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The New Zealand Paediatric Surveillance Unit: establishment and first year of operation

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The New Zealand Paediatric Surveillance Unit (NZPSU) was established in late 1997 to provide active surveillance of acute flaccid paralysis (AFP) in order to fulfil World Health Organization 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 are sent a reply-paid card on which they indicate whether in the previous month they have seen any cases of the conditions under surveillance. When a case is reported, further details on the case are sought via a short questionnaire sent to the reporting doctor. In 1998, the NZPSU received reports of 6 confirmed cases of AFP, 14 cases of HUS, 1 case of CRS, 6 pregnancies to HIV-infected women, and 2 cases of vitamin K deficiency bleeding. This system complements other forms of surveillance. It provides a method to investigate emerging problems, such as HUS, and to monitor the effectiveness of prevention programmes.

The New Zealand Paediatric Surveillance Unit (NZPSU) was established in October 1997 to improve the knowledge of rare childhood conditions in New Zealand. These conditions are of sufficiently low incidence or prevalence that case ascertainment on a national scale is needed to generate adequate numbers for meaningful study. The method was developed in the United Kingdom by the British Paediatric Surveillance Unit and has been used there since 1986.¹ Subsequently, it has been introduced into several other countries including Australia,² and is used by some other specialist groups.

The NZPSU was initially established, under a contract with the Ministry of Health, to provide active surveillance of acute flaccid paralysis (AFP). The World Health Organization (WHO), as part of the global poliomyelitis eradication programme, requires such surveillance to confirm that New Zealand is free of wild-type poliovirus infection. Since the NZPSU's establishment, the number of conditions under surveillance has increased and now includes nine uncommon childhood conditions.

This report explains how the NZPSU surveillance system works and reports some key findings from its first full year of operation.

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Method

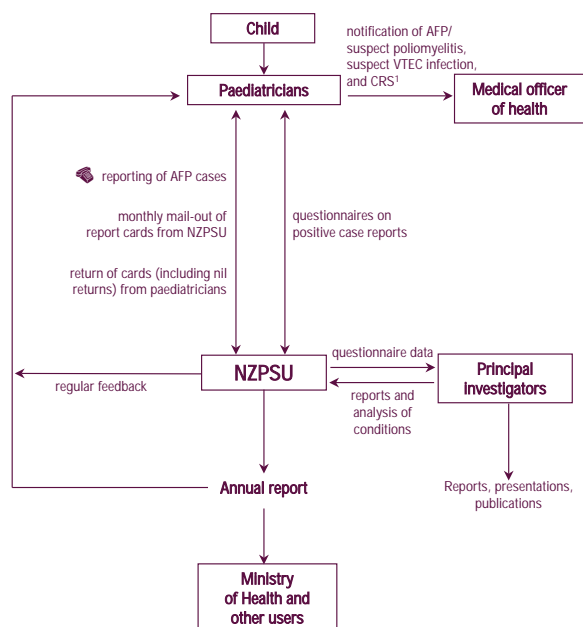
Specialist paediatricians gave their support to the surveillance system after the concept was discussed at annual meetings of the Paediatric Society of New Zealand. A database of eligible clinicians, which included specialist paediatricians and other specialists working predominantly with children, was developed using the specialist register and the membership list of the Paediatric Society. All eligible clinicians were contacted and invited to participate. Those who agreed were provided with study protocols, which included definitions of the conditions under surveillance, specific reporting instructions and a contact telephone number.

Figure 1 shows the main components and information flows of the NZPSU. Every month, participants are sent a reply-paid

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Figure 1: Operation of the NZPSU



Note: 1 AFP = acute flaccid paralysis
VTEC = verotoxigenic *Escherichia coli*
CRS = congenital rubella syndrome

card on which to report whether or not in the previous month they have seen any cases of the conditions under surveillance. Cases of AFP must also be reported immediately by phone to the NZPSU. When a case of any of the conditions is reported, the reporting clinician is sent a short questionnaire to complete on the case. The case's identity remains anonymous. Duplicate notification is recognised by a code derived from the child's initials and date of birth.

The conditions currently under surveillance are shown in Table 1. The protocols and questionnaires used for AFP, haemolytic uraemic syndrome (HUS), congenital rubella syndrome (CRS), perinatal HIV exposure, and neonatal herpes simplex infection were all adapted from those used by the Australian Paediatric Surveillance Unit. A Scientific Review Panel has been established primarily to consider the inclusion of new conditions. The decision on which conditions to add is made on the basis of the value of the information to be gained and the overall workload required of the participants. Only conditions that would be expected to be under the care of a paediatric specialist can be considered. Conditions are included for a minimum of two years.

The cases of AFP and HUS are annually checked with hospital discharge data to assess the quality of surveillance. Data on HUS cases are also compared with notifications of

verotoxigenic *Escherichia coli* (VTEC) infection. Where possible, cases of perinatal HIV exposure are matched with notifications received by the New Zealand AIDS Epidemiology Group.

Regular reports are made to the Ministry of Health, specifically updating the progress with AFP surveillance. A quarterly newsletter updating surveillance of all the conditions is sent to the participants.

Results

All specialist paediatricians and all but one of the other specialists in paediatric practice, who were contacted, agreed to participate. In 1998, 163 clinicians participated in the system. The response rate to the monthly mail-out of reporting cards was 94%, with no consistent set of non-responders. The majority of participants regularly reported having seen no cases of the conditions under surveillance.

