Some cases had more than one risk factor recorded. A history of injecting drug use remains an overwhelmingly important risk factor, being reported by 77.4% of new cases for whom this information was recorded, and by 66.7% of new cases in 2001.

Risk factors associated with hepatitis C notifications, 2002

Risk Factor	Yes	No	Unknown	Proportion[2]
History of injecting drug use	24	7	21	77.4%
Sexual contact with confirmed case or carrier	5	12	35	29.4%
Household contact with confirmed case or carrier	4	14	34	22.2%
Blood product or tissue recipients	4	17	31	19.0%
Travelled overseas during incubation period	2	17	33	10.5%
Other exposure to blood[1]	2	19	31	9.5%
Body piercing or tattooing in last 12 months	2	15	23	11.8

[1] Both cases were exposed to blood through relatives on renal dialysis.
[2] "Proportion" refers to the percentage of cases who answered "yes" out of the total number of cases for whom this information was known.

Passive surveillance of acute hepatitis C infection provides a very incomplete picture of the incidence and distribution of this infection. Modelling HCV transmission data suggests that there were likely to have been about 1280 new infections in 2000, which is about 15 times more than were indicated by the notification data [60]. A consistent feature of HCV transmission is the overwhelming importance of injecting drug use as a risk factor. [61]

Hepatitis D

There have been no notified cases of hepatitis D since the disease became notifiable in 1996. According to the first three diagnosis codes recorded on hospital discharge data, there was one hepatitis D hospitalisation in 2002, and a total of four since 1988 (ICD9 codes 070.42, 070.52).

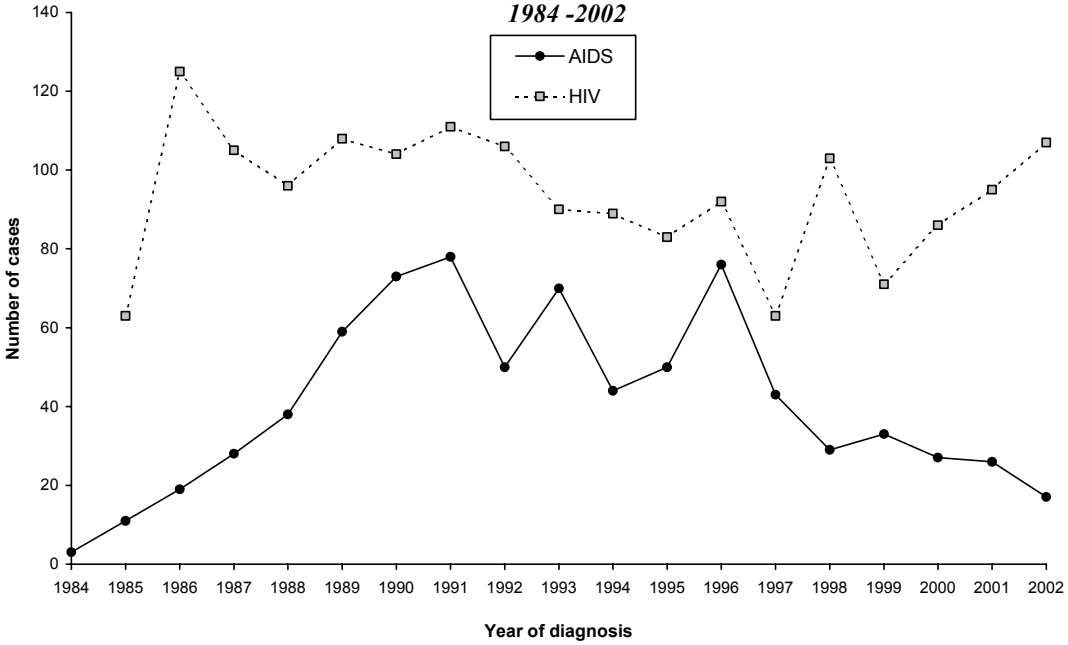
HIV/AIDS

In 2002, there were 17 cases of AIDS notified to the AIDS Epidemiology Group, representing an annual incidence rate of 0.45 per 100 000. This compares to 26 cases and a rate of 0.7 per 100 000 in 2001. A total of 772 cases of AIDS has been notified in New Zealand since surveillance began in 1983. Five deaths from AIDS were reported in 2002.

There were 136 new cases of HIV infection during 2002, of whom 107 were diagnosed through Western blot antibody test and 29 through viral load testing in New Zealand. A cumulative total of 1887 HIV infections have been diagnosed in New Zealand since testing began. This total includes those diagnosed through antibody testing in New Zealand, as well as those whose infection has been monitored with viral load testing. Many of the latter were initially diagnosed overseas and have not had antibody tests here.

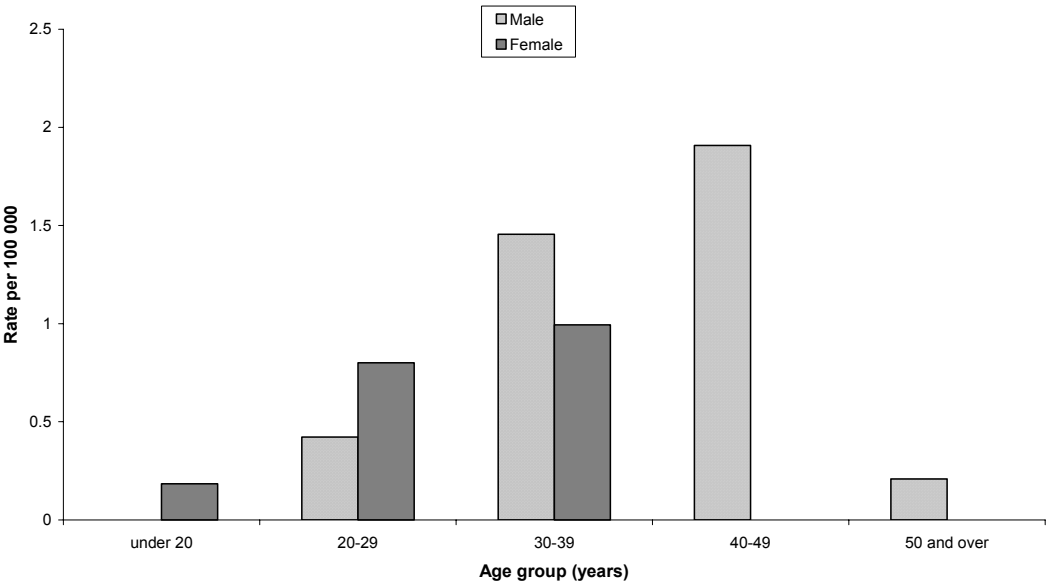
The following graph shows the number of AIDS notifications and HIV laboratory-confirmed cases since 1984. To allow comparison across years, only HIV cases diagnosed via the Western blot antibody test have been included.

AIDS notifications and HIV laboratory-confirmed cases by year, 1984 -2002



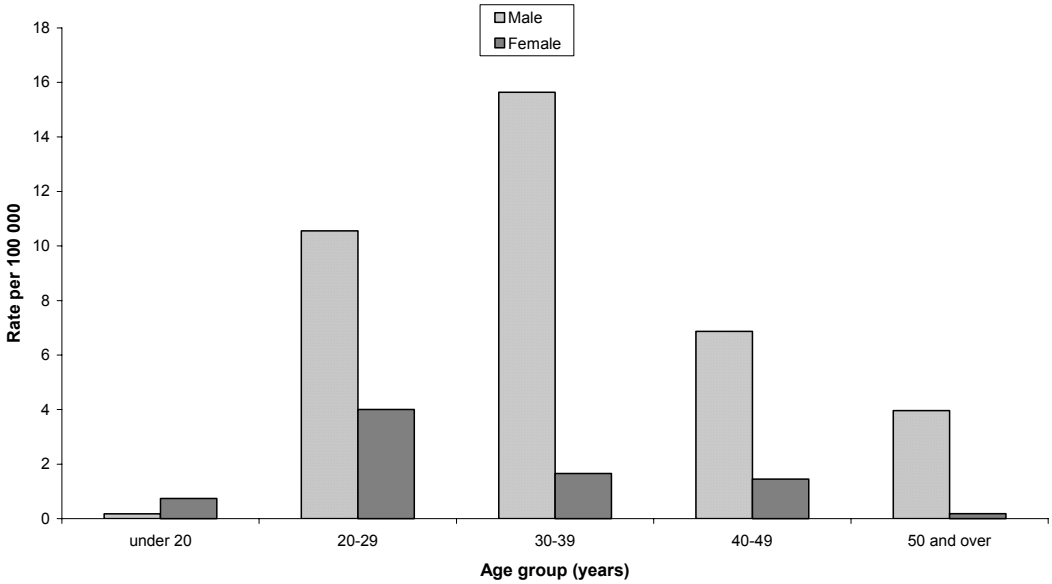
The following charts compare the age[a] and sex-specific notification rates of AIDS and HIV infection during 2002. Notification rates of AIDS were highest among males aged between 40 and 49 years, whereas HIV infection was most frequently diagnosed in males aged between 30 and 39 years.

AIDS notification rates, 2002 by age and sex



[a] Note that six male HIV cases had no age recorded.

HIV infection notification rates, 2002
by age and sex



The following table illustrates the sex and ethnic distribution of HIV and AIDS cases. Both HIV and AIDS notification rates were many times higher in people of 'Other' ethnicity, than in the general population.

HIV/AIDS notifications and rates by ethnicity, 2002

Ethnicity	AIDS			HIV infection		
	Male	Female	Total	Male	Female	Total
European	5	1	6	72	8	80
	0.4	0.1	0.2	5.7	0.6	3.1
Maori	3	1	4	8	0	8
	1.2	0.4	0.8	3.1	0	1.5
Pacific peoples	0	0	0	1	0	1
	0	0	0	1.0	0	0.5
Other	3	4	7	22	16	38
	2.5	3.1	2.8	18.6	12.4	15.4
Unknown	0	0	0	9	0	9

Number of cases
Rate per 100 000

The table below shows the most likely risk behaviour categories of people notified with AIDS or diagnosed with HIV in 2002. During 2002, whilst only five men infected through homosexual contact were notified with AIDS, there were 50 diagnosed with HIV through antibody testing in New Zealand. Although some men now being diagnosed might have acquired their infection many years ago, there is evidence that new infections are still occurring. In 2002, heterosexual contact was responsible for an increasing proportion (65%) of new AIDS notifications, compared to 54% the previous year. Injecting drug use remains an uncommon risk behaviour category for both HIV and AIDS.

Risk behaviour category for AIDS notifications and HIV infections, 2002

		AIDS[1]				HIV Infection[2]			
		2002		Total to 31 Dec 2002		2002		Total to 31 Dec 2002	
Risk category	**Sex**	**Cases**	**%**	**Cases**	**%**	**Cases**	**%**	**Cases**	**%**
Homosexual contact	Male	5	29.4	592	76.7	67	49.3	1015	53.8
Homosexual & IDU	Male	0	0.0	10	1.3	0	0.0	23	1.2
Heterosexual contact	Male	6	35.3	51	6.6	19	14.0	171	9.1
	Female	5	29.4	42	5.4	20	14.7	200	10.6
Injecting drug user (IDU)	Male	0	0	13	1.7	8	5.9	45	2.4
	Female	0	0	5	0.6	0	0.0	11	0.6
Blood product recipient	Male	0	0	16	2.1	0	0.0	34	1.8
Transfusion related	Male	0	0	2[3]	0.3	0	0.0	9	0.5
	Female	0	0	1[3]	0.1	1	0.7	7	0.4
	NS	0	0	0	0.0	0	0.0	5	0.3
Perinatal	Male	0	0	3	0.4	0	0.0	9	0.5
	Female	1	5.9	4	0.5	2	1.5	9	0.5
Awaiting information/ Undetermined	Male	0	0	30	3.9	18	13.2	298	15.8
	Female	0	0	2	0.3	1	0.7	27	1.4
	NS	0	0	0	0.0	0	0.0	13	0.7
Other	Male	0	0	0	0.0	0	0.0	4	0.2
	Female	0	0	1	0.1	0	0.0	7	0.4
Total		**17**	100.0	**772**	100.0	**136**	100.0	**1887**	100.0

[1] Reported by date of notification.
[2] Includes people who have developed AIDS. Numbers are recorded by date of diagnosis for those reported through antibody testing and by time of first viral load for those reported through viral load testing. The latter include many who have initially been diagnosed overseas and have not had an antibody test here.
[3] Acquired overseas

The following table shows the country in which HIV infection was probably acquired, for cases diagnosed via Western blot antibody testing in New Zealand. Of the 29 additional cases of HIV infection in 2002 diagnosed as a result of viral load testing, 5 cases (or 20.8% of the 24 cases for whom this information was recorded) were thought to have acquired the infection in New Zealand, and the remaining 19 cases (79.2%) overseas.

Place where infection was probably acquired, for HIV cases diagnosed via Western blot antibody testing in New Zealand, 1996-2002

Year of diagnosis	**Place where infection probably acquired**			**Total diagnosed via antibody testing**
	New Zealand[1] No. (%)[2]	**Overseas No. (%)[2]**	**Unknown**	
1996	42 (50.0%)	42 (50.0)	9	93
1997	31 (52.5%)	28 (47.5%)	4	63
1998	28 (29.8%)	66 (70.2%)	9	103
1999	26 (38.8%)	41 (61.2%)	4	71
2000	31 (38.3%)	50 (61.7%)	5	86
2001	41 (45.1%)	50 (54.9%)	4	95
2002	43 (41.7%)	60 (58.3%)	4	107

[1] Places of infection recorded as 'New Zealand or Australia' have been classified as 'New Zealand'.
[2] Percentage is of the total number of cases for whom information on place of infection was recorded.

New Zealand has been relatively successful at controlling the HIV epidemic [62] and improved treatments are slowing the progression of HIV to AIDS. However, these surveillance data illustrate the potential for continuing transmission of HIV within New Zealand, and the importance of migration and travel as sources for this infection.

Sexually-transmitted infections

Surveillance of STIs in New Zealand continues to be based on data from specialist sexual health clinics (SHCs). Although sexual health clinics see only a portion of the population with STIs, their data provide the most comprehensive source of information on the epidemiology of STIs in New Zealand. Since 1998, STI surveillance has been expanded to include data from family planning clinics (FPCs), student and youth health clinics (SYHCs), and laboratories in Waikato, Bay of Plenty and Auckland. National data on sexually transmitted infections are reported in full in the STI Annual Surveillance Report, 2002. [6]

In 2002, the 27 sexual health clinics reported 9298 confirmed STI cases. Of all sexual health clinic attendees, 12.3% were diagnosed with an STI. Genital warts was the most commonly reported STI, followed by confirmed chlamydia, NSU in males, genital herpes, confirmed gonorrhoea and syphilis. No cases of chancroid, granuloma inguinale or lymphogranuloma venereum were reported during 2002. The following table displays the number of confirmed STI cases at sexual health clinics between 1996 and 2002.

Number of confirmed STI cases at sexual health clinics, 1996-2002

STI	1996	1997	1998	1999	2000	2001	2002
Chlamydia	1665	1992	2263	2331	2870	3238	3372
Gonorrhoea	274	291	329	384	491	533	532
Genital Herpes	802	719	724	682	658	638	713
Genital Warts	3660	3691	3434	3083	3181	3304	3510
Syphilis	23	27	24	23	13	18	47
NSU (males only)	-	-	830[1]	874	825	1054	1124

[1]Annualised, based on July to December 1998 data.

Key surveillance points

- *Genital warts:* Genital warts was the most commonly diagnosed STI at SHCs, with 3510 first diagnoses reported in 2002. Highest rates were found in the '20 to 24 years' age group for males, and the '15 to 19 years' age group for females. There were comparable rates across all ethnic groups.

- *Chlamydia and gonorrhea:* In 2002, a total of 3372 infections due to chlamydia and 532 due to gonorrhoea were reported to ESR from Sexual Health Clinics. Of sexual health clinic patients diagnosed with confirmed gonorrhoea, 222 (41.7%) were diagnosed with concurrent infections. Reporting laboratories from the Auckland, Waikato and the BOP regions confirmed 10 307 cases of chlamydia and 927 cases of gonorrhoea, while FPCs reported 1373 cases of chlamydia and 184 of gonorrhoea, and SYHCs reported 391 cases

and 18 cases, respectively. These cases represent a large number of potentially curable infections.

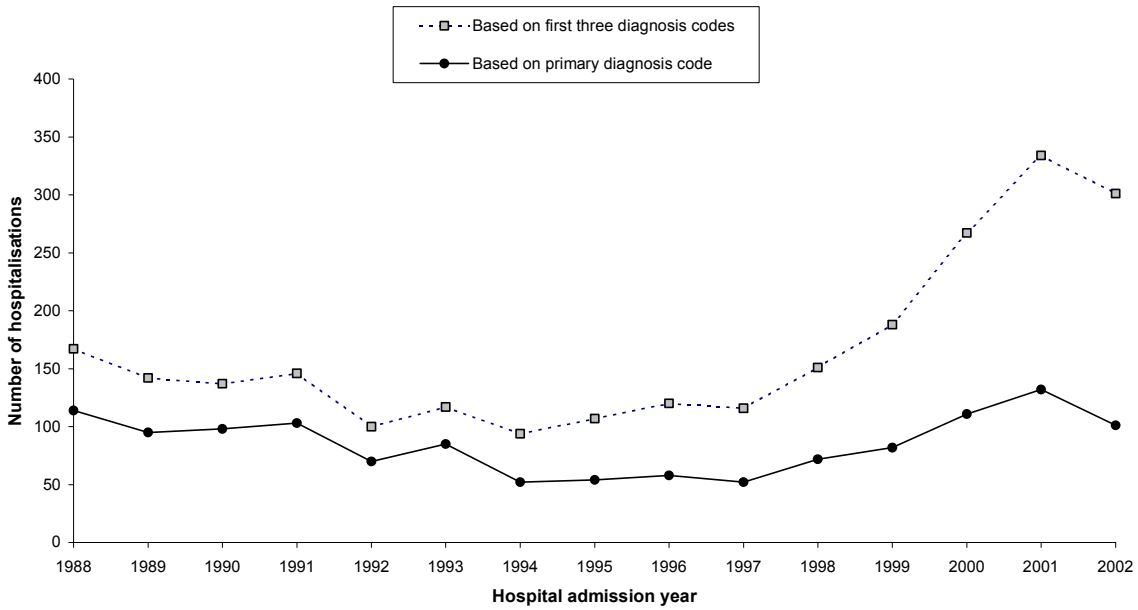
- *Age and ethnic distribution:* The majority of STIs were in teenagers and young adults, with about two-thirds of gonorrhoea, chlamydia and genital warts cases in people aged less than 25 years. Young people were also more likely to be diagnosed with concurrent infections. Rates of chlamydia and gonorrhoea at sexual health clinics and family planning clinics were considerably higher in Maori and Pacific peoples than in Europeans.

- *Trends and international comparisons:* Chlamydia infections are still increasing in New Zealand and may soon overtake genital warts as the most common infection in sexual health clinic patients. Notifications of C. trachomatis by participating laboratories during 2002 increased, representing a rate of 598 per 100 000 population - five times higher than that reported in Australia during the same period. Similarly, the rate of N gonorrhoeae, at 54 per 100 000 population, was almost double that of Australia's.

