Influenza surveillance in New Zealand
2013

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INFLUENZA SURVEILLANCE
IN NEW ZEALAND
2013

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SUMMARY

Influenza viruses can cause substantial morbidity and mortality in a short time and frequently undergo antigenic changes. National influenza surveillance is an essential public health tool for assessing and implementing strategies to control influenza. Influenza surveillance in New Zealand monitors the incidence and distribution of influenza in the community, assists with the early detection of influenza epidemics and identifies the predominant circulating strains. This report summarises the burden of disease in the community due to influenza, the circulating influenza virus strains, hospitalisations and immunisation coverage for 2013.

During the 2013 winter season, 2122 consultations for influenza-like illness (ILI) were reported from a national sentinel network of 70 general practices. It is estimated that an ILI resulting in a visit to a general practitioner (GP) affected over 25 598 New Zealanders (0.6% of total population) during the season, compared with an estimated 48 186 people in 2012 (1.1% of total population).

Influenza activity peaked in September 2013. Overall, ILI activity was at a low level compared with the 1997–2012 period. ILI consultation rates varied greatly among District Health Boards (DHBs), with the highest rates reported from South Canterbury and Waitemata DHBs.

Both the SHIVERS hospital-based Severe Acute Respiratory Infections (SARI) surveillance and sentinel practice-based ILI surveillance were fully operational in 2013. Influenza-associated primary care consultations and hospitalisations showed contrasting sociodemographic patterns and the rates of influenza-associated GP consultation and hospitalisation varied markedly with age. Influenza-associated hospitalisation rates were highest in the very young (0–4 years) and the elderly (≥65 years). Influenza-associated GP consultation rates, however, showed the opposite pattern, with higher rates in pre-schoolers, school-aged children and adults, but a lower rate in infants (<1 year) and the elderly (≥65 years). In addition, a preliminary analysis of influenza rates by ethnicity found that Māori and Pacific peoples experienced the highest rates of influenza-associated hospitalisations but the lowest rates of influenza-associated GP consultations, while the Asian ethnic group showed the opposite trend. When socioeconomic status (SES) groups were considered, the most deprived populations (NZDep 9–10) were found to have the highest rates of influenza-associated hospitalisations but the lowest rates of influenza-associated GP consultations.

In 2013, a total of 2326 influenza viruses were detected. Of these, 60.4% were influenza A and 39.6% were influenza B. Of all the viruses typed and sub-typed (2066) during the season, the predominant strains were influenza B at 44.6% and A(H3N2) at 40.9%, while 14.7% were A(H1N1)pdm09 strains. Antiviral susceptibility monitoring indicated that all influenza viruses tested were sensitive to oseltamivir and zanamivir.

No significant antigenic drift was detected for influenza A(H1N1)pdm09 viruses. A(H3N2) viruses were closely related to the A/Victoria/361/2011-like strain and the A/Texas/50/2012-like strain (a strain antigenically similar to A/Victoria/361/2011-like strain). Two lineages of influenza B viruses (B/Victoria and B/Yamagata lineages) were co-circulating in 2013 with an increased proportion of B/Yamagata lineage viruses. The B/Yamagata lineage viruses have drifted from the B/Wisconsin/1/2010-like strain to B/Massachusetts/2/2012-like strain. As a result, A(H3N2) and B components have been updated for the influenza vaccine for 2014.

The recommended influenza vaccine formulation for New Zealand in 2014 is:

A(H1N1) an A/California/7/2009 (H1N1)pdm-like strain*

A(H3N2) an A/Texas/50/2012 (H3N2)-like strain

B a B/Massachusetts/2/2012-like strain

* Note: A/California/7/2009 (H1N1)-like strain is an influenza A(H1N1)pdm09 strain.
INTRODUCTION

Influenza viruses frequently undergo antigenic changes, enabling them to evade the host immune response. This poses a real challenge for 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.

There are three active influenza surveillance systems in New Zealand which combine epidemiological and virological investigations for influenza:

1. National sentinel general practitioner (GP) based surveillance: this system was established in 1991 as part of the World Health Organization’s (WHO) Global Influenza Programme.

   The purpose of this surveillance system is to:
   - improve knowledge of the incidence and distribution of influenza in the community to assist in developing strategies to control influenza through immunisation;
   - enable early detection of influenza epidemics within the community to guide the development and implementation of public health measures; and
   - provide an indication of the predominant strains of influenza virus in the community to help in planning for the most effective influenza vaccine for the subsequent year [1].

2. SHIVERS hospital-based SARI surveillance.

   In October 2011, a five year multi-centre and multi-disciplinary project “Southern Hemisphere Influenza and Vaccine Effectiveness Research and Surveillance” (SHIVERS) led by the Institute of Environmental Science and Research (ESR) was established.

   Hospital-based surveillance for severe acute respiratory infections (SARI) is a key component of SHIVERS and it has been established and fully functioning since 30 April 2012. This is a result of an excellent collaboration between ESR, Auckland District Health Board (ADHB), Counties Manukau District Health Board (CMDHB), the University of Otago, the University of Auckland, WHO Collaborating Centre at St Jude Children’s Hospital (WHOCC) and US Centers for Disease Control and Prevention (CDC). This is an active, prospective, continuous, population-based surveillance for SARI cases admitted to four hospitals in the central, east and south Auckland region (population 838 000).

   The aims of SARI surveillance are to:
   - establish enhanced, prospective, longitudinal, population-based surveillance for hospitalised SARI cases, Intensive Care Unit (ICU) admissions and deaths caused by influenza and other respiratory pathogens in Auckland, in order to support global influenza surveillance [2];
   - measure the incidence, prevalence, demographic characteristics (including age, sex, ethnicity and socioeconomic status), clinical spectrum and outcomes for SARI cases, ICU admissions and deaths;
   - identify etiologies of SARI cases, including ICU admissions and deaths attributable to influenza and other respiratory viruses (respiratory syncytial virus, human metapneumovirus, adenovirus, parainfluenza types 1-3, rhinovirus);
   - determine the accuracy and validity of the data generated from New Zealand’s existing hospital discharge coding by comparing it with estimates of influenza and pneumonia etiology and incidence obtained from this study;
• describe any possible increased risk of influenza-related hospitalisation, ICU admission and
death associated with conditions such as asthma, pregnancy, diabetes and high BMI (body mass
index) among population sub-groups defined by age, sex, ethnicity and socioeconomic status;
• contribute directly to some of the other specific aims and objectives of the SHIVERS project by
using the data generated from this surveillance.

3. SHIVERS sentinel practice-based ILI surveillance.

SHIVERS sentinel practice based ILI surveillance has been established and fully functioning
since 29 April 2013. This is a result of a collaboration between ESR, the University of
Auckland, Primary Health Organisations (Procare, Auckland and East Tamaki Healthcare),
sentinel general practices, Auckland Regional Public Health Service, the University of Otago,
WHOCC St Jude and CDC-Atlanta.

The aims of ILI surveillance are to:
• measure the burden of disease caused by influenza and other respiratory viruses in community;
• monitor trends in disease caused by influenza and other respiratory viruses in community;
• identify high risk groups that should be prioritised for prevention and treatment;
• monitor antigenic, genetic and antiviral characteristics of influenza viruses associated with
influenza-like illness;
• provide a study base to estimate the effectiveness of the influenza vaccine.

This report summarises the results obtained from national sentinel GP-based influenza surveillance
and SHIVERS sentinel practice-based influenza surveillance in New Zealand for 2013, and includes
some comparisons with previous years. It also includes information on hospital-based influenza
morbidity and mortality (obtained from SHIVERS SARI surveillance and the Ministry of Health’s
National Minimum Dataset and Mortality Collection), Healthline and HealthStat data, laboratory-
based antiviral susceptibility data and influenza immunisation coverage data (obtained from the
Ministry of Health’s Sector Services).
METHODS

National sentinel general practitioner-based influenza surveillance

The national sentinel GP-based surveillance system, also referred to ESR’s sentinel GP-based influenza surveillance, began in 1991 as part of the World Health Organization’s (WHO) Global Influenza Programme. It is operated nationally by the ESR and locally by influenza surveillance coordinators in the public health services (PHSs). Sentinel surveillance usually operates in the winter, from May to September (weeks 18–39). Local surveillance coordinators recruited general practitioners within their region to participate on a voluntary basis. Where possible, the number of practitioners recruited was proportional to the size of the population in each DHB covered by the PHS (approximately one GP per 50,000 population).

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

For sentinel surveillance ILI was defined as an “acute upper respiratory tract infection characterised by an abrupt onset and two of the following: fever, chills, headache, and myalgia” [3].

Each participating GP collected three respiratory samples (i.e., a nasopharyngeal or throat swab) weekly; from the first ILI patient examined on each Monday, Tuesday and Wednesday. Further refinement of the sampling scheme has been implemented since 2010. For a general practice with a registered patient population of more than 10,000, a total of six nasopharyngeal or throat swabs were collected from the first two ILI patients examined on Monday, Tuesday and Wednesday of each week. The GPs forwarded these samples either 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 antigens. Influenza viruses were typed and subtyped as A, B, A(H3N2) or influenza A(H1N1)pdm09.

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 was received by Wednesday of the following week. Likewise, virology laboratories reported the total number of swabs received from each DHB and the influenza viruses identified to ESR weekly, and updated details on influenza types and subtypes. ESR reports national information on epidemiological and virological surveillance of influenza weekly, monthly and annually to relevant national and international organisations, including the WHO, with reports published on the ESR website: www.surv.esr.cri.nz/virology/influenza_weekly_update.php

Consultation rates were calculated using the registered patient populations of the participating practices as a denominator. From 1992 to 2009, the denominator for the age-specific ILI rate calculation was based on New Zealand census data with the assumption that the age distribution of the GP patient population was the same as the New Zealand population, because age-specific patient population data was not provided by the participating practices. From 2010 to 2013, age-specific patient population denominators were available for the consultation rate calculations, with the exception of a single general practice in 2013, where the former method was applied.
Influenza surveillance in New Zealand 2013

Methods

The national level of ILI activity is described using a set of threshold values [4, 5]. Based on New Zealand’s influenza ILI consultation rates during 1990–1999, various levels of influenza activity such as baseline, normal seasonal influenza, higher than expected influenza activity and severe epidemic level are described by using different ILI consultation rates. For details, see the table below.

