Annual report

# Annual Report of the National Influenza Surveillance Scheme, 2009

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## Abstract

The 2009 influenza season was considered a significant season triggered by the April 2009 emergence of a novel influenza A virus prompting a World Health Organization (WHO) declaration of a public health emergency of international concern. The overall number of notifications in the Australian 2009 influenza season was the highest since national reporting to the National Notifiable Diseases Surveillance System (NNDSS) began in 2001, and substantially higher than in prior years. Over 59,000 notifications were reported to the NNDSS, almost ten times the five year mean and representing a crude notification rate of 272.1 per 100,000. Australia's first case of confirmed influenza A(H1N1)pdm09 was identified in early May 2009. By the end of 2009, there were 37,755 laboratory confirmed cases, including 5,085 hospitalisations and 188 deaths notified. Traditionally the age distribution of influenza notifications has rates highest in very young children and the elderly, however in 2009 with the predominance of the pandemic virus, notifications were highest in older children and younger adults. Although influenza can cause very severe and fatal illness, particularly in the elderly, the impact of influenza A(H1N1)pdm09 in younger healthy adults, Aboriginal and Torres Strait Islander peoples, pregnant women and people with existing medical co-morbidities was proportionally greater than normal seasonal outbreaks, even though the absolute number of such cases remained low.[1] The establishment of a number of surveillance systems during the pandemic enabled an enhanced assessment of the epidemiological, clinical and virological characteristics to inform public health responses.

## Introduction

Influenza is a common, highly infectious respiratory viral disease which causes annual epidemics that usually peak during the winter months in temperate climates. Infection is characterised by a sudden onset of fever, cough, sore throat, runny nose, headache, fatigue and myalgia,[2] and can cause mild to severe illness and even death. Severe disease is more likely with advanced age, lack of previous exposure to an antigenically related influenza virus, greater strain virulence, chronic underlying medical conditions, and in people who are immune-compromised or pregnant.[1, 3]

Influenza viruses are single-stranded RNA orthomyxoviruses that are classified antigenically as types A, B or C, however type C is not considered clinically important in human disease.[1] The surface of influenza viruses are coated with two glycoprotein antigens, haemagglutinin (H) and neuraminidase (N). Influenza A viruses can be classified into subtypes based on differences in these surface antigens, whereas influenza B cannot. Currently, influenza A(H1N1) and A(H3N2) are the circulating seasonal influenza A virus subtypes and there are two type B seasonal virus lineages, B/Victoria and B/Yamagata.

Both influenza A and B viruses undergo continuous changes in their surface antigens and can change in two different ways. 'Antigenic drift', can occur in both influenza A and B viruses and is associated with relatively minor changes in the genes of the virus as it replicates producing viruses that are closely related but over time result in viruses that are antigenically different. This viral drift is the reason for annual seasonal

epidemics and the need to frequently review the composition of influenza vaccines. The second type of change is known as 'antigenic shift', which is an abrupt major change resulting in new haemagglutinin and/or new haemagglutinin and neuraminidase antigens that typically emerges from an animal population.[4] This viral shift occurs occasionally and unpredictably, and can cause a pandemic.

A pandemic is associated with the emergence and global spread of a novel virus which most people do not have immunity to and typically originate from animal influenza viruses. Some aspects of influenza pandemics can appear similar to seasonal influenza, whilst other characteristics may be quite different.[5] For both seasonal and pandemic influenza, the total number of people infected and the proportion who have severe illness can vary, however, the impact or severity tends to be higher in pandemics because of the potentially greater number of people in a population getting infected. So even if the proportion of those infected that go on to develop severe disease is small, the total number of severe cases can be quite large.

## Influenza A(H1N1)pdm09 emergence

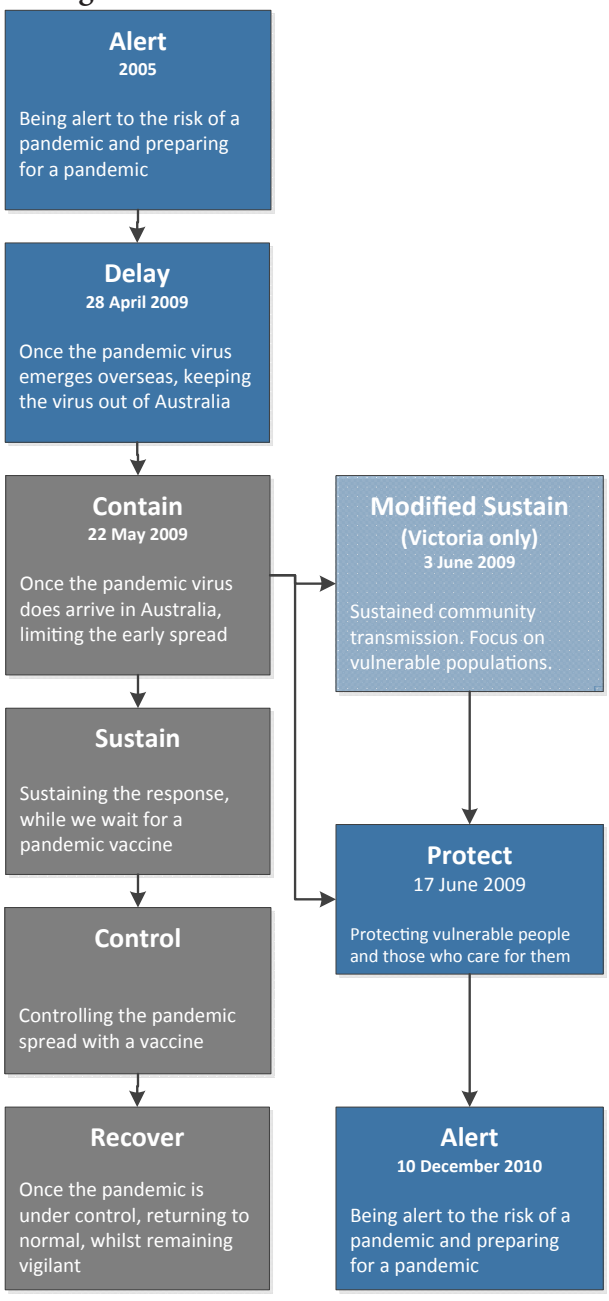
In March 2009, cases of a novel influenza virus began to emerge in Mexico and the United States of America. By mid-April 2009, atypical cases and clusters of severe pneumonia were occurring mainly among previously healthy young people in different areas of Mexico.[6] These cases were subsequently determined to be infected with a novel influenza A(H1N1) virus strain that had not circulated previously in humans and contained genetic material suggestive of swine origin.[6, 7] In late April 2009, the (WHO) determined that the situation constituted 'a public health emergency of international concern' under the International Health Regulations (2005).

Based on community spread in at least one other country in a different WHO region to where the epidemic began, and following the identification of nearly 30,000 confirmed cases in 74 countries, including Australia, the WHO declared the viruses spread a pandemic on 11 June 2009.[8] This was considered the first influenza pandemic in more than 40 years.

## National actions during the pandemic phases

Australia's response to the 2009 pandemic was guided by the *Australian Health Management Plan for Pandemic Influenza 2008* (AHMPPI) (Figure 1),[9] with the key surveillance activities by pandemic phase outlined in Appendix 1.

**Figure 1: Pandemic phases during the 2009 pandemic, based on the Australian Health Management Plan for Pandemic Influenza[9, 11]**



Australia had been in the pandemic "ALERT" phase since 2005 following the emergence of the avian influenza A(H5N1) virus infection in humans. Following the identification of the first few cases of influenza A(H1N1)pdm09 overseas and the WHO declaration of a public health emergency of international concern, Australia entered the "DELAY" phase on 28 April 2009 with the objective of delaying the entry of the virus to Australia through the early identification and management of cases and contacts. Measures during this phase included declaring the new virus a quarantinable disease under the *Quarantine Act 1908*; border surveillance measures; public messaging; and intensive case and contact management activities. Clinicians were requested to report suspected cases to jurisdictional health departments for follow-up and contact tracing, and antiviral medications were recommended for the early treatment of all confirmed cases and post-exposure prophylaxis offered to identified close contacts. Australia's first case of confirmed influenza A(H1N1)pdm09 was identified on 7 May 2009.

Australia moved to the pandemic "CONTAIN" phase on 22 May 2009, when clusters of cases began to emerge indicating community level transmission was occurring. The objective of the "CONTAIN" phase was to reduce the spread in the community, limit the number of cases and support the health system while waiting for a pandemic vaccine to become available. In the early stages of the pandemic in Australia, almost all of the activity was experienced in Victoria, thereby overwhelming the capacity of their public health responses. Due to the overwhelming case load being experienced in Victoria and the apparent moderate severity of the disease a modification to the AHMPPI's pandemic "SUSTAIN" phase occurred to ensure proportionate response strategies to the disease. On 3 June 2009, Victoria moved to the revised "MODIFIED SUSTAIN" pandemic phase.[10] All other jurisdictions remained in the "CONTAIN" phase.

When the WHO declared a pandemic in mid-June 2009, globally the pandemic was being described as mild in most but severe in some and moderate overall. In recognition that the virus was mild in most people and that a greater focus was required for managing those people more vulnerable to severe disease, Australia developed and implemented a new pandemic phase known as "PROTECT" on 17 June 2009. This phase focussed on identifying and treating those most vulnerable to severe disease. Testing during this phase was limited to those in vulnerable groups; people with moderate to severe disease; and outbreaks in institutional settings.

## The National Influenza Surveillance Scheme in 2009

The National Influenza Surveillance Scheme is coordinated by the Australian Government Department of Health. The surveillance scheme began in 1994 with the objective of essentially characterising influenza activity to inform

**Table 1: Pandemic phase changes and duration, 2009[9]**

| Phase | Date commenced | Date ceased | Duration |
|---|---|---|---|
| DELAY | 28 April 2009 | 22 May 2009 | 3 weeks |
| CONTAIN | 22 May 2009 | 17 June 2009 | 4 weeks |
| MODIFIED SUSTAIN (Victoria only) | 3 June 2009 | 17 June 2009 | 2 weeks |
| PROTECT | 17 June 2009 | 1 December 2010 | 76 weeks |
| ALERT | 1 December 2010 | Ongoing | Ongoing |

public health prevention and control activities.[12] The Seasonal Influenza Surveillance Strategy Working Group (currently known as the National Influenza Surveillance Committee (NISC)), a subcommittee of the Communicable Diseases Network Australia (CDNA), is responsible for monitoring and enhancing these systems to ensure their effectiveness in understanding influenza severity, transmission and virology.

In 2009, a variety of surveillance methods were used to monitor the incidence and severity of influenza in Australia. Severity of illness was measured through hospitalisation, including intensive care unit (ICU) admissions, as well as associated mortality. Disease incidence was monitored through laboratory confirmed notifications, emergency department presentations for influenza-like illness (ILI), sentinel general practitioner ILI consultations, absenteeism from a large national employer, health call-centre data and a community online ILI survey surveillance system. Whilst surveillance activities for influenza occur traditionally throughout the year with a focus on the peak winter epidemic activity between May and October; in 2009, due to the emergence of the pandemic influenza virus surveillance activities were heightened from late April 2009.

Prior to the 2009 pandemic, the routine surveillance scheme systems that were in place to monitor activity in a relatively timely manner throughout the season included:

- notifications of laboratory confirmed influenza required by legislation in all states and territories, and notified to the National Notifiable Diseases Surveillance System (NNDSS);
- absenteeism data from a national employer;
- consultation rates for ILI identified by sentinel general practitioners;
- consultation rates for ILI identified by sentinel hospital emergency departments (EDs) in Western Australia and New South Wales;
- paediatric hospitalisations associated with severe complications from influenza;
- mortality data from the New South Wales Registry of Births, Deaths and Marriages (BDM);
- subtype and strain data of circulating influenza viruses provided by the WHO Collaborating Centre for Reference and Research on Influenza; and
- respiratory virology testing data for influenza from the New South Wales sentinel laboratory network and the Victorian Infectious Disease Reference Laboratory.

The surveillance of influenza during 2009 was based on the already existing surveillance scheme systems, as well as either enhancements to existing systems; adoption or adaptation of other systems; or the development of additional systems in order to provide a comprehensive relatively timely assessment of the 2009 pandemic virus emergence to guide decision making. In 2009, these systems included:

- enhanced notification data of influenza A(H1N1)pdm09 confirmed cases reported to the national web-based surveillance system, by jurisdictional health departments (NetEpi);
- expansion of the data on respiratory virology testing for influenza from the Western Australian National Influenza Centre and a subset of sentinel general practitioner ILI consultations;
- intensive care unit influenza A(H1N1)pdm09 admissions (ANZICS);
- hospital admissions for influenza A(H1N1) pdm09 by public hospitals in Queensland (EpiLog);
- sentinel surveillance for acute respiratory disease requiring hospitalisation, including influenza (FluCAN); and

- community-level syndromic surveillance through a national health call centre network (NHCCN) and an online survey of voluntary participants (FluTracking).

Additionally, mortality data from the ABS National Registry of Births, Deaths and Marriages and hospitalisation data based on the Australian Institute of Health and Welfare's (AIHW) National Hospital Morbidity Database (NHMD) were retrospectively analysed as part of this report to inform the assessment of the burden of influenza activity in 2009.

During seasonal influenza epidemics many of the surveillance systems used maybe sentinel, less sensitive and less specific as it is not critical to identify every case, rather, to ascertain an indication of the burden on the community and the impact on health sector resources.[9]

Whereas following the emergence of a novel influenza virus it is crucial, at least in the initial pandemic phases of "DELAY" and "CONTAIN", to ensure a higher degree of case ascertainment through surveillance enhancements to enable the identification and management of all cases, as well as to understand the epidemiology of the new virus. For instance during seasonal influenza epidemics notifications of laboratory confirmed influenza are based and influenced on the discretional degree of patient testing for influenza by health care practitioners, whereas in the early phases a pandemic there is a strong focus on detection and so patients presenting with an acute febrile respiratory disease and associated risk factors, for example travel, would be targeted for testing.[13]

During the 2009 pandemic, the surveillance response, including case definitions (Appendix 2), testing protocols (Appendix 3) and reporting requirements were overseen by the CDNA. The CDNA developed and reviewed the case definition for reporting cases which was used to describe the degree of certainty of infection with influenza A(H1N1)pdm09. The case definition initially focussed on the identification of cases coming into Australia with illness onsets since 15 April 2009 and recent travel history to affected regions; and was later adapted to recognise the shift to Australian areas with community transmission. Throughout all phases, the case definition required the presence of fever in the absence of clear contact with confirmed cases.[14] The case definitions were used to guide the clinical management of cases, disease surveillance, testing protocols and subsequent public health decisions.

This report presents findings from a retrospective analysis of the data collected under the National Influenza Surveillance Scheme during 2009, as well as complimentary data sources that became available following the 2009 season. Data from all of these systems were utilised to describe the epidemiological characteristics of the 2009 season, with a particular focus on the confirmed case notification data reported through the NNDSS and NetEpi by jurisdictional health departments. Appendix 4 provides a summary of the influenza notification data sources during 2009. The report has been split into two main themes: a summary overall influenza activity in 2009 and also a specific focus on pandemic influenza activity.

## Surveillance and Data Analysis Methods

National surveillance of influenza is conducted by the Australian Government Department of Health. Data are collated from a range of surveillance systems to describe influenza activity and spread; to describe the impact and burden of disease; and to determine which influenza strains are circulating to inform influenza vaccine composition. These data are collected and collated by state and territory health departments, government agencies and research organisations.

### Laboratory confirmed influenza notifications

Laboratory confirmed influenza is a nationally notifiable disease under legislation in all Australian states and territories. The disease has been notifiable in all states and territories since 2001, except the ACT where it became notifiable in 2004 and South Australia where it became notifiable in 2008. Prior to influenza becoming a notifiable condition in these two jurisdictions,

laboratory reports of influenza positive results were generally provided to the NNDSS. [15, 16] The *National Health Security Act 2007* provides the legislative basis for the national notification of certain communicable diseases and authorises the exchange of health information from State and Territory Governments to the Australian Government for national collation. These data are routinely reported to the NNDSS; however during 2009, notifications of pandemic influenza cases were reported through a web-based outbreak reporting system, NetEpi, which enabled the capture of additional data fields. Notifications reported through NetEpi were retrospectively reported to NNDSS. Appendix 4 provides a summary of the types and sources of data available for notified cases throughout the 2009 influenza season.

### National Notifiable Diseases Surveillance System

The 'core' data elements normally collected and reported, with varying completeness, on influenza cases include:

- Case demographics – eg. age, sex, Indigenous status, jurisdiction of residence;
- Virus characterisation – eg. laboratory diagnosis method, virus type, subtype, strain (where available);
- Disease outcome – whether the patient died of the notifiable disease (where available); and
- Dates – eg. symptom onset, specimen collection, notification date.

In this report a summary analysis of all laboratory confirmed influenza notifications data reported during 2009, including both seasonal and pandemic influenza cases, is presented. These data were analysed by the date of diagnosis, a derived substitute for date of symptom onset and based on the earliest of specimen collection, notification or notification received dates. During the pandemic, enhanced notification data on influenza A(H1N1)pdm09 cases were initially entered into NetEpi and then a subset of the data fields collected for these cases was either simultaneously or retrospectively transmitted to NNDSS from jurisdictional health departments.

### NetEpi case reporting system

Cases of pandemic influenza were reported to state and territory health departments by general practitioners, hospitals and laboratories based on the CDNA case definition applicable at the time of case identification (see Appendix 2). State and territory health departments reported cases to the Australian Government Department of Health using NetEpi, a web-based outbreak case reporting system that was developed by New South Wales Health. The system provides a platform for enhanced data to be entered by jurisdictions using a common form and enabling real-time collation at a national level. These data were then exported to a Microsoft Excel spreadsheet for analysis.

The 'enhanced' data elements that were collected through NetEpi were in addition to those fields routinely collected in NNDSS. These enhanced fields included:

- Travel history;
- Symptoms;
- Risk factors for disease;
- Hospitalisation status; and
- Antiviral administration.

At the beginning of the pandemic, data were entered manually by jurisdictional health departments into NetEpi. However, when case numbers became too large for this to be feasible data importation methods from some alternate jurisdictional surveillance systems were implemented. This occurred at different time points during the pandemic and had an impact on the completeness and interpretability of the data fields provided in the national NetEpi enhanced data collection.

Whilst most jurisdictions used the national instance of NetEpi, Queensland and New South Wales used their own instances, which for the most part was consistent with the national form, and these data were then imported into the national NetEpi instance. Additionally New South Wales used NetEpi to maintain all notifications of laboratory confirmed influenza during 2009 and retrospectively transmitted their core NNDSS data into NNDSS. The Northern Territory used the national instance of NetEpi to provide enhanced data on confirmed cases that had been admitted to hospital or had died; core data on all other case were maintained in NNDSS.

On 6 July 2009, Queensland ceased reporting their data into NetEpi and instead a reduced dataset was reported to the NNDSS, however some enhanced case data continued to be captured for patients admitted to public and major private hospitals through the Queensland hospitalisation data collection systems, EpiLog and a purpose-designed Microsoft Excel spreadsheet.[17] EpiLog is linked to the Hospital Based Clinical Information System used in Queensland public hospitals, which stores data relating to patient demographics and hospital stay with a unique patient identifier. Additionally the same data elements were also collected from major private hospitals in Queensland using a purpose-designed Microsoft Excel spreadsheet. The data elements collected included risk factors, antiviral administration, duration of hospitalisation, ICU and ventilation requirements and Indigenous status.

In Victoria, all confirmed cases of influenza A(H1N1)pdm09 notified during the DELAY and CONTAIN phases, up to 4 June 2009, were followed up to ascertain Indigenous status, travel history, risk factors, antiviral administration and hospitalisation status. These data were reported to NetEpi.[18] Following Victoria's transition to MODIFIED SUSTAIN, limited case followup occurred and therefore enhanced case data collection and completeness was reduced. Data on Victorian ICU admissions were captured through the Australian and New Zealand Intensive Care Society (ANZICS) surveillance system.

Throughout the pandemic, updates to the NetEpi data collection form were made as case definitions and data requirements changed. Analyses of these data are based on confirmed influenza A(H1N1)pdm09 cases and using clinical onset date; however, where this date is not available the earliest of cough onset, fever onset, fever self-report onset, specimen, and notification received date is used.

Following the pandemic, states and territories reviewed the data provided to the Australian Government Department of Health to ensure completeness and accuracy, especially of priority and potentially sensitive fields, for example pandemic influenza associated deaths. Data from the NetEpi database were converted to Excel and cleaned to ensure consistency in the interpretation of the variables collected. Clarification was also sought in terms of the interpretation of the data provided against the responses that had been defined in the national form and based on the data dictionary that was approved by CDNA. Additionally for some variables of interest multiple fields or response sets captured the information, this was due to some jurisdictions making some local adaptations to the case form or reverting to alternate surveillance systems, especially later in the pandemic; as well, revisions to the national data collection form throughout the pandemic. To accommodate this in the analysis, a composite variable was created with contents based on the combining of the existing entries across the multiple fields. For example in identifying the Indigenous status of a case there were four different fields where this could be reported in the national form and the field was not completed consistently.

In this report an analysis of the enhanced data reported to NetEpi for laboratory confirmed influenza A(H1N1)pdm09 notifications during 2009, is presented. As not all pandemic influenza cases were followed up or a variety of surveillance approaches were used to ascertain enhanced case information these data represent

a subset of the notified pandemic influenza cases. Additionally, following the commencement of the PROTECT phase, the data are known to be biased towards those with moderate to severe illness due to testing recommendations rather than cases at the broader community level.

These data were analysed by the date of disease onset, or where this date was not available, the earliest of specimen collection, cough onset, temperature onset, fever self-report onset and notification received date was used.

## Community influenza-like illness surveillance

In 2009, ILI surveillance was used as a proxy measure for trends in influenza activity in the community. Absenteeism data had been collected historically for several years and during 2009 two additional surveillance data sources were adopted to further understand community ILI activity: the National Health Call Centre Network and FluTracking.

### Absenteeism data

During 2009, a major nationwide employer, representing around 33,000 employees, provided weekly absenteeism data. Absenteeism data from this system was defined as an absence recorded as 'sick-leave' for three or more consecutive days, and are presented as a rate per 100 employees per week.

### The National Health Call Centre Network

The National Health Call Centre Network (NHCCN) provides free health triage advice and information services to the public by registered nurses, and commenced delivering services in all jurisdictions except Queensland and Victoria in July 2007. Data from this network have been provided on a daily basis to the Commonwealth since mid-July 2008. All data provided are de-identified and include information regarding the caller's jurisdiction of residence, age, Indigenous status, presenting issue, patient guideline (or diagnosis) and the final triage disposition. A subset of the patient guidelines were used to define ILI for analysis.

### FluTracking

FluTracking is a national weekly online survey of ILI that commenced on 4 May 2009 and was completed by over eight thousand participating community members each week. [19] During 2009, participants were requested to complete a weekly online survey which asked whether they had experienced fever or cough and how many days they had been absent for work or normal duties because of these symptoms.

### Sentinel general practitioner influenza-like illness surveillance

Nationally, three schemes were used in 2009 to monitor rates of ILI consultations at sentinel general practices: the Australian Sentinel Practices Research Network (ASPREN), which collected data at a national level from approximately one hundred general practitioners from all jurisdictions except the Northern Territory; the Victorian Infectious Diseases Reference Laboratory General Practice Sentinel Surveillance Program (VIDRL GPSS), and the Northern Territory Tropical Influenza Surveillance Scheme (NTTISS). Both ASPREN and the NTTISS report ILI rates throughout the year, whilst the VIDRL GPSS reported from early April 2009. The case definition for ILI used by these schemes is: presentation with fever, cough and fatigue.

## Emergency department influenza-like illness surveillance

Like GP ILI surveillance, ED surveillance is an indicator of the ILI burden in the community, severity of a season and may capture groups in the community that are under-represented in GP surveillance, especially the very young.[20] Emergency department surveillance of ILI presentations was collected from 52 emergency departments across New South Wales[21,22] and nine public hospital emergency departments in Perth, Western Australia. These data

were provided to the Australian Government Department of Health on a weekly basis from the New South Wales and the Western Australian health departments.

### New South Wales emergency department influenza-like illness surveillance

In New South Wales the incidence of emergency department presentations assigned a diagnosis of influenza or ILI are assessed based on both SNOMED and ICD10 diagnosis codes relevant for influenza and ILI.[23]

### Western Australia emergency department influenza-like illness surveillance

In Western Australia, emergency department respiratory virus cases are determined from presentations that have been coded based on ICD-10 diagnosis codes as upper respiratory tract infection (J06.9) and viraemia (B34.9), which best correlates with other relevant data sources for representing respiratory viral illness presentations.[24]

## Hospital influenza surveillance

Surveillance of laboratory confirmed influenza hospitalisations is used to provide an understanding of disease severity and to identify populations at risk of severe disease. Nationally, three sources of influenza hospitalisation data were available during 2009: paediatric severe complications, intensive care unit admissions and sentinel adult acute respiratory hospitalisations. The latter two sources were set up during the pandemic and a number of systems were also set up in jurisdictional health departments and data from these systems were used to inform the NetEpi enhanced surveillance dataset.

In addition to these hospitalisation data sources, the Australian Government Department of Health's Admitted Patient Care Dataset, which were available following 2009, were used as a complimentary data source to inform analyses of hospitalisations retrospectively.

### Influenza Complications Alert Network

The Influenza Complications Alert Network (FluCAN) represented a network of eight public acute care hospitals that were established in mid-2009 to collect data on laboratory confirmed influenza or community acquired pneumonia (with or without influenza) hospitalisations in adults, including intensive care unit (ICU) admissions. Patients over 18 years of age were recruited within 48 hours of admission through active surveillance of emergency departments, infection control, pathology and radiology results, and medical admissions by designated research staff at each hospital site. Data on patients admitted to these sites were collected between 1 July and 30 November 2009. The eight hospital sites in six Australian jurisdictions in 2009 represented 10% of all Australian hospitals with over 200 beds (n=79) and overall represented 6.4% of national bed capacity (n=54,338). [30]

### Australia and New Zealand Intensive Care Society intensive care unit admissions

Between 1 June and 31 August 2009, the Australian and New Zealand Intensive Care Society (ANZICS) collected data on patients with confirmed pandemic influenza infection who were admitted to an ICU. Data were collected using electronic case report forms from all ICUs in Australia and all diagnoses were confirmed with the relevant state or territory's health department. [31] This system was the primary ICU surveillance system for ICU admitted cases for Victoria and the Australian Capital Territory.

### Paediatric severe complications hospitalisation data

The Australian Paediatric Surveillance Unit collected data on children aged less than 15 years who were admitted to hospital with severe complications from influenza. The data were collected from paediatricians and other child health clinicians using a monthly report card as a minimum, with reporters requested to report cases that meet the case definition criteria as soon as possible and complete the case report

form questionnaire. The case definition in 2009 required reporting of any child aged less than 15 years with laboratory confirmed influenza admitted to hospital with a defined complication, for example pneumonia, requirement for ventilation and encephalitis.[32,33] In 2009, surveillance was conducted from May to September.

### Admitted Patient Care Dataset

The Australian Government Department of Health's Admitted Patient Care Dataset[25] (APC Dataset) is based on the AIHW's NHMD[26]. The data are reported in accordance with the National Minimum Data Set (NMDS) for admitted patient care[27] and are based on the counting unit of 'separation'. Where separation refers to the episode of admitted patient care, which can be a total hospital stay (from admission to discharge, transfer or death) or a portion of a hospital stay beginning or ending in a change of care type.

For comparison between the 2009 season and epidemics of seasonal influenza in previous years, hospitalisation separations for the calendar years 2004 to 2009 have been included in this report. The following, 10th revision, Australian Modification (ICD-10-AM)[28], codes were utilised: J09 (Influenza due to identified avian influenza virus); J10 (Influenza due to other identified influenza virus); and J11 (Influenza, virus not identified). During the 2009 pandemic, the ICD-10-AM 6th Edition code J09 (Influenza due to identified avian influenza virus) was used by hospital coders to report influenza A (H1N1)pdm09 cases. As the J09 code descriptor in 2009 related to avian influenza, analyses of separations coded as identified influenza virus (ie. J09 and J10) have not been separated as coding of 'swine' influenza cases in 2009 are not likely to have been reliably allocated to J09. This assumption is supported by the notification data.

Analysis of the total number of hospital separations includes separations where an influenza code (J09, J10, J11) has been clinically coded as either a principal or an additional diagnosis[I]. Whilst a patient's hospitalisation can have multiple additional diagnoses recorded within the dataset, the proportion of separations that had more than one J09-J11 code recorded is likely to be negligible. Therefore, comparisons between the numbers of influenza associated separations and total hospital separations to understand the impact of influenza on hospital separations are considered appropriate.

It is not known the degree that hospitalisation data based on these three ICD-10-AM codes underestimates the true burden of influenza hospitalisation, due in particular to limited testing and attribution to influenza as one of the diagnoses in coding of hospitalisation episodes. Additionally, due to differences in testing strategies across the period analysed, especially during the 2009 season, comparisons within and across seasons are difficult and not necessarily comparable. For example, in 2009, all paediatric cases presenting with ILI admitted to hospital were tested for influenza A(H1N1)pdm09, however this did not apply to all hospitalised adults potentially resulting in an underestimation of the adult caseload by 2.7 times (90% range 1.9-4.3)[29] as well as potential changes over the time period in sensitivity of diagnosis, threshold for diagnostic testing, and case ascertainment (particularly during previous influenza seasons).

## Mortality data

Data on influenza associated mortality provides an additional indication of severity. During 2009, mortality surveillance data was collected from notifications data as well as death certificate data from New South Wales. National death certificate data were able to be analysed retrospectively.

### Notifications of influenza associated deaths

The national notifications data on laboratory confirmed influenza, within both NNDSS

I A principal diagnosis refers to the diagnosis established to be chiefly responsible for the separation and an additional diagnosis refers to the condition or complaint either coexisting with the principal diagnosis or arising during the episode of admitted patient care.

and NetEpi surveillance systems, enables the reporting of the mortality status of the case. Completeness of this field is reliant on the follow up of cases to determine the outcome of their infection and most likely do not represent the true mortality impact associated with this disease.

### New South Wales Registry of Births, Deaths and Marriages data

In 2009, death certificate data from the New South Wales Registry of Births, Deaths and Marriages provided a timely estimate of the number of deaths from pneumonia and influenza in New South Wales.[24] Death certificate data include information describing the disease or condition directly leading to a death, as well as any antecedent causes, co-morbid conditions, or other significant contributing conditions. The data submitted are scanned for the keywords 'pneumonia' and 'influenza' to generate a weekly count and population rate of deaths that mention pneumonia or influenza on the death certificate.

### National Registry of Births, Deaths and Marriages data

National influenza associated mortality were estimated based on mortality data compiled by the ABS from information provided by state and territory Registrars of Births, Deaths and Marriages. The 2009 Causes of Death Data were released by the ABS in May 2011, and therefore were not available to inform the assessment of influenza activity during 2009. ABS mortality data are coded using the 10th revision of the *International Classification of Diseases and Related Health Problems* (ICD-10).[34] To assess deaths associated with influenza infection a combination of ICD-10 codes were used, specifically J09-J18, which incorporates deaths of pneumonia; a well-known marker of seasonal and pandemic influenza activity.

## Laboratory Surveillance

Laboratory surveillance data were used to monitor testing rates for influenza; changes to the virus and antiviral susceptibility. Throughout 2009, a number of sources were used to inform laboratory testing rates and subtyping results, as well as the proportion of circulating respiratory pathogens that were attributable to influenza.

### WHO Collaborating Centre for Reference and Research on Influenza[35]

The WHO Collaborating Centre for Reference and Research on Influenza (WHOCC) in Melbourne is part of the WHO Global Influenza Surveillance Network (WHO GISN). The Centre is one of five Collaborating Centres in the world established to monitor the frequent changes in influenza viruses and inform the composition of influenza vaccines. The Melbourne WHOCC analyses viruses received from Australia and from laboratories throughout the Asia-Pacific region.

All virus isolates are analysed antigenically, with a subset undergoing further genetic analysis of the haemagglutinin and neuraminidase genes; especially those viruses exhibiting evidence of antigenic drift following antigenic characterisation. Serological analyses were also performed to monitor the extent that antigenic changes in circulating influenza viruses are able to be inhibited by antibodies produced by subjects who have been immunised with current influenza vaccines. These data were used to inform the development of candidate viruses for inclusion in the pandemic vaccine as well as seasonal vaccines.

Antiviral susceptibility testing of influenza viruses is also performed by the WHOCC to detect the emergence of drug-resistance influenza strains that could present treatment challenges. In 2009, influenza viruses were tested for their sensitivity to the neuraminidase inhibitors oseltamivir (Tamiflu) and zanamivir (Relenza) using the neuraminidase inhibition (NAI) assay. Viruses were considered to be resistant when the concentration of drug required to inhibit 50% of neuraminidase activity is greater than 200nM. Additionally, influenza A(H1N1)pdm09 and seasonal A(H1N1) viruses were also screened by pyrosequencing to detect the mutation from histidine to tyrosine at position 275 (H275Y) in the N1 neuraminidase that confers resistance to

oseltamivir. Viruses selected for pyrosequencing include those that exhibited resistance by NAI assay, as well as original clinical specimens that did not yield a virus isolate when cultured.

Summary antigenic and genetic characterisation data as well as serological and antiviral susceptibility data from the analysis of Australian viruses performed by the WHOCC have been presented in this report.

### National Influenza Centres

From mid-2009, Australia's three National Influenza Centres, the Institute of Clinical Pathology and Medical Research in New South Wales, PathWest Laboratory Medicine in Western Australia and the Victorian Infectious Diseases Reference Laboratory (VIDRL), reported influenza testing information to the Australian Government Department of Health on a weekly basis. The data reported included the number and proportion of influenza tests positive for influenza, and of the tests positive for influenza a breakdown based on influenza type and subtype.

### Serosurveillance

A national serosurvey, using plasma samples from Australian Red Cross Blood Service healthy adult blood donors aged 16 years and over, was conducted to understand the proportion of the population with demonstrated immunity to the influenza A(H1N1)pdm09 virus was commissioned by the Department of Health and Ageing in 2009. Antibodies to influenza A(H1N1)pdm09 were assessed as an indicator of immunity, primarily using haemagglutination inhibition assays, with a threshold antibody titre of 40 considered a marker of recent infection. The serosurvey was conducted over a series of time points before and after the epidemic that occurred in the winter of 2009, and taking into consideration the monovalent pandemic vaccine rollout. The study was conducted by McVernon *et al.*[36]

### Data analysis methods

Notification rates were calculated using the estimated 2009 mid-year population data published by the ABS. Population data used for these analyses included: estimated resident population, population by sex and age group, and Aboriginal or Torres Strait Islander population tables.[37, 38]

This report uses three approaches when comparing rates between population groups:

- Age-specific rates: are rates relating specifically to a certain age group. For each age group they have been calculated as the number of events in that age group divided by the mid-year estimated resident population for that age group.

- Crude rates: the number of events in a year divided by the total mid-year estimated resident population.

