Influenza Surveillance in New Zealand 2010

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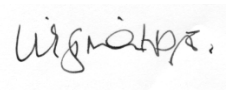

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Summary

National influenza surveillance in New Zealand is an essential public health component for assessing and implementing strategies to control influenza because influenza viruses frequently undergo antigenic changes and can cause substantial morbidity and mortality in a short space of time. The purpose of influenza surveillance in New Zealand aims at monitoring incidence and distribution of influenza in the community, early detection of influenza epidemics and identifying the predominant circulating strains. This report summaries the community disease burden of influenza, the circulating influenza virus strains, hospitalisation, mortality and immunisation coverage in 2010.

During the 2010 winter season, 4112 consultations for influenza-like illness (ILI) were reported from a national sentinel network of 91 general practices. It is estimated that ILI resulting in a visit to a general practitioner affected over 50,561 New Zealanders (1.2% of total population) during the season, compared with an estimated 116,335 people in 2009 (2.7% of total population).

Influenza activity peaked in August and overall ILI activity in 2010 was at a medium level compared with the 1997–2009 period. ILI consultation rates varied greatly among District Health Boards (DHBs) and the highest rates were reported from the Waikato and Hawke's Bay DHBs. In 2010, the vast majority of the viruses were influenza A (99.5%). Among all typed and sub-typed viruses, pandemic A(H1N1) 09 viruses (99.1%) were the predominant strain with a small number of seasonal A(H3N2) (12) and influenza B (10) viruses co-circulating during the season.

All pandemic A(H1N1) 09 viruses tested were sensitive to oseltamivir. No significant antigenic drift for influenza A(H1N1), A(H3N2) and B viruses was observed among circulating influenza viruses and no updates were required for the three components of the influenza vaccine for 2011.

1. Introduction

It is known that influenza viruses frequently undergo antigenic changes, evading host immune response. This poses a real challenge in the prevention and control of influenza. The overarching goal of influenza surveillance is to provide information to public health authorities to facilitate appropriate control and intervention measures, health resource allocation and case management, thereby minimising the impact of influenza on people. Non seasonal influenza (capable of being transmitted between human beings) became a notifiable and quarantineable disease in New Zealand on 30 April 2009. Seasonal influenza is not a notifiable disease in New Zealand. Since 31 December 2010, pandemic influenza A(H1N1) 09 has been classified as seasonal.

The purpose of influenza surveillance is:

- to understand the incidence and distribution of influenza in the community
- to assist with early detection of influenza epidemics within the community and to guide the development and implementation of public health measures
- to identify the predominant circulating strains in the community and guide the composition of the influenza vaccine for the subsequent year[1].

This report summarises the results obtained from influenza surveillance in New Zealand for 2010, and includes some comparisons with previous years. It also includes information on hospitalisations for influenza (obtained from the Ministry of Health's National Minimum Dataset (NMDS), notifiable disease information for non-seasonal influenza (obtained from EpiSurv) and influenza immunisation coverage data (obtained from Health Benefits Limited).

2. Methods

2.1. General Practice Sentinel Surveillance – Epidemiology and Virology Data

The sentinel surveillance system, in its current form, began in 1991 as part of the World Health Organization's (WHO) Global Programme for Influenza Surveillance. It is operated nationally by the Institute of Environmental Science and Research (ESR) Ltd and locally by influenza surveillance co-ordinators in the public health services (PHSs). Sentinel surveillance usually operates in the winter, from May to September, however, the sentinel time period was extended from January to September 2010 (week 1 to week 39, inclusive) due to pandemic influenza. Local surveillance co-ordinators recruited general practices within their region to participate on a voluntary basis. Where possible, the number of practices recruited was proportional to the size of the population in each DHB covered by the PHS (approximately 1:50,000 population).

General practitioners (GPs) were required to record the number of consultations for influenza-like illness (ILI) each week and the age group in years (<1, 1–4, 5–19, 20–34, 35–49, 50–64, 65+) of each of these suspected cases on a standardised form.

ILI was defined by a standardised case definition, which is, 'acute upper respiratory tract infection characterised by abrupt onset and two of the following: fever, chills, headache, and myalgia.' [2]

Each participating GP also collected three respiratory samples (ie, a nasopharyngeal or throat swab) each week from the first ILI patient examined on Monday, Tuesday and Wednesday of the week. In 2010, a general practice clinic with a registered patient population of more than 10,000 people, was required to collect a total of six respiratory samples each week from the first two ILI patients examined each on Monday, Tuesday and Wednesday. The GPs forwarded these samples to the WHO National Influenza Centre (NIC) at ESR or to hospital virology laboratories in Auckland, Waikato or Christchurch for virus characterisation. Laboratory identification included molecular detection using the polymerase chain reaction (PCR), isolation of the virus or direct detection of viral antigen. Influenza viruses were typed and subtyped as A, B, seasonal A(H1N1), seasonal A(H3N2) or pandemic A(H1N1) 09.

Information on the number of ILI consultations and swabs sent from each DHB was forwarded to ESR each week (Monday to Sunday) by local co-ordinators. ILI consultation data were received by Wednesday of the following week. Likewise, virology laboratories reported to ESR weekly with the total number of swabs received from each DHB, the influenza viruses identified and updated details on types and strains. ESR reports national information on epidemiological and virological surveillance of influenza weekly, monthly and annually to relevant national and international organisations, including the WHO, and it publishes the results on the website: http://www.surv.esr.cri.nz/virology/influenza_weekly_update.php

Consultation rates were calculated using the sum of the patient populations, reported by the participating practices, as the denominator. From 1992–2009, the denominator for the age-specific ILI rate calculation was based on New Zealand census data with the assumption that age distribution of the GP patient population was the same as the New Zealand population, because the age-specific patient population data were not provided by the participating

practices. Since 2010, age-specific ILI consultation rate calculations have been based on the age-specific patient populations as the denominator.

The national level of ILI activity is described using a set of threshold values[3,4]. A weekly rate of below 50 consultations per 100,000 patient population is described as baseline activity. A weekly consultation rate of 50–249 per 100,000 patient population per week is indicative of normal seasonal influenza activity. Within the normal seasonal activity, 50–99 consultations per 100,000 patient population per week is low activity, 100–149 consultations per 100,000 patient population per week is moderate activity and 150–249 consultations per 100,000 patient population per week is high activity. A rate of 250–399 consultations per 100,000 patient population per week indicates higher than expected influenza activity and ≥400 consultations per 100,000 patient population per week indicates an epidemic level of disease. As general practices are not uniformly spread throughout the population, this may affect the representativeness within some DHBs.

2.2. Virological Surveillance for Outpatients and Hospital Inpatients – Non-sentinel Surveillance

In addition to influenza viruses identified from sentinel surveillance, year-round laboratory surveillance of influenza (and other viruses) is carried out by the four regional virus diagnostic laboratories at Auckland, Waikato, Wellington and Christchurch Hospitals, and by the WHO NIC at ESR. This type of surveillance is called non-sentinel surveillance. Each week, all viral identifications, including influenza, and largely from outpatient and hospital inpatient clinics during routine viral diagnosis, are reported to the NIC at ESR. ESR, in turn, collates and reports virology surveillance data nationally.

The NIC at ESR (previously the National Health Institute, New Zealand Communicable Disease Centre) was designated by the New Zealand Ministry of Health and recognised by the WHO in 1954. Since that time, the NIC at ESR has been a key point of contact for both the WHO and Ministry of Health regarding virological and epidemiological surveillance of influenza. The NIC provides influenza virus isolates to the WHO Global Influenza Surveillance Network, reference testing for hospital laboratories including antigenic and genetic typing and oseltamivir susceptibility testing. The NIC collates year-round national laboratory testing information on all influenza-positive cases, including basic demographics. Most influenza viruses are forwarded to the WHO Collaborating Centre (WHOCC) in Melbourne for further characterisation.

2.3. Non-seasonal Influenza Notifications

Non seasonal influenza became a notifiable and quarantineable disease in New Zealand on 30 April 2009. In 2009–2010 this meant notifying cases of pandemic A(H1N1) 09. Since 31 December 2010, pandemic influenza A(H1N1) 09 has been classified as seasonal. Data are entered into a national web-based database (EpiSurv) operated by ESR and are available for immediate analysis. This system also records hospitalised and fatal cases of influenza. Data derived from EpiSurv as of 17 February 2011 are presented in Section 4.

2.4. Hospitalisations

Hospitalisation data for influenza (ICD-10AM-VI code I (J09-J11) for 2010 which correlate with previous versions of ICD-10AM codes J10-J11), were extracted from the New Zealand Ministry of Health's NMDS (by discharge date). In this dataset, people who received less than 1 day of hospital treatment in hospital emergency departments were excluded from any time series analysis of influenza hospitalisations during 2000–2010. Influenza-related hospitalisations were conservatively taken to include only those cases where influenza was the principal diagnosis. Repeat admissions were included, as infections with another influenza A subtype or B virus are possible.

2.5. Data used to Calculate Rates

Denominator data used to determine rates of ILI, hospitalisations, mortality, immunisation coverage have been derived from 2010 mid-year population estimates published by Statistics New Zealand.

2.6. Immunisation Coverage

In 1997, influenza vaccination was made available free to those aged 65 years and older, and in 1999, free vaccination was extended to all pregnant women and those people aged under 65 years and at high risk of complications from influenza[5,6].

Anyone aged under 65 years with any of the medical conditions in the list that follows, is eligible for free influenza vaccinations:

- Cardiovascular disease (ischaemic heart disease, congestive heart failure, rheumatic heart disease, congenital heart disease, cerebrovascular disease)
- Chronic respiratory disease (asthma if on regular preventive therapy, other respiratory disease with impaired lung function)
- Diabetes
- Chronic renal disease
- Cancer (patient currently has cancer), excluding basal and squamous skin cancers if not invasive
- Other conditions (autoimmune disease, immunosuppression, human immunodeficiency virus, transplant recipients, neuromuscular and central nervous system diseases, haemoglobinopathies, children on long-term aspirin therapy)

The data that medical practitioners provide to Health Benefits Limited to claim reimbursement were used to estimate immunisation coverage in 2010 among persons 65 years of age or older.

3. Seasonal Influenza Surveillance

3.1. Sentinel Practices

In 2010, 91 sentinel practices were recruited from all 20 DHBs. All PHSs began reporting by the fifth week (2 February 2010). Some sentinel practices did not report every week. The average number of practices participating per week was 81, with an average patient population roll of 355,222 approximately 8.1% of the New Zealand population.

3.2. Disease Burden

From January to September 2010, a total of 4112 sentinel consultations for ILI were reported. The cumulative incidence rate of ILI consultations for May to September 2010 during the influenza season was 1035.6 per 100,000 patient population. The average national weekly consultation rate in 2010 was 49.3 per 100,000 patient population. This rate is lower than the average weekly rates for 2009 (106.1 per 100,000) and 2008 (52.4 per 100,000).

Extrapolating ILI consultations obtained from the general practice patient population to the New Zealand population, it is estimated that ILI resulting in a visit to a GP affected 50,561 New Zealanders during the influenza season (1.2% of total population). This is lower than the estimated 116,335 people affected in 2009 and 50,550 people affected in 2008.