<table>
<thead>
<tr>
<th>Term used</th>
<th>Consultation rate (per 100 000 population)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline</td>
<td>≤49</td>
</tr>
<tr>
<td>Normal seasonal activity</td>
<td></td>
</tr>
<tr>
<td>low</td>
<td>50–99</td>
</tr>
<tr>
<td>moderate</td>
<td>100–149</td>
</tr>
<tr>
<td>high</td>
<td>150–249</td>
</tr>
<tr>
<td>Higher than expected</td>
<td>250–399</td>
</tr>
<tr>
<td>Severe epidemic</td>
<td>≥400</td>
</tr>
</tbody>
</table>

**SHIVERS sentinel practice based surveillance for influenza-like illness**

In SHIVERS sentinel practices, GPs and/or practice nurses screened every patient who was seeking medical attention for an ILI. The case definition was “an acute respiratory illness with a history of fever or measured fever of ≥38°C, AND cough, AND onset within the past 10 days, AND requiring a GP consultation”. If a consultation-seeking patient met this definition, a respiratory specimen (nasopharyngeal or throat swab) was collected, to test for influenza and other respiratory pathogens. Information on the patient’s demography, clinical history, co-morbidities, vaccination history, regular medication and pregnancy status was also collected. Obesity was determined by visual assessment.

Totals of patients meeting the ILI definition, numbers tested, number positive for influenza viruses, number of the enrolled patients, and total consultations, were collected. This allowed calculation of population-based incidence for ILI and associated influenza, overall and stratified by age, sex, ethnicity and socio-economic status, among the ADHB and CMDHB resident population (from 2006 census data). For example, the overall ILI incidence was calculated using the ILI patients who were enrolled in sentinel practices, residing in ADHB and CMDHB, divided by the total enrolled patient population. Incidence rates were calculated, along with 95% confidence intervals (95%CI). In addition, this allowed the calculation of the proportion of ILI and associated influenza among total consultations, by overall and stratified patients, regardless of residence or enrolment status. For example, the overall proportion of ILI consultations was calculated using total ILI patients, divided by total consultations, regardless of their enrolment and residence status.

**HealthStat**

HealthStat is a computer-based routine surveillance system based on a nationally representative random sample of approximately 100 general practices that code for ILI. The case definition used for ILI by HealthStat is: “acute upper respiratory tract infection, with abrupt onset of 2+ symptoms from chills, fever, headache and myalgia” (i.e. the same case definition as for national sentinel GP-based surveillance). This surveillance system monitors the number of people who consult GPs with an ILI. HealthStat is based on automated downloads from GP practice management computer systems. This data is provided to ESR by CBG Health Research Ltd on a weekly basis. HealthStat GP-based surveillance does not include virological surveillance.

Analysis is frequency-based, with alarms raised by identifying statistical deviations (aberrations) from previous ILI counts. The analysis of the ILI count is based on the cumulative summation algorithm implemented in the Early Aberration Reporting System (EARS) application developed by
the Centers for Disease Control and Prevention (CDC), Atlanta, United States. EARS has three sensitivity thresholds; high, medium and low. If the daily consultation count exceeds a threshold, a flag is signalled.

Healthline

Healthline is the free national 24-hour 0800 telephone health advice service funded by the Ministry of Health. Calls made to Healthline are triaged using electronic clinical decision support software. The data collected is a daily count of all phone calls from people with symptoms for any illness made to Healthline and those triaged for ILI. The Healthline data is reported by ESR on a weekly basis, but this can be switched to a daily report if required. Around 70% of all calls to Healthline are symptom-related, and other calls (that are not part of this analysis) are queries for information.

Analysis is frequency-based with alerts raised by identifying statistical deviations (aberrations) from previous patterns of call numbers. Data is reported for all ages in five age bands 0–4, 5–14, 15–44, 45–64 and 65+ years. The analysis of the call frequency is based on the cumulative summation (CUSUM) algorithm implemented in EARS.

Cases of ILI are defined in the Healthline database as having one of the following 18 symptoms: fever (adult), breathing problems, breathing difficulty – severe (paediatric), colds (paediatric), cough (paediatric), cough (adult), fever (paediatric), flu-like symptoms or known/suspected influenza, flu-like symptoms (pregnant), influenza (paediatric), headache, headache (paediatric), muscle ache/pain, sore throat (paediatric), sore throat/hoarseness, sore throat/hoarseness (pregnant), upper respiratory tract infections/colds and upper respiratory tract infections/colds (pregnant).

SHIVERS hospital-based surveillance for severe acute respiratory infections

Inpatients with suspected respiratory infections admitted overnight to any of the four District Health Board hospitals (Auckland City Hospital and the associated Starship Children’s Hospital, Middlemore Hospital and the associated Kidz First Children’s Hospital) in the two DHBs, were screened by research nurses each day. Overnight admission is defined as: “A patient who is admitted under a medical team, and to a hospital ward or assessment unit”. Case ascertainment followed a surveillance algorithm. The presence of the components of the case definition was determined through a combination of reviewing the clinician’s admission diagnoses and by interviewing patients about their presenting symptoms. Records of all patients admitted overnight to medical wards were reviewed daily to identify anyone with a suspected respiratory infection. These patients were categorised into one of ten admission diagnostic syndrome groups. Research nurses then interviewed the patients and documented the components of the case definition that were present and differentiated patients into SARI and non-SARI cases.

A WHO SARI case definition [6] has been used for all patients with suspected respiratory infections, stated as an acute respiratory illness with:

- a history of fever or measured fever of ≥38°C, and
- cough, and
- onset within the past 10 days, and
- requiring inpatient hospitalisation.

If a patient with suspected respiratory infection met the SARI case definition, a respiratory sample was collected to test for influenza and other respiratory pathogens. In addition, patient information was captured via a case report form which included patient demographics, presenting symptoms and illness, pre-hospital healthcare, medication usage, influenza vaccination history, co-morbidities, disease course and outcome, including major treatments, ICU admission and mortality, epidemiologic risk factors and laboratory results.
Methods

The total numbers of all new hospital inpatient acute admissions and newly assessed and tested patients, including ICU admissions and deaths were collected. This allowed calculation of population-based incidence for SARI and associated influenza cases overall and stratified by age, sex, ethnicity and socio-economic status among the ADHB and CMDHB resident population (from 2006 census data). Incidence rates were calculated along with 95% confidence intervals (95%CI). In addition, this allowed the calculation of the proportion of SARI and associated influenza cases, including ICU admissions and deaths, by overall and stratified patients, among all acute admissions regardless of residence status. An acute admission is defined as an unplanned admission on the day of presentation at the admitting health care facility. Admission may have been from the emergency or outpatient departments of the health care facility, a transfer from another facility or a referral from primary care.

A case may have more than one specimen taken for influenza and non-influenza virus testing. The number of specimens can therefore differ from the number of cases and specimens and cases may be reported separately.

**NMDS-coded influenza hospitalisations**

Hospitalisation data for influenza (ICD-10AM-VI code I (J09-J11) for 2013 that correlate with previous versions of ICD-10AM (codes J10-J11), was extracted from the New Zealand Ministry of Health’s National Minimum Dataset (by discharge date). In this dataset, patients who spent less than one day in a hospital emergency department were excluded from the time series analysis of influenza hospitalisations from 2000 to 2013. Influenza-related hospitalisations were conservatively taken to include only those cases where influenza was the principal diagnosis. Repeat admissions were included because infection with a different influenza A subtype or influenza B virus is possible.

**Laboratory-based non-sentinel surveillance–for outpatients and hospital inpatients**

In addition to influenza viruses identified from sentinel GP-based surveillance, year-round laboratory-based passive 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 NIC at ESR (previously the National Health Institute, New Zealand Communicable Disease Centre). This type of surveillance is referred to as non-sentinel surveillance. Each week, all viral identifications, including influenza (largely from outpatient clinics and hospital inpatient clinics during routine laboratory diagnostic investigation), are reported to the NIC, which then collates and reports virology surveillance data nationally.

The NIC was designated by the Ministry of Health and recognised by the WHO in 1954. Since that time, it has been the key point of contact for the WHO and the Ministry of Health regarding virological and epidemiological surveillance of influenza. The NIC provides influenza virus isolates to the WHO Global Influenza Surveillance and Response System (GISRS), 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 WHOCC in Melbourne for further characterisation.

**Immunisation coverage**

In 1997, influenza vaccination was made available free to those aged 65 years and older, and in 1999, free vaccinations were extended to people younger than 65 years who were at high risk of complications from influenza [7, 8]. Pregnant women have been offered free vaccinations since the influenza A(H1N1)pdm09 pandemic in 2009 [9].

People younger than 65 years are eligible for free influenza vaccinations if they have any of the following medical conditions or fit into the following population groups:
• cardiovascular disease/cerebrovascular disease (congenital heart disease, ischaemic heart disease, congestive heart failure, rheumatic heart disease, coronary artery disease, angina, heart attack, stroke, other heart conditions and cerebrovascular disease)

• chronic respiratory disease (asthma, if on regular preventive therapy, emphysema, chronic obstructive airways disease, cystic fibrosis, chronic bronchitis, other chronic respiratory disease with impaired lung function)

• certain infants & children (children under the age of 5 years who have been hospitalised for a respiratory illness, or have a history of significant respiratory illness)

• diabetes (Type I and Type II diabetes)

• pregnant women in all trimesters

• cancer (current), excluding basal and squamous skin cancers, if not invasive

• other conditions (autoimmune disease, cerebral palsy, children on long-term aspirin, chronic renal disease, congenital myopathy, epilepsy, haemoglobinopathies, human immunodeficiency virus, hydrocephaly, immunosuppression, motor neurone disease, multiple sclerosis, muscular dystrophy, myasthenia gravis, neuromuscular and central nervous system diseases, Parkinson’s disease, rheumatoid arthritis, sickle cell anaemia, transplant recipients).

Since information is not available on the number of people in the eligible groups listed above, coverage rates have been calculated using the total New Zealand population. This will certainly be an underestimate since it excludes all privately funded influenza immunisations.

Data used to calculate rates

Denominator data used to determine rates of ILI, hospitalisations, mortality and immunisation coverage was derived from 2013 mid-year population estimates published by Statistics New Zealand.

Ethnicity

For different ethnic groups, the number and rates of hospitalisations are based on a prioritised classification of ethnicity, with the Māori ethnic group at the top of the hierarchy, followed by Pacific Peoples, Asian, Middle Eastern/Latin American/African (MELAA) and European or Other ethnicity (including New Zealander) ethnic groups. The National Minimum Dataset (NMDS) and SHIVERS projects use this prioritised classification for ethnicity data.

Oseltamivir resistance monitoring

The National Influenza Centre employed a phenotypic method (fluorometric neuraminidase inhibition assay) for the surveillance of antiviral drug resistance in influenza viruses. In addition, the NIC employed a molecular method (PCR and sequencing) to monitor the H275Y mutation (histidine-to-tyrosine mutation at the codon of 275 in N1 numbering) which confers resistance to oseltamivir.
COMMUNITY-BASED SURVEILLANCE

National sentinel GP-based surveillance

In 2013, 70 sentinel practices were recruited from 18 of New Zealand’s 20 DHBs under National sentinel GP-based surveillance. No practices were recruited from Auckland or Counties Manukau DHBs since these two DHBs participated in the SHIVERS ILI surveillance instead. All DHBs began reporting by the fifth week of surveillance (2 June 2013). Some sentinel practices did not report every week. The average number of practices participating per week was 67, with an average patient roll of 370 633 – approximately 8.3% of the New Zealand population.