- Age-standardised rates: is a method of adjustment to allow for the effect of variation in the population age structure when comparing event rates for different years, locations or sub-populations. This report has used the 'direct' standardisation method, which applies the age-specific rates for a particular year to a standard population. This produces an estimate of the event rate which would have prevailed in the standard population if it had experienced the age-specific event rates in the year under study. The standard population used was the 2001 mid-year Australian population.[39]

In identifying the Indigenous status of a case there were four different fields where this could be reported in the national form and the field was not completed consistently. A combined single Indigenous status field was created. Indigenous cases were based on the aggregation of cases reported as 'Indigenous – Aboriginal but not Torres Strait Islander origin', 'Indigenous – Torres Strait Islander but not Aboriginal origin', 'Indigenous – Aboriginal and Torres Strait Islander origin'. The Indigenous status of cases,

including 'Not Indigenous', was only reported for 61.8% of cases and completeness was variable by jurisdiction and data source. In terms of analysis of influenza in the Aboriginal and Torres Strait Islander population[II] Indigenous Australian population, this likely represents the minimum number of Indigenous Australians affected by influenza during 2009. In comparing of the rates of influenza between Indigenous and non-Indigenous Australians, cases reported as 'non-Indigenous', 'unknown' or 'blank 'were aggregated to non-Indigenous Australians. Calculations of rates were based on the ABS estimates of Indigenous populations in 2009 as determined following the 2011 census. It should be noted that the ABS reported a very large increase in the estimates of Australian Indigenous persons between the 2006 and 2011 census (21%).[40]

The expected population prevalence of pregnant women, both in the total Australian population and among females of childbearing age (primarily defined as 15 to 44 years), was estimated using the ABS age-specific fertility rates and total fertility rates for 2009[41]. These estimates are based on the number of live births during the calendar year, according to the age of the mother, per 1,000 of the female estimated resident population. It should be noted that this estimate therefore does not take into account miscarriages, abortions, still births or premature deliveries, and as such is an approximation of the prevalence of pregnant women.

Rates of underlying medical conditions were calculated using data collected by the ABS through the 2007/08 National Health Survey[42] and the concurrent National Aboriginal and Torres Strait Islander Health Survey on the cumulative prevalence of at least one at-risk medical condition for which the influenza vaccine was recommended[43] and where there were corresponding data.

Valid data into a field was considered to include 'true', 'false', 'unknown' (or equivalent responses) and for New South Wales cases, not answered which in the dataset was reported as 'none'. The denominator used for interpretation of the enhanced dataset was based on the provision of valid data into any of the relevant fields related to the aspect of the analysis for a case (eg. clinical presentation, hospitalisation, risk factors). If no data were provided across all the identified relevant fields (ie. the fields were blank), these cases were not included in the denominator. The denominator completeness for each field and area of analysis was highly variable and not representative of all cases notified either geographically, by pandemic phase or health care setting. This approach has been used in attempt to more accurately represent the proportion of cases with the factor of interest that is being analysed.

## Results

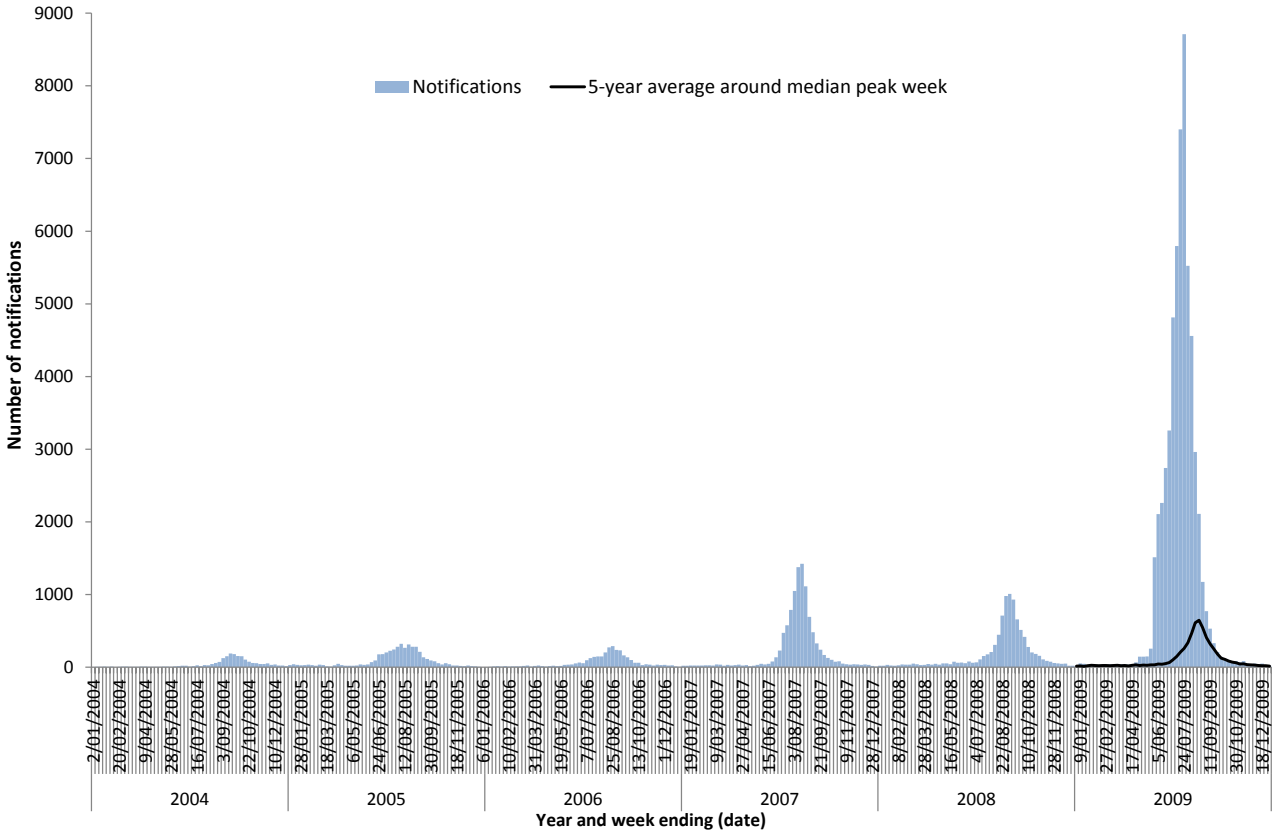
Generally the influenza season in Australia commences between late June and early July, peaking in mid-August and lasting on average approximately 17 weeks (Figure 2). In comparison, the 2009 influenza season began in early May following the emergence of the pandemic influenza virus overseas in late April 2009 and the first case detection in Australia on 8 May 2009. Notifications peaked in late July, with the season lasting approximately 22 weeks. Much of the initial rapid rise in late May is likely due to enhanced case ascertainment activities, especially testing of patients presenting with ILI in order to detect and respond to the initial emergence of pandemic influenza cases, and therefore detected background seasonal influenza activity prior to the increase associated with pandemic cases.

### Notified cases of laboratory confirmed influenza in Australia, 2009

Influenza activity in Australia between January and April 2009 was at baseline levels with around 35 notifications reported each week. Notifications started to increase in May, with a sharp distinct increase in the week ending 29 May 2009. Nationally notifications peaked at 8,711 towards the end of July (week ending 24 July 2009) and returned to inter-seasonal lev-

II From herein the term 'Indigenous Australians' will be used to refer to Aboriginal and Torres Strait Islander people in order to assist readability.

**Figure 2: Notifications of laboratory confirmed influenza, Australia, 2004 to 2009, by week of diagnosis**




Source: NNDSS

els by mid-October (Figure 4). The total number of notifications reported to the NNDSS in 2009 was 59,026, which was almost ten times the 5 year mean and represented a crude notification rate of 272.1 per 100,000 population (Figure 2 and Table 2). However, as most people with influenza do not seek medical attention or require testing, influenza notifications generally represent only a small proportion of all true cases of infection.

It should be noted that in addition to the emergence of the pandemic, and the associated increase in case numbers, testing patterns throughout 2009 were dependant on the phase of the pandemic and associated recommendations, and are therefore likely to underestimate the true incidence of the disease, especially during the later phases of the pandemic. Additionally, historic comparisons of notification data are problematic as it is difficult to know the proportion of all influenza cases that notifications data represent, especially as they are influenced by health care seeking behaviour and testing rates.

## Geographic spread

In 2009, the majority of laboratory confirmed influenza notifications occurred in Queensland (31%), followed by New South Wales (22%), South Australia (18%) and Victoria (12%). However, crude notification rates varied across the country, with the Northern Territory (876 cases per 100,000), South Australia (669 cases per 100,000) and the Australian Capital Territory (357 per 100,000) substantially above the national rate (272 per 100,000 population) (Table 2). Analysis by Australian Statistical Geography Standard Statistical Area Level 3 of the cumulative influenza notification rates in 2009 shows that rates were highest throughout the northern and central areas of Australia (Figure 3).

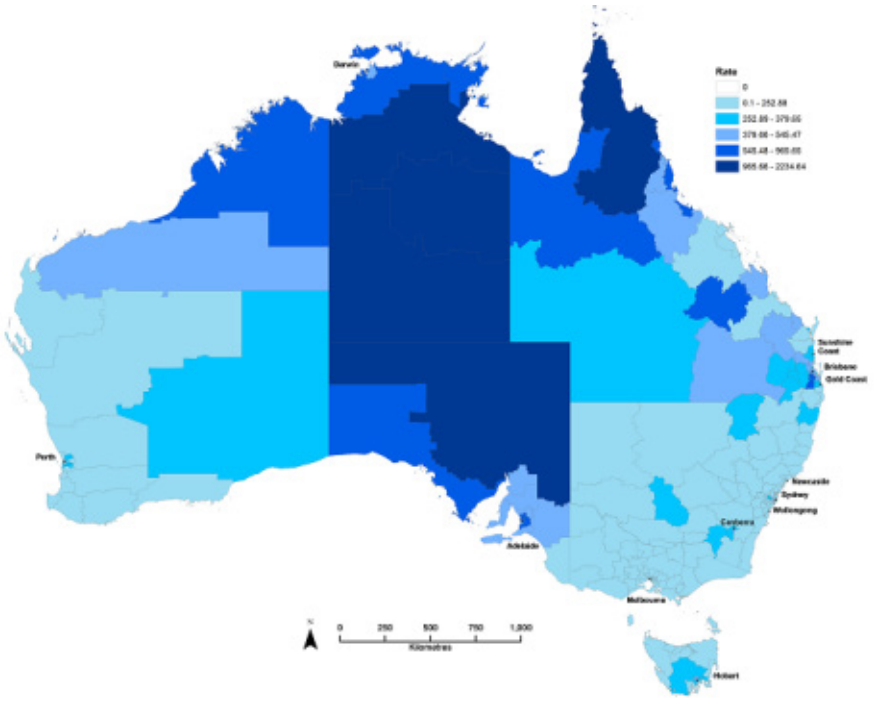
The timing and size of influenza activity increases and peaks varied across states and territories in 2009 (Figure 4). For example, Victoria experienced an overwhelming number of influenza cases from late May, much earlier

**Table 2: Notifications and rates of laboratory confirmed influenza, Australia, 2009, by state or territory**

| State or territory | Total notifications | Percentage of total (%) | Notification rate (per 100,000 population) |
|---|---|---|---|
| ACT | 1,265 | 2.1 | 356.6 |
| NSW | 12,847 | 21.8 | 182.1 |
| NT | 1,979 | 3.4 | 875.6 |
| Qld | 18,339 | 31.1 | 423.7 |
| SA | 10,763 | 18.2 | 669.0 |
| Tas | 1,313 | 2.2 | 260.3 |
| Vic | 6,996 | 11.9 | 130.2 |
| WA | 5,524 | 9.4 | 246.6 |
| Total | 59,026 | 100.0 | 272.1 |

Source: NNDSS

**Figure 3: Map of laboratory confirmed influenza rates, 2009, by Statistical Area level 3**



Source: NNDSS

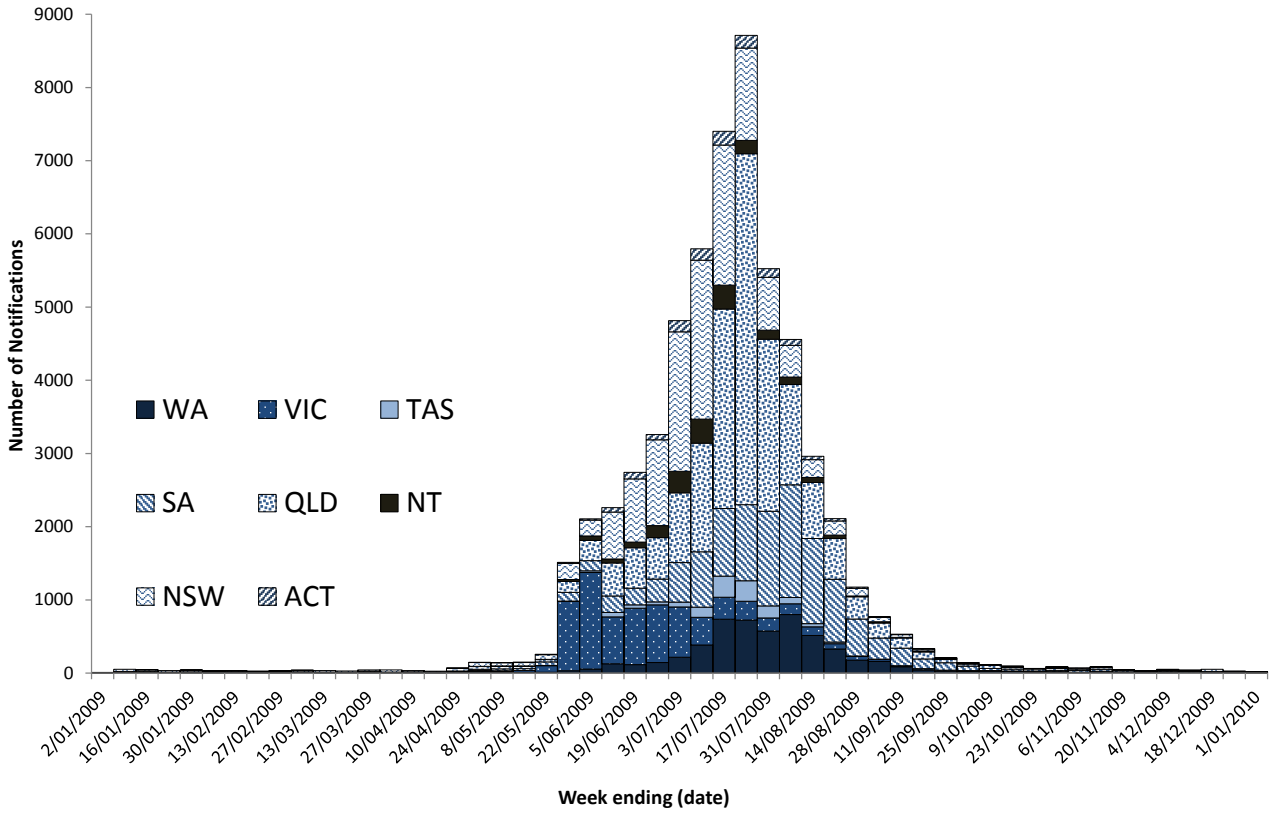
than other jurisdictions and, as a result, testing recommendations were focussed towards vulnerable individuals; therefore resulting in an overall relative reduction in the number of notifications compared to other jurisdictions.[9]

## Age-sex profile

In 2009, data completeness to determine the sex and age at onset for cases was high, 99.4% (58,654/59,026) and 99.8% (58,947/59,026) respectively, with a combined completeness of 99.3% (58,599/59,026). Females comprised just over half (51%; 29,813/58,654) of the cases and the age-standardised notification rates were also higher in females compared to males, 284.2 and 269.8 per 100,000 population respectively.

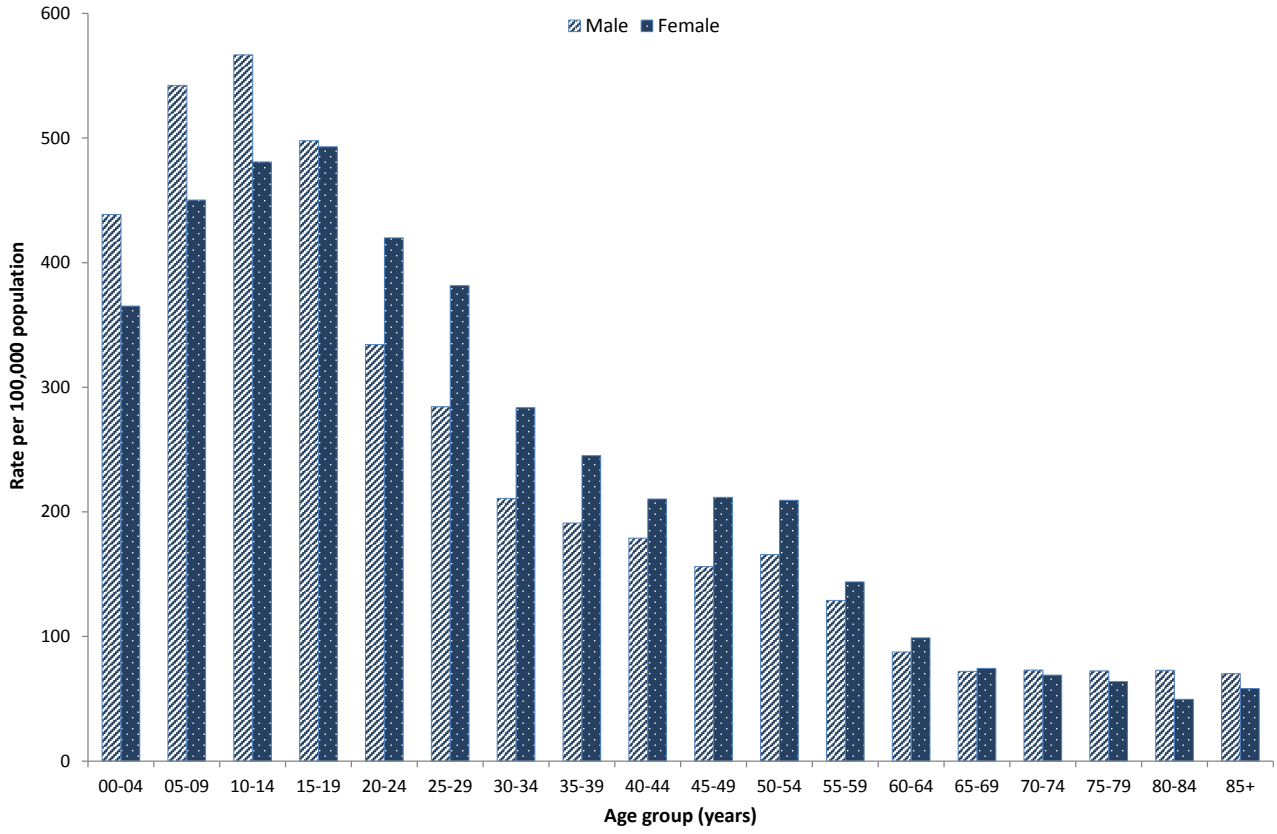
The median age of notifications in 2009 was 21 years (IQR 11-38), which is lower than the median age (26 years) among notifications for the period 2004-2008. Age-specific notifications

**Figure 4: Notifications of laboratory confirmed influenza, 2009, by state or territory and week of diagnosis**



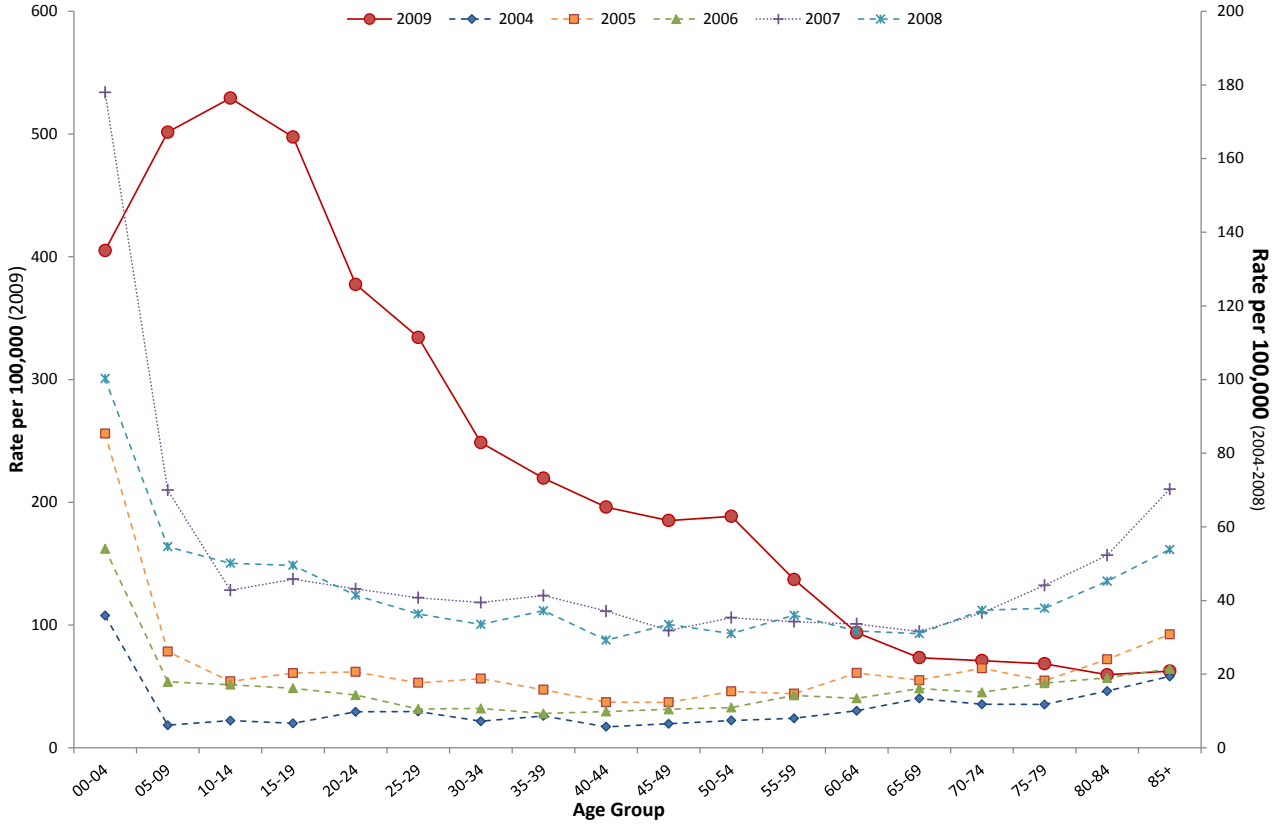

Source: NNDSS

**Figure 5: Notification rates of laboratory confirmed influenza, Australia, 2009, by sex and age group***



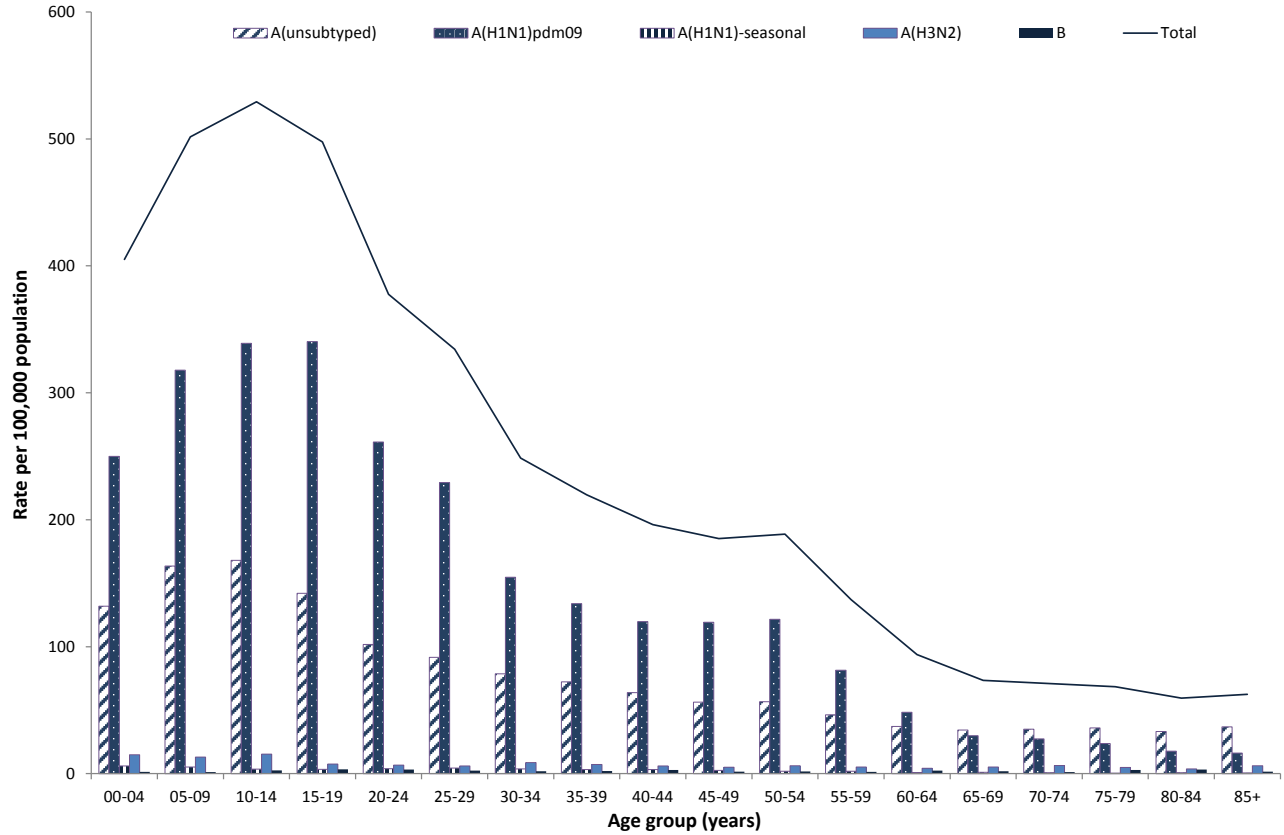

Source: NNDSS *Excludes 427 cases where age and/or sex were not reported.

**Figure 6: Notification rates of laboratory confirmed influenza, Australia, 2004 to 2009, by age group***




Source: NNDSS
*Excludes a small proportion of cases where age was not able to be determined

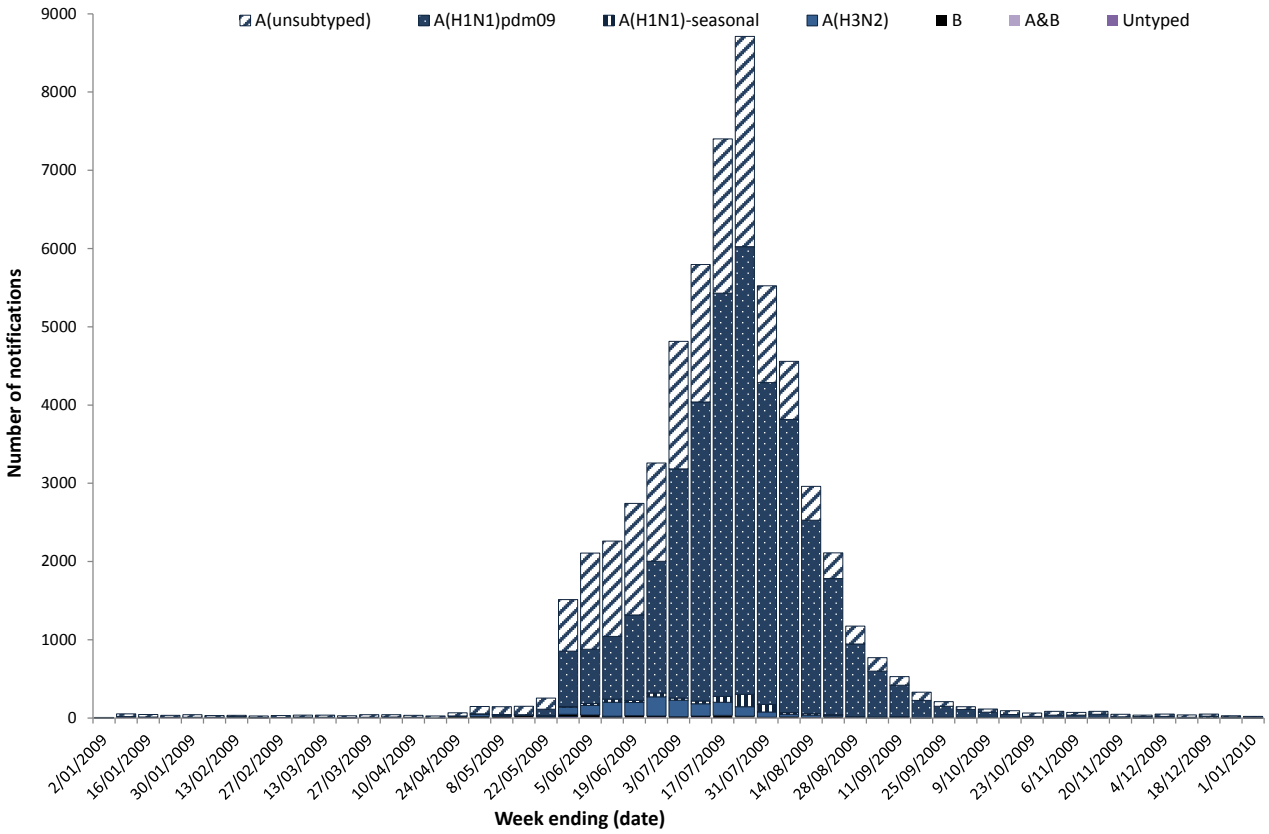
**Figure 7: Rates of laboratory confirmed influenza, Australia, 2009, by subtype and age group***




Source: NNDSS

*Excludes 79 cases where age was not able to be determined

**Figure 8: Notifications of laboratory confirmed influenza, Australia, 2009, by subtype and week of diagnosis**




Source: NNDSS

rates were higher in males compared to females amongst those aged less than 20 years and 70 years and over (Figure 5).

Traditionally the age distribution of influenza notifications has rates highest in children aged less than five years and the elderly. In 2009, notifications were highest in younger age groups, peaking in the 10-14 years age group, with a downward trend with increasing age (Figure 6). Figure 7 highlights that this apparent shift in the traditionally observed age distribution of cases is likely to be due to infection with influenza A(H1N1)pdm09 predominately occurring in younger populations.

## Virus type and subtype

Of the total influenza notifications in 2009 (n=59,026), nearly all were influenza type A (97.8%; n=58,414). Influenza type B infections accounted for 473 notifications, 13 were reported as influenza A and B co-infections and 126 were reported as untyped. Of the influenza type A infections: 64% (n=37,456) were reported as A(H1N1)pdm09; 3% (n=1,705) A(H3N2); 1% (n=678) seasonal A(H1N1); and 32% (n=18,575) were unsubtyped (Table 3). Of those cases reported as influenza A(unsubtyped), a high proportion of these are likely to be the pandemic strain. Due to the volume of tests requested and therefore the need to prioritise laboratory resources, especially once community transmission of the pandemic virus was established, laboratory confirmation of infection with the pandemic specific virus may not have been performed or reported (Figure 8). These cases are likely to have been managed as 'Suspected – with influenza A positive result' under the case definition for pandemic influenza (Appendix 2).

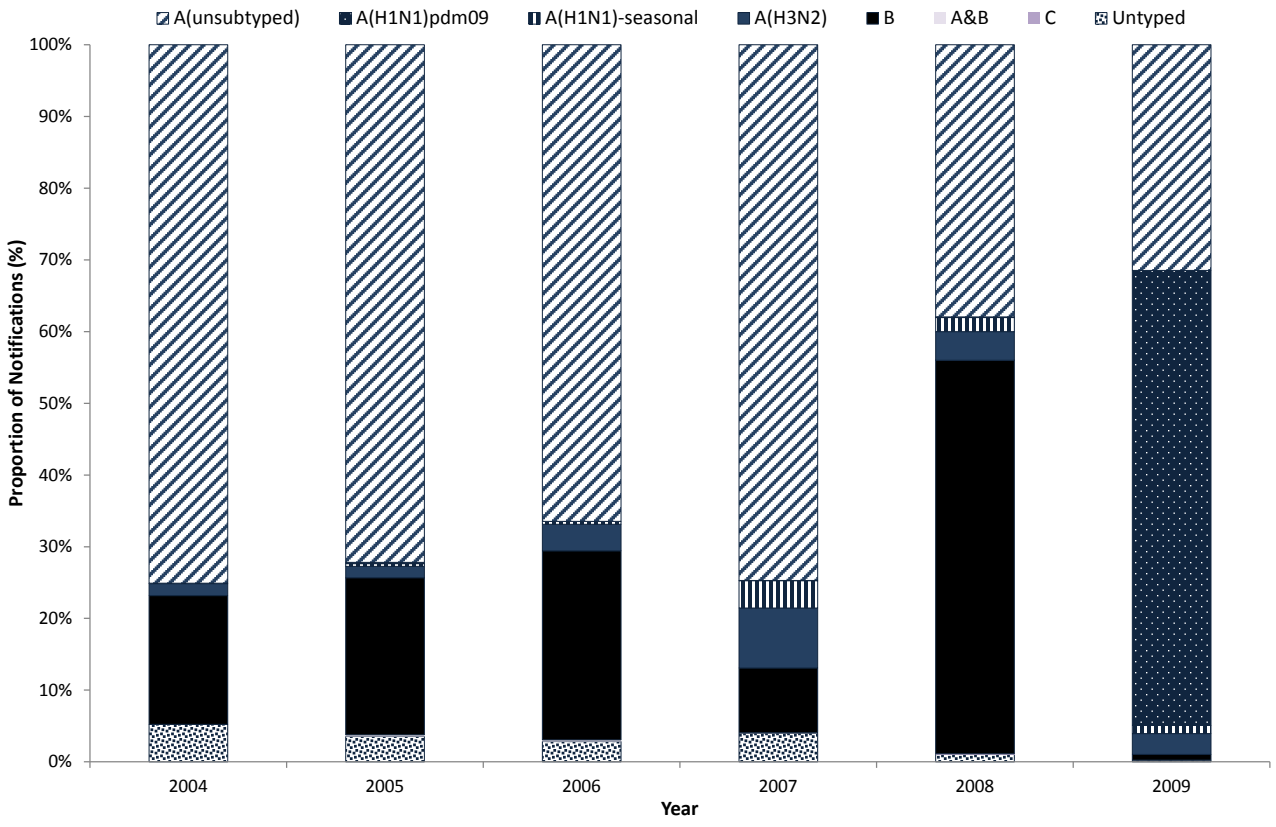
As a proportion, in 2009 there was a very small amount of influenza B infections (1%) reported compared with previous years where influenza B represented between 9 and 55% of overall notifications (Figure 9). Analysis of influenza A

**Table 3: Notifications of laboratory confirmed influenza, Australia, 2009, by state and territory and subtype**

| Type/Subtype | ACT | NSW | NT | Qld | SA | Tas | Vic | WA | Aus |
|---|---|---|---|---|---|---|---|---|---|
| A(unsubtyped) | 292 | 6,097 | 111 | 6,010 | 1,534 | 251 | 3,856 | 424 | 18,575 |
| A(H1N1)-seasonal | | 429 | 46 | 64 | | 21 | 2 | 116 | 678 |
| A(H1N1)pdm09* | 936 | 5,205 | 1,493 | 11,993 | 9,203 | 974 | 3,093 | 4,559 | 37,456 |
| A(H3N2) | 20 | 841 | 321 | 96 | | 65 | 14 | 348 | 1,705 |
| B | 17 | 161 | 6 | 166 | 26 | 1 | 29 | 67 | 473 |
| A&B | | 12 | | | | | 1 | | 13 |
| Untyped | 0 | 102 | 2 | 10 | 0 | 1 | 1 | 10 | 126 |
| Total | 1,265 | 12,847 | 1,979 | 18,339 | 10,763 | 1,313 | 6,996 | 5,524 | 59,026 |

Source: NNDSS
*The total number of pandemic influenza cases for 2009 captured in NetEpi, NNDSS and Qld EpiLog was 37,754

**Figure 9: Notifications of laboratory confirmed influenza, Australia, 2004-2009, by subtype and year of diagnosis**




Source: NNDSS

subtyping data reported to the NNDSS for the period 2004 to 2009 highlighted a substantial change in the reporting of subtype information. Previously an average of 8% (range 2-14%) of influenza A notifications had subtype information, whereas in 2009 this proportion was 68%. The substantial increase was predominately driven by the need to identify influenza A(H1N1) pdm09 cases.