Table 1 shows the number of cases reported in 1998 and the estimated incidence rate for each of the conditions under surveillance. No data on neonatal herpes simplex infection are reported, as these data are only available for the second half of 1998.

Four of the six cases of AFP were Guillain-Barré syndrome, one was caused by trauma, and one was vaccine-associated paralytic poliomyelitis.³ The WHO requirement for excluding poliomyelitis in a person with AFP is that two stool samples, taken at least 24 hours apart and within 14 days of the onset of paralysis, are negative for poliovirus. Two of the six cases had two stool samples taken at least 24 hours apart and within 14 days of the onset of paralysis. Another three cases had two samples taken, but more than 14 days after onset of paralysis, and no faecal samples were obtained from the sixth case. The five cases with stools collected, tested negative for wild poliovirus. All children with a hospital discharge diagnosis of Guillain-Barré syndrome were notified to the NZPSU.

Fourteen children were reported with HUS. Bloody diarrhoea was the commonest presentation. Seven cases needed acute dialysis and a similar number needed treatment for acute hypertension. One child died of intra-cerebral haemorrhage. VTEC was isolated from ten of the cases: seven were serotype O157:H7 and three were serotype O113. The incidence was highest in pre-school children. One child with a hospital discharge diagnosis of HUS was not notified to the NZPSU.

One child was newly diagnosed with CRS. He was born in 1989 and had moderate developmental delay, congenital heart disease, and visual and hearing disabilities. The mother was 23 years old at the time of her child's birth, had then been in New Zealand for 2 years, and her vaccination history was not known.

Table 1: Conditions currently included in the NZPSU surveillance system, and the number and rate of cases in 1998

Condition (age range included)	Surveillance started	Confirmed cases in 1998		Principal investigator(s) ¹
		No.	Incidence rate	
Acute flaccid paralysis (<15 years)	October 1997	6	0.7 per 100 000 under 15 years	Dr Nigel Dickson, Dr Paul Shillito
Haemolytic uraemic syndrome (<15 years)	January 1998	14	1.7 per 100 000 under 15 years	Dr William Wong
Congenital rubella syndrome (<15 years)	January 1998	1	- ²	Prof Diana Lennon
Perinatal HIV exposure	January 1998	4	7.0 per 100 000 births	Dr Nigel Dickson, Dr Lesley Voss
Vitamin K deficiency bleeding	January 1998	2	3.5 per 100 000 births	Assoc Prof Brian Darlow
Neonatal herpes simplex infection	January 1998	- ³	- ³	Dr Dawn Elder
Retinopathy of prematurity: stage III and beyond	January 1999	-	-	Assoc Prof Brian Darlow
Subdural haemorrhage (<2 years)	January 1999	-	-	Dr Patrick Kelly
Childhood diabetes mellitus (<15 years)	January 1999	-	-	Prof Barry Taylor, Dr Priscilla Campbell-Stokes
Fetal alcohol syndrome	July 1999	-	-	Dr Alison Leversha

Notes: 1 The principal investigator is responsible for analysing the data collected by the NZPSU.

2 The child diagnosed with congenital rubella syndrome (CRS) in 1998 was born in 1989. Rates of CRS are generally presented per 100 000 pregnant women. As this child was diagnosed many years after birth, such a rate is inappropriate.

3 Complete data could not be collected until the second half of 1998.

Table 2: Surveillance and practice points for the conditions included in the NZPSU surveillance system in 1998

Acute flaccid paralysis (AFP)	<ul style="list-style-type: none"> Two faecal specimens, taken 24 hours apart, must be collected from all cases of AFP within 14 days of the onset of paralysis. Specimens must be sent to ESR laboratories for testing. All cases of AFP must be: <ul style="list-style-type: none"> immediately notified as suspect poliomyelitis to the local medical officer of health, and reported by phone to the NZPSU (03 474 7825) if the case is <15 years old.
Haemolytic uraemic syndrome (HUS)	<ul style="list-style-type: none"> A faecal specimen must be collected early from all children with bloody diarrhoea, and cultured for VTEC. All cases of HUS must be: <ul style="list-style-type: none"> immediately notified as suspected VTEC infection to the local medical officer of health, and reported to the NZPSU if the case is <15 years old.
Congenital rubella syndrome (CRS)	<ul style="list-style-type: none"> Children should be immunised against rubella with MMR vaccine at the routinely scheduled ages of 15 months and 11 years. The rubella immunity of all women of child-bearing age must be checked. In particular, the rubella immunity of women born overseas should be checked at their first health encounter. Any susceptible women should be vaccinated if they are not pregnant. If they are pregnant, they should be vaccinated after delivery. Mothers who are breastfeeding can be vaccinated. All newly diagnosed cases of CRS must be: <ul style="list-style-type: none"> notified to the local medical officer of health, and reported to the NZPSU if the case is <15 years old.
Perinatal HIV exposure	<ul style="list-style-type: none"> Assessment of HIV infection risk should be a routine part of antenatal care. Mothers known to be HIV positive should continue antiretroviral treatment during pregnancy and be offered zidovudine chemoprophylaxis to reduce perinatal transmission of HIV. All cases of perinatal HIV exposure must be reported to the NZPSU.
Vitamin K deficiency bleeding	<ul style="list-style-type: none"> Lead maternity carers should inform prospective parents of the issues relating to vitamin K deficiency bleeding and of the recommendations for prophylaxis. The current recommendations are that all newborns be given vitamin K by a single intramuscular injection at birth. Alternatively, vitamin K may be administered orally, but multiple doses are required.¹⁰ Cases of late-onset haemorrhage may occur, particularly among breast-fed infants, if vitamin K prophylaxis is not given. Infants with late onset disease should be investigated for liver disease. All cases of vitamin K deficiency bleeding in infants must be reported to the NZPSU.