Figure 1 compares the weekly consultation rates for ILI in 2010 with weekly consultation rates for ILI in 2009 and 2008. Influenza consultation activity remained at the baseline level from week 1 to week 29, and then increased to a peak in week 33 (15–22 August 2010), with a consultation rate of 151.6 per 100,000 patient population. This occurred four weeks later than the peak in 2009 and the first peak in 2008, both of which occurred in week 29 (284.0 and 93.3 per 100,000 patient population, respectively). The 2010 peak occurred at the same time as the second peak in 2008, the latter having a consultation rate of 95.2 per 100,000 patient population. Consultation activity then gradually declined in 2010, remaining at a moderate level until week 36, and dropping below the baseline consultation rate in week 37.

Figure 1. Weekly Consultation Rates for ILI in New Zealand, 2008, 2009 and 2010

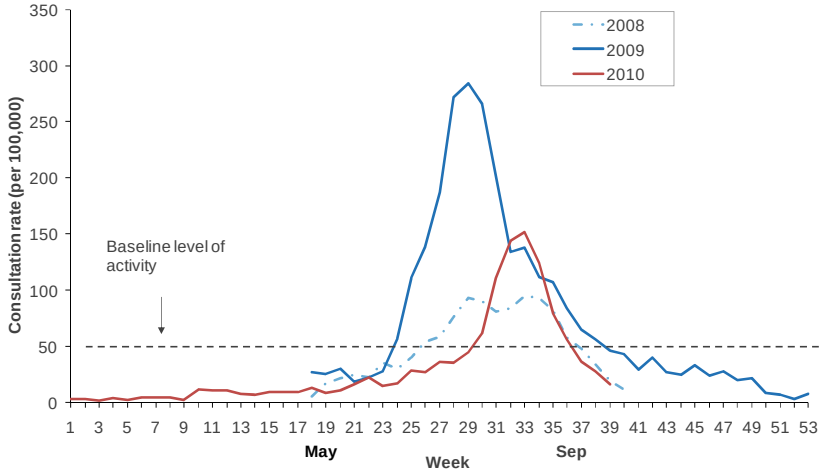


Figure 2. Total Number of Influenza Viruses Detected by Surveillance Type and Week Specimen taken, 2010

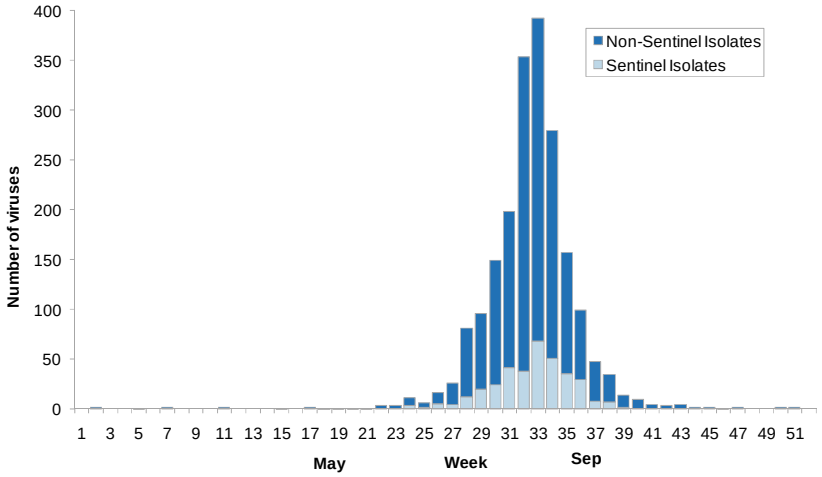
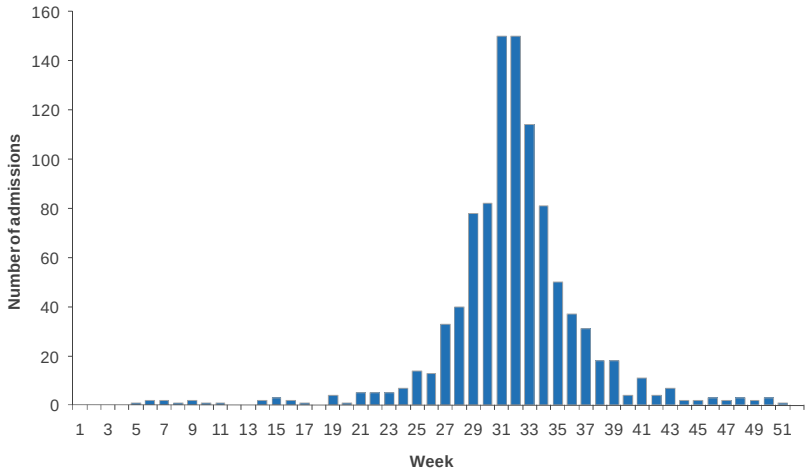


Figure 3. Influenza Hospitalisations by Week Discharged, 2010



A total of 2012 influenza viruses were identified in 2010, this is lower than the 4900 viruses identified in 2009 and higher than 1054 viruses seen in 2008. Of the 2012 viruses, 349 came from sentinel practice surveillance during January to September. This is lower than the 624 sentinel viruses identified in 2009 and lower than 466 viruses identified in 2008. There were 1663 non-sentinel viruses identified in 2010 compared with 4276 in 2009 and 588 in 2008.

Figure 2 shows the numbers of influenza viruses detected each week throughout 2010. The highest peak of influenza virus detection occurred in week 33 (393 viruses), which corresponds to the peak period for consultation rates. Sporadic influenza viruses were identified as early as January during the summer season, however, the vast majority (1970, 97.9%) were from specimens taken from June to September 2010. Sentinel and non-sentinel virus numbers peaked in week 33 (68 and 393 viruses, respectively). Overall, influenza viruses were detected in the same time period in 2010 as they were in 2009. Most sentinel and non-sentinel viruses (96.6%) were identified during the sentinel period (weeks 26–39).

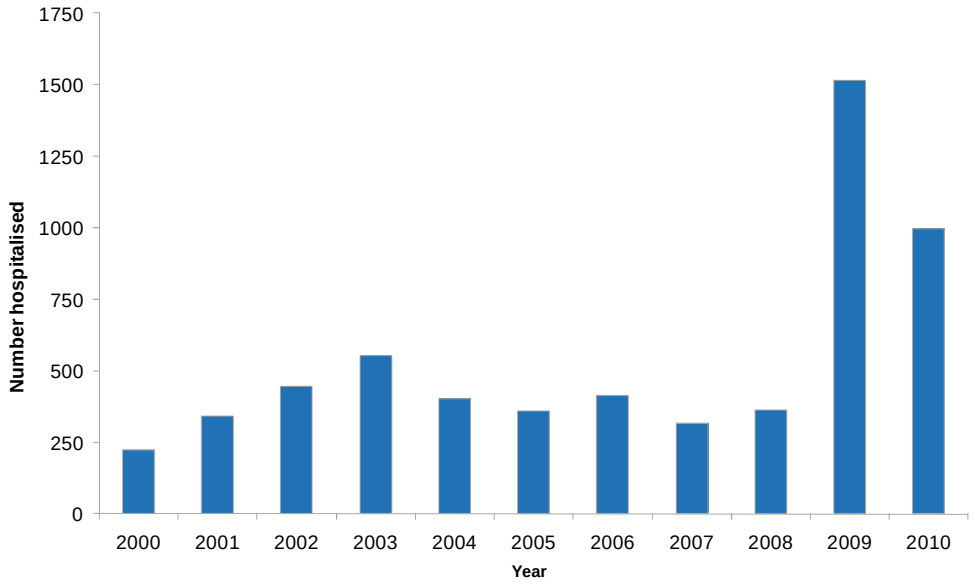
In 2010, there were a total of 998 hospitalisations for influenza, which is lower than the 1517 hospitalisations reported in 2009, but higher than the 2008 and 2007 hospitalisations of 365 and 316, respectively.

Figure 3 shows influenza hospitalisations by week discharged and indicates that 95.4% (952) of these occurred from June to October. The highest number of hospitalisations (517) occurred in August. Hospitalisations peaked in weeks 31 and 32, while sentinel and non-sentinel influenza virus detections and ILI consultations peaked in week 33.

When influenza hospitalisation data in 2010 were compared with the data from 2000–2008 (Figure 4), a substantially higher number of hospitalisations occurred in 2009 and 2010 than in previous years.

Most influenza hospitalisations in 2010 were for pandemic A(H1N1) 09 infection. See section 4 on non-seasonal influenza surveillance.

Figure 4. Influenza Hospitalisations, 1990–2010



3.3. Geographic Distribution

In addition to providing influenza incidence figures at a national level, sentinel surveillance provides information on the geographic distribution of ILI and viral strains at a regional level.

Figure 5 shows the sentinel average weekly consultation rates for each DHB from May to September 2010. A heterogeneous distribution of influenza activity among different geographical locations in New Zealand was observed, with some regions (mainly small urban and rural areas) that had relatively low levels of ILI activity in 2009 experiencing higher levels of activity during the second wave in 2010. Weekly ILI consultation rates varied among DHBs, with rates above the national average in Waikato (87.6 per 100,000 patient population), followed by Hawke's Bay (82.6), Lakes (75.4), Hutt (68.7), South Canterbury (68.7), Bay of Plenty (67.3), Capital and Coast (59.7), Canterbury (53.9) and Northland (51.3).

Figure 5. Sentinel Average Weekly Consultation Rates for Influenza by DHB, 2010

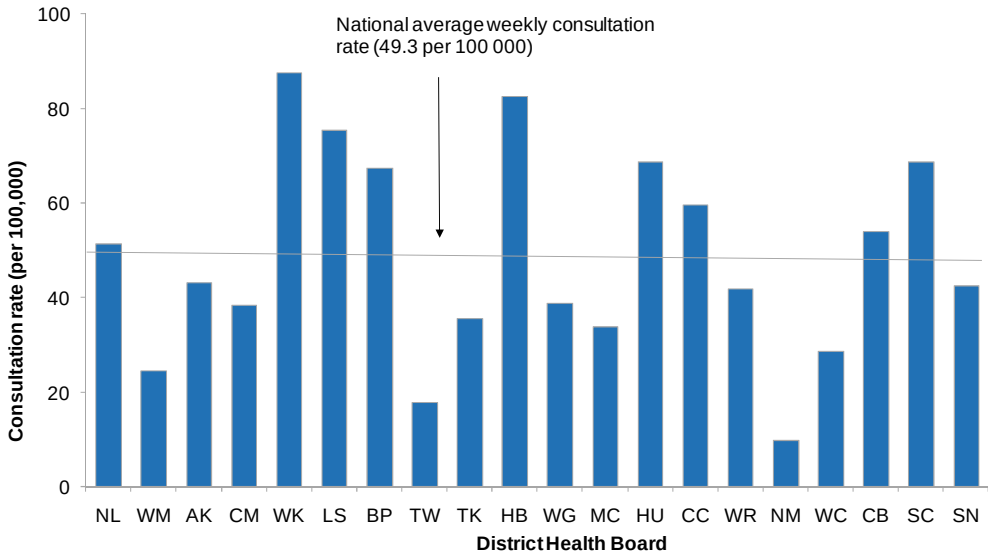


Table 1 shows the DHB codes and their descriptions.