During the 2013 influenza season (May to October), a total of 2122 sentinel consultations for ILI were reported. Based on this, the cumulative incidence rate of ILI consultations was 572.5 per 100 000 patient population. This rate is much lower than the cumulative incidence rate for 2012 (1087.0 per 100 000) and 2011 (933.8 per 100 000). The average national weekly consultation rate in 2013 was 21.6 per 100 000 patient population. This rate is also significantly lower than the average weekly rates for 2012 (50.2 per 100 000) and 2011 (37.1 per 100 000).

Extrapolating ILI consultations obtained from the general practice patient population to the New Zealand population, it is estimated that an ILI resulting in a visit to a GP affected 25 598 New Zealanders during the 2013 influenza season (0.6% of total population). This is lower than the estimated number of people affected in 2012 (48 186, 1.1% of total population) and in 2011 (41 133, 0.9% of total population).

Figure 1 compares the weekly consultation rates for ILI in 2013 with the weekly consultation rates for ILI in 2008–2012. Influenza consultation activity remained below the baseline level during the surveillance period (weeks 18–44) in 2013. It peaked in week 37 (9–15 September 2013), with a consultation rate of 47.3 per 100 000 patient population. The peak occurred six weeks later than the peak in 2012 (week 31, 154.1 per 100 000 patient population) and seven weeks later than the peak in 2011 (week 30, 66.1 per 100 000 patient population). Consultation activity then gradually declined.

Figure 1. Weekly consultation rates for ILI in New Zealand, 2008–2013

Note: Following the emergence of influenza A(H1N1)pdm09 in 2009, influenza surveillance and reporting was continued through the summer of 2009/10, beyond the normal autumn, winter and spring reporting period. In 2011, surveillance and reporting were extended by a month to cover the Rugby World Cup held during September and October. In 2013, surveillance and reporting were extended by a month to try to capture the consultation rate peak.
Figure 2 compares the weekly consultation rates for ILI in 2013 with the weekly consultation rates for ILI in 1992–2012. The peak ILI rate in 2013 was the second lowest during the period 1992–2013, the lowest was in 2000. The cumulative incidence rate of 572.5 per 100 000 patient population was the lowest recorded from 1992 to 2013.

Figure 2. Weekly consultation rates for ILI in New Zealand, 1992–2013

For sentinel virological surveillance from May to October 2013, a total of 602 specimens were tested. Of these, 196 (32.6%) specimens were positive for influenza viruses. This is lower than the 399 viruses identified through sentinel surveillance in 2012 and the 336 viruses identified through sentinel surveillance in 2011.

Figure 3 shows the temporal distribution of influenza viruses from sentinel surveillance from weeks 18–44. Overall, influenza viruses were detected in the same time period in 2013 as they were in 2012. The highest peak of influenza virus detection from sentinel surveillance occurred in week 37 (31 viruses). Influenza B viruses predominated in most of the influenza season (weeks 18–37), with a peak in week 37 (9–15 September), comprising 58.1% of all viruses detected.
The temporal distribution of the influenza viruses detected from the sentinel surveillance is compared to influenza viruses detected from all other surveillance systems (non-sentinel surveillance) (Figure 4). The highest peak of influenza virus detection from sentinel surveillance occurred in week 37 (31 viruses) and week 36 (200 viruses) from non-sentinel surveillance. Influenza viruses were identified sporadically as early as January from non-sentinel surveillance. Overall, a total of 2326 influenza viruses were identified from both sentinel and non-sentinel surveillance in 2013. This is lower than the 2425 viruses identified in 2012 but higher than the 1268 viruses identified in 2011.
Figure 5 shows the sentinel average weekly consultation rates for each DHB from May to October 2013. Weekly ILI consultation rates per 100,000 patient population varied among DHBs, with rates above the national average in Auckland (80.0), followed by South Canterbury (72.9), Counties Manukau (41.0), Waitemata (40.1), West Coast (36.3), Whanganui (28.2), Capital & Coast (27.0), Hutt Valley (26.4), Lakes (23.1), and Hawke’s Bay (22.1). See Table 2 for the DHB descriptions.

**Figure 5. Sentinel average weekly consultation rates for influenza by DHB from North to South, 2013**

Note: DHBs marked * did not participate in the national influenza sentinel surveillance, but did participate in the SHIVERS sentinel practice-based surveillance. For details about the SHIVERS ILI surveillance, please refer to the method section on SHIVERS sentinel practice-based surveillance for ILI.

<table>
<thead>
<tr>
<th>DHB code</th>
<th>DHB</th>
<th>DHB code</th>
<th>DHB</th>
</tr>
</thead>
<tbody>
<tr>
<td>NL</td>
<td>Northland</td>
<td>WG</td>
<td>Whanganui</td>
</tr>
<tr>
<td>WM</td>
<td>Waitemata</td>
<td>MC</td>
<td>MidCentral</td>
</tr>
<tr>
<td>AK</td>
<td>Auckland</td>
<td>WR</td>
<td>Wairarapa</td>
</tr>
<tr>
<td>CM</td>
<td>Counties Manukau</td>
<td>HU</td>
<td>Hutt Valley</td>
</tr>
<tr>
<td>WK</td>
<td>Waikato</td>
<td>CC</td>
<td>Capital &amp; Coast</td>
</tr>
<tr>
<td>LS</td>
<td>Lakes</td>
<td>NM</td>
<td>Nelson Marlborough</td>
</tr>
<tr>
<td>BP</td>
<td>Bay of Plenty</td>
<td>WC</td>
<td>West Coast</td>
</tr>
<tr>
<td>TW</td>
<td>Tairawhiti</td>
<td>CB</td>
<td>Canterbury</td>
</tr>
<tr>
<td>TK</td>
<td>Taranaki</td>
<td>SC</td>
<td>South Canterbury</td>
</tr>
<tr>
<td>HB</td>
<td>Hawke's Bay</td>
<td>SN</td>
<td>Southern</td>
</tr>
</tbody>
</table>
Figure 6 shows the distribution of sentinel influenza viruses based on the DHB from which the specimen (swab) was taken. Most of the viruses came from Canterbury, Hawke’s Bay and Capital Coast DHBs. No viruses were identified from Wairarapa or West Coast DHBs.

**Figure 6. Numbers of laboratory-confirmed influenza viruses from sentinel surveillance by DHB, May to October 2013**

![Bar chart showing distribution of influenza viruses by DHB](image)

NB: Auckland and Counties Manukau DHBs did not participate in the national influenza sentinel surveillance. They participated in SHIVERS sentinel practice based surveillance. For details, see relevant section of this report.

**Figure 7. Sentinel swabs received and tested positive for influenza virus by DHB, 2013**

![Chart showing number of swabs and detection rate by DHB](image)

The national influenza virus detection rate for 2013 (Figure 7) was 32.6% (196 viruses from 602 swabs received), which is lower than in 2012, (44.6%, 399 viruses from 895 swabs received) and 2011 (39.2%, 336 viruses from 858 swabs received).
Average cumulative ILI consultation rates by age group were calculated for the sentinel surveillance system (Figure 8). The highest cumulative consultation rates for ILI were in children aged 1–4 years (870.6 per 100,000 patient population) followed by those aged <1 year (658.0 per 100,000 patient population). Elderly people (aged 65 years and older) had the lowest ILI consultation rate of 230.1 per 100,000 patient population.

Figure 8. Sentinel average cumulative consultation rates for ILI by age group, 2013

Figure 9 compares the percentage of influenza viruses detected from sentinel surveillance for each age group. The highest proportions of sentinel influenza viruses were in the 35–49 years (28.0%) and lowest in the 1–4 years (2.0%) age groups.

Figure 9. Percentage of sentinel influenza viruses by age group, 2013
SHIVERS sentinel practice based ILI surveillance

The SHIVERS sentinel GP practices are based in two DHBs in the Auckland region. The ADHB and CMDHB serve a combined population of 837,696 residents and 103,752 patients are enrolled at the 18 ILI sentinel general practices (Figure 10). This is approximately 12% of the total ADHB and CMDHB population.

Figure 10. Geographical distribution of SHIVERS sentinel practices in ADHB and CMDHB

A comparison of the characteristics of the ADHB and CMDHB populations shows some differences in the ethnicity and socioeconomic distribution (age, sex, ethnicity and socioeconomic status). The population in ADHB is slightly older, has a higher proportion of European ethnicity and a higher socioeconomic status (SES) than the CMDHB population, which is slightly younger, has a higher proportion of Pacific and Asian ethnicity and a lower SES.

In the 18 sentinel practices, from 29 April to 3 November 2013, a total of 214,622 GP consultations were recorded and 1945 cases (0.9%, 1945/214,622) met the ILI case definition. Among them, 1856 (95.4%) had a specimen tested for influenza and in 518 (27.9%) cases, an influenza virus was detected. The number of ILI and influenza cases for the same period is shown in Table 3. Influenza peaked in week 37 (ending 15 September).
The temporal distribution of influenza associated ILI cases and non-influenza associated ILI cases during 29 April to 3 November 2013, is shown in Figure 11.

**Figure 11. Weekly resident ILI and influenza positive cases, 29 April to 3 November 2013**

![ILI cases graph](image)

Of the 1945 ILI cases identified, 19.9% were children aged less than five years and 4.2% were adults aged 65 and older. Of the 1945 ILI cases, 1782 were enrolled patients residing in ADHB or CMDHB. This gives an ILI incidence rate of 1717.6 per 100 000 patient population (Table 3). A total of 479 cases from ADHB and CMDHB residents were positive for influenza viruses. This gives an ILI associated influenza incidence of 461.7 per 100 000 patient population.
### Table 3. Demographic characteristics of ILI and influenza cases, 29 April to 3 November 2013

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>ILI &amp; influenza cases among sentinel practices</th>
<th>ILI &amp; influenza cases among ADHB &amp; CMDHB residents</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>ILI cases</td>
<td>ILI cases per 1000 consultations</td>
</tr>
<tr>
<td>Overall</td>
<td>1945</td>
<td>9.1</td>
</tr>
<tr>
<td>Age group (years)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1–4</td>
<td>339</td>
<td>14.4</td>
</tr>
<tr>
<td>5–19</td>
<td>578</td>
<td>17.9</td>
</tr>
<tr>
<td>20–34</td>
<td>303</td>
<td>9.0</td>
</tr>
<tr>
<td>35–49</td>
<td>380</td>
<td>8.5</td>
</tr>
<tr>
<td>50–64</td>
<td>214</td>
<td>5.2</td>
</tr>
<tr>
<td>65–79</td>
<td>70</td>
<td>2.8</td>
</tr>
<tr>
<td>&gt;80</td>
<td>70</td>
<td>1.4</td>
</tr>
<tr>
<td>Unknown</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Ethnicity</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Māori</td>
<td>95</td>
<td>4.6</td>
</tr>
<tr>
<td>Pacific Peoples</td>
<td>392</td>
<td>6.1</td>
</tr>
<tr>
<td>Asian</td>
<td>303</td>
<td>9.9</td>
</tr>
<tr>
<td>European or Other</td>
<td>1151</td>
<td>11.6</td>
</tr>
<tr>
<td>Unknown</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>DHB</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ADHB</td>
<td>1316</td>
<td>11.6</td>
</tr>
<tr>
<td>CMDHB</td>
<td>512</td>
<td>5.1</td>
</tr>
<tr>
<td>Others</td>
<td>117</td>
<td>2.8</td>
</tr>
<tr>
<td>Sex</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>1092</td>
<td>9.1</td>
</tr>
<tr>
<td>Male</td>
<td>852</td>
<td>9.0</td>
</tr>
<tr>
<td>Unknown</td>
<td>1</td>
<td></td>
</tr>
</tbody>
</table>

*Calculated as a percentage of ILI cases tested for influenza viruses. (This may differ from percentage of ILI samples tested for influenza viruses).
Influenza surveillance in New Zealand 2013

Community-based surveillance

The ILI-associated influenza incidence by age group from 29 April to 3 November 2013 is shown in Figure 12. Children aged 5–19 years had the highest ILI-associated influenza rates, followed by those aged 1–4 years and 35–49 years. Infants aged <1 year had the lowest ILI-associated influenza rates.