STI hospitalisation data

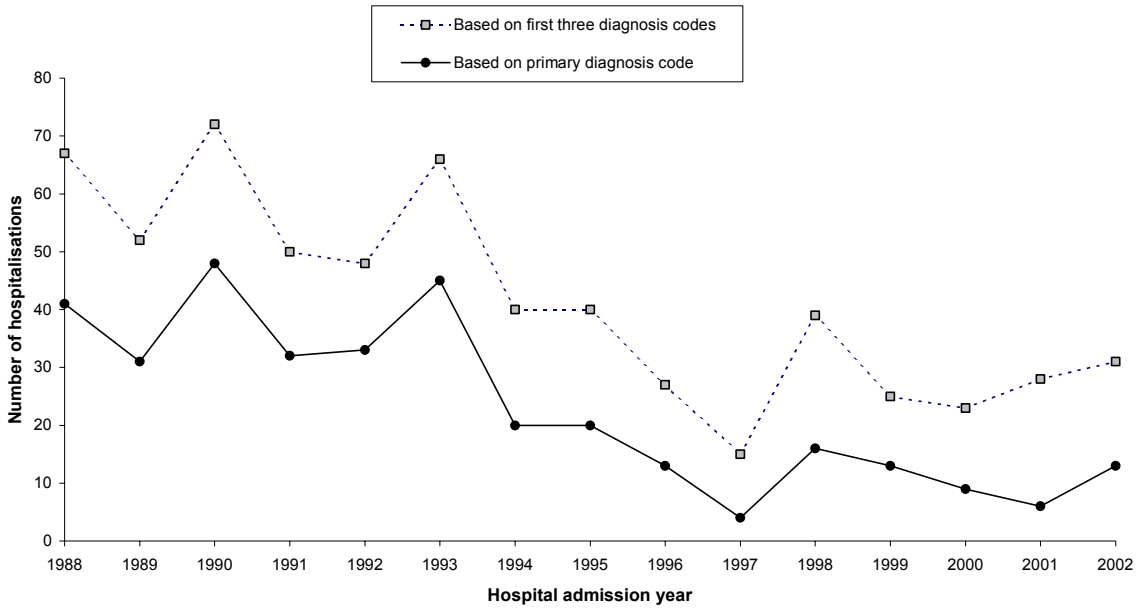
The following graph illustrates the trend in STI hospitalisations since 1998. The ICD9 codes 090-097 (for syphilis and other venereal diseases) were included in the analysis. A small drop in hospitalisations due to sexually transmitted infections was observed in 2002.

Sexually transmitted infections - hospitalisations by year, 1988 to 2002

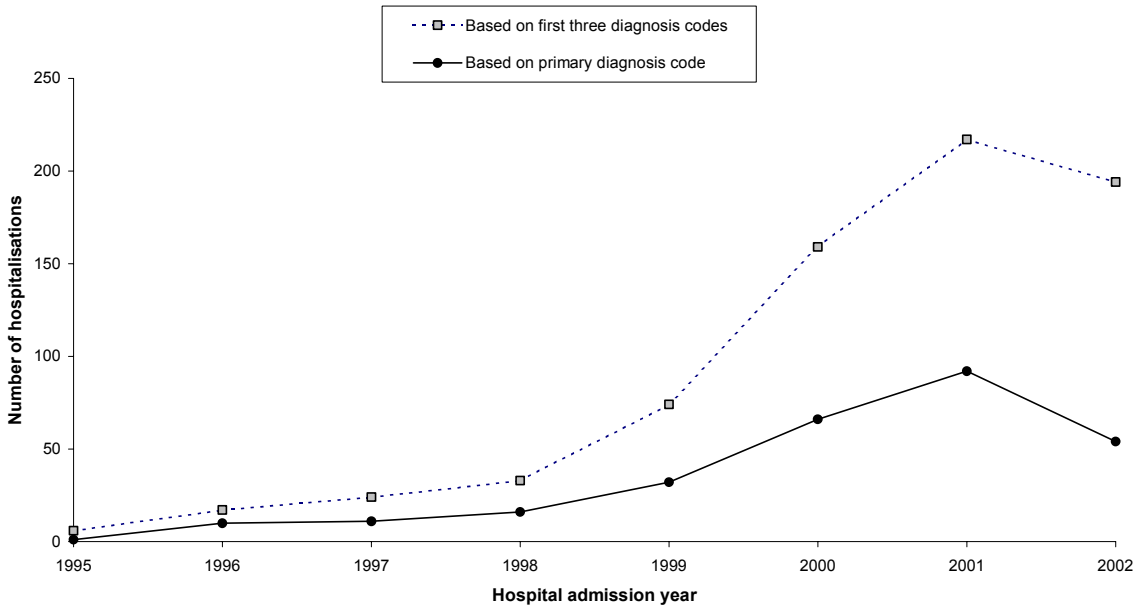


The following graphs show the trend in hospitalised cases of chlamydia, syphilis and gonococcal infections since 1988. Respective ICD9 codes of 099.5, 090-097, and 098 were included in the analysis. There was a small increase in the number of syphilis hospitalisations in 2002, but a drop in hospitalisations due to both chlamydia and gonococcal infections.

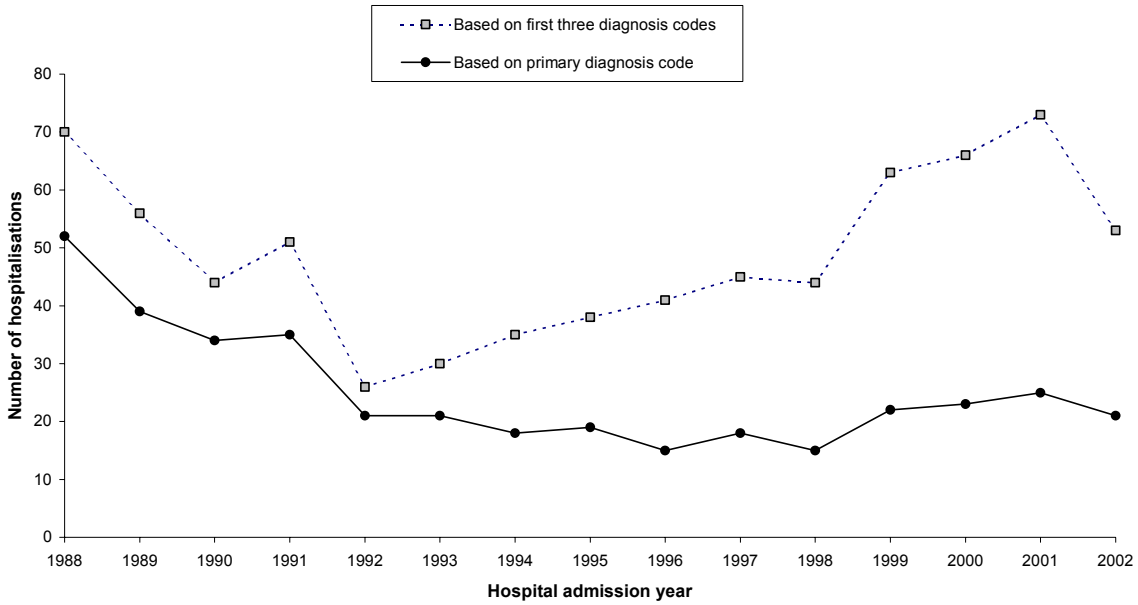
Syphilis hospitalisations by year, 1988 - 2002



Chlamydia hospitalisations by year, 1995 - 2002



Gonococcal infection hospitalisations by year, 1988 - 2002



These surveillance data all suggest a general increase in incidence of the bacterial STIs (chlamydia, gonnorrhoea and syphilis) over recent years. Limited laboratory-based surveillance has confirmed that the population rate of chlamydia, gonnorrhoea is considerably higher than that reported by sexual health clinics. [63] Overall, New Zealand rates of STIs are high compared with most other developed countries.[64] It is hard to draw conclusions about the factors contributing to this pattern without more extensive surveillance and research.

Antibiotic resistance and Hospital-acquired infections

Antimicrobial resistance

The prevalence of resistance among common, important clinical pathogens, which have been continuously monitored since 1988, is shown in the table below. Most antimicrobial resistance data are only available in a complete analysed form up to the end of 2001. Of particular note are the following trends:

- An increasing prevalence of MRSA generally - an increasing proportion of which is multiresistant (that is, resistant to at least two antibiotic classes in addition to β-lactams)
- A high prevalence of mupirocin-resistant *Staphylococcus aureus* since the mid-1990s
- Among *Streptococcus pneumoniae* from cases of invasive disease, a decrease in penicillin non-susceptibility (resistance and intermediate resistance) since 1999 and a decrease in non-susceptibility to third-generation cephalosporins (such as ceftriaxone) in 2001
- Stable levels of trimethoprim resistance among urinary *E. coli,* and continuing low levels of nitrofurantoin and fluoroquinolone resistance
- An increasing prevalence of ciprofloxacin resistance in *Neisseria gonorrhoeae*, with a four-fold increase in resistance in 2001.

However, some other important resistances emerging in other countries remain uncommon in New Zealand. Of particular note, vancomycin-resistant enterococci (VRE), while isolated in small numbers, have not become established in New Zealand hospitals. In addition, multidrug-resistant tuberculosis (MDR-TB) isolates remain uncommon, and there does not appear to have been any transmission of MDR-TB within New Zealand.

Prevalence of antimicrobial resistance, 1988-2001

Pathogen	Antimicrobial	Percent resistance[1] (number tested)				
		1988-1990	1991-1993	1994-1996	1997-1999	2000-2001
S aureus[2]	methicillin	0.5 (37466)	0.6 (42839)	2.8 (58283)	4.9 (136356)	6.8 (157015)
	erythromycin	8.1 (34900)	6.8 (40425)	8.0 (54870)	10.8 (134350)	12.0(143453)
	co-trimoxazole	1.0 (11783)	1.1 (27469)	0.8 (32926)	0.6 (91391)	1.1 (85913)
	mupirocin	NA[3]	0 (16)	10.1 (9291)	18.2 (37173)	20.7 (60216)
Methicillin-resistant *S aureus*[4]	erythromycin	52.9 (263)	58.2 (701)	31.5 (2249)	26.2 (1303)	37.0 (875)
	co-trimoxazole	24.3 (263)	24.8 (701)	8.6 (2249)	1.8 (1303)	4.3 (875)
	mupirocin	4.6 (263)	2.0 (701)	6.4 (2244)	6.0 (1303)	9.1 (875)
	rifampicin	1.1 (263)	13.0 (701)	0.3 (2249)	0.8 (1303)	0.7 (875)
S pneumoniae, non-invasive disease[2]	penicillin[5]	1.8 (2372)	0.8 (3720)	9.5 (7076)	19.0 (10976)	21.3 (8372)
	erythromycin	1.8 (2334)	1.3 (3554)	8.3 (6832)	14.5 (11212)	18.3 (10655)
	tetracycline	5.6 (1760)	1.7 (3376)	10.5 (5019)	11.2 (5993)	14.6 (4981)
S pneumoniae, invasive disease[6]	penicillin[5]	1.0 (382)	1.4 (694)	3.4 (989)	15.0 (1182)	14.5 (1003)
	erythromycin	0.8 (382)	1.9 (694)	2.6 (989)	4.1 (853)	6.5 (1002)
	cefotaxime[5]	0.3 (382)	0.1 (694)	1.8 (989)	7.3 (1182)	6.6 (1003)
Enterococcus spp[2]	amoxycillin[7]	1.6 (6127)	2.3 (2573)	1.5 (7373)	2.4 (17548)	2.5 (14905)
	vancomycin	NA	0 (148)	0.2 (1141)	0.5 (4752)	0.6 (4525)

Pathogen	Antimicrobial	Percent resistance[1] (number tested)				
		1988-1990	1991-1993	1994-1996	1997-1999	2000-2001
E coli, urinary isolates[2]	amoxycillin[7]	NA	56.2 (29394)	55.9(48706)	56.0(138712)	55.3(147841)
	amox/clav	NA	6.9 (27249)	10.6(42666)	12.2(136326)	9.5 (143922)
	trimethoprim	NA	18.8 (29340)	19.6(48098)	22.6(111710)	22.5(153982)
	nitrofurantoin	NA	2.2 (28331)	1.6 (48123)	1.7 (124362)	1.5 (152131)
	fluoroquinolone	NA	0.2 (7014)	0.5 (40032)	0.6 (118917)	1.4 (149595)
E coli, non-urinary isolates[2]	amox/clav	NA	18.3 (2318)	22.8 (7358)	21.8 (15948)	18.4 (8843)
	cefuroxime	NA	2.3 (1158)	3.2 (6309)	4.5 (6893)	3.9 (5182)
	gentamicin	NA	0.5 (3200)	0.8 (10352)	0.9 (13789)	1.7 (7951)
	fluoroquinolone	NA	0.1 (728)	0.5 (4717)	0.8 (10800)	2.0 (6868)
P aeruginosa[2]	gentamicin	6.5 (11832)	5.8 (5918)	12.5 (9556)	9.5 (20542)	11.8 (17617)
	tobramycin	1.4 (1759)	3.1 (2535)	3.9 (6757)	2.8 (11033)	3.7 (7493)
	ceftazidime	NA	6.6 (1006)	5.0 (4832)	5.2 (11147)	3.6 (9895)
	fluoroquinolone	NA	8.4 (1652)	8.8 (8123)	9.9 (16551)	9.8 (15435)
H influenzae, non-invasive disease[2]	amoxycillin[7]	10.2 (4347)	8.4 (4131)	12.0(12244)	19.3 (18852)	22.0 (21640)
	amox/clav	0.9 (555)	1.1 (1136)	1.1 (9839)	0.6 (15040)	0.7 (10850)
	co-trimoxazole	NA	11.4 (1581)	11.9 (6605)	14.7 (13964)	17.2 (17561)
	tetracycline	NA	1.7 (2082)	1.0 (7810)	1.5 (13007)	1.2 (10917)
H influenzae, invasive disease[6]	amoxycillin[7]	14.9 (388)	13.2 (478)	21.8 (179)	11.5 (122)	15.7 (102)
	amox/clav	0.3 (388)	0.2 (478)	3.4 (179)	1.6 (122)	2.0 (102)
	cefuroxime	0.3 (388)	0.8 (478)	3.4 (179)	4.9 (122)	1.0 (102)
N meningitidis, invasive disease[6]	penicillin[8]	2.2 (139)	2.1 (291)	3.9 (659)	7.9 (431)	7.3 (573)
	rifampicin	0 (139)	0.3 (291)	0 (659)	0 (431)	0 (573)
N gonorrhoeae[2,9]	penicillin	NA	16.4 (85)	11.6 (879)	10.4 (1437)	7.0 (1684)
	fluoroquinolone	NA	0 (85)	0.7 (864)	1.8 (1437)	6.5 (1600)
M tuberculosis[2]	isoniazid	NA	NA	4.6 (438)	8.2 (757)	7.9 (543)
	rifampicin	NA	NA	0.7 (438)	1.3 (757)	0.4 (543)
	MDR[10]	NA	NA	0.7 (438)	0.9 (757)	0.2 (543)

Notes:

1 intermediate-resistance not included in resistant category unless otherwise stated (refer footnotes 5 and 8 below)
2 collated clinical laboratory data
3 NA = not available
4 MRSA isolates tested by ESR
5 includes intermediate resistant and resistant isolates
6 invasive disease isolates tested by ESR
7 ampicillin used in laboratory testing
8 reduced susceptibility (MIC 0.12-0.25 mg/L)
9 data from northern North Island only up until 2000, thereafter national data used
10 multidrug resistant (ie, resistant to at least isoniazid and rifampicin)

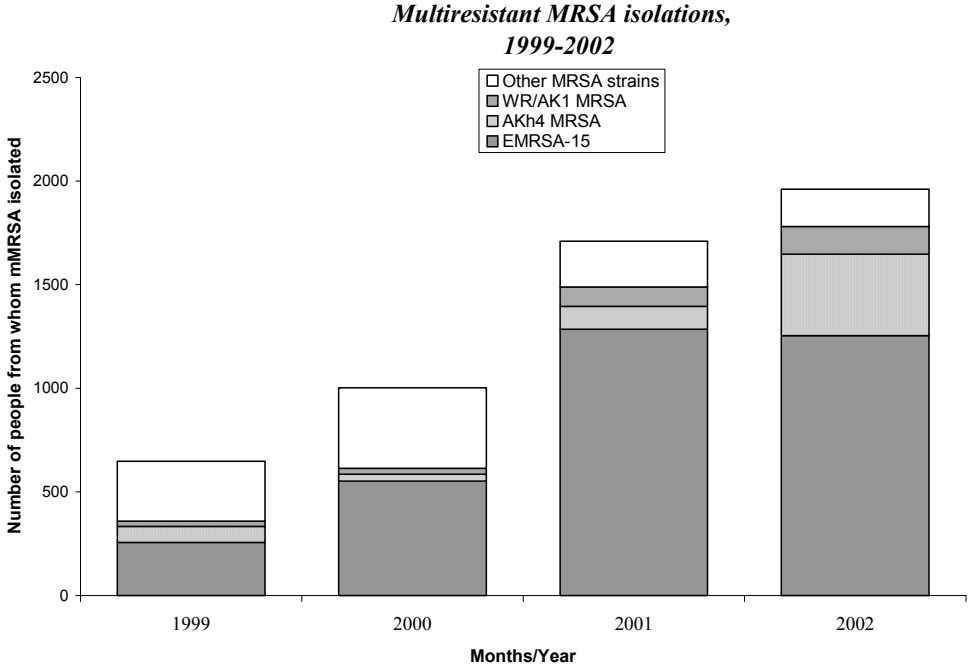
Hospital-acquired infections

This group of infections are not under national surveillance. Prevalence surveys carried out at Auckland hospital between 1996 and 1999 were used to calculate the cumulative incidence of hospital-acquired infections for all patients as 6.3% [65]. Based on this, the estimated cost is $51.35 and $85.26 million, respectively, for medical and surgical admissions to all hospitals in New Zealand. [66] A nationwide survey of medical records carried out in 1998 to identify the occurrence and impact of adverse events in New Zealand public hospitals found that 4.8% of 6579 records had hospital-acquired infection/sepsis recorded.[67]

Multiresistant methicillin-resistant *Staphylococcus aureus*

The increase in the incidence of multiresistant methicillin-resistant *Staphylococcus aureus* (mMRSA) in 2002 was smaller than in recent years, with an increase of 14.6% from an annual rate of 45.8 per 100 000 in 2001 to 52.5 per 100 000 in 2002. mMRSA are defined as *S. aureus* resistant to two or more classes of antibiotics in addition to ß-lactams. mMRSA from 1961 people (1827 patients and 134 healthcare workers) were referred to ESR in 2002.

Information on whether mMRSA was causing infection or colonising was reported for 1256 of the people from whom mMRSA was isolated. Of these, 79.1% were infected and 20.9% were colonised. Nearly three-quarters (73.7%) of the 1827 patients with mMRSA were reported to be patients in a healthcare facility (HCF) or had been in a HCF in the previous three months. The following graph shows the number of mMRSA isolations each year since 1999.

