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Estimates of HIV prevalence among pregnant women in New Zealand

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The development of interventions that markedly reduce the chance of perinatal transmission of HIV from an infected pregnant woman to her baby have spurred interest in improving detection of HIV among such women. On the basis of reports of known births to HIV infected women during 1998-2001, and the number of infected children born in New Zealand who have developed AIDS, an overall prevalence of HIV among women giving birth was estimated. This estimate is 1.5 to 4.0 per 10 000, with 45 to 80% not diagnosed before giving birth. Based on these figures, if all these infections were detected, an additional four to 18 pregnant women would be diagnosed with HIV annually and could be offered effective antenatal care. If interventions to prevent perinatal transmission were taken up by all of these women, it is estimated that on average five perinatal infections could be prevented every three years. We recommend that rapid consideration be given to the benefits, risks, and costs of moving to a policy where all pregnant women are offered and recommended to have a voluntary HIV test. In the meantime maternity carers should discuss HIV with all pregnant women. An initial prevalence study of HIV using neonatal blood spots should be undertaken to assess the true prevalence among women giving birth. In the future regular public health monitoring using neonatal blood spots, and ongoing collection on the number of women given birth known to be infected, will be required to assess the effectiveness of this policy.

Introduction

A landmark study in 1994 showed that if women with HIV infection were identified during pregnancy, the risk of perinatal transmission could be substantially reduced by the use of antiretroviral drugs.¹ Currently, in developed countries this can be reduced from around 25% to less than 2%.² In 1997, the Ministry of Health developed interim guidelines for HIV testing among pregnant women.³ These recommended screening questions for all pregnant women to assess their risk for HIV and offering testing to those "where risk factors are identified or not clear". They also recommended that data be collected to determine if routine antenatal testing was appropriate or desirable. Subsequent research on the application of those guidelines has shown that in the regions studied, most women are not questioned about risk and there is little HIV testing.^{4,5,6} Hence it is probable that not all pregnant women with HIV are being identified. In the meantime there have been calls by some,^{7,8} but not all,⁹ commentators for New Zealand to follow the United

Kingdom towards a policy where HIV testing is "offered and recommended" to all pregnant women.

Critical pieces of information needed for determining the benefits, risks, and costs of a change in policy are the current prevalence and the proportion of women with HIV giving birth who have undiagnosed infection.

To assist in reconsideration of the current policy, this paper describes the prevalence of known HIV infection among pregnant women and the incidence of diagnosed perinatal transmission for the period

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1998 to 2001. These results are used to derive a crude estimate of the number of HIV-infected women giving birth in that period. It is also discussed how the actual prevalence of HIV among women giving birth could be measured.

Methods

Information on the number of births to women known to be infected with HIV was obtained from the New Zealand Paediatric Surveillance Unit (NZPSU) scheme.¹⁰ Every month paediatricians are sent a reply-paid card on which they indicate whether in the previous month they have seen any cases of a number of conditions under surveillance. When one is reported, anonymous details on the case are sought via a short questionnaire sent to the reporting doctor. Since 1998, being born to an HIV-infected woman has been one of the 'conditions' under surveillance.¹⁰ This method can be expected to capture all infants born to known HIV-infected pregnant women under care, because of the need for paediatric follow up of their infants.

Information on the number of children with perinatally acquired HIV is collected by the AIDS Epidemiology Group. Since 1996 clinicians caring for people diagnosed with HIV have provided anonymous information including the likely mode of infection and the country in which this occurred.¹¹ Infected children who meet criteria for AIDS are notified to the group.

Estimates have been made of the number of HIV infected children likely to have been born during this period using the number of children notified with AIDS by mid-2002, who were born in New Zealand between 1998 and the end of 2001, and the data on the rate of progression to AIDS of perinatally infected children¹². Using these figures and data on the rate of perinatal transmission,¹³ estimates have been made of the number of pregnancies among HIV-infected women in this period. The number of births in New Zealand for this period was obtained from Statistics New Zealand and used to derive rates.¹⁴

Results

Between 1998 and the end of 2001, there were reports of 19 pregnancies to 18 women infected with HIV to the NZPSU. Four of the births were in 1998, two in 1999, six in 2000 (one who returned from overseas to give birth), and seven in 2001. One woman gave birth both in 1998 and 2000. There were 226 533 live births registered in this period. The prevalence of known HIV infection among women giving birth over this period was therefore 0.8 per 10 000 births. The place of birth, timing of the diagnosis of HIV, and ethnicity of mother are shown in Table 1.