Six children were recognised in 1998 as having been perinatally exposed to HIV. Of these, two were born prior to 1998: one in New Zealand and one overseas. The mother of the New Zealand-born child had epidemiological risks for HIV, but her infection was not diagnosed during pregnancy. Of the four HIV-infected pregnant women whose pregnancies ended in 1998, all were offered (and three accepted) antiretroviral therapy. Three were from high prevalence countries. One of the four pregnancies ended in a stillbirth and the other three in live born infants, none of whom have been diagnosed with HIV infection.

There were two cases of late-onset vitamin K deficiency bleeding reported. Neither case had received vitamin K. Both were breast-fed. Skin bruising occurred at 4-6 weeks of age, and one child had an intracranial hemorrhage. One of the children was diagnosed with liver disease and a peroxisomal disorder. There was an additional case of possible early-onset disease. The case responded to intramuscular vitamin K. No cause was found and investigations on this infant were limited.

Discussion

The first year of operation of the NZPSU has shown that an active surveillance system of this type can achieve reasonably complete reporting for a range of uncommon paediatric conditions, and therefore it is a sensitive system for diagnosed cases of the conditions under surveillance. All cases of AFP and all but one case of HUS identified from the hospital discharge data were also reported to the NZPSU. Additionally,

the rate of AFP detected by the system (0.7 cases per 100 000) is similar to the sensitivity of 1 case of AFP per 100 000 children under 15 years specified by the WHO.⁴

All the conditions under surveillance in 1998 occurred only infrequently, with cases of HUS and AFP being the most common (Table 1). The main practice points and the reporting requirements for the diseases under surveillance in 1998 are included in Table 2.

Surveillance of AFP has provided evidence that there have probably been no cases of wild-type poliovirus infection in New Zealand since the NZPSU was established in October 1997. Surveillance of AFP was the main impetus for the establishment of the NZPSU. Identification and investigation of all cases of AFP as suspect poliomyelitis is required to demonstrate that New Zealand is free of wild-type poliovirus infections. The certification of New Zealand's status is required as part of the final stages of the global poliomyelitis eradication programme. While the sensitivity of the NZPSU system to identify cases of AFP appears satisfactory, some deficiencies in the investigation of the cases, in particular obtaining specimens, were identified. Faecal specimens correctly taken at the recommended times (Table 2) were obtained for only two of the six cases reported. AFP cases should be reported immediately by telephone to the NZPSU (Table 2). Early reporting should help ensure that the correct samples are taken. WHO requires that two stool samples be taken at least 24 hours apart and within 14 days of the onset of paralysis.

HUS is a major reason for children needing dialysis.^{5,6} In recent years there has been a rise in children hospitalised with HUS in New Zealand and in the number of VTEC infections identified,⁷ a pattern seen in several other developed countries. Most cases reported to the NZPSU were associated with infection with VTEC serotype O157:H7, and bloody diarrhoea was the commonest presentation. A faecal specimen should be taken early from all suspect cases (Table 2), as the number of organisms decreases rapidly after about the third day of diarrhoea. Early diagnosis helps ensure appropriate care is provided, and minimises morbidity and mortality.

A high level of rubella vaccination among children to reduce virus circulation, and identification and vaccination of non-immune women is the key to the control of CRS (Table 2). While only one case of CRS was reported to the NZPSU in 1998 and was in a child born in 1989, it is likely some cases remain undiagnosed, especially as some infected infants appear normal at the time of birth.

The number of pregnancies among HIV-infected women is not known. Lead maternity providers should follow Ministry of Health guidelines for the identification of HIV-infected pregnant women, which place emphasis on identifying women with risk factors for HIV infection (Table 2).⁸ Although it is recommended that testing of pregnant women is based on risk-factor assessment, it is unlikely that this is always undertaken.⁹ As not all HIV-infected mothers might have been identified, paediatricians need to be aware of the possibility of undiagnosed HIV infection in children.

Earlier claims that intramuscular vitamin K could cause cancer may have resulted in some children receiving no, or inadequate, prophylaxis. The weight of evidence now refutes these claims.¹⁰ Neither case of late-onset vitamin K deficiency bleeding in 1998 had received the recommended prophylaxis (Table 2).

The NZPSU surveillance system does have some limitations. First, the system can only be used for conditions that can reasonably be expected to come under the care of a paediatrician. For this reason, it is only suitable as a method

of monitoring or investigating serious conditions. Second, as with any surveillance system, conditions that are under-diagnosed, for example CRS or maternal HIV infection, will be under-reported.

The third limitation is that the monthly reporting frequency is not adequate for diseases that require an immediate public health response. Three of the conditions monitored by the NZPSU are also notifiable to the local medical officer of health: AFP as suspect poliomyelitis, HUS as suspected VTEC infection, and CRS (Table 2). This allows the medical officer of health to take urgent action to identify sources of infection and prevent further cases.¹¹ Any suspect poliomyelitis case initially needs to be treated as a potential wild-type poliovirus infection, and assessed without delay to determine whether it is likely to be due to wild-type virus. VTEC infections need to be investigated to determine likely sources of infection and to identify common source outbreaks.

