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BACTERIOLOGY

INVASIVE INFECTIONS

Antimicrobial susceptibilities of these bacteria are reported in the Antibiotic Resistance section of this issue of Lablink.

Haemophilus influenzae

A total of 23 isolates of *H influenzae* were received from sterile site specimens during 2002. Only three were serotype b and all three were from adult cases. This is the first year that no Hib isolates have been received from vaccine aged children. However, it should be noted that 9/23 (39%) non-serotype b isolates were from children under the age of four years. Only one of the nine had a capsule (type f). The other eight were negative by PCR for the *bex* gene encoding capsular expression and all nine were negative for the *cap* gene encoding serotype b expression.

Neisseria meningitidis

The increase in disease rates since 1991 has largely been attributable to serogroup B meningococci expressing the PorA P1.7b,4 protein. Serogroup B disease, particularly that caused by the epidemic type, continued to dominate in 2002. Of the isolates obtained from cases 83.6% (188/225) were serogroup B and of these 173 (92%) were the epidemic type. Although case numbers were higher in 2001 (282), a comparable proportion of isolates were B with the PorA subtype P1.7b,4 (92.9%; 262/282). In fact, since 1995 greater than 85% of all serogroup B disease has been caused by this type. The complete dominance of this type with respect to all disease isolates, regardless of serogroup, is demonstrated in Figure 1.

Since 1991 the proportion of cases caused by serogroup C has varied in relation to serogroup B. W135 and Y serogroups are rarely identified in New Zealand (Figure 1). During 2000, only 10 (3.9%) cases were caused by serogroup C. In 2001, a small resurgence of cases caused by serogroup C occurred particularly in the Otago Health District, and a total of 30 cases (9.4%) were recorded. This increase was sustained in 2002, with 35 (15.5%) serogroup C cases identified throughout New Zealand. Thirteen were from the Otago District Health Board giving a rate for that area of 7.6 per 100 000. Only one case with serogroup W135 and one with serogroup Y was identified in 2002.

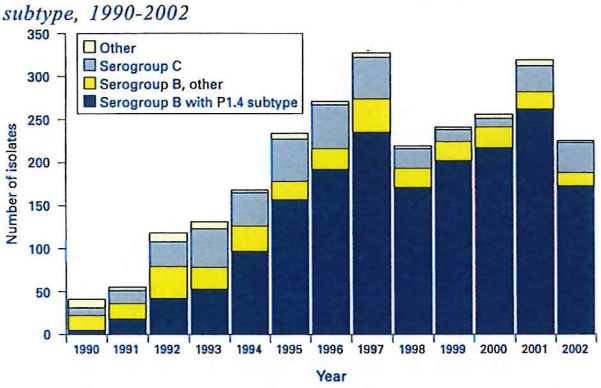
A total of 177 cases were confirmed only by a positive PCR test performed either at a regional hospital or at ESR. Of these, DNA was available for genotyping at ESR for 172. Genotyping using the *siaD* PCR showed that 147 (85.5%) encoded the group B capsular polysaccharide and 10 (5.8%) the C polysaccharide. A further 15 were unable to be defined using the *siaD* PCR. Of those genotyping as group B, 84.4% (124/147) were shown to have DNA encoding the P1.7,4 PorA type.

Consistency in PCR results was demonstrated for 36 cases where both an isolate and DNA from PCR testing was available. In all 36 instances the culture result confirmed the serogroup and/or PorA type obtained by genotyping of the DNA. Twenty-four (66.7%) were B with the P1.7b,4 PorA subtype and 12 were group C's where the PorA was either P1.5 or undefined.

By combining the sero-subtyping results for isolates (n = 225) and the *porA* genotyping results on DNA from PCR positive specimens (n= 159) it was shown that a total of 297 [173 +124] cases out of 384 (77.3%) were caused by meningococci with the P1.7b,4 PorA protein. This is marginally less than the proportion in 2001 when subtype P1.7b,4 was responsible for 80.5% (372/462) cases.

The overall estimate of the number of cases in 2002 attributable to meningococci with the P1.7b,4 PorA protein is 431 (77.3% of 557) which would give a rate of disease of 11.6 per 100 000. This estimate is based on the assumption that all confirmed and probable cases that were reported in 2002 were actual cases of meningococcal disease and that the organism distribution among probable cases was similar to that among confirmed cases.

Figure 1: Meningococcal disease isolate serogroup and dominant subtype, 1990-2002



Meningococci associated with fatal cases

Sixteen of the 18 cases of meningococcal disease who died were confirmed, 10 by isolation of *N. meningitidis* from a sterile site prior to, or at death, and six by PCR of a sterile site specimen. All five serogroup B isolates were B:4: P1.4 (epidemic strain) and four of the remaining five were C:2a P1.5. The fifth typed as C:1:P1.6. Of the six positive by PCR, four genotyped with sequences indicating they were B:P1.7,4; one typed as a C, and one was undefined.