## Antigenic and genetic characterisation

In 2009, 1,586 influenza virus isolates were subtyped by the WHO Collaborating Centre for Reference and Research on Influenza (WHOCC),[44] representing almost 3% of laboratory confirmed cases reported to the NNDSS. Influenza A(H1N1)pdm09 represented the majority (74%) of isolates subtyped, followed by influenza A(H3N2) (18%), seasonal A(H1N1) (7%) and influenza B (1%).

The WHOCC also conducted antigenic characterisation on 884 of the influenza virus isolates, in similar proportions to those subtyped. The majority of influenza A(H1N1)pdm09 isolates were characterised as A/California/7/2009-like. Seasonal influenza A(H1N1) viruses, A/Brisbane/59/2007-like, circulated sporadically throughout the year in very low numbers, being displaced by the pandemic (H1N1) 2009 strain.[45] Of the circulating influenza A(H3N2) viruses, most were antigenically similar to the 2009 A/Brisbane/10/2007 vaccine component, however the majority of these were low reactor versions indicating some drift in the strain. Although there were only a small number of influenza B viruses detected, antigenic characterisation showed a drift throughout the season in the 2009 vaccine strain, B/Florida/4/2006 (B/Yamagata lineage), to the B/Brisbane/60/2008 (B/Victoria lineage) strain.

Following the 2009 southern hemisphere influenza season, all three strains in the 2010 southern hemisphere influenza vaccine were replaced from those in the 2009 southern hemisphere vaccine. The 2010 vaccine contained A/California/7/2009 (H1N1)-like, A/Perth/16/2009 (H3N2)-like and B/Brisbane/60/2008-like viruses.[45]

Antiviral susceptibility testing for resistance to oseltamivir or zanamivir by enzyme inhibition assay (EIA) was conducted on 587 isolates of the A(H1N1)pdm09 strain by the WHOCC during 2009. Of these isolates, four showed resistance to oseltamivir. Molecular analysis of 276 isolates found 9 isolates (including the 4 oseltamivir resistant isolates identified through EIA) with the H275Y mutation, which is known to confer resistance to oseltamivir.

Oseltamivir resistance was also found in the majority (36/37) of seasonal A(H1N1) isolates tested, which is consistent with historical trends. In 2009, there were no reports of antiviral resistance in any of the A(H3N2) or influenza B isolates tested.

## Influenza-like illness surveillance

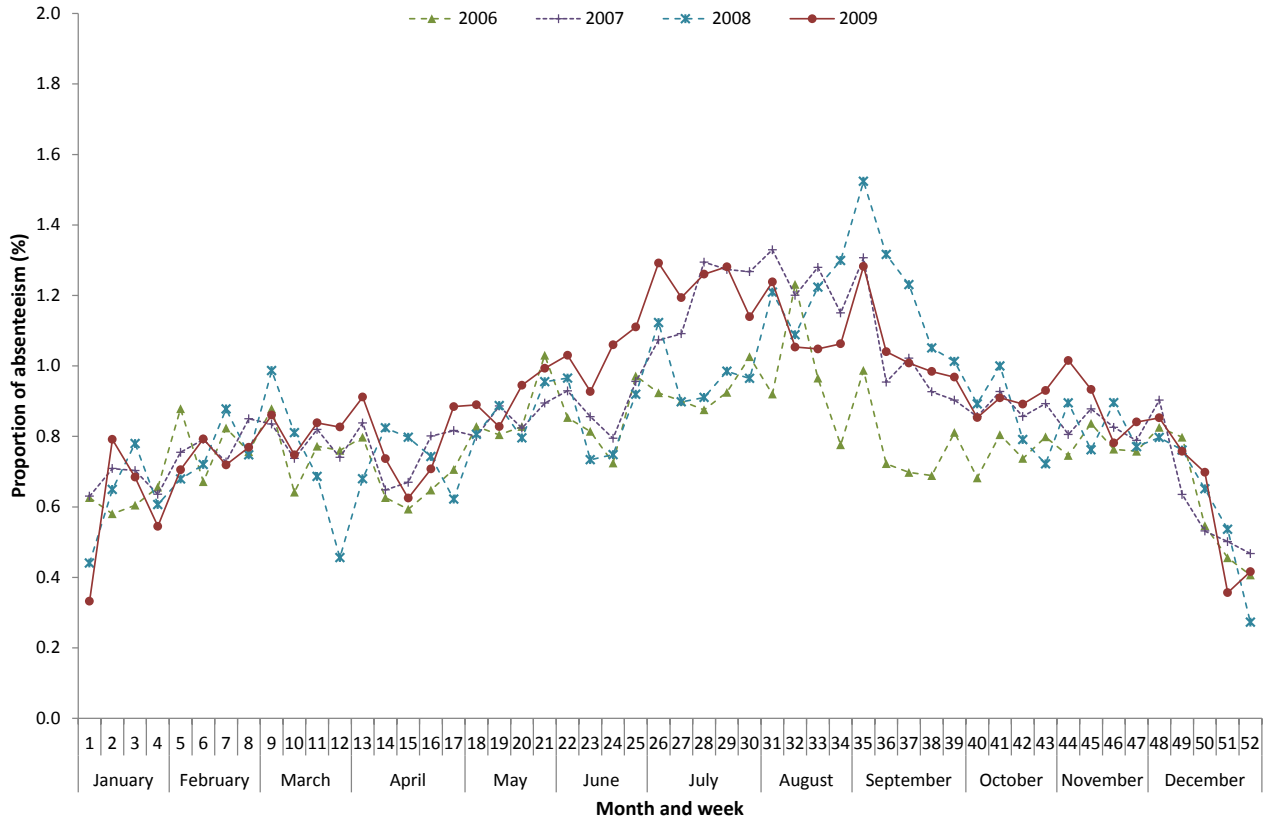
### Community level influenza-like illness surveillance

In 2009, community level ILI surveillance provided an indication of influenza activity within the community. Although these systems are less specific, as they do not include a laboratory confirmation component, they are useful for informing influenza activity detected through more specific systems with varying degrees of sensitivity, including over time and by geographic area.

Absenteeism surveillance, indicated by three or more days of consecutive sick leave, during 2009 had two general peak periods which occurred over several weeks during the month of July followed by a peak week at the beginning of September 2009 (Figure 10). The peak proportion of staff absenteeism over this period was approximately 1.3%. Peak absenteeism rates in 2009 were similar in magnitude to rates observed between 2006 and 2008, however the peak period lasted for several weeks.

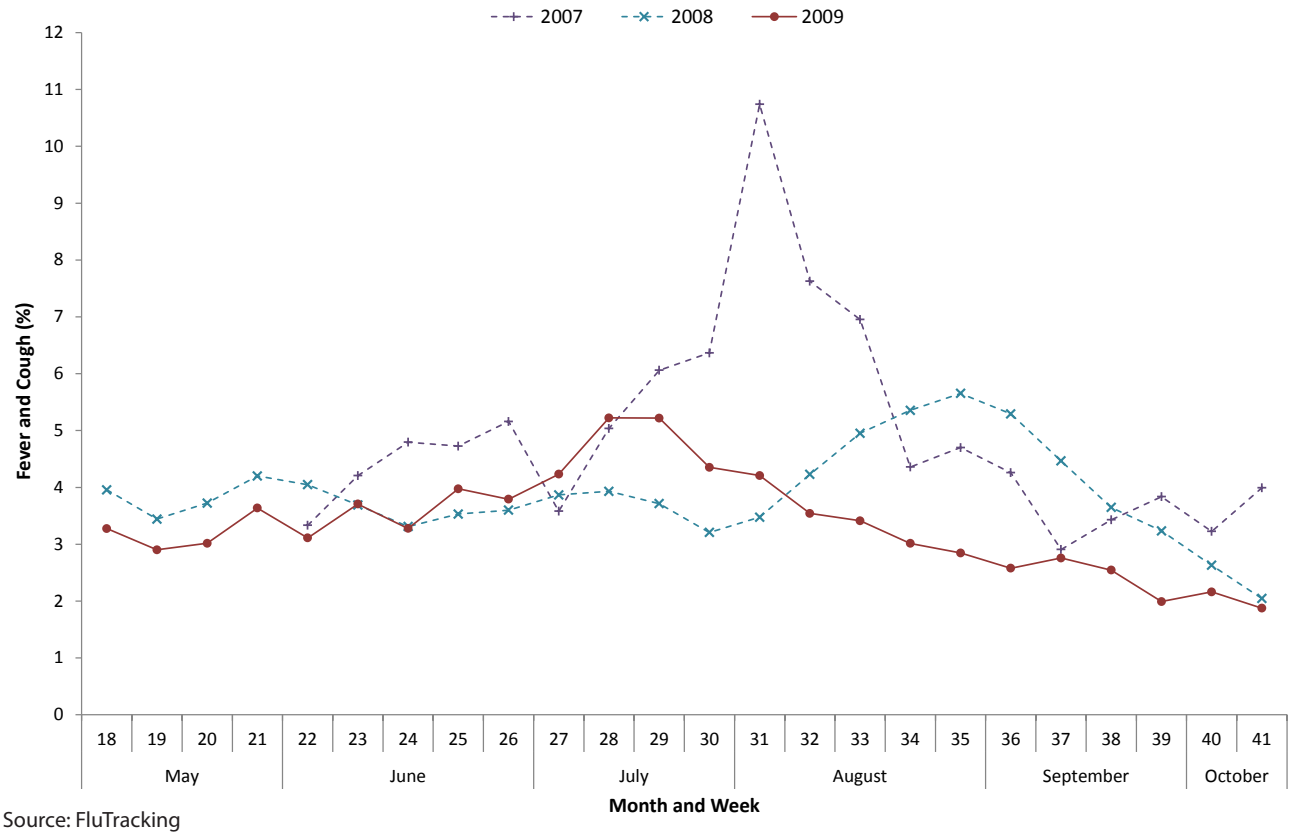
The FluTracking weekly online survey for ILI among participating community members in 2009 showed that rates of fever and cough among participants essentially peaked in mid-July at 5.2% and peaked much earlier than in 2007 and 2008 (Figure 11). Overall however, peak cough

**Figure 10: Rate of absenteeism for a period of three or more consecutive days from a national employer, Australia, 2006 to 2009, by week**



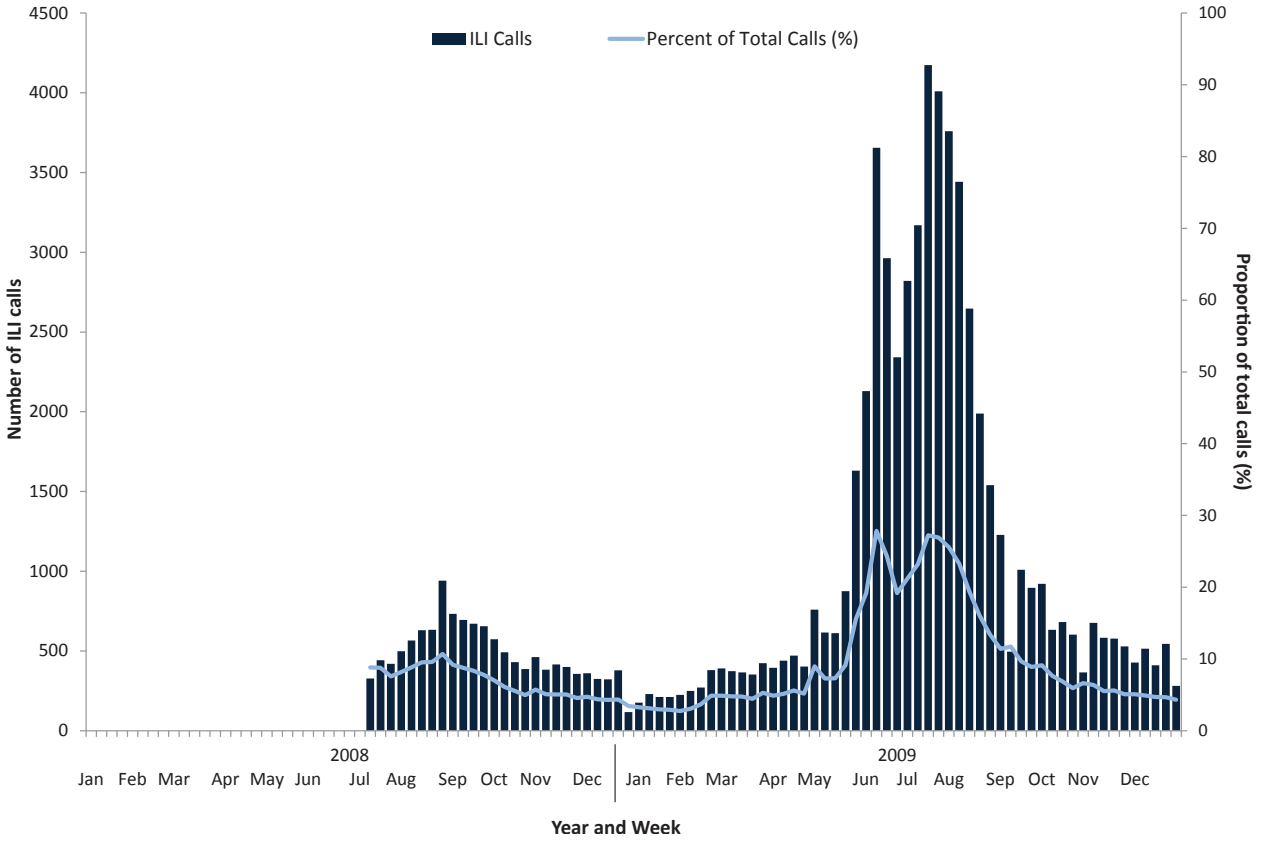

Source: A national employer (not disclosed)

**Figure 11: Proportion of fever and cough among FluTracking participants, May to October, 2007 to 2009, by week**




Source: FluTracking

**Figure 12: Number of calls to the National Health Call Centre Network related to influenza-like illness and percentage of total calls, Australia*, 7 July 2008 to 31 December 2009, by week**




Source: National Health Call Centre Network
*Data represents all states and territories except Victoria and Queensland

and fever rates were not greater than in 2007 and 2008. The attack rate pattern observed in this surveillance system potentially reflects true community ILI activity and that the 2009 season was not necessarily a significant season as these data are not affected by health-seeking behaviour and changes in clinician testing protocols.[19] It should be noted that FluTracking participants in 2007 and 2008 were mostly from New South Wales and the number of participants in 2009 substantially increased making direct comparisons between years difficult.

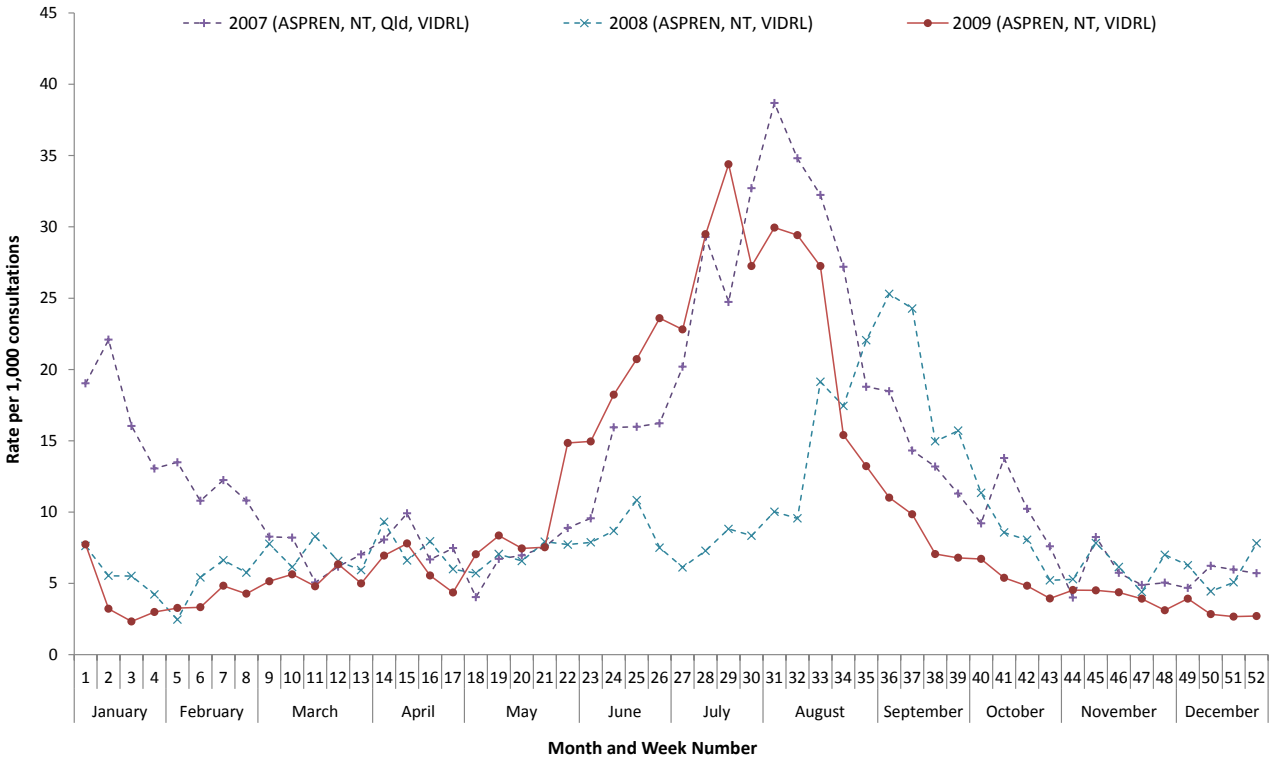
The number and proportion of calls to the National Health Call Centre Network (NHCCN) started to increase in early May 2009, had an initial peak in the week ending 14 June 2009 (27.8% of total calls; n=3,655 calls), followed by a peak in the week ending 19 July 2009 (4,174 calls; 27.2% of total calls) (Figure 12). The timing of influenza-like illness call activity was consistent with the activity observed in influenza notifications, with the sub-peak coinciding with the pandemic phase change announcement to "PROTECT".

### Sentinel general practitioner influenza-like illness surveillance

Combined data from general practitioner ILI surveillance systems throughout Australia showed that, nationally, ILI consultation rates in 2009 followed a similar trend to the notification data, with increases in ILI consultations occurring from mid-May and peaking in mid-July 2009 (Figure 13).

Overall the rate of ILI consultations were not greater than those observed in 2007, which based on influenza activity was considered to be a moderate to severe influenza season since national reporting of influenza began in 2001.[15]

**Figure 13: Weekly rate of influenza-like illness consultations reported from general practitioner surveillance systems, 2006 to 2009, by week**




Source: ASPREN, the Northern Territory Tropical Influenza Surveillance Scheme, the VIDRL General Practices Sentinel Surveillance Program, and Qld Health ILI Sentinel Surveillance in General Practice (2007 only).

Comparisons of seasonal activity observed over the period between 2007 and 2009 are difficult and several considerations need to be made. For example, with regard to the surveillance systems, general practitioner representativeness has varied over the period with the Queensland surveillance system only reporting in 2007; inconsistent representativeness across areas; and methods of data capture and reporting were also variable over the period.

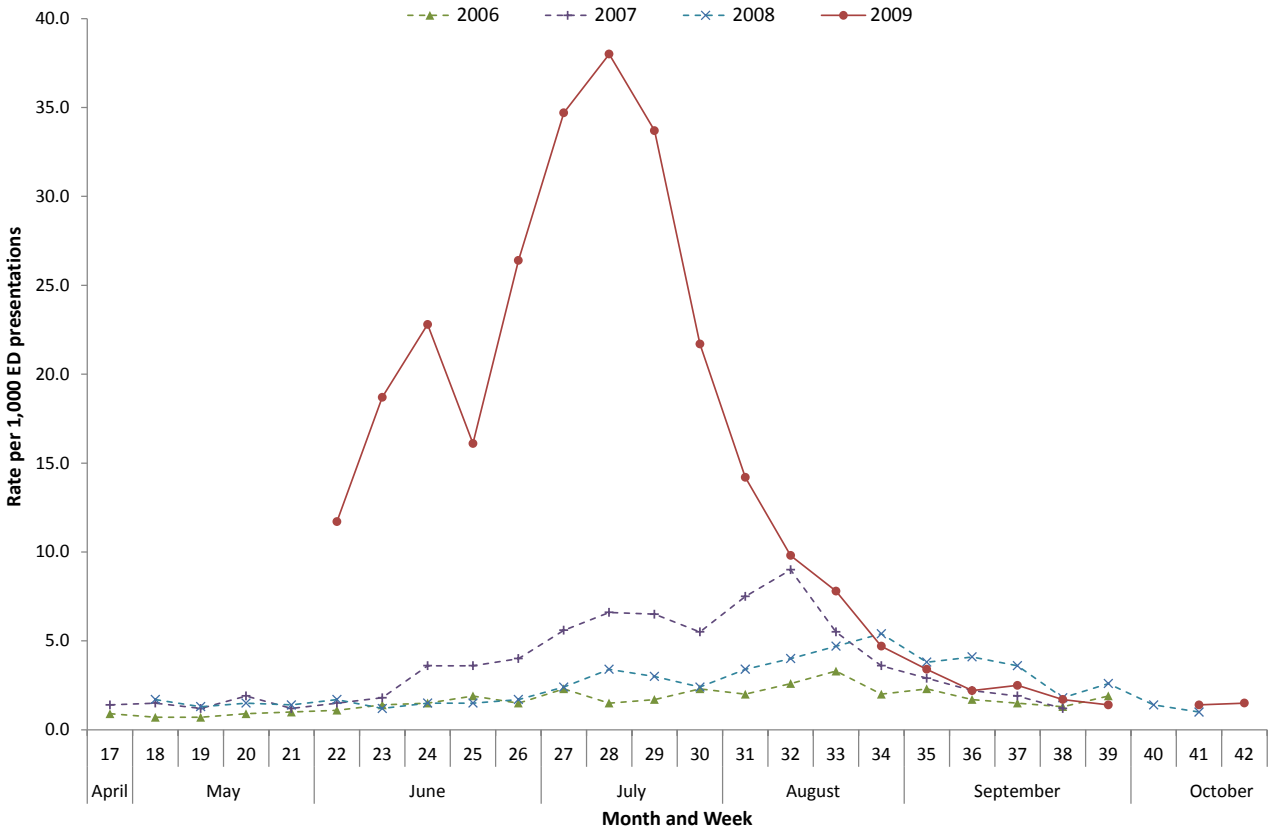
In terms of the 2009 ILI consultation rates, it is difficult to measure whether the initial community concern associated with the emergence of the pandemic influenza virus might have led to increased presentations to general practitioners, especially in people who may not have normally sought medical attention. Also following the move to the PROTECT pandemic phase, individuals with mild symptoms and without risk factors were advised not to consult their general practitioner, rather to attend specifically setup influenza clinics, in order to reduce the impact on service availability for more severe and vulnerable to severe disease cases.[9]

### Emergency department influenza-like illness surveillance

Emergency department ILI surveillance data, including the proportion of admissions, were used during the 2009 influenza season to inform illness severity. This included the comparison of activity trends to other surveillance systems such as the sentinel general practice ILI surveillance data, as well as impacts on the health-care system.

Presentations to New South Wales hospital emergency departments for ILI began to rise quite rapidly from early May 2009. The unusually early rise was noted as being for mild illnesses and associated with increased awareness of the emerging pandemic influenza virus. Presentations for ILI peaked during mid-July

**Figure 14: Rate of influenza-like illness presentations to New South Wales emergency departments, May and October, 2006 to 2009 by week**




Source: NSW Influenza Weekly Epidemiology Report

2009 with an on average 1,300 presentations per week (38.0 per 1,000 presentations) (Figure 14), and these presentations were mainly for mild illness, with around 8% of presentations during this peak week being admitted to hospital. Over this peak period, rates were highest in people aged 5 to 34 years.[21] In comparison to previous years, presentation rates were substantially higher and peaked earlier.

Respiratory viral presentations to Perth (Western Australia) hospital emergency departments during 2009 began to increase in late May and peaked in early August at 1,266 presentations (121 per 1,000 presentations) (Figure 15). The proportion of these cases admitted to hospital ranged throughout the period of peak activity (June to September) from 3.7% to 7.4% with a median admission rate of 4.8%. In comparison, the number of presentations observed in 2008 had a lower and later peak, and the duration above baseline activity was shorter. The proportion of cases admitted to hospital, both for the entire 2008 period and the 2008 peak period (August to October) was consistent with those observed in 2009.

## Hospitalisations

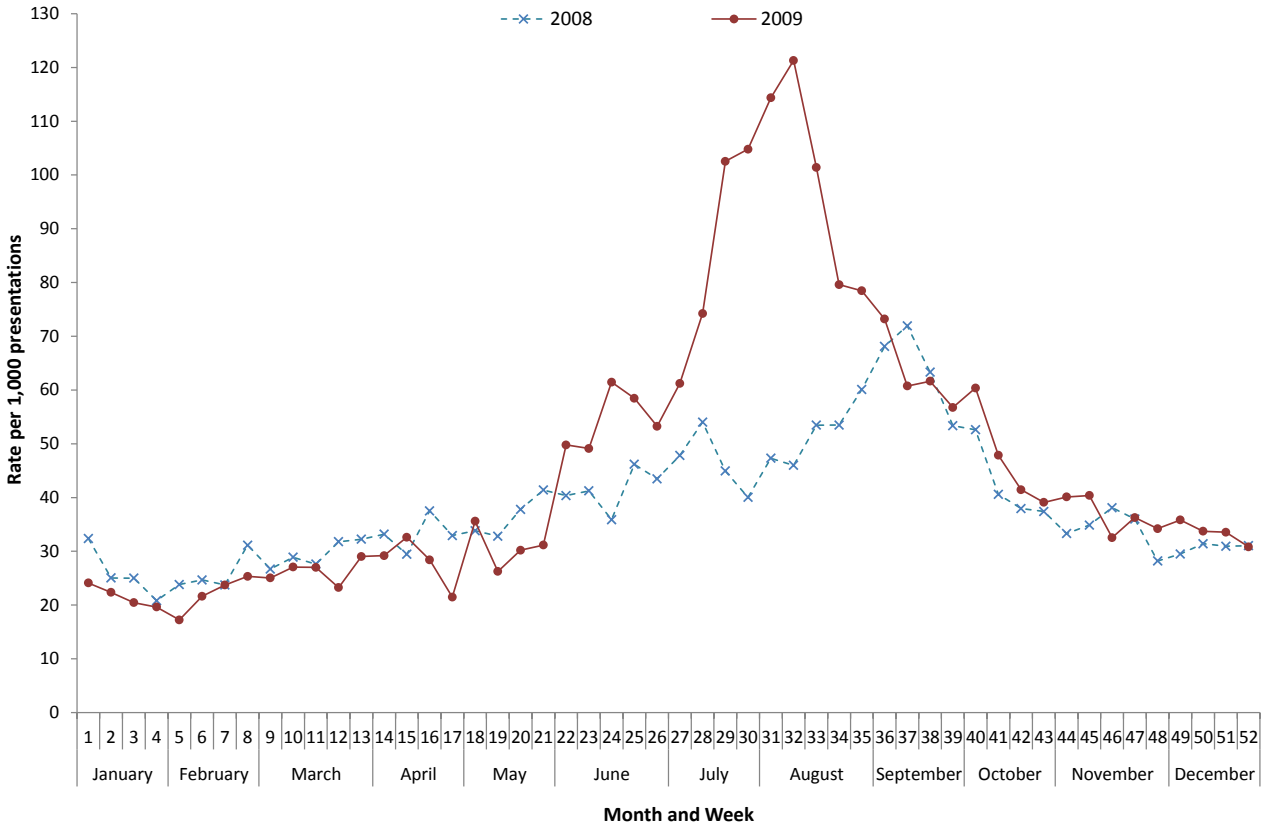
### Admitted Patient Care Dataset

For the period 2004 to 2008, the annual average number of hospital separations[III] reported either as a principal diagnosis or additional diagnosis for virologically confirmed influenza (ICD-10-AM[IV] codes J09-J10) or for influenza with or without virological confirmation (ICD-10-AM codes J09-J11) was 1,373 (range 712-2,348) and

III The process by which an episode of care for an admitted patient ceases. A separation may be formal or statistical. Formal separation: The administrative process by which a hospital records the cessation of treatment and/or care and/or accommodation of a patient. Statistical separation: The administrative process by which a hospital records the cessation of an episode of care for a patient within the one hospital stay.

IV ICD-10-AM codes used J09 – Influenza due to identified avian influenza virus (renamed in 2010 to Influenza due to certain identified influenza virus); J10 – Influenza due to other identified influenza virus; J11 – Influenza, virus not identified.

**Figure 15: Rate of respiratory viral presentations to Western Australia emergency departments, 2008 to 2009, by week**




Source: Western Australia Department of Health

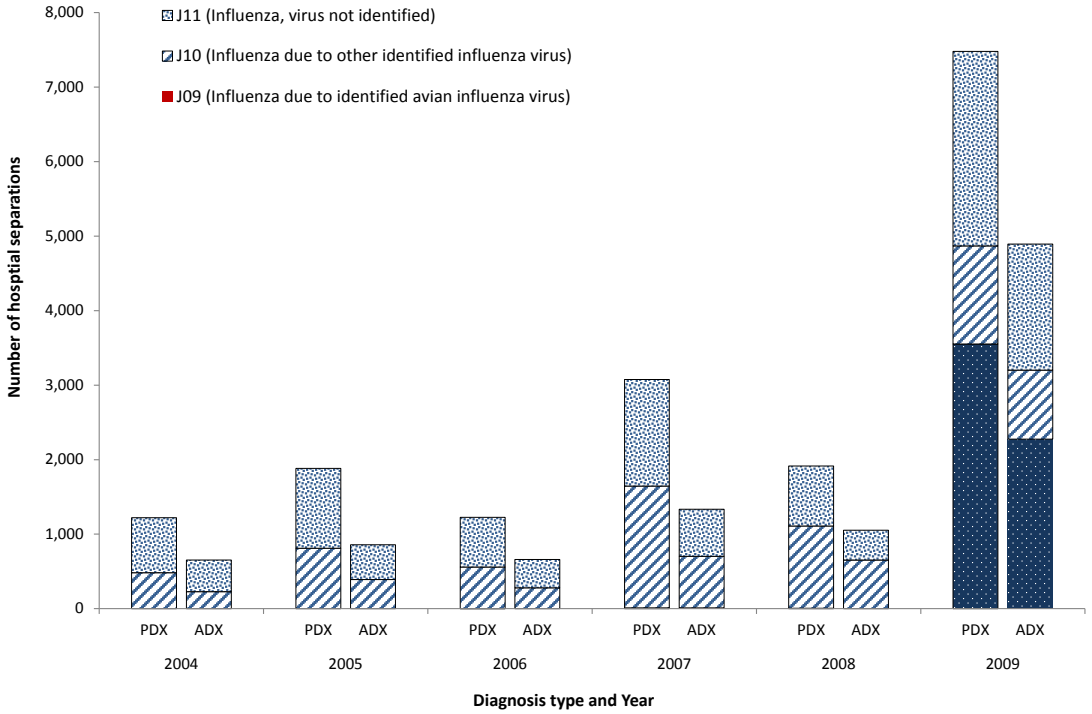
2,776 (range 1,876-2,966) respectively (Figure 16). In comparison, in 2009 there was almost a five-fold increase (n=8,069) in the number of separations for virologically confirmed influenza and a three-fold increase (n=12,374) for influenza with or without virological confirmation. Additionally the ratio of virologically confirmed influenza to non-virologically confirmed influenza was much higher in 2009 compared to the 2004-2008 mean (1.9:1 and 0.98:1 respectively); reflective of the CDNA pandemic influenza testing recommendations. As a result of the pandemic in 2009, separations coded as J09 represented the majority of influenza associated hospital separations (n=5,829), noting that the J09 code in 2009 was meant to represent identified avian influenza virus.

As a proportion of overall hospital separations, the proportion of influenza associated hospital separations increased substantially from the 2004-2008 mean of 0.04% to 0.15% in 2009. Over the 2004-2008 period, influenza was reported as the principal diagnosis for approximately two-thirds (67.2%) of influenza associated separations (ICD-10-AM codes J09-J11), whereas in 2009 there was a slightly lower proportion (60.4%). This difference was relatively consistent across all age groups, suggesting that there was a slightly higher propensity to investigate or report influenza as an additional diagnosis during a hospitalisation in 2009 regardless of age.

In comparison to the weekly 2004-2008 mean number of influenza associated separations, in 2009 there was not only a substantial increase in the number of weekly separations, but also an earlier, more intensive, increase and peak. However, the overall number of weeks that separations were above apparent baseline activity was similar (Figure 17).