**Figure 12. ILI associated influenza incidence rates by age-group, 29 April to 3 November 2013**

The ILI-associated influenza incidence by ethnic group from 29 April to 3 November 2013 is shown in Figure 13. People in the Asian ethnic group had the highest ILI-associated influenza and Māori had the lowest.

**Figure 13. ILI associated influenza incidence by ethnic groups, 29 April to 3 November 2013**
The SES distribution by quintile of ILI associated influenza cases is shown in Figure 14. The most deprived quintile (two highest deciles) had a substantially lower incidence rate than the other four.

**Figure 14. ILI associated influenza incidence by socioeconomic status, 29 April to 3 November 2013**

![ILN influenza incidence (cases per 100 000)]

Influenza viruses

From 29 April to 3 November, a total of 1866 ILI specimens were tested for influenza viruses, with 518 (27.8%) testing positive. The details are given in Table 4, below. The majority were influenza A(H3N2) followed by influenza B.

**Table 4. Influenza viruses in ILI cases, 29 April to 3 November 2013**

<table>
<thead>
<tr>
<th>Influenza viruses</th>
<th>ILI</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of specimens tested</td>
<td>1 866</td>
</tr>
<tr>
<td>No. of positive specimens (%)&lt;sup&gt;1&lt;/sup&gt;</td>
<td>518 (27.8)</td>
</tr>
<tr>
<td><strong>Influenza A</strong></td>
<td>313</td>
</tr>
<tr>
<td>A (not subtyped)</td>
<td>49</td>
</tr>
<tr>
<td>A (H1N1)pdm09</td>
<td>39</td>
</tr>
<tr>
<td>A(H1N1)pdm09 by PCR</td>
<td>18</td>
</tr>
<tr>
<td>A/California/7/2009 (H1N1)-like</td>
<td>21</td>
</tr>
<tr>
<td>A(H3N2)</td>
<td>225</td>
</tr>
<tr>
<td>A(H3N2) by PCR</td>
<td>123</td>
</tr>
<tr>
<td>A/Victoria/361/2011 (H3N2)-like</td>
<td>102</td>
</tr>
<tr>
<td><strong>Influenza B</strong></td>
<td>210</td>
</tr>
<tr>
<td>B (lineage not determined)</td>
<td>125</td>
</tr>
<tr>
<td>B (Yamagata lineage-B/Wisconsin/1/2010-like)</td>
<td>84</td>
</tr>
<tr>
<td>B (Victoria lineage-B/Brisbane/60/2008-like)</td>
<td>1</td>
</tr>
<tr>
<td><strong>Influenza and non-influenza co-detection (% of all influenza positive specimens)</strong></td>
<td>36 (6.9)</td>
</tr>
</tbody>
</table>

<sup>1</sup>Number of specimens positive for at least one of the listed viruses; note a specimen may be positive for more than one virus
The temporal distribution of the number and proportion of the influenza viruses is shown in Figure 15. Influenza B was the predominant strain over A(H3N2) from week 19 (ending 12 May) to week 29 (ending 21 July). After week 30 (ending 28 July), A(H3N2) became the predominant strain. Influenza viruses peaked in week 37 (ending 15 September).

Figure 15. Temporal distribution of the number and proportion of influenza viruses from ILI specimens by type and week, 29 April to 3 November 2013

Non-influenza respiratory viruses

From 29 April to 3 November 2013, a total of 1847 ILI specimens were tested for non-influenza viruses and 631 (34.2%) tested positive. Details are given in Table 5.

Table 5. Influenza and non-influenza respiratory viruses among ILI cases, 29 April to 3 November 2013

<table>
<thead>
<tr>
<th>Non-influenza respiratory viruses</th>
<th>ILI</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of specimens tested</td>
<td>1 847</td>
</tr>
<tr>
<td>No. of positive specimens (%)(^1)</td>
<td>631 (34.2)</td>
</tr>
<tr>
<td>Respiratory syncytial virus (RSV)</td>
<td>148</td>
</tr>
<tr>
<td>Parainfluenza 1 (PIV1)</td>
<td>15</td>
</tr>
<tr>
<td>Parainfluenza 2 (PIV2)</td>
<td>41</td>
</tr>
<tr>
<td>Parainfluenza 3 (PIV3)</td>
<td>57</td>
</tr>
<tr>
<td>Rhinovirus (RV)</td>
<td>259</td>
</tr>
<tr>
<td>Adenovirus (AdV)</td>
<td>99</td>
</tr>
<tr>
<td>Human metapneumovirus (hMPV)</td>
<td>77</td>
</tr>
<tr>
<td>Single virus detection (% positive)</td>
<td>569 (90.2)</td>
</tr>
<tr>
<td>Multiple virus detection (% positive)</td>
<td>62 (9.8)</td>
</tr>
</tbody>
</table>

\(^1\)Number of specimens positive for at least one of the listed viruses; note a specimen may be positive for more than one virus
HealthStat GP-based surveillance

Figure 17 shows the weekly rate of ILI per 100,000 GP-registered patients collected by HealthStat sentinel GPs from 2009 to 2013. The 2009 and 2010 ILI consultation data showed similar levels of influenza activity, but this differs markedly from ESR’s sentinel GP-based surveillance, laboratory-based surveillance and hospitalisation data where the overall 2009 influenza activity was much higher than the 2010 activity. The 2009 HealthStat data probably reflected the low sensitivity of coding practices in 2009, and it appears that GP coding practices have improved since 2010. The rate of ILI in 2013 was below average and peaked later in the season than in any year from 2009 to 2012.

Figure 17. HealthStat ILI consultation rates by week from 2009–2013

Data source: From responding practices of original HealthStat GP practice panel
Overall, the trend of the 2013 HealthStat data is similar to ESR’s sentinel GP surveillance (Figure 18). ESR’s sentinel GP surveillance peaked in week 37 (47.1 per 100 000) and the HealthStat data peaked in week 38 (42.9 per 100 000).

**Figure 18. ESR and HealthStat sentinel ILI rates, 2013**

![ESR and HealthStat sentinel ILI rates, 2013](chart)

**Healthline**

Figure 19 shows the weekly number of calls to Healthline for ILI from 2009 to 2013. The number of calls in 2013 was similar to 2012, and lower than most other years in the period 2009–2012. In 2013, Healthline calls peaked in week 36, with 1192 ILI-related calls.

**Figure 19. Weekly numbers of ILI-related calls to Healthline in 2009–2013**

![Weekly numbers of ILI-related calls to Healthline in 2009–2013](chart)

Data source: Healthline New Zealand
HOSPITAL-BASED SURVEILLANCE

SHIVERS hospital-based surveillance for severe acute respiratory infections

From 29 April to 29 September 2013, there were 59,688 acute admissions to ADHB and CMDHB hospitals. A total of 3,537 (5.9%) patients with suspected respiratory infections were assessed in these hospitals. Of these, 1,642 (46.4%) patients met the SARI case definition. Among these SARI patients, 1,261 (76.8%) had laboratory PCR testing for influenza. Of these, 215 (17.0%) had an influenza virus detected.

Of the 3,537 assessed patients, 1,895 (53.6%) did not meet the SARI case definition. A total of 511 (27.0%) of these non-SARI cases were also tested for influenza viruses including 156 systematically sampled cases and 355 clinician ordered cases. Among the tested non-SARI cases, 41 (8.0%) had influenza viruses detected.

The temporal distribution of influenza SARI cases (cases who met the SARI definition, and were positive for influenza) and non-influenza SARI cases from 29 April to 3 November is shown in Figure 20. Overall, weekly numbers of SARI and influenza cases among ADHB and CMDHB residents were lower in 2013 than in 2012.

Table 6 shows the demographic features of the acute admission patients, SARI cases, and SARI influenza positive cases from 29 April to 29 September 2013. Of the 3,537 (5.9%) cases with suspected respiratory infections, 1,642 met the SARI case definition, resulting in 27.5 SARI cases per 1000 acute hospitalisations. This was lower than the 34.2 per 1000 hospitalisations during the same period in 2012. Among all SARI cases, 1,372 (83.6%) were residents of ADHB and CMDHB, giving a cumulative SARI incidence of 163.8 per 100,000 population. This was lower than the 203.5 cases per 100,000 population during the same period in 2012. Of the 215 positive influenza cases, 189 (87.9%) were residents of ADHB and CMDHB, which gives a cumulative influenza incidence of 22.6 per 100,000 population. This is lower than the 34.4 per 100,000 population recorded during the same period in 2012.
### Table 6. Demographic characteristics of SARI cases, 29 April to 29 September 2013

