Annual report of the National Influenza Surveillance Scheme, 2007

Rhonda Owen, Ian G Barr, Andrew Pengilley, Conan Liu, Bev Paterson, Marlena Kaczmarek

Abstract

The year 2007 saw the most severe influenza season since national reporting of influenza began in 2001. Early in the season the National Incident Room was activated to provide effective national surveillance, reporting and management of the 2007 seasonal influenza outbreak. A surveillance team were tasked with establishing enhanced surveillance for the 2007 season and investigating unusual events in this outbreak. Key data required to comprehensively describe the number of cases, morbidity, mortality and virology of the influenza outbreak and the possible sources of these data were identified. In 2007 the number of laboratory-confirmed notifications for influenza was 3.1 times higher than the five-year mean. Forty-four per cent of notifications occurred in Queensland. High notification rates were reflected in an increase in presentations with influenza-like illness to sentinel general practices and Emergency Departments. Notifications and notification rates were highest in the 0–4 and 5–9 years age groups, possibly due to a bias towards testing in these age groups. The clinical morbidity of the infection in terms of complications or most affected groups cannot be determined but anecdotal reports indicate this season may have impacted young adults more than is usual. The available data suggest influenza has caused a significant burden on workplaces and the health care system as indicated by data on absenteeism and presentations for health care. The proportion of H1 strains of influenza circulating varied across Australia but was higher than 2006 in most jurisdictions. In 2007, 1,406 influenza isolates from Australia were antigenically analysed at the World Health Organization Collaborating Centre for Reference and Research on Influenza in Melbourne: 58.7% were A(H3N2), 34.4% were A(H1N1) and 6.9% were influenza B viruses. Antigenic drift away from the vaccine strain A/Wisconsin/67/2005 was observed with the A(H3N2) viruses and was also seen with most of the A(H1N1) viruses when compared with the vaccine strain A/New Caledonia/20/99. The small number of influenza B viruses examined were predominately of the B/Yamagata-lineage. Monitoring influenza through the National Incident Room during the 2007 season offered an excellent opportunity to conduct enhanced surveillance under conditions that were real and potentially serious but not an emergency. It enabled the current state of our surveillance systems to be assessed and opportunities for improvement to be identified. *Commun Dis Intell* 2008;32:208–226.

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.[1] 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 people 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 except South Australia and data are reported from each state or territory health department to the National Notifiable Disease 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 a moderate to severe season in 2003 but a mild season in 2006. The start of the annual influenza season is usually first detected by increased presentations at general practitioners (GPs) of 'influenza-like illness' (ILI) followed by increases in notifications of laboratory-confirmed influenza.

Surveillance methods

Data used to describe the 2007 influenza season were classified under the areas of epidemiology, morbidity, mortality and virology. Influenza surveillance was based on the following sources of data:

- notifications of laboratory-confirmed influenza required by legislation in most states and territories, and notified to the National Notifiable Diseases Surveillance System;

- 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 ILI diagnosed by sentinel general practitioners;
- consultation rates for ILI diagnosed by New South Wales sentinel hospital emergency departments;
- testing rates for influenza by New South Wales sentinel laboratories;
- media monitoring of reports of influenza outbreaks and deaths or of influenza impacts on hospitals;
- absenteeism data from a national employer; and
- New South Wales and Australian Bureau of Statistics (ABS) mortality data.

National Notifiable Diseases Surveillance System

In 2007, laboratory-confirmed influenza was a notifiable disease under state and territory legislation in all jurisdictions except South Australia. Laboratory notifications were sent to NNDSS for national collation. Although influenza was not a notifiable condition in South Australia, laboratory notifications were generally provided to NNDSS. In this report, data were analysed by the date of onset, but when this was not available the earliest date from either the specimen collection date or notification date was used.

Age, sex, method of laboratory diagnosis and postcode or locality of patient residence was included in NNDSS notifications. Maps were produced using ArcGIS.

Sentinel general practitioner surveillance

Sentinel general practitioner surveillance schemes for influenza monitor consultations for ILI. In Australia, there are five such schemes: the Australian Sentinel Practice Research Network (ASPREN), which collects data at a national level from approximately 90 general practitioners from six states and territories (Australian Capital Territory, New South Wales, Queensland, South Australia, Victoria, Western Australia); the Queensland Influenza-like Illness Sentinel Surveillance in General Practice Program; the Victorian Influenza Surveillance Scheme; Western Australian sentinel general practices; and the Northern Territory Tropical Influenza Surveillance Scheme. ASPREN and the Northern Territory Tropical Influenza Surveillance Scheme report ILI rates throughout the year, while the other sentinel surveillance schemes report only from May to October each year. 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 2007.

Emergency department surveillance

Rates for influenza-like illness presentation were collected from 30 emergency departments (ED) across New South Wales and data were provided to the Surveillance Branch, Office of Health Protection (OHP) within the Australian Government Department of Health and Ageing (DoHA), on a weekly basis, through the New South Wales Influenza Surveillance 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, 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, this forms 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[2] 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.)[3]

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

Absenteeism surveillance

A major nationwide employer, provided sick leave absenteeism data collected weekly for 2007. Absenteeism, defined as an absence for any reason for 3 or more consecutive days, was presented as a rate per 100 employees per week, on an average of 32,798 employees per week.

Media surveillance

Media information was sourced from Australian media clippings (Google News, Media Monitor) for the period 1 July to 30 August 2007. Clippings were scanned for articles relating to the 2007 influenza outbreak, particularly those relating to health system capacity.

Mortality

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

Deaths data compiled by the ABS from information provided by the state and territory Registrars of Births, Deaths and Marriages, and coded using the tenth revision of the *International Classification of Diseases and Related Health Problems* (ICD-10) were used to estimate historical levels of influenza deaths. In this report, deaths for 2006 with an underlying cause of influenza and pneumonia (ICD-10 J10–J18) are presented.[4]

Information on the sudden child deaths which were a feature of this year's season, was obtained during teleconferences or through the media.

Morbidity data

There was no effective measure of morbidity of disease readily available during the 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 collected by the Australian Paediatrics Surveillance Unit (APSU);
- emergency department presentations for ILI in New South Wales;
- ILI presentations to GP surveillance and ED networks; and
- media reports.

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

Results

The 2007 influenza season began in late May with a very gradual increase in notifications above non-seasonal levels from week 21 (week ending 27 May). At this time, consultation rates for ILI were similar to those of 2006 with small increases evident in most jurisdictions.

Increases in influenza notifications to the National Notifiable Diseases Surveillance System

At the end of July 2007, routine analysis of NNDSS data by epidemiologists in the Surveillance Branch of OHP identified a steep rise in influenza notifications diagnosed from week 28 (week ending 15 July), with levels reaching those of the peak in the 2003 season. Further analysis showed that the national increase was due to an increase in notifications in several jurisdictions, particularly Queensland and Western Australia. While it was thought likely that the increase in Western Australia was an effect of the increased media following the child deaths in that state, it was thought unlikely that this was the cause of the increase in Queensland. By this time influenza notifications in several jurisdictions had already exceeded the highest levels recorded in recent years.

Antigenic shift

Analysis of NNDSS influenza typing data indicated a change in the proportion of circulating influenza subtypes from that seen in previous years. Typing data for Queensland showed an increase in the proportion of type A(H1) relative to type A(H3). This change was reflected in the isolates typed by the World Health Organization Collaborating Centre for Reference and Research on Influenza (WHOCC) to that time, of which a significant proportion of cases were due to type A(H1).

The increase seen in influenza notifications was discussed with, and confirmed by Queensland Health. A Communicable Diseases Network Australia/

Jurisdictional Executive Group teleconference was held to discuss the epidemiology of influenza in each jurisdiction.

Jurisdictional representatives reported that influenza notifications were higher than recent influenza seasons in Queensland, New South Wales, Western Australia, the Australian Capital Territory and Tasmania. Notifications had started to rise in Victoria and the Northern Territory. South Australia was experiencing a moderate level of cases. Similar increases were seen in ILI consultation data from sentinel general practitioner sites. The age and sex distributions of influenza notifications were the same as in previous years.

The Queensland representative confirmed an increase in type A(H1) with 44% being type A(H1) compared with 1%–5% in previous years and 25% nationally.

The WHOCC reported that early testing showed a difference in the proportions of H1 and H3 strains across jurisdictions. Western Australian and Victorian isolates were mainly type A(H3) while Queensland and New South Wales isolates were a mixture of type A(H1) and type A(H3). In recent years, 2005 was the only season with a significant proportion of type A(H1) strains circulating with other seasons being predominantly type A(H3).

The WHOCC also reported that the circulating H1 strain appeared to be a genetic drift of the 2007 vaccine strain, but that the 2007 vaccine was expected to provide protection. The circulating A(H1) strains were a mixture of the vaccine strain A/New Caledonia/20/99, and the drift strain A/Solomon Islands/3/2006-like which was not in the vaccine. All Queensland H1 isolates were A/Solomon Islands/3/2006–like.

Laboratory confirmed cases

The first increases in notifications of laboratory-confirmed influenza in the 2007 season were registered in late May (week 21) when 35 cases were diagnosed. Notifications peaked in mid-August (week 33) and were almost back to inter-seasonal levels by the end of October (week 44) (Figure 1). The total number of notifications for the year was 10,577, which was 3.1 times the 5 year mean (Figure 2).

It should be noted that South Australia ceased sending influenza data to NNDSS on 31 August after changing to a new state database.