Table 1. Place of birth, timing of diagnosis and ethnicity of the mother for all known HIV-infected women giving birth, 1998–2001

	No.	%
Place of birth		
Auckland*	12	63.2
Other North Island	5	26.3
South Island	2	10.5
Time of diagnosis		
Before this pregnancy	14	73.7
During this pregnancy	5	26.3
Ethnicity of Mother		
European	3	16.7
Maori (+ European/Maori)	5	27.8
Pacific	1	5.6
African	6*	33.3
Asian	3	16.7
Total	19	100.0

* One woman gave birth twice

In 17 of the 19 pregnancies, antiretroviral treatment was given, and it was refused in the remaining two. One infant suffered an intrauterine death at 30 weeks, and of the 18 remaining infants, 15 (83.3%) were born by Caesarian section. None were breast fed. None of the 18 live born infants is known to be infected, although it is too soon to be certain for two as they were less than one year old at last follow up. There have been 10 children diagnosed in New Zealand with perinatally acquired HIV since the start of 1998 reported to the AIDS Epidemiology Group. Of these, four were born in this country, three in the period of interest 1998–2001 (in fact all in 1999), and one earlier. None of the mothers of these three children born in New

Zealand were diagnosed with HIV before their child's birth. Currently two of these children have progressed to AIDS. The remaining child, who at initial assessment had moderate immunosuppression, was then started on therapy.

The combined data of two European studies of perinatally infected infants show that approximately 30% develop AIDS within two and a half years of birth, the average age (at June 2002) of the infected children born between 1998 and 2001.¹² Thus the two children born in New Zealand in this period who developed AIDS can be expected to represent 30% of the number born with HIV, which therefore is estimated to be seven (actual figure 6.7). Only a proportion of infants born to HIV-infected women become infected. In developed countries, prior to the institution of interventions to reduce the risk of perinatal transmission, such transmission occurred in between 13% and 32% of cases.¹³ Using a figure of 25% it would be expected that the calculated number of 7 (actual figure 6.7) infected children would be born from 27 (actual number 26.8) pregnancies among infected women. This represents 1.2 pregnancies among undiagnosed infected women per 10 000 women giving birth each year, with a total (diagnosed and undiagnosed) rate of 2.0/10 000 at the end of pregnancy.

There is potential for error because of the extremely small numbers. If instead of two there had been just one or three children developing AIDS, the estimate for the rate of undiagnosed HIV-infected pregnancies would have been 0.6 and 1.8/10 000 respectively. Also, the one child diagnosed with HIV who had moderate immunosuppression at diagnosis and was started on therapy might have developed AIDS during the period of follow up if this had not been started.

These estimates would also be affected by different values for the rates of perinatal transmission and progression to AIDS. If the lower perinatal transmission rate of 13% in the studies was used, and a 20% rather than 30%, rate of progression in the period of follow up, the estimate for the undiagnosed prevalence would be 3.4/10 000. However using the higher figures of 32% and 40% respectively, the estimate would be 0.7/10 000. These are used as upper and lower estimates.

The central estimate for the rate of undiagnosed HIV-infected pregnant women giving birth, 1.2/10 000, would mean that 41% (19/46) of infected pregnancies were currently identified. The proportion derived from the upper and lower estimates would be 20% and 55% respectively.

Discussion

The point estimate in the period 1998–2001, for the total prevalence of HIV infection during pregnancy of 2.0/10,000 is higher than that made in early 2000 - "in the order of 1 in 10 000"¹⁵ - because of the higher number of HIV diagnoses among women and children in the last two years. The current estimate is of the same order of magnitude as that measured in England and Wales outside London in 2000, 2.7 per 10 000, which was determined through unlinked anonymous testing of neonatal blood spots.¹⁶ At that time the prevalence of HIV in the same region among heterosexual women attending sexual health clinics, determined through unlinked anonymous prevalence studies was 1.1/1000, very similar to the rate found in the last such study in New Zealand in 1996/7.¹⁷

The data on pregnant women with diagnosed HIV show that while a disproportionate number of women were from Africa and Asia, parts of the world where heterosexually transmitted HIV is relatively common, about half the women with HIV diagnosed in pregnancy were European, Maori or Pacific. Selectively testing that concentrates only on women from high prevalence countries would have missed up to half the cases. Although the numbers are very small, the number of Maori women diagnosed was higher than expected from the proportion in the population of childbearing age. It is not known how many of these women who would have been picked up using the recommended risk assessment because of limited information on behaviour of the infected women or their partners. However, it needs to be appreciated that a possible reason for this policy not being implemented is that it requires exploration of past sexual partnerships and injecting drug use.

Those women diagnosed before or during their pregnancy are receiving appropriate care which will lower the risk of their children acquiring HIV, and it is encouraging that none of the infants appear to have been infected.

There is clearly uncertainty about the estimates due to small numbers and the range of estimates of progression rate and transmission risk. However biases from under-reporting or under-recognition of children with AIDS, and from treatment preventing the progression to AIDS, will tend to make these estimates conservative.

The lack of information on the number of women who terminated a pregnancy on account of HIV infection will not affect the estimate of the number of undiagnosed pregnancies to infected women who give birth, but it will affect the overall rate at the start of pregnancy. The proportion undetected, even the highest in the possible range, is lower than the 84% found in England outside London in 1997, when a policy of selective testing similar to that now existing in New Zealand was operating.¹⁸ It is possible that detection is indeed more common in New Zealand, not because testing is any better among pregnant women, but because more women who have entered the country from high prevalence areas – particularly Africa – may have learnt their HIV status as part of refugee assessment. Nevertheless many people entering New Zealand from high prevalence areas are coming under standard immigration or family reunification schemes, and are therefore not being tested.

