INVASIVE PNEUMOCOCCAL DISEASE IN NEW ZEALAND, 2009

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SUMMARY

A 4-dose schedule of the 7-valent pneumococcal conjugate vaccine (PCV-7), Prevenar®, was added to the New Zealand childhood immunisation schedule in June 2008, with a catch-up programme for all children born on or after 1 January 2008. Since 17 October 2008, invasive pneumococcal disease (IPD) has been notifiable to medical officers of health under the Health Act 1956.

In this report, the data presented for 2009 is based on IPD case notifications supplemented with serotype and antimicrobial susceptibility data from ESR's national laboratory-based surveillance of invasive *Streptococcus pneumoniae* isolates. Data for earlier years is solely from ESR's laboratory-based surveillance. For this laboratory-based surveillance, diagnostic microbiology laboratories are requested to refer all invasive isolates of *S. pneumoniae* to ESR for serotyping and antimicrobial susceptibility testing.

There were 697 cases of IPD notified in 2009. A *S. pneumoniae* isolate from an invasive site was received at ESR for serotyping and susceptibility testing for 665 (95.4%) of the notified cases. Between 2007, the year before the introduction of PCV-7, and 2009, the rate of IPD in infants <2 years old halved from 96.2 to 46.4 cases per 100 000. Over the same period, the reduction in the rate of disease due to one of the serotypes in PCV-7 in this age group was more striking, reducing 77% from 78.8 to 18.1 cases per 100 000. Rates of disease caused by PCV-7 serotypes have not decreased in other age groups.

In contrast to the decrease in IPD among <2 year olds, the all-age incidence rate increased from 13.1 cases per 100 000 in 2007 to 16.1 in 2009. It should be noted that the change in 2009 from laboratory-based surveillance of invasive pneumococci to surveillance based on IPD notifications makes absolute comparisons of rates in 2009 with those for earlier years difficult. Compared with notifications, laboratory-based surveillance is likely to underestimate the incidence of IPD. Any such underestimation means that the reductions in rates of IPD among infants <2 years old since 2007 may be greater, and the all-age increase may be less, than the above rate estimates suggest.

In 2009, the all-age rate of pneumococcal meningitis was 1.1 case per 100 000. The highest rate of meningitis occurred in the <1 year age group (14.3 per 100 000). There were no cases of pneumococcal meningitis in the 1-2 year age group. The case-fatality rate was 5.6%.

Rates of IPD in Pacific Peoples and Maori were 3.8 and 3.2 times, respectively, the rate among Europeans. The rate of disease in the most deprived NZDep quintile (9-10) was 3.1 times that in the least deprived quintile (1-2).

42.8% of IPD cases, for whom the information was reported, were recorded as having a chronic illness, 40.0% of cases <5 years of age were exposed to smoking in the household, and 33.3% of cases <1 year of age had been born prematurely.

There were some regional differences in the incidence of IPD in 2009. The rate in the Midland region was significantly higher ($P \leq 0.05$) than that in any other region.

continued

SUMMARY *continued*

There has been some increase in IPD caused by non-PCV-7 serotypes over the last 3 years. This increase has been predominantly due to serotype 1 disease. The incidence of serotype 1 disease was first noted to be increasing in 2007. In 2008 it was confined mainly to older children and young adults, with very few cases in infants. But in 2009 serotype 1 was the most common type among cases <2 years old. 73.9% of all serotype 1 cases in 2009 were in Pacific Peoples or Maori, and the serotype accounted for 50.6% of all IPD cases in Pacific Peoples and 41.4% in Maori. 81.7% of serotype 1 cases were from the Northern and Midland regions.

There has not been an increase in the rate of serotype 19A disease – the non-PCV-7 type that has most commonly increased in other countries following the introduction of the vaccine.

Penicillin and cefotaxime resistance among invasive pneumococci decreased in 2009. Most resistant invasive pneumococci belong to one of the serotypes included in PCV-7, so these decreases in resistance are consistent with the decrease in disease due to vaccine types. 17.7% of isolates were categorised as penicillin resistant according to the CLSI meningitis interpretive criteria. No isolates were categorised as penicillin resistant according to the interpretive criteria for the parenteral treatment of non-meningitis infections. 2.0% of isolates were cefotaxime resistant according to the meningitis interpretive criteria and 0.6% were resistant according to the non-meningitis interpretive criteria. There is no indication that resistance is increasing in non-PCV-7 serotypes, with PCV-7 types still accounting for over 90% of the penicillin and cefotaxime resistance in 2009, as has been the trend in recent years.

While the addition of IPD to the schedule of notifiable diseases has enabled more comprehensive analysis of the epidemiology of this disease in New Zealand, some deficiencies in the notification data were noted. Nearly one-tenth of the IPD cases notified in 2009 did not include evidence that they met the case definition. The immunisation history was poorly reported with case notifications.

1. INTRODUCTION

For many years the national surveillance of invasive pneumococcal disease (IPD) in New Zealand was only laboratory based, with diagnostic laboratories referring invasive isolates of *Streptococcus pneumoniae* to the Institute of Environmental Science and Research Ltd (ESR) for serotyping and antimicrobial susceptibility testing. This laboratory-based surveillance provided information on the basic epidemiology of IPD, and the serotypes and antimicrobial susceptibility of invasive isolates.

Up until 2008, information from the laboratory-based surveillance was published periodically.[1-6] In addition, between 2002 and 2007, annual reports on the antimicrobial susceptibility of isolates from IPD cases have been published on ESR's surveillance website at http://www.surv.esr.cri.nz/antimicrobial/streptococcus_pneumoniae.php.

The first of this series of annual reports on IPD in New Zealand covered IPD in 2008 and was based on data available from national laboratory-based surveillance.[6] On 1 June 2008, the 7-valent pneumococcal conjugate vaccine (PCV-7), Prevenar®, was added to the New Zealand childhood immunisation schedule and IPD became a notifiable disease on 17 October 2008. Consequently 2009 is the first year for which a full year of IPD notification data is available. Therefore, this annual report is based on the cases of IPD notified in 2009 and supplemented with serotype and antimicrobial susceptibility data from ESR's national laboratory-based surveillance of invasive *S. pneumoniae* isolates.

2. METHODS

2.1 Surveillance methods

In this report, data for 2009 is based on IPD case notifications supplemented with serotype and antimicrobial susceptibility data from laboratory-based surveillance of invasive *S. pneumoniae* isolates. Data for earlier years is from ESR's national laboratory-based surveillance of IPD.

Since 17 October 2008, IPD has been notifiable to medical officers of health under the Health Act 1956. Data on each case is entered at public health units (PHUs) via a secure web-based portal onto a computerised database (EpiSurv). The notification data is collated and analysed on behalf of the Ministry of Health by ESR.

For the national laboratory-based surveillance of IPD, diagnostic microbiology laboratories in New Zealand are requested to refer all invasive isolates of *S. pneumoniae* (ie, isolates from cerebrospinal fluid (CSF), blood or other normally sterile site) to ESR. In addition and less frequently, laboratories refer sterile site specimens to ESR to test for the presence of pneumococcal DNA by PCR. At ESR all invasive isolates are serotyped and tested for susceptibility to a range of antibiotics (see Section 2.2).

The notification data in this report is based on the information recorded on EpiSurv as at 5 March 2010. Any changes made to EpiSurv data by PHU staff after this date are not reflected in this report. Serotype and antimicrobial susceptibility data for invasive isolates was matched with the relevant case notification.

Except for disease rates by ethnicity and deprivation index, the mid-year New Zealand population estimates were used to calculate incidence rates for the years 2004, 2005, 2007, 2008 and 2009. The 2006 census population data was used to calculate rates for 2006. The 2006 census population data was used to calculate ethnicity-specific IPD rates, and a prioritised approach was used with the order of prioritisation as: Maori, Pacific Peoples, Other (other groups except European), and European.[7] Incidence rates were not calculated for categories where there were <5 cases.

A deprivation index, which ranges from 1 (least deprived) to 10 (most deprived), is calculated for each geographical mesh block in New Zealand. Approximately equal numbers of people reside in areas associated with each of the ten deprivation levels. The deprivation index analysis was confined to those cases for which the accuracy of index designation was recorded as exact or nearest. The IPD rates by deprivation index were calculated using NZDep2006 data.

In this report, any cases for which *S. pneumoniae* was identified in CSF (by culture, PCR or antigen test) and which were not notified as meningitis cases were considered to be cases of pneumococcal meningitis.

The immunisation status of cases is based on data reported with the case notification; cases were not linked with data in the national immunisation register.

Statistical analyses were performed with SAS software v.9.1.3 (SAS Institute Inc, Cary, NC, USA). The chi-square test or Fisher's exact test, as appropriate, were used to determine the significance of any observed differences. Linear regression was used to calculate the significance and direction of time trends. An associated P value ≤0.05 was used to identify whether a difference or trend was significant.

2.2 Laboratory methods

Detection of pneumococcal DNA in clinical specimens: The presence of pneumococcal DNA in clinical specimens is detected by polymerase chain reaction (PCR).

Strain typing: *S. pneumoniae* isolates are serotyped by the capsular antigen reaction (Neufeld test) using the Danish system of nomenclature and sera obtained from the Statens Serum Institut.[8] Methods have not been established at ESR to identify the strain type when only pneumococcal DNA, rather than an isolate, is available. Therefore, the serotype can only be determined for culture-positive IPD cases.

Antimicrobial susceptibility testing: The penicillin, cefotaxime and moxifloxacin susceptibilities of *S. pneumoniae* isolates are determined by Etest (AB Biodisk, Solna, Sweden), using Mueller-Hinton agar with 5% sheep blood and incubation for 20-24 hours in 5% CO_2. Chloramphenicol, clindamycin, co-trimoxazole, erythromycin, tetracycline and vancomycin susceptibilities are determined by the Clinical and Laboratory Standards Institute's (CLSI's) disc susceptibility testing method.[9] Inducible clindamycin resistance is detected by the D-zone test.[10] All minimum inhibitory concentrations (MICs) and zone of inhibition diameters were interpreted according to the 2009 CLSI standards.[10]

In this report, the penicillin interpretive standards, which were redefined in 2008, have been retrospectively applied to historical MIC data so that time trends are comparable. Also, in this report, when associations between penicillin or cefotaxime resistance and patient demographics, geographical distribution or serotypes are made, the meningitis interpretive standards have been used.

Multidrug resistance is defined as resistance to three antibiotics in addition to penicillin. For the purposes of this definition, the meningitis interpretive standards were used for both penicillin and cefotaxime.

