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BACTERIOLOGY

INVASIVE INFECTIONS

Numbers of isolates received from cases of invasive disease caused by *Haemophilus influenzae, Neisseria meningitidis, Streptococcus pneumoniae, Streptococcus pyogenes* (Group A), and *Streptococcus agalactiae* (Group B) January to December 1999 are shown in Table 1.

Table 1. Sterile site isolates, 1999

Organism	BC	CSF or CSF/BC	Other sterile site	Total
H. influenzae	40	4	0	44
N. meningitidis	166	73	1	240
S. pneumoniae	437	33	8	478
S. pyogenes	96	2	9	107
S. agalactiae	30	2	3	35

H. influenzae: includes 9 serotype b

The age profile of the patients from whom the isolates were obtained is given in Table 2.

Table 2. Age distribution of cases of invasive disease, 1999

Organism	<1m	1-11m	1y	2y	3y	4y	5-9y	10-24y	25-59y	≥60y
H. influenzae b	0	2	2	1	0	0	0	0	3	1
H. influenzae non b	1	4	0	1	0	1	2	3	13	10
N. meningitidis	1	47	43	18	14	15	23	50	23	6
S. pneumoniae	2	51	59	28	12	6	18	22	98	181
S. pyogenes[1]	1	9	0	1	1	2	4	11	33	43
S. agalactiae[1]	12	5	0	0	0	0	0	1	10	7

[1] Information on age was not provided with one isolate of *S. pneumoniae* and two isolates of *S. pyogenes*.

Haemophilus influenzae

Haemophilus influenzae serotype b (Hib) is a notifiable disease. Since vaccination against this disease became available it has become particularly important to identify the serotypes of *H. influenzae* that are causing invasive disease since the notification is dependent on laboratory evidence. Laboratory data is matched with notification data to ensure that this is as accurate as possible.

Isolates were received from 44 cases of *H. influenzae* invasive disease in 1999. Nine of these isolates were serotype b, two were serotype e, five were serotype f, and the others were non-serotypable using serotype-specific antisera. This compares with 10 serotype b out of a total of 41 viable organisms in 1998.

All organisms that were non-serotypable were tested by PCR for the presence of the serotype b specific *cap* gene and the *bexA* gene necessary for capsular expression. Only one of these isolates possessed the *bexA* gene and none the serotype b specific *cap* gene.

The antimicrobial susceptibilities of these isolates are reported in the Antibiotic Resistance section of this issue of LabLink.

Neisseria meningitidis

Culture-confirmed cases of meningococcal disease are those from whom a meningococcus has been isolated from a sterile site. Meningococci received at ESR from cases are serogrouped and then serotyped and subtyped using monoclonal antibodies prepared against the following serotypes and subtypes:

serotype 1, 2a, 2b, 4, 14 and 15

subtypes P1.1, P1.2, P1.4, P1.5, P1.6, P1.7, P1.9, P1.10, P1.12, P1.13, P.14, P1.15 and P 1.16.

Subtyping by amplification of the *porA* gene is undertaken on organisms which are not serosubtypable with monoclonal antibodies. The *porA* gene encodes subtype-specific antigens. Restriction digestion of the PCR product enables prediction of the subtype, which is then confirmed only for subtypes P1.2, P1.4, P1.7 and P1.16 by DNA–DNA hybridisation with sequence-specific probes.

Confirmation of meningococcal disease can also be made by demonstration of meningococcal DNA in specimens of blood, CSF or tissue aspirates. The same *porA* PCR test is used to detect meningococcal DNA directly in patient specimens. It should be noted that on meningococci, the P1.7 epitope is inaccessible to monoclonal antibodies and goes unrecognised whereas the sequence-specific probes are able to detect sequences encoding this additional epitope. Thus, the subtype P1.4 detected by monoclonal antibodies equates with the sequence-specific subtype P1.7, 4 detected by PCR in patient specimens.

A total of 240 isolates were received from culture-confirmed cases of meningococcal disease. One isolate was non-viable on receipt. Of the 240 isolates, 92.9 % (223/240) were serogroup B, 5.8 % (14/240) serogroup C and 3 were serogroup Y. This compares with 217 viable invasive isolates in 1998, 88 % of which were serogroup B. In addition, 16 isolates were received from non-invasive sites from notified cases.