Table 1. DHB Codes and Descriptions

DHB code	DHB
NL	Northland
WM	Waitemata
AK	Auckland
CM	Counties Manukau
WK	Waikato
LS	Lakes
BP	Bay of Plenty
TW	Tairawhiti
TK	Taranaki
HB	Hawke's Bay
WG	Whanganui
MC	MidCentral
WR	Wairarapa
HU	Hutt Valley
CC	Capital and Coast
NM	Nelson Marlborough
WC	West Coast
CB	Canterbury
SC	South Canterbury
SN	Southern

Figure 6 shows the distribution of sentinel influenza viruses based on the DHB from which the specimen (swab) was taken. Most viruses came from Canterbury and Auckland DHBs. Viruses were not identified in Counties Manukau DHB (only one practice). The national influenza virus detection rate for 2010, illustrated in Figure 7, was 36.1% (349 viruses from 966 swabs received), which is higher than the 2009 (31.3%, 624 viruses from 1993 swabs) but lower than the 2008 detection rates (46.6%, 466 viruses from 1001 swabs).

Figure 6. Cumulative Numbers of Laboratory-confirmed Influenza Viruses from Sentinel Surveillance by DHB, May to September 2010

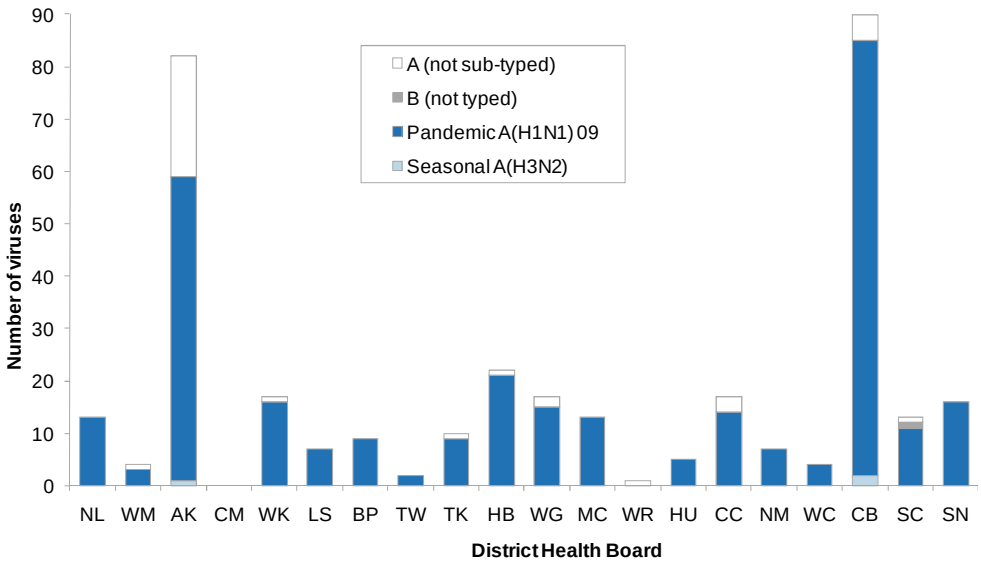
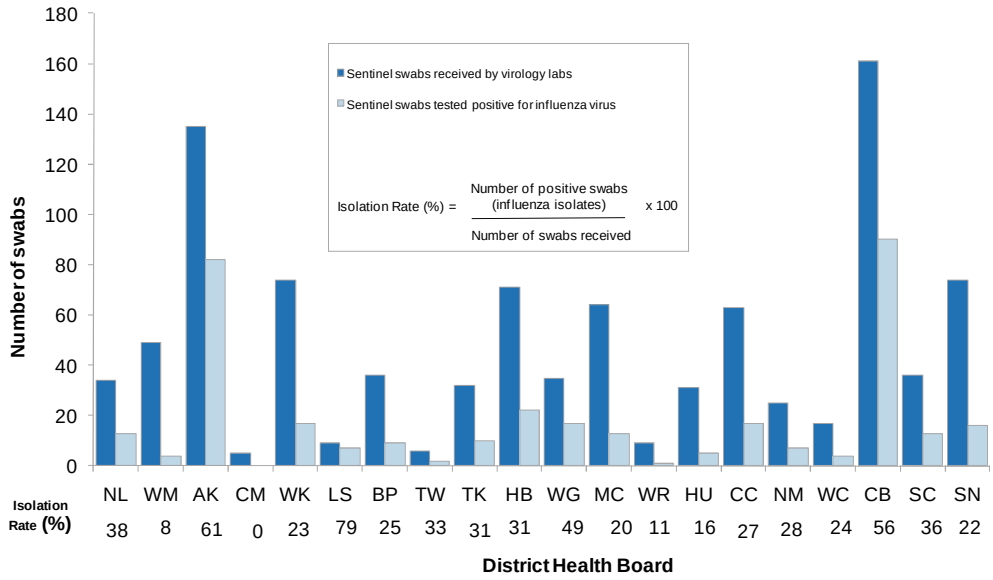


Figure 7. Sentinel Swabs Received and Tested Positive for Influenza Virus by DHB, 2010



NB: Viruses from the Auckland region were assigned to Auckland DHB if DHB was not recorded.

3.4. Age Distribution

Figure 8 compares the hospitalisation rates in 2010 by age group. In 2010, the highest hospitalisation rates occurred in children aged under 1 year (125.5 per 100,000 patient population), followed by children aged 1–4 years (49.2 per 100,000) and adults aged 20–34 years (25.4 per 100,000).

Figure 8. Influenza Hospitalisation Rates by Age Group, 2010

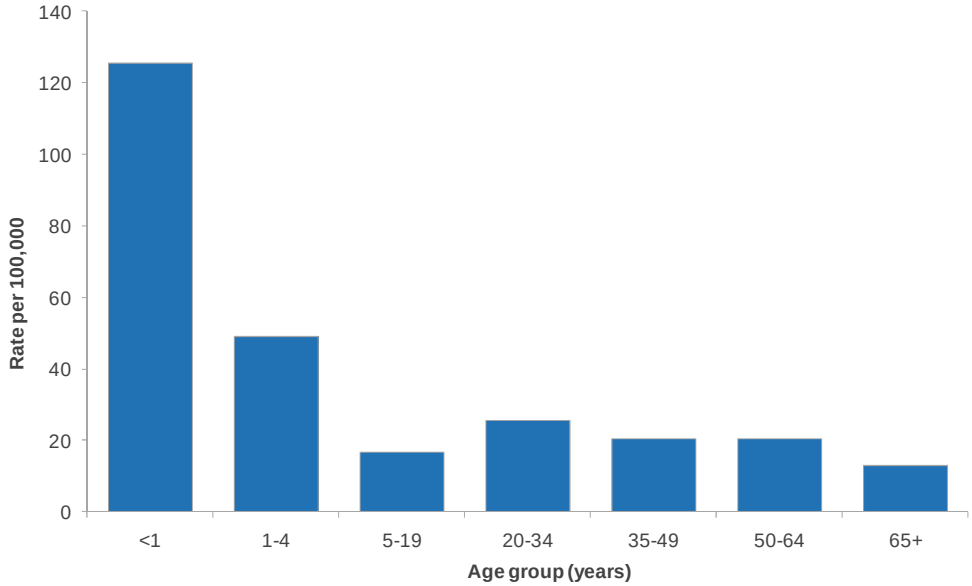
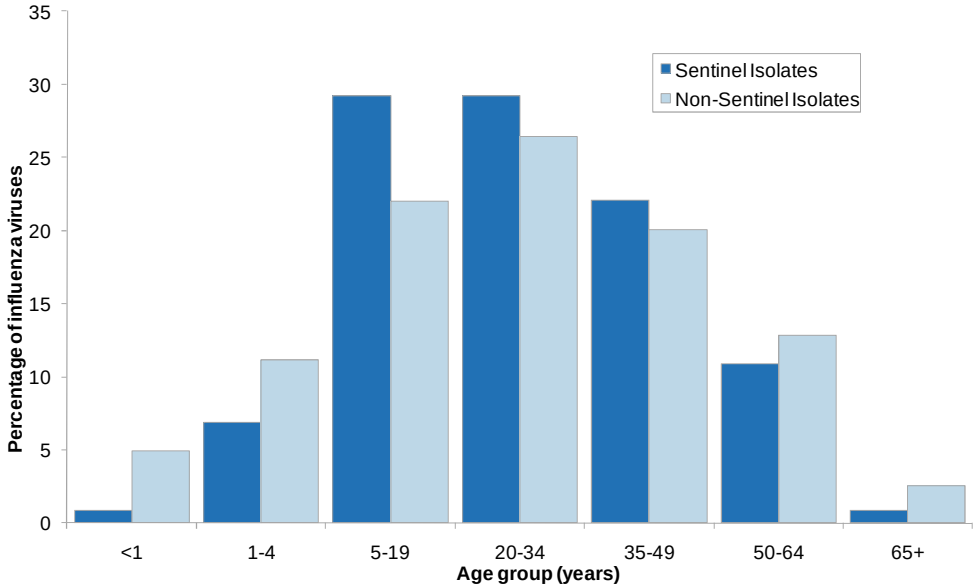


Figure 9 compares the percentage of influenza viruses detected from sentinel surveillance and non-sentinel surveillance for each age group. People aged under 1 year, 1–4 years and 65 years and older were represented more in non-sentinel surveillance than in sentinel surveillance. This is consistent with findings from the past 10 years. It may reflect the fact that influenza presents more severely in the very young and elderly populations, resulting in

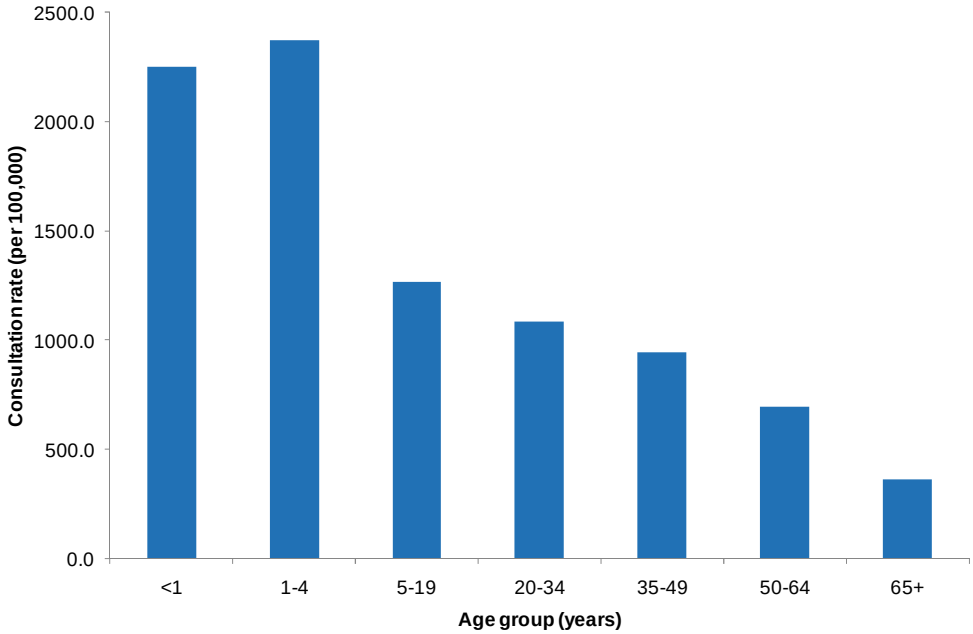
hospitalisations, or it may reflect a greater reluctance among sentinel GPs to take swabs from very young children and elderly patients. In 2009 and 2010, 50–64 year-old patients were also represented more in non-sentinel surveillance and this differs from findings in previous years.