2.3 Case definition

A case of IPD is defined as:

1 the isolation of *S. pneumoniae* from CSF, blood or other normally sterile site; or
2 the detection by nucleic acid amplification test of pneumococcal DNA in CSF, blood or other normally sterile site; or
3 a positive newer-generation *S. pneumoniae* antigen test (ie, Binax NOW) on CSF.

2.4 Abbreviations

PCV-7: 7-valent pneumococcal conjugate vaccine with serotypes 4, 6B, 9V, 14, 18C, 19F and 23F.
PCV-10: 10-valent pneumococcal conjugate vaccine with serotypes 1, 4, 5, 6B, 7F, 9V, 14, 18C, 19F and 23F.
PCV-13: 13-valent pneumococcal conjugate vaccine with serotypes 1, 3, 4, 5, 6A, 6B, 7F, 9V, 14, 18C, 19A, 19F and 23F.
PPV-23: 23-valent pneumococcal polysaccharide vaccine with serotypes 1, 2, 3, 4, 5, 6B, 7F, 8, 9N, 9V, 10A, 11A, 12F, 14, 15B, 17F, 18C, 19A, 19F, 20, 22F, 23F and 33F.

3. RESULTS

In 2009, 697 IPD cases were notified. A *S. pneumoniae* isolate from an invasive site was received at ESR for serotyping and susceptibility testing for 665 (95.4%) of these cases.

3.1 Laboratory criteria upon which diagnosis based

According to the case definition, IPD must be confirmed by the isolation of *S. pneumoniae* from CSF, blood or other normally sterile site; the detection of pneumococcal DNA in CSF, blood or other normally sterile site specimen; or a positive newer-generation pneumococcal antigen test on CSF.

The majority (85.2%) of cases were confirmed on the basis of a positive blood culture (Table 1). Fifty cases were notified with no laboratory evidence of IPD. As an invasive pneumococcal isolate from all of these cases was received at ESR, they were accepted as confirmed IPD cases. A further nine cases were notified as culture positive, although the site from which the pneumococcus was reportedly cultured was not a 'normally sterile site'. However, an invasive pneumococcal isolate from five of these cases was received at ESR and the notifying PHU, after review, opted to retain the remaining four cases.

Table 1. Laboratory criteria upon which invasive pneumococcal disease diagnosis based, as recorded in the case notification, 2009[1]

Basis of diagnosis	Number of cases	Percent of total cases (n=697)	Percent of cases notified as culture or DNA positive for which an isolate or DNA-positive specimen received at ESR
Culture of *Streptococcus pneumoniae* from:			
CSF	23	3.3	95.7
blood	594	85.2	97.0
pleural fluid	10	1.4	60.0
joint fluid	3	0.4	100
other sites	16[2]	2.3	56.3
Detection of pneumococcal DNA in:			
blood	1	0.1	100
No laboratory criteria recorded in notification	50[3]	7.2	-

1 For several cases, more than one method of laboratory confirmation was recorded. In this analysis, only one method of laboratory confirmation was counted for each case, with methods prioritised in the following order: pneumococcal culture from CSF, pneumococcal culture from blood, pneumococcal DNA in blood, pneumococcal culture from pleural fluid, pneumococcal culture from joint fluid, and pneumococcal culture from another sterile site.

2 For 9 of these 16 cases, the site specified was not a normally sterile site. However, a pneumococcal culture from an invasive site was referred to ESR from 5 of these 9 cases. The status of the remaining 4 cases was queried with the notifying public health unit, which, after review, opted to retain these cases.

3 A pneumococcal culture from an invasive site was referred to ESR from these 50 cases.

3.2 Disease incidence by age

The age and sex distribution of the 2009 cases is presented in Table 2, along with the incidence rate for each age group. The highest rates of disease were in infants <2 years of age and the elderly ≥75 years of age. There was an overall excess of males among cases. This excess was greatest in cases <1 year of age, with a male to female ratio of 2.8:1 in this age group.

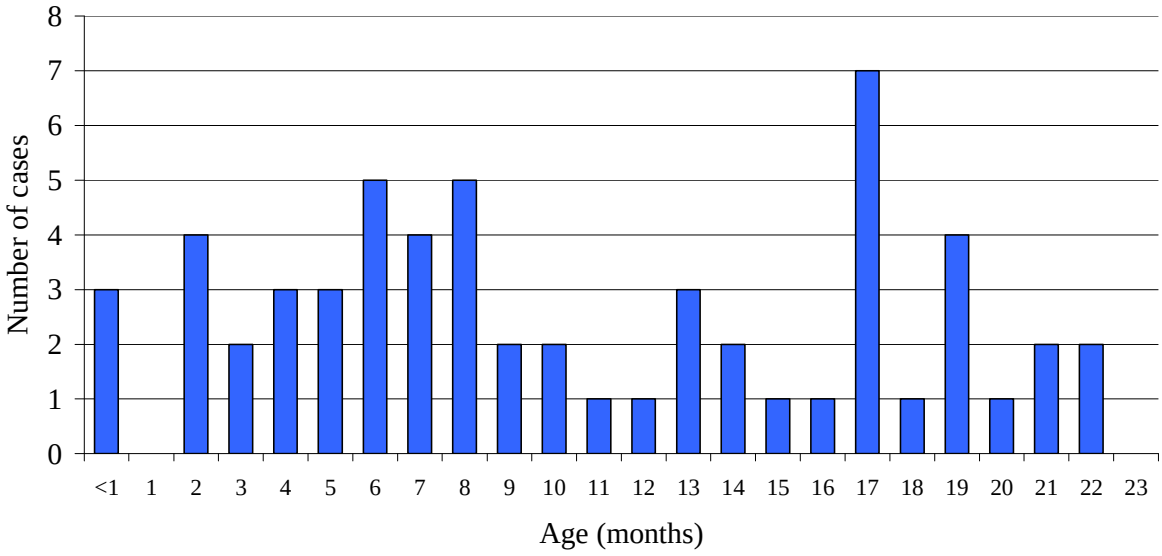
A further breakdown of the age distribution of the cases <2 years of age is shown in Figure 1.

Table 2. Numbers and rates of invasive pneumococcal disease cases by age group and sex, 2009

Age group (years)	Female		Male		All cases		
	Number	Rate[1]	Number	Rate[1]	Number	Percent	Rate[1]
<1	9	29.6	25	76.6	34	4.9	53.9
1	8	25.7	17	51.7	25	3.6	39.1
2-4	16	18.4	25	27.3	41	5.9	23.0
5-14	25	8.8	33	11.0	58	8.3	9.9
15-24	26	8.5	27	8.4	53	7.6	8.4
25-34	27	9.6	26	9.7	53	7.6	9.6
35-44	28	8.7	40	13.5	68	9.8	11.0
45-54	34	10.9	21	7.1	55	7.9	9.1
55-64	38	16.0	31	13.5	69	9.9	14.7
65-74	44	28.2	50	34.2	94	13.5	31.1
75-84	47	46.1	47	57.1	94	13.5	51.1
≥85	31	69.9	22	98.8	53	7.6	79.6
Aggregated age groups (years)[2]							
<2	17	27.6	42	64.1	59	8.5	46.4
<5	33	22.2	67	42.7	100	14.4	32.7
≥65	122	40.4	119	47.5	241	34.6	43.6
All ages	**333**	**15.1**	**364**	**17.2**	**697**	**100**	**16.1**

1 Annual incidence rate per 100 000.
2 Shaded rows indicate aggregated age groups.

Figure 1. Age distribution among invasive pneumococcal disease cases <2 years old, 2009



The all-age rate of IPD in 2009 (16.1 per 100 000) was the highest recorded in the last 5 years, 2005-2009 (Table 3). Contrary to the apparent all-age increase in IPD rates, the rates in infants <2 years old have decreased significantly (P=0.0322) since the introduction of PCV-7 to the childhood immunisation schedule.

Table 3. Rates of invasive pneumococcal disease by age group, 2005-2009[1]

Age group (years)	Annual incidence rate per 100 000				
	2005	**2006**	**2007**	**2008**	**2009**
<1	91.6	122.0	78.0	57.7	53.9
1	107.8	86.8	115.4	68.2	39.1
2-4	23.3	18.9	23.3	20.1	23.0
5-14	3.8	3.3	4.9	5.9	9.9
15-24	1.5	2.5	3.1	4.7	8.4
25-34	4.2	2.9	4.4	5.9	9.6
35-44	4.5	8.5	6.3	8.5	11.0
45-54	7.1	7.4	6.3	9.2	9.1
55-64	14.0	13.0	14.3	19.1	14.7
65-74	18.8	24.3	30.5	29.8	31.1
75-84	42.8	38.3	40.5	48.3	51.1
≥85	67.4	58.6	42.7	80.0	79.6
Aggregated age groups (years)[2]					
<2	99.7	104.6	96.2	62.9	46.4
<5	54.3	53.8	53.4	38.0	32.7
≥65	32.7	33.0	35.3	42.0	43.6
All ages	12.1	12.6	13.1	14.8	16.1

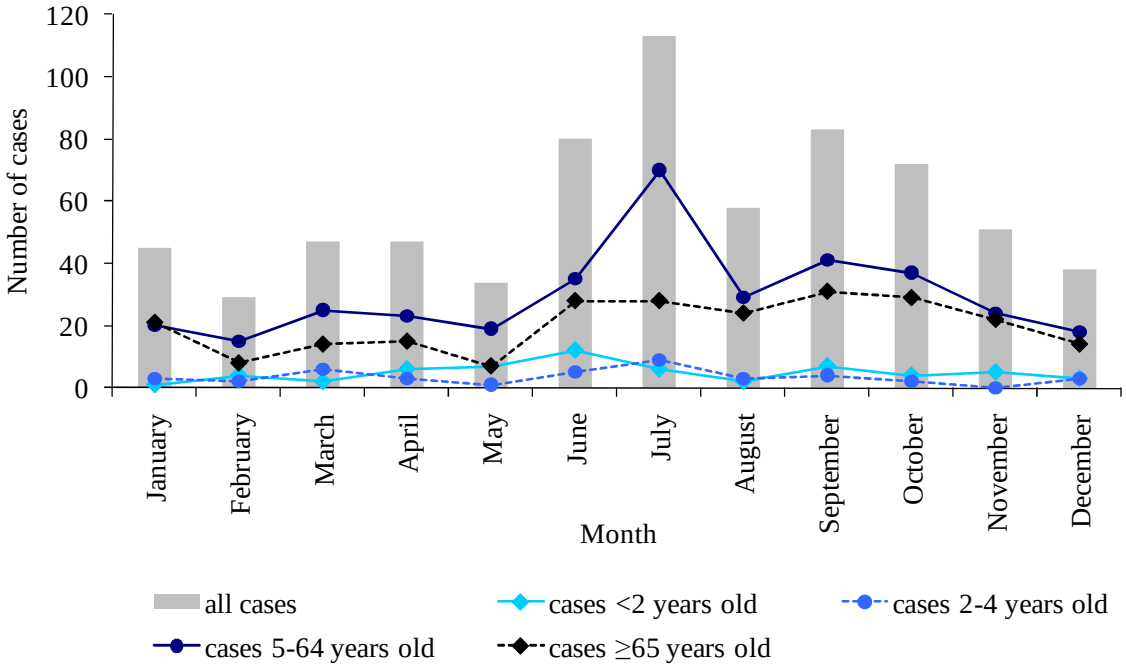
1 Data for 2005-2008 based on national laboratory-based surveillance. Data for 2009 based on IPD notifications

2 Shaded rows indicate aggregated age groups.

3.3 Disease incidence by season

IPD showed the usual seasonality in 2009, with a marked peak of cases in the winter months, although this peak is less evident in the younger age groups (Figure 2).