However, the active NZPSU surveillance system is more sensitive for AFP than the system of passive notifications to medical officers of health. Although practitioners have been advised for several years that all cases of AFP should be immediately notified to the medical officer of health, only two cases were notified in 1998 (Galloway Y. Personal communication, 1999), a third of the number reported by the NZPSU system. This current rate of notification of AFP cases to medical officers of health would not meet the WHO's specified sensitivity for AFP surveillance.

The NZPSU system complements other forms of disease monitoring. Linking information on HUS provided by paediatricians with notifications of VTEC infection, allows the burden of serious disease due to this pathogen to be determined. The system provides an active method of assessing the effectiveness of disease prevention programmes, such as those to eliminate poliomyelitis, vitamin K deficiency bleeding, and CRS. Also, with time, as children perinatally infected with HIV become symptomatic, it will be possible to estimate the effectiveness of antenatal HIV detection.

The information reported during the first full year of the NZPSU's operation predominantly provides a measure of the effectiveness of control programmes. The data that are now being collected on neonatal herpes infection, childhood diabetes, subdural haemorrhage, and fetal alcohol syndrome will provide national information on the epidemiology of these conditions that has not previously been available. Such increased understanding of these conditions is necessary to guide treatment and control programmes.

In the future we will remove some conditions from the list. Some, such as those that are providing surveillance of control programmes will probably remain, whereas others, such as those that are providing basic epidemiological information, will be included for only as long as is needed to obtain the necessary information.

Minimising the extra workload that the system imposes on paediatricians is a key factor for its success. The range of conditions under surveillance and their incidence needs to be kept under review. Confining the system to conditions that are very rare will limit the demand on clinicians' time, but conversely it will be less likely to provide useful information. Feedback both to and from clinicians is vital, as their perception of the usefulness of the scheme will influence its functioning.

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Surveillance and control notes

Further outbreaks of cryptosporidiosis associated with swimming pools

The incidence of cryptosporidiosis continued to rise in the first 4 months of 1999, with a cumulative total of 393 notified cases, compared with 249 cases in the corresponding months of 1998. The current rate of cryptosporidiosis is 27.9 per 100 000, which is significantly higher than the previous rate of 15.0 per 100 000. Over the last 12 months up to and including April 1999, rates increased in all health districts except Northland, Gisborne and Hutt. There was a large swimming pool-associated outbreak in the Hutt during March and April 1998.

Sixty-one cases of cryptosporidiosis associated with an aquatic centre in the Canterbury Health District were notified during March and April. Fifty of the cases had used the centre

and the other 11 cases were household or family contacts of people who had been at the centre. The majority (60%) of the 50 primary cases were children under 5 years of age. Most cases reported that their illness began in the middle 2 weeks of March. The centre's records showed that there had been faecal accidents in the learners' pool on 11 and 16 March. An investigation by Crown Public Health found that the centre was generally well run. The learners' pool and main pool had separate water recirculation and diatomaceous earth filtration systems. Water disinfection at the centre included chlorine dioxide and ozone treatment, both of which can be effective against *Cryptosporidium*. The pools were closed and cleaned. No further cases associated with the centre were detected. (Reported by Dianne Morrison, Health Protection Officer, Crown Public Health.)

Surveillance and control notes

During April, there was another outbreak of cryptosporidiosis associated with a swimming pool complex. The complex was in the Auckland region, and at least 23 people who had been at the complex on the afternoon and evening of 24 April were affected. No other cases of gastro-intestinal illness among people who used the complex at other times were identified, nor was there an increase in apparently sporadic cases of cryptosporidiosis in the region. It is likely that a faecal accident occurred in the afternoon of 24 April and people using the pool during the rest of that day were affected. When the outbreak became apparent on 12 May, samples of pool water and filter backwash were taken, and control measures, including changing the filter media and pool water, were taken. *Cryptosporidium* oocysts were not detected in the samples taken. (Reported by Donald Campbell, Medical Officer of Health, Auckland Healthcare.)

Prevention of *Cryptosporidium* transmission via contaminated swimming pools should focus on reducing the risk of contaminating pool water. In particular, people who have had diarrhoea in the previous two weeks should not use pools. See the *New Zealand Public Health Report* 1998; 5: 41-5 for a full discussion of the recommended prevention and control measures.

Pertussis epidemic may start in late 1999

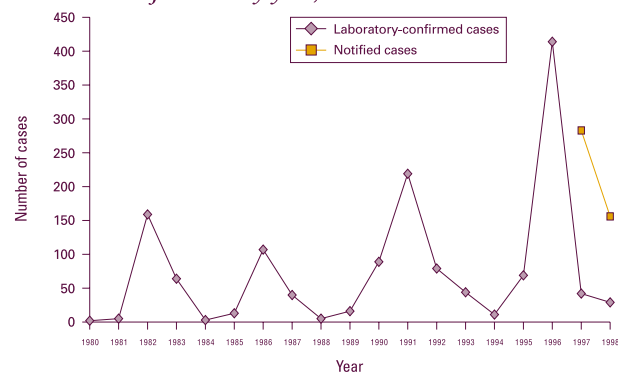
Pertussis epidemics occur every 4-5 years in New Zealand (Figure 1). The last epidemic started in late 1995 and continued throughout most of 1996. Because of the current low rate of pertussis immunisation, the inter-epidemic period could be reduced and the next epidemic could begin later this year.

