

# ANNUAL REPORT OF THE NATIONAL INFLUENZA SURVEILLANCE SCHEME, 2008

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## Abstract

The 2008 influenza season was moderate overall, with fewer laboratory-confirmed cases and influenza-like illness (ILI) presentations than in 2007, which was the most severe influenza season since national reporting of influenza began in 2001. In 2008, the number of laboratory-confirmed notifications for influenza was 1.9 times higher than the 5-year mean. High notification rates were reflected in an increase in presentations with ILI to sentinel general practices and emergency departments. Notification rates were highest in the 0–4 year age group. Unusually, the season was predominantly due to influenza B, with 54% of notifications being influenza B and 43% being influenza A (3% type unknown). The rate of influenza B was higher among the younger age groups, compared with influenza A, which was more common in the older age groups. Of influenza viruses circulating during the 2008 season, A(H3) viruses were predominant and were antigenically similar to the 2008 A(H3) vaccine strain, while the majority of A(H1) strains showed significant drift away from the 2008 A(H1) vaccine strain. There were approximately equal proportions of viruses from the 2 influenza B lineages B/Yamagata and B/Victoria. *Commun Dis Intell* 2010;34(1):8–22.

Keywords: influenza, surveillance, vaccine, influenza-like illness, sentinel surveillance

## Introduction

Influenza or ‘the flu’ is a common, highly infectious respiratory viral disease. The virus spreads from person to person by airborne droplets of exhaled respiratory secretions, especially by coughing or sneezing.<sup>1</sup> Typical symptoms include sudden onset of fever, sore throat, runny nose, cough, fatigue, headache, and aches and pains.

Influenza causes annual epidemics of respiratory disease. Influenza epidemics usually occur during the winter months in temperate climates, causing an increase in hospitalisations for pneumonia, an exacerbation of chronic diseases and also contributing to increased mortality. Those most susceptible include the elderly and very young people, or peo-

ple of any age who have a higher risk of complications (e.g. pneumonia, heart failure) due to certain chronic medical conditions, e.g. heart, lung, kidney, liver, immune, or metabolic diseases. Most healthy children and adults only have minor symptoms.

Laboratory-confirmed influenza is a nationally notifiable disease in all states and territories and data are reported from each state or territory health department to the National Notifiable Diseases Surveillance System (NNDSS).

In temperate zones of Australia, the annual influenza season runs from May to October, with a peak in notifications around the middle of August. The severity of seasons varies from year to year. Australia experienced moderate to severe seasons in 2003 and 2007 but mild seasons in other years. In recent years, influenza A has been the predominant type circulating in Australia, most commonly the A(H3) subtype.

## Surveillance methods

The surveillance of influenza during 2008 was based on the following sources of data:

- notifications of laboratory-confirmed influenza required by legislation in all states and territories, and notified to the NNDSS;
- subtype and strain data of circulating influenza viruses provided by the World Health Organization (WHO) Collaborating Centre for Reference and Research on Influenza;
- consultation rates for influenza-like illness (ILI) identified by sentinel general practitioners;
- consultation rates for ILI identified by hospital emergency departments (EDs);
- testing rates for influenza by New South Wales sentinel laboratories;
- absenteeism data from a national employer; and
- mortality data from the New South Wales Registry of Births, Deaths and Marriages and Australian Bureau of Statistics (ABS).

\* Publication of this 2008 *Annual Report of the National Influenza Surveillance Scheme* has been delayed as a result of the response to the H1N1 2009 pandemic. The 2009 annual report will be published in *CDI* later this year.

## National Notifiable Diseases Surveillance System

In 2008, laboratory-confirmed influenza was a notifiable disease under state and territory legislation in all jurisdictions. Laboratory notifications were sent to NNDSS for national collation. In this report, data were analysed by the date of diagnosis; the best substitute for the date of onset. The date of diagnosis was set as the date of onset, or where not supplied, the earliest of specimen collection, notification or notification received date. Age, sex, method of laboratory diagnosis and postcode or locality of patient residence were included in NNDSS notifications. Maps were produced using ArcGIS software.

## Sentinel general practitioner surveillance

Sentinel general practitioner surveillance schemes for influenza monitor clinical consultations for ILI. In Australia, there are three such schemes: the Australian Sentinel Practices Research Network (ASPREN), which collects data at a national level from approximately 80 general practitioners from 7 states and territories (Australian Capital Territory, New South Wales, Queensland, South Australia, Tasmania, Victoria, and Western Australia), the Victorian Infectious Diseases Reference Laboratory General Practice Sentinel Surveillance Program (VIDRL GPSS), and the Northern Territory Tropical Influenza Surveillance Scheme. ASPREN and the Northern Territory Tropical Influenza Surveillance Scheme report ILI rates throughout the year, while VIDRL GPSS reported from late April to early November in 2008. The national case definition of ILI is: presentation with fever, cough and fatigue. All sentinel surveillance schemes, including ASPREN, used the national case definition for ILI in 2008.

## Emergency department surveillance

Rates for influenza-like illness presentation were collected from 29 EDs across New South Wales and up to 9 EDs in Perth, Western Australia. Data were provided to the Surveillance Branch, Office of Health Protection within the Australian Government Department of Health and Ageing (DoHA) on a weekly basis, through the New South Wales Influenza Surveillance Report, and the West Australian Emergency Department Sentinel Surveillance News Report.

## Laboratory surveillance

### *WHO Collaborating Centre for Reference and Research on Influenza*

The WHO Collaborating Centres for Reference and Research on Influenza located in Australia, Japan, the United Kingdom and the United States

of America (USA), are responsible for analysing influenza viruses collected through an international surveillance network involving 122 national influenza centres in 94 countries. The Melbourne centre analyses viruses received from Australia and from laboratories throughout Oceania, the Asian region and beyond. All virus isolates are analysed antigenically and a geographically and temporally representative number of viruses, together with any strains demonstrating uncharacteristic reactions during antigenic characterisation, are further analysed by genetic sequencing of the viral haemagglutinin gene and the neuraminidase gene. Together with serological and epidemiological data, these form the basis from which WHO makes recommendations in February (for the Northern Hemisphere) and in September (for the Southern Hemisphere) for the vaccine formulation to be used in the following winter. WHO vaccine formulation recommendations are made in the context of strains that are antigenically 'like' laboratory reference strains that are named according to a standard nomenclature for influenza viruses. For human isolates this nomenclature is based on type, the place of isolation, sequential number and year of isolation and for influenza A, the subtype of the HA and NA may also be included in brackets after the designation. An example of a human isolate is A/Sydney/5/97(H3N2), an influenza A(H3N2) virus that was the 5th sequential influenza A isolated in Sydney in the year 1997.

The WHO recommendations<sup>2</sup> are then translated into actual virus strains acceptable to regulatory authorities and vaccine manufacturers, by national and regional committees (such as the Australian Influenza Vaccine Committee).

### *Sentinel laboratory network*

The New South Wales sentinel laboratory network collects influenza virology testing data from 6 major public laboratories, and influenza serology testing data from three. The number of laboratory requests for influenza laboratory tests was obtained weekly from New South Wales Influenza Surveillance Reports.

## Absenteeism surveillance

A major nationwide employer, provided weekly absenteeism data in 2008. Absenteeism, defined as an absence recorded as 'sick-leave' for three or more consecutive days, was presented as a rate per 100 employees per week, on an average of 33,290 employees per reporting week.

## Mortality

Death certificate data from the New South Wales Registry of Births, Deaths and Marriages provided an estimate of the number of deaths from pneu-

monia and influenza in New South Wales and compared the rate per 1,000 deaths with predicted seasonal mean plus a 95% confidence interval. These were obtained weekly from the New South Wales Influenza Surveillance Report.<sup>3</sup>

Deaths data compiled by the ABS from information provided by the state and territory Registrars of Births, Deaths and Marriages, and coded using the 10th revision of the *International Classification of Diseases and Related Health Problems* (ICD-10) were used to estimate levels of influenza deaths. In this report, deaths for 2008 with an underlying cause of influenza and pneumonia (ICD-10 J10–J18) are presented.<sup>4</sup>

### Morbidity data

There was no effective measure of morbidity of disease readily available during the 2008 influenza season. Instead, morbidity was assessed through a number of indicators including:

- absenteeism surveillance;
- Paediatric Intensive Care Unit admissions to intensive care units (ICUs) and deaths data collected by the Australian Paediatrics Surveillance Unit (APSU);
- ED presentations for ILI in New South Wales and Western Australia; and
- ILI presentations to GP surveillance networks.