Figure 2. Seasonality of invasive pneumococcal disease, 2009



3.4 Disease incidence by ethnicity

The all-age rates of IPD were highest among Pacific Peoples (50.0 per 100 000) and Maori (41.2). The rates in these two ethnic groups were 3.8 and 3.2 times, respectively, the rate among Europeans (13.0) (Table 4).

Among cases <2 years of age, the rates were also highest in Pacific Peoples and Maori, with rates in these two ethnic groups being 2.5 and 3.3 times, respectively, that in Europeans. However, these rates are based on relatively small numbers of cases in this age group.

No analysis of trends over time in IPD rates among the different ethnic groups can be made, as data on the ethnicity of cases is not available for years prior to 2009.

Table 4. Numbers and rates of invasive pneumococcal disease cases by ethnicity and age group, 2009

Age group (years)	European		Maori		Pacific Peoples		Asian		Other	Unknown
	No.	Rate[1]	No.	Rate[1]	No.	Rate[1]	No.	Rate[1]	No.	No.
<1	11	37.0	14	99.8	6	117.2	1		0	2
1	8	27.5	15	111.3	2		0		0	0
2-4	20	22.4	10	25.7	7	46.3	4		0	0
5-14	22	6.6	18	13.5	13	25.8	3		0	2
15-24	10	3.1	24	23.7	14	34.4	2		1	2
25-34	26	8.2	17	21.9	9	27.0	1		0	0
35-44	34	8.2	21	27.7	11	34.2	2		0	0
45-54	28	7.0	14	25.0	9	41.5	3		1	0
55-64	42	12.7	19	60.4	6	46.6	2		0	0
65-74	62	28.2	17	102.0	7	101.3	2		0	6
75-84	76	49.1	9	161.5	5	195.2	3		0	1
≥85	46	87.9	1		1		1		0	4
Aggregated age groups (years)[2]										
<2	19	32.3	29	105.4	8	79.6	1		0	2
<5	39	26.4	39	58.7	15	59.6	5	23.5	0	2
≥65	184	43.1	27	116.8	13	130.8	6	38.0	0	11
All ages[3]	385	13.0	179	41.2	90	50.0	24	12.2	2	17

1 Annual incidence rate per 100 000, based on prioritised ethnicity. A rate is not calculated where there are <5 cases.
2 Shaded rows indicate aggregated age groups.
3 The rates for all ages are direct-standardised to the age distribution of the total NZ population.

3.5 Disease incidence by deprivation

Accurate NZ deprivation (NZDep) index data was available for 632 (90.7%) of the 697 IPD cases in 2009. In each age group, the highest numbers of cases occurred in those in the most deprived NZDep quintiles (ie, NZDep indices 7-8 and 9-10) (Table 5). Rates of IPD within NZDep quintiles could only be calculated for all ages, as population data by NZDep index and age groups was not available. The all-age rate of IPD increased in each NZDep quintile from the least deprived (1-2) to the most deprived (9-10) quintile. The rate in the most deprived quintile (27.8 per 100 000) was 3.1 times that in the least deprived quintile (9.0).

No analysis of trends over time in IPD rates by deprivation index can be made, as data on the deprivation index of cases is not available for years prior to 2009.

Table 5. Number and percentage of invasive pneumococcal disease cases by 2006 NZ deprivation index and age group, 2009

2006 NZ deprivation index quintile	Age group (years)					
	Number (% within the age group) in each quintile[1]					Rate[2]
	<2	2-4	5-64	≥65	All ages	
1-2	7 (13.5)	5 (12.5)	31 (9.5)	31 (14.4)	74 (11.7)	9.0
3-4	8 (15.4)	0	39 (12.0)	35 (16.3)	82 (13.0)	10.1
5-6	3 (5.8)	11 (27.5)	48 (14.8)	44 (20.5)	106 (16.8)	13.3
7-8	8 (15.4)	12 (30.0)	70 (21.5)	58 (27.0)	148 (23.4)	18.7
9-10	26 (50.0)	12 (30.0)	137 (42.1)	47 (21.9)	222 (35.1)	27.8
Total	52	40	325	215	632	

1 Data available for 88.1% (52/59) of cases <2 years of age, 97.6% (40/41) of cases 2-4 years of age, 91.3% (325/356) of cases 5-64 years of age, and 89.2% (215/241) of cases ≥65 years of age.
2 Annual incidence rate per 100 000.

3.6 Disease presentation and fatalities

Information on clinical presentation was available for 615 (88.2%) of the 697 IPD cases in 2009 (Table 6).

The rate of pneumococcal meningitis was 1.1 case per 100 000 for all ages, 14.3 per 100 000 for the <1 year age group, 7.1 per 100 000 for <2 year age group, and 4.3 per 100 000 for the <5 year age group.

Of the nine cases of meningitis in infants <1 years of age, seven were Maori (which equates to a rate of 49.9 per 100 000), one was a Pacific infant and one was European.

Table 6. Clinical presentation of invasive pneumococcal disease cases by age group, 2009[1]

Age group (years)	Number of cases (% within the age group)					Number of cases for whom presentation information reported[2]
	Meningitis	Bacteraemia	Empyema	Pneumonia	Other	
<1	9 (32.1)	4 (14.3)	0	9 (32.1)	6 (21.4)	28
1	0	5 (23.8)	3 (14.3)	10 (47.6)	3 (14.3)	21
2-4	4 (10.3)	13 (33.3)	1 (2.6)	15 (38.5)	6 (15.4)	39
5-14	5 (9.4)	6 (11.3)	1 (1.9)	37 (69.8)	4 (7.5)	53
15-64	18 (6.8)	24 (9.0)	5 (1.9)	207 (77.8)	12 (4.5)	266
≥65	10 (4.8)	27 (13.0)	4 (1.9)	154 (74.0)	13 (6.3)	208
Aggregated age groups (years)[3]						
<2	9 (18.4)	9 (18.4)	3 (6.1)	19 (38.8)	9 (18.4)	49
<5	13 (14.8)	22 (25.0)	4 (4.5)	34 (38.6)	15 (17.0)	88
All ages	46 (7.5)	79 (12.8)	14 (2.3)	432 (70.2)	44 (7.2)	615

1 In this analysis, only one presentation was counted for each case, with presentations prioritised in the following order: meningitis, bacteraemia, empyema, pneumonia and 'Other'.

2 Data available for 82.4% (28/34) of cases <1 year of age, 84.0% (21/25) of cases 1 year of age, 95.1% (39/41) of cases 2-4 years of age, 91.4 % (53/58) of cases 5-14 years of age, 89.3% (266/298) of cases 15-64 years of age, and 86.3% (208/241) of cases ≥65 years of age.

3 Shaded rows indicate aggregated age groups.

Information on whether the patient survived or died was reported for 626 (89.8%) of the total 697 cases. Among the 63 cases who died, 35 were reported as dying from IPD, giving an overall case-fatality rate of 5.6% among the cases for whom this information was reported. The case-fatality rates for the different age groups are presented in Appendix 1.

3.7 Risk factors among IPD cases

The risk factors reported among IPD cases in 2009 are recorded in Table 7. Risk factors for the subset of 59 cases <2 years of age are presented in Appendix 2.

Table 7. Risk factors among invasive pneumococcal disease cases, 2009

Risk factor	Number of cases for whom information on the risk reported	Number (%[1]) of cases with the risk
Premature <37 weeks gestation (cases <1 year of age)	15	5 (33.3)
Congenital or chromosomal abnormality	555	7 (1.3)
Chronic lung disease or cystic fibrosis	570	78 (13.7)
Anatomical or functional asplenia	546	6 (1.1)
Immunocompromised[2]	567	89 (15.7)
Chronic illness[3]	584	250 (42.8)
Cochlear implants	539	2 (0.4)
Current smoker[4]	329	94 (28.6)
Smoking in household (cases <5 years of age)	35	14 (40.0)
In childcare (cases <5 years of age)	43	7 (16.3)
Resident in long-term or other chronic facility[5]	581	44 (7.6)
Other risk factors	NA[6]	114 (16.4[7])

1 Percentage based on only those cases for whom information reported for each particular risk factor, except for 'Other' risk factors (see footnote 7).
2 Includes HIV/AIDS, lymphoma, organ transplant, multiple myeloma, nephrotic syndrome, chronic drug therapy, dysgammaglobulinaemia and sickle cell anaemia.
3 Includes CSF leak, intracranial shunts, diabetes, cardiac disease, pulmonary disease, chronic liver disease, renal impairment and alcohol-related disease.
4 Only cases ≥18 years of age included in this analysis of current smokers
5 Among cases ≥75 years of age, 26.8% (33 of 123 for whom the information was reported) were residents in a long-term or other chronic facility.
6 Not applicable, as only reportable when case has 'Other' risk factors.
7 Percentage of all 697 IPD cases.

3.8 Immunisation status of cases

Among the 59 cases that were <2 years old, 20 were recorded as having been immunised, 22 as not immunised, and the immunisation status was recorded as unknown for the remaining 17 cases. Six of these 20 immunised cases were recorded as having had 1 dose of PCV-7, five had 2 doses and nine had 3 doses. No case was recorded as having had the booster fourth dose. 25.0% of the cases in immunised infants were due to a PCV-7 serotype compared with 57.1% of the cases in non-immunised infants.

Among cases ≥2 years of age, only five were recorded as having been immunised: two 2 year olds, an 8 year old, a 33 year old and an 86 year old. Both 2 year old cases were recorded as having had 3 doses of vaccine, but not a booster. No information on doses was recorded for the other three cases, except that the 8 year old case had had 1 dose of PCV-7.