The true prevalence of HIV among women giving birth can be obtained, and such data collection is required to resolve the uncertainties. HIV antibodies cross the placenta and can be detected in newborns of infected mothers, even though they may not be infected themselves. Prevalence can be determined by testing newborn blood samples that are taken for metabolic screening. In many countries this is an established method for monitoring the epidemic.^{18,19,20} To ensure complete testing, hence an unbiased result, a sample from each blood spot is taken. It is anonymised and permanently unlinked from any identifying information and then tested for HIV antibodies. Overseas this approach has passed legal and ethical scrutiny and has been recommended by the World Health Organization.^{21,22} In New Zealand an analysis of ethical issues has concluded that there is no potential harm to the individual and the knowledge gained will be beneficial to the community.²³ Nevertheless, there is now a potential legal hurdle in New Zealand in the Code of Health and Disability Services Consumers Rights (Right 7 (10)), which limits the use of even anonymous blood samples. An amendment to the Code, which is expected, should allow this form of monitoring to proceed. As well as the amendment to the Code it will be very important that there is informed public debate prior to the use of the newborn blood samples for this purpose. If people mistakenly assumed such monitoring caused harms to individuals, then the whole newborn screening programme could be put in jeopardy.

In conclusion the estimated prevalence of undiagnosed HIV in women giving birth during 1998-2001 was 1.2/10 000, with a range of 0.7 to 3.2 per 10 000. The estimate for the total (diagnosed plus undiagnosed) prevalence is 2.0 per 10 000, with a range of 1.5 to 4.0 per 10 000. These figures do not include those women who opted to terminate a pregnancy on account of the infection. These estimates mean that 59% of infected women were undetected, with a range of 45% to 80%. The possible biases in the method used mean that the estimates of the rates of infection are more likely to under- rather than over-estimate the true prevalence. Moreover, the pattern of the last few years has been for increasing number of women to be diagnosed with HIV, a further reason why this estimate is likely to be conservative for the future.

We recommend that rapid consideration be given to the benefits, risks, and costs of moving to a policy where all pregnant women are offered and recommended to have a voluntary HIV test with appropriate counseling. Based on these figures, if such testing was fully implemented it would be expected to result in an additional seven (range four to 18) pregnant women being diagnosed with HIV annually. If interventions to prevent perinatal transmission

were taken up by all of these women it is estimated that five perinatal infection could be prevented every three years. In addition these women would have the opportunity to access treatment that might improve their own well being. In the meantime maternity carers should discuss HIV with all pregnant women. An initial prevalence study of HIV using neonatal blood spots should also be undertaken. In the future regular public health monitoring using neonatal blood spots and ongoing collection on the number of women given birth known to be infected will be required to assess the effectiveness of this policy.

Acknowledgements

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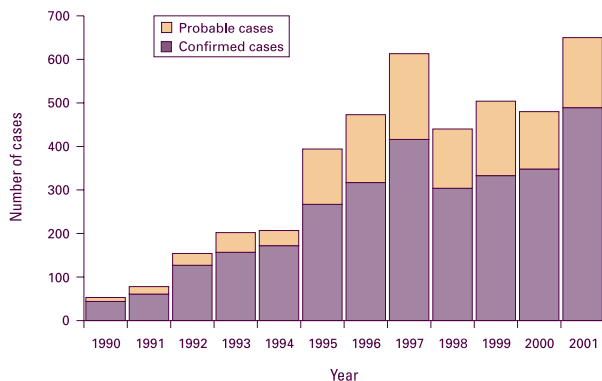
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Meningococcal disease in 2001 reaches highest incidence to date

The current New Zealand meningococcal disease epidemic continued into its eleventh year in 2001. Since the epidemic began in 1991, 4195 cases have been notified. Over the 1991 to 1997 period the number of cases increased each year, from 78 cases in 1991 to a peak of 613 cases in 1997 (Figure 1). Since then, the incidence has remained at a highly elevated level with a minimum of 440 cases a year. In 2001, the incidence increased to 650 notified cases giving a rate of 17.4 per 100 000, the highest rate to date in the epidemic. Just over three quarters (489/650, 75.2%) of the 2001 cases were laboratory confirmed, giving a rate for confirmed meningococcal disease of 13.1 per 100 000. This is the highest percentage of confirmed cases since 1994.

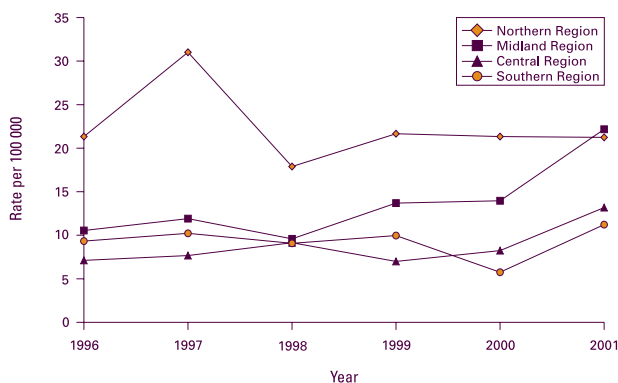
Figure 1: Meningococcal disease cases, 1990-2001



During 2001, the usual seasonal pattern continued for total cases, with 63.2% occurring during the winter and spring months (June – November). Seasonal distribution differed between regions and age bands.