Figure 9. Percentage of Sentinel and Non-sentinel Influenza Viruses by Age Group, 2010



Average weekly ILI consultation rates by age group were calculated for the sentinel surveillance system (Figure 10). The highest consultation rate for ILI was in children aged 1–4 years and those aged less than 1 year, with a cumulative consultation rate of 2370.7 and 2252.5 per 100,000 patient population, respectively. This was followed by patients aged 5–19 years (1267.0), 20–34 years (1085.1), 35–49 years (944.3) and 50–64 years (969.6). Elderly people (aged 65 years and older) had the lowest ILI consultation rate of 362.9 per 100,000 patient population.

Figure 10. Sentinel Average Weekly Consultation Rates for ILI by Age Group, 2010

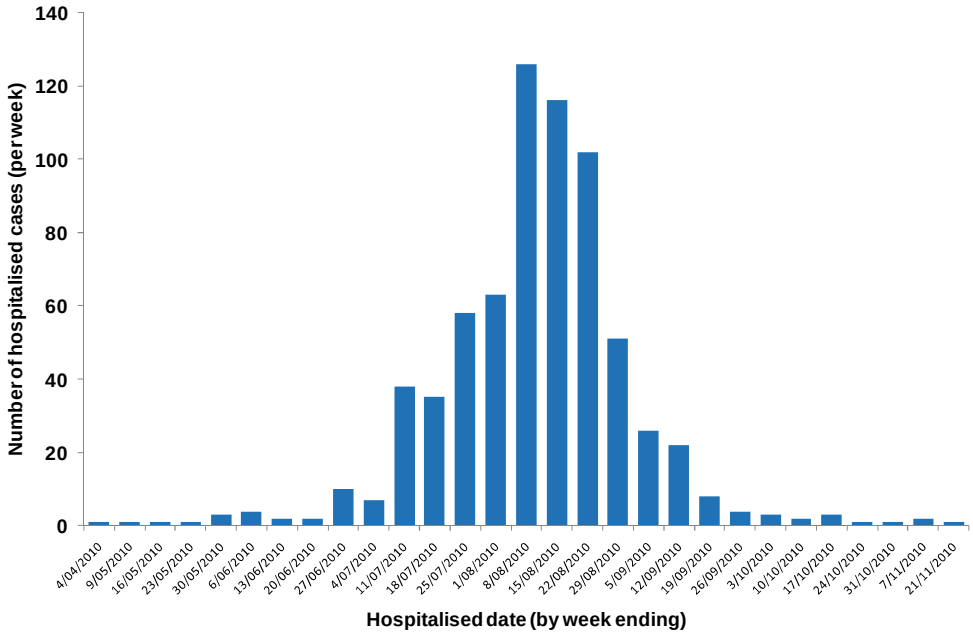


4. Non-seasonal Influenza Surveillance

Non seasonal influenza (pandemic A(H1N1) 09) became a notifiable disease on 30 April 2009. In 2010, a total of 1826 cases were reported in EpiSurv as follows, 1801 confirmed, 24 probable and one under investigation.

The epidemic curve for pandemic A(H1N1) 09 in 2010 is shown in Figure 11. This epidemic curve was constructed using the earliest date recorded in EpiSurv (onset, hospitalised or report date) and is displayed as cases per week (Monday to Sunday).

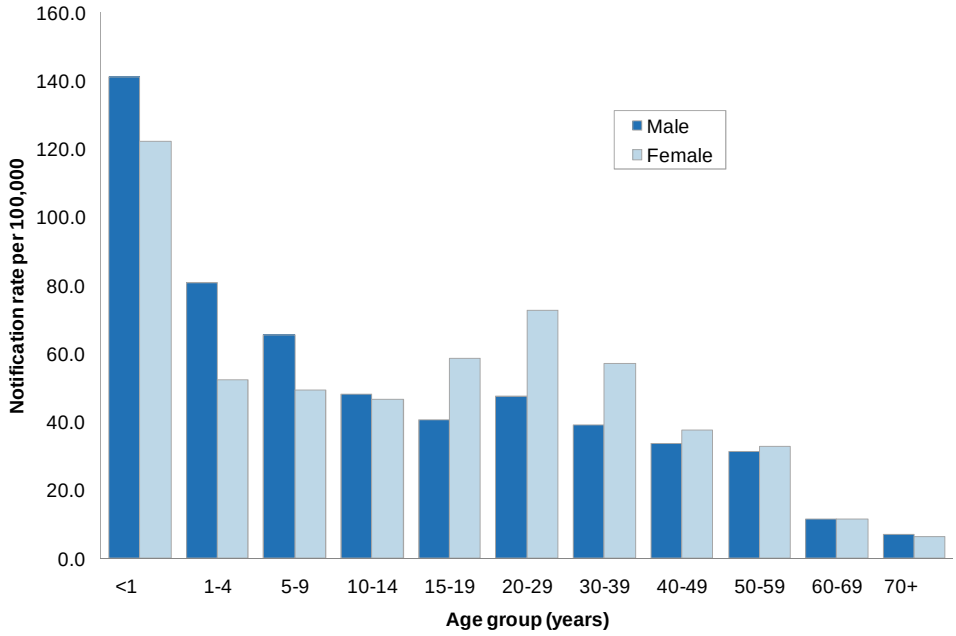
Figure 11. Total Cases of Pandemic A(H1N1) 09, 2010



Confirmed cases n = 1801, probable cases n = 24 and under investigation case n = 1

The age distribution of cases by gender is shown in Figure 12. The highest reported notification rate was in the under 1-year-old age group, followed by 1–4 year-old patients.

Figure 12. Cumulative Rate of Pandemic A(H1N1) 09, 2010 Cases by Age and Sex

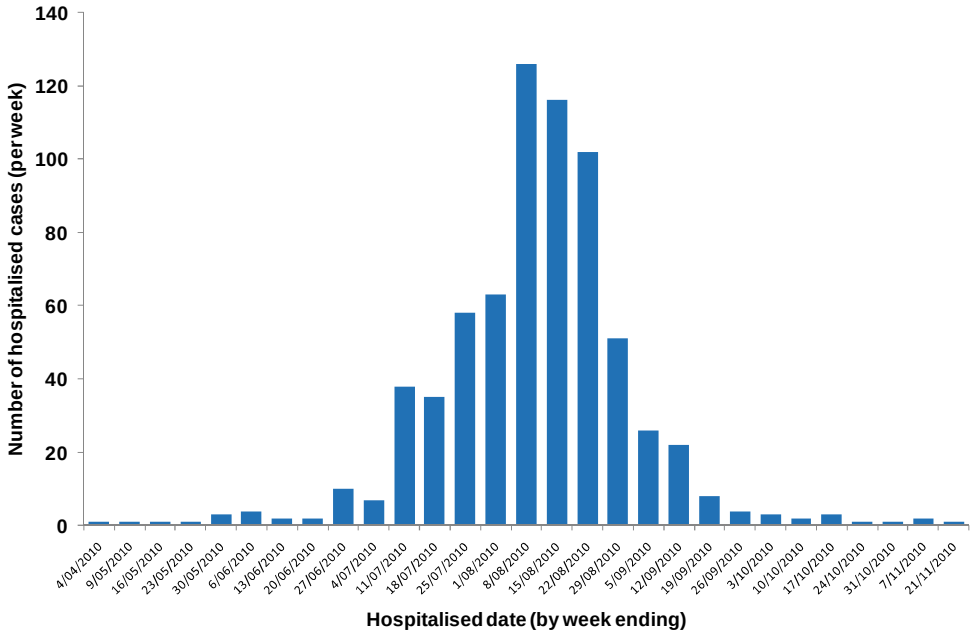


There were 746 cases reported as hospitalised from pandemic A(H1N1) 09 infection in EpiSurv in 2010. The median age was 27.0 years, and ranged from 13 days to 86 years. A total of 126 hospitalisations were reported in week 31 (2–8 August) which was two weeks earlier than the peak in notifications (week 33) (Figure 13).

The age distribution of notifications and hospitalisations for pandemic A(H1N1) 09 infections in 2010 was very similar to that in 2009 (Figure 14). Like 2009, the highest cumulative rates of notification and hospitalisation were in children under 5 years of age (80.2 and 51.3 cases per 100,000 population, respectively). The overall notification rate in 2010 was just over half of the 2009 rate. Overall hospitalisation rates were about one-third lower in 2010 compared with 2009. Notification and hospitalisation rates declined from 2009 to 2010 in all age groups, with greater reductions in patients aged 5–19 years, 0–4 years and 20–39 years.

The ethnic distribution of notifications and hospitalisations due to pandemic A(H1N1) 09 infection in 2010 was different from the one in 2009. Although the highest notification rates for pandemic A(H1N1) 09 were seen in Pacific and Māori populations in 2009, their rates dropped substantially compared with the Other groups. Similarly, hospitalisation rates due to pandemic A(H1N1) 09 infection dropped considerably among Pacific and Māori populations, though the 2010 rate remained highest among the Pacific peoples (Figure 14). Compared with the European ethnic group, the hospitalisation rate ratio for Pacific peoples in 2010 was 1.6 (95% confidence interval (CI): 1.3–2.0). This is much lower than the hospitalisation rate ratio of 3.5 (95% CI: 3.0–4.2) in 2009. The Māori population hospitalisation rate did not differ from the European ethnic group in 2010, with a rate ratio of 1.0 (95% CI: 0.8–1.2); although this is lower compared with 1.6 (95% CI: 1.4–1.9) in 2009.

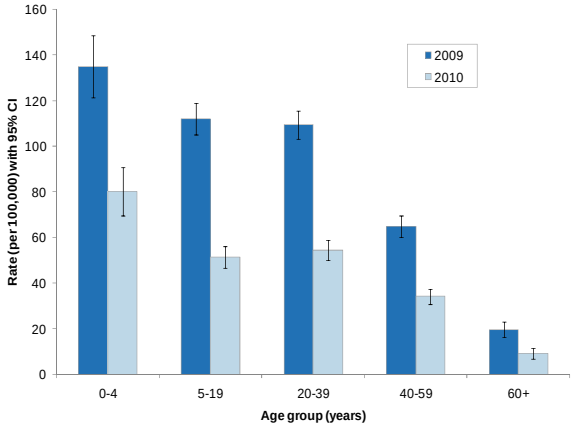
Figure 13. Hospitalisations of Confirmed Pandemic A(H1N1) 09, 2010



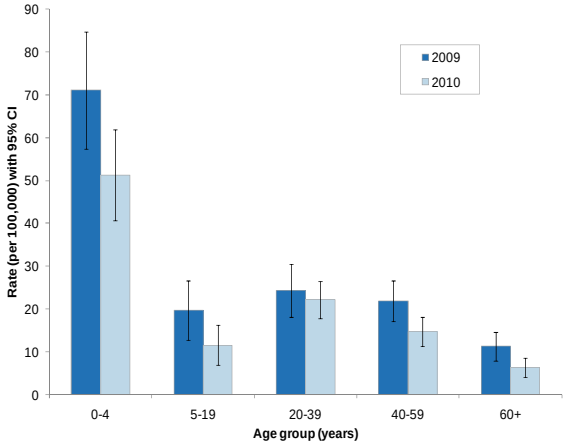
Note: There were 52 hospitalised cases with no hospitalised date recorded (excluded from the graph).