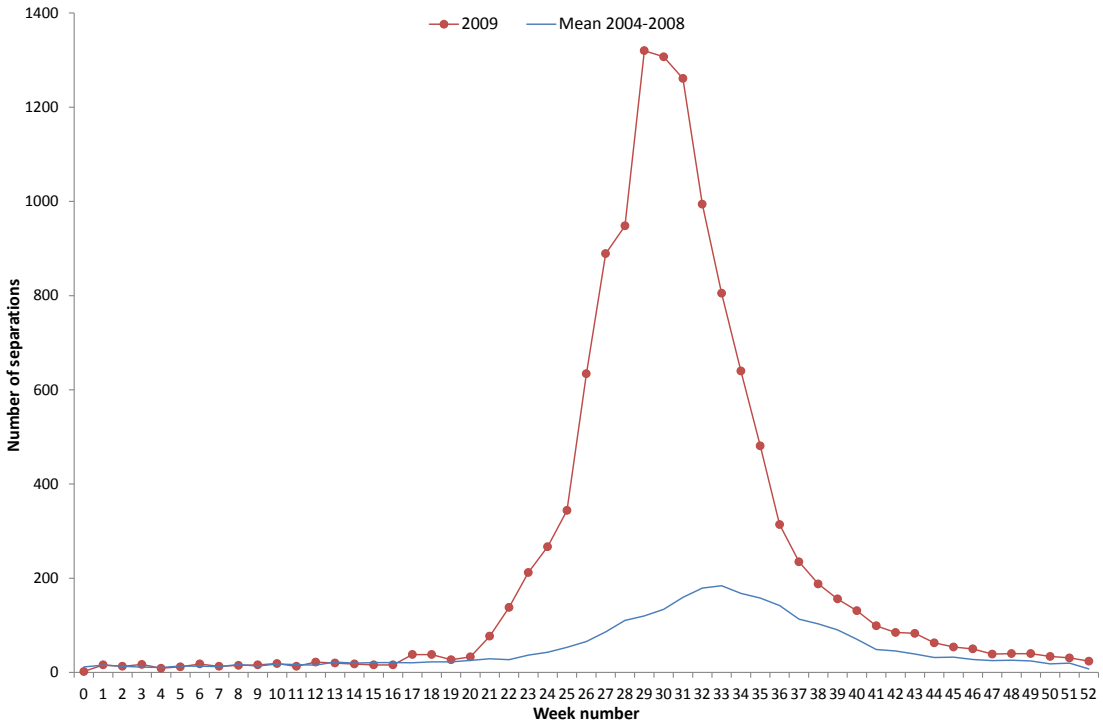
Between 2004 and 2009, hospital separations remained highest for children aged less than 5 years (Figure 18). The age-specific hospitalisation incidence profile suggests that hospitalisation

## Figure 16: Number of hospital separations ICD-10-AM coded as J09, J10 or J11, Australia, 2004-2009, by diagnosis type




Source: Admitted Patient Care Data collection 2003-04 to 2009-10 Note: PDX = Principal diagnosis and ADX = additional diagnosis

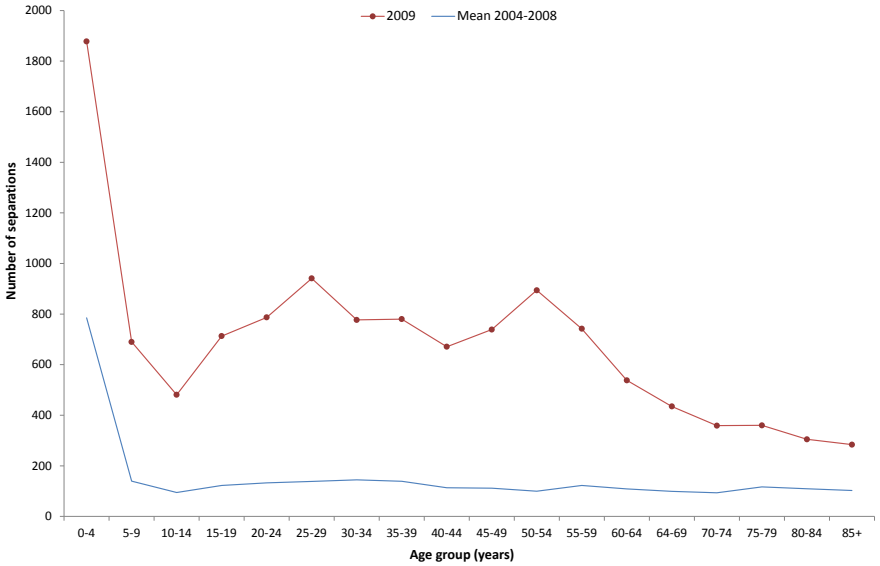
## Figure 17: Number of hospital separations for all diagnoses* ICD-10-AM coded as J09, J10 or J11, Australia, 2004-2009, by week of separation



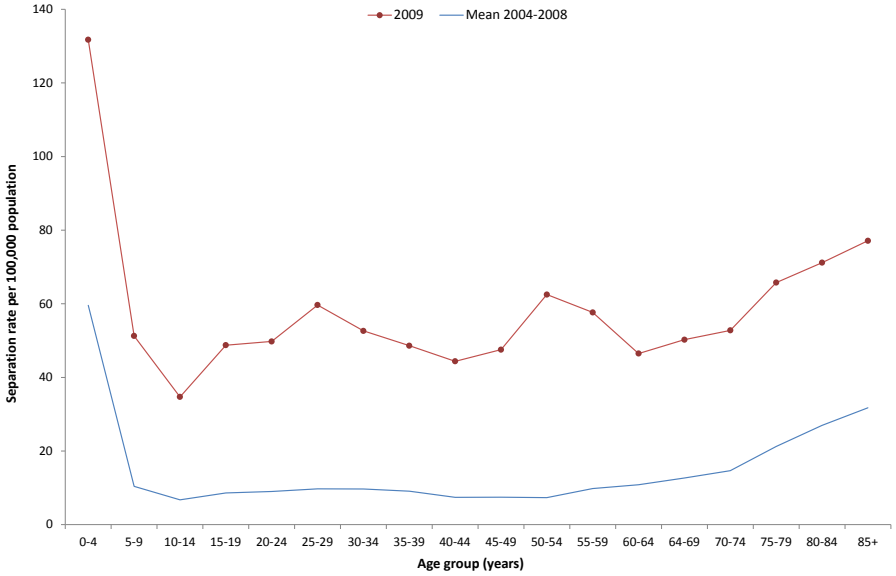

Source: Admitted Patient Care Data collection 2003-04 to 2009-10
*Includes both principal diagnosis and additional diagnoses. Where principal diagnosis is the diagnosis established after study to be chiefly responsible for occasioning a patient's service event or episode, as represented by a code; and additional diagnoses are defined as condition or complaint either coexisting with the principal diagnosis or arising during the episode of admitted patient care, episode of residential care or attendance at a health care establishment, as represented by a code.

## Figure 18: Hospital separations for all diagnoses* ICD-10-AM coded as J09, J10 or J11, Australia, 2004-2009, by 5-year age group

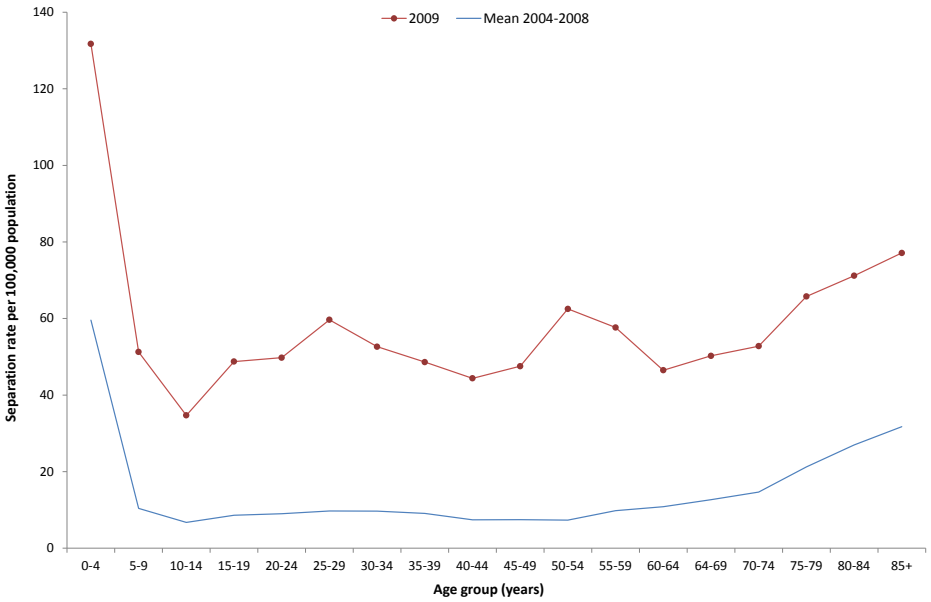
(i) Number of hospital separations.




(ii) Rate per 1,000 separations.

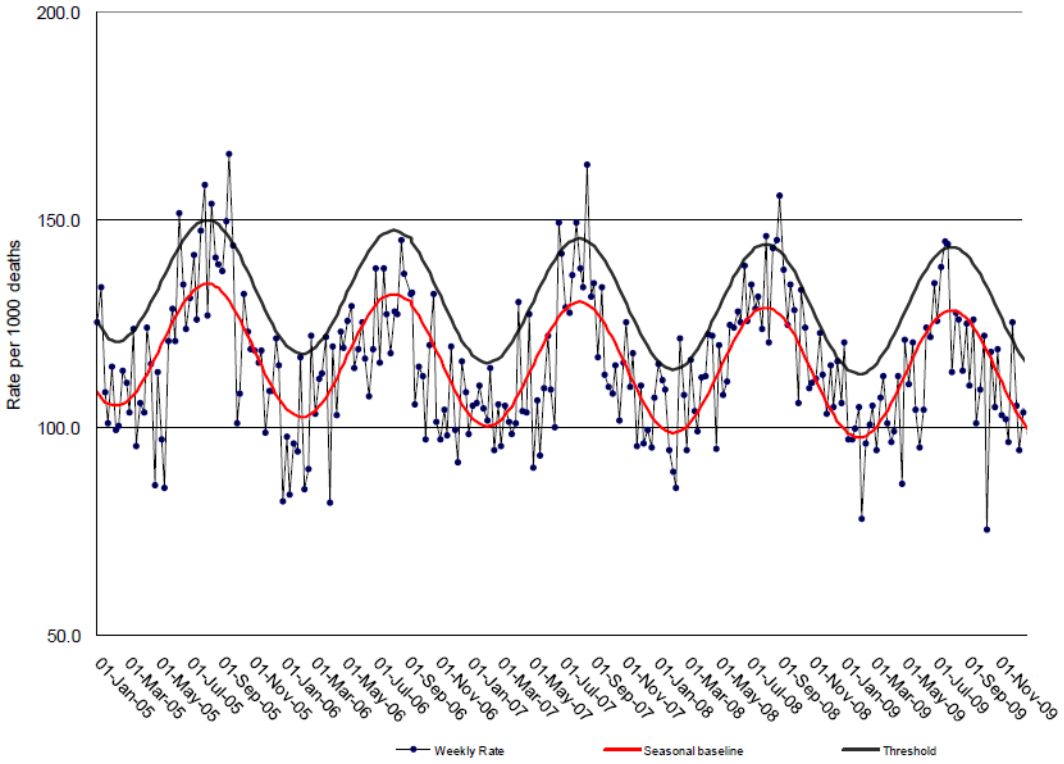



(iii) Rate per 100,000 population.
Source: Admitted Patient Care Data collection 2003-04 to 2009-10




*Includes both principal diagnosis and additional diagnoses. Where principal diagnosis is the diagnosis established after study to be chiefly responsible for occasioning a patient's service event or episode, as represented by a code; and additional diagnoses are defined as condition or complaint either coexisting with the principal diagnosis or arising during the episode of admitted patient care, episode of residential care or attendance at a health care establishment, as represented by a code.

**Figure 19: Rates of deaths classified as influenza and pneumonia from New South Wales registered death certificates, 2005 to 2009**




Source: NSW Influenza Surveillance Report[49]

with influenza in 2009 were more common in younger to middle age groups, and less common in those aged over 70 years. Hospital separation rates associated with influenza, as a potential measure of impact, showed a relatively steady decrease with increasing age in 2009; rather than an initial pronounced decrease in the 5-14 years age groups, followed by a moderate increase and almost plateau in the middle years age groups seen in previous years. As the overall number of separations across the younger and middle-years age groups over the 2004 to 2009 period remained very similar, this would indicate that there was a definite change in the casemix of admitted patient care separations attributable to influenza in these age groups.

### FluCAN[30]

From the eight public acute hospital sites represented in FluCAN, a total of 538 patients were recorded as being hospitalised with laboratory confirmed influenza over the surveillance period (1 July to 30 November 2009). Of these cases, the majority (86.4%; n=465) were due to influenza A(H1N1)pdm09, with the remainder of cases due to seasonal strains of influenza A. Co-morbidities were present in 76.3% (354/464) of patients and 30.3% of women aged 15-49 years were pregnant, with the majority in their third trimester (72.5%; n=29). FluCAN reported that 21.9% (n=102) of patients were admitted to ICUs, and of the patients admitted to hospital, 5.6% (n=26) died. Overall FluCAN results were consistent with national notification data and published ICU admissions data.

### Australian Paediatric Surveillance Unit[32]

During the 2009 surveillance period there were 124 confirmed cases of severe complications of influenza in children aged less than 15 years. The median length of stay was two days (range 1-53). All of the cases reported had influenza type A infections, with 77 having A(H1N1)pdm09, six type A but not H1N1; two A(H3N2) and 15 were reported with an unknown subtype. The median age was 2.8 years and 53% were male. Forty-five per cent of children had an underlying chronic condition. Pneumonia (69%) was the most common complication reported, followed by encephalopathy (13%). Over a third of

the cases reported were admitted to paediatric intensive care units, with a median length of stay of 6.5 days (range 2-51) and the majority (74%) required ventilator support. Six children died and all were reported as having influenza A(H1N1)pdm09.

## Mortality

Mortality from a primary influenza infection is rare and most deaths that are attributed to influenza are generally due to other causes such as pneumonia, congestive heart failure, or chronic obstructive pulmonary disease. Influenza virus infection is infrequently listed on deaths certificates as more broadly testing for influenza infection is not usually done, especially as the virus is only detectable for a short period of time and many people may only seek health care for secondary complications of influenza later in their illness when the virus can no longer be detected from respiratory samples.[46]

### ABS mortality data

Influenza and pneumonia (ICD-10 codes J09-J18) were noted as the underlying cause of death for 1,790 persons in 2009 and represented 1.3% of all deaths. More females than males died of influenza or pneumonia (1,030 females compared to 760 males); however the standardised death rate for males was higher than in females (6.4 compared to 8.0 deaths per 100,000 population, respectively).[47] In 2009, the number of influenza (J09-J11: virus identified and not identified) related deaths was 127. Influenza A(H1N1) pdm09, represented by ICD-10 code J09) was the underlying specifically identified cause of 77 of these deaths or 0.05% of all registered deaths in Australia and the median age at death was 47.8 years.47

In 2009, the number of overall influenza and pneumonia related deaths was lower in comparison to the median observed between 2004 to 2008 (2,711; range 1,760 to 3,381) seasons. However, the number of specific influenza (J09-J11: virus identified and not identified) related deaths was much higher in comparison to the median number observed between 2004 to 2008 (39; range 16 to 73). The significance of this increase is difficult to interpret as the degree of likely increase in testing as a result of the pandemic is unknown, however since the pandemic both the proportion of influenza virus identified deaths (J09-J10) and total influenza specific deaths (J09-J11) have in general been much higher compared to the pre-pandemic period.

### New South Wales Registry of Births, Deaths and Marriages

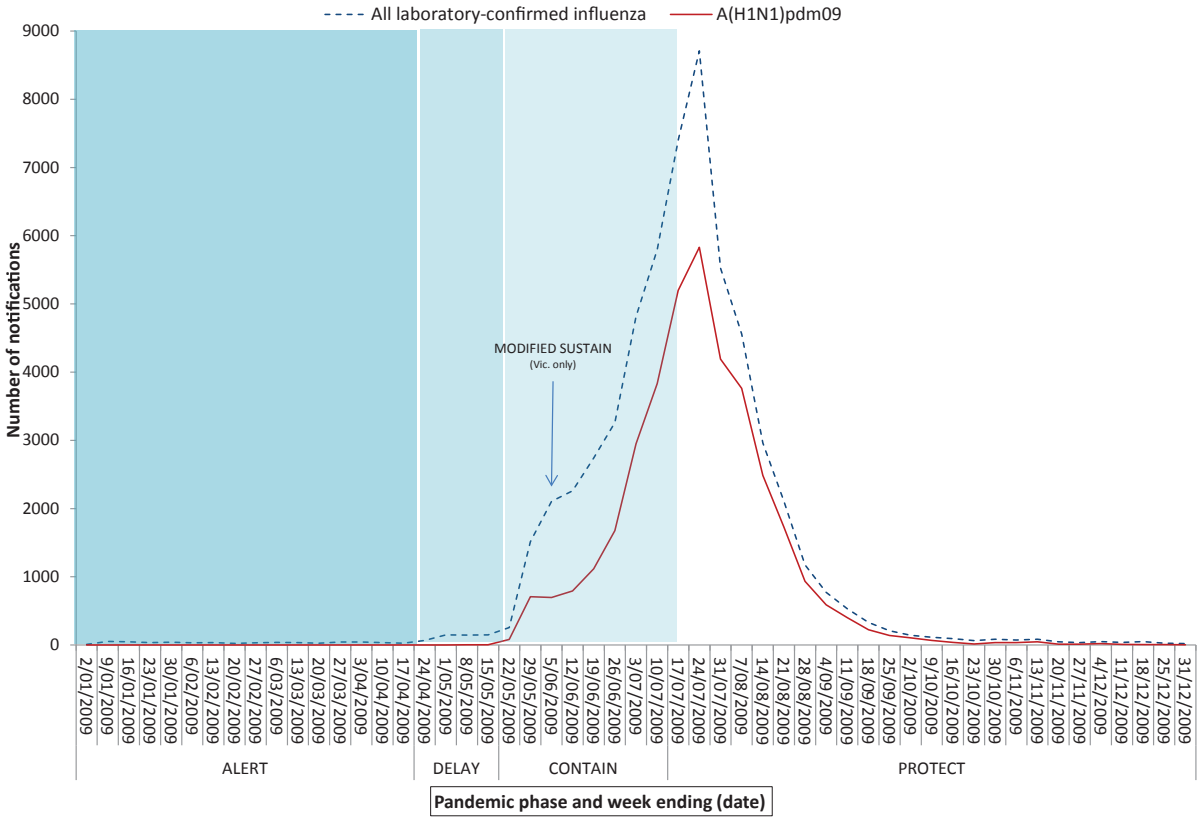
Indirect information derived from New South Wales Registry of Births, Deaths and Marriages death certificate surveillance, indicated that rates of deaths from influenza and pneumonia peaked and slightly exceeded the epidemic threshold during July 2009 at approximately 145 deaths per 1,000 NSW population (Figure 19). Overall, the combined pneumonia and influenza death rates were equal or lower than the predicted seasonal baseline for the majority of the season, but did exceed the epidemic threshold for three weeks during the season. It is noted that whilst influenza-related excess mortality was relatively low compared with seasonal activity in most recent years, there was a redistribution of deaths with a relative increase of deaths in younger age groups.[19, 21, 48]

## Pandemic Influenza A(H1N1)pdm09

In 2009, there were 37,456 notifications of influenza A(H1N1)pdm09 reported in the NNDSS; however, as a number of alternative or additional surveillance systems were utilised, in order to undertake enhanced epidemiological analyses the most representative and accurate sources of cases were combined as described in Appendix 4. Based on these systems, a total of 37,754 confirmed cases of influenza A(H1N1)pdm09 were identified for analysis.

Of the 37,754 cases of influenza A(H1N1)pdm09 identified nationally, enhanced data were reported for a subset of these cases, with field completeness varying by jurisdiction, pandemic phase and health care setting. The analysis of the enhanced surveillance data for all confirmed pandemic influenza cases have been focussed on

**Figure 20: Notifications of all laboratory confirmed influenza and influenza A(H1N1)pdm09, Australia, 2009, by pandemic phase and week of diagnosis or onset***




Source: NNDSS and NetEpi
* Diagnosis week was used for 'all laboratory confirmed influenza' notifications from NNDSS and onset week was used for cases notified as influenza A(H1N1)pdm09 in the NetEpi dataset.

the DELAY and CONTAIN phases, 28 April to 16 June 2009. This period has been selected based on its likely representation of pandemic influenza across the community and higher levels of data completeness and quality. This period however does include Victoria's MODIFIED SUSTAIN phase (3 to 16 June 2009).

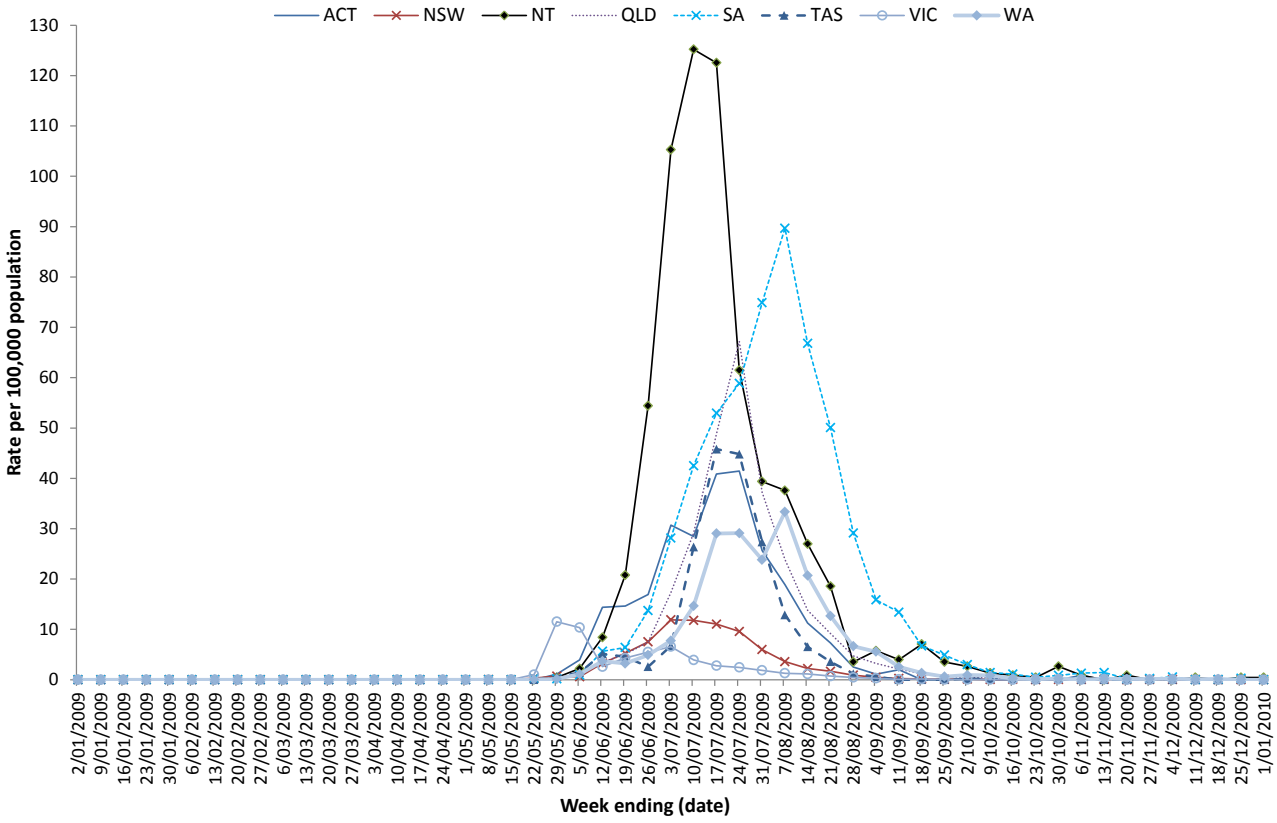
Some commentary is provided on analyses of the enhanced data during the PROTECT phase (17 June to 31 December 2009), where case ascertainment strategies were focussed towards those at risk for severe disease or those with severe disease and the conclusions are supported by sentinel systems that continued throughout the pandemic to monitor for changes in the epidemiology and virology of the pandemic.

## Source of infection or introduction

Following the emergence of the virus overseas in April 2009, public health efforts initially focused on delaying the entry of the virus into Australia through a range of measures, including border control measures, contact tracing, public health awareness, testing and isolation of possible cases, and quarantine of people in close contact with patients who tested positive for the illness.[9]

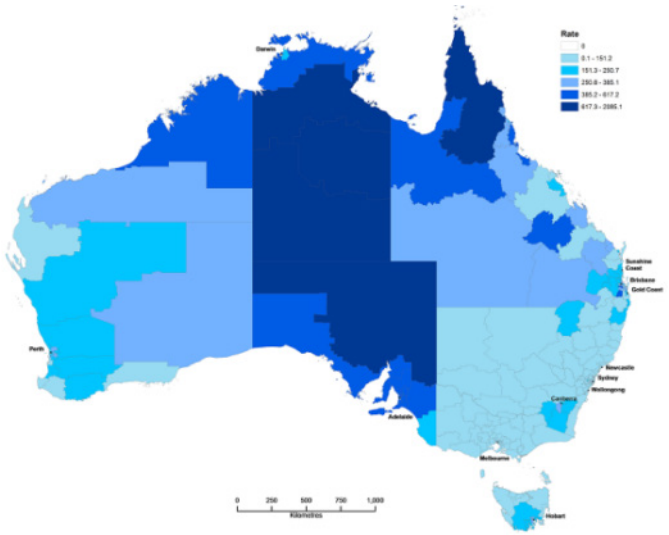
The first case of confirmed influenza A(H1N1) pdm09 infection in Australia was notified on 7 May 2009 in a traveller who had returned from the United States of America to Queensland. Following the detection of this imported case, the first case of locally acquired infection was identified on 16 May 2009. In these first few weeks of the outbreak in Australia, transmission of the virus was sporadic and generally linked to close contacts of travellers returning from countries with established community level transmission (eg. Mexico and the United States of America). By mid-June 2009, community-wide transmission of the virus was occurring across most jurisdictions.

**Figure 21: Crude rates of laboratory confirmed cases of influenza A(H1N1)pdm09, 2009, by week of onset and state and territory**



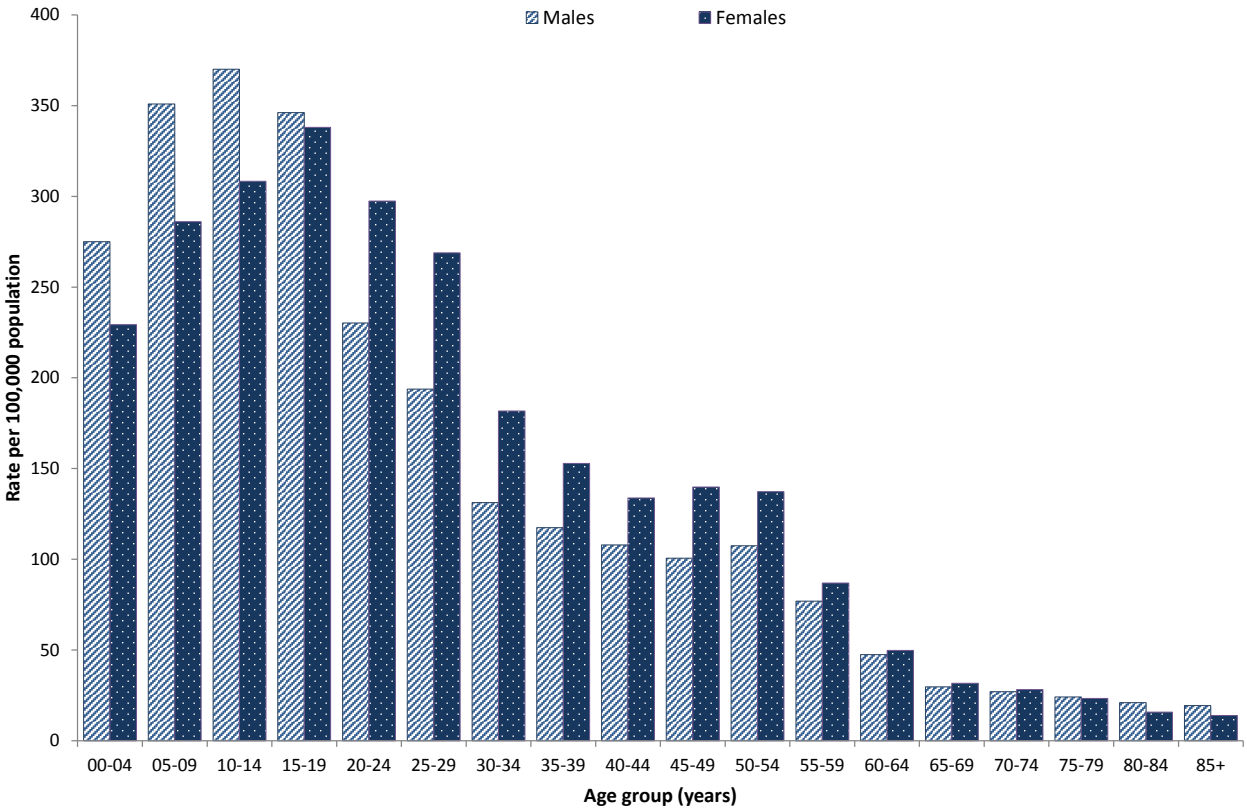

Source: NetEpi and NNDSS

**Figure 22: Map of laboratory confirmed cases of influenza A(H1N1)pdm09 rates, 2009, by Statistical Area level 3**



Source: NNDSS

**Figure 23: Rates of laboratory confirmed cases of influenza A(H1N1)pdm09, Australia, 2009, by age group and sex***




Source: NetEpi, NNDSS, EpiLog
*Excludes 71 cases where age or sex was not able to be determined

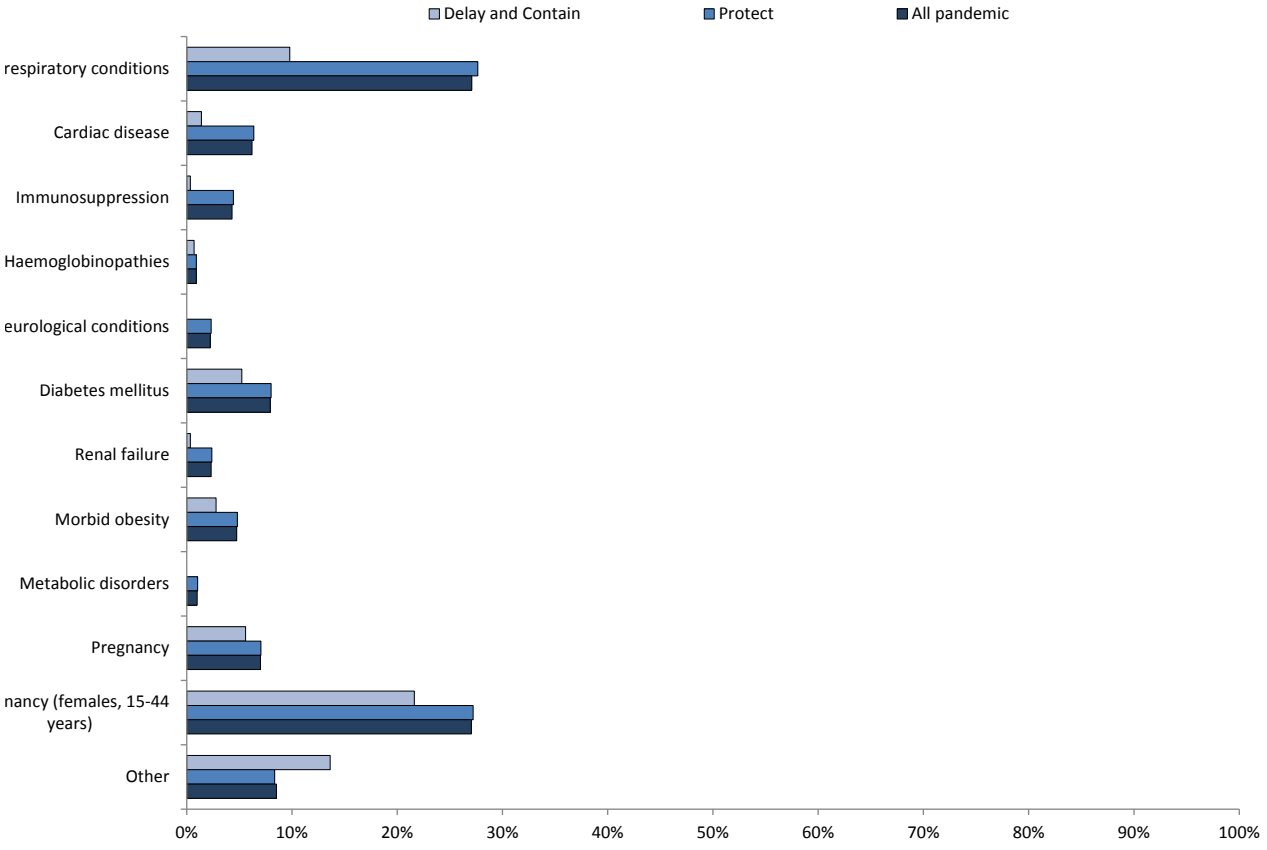
Whilst the main source of the virus' initial introduction into Australia is likely to have been through air travel; cruise ships[50] were identified as an important source of potential virus introduction, especially given that the duration of cruises can allow for multiple generations of influenza infections to develop.

## Timing and geographic distribution

At a national level, the duration of the main wave of the pandemic was around 18 weeks, from mid-May to late September, with notifications peaking in the week ending 24 July 2009 (n=5,829). The epidemic curve of influenza A(H1N1)pdm09 in comparison to all laboratory confirmed influenza notifications in 2009 is shown in Figure 20.

It should be noted while interpreting this epidemic curve that the number of confirmed cases reported are an underestimate of the true incidence of influenza A(H1N1)pdm09 infection during 2009. In the initial disease control phases of the pandemic, there was extensive diagnostic laboratory testing for influenza, and more specifically for the pandemic strain; this was also applied in varying extents in different settings and jurisdictions. From 3 June 2009, when Victoria moved to its MODIFIED SUSTAIN pandemic phase, and 17 June 2009, when Australia changed to the PROTECT pandemic phase, laboratory testing for pandemic influenza was targeted towards people with more severe disease and people more vulnerable to severe disease, although some sentinel testing to identify the levels of community transmission and viruses circulating also occurred. Additionally, although influenza testing of patients with ILI continued as part routine clinical care investigations, testing was less focussed on determining the specific type of influenza infection; this is highlighted by the divergence in the epidemic

**Figure 24: Risk factors[^] reported for confirmed influenza A(H1N1)pdm09 cases with enhanced surveillance data (n=8,838), Australia[*], 2009, by proportion[#] and pandemic phase[&]**




Source: NetEpi and EpiLog
[^] A case could report more than one risk factor.
[*] Northern Territory data represents hospitalised cases only
[#] Denominator includes all cases for which there is a valid response provided for the respective field.
[&] Queensland data represents all cases until 6 July 2009. Victoria moved to 'modified sustain' on 3 June 2009, data during the 'delay and contain' phases includes these cases.

curve (Figure 20) between notifications of all laboratory confirmed cases of influenza and the pandemic specific virus.

Throughout Australia, cases of influenza A(H1N1)pdm09 were not distributed homogenously, especially during the early phases of DELAY and CONTAIN. There was substantial variation in the incidence rates and peak times of the epidemic among states and territories (Figure 21). Sustained community transmission was initially established in Victoria, with most other jurisdictions following a fortnight later, however the rate of increase in this initial establishment phase varied by jurisdiction. The Northern Territory experienced the highest weekly rate of notified laboratory confirmed cases (124.3 per 100,000), followed by South Australia (89.6 per 100,000) and Queensland (67.2 per 100,000). Peak activity periods of the epidemic experienced by the jurisdictions ranged between the end of May and early August, with South Australia the last jurisdiction to experience their peak activity.

Analysis by Australian Statistical Geography Standard Statistical Area Level 3 of the cumulative rates of influenza A(H1N1)pm09 notifications show that rates were highest in the northern and central areas of Australia, followed by the capital cities, except Melbourne (Victoria) (Figure 22).