Serogroup C outbreak

In September, a small outbreak of serogroup C disease occurred among school pupils in South Otago. The first case admitted on

Table 1. Serologic distribution of meningococcal disease isolates, 2002

Serogroup	Serotype	Subtype	Number	Percentage of total with serologic phenotype
B	4	P1.4	146	
B	14	P1.4	5	
B	NT	P1.4	20	
B	other	P1.4	2	
Total B:P1.4			**173**	**76.9**
B	4 or other	Not P1.4	15	6.7
Total B			**188**	**83.6**
C	2a	P1.5,2 or P1.2	15	
C	2a	NST	5	
C	NT	P1.5,2 or P1.2	3	
C	NT	NST	7	
C	Other	Not P1.5,2	5	
Total C			**35**	**15.6**
Total W135			**1**	**0.4**
Total Y			**1**	**0.4**
Total isolates			**225***	**100**

NT = Non-typable
NST = Not serosubtypable
* Does not include four isolates not submitted to ESR

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antimicrobials tested except cephalothin, and was isolated from a child who had recently been in China.

In 2002, 23 *S.* Typhi, 3 *S.* Paratyphi A and 23 *S.* Paratyphi B isolates were referred to ESR. They were tested for susceptibility to the same 10 antimicrobials as the non-typhoidal *Salmonella* (Table 16). All the *S.* Typhi isolates were fully susceptible, except for one isolate that was streptomycin resistant. The three *S.* Paratyphi A isolates were fully susceptible. Over a third (9, 39.1%) of the *S.* Paratyphi B isolates were resistant, with six being multiresistant to ampicillin, chloramphenicol, streptomycin, sulphonamides and tetracycline. However, three of these six resistant isolates were part of an outbreak.

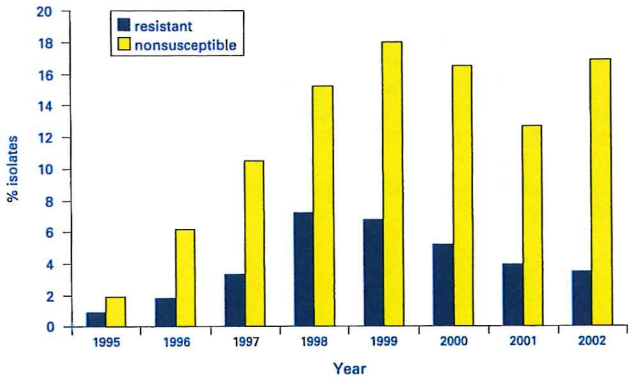
ANTIBIOTIC SUSCEPTIBILITIES OF INVASIVE PATHOGENS

These data on the antimicrobial susceptibility of isolates recovered from cases of pneumococcal, meningococcal, and *Haemophilus influenzae* invasive disease are based on isolates referred to ESR as part of the laboratory-based surveillance of these diseases. The antimicrobial susceptibility of all viable invasive isolates of these three organisms referred in 2002 was tested.

Streptococcus pneumoniae

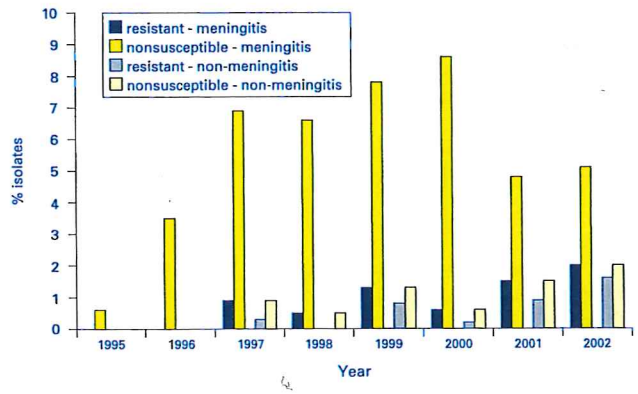
Among the 490 invasive pneumococcal isolates tested in 2002, 16.9% (83) were categorised as penicillin nonsusceptible (MIC ≥0.12 mg/L): 3.5% (17) as resistant (MIC ≥2 mg/L) and 13.5% (66) as intermediate (MIC 0.12-1 mg/L). The prevalence of penicillin resistance has decreased in each of the last four years since 1998 (Figure 9). While there was also a decline in penicillin nonsusceptibility between 1999 and 2001, the prevalence increased again in 2002.

Figure 9. Penicillin resistance and nonsusceptibility among pneumococci from invasive disease, 1995-2002



The NCCLS interpretive standards for pneumococcal susceptibility to cefotaxime/ceftriaxone were redefined in 2002, with different criteria depending on whether the isolate is from a meningitis or non-meningitis case [see *LabLink* 2002; 9(1): 9-10]. Applying the meningitis interpretive standards, 5.1% (25) of the 490 invasive isolates were categorised as cefotaxime nonsusceptible (MIC >1 mg/L): 2.0% (10) as resistant (MIC ≥2 mg/L) and 3.1% (15) as intermediate (MIC 1 mg/L). Applying the non-meningitis interpretive standards, 2.0% (10) were categorised as cefotaxime nonsusceptible (MIC ≥2 mg/L): 1.6% (8) as resistant (MIC ≥4 mg/L) and 0.4% (2) as intermediate (MIC 2 mg/L). Trends in cefotaxime resistance and nonsusceptibility since 1995 are shown in Figure 10. In general, resistance has increased, although nonsusceptibility, based on the meningitis interpretive standards, has decreased since 2000.