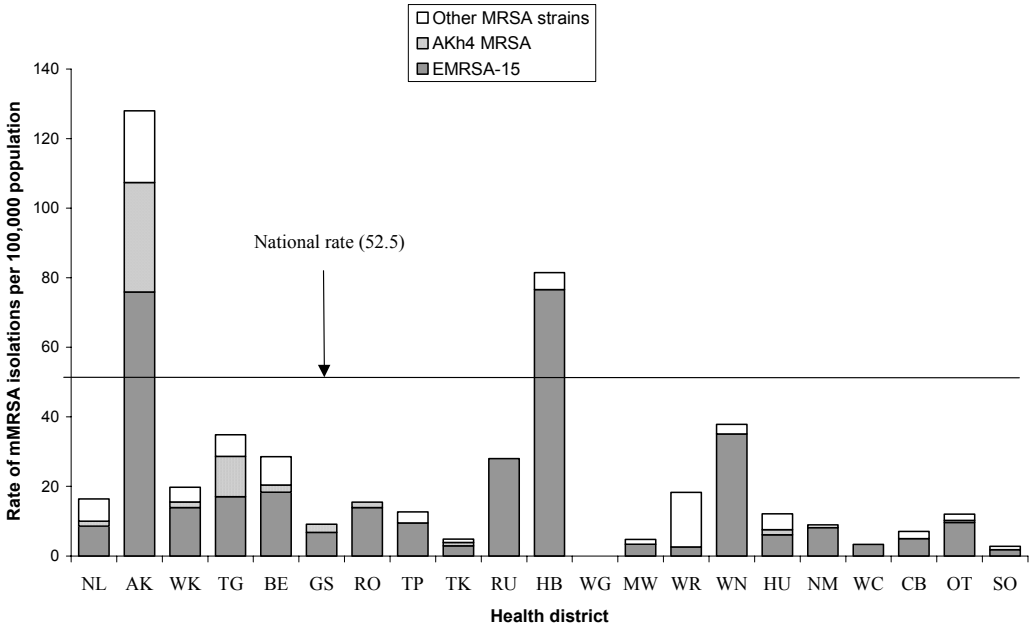


Three mMRSA strains were predominant in 2002:

- EMRSA-15, a British epidemic mMRSA strain, was isolated from 1254 people and accounted for 63.9% of all mMRSA isolations, a decrease from a proportion of 75.0% in 2001. This strain is typically isolated from elderly patients in hospital and other healthcare facilities.
- The AKh4 MRSA strain was isolated from 393 people and accounted for 20.0% of all mMRSA isolations, an increase from a proportion of 7.7% in 2001. This strain is a typical multiresistant Australian MRSA. It is predominantly isolated from patients hospitalised in the Auckland area, especially in Middlemore Hospital.
- The WR/AK1 MRSA strain was isolated from 134 people and accounted for 6.8% of all mMRSA isolations. This strain is most commonly isolated from children and young adults either in the community or hospital in the Auckland area.

The following graph compares the rate of mMRSA isolations in 2002 among different health districts. The geographic distribution of mMRSA displayed the usual pattern, with the highest rate occurring in the Auckland area. The next highest rates, in the Hawkes Bay and Wellington health districts, were comprised largely of EMRSA-15 isolations. From 2001 to 2002, rates rose in the Auckland area, decreased in Tauranga Health District, and remained at a similar level in the Hawkes Bay and Wellington health districts.

Incidence rate of multiresistant MRSA by health district, 2002



The antimicrobial resistance patterns of the three most commonly isolated mMRSA strains in 2002 are shown below.

Typical resistance patterns of the most common mMRSA strains in 2002

Strain	Resistant to:
EMRSA-15	ciprofloxacin and erythromycin[1]
AKh4	ciprofloxacin, clindamycin, co-trimoxazole, erythromycin, gentamicin and tetracycline
WR/AK1	fusidic acid and high-level mupirocin[2]

[1] Some isolates of EMRSA-15 are erythromycin susceptible and are therefore not categorised as multiresistant. Erythromycin-resistant isolates also have inducible clindamycin resistance.

[2] Some isolates WR/AK1 are also resistant to erythromycin

Congenital and perinatal infections

This category includes a number of infectious diseases that may be transmitted from an infected mother to the foetus or newborn baby during birth. Perinatal listeriosis is described in the section on enteric diseases and congenital rubella syndrome and hepatitis B in the section of vaccine preventable diseases (though perinatal infection is almost invariably asymptomatic so will not be detected by routine disease surveillance). Many congenital infections may be difficult to detect in newborn infants and surveillance data are lacking.

Group B streptococcal sepsis of the newborn

Infection with *Streptococcus agalactiae* (Group B Streptococcus) can cause serious invasive disease in newborn infants. This disease is not notifiable. Microbiological surveillance depends on the voluntary referral of isolates to the Streptococcal Reference Laboratory at ESR. During 2002, isolates were received from 121 cases of invasive group B streptococcal disease. Forty-three cases were from neonatal sepsis of which 28 were classified as early onset disease (occurring within seven days of birth) and 15 as late onset. Although fewer than in 2001, cases caused by serotype III continued to be the most common accounting for 34.9% (15/43). In 2001 60%(15/25) were serotype III. Most notable was the increase in serogroup B streptococcal blood culture isolates from adults. Seventy-eight such isolates were received and of these 29.5% (23/78) were serotype V.

In 2002, hospital discharge data for Group B Streptococcal disease (ICD9 code 038.0) recorded ten cases in newborns or infants under the age of one year (where this code was any one of the first nine diagnoses). Of these, three were ‘early onset’ disease cases. Corresponding figures for 2001 were 23 cases, including eight early onset cases.

New, exotic and imported infections

This section includes travel-associated and vector-borne diseases.

The following table shows the proportion of cases recording overseas travel during the incubation period, for commonly travel-associated notifiable diseases. All 66 dengue cases for whom travel information was recorded, reported overseas travel. All malaria cases were also imported, though only 85% reported overseas travel. The proportion of paratyphoid, typhoid, shigellosis, *Salmonella* Enteritidis, hepatitis A and giardiasis notifications recording overseas travel was significantly higher than the overall enteric disease proportion of 11.5% (95% CI: 10.9-12.2).

Number and proportion of notified cases recording overseas travel, 2002

Disease	Overseas travel	No overseas travel	Unknown travel status	Proportion[1] overseas
Cholera	1	0	0	100%
Ross river virus	1	0	0	100%
Dengue fever	66	0	4	100%
Malaria	47	5	8	90.4%
Paratyphoid	14	6	4	70.0%
Typhoid	12	6	5	66.7%
Shigellosis	41	42	29	49.4%
Salmonella Enteritidis	44	61	34	41.9%
Hepatitis A	28	51	29	35.4%
Giardiasis	133	664	751	16.7%
Salmonellosis (all non-typhoidal types)	191	1141	538	14.3%

[1]"Proportion" refers to the percentage of cases who answered "yes" out of the total number of cases for whom travel information was recorded.

The number of cases recording overseas travel during the incubation period is tabulated below for key travel-associated diseases. Cases recorded as recent visitors, migrants or refugees to New Zealand are excluded from these totals (where known), so that the rate of disease among New Zealanders travelling overseas may be estimated from Statistics New Zealand's estimates of New Zealand short-term departures by country of main destination for 2002.

Estimated number and rate of disease (per 100 000 visits) in New Zealanders travelling overseas by main overseas destination, 2002

Disease	Overseas destination[2]				
	Oceania (n=789 003)*	**Asia** (n=169 949)*	**Europe** (n=107 366)*	**Americas** (n=85 443)*	**Other** (n=141 218)*
	Number (Rate)	**Number (Rate)**	**Number (Rate)**	**Number (Rate)**	**Number (Rate)**
Dengue fever	51 (6.5)	8 (4.7)	0	0	0
Malaria	12 (1.5)	10 (5.9)	0	0	4 (2.8)
Paratyphoid[1]	0	13 (7.6)	0	0	1 (0.7)
Typhoid[1]	2 (0.3)	9 (5.3)	0	0	0
Salmonella Enteritidis[1]	6 (0.8)	34 (20.0)	3 (2.8)	0	0
Shigellosis[1]	10 (1.3)	25 (14.7)	1 (0.9)	4 (4.7)	0
Hepatitis A	14 (1.8)	3(1.8)	3 (2.8)	0	0
Giardiasis[1]	42 (5.3)	57 (33.5)	10 (9.3)	10 (11.7)	6 (4.2)

[1] Recent visitors, migrants and refugees are potentially included in numerator as the reason for travel is not recorded on EpiSurv.
[2] Overseas destinations based on Stats NZ classification: Oceania includes Australia and Pacific Islands; Asia includes South-East Asian countries (eg. Indonesia, East Timor), North -East Asian countires (eg. China, Korea), Southern and Central Asian countries (eg. India); Europe includes North-West, Southern and Eastern Europe; Americas include Northern, Southern and Central America; Other includes Sub-Saharan Africa, North Africa and the Middle East.
* Denominators for rate calculations are based on Stats NZ's estimate of NZ short-term departures by country of main destination in 2002.

Key points

Malaria: High rates of infection occurred among armed forces personnel stationed in East Timor and in travellers to Papua New Guinea and Vanuatu.

Dengue: Among travellers to oceanic countries, the rate of dengue (6.5 per 100 000 visits) was higher than that for all other typically travel-associated notifiable diseases. Rates were particularly high in travellers to the Cook Islands (158.5 per 100 000 visits).

Hepatitis A: Similar rates of infection occurred among travellers to Oceania, Asia and Europe. Rates of infection were elevated in travellers to Samoa (21.0 per 100 000 visits) and Fiji (7.5 per 100 000 visits).

Shigellosis: Assuming notified cases included few migrants, visitors or refugees to New Zealand, the highest rates of infection occurred among travellers to Asia (14.7 cases per 100 000 visits), in particular India (80.0 per 100 000 visits).

Salmonellosis: Rates of typhoid and paratyphoid fever were highest in travellers to Asia (5.3 and 7.6 per 100 000 visits, respectively). Rates of *Salmonella* Enteritidis were significantly higher among travellers to Asia than to other regions, and particularly high among travellers to Indonesia (118.5 per 100 000 visits).

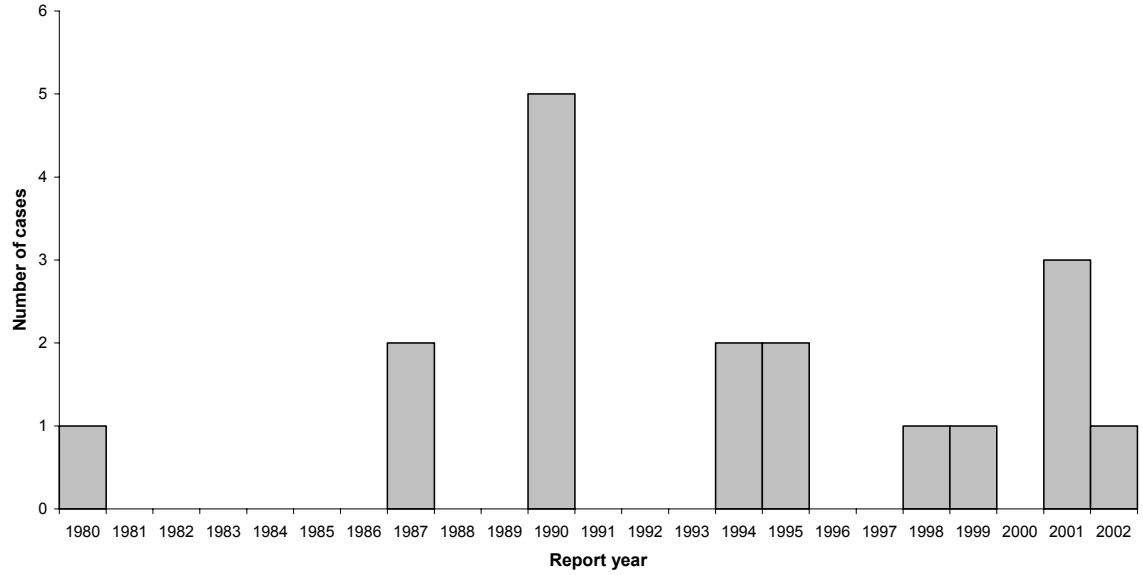
Giardia: The highest overall rates of infection for travellers were reported for giardiasis. This was particularly so among travellers to Asia (33.4 per 100 000 visits) with the highest country rate being for travel to India (205.5 per 100 000 visits).

Cholera

One laboratory-confirmed case of infection with Vibrio cholerae O1, biotype El Tor, subtype Ogawa, was notified from Waitemata District Health Board in April 2002. The case was a 63-year-old female New Zealander who had recently travelled to India and Thailand. She drank iced water in Bangkok, swam in an Indian hotel swimming pool and had contact with cow dung in New Delhi. The case was hospitalised. In comparison, three cases of cholera linked to overseas travel to India, China and Bali, respectively, were notified in 2001.

The following chart shows the number of cholera notifications each year since 1980. The total number of cases notified since 1980 is 18, six of whom have been notified in the past five years.

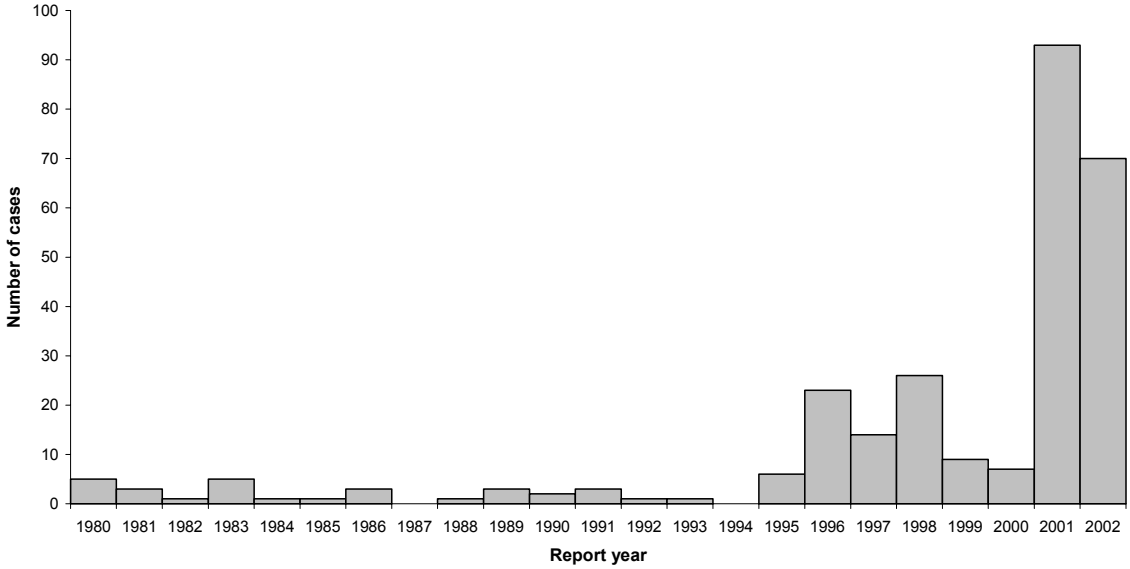
Cholera notifications by year, 1980 - 2002



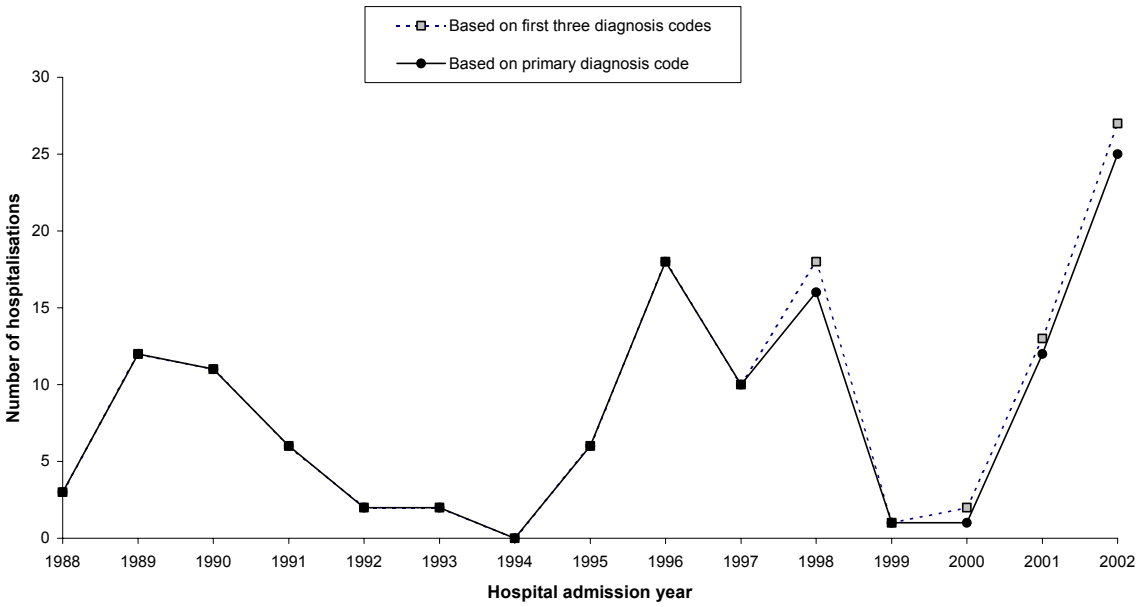
Dengue fever

Seventy cases of dengue fever were notified in 2002, representing a rate of 1.9 per 100 000, compared to a rate of 2.5 the previous year. Of the 63 cases for whom this information was recorded on EpiSurv, 16 (25.4%) were hospitalised. According to hospital discharge data, 25 cases were hospitalised in 2002 for dengue fever. This is more than double the number (12) hospitalised during 2001. The following two graphs show (i) the number of notified cases each year since 1980, and (ii) the number of hospitalisations (ICD9 code 061) each year since 1988. The trends they exhibit are similar, and mirror those seen in Pacific Islands in recent years: namely, an increase in cases during 1996, 1997 and 1998, a lull during 1999 and 2000, followed by a sharp rise in 2001, and elevated incidence during 2002.

Dengue notifications by year, 1980 - 2002



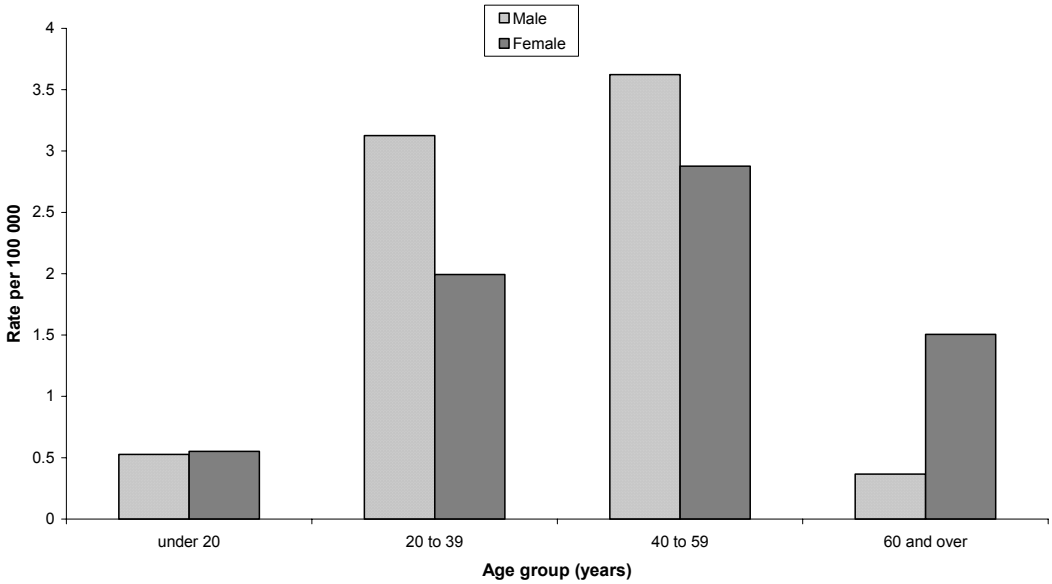
Dengue fever hospitalisations by year, 1988 - 2002



All 66 cases for whom travel information was recorded, reported overseas travel during the incubation period. Implicated overseas destinations included the following: Cook Islands (41 cases), Fiji (5), Thailand (5), Samoa (4), Tahiti (3), Indonesia (2), and one case each from Australia, Solomon Islands, East Timor, Malaysia, and Pakistan. Six dengue fever cases were recent visitors to New Zealand. Excluding these, rates of infection were highest among travellers to the Cook Islands (158.5 per 100 000 visits). In comparison, during 2001, incidence was highest among New Zealanders travelling to Samoa, with 52 cases in this group representing a rate of 349.8 per 100 000 visits.