The median time from diagnosis to notification was estimated at 4 days in 2007, with 90% of notifications received within 14 days. Twenty-six per cent of notifications were received after seven days and 10% after 14 days. This time was calculated as the earliest recorded date (either onset date, diagnosis date

Figure 1. Laboratory-confirmed influenza notifications, 2007, by state or territory and week of diagnosis

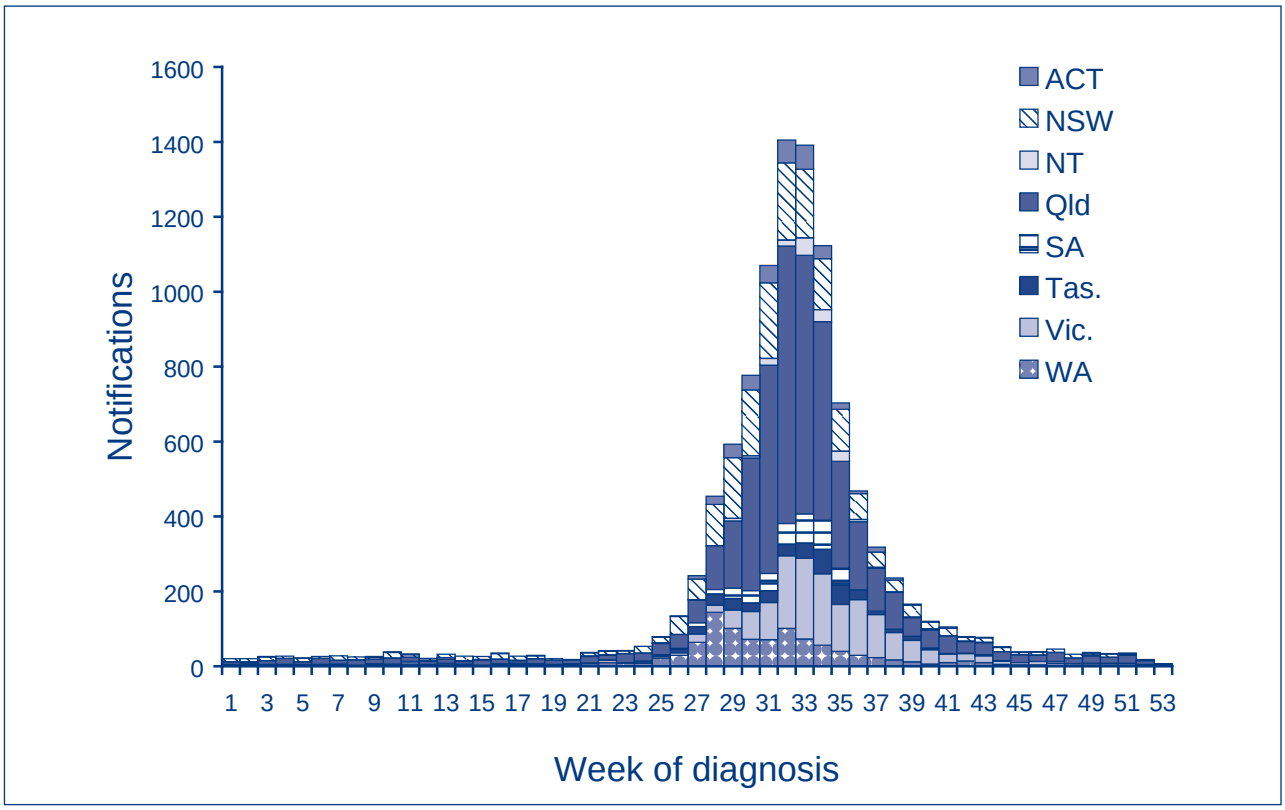


Figure 2. Laboratory-confirmed influenza notifications, 2003 to 2007, Australia, by month and year of diagnosis

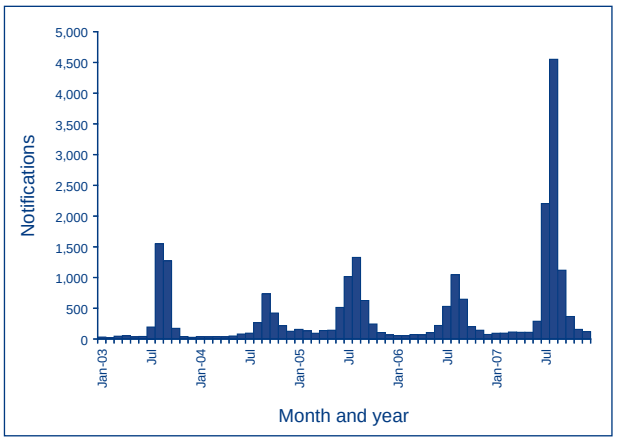
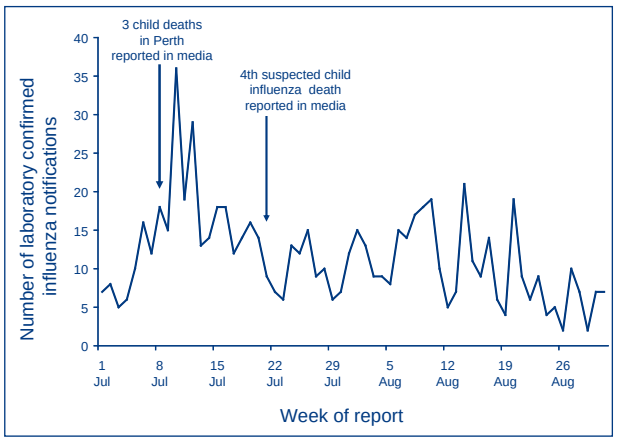


Figure 3. Number of laboratory confirmed notifications of influenza following paediatric deaths in Western Australia, National Notifiable Diseases Surveillance System, July to August 2007, by day of report



or laboratory date) to the date the information was received by the state or territory health department. In reality the timeliness was longer than this because of the additional time taken for notifications to be sent from the health department to NNDSS.

NNDSS laboratory confirmed notifications represent an unknown proportion of all influenza cases. Jurisdictions vary in the number and representativeness of samples they provide for laboratory confirmation and this affects the number of cases diagnosed and notified. The number of actual cases that each confirmed case represents varies between jurisdictions and between urban, rural and remote areas.

Media coverage of influenza deaths may also have increased the rate of presentations for health care and testing for influenza in children and thus laboratory diagnosis and notification. There appears to have been a large increase in influenza notifications from Western Australia following the deaths of 3 children, which were due to influenza (Figure 3).

Influenza-like illness consultations from sentinel general practitioner surveillance systems

Data from the ASPREN Sentinel GP Surveillance System showed that for 2007 there were 6,125 notifications for ILI. An average of 63 doctors reported to ASPREN each week, with an average of 6,437 consultations per week (range 2,530–9,356).

The rate of consultations for ILI began to increase in early June, peaking mid-July to mid-August and began to drop at the end of August (Figure 4). The rate of consultations for ILI mirrors that of laboratory-confirmed influenza notifications but increases in ILI consultation rates precedes increases in notifications by 1 to 2 weeks. The peak rate of ILI consultations to ASPREN in 2007 was approximately 50% higher than the peak rate reported in 2006.

Rates of ILI in individual state and territory sentinel GP surveillance systems, in general, mirrored the rates of influenza notifications in their jurisdictions (Figure 5). Western Australia was the only state where ILI consultation rates were lower in 2007 than in 2006.

Data provided by ASPREN were aggregated and did not allow for further analysis. A breakdown of sentinel GP data by state, postcode, age and sex would help to identify the demographics and geographic location of people presenting to general practitioners with ILI.

Figure 4. Consultation rates for influenza-like illness, ASPREN, 2006 and 2007, by week of report

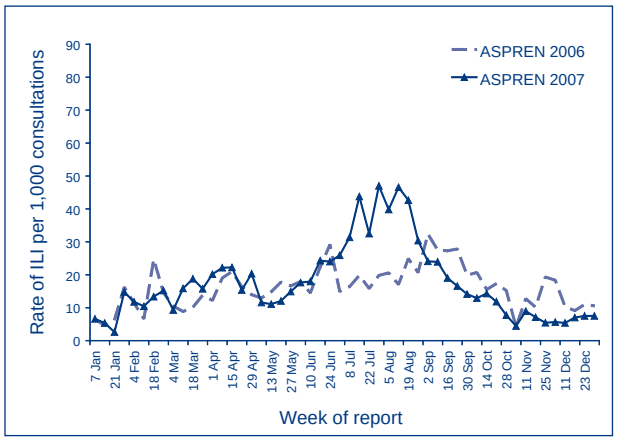
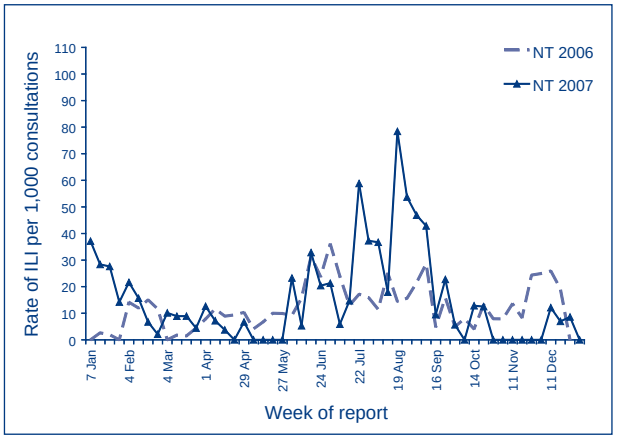
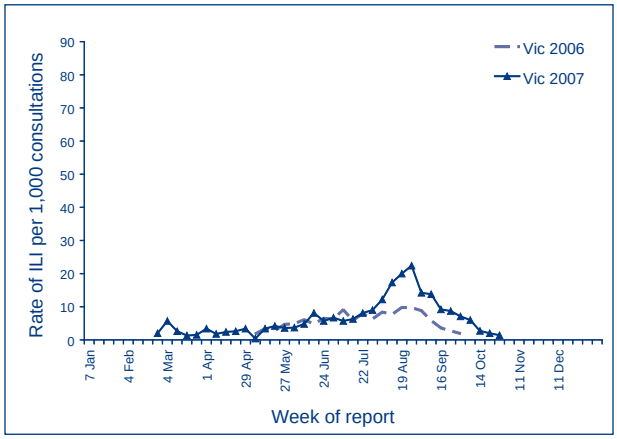


Figure 5. Consultation rates for influenza-like illness, 2006 and 2007, by sentinel surveillance scheme and week of report