**Table 4: Risk factors[^] reported for confirmed influenza A(H1N1)pdm09 cases with enhanced surveillance data[#] (n=8,838) and the general population prevalence, Australia[*], 2009, by pandemic phase[&]**

| | DELAY and CONTAIN | | PROTECT | | ALL | | Population prevalence |
|---|---|---|---|---|---|---|---|
| | n | (%) | n | (%) | n | (%) | (%) |
| Total | 286 | | 8,552 | | 8,838 | | |
| **Underlying medical conditions[*&]** | | | | | | | |
| Chronic respiratory conditions | 28 | (9.8) | 2,366 | (27.7) | 2,394 | (27.1) | 12.3 |
| Cardiac disease | 4 | (1.4) | 545 | (6.4) | 549 | (6.2) | 5.2 |
| Immunosuppression | 1 | (0.3) | 380 | (4.4) | 381 | (4.3) | |
| Haemoglobinopathies | 2 | (0.7) | 78 | (0.9) | 80 | (0.9) | 1.8 |
| Neurological conditions | 0 | (0.0) | 198 | (2.3) | 198 | (2.2) | 1.7 |
| Diabetes mellitus | 15 | (5.2) | 686 | (8.0) | 701 | (7.9) | 4.0 |
| Renal failure | 1 | (0.3) | 205 | (2.4) | 206 | (2.3) | |
| Morbid obesity | 8 | (2.8) | 411 | (4.8) | 419 | (4.7) | 2.4[52] |
| Metabolic disorders | 0 | (0.0) | 89 | (1.0) | 89 | (1.0) | |
| Pregnancy | 16 | (5.6) | 603 | (7.1) | 619 | (7.0) | 1.3 |
| *Pregnancy (females, 15-44 years)* | *16* | *(21.6)* | *598* | *(27.2)* | *614* | *(27.0)* | *6.4* |
| Other | 39 | (13.6) | 714 | (8.3) | 753 | (8.5) | |
| *Alcoholism* | *1* | *(0.3)* | *44* | *(0.5)* | *45* | *(0.5)* | |
| *Blood cancers* | *3* | *(1.0)* | *50* | *(0.6)* | *53* | *(0.6)* | |
| *Downs Syndrome* | *2* | *(0.7)* | *11* | *(0.1)* | *13* | *(0.1)* | |
| *Epilepsy* | *0* | *(0.0)* | *29* | *(0.3)* | *29* | *(0.3)* | |
| *Hepatitis B or C or other liver disease* | *0* | *(0.0)* | *31* | *(0.4)* | *31* | *(0.4)* | |
| *Transplant history* | *0* | *(0.0)* | *32* | *(0.4)* | *32* | *(0.4)* | |
| *Smoking history* | *6* | *(2.1)* | *36* | *(0.4)* | *42* | *(0.5)* | *18.9* |
| **Number of medical conditions[*&]** | | | | | | | |
| None | 194 | (67.8) | 4,017 | (47.0) | 4,211 | (47.6) | |
| One | 75 | (26.2) | 3,282 | (38.4) | 3,357 | (38.0) | |
| Two | 12 | (4.2) | 877 | (10.3) | 889 | (10.1) | |
| Three | 5 | (1.7) | 288 | (3.4) | 293 | (3.3) | |
| Four or more | 0 | (0.0) | 88 | (1.0) | 88 | (1.0) | |

Source: NetEpi, EpiLog, NNDSS and the National Health Survey 2007-2008[42]
[^]A case could report more than one risk factor.
[*]Northern Territory data represents hospitalised cases only. No data provided for the field 'other' from Queensland.
[#]Denominator includes all cases with any data provided in the underlying medical conditions fields.
[&]Queensland data represents all cases until 6 July 2009. Victoria moved to 'modified sustain' on 3 June 2009, data during the 'delay and contain' phases includes these cases.

## Groups affected

During the DELAY and CONTAIN phases, including cases reported during Victoria's MODIFIED SUSTAIN phase, the median age of pandemic influenza A(H1N1)pdm09 cases was 17 years (IQR 12 – 29), however the median age of cases increased to 21 years (IQR 11-36) during the PROTECT phase. Throughout all phases of the pandemic, the 10-14 and 15-19 years age groups had the highest cumulative population incidence rates (339.0 and 340.3 per 100,000 respectively). The relatively low rates among adults aged 60 years and over is thought to be due to historical exposure to antigenically related influenza viruses earlier in their lives, resulting in the development of cross-protective antibodies.[51] There was an approximately equal distribution of cases by gender (51% female) overall, however there was variability in the ratio of males to females across each age group. There tended to be a notably higher proportion of males compared to females in younger populations, whereas the proportion of female cases tended to be higher in the 20 and 59 years age groups (Figure 23).

Valid underlying medical condition risk factor data were reported for almost a quarter of all influenza A(H1N1)pdm09 cases (23.4%; 8,838/37,754) during 2009.

### DELAY and CONTAIN phases

Of the cases reported during the DELAY and CONTAIN phases, including cases reported as part of Victoria's MODIFIED SUSTAIN phase, a third (32.2%; 92/286) of these cases reported at least one underlying medical condition (Table 4 and Figure 24).

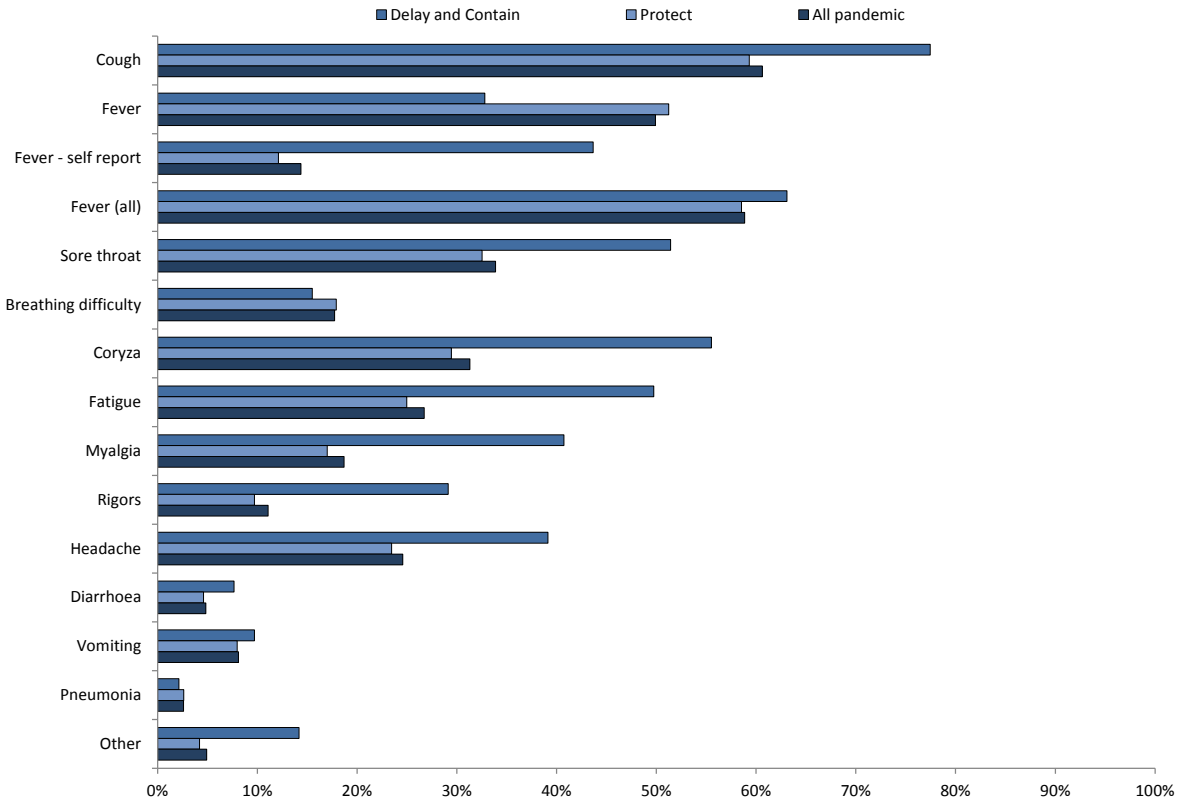
The most commonly reported underlying medical condition during these early phases were chronic respiratory conditions (9.8%), which included asthma and chronic obstructive pulmonary disease, followed by diabetes mellitus (5.2%). A total of 16 cases were pregnant (21.6%

**Table 5: Symptoms[^] reported for confirmed influenza A(H1N1)pdm09 cases with enhanced surveillance data[#] (n=15,723), Australia[*], 2009, by pandemic phase[&]**

| | Delay and Contain | | Protect | | All pandemic | |
|---|---|---|---|---|---|---|
| | n | (%) | n | (%) | n | (%) |
| Total | 1,122 | | 14601 | | 15723 | |
| **Symptoms** | | | | | | |
| Cough | 869 | (77.5) | 8,662 | (59.3) | 9,531 | (60.6) |
| Fever (all) | 708 | (63.1) | 8,546 | (58.5) | 9,254 | (58.9) |
| Sore throat | 577 | (51.4) | 4,749 | (32.5) | 5,326 | (33.9) |
| Breathing difficulty | 174 | (15.5) | 2,616 | (17.9) | 2,790 | (17.7) |
| Coryza | 623 | (55.5) | 4,299 | (29.4) | 4,922 | (31.3) |
| Fatigue | 558 | (49.7) | 3,646 | (25.0) | 4,204 | (26.7) |
| Myalgia | 457 | (40.7) | 2,483 | (17.0) | 2,940 | (18.7) |
| Rigors | 327 | (29.1) | 1,416 | (9.7) | 1,743 | (11.1) |
| Headache | 439 | (39.1) | 3,424 | (23.5) | 3,863 | (24.6) |
| Diarrhoea | 86 | (7.7) | 673 | (4.6) | 759 | (4.8) |
| Vomiting | 109 | (9.7) | 1,165 | (8.0) | 1,274 | (8.1) |
| Pneumonia | 24 | (2.1) | 384 | (2.6) | 408 | (2.6) |
| Other | 159 | (14.2) | 615 | (4.2) | 774 | (4.9) |

Source: NetEpi.
[^] A case could report more than one symptom. Fever combines the fields of a measured temperature of greater than 38°C and a self-reported history of fever.
[*] Northern Territory data represents hospitalised cases only
[#]Denominator includes all cases for which there is a valid response provided for the respective field.
[&]Queensland data represents all cases until 6 July 2009. Victoria moved to 'modified sustain' on 3 June 2009, data during the 'delay and contain' phases includes these cases.

**Figure 25: Symptoms[^] reported for confirmed influenza A(H1N1)pdm09 cases with enhanced surveillance data, Australia[*], 2009, by proportion[#] and pandemic phase[&]**




Source: NetEpi
[^] A case could report more than one symptom. Fever combines the fields of a measured temperature of greater than 38ºC and a self-reported history of fever.
[*] Northern Territory data represents hospitalised cases only
[#]Denominator includes all cases for which there is a valid response provided for the respective category.
[&]Queensland data represents all cases until 6 July 2009. Victoria moved to 'modified sustain' on 3 June 2009, data during the 'delay and contain' phases includes these cases.

of females aged between 15-44 years). Compared with the known general population prevalence of these medical conditions, the prevalence of influenza in these populations was much higher, especially among those with diabetes mellitus and pregnant women.

### PROTECT phase

During the PROTECT phase the proportion of cases with an underlying medical condition increased substantially to represent half of the confirmed cases during this phase (53.0%; 4,535/8,552), however given the known susceptibility and focus of case ascertainment towards those at risk populations, the increased proportion of cases reported during this phase is not unexpected. During this period the most common reported underlying medical condition continued to be chronic respiratory conditions (27.7%) and diabetes mellitus (8.0%).

### All 2009 pandemic phases

Analysis of the underlying medical conditions risk factor field 'other' for the whole period showed blood cancers (0.6%), alcoholism (0.5%) and a history of smoking (0.5%) to be additional risk factors also associated with infection. These risk factors are likely to be under reported in the dataset as information regarding these specific risk factors were not actively sought.

In comparison to the estimated population prevalence for some of the underlying medical conditions analysed, the proportion of cases with chronic respiratory conditions, morbid

**Table 6: Duration of symptoms[^] (days) reported for confirmed influenza A(H1N1)pdm09 cases with enhanced surveillance data[#] (n=1,189), Australia[*], 2009, by pandemic phase[&]**

| | Delay and Contain | | | Protect | | | All pandemic | | |
|---|---|---|---|---|---|---|---|---|---|
| Total | | | 346 | | | 843 | | | 1,189 |
| Symptom | Median (days) | IQR (days) | n | Median *(days)* | IQR (days) | n | Median (days) | IQR (days) | n |
| Cough | 4 | 2-7 | 269 | 5 | 3-7 | 542 | 5 | 3-7 | 811 |
| Fever all | 2 | 1-3 | 239 | 3 | 2-4 | 648 | 3 | 2-4 | 887 |
| Sore throat | 3 | 2-5 | 173 | 3 | 2-5 | 245 | 3 | 2-5 | 418 |
| Breathing difficulty | 3 | 2-4 | 65 | 3 | 2-5 | 257 | 3 | 2-5 | 322 |
| Coryza | 3 | 2-5 | 216 | 4 | 3-7 | 324 | 4 | 2-7 | 540 |
| Fatigue | 3 | 2-5 | 207 | 4 | 2-7 | 373 | 4 | 2-6 | 580 |
| Myalgia | 3 | 2-5 | 139 | 3 | 2-4 | 284 | 3 | 2-5 | 423 |
| Rigors | 2 | 1-3 | 110 | 2 | 2-4 | 232 | 2 | 2-3 | 342 |
| Headache | 2 | 1-4 | 154 | 3 | 2-5 | 277 | 3 | 2-5 | 431 |
| Diarrhoea | 1 | 1-2 | 37 | 2 | 1-3.25 | 148 | 2 | 1-3 | 185 |
| Vomiting | 1 | 1-2 | 50 | 1 | 1-3 | 192 | 1 | 1-2 | 242 |

Source: NetEpi.
[^] A case could report more than one symptom. Fever combines the fields of a measured temperature of greater than 38ºC and a self-reported history of fever.
[*] Cases from NSW (n=1,176), SA (n=12) and Vic (n=1).
[#]Denominator includes all cases for which there is a valid response provided for the respective field.
[&]Queensland data represents all cases until 6 July 2009. Victoria moved to 'modified sustain' on 3 June 2009, data during the 'delay and contain' phases includes these cases.

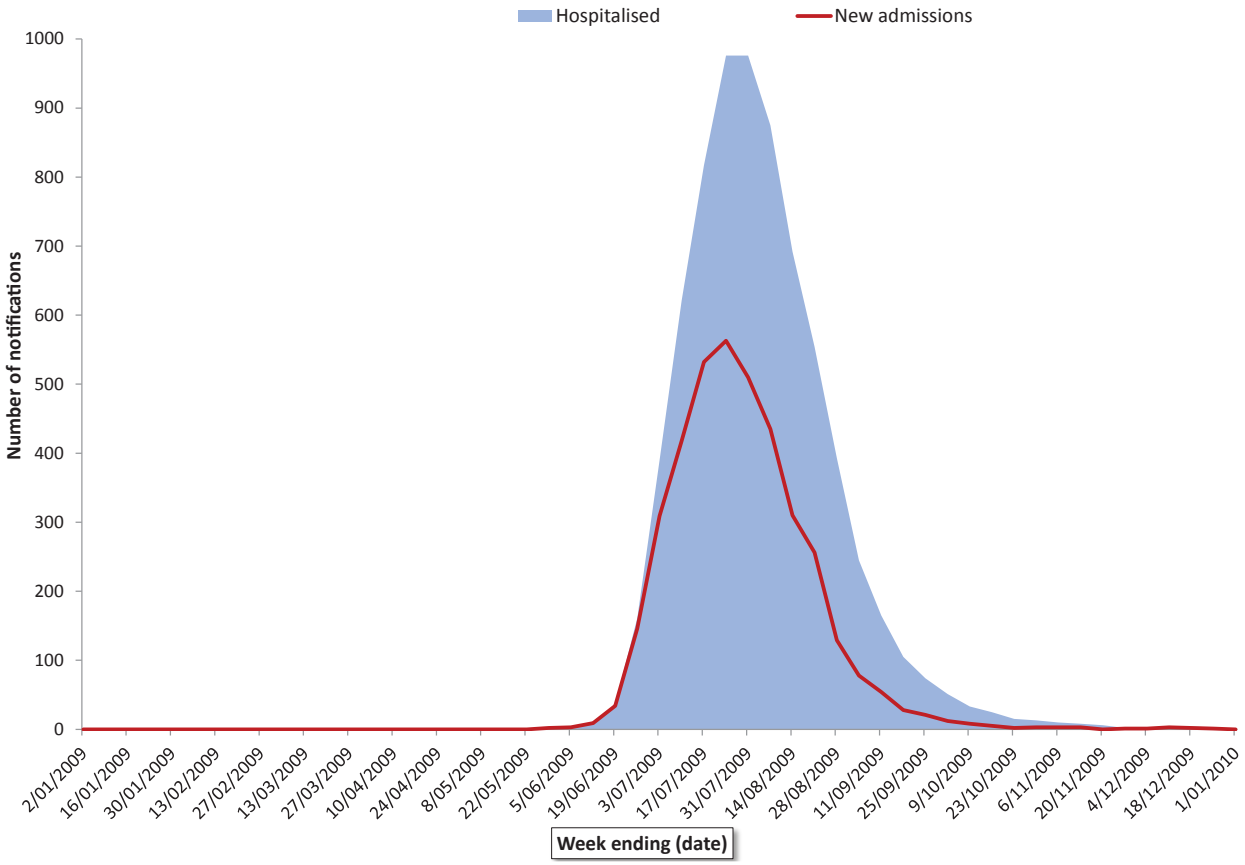
obesity and who were pregnant was much higher than the expected population prevalence for these conditions (Table 4).

Of females aged 15-44 years, 27.0% were reported as pregnant (n=614). Additionally, the proportion of cases among Indigenous Australians increased as the pandemic progressed and represented 10.5% of all influenza A(H1N1)pdm09 cases in 2009 (Table 12). Further analysis regarding the burden of influenza among Indigenous Australians and pregnant women are provided in the section '*Specific risk group analysis*'.

## Clinical presentation

Analysis of enhanced surveillance data (n=15,723) during the DELAY and CONTAIN phases, including Victoria's MODIFIED SUSTAIN phase, and also the PROTECT phase showed that infection with influenza A(H1N1) pdm09 caused a broad spectrum of symptoms, with most cases experiencing symptoms consistent with seasonal influenza infection. The prevalence of the majority of symptoms appeared to be higher during the DELAY and CONTAIN pandemic phases. This is likely due to a change in case ascertainment and followup strategies associated with the move to the PROTECT pandemic phase, however completeness of these may have been affected by capacity for public health follow-up. Overall, cough and fever, appeared to be the most commonly reported symptoms, followed by sore throat, coryza and fatigue (Figure 25 and Table 5). The relatively high rates of fever and cough symptoms may also be associated with the clinical presentation criteria for laboratory testing and case definition (Appendix 2 and Appendix 3), which depending on the epidemiological linkage of a case to a confirmed case, required the presence of acute respiratory disease and fever. Gastrointestinal symptoms, such as vomiting and diarrhoea, were reported in 10.7% of cases and were generally considered to occur more frequently, especially among adults, in comparison to seasonal influenza.[51] Compared with some other countries or regions, however, the prevalence of fever and gastrointestinal symptoms reported among Australian cases appeared to be lower.[51,53] Analysis of the 'other symptoms' free-text field showed symptoms such as nausea (0.7%); chest pain (0.5%); dizziness (0.4%); abdominal pain (0.3%); and ear aches (0.3%) were also reported amongst cases.

**Figure 26: Number of hospitalised cases with confirmed influenza A(H1N1)pdm09[#], Australia, 2009, by admission date, hospitalised and week[*]**




Source: NetEpi and EpiLog
[*]Where admission date is greater than 7 days prior to influenza onset date, influenza onset date was used.
[#]Excludes 1,203 cases where hospital admission date was not reported.

Although the duration of a cases illness as a marker of severity was not able to be determined from the dataset, the duration of each symptom, excluding pneumonia, was able to be analysed. Data on the duration of each symptom was reported for 7.6% of cases with clinical presentation data (1,189/15,723), with the majority of these cases from New South Wales (98.9%; 1,176/1,189) and a small number of cases from South Australia (n=12) and Victoria (n=1). The symptom with the highest median duration throughout all phases of the pandemic was cough (5 days; IQR 3-7), followed by coryza (4 days; IQR 2-7) and fatigue (4 days; IQR 2-6) (Table 6).

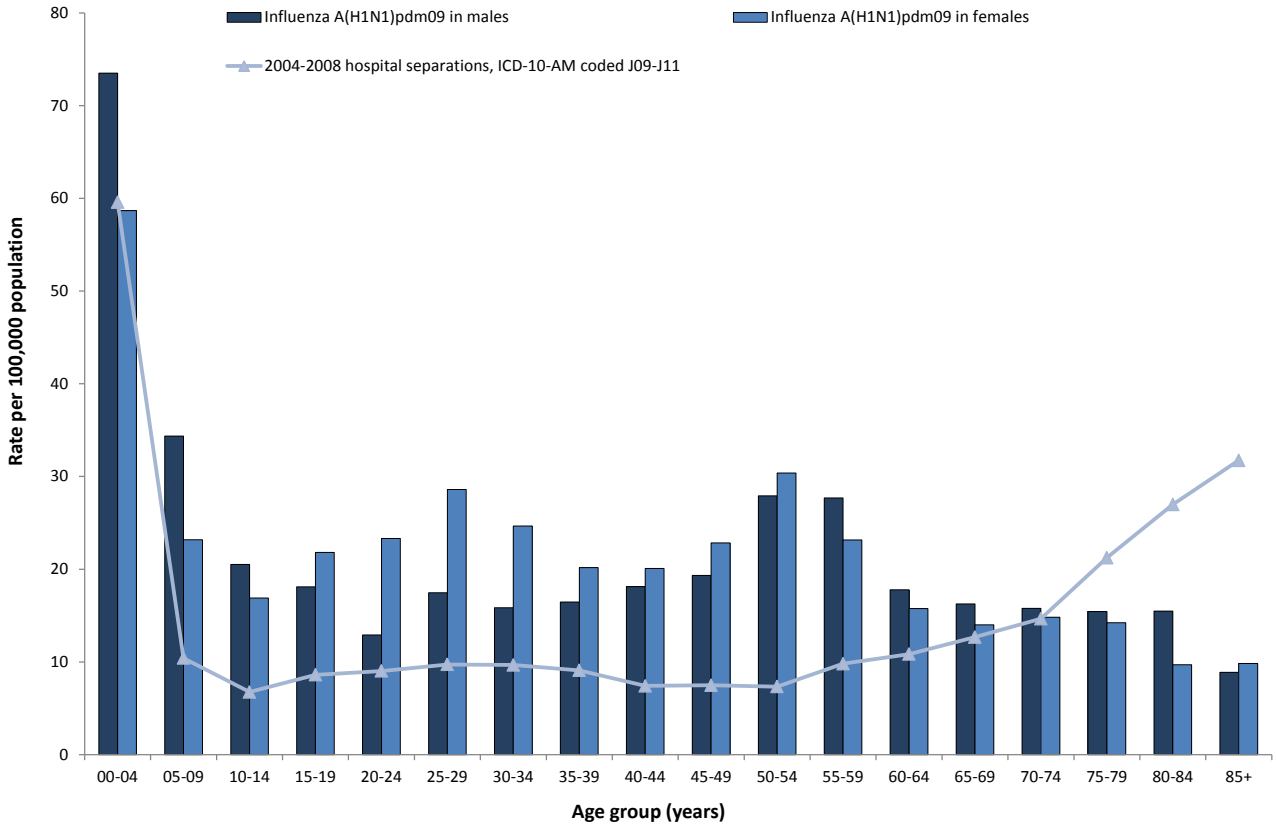
## Severity and complications

The greatly increased rate of testing, and changes to thresholds for diagnostic testing for influenza, especially influenza A(H1N1)pdm09, in the protocols of the pandemic phases, have made the assessment of overall disease burden problematic, especially in comparison with previous influenza seasons. Although a large number of mild cases were identified at the community level, particularly in the early stages of the pandemic where containment measures were being attempted, severe cases of pandemic influenza were also reported. Three indicators of progressively increasing severity have been utilised for analysis in this report: (i) hospitalisation; (ii) intensive-care unit admission; and (iii) mortality.

### Hospitalisations

In 2009, there were 5,085 cases of influenza A(H1N1)pdm09 reported as being hospitalised in Australia. This figure included both cases where influenza A(H1N1)pdm09 was their primary diagnosis, as well as those where infection

**Figure 27: Rates of hospitalisation with confirmed influenza A(H1N1)pdm09 in 2009* and average annual rates of hospital separations for all diagnoses† ICD-10-AM coded J09-J11 for 2004-2008, Australia, by age group**




Source: NetEpi, EpiLog and Admitted Patient Care Data collection 2003-04 to 2008-09
* Excludes 3 cases for whom age or sex were not reported
† Includes both principal diagnosis and additional diagnoses. Where principal diagnosis is the diagnosis established after study to be chiefly responsible for occasioning a patient's service event or episode, as represented by a code; and additional diagnoses are defined as condition or complaint either coexisting with the principal diagnosis or arising during the episode of admitted patient care, episode of residential care or attendance at a health care establishment, as represented by a code.

with the virus was not the primary diagnosis. The number of hospitalisations equated to an overall crude rate of 23.4 per 100,000 population.

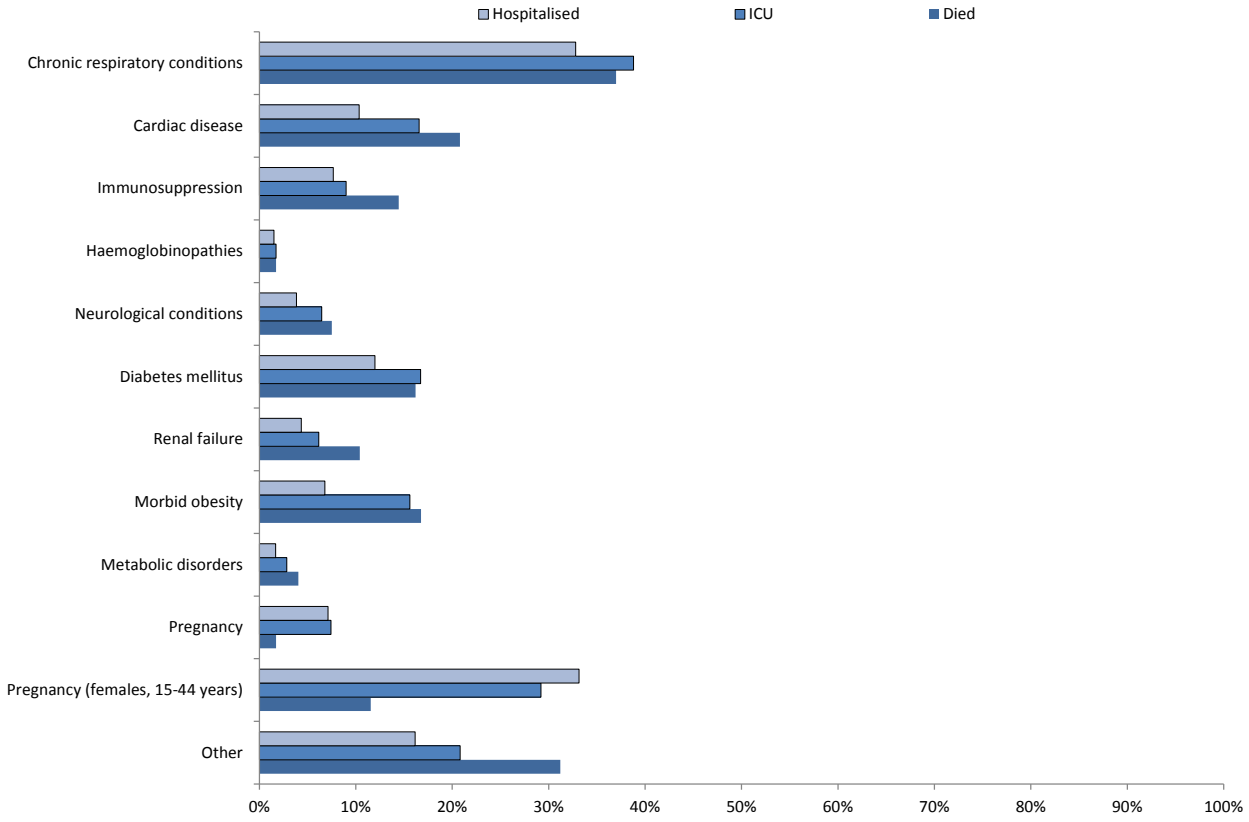
The number of hospital admissions peaked in the week ending 24 July 2009 at 562 (Figure 26). During this peak week of hospital admissions, there were over 970 people with confirmed influenza A(H1N1)pdm09 being cared for in hospital, representing 1.2% of beds available in both private and public acute hospitals or 1.8% in public acute hospitals only (974/79,636 and 974/54,338 respectively).[54]

### Groups affected

The median age of hospitalised cases was 30 years (IQR 8-50). Rates of hospitalisation were highest among children aged less than 5 years (66.3 per 100,000 population), with sub-peaks observed in the 5-9, 25-29 and 50-54 years age groups (28.9, 23.0, 29.2 per 100,000 respectively) (Figure 27). There was an approximately equal distribution of hospitalised cases by gender (51% female), however hospitalisation rates were markedly higher among males aged less than 10 years and lower among females in the 20-34 years age range.

Similar to previous seasonal influenza epidemics, hospitalisation rates associated with pandemic influenza in 2009 remained highest among children aged less than 5 years, however, in contrast to previous seasons, hospitalisation in 2009 was also more common among those aged 50-59 years and lower amongst those aged

**Figure 28: Risk factors reported for cases with laboratory confirmed influenza A(H1N1) pdm09 with enhanced surveillance data, Australia, 2009, by hospitalisation and mortality status and proportion**




Source: NetEpi and EpiLog

75 years and over (Figure 27). The age distribution of hospitalised cases appears markedly different from the trends observed amongst all notified cases (Figure 23).

Valid underlying medical condition risk factor data were reported for 84.5% (4,297/5,085) of hospitalised cases during 2009, and over two-thirds (68.6%; 2,947/4,297) of these cases reported at least one underlying medical condition (Table 7). The most common reported underlying medical condition was chronic respiratory conditions (38.6%; 1,409/3,469), with diabetes mellitus (15.1%; 515/3,418) and cardiac disease (13.2%; 445/3,378) also common. Analysis of the underlying medical conditions risk factor field 'other' showed blood cancers (2.7%, 52/1,898), including leukaemia, lymphoma and myeloma; alcoholism (2.3%; 44/1,898) and a history of smoking (2.1%; 40/1,898) to be common risk factors associated with pandemic influenza associated hospitalisations (Table 7).

A total of 306 hospitalised cases were pregnant. Among hospitalised females aged 15-44 years, pregnancy accounted for 28.7% (302/1,054) of these cases and 39.4% of cases with valid data (302/767). Sixteen per cent of patients admitted to hospital with confirmed influenza A(H1N1)pdm09 were Indigenous Australians (Table 11).

### Severity

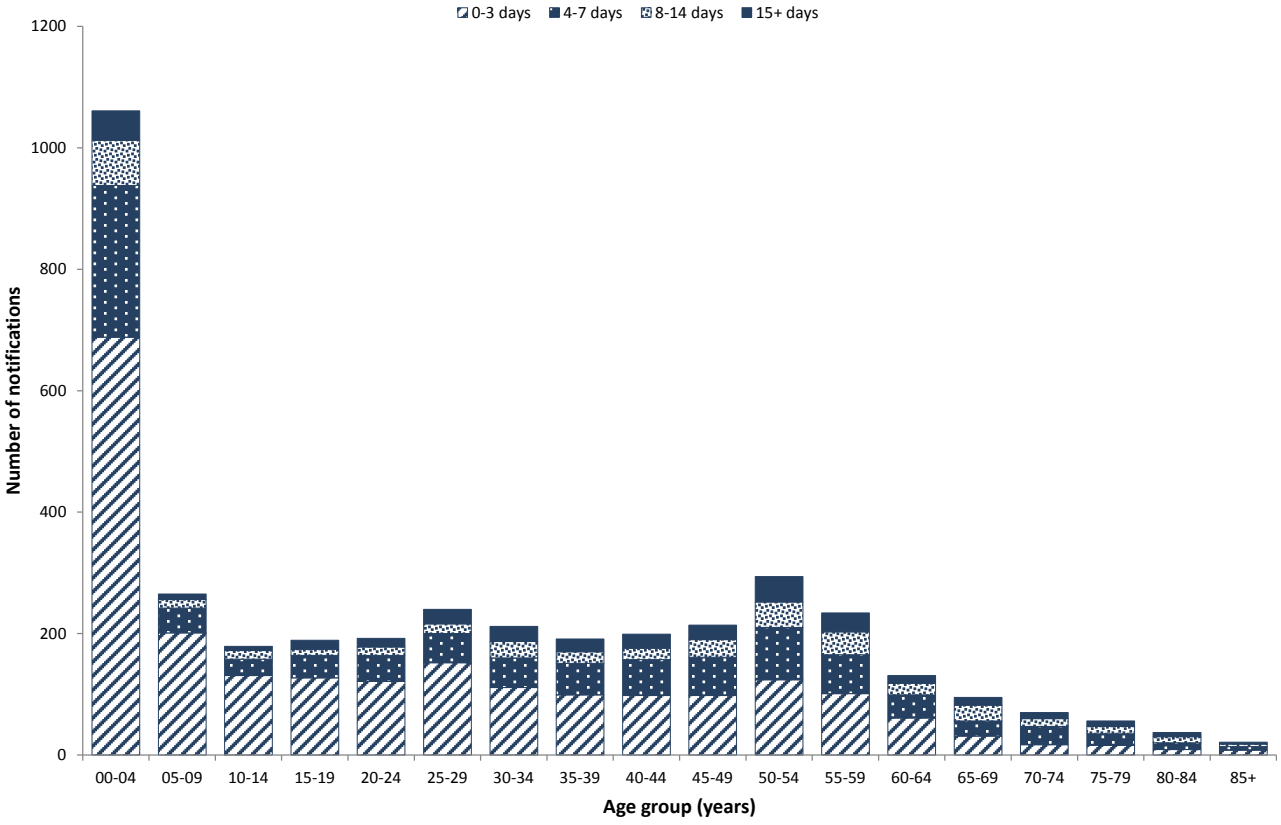
Information on length of stay was available for 69% (3,881/5,085) of hospitalised cases, which includes those cases also admitted to an ICU. A case may have already been hospitalised due to another condition; therefore if the period between date of admission and onset was greater than 7 days, date on onset was used. Additionally, if no discharge date or date of death was provided, the case was considered to have been hospitalised for less than one day.