Hospital admissions for influenza and pneumonia were not available during the 2008 season.

## Results

The 2008 influenza season began in mid-July, although there was a very gradual increase in notifications above non-seasonal levels from much earlier in the year. Between February and the start of the season in July, NNDSS notifications were above the 5-year mean. However all sentinel data sources were tracking below or similar to trends seen in previous years.

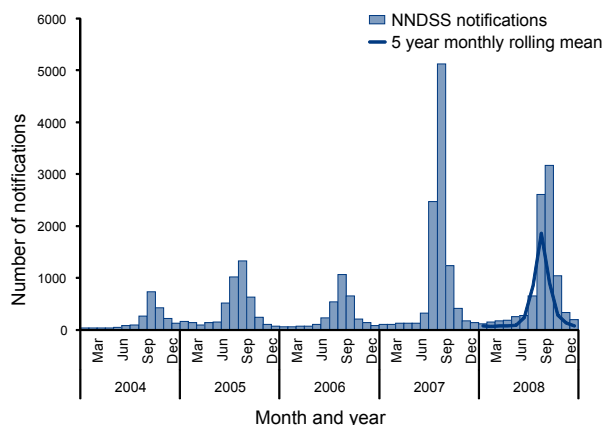
### Laboratory confirmed cases

The first increases in notifications of laboratory-confirmed influenza in the 2008 season were registered in early July (week 28) when 105 cases were diagnosed. Notifications peaked in early September (week 36) and were almost back to inter-seasonal levels by the middle of November (week 64) (Figure 1). The total number of notifications for the year was 9,137, which was 1.9 times the 5-year mean.

### Geographic spread

In 2008, 41% of laboratory-confirmed influenza notifications occurred in Queensland, 20% in New

**Figure 1: Laboratory-confirmed influenza notifications, 2004 to 2008, Australia, by month and year of diagnosis**



South Wales, 14% in Victoria, 11% in Western Australia, 5% in South Australia, 4% in Tasmania, 3% in the Australian Capital Territory and 2% in the Northern Territory (Figure 2). The number of notifications peaked at a similar time in most jurisdictions, (in weeks 35–36; weeks ending 29 August and 5 September respectively).

Laboratory-confirmed influenza notification rates for 2008 varied across the country, ranging from 24 cases per 100,000 population in Victoria to 91 cases per 100,000 population in the Northern Territory. The crude annual notification rate of influenza infection for Australia was 43 cases per 100,000 population (Table 1).

The Map shows rates of laboratory-confirmed influenza in 2008 by Statistical Division\* of residence. The highest rates of influenza occurred in Statistical Divisions that encompassed the central region of the Northern Territory and south-west region of Queensland.

### Age-sex profile

Age-specific notification rates for laboratory-confirmed influenza reported to the NNDSS in 2008 are shown in Figure 3. The highest notification rates were seen in children aged 0–4 years, which were around 3.4 times higher than for other age groups (98 per 100,000 population compared with a total rate of 39 per 100,000 population for all notifi-

\* A Statistical Division (SD) is an Australian Standard Geographic Classification defined area, which represents a relatively homogeneous region characterised by identifiable social and economic links within the region. They consist of one or more Statistical Subdivisions and cover, in aggregate, the whole of Australia without gaps or overlaps. They do not cross state or territory boundaries and are the largest statistical building blocks of states and territories. (Source: ABS)

Map: Notification rates of laboratory-confirmed influenza, Australia, 2007, by Statistical Division of residence

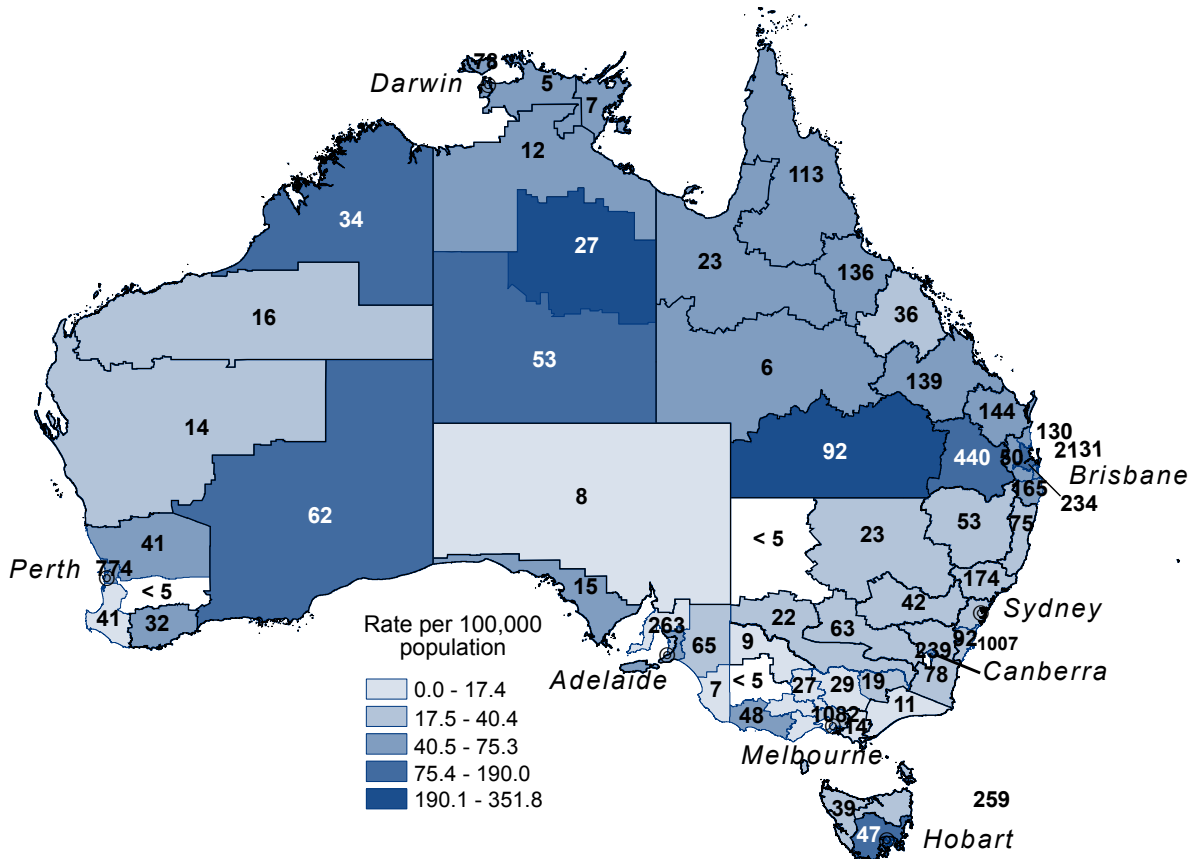
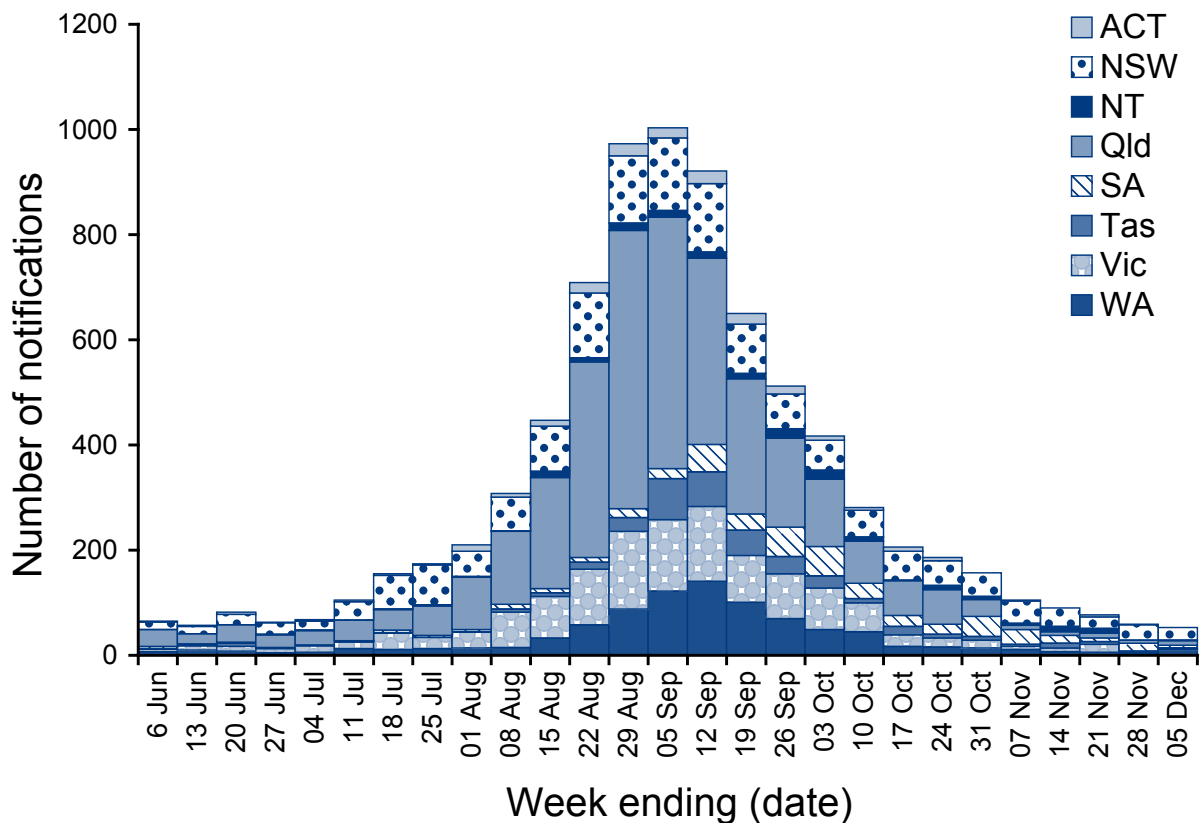