There has already been increased pertussis activity in some parts of the country. Late last year there was a cluster of cases in Nelson. In April this year, a cluster of nine cases was reported in the Wellington Health District, with five of the cases from the Kapiti area. Eight of the cases were children: one was under one year old, four were 1-4 years and three were 5-9 years.

Medical practitioners should notify all cases of pertussis on suspicion to their local officer of health. The case definition for a suspect case includes any child with paroxysmal cough with whoop, vomiting or apnoea for which there is no other known cause. Practitioners are encouraged to obtain laboratory confirmation for cases. The preferred method is by culture from a nasopharyngeal swab. Isolation of the organism from throat swabs is less sensitive.

Infants should be protected against pertussis as early as

Figure 1: Pertussis laboratory-confirmed cases and notifications by year, 1980-1998

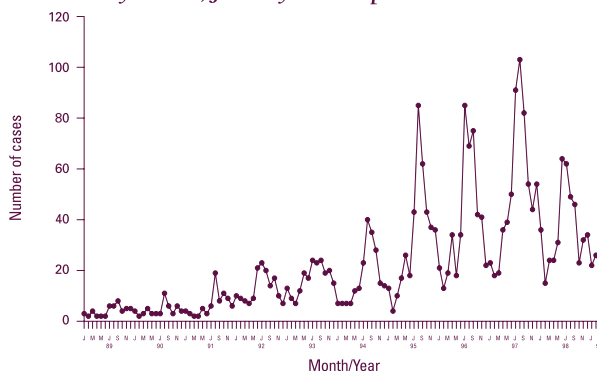


possible by ensuring that they receive their vaccinations on time. Delaying immunisation not only delays protection but may cause more reactions. About half of vaccine recipients experience minor reactions to the whole cell pertussis vaccine in DTPH. More serious reactions (persistent and unusual crying, convulsions and hypotonic hyporesponsive episodes) are rare, occurring in less than 1% of children. Less reactogenic acellular pertussis vaccines are now available (as combined diphtheria-tetanus-pertussis vaccine) in New Zealand, but are not state funded. Use of these vaccines could be considered to complete the immunisation of children who have a severe reaction to their first DTPH.

Meningococcal disease epidemic continues

The incidence of meningococcal disease has increased again this year following a sharp fall in the second half of 1998, and is now similar to the incidence during the first half of 1998. One hundred cases were notified during the first 4 months of 1999, which is very close to the number (102) of cases notified during the same months in 1998 (Figure 2).

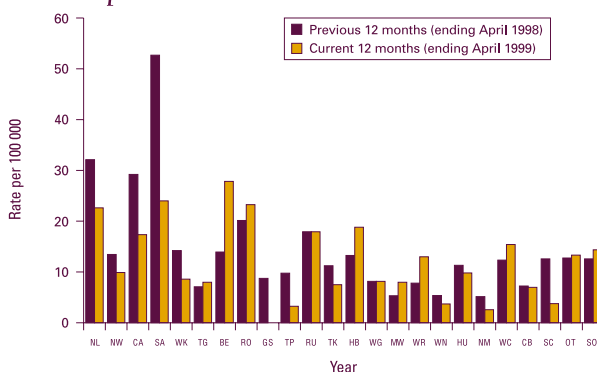
Figure 2: Total and confirmed meningococcal disease cases by month, January 1991-April 1999



During the 12 month period up to and including April 1999, the rates of meningococcal disease fell in 13 of the 24 health districts but increased in 9 districts (Figure 3). During the same 12 months, the rate of meningococcal disease decreased in Pacific Islands people (99.9 to 50.8 per 100 000) and Maori (38.6 to 27.5), but was unchanged in Europeans (7.4).

Medical practitioners should maintain a high level of suspicion for meningococcal disease, especially during winter and spring months. Prior to transfer to hospital, practitioners should administer parenteral antibiotics to suspected cases who have any haemorrhagic rash and to all suspected cases who are not likely to reach hospital within 30 minutes.

Figure 3: Meningococcal disease rates by health district, previous and current rates



Surveillance data

National surveillance data - April 1999

Disease ¹	Current year - 1999 ²			Previous year - 1998			Trends - April 1999
	Apr 1999 cases	Cumulative total year-to-date	Current rate ³	Apr 1998 cases	Cumulative total year-to-date	Previous rate ³	
AIDS	2	6	0.7	3	11	1.2	<p>Percentage change⁷</p> <p>Significance of difference: * p<0.05 ** p<0.01 *** p<0.001</p>
Acute gastroenteritis ⁴	26	169	12.9	23	114	9.6	
Campylobacteriosis	514	2750	290.4	821	3746	253.9	
Cholera	0	0	0.0	0	0	0.0	
Creutzfeldt-Jakob disease	0	0	0.0	0	0	0.0	
Cryptosporidiosis	110	393	27.9	158	249	15.0	
Dengue fever	0	7	0.6	3	10	0.6	
Giardiasis	182	747	57.5	200	830	58.1	
<i>H influenzae</i> type b disease	2	3	0.4	1	3	0.2	
Hepatitis A	5	49	3.8	13	61	8.0	
Hepatitis B (acute) ⁵	9	32	2.5	5	22	3.2	
Hepatitis C (acute) ⁵	9	33	2.5	7	41	3.0	
Hydatid disease	0	3	0.1	1	1	0.1	
Influenza ⁶	7	15	12.5	0	3	20.5	
Lead absorption	17	56	3.1	6	21	1.8	
Legionellosis ⁶	3	15	1.7	14	64	4.4	
Leprosy	0	2	0.1	0	0	0.1	
Leptospirosis	1	14	1.6	10	30	1.8	
Listeriosis	1	5	0.4	0	8	0.7	
Malaria	3	17	1.5	7	33	2.0	
Measles	10	30	3.3	10	88	52.7	
Meningococcal disease	25	100	12.4	24	102	17.1	
Mumps	4	19	2.0	8	33	2.5	
Paratyphoid	0	5	0.5	0	1	0.5	
Pertussis	17	36	4.1	11	45	6.1	
Rheumatic fever	3	18	1.9	6	14	2.0	
Rubella	2	8	1.1	5	25	1.7	
Salmonellosis	253	1098	61.2	206	954	44.1	
Shigellosis	12	66	4.0	10	43	3.2	
Tetanus	0	3	0.1	0	1	0.0	
Tuberculosis	23	134	10.8	31	113	9.2	
Typhoid	0	4	0.7	1	11	0.6	
VTEC/STEC infection	11	28	1.6	9	17	0.7	
Yersiniosis	30	216	14.0	44	252	15.6	