The immunisation status of three of the six asplenic cases was recorded. One of these three cases had been immunised, although no details of the doses were recorded.

3.9 Incidence by district health board

Table 8 shows the number of cases by age group and the incidence rates in each region and district health board (DHB). Care should be taken with comparing the DHB rates, as some DHBs had relatively small numbers of cases. The incidence of IPD in the Midland region (23.1 per 100 000) was significantly higher than that in any of the other regions.

Table 8. Invasive pneumococcal disease cases by region, district health board (DHB) and age group, 2009

	Number of cases by age group (years)					Rate[1]
Region and DHB	**<2**	**<5**	**5-64**	**≥65**	**All ages**	**(all ages)**
Northern	**22**	**38**	**133**	**65**	**236**	**14.7**
Northland	5	7	13	12	32	20.5
Waitemata	8	10	30	23	63	11.9
Auckland	1	8	30	13	51	11.5
Counties Manukau	8	13	60	17	90	18.7
Midland	**20**	**26**	**99**	**65**	**190**	**23.1**
Waikato	10	12	48	22	82	22.8
Lakes	2	4	14	11	29	28.5
Bay of Plenty	6	7	24	19	50	24.1
Tairawhiti	1	2	4	3	9	19.5
Taranaki	1	1	9	10	20	18.5
Central	**10**	**23**	**72**	**63**	**158**	**16.0**
Hawke's Bay	0	2	18	15	35	22.7
Whanganui	0	0	5	7	12	19.0
MidCentral	2	4	6	7	17	10.2
Hutt	2	4	16	10	30	21.0
Capital and Coast	5	8	11	11	30	10.4
Wairarapa	0	1	4	7	12	30.0
Nelson Marlborough	1	4	12	6	22	16.1
Southern	**7**	**13**	**52**	**48**	**113**	**12.7**
West Coast	0	0	0	0	0	
Canterbury	5	8	25	22	55	11.0
South Canterbury	0	0	2	4	6	10.8
Otago	2	2	18	12	32	17.0
Southland	0	3	7	10	20	17.9
New Zealand total	**59**	**100**	**356**	**241**	**697**	**16.1**

1 Annual incidence rate per 100 000. A rate is not calculated where there are <5 cases.

3.10 Serotype distribution

Table 9 shows, for the different age groups, the proportion of the 665 culture-positive IPD cases in 2009 caused by each of the serotypes included in the 7, 10 and 13-valent pneumococcal conjugate vaccines and any other serotypes that accounted for ≥1% of cases. A full list of the serotypes of all culture-positive cases is presented in Appendix 3.

92.8% of the isolates from cases ≥65 years of age were due to one of the serotypes included in PPV-23. Vaccination with PPV-23 is recommended for people in this age group.

Table 9. Serotypes among invasive pneumococcal disease cases and vaccine coverage by age group, 2009

Serotype	Proportion (%) of IPD cases within the age group (years) due to the serotype:					
	<2 (n=55)	**2-4** (n=38)	**<5**[1] (n=93)	**5-64** (n=342)	**≥65** (n=230)	**All ages** (n=665)
Serotypes in PCV-7:						
4	1.8	2.6	2.2	9.4	10.0	8.6
6B	7.3	10.5	8.6	2.3	7.4	5.0
9V	0.0	2.6	1.1	4.4	8.3	5.3
14	12.7	26.3	18.3	6.7	15.2	11.3
18C	1.8	7.9	4.3	2.9	2.6	3.0
19F	14.6	13.2	14.0	7.6	8.3	8.7
23F	3.6	7.9	5.4	4.7	11.7	7.2
Total for PCV-7 serotypes	**41.8**	**71.1**	**53.8**	**38.0**	**63.5**	**49.0**
Additional serotypes in PCV-10:						
1	21.8	7.9	16.1	36.3	6.1	23.0
5	0.0	0.0	0.0	0.0	0.0	0.0
7F	1.8	0.0	1.1	3.8	1.7	2.7
Total for PCV-10 serotypes	**65.5**	**79.0**	**71.0**	**78.1**	**71.3**	**74.7**
Additional serotypes in PCV-13:						
3	5.5	0.0	3.2	3.5	4.8	3.9
6A	3.6	2.6	3.2	1.5	2.2	2.0
19A	14.6	10.5	12.9	4.7	3.9	5.6
Total for PCV-13 serotypes	**89.1**	**92.1**	**90.3**	**87.7**	**82.2**	**86.2**
Non-PCV serotypes[2]:						
8	0.0	0.0	0.0	2.3	1.7	1.8
9N	0.0	5.3	2.2	1.2	0.9	1.2
22F	1.8	0.0	1.1	3.2	4.4	3.3
other types	9.1	2.6	6.5	5.6	10.9	7.5

1 Shaded column indicates an aggregated age group.

2 The specific serotypes listed are those that accounted for ≥1% of all cases. See the Appendix for a full list of all serotypes.

The serotypes causing IPD during the two years before the introduction of PCV-7 to the childhood immunisation programme (2006 and 2007), and the year of the introduction and the year after (2008 and 2009) are shown in Table 10. The proportion of IPD among <2 year olds caused by PCV-7 serotypes decreased from 83.8% in 2006-7 to 41.8% in 2009 (Table 9).

Table 10. Serotypes among invasive pneumococcal disease cases and vaccine coverage by age group, 2006-7 compared with 2008-9

Serotype	Proportion (%) of IPD cases within the age group (years) due to the serotype:									
	<2		<5		5-64		≥65		All ages	
	2006-7 n=235	2008-9 n=133	2006-7 n=307	2008-9 n=205	2006-7 n=415	2008-9 n=633	2006-7 n=355	2008-9 n=457	2006-7 n=1077	2008-9 n=1295
Serotypes in PCV-7:										
4	5.5	4.5	5.2	4.4	18.3	10.3	11.0	9.6	12.2	9.1
6B	15.3	18.8	15.3	16.1	5.5	2.7	6.2	7.2	8.5	6.4
9V	3.8	2.3	4.6	2.4	5.3	5.4	8.2	8.1	6.0	5.9
14	32.8	21.1	30.9	23.4	14.9	8.2	20.0	18.2	21.2	14.1
18C	5.1	5.3	6.8	4.9	2.7	2.8	1.7	3.1	3.5	3.2
19F	13.6	10.5	12.7	11.7	5.8	6.5	9.3	7.7	8.9	7.7
23F	7.7	3.8	6.2	4.9	5.8	4.9	8.5	9.4	6.8	6.5
Total for PCV-7 serotypes	**83.8**	**66.2**	**81.8**	**67.8**	**58.3**	**40.8**	**64.8**	**63.2**	**67.1**	**53.0**
Additional serotypes in PCV-10:										
1	1.7	9.8	1.6	9.8	9.2	28.4	2.0	5.0	4.6	17.2
5	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
7F	0.4	0.8	0.3	0.5	2.9	4.0	2.0	1.3	1.9	2.5
Total for PCV-10 serotypes	**86.0**	**76.7**	**83.7**	**78.1**	**70.4**	**73.1**	**68.7**	**69.6**	**73.6**	**72.7**
Additional serotypes in PCV-13:										
3	0.9	2.3	0.7	1.5	4.1	4.4	7.0	5.0	4.1	4.2
6A	2.6	1.5	2.9	2.4	2.4	1.6	1.4	2.0	2.2	1.9
19A	5.1	9.8	6.8	9.3	4.8	6.0	4.5	4.4	5.3	6.0
Total for PCV-13 serotypes	**94.5**	**90.2**	**94.1**	**91.2**	**81.7**	**85.2**	**81.7**	**81.0**	**85.2**	**84.6**
Non-PCV serotypes[1]:										
8	0.0	1.5	0.0	1.0	5.8	3.0	2.0	1.8	2.9	2.2
9N	0.0	0.0	0.0	1.0	1.9	1.6	2.3	1.5	1.5	1.5
10A	0.4	0.8	0.7	1.0	1.5	0.3	1.1	0.4	1.1	0.5
11A	0.4	0.8	0.3	0.5	1.7	1.1	2.0	1.1	1.4	1.0
20	0.0	0.0	0.0	0.0	0.5	1.7	1.4	0.9	0.7	1.2
22F	0.9	0.8	0.7	0.5	2.4	2.5	2.5	4.4	2.0	2.9
33F	0.4	0.8	0.7	0.5	0.0	0.5	0.9	2.2	0.5	1.1

1 The specific serotypes listed are those that accounted for ≥1% of all cases in either of the two 2-year periods.

Figure 3 shows the trends since 2004 in the rates of disease, among infants <2 years of age, due to each of the serotypes included in PCV-7, and the other common serotypes in this age group (serotypes 19A, 6A and 1). There have clearly been marked decreases in the rates of disease caused by most of the PCV-7 serotypes since the introduction of PCV-7 into the national immunisation schedule in 2008. There has been no change in the rate of disease due to the non-PCV-7 serotype 19A in this age group.

The rate of disease among <2 year olds caused collectively by PCV-7 serotypes decreased from an annual average of 86.0 per 100 000 during the years 2004-2007 to 18.1 per 100 000 in 2009 (Figure 4). In 2009 the rates of disease caused by PCV-7 serotypes varied among infants <2 years according to ethnicity. The rate for Maori (40.0 per 100 000) was the highest and over twice the average rate of 18.1. The rate for Europeans was 18.7 per 100 000 and there were no cases of IPD due to a PCV-7 serotype among Pacific infants.

The decrease in the rate of disease caused by the PCV-7 serotypes observed in the <2 year age group has not occurred in other age groups (Figure 4).