Figure 2. Laboratory-confirmed influenza notifications, June to December 2008, by state or territory and week of diagnosis



**Table 1: Notifications and rates of laboratory-confirmed influenza, 2008, by state or territory and sex**

State or territory	Total notifications	% of total	Notification rate (per 100,000 population)	Notifications*		Notification rate* (per 100,000 population)	
				Male	Female	Male	Female
ACT	244	3	71	99	145	58	83
NSW	1,814	20	26	863	944	25	27
NT	199	2	91	97	102	85	96
Qld	3703	41	86	1,718	1,985	80	92
SA	473	5	30	238	235	30	29
Tas	388	4	78	180	207	73	82
Vic	1,300	14	24	628	670	24	25
WA	1,016	11	47	521	495	47	46
Aus	9,137	100	43	4,344	4,783	41	44

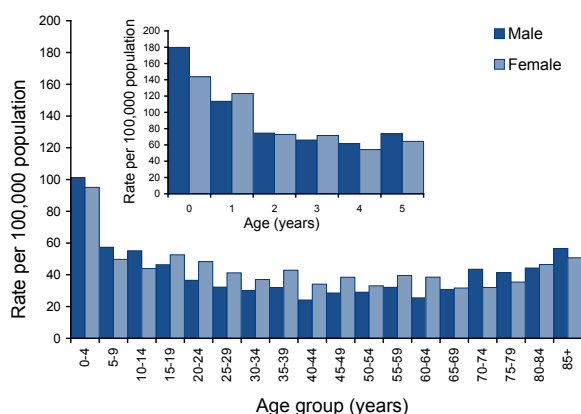
\* Ten notifications of unknown sex were excluded from analysis.

cations). People aged 65 years or over are the target for influenza vaccination as they are at an increased risk of complications from influenza. Notification rates for people in this age group were 40 per 100,000 population for males and 38 per 100,000 population for females. This compares with 2007 where influenza notification rates in the 65 years or over age group were 43 per 100,000 population for both males and females.

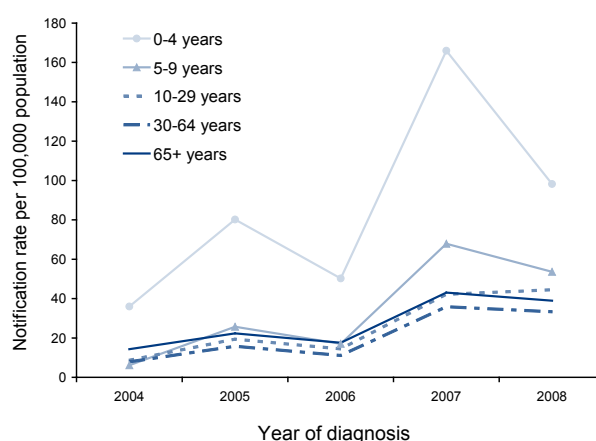
Total notifications in 2008 were approximately equal for both males (47.5%) and females (52.5%). Notifications were slightly higher in females than in males for persons aged between 15 and 69 years, and 80–84 years. For children and the elderly, notifications for males exceeded those for females. The ratio of males to females in the population was approximately 1:1 for age groups up to 69 years. The ratio of males

to females aged 70 years or older was 0.8:1. Although the number of notifications was higher in females aged over 70 years, there are fewer males in the population aged over 70 years, which accounts for the higher rate compared with females in this age group.

Figure 4 shows the notifications rates for key age groups for the years 2004 to 2008. Overall, notification rates were higher in 2008 for all age groups compared with the previous 4 years, with the exception of the 0–4 and 5–9 year age groups in 2007. Notification rates decreased dramatically in the 0–4 year age group, from 166 per 100,000 population in 2007 to 98 per 100,000 population in 2008. A decrease was also observed in the 5–9 year age group (68 notifications per 100,000 in 2007 to 54 notifications per 100,000 in 2008). Rates in all other age groups remained similar to those in 2007.

**Figure 3: Notification rate of laboratory-confirmed influenza, Australia, 2008, by age group (insert – age) and sex\***

\* Notifications of unknown age (n = 4) or sex (n=10) were excluded from analysis.

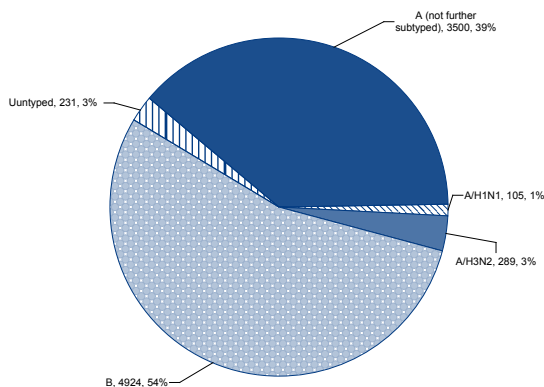
**Figure 4: Notification rate of laboratory-confirmed influenza reported to the National Notifiable Diseases Surveillance System, Australia, 2003 to 2008, by age group\***

\* Notifications of unknown age (n=4) were excluded from analysis

## Virus type and subtype

Analysis of NNDSS influenza typing data indicated a change in the proportion of circulating influenza subtypes from that seen in previous years. Of the 9,137 influenza cases notified to NNDSS in 2008, 98% included typing data. Influenza B was the predominant circulating type comprising 54% of isolates typed compared with 43% that were type A (type was unknown for 3% of influenza cases) (Figure 5). In 2007, 86% of typed isolates were influenza A and 9% were influenza B.

**Figure 5: Number of influenza notifications reported to the National Notifiable Diseases Surveillance System, Australia, 2008, by type\***



\* Notifications of influenza type 'A&B' (n=86) and 'C' (n=2) were excluded from this analysis.

Prior to the influenza season, notifications were predominantly influenza A. However as the season commenced in mid-July, influenza B became more predominant (Figure 6). A predominantly influenza B season has not been recorded in Australia since influenza became nationally notifiable. Since 2004, the proportion of influenza B was 20%, 23%, and 26% in 2004–2006 (respectively), 9% in 2007 and 54% in 2008 (Figure 7).