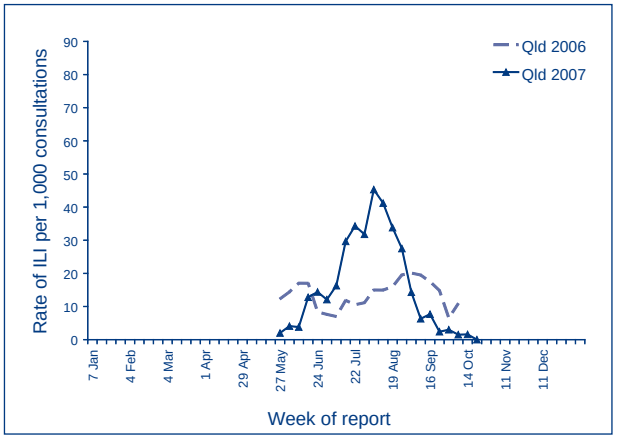
Northern Territory sentinel general practice



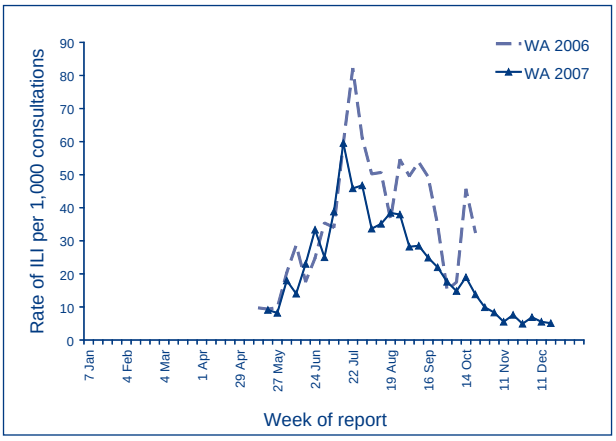
Victoria sentinel general practice



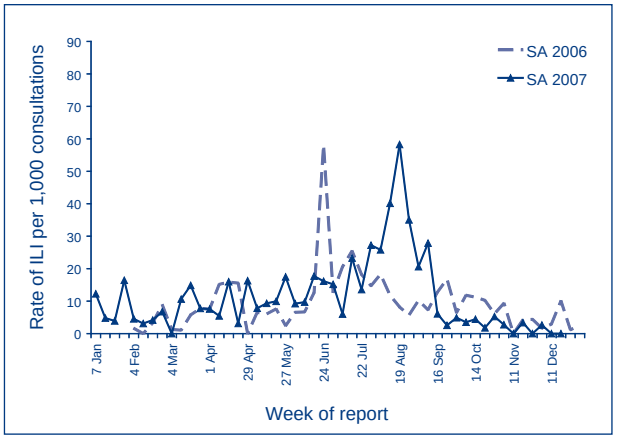
Queensland sentinel general practice



Western Australia sentinel general practice



South Australia sentinel general practice



While most ASPREN data are provided within a week of collection from GPs, some delay in reporting by some GPs mean that ILI rates are not stable for up to 3 weeks. Improvements to the timeliness of ASPREN data is important if increases in ILI are to be relied upon for early warning of the start of the influenza season.

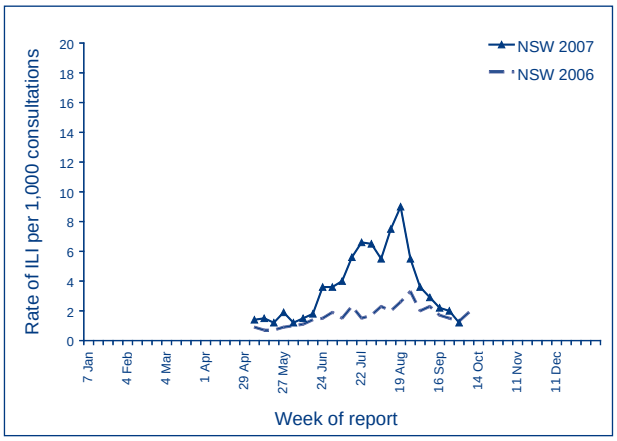
ASPREN data are not completely representative of the Australian population. There are no GPs from the Northern Territory or Tasmania that contribute data to ASPREN. Representativeness is also reduced with time due to declines in the number of reporting doctors.

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. The proportion positive of ILI presentations could not be determined.

Influenza-like illness – sentinel emergency department surveillance (New South Wales only)

Presentations to New South Wales emergency departments for ILI began to rise in mid-June and peaked at 9 per 1,000 consultations in mid-August (Figure 6). The increase in presentation rates reflected the rise in laboratory confirmed notifications of influenza to NNDSS. Presentation rates in 2007 exceeded those for any part of the 2006 influenza season.

Figure 6. Rate of influenza-like illness consultations from hospital emergency departments, New South Wales, April to September 2006 and 2007, by week of report



Sentinel ED surveillance data were timely but available from New South Wales only. ED surveillance systems operate in other jurisdictions but these do not routinely report data to the Australian Government Department of Health and Ageing.

Further work to identify how representative the ED data are and whether the demographics of people presenting with ILI to EDs are different to those who present to GPs will add to our understanding of this data set in future years.

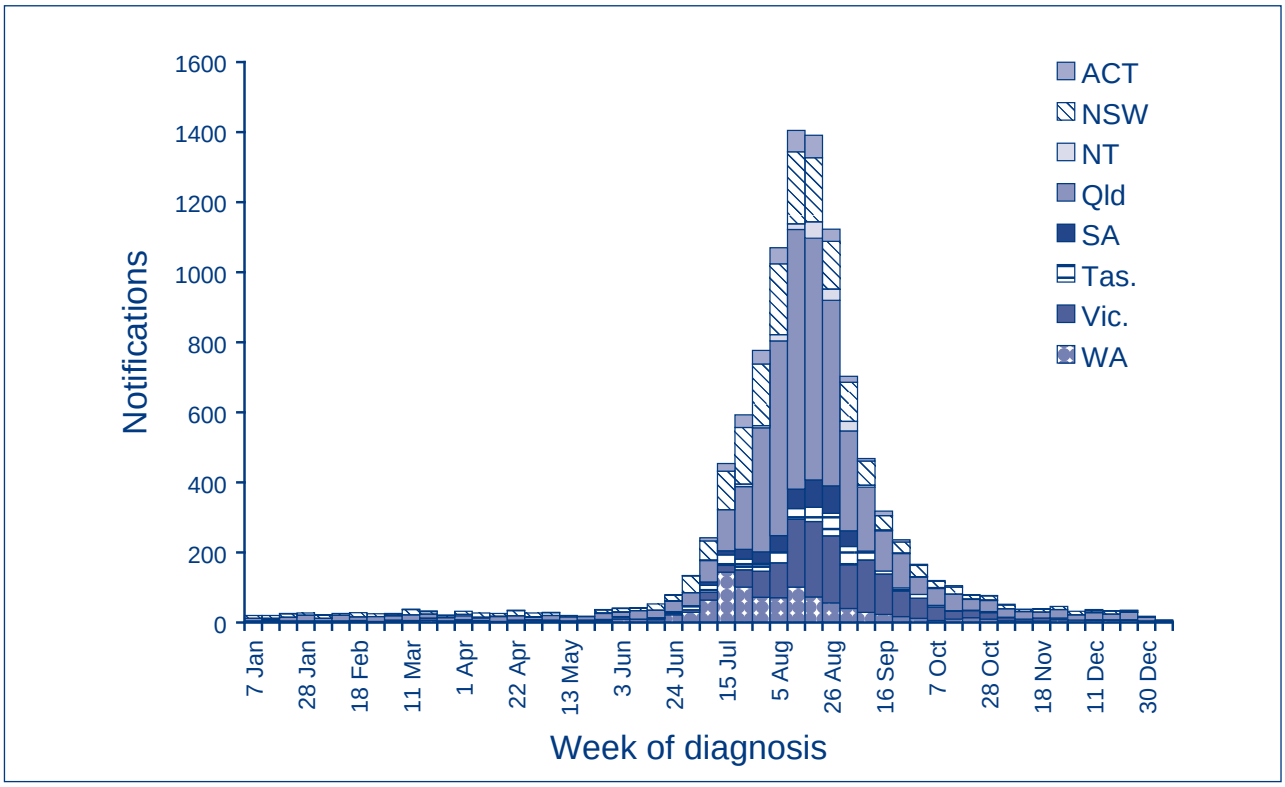
Geographic spread

In 2007, 44% of laboratory-confirmed influenza notifications occurred in Queensland, 18% in New South Wales, 15% in Victoria, 10% in Western Australia, 4% in South Australia, 4% in Tasmania, 4% in the Australian Capital Territory and 2% in the Northern Territory (Figure 7).

While the number of notifications peaked at a similar time in most jurisdictions, (at about week 33; week ending 19 August), the epidemic curve varies considerably by jurisdiction. Western Australia experienced a sharp, early increase in notifications at week 28 (week ending 15 July) likely caused by an increase in presentations following the influenza-related deaths of 3 children. A second peak occurred at about week 33. New South Wales also experienced an early increase in cases at week 28, while increases in Victoria commenced about four weeks later (week 32, week ending 12 August). In addition, notifications remained high for longer in Queensland, Western Australia and New South Wales (Figure 7).

Rates of notification for laboratory-confirmed influenza for 2007 varied across the country, ranging from 28 per 100,000 population in New South Wales to 115 per 100,000 population in the Australian Capital Territory. The rate of notification of influenza infec-

Figure 7. Laboratory-confirmed influenza notifications, May to October 2007, by state or territory and week of diagnosis



tion for Australia was 50 cases per 100,000 (Table 1). This was 3 times the mean rate over the previous 5 years (17.0 cases per 100,000 population).

The Map shows rates of laboratory-confirmed influenza in 2007 by Statistical Division. The highest rates of influenza occurred in statistical divisions, which encompassed the Northern Territory, northern Western Australia, parts of southern Western Australia, southern Queensland, Brisbane, the Australian Capital Territory and southern Tasmania.