The following bar chart shows the age and sex-specific notification rate during 2002. Males in the '40 to 59 years' age group experienced the highest rate of 3.6 per 100 000.

Dengue fever notification rates, 2002 by age and sex



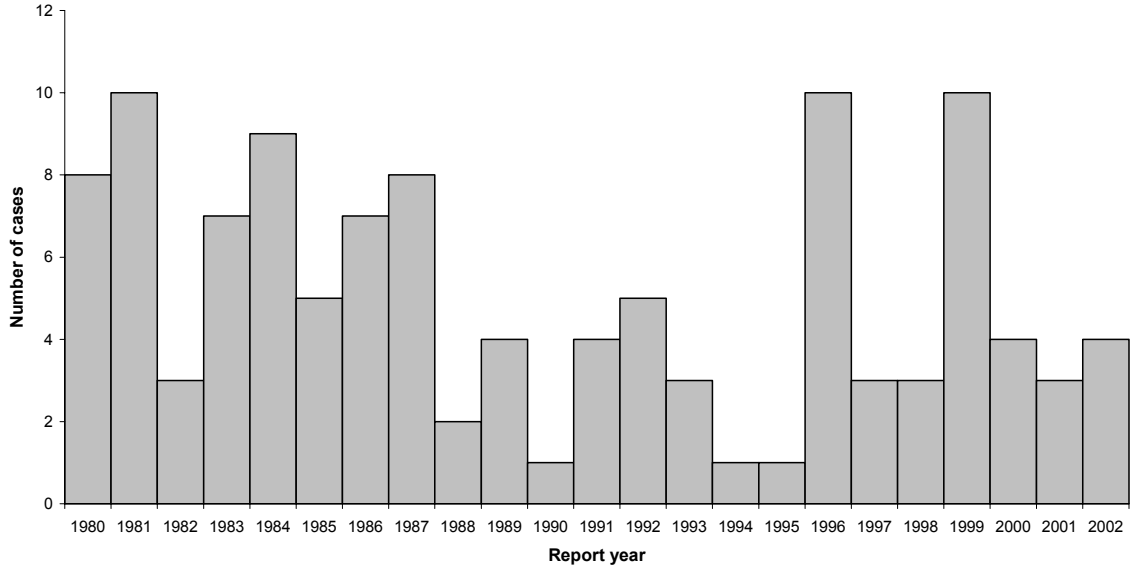
These surveillance data suggest that dengue fever is an increasing risk for New Zealanders travelling to tropical countries. Consequently, there is also an increasing risk that this disease will be introduced into New Zealand. Both of these factors reinforce the need to emphasise personal protection measures for travellers.[68]

Leprosy

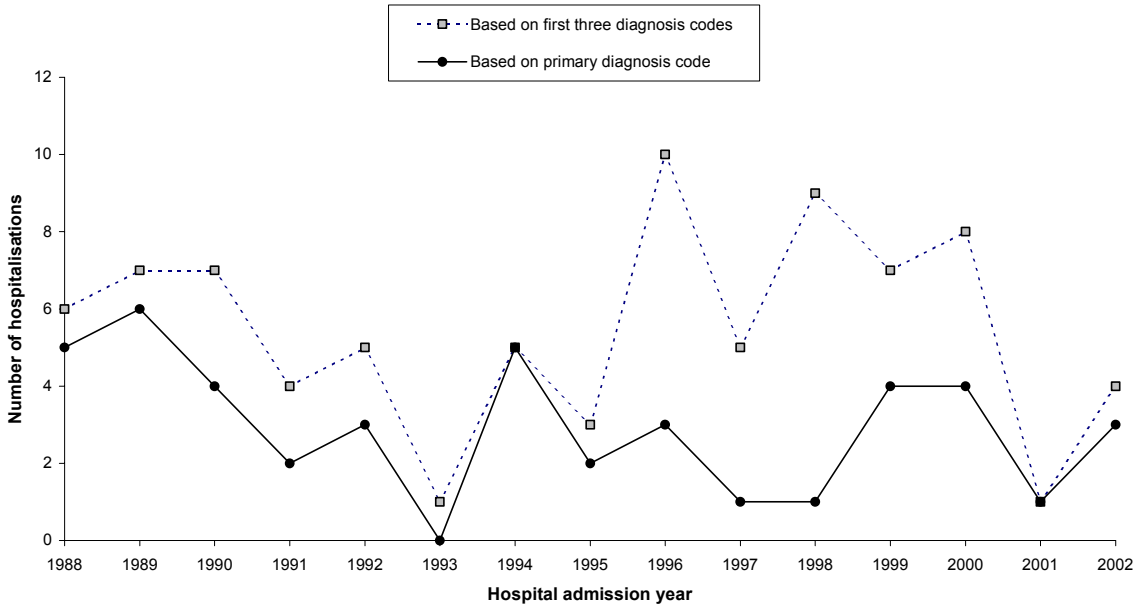
Three cases of leprosy were notified in 2002, two of whom were laboratory confirmed. One case was lepromatous leprosy, one was borderline leprosy and one was of unknown form. The three cases all resided in the Auckland region and were aged between 40 and 50 years. Two female cases were Pacific peoples and one male case was of Ethiopian ethnicity. The majority (24/37 or 64.9%) of cases notified since 1995 have been Pacific peoples, and the remainder (35.1%) have been of 'Other' ethnicity i.e. nine Asians (including five Indians) and four Africans. Over the past eight years the male to female notification ratio was 2.1:1. There have been no notified cases aged under 18 years since 1995.

No hospitalisations among 2002 notifications were recorded on EpiSurv, although according to hospital discharge data hospitalisations due to leprosy totalled three. The following graphs show (i) the number of notified cases each year since 1980, and (ii) the hospitalisations each year since 1988.

Leprosy notifications by year, 1980 - 2002

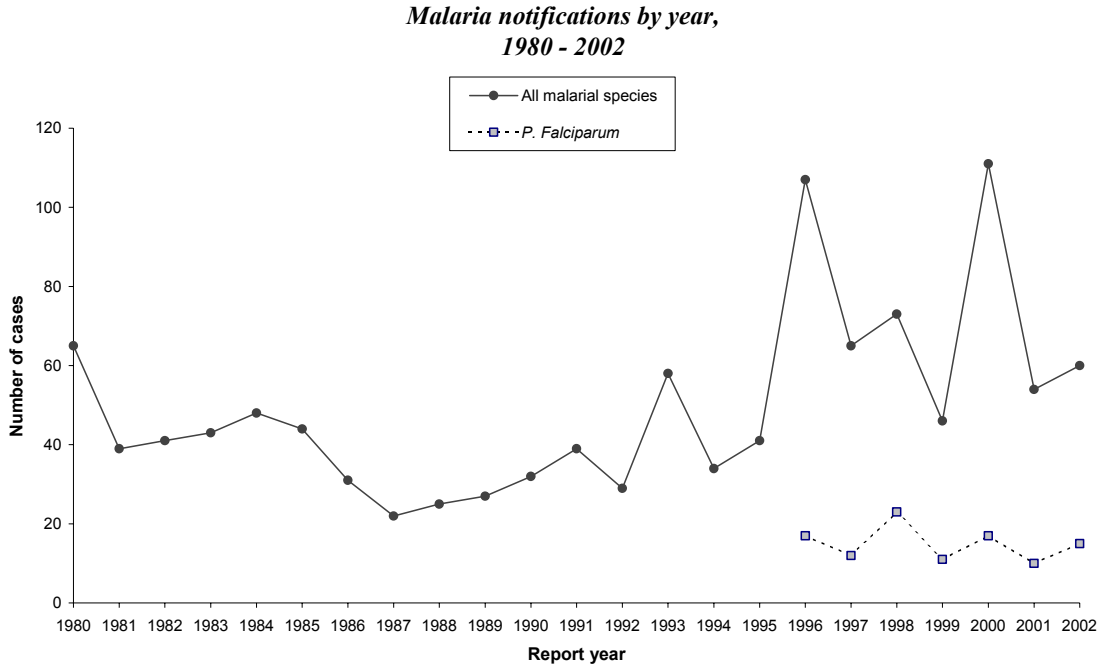


Leprosy hospitalisations by year, 1988 - 2002

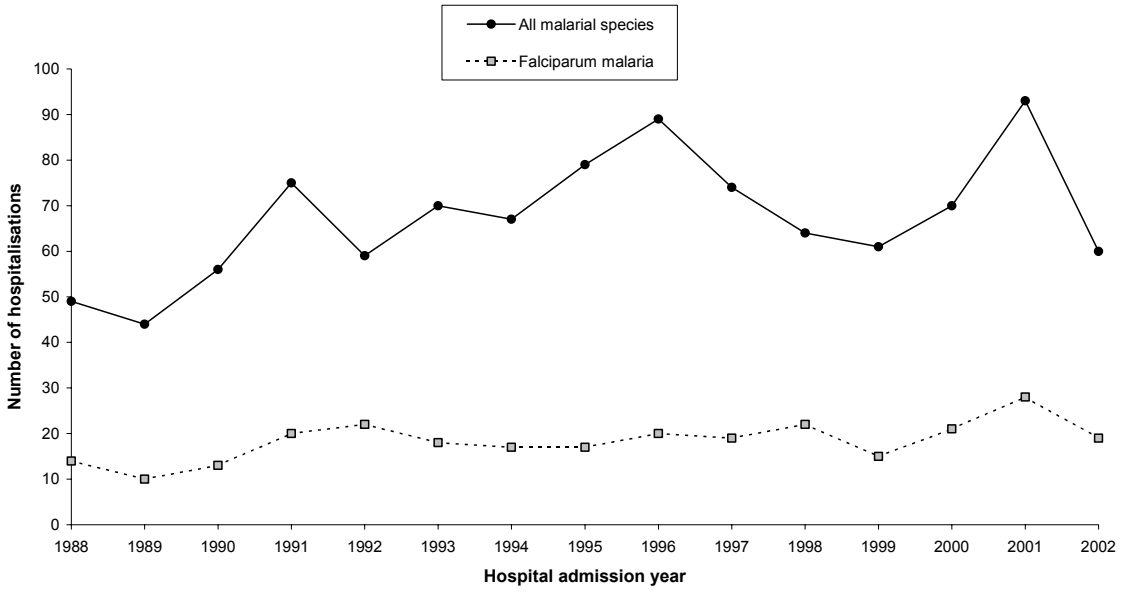


Malaria

A total of 60 cases of malaria was notified in 2002, representing a rate of 1.6 per 100 000. This was a slightly higher than the incidence rate during 2001 (1.4), although significantly lower than the rate in 2000 of 3.0 per 100 000. Of the 49 cases for whom hospitalisation status was recorded on EpiSurv, 32 cases (65.3%) were hospitalised. According to hospital discharge data, malaria hospitalisations (ICD9 code 084) totalled 60 during 2002, and 93 the previous year. The following graphs show (i) the total number of cases of malaria notified each year since 1980, and the number of cases of *P. falciparum* notified since 1995; and (ii) the hospitalisations since 1988.

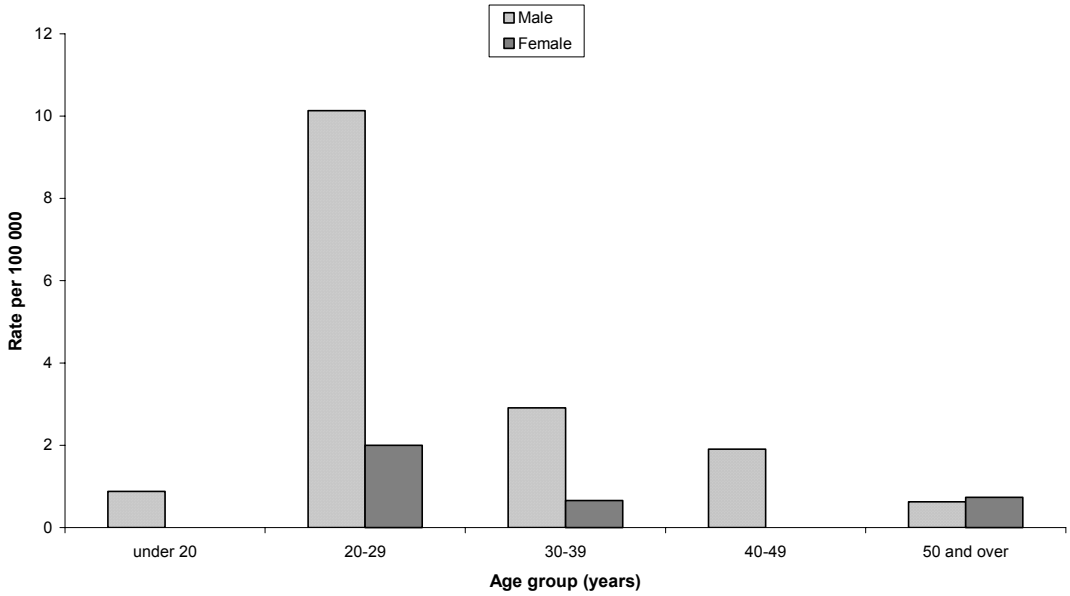


Malaria hospitalisations by year, 1988 - 2002



A total of fifteen cases had travelled overseas due to army deployment. Nine cases recorded travel to East Timor as part of New Zealand's military/ peacekeeping involvement in the region. This is reflected in the age and sex distribution of 2002 notifications (see chart below), in which a rate of 10.1 per 100 000 was experienced by males in the '20 to 29 years' age group, compared to the national rate of 1.6. Since 1998, a total of 76 confirmed cases have occurred among armed forces personnel in East Timor.

Malaria notification rates, 2002 by age and sex



Overseas travel information was recorded for 50 cases, of whom 47 cases (84.9%) had recently been overseas. Implicated overseas destinations were the following: Papua New Guinea (11 cases), Africa (11), East Timor (10), India (7), Vanuatu (3), Solomon Islands (2), Korea (2), Pakistan (2), Fiji (1), and Vietnam (1).

The reason for travel was recorded for 45 cases. A total of 26 (57.8%) cases were New Zealanders travelling overseas, including 15 cases travelling overseas due to military service. There were also eight visitors to New Zealand, eight migrants, and two refugees.

The malarial species was identified in 54 (90%) cases: *P. vivax* was identified in 74% (40/54) of cases and *P. falciparum* in 28% (15/54) of cases. Both *P. vivax* and *P. falciparum* were identified in three cases. *P. ovale* was identified in two cases.

The following table shows the countries where the infection was most likely to have been acquired, by malarial species identified. Among cases for whom travel information was recorded, the majority (20/33 or 60.6%) of *P. vivax* cases had travelled to or from Asian countries, whereas the majority (7/13 or 53.8%) of *P. falciparum* cases were linked to travel to or from African countries. Among the 17 cases recording travel to or from Oceanic countries, 10 (58.8%) were identified as *P. vivax.*

Malarial species and countries/regions where infection probably acquired, 2002 cases

Overseas destination	**Malarial species**			
	P. vivax	***P. falciparum***	***P. ovale***	**Unknown species**
Oceania				
Papua New Guinea	6	2	0	3
Solomon Islands[1]	1	0	0	1
Vanuatu	2	0	1	0
Fiji	1	1	0	0
Asia				
East Timor	9	1	0	0
India	5	2	0	1
Pakistan	2	0	0	0
Korea	2	0	0	0
Vietnam	1	0	0	0
Thailand	1	0	0	0
Africa	3	7	1	0
Unknown travel	7	2	0	1
Total	**40**[*]	**15**[*]	**2**	**6**

* Three cases had both *P. Falcip and P. vivax* identified

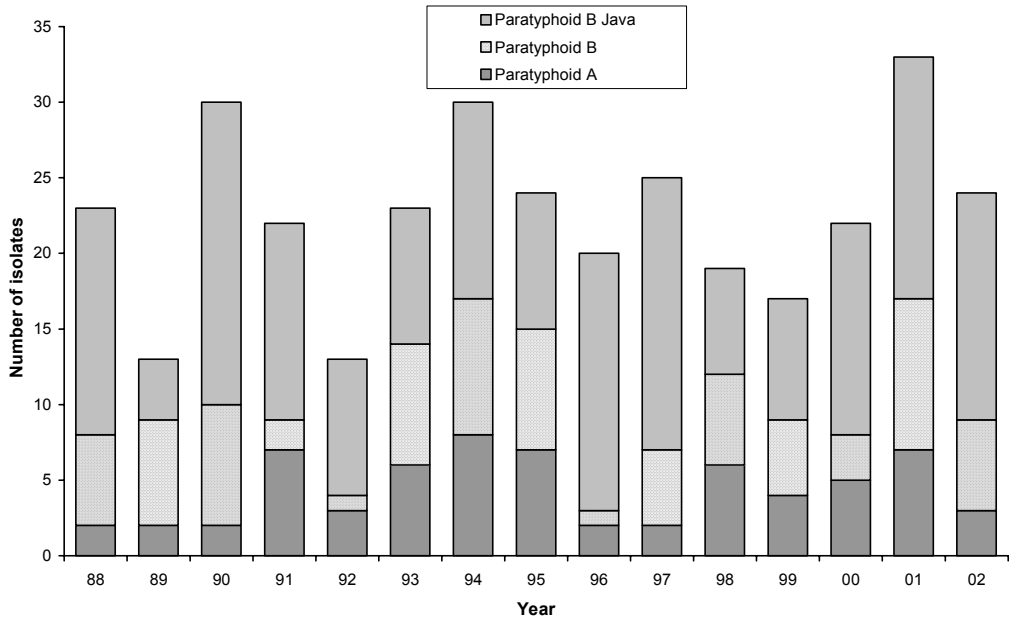
These surveillance data suggest a modest increase in imported malaria in New Zealand over the past decade. Most cases continue to be linked to travel to Asia and those parts of Oceania where malaria is endemic (Papua New Guinea, Solomon Islands, and Vanuatu). The more severe, and potentially fatal, infection with *P. falciparum* continues to be most strongly associated with travel to Africa.

Paratyphoid fever

There were 24 cases of paratyphoid[a] notified in 2002. These were an exact match with the 24 cases confirmed by ESR Enteric Reference Laboratory. In comparison, during 2001 there were 33 laboratory-reported cases and 32 notifications. During 2002, fifteen isolates were identified as *Salmonella* Paratyphi B var Java, six as *S.* Paratyphi B, and three as *S.* Paratyphi A. Of the nineteen cases for whom hospitalisation status was recorded on EpiSurv, 5 (26%) were hospitalised.