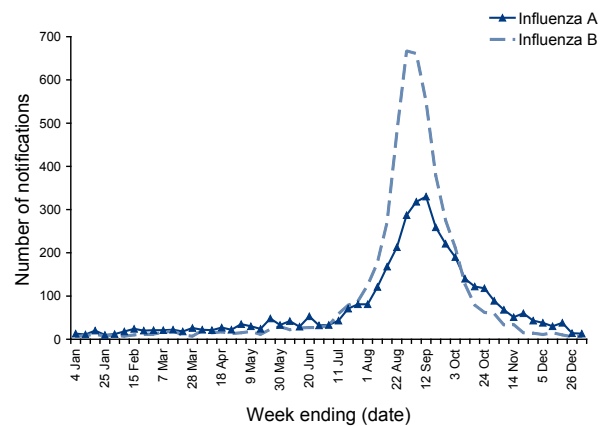
A breakdown of notifications by type and age indicated that compared with influenza A, the rate of influenza B was higher in younger age groups and lower in older age groups (Figure 8).

## Virology

### Antigenic characterisation

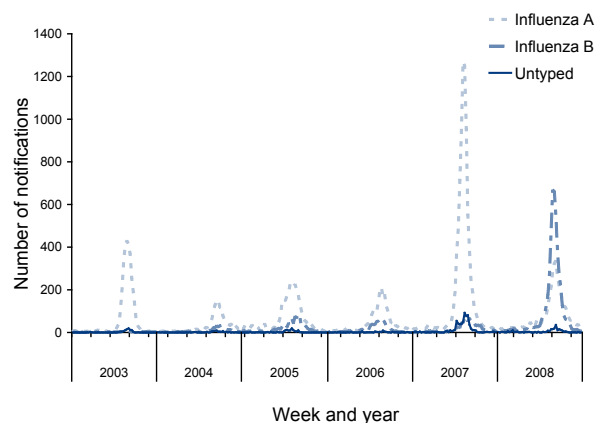
The WHO Collaborating Centre for Reference and Research on Influenza received 1,512 isolates or clinical specimens from Australian laboratories in 2008 that yielded 1,224 viable influenza viruses. All of the viable 2008 viruses were analysed antigenically

**Figure 6: Number of influenza notifications reported to the National Notifiable Diseases Surveillance System, Australia, 2008, by type and week of diagnosis\***



\* 319 notifications of influenza were excluded from this analysis: type 'A&B' (n=86), 'C' (n=2) and 'Untyped' (n=231)

**Figure 7: Number of influenza notifications reported to the National Notifiable Diseases Surveillance System, Australia, 2004 to 2008, by type and week of diagnosis**

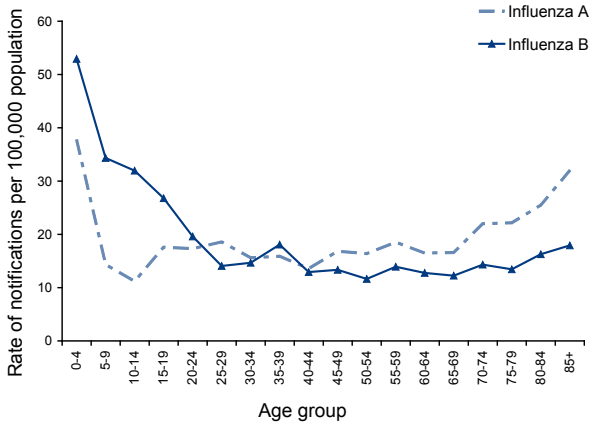


cally using the haemagglutination inhibition (HI) assay, which identified 266 (21.7%) as A(H3N2) strains, 144 (11.8%) as A(H1N1) strains and 814 (66.5%) as influenza B strains. This was by far the highest proportion of influenza B viruses seen in the last 10 years with the previous highest proportion occurring in 2006 with 38.5% of the viruses typed as influenza B. Over this period, A(H3N2) viruses have predominated in 8 years, A(H1) in 1 year and now influenza B in 1 year (2008). The 2008 Australian A(H3N2) viruses were mostly antigenically similar to the vaccine strain A/Brisbane/10/2007 by HI assay, with few A/Wisconsin/67/2005 viruses detected. Antigenic analysis of the Australian 2008 A(H1) strains, showed that there was significant drift away from the 2008 Australian vaccine strain A/Solomon

Islands/3/2006 to the A/Brisbane/59/2007-like viruses. A large number of influenza B viruses were isolated in 2008, with almost equal proportions of

the 2 lineages being present; 50.7% (413) of viruses of the B/Victoria-lineage (B/Victoria/2/87) and 49.3% (401) of viruses of the B/Yamagata-lineage (B/Yamagata/16/88). The B/Victoria-lineage viruses reacted well with ferret sera raised to cell cultured recent B/Victoria-lineage like-viruses such as B/Victoria/304/2006 while the B/Yamagata-like viruses reacted well with antisera raised against the 2008 Australian vaccine strain B/Florida/4/2006 (Table 2).

**Figure 8: Rate of influenza notifications reported to the National Notifiable Diseases Surveillance System, Australia, 2008, by type and age group\***

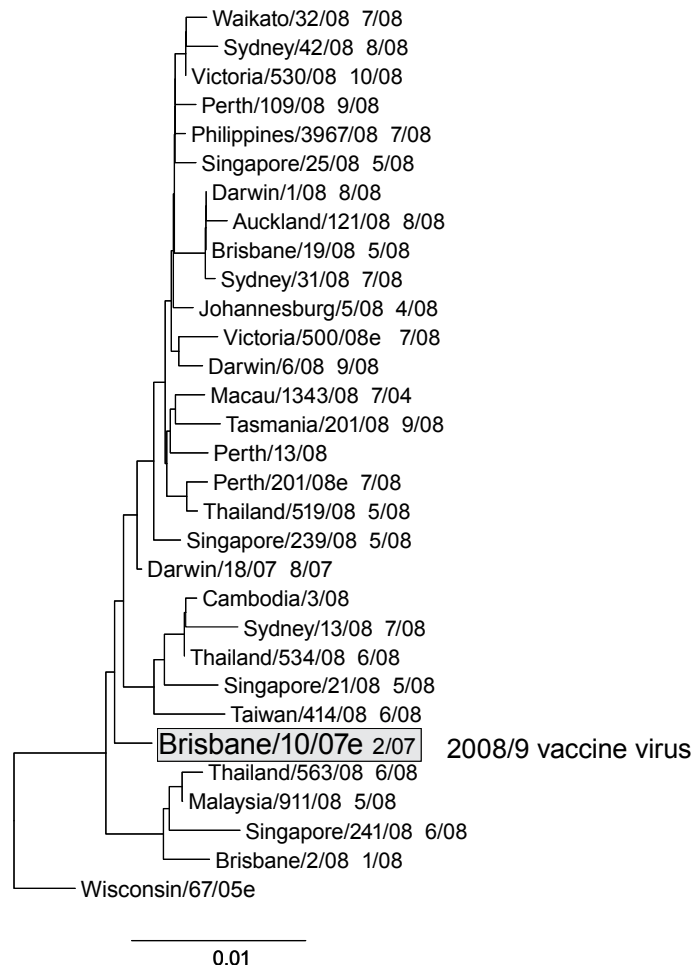


\* Notifications of 'unknown' age (n=4) and influenza types: 'A&B' (n=86), 'C' (n=2) and 'unknown' (n=231) were excluded from analysis.

Sequence analysis of the variable (HA1) region of the haemagglutinin (HA) gene was undertaken for 115 Australian 2008 viruses (33 A(H1), 29 A(H3) and 53 B) and for 111 neuraminidase genes, (67 H1, 16 H3, 28 B). The phylogenetic analysis of the 2008 (H3) virus HA1 sequences showed that most Australian A(H3) viruses were closely related to A/ Brisbane/10/2007-like viruses with the majority having a conserved amino acid change at position K173Q, although some viruses also fell into other closely related sub-groups (Figure 9).