Notes: 1 No cases of the following notifiable diseases were reported in April: anthrax, brucellosis, cysticercosis, diphtheria, meningococcal disease - primary amoebic, plague, poliomyelitis, rabies, rickettsial diseases, trichinosis, viral haemorrhagic fever, or yellow fever

2 These data are provisional

3 Rate is based on the cumulative total for the current year (12 months to April 1999) or the previous year (12 months to April 1998), expressed as cases per 100 000

4 Cases with suspected common source, person in a high-risk category (eg, foodhandler, childcare worker, healthcare worker)

5 Only acute cases of this disease are currently notifiable

6 Surveillance data based on laboratory-reported cases only

7 Percentage change is the difference between the number of cases in the current year (12 months to April 1999) and the previous year (12 months to April 1998). This difference is expressed as a percentage of the number of cases seen in the previous year.

Surveillance data

Surveillance data by health district - April 1999

Cases this month Current rate¹

Disease	Cases for April 1999, ² and current rate ^{1,2} by health district ^{3,4}																							
	Northern				Midland						Central						Southern							
	Northland	NW Auck	Central Auck	South Auck	Waikato	Tauranga	Eastern BOP	Gisborne	Rotorua	Taupo	Taranaki	Ruapehu	Hawkes Bay	Wanganui	Manawatu	Wairarapa	Wellington	Hutt	Nelson-Marl	West Coast	Canterbury	South Cant	Otago	Southland
AIDS ³	2				0						0						0							
	1.2				0.4						0.9						0.6							
Acute gastroenteritis	1	4	3	4	0	1	0	0	0	0	0	0	0	0	1	0	0	2	2	7	2	0	0	
	4.4	11.7	24.3	11.1	5.0	1.8	0	74.3	1.5	0	1.9	0	0	0	1.3	0	3.3	0.8	6.0	6.2	55.6	3.8	0.6	3.6
Campylobacteriosis	9	60	82	30	40	12	2	0	14	0	9	1	24	5	11	5	47	21	9	3	89	12	14	15
	137.9	304.9	324.5	230.6	354.3	218.1	165.1	144.3	199.9	198.7	180.6	185.1	327.6	172.6	175.5	156.0	520.4	446.4	129.5	200.5	370.4	310.6	267.0	235.4
Cholera	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	0	0.3	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Creutzfeldt-Jakob disease	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Cryptosporidiosis	1	9	3	4	12	2	0	2	0	0	0	0	6	1	8	0	6	2	0	0	52	1	0	1
	13.9	10.1	6.7	8.8	73.0	19.5	2.0	32.8	4.6	55.4	7.5	35.8	35.5	21.2	52.5	23.4	31.3	56.6	4.3	117.2	38.8	50.3	19.7	38.6
Dengue fever	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	0	1.0	2.3	0.6	0.3	0	0	0	0	0	0	0	0	0	0	0	0.8	0	0	0	0.8	0	1.2	0
Giardiasis	1	33	19	12	16	5	0	1	5	2	3	0	10	1	11	5	13	6	5	6	25	3	0	0
	38.7	66.0	70.6	58.8	80.6	63.8	21.9	102.7	54.2	48.9	16.8	23.9	84.3	42.3	33.9	36.4	90.2	67.1	18.0	111.0	46.3	42.7	29.0	43.1
H influenzae type b disease	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0
	0	0.5	0.3	0.9	0.3	0	2.0	0	0	0	0	0	0.7	1.6	0	0	0.4	2.3	0	0	0.3	0	0	0
Hepatitis A	0	2	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0
	20.4	6.3	4.9	7.3	1.3	1.8	0	2.2	1.5	0	0	0	2.1	1.6	0	0	3.3	0	2.6	0	4.4	2.5	0	1.8
Hepatitis B	0	0	0	2	3	0	0	0	0	0	0	0	2	0	0	0	0	0	1	0	0	0	0	1
	5.1	1.3	1.7	4.1	3.6	2.7	0	13.1	0	0	0	11.9	5.6	0	2.0	0	1.6	3.8	1.7	3.1	2.8	0	1.7	1.8
Hepatitis C	0	0	1	1	0	2	0	0	0	0	0	0	0	0	0	0	0	0	0	0	4	1	0	0
	0	1.3	1.7	0.6	0.3	15.1	2.0	0	12.4	0	0	0	1.4	0	1.3	2.6	1.2	0.8	1.7	6.2	8.0	5.0	0.6	0