Figure 14. Notification and Hospitalisation Rates for Pandemic A(H1N1) 09 by Age Group (A, B) and Ethnicity (C, D), Stratified by year, 2009 and 2010

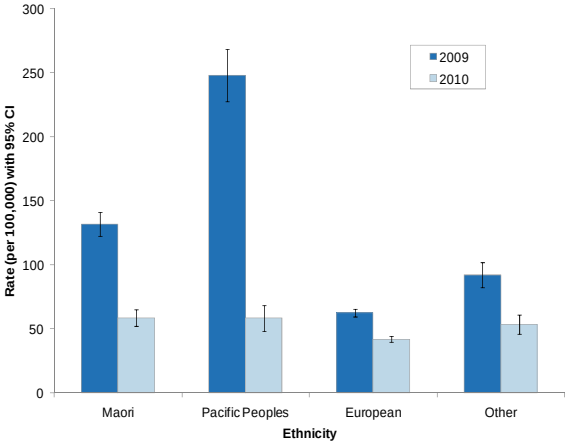
A. Notifications by Age Group



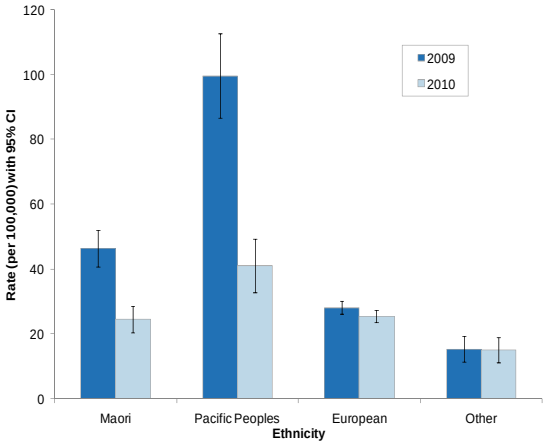
B. Hospitalisation Rates by Age Group



C. Notification Rates by Ethnicity (Age-adjusted)



D. Hospitalisation Rates by Ethnicity (Age-adjusted)

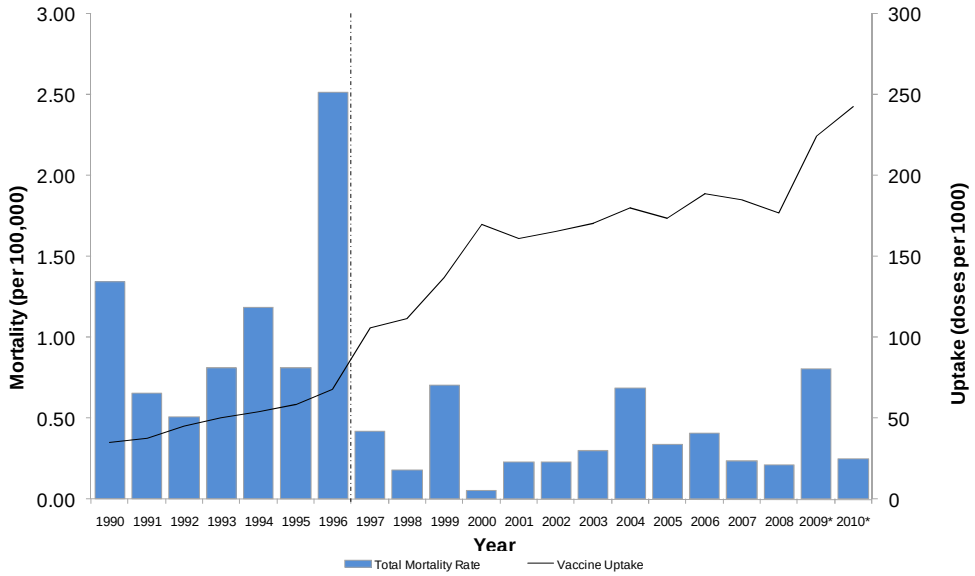


95%CI= Rates +/- margin error (II*SE)

At the time of writing this report, 16 deaths had been reported in New Zealand as pandemic A(H1N1) 09-associated deaths[6]. The median age was 51 years, and ranged from 15 to 86 years of age. This gave rise to the mortality rate of 0.37 per 100,000 for 2010, which is lower than 0.81 per 100,000 in 2009. When the 2009 influenza mortality rate was compared with that of 1990–2007 (Figure 15), the 2009 mortality rate was the fifth highest rate recorded from 1990–2010. The first (2.52 per 100,000) and second (1.34 per 100,000) highest mortality rates were recorded in 1996 and 1990, respectively.

[6] A pandemic influenza A(H1N1) 09-associated death is defined as a person with confirmed pandemic A influenza (H1N1) 09 infection determined from ante-mortem or post-mortem specimens, and who died from a clinically compatible illness or complications attributable to that infection. There should be no period of complete recovery between illness and death, and no alternative agreed upon cause of death.

Figure 15. Influenza Mortality Rates and Vaccine Uptake, 1990–2010



*2009 and 2010 mortality data are from EpiSurv.
Note: In 1997, the Ministry of Health made influenza vaccination available free to persons aged 65 years and older. In 1999, this policy was extended to at risk groups <65 years old. 2007 mortality data are provisional.

5. Immunisation Coverage

The uptake of seasonal influenza vaccine in New Zealand in 2010 increased substantially. The number of doses of influenza vaccine, both publically and privately funded used during the 2010 seasonal influenza programme was 243 doses per 1000 population, 9% higher than the 224 doses per 1000 population 2009. The overall uptake rate for funded vaccine among persons 65 years and older was 63.5%, up from 60.5% in 2009 (Immunisation benefit claims data, Ministry of Health 2010). It is not possible to estimate vaccine uptake for persons under 65 years as the 2010 Influenza programme was extended to include pregnant women and children less than 5 years. Pandemic influenza vaccine doses administered as part of the early protection programme in 2010 are not included.

At least 1,046,000 doses of the seasonal trivalent influenza vaccine were distributed in New Zealand in the 2010 season. Over 624,000 claims had been received up to the end of October 2010 for the subsidised programme. In 2010, a considerable number of doses must have been purchased privately to explain stock exhaustion and the need for replenishment. Table 2 shows the estimated numbers of people that received immunisation for five age groups.

Table 2. Influenza Immunity by Age Group, 2010

Age group (years)	Immunity 2010 (pre-second wave)
1–4	30 023 (10.0%)
5–19	27 523 (3.0%)
20–39	44 089 (3.8%)
40–59	105 968 (9.2%)
60+	416 832 (55.6%)

6. Virus Strain Characterisation

6.1. Circulating Viral Strains in 2010

Figure 16 shows influenza virus identifications by type and subtype for each week throughout 2010, and the total percentage contribution of each. Table 3 shows influenza virus identifications by type and subtype for 2010.

The majority of influenza viruses (2002/2012 or 99.5% of all viruses) were characterised as influenza A. A very small number of influenza B viruses (10) were detected and represented 0.5% (10/2012) of all viruses.

Overall, the pandemic A(H1N1) 09 virus was the predominant strain among all influenza viruses. The pandemic A(H1N1) 09 strain represented 89.9% (1808/2012) of all viruses and 98.8% (1808/1830) of all typed and subtyped viruses.

The seasonal influenza A(H3N2) strain represented 0.6% (12/2012) of all viruses and 0.7% (12/1830) of all typed and subtyped viruses.

Figure 17 shows the general pattern of influenza virus identifications. This indicates the early onset of ILI activity, followed by a rapid rise to the peak in week 33. The majority of influenza A viruses occurred in the middle of the season. Pandemic A(H1N1) 09 viruses predominated for most of the influenza season (from week 18 to week 40).

Table 3. Influenza Virus Identifications by Type and Subtype, 2010

Viruses	No. viruses (%)	Typed/Sub-typed (%)
Influenza A		
A (not sub-typed)	182 (9.0)	
Pandemic A(H1N1) 09		
Pandemic A(H1N1) 09 by PCR	1270 (63.1)	1270 (69.4)
A/California/7/2009 (H1N1) – like	538 (26.7)	538 (29.4)
Subtotal pandemic A(H1N1) 09	**1808 (89.9)**	**1808 (98.8)**
Influenza A(H3N2)		
Seasonal A (H3N2) by PCR	8 (0.4)	8 (0.4)
A/Perth/16/2009 (H3N2) – like	4 (0.2)	4 (0.2)
Subtotal seasonal A(H3N2)	**12 (0.6)**	**12 (0.7)**
Influenza B		
B by PCR	6 (0.3)	6 (0.3)
B/Brisbane/60/2008 – like	4 (0.2)	4 (0.2)
Subtotal B	**10 (0.5)**	**10 (0.5)**
Total	**2012 (100)**	**1830 (100)**