Both state code (a mandatory code) and postcode of residence have high completion rates in NNDSS allowing the possibility of monitoring cases of influ-

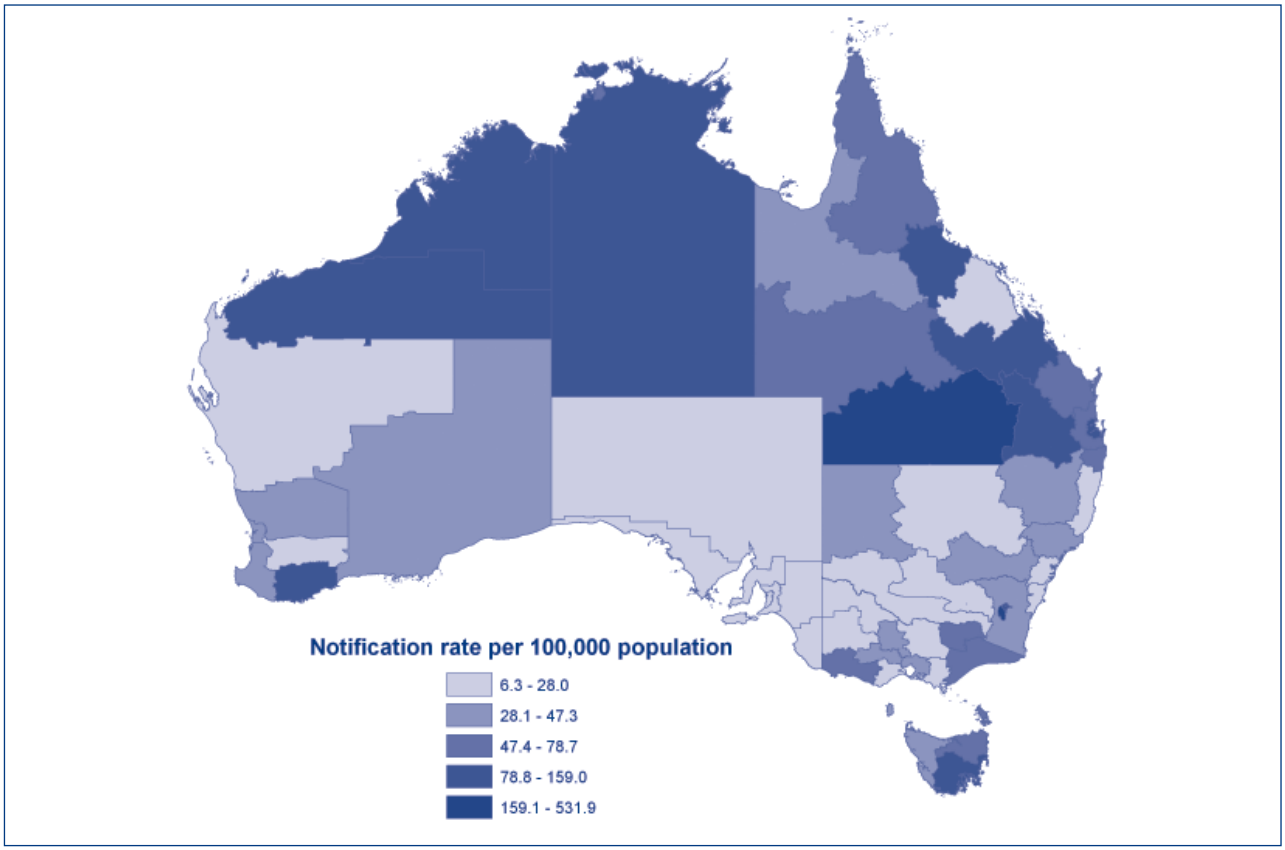
Table 1. Notifications and rates of laboratory-confirmed influenza notifications, 2007, by state or territory

State or territory	Total notifications	% of total	Notifications		Notification rate*		
			Male	Female	Male	Female	Total
Qld	4,644	43.9	2,215	2,429	106.0	116.0	111.0
NSW	1,918	18.1	991	923	29.0	26.6	27.8
Vic.	1,584	15.0	736	840	28.6	32.0	30.4
WA	1,038	9.8	522	516	49.0	49.6	49.3
SA	415†	3.9	180	234	74.0	93.6	84.1
Tas.	402	3.8	211	190	27.0	23.7	25.4
ACT	390	3.7	203	187	120.5	109.1	114.8
NT	186	1.8	109	77	97.6	74.5	86.5
Aus.	10,577	100.0	5,167	5,396	49.4	51.1	50.3

* Rate of notification per 100,000 population.

† South Australia ceased reporting notifications to the National Notifiable Diseases Surveillance System on 31 August 2007.

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

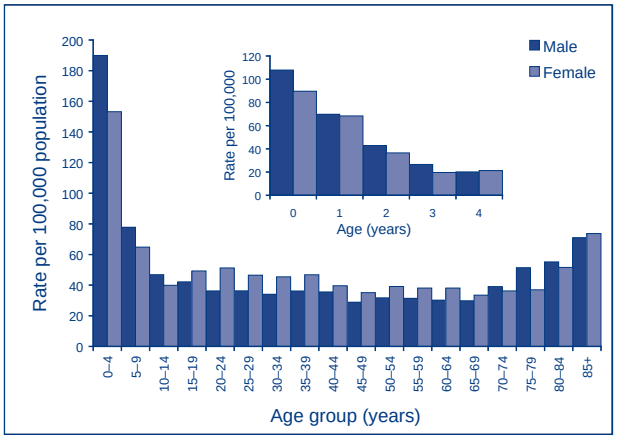


enza by time and location. Improved completion of typing data in NNDSS would also allow monitoring of virus type by location and time.

Age-sex profile

Age-specific notification rates for laboratory-confirmed influenza reported to the NNDSS in 2007 are shown in Figure 8. The highest notification rates were seen in those aged 0–4 years followed by the 5–9 years and 80–84 years age groups. Rates in the 0–4 years age group were around 3.4 times higher than for other age groups (170 cases per 100,000 population compared to an all-ages rate of 50 cases per 100,000 population).

Figure 8. Notification rate of laboratory-confirmed influenza, Australia, 2007, by age group and sex



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 43 cases per 100,000 population for both males and females. This compares with 2006 where influenza rates in 65 or over age group were 18 cases per 100,000 population for males and 17 cases per 100,000 population for females.

Forty-nine per cent of all notifications were male. Notifications were higher in females than in males for persons aged between 15 and 69 years. For children and the elderly, notifications for males exceeded those for females. The ratio of males to females in both the 0–4 years and 5–9 years age groups was 1.2:1.

Figure 9 shows rates of notifications for key age groups for the years 2003 to 2007. While rates increased in all age groups in 2007, the most marked were in the 0–4 and 5–9 year age groups.

Figure 9. Notification rate of laboratory-confirmed influenza reported to the National Notifiable Diseases Surveillance System, Australia, 2003 to 2007, by age group

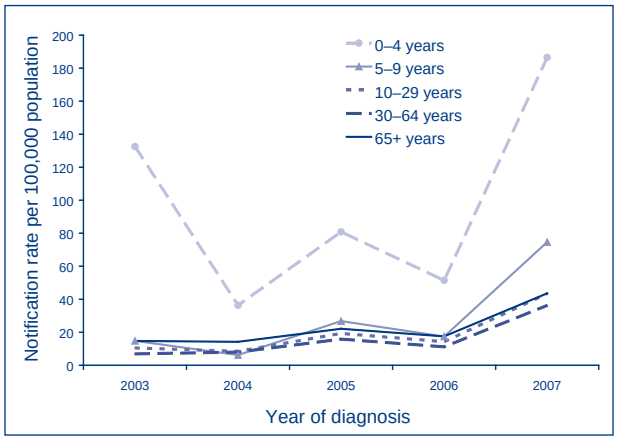
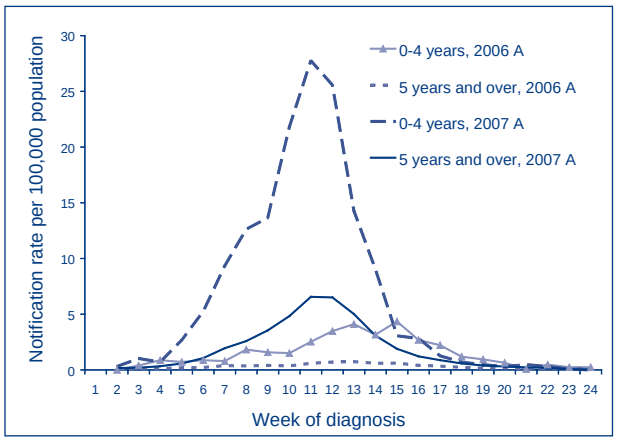


Figure 10 shows notification rates in key age groups in 2007 for influenza A. The data show that from mid-June, the notification rate for influenza A in children under 5 years of age increased significantly when compared to rates in other age groups. This increase was substantially higher than in 2006.