Hydatids	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	0	0	0.3	0	0	0.9	0	0	0	0	0	0	0.7	0	0	0	0	0	0.9	0	0	0	0	0
Influenza ⁵	0	1	3	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0	1	0
	1.5	5.8	4.9	10.0	16.9	0.9	13.9	0	0	13.0	6.6	0	2.8	8.1	0.7	2.6	7.0	0	1.7	6.2	68.0	6.3	2.3	2.7
Lead absorption	1	0	1	0	6	1	0	0	0	0	0	0	2	0	1	0	1	0	0	0	2	1	1	0
	2.2	0.8	3.8	1.8	5.6	3.5	2.0	2.2	10.8	0	5.6	0	9.1	1.6	2.7	0	1.6	1.5	1.7	0	3.9	6.3	1.2	3.6
Legionellosis ⁵	0	0	0	0	1	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0	1	0	0	0
	0	0	4.6	0	3.0	0	0	0	0	0	2.8	0	2.1	1.6	1.3	10.4	1.6	2.3	0	0	4.1	0	0.6	0.0
Leprosy	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	0	0	0.3	0.9	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Leptospirosis	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0	0
	4.4	0	0	0.9	3.3	2.7	0	4.4	1.5	0	3.7	6.0	3.5	3.3	2.7	7.8	0.4	0	2.6	9.3	0.3	5.0	0.6	0.9
Listeriosis	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	0.7	0.5	1.2	0.3	0.7	0	0	0	0	0	0	0	0	0	0	0	0.4	0	0	0	0.3	2.5	0	0
Malaria	0	0	0	0	1	0	0	1	0	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0
	0.7	2.0	1.2	1.2	1.7	1.8	0	2.2	3.1	0	2.8	6.0	0.7	6.5	2.0	0	1.2	0	0.9	0	1.6	2.5	0.6	2.7
Measles	0	0	2	1	1	0	0	1	0	0	0	2	0	0	0	2	0	0	0	0	1	0	0	0
	0.7	3.0	4.6	2.3	4.3	4.4	4.0	2.2	1.5	0	0.9	0	4.9	0	5.3	5.2	2.5	2.3	2.6	0	4.1	0	6.9	2.7
Meningococcal disease	2	2	4	7	2	1	1	0	2	0	0	0	0	1	0	0	0	0	0	0	2	0	1	0
	21.9	10.1	18.8	28.7	8.6	8.0	21.9	0	27.9	3.3	8.4	17.9	18.1	8.1	7.3	13.0	4.1	9.8	2.6	18.5	6.5	3.8	13.3	14.4
Mumps	0	0	0	0	0	0	0	0	0	0	0	0	2	0	0	0	0	1	0	0	0	0	1	0
	2.9	0.5	0.9	3.5	0.7	1.8	2.0	0	3.1	9.8	1.9	0	5.6	1.6	0	10.4	3.7	1.5	1.7	0	2.3	0	3.5	0.9
Paratyphoid	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	0	1.3	0.9	0.9	0.3	0	0	0	0	3.3	0	0	0	0	0	0	0.4	0	0	0	1.3	0	0	0
Pertussis	1	0	0	0	3	0	0	0	0	0	0	1	0	0	0	7	2	0	0	1	2	0	0	0
	0.7	0.3	0.9	0.6	22.8	3.5	6.0	0	0	0	0	0	3.5	1.6	0.7	0	3.7	2.3	25.7	0	2.1	3.8	1.7	0.9
Rheumatic fever	1	0	0	0	1	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	2.9	0.0	0.6	4.1	5.3	0	2.0	15.3	3.1	0	0.9	17.9	3.5	0	0	5.2	4.5	0.8	0.9	0	0	0	0	0
Rubella	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	2	0
	0	1.3	0.3	0.6	0.3	0.9	0	0	0	0	0.9	0	4.9	0	0.7	2.6	2.1	1.5	0.9	0	2.1	0	2.9	0
Salmonellosis	12	34	37	14	22	6	1	0	4	1	7	1	4	2	7	2	27	10	6	0	35	5	9	7
	47.4	47.7	50.9	45.1	66.8	60.3	21.9	45.9	32.5	140.1	45.9	71.6	50.2	35.8	78.5	96.2	93.9	97.3	45.4	43.2	65.4	81.7	65.4	90.7
Shigellosis	1	1	5	2	1	0	0	0	0	0	0	0	0	0	0	1	1	0	0	0	0	0	0	0
	4.4	4.6	12.1	10.8	1.7	3.5	0	0	4.6	3.3	0.9	0	0.7	3.3	1.3	2.6	0.4	2.3	2.6	0	2.6	1.3	0.6	1.8
Tetanus	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	0	0	0	0	0	0	0	0	0	0	0.9	0	0	0	0	0	0.4	0	0	0	0	0	0.6	0.9
Tuberculosis	0	3	9	5	2	0	0	0	0	0	0	0	0	0	0	1	1	1	0	0	0	0	0	1
	7.3	7.4	24.0	24.9	10.9	8.0	8.0	2.2	6.2	6.5	1.9	6.0	4.2	0	8.0	5.2	16.5	12.8	3.4	3.1	9.3	3.8	2.3	12.6
Typhoid	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	0	0.8	1.4	2.6	0	3.5	0	0	0	0	0	0	0	0	0	0	0.8	1.5	0	0	0	0.3	0	0
VTEC/STEC infection	0	0	0	0	6	2	0	0	0	0	0	0	0	0	1	0	0	1	0	0	0	0	0	0
	0	0	0.6	0.3	10.6	5.3	4.0	0	3.1	3.3	1.9	0	0	0	0.7	5.2	0.4	0.8	0	0	0.3	2.5	1.2	0
Yersiniosis	2	4	5	1	2	0	0	1	0	0	2	0	0	0	1	0	4	2	0	2	2	2	0	0
	10.2	15.0	13.6	14.6	13.6	8.9	8.0	10.9	12.4	13.0	8.4	0	16.7	4.9	4.0	2.6	17.3	12.1	4.3	40.1	30.0	26.4	3.5	9.0