Rates per 100 000 population were highest in Midland, rather than the Northern region for 2001, the first time this has occurred since the epidemic began (Figure 2). Between 1996-2001, the rate for the Midland region has shown a significant increase (chi-square for trend, $p=0.04$). Incidence of disease by health district was highest in Taupo (41.3 per 100 000), Rotorua (40.3), South Auckland (33.0), Eastern Bay of Plenty (32.6) and Otago (32.5), while the lowest rates were in South Canterbury (3.8), Canterbury (4.5), and Ruapehu (7.0).

Figure 2: Meningococcal disease rates by region, 1996-2001



Rates of meningococcal disease continued to be highest in the <1 and 1-4 year age groups (205.0 and 81.4 per 100 000 respectively) (Table 1). There is also a second much smaller peak in incidence around 18 years of age. Otherwise, rates decline markedly with age, with cases aged over 20 years accounted for less than 17% of the 2001 cases. The age-standardised rate for Maori in 2001 was over two times the rate seen in the European population, while the rate for Pacific peoples was over four and a half times that seen in the European population. The highest rate for a specific age-ethnicity group was among Pacific

infants <1 year old (639.9 per 100 000). The median age for cases of meningococcal disease by ethnicity differed markedly between ethnicities, being 2.6 years for people of Maori ethnicity, and 3.8 years for Pacific peoples compared with 16.6 years among the European population.

Table 1: Age group and ethnicity distribution of meningococcal disease cases, 2001

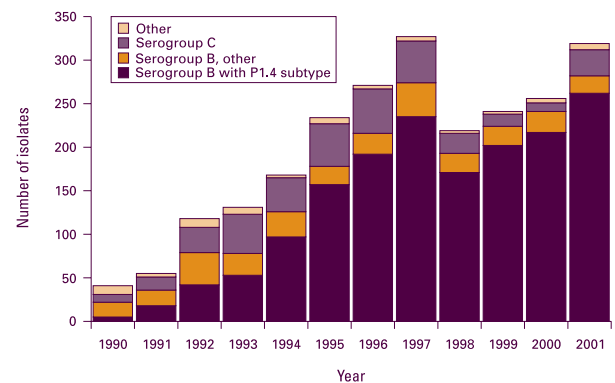
Age group (years)	European		Maori		Pacific		Other		Unknown	Total	
	Cases	Rate ¹	Cases	Rate ¹	Cases	Rate ¹	Cases	Rate ¹		Cases	Rate ¹
<1	17	57.1	60	428.8	33	639.9	2	53.8	0	112	205.0
1-4	48	39.5	73	136.3	53	276.4	1	7.1	1	176	81.4
5-9	26	15.4	19	28.7	28	118.6	2	10.9	0	75	28.2
10-14	31	17.5	23	36.6	13	60.0	0	0.0	3	70	24.1
15-19	59	36.8	16	32.3	16	88.2	3	10.7	1	95	35.8
20-29	38	12.4	10	12.2	5	14.8	2	4.6	2	57	11.7
30-39	17	4.3	6	7.7	1	3.2	1	2.2	0	25	4.3
40+	28	2.2	4	3.3	6	12.8	2	2.7	0	40	2.6
Total	264	11.5	211	25.7	155	53.1	13	4.9	7	650	17.4

Note:

¹ Rates per 100 000 based on 2001 census data. The total rate for each ethnic group is standardised to the age distribution of the New Zealand population.

During the epidemic, *Neisseria meningitidis* serogroup B meningococcal disease has been responsible for an increasing proportion and number of meningococcal disease cases. The proportion of serogroup B cases peaked in 2000 at 94.1% (Figure 3). Serogroup B continued to dominate in 2001, although an increase in serogroup C meningococcal disease to 9.4% of cases proportionately lowered serogroup B meningococcal disease percentage to 88.4%. The increase in serogroup B disease has been largely due to the one epidemic strain with the serologic type B:4:P1.7b,4. As a proportion of serogroup B isolates, isolates with the P1.7b,4 subtype have increased from 50.0% in 1991 to 92.9% in 2001. Meningococci with the P1.7b,4 subtype, regardless of serogroup accounted for 80.5% of all 2001 cases for which the subtype could be determined. The PorA proteins on the surface of the meningococcus define the subtype. This P1.7b,4 PorA subtype is the target antigen in the vaccine that is being used in clinical trials in New Zealand aimed at controlling the epidemic.

Figure 3: Meningococcal disease cases, 1990-2001



Since the meningococcal disease epidemic began in 1991, 185 deaths have been notified, giving an average case fatality rate for the epidemic of 4.4%. Meningococcal disease resulted in 26 deaths in 2001, the highest number for a single year to date. The case fatality rate for 2001 was 4.0%. Based on clinical presentation data recorded in the EpiSurv notification database, cases with septicaemia only, had a markedly higher case fatality rate (8.1%) than those cases with meningitis only (2.1%) or meningitis and septicaemia (2.7%).