Figure 3. Rates of invasive pneumococcal disease for PCV-7 serotypes, and types 19A, 6A and 1, <2 years olds each year 2004-2009

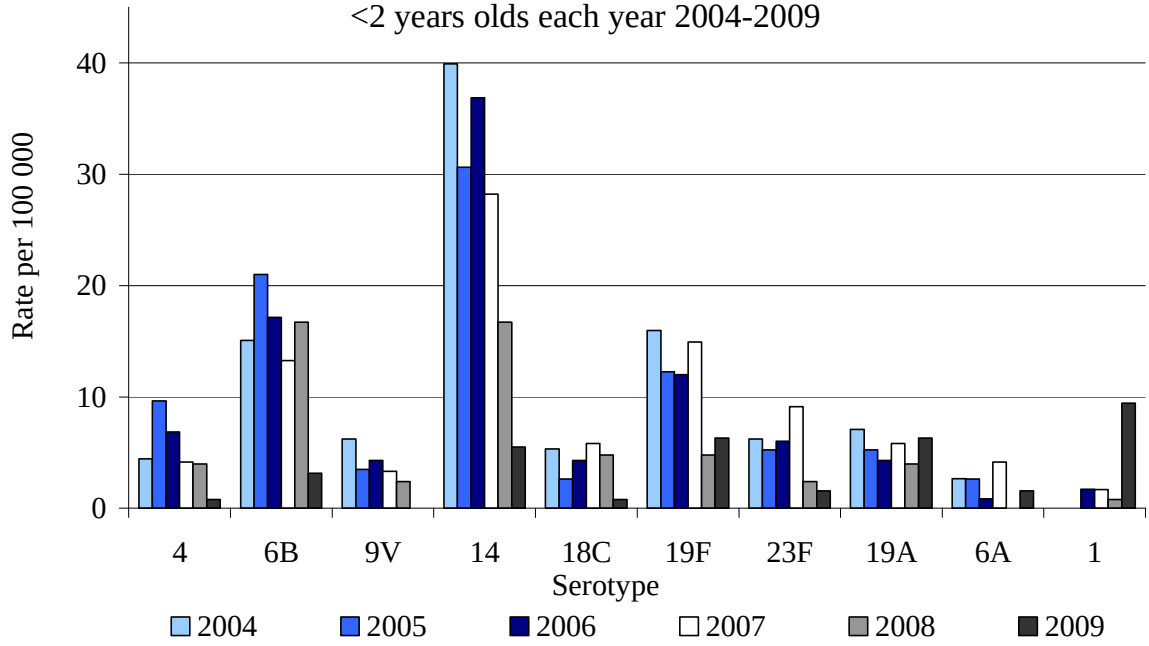


Figure 5 shows the trends since 2004 in the rates of disease due to serotypes not included in PCV-7. Increases in rates are evident over the last 1-2 years for all age groups except the 2-4 year olds. These increases were predominantly due to serotype 1 disease.

An increase in serotype 1 disease was first noted in 2007, but in older age groups only. In 2008, serotype 1 accounted for 42.9% of the IPD cases in the 5-14 year age group, 37.9% in the 15-24 year group and 31.3% in the 25-34 year group, but only 1.3% of cases <2 years old. However in 2009, this serotype was the most common type among <2 year olds, accounting for 21.8% of cases in this age group (Table 9). In 2009, the rate of serotype 1 disease among <2 year olds was 9.4 per 100 000, whereas the rate over the 2004-2007 period averaged 0.9 per 100 000. 73.9% of the

serotype 1 cases were in Pacific Peoples or Maori. This serotype accounted for 50.6% of the all IPD cases in Pacific Peoples and 41.4% in Maori. 81.7% of serotype 1 cases were from the Northern and Midland regions.

Figure 4. Rates of invasive pneumococcal disease caused by PCV-7 serotypes, by age group each year 2004-2009

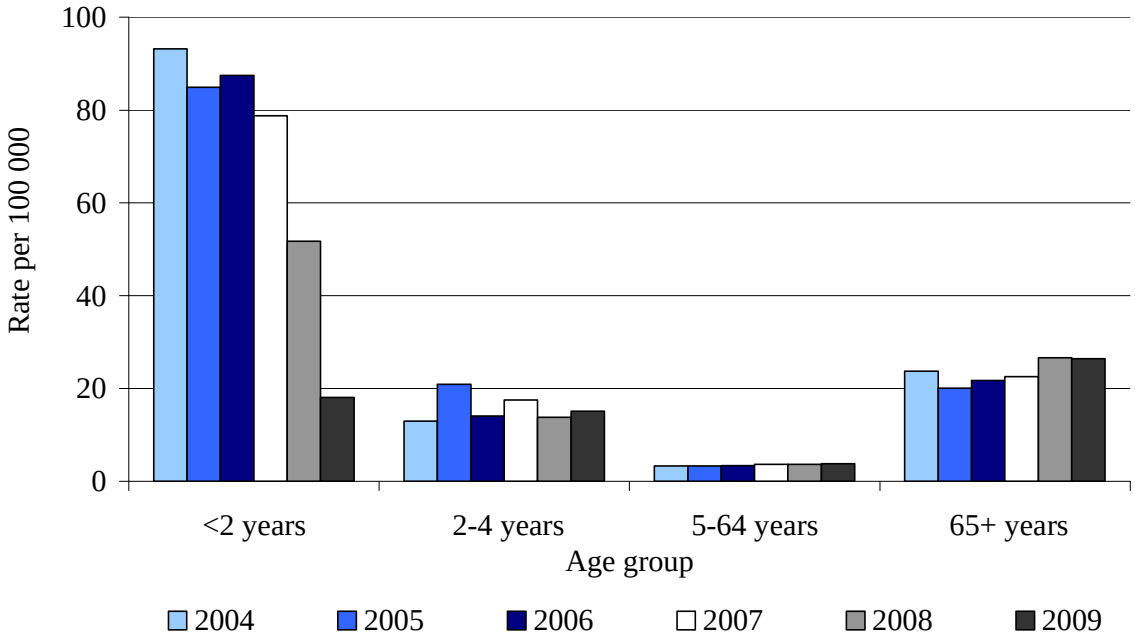
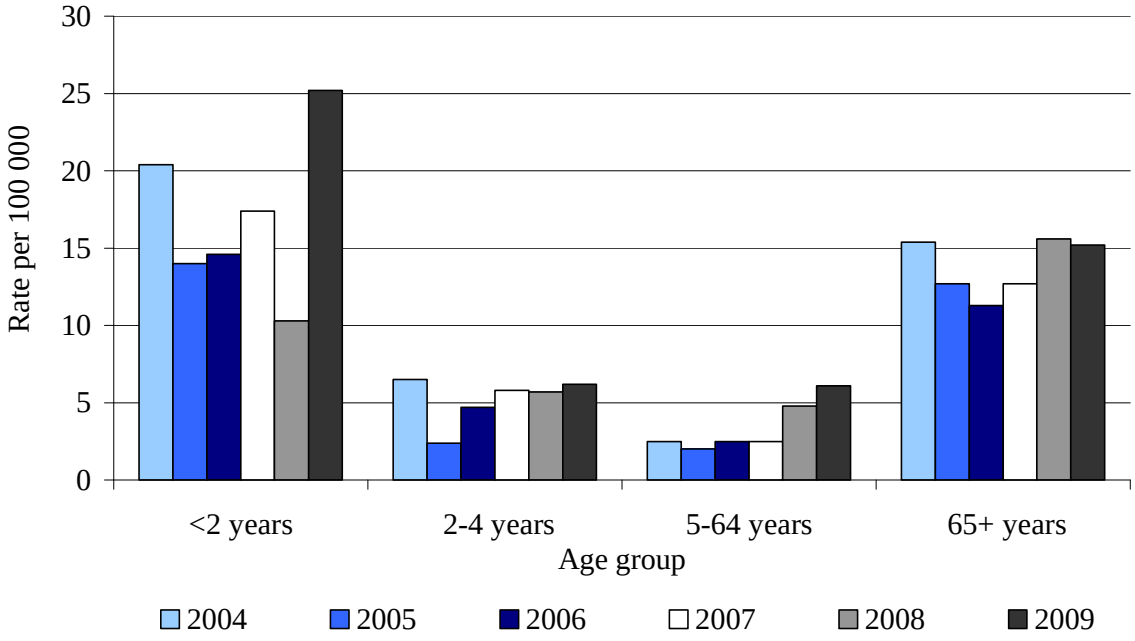


Figure 5. Rates of invasive pneumococcal disease caused by non-PCV-7 serotypes, by age group each year 2004-2009



3.11 Antimicrobial susceptibility

Table 11 shows the antimicrobial susceptibility of the 665 culture-positive IPD cases in 2009.

6.3% of isolates had combined penicillin (meningitis interpretation) and erythromycin resistance, and 0.3% had combined penicillin-intermediate resistance (non-meningitis interpretation) and erythromycin resistance. Among the penicillin-resistant isolates (meningitis interpretation), 29.7% (35/118) were multiresistant to ≥3 additional antibiotics, commonly co-trimoxazole, erythromycin and tetracycline with or without cefotaxime resistance.

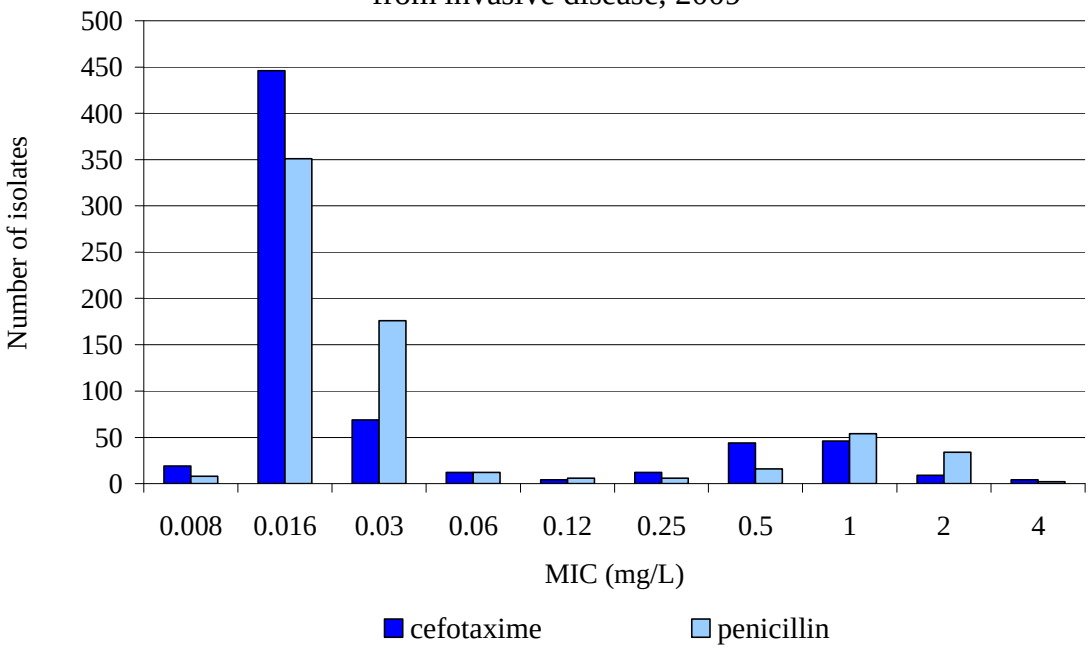
Table 11. Antimicrobial susceptibility among isolates from invasive pneumococcal disease cases, 2009

	Interpretive standards			Percent		
	S[1]	I	R			
	MIC (mg/L)			S	I	R
penicillin						
meningitis	≤0.06	-	≥0.12	82.3	-	17.7
non-meningitis	≤2	4	≥8	99.7	0.3	0.0
oral treatment	≤0.06	0.12-1	≥2	82.3	12.3	5.4
cefotaxime						
meningitis	≤0.5	1	≥2	91.1	6.9	2.0
non-meningitis	≤1	2	≥4	98.1	1.4	0.6
moxifloxacin	≤1	2	≥4	100	0.0	0.0
	Zone diameter (mm)					
chloramphenicol	≥21	-	≤20	98.8	-	1.2
clindamycin[2]	≥19	16-18	≤15	95.5	0.5	4.1
co-trimoxazole	≥19	16-18	≤15	72.6	2.1	25.3
erythromycin	≥21	16-20	≤15	90.2	0.2	9.6
tetracycline	≥23	19-22	≤18	92.5	0.3	7.2
vancomycin	≥17	-	-	100	-	-

1 S, susceptible; I, intermediate; R, resistant.
2 The percentage resistant given is for constitutive clindamycin resistance. A further 3 isolates (0.5%) had inducible clindamycin resistance.