The following graph shows the number of *Salmonella* paratyphi isolates by type, each year since 1988.

Salmonella paratyphi isolates, by type and year
1988 - 2002



Of the 20 cases for whom travel information was recorded, 14 (70%) had been overseas during the incubation period. Implicated overseas destinations included Thailand (4 cases), Indonesia (3), Morocco, Taiwan, Korea, Malaysia, Singapore and Bangladesh (1 case each). Rates of infection were highest among travellers to Asian countries, in particular Thailand (29.4 per 100 000 visits).

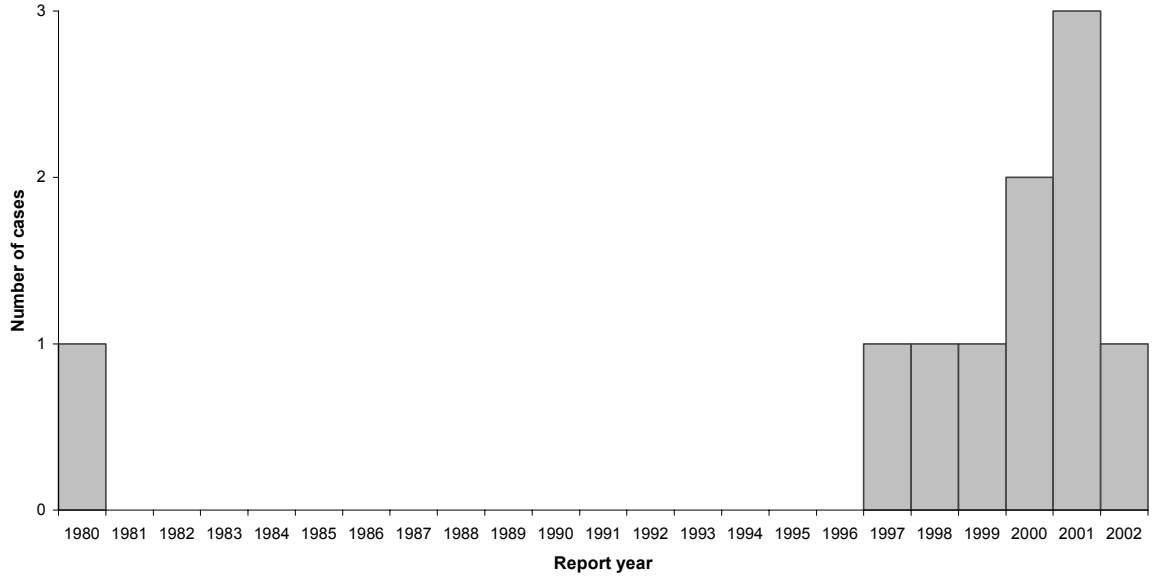
Ethnicity was recorded for 17 cases, of whom 15 (88.2%) were of European ethnicity. The male to female ratio was 1:1. Cases ranged in age from one to 69 years. A total of five (20.8%) cases occurred in children aged between one and four years. Among the six cases recording no overseas travel during the incubation period, one case recorded contact with a confirmed case, one case consumed mussels from an anchor chain in open sea, one case was a pet shop products salesman, and another had handled a turtle.

[a] Includes eight cases who were notified under the 'salmonellosis' disease heading.

Ross River virus

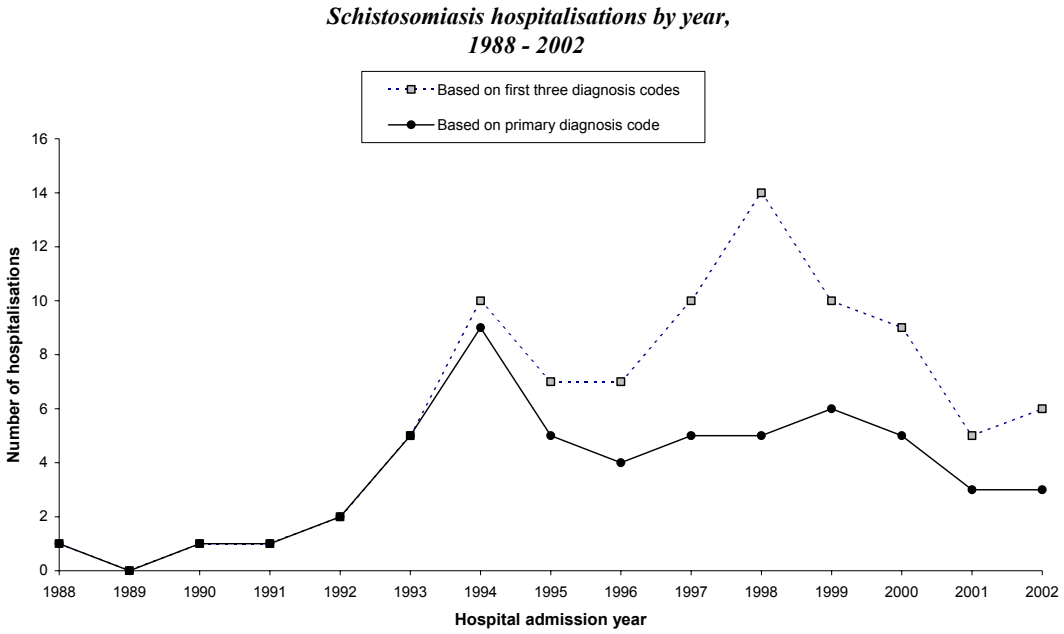
One confirmed case of Ross River virus infection was notified in 2002. The case was a 65-year-old European male who had been in Australia during the incubation period. During the previous year, three imported cases, linked to travel to Australia, Thailand and Fiji, respectively, were notified. The total number of cases reported since the disease became notifiable in the late 1970s is ten. The following chart shows the number of cases of Ross River virus notified each year since 1988.

Ross River virus notifications by year, 1980 - 2002



Schistosomiasis

Between 1960 and 1996, the highest annual schistosomiasis notification total was reported in 1994 (23 cases). Schistosomiasis was removed from the list of notifiable diseases in 1996. Hospital discharge data therefore provide one of the only ongoing forms of surveillance for this disease. These data show three hospitalisations due to schistosomiasis in 2002 and no evidence of an increase in the incidence of this imported disease over the past decade.

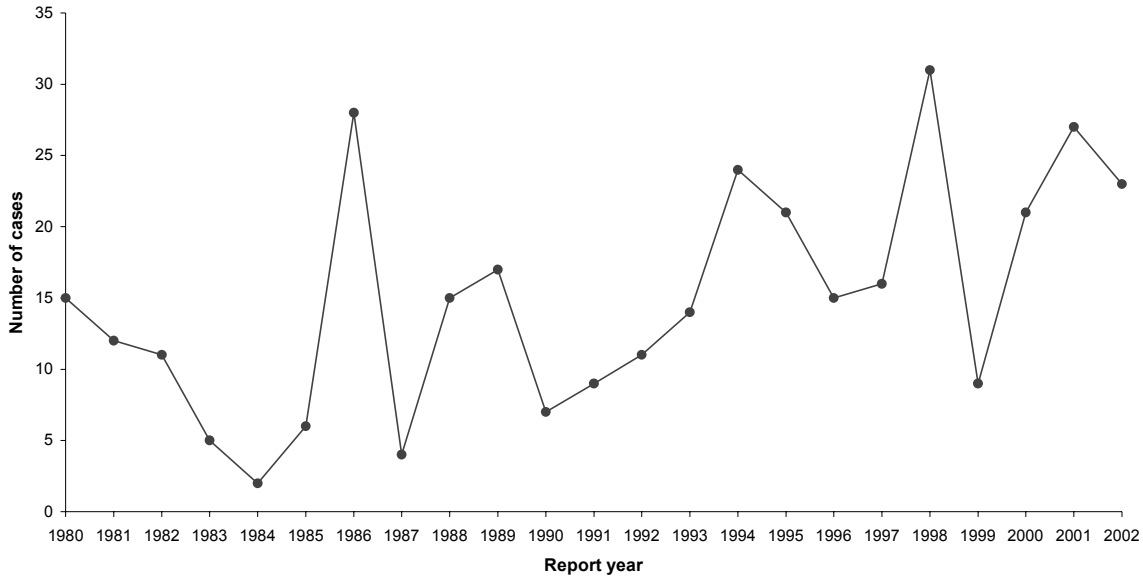


Typhoid

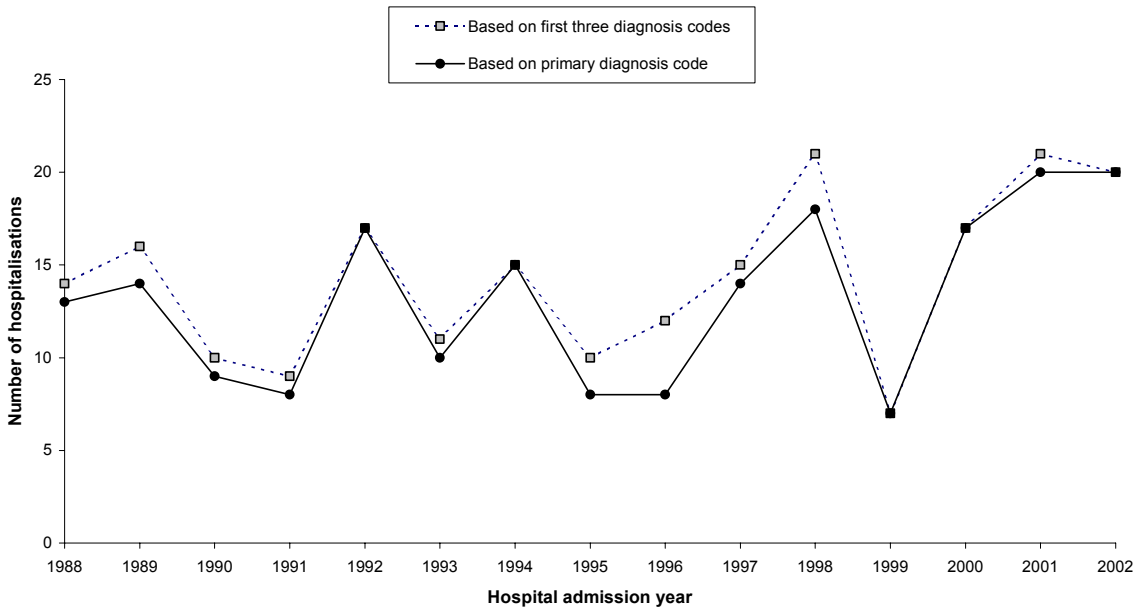
Twenty-three cases of typhoid fever were notified in 2002. There were also 23 isolates of *Salmonella* Typhi received by the ESR Enteric Reference Laboratory. Of these, 22 matched to notifications: one laboratory-reported case was not notified as it did not meet the clinical criteria for a case, and one notified case (the mother of a confirmed case) was not laboratory-confirmed. The typhoid notification rate in 2002 of 0.6 per 100 000 was similar to the previous year's rate of 0.7. Hospitalisation status was recorded for 22 cases, of whom 17 (77.3%) were hospitalised. According to hospital discharge data, the hospitalisations (ICD9 code 002.0) in 2002 totalled 20. Typhoid has had the highest case hospitalisation rate among notifiable enteric diseases since 1996.

The following graphs show (i) the number of cases notified each year since 1980, and (ii) hospitalisations each year since 1988.

Typhoid notifications by year, 1980 - 2002



Typhoid hospitalisations by year, 1988 to 2002



Overseas travel information was recorded for 18 cases, of whom 12 (66.7%) had been overseas during the incubation period. Implicated overseas destinations included Indonesia (3 cases), Malaysia (2), India (2), Samoa (2), Singapore (1) and Pakistan (1). Rates of infection in New Zealanders travelling overseas are hard to estimate, as the reason for travel is not recorded on EpiSurv for typhoid notifications. Of the six cases who had not been overseas during the incubation period, four reported contact with a confirmed case of the disease. Ethnicity was recorded for 21 cases, all of whom were either Pacific peoples (9/21 or 42.9%)

or of ‘Other’ ethnicity (12/21 or 57.1%). Seven cases (33.3%) were of Samoan ethnicity. The proportion of Pacific peoples in 2001 cases was also high (11/27 or 40.7%) and included eight people of Samoan ethnicity.

Cases ranged in age from four to 65 years. Nine (39.1%) cases were aged under 20 years. The male to female ratio was 0.5:1.

Outbreak surveillance

A detailed analysis of the outbreak surveillance data can be found in the Annual Summary of Outbreaks 2002.[9] A total of 333 outbreaks were reported to ESR in 2002, representing a national rate of 8.9 outbreaks per 100 000 population. The outbreaks involved a total of 2870 cases: 990 confirmed (according to the case definition reported for the outbreak) and 1880 probable cases. The average number of cases per outbreak for 2002 was 8.6 compared to 6.0 in 2001. The number of outbreak-associated cases reported during 2002 exceeded that of 2001 (although the total number of outbreaks decreased), and is the highest annual total of outbreak cases since this surveillance began operating in 1996.

Of the 333 outbreaks reported during 2002, a total of 28 outbreaks involved cases that were hospitalised, with 77 cases hospitalised in total. There were two recorded deaths (both due to Norovirus) among outbreak-associated cases. The following table summarises key characteristics of reported outbreaks in 2002 and 2001.

Characteristics of reported outbreaks in 2002 and 2001

	2002		2001	
Characteristics	**Number**	**Rate[1]**	**Number**	**Rate[1]**
Outbreaks	333	8.9	389	10.4
Cases				
Confirmed	990	26.5	1049	28.1
Probable	1880	50.3	1274	34.1
Total	2870	76.8	2323	62.2
Exposed Persons	5275	141.1	6258	167.4
Hospitalized Persons	77	2.1	78	2.1
Deaths	2	0.1	2	0.1

[1] Rate per 100 000 population

The most frequently reported type of outbreak was common source outbreaks (201/333, 60.4%), followed by outbreaks in defined settings (106, 31.8%) and community-wide outbreaks (3, 0.9%). The number of reported 'household' outbreaks decreased from 104 in 2001 to 57 in 2002, whereas the number of institutional outbreaks rose from 33 in 2001 to 49 in 2002. The average number of cases per institutional outbreak in 2002 was 24.6. There were 28 outbreaks in rest homes in 2002 with on average 33 cases per outbreak.

Most outbreaks were at least partly recognised by cases linked to a common source (248 outbreaks, 74.5%), attending a common event (167, 50.2%) and person to person contact with other case(s) (70, 21.0%). A total of 137 outbreaks (41.1%) were attributed at least in part to food-borne transmission. Person to person, water-borne and zoonotic transmission contributed to 114, 24 and 17 outbreaks respectively. The following table shows the *principle* mode of number of transmission for outbreaks reported in 2002.

Principle mode of transmission for outbreaks reported in 2002

Principle mode of transmission	No. of outbreaks	Percent (n=333)	No. of cases	Percent (n=2870)
Foodborne	132	39.6%	677	23.6%
Waterborne	6	1.8%	18	0.6%
Person to person	68	20.4%	1075	37.5%
Environmental	1	0.3%	9	0.3%
Zoonotic	7	2.1%	22	0.8%
Other	0	0.0%	0	0.0%
Unknown	56	16.8%	267	9.3%
Not Specified	8	2.4%	15	0.5%
Multiple modes	55	16.5%	787	27.4%
Total	**333**	100%	**2870**	100%

During 2002, outbreaks were reported from all health districts except Eastern Bay of Plenty (see table below). The Auckland region, incorporating North West, Central and South Auckland districts, reported the most outbreaks (177 outbreaks involving almost one thousand cases), followed by Canterbury (39 outbreaks), Wellington/Hutt (27), Manawatu (16), West Coast (10) and Otago (10) health districts. The crude national outbreak rate was 8.9 per 100 000 population. Several health districts had significantly higher rates, including West Coast, Auckland, Rotorua, and Manawatu.

Number and rate of outbreaks and outbreak-associated cases, by health district, 2002

Health district[1]	No. of outbreaks	Rate per 100 000	No. of cases	Rate per 100 000	Average no. of cases per outbreak
Northland	1	0.7	15	10.7	15.0
Auckland	177	15.1	967	82.4	5.5
Waikato	8	2.6	73	23.7	9.1
Rotorua	7	10.9	20	31.0	2.9
Taupo	1	3.2	3	9.5	3.0
Tauranga	5	3.9	32	24.8	6.4
Gisborne	2	4.6	12	27.3	6.0
Hawkes Bay	4	2.8	24	16.7	6.0
Taranaki	9	8.7	130	126.0	14.4
Manawatu	16	10.9	66	44.8	4.1
Ruapehu	1	7.0	2	14.0	2.0
Wanganui	3	5.1	25	42.8	8.3
Wairarapa	1	2.6	8	20.9	8.0
Wellington	18	7.1	358	141.1	19.9
Hutt	9	6.8	116	88.0	12.9
Nelson-Marlborough	6	4.9	136	111.1	22.7
Canterbury	39	9.7	690	171.8	17.7
South Canterbury	4	5.1	83	106.2	20.8
West Coast	10	33.0	32	105.5	3.2
Otago	10	6.0	46	27.7	4.6
Southland	1	0.9	5	4.6	5.0
New Zealand	**333**	**8.9**	**2870**[2]	**76.8**	**8.6**

[1] Health district assignment is based on the reported Territorial Local Authority (TLA). Where no TLA was recorded (32 outbreaks), health district was assigned according to the PHU where the outbreak was entered into EpiSurv.
[2] Includes 27 cases from one 'nationwide' outbreak.

A specific food type was implicated in 75 outbreaks. The most commonly implicated food type was chicken (26 outbreaks) followed by fish (13 outbreaks). Commercial food operations were the most frequently recorded outbreak setting and were implicated in 133 outbreaks, 66 of which were restaurants or cafés. A total of 110 outbreaks were reported as having occurred in the home. Control measures were recorded for 250 (75.1%) outbreaks in 2002. Of these, 168 (67.2%) were controlled at source - with health and education advice (60.7%) and modification of procedures (54.2%) being the most common control measures applied.

The most commonly implicated pathogen or toxin was Norovirus (76 outbreaks, 22.8%), followed by *Campylobacter* (51 outbreaks, 15.3%) and *Salmonella* (35, 10.5%). Enteric pathogens were identified or suspected in 317 (95.2%) outbreaks. The following table illustrates the distribution of outbreaks by suspected pathogen or toxin. A detailed table showing outbreaks cross-tabulated by region and pathogen/toxin is contained in the appendix.