Figure 10. Cefotaxime resistance and nonsusceptibility among pneumococci from invasive disease, 1995-2002



The rates of resistance to other antibiotics among the 490 invasive isolates tested in 2002 included 2.5% chloramphenicol resistance, 36.1% co-trimoxazole resistance, 9.0% erythromycin resistance, and 6.9% tetracycline resistance. All isolates were vancomycin susceptible.

The majority of the penicillin-nonsusceptible isolates belonged to the capsular types usually associated with penicillin resistance (Table 17).

Table 17. Distribution of capsular types among penicillin-nonsusceptible and cefotaxime-nonsusceptible invasive pneumococcal isolates, 2002

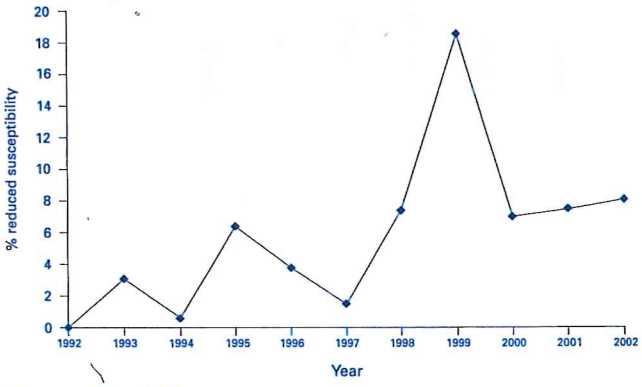
Capsular type	Number (%[1]) isolates			
	Penicillin		Cefotaxime	
	Nonsusceptible MIC ≥0.12 mg/L	Resistant MIC ≥2 mg/L	Nonsusceptible[2] MIC ≥1 mg/L	Resistant[2] MIC ≥2 mg/L
9V	33 (39.8)	2 (11.8)	4 (16.0)	1 (10.0)
19F	16 (19.3)	11 (64.7)	13 (52.0)	9 (90.0)
6B	11 (13.3)	0	3 (12.0)	
23F	11 (13.3)	3 (17.7)	3 (12.0)	
14	6 (7.2)	1 (5.9)	2 (8.0)	
19A	3 (3.6)			
Others	3 (3.6)[3]			
Total	**83 (100)**	**17 (100)**	**25 (100)**	**10 (100)**

[1] Percentage of the nonsusceptible or resistant isolates.
[2] Based on meningitis interpretive standards.
[3] One serotype 6A, one 9N and one 29.

Neisseria meningitidis

In 2002, 223 isolates from cases of invasive meningococcal disease were tested, and all were susceptible to penicillin, ceftriaxone, ciprofloxacin and rifampicin. However, 8.1% (18/223) had reduced penicillin susceptibility, with MICs of 0.12-0.5 mg/L. The proportion of isolates with reduced penicillin susceptibility since 1992 is shown in Figure 11, and shows a trend of increasing prevalence. Until 2002, all isolates with reduced penicillin susceptibility had MICs of 0.12 or 0.25 mg/L. In 2002, one isolate had a penicillin MIC of 0.5 mg/L.

Figure 11. Reduced susceptibility to penicillin among meningococci from invasive disease, 1992-2002



Haemophilus influenzae

Among the 23 invasive *H. influenzae* isolates tested in 2002, eight (34.8%), including two of the total three serotype b isolates, were ampicillin resistant. These eight ampicillin-resistant isolates all produced ß-lactamase. All isolates were susceptible to cefotaxime, chloramphenicol and rifampicin.

VIROLOGY

Table 18 summarises viral identification and mycoplasma infections in New Zealand in 2002. The information is based on weekly data collated from the virology laboratories of Auckland Healthcare, Healthcare Waikato, Canterbury Health Laboratories, Health Otago, Capital Coast Health and ESR.