**Table 7: Risk factors reported for cases with laboratory confirmed influenza A(H1N1)pdm09 with enhanced surveillance data, Australia, 2009, by hospitalisation and mortality status and proportion**

| | Hospitalised | | ICU | | Died | |
|---|---|---|---|---|---|---|
| | n | (%) | n | (%) | n | (%) |
| Total | 5,085 | | 686 | | 188 | |
| Total with underlying medical conditions data | 4,297 | (84.5) | 634 | (92.4) | 173 | (92.0) |
| **Age (years)** | | | | | | |
| 00-04 | 1,246 | (24.5) | 31 | (4.9) | 4 | (2.1) |
| 05-09 | 373 | (7.3) | 20 | (3.1) | 2 | (1.1) |
| 10-14 | 243 | (4.8) | 16 | (2.5) | 5 | (2.7) |
| 15-19 | 276 | (5.4) | 23 | (3.6) | 3 | (1.6) |
| 20-24 | 262 | (5.2) | 26 | (4.1) | 8 | (4.3) |
| 25-29 | 344 | (6.8) | 55 | (8.6) | 12 | (6.4) |
| 30-34 | 277 | (5.4) | 48 | (7.5) | 7 | (3.7) |
| 35-39 | 258 | (5.1) | 57 | (8.9) | 14 | (7.4) |
| 40-44 | 260 | (5.1) | 48 | (7.5) | 18 | (9.6) |
| 45-49 | 290 | (5.7) | 57 | (8.9) | 17 | (9.0) |
| 50-54 | 381 | (7.5) | 86 | (13.5) | 19 | (10.1) |
| 55-59 | 311 | (6.1) | 75 | (11.8) | 28 | (14.9) |
| 60-64 | 175 | (3.4) | 30 | (4.7) | 12 | (6.4) |
| 65-69 | 128 | (2.5) | 28 | (4.4) | 8 | (4.3) |
| 70-74 | 98 | (1.9) | 16 | (2.5) | 11 | (5.9) |
| 75-79 | 78 | (1.5) | 16 | (2.5) | 10 | (5.3) |
| 80-84 | 50 | (1.0) | 5 | (0.8) | 5 | (2.7) |
| 85+ | 33 | (0.6) | 1 | (0.2) | 5 | (2.7) |
| Unknown | 2 | (0.0) | 0 | (0.0) | 0 | (0.0) |
| **Underlying medical conditions** | | | | | | |
| Chronic respiratory conditions | 1,409 | (32.8) | 246 | (38.8) | 64 | (37.0) |
| Cardiac disease | 445 | (10.4) | 105 | (16.6) | 36 | (20.8) |
| Immunosuppression | 329 | (7.7) | 57 | (9.0) | 25 | (14.5) |
| Haemoglobinopathies | 65 | (1.5) | 11 | (1.7) | 3 | (1.7) |
| Neurological conditions | 165 | (3.8) | 41 | (6.5) | 13 | (7.5) |
| Diabetes mellitus | 515 | (12.0) | 106 | (16.7) | 28 | (16.2) |
| Renal failure | 187 | (4.4) | 39 | (6.2) | 18 | (10.4) |
| Morbid obesity | 292 | (6.8) | 99 | (15.6) | 29 | (16.8) |
| Metabolic disorders | 72 | (1.7) | 18 | (2.8) | 7 | (4.0) |
| *Pregnancy* | *306* | *(7.1)* | *47* | *(7.4)* | *3* | *(1.7)* |
| Pregnancy (females, 15-44 years) | 302 | (33.2) | 47 | (29.2) | 3 | (11.5) |
| *Other* | *694* | *(16.2)* | *132* | *(20.8)* | *54* | *(31.2)* |
| *Alcoholism* | *44* | *(1.0)* | *13* | *(2.1)* | *5* | *(2.9)* |
| *Blood cancers* | *52* | *(1.2)* | *5* | *(0.8)* | *12* | *(6.9)* |
| *Downs Syndrome* | *11* | *(0.3)* | *2* | *(0.3)* | *1* | *(0.6)* |
| *Epilepsy* | *26* | *(0.6)* | *4* | *(0.6)* | *1* | *(0.6)* |
| *Hepatitis B or C or other liver disease* | *30* | *(0.7)* | *9* | *(1.4)* | *2* | *(1.2)* |
| *Transplant history* | *29* | *(0.7)* | *8* | *(1.3)* | *3* | *(1.7)* |
| *Smoking history* | *40* | *(0.9)* | *11* | *(1.7)* | *3* | *(1.7)* |
| **Number of medical conditions** | | | | | | |
| None | 1,350 | (31.4) | 122 | (19.2) | 64 | (37.0) |
| One | 1,859 | (43.3) | 265 | (41.8) | 75 | (43.4) |
| Two | 747 | (17.4) | 140 | (22.1) | 34 | (19.7) |
| Three | 260 | (6.1) | 81 | (12.8) | 28 | (16.2) |
| Four or more | 81 | (1.9) | 26 | (4.1) | 12 | (6.9) |

Source: NetEpi and EpiLog

**Figure 29: Number of hospitalised cases with confirmed cases of influenza A(H1N1)pdm09[#], Australia, 2009, by age group and duration hospitalised[*]**




Source: NetEpi and EpiLog
[*]Where admission date is greater than 7 days prior to influenza onset date, influenza onset date used.
[#] Excludes 1,205 cases where duration hospitalised and/or age were not able to be determined.

The median length of hospitalisation was 3 days (IQR 2-6 days). Approximately 19% of hospitalised cases were hospitalised for a period of greater than 7 days. Although children aged less than 5 years were more likely to be hospitalised, their duration of hospitalisation tended to be shorter in comparison to older children and adults. Almost 9% (68/760) of children aged less than 5 years were hospitalised for a period of greater than 7 days, compared to over a quarter (26.5%; 521/1,967) among those aged 30 years and over (Figure 29); suggesting that hospitalisations in older children and adults were relatively more severe than in younger children aged less than 5 years. This finding is consistent with the observed upward trend in the median age of the various severity indices (Table 7).

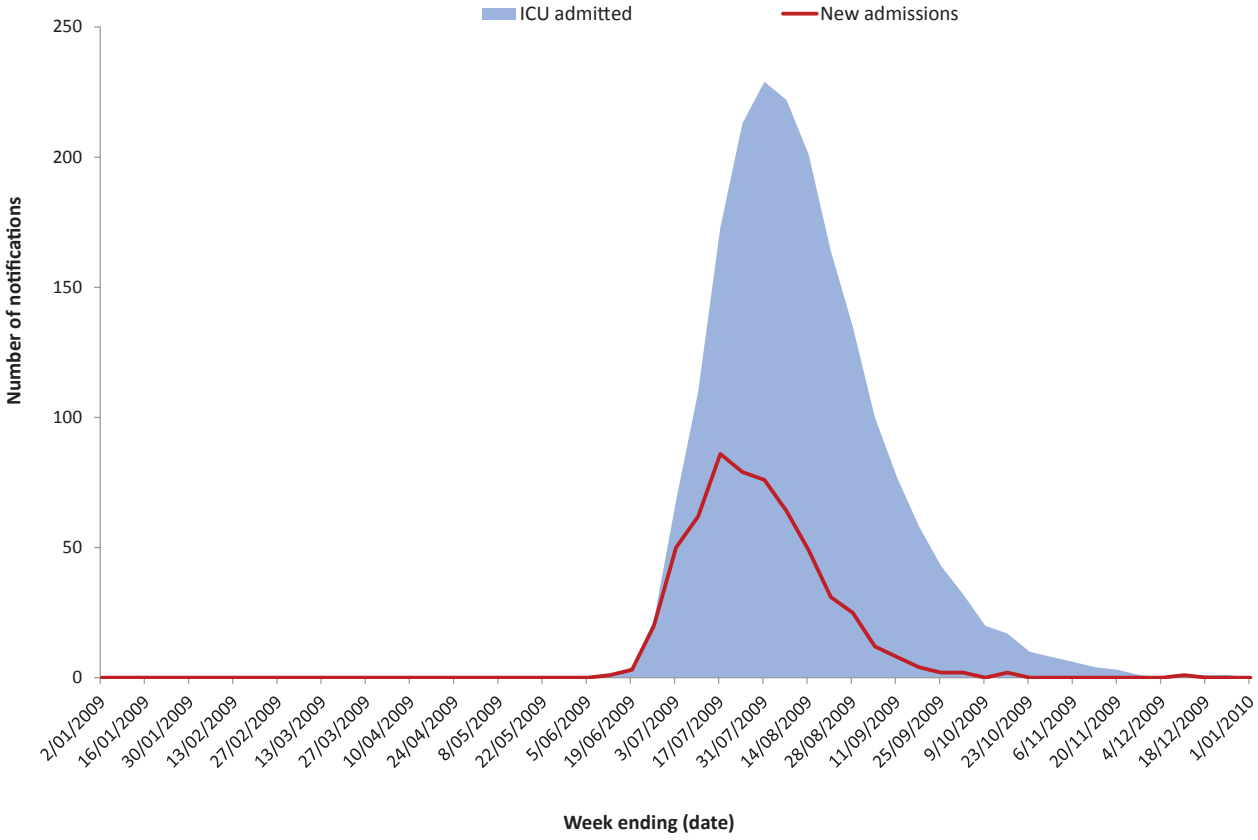
Of the hospitalised cases with clinical presentation data (3,181/5,085), three-quarters of cases presented with cough or fever (74.5% and 76.3% respectively) and almost half with breathing difficulty (45.6%). Twelve per cent of cases (371/3,181) presented with pneumonia. Data on the duration of each symptom, excluding pneumonia, were reported for a third of the hospitalised cases reported from New South Wales (467/1,430). The symptoms with the highest median duration, 5 days, were cough (IQR 3-9) and fatigue (IQR 3-7). The duration of pneumonia was able to be estimated for New South Wales cases hospitalised where pneumonia onset and hospital discharge dates were provided, and the date of discharge was greater than pneumonia onset (n=179). Of these cases, the median duration of hospitalisation for confirmed cases admitted with pneumonia was estimated to be 6 days (IQR 3-13).

**Table 8: Comparison of NetEpi* and ANZICS ICU admissions, Australia, 2009, by state or territory**

| | ACT | NSW | NT | Qld | SA | Tas | Vic | WA | Total |
|---|---|---|---|---|---|---|---|---|---|
| NetEpi* | 1 | 262 | 48 | 187 | 100 | 9 | - | 79 | **686** |
| ANZICS | 12 | 254 | 27 | 162 | 72 | 8 | 106 | 77 | **718** |

Source: NetEpi, EpiLog and ANZICS
* NetEpi cases includes cases reported through Qld's EpiLog system

**Figure 30: Number of laboratory confirmed influenza A(H1N1)pdm09 admitted to an intensive-care unit#, Australia, 2009, by week* and admission status**




Source: NetEpi and EpiLog
# Excludes 109 cases were hospital admission date was not reported.
*Duration of stay could incorporate periods of care where the case was not in an ICU, but was still hospitalised. Where admission date is greater than 7 days prior to influenza onset date, influenza onset date used.

## Intensive-care unit admission

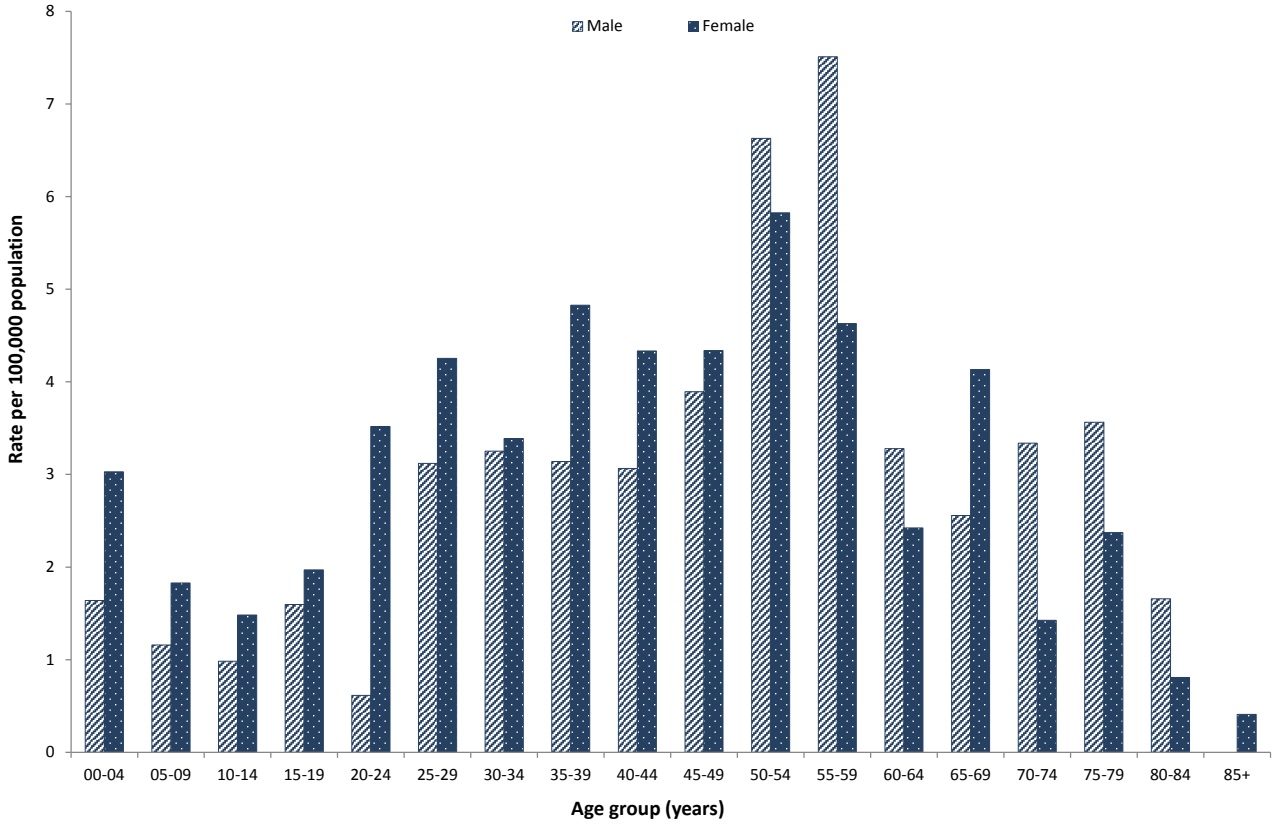
Cases admitted to intensive-care units (ICUs) represented 686 of the 5,085 (13.5%) hospitalised cases with confirmed influenza A(H1N1) pdm09 that were reported through NetEpi and Queensland's EpiLog system. However, Victoria and the ACT are not represented in the NetEpi dataset as these jurisdictions used the ANZICS system to capture ICU admissions data (Table 8) and an analysis of this dataset is provided later in this report.

Hospital admissions requiring intensive care peaked during the week ending 17 July 2009 with 86 admissions, and by the end of July there was a subsequent peak week in the number of people hospitalised who required intensive care at 229 (Figure 30).

### Groups affected

Cases admitted to ICU were more likely to be older compared to general hospital admissions, with a median age of 44.5 years (IQR 28-55) compared to 30 years (IQR 8-50). The peak

**Figure 31: Rates of intensive-care unit admissions with laboratory confirmed influenza A(H1N1)pdm09, Australia, 2009, by sex and age group**




Source: NetEpi and EpiLog

occurrence of admissions to ICU occurred in the 50-54 years age group (n=89) (Figure 31) and the proportion of hospitalised cases admitted to ICU were highest in the 30-59 years age groups (range 19.4 to 23.9%). Fifty-three per cent (367/686) of admission to ICU were female, with rates of admissions generally higher among females aged less than 50 years. Rates of ICU admissions peaked among females in the 50-54 years age group (5.8 ICU admissions per 100,000 population), compared to the 55-59 years age group for males (7.5 ICU admissions per 100,000 population) (Figure 31).

Valid underlying medical condition risk factor data were reported for 92% (634/686) of cases admitted to an ICU, with over 80% (512/634) of these cases reporting at least one underlying medical condition (Table 7). Consistent with all hospitalised cases, the most common reported underlying medical condition associated with intensive care unit admission was chronic respiratory conditions (44.0%; 246/559), with diabetes mellitus (20.4%; 106/519) and cardiac disease (20.3%; 105/518) also common. Additionally, cases reported as being morbidly obese was a common underlying medical condition risk factor (19.0%; 99/520).

Analysis of the underlying medical conditions risk factor field 'other' identified alcoholism (4.4%; 13/298) and a history of smoking (3.7%; 11/298) as common risk factors associated with pandemic influenza associated hospitalisations. This quantification is a likely underestimate as information regarding these specific risk factors were not systematically collected or measured (Table 7 and Figure 28).

A total of 47 cases admitted to an ICU were pregnant, and among ICU admitted females aged 15-44 years, pregnant women accounted for just over a quarter of these cases (27.5%; 47/171). Just over 14% of patients admitted to an ICU with confirmed influenza A(H1N1)pdm09 were Indigenous Australians (Table 11).

Overall, the proportion of cases admitted to an ICU and who were pregnant, had chronic lung disease, had a BMI of 40 or more, or were Indigenous were all higher than the corresponding distribution of these risk factors in the general population.

### Severity

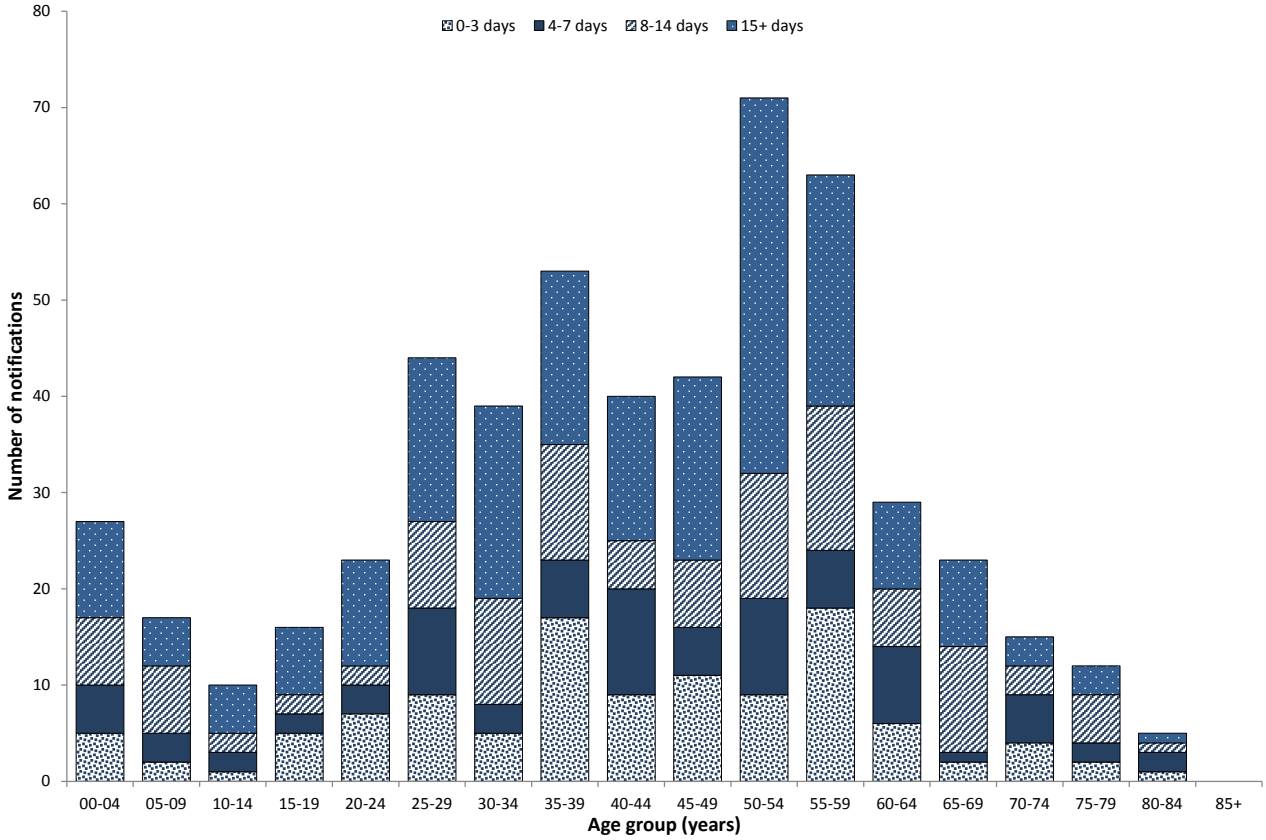
Information on the duration of hospitalisation for cases admitted to an ICU at some point during their hospitalisation was available for 84% (577/686) cases. It should be noted that the calculation of duration of stay could incorporate periods of care where the case was not in an ICU, but was still hospitalised. The median duration of hospitalisation for cases admitted to an ICU was 13 days (IQR 4-22), which, as expected, was substantially longer than compared to hospitalised cases. Among those in the age groups of less than 70 years admitted to an ICU, more than half of the cases in each of these age groups were hospitalised for greater than 7 days; peaking at 87.5% for cases in the 65-69 years age group (Figure 32).

A total of 361 cases admitted to hospital required mechanical ventilation, with the majority of these cases (n=352) admitted to an ICU and representing over half of all ICU admissions (51.3%; 352/686). The median age of cases requiring ventilation was 47 years (range 0 to 77), with the age distribution of cases similar to those requiring ICU admission with peaks among older adults (50-59 years) (Figure 33).

### Australian and New Zealand Intensive Care Society (ANZICS)

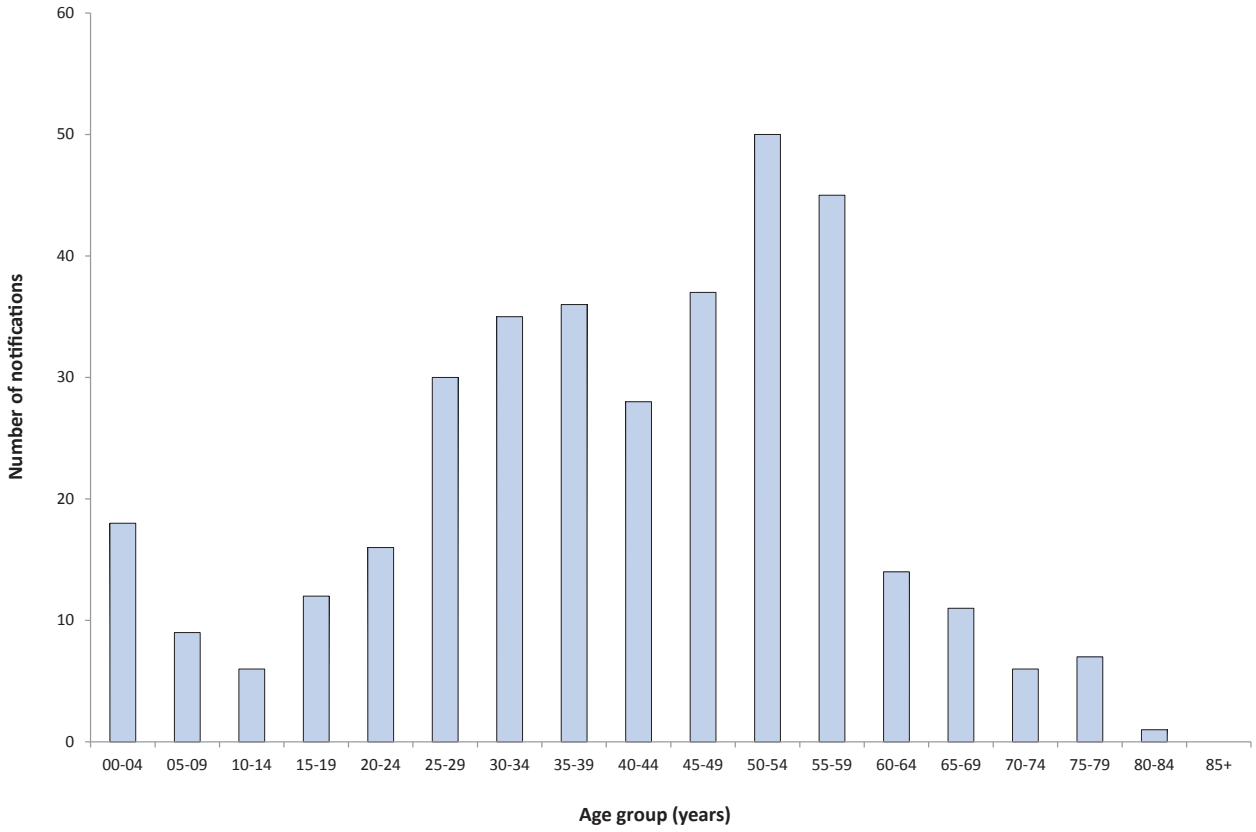
Data on influenza A(H1N1)pdm09 patients admitted to Australian ICUs were also col-

**Figure 32: Number of laboratory confirmed influenza A(H1N1)pdm09 admitted to an intensive-care unit[#], Australia, 2009, by age group and duration hospitalised[*]**




Source: NetEpi and EpiLog
[#] Excludes 109 cases where duration hospitalised and/or age were not able to be determined.
[*] Duration of stay could incorporate periods of care where the case was not in an ICU, but was still hospitalised. Where admission date is greater than 7 days prior to influenza onset date, influenza onset date used.

**Figure 33: Number of hospitalised cases with confirmed influenza A(H1N1)pdm09 who required mechanical ventilation, Australia, 2009, by age group**




Source: NetEpi and EpiLog

lected by the Australian and New Zealand Intensive Care Society (ANZICS) and provided to the Department. During 2009, 718 cases were admitted to an ICU with confirmed influenza A(H1N1)pdm09 (Table 9), an additional 32 cases in comparison to the number of cases reported as being admitted to an ICU in the NetEpi dataset (n=686). As noted previously, Victoria and the ACT were not represented in the NetEpi dataset as these jurisdictions used the ANZICS system to record ICU admissions. Additionally, based on the differences in case counts between the two systems by jurisdiction, the additional ICU cases in ANZICS are unlikely to reflect missing influenza A(H1N1)pdm09 notifications, rather cases whose ICU status was either not captured in NetEpi or their residential jurisdiction was different from the jurisdiction in which they were admitted to an ICU (Table 8).

The median duration of treatment in an ICU was 7 days (IQR 3-15); and an overall median duration of hospitalisation of 14 days (IQR 6-26), similar to the findings in the NetEpi combined dataset. The number of cases being cared for concurrently in an ICU peaked in the week ending 31 July 2009 with a median of 153 cases per day (range 147 to 157) (Figure 34). Based on available intensive care bed stocks across Australia in 2009,[55] the proportion of ICU beds occupied by influenza A(H1N1)pdm09 cases nationally during this peak week was around 10%. However, as the peak timing and intensity of the pandemic varied by jurisdiction, the peak percentage of ICU beds occupied by these cases across the jurisdictions ranged from 5.1 to 38.3%.

In comparison to hospitalisations captured in the NetEpi combined dataset, the age distribution of influenza A(H1N1)pdm09 ICU admissions varied substantially; with the median age of ICU admissions being much older (30 versus 42 years respectively) (Figure 35). Figure 35 shows the age distribution of ICU admissions by risk factor category and highlights that the proportion of cases with a risk factor gener-

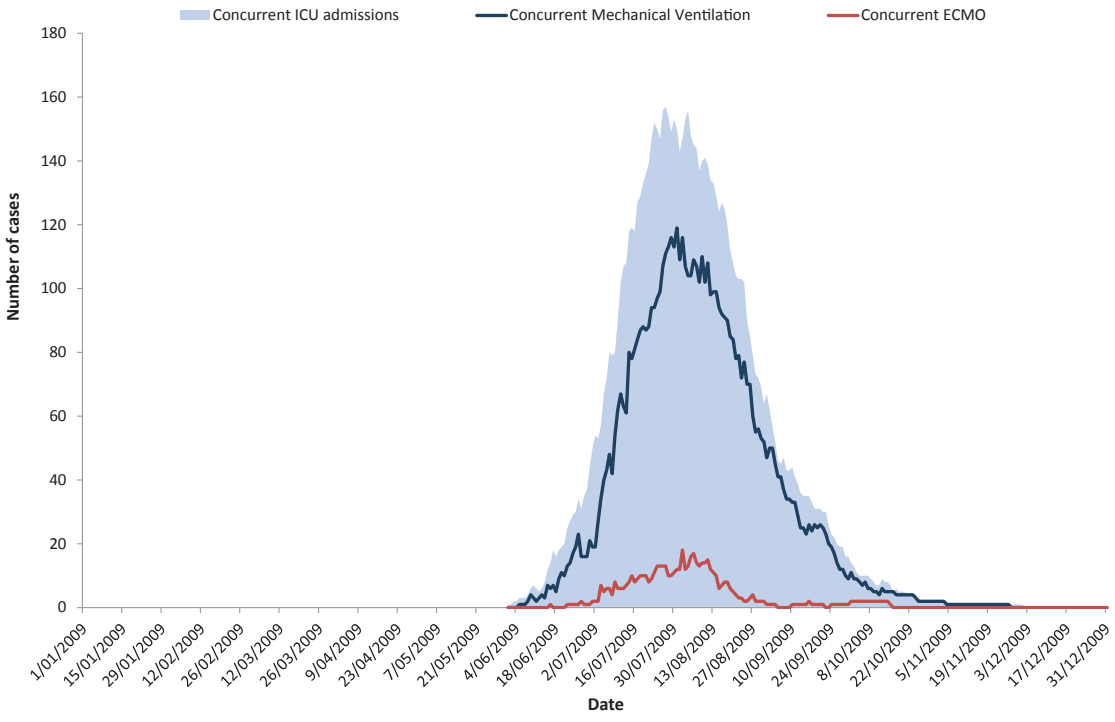
## Table 9: Characteristics of cases admitted to an intensive care unit with confirmed influenza A(H1N1)pdm09 (n=718), Australia, 2009

| | ICU admission | | Mechanical ventilation | | ECMO | |
|---|---|---|---|---|---|---|
| Total | 718 | | 481 | | 54 | |
| **Age (years)** | | | | | | |
| Median | 42 | | 40 | | 35 | |
| IQR | 27-54 | | 27-53 | | 27-44 | |
| **Sex** | | | | | | |
| Male (%) | 348 | (48.5) | 231 | (48.0) | 24 | (44.4) |
| Female (%) | 370 | (51.5) | 250 | (52.0) | 30 | (55.6) |
| **Risk factor** | | | | | | |
| Pregnant (%) | 62 | (8.6) | 42 | (8.7) | 8 | (14.8) |
| Indigenous (%) | 73 | (10.2) | 45 | (9.4) | 3 | (5.6) |
| BMI ≥40kg/m$^2$ (%) | 79 | (11.0) | 64 | (13.3) | 9 | (16.7) |
| Diabetes mellitus (%) | 115 | (16.0) | 76 | (15.8) | 8 | (14.8) |
| Chronic lung disease (%) | 246 | (34.3) | 145 | (30.1) | 17 | (31.5) |
| Chronic heart failure (%) | 87 | (12.1) | 48 | (10.0) | 3 | (5.6) |
| APACHE III co-morbidity (%)* | 213 | (29.7) | 134 | (27.9) | 7 | (13.0) |
| **Duration (days)** | | | | | | |
| Median | 7 | | 8 | | 8 | |
| IQR | 3-15 | | 3-17 | | 4-13 | |
| **Outcome** | | | | | | |
| Died (%) | 110 | (15.3) | 94 | (19.5) | 8 | (14.8) |

Source: Australia and New Zealand Intensive Care Society

* Acute physiological, age, chronic health evaluation 3rd revision co-morbidities: *Adults aged≥16 years* – AIDS, hepatic failure, lymphoma, metastatic carcinoma, leukaemia or myeloma, cirrhosis, chronic respiratory disease, chronic cardiovascular disease, chronic renal failure, immunosuppression due to disease, immunosuppression due to therapy; and *Paediatric cases aged <16 years* – prematurity, immunodeficiency, cystic fibrosis, congenital heart disease, neuromuscular disorder or chronic neurological impairment.

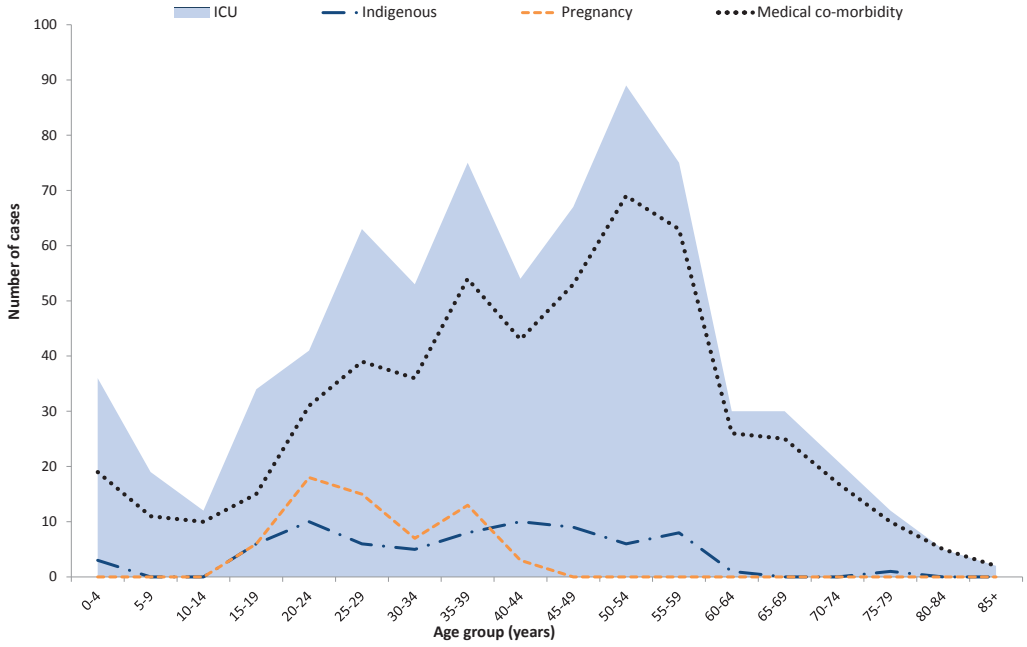
## Figure 34: Number of confirmed influenza A(H1N1)pdm09 cases concurrently admitted to an ICU and either mechanically ventilated or receiving extracorporeal membrane oxygenation, Australia, 2009*, by date




Source: Australia and New Zealand Intensive Care Society

*ANZICS data collection commenced 1 June 2009

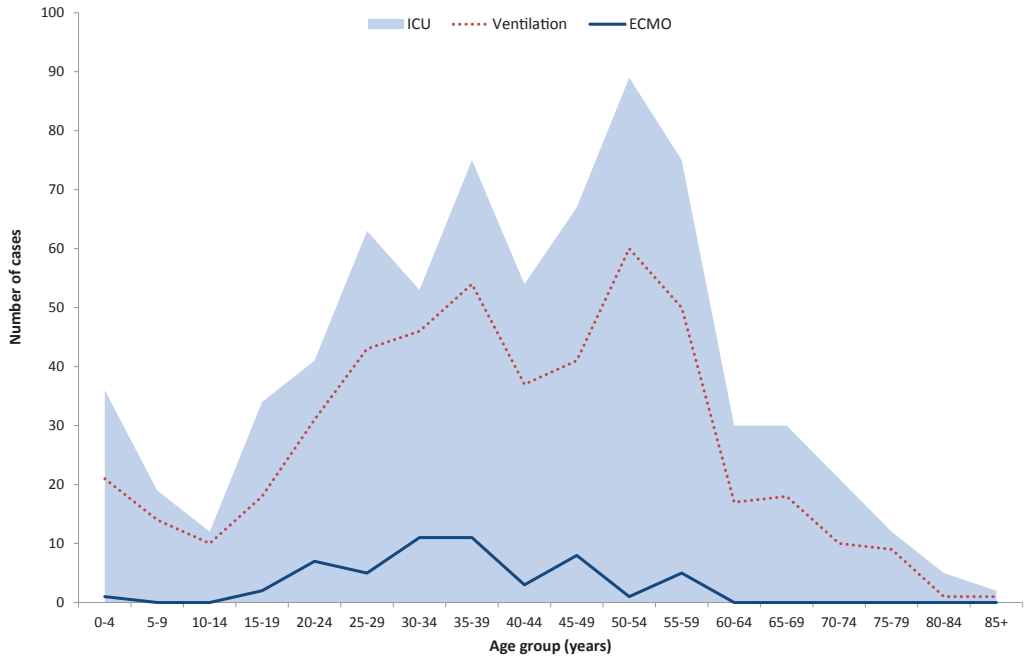
## Figure 35: Number of confirmed influenza A(H1N1)pdm09 cases admitted to an ICU, Australia, 2009, by age group and risk factor*




Source: Australia and New Zealand Intensive Care Society

*Medical co-morbidity includes APACHE III co-morbidities, chronic lung disease, chronic heart failure, diabetes mellitus and morbid obesity (BMI≥40kg/m$^2$)

## Figure 36: Number of confirmed influenza A(H1N1)pdm09 cases admitted to an ICU and mechanically ventilated and/or received extracorporeal membrane oxygenation, Australia, 2009, by age group




Source: Australia and New Zealand Intensive Care Society

ally increased with increasing age, with a peak additionally noted in the 20-24 years age group. Although pregnant women represent just over 1% of the general Australian population,[41, 56] of the 718 cases admitted to an ICU with influenza A(H1N1)pdm09, 8.6% (62/718) were pregnant (Table 9). The proportion of cases with a BMI of greater than or equal to 40kg/m$^2$ was 11% (79/718), which is much higher than the estimated 2.4% prevalence amongst Australian adults aged 18 years or over.[52] Data also indicated that Indigenous patients were also relatively overrepresented and accounted for 10.1% (73/718) of admissions to ICUs in Australia.