Number of outbreaks and outbreak-associated cases by suspected pathogen or toxin, 2002

Suspected pathogen or toxin	No. of outbreaks	Percent (n=333)	No. of cases[1]	Percent (n=2870)	Average no. of cases per outbreak	Notified cases[2] part of outbreak No. (%)[3]
Enteric	317	95.2%	2783	97.0%	8.8	800 (4.3%)
Bacillus cereus	4	1.2%	16	0.6%	4.0	-
Campylobacter	51	15.3%	239	8.3%	4.7	75 (0.6%)
Ciguatera poisoning	2	0.6%	9	0.3%	4.5	-
Clostridium perfringens	7	2.1%	127	4.4%	18.1	-
C. perfringens and *B. cereus*	1	0.3%	7	0.2%	7.0	-
Cryptosporidium parvum	15	4.5%	120	4.2%	8.0	111 (11.4%)
Cucurbitacin toxin	1	0.3%	3	0.1%	3.0	-
Escherichia coli O157	1	0.3%	3	0.1%	3.0	3 (4.1%)
Gastroenteritis (not specified)	87	26.1%	317	11.0%	3.6	378 (34.9%)*
Giardia	11	3.3%	68	2.4%	6.2	58 (3.7%)
Hepatitis A virus	4	1.2%	34	1.2%	8.5	30 (27.8%)
Histamine poisoning	3	0.9%	10	0.3%	3.3	-
Norovirus	73	21.9%	1419	49.4%	19.4	-
Norovirus, *B. cereus*	1	0.3%	3	0.1%	3.0	-
Norovirus, Aeromonas caviae	1	0.3%	23	0.8%	23.0	-
Rotavirus	2	0.6%	37	1.3%	18.5	-
Salmonella	35	10.5%	249	8.7%	7.1	164 (8.8%)
Scombroid fish poisoning	2	0.6%	22	0.8%	11.0	-
Shigella	7	2.1%	27	0.9%	3.9	9 (8.0%)
Solanine toxin	1	0.3%	2	0.1%	2.0	-
Staphylococcus aureus	4	1.2%	9	0.3%	2.3	-
Staph, *C. perf.*, Norovirus, *B. cer.*	1	0.3%	29	1.0%	29.0	-
Yersinia	3	0.9%	10	0.3%	3.3	4 (0.8%)
Non enteric	16	4.8%	87	3.0%	5.4	83 (3.0%)
Bordetella pertussis	7	2.1%	32	1.1%	4.6	30 (2.8%)
Hepatitis C virus	1	0.3%	3	0.1%	3.0	0 (0%)
Leptospira Pomona	2	0.6%	5	0.2%	2.5	4 (2.8%)
Mycobacterium tuberculosis	4	1.2%	39	1.4%	9.8	40 (10.4%)
Neisseria meningitidis group C	2	0.6%	8	0.3%	4.0	8 (1.4%)
Total	**333**	100%	**2870**	100%	**8.6**	**883 (4.1%)**

[1] Number of cases includes laboratory-confirmed, other confirmed and probable cases
[2] Cases individually notified on EpiSurv which were recorded as being part of a recognised outbreak
[3] Percentage of the total number of individually notified cases
* Cases individually notified as gastroenteritis include many for whom the causal organism was identified

Identified outbreaks contribute a significant proportion of the burden of infectious diseases in New Zealand. This is particularly the case for enteric diseases, where approximately four percent of cases of notifiable enteric diseases are part of identified outbreaks. (The proportion of notified cases occurring as part of recognised outbreaks is shown in the table above). Much of this burden is preventable, particularly by focusing on food safety, enteric hygiene precautions and hygiene after handling animals.

There are two major limitations of this data. These are that the surveillance system does not record information on all outbreaks occurring in New Zealand, and that recording of information on reported outbreaks is often incomplete or inconsistent. Further work is necessary to improve the coverage of the surveillance system and to standardise information collection and application of case definitions.

Discussion

Key Results

These surveillance data identify many of the same infectious disease problems described in previous annual surveillance reports.[69,70] The incidence of several important communicable diseases is increasing in New Zealand compared to historical levels. Moreover, rates of some diseases are either high compared with other developed countries or failing to decline at an acceptable rate. The following disease events are of particular note:

- The meningococcal disease epidemic continued for its thirteenth year. This is following a typical pattern of group B meningococcal disease epidemics which cause prolonged, moderate increases in disease incidence.
- Pertussis remains poorly controlled with epidemics recurring every 4-5 years and an increasing incidence of disease in the inter-epidemics periods.
- The incidence of tuberculosis and rheumatic fever is failing to decline. Along with meningococcal disease, all are causing disproportionately high rates in Maori and Pacific peoples and are strongly associated with deprivation.
- Enteric disease rates continue to climb. The increase is largely driven by campylobacteriosis, which reached a new high of 12489 cases in 2002 and remains the most frequently notified disease.
- Rates of the bacterial STIs, gonorrhoea, chlamydia and syphilis, continue to increase and remain elevated compared with other developed countries.
- Leptospirosis appears to be re-emerging following a marked decline that resulted from successful control programmes in the 1970s. Sheep may be a more important reservoir of infection than previously thought.
- The first local transmission of brucellosis in New Zealand for at least a decade occurred in 2002. This was a case of *Brucella suis*, probably transmitted from an infected pig.
- Hospitalisations for cellulitis increased markedly during the 1990s, and this group of infections now cause over 6000 hospitalisations a year. This rise has coincided with an increase in methicillin-resistant *Staphylococcus aureus* (MRSA) infection in the communitiy.
- Dengue fever incidence in returning travellers remains high, reinforcing its importance as a hazard for travellers, and the introduction potential within New Zealand.
- Identified outbreaks (333) caused a total of 2870 cases, more than in any year since the outbreak surveillance system was established in 1996. Among these outbreaks was the first large dispersed foodborne outbreak of hepatitis A for several years. There were also 93 Norovirus outbreaks or clusters of laboratory-confirmed Norovirus-associated gastroenteritis cases in 2002, and extensive spread of cases through institutionalised settings.

New Zealand has had some successes in prevention and control of infectious diseases. Historically, well-organized, government-run programs have eliminated or controlled several zoonoses.[71] There have also been successes with control of some vaccine preventable diseases. The continuing decline in the incidence of Hib to a new low of three cases in 2002, (none of whom were children), is one good example.

The importance of infectious diseases was highlighted by a re-analysis of mortality records for the years 1980-1998 and hospital discharges for the period 1988-2000.[72] Following recoding, the proportion of deaths attributable to infectious disease increased from 0.7% to 6.6% of deaths, and hospitalisations from 2.2% to 12.6%. Over the study period infectious disease mortality rates showed little decline, and infectious disease hospitalisations rose by almost 60%.

These data also show that the burden of serious infectious diseases is not spread evenly across the population, with higher rates among Maori and Pacific populations and those living in more deprived neighbourhoods.

Implications

Surveillance data provide support for many of the control priorities already described in the Integrated Approach to Infectious Diseases:[73]

- Reducing socio-economic deprivation and household crowding
- Implementing the meningococcal disease vaccine strategy
- Raising vaccination coverage via the proposed immunisation register and other strategies
- Developing and implementing the sexual health strategy
- Implementing the Hepatitis C action plan.[74]

The data in this report also identify a number of potential research priorities, several of which are already well described:

- Research to identify sources and modes of transmission for enteric disease and to support effective control measures
- Investigation of the rising incidence of leptospirosis
- Operational research to better quantify and evaluate characteristics of the notifiable diseases surveillance system.

Limitations of data

The major limitation of infectious disease surveillance is that not all important infectious diseases, or groups of diseases, are under effective public health surveillance. A number of important diseases are not notifiable under the Health Act 1956. These include most sexually transmitted infections, pneumococcal disease, hospital-acquired, and congenital infections. While some surveillance data on these diseases are available, they remain incomplete.

Even where a disease has been placed under surveillance, the data inevitably have quality limitations. This report attempts, for the first time, to use hospitalisation data for infectious diseases in a systematic way to supplement surveillance data from other sources. This approach also allows some key quality dimensions of the notifiable diseases surveillance system to be assessed, as follows:

- *Sensitivity* – The sensitivity of the meningococcal disease surveillance system appears to be at least 87%. The sensitivity of surveillance for other diseases will almost invariably be less, particularly for the least serious enteric disease where only a small proportion of

those infected will present to the health system. The notifiable disease surveillance system also tends to focus on acute infections and is less effective for surveillance of chronic infections, notably hepatitis B and C and HIV/AIDS, where initial infection is frequently asymptomatic (the same is true for surveillance of lead absorption). More work is needed to identify areas where high sensitivity is critical to the success of infectious disease surveillance.

- *Positive predictive value (PPV)* – Although this has not been measured for any of the infectious diseases under surveillance it will be adequate for most purposes. It may be insufficient for meningococcal disease where surveillance will be used to evaluate the effectiveness of the planned vaccination programme. The PPV of measles surveillance may also need to be enhanced if this surveillance system is going to be able to measure measles elimination in New Zealand. This enhancement could be achieved by requiring laboratory confirmation of all cases.

- *Representativeness* – The analysis contained in this report suggest that for some diseases notification data may be poorly representative of disease epidemiology. For example, hospitalisation data suggest that Maori and Pacific people have higher rates of salmonellosis and pertussis than Europeans, which is opposite to the patterns shown by notification data. Further work could be carried out to quantify some of these biases in existing surveillance systems to see if correction factors can be applied to adjust surveillance data.

- *Completeness of associated data* – This report has also identified gaps in the completeness of associated data that is used to describe important demographic, outcome and risk factor characteristics of cases. More work is needed to define core data sets and how they can be collected more efficiently.

- *Data errors* – There are unquantifiable errors in the data contained in this report. These errors are likely to be greater for rates than for simple counts of cases, since rates depend on both an accurate numerator and an accurate and appropriately chosen denominator. This potential source of error is most apparent in ethnic-specific rates, due to the variability in the recording of ethnicity data. Data errors can also result from inaccurate data entry or incorrect information supplied by the person notifying the case.

Improvements to surveillance

One of the strengths of New Zealand's infectious disease surveillance system is its increasing ability to integrate data from multiple sources for increased sensitivity of surveillance. This strength is best illustrated by meningococcal disease surveillance.

A number of improvements have recently been made or are in the process of being made to New Zealand's infectious disease surveillance system. These include the following:

- Routine integration of EpiSurv notification data with laboratory data sources
- Regular comparison and/or integration of EpiSurv disease data with external data sources such as hospital discharge data.
- Improved geocoding of addresses in EpiSurv.

- The development of an EpiSurv quality assurance and performance measurement programme.

This report has identified a number of areas where New Zealand's infectious disease surveillance system could be improved, including:

- Establishing national surveillance for hospital acquired infections (HAIs)
- Upgrading STI surveillance so that it can provide information on population rates of STI infection and risk factor data to help formulate control polices
- Upgrading surveillance of chronic infections, notably hepatitis B and C (including disease sequelae), to improve sensitivity, particularly in high risk populations
- Greater use of laboratory-based surveillance. An audit carried out in Auckland suggested that such a change could improve public health investigation and control of foodborne disease.[75]
- Establishing processes to regularly review the methods, rationale and priorities for infectious diseases under surveillance. This process is needed so that surveillance remains responsive to emerging threats, such as SARS. It is also needed so that surveillance is responsive to diseases that are becoming vaccine preventable, such as pneumococcal disease.

A major review of the schedule of notifiable diseases is underway[76] and this, together with proposed changes to the Health Act, is likely to have a considerable effect on the future direction of infectious diseases surveillance in New Zealand.

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Appendix: National surveillance data and trends

A. Table of data from multiple surveillance sources

Cases and outbreaks of notifiable diseases during 2002, from multiple surveillance sources

2002 Disease	Total cases notified	Notified cases part of outbreak[1]	Laboratory-reported cases[2,3,4]	Hospitalisations		No. of outbreaks[6]	Outbreak cases[6]
				Hospital data[4,5]	EpiSurv	EpiSurv Outbreak Module	
Brucellosis	2	0	-	1	1	0	0
Campylobacteriosis	12489	75	-	570	515	51	239
Cholera	1	0	1	1	1	0	0
Creutzfeldt-Jakob disease	3	0	-	5	-	0	0
Cryptosporidiosis	974	111	-	35	40	15	120
Dengue fever	70	0	-	25	16	0	0
Diphtheria	1	0	1[10]	1	1	0	0
Gastroenteritis	1084	378	-	47[7]	21	87	317
Giardiasis	1548	58	-	24	28	11	68
Haemophilus influenzae type b	3	0	3	2	3	0	0
Hepatitis A	108	30	-	32	24	4	34
Hepatitis B*	69	0	-	191	11	0	0
Hepatitis C*	52	0	-	379	3	1	3
Hydatid disease	2	0	-	1	1	0	0
Lead absorption	90	0	-	-	3	0	0
Legionellosis	51	0	53	84	27	0	0
Leprosy	3	0	-	3	0	0	0
Leptospirosis	142	4	181	63	55	2	5
Listeriosis	19	0	19	6	17	0	0
Malaria	60	1	-	60	32	0	0
Measles	25	0	6	5	0	0	0
Meningococcal disease	557	8	413[11]	492	531	2	8
Mumps	64	0	18	9	1	0	0
Paratyphoid	24	0	24	1	5	0	0
Pertussis	1071	30	-	133	98	7	32
Rheumatic fever (acute)	87	0	-	145	31	0	0
Rickettsial disease	6	0	-	4	2	0	0
Rubella	33	0	4	4	1	0	0
Salmonellosis	1870	164	2019	146	206	35	249
Shigellosis	112	9	123	22	24	7	27
Tetanus	1	0	-	0	1	0	0
Toxic (shell)fish poisoning	1	0	-	13	0	2[8]	22
Tuberculosis	384	40	271	333	193	4	39
Typhoid	23	1	23	20	17	0	0
VTEC/STEC infection	73	3	66	8[9]	16	1	3
Yersiniosis	476	4	-	14	31	3	10
Total	**21578**	**916**	**-**	**2824**	**1956**	**230**	**1176**

* Only acute cases of this disease are currently notifiable

[1] Number of cases individually notified on EpiSurv who were recorded as part of a recognised outbreak

[2] May represent an undercount of true laboratory-confirmed cases for diseases in which ESR has been unable to collate results from all laboratories nationwide.

[3] Counts based on laboratory specimen date.

[4] A dash indicates the data is either unavailable or unknown

[5] Based on primary ICD9 diagnosis code and hospital admission date. Multiple admissions excluded.

[6] According to outbreaks reported on the EpiSurv Outbreak Module. Counts based on outbreak report date.

[7] Cases of other bacterial food poisoning

[8] Cases of scromboid fish poisoning

[9] E. coli (generally)

[10] Toxigenic strain was isolated from a hip aspirate

[11] This figure represents the total number of laboratory-confirmed cases in 2002

B. Table of disease notifications and rates

Cases and rates of notifiable diseases in New Zealand during 2001 and 2002

Disease	Notifications 2002*			Notifications 2001		
	Cases	Rate	95% CI	Cases	Rate	95% CI
AIDS	17	0.5	[0.3, 0.7]	26	0.7	[0.5, 1.0]
Brucellosis	2	0.1	[0.0, 0.2]	0	0.0	[0.0, 0.1]
Campylobacteriosis	12489	334.2	[328.3, 340.0]	10145	271.5	[266.2, 276.7]
Cholera	1	0.0	[0, 0.1]	3	0.1	[0.0, 0.2]
Creutzfeldt-Jakob disease	3	0.1	[0, 0.2]	1	0.0	[0.0, 0.1]
Cryptosporidiosis	974	26.1	[24.4, 27.7]	1208	32.3	[30.5, 34.1]
Dengue fever	70	1.9	[1.5, 2.4]	93	2.5	[2.0, 3.0]
Diphtheria	1	0.0	[0.0, 0.1]	0	0.0	[0.0, 0.1]
Gastroenteritis[1]	1084	29.0	[27.3, 30.7]	940	25.2	[23.5, 26.8]
Giardiasis	1548	41.4	[39.4, 43.5]	1603	42.9	[40.8, 45]
Hib	3	0.0	[0.0, 0.2]	11	0.3	[0.1, 0.5]
Hepatitis A	108	2.9	[2.3, 3.4]	61	1.6	[1.2, 2.1]
Hepatitis B[2]	69	1.8	[1.4, 2.3]	56	1.5	[1.1, 1.9]
Hepatitis C[2]	52	1.4	[1.0, 1.8]	59	1.6	[1.2, 2.0]
Hydatid disease	2	0.1	[0.0, 0.2]	7	0.2	[0.1, 0.4]
Lead absorption	90	2.4	[1.9, 3.0]	130	3.5	[2.9, 4.1]
Legionellosis	51	1.4	[1.0, 1.8]	46	1.2	[0.9, 1.6]
Leprosy	3	0.1	[0.0, 0.2]	3	0.1	[0.0, 0.2]
Leptospirosis	142	3.8	[3.2, 4.4]	105	2.8	[2.3, 3.3]
Listeriosis	19	0.5	[0.3, 0.5]	18	0.5	[0.3, 0.8]
Malaria	60	1.6	[1.2, 2.1]	54	1.4	[1.1, 1.9]
Measles	25	0.7	[0.4, 1.0]	83	2.2	[1.8, 2.8]
Meningococcal disease	557	14.9	[13.7, 16.1]	650	17.4	[16.1, 18.7]
Mumps	64	1.7	[1.3, 2.2]	56	1.5	[1.1, 1.9]
Paratyphoid	24	0.6	[0.4, 1.0]	32	0.9	[0.6, 1.2]
Pertussis	1071	28.7	[26.9, 30.4]	1334	35.7	[33.8, 37.6]
Rheumatic fever (acute)	87	2.3	[1.9, 2.9]	114	3.1	[2.5, 3.6]
Rickettsial disease	6	0.2	[0.1, 0.3]	5	0.1	[0.0, 0.3]
Ross River virus	1	0.0	[0.0, 0.1]	3	0.1	[0.0, 0.2]
Rubella	33	0.9	[0.6, 1.2]	30	0.8	[0.5, 1.1]
Salmonellosis	1870	50.0	[47.8, 52.3]	2417	64.7	[62.1, 67.3]
Shigellosis	112	3.0	[2.4, 3.6]	157	4.2	[3.5, 4.9]
Tetanus	1	0.0	[0.0, 0.1]	4	0.1	[0.0, 0.3]
Toxic (shell)fish poisoning	1	0.0	[0.0, 0.1]	2	0.1	[0.0, 0.2]
Tuberculosis	384	10.3	[9.2, 11.3]	373	10.0	[9.0, 11.0]
Typhoid	23	0.6	[0.4, 0.9]	27	0.7	[0.5, 1.1]
VTEC/STEC infection	73	2.0	[1.5, 2.5]	76	2.0	[1.6, 2.5]
Yersiniosis	476	12.7	[11.6, 13.9]	429	11.5	[10.4, 12.6]

***** No cases of the following notifiable diseases were reported in 2002: anthrax, botulism, plague, poliomyelitis, rabies, cysticercosis, trichinosis, primary amoebic meningoencephalitis

[1] Cases of gastroenteritis from a common source or foodborne intoxication eg. staphylococcal intoxication

[2] Only acute cases of this disease are currently notifiable

C. International rates

Incidence rates of notifiable diseases in New Zealand during 2002, compared with rates in other developed countries