Table 18. Summary of virus identification and mycoplasma infections, 2002

Year 2002	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec	Total
*Influenza A (not subtyped)	0	1	0	5	42	73	63	26	10	3	0	1	224
*Influenza A H3N2	2	3	1	15	17	75	90	89	31	2	0	0	325
*Influenza A H1N1	1	0	0	0	0	0	1	0	0	0	0	0	2
*Influenza B	0	0	1	0	4	20	26	66	28	5	1	0	151
Parainfluenza 1	1	1	1	3	9	10	11	8	0	0	2	0	46
Parainfluenza 2	0	0	0	1	2	1	0	0	1	0	1	0	6
Parainfluenza 3	0	0	0	0	1	0	2	2	4	13	20	6	48
RSV	2	2	4	3	27	87	242	316	95	32	5	1	816
Rhino	0	2	2	3	7	12	4	10	7	5	10	10	72
Measles	1	0	2	0	1	0	1	0	0	0	1	0	6
Mumps	2	5	3	0	6	1	0	2	1	2	0	0	22
Rubella	0	0	0	0	2	1	1	0	0	0	0	0	4
Varicella Zoster	30	22	26	23	23	20	34	23	18	34	27	26	306
Rotavirus	6	1	5	10	7	8	12	16	23	14	8	4	114
Mycoplasma	64	62	42	41	40	23	24	54	34	10	24	13	431
Adenoviruses	17	18	14	7	25	14	10	25	18	19	24	32	223
Adeno type 1	1	0	0	3	1	1	2	1	2	4	2	7	24
Adeno type 2	2	2	0	0	2	2	0	3	6	1	3	3	24
Adeno type 3	6	7	4	6	9	5	4	14	10	17	7	12	101
Adeno type 4	0	1	0	0	0	0	0	0	0	0	0	0	1
Adeno type 5	0	0	0	0	0	0	0	0	1	0	0	1	2
Adeno type 7	3	1	3	0	3	3	0	0	0	0	0	0	13
Adeno type 8	0	0	0	0	0	1	0	0	3	0	2	0	6
Adeno type 19	0	0	0	0	0	1	0	0	0	0	1	0	2
Adeno type 21	1	0	0	0	0	0	0	0	1	0	0	2	4
Adeno type 22	1	0	0	0	0	0	0	0	0	0	0	0	1
Adeno type 6	0	0	0	0	0	1	0	0	0	0	0	0	1
Adeno type 24	1	0	0	0	0	0	0	0	0	0	0	0	1
Untypable Adenovirus	2	4	5	0	3	2	5	5	4	1	1	0	32
Enteroviruses	126	19	4	3	4	2	5	8	9	12	16	11	219
*Polio 1+2	0	1	0	0	0	0	0	0	0	0	0	0	1
Coxsackie B1	1	0	0	0	0	0	0	0	0	0	0	0	1
Coxsackie B2	0	0	0	0	0	0	0	0	0	2	0	3	5
Coxsackie B3	0	0	0	0	0	0	0	0	0	1	0	0	1
Coxsackie B4	1	1	0	0	3	0	0	0	0	1	0	1	7
CA6	0	0	0	0	0	0	0	2	0	0	0	0	2
CA9	1	0	0	0	1	0	0	0	0	0	0	0	2
CA16	0	0	0	0	0	0	0	0	0	0	0	1	1
Echo 3	0	0	0	0	0	0	0	1	5	0	1	1	8
Echo 6	0	0	0	0	0	0	0	0	0	0	3	4	7
Echo 7	1	1	0	0	0	0	0	0	0	0	0	0	2
Echo 9	0	0	0	0	0	0	0	0	2	0	1	0	3
Echo 25	0	0	0	0	0	0	0	0	0	0	2	0	2
Echo 30	16	4	3	0	0	0	0	0	0	0	0	0	23
*Echo 13	27	1	0	1	0	0	0	0	0	0	0	0	29
Entero 71	0	0	0	0	0	0	1	0	0	0	0	0	1
Untypable Entero	2	2	1	0	1	1	1	1	0	0	1	2	12

* Note: Viruses with sign "*" were reported based on the specimen taken date, whereas other viruses were based on lab reporting date.

RESPIRATORY VIRUSES

Influenza

Influenza activity from January to December 2002 was low to moderate (Figure 12 & 13). A total of 702 influenza isolates from sentinel and non-sentinel surveillance was identified in 2002. Of these, 241 came from sentinel practice surveillance during May to September. This is lower than the 313 sentinel isolates identified in 2001, but more than three times higher than the 73 sentinel isolates identified in 2000. There were 461 non-sentinel isolates identified in 2002 compared to 341 in 2001 and 230 in 2000.

Figure 12. Influenza isolates, 1998-2002

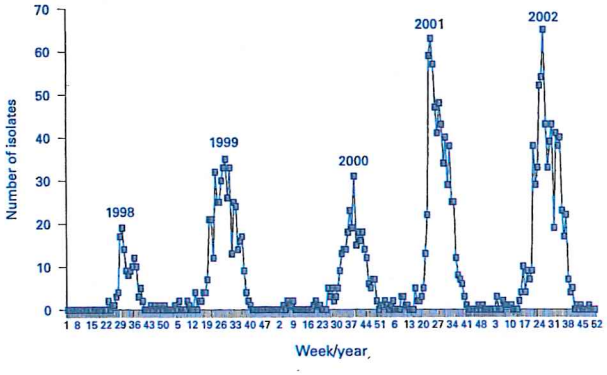
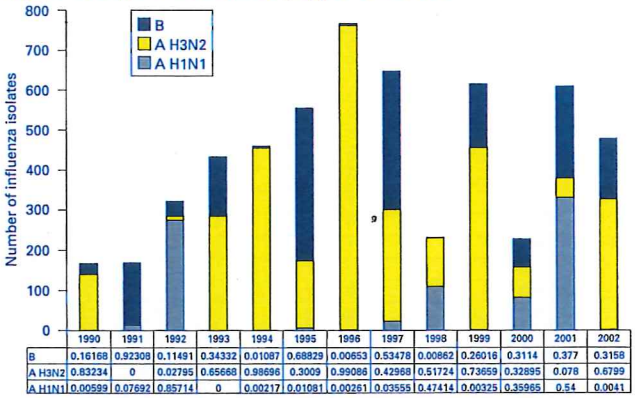