For some of the ICU admitted cases that presented with, or developed, severe acute respiratory distress syndrome, mechanical ventilation and/or extracorporeal membrane oxygenation (ECMO) treatment was required.[57] Approximately 67% (481/718) of ICU admissions required mechanical ventilation for a median of 8 days (IQR 3 to 17) (Table 9). The concurrent peak demand for mechanical ventilation was in the week ending 31 July 2009 with a median of 113 cases per day (range 99 to 119) (Figure 34). The median age of cases requiring mechanical ventilation was 40 years, and there were peaks in the 35-39 and 50-54 years age groups (Figure 36).

Of the 481 cases that underwent mechanical ventilation, 54 (11.0%) were subsequently treated with ECMO for a median duration of 8 days (IQR 4.25-13) (Table 9). The concurrent peak demand for ECMO occurred during the week ending 7 August 2009 with a median of 14 cases per day (range 12 to 18) (Figure 34). Cases requiring ECMO were often young adults (median age 35 years) (Figure 36), pregnant or postpartum women, obese, had severe respiratory failure before ECMO, and received prolonged mechanical ventilation and ECMO support.[57]

Around 81% (579/718) of ICU admitted cases were reported to have received antivirals either prior to or during their hospital admission. The majority of cases 82.2% (476/579) were reported to have received antivirals in hospital only; a further 17.4% (101/579) were reported as having received antivirals both prior to admission and in hospital, and the remainder of cases received antivirals prior to their hospital admission only (0.4%; 2/579). Whilst date of antiviral administration prior to hospitalisation data were not available, among cases who were admitted to an ICU and were reported as only receiving antivirals in hospital 96% (457/476) had antiviral admission data reported. The median time from onset of illness to the initiation of hospital based antiviral therapy for these cases was 5 days (IQR 3-8) and 24.9% (114/457) received antiviral therapy within 2 days of the onset of symptoms.

### Mortality

There were 188 deaths notified as being associated with confirmed pandemic influenza infection, representing 0.5% of all confirmed cases (188/37,554). Whilst there were several additional deaths reported during 2009, following further investigation, including coroner investigation outcomes, these cases were retrospectively considered to have died from other causes not associated with pandemic influenza infection.

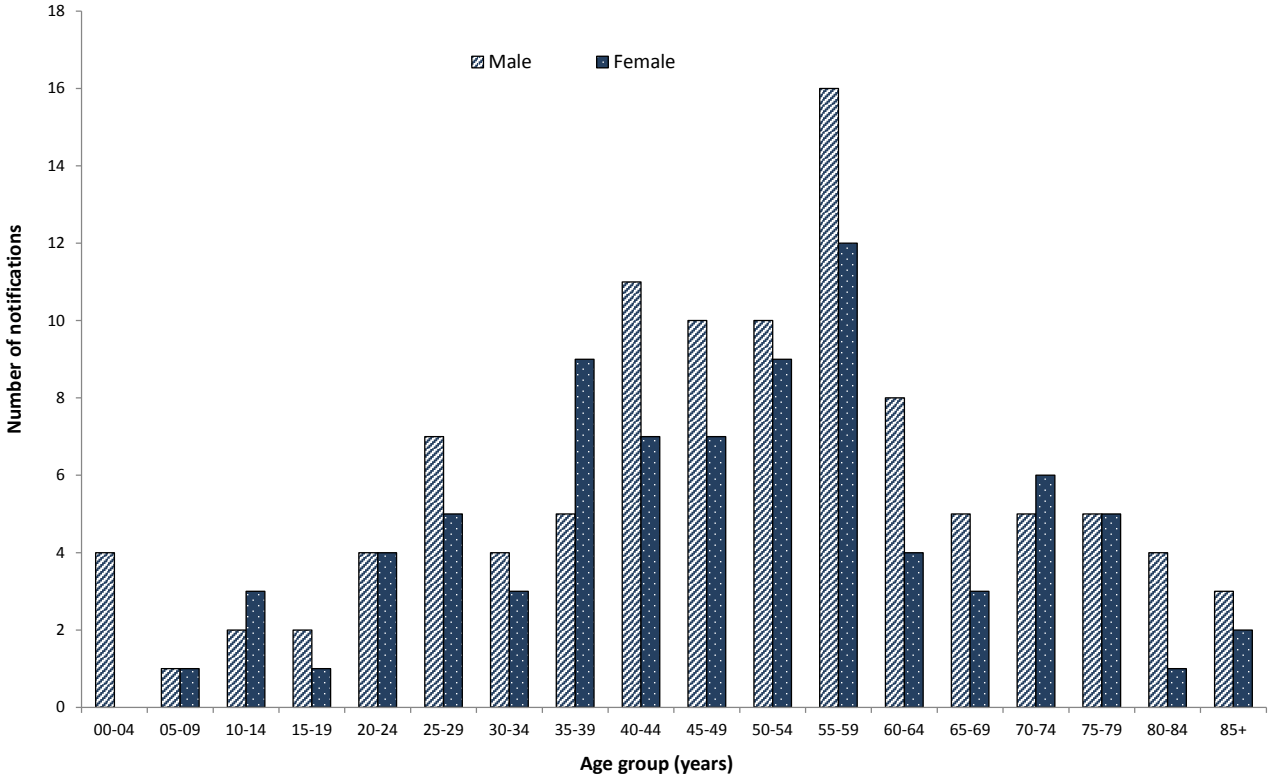
Of the cases who died, 82% (155/188) had been reported as being hospitalised, with the remainder reported as dying in another setting, such as palliative care services or at home.

Duration of illness was able to be estimated for 80.9% (152/188) of cases where onset of illness or specimen dates was pre-mortem. Of these cases the median time from onset of illness to death was 11 days (IQR 2-16 days).

#### Groups affected

The median age of cases who died was 50 years (IQR 37-62), which was considerably lower than the median age (83 years) among deaths with influenza recorded as the underlying cause of death on death certificate data for the period 2001-2006[58]. Forty four per cent of deaths were females. The age distribution of deaths peaked in the 55-59 years age group for both males and females, with females also experiencing an apparent sub-peak in the 35-39 years age group (Figure 37).

**Figure 37: Number of confirmed influenza A(H1N1)pdm09 associated mortality, Australia, 2009, by sex and age group**




Source: NetEpi, NNDSS and EpiLog

Valid underlying medical condition risk factor data were available for 173 (92.0%; 173/188) of the influenza A(H1N1)pdm09 deaths reported during 2009, with 86.1% (149/173) of these cases having at least one underlying medical condition reported (Table 7). Chronic respiratory conditions represented the most commonly reported underlying medical condition (47.1%; 64/136), followed by cardiac disease (29%; 36/124), morbid obesity (22.1%; 29/131), diabetes mellitus (%21.9; 28/128) and immunosuppression (20.0%; 25/125) (Figure 28).

## Viral characteristics

The influenza A(H1N1)pdm09 virus had six genes derived from triple-reassortant North American swine lineages and two genes, which encode the neuraminidase and matrix proteins, from the Eurasian swine virus lineages. [51, 59] This combination of gene segments had not previously been reported in swine or human influenza viruses. Although the influenza A(H1N1)pdm09 virus is antigenically distinct from other human and swine influenza A(H1N1) viruses, strains for this virus have remained antigenically homogeneous and closely related to the A/California/7/2009 strain that was selected for pandemic influenza vaccines worldwide.[51]

## Antiviral Treatment

Data on antiviral drug administration were available for 17.2% (6,522/37,754) of cases overall, with hospitalised cases representing half of these cases (48%; 3,146/6,522). Of the non-hospitalised cases, (46% (1,542/3,376) were prescribed antivirals and the median age of these cases was 24 years (IQR 14-37).

Antiviral treatment data were available for 62% (3,146/5,085) of hospitalised cases (Table 10). Of these cases, 71% (2,240/3,146) were recorded as having received antiviral therapy. The median age of these cases was 36 years (IQR 18-53); much higher than the non-hospitalised cases.

Among hospitalised cases for whom data on antiviral therapy timing were available (86.6%; 1,940/2,240), 55% (1,071/1,940) had received antiviral treatment within 2 days of their reported onset of symptoms, with the majority of cases receiving their antivirals on their date of hospital admission. The median time from onset of illness to the initiation of antiviral therapy was 2 days, with a range of 37 days prior to illness onset (likely to represent a prior prophylactic course) and 51 days post illness onset.

Of cases who were admitted to an ICU for whom antiviral therapy data were available (64%; 442/686), almost 90% (396/442) had received antiviral drugs (Table 10). Where timing of antiviral therapy data were available (88%; 350/396), antiviral therapy was initiated within 2 days of symptom onset for 41% (143/350) of cases, with antiviral therapy being initiated greater than 2 days of symptom onset for 57% (199/350).

The median duration of hospitalisation for cases where antiviral therapy was initiated within 2 days of symptom onset was 3 days (IQR 2-6), one day shorter in comparison to those who initiated antiviral treatment more than 2 days following symptom onset (4 days; IQR 2-8) (Table 10). However, in comparison to those who were reported as not receiving antiviral treatment (2 days; IQR 1-4), the median duration was longer by one day. An explanation for the difference in the expected effect on disease severity using hospital duration as a proxy and the apparent non-beneficial effect may be explained by disease severity at presentation as an indicator for antiviral initiation. For more severe cases, based on the median duration of hospitalisation for cases admitted to an ICU during their hospitalisation, there is an apparent reduction in the median duration of hospitalisation where antiviral treatment was initiated within 2 days of symptom onset (9 days; IQR 4-17), compared to those that did not receive antivirals (10 days (IQR 3- 27.5) or where treatment was initiated greater than 2 days following symptom onset (12 days; IQR 5-22).

## Seroprevalence

Pre-pandemic samples were available from the Cairns and Townsville donor collection centres in north Queensland collected in late April - early May 2009. Post-pandemic samples were prospectively collected from seven sites across five states from late October to early December 2009 following the winter wave of the pandemic in Australia.

McVernon *et al* (2011)[36] found that there was an increase in the influenza-seropositive propor-

**Table 10: Duration of hospitalisation for confirmed influenza A(H1N1)pdm09, 2009, by antiviral initiation and hospital ward type**

| Antiviral initiated | Antiviral timing | Hospitalised | | | ICU admission | | |
|---|---|---|---|---|---|---|---|
| | | Total | Median duration (days) | IQR | Total | Median duration (days) | IQR |
| Yes | Greater than 2 days | 830 | 4 | 2-8 | 199 | 12 | 5-22 |
| | 0-2 days | 1,071 | 3 | 2-6 | 143 | 9 | 4-17 |
| | Prior to symptom onset | 39 | 3.5 | 2-5 | 8 | 9 | 4.25-15.5 |
| | No timing information | 300 | 3 | 2-5 | 46 | 9 | 4-16.25 |
| No | N/a | 864 | 2 | 1-4 | 46 | 10 | 3-27.5 |
| Unknown | N/a | 1,981 | 3 | 2-6 | 244 | 11 | 4-22 |
| **Total** | | **5,085** | | | **686** | | |

Source: NetEpi and EpiLog

**Table 11: Notifications and rates of laboratory confirmed influenza A(H1N1)pdm09, Australia, 2009, by Indigenous status and morbidity**

| Morbidity | Indigenous | | Non-Indigenous* | | Rate ratio of Indigenous to non-Indigenous | Standardised morbidity or mortality ratio |
|---|---|---|---|---|---|---|
| | Cases (n) | Crude rate# | Cases (n) | Crude rate# | | |
| All cases | 3,966 | 616.7 | 33,788 | 160.5 | 3.8 | 3.5 |
| Admitted to hospital | 807 | 125.5 | 4,278 | 20.3 | 6.2 | 7.0 |
| Admitted to ICU | 99 | 15.4 | 587 | 2.8 | 5.5 | 7.3 |
| Died | 23 | 3.6 | 165 | 0.8 | 4.6 | 7.6 |

Source: NetEpi, EpiLog and NNDSS
* Includes cases reported as 'non-Indigenous', 'unknown' or no data reported.
# Crude rate per 100,000 population

tion amongst donors from 12%, likely to represent broadly cross-reactive antibody responses induced by prior exposure to like influenza A viruses; to 22%, representing an attack rate of 10%. Noting the limitations of the sample source, the serosurvey suggested that exposure to the novel pandemic virus during the 2009 winter outbreak was relatively uncommon amongst the healthy Australian adult population. However, the true attack rate may be higher if first exposure to the novel virus was poorly immunogenic, resulting in low and/or rapidly declining antibody responses.

These seroprevalence findings were consistent with trends observed in other serosurveys conducted across Australia,[60, 61] where rates of infection differed by age group with the highest attack rates observed among adolescents and young adults of up to 21%. Findings from other country serosurveys noted that prior to the start of the pandemic the proportion of individuals with pre-existing antibodies that cross-reacted with the pandemic virus increased with age; and that seropositivity rates after the virus had been circulating were highest in younger adults.[62]

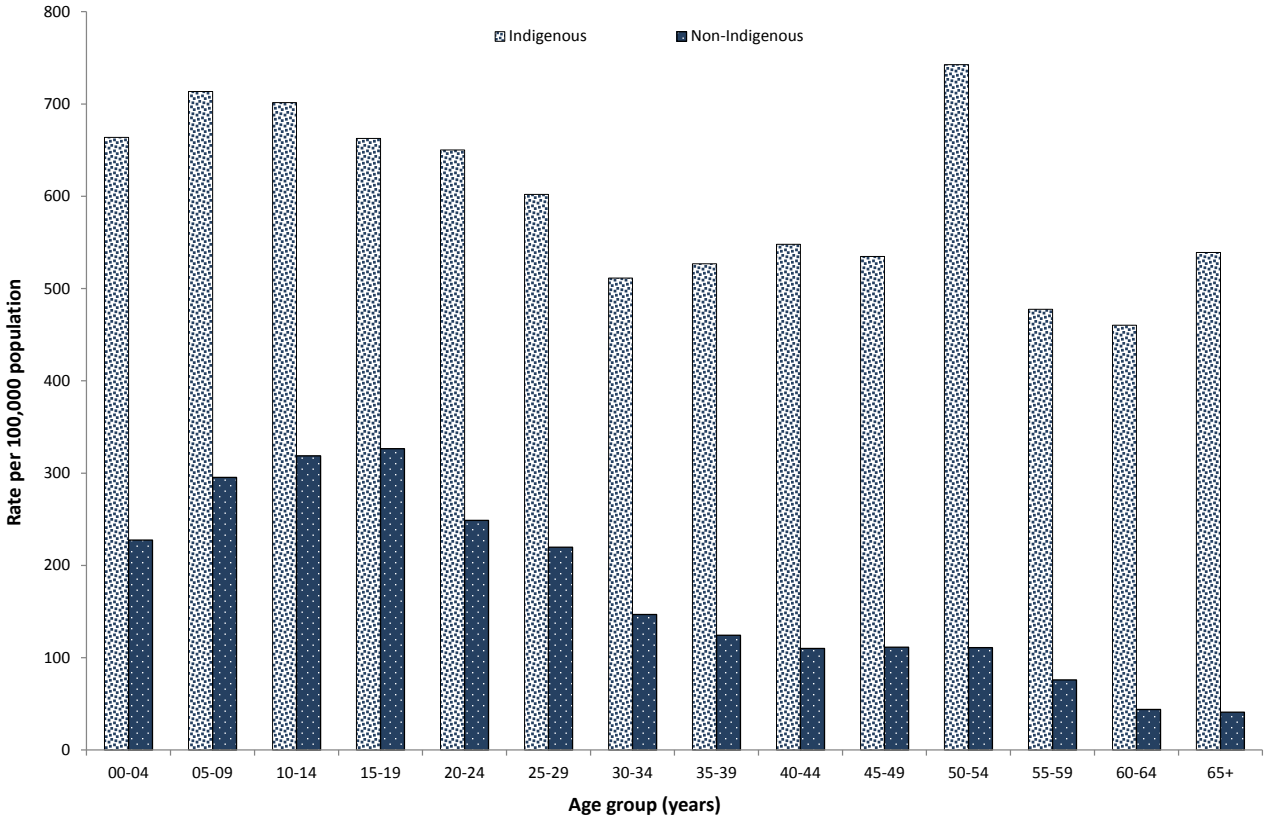
## Specific risk group analysis

### Indigenous

Indigenous status was reported for 61.8% (23,347/37,754) of cases throughout the pandemic, and 3,966 were identified as Indigenous Australians (Table 11). During the DELAY and CONTAIN phases, including cases reported as part of Victoria's MODIFIED SUSTAIN phase, Indigenous cases represented 1.6% (46/2,891) of cases, increasing to 11.2% (3,920/34,863) of cases during the PROTECT phase (Table 12). The increased proportion of Indigenous cases later in the pandemic, where case ascertainment focussed on those at risk of severe disease or those with severe disease, is likely to be due to both the known increased influenza susceptibility and substantially increased risk of severe disease among Indigenous persons[1], as well as increased geographic spread of the disease especially from urban into more rural and remote areas as the pandemic progressed.

Nationally, among Indigenous Australians, the age standardised notification rate of confirmed influenza A(H1N1)pdm09 was 595.9 per 100,000 population, which was over three times the rate experienced by the non-Indigenous Australian population (168.4 per 100,000 population). The highest crude rate of cases was reported in the Northern Territory (1,438.5 per 100,000 population), followed by Queensland (1,039.7 per 100,000 population) (Table 13). Differences in transmission patterns in the community setting, testing practices, prevalence of co-morbidities, population structure as well as ascertainment of Indigenous status, may partially explain some of the differences in crude notification rates between jurisdictions.[63]

**Figure 38: Notification rates of confirmed influenza A(H1N1)pdm09, Australia, 2009, by Indigenous status[#] and age group[*]**




Source: NetEpi, EpiLog and NNDSS
[#] Non-Indigenous includes cases whose Indigenous status was reported as 'unknown' (n=14265) or was missing (n=142).
[*]Excludes 48 cases where age was not able to be determined.

Compared to the non-Indigenous population, rates of influenza A(H1N1)pdm09 among the Indigenous population were relatively high across all age groups and remained quite high among older age groups (Figure 38). However, overall Indigenous cases were younger than non-Indigenous cases (median age of 18 years compared with 21 years), and the ratio of males to females was lower for Indigenous Australians (0.90:1) compared to non-Indigenous Australians (0.97:1).

## Hospitalisation

Of the 5,085 cases hospitalised with influenza A(H1N1)pdm09, Indigenous status was reported for 3,688 (72.5%) of these cases, and 807 (15.9%) were reported as Indigenous Australians. The age standardised ratio for admission to hospital was much higher in the Indigenous Australian population compared with the non-Indigenous population (Table 11).

Indigenous Australians hospitalised with confirmed influenza A(H1N1)pdm09 were slightly older than non-Indigenous Australians (median age of 32 years compared to 30 years). The highest rates of hospitalisation for Indigenous Australian cases were 271 per 100,000 population in the 50-54 years age group, and 58.2 per 100,000 for cases aged less than five years in the non-Indigenous population (Figure 39). The ratio of males to females admitted to hospital was 0.89:1 among Indigenous Australian cases, with the proportion of males being lower in comparison to the ratio observed in among non-Indigenous Australians (1:1).

Risk factor data were available for 706 (87.5%) Indigenous Australian cases who were hospital-

**Table 12: Notifications of confirmed influenza A(H1N1)pdm09, Australia, 2009, by Indigenous status and pandemic phase**

| | Delay and Contain | | | Protect | | | All pandemic | | |
|---|---|---|---|---|---|---|---|---|---|
| | *n* | % | Rate* | n | % | Rate* | n | % | Rate* |
| Indigenous status | | | | | | | | | |
| Indigenous | 46 | 1.6 | 7.2 | 3,920 | 11.2 | 609.6 | 3,966 | 10.5 | 616.7 |
| Non-Indigenous | 1,147 | 39.7 | 13.5 | 18234 | 52.3 | 147.0 | 19381 | 51.3 | 106.5 |
| Unknown/blank | 1,698 | 58.7 | - | 12709 | 36.5 | - | 14407 | 38.2 | - |
| Total | 2,891 | 100.0 | 13.3 | 34863 | 100.0 | 160.7 | 37754 | 100.0 | 174.0 |

Source: NetEpi, EpiLog and NNDSS
*Crude rate per 100,000 population[37, 38]

ised. Of these cases, 485 (68.7%) were recorded as having at least one pre-existing medical condition. A total of 43 patients were reported as pregnant (26.2% of Indigenous Australian female hospitalised cases aged 15-44 years with risk factor data). A third of cases (n=221) had chronic respiratory conditions; 22.0% (n=155) had diabetes mellitus; and 14.2% (n=100) had a chronic cardiac condition (Table 14).

The duration of hospitalisation among Indigenous Australians was comparable to that of non-Indigenous Australians (median 3 days).

### ICU admission

A total of 99 Indigenous Australians were admitted to an ICU with influenza A(H1N1)pdm09 in 2009. This represented 16.8% of all ICU admissions reported to NetEpi and Queensland's EpiLog system (n=587), noting that Victorian and ACT ICU admissions are not represented in these data. A slightly smaller proportion of ICU admissions among Indigenous Australians (10.2%) were identified in the ANZICS dataset (Table 9).

The highest rate of admission to ICU for Indigenous Australian cases were in the 55-59 years age group with 66.8 admissions per 100,000 population (Figure 40). For non-Indigenous cases rates of ICU admissions were highest in the 50-54 years age group (5.6 per 100,000 population). The age standardised rate for Indigenous Australians admitted to an ICU was 7.3 per 100,000 population. The median age for Indigenous Australian cases admitted to ICU was 41 years (IQR 25.5-51) and 44 years (IQR 28.5-55) for non-Indigenous cases.

Risk factor data were available for 94 (94.9%) of Indigenous Australian cases admitted to an ICU. Of these cases, almost 90% (n=84) were recorded as having at least one pre-existing medical condition. Forty-three per cent (n=41) had a chronic respiratory condition; 28% (n=26) diabetes mellitus and 26% (n=24) cardiac disease. A total of 12 patients were reported as pregnant, representing a third of Indigenous Australian female cases aged 15-44 years admitted to an ICU (Table 14).

The duration of hospitalisation for cases admitted to an ICU among Indigenous Australians was comparable to that of non-Indigenous Australians (median 10.5 and 11 days respectively).

### Mortality

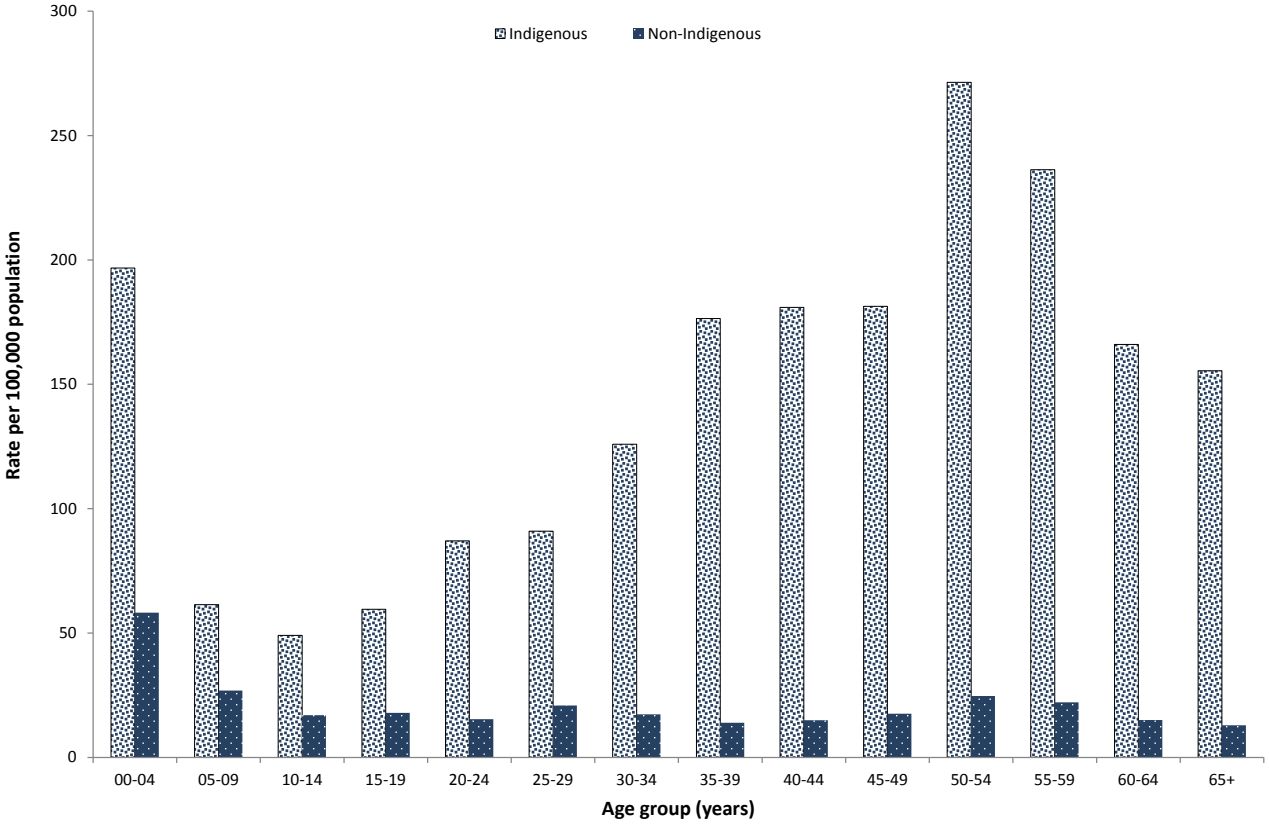
Of the 188 deaths reported to be associated with influenza A(H1N1)pdm09 in 2009, 23 (12.2%) were reported as Indigenous Australians. The age standardised mortality ratio for Indigenous Australians was 7.6 (Table 11).

**Table 13: Notifications and rates of confirmed influenza A(H1N1)pdm09, Australia, 2009, by Indigenous status and state or territory**

| State or territory | Indigenous | | Non-Indigenous* | |
|---|---|---|---|---|
| | n | Crude rate# | n | Crude rate# |
| ACT | 18 | 316.0 | 914 | 261.8 |
| NSW | 202 | 100.7 | 5,294 | 77.2 |
| NT | 974 | 1,438.5 | 514 | 324.7 |
| Qld | 1,871 | 1,039.7 | 10128 | 244.1 |
| SA | 325 | 910.7 | 8,881 | 564.5 |
| Tas | 10 | 43.1 | 953 | 198.1 |
| Vic | 9 | 20.2 | 3,086 | 57.9 |
| WA | 557 | 653.6 | 4,018 | 186.4 |
| Total | 3,966 | 616.7 | 33788 | 160.5 |

Source: NetEpi, EpiLog and NNDSS
* Includes cases reported as 'non-Indigenous', 'unknown' and 'blank'.
# Crude rate per 100,000 population

**Figure 39: Rates of laboratory confirmed influenza A(H1N1)pdm09 hospitalisations, Australia, 2009, by age group and Indigenous status**



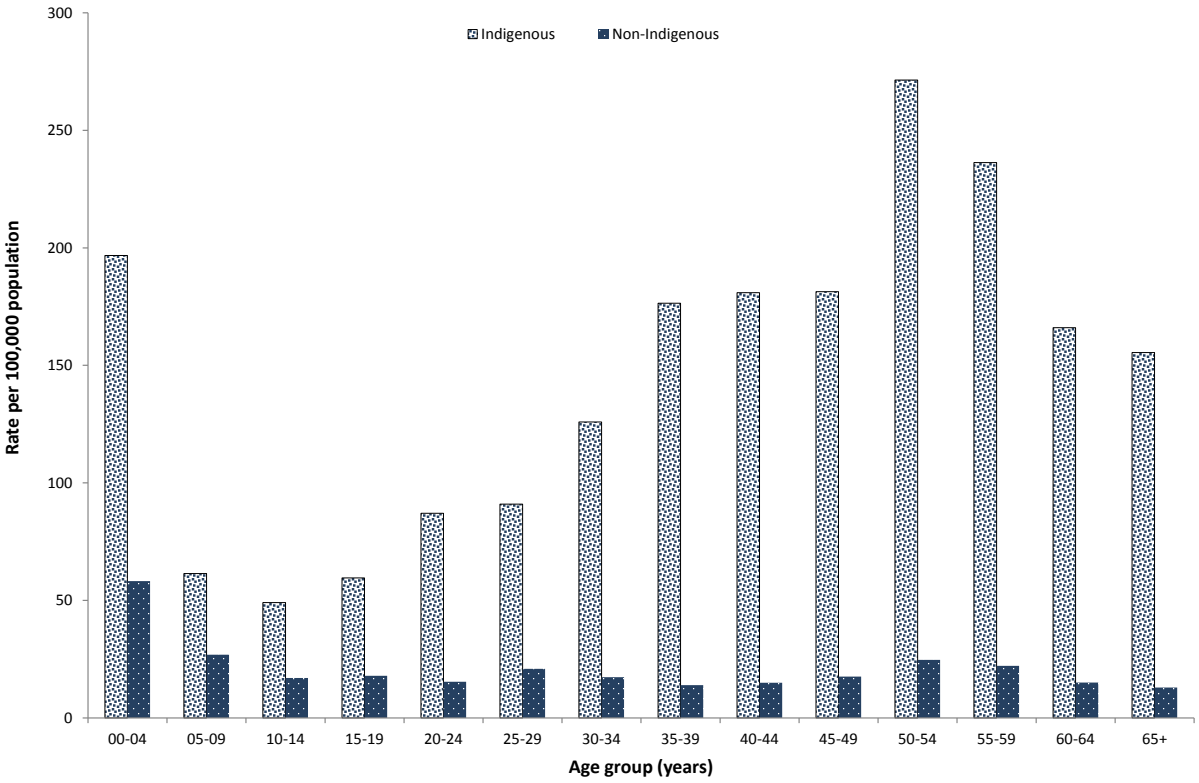

Source: NetEpi and EpiLog

**Table 14: Characteristics of notifications of laboratory confirmed influenza A(H1N1)pdm09 among Indigenous Australians, Australia, 2009, by hospitalisation and mortality status**

| | Hospitalisation | | ICU | | Deaths | |
|---|---|---|---|---|---|---|
| | n | (%) | n | (%) | n | (%) |
| Total | 807 | | 99 | | 23 | |
| Total cases with underlying medical conditions data | 706 | (87.5) | 94 | (94.9) | 20 | (87.0) |
| Age (years)‡ | | | | | | |
| 00-04 | 163 | (20.2) | 3 | (3.0) | 1 | (4.3) |
| 05-09 | 48 | (5.9) | 0 | (0.0) | 0 | (0.0) |
| 10-14 | 38 | (4.7) | 3 | (3.0) | 0 | (0.0) |
| 15-19 | 41 | (5.1) | 6 | (6.1) | 0 | (0.0) |
| 20-24 | 49 | (6.1) | 11 | (11.1) | 1 | (0.1) |
| 25-29 | 42 | (5.2) | 7 | (7.1) | 1 | (0.1) |
| 30-34 | 50 | (6.2) | 5 | (5.1) | 0 | (0.0) |
| 35-39 | 76 | (9.4) | 11 | (11.1) | 3 | (0.4) |
| 40-44 | 68 | (8.4) | 15 | (15.2) | 4 | (0.6) |
| 45-49 | 60 | (7.4) | 9 | (9.1) | 2 | (0.3) |
| 50-54 | 72 | (8.9) | 10 | (10.1) | 3 | (0.4) |
| 55-59 | 46 | (5.7) | 13 | (13.1) | 6 | (0.8) |
| 60-64 | 22 | (2.7) | 3 | (3.0) | 0 | (0.0) |
| 65+ | 32 | (4.0) | 3 | (3.0) | 2 | (0.3) |
| Underlying medical conditions*# | | | | | | |
| Chronic respiratory conditions | 221 | (31.3) | 41 | (43.6) | 10 | (50.0) |
| Cardiac disease | 100 | (14.2) | 24 | (25.5) | 8 | (40.0) |
| Immunosuppression | 16 | (2.3) | 3 | (3.2) | 4 | (20.0) |
| Haemoglobinopathies | 9 | (1.3) | 2 | (2.1) | 1 | (5.0) |
| Neurological conditions | 24 | (3.4) | 6 | (6.4) | 4 | (20.0) |
| Diabetes mellitus | 155 | (22.0) | 26 | (27.7) | 6 | (30.0) |
| Renal failure | 77 | (10.9) | 14 | (14.9) | 3 | (15.0) |
| Morbid obesity | 51 | (7.2) | 13 | (13.8) | 3 | (15.0) |
| Metabolic disorders | 21 | (3.0) | 5 | (5.3) | 2 | (10.0) |
| Pregnancy | 43 | (6.1) | 12 | (12.8) | 1 | (5.0) |
| *Pregnancy (females, aged 15-44)*† | 43 | (26.2) | 12 | (33.3) | 1 | (25.0) |
| Other | 111 | (15.7) | 23 | (24.5) | 5 | (25.0) |
| Number of medical conditions | | | | | | |
| None | 220 | (31.2) | 10 | (10.6) | 1 | (5.0) |
| 1 | 257 | (36.4) | 32 | (34.0) | 4 | (20.0) |
| 2 | 146 | (20.7) | 27 | (28.7) | 7 | (35.0) |
| 3 | 59 | (8.4) | 19 | (20.2) | 5 | (25.0) |
| 4 | 18 | (2.5) | 4 | (4.3) | 1 | (5.0) |
| 5 | 6 | (0.8) | 2 | (2.1) | 2 | (10.0) |

Source: NetEpi and EpiLog
‡Total cases used as the denominator for determining the proportion.
* More than one pre-existing medical condition could be reported for a case.
# Total cases with risk factor data used as the denominator for determining the proportion.
† The denominator used was females aged 15-44 years with risk factor data reported, hospitalised cases n=164, ICU admissions n=36 and deaths n=4.