Of the 632 cases in 2001 for whom hospitalisation status was recorded, 619 (97.9%) were hospitalised. Pre hospital antibiotics were given to 22.9% of cases (135/589) for whom this information was recorded. Antibiotic treatment was highest in Midland region (27.5%) and 1-4 year olds (29.7%) and lowest in Southern region (13.0%) and 30-39 year olds (9.1%). The case fatality rate among cases who saw a doctor and were given antibiotic treatment was 2.2%, compared with 2.6% among cases who saw a doctor and did not receive antibiotics, and

7.5% among cases not seen by a doctor prior to hospital admission (and consequently not given pre hospital antibiotics).

One consequence of pre hospital antibiotic treatment is a reduction in confirmation of cases by the isolation of meningococcus from blood, CSF and other normally sterile sites. In 2001 an organism was cultured from only 30.4% of 135 cases treated with antibiotics prior to sampling, compared with 56.1% of 460 cases not treated before sampling. This difference was statistically significant (chi square, $p < 0.0001$). The use of polymerase chain reaction (PCR) which can detect the DNA of non-viable meningococci enabled the confirmation of 38.3% of the cases treated with antibiotics prior to testing by PCR. These results affirm the value of PCR testing and should encourage the promotion of its use. Geographic differences in the use of PCR are present. In 2001, only 11.5% of cases were confirmed by PCR in the Northern region, compared with 26.3% in the Central region, 33.0% in the Southern region, and 40.6% in the Midland region.

Most cases of meningococcal disease were sporadic. However, 25 cases (5.2%) were reported to be contacts, that is, possible associated cases. Information on the suspect index cases was available for 18 possible associated cases, and eight were classified as secondary cases (symptoms began within 2-60 days of the onset of disease in the index case) and ten were considered co-primary cases (symptoms began within one day of disease in the index case). Chemoprophylaxis was reported to have been taken by five of the 18 associated cases.

Meningococcal disease continues to be one of the most serious infectious diseases in New Zealand, causing more hospitalisations and deaths than any other notifiable disease. The disease also results in significant morbidity among cases that survive. Several strategies have or are being developed to control the current epidemic to improve outcome among cases, and to improve the diagnosis and surveillance of meningococcal disease.

In May 2002 phase I clinical trials to test the safety and immunogenicity of a strain-specific vaccine began in 75 healthy adults. Further phases of clinical trials will continue for at least 18 months in the target population for the mass immunisation campaign. It is anticipated that a mass vaccination campaign targeting all under 20 year olds will proceed once clinical trials are complete, assessed against safety and effectiveness guidelines and regulatory approval is granted by the Director-General of Health on the recommendations of Medsafe.

To improve disease outcome, medical practitioners should maintain a high degree of suspicion for meningococcal disease and administer antibiotics to suspected cases prior to sending them to hospital. Hospital clinicians are encouraged to make greater use of PCR testing to increase the rate of case confirmation and the proportion of cases for which the causative organism can be typed. Public health staff should ensure contacts are identified and offered prophylaxis, encourage greater confirmation of disease and continue to collect a complete set of data on each case to improve the current enhanced surveillance of this disease. It will be of particular importance to collect reliable information on vaccine status to assess the effectiveness of the vaccine programme.

(Information in this report was extracted from Martin D, McDowell R, Garrett N, Baker M. 2002. The Epidemiology of Meningococcal Disease in New Zealand in 2001. A report prepared for the Ministry of Health by the Institute of Environmental, Science and Research (ESR). Wellington: Ministry of Health. (Unpublished report)

New Zealand Paediatric Surveillance Unit Update

The New Zealand Paediatric Surveillance Unit (NZPSU) was established in late 1997 to provide active surveillance of acute flaccid paralysis (AFP) in New Zealand to fulfil the World Health Organization (WHO) requirements for certification of polio eradication.

The way NZPSU operates was detailed in a previous report published in the New Zealand Public Health Report.¹ Briefly, all paediatricians are asked to report by phone all children with AFP, the key clinical feature of poliomyelitis, to the NZPSU as soon as possible after admission and definitely while the child is still in hospital. Two stool samples are sent for testing at ESR so that polio can be discounted.

In addition, each month all paediatricians throughout New Zealand are sent either a reply-paid card or email (depending on their preference) on which they indicate whether they have seen a child with AFP in the previous month. The process has enabled the incidence of a number of other conditions seen by paediatricians to be investigated. These studies are generally undertaken for a period of two or three years, although some will probably remain ongoing. The response rate by paediatricians is high, with a monthly average of 94%.

When a case of any of the conditions under surveillance is notified to NZPSU, a short anonymous questionnaire is sent to the notifying paediatrician to ascertain demographic details and a few clinical details. Several studies also require a follow-up questionnaire to ascertain outcome.

Currently there are ten conditions on the NZPSU card. Conditions of particular interest are shown in the following table. Investigation of the AFP cases has confirmed that New Zealand is polio free but surveillance for AFP needs to continue for several more years.