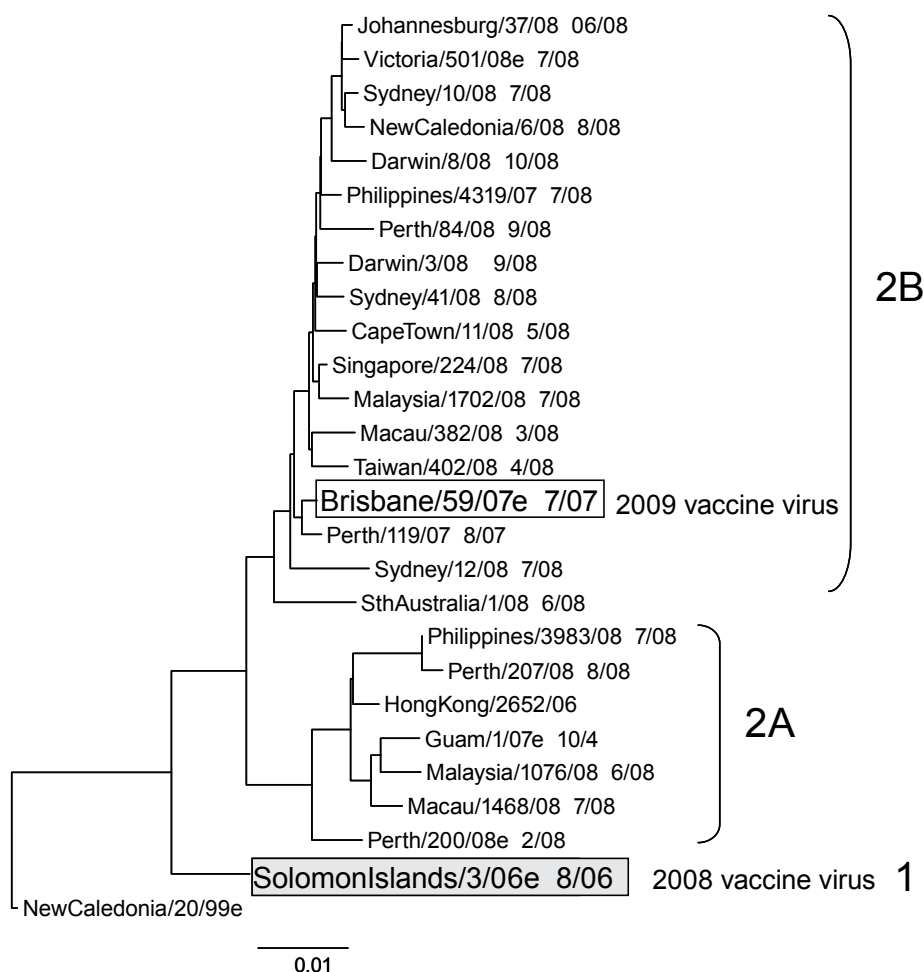
When the HA1 genes from A(H1) viruses isolated in Australia in 2008 were compared phylogenetically, they fell almost exclusively into the group of viruses represented by A/Brisbane/59/2007 (Figure 10) and now

**Figure 9: Evolutionary relationships between influenza A (H3) haemagglutinins (HA1 region)**



**Table 2: Antigenic comparisons of the influenza B viruses, by the haemagglutination-inhibition assay**

Viruses	Lineage	Ferret antisera Reciprocal haemagglutination-inhibition titre			
		B/Yamagata-lineage		B/Victoria-lineage	
		B/Florida/4/ 2006	B/Brisbane/3/ 2007	B/Malaysia/ 2506/2004	B/Brisbane/60/2008
<b>Reference viruses</b>					
B/Florida/4/2006	Yamagata	640	640	160	20
B/Brisbane/3/2007	Yamagata	320	640	80	20
B/Malaysia/2506/2004	Victoria	<20	<20	1280	160
B/Brisbane/60/2008	Victoria	<20	<20	640	320
<b>Test viruses</b>					
B/Victoria/227/2008	Victoria	<20	<20	80	320
B/Victoria/523/2008	Yamagata	160	160	<20	<20
B/Sydney/529/2008	Yamagata	160	320	20	<20
B/Perth/213/2008	Victoria	<20	<20	20	80
B/South Australia/180/2008	Victoria	–	–	20	160
B/South Australia/88/2008	Yamagata	320	1280	40	<20
B/Brisbane/134/2008	Yamagata	160	320	–	–
B/Canberra/100/2008	Victoria	–	–	40	160
B/Tasmania/205/2008	Victoria	–	–	40	320

**Figure 10: Evolutionary relationships between influenza A (H1) haemagglutinins (HAI region)**



distinguished as clade 2B viruses. Few viruses fell into other groups represented by A/Hong Kong/2652/2006 (clade 2C viruses), A/Solomon Islands/3/2006 (clade 2A viruses) or A/Brisbane/193/2004 (clade 1 viruses) (Figure 10).

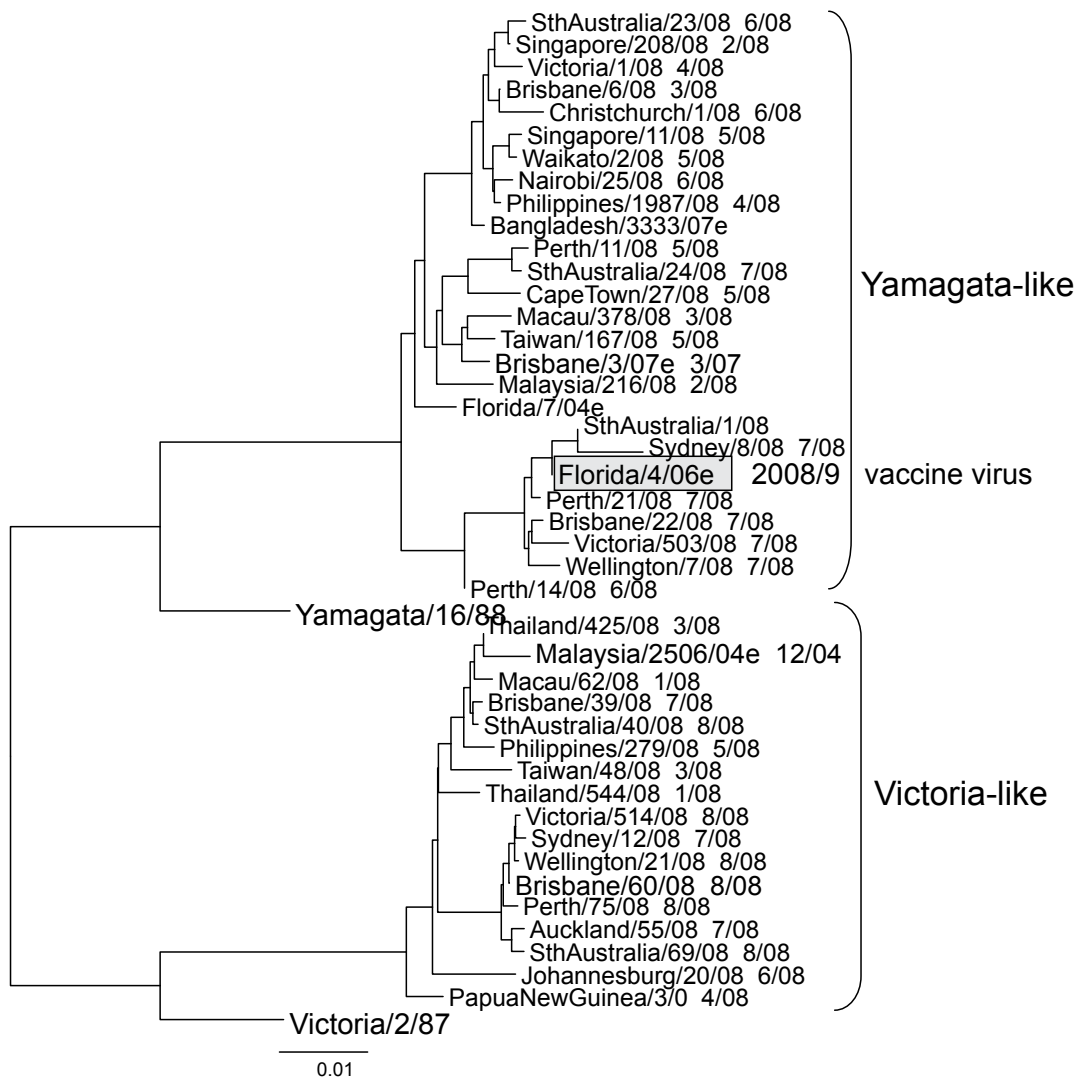
The Australian 2008 influenza B viruses phylogenetically grouped into their respective lineages, either the B/Victoria or B/Yamagata lineage, with the B/Victoria lineage viruses showing a number of amino acid change from the reference/vaccine strain B/Malaysia/2506/2004 and grouping with the reference virus B/Brisbane/60/2008. The B/Yamagata-like viruses from Australia fell into 3 genetically distinct subgroups represented by B/Florida/4/2006 (Group 1) B/Brisbane/3/2007 (Group 2) and B/Bangladesh/3333/2007 (Group 3) in similar proportions (Figure 11).