Figure 10. Notification rate of laboratory-confirmed influenza type A from week 23 to week 45, Australia, 2006 and 2007, by age group and week

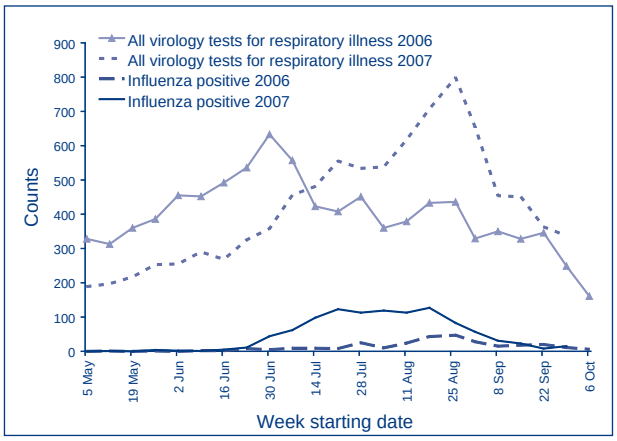


Laboratory surveillance

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 July 2007, to over 800 tests per week in late August, exceeding the peak of approximately 600 tests per week in 2006 (Figure 11). The percentage of virological tests

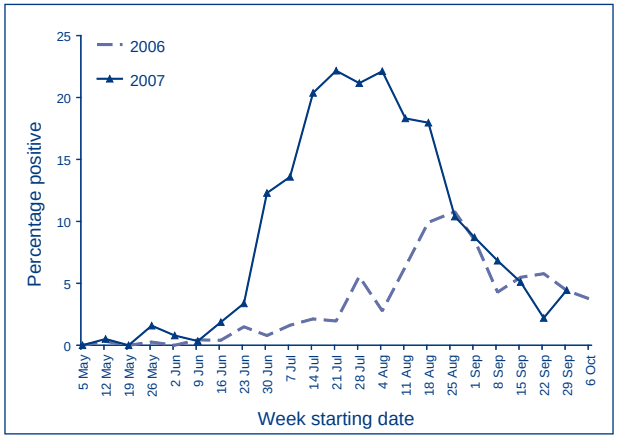
positive for influenza increased over the season from 1.9% in June to over 20% for the period between mid-July and early-August (Figure 12). Serology positive tests showed similar trends. This confirms that the increase in the number of cases of seasonal influenza was not an artefact of increased testing.

Figure 11. Total virology specimens tested and number positive for influenza A, New South Wales, May to October 2006 and 2007



Data source: NSW Influenza Surveillance Report, 22 to 28 September 2007

Figure 12. Percentage of virology specimens testing positive for influenza A, New South Wales, May to October 2006 and 2007



Data source: NSW Influenza Surveillance Report, 22 to 28 September 2007

Laboratory diagnosis method analysis

NNDSS notifications for influenza were analysed for laboratory diagnosis methods used.

Table 2 shows that during the period 1 June 2007 to 31 October 2007, 37% of laboratory-confirmed notifications were diagnosed by polymerase chain reaction (PCR) alone (n=3,545 of 9,672). Serology and culture-based diagnoses accounted for 21% and 9% of notifications respectively (n=3,545, n=880). Laboratory-confirmed influenza notifications based on antigen detection with and without confirmatory testing accounted for 16% of notifications (n=1,547) overall. The laboratory method was unknown in 14% of the total notifications for this period.

Trends in laboratory diagnosis methods used during the 2007 season are shown in Figure 13. At the peak of the season (week 33; week ending 19 August), 39% of notifications were diagnosed by PCR alone (n=559 of 1,441). Notifications based on antigen detection and antigen detection alone peaked in week 31 (16%, n=173). The proportion of weekly notifications based on less specific serological tests were low and use of these tests declined in the peak of the season.

Morbidity

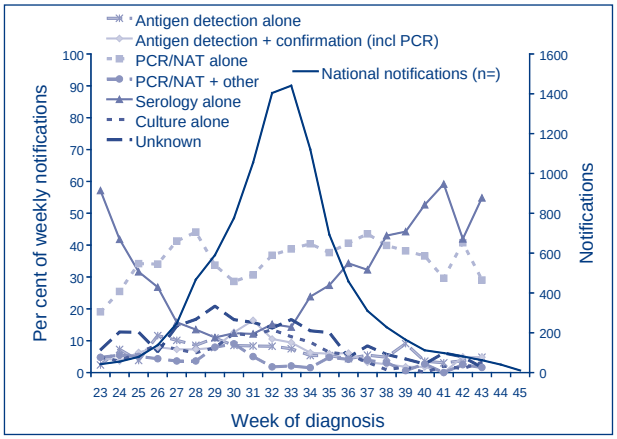
APSU surveillance

Results from the APSU survey commissioned by DoHA showed that for the month of September 2007, 15 cases aged under 5 years were admitted

Table 2. Laboratory diagnosis method of influenza notifications, Australia, 1 June to 31 October 2007

Laboratory diagnosis method	n	%
Antigen detection alone	704	7
Antigen detection + confirmation (including polymerase chain reaction)	843	9
Polymerase chain reaction or nucleic acid testing only	3,545	37
Polymerase chain reaction or nucleic acid testing + other laboratory test	361	4
Serology only	1,997	21
Culture only	880	9
Unknown laboratory method	1,342	14
Total	9,672	100

Figure 13. Percentage of weekly notifications reported to the National Notifiable Diseases Surveillance System, 4 June, 2007 (week 23) to 5 November (week 45), by method of laboratory diagnosis and week of diagnosis



to hospital with complications from influenza. Of the 15 cases, the ratio of male to female was 4:1 and 3 of the cases were identified as Aboriginal or Torres Strait Islander. Thirteen cases were influenza A and 2 were influenza B. The survey was not conducted for the whole of the influenza season.

Media reporting

Media reports discussed the clinical severity of the 2007 influenza strains by emphasising deaths (many descriptions of 'killer flu' in national print media) but also noted the impact of influenza on emergency department waiting times. Media clippings reported that during the 2007 influenza season:

- there were long waiting lists to see doctors;
- doctors were working longer hours;
- pharmacists saw increases in the demand for anti-virals and flu treatments;
- medical advice lines (such as Health Direct in Western Australia) had unprecedented demand with an 80% increase in calls;
- blood supplies were low because of illness in the donor population; and
- hospitals were regularly put on divert as the influenza season reached its peak.

In areas with a particularly severe influenza season, the media reported that hospitals were overwhelmed with both the sick and the worried well. In response to this, nurses worked extra shifts; holidays were cancelled; doctors were recruited from other departments to work evenings and weekends to relieve pressure; elective surgery was postponed; hospitals wards were closed down to enable staff to be freed up to work in other areas; and additional influenza clinics were opened next to the Emergency Departments.

Media also discussed absenteeism issues relating to the influenza season; recommendations that sick people stay at home and the use of masks by those who were infectious.

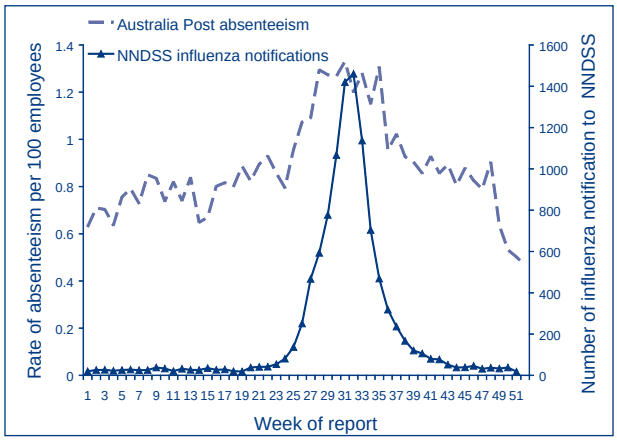
The media proved a useful mechanism for understanding public concerns relating to the influenza season. The media provides an early indicator of the perceived severity of the influenza season. Media can provide useful information that is not accessible through standard data sources. Following media attention on outbreaks, Emergency Departments may be overwhelmed with worried parents and the worried well.

Absenteeism surveillance

Absenteeism rates generally increase several weeks before notifications start increasing. This can provide an early warning system of the severity of the influenza season. Absenteeism is likely to be elevated by public health messages to stay home if unwell.

Absenteeism was higher during the 2007 influenza season than in the past five years. The absenteeism figures for 2007 (Figure 14) were the highest since collection commenced in 2002. The proportion of workers absent for more than 3 consecutive days peaked at 1.33% for the week ending 8 August 2007 (Week 31): the highest rate recorded since data has been collected.

Figure 14. National absenteeism (more than 3 consecutive days) rates and National Notifiable Diseases Surveillance System influenza notifications, 2007, by week of report



A comparison of absenteeism rates and laboratory-confirmed influenza notifications is shown in Figure 14. The 2007 figures show a broader peak than the laboratory confirmed notifications with the highest rates of absenteeism during the period 12 July to 5 September 2007.

The absenteeism rate was only measured in workers and does not reflect an 'incapacity' rate among the population in general. There is also a time lag as illness is only reported on return to work.

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.

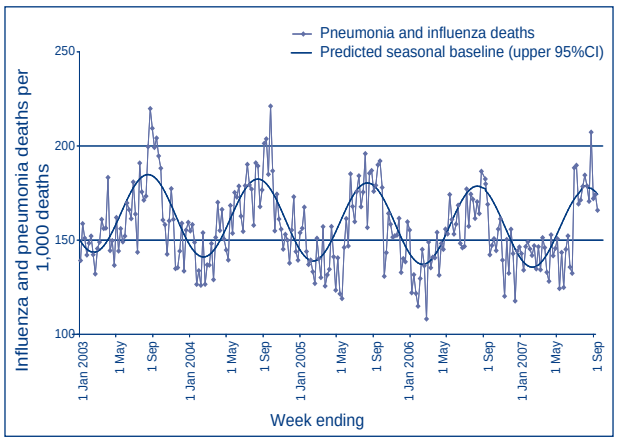
Deaths from pneumonia and influenza – New South Wales

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 late August at 210 per 1,000 deaths (Figure 15). The combined pneumonia and influenza death rates were higher than those of 2006 and for several weeks were equal to or higher than the upper 95% confidence interval of the predicted seasonal baseline.

Australian Bureau of Statistics death data

The most recent data on causes of death in Australia are for 2006. Influenza and pneumonia (ICD-10 codes J10–J18) were noted as the underlying cause of death for 2,715 persons in 2006 (2.0% of all deaths). More females than males died of influenza or pneumonia (1,495 females compared to 1,220 males); however the standardised death rate for males was higher than for females (14.1 versus 10.2).[4]

Figure 15. Observed and predicted rate of influenza and pneumonia deaths as per New South Wales registered death certificates, January 2003 to September 2007



Source: New South Wales Influenza Surveillance Report, 22 to 28 September 2007.