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>All acute admissions</th>
<th>SARI &amp; influenza cases among all hospital patients</th>
<th>SARI &amp; influenza cases among ADHB &amp; CMDHB residents</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>SARI Cases</td>
<td>Cases per 1000 hospitalisations</td>
</tr>
<tr>
<td>Overall</td>
<td>59 688</td>
<td>1 642</td>
<td>27.5</td>
</tr>
<tr>
<td><strong>Age group (years)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;1</td>
<td>2 434</td>
<td>284</td>
<td>116.7</td>
</tr>
<tr>
<td>1–4</td>
<td>4 658</td>
<td>239</td>
<td>51.3</td>
</tr>
<tr>
<td>5–19</td>
<td>7 223</td>
<td>86</td>
<td>11.9</td>
</tr>
<tr>
<td>20–34</td>
<td>10 875</td>
<td>108</td>
<td>9.9</td>
</tr>
<tr>
<td>35–49</td>
<td>9 203</td>
<td>144</td>
<td>15.7</td>
</tr>
<tr>
<td>50–64</td>
<td>10 074</td>
<td>224</td>
<td>22.2</td>
</tr>
<tr>
<td>65–79</td>
<td>9 047</td>
<td>243</td>
<td>26.9</td>
</tr>
<tr>
<td>&gt;80</td>
<td>6 169</td>
<td>154</td>
<td>25.0</td>
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<tr>
<td>Unknown</td>
<td>5</td>
<td>160</td>
<td>12 (12.4)</td>
</tr>
<tr>
<td><strong>Ethnicity</strong></td>
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</tr>
<tr>
<td>Māori</td>
<td>8 143</td>
<td>262</td>
<td>32.2</td>
</tr>
<tr>
<td>Pacific Peoples</td>
<td>12 581</td>
<td>429</td>
<td>34.1</td>
</tr>
<tr>
<td>Asian</td>
<td>8 386</td>
<td>114</td>
<td>13.6</td>
</tr>
<tr>
<td>European or Other</td>
<td>30 133</td>
<td>522</td>
<td>17.3</td>
</tr>
<tr>
<td>Unknown</td>
<td>428</td>
<td>315</td>
<td>23 (15.5)</td>
</tr>
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<td><strong>DHB</strong></td>
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</tr>
<tr>
<td>ADHB</td>
<td>33 097</td>
<td>761</td>
<td>23.0</td>
</tr>
<tr>
<td>CMDHB</td>
<td>26 591</td>
<td>881</td>
<td>33.1</td>
</tr>
<tr>
<td><strong>Sex</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>31 719</td>
<td>741</td>
<td>23.4</td>
</tr>
<tr>
<td>Male</td>
<td>27 963</td>
<td>743</td>
<td>26.6</td>
</tr>
<tr>
<td>Unknown</td>
<td>6</td>
<td>158</td>
<td>12 (12.4)</td>
</tr>
</tbody>
</table>

*Calculated as a percentage of SARI cases tested for influenza viruses. (This may differ from percentage of SARI samples tested for influenza viruses).
The cumulative SARI-associated influenza incidence by age group from 29 April to 29 September presents a U-shaped curve, as shown in Figure 21. This was very similar to previous results obtained for the 2011/2012 result. High rates of influenza hospitalisation were recorded in very young and very old people. Infants aged <1 year had the highest influenza hospitalisation rate (122.0 per 100 000) followed by the elderly (72.3 per 100 000 for those aged 65–79 years and 69.3 per 100 000 for 80 years and over).

Figure 21. Cumulative SARI associated influenza hospitalisation rate by age group, 29 April to 29 September 2013

The cumulative SARI associated influenza incidence by ethnic group from 29 April to 29 September is shown in Figure 22. Pacific Peoples ethnic group had the highest hospitalisation rate. This was followed by Māori, European or Other and Asian ethnic groups. This trend is similar to the SARI 2011/12 result obtained in 2012.

Figure 22. SARI associated influenza hospitalisation rate by ethnic group, 29 April to 29 September 2013
Rates of influenza incidence among SARI cases by SES are shown in Figure 23. Cases in the lowest SES quintile have the highest rate, being much higher than any other group.

**Figure 23. SARI-associated influenza hospitalisation rate by socioeconomic status, 29 April to 29 September 2013**

![Influenza incidence by SES quintile](image)

<table>
<thead>
<tr>
<th>NZDep1–2</th>
<th>NZDep3–4</th>
<th>NZDep5–6</th>
<th>NZDep7–8</th>
<th>NZDep9–10</th>
</tr>
</thead>
<tbody>
<tr>
<td>15.8</td>
<td>9.7</td>
<td>29.0</td>
<td>24.2</td>
<td>51.2</td>
</tr>
</tbody>
</table>

**Severe hospital outcomes**

A measure of the severity of an acute hospitalisation is an admission to an intensive care unit (ICU), or death recorded whilst in hospital.

During the period from 29 April to 29 September 2013, there were a total of 539 admissions to an ICU, 66 (12.2%) of which met the SARI definition; 4.0% of SARI cases (66/1642) were therefore admitted to ICU. A total of 15.6% (10/64) of the tested ICU cases were positive for influenza viruses, and the influenza incidence rate among SARI cases who were admitted to ICU was 1.0 per 100 000 (95% CI: 0.4, 1.9).

The ICU incidence rate was four to six times higher among Maori and Pacific Peoples than the other ethnicities, and concentrated among very young cases.

During the period from 29 April to 29 September 2013 a total of 426 hospital deaths were recorded, seven (1.6%) of which met the SARI definition; 0.4% of SARI cases (7/1642) died. Of the six fatal cases that were tested, one case (16.7%, 1/6) was positive for influenza virus, from a non-ADHB and non-CMDHB resident.
Influenza surveillance in New Zealand 2013
Hospital-based surveillance

Influenza viruses

From 29 April to 29 September 2013, 1353 specimens from SARI patients were tested and 232 (17.1%) were positive for influenza viruses including 156 influenza A and 76 influenza B viruses (Table 7).

Table 7. Influenza viruses among SARI cases, 29 April to 29 September 2013

<table>
<thead>
<tr>
<th>SARI cases virology</th>
<th>Cases</th>
<th>ICU</th>
<th>Deaths</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of specimens tested</td>
<td>1353</td>
<td>81</td>
<td>12</td>
</tr>
<tr>
<td>No. of positive specimens (%)¹</td>
<td>232 (17.1)</td>
<td>11 (13.6)</td>
<td>1 (8.3)</td>
</tr>
<tr>
<td><strong>Influenza A</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A (not subtyped)</td>
<td>30</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>A (H1N1)pdm09</td>
<td>15</td>
<td>4</td>
<td>0</td>
</tr>
<tr>
<td>A(H1N1)pdm09 by PCR</td>
<td>9</td>
<td>4</td>
<td>0</td>
</tr>
<tr>
<td>A/California/7/2009 (H1N1)-like</td>
<td>6</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>A/H3N2</td>
<td>111</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>A(H3N2) by PCR</td>
<td>63</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>A/Victoria/361/2011 (H3N2)-like</td>
<td>48</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td><strong>Influenza B</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>B (lineage not determined)</td>
<td>48</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>B (Yamagata lineage-B/Wisconsin/1/2010-like)</td>
<td>26</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>B (Victoria lineage-B/Brisbane/60/2008-like)</td>
<td>2</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td><strong>Influenza and non-influenza co-detection (% positive)</strong></td>
<td>27 (11.6)</td>
<td>0 (0.0)</td>
<td>0 (0.0)</td>
</tr>
</tbody>
</table>

¹Number of specimens positive for at least one of the listed viruses; note a specimen may be positive for more than one virus
The temporal distribution of the number and proportion of the influenza viruses is shown in Figure 24. Influenza B was the predominant strain from week 18 (ending 5 May) to week 27 (ending 27 July). After week 27 (ending 27 July), A(H3N2) became the predominant strain. Influenza viruses peaked in week 38.

**Figure 24. Temporal distribution of the number and proportion of influenza viruses from SARI specimens by type and week**

In addition to testing for influenza viruses, the SARI surveillance also tested for the presence of seven non-influenza viruses. From 29 April to 29 September 2013, 870 SARI specimens were tested for non-influenza respiratory viruses. Of these, 388 (44.6%) were positive. Details are given in Table 8.

**Table 8. Non-influenza respiratory viruses among SARI cases, 29 April to 29 September 2013**

<table>
<thead>
<tr>
<th>SARI cases virology</th>
<th>Cases</th>
<th>ICU</th>
<th>Deaths</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of specimens tested</td>
<td>870</td>
<td>24</td>
<td>3</td>
</tr>
<tr>
<td>No. of positive specimens (%)</td>
<td>388 (44.6)</td>
<td>12 (50)</td>
<td>2 (66.7)</td>
</tr>
<tr>
<td>Respiratory syncytial virus (RSV)</td>
<td>162</td>
<td>4</td>
<td>0</td>
</tr>
<tr>
<td>Parainfluenza 1 (PIV1)</td>
<td>3</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Parainfluenza 2 (PIV2)</td>
<td>18</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Parainfluenza 3 (PIV3)</td>
<td>34</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Rhinovirus (RV)</td>
<td>168</td>
<td>4</td>
<td>0</td>
</tr>
<tr>
<td>Adenovirus (AdV)</td>
<td>60</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>Human metapneumovirus (hMPV)</td>
<td>46</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Single virus detection (% positive)</td>
<td>303 (78.1)</td>
<td>8 (66.7)</td>
<td>2 (100.0)</td>
</tr>
<tr>
<td>Multiple virus detection (% positive)</td>
<td>85 (21.9)</td>
<td>4 (33.3)</td>
<td>0 (0.0)</td>
</tr>
</tbody>
</table>

1Number of specimens positive for at least one of the listed viruses; note a specimen may be positive for more than one virus
The temporal distribution of the number and proportion of non-influenza respiratory viruses is shown in Figure 25. High RSV activity occurred in July and August with a peak in week 27 (ending 7 July), nine weeks earlier than the influenza peak. The rhinovirus activity was at a constant level from May to September.

**Figure 25. Temporal distribution of the number and proportion of non-influenza viruses from SARI specimens by type and week, 2013**
Ministry of Health data on publicly funded hospital discharges

Influenza hospitalisations by week discharged are shown in Figure 26 and indicate that 86.7% (678) of these occurred from June to October. The highest number of hospitalisations (248) occurred in September (weeks 35–39). Hospitalisations peaked in weeks 36 and 37 which was similar to non-sentinel virus numbers which peaked in week 36, and sentinel ILI consultations and sentinel virus numbers which peaked in week 37.

Figure 26. Influenza hospital discharges by week, 2013

![Figure 26. Influenza hospital discharges by week, 2013](image)

Source: Ministry of Health, NMDS (Hospital Events)

The number of influenza hospitalisations in 2013 ranked the fourth highest during the period from 2000 to 2013 (Figure 27). In 2013, there were 782 hospitalisations for influenza, lower than the 1076 hospitalisations reported in 2012 but higher than the 526 hospitalisations reported in 2011. A substantially higher number of hospitalisations occurred in 2009 and 2010, due to the 2009 pandemic. The relatively high number of hospitalisations in 2012 and 2013 may be partly due to the SHIVERS SARI surveillance with comprehensive case ascertainment and thorough laboratory testing.
Influenza surveillance in New Zealand 2013

Hospital-based surveillance

Figure 27. Influenza hospital discharges, 2000–2013

Source: Ministry of Health, NMDS (Hospital Events)

Figure 28 compares the hospitalisation rates in 2013 by age group. In 2013 by far the highest hospitalisation rates occurred in children aged <1 year (142.0 per 100 000 patient population, 85 hospitalised). This was over six times the next highest rate of 22.0 per 100 000, 140 hospitalised, for adults aged 65 years and over.