Figure 16. Total Influenza Viruses by Type and Week Specimen taken, 2010

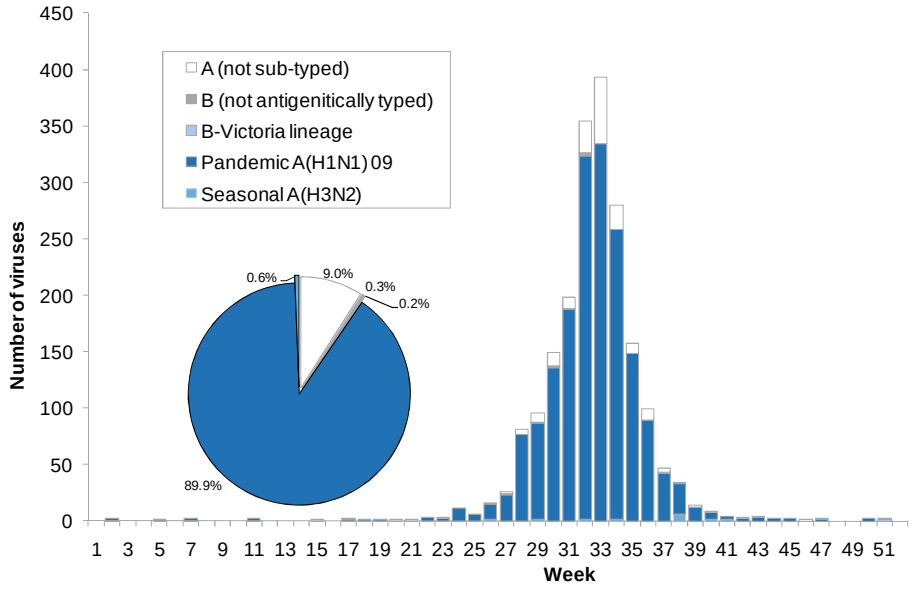


Figure 17. Total Influenza Virus Viruses by Type and Week Specimen taken, 2010

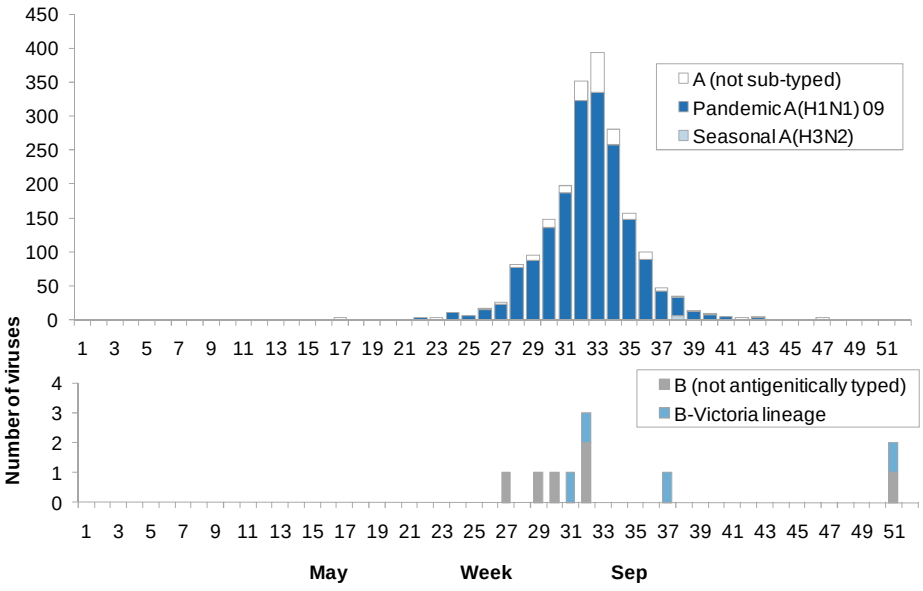
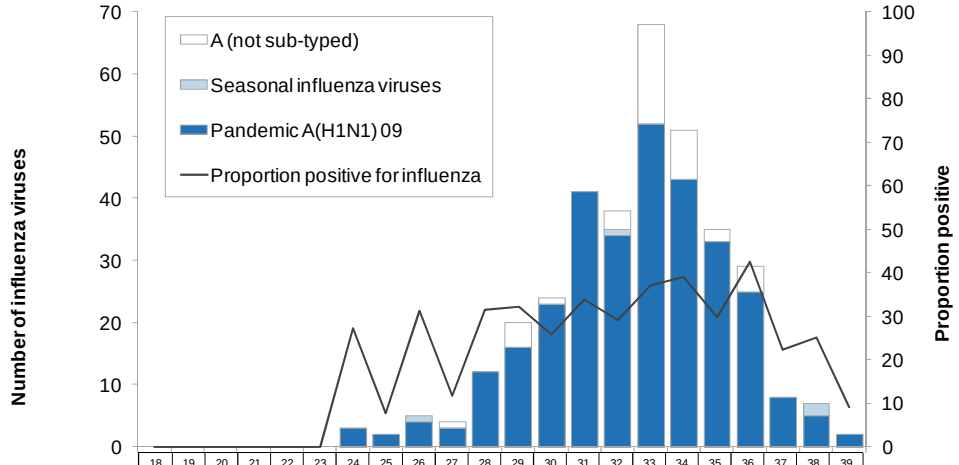


Figure 18 shows the temporal distribution of influenza viruses from sentinel surveillance from weeks 18–39. The pandemic A(H1N1) 09 virus strain predominated throughout the influenza season with a peak in week 33 (16–22 August), comprising 76.5% of all viruses.

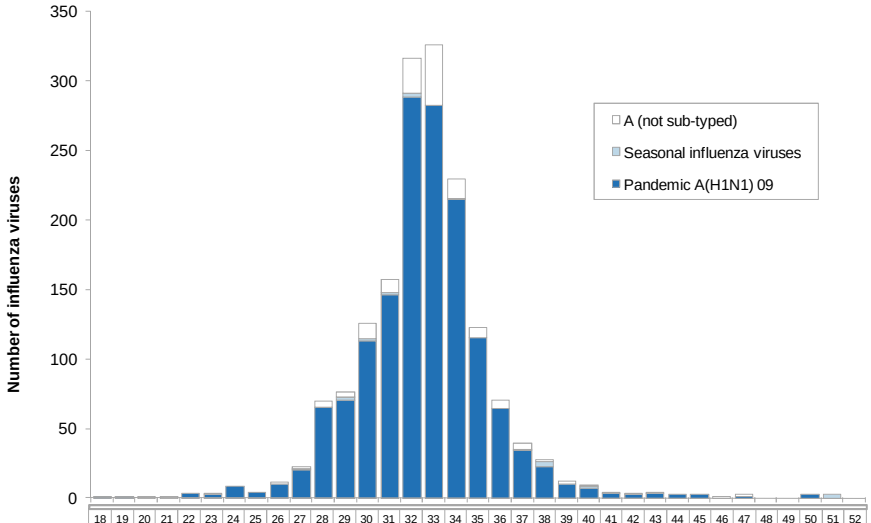
Figure 18. Total Influenza Viruses from Sentinel Surveillance by Type and Week Reported, 2010



	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37	38	39
A (not sub-typed)	0%	0%	0%	0%	0%	0%	0%	0%	0%	25%	0%	20%	4%	0%	8%	24%	16%	6%	14%	0%	0%	0%
Seasonal influenza viruses	0%	0%	0%	0%	0%	0%	0%	0%	20%	0%	0%	0%	0%	0%	3%	0%	0%	0%	0%	0%	29%	0%
Pandemic A(H1N1) 09	0%	0%	0%	0%	0%	0%	100%	100%	80%	75%	100%	80%	96%	100%	89%	76%	84%	94%	86%	100%	71%	100%

Figure 19 shows the temporal distribution of influenza viruses reported by type and subtype for each week from non-sentinel surveillance for weeks 18–52. Again, the pandemic A(H1N1) 09 virus has become the predominant strain in New Zealand with a peak in week 32 (9–15 August 2010) comprising 91.1% of all viruses.

Figure 19. Total Influenza Viruses from Non-sentinel Surveillance by Type and Week Reported, 2010



	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37	38	39	40	41	42	43	44	45	46	47	48	49	50	51	52
A (not sub-typed)	0%	0%	100	100	0%	33%	0%	0%	9%	5%	6%	5%	9%	6%	8%	13%	6%	6%	9%	10%	4%	17%	11%	0%	33%	25%	0%	0%	100	50%	0%	0%	0%	0%	0%
Seasonal influenza viruses	0%	0%	0%	0%	0%	0%	0%	0%	0%	5%	0%	3%	1%	1%	1%	0%	0%	0%	0%	3%	15%	0%	11%	25%	0%	0%	0%	0%	0%	0%	0%	0%	0%	100	0%
Pandemic A(H1N1) 09	100	100	0%	0%	100	67%	100	100	91%	91%	94%	92%	90%	93%	91%	87%	93%	94%	91%	87%	81%	83%	78%	75%	67%	75%	100	100	0%	50%	0%	0%	100	0%	0%

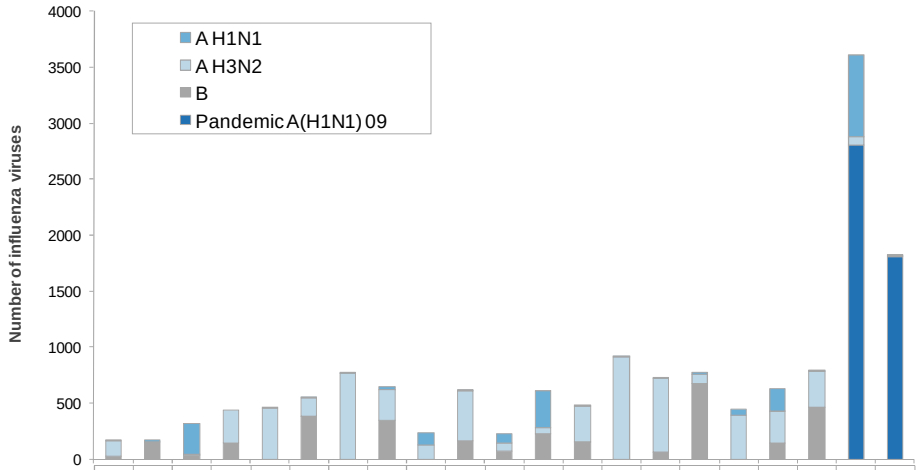
*Data shown from week 18 onwards.

6.2. Predominant Influenza Virus Strains from 1990–2010

Figure 20 shows the number and percentage of typed and subtyped (not total) influenza viruses from 1990–2010. There are noticeable changes in terms of the predominance patterns.

- The pandemic A(H1N1) 09 strain predominated in 2009 and 2010.
- The seasonal A(H1N1) strain predominated in three seasons (1992, 2000 and 2001) and was associated with relatively low hospitalisations (193 in 1992, 228 in 2000 and 379 in 2001).
- The seasonal A(H3N2) strain predominated for 11 seasons (1990, 1993, 1994, 1996, 1998, 1999, 2002, 2003, 2004, 2006 and 2007). The A/Fujian/411/02 (H3N2)-like strain predominated in 2003 with the highest recorded hospitalisations from 1990–2008. A A/Wuhan/359/95 (H3N2)-like strain predominated in 1996 with 94 deaths associated with it (93 out 94 deaths occurred in people aged 65 years and older).
- Influenza B strains predominated for five seasons (1991, 1995, 1997, 2005 and 2008). B/HongKong/330/2001-like strain (B-Victoria lineage) predominated in 2005. The disease burden was high in children aged 5–19 years with deaths in three children associated with this strain.

Figure 20. Influenza Viruses by Type, 1990–2010



	1990	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000	2001	2002	2003	2004	2005	2006	2007	2008	2009	2010
A H1N1	0.6%	7.7%	85.7%	0.0%	0.2%	1.1%	0.3%	3.6%	47.4%	0.3%	36.0%	54.4%	0.4%	0.1%	0.1%	2.3%	12.6%	32.1%	0.8%	20.1%	0.0%
A H3N2	83.2%	0.0%	2.8%	65.7%	98.7%	30.1%	99.1%	43.0%	51.7%	73.7%	32.9%	7.9%	68.0%	99.6%	91.3%	10.7%	86.3%	45.0%	41.0%	2.2%	0.7%
B	16.2%	92.3%	11.5%	34.3%	1.1%	68.8%	0.7%	53.5%	0.9%	26.0%	31.1%	37.8%	31.6%	0.3%	8.6%	87.0%	1.1%	23.0%	58.3%	0.0%	0.5%
Pandemic A(H1N1) 09	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	77.7%	98.8%

6.3. Pandemic A(H1N1) 09

Representative pandemic A(H1N1) 09 isolates (538) were antigenically subtyped. Some of these isolates were also sent to WHOCC-Melbourne. Results from WHOCC indicate that most of the currently circulating pandemic A(H1N1) 09 viruses are closely related to the vaccine candidate strain A/California/7/2009 (H1N1) [7].

Genetic analysis of the hemagglutinatin (HA) gene of representative pandemic A(H1N1) 09 viruses showed that the isolates from New Zealand, as well as isolates from Australia and Singapore, exhibited increasing genetic drift with two major subclades both with E374K and N125D amino acid changes from previously circulating viruses (see Figure 1 in the Appendix). Genetic analysis of the neuraminidase (N1) gene of representative pandemic A(H1N1) 09 viruses showed that viruses with HA E374K and N125D amino acid changes

had amino acid changes at M15I and N189S and fell into a separate group (see Figure 2 in the Appendix). However, it appears that these genetic changes have not resulted in significant antigenic changes [8]. No H275Y mutations were detected, suggesting they were sensitive to oseltamivir.

6.4. Seasonal A(H1N1)

No seasonal A(H1N1) virus was detected in 2010.

6.5. Influenza A(H3N2)

Only 12 influenza A(H3N2) viruses were identified in 2010. Four were antigenically subtyped and these isolates were antigenically closely related to the A/Perth/16/2009-like strain.