Australian Institute of Health and Welfare death data

Australian Institute of Health and Welfare (AIHW) data show a mean of 2.6 deaths per year from influenza, in children aged 0 to 4 years, over the 9 years from 1997 to 2005 (Table 3). In 2007, 7 child deaths were reported by jurisdictions as associated with influenza.[5]

Virology

Antigenic characterisation

Of the 10,577 influenza cases notified to NNDSS in 2007, 95% included typing data. Influenza A was the predominant circulating type comprising 86% of isolates typed compared to 9% that were type B and 0.4% mixed type A&B (Figure 16). The finding of 0.4% of notifications having both types of influenza was lower than in previous years but is still higher than would be expected, as documented reports of dual infections are rare.[6] This may warrant further investigation in the future to confirm true dual influenza infections. In 2006, 75% of typed isolates were influenza A and 25% influenza B.

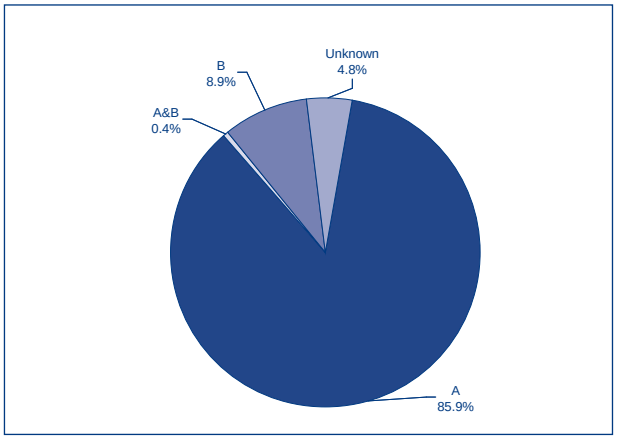
Table 3. Influenza deaths, Australia, 1997–2005, in children aged less than 5 years

	1997	1998	1999	2000	2001	2002	2003	2004	2005
Influenza deaths 0–4 years (J10, J11)	2	6	2	3	2	1	2	3	2

Source: Australian Institute of Health and Welfare 2007.

GRIM (General Record of Incidence of Mortality) Books. Australian Institute of Health and Welfare: Canberra.

Figure 16. Number of influenza notifications reported to the National Notifiable Diseases Surveillance System, Australia, 2007, by type



The WHO Collaborating Centre for Reference and Research on Influenza received 1,406 isolates or clinical specimens from Australian laboratories in 2007 that yielded viable influenza viruses. This was the second highest number of isolates received over the last 10 years (the highest being in 2002). All of the 2007 viruses were analysed antigenically using the haemagglutination inhibition (H1) assay, which identified 826 (58.7%) as A(H3N2) strains, 483 (34.4%) as A(H1N1) strains and 97 (6.9%) as influenza B strains. The 2007 Australian A(H3N2) viruses were antigenically similar to either the 2007 vaccine strain A/Wisconsin/67/2005 or the newly emergent variant A/Brisbane/10/2007, with a few viruses not showing either of these HI patterns (Table 4). Consistent with the antigenic drift seen with a large number of the A(H3N2) isolates when tested with specific ferret antisera, serological studies conducted with pre– and post-vaccination human sera from recipients of 2006 vaccine which contained the A/Wisconsin/67/2005 strain, showed that there was a reduction in antibody titres to many 2007 A(H3N2) isolates. Antigenic analysis of the Australian 2007 A(H1) strains, also showed that there was significant drift away from the 2007 vaccine strain A/New Caledonia/20/99. Few influenza B viruses were isolated in 2007, but from those analysed, only 21% were antigenically related to the 2007 vaccine strain B/Malaysia/2506/2004 (B/Victoria/2/87-lineage viruses) while the remaining 79% were closely related to B/Florida/7/2004-like viruses (B/Yamagata/16/88-lineage viruses) representing the alternative B lineage.

Sequence analysis of the variable (HA1) region of the haemagglutinin (HA) gene was undertaken for 133 Australian 2007 viruses (40 A(H1), 72 A(H3) and 21 B) and for 73 the neuraminidase genes, (23 H1, 41 H3, 9 B). The phylogenetic analysis of the 2007 (H3) virus HA1 sequences showed that most Australian A(H3) viruses were closely related to A/Brisbane/10/2007-like viruses although some also grouped closely with related viruses recently isolated from South East Asia (such as A/Thailand/31/2007 and A/Macau/200/2007). Viruses isolated from fatalities involving 3 young children (<5 years) in Perth in July 2007 and 1 from Victoria also grouped in the A/Brisbane/10/2007 phylogenetic group, as did viruses from non-fatal childhood cases (e.g. A/Perth/26/2007) (Figure 17).

When the HA1 genes from A(H1) viruses isolated in Australia in 2007 were compared phylogenetically, they fell almost exclusively into the group of viruses represented by A/Brisbane/59/2007 (Figure 18). A few viruses fell into in other groups

Table 4. Antigenic comparisons of influenza A(H3) viruses by the haemagglutination-inhibition test

Virus antigen	Ferret antiserum Reciprocal haemagglutination-inhibition titre:	
	A/Wisconsin/67/2005	A/Brisbane/10/2007
A/Wisconsin/67/2005	**640***	1,280
A/Brisbane/10/2007	640	**640***
A/Perth/117/2007	640	1,280
A/Sydney/103/2007	320	320
A/South Australia/61/2007	80	160
A/Victoria/290/2007	80	320
A/Darwin/12/2007	80	320
A/Brisbane/188/2007	80	320
A/South Australia/41/2007	80	40
A/Canberra/1/2007	<40	160

* An A/California/7/2004-like strain (A/New York/55/2004) was the H3 strain used in the 2006 Australian influenza vaccine.

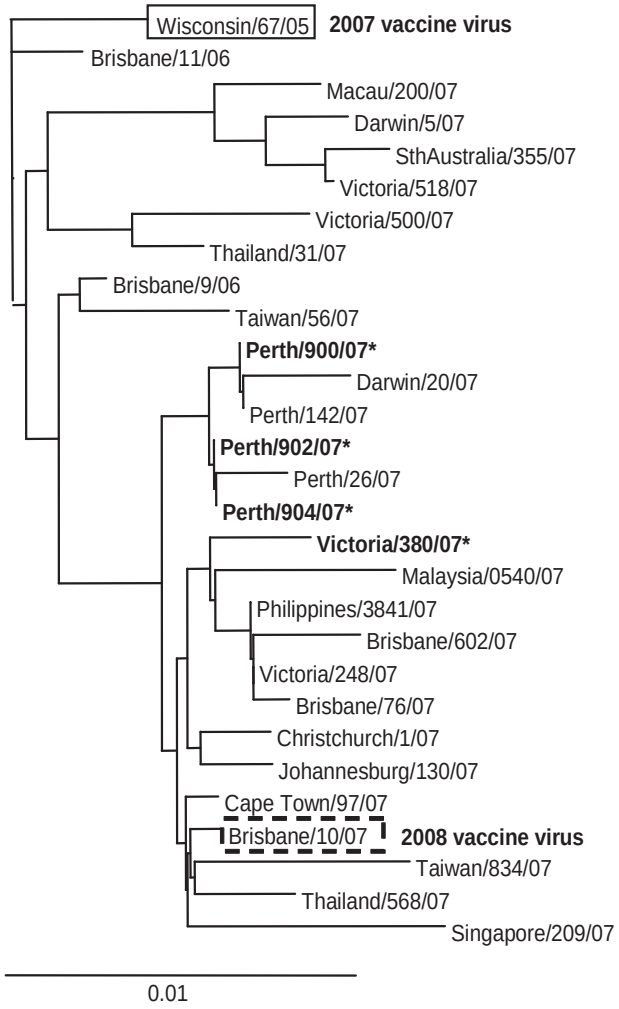
represented by the A/Hong Kong/2652/2006 or A/Brisbane/193/2004. Two A(H1) viruses associated with fatalities in young children also grouped with the A/Brisbane/59/2007-like viruses as did the 3 Tamiflu resistant A(H1) viruses isolated from Australian patients in late 2007 (Figure 18). The Australian 2007 influenza B viruses phylogenetically grouped into their respective lineages, either the B/Victoria or B/Yamagata lineage, with the B/Victoria-lineage viruses showing little change from the reference/vaccine strain B/Malaysia/2506/2004 while the B/Yamagata-like viruses showed only minor changes from the reference virus A/Florida/7/2004 but were somewhat better represented by the A/Brisbane/3/2007 virus (Figure 19).

International trends in influenza

In 2007 global influenza activity was generally moderate when compared with previous years. Influenza was detected in most countries and typically all 3 types/subtypes co-circulated (i.e. A(H1N1), A(H3N2) and influenza B). Levels of influenza A(H1) activity however, did increase in many countries compared with previous years. Outbreaks of A(H1) were reported in several countries, including Australia, Japan, Mexico, New Zealand, South Africa and the United States of Australia. In the Southern Hemisphere, influenza activity began in April and increased until July and then began to decline through August. In South America influenza A(H3N2) and B viruses co-circulated while influenza A(H1N1) and A(H3N2) (44 % and 31% respectively of all influenza viruses New Zealand and A(H1N1) viruses predominated in South Africa.