Figure 13. Influenza isolates by type, 1990-2002



	1990	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000	2001	2002
B	0.16168	0.92308	0.11491	0.34332	0.01087	0.68829	0.00653	0.53478	0.00862	0.26016	0.3114	0.377	0.3158
A H3N2	0.83234	0	0.02795	0.65668	0.98696	0.3009	0.99086	0.42968	0.51724	0.73659	0.32895	0.078	0.6799
A H1N1	0.00599	0.07692	0.85714	0	0.00217	0.01081	0.00261	0.03555	0.47414	0.00325	0.35965	0.54	0.0041

Influenza A(H1N1)

In 2002, influenza A(H1N1) represented a very small proportion of isolates, 0.4% (2) of typed and subtyped isolates (478) and 0.3% of all isolates (702). There are two antigenically distinct lines of influenza A(H1N1) circulating the world in recent years and the current reference strains for these are A/New Caledonia/20/99 and A/Bayern/7/95. Influenza A(H1N1) viruses predominated in most regions worldwide during 2002. Viruses of the A/New Caledonia/20/99 lineage have continued to replace A/Bayern/7/99-like strains.

The Australian WHO Collaborating Centre showed that most A(H1N1) isolates from the Southern Hemisphere in 2002, including New Zealand, were A/New Caledonia/20/99. Based on the global data, the WHO Consultative Group concluded that there was no need to change the vaccine strain from an A/New Caledonia/20/99-like virus. Two factors still remain true for the recommendation of A/New Caledonia/20/99-like virus for the year 2003 vaccine formulation:

- Increasing incidence of viruses of this type, and
- The demonstration that, in humans, vaccines containing viruses of this lineage induce similar antibody responses against both the homologous virus and A/Bayern-like strains whereas the converse was not true.

Influenza A(H3N2)

A total of 325 influenza A(H3N2) isolations (68% of typed and subtyped isolates and 46% of all isolates) were obtained in 2002. Influenza A(H3N2) has been frequently associated with severe disease and excess mortality in high-risk groups. This subtype has also shown the greatest tendency for antigenic drift as illustrated by the frequency of vaccine formulation changes recommended by the WHO and the AIVC. The circulating viruses in this subtype fall into a single lineage, although a degree of antigenic heterogeneity is often observed. Influenza A(H3N2) was the predominant subtype in many countries including New Zealand during the past 12 months.

The Australian WHO Collaborating Centre showed that most A(H3N2) isolates from the Southern Hemisphere including New Zealand remain closely related to the A/Moscow/10/99 reference strain and A/Panama/2007/99 vaccine virus. There is evidence of antigenic heterogeneity among the isolates with no single evolutionary lineage at this time. Based on the global data, the WHO Consultative Group concluded that there was currently no pressing need to change from a recommendation for an A/Moscow/10/99-like virus as the A(H3N2) vaccine component for 2003 and there is no obvious new candidate reference strain.

Influenza B

There were 151 isolations of influenza B (35% of all isolates) in 2002. There have been two distinct lines of influenza B circulating in recent years. This dates back to 1990 when the B/Panama/45/90 variant of influenza B arose whilst strains of the previous B/Victoria /2/87-like viruses continued to circulate in Asia. Further variation of the B/Panama/45/90 line gave rise to the B/Beijing/184/93-like viruses. Meanwhile in Asia, independent antigenic evolution of the B/Victoria/2/87-like virus continued and gave rise to the B/Shangdong/7/97-like strains that were prominent in parts of Asia during 1998-9. During the previous 12 months, influenza B viruses co-circulated with influenza A in most parts of the world although levels have been variable. Viruses of the B/Sichuan/379/99 lineage have predominated with only a small number of isolates from the B/Shangdong/7/97 lineage. For reasons not understood these remained geographically restricted to Asia until 2001. In May-June 2001 some isolates of the B/Victoria lineage were found in Hawaii, but not in other non-Asian countries. Further spread of viruses of this lineage then commenced in the 2001-2002 Northern winter and they

progressively became prominent in some countries, particularly in North America. Earlier human vaccination studies had indicated that a virus of this lineage, B/Shangdong/7/97, induced moderate antibody responses to the alternate lineage, whereas the converse was not true. Based on this, the lack of recent exposure to related viruses and apparent emergence of this lineage, a recent virus of this type (B/Hong Kong/330/2001) was recommended for vaccines for the 2002-3 Northern winter. Europe accepted B/Shangdong/7/97 as a B/Hong Kong/330/2001-like strain whereas the USA accepted B/Hong Kong/330/2001 or B/Hong Kong/1434/2002 as suitable vaccine strains.

The Australian WHO Collaborating Centre showed that majority of B isolates (90%) from the Southern Hemisphere in 2002 including New Zealand were B/Hong Kong/330/2001 lineage viruses. There was only one B/Sichuan/379/99-like virus isolated in New Zealand in 2002. Current vaccines containing influenza B/Hong Kong/330/2001 antigen induced anti-HA antibodies to recently isolated viruses, which were of similar titre and frequency to those against the vaccine virus. Based on the global data, the WHO consultation group concluded that vaccines containing a B/Hong Kong/330/2001-like strain for 2003 as the B component.