Figure 6 shows the penicillin and cefotaxime MIC distribution, with the typical bimodal distribution for both antibiotics.

Figure 6. Penicillin and cefotaxime MIC distribution of pneumococci from invasive disease, 2009



Trends in penicillin resistance based on the meningitis interpretive standards and the non-meningitis interpretive standards, for the 10 years, 2000-2009, are shown in Figures 7 and 8, respectively. Between 2000 and 2008 there was a significant trend of increasing penicillin resistance (meningitis interpretation) (P=0.0457), but between 2008 and 2009 resistance decreased, although this decrease was not highly significant (P=0.0515) (Figure 7).

Intermediate resistance (non-meningitis interpretation) also decreased in 2009, and there has been a significant trend of decreasing intermediate resistance since 2004 (P=0.0003) (Figure 8).

Figure 7. Penicillin resistance (meningitis interpretation) among pneumococci from invasive disease, 2000-2009

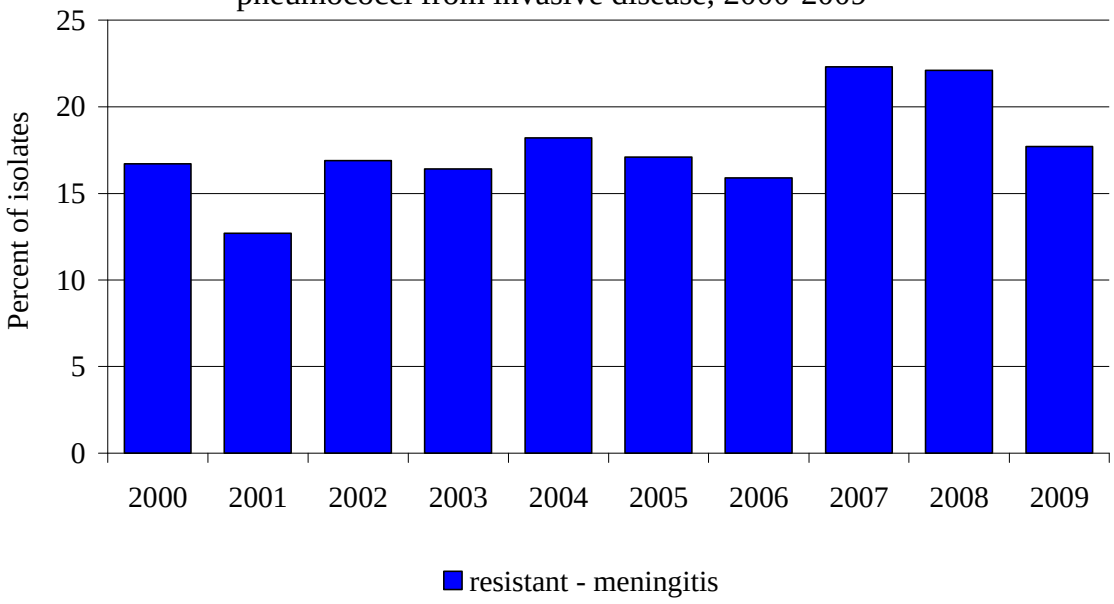
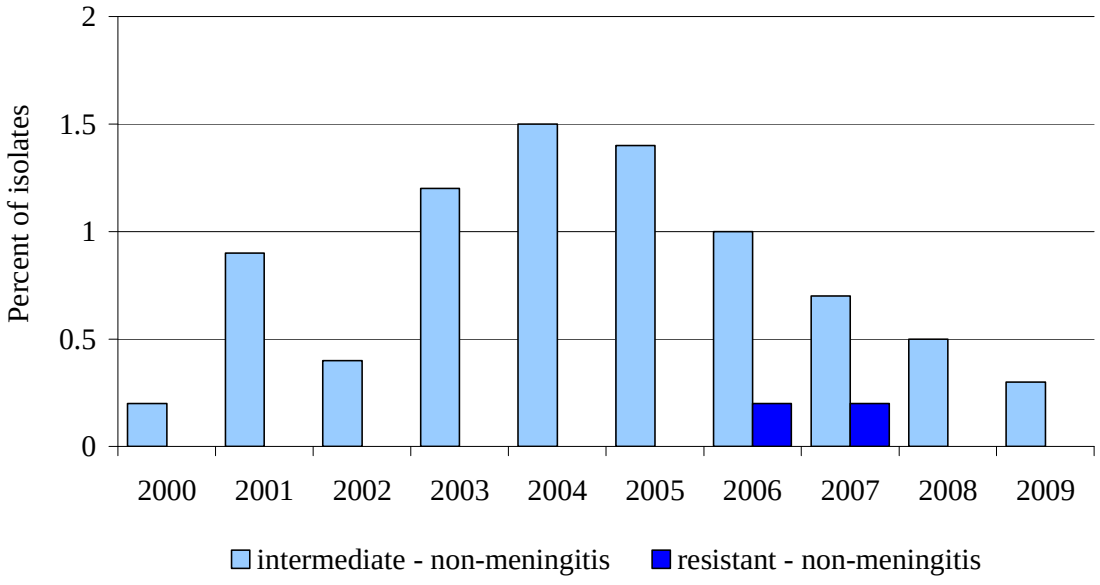


Figure 8. Penicillin intermediate resistance and resistance (non-meningitis interpretation) among pneumococci from invasive disease, 2000-2009



Trends in cefotaxime resistance based on the meningitis interpretive standards and the non-meningitis interpretive standards, for the 10 years, 2000-2009, are shown in Figures 9 and 10, respectively. Between 2000 and 2008 there was a significant trend of increasing cefotaxime resistance (meningitis interpretation) (P=0.0110), but between 2008 and 2009 there was a significant decrease (P=0.0220) (Figure 9).

Similarly there was a trend of increasing but not significant (P=0.0797) cefotaxime resistance (non-meningitis interpretation) between 2000 and 2008, but a significant decrease between 2008 and 2009 (P=0.0264) (Figure 10)

Figure 9. Cefotaxime intermediate resistance and resistance (meningitis interpretation) among pneumococci from invasive disease, 2000-2009

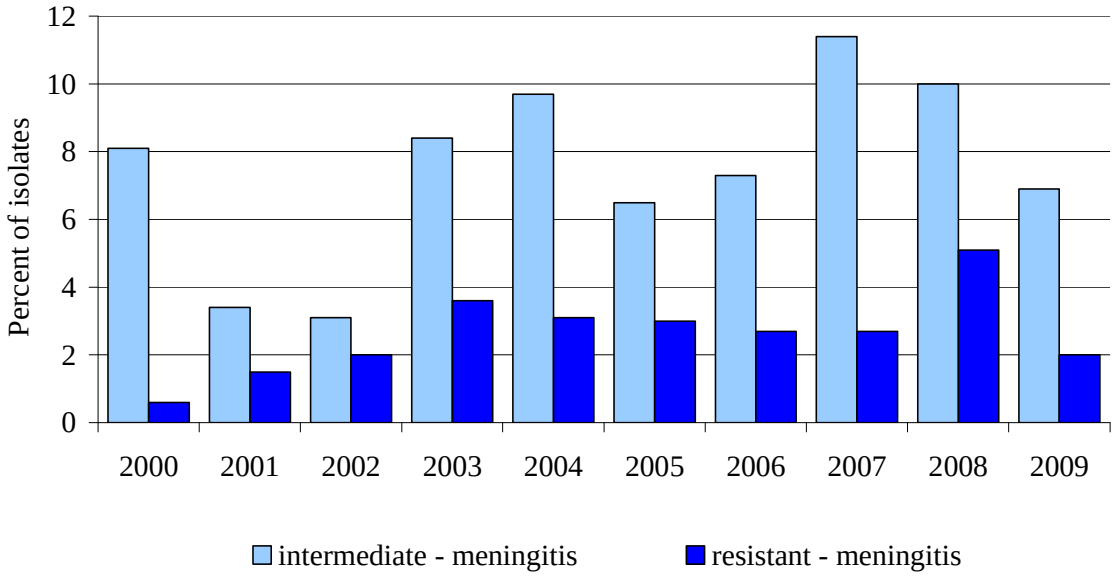
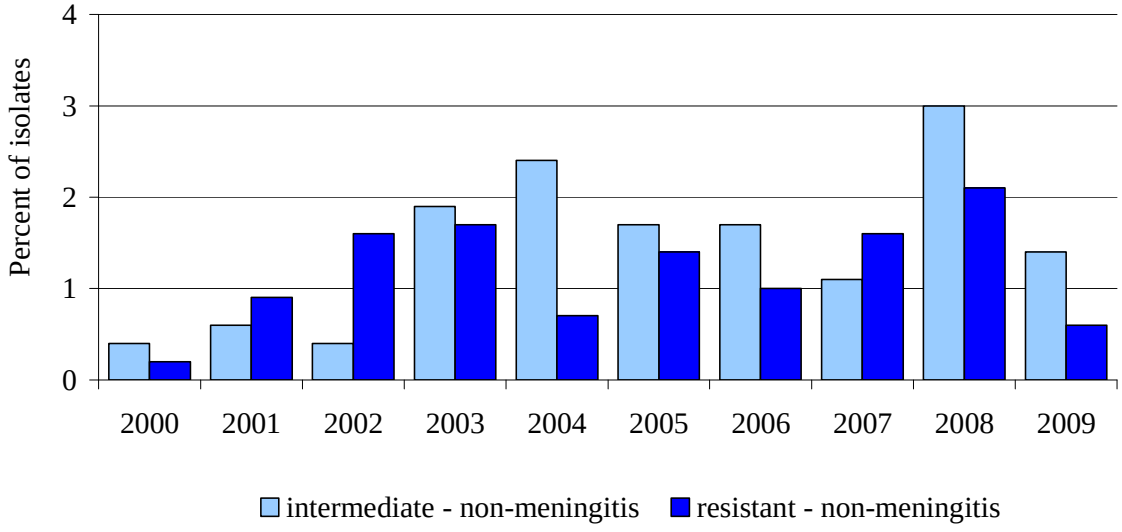


Figure 10. Cefotaxime intermediate resistance and resistance (non-meningitis interpretation) among pneumococci from invasive disease, 2000-2009



Penicillin and cefotaxime resistance in each DHB is shown in Table 12.