Disease	Country					
	New Zealand (2002)	Australia[a,b] (2002)[c]	USA[d,e] (2000)	Canada[f,g] (2000)	England and Wales[h,i] (2001)	Scotland[j,k] (2002)
AIDS[c]	0.5	0.8	14.5	1.3	-	-
Campylobacteriosis	334.2	78.1	-	40.0	-	-
Cholera	0.0	0.0	0.0	0	0.1	0.1
Cryptosporidiosis	26.1	17.5	1.1	2.0	-	-
Dengue fever	1.9	1.1	-	-	-	-
Giardiasis	41.4	-	-	16.3	-	-
H. influenzae type b disease	0.1	0.2	0.5	0.1	-	-
Hepatitis A	2.9	2.1	4.8	1.6	2.2	-
Hepatitis B	1.8	2.3	2.9	3.2	2.0	-
Hepatitis C	1.4	2.4	1.1	61.1	2.0	-
Legionellosis	1.4	1.5	0.4	0.2	-	0.3
Leptospirosis	3.8	0.9	-	-	0.0	-
Listeriosis	0.5	0.3	0.3	-	-	-
Malaria	1.6	2.6	0.6	1.4	2.1	0.6
Measles	0.7	0.1	0.0	0.6	4.3	6.4
Meningococcal disease	14.9	3.6	0.8	0.8	2.0	4.9
Mumps	1.7	0.4	0.1	0.3	5.3	3.1
Paratyphoid	0.6	-	-	-	0.2	-
Pertussis	28.7	31.6	2.8	16.1	1.7	1.9
Ross River virus infection	0.0	7.6	-	-	-	-
Rubella	0.9	1.3	0.1	0.1	2.8	4.7
Salmonellosis	50.0	42.1	14.1	18.4	-	-
Shigellosis	3.0	2.7	8.1	3.7	-	-
Tetanus	0.0	0.0	0.0	0	0.0	-
Tuberculosis	10.3	5.7	5.8	5.5	12.9	8.4
Typhoid	0.6	0.4	-	0.3	0.2	-
VTEC/STEC infection	2.0	0.3	-	8.8	-	-

-: Disease not notifiable or information not available.

a: (Australian) National Notifiable Diseases Surveillance System, personal communication. (Communicable Diseases Network Australia). Web:http://www.health.gov.au/pubhlth/cdi/nndss/nndss2.htm

b: Population figure (18 972 350) 2001 census. Australian Bureau of Statistics. Web: http://www.abs.gov.au/

c: Australian Aids data (only) is for the calendar year 2001 and is from the (Australian) National Centre in HIV Epidemiology and Clinical Research reported by the (Australian) Communicable Diseases Network.

d: Centers for Disease Control and Prevention (CDC). Summary of notifiable diseases, United States, 2000. MMWR 2000; Pages 84-85 via web as at 28/04/03: http://www.cdc.gov/mmwr/PDF/wk/mm4953.pdf

e: Population figure (281 421 906) census 2000. United States Census Bureau. Web:http://www.census.gov.main/www/cen2000.html

f: Health Canada. "Notifiable Diseases Annual Summary". Canada Communicable Disease Report 2003;29S2:1-149 (published report).

g: Population estimate (30 769 669) provided by Statistics Canada (Annual Demographic Statistics 2000, catalogue no. 91-213) quoted in Health Canada publication quoted above.

h: "NOIDS Report: 2001 Statutory Notifications of Infectious Diseases in England and Wales...". Via web as at 29/01/03: http://www.phls.org.uk/topics-az/noids/annual.htm

i: Population figure 52 041 916 (England 49 138 831, Wales 2 903 085) census 2001, National Statistics (UK) Via Web as at 29/01/03: http://www.statistics.gov.uk/census2001/press_release_uk.asp

j: Scottish Centre for Infection and Environmental Health (SCIEH). SCIEH Weekly Report Volume 37 2003:/01 7 January 2003 page 4 (provisional data missing four days of the year). (Published report).

k: Population figure (5 062 011) census 2001, National Statistics (UK) details above.

Note that data presented as international comparisons should be interpreted with caution due to differences in the surveillance systems from which the figures are obtained. Differences in data collection methods (e.g. through laboratory diagnosis and/or doctor notification) and data collected according to different case definitions (especially those diseases involving both acute and chronic cases such as hepatitis C), will result in rates of disease that are not necessarily comparable.

D. Table of fatalities

Fatal cases of notifiable diseases in 2001 and 2002, according to EpiSurv

Disease	2001			2002		
	No. of fatal cases	**Total notified cases**	**Case-fatality rate**	**No. of fatal cases**	**Total notified cases**	**Case-fatality rate**
AIDS	12	26	-	5	17	-
Campylobacter	1	10145	0.01%	1	12489	0.008%
Gastroenteritis	0	940	0%	1	1084	0.09%
Creutzfeldt Jakob disease	1	1	100%	3	3	100%
Haemophilus influenzae b	1	11	9.1%	1	3	33.3%
Hepatitis B	1	56	1.8%	0	69	0%
Legionellosis	2	46	4.3%	3[2]	51	5.9%
Listeriosis	2[1]	18	11.1%	3[3]	19	15.8%
Meningococcal disease	26	650	4.0%	18	557	3.2%
Pertussis	1	1334	0.07%	1	1071	0.09%
Salmonellosis	2	2417	0.1%	1	1870	0.05%
Tetanus	1	4	25%	0	1	0%
Tuberculosis disease	2	373	0.5%	6	384	1.6%
Total	**52**	**-**	**-**	**43**	**-**	**-**

[1] There was one fatality from perinatal listeriosis and one from non-perinatal listeriosis.
[2] One further death occurred in a laboratory-reported but non-notified case.
[3] All three fatalities occurred in perinatal cases.

E. Table of notifiable diseases by year

Note: cell is blank if data unavailable

Disease	Source	1980	1981	1982	1983	1984	1985	1986	1987	1988	1989	1990	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000	2001	2002
AIDS	Notification				0	3	11	19	28	38	59	73	78	50	70	44	50	76	43	29	33	27	26	17
Campylobacteriosis	Notification	271	442	769	1251	1915	2390	2786	2921	2796	4187	3850	4148	5144	8101	7714	7442	7635	8924	11575	8161	8418	10145	12489
Cholera	Notification	1	0	0	0	0	0	0	2	0	0	5	0	0	0	2	2	0	0	1	1	0	3	1
Creutzfeldt-Jakob disease	Notification																	2	1	0	2	3	1	3
Cryptosporidiosis	Notification																	119	357	866	977	774	1208	974
Dengue fever	Notification	5	3	1	5	1	1	3	0	1	3	2	3	1	1	0	6	23	14	26	9	7	93	70
Gastroenteritis	Notification																	555	308	490	600	725	940	1084
Giardiasis	Notification																	1235	2127	2183	1793	1684	1603	1548
H. influenzae serotype b	Laboratory								93	107	121	143	148	166	118	75	14	24	8	10	9	10	8	3
	Notification																	26	9	11	10	13	11	3
Hepatitis A	Notification	1649	1145	750	674	539	380	251	158	176	134	150	224	288	257	179	338	311	347	145	119	107	61	108
Hepatitis B	Notification	393	377	477	571	609	530	488	474	370	309	242	227	221	145	133	125	104	138	88	94	79	56	69
Hepatitis C (formerly non A or B)	Notification	5	3	7	20	29	31	17	18	20	13	11	25	89	91	79	88	59	92	102	96	80	59	52
Hydatid disease	Notification	7	7	11	9	6	4	5	2	2	0	4	0	4	4	1	5	3	2	2	8	3	7	2
Influenza	Sentinel isolates	33	12	18	30	9	6	8	18	136	119	343	183	317	423	441	521	673	743	127	425	73	313	241
Legionellosis	Notification	5	3	9	25	48	87	95	91	62	17	20	14	11	24	66	33	36	63	43	51	61	46	51
	Laboratory											21	42	60	76	121	76	60	109	107	65	56	56	53
Leprosy	Notification	8	10	3	7	9	5	7	8	2	4	1	4	5	3	1	1	10	3	3	10	4	3	3
Leptospirosis	Notification	582	325	179	169	201	174	139	129	99	90	117	106	70	116	70	65	56	52	75	59	98	105	142
	Laboratory									192	182	229	176	218	234	168	183	140	84	117	76	114	113	181
Listeriosis	Notification	32	18	15	5	6	6	6	12	7	10	16	26	16	11	8	13	10	35	17	19	22	18	19
Malaria	Notification	65	39	41	43	48	44	31	22	25	27	32	39	29	58	34	41	107	65	73	46	111	54	60
Measles	Notification																	68	1984	164	107	64	83	25
	Laboratory	8	7	1	4	11	145	135	26	5	5	7	355	53	4	4	15	25	1220	35	2	9	21	6
Meningococcal disease	Notification	26	25	15	38	34	107	190	179	83	49	53	71	153	202	208	394	473	613	440	505	480	650	557
Mumps	Notification																	76	90	85	56	50	56	64
	Laboratory	7	5	22	2	0	61	132	28	5	105	26	23	10	25	245	66	20	14	8	5	2	22	18
Paratyphoid	Laboratory									23	13	30	22	13	23	30	24	20	25	19	17	23	33	24
Petussis	Notification																	1022	284	153	1046	4140	1334	1071
Rheumatic fever (initial attack)	Notification							12	215	153	148	90	97	70	81	98	88	110	95	65	71	136	114	87
Rubella	Notification																	306	80	53	35	26	30	33
	Laboratory	134	44	12	62	155	120	30	50	95	114	168	81	27	244	104	1581	339	21	2	0	0	3	4
Salmonellosis	Notification	799	845	1261	995	1138	1234	1335	1140	1128	1860	1619	1244	1239	1340	1522	1334	1141	1177	2069	2077	1796	2417	1870
Shigellosis	Notification	228	173	215	173	127	192	189	143	145	137	197	152	124	128	185	191	167	117	122	147	115	157	112
Tetanus	Notification	2	4	5	5	7	3	3	4	1	0	0	0	8	2	2	2	3	0	2	6	1	4	1
Tuberculosis	Notification	474	448	437	415	404	359	320	296	295	303	348	335	327	323	352	391	352	321	365	446	354	373	384
Typhoid	Notification	15	12	11	5	2	6	28	4	15	17	7	9	11	14	24	21	15	16	31	9	21	27	23
VTEC/STEC infection	Notification														3	3	6	7	12	48	64	67	76	73
Yersiniosis	Notification																	330	488	546	503	396	429	476

F. Table of disease notifications and rates by District Health Board, 2002

Note: Blank cell denotes a value of zero

District Health Board	Northland	Waitemata	Auckland	Counties Manukau	Waikato	Lakes	Bay of Plenty	Tairawhiti	Taranaki	Hawke's Bay	Wanganui	Midcentral	Hutt	Capital and Coast	Wairarapa	Nelson Marlborough	West Coast	Canterbury	South Canterbury	Otago	Southland
Campylo-bacteriosis	292	1799	1567	1196	1104	270	378	89	307	439	161	284	442	1088	75	214	84	1510	223	626	334
	208.4	418.6	426.1	318.5	347.4	281.3	212.2	202.4	298.0	305.8	253.2	183.2	335.2	442.5	196.3	174.7	277.3	353.6	422.5	366.6	323.1
Crypto-sporidiosis	8	41	26	26	127	32	21	5	39	61	20	65	27	150	8	24	17	107	40	83	46
	5.7	9.5	7.1	6.9	40.0	33.3	11.8	11.4	37.9	42.5	31.4	41.9	20.5	61.0	20.9	19.6	56.1	25.1	75.8	48.6	44.5
Dengue fever		6	18	12	4	2	3		2		1	3	4	3	1			10		1	
		1.4	4.9	3.2	1.3	2.1	1.7		1.9		1.6	1.9	3.0	1.2	2.6			2.3		0.6	
Gastroenteritis	11	102	101	47	33	11	5	15	11	7	19	34	49	84	10	16	5	364	86	54	19
	7.9	23.7	27.5	12.5	10.4	11.5	2.8	34.1	10.7	4.9	29.9	21.9	37.2	34.2	26.2	13.1	16.5	85.2	162.9	31.6	18.4
Giardiasis	30	188	236	135	164	35	61	8	15	127	28	46	71	129	8	31	20	130	16	54	16
	21.4	43.7	64.2	35.9	51.6	36.5	34.2	18.2	14.6	88.5	44.0	29.7	53.8	52.5	20.9	25.3	66.0	30.4	30.3	31.6	15.5
Hepatitis A	2	17	29	20	17	2	1			5		2	5	2		2		2			1
	1.4	4.0	7.9	5.3	5.4	2.1	0.6			3.5		1.3	3.8	0.8		1.6		0.5			1.0
Hepatitis B	4	8	10	6	7	2	4	6	1	2	2	1	1	3	1	1		8		1	
	2.9	1.9	2.7	1.6	2.2	2.1	2.2	13.6	1.0	1.4	3.1	0.6	0.8	1.2	2.6	0.8		1.9		0.6	
Hepatitis C		3	2	3	1	5	16			2		1		9	1		3	4	2		
		0.7	0.5	0.8	0.3	5.2	9.0			1.4		0.6		3.7	2.6		9.9	0.9	3.8		
Lead Absorption	5	7	7	2	11		2	4	5	3	2	6	1	2	1	1		15	4	9	3
	3.6	1.6	1.9	0.5	3.5		1.1	9.1	4.9	2.1	3.1	3.9	0.8	0.8	2.6	0.8		3.5	7.6	5.3	2.9
Legionellosis	3	3	4	3	3	1	2		2	4	1		4	5	2	2		9	1	2	
	2.1	0.7	1.1	0.8	0.9	1	1.1		1.9	2.8	1.6		3	2.0	5.2	1.6		2.1	1.9	1.2	
Leptospirosis	9	6		2	22		8	7	6	28	4	12		1	2	12	2	6	7	6	2
	6.4	1.4		0.5	6.9		4.5	15.9	5.8	19.5	6.3	7.7		0.4	5.2	9.8	6.6	1.4	13.3	3.5	1.9
Listeriosis		3	3	2	1		2					1	1					4			1
		0.7	0.8	0.5	0.3		1.1					0.6	0.8					0.9			1.0
Malaria		6	3	4	7	3	2		1	1	2	10	1	5		2		7	2	3	1
		1.4	0.8	1.1	2.2	3.1	1.1		1.0	0.7	3.1	6.5	0.8	2.0		1.6		1.6	3.8	1.8	1.0
Measles		2	1	1	2		1							1		6	5	6			
		0.5	0.3	0.3	0.6		0.6							0.4		4.9	16.5	1.4			
Meningococcal disease	29	35	45	87	37	53	51	6	9	31	6	11	12	28	3	3	9	33	6	49	14
	20.7	8.1	12.2	23.2	11.6	55.2	28.6	13.6	8.7	21.6	9.4	7.1	9.1	11.4	7.9	2.4	29.7	7.7	11.4	28.7	13.5
Mumps	3	9	6	7	1	2	2			4	1		1	3		6	1	5	1	9	3
	2.1	2.1	1.6	1.9	0.3	2.1	1.1			2.8	1.6		0.8	1.2		4.9	3.3	1.2	1.9	5.3	2.9
Paratyphoid	4	5	2	2	2	1				2				2		2			1		1
	2.9	1.2	0.5	0.5	0.6	1.0				1.4				0.8		1.6			1.9		1.0
Pertussis	8	96	29	33	89	7	13	1	21	17	35	32	57	62	6	84	79	215	149	7	31
	5.7	22.3	7.9	8.8	28	7.3	7.3	2.3	20.4	11.8	55	20.6	43.2	25.2	15.7	68.6	260.8	50.3	282.3	4.1	30
Rhuematic fever	8	4	15	31	5	1	7	2	1	3	1		1	5	1			1			
	5.7	0.9	4.1	8.3	1.6	1.0	3.9	4.5	1.0	2.1	1.6		0.8	2.0	2.6			0.2			
Rubella	3		2	1	1		3		1	11			1	2		3	2	2		1	
	2.1		0.5	0.3	0.3		1.7		1.0	7.7			0.8	0.8		2.4	6.6	0.5		0.6	
Salmonellosis	48	173	172	151	139	57	60	40	48	86	38	58	48	143	23	138	16	215	43	98	73
	34.3	40.3	46.8	40.2	43.7	59.4	33.7	91.0	46.6	59.9	59.8	37.4	36.4	58.2	60.2	112.7	52.8	50.3	81.5	57.4	70.6
Shigellosis	2	14	26	23	4	3	1		2	7	1		1	4		1		13	4	5	1
	1.4	3.3	7.1	6.1	1.3	3.1	0.6		1.9	4.9	1.6		0.8	1.6		0.8		3.0	7.6	2.9	1.0
Tuberculosis	12	66	78	61	24	7	9	2	2	30	2	6	18	28	5	2		21	1	8	2
	8.6	15.4	21.2	16.2	7.6	7.3	5.1	4.5	1.9	20.9	3.1	3.9	13.7	11.4	13.1	1.6		4.9	1.9	4.7	1.9
Typhoid		2	3	4									13	14				18			
		1.4	0.5	2.7									0.8	1.2				0.2			
VTEC/STEC	1	3	5	3	15	4	6		5	2	1	1	1	4				18	1	1	2
	0.7	0.7	1.4	0.8	4.7	4.2	3.4		4.9	1.4	1.6	0.6	0.8	1.6				4.2	1.9	0.6	1.9
Yersiniosis	5	70	66	45	36	12	24	3	3	16	6	12	10	38	4	4	20	65	10	18	9
	3.6	16.3	17.9	12.0	11.3	12.5	13.5	6.8	2.9	11.1	9.4	7.7	7.6	15.5	10.5	3.3	66.0	15.2	18.9	10.5	8.7