6.6. Influenza B

Only 10 influenza B viruses were identified in 2010. Four were further antigenically typed as the B/Brisbane/60/2008-like strain.

6.7. Oseltamivir Resistance Monitoring

The WHO NIC at ESR has established a phenotypic method (fluorometric NA inhibition assay) for the surveillance of anti-viral drug resistance in influenza viruses. In addition, a molecular method (PCR and sequencing) was also developed to monitor the H275Y mutation (histidine-to-tyrosine mutation at the codon of 275 in N1 numbering) which confers resistance to oseltamivir. Since January 2008, the global emergence and rapid spread of oseltamivir-resistant seasonal AH1N1 viruses has been observed. During New Zealand's 2009 winter season, a total of 25 seasonal AH1N1 viruses were tested using fluorometric NA inhibition. The results indicated that these viruses had a highly reduced sensitivity to oseltamivir with IC50 values in the range of 305–7912 nM, typical of the recent globally emerging oseltamivir-resistant A(H1N1) viruses (Table 3).

In 2010, the fluorometric NA inhibition assay tested a total of 334 pandemic A(H1N1) viruses. All viruses were sensitive to oseltamivir with IC50 values in the range of 0.01–2.9 nM (Table 3).

Table 3. Oseltamivir Resistance, 2006–2010

Influenza Type/Sub-type	Seasonal A(H1N1)				Pandemic A(H1N1) 09	
Year	2006	2007	2008	2009	2009	2010
Number of viruses	17	138	4	25	483	334
Mean IC50*	1.84	0.83	728	1399	0.4	0.68
Std. dev.	0.71	0.63	136	1990	0.24	0.41
Min IC50	0.25	0.01	547	305	0.09	0.01
Max IC50	3.099	4.219	870	7912	1.4	2.05

**IC50: Concentration of oseltamivir (nM) at which there is 50% inhibition of neuraminidase activity.*

7. Southern Hemisphere Vaccine Strain Recommendations

In October 2010, the Australian Influenza Vaccine Committee (AIVC), with a New Zealand representative, met to decide on the composition of the influenza vaccine for the 2011 winter season for New Zealand, Australia and South Africa. During these discussions, the following trends were noted, these are outlined next.

7.1. Influenza A(H1N1)

The epidemiological data from the New Zealand 2010 influenza season and most other southern hemisphere countries indicate that the pandemic A(H1N1) 09 virus was the predominant circulating strain. The WHOCC-Melbourne has analysed 1216 pandemic A(H1N1) 09 isolates from 16 countries, including New Zealand, since January 2010. The antigenic data from these isolates indicate that the current circulating pandemic A(H1N1) 09 viruses are antigenically similar to the vaccine candidate strain A/California/7/2009 (H1N1). Current vaccines containing A/California/7/2009 antigen stimulated anti-HA antibodies of similar geometric mean haemagglutination inhibition (HI) titres to the vaccine virus and recent pandemic A(H1N1) 09 isolates.

Based on southern hemisphere and global data, the WHO Consultative Group and the AIVC recommended vaccines containing a pandemic influenza A/California/7/2009 (H1N1)-like strain as the H1 component for 2011.

7.2. Influenza A(H3N2)

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 WHOCC-Melbourne has analysed 119 A(H3N2) isolates from nine countries since January 2010. Most recent isolates had antigenically drifted away from the A/Brisbane/10/2007 (H3N2)-like strain and were antigenically closely related to the A/Perth/16/2009-like strain. Current vaccines containing the A/Perth/16/2009 antigen stimulated anti-HA antibodies of similar geometric mean HI titres to the vaccine virus and to recent A(H3N2) isolates. As a result, an A/Perth/16/2009-like strain was recommended by the WHO Consultative Group and the AIVC to be the H3 component of the influenza vaccine for the southern hemisphere for 2011.

7.3. Influenza B

Two distinct lines of influenza B have co-circulated in many countries during recent years. This dates from the late 1980s when the B/Panama/45/90 variant of influenza B was first observed. This strain and its further variants of the B/Yamagata/16/88 lineage (the most recent representative strain is B/Florida/4/2006) spread worldwide, whereas strains of the previous B/Victoria/2/87 lineage viruses continued to circulate in Asia and subsequently underwent independent evolution as an antigenically distinct lineage (the most recent representative strain is B/Brisbane/60/2008). For reasons not wholly understood, these remained geographically restricted to Asia until 2001. In 2002, the B/Victoria-lineage strains spread to the rest of the world.

Both recent B/Victoria-like strains (B/Brisbane/60/2008 is the current reference strain) and B/Yamagata-like strains (B/Florida/4/2006 is the current reference strain) continued to be isolated worldwide in 2010. Varying proportions of the two lineages were seen in many countries with mainly B/Victoria-like lineage strains circulating in southern hemisphere countries. The majority of isolates were antigenically closely related to B/Brisbane/60/2008-like strain. Current vaccines containing B/Brisbane/60/2008 antigen stimulated HA antibodies that were similar in titre to the vaccine virus and to recently isolated B/Brisbane/60/2008-like viruses. Based on the southern hemisphere and global data, the WHO Consultative Group and the AIVC recommended vaccines containing a B/Brisbane/60/2008-like strain to be the B component of the influenza vaccine for the southern hemisphere for 2011.

In summary, the AIVC agreed to adopt the recommendations made by the WHO consultation group as shown below.

The recommended influenza vaccine formulation for New Zealand in 2011 is:

- **A(H1N1) an A/California/7/2009 (H1N1) - like strain***
- **A(H3N2) an A/Perth/16/2009 (H3N2) - like strain**
- **B a B/Brisbane/60/2008 - like strain**

***Note: A/California/7/2009 is a pandemic A(H1N1) 09 strain**

8. Discussion

Sentinel surveillance, as a syndromic surveillance system in New Zealand, is one of the best systems for monitoring the burden of disease in the community during an epidemic. It has operated continuously in New Zealand since its establishment in 1991 [3]. It is a relatively stable system that monitors year-to-year disease trends in the community. Active syndromic surveillance systems are increasingly being used to detect emerging and re-emerging pathogens [9,10]. Enhanced influenza surveillance is also a key strategy for improving New Zealand's preparedness for pandemic influenza[11]. The usefulness of sentinel surveillance during a pandemic was tested in 2009 and the system has been adapted to monitor the early and late stages of a pandemic. While the sensitivity of sentinel surveillance during the containment phase of a pandemic is encouraging, it requires further evaluation. Furthermore, the performance characteristics of sentinel surveillance (sensitivity, specificity, representativeness and robustness) for a pandemic at a national and regional level during the containment phase for early detection, as well as the management phase for monitoring, should be evaluated against other influenza surveillance systems.

Based on sentinel consultation data, the overall influenza activity in 2010 is described as being at a medium level. Comparing data for the past 14 years (from 1997–2010), the weekly consultation rates peak for ILI in 2010 was the sixth highest and the cumulative incidence rates peak in 2010 was the ninth highest.

It is estimated that ILI resulting in a visit to a GP affected over 50,561 New Zealanders in 2010 or about 1.2% of the population. The number of cases reported through the sentinel network is likely to be a considerable underestimate of the true number, as many people do not consult a GP when they have an ILI.

The second year of pandemic A(H1N1) 09 showed marked geographic heterogeneity. In particular, some regions (mainly small urban and rural areas) that had relatively low ILI activity in 2009, experienced higher levels of activity during the second wave in 2010. This finding supports the hypothesis that areas that were more affected in 2009 were protected to a certain extent in 2010. If this was not the case, we would expect (as we see for most diseases) that rates from one year to the next would be highly positively correlated because patterns of vulnerability tend to persist. Regional variations of pandemic A(H1N1) 09 infections were also observed in 2009 in clinical surveillance as well as in the pandemic A(H1N1) 09 seroprevalence survey [12,13,14]. It is possible that this variability allowed areas (mainly rural and small urban areas) with low pandemic A(H1N1) 09 activity to maintain more susceptible populations and to sustain more pandemic A(H1N1) 09 infections and transmission in 2010 than in 2009.

The age distribution of pandemic A(H1N1) 09 infections in 2010 was broadly similar to 2009 with the highest infection rates in children under the age of 5 years. Hospitalisation rates declined significantly for most age groups, except for the 20–39 year age group. This decline was particularly marked for the 5–9 year age group, although notification rates remained higher in children aged 5–19 years. This probably reflected a feature of the 2009 pandemic which caused relatively mild disease in people in the 5–19 year age group.

The distribution of pandemic A(H1N1) 09 infections among ethnic groups in 2010 changed markedly compared with 2009. Rates for Pacific and Māori populations remained significantly higher than for European and Other ethnic groups, but the disparity was far less pronounced. These changes in the age and ethnicity distribution of the disease may reflect immunity from a combination of sources, including immunisation and natural infection. Reasons for ethnic differences in hospitalisation rates may include a higher incidence of infection among Pacific and Māori people, a higher prevalence of co-morbidities (such as asthma and diabetes), unfavourable environmental factors (such as household crowding and poor quality housing), behavioural differences in responding to influenza, differences in socio-cultural-economic status, differences in health service utilisation and increased genetic susceptibility [15]. Further studies on the contributing factors to ethnic differences in the risk of pandemic A(H1N1) 09 infection and severe disease are underway in New Zealand.

One of the strengths of the sentinel surveillance system in New Zealand is the combination of disease surveillance (influenza-like illness) with strain surveillance (virological identification). A definitive diagnosis of influenza requires laboratory confirmation, because clinical diagnosis on the basis of clinical symptoms is not highly specific. In fact, sentinel surveillance is the only syndromic surveillance system that obtains appropriate respiratory swabs for verification of clinical diagnosis. Consequently, an important part of the sentinel system is for GPs to take nasopharyngeal and/or throat swabs from patients presenting with an influenza ILI. In the current protocol, three swabs each week are required to be collected from each GP. An evaluation should be conducted to find out whether this number provides sufficient information about the predominant circulating strains over time at a national and regional level during influenza epidemics.

During sentinel surveillance from January to September 2010, four virology laboratories tested 966 respiratory specimens for influenza viruses and 349 (36.1%) specimens were positive. However, the influenza isolation rate varied among the different DHBs. Some DHBs had an influenza virus isolation rate that was lower than the national average of 36.1%. Many factors contribute to low isolation rates, including sampling techniques. Sampling of the respiratory tract for clinical viral isolation should maximise the harvest of virally infected columnar epithelial cells. Ideally, nasopharyngeal washes or aspirates are the best specimens as they contain a higher cellular content than nasopharyngeal swabs[16]. By comparison, throat swabs or throat washings are of limited use in the diagnosis of influenza because the majority of cells captured by this technique are squamous epithelia. However, a combined nose (ie, nasopharyngeal) and throat swab can be a useful specimen for influenza virus isolation and it is selected for influenza surveillance because of its convenience. Nasopharyngeal swabs should be cotton-, rayon- or dacron-tipped, plastic-coated swabs. The swab should be inserted deeply into the nasopharynx, rotated vigorously to collect columnar epithelia cells, removed, placed into viral transport medium, chilled and couriered to the virology laboratory without delay.