Condition	2000	2001	2002 (til June)
Acute Flaccid Paralysis	14	11	4
Perinatal HIV exposure	5	9	3
Vitamin K Deficiency Bleeding	2	2	5
Haemolytic Uraemic Syndrome	7	6	9
Congenital Rubella	0	0	0

As these are currently being investigated, duplicate reports might be discovered.

The NZPSU prepares and distributes an annual report that summarises the year's activities and findings. The Principal Investigators for all studies are asked to update their study for this report, and are encouraged to disseminate their findings in a variety of ways.

Further information on how the NZPSU operates, and previous annual reports can be found at: www.paediatrics.org.nz/nzpsu/nzpsu1.html (Reported by Melissa Carter, NZPSU)

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Three HUS cases in Rotorua

Two children with VTEC (verotoxigenic *Escherichia coli*) infection were notified by Rotorua Hospital to Toi te Ora Public Health late in June. They were siblings, aged one and four years. The younger of the two developed symptoms three days prior to his sister, who was fed by naso-gastric tube due to cerebral palsy. Both were referred to Starship Hospital for treatment of haemolytic-uraemic syndrome (HUS). Routine public health follow-up failed to reveal a likely cause although person-to-person transmission from the first case to his sister was suspected.

A third case of VTEC infection, in a child aged seven, was notified to A+ Public Health Unit in early July. This child also required treatment for HUS at Starship Hospital. She lives on a farm in Auckland and had visited Rotorua during the incubation period of her illness. Her symptoms began some 18 days after the second case became unwell. Further investigation revealed the three cases had all eaten at the same restaurant in Rotorua, and the third case had also visited a petting zoo in Rotorua.

Inspection of the food premises confirmed good food handling and cooking practices. The home water supply of the third case is from a bore supplemented with roof rainwater. Testing confirmed significant bacterial contamination. Inspection of the petting zoo showed some significant hazards including direct farm animal contact, the possibility of consumption of raw dairy products, and inadequate hand-washing facilities.

Other than consumption of food at the same restaurant, there was no obvious connection between the first two cases and the third case. Pulsed field typing of isolates has since confirmed that a different organism was responsible for the third case. The cause of the first case remains unknown, but based on the assessed risk and the incubation time the petting zoo is the presumed source for the third case (reported by Dr Phil Shoemack, Medical Officer of Health, and Steve Goodin, Senior Health Protection Officer).

Surveillance data

National surveillance data - July to September 2002

Disease ^{1,2}	Current year - 2002 ³			Previous year - 2001			Disease trends - Year ending September 2002
	Jul-Sep 2002 cases	Cumulative total since 1 January	Current rate ⁴	Jul-Sep 2001 cases	Cumulative total since 1 January	Previous rate ⁴	
AIDS	6	16	0.6	7	19	0.7	
Campylobacteriosis	3304	9011	349.2	2132	6106	230.6	***
Cholera	0	1	0.1	2	2	0.1	
Cryptosporidiosis	379	568	25.5	301	823	33.0	***
Dengue fever	21	60	2.2	64	69	2.0	
Gastroenteritis ⁵	201	729	26.0	320	699	24.8	
Giardiasis	357	1211	42.9	386	1209	42.0	
<i>H influenzae</i> type b disease	1	4	0.1	3	10	0.4	**
Hepatitis A	6	94	3.0	15	44	1.8	***
Hepatitis B (acute) ⁶	17	52	1.7	10	43	1.5	
Hepatitis C (acute) ⁶	18	45	1.6	20	46	1.6	
Hydatid disease	1	1	0.1	2	4	0.2	
Influenza ⁷	469	675	18.6	425	645	20.0	
Lead absorption	24	73	2.5	36	108	3.9	***
Legionellosis ⁷	15	38	1.3	6	46	2.0	*
Leprosy	1	2	0.1	1	3	0.1	
Leptospirosis	34	111	3.7	26	78	2.6	**
Listeriosis	6	13	0.5	6	13	0.4	
Malaria	15	53	1.7	9	43	2.5	*
Measles	7	21	1.5	18	48	1.5	
Meningococcal disease ⁸	229	456	17.2	218	461	15.7	
Mumps	14	45	1.4	24	50	1.7	
Paratyphoid	4	13	0.6	10	24	0.9	
Pertussis	290	785	26.1	234	1143	68.7	***
Rheumatic fever	17	70	2.1	38	109	3.8	***
Rickettsial disease	4	6	0.2	3	3	0.1	
Rubella	7	30	0.9	12	26	0.9	
Salmonellosis	312	1486	59.7	560	1672	58.2	
Shigellosis	24	91	3.0	42	136	4.4	**
Tetanus	0	1	0.1	0	3	0.1	
Tuberculosis	109	272	10.1	82	269	9.6	
Typhoid	2	19	0.7	4	20	0.7	
VTEC/STEC infection	19	60	1.9	22	64	2.1	
Yersiniosis	85	350	12.9	89	298	10.2	***