Figure 28. Influenza hospital discharge rates by age group, 2013

Source: Ministry of Health, NMDS (Hospital Events)
The ethnic distribution of influenza hospitalisations in 2013 is shown in Figure 29. Pacific Peoples had the highest hospitalisation rate (51.8 per 100 000, 143 hospitalised), followed by MELAA with 30.0 per 100 000 (15 hospitalised). European or Other ethnic group had the lowest rate of hospitalisations (13.1 per 100 000, 389 hospitalised).

![Figure 29. Hospital discharge rates by prioritised ethnic group in 2013](image)

Source: Ministry of Health, NMDS (Hospital Events)

There were 16 deaths from influenza in 2011 (the most recent year for which mortality data is available) – a mortality rate of 0.39 per 100 000 population. This rate is lower than the rate of 1.14 per 100 000 population reported in 2009. The highest mortality rate between 1990 and 2011 was recorded in 1996, with a rate of 2.52 per 100 000 (Figure 30).

![Figure 30. Influenza mortality rates 1990–2011](image)

Source: Ministry of Health, Mortality Collection
Comparison of SARI and National Minimum Dataset (NMDS) hospitalisations

The SARI influenza hospitalisation rates from SHIVERS (SHIVERS ADHB and CMDHB populations) were compared to the national data (New Zealand population) from the NMDS influenza hospitalisation rates for each age group. Both the SARI hospitalisations and NMDS recorded the highest influenza hospitalisation rates in the <1 year age group, with rates of 137.2 per 100,000 and 95.2 per 100,000 respectively. People aged 80 years and over had the next highest rates (79.2 and 17.8 per 100,000), followed by those aged 65–79 years (77.6 and 15.2 per 100,000) (Figure 31). Higher influenza hospitalisation rates from SARI surveillance are likely to be due to the rigorous case ascertainment and more frequent testing of respiratory illness admissions as a result of the SHIVERS research programme.

Figure 31. Age-specific influenza hospitalisation rates from the NMDS and SHIVERS data, 29 April to 29 September, 2013, ADHB and CMDHB

The SARI influenza hospitalisation rates from SHIVERS were also higher than the national data from the NMDS influenza hospitalisation rates for each ethnic group. SARI surveillance and NMDS-coded influenza hospitalisations both recorded the highest influenza hospitalisation rates for Pacific Peoples (52.2/100,000 and 39.5/100,000 respectively). Māori ethnic group had the second highest rate (29.9/100,000 and 18.3/100,000) (Figure 32).
Influenza surveillance in New Zealand 2013

Hospital-based surveillance

Figure 32. Ethnic-specific influenza hospitalisation rates for NMDS and SHIVERS data, 29 April to 29 September 2013

Laboratory-based non-sentinel surveillance – for outpatients and hospital inpatients

For non-sentinel surveillance from January to December 2013, a total of 7956 specimens were tested. Of these, 2130 (26.8%) specimens tested positive for influenza viruses. This is higher than the 2026 and 932 viruses identified through non-sentinel surveillance in 2012 and 2011 respectively, but lower than the 4276 viruses identified in 2009.

Figure 33 shows the temporal distribution of influenza viruses reported by type and subtype each week from non-sentinel surveillance for weeks 18–44. Influenza A(H3N2) and B viruses co-circulated in New Zealand with a peak in week 36 (2–8 September 2013) and accounted for 45.7% and 31.8%, respectively of all influenza viruses identified.

Figure 33. Influenza viruses from non-sentinel surveillance by type and week reported, 2013

*Data shown from weeks 18–44 only.
IMMUNISATION COVERAGE

Influenza vaccine coverage in New Zealand in 2013 was higher than the previous peak in 2010 (Figure 34). The coverage rate of publicly funded influenza vaccine, used during the 2013 seasonal influenza immunisation programme was 280 doses per 1000 population, 19.3% higher than the 226 doses per 1000 population administered in 2012. The coverage rate for people 65 years and older was 67.4%; slightly higher than the coverage rate of 64.4% achieved in 2012 (Immunisation Benefit Claims Data, Sector Services, Ministry of Health). No data are available on privately funded immunisations.

Figure 34. Influenza vaccine coverage, 1990–2013

At least 1 251 240 doses of the seasonal trivalent influenza vaccine were distributed in New Zealand in the 2013 season. Table 9 shows the estimated number of people who received the funded influenza vaccine in seven age groups.

Table 9. Influenza coverage by age group, 2013

<table>
<thead>
<tr>
<th>Age group (years)</th>
<th>Total vaccines received</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;1</td>
<td>1 938</td>
</tr>
<tr>
<td>1–4</td>
<td>20 198</td>
</tr>
<tr>
<td>5–19</td>
<td>47 220</td>
</tr>
<tr>
<td>20–34</td>
<td>28 205</td>
</tr>
<tr>
<td>35–49</td>
<td>54 474</td>
</tr>
<tr>
<td>50–64</td>
<td>121 060</td>
</tr>
<tr>
<td>65+</td>
<td>428 036</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>701 131</strong></td>
</tr>
</tbody>
</table>

Source: Immunisation benefits claims data, Sector Services, Ministry of Health
VIRUS STRAIN CHARACTERISATION
VIRUS STRAIN CHARACTERISATION

Circulating viral strains in 2013

A total of 2326 influenza viruses were detected and reported in 2013, with influenza A representing 60.4% (1405/2326) and influenza B 39.6% (921/2326) of all influenza viruses (Table 10). The influenza B virus strain represented 44.6% (921/2066) of all typed and subtyped viruses. Seasonal influenza A(H3N2) strain represented 36.3% (845/2326) of all viruses and 40.9% (845/2066) of all typed and subtyped viruses. The influenza A(H1N1)pdm09 virus represented 12.9% (300/2326) of all viruses and 14.5% (300/2066) of all typed and subtyped viruses.

Table 10. Influenza virus identifications by type and subtype, 2013

<table>
<thead>
<tr>
<th>Viruses</th>
<th>All viruses (%)</th>
<th>Typed/Sub-typed (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Influenza A (not sub-typed)</td>
<td>260 (11.2)</td>
<td></td>
</tr>
<tr>
<td>Influenza A(H1N1)pdm09</td>
<td>300 (12.9)</td>
<td>300 (14.5)</td>
</tr>
<tr>
<td>A(H1N1)pdm09 by PCR</td>
<td>224 (9.6)</td>
<td>224 (10.8)</td>
</tr>
<tr>
<td>A/California/7/2009 (H1N1)-like</td>
<td>76 (3.3)</td>
<td>76 (3.7)</td>
</tr>
<tr>
<td>Influenza A(H3N2)</td>
<td>845 (36.3)</td>
<td>845 (40.9)</td>
</tr>
<tr>
<td>A(H3N2) by PCR</td>
<td>544 (23.4)</td>
<td>544 (26.3)</td>
</tr>
<tr>
<td>A/Victoria/361/2011 (H3N2)-like</td>
<td>301 (12.9)</td>
<td>301 (14.6)</td>
</tr>
<tr>
<td>Influenza B</td>
<td>921 (39.6)</td>
<td>921 (44.6)</td>
</tr>
<tr>
<td>B by PCR</td>
<td>602 (25.9)</td>
<td>602 (29.1)</td>
</tr>
<tr>
<td>B/Victoria (B/Brisbane)</td>
<td>6 (0.3)</td>
<td>6 (0.3)</td>
</tr>
<tr>
<td>B/Yamagata (B/Wisconsin)</td>
<td>313 (13.5)</td>
<td>313 (15.2)</td>
</tr>
<tr>
<td>Total</td>
<td>2326 (100.0)</td>
<td>2066 (100.0)</td>
</tr>
</tbody>
</table>

Figure 35 shows the influenza virus identifications by type and subtype for each week throughout 2013.

Figure 35. Total influenza viruses by type and week specimen taken, 2013
Figure 36 shows the majority of influenza B infections were in those aged 5–19 years and 35–49 years, while influenza A(H3N2) infections were highest in the 65 years and over age group. There were very few A(H1N1)pdm09 viruses identified in those aged 65 years and older.

Figure 36. Number of laboratory confirmed influenza, 1 January to 31 December 2013, by subtype and age group

Figure 37 shows the general pattern of influenza virus identifications. Influenza A and B viruses co-circulated throughout the season.

Figure 37. Total influenza A and B viruses by week specimen taken, 2013
Figure 38 shows the number and percentage of typed and subtyped (not total) influenza viruses from 1990–2013. Higher annual numbers of influenza viruses were detected in New Zealand during 2009–2013 than in the previous years. There were two reasons: 1) Since PCR was introduced as a screening assay in NZ virus laboratory network in 2009, more specimens were tested as PCR can cope with higher volume of testing than viral isolations; 2) SHIVERS SARI and ILI surveillance have been established in New Zealand since 2012, resulting in more influenza testing.

There are noticeable changes in terms of the predominant strains.

- The influenza A(H1N1)pdm09 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 numbers of hospitalisations (193 in 1992, 228 in 2000 and 379 in 2001). No seasonal A(H1N1) viruses have been detected since 2010.
Influenza surveillance in New Zealand 2013
Virus strain characterisation

Figure 39 shows the number and percentage of all antigenically typed B viruses from 1990 to 2013. Since the introduction of the B/Victoria lineage viruses into New Zealand in 2002, this strain has predominated over the B/Yamagata lineage viruses in three yearly cycles (2002, 2005, 2008 and 2011).

Influenza A(H1N1)pdm09

In 2013, 76 representative influenza A(H1N1)pdm09 isolates were antigenically subtyped. The results from the NIC and WHOCC-Melbourne indicated that most of the currently circulating influenza A(H1N1)pdm09 viruses were antigenically closely related to the vaccine candidate strain A/California/7/2009 (H1N1) [10].

The genetic analysis was conducted for the hemagglutinin (HA) gene and neuraminidase gene of representative influenza A(H1N1)pdm09 viruses. The New Zealand isolates along with isolates from Australia and other countries, exhibited increasing genetic diversity with two major subclades designated as groups six and seven (CDC designation, Appendix A). However, it appears that these genetic changes have not resulted in significant antigenic changes [11]. No H275Y mutations (see below) were detected from any A(H1N1)pdm09 virus from New Zealand.