**Influenza-like illness consultations from sentinel general practitioner surveillance systems**

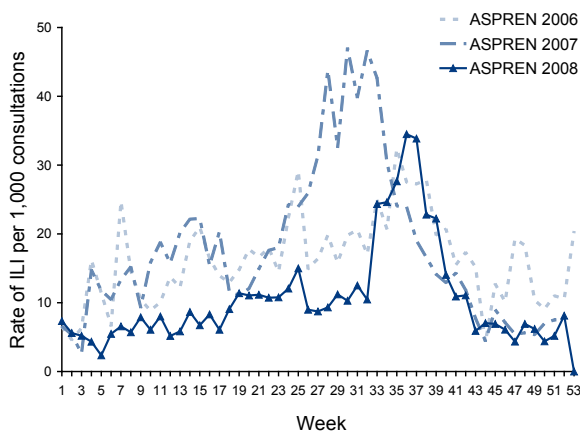
Data from the ASPREN Sentinel GP Surveillance System showed that for 2008 there were 4,213 notifications for ILI. An average of 80 doctors reported to ASPREN each week (range 69–85), with an average of 7,453 consultations per week (range 3,573–8,464).

Overall, the rate of consultations for ILI was lower in 2008 than in 2006 and 2007 (Figure 12). Consultation rates were higher in May and June (weeks 18–25) compared with earlier in the year, decreased slightly in July and early August (weeks 26–32), and then increased sharply in mid-August (week 33). The rate peaked in early September (weeks 36–37), consistent with NNDSS notifications, at 34 ILI cases per 1,000 consultations, and returned to baseline levels by the end of October (week 43). Although ILI and NNDSS notifications

**Figure 11: Evolutionary relationships between influenza B haemagglutinins (HAI region)**



**Figure 12: Consultation rates for influenza-like illness, ASPREN, 2006 to 2008, by week**



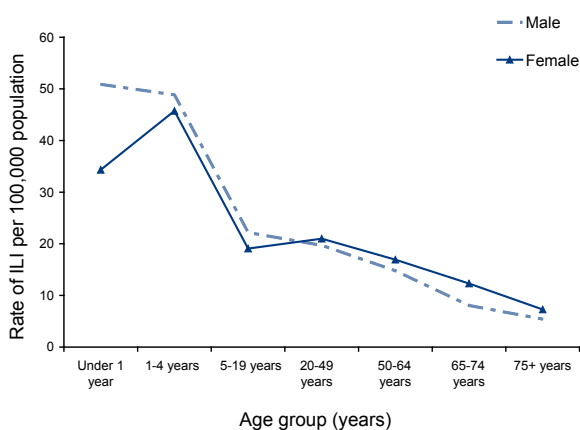
follow the same trends, during the 2008 season ILI notifications were more timely than NNDSS notifications and were used as an early alert and predictor of influenza in the community.

At a state level, ASPREN GPs in Western Australia and New South Wales had higher rates of ILI notifications compared with all the other states and territories.

The VIDRL GPSS followed a similar trend to ASPREN nationally, with more ILI presentations in 2008 than in 2006 but less than 2007, and similar timing for the start and end of the season. This trend was also seen in data from the Northern Territory Tropical Influenza Surveillance System.

A breakdown of ASPREN data by age and sex indicates that the highest rate of ILI presentations were recorded in children under 1 year of age and aged 1–4 years (Figure 13). This is consistent with NNDSS influenza notifications. Male children under 1 year of age had a higher number of presen-

**Figure 13: Consultation rates for influenza-like illness, ASPREN, 2008, by age group and sex**



tations compared with female children of the same age. The rate of ILI presentations were similar for both males and females in all other age groups.

ASPREN data are not completely representative of the Australian population. In 2008, there were no GPs from the Northern Territory contributing data to ASPREN, and several states and territories had small numbers of GPs reporting. It is also difficult to compare across different years, as representativeness varies over time, due to changes in the number of reporting doctors.

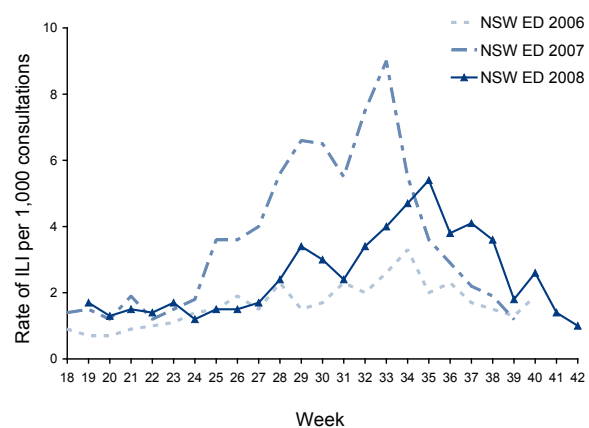
No sampling of ILI patients for laboratory influenza testing occurred at a national level through the ASPREN scheme, although some state systems conducted random testing. Therefore the proportion of ILI presentations tested for influenza, and of those, the proportion positive for influenza could not be determined.

#### **Influenza-like illness – emergency department surveillance (New South Wales and Western Australia only)**

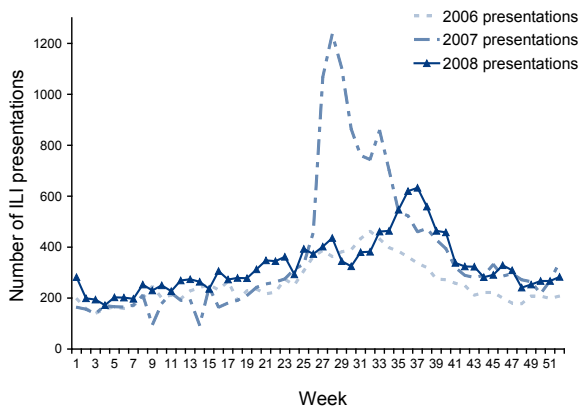
Presentations to New South Wales EDs for ILI began to rise in early July (week 27) and peaked at 5 presentations per 1,000 consultations in late August (week 35) (Figure 14). The increase in presentation rates reflected the rise in laboratory confirmed notifications of influenza to NNDSS. Presentation rates in 2008 were higher than in 2006 but did not exceed rates during the peak of the 2007 influenza season.

Presentations to emergency departments in Western Australia for ILI rose gradually from the beginning of the year, following trends seen in 2006 and 2007 (Figure 15). The number of presentations peaked in the week ending 14 September (week 37), which

**Figure 14: Rate of influenza-like illness consultations from hospital emergency departments, New South Wales, April to September 2006 to 2008, by week of report**



**Figure 15. Rate of influenza-like illness consultations from hospital emergency departments, Western Australia, January to December, 2006 to 2008, by week of report**



was consistent with the peak of Western Australia influenza notifications to NNDSS. ED presentations for ILI in 2008 were lower than in 2007.

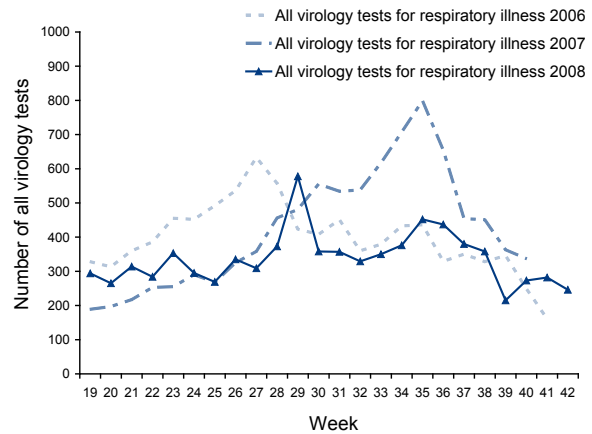
Sentinel ED surveillance data were timely, and a useful indicator of illness severity, but were only available from New South Wales and Western Australia. ED surveillance systems operate in other jurisdictions but these do not routinely report data to DoHA.