In summary, Australia Influenza Vaccine Committee, with representatives from New Zealand, Australia and South Africa, agreed to adopt the recommendations made by the WHO consultation group. The recommended composition for 2003 was:

- A(H1N1) an A/New Caledonia/20/99-like strain
- A(H3N2) an A/Moscow/10/99-like strain
- B a B/Hong Kong/330/200/-like strain

Respiratory Syncytial Virus (RSV)

The 2002 RSV activity was at the high level based on laboratory-confirmed RSV cases reported to ESR from 1990 to 2001 (Figure 14). During January to December 2002, a total of 816 RSV infections was reported compared with 565 during the same period in 2001 (Figure 14). The highest RSV activity occurred in 1999 with 858 cases reported.

In 2002, the RSV activity started to increase in May and peaked in Weeks 31 (at the beginning of August), 3 weeks earlier than the peak in 2001 (Figure 15). The RSV activity remained at the high level till Week 37 (early September). Since then, the number of RSV cases has declined to baseline level.

Figure 14. Annual laboratory-confirmed RSV cases, 1990-2002

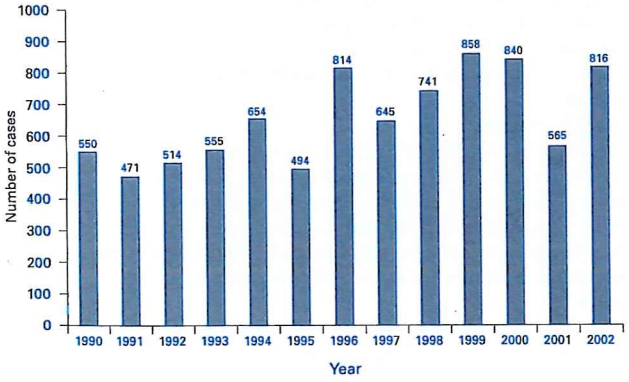
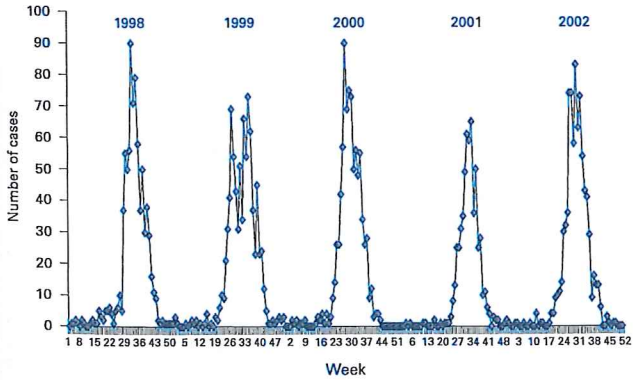


Figure 15. RSV laboratory-confirmed cases by week, 1998-2002



ENTEROVIRUSES

The New Zealand enterovirus laboratory network comprises five laboratories: one public health virology laboratory (ESR, Wellington) and four hospital virology laboratories in Auckland, Christchurch, Waikato and Dunedin. These five virology laboratories cover 100% of the population and all geographical areas of the country. Enterovirus surveillance is a year-round routine diagnostic surveillance for hospital in-patients and outpatients. Hospital laboratories report all enterovirus isolations and/or typing results weekly to ESR and these data are then available nationally. Untyped or untypable enteroviruses are referred to ESR for identification.

There was a total of 219 enterovirus isolations in 2002, compared with 381 in 2001. Echovirus type 13 was the most predominant serotype with 32 isolates from 29 cases (14.6%). There were 23 isolations of Echovirus type 30 (10.5%), compared with 32 in 2001 (8.4%). A total of 7 Coxsackie B type 4 isolations was reported in 2002, compared with 6 isolations in 2001. A total of 8 isolations of Echovirus type 3 and 7 isolations of Echovirus type 6 were reported in 2002, compared with 1 Echovirus type 3 and nil report of Echovirus type 6 in 2001.

Echovirus type 13 in 2001-2002

Note: The Echovirus type 13 (E13) outbreak in 2001 was reported in *LabLink* 2002;9(1):13-14. This report is a summary of the entire E13 outbreak during 2001-2. The E13 isolate details were provided by 5 virology laboratories. Professor Keith Grimwood reviewed the medical notes for 29 confirmed E13 cases from the Wellington region.

- **Epidemiology:**

During a 14-month period from February 2001 to April 2002, a total of 153 E13 isolates from 129 cases (100 in 2001 and 29 in 2002) of mainly aseptic meningitis was identified. These cases were distributed across the Waikato, Auckland, Wellington, Christchurch and Dunedin regions (Figure 16). The outbreak started in February 2001 with the first E13 isolation from faecal and respiratory specimens taken from a 2-month old boy in Waikato. There was a long lag phase in the winter of 2001 with only 2 more cases of E13 reported. The number of cases started to increase in the spring of 2001, peaked in the summer and subsided in the autumn of 2002. The outbreak was initially restricted to Waikato but spread to Auckland, then to the Wellington region and reached Christchurch in week 43 (the end of October). The last E13 isolation was obtained from a CSF specimen taken 15th April 2002 from a 23 day-old infant boy in Wellington. The ages ranged from 10 days to 39 years, with a median of 4 years. Seventy-six were male and 53 were female (ratio M:F = 1.4:1).