Notes: 1 Data on surveillance of Creutzfeldt-Jacob disease have been removed from the quarterly surveillance data tables, and replaced by rickettsial disease surveillance data. Annual summary statistics for Creutzfeldt-Jacob disease will be available in the Annual Surveillance Summary.
 2 Other notifiable infectious diseases reported in July to September 2002: Nil
 3 These data are provisional
 4 Rate is based on the cumulative total for the current year (12 months up to and including September 2002) or the previous year (12 months up to and including September 2001), expressed as cases per 100 000
 5 Cases of gastroenteritis from a common source or foodborne intoxication eg, staphylococcal intoxication or toxic shellfish poisoning
 6 Only acute cases of this disease are currently notifiable
 7 Surveillance data based on laboratory-reported cases only
 8 Totals and rates are based on the EpiSurv report date as opposed to the earliest available date used in the meningococcal disease section
 9 Percentage rate change is the difference between the number of cases in the current year (12 months up to and including September 2002) and the previous year (12 months up to and including September 2001). This difference is expressed as a percentage of the number of cases in the previous year

Surveillance data

Surveillance data by health district - July to September 2002 quarter

Cases this quarter Current rate²

Disease	Cases for July to September 2002, ² and current rate ^{1,2} by health district ^{3,4,5}																							
	Northland	NW Auck	Central Auck	South Auck	Waikato	Tauranga	Eastern BOP	Gisborne	Rotorua	Taupo	Taranaki	Ruapehu	Hawkes Bay	Wanganui	Manawatu	Wairarapa	Wellington	Hutt	Nelson-Marl	West Coast	Canterbury	South Cant	Otago	Southland
AIDS ⁴	0	5	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Campylobacteriosis	89	495	467	330	303	78	16	27	45	18	90	3	142	40	53	20	313	105	49	28	268	88	147	89
Cholera	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Cryptosporidiosis	5	11	10	9	66	7	1	3	3	8	24	1	19	2	29	2	93	8	3	3	31	15	14	11
Dengue fever	0	0	6	5	2	1	0	0	0	0	1	0	0	0	1	0	1	0	0	0	0	3	0	1
Gastroenteritis	10	23	20	9	14	0	0	1	1	1	6	0	2	9	0	3	21	13	1	1	53	3	3	7
Giardiasis	5	41	61	24	38	14	0	0	3	3	3	0	20	7	9	2	35	28	4	3	35	3	16	3
H influenzae type b disease	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0
Hepatitis A	0	1	2	1	0	0	0	0	0	1	0	0	0	0	1	0	0	0	0	0	0	0	0	0
Hepatitis B	0	3	1	1	1	3	0	3	0	1	1	1	0	1	0	0	1	0	0	0	0	0	0	0
Hepatitis C	0	2	1	2	0	3	0	0	1	0	0	0	1	0	1	1	2	0	0	0	3	1	0	0
Hydatids	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Lead absorption	3	3	2	0	1	0	0	0	0	0	2	0	1	1	3	1	0	0	0	0	4	1	2	0
Legionellosis ⁶	1	1	1	0	0	1	0	0	0	0	1	1	0	0	0	1	1	1	0	0	3	0	3	0
Leprosy	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Leptospirosis	3	1	0	1	7	2	0	0	0	0	2	0	7	2	2	0	0	0	2	0	1	2	1	1
Listeriosis	0	1	0	1	0	2	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	1
Malaria	0	2	1	2	2	2	0	0	0	0	0	0	1	0	0	0	4	1	0	0	0	0	0	0
Measles	0	2	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	4	1	0	0	0
Meningococcal disease ⁷	12	9	13	30	19	13	7	3	12	10	3	3	13	2	5	1	15	7	2	5	11	3	23	8
Mumps	0	2	1	1	0	1	0	0	0	0	0	0	3	0	0	0	2	0	0	0	2	1	1	0
Paratyphoid	0	2	0	0	0	0	0	0	0	0	0	0	1	0	0	0	1	0	0	0	0	0	0	0
Pertussis	0	16	5	7	17	0	1	0	0	0	3	0	2	1	5	2	12	12	14	17	84	82	2	8
Rheumatic fever	3	1	1	2	3	1	0	0	2	0	0	0	0	0	0	0	3	1	0	0	0	0	0	0
Rickettsial disease	0	4	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Rubella	0	0	1	0	0	0	2	0	0	0	0	0	0	0	0	1	1	0	0	0	1	0	1	0
Salmonellosis	8	36	26	25	34	3	2	1	4	4	9	1	15	5	7	5	27	5	7	3	27	11	22	25
Shigellosis	1	2	6	5	0	1	0	0	2	0	0	0	0	0	0	0	0	0	0	0	6	0	1	0
Tetanus	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0
Tuberculosis	1	19	21	15	10	3	0	2	0	1	0	0	9	0	2	0	9	5	0	1	5	1	4	1
Typhoid	0	0	0	2	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
VTEC/STEC infection	0	0	0	3	3	0	3	0	0	1	3	0	1	0	0	0	0	0	0	0	4	1	0	0
Yersiniosis	0	8	12	9	8	4	0	1	0	1	1	0	2	1	0	1	14	3	1	2	9	4	3	1