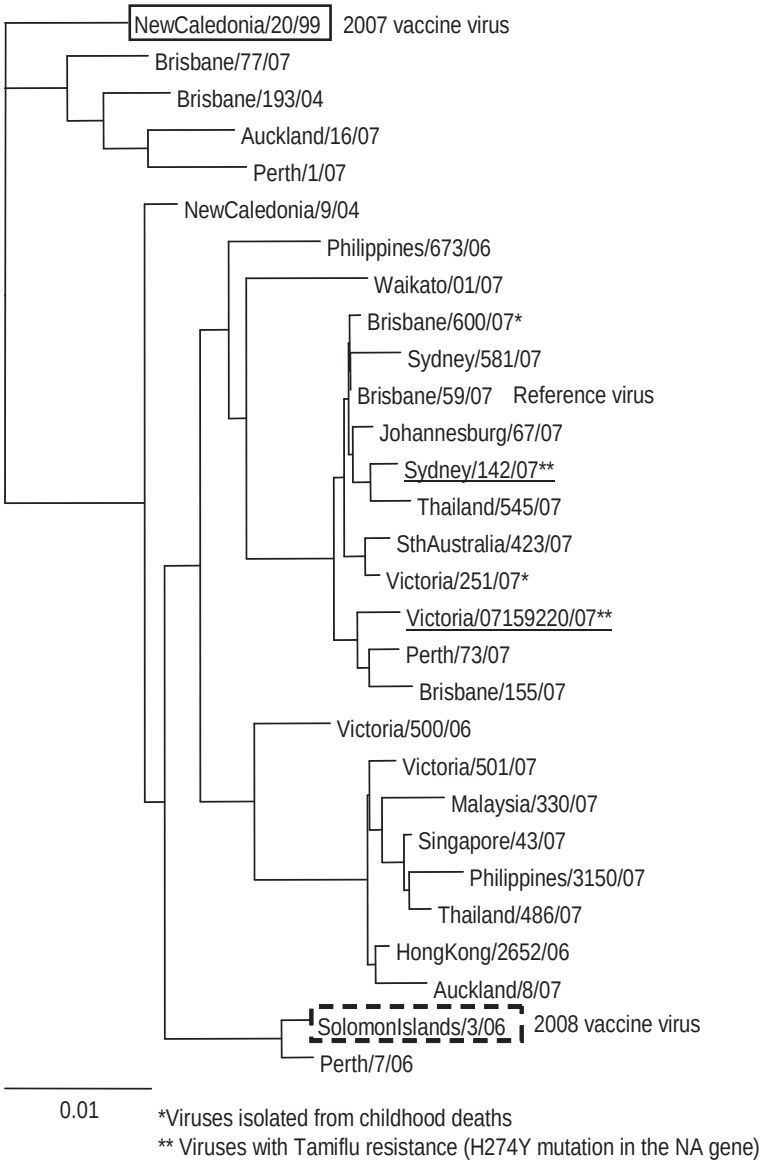
Outbreaks of A(H5N1) highly pathogenic avian influenza (HPAI) in poultry and wild birds still occurred in many parts of the world in 2007, with the continued exceptions of the Americas and Oceania. According to the official WHO figures, 88 H5N1 human infections occurred in 7 countries during 2007 resulting in 59 deaths. This was lower

Figure 17. Evolutionary relationships between influenza A(H3) haemagglutinins (HA1 region)



*Viruses isolated from childhood deaths

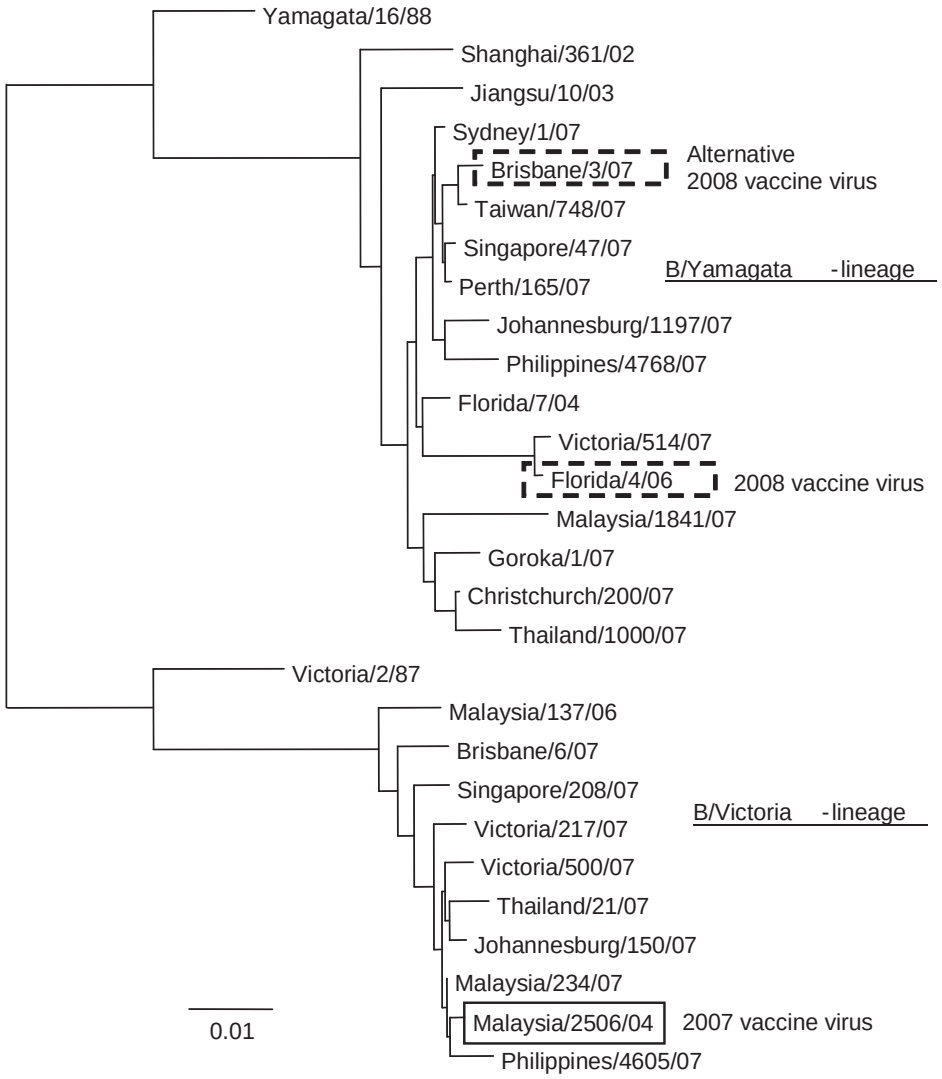
Figure 18. Evolutionary relationships between influenza A(H1) haemagglutinins (HA1 region)



than seen in either 2005 or 2006, (for details see the WHO avian influenza web site http://www.who.int/csr/disease/avian_influenza/en/). No highly pathogenic H5N1 infections were detected in humans or in birds in Australia in 2007.

While the temporal pattern of the annual influenza season in New Zealand is broadly similar to Australia, outbreaks often begin earlier in the year. In 2007 the New Zealand consultation rates for ILI started to increase in late May, with a minor peak at week 27 (first week of July) reaching a major peak at week 30–31 (late July) with levels then declining and returning to baseline during September. A lower level of hospitalisation with influenza was seen in 2007 (347 people) compared with previous years (2005; 390 people, 2006; 464 people). Of the 345 New Zealand isolates typed at the WHOCC, the majority were A(H1) (44.1%) with fewer A(H3) viruses (19.7%) and influenza B viruses (36.2%). This was in contrast to 2006 where the vast majority of New Zealand viruses tested at the Centre were influenza A(H3) viruses (80.4%). Interestingly, the majority of the New Zealand A(H1) viruses were antigenically closely related to the A/New Caledonia/20/99 vaccine strain with only one third typed as A/Solomon Islands/3/2006-like along with a few A/Brisbane/59/2007-like viruses. A(H3) viruses were mainly A/Brisbane/10/2007-like while the influenza B viruses were mainly of the B/Yamagata-lineage (B/Florida/7/2004-like) with very few B/Malaysia/2506/2004-like viruses (B/Victoria-lineage). Overall influenza activity in New Zealand in 2007 was low and below levels seen in 2003–2006. The full Environmental Science and Research report

Figure 19. Evolutionary relationships between influenza B haemagglutinins (HA1 region)



on the 2007 influenza season in New Zealand is available on their web site at: http://www.surv.esr.cri.nz/virology/influenza_annual_report.php

Review of paediatric deaths from influenza, 2007

During the 2007 influenza season, eight children under five years of age died within 24 hours of developing mild and non-specific symptoms of respiratory infection. Although the cause of death has not been reported in many of these cases, these deaths appeared to be associated with influenza A. The apparent virulence of the infection led DoHA to attempt to collate a case-series to identify any common or predictive features of influenza A infection in these cases. Paediatric deaths are not routinely reported as a separate finding in seasonal influenza surveillance.

Surveillance of paediatric deaths was undertaken using several sources of data:

- jurisdictional communicable diseases units were asked to report any influenza associated deaths occurring in children aged 0–4 years, or any instances of children this age being admitted to ICU within 24 hours of the onset of influenza. A case report form was constructed in NetEpi and made available to the jurisdictions although information was accepted by DoHA in any format.
- The Australian Paediatric Surveillance Unit was requested to report any cases of children aged 0–4 years who had presented to ICU within 24 hours of the onset of influenza. A case report form was constructed in consultation with APSU. Reports of paediatric deaths from influenza appearing in the national media were reviewed and significant information noted.

The NetEpi and ASPU forms differed in some details, and the NetEpi form surveyed greater detail about the child's presenting illness and possible contacts. This was not considered problematic because both the jurisdictional public health units

and APSU should have been reporting the same cases, allowing the data received to be pooled into single case reports.

Case data

Details of a single case were received from NSW Health using the NetEpi system. Of the 15 cases aged 0–4 years presenting to ICUs within 24 hours of the onset of influenza, identified by APSU, there were no deaths. Other details were collated from teleconference notifications to the Communicable Diseases Network of Australia or media reports.

Sufficient data were available to include 8 paediatric deaths in the series. Minimal information was available on a further 3 children who were admitted to ICU and survived, and another possible further case of death from influenza B. Influenza was isolated in seven of the children who died, while adenovirus was isolated from the eighth child. Where data were provided on the duration of illness (6 of 7 influenza deaths), death occurred within 48 hours of the onset of symptoms. Influenza A subtype H3N2 was isolated from 3 cases and H1N1 from 4 cases in the 7 deaths where influenza was detected. Insufficient information was provided on medications, previous illnesses or exposure to animals to allow hypothesis generation of the cause of death. It was notable that the first 3 Western Australian deaths had group B streptococcus isolated from blood samples post-mortem but the significance of this is not clear.