E13 is a relatively rare serotype of enterovirus with few reports in the literature. The occurrence of E13 in New Zealand has generally followed this trend with no E13 isolations recorded between 1975 and 2000. However, E13 was the predominant echovirus in 2001 and 2002 accounting for 69.5% (153/220) of all echovirus isolates and 31.5% (153/486) of all enterovirus isolates during Feb 2001 to April 2002. This outbreak was also the largest recorded echovirus outbreak in New Zealand.

- **Virological charaterisation:**

Faecal specimens (43.8%, 67/153), CSF (33.3%, 51/153), respiratory specimens (17.6%, 27/153) and urine (1.3%, 2/153) most readily yielded E33 viruses.

Sequencing of the New Zealand E13 outbreak isolate showed that it is most similar to one found in Australia in 2001 and Germany (DEU) in 2000. Amongst the 2001 outbreak strains of E13 from around the world there is no more than 4.5% nucleotide difference, all of the strains being closely related.

- **Clinical features:**

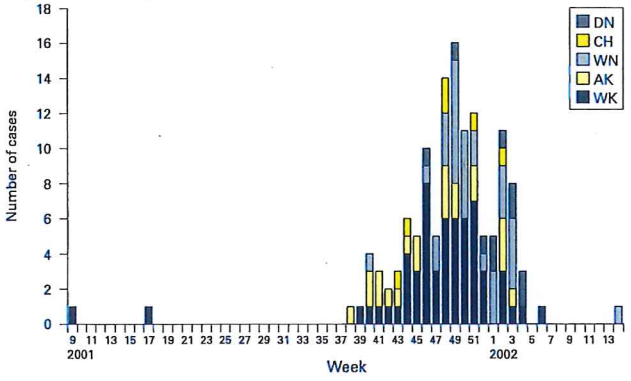
The medical records of the 29 cases (22%, 29/129) from the Wellington region were reviewed. None were immunocompromised and no patient received pleconaril. Overall, all 29 patients were hospitalized and all received 2-7 days of antimicrobial therapy.

Infants ≤ 12-months. Of 8 cases, 5 (62.5%) cases were diagnosed as viral meningitis and 3 as viral infections. The presenting symptoms included rash (morbilliform or petechial; 87.5%, 7/8), vomiting (37.5%, 3/8), fever (100%), stiff neck (12.5%, 1/8), and lethargy (75%, 6/8). Their median hospital stay was 3.5 (range 3-5) days. No infants died due to E13 infection.

Patients > 12-months. Of 21 cases with discharge diagnosis, 20 (95%) had aseptic meningitis and one had bacterial meningitis. The most common complaints were headache (100%), stiff neck (100%), photophobia (86%, 18/21), vomiting (90%, 19/21), lethargy (95%, 20/21) and fever (81%, 17/21). No rash was presented in patients older than 12 months during the medical examinations.

CSF results. The median white blood cell count in the CSF from 28 cases was 197.5 (range 1-1043) x 10^6/L with 50% (14/28)≤ 200 x 10^6/L. Differential cell counts were available for 25 cases; monocytes predominated in 80% (20/25) cases, and neutrophils predominated in 56% (5/25) cases. 69% of cases had CSF protein concentration more than 0.45g/L and 23% with glucose concentration less than 2.5 mmol/L. E13 viruses were isolated from 23/29 (79%) CSF specimens.

Figure 16 . Temporal and geographical distribution of the E13 outbreak



MEASLES, MUMPS AND RUBELLA

Measles, mumps and rubella have been notifiable diseases since June 1996. For demographic and vaccination data on MMR notification cases and hospitalisations, please refer to "Infectious diseases in New Zealand: 2002 annual surveillance summary" produced by ESR. This report focuses on the laboratory-confirmed MMR cases.

Measles

In 2002, a total of 6 laboratory-confirmed measles cases was reported from Canterbury (3), Otago (1), and Waikato (2). Patients ranged in age from 12m (1), 15m (2), 7y (1), 22y (1) and 30y(1).

Mumps

A total of 22 laboratory-confirmed mumps cases was reported from Otago (8), Waikato (3), Wellington (3), Taranaki (2), South Auckland (1), Canterbury (1), Rotorua (1), Wanganui (1), Southland (1) and Nelson-Marlborough (1). Patients ranged in age from 7 month to 71 years (average 29 years). Mumps IgM was positive for 21 cases and a mumps virus was isolated by tissue culture from one case with intrauterine death.

Rubella

A total of 4 laboratory-confirmed rubella cases was reported in 2002. A 16-month girl from Taranaki was positive with rubella IgM presumably due to the recent vaccination. The remaining 3 cases occurred in adults, 18y male from Nelson-Marlborough, 21y female from Canterbury and 28y female from Hawkes Bay.