Among the serogroup B isolates received, one strain type, B:4:P1.4, accounted for 73.0 % (162/222) and one subtype, B:P1.4, accounted for 90.6% (202/223). This compares with 69.1% and 88.0% respectively for 1998. Organisms with this serosubtype have been identified from the majority of cases of meningococcal disease since the beginning of the current epidemic in mid-1991. One non-viable organism was able to be serogrouped as a B and subtyped as P1.7,4 by PCR testing. The serotype was not determined. The serogroup Y isolates typed as Y:14:NST, Y:14:P1.2 and Y:14:P1.5,2

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VIROLOGY

Table 21 summarises viral identification and mycoplasma infections in New Zealand in 1999. The information is based on weekly data collated from the virology laboratories of Auckland Healthcare, Healthcare Waikato, Canterbury Health Laboratories, Healthcare Otago, and ESR.

Table 21. Summary of virus identification and mycoplasma notifications, 1999

	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec	Total
Influenza A H3N2	2	2	2	0	103	152	135	54	3	0	0	0	453
Influenza A H1N1	0	0	0	0	0	0	1	1	0	0	0	0	2
FluA not subtyped	1	0	3	10	21	57	62	37	2	3	3	2	201
Influenza B	0	0	0	2	13	5	10	81	35	9	4	1	160
Parainfluenza 1	0	0	0	0	0	0	0	1	0	0	0	0	1
Parainfluenza 2	0	0	0	0	0	0	1	1	0	0	0	0	2
Parainfluenza 3	1	1	6	0	3	8	21	40	20	15	5	2	122
RSV	1	4	4	5	30	162	179	289	128	42	9	5	858
Rhino	1	0	3	1	4	6	28	9	4	23	31	3	113
Mycoplasma	2	3	0	3	6	0	1	4	0	0	0	2	21
CMV	0	2	0	2	0	4	0	1	0	4	1	1	15
Varicella zoster	2	1	7	3	2	6	5	7	5	5	4	7	54
Rubella	0	0	0	0	0	0	0	0	0	0	0	0	0
Mumps	1	1	1	0	0	0	1	0	0	0	0	0	4
Measles	0	0	1	0	1	0	0	0	0	0	0	0	2
Adeno	9	5	5	6	2	7	5	24	18	11	8	5	105
Adeno type 1	2	0	0	4	1	1	2	1	3	2	1	1	18
Adeno type 2	2	2	0	0	1	1	2	0	3	1	1	3	16
Adeno type 3	19	3	2	2	6	2	4	1	1	2	1	0	43
Adeno type 4	0	0	1	0	0	0	0	0	0	0	3	1	5
Adeno type 5	0	1	0	0	0	0	0	1	2	1	1	1	7
Adeno type 11	1	2	0	0	0	1	0	1	0	0	0	0	5
Adeno type 13	0	0	3	0	0	0	0	0	0	0	0	0	3
Adeno type 14	0	1	1	0	0	0	0	0	0	0	0	0	2
Adeno type 15	0	0	0	1	1	0	0	0	0	0	0	0	2
Adeno type 21		0	0	0	0	0	0	0	1	1	0	0	2
Untypable adeno		0	0	3	2	0	0	0	2	0	0	0	7
Entero	1	2	5	3	3	3	1	6	6	7	10	8	55
Polio 1	0	0	1	1	1	1	2	3	5	4	2	2	22
Polio 2	0	0	1	0	1	0	3	1	2	0	3	2	13
Polio 3	0	0	0	0	0	0	2	0	0	1	0	0	3
Coxsackie A9	0	0	0	0	0	0	1	0	0	0	0	0	1
Coxsackie A16	0	0	0	4	2	2	6	2	0	0	0	0	16
Coxsackie B4	0	0	0	0	0	0	1	0	1	1	2	0	5
Echo 3	0	0	0	0	1	0	0	1	0	0	1	0	3
Echo 6	0	0	0	0	0	0	0	1	0	1	0	0	2
Echo 7	0	0	0	0	0	0	0	0	0	0	0	1	1
Echo 9	0	0	0	0	0	0	1	1	2	0	1	4	9
Echo 11	2	1	1	0	0	1	0	0	0	0	0	0	5
Echo 21	0	0	0	1	0	0	0	0	0	0	0	0	1
Echo 30	0	1	0	0	0	0	1	0	0	0	0	0	2
Entero 71	0	0	0	0	1	0	0	0	0	0	0	0	1
Untypable entero	0	1	1	0	1	1	8	3	0	2	0	3	20

RESPIRATORY VIRUSES

Influenza

The 1999 influenza activity has been higher than 1997 and 1998 (Figure 10 and 11). Through the sentinel surveillance programme, a total of 425 influenza isolates were identified. There were 264 (62.1%) isolations of A(H3N2). This strain predominated in New Zealand from May to the end of August, causing several outbreaks in Auckland, Waikato, Christchurch and Wellington, and was associated with severe disease in all age groups. All A (H3N2) isolates were antigenically similar to the A/Sydney/5/97. Again, similar to last year, two major groups of A/Sydney/5/97 were identified: one which showed a typical reaction with A/Sydney/5/97) antisera and the other showing a reduced reaction to this antiserum (a reduction of four-fold or more). This latter group of low reacting strains constituted an even greater proportion of isolates (45%) in 1999 compared with that of 1998 (30%).