All seven of the influenza deaths were boys. It is improbable that all 7 deaths would be male if the probability of a male and a female being in the sample was equal ($p=0.07$). This does not mean that the observed sex-bias was due to differential susceptibility to disease as the case series is not complete and the numbers are small. The finding is, however, interesting, given the higher preponderance of asthma and other possibly significant co-morbidities in boys aged 0–4 years.

Death from influenza is rare in children aged 0–4 years. The AIHW reported between 2 and 6 deaths per year from influenza in this age group between 1997 and 2005, with no data available for the past 2 years. Eleven child deaths in 2007 appears higher than expected, but this cannot be quantified without an estimate of the number of cases of infection among children occurring in different years.

Since enhanced surveillance was unable to identify definitively either the number of deaths in children 0–4 years of age which occurred during the 2007 season or many of the significant clinical details regarding these deaths, no epidemiological or clinical conclusions can be drawn from the data reported. It has not been possible to inform public health interventions based on an analysis of sudden deaths in this age group.

It was considered an important element of influenza surveillance to attempt to examine the number and circumstances of sudden deaths occurring in children. Deaths from influenza in this age group are rare, and therefore a measurable increase could reflect the emergence of a virulent strain of influenza. Acquiring a defined case series was assisted by the low 'background' rate of influenza deaths among children but this also necessitated active surveillance to avoid these deaths being lost in aggregate death data. Children are not usually a target of measures to prevent influenza, such as vaccination, and evidence of an increased level of risk may have helped inform decisions about management in the 2007 season. It was also not immediately apparent that the first 3 reported deaths were due to influenza, this diagnosis being made post-mortem, and accurate case reporting was necessary to exclude a novel emerging pathogen.

A more consistent national system is required for the early identification, collection and provision of unexplained paediatric deaths. Information should be collated nationally to detect any changes in the virulence, host-risk-factors or co-morbidities associated with death from circulating strains of influenza. If all deaths are reported and reported early this will provide better data on the number of deaths which are expected during an influenza season. Accurate baseline data would provide a quantitative measure against which to assess 'bad' influenza seasons, as well as form part of the surveillance for new or potentially pandemic strains of the virus.

Discussion

The impact of an influenza season and its severity is difficult to measure. Ideally a severity index would include the number of cases along with the burden of disease on individuals (such as hospitalisations) and the burden on the health system. At this time only the number of cases in an influenza season are able to indicate how severe the season was in Australia. In 2007, the largest number of notifications was seen since inception of influenza as a notifiable disease in 2001.

The 2007 influenza season was considered a moderate to severe season as measured by the number of cases. While the role of heightened media attention, differing diagnostic tests, and non-representative referral of samples makes an estimate of the true number of cases of influenza impossible, there were more than 3 times the number of laboratory-confirmed notifications compared with the five year mean. Furthermore,

an epidemic case load was recorded through ILI surveillance and absenteeism was higher than previous years.

The majority of the Australian isolates analysed at the WHO Influenza Centre were A(H3N2) viruses however, there was a substantial proportion of A(H1N1) viruses co-circulating along with a few B viruses. This was the highest proportion of A(H1N1) viruses that have circulated in Australia since 2001 when they were the predominant strain. Over the next 5 years from 2002 to 2006, the proportion of A(H1N1) strains seen annually in Australia has been <1%, <1%, 18.8%, and 3.5%, respectively. Accordingly, the extensive co-circulation of 2 influenza A strains may have contributed to the severity of the influenza season seen in Australia in 2007. Both A(H1N1) and A(H3N2) viruses were isolated from fatal cases involving children under 5 years of age as well as in adults, although any contribution that these influenza infections may have had in these deaths remains to be determined.

Antigenic drift away from the 2007 vaccine strains A/New Caledonia/20/99 and A/Wisconsin/67/2005 was seen for both the influenza A(H1N1) and the A(H3N2) viruses respectively. Antigenic and genetic analysis of these viruses showed the majority of A(H1N1) viruses isolated in Australia in 2007 were more closely related to the reference strain A/Brisbane/59/2007 while the A(H3N2) viruses were more closely related to the reference virus A/Brisbane/10/2007. Interestingly, in New Zealand the majority of the A(H1N1) strains were still similar to the vaccine strain A/New Caledonia/20/99. Three A(H1N1) strains with the H274Y mutation in the neuraminidase gene were detected in Australian 2007 isolates. This mutation confers oseltamivir (Tamiflu) resistance and appears to have arisen in Europe during the 2007–08 season[7] and was prevalent in a number of countries (e.g. Norway had 67% resistant A(H1) viruses). Few influenza B viruses were isolated in Australia in 2007 (97) and the majority of these (79%) were B/Florida/7/2004-like (B/Yamagata-lineage) while the remaining viruses (21%) were similar to the 2007 vaccine virus, B/Malaysia/2506/2004 (B/Victoria-lineage). This was in contrast to the 2006 season where B/Malaysia/2506/2004-like viruses predominated. Influenza A(H3N2) and B viruses in New Zealand were similar to those seen in Australia in 2007.

The WHO annual consultation on the composition of influenza vaccines for the Southern Hemisphere, 2008 took place in Geneva from 17–19 September 2007. The recommended composition of influenza virus vaccines for use in the 2008 Southern Hemisphere influenza season was:

— an A/Solomon Islands/3/2006 (H1N1)-like virus;

— an A/Brisbane/10/2007 (H3N2)-like virus;

— a B/Florida/4/2006-like virus.

The recommendation for the 2008 vaccine had changes to all 3 viruses compared to the previous year's Southern Hemisphere vaccine. A further change has been recommended for the 2008–09 Northern Hemisphere influenza vaccine, with the A(H1N1) component being updated to incorporate an A/Brisbane/59/2007-like virus. B/Brisbane/3/2007 is considered an B/Florida/4/2006-like virus and so can be used for the 2008 vaccine instead of B/Florida/4/2006.

Many sources of data were available to characterise the epidemiology of the 2007 influenza season. However, important opportunities to improve the surveillance data were also identified. These included enhanced data collection to maximise completeness; improved data quality; and opportunities to ease the collection of data. Improved and consistent protocols for data collection would also help to improve the representativeness of information across jurisdictions.

Potential sources of additional data and steps required to improve seasonal influenza surveillance data were identified. These should now be developed, in collaboration with key stakeholders, into specific actions plans with assigned responsibilities to ensure an ongoing improvement process for seasonal influenza surveillance.

Ongoing, continuous improvement of surveillance should be the goal each season, in the knowledge that effective seasonal surveillance will also be an essential precursor to the surveillance of pandemic influenza.

Monitoring influenza through the National Incident Room during the 2007 season offered an excellent opportunity to conduct enhanced surveillance under conditions which were real and potentially serious but not an emergency. It enabled the current state of our surveillance systems to be assessed and opportunities for improvement to be identified. Areas for improvement in the collection and presentation of influenza during outbreaks, as well as the ability to investigate emerging changes in the behaviour of infectious disease were recognised.

Acknowledgements

The authors would like to acknowledge to efforts of the NIR surveillance team who worked to collect the information for this report: Gerard Fitzsimmons,

Julie Hall, Dougald Knuckey, Fiona Kong, Krissa O'Neil, Leslee Roberts, Paul Roche, Sam Ryan, Stefan Stirzaker.

The authors would also like to thank the National Influenza Centres, laboratories in Australia, South East Asia, New Zealand and Oceania for supplying influenza viruses to the Melbourne WHO Collaborating Centre for Reference and Research on Influenza. The Centre is supported by the Australian Government Department of Health and Ageing.

Author details

Rhonda Owen[1]
Ian G Barr[2]
Andrew Pengilley[3]
Conan Liu[1]
Bev Paterson[4]
Marlena Kaczmarek[1]

1. Surveillance Systems and Policy Section, Office of Health Protection, Australian Government Department of Health and Ageing, Canberra, Australian Capital Territory
2. Who Collaborating Centre for Reference and Research on Influenza, Parkville, Victoria
3. Medical Advisor, Office of Health Protection, Australian Government Department of Health and Ageing, Canberra, Australian Capital Territory
4. Food Safety and Surveillance Section, Office of Health Protection, Australian Government Department of Health and Ageing, Canberra, Australian Capital Territory

Corresponding author: Ms Rhonda Owen, Surveillance Policy and Systems Section, Office of Health Protection, Australian Government Department of Health and Ageing, MDP 14, GPO 9848, Canberra ACT 2601. Telephone: +61 2 6289 2709. Facsimile: +61 2 6289 2600. Email: rhonda.owen@health.gov.au

References

1. Nicholson KG. Clinical features of influenza. *Seminars in Respiratory Infections* 1992;7:26–37. In: *The Australian Immunisation Handbook*. 8th edn. Canberra; 2003.
2. World Health Organization. Epidemic and Pandemic Alert and Reponses. Influenza vaccine recommendations. Available from: http://www.who.int/csr/disease/influenza/vaccinerecommendations/en/index.html
3. Australian Government Department of Health and Ageing. Therapeutic Goods Administration. Australian Influenza Vaccine Committee. Available from: http://www.tga.gov.au/committee/aivc.htm
4. Australian Bureau of Statistics. Causes of deaths, Australia, 2005. ABS cat. no. 3303.3. Canberra: 2007.
5. Australian Institute of Health and Welfare. GRIM (General Record of Incidence of Mortality) Books. Australian Institute of Health and Welfare: Canberra.
6. Fonseca K, Tarrant M, Lam S, Li Y. Dual infection with influenza A and B viruses. *Pediatr Infect Dis J* 2002;21:795–796.
7. World Health Organization. Epidemic and Pandemic Alert and Responses. Influenza A(H1N1) virus resistance to oseltamivir. Available from: http://www.who.int/csr/disease/influenza/h1n1_table/en/index.html