Table 12. Penicillin and cefotaxime resistance among isolates from invasive pneumococcal disease by region and district health board (DHB), 2009

Region and DHB	Number of isolates	Penicillin	Cefotaxime	
		% resistant[1] MIC ≥0.12 mg/L	% intermediate[1] MIC 1 mg/L	% resistant[1] MIC ≥2 mg/L
Northland	**223**	**21.5**	**9.0**	**1.4**
Northland	31	25.8	12.9	3.2
Waitemata	57	28.1	10.5	1.8
Auckland	49	32.7	18.4	2.0
Counties Manukau	86	9.3	1.2	0.0
Midland	**179**	**11.7**	**2.8**	**1.7**
Waikato	80	11.3	2.5	3.8
Lakes	25	12.0	4.0	0.0
Bay of Plenty	45	6.7	0.0	0.0
Tairawhiti	9	11.1	0.0	0.0
Taranaki	20	25.0	10.0	0.0
Central	**152**	**16.5**	**9.2**	**1.3**
Hawke's Bay	35	11.4	8.6	0.0
Whanganui	12	16.7	0.0	0.0
MidCentral	16	18.8	18.8	0.0
Hutt	30	30.0	16.7	3.3
Capital and Coast	30	13.3	6.7	3.3
Wairarapa	12	8.3	8.3	0.0
Nelson Marlborough	17	11.8	0.0	0.0
Southern	**111**	**21.6**	**6.3**	**4.5**
West Coast	0	-	-	-
Canterbury	53	22.6	7.6	5.7
South Canterbury	6	16.7	0.0	0.0
Otago	32	31.3	9.4	6.3
Southland	20	5.0	0.0	0.0
New Zealand total	**665**	**17.7**	**6.9**	**2.0**

1 Meningitis interpretations; no intermediate category for penicillin.

Penicillin and cefotaxime resistance among isolates from the different age groups is shown in Table 13. Penicillin resistance (meningitis interpretation) was significantly higher among isolates from cases 2-4 and ≥65 years of age (P=0.0062 and 0.0049, respectively), and significantly lower among isolates from cases 5-64 years of age (P=0.0001). There were no significant differences in cefotaxime resistance (meningitis interpretation) between the age groups.

Table 13. Penicillin and cefotaxime resistance among isolates from invasive pneumococcal disease cases by patient age, 2009

Age group (years)	Number (%[1]) isolates		
	Penicillin	Cefotaxime	
	resistant[2] MIC ≥0.12 mg/L	intermediate[2] MIC 1 mg/L	resistant[2] MIC ≥2 mg/L
<2 (n=55)	9 (16.4)	3 (5.5)	2 (3.6)
2-4 (n=38)	13 (34.2)	9 (23.7)	0
5-64 (n=342)	42 (12.3)	14 (4.1)	7 (2.1)
≥65 (n=230)	54 (23.5)	20 (8.7)	4 (1.7)
All ages (n=665)	118 (17.7)	46 (6.9)	13 (2.0)

1 Percentage of the cases within the age group.
2 Meningitis interpretations; no intermediate category for penicillin.

The majority of the penicillin-resistant (meningitis interpretation) invasive pneumococci were one of the serotypes usually associated with penicillin resistance (Table 14). 91.5% of the penicillin-resistant isolates, and an even higher proportion of cefotaxime-intermediate, cefotaxime-resistant and multiresistant isolates, were serotypes included in PCV-7. Seven (77.8%) of the nine penicillin-resistant (meningitis interpretation) isolates from cases <2 years of age were PCV-7 serotypes. The other two penicillin-resistant isolates from cases in this age group were both serotype 19A. All cefotaxime-intermediate and cefotaxime-resistant isolates (meningitis interpretations) from cases <2 years old were PCV-7 serotypes.

Table 14. Serotypes among penicillin resistant, cefotaxime resistant and intermediate, and multiresistant isolates from invasive pneumococcal disease cases, 2009

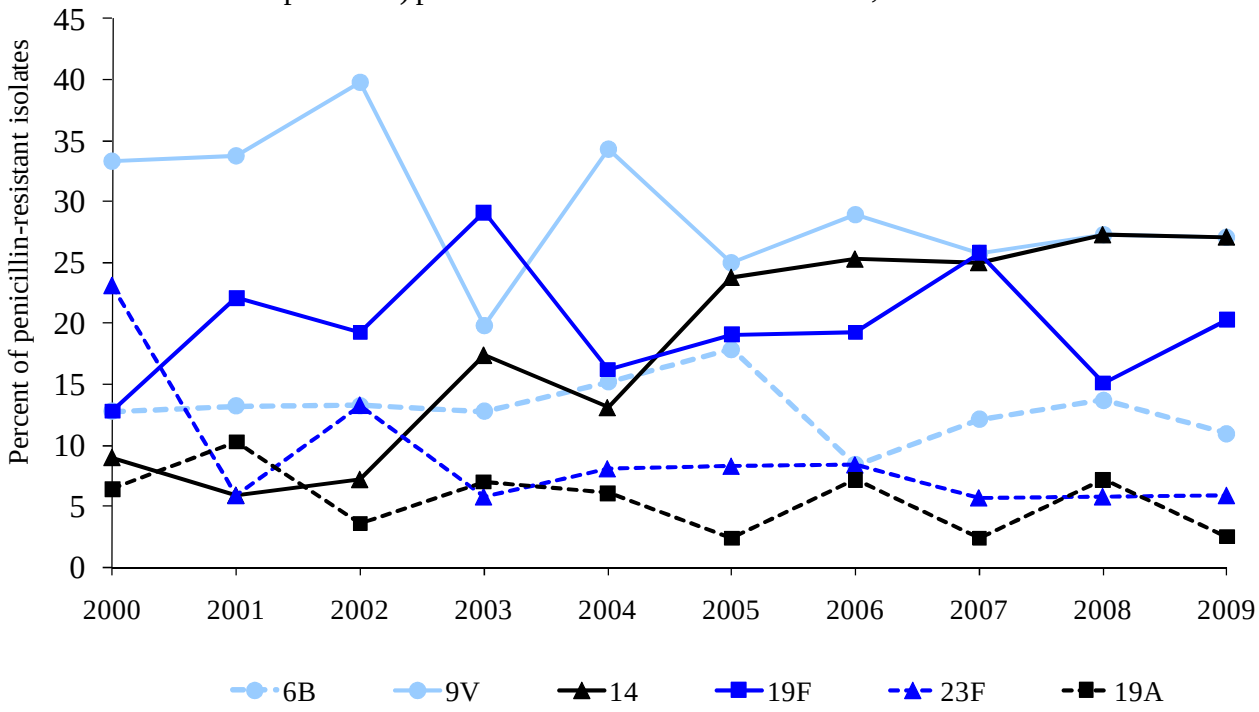
Serotype	Number (%[1]) isolates			
	Penicillin	Cefotaxime		Multi-resistant[3] (n=35)
	resistant[2] MIC ≥0.12 mg/L (n=118)	intermediate[2] MIC 1 mg/L (n=46)	resistant[2] MIC ≥2 mg/L (n=13)	
Serotypes in PCV-7:				
4	0	0	0	0
6B	13 (11.0)	6 (13.0)	0	4 (11.4)
9V	32 (27.1)	10 (21.7)	0	0
14	32 (27.1)	16 (34.8)	2 (15.4)	6 (17.1)
18C	0	0	0	0
19F	24 (20.3)	10 (21.7)	9 (69.2)	20 (57.1)
23F	7 (5.9)	1 (2.2)	2 (15.4)	4 (11.4)
Total for PCV-7 serotypes	**108 (91.5)**	**43 (93.5)**	**13 (100)**	**34 (97.1)**
Additional serotypes in PCV-10:				
1	0	0	0	0
5	0	0	0	0
7F	0	0	0	0
Total for PCV-10 serotypes	**108 (91.5)**	**43 (93.5)**	**13 (100)**	**34 (97.1)**
Additional serotypes in PCV-13:				
3	0	0	0	0
6A	1 (0.9)	1 (2.2)	0	1 (2.9)
19A	3 (2.5)	0	0	0
Total for PCV-13 serotypes	**112 (94.9)**	**44 (95.7)**	**13 (100)**	**35 (100)**
Non-PCV serotypes:				
6 non-typable	1 (0.9)	0	0	0
9N	3 (2.5)	2 (4.4)	0	0
9 Non-typable	1 (0.9)	0	0	0
Non-typable	1 (0.9)	0	0	0

1 Percentage of the intermediate or resistant isolates.
2 Meningitis interpretations; no intermediate category for penicillin.
3 Resistant to penicillin (meningitis interpretation) and three additional antibiotics.

Serotype 19F accounted for the majority of the multiresistant isolates (Table 14). In recent years, the multiresistant 19F isolates have been resistant to at least penicillin, cefotaxime, co-trimoxazole, erythromycin and tetracycline. However in 2009, only about half (9/20) of the multiresistant 19F isolates were cefotaxime resistant.

Over the last 10 years, serotype 9V has been the prevalent penicillin-resistant serotype. Serotype 19F and, in more recent years, serotype 14 are the other two more prevalent serotypes among penicillin-resistant invasive pneumococci (Figure 11).

Figure 11. Serotype distribution among penicillin-resistant (meningitis interpretation) pneumococci from invasive disease, 2000-2009



4. DISCUSSION

A 4-dose schedule of PCV-7 (3-dose primary series plus booster) was added to the New Zealand childhood immunisation schedule in June 2008, with a catch-up programme for all children born on or after 1 January 2008. The impact of the vaccine is already clearly evident among children eligible for vaccination, that is, infants <2 years old as at the end of 2009. The incidence of IPD in infants in this age group has halved in the last 2 years from 96.2 cases per 100 000 in 2007 to 46.4 per 100 000 in 2009. The reduction in rates of disease caused by PCV-7 serotypes was, as expected, more striking, with a 77% decrease from 78.8 cases per 100 000 in 2007 to 18.1 per 100 000 in 2009 in infants <2 years of age. The actual reductions in disease rates may be greater than these figures indicate, as the 2009 rates are based on IPD notifications whereas the rates for earlier years are based on case numbers captured by laboratory-based surveillance, which, compared with notifications, is likely to underestimate the burden of IPD.