There have been sporadic cases of influenza A(H1N1) isolated in the Americas, Asia and Oceania in 1999. These A(H1N1) viruses were related either to the A/Bayern/7/95-like lineage or A/Beijing/262/95-like lineage, the two distinct lines of influenza A(H1N1) circulating in recent years. A/Beijing/262/95 viruses were initially restricted in Asia, but have spread to Oceania this year. An outbreak in New Caledonia in May-June 1999 was caused by viruses of the A/Beijing/262/95 lineage. There were two cases of influenza A(H1N1) isolated in New Zealand this year. The first A(H1N1) isolate was designated as A/Auckland/176/99. It was isolated in Auckland through the sentinel surveillance programme. A nasal swab was taken on 6 July from a 6 year old female who had a flu-like illness. The WHO collaborating centre in Melbourne has shown that it is more closely related to A/Beijing/262/95 viruses than A/Bayern/7/95 viruses. This is the first time a virus of A/Beijing/262/95 lineage has been isolated in New Zealand. The second A(H1N1) isolate was designated as A/Waikato/86/99. It was isolated through the hospital surveillance programme (also referred as the non-surveillance programme). A throat swab was taken on 23 August from a 10 year old female. The ESR virology laboratory has shown that this isolate was more closely related to the A/Beijing/262/95 virus than the A/Bayern/7/95 virus.

The majority of influenza B viruses world-wide were antigenically related to the B/Beijing/184/93 reference strain. In New Zealand, all 84 (19.2% of total isolates) laboratory-confirmed influenza B viruses were related to B/Beijing/184/93 and influenza B became the predominant type from the end of August.

Based on all epideomiology data from the southern hemisphere countries such as Australia, South Africa and New Zealand, the Australian Influenza Vaccine Committee, with a New Zealand representative, met on 7 October and 9 November 1999 to consult on the influenza vaccine composition for 2000. The recommended composition was:

- A(H1N1) an A/New Caledonia/20/99-like strain
- A(H3N2) an A/Sydney/5/97-like strain
- B a B/Beijing/184/93-like strain

Figure 10. Laboratory- confirmed influenza isolates, January 1995 - September 1999

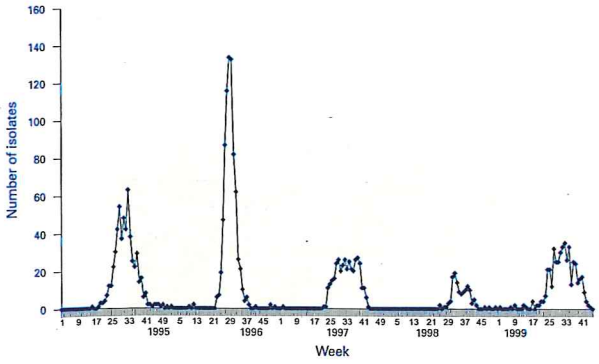
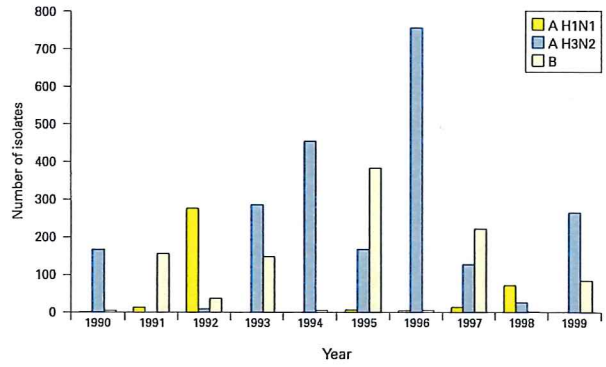


Figure 11: Laboratory-confirmed influenza isolates by type, 1990-99



Respiratory Syncytial Virus (RSV)

The 1999 RSV outbreak was the highest (858 cases) since 1990 (Figures 12 and 13). It had an early onset in the middle of June (31 cases in week 24), four weeks earlier than that of 1998. RSV activity remained at a high level through June, July, August and the end of September, showing a broader and shorter peak than 1998. The largest number of cases was reported at the end of August with 73 cases in the 34th week which was three weeks later than 1998 (90 cases in the 31st week). The number of reported cases declined rapidly around the middle of October.