ADENOVIRUSES

There was a total of 224 adenovirus isolations in 2002, compared with 216 in 2001. The predominant serotypes in 2002 were adenovirus type 3 (101 isolates, 45.1%), adenovirus type 1 (24 isolates, 10.7%), adenovirus type 2 (24 isolates, 10.7%), adenovirus type 7 (13 isolates, 5.8%) and adenovirus type 8 (6 isolates, 2.7%). In comparison, in 2001 there were 21 isolations of adenovirus type 3 (9.7%), 16 of adenovirus type 1 (7.4%), 13 of adenovirus type 2 (6.0%), 10 of adenovirus type 7 (4.6%) and 4 of adenovirus type 8 (1.8%).

Adenovirus type 3

Based on DNA homology, adenovirus type 3 (Ad3) belongs to subgenus B, cluster 1 (B:1) of human adenoviruses (*Adenoviridae* family: *Mastadenovirus* genus). Ad3 accounts for 13% of all adenovirus isolates typed and reported to WHO. It shows an epidemic appearance with 4-5 year intervals. It is most frequently isolated from children below the age of 4 years. Ad3 can cause pharyngitis with an exudative tonsillitis and frequently conjunctivitis, together with nasal congestion and cough. It can also cause laryngotracheobronchitis, but the pneumonias that occur in young children are the most serious clinical manifestations. Ad3 can also cause gastroenteritis, probably a sign of a systemic infection. Adenoviruses may be an infrequent cause of meningitis, and Ad3 and Ad 7 account for two-thirds of all adenovirus-associated cases of meningitis or meningoencephalitis.

A total of 101 Ad3 isolates was reported in 2002, higher than that of 2001 (21), 2000 (17), 1999 (43), 1998 (74), 1997 (29), and 1996 (28). Of 101 isolates, 99 had detailed demographic information. Patients ranged in age from 3 months to 69 years with a median of 13.5 years. Fifty-one were male and 48 were female (ratio M:F=1.06:1). Ad3 was reported from Auckland (46), Canterbury (22), Waikato (14), Otago (6), Tauranga (3), Southland (2), Nelson-Marlborough (2), Eastern Bay of Plenty (1), Gisborne (1), Hawkes Bay (1) and Taupo (1). Of 97 isolates with known specimen information, Ad3 was isolated from eye swabs (54), respiratory specimens (36), faeces (6) and urine (1). Ad3 was isolated from patients whose illness ranged from febrile respiratory illness, pneumonia, conjunctivitis and gastroenteritis. Two encephalitis patients yielded adenovirus type 3 from their faeces.

NOROVIRUS

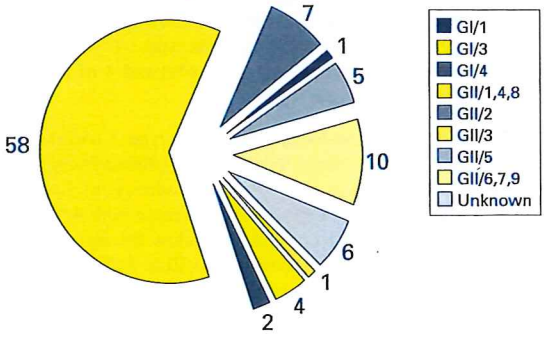
Characterisation of Norovirus strains from gastroenteritis outbreaks occurring in 2002.

In August 2002, the International Committee on Viral Taxonomy reclassified the Norwalk-like virus group into a new Genus *Norovirus* in the Caliciviridae Family.

There were 93 outbreaks or clusters of laboratory-confirmed Norovirus - associated gastroenteritis to December 30 2002. Norovirus was also confirmed in a number of individual cases not known to be linked to any outbreaks. As in 2001, there was no seasonal peak in 2002, with outbreaks being reported during all months but 35.5% of the outbreaks occurred in October, November and December. In 2002 extensive spread of NLVs through institutionalised settings was observed both in New Zealand and overseas. In New Zealand, 34 institutional rest home and hospital outbreaks were reported; the majority of these (29, 85.3%) were caused by the GII/1,4,8 'global strain cluster' and occurred in the winter months. Eight outbreaks occurred in child- related settings. Of these, four occurred in child care centres or commercial children's play centres, two in school camps and two in school hostels. Other settings included restaurants, cafes, takeaway bars and catered functions and several family groups around the country. The extent of Norovirus infection originating in the home is unknown.

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

Figure 17. Norovirus genotypes in 2002 (n=94, 2 strains identified in 1 outbreak)



Editorial Note

This will be last issue of Lablink. A new publication, "interPHace", encompassing the material present in both Lablink and New Zealand Public Health Report will be published in the near future.

ESR LabLink is produced quarterly by the Institute of Environmental Science & Research Limited (ESR), Kenepuru Science Centre, Kenepuru Drive, PO Box 50-348, Porirua, Wellington. Telephone: 04-914 0700, Facsimile: 04-914 0770.

Editorial Team: Philip Carter, Sue Huang, Carolyn Nicol.