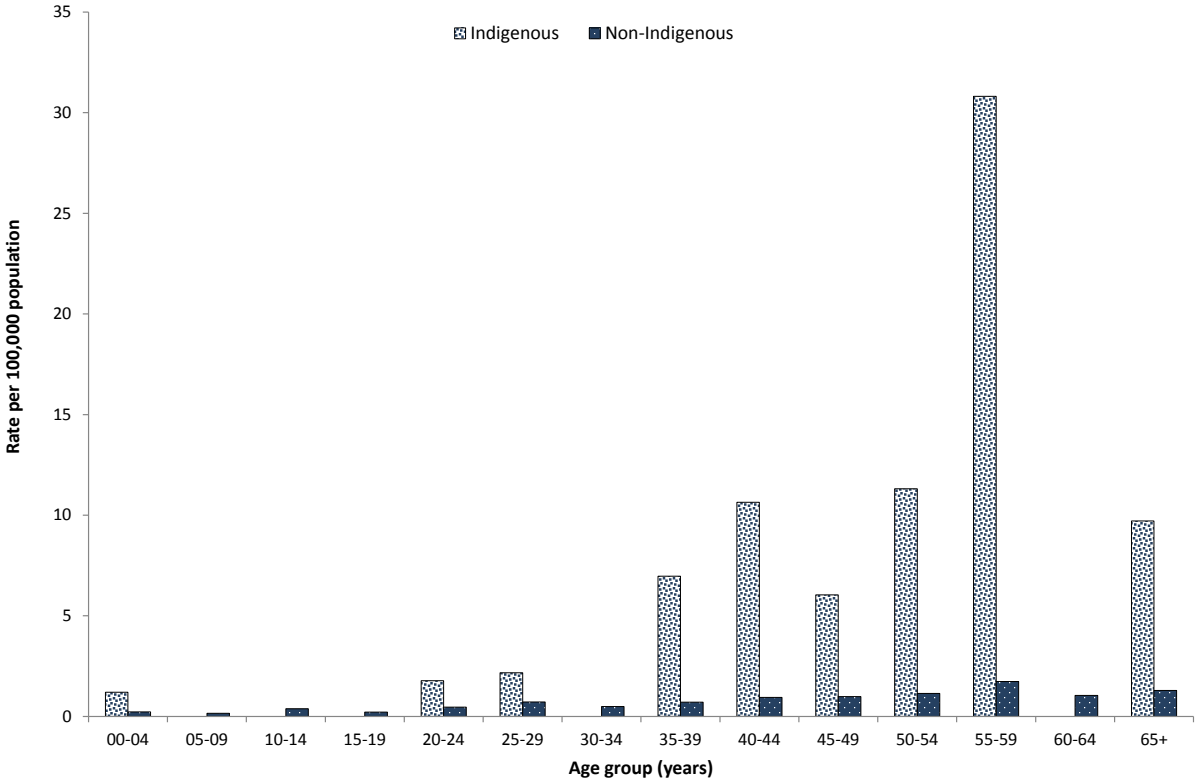
**Figure 40: Rates of laboratory confirmed influenza A(H1N1)pdm09 among Indigenous Australians admitted to an ICU*, Australia, 2009, by age group and Indigenous status**




Source: NetEpi and EpiLog

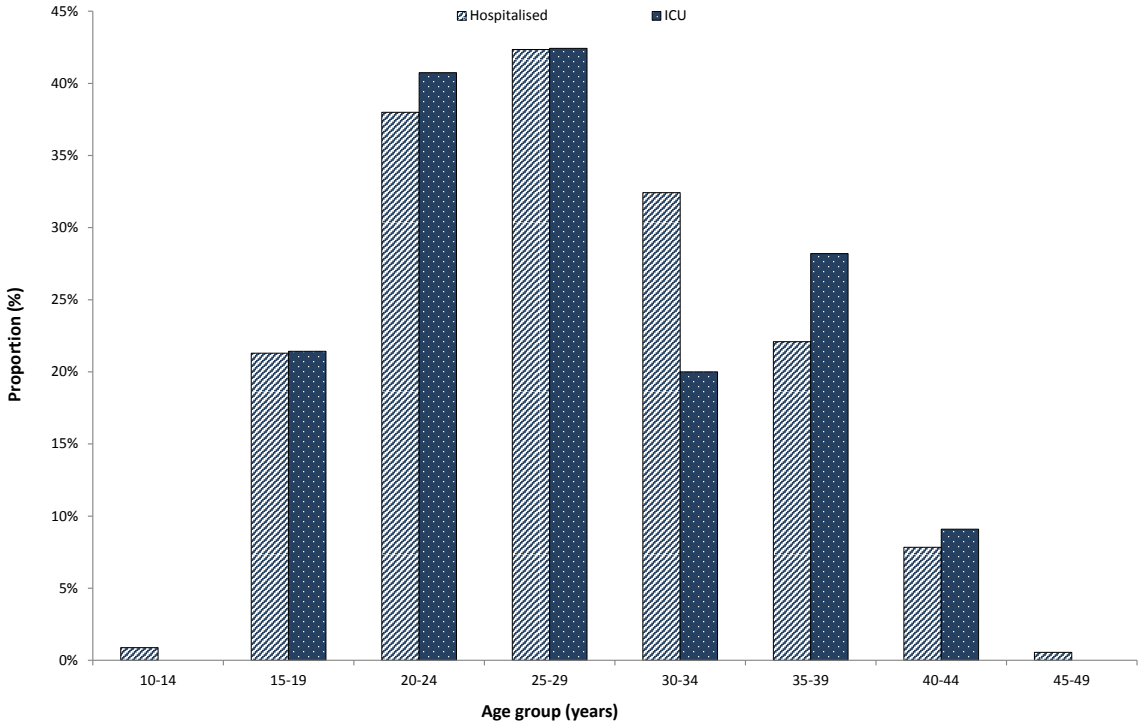
*Victoria and ACT ICU admissions are not represented in these data

**Figure 41: Rates of laboratory confirmed influenza A(H1N1)pdm09 associated mortality, Australia, 2009, by age group and Indigenous status**



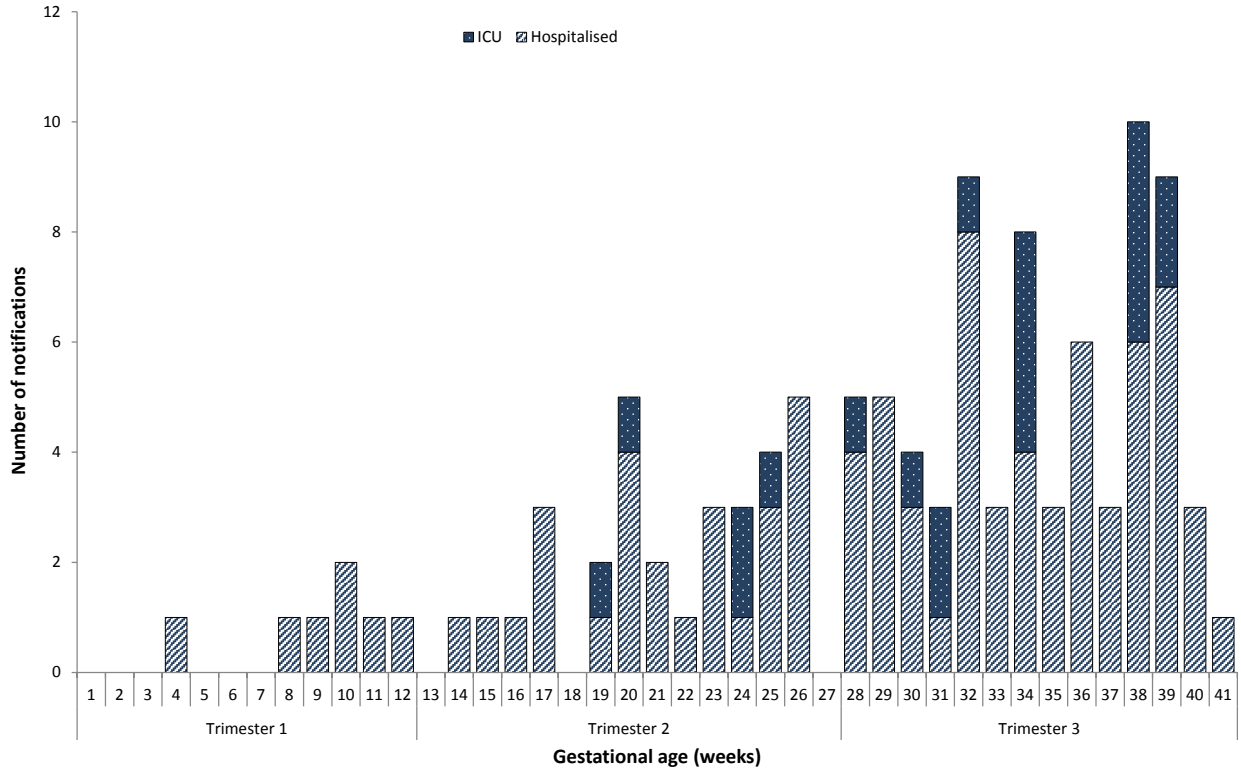

Source: NetEpi, EpiLog and NNDSS

**Figure 42: Proportion of laboratory confirmed influenza A(H1N1)pdm09 notifications reported as pregnant* among females, Australia, 2009, by 5-year age group and ward type**




Source: NetEpi and EpiLog
* Excludes 2 pregnant cases whose age was not able to be determined.

**Figure 43: Notifications of laboratory confirmed influenza A(H1N1)pdm09 in pregnant women* admitted to hospital, Australia, 2009, by weeks of gestation and ward type**




Source: NetEpi and EpiLog
*Excludes 196 cases where gestational age was not reported.

**Table 15: Characteristics of notifications of laboratory confirmed influenza A(H1N1)pdm09 among pregnant women admitted to hospital, Australia, 2009**

| | Hospitalised n(%) | | ICU n(%) | |
|---|---|---|---|---|
| Total | 306 | | 47 | |
| **Age (years)** | | | | |
| 10-14 | 1 | (0.3) | 0 | (0.0) |
| 15-19 | 33 | (10.8) | 3 | (1.0) |
| 20-24 | 68 | (22.2) | 11 | (3.6) |
| 25-29 | 94 | (30.7) | 14 | (4.6) |
| 30-34 | 59 | (19.3) | 5 | (1.6) |
| 35-39 | 36 | (11.8) | 11 | (3.6) |
| 40-44 | 12 | (3.9) | 3 | (1.0) |
| 45-49 | 1 | (0.3) | 0 | (0.0) |
| Unknown | 2 | (0.7) | 0 | (0.0) |
| **Indigenous status** | | | | |
| Indigenous | 43 | (14.1) | 12 | (25.5) |
| Non-Indigenous | 185 | (60.5) | 29 | (61.7) |
| Unknown | 78 | (25.5) | 6 | (12.8) |
| **Underlying medical conditions*** | | | | |
| Chronic respiratory conditions | 40 | (13.1) | 0 | (0.0) |
| Cardiac disease | 7 | (2.3) | 4 | (8.5) |
| Immunosuppression | 1 | (0.3) | 1 | (2.1) |
| Haemoglobinopathies | 5 | (1.6) | 0 | (0.0) |
| Neurological conditions | 1 | (0.3) | 0 | (0.0) |
| Diabetes mellitus | 11 | (3.6) | 2 | (4.3) |
| Renal failure | 2 | (0.7) | 0 | (0.0) |
| Morbid obesity | 7 | (2.3) | 3 | (6.4) |
| Metabolic disorders | 4 | (1.3) | 0 | (0.0) |
| Other | 19 | (6.2) | 6 | (12.8) |
| **Number of underlying medical conditions*** | | | | |
| 0 | 222 | (72.5) | 30 | (63.8) |
| 1 | 74 | (24.2) | 14 | (29.8) |
| 2 | 8 | (2.6) | 2 | (4.3) |
| 3 | 1 | (0.3) | 0 | (0.0) |
| 4 | 1 | (0.3) | 1 | (2.1) |
| **Trimester of admission** | | | | |
| 1st | 7 | (2.3) | 0 | (0.0) |
| 2nd | 31 | (10.1) | 5 | (10.6) |
| 3rd | 72 | (23.5) | 15 | (31.9) |
| Unknown | 196 | (64.1) | 27 | (57.4) |

Source: NetEpi and EpiLog

* More than one pre-existing medical condition could be reported for a case. Pre-existing medical conditions are in addition to pregnancy

The pattern of age-specific mortality rates was similar between the Indigenous and non-Indigenous Australians; however the age-specific rates were substantially higher amongst Indigenous Australians (Figure 41). The median age of Indigenous Australians whose death was associated with influenza A(H1N1)pdm09 was 48 years (IQR 25.5-51) and 54 years (38.5-66) among non-Indigenous Australians.

Risk factor data were available for 20 of the 23 (87%) influenza A(H1N1)pdm09 associated deaths in Indigenous Australians. Of these cases, all except one case was reported as having at least one pre-existing medical condition. Although based on small numbers, half (n=10) were recorded as having a chronic respiratory condition and 40% cardiac disease (Table 14).

## Pregnant women

In Australia pregnant women represent approximately 1% of the total population and 6.5% among women aged 15-44 years. The median age of all mothers in 2009 was 30.6 years. [41,56] In 2009, 619 cases were reported as being pregnant, representing 1.6% (619/37,754) of all confirmed cases; however based on completeness of this risk factor field, 9.2% of cases were reported as being pregnant at the time of their infection (619/6,746), with a median age of 27 years. Among females aged 15-44 years, approximately 5.9% (614/10,367) of confirmed cases, with or without risk factor data, and 27.0% (614/2,271) of cases with risk factor data were pregnant at the time of their infection.

### Hospitalisation

In 2009, a total of 306 hospitalised cases were reported as being pregnant, representing just over 6% of all hospitalised cases (306/5,085); with a median age of 25 years. Of hospitalised cases among females aged between 15-44 years, those reported as pregnant comprised 28.7% (302/1,054) of these cases. This suggests that compared to the expected prevalence of pregnancy among females aged 15-44 years in the Australian population (6.5%)[41], pregnant women were about four times more likely to be admitted to hospital with influenza A(H1N1)pdm09.

Information regarding gestational age was available for a third (110/306) of the cases. Approximately 6% (7/110) were in their first trimester; 28% (31/110) in their second trimester; and 66% (72/110) in their third trimester (Table 15, Figure 43).

As an indication of severity, pregnant women were disproportionately represented among hospitalisations compared to other females of child bearing age (Figure 42). However, the median duration of hospitalisation for pregnant women was 3 days (IQR 1-5), which was comparable to the median duration among females aged 15-44 years who were not pregnant (3 days; IQR 2-6).

Over a quarter (27.5%; 84/306) of the cases reported had at least one underlying medical condition, in addition to pregnancy. The most common reported underlying medical condition among this group were chronic respiratory conditions (13.1%; 40/306). Just over 14% (43/306) of pregnant cases hospitalised were identified as Indigenous Australians (Table 15), representing almost 1.5 times the expected prevalence of pregnancy among Indigenous females aged 15-44 years in the Australian population (9.0%)[41].

### ICU Admission

A total of 47 pregnant women were admitted to an ICU, with a median age of 22 years. Of the 20 cases (43%) where gestational age was known, 75% (15/20) were in their third trimester, with the remainder in their second trimester (Table 15, Figure 43).

As an indication of severity, pregnant women were disproportionately represented among ICU admissions compared to other females of child bearing age (Figure 42). However, the median duration of hospitalisation for cases who were pregnant and admitted to an ICU was comparable to the median duration observed

among females aged 15-44 years who were not pregnant, 9 days (IQR 3-18) and 10 days (IQR 4-18) respectively.

### Mortality

Of the 188 influenza A(H1N1)pdm09 associated deaths reported, three were in pregnant women. All three cases were reported to have had additional risk factors predisposing to severe disease.

## Discussion

Influenza derives its public health significance from the rate with which the virus evolves, it's widespread morbidity and the seriousness of complications.[2] The overall number of notifications during the Australian 2009 influenza season was the highest since national reporting to the NNDSS began in 2001, and substantially higher than years immediately prior. Notifications started to increase in May, following the emergence of a novel influenza A virus overseas, peaking towards the end of July and returning to inter-seasonal levels by mid-October. The timing and size of influenza activity increases and peaks varied across states and territories in 2009 and notification rates tended to be highest overall throughout the northern and central areas of Australia. Of the total influenza notifications in 2009 (n=59,026), nearly all were influenza type A, with the majority of these associated with the newly emerged pandemic virus. Although a third of influenza A cases were unsubtyped, a high proportion of these are likely to have been the pandemic virus.

The first case of confirmed influenza A(H1N1) pdm09 infection in Australia was notified on 7 May 2009 in a traveller and by mid-June 2009, community-wide transmission of the virus was occurring across most jurisdictions. By the end of 2009, there were 37,755 laboratory confirmed cases, including 5,085 hospitalisations and 188 deaths notified.

Throughout Australia, cases of influenza A(H1N1)pdm09 were not distributed homogenously, especially during the early phases of DELAY and CONTAIN and there was substantial variation in both the apparent incidence and peak activity timing among states and territories. The true incidence of infection is difficult to know and was most likely considerably higher than reported as not everyone who was infected would have been tested, both due to the apparent mild nature of infection for most and the targeting of testing especially from 22 June 2009 onwards as part of the 'PROTECT' pandemic phase.

The number of people receiving care in hospital or admitted to an ICU peaked in late July. The proportion of pandemic influenza cases during this peak represented 1.2% of available private and public acute hospital beds and around 10% of available ICU beds. Although, as expected the median age of cases tended to increase by severity measurement indicators, as defined through the comparison of cases who were non-hospitalised, hospitalised, ICU admitted and died; compared to seasonal influenza, these median ages tended to be younger. In comparison to previous seasonal influenza, hospitalisation rates associated with pandemic influenza in 2009 were consistently highest for children aged less than 5 years, however their duration of hospitalisation tended to be shorter in comparison to older children and adults, suggesting a higher propensity to admit children presenting with ILI or confirmed influenza compared to older populations where a higher severity threshold may be required.

Although laboratory testing of people presenting with influenza-like illness to primary care varied throughout the phases of the pandemic, it is clear that the pandemic had a substantial impact on three key risk groups: Indigenous Australians, pregnant women and people with co-morbidities, especially people with chronic respiratory conditions. Additionally, there was a noticeable shift in the age distribution of cases with mostly older children and young adults being affected, as opposed to young children and the elderly.

Following the winter pandemic period there was ongoing summer activity of influenza A(H1N1) pdm09 in late 2009. Since 2009, the pandemic virus has continued to circulate, replacing the

previously circulating seasonal A(H1N1) strain. In August 2010, the WHO announced the end of the pandemic as the virus had adopted a seasonal pattern of circulation in both the northern and southern hemispheres. Today the virus continues to circulate on a seasonal basis.

The objective of Australia's pandemic activities throughout 2009 was to essentially flatten the epidemic curve so as to manage the impact and burden of the disease on the community and also the strain on public health resources. Influenza surveillance in Australia relied on a myriad of surveillance systems and data sources, with varying degrees of representativeness and ability to measure incidence, severity and impact. As it is impossible to identify and count every influenza infection, there was an overarching need to rapidly ensure an understanding of these factors to inform public health actions and balance the level of detail required with resource and logistical constraints.

The collection and reporting of enhanced information, although highly beneficial for informing decision making, is not traditionally undertaken during seasonal influenza and over time became difficult as the case numbers increased. This had a significant impact on the completeness and interpretability of the data nationally. Maintaining a national instance of these enhanced data through NetEpi and alternative systems required enormous effort on the part of jurisdictional health departments, with some jurisdictions double handling data in order to meet local and national requirements and many reverting to providing the core dataset through NNDSS only.

Data on hospitalisations were very beneficial in assessing severity; however these data were identified as being extremely difficult to access or collect effectively within the time frame needed for surveillance. Many jurisdictions relied on the resource intensive manual follow-up and reporting of these data. Later in the pandemic a number of national systems were implemented to capture these data, with some jurisdictions able to develop more automated extracts from hospital surveillance systems through relatively real-time data linkage activities.

As outlined in Appendix 4, there were a number of different sources of enhanced data provided throughout the pandemic, with varying degrees of completeness and representation by pandemic phase, jurisdictions and health care setting. This meant that the combining, cleaning and analysis of data on notified cases from 2009 was extremely complex.

The ability to accurately interpret surveillance data continues to be highly dependent on understanding its representativeness, stability and comparability, both between systems and over time. Many influenza surveillance systems are affected by degrees of underlying awareness to investigate an ILI presentation for influenza, which vary by jurisdiction, health care setting and over time. Following the 2009 pandemic, many of Australia's influenza surveillance systems have been reporting relatively higher levels of influenza activity in comparison to the pre-pandemic period; most likely associated with an apparent increased awareness and investigation propensity. In trying to understand these potential artefacts, there is a need to further enhance surveillance systems to enable the capture of denominator data across many of these systems.

Findings from the *Review of Australia's Health Sector Response to Pandemic (H1N1) 2009*[9] have informed the completion of the surveillance plan as part of the AHMPPI[64] for the collection, analysis and reporting of data at the national level, especially with regard to defining the level of detail needed to inform decision making appropriate for the changing phases of the pandemic. The majority of surveillance activities during a pandemic aim to be consistent with seasonal activities. Whilst an enhanced surveillance component has been identified, it is targeted towards enabling the initial understanding of the early clinical, epidemiological and virological parameters of a pandemic virus; followed by monitoring for change through a limited ongoing enhanced data collection. Additionally, work continues to be progressed

with regard to data management efficiency and scaling between national and jurisdictional systems.

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## Appendices

### Appendix 1: Summary of key surveillance related activities during the pandemic[10, 11, 65, 66]

| Phase | Date commenced | Activity |
|---|---|---|
| DELAY | 28 April 2009 | Testing of all patients with acute febrile respiratory illness who had been to a country with sustained community transmission or close contact with a confirmed or suspected case within the previous 7 days.<br>Suspected and confirmed cases treated with antivirals and isolated. If a suspected case tested negative these measures were ceased.<br>All contacts of suspected cases traced and if meeting the national contact definition were provided with antiviral prophylaxis and quarantined.<br>Border measures including positive pratique, thermal scanning and health declaration cards were implemented. |
| CONTAIN | 22 May 2009 | Testing of all suspected cases with acute febrile respiratory illness.<br>Suspected and confirmed cases treated with antivirals and isolated. If a suspected case tested negative these measures were ceased.<br>All contacts of suspected cases traced and if meeting the national contact definition were provided with antiviral prophylaxis and requested to remain in quarantine. |
| MODIFIED SUSTAIN (Victoria only)[10, 67] | 3 June 2009 | Testing recommended to those with moderate to severe disease or those with symptoms in vulnerable populations.<br>Antiviral treatment provided to people with acute febrile respiratory illness and immediate household contacts.<br>Confirmed cases requested to isolate themselves for 3 days following commencement of antiviral treatment. No quarantine required for household contacts.<br>Contact tracing in high risk settings intensified to protect those at greater risk of severe complications.<br>Victoria's sentinel general practitioner ILI surveillance program enhanced, including increased sampling to monitor the distribution of the virus and changes in the dominant circulating influenza strain. |
| PROTECT | 17 June 2009 | Testing focused on those with moderate to severe disease or those with symptoms from vulnerable settings.<br>Sentinel testing also continued at hospital and community level for surveillance purposes and to monitor virus behaviour.<br>Clinical cases offered antiviral treatment through consultation with healthcare professionals with an emphasis on treatment for persons in higher risk groups<br>Contacts of cases not offered prophylaxis.<br>Specific border surveillance for influenza activities ceased. |

## Appendix 2: Pandemic Influenza Case Definition, Australia, 2009, by version*

| Case classification | Version 3.0A, 1 May 2009[68] | Version 4, 15 May 2009[69] | Version 5, 23 May 2009[13] | Version 6C, 3 June 2009[70] |
|---|---|---|---|---|
| Suspected | A suspected case of human swine influenza A (H1N1) virus infection is defined as:<br>a person with acute febrile respiratory illness† with onset:<br>within 7 days of close contact with a person who is a confirmed case of human swine influenza A (H1N1) virus infection or a suspected case with an influenza A positive test OR<br>within 7 days of travel to Mexico, USA or Canada (countries to be updated where evidence of local transmission).<br>a person who meets the above criteria AND who is positive for influenza A (but not for influenza A H3 sub-type) by RT-PCR, OR by an influenza rapid test, OR by an influenza immunofluorescence assay (IFA). | A suspected case of H1N1 Influenza 09 (human swine influenza) virus infection is defined as a person with acute febrile respiratory illness‡ with onset:<br>within 7 days of close contact with a person who is a confirmed case of H1N1 Influenza 09 (human swine influenza) virus infection or a suspected case with an influenza A positive test result, OR<br>within 7 days of travel to Mexico, USA or Canada (countries to be updated where evidence of local transmission). | A suspected case of H1N1 Influenza 09 (human swine influenza) virus infection is defined as a person with acute febrile respiratory illness§ with onset:<br>within 7 days of close contact with a person who is a confirmed case of H1N1 Influenza 09 (human swine influenza) virus infection or a suspected case with an influenza A positive test result, OR<br>within 7 days of travel to Mexico, USA, Canada, Japan or Panama (countries to be updated where evidence of local transmission). | |
| Suspected – with influenza A positive result | | A suspected case with an influenza A positive result is defined as a person who meets the suspected case definition AND who is positive for influenza A by:<br>PCR (Matrix or other conserved region), OR<br>an influenza rapid antigen, OR<br>other antigen test e.g. immunofluorescence assay (IFA]).<br>A suspected case with an influenza A positive test result is excluded where the sample tests:<br>positive for human influenza A H1 and negative for H1N1Influenza 09 (human swine influenza) OR<br>positive for human influenza A H3 and negative for H1N1Influenza 09 (human swine influenza) | A suspected case with an influenza A positive result is defined as a person who meets the suspected case definition AND who is positive for influenza A by:<br>PCR (Matrix or other conserved region), OR<br>an influenza rapid antigen, OR<br>other antigen test e.g. immunofluorescence assay (IFA]).<br>A suspected case with an influenza A positive test result is excluded where the sample tests:<br>positive for human influenza A H1 and negative for H1N1Influenza 09 (human swine influenza) OR<br>positive for human influenza A H3 and negative for H1N1Influenza 09 (human swine influenza) | |
| Probable | | | A probable case is a person who has a strong epidemiological link to a confirmed case during that case's infectious period, and who:<br>has an acute respiratory illness§, with or without fever, for which no other cause is identified, but<br>tests negative on human swine influenza test OR has no appropriate sample collected for testing. | A probable case is a person who has a household or intimate epidemiological link to a confirmed case during that case's infectious period, and who has an acute respiratory illness (defined as recent onset of at least one of the following symptoms: rhinorrhoea, nasal congestion, sore throat or cough, with or without fever) for which no other cause is identified. |
| Confirmed | A confirmed case of human swine influenza A (H1N1) virus is defined as a person with an acute febrile respiratory illness† with laboratory confirmed human swine influenza A (H1N1) virus infection by one or more of the following tests:<br>viral sequencing<br>real-time RT-PCR<br>viral culture | A confirmed case of H1N1 Influenza 09 (human swine influenza) virus is defined as a person with an acute febrile respiratory illness‡ with laboratory confirmed H1N1 Influenza 09 (human swine influenza) virus infection by one or more of the following tests:<br>viral sequencing<br>human swine influenza (H1N1) specific-PCR<br>isolation of human swine influenza A (H1N1) virus | A confirmed case of H1N1 Influenza 09 (human swine influenza) virus is defined as a person with an acute respiratory illness§ with laboratory confirmed H1N1 Influenza 09 (human swine influenza) virus infection by one or more of the following tests:<br>viral sequencing<br>human swine influenza (H1N1) specific-PCR<br>isolation of human swine influenza A (H1N1) virus. | A confirmed case of H1N1 Influenza 09 (human swine influenza) virus is defined as a person with laboratory confirmed H1N1 Influenza 09 (human swine influenza) virus infection by one or more of the following tests:<br>viral sequencing<br>human swine influenza (H1N1) specific-PCR<br>isolation of human swine influenza A (H1N1) virus |

* Start date for case collection was based on an onset date of 15 April 2009.
† An acute febrile respiratory disease: is defined as a measured temperature of 38$^0$C or greater OR a good history of fever, AND recent onset of at least one of the following symptoms: rhinorrhoea, nasal congestion, sore throat or cough.
‡ An acute febrile respiratory disease is defined as a measured temperature of 38$^0$C or greater OR a good history of fever, AND recent onset of at least one of the following symptoms: rhinorrhoea, nasal congestion, sore throat or cough.
§ For cases not epidemiologically linked to a confirmed case an acute febrile respiratory disease is defined as a measured temperature of 38oC or greater OR a good history of fever, AND recent onset of at least one of the following symptoms: rhinorrhoea, nasal congestion, sore throat or cough.
- Persons who are epidemiologically linked to a potentially infectious confirmed case do not require a measured or well described fever to warrant investigation, but should have symptoms consistent with an acute respiratory illness (see Probable case).

## Appendix 3: Enhancing case ascertainment, testing protocols, Australia, 2009

| Source | Pandemic phase commenced | Testing protocol |
|---|---|---|
| Draft AHMPPI Surveillance Annex* | ALERT/DELAY | All suspected cases |
| CDNA case definition, version 5, 23 May 2009 | CONTAIN | To enhance case ascertainment in the early phases of the CONTAIN phase, CDNA have agreed that for the present time anyone with an acute febrile respiratory disease , regardless of travel history, should be considered for swabs and testing for influenza, within routine diagnostic procedures. Any influenza A positive specimen should be sub-typed and tested for H1N1 Influenza 09 (human swine influenza), and classified according to results. |
| CDNA case definition, version 6C, 3 June 2009 | CONTAIN (late)<br>VIC SUSTAIN | **Australian areas without community transmission:** Clinicians should prioritise taking nose and throat swabs for influenza testing from people who present with an acute respiratory illness (history of fever and either cough, sore throat, runny or blocked nose) and who:<br>have travelled to an area with community transmission (anywhere overseas or to an Australian area of high prevalence) in the previous 7 days, OR<br>are at risk of severe complications following human swine flu infection (pregnant women, people with diabetes or other chronic underlying illnesses, morbidly obese).<br>Any Influenza virus A positive specimen should be subtyped and tested for H1N1<br>Influenza 09 (human swine influenza).<br>**Australian areas with community transmission:** Clinicians should prioritise taking nose and throat swabs for influenza testing from people who present with an acute respiratory illness (a history of fever and either cough, sore throat, runny or blocked nose) and who:<br>are at risk of severe complications following human swine flu infection (pregnant women, people with diabetes or other chronic underlying illnesses, morbidly obese).<br>Once the first case in a cluster tests positive for H1N1 Influenza 09 (human swine influenza) the remaining members of the cluster do not need to be tested routinely. |

* A draft surveillance annex to the AHMPPI was being considered by the Scientific Influenza Advisory Group (SIAG) and the Australian Health Protection Committee (AHPC) Inter-jurisdictional Pandemic Planners Working Group (IPPWG) when the pandemic emerged.

## Appendix 4: Sources of notification surveillance data for laboratory confirmed cases of influenza, by State and Territory and pandemic phase, 2009

| State | Pre-Pandemic | Pandemic Phase | | | | Source for post-pandemic analysis |
|---|---|---|---|---|---|---|
| | | DELAY | CONTAIN | **MODIFIED SUSTAIN** ***(Victoria only)*** | PROTECT | |
| Seasonal influenza notifications | | | | | | |
| Demographic data | | | | | | |
| ACT<br>NT<br>Qld<br>SA<br>Tas<br>WA | NNDSS | NNDSS | NNDSS | n/a | NNDSS | NNDSS |
| Vic | NNDSS | NNDSS | NNDSS | NNDSS | NNDSS | NNDSS |
| NSW | NNDSS | NetEpi | NetEpi | n/a | NetEpi | NetEpi NNDSS |
| Pandemic influenza confirmed case notifications | | | | | | |
| All cases - Demographic data | | | | | | |
| ACT<br>NSW[#]<br>SA<br>Tas<br>WA | n/a | NetEpi | NetEpi | n/a | NetEpi | NetEpi NNDSS |
| Vic | n/a | NetEpi | NetEpi | NetEpi | NetEpi | NetEpi NNDSS |
| Qld | n/a | NetEpi | NetEpi (until 6 July)<br>NNDSS | n/a | NNDSS | NetEpi NNDSS |
| NT | n/a | NNDSS | NNDSS | n/a | NNDSS | NNDSS |
| All cases - Enhanced data[&] | | | | | | |
| ACT<br>NSW[#]<br>SA<br>Tas<br>WA | n/a | NetEpi | NetEpi | n/a | NetEpi | NetEpi |
| Vic | n/a | NetEpi | NetEpi | NetEpi | NetEpi | NetEpi |
| Qld | n/a | NetEpi | NetEpi (until 6 July)[*] | n/a | -* | NetEpi (until 6 July) |
| NT[*] | n/a | - | - | n/a | - | - |
| Hospitalised cases+ – Demographic data[&] | | | | | | |
| ACT<br>NSW[#]<br>NT<br>SA<br>Tas<br>WA | n/a | NetEpi | NetEpi | n/a | NetEpi | NetEpi |
| Vic | n/a | NetEpi | NetEpi | NetEpi | NetEpi | NetEpi |
| Qld (all) | n/a | NetEpi | NetEpi (until 6 July) | n/a | - | NetEpi |
| Qld (public and major private hospital patients only) | n/a | NetEpi | NetEpi (until 6 July)<br>EpiLog (public)<br>MS Excel Spreadsheet (private) | n/a | EpiLog (public)<br>MS Excel Spreadsheet (private) | NetEpi<br>EpiLog (public)<br>MS Excel Spreadsheet (private) |
| Hospitalised cases+ – Enhanced data[&] | | | | | | |
| ACT<br>NSW[#]<br>NT<br>SA<br>Tas<br>WA | n/a | NetEpi | NetEpi | n/a | NetEpi | NetEpi |

| State | Pre-Pandemic | Pandemic Phase | | | | Source for post-pandemic analysis |
|---|---|---|---|---|---|---|
| Vic | n/a | NetEpi (risk factors) | NetEpi (risk factors) | - | - | NetEpi (risk factors) |
| Qld (all) | n/a | NetEpi | NetEpi (until 6 July) | n/a | - | NetEpi |
| Qld (public and major private hospital patients only) | n/a | NetEpi | NetEpi (until 6 July) EpiLog (public) MS Excel Spreadsheet (private) | n/a | EpiLog (public) MS Excel Spreadsheet (private) | NetEpi EpiLog (public) MS Excel Spreadsheet (private) |
| Intensive care unit cases+ – Demographic and enhanced data[&] | | | | | | |
| NSW[#] NT SA Tas WA | n/a | NetEpi | NetEpi | n/a | NetEpi | NetEpi |
| ACT Vic | n/a | ANZICSi | ANZICS | ANZICS | ANZICS | ANZICS |
| Qld (all) | n/a | NetEpi | NetEpi (until 6 July) | n/a | - | NetEpi |
| Qld (public and major private hospital patients only) | n/a | NetEpi | NetEpi (until 6 July) EpiLog (public) MS Excel Spreadsheet (private) | n/a | EpiLog (public) MS Excel Spreadsheet (private) | NetEpi EpiLog (public) MS Excel Spreadsheet (private) |
| Mortality[$] – Demographic details | | | | | | |
| ACT NSW[#] NT SA Tas WA | n/a | NetEpi | NetEpi | n/a | NetEpi | NetEpi NNDSS |
| Vic | n/a | NetEpi | NetEpi | NetEpi | NetEpi | NetEpi NNDSS |
| Qld | n/a | NetEpi | NNDSS NetEpi (until 6 July) EpiLog | n/a | NNDSS EpiLog | NNDSS EpiLog |
| Mortality[$] – Enhanced data[&] | | | | | | |
| ACT NSW[#] NT[*] SA Tas WA | n/a | NetEpi | NetEpi | n/a | NetEpi | NetEpi |
| Vic | n/a | NetEpi | NetEpi | NetEpi | NetEpi | NetEpi |
| Qld | n/a | NetEpi | NetEpi (until 6 July) EpiLog | n/a | EpiLog | EpiLog |

[*] Enhanced data were only collected on hospitalised cases.
[&] Enhanced data completeness is highly variable by jurisdiction, data element and pandemic phase.
[+]Hospitalised cases represent a subset of all pandemic influenza confirmed cases. The NNDSS does not contain data on the hospitalisation status of cases or enhanced data.
[#] NSW maintained a separate instance of NetEpi and data were regularly imported into the national instance of NetEpi.
[$] Mortality data likely represents cases that have died in a healthcare based setting rather than out in the community.

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