Public health abstracts

Evidence that human herpesvirus 8 is sexually transmitted in men

A United States study has found further evidence that human herpesvirus 8 (HHV-8) has a causal role in Kaposi's sarcoma and is sexually transmitted among men. The study was conducted among men enrolled in the San Francisco Men's Health Study, which was initiated in 1984 to study the natural history of HIV infection. Serum samples were taken from the men at the beginning of the study (1984-5). Antibodies to HHV-8 latency-associated nuclear antigen were found in 223 (37.6%) of 593 men who reported having any homosexual activity in the previous 5 years, but in none of 195 exclusively heterosexual men. HHV-8 infection correlated with a history of sexually transmitted diseases, the number of years of regular homosexual intercourse, and the number of male intercourse partners. Among men who were infected with both HIV and HHV-8, the probability of developing Kaposi's sarcoma within

10 years was 49.6%. HHV-8 infection preceded and was associated with Kaposi's sarcoma independently of the degree of sexual activity and HIV-related immune compromise (Sexual transmission and the natural history of human herpesvirus 8 infection. Martin JN, Ganem DE, Osmond DH, et al. *N Engl J Med* 1998; 338: 948-54).

Editorial note: There is now very good evidence that HHV-8 has a causative role in the development of Kaposi's sarcoma in HIV-infected men. This study provides the strongest evidence to date that HHV-8 is sexually transmitted among homosexual men. It is still unclear which specific practices lead to transmission. Kaposi's sarcoma was reported as the AIDS defining condition in 66 (9.7%) of the total 678 cases of AIDS that have been notified in New Zealand up until the end of March 1999. Additional cases may have developed Kaposi's sarcoma after notification of the case.

Insufficient evidence to recommend screening with the faecal occult blood test

The effectiveness of screening for colorectal cancer using the faecal occult blood test, Hemoccult, was evaluated by meta-analysis of results from four randomised controlled studies. The studies were conducted in Denmark, the United Kingdom, Sweden, and the United States, and involved people over 40 years of age. Mortality from colorectal cancer was reduced by 16% among study participants allocated to screening and by 23% among those who were actually screened. It was estimated that a biennial Hemoccult screening programme offered to 10 000 people ≥ 40 years of age, of whom two-thirds attended at least one test, would prevent 8.5 deaths over a 10 year period. Put another way, 1173 people would need to be screened to prevent one colorectal cancer death over 10 years. The authors concluded that the benefits of screening are likely to outweigh the possible physical and

psychosocial harm, at least among high-risk groups. However, before widespread screening can be recommended, more information is needed about the harmful effects, the community's responses to screening, and the costs (Towler B, Irwig L, Glasziou P, et al. A systematic review of the effects of screening for colorectal cancer using the faecal occult blood test, Hemoccult. *BMJ* 1998; 317: 559-65).

Editorial note: In 1998, following an 18-month review of the scientific evidence published up to May 1998, the National Health Committee Working Party on Population Screening for Colorectal Cancer recommended against population-based screening for colorectal cancer with faecal occult blood tests. This recommendation was based on the fact that the potential benefit was only modest, while a considerable commitment of health sector resources would be needed and there was a small but real potential for harm.

Travel health

Traveller's diarrhoea remains common in visitors to developing countries

The first in a planned series of studies of the current epidemiology of traveller's diarrhoea has found an attack rate of 23.6% for short-term visitors to Jamaica. For a mean duration of stay of 4-7 days, the risk was 20.9% for all traveller's diarrhoea and 10.0% for classical traveller's diarrhoea. This cross-sectional study was based on questionnaires administered to 30 369 short-term visitors to Jamaica just before boarding their homebound aircraft. Classical traveller's diarrhoea was defined as ≥ 3 unformed stools in 24 hours, with at least one accompanying gastrointestinal symptom. Incapacity lasted a mean of 11.6 hours. Less than 3% of all travellers avoided potentially high-risk foods and beverages. In a second arm of the study to investigate aetiology, 322 hotel guests with traveller's diarrhoea provided stool samples. The most frequently detected pathogens were enterotoxigenic *Escherichia coli*, rotavirus, and *Salmonella* (Steffen R, Collard F, Tornieporth N, et al. *Epidemiology, etiology, and impact of traveler's diarrhea in Jamaica. JAMA* 1999; 281: 811-7).

Editorial note: Prevention of traveller's diarrhoea remains difficult. Effective vaccines against the agents involved are not yet available and prophylactic antibiotic use should be reserved for only the most vulnerable patient groups. Travellers should be advised that most cases are self-limiting and require only oral replacement of fluids and salts.

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