Figure 12. Annual laboratory-confirmed RSV cases, 1990-1999

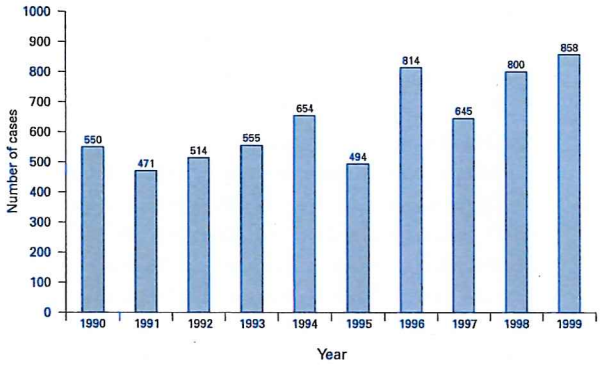
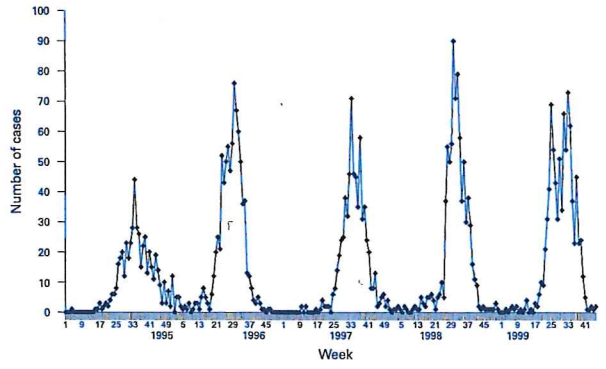


Figure 13. RSV laboratory-confirmed cases by week, 1994-1999



ENTEROVIRUSES

Pericarditis outbreak in Wairarapa

Six cases of pericarditis in Wairarapa health district were identified during the first three weeks of November 1999. The diagnosis in each case was based on clinical presentation and abnormalities present on ECG and echocardiograms. Faecal and throat specimens were taken from two cases for virological investigation. Coxsackie B4 was isolated and identified from faeces from an 18-year-old male. No virus was detected from the other case. Virological investigation was not performed on the remaining four cases. The cases had a median age of 36.5 years, five were male and one female. Three were admitted to Masterton Hospital for 1-3 days.

Pericarditis is uncommon in New Zealand, with an admission rate of 2.6/100,000 per annum for 1996-98 inclusive. Wairarapa would expect to admit one case per year, if following the New Zealand trend.

A review in England and Wales[1] found group B coxsackievirus to be the most common cause of infective pericarditis and myocarditis, followed by influenza virus, mycoplasma, chlamydia and *Mycobacterium* tuberculosis. Group B coxsackievirus is an enterovirus, transmitted by faecal-oral or respiratory droplet spread, with an incubation time of 3-5 days.[2] It has been estimated that about 5% of all symptomatic coxsackievirus infections induce heart disease[3]. The virus may affect the endocardium, pericardium, myocardium, or all three. After acute coxsackie carditis, lasting heart damage has been reported with virus persistence in diseased tissue.[3]

Outbreaks of viral myocarditis have been reported in Malaysia. Although no common source of infection was detected for the Wairarapa cases, this outbreak serves as a reminder that pericarditis and myocarditis often have an infectious origin and appropriate testing should be performed to identify the aetiological agent. Pericarditis and myocarditis should be suspected in any patient with sharp retrosternal chest pain. Individuals diagnosed with pericarditis or myocarditis should have a throat swab and faecal specimen tested for enteroviruses, appropriate testing for mycobacteria and a rheumatology screen.

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1. Fairley CK, Ryan M, Wall PG, Weinberg J. The organisms reported to cause infective myocarditis and pericarditis in England and Wales. *Journal of Infection* 1996; 32:223-225.
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3. Melnick, JL. Enteroviruses: Polioviruses, Coxsackieviruses, Echoviruses, and Newer Enteroviruses. Fields Virology. pp. 655-712. Third Edition. Edited by BN Fields, DM Knipe, PM Howley et al. Lippincott-Raven Publishers, Philadelphia 1996.

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