Influenza A(H3N2)

In 2013, 301 representative seasonal influenza A(H3N2) isolates were antigenically subtyped. The results indicated that most of the New Zealand isolates, as well as isolates from Australia and other countries, are antigenically closely related to the vaccine strain A/Victoria/361/2011-like or A/Texas/50/2012-like strain. Genetically, most of the A(H3N2) viruses were closely related to A/Victoria/361/2011-like strain, falling into group three with a few viruses in groups five and six (CDC designations, Appendix B).
Influenza B

In 2013, representative seasonal influenza B/Victoria lineage isolates (B/Brisbane/60/2008-like) (6) and B/Yamagata lineage isolates (B/Wisconsin/1/2010-like, the current vaccine strain) (313) were antigenically typed. The results indicated that the B/Yamagata isolates from New Zealand, as well as isolates from Australia and other countries, had antigenically drifted from B/Wisconsin/1/2010-like viruses to B/Massachusetts/2/2012-like viruses. The results of the genetic analysis of the HA gene of influenza B viruses indicated that the B/Victoria and B/Yamagata lineage viruses fell into group one and groups two and three respectively (CDC designations, Appendices C and D).

Oseltamivir resistance monitoring

In 2013, 712 influenza viruses were tested for resistance to oseltamivir and 710 for resistance to zanamivir by a phenotypic assay. All viruses were found to be sensitive to oseltamivir (Table 11) and zanamivir (Table 12).

From 2006 to 2007, all influenza A(H1N1) viruses tested were sensitive to oseltamivir. In 2008, only six seasonal A(H1N1) viruses (0.8%) were detected, of which only four were available for antiviral susceptibility testing, and all were found to be resistant to oseltamivir. The results of the fluorometric neuraminidase inhibition assay indicated that the four viruses had highly reduced sensitivities to oseltamivir, with IC50 values in the 500–1700 nM range, typical of the oseltamivir-resistant A(H1N1) viruses that have been emerging globally in recent years. Genetic analysis of the neuraminidase gene confirmed that the four viruses had the H275Y mutation (histidine-to-tyrosine at codon 275 in N1 nomenclature), conferring resistance to oseltamivir. None of the patients or their close contacts had received oseltamivir prior to sample collection. In 2009, 25 seasonal A(H1N1) viruses were phenotypically tested and all were resistant to oseltamivir. However, all influenza A(H1N1)pdm09 isolates tested between 2009 and 2011 were sensitive to oseltamivir.

In 2012, two A(H1N1)pdm09 viruses were resistant to oseltamivir. The first was detected from a 26 year old male who was hospitalised with acute upper respiratory infection within seven days of returning to New Zealand from India. The results of the fluorometric neuraminidase inhibition assay indicated that the virus had highly reduced sensitivity to oseltamivir with an IC50 value of 271 nM; 821 times higher than the mean IC50 value (0.32 nM). The sequencing analysis of the neuraminidase gene confirmed that the virus had the H275Y mutation (histidine-to-tyrosine at codon 275 in N1 nomenclature), conferring resistance to oseltamivir. In addition, this virus is genetically closer to the Indian A(H1N1)pdm09 viruses than the New Zealand A(H1N1)pdm09 viruses. The second oseltamivir resistant virus was detected in a seven month old female Samoan, who was hospitalised with suspected pneumonia but had not travelled overseas prior to hospitalisation. The results of the fluorometric neuraminidase inhibition assay indicated that the virus had highly reduced sensitivity to oseltamivir with an IC50 value of 316 nM, 958 times higher than the mean. Neither patient took any oseltamivir medication before or during hospitalisation.
Table 11. Antiviral susceptibility to oseltamivir for influenza viruses in New Zealand, 2006–2013

<table>
<thead>
<tr>
<th>Influenza type/sub-type</th>
<th>2006</th>
<th>2007</th>
<th>2008</th>
<th>2009</th>
<th>2010</th>
<th>2011**</th>
<th>2012**</th>
<th>2013</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Influenza B</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Number of isolates tested</td>
<td>1</td>
<td>132</td>
<td>306</td>
<td>-</td>
<td>1</td>
<td>244</td>
<td>64</td>
<td>316</td>
</tr>
<tr>
<td>Mean IC50* (nM)</td>
<td>-</td>
<td>37.5</td>
<td>26.5</td>
<td>-</td>
<td>-</td>
<td>32.1</td>
<td>11.2</td>
<td>14.3</td>
</tr>
<tr>
<td>Standard deviation (nM)</td>
<td>-</td>
<td>22.5</td>
<td>16.9</td>
<td>-</td>
<td>-</td>
<td>20.2</td>
<td>5.8</td>
<td>8.1</td>
</tr>
<tr>
<td>Minimum IC50 (nM)</td>
<td>-</td>
<td>0.9</td>
<td>0.22</td>
<td>-</td>
<td>-</td>
<td>4.1</td>
<td>4.8</td>
<td>0.1</td>
</tr>
<tr>
<td>Maximum IC50 (nM)</td>
<td>-</td>
<td>97.4</td>
<td>87.8</td>
<td>-</td>
<td>-</td>
<td>182.7</td>
<td>31.8</td>
<td>51.1</td>
</tr>
<tr>
<td><strong>Influenza A(H3N2)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Number of isolates tested</td>
<td>189</td>
<td>45</td>
<td>120</td>
<td>-</td>
<td>1</td>
<td>224</td>
<td>271</td>
<td>321</td>
</tr>
<tr>
<td>Mean IC50 (nM)</td>
<td>0.7</td>
<td>0.38</td>
<td>0.28</td>
<td>-</td>
<td>-</td>
<td>0.4</td>
<td>0.41</td>
<td>0.32</td>
</tr>
<tr>
<td>Standard deviation (nM)</td>
<td>0.27</td>
<td>0.26</td>
<td>0.17</td>
<td>-</td>
<td>-</td>
<td>0.21</td>
<td>0.19</td>
<td>0.15</td>
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<tr>
<td>Minimum IC50 (nM)</td>
<td>0.06</td>
<td>0.07</td>
<td>0.01</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>0.06</td>
<td>0.06</td>
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<tr>
<td>Maximum IC50 (nM)</td>
<td>1.4</td>
<td>1.13</td>
<td>1.08</td>
<td>-</td>
<td>-</td>
<td>1.5</td>
<td>1.22</td>
<td>0.88</td>
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<td><strong>Seasonal influenza A(H1N1)</strong></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Number of isolates tested</td>
<td>18</td>
<td>136</td>
<td>4</td>
<td>25</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
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<tr>
<td>Mean IC50 (nM)</td>
<td>1.26</td>
<td>0.81</td>
<td>768</td>
<td>1385</td>
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<tr>
<td>Standard deviation (nM)</td>
<td>0.89</td>
<td>0.64</td>
<td>287</td>
<td>1996</td>
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<td>-</td>
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<tr>
<td>Minimum IC50 (nM)</td>
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<td>0.05</td>
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<tr>
<td><strong>Influenza A(H1N1)pdm09</strong></td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
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<td>Number of isolates tested</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>483</td>
<td>334</td>
<td>29</td>
<td>95</td>
</tr>
<tr>
<td>Mean IC50 (nM)</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>0.4</td>
<td>0.68</td>
<td>0.53</td>
<td>0.33</td>
</tr>
<tr>
<td>Standard deviation (nM)</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>0.24</td>
<td>0.41</td>
<td>0.26</td>
<td>0.19</td>
</tr>
<tr>
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<td>-</td>
<td>-</td>
<td>-</td>
<td>0.09</td>
<td>0.01</td>
<td>0.18</td>
<td>0.09</td>
</tr>
<tr>
<td>Maximum IC50 (nM)</td>
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<td>-</td>
<td>-</td>
<td>-</td>
<td>1.4</td>
<td>2.05</td>
<td>1.31</td>
<td>316</td>
</tr>
</tbody>
</table>

*IC50; inhibitory concentration of the drug at which a 50% reduction in enzymatic activity is observed.
** Mean and standard deviation calculated for 2011 and 2012 includes four outliers deemed to be resistant to oseltamivir (having IC50 values >10-fold higher than the overall mean for a given subtype recorded for all years). Four outliers were excluded in mean and standard deviation calculations: two pandemic influenza A(H1N1)pdm09 virus in 2012 and two influenza B viruses in 2011.

Table 12. Antiviral susceptibility to zanamivir for influenza viruses, 2013

<table>
<thead>
<tr>
<th>Zanamivir</th>
<th>Influenza B</th>
<th>A(H1N1)pdm09</th>
<th>A(H3N2)</th>
</tr>
</thead>
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<tr>
<td>Number of isolates tested</td>
<td>314</td>
<td>72</td>
<td>324</td>
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<td>Mean IC50 (nM)</td>
<td>1.28</td>
<td>0.192</td>
<td>0.339</td>
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<tr>
<td>Standard Deviation (nM)</td>
<td>0.78</td>
<td>0.15</td>
<td>0.164</td>
</tr>
<tr>
<td>Minimum IC50 (nM)</td>
<td>0.02</td>
<td>0.01</td>
<td>0.07</td>
</tr>
<tr>
<td>Maximum IC50 (nM)</td>
<td>5.6</td>
<td>1.07</td>
<td>1.42</td>
</tr>
</tbody>
</table>
SOUTHERN HEMISPHERE VACCINE STRAIN RECOMMENDATIONS

In October 2013, the Australian Influenza Vaccine Committee (AIVC), which includes a New Zealand representative, met to decide on the composition of the influenza vaccine for the 2014 winter season for New Zealand, Australia and South Africa. During these discussions, the following trends were noted.

**Influenza A(H1N1)**

The epidemiological data from the New Zealand 2013 influenza season, along with most other Southern Hemisphere countries, indicated that the influenza A(H1N1)pdm09 virus has replaced seasonal A(H1N1) virus since 2009. The WHOCC in Melbourne has analysed 616 influenza A(H1N1)pdm09 isolates from three countries (including New Zealand) since January 2013 and the antigenic data from these isolates indicates that the current circulating influenza A(H1N1)pdm09 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 influenza A(H1N1)pdm09 isolates.

Based on Southern Hemisphere and global data, the WHO Consultative Group and the AIVC recommended that the 2014 vaccines contain a pandemic influenza A/California/7/2009 (H1N1)-like strain as the H1 component.

**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 has analysed 358 A(H3N2) isolates from 11 countries since January 2013. Most of the recent isolates were genetically and antigenically similar to A/Victoria/361/2011-like or A/Texas/50/2012-like viruses (a virus antigenically similar to A/Victoria/361/2011-like virus). Current vaccines containing the A/Texas/50/2012-like antigen stimulated anti-HA antibodies of similar geometric mean HI titres to the cell-propagated vaccine virus and to recent A(H3N2) isolates. As a result, an A/Texas/50/2012-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 2014.

**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 further variants of the B/Yamagata/16/88 lineage (the most recent representative strain being B/Wisconsin/1/2010) 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, with the most recent representative strain being 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/Wisconsin/1/2010 is the current reference strain) continued to be isolated worldwide in 2013. Varying proportions of the two lineages were seen with an increase of the proportion of B/Yamagata/16/88 lineage viruses in many Southern Hemisphere countries. The majority of B/Yamagata/16/88 lineage isolates had drifted antigenically from the B/Wisconsin/1/2010-like strain to B/Massachusetts/2/2012-like. Current vaccines containing the B/Massachusetts/2/2012-like antigen stimulated HA antibodies that were similar in titre to the vaccine virus and to recently isolated B/Yamagata lineage viruses. In light of the increase in the proportion of B/Yamagata/16/88 lineage viruses relative to B/Victoria/2/87 lineage viruses, the WHO Consultative Group and the recommended vaccines containing a B/Massachusetts/2/2012 strain be the B component of the influenza vaccine for the Southern Hemisphere for 2014.