Key: Cases
Rate per 100 000

G. Table of disease notifications and rates by health district, 2002

Note: Blank cell denotes a value of zero

Health District	Northland	North West Auckland	Central Auckland	South Auckland	Waikato	Eastern Bay of Plenty	Tauranga	Rotorua	Gisborne	Taupo	Hawke's Bay	Ruapehu	Taranaki	Wanganui	Manawatu	Wairarapa	Wellington	Hutt	Nelson-Marlborough	West Coast	Canterbury	South Canterbury	Otago	Southland
Campylo-bacteriosis	295	1790	1568	1187	1088	64	312	177	88	94	440	17	314	156	260	75	1112	441	215	83	1424	321	608	360
	211.0	417.0	426.0	316.0	353.0	131.0	242.0	274.0	200.0	298.0	307.0	119.0	304.0	267.9	177.0	196.0	438.0	334.0	176.0	274.0	355.0	411.0	366.0	333
Crypto-sporidiosis	8	42	27	26	122	1	20	16	5	19	60	7	39	14	63	8	151	27	24	18	93	54	81	49
	5.7	9.8	7.3	6.9	39.5	2.0	15.5	24.8	11.4	60.3	41.8	49.0	37.8	24.0	42.8	20.9	59.5	20.5	19.6	59.3	23.2	69.1	48.8	45.4
Dengue fever		6	18	12	4	1	2	1		1			2	1	3	1	3	4			10		1	
		1.4	4.9	3.2	1.3	2.0	1.5	1.6		3.2			1.9	1.7	2.0	2.6	1.2	3.0			2.5		0.6	
Gastroenteritis	11	102	102	47	33	1	4	6	15	5	7		11	19	33	10	84	49	17	5	362	89	54	18
	7.8	23.7	27.7	12.5	10.7	2.0	3.1	9.3	34.1	15.9	4.9		10.7	32.5	22.4	26.1	33.1	37.2	13.9	16.5	90.1	113.9	32.5	16.7
Giardiasis	30	189	235	135	164	5	55	20	8	15	123		15	28	45	11	130	71	31	20	127	21	50	20
	21.4	44.0	63.9	35.9	53.1	10.2	42.6	31.0	18.2	47.6	85.7		14.5	48.0	30.6	28.7	51.2	53.8	25.3	65.9	31.6	26.9	30.1	18.5
Hepatitis A	2	17	29	20	17	1		1		1	5				2		2	5	2		2		1	1
	1.4	4.0	7.9	5.3	5.5	2.0		1.6		3.2	3.5				1.4		0.8	3.8	1.6		0.5		0.6	0.9
Hepatitis B	4	8	10	6	6		4		6	2	2	1	1	2	1	1	3	1	2		8		1	
	2.9	1.9	2.7	1.6	1.9		3.1		13.7	6.3	1.4	7.0	1.0	3.4	0.7	2.6	1.2	0.8	1.6		2.0		0.6	
Hepatitis C		3	2	3	1	1	15	4		1	2				1	1	9			3	4	2		
		0.7	0.5	0.8	0.3	2.0	11.6	6.2		3.2	1.4				0.7	2.6	3.5			9.9	1.0	2.6		
Lead Absorption	5	7	7	2	11		2		4		3		5	2	6	1	2	1	1		14	5	9	3
	3.6	1.6	1.9	0.5	3.6		1.5		9.1		2.1		4.8	3.4	4.1	2.6	0.8	0.8	0.8		3.5	6.4	5.4	2.8
Legionellosis	3	3	4	3	1		2			1	4	2	2	1		2	5	4	2		8	2	2	
	2.1	0.7	1.1	0.8	0.3		1.5			3.2	2.8	14.0	1.9	1.7		5.2	2.0	3.0	1.6		2.0	2.6	1.2	
Leptospirosis	9	6		2	20	1	7		6		29	2	6	4	12	2	1		12	2	4	9	6	2
	6.4	1.4		0.5	6.5	2.0	5.4		13.7		20.2	14.0	5.8	6.9	8.2	5.2	0.4		9.8	6.6	1.0	11.5	3.6	1.9
Listeriosis		3	3	2	1		2								1			1			3	1		1
		0.7	0.8	0.5	0.3		1.5								0.7			0.8			0.7	1.3		0.9
Malaria		6	3	4	7		2	2		1	1	2	1		10		5	1	2		7	2	3	1
		1.4	0.8	1.1	2.3		1.5	3.1		3.2	0.7	14.0	1.0		6.8		2.0	0.8	1.6		1.7	2.6	1.8	0.9
Measles		2	1	1	2		1										1		6	5	5	1		
		0.5	0.3	0.3	0.6		0.8										0.4		4.9	16.5	1.2	1.3		
Meningococcal disease	29	35	44	87	37	15	38	34	6	19	31	1	10	5	10	3	28	12	3	8	32	7	49	14
	20.7	8.1	12.0	23.2	12.0	30.6	29.4	52.7	13.7	60.3	21.6	7.0	9.7	8.6	6.8	7.8	11.0	9.1	2.5	26.4	8.0	9.0	29.5	13.0
Mumps	3	9	6	7	1		2	2			4			1			3	1	6	1	4	1	9	4
	2.1	2.1	1.6	1.9	0.3		1.5	3.1			2.8			1.7			1.2	0.8	4.9	3.3	1.0	1.3	5.4	3.7
Paratyphoid	4	5	2	2	2			1			2						2		2			1		1
	2.9	1.2	0.5	0.5	0.6			1.6			1.4						0.8		1.6			1.3		0.9
Pertussis	8	97	28	33	89	3	10	3	1	4	17	1	22	34	32	6	62	57	83	79	205	159	5	33
	5.7	22.6	7.6	8.8	28.8	6.1	7.7	4.7	2.3	12.7	11.8	7.0	21.3	58.2	21.7	15.7	24.4	43.2	67.8	260.4	51.0	203.5	3.0	30.5
Rhuematic fever	8	4	15	32	5	2	5	1	2		3		1	1		1	5	1			1			
	5.7	0.9	4.1	8.5	1.6	4.1	3.9	1.6	4.6		2.1		1.0	1.7		2.6	2.0	0.8			0.2			
Rubella	1	1	2	1		2	1				13		1				2	1	3	2	2		1	
	0.7	0.2	0.5	0.3		4.1	0.8				9.1		1.0				0.8	0.8	2.5	6.6	0.5		0.6	
Salmonellosis	48	174	172	150	136	22	38	35	40	22	86	6	47	36	57	23	144	48	137	16	187	72	93	80
	34.2	40.5	46.8	39.9	44.1	44.8	29.4	54.3	91.0	69.8	59.9	42.0	45.6	61.7	38.7	60.1	56.8	36.4	111.9	52.7	46.6	92.1	56.0	74.1
Shigellosis	3	14	25	23	4		1	3			7		2	1			4	1	1		13	4	5	1
	2.1	3.3	6.8	6.1	1.3		0.8	4.7			4.9		1.9	1.7			1.6	0.8	0.8		3.2	5.1	3.0	0.9
Tuberculosis	12	64	78	62	24		9	3	2	4	30		2	2	6	5	28	18	2		19	4	8	2
	8.6	14.9	21.2	16.5	7.8		7.0	4.7	4.6	12.7	20.9		1.9	3.4	4.1	13.1	11.0	13.7	1.6		4.7	5.1	4.8	1.9
Typhoid		6	2	10													3	1			1			
		1.4	0.5	2.7													1.2	0.8			0.2			
VTEC/STEC	1	3	5	3	15	5	1	3		1	2		5	1	1		4	1			18	1	1	2
	0.7	0.7	1.4	0.8	4.9	10.2	0.8	4.7		3.2	1.4		4.8	1.7	0.7		1.6	0.8			4.5	1.3	0.6	1.9
Yersiniosis	5	70	67	44	36	2	21	5	3	7	16	2	3	4	12	4	38	10	4	20	61	15	18	9
	3.6	16.3	18.2	11.7	11.7	4.1	16.3	7.8	6.8	22.2	11.1	14.0	2.9	6.9	8.2	10.5	15.0	7.6	3.3	65.9	15.2	19.2	10.8	8.3

Key: Cases
Rate per 100 000

H. Table of age-standardised rates and 95% CIs by DHB, 2002

Note: Rates (per 100 000) are directly standardised to the New Zealand population

District Health Board	Northland	Waitemata	Auckland	Counties Manukau	Waikato	Lakes	Bay of Plenty	Tairawhiti	Taranaki	Hawke's Bay	Wanganui	Midcentral	Hutt	Capital and Coast	Wairarapa	Nelson Marlborough	West Coast	Canterbury	South Canterbury	Otago	Southland
Campylo-bacteriosis	211.4 [187.5, 237.8]	411.4 [392.4, 431.1]	409.6 [389.1, 430.9]	319.1 [301, 338.1]	346.2 [326, 367.3]	279.9 [247.3, 315.7]	215.4 [194, 238.7]	206.2 [165.4, 254.4]	307.2 [273.6, 343.9]	305.6 [277.6, 335.8]	257 [218.6, 300.5]	183.2 [162.4, 205.9]	334.8 [304.2, 367.8]	430.6 [405.1, 457.3]	208.4 [163.3, 263.1]	177.1 [153.8, 203.1]	298.2 [237.3, 370.9]	355.3 [337.6, 373.8]	457.7 [398.7, 523.5]	363.7 [335.3, 394]	325.2 [291.1, 362.2]
Crypto-sporidiosis	4.9 [1.9, 10.7]	9.5 [6.8, 12.9]	7.3 [4.8, 10.8]	5.8 [3.7, 8.8]	37.7 [31.4, 44.9]	30.1 [20.4, 43.1]	11.4 [7.0, 17.7]	10.7 [3.4, 26.1]	37.7 [26.8, 51.8]	42.1 [32.2, 54.3]	31.9 [19.4, 49.8]	42.5 [32.8, 54.2]	20 [13.2, 29.3]	63.1 [53.4, 74.2]	22.1 [9.5, 45.4]	21.2 [13.6, 31.8]	63.2 [36.7, 102.4]	26.4 [21.6, 31.9]	83.2 [59.3, 114.2]	55.7 [44.3, 69.2]	46.1 [33.7, 61.6]
Gastroenteritis	8.3 [4.1, 15.3]	22 [17.8, 26.9]	26.2 [21.2, 32.1]	12.3 [8.9, 16.7]	8.6 [5.7, 12.6]	10.6 [5.1, 19.7]	3 [0.9, 7.3]	33.2 [18.1, 56.1]	9.8 [4.7, 18.4]	5.1 [2.0, 10.7]	30.2 [18.1, 47.7]	13.6 [8.6, 20.6]	36 [26.4, 48.0]	29.9 [23.5, 37.6]	28.7 [13.6, 54.4]	12.6 [7.2, 20.8]	15.7 [5.1, 39.2]	83.6 [75.2, 92.7]	182.7 [145.3, 227.3]	31.4 [23.5, 41.3]	16.5 [9.6, 26.5]
Giardiasis	21.2 [14.3, 30.7]	42 [36.1, 48.6]	62.3 [54.5, 71.0]	35.1 [29.4, 41.8]	51.1 [43.5, 59.6]	34.8 [24.1, 48.9]	36.3 [27.7, 46.8]	17.7 [7.5, 35.8]	14.9 [8.3, 24.9]	88.7 [73.9, 105.7]	45.2 [30.0, 65.8]	30.8 [22.5, 41.1]	52 [40.6, 65.7]	52 [43.4, 61.9]	21.2 [9.0, 44.0]	26.7 [18.1, 38.2]	73.3 [44.6, 114.5]	31.1 [26.0, 37.0]	33.7 [19.2, 55.6]	32.7 [24.4, 43.0]	15.5 [8.8, 25.3]
Hepatitis A	1.5 [0.2, 6]	3.9 [2.3, 6.3]	7.8 [5.1, 11.4]	5.3 [3.2, 8.4]	5.3 [3.1, 8.5]	2.2 [0.3, 8.1]	0.7 [0, 4]			3.6 [1.2, 8.6]		1.2 [0.2, 4.6]	3.7 [1.2, 8.9]	0.8 [0.1, 3]		1.7 [0.2, 6.5]		0.5 [0.1, 1.8]			0.9 [0, 5.4]
Lead Absorption	3.6 [1.1, 9]	1.6 [0.6, 3.4]	2 [0.8, 4.2]	0.5 [0.1, 2.2]	3.5 [1.7, 6.3]		0.6 [0, 3.7]	9 [2.5, 23.9]	4.8 [1.6, 11.6]	2.2 [0.5, 6.6]	3.2 [0.4, 12.3]	4 [1.5, 8.8]	0.8 [0, 4.5]	0.7 [0.1, 2.8]	2.7 [0.1, 17.9]	0.7 [0, 4.8]		3.4 [1.9, 5.7]	8.1 [2.1, 22.1]	5.3 [2.4, 10.3]	2.8 [0.6, 8.5]
Leptospirosis	6.3 [2.9, 12.5]	1.4 [0.5, 3.1]		0.6 [0.1, 2.4]	7.1 [4.4, 10.8]		4.7 [2.0, 9.5]	17.1 [6.9, 35.6]	5.9 [2.2, 13.2]	20.2 [13.4, 29.4]	7.1 [1.9, 18.5]	8 [4.1, 14.1]		0.5 [0.0, 2.6]	4.8 [0.6, 20.7]	9.7 [5.0, 17.3]	6 [0.7, 25.3]	1.4 [0.5, 3.1]	14.2 [5.6, 30.6]	3.7 [1.4, 8.3]	2 [0.2, 7.3]
Meningococcal disease	21.3 [14.2, 31.1]	8.2 [5.7, 11.4]	12.7 [9.2, 17.1]	20 [16.0, 24.9]	11.2 [7.9, 15.5]	53 [39.7, 69.6]	29.7 [22.1, 39.3]	12.1 [4.4, 27.7]	9.2 [4.2, 17.8]	21 [14.3, 30.0]	9.2 [3.4, 20.8]	7.3 [3.7, 13.1]	8.8 [4.5, 15.6]	11.7 [7.8, 17.0]	7.5 [1.5, 24.9]	2.7 [0.6, 8.1]	33 [15.0, 64.0]	8.1 [5.6, 11.4]	12.6 [4.6, 28.4]	29 [21.4, 38.6]	14 [7.6, 23.6]
Mumps	2 [0.4, 6.7]	2.1 [1, 4]	1.8 [0.7, 4]	1.7 [0.7, 3.8]	0.3 [0, 1.8]	1.9 [0.2, 7.4]	1.2 [0.1, 4.7]			2.6 [0.7, 7]	1.5 [0, 9.5]		0.8 [0, 4.5]	1.3 [0.3, 3.9]		5.5 [2, 12.1]	4.1 [0.1, 23.6]	1.3 [0.4, 3]	2 [0.1, 12.9]	5.9 [2.7, 11.3]	3 [0.6, 8.8]
Pertussis	5.6 [2.4, 11.5]	22.3 [18.1, 27.3]	8.5 [5.7, 12.3]	7.8 [5.4, 11.2]	26.8 [21.5, 33.1]	6.6 [2.6, 14.0]	7.1 [3.8, 12.4]	1.8 [0.1, 12.5]	19.8 [12.2, 30.7]	11.5 [6.7, 18.7]	53 [36.9, 74.3]	21.2 [14.5, 30.0]	41.5 [31.4, 53.9]	26.5 [20.3, 34.1]	15.9 [5.8, 36.7]	72.5 [57.8, 90.0]	258.5 [204.4, 323.9]	53.7 [46.7, 61.4]	304.9 [257.6, 359.0]	4.8 [1.9, 10.0]	31.1 [21.1, 44.2]
Rhuematic fever	5.5 [2.3, 11.4]	0.9 [0.2, 2.4]	4.7 [2.6, 7.8]	7.4 [5, 10.7]	1.5 [0.5, 3.6]	0.9 [0, 5.9]	3.9 [1.5, 8.4]	3.8 [0.5, 15.6]	0.9 [0, 5.8]	2.1 [0.4, 6.4]	1.4 [0, 9.3]		0.7 [0, 4.4]	2.1 [0.7, 5.1]	2.6 [0.1, 17.7]			0.3 [0, 1.5]			
Salmonellosis	33.2 [24.4, 44.5]	39.7 [34.0, 46.2]	46 [39.3, 53.6]	38.5 [32.5, 45.4]	42.7 [35.9, 50.5]	56.9 [42.8, 74.3]	34.3 [26.1, 44.4]	88.5 [63, 121.4]	45.7 [33.5, 61.1]	59 [47.2, 73.1]	61.5 [43.4, 84.9]	37.8 [28.7, 48.9]	36 [26.5, 47.9]	56.4 [47.4, 66.7]	64.3 [40.3, 98.4]	116.6 [97.9, 138]	50 [27.8, 84.5]	52.1 [45.3, 59.6]	88.6 [63.9, 120.3]	59.9 [48.5, 73.2]	72.2 [56.6, 90.8]
Shigellosis	1.6 [0.2, 6.3]	3.2 [1.7, 5.4]	6.8 [4.4, 10.1]	5.4 [3.4, 8.3]	1.3 [0.4, 3.3]	3.2 [0.7, 9.6]	0.5 [0, 3.5]		2 [0.2, 7.7]	5 [2, 10.6]	1.3 [0, 9]		0.7 [0, 4.4]	1.7 [0.5, 4.5]		0.7 [0, 4.8]		3.2 [1.7, 5.5]	8.7 [2.4, 23.2]	3.1 [1, 7.4]	1 [0, 5.7]
Tuberculosis	8.4 [4.3, 15.2]	15.5 [12.0, 19.8]	20.1 [15.8, 25.3]	16.7 [12.7, 21.7]	7.4 [4.7, 11.1]	7.5 [3.0, 15.6]	5.3 [2.4, 10.3]	4.7 [0.6, 17.8]	2.4 [0.3, 8.5]	22.2 [15.0, 31.8]	3 [0.4, 11.8]	3.8 [1.4, 8.4]	13.7 [8.1, 21.8]	11.4 [7.6, 16.6]	13.6 [4.3, 34.0]	1.4 [0.2, 5.9]		4.8 [3.0, 7.4]	1.7 [0.0, 12.1]	4.4 [1.9, 9.0]	2 [0.3, 7.2]
VTEC/STEC	0.7 [0, 4.7]	0.7 [0.1, 2.1]	1.5 [0.5, 3.6]	0.7 [0.1, 2.4]	4.5 [2.5, 7.5]	3.7 [1, 10]	3.2 [1.2, 7.3]		4.8 [1.6, 11.7]	1.3 [0.2, 5.1]	1.5 [0, 9.5]	0.7 [0, 3.8]	0.8 [0, 4.5]	1.7 [0.5, 4.5]				4.4 [2.6, 7]	2 [0.1, 12.9]	0.5 [0, 3.4]	2.1 [0.3, 7.5]
Yersiniosis	3.4 [1.1, 8.5]	15.8 [12.3, 20.1]	17.9 [13.8, 22.9]	11.3 [8.2, 15.4]	11.3 [7.9, 15.7]	12.8 [6.6, 22.5]	13.4 [8.5, 20.2]	7.2 [1.5, 21.7]	3.1 [0.6, 9.4]	11.3 [6.4, 18.6]	9.6 [3.5, 21.4]	7.9 [4.1, 13.9]	7.5 [3.6, 14.0]	15.1 [10.7, 20.9]	10.1 [2.7, 28.5]	3.5 [1.0, 9.2]	65 [39.5, 102.4]	15.6 [12.0, 19.9]	20.3 [9.6, 38.5]	10.8 [6.4, 17.3]	8.8 [4.0, 16.8]

Key: Age-standardised rate
95% confidence interval

I. Table of outbreaks by pathogen/toxin and region

Outbreaks reported during 2002, by pathogen/toxin and reporting Public Health Unit

Pathogen or Toxin	Northland	Auckland	Waikato	Tauranga	Gisborne	Rotorua	Taranaki	Hawkes Bay	Wanganui	Manawatu	Wairarapa	Wellington	Nelson	Marlborough	West Coast	Canterbury	South Canterbury	Otago	Southland	National	Total no. of outbreaks	Total outbreak cases
Enteric																						
Bacillus cereus		1										1			1	1					4	16
Campylobacter		30				2	2	1	1	4		1				8		2			51	239
Ciguatera poisoning		2																			2	9
Clostridium perfringens		3							1			1				2					7	127
C. perfringens and *B. cereus*												1									1	7
Cryptosporidium parvum		2	1			2			1	4		3			1			1			15	120
Cucurbitacin toxin		1																			1	3
Escherichia coli O157																1					1	3
Gastroenteritis (not specified)	1	68	3	1	1					1		7	2			1		2			87	317
Giardia		5		1		1					1	1				1		1			11	68
Hepatitis A virus		1						1				1								1	4	34
Histamine poisoning		3																			3	10
Norovirus		29	2	3			5	1	1			8				19	2	2	1		73	1419
Norovirus, *B. cereus*																1					1	3
Norovirus, Aeromonas caviae																1					1	23
Rotavirus			1									1									2	37
Salmonella		20			1	3	1			3		2	2	1	1			1			35	249
Scombroid fish poisoning		2																			2	22
Shigella		5														1	1				7	27
Solanine toxin																1					1	2
Staphylococcus aureus		3					1														4	9
Staph, *C. perf.*, Norovirus, *B. cer.*																1					1	29
Yersinia		1								1					1						3	10
Non enteric																						
Bordetella pertussis									1	1					5						7	32
Hepatitis C virus		1																			1	3
Leptospira Pomona										1			1								2	5
Mycobacterium tuberculosis		1						2									1				4	39
Neisseria meningitidis group C															1			1			2	8
Total	**1**	**178**	**7**	**5**	**2**	**8**	**9**	**5**	**5**	**15**	**1**	**27**	**5**	**1**	**10**	**38**	**4**	**10**	**1**	**1**	**333**	**2870**

Note: Blank cell denotes a value of zero