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

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

3 These data are provisional

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

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

6 Surveillance data based on laboratory-reported cases only

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

Finnish study estimates 35% of asthma is attributable to work place mould exposure

A population based incident case control study assessed the effects of indoor dampness and mould, at work and home, on the development of asthma in adults in Finland. Cases (521 adults) were clinically diagnosed asthma patients recruited from all health care facilities in the area. 932 controls were selected from the national population registry. Exposure was assessed by a questionnaire inquiring about water damage, damp stains and other marks of structural dampness, visible mould and mould odour both at home and indoors at work. The study found that the risk of asthma was related to the presence of visible mould and/or mould odour in the workplace (Odds Ratio [OR] 1.54, 95% CI 1.01-2.32) but not to water damage or damp stains alone. Confounding was taken account of by adjusting for sex, age, parental atopy, education, smoking, environmental tobacco smoke, and pets. They also found that women, the young (20-29 years), and smokers were more susceptible to the effects of workplace mould. From this study, they

estimated the fraction of asthma attributable to workplace mould exposure to be 35% among the exposed (Jaakkola MS, Nordman H, Piipari R, et al. Indoor dampness and molds and development of adult-onset asthma: a population based incident case control study. *Environ Health Perspect* 2002; 110: 543-7).

Editorial Note: There has not been a population based case control study assessing occupational asthma in New Zealand. However, there have been studies assessing asthma risks in high risk occupations like saw milling and welding where working in these occupations was associated with an increased prevalence of asthma. Other occupations where workers are more likely to develop asthma include laboratory work, baking, chemistry, and seafood work. The most common agents causing occupational asthma in New Zealand are understood to be isocyanate paints, foams and plastics, animal fur and proteins, flour and grain dusts and epoxy resins and other plastics.

NLV's found to be leading cause of non bacterial gastroenteritis outbreaks in the US.

This CDC study assessed laboratory and epidemiologic data from outbreaks between July 1997 and June 2000 of non-bacterial gastroenteritis in the US. 93% (217) of the 233 reported non bacterial outbreaks were positive for NLV. The most common settings reported were restaurants and events with catered meals (39%), followed by nursing homes and hospitals (25%). Contaminated food (57%) was the most commonly reported vehicle of infection, followed by person to person (16%) and contaminated water (3%). For 24% of the outbreaks, mode of transmission was unknown. The median number of people affected were 40 and the mean age was 43 years old (range two months to 104 years). During this study, 213 strains of NLV were detected. Genogroup II (GI) strain was the predominant type (73%) and Genogroup I caused 26% of the NLV outbreaks. An interesting finding was that none of the food borne outbreaks were related to the consumption of contaminated oysters (Fankhauser RL, Monroe SS, Noel JS et al.

Epidemiologic and molecular trends of "Norwalk-like viruses associated with outbreaks of gastroenteritis in the United States. *J Infect Dis* 2002; 186: 1-7).

Editorial note: In New Zealand, NLV is also the most common identified cause of reported non-bacterial gastroenteritis outbreaks. This agent accounted for 115 outbreaks for the same time period as the above study. A New Zealand study (Greening GE, Mirams M, Berke, T. Molecular epidemiology of 'Norwalk-like viruses' associated with gastroenteritis outbreaks in New Zealand. *J Med Virology* 2001; 64:58-66.) analysed 83 reported outbreaks during August 1995 to July 1999, showing similar features to the above US study. The most common settings were restaurants (48%) and rest homes (29%). Almost half (49%) of the outbreaks were associated with food or waterborne transmission. The food type implicated most commonly was seafood, especially oysters. 66% of the outbreaks were caused by Genogroup II and 31% by Genogroup I.

Travel health

Escalating problem of dengue

Dengue is the most common arboviral disease world-wide with an estimated 50-100 million cases and 24 000 deaths annually. During the past 60 years, its incidence, distribution, and clinical severity have increased markedly. Population growth in the tropics, uncontrolled urbanisation, failure of prevention programmes, and air travel have all contributed to this emerging problem. The authors of this review conclude that until the main vector, the *Aedes aegypti* mosquito, can be effectively controlled or a cost-effective vaccine developed, dengue can be expected to continue escalating. Travellers to tropical countries can be advised to take precautions against mosquito bites, including use of protective clothing and insect repellents which should be used in the early morning and late afternoon when *Aedes* mosquitoes are most active. Dengue is a relatively common cause of fever in travellers returning from the tropics. (Gibbons RV, Vaughn DW. Dengue: an escalating problem. *BMJ* 2002; 324: 1563-6).

Editorial note: New Zealand is affected by this world wide increase in dengue. There were 93 notified cases in 2001, the highest incidence year to date. This pattern looks set to continue with 60 imported cases in the first nine months of 2002. It is important to advise travellers to the Pacific and other tropical regions about precautions to prevent mosquito bites. Because of the potential for dengue fever to become established in New Zealand, it is also crucial that medical practitioners test for the disease in those with suggestive symptoms, and notify the medical officer of health of all suspect cases.

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