Similar rapid reductions in IPD among infants have been observed in other countries following the introduction of PCV-7.[11,12] For example, in the United States there was a 69% reduction in IPD in infants <2 years old by the end of the second year following the introduction of the vaccine.[11]

So far there is no evidence of herd immunity, with little change or even increases in IPD in people ≥2 years of age. The all-age rate actually increased between 2007 and 2009 from 13.1 to 16.1 cases per 100 000. As noted above, the change in 2009 from laboratory-based surveillance of invasive pneumococci to surveillance based on IPD notifications makes absolute comparisons of rates in 2009 with those for earlier years difficult. Some of the apparent increase in 2009 may be due to the greater sensitivity of the notification-based surveillance system. On the other hand, there has also been an increase in cases captured by laboratory-based surveillance over the same time period: from 554 cases in 2007 to 631 in 2008 and 666 in 2009. These figures suggest there may well have been a real increase in IPD in recent years. However, as discussed in last year's report, referrals of invasive isolates to ESR for the laboratory-based surveillance of IPD may have been more complete since at least mid-2008 due to a heightened awareness of the disease following the introduction of PCV-7 to the national immunisation schedule and IPD becoming a notifiable disease.[6]

In this report for 2009, the analysis of the epidemiology of IPD in New Zealand has been extended to include, for the first time, analyses of IPD according to disease presentation, and the cases' ethnicity, deprivation levels and risk factors. This information has only become available since IPD became a notifiable disease.

The all-age rate of pneumococcal meningitis was 1.1 case per 100 000. The highest rate of meningitis occurred in the <1 year age group (14.3 per 100 000). Equal with pneumonia, meningitis was the most common IPD presentation among cases <1 year of age. There were no cases of pneumococcal meningitis in the 1-2 year age group.

In New Zealand, Maori and Pacific Peoples suffer a disproportionate burden of infectious disease. It seems this is also the pattern for IPD, with the rates for Maori and Pacific Peoples over three times that for Europeans. And correspondingly the rates of IPD were highest among the most deprived groups of the population. Similar associations have been reported in other countries, including higher rates among Australian aborigines and native Americans.[13,14]

As with all vaccines that target only specific types, there is concern that pneumococcal serotypes not included in PCV-7 will increase and essentially 'replace' vaccine types as the principal cause of IPD. This appears to have happened to some extent in several countries, although any increases in

disease due to non-vaccine types have usually been somewhat smaller than the reductions in disease due to vaccine types. Serotype 19A is the non-PCV-7 type most frequently reported to have increased.[15,16] Increases in 19A disease have been of particular concern as this serotype is often associated with antibiotic resistance.[17,18] As yet there has not been an increase in the rate of 19A disease in New Zealand. Moreover, serotype 19A isolates in this country are not especially associated with resistance.

However, since 2007 there has clearly been an increase in IPD due to another non-PCV-7 type: serotype 1. In 2009 this serotype was strongly associated with IPD in Pacific Peoples and Maori. The increase in serotype 1 may not be a result of serotype replacement following the introduction of PCV-7 since the increase in serotype 1 disease commenced in 2007, and in 2008 it was mainly associated with school-age children and young adults who were not eligible for PCV-7 vaccination. In addition, globally this serotype is often associated with outbreaks that occur cyclically every few years.[19] ESR's serotyping of pneumococci from 2010 IPD cases indicate that the prevalence of serotype 1 may already be declining.

As most resistant invasive pneumococci belong to one of the serotypes included in PCV-7, a decrease in IPD caused by vaccine types would be expected, and has been observed, to have the concomitant effect of reducing the incidence of IPD caused by resistant pneumococci.[20] This appears to be happening in New Zealand with both penicillin and cefotaxime resistance decreasing in 2009. There is no indication that resistance is increasing in non-PCV-7 serotypes in this country, with PCV-7 types still accounting for over 90% of the penicillin and cefotaxime resistance in 2009 as has been the trend in recent years.

Nearly one-tenth (59/697) of the IPD cases notified in 2009 did not include evidence that they met the case definition. PHUs need to ensure that the basis of the diagnosis is completed when IPD cases are notified and that this diagnosis meets the case definition, that is, pneumococci or pneumococcal DNA isolated or detected in a normally sterile site, or pneumococcal antigen detected in CSF using a newer generation antigen test.

The immunisation history was poorly reported with case notifications. It is hoped that, in future annual reports, information on the immunisation status of cases will be enhanced by matching notification data with data in the national immunisation register, rather than relying on information reported with the notification. In addition, it is hoped to include information of PCV coverage in future reports.

The addition of IPD to the schedule of notifiable diseases has enabled more comprehensive analysis of the epidemiology of this disease in New Zealand. This information will be more valuable in future years when trends can be analysed and multiyear analyses can provide sufficient numbers and power to undertake meaningful examination of associations with various demographic features, such as risk factors for IPD and the serotypes associated with certain disease presentations.

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APPENDIX 1. Case-fatality rates for invasive pneumococcal disease cases by age group, 2009

Age group (years)	Number of cases who died[1]	Case-fatality rate (%[2])	Number of cases for whom information on whether they survived or died was reported[3]
<1	1	3.6	28
1	0	-	24
2-4	0	-	38
5-14	0	-	51
15-64	10	3.7	273
≥65	24	11.3	212
All ages	35	5.6	626

1 Only includes cases for whom IPD was recorded as the primary cause of death.
2 Calculated on the basis of the number of cases for whom information on outcome was reported.
3 Outcome information available for 82.4% (28/34) of cases <1 year of age, 96.0% (24/25) of cases 1 year of age, 92.7% (38/41) of cases 2-4 years of age, 88.0% (51/58) of cases 5-14 years of age, 91.6% (273/298) of cases 15-64 years of age, and 88.0% (212/241) of cases ≥65 years of age.

APPENDIX 2. Risk factors among invasive pneumococcal disease cases <2 years of age, 2009

Risk factor	Number of cases for whom information on the risk reported	Number (%[1]) of cases with the risk
Premature <37 weeks gestation (cases <1 year of age)	15	5 (33.3)
Congenital or chromosomal abnormality	43	2 (4.7)
Chronic lung disease or cystic fibrosis	44	2 (4.5)
Anatomical or functional asplenia	41	0
Immunocompromised[2]	43	0
Chronic illness[3]	44	5 (11.4)
Cochlear implants	44	0
Smoking in household	23	9 (39.1)
In childcare	32	5 (15.6)
Resident in long-term or other chronic facility	47	0
Other risk factors	NA[4]	4 (6.8[5])

1 Percentage based on only those cases for whom information reported for each particular risk factor, except for 'Other' risk factors (see footnote 5).
2 Includes HIV/AIDS, lymphoma, organ transplant, multiple myeloma, nephrotic syndrome, chronic drug therapy, dysgammaglobulinaemia and sickle cell anaemia.
3 Includes CSF leak, intracranial shunts, diabetes, cardiac disease, pulmonary disease, chronic liver disease, renal impairment and alcohol-related disease.
4 Not applicable, as only reportable when case has 'Other' risk factors.
5 Percentage of all 59 IPD cases ≤2 years of age.

APPENDIX 3. Serotypes among invasive pneumococcal disease cases by age group, 2009

Serotype	Number (% within age group) of IPD cases due to each serotype:									
	<2 (n=55)		2-4 (n=38)		5-64 (n=342)		≥65 (n=230)		All ages (n=665)	
1	12	(21.8)	3	(7.9)	124	(36.3)	14	(6.1)	153	(23.0)
3	3	(5.5)	0		12	(3.5)	11	(4.8)	26	(3.9)
4	1	(1.8)	1	(2.6)	32	(9.4)	23	(10.0)	57	(8.6)
6 nt[1]	0		0		1	(0.3)	1	(0.4)	2	(0.3)
6A	2	(3.6)	1	(2.6)	5	(1.5)	5	(2.2)	13	(2.0)
6B	4	(7.3)	4	(10.5)	8	(2.3)	17	(7.4)	33	(5.0)
7 nt	0		0		1	(0.3)	0		1	(0.2)
7A	0		0		1	(0.3)	1	(0.4)	2	(0.3)
7F	1	(1.8)	0		13	(3.8)	4	(1.7)	18	(2.7)
8	0		0		8	(2.3)	4	(1.7)	12	(1.8)
9 nt	0		0		1	(0.3)	0		1	(0.2)
9N	0		2	(5.3)	4	(1.2)	2	(0.9)	8	(1.2)
9V	0		1	(2.6)	15	(4.4)	19	(8.3)	35	(5.3)
10A	0		0		2	(0.6)	2	(0.9)	4	(0.6)
11A	1	(1.8)	0		2	(0.6)	3	(1.3)	6	(0.9)
12F	0		0		1	(0.3)	1	(0.4)	2	(0.3)
13	1	(1.8)	0		0		0		1	0.2)
14	7	(12.7)	10	(26.3)	23	(6.7)	35	(15.2)	75	(11.3)
15 nt	1	(1.8)	0		0		0		1	(0.2)
15B	0		0		0		2	(0.9)	2	(0.3)
17F	0		0		0		2	(0.9)	2	(0.3)
18C	1	(1.8)	3	(7.9)	10	(2.9)	6	(2.6)	20	(3.0)
19A	8	(14.6)	4	(10.5)	16	(4.7)	9	(3.9)	37	(5.6)
19F	8	(14.6)	5	(13.2)	26	(7.6)	19	(8.3)	58	(8.7)
20	0		0		3	(0.9)	3	(1.3)	6	(0.9)
22 nt	0		0		0		1	(0.4)	1	(0.2)
22A	1	(1.8)	0		0		0		1	(0.2)
22F	1	(1.8)	0		11	(3.2)	10	(4.4)	22	(3.3)
23A	1	(1.8)	0		1	(0.3)	2	(0.9)	4	(0.6)
23F	2	(3.6)	3	(7.9)	16	(4.7)	27	(11.7)	48	(7.2)
31	0		1	(2.6)	0		0		1	(0.2)
33 nt	0		0		1	(0.3)	0		1	(0.2)
33F	0		0		1	(0.3)	3	(1.3)	4	(0.6)
34	0		0		1	(0.3)	1	(0.4)	2	(0.3)
35	0		0		1	(0.3)	1	(0.4)	2	(0.3)
37	0		0		0		1	(0.4)	1	(0.2)
38	0		0		0		1	(0.4)	1	(0.2)
non-typable	0		0		2	(0.6)	0		2	(0.3)

[1] nt, serogroupable but not serotypable