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

The recommended influenza vaccine formulation for New Zealand in 2014 is:

- A(H1N1) an A/California/7/2009 (H1N1)pdm-like strain*
- A(H3N2) an A/Texas/50/2012 (H3N2)-like strain
- B a B/Massachusetts/2/2012-like strain

*Note: A/California/7/2009 is an influenza A(H1N1)pdm09 strain
**DISCUSSION**

Sentinel GP based influenza surveillance, a syndromic surveillance system, is an effective tool for monitoring the disease in the community during an epidemic. It has operated continuously in New Zealand since its establishment in 1991 [3, 5]. Sentinel influenza surveillance is a relatively stable system that monitors disease trends in the community year by year. Active syndromic surveillance systems are increasingly being used to detect emerging and re-emerging pathogens [12, 13]. Influenza surveillance is also a key strategy for improving New Zealand’s preparedness for an influenza pandemic [14]. The usefulness of sentinel surveillance during a pandemic was tested in 2009 and the system has since been adapted to monitor the early and late stages of a pandemic.

Based on sentinel consultation data, the overall influenza activity in 2013 is at a low level. Comparing data for the past 16 years (1997–2013), the weekly consultation rate peak and cumulative consultation rate for ILI in 2013 were the second lowest during this period. It is estimated that ILI resulting in a visit to a GP affected over 25 598 New Zealanders in 2013; about 0.6% of the population. The number of cases reported through the sentinel network, however, is likely to considerably underestimate the true number, as many people do not consult a GP when they have an ILI.

Consultation rates varied greatly among DHBs. The use of a common case definition for the purposes of surveillance should minimise regional differences in the diagnosis criteria but in DHBs where a single practice or a small number of practices participate, consultation rates are more likely to be subject to variations in individual diagnostic practices. Sentinel practices with small registered populations can also produce much greater fluctuations in ILI consultation rates.

Virological surveillance for outpatients and hospital inpatients (referred to as non-sentinel surveillance) complements sentinel surveillance. Non-sentinel surveillance provides useful information for the characterisation of circulating influenza viruses and monitors the emergence of novel strains with pandemic potential. However, current non-sentinel surveillance does not provide robust epidemiologic data with good denominator information. The recent emergence of the influenza A(H1N1)pdm09 virus highlights the need for surveillance to better define those most at risk from SARI resulting from influenza [15]. Expansion of existing non-sentinel surveillance to include the systematic collection of epidemiological data on hospitalised SARI cases would enable the factors that place the most vulnerable people at risk to be described, and facilitate targeted intervention. This would also establish a surveillance platform for other endemic and emerging respiratory pathogens such as MERS Coronavirus.

In October 2011, the CDC commissioned ESR to conduct a five-year study of influenza, including vaccine effectiveness, in the Southern Hemisphere. The study (SHIVERS) is a multi-disciplinary and multi-centre collaboration between ESR, Auckland and Counties Manukau DHBs, the Universities of Otago and Auckland, the WHOCC at St Jude Children Hospital in Memphis and the CDC in Atlanta. Hospital-based SARI surveillance has been established and fully functioning since 30 April 2012 in Auckland and Counties Manukau DHBs. In 2013, its second year, the SHIVERS project was extended to include sentinel practice-based ILI surveillance in ADHB and CMDHB.

Rates of influenza-associated GP consultations and hospitalisations from the SHIVERS study were markedly different by age group. Influenza-associated hospitalisation rates were highest in the very young (0–4 years) and the elderly (≥65 years). Influenza-associated GP consultation rates, however, showed the opposite pattern, with a higher rate in pre-schoolers, school-aged children and adults, but a lower rate in infants (<1 year) and the elderly (≥65 years). The differences in hospitalisation and GP consultation rates by age are well documented [16, 17] and are likely to result from multiple influences including differences in host immune response, virus pathogenesis, clinical severity and health seeking behaviour among different age groups and their parents/caregivers.
A preliminary analysis of influenza rates by ethnicity, found that Māori and Pacific Peoples experienced the highest rates of influenza-associated hospitalisations but the lowest rates of influenza-associated GP consultations, while conversely, the Asian ethnic group showed the opposite trend. When SES was considered, the most deprived populations (NZDep 9–10) were found to have the highest rates of influenza-associated hospitalisations but the lowest rates of influenza-associated GP consultations. Higher hospitalisation rates from seasonal and pandemic influenza have been reported in indigenous groups in the United States, New Zealand and Australia [18, 19]. However, it is difficult to explain why Māori and Pacific Peoples had the highest rates of influenza-associated hospitalisations but the lowest rates of influenza-associated GP consultations. It remains unclear if these patterns are due to genetic susceptibility, differences in baseline health status and co-morbidities, or a host of possible contributory factors such as SES and healthcare access, unfavourable environmental conditions, health perception, health seeking patterns and health literacy. Further research is required to understand the independent effects of these factors.

The 2009 influenza pandemic A(H1N1)pdm09, identified a major gap in global influenza surveillance capacity that compromised the assessment and monitoring of the pandemic. The lack of any established surveillance for severe disease in most countries, including developed countries, resulted in an absence of historical data to evaluate the severity of the pandemic in the context of previous seasons or to observe changes in the behaviour of the virus. One main objective of SARI surveillance is to measure the incidence and prevalence of SARI cases, including ICU admissions and deaths, in a timely manner. Of the 837 696 residents in ADHB and CMDHB areas, 1372 (164 per 100 000 population) were hospitalised with severe acute respiratory infections including 44 (5 per 100 000 population) ICU admissions and 4 (0.5 per 100 000 population) deaths in 2013. Influenza viruses were detected in 189 (23 per 100 000 population) tested SARI cases, 8 (1 per 100 000 population) in SARI ICU cases and none in SARI deaths. This SARI surveillance has generated timely disease incidence and severity data on the impact or burden of influenza that will help policy makers develop strategies for reducing the incidence and impact of influenza. On-going active (real-time) surveillance being conducted for SHIVERS will enhance the early detection of any impending pandemic or novel virus resulting in severe illness.

One of the strengths of the ILI sentinel surveillance system in New Zealand is the combination of disease surveillance with virus strain surveillance (virological identification). A definitive diagnosis of influenza requires laboratory confirmation, because diagnosis on the basis of clinical symptoms is not specific. In fact, sentinel surveillance is the only syndromic surveillance system that obtains good quality 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 ILI. During sentinel surveillance from May to October 2013, four virology laboratories tested 602 respiratory specimens for influenza viruses, of which 196 (32.6%) specimens were positive. The influenza detection rate varied among the different DHBs with a range from 18.0%–77.8%. Sampling techniques may contribute to low detection rates. Sampling of the respiratory tract for viral isolation should maximise the harvest of virally-infected columnar epithelial cells which would then improve the influenza detection rate [20].

The emergence and rapid global 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 were resistant to oseltamivir. By contrast, all influenza A(H1N1)pdm09 viruses tested in 2009, 2010, 2011 and 2013 were sensitive to oseltamivir. In 2012, two oseltamivir resistant influenza A(H1N1)pdm09 viruses were detected. Oseltamivir-resistant viruses pose challenges for the selection of antiviral medications for the treatment and chemoprophylaxis of influenza. It has become increasingly important to maintain a national antiviral monitoring programme in New Zealand to provide timely surveillance information, to assist clinicians choosing antiviral agents for their patients and public health officials making decisions on stockpiling and using antiviral agents during a pandemic or epidemic.
Timely surveillance information also encourages clinicians to test patients for influenza virus infection in order to select appropriate antiviral medications.

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

Because the impact of influenza on people and health systems can be reduced by annual immunisation, information about influenza vaccination coverage is particularly important in raising awareness of the disease among 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, has been key to reach the vaccination coverage to 67.4% for people aged 65 years and older in 2013.

Influenza vaccines are recommended for people at risk of developing complications following infection because of their age or because of underlying chronic conditions [21]. In 1997, New Zealand introduced a programme of free influenza vaccinations to all New Zealanders aged 65 years and older, and set a target of 75% coverage for the year 2000. In 1999, the free vaccination programme was extended to include those under 65 years with specified chronic medical conditions [9]. In 2010, the free vaccination was extended to all pregnant women [9]. In 2013, the free vaccination was extended to children aged less than five years with significant respiratory illness [22]. Quality coverage data is essential for evaluating the effectiveness of influenza vaccines. Therefore, it is crucial that influenza vaccination information from multiple sources is included on the national immunisation register.

In summary, we have described the national influenza surveillance data collected in 2013 in terms of community disease burden, circulating viral strains, hospitalisations, mortality, and immunisation coverage. The national sentinel GP-based surveillance recorded influenza activity at a low level in the community. SHIVERS hospital-based SARI surveillance and general practice-based ILI surveillance showed contrasting socio-demographic patterns. Higher rates of influenza-associated GP consultations occurred in school age children, pre-schoolers and adults, those of Asian ethnicity and those from least deprived socio-economic groups. Whereas, influenza-associated hospitalisations were more frequent in the very young, the elderly, Māori and Pacific Peoples and those from most deprived socio-economic groups. The highest influenza vaccination coverage since 1990 was recorded in 2013. This report demonstrates that an integrated virological and epidemiological surveillance system for influenza is essential for monitoring the disease burden, identifying circulating strains, guiding effective vaccination and planning for a potential pandemic.
REFERENCES


Appendix A. Phylogenetic analysis of HA gene sequence of influenza A(H1N1) viruses

Influenza A H1N1

Legend
- New Zealand 2013 viruses
- New Zealand 2012 viruses
- Vaccine strain 2013
Appendix B. Phylogenetic analysis of HA gene sequence of A(H3N2) viruses

Influenza A H3N2

Legend
- New Zealand 2013 viruses
- New Zealand 2012 viruses
- Vaccine strain 2013
- Vaccine strain 2012

[Phylogenetic tree diagram showing evolutionary relationships among influenza A H3N2 viruses from New Zealand 2013, New Zealand 2012, Vaccine strain 2013, and Vaccine strain 2012.]
Appendix C. Phylogenetic analysis of HA gene sequence of B/Victoria lineage viruses

Influenza surveillance in New Zealand 2013

Appendix

Influenza B/Victoria

Legend

- New Zealand 2013 viruses
- New Zealand 2012 viruses
- Vaccine strain 2012

Influenza B/Victoria
Appendix D. Phylogenetic analysis of HA gene sequence of B/Yamagata lineage viruses