**Laboratory surveillance**

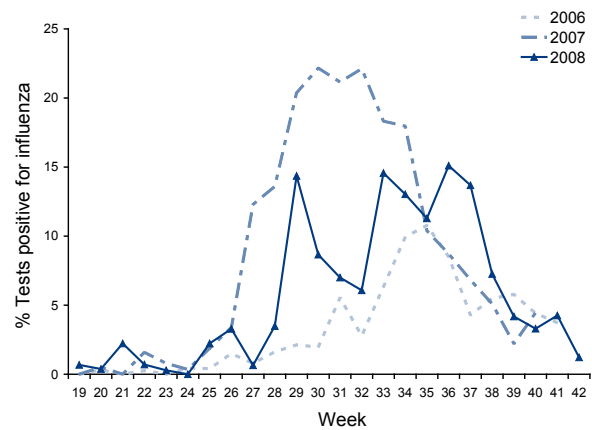
Data from New South Wales sentinel laboratory networks showed that the number of laboratory virology tests for respiratory illness (direct immunofluorescence, nucleic acid tests and viral culture) increased rapidly from baseline levels prior to early July 2008 (approximately 300 per week), to a peak of 578 tests per week in mid-July. This peak resulted from laboratory testing of influenza-like illness outbreaks in World Youth Day pilgrim groups. A second increase in testing during late August and early September were associated with the influenza season. The peak number of tests in 2008 did not exceed peaks in 2006 and 2007 (Figure 16).

The percentage of virology specimens testing positive peaked at approximately 15% in weeks 29, 33 and 36 (weeks ending 18 July, 15 August and 5 September respectively) (Figure 17). The peak in week 29 was again associated with World Youth Day pilgrims, and confirmed that these were influenza outbreaks. The peaks in weeks 33 and 36 were associated with the influenza season, and confirm the increase in the number of seasonal influenza cases. As the number of tests did not increase greatly during these weeks, it also indicates that increasing numbers were not an artefact of increased testing.

**Figure 16: Total virology specimens tested and number positive for influenza, New South Wales, May to October 2006 to 2008**



**Figure 17: Percentage of virology specimens testing positive for influenza, New South Wales, May to October 2006 to 2008**

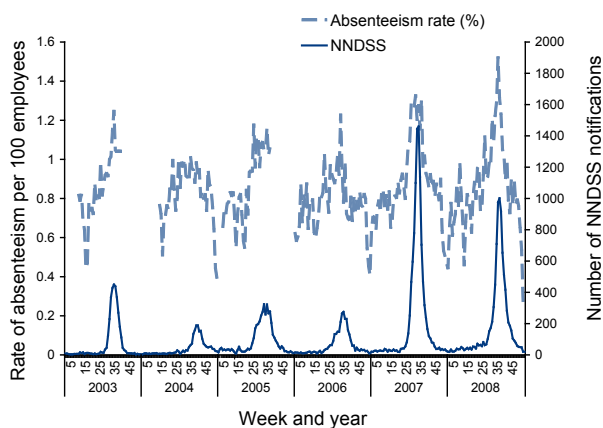


**Absenteeism surveillance**

Absenteeism surveillance can provide an early warning system of the severity of the influenza season as rates generally begin to increase several weeks before laboratory-confirmed notifications start increasing. Absenteeism rates are likely to be elevated by public health messages to stay home if unwell.

Absenteeism during the 2008 influenza season peaked in the week ending 3 September, which was consistent with the peak in laboratory-confirmed influenza notifications to NNDSS. Although laboratory-confirmed influenza notifications were lower in 2008 compared with 2007, absenteeism peaked higher in 2008, at 1.5% of employees (Figure 18).

**Figure 18: National absenteeism (more than 3 consecutive days) rates and National Notifiable Diseases Surveillance System influenza notifications, 2003 to 2008, by week of report**



### Morbidity

#### *Australian Paediatric Surveillance Unit surveillance*

APSU reported that between 1 July and 17 October 2008, there were 43 cases of children aged 15 years or younger admitted to ICUs in Australia, following complications due to influenza infection. Of the admissions, 29 were influenza B, 12 were influenza A and two were unknown. Only 6 children were reported to have been vaccinated for influenza (27 reportedly unvaccinated; 10 unknown). At the

time of admission, 12 cases were under 1 year of age, 14 were aged 1–4 years, and 15 were aged 5–15 years (2 ages unknown). The ages at admission ranged from 5 weeks to 14 years, with a median age of 3 years.

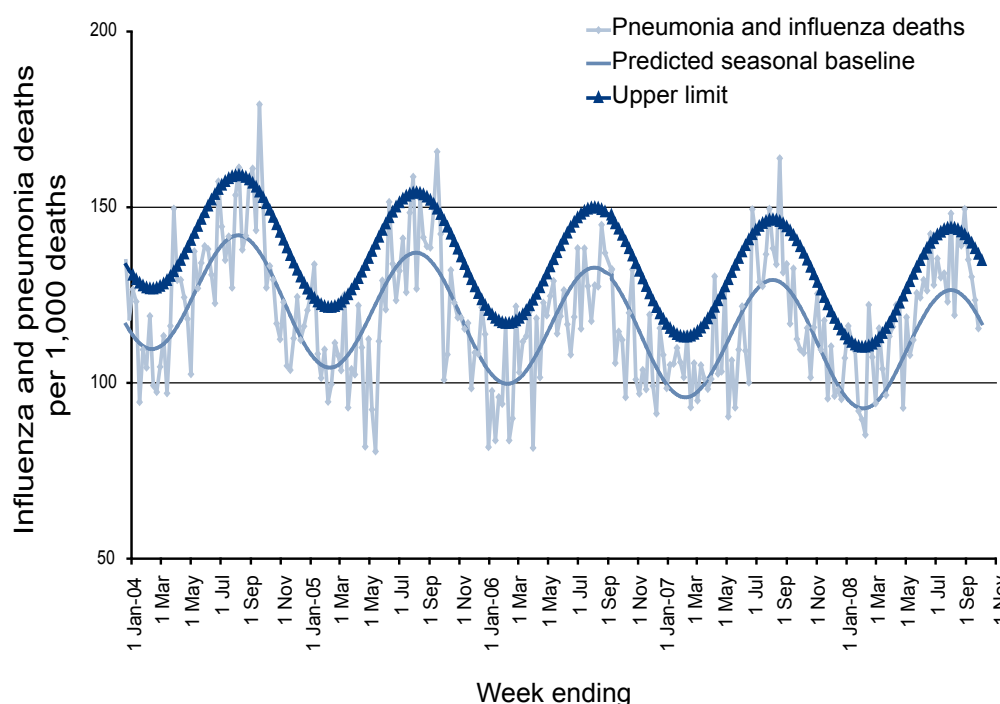
### Mortality

Mortality from a primary influenza infection is rare and most of the deaths attributed to influenza occur from complications including pneumonia, obstructive airways disease and sudden cardiac deaths. These occur predominantly in identified risk groups such as those over 65 years or under 6 months of age; or those with chronic medical conditions.

#### *Deaths from pneumonia and influenza – New South Wales*

Mortality rates from influenza in New South Wales reported by the Registry of Births, Deaths and Marriages showed that rates of deaths from influenza and pneumonia peaked in early September at approximately 150 per 1,000 deaths (Figure 19). The combined pneumonia and influenza death rates were equal to or higher than the predicted seasonal baseline for the majority of the season. Death rates were equal to or higher than the upper 95% confidence interval of the predicted seasonal baseline for 4 weeks during the season.

**Figure 19: Observed and predicted rate of influenza and pneumonia deaths as per New South Wales registered death certificates, 1 January 2004 to 3 October 2008**



*Australian Bureau of Statistics death data<sup>†</sup>*

Influenza and pneumonia (ICD-10 codes J10–J18<sup>†</sup>) were noted as the underlying cause of death for 1,554 persons in 2008 (1.2% of all deaths). The rate of influenza and pneumonia deaths was 7 per 100,000 deaths. In 2008, there were 932 female and 622 male deaths noted as influenza and pneumonia being the underlying cause of death. The standardised death rate was higher in females, with 9 per 100,000 deaths, compared with males at 6 per 100,000 deaths.