A global emergence and rapid spread of oseltamivir-resistant influenza A(H1N1) viruses carrying an NA gene with an H274Y (histidine to tyrosine mutation at the codon of 274 by N2 numbering) amino acid substitution has been observed in New Zealand since January 2008. All seasonal influenza A(H1N1) viruses (25) tested in 2009 showed that they were resistant to oseltamivir. On the other hand, all pandemic A(H1N1) 09 viruses tested in 2009 and 2010 showed that they were sensitive to oseltamivir. Oseltamivir-resistant viruses pose challenges for the selection of antiviral medications for the treatment and chemoprophylaxis of influenza. They also pose potential risks in terms of the generation of new variants of the

pandemic A(H1N1) 09 virus that carry the oseltamivir-resistant gene via co-infection and reassortment. It has become increasingly important to establish and sustain a national antiviral monitoring programme in New Zealand that would provide timely surveillance information to assist clinicians in choosing appropriate antiviral agents for their patients, and assist public health officials making evidence-based decisions on stockpiling of antiviral agents and their usage during a pandemic or epidemic. The timely surveillance information also provides compelling reasons for clinicians to test patients for influenza virus infection to select appropriate antiviral medications.

Virological surveillance for outpatients and hospital inpatients (also referred to as non-sentinel surveillance) complements sentinel surveillance. Non-sentinel surveillance provides useful information on the characterisation of circulating influenza viruses and monitors the emergence of novel strains with pandemic potential. However, the current non-sentinel surveillance does not provide robust epidemiologic data with good denominator information on the severe end of the disease burden in terms of its morbidity and mortality, and the risk factors caused by influenza. The recent emergence of the pandemic A(H1N1) 09 virus highlights the need for surveillance to better define persons most at risk for severe acute respiratory illness (SARI) resulting from influenza [17]. Expansion of the existing non-sentinel surveillance to include the systematic collection of epidemiologic data on hospitalised SARI cases would enable the factors that place the most vulnerable persons at risk to be described and targeted intervention to be facilitated. It would also establish a platform for broader respiratory disease surveillance. It would be beneficial to evaluate the current status of non-sentinel surveillance in New Zealand and consider an expansion of the system to establish SARI surveillance for hospital inpatients.

Since 2001, the four virology laboratories have been using the ESR-designed electronic influenza virus input form for data entry. This process requires the retrieval of the necessary demographic data from the hospital information system and re-keying this information onto the ESR virus input form. This is a time-consuming system and inevitably creates data error. Timely reporting for the virology weekly report was one of the biggest challenges during the pandemic response. Advances in information transfer using systems such as Health-Link would greatly streamline this process.

As the impact of influenza can be reduced by annual immunisation, the information on influenza vaccination coverage is particularly important in raising awareness of the disease amongst health professionals and the public, and for planning the vaccine's formulation and delivery. The National Influenza Immunisation Strategy Group was established in 2000 with the purpose of improving coverage through public and healthcare provider education. A national approach to promotion, coupled with local initiatives, is key to lifting vaccination coverage to 65% amongst those at greatest risk, including people aged 65 years and older.

Influenza vaccines are recommended for people at risk of developing complications following infection because of their age or because of underlying chronic conditions, and are available free each year. [18]. In 1997, New Zealand introduced free influenza vaccination to all New Zealanders aged 65 years and older, and set a target of 75% coverage for the year 2000. In 1999, free vaccination was extended to include those under 65 years with certain chronic medical condition[18,19]. Quality coverage data are essential for the continuing development of this programme, while continuing surveillance ensures the provision of effective vaccines to reduce the burden of influenza in New Zealand.

9. References

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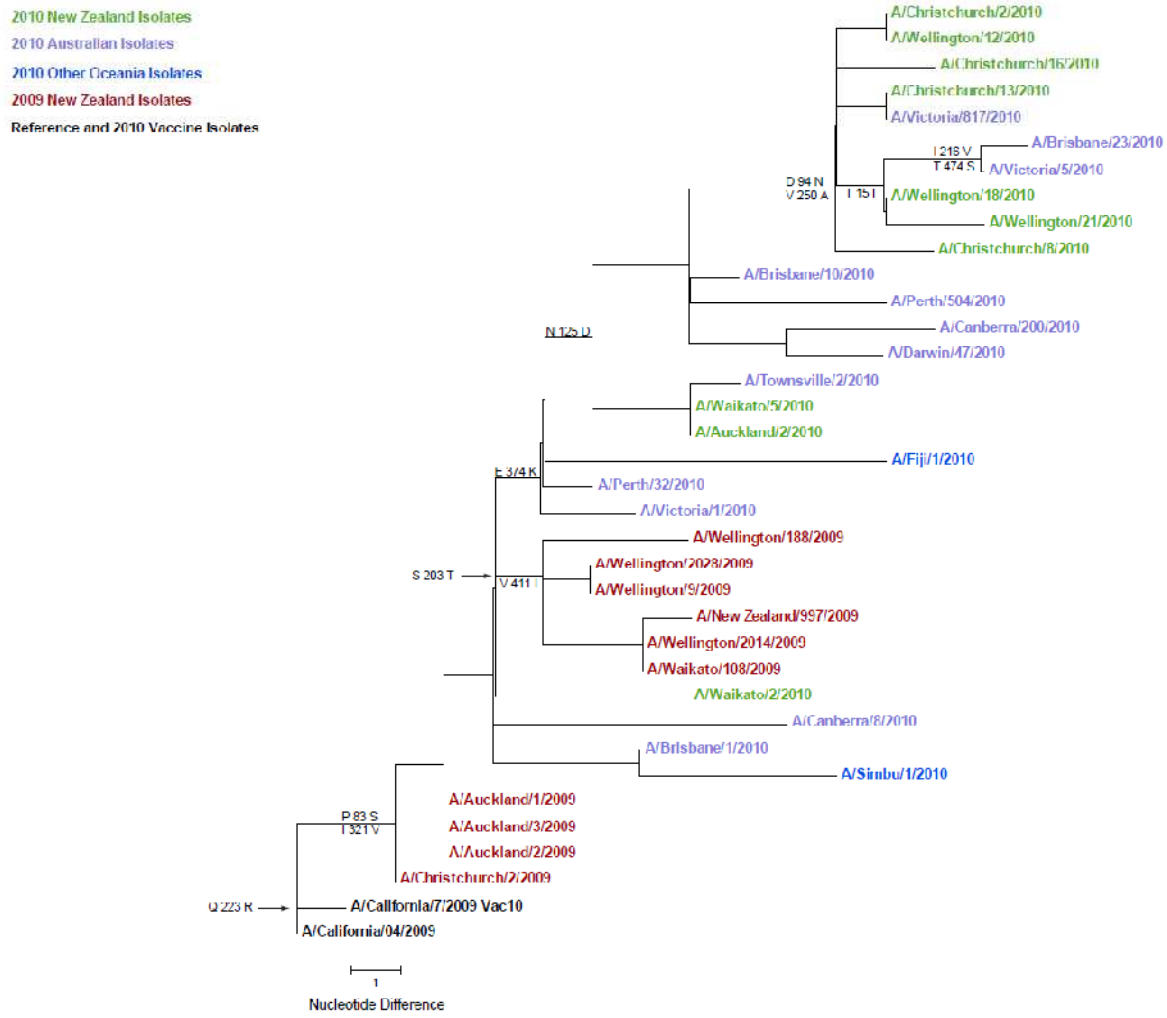
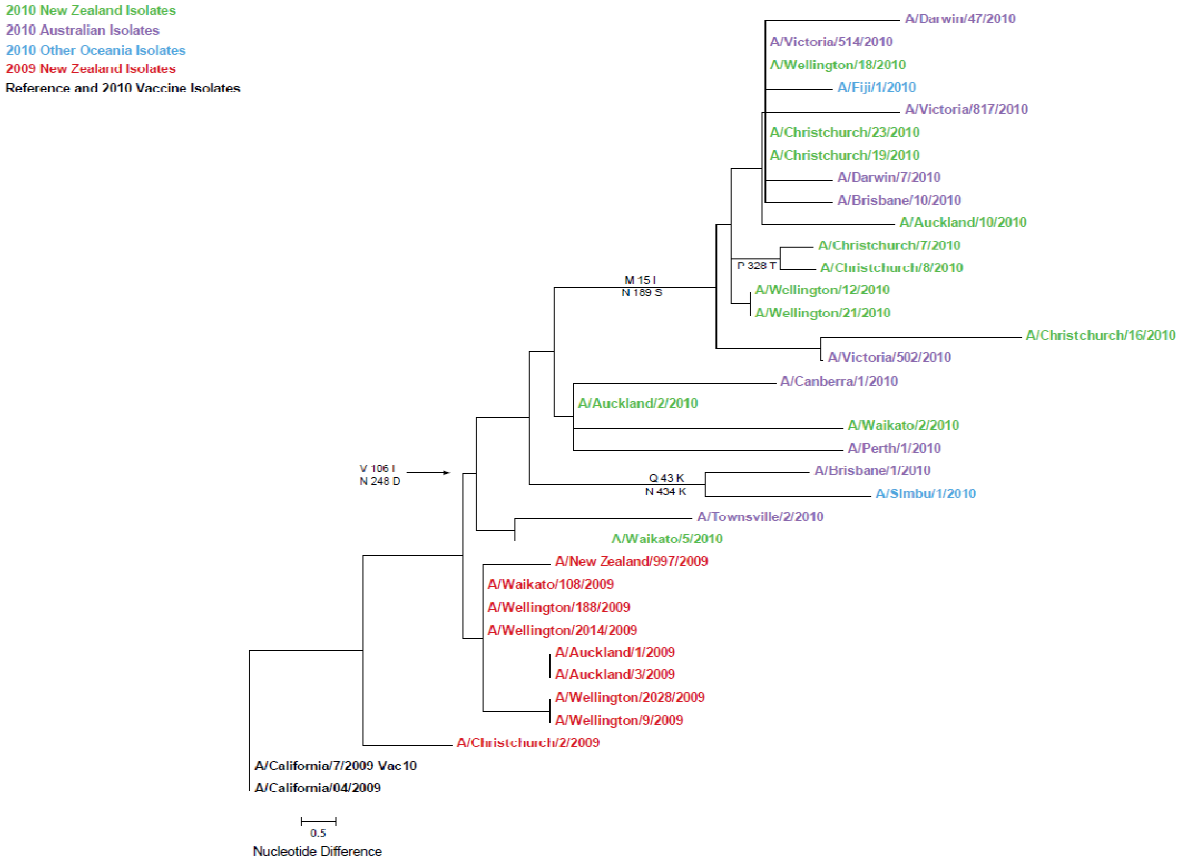
Appendix- Figure 1. Phylogenetic analysis of HA gene sequence of pandemic A(H1N1) viruses



Appendix- Figure 2. Phylogenetic analysis of NA gene sequence of pandemic A(H1N1) viruses



Note: The evolutionary history was inferred using the Neighbor-Joining method [1]. The bootstrap consensus tree inferred from 1000 replicates [2] is taken to represent the evolutionary history of the taxa analysed [2]. Branches corresponding to partitions reproduced in less than 50% bootstrap replicates are collapsed. The percentage of replicate trees in which the associated taxa clustered together in the bootstrap test (1000 replicates) is shown next to the branches [2]. The tree is drawn to scale, with branch lengths in the same units as those of the evolutionary distances used to infer the phylogenetic tree. Codon positions included were 1st+2nd+3rd+Noncoding. All positions containing gaps and missing data were eliminated from the dataset (Complete deletion option). There were a total of 1681 positions in the final dataset. Phylogenetic analyses were conducted in MEGA4 [3].

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