**International trends in influenza**

Between February and September 2008, influenza activity reported by WHO in Africa, the Americas, Asia, Europe, Australia and New Zealand, was in general mild.

In the Northern Hemisphere, influenza activity following the 2007/08 influenza season began to decline in March in Europe and in April in both Asia and North America. It was reported that A(H1) predominated in many countries in Asia and Eastern Europe, while A(H3) predominated in the USA. Influenza B viruses co-circulated and outbreaks were reported in some countries.

In the Southern Hemisphere, influenza activity began in March, increasing in April, in South America, while in Africa activity started in May, increasing in July. Overall activity declined in August, except for Australia, Brazil and New Zealand. In South America, A(H1) and B viruses co-circulated, in Africa A(H1) predominated, and in Australia and New Zealand A(H3) and B viruses co-circulated.

Overall, influenza activity in New Zealand in 2008 was described as moderate compared with other influenza seasons between 1997 and 2007. In 2008, consultation rates for ILI in New Zealand started to increase at the start of May, and remained at a high level between the start of July (week 27) to the start of September (week 36). During this time there were 2 peaks, one in the middle of July (week 29) and another in the middle of August (week 33). ILI consultation rates returned to baseline levels by the middle of September (week 37). There were a total

of 474 hospitalisations in 2008, which was more than in 2007 (347 people), and approximately equal to hospitalisations in 2006 (464 people).

The highest morbidity rates occurred in children aged under one year (120.1 per 100,000 population), followed by children aged 1–4 years (28.8 per 100,000 population) and adults aged over 65 years (15.5 per 100,000 population). These rates are consistent with morbidity seen in Australia during the 2008 season.

Of typed and subtyped viruses in 2008, the majority of viruses were characterised as influenza B (58.3%), with influenza A(H3) representing 41% and influenza A(H1) representing 0.8%.

Interestingly, influenza A(H3) was predominant at the start of the season (during June and July) however during August and September, influenza B was the most widely circulating strain in New Zealand. Prior to the start of the season, influenza B/Yamagata viruses (B/Florida/4/2006-like) predominated, however B/Victoria viruses (B/Malaysia/2506/2004) became more prevalent during the second half of the season and were predominant for the season overall (B/Yamagata 13.6%; B/Victoria 44.6% – of all influenza viruses). Since 1990, there have been 5 influenza seasons in New Zealand that have been predominantly type B: 1991 (92.3%), 1995 (68.8%), 1997 (53.5%), 2005 (87.0%), and 2008 (58.3%).

The full report on the 2008 influenza season in New Zealand is available on their web site.<sup>6</sup>

Outbreaks of A(H5N1) highly pathogenic avian influenza (HPAI) in poultry and wild birds still occurred in many parts of the world in 2008, with the continued exceptions of the Americas and Oceania. According to the official WHO figures, 44 H5N1 human infections occurred in 6 countries during 2008 resulting in 33 deaths. This was lower than cases and deaths in the previous 3 years (2005, 2006 and 2007). No highly pathogenic H5N1 infections were detected in humans or in birds in Australia in 2008. Details of avian influenza cases and deaths is available on the WHO avian influenza web site.<sup>7</sup>

**Discussion**

The severity and impact of influenza are difficult to measure due to the nature of the illness and limitations of surveillance systems. Influenza surveillance in Australia relies on a myriad of data sources and systems, varying in their ability to detect true cases of influenza. Ideally, the number of laboratory-confirmed notifications would include all cases, rather than just those that have been tested, and sentinel GP and ED surveillance systems would indicate the burden of disease on health systems

† The International Classification of Diseases (ICD) is the international standard diagnostic classification, and is used to classify diseases and other health problems recorded on many types of health and vital records, including death certificates and health records. ICD-10 (ICD version 10) codes J00–J99 relate to diseases of the respiratory system, and J10–J18 are specific for influenza and/or pneumonia.<sup>5</sup>

and the community. Hospitalisation and death data could also be improved to allow for true indicators of severity, morbidity and mortality related to influenza infection. It is possible that notifications in 2008 may have been affected by heightened media attention following the severity of the 2007 season, and it is not currently possible to measure the extent of this impact (if any). Influenza notifications following the 2007 season did not return to the same background levels as in previous years, and as such, notifications at the start of 2008 were slightly elevated in comparison. As all sentinel sources were indicating low levels of influenza-like illness in the community, it is possible that increased notifications early in 2008 were the result of increased testing and as such increased case finding.

Based on available data, the 2008 influenza season in Australia was considered moderate overall in comparison to previous seasons. Laboratory-confirmed influenza notifications were almost 2 times the 5-year mean, although this was less than notifications in 2007 (3 times the 5-year mean). There were no significant differences in laboratory-confirmed influenza notifications during the 2008 season compared with previous seasons in terms of geographic spread, with larger jurisdictions recording higher case numbers, and smaller jurisdictions having higher rates (due to smaller populations). Like previous years, the highest rates of laboratory-confirmed influenza occurred in children under 5 years of age, especially in those under 1 year of age. Although laboratory-confirmed influenza rates remained stable in ages over 10 years, the rate in children aged under 10 years decreased in comparison to 2007. This decrease was significant in children under 5 years of age, which may reflect higher rates of testing in this age group in 2007 following the heightened media attention.

In 2008 for the first time, sentinel GP surveillance system data were analysed by age and sex. It has been widely hypothesised that young children were under-represented in these data, as the vast majority of ILI presentations are in the 20–49 year age group. However, calculated rates indicate that the rate of ILI presentations to ASPREN GPs was highest in children under 5 years of age, especially male children under 1 year of age.

All sentinel data sources indicated that the 2008 season was moderate overall, consistent with laboratory-confirmed influenza data. ILI presentations to GPs and EDs, as well as influenza and pneumonia deaths, were lower in 2008 than in 2007, but higher than 2006.

Australia hosted World Youth Day (WYD) events in Sydney on 15–20 July 2008. Prior to WYD, influenza activity in Australia had been at baseline

levels, with influenza notifications and outbreaks predominantly influenza A. Several influenza outbreaks (including a number due to influenza B) occurred in pilgrims who had travelled to Sydney for WYD events. An increase in influenza coincided with pilgrims travelling around Australia, but it cannot be concluded whether this was due to spread of the virus by pilgrims or normal seasonal spread.

The 2008 season was predominantly influenza B, which accounted for 54% of all laboratory-confirmed influenza notifications. Younger age groups had higher rates of influenza B infection than older age groups, who were more commonly infected with influenza A. The WYD outbreaks may have contributed to this trend, as influenza B may have been spread from pilgrim groups to similar age groups as they travelled around Australia or as Australian pilgrims returned home. To confirm that this change had been as a result of pilgrim travel would have required detailed case follow-up and contact tracing, which was not possible due to the high numbers of influenza during the 2008 season.

Of influenza viruses circulating during the 2008 season A(H3) viruses were mostly antigenically similar to the vaccine strain A/Brisbane/10/2007. A(H1) strains however, showed significant drift away from the vaccine strain A/Solomon Islands/3/2006 to the A/Brisbane/59/2007-like viruses. As the season was predominated by influenza B, a large number of influenza B viruses were isolated in 2008. While overall there were approximately equal proportions of viruses from the 2 influenza B lineages (B/Victoria and B/Yamagata), B/Yamagata viruses were predominant at the start of the season, and B/Victoria at the end of the season.

Based on data available during the 2008 Southern Hemisphere influenza season, at their technical meeting in September 2008, the WHO recommended the following influenza virus strains for inclusion in the 2009 Southern Hemisphere seasonal influenza vaccine:

- A/Brisbane/59/2007-like virus (H1N1);
- A/Brisbane/10/2007-like virus (H3N1); and
- B/Florida/4/2006-like virus.

The recommendation for the 2009 Southern Hemisphere vaccine had only one change compared with the 2008 Southern Hemisphere vaccine: a change to the A(H1) virus from A/Solomon Islands/3/2006-like virus to A/Brisbane/59/2007-like virus.

Although various data sources were used to characterise the 2008 Australian influenza season, they were all consistent in indicating that the season

occurred later than usually seen but was moderate overall, which is characteristic of a predominantly influenza B season.

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